MAIZE GENETICS COOPERATION

NEWSLETTER

68

March 15, 1994

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

Cloned it? Map it! (see pages 157 and 254)
Gened it? Name it! (see pages 154 and192)
The 1993 Standard for Nomenclature is reprinted in this issue
Mapped it? Share the data! (see page 213)
Can't find it? See pages 214-215 and inside back cover
Subscription wanted? See pages 255-256
Found an error? Say aha! and share it!

How to do it?

(see M. Freeling and V. Walbot, 1993,

The Maize Handbook, New York: Springer-Verlag)

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I. FOREWORD

The 'Cooperation' exists because you are a 'Cooperator' in keeping up the tradition of sharing maize genetics information with colleagues. The working research information here is shared with the understanding that each item is unpublished and is not to be cited in publications without specific consent of the authors.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:
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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 $85,000. 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 the regular research project of Ed Coe and Mike McMullen, and most recently as part of the Plant Genome Initiative project for development of a database prototype for maize. The extent and depth of the database and data syntheses will depend on continuity of the database support in the future.

Warm acknowledgment for help, advice and ideas during the past year is given to my colleagues, Mike McMullen, Mary Polacco, Georgia Davis, and Pat Byrne, for editing, for compilations and summarizations, for evaluating contents, and for providing creative advice. Their advice and encouragement, not to mention tolerance, is greatly appreciated.

Shirley Kowalewski once again skillfully edited and nurtured the contents from rough into fine form, twisted diverse electronic sources to suit and interpreted exotic scripts, structured the year's literature, and questioned quality or content, or 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. Thanks are also given to Lou Butler for 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 69, 1995), 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.

Details about the 1995 Maize Genetics Conference, at Asilomar, California, will be mailed to former attendees in November, 1994; 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 1994 Maize Genetics Conference is:

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It's a Gnarley One! (*Gn1*)

--Toshi Foster and Sarah Hake

Gnarley 1 (*Gn1*) is a new dominant mutation that was recovered by Dr. Tony Pryor at CSIRO, Australia. The mutation arose spontaneously as a single plant in a control population of a transposon tagging experiment, specifically, a family selected for the absence of active *Ac* at the *P* locus.

The *Gn1* phenotype is characterized by reduced internodal length, a sinuously curving culm (Fig. 1), lack of a distinct boundary between blade and sheath, and extra silks that originate from the base of mature carpels. The position of the extra silks suggests that they are transformed stamens. The mutation is evident in young seedlings, affects all nodes, and is fully penetrant in several backgrounds. We mapped the *Gn1* mutation to 2L using waxy reciprocal translocation stocks.

The *Gn1* phenotype is reminiscent of the *Knotted1* and *Rough Sheath1* phenotypes in the disturbance of the ligule region and the twisted stature. Since both *Kn1* (Vollbrecht et al., Nature 350:241-243) and *Rs1* (Freeling, Dev. Bio. 153:44-58) encode homeodomains, we speculated that *Gn1* may also fall in this class of homeobox-containing genes. Using the *kn1* homeobox as a low stringency hybridization probe, members of the Hake laboratory have isolated approximately 12 additional homeobox genes which have been designated *knox* for *kn*otted like homeob*ox*. The clones were mapped using Recombinant Inbred populations and one of them, *knox4*, was shown to map to 2L within one map unit of *bnl17.19b* at 2L162. When we used *knox4* as a probe on a Southern of a segregating population of 52 *Gn1* and 64 normal plants, we found only one recombinant, which was possibly misidentified as normal. Preliminary northern data indicates that *knox4* hybridizes to a transcript of about 1.6 kb. *knox4* is highly expressed in both normal and *Gn1* vegetative and ear meristems, is absent from normal leaves and is ectopically expressed in *Gn1* leaves. *knox4* is not ectopically expressed in *Kn1* mutant leaves, nor is *kn1* ectopically expressed in *Gn1* mutant leaves. These observations strongly support the hypothesis that *knox4* corresponds to *Gn1*. In situ analysis with *knox4* is in progress to determine which tissues or cells are ectopically expressing *knox4*. In order to prove that *Gn1* is a mutation of the *knox4* gene, we are trying to knock out the dominant phenotype with transposon insertions.

Figure 1. Photograph of the *Gnarley1* mutation courtesy of Dr. Tony Pryor.

***lethal ovule2* causes aberrant embryo sac development**

--Erik Vollbrecht

In each female floret, a single diploid cell within the ovule undergoes meiosis. Four haploid megaspores result, but only one persists and enters into megagametogenesis, or embryo sac (ES) development. In early megagametogenesis, the functional megaspore enlarges gradually and undergoes three rapid free-nuclear mitoses. In the next stage of ES development, simultaneous cellularization partitions the 8-nucleate structure into 7 cells: 3 antipodal cells, a binucleate central cell, an egg cell and 2 synergid cells. Only the antipodals undergo further cell divisions as the ES enlarges, differentiates and matures. Thus, the haploid megagametophyte phase of maize begins upon completion of meiosis and ends when the egg cell and the central cell of the embryo sac (ES) are fertilized by pollen-delivered sperm cells.

In 1952, O. E. Nelson and G. B. Clary serendipitously recovered the *lethal ovule2 (lo2)* mutation in the course of a screen for new male steriles (J. Hered. 43:205-210). They showed that although *lo2* transmits at normal frequencies through the male gametophyte (i.e., pollen), *lo2* does not transmit through the female gametophyte (i.e., ES). Thus, the most straightforward interpretation of *lo2* genetics suggests *lo2* is a mutation in a gene which is expressed by the haploid ES and required for normal ES function. Nelson and Clary did not further pursue *lo2*, but their initial, brief report is the source of the contemporary description of the *lo2* phenotype: "ovules containing *lo2* megaspores abort."

Confocal laser scanning microscopy (CLSM) was used to observe ovules and ESs on *lo2*-containing plants. CLSM analysis is much more rapid than traditional sectioning techniques, is nondestructive, and allows for detailed three-dimensional interpretations of histological features. *lo2* stocks were kindly provided by the Stock Center and by Ed Coe. Developing ears from *lo2*/+ plants were harvested and fixed, and the lower ears were testcrossed to verify the presence of *lo2*. Samples were stained to detect nuclei &/or cell walls with CLSM. In florets staged to contain mature ESs (as indicated by silk length), every ovule appeared to contain structurally normal sporophytic (diploid) tissues (i.e., nucellus, integuments, micropyle), and every ovule contained an ES-like structure. However, roughly 50% of the ESs

appeared wild-type and mature, while the remaining 50% lagged considerably in their development, displaying slightly enlarged 2- or 4-nucleate phenotypes. The 2- and 4-nucleate stages of ES development normally transpire very rapidly, such that they are difficult to observe even in appropriately staged (younger) florets. Even in very "old" florets, 2- and 4-nucleate ESs persisted and there was no sign of ovule degeneration, abortion, or abnormality associated with the presumptive *lo2*-containing ESs.

These observations are consistent with the hypotheses that *lo2* is a loss of function mutation, the *lo2* gene function is normally expressed by the haploid ES and that the *lo2* gene product is required during megagametogenesis as early as the 2-nucleate stage. Alternatively, the *lo2* mutation could be a gain of function in which a novel function (sporophytic or gametophytic in origin) is expressed at a similar stage. Moreover, ovule development can apparently continue undisturbed in the presence of aberrant ES development, and ES expansion and viability can be uncoupled from nuclear division patterns and cellularization. Additional genetic experiments are underway to determine whether *lo2* is transmitted at any detectable frequency through the ES, and to determine, using primary trisomic stocks, whether *lo2* behaves as dominant or recessive in the megagametophyte.

Deficiency analysis of megagametogenesis

--Erik Vollbrecht

By crossing together appropriately chosen reciprocal translocation stocks, one can construct euploid genotypes that, after meiosis, segregate (haploid) spores containing relatively small interstitial deficiencies (for discussion, see Birchler's chapter on segmental aneuploidy in The Maize Handbook.). Since haploid spores are the predecessors of the gametophytes (pollen and embryo sac [ES]), and deficient genotypes rarely transmit through either pollen or ES, gametophyte-specific functions are probably encoded by loci within such deficiency-defined regions. By determining the gametophytic defects conferred by deficiency for a particular region, one can infer the function(s) of gametophyte-expressed genes within that region. Thus, I am using translocation heterozygotes that segregate (balanced:duplicate:deficient) spores in a ratio of (2:1:1) to determine the affects of various loss of function genotypes on ES development. Stocks for segmental analysis of 1L and 3L were kindly supplied by Jim Birchler. The Stock Center supplied the *Tp9; Df3* stock, which was described in some detail by M. M. Rhoades (in Replication and Recombination of Genetic Material, Peacock and Brock, eds., 1968). The *Tp9; Df3* stock also segregates 25% deficient spores, which do not transmit through either gametophyte due to deficiency for ~10% of 3L. Immature ears were fixed and processed for analysis by confocal laser scanning microscopy (CLSM). Deficiency phenotypes are inferred from the frequency of occurrence of novel ES phenotypes in a population from a single ear. Analysis is still in progress, but phenotypes observed thus far include ESs that develop to the 2-nucleate stage and persist as immature ESs, ESs that degenerate precociously, 2-nucleate ESs with non polar nuclear distribution, and 4-nucleate ESs with nuclear degeneration at the micropylar pole but not the chalazal pole. Initial observations suggest that ESs containing the corresponding duplicate genotypes, which also segregate at 25% on the ears analyzed but transmit with regularity, may develop slower than do their euploid "siblings".

Loss of the dominant *knotted1* phenotype by EMS mutagenesis

--Laurie Smith, Randall Kerstetter and Sarah Hake

The *knotted1* (*kn1*) locus in maize is defined by a series of dominant alleles which cause sporadic localized outgrowths of tissue or knots on the lateral veins of the leaf blade. All of the existing alleles appear to be caused by insertion of transposable elements into introns of the gene except for *Kn1-O*, which is caused by a tandem duplication of the entire locus (reviewed by S. Hake, TIGS 8:109-114, 1992). The gene was cloned by transposon tagging (S. Hake, E. Vollbrecht and M. Freeling, EMBO J. 8:15-22, 1989) and shown to encode a homeodomain (E. Vollbrecht, B. Veit, N. Sinha and S. Hake, Nature 350:241-243, 1991). Analysis of the normal pattern of expression of *kn1* indicates that the mRNA and the protein are abundant in apical meristems and immature, unexpanded axes of vegetative and floral shoots (L. Smith, B. Greene, B. Veit and S. Hake, Development 116:21-30, 1992; D. Jackson, B. Veit and S. Hake, in press). *kn1* mRNA and KN1 protein are not detected in lateral organs of the plant and are apparently down-regulated even before the organ primordia are visible on the flanks of the meristem (Smith et al., 1992; Jackson et al., in press). Dominant *Kn1* mutations cause the gene to be expressed ectopically in lateral veins of immature leaves (Smith et al., 1992). None of the dominant alleles appear to disrupt the normal pattern of expression; they merely add a new component.

The dominant mutations provide information about the role of the *knotted1* gene outside of its normal context. In order to understand the role of *kn1* in the meristem, loss of function alleles are needed. A small deletion of *kn1*, *Def(Kn1)O*, was isolated which removes the entire gene but not the closely linked loci *alcohol dehydrogenase*, *adh1* (~1 cM), and *lemon white* (~1 cM) (J. Mathern and S. Hake, MNL 63:2, 1989). The deletion has no visible effects on the sporophyte as a heterozygote, but fails to pass through the male gametophyte. When *Def(Kn1)O* is uncovered in the progeny of crosses by the TB-1La translocation, the *Def(Kn1)O*-hypoploid embryo class displays an early embryonic lethal phenotype. We have recently found that at least one additional gene (*knox3*, a homeobox gene very similar in sequence and expression pattern to *kn1*) is missing in this deletion. Since it is unclear how many genes are missing in *Def(Kn1)O*, we cannot infer the phenotype of a loss of function allele of *kn1* from the phenotype of the deletion.

We have used the chemical mutagen ethane methylsulfonic acid, EMS, to induce loss of function mutations, which can be selected by screening for the loss of the dominant *Kn1-N2* phenotype. The *Kn1-N2* allele is correlated with the presence of a *receptor of Dotted* element, *rDt*, in the fourth intron of the gene (N. Sinha, E. Vollbrecht and S. Hake, unpublished results) and is closely linked to the *S* allele of *adh1*. The phenotype is both highly expressive and fully penetrant in seedlings and it is characterized by wide, white veins and one to many knots on the first several leaves. Pollen was collected from plants heterozygous for *Kn1-N2* and for *Def(Kn1)O* (which is never transmitted through the male gametophyte). The pollen was treated for 30 to 50 minutes in a 0.1% (v/v) emulsion of EMS in mineral oil according to the method of M. G. Neuffer (Mutagenesis in The Maize Handbook, eds. M. Freeling and V. Walbot, Springer-Verlag New York, Inc., pp. 212-219, 1993) and applied with a brush to silks of the inbred line B73 (Pioneer), which carries a normal allele of *kn1* closely linked to the *F* allele of *adh1*. Seeds were collected and planted at high density and screened as seedlings for individuals without knots.

Of approximately 13,000 seedlings screened, 60 showed no sign of knots (this number included many self contaminants which might have been reduced had we detasseled the female parent). Various other mutations were observed in the population, including putative oil yellow seedlings, indicating the mutagenesis was effective. The EMS induced derivatives no longer expressing the *knotted* phenotype were selfed (where possible) and the progeny were examined for absence of knots, and tested for the closely linked *adh1-S* allele and for the presence of the *rDt* element associated with the *Kn1-N2* allele in order to weed out normal individuals resulting from pollen contaminants. Ten families carried the proper markers and continued to show no sign of knots.

As a preliminary step toward characterizing the changes at the *kn1* locus resulting from EMS mutagenesis, we looked for changes in the KN1 protein produced by these *Kn1-N2* derivatives on Western blots. Homozygous *adh1-S* seeds from each were planted, and seedlings were harvested at 3 to 5 weeks after planting. Leaves longer than 5mm were removed and the meristem and a few millimeters of ground tissue were harvested and proteins extracted. Western blots were made and the KN1 protein was visualized with affinity purified polyclonal antibody specific to KN1 (as in Smith et al., 1992). Equal quantities of protein were loaded in each lane and protein from B73 meristems was run for comparison. The results for 6 out of 10 of the derivatives are shown in Figure 1. Each derivative family produces at least some KN1 protein, and no novel proteins reacting with the anti-KN1 antibody were observed. Four derivative families produce normal levels of KN1 protein (shown in Figure 1: ems34, ems27, ems18 and ems16), while six show significant reductions in the levels of KN1 protein, possibly due to a point mutation rendering the protein unstable (two of these shown in Figure 1: ems38 and ems4). Several of the derivatives still show some level of ectopic expression in young leaf tissue (data not shown). In each case, however, the levels appear lower than in leaf tissue from a homozygous *Kn1-N2* mutant. All of the homozygous *adh1-S* progeny of the derivatives appear normal as seedlings, including the two with the greatest reductions in KN1 protein levels, ems38 and ems4. A thorough examination of adult phenotypes is in progress. So far, homozygotes from one derivative (R848-6) appear to have markedly reduced seed set when compared to heterozygous sibs.

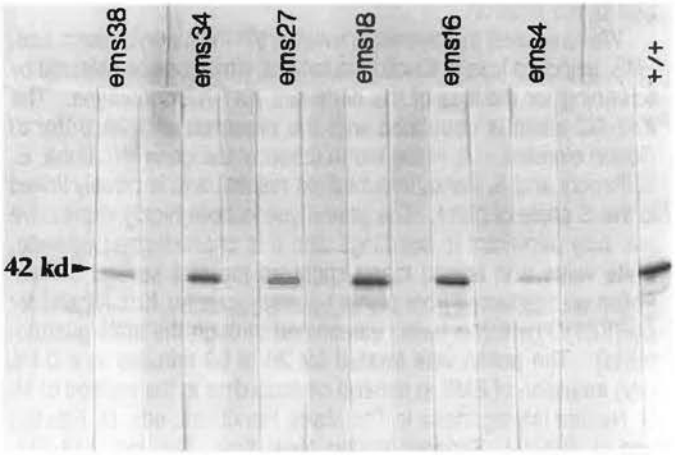

Figure 1. Western blot with anti-KN1 antibody on proteins extracted from individuals homozygous for six different EMS-induced derivatives of *Kn1-N2* that no longer condition the *Kn1* mutant phenotype, and a wild-type control (right end). Methods and materials as described in Smith et al. (Development 116:21-30, 1992).

Our efforts to generate loss of function alleles at the *knotted1* locus by EMS mutagenesis of a mutant allele have yielded several new alleles that no longer condition the *Knotted* phenotype. Since all of them make at least some KN1 protein, we may not yet have isolated a complete null allele. The isolation of derivatives that have significantly reduced levels of the KN1 protein suggests we have isolated events that reduce expression below a threshold necessary to induce knot formation or that alter the stability of KN1 protein. Derivatives with levels of protein comparable to normal suggest qualitative alterations of the KN1 protein that may impair its function or cause loss of its expression in the cells necessary to cause knot formation. Further characterization of the specific defects may point toward significant elements of the protein or promoter of the maize homeobox gene, *kn1*.

AMES, IOWA
Iowa State University

An attempt to tag *rhm* with transposable elements

--Ru-Ying Chang and Peter A. Peterson

The *rhm* gene controls resistance of maize plants to the disease southern leaf blight, which is caused by the fungus *Helminthosporium maydis (Bipolaris maydis)*. The homozygous recessive allele *rhm/rhm* confers resistance, while the dominant allele *Rhm* is associated with susceptibility. Lines are susceptible (*Rhm/Rhm*) unless bred for resistance. This greatly accelerates the transposon tagging process due to ease of constructing element-laden genotypes for mutant screening.

Transposable elements randomly insert into a locus at a frequency of 10^{-6} to 10^{-5} (Peterson, MNL 59:3, 1985; Döring Maydica 34:73-78, 1989). This project is designed to tag the *rhm* gene with transposable elements based on random insertion of transposable elements into the gene. Because normal lines are susceptible to the fungus, transposable element lines are of the allele composition of *Rhm/Rhm El El* (El, abbreviation of transposable elements). Element insertion into the *Rhm* gene will abolish the function of the *Rhm* allele. Hybridization of genomic Southerns from the mutant with a probe from the element and subsequent cloning will enable us to isolate the gene.

In order to detect an element insertion into the *Rhm* allele, the genotype *Rhm/Rhm El El* was testcrossed by an *rhm/rhm* tester. This cross yields an *Rhm/rhm El* F1, which is the genotype used for mutant screening. This genotype is susceptible to the fungus, while the mutants (designated *rhm*/rhm*) with an element insertion are resistant.

As of now, eighteen mutants have been obtained out of approximately four hundred thousand seedlings screened. The results are listed in Table 1 by element categories.

Table 1. Screening results of *rhm* tagging. The screening material, *Rhm/rhm El*, was the progeny from the crosses of *Rhm/Rhm El El* x *rhm/rhm*. Artificial inoculation was used at 3-5 leal stage in a greenhouse.

Pop'n	Element	Total screened	No. of mutants	Designation	Mutation rate
1	*En*	12,000	1	rhm-m-1	8.33×10^{-5}
2	*CyTEL*	78,200	6	rhm-m-1-rhm-m-7	7.67×10^{-5}
3	*Cy*	263,840	11	rhm-m-8-rhm-m-18	4.17×10^{-5}
4	*T4-6(033-16)~c2-m1~Rhm*	47,391	0		0.00
5	*T4-6~c2-m1~Rhm/Cy bz-rcy*	26,282	many		$\sim 5.00 \times 10^{-2}$

The mutation rates are similar for the first three classes, *En, Cy* and *CyTEL*. No mutants were found out of 47,391 seedlings screened from the T4-6(033)-16~*c2-m1*~*Rhm* line (line with *c2-m1* [*En* in the *C2* gene] labeled 6S). The labeling was to increase the frequency of transposon insertion into the *Rhm* allele by taking advantage of the transposition preference of transposons to closely linked sites. These results with unexpected low frequency of mutation may be explained in the following two ways. i) It was originally suspected that the *rhm* gene was at the centromere of chromosome 6. Recently it was shown to be located at the end of 6S (Zaitlin et al., Genome 36:555-564, 1993). T4-6(033-16) has a break point of 0.9 on 6S. It is possible that the *rhm* locus is not linked to *c2-m1*, but moved to 4L, which has the other break point of the translocation. The *c2-m1* allele was moved from 4L to 6S. Thus the enhancement expected with close linkage was not in effect. ii) Based on random insertion, 10^{-5} to 8 x 10^{-5} as shown in Table 1, 0.5 to 4 mutants are expected. Because of this expectation, the screened population may not be large enough to ensure a mutant to be found.

Another unexpected result deserves careful examination. When *Cy* and *T4-6(033-16)~c2-m1~Rhm* line were used separately in the screening, the mutation rates were 4.17 x 10^{-5} and zero, respectively. However, when the two lines were crossed together to increase vigor and the F1 was testcrossed by *rhm/rhm* to develop the screening seed, mutants were found at a high frequency of around 5% from screening the testcross population. This result was unexpected.

To explain these results, it is hypothesized that two closely linked recessive genes are involved in determining resistance in this case. Each of the two parents has a dominant allele of one gene and a recessive allele of the other. This aspect is discussed in some detail in an accompanying report (see Chang and Peterson in this issue).

The mutants obtained (*rhm*/rhm*) were crossed to an *Rhm/Rhm* line in order to separate *rhm** from *rhm*. The two genotypes yielded from this cross, *Rhm/rhm** and *Rhm/rhm* in equal ratio, can be distinguished using RFLP analysis provided a suitable probe is chosen. A probe, *umc85*, which detected polymorphisms between *Rhm/Rhm* and *rhm/rhm* lines, was chosen in this case. Because *rhm** was derived from *Rhm*, it should possess the band specific to the *Rhm/Rhm* line, while *rhm* should possess a band specific to the *rhm/rhm* line. Nine of the 18 mutants were analyzed in this way by our collaborators in Dr. A. Gierl's lab in Köln, Germany. Five of the nine showed the expected banding pattern: the 1/2 *Rhm/rhm** possessed a single band while the 1/2 *Rhm/rhm* possessed both bands. When genomic Southerns from the mutants as well as from the wild-type lines were probed with an element sequence, no cosegregating band was detected.

Failure to detect a cosegregating band may stem from several reasons. First, the number of mutants tested is not large enough. Secondly, the RFLP analysis used distinguishes between *rhm** and *rhm*. It tells us whether *rhm** is derived from the *Rhm/Rhm* line. It does not show the nature of the mutation. The *rhm** allele might have been derived from spontaneous mutations from *Rhm* to *rhm* or due to another transposon. This project is currently underway. More mutants are being generated and analysed at this time.

Resistance to *Helminthosporium maydis*: one gene or two genes?

--Ru-Ying Chang and Peter A. Peterson

In the *rhm* tagging project, the mutation rates (susceptible *Rhm* to resistant *rhm*) for different elements usually ranged from 4 x 10^{-5} to 8 x 10^{-5} (see Chang and Peterson, accompanying report). The *Cy* population yielded a mutation rate of 4.17 x 10^{-5}. No mutant was found for the T4-6(033-16)~*c2-m1*~*Rhm* population out of 47,391 seedlings screened. The two populations were combined into a heterozygote (crossed to increase vigor) and testcrossed by an *rhm* tester to develop screening seed. Screening of the testcross progeny from this heterozygote, however, yielded around 5% resistant mutants. The figure 5% was an estimate since no precise counting was made. This result certainly cannot be explained by random insertion of the element or natural mutation or both.

It is hypothesized that two closely linked recessive genes (*rhm1* and *rhm2*) control resistance to *Helminthosporium maydis* (*Bipolaris maydis*), the causal fungus of southern leaf blight. The linkage distance is roughly 5% x 2 = 10%. Figure 1 is a diagram of the hypothesis proposed.

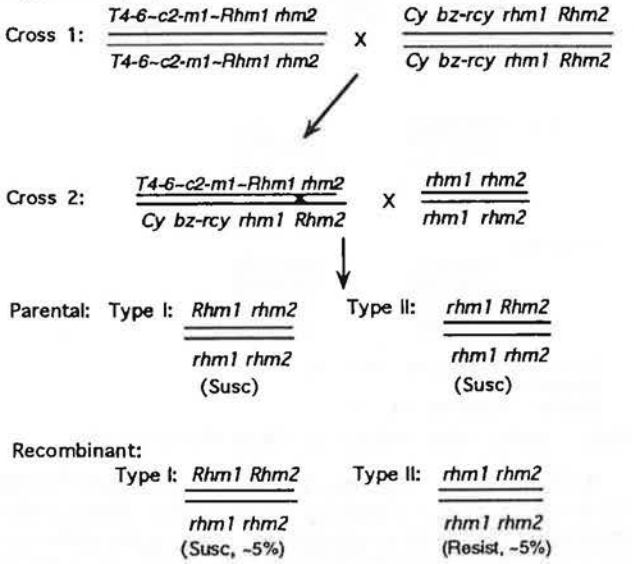

Figure 1. Model for resistance controlled by two linked recessive genes. T4-6, T4-6(033-16)

The two closely linked genes are temporarily designated *rhm1* and *rhm2*, with no firm knowledge of the relationship of the two genes to the *rhm* gene proposed by Smith and Hooker (Crop Sci. 13:330-331, 1973). The dominant gene in T4-6(033-16)~*c2-m1*~*Rhm* is arbitrarily designated *Rhm1* and that in the *Cy* line, *Rhm2*. According to this, the *rhm* tester will have two recessive genes. The two genes have coordinated effects so that only the genotype *rhm1 rhm2//rhm1 rhm2* is resistant to the fungus. A dominant allele at either locus will abolish resistance. The cross described above between the two parents would have yielded a genotype with the two genes in repulsion phase, *Rhm1 rhm2//rhm1 Rhm2*. In the testcross progeny with the *rhm* tester, there are two types of parental genotypes which produce susceptible seedlings. There are two types of recombinants expected in the progeny, whose frequency depends on the linkage between the two genes. One of the two recombinants is *rhm1 rhm2//rhm1 rhm2*. It is this recombinant type that yielded the ~5% resistant "mutants". The other recombinant genotype *Rhm1 Rhm2//rhm1 rhm2* is phenotypically indistinguishable from the majority (parental) seedlings. Therefore, the linkage distance between the two genes should be 5% x 2 = 10%.

A strategy has been developed to test this hypothesis. The

rhm locus has been shown to be located at the end of 6S. Though an RFLP marker, *umc85*, was located close to the locus (Zaitlin et al., Genome 36:555-564, 1993), it was not determined whether this marker is proximal or distal to the locus. Thus a strategy based on RFLP analysis awaits a firm distal marker to the locus. The strategy presented here (Fig. 2) is a purely genetic one.

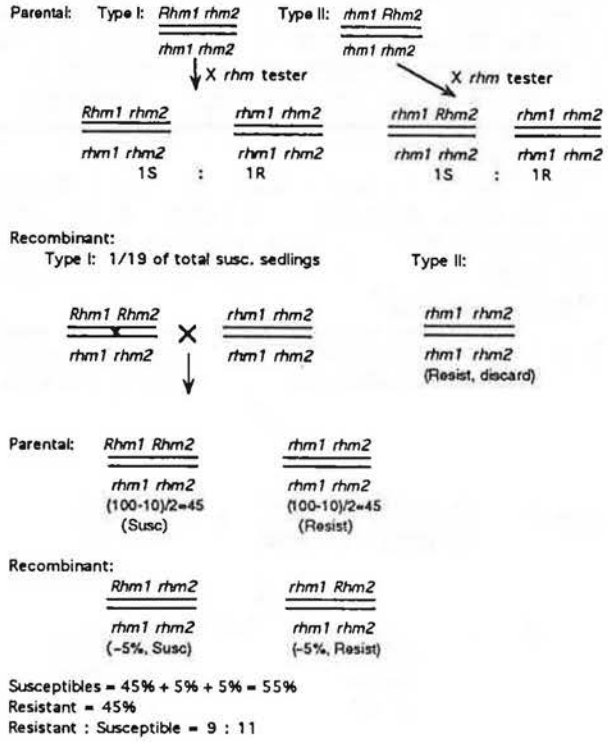

Figure 2. Test of hypothesis of resistance controlled by two linked recessive genes

1) The seed for screening (testcross progeny from the cross T4-6~*c2-m1~Rhm1 rhm2//Cy bz-rcy rhm1 Rhm2* x *rhm1 rhm2//rhm1 rhm2*) is to be planted again. Among the progeny are the two types of nonrecombinant seedlings as well as the two types of recombinant seedlings. 2) Susceptible seedlings from lines that produce ~5% resistant individuals are to be transplanted and testcrossed by the same *rhm* tester. The two parental types will produce 1R : 1S seedlings since no crossover will yield a different phenotype. The recombinant type, however, will yield new recombinants. This will increase the proportion of susceptible seedlings by ~10%. Thus a new ratio 9R : 11S will result instead of 1R : 1S (10R : 10S).

The hypothesis of two linked *rhm* loci can be tested as outlined in Figure 2. The expected frequency of the susceptible recombinant class *Rhm1 Rhm2//rhm1 rhm2* from cross 2 (Fig. 1) is 5% in the progeny. If the 5% resistant class is excluded, the frequency of susceptible recombinant class among all susceptible seedlings will be expected to be ~5% out of 95%, i.e., 1/19. This class can be recognized since it will produce a 9R : 11S distorted ratio. Using the formula given by Sedcole (Crop Sci. 17:667-668, 1977), the number of plants needed to be testcrossed is 67 in order to obtain at least one progeny yielding the distorted ratio at the 95% confidence level. Using the formula given by Clarke and Carbon (Cell 9:91-99, 1976), the number would be 55. Lastly, the testcross progeny will be tested for the ratio of resistant versus susceptible seedlings.

Chlorotic lesion resistance to southern leaf blight was first identified by Craig and Daniel-Kalio (Plant Dis. Rep. 53:134-136, 1968) in Nigeria. Craig and Fajemisin subsequently studied the inheritance of resistance. They reported (Craig and Fajemisin, Plant Dis. Rep. 53:742-743, 1969) that resistance was controlled by two closely linked recessive genes with a linkage of 16.83%. Using the material of these authors, Smith and Hooker (Crop Sci. 13:330-331, 1973) tested the inheritance of resistance on a large scale. From the data obtained, they concluded that resistance is controlled by a single recessive gene, designated *rhm*. However, Smith and Hooker used only one plant of the Nigerian material in the crosses. Utilization of a single plant could have excluded the second gene if the Nigerian material was not homogeneous. Later, Thompson and Bergquist (Crop Sci. 24:807-811, 1984) reported two independent recessive genes controlled seedling resistance, among other types. Holley and Goodman (Plant Dis. 73:562-564, 1989) reported new sources of resistance with more complicated inheritance patterns.

A few inferences can be drawn from this study. i) The data obtained in this experiment exclude the possibility of two independently segregating loci. The two genes involved in the two parental lines (cross 1, Fig. 1) were apparently linked. They behaved like the genes originally described by Craig and Fajemisin. ii) The two genes in this study interact in a coordinated manner similar to that proposed by Craig and Fajemisin and by Thompson and Bergquist. iii) One of the two genes could be the *rhm* gene proposed by Smith and Hooker, since the two parents, when separate, behaved as though only one gene were involved. iv) Although not possible to exclude, other epistatic effects, except the coordinated interaction described above, are not likely to have been a major force in determining resistance since the newly derived resistance type strongly resembled the *rhm* tester. This, at least, excludes the types of interaction observed by Holley and Goodman that resulted in intermediate levels of resistance.

How then to visualize the coordinated gene action that results in resistance with two genes? The most likely explanation is that one of the two genes is a copy of the other created by duplication. The dominant alleles are responsible for a gene product that permit the fungus to proliferate. The recessive alleles are null alleles. A dominant allele in either of the two copies of the gene will be able to make the gene product, and thus, will abolish resistance.

Multiple gene loss on the short arm of chromosome 9 in *C-m925408U* is not induced by *Ac*

--Vijay Thatiparthi and Peter A. Peterson

The allele *C-m925408U* is characterized by the coordinate loss of *Yg2, C, Sh, Bz* and *Wx* genes (sectored phenotype) present on the short arm of chromosome 9. This coordinate loss is ascribed to chromosome breakage. This breakage is initiated late in kernel development and is evidenced by small colorless or bronze (*bz*) sectors (depending on the genotype). Some of the sectors are so small that the bronze sectors (resulting from the loss of the *Bz* gene) are not readily revealed because of the diffusion of the *Bz* product from the surrounding *Bz* tissue into the bz sectors. Large sectors illustrative of early breakage are observed occasionally.

As expected, the sectored phenotype is more obvious when the mutant is crossed as male than when crossed as female. Kernels which are colored (with no discernible bronze sectors) when stained with I/KI solution revealed the loss of the *Wx* gene. We presume that the lack of bronze sectors might be due to diffusion

of *Bz* product rather than due to the insertion of breaker or related elements into the *Wx* gene. However, the number of waxy sectors is less than observed when the mutant is crossed as male. Thus the dosage of breaker (which is thought to be autonomous) does not seem to influence the frequency of breakage. We recently obtained kernels with the *C-I* allele linked to the breaker. The loss of *C-I* in these kernels can be identified easily and we hope the pattern of breakage in these kernels can be used to identify more clearly the male-female differences observed in this mutant.

In plants grown from the *Yg2 C Sh Wx bk/ yg2 c sh wx* kernels, the loss of *Yg2* is revealed when the plants are as young as 7-8 day old seedlings (two leaf stage). Among twenty plants screened, one plant showed 3 to 4 very early occurring *yg2* sectors. In the remaining plants the *yg2* sectors range from very late occurring loss to early occurring loss. The number of *yg2* sectors ranges from 1-4. The colored round waxy kernels segregating on the same ear as the above kernels are used as control. Three out of sixteen control plants show 1 to 3 late occurring *yg2* sectors. A much more thorough quantitation of the *yg2* loss will be undertaken using one month old seedlings.

The loss of *C* in kernels is always correlated with the loss of *Wx*, indicating that the chromosome breaking structure is located proximal to the *Wx* locus. The limited mapping data obtained indicate that the breaker is located about 3 to 5 map units proximal to the *Wx* locus. A few exceptional kernels were obtained in which the breakage is initiated between the *Bz* and *Wx* genes. This phenotype indicates that the breaker carrying unit is transposable. The genetic ratios of the sectored kernels suggest that the breakage is caused by an autonomous transposable element.

This breakage mutant was screened for the presence of *Ac, Uq, En* and *Cy* transposable elements. System tests revealed that *C-m925408U* contains only *En* and *Cy* elements. Thus *Ac*, which is known to cause chromosome breakage, is not involved in causing the breakage observed in this mutant. Our initial attempts to correlate the breakage either with *En* or *Cy* were not successful because of high copy number of both *En* and *Cy*. Further tests will be undertaken to establish the system relationship.

In maize most of the transposable element mediated chromosome breakage alleles are caused by the *Ac-Ds* system (McClintock, 1946, 1947, 1948, 1949; Dooner and Belachew, 1991; Weil and Wessler, 1993). Apart from *Ac* only *En/Spm* is known to cause chromosome breakage (Cormack and Peterson, in press). Since the *Mu* element is shown not to cause chromosome breakage (Rowland et al., 1989) it will be interesting to see whether the chromosome breakage in *C-m925408U* is caused by *Cy*.

Genomic regions affecting plant height in maize and sorghum

--M. G. Pereira and M. Lee

We have constructed a complete genetic linkage map of sorghum based on restriction fragment length polymorphisms (RFLPs) detected by maize genomic and cDNA probes (Pereira et al., Genome 1994). To facilitate comparisons with maize, some probes were selected on the basis of linkage information reported in newsletter compilations, notes, and our unpublished results (Spike and Lee, MNL 67:6-7, 1993; M. Lee, unpublished). The sorghum parents used to create the mapping population (CK60 and PI229828) were chosen to represent phenotypic extremes for panicle morphology, leaf dimensions, tillering, plant height and resistance to biotic stresses. In this note, we summarize the mapping results for RFLP loci associated with plant height in sorghum and review some evidence for orthologous regions in maize. A complete presentation will be reported elsewhere (Pereira and Lee, Theor. Appl. Genet., 1994).

Linkage between RFLP and quantitative trait loci (QTL) in sorghum was assessed with a sample of 152 F2 plants at 111 loci. Four independent QTL for plant height were identified (linkage groups A, B, E, and H). Individually, the QTL accounted for 9 to 29% of the phenotypic variation (63%, collectively) with positive additive effects of 15 to 32 centimeters. Alleles for increased plant height were derived from the tall parent (PI229828) and with one exception (QTL of linkage group H) tallness was dominant. Three of the QTL were also significantly associated with several other traits while the effect of the fourth QTL (linkage group E) was limited to plant height. Based on these observations, we have hypothesized that the plant height QTL of linkage groups A, E, and H correspond to sorghum genetic loci *Dw3*, *Dw4*, and *Dw2*, respectively. The *Dw* (dwarf) loci have been identified usually on the basis of recessive alleles with highly qualitative effects on plant height (primarily a reduction of internode length). Dwarfing alleles at the loci (*Dw1-4*) have been routinely manipulated in breeding programs and have been backcrossed in germplasm adaptation programs.

Placement of RFLP loci in maize and sorghum maps with common probes suggests the plant height QTL of sorghum linkage groups A, E, and H may be orthologous to plant height QTL reported for maize chromosomes 1, 6, and 9, respectively. In each case, the confidence intervals of the sorghum QTL are within those reported for maize (Beavis et al., Theor. Appl. Genet. 83:141-145, 1991 for QTL on chromosomes 1 and 9; Veldboom et al., Theor. Appl. Genet., 1994 for QTL on chromosomes 1 and 6). Also, linked or pleiotropic effects of some sorghum plant height QTL resemble those of mutant alleles at maize genetic loci in the putatively orthologous regions. On chromosome 1, two genetic loci (*an1* and *br1*) seem to be included within the confidence intervals reported by Beavis et al. and Veldboom et al. The mutant phenotype of *an1* is an andromonoecious, gibberellin-responsive dwarf with short leaves and few tassel branches. The sorghum QTL of linkage group A has significant effects on the number of primary branches per panicle. On chromosome 9, the *d3* locus appears to be within the maize QTL confidence interval. The mutant phenotype of *d3* is an andromonoecious, gibberellin-responsive dwarf with thickened broad leaves and a compact tassel. Likewise, the sorghum QTL of linkage group H has significant effects on leaf blade and panicle dimensions (length and width). Similar parallel effects for maize chromosome 6 and sorghum linkage group E were not apparent.

Comparative linkage analysis of RFLP loci and QTL in F2:3 and F6:7 recombinant inbreds

--D.F. Austin and M. Lee

The first objective of our study was to identify RFLP loci associated with agronomic traits in inbred progeny of an elite maize population. The second was to determine the minimum number of QTL controlling each trait and the proportion of the total phenotypic variation explained by each locus. The recombinant inbred (RI) population was derived from a cross between elite inbred lines Mo17 and H99, which differ for several traits including insect resistance, kernel size, grain yield, ear length, plant height, and flowering date. From the original cross, 186 unselected F6:7

lines were developed. By using RIs, we expect to detect smaller phenotypic effects because of increased replication of the homozygous parental marker classes. The power of detecting significant differences between the homozygous marker classes in a RI mapping population is increased over an F2 derived population of equivalent size. This is due to the reduction of the expected frequency of heterozygous individuals from 50% in the F2:3 to 3% in the F6:7 at any given locus.

A linkage map consisting of 100 RFLP loci and 1 morphological marker was developed using Mapmaker. The map consisted of ten well characterized linkage groups with a total map length of 1408 cM and an average interval between loci of 15.4 cM. Veldboom et al. (Theor. Appl. Genet., 1994) produced an RFLP map in the same population using 150 F2:3 lines with 103 RFLP loci and 1 morphological marker. Their total map length was 1419 cM with an average interval length of 15.0 cM. Between the two studies, 84 loci are in common. Marker order within chromosomes is conserved between maps with one exception. At the end of the long arm of chromosome 9, the order of two loci, separated by 2 cM on the RI map, is reversed. Burr et al. (Genetics 118:519-526, 1988) discussed the expectation of a two-fold expansion of the map for closely linked markers when mapping with RIs. To analyze this expectation, we compared common loci intervals which were less than 15 cM in the F2:3 map. Of the 37 intervals, 17 are larger and 21 smaller in the RI map. The average expansion of the 17 intervals which are larger in the RI map is 1.3X.

Single-factor analysis of variance was conducted on all locus-trait combinations to test for significant differences between the homozygous marker classes. For plant height, 32 marker loci were significant (.05) and were located on 7 chromosomes. Significant regions include 1L, 2S, 2L, 3L, 4S, 4L, 5L, 7L, and 8L. The proportion of phenotypic variance explained by marker classes for individual markers ranged from 2.1% to 12.3%. In three of the regions (3L, 4S, and 7L) H99 alleles are associated with increased plant height. The remaining regions are associated with Mo17, the taller parent, contributing the positive effects. Veldboom et al. identified putative QTL using Mapmaker QTL for plant height on 1L, 2S, 4S, 6L, and 7L. Four of the five regions were also identified in our study. Loci *umc37* (1L), *umc34* (2S), and *umc35* (7L) were identified in the previous study and were significant(.01) in the present study. Both studies identified region 4S but different loci were indicated. In all four regions, the parent contributing the positive effect is consistent between studies.

Several additional traits have been measured on the RI population and will be analyzed in a similar manner. Additional traits to be analyzed include ear height, silking date, anthesis date, silk delay, yield and yield components.

Cloning of *sugary1* by transposon tagging with *Mutator*

--Martha G. James and Alan M. Myers

A previous report identified several alleles of *sugary1* (*su1*) in *Mutator* backgrounds (MNL 66:8). These alleles exhibit a range of phenotypic expression, from wrinkling only at the crown to extremely shrunken and glassy kernels. These variations may be due to background effects, or they may result from position effects of the transposon insertions. Recently, we have identified three additional alleles of *su1*. These are *su1-489* (a gift from Barbara Kloeckener-Gruissem), *su1-A1*, and *su1-A2* (gifts from Mark Alfenito). *su1-489* arose in a *Mutator* background, and *su1-A1* and *su1-A2* arose in an *Ac*-background.

Southern hybridization analysis of DNAs from one of the putative *Mu*-induced alleles, *su1-4582*, identified a *Mu1*-homologous, 4.0 kb *Eco*RI restriction fragment that cosegregated with the mutant phenotype. The 4.0 kb fragment was present in 60 *su1-Ref/su1-4582* DNA samples, and absent in 57 *su1-Ref/+* sibling DNA samples. A genomic clone corresponding to this 4.0 kb *Eco*RI fragment was identified and isolated from a bacteriophage lambda library by hybridization with *Mu1* (Fig. 1). A hybridization probe from the genomic region flanking the *Mu1* insertion ("*Su1* probe") also identified a specific 4.0 kb *Eco*RI fragment in DNAs derived from mutant kernels. Furthermore, the *Su1* probe identified similar polymorphisms in DNAs from two independent alleles of *su1*, *su1-7110* and *su1-3162*. Thus, it is likely that the genomic DNA flanking the *Mu1* element in the *su1-4582* clone contains at least a portion of the *Su1* locus.

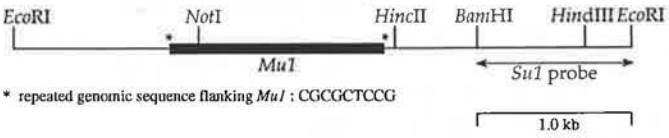

Figure 1. Restriction map of the cloned genomic *Eco*RI fragment that cosegregates with *su1-4582*. The black bar indicates the position of *Mu1* within this fragment.

A transcript of approximately 3.5 kb was identified in polyadenylated RNA isolated from wild type kernels by hybridization with the *Su1* probe. Comparisons with poly A^+ RNAs from three of the *Mu*-induced *su1* alleles showed that this transcript was missing in RNA from *su1-2412* kernels, was greatly reduced in both size and abundance in *su1-4582* kernels, and was slightly larger than the wild type transcript and of roughly equal abundance in RNA from *su1-7110* kernels. These differences and their possible relevance to observed phenotypic variations among the transposon-induced alleles are being investigated. A partial cDNA clone homologous to the genomic clone has been isolated from a kernel cDNA library (a gift from Karen Cone).

Some thoughts on the nature and utilization of the *Mutator* system

--Donald S. Robertson

Because I have retired and my health will not permit me to continue my research, this will probably be the last article to be included in the Newsletter. Thus, I think it would be helpful to take this opportunity to summarize various miscellaneous aspects of the *Mutator* system that have not been reported before or to emphasize certain observations previously made and, where appropriate, comment on their significance for future research.

1. Tests for *Mutator* activity. The standard test I have used for the presence of an active *Mutator* has included the following elements: a) Self-pollinate the *Mutator* parent. b) Outcross the *Mutator* plant to a standard line (or any non-*Mutator* stock). c) If possible self-pollinate the second ear of the outcross parent. d) Seedling test the selfed ears of both parents to determine that neither is segregating for a new mutant. e) Seedling test the progeny of self-pollinated ears of 50 plants of the outcross progeny and score for new seedling mutations expected if the *Mutator* parent was active. Instead of seedling tests, some investigators have used the segregation of defective kernels on the self-pollinated ears of the outcross plants. This is probably an acceptable technique but I think there is a higher risk of error in scoring a given plant as having an active *Mutator* system than when seedling tests are used. I have found that spontaneous defective kernel

mutants occur with a relatively higher frequency than seedling mutants and that environmental factors also can result in the production of the defective kernel phenotype. An added advantage of the seedling test is the opportunity to observe instability (mutability) in the mutants, which is seldom possible if only the defective kernel phenotype is utilized. The presence of mutability is a sure indicator that a *Mutator*-induced mutation has occurred.

2. Somatic mutability is an unreliable indicator of *Mutator* activity. In my research I have extensive data demonstrating that there is no correlation between somatic instability and *Mutator* activity (i.e., the ability to induce a high frequency of new mutants) of a plant from a mutable kernel or a mutable plant. From what now is known about the molecular basis of the *Mutator* system, such a lack of correlation is not surprising. All that is required for somatic mutability is a *MuDR* element at the mutant locus, or one of the receptor elements at the locus in addition to a *MuDR* element elsewhere in the genome. Thus, there can be somatic mutability with only one or two elements in the genome, while active *Mutator* stocks usually possess numerous elements. It is the large number of elements in the genome of active *Mutator* plants that is responsible for their high mutation frequencies. This does not mean that plants from mutable kernels or mutable plants with only one, two, or just a few elements are incapable of producing new mutations. However, the mutation frequency will be much lower in such plants than in plants with numerous elements.

3. The effects of an inbred condition on germinal and somatic mutability. Generally, the more inbred a *Mutator* stock the less likely it is to have *Mutator* activity in both the germ line and the soma. This is true whether the inbred condition is due to inbreeding per se or is the result of crossing the germinal *Mutator* system or an unstable *Mutator*-induced mutant into an inbred line. In some genetic backgrounds, this loss of activity may happen in fewer generations than others, but I have not observed any background where eventually an inbred condition will not eliminate both types of *Mutator* activity.

4. *Mutator*-induced mutants. *Mutator*-induced mutants and their storage location are summarized in the accompanying table..

5. The response of the *Mutator* system to ultraviolet light irradiation. When mature pollen from active *Mutator* plants was irradiated with U. V. for 30, 35, 40, 45 seconds a synergistic affect was observed. If these observations are valid (they need to be repeated), they suggest that the mechanism involved in repairing U.V. damage to DNA might create a situation that is amenable to the transposition of *Mutator* elements.

6. *Mutator* activity in the early development of the embryo. The mutants segregating in the progeny of many self-pollinated ears occur in less than the 3:1 ratio, which is expected if the mutant allele was carried by the pollen grain or the egg responsible for the self-pollinated plant. One of the possible explanations for this phenomenon is that instead of the mutant being carried in the gamete from the *Mutator* parent, it was induced early in the development of the embryo of the plant to be self-pollinated. A mutation occurring in a cell before the cell lineage of the tassel and ear separated, and whose descendent cells made up a portion of the meristems of both inflorescences, could account for such non-Mendelian ratios. Another evidence of mutations occurring in the early development of the embryo was the observation that in some tests for *Mutator* activity self-pollinated ears of *Mutator* plants, which were outcrossed, would not segregate for a new mutant. However, half or less than half, but a good portion, of self-pollinated ears from the plants of the outcross progenies would segregate for a new mutant. Such an observation suggests that a mutation occurred during the development of the embryo in the cell lineage giving rise to the tassel after the tassel and ear cell lineages had separated. Preliminary tests support the occurrence of both of these kinds of events, but much more data is needed to confirm this conclusion.

	Storage location	
	COOP	ISU
vp5-Mum (Several independent isolates.)	X	
vp5-Mu3076-36	X	
vp9-Mum (Several independent isolates.)	X	X
vp9-Mum2(3111-5)	X	
a1-Mum (5 independent isolates.)	X	X
a1-Mus (When first isolated mutants were stable. 4 independent isolates.)	X	
bz1-Mum (17 independent isolates.)	X	X
bz1-Mus (When first isolated mutants were stable. 10 independent isolates.)	X	X
a2-Mum (4 independent isolates.)	X	X
a2-Mus (When first isolated mutants were stable. 3 independent isolates.)	X	X
wx1-Mum (15 independent isolates.)	X	X
wx1-Mus (When first isolated mutants were stable. 3 independent isolates.)	X	X
sh1-Mu (7 independent isolates.)	X	
bt2-Mu1(9626-11)	X	
Mutator-induced opaques (8 independent isolates, not *o2*.)	X	
bt1-Mu4206	X	
fl2-9234	X	
o2-Mum1	X	
o2-Mum3b	X	
vp1-Mum1	X	
vp1-Mum3	X	
c2-Mum1	X	
Dap1	X	
Dap2	X	
Dap-py	X	
yg2-Mum (13 independent isolates.)	X	X
lw1-Mum3108	X	
lt0-Mus1359 (When first isolated mutant was stable.)	X	

7. *Mutator*-induced deletions. Deletions have been demonstrated in progenies of *Mutator* plants for the distal portion of both the short arm and the long arm of chromosome 9. However, genetic evidence for deletions in interstitial regions is meager. One has been found involving the *a1 sh2* region of chromosome 3 and a putative deletion linked to *a1* also has been found. These were just found incidental to other studies. A systematic search for such deletions may reveal whether they are a common phenomenon of the *Mutator* systems or not. The following regions would lend themselves to such studies: Chrom. 1 - *an1 bz2*, Chrom. 3 - *a1 sh2*, Chrom. 5 - *pr1 gl8*, Chrom. 9 - *c1 sh1 bz1*.

8. The presence of *Spm* in I.S.U. *Mutator* stocks. Dr. Vicki Chandler's laboratory has reported the presence of *Spm* in active *Mutator* stocks. I have attempted to test several I.S.U. stocks for the presence of *Spm*. A heterozygous *Spm* stock segregating for mutable and stable *c2-m2*, *wx1-m8* kernels was obtained from another laboratory. This stock was supposed to carry all the genes other than *c2-m2* necessary for aleurone color. Stable kernels were selected and the plants from these kernels were crossed to five different purple aleurone *Mutator* stocks, three *a1-Mum2* mutable aleurone stocks, and four different *Mu2* stocks. Unexpected results were obtained in the crosses to the *Mu2* stocks. All of the F1 ears lacked kernels with aleurone pigmentation. Because our *Mu2* stocks are *A1 A1, A2 A2, C2 C2, c1 c1, r1 r1* and the *Spm* was supposed to be homozygous for the aleurone genes, these F1 ears should have all had mottled kernels. However, if the *Spm* stock was segregating for *c1* and/or *r1*, the stable kernels selected for a source of plants used in these tests could have been

stable because they were homozygous for *c1* and/or *r1* and not because they lacked *Spm*. Such kernels would have been classified as lacking *Spm* when in reality they could have carried *Spm*, which would not be detected because they lack a *C1* and/or *a R1* allele. Some F1 plants from all these tests, when backcrossed to plants from stable kernels of the *Spm* stock, did indeed segregate for mutable kernels, indicating the presence of a *Spm*. However, it can not be determined whether this controlling element came from the original *Spm* stock or the *Mutator* stocks. Unfortunately I was not able to repeat these experiments before I had to cease my research. Thus, the *Spm* status of the I.S.U. *Mutator* lines is undetermined as yet and, unless someone is inclined to make the appropriate tests in the future, it may never be known. (Note: I have included this information here so that workers in other laboratories, who were informed about our preliminary observations, suggesting the widespread presence of *Spm* in our stocks, will be aware that the prevalence of *Spm* in I.S.U. stocks is yet to be determined.)

Information on the Iowa State *Mutator* and other stocks from the Robertson laboratory

--Donald S. Roberston

In last year's News Letter, I indicated that I would be sorting through the stocks I had stored at ISU. This task has been completed and there are now two sources of the stocks that were retained, which are now available for distribution to interested researchers. One set of stocks was sent to the Maize Genetics Cooperation Stock Center at the University of Illinois, Urbana, IL. The second set of stocks has been retained at ISU. The Coop stocks are those I thought would be of more general interest to the maize genetic community, while the ISU stocks are those that I thought would be of more interest to Mutator connoisseurs. Some items have been retained in both collections. (Ed. Note: Lists of the two sets of stocks can be requested from the Stock Center or from the editor of MNL; please ask for a hard copy or an electronic copy; seed of these materials will be available only after Stock Center reproduction, i.e., not until 1995). All stocks except those used as planting sources for, or produced in, the years 1989, 1990, and 1991 have been stored in our cold storage facilities. These facilities were not the most reliable and at times the stocks were in less than optimal conditions for periods of a week or two until repairs could be made. Thus, it would be advisable to test a few kernels of each stock for germination before planting them in the field. The older the stocks the higher the likelihood of poor germination.

AMES, IOWA
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JOHNSTON, IOWA
Pioneer Hi-Bred International, Inc.

Analysis of the 5' region of the *P* gene as a potential floral-specific promoter

--Xianggam Li, Laura Tagliani, Bruce Drummond, Ben Bowen and Thomas Peterson

The *p* locus is involved in the synthesis of a phlobaphene-like red pigment found in mature cob glumes and pericarps. The *P* gene encodes a Myb-like transcription factor (Grotewold, Athma and Peterson, PNAS 88:4587) that binds the sequence $CC^{T}/_{A}ACC$ and activates transcription of the *A1* gene, but not the *Bz1* gene (Grotewold et al., Cell, in press). The 1.2 kb region 5' of the *P* transcription start site has partial homology to a *Trypanosoma brucei* tRNA gene, the maize *B-I* promoter, and maize pollen-specific pectate lyase gene 3' region. The *P* promoter region was fused to various reporter genes (Gus, luciferase, and anthocyanin markers) for transient assay experiments via particle gun bombardment. The following preliminary results were obtained: (1) the *P::GUS* reporter fusion was much less active in endosperm suspension culture than either embryogenic cells or BMS non-embryogenic suspension cells; (2) qualitative GUS assays indicated more blue spots in pericarp and cob glumes than aleurone or young shoots; (3) the 5' region of the *P* gene contains a relatively weak promoter since only *P::GUS* or *P::Luciferase* plasmids containing *Adh1* intron 1 gave a detectable signal in transient assays, whereas no signal was obtained using similar constructs lacking the *Adh1* intron 1. Further subcloning and quantitative assays will be used to understand how the *P* gene is expressed specifically in floral tissues, and to determine whether the *P* promoter could be used to direct foreign gene expression in pericarps, glumes, silks and husks.

A new *P-ww* allele and *Ac* element with high negative dosage effect and novel suppressing activity

--Jianbo Zhang and Thomas Peterson

A new *P* gene allele (*P-ww*-12:27-3*) which specifies colorless pericarp and cob glumes was derived in two steps from *P-ovov-1114*. The *P-ww*-12:27-3* allele carries an *Ac* element with a very high negative dosage effect. In crosses to *P-vv* and the *Ac* tester line *r-m3*, both *Ac* transposition in pericarp and *Ds* excision in aleurone occurred very late. Also, *P-ww*-12:27-3* suppresses the orange pericarp pigmentation specified by *P-ovov-1114* (which carries *Ac* inserted in the *P-rr* intron 2) and two other *P* gene alleles carrying *Ds* elements. Thus, *P-ww*-12:27-3* suppresses the expression of *Ac*- and *Ds*-induced alleles, similar to the *Suppressor* function of the *En/Spm* transposon system. Preliminary Southern blot analysis indicates that *P-ww*-12:27-3* has a duplication of part of the *P* gene. Further molecular studies are in progress to determine what molecular changes occurred to generate the novel *Ac* element in *P-ww*-12:27-3* from the standard *Ac* in *P-ovov-1114*.

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Four new tropical lowland downy mildew resistant maize populations

--C. De Leon, G. Granados and R. N. Wedderburn

Four genetically broad based downy mildew resistant (DMR) maize populations have been developed by CIMMYT-Asian Regional Maize Program (ARMP) based in Thailand. These are Pops. 100 (Early White DMR), 145 (Early Yellow DMR), 300 (Late White DMR) and 345 (Late Yellow DMR) adapted to tropical lowlands. Until now, the two white and the two yellow populations have undergone five and four cycles of S1-S2 recurrent selection, respectively.

Populations were developed in 1985 by crossing several selected white and yellow maize cultivars in the early (90-95 days to harvest) and late (110-115 days to harvest) maturity groups.

Table 1. Means of three traits in different cycles of selection in EW-DMR, EY-DMR, LW-DMR, and LY-DMR populations, selected for downy mildew resistance and other agronomic traits.†

Population	Cycle of selection	Grain yield	Days to 50% silking	DM infection	
				Transformed	Raw
		kg ha^{-1}	no.	angle‡	%
EW-DMR	C0	5171	48	50.6	59.7
	C1	5633	49	46.4	53.4
	C2	6350	48	19.8	11.5
	C3	6110	49	29.6	24.4
	Mean	5816	48	36.6	35.5
	b (linear)	354**	—	− 9.0*	—
EY-DMR	C0	4638	48	60.0	75.0
	C1	5195	47	53.7	65.0
	C2	5834	48	21.2	13.1
	C3	6510	48	25.4	18.4
	Mean	5544	48	40.1	41.5
	b (linear)	626**	—	−13.6**	—
LW-DMR	C0	5770	50	53.0	63.8
	C1	5685	51	46.7	53.0
	C2	6100	52	39.4	40.3
	C3	6724	52	24.3	16.9
	Mean	6070	51	40.8	42.7
	b (linear)	324**	0.6**	− 9.4**	—
LY-DMR	C0	5152	50	46.8	53.1
	C1	5183	52	56.8	70.0
	C2	6510	52	39.9	41.1
	C3	7113	52	23.5	15.9
	Mean	5990	52	41.8	44.4
	b (linear)	721**	0.7**	− 8.7**	—
	Mean C0	5183	49	52.6	63.1
	Mean C1	5424	50	50.9	60.2
	Mean C2	6199	50	30.1	25.1
	Mean C3	6615	50	25.7	18.8
	Overall mean	5855	50	39.8	41.0
	b (linear)	507**	0.4**	−10.2**	—
LSD (0.05) between populations		361	1.0	5.2	—
LSD (0.05) between cycles		305	0.6	5.2	—
LSD (0.05) between cycles within populations		610	1.2	10.4	—

*,** Significant at the 0.05 and 0.01 probability levels, respectively.
† Means based on combined analysis of 1990 summer data from the Philippines and Thailand.
‡ Percent DM infection values were transformed to the arcsin of the square root of % DM infection.

Philippine DMR Comp. 1 and Philippine DMR Comp. 2 were used as donors of DMR in the yellow and white populations. The crosses were made in isolated crossing blocks at Suwan Farm, Thailand (14.5°N, 101°E, 360 masl). After random mating twice, a S1-S2 recurrent selection program was initiated. During each cycle of selection, approx. 500 S1 families were generated from 500 bulk pollinated ears at Suwan in the dry winter season under no downy mildew conditions and evaluated in DM nurseries at Suwan and the Univ. Southern Mindanao Agric. Res. Center (USMARC) in the Philippines (7°15'N, 124°50'E, 300 masl) during the early planting season (April-June). Selected plants in the superior 60% of the S1 progenies were self-pollinated at both Suwan and USMARC. Seeds of approx. 500 S2 ears selected at both places were planted again in disease nurseries at these two locations during the late season (July-Oct.). Screening for DM reaction was done during the early and late planting seasons when disease incidence is high. Information on S2 progeny performance in both nurseries was used to bulk pollinate among 60% of the selected progenies at Suwan. Progenies were selected mostly for desirable agronomic characters and disease resistance.

In the summer of 1990, bulks of Cycles 0, C1, C2 and C3 of the four populations were evaluated at Suwan and USMARC under disease-free conditions for grain yield and days to 50% silking. The response for DMR in the four populations was measured by planting bulks of Cycles 0, 1, 2 and 3 in DM nurseries at two locations.

Data showed that grain yields were higher and plant height lower at Suwan than at USMARC. Throughout the tests CV's were low for yield and days to 50% silking, but high for DM infection. Early populations yielded less and flowered earlier than the late ones (Table 1); in all populations C3's yielded higher than the C0's with a highly significant gain across populations. Highly significant decreases in DM infection were recorded with averages of 63.1% for C0 and 18.8% for C3 of all populations with a highly significant progress from selection for DMR averaging -11% per cycle across populations. During the selection cycles, the S1 progenies showed greater DM infection at USMARC than at Suwan and infection values at both locations were highly correlated.

These new broad based DMR populations can be either directly released by national programs, or used as sources of resistance in breeding programs. Breeder seedstocks are available from CIMMYT-ARMP, P.O. Box 9-188, Bangkok 10900, Thailand.

BEIJING, CHINA
Academia Sinica

Origin of Chinese waxy maize (*Zea mays sinensis*)

--Mengqian Zeng and Yannan Liu

In this paper, we state the research results on the morphology, physiology-biochemistry, genetics and origin time of Chinese waxy maize (*Zea mays sinensis*). The main results of the research are summarized as follows:

(1) Chinese waxy maize showed many characters of wild maize. For example, the ear was smaller, there were smaller grains covered partly by the bracts of the spikelet, rachises of the tassel were on the top of the ear, the number of ear rows was less, and each plant possessed many ears and tillers.

(2) The compositions of grain protein and amino acids of Chinese waxy maize were between normal and high lysine maize. The genetic resource of high lysine maize was screened in Chinese waxy maize.

(3) The fifth band of peroxidase isozymes was a marked band of Chinese waxy maize from South China; on the other hand, the fourth band was a marked band of dent maize (including waxy dent) from America. The fourth and fifth band of the peroxidase isozymes were genetically controlled by a locus with codominant alleles. The fourth and fifth band were a pair specific to the green tissues.

(4) According to these observations, it was reasonably believed that Chinese waxy maize actually originated from the tropical and sub-tropical regions of Xishuangbannan of China.

(5) We bred waxy maize hybrids with higher grain yield and high resistance to maize disease. For instance, SASC No. 1 (Science Academy Single cross No. 1) was crossed by excellent waxy maize lines (Shanghai wx and Yinou303 wx). The hybrid has a grain yield of 6750 kg/ha in general and the maximum grain yield will reach more than 8500 kg/ha.

Studies on the superior new germplasms in sweet corn (*Zea mays saccharata*)

--Yannan Liu, Mengqian Zeng and Taolan Yang

During the period from 1985 winter to 1993 autumn, two normal sweet corn lines (Yitain No. 83 and Yitain No. 33) and one super sweet corn line (Yitain dwarf No. 1) were developed. These lines have been tested and used in hybrid production as new germplasm resources. Three lines are maintained by and are available from the Institute of Genetics, Academia Sinica.

(1) Yitain No. 83 and Yitain No. 33: These lines were developed by continued inbreeding and selection from the sweet corn varieties A and B. They impart high general and specific combining ability. Yield of young ears of the cross combination Yitain No. 83 x Yitain No. 33 and Yitain No. 83 x Yitain No. 185 (baby corn) were high and stable, and were 24.0% and 35.9% higher than the control, normal sweet corn No. 8701 (ck), respectively. The two lines are also fairly resistant to *Helminthosporium turcicum, H. maydis, Fusarium graminearum,* and *Diplodia maydis* and *D. macrospora*. The degree of resistance to *H. turcicum* is 1, indicating polygenes or horizontal resistance. They are mid-late in maturity, mid-tall in plant and ear height. Stalks are fair, roots are vigorous, leaves are semi-erect and mid-sized. They usually have two or three cylindrical young ears per plant and produce yellow kernels on white cobs.

(2) Yitain dwarf No. 1: We bred this line by using continued inbreeding and dwarf selection from improvement population I *sh2* which was bred by recurrent selection. Yitain dwarf No. 1 has good general and specific combining ability. The yield of the cross combination Yitain dwarf No. 1 x Yitain No. 20m is high, and is higher than the control (ck).

Yitain dwarf No. 1 showed a wide spectrum of resistance and ideal plant morphology. The degree of resistance is 1, which indicates polygenes or horizontal resistance. It is medium in maturity and medium in plant and ear height. Stalk and root qualities are good. The percentage of double ears per plant is high.

The lines have different responses to different types of cms (Table 1).

Table 1. Fertile reaction of three lines to different cms types.

		Cytoplasm	
Line	T	S	C
Yitain No. 83	-	-	+
Yitain No. 33	-	+	-
Yitain dwarf No. 1	-	-	-

Notes: +, full restoration; -, full maintenance.

To sum up, three inbred lines were developed by different methods from various sweet corn materials. Though each possessed its own characters and specific properties, all showed good disease resistance, superior agronomic characters and high combining ability. The lines have different responses to different types of cms. The breeding of the three inbred lines has enriched the maize germplasm in our country.

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Role of the transcriptional regulator *opaque2* in carbon partitioning between starch and proteins in the sink

--M. Maddaloni, G. Donini, F. Forlani, L. Stasse and M. Motto

The endosperm is considered the primary sink for carbon (C) and nitrogen (N) assimilates in maize. Starch and zein protein are major storage components in the endosperm sink that affect grain yield and nutritional quality. The synthesis of these two storage components requires sucrose and amino acids, which are provided by vegetative tissues. Previous studies indicate that the relative level of C and N in kernels may vary among maize hybrids. Moreover, evidence has suggested that the amount and the relative proportion of starch and proteins in the endosperm, i.e. the C/N ratio, are determined by the nutrient supply, the sink demand and the interaction between them (Balconi et al., Plant Sci. 73:1-9, 1991). The coordinate regulation of C and N supply is a subject of great interest because it is responsible for the accumulation of starch, protein and corresponding increases in dry weight of the kernel. However, the biochemical and physiological background of this relationship is complex and not fully understood.

The maize *o2* locus, which is known by classical genetic and molecular studies to activate the 22 kD zein and b-32 genes in trans, encodes a protein which belongs to the basic/leucine zipper (bZIP) class of transcription factors (Lohmer et al., EMBO J. 10:617-624, 1991). We have recently shown that in transformed yeast cells the O2 protein could substitute for GCN4 protein, a yeast transcriptional activator of amino acids biosynthetic genes which are subjected to general amino acid control. It is conceivable that *o2* may play a similar role in maize endosperm, namely that it

regulates amino acids biosynthetic genes and/or genes involved in C partitioning between proteins and starch.

The gene encoding the cytosolic form of pyruvate orthophosphate dikinase (PPDK:EC2.7.9.1), which catalyzes the conversion of pyruvate, ATP and Pi to PEP, AMP, and PPi. Three genes have been described in maize by Sheen (Plant Cell 3:225-245, 1991): one encoding the C4, chloroplastic PPDK (C4PPDK) and two encoding cytosolic PPDK activities, cyPPDK1 and cyPPDK2, the last being poorly expressed in all tissues examined.

An assay for transient gene expression in tobacco protoplast has been employed to investigate the possible activation of two different PPDK promoters by the *O2* product. The assay was based on cotransfection of tobacco mesophyll protoplasts with an expression and a reporter plasmid. The expression plasmid, pCaMVO2, consisted of the full length *O2* cDNA placed as a transcriptional fusion under the control of the 35S gene promoter from CaMV. The reporter plasmids consisted of different PPDK gene promoters fused to the coding region of chloramphenicol acetyltransferase (CAT) gene. These constructs were generously provided by J. Sheen, Massachusetts General Hospital.

The results of the transient expression experiments showed that the promoter of the C4PPDK gene is not transactivated by *O2*, while the promoter of cyPPDK1 was strongly activated by *O2*, suggesting that cytosolic PPDK1 expression is under the control of *O2*.

Such observation is congruent with the hypothesis that *O2* may be active in the diversion of C flux from sugars to amino acids, because PEP is generally believed to play a crucial role in amino acid biosynthesis. In fact, PEP can be carboxylated by cytosolic PEPcase to generate oxaloacetate, which is easily transformed into aspartate. This molecule is in turn the first compound for the synthesis of branched amino acids. PEP itself is also the first compound common to the aromatic amino acids biosynthetic pathway. Experiments are in press to assess the real impact of this diversion route in the in vivo partitioning of photosynthates.

RFLP mapping of QTLs for grain yield and agronomic traits

--P. Ajmone Marsan, G. Monfredini, W. Ludwig, A. E. Melchinger, G. Pagnotto and M. Motto

The use of RFLP markers has proven useful in the rapid construction of detailed linkage maps in several crop species and made possible the dissection of quantitative traits into Mendelian factors. The identification and examination of individual quantitative genes should provide information about the organization of genomes and insight into the relative contributions of quantitative genes to continuous variation. In this respect, genetic markers linked to factors associated with metric traits have been advanced in the literature to study quantitative inheritance.

The objective of this research was to identify RFLP loci associated with QTLs affecting expression of yield and other agronomic traits in a cross between two maize inbred lines, B73 and A7. From this cross 294 F3 lines were developed through two selfing generations with each F3 line tracing back to a different F2 plant. In the 1990 breeding nursery at Bergamo, Italy, two sets of testcrosses for each of the 294 F3 lines were produced: one with the tester inbred line A1, and the other with Mo17.

For each kind of testcross, the materials were subdivided into 3 sets and evaluated in simple 10x10 lattices at two locations, Bergamo and Brescia, Italy, in 1992. A total of 75 genomic maize clones were selected from collections of mapped clones available from Brookhaven National Laboratory and the University of Missouri to provide a uniform coverage of the genome.

Mapping of QTLs and estimation of their genetic effects were performed according to the method of interval mapping described by Lander and Botstein (Genetics 121:185-199, 1989) using the computer package MAPMAKER/QTL (Lincoln and Lander, Whitehead Inst. for Biomed. Res., Tech. Rep., Cambridge, MA, 1990). Presence of a putative QTL in a given genomic region was declared when the LOD scores of the additive model exceeded 2.5, corresponding to a probability $P<0.05$ that a false positive occurs somewhere in the genome. The total variation accounted by significant QTLs and the total LOD score were obtained by fitting a model including all putative QTLs for the respective trait simultaneously.

For grain yield in the testcross to Mo17, the long arm of chromosome 4 and the short arm of chromosome 6 showed highly significant effects with LOD scores of 3.2 and 6.1, respectively. In the testcross to A1, the short arm of chromosome 6 and the long arm of chromosomes 9 and 10 showed highly significant effect with LOD scores of 2.9, 5.4, and 2.9, respectively. Thus, the testcross to Mo17 showed at least two QTLs which collectively accounted for 21.7% of the variation for grain yield, while the backcross to A1 showed at least three QTLs which collectively accounted for 25.2% of the variation for grain yield. In the combined analyses for means over testcrosses, three genomic regions located on 4L, 6S, and 10L were found to significantly affect grain yield. LOD scores at peaks of QTL likelihood maps ranged from 2.6 to 7.4 for the genomic regions on 4L and 6S, respectively. The multiple QTL model indicated that these QTL, collectively, accounted for 35.4% of the variation for grain yield. It was interesting to note that the QTL on the short arm of chromosome 6 (LOD 7.4) accounted for 24.5% of the total phenotypic variation.

A total of three QTLs influencing grain dry matter content were detected. Analysis of Mo17 testcross data revealed two factors on chromosomes 1 and 2 with LOD scores of 4.8 and 4.9, respectively. The two loci together accounted for 22.7% of the phenotypic variance. Analysis of A1 testcross data confirmed the presence of the QTL on chromosome 2 between *umc134* and *umc131* markers, and suggested a second locus on chromosome 8. Collectively they accounted for 10.7% of the phenotypic variation for grain dry matter. The loci on chromosome 1 and 2 influencing grain dry matter content were confirmed in the combined analysis across testers. LOD scores at peak of QTL likelihood maps were 3.6 and 8.9, respectively. In total, 26.4% of the phenotypic variance was explained by the two QTLs.

Only a single QTL influencing plant height was detected in the testcross to Mo17. This QTL mapped on chromosome 3 and had a LOD score exceeding 2.7, which accounted for 16% of the phenotypic variation for plant height. In the A1 testcross, chromosomes 3, 5, 9, and 10 showed LOD scores exceeding 2.7, which altogether accounted for 28.8% of the phenotypic variation. QTLs found on chromosome 3 in the two testcrosses had extremely large, and overlapping, support intervals (>50 cM). Hence they were considered as identical loci even if they map between different flanking markers. It was also evident that for the QTL on chromosome 3 the B73 allele performed better than the A7 allele; the reverse was true for all the other QTLs detected.

Most QTLs found for the traits evaluated in our study were consistent across locations, although variations were observed in

the LOD score levels, indicating that expression of genes controlling these traits was mainly independent of the environments. Only QTLs with larger effect were consistent across testcrosses suggesting that genetic background may contribute to the identification of the QTLs in a specific fashion. It is conceivable that data averaged over more than one testcross should be used for QTL identification. Obviously, further experiments will be required before sufficient evidence is available to verify this effect.

Loci for grain yield found on chromosomes 9 and 10 in our study are likely to have overlapping support intervals with QTLs for grain yield found in the cross B73xMo17 by Stuber et al. (Genetics 132;823-839, 1992). Moreover, all the loci for plant height found in our study mapped in chromosomal regions where the previous authors have found QTLs for plant height, although only the QTLs located on chromosome 3 have overlapping confidence intervals. A further observation which originates from our data is that on chromosomes 9 and 10, the likelihood peaks for the putative QTLs for plant height and grain yield fell in the same marker intervals. The direction of the effects of allele substitution was also consistent. The A7 allele increased both plant height and grain yield, suggesting evidence of an interrelationship of the genes regulating the two traits in this genomic region. The phenomenon of significant association of molecular markers with more than one trait has also been observed by others.

In conclusion, although further investigations will be required to establish the consistency of the detected effects in other genetic backgrounds, our results demonstrated the value of this type of investigation for identifying and localizing genetic factors (QTLs or specific genomic regions). This approach should be useful for marker-facilitated improvement programs, including intrapopulation selection or transfer of desired factors to other germplasms. Research involving facilitated breeding approaches is currently being addressed in our laboratory.

Effect of sucrose and asparagine on the synthesis of storage products in in vitro grown maize endosperms

--D. Bosio, C. Balconi, E. Rizzi, L. Nembrini, A. Morselli and M. Motto

Regulation of nitrogen supply to the developing maize kernel is a subject of great interest since it is responsible for the accumulation of starch protein and corresponding increases in dry weight of the kernel. In addition, the synthesis of storage protein and the potential for cell and/or starch granule formation have been associated, through correlative studies, to the control of starch synthesis. Therefore, understanding the relative importance for these factors and how they interact in controlling endosperm growth could be useful in developing strategies for improving maize productivity.

The aim of this research was to examine the effect of C and N supply on growth, starch, and protein composition of maize endosperms. In vitro culture of maize endosperms, on well defined media, offers a convenient opportunity to study various factors affecting kernel growth and endosperm starch and protein synthesis; this will avoid the complex relationship between the growing seed and the mother plant. Immature endosperms of 26 maize inbred lines (Table 1), differing in starch and protein content in the grain, were collected at 9 days after pollination (DAP) and grown for five days on solid media containing different sucrose to asparagine ratios (Table 2).

Table 1. Inbred lines with high (HP), medium (+) and low (LP) protein content as percent (%) of dry matter, in the mature seed.

No.	Inbred line	% of proteins	LP<10.5%	HP>13
	IHP	25.50		HP
1	Lo5	14.85		HP
2	A69Y	14.50		HP
3	W25	14.42		HP
4	38-11	13.52		HP
5	Lo881	13.48		HP
6	W64A	13.41		HP
7	B14A	13.11		HP
8	Pa83	13.05		HP
9	B14	13.02		HP
10	R193	12.61	+	+
11	A637	12.55	+	+
12	Lo863	12.18	+	+
13	B37	11.52	+	+
14	A632	10.61	+	+
15	Lo1066	10.40	LP	
16	N7B	9.39	LP	
17	Lo1069	9.37	LP	
18	Lo964	8.93	LP	
19	Lo904	8.92	LP	
20	Ms213	8.83	LP	
21	ND385	8.78	LP	
22	N28	8.75	LP	
23	Lo1016	8.41	LP	
24	K44W	8.24	LP	
25	R227	8.10	LP	
26	B84	7.61	LP	
	ILP	3.90	LP	

Table 2. Culture media. All media contained 0.4 mg/l thiamine, 100 mg/l inositol, salts as described in Nitsch and Nitsch and 8 g/l agar.

Medium	Sucrose (g/l)	Asparagine (g/l)
1	10	0.02
2	10	4
3	30	0.02
4	30	4

For all the inbred lines tested, it was evident that increased dry weight accumulation by cultivated endosperms and increased sucrose and asparagine concentrations in the media were positively correlated. In addition, from this study it was possible to identify groups of lines differing in the trend of total nitrogen and starch accumulation in the endosperm, during in vitro culture. The data suggest that for some inbred lines the control of synthesis of endosperm proteins and of starch was, at least in part, at the source level rather than at the sink level.

The antagonism between starch and N accumulation, observed in some inbred lines, could be explained by the fact that the highest amount of asparagine supplied was greater than that required for maximum N content in the endosperm; in these conditions the efficiency of N use declined and less starch was deposited.

Our data suggest that, in the maize endosperm, starch and protein accumulation were interdependent and were controlled by carbon and nitrogen nutrient supply. However, a large variability among the inbred lines in the trend of response to the nutrients was evident.

Conditions for electroporation of intact type II maize calli

--E. Lupotto, P. A. Della Torre, M. Albano and G. M. Borrelli

During our studies on maize tissue culture, particular attention was focused on the development of embryogenic regenerable friable type II callus from elite maize genotypes and crosses of Lo inbred lines produced by the Section of Bergamo (Locatelli et al., MNL 66:17-18, 1992). The work was developed with the aim of evaluating the in vitro culturability of the Lo inbred lines, and establishing cultures for direct genetic transformation via

electroporation in callus tissues as described by D'Halluin and coworkers (Plant Cell 4:1495-1505). In that paper, stable genetic transformation of maize was afforded by direct introduction of DNA into callus and immature zygotic embryos via electroporation. The advantage of that work, besides the importance of having established a new and easy tool for transformation, resides in the fact that DNA transfer can directly be applied to zygotic embryos after explant, or to type I primary calli, which can easily be induced in several maize genotypes (Hodges et al., Bio/Technol. 4:219-223, 1986).

On the other hand, we observed that the constant availability of immature embryos throughout the year needs a time consuming and sometimes problematic continuous breeding of donor plants in greenhouse or phytotron. Often, when greenhouse conditions can not be controlled perfectly, it is difficult to establish the optimal "physiological age" of the embryos to be explanted. Therefore genotypes that respond perfectly when evaluated from field conditions, do not respond as well when evaluated using embryos obtained from greenhouse grown plants. Furthermore, type I calli are easy to establish but difficult to maintain in culture for long periods, and their regenerative capability declines in a few months. A long term effort for the introduction of genes in maize would then be favored by a continuous supply of regenerable cultures, needing to be established only once a year. In this respect, we have chosen to direct our efforts to the establishment of optimal conditions for electroporation of selected embryogenic lines obtained from crosses of Lo inbreds with A188. Some selected lines behave as expected in that: i) they are friable, highly embryogenic and fast growing; ii) they are started yearly from immature embryos of the summer nursery; iii) they are easily maintained in propagation on N6P medium in the dark under standard routine conditions (as described in Lupotto and Lusardi, Maydica 33:163-177, 1988); iv) they are promptly regenerated after plating of calli in the light directly onto MS hormone-free medium, or after a 10 day culture on MS medium supplemented with 5 mg/l zeatin, in the light, and with subsequent transfer to hormone free conditions.

Besides maize we have established a variety of embryogenic callus cultures of cereal species: sorghum, bread wheat and durum wheat. Our objective is to develop a protocol of transformation in callus cultures via electroporation, amenable to utilization for gene delivery into the various species with minor modifications.

We have established a routine procedure for electroporation of maize type II calli by using a Bio-Rad Gene Pulser with capacitance extender, discharging one electrical pulse per sample with a field strength of 375-400 $V \cdot cm^{-1}$ from a 960 uF capacitor. The callus used in each electroporation was about 80-150 mg fresh weight tissue chosen from the upper part of calli at the mid growth stage during subculture on N6P medium in the dark. In these conditions the tissue which is electroporated is mainly formed of globular shaped somatic embryos. Before electroporation, callus tissue is plasmolized for 2 hours at 24 C in 800 ul 0.4 M mannitol, 10 mM $CaCl_2 \cdot H_2O$, 10 mM MES, pH 7.2, with subsequent incubation 10 min on ice. After electroporation and a further incubation on ice for 10-15 minutes, callus pieces are transferred onto the surface of 0.6% agarose gelled N6P medium supplemented with 0.2 M mannitol, air dried for 30 minutes at the flow hood and incubated thereafter in the dark for subsequent growth. Various types of electrolytes can be utilized in the electroporation buffer such as KCl, NaCl, K-glutamate and Na-glutamate, in accordance with Songstad et al. (Plant Cell Tissue Organ Cult. 33:195-201, 1993). The duration of the electrical pulse, measured as time constant (TC) in msec, varies according to the electrolyte and its concentration, with stable inoculum size of callus tissue (Fig. 1). We could detect GUS expression in histochemically stained calli 48 h after electroporation in a range of values of TC, from about 70 msec, obtained with 80 mM KCl or NaCl, up to 300 msec, with 5-10 mM KCl or NaCl. Values of TC are slightly higher when Na-glutamate and K-glutamate are employed. Since our studies are focused on the stable introduction of genes into maize, a major requirement in the transformation procedure is represented by the ease and efficiency of regeneration of the electroporated tissues. We have noticed that when Na-glutamate and K-glutamate are utilized as electrolytes, independently from the concentrations, a strong decrease in the callus regenerative capability was observed. Furthermore, callus growth and somatic embryogenesis was also negatively influenced when calli were let grow on N6P medium. The highest regenerative efficiency was monitored when 10-20 mM NaCl was used for electroporation, and for this reason we

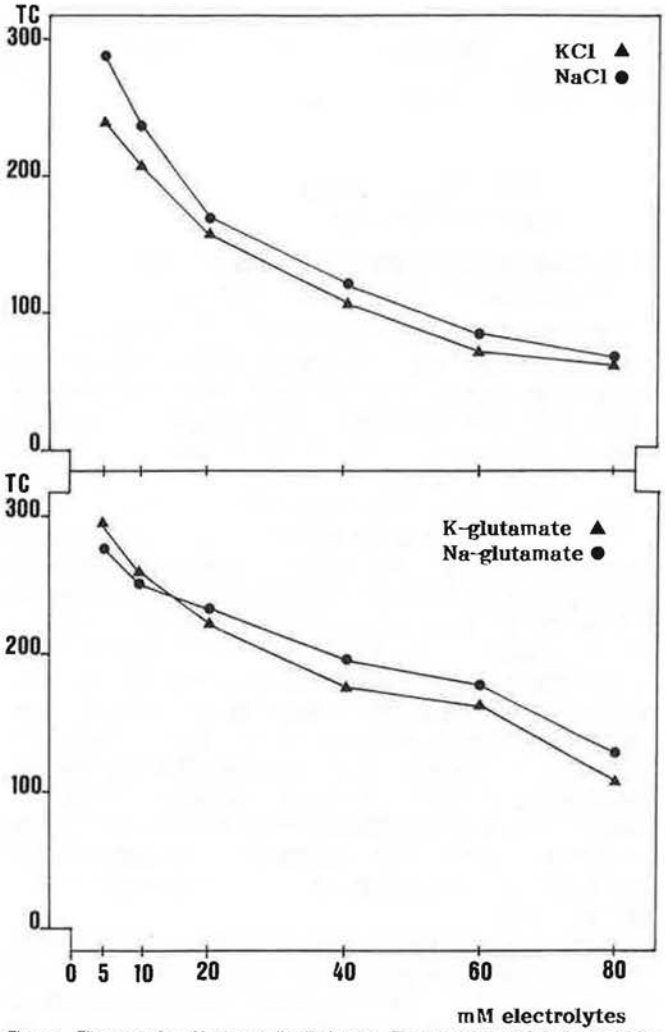

Figure 1. Electroporation of intact type II calli of maize. Electroporation was in buffer containing two types of inorganic electrolytes (NaCl and KCl) and two types of organic electrolytes (Na-glutamate and K-glutamate) at various concentrations from 5 to 80 mM. TC is expressed in msec and indicates time constant for a pulse discharged with 375 $V \cdot cm^{-1}$ from 960 µF capacitor.

currently utilize such conditions for stable transformation of maize. By using this procedure we have obtained stable transgenic maize callus lines containing marker genes, and work is in progress for plant regeneration and genetic analyses.

BERKELEY, CALIFORNIA
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The *lg3* locus maps to the short arm of chromosome 3

--Yong Chi, John Fowler and Michael Freeling

Although *lg3* is usually represented on the genetic map of chromosome 3 as being on the long arm of the chromosome, no definitive placement on either side of the centromere has previously been possible. Some data (R. S. Poethig, MNL 62:99, 1988) suggested that *lg3* is on the short arm of chromosome 3. We have used a method described first by Weber and Helentjaris (Genetics 121:583-90, 1989) to locate *lg3* to chromosome 3S.

Essentially, a DNA probe corresponding to the *lg3* locus (Fowler and Freeling, unpublished) was used to determine the copy number of the *lg3* locus in plants that were either hypoploid or hyperploid for various segments of chromosome 3. Genomic DNA from these plants was digested with various enzymes to give distinct RFLPs for both alleles that were potentially present, Southern blotted, and probed. If the probe corresponded to a locus that was located on a chromosomal segment for which a particular plant was hypoploid, only one band (DNA fragment) appeared; otherwise, two distinct bands were present. In the converse situation, a probe corresponding to a locus on a segment that was hyperploid in a plant yielded two bands, with the RFLP allele present in two copies producing a band twice as intense in signal as the other.

Plants hypoploid and hyperploid for the B-A translocation TB-3Sb were generated by crossing the translocation stock as a male to an *h1 v19/ h1* + stock. Hypoploid embryos expressed the *h1* (starchy) phenotype, and produced plants hyperploid for the translocated segment of 3S. Plants hypoploid for the same segment in the population were recognized by the expression of the *v19* (virescent) phenotype. Testers homozygous for *ys3 a1*; *R-scm* were used as females in crosses with a TB3-La translocation stock; colorless endosperm/colored scutellum kernels produced 3L hyperploids and *ys3* (yellow stripe) plants were hypoploid for the same segment of 3L.

A chromosome stock provided by K. L. Rose and R. W. Staub (Carleton College) was used to generate plants hypoploid for either the entire 3S or 3L chromosome arm. This stock resulted from the centric fission of chromosome 3, followed by recovery of two stable telocentric chromosomes corresponding to arms 3S and 3L. In the presence of B chromosomes, at a low frequency, one or the other of the telosomes is not transmitted to the zygote through the pollen, perhaps due to non-disjunction at the second microspore division (K.L. Rose and R.W. Staub, personal communication). This results in a plant hypoploid for the entire length of either chromosome arm. A plant carrying both telocentric 3 chromosomes was crossed as a male to several female testers, which were either heterozygous for the *v19* marker on 3S or homozygous for the *ys3* marker on 3L. One plant out of 90 in a population derived from the *v19* tester cross expressed the v19 phenotype and was classified as a 3S hypoploid. One plant out of 37 in a population derived from the *ys3* tester expressed the ys3 phenotype and was classified as a 3L hypoploid.

DNA from one of each of these types of plants (hypo- and hyperploid for TB-3Sb, hypo- and hyperploid for TB-3La, hypoploid for the entire 3S arm, and hypoploid for the entire 3L arm) was subjected to Southern analysis. When probed with the *lg3* probe, all of the plants except the entire 3S hypoploid exhibited two bands, corresponding to the two alleles of *lg3* present in each plant. The 3S hypoploid exhibited only one band, indicating only one copy of *lg3* in this plant. These data indicate that *lg3* is on the short arm of 3S, proximal to the TB-3Sb translocation breakpoint. The same filters were stripped and reprobed with both the *umc92* RFLP probe (located on 3S distal to the TB-3Sb breakpoint) and the *a1* gene probe (located on 3L distal to the TB-3La breakpoint) to confirm the chromosomal constitutions of these plants. In all cases, the predicted number and intensity of bands for each DNA sample was observed (i.e., *umc92* picked up two bands in each of the plants aneuploid for segments of 3L, one band in the entire 3S and TB-3Sb hypoploids, and two bands, one twice as intense as the other, in the TB-3Sb hyperploid).

The pleiotropic mutation *dek*-Mu1364* maps to chromosome arm 9L

--Mike Scanlon, Mi Chang and Michael Freeling

The recessive mutation *dek*-Mu1364* was generated from *Mutator* stocks and causes numerous developmental aberrations: small endosperm, small embryo, 50% germination rate among homozygous kernels, brachytic plants, drooping leaves exhibiting ligule disruption over the midrib, and male sterility (Scanlon et al., Genetics, in press).

Subsequent analyses indicated the presence of both ectopic ligule and displaced ligule along the midrib of all leaves in *dek*-Mu1364* homozygous plants. Transverse sections of leaves were treated with phloroglucinol (stains lignin) to demonstrate that the ligular region of *dek*-Mu1364* homozygotes is markedly delignified as compared to wild type siblings. The reduction in lignin accumulation in leaves of *dek*-Mu1364* homozygotes explains the drooping leaf phenotype observed in these plants. Histological analyses of the vegetative shoot apex of 14 day-old mutant seedlings have revealed that the meristem of *dek*-Mu1364* homozygotes is abnormally short and flattened.

Plants heterozygous for the *dek*-Mu1364* mutation were crossed to TB-9Lc and several ears were identified which segregated kernels with small endosperm and large embryos, and plump endosperm and small embryos. The discordant kernel classes were planted in the greenhouse and those with small endosperm and large embryos (putative hyperploid embryos) produced normal seedlings whereas the kernels with plump endosperm and small embryos (putative hypoploid embryos) yielded small brachytic plants with ligule disruption. These data indicate that *dek*-Mu1364* is located on the long arm of chromosome 9.

The *lxm1* gene maps near position 88 on 3L

--Denise Schichnes, Claudine Woo and Michael Freeling

The gene *lxm1* is identified by a single, EMS induced dominant mutant allele. *lxm1* was originally described and mapped to chromosome 3 by M. G. Neuffer (MNL 62:53). *lxm1* shows linkage to *lg2* on chromosome 3L.

Parentals		Recombinants	
lg2	*Lxm1*	*Lxm1:lg2*	W.T.
80	42	7	5

Based on these data, *lxm* is 8.3 map units from *lg2*.

Using RFLP probes for 3L, we determined the location of *lxm1* at approximately position 88 on chromosome 3.

Probe	Position	# crossovers	Total observed	Map units
bnl5.37	81	12	183	6.6
bnl8.01	92	6	140	4.3
bnl10.24	93	2	50	4.0

BOMBAY, INDIA
Bhabha Atomic Research Centre

Non-Mendelian breeding behaviour of *sh1-B* (*shrunken1-Bombay*) allele

--S. Nadiger and N. K. Notani

We have reported that upon self-pollination, plants carrying *sh1-B* allele yield kernels with varying 'shrunkenness'. Upon testcrossing to *sh1-A* (American tester stock), all the kernels are completely shrunken (Allagikar et al., J. Genet. 70:33-41, 1991). Self-pollination of *sh1-A/sh1-B* hybrids yields progeny kernels that are, with few exceptions (~1%), completely shrunken (MNL 67:21, 1993). This suggests that the 'silenced' *sh1-B* allele continues to remain repressed in the following generation. Non-shrunken kernels, when testcrossed to *sh1-A*, are no longer inhibited. We interpret these observations as follows:

1. There are alleles in maize, the expression of which is variable (metastable?). The molecular basis for this is not clear.
2. Hybrids of *sh1-A/sh1-B* have completely shrunken kernels indicating that the *sh1-B* allele has been 'silenced'.
3. The 'silenced' allele *sh1-B* continues to remain so in the next generation following selfing of *sh1-A/sh1-B* plants.
4. A few non-shrunken kernels in the progeny of *sh1-A/sh1-B*, when testcrossed to *sh1-A*, remain non-shrunken, presumably having become refractory.

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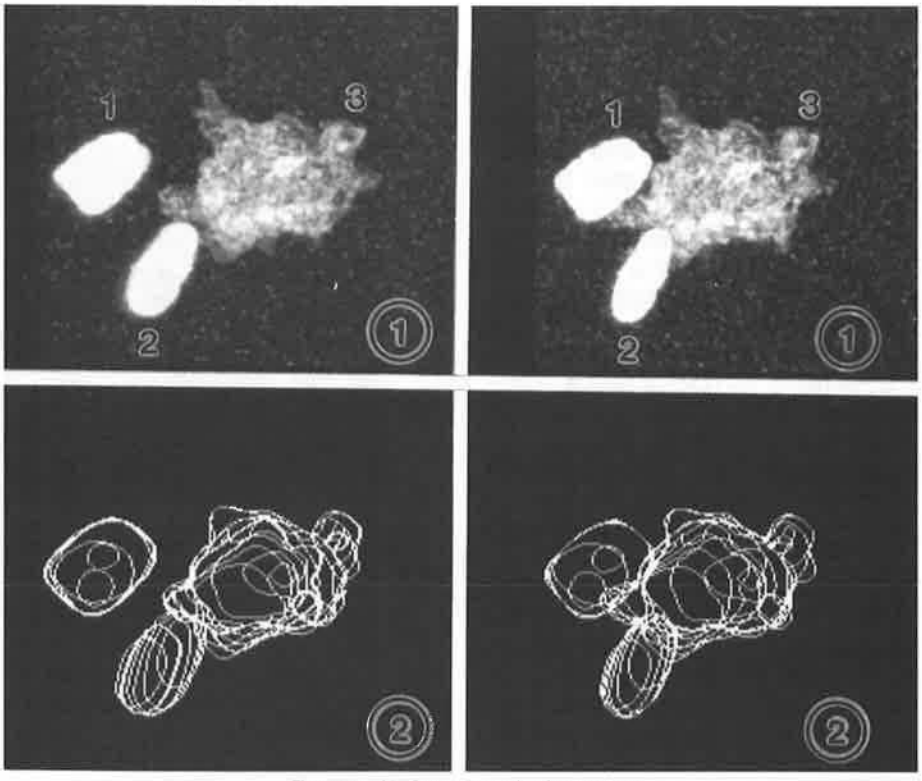
State University of New York

Determining the nuclear volume in a pollen grain by using laser scanning confocal microscopy and multi-dimensional image analysis

--Ping-chin Cheng and J. K. Samarabandu

We have developed an automatic image processing tool for determining the volume of vegetative and sperm nuclei in a maize pollen grain. The procedure involved collection of confocal fluorescence images from a DAPI-stained/Feulgen-stained pollen grain at a 0.5 μm (or 1 μm) sectioning interval and processing the 3D image data with an automatic image processing system developed at our laboratory. To prepare for confocal microscopy, the stained pollen was dehydrated in EtOH, and cleared in methyl salicylate (Cheng et al., in Multi-dimensional Microscopy, P. C. Cheng, et al., eds., Springer-Verlag, New York, pp. 339-380, 1993).

An attempt at manually contouring the cell nucleus in a sea urchin embryo in 3D was reported by Summers et al. (J. Electron Micro. Tech. 18:24-30, 1990). Apart from being labor intensive, this 3D digitization technique suffers from the inaccuracies of manual 3D tracing related to the depth perception of the operator (Cheng et al., in Visualization in Multi-Slice Imaging Microscopies, A. Kriete, ed., VCH-Publ., Weinheim, pp. 361-398, 1993b). However, it does demonstrate that reducing a stack of confocal images to a 3D graphic representation helps to visualize and analyze complex tissue. This procedure also significantly reduces the computational burden in an interactive operation. These



Figures 1 and 2. Stereogram and surface contours.

image analysis tools can also be employed for numerical and volumetric study of cell nuclei.

To overcome the disadvantage of manual tracing, an automatic data reduction procedure based on multi-dimensional image analysis algorithms was developed in our laboratory. We also developed a system to visualize and extract morphometrical parameters from the data generated by this method (Cheng et al., 1993; Samarabandu, in Multi-dimensional Microscopy, P. C. Cheng et al., eds., Springer-Verlag, New York, pp. 231-250, 1993). Our confocal image processing system is implemented as a set of tools whose activities are coordinated by a blackboard control structure and is modeled after the image understanding model introduced by Kanade (Comp. Graph. Imag. Proc. 13:279-297, 1980).

Figure 1 is a stereogram of a vegetative and two sperm nuclei stained by Feulgen reaction. The image was obtained by optical sectioning of the pollen grain at 1 μm intervals with an Olympus GB-200 laser scanning confocal microscope. The 514nm green line of a 25mW Ar ion laser was used as the excitation wavelength, and red fluorescence was recorded. Note the two intensely stained sperm nuclei and a highly convoluted vegetative nucleus. Figure 2 shows the surface contours of the nuclei generated by our automatic image processing program. The program, at the present time, does show some difficulty in contouring weakly stained fine projections of the vegetative nucleus. With the contour generated, the volume of the nuclei can be calculated. In this case, the volumes of the two sperm nuclei are (1) 249 μm^3 and (2) 209 μm^3; the vegetative nucleus (3) is 973 μm^3.

CEDAR FALLS, IOWA
The University of Northern Iowa

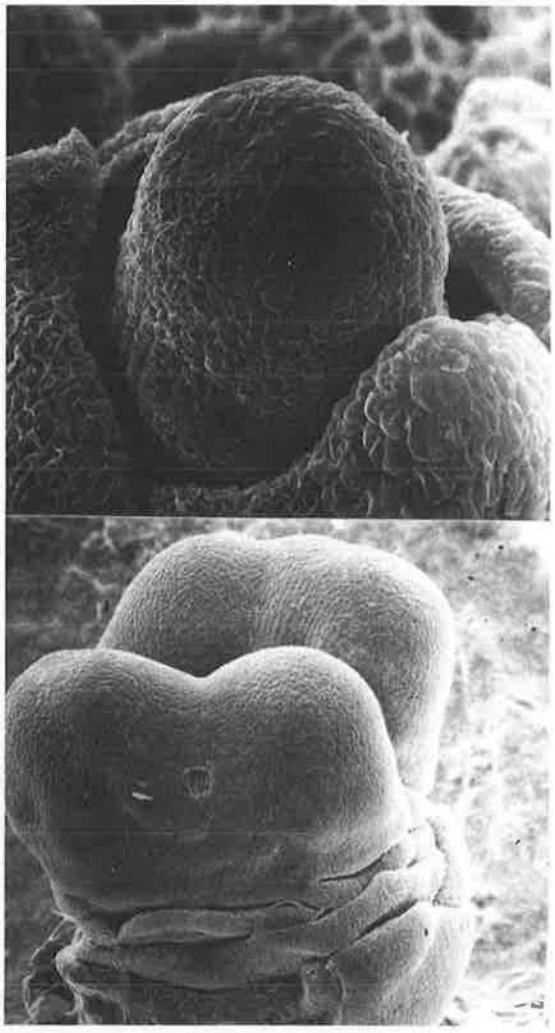
Organogenesis of the malze mutant *Fascicled ear* (*Fas*)

--Gretchen Haas and Alan Orr

Fas is one of the earliest acting mutants on the development of the inflorescence axis of maize. The pronounced, normal elongation of the apical meristem that occurs upon transition from the vegetative state to the reproductive state is disrupted by *Fas* (Postlethwait and Nelson, MNL 64:81-82, 1990). Key abnormal organogenic features include a shift in the direction of transition meristem growth (width doubles its height) and a bifurcation of the broad transition meristem into two primary inflorescence axes, each with a terminal apical meristem. This bifurcation of each terminal apical meristem may be repeated several times in the ear and central spike of the tassel before the process ceases. Since the appearance of terminal apical meristems of *Fas* inflorescences is similar to the spikelet pair (branch) primordia before the formation of pedicellate and sessile spikelet pairs, it was suggested by Hessler (1963) and Postlethwait and Nelson (1990) that *Fas* "time-shifts" the bifurcation event to an earlier ontogenetic stage.

There is no previous SEM organogenic study of this mutant, although an examination of Ruth Hessler's dissertation reveals a histological description of *Fas* ear and tassel development. We undertook this investigation to establish a morphological series of development stages for sampling stage-associated proteins in *Fas* inflorescences. This permits us to test, at the molecular level, the 'time-shift hypothesis': whether a subset of putative protein markers of normal spikelet pair primordia (Coffe, Findlay, Wagner and Orr, Int. J. Plant Sci. 153:31-39, 1992) is found in the terminal meristem of mutant inflorescences.

An SEM examination of more than 40 developing ears and tassels of *Fascicled ear* confirmed that, in the majority of cases, mutant organogenesis followed the pattern described in Hessler's histological studies. Figure 1 illustrates an ear inflorescence whose apical meristem is at the transition stage. This enlarged width stage is followed by a bifurcation of the apical meristem. Figure 2 shows an ear where a second bifurcation cycle has been initiated. Initiation and early development of spikelet pair, spikelet, and floret primordia on each derived *Fas* inflorescence axis is essentially the same as expected for normal maize ear and tassel development, except the inner surface of each axis is often barren of primordia.



Figures 1-2. Early ear organogenesis. 1. Ear at transition stage of development. The specimen shows a broad shoot apical meristem prior to the first bifurcation at the distal tip. 2. A second bifurcation of each new inflorescence axis is shown.

However, based on these SEM studies of ear and tassel development in *Fas* we found additional alterations to the normal sequence of inflorescence organogenesis. In several instances 1-2 additional ranks of spikelets were produced along the clustered, bifurcated ear axes (Fig. 3) and the bifurcated central spike of the tassel. Figure 3 illustrates, at the base of the photo, the usual paired arrangement of pedicellate and sessile spikelets (each with a lower floret initial); note the additional spikelets at the two

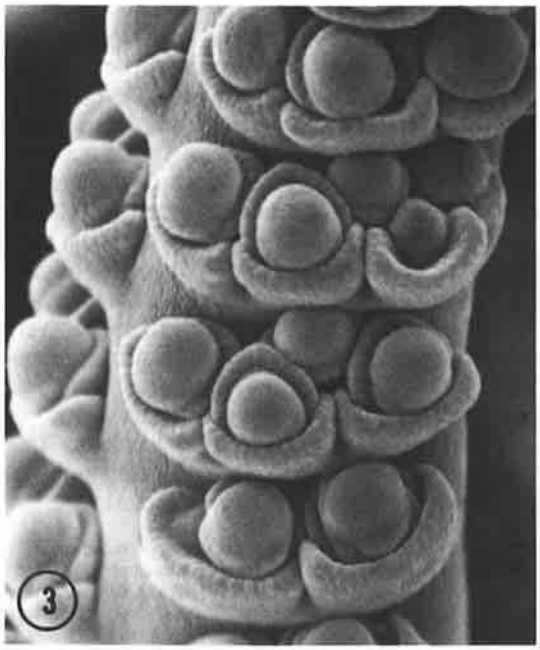


Figure 3. Single inflorescence axis from a *Fascicled ear* cluster of female inflorescence after several bifurcation cycles. Inflorescence is characterized by extra spikelets at the three upper nodes. The lower node shows paired spikelets with glumes, lemmas and lower floret primordia.

nodes proximal to the lower paired spikelets. In these cases the extra spikelets were smaller and at an earlier stage of development. If the execution of the mutant bifurcation program is pronged into the spikelet pair stage, it appears the *Fas* gene occasionally is expressed at the switch point between spikelet pair primordia and spikelet primordia. This is similar to a second round of bifurcations in spikelet pair primordia of Argentine popcorn (Sundberg and Doebley, MNL 64:21-22; Sundberg and Orr, unpublished), where a doubling of the row number shifts the popcorn inflorescence from distichy to polystichy.

We also observed in tassels a previously unreported bifurcation of the distal tips of the secondary (lateral) branches (Fig. 4,

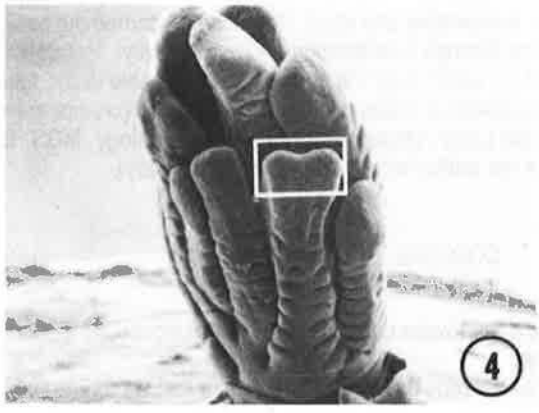


Figure 4. *Fas* tassel after a few bifurcations of central spike tissue. Tips of long lateral branches are bifurcated (box).

box). It seems the mutant program is retained in the branch primordia that give rise to the elongate lateral branches of the tassel. This also is consistent with a prolonged expression (bifurcation) of the *Fas* gene at the spikelet-pair-primordia switch point. Another new observation is the presence of numerous bract primordia within the ring, and at the base, of a cluster of mutant derived female inflorescence axes (Fig. 5). Note in Figure 5 (arrow) the bifurcation of one bract primordium. We are unclear whether these observations represent an expression of the *Fas* gene in the vegetative phase.

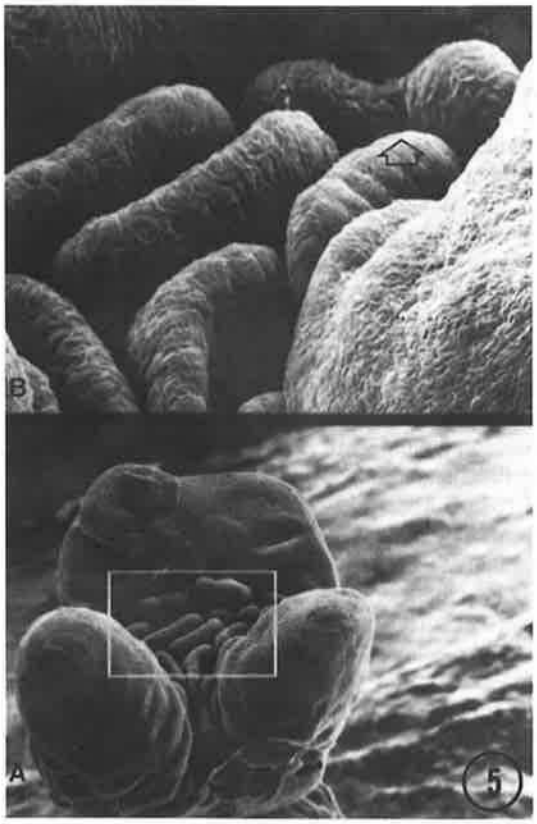


Figure 5. Female inflorescence of *Fas* with numerous bracts clustered at base (A, box) of several inflorescence axes derived from bifurcations of the terminal meristem. Note bifurcation of a single bract (B, arrow).

We are currently testing a time displacement model for a better understanding of the regulatory events in maize floral development. This is based on an analysis of 2-D PAGE protein extracts from selected inflorescence primordia of *Fas*.

CHESTNUT HILL, MASSACHUSETTS
Boston College

Molecular markers of anther culture-derived plants
--Y. C. Ting

In last year's Maize Genetics Cooperation Newsletter, I reported studies on the mutants *cur* and *cfm*, curling tassel and chromosome fusion at meiosis respectively, produced by maize anther culture in vitro. Since then, different studies on the progeny plants of maize anther culture-derived microspore plants were carried out. The procedures followed were RAPD (random amplification of polymorphic DNA).

Williams et al. (Nucl. Acids Res. 18:6531-6535, 1990) reported findings of polymorphic DNA in maize and other eukaryotic as well as prokaryotic organisms. They employed single primers without previous knowledge of their sequence to amplify segments of genomic DNA. The products were reproducible and could be

used for physical mapping.

For the present studies, genomic DNA was extracted by following the protocols of Dellaporta et al. (Plant Mol. Biol. Rep. 1:19-21, 1983). For example, instead of using the crude preparation as directed in the PCR protocol, the preparation was subjected to further purification by the following treatment with RNase. The untreated DNA was removed from the original preparation and transferred into TE buffer in a total volume of 400 µl. Five µl of RNase (10 mg/µl) was added to the solution, which was kept at 37 C for four hr or overnight. The DNA was repeatedly precipitated with NH_4OAc three times with the following components: one v. of DNA, 0.5 v. of NH_4OAc (7.5 M) and two v. EtOH (100%), then washed with 70% EtOH three times. The DNA was dried at room temperature and resuspended in TE (total 400 µl). Then, the concentration of DNA was estimated with a spectrophotometer (1.0 OD_{260} = 50 µg DNA; the sample was diluted in TE for 2^{-2} dilution; for example, 10 µl ---- 1.0 ml).

PCR procedure was carried out in a Perkins-Elmer Cetus Thermal Cycler with the following specifications for each amplification: template DNA (genomic), 1.37 µg; 5' primer 500 ng; 10 x PCR reaction buffer, 10 µl; 10 x dNTP (2 mM), 10 µl; Taq polymerase (Boehringer) 5 µ, 0.5 µl; with H_2O to make up a final volume of 100 µl. Then a few drops of mineral oil were added to the mixture. The cycler was programmed for 40 cycles; for each cycle it took 1 min at 94 C for denaturation of the template DNA, 2 min at 45 C for annealing, and 3 min at 72 C for extension of the primer action. The amplification products were analyzed with agarose gel electrophoresis prepared with 1.2% agarose. Each lane was loaded with 40 µl aliquots. The gel was stained with ethidium bromide, viewed under ultraviolet lamp and photographed with Polaroid film 655. Tubes containing all of the above components except genomic DNA templates were used as a check for all the primers employed. Molecular weight standard was the lambda DNA digested with *Bst*EII.

The employment of eight primers revealed a certain number of polymorphisms of the amplified DNA sequences, which varied from

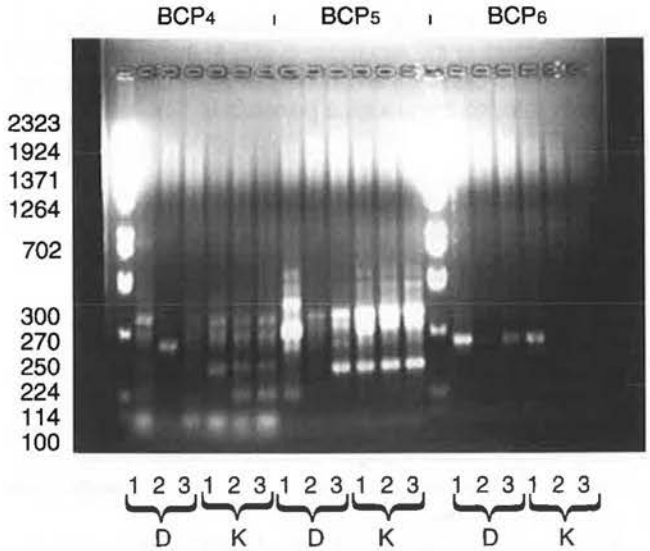


Figure 1. Polymorphisms detected by RAPD markers in maize anther culture-derived progeny plants and their parental individuals. Top: different primers used for three sets of plants. Bottom: D designates Dan-San 91 line and K, King Huang 13 line. In each set, the first lane contains parental DNA from D or K. The second and third lanes contain DNA from the progenies. Numbers to the left side indicate molecular size specified by the standard.

1370 to 100 bp in size. The number of products (bands) obtained with a single primer ranged from one to five. Figure 1 depicts the results for three of the primers. These products, or molecular markers, represent the sequence variations in the genomic DNA of different populations. They are dominant and scorable, and caused by base changes in the primer binding sites or by chromosome alterations within the amplified sequence during culturing. If a maize hybrid and its parents were analyzed with RAPD procedures, the markers appeared to be inherited only from one parent. Amplification of alternate alleles has not yet been found. For primer BCP-4, a band of size 270 bp was present in parental "D and K" (Dan-San 91 and King Huang 13 respectively), and progeny populations of "K", but absent from both progenies of "D" (Table 1). In the same table, it is apparent that polymorphic DNAs were manifested by the different primers. These polymorphisms demonstrated the occurrence of mutations. Thus, they constitute one more piece of evidence supporting the previous conclusion that maize anther culture per se is mutagenic.

Table 1. Polymorphism of genomic DNA of maize anther culture derived progeny plants and their parents revealed by amplification of DNA segments with arbitrary primers, "D" and "K" designate maize varieties. Under "D" and "K", "number 1" indicates parents; 2 and 3, progenies. Nomenclature of primers, BC means Boston College. "P-numeral" numeral means primer code number; "-number" means molecular weights of the products (bands) in base pairs. "+" means present; "0" absent.

	Varieties					
	D			K		
Primers	1	2	3	1	2	3
BCP-4-270	+	0	0	+	+	+
BCP-4-250	+	+	0	+	+	+
BCP-4-224	0	0	0	+	+	+
BCP-4-114	0	0	0	0	+	+
BCP-4-100	+	0	+	+	+	+
BCP-5-300	+	0	0	0	0	0
BCP-5-270	0	+	+	+	+	+
BCP-5-250	+	0	+	+	+	+
BCP-5-224	+	0	+	+	+	+
BCP-5-114	+	0	0	0	0	0
BCP-6-250	+	0	+	+	0	0

Arbitrarily primed RAPDs are molecular markers which are transmitted as genes (mutations). Thus, the procedures of RAPD can add one more category of genes to the existing ones for physical mapping of maize linkage. The protocols involved are simple, inexpensive and rapid. They can be carried out easily without going through hybridization and radioactivity. It is conceivable that RAPD technology will play an important role in the future genetic research of maize and other organisms (credits are due to Dr. Gabin Lazar, Department of Molecular Biology, MGS, Boston, MA, for his skillful help in the PCR of this study).

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The *Ac* transposase consists of several, functionally distinct domains

--Ute Behrens-Jung, Reinhard Kunze and Sandra Kuehn

By means of a transient *Ds*-excision assay we have recently investigated the transpositional activities of several *Ac* transposase (TPase) mutants, and their effects upon coexpression with the active TPase derivative TPase(103-807) (Kunze et al., Proc. Natl. Acad. Sci. USA 90, 7094-7098, 1993). Some of these mutant TPase derivatives act as dominant inhibitors of transposition, leading to the conclusion that the active TPase is an

oligomeric protein.

Since a fraction of the mutant, inactive TPase expression vectors used in that study gave rise to much lower protein levels in the transfected protoplasts than the "wild-type" TPase vector, it could not be determined whether they influence the *Ds* excision frequency upon coexpression with the "wild-type" TPase. Therefore, we have constructed modified plasmids which express the mutant TPases with similar levels as the "wild-type" TPase vector. The results of these experiments - performed under the same conditions as described in the above-mentioned publication - are summarized in Figure 1. Extending our previous results, we found four additional dominant mutants (369TR, 390RV, 445TR and 462TR), and a number of inactive, recessive mutants. The weird transposition frequency boost caused by coexpression of small amounts of mutants Δ(710-807) and Δ(755-807) with "wild-type" TPase is completely abolished [Δ(710-807)] or reduced [Δ(710-807)] upon strong expression of these mutant proteins.

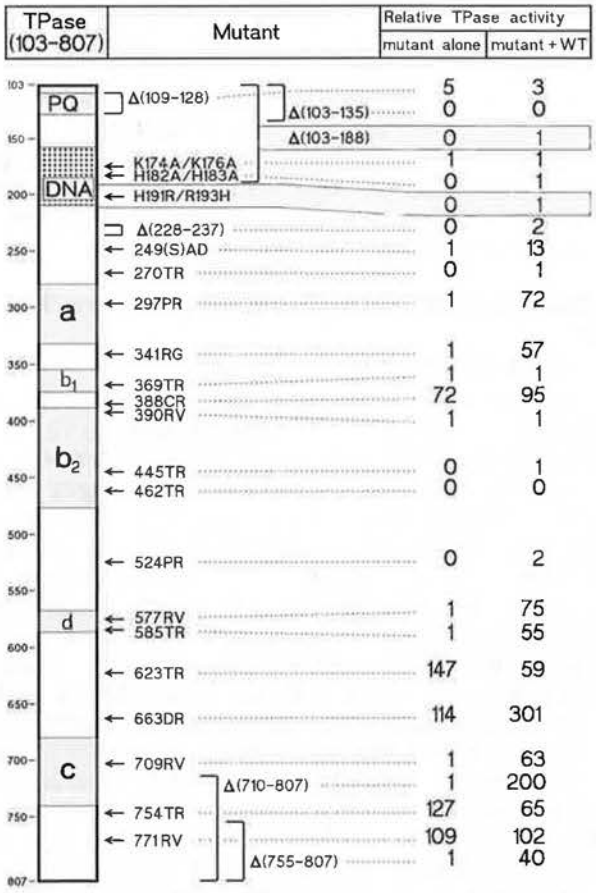


Figure 1. Distribution and relative activities of mutations along the N-terminally truncated TPase(103-807). Certain segments of the protein are highlighted in column "TPase(103-807)": "PQ" = P_{109}QPQPQPQPEPQPQPQPEPE$_{128}$. "DNA" = DNA binding domain. Protein regions with more than 30% sequence identity between the *Ac* TPase and the putative *Tam3* TPase are indicated as "a", "b_1", "b_2", "c" and "d". Column "Mutant" shows the approximate locations of individual mutations. For the nomenclature of mutations the single amino acid letter code is used: Δ(n-m) = deletion of ORFa residues n to m. HnA = substitution of His_n with Ala. nPR = insertion of Pro and Arg behind ORFa residue n. 249(S)AD = substitution of Tyr_{250} with Ser and insertion of Ala and Asp. Protoplasts were co-transfected with 10 µg reporter plasmid and, either 10 µg (mutant) TPase plasmid alone (Column "mutant alone"), or 3 µg "wild-type" TPase plasmid and 15 µg mutant TPase plasmid (column "mutant + WT"). The number of blue-stained, i.e. GUS- positive protoplasts obtained with "wild-type" plasmid alone was taken as 100%. The values shown in columns "mutant alone" and "mutant + WT" are the averages of three to six independent co-transfections and two platings per transfection.

The distribution of dominant and recessive mutations along the polypeptide chain indicates functionally distinct regions of the TPase. With only one exception (mutant 388CR) all mutants between the N-terminus of the active TPase derivative (amino acid [aa] 103) and aa 585 are transpositionally inactive. The C-terminal about 180 aa's seem to be more tolerant against (presumably small) structural disturbances, as four out of five two-aa-insertions in this region are transpositionally active. Nonetheless, the C-terminal about 100 aa's are required for the transposition reaction, as their deletion inactivates the protein.

The inactive TPase mutants fall into two groups - dominant and recessive - which seem to be clustered along the polypeptide chain. Except for mutant 249(S)AD which possibly exhibits an intermediate effect, all inactive mutants between the N-terminus and aa 270, and between aa's 369 and 524 are dominant, whereas two-aa-insertion mutants between aa's 297 and 341, and between aa's 577 and 709 are recessive, respectively. The dominant mutants are most likely still capable of interacting with the "wild-type" TPase. The recessive phenotype could have different causes: (a) the disturbance of protein structure is severe and not locally restricted, preventing any functional interaction with the "wild-type" TPase; (b) the mutants have specifically lost the ability to interact with the "wild-type" TPase; or (c) the mutants can specifically interact with the "wild-type" TPase, but the affected functions are not required in every subunit of the active TPase oligomer.

In order to distinguish between these three possibilities, we have begun to do complementation experiments with pairs of inactive, recessive mutants. We obtained preliminary results indicating that coexpression of mutants 297PR and 709RV leads to the formation of active TPase. Accordingly, these two mutants can specifically interact and fall into two different complementation groups. Presumably, the *Ac* TPase consists of several distinct domains with independent functions.

The DNA-binding sites of the *Ac* transposase

--Heinz-Albert Becker and Reinhard Kunze

The *Ac* transposase (TPase) binds in vitro to short sequence motifs (AAACGG) which occur in multiple copies but different arrangements in both ends of *Ac* and *Ds*. By testing a variety of subterminal *Ds* mutants for transpositional competence in vivo (see report of Shivani Chatterjee and Peter Starlinger) it was found that regions free of AAACGG motifs in the 5'-end of *Ds* are also important for transposition. However, these regions contain motifs which are similar to AAACGG. We have begun to analyze the structural and sequence requirements for specific binding of TPase to DNA in vitro. We performed gel shift assays with the N-terminally truncated TPase(103-807) and radiolabelled plasmid fragments containing various oligonucleotides in different copy numbers, distances and orientations.

When using tandem arrays of AAACGGs as probe, two copies are very weakly complexed, whereas the amount of stable TPase/DNA-complex increases dramatically with higher copy numbers. It turned out that a DNA-unit length of 5 nucleotides is sufficient for complexation. Tandem arrays of six AAACG-, AACGG- and ACGGG-motifs are efficiently bound by the TPase. In contrast, the tetramer ACGG and several motifs with base substitutions in different positions (AGCGG, CGCGG, AAAGGG, AAACTG) are not complexed. The arrangement of motifs in

opposite orientations leads to a strongly reduced but not completely abolished binding efficiency. In summary, these experiments show that DNA-recognition by the TPase is not restricted to a single DNA motif. A WCG-motif repeated in tandem several times and interrupted by two or three nucleotides long spacers (longer spacers have not yet been analyzed) seems to be sufficient for specific binding by the TPase. Individual binding motifs are - except for the central CG-dinucleotide - not palindromic, and tandem arrays of binding motifs result in strongly increased amounts and/or stability of TPase/DNA-complexes. These observations could indicate that individual TPase molecules interact non-symmetrically with each other, forming oligomers variable in size.

In vivo aggregation of *Ac* transposase in nuclei of maize endosperm and petunia protoplasts

--Manfred Heinlein, Torben Brattig, Sandra Kuehn, Ute Behrens and Reinhard Kunze

We visualized the transposase (TPase) of the maize transposable element *Activator* (*Ac*) by immunofluorescence in maize endosperm and in transfected petunia protoplasts. The TPase is detected in the nuclei of both, where it aggregates into large, rod-like complexes about 2 µm in length. Outside of these aggregates, no TPase is detectable. In petunia protoplasts the amount of the complexes is directly related to the strength of the promoter fused to the *Ac* coding region. However, no correlation is seen between the excision frequency of a *Ds* element in the petunia protoplast assay and the amount of TPase aggregates. This is an indication that the TPase protein bound in the aggregates has no TPase activity. We therefore consider the possibility that TPase aggregation serves as a sequestration mechanism which controls TPase activity in the cell. Consistent with such a posttranslational mechanism is the observation that in transgenic tobacco transpositions occur only below a certain threshold in TPase expression (Scofield et al., Cell 75:507-517, 1993).

A functional TPase derivative, lacking the amino-terminal 102 amino acids, differs from the full-length TPase with respect to the formation of aggregates. At low expression levels, no difference in nuclear accumulation is observed between the two proteins. At high expression levels, however, aggregates of the truncated TPase appear in the cytoplasm of many cells, and the amount of nuclear aggregates does not exceed a certain level. In contrast, the wild type TPase still almost exclusively accumulates in the nuclei. Therefore, the N-terminus of the TPase contains sequences involved in nuclear localization or aggregation of the protein.

Correlation of aggregation phenotypes and activity of mutant *Ac* transposase derivatives

--Manfred Heinlein, Sandra Kuehn, Ute Behrens and Reinhard Kunze

In a previous publication it has been reported that some inactive TPase mutants are dominantly inhibiting the transpositionally active TPase. Since two of these mutants are DNA-binding deficient, it has been proposed that the TPase acts as an oligomer or multimer during the excision reaction (Kunze et al., PNAS 87:7094-7098, 1993). We are interested to know whether the oligomerization of the TPase during the excision reaction and the tendency of the protein to form aggregates in vivo (see previous report) are related. Therefore, we started to look for correlations between the activity and the aggregation phenotype of various TPase mutants expressed in transfected petunia protoplasts.

Our tentative results indicate that 1) mutant TPase derivatives which have retained their activity also form aggregates identical to those formed by the wildtype protein and 2) that inactive TPase mutants are able to form aggregates if they are dominant. Both observations are consistent with the assumption that the ability to form aggregates is restricted to TPase molecules that also are able to form oligomers during the excision reaction.

One recessive and one dominant mutant give rise to a homogeneously distributed immunohistochemical signal. Obviously these mutants are not able to form aggregates, however, we also consider the possibility that the observed signal is due to aggregation intermediates. The fact that one of these mutants is dominant could suggest that the formation of TPase oligomers during the excision reaction and the aggregation of the protein occur via different pathways. However, it could also indicate that the protein-protein interactions necessary for oligomerization are not sufficient for the protein to aggregate. According to this hypothesis, aggregation would require an additional protein interactive surface.

Five of the six recessive mutant proteins we investigated are not detectable immunohistochemically although expression in the transfected petunia cells could be shown by Western blot analysis. In addition, certain pairs of these recessive TPase mutants complement to wildtype activity. We are presently trying to find an explanation for this apparent discrepancy.

***Ac* allele-specific variegation patterns are not due to modifier genes**

--Manfred Heinlein and Peter Starlinger

The timing and the frequency of *Ds* excisions differ between maize kernels which carry the *Ac* elements present in the *wx-m7* and *wx-m9* alleles, respectively, although the two elements are identical in sequence and are located within the same gene in identical orientation (Heinlein and Starlinger, Maydica 36:309-316, 1991). Immunohistochemical staining of 30 DAP (days after pollination) endosperm cells for the TPase revealed that during this late stage of endosperm development the *wx-m7* endosperm contains much more of the TPase aggregates than *wx-m9* endosperm. The two *Ac* elements therefore might be differentially expressed. This is further corroborated by the observation that the *Ac* alleles can not be replaced by each other in kernels carrying the *bz-m2(DI)* allele without altering the pattern of revertant *Bz* sectors in the aleurone. Whereas *wx-m7/wx-m7/wx-m7* endosperms are characterized by very large revertant sectors due to excision events having occurred during early developmental stages, the phenotype of the *wx-m7/wx-m7/wx-m9* endosperms exhibits very rare and unicellular sectors due to late events. However, the history of the *wx-m7* and *wx-m9* maize lines used in the crosses are not known in detail and these lines therefore cannot be considered isogenic. Consequently, we performed genetic experiments aimed to reveal whether other gene products (encoded by modifier genes) are involved in the control and regulation of transposition.

We started our experiments with heterozygotes between *wx-m7* and *wx-m9* and crossed this line to an appropriate tester strain, e.g. *bz-m2(DI)*. If the differences between the *wx-m7* and *wx-m9* lines were due to the presence of modifier genes, we expected either a new variegation pattern in the progeny of this cross if several unlinked or loosely linked modifier genes were in-

volved, or the reappearance of the two previously known patterns segregating independently of the *Wx* alleles. The outcome of this experiment was the reappearance of the two previous patterns, which segregated in the expected ratios. The particular patterns seen in the aleurone were concordant with the respective *wx-m7* and *wx-m9* variegation patterns in the inner endosperm. However, this was not taken to be diagnostic for the presence of the specific *Wx* alleles since the variegation patterns of these alleles to some degree might underlie the action of the putative modifier genes also. Hence, we extracted the DNA from 120 kernels showing either of the two variegation patterns and probed this DNA by PCR for the presence of the respective waxy-alleles. In all but two cases (which might be misselections) we found that the kernels which exhibit an *Ac*-specific variegation phenotype also carry the respective *Ac* element. The outcome of this experiment therefore excluded the presence of a modifier gene linked to *Ac*, unless this gene maps very close (within 2 map units) to *Ac*.

As a next step we crossed plants grown from selected kernels that did not receive an *Ac* element due to a meiotic excision event to plants that carried the other *Ac* element combined with the *bz-m2(DI)* allele. The chromosome that previously carried *Ac* should still carry the putative tightly linked modifier gene. For the case that this modifier gene gives rise to the "*Ac* specific" variegation pattern rather than *Ac* itself, we expected the appearance of the weakly variegated phenotype characteristic for *Ac7/Ac9* heterozygotes (see above) on 25% of the progeny kernels. However, this phenotype was not found on the ears. Instead, we solely observed variegation patterns characteristic for the *Ac* element brought in by the tester plant.

Taken together, these results strongly suggest that the *Ac* allele specific variegation patterns are not due to modifier genes. Accordingly, it seems reasonable to assume that the differences between *Ac*-specific variegation patterns are due to differential expression of the *Ac* elements.

Dosage effects of *Ds*

--Manfred Heinlein and Peter Starlinger

Previous experiments employing the *Ds9* element of the *wx-m9Ds* allele (which differs from *Ac* in *wx-m9* by a deletion of 194 bp) have shown that the frequency of *Ds9* excisions is not only dependent on the dosage of the transactivating *Ac* element (we used the *Ac* element present in the *bz-m2* allele of the *Bz1* gene) but also on its own dosage. We found that the *Ds9* element is more often excised if inherited from the male than if inherited from the female and that, if homozygous, the *Ds9* element is excised with a frequency which by far exceeds the sum of the frequencies obtained with one and two *Ds* elements. Accordingly, the maternally and paternally transmitted *Ds9* elements seem to be much better substrates for excision if combined in one nucleus than if separated. These results were independent of the dosage of the transactivating *Ac* element.

We have repeated the same kind of experiment with the *Ds* element of the *bz-m2(DI)* allele and used either one, two or three doses of the *Ac* element present in the *wx-m7* allele or of the *Ac* element present in the *wx-m9* allele for transactivation. The patterns seen on the ears are currently being evaluated.

Studies on *Ac/Ds* methylation

--Lihua Wang, Manfred Heinlein and Peter Starlinger

In vitro, the DNA-binding affinity of the *Ac* transposase is enhanced if its hexameric target motif AAACGG is hemimethylated. This observation by Reinhard Kunze seems to fit to the earlier finding by geneticists that transposition is often linked to replication. In order to test the in vivo significance of this correlation, we have begun genomic sequencing of the subterminal sequences of *Ac* by a PCR-based method developed by Frommer et al. (PNAS 89:1827-1831, 1992). We make use of the fact that the mutagen bisulfite is able to distinguish between methylated cytosin residues, which are stable, and non-methylated cytosin residues, which are oxidatively desaminated to yield uracil.

Characterization of the *Ac* sequences required in cis for transposition

--Shivani Chatterjee and Peter Starlinger

This project comprises the investigation of the cis-acting sequences in the termini of the mobile element *Ac*, which are required for excision. The excision event results from the interaction of the terminal sequences with some trans-acting components, the *Ac*-encoded protein and perhaps one or several host-encoded factors. Upon the mutation of an essential terminal sequence this interaction should be disturbed and the excision ability of the resulting element should be reduced or even lost.

Mutations were introduced by applying the technique of oligonucleotide-directed mutagenesis and the resulting elements were tested on their excision ability in the "Petunia-filter-assay". In this transient assay system excision events can be visualized as beta-glucuronidase-expressing protoplasts (blue spots). Because deletion experiments had restricted the location of the cis-acting sequences to the terminal 200 bp of each end, the mutations were established in these regions.

A first series of mutations altered the sequence motif GGTAAA, which was protected by nuclear extracts of *Ac*-free maize (H. A. Becker), implying the involvement of a host-factor. Substitutions of individual copies of this motif did not result in a loss of excision in vivo, indicating either that the host-factor binding is not critical for the excision-reaction or that the loss of a single motif is not severe enough to prevent excision. Another motif that was altered is the hexameric sequence AAACGG, which is reiterated many times in both ends of *Ac* , and which is bound in vitro by the *Ac* protein. This motif was substituted block-wise by unrelated sequences. While the substitution of four perfectly repeated motifs resulted in no loss of function, the substitution of a less conserved block of hexameric motifs located in the vicinity led to a nearly tenfold reduction of excision frequency.

The combination mutant with both blocks substituted could not be excised at all. Furthermore, a group of point mutations located in the vicinity of the inverted repeats resulted in a dramatic reduction of excision frequency. Only one of eight of these mutations proximal to the 5' or 3' inverted repeat altered a perfect AAACGG-sequence, whereas the other mutations did not change obvious sequence motifs. The exchange of the inverted repeats of *Ac* with those of the Tam3 element resulted in a hybrid element that is no longer mobilized by the *Ac* protein.

From these findings the following conclusions can be tentatively drawn, although the role of the different sequence elements is not yet completely understood:

1. The inverted repeats are indispensable for transposition.
2. The blocks of AAACGG hexamers contribute to the excisability of the element. With the exception of one such motif very close to the 3'-terminus, none of these elements seems to play an

exclusive role. This does not make it likely that single elements are absolutely necessary for transposition, while the others help in setting up a transposition complex. On the other hand, no such alteration has yet yielded an increase of excisability. This renders the hypothesis unlikely that some of these hexamers bind transposase in a non-productive way and thus help in keeping the transposition rate low.

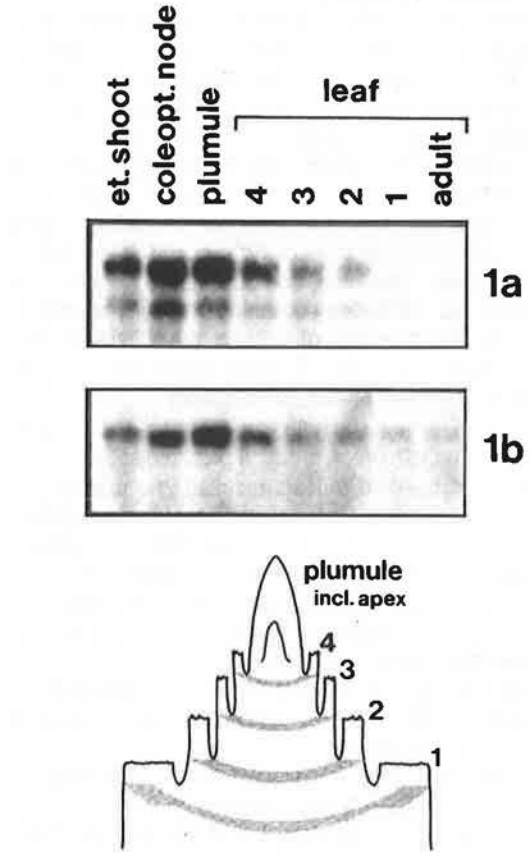
3. Sequences between the inverted termini and the AAACGG hexamer block can be point-mutated to yield pronounced decreases in excisability, though they do not bind transposase in vitro. The role of these sequences is yet unclear.

Ectopic expression of *Zmhox1b* alters the development in transgenic tobacco

--Bärbel Überlacker, Claudia Mehlem and Wolfgang Werr

Zmhox1a (Zea mays homeobox gene 1a), our first homeobox gene, was isolated by screening a λgt11 expression library with the 26 bp feedback control element of the *Shrunken* promoter. Its homeodomain is only distantly related to other plant homeodomains, including the maize *Knotted* class. Therefore *Zmhox1a* is a member of an unrelated class of maize homeobox genes. A close relative, *Zmhox*1b, was isolated using the *Zmhox1a* homeobox as a probe (Bellmann and Werr, 1992, EMBO J. 11:3367-3374). Both genes represent a highly related gene pair, the gene products share 91% similarity on the protein level. They are not alleles because *Zmhox1a* maps on chromosome 8, while *Zmhox1b* is located on chromosome 6.

Northern experiments show that *Zmhox1a* and *Zmhox1b* are transcribed at low level in most tissues. In contrast to *Zmhox1b* probes, with which similar RNA amounts were detected in all tissues except in leaves, *Zmhox1a* transcript levels peaked in suspension cells and during embryonic development. From young to adult leaves the *Zmhox1a/b* transcripts decrease, *1a* drops below the detection level while the *1b* RNA remains visible at a very low level (see Fig. 1).



To gain insight into biological functions we raised transgenic tobacco plants ectopically expressing the *Zmhox1a/b* gene products. Only the protein coding regions were expressed to exclude posttranscriptional regulation, which may involve the 5' and 3' untranslated sequences of *Zmhox1a* or *b* cDNA clones. The open reading frames were fused behind the Ω leader sequence of TMV and expressed under the control of the CaMV 35S promoter. Due to differences in the cloning strategy, so far the experiment with *Zmhox1b* is most advanced.

Ectopic expression of the *Zmhox1b* gene affects the development of the vegetative plant body and the flower. Out of 137 independent primary transformants 70% show an abnormal flower phenotype. Two whorls are affected: the stamen and the carpel. Different flowers contain between 1-5 petaloid stamens which often are also fused. The filament is unchanged but petals grow out below the anthers; the pollen of these transformed stamens is sterile. Severe phenotypes carry the homeotic transformation in all stamens and are sterile even in wild-type backcrosses. In addition these plants show alterations in the carpel. The stigma is changed in shape, the position of the ovary is displaced from the receptacle towards the stigma and the number of ovules per placenta is highly reduced. Besides changes in the flower, the ectopically expressing tobacco plants are often more branched than control plants. Most plants are shorter than the wildtype and exhibit outgrowth of additional flowering side shoots below the main flower. Severely affected plants grow adventitious shoots from every axillary bud, but unfortunately are often sterile. The abnormal flower development, the weaker bushy growth habit and plant shortness are stably inherited into the next plant generation.

The *Zmhox2a/b* gene pair is highly transcribed in meristematic maize tissues

--Bettina Klinge, Christian Korfhage and Wolfgang Werr

Screening of different embryonic cDNA libraries with the *Zmhox1a* homeobox (Bellmann and Werr, EMBO J. 11:3367-3374, 1992) led to the isolation of two highly related genes, each containing two homeoboxes. *Zmhox2a* and *Zmhox2b* (Zea mays homeobox) encode mRNAs of 6 kb length, and the deduced amino acid sequences exihibit modular proteins of 89% identity. The products of both genes are composed of eight basic NLS-like repeats at the N-terminus, followed by a cysteine rich domain conserved in the *Arabidopsis HAT3.1* gene (denoted plant homeodomain finger, Schindler et al., Plant J. 4:137-150, 1993) and two central 159 aa repeats each containing one functional homeodomain. Both homeodomains function independently as DNA-binding motifs. Furthermore eleven proline/glycine rich repeats are found at the C-terminus of the Zmhox2a gene product whereas there are ten in the Zmhox2b protein. Interestingly the central part of the Zmhox2a/2b pair exhibits significant similarity to the Zmhox1a/1b proteins which comprises two N-terminal repeats, the cysteine rich domain and one homeodomain (Fig. 1). Therefore both gene pairs have a common ancestor in evolution.

Northern analysis shows identical transcription patterns for both genes in different maize tissues. High mRNA levels are found

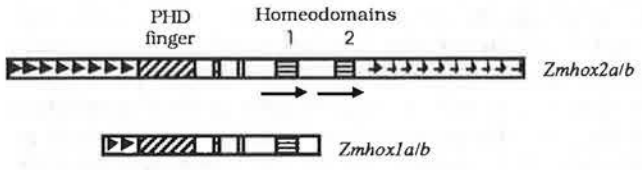


Figure 1.

in all embryonic stages (Abbe and Stein, Amer. J. Bot. 41, 1954), the plumule and the developing male or female flower, reduced amounts in roots and shoots while only a low transcript level can be detected in dormant embryos and mature leaves.

In situ hybridization experiments (Fig. 2) confirm that the *Zmhox2a/b* transcripts accumulate in the meristematic regions of

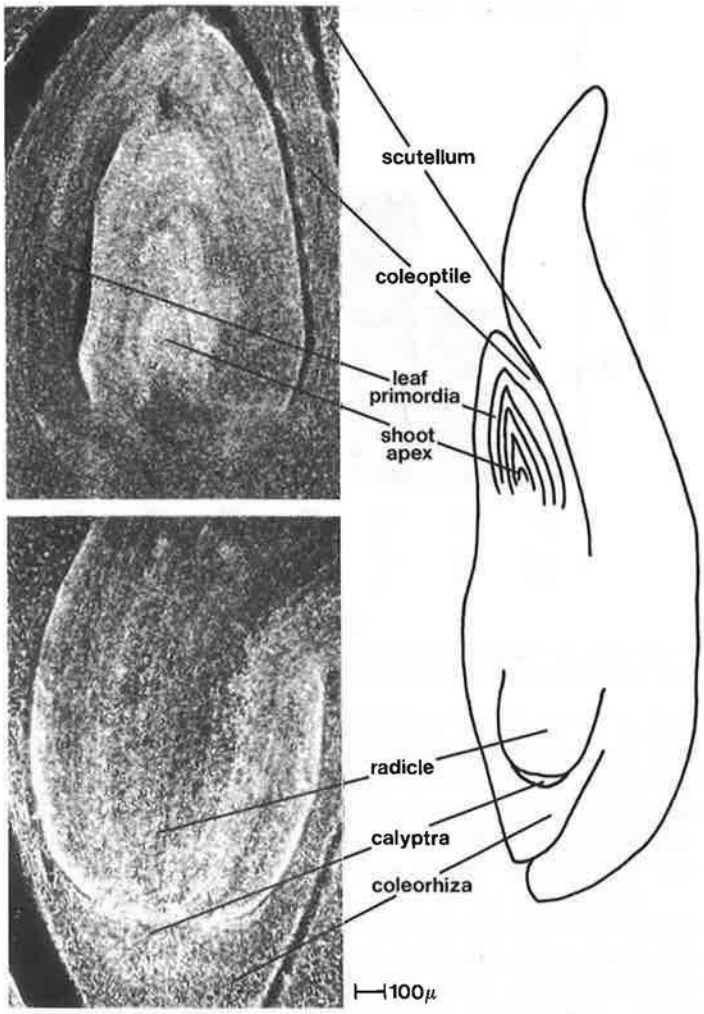


Figure 2. In situ hybridization.

all organs analysed. These include the embryonic shoot and root apical meristem, the youngest leaf primordia, the plumule and root tip of the maize seedling and the spikelets of the developing female flower. At the moment transgenic tobacco plants ectopically expressing the Zmhox2a/2b proteins are being regenerated. By this type of gain of function experiments we hope to get hints on the biological function of this double-homeobox gene pair.

COLUMBIA, MISSOURI
University of Missouri and USDA-ARS

The solid-state chlorophyll meter: a novel instrument for rapidly and accurately determining the chlorophyll concentrations in seedling leaves

--Brent Krugh, Lisa Bickham and Donald Miles

The amount of chlorophyll per unit leaf area in maize is a good indicator of the overall condition of the plant. Healthy plants, those capable of maximum growth, generally can be expected to have larger amounts of chlorophyll than unhealthy plants. Therefore, determination of the chlorophyll content of a leaf can be used to detect and study plant mutations, stress, and nutritional state. The standard method for determining the amount of chlorophyll in a leaf sample is to homogenize the leaf tissue in 80% acetone, measure the absorbance at 663 and 645 nm, and then calculate the chlorophyll concentration using the specific absorption coefficients for chlorophyll a and b (MacKinney, J. Biol. Chem. 140:315-322, 1941; Arnon, Plant Physiol. 24:1-15, 1949). Although this method works well, it has two drawbacks. First, this method is time consuming, especially when there are numerous specimens to analyze. Secondly, the leaf specimen for which the chlorophyll amount has been determined is destroyed, thus making further study of that specimen impossible.

The Minolta Chlorophyll Meter SPAD-502 (Spectrum Technologies, 12010 S. Aero Dr., Plainfield, IL, 60544, 1-800-248-8873) (Minolta Corporation, 101 Williams Drive, New Jersey 07446, USA) is a lightweight handheld meter which allows one to quickly read the chlorophyll concentration of a leaf with no damage (Fig. 1). The SPAD-502 was initially developed to aid rice growers in determining when their crops were in need of nitrogen supplementation (Turner and Jund, Agron. J. 83:926-928, 1991). They found a direct correlation between available nitrogen and leaf chlorophyll during the pre-panicle initiation and panicle differentiation growth stages. The meter utilizes two LEDs which emit light onto the upper surface of a leaf; a red LED with a peak

Figure 1. The Minolta Chlorophyll meter SPAD-502 being used to determine the chlorophyll concentration of a maize seedling leaf.

wavelength of 650 nm and an infrared LED with a peak wavelength of 940 nm. The light enters the leaf where a portion of the light is absorbed by chlorophyll and the remainder is transmitted through the leaf where it contacts a silicon photodiode detector and is converted into an electrical signal. The amount of light reaching the photodiode detector is inversely proportional to the amount of chlorophyll in the light path. After the signal is processed the absorbance is displayed in arbitrary units from 0 to 199. The procedure takes only seconds to perform and the meter is equipped to store 30 readings, average the data, and make data deletions when necessary. To assure accuracy and consistency the meter is calibrated prior to each use with a standard calibration filter which is supplied with the meter.

Since the SPAD-502 meter gives the data only in arbitrary units, it is more useful and meaningful if the data were correlated to actual amounts of chlorophyll per unit area of leaf tissue. In order to accomplish this, leaf disks were excised from 8-15 day old B73 maize (*Zea mays* L.) seedlings. The leaf disks were used to obtain SPAD values and for the calculation of total chlorophyll. A section of leaf was selected and a circular disk 0.87 cm in diameter was cut from the section. SPAD values were obtained from five locations on the leaf disk and averaged. The disk was homogenized in 80% acetone to extract chlorophyll and then, after a brief centrifugation to remove leaf material, the absorbance was measured at 663 nm and 645 nm. Using these absorbance values, the chlorophyll concentration was calculated with the formula described by Arnon (1949). The SPAD values were plotted against the calculated chlorophyll concentrations (adjusted to a leaf disk area of 1.0 cm^2) and when a line was fit to the data points, a linear relationship with a 0.96 correlation resulted (Fig. 2).

In addition, we also used the SPAD-502 to address the common practice of classifying maize mutants as "yellow", "yellow-green", "green", or similar phenotype. The usual method of making these classifications is to simply look at the plant and decide what general color it appears. This method seems somewhat ambiguous and assigns no real parameters to these classifications. To address this situation, we used 10 day old seedlings of the maize mutant *Oy-700*, which segregates into three phenotypes designated

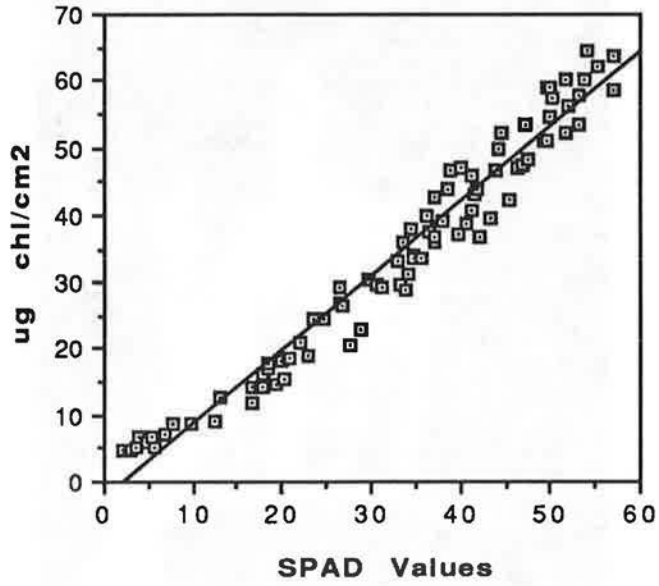


Figure 2. Graphic representation of the correlation of SPAD values with chlorophyll concentration per cm^2 in maize seedling leaves.

"yellow", "yellow green" (oil yellow), and "green" (Hopkins et al., Z. Pflanzenphysiol. 99: 417-426, 1980). One hundred seeds were planted and on the tenth day, each was visually assigned to one of the three color classifications: two were yellow, 24 yellow-green, and 70 green. In order to maintain some sense of uniformity in numbers, 25 of the green plants were randomly chosen to be included with the 24 yellow-green and 2 yellow plants in this study. Five SPAD values were obtained from the second leaf of each plant ranging in a random array from mid leaf to the leaf tip. Regardless of where the measurement was attempted, the SPAD-502 was unable to detect any chlorophyll in the yellow plants. The five SPAD values obtained from each of the yellow-green and green plants were averaged and used to generate a plot for each color classification (Figs. 3 and 4). To generate plots for the

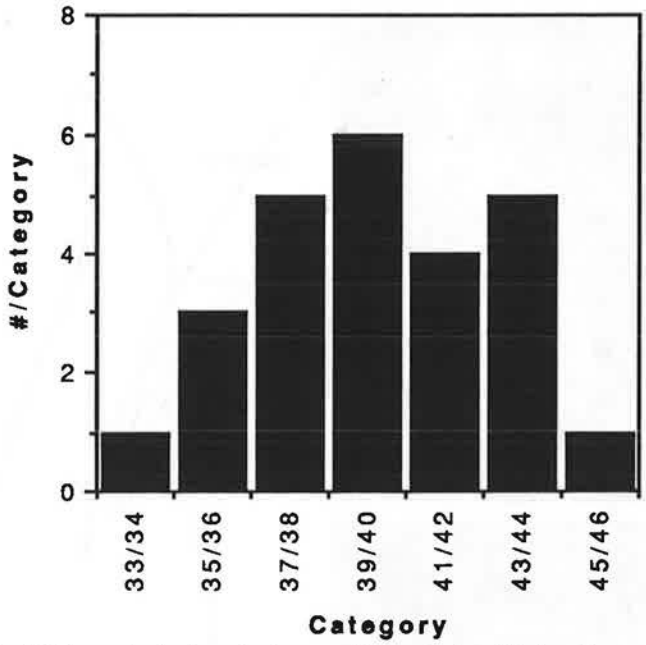


Figure 3. Histogram showing the number of representative "green" plants falling in each category constructed using the averages of the five SPAD readings from each plant.

data, whole numbers throughout the range of averages for each color classification were grouped in consecutive pairs (categories) and plotted against the number of individual averages falling within each category (#/category). For example category 12/13 would represent all SPAD averages from 12.00 to 13.99. The mean SPAD value averages were determined to be 17.35 for yellow-green plants and 40.02 for green plants. In both cases, greater than 95% of the SPAD value averages were within two standard deviations. Our data suggest the possibility of using the Minolta Chlorophyll Meter SPAD-502 to develop a color classification system that would be more precise than the visual method of making these assignments. For instance, due to the inability of the SPAD-502 to measure any chlorophyll in the yellow plants, a possible "yellow" category would be SPAD values very near 0. Furthermore, yellow-green and green categories could be set up encompassing their respective mean of the SPAD value averages (Fig. 5).

It is important to note that these data were obtained from 8-15 day old maize seedlings and due to variations in leaf thickness and morphology, it may not be applicable to other species or developmental stages of plants. However, the data may prove useful

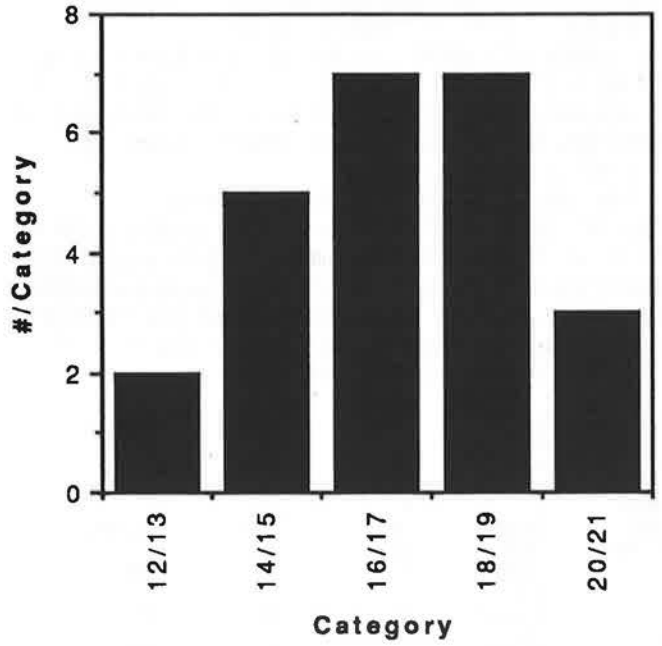


Figure 4. Histogram showing the number of representative "yellow-green" plants in each category constructed using the averages of the five SPAD values from each plant.

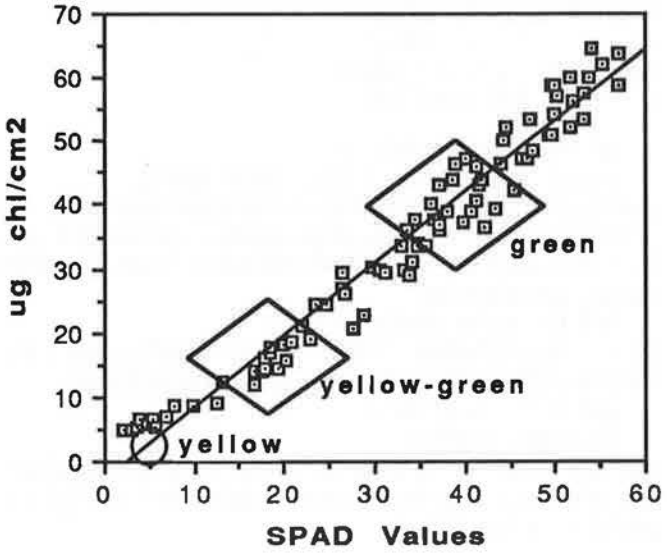


Figure 5. Graphic representation of the correlation of SPAD values with chlorophyll concentration per cm^2 in maize seedling leaves. Possible ranges for a color classification scheme are marked on the graph.

when applied to maize research. It will enable researchers to use the Minolta Chlorophyll Meter SPAD-502 on maize seedlings and then refer to the plot (Fig. 2) as a standard curve and obtain "real" chlorophyll concentrations per unit area. These data may allow this instrument to be used more extensively and in a broader range of analyses. For instance, the same method could be used to create standard curves for other species and developmental stages of plants. Aside from its initial intended use (to monitor the levels of nutrients that affect leaf greening), the SPAD-502 could be used to monitor the effects of environmental pollutants on chlorophyll content of plants or to study and identify plants carrying mutations that affect chlorophyll biosynthesis. Furthermore, the instrument could be used to more accurately and reproducibly classify the effects of mutant genes as "green", "yellow-green", "yellow" etc., thus replacing the visual method of making these judgments. Ranges of SPAD values could be assigned for each color classification (Fig. 5) resulting in a clear, concise classification system.

The Minolta Chlorophyll Meter SPAD-502 can be used to rapidly determine chlorophyll concentrations in plant leaves without damage to the leaf. Initially, one was limited to the arbitrary units which the instrument displays. However, the data and graphs presented above show that there is a linear relationship between the SPAD values and the total chlorophyll (calculated by conventional methods) in maize seedling leaves. This relationship makes it possible to use the graph as a standard curve and determine actual amounts of chlorophyll per unit area from SPAD values. The method presented above can be used to construct standard curves for other species and developmental stages of plants which may not correlate directly to our data due to differences in leaf thickness and morphology. Furthermore, we have shown that it may be possible to assign real parameters to the color classifications that are now typically determined visually. Finally, our data indicate the possibility for a wide variety of uses for the Minolta Chlorophyll Meter SPAD-502 including the detection and classification of mutant plant lineages.

Location of *blh*-2359* on chromosome 8L

--M. G. Neuffer and Dan England

In an EMS-induced recessive mutation, *blh*-2359*, bleached expression begins at the leaf tips and moves toward the midleaf area. This mutant was located on 8L as follows: *+/blh*-2359* x TB-8Lc normal kernels were planted and thinned to 20 hypoploid plants. Nine of these were bleached.

Location of *Yg*-2448* on chromosome 1S

--M. G. Neuffer and Dan England

A bright yellow EMS-induced dominant yellow-green plant mutant was tested for location to chromosome with the full set of *wx*-marked translocations. *Yg*-2448* showed linkage with *wx1* T1-9c(1S.48), as shown in the following data. This was the only one that showed linkage. It indicates a location in the distal region of 1S.

Wx Yg	*Wx +*	*wx Yg*	*wx +*	Rec.
66	22	23	69	.25 ± .032

Bif1-pro1-Lg4 linkage on chromosome 8

--M. G. Neuffer and Dan England

Data from the cross of *Bif1 pro1 Lg4*/+++ on +++ are given below. We had some problems such as reduced numbers of the double dominant *Bif1 Lg4*, and poor expression of *Lg4* in the A632 background used. However, we have been able to establish the order of these 3 genes as *Bif1 pro1 Lg4*. The plants were selfed to test for *pro1*.

+++ x *Bif1 pro1 Lg4*/+++

Locus	*Bif1*	*pro1*	*Lg4*	TOTAL
No. Plts.	37	41	29	89
%	41.6	46.1	32.6	

Classes

Region	Genotype	No.	Totals	Expected
Parental	*Bif1 pro1 Lg4*	19		
	+++	39	58	56.2
R1	*+pro1 Lg4*	4		
	Bif1++	3	7	8.8
R2	*++Lg4*	5		
	Bif1 pro1 +	14	19	20.8
R1,2	*Bif1 + Lg4*	1		
	+ pro1 +	4	5	3.2

Paired Recombination Values
Bif1 --- *pro1* 0.1348 ± 0.0362
pro1 --- *Lg4* 0.2697 ± 0.0470
Bif1 --- *Lg4* 0.2921 ± 0.0482

Designation of *bif2*

--M. G. Neuffer and Steve Briggs

We have recently located a recessive barren inflorescence mutant (*bif*-2354*, from EMS treatment) to 3L using a B-A hypoploid test. This mutant turns out to be allelic to Briggs' *bif*-47330* (Briggs and Johal, MNL66:51 1992). We are designating this gene as *bif2*, with the two alleles described above.

Another pair of factors expressing *orange pericarp*

--M. G. Neuffer and Allen Wright

A second pair of duplicate factors for *orange pericarp*, that are not allelic to *orp1* and *orp2*, have been found in M2 progeny from treatment of pollen with EMS. They differ from *orp1* and *orp2* in that they have separate distinguishable individual phenotypes. The double heterozygote selfed produces an ear segregating approximately 9 normal:3 small dented:2 collapsed nonviable:1 collapsed orange nonviable kernels.

The first of these factors, *cp*-888A* designated *cp3*, which appears as a variably collapsed floury non-pigmented nonviable kernel, segregates 3 normal:1 collapsed from a single heterozygote selfed. It has been located by a hypoploid test as proximal to the breakpoint of TB-1La but not uncovered by TB-1Sb. This places it between the breakpoint of these 2 translocations on chromosome 1.

The second factor, *smk*-888C*, designated *mn4*, segregates in heterozygote selfs as 3 normal:1 nearly normal slightly smaller dented viable kernels that germinate to produce a normal plant. This mutant has not been located to chromosome but it apparently segregates independently of *cp*-888A*. The double mutant phenotype, which occurs in a frequency of 1/16 from the double heterozygote selfed, is a slightly more collapsed nonviable kernel that has a distinctive orange pigmentation of the pericarp. The nature and relationship of the pigment to the indole-induced pigment from *orp1* and *orp2* has not been determined.

New mutant designations

--M. G. Neuffer

The following mutants have been assigned gene symbols. They will also be described in the forthcoming edition of *Mutants of Maize*, which will include pictures of *emp3*, *Lld1*, *nld1*, *rgh1*, and *rli1*.

csp1 not located *white spot*

Expression: good Viability: normal

Originally isolated by R. Kerstetter; our lab number was *lws*-A1173*. Tiny- to medium-sized elliptical, nearly transparent spots that appear almost white and are scattered on the leaf blade beginning at 8-leaf stage and continuing to maturity. No appreciable effect on plant vigor.

dlf1 not located *delayed flowering*

Expression: excellent Viability: normal

(Our number *dlf*-2389A*). Tall late plant with additional nodes and leaves at flowering. Mature plant 2 to 3 feet taller and 7 to 10 days later in flowering with 4 to 6 more nodes and leaves than normal sibs. No apparent response to variations in day length.

emp3 8L-89 *empty pericarp*

Expression: excellent Viability: lethal embryo

Was *dek*-1386A*. Small extremely collapsed defective poorly viable kernel. Practically endospermless, giving an empty pericarp appearance. Embryos are small but morphologically and functionally complete and under optimum conditions will germinate to produce small green seedlings. Locating data from *ms8 j1* + X *ms8 j1* +/+ + *emp3*, progeny tested for *emp3*, follow.

Locus	*ms*	*j*	*emp*	TOTAL
No. Plts.	96	97	129	219
%	43.8	44.3	58.9	

Classes

Region	Genotype	No.	Totals	Expected
Parental	*ms j +*	71		
	++emp	106	177	173.5
R1	*+j+*	13		
	ms + emp	10	23	26.5
R2	*+++*	2		
	ms j emp	11	13	16.5
R1,2	*ms ++*	4		
	+ j emp	2	6	2.5

Paired Recombination Values
ms --- *j* 0.1324 ± 0.0229
ms --- *emp* 0.1644 ± 0.0250
j --- *emp* 0.0868 ± 0.0190

lld1 not located *lethal dwarf*

Expression: excellent Viability: lethal seedling.

Dominant *Lld1* small plant with up to three short fleshy leaves that glisten in sunlight. Seen as single seedling and distorted half-plant chimeras in M1 from EMS-treated pollen. No progeny tests possible due to lethality.

nld1 not located *narrow leaf dwarf*

(Our number *nld*-2346*). Small compact plant with narrow rolled leaves that are bleached pale green, especially along the midrib.

rgh1 8L-111 *rough kernel*

Expression: excellent

Was *rgh*-1285*. Small floury kernel with rough and pitted surface and nonviable embryos. Locating data from (+/*pro1*) *j1* + X *pro1 j1* +/+ + *rgh1* follow.

Locus	*pro1*	*j*	*rgh*	TOTAL
No. Plts.	52	56	64	136
%	38.2	41.2	47.1	

Classes

Region	Genotype	No.	Totals	Expected
Parental	*pro1 j+*	13		
	++rgh	37	50	51.1
R1	*+j+*	30		
	pro1 + rgh	14	44	42.9
R2	*+++*	14		
	pro1 j rgh	10	24	22.9
R1,2	*pro1 ++*	15		
	+ j rgh	3	18	19.1

Paired Recombination Values
pro1 --- *j* 0.4559 ± 0.0427
pro1 --- *rgh* 0.5000 ± 0.0429
j --- *rgh* 0.3088 ± 0.0396

rli1 not located *rough lineate*
Expression: good Viability: fair.
(Our number *rgli*-2302*). Lineate-like streaks of protruding tissue on leaf blade which produce a rough texture.

Dominant *Lesion* mutants on chromosome 2 and designation of *Les18* and *Les19*

--M. G. Neuffer and Dan England

We have located 2 more EMS-induced dominant *Les* mutants to chromosome using the *wx*-marked reciprocal translocation method. The data for the locating crosses listed in Table 1 indicate that *Les*-2441* is midway on 2S and *Les*-2450* is proximal to *wx1* T2-9d. Of the 31 known dominant *Lesion* mutants that we have worked with, 21 have been located to 7 chromosomes: 8 on chromosome 2, 4 on chromosome 1, 4 on chromosome 10, 2 on chromosome 3, and 1 each on chromosomes 6, 7 and 9. The high number on chromosome 2 suggests non-random distribution favoring that chromosome.

For comparison we have brought the available location data for all 8 mutants on chromosome 2 together in the table below. The data are not extensive nor highly accurate but nevertheless instructive about the possible positions of these mutants along the chromosome.

Les1, which is located on the genetic map at 58 between *sk1* and *wt1*, has a recombination value of 14 ± 2% with *wx1* T2-9b.

Les4 and *Les*-1378* both show fairly close linkage (2 ±3% and 7 ±3%, respectively) to *wx1* T2-9d which at .83 on the long arm (cytological map), and no linkage to *wx1* T2-9b which is proximal on the short arm. This places these two mutants in the distal region of 2L.

Les10 shows moderate linkage with both *wx1* T2-9b (25 ± 3% recombination) and *wx1* T2-9d (33 ± 2% recombination), indicating that it is probably located between them in the proximal region of 2L.

Les11 shows moderate linkage (23 ± 4% recombination) with *wx1* T2-9c and no linkage with *wx1* T2-9d, indicating a location in the distal region of 2S.

Les15 shows close linkage (2 ± 1.5% recombination) with *wx1* T2-9b suggesting location in the proximal region of 2S near *Les1*.

Les-2441* shows good linkage (15 ±3 recombination) with *wx1* T2-9c, moderate linkage (22 ± 4) with *wx1* T2-9b, and no linkage with *wx1* T2-9d, suggesting a distal location on 2S but not so far out as *Les11*, therefore we are designating this mutant *Les18*.

Les-2450* shows moderate linkage with *wx1* T2-9b (24 ±3% recombinant) and *wx1* T2-9d (26 ± 5% recombination) but very loose linkage with *wx1* T2-9c, suggesting a position between *wx1* T2-9b and *wx1* T2-9d in the mid-arm region of 2L. This is near the location of *Les10*, but since the two are much different in expression we are designating this mutant *Les19*.

With this information we can propose a tentative order along chromosome 2 as follows: *Les11* distal 2S, *Les18* distal 2S, *Les1* mid-arm 2S, *Les15* proximal 2S, *Les10* and *Les19* proximal-mid 2L, and *Les4* and *Les*-1378* distal 2L.

More precise data which may confirm or invalidate these positions will require linkage tests and/or interval mapping.

Table 1: Data from the cross of *Wx Les/wx1* T2-9 on *wx* Normal for the *wx* translocations T2-9c (2S.49 bkpt), T2-9b (2S.18 bkpt) and T2-9d (2L.83 bkpt). The seed was separated for wx, plants and progeny were noted for *Les* phenotype. The mutant *Les10-A607* arose spontaneously; the other *Les* mutants are EMS-induced.

	Bkpt.	*Wx Les*	*Wx +*	*wx Les*	*wx +*	Recombination ±	Region
Les1-843							
wx1 T2-9b	2S.18	171	30	19	126	.1416 ± .0187	2S (mid)
Les4-1375							
wx1 T2-9b	2S.18	30	38	37	40	.4828 ± .0415	
wx1 T2-9d	2L.83	75	14	6	74	.1183 ± .0248	2L (distal)
Les-1378*							
wx1 T2-9b	2S.18	27	30	30	28	.4783 ± .0465	
wx1 T2-9d	2L.83	47	1	5	39	.0652 ± .0257	2L (distal)
Les10-A607							
wx1 T2-9b	2S.18	102	32	32	88	.2520 ± .0272	
wx1 T2-9d	2L.83	157	74	71	134	.3326 ± .0226	2L (prox)
Les11-1438							
wx1 T2-9c	2S.49	46	16	13	51	.2302 ± .0375	
wx1 T2-9b	2S.18	65	75	61	85	.4755 ± .0295	2S (distal)
Les15-2007							
wx1 T2-9b	2S.18	46	1	1	46	.0213 ± .0149	2S (prox)
Les18-2441							
wx1 T2-9c	2S.49	62	3	18	58	.1489 ± .03	
wx1 T2-9b	2S.18	47	17	9	46	.2185 ± .0379	
wx1 T2-9d	2L.83	29	38	29	36	.4924 ± .0435	2S (distal)
Les19-2450							
wx1 T2-9c	2S.49	45	30	35	46	.4167 ± .0395	
wx1 T2-9b	2S.18	64	31	17	91	.2365 ± .0298	
wx1 T2-9d	2L.83	25	9	9	26	.2609 ± .05	2L (prox)

Tests for allelism among dominant lesion mutants

--M. G. Neuffer

Les4 and *Les*-1378* closely resemble one another and are located on chromosome 2L with similar linkage to the *wx1* T2-9d breakpoint. It is therefore possible that they are allelic. After considerable effort we were able to obtain a plant that carried both mutants in repulsion and were able to cross pollen from it on a normal ear. We planted 100 seeds from this cross for observation with the hope of determining whether crossovers could be obtained between the two mutants. Among 98 progeny, 1 normal plant was obtained. This is expected if the two were not allelic; however, it is not possible to exclude contamination since pollen was used and a stray normal pollen grain could give this result. It should be noted, however, that three more extreme plants were also observed and these could be the reciprocal double mutant crossover. The results of this test are indicative but inconclusive. There are still no proven cases of allelism among 19 dominant lesion mutants that we have tested.

Increasing sensitivity and reducing cost and prep time using the "modified dry blot" procedure for Southern and Northern analyses

--Pamela S. Close, Darren Gruis and Kevin D. Simcox

Last year we reported a "modified dry blot" procedure (Simcox and McMullen, MNL 67:116-117, 1993), which is a modification of the standard Southern DNA transfer method (Southern, J. Mol. Biol. 98:503, 1975). The dry blot procedure simply involves using the buffer contained within the agarose gel to transfer DNA fragments onto a charged membrane. The entire process from removing the gel from the electrophoresis unit to pre-hybridizing the membrane requires less than 4 hours. Although many labs have been using a variation of the dry blot procedure to transfer plasmid DNA to membranes, the first report of the use of the dry blot technique in the analysis of complex genomes was just recently published (Kempter et al., TIG 7:109-110, 1991). The dry blot procedure is extremely simple, but effective.

Initially, the dry blot technique was used for F2 and interval mapping procedures in which membranes were stripped and reprobed numerous times. We have stripped several sets of mem-

branes more than 18 times and re-probed with single copy RFLP probes, with no appreciable loss of signal. Membranes produced using the dry blot technique with single copy maize probes require a 1 -2 day exposure. The dry blot procedure has been used by different labs in a number of different applications with the same degree of success. In addition to RFLP analysis, we have used the dry blot procedure for CHEF analysis of YAC clones and high MW maize genomic DNA, co-segregation analysis using *Mutator* probes, and northern analysis.

Several changes were made to adapt the dry blot procedure for northern analysis. In one case, glyoxal-treated poly(A+)RNA from soybean somatic embryos was neutralized in 10 mM $NaPO_4$, pH 6.5 and transferred onto Gene Screen Plus transfer membrane (Ma et al., Plant Mol. Biol. in press, 1994). Using this method, Hongchang Ma and co-workers detected expression of a soybean homeobox-containing gene, homologous to the maize *knotted1* cDNA, after a 2 day exposure. Another variation involved equilibrating and transferring glyoxal-treated total RNA collected at different times after maize leaves were inoculated with *Cochliobolus carbonum* race 1, in 50 mM NaOH onto Hybond N+ transfer membrane (Gruis and Johal, unpublished). Several rare transcripts were detected using the *Hm1* cDNA after a 3-4 day exposure.

We believe that the sensitivity of the dry blot procedure is derived from the use of charged transfer membranes. When other sources of transfer membranes are used, the method used to strip membranes after hybridization should be adapted according to the manufacturer's protocol. The procedure as described in the 1993 MNL article was developed using DuPont's Gene Screen Plus membrane. We found that stripping membranes with 0.4 M NaOH was far superior to high temperature treatments using high stringency solutions. (Names are necessary to report factually on available data; however, the USDA and the University of Missouri neither guarantees nor warrants the standard of the product, and the use of the name by USDA and the University of Missouri implies no approval of the product to the exclusion of others that may also be suitable.)

Combined F2 and IF2 RFLP map

--Oscar Heredia-Díaz, Jack Gardiner, Dave Hoisington, Shiaoman Chao, Ed Coe, Theresa Musket and Guilin Xu

Our initial RFLP mapping research was reported in MNL 63:141-151 in 1989. The first UMC maize RFLP map was generated using an F2 population derived from Tx303 x CO159. The map included 256 RFLP loci, scored in 46 individuals, and the linkage analysis was done using Mapmaker v2.0 (Unix). In order to be able to continue mapping after the F2 tissue was exhausted, a new immortalized F2 (IF2), consisting of 56 individuals, was generated during the spring of 1989 (MNL64:47). The latest count of loci mapped on the IF2 is 404, which includes 389 RFLP-defined loci (268 genomic, 78 leaf cDNA, 43 cloned gene candidates), and 15 isozymes.

Toward our effort to integrate available information on RFLP and morphological loci into a consensus map, we have combined segregation data from both populations (current IF2 and previous F2) to generate a combined map, which consists of segregation data for 616 loci scored on 102 individuals in total.

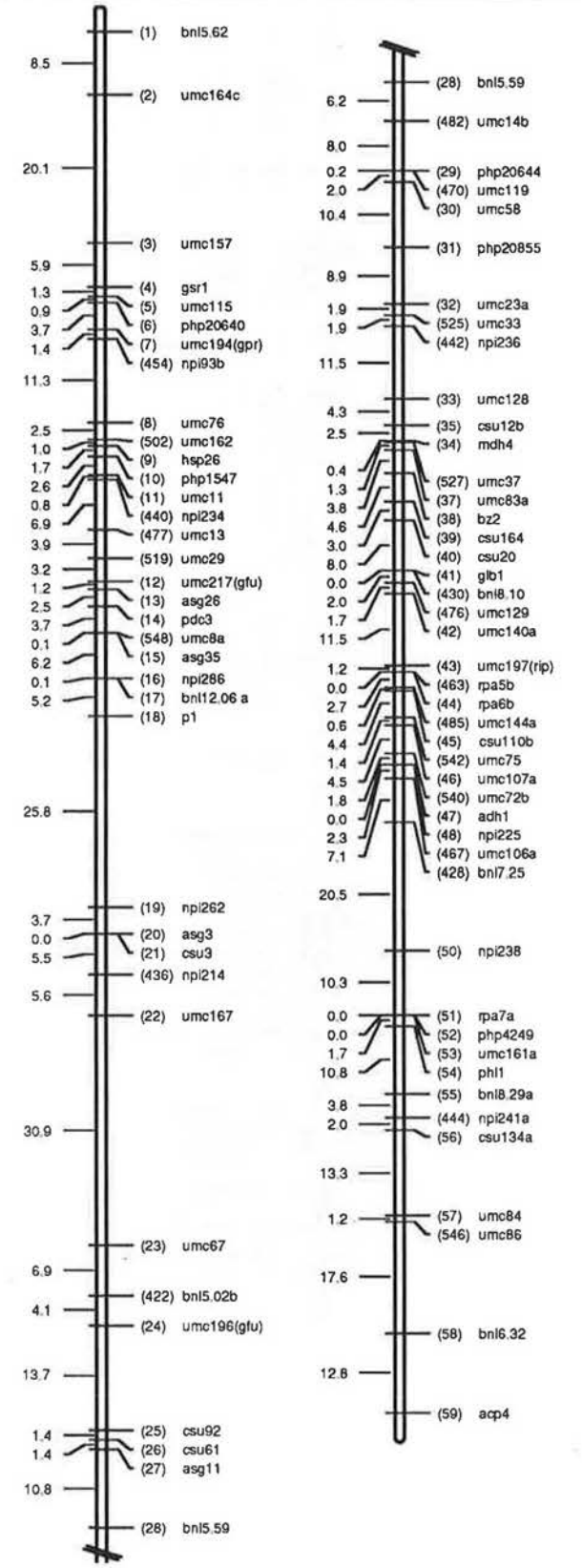
We used SAS (Statistical Analysis System) to concatenate both data sets (see program below). Whenever no segregation information was available for any marker in one of the two populations, it was filled with missing data ("-" in Mapmaker). The combined data set was subjected to linkage analysis using Mapmaker v3.0 (Unix). After identifying linkage groups (LOD=6.0 and θ=0.4), a framework was set identical to the IF2 map order, and thereafter all remaining loci (those mapped on the first F2 , but not on the IF2), were placed by implementing the command "try". The "try" command places a given marker into a known framework of markers by determining the interval into which it can be inserted to give the highest likelihood map. Mapmaker computes the maximum likelihood maps for the given framework and the trial locus inserted into each interval, and will display each map's likelihoods relative to the best likelihood found. All map distances are recomputed for each map calculation. By repeating this procedure, 112 additional RFLP loci were mapped. Whenever a marker was inconsistently placed, i.e. more than one possible placement, it was re-tried after all other loci with solid placement were incorporated into the map sequence. Graphic representation of the combined map is shown in the accompanying figure. Markers with Id number out of sequential order are the newly incorporated loci. Loci with *php* and a 4-digit number are new, detected with recently defined Pioneer probes. Loci with *csu* and a number are ones detected with cDNA sequences isolated by Chris Baysdorfer. These loci were given *umc* numbers in the 300 range on last year's maps, but our consensus (among the Baysdorfer, Helentjaris, Burr, and Missouri labs) is now to use the *csu* designation, with the sequential numbers assigned by Baysdorfer. The mapping data, and the identities and parameters for the probes, are being incorporated into the Maize Genome Database (Maizedb). Please refer to the item on Maizedb, in this issue, for criteria that are being used to define and designate loci and genes (examples: cDNA csu77 shows 68% identity to a bacterial malate dehydrogenase, probes a site matching *mdh4*, mapped as an isozyme; the site is designated *mdh4*).

Incorporation of more data sets with the already existing ones, linkage analysis, and refinement of the integrated maps will provide useful and powerful tools for mapping of complete genomes, genetic studies, gene tagging, gene cloning, and integration of more complex traits.

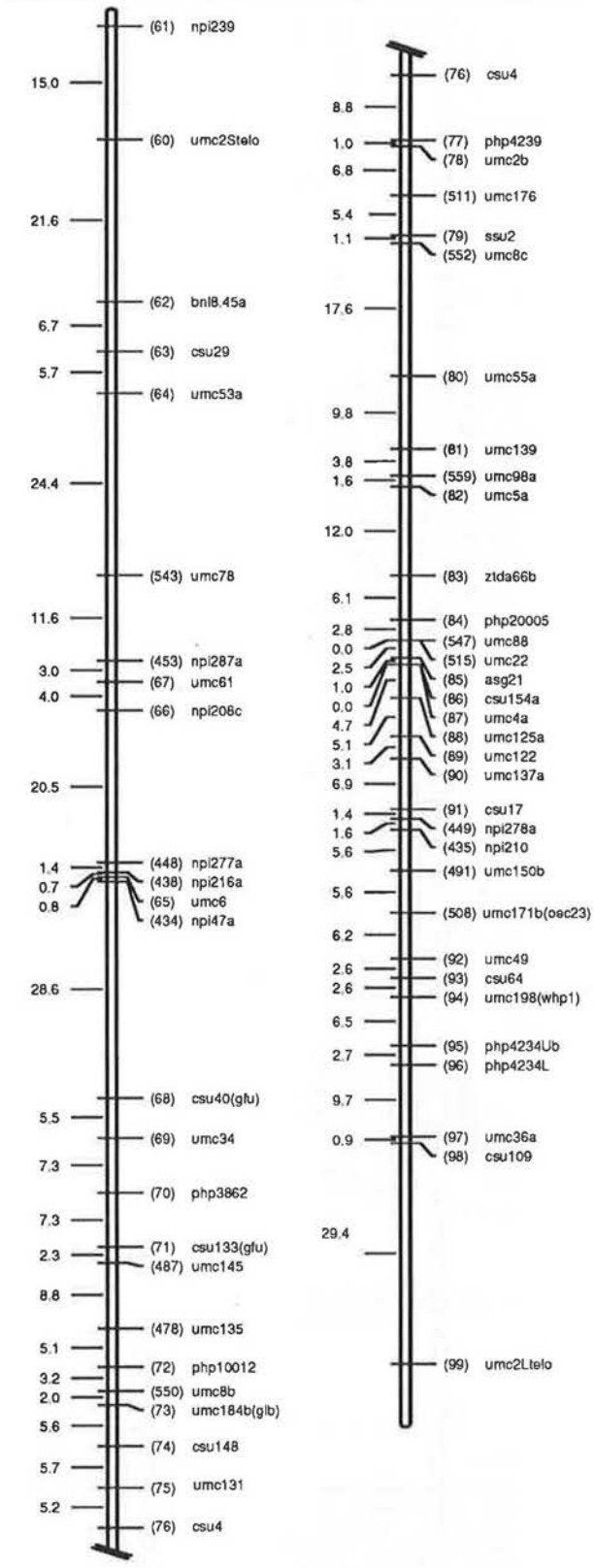
The SAS program used to merge the segregation data sets from both populations follows:

```
FILEDEF POP1 DISK POP1 DATA A1;
FILEDEF POP2 DISK POP2 DATA A1;
FILEDEF POPn DISK POPn DATA A1;

DATA POP1;
INFILE POP1;
INPUT LOCUS $ 1-14 @ 15 (IND1-56) ($1.);
PROC SORT; BY LOCUS;

DATA POP2;
INFILE POP2;
INPUT LOCUS $ 1-14 @ 15 (IND57-102) ($1.);
PROC SORT; BY LOCUS;

DATA POPn;
INFILE POPn;
INPUT LOCUS $ 1-14 @ 15 (IND103-n) ($1.);
PROC SORT; BY LOCUS;

FILEDEF OUT DISK MERGED DAT A1 (LRECL 200 BLOCK 200 RECFM F;

DATA ALL;
MERGE POP1 POP2 POPn;
BY LOCUS;
ARRAY CHANGE IND1-INDn;
DO OVER CHANGE;
IF CHANGE=' ' THEN CHANGE='-';
END;
FILE OUT;
PUT LOCUS $ 1-14 @ 15 (IND1-INDn) ($1.);
```

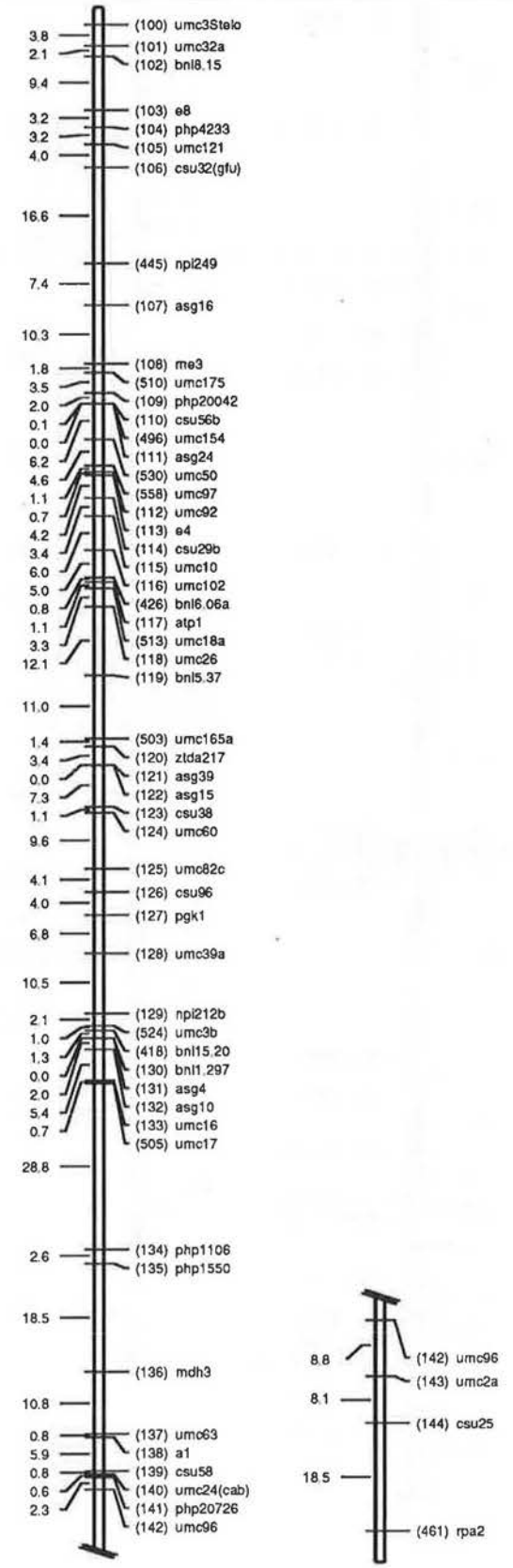
Chromosome 1



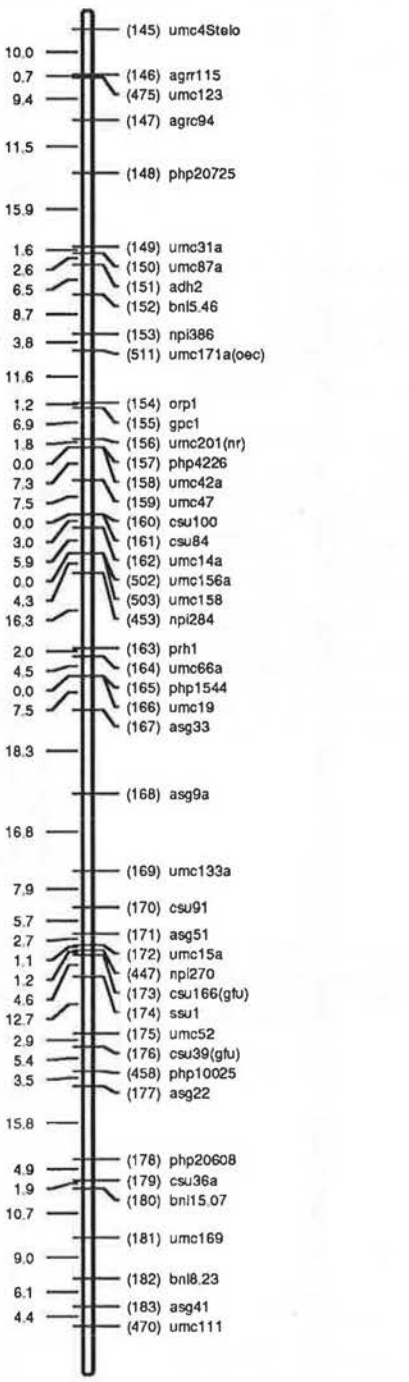
Chromosome 2

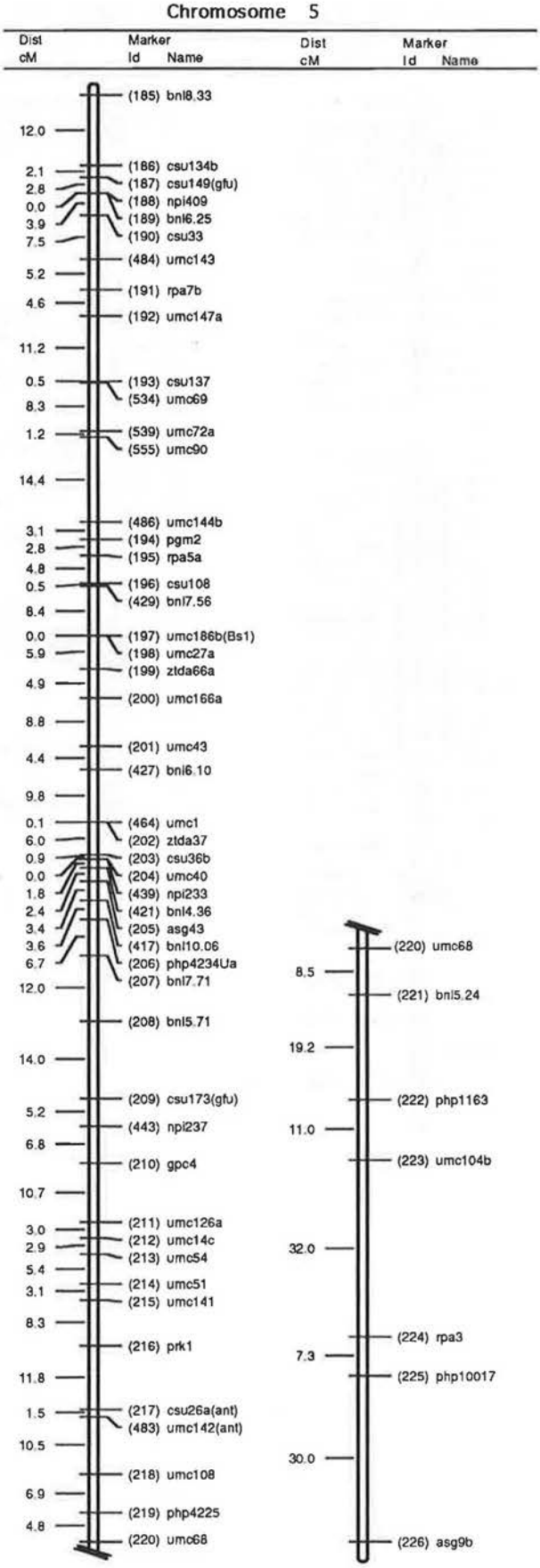


Chromosome 3



Chromosome 4




Chromosome 5
Dist cM
Marker Id Name
(185) bnl8.33
12.0
2.1 (186) csu134b
2.8 (187) csu149(gfu)
0.0 (188) npi409
3.9 (189) bnl6.25
7.5 (190) csu33
(484) umc143
5.2
(191) rpa7b
4.6
(192) umc147a
11.2
0.5 (193) csu137
8.3 (534) umc69
1.2 (539) umc72a
(555) umc90
14.4
3.1 (486) umc144b
2.8 (194) pgm2
(195) rpa5a
4.8
0.5 (196) csu108
(429) bnl7.56
8.4
0.0 (197) umc186b(Bs1)
5.9 (198) umc27a
(199) ztda66a
4.9
(200) umc166a
8.8
(201) umc43
4.4
(427) bnl6.10
9.8
0.1 (464) umc1
6.0 (202) ztda37
0.9 (203) csu36b
0.0 (204) umc40
1.8 (439) npi233
2.4 (421) bnl4.36
3.4 (205) asg43
3.6 (417) bnl10.06
6.7 (206) php4234Ua
(207) bnl7.71
12.0
(208) bnl5.71
14.0
5.2 (209) csu173(gfu)
(443) npi237
6.8
(210) gpc4
10.7
3.0 (211) umc126a
2.9 (212) umc14c
(213) umc54
5.4
3.1 (214) umc51
(215) umc141
8.3
(216) prk1
11.8
1.5 (217) csu26a(ant)
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10.5
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6.9
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4.8
(220) umc68
Dist cM
Marker Id Name
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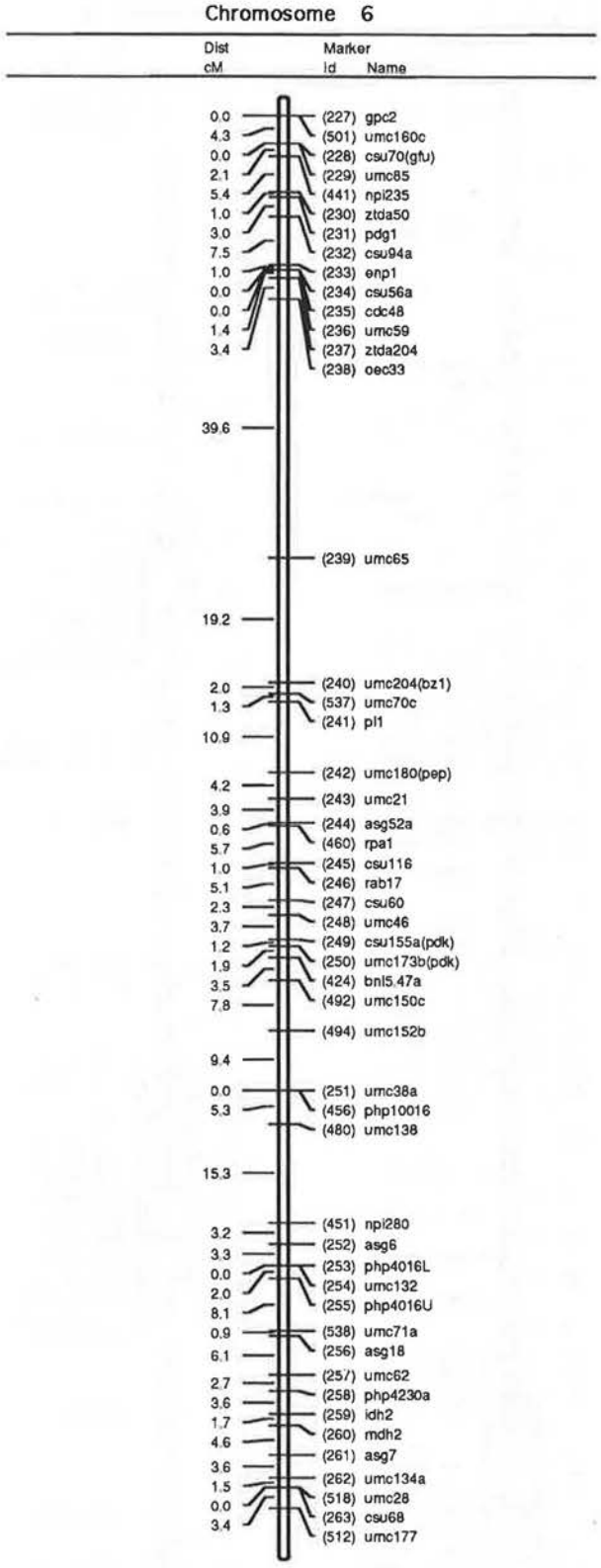
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Marker Id Name
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1.0 (230) ztda50
3.0 (231) pdg1
7.5 (232) csu94a
1.0 (233) enp1
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0.0 (235) cdc48
1.4 (236) umc59
3.4 (237) ztda204
(238) oec33
39.6
(239) umc65
19.2
2.0 (240) umc204(bz1)
1.3 (537) umc70c
(241) pl1
10.9
(242) umc180(pep)
4.2
(243) umc21
3.9
0.6 (244) asg52a
5.7 (460) rpa1
1.0 (245) csu116
5.1 (246) rab17
2.3 (247) csu60
3.7 (248) umc46
1.2 (249) csu155a(pdk)
1.9 (250) umc173b(pdk)
3.5 (424) bnl5.47a
7.8 (492) umc150c
(494) umc152b
9.4
0.0 (251) umc38a
5.3 (456) php10016
(480) umc138
15.3
3.2 (451) npi280
3.3 (252) asg6
0.0 (253) php4016L
2.0 (254) umc132
8.1 (255) php4016U
0.9 (538) umc71a
6.1 (256) asg18
2.7 (257) umc62
3.6 (258) php4230a
1.7 (259) idh2
4.6 (260) mdh2
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3.6
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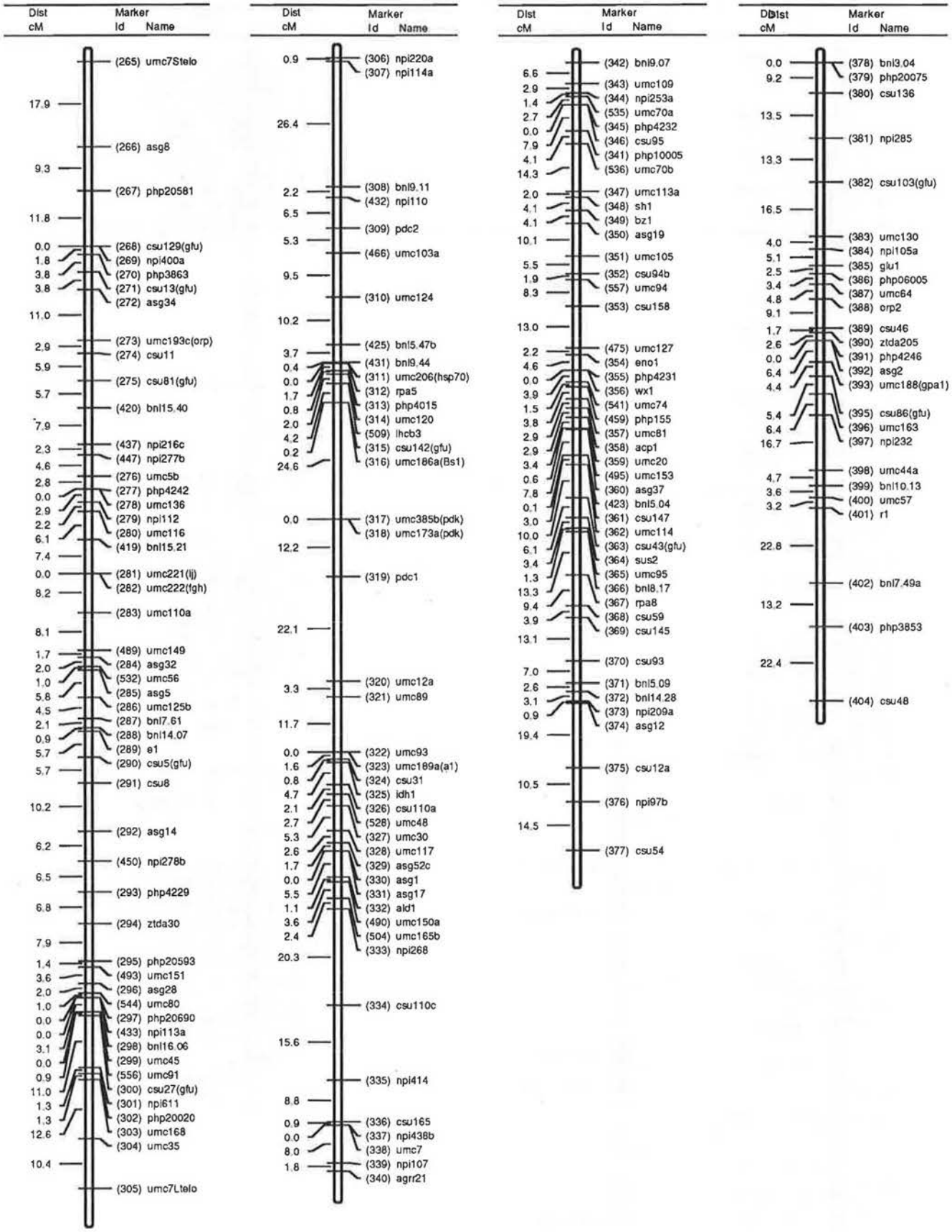
(512) umc177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(266) asg8
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(267) php20581
11.8
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1.8 (269) npi400a
3.8 (270) php3863
3.8 (271) csu13(gfu)
(272) asg34
11.0
2.9 (273) umc193c(orp)
5.9 (274) csu11
(275) csu81(gfu)
5.7
(420) bnl15.40
7.9
2.3 (437) npi216c
4.6 (447) npi277b
2.8 (276) umc5b
0.0 (277) php4242
2.9 (278) umc136
2.2 (279) npi112
6.1 (280) umc116
(419) bnl15.21
7.4
0.0 (281) umc221(ij)
8.2 (282) umc222(fgh)
(283) umc110a
8.1
1.7 (489) umc149
2.0 (284) asg32
1.0 (532) umc56
5.8 (285) asg5
4.5 (286) umc125b
2.1 (287) bnl7.61
0.9 (288) bnl14.07
5.7 (289) e1
5.7 (290) csu5(gfu)
(291) csu8
10.2
(292) asg14
6.2
(450) npi278b
6.5
(293) php4229
6.8
(294) ztda30
7.9
1.4 (295) php20593
3.6 (493) umc151
2.0 (296) asg28
1.0 (544) umc80
0.0 (297) php20690
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3.1 (298) bnl16.06
0.0 (299) umc45
0.9 (556) umc91
11.0 (300) csu27(gfu)
1.3 (301) npi611
1.3 (302) php20020
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(304) umc35
10.4
(305) umc7Ltelo
Chromosome 8
Dist cM
Marker Id Name
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(307) npi114a
26.4
2.2 (308) bnl9.11
(432) npi110
6.5
(309) pdc2
5.3
(466) umc103a
9.5
(310) umc124
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0.8 (313) php4015
2.0 (314) umc120
4.2 (509) lhcb3
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24.6 (316) umc186a(Bs1)
0.0 (317) umc385b(pdk)
(318) umc173a(pdk)
12.2
(319) pdc1
22.1
3.3 (320) umc12a
(321) umc89
11.7
0.0 (322) umc93
1.6 (323) umc189a(a1)
0.8 (324) csu31
4.7 (325) idh1
2.1 (326) csu110a
2.7 (528) umc48
5.3 (327) umc30
2.6 (328) umc117
1.7 (329) asg52c
0.0 (330) asg1
5.5 (331) asg17
1.1 (332) ald1
3.6 (490) umc150a
2.4 (504) umc165b
(333) npi268
20.3
(334) csu110c
15.6
(335) npi414
8.8
0.9 (336) csu165
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8.0 (338) umc7
1.8 (339) npi107
(340) agrr21
Chromosome 9
Dist cM
Marker Id Name
6.6 (342) bnl9.07
2.9 (343) umc109
1.4 (344) npi253a
2.7 (535) umc70a
0.0 (345) php4232
7.9 (346) csu95
4.1 (341) php10005
14.3 (536) umc70b
2.0 (347) umc113a
4.1 (348) sh1
4.1 (349) bz1
10.1 (350) asg19
5.5 (351) umc105
1.9 (352) csu94b
8.3 (557) umc94
13.0 (353) csu158
2.2 (475) umc127
4.6 (354) eno1
0.0 (355) php4231
3.9 (356) wx1
1.5 (541) umc74
3.8 (459) php155
2.9 (357) umc81
2.9 (358) acp1
3.4 (359) umc20
0.6 (495) umc153
7.8 (360) asg37
0.1 (423) bnl5.04
3.0 (361) csu147
10.0 (362) umc114
6.1 (363) csu43(gfu)
3.4 (364) sus2
1.3 (365) umc95
13.3 (366) bnl8.17
9.4 (367) rpa8
3.9 (368) csu59
13.1 (369) csu145
(370) csu93
7.0
2.6 (371) bnl5.09
3.1 (372) bnl14.28
0.9 (373) npi209a
(374) asg12
19.4
(375) csu12a
10.5
(376) npi97b
14.5
(377) csu54
Chromosome 10
Dist cM
Marker Id Name
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9.2 (379) php20075
(380) csu136
13.5
(381) npi285
13.3
(382) csu103(gfu)
16.5
4.0 (383) umc130
5.1 (384) npi105a
2.5 (385) glu1
3.4 (386) php06005
4.8 (387) umc64
9.1 (388) orp2
1.7 (389) csu46
2.6 (390) ztda205
0.0 (391) php4246
6.4 (392) asg2
4.4 (393) umc188(gpa1)
5.4 (395) csu86(gfu)
6.4 (396) umc163
16.7 (397) npi232
4.7 (398) umc44a
3.6 (399) bnl10.13
3.2 (400) umc57
(401) r1
22.8
(402) bnl7.49a
13.2
(403) php3853
22.4
(404) csu48

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Silk browning, maysin content, and corn earworm resistance

--P. F. Byrne, L. L. Darrah, D. J. Moellenbeck, B. D. Barry, M. E. Snook, B. R. Wiseman and N. W. Widstrom

Silks of some genotypes turn brown after wounding, while those of other genotypes do not change color. The trait is controlled by a factor at the *p1* locus (Han and Coe, MNL 60:55, 1986; MNL 61:46, 1987) and is believed to be due to enzymatic oxidation of flavonoid compounds to quinones, which condense with themselves or proteins to produce brown pigments (Levings and Stuber, Genetics 69:491-498, 1971). A previous study found a significant correlation between percentage of plants with browning silks and damage caused by corn earworm, *Helicoverpa zea* (Boddie) (CEW), in cycles of selection of the population 10LDD, which had been selected for CEW resistance (Byrne et al., Environ. Entomol. 18:356-360, 1989). Subsequently, 10LDD Cycle 0 was divergently selected to form subpopulations with silks that were nearly all browning (10LDD BR) or nearly all nonbrowning (10LDD NBR).

In the summer of 1993, these subpopulations were evaluated along with 10LDD Cycles 0, 2, 4, 6, and 7A1. Cycles 2, 4, and 6 were selected on the basis of CEW ear penetration and husk tightness, and Cycle 7A1 was selected based on silk maysin content and dried silk bioassays. Maysin, a flavone glycoside, is a major factor responsible for antibiotic resistance to CEW (Waiss et al., J. Econ. Entomol. 72:256-258, 1979). Two commercial hybrids, Pioneer Brand 3369A and Pioneer Brand 3184, were included as check entries. The CEW resistant variety 'Zapalote Chico 2451# (P) C3' was delay-planted in an adjacent plot. The trial was arranged in a randomized complete block design with five replications, and was grown at three locations in or near Columbia, MO. Experimental plots were two rows 4.9 m long with 0.91 m between rows.

Primary ear shoots were covered with shoot bags to prevent pollination and subsequent physical and chemical changes in the silks. Approximately three days after silks appeared, 10 primary ears per plot of 10LDD BR, NBR, Cycle 0 and Cycle 6, and Zapalote Chico were harvested, packed in coolers with blue-ice packs, and shipped to the USDA-ARS Phytochemical Research Unit, Athens, GA. All silks from the same plot were bulked, and concentrations of maysin and related compounds were determined by reversed-phase high-performance liquid chromatography (Snook et al., J. Chromatogr. 477:439-447, 1989).

At about the same stage of silk development, four silk masses per plot of each entry were collected, dried at 41 C for 10 days, and bulked across replications for each entry at each location. Dried silks were shipped to the USDA-ARS Insect Biology and Population Management Research Laboratory, Tifton, GA. Silks were ground and mixed with a pinto bean diet, and bioassays carried out as described by Wiseman (Toward insect resistant maize for the third world, p. 94-100, CIMMYT, Mexico, D.F., 1989). Fifteen replications of a split-plot design (whole plots=sampling locations, subplots=entries) were conducted, and data recorded on eight-day larval weight, time to pupation, and pupal weight. Silks of eight to ten ears per plot were evaluated for the browning reaction about two weeks after silking began.

Eight to ten ears per plot were artificially infested with CEW eggs at three to five days after silk emergence. Due to environmental conditions, high predator populations, or other factors, the infestation was not uniformly successful; data for depth of CEW penetration were highly variable and are not presented here.

Selecting solely for browning and nonbrowning silks resulted in subpopulations with high and undetectable levels of silk maysin, respectively (Table 1). In the bioassay, the browning subpopulation resulted in significantly lower ($P<0.01$) eight-day larval weights and longer ($P<0.01$) time to pupation than the nonbrowning entry. Little change in the measured parameters was observed from Cycle 0 to Cycle 6, probably because good husk tightness in this population obscured differences in antibiosis. However, after selection criteria were changed in Cycle 7A1, a large decrease in percentage of nonbrowning silks, smaller larval weight, and a longer time to pupation resulted. Pioneer Brand 3184, which had previously demonstrated resistance to European corn borer, *Ostrinia nubilalis* (Hübner), (B. D. Barry, personal communication), showed a high degree of antibiosis to CEW based on the bioassay.

Table 1. Treatment means for various corn earworm resistance factors, combined over three locations and five replications.

Entry	Nonbrowning silks	Maysin	Eight-day larval weight	Time to pupation	Pupal weight
	%	% fresh weight	mg	days	mg
10LLD BR	0.7	0.185	61.0	18.6	466.3
10LDD NBR	97.3	0.000	335.1	13.3	530.0
10LDD C0	34.1	0.077	191.8	14.9	621.5
10LDD C2	39.0	--	197.6	14.4	534.5
10LDD C4	62.2	--	217.4	14.0	538.3
10LDD C6	54.6	0.064	207.0	14.3	535.9
10LDD 7A1	8.5	--	101.1	17.0	492.3
Pioneer Brand 3369A	0.0	--	66.6	17.8	503.1
Pioneer Brand3184	0.0	--	18.6	23.8	409.6
Zapalote Chico	0.0	0.242	48.2	21.3	445.4
Significant difference	10.0†	0.027†	22.9‡	0.6‡	104.2‡

-- Maysin levels of these entries were not measured.
† LSD (0.05)
‡ Minimum significant difference (0.05), Waller-Duncan K-ratio *t*-test.

Because maysin synthesis occurs as part of the flavonoid pathway, a blockage early in the pathway effected by a recessive allele at *p1* will result in silks lacking maysin and other compounds contributing to the browning reaction. Although in the 10LDD population variation at the *p1* locus (and/or loci controlling nearby steps in the pathway) apparently is the key factor determining maysin content, in other materials (those with all browning silks, for example), other loci along the pathway presumably will be responsible for variation in maysin level.

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Current status of the *Tripsacum dacyloides* (Eastern gamagrass) RFLP molecular genetic map

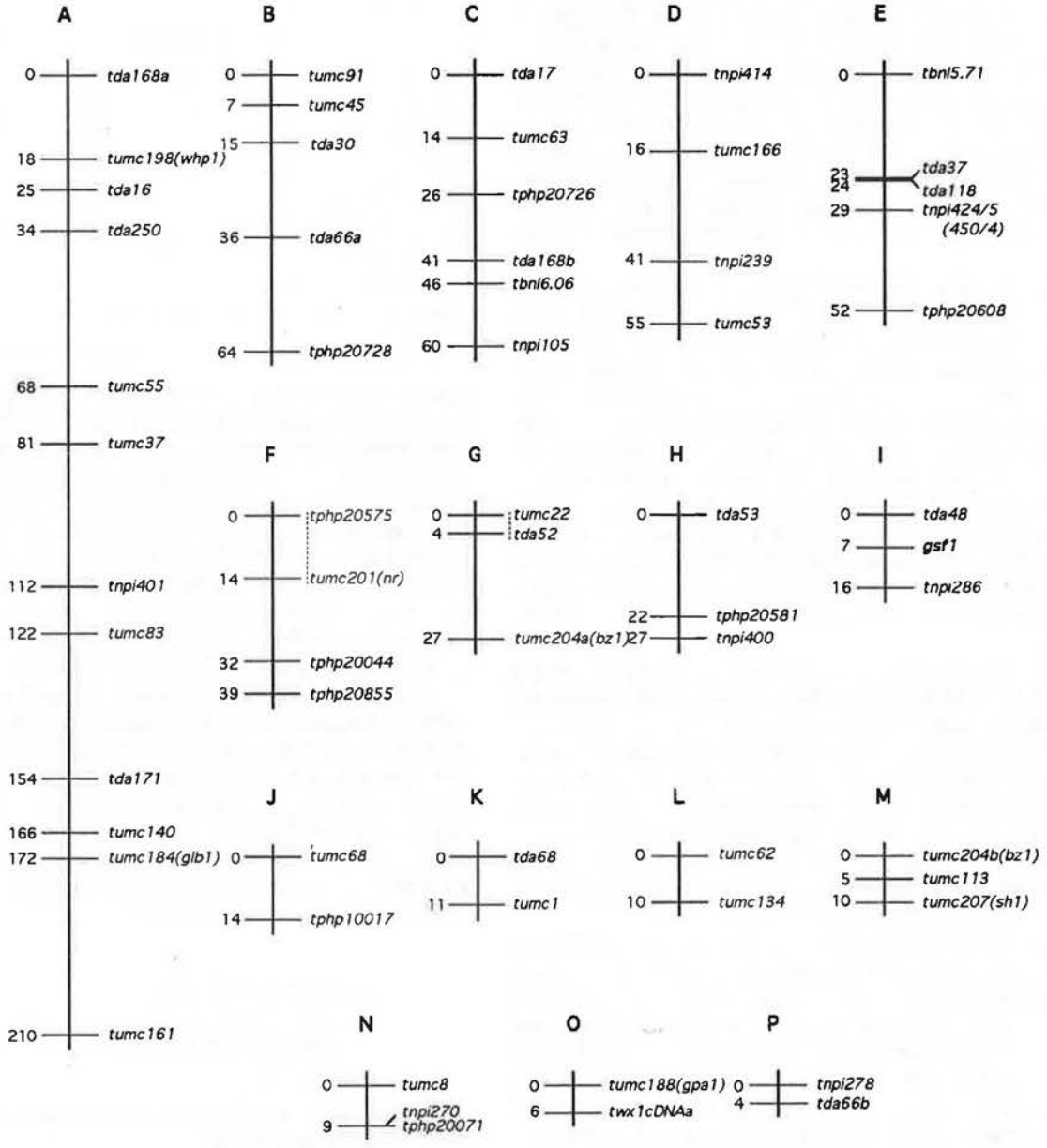
--C.A. Blakey, E.H. Coe, Jr. and C.L. Dewald

***Tripsacum* genetic map.** The restriction fragment length polymorphism (RFLP) genetic map developed from the segregation of

molecular markers in a diploid (2n=36) F2 population of *Tripsacum dactyloides* (common name: Eastern gamagrass) currently stands at 16 linkage groups markers (see Figure) (Blakey, Ph.D Dissertation, 1993; Blakey et al., manuscript in preparation). The map includes 61 loci identified by 57 molecular markers and one phenotypic marker (*gsf1*) (Blakey et al., submitted), for a total genetic length of 609 cM (Group A = longest at 210 cM, Group P = shortest at 4 cM). Complete map data will be available in the Maize Database, upon publication of the RFLP map.

A total of 358 molecular probes have been screened, including 20 functionally defined maize probes, and 197 detected polymorphisms in *T. dactyloides* with one or more enzymes. The polymorphic markers included 65 *T. dactyloides* genomic DNA probes (TDA), 117 maize genomic DNA probes (BNL, NPI, PHP, UMC), 13 functionally defined maize probes, one functionally defined barley probe, and one wheat genomic DNA probe. Over 50 TDA markers and 60 maize RFLP markers that exhibited polymorphism between the *T. dactyloides* parental lines remain to be placed on the existing *Tripsacum* map.

Maize map: All 112 TDA genomic DNA markers screened against the *T. dactyloides* parental lines have also been screened for polymorphisms in the maize lines Tx303 and CO159. Seven TDA markers representing 8 loci in maize (*ztda* loci, for *Zea mays* ssp. *mays* loci identified by TDA genomic DNA probes--see Blakey et al., next article) have been placed on the UMC maize map (MNL 67 and MNL 68). Additional mapping is in progress.



Analysis of *Zea/Tripsacum* genome synteny: Analysis of genomic synteny between *Tripsacum* and maize has revealed regions/blocks of markers that appear to be conserved between these two species from different genera (Blakey, Ph.D Dissertation, 1993; Blakey et al., manuscript in preparation). Every RFLP linkage group of these two species exhibits some degree of synteny, from having a single molecular marker in common to four-marker blocks of conserved order. And, in 9 out of 14 cases, the conserved linkage blocks indicate approximately the same genetic distance (within 10 cM) over the conserved region.

Relevant to linkage group conservation, most of the *T. dactyloides* linkage groups have markers in common with two maize linkage groups. Five *T. dactyloides* groups have markers in common with only one maize group, nine *T. dactyloides* groups have markers in common with two maize groups, and two *T. dactyloides* groups (D and N) each have markers in common with three different maize linkage groups. In general, the particular maize linkage groups in common with a *T. dactyloides* group differ from those maize groups found in common with individual sorghum linkage groups seen by Whitkus et al. (Genetics 132:1119-1130, 1992) and Binelli et al. (TAG 84:10-16, 1992). The exception was *Tripsacum* group E and Whitkus's sorghum group H, both of which have markers from maize groups 4 and 5, and have overlapping or conserved regions in common.

Summary: Through the use of molecular probes derived from both maize and *Tripsacum dactyloides*, a genetic map using molecular markers has been constructed in *T. dactyloides* resulting in 16 linkage groups. The mapped maize-derived markers included loci from all ten maize chromosomes scattered on 16 *Tripsacum* linkage groups with large numbers of loci clustered in fourteen syntenic regions conserved between the two genomes on 12 different *Tripsacum* linkage groups and 8 different maize chromosomes. In general, the recombination distances of these regions in maize were greater than or equal to homoeologous regions in *Tripsacum*, with the exception of *tumc83 - tumc161* (*Tripsacum* group A; homoeologous region on maize chromosome 1) covering a distance of 132 cM in *Tripsacum* and only 55 cM in maize. In comparing the *Tripsacum* linkage groups to the maize molecular and genetic maps, a few of the homoeologous *Tripsacum* chromosomes described by Galinat (1974) were tentatively identified, Tr5, Tr7, and Tr9 (see Table), correlating the cytogenetic, genetic and molecular maps for these chromosomes.

RFLP locus-site designations for interspecific mapping of molecular markers derived from *Tripsacum dactyloides* and maize

--C.A. Blakey, E.H. Coe, Jr. and C.L. Dewald

In constructing a genetic map utilizing multi-species/genus probe sets, it is desirable to designate loci such that the DNA sources of the markers can be readily identified. Over the past several years, it has been our practice to designate the locus-sites for maize-derived molecular markers placed on the *Tripsacum dactyloides* genetic map with the prefix letter "t," as in *tumc1*, to designate the *Tripsacum* locus-site of high homology to a molecular marker (i.e., *umc1*) which identifies a homoeologous locus-site in maize. Reciprocally, *Tripsacum*-derived molecular markers (TDA probe set) are given a prefix letter "z," as in *ztda50*, when mapped in maize (*Zea mays* ssp. *mays*). This creates a four letter locus-site designation that identifies both the DNA source of the molecular marker and the genus-specific locus-site being discussed.

Our justification was that it provides a simplified system that allows for the identification of both the locus and the DNA source of the marker used to identify the locus. This also allows for homoeologous locus identification using a specific molecular marker or cDNA from different species, especially where there may be some degree of lack of exact sequence homology due to the level of hybridization stringency. For example, the *zag1* and *zag2* genes (for *Zea agamous-1* and *-2*) designated by Schmidt et al. (Plant Cell 5:729-737, 1993) were cloned from a maize cDNA library by low stringency hybridization with the *agamous* (*AG*) cDNA from

Table. Molecular, genetic, and cytogenetic correspondence of several maize and *Tripsacum* chromosomes.

Maize Chromosome	Locus	*Tripsacum*[a] Chromosome	*Tripsacum*[b] Group(s)	Molecularly defined region in maize used as evidence for *Tripsacum* group assignment
2S	*ws3, lg1, gl2*	Tr9	D	*ws3* & *lg1*: loci near or in region *npi239 - umc53*
2S	*b1, sk1, fl1*	Tr9	D?	*b1*: proximal to *umc61* at position 60
4S	*su1 - ra3?*	Tr7	F**	*su1* & *ra3*: distal to *umc201(nr)*, proximal to *umc87*
9S	*yg2, c1, sh1, bz1*	Tr5	M	*sh1* & *bz1*: cDNA clones map to group M (*bz1* clone = probe for homolog on 6L)
9S	*wx1*	Tr5	O	maize cDNA for gene mapped in *Tripsacum*

[a] *Tripsacum* chromosome corresponding to the identified maize locus based on the work of Galinat (1974).

[b] *Tripsacum dactyloides* RFLP linkage group.

** 2° map order; ? distance to nearest RFLP greater than 15 cM.

Arabidopsis. In dealing with genomic synteny, the same molecular marker may or may not identify a homoeologous locus region in another species or genus; therefore, each locus in a particular species (or genus) has a given name which identifies the specific locus and the history (source) of the marker used in its identification.

Gynomonoecious sex form1 gene (_gsf1_) of _Tripsacum dactyloides_: Description and _Tripsacum_ linkage map location

--C.L. Dewald, C.A. Blakey and E.H. Coe, Jr.

The gene designated *gynomonoecious sex form1* (*gsf1*) (Blakey et al., submitted) in the homozygous recessive form, in a diploid, confers a high degree of feminization to the otherwise primarily male floral structure of the *Tripsacum* rachis. It was named after the phenotype of a variant of *Tripsacum dactyloides* var. *dactyloides* identified by C. L. Dewald and R. S. Dayton (Crop Sci. 25:715, 1985; Phytologia 57:156, 1985), germplasm accession WW1582 (GSF-I), Woodward, OK. This gene had been previously characterized as a monogenic recessive with the proposed gene name ts^{tr} for *tassel seed-Tripsacum* (Dewald et al., Am. J. Bot. 74:1055-1059, 1987).

The normal (*Gsf1*) phenotype, typical of most *Tripsacum* species, has solitary pistillate spikelets in the lower one-eighth to one-third of the raceme and paired staminate spikelets throughout the remaining portion of the raceme. The recessive mutant phenotype (*gsf1*) has paired pistillate spikelets in the midsection and bisexual spikelets in the terminal portion of the raceme, in addition to the "normal" solitary pistillate spikelets in the lower portion of the raceme.

A diploid (2n=36) F2 population of 113 individuals from the cross WW1582 (*gsf1/gsf1*) X WW1218 (*Gsf1/Gsf1*) was scored for segregation of the mutant phenotype. Phenotypic scoring data were combined with the restriction fragment length polymorphism (RFLP) map data set and linkage relationships were determined using the program MAPMAKER. Two-point analysis revealed linkage of *gsf1* to two genomic DNA probes. The RFLP loci were approximately 7 cM (*tda48*) and 9 cM (*tnpi286*) on either side of the *gsf1* locus on linkage "Group I" of the *Tripsacum* genetic map (Blakey et al., in preparation), where the molecular marker DNA probes were derived from *Tripsacum* (probe: *tda48*) and maize (probe: *npi286*), respectively.

The alteration in floral structure of *gsf1* mutant is morphologically similar to the feminizing effect of the tassel seed mutants of maize (Emerson, J. Hered. 11: 65 - 76, 1920; Nickerson and Dale, Ann. Mo. Bot. Gard. 42: 195-212, 1955; Irish and Nelson, Am. J. Bot. 80: 292-299, 1993). Based on this similarity and molecular evidence of probable synteny between the linkage map region of *Tripsacum* linkage group I and maize chromosome 1 (Blakey et al., submitted; Blakey et al., in preparation), *gsf1* may be homoeologous to the *tassel seed2* (*ts2*) gene of maize.

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Three genes control resistance to wheat streak mosaic virus in the maize inbred Pa405

--M. D. McMullen, M. W. Jones, K. D. Simcox and R. Louie

Wheat streak mosaic virus (WSMV), a mite-transmitted potyvirus, infects certain maize inbreds. We (McMullen and Louie, Mol. Plant-Microbe Interact. 2:309-314) have previously reported on a gene (*Wsm1*) for resistance to WSMV on chromosome 6S of B73 and Pa405. However, segregation ratios of resistant to susceptible plants from a cross involving Pa405 and Oh28 indicated that, in addition to *Wsm1*, other genetic factors controlling symptom resistance to WSMV are present in Pa405. To identify these other genes in Pa405 controlling resistance to WSMV, (Pa405 x Oh28) F2 plants were inoculated with WSMV and symptom responses observed. In addition to resistant (symptomless) plants, two types of symptomatic plants were noted: plants with generalized mosaic (GM) similar to the symptoms observed on the susceptible inbred Oh28, and plants with dispersed, chlorotic spots and rings (DSR). DNAs pooled from 25 plants with GM symptoms and from 25 plants with DSR symptoms were used to detect linkage to RFLP loci by a "bulked segregant" approach. Southern hybridization analysis was performed with DNAs of Pa405, Oh28, F1, GM pool, and DSR pool, all cleaved with three to six restriction enzymes, and hybridized with RFLP probes. This analysis identified two additional genes in Pa405 for resistance to WSMV, designated *Wsm2* (chromosome 3 near *umc102*) and *Wsm3* (chromosome 10 near *umc163*), and reconfirmed the presence of *Wsm1* on chromosome 6S. RFLP analyses of DNA from individual plants revealed that the plants that exhibited GM symptoms were homozygous for Oh28 alleles at the *wsm1, wsm2* and *wsm3* loci. Plants that exhibited DSR symptoms were homozygous for Oh28 alleles at *wsm1* and *wsm2*, but had one or two alleles from Pa405 at the *wsm3* locus.

DEFIANCE, OHIO
Defiance, Ohio

When does paramutation take place?

--Bernard C. Mikula and Beth Besaw

In MNL 1993 I reported that significant, heritable differences in the level of paramutation could be related to controlled conditions in which seedlings were grown for the first two or three weeks before being transplanted to field conditions for maturity. If seedlings were grown for 15 days under 32 C in LL (constant light) before being transplanted to field conditions for maturation, then paramutant *R* expression in the aleurone was essentially colorless. If seedlings were grown for 10 days at 32 C LL then shifted to LD (12 hr. light:12 hr. dark) conditions for days 11-15, then the paramutant *R* gene showed significantly more aleurone cells with dark pigmentation. During this first two weeks of somatic development, in these *R/R-st* heterozygotes undergoing paramutation, what is the functional status of the *R* gene? Is it on or off? The paramutant *R*-gene phenotype, observed in the aleurone of testcross kernels at the end of the life cycle, is expressed as a mosaic of cells in which the gene is on or off. The data reported in MNL, 1993, can be interpreted as a turning off of the *R* gene just prior to or during tassel determination under 32 C and constant light. This "off condition" was accomplished by the 15th day when no tassel primordia were yet visible. If in the last five days of this 15-day period the seedlings were given LD cycles, significantly more pigment could be observed in the testcrosses at maturity. This can be interpreted as meaning the *R* gene was on until turned off by the 32 C LL conditions. Additional evidence which can be interpreted to support this conclusion comes from a

comparison of *R* gene expressions of the pollen sampled from the upper and lower tassel branches of the same plant. If seedlings started at 32 C were switched to 22 C for the last five days of the first 15 days of seedling development, the greatest reduction in paramutated *R*-gene expression occurred in samples from the lower tassel branches. *R*-gene expression of pollen from upper tassel branches of plants which as seedlings received treatments of 22 C or 32 C did not differ. This is interpreted to mean that in 32 C-LL conditions the *R* gene undergoes more repression in pollen tested from the lower tassel branches. The amount of repression, represented in the pigment scores of kernels from testcross ears, is dependent on the number of days the environmental conditions are applied. This raises another question. For an individual plant, is the entire five-day period essential to bring about the high degree of repression or is a five-day period essential simply to ensure that all treated plants will respond equally? To restate the question, for how long must the signal to repress the *R* gene be sustained to achieve a given level of paramutant *R*-gene expression in the treatment of a single meristem? How can this environmental signal, reported in the kernels as degrees of variegation, be accounted for as quantitatively stored "genetic memory" whose phenotype can be incremented in the positive or negative direction? What accounts for the clonal sectoring pattern reported in the aleurone where the *R* gene is highly paramutated and no transposable element has yet been implicated for the *R* locus?

If it is assumed the *R* gene is "on" throughout development, what assumptions must be made about the ear-shoot meristem as well as tiller meristem? Under the 15 days of controlled conditions on which I have reported, these two lower meristems are farther behind in development than the terminal meristem. If as reported, the terminal meristem is sensitive to environmental conditions, then it should not be surprising that the lateral meristems would differ in *R*-gene expression, since under our conditions control of seedling environment is terminated on the 15th day or the 21st day, depending on temperature. The lateral meristems are, subsequently, controlled by field conditions which in early spring vary over a wide range of temperatures; periodicity of light conditions will depend on season and latitude. Where the terminal meristem was initiated under controlled conditions to generate repression of the *R* gene, testcrosses of tillers of these same plants show significantly more pigmented cells than those from the main tassel. That the level of paramutation in the ear is different from the tassel was found in some preliminary evidence where a strong paramutation expression was induced in the terminal meristem. A bimodal distribution of *R*-gene expression was found among the *R/R-lst* heterozygotes which were selfed the previous year. Seeds from the self-pollinated heterozygote could be separated, phenotypically, according to whether the paramutant *R* gene came through the tassel or through the ear. Because of the high degree of paramutation, the *R* gene from the male was nearly colorless and the *R-lst* phenotype in the same kernel, contributed by the female, could be identified. The paramutated *R* gene inherited through the female, because of endosperm dosage, was strongly pigmented. The two classes of seeds from the self-pollinated heterozygote, one showing the *R-lst* phenotype, the other darkly pigmented, were planted. A second round of paramutation with *R-lst* showed the *R* genes from male gametes in the F1 were significantly lighter than those from the female gametes of the same plant. More extensive testing of this phenomenon is planned now that it is possible to influence the terminal meristem unequivocally.

Clonal pattern of pigmented cells in aleurone is host- determined in the second week of seedling development.

--Bernard C. Mikula and Beth Besaw

Highly paramutated *R* gene expression shows up in the aleurone as clonal patterns or sectors of cells similar to pattern alleles *R-st* and *R-mb* . Sector sizes, based on excision patterns for three different transposable elements, were assigned to specific cell divisions in aleurone development (Levy and Walbot, Science 248:1534-1537, 1990). The number of cells per sector can be related to the number of cell divisions that followed the loss of the TE. When in ontogeny is it decided that TE excision will be early or late? If the size of the clonal sectors can be controlled, then the sizes of the paramutated *R* sectors can be used as a reporter system to identify when the developmental program of the host can be set for clonal pattern expression in the aleurone. Under paramutagenic conditions, the sizes of aleurone sectors are determined in the second week of seedling development, under constant light and 32 C or a week later under 22 C conditions. Unlike the *R-st* or *R-lst* alleles responsible for paramutation, the pigmented sectors of cells in the aleurone lack sharp boundaries. Nevertheless the clonal nature of these sectors is evident upon magnification. The aleurone patterning can be considered to have been host-determined, before tassel primordia were visible, by conditions administered to the seedling when the meristem was ready to switch from the vegetative to the reproductive phase of development.

Host-controlled timing of clonal-pattern expression in the third week of seedling development

--Bernard C. Mikula and Beth Besaw

Under paramutagenic conditions for the *R* locus I reported in MNL, 1993, that it was possible to condition near colorless expression of the paramutant *R* gene in testcrosses of plants which as seedlings were held in constant light at 32 C for their first two weeks of development. Seedlings maintained in 22 C for three weeks showed significantly more pigmentation in their testcrosses at maturity. It was possible to bring about the near colorless level of paramutant *R* expression if seedlings which were started for two weeks in 22 C under constant light are shifted to 32 C and constant light in the third week. Figure 1 shows the testcross

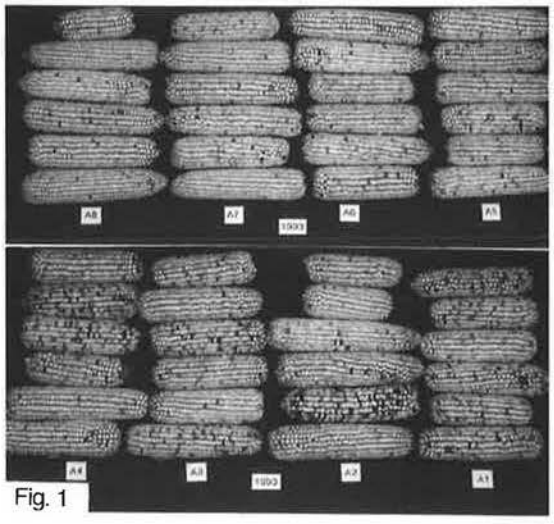


Fig. 1

phenotypes of paramutated *R*-gene expression from *R/R-lst* plants which as seedlings received decreasing numbers of 32 C-LL cycles during the third week of seedling development. On all testcross ears, 50% of the kernels show different degrees of paramutant *R* expressions. Testcrosses labelled A8 through A1 represented plants which as seedlings received successively fewer days of the 32 C treatment. As seen in the lower half of Figure 1, with fewer than five days of 32 C and LL (A4-AI), a greater degree of variation in *R*-gene expression is encountered from ear to ear. Nevertheless, among all the treatments, A4 through A1, testcross ears are found whose *R*-gene expressions are as light as those from the treatments A8 to A5. An interpretation that can account for this heterogeneity is that the seedlings are not all receptive to the signal at the same time because of different stages of development. It could be inferred that a minimum of five days is necessary to have all plants respond to the applied temperature conditions. A corollary to this line of reasoning is that the changeover to the highly repressed state could be accomplished within a day or less if all seedlings were developmentally synchronized. Figure 2 shows, schematically, the treatment schedule for the first 21 days of seedling growth. Plants were kept in constant light for 21 days; beginning on day 13 the length of the line represents the number of days different seedling groups received 32 C-LL conditions. The only change throughout this three-week period was in the number of days seedlings were held in 32 C-LL conditions.

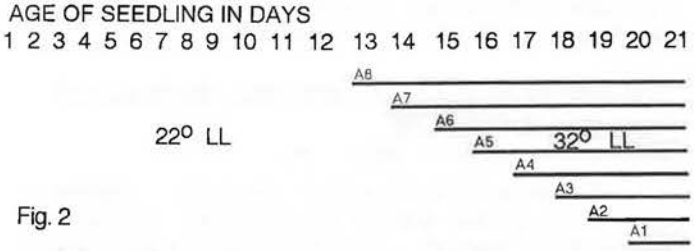


Fig. 2

Figure 3 summarizes the programming strategies which can achieve different levels of paramutation as represented by the pigment scores to the right. Scoring was done as described in MNL, 1993. Unparamutated *R* genes score 18-20. LD (12 hr. light:12hr. dark) conditions administered the third week produced testcrosses with a pooled mean of 13; LL conditions for this same period produced plants at maturity whose testcrosses scored 9. Maximum reduction in pigment was achieved when seedlings were given 32 C for the third week of seedling treatment. For the first two weeks seedlings were held in constant light and 22 C. At the end of 21 days seedlings were moved to field conditions to complete their life cycle.

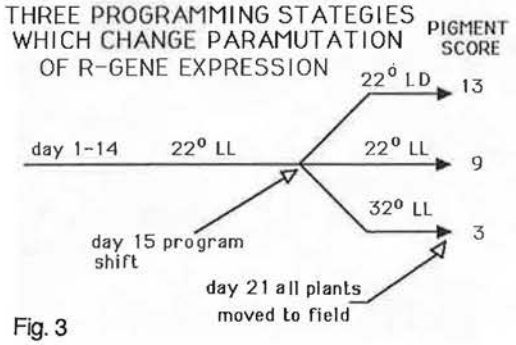


Fig. 3

The significance of paramutation first reported by Brink in 1956 was that in a single generation a heritable change could be made in the expression of a Mendelian gene. **All** the *R*-gene expressions from the heterozygote with *R-st* were changed. A system which provided such a high frequency of responsiveness was an excellent place to see if the 20th Century dogma "the program does not learn from experience" (Jacob) could be challenged. The evidence outlined above and in previous issues of MNL shows that the genetic program learns from its genetic as well as its environmental experience. And, what is more important for a "good learner", the *R* gene has a "good memory" which can be incremented from generation to generation. Since the *R* gene controls a transcriptional activator and is responsive to both light and temperature, this memory capability should have important evolutionary significance for meeting the adaptive challenges demanded by the environment. Information processing, programmed for a specific stage of development, can now be thought of as a part of the regulatory apparatus of the paramutation system. The behavior of *Spm* elements as described by Fedoroff (Genetics 121:591-608 1989) shares some of the characteristics of the paramutation system described above.

DURHAM, NORTH CAROLINA
Duke University

Rootworm Resistance in F1 *Tripsacum* X *Zea diploperennis*
--Mary Eubanks

A bioassay for resistance to western corn rootworm *Diabrotica virgifera* LeConte in F1 progeny of a cross between *Tripsacum dactyloides* (L.) L. and *Zea diploperennis* Iltis, Doebley & Guzman was conducted to determine if rootworm resistance is inherited in offspring of the original hybrid plant designated Tripsacorn (MNL 67:39-40). If resistance occurs in the F1, tests are needed to determine if the trait segregates according to Mendelian inheritance.

Seed was germinated on moist filter paper in petri dishes. Twelve seedlings were then planted in potting soil in 2.25 inch square plastic pots and grown indoors under a 33 watt fluorescent grow light. For infestation, 1,000 non-diapausing western corn rootworm (WCR) eggs in soil were shipped from French Agricultural Research, Inc., Lamberton, MN, to Seeds for the Future, Durham, NC, under USDA permit no. 922762. At 42 days old, when plants had 2 to 4 leaves and were approximately 4 inches tall, 25 newly hatched WCR first instar larvae were transferred to pots using a small paintbrush. Four days later 25 additional larvae were transferred to the twelve pots, giving an infestation total of 50 larvae per pot. Ten days after final infestation, individual plants were immersed in a container of water and roots gently washed for subsequent examination.

There was a range of variation from plants with roots that had been severely chewed and died, to healthy plants with vigorous growth whose roots exhibited almost no larval feeding. The results of this preliminary bioassay to screen for rootworm resistance in progeny indicate a gene for rootworm resistance that has a dosage effect is inherited from *Tripsacum*. Presumably, plants with severely chewed roots that died were homozygous susceptible; plants intermediate in root feeding and growth were heterozygous, and healthy plants with minimal root feeding were homozygous resistant. The ability to use homozygous resistant

plants for crossing would greatly facilitate a breeding program to introduce rootworm resistance into maize via the Tripsacorn bridge species. An experiment to determine if results of laboratory bioassays can be replicated in greenhouse infestations is underway.

EUGENE, OREGON
University of Oregon

Isolation and genomic mapping of cDNAs encoding the chloroplast Rieske Fe/S protein: two unlinked genes are transcribed in maize

--Alice Barkan and Macie Walker

The chloroplast cytochrome f/b6 complex contains a single nuclear-encoded subunit, the Rieske Fe/S protein. cDNAs encoding this protein were isolated by using a monospecific antiserum to screen a cDNA expression library prepared from B73 seedling leaf RNA. Partial sequence data indicated that two classes of cDNA were obtained. The two classes are over 95% identical in the protein coding region that has been sequenced. They diverge more extensively in the 3'-untranslated region.

In high stringency Southerns (0.2 X SSC, 65 C washes) the cDNAs hybridized to two bands in DNA from the inbred lines B73, CM37, T232, Tx303 or CO159 that had been digested with each of several different enzymes . The hybridizing DNA fragments were mapped using the Burr T x CM and CO x Tx recombinant inbred populations. The two bands were unlinked. The two genes they correspond to were named *ris1* and *ris2*. They mapped to the following locations:

ris1: maps to chromosome 5, within 2 cM of *bt1*.
ris2: maps to chromosome 4L, 9 cM distal to *c2*.

These results suggest that there are two closely related genes encoding the chloroplast Rieske Fe/S protein in maize, and that both genes are transcribed in leaf tissue. It seems unlikely that mutation of either gene alone would result in the loss of the cytochrome f/b6 complex. Therefore, the numerous nuclear mutations in maize that lead to the loss of the cytochrome f/b6 complex are unlikely to lie in a gene encoding the Rieske protein.

A cDNA encoding the mitochondrially localized Rieske Fe/S protein in maize was reported by Huang et al. (Proc. Natl. Acad. Sci., 1991). This cDNA is no more than 40% similar to the cDNAs encoding the chloroplast-localized protein, even in the most conserved portions of their protein coding region. Therefore the genes for the mitochondrial and chloroplast Rieske Fe/S proteins are distinct.

Mapping and allelism results: nuclear mutations affecting chloroplast biogenesis

--Alice Barkan, Rodger Voelker, Melanie Johnson, Lisa Cipolla and Laura Roy

***crp1* maps to chromosome 7L.** *crp1* is a nuclear mutation that causes the loss of the cytochrome f/b6 complex and a decrease in photosystem I. These protein losses are due to a defect in the processing and translation of specific chloroplast mRNAs (manuscript in preparation). This mutation was unmasked in crosses with TB-7Lb. Therefore, it maps to the long arm of chromosome 7.

***crp1* is allelic to *hcf111*.** *hcf111*, a mutation isolated by Cook and Miles (MNL63:65-66), and *crp1* map to the same chromosome arm (7L). Like *crp1*, *hcf111* is also deficient in the cytochrome f/b6 complex. We have determined that *hcf111* shares the defect in chloroplast RNA processing previously seen only in *crp1*. Because the map position and phenotypes of the two mutations were similar, it seemed likely that they represented two alleles of the same gene. In complementation tests, six different crosses between pairs of heterozygous plants each yielded one-quarter mutant progeny. Therefore, *crp1* and *hcf111* do not complement and are most likely allelic.

***cps1* maps to chromosome 1L.** *cps1* is a nuclear gene that is required for chloroplast protein synthesis (A. Barkan, Plant Cell 5:389-402). Crosses of five *cps1*/+ plants by TB-1La pollen yielded pale green seedlings with hypoploid morphology and lacking Rubisco. Therefore, *cps1* maps to the long arm of chromosome 1.

***hcf6* is allelic to *pet3-1* and *pet3-2*.** *pet3-1* and *pet3-2* are two independent alleles of a gene, *pet3*, that is required specifically for the accumulation of the chloroplast cytochrome f/b6 complex. Both alleles were obtained from *Mutator* lines and both block the accumulation of the cytochrome f/b6 complex at a post-translational step (Voelker and Barkan, in preparation). Complementation tests were performed between these mutations and *hcf6*, an EMS-induced mutation isolated by Don Miles (University of Missouri) that also lacks the cytochrome f/b6 complex. Three crosses between plants heterozygous for *pet3-1* and *hcf6* and one cross between plants heterozygous for *pet3-2* and *hcf6* each yielded one quarter mutant progeny. Therefore, *pet3* and *hcf6* mutations do not complement and are most likely allelic.

GAINESVILLE, FLORIDA
University of Florida

A summary of the chromatin structure and other architectural features of the maize *Adh1* 5' flanking region

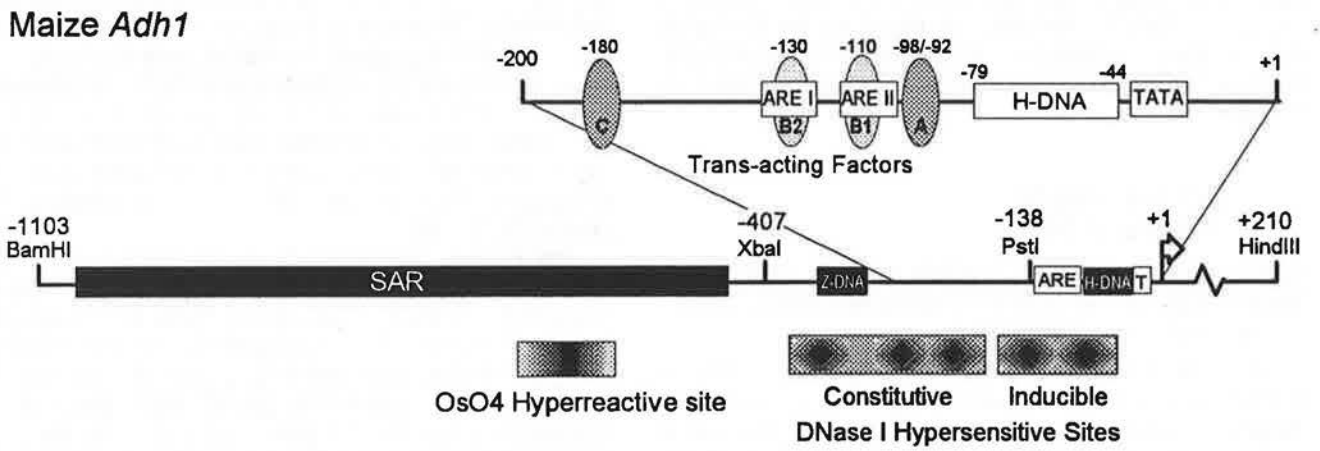
--Anna-Lisa Paul and Robert J. Ferl

The chromatin structure of the *Adh1* promoter influences and is influenced by transcriptional activation. There are components that are constitutively present, as well as those that are apparent only when the gene is active. Figure 1 (next page) summarizes our current understanding of the chromatin structure of the maize *Adh1* promoter, together with the appropriate references.

Brain protein homologs and gene names

--Robert Ferl, Nick deVetten, Guihua Lu, Paul Sehnke, Christine Daugherty, Beth Laughner and Ke Wu

There have been several reports of cloned plant sequences with homology to the 14-3-3 class of mammalian brain proteins (Brandt et al., Plant J 2:815-820,1992; Hirsch et al., FEBS Lett 296:222-224, 1992; Keith et al., Plant Physiol 101:329-332, 1993; Kidou et al., Plant Mol Biol 21:191-194, 1993). Our own work has focussed on the potential involvement of these proteins in the DNA binding complex that is associated with the G-box and its regulatory properties (Lu et al., PNAS 89:11490-11494, 1992; deVetten et al., The Plant Cell 4:1295-1307, 1992), but work from other laboratories indicates that the potential activities and responses involving 14-3-3 brain protein homologs are far-ranging. Most of the known biochemical activities involve regulation of protein kinase mediated events, and our current model is that their participation in the G-box complex is within their role as moderators of signal transduction via phosphorylation. We have recently



SAR: Scaffold Attachment Region. The SAR lies between the *Xba*I site and the *Bam*HI site. A likely point of attachment is within the OsO_4 hyperreactive site centered around -589. Avramova and Bennetzen, PMB 22:1135, 1993; Paul and Ferl, PMB 22:1145, 1993.

Z-DNA: A tract of alternating purines and pyrimidines between -325 and -311 that assumes a Z-DNA configuration under superhelical stress in vitro. Ferl and Paul, PMB 18:1181, 1992.

H-DNA: A tract of extreme homopurine/homopyrimidine (PuPy) asymmetry between -79 and -44 that assumes an H-DNA configuration under superhelical stress in vitro. The region is also capable of forming a triple helix in vitro, and has been shown to play a role in *Adh1*-GUS expression in vivo. Ferl et al., PMB 8:299, 1987; Lu and Ferl, PMB 19:715, 1992.

DNase I: There are two sets of DNase I hypersensitive sites in the *Adh1* promoter. There are three major constitutively present sites that lie between -400 and -160 and two inducible sites located between -150 and -35. Paul et al., PNAS 84:799, 1987.

Trans-factors: The functionally defined cis-regulatory anaerobic response element in *Adh1* (ARE; *Walker et al., 1987, PNAS 84:6624*) is associated with trans-acting DNA binding factors in vivo. There are two types of DNA binding factors. One set (B1 and B2) is constitutively bound to the ARE between -133 and -124 (ARE I) and between -113 and -99 (ARE II). The second set of factors (C and A) bind outside the ARE, in response to hypoxic stress, at positions centered around -180 and -95. Ferl and Nick, JBC 262:7947, 1987; Paul and Ferl, Plant Cell 3:159, 1991.

cloned, mapped, sequenced and characterized several genomic clones from maize and are therefore faced with the necessity of providing a gene name for this family of proteins.

The maize proteins and cDNAs that we have worked with to date have been called GF14, for *G*-box *F*actor *14*-3-3 homolog. Particular isoforms of the proteins have been given either Greek letter designations to follow the lead of the animal literature or simpler laboratory designations. Thus, for example, cDNAs and proteins are currently referred to as GF14ω or GF14χ. In order to meet accepted standards for gene names, we propose to use *Grf* for *G*-box *r*egulatory *f*actor to refer to the genomic clones and their loci, and will designate the genes as *Grf1*, *Grf2*, etc as they are cloned and mapped. In order to maintain contact with the previous literature and with the animal literature, the allele designations will retain the original cDNA and protein name, such as *Grf1*-GF14ω. Another aspect of the *Grf* gene name is that if this family of proteins becomes regarded as having much wider regulatory roles, the gene name could be modified to *G*eneral *r*egulatory *f*actor to accomodate additional perspectives.

GIF/YVETTE, FRANCE
INRA/UPS/CNRS

A composite map of expressed sequences, based on four individual maps.

--Mathilde Causse, Catherine Damerval, Alexandrine Maurice, Alain Charcosset, Sylvain Santoni and Dominique de Vienne

Candidate gene approach is a straightforward way to identify QTLs and to increase the efficiency of marker assisted selection. For this purpose, we are constructing a maize genetic map mainly based on expressed sequences. Our map relies on the data from 4 segregating populations. Three recombinant inbred line (RIL) populations were derived from the three possible crosses between 3 inbred lines, an early flint (F2), an Iodent (coded Io) and an early dent line (F252). A map derived from an independent F_2 progeny from the cross Io x F2 was also used. Each progeny contains between 100 and 150 genotypes, and a total of more than 500 individuals were genotyped. With such sample size, a good confidence in gene order is expected. Around 75% of the tested probes appeared polymorphic in each cross (among more than 200 probes). Four sources of markers were used, and the present map is composed of: - 60 loci corresponding to known function genes obtained from laboratories which cloned them (Table1); - 39 loci (coded PSL) controlling position shifts of proteins revealed by two-dimensional electrophoresis in our laboratory (submitted); - 27 loci (coded SC) of sequenced cDNA for which no homology was found in gene banks, kindly provided by C. Baysdorfer (California State Univ.); - 98 loci of anonymous probes (coded umc and bnl) of the maize core map (Gardiner et al., Genetics 134:917-930, 1993). These markers were useful to integrate our map with the other maize maps.

Mapping cDNA revealed some problems, among which is the high frequency of multiple copy probes. Among the mutiple bands, it is rare that more than one locus per progeny could be mapped. Working with 4 populations and 2 restriction enzymes sometimes allowed maping of a higher number of loci. Depending on the region of the gene used (3' end versus 5' end) we could also reveal different patterns and map additional loci for the *Sh2* gene. The development of locus specific probes would be necessary for many known function cDNAs. No specific organization was deduced, except the duplications already mentioned by Helentjaris et al. (Genetics 118:356-363, 1988). Depending on the population, segregation distortions concerned between 4% and 12% of the probes ($p<0.01$)

Individual maps were first constructed using Mapmaker V3.0

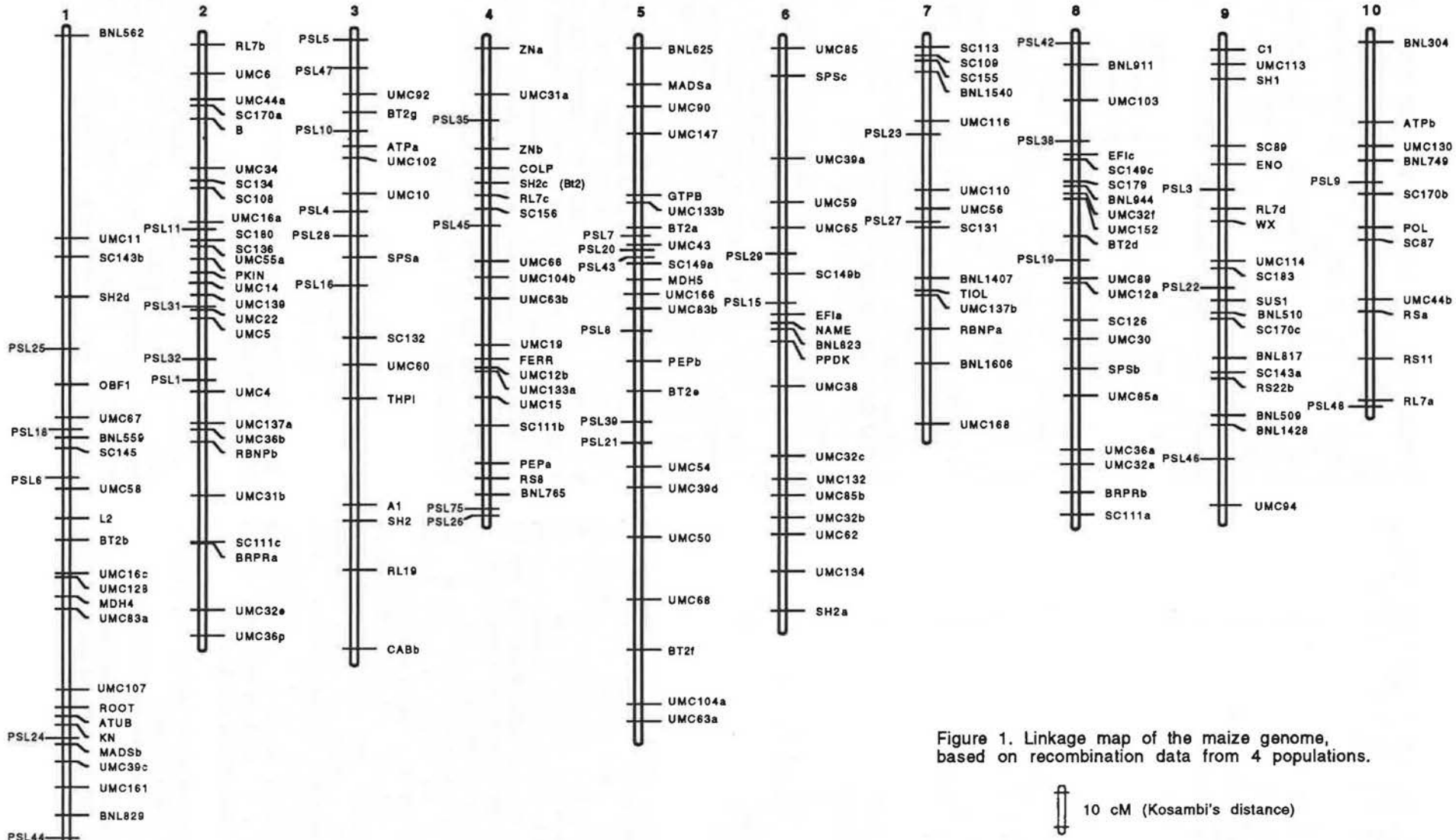


Figure 1. Linkage map of the maize genome, based on recombination data from 4 populations.

10 cM (Kosambi's distance)

Table 1. Chromosomal location of known function genes mapped on the composite map. Probes whose function has been deduced from sequence homology are indicated with an asterisk. The percentage of homology is in parenthesis, with the corresponding organism (ZM: maize; P: other plant; A: animal; Y: yeast; B: bactery).

Code	Function	Chromosome
A1	A1, anthocyanin metabolism	3
ATP*	ATP/ADP translocator (100% ZM)	3,1
ATUB1	a tubulin 1	1
B	B, anthocyanin metabolism regulator	2
BRPR*	brain specific 14-3-3 protein (70% A)	2,8
BT2	brittle2 (ADPG pyrophosphorylase, endosperm)	6
C1	anthocyanin metabolism	9
CAB*	chlorophyll a/b binding protein (99% ZM)	3
COLP	cold induced protein	4
EFI*	elongation factor I a (100% P)	6,8
ENO*	enolase (99% ZM)	9
FERR	ferritin	4
GTPB*	GTP binding protein (79% A)	5
KN	knotted, transcription factor	1
L2	ADPG pyrophosphorylase, leaf	1
MADS*	MADS box (62% P)	1,5
MDH*	malate dehydrogenase (68% B)	1,5
NAME*	NADP malic enzyme (100% ZM)	6
OBF1	OCSBF-1, transcription factor	1
PEPC	phosphoenol pyruvate carboxylase	4,5
PKIN	protein kinase	2
POL	pollen specific cDNA	10
PPDK*	pyruvate phosphate dikinase (100% ZM)	6
RBNP*	31 KD ribonucleic protein (60% P)	2,7
RL19*	ribosomal protein L19 (71% A)	3
RL7	ribosomal protein L7	2,4,10
ROOT	root specific cDNA	1
RS	R-S, anthocyanin metabolism	10
RS11	ribosomal protein S11	10
RS22*	ribosomal protein S22 (76% A)	9
RS8*	ribosomal protein S8 (70% A)	4
SH1	shrunken1 (sucrose synthase)	9
SH2	shrunken2 (ADPG pyrophosphorylase, albumen) 3' end	3
SH2	shrunken2 (ADPG pyrophosphorylase, albumen) 5' end	1,4
SPS	sucrose phosphate synthase	3,6,8
SUS1	SuS1 (sucrose synthase)	9
THPI*	thiol protease inhibitor (73% P)	3
TIOL*	thiol protease (63% P)	7
WX	waxy	9
ZN	zein	4,4

software. A few differences with the core map were detected in the locus position, usually in regard to multiple copy probes. As many loci were common to all maps, we checked for heterogeneity between recombination fractions. The comparison of recombination fractions following the procedure of Beavis et al. (Theor. Appl. Genet. 82:636-644, 1991) procedure revealed: (i) a very good correspondence of the recombination fractions between the F2 and the RIL progeny derived from the same cross; (ii) few significant differences in interval distances between the 3 RIL populations; and (iii) global differences, which can reach 20% of the total map length (when the same subset of loci is mapped). The consistency of probe order over the progenies was confirmed. A composite map has thus been constructed using JoinMap software (Fig. 1). With a total of 233 loci, we approximately cover 90% of the maize genome (when compared with the most recent MNL compilation). The mapping effort is continuing and we would enjoy mapping any known function gene, newly cloned, on our material. The three RIL populations are involved in various QTL location projects (see companion papers), which should lead to a large data set interesting both for maize breeders and geneticists.

Investigation of the effect of genetic background on QTL expression using three connected RIL populations

--Alain Charcosset, Mathilde Causse and André Gallais

A set of maize recombinant inbred lines (RIL) has been developed to investigate the genetics of several quantitative traits. Three parental lines were chosen to provide a good representation of the germplasm that can be used in the north of France. F2 is an early flint line derived from the French population Lacaune, F252 is an early dent line developed from US and Canadian germplasm (F186*Co125), Io is a later dent line from the Iodent group. The three possible hybrids between these three lines have been selfed. Resulting F_2 populations have then undergone a classical single seed descent process yielding F_5 lines. 145, 129 and 152 lines were developed for populations Io*F2, F252*F2 and Io*F252, respectively. These three populations will be called respectively D, E and G in the following text.

The lines were analyzed for their RFLP and a synthetic map was built (see companion paper). Field data were observed on F_6 families in years 1992 and 1993. Earliness data (days to silking) were recorded in three environments in 1992 and one in 1993. QTL analysis (ANOVA and Mapmaker QTL) was performed for each population. Results appeared to be very dependent on the population. In population D, 5 chromosomal regions showed significant effects (1% alpha level) in the two northern environments. This number was three and one for populations G and E, respectively. The number of detected QTLs appeared to be positively related to the difference in silking time of the parents. Most important effects were detected for population D, near probe *umc67* (chr. 1) with a substitution effect of 3.6 days (15% of the variation explained), and near probe *umc103* (chr. 8) with a substitution effect of 4.5 days (17% of the variation explained).

Two chromosomal segments were clearly common to populations D and G. However, consistently with the results of Beavis et al. (Theor. Appl. Genet. 83:141-145, 1991), other segments were involved in the variation of a single population. For instance, a QTL near *umc67* was observed in population D and not in the two other populations. Several causes can be evoked to explain such a result: (i) the power of the statistical tests, (ii) allelic relationships between parents, and (iii) epistatic effects. A simultaneous analysis of variance was performed for the three populations (for loci that were polymorphic in the three populations). Significant (alpha 5% level) interaction effects between marker and population were observed for 18% of the tests, which suggests that epistatic effects play a role in the differences that we observed.

Further analyses will be carried out on the hybrids between the RILs and the three parental lines to investigate dominance effects for several traits. Selfing of the RILs is underway to develop F_7 lines that will be available for cooperations.

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Locating QTLs for carbon metabolism and early growth, using candidate gene approach

--Mathilde Causse and Jean Paul Rocher

Genetic determinism of physiological and early growth traits in maize has been studied in a population of recombinant inbred lines using the QTL/RFLP methodology. Sixty five F6 lines, derived from the cross between F2, an early flint line and an Iodent line, were grown in controlled conditions in a greenhouse, until the third expanded leaf stage. Growth measures concerned leaf size,

growth duration and dry matter weight. The carbon metabolism was characterized by the concentrations of carbohydrates and the activities of four key-enzymes : sucrose phosphate synthase (SPS, which regulates sucrose synthesis), ADP-glucose pyrophosphorylase (AGPase, which regulates starch synthesis), and invertase and sucrose synthase (INV and SuS, which both hydrolyze sucrose in sink organs). Differences between parental mean values, and a wide range of variation in RILs have been found for growth as well as for the physiological traits. Strong correlations were found between growth traits and invertase activity, which reflects sink organ strength.

The population has been genotyped for more than 100 RFLP marker loci and a genetic map was constructed (see companion paper). QTLs, located on thirteen chromosomal regions, were detected (by one-way ANOVAs, $p<0.01$) for every trait. Between one and four QTLs were detected for every trait, with R^2 values (determination coefficient) between 0.07 and 0.35. Each chromosomal region frequently concerned more than one trait, and common locations of QTLs for growth traits and activity of enzymes was observed in 3 of the 13 regions (in 8 of these 13 regions when decreasing the probability threshold to 0.05). For instance, a segment on chromosome 8 exhibited QTLs for invertase activity (with $R^2=0.35$) and dry matter weight (with $R^2=0.20$). QTLs common to these traits also appeared on chromosome 10. On chromosome 9, a region was found where QTLs were detected for growth duration until the 3rd expanded leaf stage, SuS and AGPase activities. These common locations possibly reflect the impact of the physiological traits on growth characteristics.

We mapped loci corresponding to the structural genes of 3 of the 4 studied enzymes. Some of the genes coding for the key-enzymes were located close to or at the most likely position of the QTL for the activity of the enzyme. This emphasizes the role of these candidate genes in physiological processes. For AGPase, the gene *L2* coding for the enzyme form expressed in leaves (cloned by Prioul et al, Plant Physiol., in press), unmapped until now, mapped on chromosome 1, near *umc58*. Hybridization with *Sh2* and *Bt2* clones, the two isoforms expressed in endosperm, revealed homology with 7 other loci, 3 with *Bt2* and 4 with *Sh2*. The only QTL detected for AGPase activity did not map near one of these loci. For the sucrose synthase, the two genes *Sh1* and *Css1* mapped as expected on chromosome 9. A QTL for the activity of this enzyme was found in the *Sh1* region, suggesting a possible involvement of this gene in the expression of its activity. For SPS, three loci were mapped on chromosomes 3, 6 and 8. A QTL for the SPS activity was found near the QTL on chromosome 8. The role of an allelic variation at these candidate loci in the activity of their enzyme still remains to be proven, as confidence intervals of QTL location are very large. Mapping the invertase gene would be also of a great interest. Finally, the carbohydrate enzyme loci were found to be involved in epistatic interactions more frequently than anonymous loci, suggesting their implication in regulation networks.

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Associations among inbred lines of maize using RFLP and DNA amplification technologies (AFLP and AP-PCR), and correlations with pedigree, F1 yield and heterosis

--Stephen Smith, Stella Luk, Bruno Sobral, Salah Muhawish, Johann Peleman and Marc Zabeau

Thirty-five and thirty-six of the thirty-seven inbred lines that previously have been reported upon (Smith et al., Theor. Appl. Genet. 80:833-840, 1990) for pedigree, F1 yield, heterosis and RFLP data were profiled using two DNA amplification procedures. These were Amplified Fragment Length Polymorphisms (AFLPs) or Selective Restriction Fragment Amplification (Zabeau and Vos, European Patent Application No. 0 534 858 A1, 1993) and Arbitrarily Primed Polymerase Chain Reaction (AP-PCR), (Welsh and McClelland, Nucl. Acids Res. 18:7213-7218, 1990).

Twenty AFLP primers were used to score 347 bands. Forty primers were used in AP-PCR and 258 bands were scored. Correlations for pairwise distances between inbreds from AFLP data and other data were $r = 0.91$ (F1 yield), $r = 0.84$ (heterosis), $r = 0.90$ (pedigree), $r = 0.91$ (RFLP) and $r = 0.88$ (AP-PCR). Correlations for pairwise distances between inbreds from AP-PCR data and other data were $r = 0.92$ (F1 yield), $r = 0.84$ (heterosis), $r = 0.85$ (pedigree) and $r = 0.85$ (RFLPs). As would be expected from the correlation data, cluster analyses of inbred lines using distance data from AFLP and AP-PCR resulted in associations of inbreds that were in close agreement with those generated from RFLP and pedigree distance (1 - Malecot Coefficient of Similarity) data.

AP-PCR and AFLP are different methods to sample DNA sequence diversity and they possibly differ in the regions of the genome that are targeted by RFLP probes made using methylation sensitive enzymes. Nevertheless, these data show that different perspectives on inbreds that have degrees of relatedness usually encountered in breeding programs are in concurrence. Therefore, data from these arbitrary primer DNA amplification methods further support that molecular marker data can provide useful information on inbred identities and relationships for the support of plant breeding. It will not be necessary to generate data from several marker technologies; technological choice can be made as suits the issues and circumstances (Ragot and Hoisington, Theor. Appl. Genet. 86:975-984, 1993).

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Distribution of carotenoids and *Y1* mRNA in maize kernels

--Brent Buckner and Diane Janick-Buckner

Yellow-kerneled maize is known to contain carotenoids, while white-kerneled maize lines are thought to contain little if any carotenoids. The plant hormone abscisic acid (ABA), which is involved in maintaining kernel dormancy, is derived from carotenoids. Therefore, if white kernels contain no carotenoid biosynthetic ac-

tivity the kernels would not contain ABA and, subsequently, would be expected to be viviparous. Viviparous mutants of maize are known, however, kernels that are white as a result of being homozygous for a recessive allele of *Y1* are not viviparous. Therefore, we have investigated the distribution of carotenoids and *Y1* mRNA in endosperm and embryos that are homozygous for dominant or recessive alleles of *Y1*.

The standard *Y1* line used in these experiments was a hybrid of inbred lines Q66 and Q67. The *y1* standard line used was derived from a heterozygous translocation T6-9e stock obtained from the Maize Genetics Cooperation in 1961 (stock no. 57-413-2) by D.S. Robertson. To analyze the distribution of carotenoids in the maize kernel we extracted total carotenoids separately from endosperms and embryos. The carotenoids were separated and quantified by using high pressure liquid chromatography. The major carotenoids found in the kernels were zeaxanthin/lutein, a xanthophyll monoester, β-cryptoxanthin, α-carotene and β-carotene. The types and amounts of carotenoids were not significantly different in the embryos of kernels which were homozygous for either the dominant or recessive allele of *Y1* (Table 1). The types of carotenoids present in the endosperm of kernels homozygous for the dominant or recessive allele of *Y1* were the same, however, they were found in significantly lower quantities in the endosperm of homozygous recessive kernels (Table 1).

Table 1. Distribution of carotenoids in maize kernels[a].

	Lutein/zeaxanthin µg/g	β-cryptoxanthin µg/g	β-carotene µg/g	α-carotene µg/g[b]
Y1 Y1 embryo	16.54 ± 3.20	3.86 ± 1.14	2.07 ± 0.43	0.55 ± 0.24
y1 y1 embryo	12.65 ± 1.14	2.80 ± 0.80	2.72 ± 1.03	0.86 ± 0.29
	N.S.	N.S.	N.S.	N.S.[c]
Y1 Y1 Y1 endosperm	10.00 ± 0.59	0.54 ± 0.07	2.02 ± 0.07	0.33 ± 0.07
y1 y1 y1 endosperm	0.62 ± 0.32	0.23 ± 0.03	0.29 ± 0.06	0.08 ± 0.02
	p<0.001	p<0.001	p<0.001	p<0.01[c]

[a]Each value represents the mean ± standard deviation of embryo or endosperm samples from three separate ears measured in triplicate, with the exception of the *Y1 Y1* embryos, which were done in duplicate.

[b]Data are expressed as µg carotenoid per gram of wet tissue.

[c]Values were compared by using an unpaired t test. Samples were not considered to be significantly different (N.S.) if the calculated p value was greater than 0.05.

The *Y1* gene codes for phytoene synthase, the enzyme that converts two molecules of geranylgeranyl pyrophosphate to phytoene, the first C_{40} carotenoid. RNA blot hybridization analyses were performed to investigate the expression of the *Y1* gene. Rehybridization of the RNA blots used in this study with a human actin hybridization probe indicated that all samples contained RNA. Densitometric analysis of the RNA blot hybridization autoradiographs allowed us to compare the amount of *Y1* mRNA present in each tissue. Embryos isolated 30 days after pollination (DAP) from kernels which were homozygous for the dominant or recessive allele of *Y1* both contain *Y1* mRNA, however, the *Y1* mRNA in the *Y1 Y1* embryo was approximately 6 times more abundant. A *Y1* mRNA transcript was detected in endosperm isolated from 30 DAP kernels which were homozygous for the dominant allele of *Y1*, however, no transcript was detected in 30 DAP endosperm which was homozygous for the recessive allele of *Y1*.

There was no significant difference in the amount of carotenoids detected in the embryo of kernels homozygous for either the dominant or recessive alleles of *Y1*. However, approximately 6 times more *Y1* mRNA was detected in embryos that were homozygous for the dominant allele of *Y1*. There are several hypotheses that might account for this observation. One possibility is that the quantity of carotenoids in 30 DAP embryos is reflective of a peak amount of *Y1* gene expression that occurred prior to 30 DAP. An analysis of the expression of the *Y1* gene during the development of kernels may indicate if this is the case. Alternatively, if the biosynthetic steps subsequent to phytoene synthesis, but prior to the synthesis of the carotenoids analyzed in this study, are rate limiting, then phytoene may accumulate in the embryos of both genotypes. If this were true, then the lower level of *Y1* mRNA present in the embryos that are homozygous for the recessive allele of *Y1* might be sufficient to produce the level of colored carotenoids measured in this study. Analysis of phytoene and all subsequent carotenoids in the embryo of both genotypes should address this possibility.

Carotenoids were detected in 30 DAP endosperm of plants that were homozygous for the standard recessive allele of *Y1*, however, no *Y1* mRNA was detected in this tissue. The inability to detect *Y1* mRNA in this tissue may be due to the *Y1* gene being expressed in this tissue at an earlier time or due to the expression of the *Y1* gene below the level of sensitivity afforded by RNA blot hybridization analysis. Using a reverse transcriptase and polymerase chain reaction assay for expression should indicate if and when the recessive allele of *Y1* is transcribed in the endosperm of plants which are homozygous for the recessive *Y1* allele.

Alternatively, if maize has more than one phytoene synthase gene, the additional locus (loci) may be expressed in endosperm of plants that are homozygous for the recessive allele of *Y1* and could thereby be partly responsible for the presence of carotenoids at the low levels found in this tissue. Additional phytoene synthase loci would also explain why no albino or viviparous alleles of *Y1* have been described. DNA blot hybridization analysis using the *Y1* gene as a hybridization probe and high stringency hybridization and wash conditions often exhibits DNA fragments in addition to those expected from restriction endonuclease map and sequence data of the cloned *Y1* gene. These additional DNA fragments do not hybridize to the same extent as the *Y1* sequences and might represent other loci that are members of a phytoene synthase gene family. Transcription of multiple phytoene synthase loci may not have been detected in this study if transcripts are present at a concentration below the level of detection afforded by RNA blot hybridization analyses or if the sequence of the non-*Y1* phytoene synthase loci has diverged significantly from the *Y1* sequence.

Carotenoid content in the endosperm of pale yellow and white kernels that are homozygous for a recessive allele of *Y1*

--Brent Buckner, Lian A. Bonds and Diane Janick-Buckner

It is commonly observed that kernels homozygous for a recessive allele of *Y1* can be white to pale-yellow in color. This variable expressivity is usually associated with the genetic background of the plant. In some of our stocks we have observed F2 ratios of 12 yellow: 3 pale yellow: 1 white kernels in self pollinations of plants heterozygous for *Y1*. Vivipary has not been observed in association with the gene(s) responsible for pale yellow color in these crosses. The purpose of this study was to determine whether the pigments responsible for the pale yellow phenotype were carotenoids. Therefore, we extracted total carotenoids from endosperms of yellow, pale yellow and white kernels and separated and quantified them by using high pressure liquid chromatography. The major carotenoids found in all kernels were zeaxanthin/lutein, a xanthophyll monoester, β-cryptoxanthin, α-carotene and β-

carotene. We found that there was a significant difference in the carotenoid levels of pale yellow and white kernels for the lutein/zeaxanthin, α-carotene, β-carotene and total carotenoid content (Table 1).

Table 1. Carotenoid content in the endosperm of pale yellow and white kernels that are homozygous for a recessive allele of *Y1*[a]

	White	Pale yellow[b]	
Lutein/zeaxanthin	100	112 ± 5	p≤0.05[c]
β-cryptoxanthin	100	100 ± 4	N.S.
Lutein/zeaxanthin monoester	100	100 ± 2	N.S.
α-carotene	100	132 ± 17	p≤0.05
β-carotene	100	130 ± 6	p≤0.01
Total carotenoids	100	109 ± 2	p≤0.05

[a]Data are expressed as the percentage of carotenoids present in white endosperm.

[b]Each value represents the mean ± standard error of the mean of endosperm samples from three separate ears measured in triplicate.

[c]Values were compared by using a paired t test. Samples were not considered to be significantly different (N.S.) if the calculated p value was greater than 0.05.

The allele of the gene responsible for the pale yellow phenotype in the kernels analyzed was contributed from a hybrid of inbred lines M14 and W22. Donald S. Robertson has informed us that in his experience stocks which are homozygous for a recessive allele of *Y1* are pale yellow in an M14 background and a "truer white" in W22 background. Therefore, it is likely that the allele of the gene responsible for the pale yellow color is present in the M14 line.

The identity of the gene responsible for pale yellow kernel color in the kernels analyzed in this study is not known. There are several genes such as *Y6*, *Y8*, *Y11*, and *Y12* which, when present in a homozygous recessive condition, do not result in vivipary, however, they do influence the intensity of yellow pigmentation in the kernels of plants that have a standard dominant allele of *Y1*. Plants that are homozygous for the recessive alleles of these genes have decreased yellow pigmentation in their endosperm, presumably due to decreased levels of carotenoids, even when the plants contain a dominant *Y1* allele. Other alleles of these genes might be expected to increase the quantity of carotenoids found in the endosperm of maize kernels. Therefore, if these loci, or others with similar effects on carotenoid biosynthesis, segregate independently from the *Y1* locus, epistatic ratios such as those described in this study would be expected.

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Transgressive segregation in the progeny of a cross between two inducers of maize maternal haploids

--S. T. Chalyk, V. G. Bylich and O D. Chebotar

The production of new inducers of maternal haploids is of some importance to maize genetics and breeding. This work may be successful, provided the number of genes responsible for the induction of haploids is known. To determine this number of genes, the following test was carried out. The parental stocks used were two lines: a haploid inducer, Korichnevy Marker Saratovsky (KMS), and a usual commercial line, MK01, in the progeny of which no haploids have ever occurred. Previously it has been established that the yield of maternal haploids ranges from 0.75 to 2.94% when KMS is used as a pollen parent. Long-term experiments have shown that the range of variation of the KMS haploid-inducing capacity is largely dependent on the female parent genotype (Tyrnov and Zavalishina, 1984). A backcross (MK01 x KMS) x KMS, was made. The backcross plants were selfed and the resulting seeds were sown in a field plot to determine the frequency of haploids. Haploid and diploid plants were identified during flowering by a set of morphological traits. Previously it has been found that morphological identification is more reliable than cytological analysis. This is due to the fact that a small proportion of cells with diploid chromosome sets occur in almost every tissue of haploid plants, which can lead to distorted results (Khokhlov et al., 1976). A total of 54 progenies by selfing were examined. The results are listed in the table below. The upper row of the table shows the percentage of haploids recovered, the bottom one indicates the number of progenies.

%	0.25	0.75	1.25	1.75	2.25	2.75	3.25
No.	30	4	8	5	3	2	2

Thirty progenies either showed no haploids, or their frequency of haploids was very low, tending to zero. Twenty-four progenies by selfing backcross plants had the proportion of haploids which corresponded to that of the KMS and varied between 0.75 and 3.25%. The segregation ratio 30:24 is rather close to 1:1. Ideally, the ratio should be 27:27. Chi-square was 0.67, indicating close agreement with the expected segregation. It can be suggested, therefore, that, with respect to the trait concerned, KMS only differs from the usual MK01 line by a single gene. Lack of haploids is dominant, although the dominance is incomplete. It can be inferred from these findings that the development of new haploid inducers is unlikely to be a very difficult task.

In addition to KMS, we have extensively used in our work the inducer line Zarodyshevy Marker Saratovsky (ZMS). Its haploid-inducing capacity closely approximates that of KMS, ranging from 1.7 to 3.4% in different female parent genetic backgrounds. To test for allelism between the genetic factors controlling haploid-inducing capacity of KMS and ZMS, a cross between these two lines was made. The resulting hybrid plants were selfed to produce the F2 progeny. Segregation analysis was based on the frequency of haploids resulting from selfing of the F2 plants. Haploids were identified according to the method described above. A total of 22 progenies were examined. Out of these, 15 have been found to correspond to parental lines in the trait concerned, their haploid frequencies varying between 0.93 and 3.77%. Three progenies lacked haploids (0%). The third group of genotypes are of particular interest. These obviously were superior to parental lines KMS and ZMS. Their frequency of haploids varied between 6.94 and 8.75%. This is more than two times higher than in the parental inducer lines. There were four such progenies. Unfortunately, only 22 progenies were examined, and the F2 segregation ratio allows no definitive conclusions to be made. However, the presence of obviously transgressive forms suggests that KMS and ZMS differ by no less than two genes. In the future, homozygosity of the four progenies superior to the parental lines should be tested.

Both ZMS and KMS exhibit haploid-inducing potential. So it can be assumed that one of the differing genes should be carried by KMS and the other or others by ZMS. This assumption is confirmed by the fact that one of the genes considered was found, in the above experiment, to be carried by KMS.

Thus, it can be suggested that KMS and ZMS are sources of no less than two different genes whose joint action may result in 7 to 9% yield of haploids.

The influence of chronic gibberellin treatment on the expression of the heterochronic mutation *Cg2*

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

In order to understand a specific nature of the hormonal disequilibrium in corn heterochronic mutations and its influence on developmental processes, plants of *Cg1* and *Tp1* were treated with a gibberellin solution (Nickerson, Am. J. Bot. 47:809, 1960). Recently, this experiment has been carried out once more (Ritchings and Tracy, Maydica 34:297, 1989).

One of the sublines of the heterochronic mutation *Cg2* carriers (*Cg2/Cg2*) was also GA_3 treated according to Nickerson's protocol at a dose of 82 ppm. Twenty-three corn plants were GA_3 treated, and 25 plants were treated with H_2O. Gibberellin changed the corngrass phenotype, i.e. the plant grew significantly higher and side shoots were not formed (Fig. 1). The node number and leaf length increased, leaf width remaining nearly unchanged. The male inflorescence in the form of a spikelet was observed only in single corn plants, and ears were small as before (Table 1).

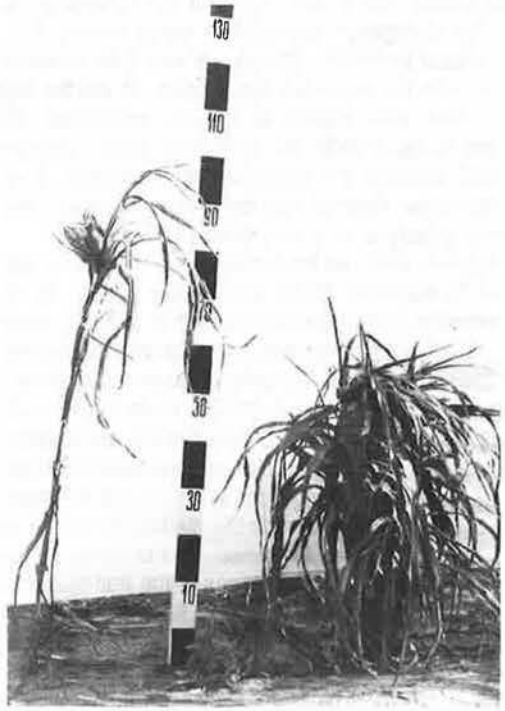

Figure 1. The corn plant (left) was chronically treated with a GA_3 solution. The corn plant (right) was chronically treated with H_2O.

Table 1. Response of the *Cg2* 220-569-205-315 subline to gibberellin treatment.

Treatment	Plant height, cm	Tassel length, cm	Cobs no.	Stems no.	Stem diameter, mm	Nodes no.	Leaf length, cm	Leaf width, cm
Control	75.1±7.8	7.3±3.6	9.9±1.4	10.3±2.6	8.9±1.6	12.6±1.2	41.9±3.0	2.1±0.4
GA	113.3±11.6	-	1.8±0.5	1.0	9.1±0.5	17.3±1.2	46.7±2.2	2.3±0.2

According to its response to exogenic gibberellin application, the heterochronic mutation *Cg2* is deficient in the growth hormone. Nevertheless, like the heterochronic *Cg1* mutation, *Cg2* is not a simple biochemical mutant resembling dwarf mutants and, despite the absence of a complete and clear pattern of hormonal balance changes occurring in heterochronic corn mutations, a hormonal explanation of the sex display in corn does exist. This explanation implies a seasonal alteration of the hormonal balance between cytokinins, which cause a female expression, and gibberellins, which are produced by leaves and cause a male expression (Iltis, Science 222:886, 1983). Apparently, something like that occurs in heterochronic corn mutations.

Expressivity of the heterochronic mutation *Cg2* and its correlation with gene dose

--N. V. Krivov

In the first report on *Cg1* this mutation was referred to as a second "teopod" (Singleton, MNL 21:6, 1947). Subsequently. corngrass was shown to be highly susceptible to genetic modification. The early selections for weak expression of the corngrass gene were similar to the teopod mutant while later selections approached normal corn (Galinat, Amer. Nat. 88:101, 1954).

According to the expression degree, the heterochronic mutation *Cg2*, like *Cg1* segregates for two relatively discrete phenes: "teopod", a moderate expression of *Cg2*, *Cg2(m)* and "corngrass", a strong expression of *Cg2*. This made it possible to study the kind of *Cg2* expression in homo- and heterozygotes, or in other words, how expressivity depends on gene dose (Table 1).

Table 1. Correlation between heterochronic mutation *Cg2* expressivity and gene dose.

		Cg2/Cg2		*Cg2/+*	
Lines no.	Phenotypes of plants tested	No. plants	%	No. plants	%
143	Cg2(m)	9	75	48	96
	Cg2(s)	3	25	2	4
220	Cg2(m)	12	60	67	95.7
	Cg2(s)	8	40	3	4.3

The table shows that among mutant plants which, according to analyses, turned out to be heterozygous for the progeny, 96% had a teopod phene while the remaining 4% belonged to the corngrass phenotype. Among homozygous plants (*Cg2/Cg2*) tested for the progeny a share of plants with a corngrass phene rose from 25% in the line No. 143 to 40% in No. 220. Thus, the possibility of finding a homozygous plant in the line No. 143 is 6 times, and in No. 220 10 times higher among specimens showing a strong mutant phenotype, *Cg2(s)*. Hence, two doses of the *Cg2* gene increase mutant phene expressivity while alleles of the wild type reduce *Cg* expressivity. *Cg2* is likely to encode a product which is antagonistic to normal gene activity.

The interaction between genes suppressing heterochronic mutant *Cg2* manifestation and the cytoplasm genome

--N. V. Krivov

The presence of genes modifying *Cg1* penetrance was known in self-pollinated corn lines long ago (Galinat, MNL 26:52, 1952). Heterochronic mutations *Tp1*, *Tp2* and *Tp3* are also sensitive to genes that modify their penetrance and expressivity (Poethig, Genetics 119:959, 1988). The heterochronic mutation *Cg2* reacts to a foreign genetic background like the four dominant mutations (Krivov and Lysikov, Buletinul AS RM, No. 2:20, 1988).

During corn domestication, which took place 4 thousand years ago (Mangelsdorf, Sci. Amer. 255:72, 1986), a lot of dominant and recessive genes maintaining the apical dominance of the main stem have accumulated in the process of corn breeding (Iltis, Science 222:886, 1983). Genes suppressing *Cg2* manifestation are supposed to occur more frequently in lines with a high combining ability than in lines with a low one. In order to test this supposition crossings between *Cg2 gs bm2* and self-pollinated lines D5, Pr01a, F2, F7, O92, Co125, 346, VIR-44, N384, MK01, Rad.

dwarf USA, kindly presented by S. T. Chalyk and A. J. Dukhovniy, have been conducted. All these lines, with the exception of D5 and Pr01a, possess a high combining ability and contain genes that reduce the expression and inhibit the manifestation of the heterochronic *Cg2* mutation. In all cases the impact of foreign genetic backgrounds is generally displayed through the reduction of expression of the whole complex of mutant traits characteristic of the heterochronic *Cg2* mutation, and through the F1 uniformity disturbance as many plants must be referred to the wild type corn.

Reciprocal crossings have shown that the manifestation of the heterochronic mutation *Cg2* depends on the line which is taken as mother, i.e. on the cytoplasm genotype (Table 1). Only the cytoplasm of the 346 line turned out to be quite indifferent and, perhaps genetically similar to the *Cg2 gs bm2* line. Two cases of *Cg* suppression should be mentioned particularly: (1) when the suppressor gene, which is contained in MK01, suppresses *Cg2* manifestation completely at the interaction with the *Cg2 gs bm2* cytoplasm genome while the suppression of the *Cg2* manifestation is incomplete at the interaction with its own cytoplasm genome, and (2) when *Cg2* manifestation is suppressed completely by the suppressor gene at the interaction with the cytoplasm genome of the Co125 and VIR-44 lines.

Table 1. *Cg2* suppression in F1 when *Cg2 gs bm2/Cg2 gs bm2* is hybridized with the lines contained in recognized hybrids.

		Phenotypic classes					
Combinations	wt	Cg2w	Cg2m	Cg2s	m(Cg2)	Cg2 %	Total
Cg2 gs bm2 gl x D5	2		29		3	94.1	34
Cg2 gs bm2 x Pr01a	10		30			75.0	40
Cg2 gs bm2 x F2	11		27			71.0	38
O92 x *Cg2 gs bm2*	6	13			2	71.4	21
O92 x *Cg2 gs bm2*	15	17	1			54.5	33
Cg2 gs bm2 x F7	16	18	1			54.3	35
F7 x *Cg2 gs bm2 gl*	18	12				40.0	30
Cg2 gs bm2 x Co125	14		20		1	60.0	35
Co125 x *Cg2 gs bm2*	14					0.0	14
Cg2 gs bm2 x 346			19	1		100.0	20
346 x *Cg2 gs bm2*			39			100.0	39
Cg2 gs bm2 x VIR-44	14		20		2	61.1	36
VIR-44 x *Cg2 gs bm2*	37					0.0	37
Cg2 gs bm2 x N384	37		1			2.6	38
N384 x *Cg2 gs bm2*	31		1	4		13.9	36
Cg2 gs bm2 x MK01	37					0.0	37
MK01 x *Cg2 gs bm2*	7		7		3	58.8	17
Cg2 gs bm2 x Rad. dwarf	22		16		1	43.6	39
Rad. dwarf x *Cg2 gs bm2*	18		14		6	52.6	38

wt - wild type; Cg2w - weak expression of the mutant phene; Cg2m - moderate expression of the mutant phene; Cg2s - a strong expression of the mutant phene; m(Cg2) - a mosaic expression of the *Cg2* phene.

A significant excess of the wild type, and *Cg2* deficit are observed in the progeny of self-pollinated heterozygotes (Table 2). Accordingly, among 174 plants in the MK01 x *Cg2 gs bm2* combination only one had a weak expression of the *Cg2* phenotype in F2, whereas in the VIR-44 x *Cg2 gs bm2* and Co125 x *Cg2 gs bm2* combinations not a single plant with the *Cg2* phenotype was found among progenies. The *Cg2 gs bm2* x D5 combination is an exception as the deviation from the theoretically expected ratio 3:1 is not significant (X^2=2.7). As the *Cg2* phene suppression in VIR-44 x *Cg2 gs bm2* and Co125 x *Cg2 gs bm2* hybrids is supposed to be related to the interaction between the suppressor gene and cytoplasm genome of the Co125 and VIR-44 lines, the *Cg2* gene should be transferred to another cytoplasm through hybridization in order to receive segregants with the *Cg2* phenotype.

Table 2. Segregation for *Cg2* and wt in F2 when *Cg2 gs bm2/Cg2 gs bm2* is hybridized with the lines contained in recognized hybrids.

		Phenotypic classes					
Combinations	Phenotype in F1	wt	Cg2w	Cg2m	Cg2s)	Cg2 %	Total
Cg2 gs bm2 x VIR-44	Cg2w	57	14	16		34.5	87
VIR-44 x *Cg2 gs bm2*	wt	168					168
Cg2 gs bm2 x Co125	Cg2w	99	14	3		14.7	116
Co125 x *Cg2 gs bm2*	wt	230					230
Cg2 gs bm2 x D5	Cg2m	16	20	9		64.4	45
Cg2 gs bm2 x Pr01a	Cg2m	34	23	10		49.3	67
Cg2 gs bm2 x F2	Cg2w	84	4	71	4	48.5	163
Cg2 gs bm2 x 346	Cg2w	23	15	18	4	61.7	60
346 x *Cg2 gs bm2*	Cg2w	37	10	15	1	41.3	63
Cg2 gs bm2 x N384	wt	120	14	40	5	33.0	179
N384 x *Cg2 gs bm2*	wt	127	6	27	5	23.0	165
MK01 x *Cg2 gs bm2*	wt	173	1			0.6	174
Cg2 gs bm2 x MK01	wt	106	1	13	2	13.1	122

With this aim crossings between the VIR-44 x *Cg2 gs bm2* and Co125 x *Cg2 gs bm2* hybrids and Rad. dwarf USA, F2 and F7 used as mother were carried out (Table 3). However, plants with

Table 3. Crossing test data of *Cg2/+* heterozygote transferences to the cytoplasm of Rad. dwarf USA, F2 and F7 lines.

Plant tested	Crossing	Progeny phenotype wt
N1	Rad. dwarf USA x (VIR-44 x *Cg2 gs bm2*)	21
N2	Rad. dwarf USA x (VIR-44 x *Cg2 gs bm2*)	39
N4a	Rad. dwarf USA x (VIR-44 x *Cg2 gs bm2*)	32
N4b	Rad. dwarf USA x (VIR-44 x *Cg2 gs bm2*)	34
N7	Rad. dwarf USA x (VIR-44 x *Cg2 gs bm2*)	35
N1	Rad. dwarf USA x (Co125 x *Cg2 gs bm2*)	38
N2	Rad. dwarf USA x (Co125 x *Cg2 gs bm2*)	21
N2	F2 x (Co125 x *Cg2 gs bm2*)	40
N2	F7 x (Co125 x *Cg2 gs bm2*)	25
N4	F7 x (Co125 x *Cg2 gs bm2*)	26
N4	F7 x (Co125 x *Cg2 gs bm2*)	35
N4	F2 x (Co125 x *Cg2 gs bm2*)	39
N5	Rad. dwarf USA x (Co125 x *Cg2 gs bm2*)	32
N6	Rad. dwarf USA x (Co125 x *Cg2 gs bm2*)	39
N9	Rad. dwarf USA x (Co125 x *Cg2 gs bm2*)	27
N9	F2 x (Co125 x *Cg2 gs bm2*)	38
N9	F7 x (Co125 x *Cg2 gs bm2*)	32

the expected *Cg2* phenotype have not been obtained. Thus, self-pollinated MK01, VIR-44 and Co125 lines having a very high combining ability contain dominant genes unique for their potentials which maintain corn (*Zea mays* L.) habitus, while in lines with a low combining ability D5 and Pr01a are very weak recessive genes.

The effect of the chromosome 1 segment marked by the *Adh1* locus on quantitative traits

--A. A. Chernov, M. E. Mihailov and S. V. Ursul

The objective of the present study was to establish the genotypic relationship between the chromosome 1 segment marked by the *Adh1* locus and a number of agronomic traits. The study was carried out on Moldavsky 291, a high-yielding hybrid widely cultivated in Moldova. The parental lines, F1 and 188 F2 plants were estimate for the following quantitative traits: 1) time from emergence to the flowering of panicles, days; 2) time from emergence to the flowering of top ears, days; 3) time lag of the onset of flowering between panicle and top ear, days; 4) time from flowering to the maturation of top ears, days; 5) time from emergence to the maturation of top ears, days; 6) number of stems; 7) plant height, cm; 8) stem length, cm; 9) panicle length, cm; 10) top ear position on the stem, cm; 11) diameter of the bottom first internode, mm; 12) number of the above-ground nodes; 13) number of ears with kernels; 14) internode mean length, cm; 15) stem volume parameter, litre; 16) ratio of stem length to bottom first intern-

ode diameter; 17) weight of top ear at harvest, gm; 18) weight of the remaining ears at harvest, gm; 19) total weight of ears, gm; 20) the proportion of second top ears in total ear weight, %; 21) daily increment in ear weight, gm.

Enzyme electrophoresis extracts from pollen were run in 14% starch gel (buffer system "G", pH=7.0) (Wendel and Stuber, Isozyme Bull. 17:4-11, 1984). The gel staining was performed using reaction mixtures from Levites' list (Genetika isozymes of the plants, Nauka, Novosibirsk, 1986).

Isoenzyme analysis has shown the parental lines to differ with respect to the *Adh1* locus. This allows 3 genotype classes to be distinguished in the F2 population. For the quantitative traits studied, the following statistically significant differences between classes FF, FS, and SS were observed (Table 1): 1) panicles of the FS heterozygotes enter flowering earlier than those of SS; there is a larger time lag for the onset of flowering between panicle and ear compared with FF and lower weight of the first ear compared with FF; 2) the higher position of the top ear in the FF homozygote.

Table 1. Mean values of quantitative traits for three *Adh1* segregation classes in maize F2.

	FF			FS			SS		
Traits	N	Mean	SE	N	Mean	SE	N	Mean	SE
1	52	65.81	0.40	86	65.31*	0.31	50	66.22*	0.34
2	52	66.31	0.39	86	66.51	0.35	50	67.28	0.41
3	52	0.50**	0.17	86	1.20**	0.18	50	1.06	0.24
4	52	39.33	0.72	86	38.83	0.65	50	37.52	0.69
5	52	105.63	0.86	86	105.33	0.75	50	104.80	0.78
6	52	1.02	0.02	86	1.02	0.02	50	1.06	0.04
7	47	197.42	2.59	81	195.22	2.15	47	193.17	3.06
8	52	163.13	2.28	86	162.10	1.94	50	161.52	2.78
9	47	34.02	0.60	81	33.59	0.47	47	32.45	0.69
10	52	60.88*	1.58	86	56.69	1.46	50	55.94*	1.73
11	52	22.17	0.37	86	22.05	0.33	50	21.53	0.44
12	52	11.73	0.13	86	11.52	0.12	50	11.46	0.15
13	52	1.65	0.11	86	1.74	0.09	50	1.90	0.11
14	52	14.55	0.17	86	14.75	0.13	50	14.75	0.18
15	52	0.82	0.03	86	0.81	0.03	50	0.78	0.04
16	52	74.21	1.19	86	74.47	1.08	50	75.99	1.51
17	32	196.46*	7.20	37	175.91*	5.42	26	182.96	10.25
18	32	55.88	12.45	37	57.84	10.08	26	71.92	14.49
19	32	252.34	13.43	37	233.75	12.05	26	254.88	18.25
20	32	17.36	3.69	37	19.88	3.09	26	22.68	4.07
21	32	6.77	0.39	37	6.56	0.33	26	7.13	0.52

Note: *, ** means significantly different at 5% and 1% levels.

The results suggest that the chromosome 1 segment marked by the *Adh1* locus genetically affects the above traits in maize. Future research is needed to ascertain the nature of the effects observed.

Mutagenic effects of laser radiation and 6-mercaptopurine on seedlings

--V. K. Burilkov, V. M. Paschenko and V. N. Lysikov

Mutagenic effects of acridine orange (AO) and ethidium bromide (EB) on prokaryotes and eukaryotes have been studied (Burilkov and Krochik,in Laser in the Life Sciences, pp. 253-274, 1988; Dragan and Khrapunov, Cytol. Genet. 26:32-35, 1992). We have compared cytogenetic effects of the known sensitizers, AO and EB, and of ones not used previously, such as 6-mercaptopurine (6-MP) and cloroxine (CX), each used in combination with laser radiation (LR).

Seedlings of A-346 grown on media containing the above sensitizers at a concentration of 0.00001 M each, were exposed to LR for 1 min (λ=337.1 nm, I=70 MWt/m·m·sec). Counts of chromosome aberrations were made in temporary preparations during mitotic anaphase and telophase of maize (Gostimsky, Practical Guide on Cytogenetics, 1974).

The studies have shown that the highest rate of chromosome aberrations occurred when 6-MP was used as a sensitizer (Fig. 1). This much exceeded the rates of chromosome aberrations resulting from exposure to EB+LR, CX+LR, and AO+LR.

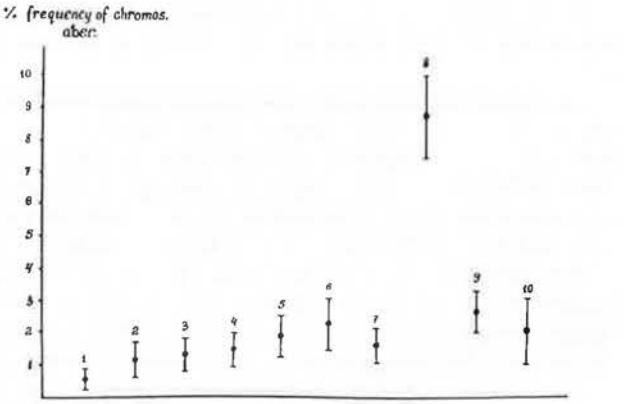

Figure 1. The rate of chromosome aberrations during mitotic anaphase-telophase in maize rootlets resulting from exposure to sensitizers and LR. 1 - control; 2 - LR; 3 - AO; 4 - AO+LR; 5 - CX; 6 - CX+LR; 7 - 6-MP; 8 - 6-MP+LR; 9 - EB; 10 - EB+LR.

One possible cause of chromosome aberrations induced by LR and sensitizers may be the formation of one- and two-strand breaks due to laser radiation energy which is transferred from the sensitizer molecule to certain DNA sites. The resultant one- and two-strand breaks may be repaired during mitosis, or they may eventually turn into chromosome aberrations. To test this hypothesis, we studied maize genomic DNA by the gel electrophoresis technique. Electrophoretic patterns and break counts from densitograms (Zhizina et al., Radiobiology 23:783-786, 1983) have suggested that the hypothesis is not implausible (Fig. 2, Table 1).

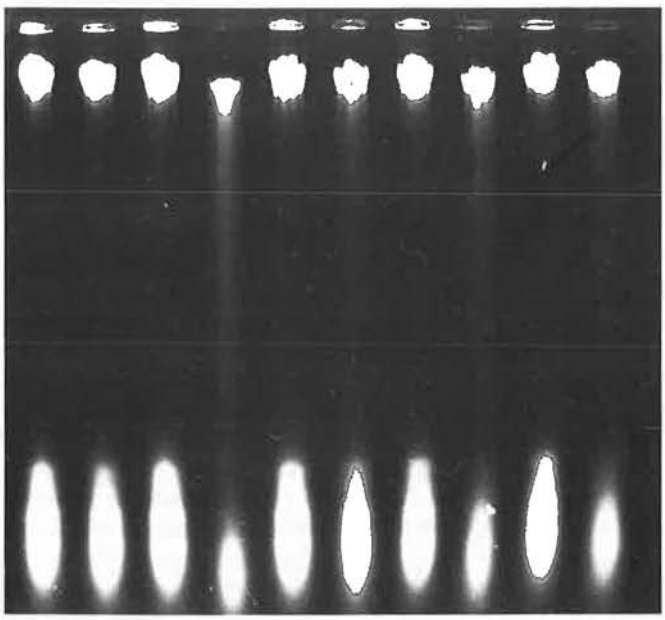

Figure 2. Electrophoretic patterns (non-denaturating) for maize DNA and DNA+sensitizer complex. Lanes: 1 - DNA; 2 - DNA+LR; 3 - DNA+6-MP; 4 - DNA+6-MP+LR; 5 - DNA+AO; 6 - DNA+AO+LR; 7 - DNA+CX; 8 - DNA+CX+LR; 9 - DNA+EB; 10 - DNA+EB+LR.

Table 1. The relative number of breaks in total DNA of maize resulting from exposure to sensitizers and LR.

LR	6-MP+LR	EB+LR	AO+LR	CX+LR
3.87	64.50	24.94	12.47	14.62

The induction of chromosome aberrations in maize plants with the aid of 6-MP and LR has a number of advantages over the conventional techniques: (1) it shows relative selectivity--interaction is primarily with DNA; (2) it enables the molecular mechanisms of mutation and recombination to be more precisely identified; and (3) it produces no cytotoxic effects.

A high level of mutations of various types, inherited in M2 and M3, were discovered following the treatment of maize seedlings of the A-346 line. The analysis is in progress.

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Effect of growth environment on development of *Zea* x *Tripsacum* hybrid kernels

--E. Erygina and A. Mashnenkov

Our experiments were aimed at clarifying the exact relationship between response to mutagens and mutability of corn lines and their compatibility with *Tripsacum dactyloides* L. (MNL 65:72, 79, 1991).

The lines resistant to mutagen effects (R) demonstrate complete incompatibility regardless of environment (Table 1). A great environmental effect on the rate of hybrid kernel development proves typical in the lines of moderate resistance (MR) and in the susceptible lines (S) in particular. The better the environment is during the vegetation period, the greater the percent of developed kernels per ear. Line lg57 appears to be an expected exception. It shows a high value and relative stability of the trait. Probably the distinct relationship between mutability, response to mutagens and compatibility only manifests in a favourable environment (1989). In case of unfavourable conditions it partially (1990, 1992) or completely (1991) changes because of the different adaptability of the lines.

Table 1. Percentage of viable hybrid kernels per ear.

		Year			
Line	Response to Mutagens	1989	1990	1991	1992
T22	R	0.00	0.00	0.00	0.00
Gk26	R	0.00	0.00	0.00	0.00
W23	MR	3.55	--	0.00	4.40
A344	MR	28.20	12.24	5.52	24.51
PLS61	MR	33.99	2.52	0.00	--
F2	S	3.62	0.00	0.00	0.00
Hy2	S	7.92	--	0.00	0.00
A663	S	12.96	5.24	0.00	--
lg57	S	--	38.22	21.13	36.41

Mass induction of maternal haploids in corn

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

The method of genetic marking proves superior to the other methods of haploid induction in corn, including pollen culture, in number of induced haploids and in simplicity and inexpensiveness of the procedure. The markers developed at our laboratory enable us to induce rather easily a large number of haploids (from some thousands to some dozens of thousands) annually. This method does not require expensive reagents and complicated equipment. The Krasnodar markers are involved in crosses as males. They carry *A C R-nj* dominant genes for embryo and endosperm colour. This makes it possible to select marked stock at the stage of dry kernel. This is the merit of the method because by using other methods of selection for haploidy it is necessary to germinate all marked kernels. The other merit of it is a high frequency of haploidy due to these markers in hybrids and populations as females.

Involvement of 3MC line, one of the lines obtained from Stock 6 and selected for "haploid stimulation" (Tyrnov and Zavalisha, Dokl. Akad. Nauk SSSR 276:3, 1984) into the genotype of the advanced Chase's marker (PEM) resulted in a higher frequency of maternal haploid induction. For some years the developed markers were used in ear-to-row selection for a frequency of haploidy. The group of the markers stimulating maternal haploidy up to 6.3% and even over 10% in some ears was selected in 1992 (Table 1).

Table 1. Stimulation of maternal haploidy by various groups of markers*.

				Frequency of haploidy	
Group of markers	No. ears	No. kernels	Haploids	%	per ear
PEM	68	24,834	46	0.19	0.7
3MC	24	8,206	60	0.73	2.5
Krasnodar markers	52	10,398	655	6.30	12.6

Use of such markers at a sufficiently high frequency of diploidization (20-30%) enable us to obtain annually more than a thousand new homozygous lines. Thus, haploidization becomes a competitive method comparable to traditional inbreeding in number of developed lines, reducing the period for line development by 2-3 times.

Application of the Krasnodar markers made it possible to produce 760-1,500 new autodiploid lines in 1992 and 1993, respectively.

Autodiploid lines as sources of haploid spontaneous diploidization in corn

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

In our search for the cultivars displaying a high frequency of spontaneous diploidization of haploids, the 613/2 c4 line was revealed (MNL, 1993). In 1993 the frequency of spontaneous diploidization (the ratio of the number of plants with fertile anthers to the total number of haploids) was as high as 22% in 613/2 c4 (Table 1). Some lines of the fifth cycle of haploidy were derived from 613/2 c4 by genetic marking and spontaneous diploidization. The trait under research, haploids originating in these lines, varies within the range of 11-43%. Stimulation of diploidization shows intermediate inheritance in hybrids.

Genotypes with a high frequency of spontaneous diploidization in some lines and hybrids were selected among the autodiploid lines of 1-3 cycles. In the haploids of these cultivars spontaneous chromosome doubling without a colchicine treatment was observed at a rate of 23-46%. These plants carry a vital ear and some fertile anthers or a fertile sector in a sterile tassel.

Table 1. Frequency of spontaneous diploidization of haploids in corn hybrids and their parents.

Origin of haploids	No. haploids	No. plants with fertile anthers	Frequency of diploidization, %
Ts8	57	3	5.3
Ts8 x 613/2 c4	89	12	13.5
Ts16	59	2	3.4
Ts16 x 613/2 c4	220	31	14.0
Intermated Mo17	170	2	1.2
Intermated Mo17 x 613/2 c4	59	8	13.6
613/2 c4	151	33	21.9

Thus, the sources of a high frequency of spontaneous diploidization could be found with a greater success within the cultivars (lines and hybrids) having passed over a haploidy state than within the lines of traditional selection.

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Cytological studies in alloplasmic lines of maize

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We studied the meiotic behavior of different alloplasmic lines of *Zea mays* ssp. *mays*, with cytoplasm of teosinte (*Z. mays* ssp. *mexicana*; Florida variety, Huixtla, Mexico). These lines were obtained by L. B. Mazoti using inbred lines of maize as the recurrent male parent, and teosinte as the cytoplasmic female donor. The maize inbred lines "*c*-tester", "*gl ij*" and "Multiple Dominant" were introduced in Argentina in 1933 by Ing. Agr. S. Horovitz, and they were maintained in the IFSC since their introduction. To obtain the alloplasmic lines, these inbred lines were backcrossed for 5 ("flint"), 7 ("*c*-tester", "*gl ij*" and "*r*-tester") and 20 ("Multiple Dominant") generations (Mazoti, 1978, 1987).

In the "Multiple Dominant" line with teosinte cytoplasm, Mazoti and Velazquez (1962) found a greater percentage of pollen sterility and greater variation of nucleolus diameter, than in the normal line. In addition, they found stickiness of meiotic chromosomes, and frequent intercellular contacts observed in sectioned anthers. Moreover, Mazoti (1987) reported that in this alloplasmic line, the knobs have greater size and higher DNA content. Poggio et al. (1990, 1991) using Feulgen microdensitometry, found higher DNA content per nucleus and higher heterochromatin percentage in the alloplasmic lines "Multiple Dominant" "*c*-tester" and "*gl ij*" than in the inbred lines. The same authors found that some individuals of the "Multiple Dominant" alloplasmic line showed desynapsis, cytomixis, cell fusion and pseudomultivalents in various places of the panicle.

In the present work we further analyze the meiotic behaviour and pollen stainability of these alloplasmic lines. From the previous work as well as from the present results we can list the following common features:

i) In the majority of the cells, the lines with teosinte cytoplasm showed 10 bivalents distributed in two groups of 5 bivalents each. This disposition was more remarkable than that found in the inbred lines (Fig. 1A, B).

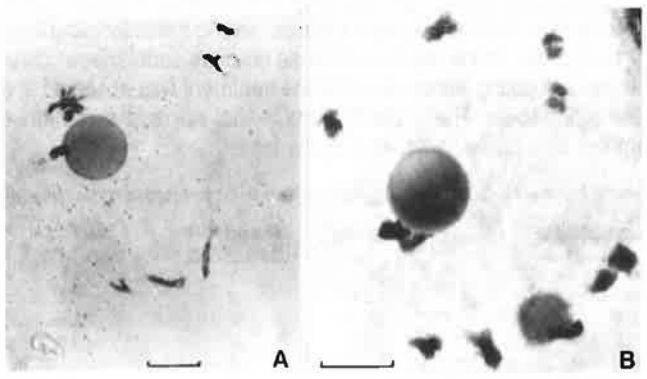

Figure 1. A and B: see explanation in the text. Bars=10 um.

ii) In about 20% of the PMCs in prophase, metaphase and anaphase I, the two groups of 5 bivalents are slightly asynchronous to each other (Fig. 1A).

iii) In contrast to the usual formation of only one nucleolus per PMC in *Zea* species and hybrids, two nucleoli were observed in 30% of the cells of the alloplasmic lines, each being associated with one of the groups of 5 bivalents (Fig. 1B). The presence of two nucleoli in the alloplasmic lines can be interpreted as if the teosinte cytoplasm permitted the expression of one inactivated NOR in the normal maize. There are evidences supporting a basic chromosome number of x=5 in the genus *Zea* (Molina and Naranjo, 1987; Poggio et al., 1990); therefore, each nucleolus could correspond to each genome of 5 bivalents, both being expressed in many of the cells of the alloplasmic lines.

These results support the hypothesis that maize is an allotetraploid and each group of 5 bivalents would correspond to a different genome. Additionally, the stainability of pollen grains was evaluated using the Alexander stain (1969). In the alloplasmic "Multiple Dominant" line with teosinte cytoplasm, a variation between 0-48% of stained pollen was recorded. This result agrees with that of Mazoti (1978) who found that this alloplasmic line shows mosaicism for pollen viability, and with that of Poggio et al. (1990), who found mosaicism for meiotic behaviour. In the other alloplasmic lines, pollen stainability was very low, 35% in *r*-tester and about 0% in the other lines in all anthers, and mosaicisms for meiotic behaviour were not observed.

Meiotic behavior of maize B chromosomes in the native race "Pisingallo" from NW Argentina

--A. M. Chiavarino*, L. Poggio and C. A. Naranjo

We are interested in the population genetics and meiotic behavior of the native races of maize from Argentina. B chromosomes are frequent in these races (Rosato et al. MNL 15:67; Chiavarino et al., MNL 15:68, 1993). In the present work we analyze the polymorphism frequencies and the meiotic behavior of B's in 78 individuals of the maize native population "Pisingallo" from NW Argentina. B's were found in 43.6% of the individuals, the distribution being: 64% 1B, 30% 2B, 4% 3B and 3% 4B. The meiotic behavior of B's can be summarized as follows:

1B plants. Metaphase I: The B univalent remains outside the plate. Anaphase I: The B chromosome migrates precociously to one pole in 50-95% of cells studied, and is included in the pole or forms a micronucleus; lags at the metaphase plate (0-8%) and forms a micronucleus; or divides equationally (0-7%), and is included in the poles or forms 1 or 2 micronuclei. The percentage of dyads with micronuclei was 5-35%, indicating that the B is frequently lost in the first meiotic division.

2B plants (Fig. 1). Diakinesis: The B's can be observed forming 2 univalents (0-21%), 1 bivalent (65-94%) or 1 pseudobivalent (0-14%). Metaphase I: Univalents remain outside the plate (5-35%) and can migrate to the same or to different poles, or divide equationally in very few cells; in all the cells the two univalents are secondarily associated. Bivalents remain in the metaphase plate together with the A bivalents (92-100%), or outside the plate. In the former case the B's migrate precociously to the poles in a few cells or migrate together with the A's. In the latter the bivalent lags in AI or the B's migrate precociously to the poles. At metaphase II the B's frequently remain outside the plate. Micronuclei were observed.

3B plants. At diakinesis the B's can be associated, forming a

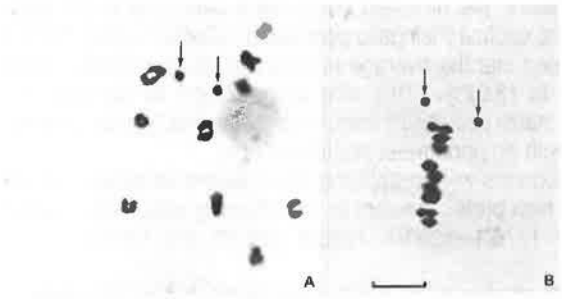

Figure 1. A and B: 2n=20+2B. A=diakinesis; B=metaphase I. Arrows show the B univalents. Bar=10 um.

trivalent (35%), bivalent plus univalent (60%) or 3 univalents (5%). The bivalent and univalent have secondary association in all the cells studied. Metaphase I: The 3 univalents remain outside the plate, migrating to the poles at random. The bivalent remains in the plate with the A's migrating normally, while the univalent migrates precociously to one pole. The trivalent always remains in the plate, orientating one B to one pole and two to the other.

The percentages of the different meiotic configurations given above are the pooled data scored in the observed individuals. It is worth mentioning that there was large interindividual variation; for example, in some individuals the B's tended to behave like the normal complement, while in others they tended to form more univalents, laggards, equational division, etc. For this reason we include the range of variation in the most remarkable cases. It is interesting that this variation in the meiotic behavior may be due to a genetic variation present in the native race, which may affect the B chromosome transmission and polymorphism, as occurs in other organisms (Puertas et al., Chromosomes Today 11:391-399, 1993).

Development of waxy maize inbred lines

--V. R. Corcuera and C. A. Naranjo

The objective is the development of inbred lines to get waxy endosperm hybrids suitable for the wet-milling process. Such hybrids would allow the extraction of modified starch useful for food industry, adhesives and others.

With this purpose during the summer of 1990-1991, a breeding program similar to the one proposed by Cornelius and Dudley (1974) was initiated. The basic germplasm used were four waxy endosperm maize populations (S0) called: SCV1, SCV2, WEM, and FW as well as two hard endosperm populations named CP27 and S80. In each population, couples of the best individuals were crossed, whilst some others were selfed, and free pollination was also allowed. This way, from each one of the original populations, two subpopulations were obtained as well as the first generation of selfings (S1) during the first year. These materials were agronomically evaluated during 1991-92 and then the second generation of selfings (S2) and the first generation of sib matings (SI1) was obtained. These materials, along with the S1's and S0's, were agronomically evaluated during the field crop 1992-93 for different plant and prolificacy traits. The plant traits measured were: plant height in meters (PH) - ear insertion height in meters (EIH) - number of tillers (NT) and number of leaves (NL). The prolificacy traits measured were: number of productive nodes (PN) - number of ears in the uppermost node (EUN) - number of ears per tiller (ET) and number of ears per plant (EP).

Tables 1 and 2 show the average values obtained for each trait in each generation as well as the relative values (in percentage) for each trait in the inbreeding generations referred to as the S0 generation average values. It is easily noticed that the average values decreased through the generations of inbreeding, though the speed of this process depends on both the nature of the original population and on the inbreeding method applied.

Table 1. Plant trait average values and relative values for the original populations and their derived inbred lines.

		PH (m)		EIH (m)		NT		NL	
Generation		abs.	rel.	abs.	rel.	abs.	rel.	abs.	rel.
SCV1	(S0)	1.62	100.0	0.67	100.0	1.58	100.0	10.65	100.0
	(S1)	1.49	92.0	0.48	71.6	1.27	80.4	10.51	98.7
	(SI1)	1.53	94.4	0.63	94.0	1.38	87.3	10.85	101.9
SCV2	(S0)	1.40	100.0	0.43	100.0	1.14	100.0	10.32	100.0
	(S1)	1.19	85.0	0.39	90.7	1.08	94.7	9.61	93.1
WEM	(S0)	1.33	100.0	0.33	100.0	1.42	100.0	10.17	100.0
	(S1)	1.26	94.7	0.25	75.7	1.26	88.7	9.78	96.2
	(SI1)	1.30	97.7	0.31	93.9	1.28	90.1	9.40	92.4
FW	(S0)	2.01	100.0	0.68	100.0	1.12	100.0	13.03	100.0
	(S1)	1.86	92.5	0.65	95.6	1.10	98.2	12.11	92.9
	(SI1)	1.92	95.5	0.69	101.5	1.05	93.7	11.00	84.4
CP27	(S0)	1.88	100.0	0.67	100.0	1.12	100.0	12.00	100.0
	(S1)	1.84	97.9	0.62	92.5	1.05	93.7	12.53	104.4
	(S2)	1.78	94.7	0.57	85.0	1.06	94.6	11.94	99.5
S80	(S0)	1.90	100.0	0.62	100.0	1.18	100.0	11.92	100.0
	(S1)	1.72	90.5	0.49	79.0	1.25	105.9	10.25	86.0
	(S2)	1.70	89.5	0.42	67.7	1.12	94.9	11.87	99.6

Table 2. Prolificity trait average values and relative values for the original populations and their derived inbred lines.

		PN		EUN		ET		EP	
Generation		abs.	rel.	abs.	rel.	abs.	rel.	abs.	rel.
SCV1	(S0)	1.46	100.0	1.00	100.0	1.46	100.0	2.23	100.0
	(S1)	1.20	82.2	1.00	100.0	1.20	82.2	1.57	70.4
	(SI1)	1.19	81.5	1.00	100.0	1.19	81.5	1.61	72.2
SCV2	(S0)	1.14	100.0	1.00	100.0	1.14	100.0	1.27	100.0
	(S1)	1.27	111.4	1.00	100.0	1.27	111.4	1.35	106.3
WEM	(S0)	1.50	100.0	1.00	100.0	1.50	100.0	2.02	100.0
	(S1)	1.09	72.7	1.00	100.0	1.09	72.7	1.35	66.8
	(SI1)	1.48	98.7	1.00	100.0	1.48	98.7	1.86	92.0
FW	(S0)	1.25	100.0	1.00	100.0	1.25	100.0	1.37	100.0
	(S1)	1.04	83.2	1.00	100.0	1.04	83.2	1.14	83.2
	(SI1)	1.00	80.0	1.00	100.0	1.00	80.0	1.06	77.4
CP27	(S0)	1.53	100.0	1.14	100.0	1.53	100.0	1.71	100.0
	(S1)	1.43	93.5	1.00	87.7	1.43	93.5	1.47	86.0
	(S2)	1.37	89.5	1.00	87.7	1.37	89.5	1.44	84.2
S80	(S0)	1.22	100.0	1.06	100.0	1.22	100.0	1.37	100.0
	(S1)	1.17	95.9	1.08	101.9	1.17	95.9	1.35	98.5
	(S2)	1.08	88.5	1.00	94.3	1.08	88.5	1.25	91.2

Table 3 shows the potential yield estimated for the original populations and for the lines derived from them. This estimation resulted from relating the average weight of kernels/ear (in

Table 3. Potential yield for the original populations and their derived inbred lines.

Generation		Kernel weight (g/ear)	EP	Yield (Kg/ha)
SCV1	(S0)	60.45	2.23	7,702
	(S1)	44.00	1.57	3,947
	(SI1)	55.88	1.61	5,140
SCV2	(S0)	57.40	1.27	4,165
	(S1)	48.70	1.35	3,756
WEM	(S0)	62.00	2.02	7,156
	(S1)	37.50	1.35	2,892
	(SI1)	59.43	1.86	6,316
FW	(S0)	133.00	1.37	10,411
	(S1)	80.00	1.14	5,211
	(SI1)	119.00	1.06	7,208
CP27	(S0)	92.29	1.71	9,018
	(S1)	91.84	1.47	7,714
	(S2)	80.62	1.44	6,634
S80	(S0)	76.07	1.37	5,955
	(S1)	65.08	1.35	5,020
	(S2)	58.64	1.25	4,188

grams) with the number of ears per plant (EP) and the sowing density used (57,142 plants/hectare).

Actually we have S1 lines (F:0.5 - Hr:0.5); S2 lines (F:0.75 - Hr:0.25) and SI1 lines (F:0.25 - Hr:0.75) from which we are obtaining new lines and generations by selfing.

The materials obtained to date showed low yield, by which they will be employed to obtain waxy endosperm inbred lines that later on could be used to incorporate the waxy gene (*wx*) into high yielding hard endosperm inbred lines using backcrosses. Finally the modified endosperm lines will be used to obtain high yielding waxy endosperm hybrids.

Evaluation of protein content in a maize native race from Argentina

--V. R. Corcuera and C. A. Naranjo

With the purpose of incorporating germplasm of Argentine native races to a breeding program specially designed to improve the protein content of hard endosperm maizes, individuals of the race Pisingallo were evaluated.

The Pisingallo race has hard endosperm kernels that may be of different colors (white, red, yellow) and denotes a long evolutive cycle. The seeds of Pisingallo were collected at the location of Piedras Blancas in the province of Catamarca (Argentina) by A. M. Chiavarino and C. A. Naranjo during 1991.

Fifty ears were taken at random from the original population at Piedras Blancas, and a pedigree number was assigned to each one of them. Then, fifteen kernels were taken from each ear and were sown in a separate row. In each row, the best 2 or 4 plants were selfed and identified by a second number. The selfed ears (S1 lines) were harvested by hand and fifteen kernels were taken from each. Pericarp and germ were removed to evaluate endosperm protein content using microKjeldahl method (A.O.A.C., 1985) titrating with 0.025 N H_2SO_4.

The protein content values of each sample and replicates are shown in Table 1. In Table 2 the protein content range and average value for each pedigree may be seen.

Table 1. Average protein content for each ear evaluated.

Pedigree	Sample	Replicate	Average
1265/1	12.93%	12.70%	12.81%
1265/2	10.27%	9.83%	10.05%
1265/3	8.95%	9.17%	9.06%
1265/4	8.95%	8.73%	8.84%
1267/1	13.59%	14.25%	13.92%
1267/2	8.51%	8.51%	8.51%
1267/3	10.05%	11.16%	10.60%
1267/4	7.18%	8.06%	7.62%
1272/1	13.37%	12.04%	12.70%
1272/2	9.61%	9.39%	9.50%
1276/1	10.71%	11.38%	11.04%
1276/2	10.27%	10.71%	10.49%
1276/4	8.51%	8.06%	8.28%
1280/1	12.48%	12.92%	12.70%
1280/2	10.71%	11.38%	11.04%
1280/3	8.51%	9.17%	8.84%
1284/1	10.27%	9.83%	10.05%
1284/2	9.39%	10.05%	9.72%
1284/3	5.85%	4.97%	5.41%

Table 2. Protein content range for each pedigree studied.

Pedigree	Minimun	Maximun
1265	8.84%	12.81%
1267	7.62%	13.92%
1272	9.50%	12.70%
1276	8.28%	11.04%
1280	8.84%	12.70%
1284	5.41%	10.05%

Considering the nineteen selfed ears belonging to six pedigrees of the original Pisingallo population collected during 1991, it can be noted that the average endosperm protein content varies from 5.41 to 13.92%. This wide range, found for the trait in a Pisingallo maize population from Piedras Blancas, allows us to select ears with an uppermost protein content.

The next step will be applying the classic methodology of selection by high protein content to the following pedigrees: 1265/1 - 1272/1 - 1276/1 -1280/1 -1280/2 -1267/1 and 1267/3.

Cytogenetic abnormalities in callus and plants derived from one maize embryo after 60 months in culture

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The occurrence of numerical and structural chromosome variation in cell and tissue culture and regenerated plants is now a widely accepted component of the general phenomenon of somaclonal variation (Larkin and Scowcroft, Theor. Appl. Genet., 1981). Many types of cytogenetic alterations have been described in tissue cultures and regenerated plants, for example polyploidy, aneuploidy, chromosomal rearrangements, deficiencies, dicentric chromosomes, deletions, duplications, inversions and translocations. The genotype and culture medium, mainly the kind and concentrations of plant growth regulators, influence the chromosomal aberration frequencies and types (Edallo et al., Maydica 26:39-56, 1981; McCoy et al., Can. J. Genet. Cytol. 24:37-50, 1982; Puolimatka and Karp, Heredity 71:138-144). Otherwise, Lee and Phillips (Genome 29:122-128, 1987) observed no chromosomal abnormalities in maize plants regenerated from 3 or 4 month old cultures, but 50% of plants regenerated from 8 or 9 month old cultures showed chromosomal alterations. Of these, 96% had changes in the chromosomal structure, 42% deficiencies and 19% heteromorphic pairs. This indicates that somaclonal variation is influenced not only by the medium and genotype stability, but also by the time in culture.

The objectives of this research were to determine frequency and types of chromosomal aberrations among maize somaclones, and whether cytogenetic abnormalities could explain the extreme phenotypic variation among somaclones after 60 months in culture.

Organogenic callus cultures were initiated from one Colorado Klein embryo on media supplemented with 0.5 mg l^{-1} 2,4-dichlorophenoxyacetic acid (2,4-D). Calli have been maintained by monthly subculturing on media containing 1 or 2 mg l^{-1} 2,4-D (Garcia et al., Rev. Fac. Agron. La Plata 68:15-25, 1992). For cytogenetic studies, callus was pretreated for 3 hours in 1-4-dichlorobenzene saturated solution. Callus and tassels were fixed in an acetic acid-ethanol (1:3) solution, then stained with acetic haematoxylin.

The frequencies of cytogenetically abnormal plants from 17, 32 and 52 month old callus were 30%, 95% and 100%, respectively. The frequencies of phenotypically abnormal plants from 17, 32 and 52 month old cultures were 0%, 92% and 100%, respectively. Pollen fertility also decreased with the age of cultures (Garcia et al., Rev. Fac. Agron. La Plata 68:15-25, 1992).

Cytogenetic analysis of 60 month old tissue cultures revealed great variability in chromosome number with 2n=18, 19, 20, 21, 22 and 23. Some cells showed 2 or 4 chromosomes with satellite, heteromorphic pairs and a ring of chromosomes. However, 90% of plants regenerated from these tissues had 2n=21 and 10% 2n=20. The types of chromosomal alterations observed in these plants

were deficiencies, duplications, high number of univalents, one or more extra chromosomes and inversions, chromosomes with two nucleolus organizer regions and translocations. The analysis of anaphase showed 30% normal plants and 70% with a different chromosome number at each pole and one or more chromosome bridges. All regenerated plants were phenotypically abnormal and completely sterile.

In conclusion, after 60 months on 2,4-D containing media, maize tissues showed changes in chromosome number (2n=18, 19, 20, 21, 22 or 23) and other alterations. We were only able to regenerate plants from tissues with cells with 2N=10 or 21. These plants exhibit also phenotypic and chromosome aberrations, which increased with the age of cultures.

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Expression of some maize 18 kDa HSPs result from the translation at internal AUG codons

--J. Roger H. Frappier, Robert A. Bouchard, David B. Walden and Burr G. Atkinson

Initiation of translation of eukaryotic mRNA's occurs typically at the first AUG triplet from the 5' end of the message although exceptions have been described (Suzuki et al., Eur. J. Biochem. 207:767-772, 1992). Herein, we report an exception which appears to be common among mRNA transcripts encoding the 18 kDa HSPs in radicles from heat-shocked maize seedlings. We have isolated, sequenced and characterized cDNAs encoding different members of the 18 kDa HSP family and found that the open reading frames of some transcripts contain in-frame, internal AUG codons. Transcription and translation of 18 kDa HSP cDNAs containing internal AUGs in the transcribed RNA appear to synthesize a polypeptide initiating from each AUG codon. Furthermore, an HSP 18 cDNA was expressed in the Invitrogen pTrcHis expression system containing a 33 amino acid leader sequence, resulting in the production of three proteins (corresponding to the internal HSP AUGs). Moreover, translation of hybrid-selected poly(A)+ mRNAs from radicles or plumules of heat-shocked maize seedlings (utilizing oligonucleotide sequences for hybrid selection which are specific for each cDNA) results in the synthesis of different proteins which correspond to the number of AUGs in the hybrid-selected transcript. Both the deduced and observed molecular mass and isoelectric point of each of the proteins synthesized from the AUG most proximal to the 5' end of the transcripts as well as those synthesized from internal AUGs correspond to a member of the 18 kDa HSP family synthesized in vivo. We suggest that the expression of some members of the maize 18 kDa HSP family results from initiation of translation at internal AUG codons.

In situ hybridization of 18 kDa HSP antisense RNA in maize root tips using digoxigenin detection

--R. I. Greyson, E. Banisikowska and D. B. Walden

We have reported on the inducible heat shock (hs) genes of maize and the modulated transcriptional activity of some, but not all, of these genes during microsporogenesis and gametogenesis (Atkinson et al., Dev. Genetics 14:15-26, 1993; Bouchard et al., Maydica 38:135-144, 1993). Brothers et al. (MNL 67:73-74, 1993) reported on the distribution in maize root tip cells of polyclonal antibodies raised to the 18 kDa family of HSPs. The antibodies were raised against protein isolated from hs root tips.

To extend our study, we undertook to locate the sites in root tip cells of mRNA for the 18 kDa family of HSPs. We report below the hybridization procedures employed on root tip sections for the antisense RNA (and sense controls) to the mRNAs containing the open reading frame (ORF) from the family of 18 kDa HSPs.

Preparation of probes. Several of the 18 kDa maize heat shock protein (HSP) genes (Goping et al., Plant Mol. Biol. 16:699-711, 1991) were cloned into pBluescript II Sk- vector containing T7 and T3 RNA polymerase promotors. Template DNA of scMHSP 18-9-2 (a 342 bp fragment containing the ORF) was linearized prior to RNA synthesis and both sense (control) and antisense transcripts were synthesized. The scMHSP 18-9-2 fragment was cut with *Xba*I + T7 polymerase for the sense strand and *Pst*I +T3 polymerase for the antisense strand.

After restriction enzyme digestion, the template was purified by phenol/chloroform extraction according to standard procedures and dissolved in DEPC-H_2O. Transcripts were produced from 1 µl of template DNA and 6-8 µl of digoxigenein (DIG) RNA labelling mixture (Bohringer-Mannheim) [60-80 mM of all nucleotides]. The concentration of the sense and antisense probes was 10 ng per 1 µl of hybrization solution (buffer prepared according to Langdale (In Freeling and Walbot, eds., The Maize Handbook, pages 165-180, Springer-Verlag, 1993). We used 15 µl per slide (for a 22 mm^2 cover slip).

Hybridization procedures. 10 µm longitudinal sections (l/s) of FAA-fixed, paraffin embedded root tips from heat shocked and

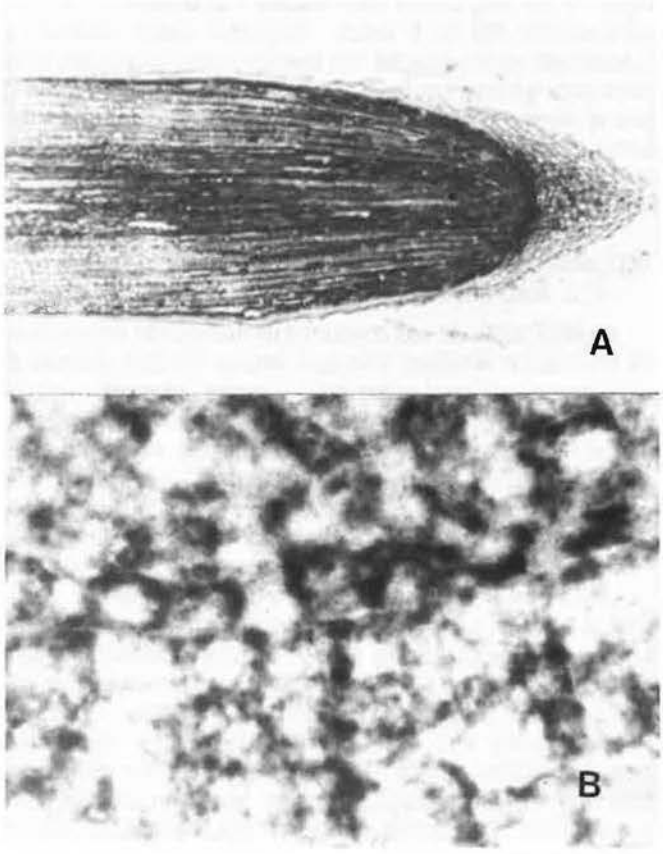

Figure 1A: Near-median l/s through maize root tip documenting the typical staining response to the antisense DIG probe (X25). Figure B: View of response of cells from the region of the root tip meristem and the root cap to antisense DIG probe (X900).

control seedlings (4-5 day) were tested for in situ hybridization of hs mRNA using a modified procedure from Langdale (1993). Depending upon the length of treatment with the substrate solution (currently 10 h) duration and temperature of post hybridization washes, obvious and consistent hybridization (dark blue-purple staining) was observed with the antisense mRNA of the 18-9-2 clone (Fig. 1A). Considerably lighter or no staining was detected with the sense probes on hs root tip sections and with both sense and antisense probes on sections of control root tips.

The hybridization was distributed unevenly throughout the 1cm portion of the root with the greatest intensity towards the meristem (Fig. 1A). Cytologically the staining was restricted to the cytoplasm with dense accumulations and with many dispersed punctate sites (Fig. 1B). Attempts to quantify the different responses of different tissues of the root are continuing as are comparative studies with probes derived from other members of the 18 kDa family of HSPs.

RI mapping of two ubiquitin sequences in maize

--Dan Maillet, Burr G. Atkinson and David B. Walden

In MNL 67:75 (1993) we communicated the location of a ubiquitin fusion protein gene *uwo1* (MubG7), which was mapped to the long arm of chromosome 8 position 92 (similar to *umc7*) with the RI families TxCM and TxxCo. We now report the map positions of two more ubiquitin sequences that have been isolated and characterized (Liu et al., MNL 67:74, 1993; Bouchard et al., Maydica 38:135-144, 1993). Sequence *uwo2* (MubG10), a second ubiquitin fusion protein gene, has been assigned to a sub-centromeric region of the long arm of chromosome 1 at position 74 using a gene-specific 550 bp 5' probe. Sequence *uwo3* (MubG9), a polyubiquitin which encodes five tandem copies of ubiquitin in its open reading frame, has been mapped to a distal region of the long arm of chromosome 4 between positions 155 and 159 (between *bnl15.07* and *bnl8.23*) employing a gene-specific 1.2 kb 5' sequence. Ascertainment of the map positions of other maize ubiquitin sequences is underway.

RFLP analysis of genotypic variation in callus

--K. J. Bates and D. B. Walden

An RFLP analysis was conducted on maize DNA derived from six time-course 'windows': immature embryo (20 day), plumule (5 day), mature leaf, and callus aged 4 weeks, 12 weeks, and 18 weeks. The generation of callus tissue and the variation observed was reported in MNL (67:75,1993). Samples from genotypes A188, A632, B37, B73, CO159, F2, M14, Mo17, N28, Oh43, ONTARIO FLINT, VA26, W23, and W64A were digested with *Eco*RI, *Bam*HI, *Hin*dIII, *Bst*I, or *Eco*RI/*Bam*HI and probed with a 14 kb *Eco*RI-waxy fragment, and 2 ubiquitin clones. Variation was observed for all waxy/enzyme combinations at each 'window'. Variations in fragment size and number, indicative of genotypic instability was observed particularly during the callus 'windows'. All data were generated using the DIG (digoxygenin) detection system, the method described by our lab in MNL (67:74, 1993). Genotypic RFLP grouping was observed at each 'window' for all enzyme/waxy combinations. This report identifies the differences observed for the genotypes for three restriction enzyme/waxy combinations at the immature embryo and callus (4, 12 and 18 week) 'windows'.

The most common restriction fragment pattern observed for *Eco*RI/waxy hybridizations of seven genotypes was a single fragment with an increase in fragment number at the 12 week callus stage. An increase in fragment number was observed only after 18 weeks of callus culture for Ontario Flint. In addition, a decrease in fragment size was observed in four of five genotypes after 18 weeks, however, A188 remained unchanged. Genotypic grouping in size RFLP was observed for DNA from all four 'windows' (immature embryo and callus at 4, 12 and 18 weeks). A trend towards increasing fragment size was observed for four of seven genotypes until the 12th week of callus, with a size decrease observed at the 18 week window. Deviations from this pattern included Ontario Flint, W64A and B73. An increase in fragment size was observed from the immature embryo 'window' to the first four weeks of callus only; fragment size stability was observed in the 12 and 18 week 'windows'. W64A also showed an increase in fragment size until the 12 week 'window', then stabilized. B73 exhibited the most deviant pattern, with a decrease in fragment size and number between the 4, 12 and 18 week callus windows.

RFLP's for seven genotypes were observed from *Hin*dIII/waxy hybrizations. Five of seven genotypes exhibited a single fragment for immature embryo and callus at 4 and 12 weeks, with an increase in fragment number at the 18 week 'window' for three of the five genotypes. B73 and B37 exhibited fragment number stability for all four stages. A decrease in fragment size was observed for three of seven genotypes. The smallest fragment observed was at the four week callus stage for B73. Both B73 and B37 exhibited an increasing size trend as callus aged. Oh43 exhibited a completely different, unstable size pattern, whereas A188 exhibited the most stability in fragment size, for the same fragment was observed in all four 'windows', even as a double restriction fragment pattern at 18 weeks. Overall, Ontario Flint exhibited the most variation in fragment size and in number when the immature embryo, and callus at 4, 12, and 18 week 'windows' were compared.

RFLP's for eight genotypes were observed from *Bam*HI/waxy hybridizations. For five genotypes, an increase in fragment number at the 12 week callus phase was observed, with a decrease to a single fragment at the 18 week 'window'. A188 exhibited the most variation in fragment number, with single, double and triple restriction pattterns observed. A single fragment was observed for Ontario Flint at all 'windows' except the immature embryo stage, where a triple fragment pattern was found. All genotypes exhibited band sizes within a 3-5 kb size range, however, four of eight genotypes showed an increasing size trend as callus aged. Larger fragments up to 13 kb were observed at the 12 week stage for Ontario Flint, M14 and Oh43. Most changes observed amongst the genotypes and subsequent 'windows' involved fragment number alterations, with sizes remaining within the 3-5 kb range.

For *Hin*dIII and *Eco*RI digests, most double fragment patterns included a common sized band as well as one larger, > 30 kb fragment. These fragments were prevalent in DNA isolated after the 12 - 18 week callus phase.

Collectively, these data may give an indication of the loss of restriction sites due to either direct changes in the DNA, its packaging, or other factors interacting with the DNA after isolation. However, these patterns were observed for many genotypes and were particular in size, indicating a deliberate change, instead of a random process. In general, the most frequently observed *Eco*RI and *Bam*HI fragment changes were at

the 12 week callus window, whereas most *Hin*dIII RFLP's were observed after the 18th week of callus. Further research is required to address the question of whether these apparent changes in DNA during callus reproduction could be fore-runners of somaclonal variants.

Analysis of environmental effects on RFLP stability in maize inbreds

--A. S. Richman and D. B. Walden

The utility of RFLPs as molecular markers relies in part on their stability through successive generations even when exposed to different environmental conditions. In order to investigate the potential contribution of "environment" to RFLP stability, six established inbred lines were maintained under two environmental regimes for seven generations and then analyzed.

In 1978 seed from each of maize inbreds Oh51A, Oh43, Mo17, B73, W64A and A632 were collected and separated into two lots, one designated for Molokai, Hawaii and the other for London, Canada. Sibling (or isogenic) lines were maintained in their respective locations through self pollination for seven generations, and in the final generation a sample from each isogenic line was grown at the alternate location. It is implicit in this study that no apparent phenotypic variation was introduced during the pedigreed breeding, thereby confounding any observed variation in the DNA. To document the absence of phenotypic variation, three nursery plots, one at London in 1992 and two at London in 1993, included seed from both locations. Data were collected on node number, plant height and tassel branch number. No obvious differences were visible based on observations of field grown material at the London nursery.

Genomic DNA was isolated from six day etiolated plumules following a modified urea extraction protocol (Shure et al., Cell 35:225-233, 1983). DNA was digested using one of four restriction enzymes (*Eco*RI, *Hin*dIII, *Bam*HI and *Bst*I) according to manufacturer's instructions (Pharmacia). Fragments were separated by gel electrophoresis then capillary transferred to positively charged nylon membranes (Boehringer Mannheim, BM). Four gene specific sequences were random primer labelled using the digoxigenin system from BM. Detections were carried out as outlined by Engler-Blum et al. (Analyt. Biochem. 210:235-244, 1993) with modifications based on Maillet et al. (MNL 67:75, 1993). The sequences employed as probes were: Waxy (Wx), a 14 kb *Eco*RI fragment obtained from P. Dietrich; BI, a 1.9 kb *Eco*RI cDNA from V. Chandler; scMubG7-J, a 2.0 kb *Eco*RI/*Xba*I fragment, specific for the 5' region of a ubiquitin fusion protein gene; C1-5C, a 1.0 kb fragment specific for a polyubiquitin gene. The maize ubiquitin clones were provided by L. Liu. In total, sixteen clone enzyme combinations (CECs) were used to investigate all entries for each of the six genotypes.

There were no differences observed in either the number or size of fragments between the London and Hawaiian isogenic lines (all inbreds). We conclude that these RFLP markers remained stable over seven generations under the two different seed production locations. Though intervening generations were not examined, it seems unlikely that mutations arose and subsequently reverted to their previous state. Viable material is available for analysis; had there been any differences found they could have been traced to their origin.

All CECs revealed polymorphisms among at least two of the inbreds, while on average 4.2 different banding patterns were produced per CEC. The number of CECs used was not sufficient to determine accurately the distances among inbred lines as outlined by Smith et al. (MNL 65:66, 1991); however, it is interesting to note that the highest genetic similarity, as calculated according to Nei and Li (PNAS 76:5269-5273, 1979), was obtained between Mo17 and W64A (0.597), two inbreds that share a common parent (187-2).

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The absence of debranching enzyme activity and the presence of phytoglycogen in the germinating seeds of *sugary1* mutants and commercial sweet corns

--David Pan and Oliver E. Nelson

In an earlier study (Plant Physiol. 74:324-328, 1984), we reported that the *sugary1* mutants have reduced debranching enzyme activity in the developing endosperms. The debranching enzymes of nonmutant endosperms can be separated into three fractions on a hydroxyapatite column; the extract of *sugary1* endosperms lacks the first fraction; the second and third fractions are also much reduced in activity. In no case is debranching enzyme activity completely absent from *sugary1* mutant endosperms. In this note, we report that debranching enzyme activity was not detected in the extract of the germinating seeds of *sugary1* mutants nor commercial sweet corns that have *sugary1* alleles. As shown in Table 1, the absence of debranching enzyme activity in the germinating seeds of these genotypes is correlated with the presence of phytoglycogen in seed tissues except for the anomalous *su1-starchy* allele reported by Dahlstrom and Lonnquist (J. Hered. 55:242-246, 1964). While somewhat variable in expression, the homozygous *su1-starchy* seeds are starchy in appearance. The *su1-starchy* allele is recessive to the *su1* alleles that condition the production of the typical wrinkled seeds. In developing endosperms at 22 DAP, the *su1-st* endosperms have 56% of the debranching enzyme activity of a *Su1* control while other *su1* mutants have 9-30% of the control value. Thus, the data of Table 1 are compatible with our previous finding that reduced debranching enzyme activity is correlated with the production of phyto

Table 1. The presence of phytoglycogen and the absence of debranching enzyme activity in the germinating seeds of *sugary1* mutants and commercial sweet corns.

Genotypes	Debranching enzyme activity	Phytoglycogen
Golden Beauty Hybrid	-	+
Wis. Golden 900	-	+
Seneca Chief	-	+
Early Sunglow	-	+
So Sweet	-	+
Tendertreat	-	+
Jubilee	-	+
Sugar Buns	-	+
Mainliner	-	+
Commander	-	+
Silver Treat	-	+
Natural Sweet 9000 (*sh2*)	+	-
Golden Cross Bantam	-	+
NK 199	-	+
Miracle	-	+
su1-starchy	+	+
W64A *su1-R*	-	+
Oh43 *su1-R*	-	+
bt1	+	-
bt2	+	-
sh1	+	-

- not detected
+ presence.

glycogen by developing endosperms. It has been known that the germination rate of sweet corn seeds is usually lower than dent corns. We suggest that the lack of a debranching enzyme activity that would hydrolyze the α,1-6 glycosidic bonds of amylopectin and phytoglycogen would limit their complete degradation during germination and may partially account for the poor germination of sweet corn. The digestion of oligosaccharides extracted from germinating seeds of a *su1-R* (W64A) mutant and *Su1* control with isoamylase and β-amylase to hydrolyze α,1-6 and α,1-4 linkages respectively, indicates that there are many more α,1-6 glycosidic bonds in the oligosaccharides of *su1-R* mutant than nonmutant seeds (Table 2). The evidence supports the hypothesis that debranching enzymes have an important role in degrading amylopectin and phytoglycogen in germinating seeds.

Table 2. Degradation of oligosaccharides extracted from germinating seeds of *sugary1* mutant and nonmutant by isoamylase and β-amylase.

	% increase in reducing sugar liberated from oligosaccharides after digestion	
Enzymes	*su1-R* (W64A) mutant	Nonmutant
Isoamylase	10.2	7.9
β-amylase	7.4	18.6

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Evaluation of tropical inbred lines for resistance to *Fusarium moniliforme* ear rot

--D. Jeffers, S. K. Vasal, S. McLean, G. Srinivasan

Advanced lowland tropical maize inbred lines developed at CIMMYT were evaluated for resistance to *Fusarium moniliforme* ear rot in a preliminary study in Poza Rica, Vera Cruz during the 1993A growing season using one, 5 m row subplots. Two inoculation treatments were used to compare infection levels: 1) the nailpunch/sponge technique with a spore suspension of 5.0×10^5 spores/ml applied through the husk in the middle of the ear (Drepper et al., Plant Dis. 74:952-956, 1990), and 2)application in the silk channel of a 1 ml spore suspension of 5.0×10^5 spores/ml prepared in a 4 M sucrose solution. Inoculations were made 7-10 days after 100% extrusion of the silks. Ears were evaluated at physiological maturity using a visual rating scale of 1-6 for percent infection (1=0%, 2≤10%, 3≤25%, 4≤50%, 5≤75%, and 6≤100%). A total of 164 lines, including 58 announced CIMMYT maize lines (CML), and 53 promising lines from both white and yellow grain type, were evaluated. From the 1993A results 23 inbreds with the lowest levels of *F. moniliforme* infection and 2 lines used as checks were evaluated in 1993B in a split plot design using the same two inoculation techniques and 4 replications. Subplots were one, 2.5 m row. The purpose of this trial was to evaluate the lines for resistance to infection, to determine if the sucrose solution method improved the level of infection compared to the standard nailpunch technique presently being used, and to determine if there was a correlation between the two techniques. Results obtained indicate that the sucrose technique was significantly better than the standard nailpunch technique (p=0.05) as determined by Fisher's LSD test for percent infection. The correlation between the rankings of the lines for resistance to ear rot with the two techniques was 0.81. Five inbreds in the evaluations had ratings between 2 and 3 with the two treatments and are considered to have high levels of resistance to *F. moniliforme* ear rot infection. An additional 3 lines are included which had low ear rot ratings from earlier evaluations. Another season of ear rot evaluations will be performed with these lines to confirm our results. The lines are:

CML 48 Porillo 8073-11-1-1-##
(Pop 22 TSR)-4-3-1-3-1-BB-####
CML 52 Sta Rosa 8079-22-2-2-##
CML 1 Pop 21 C5 HC57-1-2-B-##
Sint Am TSR-76-1-1-1-2-BB-####
(Across 7643 x 43 F7)-2-3-4-3-BB-####
CML 40 Pop 36 C5 HC144-2-2-B-###
Sint Am TSR 23-3-1-2-3-BB-####

Evidence for the tri-hybrid origin of *Tripsacum andersonii* Gray

-- Marc Barré, Julien Berthaud, Diego González-de-León and Yves Savidan

Tripsacum andersonii has 64 chromosomes (Levings et al., Crop Sci. 16:63-66, 1976). Since this counting, this species was postulated to be the result of a hybridization event between a *Zea* (10 chr) and a *Tripsacum* (3x=54) species. De Wet et al. (Amer. J. Bot. 70:706-711, 1983) proposed *T. latifolium* (2n=2x=36) as the putative *Tripsacum* parent based on its highly unique morphological features. Studies by Talbert et al. (Amer. J. Bot. 77:722-726, 1990) have suggested that the *Zea* genome is from *Zea luxurians* and the *Tripsacum* genome is from *T. maizar* or *T. laxum* rather than from *T. latifolium* (2x). This conclusion was based on analysis of restriction fragments revealed by an rDNA probe (pzmr1) of *Bam*HI-digested DNA: clearly, a 1.6 kb band is present in *T. andersonii*, *T. maizar* and *T. laxum* but not in *T. latifolium* (2x), *T. dactyloides* or *T. peruvianum*.

Since then we have surveyed the diversity of wild *Tripsacum* populations in Mexico and have assembled a large collection. Among the *T. latifolium* accessions, we found two types that have the same gross morphology except that one has paired sessile spikelets and is diploid (as described for the botanical type of the species), while the other is triploid and has paired spikelets, one sessile, one shortly pedicellate. From these unpublished observations we had derived the hypothesis that triploid *T. latifolium* would be a hybrid between a diploid *T. latifolium* and another *Tripsacum* species belonging to the *Fasciculata* section (to explain pedicellate spikelets) and, as a corollary, that this hybrid is the putative *Tripsacum* progenitor of *T. andersonii*.

Using the same probe/enzyme combination as Talbert et al., we analyzed DNA samples from different species of *Tripsacum* to determine the occurrence of the 1.6 kb band and test the robustness of a conclusion based on the presence/absence of such a band. Some results are shown in Table 1 and Figure 1.

Three bands of interest were detected in our collections: "A", a high intensity 1.6 kb band similar to that described by Talbert et al.; "B" a low intensity 1.6 kb band; and "C" a low intensity 1.9 kb band that is always found when B is present (Fig. 1).

We believe that bands B and C are here reported for the first time and that they are essentially different from band A but have, so far, no bearing on the evidence used for supporting our hypothesis on the origin of triploid *T. latifolium*.

As was recorded by Talbert et al., band A is absent from diploid *T. latifolium*. It is also absent from most other species with the exception of *T. manisuroides*, *T. maizar*, *T. laxum*, triploid *T. latifolium* and *T. andersonii* (Table 1). This narrow distribution

Table 1. Distribution of bands A (1.6 kb, intense), B (1.6 kb, faint) and C (1.9 kb, faint) in samples from our *Tripsacum* collection (S.A. = South America; MEX = Mexico).

Pop #	Species	Origin	Ploidy	Pattern	# in Fig. 1
507	*andersonii*	S.A.	64 chr.	A	a
528	*australe australe*	S.A.	2x	none	
544	*australe hirsutum*	S.A.	2x	none	
606	*cundinamarce*	S.A.	2x	none	
612		S.A.	2x	none	
57	*bravum*	MEX	2x	BC	
15		MEX	4x	BC	
38		MEX	4x	BC	
121		MEX	4x	BC	
127		MEX	4x	BC	
132		MEX	4x	BC	
148		MEX	4x	BC	
111	*dactyloides hispidum*	MEX	2x	BC	
151		MEX	2x	BC	
67		MEX	4x	BC	
37	*dactyloides mexicanum*	MEX	4x	BC	
38		MEX	4x	BC	
39		MEX	4x	BC	
40		MEX	4x	BC	
60		MEX	4x	BC	
83		MEX	4x	BC	
156		MEX	4x	none	
14	*intermedium*	MEX	4x	BC	
96	*jalapense*	MEX	4x	BC	
126	*lanceolatum*	MEX	4x	BC	
142		MEX	4x	BC	
77	*latifolium*	MEX	2x	none	c
106		MEX	2x	none	b
73		MEX	3x	A	d
109		MEX	3x	A	e
76	*laxum*	MEX	2x	A	f
95	*manisuroides*	MEX	2x	A	
3	*maizar*	MEX	2x	A	h
21		MEX	2x	A	i
99		MEX	2x	A	g
39	*pilosum*	MEX	2x	none	
46		MEX	2x	none	
47		MEX	2x	none	
139		MEX	2x	none	
68		MEX	4x	BC	
49	*zopilotense*	MEX	2x	BC	

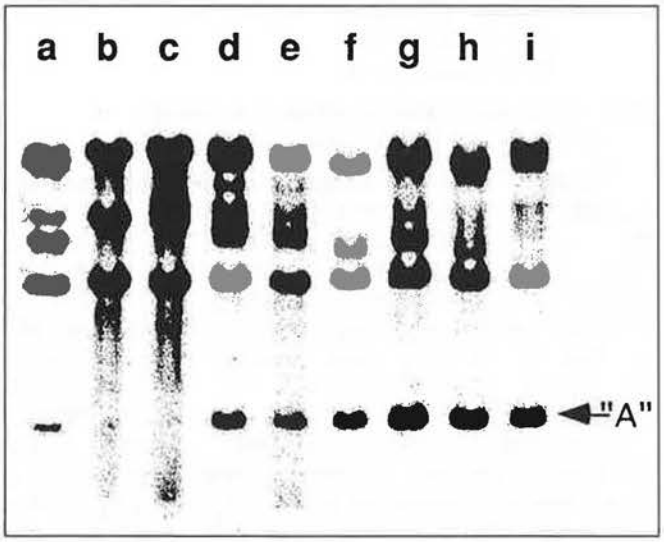

Figure 1. Luminograph of Southern blots probed with rDNA probe pzmr1 after digestion with *Bam*HI. Lanes: a: *T. andersonii*; b&c: *T. latifolium* (2x), d&e: *T. latifolium* (3x), f: *T. laxum*; g h&i: *T. maizar*.

suggests that one of the latter species could be the putative *Tripsacum* progenitor of *T. andersonii*. On the basis of their morphological differences with *T. andersonii*, it is improbable that *T. manisuroides*, *T. maizar* or. *laxum* would be good candidates. By contrast, the morphologically unique resemblance between *T. latifolium* and *T. andersonii* (supporting De Wet et al.'s observations on the diploid), as well as precisely the adequate number of chromosomes (54) and the presence of the diagnostic A band, make triploid *T. latifolium* the best putative progenitor of *T. andersonii*. Under this hypothesis, we would also propose that the two collected triploid *T. latifolium* accessions, both having pedicellate spikelets, may well be derived from hybridization events between diploid *T. latifolium* (no 1.6 kb band) and *T. laxum* (1.6 kb band), which has pedicellate spikelets and is the only species that we have found to be sympatric with *T. latifolium* in our surveys.

In conclusion, we propose that the data discussed above support the hypothesis that the genetic constitution of *T. andersonii* was derived from two hybridization events :

1) *T. latifolium* (2x) x *T. laxum* (2x) => *T. latifolium* (3x=54)

2) *T. latifolium* (3x) x *Zea luxurians*(2n=20) => *T. andersonii* (54+10 chromosomes)

The first event may have occurred at least twice given that the two *T. latifolium* populations have different isozyme profiles (data not shown). That the second event has probably been unique is strongly suggested by at least two lines of evidence: more than 20 different accessions of *T. andersonii* from several South American countries show exactly the same morphology and the same isozyme pattern (data not shown).

T. andersonii may therefore be an example of how an apparently very improbable set of events can give rise to a new species.

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Identification of a RAPD marker associated with *Rf3*

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Cytoplasmic male sterility (cms) is a maternally inherited trait, widely used in many crop plants for the production of hybrid seed because male fertility can be restored by the action of one or more nuclear restorer genes. cms has been extensively studied in maize, where four major cytoplasm types have been described: the normal cytoplasm (N) and three cytoplasm types (designated as C, T and S) causing male sterility, distinguishable on the basis of their interaction with specific nuclear restorer genes.

Rf3, a nuclear gene mapped on the long arm of chromosome 2, is required for the restoration of fertility in cms type S (Laughnan and Gabay-Laughnan, Annu. Rev. Genet. 17:27-48, 1983). *Rf3* acts as a dominant gene with gametophytic expression: cms-S plants heterozygous for the restorer locus (*Rf3 rf3*) produce 50% normal pollen, according to a 1:1 segregation pattern of the two alleles in microspores after meiosis. Little is known about the nature or function of *Rf3*, or about the mechanism(s) by which male sterility is overcome.

In order to identify molecular markers tightly linked to *Rf3*, we have analyzed Near Isogenic Lines (NIL) sharing the same Ny821 genetic background by means of RAPD markers: differences in the amplification patterns obtained from NILs should indicate polymorphisms in a region linked to *Rf3*. Amplifications have been performed on DNA extracted from all four genetic combinations of N and S cytoplasm with the two allelic forms of the restorer in homozygous condition, to avoid misinterpretations due to the amplification of cytoplasmic components. Several decamers from Operon Inc. (Alameda, CA) have been used under the conditions

indicated by Williams et al. (in Methods in Enzymology, Academic Press, Orlando, Florida, 1991).

One of the primers tested has revealed an amplification product of approximately 1900 bp, present in the combinations cyt-N *Rf3 Rf3* and cyt-S *Rf3 Rf3*, but not in the combinations cyt-N *rf3 rf3* and cyt-S *rf3 rf3* (Fig. 1). DNA corresponding to the

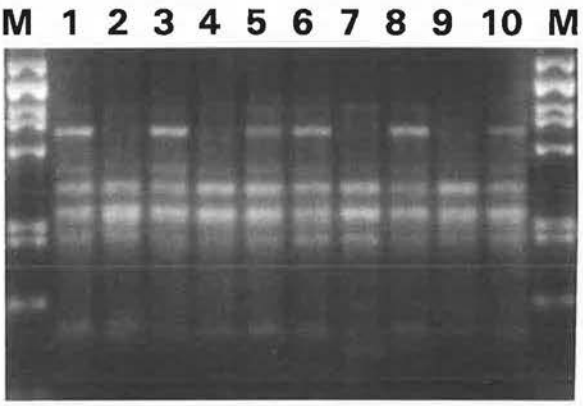

Figure 1. RAPD amplification of four Ny821 Near Isogenic Lines. Lanes 1, 6: Ny821 cms-S *Rf3 Rf3*. Lanes 2, 7: Ny821 cms-S *rf3 rf3*. Lanes 3, 8: Ny821 normal *Rf3 Rf3*. Lanes 4, 9: Ny821 normal *rf3 rf3*. Lanes 5, 10: Ny821 cms-S *Rf3 rf3*. M: lambda *Pst*I marker. *polymorphic band observed.

polymorphic band has been extracted and used as a probe onto genomic DNA in a Southern blot. Our results indicate that this DNA corresponds to sequences present in low or medium number copy in the maize genome. We are now performing cosegregation analysis on a backcross population in order to confirm the linkage of this sequence with *Rf3*.

This result, although preliminary, is of particular interest because it indicates the possibility of saturating the region surrounding *Rf3* with closely linked molecular markers.

Mapping QTLs for pollen thermotolerance in recombinant inbreds

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Pollen thermotolerance is a major component of yield stability under high temperature stress. Here we report its genetic dissection through RFLP analysis of a recombinant inbred line population (T232 X CM37, provided by B. Burr, Brookhaven National Laboratory). The character was measured in vitro as the degree of injury [I = (1 - T/C) * 100] of pollen germination ability (IPGG) and of pollen tube growth (IPTG), caused by high temperature treatment (T = 41 C) in comparison with control (C = 27 C) growth conditions. Both IPGG and IPTG showed a typical quantitative distribution among RIs and high heritability: h^2 = 0.64 and 0.68 respectively. Regression analysis between each RFLP locus and trait expression identified several markers significantly correlated with pollen thermotolerance. They are shown in Figure 1A and B, where each marker is represented by its R^2 value. In order to avoid false QTL assignments, the correlation matrices between all significant markers for each trait were analyzed; in each group of correlated markers only the one with the highest R^2 value was considered indicative of the presence of a QTL in that region. The results show that at least five genomic regions are involved in IPGG and six in IPTG determination, and that the two traits are controlled by different sets of genes. A comparison between the regions identified and those (also determined in this study) containing putative QTLs for pollen germination and tube

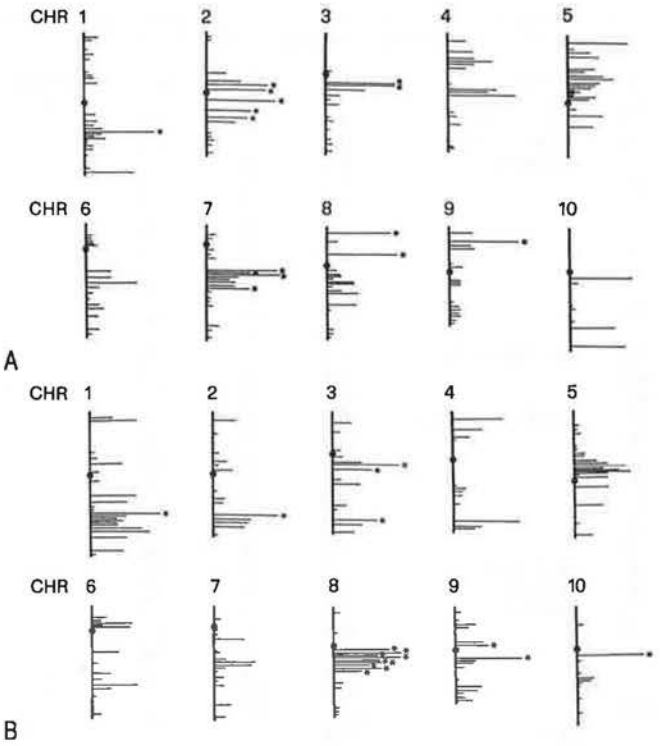

Figure 1. Localization of putative QTLs controlling IPGG (A) and IPTG (B) in maize. Horizontal bars indicate degree of correlation between RFLP loci and the characters in terms of R^2. Significant loci are indicated by asterisks.

growth in non-stress conditions, indicate that the "base" and the injury traits are largely independent.

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Sequence analysis of an *opaque2* mutant of *Zea mays*

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The *opaque2* mutant of the Bianchi *o2* maize line, recovered as a spontaneous mutation in the early sixties (Bianchi, personal communication), previously analysed at the genetic level (Nelson, Maydica 12:81-96, 1967; Salamini, Cold Spring Harbor Symp. Quant. Biol. 45:467-476, 1980) and more recently investigated at the molecular level, has been introgressed in four different lines: W22 and A69Y (Istituto Sperimentale per la Cerealicoltura, Bergamo, Italy), NYR and 3316 (Dipartimento di Genetica e Biologia dei Microorganismi, Milan, Italy). Southern and Northern analysis of these different lines reveals no difference in regard to *o2* gene and transcript patterns. It should be remembered that this *o2* allele produces a transcript normal in size and similar in level in respect to the wildtype line (Dolfini et al., Dev. Genet. 13:264-276, 1992). In order to obtain the cDNA clone of this allele we synthesised four oligonucleotides: one in the leader sequence of the *O2* wildtype allele, one in the trailer sequence, and two internal to the coding region (Schmidt et al., PNAS 87:46-50, 1990). Specific amplification of the two regions of the *o2* allele has been carried out by PCR on total RNA extracted from 20 DAP (days after pollination) endosperms of the A69Yo2 maize line and reverse transcribed using oligo-dT primer and MoMuLV reverse tran-

scriptase. The amplified sequences were treated with the large fragment of DNA polymerase I (Klenow fragment) and cloned in the pBSKS vector (Stratagene). We obtained two clones that represent the whole cDNA sequence of the *o2* Bianchi allele, named *o2-Italian*, one spanning from about 100 bp before the ATG codon to base 936 of the coding sequence (after the leucine zipper motif), and the second from base 667 of the coding sequence (before the basic domain) to some 40 bases after the stop codon. More copies of the two clones have been prepared, coming from different amplification reactions, in order to verify the reliability of the method. All these clones have been sequenced and compared, showing no significant difference among the nucleotide sequences. However, comparison of the deduced amino acid sequence of *o2-Italian* with the *O2* wildtype sequences of both A69Y and W22 maize lines revealed various differences. The most important difference is a deletion of a sequence of 7 amino acids in the basic domain.

As the basic domain of the O2-bZip transcriptional factor is involved in DNA binding, these preliminary data suggest the possibility of a loss of function of the *o2-Italian* allele due to the lack of binding activity. This hypothesis is supported by the results of Southwestern experiments carried out using oligonucleotides containing the O2 target sequence as probes. Moreover, the deleted amino acids include the short RKRK sequence which constitutes the first part of the bipartite NLS (nuclear localising sequence) contained in the *opaque2* basic domain (Varagona et al., Plant Cell 4:1213-1227, 1992). This could lead to lower efficiencies in transporting O2 to the nucleus, even if O2 contains another short low-efficiency NLS in the amino-terminal region.

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Pollen-specific peroxidase *Px2*

--Emil E. Khavkin and M. V. Zabrodina

Among numerous maize peroxidases (MNL 67:83, 1993), the pollen-specific *Px2* is of particular interest. The enzyme activity was absent from any other (seedling and tassel) tissues, including pre-shedding anthers, and rapidly increased in the pollen during the first hours after shedding. By comparing peroxidase patterns of the pollen extracted with hypo- and isotonic buffer solutions, we suggest that *Px2* is apparently located in the exine and might participate, in a yet undefined way, in pollen recognition and germination.

Two *Px2* allelomorphs were found, with the predominant fast isozyme in 2/3 of screened inbreds. As a first step to mapping *px2*, we compared peroxidase isozyme patterns in the inbreds commonly employed as parental lines for molecular mapping. Both B73 and Mo17 had the fast *Px2* allelomorph, whereas Tx303 and CO159 differed by this marker, with the slow and the fast isozymes, respectively.

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Are there clusters of growth-related genes in maize?

--Emil E. Khavkin and Ed Coe

In the maize genome, genes with phenotypical expression related to growth and development appear to form clusters about 10 to 30 cM long distributed nonrandomly along the chromosomes. A typical cluster includes mutants expressing retarded stem growth, changed attitude and disturbed growth of leaves, stems and roots, or their components, reduction and various malformations of inflorescences, and vivipary. This pattern is repeated , with considerable consistency, in different regions of the genetic map. Admittedly our identification of clusters and their boundaries is being done arbitrarily, but the number of clusters that can be identified may be as many as 16. The combined length of the clusters is about 30% of the total map length.

At first glance at the maize genetic map, we observe: (1) an apparently uneven distribution of growth-regulating genes (GRGs) in the maize genome, and (2) an obvious regularity of gene constellations in different chromosomes. Clusters of closely mapped GRGs comprise much the same categories of (a) genes governing hormone-sensitive changes in plant growth and development, complemented in most constellations with (b) genes apparently related to hormone metabolism and sensing, and (c) master genes that manifest profound influence on spatial and temporal pattern of cell and tissue differentiation. An example that illustrates the point is the region from *an1* and *id1* through *kn1* and *lw1* on chromosome 1, approx. 40 cM long, where these categories are represented. The 15-cM region including *rd1*, *py2*, *vp8*, *tls1*, and *ts6* might be considered to be part of the same cluster, or separate, but the criteria by which we have done this first-approximation suggest that it would more likely be separate.

The majority of already mapped QTLs for plant growth, architecture and productivity, and master genes apparently related to transcription factors, participating in spatial and temporal control over plant development and /or hormone-response functions, map within these clusters. The *an1...kn1* region of chromosome 1, for example, displays QTLs for a number of relevant traits studied by Doebley and Stec (1991) and by Stuber et al. (1992).

We suggest that these clusters are functional units comprising genes for environmental sensors and signal transducers, receptor sites to translate environmental and hormonal signals to growth machinery, and master genes to govern critical spatial and temporal transitions in cell growth and differentiation. When clustered in such a functional unit, genes expressed in concert gain more efficient short-distance cis-control or proximity control by transcription factors engaged in protein-protein and DNA-protein interactions. The interactions with different factors may provide a great diversity of growth and developmental reactions to a limited number of environmental stimuli. Some clusters quite distant on the map might also interact in trans if clusters come into spatial proximity in the interphase nucleus, and if some clusters can be identified as "incomplete" they are candidates for such trans-complementation.

Many heritable traits concerning plant form, growth and development are well-documented and mapped (Coe et al., in: Corn and Corn Improvement, 1988; Sheridan, Annu. Rev. Genet. 22: 353-385, 1988), and recently rapid progress has been made in the cloning and sequencing of several GRGs (Freeling et al., BioEssays 14:227-236, 1992). Expression of GRGs provides for hormonal regulation, i.e., production, degradation and interaction of endogenous hormones as well as response to endogenous or exogenous hormonal signals, including transduction of signals and such loosely defined processes as commitment, competence, determination, evocation or sensitivity (Trewavas and Cleland, Trends Bioch. Sci.

8:354-357, 1983). While environmental changes induce profound effects on hormone content and distribution, some environmental effects are not mediated by hormones. Yet in both cases there must be genes for sensors to translate environmental signals into differential gene expression. On the opposite end of this GRG chain displayed as a sequence of growth events we presume to find master genes channeling differential gene expression into specific patterns of cell division, enlargement and specialization.

A working hypothesis: We suggest that the maize genome contains functionally significant units of clustered genes for plant growth and development. Such a unit must comprise:

- sensors for environmental signals, e.g., daylength, light quality, gravity, temperature, etc., capable of transforming these signals into primary (hormones) or secondary (Ca - calmodulin, transmembrane potential) messages to genes;

- receptor sites or independent transmitters to translate the message into the growth machinery within a particular cell (cell-autonomous trait) or in a wider context (non-cell-autonomous trait);

-service pathways to control these chains of events by producing low- and high-molecular-weight products that are signal transducers or modulators;

-master genes presumably operating in cascade fashion to govern cell and tissue differentiation at the critical points of development; apparently some of these master genes could also play the role of receptor sites for environmental messages.

Notably, the most prominent GRG clusters seem to contain all the listed components. Mutations at the genes comprising clusters produce several classes of disturbances in plant growth and development associated with specific hormones: ABA-related vivipary of embryos, dwarfism usually related to deficiency in gibberellin metabolism, transport or sensing, auxin-related alteration of apical dominance leading to changes in the branching pattern, and various malformations, including developmental displacements, as ectopic effects of hormone interplay. Some of the clusters include genes that could be environmental sensors (*Phy1*, *Phy2*), or hormone sensors (*Abp1*, *D8/D9*, *Rab*, *Vp*). Finally, in most clusters we find genes regulating cell fates in development; these genes usually contain sequences related to DNA transcription factors (*Kn* being the best example).

The advantages of functional gene clustering are intuitively attractive. Compartmentalization within a nucleus of signal molecules, transcription factors and co-factors of transcription can facilitate temporal regulation of gene expression and amplify regulatory cascades. Multienzyme complexes are the most extensively studied example of such compartmentalization of functional coordination and control signals.

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Cytochrome P450 enzymes of the maize seedling

--Monika Frey, Ralf Kliem, Heinz Saedler and Alfons Gierl

Cytochrome P450 enzymes (P450s) are heme-containing enzymes that are most commonly integrated in microsomal membranes. Reactions catalyzed by P450s are characterized by the requirement of NADPH as a co-substrate and the photo-reversible inhibition by CO. NADPH is the substrate of the reductase that represents an integral part of the multi-enzyme complex. The ratio of reductase and P450 may be different at developmental stages and the reductase moiety does not influence the specificity of the reaction, rather substrate and reaction specificity is conferred by the P450 part of the enzyme complex. Furthermore, the reductase is exchangeable even between distantly related species. In plants the importance of P450s as key enzymes in the synthesis of secondary metabolites (e.g. gibberellins, terpens, flavonoids) has been recognized for a long time, but only recently have P450 genes been isolated molecularly (e.g. Bozak et al., Proc. Natl. Acad. Sci. USA 87:3904-3908, 1990).

It has been speculated that plants have evolved highly specific P450-linked secondary pathways to produce defense-related compounds, while in turn animals invented P450-linked systems to detoxify ingested phytoalexins or xenobiotics. The seedling has been one major source for the demonstration of P450 enzyme activity in plants. It has been shown for various species including maize that P450s are transiently expressed in the seedling. Since the seedling is a fragile structure that has to be especially protected for the successful establishment of the plant, part of these seedling specific P450 enzymes might be defense related. An example for such a seedling specific P450 enzyme having impact on defense is given in maize. A P450 N-monooxygenase participates in the synthesis of DIMBOA, a secondary metabolite belonging to the graminean specific class of benzoxazine-ones conferring general resistance to the plant (Niemeyer, Phytochemistry 27:3349-3358, 1988, for review).

We have isolated four cDNA clones representing P450 genes highly expressed in the seedling. Comparison of the amino acid sequence of the four maize P450 clones with the available plant enzyme sequences demonstrates that all plant enzymes are quite related. According to the criteria established for the animal enzymes, the maize genes belong to the same family as the ripening related avocado enzyme Cyp71A. Within this family three of the maize genes build a gene subfamily. The differences within the four maize enzymes are big enough to account for different enzymatic functions. Even for the P450s of animals that have been investigated molecularly for a long time, not much is known about involvement of protein domains in substrate recognition and reaction specificity. Comparison of the amino acid sequences of the four maize members of the Cyp71 family revealed several regions of intrafamiliar conservation that will be tested for their significance in the catalyzed reaction by site directed mutagenesis.

Making use of the recombinant inbred maize population (Burr and Burr, TIG 7:55-60, 1991) the P450 genes were mapped within a four map unit cluster on the short arm of chromosome 4 (4S023 to 4S027). Therefore, as in animals, P450 genes of families and subfamilies are clustered in maize and might have evolved via gene duplication. In contrast to the situation in animals where several large introns disrupt the gene, the structure of the isolated maize P450 genes is simple. One small intron is present close to the dioxygen binding site in all four genes and an additional intron is found in two of them. The number of introns is therefore not conserved within the gene family while intron conservation is a common feature in animal P450 gene families.

Northern analysis revealed that all four genes have a similar expression pattern: they are most highly expressed in the shoot where the maximum is reached seven days after imbibition, while in the root a distinct maximum is displayed at day three. Fourteen days after imbibition only a low level of the transcripts is discovered in the seedling and minor amounts of the transcript are found

in the leaves of the mature plant. No transcript at all is detectable in the kernel. Between different maize lines the relative amount of the four P450 genes might vary. A hint for the function of the isolated maize P450 enzymes might come from their distribution in the maize seedling. In the shoot there is a shift of the major hybridization signal from the coleoptile to the outer leaves. However, the youngest, smallest leaves and the apical meristem display only background hybridization throughout the span of P450 gene expression. Transcript is detected at the base of all developed leaves and at the tip of the outer leaves but hybridization throughout the leaf blade is demonstrated only for the coleoptile and the two outer leaves. These organs build a kind of shield for the seedling but have no function for the major plant and are even degraded. The parenchymatic cells of the first internode and the compressed nodule complex that is the site for the generation of secondary roots of maize were highly decorated with silver grains after in situ hybridization. In the primary root the hybridization is restricted to the region of cell division and here to the cortex and the pith of the pro-vascular tissue. Due to this expression pattern it seems unlikely that the maize P450 genes of the Cyp71 family are involved in hormone synthesis or in the synthesis of cell wall related compounds, but it might be that they have an implication in defense mechanisms. The enzyme function will be tested by heterologous expression and the function of the genes will also be assayed by 'reverse genetics'.

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Genetic characterization of *R-mb:cc*, a mutable derivative from *R-mb*

--V. Niral, B. M. Prasanna and K. R. Sarkar

R-mb:cc, a new variant from *R-mb*, was reported by Prasanna and Sarkar (MNL 67:87-88, 1993). The phenotype characterized by *R-mb:cc* on the aleurone has some exceptional features. Colored sectors on colorless background appear in the form of concentric rings or stripes on either side of the scutellum. The sectors may extend onto the crown and the abgerminal side (Fig. 1). However, the flow region on the abgerminal side might show irregular spots as in *R-mb*. We tried to further characterize this derivative from *R-mb* through genetic analysis.

Wide variation could be observed in the degree of pigmentation of *R-mb:cc* kernels, evidenced by varying number of stripes on the kernels from the same ear. To test if this variation has any genetic basis, kernels were categorised into six different scores based on a 'striping scale' (cc1 with only one colored stripe to cc6 with almost full coloration on the aleurone except for one or two colorless sectors). Although each of these scores segregate for different scores on selfing, distinct segregation profiles could be observed. By analysing the mean visual scores of ears belonging to the different classes through Student's t-test, we could categorise the striping pattern of *R-mb:cc* into three classes: very light striping (cc1), light striping (cc2) and medium striping (cc3, cc4 and cc5). The very heavily pigmented class (cc6) could not be statistically analysed due to small sample size.

Unlike the 'sister' pattern alleles *R-nj* and *R-st*, the *R-mb* allele shows a drastic reduction in both penetrance and expressivity when transmitted through the pollen parent in a single dose (Weyers, Genetics 47:1061-1067, 1961; Prasanna and Sarkar, MNL 67:85-86, 1993). To determine if *R-mb:cc* displays a genetic behaviour similar to that of the parental allele *R-mb*, we carried out reciprocal crosses between homozygous *R-mb:cc* lines and the recessive tester lines carrying *r-g*, besides selfing *R-mb:cc* lines. Analysis of the mean visual scores in *R-mb:cc R-mb:cc R-mb:cc, R-mb:cc R-mb:cc r-g* and *R-mb:cc r-g r-g* by t-test showed statistically significant differences in striping potential of the three classes (Fig. 2). In a single dose the penetrance of *R-mb:cc* was drastically reduced, with only 5-6% of the kernels showing the mb:cc phenotype as compared to 61-62% in two doses and 88-89% in three doses. We could also observe that such a significant effect of dosage on penetrance and expressivity is restricted only to the *R-mb* and *R-mb:cc* among the pattern alleles at the *R* locus.

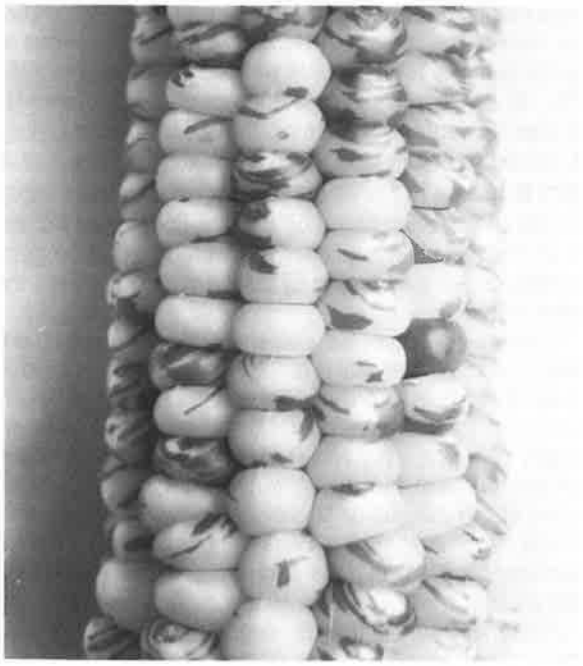


Figure 1. Ear showing the characteristic striping pattern of *R-mb:cc*. The two phenotypic extremes, a self-colored revertant (*R-scm*) and a colorless kernel, can also be seen.

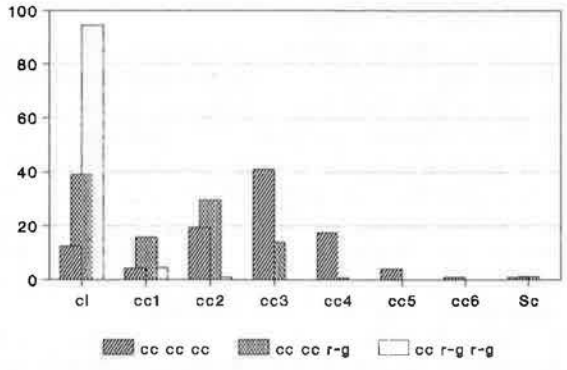


Figure 2. Segregation profiles of various doses of *R-mb:cc*.

Reversions to the self-colored form in a high frequency signify the influence of a transposable element on a specific pattern. The *R-mb:cc* ears showed frequent reversions to the fully colored form (Fig. 1). Progeny testing showed that the self-colored kernels were either somatic or germinal revertants. The germinal re-

version rate from *R-mb:cc* to *R-scm* was 17.97 x 10^{-4}. However, in the case of test crosses of *R-mb:cc/g r-g* with recessive tester *g r-g* the germinal reversion rate was found to be higher (26.98 x 10^{-4}). In addition, discordant endosperm-embryo phenotypes were also recovered from *R-mb:cc*. Kernels with mb:cc endosperm and colored scutellum from test crosses were observed at a frequency of 18.40 x 10^{-4} while the frequency of colorless endosperm with colored scutellum was 3.07 x 10^{-4}. Both classes of discordant endosperm-embryo phenotypes were found to be germinal revertants on progeny testing by selfing or crossing onto the recessive tester.

To ascertain whether the element system at *R-mb:cc* is influenced by other pattern alleles at the *R* locus, we made reciprocal crosses of *R-mb:cc* with *R-nj, R-st* and the parental allele *R-mb*. It was found that there are no dominance-recessive relationships between *R-mb:cc, R-nj* and *R-st*, evident by occurrence of a codominant pattern. However, when *R-mb:cc* as a female was crossed with *R-mb*, mb:cc phenotype was predominant and the converse was true when *R-mb* was used as a female parent. The observations indicate that the *R-mb:cc* pattern is not influenced by the parental allele *R-mb* in a *R-mb:cc/R-mb* heterozygote, and the differences in phenotypic segregation in the reciprocal crosses can be attributed to the single dose effect.

A plausible mechanism for the origin of *R-mb:cc* from *R-mb* can be a 'change in state' of the element in the parental allele where the element at *R-mb:cc* might be responding to different developmental signals or host factors. Characterization at the molecular level can help us dissect out the differences between *R-mb* and *R-mb:cc*. This can also provide significant clues in understanding the genetic and epigenetic phenomena underlying the formation of this symmetric and germinally transmissible pattern.

Tassel maturation and *R-mb:cc* expression

--V. Niral, B. M. Prasanna and K. R. Sarkar

We were interested in finding out if tassel maturity has any effect on excision behaviour of the transposable element system operating at *R-mb:cc*. In the maize tassel, the florets shed pollen over a period of a week. Anthesis proceeds in a systematic and predictable manner, with the two florets in a spikelet shedding pollen on two subsequent days.

In *R-mb:cc* lines, pollinations were made on different days of pollen shed (day 1 to day 7). The resultant ears were scored on the basis of 'striping scale' and the average ear scores computed. Comparisons of the average scores of ears pollinated on different days of tassel maturation showed no statistically significant changes in phenotypic expression. In an earlier study, Levy and coworkers (Dev. Genet. 10:520-531, 1989) studied the association between tassel maturation and somatic excision frequency of *Mu* and found that there were no overall changes in *Mu* activity in the tassel during the period of pollen shedding.

It appears, therefore, that the excision behaviour of a transposable element may not show significant differences in different sectors of the tassel.

Anthocyanin pattern formation in vitro

--V. Niral, B. M. Prasanna and K. R. Sarkar

The majority of studies on anthocyanin pattern formation in maize have been done in vivo by screening kernels collected from the field at various days after pollination (DAP). Such studies have contributed immensely towards the understanding of phenomena such as clonal development of the aleurone using somatic reversions of *R-st* (Coe, in Maize Breeding and Genetics, Walden, ed., pp. 447-459, 1978) and elucidation of the intra-tissue differentiation in maize aleurone through studies on *R-nj* pigmentation (Styles et al., Can. J. Genet. 19:111-117, 1977; Prasanna and Sarkar, MNL 67:86-87, 1993). However, to closely follow the sequence of events in a single kernel/group of kernels simultaneously, the in vivo approach has inherent limitations. To circumvent these, we have adopted an in vitro approach.

Immature kernels (4-6 DAP) were transferred to test tubes containing 20 ml of culture medium (medium composition as in Gengenbach, Crop Sci. 17:489-492, 1977) under aseptic conditions as blocks of 4-8 kernels each. The tubes were incubated at 25±1 C in the dark. Kernels from homozygous *R-mb:cc* ears showed formation of the characteristic mb:cc pattern. Pigment initiation in vitro (14-16 DAP) was comparable to that in vivo except for a slight delay of 1-2 days. In aleurone patterns like that of *R-mb:cc*, which are symmetric and location-specific, it is important to ascertain whether the final pattern is due to progression of stripes arising from different regions of the kernel that coalesce thereafter or the pattern is pre-programmed without dependence on progression and coalescence. On the basis of the observations on pattern formation in several in vitro grown kernels, we conclude that in *R-mb:cc*, pigmentation pattern follows the latter. Intensity of pigmentation was less on initiation, with a gradual increase over time without any alteration in the basic pattern. However, colored spots appearing at the base of the kernel were relatively late in onset compared to those on the crown. Besides kernels showing mb:cc pattern, colorless and self-colored kernels were also recovered.

In order to ascertain differences in pattern formation of *R-mb:cc* and *R-nj*, immature ears from *R-mb:cc/R-nj* selfed lines were also cultured. As observed in vivo, the Navajo pattern showed a clear delay in its onset (a minimum of 3 days) in comparison to that of *R-mb:cc*. The kernels showed either nj, mb:cc or both nj+mb:cc expressions as would be expected on selfing of a *R-nj/R-mb:cc* heterozygote. Through these in vitro cultures, we could also obtain mature, fully grown/differentiated kernels. This approach, coupled with image analysis, can serve as a powerful tool to analyze the events underlying the formation of anthocyanin patterns like that of *R-mb:cc*.

Stabilization of high haploid inducer lines

--K. R. Sarkar, A. Pandey, P. Gayen, Jasbir Kaur Madan, Rajesh Kumar and J. K. S. Sachan

The haploid inducer lines developed earlier with 75 percent stock-6 background yielded on an average, about 3 percent maternal haploids in the stock-6 derived tester line carrying appropriate genetic markers. The haploid induction potential was still segregating in the plants within the lines, but further selection was not effective. Therefore, intercrossing among the lines and selection was tried to increase the haploid induction potential still further.

With this in view, the top 15 to 20 high haploid inducer males in each season were carried forward to the next generation and tested. Haploid induction higher than 5 percent was obtained during the last five seasons (Table 1). For the last three seasons haploid percentage induced by selected male families ranged from

Table 1. Mean haploid percentage over the season.

Season	Mean haploid %	Range of haploid %
1990-91 winter	5.22	2.19-12.50
1991 summer	4.86	2.50-9.23
1991-92 winter	5.81	3.05-14.29
1992 summer	5.22	4.03-13.35
1992-93 winter	5.47	3.76-18.57

3.05% to 18.57%. Further, rigorous selection for the last five seasons resulted in identification and stabilization of a few high haploid inducer *C-I* lines (Table 2). In the 1992-93 winter, one ear

Table 2. Selected males from 1992-93 winter.

		Haploids	
Male	Total kernels	Number	Percent
5329 C.1-17	77	10	12.98
-23	140	26	18.57
-28	457	44	9.63
-29	484	25	5.17
5329 C.2-8	95	6	6.31
-12	679	26	3.83
5329 D-6	1006	50	4.97
-10	31	3	9.68
-1	27	3	11.11
-11	161	8	4.96
-12	203	16	7.88
-23	519	28	5.40
5332 C.1-2	581	37	5.43
-11	301	20	6.65
5332 C.2-2	1081	48	4.44
-11	432	22	4.84
-16	339	18	5.31
-18	3015	148	4.91
-27	1099	43	3.76
-28	366	26	7.10
-30	58	6	10.34
5332 D.1-12	1793	90	5.02
5332 D.3-24	589	44	7.47
5332 D.5-4	322	16	4.97
-18	566	27	4.77
-27	882	59	6.69
5332 E.1-9	217	16	7.37

produced as many as 26 haploids out of 140 kernels of the tester female. The earlier maximum number was 23 out of 300 kernels on one ear. During the last winter, 15 ears with more than 20 haploids/ear were obtained as opposed to 5 ears with more than 20 haploids in the 1992 summer. These haploid inducer lines are being tested on breeding populations to explore the possibility of mass scale haploid induction for homozygous line development.

Chromosome doubling in haploids through colchicine

--P. Gayen, Jasbir Kaur Madan, Rajesh Kumar and K. R. Sarkar

Maternal haploids isolated through the genetic selection technique were used for chromosome doubling. Seeds (soaked for 48 hours) and seedlings (5 days after germination) were treated with 0.03%, 0.06% and 0.1% aqueous solution of colchicine (SRL) with DMSO (dimethyl sulfoxide) 0.5% (by volume) for 6 hours, 12 hours and 24 hours.

Seed treatment. A small portion of the plumule tip was cut off before treatment to ensure better penetrance of colchicine to the growing meristem. A set of controls without cutting the plumule tip was also included. Treatment with 0.06% colchicine concentration was carried out at two different temperatures, 18±1 C and 25±2 C. Fifty seeds per treatment were dipped in aqueous solutions of colchicine and kept in the dark. After completion of the treatment, the seeds were thoroughly washed with distilled water and sown in the field. Light watering in the evening up to 7 days after sowing is very essential for better germination and establishment of seedlings.

Seedling treatment. Shoot tips were nipped off to expose the apical meristem for better colchicine action. A set of controls without cutting the shoot tip was also included. Fifty seedlings per treatment were maintained by moistening cotton wads at 1 hour intervals. After completion of the treatment the shoot tips were thoroughly washed with distilled water. The seedlings were maintained in paper cups for 2-3 days in shade and then transferred to the field. Significant success (18.05%) was obtained from seed treatment (with plumule tip cut) with 0.06% colchicine concentration at 18±1 C for 12 hours (Table 1). Cutting of the

Table 1. Effect of varying colchicine concentrations on doubling of maize haploids obtained through 'Scutellum Marker Technique.'

			% doubled haploids Duration of treatment		
Colchicine Conc.	Material		6 hrs	12 hrs	24 hrs
0.03%	Seedling	Tip cut	1.02	1.20	1.80
		Tip intact	0.00	0.29	0.50
	Seed	Tip cut	0.60	0.42	0.98
		Tip intact	0.00	0.00	0.21
0.06%	Seedling	Tip cut	6.23	8.24	No plant estab.
		Tip intact	3.29	4.26	-do-
	Seed	Tip cut 18 C	10.23	18.05	-do-
		Tip cut 25 C	5.98	12.21	-do-
		Tip intact	2.97	6.22	-do-
0.1%	Seedling	Tip cut	6.42	-do-	
		Tip intact	3.41	-do-	
	Seed	Tip cut	4.32	5.38	-do-
		Tip intact	2.21	3.61	-do-

plumule tip in the case of seed, and shoot tip in the case of seedlings, gave better response. No plant establishment was obtained from treatments with 0.06% and 0.1% concentrations for 24 hours. Chromosome doubling of treated haploids was confirmed by chromosome counting at diakinesis and anaphase I from the fixed male inflorescence.

Morphometric characters of seed in relation to callusing ability (%) and callus growth

--Jasbir Kaur Madan, P. Gayen and K. R. Sarkar

A study was carried out to ascertain whether there is any relationship between morphometric characters of seed and callusing ability (%) and callus growth. Mature seeds of thirty different inbreds were used in this experiment. Morphometric characters (except seed weight and seed volume) were recorded from 20 random seeds of each inbred. Sixty embryos from each inbred were cultured in MS media supplemented with 2,4-D (2 mg/l), casein hydrolysate (1 g/l), inositol (100 mg/l), sucrose (3%) and agar (0.7%). Records on callusing ability and callus growth were taken 14 days after inoculation. It was observed that morphometric characters of seed have no significant association with callusing ability (Table 1). However, seed weight, seed volume and seed width showed positive and significant correlation with callus growth.

Table 1. Association of seed morphology with callusing ability (%) and callus growth (r values).

Seed characters	Callusing ability	Callus growth[a]
Seed length (cm)	0.06	0.40
Seed breadth (cm)	-0.03	0.56*
Single seed wt. (g)	-0.24	0.63**
Single seed vol. (cc)	-0.38	0.59**
Embryo length (mm)	-0.13	0.37
Embryo width (mm)	-0.24	0.41

*, **Significant at P=0.05 and P=0.01, respectively

[a]Callus growth (fresh weight basis) taken after 14 days of inoculation.

Effect of silver nitrate on callusing ability

--Jasbir Kaur Madan, P. Gayen and K. R. Sarkar

Mature and immature (16-18 days after pollination) embryos of seven inbreds were tested for callusing ability with and without silver nitrate (10 mg/l). MS medium was supplemented with 2,4-D (2 mg/l), casein hydrolysate (1 g/l), inositol (100 mg/l), sucrose (3%) and agar (0.7%). Fifty mature and fifty immature embryos from each inbred were cultured in the media with silver nitrate, and the same number of embryos were cultured without silver nitrate. Callus induction frequency (%) was taken 20 days after inoculation. For all the inbreds, better callus induction was observed with silver nitrate either in mature or immature embryos (Table 1). It was also noted that immature embryo was better than mature embryo to induce callus for all the inbreds. The variation in callus induction frequency was significant among the inbreds, between two different embryo ages and also between the silver nitrate treatments.

Table 1. Impact of silver nitrate and embryo age on callusing ability of different inbreds.

	Mature embryo		Immature embryo	
Inbred	With $AgNO_3$	without $AgNO_3$	With $AgNO_3$	Without $AgNO_3$
2100	47.02	9.23	62.19	20.40
2702	38.92	10.20	58.62	15.23
2122	65.02	25.90	81.23	39.42
3498	72.91	30.00	80.21	35.40
3508	80.29	40.21	85.20	44.21
3994	35.80	6.29	50.29	16.20
4603	50..02	20.41	90.49	40.21

Meiotic studies on haploids

--P. Gayen, J. K. S. Sachan, Jasbir Kaur Madan and K. R. Sarkar

Three haploid plants (stock-6 derived) were analyzed meiotically. Grouping of chromosomes in diakinesis and metaphase I was very pronounced. About 60% of the cells had 5:5 grouping, 15% of the cells had 6:4 grouping (which may be potentially 5:5), five groups of two bivalents were noticed in 15% and in the rest of the cells, no grouping was observed. This raises some doubts about the exact basic chromosome number in maize, indicating that it might be 5 rather than 10.

Besides grouping of chromosomes, it was also noted that three chromosomes were attached to the nucleolus in more than 75% of the cells in diplotene and diakinesis (Fig. 1). The presence of 1-4 micronucleoli was found in more than 50% of the cells. Two diploid plants of the same stock-6 background grown in moisture stress and high temperature had a high frequency of cells with 3 bivalents attached to the nucleolus, and the presence of micronucleoli was very common. But the diploid plants of the same genotype grown under normal conditions had no cells with more than one bivalent attached to the nucleolus. Adverse microenvironment of the cells in the plants grown in moisture and temperature stress or genomic imbalance in the haploids may be responsible for the occurrence of micronucleoli. These observations also indicate that there might be two other chromosomes, other than chromosome 6, in the maize genome which have nucleolar organiser regions (NOR) expressed only under adverse conditions.

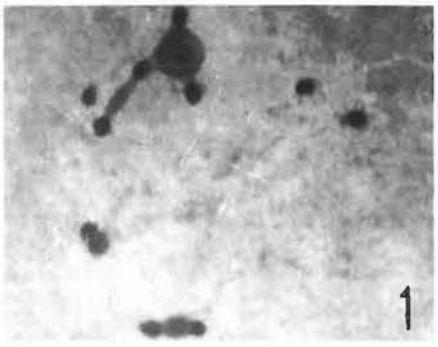


Figure 1. Diakinesis of a stock-6 derived haploid plant showing three chromosomes attached to the nucleolus and presence of three micronucleoli.

Somatic pairing in maize and teosinte

--J. K. S. Sachan, K. R. Sarkar and Ryuso Tanaka

Somatic pairing refers to close or loose association of homologous chromosomes during mitotic divisions. Somatic pairing and somatic crossing over are genetically controlled events and have been reported in a wide array of animals and plants. These events assume special evolutionary significance in sexual fungi like *Aspergillus*, in *Drosophila* and in maize.

Indirect evidence of premeiotic pairing in maize has been reported by Rhoades (The Cell 2, 1961) and Maguire (Genetics 53:1071-1077, 1966). We report here somatic pairing and somatic crossing over at metaphase in root tip meristems of a Colombian popcorn race Pira Naranja and Xochimilco teosinte (K68-3), seeds of which were received from CIMMYT. The conventional procedures described by Sachan and Tanaka (Chromosome Inf. Serv. 20:3-4, 1976) for Feulgen staining and by Sachan and Tanaka (Jpn. J. Genet. 51:139-141, 1976) for C-banding in the root meristem of *Zea* chromosomes were used.

The illustrations (Figs. 1 and 2) are self-explanatory of the perfect pairing of homologous chromosomes in maize at mitotic metaphase arrested in root tips by pretreatment with 0.002 M 8-hydroxyquinoline at 14±18 C for 3.5 hours. The event of somatic pairing occurs in the root tips of *Zea* roughly with a frequency of one in 500 metaphase plates. The photographs can not be confused with C-mitosis as there are strong evidences in support of somatic pairing and somatic crossing over. Firstly, both conventional and C-banded karyotype preparations of maize and teosinte had a chromosome count of 2n=20. Secondly, in haploid C-mitosis, one expects complete correspondence in morphometry of both the chromatids. We know that chromosome 6 of *Zea* is highly hetero-

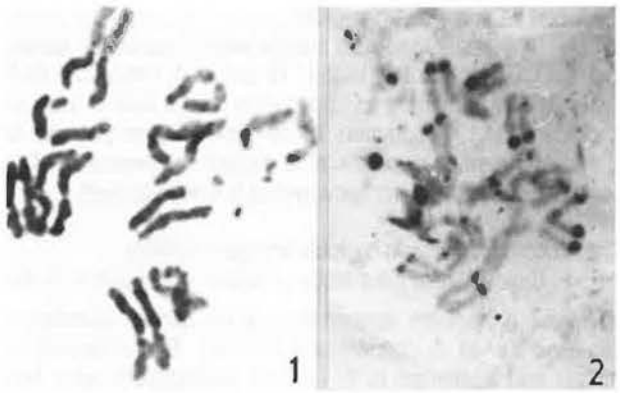


Figure 1. Somatic pairing and crossing over in teosinte (K68-3).
Figure 2. C-banded somatic karyotype of Pira showing pairing and crossing over.

morphic. Figure 2 gives clear identity of heteromorphic pairing of chromosome 6. One can also note chiasmata formation and probable exchange of genetic material in some of the paired homologues both in maize and teosinte.

Amphidiploid theory of maize origin - revisited

--J. K. S. Sachan, M. S. Ramesha, P. Gayen and Vinita Lakkawar

In view of the possible tetraploid nature of maize and consideration of its amphiploid derivative of n=5 of sorghum and n=5 of *Coix* by Anderson (Chronica Bot. 9:88-92, 1945) and Stonor and Anderson (Ann. Mo. Bot. Gard. 36:355-404), revisiting seems necessary with appropriate modification.

Tangible evidence in favour of x=5 as the basic chromosome number of the genus *Zea* has been provided from the secondary association in diploid maize (Chantekar, Cytologia 30:426-435, 1965), haploid maize (Majumdar and Sarkar, Cytologia 39:83-89, 1974; Ting, Maydica 30:161-169, 1985) and maize x teosinte hybrids by Ting (Bussey Inst. Harvard Univ. (monograph), p. 64, 1964), and in a series of papers by Molina and her associates (Cytologia 50:643-648, 1985; MNL 60:77-79, 1986, etc.). Electron micrographs of somatic metaphase chromosomes (Bennett, Kew Bot. Conf. II, pp. 71-79, 1983) also suggest the tetraploid nature of the maize genome. Buffering capacity and survival of monosomics (Weber, in W. F. Sheridan, ed., pp. 79-83, 1982) add further evidence for the amphiploid origin of maize.

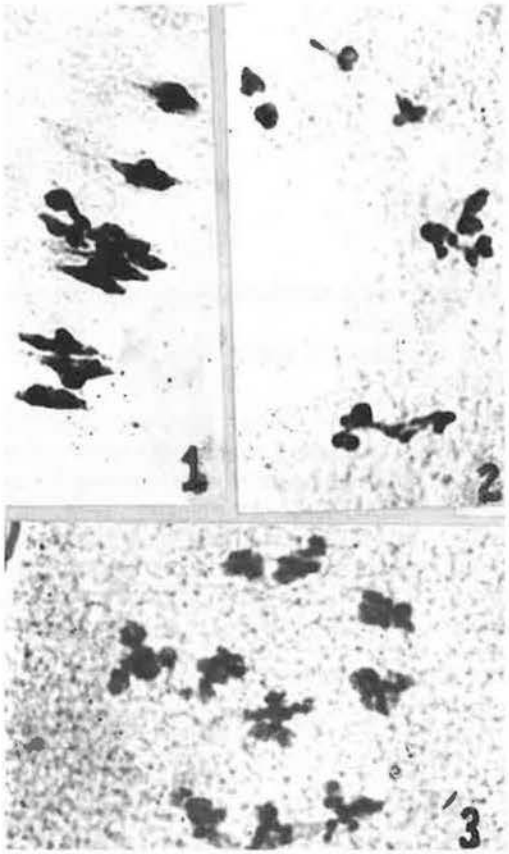


Figure 1. Secondary association (3:3:2:2) at metaphase I in Nal-Tel x *Zea diploperennis*.
Figure 2. Equatorial view of 3:3:2:2 constellation at metaphase II in *Zea diploperennis* x CM105.
Figure 3. Polar view showing 3:3:2:2 constellation at metaphase II in CM105 x *Zea diploperennis*.

Our studies on secondary association in maize, teosinte and their hybrids have supported the amphiploid (2n=4x=20) nature of maize evolution. In maize (2n=20) and teosinte (2n=20) chromosome association has been observed in almost all the maize and teosinte hybrids. For example, maize x *Zea diploperennis*, maize x *Z. luxurians*, maize x *Z. parviglumis* and maize x *Z. mexicana* secondary association has been observed at different stages of meiosis in varying frequencies such as 5:5, 6:4, 3:3:2:2 and 2:2:2:2. The most frequent association at metaphase I was 3:3:2:2 (Fig. 2). It is interesting to note that the pattern of chromosome association is also retained in metaphase II (Figs. 1 and 3). Chromosome constellations observed in metaphase I are also faithfully reflected in metaphase II, which provides an opportunity to observe chromosome pairing and association etc. with more clarity than in metaphase I. Finally, it appears quite probable that the *Zea* species possess two homologous genomes, where pairing is restricted due to the presence of a *Ph*-like gene (Poggio et al., MNL 64:72, 1990).

Centromeric fusion and knob fusion in maize

--S. Dash, P. Gayen, Vinita Lakkawar and J. K. S. Sachan

Centromeres and knobs are the topographical references of pachytene chromosomes in maize and teosinte. Structurally and functionally both identities are distinct from the rest of the chromosome. It appears that DNA sequences among the centromeres of different bivalents, and the repetitive DNA within knob-heterochromatin in the genome, have considerable homology to pair and exchange the genetic material from nonhomologous counterparts.

The phenomenon of random centromeric fusion has been reported earlier by Gurgel (MNL 30:54-57, 1956). While studying knob constellations of the Northeastern Himalayan maize we came across centromeric fusion of nonhomologous bivalents at pachytene in Sikkim Primitives from Tripura (T-2 and T-26) and in other strains from Meghalaya (M-1, M-5 and M-12). In our case, it was observed that a particular chromosome with a subterminal knob on the short arm is frequently involved in centromeric fusion events (Fig. 1). Similarly, random homoeologous association and fusion of knobs between nonhomologous bivalents at pachytene in maize (M-5) and maize (CM-111) x *Z. parviglumis* crosses (Fig. 2) have been frequently observed. The consequences of centromeric fusion and knob fusion may lead to structural differences and thus have an evolutionary significance in maize.

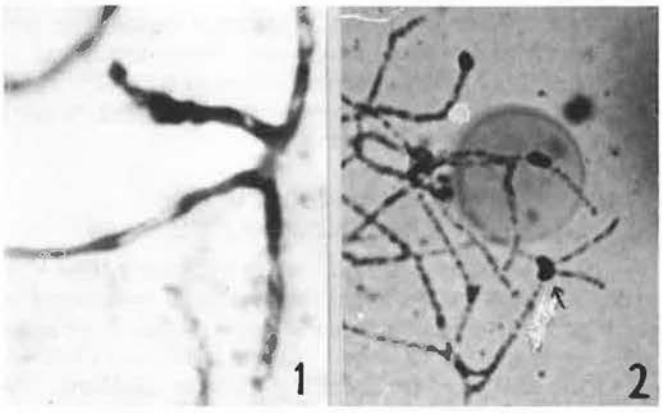


Figure 1. Centromeric fusion in maize (M-5).
Figure 2. Knob fusion (→) in maize (CM-111) x teosinte (*Zea parviglumis*) hybrid.

Translocation heterozygosity in *Coix*

--P. Gayen, J. K. S. Sachan, Rajesh Kumar and K. R. Sarkar

Adaptive polymorphism for translocations exists sporadically in the genus *Coix*, an Asiatic relative of maize. There are a number of forms and species of *Coix* with chromosome counts of 10, 20 and 40. In naturally occurring populations of *Coix* from Western Ghat (Coix-21) and from Madhya Pradesh (Coix-63), rings of various sizes have been observed. In Coix-21 a ring quadrivalent was obtained in 60 percent of cells at diakinesis (Fig. 1) and one hexavalent was noted in about 12.5 percent of cells. The rest of the cells had 5 bivalents. The presence of quadrivalents (ring) or hexavalents (ring) is due to spontaneously occurring reciprocal translocations. In anaphase I, separation was observed, but a few cells with 1-2 lagging chromosomes, which might be a case of late separation, was noted. More than 80 percent of the cells had 5:5 separation in anaphase I and pollen fertility was 98 percent accompanied by high (about 80%) seed fertility. On the basis of these observations it might be assumed that this translocation heterozygosity is analogous to that which exists in *Rhoeo* and *Tradescantia*. In another population, Coix-63, 75 percent of cells had one quadrivalent, 7.5 percent of cells had one hexavalent (Fig. 2) and the rest of the cells had 5 bivalents at diakinesis. In

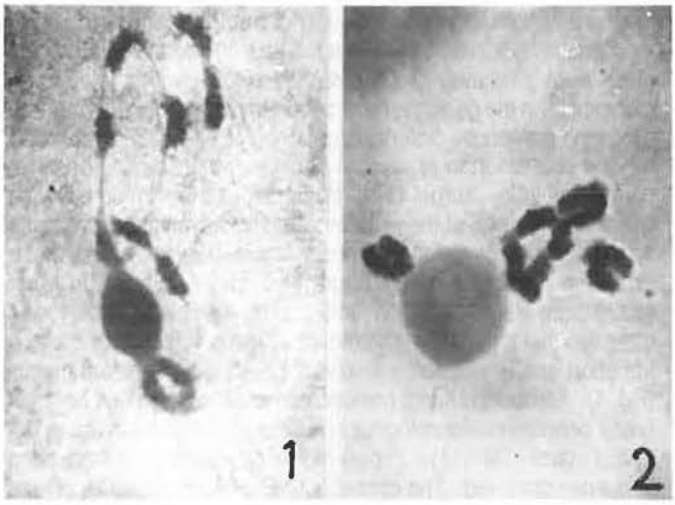


Figure 1. Ring quadrivalent at diakinesis in Coix-21.
Figure 2. Hexavalent at diakinesis in Coix-63.

anaphase I the majority of the cells (80%) had 5/5 separation, with unequal separation (6/4) in 20 percent of the cells. No cells with lagging chromosomes were noted. Pollen fertility was 75 percent and seed fertility was not as normal as in Coix-21. In this collection probably alternate segregation is higher, but the same amount of adjacent type is also there.

Comparative pollen grain size in the tribe Maydeae

--T. M. Shivakumar, Rajesh Kumar and J. K. S. Sachan

The pollen grains of different species and genera of the Maydeae and sorghum (Andropogoneae) studied were round or slightly oval with a single pore (uniporate). In Maydeae, the pollen grain diameter along the longest axis was found to vary between 31.25 µm and 124.70 µm under Delhi summer conditions. The pollen of *Chionachne* (Chio-1) was the smallest, and the largest pollen belonged to Sikkim primitive maize (S-44). The pollen of maize was generally largest followed by teosinte, *Coix*, sorghum and *Chionachne*. As to the pollen size among teosintes, *Zea diploperennis* had the smallest (77.92 µm) and the Mexican teosinte (Teo-2) had the largest (95.83 µm). In *Coix*, the minimum pollen size noted was 55.83 µm and the maximum was 97.92 µm. *Chionachne* pollen ranged from 31.25 µm and 39.17 µm in size. The pollen size of the two sorghum lines examined, viz. CK 60B and 2077B, were 37.50 and 38.50 µm, respectively. Based on the C.D. value for pollen grain size no difference was observed between *Chionachne* and sorghum. The size ranges of maize, teosinte and *Coix* were overlapping with one another (see Table 1).

Table 1. Comparison of pollen grain size based on C.D. value (7.15).

Material	Pollen size (µm)
Chionachne-1	31.25
CK 60B	37.50
2077 B	38.75
Chionachne-5	39.17
Coix-9	55.83
Coix-29	60.00
Coix-11	69.17
Z. diploperennis	77.92
S-44	81.67
Coix-49	83.33
Teo-1	85.00
Teo-6	85.83
Teo-5	87.08
Arrocillo Amarillo	90.00
Teo-3	90.83
Z. luxurians	92.50
Palo. Toluqueno Mex-5	94.17
Tepecintle, Teo-4	95.42
Teo-2	95.83
T-2	96.67
Coix-48	97.92
S-18	100.00
Pira	101.25
T-26	101.67
S-27	104.17
Conico Norteno	111.25
Chapalote, M-15	113.33
N-4	115.00
M-25	120.83
M-1	121.67
S-23	124.17

Interracial differences in mechanical properties of the cob in relation to knob composition

--J. K. S. Sachan and Y. Nath*

*IIT, Delhi

Mechanical properties of corn cobs were determined from quasi static radial compression force deformation curves. Cobs were cut axially to small pieces of approximately 2.5 cm length. Different races, including Sikkim Primitive maize, with varying knob number were tested for their relative contribution to the macro-structure components of the corn cob: to its apparent modulus of elasticity, crushing strength and modulus of toughness in relation to knob composition. The studies showed the variation

Table 1. Main effects of races on physical and mechanical properties of the cob.

Races	Knob no.	Cob softness/ flexibility	Apparent elasticity modulus	Crushing strength	Modulus of toughness	Dia. (mm)	Pith dia. (mm)
Palomero Toluqueno	2-4	soft	6.20	2.70	0.437	12.99	4.43
Confite Morocho	2-4	flexible	4.84	2.26	0.428	9.91	2.21
Chapalote	10-12	---	7.99	1.89	0.227	20.25	6.00
Pira	6-8	soft/flex	5.90	1.62	0.881	10.00	2.42
Celaya	10-12	hard	8.29	1.91	0.164	23.88	8.84
Bolita	12-14	hard	12.90	2.42	0.265	21.61	5.68
Sikkim	8-10	soft/flex	4.34	1.76	0.699	11.28	3.23

of modulus of elasticity along its length. It is less in races with soft cob and low knob number. It has been found that Young's modulus has the higher value near the middle of the cob towards the butt for all the corn races except Palomero Toluqueno. Table 1 shows the average values of the properties. It was observed that the race Bolita has the highest, and Sikkim Primitive (T-26) the lowest value of modulus of elasticity. Palomero Toluqueno has the highest crushing strength and T-26 has the lowest. Pira has the highest modulus for toughness and Celaya has the lowest value.

Our experience on working with landraces of Northeastern Himalayan maize suggests that maize strains with soft and flexible cobs possess relatively low knob number. Hardness of the cob reflects introgression of teosinte into maize.

Restructuring maize plant type for higher productivity

--J. K. S. Sachan

Ever since the advent of the modern era in maize cultivation, there always existed a stereotypic image of a maize plant type marked by the presence of one or two cobs in the middle of the plant. Our analyses of prehistoric wild corn vis-a-vis Sikkim Primitive maize (SP) and other primitive and advanced races of maize, including inbreds and local varieties of the Northeastern Himalayan region and the plains of India, effectively emphasize the values of landraces in developing maize cultivars. Identity of landraces or the prehistoric corn is progressively lost during 10,000 years of domestication, population growth and deforestation. Breeders are responsible, to some extent, for the extinction of maize diversity. Inbreds and hybrids with one or two ears in the middle of the plant were preferred to better suit mechanized farming and ease of harvesting by combine harvester. This had a devastating effect on potentialities of this taxon, fondly called corn. With a growing realization of the stagnation in the yield potential of maize, one begins to ponder the importance of wild relatives and landraces. A significant question arises--do we really have such a germplasm that can contribute to a quantum jump in maize yield? The answer is positively 'yes'. The SP maize, a germplasm par excellence, offers advantageous opportunities to the breeder for a sizable leap in yield and productivity. It is worthwhile in this context to mention the salient features of SP maize.

Information obtained from our studies on botanical, C- and Q-banding; pachytene analysis, ethnobotany, and interpretation of the archaeological findings have provided tangible evidence that pre-historic wild corn, which evolved in the extreme desert environment in the Tehuacan valley of Mexico, is well preserved in the form of Sikkim Primitive (SP) in remote and isolated pockets of the Northeastern Himalayan Region (NEH) (Sachan and Sarkar, Indian J. Genet. 46:153-161, 1986). SP maize, which has 10,000 years of history, was evolved under extreme desert conditions. With its unique plant architecture, it offers a golden opportunity to breeders to utilize a reservoir of favourable genes, both for biotic and abiotic stresses as well as several other agronomic traits. There is strong evidence that SP maize plant type conforms to the most competitive and advantageous attributes for survival in wilderness. Reproductive efficiency and the defense mechanisms of SP maize are the fittest attributes to survive against the vicissitudes and vagaries of weather, potential enemies, biotic and abiotic stresses. Studies have revealed that SP maize harbours a gene for drought tolerance (Mani et al., MNL 61:2, 1987). The most important physiological attributes of SP maize are a complete lack of apical dominance, prolificacy (5-9 ears) with uniformity in ear size (Fig. 1); erect leaves for developing maize varieties for high population density, top bearing habit and drooping tassel to ensure effective fertilization (Sachan and Sarkar, MNL 56:121-124, 1982). It stays green after maturity; thus it is also good for fodder purposes. It is resistant to stalk rot and has tremendous stem strength which prevents lodging.

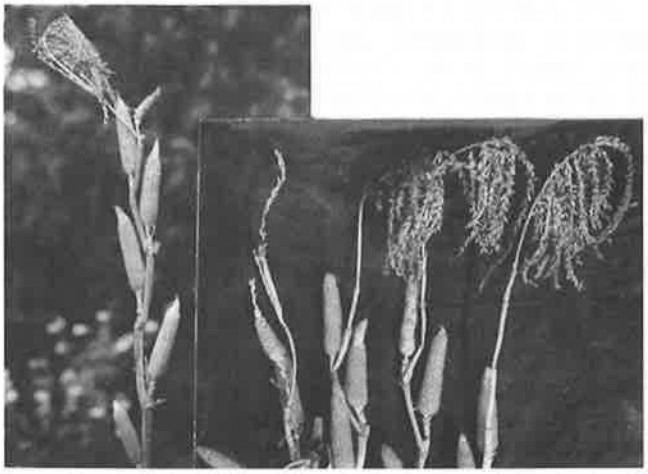


Figure 1. Different collection of Sikkim primitives showing drooping tassel, upper bearing habit, prolificacy and uniformity in ear size (L-R; strains from Kumaon Hills, Tripura, Sikkim, Meghalaya and Nagaland.

Some of the factors influencing photosynthetic efficiency are the availability of water, carbon dioxide, light, nutrients, temperature, plant age, leaf age and the genetic makeup of the plant. The internal control of photosynthesis in maize is the rate at which photosynthetic products are translocated from subtending leaf and leaves in the upper canopy to the sink (cob). For efficient source-sink relationship, the essential contributing factors are longevity and health of the leaf-canopy and minimal distance between the source and the sink. SP maize, having erect leaves near the top of the canopy, should synthesize food more rapidly than those strains having horizontally oriented leaves. Furthermore, the middle leaves and lower leaves are unable to photosynthesize because of chlorophyll breakdown and loss of functional chloroplasts. Thus SP maize possesses all the required parameters to increase the yield potential of maize.

Among all the contributing factors, the placement of the ear is the most important. Ears must occupy positions in the upper one-third of the plant height rather than the middle so that photosynthate is translocated right from subtending and upper leaves to the sink. Our analysis of the fifty strains of maize (as given earlier in the text) has shown that in modern maize, on an average, there are five internodes between the uppermost ear and the tassel, whereas in SP maize there are only two internodes between peduncle and the uppermost ear. Obviously the modern cultivars have inherent limitations in utilizing the photosynthesized product of the upper canopy.

In view of the above facts a hypothesis can be advanced that if the maize plant can be restructured and harnessed to have an upper bearing habit (rather than the present middle bearing habit) with two to three productive ears, a breakthrough in yield may be obtained. Many strains of the Northeastern Himalayan region possess these characteristics. SP maize specially can play a greater role in maximizing the yield potential of modern maize.

NEW HAVEN, CONNECTICUT
Yale University

A microsatellite linked to the *ts2* locus

--Alejandro Calderon-Urrea and Stephen L. Dellaporta

The utility of microsatellite sequences as markers for mapping or for rapid genotyping is becoming a routine practice in plant and animal genetics. This technology requires knowledge of DNA sequences flanking the repeat so that the locus-specific PCR primers can be designed. This information may come from DNA sequences already present in DNA databases or from a systematic search for a particular microsatellite repeat by cloning and sequencing. Here we report on the presence of a microsatellite tightly linked to the recently cloned *tasselseed2* (*ts2*) gene (DeLong et al., Cell 74:757-768, 1993). After sequencing 10 kb of genomic DNA from the *ts2* locus in W22 inbred material, we found the following repeat: 5'TGGC$(AG)_{32}$AACGAA3' located 1.5 kb 3' to the *ts2* gene. Microsatellite length variability was found in different genetic backgrounds using primers flanking the repeat (OTS98 = 5'TGACGGACGTGGATCGCTTCAC3'; OTS99 = 5'AGCAGGCAGCAGGTCAGCAGCG3'). In the W22 genetic background, these primers amplify a 119 bp PCR product, a size consistent with the product predicted from sequence data. As shown in Figure 1, these primers amplify different length

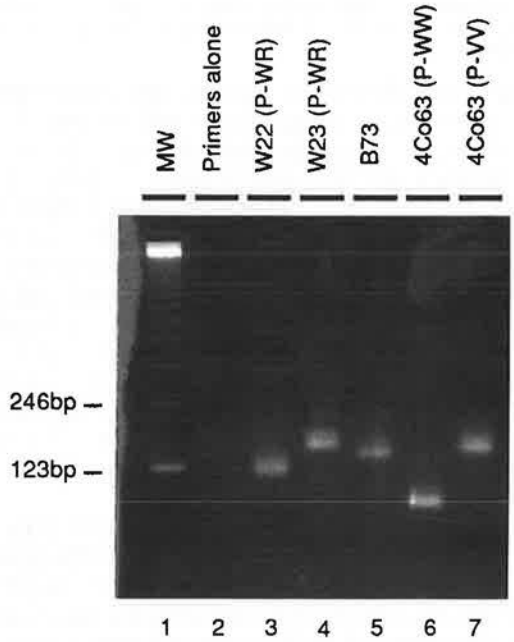


Figure 1. Agarose gel electrophoresis of the *ts2* linked microsatellite in four different maize inbred lines. MW, molecular weight markers (BRL 123 bp ladder).

products in all the genetic backgrounds tested that range from 100 bp (lane 6) to 160 bp (lane 4) (all backgrounds carry a functional *Ts2* allele). It is interesting to notice that two phenotypically identical *P* alleles (*P-wr*), located 2 cM proximal to *ts2*, are polymorphic for the microsatellite repeat. In summary the data indicate that the *ts2*-linked microsatellite repeat should be a useful genetic locus for mapping and genotyping purposes.

Conditions for PCR amplification are as follows: 100 ng of genomic DNA were amplified in a 50 uL final volume containing 20 pmoles of each primer, 10 umoles each dNTP, 5 uL of 10X Taq buffer (USB), 2 uL of $MgCl_2$ solution (USB) and one unit of Taq DNA polymerase (USB). PCR was performed in a Perkin-Elmer-Cetus Thermal Cycler with the following profile: i) 95 C for 5 min., 1 cycle; ii) 95 C for 40 sec., 60 C for 40 sec., 72 C for 40 sec., 30 cycles; iii) 72 C for 15 min. PCR products were fractionated in a 4% (3 NuSieve:1 SeaKem FMC) agarose gel run for 4 hours at 65 volts.

NORMAL, ILLINOIS
Illinois State University

A study of the progeny of monosomic-4 plants in maize

--N. I. Teissonniere, D. F. Weber and M. C. Schneerman

We have explored a limited number of progeny of monosomic-4 maize plants. Monosomic-4 plants were produced utilizing the *r-x1* system as described by Weber (Maize Handbook, M. Freeling and V. Walbot, eds., pp 350-358, 1993). Monosomic-4 plants were selected and crossed as male parents to the inbred B73. The monosomic-4 plants had *su* on their single chromosome 4 and B73 was *Su/Su*; thus, the F1s were *Su/su*.

F1s from the above cross were reciprocally testcrossed, and the number of non-sugary (*Su/su*) and sugary (*su/su*) kernels were determined. Forty-nine F1s were testcrossed as female parents, and none of these had ratios that were significantly different from a 1:1 ratio of non-sugary to sugary kernels. However, 15 of 49 F1s testcrossed as male parents had ratios that deviated significantly from a 1:1 ratio as shown in the table. Thirteen had more sugary than non-sugary kernels and 2 had fewer sugary than non-sugary kernels.

Plant #	Non-sugary#	Sugary	X^2
245-3	149	115	4.38
247-9	217	170	5.71
246-7	88	119	4.64
254-5	0	16	16
246-3	9	428	428
247-6	114	168	10.3
252-11	130	172	5.84
246-6	148	192	5.69
246-5	114	165	9.32
253-6	90	124	5.40
253-3	63	146	33.0
246-3	1	285	282
246-11	177	239	9.24
252-7	108	141	4.37

The reason for these deviations from a 1:1 ratio is not known. In wheat, univalent chromosomes frequently misdivide producing telocentric chromosomes (Sears, Chromosoma 4:535-550, 1952). If the univalent chromosome 4 in the monosomic-4 plant underwent misdivision to produce telocentrics for both arms of chromosome 4, the F1s would have a normal chromosome 4 with *Su* and a telocentric for 4S with *su*. It is possible that the telocentric would demonstrate reduced transmission, and if such an individual were testcrossed, less than half non-sugary kernels would be produced. Two of the plants demonstrated reduced transmission of *su* when they were testcrossed as male parents; however, neither of these demonstrate reduced transmission when testcrossed as female parents. We believe it is unlikely that these plants contain telocentrics for chromosome 4.

NORMAL, ILLINOIS
Illinois State University
COLUMBIA, MISSOURI
University of Missouri

Mapping the centromere of chromosome 4 in maize using a telocentric for 4S

--W. Lee, D. F. Weber, M. C. Schneerman, and G. Doyle

It is critical to correlate the maize genetic and cytological maps so that the rich array of maize cytogenetic variants can be used to manipulate its genome for use in molecular studies. One of the continuing goals of the Weber lab is to better correlate the genetic and cytological maps of maize. We are currently attempting to more accurately map the centromeres of each of the chromosomes on the maize RFLP genetic map. One approach we are using is with telocentric chromosomes.

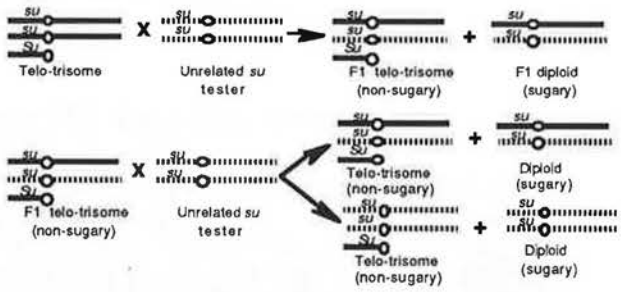
Telocentrics have been recovered for several chromosome arms of the maize genome (Rhoades, Genetics 25:483-521, 1940; Doyle, MNL 62:49-50, 1988, Staub unpublished). Unfortunately, the telocentric for 5S that Rhoades (1940) recovered appears to have been lost (Dempsey, personal communication).

One of us (Doyle) has been analyzing progeny of trisomic maize plants that were heterozygous for a morphological marker locus on the trisomic chromosome. Genetic ratios were observed in certain of these crosses that indicated that a telocentric chromosome plus two normal homologs were present. A plant of this type is a telotrisome. Here we report the use of a telocentric for the short arm of chromosome 4 (telo-4Sc) to more accurately map the position of the centromere of chromosome 4 on the maize RFLP map. One of us (Lee) analyzed telotrisome-4Sc plants cytologically and observed trivalents at diakinesis where one of the three members was smaller than the other two members. We are in the process of analyzing these at pachytene.

A telotrisomic stock for the short arm of chromosome 4 (telo-4Sc) had the recessive allele *su* on the normal chromosome 4's and *Su* on the telocentric chromosome. Part of the gametes produced by such a plant will contain a chromosome 4 (with *su*) and part will contain a chromosome 4 (with *su*) and a telo-4Sc (with *Su*). When a plant of this type is self-pollinated or testcrossed, two types of kernels are produced (not considering recombination between the *Su* locus on the telocentric and the centromere). Some are sugary, and these contain two normal 4s with *su* alleles. Others are non-sugary and contain two normal 4's plus the telo-4Sc. The progeny that contain the telocentric chromosome can be easily identified in this way.

Because this stock had been self-pollinated for several generations, the normal chromosome 4's and the telo-4Sc may be similar genetically. Therefore, the telotrisomic-4Sc stock was crossed as a female parent by an unrelated diploid tester stock that had the same recessive marker mutation (*su*), and the F1 non-sugary progeny were backcrossed as female parents to the same *su* tester stock. These crosses are diagrammed below. The chromosomes of the original stock are shown with solid lines and the chromosomes of the unrelated *su* tester are shown with broken lines.

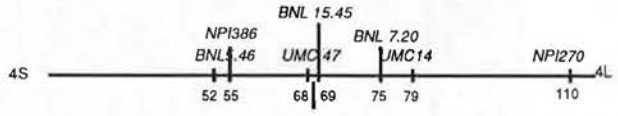
When the F1 non-sugary progeny are backcrossed to the unrelated *su* tester, four types of progeny are produced. Half of the non-sugary kernels have both chromosome 4's from the tester and the other half have one from the original telotrisomic-4Sc stock and one from the *su* tester. Also, half of the sugary kernels have both chromosome 4's from the *su* tester and the other half have



one from each parent.

For RFLP loci that have different alleles in the original telocentric-4Sc and *su* tester parents, the following relationships will exist. If the RFLP locus is located on the long arm of chromosome 4, half of the non-sugary backcross progeny will display both alleles and half will only display the allele that was present in the tester parent. However, if the locus is located on the short arm, both alleles will be displayed in all of the non-sugary progeny.

Leaf samples from the original telocentric-4Sc plant, the unrelated *su* tester, and non-sugary and sugary backcross progeny were harvested, freeze-dried, and DNAs were isolated from each plant type. We analyzed DNAs of the original telotrisomic-4Sc plant, the unrelated *su* tester, and ten telotrisomic (non-sugary) backcross progeny with a probe for RFLP locus BNL15.45. We found that the RFLP alleles for both the original telotrisome-4Sc and the tester parent were present in six of the F1 progeny and only the allele from the original telotrisome-4Sc was present in the other four F1s. A similar banding pattern was obtained for RFLP locus *bnl7.20*. These results indicate that these RFLP loci reside in the long arm of chromosome 4. When these same plants were explored with probes for RFLP loci *bnl5.46*, *npi386*, and *umc47*, both alleles were present in each of the F1s. Clearly, these RFLP loci are located in the short arm. Therefore, the centromere of chromosome 4 is located between RFLP loci *bnl15.45* and *umc47* as shown below:



Previously, we (Weber and Helentjaris, Genetics 121:583-590, 1989) explored B-A translocation hypoploids with RFLP probes. We were able to localize the centromere of chromosome 4 to a region between RFLP loci *npi27*, *npi77*, and *npi95* (which mapped near each other in the short arm) and NPI250 in the long arm (a region spanning 26 map units). The current study localizes the position of the centromere to a region of 1 map unit. We will be exploring other telocentrics using the same experimental approach.

NORTHFIELD, MINNESOTA
BioTec Innovation

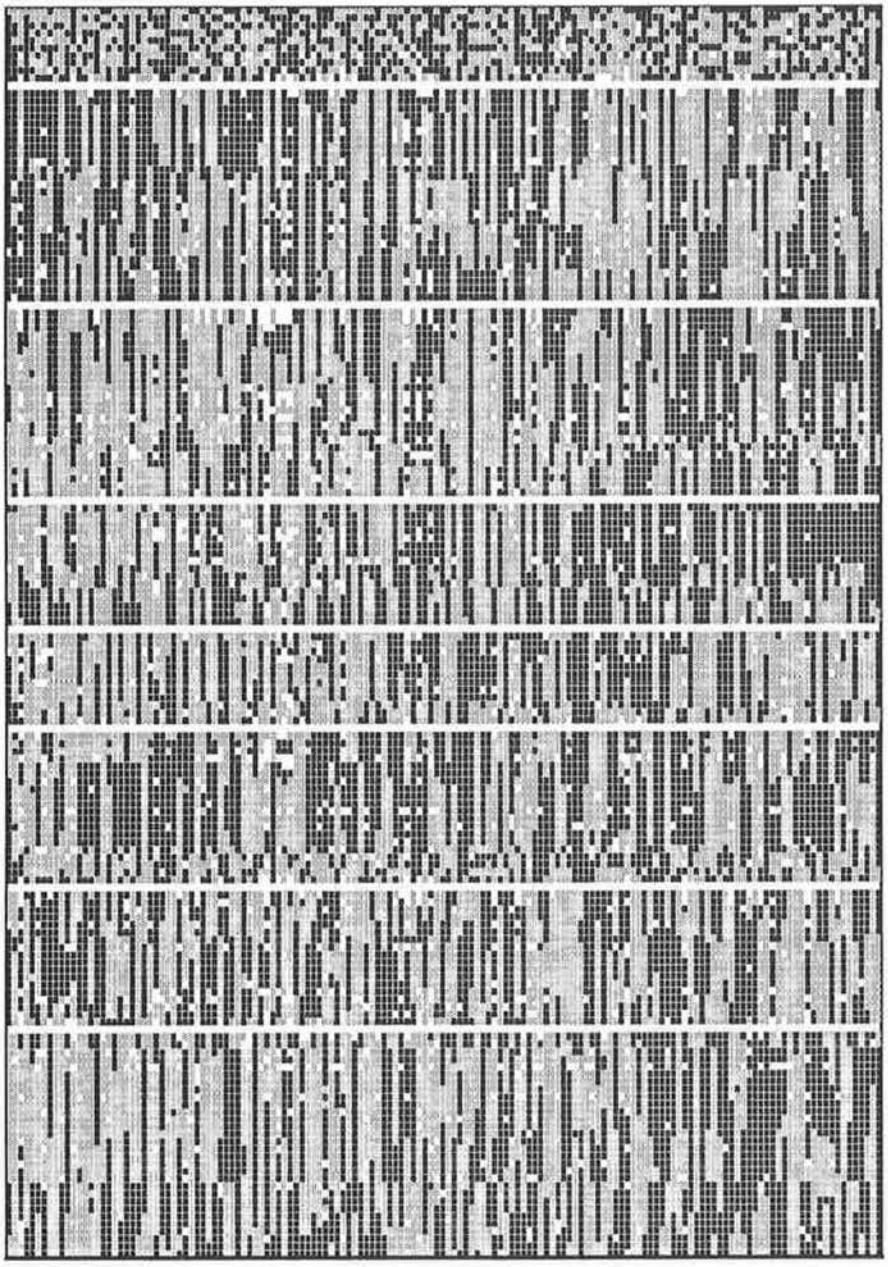
Illustrating multigene mapping data in a spreadsheet format

--Edward Weck

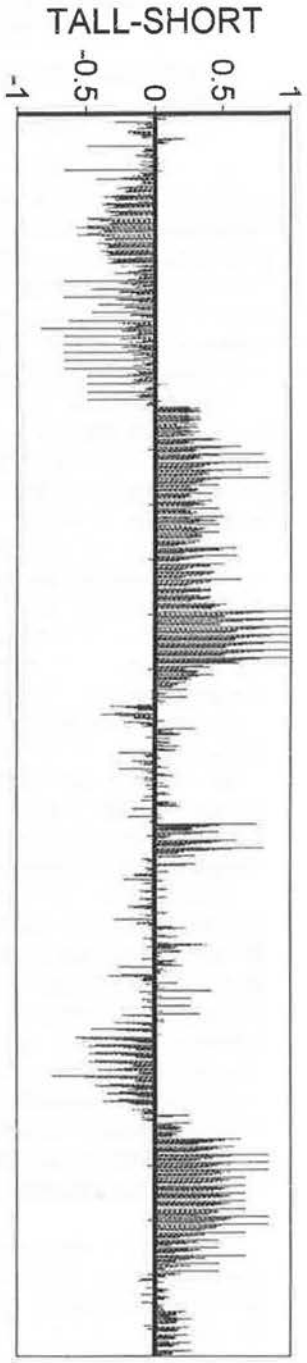
A number of statistical methods are available for distinguish-

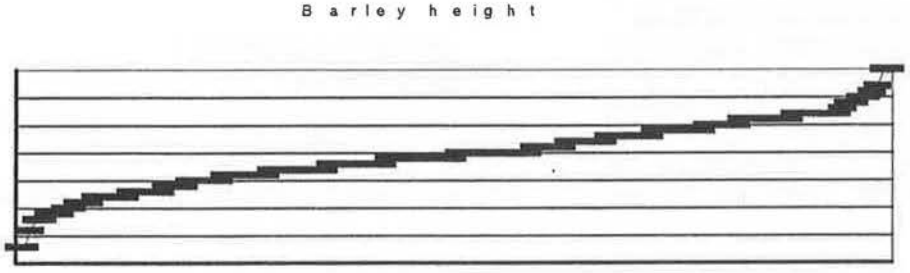
Barley Hite Sort

Individual Plant



Chromosome





ing genetically important regions from random effects in mapping populations. These methods vary in complexity from regression analysis to interval mapping. Plant F2 pedigrees are much simpler than the human pedigrees used for mapping multigenic traits. In a plant F2 population, the entire dataset consists of but three genetic alternatives; the two parental alleles and the heterozygote of the two parents. With plants, it is also possible to grow populations of unlimited size which helps reduce the complexity of multigenic mapping. I thought it would be possible, because of this underlying genetic simplicity, to gain further insight into the genetic underpinnings of a phenotypic trait via a graphical data representation. The entire data set could be "spectrally mapped," with each possible allele represented by a different gray scale value.

Because of the extremely high economic and scientific value of maize datasets, I was unable to obtain any maize molecular marker data associated with phenotypic traits. To allay this problem, I obtained a barley dataset from the Grain Genes database on the Internet (courtesy of the North American Barley Genome Mapping Project). The molecular marker and phenotypic data were from a doubled haploid cross of Steptoe x Morex (150 plants, 150 probes). A doubled haploid dataset is simpler than an F2 dataset with only the two parental alleles possible. Along with the genetic data, I also downloaded the marker mapping information and phenotypic data for plant height.

An ordered array of genotypic and phenotypic information is displayed in Figure 1. The Steptoe allele is encoded by a gray square, the Morex allele by a black square and missing data by a white square. The individual lines are sorted by increasing plant height (shown at the bottom) along the x-axis and by chromosome number (and probe position) from top to bottom along the y axis. Unmapped probes are shown at the top, followed by probes on the seven barley chromosomes separated from one another by white lines in the graphic.

The unmapped probes at the top of the figure exhibit a random pattern, as they are not organized with regard to either genotype or phenotype. In contrast, one parental allele predominates on certain chromosomal segments when comparing the tallest and shortest plants. A preponderance of the Morex allele on chromosome three, and possibly seven, and the Steptoe allele on chromosome one seemed apparent from looking at this graphic.

After pondering the graphic for some time, I decided some sort of summary statistics would be needed to assure completeness in interpretation. The most intuitive way to analyze the data, I thought, was to compare multiple slices of the data. The tall-short summary on the right of the figure compares the averages of: the (tallest five - shortest five) genotypes, the (tallest ten - shortest ten) genotypes, ..., the (tallest half-shortest half) of the population. The same result was observed for the (tallest five - shortest five) as with the (tallest half of the population -shortest half of the population) with the exception of the long arm of chromosome one. Here the genetic effect was exaggerated in the (tallest five - shortest five) because of missing data. An effect more consistent with the remainder of the population was, however, observed in the (tallest ten - shortest ten).

There is more than one way to be tall. Although a region on chromosome three appears to be necessary for plant height, tall plants do not have an absolute requirement for the other "tall" genes. Breeding for tallness would require the inclusion of the top three or four "tall" genes when these genetic regions didn't conflict with other phenotypes of breeding importance. This lack of absolute necessity for certain "tall" genes would seem to allow a certain flexibility in the creation of improved breeding materials.

When I started this analysis I thought that I should be able to see the genetically responsible regions in a population by merely looking at the raw data. With this graphical data presentation one can readily see the complete genotype of every individual within the population as well as where recombination has taken place in the creation of that individual. It is also easy to envision the effects of pooling individuals for measurements involved in mapping multigenic traits. Improving a population for a single multigenic trait simply requires growing a large population and analyzing the five or ten best and worst phenotypes. In addition, it should be possible to learn more from available inbred databases if the molecular marker data are presented in conjunction with phenotypic data.

I have taken advantage of a number of commercially available programs in creating this graphic, including: Access, Corel Chart, Lotus Improv, and Quattro Pro. (The original inspiration for this graphic came from The Computational Brain, p.114, by Churchland and Sejnowski, 1992.) I am currently working on an application that uses the spreadsheet format to display genetic information and allow rapid switching between various phenotypic sorts.

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QTL for drought responses in an F2 population

--Steve Quarrie, Claude Lebreton, Vesna Lazic-Jancic and Andrew Steed

We have been studying the physiological basis of differences in drought resistance amongst inbred maize lines for several years. Much of this work has been focused on the role of the stress hormone abscisic acid (ABA) in mediating stress responses. As ABA content appears to be inherited quantitatively we have started a program to map loci that regulate ABA production so that we can test for the presence of coincident QTL for a wide range of other traits associated with drought response.

We chose as our mapping population 81 F2 plants derived from a cross between two inbred lines that have been shown in previous work to differ in a wide range of responses to drought stress. In particular, they differ markedly in their leaf ABA contents, both under non-stressed and drought stressed conditions: F-2 (low-ABA, drought sensitive) and Polj17 (high-ABA, drought tolerant). Plants were grown in a soil glasshouse and sampled for leaf ABA content under non-stressed conditions at flowering time and then under mild drought stress after three weeks without water. Xylem sap was expressed from droughted leaves in a pressure chamber. At maturity plants were pulled out of the ground using a screw mechanism attached to a spring balance to measure the maximum force required to pull up the plant (root pulling force), and the number of roots in the root whorl at the base of the stem was counted. Leaf samples were also collected for anatomical measurements as the parents are known to differ significantly in xylem vessel dimensions and cuticular thickness.

So far about 50 RFLP markers (mainly from the UMC collections) have been mapped, with an average of ten markers covering each of chromosomes 1, 3, 6 and 9. This allowed us to estimate the location on these chromosomes of QTL for the traits using MAPMAKER-QTL. Chromosome 1 showed evidence of QTL regulating

ABA content in non-stressed and stressed leaves and in xylem sap. Chromosome 3 had major QTL for stressed leaf (maximum log-likelihood 8.0) and xylem sap (maximum log-likelihood 4.5) ABA contents, but on different regions of the chromosome (Fig. 1). No QTL for non-stressed leaf ABA content was found on this chromosome. Chromosome 6 had a significant QTL for xylem ABA content (maximum log-likelihood 2.6), but no evidence of any QTL for leaf ABA content.

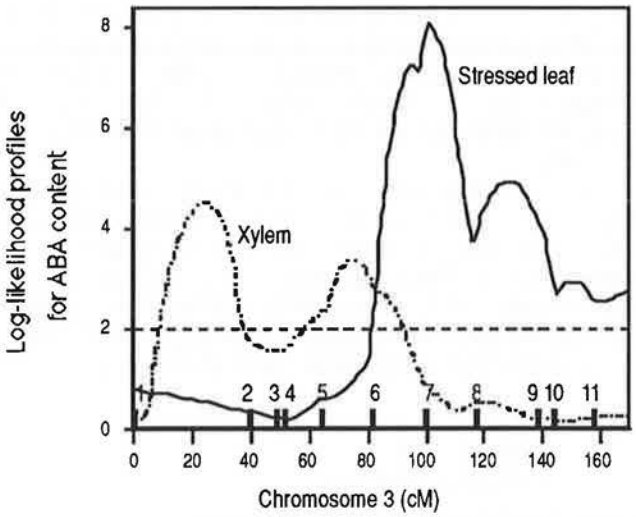

Figure 1. Log-likelihood profiles for stressed leaf and xylem sap ABA contents for chromosome 3. Thick bars indicate the position of RFLP markers on the chromosome. Markers: 1-*umc121*, 2-*umc10*, 3-*umc102*, 4-*umc26*, 5-*bnl5.37*, 6-*umc60*, 7-*umc39*, 8-*umc16*, 9-*umc63*, 10-*umc96*, 11-*umc2a*.

QTL for stomatal conductance were widespread throughout the genome, and were consistently associated with QTL for leaf ABA content. The QTL for xylem ABA content on chromosome 6 was not associated with any effects on stomatal conductance, and it seems likely that in these plants the ABA content of the whole leaf had a greater role in the regulation of stomatal conductance than xylem ABA content. A major QTL determining root number was found on chromosome 8 and root pulling force was determined by loci on chromosomes 1, 3 and 8. In general, QTL for root pulling force coincided with QTL for stressed leaf ABA contents, high ABA content being associated with a high root pulling force. Thus, it is possible that ABA made in the leaves may have a role in stimulating root development in a drying soil.

We are currently adding further RFLP markers to the map and have collected seed from the self-pollinated F2 plants for further growth cabinet and field tests on the F3 generation to study the robustness of these drought-related QTL.

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Light requirement for anthocyanin pigmentation of *C* aleurones

--Hugo K. Dooner and Edward Ralston

The anthocyanin pigmentation conditioned in the maize aleurone by the *c-p* allele of *C* has been described as being "light-dependent". This usage implies that the pigmentation conditioned by a *C* allele might be light-independent. Yet, it has been known for years that pigmentation of *C* aleurones can be significantly reduced in plants carrying all the factors required for aleurone pigmentation and also *B-s*, a strong *B* allele, and *Pl*. These plants have very darkly pigmented husks which could partially block the amount of light that reaches the aleurone. This observation suggests that anthocyanin pigmentation in *C* aleurones is also light-dependent. To investigate this further we conducted a simple test.

We introduced the *B-s* and *Pl* factors, required for strong plant pigmentation, into a W22 stock carrying all the factors necessary for aleurone pigmentation. We grew these *B-s Pl* plants in the greenhouse and as soon as the first ear shoot tip emerged, we removed the subtending leaf and wrapped several layers of aluminum foil around the husks. The ear shoots were allowed to silk out under the aluminum foil and on the day of pollination the plants were moved into a dark chamber, where the pollen was collected and the self-pollination was performed. The foil was replaced and the plants were returned to the greenhouse, where they were allowed to continue development. One plant was left unwrapped until the day of pollination, at which time it was covered with aluminum foil, like in the first group; several control plants were not covered at all. Six weeks after pollination, a sufficient time for full aleurone pigmentation under standard greenhouse growing conditions, the wrapped ears were harvested, husked in the dark, and dried in the dark for several days in a commercial food dehydrator. Of the six ears that were harvested, two were moldy (probably because of the poor air circulation under the aluminum foil) and could not be scored; the remaining four had only colorless seed, including the ear that was only wrapped after pollination. The unwrapped controls produced, as expected, kernels with a moderate level of pigmentation. This first set of observations suggests that light is, in fact, required for anthocyanin production in *C* aleurones. The ears with colorless kernels were set aside for several days on a table by a window, where, unexpectedly, they developed pigment in kernels exposed to the light, but not in those covered by the paper label (Fig. 1). This second observation suggests that light can induce pigmentation of unpigmented, mature *C* aleurones. (Though the ears were dried down in a dehydrator, we did not determine the kernels' final moisture content). When germinated in the light, the colorless *C* seeds developed very strong red pigment in patches, as *c-p* kernels do.

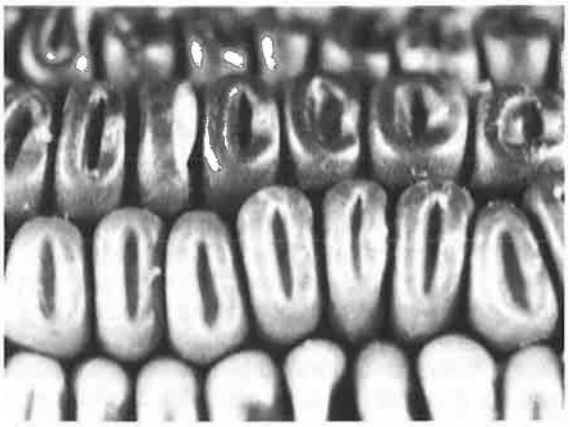

Figure 1. Close-up of *C* mature ear, which was colorless at harvest. The kernels in the top two rows were exposed to light and developed extensive pigmentation. The kernels in the bottom two rows were covered by a paper label and failed to develop appreciable pigments.

The above observations indicate that alleles of *C* that can elicit pigmentation differ in their relative, not their absolute, light requirement and suggest that anthocyanin pigmentation in the developing (and mature) aleurone requires light. These observations are in agreement with current knowledge of the photoregulation of anthocyanin biosynthesis, which indicates that anthocyanin pigmentation in plants is light-dependent (see e.g., A. Mancinelli, The Genetics of the Flavonoids, pp. 9-21. Unicopli, Milano, 1988).

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The pattern of distribution of the allele *Bg-3449* in inbred Zpl 2077/54-14

--Vladimir V. Koterniak

Earlier (MNL 67:90-91) *Bg-3449*, a new allele of the regulatory element *Bg*, was reported. This allele was found in a selfed generation of the hybrid between the normal line, Zpl 2077/54-14, and opaque line Sp168o2. Inbred Zpl 2077-54-14, unlike Sp168o2, when crossed with plants possessing receptive alleles of *o2*, gave in selfed generations variegated kernels, i.e. kernels with flint and opaque sectors. This indicates that the somatic instability observed earlier in 3449o2 is contributed by inbred Zpl 2077/54-14. For determining the pattern of distribution of *Bg-3449* in Zpl 2077/54-14, 53 plants of this line were crossed (as male parents) with an *o2-m(r)* no-*Bg* tester. All tested plants of inbred Zpl 2077/54-14 were obtained from a mixture of 13 selfed ears of the second generation of ear 83-864-1. This generation was obtained from the mixture of 6 selfed ears of the first generation of the same ear. F1 hybrids were selfed (3-5 ears on each cross). On selfed ears normal, variegated and opaque kernels were scored and the ratio of the sum of normal and variegated kernels to opaque ones was calculated. Evaluation of the significance of the difference between observed and theoretical frequencies was determined by the chi-square method.

All 177 selfed ears of testcrosses possessed normal, variegated and opaque kernels, which indicates homozygosity of all analyzed plants for *Bg-3449*. The presence of an active regulatory element in all plants tested occurs very rarely (Peterson and Salamini, Maydica 31:163-172, 1986; Peterson, MNL 64:8), though Peterson described the homozygous state of *En* in all 9 plants studied of the line 4Co63.

The major part of selfed ears (progenies of 38 plants out of 53) showed a ratio of normal and variegated to opaque kernels which did not differ significantly from 15:1. This suggests the presence of one regulatory element not linked to the *o2* locus. A ratio close to 63:1 that shows the presence of two independently assorting copies of *Bg-3449* was observed in the descendants of 21 plants of Zpl 2077/54-14. In progenies of 6 of them were ears with a ratio of 63:1 and ears with a ratio of 15:1.

In the selfed progenies no 3:1 ratio of normal and variegated kernels to opaque was observed. However, the results of kernel segregation on a set of ears suggest partial linkage between the *Bg-3449* allele and the *o2* locus in some cases. Thus, on 14 selfed ears, the progeny of 12 plants, the observed ratios differed from all theoretical ones studied. One part of these ears (5 ears belonging to descendants of 5 plants of inbred Zpl 2077/54-14) had a ratio of the sum of normal and variegated kernels to opaque that was intermediate between 3:1 and 15:1 (Table 1). The observed ratios of another part of the ears indicated (9 ears belonging to descendants of 8 plants) were intermediate between 15:1 and 63:1. Testcross progenies of the plant 92-5276p305 were from both parts. It is possible to consider the former part of the ears as descendants of plants possessing the regulatory element *Bg-3449* partially linked to the *o2* locus. The percentage of recombination between them was in the limits of 28.9-40.9 (Table 1). Accordingly, in the latter part it is possible to assume the presence of two copies of *Bg-3449* at least one of which is linked either to the *o2* locus or to another copy of the regulatory element.

Table 1. Recombination value between *Bg-3449* and the *o2* locus in ears where the ratio of the sum of normal and variegated kernels to those opaque is intermediate between the theoretical ratios 3:1 and 15:1 and is significantly different from both of them.

Plant no. of inbred Zpl 2077/54-14	No. of analyzed ears of testcrosses	Phenotype of kernels* n	v	o	Ratio (n+v)/o	% recombination between *Bg-3449* and *o2* locus**
91-5276p305	92-5895-1	322	65	38	10.2	40.2
91-5280p301	92-5945-1	294	51	33	10.5	40.9
91-5281p297	92-5961-2	352	66	40	10.5	40.9
91-5682p71	92-5971-1	228	35	38	6.9	28.9
91-5682p74	92-5977-2	249	45	31	9.5	38.2

*n, v, o - normal, variegated and opaque kernels, respectively
**calculation was made using equation $r^2-2r+1-4c=0$, where r is recombination fraction and c is the ratio of opaque kernels to the total number of kernels from ear

In some tested plants, it also may be suggested that there is the presence of 3 copies of *Bg-3449*, at least one of which is linked either to the *o2* locus or to another copy. This assumption may be made proceeding from segregation on 13 ears (belonging to descendants of 10 plants) on which the ratio of the sum of normal and variegated kernels to opaque ones did not differ significantly from both 255:1 and from 63:1, and was in the limits of 100.3-166.0. It is necessary to mention that all the ratios which did not differ significantly from 255:1 also did not deviate significantly from 63:1.

It was established that the mutation of *Bg-3449* to the inactive state occurs very rarely (unpublished data), likewise the inactivation of the standard *Bg* allele (Salamini et al., Heredity 49:111-115, 1982). Approximate evaluation showed that the gametic frequency of this event is no more than $9x10^{-3}$. Therefore the diversity observed in inbred Zpl 2077/54-14 in the number of copies of *Bg-3449*, and its linkage strength with the *o2* locus, is possibly the consequence of the transposition ability of this allele.

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Selection of plants resistant to S-2-aminoethyl-L-cysteine

--Ricardo A. Azevedo and Paulo Arruda

Substantial progress has been made with genetic, biochemical and molecular techniques in the study of metabolism and development of plants. Plant tissue culture techniques have been widely used and have proved to be a useful tool for the induction, selection and study of mutants. They have also brought new insight into the role of enzymes involved in primary metabolism. However, for such an approach to be viable, protocols are needed for the establishment of callus, cell suspension and protoplast cultures, as well as plant regeneration. Furthermore, strategies for the in vitro

selection of mutants are fundamental.

Mutants selected in vitro with an altered aspartate kinase (AK) have been obtained in maize, leading to an overproduction of threonine in different plant tissues, including endosperm (Hibberd and Green, PNAS 79:559, 1982; Azevedo et al., Plant Sci. 70:81, 1990; Diedrick et al., TAG 75:209, 1990). The lysine analog S-2-aminoethyl-L-cysteine (AEC) has been used to select mutants with an altered dihydrodipicolinate synthase (DHDPS), which is strongly inhibited by lysine and AEC. The relaxation of this regulatory step of the pathway could lead to an accumulation of soluble lysine in the maize seed.

Maize tissue culture was used to select plants resistant to AEC. Calli treated with NaN_3 and selected in MO9-2 medium containing 0.25 mM AEC showed the formation of necrotic and non-necrotic sectors. Normally growing sectors, when subcultured continuously into the same medium, showed resistance to AEC and were then transferred to the regeneration medium. Although normal callus growth has been observed after several subcultures in the presence of AEC, a small number of regenerating plants did not survive the AEC inhibition of root growth in the regeneration medium. However, 173 plants were regenerated, from which 63 reached the field stage and 35 of these reached maturity with the production of a panicle and ears, which allowed self-pollination and crossings to the original Cat 100-1 inbred line.

The mutagenic NaN_3 proved to be very efficient by the number of mutants observed. Meanwhile, the selection system still allowed a large number of escapes, since many mutants not related to AEC inhibition were obtained, as well as plants showing no alteration. This could be due to a gradient effect of AEC produced by a differential uptake of AEC by cells in the upper layers of the calli, where most of the normal growing cells were observed. Thus, successive subcultures of these normal sectors during the selection stage for a shorter period of time might reduce the number of undesirable mutants. Furthermore, the use of cell suspension and protoplast cultures could also show a better efficiency; however, even using cell suspension cultures, escapes are still a common feature, with at least 25% sensitive plants.

Anther spikelets from each regenerated plant were tested for soluble amino acid overproduction with special attention to lysine. Chromatograms showed a large variation in the concentration of different amino acids, including lysine. Plants AEC-5, AEC-6, AEC-11, AEC-8, AEC-14, AEC-23 and AEC-25 showed strong bands with the same R_f as that of lysine, while the control Cat 100-1, as well as other regenerated plants tested, did not show a band which corresponds to lysine.

Progenies obtained from regenerated plants were tested for resistance to AEC by inoculating excised immature embryos in the presence of AEC (Fig. 1). Among all progenies tested, only two, AEC-1 and AEC-5, showed segregation for AEC resistance.

Genetic analysis showed that for the AEC-1 resistant mutant, the resistance was a dominant trait segregating according to Mendelian laws (Fig. 1B). For the AEC-5 resistant mutant, on the other hand, the resistance was a recessive trait (Fig. 1C). These results were further confirmed when F1 progeny were produced by crossing AEC-1 (in the recessive homozygous condition, *aec-1/aec-1*) and AEC-5 (in the dominant homozygous condition, *Aec-5/Aec-5*). Both together, in the sensitive genotype condition to AEC inhibition, showed only embryos sensitive to AEC (Fig. 1D) as observed for the Cat 100-1 inbred line (Fig. 1A). All other regenerated plants were sensitive to the inhibition caused by AEC.

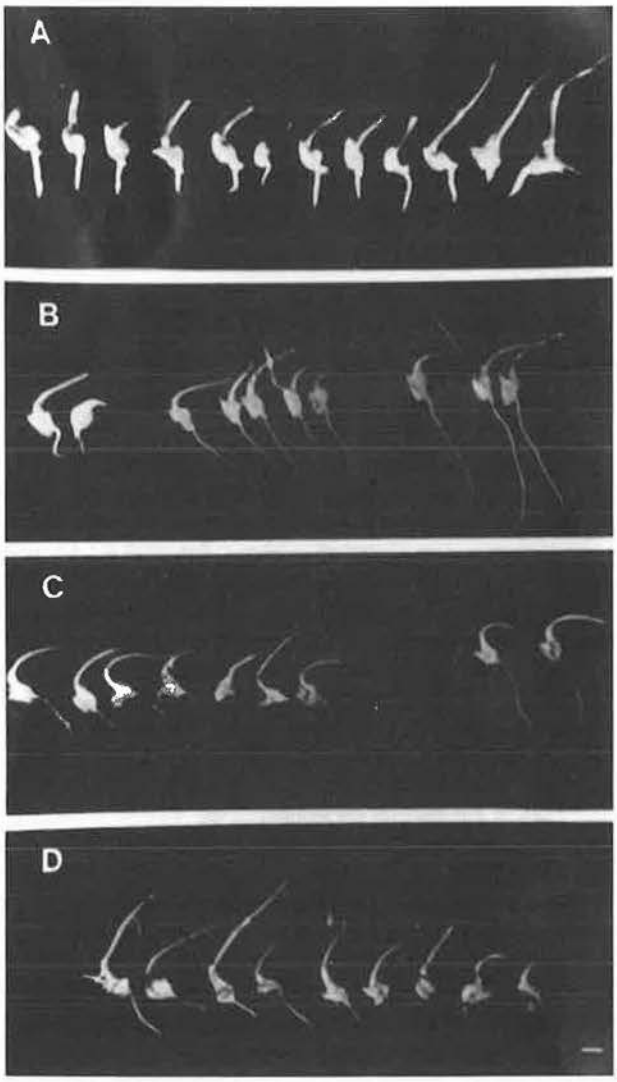

Figure 1. Growth of excised embryos of regenerated plants in medium MO9-2 containing 0.25 mM AEC. (A) Cat 100-1 sensitive control, (B) regenerated progeny AEC-1 showing dominant segregation for AEC resistance, (C) regenerated progeny AEC-5 showing a recessive trait for AEC resistance and (D) hybrid progeny (*aec1 aec1 Aec5 Aec5*) sensitive to AEC resistance. Bar = 5 mm.

Progenies derived from AEC-1 and AEC-5 resistant mutants and the AEC-10 and AEC-18 sensitive selected progenies, along with the Cat 100-1 inbred line (control), were used for soluble amino acid extraction and quantification by HPLC. Endosperms (30 per progeny) were individually analysed. It was observed that the AEC-10 sensitive progeny showed a 56% increase in the level of total soluble amino acids in comparison with the control, and also higher than the total soluble amino acid fraction of the resistant progenies analysed. Table 1 shows the results for soluble lysine levels. Although the absolute level of lysine was a little higher than in the control (0.24 and 0.19 $\mu mol \cdot g^{-1}$ endosperm in AEC-10 and Cat 100-1, respectively), its relative level was lower (3.78% of the total in the control and 3% of the total in AEC-10). For AEC-1 and AEC-5 the levels of soluble lysine were higher both in absolute (0.24 and 0.34 $mol \cdot g^{-1}$ endosperm for AEC-1 and AEC-5, respectively) and relative (5.85% and 5.55% of the total for AEC-1 and AEC-5, respectively) levels.

Table 1. Soluble lysine of the endosperm of regenerated plans after selection in medium containing AEC. Cat 100-1 represents the sensitive control.

Progeny	Soluble lysine (μmol·g^{-1} endosperm ± sd)	% of total
Cat 100-1	0.19±0.01	3.78
AEC-1	0.24±0.07	5.85
AEC-5	0.34±0.03	5.55
AEC-10	0.24±0.04	3.00
AEC-18	0.21±0.02	2.16

In maize, an accumulation of lysine in the endosperm has not been reported. This may be due to two main factors: first, for the lysine plus threonine resistant mutants, the DHDPS enzyme may still be sensitive to lysine blocking its synthesis at this regulation point of the aspartic acid pathway. Second, maize endosperms carry out a high rate of lysine degradation during seed development. This fact was first reported using [^{14}C]-lysine (Sodek and Wilson, Arch. Biochem. Biophys. 140:25, 1970). Two other enzymes, lysine-α-ketoglutarate reductase (LKR) and saccharopine dehydrogenase (SDH), were detected and characterized in maize (Arruda et al., Plant Physiol. 69:988, 1982), and these enzymes are involved in the degradation of lysine. LKR converts lysine into saccharopine and is present at high activities in developing endosperms. This enzyme is also involved with the translocation of nitrogen to the seed (Arruda and Silva, Phytochemistry 22:2687, 1983). Two hypotheses should also be considered for the importance of lysine degradation as one of the main factors for the accumulation of lysine in seeds. Firstly, during seed development, translocation of lysine to the endosperm occurs in excess of 2-3 times the necessary level of lysine for protein synthesis (Arruda and Silva, Phytochemistry 18:409, 1979). The excess lysine translocated is degraded in normal endosperms, but this does not occur in mutants rich in lysine. Secondly, LKR shows very low activity in *opaque2* endosperms, which are characterized by high levels of lysine (Arruda et al., MNL 58:50, 1984). This mutation does not affect SDH, therefore, LKR may be metabolising the excess lysine that could be accumulated in the resistant mutants. So, a further analysis for DHDPS in the AEC resistant mutants is being carried out to test these hypotheses.

Isolation of aspartate kinase from *Coix lacryma-jobi*

--Juverlandi Lugli and Ricardo A. Azevedo

Coix lacryma-jobi, together with maize, *Tripsacum* and sorghum, belongs to the grass tribe Andropogoneae. This cereal is native to Southeast Asia and has been used as a food source for humans and livestock, in the production of alcoholic beverages, and as a medicinal plant. Seeds of *Coix lacryma-jobi* contain around 29% protein, the major constituent of which is a prolamin called coixin. Like other cereal prolamins, the coixin polypeptides contain very low levels of lysine and tryptophan (Ottoboni et al., J. Agric. Food Chem. 38:631, 1990).

This cereal is now under biochemical investigation in order to study the biosynthesis of lysine, threonine, methionine and isoleucine (the aspartate family of amino acids).

The enzyme aspartate kinase, which has been isolated and purified in many higher plants, was extracted from *Coix* endosperms at different stages of development with 50 mM Tris buffer (pH 7.4) containing 200 mM KCl, 2 mM lysine, 2 mM threonine, 1 mM DTT, 0.1 mM EDTA and 15% (v/v) glycerol. Proteins from crude extracts were precipitated with ammonium sulphate (35-60%) and desalted on a Sephadex G50 column equilibrated with 25 mM Tris buffer (pH 7.4) containing 50 mM KCl, 1 mM DTT, 0.1 mM lysine, 0.1 mM threonine and 10% (v/v) glycerol. The desalted sample was applied to a Fast Flow Q Sepharose column equilibrated in the same buffer and eluted "step-wise" with 100 mM, 200 mM, 300 mM, 400 mM and 500 mM KCl in the same buffer. Aspartate kinase activity was determined by the optimized hydroxamate assay (Azevedo et al., Phytochemistry 31:3725, 1992) and protein by Bradford.

Aspartate kinase activity was extracted from 5 g of endosperm from each developmental stage. Stages 1 and 2 presented the highest levels of activity, and in both stages the amino acids threonine and lysine, at a concentration of 5 mM each, partially inhibited the activity of aspartate kinase, showing an additive effect when the amino acids were added together. Stage 2 was selected for further experiments based on the amount of endosperm that can be obtained in comparison to stage 1.

The anion exchange chromatography step showed that aspartate kinase could be eluted with 300 mM KCl, and the peak was also inhibited by threonine and lysine. These results indicated the presence of at least two forms of the enzyme in *Coix*; one sensitive to threonine inhibition, which corresponds to around 50% of the total activity (the major component) and the other sensitive to lysine (around 30% of the total activity). This result is different from those obtained in other plants, since in the majority the isoenzyme sensitive to lysine represented the major component. In other plants, aspartate kinase activity could be eluted with around 200 mM KCl, which was not enough to elute aspartate kinase from *Coix*. The peak containing aspartate kinase activity eluted from the Fast Flow Q Sepharose column was concentrated with 70% ammonium sulphate and is being tested in a Sephacryl S200 gel filtration column for molecular weight determination.

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Aspartate kinase activity extracted from seedlings of the *ask1* mutant

--Ricardo A. Azevedo and Peter J. Lea

The growth of cell cultures and seedlings of many plants can be inhibited by lysine plus threonine due to feedback inhibition at one or more steps in the aspartic acid metabolic pathway (Green and Phillips, Crop Sci. 14:827, 1974). The selection of mutants showing resistance to the inhibition caused by lysine plus threonine has shown that less sensitivity to feedback regulation leads to an overproduction of soluble threonine (Hibberd and Green, PNAS 79:559, 1982). In barley, lysine plus threonine resistant mutants showed that mutant forms of aspartate kinase isoenzymes were less sensitive to lysine feedback inhibition (Arruda et al., Plant Physiol. 76:422, 1984).

In maize, mutants resistant to inhibition by lysine plus threonine were also obtained and the enzyme aspartate kinase was extracted from maize ears and cell culture and analysed (Dotson et al., Planta 182:546, 1990). The *ask1* gene, which is the structural gene for aspartate kinase, was transferred to the Cat100-1 inbred line, mapped in the short arm of chromosome 7, and shown to be regulated by the *opaque2* gene when soluble and total amino acid fractions were analysed (Azevedo et al., Plant Sci. 70:81, 1990). This work represents a continuation of investigations on the *ask1* mu-

tant gene.

The mutant *ask1* gene was transferred from line A619 to Cat100-1 by backcrossing to near isogenic conversion. Maize seeds from ears containing separately *Ask1/Ask1* and *ask1/ask1* genotypes were planted in trays containing organic compost, incubated at 28 C in a 16/8 h light/dark period for 10 days, and water added at 48 h intervals. At the end of this period (seedlings normally showing 5 leaves) the leaves were harvested and the weight recorded. The leaf samples were immediately frozen with liquid nitrogen and used for aspartate kinase extraction.

For the identification of the presence of the *ask1* gene in the homozygous dominant form, 40 seeds from each of 8 segregating ears were planted and grown to maturity in a greenhouse (Lancaster University Field Station) at 24 C. Anther spikelets from each plant were collected and stored at -80 C. Anthers were then used for extraction of soluble amino acids. One anther spikelet from each plant was homogenized with 30 µl of distilled-deionised water in 0.5 ml microfuge tubes using a glass rod. The homogenates were centrifuged in a microcentrifuge at 16000 rpm and the supernatant used for amino acid analysis. Aliquots of 5 µl were applied to thin-layer chromatography (TLC) on glass plates (20x20 cm) coated with 0.5 mm of a 2:5 (w/w) mixture of silica gel and cellulose. The chromatogram was developed in a solvent mixture containing butanol, acetone, ammonium hydroxide and water (5:5:2.5:1, by volume) for 90 min. After the separation of the amino acids, the plates were dried for 3 days at room temperature and sprayed with 0.2% (w/v) ninhydrin in acetone. Standard threonine (1%, w/v) solution in water was used to identify the amino acid spots.

The segregation of the *ask1* gene was recorded and seeds from ears homozygous (*Ask1/Ask1*) for the gene were selected for the experiment.

Aspartate kinase was extracted and partially purified from 10 g of tissue for each genotype. Samples were ground with liquid nitrogen in a pestle and mortar with 5:1 (v/w) extraction buffer to 5% (w/v) polyvinylpyrrolidone. The extraction buffer contained 50 mM Tris/HCl ph 7.4 with 50 mM KCl, 2 mM lysine, 2 mM threonine, 1 mM DTT, 0.1 mM PMSF, 20% (v/v) ethanediol and the extract was filtered through several layers of gauze. After ammonium sulphate precipitation (35-60%), protein pellets were resuspended and desalted on G25 Sephadex columns equilibrated with Tris buffer (pH 7.4) containing 50 mM KCl, 0.1 mM lysine, 0.1 mM threonine and 10% (v/v) ethanediol. Aspartate kinase activity was measured by the hydroxamate assay method (Azevedo et al., Phytochemistry 31:3725, 1992).

The identification of the *ask1* gene had to be carried out since the seeds were segregating (*Ask1/Ask1:Ask1/ask1:ask1/ask1*) for the gene. A large number of seeds from each of the 8 ears were selected and planted, and grown to maturity, producing panicles from which anther spikelets were collected and tested. Some of the plants were self-pollinated or crossed. The extraction of soluble amino acids from anther spikelets and their identification and quantification by TLC has been shown to be a reliable indicator of the presence of the *ask1* gene. This was also the case in these experiments, since very clear differences in the levels of threonine among the genotypes could be observed (Fig. 1). From 8 ears tested, only ear number 7 showed the gene in the homozygous dominant form (*Ask1/Ask1*) with all plants being threonine overproducers. The analysis of the other 7 ears showed that two of them, 3 and 4, were normal (*ask1/ask1*) whereas ears 1, 2, 5, 6 and

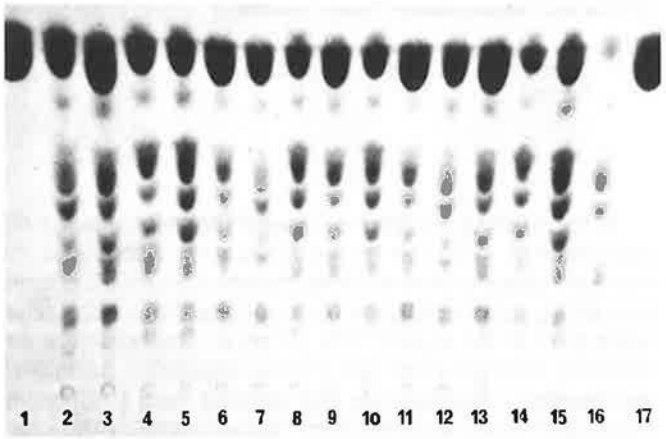

Figure 1. Thin layer chromatograms of amino acids extracted from anthers for the identification of the *ask1* gene, which leads to overproduction of threonine. Lanes 1 and 17 correspond to the threonine standard; 2-15 correspond to different plants showing threonine accumulation and 16 corresponds to the negative control (wild type).

8 showed segregation for the gene (Table 1). This experiment was carried out with the objective of identifying the *ask1* gene, therefore no quantitative measurements were carried out to separate *Ask1/Ask1* from *Ask1/ask1* since both genotypes overproduce threonine. Table 2 shows the results obtained when aspartate kinase activity was determined in wild type (*ask1/ask1*) and in the mutant (*Ask1/Ask1*) plants. Aspartate kinase was only slightly higher in the mutant than in the wild type plants. However, variation in the inhibition caused by lysine was observed. Lysine (5 mM) produced a 65% inhibition of aspartate kinase activity in the wild type, while the same concentration of lysine showed a 41% inhibition in the mutant. Threonine, on the other hand, did not show any variation, giving identical levels of inhibition of aspartate kinase activity between the two genotypes tested. This result confirmed the results reported by Dotson et al. (Planta 182;546, 1990), who analysed two mutations (one of them the *ask1* gene) in

Table 1. Segregation of the *ask1* gene in 8 progenies obtained from self-pollination of *Ask1/ask1*. The segregation of the gene was verified by the threonine levels of different genotypes in TLC plates.

Maize ear no.	Seeds planted	Produced panicle	Threonine overproduction +	Threonine overproduction -	Genotype of self-pollinated parent
1	40	7	5	2	*Ask1/ask1*
2	40	4	3	1	*Ask1/ask1*
3	40	11	0	11	*ask1/ask1*
4	40	7	0	7	*ask1/ask1*
5	40	9	6	3	*Ask1/ask1*
6	40	5	4	1	*Ask1/ask1*
7	40	12	12	0	*Ask1/Ask1*
8	40	15	11	4	*Ask1/ask1*

Table 2. Aspartate kinase activity (nKat/mg protein) extracted from wild type and mutant seedlings for the *ask1* gene.

	Genotypes for *ask1*			
	ask1/ask1		*Ask1/Ask1*	
Treatment	Activity	Inhibition (%)	Activity	Inhibition (%)
Control	0.0141	0	0.0153	0
Thr (2 mM)	0.0104	26	0.0112	27
Thr (5 mM)	0.0097	31	0.0109	29
Lys (2 mM)	0.0062	56	0.0109	29
Lys (5 mM)	0.0049	65	0.0090	41
Lys+Thr (2 mM)	0.0028	80	0.0076	50
Lys+Thr (5 mM)	0.0015	89	0.0067	56

which aspartate kinase activity was isolated and purified from immature maize ears. However, the extent of the lysine inhibition was different. This may be due to the fact that a different tissue was used, which might alter the effect of these amino acids on the enzyme activity. These results also confirmed that the *ask1* gene is one of the structural genes for the lysine-sensitive aspartate kinase in maize. Although Dotson et al. (Planta 182:546, 1990) could not identify the threonine-sensitive form of aspartate kinase, these results strongly support the affirmative above, since the inhibition by threonine was not altered in the mutant. Thus other genes may be responsible for the threonine-sensitive aspartate kinase isoenzyme in maize.

The addition of lysine and threonine together showed a clear additive pattern of these two amino acids on the aspartate kinase activity. In the mutant, the addition of lysine plus threonine did not show a strong aspartate kinase inhibition as observed for the wild type due to the reduced sensitivity of the lysine-sensitive aspartate kinase. However, this reduction was smaller than the levels expected for an additive effect of the two amino acids as shown by wild type plants.

Additional experiments with anthers, seedlings and endosperms are being carried out to better characterize the *ask1* gene in maize.

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Quantitative extraction of pericarp pigments

--O. Prem Das, Margaret Morales and Joachim Messing

Several genes, including *P*, *R* and *A1*, control pigmentation in kernel pericarp, and for *P*, alleles that vary over a wide range in phenotype are known. For example, mutable alleles of the *P-vv* type can give rise to different degrees of variegation and background pigmentation: stable alleles can also condition different degrees and patterns of pigmentation. In addition, we (MNL 67; Das and Messing, Genetics, in press) have described an allele (termed *P-pr*), originating from epimutation of *P-rr*, that can generate a range of variegated pigmentation on its own, as well as in heterozygotes with *P-rr* (see note below). A quantitative measure of pigmentation would be useful in the characterization of these alleles and their interactions, and therefore, we have developed the following simple method for pigment extraction from pericarp.

Kernels (5 per ear) are soaked in water for 2 hrs or more, and pericarps are manually peeled and placed in a tared Eppendorf tube with a pierced cap. Tissue is dried by overnight lyophilization, and the tube is weighed to determine dry tissue weight (~50mg). Then 0.2 ml conc. HCl is added to this tube, followed by 0.8 ml dimethyl sulfoxide (DMSO). These reagents should be added sequentially with vigorous vortex mixing after each addition. The tube is centrifuged briefly to clarify the suspension, and 0.05-0.2 ml, depending on the intensity of color, is diluted with 1 ml methanol for absorbance readings (λ-max = 510 nm for *P* pigments, 530 nm for *r-ch* pigments).

The extracted pigment is stable; there is only a slight increase (<10%) in the absorbance and little change in the absorption spectrum of the extract upon overnight extraction compared to a 0.5 hr treatment, and a similar increase in the absorbance of the methanol-diluted sample. More than 90% of the pigment is recovered in a one-hour extraction. Grinding of the tissue slightly increases extraction efficiency, but is unnecessary for routine comparative analysis. Pigments that depend on the *P* gene can be recovered after workup in acetone for further analysis, which we have not pursued. It is likely that the strong acid treatment affects the chemical structure of these pigments. However, treatment with milder acid does not give quantitative extraction of P pigments, and results in two absorption maxima at 510 and 570 nm. Longer treatment or stronger acid conditions result in a progressive loss of the 570 nm peak, and a concomitant increase in OD at 510 nm, indicating a precursor-product relationship between the species that give rise to these peaks. Under the conditions given above, the 570 nm peak is present, if at all, as a slight shoulder. The *r-ch* pigment gives a simple, narrow absorption spectrum in the visible range, suggesting that one or a closely related set of chromophores are present. The absorption spectrum of the *P* pigment is more complex, with strong absorbance into the near UV. For both, absorbance accurately reflects pigmentation, as shown by testing mixtures of colorless and colored pericarps in known ratios. Furthermore, extraction is nearly quantitative, since the residue after one or two extractions is almost colorless. It is possible that this simple method may apply to the quantitation of pigmentation conditioned by other genes, and in other tissues.

Effect of *P-pr* on pigmentation conditioned by *P-rr*

--O. Prem Das, Barton Scott, John Lena and Joachim Messing

Two isolates of a new allele of the *P* gene, termed *P-pr* (for patterned pericarp, red cob), that originated by transmission of somatic epimutation of the *P-rr* allele were described previously (MNL 67; Das and Messing, Genetics, in press). *P-pr* was similar to *P-rr* in sequence, but was more methylated at both CG and CNG motifs. Its phenotype was characterized by variegated pigmentation in pericarp, but not cob, variability in pigmentation between siblings, and the presence of large clonal sectors that differed in pericarp pigmentation. In heterozygotes with this allele, the uniform red pigmentation conditioned by *P-rr* was reduced, and rendered qualitatively similar to that of *P-pr* in all the above respects. However, the reduction was variable, and many ears appeared fully red. To quantitatively demonstrate the reduction in pigmentation, we have used the assay described in the preceding note, and the following genetic schemes.

Two genetic schemes (Fig. 1, next note) were used to control for genetic background effects on *P-pr-1*. In one (Table 1, left columns), 8 plants of the genotype *P-ww/P-pr*, 4 sibling plants of genotype *P-ww/P-ww* and one *P-pr/P-rr* plant (all in the original background that *P-pr-1* was isolated in) were crossed to *P-rr* in the W22 inbred background. The genotype of the resulting plants (control genotype of *P-ww/P-rr* or test genotype of *P-pr/P-rr)* was determined by segregation in the next generation, and five random kernels from each of the resulting ears were used for quantitation of pigmentation. In the second scheme

Table 1. Reduction of pericarp pigmentation* of *P-rr* by *P-pr*.

	P-pr/P-ww, sibling *P-ww/P-ww* and *P-pr/P-pr* X *P-rr/P-rr*		*P-pr/P-rr* X *P-rr/P-rr*	
	Test *P-pr/P-rr*	Control *P-ww/P-rr*	Test *P-pr/P-rr*	Control *P-rr'/P-rr*
MEAN	48.8	99	58.9	145.8
σ	20.4	25	30.6	24.4
n	95	48	30	23

*Expressed as OD/mg dry tissue from pericarps of 5 random kernels of each ear.

(right two columns), 5 plants of the genotype *P-pr/P-rr* were crossed to *P-rr* in the W22 background, and again, the two resulting genotypes *P-pr/P-rr* and *P-rr'/P-rr* were distinguished by segregation in the next generation (*P-rr'* is used to designate the *P-rr* allele that has interacted with *P-pr* in the previous generation).

Relative to *P-ww*, *P-pr* reduces pigmentation of *P-rr* by two-fold; relative to *P-rr'*, reduction is closer to 2.5-fold. The true value is likely intermediate, since *P-pr* is capable of generating, by itself, more pigmentation than *P-ww* and less than *P-rr'* (see next note). The differences between the *P-pr/P-rr* mean values in the two schemes may reflect the genetic background, since the second scheme uses two successive crosses to W22 before the testcross, whereas the first uses only one. However, in each scheme, factors unlinked to *P* should be equalized between test and control. Individual pigmentation values in each data set fit a normal distribution; when grouped by frequency in ascending intervals, they approximated the expected bell-shaped curve. Quantitative measures of the fit, obtained by comparing the number of entries within 0.5, 1, 2 and 3 standard deviations of the mean in each data set to expected values, did not differ from expectations at a confidence level of 90%.

A heritable interaction between *P-pr* and *P-rr*

--O. Prem Das and Joachim Messing

The preceding note demonstrates that *P-pr* reduces the pigmentation caused by *P-rr*, indicating that the alleles interact. Here we present evidence for heritability of interaction, determined as modification of the phenotype of *P-rr'* (in analogy with the convention for paramutation at *R* and *B* loci) transmitted after interaction with *P-pr*. The genetic scheme used is shown in Figure 1. Test plants of the genotype *P-pr/P-rr*, and control plants of genotype either *P-ww/P-rr* or *P-rr'/P-rr* were testcrossed to *P-ww*. The resulting kernels were planted in blocks that yielded between 90 and 160 plants, and phenotypes were determined in the field on open-pollinated ears. Phenotypes were classified as white (colorless, expected from *P-ww/P-ww*), red (solid red color on all kernels, expected from *P-rr/P-ww*), patterned (variegated pigmentation on all kernels, expected from *P-pr/P-ww)* and a novel phenotype designated variegated red, which was largely red, but resembled the *P-pr* phenotype on some or all kernels of an ear. The distinction between variegated red and patterned was somewhat subjective, since it was based on the

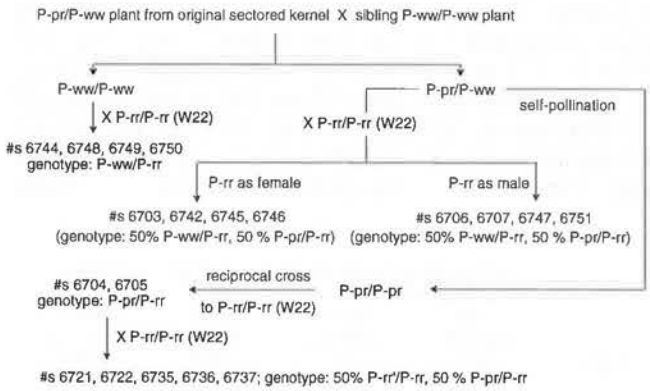

Figure 1. Genetic Scheme: the chart shows the lineage of the families whose members were testcrossed to *P-ww* to obtain the data in Figure 2 and Table 1.

criterion of which color, red or white, was more predominant on the ear. However, this does not affect the conclusions, as shown below.

Segregation data for the families described in Figure 1 are shown in Table 1. Testcrosses to *P-ww* of control populations of

Table 1. Segregation in testcrosses to *P-ww/P-ww* (4Co63).

Control populations of genotype *P-ww/P-rr*					
Family # (see Fig. 1)	# plants crossed	Red ears	White ears	Variegated ears	Chi-square
6744	4	301	308	0	0.08
6748	5	333	333	1	0
6749	7	304	362	0	5.05
6750	3	189	197	0	0.17
6703	6	294	306	0	0.24
6742	2	147	113	0	4.45
6706	15	1004	1079	0	2.7
6707	4	322	310	0	0.23
Total	46	2894	3008	1	2.2

Test populations of genotype *P-pr/P-rr*					
Family # (see Fig. 1)	# plants crossed	Red ears	Patterned ears	Variegated ears	Chi-square
6735	10	602	602	41	1.35
6736	4	167	224	43	23
6737	6	50	526	155	544.7
6722	3	189	190	2	0.02
6721	2	121	110	20	0.32
6703	9	514	505	31	0.46
6704	32	1658	2098	185	99.1
6705	24	1174	1666	211	162
6706	10	555	664	66	23.8
6707	6	394	472	51	18.1
6742	1	63	65	1	0.07
6745	5	284	379	44	27.3
6746	4	331	333	8	0.15
6747	2	145	160	9	1.83
6751	2	148	192	9	2.2
Total	120	6395	8186	876	460.7

genotype *P-ww/P-rr* gave 5903 ears from 46 subfamilies, representing 8 progenitor families. Virtually all ears were red or white; the single variegated red ear may be due to pollen contamination. Testcrosses to *P-ww* of a second control population of genotype *P-rr'/P-rr* yielded 2937 ears from 22 subfamilies, representing families 6721, 6722, 6735, 6736 and 6737 (Fig. 1); these were also all solid red, with no variegated red ears (data not shown in the Table). In contrast, almost all of the 15 test families of genotype *P-pr/P-rr* gave a significant number of variegated red ears, in addition to patterned red and solid red ears. A few white or almost white ears were also seen, which, if confirmed for the presence of a new *P-ww* allele, may represent loss of function derivatives of *P-pr* or *P-rr*.

The presence of the variegated red class in testcrosses of *P-pr/P-rr* may result either from an increase in pigmentation capacity of *P-pr*, or from a decrease in pigmentation capacity of *P-rr'*. These alternatives were tested by analysis of segregation ratios. The first alternative predicts that the sum of variegated and patterned ears should equal the number of red ears, while the second predicts that they should exceed it. Chi-square analysis of the departure from 1:1 segregation for both control and test populations is shown in Table 1 (underlined values are significant at the 95% level). In the control population, only two families deviate significantly from a 1:1 ratio of red and white ears, and these show opposing biases. The total shows no significant deviation, indicating that the genetic background or unlinked factors do not lead to consistently biased segregation. In contrast, in the test population, 7/15 families deviate at the 95% confidence level from

1:1 segregation of the patterned + variegated class relative to the solid red class. All of these represent decreases in the solid red class, leading to a highly significant chi-square value of 461 for the total.

Figure 2 shows the same data for each subfamily, ordered by decreasing frequency of red ears. For the control population

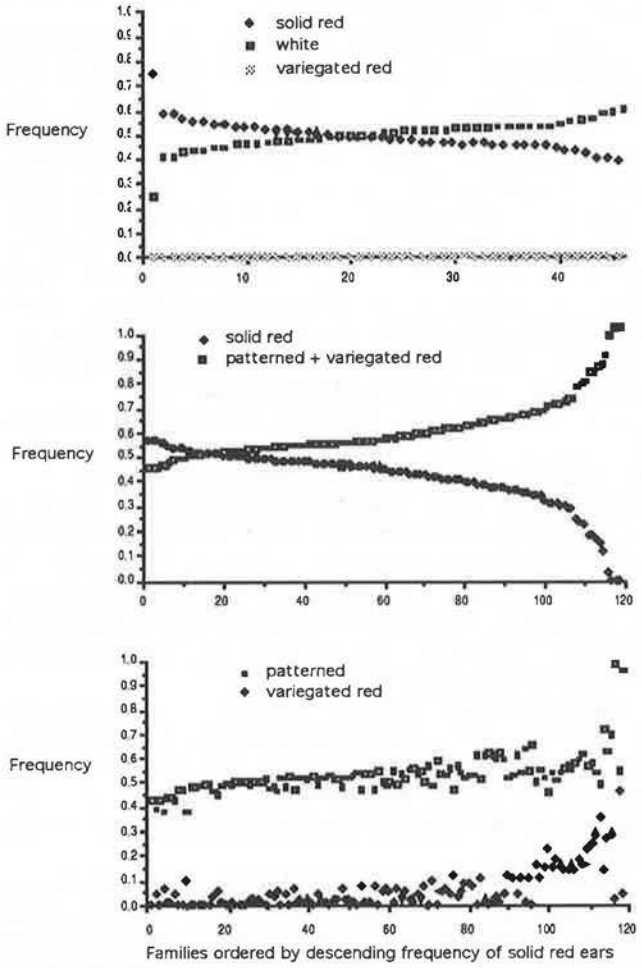

Figure 2. Segregation in Control and Test Populations: Segregation frequencies from the 46 subfamilies of the control population (upper panel) and 120 subfamilies of the test population (middle and lower panels) were ordered by decreasing frequency of red ears, and plotted.

(upper panel), all values adhere fairly closely to the expected value of 0.5, and deviations cancel out as indicated by the fact that the two curves cross close to the middle. In contrast, for the test population, segregation values are distorted, with fewer solid red ears in most families, as indicated by the crossing of the two curves near the left end. Progeny from the ears represented on the right-hand side of the range are highly deficient in red ears, suggesting nearly complete loss of the normal pigmentation capacity of *P-rr*. These observations indicate a similarity in the interaction between these two alleles to paramutation at the *R* locus.

Mapping of a novel δ-zein and a proposal for revising nomenclature of the δ-class zeins

--Sanjay Swarup and Joachim Messing

We have cloned and sequenced *dzs23*, a homolog of the 10 kDa zein gene. In order to map this duplicate gene, we used the recombinant inbred lines derived from the cross T232 X CM37 kindly provided by Ben Burr (Brookhaven National Labs.). *dzs23* maps to 6L-57 on the BNL map; the same as the *hex2* locus. This map location has been confirmed independently by Krone and Phillips (refer to their article in this issue) using the segregants from BSSS-53 X Mo 17 cross.

Since we know of at least two members of the δ-class of zeins, we would like to propose a revised nomenclature of these zeins. In doing so, we would like to include both the class designation as well as the molecular weight of the protein as estimated from SDS-PAGE analysis. We, therefore, propose to use the names *dzs10* (delta zein structural 10) for the 10 kDa locus (previously also called *zps10*) on chromosome 9 and *dzs23* for the 23 kDa locus (see accompanying note) on chromosome 6. In accordance with the standard nomenclature, their respective protein products would be DZS10 and DZS23, respectively.

Analysis of *dzs23*, which encodes the highest methionine containing zein

--Sanjay Swarup, Sumita Chaudhuri and Joachim Messing

Maize endosperm prolamins, or zeins, fall into four major classes viz., α, β, γ, and δ. Of these, zeins belonging to β- and δ-classes contain 10% and 22% of methionine/mol protein, respectively. Being an essential amino acid, methionine forms an important constituent in both human and animal diets. Previously, the cloning of a 10 kDa high-methionine zein gene belonging to the δ-class was reported by our laboratory (Kirihara et al., Mol. Gen. Genet. 211:477-484, 1988). We report here the cloning and analysis of *dzs23*, which has 86% DNA sequence similarity to the 10 kDa structural gene (referred to from here on as *dzs10*).

The predicted zein encoded by *dzs23* has a 21 aa long leader similar to that of DZS10 zein and the mature protein is 1.5 times longer than DZS10 (Fig. 1A). DZS23 is longer, due to an internal duplication thereby increasing its methionine content to 26% as compared to 22%/mol of DZS10. Surprisingly, the predicted DZS23 zein contains 1 lysine and 2 tryptophan residues. Presence of the lysine residue was confirmed by endoproteinase Lys-C digestion of DZS23 which released an 18 kDa peptide (data not shown). Both lysine and tryptophan are absent in the 10 kDa zein and normally underrepresented in other zeins.

Synthesis of a T7 Tag-DZS23 fusion protein allowed us to test the cross-reactivity of a polyclonal antibody directed against DZS10. The δ-zein antibody cross-reacted to the in vitro synthesized DZS23 fusion protein (data not shown). This result led us to study the levels of both DZS10 and DZS23 in various maize inbred lines (Fig. 1b). Three patterns of δ-zein levels were found. Inbred Mo17 was low in both; BSSS-53 was low in DZS23 but very high in DZS10; B37 and A619 had moderately high levels of both δ-zeins. The antisera used as control to detect α-zeins also react with lower sensitivity to δ-zeins. Protein blot of zeins from mature kernels (Fig. 1B) and from in vitro synthesis (not shown) provided a size estimate of 23 kDa for the novel δ-zein, thus the designation DZS23.

In order to simultaneously study the expression of both *dzs10* and *dzs23* in developing kernels, we resorted to using primer extension. An antisense oligo was synthesized which fit the following three criteria: (i) whose binding site was conserved between the two genes, (ii) which was ~ 100 bp from the transcription start site and (iii) whose extension products would show size polymorphism. The primer extended products corresponding to both δ-

Fig. 1A Sequence Comparison of δ-Class Zeins

```
DZS23  1 MAAKMFALFALLALCATATSATHIQALVTTTDAIGYHEPWMQYCMKQQGV  50
         |||||:|||||||||||.|||||||.: :... ::|  :| |||||.|||:
DZS10  1 MAAKMLALFALLALCASATSATHIPGHLPPVMPLGTMNPCMQYCMMQQGL  50

        51 ANLLAWPTLMLQQLLASPLQQCQMPMMMPGMMPPMTMMPMPSMMPSMVPT 100
           |.|:| |.||||||||| |||
        51 ASLMACPSLMLQQLLALPLQ.............................. 70

       101 MMSPMTMASMMPPMMMPSMISPMTMPSMMPSMIMPTMMSPMIMPSMMPPM 150
                                  ||| ||| |: |.|||||:|||||.||
        71 ......................TMPVMMPQMMTPNMMSPLMMPSMMSPM  97

       151 MMPSMVSPMMMPNMMTVPQCYSGSISHIIQQQQLPPMFSPTAMAIPPMFL 200
           ::|||:|.|||      |||.:::|:|: ||||||||.| ||.|||||| 
        98 VLPSMMSQMMM......PQCHCDAVSQIMLQQQLPFMFNPMAMTIPPMFL 141


       201 QQPFVGAAF 209
           |||||||||
       142 QQPFVGAAF 150
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Fig. 1B

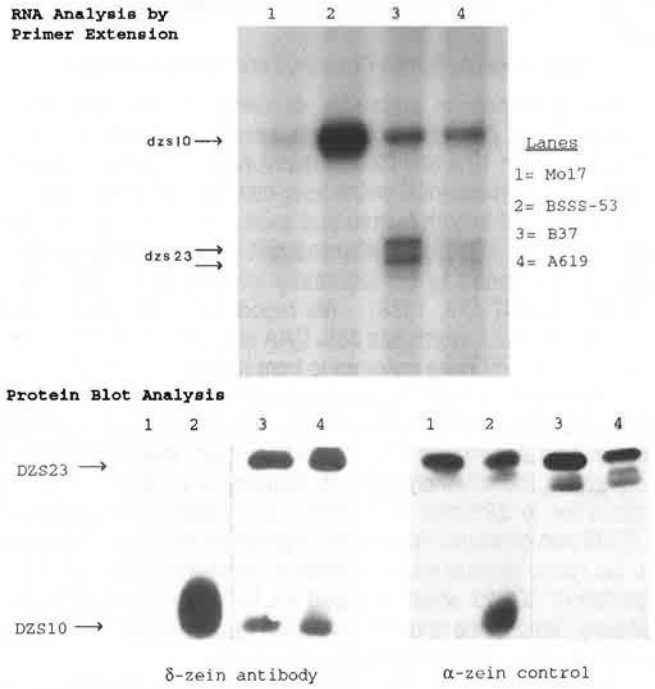

Figure 1. Characterization of *dzs23* gene.

zeins in four maize inbred lines is shown in Fig. 1B. The doublet corresponding to *dzs23* transcript is possibly due to strong secondary structures at the 5'-end as predicted from its sequence. δ-zein transcript levels at 18 DAP follow a similar profile as the proteins in mature kernels. Whereas low levels of *dzs23* RNA are detected in Mo17, no transcripts are detected by this analysis in BSSS-53. Identity of *dzs23* transcript from Mo17 was confirmed by cloning and sequencing the RT PCR product. This analysis also revealed at least two polyadenylation sites in the mRNA. Based on its high methionine content as well as the presence of both lysine and tryptophan, DZS23 may be a good candidate for improving the nutritional quality of corn using the transformation technology.

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A new maize ring chromosome - *ring10:A1179*

--Mary Alleman

Ring chromosomes, although rare, have been described occasionally involving maize chromosomes. In this report, I announce the identification of a chromosome 10 ring.

Origin. *ring10:A1179* originated in a *Mutator* stock of maize and was recovered while I was screening kernel populations for *Mu*-induced mutations of the *R* locus. The initial selection appeared as a coarsely sectored kernel quite dissimilar from a typical finely spotted, *Mu*-induced mutation of a maize anthocyanin pathway gene. In subsequent crosses I found that the transmission of the "mutation" was not consistent with the segregation of one or two genes: progeny ears from test crosses of A1179 with *r-g* consistently produced 0-10% sectored progeny kernels with most ears having no sectored kernels. Although loss of *Mutator* activity could explain the colorless kernels, a ring chromosome was the preferred explanation because of the atypical *Mu* spotting pattern and the absence of *Mutator* symptoms in all plants.

Cytological evidence. In order to make cytological verification of a ring chromosome, I harvested several immature tassels from A1179 plants derived from sectored kernels and visited Miss Ellen Dempsey and Dr. Marcus Rhoades in Bloomington, IN. Because the suspected ring chromosome was transmitted only occasionally, many preparations were examined. Miss Dempsey's gifted hands and eyes found that a ring chromosome, best indicated in diakinesis, was unmistakable in several chromosome spreads deriving from one plant. From rough estimates, *ring10:A1179* appeared to involve approximately one half of chromosome 10.

Genetic evidence. Overlap of *ring10:A1179* with 10L markers besides *R* has not been especially enlightening. The genotype *golden1/golden1*; *ring10:A1179* produces sectored *g1, G1* plants indicating overlap of *ring10:A1179* with *g1* (not surprising as *g1* is proximal to *R*). A similar experiment using *w2* (distal to *R*) has been inconclusive.

Transmission. Transmission of *ring10:A1179* was recently tested using the stock *r-g/r-g*; *ring10:A1179*; W22 (six generations backcrossed to W22). In reciprocal crosses with *r-g* tester plants, *ring10:A1179* is transmitted in different frequencies dependent on the mode of transmission. Pollen transmission produces the highest frequency of pigmented kernels or embryos, approximately 13%. Maternal transmission produces approximately 6% pigmented kernels. Plant by plant frequency data follow: (plant number - male transmission frequency/female transmission frequency): (1 - 0.11/0.04), (2 - 0.10/0.04), (5 - 0.12/0.07), (6 - 0.18/0.07), (9 - 0.12/0.05), (12 - 0.17/0.03), (13 - 0.17/0.07), (14 - 0.09/0.08), (16 - 0.18/0.04), (17 - 0.12/0.05), (18 - 0.13/0.08). Kernels with pigmented embryos and colorless endosperm were only produced from pollen-transmitted ring 10 (4% of the pigmented kernels), possibly by heterofertilization. These were verified as noncontaminant by progeny testing and were included in the above data set. There were no plants grown from sectored kernels which did not produce sectored progeny kernels. This contrasts with plants grown in the first three generations in which most of the plants produced only colorless kernels. Possible explanations include the ring stabilizing spontaneously, in the W22 background, or the increase in number of ring chromosomes per cell.

ring10:A1179 may have originated via abortive transposition of a *Mu* element on chromosome 10 or by crossing over between two similar *Mu* elements. The origin of chromosomal abnormalities as a result of transposable element action is well documented, especially in *Drosophila*.

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Rajendra Agricultural University

Embryoid formation from cultured anthers of two inbreds and their hybrid

--Arti Kumari, Harsh Kumar, S. K. T. Nasar and M. Kumar

Low frequency of response has limited the use of anther culture technique for the production of haploid plants. With a view to improving results and gaining insight into genetical factors for tissue culture response, anther culture was done on two inbreds, PKMS and MSIDR, and their hybrid PKMS x MSIDR. Young tassels from selected healthy plants were cut from their bases, surface sterilized with 10% calcium hypochlorite solution, rinsed thrice with sterile distilled water and dissected. Anthers were placed on MS and N6 media supplemented with casein hydrolysate (500 mg/l), kinetin (1 mg/l) and 2,4-dichlorphenoxy acetic acid (2 mg/l). Sucrose was added at the level of 12% and 3% for N6 and MS media respectively. Active charcoal at the level of 0.5% was also added to the two media. An anther from each spikelet was kept and analysed for the pollen developmental stage.

On MS medium more anthers showed swelling but on N6 medium more anthers formed embryoids. A comparison of the anther size, pollen size and pollen developmental stage to the frequency of embryoid formation from the cultured anther was made. Results have shown that, contrary to earlier reports of middle uninucleate pollen stage being the most responsive, the pollen division (mitosis) stage and early binucleate stage showed better response (Table 1). Among the two inbreds and their hybrid, MSIDR showed better response than PKMS and the hybrid PKMS x MSIDR showed intermediate response for the frequency of embryoid formation from the cultured anthers (Table 2).

Table 1. Effect of pollen developmental stage on the frequency of embryoid formation from cultured anthers of MSIDR on N6 medium.

Sl no.	Anther size (length-breadth), mm	Pollen size (dia.), mm	Pollen development stage	Frequency of embryoid formation, %
1	1.71 - 0.28	0.049	Tetrad	0.86
2	1.99 - 0.32	0.062	Early uninucleate	1.37
3	2.28 - 0.37	0.068	Late uninucleate	3.87
4	2.57 - 0.42	0.074	Pollen mitosis	4.53
5	2.78 - 0.57	0.087	Early binucleate	4.35
6	2.99 - 0.71	0.101	Late binucleate	0.54

Table 2. Effect of genotype on frequency of embryoid formation from cultured anthers on N6 medium (approximate size 2.5x0.5 mm).

Sl no.	Genotype	Frequency of embryoid formation (%)
1	MSIDR	4.31
2	PKMS	3.06
3	PKMSxMSIDR	4.03

Cytological analysis of cultured anthers has shown that equal division of uninucleate pollen, development of the vegetative cell, and development of the generative cell were the three pathways for pollen embryoid formation.

A simple method for pollen karyotyping in maize

--Arti Kumari, S. K. T. Nasar, M. Kumar and H. Kumar

Except for the preliminary work of McClintock (1929) on pollen karyotyping in the first microspore division, no further attempt has been made in this regard. The present study was undertaken to standardize an efficient and easy method of pollen karyotyping in maize. A simple smear-cum-squash technique with 2% acetocarmine staining has been established for maize pollen using mature anthers of two inbreds, PKMS and MSIDR. Appropriate sized mature anthers were dissected in 2% acetocarmine and then were stored in 2% acetocarmine stain for 4-5 days. Then these anthers were squashed in a drop of fresh stain on the slide. Alternate warming, cooling and tapping of a coverglass placed on the material was done repeatedly for obtaining better spread of chromosomes and bursting of pollen grains. Overstaining followed by destaining with 45% glacial acetic acid resulted in good preparations. Another protocol of fixation of mature tassels in 3:1 alcohol-acetic acid mixture followed by overstaining and destaining of pollen also gave a high degree of success. The first microspore division was the appropriate phase for identifying individual chromosomes of the haploid complement.

Clearly delineated heterochromatic bands and knobs were visible during early prophase of the 1st microspore division. Gradually the bands, due to chromosomal condensation, lose their individual identity, while the knobs continue to maintain identity up to late prophase. The total length, relative length and arm ratio of individual chromosomes was found to be constant along with fixed position of heterochromatic knobs and bands. The karyotype of pollen chromosomes gives us a clear and better understanding of the genome being contributed to the next generation.

Cytotoxicity of a herbicide in maize

--M. Prasad, M. Kumar, H. Kumar and S. K. T. Nasar

Toxic effects of a commonly used herbicide, 'taffacide' of the triazine group, have been evaluated in the root tip mitosis of maize,

Table 1. Effects of taffacide on root tip mitosis in maize.

Sl no.	Concn. (ppm)	Treatment (h)	Recovery period (h)	Mitotic index (%)	Total abnormal cells	Frequency of abnormalities (%)						
						Despiralization	Fragmentation	Stickiness	Pulverization	C-mitosis	Laggard	Binu. cell	Giant cells
1	5	6	4	9.22	2.28	1.54	0.33	-	-	0.42	-	-	-
2	5	6	8	10.23	2.09	1.48	-	0.30	-	0.31	-	-	-
3	5	12	4	4.36	0.70	-	-	-	-	-	-	-	0.70
4	5	12	8	5.52	0.55	-	-	-	-	-	-	-	0.55
5	25	6	4	7.21	3.08	2.13	0.45	-	-	0.50	-	-	-
6	25	6	8	9.19	3.47	2.38	-	0.53	-	0.39	0.17	-	-
7	25	12	4	3.39	0.97	-	-	-	-	-	-	-	0.97
8	25	12	8	4.47	0.97	-	-	-	0.39	-	-	-	0.58
9	50	6	4	5.91	5.87	3.75	0.83	0.55	-	0.69	-	-	-
10	50	6	8	5.88	5.16	2.60	-	0.84	-	0.35	-	1.37	-
11	50	12	4	2.09	1.19	-	-	-	-	-	-	-	1.19
12	50	12	8	3.35	1.20	-	-	-	0.47	-	-	-	0.73
13	control	-	-	10.54	-	-	-	-	-	-	-	-	-

cv. Suwan. Germinated root tips were treated with three concentrations of the herbicide, viz. 5, 25 and 50 ppm for 6 and 12 h durations with two recovery periods for 4 and 8 h along with control. The root-tips were fixed in 3:1 alcohol-acetic acid fixative and examined cytologically through routine acetocarmine squash preparations. Marked mitotic depression was duration- and concentration-dependent. Reversal of adverse effects of the herbicide was observed during recovery treatments even in treatments with higher doses for longer durations (Table 1).

Various cytological abnormalities with varying frequency, viz. fragmentation, despiralization, C-mitosis, laggards, etc. were observed in 6 h treatment only, while giant cells and pulverisation were observed in 12 h treatments only. Maximum frequency of C-mitosis (0.69%) and fragmentation of chromosomes (0.78%) was scored in 50 ppm treatments for 6 h and recovery of 4 h.

Effect of media on callusing and rhizogenesis from cultured root explants of genotype TUXP237-2

--Ashok Kumar, Harsh Kumar, S. K. T. Nasar and M. Kumar

The success of tissue culture experiments depends on selection of suitable media combinations. Thus, in order to select suitable media for callusing as well as for rhizogenesis, root explants from in vitro germinated seedlings of the genotype TUXP237-2 were inoculated in 15 different MS media combinations supplemented with various concentrations of 2,4-dichlorophenoxyacetic acid (2,4-D), indole butyric acid (IBA), indole 3-acetic acid (IAA) and kinetin (KN), either singly or in combinations of two. The responses were recorded after 45-50 days of culture. The best response with 2,4-D and IBA was at 5 mgl^{-1} and 2.5 mgl^{-1} respectively, with 2,4-D favouring more callusing and IBA favouring more rooting. Among the media with 2,4-D and IAA, the maximum response was observed at 2,4-D 5 mgl^{-1} and IAA 2 mgl^{-1}. Similarly, the best response with 2,4-D and IBA was with 5 mgl^{-1} and 2.5 mgl^{-1}. When 2,4-D was used with KN, the response was moderated and for this combination the maximum response was found when 2,4-D was used at 3 mgl^{-1} and KN at 2 mgl^{-1} (Table 1). Normally the medium favouring callusing also favoured rhizogenesis.

Table 1. Callusing and rhizogenesis from cultured root explants on different media.

Sl no.	Medium MS plus	Callusing % cultures	Callusing Growth	Rhizogenesis % cultures	Rhizogenesis No.
1	2,4-D 0.5 mgl^{-1}	55.55	++	27.77	2-4
2	2,4-D 2.5 mgl^{-1}	61.11	++	44.44	4-6
3	2,4-D 5.0 mgl^{-1}	72.22	+++	55.55	4-8
4	IBA 0.5 mgl^{-1}	55.55	++	66.66	8-10
5	IBA 2.5 mgl^{-1}	66.66	+++	94.44	15-20
6	IBA 5.0 mgl^{-1}	50.00	++	72.22	8-12
7	2,4-D 0.5 mgl^{-1} + IAA 0.5 mgl^{-1}	44.44	+++	44.44	2-6
8	2,4-D 2.0 mgl^{-1} + IAA 5.0 mgl^{-1}	66.66	+++	55.55	4-7
9	2,4-D 5.0 mgl^{-1} + IAA 2.0 mgl^{-1}	88.88	++++	66.66	5-10
10	2,4-D 0.5 mgl^{-1} + IBA 0.5 mgl^{-1}	55.55	++	55.55	2-6
11	2,4-D 2.5 mgl^{-1} + IBA 0.5 mgl^{-1}	55.55	+++	61.11	6-8
12	2,4-D 5.0 mgl^{-1} + IBA 2.5 mgl^{-1}	77.77	++++	72.22	6-12
13	2,4-D 0.5 mgl^{-1} + KN 0.5 mgl^{-1}	27.77	+	33.33	2-4
14	2,4-D 3.0 mgl^{-1} + KN 2.0 mgl^{-1}	33.33	++	38.88	2-6
15	2,4-D 5.0 mgl^{-1} + KN 5.0 mgl^{-1}	22.22	+	27.77	1-3

Callusing - + = low; ++ = moderate; +++ = high; ++++ = very high

Differential tissue culture response of seedling explants of cv. Swan

--Harsh Kumar and M. Kumar

Surface sterilized seeds of maize cv. Swan were germinated on Murashige and Skoog's (MS) basal medium. The seedlings were grown to 4-5 cm shoot length. Three seedling explants, namely about 1 cm primary root segments, about 0.3 cm seedling shoot segments taken 1 cm above the seedling node, and unrolled young leaves, were cultured to assess their response for callusing and rhizogenesis.

The MS basal medium supplemented with 2,4-D, IAA and kinetin (KN) in different concentrations, either singly or in combinations of two were used for callusing and rhizogenesis. Callus formation and subsequent rhizogenesis from all the three explants were best achieved on 2,4-D (5.0 mgl^{-1}) + IAA (2.0 mgl^{-1}). Among the three explants, root segments gave the best response, followed by young leaves. Shoot segments were the least responsive explants for both callus formation and subsequent rhizogenesis (Table 1). Among root and shoot segments, the basal portions were more responsive than the apical portions or explants. The younger the leaves, the better the response.

Table 1. Response of different seedling explants for callusing and subsequent rhizogenesis on MS + 2,4-D (5.0 mgl^{-1}) + IAA (2.0 mgl^{-1}).

Sl no.	Explants	Callusing % cultures	Callusing Growth	Rhizogenesis % cultures	Rhizogenesis No. culture
1	root	86.50	++++	65.07	6-12
2	shoot	10.34	+	2.75	2-4
3	leaf	23.52	++	11.76	2-5

Callusing - + = low; ++ = moderate, +++ = high; ++++ = very high

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Size and distribution of stomata in maize and its wild relatives

--G. Chandel, Rajesh Kumar and S. Katiyar

Stomatal characteristics have been realised as an asset to evolutionary studies. Stomatal size and distribution in twenty-six distinct collections of *Coix*, including both *Coix aquatica* (2n=10) and *Coix lacryma-jobi* (2n=20), four collections of *Chionachne koenigii*, three collections of teosinte, including *Zea diploperennis, Z. parviglumis, Z. luxurians* and three collections of maize (*Zea mays* L.) were used for observation.

Observations were recorded on size and number of stomata/unit area in all the members of the Maydeae. In general, *Chionachne* has the largest stomata with a mean stomatal size of 52.50 µm, followed by *Coix aquatica* (51.97 µm), *Coix lacryma-jobi* (48.00 µm), teosinte (46.75 µm) and maize (43.50 µm) (Table 1). Although *Coix aquatica* (2n=10) has a lower chromosome number, size of the stomata was found to be larger than that of *Coix lacryma-jobi* (2n=20). Mean number of stomata/unit area

Table 1. Mean stomatal size (µm) and number per unit area in maize and its wild relatives.

S.N.	Materials	Stomatal size Mean	Stomatal size Range	No. of stomata/unit area Mean	No. of stomata/unit area Range
1	Maize	43.50±0.00	37.50-45.00	136.00±0.00	133.00-139.00
2	Teosinte	46.75±1.03	45.00-52.50	131.83±12.9	120.00-171.00
3	*Coix aquatica*	51.97±1.30	37.50-60.00	134.01±4.75	102.00-194.00
4	*C. lacryma-jobi*	48.00±0.77	45.00-60.00	163.94±6.58	139.00-186.00
5	*Chionachne*	52.50±1.93	45.00-60.00	137.41±12.7	110.00-168.00

Maize - mean of four collections; teosinte - mean including *Zea diploperennis, Z. parviglumis* and *Z. luxurians*; *Coix aquatica* - mean of twenty collections; *C. lacryma-jobi* - mean of six collections; *Chionachne* - mean of four collections.

in Maydeae varied from 131.83 in teosinte to 163.94 in *Coix lacryma-jobi*. The size of the stomata in maize and teosinte was smaller than in the members of *Coix* and *Chionachne*. Number of stomata/unit area was also less in *Zea* than in *Coix* and *Chionachne*.

Pollen size variation in *Coix*

--G. Chandel, S. Katiyar, Rajesh Kumar and J. K. S. Sachan

Pollen size is one of the important characteristics to be used in evolutionary studies. Twenty-nine distinct collections of *Coix*, including both *Coix aquatica* (2n=10) and *C. lacryma-jobi* (2n=20), collected from different geographical regions of the Indian subcontinent, were taken for the present study. All the populations of *Coix* were field grown in two different climatic zones of India: (i) IGAU, Raipur and (ii) IARI, New Delhi.

In general, the pollen grains of *Coix* were round or oval in shape with a single pore. Although *Coix aquatica* has a lower chromosome number than *C. lacryma-jobi*, the mean pollen grain size of *C. aquatica* (87.26 and 60.00 µm) was found to be more than that of *C. lacryma-jobi* (63.05 and 35.83 µm) at IGAU, Raipur and IARI, New Delhi, respectively (Table 1.). Significant reduction in pollen

Table 1. Pollen grain size (in µm) in *Coix*

	Mean pollen grain size	
Collection	At IGAU	At IARI
(A) *Coix aquatica* (2n=10)		
Coix-20	105.00	68.00
Coix-24	69.00	54.00
Coix-25	93.00	65.00
Coix-27	73.50	49.00
Coix-28	67.50	46.00
Coix-29	72.00	48.00
Coix-30	75.00	46.00
Coix-36	96.00	64.00
Coix-44	114.00	70.00
Coix-48	105.00	68.00
Coix-55	87.00	58.00
Coix-56	102.00	68.00
Coix-57	105.00	75.00
Coix-59	82.50	61.00
Coix-62	75.00	50.00
Coix-63	90.00	60.00
Coix-64	90.00	65.00
Coix-65	82.50	58.00
Coix-66	90.00	62.00
Coix-67	90.00	60.00
Coix-68	75.00	50.00
Coix-70	90.00	70.00
Coix-71	78.00	65.00
MEAN	87.26	60.00
(B) *Coix lacryma-jobi* (2n=20)		
Coix-50	51.00	38.00
Coix-51	63.00	39.00
Coix-52	67.50	40.00
Coix-53	64.80	46.00
Coix-54	72.00	50.00
Coix-60	60.00	40.00
MEAN	63.03	35.83

size has been observed in all the populations of *Coix* studied. Various sized pollen grains have been recorded within and between different collections of both species of *Coix* studied.

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Linkage of RFLP markers to genes controlling resistance to southern corn rust

--J. B. Holland, D. V. Uhr and M. M. Goodman

Inheritance of resistance to southern corn rust (*Puccinia polysora*) in two maize populations was studied using RFLP markers. Two 100% tropical inbred lines (1416-1 and 1497-2) adapted to temperate environments had been identified as highly resistant to southern rust in a previous study (Uhr, Ph.D. thesis, North Carolina State University, 1991). Two populations segregating for resistance to southern rust were developed by crossing each resistant line to a susceptible hybrid, B73xMo17, and self-pollinating a single (F1) plant from each cross. 145 F2 plants from each population were scored for disease phenotype under natural infestation using a 1-9 scale (1=highly susceptible, 9=highly resistant). Each plant was also selfed to produce F2 families in the F3 generation. The F2 families were then scored for disease phenotype under natural infestation in a replicated field trial using natural inoculum.

Based on these phenotypic scores, the most resistant and most suceptible families in each population were chosen for genotypic evaluation. Specifically, 32 resistant and 13 susceptible families were chosen from the 1416-1 population, and 33 resistant and 20 susceptible families were chosen from the 1497-2 population. Leaf tissue from ten plants of each family was bulked to isolate genomic DNA representing each family. RFLP probes marking regions of the genome known to contain rust resistance loci (either *Rpp* or *Rp* genes) were used to determine genotypes of the families. Single factor ANOVA's were used to determine which markers were linked to chromosomal regions that significantly affected disease resistance (Table 1).

Table 1. Additive (a) and dominance (d) effects and coefficients of determination (R^2) of RFLP markers on southern rust resistance. (Note that negative a effects indicate B73xMo17-contributed resistance allele.)

		1416-1 population			1497-2 population		
Chromosome	Marker	a	d	R^2	a	d	R^2
3	*umc10*	ns	ns	-	nd	nd	nd
3	*umc161*	ns	ns	-	-0.38**	1.30**	0.08
3	*umc102*	nd	nd	nd	-1.24**	-0.73*	0.06
3	*umc26*	0.74*	-1.04*	0.11	-1.48**	-0.37*	0.11
4	*bnl5.46*	-0.24**	0.36**	0.01	-1.13**	ns	0.09
10	*bnl10.17*	1.93**	1.03**	0.52	nd	nd	nd
10	*bnl3.04*	1.94**	ns	0.57	nd	nd	nd
10	*npi285*	1.98**	0.64**	0.57	2.11**	ns	0.41
10	*umc130*	1.54*	ns	0.27	1.75**	ns	0.22

*,** - significant at the p=0.05, p=0.01 levels, respectively.
ns - not significantly different from 0 at p=0.05.
nd - no data available yet.

Markers on the short arm of chromosome 10 showed the largest effects in both populations, indicating that each resistant inbred parent carries a major gene or genes in this region. It is possible that the resistance could be caused by alleles of *Rpp9* on chromosome 10. Genes with smaller effects on resistance to southern rust also appear to be located on chromosomes 3 and 4. Although major common rust (*P. sorghi*) resistance genes are known to be located in these genomic regions, to our knowledge this is the first report indicating that southern rust resistance genes exist on these chromosomes.

ST. PAUL, MINNESOTA
University of Minnesota

***Teosinte glume architecture1* controls silica deposition in the glumes of maize**

--Jane E. Dorweiler and John Doebley

Teosinte glume architecture1 (*tga1*) has several effects on the glumes of maize, with the homozygous teosinte allele (*tga1/tga1*)

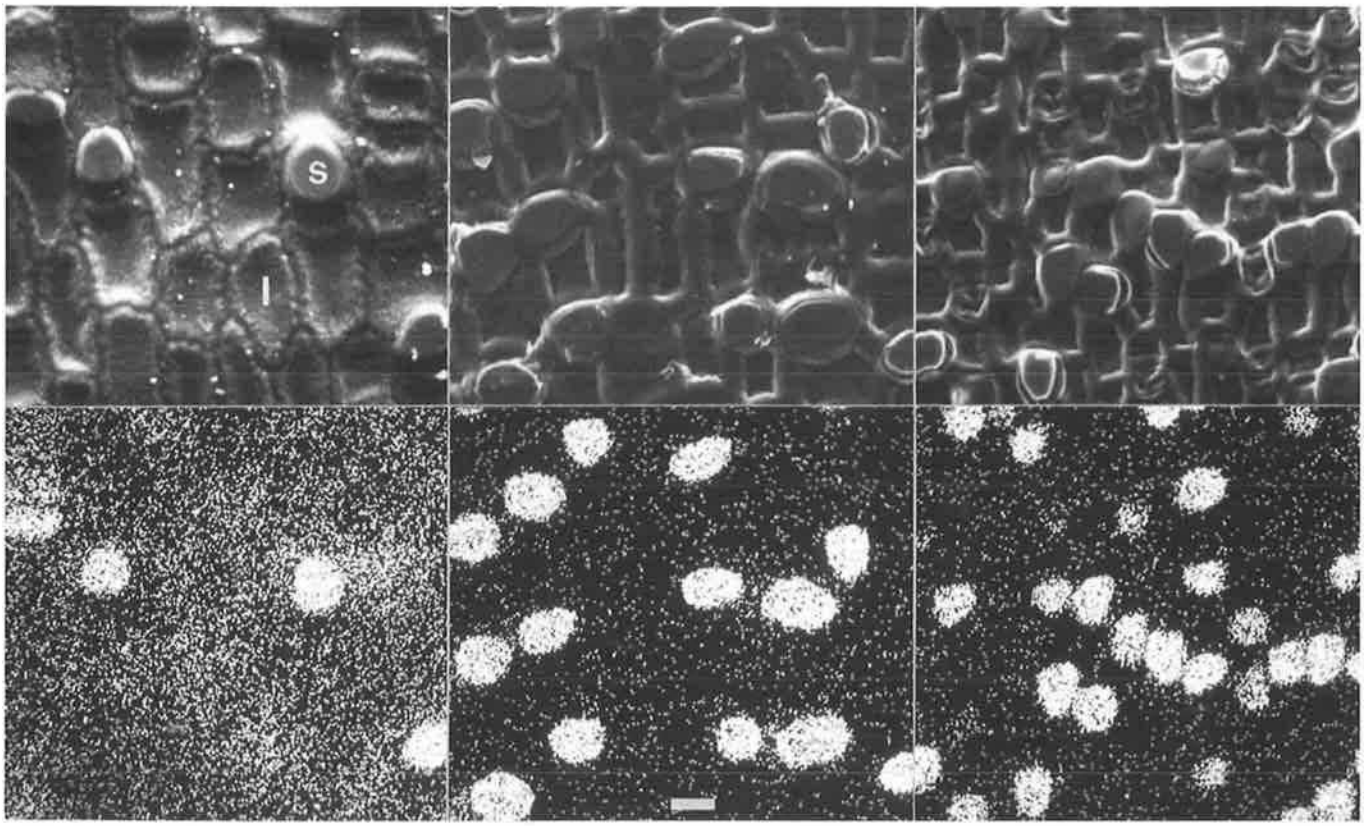

Figure 1. SEM micrographs of glumes of the three genotypes at *tga1* (top row) and their corresponding silica dot maps (bottom row). In each SEM micrograph, silica cells (s) are visible. The corresponding dot maps show a high density of silica X-rays (dots) mapping to these silica cells. The teosinte allele homozygotes (*tga1/tga1*) at left also have a high number of silica X-rays mapping to the long epidermal cells (l). Bar represents 15 μm for all six photos.

making the glumes more teosinte-like (Dorweiler et al., Science 262:233-235, 1993). One of these effects is to make the glumes of maize more highly indurated (harder).

In an effort to understand the developmental basis for this difference, we have looked at silica deposition in the glumes representing the three genotypes at *tga1* in the W22 background. We used X-ray microanalysis to investigate silica deposition. We found that the standard W22 line (*Tga1/Tga1*) has high concentrations of silica in the short cells of the glumes, but that the long cells have virtually no deposition of silica. The heterozygotes (*Tga1/tga1*) have a similar silica distribution to *Tga1/Tga1* homozygotes. The teosinte allele homozygotes (*tga1/tga1*), though they still have high silica deposition in their short cells, have additional silica deposited in the long cells of the glumes (Figure 1).

The amount of silica in the long and short cells was also quantified using X-ray microanalysis. The amount of silica in the short cells of all three genotypes is essentially the same. The amount of silica in the long cells of the homozygous teosinte allele at *tga1* is nearly 40x the amount of silica in the long cells of both the maize homozygote and the heterozygote (Figure 2).

Thus, in addition to the effects previously noted for *teosinte glume architecture1* (Dorweiler et al., Science 262:233-235, 1993), this locus seems to have an effect on the distribution of silica in the abaxial epidermal cells of the glumes. This phenomenon appears to at least partially explain the induration differences between the maize and teosinte alleles at *tga1*, and thus the differences in glume induration between maize and teosinte.

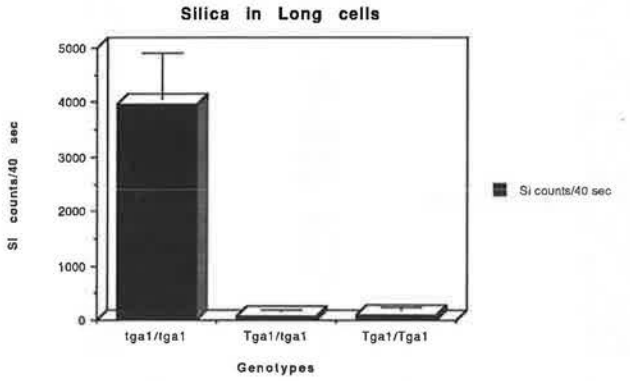

Figure 2. Silica in long cells for each genotype. This graph shows the amount of silica in long cells for the abaxial epidermis of glumes from the three genotypes at *tga1*. The y-axis represents the average number of silica (Si) X-rays emitted from long cells during a 40 second interval. Averages calculated from three 40 second readings on 3 cells for each genotype.

Acknowledgments: Special thanks to Dr. Anne Sylvester for suggesting the analysis of silica deposition and teaching one of us (JED) this technique.

***Suppressor of sessile spikelets1* (*Sos1*): a dominant mutant affecting inflorescence development**

--John Doebley, Beth Kent and Adrian Stec

The spikelets of the grass tribe Andropogoneae, to which maize belongs, are borne in pairs, one sessile and one pedicellate. This paired arrangement is also found in maize ears, although the highly compact nature of the maize ear makes it difficult, but not impossible, to distinguish the sessile and pedicellate spikelets since both are essentially sessile (H. C. Cutler, Bot. Mus. Leaflet, Harvard Univ. 12:257-291). We describe a dominant mutant that suppresses the formation of sessile spikelets in maize ears. Seeds of the mutant were taken from an ear showing the mutant phenotype in the University of Wisconsin Herbarium. According to Dr. Hugh Iltis (Director of the Herbarium), he obtained the ear from Dr. John Lonnquist who reported to him that the mutant arose spontaneously in a maize population.

Seeds originally obtained from the herbarium specimen were selfed for several generations until a line (91-31) that was true-breeding for the single spikelet trait was obtained. This line also had a poor tassel with few short branches. Subsequently, 91-31 was crossed to W22 and W22-TGA (a W22 derivative carrying a segment of teosinte chromosome 4 and the teosinte allele at *tga1* within this segment). The F1's of both crosses were grown and found to exhibit the single spikelet trait, indicating that this trait was dominant to the normal maize condition of paired spikelets. The 91-31xW22 F1 was backcrossed to W22, and the 91-31xW22-TGA F1 was selfed to produce an F2 population.

During the summer of 1992, we analyzed the 91-31xW22 backcross population. This was one of the coldest summers on record in Minnesota and plant growth was poor. Among the 58 progeny analyzed, 31 exhibited paired spikelets and normal tassels. The remaining 27 plants were barren (without ears) and had tassels that consisted only of a central spike without any branches. The 31:27 ratio suggested a 1:1 ratio expected if a single locus controlled the differences in tassel structure and barrenness. The absence of the single spikelet trait among those plants that had ears prevented us from scoring this trait. One possibility we considered was that the cold weather had induced the barrenness in plants carrying the factor that causes the single spikelet trait and that this factor also affects the production of tassel branches. Working on this assumption, we analyzed the 58 plants for RFLP markers, using one marker per chromosome arm. We detected linkage between an RFLP marker and barrenness/unbranched tassels on chromosome arm 4S. After mapping additional RFLPs on this chromosome arm, we mapped these traits between *php20725* and *bnl5.46*.

php20725 - 2.6 - barrenness/unbranched tassels - 4.4 - *bnl5.46*

Distances are the recombination fractions.

In the summer of 1993, we again attempted mapping the single spikelet trait using the 91-31xW22-TGA F2 population. Plant growth this year was better and, in a population of 58 F2 plants, we observed 46 with single spikelets and 12 with paired spikelets in the ear. These numbers do not differ from the expected 3:1 ratio if the single spikelet trait is controlled by a single dominant locus ($X^2 = 0.32$, d.f = 1, $p > 0.5$). We designate this locus *Suppressor of sessile spikelets1* (*Sos1*). RFLP analysis demonstrated that *Sos1* maps between *php20725* and *bnl5.46*.

php20725 - 3.7 - *Sos1* - 9.6 - *bnl5.46*

Because the single spikelet trait mapped to the same location as barrenness/unbranched tassels in the 1992 backcross population and because all these traits behaved as dominants, we infer that *Sos1* alone controls these traits and that *Sos1* plants have a tendency toward barrenness under poor growth conditions. We should also note that the introgressed teosinte chromosome segment in W22-TGA does not extend between *php200725* and *bnl5.46*, and, thus, *Sos1-Ref* was segregating with *sos1+W22* in 91-31xW22-TGA F2 population.

Figure 1 shows an ear carrying *Sos1-Ref*. The absence of the sessile spikelets leaves a gap between the rows of pedicellate spikelets. We were able to confirm that it is the sessile spikelet that is suppressed because this spikelet arises as a branch of the primordium that forms the pedicellate spikelet. Examination of ear primordia of *Sos1-Ref* maize revealed that this branch is not formed. *Sos1-Ref* also affects the formation of tassel branches, formation of sessile spikelets in the tassel, and the number of rows of cupules in the ear. The wild type function of *sos1* may be in some aspect of the formation of inflorescence primordia, such as governing the number of cells committed to each branch primordium.

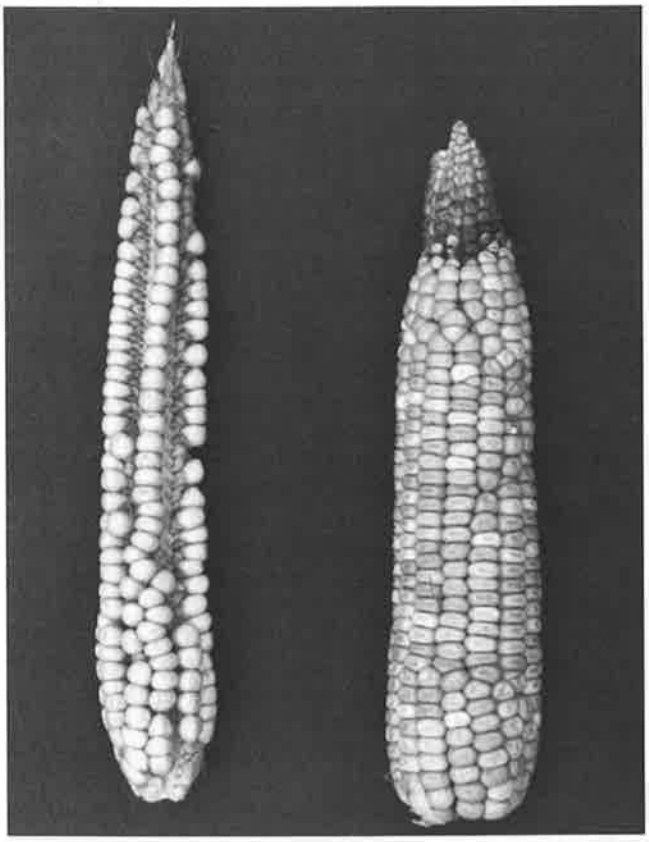

Figure 1. Ears of *Sos1-Ref* and *sos1+W22* maize.

Our interest in *Sos1* arose because the single spikelet trait of this mutant seems to resemble the single spikelets of teosinte ears, the probable ancestor of maize. Thus, *Sos1* is a candidate for a gene involved in the evolution of maize from teosinte. Three observations suggest that this is not the case. (1) In teosinte ears, it is the pedicellate and not the sessile spikelet that is lacking. (2) In teosinte ears, both the pedicellate and sessile spikelet primordia are formed, but then the pedicellate spikelet is aborted. With *Sos1*, only a single spikelet primordium is formed. (3) In our

QTL (quantitative trait locus) mapping studies (Doebley and Stec, Genetics 134:559-570, 1993), the QTLs controlling the difference of paired versus single spikelets between maize and teosinte does not map to this region of chromosome arm 4S. We are currently testing whether the *sos1* allele of teosinte is equivalent to the maize wild type allele (*sos1+maize*) by examining an F2 population derived from a teosinte x *Sos1-Ref* maize cross. If the teosinte allele is functionally the same as *sos1+maize*, then we should recover plants with paired spikelets in the F2 population.

Teosinte branched1 and the origin of maize

--John Doebley and Adrian Stec

Teosinte branched1 (*tb1*) is a recessive mutant of maize that affects plant architecture and maps to chromosome arm 1L. Mutant plants (*tb1-ref*) have long lateral branches tipped by tassels at some upper nodes of the main culm and tillers at the basal nodes. We will refer to this syndrome as **teosinte branched plant architecture**. This contrasts with **normal maize plant architecture** conferred by the dominant maize allele (*Tb1*): short lateral branches tipped by ears at some upper nodes and few or no tillers at the basal nodes. Since both tillers (basal lateral branches) and upper lateral branches arise from axillary meristems, in a general sense, *tb1* controls the fate of the axillary meristems, although with different effects depending on the position (basal or upper) of the axillary meristem within the plant.

The architecture of *tb1-ref* maize plants resembles that of the nearest wild relatives of maize, the annual teosintes. Like *tb1-ref* plants, annual teosinte grown in its native habitat in Latin America "normally" produces long lateral branches tipped by tassels at upper nodes of the main culm. We say "normally" because annual teosinte plants in Latin America may produce short lateral branches tipped by normal teosinte ears or mixed male-female inflorescences in some environments such as dry shallow soils or low light (shading). Also like *tb1-ref* plants, annual teosinte can tiller profusely. Tillering in teosinte is extreme when the plants are grown at temperate latitudes, apparently in response to the long days of these regions which may prolong the juvenile phase of development during which tillers are formed. Tillering is uncommon for teosinte in Latin America, perhaps because, under the short Latin American days, the plants begin adult development earlier, restricting the opportunity to produce tillers. Thus, while teosinte resembles *tb1-ref* maize, the extent and nature of the resemblance is dependent on the environment in which the teosinte plants are grown. This situation suggests that the developmental pathway controlling plant architecture in teosinte is responsive to environmental signals, most likely in a way that best adapts the plant to the local environment.

Evidence from our QTL (quantitative trait locus) mapping studies (Doebley and Stec, Genetics 134:559-570, 1993) demonstrated that a QTL on chromosome arm 1L largely controls the differences in plant architecture between maize and teosinte. This QTL is very near (within 10 map units) to the location of *tb1*. For this reason, we proposed that *tb1* is our QTL and that *tb1* was largely responsible for the evolution of normal maize plant architecture from the ancestral teosinte plant architecture. To test this hypothesis, we performed a simple complementation test. First, we transferred the region of teosinte chromosome arm 1L encompassing *tb1* and our QTL into maize inbred W22 by four generations of backcrossing using molecular markers to retain the teosinte segment of 1L. No phenotypic selection was exercised. A fourth generation backcross plant heterozygous for the teosinte chromosome segment was used to pollinate a maize plant carrying *Tb1/tb1-ref* (seed obtained from Charles Burnham). We considered two possible outcomes. (1) Our QTL is not allelic to *tb1* in which case all plants should have normal maize plant architecture. (2) Our QTL is allelic to *tb1* in which case one-fourth of the plants should have teosinte branched plant architecture. These expectations arise because among the F1's there are four genotypic classes, only one of which should give teosinte branched plant architecture:

Genotypes	Expected Phenotypes
Tb1 / Tb1+W22	normal maize
Tb1 / tb1-teosinte	normal maize
tb1-ref / Tb1+W22	normal maize
tb1-ref / tb1-teosinte	teosinte branched

The expectation that one-fourth of the F1 progeny should have the teosinte branched phenotype assumes that *tb1-teosinte* will behave as a recessive. This assumption is based on our observation that the BC1, BC2 and other backcross generations all exhibited normal maize plant architecture despite the fact that they were heterozygous (*Tb1+W22/tb1-teosinte*).

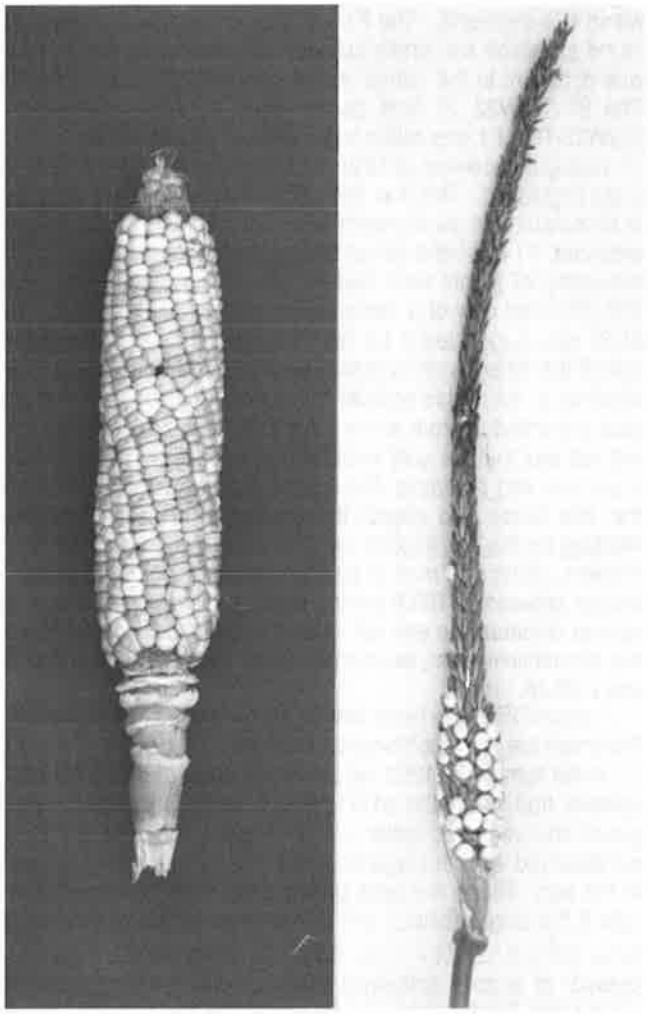

Figure 1. Inflorescences terminating the primary lateral branch from the complementation test discussed in the text: a female inflorescence (ear) showing the normal maize phenotype of the wild type maize *Tb1* allele (left); and a mixed male-female inflorescence showing the phenotype of a *tb1-ref / tb1-teosinte* plant (right).

Seventy-two F1's from the cross were classified with 57 having normal maize and 15 having a weak teosinte branched plant architecture. Plants with weak teosinte branched plant architecture had elongated lateral branches tipped by mixed male-female inflorescences (Fig. 1). The ratio of 57:15 does not differ significantly (X^2 = 0.67, 1 d.f., p > 0.25) from the 3:1 ratio expected if our QTL is an allele of *tb1*. Although none of the progeny had a strong teosinte branched phenotype, since one-fourth did show a weak teosinte branched phenotype, we conclude that our QTL is allelic to *tb1* but that our QTL represents a weak allele (*tb1-teosinte*) relative to the maize mutant (*tb1-ref*).

A model for *teosinte branched1*. We propose the following model for the function of *tb1* and how it changed during the evolution of maize. In teosinte, *tb1* encodes a repressor of the elongation of the lateral branches or of the development of axillary meristems. In good environmental conditions and full sunlight, this locus is not expressed in teosinte and thus the axillary meristems develop into basal tillers or upper lateral branches tipped by tassels. In poor environmental conditions (low moisture, shallow soil) and/or shading, *tb1* is expressed in teosinte and it directly or indirectly represses the development of the axillary meristems such that few (or no) basal tillers and only short upper lateral branches tipped by ears are produced. Thus, *tb1* is a locus whose original function was in adapting the teosinte plant to its local environmental situation by altering plant architecture. To explain the evolution of maize plant architecture, we propose that the expression of *tb1* is no longer tied to an environmental signal but rather that *tb1* in maize is constitutively expressed during the early development of the axillary meristems, keeping both tillering and full elongation of the upper lateral branches repressed. Under this model, both the *tb1-teosinte* and *Tb1+maize* alleles would encode functional products, although ones that are differently regulated. *tb1-teosinte* is recessive to *Tb1+maize* because the latter will produce the repressor whether or not the former allele is activated by an environmental signal. Finally, under this model, the maize mutant (*tb1-ref*) can be explained as a recessive loss of function allele. With complete loss of the repressor function, the axillary meristems of homozygous *tb1-ref* plants elongate to produce either basal tillers or elongate upper lateral branches tipped by tassels.

Photos of teosinte glume architecture1

-- Jane Dorweiler and John Doebley

In our recent paper on *Teosinte glume architecture1* (*tga1*) (Dorweiler et al., Science 262:233-235, 1993), the reproduction of Figures 1, 3, 4 and 5 was not what we hoped because the printer reduced some figures more than desirable and printed others from polaroids sent for the reviewers rather than from the originals. These figures are reproduced here to more clearly show the effects of *tga1* on ear and glume morphology.

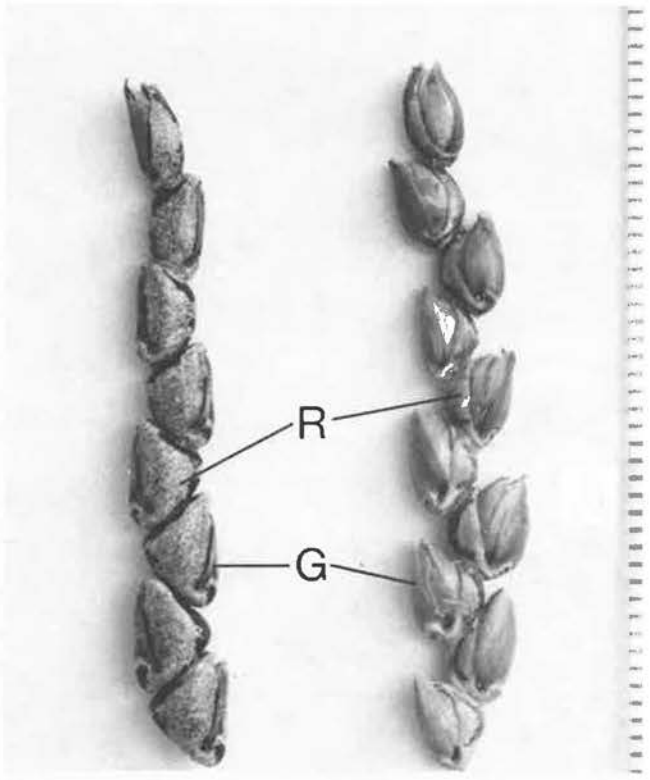

Figure 1. Ear of pure teosinte (left) composed of eight cupulate-fruitcases and an ear of teosinte homozygous for the maize allele at *tga1* (right). The rachids of teosinte (R) are fully developed, forming a deep invagination in which the kernels are housed. The glume (G) seals the opening of the invagination so that the kernel is completely hidden and protected. The rachids of teosinte with maize allele at *tga1* are less developed, forming only a short, shallow invagination that does not fully encase the kernel. Scale in mm.

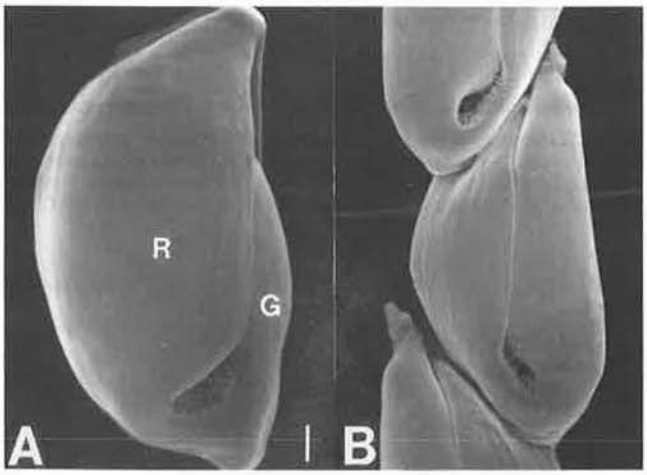

Figure 3. Scanning electron micrographs of single fruitcases of teosinte (**A**) and of teosinte homozygous for the maize allele at *tga1* (**B**), both collected just prior to pollination. The maize allele makes the rachid (R) shorter and narrower, exposing more of the outer glume (G). In teosinte, the glume arches into the cupule, while the maize allele causes the glume to be oriented directly upward at this stage. Bar represents 0.5 mm.

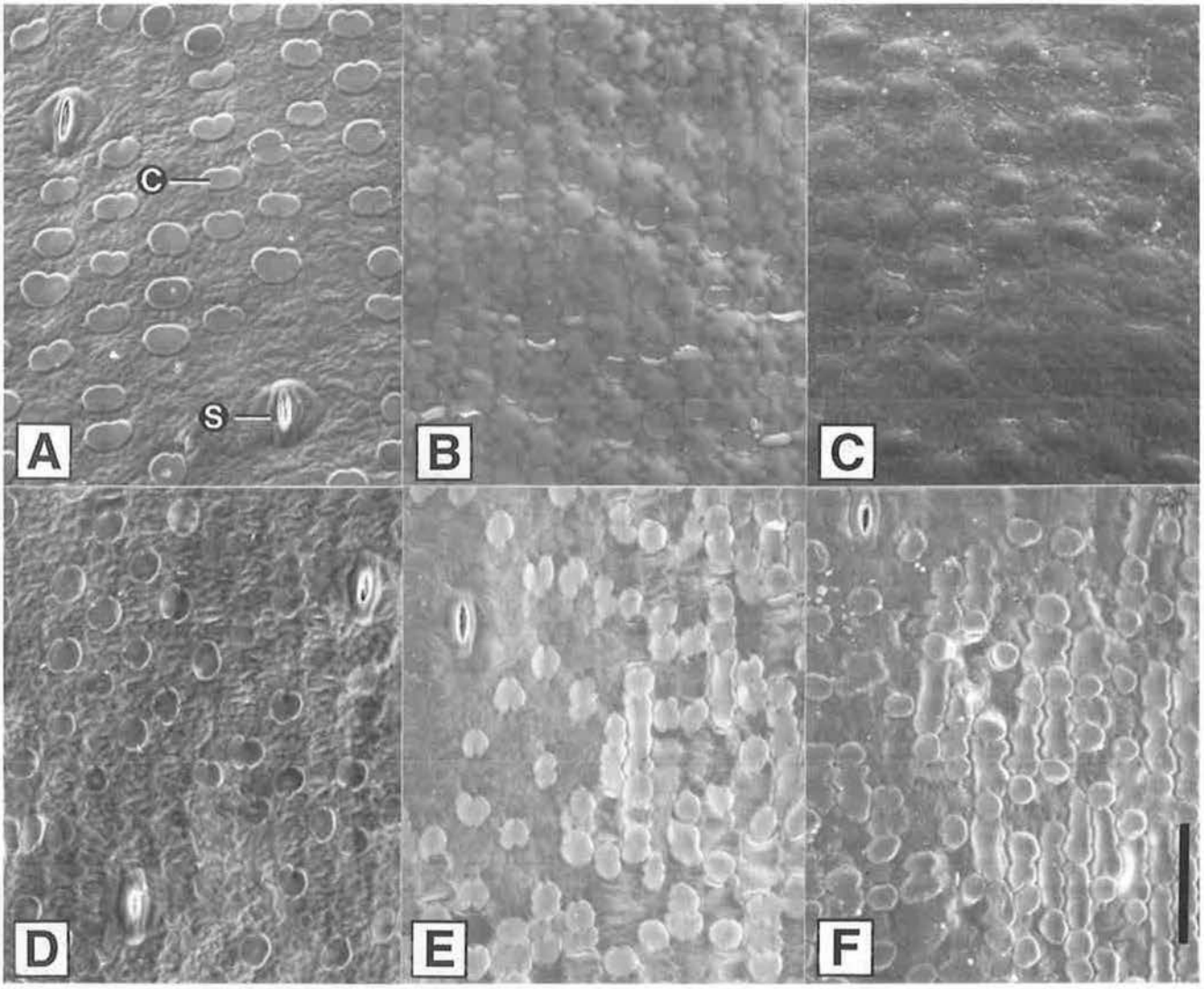

Figure 4. Scanning electron micrographs of the surface of the outer glume of the ear of teosinte (**A**, **B**, **C**) and of teosinte homozygous for the maize allele at *tga1* (**D**, **E**, **F**). Glume surfaces just prior to pollination (**A**, **D**), and approximately two (**B**, **E**) and five (**C**, **F**) weeks post-pollination are shown. In teosinte, the short cells (c) are arranged in distinct rows (**A**) and the glume epidermis forms a smooth regular surface as it matures (**B**, **C**). The maize allele at *tga1* appears to alter both the arrangement of short cells and the formation of a smooth regular surface at maturity (**F**). Stomata (s) are visible before pollination in teosinte but are obscured as the glume matures (**B**, **C**). With the maize allele at *tga1*, the stomata remain visible even in the mature glume (**F**). Bar represents 50 microns.

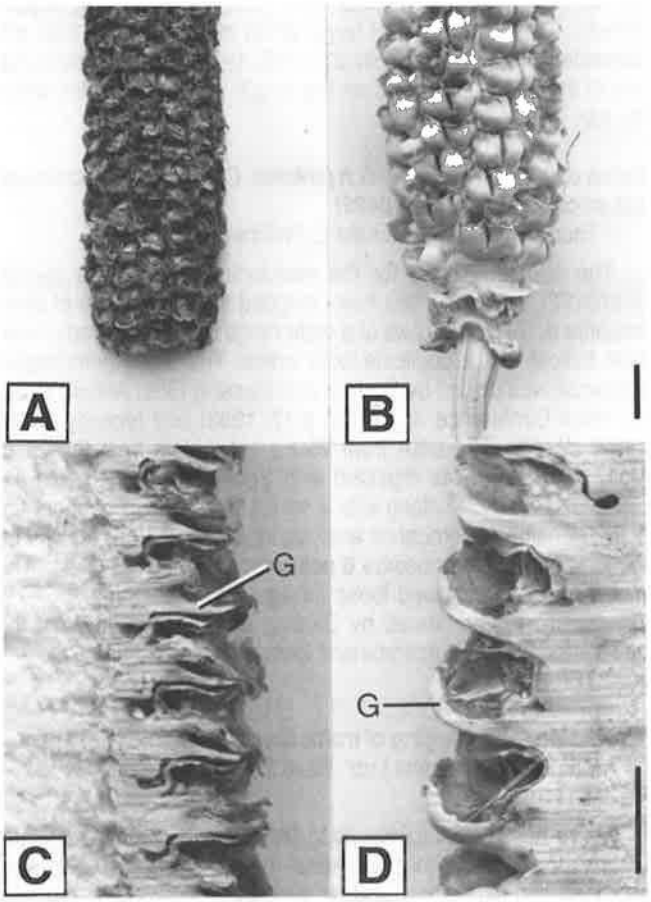

Figure 5. Mature ears (without kernels) of maize line W22 homozygous for the maize (**A**, **C**) and teosinte alleles (**B**, **D**) at *tga1*. With the maize allele (**A**), the relatively small outer glumes are not visible, being obscured by the red pigmented bracts (paleas and lemmas). With the teosinte allele (**B**), the paleas and lemmas are obscured by the enlarged, unpigmented outer glumes. Longitudinal cross-sections show that W22 with the maize allele at *tga1* has outer glumes (G) that are thin and perpendicular to the axis of the ear (**C**), while those of W22 with the teosinte allele at *tga1* are thicker and curved upward (**D**). The black bar in **B** represents 1 cm and applies to both **A** and **B**; the black bar in **D** represents 5 mm and applies to both **C** and **D**.

Terminal ear1 and the origin of maize

-- John Doebley

QTL (quantitative trait locus) mapping identified the region between *umc18* and *umc60* on chromosome arm 3L as affecting several aspects of inflorescence structure that distinguish maize and teosinte (Doebley and Stec, Genetics 134:559-570, 1993). These include (1) the extent to which inflorescences terminating the primary lateral branches are male or female, (2) inflorescence phyllotaxy, (3) the length of the internodes in the primary lateral branches, (4) the frequency of paired versus single spikelets in the female inflorescence, and (5) the number of cupules (internodes) within the inflorescence. One possibility is that all of these differences represent the pleiotropic effects of a single locus located within this segment. Previously, I proposed that this locus might be *terminal ear1* (*te1*) (Doebley, Cell. Biochem., Supl. 17B, p. 5,1993). In this note, I explain this hypothesis and present new evidence consistent with it.

A model for *terminal ear1* in maize evolution. *Terminal ear1* plays a role in internode elongation in the vegetative culm. Mutant *te1-ref* plants have shorter internodes in the vegetative culm of the plant and often some female spikelets in the tassel. The internodes just below the tassel are the most severely affected relative to wild type. The model presented here presumes that, in teosinte, *te1* plays a role in internode elongation in both the vegetative culm and in the inflorescences. Then, during the evolution of maize, *te1* was altered such that the maize allele (*Te1-Maize*) produces shorter internodes in the inflorescences and the primary lateral branch (or ear shank) than does the teosinte (progenitor) allele (*Te1-Teosinte*). One mechanism by which this could be accomplished is if *Te1-Maize* has a lower level of expression in the inflorescence than does *Te1-Teosinte*. Shorter internodes in the ear and ear shank would have pleiotropic effects on phyllotaxy, inflorescence sex, and single versus paired spikelets. Also, if the internodes are shorter in the ear, then it may be possible to produce a larger number of internodes. Under this model, both the maize and teosinte alleles of *te1* would produce functional products. Finally, since *te1-ref* represents a loss of function mutation, it may also cause shorter internodes in the ear shank and the inflorescence. These effects may be difficult to detect because internodes in the inflorescences and ear shank are already very short in most types of modern maize.

New evidence. Two new pieces of information are consistent with the proposal that *te1* is our QTL. First, I scored internode length in the ear as a quantitative trait in a maize-teosinte F2 population. The ears of the F2 plants showed a range of variation from those in which all internodes were relatively long and of equal length (like teosinte) to others with alternating short and long internodes (Fig. 1). The later condition is both more maize-like and

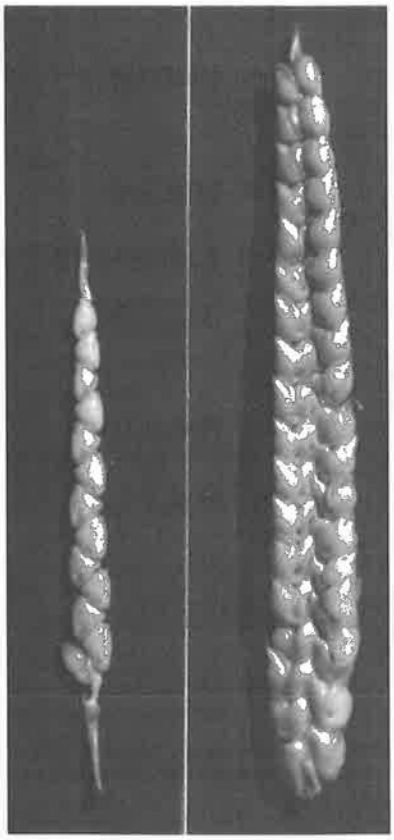

Figure 1. Ears from a maize-teosinte F2 population showing variation in the length of internodes within the ear: teosinte-like ear with internodes that are long and of relatively equal lengths so that the cupulate fruitcases appear to be stacked one on top of the other (left), and more maize-like ear with alternating short and long internodes so that the cupulate fruitcases appear side-by-side (right).

reminiscent of the effect that *te1* can have on the vegetative culm. Interval mapping revealed seven QTLs affecting internode length in the ear (Table 1). The QTL with the largest effect accounts for 47% of the variation (a very large effect) and maps to chromosome arm 3L where *te1* is located. Substitution of two maize alleles at this QTL for two teosinte alleles reduces the average length of the internodes in the ear from 4.5 to 3.0 millimeters.

Table 1. Location and effects of quantitative trait loci affecting internode length in the ear based on an analysis of 290 F2 plants derived from a maize (race Reventador) by teosinte (ssp. *parviglumis*) cross. This population previously described by Doebley and Stec (Genetics 134:559-570, 1993).

Marker Locus	Chromosome	LOD-score	R-squared	Candidate
umc157	1S	6.06	12.1	
umc107	1L	2.63	4.4	*tb1*
umc5a	2L	2.42	4.1	
umc60	3L	19.19	47.0	*te1*
umc42	4S	10.68	26.2	*tga1*
umc27	5S	2.47	4.6	
umc110a	7L	5.71	12.0	

A second piece of supporting evidence was possible because *te1* has recently been molecularly cloned (Bruce Veit, pers. comm.). Using a clone of *te1* (generously provided by B. Veit and S. Hake), I was able to map *te1* relative to our QTL. *te1* is located in the interval between *umc18* and *umc60* where our QTL for inflorescence internode length maps (Fig. 2). These new data make *te1* a strong candidate for our QTL. The recent cloning of *te1* makes it possible to test definitively both the hypothesis that our QTL is *te1* as well as the specific predictions of the model outlined above.

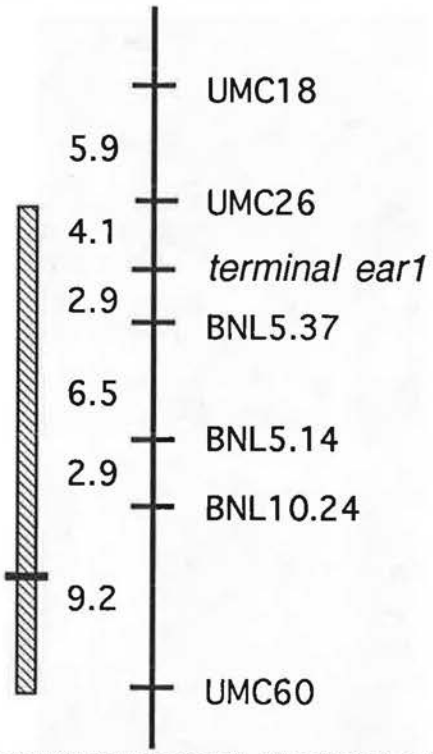

Figure 2. Map of a portion of chromosome arm 3L. Hatched rectangle on the left represents the 2-LOD support interval for the position of the QTL affecting internode length in the ear.

Finally, it is interesting that two of the other QTLs, affecting internode length in the ear, map near other genes involved in the evolution of maize (Table 1). There is a QTL of small effect on 1L near *tb1*. Our analysis of *tb1* indicates that it primarily affects internode length in the ear shank but that it also has some effect on internode length in the ear itself (Doebley and Stec, unpublished). A second QTL of large effect maps near *tga1* on 4S. Dorweiler et al. (Science 262:233-235, 1993) have reported that one of the effects of *tga1* is on the length of the internodes within the ear.

Genes encoding methionine-rich proteins: Chromosomal location of a duplicate locus of *zps10/(22)*

--Todd L. Krone and Ronald L. Phillips

The structural gene for the methionine-rich 10 kDa zein-2, *zps10/(22)*, previously has been mapped to the long arm of chromosome 9. Southern blots of a wide range of maize inbreds, however, indicate that a duplicate locus exists. This highly homologous sequence was cloned by Swarup and Messing (35th Annual Maize Genetics Conference Abstracts, p.12, 1993) and recently designated *dzs23*. The DNA from 25 F2 individuals from the cross Mo17 x BSSS53 was digested with *Eco*RI and probed with the clone for *zps10/(22)* along with a series of probes throughout the maize genome. Segregation analysis indicates *dzs23* is located on the long arm of chromosome 6 near *umc21*, 4 ± 3 cM distal. The *dzs23* gene also showed loose linkage with *umc65* (24 ± 6 cM). This position is confirmed by Swarup et al. in this issue of the newsletter using a recombinant inbred line population and the *dzs23* probe.

Identification and mapping of maize acetyl-CoA carboxylase genes

--Margaret Egli, Sheila Lutz, Dave Somers and Burle Gengenbach

Maize acetyl-CoA carboxylase (ACCase) is a multifunctional, biotinylated protein that provides malonyl-CoA for fatty acid synthesis and elongation, and for synthesis of secondary metabolites. Fatty acid synthesis is proportional to and rate-limited by ACCase activity in seeds and leaves. Knowledge of how ACCase is regulated may be useful for increasing kernel oil content, especially in combination with the ability to alter relative amounts of specific fatty acid components.

Most ACCase activity in maize leaves and in developing endosperm tissue and embryos is encoded by the *Acc1* gene (Somers et al., Plant Physiol. 101:1097, 1993). *Acc1* is a semi-dominant nuclear gene, mutant forms of which confer tolerance to certain herbicides (Marshall et al., Theor. Appl. Genet. 83:435, 1992). Both wildtype and mutant *Acc1* maize leaves contain two forms of ACCase activity (I and II) that differ in charge, size, cellular location, immunoreactivity with ACCase I antiserum, and herbicide inhibition. The predominant form, ACCase I, is plastid-localized and its inhibition by herbicides is altered by mutations in the *Acc1* gene (Egli et al., Plant Physiol. 101:499, 1993; Egli et al., MNL 66:94, 1992).

Antiserum to SDS-denatured ACCase I (Egli et al., 1993) was used to screen a λ-gt11 cDNA expression library derived from oligo-dT-primed A188 seedling mRNA. Seven incomplete cDNAs of 1.2 to 5.44 kb were obtained (type A cDNA); partial sequencing and restriction mapping indicated they were identical and were significantly similar to known ACCases. A 2 kb *Eco*RI subclone of ACCase type A cDNA hybridized to an 8.25 kb mRNA, which is large enough to encode the 227 kD ACCase I polypeptide plus an expected plastid transit peptide (about 6.3 kb).

A W22 genomic library (Clontech) was probed with the 2 kb *Eco*RI ACCase type A cDNA subclone to obtain additional 5' cod-

ing sequence. Two different 15 kb genomic clones (types A and B) were identified. A 2.7 kb coding sequence from genomic clone type A was 100% identical to type A ACCase cDNA; the genomic clone also extends 5' from the cDNA and should contain the remaining coding sequence plus the promoter. A partial sequence (750 nt) of the type B genomic clone was 96% identical to ACCase type A cDNA. The genomic type B clone extends 3' from the gene and may lack the 5' end. The cDNA library was rescreened and a low frequency of clones corresponding to type B were obtained. We are continuing to sequence type A and B cDNA and genomic clones to obtain gene-specific probes that may be useful for mapping and for mRNA expression analyses.

PCR primers derived from the 5' end of type A cDNA clones and from genomic type A coding sequence were used to amplify A188 cDNA. Sequence analysis of these PCR clones is in progress. Currently available cDNA clones cover 87% of the expected ACCase coding sequence. Additional 5' coding sequence will be obtained from RACE-PCR products and genomic clone A.

Peptide sequence comparisons to yeast, rat and chicken ACCase indicate they are colinear with maize ACCase cDNA. High sequence identities in the biotin carboxylase (> 33% for 57 aa), biotin binding (48% for 40 aa), and transcarboxylase domains (51% for 602 aa) were observed. The typical eukaryotic ACCase biotin binding site (VMKM) was located approximately 4.36 kb 5' of the C-terminus of the maize ACCase type A coding sequence.

Recombinant inbred maize lines from Tx303 x CO159 were used for mapping. Blots of *Hin*dIII-digested parent and progeny DNA were hybridized with a 1.2 kb *Eco*RI type A cDNA subclone that contained about 50% non-coding 3' sequence. The location of the only polymorphic band was mapped (Mapmaker, Kosambi mapping function) to chromosome 2S, between *umc131* (4.6 cM) and *umc2b* (10.4 cM). Two other monomorphic bands were also present and could not be mapped in this population. We are currently investigating whether herbicide tolerance conferred by the mutant *Acc1* gene cosegregates with RFLPs identified with ACCase cDNA probes.

Threonine-overproducing, lysine-insensitive aspartate kinase (*Ask2*) map location

--Gary J. Muehlbauer, Burle G. Gengenbach and David A. Somers

Ask2 encodes a threonine-overproducing, lysine-insensitive aspartate kinase mutant (Dotson et al., Planta 182:546-552, 1990). The *Ask1* threonine-overproducing trait was previously mapped to chromosome 7S (Azevedo et al., Plant Sci, 70:81-90, 1990) and shown to be nonallelic to *Ask2* (Diedrick et al., Theor. Appl. Genet. 79:209-215, 1990). To locate the *Ask2* threonine-overproducing trait in the maize genome, a cross was made to wildtype B73 and F3 lines were derived. Free threonine concentrations were measured in the bulked samples of F3 kernels to determine the *Ask2* genotype of the segregating F2 plants. DNA blot analysis was conducted on bulked samples of genomic DNA from 16 F3 *Ask2* lines. These blots were hybridized with a chromosome 2L marker from a cDNA encoding an aspartate kinase-homoserine dehydrogenase bifunctional enzyme (pAKHSDH2) (Muehlbauer et al., MGN, 1994). Linkage analysis demonstrated that pAKHSDH2 was positioned 6.5 +/- 4.5 cM from *Ask2* (Table 1). To further position *Ask2*, chromosome 2L DNA markers *umc55* and *umc5* were used on the B73 x A188 F3 *Ask2* population. *umc55* exhibited no recombination with pAKHSDH2 in this population and was

Table 1. Chi-square analysis of the segregation data for the *Ask2* locus (lysine-insensitive AK, threonine-overproducing mutant) with chromosome 2L markers, pAKHSDH2, *umc55* and *umc5*.

Loci pair	Data type	n	X^2	df	*P*	r +/- S.E.
Ask2/pAKHSDH2	F3	16	22.04	4	0.0002	6.5 +/- 4.5
Ask2/*umc55*	F3	16	22.04	4	0.0002	6.5 +/- 4.5
Ask2/ *umc5*	F3	16	8.12	2	0.017	13.4 +/-9.1
umc5/pAKHSDH2	F3	16	11.07	2	0.0039	6.4 +/- 6.3

n = Number of F3 lines.
P = Probability of a greater value of X^2.
r +/- S.E. = recombination frequency plus or minus the standard error.
The Linkage-1 computer program (Suiter et al., J. Hered. 74:203-204, 1983) was used in this linkage analysis.

also positioned 6.5 +/- 4.5 cM from *Ask2* . *umc5* was positioned 13.4 +/- 9.1 cM from *Ask2*. These data demonstrated that the threonine-overproducing trait conferred by *Ask2* is located on chromosome 2L. However, the small population size precludes determination of the marker and gene order.

Identification of point mutations which confer lysine-insensitivity to maize dihydrodipicolinate synthase

--Jonathan M. Shaver, Douglas C. Bittel, David A. Somers and Burle G. Gengenbach

Dihydrodipicolinate synthase (DHPS) catalyzes the first step specific to lysine biosynthesis in plants. DHPS is a key regulatory enzyme in the lysine biosynthetic pathway as it is sensitive to feedback inhibition by lysine. DHPS is also inhibited by a number of lysine analogs. The DHPS cDNA has been isolated from maize by selection for restoration of growth to an *E. coli* auxotroph DHPS- transformed with sequences from a maize cDNA library (Frisch et al., MGG, 1991). In an attempt to alter lysine regulation of DHPS, we have mutagenized the auxotrophic strain containing maize DHPS and selected for growth in the presence of a lysine analog, S-2-aminoethyl-L-cysteine.

From the time of the initial report in 1991 (Sellner et al., MNL 66:94, 1992), 12 additional mutants have been identified which are lysine-insensitive. Preliminary kinetic analyses of crude DHPS preparations have shown each mutant to be insensitive to 1 mM lysine compared to 50% inhibition at 25 uM for wildtype maize DHPS. No differences in sensitivity to lysine among the mutant forms of maize DHPS have been identified. The DHPS cDNA of six independently derived mutants has been sequenced resulting in the identification of three different mutations (see Figure 1).

```
WT  5' GGC AAC ACA GGA AGC AAC TCA ACC AGA GAA GCC GTC CAC GCA ACA GAA CAG 3'
       Gly Asn Thr Gly Ser Asn Ser Thr Arg Glu Ala Val His Ala Thr Glu Gln
                                                            *
1-1 5' GGC AAC ACA GGA AGC AAC TCA ACC AGA GAA GCC GTC CAC GTA ACA GAA CAG 3'
       Gly Asn Thr Gly Ser Asn Ser Thr Arg Glu Ala Val His Val Thr Glu Gln
                        *
A   5' GGC AAC ACA GGA AAC AAC TCA ACC AGA GAA GCC GTC CAC GCA ACA GAA CAG 3'
       Gly Asn Thr Gly Asn Asn Ser Thr Arg Glu Ala Val His Ala Thr Glu Gln
                                            *
B   5' GGC AAC ACA GGA AGC AAC TCA ACC AGA AAA GCC GTC CAC GCA ACA GAA CAG 3'
       Gly Asn Thr Gly Ser Asn Ser Thr Arg Lys Ala Val His Ala Thr Glu Gln
                                                        *
C   5' GGC AAC ACA GGA AGC AAC TCA ACC AGA GAA GCC GTC CAC ACA ACA GAA CAG 3'
       Gly Asn Thr Gly Ser Asn Ser Thr Arg Glu Ala Val His Thr Thr Glu Gln
```

Figure 1. Nucleotide sequence 458 to 508 and amino acid sequence 153 to 169 of maize DHPS are shown. The asterisk and boldface type represent the respective nucleotide and subsequent residue changes which confer lysine insensitivity to maize DHPS. The wildtype sequence is shown in the first block. Mutant 1-1 was described by Janita M. Sellner (MNL 66:94, 1992).

The identification of four mutations within 10 residues of one another suggests that this particular region is an important domain for conferring lysine sensitivity to DHPS. This hypothesis is being tested by computer analysis of secondary structure of this region and by prediction of the changes caused by these mutations

which alter the sensitivity to lysine. In addition, we are attempting to identify this region as a lysine-binding domain by comparison to other known lysine-binding proteins and by comparison to forms of DHPS from other species which are not inhibited by lysine.

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Aspartate kinase-homoserine dehydrogenase bifunctional enzyme

--Gary J. Muehlbauer, David A. Somers, Benjamin F. Matthews and Burle G. Gengenbach

Aspartate kinase (AK) and homoserine dehydrogenase (HSDH) are enzymes in the aspartate-derived amino acid pathway which leads to the production of lysine, methionine, threonine and isoleucine. Regulatory control of AK and HSDH is mediated through feedback inhibition by end product amino acids. AK catalyzes the first reaction in this pathway and exists in isoforms that are feedback inhibited by lysine, lysine plus S-adenosyl methionine and threonine. Recently in maize, threonine-sensitive AK was shown to copurify with threonine-sensitive HSDH, indicating the possibility of an AK-HSDH bifunctional enzyme (Azevedo et al., Phytochem. 31:373-374, 1992). The putative AK-HSDH bifunctional enzyme was proposed to be a 180 kDa dimer. HSDH catalyzes the first committed step in the synthesis of threonine and exists in threonine-sensitive and insensitive forms. Threonine-sensitive HSDH was previously shown to be a 180 kDa dimer composed of 89 kDa subunits (Walter et al., J. Biol. Chem. 254:1349-1355, 1979). Therefore, it seems likely that threonine-sensitive AK and HSDH exists as a bifunctional enzyme, whereas, lysine-sensitive AK and threonine-insensitive HSDH exist as monofunctional enzymes.

To further investigate this hypothesis we isolated one partial and two full-length cDNAs encoding AK-HSDH from a λgt11 seedling leaf cDNA library using a carrot AK-HSDH cDNA (Weisemann and Matthews, Plant Mol. Biol. 22:301-312, 1993) as a probe.. pAKHSDH1 is a 3178 bp cDNA consisting of a 41 nucleotide 5' untranslated sequence, a 92 amino acid putative transit peptide sequence, an 828 amino acid coding region and a 377 bp 3' untranslated sequence. pAKHSDH2 is a 3051 bp cDNA consisting of a 49 bp 5' untranslated sequence, a 89 amino acid putative transit peptide sequence, a 828 amino acid coding sequence and a 251 bp 3' untranslated sequence. The predicted molecular weights for subunits encoded by pAKHSDH1 and pAKHSDH2 were 90,319 and 89,818 Da, respectively. pAKHSDH1 and pAKHSDH2 were 82 and 88% identical at the nucleotide and amino acid level, respectively. pAKHSDH1 and pAKHSDH2 were 77 and 75% identical at the amino acid level to carrot AK-HSDH, respectively. Both clones are divided into three domains; an amino terminal AK domain, a central interface domain, and a carboxy terminal HSDH domain. Of potential regulatory interest was the conservation of sequences observed in the HSDH domain with four sequence motifs from transmitter modules of prokaryotic two-component regulatory proteins. Two-component regulatory proteins produce adaptive responses to environmental stimuli via phosphorylation mechanisms. Therefore, AK-HSDH may possess a phosphorylation mechanism as a way to regulate enzyme activity, sensitivity or both.

To verify that the clones encode functional AK and HSDH activity, a biochemical and immunological study was conducted. Antibodies were raised against a 13-amino acid peptide sequence from pAKHSDH1. AK and HSDH activities were copurified using a Blue Sepharose column. The Blue Sepharose fraction contained only threonine-sensitive AK activity and no lysine-sensitive AK activity, whereas 72% of the HSDH activity in this preparation was threonine sensitive. Threonine-sensitive AK and partially threonine-sensitive HSDH activities migrated to the same position on native PAGE. The pAKHSDH1-derived antibodies cross-reacted on a native protein blot to a protein that corresponded to threonine-sensitive AK and partially threonine-sensitive HSDH activities. The antibodies also cross-reacted with an 89 kDa protein on SDS PAGE, which is the same molecular weight as previously reported for threonine-sensitive HSDH and the same size as the predicted subunit molecular weights for pAKHSDH1 and pAKHSDH2. These data indicated that pAKHSDH1 encodes a subunit of threonine-sensitive AK-HSDH. pAKHSDH2 contains high identity to pAKHSDH1, indicating that it probably also encodes for a subunit of threonine-sensitive AK-HSDH.

RNA blot analyses of AK-HSDH demonstrated hybridization to a single 3.2 kb transcript in embryo, endosperm, leaf and Black Mexican Sweet tissue culture cells. These data demonstrated that threonine-sensitive AK and HSDH activities are encoded by a single transcript. Low stringency hybridizations and washes did not detect smaller transcripts that might encode monofunctional AK or HSDH. These data indicated that AK-HSDH has diverged significantly from monofunctional AK and HSDH.

The chromosomal locations of pAKHSDH1 and pAKHSDH2 were determined using the immortalized F2 population created at the University of Missouri and provided by E. Coe (Gardiner et al., Genetics 134:917-930, 1993). DNA blots containing the immortalized F2 population were hybridized with gene-specific probes from pAKHSDH1 and pAKHSDH2. Using the MAPMAKER computer program (Lander et al., Genomics 1:174-181, 1987) and the University of Missouri core RFLP data base, pAKHSDH1 and pAKHSDH2 were positioned on chromosomes 4S and 2L, respectively. pAKHSDH1 was positioned between *umc191*(gpc1) and *umc201*(nr) at 7.5 cM and 2.7 cM, respectively. pAKHSDH2 was positioned on chromosome 2L between *umc055* and *umc139* at 3.6 cM and 4.9 cM, respectively. Nonspecific probes for the partial cDNA clone, pAKHSDH3, detected polymorphisms only on chromosomes 2L and 4S in the same locations as pAKHSDH1 and pAKHSDH2. These blots also contained other monomorphic bands; therefore, an alternate location for pAKHSDH3 may be possible. A gene-specific probe for pAKHSDH3 did not detect a polymorphism between the F2 parents, Tx303 and CO159, with 20 restriction enzymes and it has not been possible to determine the map location of pAKHSDH3.

Further efforts will be directed at isolating the full length cDNA for pAKHSDH3 and determining its map location. However, highest priority will be investigating the potential phosphorylation mechanism of AK-HSDH.

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***MuDR*-like elements in Zapalote chico maize**

--Christine Warren and Virginia Walbot

MuDR is the new designation for the transposase-encoding master *Mutator* element previously designated as *Mu9*, *MuR1* or

MuA2. *MuDR* encodes two major sense transcripts that, when fully spliced, yield mRNAs of ~2.9 kb and ~1.0 kb. The transcripts are convergent, initiating on opposite strands, but in the same sequence, in the nearly identical terminal inverted repeat elements. Between the polyadenylation sites mapped from cDNAs, there is a several hundred base pair region containing several classes of short direct and inverted repeat elements.

In a Southern blot survey of inbred maize lines, we found no evidence for an intact *MuDR*-like element, using probes from both transcribed regions. The collection of exotic lines available from the Co-op was also checked, and Zapalote chico contained multiple (10 - 20) copies of a *MuDR*-like element. Detailed genomic Southern mapping indicates that all restriction sites expected from the sequenced *MuDR* (Hershberger et al., PNAS 88:10198, 1991) are present in the Zapalote chico element, however, the intergenic region is several hundred bases longer in Zapalote chico. This was confirmed by PCR amplification using 8 pairs of primers that span *MuDR* followed by diagnostic restriction digests of the PCR products: *MuDR* and the Zapalote chico elements are identical in 7 regions, but differ by several hundred bases in the product that spans the intergenic region. To test whether the Zapalote chico elements are "active," northern blot analysis was performed; there are weak signals for the 2.9 kb and 1.0 kb transcripts, but the level of transcript is about 1% of that found in a typical *Mutator* line.

The data available are sufficient to suggest that *Mutator* is a normal part of the maize genome--there is no need to invoke horizontal transmission from another species. The unusual distribution of *MuDR*-like elements in modern maize, in contrast to the widespread occurrence of *Ac* and *En/Spm*, may reflect stronger selection against *Mutator* activity resulting in element loss in most lines.

The single Zapalote chico line available from the Co-op was recovered during a collecting trip to Southern Mexico about 40 years ago. The line has been propagated by self- or sib-crossing since then. The seed is pure white, eliminating the easiest method to visualize transposable element activity, and early selection for vigor to overcome "problems" with an exotic line from a different latitude might also have effectively selected against *Mutator* activity in the line available. Questions under study include: (1) Is Zapalote chico maize still grown, and, if so, where and why? Zapalote chico turns out to be the "staff of life" of the Zapotec people, a group of about 300,000 native Americans living in Oaxaca, Mexico. The masa prepared from Zapalote chico flour is ideal for preparing totopos, a dry cracker baked in a clay oven. Unlike tortillas, totopos store well for up to 6 months. Totopos are a key element in the 3,500 year oral history of the Zapotecs, suggesting that this line is very ancient. (2) Is there evidence for instability of Zapalote chico in its local environment? The Zapotecs believe that thieves who steal their corn will suffer because the stolen corn will poison the fields when mixed with other corn. Mexican corn breeders have found that crosses between inbred lines and Zapalote chico yield hybrid dysgenesis rather than hybrid vigor, giving scientific support to the Zapotec legend. The instabilities observed in F1 hybrids are reminiscent of an active *Mutator* system. It has been impossible for Mexican breeders to establish stable selfed lines from the hybrid populations. Similarly, Georgia Davis and R. Kowles made crosses between Zapalote chico and Wilbur's Knobless Flint and noted that the hybrid and backcrossed lines were not vigorous (personal communication). We plan to check the hypothesis that the low level of *MuDR* transcripts typical of Zapalote chico is increased in the hybrids. (3) How widespread are *MuDR*-like elements in Zapalote chico? We have 55 new accessions of Zapalote chico, and a survey will be performed on these materials. (4) Can crosses with Zapalote chico reactivate quiescent *Mu* elements in inactive *Mutator* lines? Although Zapalote chico has clear plant and kernel phenotypes, the line is not homozygous for anthocyanin markers: most populations are *r-g Bz2 Bz1* and segregating for dominant and recessive (low or non-functional) alleles of *b*, *c1* and *c2*. Some individuals are *r-r* and have purple anthers. Reactivation tests are in progress with inactive lines carrying mutable alleles of *bz2* and *bz1*. (5) What is the exact structure of the *MuDR*-like elements in Zapalote chico? This will require cloning and sequencing an element.

Structure and regulation of the *Bronze-2* promoter

--John P. Bodeau and Virginia Walbot

Anthocyanin biosynthesis in maize requires two classes of regulatory proteins, C1 and R, proposed to be transcriptional activators responsible for the coordinate expression of a suite of structural genes, including *Bronze2* (*Bz2*). Structural analysis of the *Bz2* promoter was performed in electroporated BMS maize protoplasts in which 35S:R ("pR") and 35S:C1 ("pC1") plasmids activate the endogenous anthocyanin structural genes resulting in pink protoplasts within 24 hr in virtually all viable cells. These plasmids also activate expression of a *Bz2*:luciferase reporter construct (Bodeau and Walbot, Mol. Gen. Genet. 233: 379-387, 1992). To better understand the coordinate regulation of the maize anthocyanin structural genes, we analyzed the structure and function of the *Bz2* promoter by deletional and site-directed mutagenesis of chimeric reporter gene constructs.

Deletional analysis showed that sequences necessary for regulated expression of *Bz2* reside between -63 and -84 relative to the major transcription start site. Sequences from -2200 to -224 could be deleted with no effect on luciferase expression either in the presence or in the absence of co-electroporated pR and pC1. Deletion to -134 and to -84 decreased expression about 50% and 90%, respectively, but these promoters were still inducible. More severe deletions, to -63 and to -48, virtually eliminated inducibility. Thus, sequences critical for R and C1 inducibility likely reside between -63 and -84 in the *Bz2* promoter, with sequences out to -224 contributing to the overall induction. There is no evidence for negative elements whose deletion would allow R- or C1- independent expression, because expression remained very low in the absence of the two regulators.

Previous analysis of the *Bz1* promoter indicated that two sequence motifs were important for induction by R and C1 (Roth et al., Plant Cell 3: 317-325, 1991). The first is a TAACTG element (which we designate a "C1-motif"), the sequence bound by Myb, an animal homologue of C1. The second element is a CAGGTG sequence similar to CACGTG (which we designate an "R-motif"), the consensus binding site of many bHLH proteins, homologous to R. Site-directed mutagenesis of our *Bz2*:luciferase plasmid demonstrated that R- and C1-motifs located between -63 and -112 are important for *Bz2* promoter activation as well. Three putative R-motifs were mutated: R:-91 (CACGAG from -91 to -86), R:-68 (CACGAC from -68 to -63), and R:+5 (GAGGTG from +5 to +10). Two putative C1-motifs were mutated: C1:-112 (CAGTTA from -112 to -107) and C1:-78 (CGGTCA from -78 to -73). Mutating either the C1:-78 element or the R:-68 el-

ement alone decreased expression of the reporter gene, but the other three motifs could be mutated without a significant change in luciferase levels. Combinations of double and triple mutants indicated that the C1:-112 element is also important for full promoter activity.

We noticed a region from -191 to -139 that contained several R- and C1-motifs, but that could be deleted with less than a 50% reduction in promoter inducibility by R and C1. To test whether this was a redundant R/C1 responsive region, we fused it to two non-inducible *Bz2* promoter deletions and to a truncated 35S promoter, and assayed the inducibility of the chimeric promoters. Each of the base plasmids lacking the upstream promoter region was at most 2-fold inducible by pR and pC1. With the addition of the -191 to -139 promoter fragment, the resultant promoters were induced about 10-fold by pR and pC1. The absolute expression level was, however, only about 3% the level of the wild-type *Bz2* promoter. Interestingly, the upstream fragment inserted in the reverse orientation was equally functional. Thus the -191 to -139 region may act as an enhancer-like R and C1 responsive region that is qualitatively, but not quantitatively similar to the -63 to -84 R and C1 control region.

While chimeric reporter genes are powerful tools for regulatory studies, a potential hazard is that regulatory regions present in the coding sequence of the native gene may be overlooked. To test for such control, we introduced a 4 nt insertion into a genomic *Bz2* clone, in order to differentiate transcripts from the endogenous gene, and reconstructed a series of *Bz2* genes with truncated and mutated promoters. We compared the mRNA expression levels in electroporated protoplasts using RNase protection. Just as we observed using reporter constructs, deletion of promoter sequences to -224 or to -134 had virtually no effect on mRNA level, while deletion to -48 resulted in very low mRNA accumulation. Mutating the C1:-78 motif also resulted in about a 75% decrease in mRNA accumulation. These results confirmed that R/C1 regulation of *Bz2* is primarily transcriptional and that the regulation is mediated through the promoter elements identified in reporter gene expression assays.

Shielding and repair: responses to ultraviolet radiation

--Ann E. Stapleton and Virginia Walbot

Plants use sunlight for photosynthesis, and are unavoidably exposed to the ultraviolet radiation that is also present in sunlight. Because plants lack the behavioral mechanisms that mobile animals use to respond to excess radiation, they may have evolved novel and effective mechanisms to protect essential functions from radiation damage. Plants have two basic mechanisms for coping with UV damage--shielding and repair.

Between 95% and 99% of incoming UV radiation does not penetrate through the epidermal layer. Most of this UV shielding is due to absorption by epidermal flavonoid compounds (Robberecht and Caldwell, 1978, Oecologia 32:277). We show that anthocyanins synthesized under the control of the regulatory genes *B* and *Pl* can protect maize leaf DNA from damage induced by UV radiation in vivo (Fig. 1).

Thus the interior of the plant is protected from the induction of UV radiation damage by epidermal anthocyanins. Shielding, however, does not provide complete protection from UV damage, and is not sufficient to explain differences in UV tolerance between different plants (Sullivan et al., Amer J Bot 79:737, 1992).

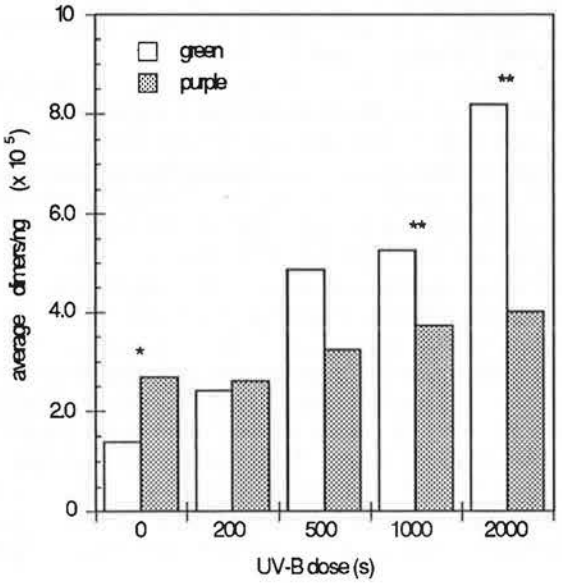

Figure 1. DNA damage levels in green (*b pl r-g*) and purple (*B Pl*) plants after UV-B irradiation. Sheath tissue was irradiated, DNA extracted and amount of cyclobutane pyrimidine dimer damage measured using our antibody assay (Stapleton and Walbot, Plant Mol. Biol. Rep. 11:230, 1993).

We have therefore measured the repair of UV-induced DNA damage in maize seedlings. We have measured both photoreactivation repair (in which the enzyme photolyase uses light to remove UV-induced DNA damage) and excision repair (which occurs in the light and in the dark).

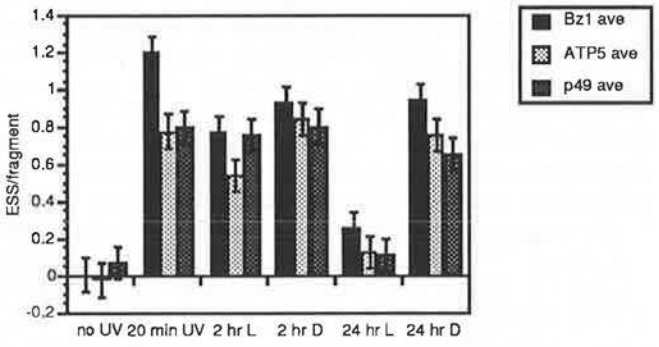

Figure 2. Repair of UV-induced DNA damage in maize seedlings. Seedlings (*bz2/bz2*) were grown for 1 week, irradiated with 6 germicidal UV-C bulbs for 20 min., and allowed to recover for 2 or 24 hrs. in light or dark. Damage was measured using a technique originally developed for determination of repair rates in mammalian tissue cultures (Bohr and Okumoto, *in* DNA Repair, a Laboratory Manual, E. C. Friedberg and P. C. Hanawalt, eds., Marcel Dekker, New York, 1988). We determined damage levels by determination of the number of sites in each *Hpa*I fragment sensitive to T4 endonuclease V (ESS/fragment) that hybridized to a nuclear (*Bz1*), chloroplast (ATP5) or mitochondrial (p49) probe.

We demonstrate (Fig. 2) that there is photoreactivation-type repair (reduced damage after 24 hrs. of recovery in the light) of UV-induced DNA damage in nuclear, mitochondrial and chloroplast genes in maize seedlings. This suggests that all three compartments contain photolyase. Developmental and molecular characterization of maize photolyase(s) is proceeding.

Bronze2 and related genes: a clue to the function of the Bz2 protein?

--Kathleen A. Marrs and Virginia Walbot

The protein encoded by the *Bronze2* gene in maize performs the last genetically defined step in the anthocyanin biosynthetic pathway (*C2* -->*A1*-->*A2*-->*Bz1*-->*Bz2*), resulting in the purple pigmentation seen in various plant tissues such as the leaves, husks, and aleurone. While its biochemical function in this pathway has not yet been shown, several functions for the BZ2 protein can be envisioned: the BZ2 protein may function as a malonyltransferase to catalyze the addition of malonic acid to the precursor cyanidin-3-glucoside to produce the cyanidin-3-malonylglucoside found in the maize vacuole (Harborne and Self, Phytochemistry 26:2417-18, 1987), or as a transporter to ensure that the anthocyanin is delivered into the vacuole, or it may stabilize the anthocyanin in the vacuole by complexing with metal ions. This lab has previously shown (Nash and Walbot, MNL 66:104, 1992; Nash and Walbot, Plant Physiol. 100:464-71, 1993) that the BZ2 amino acid sequence is highly similar to the amino acid sequence of the soybean small heat shock protein (HSP)-like *hsp26A* gene; there is 66% similarity between the first exons of these genes. In addition, Schmitz and Theres (Mol. Gen. Genet. 233:269-77, 1992) also reported the similarity of *Bz2* with a number of other plant genes. However, only the percent similarity, and not the actual alignment, was reported. We have aligned the first exon sequences of all genes related to *Bz2*, shown in Figure 1.

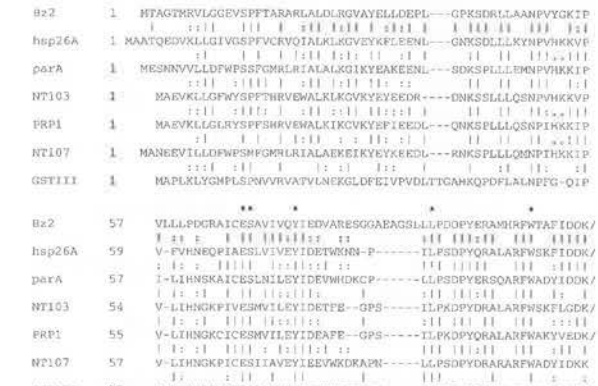

Figure 1: Amino acid sequence alignment between the first exon of *Bz2* and the first exon (or amino terminal "half") of other related genes: soybean *hsp26A* (Czarnecka et al., Mol. Cell. Biol. 8:113-22), tobacco *parA* (Takahashi et al., PNAS 86:9279-83), tobacco *NT103* and *107* (van der Zaal et al., Plant Mol. Biol. 16:983-98; Droog et al., Plant Mol. Biol. 21:965-72, 1993), potato *PRP1* (Taylor et al., Mol. Plant-Microbe Interact. 1:157-60), and maize *GSTIII* (Grove et al., Nucl. Acids Res. 16:425-38). The paper by Droog et al. (Plant Mol. Biol. 21:965-72, 1993) reports the complete alignment (both first and second exon) for all these genes with the exception of *Bz2*. Residues identical and very similar to *Bz2* are indicated by (|) and (:), respectively. Similarity groups used are I,L,V,F,W,Y; K,H,R; D,E,N,Q; S,T. Hyphens indicate gaps introduced to optimize alignment. Asterisks (*) indicate residues identical in all seven sequences. A slash (/) indicates the position of the exon/intron boundary, where known.

In addition to the high amino acid homology only within the first exons of all these genes (amino terminus of protein), there are several other striking characteristics suggesting that these proteins are related:

•Structurally, the organization of all the genes is highly conserved: each gene for which the exon:intron structure was reported contains a single intron in the exact same position in each message. Beyond the first exon, the similarity of Bz2 with the other genes is extremely low. This conservation of exon:intron boundaries supports the suggestion (Nash and Walbot, MNL 66:104, 1992) that this class of proteins may have been constructed by the process of exon-shuffling.

•Functionally, all are involved in responses to environmental stress conditions, particularly auxins and heavy metals, although the biochemical function of most is poorly understood (the exception being the glutathione-S-transferases or GSTs):

Gene	Possible or known function	Induced by:
Bz2	Anthocyanin pathway, malonyltransferase(?)	ABA?, cadmium, cold
Hsp26	Weak homology (in 2nd exon only) to small HSPs	Cadmium, heat, auxin
parA	Auxin regulated protein	Auxin
NT103	Cell-cycle, auxin regulated; in vitro GST activity	Auxin
PRP1A	Potato pathogenesis-related protein	Fungal attack
NT107	Cell-cycle, auxin regulated	Auxin
ZmGSTIII	Glutathione-S-transferase	Herbicides

•Transcripts of both the *Bz2* and the *hsp26A* genes are unusual in that high amounts of unspliced, intron-containing message have been reported, particularly as a result of heavy metal stress as well as a generalized "field" stress (Czarnecka et al., Mol. Cell. Biol. 8:1113-22, 1988; Nash et al., Plant Cell 2:1039-49, 1990; Marrs and Walbot, in preparation). Both the *Bz2* and the *hsp26A* unspliced transcripts, if translated, would encode highly related, truncated proteins of about 14 kD (vs. the "full length" protein of ~26 kD) because of a stop codon in the intron of each gene. The production of the 14 kD protein was observed by Czarnecka et al. after hybrid-release translation using RNA from cadmium-stressed seedlings. While the presence of unspliced transcripts of the other genes in this class is not mentioned in the literature, their presence would also encode for truncated proteins of 14-17 kD because of stop codons in the introns.

Could this set of stress-related RNAs be a "barometer" of environmental stress conditions? We postulate that the 14 kD proteins generated from *Bz2* and related genes may be specialized stress proteins. These genes may respond to stress by encoding two proteins, each with a separate function - a "correctly" processed, larger form (26 kD) that is involved in a specific function -- anthocyanin production, response to auxin, etc.-- and an unspliced "stress" form of 14 kD, whose role could either be a stress indicator or even play some direct role in the response to stress. In this case, the intron (presence or absence) could indicate a boundary between functional domains of the proteins, with the barometer function specified by the first exon. This "barometer" concept was first suggested by Czarnecka et al. (Plant Mol. Biol. 3:45-58, 1984) to describe the function of a class of soybean small HSPs, but it would seem to apply to the proteins encoded by the other genes in this class as well.

Another possibility is that the proteins encoded by these genes could have dual roles in the cell both during normal cell metabolism and during stress. Several of these proteins share significant homology with authentic plant and animal GSTs, including the BZ2 protein sequence to some extent. GSTs are involved in the detoxification of a wide variety of xenobiotic compounds and herbicides (and heavy metals indirectly, which are detoxified by the synthesis of phytochelatin from glutathione). In addition, GSTs also play a role in normal plant secondary metabolism, and are also thought to protect cells against oxidative damage. Malonyltransferase enzymes are another class of enzymes in plants that have roles in both detoxification of xenobiotic compounds as well as during normal secondary metabolism (Sandermann, TIBS 17:82-84, 1992). Genes for known malonyltransferases have yet to be cloned. The BZ2 protein could have a dual function as a malonlytransferase, malonating either cyanidin-3-glucosides during anthocyanin production or other xenobiotic substrates for detoxi-

fication during stress. Alternatively, BZ2 could function as a metal binding protein either to stabilize anthocyanins or to chelate heavy metals during stress.

Our current findings concerning *Bz2* regulation during cadmium stress as well as previous results from this lab imply that alternative forms of *Bz2* may play a role in the cell during stress. We are currently testing whether the BZ2 protein functions biochemically as a malonyltransferase or as a glutathione-S-transferase both during anthocyanin production and during stress. Positive results would provide the first evidence of the biochemical function of *Bz2*.

Anthocyanin genotypes in an A188 background, and their pigment phenotypes in embryogenic calli

--John P. Bodeau and Virginia Walbot

Maize anthocyanin genotypes that efficiently give rise to friable, Type II embryogenic callus were produced by repeated backcrossing to the inbred line A188. These lines were produced to better understand the endogenous genetic regulation of anthocyanin synthesis in such callus and to facilitate use of the anthocyanin genes as visible markers for transformation. These lines, summarized in Table 1, are available upon request and have been

Table 1. Maize anthocyanin genotypes introgressed into A188

Genotype	Description	Callus color	Nuclear background	Derivation
R-r C1 [B/?](x) Pl	Full color	Red	.93A188/.07W23	JB:F1-6(x)
R-r C1 b Pl	Full color - *b*	Red	.97A188/.03W23	JB:F2-1(x)
R-r C1 B pl	Sun-red	Sun-red	.97A188/.03W23	JB:F10-4(x)
R-g C1 b pl	Aleurone red	Sun-red	.93A188/.07K55	JB:F5 (x)
r-r C1 b Pl	*r-r* tester	Red	.93A188/.07W23	JB:F4-2(x)
r-g C1 b pl	*r-g* tester	Colorless	.75A188/.25W23	JB:EH5 (x)
R-r c1 b [Pl/?](x)	*c1* tester	Red	.93A188/.07W23	JB:F9-5(x)
R-r c1 b pl	*c1* tester	Sun-red	.93A188/.07W23	JB:F8-8(x)
r-g c1 b pl	All colorless	Colorless	.87A188/.12W23	JB:F7 (x)
r-r C1 B-peru Pl	*B-peru*	not tested	.93A188/ .07(Neuffer's)	JB:F11-2(x)
R-nj C1 b pl	*R-navajo*	Sun-red	.93A188/ .07(Neuffer's)	JB:F12 (x)
R-scm2 C1 b pl	*R-scm2*	not tested	.75A188/ .25(Neuffer's)	JB:F13 (x)
R-scm2/r-r C1/c1 b pl	*R-scm2* construction	not tested	.87A188/ .12(Neuffer's)	JB:F33 x F13-2
[r-ch:H/r-r Pl/pl](x) c1 b	*r-cherry:Hopi*	not tested	.5A188/ .5(Racchi's)	JB:F14-5(x)
1/4=r-ch:H/r-r c1 b Pl/pl	*r-ch:Hopi* construction	not tested	.75A188/ .25(Racchi's)	JB:F33 x F14-5
a1(R C1 B Pl seg.)	*a1*	Colorless	.90A188/.10W23, K55,N1	JB:F18-2(x)
a2 R-g C1 b pl	*a2*	Colorless	.93A188/.07W23 or K55	JB:F20-3(x)
bz1 R-r C1 B pl	*bz1*	Colorless	.75A188/.25W23	JB:F21-1(x)
bz1 R-r/? C1/? B/? pl	*bz1*	Colorless	.93A188/.07W23	JB:F23-2,-7(x)
bz2 R-r C1 b pl	*bz2*	Colorless	.87A188/.12K55	JB:F24(x)
bz2 R-r C1 b Pl	*bz2*	Colorless	.87A188/.12W23, K55	JB:F25-2(x)
Also included is the A188 line used as backcross parent:				
r-r b c1 pl	A188 inbred	Weak sun-red	1.0A188 Stanford	JB:F33(x)

The genotype relevant to anthocyanin accumulation is shown. Alleles of the regulatory loci *R, B, C1,* and *Pl* are shown for all lines, with uncertain or segregating loci indicated by brackets. All lines were homozygous for the structural genes *C2, Pr, A1, A2, Bz1,* and *Bz2* unless otherwise indicated. The pigmentation phenotype of embryogenic, Type II callus of these genotypes is shown. The approximate fractional composition of each nuclear background is given by inbred source; all lines have A188-derived cytoplasm. The parental derivation of each seed-stock is given for reference; John Bodeau's "F" season was grown during summer, 1993 at Stanford.

submitted to the Stock Center. The overall morphology of the backcrossed lines resembles inbred A188 plants, which under summer conditions at Stanford typically are short in stature, thick stemmed, rarely tillered, early flowering, and form kernels with a pronounced sharp point at the silk attachment site. Immature embryos of most of the lines efficiently initiated embryogenic callus when plated on N6 media.

Anthocyanin accumulated in callus of several genotypes. When present, anthocyanin accumulated in the surface cells of undifferentiated, friable tissue forming the bulk of Type II callus. The embryoids arising from red callus, however, were colorless, although the suspensor-like supporting stalk was often pigmented. Prolonged light treatment increased anthocyanin accumulation in all pigmented genotypes, and was absolutely required for pigmentation in *pl* calli.

As in other tissues, both an *R*-family member (*R* or *B*) and a *C1*-family member (*C1* or *Pl*) were required for callus pigmentation. Multiple *R*-family alleles conferred pigmentation. The genotype *r-g b C1 pl*, lacking any functional *R* or *B* allele, was colorless, indicating that at least one *R*-family member is required in callus. Both the *S* (seed) or the *P* (plant) component of the complex *R-r* locus were functional in callus. *R-g* calli, lacking the plant component, *R(P)*, and *r-r* calli, lacking the seed component, *R(S)*, were both pigmented. *R-r* genotypes were, of course, also strongly pigmented. An additional *R* allele, *R-navajo* (*R-nj*), was also functional in callus. *R* alleles *R-scm2* and *r-cherry:Hopi*, and *B* alleles *B-Intense* and *B-peru*, were not required in addition to a functional *R* allele as discussed above. Type II calli of the correct genotypes were not successfully initiated to test whether these alleles were individually sufficient.

The requirement for a *C1*-family member resembled that of mature plant tissues: either *Pl* or *pl*, plus prolonged light treatment, were necessary and sufficient for callus pigmentation. *C1* is not required, nor is it sufficient for callus pigmentation in the dark in the genotype *R-r B C1 pl*, or any other. Interestingly, light-induced pigmentation was seen in the genotype *R-g b C1 pl*, which in planta accumulates anthocyanins independently of light, but only in aleurone tissue.

In addition to regulatory genotypes, we initiated callus lines individually homozygous recessive for the structural genes *a1*, *a2*, *bz1*, and *bz2*. None of these callus lines accumulated visible anthocyanin, but some turned brownish or necrotic more quickly after subculture than did wild-type calli. These observations suggest that flavonoid intermediates accumulated in calli, much as they do in plant tissues.

While embryogenic callus pigmentation has similar genetic requirements as intact plant tissues, normal patterns of tissue-specific gene expression are not maintained in callus. The *R*-family alleles *R-nj*, *R(S)*, and *R(P)* were each functional in embryogenic callus; in planta *R(S)* and *R(P)* act in mutually exclusive seed or plant tissues, while *R-nj* acts in both. For the *C1* requirement, either *Pl* or *pl* plus prolonged light treatment, but not *C1*, was sufficient. Pigmentation conferred by the combination of *R(S)* and *pl* plus light (genotype *R-g b C1 pl*) was unexpected because *R(S)*, normally active only in the aleurone, in this case required either light-induced expression of *pl*, which is not usually active in aleurone, or light-induced (enhanced?) expression of *C1*, which is strongly active in aleurone in the dark. Thus either alleles that function in mutually exclusive aleurone and somatic tissues are co-expressed, or the wild type *C1* allele acquires a light requirement in callus. In any case Type II callus appears to represent a novel tissue-type with similarities to both seed and plant tissues.

STUTTGART, GERMANY
Universität Hohenheim

Herbicide resistance as a marker in screening for maternal haploids

-- H. H. Geiger, S. R. Roux and S. Deimling

In crosses with inbred line Stock 6 as pollinator parent, Coe (Am. Nat. 93:381-382, 1959) observed 2.3% maternal haploids. Recently, Lashermes and Beckert (TAG 76:405-410, 1988) were able to increase the haploid frequency to 2 - 5 % using inbred WS 14 (derived from a cross between W23ig and Stock 6) as the inducer line. This phenomenon could be used as a simple, fast, and inexpensive means of haploid production if a genetic marker existed which would allow efficient screening for haploids among the regular sexual diploids.

In our experiments we investigated the usefulness of the transgenic resistance against the herbicide BASTA as a physiological marker. The resistance is inherited as a monogenic dominant trait. Resistant and sensitive genotypes can be distinguished in young seedlings by applying BASTA in a concentration of 1% to the terminal half of one leaf. Three to four days later the herbicide damage becomes visible on the sensitive seedlings whereas the resistant ones remain unaffected.

A BASTA-resistant line (kindly provided by Dr. G. Donn, Hoechst AG, Frankfurt/M.) was crossed to the inducer line WS 14 (kindly provided by Dr. M. Beckert, INRA, Clermont Fd., France), backcrossed three times to WS 14, and subsequently selfed using resistant plants for backcrossing and selfing. From the resulting BC3S1 versions of WS 14, 6 homozygous resistant plants were used as pollinator parents in the present study. Homozygosity of the resistance gene was determined *a posteriori* in the second selfing generation (BC3S2).

To check the effectiveness of the BASTA marker, we used an S2 line with the monogenic recessive mutant *liguleless* as female parent. Maternal haploids, as well as spontaneously doubled maternal haploids, should be both sensitive to BASTA and *liguleless*, whereas sexual (F1) seedlings should be heterozygous at the two loci and thus display BASTA resistance and normal leaf morphology. In four progenies consisting of 111, 179, 202, and 259 seedlings, the frequency of maternal haploids (or doubled haploids) was 1.0, 1.1, 1.6, and 3.8%, respectively. In all cases the BASTA sensitive plants were *liguleless* and the resistant ones were normal.

These results clearly demonstrate the usefulness of BASTA resistance as a foolproof marker system to identify maternal haploids. In comparison to the *R-nj*-embryo marker (Greenblatt and Bock, J. Hered. 58:9-13, 1967), BASTA resistance has the advantage of unambiguity and independence of the genetic background of the female parent. However, using a transgenic inducer genotype for haploid production on a commercial scale would require field experiments, for which a permit might be difficult to obtain in certain countries. The final doubled haploid lines, on the other hand, could be grown without any such restrictions.

TAEJON, KOREA
Chungnam National University

Genetics of super thin pericarp

--Insup Lee, Bongho, Choe, Wonkoo Lee and Heebong Lee

We reported that one of the Korean waxy inbreds developed by the authors to improve the table quality of waxy hybrids had very thin pericarp (MNL 67:109, 1993). In order to determine the genetic nature of the thin pericarp inbred, we made diallel crosses among six waxy inbreds. We measured pericarp thickness of the parents as well as the hybrids using the method reported by Wolf et al. (Agron. J. 61:777-779, 1969). Results indicated that waxy hybrids with thin pericarp can be developed by choosing proper parental lines. Of the six inbreds, the pericarp thickness of Jewon inbred was about 40 μm and the pericarp thickness of Danyang inbred was about 100 μm. Hybrids crossed with Jewon also showed thinner pericarp compared with other hybrids. However, Danyang inbred which has thick pericarp showed thicker pericarp in hybrid combinations. Variance due to general combining abilities was much greater than the variance due to the specific combining abilities, indicating that additive gene effects are more important. The pericarp thickness of waxy hybrid endosperm varied with the parts of pericarp. The germinal side of the pericarp is thinner than the abgerminal part. The upper part (crown) has thicker pericarp than the lower part (tip) of the kernel.

Tillers taller than the main stem are heritable

--Heebong Lee, Wonkoo Lee, Insup Lee, Bongho Choe and Seungkeunn Chung

We have reported one inbred whose tillers are taller than the main stem (MNL 67:108-109, 1993). Tillers of inbreds and hybrids are generally shorter than the main stem. Most inbreds and hybrids which were developed by the authors for high performing tillering maizes have short tillers compared with the main stem height at maturity. The short tiller heights may be partly responsible for poor ear set of tillers. In a series of developing tillering inbreds, we found one inbred with tillers taller than the main stem as shown in Table 1. The first and second tiller heights of IK4 inbred were taller than the main stem. However, tiller heights of IK1 inbred were shorter than the main stem without exception. Ear heights of tillers were higher than the ear height of the main stem in both inbreds.

Table 1. Main stem and tiller heights and ear height of IK4 and IK1 at maturity, cm.

	IK4		IK1	
Characters	Plant Ht.	Ear ht.	Plant ht.	Ear ht.
Main stem	176.0±9.4*	74.2±5.6	180.0±7.6	83±5.4
1st tiller	198.5±14.5	87.8±6.4	151.7±18.5	116±7.8
2nd tiller	182.0±14.4	85.6±5.5	85.0±20.0	112±10.3
3rd tiller	115.5±19.2	61.8±4.1	--	--

*standard error

Table 2. Main stem and tiller height of F1 hybrids between IK4 and four tillering inbred lines, cm.

Characters	IK4/IK1CI66	IK4/PI213749	IK4/IK1FR3019	IK4/IK1US	P3160**
Main stem	283.0±4.9*	247.7±9.6	227.0±9.5	233.5±4.6	275±8.5
1st tiller	281.7±6.0	253.7±7.2	234.0±6.0	258.0±8.8	--
2nd tiller	221.7±10.7	244.3±6.7	218.3±11.7	251.4±12.4	--
3rd tiller	--	--	--	169.7±16.4	--

*standard error
**check hybrid with no-tillers

Tillering habits of IK4 were well manifested in some of the hybrid combinations (Table 2). Hybrids between IK4 and other tillering inbreds showed almost equal tiller heights to main stem height. The first and second tillers of hybrids between IK4 and IK1US showed much higher tiller heights than the main stem.

Tillering and prolific inbreds

--Bongho Choe, Heebong Lee, Wonkoo Lee and Heechung Ji

Since we reported the tillering characteristics of some of the Korean local open pollinated flint lines in 1980, our efforts to develop high performing tillering hybrids have been continued (MNL 56:62, 1982; IBPGR Newsletter 68:1, 1986; 13th Cong. EUCARPIA, 1985; MNL 62:54, 1987; MNL 63:75, 1988; SABRAO 19:119-122, 1987; MNL 67:108, 1993). We have developed four tillering inbreds, IK1, IK2, IK3 and IK4. IK1 and IK3 inbreds were strictly developed from Korean local flint lines and IK2 inbred was developed from crosses between IK1 and U. S. derived dent type. All inbreds were selfed for more than ten generations and selected based on the number of tillers and tillering characteristics. Table 1 shows some of the unique characteristics of the inbreds.

Table 1. Main characteristics of three tillering inbreds.

Inbreds	Main stem ht cm	Tiller* ht cm	Tillers plant^{-1}	Effective tillers plant^{-1}	Ear ht cm	Ears plant^{-1}	100 kernel weight gr
IK1	158.0	118.5	2.1	2.1	70.3	4.6	15.3
IK2	160.5	90.4	1.9	1.5	80.7	2.5	16.5
IK3	165.3	100.3	1.7	1.5	78.3	3.2	13.4

*average of first and second tiller heights

The characters shown in Table 1 were all based on trials conducted in Korea. Our past experience shows that the inbreds failed to show tillers when planted at Los Banos, Philippines, probably due to high temperature (MNL 67:108, 1993). The inbreds shown in Table 1 were planted on May 1 at Taejon, Korea. The plant density was about 50,000 ha^{-1}. The tiller heights of inbreds are shorter than the main stem height. Each inbred has one to two tillers per plant. The number of ears per plant ranged from three to five. The 100 kernel weights of inbreds are comparatively low, ranging from 13 to 17 grams. In addition to these three inbreds, IK4 was recently developed and its general characteristics are reported in this newsletter (see previous article).

TAICHUNG, TAIWAN
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A new type of non-chromosomal stripe from Taiwanese maize

--Bor-yaw Lin and Hao-Jan Yu

A variant plant was found in the fall planting of 1992, among more than one thousand individuals of a local maize race called Tainan White, the origin of which is sketchy. This race has been cultivated on the island for more than eighty years, presumably originating from the United States through Japan many years ago. Based on Chang's literature review (Know-You News Letter 157:76-83, 1993), the earliest local cultivated maizes included White Flint, Hickory King, Large Yellow, Longfellow and Chiachow. Among these, the phenotype of Hickory King is closest to that of Tainan White, including dent kernels, large grain, white endosperm, ears with 8-10 kernel rows, tall stand, and relatively slow maturity rate (60-75 days). Total SDS-protein gel electrophoretic analysis revealed that the protein pattern of Tainan White was similar to that of Hickory King, a subrace of Southern Dent.

This variant plant exhibited white stripes on all leaves, and the stripes extended from leaf blades to sheath and to the internode below the sheath. Most striped areas were white or yellow white, but some were light yellow green. The transition from green to white tissues was sharp and clear in some areas but was filled with yellow greenish tissues in others. The width of stripes varied, from about 2 cm to less than 0.5 cm, but there was no striation on this plant or its progeny. In those leaves with two halves divided by the midrib, no displacement of the midrib from the leaf center was evident. This plant was somewhat shorter than the surrounding plants, but since Tainan White is not a uniform line, the difference in height may not be part of the variant phenotype.

This plant was self-pollinated to result in an infected ear with about 200 clean healthy kernels. Twenty kernels were planted the following spring and all of them were destroyed by insects. Ten more kernels were immediately germinated in late spring and transplanted to pots, which were placed on the roof of a head house (the only place that we could find by then) with proper protection from insects. The plants did not grow normally, because of inappropriate pot size, constant high temperature (av. 40 to 43 C) and periodical water shortage. Yet, one plant managed to have enough pollen for crossing to produce 85 kernels.

Preliminary crosses involving this variant demonstrated that it was inherited in a manner indicating that the gene responsible for the striped phenotype is not located on chromosomes. First, the variant, after it was crossed reciprocally with hybrid W22/W23 plants, gave different results. The variant, when mated as female, produced an ear with a normal phenotype like that of sib ears, without any small or other off-type kernels. Eighty-five kernels were germinated before planting in the field and resulted in 73 seedlings; 70 of these had yellow greenish leaves which turned yellow white at the 6-leaf stage and died. Three others had striped leaves; two were about two-thirds as tall as their sib plants, and stalks were thinner and leaves were narrower than their sib counterparts. Their leaves had yellow greenish stripes which turned white at maturity and produced normal pollen. The striped leaves had about 50% white area. The last plant resembled its female parent with about 25% white area and had a normal size and height. The reverse cross gave an ear of normal appearance, 80 kernels from the ear were planted directly to the field to give rise to 60 plants, and all were green and tall, indistinguishable from hybrid plants of the same background planted in the same field. Second, a small-scale ear mapping analysis using a limited number of kernels (from a segment of the ear with 10, 10-kernel rows) revealed clonal distribution of variant kernels. Four variants were observed, three of these appeared in the same row, with one and two green sib plants between them, and the last variant was found in the adjacent row at the same relative position at the three others.

In an attempt to understand if the chloroplast was affected in the variant tissue, fresh mature leaves were sectioned with a razor blade and examined under light and phase microscope. Two different leaves were analyzed: one was from one of the three viable striped plants germinated from kernels borne on the variant pistillate plant crossed with the W22/W23 hybrid pollen, and the other was from a normal green plant derived from the reciprocal cross mentioned above. For the striped leaf, most cells had no visible chloroplasts, but a few others had less than three yellow greenish chloroplasts. There were 9 to 13 green chloroplasts per cell in green and yellow greenish tissues of the same leaf. The color of these chloroplasts was not uniform; there was a gradient of color intensity. More light-colored chloroplasts were present in the yellow greenish tissue than the green one. The normal leaf from the reciprocal cross had about 23 chloroplasts, with color intensity similar to the green tissue of the first leaf.

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Compilation of mapping/sequencing results for randomly selected maize cDNAs

--Tim Helentjaris, Ivone Torres-Jerez, Bo Shen, Newton Carneiro, Becky Stevenson, Tom McCreery, Jeff Habben, Brian Larkins, Rob Ferl, Ernie Almira and Chris Baysdorfer

As first described in an article in the Newsletter last year, we have continued our efforts at gene identification/isolation through the analysis of randomly selected cDNAs. The bulk of the effort over the last year has concentrated upon two libraries prepared at Tucson, one from etiolated seedlings and the second from membrane-free polysomes from endosperm. These libraries consist of size-selected cDNAs which are directionally cloned into the ZipLox vector from Gibco-BRL.

After construction, each of the clones is screened for expression pattern by hybridizing a colony lift of several hundred clones at a time with a probe prepared from 1st strand cDNA from each of the two tissues. Those colonies not hybridizing with either probe are characterized as "rarely expressed". Those hybridizing with only one of the probes are denoted as "abundantly expressed and tissue-specific", while those which hybridize with both tissue-derived probes are characterized as "abundantly and generally expressed".

Clones are then submitted to "single-pass" sequencing from the presumed 5' end of the original mRNA. The data is submitted to GenBank by BlastX analysis and subsequently by BlastN if no homologies are identified. Strong homologies indicative of conserved function are usually indicated by BlastX scores of more than 180 and related functions are usually indicated by scores of more than 100. Some clones were also sequenced from the presumed 3' end but the data did not prove useful in identifying putative matches in GenBank.

Probes are then prepared from clones and hybridized to genomic DNA from the Brookhaven RI parents digested with one of three restriction enzymes. Informative clone:enzyme:cross combinations are noted and then all clones with putative identifications from sequencing and others with simple hybridization patterns are also applied to the RI progeny to determine map positions for these cloned sequences.

The results from this analysis to date are presented in the accompanying table, which lists only those clones with sequences indicative of some homology or function. In the future this table will be regularly updated, and will become part of the maize database at Columbia, MO, from which it can be easily accessed. All sequences are also being deposited in GenBank and can be accessed from there. All mapping data are being forwarded to Ben Burr to

be included in the Brookhaven database and will be published annually in the MNL. All clones and data are currently available from Tucson upon request and without restriction. If investigators have requests, such as sequence types or probes in particular genomic regions, they can be communicated to Tucson and we will track the developing database for those requests and forward them as they are discovered. We would like to thank the following companies for providing unrestricted funding to help support this effort along with that from the USDA Plant Genome Program: CIBA-GEIGY (D. Alexander), Monsanto (M. Fromm), Pioneer Hi-Bred International (J. Howard), Rhone-Poulenc (G. Freyssinet), and Sandoz Crop Protection (K. Brunke).

Annotations to the Accompanying Table:

The lab designations for every putatively identified clone are listed in the first column. Those with a first number of "2" or "5", or names beginning with "RSP" or "SPF" originated from endosperm libraries. Those beginning with a "6" originated from a B73 etiolated seedling library. Those denoted by CSU were originally isolated from a mature vegetative tissue library and sequenced by CSU, many of them then mapped subsequently by UAZ.

In the second column, the asterisk defines this sequence as having been first isolated and identified here in maize. Homologies are detected either by BlastX searches of the GenBank at the amino acid level, or if unsuccessful with this approach by BlastN searches at the nucleic acid level. The sequences are grouped roughly according to the function of their putative homologies.

In the third column, a GenBank accession number for one of the high scoring matches is given.

In the fourth column, clones are described as either "abundantly expressed" or "rarely expressed" depending upon whether they exhibit significant signal in a colony hybridization with a 1st strand cDNA probe. The probes they hybridize to are also indicated in this column (i.e. either "Endosperm" or "Seedling").

In the fifth column, a "Complex" pattern indicates somewhat more than three significant hybridizing fragments on a genomic Southern with more than one restriction enzyme. Those clones with "Simple" patterns possess three or less fragments. The designation in the sixth column refers to the map name for the locus(i) detected by this clone. Map locations in the last column are denoted as chromosome(s) and either short arm ("s"), long arm ("L"), or centromeric region ("c").

PUTATIVE IDENTIFICATIONS OF MAIZE CDNAS

Clone Designation	Sequence Homologies	GenBank Accession #	Abund/Rare Tissue-Spec?	Genomic Complexity	Map Number	Genomic Location
2C01H10 6C02G05	triosephospate isomerase - maize	A25501	Abund/S	Simple	UAZ093(TPI)	8L
RSP12,64 5C04A07, D10	sucrose synthase - maize	L01626 susy_maize	Abund/E	Simple	UAZ154(SuS)	9s
5C03G08	Starch-branching enzyme II - maize	L08065	Rare	Simple	UAZ229(SBE2)	6L
5C04B10	*starch synthase precurs (*wx* homol?) - potato	P19395	Rare	Complex	UAZ218(StrS)	4c,3L
RSP33	*alpha-glucan phosphorylase - Ipomoea	phsg_ipoba				
5C04H06	ADPG pyrophosphorylase (*sh2* subunit)	S48563	Abund/E			1,3L,4,5
6C02D08 5C02H07	*UDPG pyrophosphorylase - potato	P19595	Rare	Simple	UAZ194(UDPG)	2L,2L
CSU149	*short-chain alcohol dehydr. (*ts4* homol?)			Simple	CSU149(SADh)	5s
5C04A01	*sorbitol dehydrog homol? - human	Q00796	Rare	Simple	UAZ152(SrDh)	9L
2C02D11 CSU140 6C02B05	glycerald-3-phosphate-dehydr - maize	PQ0178	Rare	Simple	UAZ073(GaPD) UAZ271(GaPD)	4L 3L
5C01C11, H01 02E03, 04E09 CSU152	*glycerald-3-phosphate-dehydr - plant	P08735	Rare	Simple	UAZ190(GaPD)	5L
6C02A09	*alpha ketoglutarate dehydrog - yeast	P20967	Abund/E	Simple		
CSU158 5C01C07	enolase - maize	X55981 P26301	Rare	Simple	CSU158(Enol)	9s
6C02C04	catalase1 - maize	GB-M33104	Abund/ES	Simple	UAZ226(Cat1)	5s
RSP27	catalase 2? - maize	MZECAT2R				1s
6C02C06	catalase3 - maize	GB-X12539	Abund/S	Simple		mitochond?
CSU044	glutathione-S -transferase - maize	X04455				
RSP13,145	*aspartate amino transferase - millet	D14673				
5C04B05	*alanine transaminase - millet	S28429	Rare	Simple	UAZ158(ATas)	5c
5C01C04, 02C04	peptidyl-prolyl c-t isomerase	P21569	AbundE	Simple	UAZ238(PPcl)	5L
FPL0	*protein disulfur isomerase -			Simple	UAZ239(PDsi)	2L,4s
6C02D10	*peroxidase (lignin form enz) - rice	S22087	Rare	Simple	UAZ235(Perx)	2c
CSU160 6C02E12, 02F07	*lipoxygenase L-2 - rice	J03211 P29250	Abund/S	Complex	UAZ225(Lipx)	7L
CSU156	*phenylala ammonia lyase - plant	X16099				
CSU065	*anthranilate synthase II - yeast	M95067				
CSU262	*6-phosphogluconate dehyd - plant	X58719				
5C04A02, A04, C06 CSU155	pyruvate phospate dikinase - maize	M58656	Abund/E	Simple	UAZ153(PPDk)	8L,6L
CSU324	*citrate synthase - animal	M21197				

CSU198	*malate dehydrogenase - animal	M29463				
CSU077	*malate dehydrogenase - bacteria	M95069			CSU077(MDh)	1
CSU016	NADP malic enzyme - maize	J05130		Simple	CSU046(ME)	3L, 6
SPF1,11 CSU269	*nucleoside diphosphate kinase - animal	M65037		Simple	UAZ031(NDpK)	1c
SPF3,4	*nucleoside diphosphate kinase - Spinacia	S24165		Simple	UAZ091(NDpK)	7c
CSU074	ferredoxin - maize	M73828				
5C01E01	ubiquitin precursor - maize	S04863	Abund/E	Simple	UAZ247(Ubiq)	4L
CSU204 2C06C11 5C02F11, 04D06	*ubiquitin-conjugating enzyme E2 - wheat	P16577	Rare	Simple	UAZ102(UCE2)	6S
2C01H08	*acyl-carrier protein - plant	S19832		Simple	UAZ099(ACP)	10c
CSU136, 205 5C02A01	phospholipid transfer protein - maize	J04176 P19656	Rare		CSU136(PITP)	10s
CSU257	*plastocyanin - plant	Y00704				
CSU229	*16kd O2 evolving factor - plant	X05512				
CSU117	*Chloroph A/B-binding protein - plant	M63931				
CSU102	*Chloroph A/B-binding protein - plant	X13909				
CSU066	*Chloroph A/B-binding protein - plant	D00642				
CSU071	Chloroph A/B-binding protein - maize	X14794		Complex	CSU071(CAB)	1L, 6s
6C02C05	Rubisco, Large Subunit - maize	GB-V00171	Abund/S	Simple		Chloroplast
6C02A04	*Arabidopsis ORF, potent chloropl-target prot?	X71878	Rare	Simple	UAZ200	7L
CSU026	ATP/ADP Translocator - maize	X02842		Simple	CSU026(ATPT)	5L
5C04B04	*ATP synthase alpha mitoch homol? - yeast	P07251	Rare	Simple	UAZ144(ATPS)	4L
5C04E07	ATP synthase beta mitoch - maize	P19023	Rare	Complex	UAZ243(ATPS)	3L,6L,8s
CSU030	*vacuolar ATPase, proteolipid sub - plant	M73232		Simple	CSU030(ATPs)	3s
6C02E07	*vacuolar ATPase, nuc. bind. sub - barley	L11862	Abund/E	Complex	UAZ223(ATPs)	9c
5C02E08	*vacuolar membrane proton pump (PPase) - Arabidopsis	P31414	Rare	Complex	UAZ280(PPas)	4s,9L
CSU125	*carbonic anhydrase - plant	X52558				
5C01A12	*40s ribosomal prot S6 homol? - tobacco	S25550	Abund/E	Complex	UAZ119(S6)	7L,8L,9L
5C01A09, 04G09 CSU034	*40s ribosomal prot S8 homol? - human	P09058	Rare	Simple	UAZ115(S8)	4L
6C02A02 5C02F12	40s ribosomal prot S11 - maize	P25460	Abund/S	Simple	UAZ251(S11)	2s,3L,6L,8c, 10L
2C01G06	*40s ribosomal prot S21	P05764		Complex		
CSU28	*40s ribosomal prot S22 - animal	M34706				
5C02D01	40s ribosom prot S27A (ubiq. fus. prot. 9)	JS0657	Rare	Complex	UAZ249(S27A)	1c,3c,8c,8L
5C01A05	*40s ribosomal prot S28 - rat	P25112	Rare	Simple	UAZ146(S28)	1s,1s
5C04D11	*60s ribosomal prot L5 - yeast	P15125	Rare	Simple	UAZ189(L5)	3
RSP81	*60s ribosomal prot L7 - human	rl7_human				2,10
5C01D03, 04F09 02H06	*60s ribosomal prot L10e homology - yeast	P15826	Rare	Complex	UAZ198(L10e)	3L
CSU245	*60s ribosomal prot L14 homol?- animal	X06222				
CSU036 5C04B03, G01	*60s ribosomal prot L19 homol?- human	P14118	Rare	Simple	CSU036(L19) UAZ157(L19)	3L,4L,5c
5C01A04	*60s ribosomal prot L24 homol? - Nicot.	M87838	Rare	Complex	UAZ270(L24)	4L
CSU236	*translation initiation factor-2 - bacteria	X04399				
6C02E11	*translation initiation factor eiF-2 - human	L19161	Abund/S	Simple	UAZ224(TIF2)	7L
RSP35,37 5C03C06, 04H09 CSU116,226	*elongation factor 1-alpha - Arabidopsis	P17786	Rare	Complex	UAZ220(EF1α)	6L,8
5C04C04	*elongation factor 1-gamma - Trypanosoma	L17307	Rare	Complex	UAZ161(EF1γ)	6L,9s,3L,4L
6C02G11	* seryl-tRNA synthetase - yeast	P07284	Abund/ES	Complex	UAZ236(StRS)	9L,2s
5C01B12	*aspartyl-tRNA synthetase alpha chain - rat	P15178	Rare			
5C04F01	RIP-3 - maize (*rip1*)	P25898	Abund/E	Simple	UAZ193(RIP3)	8c
5C04B01	RIP-9,	P25892	Abund/E	Simple	UAZ156(RIP9)	3L
2C01B05	19kd alpha-zein 19A2	zizma2	Abund/E	Complex	UAZ049(19αZ)	2s,4s
2C06H03 SPF19,24	19kd alpha-zein A20	zizm2	Abund/E	Complex	UAZ068(19αZ)	4s
2C01B06	19kd alpha-zein A30	zizm3	Abund/E	Complex	UAZ049(19αZ)	4s
2C06H04	19kd alpha-zein 19B1	zizmb1	Abund/E			
5C01H08	19kd alpha-zein 19C2	P06677	Abund/E			
SPF6,28 5C02A08	19kd alpha-zein 19D1	P06678	Abund/E	Simple	UAZ005(19αZ)	1c
5C03G02 SPF20	19kd alpha-zein PMS 2	P24450	Abund/E	Complex	UAZ149(19αZ)	4s
2C06D05	19kd alpha-zein ZG31A	S21965	Abund/E	Complex		
5C04D05	22kd alpha-zein pML1	X14334	Abund/E			
5C03B06	22kd alpha-zein PZ22.1	P04700	Abund/E	Complex	UAZ185(22αZ)	4s
5C02C08, 02C05	22kd alpha-zein PZ22.3	P04698	Abund/E			
SPF10	22kd alpha-zein ZA1	B22831	Abund/E			
2C07D02 SPF8 5C02A02, 04E12 04G06	16kd beta-zein ZC1	P06673	Abund/E			
RSP80,92	gamma-zein	mzezzg_1	Abund/E			
5C04B07	*glutenin homol? - wheat	A30843	Rare	Simple	UAZ230(Glu?)	4c,6L
6C02H08	*cathepsin B - wheat lysosomal protease?		Rare	Simple	UAZ234(Pros)	2c
CSU005	*thiol protease - plant	X15732		Simple	CSU005(Pros)	7
CSU096	*thiol protease inhibitor - plant	M29259		Simple	CSU096(Prol)	3
5C03B04 RSP11,21	trypsin/Factor XIIA inhibitor - maize	P01088	Abund/E	smear		

RSP96,111 5C04A08	*chymotrypsin inhibitor - barley	A29537	Rare	Simple	UAZ232(Ctl)	2L
5C02D08	*chloropl. ATP-depend. proteinase - pea	P31542	Rare	Simple	UAZ242(Pros)	10L
5C02A05	*proteasome C9 endopeptidase. - human	P25789	Rare	Simple	UAZ237(Pros)	6c,9s
RSP75	proteinase inhibitor, wound-induc? - potato	potinhwi_1				
5C02G08	*alkaline extracell protease homol - yeast	P09379	Rare			
2C01C07	*oligopeptidase A - E . coli	A42298		Simple	UAZ100(Pros)	10s
6C02E06	*chaperonin 10 (chloroplast) - spinach	Q02073	Rare	Simple	UAZ222(Chap)	4L
6C02F11	*actin AC1 - carrot	J01238	Abund/S	Complex	UAZ233(Act)	8c,6c,7L
CSU272	a-tubulin 1 - maize	X15704				1
6C02D05	a-tubulin 3 - maize	S28429	Abund/E	Complex	UAZ201(αTub)	5s
5C04C03, 04F07	Ser-rich protein		Rare			
6C02C10	Ser-Lys-rich protein		Abund/S			
5C04B11	Ala-repeat protein		Rare	Complex	UAZ159(AlaR)	5c
6C02B04	Lys-Glu-rich prot		Rare	Simple		
5C02D07	Pro-rich prot					
6C02F01	Pro-Pro-rich prot (extensin-like)		Abund/E	Complex		
6C02F03	Pro-Val-rich prot (extensin-like)		Abund/E	Simple	UAZ192(PrVa)	7L
6C02D09	*glycine-rich prot - tomato	X55691	Rare	Smear		
CSU208 6C02G01, 02G12 5C02G05	glycine-rich prot, ABA-inducible - maize	P10979	Abund/ES	Simple		
5C04A11	Bt1precursor - maize	P29518	Rare	Complex	UAZ155(Bt1)	10c
2C01F03	Bt-homolog? - maize	mzebtia		Complex	UAZ025(Bt?)	2,5,6,8
5C02F05, 02H10	*mitochondral carrier protein - yeast	P32331	Rare	Complex	UAZ282(MCP)	1c
5C04C02	*TDR3 homeotic (MADS) - Tomato	X60756	Rare	Complex	UAZ231(MADS)	9L
CSU137	*MADS box - plant	X53579		Simple	CSU137(MADS)	1s,5s
5C04A03	*CA-depend. protein kinase - carrot	P28582	Rare	Complex	UAZ130(PKas)	1L,4c,5s
5C04G11	*CA-depend. protein kinase - carrot	L14771	Rare	Complex	UAZ197(PKas)	6c,6s
CSU231	*protein kinase - yeast	M13971				
CSU252	*protein kinase - yeast	M76585				
5C02A07	*protein kinase (tyr-ser-thr) - Arabidopsis	L07428	Rare	Simple	UAZ252(PKas)	4L,8L
5C04C05	*phosphoprotein phosphatase - Drosophila	S29396	Rare	Complex	UAZ244(PpPs)	6L,8L
CSU108	*GTP-binding protein - plant	M35520			CSU108(GTPB)	5s
5C03G12	*GTP-binding protein SAR1 - Arabidopsis	Q01474	Rare	Simple	UAZ151(GTPB)	1L
5C04D03	*GTP-binding protein (dev-regul) - mouse	D10715	Rare	Simple	UAZ245(GTPB)	7L
2C07F04	*signal recognition particle receptor - dog	dogsrpr_1		Simple	UAZ008(SRPR)	3L
CSU150	*RNA pol II - yeast	M15693		Simple	CSU150(RPI2)	5s,6s
CSU017	*31kd ribonucleoprot - plant	X53942		Simple	CSU017(RnP)	2L,7
5C02D05	*U1 small nuclear ribonucleoprot? - human	M18465	Rare	Simple		
6C02F05	*DNA/RNA-binding protein - Drosophila	P13469	Abund/ES			
FLP6	RNA-binding protein - maize			Complex	UAZ240(RNAB)	1L,5L,6L
2C06G06	CAAT-box-binding protein - maize	zmnfyb		Simple	UAZ007(CAAT)	7s
5C01A07	*TAT-binding protein homol? - yeast	L01626	Rare	Simple	UAZ118(TATB)	4L
5C04C08	*myb transform protein homol.? - mouse	A28013	Rare	Complex	UAZ216(myb?)	4c
5C04D07	*putative transcription factor?	P27426	Rare	Simple	UAZ207(TF?)	2s
6C02C08	*histone H2A - pea	P25470	Abund/ES	Complex	UAZ221(H2A)	7L
CSU285 5C04D12	histone H2B.2 - maize	P30756	Rare	Complex	UAZ228(H2B)	2L,10c,4L, 1L
5C03H09	*histone H3.3 - Arabidopsis	S24346	Abund/ES	Simple	UAZ248(H3)	1s,5L
5C02C03	histone H4 - maize	A25642	Rare	Simple		?
CSU209	*GOS 2 homolog - plant	X51910				
5C04A12	*early nodulin - soybean	D13506	Rare	Simple	UAZ227(ENod)	6c
CSU146	*cell cycle protein CDC48 - yeast	X56956		Simple	CSU146(cdc)	6c
CSU052 RSP30	*DNA J protein homolog - yeast	X56560			UAZ109(DNAJ)	3s
CSU012	CIN 4 retroelement - maize	Y00086		Complex	CSU012(CIN4)	2s,4L
5C04E06, 04G04	Ac transposase homology - maize	X01380	Rare	Complex	UAZ285(ACTr)	1c,8L,8L,5c
CSU190	*auxin-induced gene - plant	X56267				
6C02E02	*male sterility gene homol? (cholesterol dehydrog?) - Arabidopsis	X73562	Abund/E	Simple	UAZ195(MS?)	4c
5C02D11	*chloropl. 17.6kd heat shock - chenopod.	P11890	Rare	Simple	UAZ171(HtSh)	4L
5C04F02, 04H02	18kd heat shock - maize	P24631	Abund/E	Simple	UAZ210(HtSh)	3s
5C04F03	*70kd chloropl heat shock homol?	Q02028	Rare			
5C04D01	*70kd mitoch. heat shock - Phaseolus	Q01899	Rare	Complex	UAZ205(HtSh)	5s,1L
5C04H04	*80kd heat shock prot (chloropl) - spinach	M99565	Rare	Simple	UAZ219(HtSh)	5s
CSU274	*83kd heat shock - plant	M62984				
CSU019	*cold-regulated gene - plant	M60733		Simple	CSU019(Cold)	4s
2C02A04 CSU206	*barley ORF (Fe-deficiency induced)	bylids3		Simple	UAZ080(FeDf)	6s
5C01G10	*salt stress-induc hydrophob - wheatgrass	U00966	Abund/ES	Simple	UAZ250(NaCl)	10L
5C03H11	*rice ORF 0962A (ribonucl reduct M1 homol?)	D15619	Abund/ES	Simple	UAZ186(REST)	5L
5C02A11	*rice cDNA 2022A	D16016	Rare	Complex	UAZ274(REST)	2L,1c
5C04B06	*YBL0507 - yeast cDNA aminogly-acetyl transf homology?	Z23261	Rare	Complex	UAZ241(YEST)	2L,7L
5C01C06	*vegetative-specific protein - slime mold	P14327	Abund/E	Simple	UAZ246(VSP)	4c
5C04D09	*p23 tumor-specific transpl. antigen (highly basic protein) - human?	A44367	Rare	Simple	UAZ208(TSTA)	1L
5C02B05	*human orf HUMRSC399-1	D13642	Rare	Simple	UAZ275(HEST)	5s
CSU064	*brain specific prot., 14-3-3 Protein, Tau ch. - human	J03868		Simple	CSU064(BSPτ)	1L,2,8
6C02C02	*retinoblastoma-associated protein - human	P28749	Abund/SE	Simple	UAZ191(RAP)	2L

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Stocks and new factors

--G. F. Sprague

Due to poor health I have given up my field plantings. I have attempted to provide seed to the Coop of all my stocks which might have the most general interest. The following notes refer to several unfinished items which, hopefully, may be of interest to someone.

Dotted stocks. The dotted stocks involved in these studies involve *Dt1, Dt2, Dt3* and *Dt4* from the Maize Coop and *Dt6, Dt7, Dt(a), Dt(b), Dt(c), Dt(d), Dt(e), Dt(f), Dt9* and *Dt(n)* derived from the earlier virus studies. *Dt(b)* may not be workable as it exhibits only 1 or 2 small dots per kernel. There are a few other instances where dotting appears to involve either the *c* or *r* alleles. These are not considered here.

Linkage relations for *Dt(a)* through *Dt(n)* have not been established. Two approaches have been used for allelism testing. *Dt/Dt* combinations have been produced and advanced to F2. Failure of segregation for non-dotted kernels would indicate the two parental types were alleles. On this basis none of the (a)-(n) parental types were allelic. Diallel crosses were also made among their true breeding non-dotted counterparts. Essentially all produced non-dotted F1 kernels. However when the non-dotted types were advanced to F2 they exhibited *Dt:dt* segregation. Proportions were quite variable with the *dt* class often exceeding 50%. With selection of the more heavily dotted kernels homozygous *Dt/Dt* types can be recovered in F3 or later generations.

These generalizations suggest some special types of reactivation or transposable element involvement. If transposable element or elements are involved they must differ from the typical transacting types such as *Ac, Sm, Uq*--as *dt/dt* crosses are non-dotted in F1.

After stabilization the *Dt*s recovered from *dt/dt* crosses may exhibit one of the parental dotting patterns (i.e. the *Dt6* pattern from the *dt1/dt6* cross) with or without its characteristic linkage pattern. This suggests either that patterns are conditioned by factors other than the *Dt* allele or the *Dt* allele has been transposed to a new site. The linkage relations of *Dt(a)* through *Dt(n)* are still unknown. *Dt1, Dt2* and *Dt6* are the best candidates for further exploration of this phenomenon and tests are underway.

Relevant stocks have been given to the Coop.

A new dwarf. A rosette type dwarf was found and tested against other recessive rosette dwarfs. No allelism was observed. In tests performed by B. O. Phinney this dwarf does not respond to gibberellic acid. Preliminary tests suggested it was allelic to *an1*. Test crosses were made with the *an1-bz2-6923* deficiency stock from the Coop. The F1 seedlings were rosette dwarfs. However, this test was inconclusive as the deficiency stock shed poorly, increasing the possibility of contamination. Following a severe aphid infestation the F1 plants developed a soft rot and no progeny were obtained.

A gametophyte factor on chromosome 6. In the course of testing for allelism among the Coop's collection of glossy mutants, one cross exhibited an unexpected *Y:y* segregation. The numbers observed suggested either a gametophyte factor or a second *y* locus, similar to *y1*, conditioning the near absence of carotenoid pigments. F3 progeny tests supported the gametophyte assumption. The mean percentage of *y* kernels or segregating ears was 40.6. Other presumed instances of gametophyte factors include *su* and *pr*. These have been given to the Coop.

Green corn. Seed was obtained from a former County Extension Agent now located in Oklahoma who has a hobby of maintaining specialty corn. This type he calls his John Deere corn. The corn has aleurone which is an off color shade of green. A sample was supplied to Dr. E. D. Styles who indicated (personal communication) that the color was definitely in the aleurone, and that the anthocyanins are the normal acylated cyanidin glycosides present in *Pr* aleurones with no other flavonoids in easily detectable concentrations. Tests indicated the presence of an additional pigment, but this could not be resolved due to a lack of a colorless counterpart. Further work on identification will require additional material.

In crosses of "green" with the aleurone tester stocks the green color is so diluted in the F2 kernels as to make separations difficult and questionable.

Silencing of *restorer-of-fertility* genes of cms-S

-- S. Gabay-Laughnan and J. R. Laughnan

The mitochondrial alteration causing S-type cytoplasmic male sterility (cms-S) in maize can be overruled by certain nuclear genes called *restorer-of-fertility* (*Rf*) genes. The mode of restoration of these *Rf* genes is gametophytic in the cms-S system meaning that they act postmeiotiallly. Among the many spontaneously occurring *Rf* genes that we have identified is a class we refer to as pseudorestorer. When "fertile" plants carrying a pseudorestorer gene are crossed as male parents onto *rf rf* cms-S (male-sterile) testers, or onto male-fertile isogenic maintainer (*rf rf*) plants with normal cytoplasm, there is no seed set on the ears. Because this class of "restorer" gene produces nonfunctional pollen, we have given it the symbol *Rf-nf* (MNL 63:122, 1989; MNL 63:122-123, 1989). To date, seven independently occurring spontaneous revertants arising in four inbred line-cytoplasm combinations have been classified as *Rf-nf* genes.

In the course of studies on the allelic relationships of the *Rf-nf* genes we found that crossing *Rf-nf* plants by unrelated, nonrestoring inbred lines yields F1 plants that produce functional pollen. As part of our effort to understand this phenomenon the F1 plants are being successively crossed as male and female parents with each of the two inbreds that constitute the F1. In the course of these crosses we were able to compare the performance of an *Rf-nf* gene of an F1 (*Rf-nf/rf*) plant crossed both as male and as female parent. We have observed differential effects on pollen production in the backcross progeny depending on whether the *Rf-nf* -carrying F1 plant was crossed as the male or female parent. Crosses of the F1 plants as pollen parents often produced progeny segregating sterile plants and, in some cases, all sterile progeny, whereas such crosses should produce all fertile progeny. Crosses of these same F1 plants as female parents gave the expected fertile and sterile plants. Since the backcross progeny in both crosses have the same nuclear constitution the difference cannot be explained by the failure of the *Rf-nf* gene to express in a particular nuclear background. We have only recently begun studying this phenomenon and have hypothesized that the differential behavior of the *Rf-nf* genes, depending on whether they were transmitted through the maternal or paternal parent, is due to imprinting.

As a control, we have crossed ears on plants carrying functional *Rf* genes in inbred nuclear backgrounds, both the standard

Rf3 gene and other *Rfs* that arose spontaneously in our hands, by unrelated inbred maintainer pollen. The fertile *Rf*-carrying F1 plants were crossed both as male and female parents and exhibited the expected results; crosses of these F1 plants as pollen parents produced only fertile offspring. Since there is no evidence of silencing of functional *Rf* genes, our *Rf-nf* genes may represent a unique system in which to study gene silencing in maize.

The unexpectedly sterile plants resulting from crosses of *Rf-nf*-carrying F1 plants as pollen parents should all carry the *Rf-nf* allele even though it is not being expressed. We have begun testing such plants to determine if the "imprinting" can be erased by passage of the silent *Rf-nf* gene through a sporophytic generation and have found that the "imprinting" persists. The resulting sterile plants (now only half of which are expected to carry the *Rf-nf* allele) have been crossed again by maintainer pollen and were scored in our 1993 summer nursery. There is no indication that a second passage of the silenced *Rf-nf* genes through a sporophytic generation has erased the imprinting. Since we have carried the crossing of the silenced *Rf-nf* genes by maintainer pollen as far as we reasonably can, we will now try a different approach. Sterile plants resulting from the backcross of an *Rf-nf*-carrying F1 plant as pollen parent will be crossed as female parents with pollen from the F1 plants. These sterile plants carry a silenced *Rf-nf* gene. Will additional copies of the *Rf-nf* gene also be silenced?

A phototoxin in maize leaves, disease resistance?

--Robert Tuveson and Dale M. Steffensen

Recently, there has been some interest in light activated compounds, so-called phototoxins. We decided to extract maize leaves to see if any such compounds could be identified using a bacterial assay. After several trials we settled on extracting maize leaves with 70% methanol in a Waring blender going full speed for 2 min.

The first successful response came from extracts of Fr 632 *Ht1* using the leaves of 5-6 leaf maize plants. The standard procedure has been to grind 10-20 grams of leaves without the sheath. The methanol mixture is 70% methanol, 30% water with one drop of beta-mercaptoethanol per 10 ml. The grinding ratio was 1 g of tissue per 10 ml of 70% methanol. After grinding, the mixture was spun in a Sorvall centrifuge for 10 min at 5,000 rpm. The liquid was taken off with a Pasteur pipette and spotted immediately onto filter discs, held by pins. The remaining liquid was stored at -20 C. Storage for several weeks did not seem to reduce activity.

Cultures of *Escherichia coli* strain RT 7 rfa were grown the night before on Petri plates. Next morning the dry filter paper discs with the leaf extracts were allowed to absorb and diffuse on the Petri plates for 1 1/2 hours. A control compound, 8-MOP, was also spotted and blotted. Duplicate plates were always made up as dark controls. After the absorption from the discs was completed, and the paper removed, the plates were irradiated with UV-A (315-400 nm) for one hour. The other half of the plates (dark controls) were not. After the hour all of the plates were put at 37 C to grow until the next day, when they were examined. The UV-A 8-MOP control usually gave lethal circles, 25 mm in diameter. The positive genotypes with 0.060 ml of methanol extract had rings of killing nearly that large. Measurements of these diameters gave the bioassay a semi-quantitative measure. A typical experiment is shown in Figure 1.

The initial response was from a *Ht1* genotype. Other maize

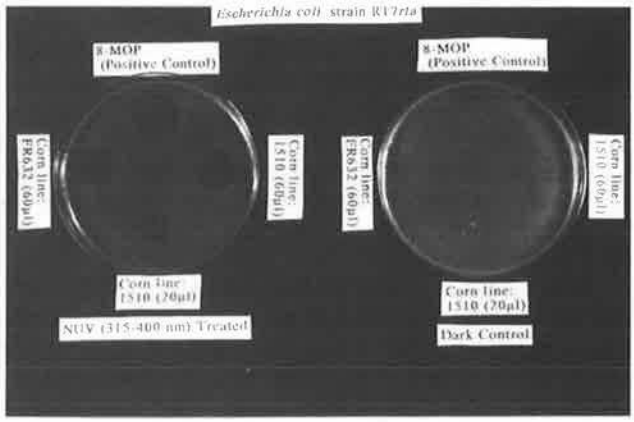

Figure 1. Petri dishes with *E. coli* cultures spotted with methanol extracts of maize leaves. The plate on the left received UV, the right did not. FR632 is an inbred from Illinois Foundation Seed. 1510 is a Mexican flint. The clear circles are where the phototoxin killed the bacteria.

stocks we tried were negative or had slight responses. To do a proper experiment we obtained a large series of inbreds that were *Ht1* converted and *ht1* from Illinois Foundation Seed. As seen in Table 1 below there was no correlation between *Ht1* and reactivity.

Table 1.

Genotype	Reactivity	Diameter of reactive zone (mm)
O7A *ht1*	-	
O7A *Ht1*	-	
M14 *ht1*	+-	6
M14 *Ht1*	-	
FR Mo17 *rhm, ht1*	+	9
Mo17 *Ht1* (Callahan)	-	
W22G *ht1*	-	
W22G *Ht1*	+	10-11
B37 *ht1*	+	9
B37 *Ht1*	+-	3
Oh51A *ht1*	-	
Oh51A *Ht1*	+	8
B68 *ht1*	-	
B68, *rhm, Ht1*	+	16
A619 *ht1*	+	9
A619 *rhm, Ht1*	-	
A635 *ht1*	-	
A632 *Ht1*	-	
MS1334 *ht1*	-	
MS1334 *Ht1*	-	

The most reactive genotype was a B68 inbred. B68 is known to have a high content of DIMBOA. For this reason we obtained seed from K. Simcox that are DIMBOA negative (*bx/bx*). In Table 2 *bx/bx* leaves are quite reactive in the bioassay. We are left without leads as to the role of this phototoxin in the plant. Finally, in mature plants (B37 *ht1*) we determined that the phototoxin was evenly distributed. The oldest leaves had the same reactivity as the youngest leaves on a per gram basis.

Table 2.

Genotype	Reactivity	Diameter of reactive zone (mm)
bx/bx (Simcox)	+	18
Bx/Bx	+-	3
bx/bx (#16, Simcox)	+	20
B68 *Ht1*	+	16
B68 *ht1*	-	
B37 *Ht1*	+-	3
B37 *ht1*	+	9

A number of varied experiments had been planned, including identifying the compound(s) by chemical methods. However the

senior author (RWT) met an untimely death more than a year ago, so the studies were ended.

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Three-point linkage data for *su1*, *lw4*, and *gl4* on chromosome 4
--Philip S. Stinard

The results of a three-point linkage test for *su1*, *lw4*, and *gl4* on chromosome 4 are reported in Table 1. The linkage test was set up as a modified backcross as indicated in Table 1. Since *lw3* and *lw4* are a duplicate factor pair, the backcross to *lw3* was necessary in order to score for lemon-white in the next generation. Kernels from the backcross ears were planted in the field, the resulting plants self-pollinated, and the ears scored for *su1* and *lw4*. Kernel samples from each ear were planted in the sand bench, and the resulting seedlings were scored for *lw4* (for confirmation of kernel phenotype) and *gl4*. The following linkage relationship was established: *su1* - 11.7 - *lw4* - 7.0 - *gl4*. The distance between *su1* and *gl4* (18.7 cM) is greater than that reported on the 1993 linkage map (15 cM), but is within the standard error of that reported in a previous linkage study of *su1*, *lw4*, and *gl4* (MNL 65:17-18, 1991; 21.0 cM). When the data from this study are combined with the previous data (MNL 65:17-18, 1991), one obtains the following linkage relationship:

Table 1. Three-point linkage data for *su1 - lw4 - gl4*. Testcross: *Su1 Lw4 Gl4 lw3* X (*su1 Lw4 gl4 Lw3 / Su1 lw4 Gl4 Lw3*).

Reg.	Phenotype	No.	Totals
0	*su1 + gl4*	68	
	+ lw4+	72	140
1	*su1 lw4+*	9	
	++ gl4	10	19
2	*su1 + +*	4	
	+ lw4 gl4	7	11
1+2	*su1 lw4 gl4*	0	
	+++	1	1

% recombination *su1--lw4* = 11.7 +/- 2.5
% recombination *lw4--gl4* = 7.0 +/- 2.0
% recombination *su1--gl4* = 18.7 +/- 2.5

% recombination *su1--lw4* = 11.3 +/- 2.0
% recombination *lw4--gl4* = 8.2 +/- 1.7
% recombination *su1--gl4* = 19.9 +/- 2.5

The data were scaled to fit the current linkage map; the suggested map revision is:

```
--su1----------lw4---------gl4--
  47           56          62
```

Three-point linkage data for *pr1*, *lw3*, and *v2* on chromosome 5
--Philip S. Stinard

The results of a three-point linkage test for *pr1*, *lw3*, and *v2* on the long arm of chromosome 5 are reported in Table 1. The linkage test was set up as a modified backcross as indicated in Table 1. Since *lw3* and *lw4* are a duplicate factor pair, the backcross to *lw4* was necessary in order to score for *lw3* in the next generation. Kernels from the backcross ears were planted in the field, the resulting plants self-pollinated, and the selfed ears scored for *pr1* and *lw3*. Kernel samples from each ear were planted in the sand bench, and the resulting seedlings were scored for *lw3* (for confirmation of kernel phenotype) and *v2*. The following linkage relationship was established: *pr1* - 14.4 - *lw3* - 37.9 - *v2*. The distance between *pr1* and *v2* (52.4 cM) is longer than that reported on the 1993 linkage map (40 cM). The placement of *lw3* 14 cM distal to *pr1* is consistent with the F2 data of Tulpule (Am. J. Bot. 41:294-301, 1954), which gives a *pr1* - *lw3* distance of 19 cM. The following map revision is suggested:

Table 1. Three-point linkage data for *pr1 - lw3 - v2*. Testcross: (*Pr1 lw3 V2 Lw4 / pr1 Lw3 v2 Lw4*) X *Pr1 Lw3 V2 lw4*.

Reg.	Phenotype	No.	Totals
0	*pr1 + v2*	117	
	+ lw3+	104	221
1	*pr1 lw3+*	23	
	++ v2	31	54
2	*pr1 + +*	74	
	+ lw3 v2	84	158
1+2	*pr1 lw3 v2*	5	
	+++	5	10

% recombination *pr1--lw3* = 14.4 +/- 1.7
% recombination *lw3--v2* = 37.9 +/- 2.3
% recombination *pr1--v2* = 52.4 +/- 2.4

```
            lw3
--pr1-----ys1----------------got2-------v2--
  67       75                96          107
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URBANA, ILLINOIS
USDA/ARS/MWA
AMES, IOWA
Iowa State University

o12* and *cp2* are allelic to *dek7
--Philip S. Stinard and Patrick S. Schnable

We reported the location of *o12* to the short arm of chromosome 4 in last year's MNL (67:10, 1993). Because of the similarities in phenotype of *o12* and *dek7* (sugary/shrunken endosperm, aleurone mosaicism, striate seedlings), which is also on 4S, allelism tests were conducted between these two mutants. Nonmutant kernels from selfed segregating ears of *dek7* and *o12* heterozygotes were planted in our 1992-1993 winter nursery, and the resulting plants were intercrossed between families. Several segregating ears were obtained, indicating allelism between *o12* and *dek7*.

Because of the similarities in phenotype between *cp2* (reportedly on chromosome 7) and *dek7*, allelism tests were conducted between these two mutants. Nonmutant kernels from selfed segregating ears of *dek7* and *cp2* heterozygotes were planted in the 1993 Urbana summer nursery, and allelism tests were conducted as described above. Several segregating ears were obtained, indicating allelism between *cp2* and *dek7*. (Linkage tests of *cp2* with *wx* T7-9(4363) (7 ctr.; 9 ctr.) demonstrated independent segregation, also indicating that the original placement of *cp2* to chromosome 7 was in error.) Allelism tests of *cp2* with *dek5* (on 3S), which also has a phenotype similar to that of *cp2*, were negative.

Of the three mutants, *cp2* has precedence (Neuffer et al., The Mutants of Maize, 1968), so the recommended nomenclature for *o12* and *dek7* is *cp2-o12* and *cp2-dek7*.

New alleles of *et2* and *su3*
--Philip S. Stinard and Patrick S. Schnable

Allelism tests are continuing on endosperm mutants arising

from *Mutator* populations. This report describes new alleles of *et2* and *su3* that arose in *Mutator* populations.

An etched endosperm mutant was found segregating on the self-pollinated ear of a *Mu1* outcross plant (91g6290-26). When planted in the sand bench, etched kernels gave rise to albino seedlings. Since the mutant *et2* has a similar phenotype, allelism tests were conducted between these two mutants in the 1993 Urbana summer nursery. These tests gave positive results. We name the new *et2* mutant allele *et2-91g6290-26.*

A homozygous viable sugary/shrunken endosperm mutant was found segregating in low frequency on the self-pollinated ear of a plant (89-1303-18) grown from the cross *wc1 bm4* X (*Wc1 Bm4 / wc1 bm4* [*Mu*]). Allelism tests with *ae1*, *bt1*, *bt2*, *sh2*, and *su1* proved negative. Positive allele tests were obtained with *su3* in our 1991-1992 winter nursery, and were replicated in our 1992 summer nursery. We name this new *su3* allele *su3-89-1303-18. su3-89-1303-18* shares with the *su3* reference allele reduced frequencies of transmission and segregation. We are investigating the possibility that *su3* is a duplicate factor pair.

The new, improved TB-9Lc

--Philip S. Stinard and Patrick S. Schnable

The original TB-9Lc stocks were time consuming to work with because no endosperm markers were uncovered by this translocation, making selection of known hyperploids for use in mapping experiments difficult. In the absence of homozygous TB-9Lc lines, each plant in a segregating TB-9Lc population had to be examined for pollen sterility, and those plants which showed slight pollen abortion (approximately ten percent in a year with good expression) had to be test crossed to a 9L seedling marker (such as *v1* or *Bf1*), and the progeny seedling tested in order to determine with certainty which plants carried the translocation.

An innovation was made when the *Wc1* (dominant pale yellow endosperm) mutation was transferred to the translocated 9L arm, allowing the uncovering of the recessive *wc1* allele (J. B. Beckett, Locating recessive genes to chromosome arm with B-A translocations, in The Maize Handbook, ed. by Freeling and Walbot, Springer-Verlag, 1993) in hypoploid endosperms. In this method, plants homozygous for *Wc1* and segregating for TB-9Lc are crossed onto *wc1 Y1* (yellow endosperm) standards. If the male parent in a cross carries TB-9Lc, the resulting ears segregate for large pale yellow (balanced and hyperploid endosperms) and smaller yellow kernels (hypoploid endosperms). Barring heterofertilization (a relatively rare event), the yellow kernels will have hyperploid embryos and thus carry the translocation. Crosses in which the male parent does not carry TB-9Lc will produce ears with only pale yellow kernels.

In order to improve the selection for homozygous *Wc1* plants carrying TB-9Lc, we have taken advantage of the close linkage of the *wx1* locus on the short arm of chromosome 9 to the TB-9Lc breakpoint (see 1993 linkage map). We have crossed a homozygous *wx1 Wc1* line to a TB-9Lc *Wc1* line for two generations, generating homozygous *Wc1* ears segregating for *wx1* kernels. On such ears, the majority of *Wx1* kernels should carry TB-9Lc since TB-9Lc is in close coupling with the *Wx1* allele. Of 44 plants grown from *Wx1* kernels so far, 33 (75%) have carried TB-9Lc. After further testing and increase, our *Wx1* 9-B *Wc1* B-9 (TB-9Lc) and *wx1 Wc1* lines will be made available for distribution from the Maize Genetics Cooperation Stock Center.

VICTORIA, BC, CANADA
University of Victoria

Notes from a corner in Victoria

--E. D. Styles

A unique *R1* allele? Although Stadler and Fogel initially described four different classes of *R-r* alleles, the only class that seems to have been easily distinguished from the rest was 'Class 4' (or 'Group D') *R-r* alleles, and that on the basis of a specific response to a plant color modifier. In later papers Stadler made a point of emphasizing that because of differences in expression in different tissues, it was virtually impossible to arrange different *R-r* alleles in any sort of continuous series. True as this may be, for convenience it is sometimes necessary to attach labels to particular *R-r* alleles that have unique enough features that allow them to be distinguished from other *R-r* alleles. One such *R-r* allele came into my stocks through the generosity of Ed Coe. In most *pl* stocks that I grow in Victoria, this allele determines green or near green anthers. It is not a 'Group D' allele, but sometimes I have mislabelled it as '*R-g*' until for whatever reason I have crossed it with *Pl*, when it determines purple anthers as dark as any determined by the more predictable *R-r* alleles. As this allele seemed worthy of a special designation, and as it is in fact an '*R1*' allele, I call it, as you may by now have guessed, '*E. Coe R1*'. It is worth noting that the best expression of this allele in the anthers is 'restricted' to backgrounds that carry *Pl*. Any similarity to the terminology used by those working with inferior organisms is of course purely incidental.

***Whp* vs. *whp*.** As with many others I am sure, I am trying to get parallel lines of stocks that, except for differences at the *whp* locus, are similar with respect to allelic variations at other flavonoid loci. The original reason for this attempt goes way back to when Ed Coe and I had the rather naive hope of being able to characterize the differences in expression of flavonoid genes in different inbred lines in terms of the known flavonoids. Apart from the fact that variations in the glycosidic patterns were more complex than thought, I at least had not recognized the difficulties in distinguishing possible effects of known differences between the inbreds (e.g., *P-WW* in K55 vs. *P-WR* in W22 and W23) from unknown specific or non-specific background effects. As Coe and his Coe-workers later found, K55 differs from other inbreds in carrying *whp*, and conceivably some of the differences we found between K55 and the other inbreds could have been due to differences at the *whp* locus. Of course there is no immediate way of testing that possibility except by evaluating the two alleles in backgrounds other than K55. Until recently I have not given much priority to the development of such lines, but have simply been saving those stocks that might one day yield testable comparisons. As I have gathered the *whp C2* lines I need, I have noticed quite frequently a patchy or 'splotchy' anthocyanin phenotype in leaves of some *whp C2* plants. In some families segregating for *c2* and *C2*, the leaves of the *c2* (*whp*) sibs shown necrotic patches in the same regions that their *C2* (*whp*) sibs show anthocyanin patches. This patchiness is not present in *r-g b* lines, so that some *R* (or *B*) function seems to be involved. I am testing further to see what other genes may affect this phenotype (*P, Pl, A1, bz1,* etc.).

Defective *Spm/En*'s at the *P* locus? In last year's Newsletter (page 111), I reported briefly on a *P* allele determining a 'grainy' pericarp that can mutate to a sectored form that in turn is capable of mutating to a stable *P-RR*. Although I have made no inde-

pendent tests for *Spm/En*, I have traced back through my pedigrees, and it seems that I unwittingly introduced a non-defective *Spm/En* or *Spm/En*-like factor via a cross to a 'Rainbow Flint' stock. The original 'grainy' pericarp appears to result from the presence of a defective *Spm/En*(?) at the *P* locus that can be excised in the presence of the non-defective factor. Having satisfied my own mind as to the probable origin of this particular sectoring *P* allele, I then attempted to trace back through my pedigrees for the origin of another sectoring *P* allele that 'arose' in my stocks, distinguished by giving only very fine and infrequent pericarp sectors. As far as I can determine, this sectored form arose from a cross of the same 'Rainbow Flint' stock mentioned above, with a '*P-RW*' allele from a Northwestern Dent source. This Northwestern Dent *P-RW* is a 'frustrating' *P-RW* because it maintains reasonably good pericarp expression in a Northwestern Dent background (where it segregates with a *P-RR* allele) but tends to 'lose' the pericarp expression when it is isolated from its partner *P-RR* or from its Northwestern Dent background. To all intents and purposes it becomes close to a '*P-WW*' allele. Finding that a sectoring form can be derived by crossing it with a stock carrying a non-defective *Spm/En* or *Spm/En*-like factor, may be the clue that can lead to an understanding of the variable nature of this allele.

WALTHAM, MASSACHUSETTS
University of Massachusetts

The Identity of *Mga* (*maize glume architecture*) on 4S confused with a multiple allelic series at the *Tu* (*tunicate*) locus

--Walton C. Galinat

The old defunct and discredited wild podcorn hypothesis of Mangelsdorf and Reeves, as the key part of their tripartite theory that controlled thinking on the origin of maize for more than 25 years, has left a legacy of prejudice against both Mangelsdorf and the *tunicate* locus. For example, Doebley and Kermicle (In: Dorweiler et al., Science 262:233-235, 1993) would allow only two alleles (*Tu1* and *tu1*) at the *tunicate* locus, apparently because of this prejudicial barrier rather than actual facts. A multiple allelic series of *tunicate* alleles (*Tu1 tu1-l, tu1-d, tu1-f, tu1-w, tu1*) at the *Tu* locus was identified long ago (Mangelsdorf and Edwardson, MNL 27:24, 1953) and its components separable by mutation and reconstructible by recombination (Mangelsdorf and Galinat, PNAS 51:147, 1964). The various *tunicate* alleles tend to focus on the architecture of the female spikelets to different degrees. The strongest allele (*Tu1*), key to the wild podcorn hypothesis, has monstrous effects, especially when homozygous, with large amounts of developmental activity going into the foliaceous elongation of just the first and second glumes, with the lemmas and paleas usually left wanting. This hyperactivity of the strongest *tunicate* allele is not grounds for rejecting or ignoring the remainder of the allelic series, which are much more modest in their effects. The *tu1-f* allele from Chapalote and Reventador is close to normal (*tu*) for most modern maize, with slight increases in the foliaceous and length traits of the outer female glume. The *tu1-f* allele has domestic values by its interactions with the *Vg* (*vestigial glume*) on chromosome 1 with partial restoration of glumes that are important in the tassel for sunburn protection. The *tu1-f* allele was important in the past by its interaction with the teosinte allele *mga* linked to *su*. In the combination *tu1-f tu1-f mga mga* (Fig. 1E), the outer female glumes become more foliaceous and elongate, the rachilla more elongate and reflexed, the cupule more reduced. Together these modifications to the teosinte fruitcase made the teosinte grain easily threshable from its enclosure.

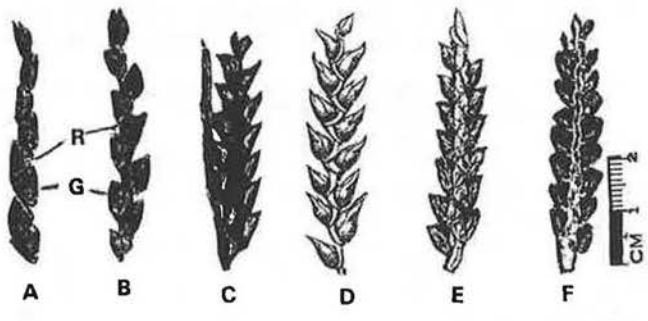

Figure 1. Ear of teosinte (A) compared with its *maize glume architecture* (*Mga* or *Tga*) and *tunicate* (B to E) and non-tunicate derivative (F). Ears (A) and (B) are adapted from Dorweiler et al. (1993) in which the rachis segment (R) of teosinte (A) is fully developed with the cupule sealed by the outer glume (G) in forming a fruitcase type of protective device about the kernel. Ear (B) is claimed by Dorweiler et al. to be *Tga* teosinte. On a basis of comparison with the other teosinte derivatives (C to F), it is suggested here that it may be *tga tu1-f*. Ears (C) and (D) are *tu1-d mga* teosinte adapted from Beadle in 1971 and 1980, respectively. The 1980 line drawing may be made from the 1972 photograph. Ears (E) and (F) are from my large collection of connecting link stocks that I have developed from my studies still underway on the origin of maize. Ear (E) is believed to be *mga tu1-f* and similar to ear (B). This ear is typical of about 300 ears all from one plant with about 20 tillers, each 7 to 9 feet tall with the main stalk 6-1/2 feet tall. Ear (F) has a combination of the modern maize genes *Mga* and *tu*. All ears appear to be stable for single female spikelets. A common metric scale is on the right.

More important than the modifying interaction of the *tu1-f* allele with the teosinte *mga* gene was a dominant mutation at *mga* to *Mga* (*maize glume architecture*), which then made the teosinte spike even more threshable and opened the way for an even lower allele (*tu*) at the *Tu* locus (Fig. 1F). It is not clear at this point if the *tu* allele came by means of mutation from *tu1-f* or by backcross introgression from teosinte.

In attempting to deal with the *tu1-f* allele extracted from Reventador, Doebley would place it at the *mga* locus rather than the *Tu* locus. Such a switch could appear to occur by an accidental mixing of *su gl3* stocks with *su gl4* stocks. Thus, *mga* at point 48 just 14 units above *gl4* could appear to be the location of the *tu1-f* gene instead of *Tu*, at just 11 units above *gl3*.

A comparison of some of the *mga-Mga* tunicate phenotypes is made here in Figure 1 with caption. It is important to note that the tunicates shown here do not include full (*Tu1*). All of the tunicate teosinte which Beadle grew and observed was entirely *tu1-d tu1-d* which he obtained from me. I had explained this to him but, in his enthusiasm, it somehow got overlooked.

Significant differences between populations grown from single *pd* compared with paired *Pd* spikelet seed borne in variegated arrangements on individual ears.

--Walton C. Galinat

In last year's MNL, I reported that in certain of my connecting link stocks between teosinte and maize there was an unstable or variegated expression of the *pd* gene for single female spikelets. Being a non-molecular, old fashioned Mendelian geneticist, I have made a progeny test for the heritability of the differences between the single and paired spikelets on 24 ears variegated for

these conditions. Every precaution was taken not to influence the data with bias. Several people, who were not directly interested in the results, were involved in the sorting of seed to plant, harvesting and scoring ears and analyzing the data as acknowledged. The results reveal a significant difference between the two populations, the one grown from single spikelet kernels and the other from paired spikelet kernels. The data flow sheet is in Table 1. The analysis of variance showing highly significant heritability of paired spikelets is in Table 2 and for single spikelets in Table 3.

Table 1. Number of progeny ears with single (S) or paired (P) spikelets in two populations grown from S and P parental seed borne in variegation by 24 parental ears.

Parent Ear	Total Seed	Progeny Total	from S	from P	Progeny Total	from S	from P
2004	14	3	0	3	11	0	11
2006-4	22	14	1	13	8	1	7
2012	19	8	3	5	11	2	9
2013-1	25	11	4	7	14	0	14
2025-1	44	12	3	9	32	3	29
2028-5	18	2	1	1	16	4	14
2030-1	29	9	4	5	20	9	11
2030-4	13	8	2	6	5	0	5
2033	21	14	8	6	7	2	5
2035	19	6	3	3	13	4	9
2046-1	18	7	0	7	11	4	7
2046-2	46	21	7	14	25	9	16
2062-1	34	14	2	12	20	2	18
2088-1	29	7	3	4	22	2	20
2089	14	4	2	2	10	8	2
2091-2	24	13	4	9	11	4	7
2093	20	7	1	6	13	4	9
2097-2	16	6	0	6	10	2	8
2103-1	17	5	1	4	12	2	10
2124-1	28	8	3	5	20	4	16
2125-1	32	7	0	7	25	2	23
2127-1	10	4	0	4	6	4	2
2132	16	7	4	3	9	8	1
2192-2	64	40	13	27	24	11	13
Totals	592	237	69	168	355	91	264
% of totals		40%	12%	28%	60%	15%	45%
% within treatment			29%	71%		26%	74%

The above data was analyzed as a completely random design. The analysis of variance, one-way classification with unequal replication, was conducted according to the methods described by Steel and Torrie (1980).

1980. R.G.D. Steel and J. H. Torrie. Principles and Procedures of Statistics A Biometrical Approach. 2nd Edition. McGraw-Hill Book Company.

Table 2. Number of paired spikelet ears from single (S) spikelet seed and paired spikelet ears from paired (P) spikelet seed borne in variegated ears.

Trt	ri	Yij	meanYi	Y^2ij	Y^2i/ri	(Yij-meanYi)^2
S	237	168	0.71	1846	119.1	1619.54
P	355	264	0.74	3970	196.3	3592.42
sum	592	432		5816	315.4	5211.96

Analysis of variance for data summarized above

Source of variation	df	SS	MS	F
Among treatments	1	288.84	288.84	32.71**
Within treatments	590	5211.96	8.83	
Total	591	5500.8		

The analysis of variance given above is significant; tabulated F(0.005)=7.88 for 1 and 590 degrees of freedom. Therefore sufficient evidence exists to suggest that the mean number of paired spikelet types produced from single (0.71) and paired (0.74) spikelet type parent plants are different for reasons other than chance.

Because both the single and paired conditions on these variegated ears are inherited and because a pair of spikelets produces twice as many kernels as the single ones, the population would of its own accord shift to the paired condition without human help.

The data clearly demonstrate at least an inherited component,

Table 3. Number of single spikelet ears from single (S) spikelet seed and single spikelet ears from paired (P) spikelet seed borne in variegated ears.

Trt	ri	Yij	meanYi	Y^2ij	Y^2i/ri	(Yij-meanYi)^2
S	237	69	0.29	407	20.09	369
P	355	91	0.26	561	23.33	515.36
sum	592	160		968	43.42	884.36

Analysis of variance for data summarized above

Source of variation	df	SS	MS	F
Among treatments	1	40.4	40.4	26.93**
Within treatments	590	884.36	1.5	
Total	591	924.76		

The analysis of variance given above is significant; tabulated F(0.005)=7.88 for 1 and 590 degrees of freedom. Therefore sufficient evidence exists to suggest that the mean number of single spikelet types produced from single (0.29) and paired (0.26) spikelet type parent plants are different for reasons other than chance.

possibly of a transposon nature, that is regulating *pd-Pd* expression and is passed on to the next generation. The random distribution of paired and single spikelets is mostly in the central region of the ear with more paired ones near the base and more single ones high on the ear. If we assume that it takes more energy (glucose) to differentiate a pair of spikelet primordia than just a single one, this pattern would fit. But even so, this does not exclude transposon regulation. It is generally agreed that the internal environment of the host controls transposon movement during morphogenesis. Perhaps in this case it is the rhythms of distribution of photosynthate that stem from day-night cycles, just as plastochrons of phytomers do, that influence transposon movement which then regulates gene activity. In any case, whether or not energy level is involved, the *pd-Pd* states of expression in a variegated arrangement are inherited. I previously reported a similar situation regarding floral and vegetative multiranking (*mr-Mr*). In this case there was increased transmission of full vegetative multiranking through pollen from the central spike of the tassel in comparison with pollen from the two-ranked lateral branches (MNL 64:120, 1990).

Acknowledgments: Dr. Ann E. Kennedy for carefully separating kernels borne in paired spikelets, even when only rudiments of the second spikelet were apparent, from those kernels borne only one per cupule, and putting them in planting envelopes. Dr. Neelima Sinha for planting this single spikelet seed and paired spikelet seed from 24 divided ear-to-row families. Mr. Miguel Sosa and Mr. Oscar Hernandez for harvesting the uppermost ear on the main stalk from each plant and labeling these ears for classification. Bill Ebener of Mesa Inc. for crucial help with the statistical analysis.

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ICI Seeds

QTLs for degree of pollen-silk discordance, expression of disease lesion mimic, and leaf curl response to drought

--B.E. Zehr, J.W. Dudley and G.K. Rufener

QTL identification has been carried out using a set of 224 S2 progeny derived from the cross of inbred line Mo17 with population BS11(R)C7. Specific RFLP markers and statistical approaches used for QTL analysis are as previously described in a related study by Zehr et al. (TAG 83:903, 1992). Replicated S2

progeny rows were grown at two Illinois locations in both the summers of 1988 and 1989, the former year being a drought environment and the latter year having relatively normal rainfall. Measurement of discordance between average silk emergence and pollen shed was made in 1989 only; data were taken as days from planting. Four RFLP markers showed greatest significance of association with the difference between average pollen and silking dates (Table 1). These data are in partial agreement with those of Phillips et al. (Proc. 47th Annu. Corn Sorgh. Res. Conf., 1992), who described *umc12* as having close association with a gene conferring major influence on maturity in corn, as determined by relative date of pollen shed and silk emergence. However, our data did not indicate significance of *umc12* for these two maturity measures directly, only in their relative degree of separation.

Table 1. RFLP markers having greatest association with degree of discordance between pollen and silking dates.

Chrom. Arm	Marker	Prob. > F
2S	*npi239*	0.0001
4L	*umc66*	0.0093
6L	*npi223*	0.0016
8L	*umc12*	0.0064

Expression of a disease lesion mimic is characteristic of Mo17 and material derived from this inbred line. In the S2 progeny described above, lesions were evident as brown necrotic spots with clear centers and chlorotic halos on leaf blades. Two levels of expression were generally seen; either numerous small lesions, or relatively fewer yet large oblong lesions due to expanded halo width. Data were taken in the drought stress environment of 1988, which seemed to enhance lesion expression. Plants showing lesion mimic were segregating both within and among progeny rows, and data were recorded as the number of plants per row with easily identifiable lesion phenotype at the time of flowering. Four markers representing three chromosome arms showed highest degree of association (Table 2). Two of the chromosome arms represented contain previously identified disease lesion mimic mutations: *Les14* and *Les17* on chromosome arm 3L, and *Les8* on chromosome arm 9S. The small lesion phenotype observed in this study is very similar to that described for *Les14* (Neuffer, MNL 66:39, 1992), suggesting that these two mutations may be allelic. The second phenotype of this lesion mimic (few but big lesions) could be due to the effect of genetic modifiers present in other genomic regions detected in this study (i.e., 3L and 9S). It is well documented that expression of lesions in almost all mimic mutations is highly dependent on genetic background (Walbot et al., Disease lesion mimic mutations, in: Genetic Engineering of Plants, Plenum Pub. Corp., New York, 1983).

Table 2. RFLP markers having greatest association with disease lesion mimic phenotype.

Chrom. Arm	Marker	Prob. > F
3L	*umc96*	0.0088
4L	*umc66*	0.0094
4L	*npi451*	0.0003
9S	*bz1*	0.0013

In the drought environment of 1988, pre-flowering stress response was characterized by leaf curling. Differences in degree of leaf curl were apparent among progeny rows. Data were taken pre-flowering at approximately the eight leaf stage using a rating scale from 1 to 5 on a row average basis; a rating of 1 indicating little or no leaf curl for all plants within a progeny row, and a rating of 5 indicating extreme curling for all plants of a row. Major associations with leaf curl response were found for the 8 markers (Table 3). Mapping studies using maize RFLP probes have shown a large degree of colinearity between the genomes of corn and

Table 3. RFLP markers having greatest association with leaf curl response to drought.

Chrom. Arm	Marker	Prob. > F
2S	*npi239*	0.0005
4L	*bnl15.07*	0.0023
4L	*npi451*	0.0061
5S	*umc27*	0.0085
6L	*umc38*	0.0051
7L	*bnl16.06*	0.0031
7L	*umc80*	0.0011
8S	*npi114*	0.0078

sorghum (Whitkus et al., Genetics 132:1119, 1992; Melake Berhan et al., TAG 86:598, 1993), sorghum being a related crop with good drought tolerance characteristics. Three RFLP markers with significance in this study (*umc27, bnl16.06, npi114*) have been mapped directly in sorghum, making possible any future transfer of such information between the two crops.

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Regions of genomic similarity among four 'Stiff Stalk' inbred lines as measured by multiple restriction enzymes in RFLP analysis

--B.E. Zehr and S. Wright

Multiple restriction enzymes have been used for each of 157 probes in RFLP analysis in order to reveal the extent of genomic similarity among four inbred lines derived from Iowa Stiff Stalk Synthetic (BSSS). Data for 142 probes were obtained using four restriction enzymes per probe (*Hin*dIII, *Eco*RI, *Eco*RV, *Sst*I), while three enzymes were used for each of the 15 remaining probes. The extent of marker coverage per chromosome ranged from 26 probes (chromosome 1) to 9 probes (chromosome 10). The inbred lines used in this analysis were B14A, B37, B73, and B84; each line having proven to be of some historical significance in hybrid breeding, and each derived from either the initial cycle BSSS or an improved version.

Out of 157 probes total, 24 (15%) showed monomorphism among all four inbred lines for each restriction enzyme (Table 1). Thirteen of the 24 completely monomorphic probes were single banded, while the remainder identified multiple banding patterns with the primary band common (monomorphic) among all lines. Over 100 probes (64%) showed monomorphic expression for at least one restriction enzyme, while 56 probes (36%) identified no monomorphism across all four lines. (Table 1).

Table 1. Number of RFLP probes showing monomorphism among lines B14A, B37, B73, B84 for varying levels of restriction enzymes.

Monomorphism	Number of probes	% of total probes
For all 4 enzymes	24	15%
For at least 3 enzymes	36	23%
Monomorphism for at least 2 enzymes	59	38%
Monomorphism for at least 1 enzyme	101	64%
No monomorphism	56	36%

Chromosome numbers 1, 6 and 9 contained the most probes which were monomorphic for all restriction enzymes (Table 2), suggesting a greater degree of genomic conservation for these

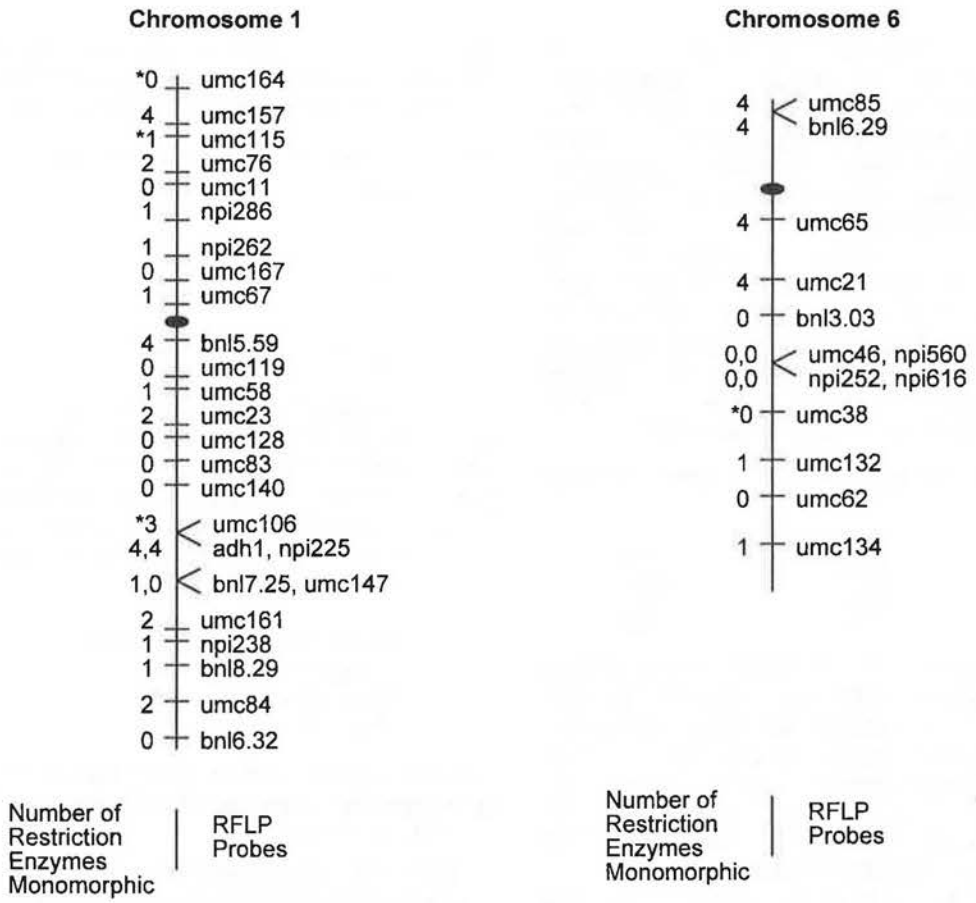

Figure 1. Number of restriction enzymes (out of 4 total, or * = out of 3 total) showing monomorphism among inbred lines B14A, B37, B73, B84 for probes on chromosomes 1 and 6 (note: relative distances between markers are not proportionally accurate).

Table 2. Probes showing monomorphism among lines B14A, B37, B73, B84 for all restriction enzymes used in RFLP analysis.

Chrom. Arm	Probe	Chrom. Arm	Probe
1S	*umc157*	6S	*bnl6.29*
1L	*adh1*	6S	*umc85*
1L	*bnl5.59*	6L	*umc21*
1L	*npi225*	6L	*umc65*
1L	*umc106*		
		7S	*npi400*
2L	*umc98*	7S	*opaque2*
		7L	*bnl16.06*
3L	*umc02*		
3L	*umc97*	8L	*umc93*
		8L	*umc120*
5S	*bnl6.25*		
5L	*bnl5.71*	9S	*umc105*
		9L	*bnl14.28*
		9L	*bnl3.06*
		9L	*npi97*
		9L	*npi291*

chromosomes among the four Stiff Stalk lines. Chromosomes 4 and 10 were not represented among probes showing monomorphism over all restriction enzymes. However, these chromosomes also contained the least number of probes; 10 probes on chromosome number 4, and 9 probes on chromosome number 10.

Genomic regions showing greatest conservation among lines were present both as isolated loci and as larger linkage groups. Figure 1 illustrates relative linkage arrangements and levels of monomorphism for probes on chromosome numbers 1 and 6. For chromosome 1, loci with high degrees of allelic conservation (monomorphic for all enzymes) were adjacent to regions with little or no monomorphism. In contrast, all highly monomorphic probes on chromosome 6 were adjacent, and located on the short arm and centromeric regions. However, marker saturation in these regions of chromosome 6 was somewhat limited.

These data are in agreement with that of Neuhausen (MNL 63:110) with regard to isolated regions of conservation among Stiff Stalk material on chromosome 1, and monomorphic banding patterns at most probes on chromosome 7 among inbred lines B73 and B84 (data not shown), both derived from later cycles of BSSS. Our data indicate a high degree of genomic conservation among both early cycle (B14A, B37) and later cycle (B73, B84) lines only at loci toward either end of chromosome 7. However, conservation among all lines could have been inferred for loci spread across this entire chromosome if ≤ 2 restriction enzymes had been used. This illustrates the value of utilizing multiple restriction enzymes for RFLP analysis when interpreting genomic similarity among individuals or lines.

Maize should be an excellent organism for studying long-term effects of selection with respect to genomic conservation. Each inbred line in this study represents an end point for separate selection experiments which have utilized the same genetic source material, and which have had similar goals. The rationale behind long-term recurrent selection in population improvement, like that ongoing for BSSS, should favor a degree of genomic conservation which would then be reflected through inbred development. We

suggest that regions showing greatest degree of conservation among the lines in this study, as evidenced by monomorphic patterns viewed over multiple restriction enzymes for each RFLP probe, could be prime candidates to contain the genetic factors which have helped define 'Stiff Stalk' as an important heterotic group of hybrid corn.

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Allozyme polymorphisms within and among local varieties of maize in Southwestern China

--H. Lu, Y. L. Zheng, J. S. Li, X. Z. Xiong and J. L. Liu

There is abundant germplasm of maize in China, and more than 8000 local varieties have been collected in 1984. Although local varieties have provided much of the germplasm currently available to maize genetics and breeders in China, they have been little evaluated for their genetic variability.

In this paper, genetic polymorphisms on 18 isozyme loci were investigated for 27 local varieties in Southwestern China. The number of seedlings assayed per variety ranged from 35 to 105. A random sample of seedlings from each variety was assayed by horizontal starch (Sigma Co.) gel electrophoresis. One sample of each of inbred lines Mo17 and Oh43 was taken as a standard on each gel to aid gel reading. The methods described by Stuber (North Carolina Univ. Techn. Bull. 286, 1988) were employed for electrophoresis and denoting alleles for each locus.

Allele numbers on each locus are listed in Table 1. In order to compare with other studies, Table 1 also includes allele numbers

Table 1. Allele numbers detected at isozyme loci in different maize germplasm.

Locus	(1)	(2)	(3)	(4)
acp1	5	4	3	6
acp4	6	-	6	-
adh1	3	3	2	4
cat3	7	3	-	8
est1	4	-	3	-
est3	3	-	5	-
est6	4	-	-	6
got1	3	2	2	4
got2	4	2	-	3
got3	3	1	-	2
mdh1	5	2	-	7
mdh2	4	4	2	5
mdh3	3	2	-	4
mdh5	2	2	-	6
mmm	2	1	-	2
pgd1	5	3	2	5
pgd2	4	2	2	5
phi1	3	4	-	6

(1) Results in this study; (2) adapted from smith et al. (Crop Sci. 25:550, 1985); (3) adapted from Kahler et al. (TAG 72:592, 1986); (4) adapted from Goodman and Stuber (Maydica 28:169-187, 1983).

detected in 72 U.S. important inbred lines (Smith et al., 1985), 12 U.S. Corn Belt open-pollinated populations (Kahler et al., 1986) and 31 races of maize in Bolivia (Goodman and Stuber, 1983). It was shown that at most loci, the number of alleles observed per locus in 27 local varieties was more comparable to the number of alleles observed in 72 U.S. important inbred lines and 12 Corn Belt open-pollinated populations, but less than the number of alleles observed in 31 races of maize in Bolivia. The results indicated that there were extensive allozyme polymorphisms and abundant genetic variability in local varieties in Southwestern China, and that those local varieties were valuable germplasm.

Gene diversity was analysed by Nei's methods (Molec. Pop. Genet. Eval., Elsevier Publ, 1975). The gene diversity in the total population (Ht) can be divided into the gene diversities within and between subpopulations (varieties), signified by Hs and Dst (Dst = Ht-Hs), respectively. The relative and absolute magnitude of gene differentiation among varieties may be measured by Gst (Gst = Dst/Ht) and Dm, respectively. Analysis of gene diversity in 27 local varieties is presented in Table 2. *acp4* had the maxi

Table 2. Gene diversity at 18 isozyme loci of 27 local varieties of maize in Southwestern China.

Locus	Hs	Ht	Gst	Dm
acp1	0.462	0.600	0.230	0.143
acp4	0.432	0.650	0.337	0.232
adh1	0.094	0.106	0.113	0.088
cat3	0.372	0.439	0.150	0.069
est1	0.379	0.537	0.294	0.163
est3	0.163	0.242	0.326	0.073
est8	0.281	0.294	0.044	0.013
got1	0.090	0.147	0.388	0.059
got2	0.087	0.103	0.156	0.017
got3	0.056	0.063	0.111	0.007
mdh1	0.287	0.470	0.389	0.189
mdh2	0.325	0.434	0.251	0.113
mdh3	0.281	0.440	0.361	0.164
mdh5	0.031	0.032	0.030	0.001
mmm	0.004	0.0042	0.048	0.0002
pgd1	0.186	0.332	0.440	0.151
pgd2	0.169	0.320	0.470	0.170
phi1	0.069	0.072	0.420	0.031
Mean	0.209	0.294	0.289	0.088

mum gene diversity in total population (0.650), followed by *acp1, mdh1, mdh3* and *cat3*, at which there were extensive allozyme polymorphisms. The minimum Ht was 0.0042 at locus *mmm*, and Ht's at loci of *mdh5, got3* and *phi1* were less than 0.100, indicating that those loci were stable and had few allozyme polymorphisms. Gst ranged from 0.003 (*mdh5*) to 0.497 (*pgd2*) below 0.500, indicating that gene diversity at a single locus is mainly contained within varieties. Average Gst calculated from 18 isozyme loci was 0.289, and revealed 71.1% and 28.9% of gene diversity contained within and among varieties, respectively. Average Dm from 18 loci was 0.088, meaning that an estimate of the net gene codon differences between varieties and independent of the gene diversity within varieties was 0.088, higher than in most other organisms.

The results of the electrophoretic assays showed that extensive allozyme polymorphism was the predominant feature of the 27 local varieties. The gene diversity in the total population mainly occurred within varieties rather than among them.

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Chromosome linkage study of *Rf* locus for cms-C

--Shaojiang Chen and Weicheng Chen

Restoration of the cms-C group of cytoplasms has been shown to be controlled by three complementary loci, designated *Rf4, Rf5* and *Rf6* (MNL 66:140, 1992). *Rf4* has been located on the long arm of chromosome 8 (Sisco, Crop Sci. 31:1263, 1991); the location of *Rf5* and *Rf6* is unknown. No studies have been performed on the location of the *Rf* loci from China, though studies have shown that the restoration of cms-C is controlled by at least two loci (Chen, Acta Agron. Sin. 5:21-28, 1979). For this reason, it was hoped in this study that the restoring locus could be mapped and compared with the results above.

A series of crosses were made using two different cms-C fertile lines (Guang10-2, Jixi21), which had been confirmed to have one *Rf* gene, and a series of *wx*-translocation stocks which had been converted to cms-C sterility by crossing onto two different sterile lines (cms-ELB73 and cms-Ernan24).

The method used in our study was the same as Alice Johnson's (Johnson, MNL 58:102-103, 1984). The expected ratio was 3:1 (fertile:sterile).

Any cross exhibiting significantly > 25% sterility might be indicative of linkage of the *Rf* gene to the particular translocation since the *wx* T stocks were *rf rf*.

Tables 1-2 and Tables 3-4 were the results of two restoring lines on the sterile backgrounds cms-ELB73 and cms-Ernan24, respectively. Two crosses in Table 1 (No. 3, 4), one in Table 2 (No. 1), one in Table 3 (No. 5) and one in Table 4 (No. 12) exhibited a higher degree of fertility than expected, which could be explained by misclassification. The following crosses in the tables exhibited a much higher degree of sterility than expected: Table 1: T4-9b, T7-9a, T8-9d; Table 2: T6-9a, T7-9a; Table 3: T5-9a, T7-9a, T8-9d; Table 4: T7-9a.

Table 1. Fertility for crosses Guang 10-2/translocations on the background of cms-ELB73.

No.	wx translocation	Observed F	Observed S	Expected F	Expected S	X^2	P
1	T1-98389	193	64	192.75	64.25	0.001	>0.90
2	T1-9c	161	55	162.00	54.00	0.03	0.75~0.90
3	T2-9b	206	45	188.25	62.75	6.69	0.005~0.01 *
4	T3-9c	157	35	144.00	48.00	4.69	0.025~0.05 *
5	T4-9b	183	92	206.25	68.75	10.26	<0.005 * *
6	T4-95657	143	54	147.75	49.25	0.61	0.50~0.75
7	T5-9a	125	33	118.50	39.50	1.43	0.10~0.25
8	T6-9a	12	149	120.75	40.25	2.54	0.10~0.25
9	T7-9a	62	89	113.25	37.75	93.70	<0.005 * *
10	T8-96673	163	58	165.75	55.25	0.18	0.50~0.75
11	T8-9d	98	57	116.25	38.75	11.46	<0.005 * *
12	T9-10b	173	64	177.75	59.25	0.51	0.25~0.50

Table 2. Fertility for crosses of Jixi21/translocations on the background of cms-ELB73.

No.	wx translocation	Observed F	Observed S	Expected F	Expected S	X^2	P
1	T1-98389	106	22	96.00	32.00	4.17	0.025~0.05 *
2	T1-9c	121	33	117.00	39.00	0.55	0.25~0.50
3	T2-9b	163	42	153.75	51.25	2.23	0.10~0.25
4	T3-9c	112	36	111.00	37.00	0.04	0.75~0.90
5	T4-95657	147	36	137.25	45.75	2.77	0.05~0.10
6	T4-9b	97	32	96.75	32.75	0.003	>0.90
7	T5-9a	122	32	115.50	38.60	1.46	0.10~0.25
8	T6-9a	130	83	159.75	53.25	22.16	<0.005 * *
9	T7-9a	25	30	41.25	13.75	25.61	<0.005 * *
10	T8-96673	135	33	126.00	42.00	2.57	0.10~0.25
11	T8-9d	51	20	53.25	17.75	0.38	0.50~0.75
12	T9-10b	86	25	83.25	27.75	0.36	0.50~0.75

It was very clear that the translocation, which couldn't be explained by misclassification or small size, was T7-9a. Therefore, the *Rf* locus for cms-ELB73 and cms-Ernan24 was the same one, and is on chromosome 7 near the breakpoint of T7-9a (7L.63, 9S.07). This showed a difference from the studies before. It seems reasonable to name it *Rf5*.

Table 3. Fertility for crosses of Guang10-2/translocations on the background of cms-Ernan24.

No.	wx translocation	Observed F	Observed S	Expected F	Expected S	X^2	P
1	T1-98389	229	59	216.00	72.00	3.12	0.05~0.10
2	T1-9c	185	60	183.75	61.25	0.03	0.75~0.90
3	T2-9b	171	51	166.50	55.50	0.49	0.25~0.50
4	T3-9c	89	29	88.50	29.50	0.01	0.50~0.75
5	T4-9b	144	25	126.75	42.25	9.39	<0.005 * *
6	T4-95657	167	48	161.75	52.75	0.69	0.50~0.75
7	T5-9a	73	58	98.25	32.75	25.96	<0.005 * *
8	T6-9a	71	17	66.00	22.00	1.17	0.10~0.25
9	T7-9a	59	106	123.75	41.25	135.52	<0.005 * *
10	T8-96673	262	71	247.75	83.25	3.26	0.10~0.25
11	T8-9d	99	94	144.75	48.25	57.84	<0.005 * *

Table 4. Fertility of crosses of Jixi21/translocations on the background of cms-Ernan24.

No.	wx translocation	Observed F	Observed S	Expected F	Expected S	X^2	P
1	T1-98389	180	53	174.74	58.25	0.63	0.25~0.50
2	T1-9c	129	51	135.00	45.00	1.07	0.25~0.50
3	T2-9b	91	21	84.00	28.00	1.71	0.10~0.25
4	T3-9c	136	55	143.25	47.75	1.47	0.10~0.25
5	T4-9b	84	25	81.50	27.50	0.30	0.50~0.75
6	T4-95657	146	32	135.00	45.00	3.58	0.05~0.10
7	T5-9a	97	26	92.25	30.75	0.86	0.25~0.50
8	T6-9a	9	5	10.50	3.50	0.86	0.25~0.50
9	T7-9a	18	24	31.50	10.50	23.14	<0.005 * *
10	T8-96673	22	8	22.50	7.50	0.04	0.75~0.90
11	T8-9d	184	49	174.75	58.25	1.91	0.10~0.025
12	T9-10b	154	32	139.50	46.50	6.03	0.01~0.025 *

F :number of fertile plants
S :number of sterile plants
* :significant at P = 0.05 level
* * :significant at P = 0.01 level

III. USING MAIZE IN K-12 EDUCATION

Last year at the Education Workshop during the Maize Genetics Meeting, there was a great deal of enthusiasm for promoting the use of maize in K-12 Science Education. During the workshop several ideas were discussed. Among them was establishing a section of the Maize Cooperation Newsletter that contained ideas for using maize in the classroom. This year is the first, of what we hope is an annual section in the newsletter. Thanks to everyone who submitted their ideas and activities!

Your ideas are needed for future editions. If you have visited classrooms, given advice to or worked with teachers, or in any other way used maize in the classroom, your colleagues would love to hear about it. I will act as the coordinator to assemble the Education Section of the newsletter. Your ideas can be submitted to me via E-mail, Fax, diskette, or old-fashioned mail. I will put the section together and submit it as one piece to Ed Coe. In future years, I will need to get the information from you by December 15th, so that it can be submitted by the January 1st deadline.

At the 1993 Maize Genetics Meeting, it was decided that a committee would be established to gather information on current programs, establish contacts with those programs and national teaching organizations, and report back to the maize community what type of help it could best provide. Coordination with scientists using other genetic organisms will also be crucial to prevent redundancy of efforts. Ten persons, representing academic and company perspectives, volunteered to serve on this committe. Vicki Chandler (Univ. of Oregon), Ralph Bertrand (Colorado College), David Duncan (Monsanto), Julia Bailey-Serres (UC Riverside), Roger Krueger (American Cyanamid), Ronald Phillips (Univ. of Minnesota), Torbert Rocheford (Univ. of Illinois), Mary Schramke (BioRad), and William Tracy (Univ. of Wisconsin). Numerous other scientists had excellent suggestions and expressed a willingness to participate in various projects. I am attempting to keep an updated mailing list of all persons interested in receiving information regarding using maize in the classroom. If you did not receive a mailing from me last spring and fall and would like to be added to the list, please send me your complete address, telephone, Fax, and E-mail.

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Industry/Education Interactions

--Roger Krueger, American Cyanamid

American Cyanamid performs several science teacher workshops, better known as "Fast Plants." These workshops are coordinated by Fred Stillwagon. We work with other companies as well as school systems. We would like to add some new labs using maize. Anyone with ideas for maize labs should contact Roger Krueger, coordinator of Industry/Education at American Cyanamid.

American Cyanamid also hosts a Teacher Industry and Environment workshop, drawing participants from all over the country.

Partners for Progress

--Debi Blair, Pioneer Hi-Bred

Pioneer belongs to a program called "Partners for Progress." This is an alliance between local businesses and schools. One of our school partners is a grade school. The school is located on a main street in Des Moines and we had a VERY cooperative principal. We ripped up about a 10 by 20 foot patch of their lawn and set up a small plot of hybrid corn. We went out and helped them hand plant the seed. Then they took turns signing up for a week to weed and water through the summer. This let them see the entire growth process from spring planting to fall harvest. We kept this plot going for several summers. It was a nice way to "advertise the alliance" by putting a Pioneer "test plot sign" out front, and gave the children a chance to work with the plants. If we do this again, I would suggest planting different varieties or some interesting mutants. The older children could then make observations.

Providing Ears, Seeds to Local High Schools

--Virginia Walbot, Stanford University

Most schools are pressed if they have to purchase ears from Carolina Biological ... apparently they are about $5 each! So with a minimum of work, a corn lab can supply at least one teacher with materials for every student that will last for several years.

I have provided selfed ears of *A2 Bt Pr/a2 bt pr* and the test cross to *a2 bt pr*. With 150 ears selfed and 150 test cross ears, the teachers can let students really use the ears. Once this was combined with *gl* vs. *Gl*, once with *Y*, and once with *R-nj* vs. *R-scm* vs. *R* so that the students could look at independent assortment, look at different tissues, and appreciate that there are multiple alleles. If large ears are made, they apparently last for several years, even if students remove some kernels.

I've also supplied seed for dwarfs and recipes for applying gibberellic acid. The demonstration that some dwarfs respond to GA, and some don't, was apparently very popular with an 8th grade class in conjunction with a discussion of animal growth hormone.

A Presentation to my Daughter's First Grade Class

--Susan R. Wessler, Department of Genetics/Botany, University of Georgia

Schools in University communities often set aside time for parents/educators to present "show and tell" demonstrations that highlight their area of expertise. In my daughter Nicole's first-grade classroom, this time-period was called "Freaky Friday." My husband, Mark Schell, is a microbiologist. As part of his presentation, he conducted an experiment where half the children washed their hands prior to using their fingers to streak out petri dishes. The following Friday, after the plates had incubated for one week, he returned to the classroom with both the plates and several dissecting microscopes. Although the children were impressed by the variety and intricacies of the bacterial and fungal colonies, they were also given a powerful visual lesson in the importance of washing their hands!

The success of my husband's presentation led me to put together a lesson that would draw on the day-to-day experiences of first graders. My goal was to introduce them to the corn plant "up close" and point out the prevalence of corn products in their daily lives. To this end, the following was done:

1. I brought in a flowering plant and introduced them to the immature ear, the shedding tassel, and how the pollen falls on the silk and initiates seed development.
2. I passed around an immature ear with attached silks so that they could see how each silk was attached to the site of a future kernel.
3. I also brought in and compared an ear from the grocery store

and mature, dried ears with either red or colorless seed. They picked off either red (the girls) or colorless (the boys) seed and planted them in starter-pots provided by the Botany greenhouse staff. Small plastic stakes were also provided so that they could identify their pot and watch their own seedling grow. These were ultimately taken home by the children. The red seeds also carried the *B-I* allele; I was hoping that the seedlings grown from the red kernels and not the colorless ones would display some pigment.

4. I read to them from a simple picture book called "Corn: What it is, What it Does" by Cynthia Kellogg (47p, Greenwillow Books, NY 1989). This book has an excellent section on the more than 2,000 products in the supermarket and in our homes and offices that contain corn products. The students were instructed to check the ingredients of their favorite foods to see if they contain corn products.
5. At the end of the hour, corn muffins and soda (with high fructose corn syrup!) were served. A great time was had by all.

Corn Webbing in Wisconsin
--Bill Tracy, University of Wisconsin-Madison

Against my better judgment I agreed to teach a module (one week) in the UW teacher enhancement program during 2-6 August, the peak of pollinating season. The class consisted of 15 elementary school teachers. Most of the teachers had only introductory college biology. Most knew nothing about genetics. I spent nearly the entire week with them, and it was a wonderful experience.

The class was structured around the idea of "webbing." This is where one thing, in this case corn, is used to teach concepts in all academic subjects ranging from music to math. I had the help of two facilitators who were professional elementary teachers. They were absolutely invaluable. In addition to lectures and demos on many aspects of corn (scientific, social, economic), we had three field trips. Two of the trips were to my field. On the last day, they made their own pollinations among a set of endosperm mutants I had set up. The pollinations were their favorite part of the experience. The other field trip was to a seed production company and a sweet corn processing company.

The cornweb and syllabus is available upon request. In addition, I have a 3" binder filled with my handouts, and activities that the teachers developed, songs, maps, recipes, games, and projects.

As a result of this program I have visited the classrooms of some of "my students." I was very impressed by the enthusiasm the kids had for science in general and corn in particular. Two observations: 1) At the elementary level they really want hands-on activities that will excite the students about science, and 2) while the teachers may be very interested in genetics, their background is usually weak. Do not overshoot your audience. Unless you know they know more, start out with Mendel's laws and meiosis and mitosis.

This was a great experience. I learned many ways to improve my teaching from working with teachers. I am going to do the course again this summer.

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V. MAIZE GENETICS COOPERATION STOCK CENTER

Maize Genetics Cooperation • Stock Center

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During 1993, 2,758 seed samples were provided in response to 301 requests. These totals include 688 seed samples provided in response to 57 requests from 17 foreign countries. The total number of requests exceeds all previous annual totals.

As a result of heavy rainfall, most genotypes grew well but there were more disease and insect problems than normal. Still, we obtained good increases on most stocks that were grown. We had extensive plantings of traits located on Chromosomes 1, 2, and 3, and plantings of stocks in short supply on Chromosomes 4, 5, 6, 7, and 8. Increases were made of the *wx1* marked translocation set, and new *wx1* marked translocations submitted by Donald Robertson and Paul Sisco were increased. It is expected that the new translocation stocks will be ready for distribution within a year or two. Special increases were made of viviparous and yellow stripe mutants of unknown allelism. Field plantings were made of mature plant traits in order to confirm pedigrees. Greenhouse sand bench plantings are being conducted on a record scale in order to confirm seedling mutants. We are hoping that funds for new greenhouse space become available for FY95, so that mutant stocks that do not do well under field conditions can be grown and seedling tests can be done more efficiently.

Philip Stinard has joined us this past May and assumed the role of Curator of the Maize Genetics Cooperation • Stock Center. Earl Patterson continues to play a very active role, allowing for a very smooth transition in the operations at the stock center. Janet Day went from half-time to full-time, as a research specialist with the stock center at the beginning of the year. In addition, two graduate students are presently doing their dissertation research at the stock center. Mirian Maluf is studying a mutable allele of a novel *viviparous* locus, and Dinakar Bhattramakki is studying polymorphism at the *globulin1* locus.

During the year, work on our second coldroom, which essentially doubles current seed storage capacity, was completed and is now fully operational. The new cold room is currently serving as a repository for newly acquired maize collections. We have obtained stocks from the collections of Marcus Rhoades, George Sprague, and Donald Robertson, and are in the process of obtaining stocks from the collections of Barbara McClintock, Charles Burnham, and Walton Galinat. We expect the collection to expand considerably over the next few years to accommodate the collections of maize geneticists retiring from active research.

As the maize gene list expands, we are becoming aware of deficiencies in the Stock Center collection. During the coming year, **we call upon all maize researchers to contribute mutants in their possession that are not in the collection**. Loss of these stocks would represent an irreparable loss of knowledge. Only by propagating these mutants for posterity does the original research done on these mutants preserve its meaning. Mutants that are lost become dropped from the gene list, and they become the object of longing for young researchers poring through dusty volumes of newsletters past. Don't disappoint them. Lost mutants mean that experiments cannot be replicated and expanded upon. If experiments cannot be replicated, conclusions could be discarded. Think about it.

Because of the increasing size of the Stock Center collection and the Stock Center's limited resources, stocks that are rarely requested (particularly multiple combinations of mutants that span 50+ centimorgans, that have phenotypes that are difficult to distinguish from one another, or that show a high degree of redundancy with other stocks) will be dropped from the Stock Center catalogue; however, these stocks will be placed in long term storage at the National Seed Storage Laboratory in Fort Collins, Colorado, and will still be available upon request. We hope to have a list of such stocks by next year. Regardless of which combinations are discontinued, all individual mutants will be actively maintained and supplied from the Stock Center.

We have started accepting stock requests via e-mail (our internet address is maize@uiuc.edu). We have begun entering stock pedigree and availability data into our internal database (using 4th Dimension software on a Macintosh Quadra 950 computer). This information will make pedigree analysis and planting decisions easier.

We have been continuing our collaboration with Ed Coe's efforts in creating the Maize Genome Database (MaizeDB). This is part of the Plant Genome Database (PGD) effort being sponsored by the National Agriculture Library (see details elsewhere in the Newsletter). We have plans to tie our internal stock center database in with MaizeDB (and therefore also with the PGD at NAL and also with GRIN (Germplasm Resources Information Network) to allow users access to the latest information about available maize genetic stocks. Presently, our list of available stocks is accessible via gopher from PGD and MaizeDB (the full Sybase version is now accessible to researchers). With the help of Quinn Sinnott, data on available maize genetic stocks has also been entered into GRIN. A list of available stocks will continue to be published annually as part of the Maize Genetics Cooperation Newsletter.

We anticipate that in addition to current methods for requesting stocks, a user will be able to find a stock of interest on 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	Earl Patterson	Philip Stinard	Janet Day
Director	Co-director	Curator	Research Specialist

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 *an1 bm2 sr1 P1-WR*
102C *ad1 bm2 sr1 P1-RW*
102D *ad1 bm2 sr1 P1-RR*
103C *bm2 sr1 P1-WR*
103D *vp5*
103E *zb4 ms17 P1-WW*
103G *bm2 sr1 P1-RR*
104B *bm2 ts2 zb4 P1-WW*
105A *zb4 P1-WW*
105E *ms17 P1-WR*
105F *ms17 P1-WW*
106A *bm2 zb4 P1-WW*
106B *ts2 P1-RR*
106C *bm2 ts2 P1-WW*
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 *f1 an1 bm2 br1 gs1 P1-RR*
109A *ad1 an1 bm2 P1-RR*
109B *an1 bm2 gs1 P1-RR*
109D *ad1 bm2 P1-RR*
109E *f1 br1 P1-WR*
110B *Kn1 an1 P1-WR*
110C *ad1 an1 bm2 P1-WR*
110D *an1 bm2 P1-WR*
110E *ad1 bm2 P1-WR*
110F *Vg1 br1 P1-WR*
110G *f1 bm2 br1 gs1 P1-WR*
110K *br1 P1-WR*
111A *rs2 P1-WW*
111D *f1 br1 hm1 P1-WW*
111G *rs2 P1-WR*
112B *f1 bm2 br1 P1-WW*
112E *as1*
112H *br1 P1-WW*
113A *as1 br2*
113B *rd1*
113C *f1 br1*
113E *f1 Kn1 br1*
113K *hm1; hm2*
113L *Hm1; hm2*
114B *f1 Kn1 bm2 br1*
114D *Vg1*
114E *f1 Vg1 br1*
114F *br2 hm1*
115B *Vg1 bm2 br2*
115C *v22*
115D *bz2-m; A1 A2 C1 no Ac Pr1 R1*
115E *Vg1 br2*
116A *bz2-m; A1 A2 Ac C1 Pr1 R1*
116C *an1 bm2*
116D *an1-bz2-6923*
116I *Ts6 bm2 bz2 gs1*
117A *br2*
117B *bm2 br2*
117D *tb1*
117E *Kn1*
118A *Kn1 Ts6*
118B *Kn1 bm2*
118C *lw1*
119B *vp8*
119C *gs1*
119D *bm2 gs1*
119E *Ts6*
119F *bm2*
120A *id1*
120B *nec2*
120C *ms9*
120D *ms12*
120F *Mpl1*
121A *ms14*
121B *mi1*
121C *D8*
121D *lls1*
121J *br2 ms14*
122A TB-1La
122B TB-1Sb (1S.05; BL)
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 *Les2-845A*
127A *bm2 bz2 zb7*
127B *dek1*
127C *dek2*
127D *dek22*
127E *f1*
127G *Tlr1-1590*
128A *ij2-8*
128B *l16-515*
128C *l17-544*
128D *pg15-340B*
128E *pg16-219*
128F *v25*
129A *w18*
129B *wlu5*
130A *o10-1356*

CHROMOSOME 2

201F *b1 gl2 lg1 ws3*
203B *al1*
205B *lg1*
205C *gl2 lg1*
206A *B1 gl2 lg1*
206B *B1 gl2 gs2 lg1*
208B *B1 gl2 lg1 sk1*
208D *B1 v4 gl2 lg1*
208E *b1 gl2 lg1*
208F *b1 gl2 gs2 lg1*
208H *gl2*
209B *b1 v4 gl2 gs2 lg1*
209E *b1 gl2 lg1 sk1*
209F *b1 fl1 gl2 lg1 sk1*
210A *b1 v4 gl2 lg1 sk1*
211A *b1 fl1 gl2 lg1*
211D *b1 gl2 wt1*
212B *b1 v4 fl1 gl2 lg1*
212D *b1 v4 gl2 lg1*
212E *b1 v4 Ch1 gl2 lg1*
213A *v4 gl2 lg1 mn1*
213B *gl2 lg1 wt1*
213C *w3 gl2 lg1*
213D *w3 Ch1 gl2 lg1*
213E *b1 Ch1 gl2 lg1*
213F *Ch1 lg1; B1-V*
213G *Ch1 lg1*
214C *d5*
214D *B1 gl11*
214E *B1 ts1*
214F *v4 Ch1 gl2*
214G *v4 gs2 lg1*
215B *gl11*
215C *wt1*
215E *fl1*
215G *v4 fl1*
216A *v4 Ch1 fl1*
216D *w3 fl1*
216E *v4 w3 fl1*
216F *w3 Ch1 fl1*
217A *ts1*
217B *v4*
217E *w3 Ch1 Ht1*
217H *v4 ba2*
218A *w3*
218C *w3 Ch1*
218D *Ht1*
218E *ba2*
218F *B1 ba2*
219B *B1+Peru; A1 A2 C1 r1-g*
219C *Ch1*
220A *Les1-843*
220B *gl2 lg1 ws3;* Alien Addition T2-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 *Les4-1375*
227I *nec4-516B*
228A *l18-1940*
228B *spt1-464*
228C *v26*
229A *Ch1 rl3*
229B *v24*

CHROMOSOME 3

301A *cr1*
302A *d1-6016*
302E *d1-tall*
303A *d1 Lg3 rt1*
303B *d1 Rl1 lg2*
303F *g2*
303G *d1 g2*
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 lg2 ts4 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 *Rg1 lg2 ra2*
309E *lg2 pm1 ra2*
310A *ra2 ts4*
310C *lg2 ra2*
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 *lg2 ts4 ys3*
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 *et1 lg2 a1-m; A2 C1 dt1 R1*
319D *et1 lg2 a1-m; A2 C1 Dt1 R1*
319F *et1 lg2 a1-st; A2 C1 C2 Dt1 R1*
320A *lg2*
320C *lg2 na1*
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 *sh2 A1-d31; A2 C1 dt1 R1*
322B *sh2 A1-d31; 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 *sh2 a1-m; A2 B1 C1 dt1 Pl1 R1*
324A *a1-st; A2 C1 Dt1 R1*
324E *et1 a1-st; A2 C1 Dt1 R1*
324G *a1-st; A2 C1 dt1 R1*
325A *et1 a1-p; A2 C1 dt1 R1*
325B *et1 a1-p; 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*
332F *gl19-169*
332G *dek6*
332H *dek17*
332I *Lxm1-1600*
332J *ms23*
332L *brn1*
332N *wlu1*
332P *g2 brn1*
332Q *cr1 brn1*

CHROMOSOME 4

401A *Rp4*
401D *Ga1-S*
401J *Ga1-M*
402A *st1*
402C *fl2 st1*
402D *Ts5*
403A *Ts5 fl2*
404A *Ts5 su1 zb6*
405B *la1*
405D *gl3 la1 su1*
405G *gl4 la1 su1*
406C *fl2*
406D *fl2 su1*
407B *bm3 fl2 su1*
407D *su1*
407E *su1-am*

408B *bm3 su1*
408E *bm3*
408K *se1 su1*
409A *Tu1 su1 zb6*
410D *gl3 su1 zb6*
412C *gl3 su1*
412E *j2 gl3 su1*
413B *gl4 su1*
414A *bt2*
414B *gl4*
414C *o1 gl4*
414E *de*-414E*
415A *j2*
415C *C2 j2; 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 *o1 gl3*
418A *dp1 gl3*
418B *c2; A1 A2 C1 R1*
418C *C2; A1 A2 C1 R1*
418E *dp1*
418F *o1*
418G *v17*
419B *gl3 ra3 su1*
419F *Dt6 gl3; a1-m A2 C1 R1*
420A *Dt4 su1; a1-m A2 C1 R1*
420B TB-9Sb-4L6504
420C *nec*-rd*
420D *yel*-8457*
420H *C2 Dt4; a1-m A2 C1 R1*
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 *cp2-211C* (was *dek7*)
427B *dek25*
427C *Ysk1*
427D *orp1; orp2*
427E *dek8*
427F *dek10*
427G *Ms41-1995*
427H *dek31*
428A *gl5; gl20*
428B *lw4; lw3*
428C *nec5-642A*
428D *spt2-1269A*
428E *wt2*
428F *lw4; Lw3*
428G *bx1*

CHROMOSOME 5

501A *a2 am1; A1 C1 R1*
501B *lu1*
501C *lu1 sh4*
501D *ms13*
501E *gl17*
501H *a2 bt1 gl17; A1 C1 R1*
501I *am1*
502A *a2 v2 bt1 gl17; A1 C1 R1*
502B *A2 pr1 ps1-vp7; A1 C1 R1*
502D *A2 bm1 pr1; A1 C1 R1*
503A *A2 bm1 pr1 ys1; A1 C1 R1*
503D *A2 v3 bt1 pr1; A1 C1 R1*
504A *A2 bt1 pr1; A1 C1 R1*
504B *A2 v2 bm1 pr1 ys1; 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 v2 pr1; A1 C1 R1*
506D *A2 na2 pr1; A1 C1 R1*
506F *A2 pr1 v12; A1 C1 R1*
506L *A2 br3 pr1; A1 C1 R1*
507A *a2; A1 C1 R1*
508C *a2 bt1 bv1 pr1; A1 C1 R1*
508F *a2 bm1 pr1 ys1; A1 C1 R1*
510A *a2 v2 bm1 pr1; A1 C1 R1*
510B *A2 bm1 eg1 pr1; A1 C1 R1*
510G *a2 bm1 eg1 pr1; A1 C1 R1*
511A *a2 v3 bt1 pr1; A1 C1 R1*
511C *a2 bt1 pr1; A1 C1 R1*
512A *a2 v2 bt1; 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 v2 pr1; 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 *ae1 td1*
516G *A2 bm1 pr1 yg1; A1 C1 R1*
516I *td1; Rp1*
517A *v3*
517B *ae1*
517E *ae1 gl8 pr1*
518A *sh4*
518B *gl8*
518C *na2*
518D *lw2*
518F *v2 sh4*
518H *v2 gl8*
519A *ys1*
519B *eg1*
519C *v2*
519D *yg1*
519E *A2 pr1 yg1; A1 C1 R1*
519F *A2 gl8 pr1; A1 C1 R1*
519G *zb3*
520B *v12*
520C *br3*
520F *A2 Dap1; A1 C1 C2 R1*
520G *A2 pr1 Dap1; A1 C1 C2 R1*
521A *nec3-409*
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 *nec7-756B*
527G *pr1 sh5*
528A *Hsf1-1595*

CHROMOSOME 6

601D *Y1 rgd1*
601E *po1-ms6*
601F *y1 pl1 po1-ms6*
601G *y1 Pl1 po1-ms6*
602A *y1 wi1 po1-ms6*
602K *y1-gbl*
603A *y1 l10*
603C *y1 l12*
603D *y1 w15*
603H *y1 mn3*
604A *y1 pb4 pl1*
604B *y1 Pl1 pb4*
604D *y1 l15*
604F *y1 si1-mssi*
604H *y1 ms1*
604I *Y1 ms1*
605A *y1 Pl1 wi1*
605F *Y1 pl1 wi1*
606A *Y1 pg11; pg12 Wx1*
606B *y1 pg11; pg12 wx1*
606C *Y1 pg11; pg12 wx1*
606D *y1 pg11; pg12 Wx1*
606E *y1 pl1*
606F *y1 Pl1*
607A *y1 Pl1 Pl1-Bh1; A1 A2 c1 R1 sh1 wx1*
607B *y1 pl1 Pl1-Bh1; A1 A2 c1 R1 sh1 wx1*
607C *y1 su2*
607D *y1 pl1 su2*
607F *y1 Pl1 su2*
608G *Y1 l11*
609B *Y1 pl1 wi1*
609C *Y1 Pl1 wi1*
609D *Y1 su2*
610B *Dt2 Pl1; a1-m A2 C1 R1*
610C *pl1 sm1; P1-RR*
610H *Y1 Dt2 pl1; a1-m A2 C1 R1*
611A *Pl1 sm1; P1-RR*
611D *Pt1*
611E *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*
701F *Hs1*
702B *o2 v5 gl1 ra1*
703A *o2 v5 gl1*
703J *Rs1-O*
703K *Rs1-Z*
705A *o2 gl1*
705B *o2 gl1 sl1*
705C *o2 ij1-ref::Ds*
705D *o2 bd1*
706A *o2 sl1*
707A *v5 y8 gl1*
707B *in1; A1 A2 C1 pr1 R1*
707D *v5*
707E *vp9*
707F *y8 gl1*
708A *ra1*
708G *y8*
709A *gl1*
710H *Tp1 gl1 ms7*
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 *Dt3 gl1; a1-m A2 C1 R1*
716A *v*-8647*
716B *yel*-7748*
716F *Las9-2008*
717A TB-7Lb
718A Trisomic 7
727A *dek11*
727B *wlu2*

CHROMOSOME 8

801A *gl18*
801B *v16*
801C *j1 v16*
801D *j1 ms8 v16*
803A *ms8*
803B *nec1*
804A *v21*
804D *wh*-053-4*
804E *w*-017-4*
804F *w*-034-16*
804G *w*-8635*
804H *w*-8963*
805A *fl3*
805D *j1 fl3 ms8*
805E *el1*
806A TB-8La
806B TB-8Lb
808 *ct1*
809A TB-8Lc
827A *dek20*
827B *dek29*
827C *Bif1-1440*
827D *Sdw1-1592*
827E *Clt1-985*
827K *pro1*

CHROMOSOME 9

901D *bz1 sh1 wx1 yg2 C1-l; A1 A2 R1*
901E *C1 bz1 wx1 yg2; A1 A2 R1*
902A *c1 bz1 sh1 wx1 yg2; A1 A2 R1*
902B *c1 sh1 wx1 yg2; A1 A2 R1*
902C *c1 sh1 wx1 yg2 gl15; A1 A2 R1*
902D *c1 sh1 wx1 yg2 gl15; A1 A2 K9S-s R1*
902E *c1 bz1 wx1 yg2; A1 A2 R1*
903A *C1 bz1 sh1; A1 A2 R1*
903B *C1 bz1 sh1 wx1; A1 A2 R1*
903D *bz1 sh1 wx1 C1-l; A1 A2 R1*
904B *C1 sh1; A1 A2 R1*
904C *C1 sh1 wx1; A1 A2 R1*
904D *C1 ar1 wx1; A1 A2 R1*
905A *C1 sh1 wx1; A1 A2 K9S-l R1*
905B *C1 ms2 sh1; A1 A2 R1*
905C *C1 Wx1 bz1; A1 A2 R1*
905D *C1 sh1 wx1; A1 A2 K10 K9S-l R1*
905E *C1 v1 sh1 wx1; A1 A2 C2 R1*
906A *C1 wx1; A1 A2 Ds Pr1 R1 y1*
906B *C1 wx1; A1 A2 Ds pr1 R1 Y1*
906C *Wx1 C1-l; A1 A2 Ds R1*
906D *C1-l; A1 A2 R1*
906G *Wx1 C1-l; A1 A2 Ds R1*
907A *C1 wx1; A1 A2 R1*
907D *C1 wx1; A1 A2 B1 pl1 R1*
907E *wx1 C1-l; 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 v1 wx1; A1 A2 R1*
908D *C1 wx1 gl15; A1 A2 R1*
908E *C1 wx1 gl15; A1 A2 pr1 R1*
908F *C1 da1 wx1; A1 A2 R1*
908H *C1 wx1; A1 A2 R1 y1*
909A *C1 Bf1 wx1; A1 A2 R1*
909B *c1 bz1 wx1; A1 A2 R1*
909C *c1 bz1 sh1 wx1; A1 A2 R1 y1*
909D *c1 sh1 wx1; A1 A2 R1*
909E *c1 v1 sh1 wx1; A1 A2 R1*
909F *c1 sh1 wx1 gl15; A1 A2 R1*
910B *c1 Bf1 sh1 wx1 gl15; A1 A2 R1*
910C *c1 bk2 sh1 wx1; A1 A2 R1*
910D *c1; A1 A2 R1*
910G *C1 Wx1 sh1-bz1-x2; A1 A2 R1*

911A *c1 wx1; A1 A2 R1 y1*
911B *c1 v1 wx1; A1 A2 R1*
911C *c1 wx1 gl15; A1 A2 R1*
911D *c1 Bf1 wx1; A1 A2 R1*
912A *sh1*
912B *v1 sh1 wx1*
913A *sh1 wx1*
913C *l7 sh1*
914A *d3 wx1*
914E *Wx1 pg12; pg11 y1*
914F *wx1 pg12; pg11 y1*
914G *Wx1 pg12; pg11 Y1*
914H *wx1 pg12; pg11 Y1*
915A *wx1*
915B *wx1-a*
915C *w11*
916A *v1 wx1*
916C *bk2 wx1*
917A *Bf1 wx1*
917C *v1*
917D *ms2*
917E *gl15*
917F *d3*
918A *Bf1 gl15*
918D *Wc1*
918E *Wx1 bk2 bm4*
918F *Bf1*
918G *Bf1 bm4 Wc1-Wh*
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-I; A1 A2 R1* Ring 9S Ring Chromosome 9S
927A *dek12*
927B *dek13*
927C *dek30*
927D *Les8-2005*
927E *Zb8*
927F *C1; a1-r A2 Dt7 R1*
928A *v28*
928B *wlu4*
928C *C1 Bf1 wx1; A1 A2 r1*
930C *Bf1 ms2 wx1; A1 A2 r1*

CHROMOSOME 10

X01A *oy1*
X01B *R1 oy1; A1 A2 C1*
X01E *R1 bf2 oy1; A1 A2 C1*
X02G *oy1 zn1*
X02I *bf2 oy1 ms10*
X02K *oy1 zn1*
X03A *sr3*
X03B *Og1*
X04A *R1 Og1 du1; A1 A2 C1*
X04B *ms11*
X04C *bf2 ms11*
X04D *bf2*
X05A *bf2 zn1*
X05E *bf2 sr2*
X06C *R1 g1 nl1; A1 A2 C1*
X07C *y9*
X07D *nl1*
X09B *R1 g1 li1; A1 A2 C1*
X09F *ms10*
X10A *du1*
X10D *g1 r1 du1; A1 A2 C1*
X10F *zn1*
X10G *du1 v18*
X11A *g1 zn1*
X11F *g1 r1; A1 A2 C1*
X11H *zn1 R1-r; A1 A2 C1*
X12A *g1 r1 sr2*
X12E *R1 g1; A1 A2 C1*
X13D *g1 sr2 r1-r; 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*
X18A *R1-lsk; A1 A2 C1*
X18C *R1-st; A1 A2 C1*
X18D *R1-sk; A1 A2 C1*
X18E *Mst1 R1-st*
X18G *R1-scm2; A1 A2 bz2 C1 C2*
X18H *R1-nj; 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 *Les6-1451*
X27E *gl21-478B*
X27F *Vsr1*
X27G *Oy1-700*
X27H *orp2; orp1*
X27I *I19-425*
X28F *cr4*

UNPLACED GENES

U140C *l4*
U141A *ms22*
U141B *ms24*
U141C *o9*
U141D *o11*
U142B *o13*
U142C *rd3*
U142D *ub1-76C*
U142E *y11*
U142F *y12*
U240A *Les7-1461*
U240B *vp10*

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 *A1; A2 B1 C1 C2 Pl1 R1-r*
M741F Stock 6 *A1; A2 C1 C2 pl1 R1-g* scutellum colored *y1*
M741G Stock 6 *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 *Ht1-Ladyfinger*
P242C Ohio Yellow Popcorn
P342A Red Popcorn
P342B Strawberry Popcorn
P342C Supergold Popcorn *Adh1+33F*
P342D South American Popcorn
P442B White Rice Popcorn

EXOTICS

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 *Adh2-33*
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 Autotetraploid; *P1-RR*
N103D Autotetraploid; *P1-WR*
N104B *A1; A2* Autotetraploid; *C1 pr1 R1*
N104C Autotetraploid; *su1 wx1*
N106D Autotetraploid; *sh1 Wx1 Y1*
N106E Autotetraploid; *sh1 wx1 y1*
N107B W23 Autotetraploid
N107C Synthetic B Autotetraploid

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 N cytoplasm *rf1; rf2*

WAXY RECIPROCAL TRANSLOCATIONS

wx01A T1-9c (1S0.4; 9L.22); *wx1*
wx01B T1-9(5622) (1L.1; 9L.12); *wx1*
wx03A T1-9(8389) (1L.74; 9L.13); *wx1*
wx04A T2-9c (2S0.4; 9S0.3); *wx1*
wx05A T2-9b (2S0.1; 9L.22); *wx1*
wx06A T2-9d (2L.83; 9L.27); *wx1*
wx07A T3-9(8447) (3S0.4; 9L.14); *wx1*
wx08A T3-9c (3L.09; 9L.12); *wx1*
wx10A T4-9e (4S0.5; 9L.26); *wx1*
wx11A T4-9g (4S0.2; 9L.27); *wx1*
wx12A T4-9(5657) (4L.33; 9S0.2); *wx1*
wx13A T4-9b (4L.9; 9L.29); *wx1*
wx15A T5-9(4817) (5L.06; 9S0.0); *wx1*
wx16A T5-9d (5L.14; 9L.1); *wx1*
wx17A T5-9a (5L.69; 9S0.1); *wx1*
wx18A T6-9(4778) (6S0.8; 9L.3); *wx1*
wx20A T6-9b (6L.1; 9S0.3); *wx1* y1
wx21A T6-9(4505) (6L.13; 9); *wx1*
wx22A T7-9(4363) (7; 9); *wx1*
wx23A T7-9a (7L.63; 9S0.0); *wx1*
wx24A T8-9d (8L.09; 9S0.1); *wx1*
wx25A T8-9(6673) (8L.35; 9S0.3); *wx1*
wx26A T9-10(8630) (10L.37; 9S0.2); *wx1*
wx27A T9-10b (10S0.4; 9S0.1); *wx1*
wx28A T5-9(8386) (5L.87; 9S0.1); *wx1*

NON-WAXY RECIPROCAL TRANSLOCATIONS

Wx30A T1-9c (1S0.4; 9L.22); *Wx1*
Wx30B T1-9(4995) (1L.19; 9S0.2); *Wx1*
Wx30C T1-9(8389) (1L.74; 9L.13); *Wx1*
Wx31A T2-9c (2S0.4; 9S0.3); *Wx1*
Wx31B T2-9b (2S0.1; 9L.22); *Wx1*
Wx32A T3-9(8447) (3S0.4; 9L.14); *Wx1*
Wx32B T3-9(8562) (3L.65; 9L.22); *Wx1*
Wx32C T3-9c (3L.09; 9L.12); *Wx1*
Wx33A T4-9e (4S0.5; 9L.26); *Wx1*
Wx33B T4-9(5657) (4L.33; 9S0.2); *Wx1*
Wx33C T4-9g (4S0.2; 9L.27); *Wx1*

Wx34A T5-9c (5S0.0; 9L.1); *Wx1*
Wx34B T5-9(4817) (5L.06; 9S0.0); *Wx1*
Wx34C T4-9b (4L.9; 9L.29); *Wx1*
Wx35A T5-9(8386) (5L.87; 9S0.1); *Wx1*
Wx35B T5-9a (5L.69; 9S0.1); *Wx1*
Wx35C T5-9d (5L.14; 9L.1); *Wx1*
Wx36A T6-9(4778) (6S0.8; 9L.3); *Wx1*
Wx37A T6-9(8768) (6L.89; 9S0.6); *Wx1*
Wx37B T7-9(4363) (7; 9); *Wx1*
Wx37C T6-9(4505) (6L.13; 9); *Wx1*
Wx38A T7-9a (7L.63; 9S0.0); *Wx1*
Wx38B T8-9d (8L.09; 9S0.1); *Wx1*
Wx38C T8-9(6673) (8L.35; 9S0.3); *Wx1*
Wx39A T9-10(8630) (10L.37; 9S0.2); *Wx1*
Wx39B T9-10b (10S0.4; 9S0.1); *Wx1*

INVERSIONS

I143B Inv1c (1S0.3-1L.01)
I143C Inv1d (1L.55-1L.92)
I143D Inv1(5131-10) (1L.46-1L.82)
I243A Inv2(8865) (2S0.0-2L.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 (9S0.7-9L.9)
I443A Inv4b (4L.4-4L.96)
I443B Inv4c (4S0.8-4L.62)
I444A Inv2a (2S0.7-2L.8)
I543A Inv4e (4L.16-4L.81)
I743A Inv5(8623) (5S0.6-5L.69)
I743B Inv6(8452) (6S0.7-6L.33)
I743C Inv6(3712) (6S0.7-6L.63)
I843A Inv6(8604) (6S0.8-6L.32)
I943A Inv7(5803) (7L.17-7L.61)
I943B Inv7(8540) (7L.12-7L.92)
I943C Inv7(3717) (7S0.3-7L.3)
IX43A Inv8a (8S0.3-8S0.1)
IX43B Inv9b (9S0.0-9L.87)

VI. ZEALAND 1994

This is a summary of selected genetic research information reported in recent literature and in this News Letter. Numbers preceded by "r" refer to numbered references in the Recent Maize Publications section. New loci; mapping; cloning; sequencing; and trait inheritance information added this year to the Maize Genome Database (MaizeDB) have been extracted here. The term 'genelist' refers to references with information central to the uniqueness and designation of the gene, and may include references that are the first report for that gene. The Symbol Index also provides access to journal publications in which studies on gene expression, gene products, developmental control, physiological responses, techniques, etc., are reported. Comments or suggestions on these research aids, assembled by an unrestricted, Prof. Ligate Committee (Pat Byrne, Ed Coe, Georgia Davis, and Mary Polacco), are always welcome.

* with symbols identifies genes that may be allelic to previously designated genes. For guidance in choosing and assigning symbols, please refer to the section, A Standard for Maize Nomenclature, in this News Letter.

CHROMOSOME 1

acp4: bin 1.16, map note --r290
Adh1+Cm, evolution, origin --r293
adh1, map data --r696
adh1, promoter analysis --MNL68:41-44
adh1: bin 1.12, map note --r290
bz2: bin 1.10, map note --r290
cp3, first report --MNL68:28
cps1, first report --r46
cps1: uncovered by TB-1La, map note --MNL68:41
csu61, map note --MNL68:30-34
csu92, map note --MNL68:30-34
Def(Kn1)O: includes *kn1* and *knox3* but not *adh1* or *lw1*; not male transmissible, TB-1La hypoploids embryonic lethal, map note --MNL68:3-4
dek1, vp5 uncovered by TB-1Sb, map note --MNL68:28
glb1: bin 1.10, map note --r290
gsr1, map note --MNL68:30-34
hcf6, map note --MNL68:41
hsp26, map data --r290
knox3, first report --MNL68:3-4
mdh4, genelist --r414
mdh4, map note --MNL68:30-34
p1: bin 1.04, map note --r290
pdc3: *umc11 - wusl1032(gfu) - pdc3 - npi286* (bin 1.03); *umc11* -18- *npi286*, map --r610
phi1: bin 1.12, map note --r290
tb1, origin --MNL68:88-89
ts2, map note --MNL68:70
ts2, sequence, *ts2-m1*, *ts2-m2*, restriction map --r206
umc184a(glb1), map data --r290
umc185(p1), map data --r290
umc194a(gpr), map data --r290
umc196(gfu), map data --r290
umc197(b32), map data --r290
umc217(gfu), map note --MNL68:30-34
uwo2, map note --MNL68:56
yg-2448* -25- T1-9c(1S.48), map data --MNL68:27

CHROMOSOME 2

akh2, first report, bin2.07: *umc55a* -3.6- *akh2* -4.9- *umc139*, map note --MNL68:94
al1, *lg1*, *gl2*, *d5*, *gl11*, *wt1* uncovered by TB-3La-2S6270, map data --r55
ask2: bin 2.07±, *ask2* -6.5- *umc55*, *ask2* -13.4- *umc5*; data from 16 bulked F3 *Ask2* lines, map note --MNL68:93
csu109, map note --MNL68:30-34
csu17(rnp), map note --MNL68:30-34
csu40(grx), map note --MNL68:30-34
csu64(tau), map note --MNL68:30-34
et2-91g6290-26, first report --MNL68:107-108
gn1 first report, tightly linked or cosegregates with *knox4*, 1 cM from *bnl17.19b*, in bin 2.11, map note --MNL68:2
knox4, first report, map data --MNL68:2
les-1378* -7- T2-9d, *les*-1378* -48- T2-9b, *les4/les*-1378*, 98 testcross plants showed 1 normal and 3 more-extreme; suggests non-allelic (to date, no proven cases of allelism among lesion mutants), map data --MNL68:29
les1 -14- T2-9b(2) *wx1*, map data --MNL68:29
les10 -25- T2-9b(2) *wx1*; *les10* -33- T2-9d(2) *wx1*, map data --MNL68:29
les11 -48- T2-9b(2) *wx1*; *les11* -23- T2-9d(2) *wx1*, map data --MNL68:29
les15 -2- T2-9b(2) *wx1*, map data --MNL68:29
les18 , first report, -22- T2-9b(2) *wx1*; *les18* -15- T2-9c(2) *wx1*; *les18* -49- T2-9d(2) *wx1*, map data --MNL68:29
les19, first report, -24- T2-9b(2) *wx1*; *les19* -42- T2-9c *wx1*; *les19* -26- T2-9d(2) *wx1*, map data --MNL68:29
les4 -48- T2-9b(2) *wx1*; *les4* -12- T2-9d(2) *wx1*, map data --MNL68:29
prp2, map note --MNL68:30-34
ssu2: bin 2.06, map note --r290
umc131 -4.6- *umn1(acc)* -10.4- *umc2b* (bin 2.06), map note --MNL68:92-93
umc184b(glb), map data --r290
umc198(whp1), map data --r290
whp1: bin 2.10, map note --r290

CHROMOSOME 3

a1: bin 3.09, map note --r290
Abp1+W22, sequence, *abp1*, sequence --r691
atp1, genelist --r414
atp1, map note --MNL68:30-34
bif2, map note --MNL68:28
csu25(P450), map note --MNL68:30-34
csu32, map note --MNL68:30-34
csu96, map note --MNL68:30-34
e4: bin 3.04, map note --r290
e8: bin 3.01, map note --r290
g3, map data --MNL68:16
lg2 -8- *lxm1*, map data --MNL68:16
lg3: probed site uncovered in TB-3Sb hypoploids and monotelo3L (i.e., hypo3S) plants, map data --MNL68:16
lhcb1: bin 3.09, map note --r290
lxm1: *bnl5.37* -6.6- *lxm1* -4.3- *bnl8.01* (bin3.06), map data --MNL68:16
mdh3: bin 3.08, map note --r290
me3, genelist --r414
me3, map note --MNL68:30-34
npi477(cab), map data --r290
obf-A1* and *-A2* band polymorphism maps near *bnl15.20* (bin 3.07), map note --r273
te1: bin 3.05, *umc18* -5.9- *umc26* -4.1- *te1* -2.9- *bnl5.37* -6.5- *bnl5.14*, origin, map note --MNL68:91-92
tlr2, first report --r403
umc199(a1), map data --r290
umc208(cppgk), map data --r290
umc92 uncovered by TB-3Sb, map note --MNL68:16
umc92 uncovered by TB-3Sb, map note --MNL68:16
zag2, first report --r688

CHROMOSOME 4

adh2, evolution --r304

adh2: bin 4.02, map note --r290

akh1: first report, bin4.04, *umc191(gpc1)* -7.5- *akh1* -2.7- *umc201(nr)*, map note --MNL68:94

bnl8.23 - *uwo3* - *bnl15.07*, map note --MNL68:56

c2-m881058Y, sequence --r564

Cat3+W64A, sequence, *cat3*, sequence --r2

cat3: *npi333* -7- *[cat3, ncr(b70b)]* -1- *umc111* (bin 4.10), map data --r713

cent4: bin 4.05 by telotrisomic mapping: *bnl5.46* - *npi386* - *umc47* - *cent4* - *bnl15.45* - *bnl7.20* - *umc14* - *npi270*, map data --MNL68:71

cp2, map note --MNL68:107

csu39(gfu), map note --MNL68:30-34

Dt6 left of TB-4Sa, map note --r86

gl4, map location --r222

gpc1: bin 4.04, map data --MNL68:30-34 r290 r696

gpc3, map note --MNL68:30-34

la1 (bin 4.03) -1.3- *npi386* -4.5- *dek7* (bin 4.04±.01) -1.3- *orp1* -0.6- *gpc1* -1.9- *su1* (bin 4.04±.01) -3.4- *(tga1, umc42, bt2)* -3.4- (*umc156, php20597*) -3.4- *npi584* -6.8- *gl4* (bin 4.06), map location --*r222*

m-adh2n microsatellite cosegregates with *adh2* RFLP/isozyme, map data --r696

m-gpc1 microsatellite cosegregates with *gpc1* RFLP/isozyme, map data --r696

mpik5a, clone isolation, map note --MNL68:62-63

mpik5b, map note --MNL68:62-63

mpik6, map note --MNL68:62-63

mpik7, map note --MNL68:62-63

mpik8, map note --MNL68:62-63

orp1: bin 4.04, map note --r290

prh1, map data --r290

ris2, first report, map location --MNL68:41

sos1, first report --MNL68:87-88

ssu1: bin 4.08, map note --r290

su1 -11.3- *lw4* -8.2- *gl4*, map data --MNL68:107 r222

su1, restriction map --MNL68:8

su1-2412, first report, *su1-3162*, first report, *su1-4582*, restriction map, *su1-7110*, polymorphism with *Su1* probe, first report --MNL68:8

tga1, map location, origin --MNL68:109 r222

tga1: bin 4.04, *umc193a(orp1)* -0.7- *umc191(gpc1)* -6.2- *(tga1, npi316)* -2.0- *umc201* -1.3- *(bt2, umc47)* -0.4- *npi27* -7.5- *umc42*, map location --r222

umc191(gpc1), map data --r290

umc200(adh2), map data --r290

umc201(nr), map data --r290

uwo3, map note --MNL68:56

zbr1, map note --MNL68:30-34

zrp4, first report --r352

CHROMOSOME 5

Cat1+W64A, sequence --r332

csu108(gtpb), map note --MNL68:30-34

csu149(ts2), map note --MNL68:30-34

csu173(gfu), map note --MNL68:30-34

lw2, map note --r232

pgm2: bin 5.03, map note --r290

pr1 -14.4- *lw3* -37.9- *v2*, map data --MNL68:107

ris1, first report, map location --MNL68:41

tbp2, first report --r787

umc186b(Bs1), map data --r290

umc209(prk), map data --r290

CHROMOSOME 6

csu116(elf), first report --r414

csu70(gfu), map note --MNL68:30-34

Dt2 right of TB-6Lc, map note --r86

dzs23, first report, sequence --MNL68:81-82

dzs23: bin 6.06, distal to *umc21*, 4±3 cM, map note --MNL68:92

enp1, map note --MNL68:30-34

ga-GFS1994*, first report --MNL68:105

gpc2: bin 6.00, map note --r290

hox2, map note --MNL68:24

idh2: bin 6.10, map note --r290

m-ppdka2 microsatellite cosegregates with *pdk1* RFLP/isozyme, map data --r696

mdh2: bin 6.10, map note --r290

npi(pdk1), map data --r696

oec33: bin 6.02, map note --MNL68:30-34 r290

pgd1: bin 6.01, map note --MNL68:30-34 r290

pl1, sequence --r164

pl1: bin 6.04±.01, map note --r290

rab17 (=dhn1): *bnl3.03* -4- *rab17* -20- *umc138* (bin 6.06), map --r849

rab17: bin 6.06, map note --r290

rhm1, *agrp144*, *umc85* closely linked (some variation in order among experiments); distal to *bnl6.29* (bin 6.00), map --r849

umc173b(pdk), map data --r290

umc180(pep), map note --r290

umc204(bz1), map data --r290

zag1, first report --r688

Zp15+A5707, restriction map, sequence, *zp15*, sequence --r228

CHROMOSOME 7

crp1, map note --MNL68:41

csu129(ntm9), map note --MNL68:30-34

csu13(h1), map note --MNL68:30-34

Csu13+B73, first report --r414

csu27(bcl), map note --MNL68:30-34

Dt3 right of TB-7Lb; *crp1* right of TB-7Lb, map note --MNL68:41 r86

e1: bin 7.05, map note --r290

m-tpi1 microsatellite polymorphism cosegregates with RFLP/isozyme, map data --r696

o2, evolution --r585

o2-23::*En*, restriction map --r31

o5 -2.6- *gl1* -9.7- *tp1*, map data --r232

rs1, map note --r788

thp1, genelist, map note --MNL68:30-34 r414

tpi1, map data --r696

umc193c(orp), map data --r290

CHROMOSOME 8

ald1, map note --MNL68:30-34

bif1 -14- *pro1* -27- *lg4*, map data --MNL68:27-28

blh-2359*, map note --MNL68:27

csu31, map note --MNL68:30-34

emp3, map data --MNL68:28

ldh1: bin 8.07, map note --r290

lhcb3, map note --MNL68:30-34

ms8 -13- *j1* -9- *emp3*, map data --MNL68:28

obf-B1* and *B2* band polymorphism maps near *umc7* (bin 8.10), map note --r273

pdc1: *umc173* - *pdc1* - *umc12* (bin8.05); *umc173* -18- *umc12*, genelist, map data --r610

pdc2: *bnl9.11* - *pdc2* - *umc124* (bin 8.03); *bnl9.11* -24- *umc124*, genelist, map data --r610

pro1 -46- *j1* -31- *rgh1*, map data --MNL68:28

rgh1, map data --MNL68:28

stp1, map note --MNL68:30-34

umc173a(pdk), map data --r290

umc186a(Bs1), map data --r290

umc189a(a1), map data --r290

umc206(hsp70a), map data --r290

CHROMOSOME 9
ar1, map note --r788
bz1-m13CS13, first report, *bz1-m13CS14*, first report, *bz1-m13CS15*, first report, *bz1-m13CS16*, first report --r113
bz1: bin 9.02, map note --r290
C1-m925408U, first report --MNL68:6-7
csu147, map note --MNL68:30-34
csu43(gfu), map note --MNL68:30-34
csu59, map note --MNL68:30-34
csu93, map note --MNL68:30-34
dek-Mu1364*, map note --MNL68:16
eno1, map note --MNL68:30-34
*Eno*csu158+W64A2*, first report --MNL68: 101-104
pep1: bin 6.05, map note --r290
sh1: bin 9.02, map note --r290
sus2: bin 9.04, map note --r290
umc194b(gpr), map data --r290
wx1: bin 9.03, map note --r290

CHROMOSOME 10
csu6, map note --MNL68:30-34
csu86, map note --MNL68:30-34
Dsl - 1.4 - *tp2* - 12.4 - *r1*, map data --r232
glu1: bin 10.04, map note --r290
gpa1: bin 10.06, map note --MNL68:30-34 r290
hupm1, map note --MNL68:30-34
npi371c, structure --r358
orp2: bin 10.04±.01, map note --r290
oy1: see *rp1* mapping data, map note --r358
R1-mb1994, first report --MNL68:63-64
r1: bin 10.08, map note --r290
rp1: *(php20075, bnl3.04)* -1.5- *Rp1-G* -2- *Rp1* -1- (*ksu3/4*, *npi422=npi371c*) -4- *npi285* -11- *oy1*, map data --r358
Sn1-bol3, sequence --r175
T10S-B-10L18a uncovers *y9* (10S) and *r1* (10L), map note --r88
T5-10(4801)(10) - *7.0* - *tp2* - *14.1* - *r1*, map data --r232
umc182(r1), map data --r290

UNPLACED
aat1, first report --MNL68: 101-104
abp4, first report --r691
abp4, sequence --r691
abp5, first report --r691
abt1, first report --MNL68: 101-104
Ac2, genelist --r207
*agp*uazT14743*, first report --MNL68: 101-104
ans1, genelist --r414
*ant*uaz155*, first report --MNL68: 101-104
app1, first report --r733
asp1, first report --r755
*atp*uaz243*, first report --MNL68: 101-104
atpc1, first report --r357
atpc1, sequence --r357
ba3, first report --r591
bvp1, first report --MNL68: 101-104
cah1, genelist --r414
*cin*csu12*, genelist --r414
clp1, first report --MNL68: 101-104
cry1, first report --r447
cry2, first report --r447
cry3, first report --r447
csu54b, map note --MNL68:30-34
d-GFS1994*, first report --MNL68:105
Def(Kn1)O, first report --MNL68:3-4
Ds-r, restriction map --r665
*elf*uaz220*, first report --MNL68: 101-104
elf1, genelist --r414
elf2, first report --MNL68: 101-104
end1, first report --MNL68: 101-104
gbp1, first report --r414
gpb1, genelist --r414
*gpc*uaz190*, first report --MNL68: 101-104
gss1, first report --MNL68: 101-104
*gst*csu44*, first report --r414
gst1, genelist --r680
hca1, first report --MNL68: 101-104
his2b(uaz228)*, first report --MNL68: 101-104
*his3*uaz248*, first report --MNL68: 101-104
hsk1, first report --MNL68: 101-104
*hsp18*uaz171*, first report --MNL68: 101-104
*hsp18*uaz210*, first report --MNL68: 101-104
hsp18a, first report --r28
*hsp70*uaz219*, first report --MNL68: 101-104
*hsp90**, sequence --r527
Htm1, first report --r656
Irma, first report --r564
Irma, sequence --r564
*lhcb*csu66*, genelist --r414
*lhcb*X68682*, first report --r785
lop1, first report --r107
ltf1, first report --MNL68: 101-104
MARZadh1, first report --r34
MDMV-cp, first report --r563
Med, first report --r564
mfs14, first report --r832
mfs18, first report --r832
mn4, first report --MNL68:28
mnb1, sequence --r838
mta1, first report --MNL68: 101-104
Mu1, origin --r136
MuDR, sequence --r382
Mx, first report --r555
*myb*uaz216*, first report --MNL68: 101-104
nac1, first report --MNL68: 101-104
NCS-1994*, first report --MNL68:100-102
nsf1, first report --r404
*obf*X69152*, first report --r273
*obf*X69152*, map note --r273
*obf*X69153*, first report --r273
*obf*X69153*, map note --r273
*ohp1**, first report --r628
*ohp2**, first report --r628
pal1, genelist --r414
pbp1, first report --r406
*pgl*X65847*, sequence --r45
*pgl*X65849*, first report --r45
*pgl*X65849*, sequence --r45
*pgl*X65850*, first report --r45
*pgl*X65850*, sequence --r45
*pgl*X66422*, first report --r45
pgl1, sequence --r45
pgl2, sequence --r45
pgl3, sequence --r45
pgl6, sequence --r17
pgl7, sequence --r45
pgl8, first report --r45
pgl8, sequence --r45
*plt*csu136*, genelist --r414
*plt*uazT14763*, first report --MNL68: 101-104
pop1, first report --MNL68: 101-104
*ppi*uaz238*, first report --MNL68: 101-104
*ppi*uaz288*, first report --MNL68: 101-104

VII. 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. GENE LIST AND WORKING MAPS

GENELIST: A table of the defined gene loci of maize, extracted from the Maize Genome Database (MaizeDB), follows. The table includes the symbol for the locus; the location in 'bins' as described below ("±" denotes a location near the position listed); the locus name with a brief phenotypic description; and references to first reports or publications central to the designation of the locus. Stocks of variants may be obtained from the Maize Genetics Stock Center, as described in that section; many variations (e.g., isozymes and RFLPs) occur naturally among generally available strains.

LARGE LEAP: The genelist is much expanded over previous versions. There is continued rapid growth in new mapped loci identified by directly visible mutations, and in new mapped loci defined by probing with sequences from clones with specifically targeted functions. But the growing volume of cDNA sequences for which a defined function can be identified is unparalleled, and the result is a conspicuous expansion of our knowledge resource. There are 677 genes in this list that have been defined to linkage group, and approximately 1,200 genes are known.

MENDELIAN CRITERIA, MATCHING CRITERIA, AND CANDIDATES: The traditional criteria for designating a unique gene (Mendelian inheritance of a variation accompanied by evidence that it is different from ones previously defined) are today complemented by criteria based on evidence for existence of a function, for possible matching to known genes in the universe of biological systems, for specific functionality of a genomic site, and for uniqueness of a genomic site. Our application of these criteria is shown in a chart accompanying the Table of cDNA Candidates that follows the Genelist.

NOMENCLATURE: The Standard for Maize Nomenclature is reprinted in the preceding section.

MAPS: Conventional representation of order and distance relationships for all entities in a single diagram has finally become unrealistic. Because segmentation of the genome into experimentally defined parts is one of the most effectively applied strategies (e.g., *Drosophila melanogaster*, *E. coli*, *Homo sapiens*), a 'bin' representation is offered for your assessment in this issue of MNL.

Following the gene list is the published UMC Core Map (Gardiner et al., Genetics 134:917-930, 1993), revised to be current with respect to nomenclature and new information. The bin numbers are on the left, and the boundary markers for each bin are boxed; the bin locations in the Gene List refer to these segments. PLEASE NOTE that the bin assignments have been specified on the basis of available data, but that they are subject to the same statistical constraints as those for map order, because interval placements are determined almost exclusively through analysis of recombination data. Distances in the map are in centiMorgans (1% recombination = 1 cM). Each chromosome begins at the top with a distal marker mapped in the short arm. Traditional map diagrams, and cytological maps, may be found in MNL 67 or in sources identified inside the back cover.

Following the Core Map is the current BNL map, re-analyzed with MapMaker and represented as described in the accompanying note. We are all indebted to Eileen Matz and Ben and Francis Burr for the development and sharing of this invaluable resource.

Construction of maps that integrate the locations of genes, cytogenetic variants, and molecular markers requires systematic compilations of data, ongoing in the MaizeDB program. More importantly, integrated maps require the development of complex mapping utilities. Enhanced utilities that can be applied to the data are in preparation.

The current Plastid Chromosome Genetic Map, prepared by Carolyn Wetzel and Steve Rodermel, and Mitochondrial Maps, prepared by Christiane Fauron, follow the nuclear working maps.

The data shared by all Cooperators is represented in these summaries, and we know we speak for all Cooperators in appreciating their availability. Compilation, verification, and encapsulation of the information was specifically aided by the care and efforts of Lou Butler, Oscar Heredia-Diaz, and Theresa Musket. Mike McMullen and Georgia Davis gave us key advice on the criteria and representation for the many new loci.

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 the area of quantitative genetics) progress. IF YOU HAVE A CLONE for a known function and know or believe that it hybridizes to maize genomic sequences, you should 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. We would also use the probe in correlation to physical and conventional markers. We have included in this Newsletter 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 and Mary Polacco

SYMBOL	BIN	NAME, PHENOTYPE	REF
a1	3.09	anthocyaninless1, colorless aleurone, green or brown plant, brown pericarp with *P1-RR*, encodes NADPH dihydroflavonol reductase	158
a2	5.06	anthocyaninless2, like *a1*, but red pericarp with *P1-RR*, may encode? naringenin, 2-oxoglutarate 3-dioxygenase	263
a3	3.08+-0.01	anthocyanin3, recessive intensifier of expression of *R1* and *B1* in plant tissue, encodes *a3*-product	326
abp1	3.05	auxin binding protein1, putative auxin receptor, single band in Southerns, encodes auxin binding protein	534, 606
abp4		auxin binding protein homolog4, putative auxin receptor, genomic clone promoter-reporter gene fusion functional in maize leaf protoplasts. cDNA ZmERabp4 probes one band on Southerns, may encode? auxin binding protein	534
abp5		auxin binding protein homolog5, genomic sequence, promoter-reporter gene fusion functional in maize protoplasts, may encode? auxin binding protein	534
abt1		ATP-binding transport protein homolog1, (was *uaz230*) partially sequenced cDNA to endosperm mRNA, homologous to membrane carrier proteins, may encode? membrane permease	236
Ac		Activator, autonomous transposable element; regulates *Ds* transposition and dissociation; *Ac9* is element isolated from *wx1-m9*, encodes TPASE	63, 360
Ac2		similar to *Ac*, but one dose engenders no excisions, and higher doses show exponential increases	133, 134
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	448
aco1	4.04	aconitase1, electrophoretic mobility; monomeric, encodes aconitate hydratase	627
aco2		aconitase2, electrophoretic mobility, encodes aconitate hydratase	627
aco3		aconitase3, electrophoretic mobility, encodes aconitate hydratase	627
aco4		aconitase4, electrophoretic mobility; monomeric, encodes aconitate hydratase	627
acp1	9.04	acid phosphatase1, electrophoretic mobility; cytosolic; dimeric, encodes acid phosphatase	151, 152, 215
acp2		acid phosphatase2, electrophoretic mobility; dimeric, encodes acid phosphatase	152, 215, 279
acp4	1.16	acid phosphatase4, electrophoretic mobility; monomeric, encodes acid phosphatase	279
acpt1		acyl carrier protein1, acyl carrier protein (*acp*) cDNA, encodes 121 aa polypeptide, contains transit peptide sequence, encodes acyl carrier protein	563
ad1	1.1	adherent1, seedling leaves, tassel branches, and occasionally top leaves adhere	290, 292
ad2		adherent2, upper leaves and tassel tend to adhere and fuse; seedling and juvenile stages normal	414, 429
adh1	1.12	alcohol dehydrogenase1, electrophoretic mobility; null alleles are known; dimeric; intra/interlocus hybrid bands occur), encodes alcohol dehydrogenase	279, 530, 532
adh2	4.02	alcohol dehydrogenase2, electrophoretic mobility; null alleles are known; dimeric; intra/interlocus hybrid bands occur, encodes alcohol dehydrogenase	530
adk1	6+-0.01	adenylate kinase1, electrophoretic mobility; plastidial, encodes adenylate kinase	632
adr1		alcohol dehydrogenase regulator1, recessive (in strain R6-67) sustains higher levels of scutellar ADH vs. usual decline (in W64A) during germination	306
ae1	5.07	amylose extender1, glassy, tarnished endosperm; high amylose content, encodes 1,4-alpha-glucan branching enzyme	616
afd1	6.07+-0.04	absence of first division1, male and female sterility; failure of synapsis, anaphase I equatorial	122, 208
agt1		agravitropic1, primary root unresponsive to gravity	145
akh1	4.04	aspartate kinase-homoserine dehydrogenase1, cDNA clone 77% homologous to carrot threonine-sensitive AK-HSDH bifunctional enzyme, encodes aspartate kinase homoserine dehydrogenase	392
akh2	2.07	aspartate kinase homoserine dehydrogenase2, cDNA clone, sequence 75% homologous to carrot threonine-sensitive AK-HSDH bifunctional enzyme, encodes aspartate kinase homoserine dehydrogenase	392
al1	2.01+-0.01	albescent plant1, variably cross-banded to white leaves, pale yellow endosperm, some alleles viviparous; see *y3*, which evidently is an allele	454, 463
ald1	8.08	aldolase1, cytosolic aldolase; cDNA and genomic clones; Southern blots give single or double band; promoter functional in transient expression assay, encodes aldolase	225, 287
alh1	1.15+-0.01	histone la, (was H1a); electrophoretic mobility, encodes histone la	592
alpha		*a1* locus component (see beta), determines reduced aleurone and plant color, brown pericarp	313
als1	4.04	acetolactate synthase1, sensitive to imidazolinone herbicides; acetohydroxyacid synthase has altered herbicide inhibition kinetics, encodes acetohydroxyacid synthase	13, 430
als2	5.07	acetolactate synthase2, sensitive to imidazolinone herbicides; acetohydroxyacid synthase has altered herbicide inhibition kinetics, encodes acetohydroxyacid synthase	13, 430

SYMBOL	BIN	NAME, PHENOTYPE	REF
alt1		L-alanine:2-oxoglutarate aminotransferase1, electrophoretic mobility; *alt1* and *alt2* interact to form heterodimers, encodes L-alanine:2-oxoglutarate aminotransferase	623
alt2		L-alanine:2-oxoglutarate aminotransferase2, electrophoretic mobility; *alt1* and *alt2* interact to form heterodimers, encodes L-alanine:2-oxoglutarate aminotransferase	623
alt3		L-alanine:2-oxoglutarate aminotransferase3, electrophoretic mobility, encodes L-alanine:2-oxoglutarate aminotransferase	623
am1	5.04+-0.01	ameiotic1, male and female sterility; anaphase I equatorial	206, 444, 486
am2		ameiotic2, like, but not allelic to, *am1*	122
amp1	1.1	aminopeptidase1, electrophoretic mobility; cytosolic; monomeric, encodes aminopeptidase	440
amp2	1.08+-0.01	aminopeptidase2, electrophoretic mobility; monomeric, encodes aminopeptidase	440
amp3	5.06	aminopeptidase3, electrophoretic mobility; monomeric, encodes aminopeptidase	440
amp4		aminopeptidase4, electrophoretic mobility; monomeric, encodes aminopeptidase	440
amy1		alpha amylase1, electrophoretic mobility; monomeric, encodes alpha amylase	89
amy2	5.044+-0.01	beta amylase2, electrophoretic mobility; monomeric, encodes beta amylase	88
an1	1.1	anther ear1, andromonoecious dwarf, intermediate stature; few tassel branches; responds to gibberellins; *an1-6923* includes deletion of *Bz2+*	155, 165
anl1	5.05+-0.01	anthocyaninless lethal1, Colorless aleurone; small kernels; embryo inviable	105
ans1		anthranilate synthase homolog, (was *csu65*) partially sequenced cDNA to leaf mRNA, homologous to yeast TRP3, anthranilate synthase component II, may encode? anthranilate synthase	285
ant1		adenine nucleotide translocator1, open reading frame encodes polypeptide of 40,519 Da; previous single site (5L, MNL 67) contradicted by two sites probed by p-csuh26 in Tropical Maize F2's, may encode? adenine nucleotide translocator, mitochondrial	21
ant2		adenine nucleotide translocator2, cDNA sequence corresponds to genomic sequence; actively transcribed in basal meristem, not in green leaves, may encode? adenine nucleotide translocator, mitochondrial	26
aph1		aphid resistance1, recessive resistance	53, 87
app1		abnormal phragmoplast formation1, phragmoplasts in microsporocytes abnormal, cytokinesis disorganized	581
ar1	9.04+-0.01	argentia1, virescent seedling, greens rapidly; husk leaf tips striped	171
ars1		autonomously replicates in yeast; 11,000 copies in maize	45
ars2		autonomously replicates in yeast; 10,000 copies in maize	45
ars3		autonomously replicates in yeast; 28,000 copies in maize	45
as1	1.05	asynaptic1, synaptic failure in male and female	36
ask1	7.01+-0.01	aspartate kinase1, lysine-threonine resistance in cultures and seedlings, increased threonine in kernels, altered kinetics of aspartate kinase, encodes aspartate kinase	136
ask2	2.07+-0.01	aspartate kinase2, lysine-threonine resistance, encodes aspartate kinase	136
asp1		absence of meiotic spindle1, meiosis normal up to diakinesis; spindle absent, telophases contain 3-10 nuclei	600
asr1	4	absence of seminal roots1, dominant *Asr1* is absence of seminal roots	382
atn1		anaerobic tolerant null1, enhances survival of ADH-null under anoxia	314
atp1	3.05	ATPase1, (was csu30) single copy; amino acid sequence, from partial cDNA sequence, is identical to proteolipid of Avena sativa vacuolar ATPase, encodes proteolipid, vacuolar ATPase	285
atp2		ATP synthase2, cDNA clone, encodes ATP synthase beta chain, mitochondrial	639
atpc1		ATP synthase gamma subunit1, N-terminal amino acid sequence, cDNA sequence from clone selected using anti-gammaCF1 serum, encodes ATP synthase, gamma subunit, chloroplast	239
ats1	8.06+-0.03	atrazine susceptible1, lacks glutathione S-transferase	222
B chromosome		supernumerary chromosome; occurs naturally in many maize and teosinte populations	478
B-A translocation		interchange between a B chromosome and a member of the basic (A) set of chromosomes	504
b1	2.04	colored plant1, dominant *B1* plants have anthocyanin in major plant tissues; some alleles affect aleurone and embryo color; regulates flavonoid enzymes, encodes *B1* (myb) protein	161
ba1	3.06+-0.01	barren stalk1, ear shoots and most tassel branches and spikelets absent	240
ba2	2.05	barren stalk2, like *ba1*, but tassel more normal	240
ba3		barren stalk3, no ear produced	446, 447
baf1	9.03	barren stalk fastigiate1, ear shoots often absent; tassel branches erect	106
bcl1		B cell lymphoma homolog1, (was *csu27*) cDNA to leaf mRNA, homologous to human lymphoma protein, BCL-3, may encode? cell cycle protein CDC10	28
bd1	7.06+-0.01	branched silkless1, ear silkless, branched at base; tassel proliferated, bushy	291
ben1		bentazon resistance1, dominant resistance	179
beta		*a1* locus component (see *alpha*), determines aleurone and plant color, red pericarp	313
bf1	9.09	blue fluorescent1, homozygous *bf1* seedlings, homozygous or heterozygous anthers, fluoresce blue under ultraviolet light; anthranilic acid accumulates, anthranilate synthase has altered inhibition kinetics, may encode? anthranilate synthase	60

SYMBOL	BIN	NAME, PHENOTYPE	REF
bf2		blue fluorescent2, similar to *Bf1* in expression; shows earlier, stronger seedling fluorescence	9
Bg		transposable element, Bergamo, regulatory element mediating *o2-mr*	511
bif1	8.03+-0.01	barren inflorescence1, dominant *Bif1* plants have ear and tassel with many fewer spikelets, bare rachis appendages	419, 422
bif2	3.07+-0.03	barren inflorescence2, variable expression on ear with 0-2 spikelets produced at each floral node. Few-branched, normally no pollen shed	58
bip1		BiP homolog1, cDNA clone, protein body, putative molecular chaperone of hsp70 family, encodes maize BiP	50
bk2	9.04	brittle stalk2, brittle plant parts after 4-leaf stage	311
blh1	1.04+-0.03	bleached1, dominant *Blh1* plants have pale green midveins and base in upper leaves	407
bm1	5.05+-0.01	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	170
bm2	1.14	brown midrib2, like *bm1*	77
bm3	4.05	brown midrib3, like *bm1*; has lowered activity of catechol O-methyl transferase. Silage corn with *bm3*, having improved digestibility, is in production	72, 166, 301
bm4	9.09	brown midrib4, like *bm1*	74
bn1	7.05	brown aleurone1, yellowish brown aleurone color	305
br1	1.09	brachytic1, short internodes, short plant; no response to gibberellins	288, 290
br2	1.08+-0.01	brachytic2, like *br1*	316
br3	5.07+-0.07	brachytic3, like *br1*	550
bre1		branching enzyme1, maize kernel cDNA highly homologous to starch branching enzyme I of bacteria, putative 64-amino acid transit peptide, highly expressed in early stages of kernel development, may encode? starch branching enzyme	18
brn1	3.02+-0.01	brown aleurone1, brown kernel, brown embryo; seedling lethal	500
Bs-1		barley stripe, transposable element, retrovirus-like; 1-5 copies in genome	270
bs1		barren sterile1, Plant weak, with little or no tassel and usually with only a vestige of pistillate inflorescence, shank, husks	371, 640
bt1	5.06	brittle endosperm1, mature kernel collapsed, angular, often translucent and brittle, may encode? amyloplast adenylate translocator	342, 633
bt2	4.04	brittle endosperm2, like *bt1*; endosperm ADPG pyrophosphorylase subunit (compare *sh2*), encodes ADP glucose pyrophosphorylase	166, 605
btn1		brittle node1, tassel breakage in B73 inbred line	281
bu1		leaf burn1, leaves show burning, sometimes horizontal bands, accentuated by high temperature	194
bv1	5.07+-0.01	brevis plant1, short internodes, short plant	318
bv2		brevis plant2, plant height 50-70% of normal; possible allelism with *rd1*	464
bvp1		bovine virus protein homolog1, (was *uaz207*) partially sequenced cDNA to endosperm mRNA, strong homology to bovine virus protein, may encode? transcription factor	236
bvp2		bovine virus protein homolog2, cDNA to endosperm mRNA, homologous to bovine virus glycoprotein, may encode? glycoprotein	237
bx1	4.01+-0.01	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*	120
bz1	9.02	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	477, 485
bz2	1.1	bronze2, like *bz1*; anthers not fluorescent; *an1-6923* mutation includes deletion for *Bz2*; potential function flavonoid acylation, glycosylation, transport, or deposition, encodes BZ2 product	435, 436
c1	9.01	colored aleurone1, *C1* colored; *c1* colorless; *C1-I* dominant colorless; *c1-p* pigment inducible by light, encodes *C1* (myb) protein	149
c2	4.08	colorless2, colorless aleurone, reduced plant and cob color; chalcone synthase; *C2-Idf* dominant inhibitor; duplicate factor with *whp1* for pollen color and for anthocyanins, encodes chalcone synthase	62
cah1		carbonic anhydrase homolog, (was *csu125*) partial cDNA to leaf mRNA, sequence homologous to pea carbonic anhydrase, may encode? carbonic anhydrase	285
cal1		calmodulin homolog1, cDNA sequence, may encode? calmodulin	221a
car1	1.04+-0.03	catalase regulator1, dominant *Car1* determines increased enzyme activity level, encodes CAR1 product	519
cat1	5.05	catalase1, electrophoretic mobility; cytosolic/glyoxysomal; tetrameric; intra/interlocus hybrid bands occur, encodes catalase	40, 528
cat2	1.01	catalase2, electrophoretic mobility; null allele is known; cytosolic/glyoxysomal; tetrameric; intra/interlocus hybrid bands occur, encodes catalase	516
cat3	4.1	catalase3, electrophoretic mobility; null allele is known; mitochondrial; tetrameric; intralocus hybrid bands occur, encodes catalase	518
cdc2		cell division control protein2 homolog, cDNA sequence homologous to CDC2/CDC28 subfamily of serine/threonine protein kinases, may encode? serine/threonine protein kinase	108

SYMBOL	BIN	NAME, PHENOTYPE	REF
cdc48	6.02	cell division protein48 homolog, (was *csu146*) single map site; partially sequenced cDNA to leaf mRNA; strong homology to FTSH, an E. coli cell division protein, class CDC48, encodes cell cycle protein CDC48	28, 29
cdh1		cinnamyl alcohol dehydrogenase1, electrophoretic mobility, encodes cinnamyl alcohol dehydrogenase	184
cdj1		chaperone DNA J homolog1, (was *csu63*) partially sequenced cDNA to leaf mRNA, deduced amino acid sequence nearly identical to chaperone DNA J, multiple copies, may encode? chaperone DNA J	28, 29
ce1		curled entangled1, dominant Ce1 plants have rolled leaves that tend to be entangled	95, 449
cfl2		complementary to *fl2*, recessive female gives floury with *fl2* pollen; heterozygous female gives normal phenotype	441, 442
cfr1	1.04+-0.03	coupling factor reduction1, chloroplast ATP synthase affected; seedlings pale green and greatly reduced in vigor	150
cg1	3.02+-0.01	corngrass1, semidominant *Cg1* plants have narrow leaves, extreme tillering	549
cg2	3.02+-0.01	corngrass2, dominant *Cg2* plants have narrow leaves, high tillering; mutable	334
cgl1		*Colletotrichum graminicola* resistance1, dominant *Cgl1* plants are resistant	20
cgx1		chloroplast gene expression1, reduced RUBISCO, thylakoid polypeptides, chloroplast rRNA, mRNA's appear normal and mostly associated with polysomes	25
cgx2		chloroplast gene expression2, reduced RUBISCO and thylakoid polypeptides; plastid mRNA's, rRNA's normal and mostly associated with polysomes	25
ch1	2.1+-0.01	chocolate pericarp1, dominant *Ch1* ears have tan to dark brown pericarp and cob	11
chs1		chitin synthase homolog1, partially sequenced cDNA to endosperm mRNA, homologous to chitin synthase of Candida albicans, may encode? chitin synthase	237
cif1		cross-incompatibility(female)1, cross-incompatibility when homozygous *cif1* female is crossed with male homozygous recessive for *cim1* and *cim2*	479
cim1		cross-incompatibility(male)1, reduced seed set when male parent is homozygous recessive *cim1* and *cim2* and female parent is homozygous recessive *cif1*	479
cim2		cross-incompatibility(male)2, reduced seed set when male parent is homozygous recessive *cim1* and *cim2* and female parent is homozygous recessive *cif1*	479
Cin		Cinteotl corn insert: repetitive sequences dispersed in the genome	223
ck2		casein kinase2, partial cDNA has three regions of identity to all other known casein kinase 2 alpha subunit genes, encodes casein kinase	136a
cl1	3.04+-0.01	chlorophyll1, white to green seedlings, depending upon alleles of modifier *clm1*; pale yellow endosperm	168
cld1		cold regulated protein homolog1, (was *csu19*) cDNA to leaf mRNA, strong homology to barley cold-regulated protein2, may encode? cold-regulated protein	29
clh1		histone lc1, electrophoretic mobility, encodes histone lc	592
clm1	8.06+-0.05	modifier of *cl1*, dominant *Clm1* alleles confer greening in cl1 seedlings but do not restore endosperm carotenoids	168
clp1		CLP protease homolog1, (was *uaz227*) partially sequenced cDNA to endosperm mRNA, identical to chloroplast Clp ATP-dependent protease, may encode? Clp ATP-dependent protease, chloroplast	236
clt1	8.05+-0.02	clumped tassel1, dominant *Clt1* plants have variable dwarfing, developmental anomalies	199, 419
clx1		calnexin homolog1, (was *csu148*) low copy number, cDNA to leaf mRNA, strong homology to *Arabidopsis* calnexin, may encode? calnexin	29
cm1		chloroplast modifier1, white or yellow stripes on leaves (compare *ij1*); conditions chloroplast modifications that are maternally inherited	594
cms-C		cytoplasmic male sterility, female-transmitted male sterility, C type; restored by *Rf4*	37, 148, 614
cms-S		cytoplasmic male sterility, female-transmitted male sterility, S type; restored by *Rf3*	148, 273, 276
cms-T		cytoplasmic male sterility, female-transmitted male sterility, Texas type; restored by *Rf1 Rf2*	148, 273, 276
cms-Y		cytoplasmic male sterility, female-transmitted male sterility, Y type; partially restored by *Rf7*	123
cp1	7.01+-0.01	collapsed1, endosperm collapsed and partially defective	331, 332
cp2	4.03	crumpled2, shrunken sugary endosperm; white seedling with green stripes	410, 423, 425
cp3	1.07+-0.01	collapsed kernel3, variably collapsed floury non-pigmented nonviable kernel; double mutant combination with mn4 has orange pericarp	426
cps1	1.12+-0.04	chloroplast protein synthesis1, reduced levels of RUBISCO and all thylakoid membrane complexes; unaltered chloroplast mRNA; decreased chloroplast polysomes	23
cps2	6.07+-0.04	chloroplast protein synthesis2, 20-fold reduced RUBISCO, 2-fold reduced thylakoid polypeptides, decreased chloroplast polysomes	23
cr1	3.02	crinkly leaves1, plant short; leaves broad, crinkled, foreshortened	162
cr4		crinkly leaf4, crinkly seedling leaves; plants short with rough, extremely crinkly leaves and club tassel; aleurone mosaic	587, 589
crp1	7.05+-0.02	chloroplast RNA processing1, (was *hcf136*; *hcf111* allelic); fails to accumulate monocistronic petB and petD mRNA's; lacks cytochrome bf	25, 115
crp2		chloroplast RNA processing2, fails to degrade group II introns in chloroplast	25

SYMBOL	BIN	NAME, PHENOTYPE	REF
cry1		crystal proteinIA(b)1, synthetic gene, CaMV 35S promoter, transferred by microprojectile bombardment, confers dominant resistance to European corn borer, encodes modified delta endotoxin, *Bacillus thuringiensis*	298
cry2		crystal proteinIA(b)2, synthetic gene with PEPC promoter, transferred, with *cry3*, by microprojectile bombardment, confers dominant resistance to European corn borer, encodes modified delta endotoxin, *Bacillus thuringiensis*	298
cry3		crystal proteinIA(b)3, synthetic gene, pollen specific promoter, transferred, with *cry2*, by microprojectile bombardment, confers pollen specific resistance to European corn borer, encodes modified delta endotoxin, *Bacillus thuringiensis*	298
csp1		white spot1, tiny to medium elliptical nearly transparent spots, scattered on leaf blade beginning at 8-leaf stage.	296
csu173(gfu)	5.07	gene specific probe, partially sequenced cDNA to leaf mRNA	28, 29
csu39(gfu)	4.09	gene specific probe, partially sequenced cDNA to leaf mRNA	28, 29
csu43(gfu)	9.04	gene specific probe, partially sequenced cDNA to leaf mRNA	28, 29
csu70(gfu)	6.01	partially sequenced cDNA to leaf mRNA, single map site	28, 29
ct1	8.03+-0.01	compact plant1, semi-dwarf plant, ear furcated	199, 401
ct2	1.01	compact plant2, semi-dwarf plant with club tassel	203
cta1		chitinase *a1*, cDNA sequence corresponds to peptide sequence of maize 28kDa chitinase A, encodes chitinase	257
ctb1		chitinase *B1*, cDNA sequence corresponds to protein sequence of maize chitinase B, encodes chitinase	257
cto1		cob turned out1, ear inverted to a sheet or tube, kernels internally placed; variable expression	514, 612
cx1		catechol oxidase1, electrophoretic mobility; null allele is known; monomeric; no hybrid bands, encodes catechol oxidase	470, 473
Cy	5.09+-0.01	Cycler: regulatory element mediating *bz1-rcy*	525
cyp1		cytochrome P450 homolog1, (was *csu25*) cDNA to leaf mRNA, strong homology to rat cytochrome P450, may encode? cytochrome P450	28, 29
d1	3.03	dwarf plant1, andromonoecious, short, compact plants; responds to gibberellins; *d1-t* intermediate in height	155
d2	3.05+-0.05	dwarf plant2, like *d1*	597
d3	9.03	dwarf plant3, like *d1*	129
d5	2.02+-0.02	dwarf plant5, like *d1*	597
d8	1.11	dwarf plant8, dominant *D8* plants resemble *d1*; not responsive to gibberellins; (compare *Mpl1*, probable allele)	461
d9	5.02+-0.02	dwarf plant9, dominant *D9* plants semidwarf with broad, dark green leaves; not andromonoecious, not responsive to gibberellins	409, 411
da1	9.05+-0.04	dilute aleurone1, aleurone color diluted	175
dap1	5.12+-0.01	dappled aleurone1, dominant *Dap1* kernels show patches of normal and abnormal aleurone cells; effect with colored aleurone is conspicuous	589
Def		deficiency, general symbol for a loss of a chromosome segment	356
Def(Kn1)O		deficiency of *Kn1*, deletion of *Kn1* but not of *adh1* or *lw1*; fails to pass through the male gametophyte; hemizygotes with TB-1La are embryo lethal; also deletes *knox3* homeobox gene, very similar in sequence and expression pattern to *kn1*	556
dek1	1.04	defective kernel1, germless; floury endosperm; anthocyanins and carotenoids absent; cultured embryos not obtained	423, 424
dek2	1.12+-0.04	defective kernel2, discolored, scarred endosperm; lethal; cultured embryos green	423, 424
dek3	2.03+-0.02	defective kernel3, germless; cultured embryos white with green stripe	423, 424
dek4	2.08+-0.03	defective kernel4, germless; floury endosperm; cultured embryos green, narrow leaved	423, 424
dek5	3.02+-0.02	defective kernel5, shrunken endosperm; white seedling with green stripes	423, 424
dek6	3.08+-0.02	defective kernel6, shrunken endosperm; lethal; cultured embryos normal	423, 424
dek8	4.09+-0.03	defective kernel8, shrunken endosperm; lethal; cultured embryos green, small	423, 424
dek9	5.1+-0.04	defective kernel9, crumpled endosperm; lethal; anthocyanins and carotenoids reduced; cultured embryos not obtained	423, 424
dek10	4.08+-0.04	defective kernel10, collapsed endosperm; lethal; cultured embryos green, curled, stubby	423, 424
dek11	4.02+-0.02	defective kernel11, etched endosperm; lethal; cultured embryos white with green stripes	423, 424
dek12	9.02+-0.02	defective kernel12, collapsed endosperm; lethal; cultured embryos green, narrow-leaved, curled	423, 424
dek13	9.06+-0.03	defective kernel13, defective opaque endosperm; lethal; cultured embryos pale green with green stripes	423, 424
dek14		defective kernel14, collapsed endosperm; lethal; cultured embryos yellow-green	423, 424
dek15		defective kernel15, collapsed floury endosperm; lethal; cultured embryos green	423, 424
dek16	2.08+-0.03	defective kernel16, floury endosperm; lethal; cultured embryos normal	541
dek17	3.07+-0.03	defective kernel17, collapsed endosperm; lethal; cultured embryos not obtained	541
dek18	5.03+-0.03	defective kernel18, collapsed endosperm; lethal; cultured embryos green, narrow-leaved	541
dek19	6.07+-0.04	defective kernel19, collapsed opaque endosperm; lethal; cultured embryos green	541

SYMBOL	BIN	NAME, PHENOTYPE	REF
dek20	8.05+-0.02	defective kernel20, collapsed endosperm; lethal; cultured embryos green	541
dek22	1.12+-0.04	defective kernel22, collapsed endosperm; lethal; cultured embryos not obtained	100, 542
dek23	2.08+-0.03	defective kernel23, defective crown; lethal; cultured embryos not obtained	100, 542
dek24	3.02+-0.02	defective kernel24, collapsed endosperm; lethal; cultured embryos normal	542
dek25	4.01+-0.01	defective kernel25, shrunken endosperm; lethal; cultured embryos normal	542
dek26	5.1+-0.04	defective kernel26, collapsed endosperm; lethal; cultured embryos normal	542
dek27	5.1+-0.04	defective kernel27, collapsed endosperm; lethal; cultured embryos green	542
dek28	6.02+-0.02	defective kernel28, opaque endosperm	542
dek29	8.06+-0.02	defective kernel29, collapsed endosperm; viable; cultured embryos green, narrow-leaved	542
dek30	9.06+-0.03	defective kernel30, floury endosperm; lethal; cultured embryos green, narrow-leaved	542
dek31	4.07	defective kernel31, pitted endosperm; lethal	540
dek32	1.03+-0.03	large shrunken floury nonviable kernel	413
dek33	5.07	defective kernel33, opaque, floury, dented, wrinkled kernel with floury endosperm; occasionally viviparous	413
dep1	6.05+-0.05	defective pistils1, female florets have abnormal structure; the ovaries form two or more short defective pistils that do not function	372
des17	8.07+-0.04	defective seedling17, reduced height, partial suppression of primary root growth, contorted leaves, lethal	188
dHbr		defective Heartbreaker, Heartbreaker: element similar to Tourist	269
dia1	2.06	diaphorase1, electrophoretic mobility; cytosolic; monomeric, encodes dihydrolipoamide dehydrogenase	627
dia2	1.14+-0.01	diaphorase 2, electrophoretic mobility; cytosolic; dimeric, encodes dihydrolipoamide dehydrogenase	627
dib1		dichotomously branched1, main axis branches into two normal tops, most often at node 4-8 but variable; associated with aneuploidy	370, 371, 373
dlf1		delayed flowering1, tall late plant with additional nodes and leaves at flowering; no apparent response to day length	429
dp1	4.11+-0.01	distal pale1, seedling leaf tip virescent	12
dps1		dihydrodipicolinate synthase1, cDNA complements *E. coli* mutant (AT997dapA-) blocked in DHPS, encodes dihydrodipicolinate synthase	187
Ds		dissociation, designator for transposable factors regulated by *Ac*; modifies gene function and/or chromosome breakage (termed "*Ds-2*"); *Ds2* designates element isolated from *Adh1-2F11*	359, 360
Ds-r		*Ds* element equipped with bacterial plasmid sequences to permit rescue from the plant genome	504a
dsc1	4.02+-0.02	discolored kernel1, crumpled, discolored, germless lethal	258, 521
Dsl		State I *Ds*, One of the two "states" of *Ds*, generates a high frequency of chromosome breaks. Molecular evidence is consistent with McClintock model of locally repeated *Ds* elements.	359
dSpm		designator for transposable factors regulated by *Spm*	523
dsy1		desynaptic1, male and female sterility; synapsis and chiasmata occur, followed by separation after pachytene	122, 209
dsy2		desynaptic2, like *dsy1*	207
dsy3		desynaptic3, like *dsy1*	206
dsy4		desynaptic4, like *dsy1*	206
Dt1	9	Dotted1, regulates controlling element at *A1*; responding *a1*-m alleles express colored dots on colorless kernels and purple sectors on brown plants, encodes *Dt* transposase	481
Dt2	6.05+-0.01	like *Dt1*	437
Dt3	7.05+-0.02	like *Dt1*, but expression variable	437
Dt4	4.06+-0.06	like *Dt1*, but dots chiefly on crown of kernel	139
Dt5	9	like *Dt1*	139
Dt6	4.03+-0.01	like *Dt1*	568
dts1		aspartyl-tRNA synthetase1, (was *uaz131*) cDNA to endosperm mRNA, strong homology to rat aspartyl-tRNA synthetase, alpha-2 subunit, encodes aspartyl-tRNA synthetase, alpha-2 subunit	237
du1		dull endosperm1, glassy, tarnished endosperm; affects soluble starch synthase and branching enzyme IIa	166, 343
dv1		divergent spindle1, chromosomes unoriented at metaphase I; partial male and female sterility	98, 99
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	400
dzs10	9.03	delta zein10, [was *Zps10/(22)*]; high (22.5%) methionine 10kD zein; RFLP (probe 10kZ-1), encodes 10-kDa zein (delta zein)	44
dzs23	6.06	delta zein 23, genomic sequence similar to dzs10, deduced methionine content 26%, primer extension indicates expressed at low levels in B37 but not expressed in line BSSS-53, encodes delta zein, 23kDa	598
e1	7.05	esterase1, electrophoretic mobility; null allele is known; dimeric; intralocus hybrid bands occur, encodes esterase	279

SYMBOL	BIN	NAME, PHENOTYPE	REF
e2		esterase 2, presence-absence only, encodes esterase	533
e3	3.05+-0.05	esterase3, electrophoretic mobility; dimeric; intralocus hybrid bands occur, encodes esterase	529
e4	3.04	esterase4, electrophoretic mobility; null allele is known; monomeric, encodes esterase	230
e5(I)		esterase, electrophoretic mobility, duplicate factor with E5(II), encodes esterase	336, 337
e5(II)		esterase, electrophoretic mobility; duplicate factor with E5-(I), encodes esterase	336, 337
e6		esterase6, presence-absence only, encodes esterase	336, 337
e7		esterase7, presence-absence only, encodes esterase	336, 337
e8	3.01	esterase8, electrophoretic mobility; null allele is known; dimeric; intralocus hybrid bands occur, encodes esterase	336, 337
e9		esterase9, electrophoretic mobility; null allele is known, encodes esterase	336, 337
e10		esterase 10, electrophoretic mobility, encodes esterase	336, 337
eg1	5.1+-0.04	expanded glumes1, glumes open at right angle	75
eif5		elongation initiation factor 5, cDNA to leaf mRNA, strong homology to yeast and rat tranlation initiation factor eIF-5, may encode? elongation initiation factor 5	28, 29
el1	8.07+-0.04	elongate1, chromosomes uncoiled during meiotic metaphase and anaphase in male and female; frequent unreduced gametes	205, 486
elf1		elongation factor1, (was *csu116*) multiple copies; cDNA to leaf mRNA, partial sequence identical to tomato elongation factor 1-alpha, encodes elongation factor 1-alpha subunit	285, 489
elf2		elongation factor2, (was *uaz161*) cDNA to endosperm mRNA, homologous to *Trypanosoma* elongation factor 1-gamma, may encode? elongation factor 1-gamma	236
emb1	1.04+-0.03	embryo development blocked at late proembryo to early transition stage	101
emb2	9.02+-0.02	embryo development blocked at the transition stage	101
emb3	4.09+-0.03	embryo development blocked at transition stage; suspensor bulbous; embryo proper enlarged	101
emb4	1.04+-0.03	embryo development blocked at late transition stage	101
emb5	2.08+-0.03	embryo development blocked at transition to early coleoptilar stage	101
emb6	4.09+-0.03	embryo development blocked at coleoptilar stage	101
emb7	1.04+-0.03	embryo development blocked at the transition stage through stage 2	101
emb8	4.09+-0.03	embryo development blocked at coleoptilar stage or later	101
emb9	3.07+-0.03	embryo development blocked at the transition stage or later	101
emb10	1.04+-0.03	embryo development blocked during elaboration of embryonic structures (stage 4)	101
emb11	4.09+-0.03	embryo development blocked from stage 3 to stage 6; small embryo	101
emp1	1.04+-0.03	empty pericarp1, germless, unfilled kernel	521
emp2	2.06	empty pericarp2, germless, unfilled kernel	258, 521
emp3	8.09+-0.01	empty pericarp3, small, extremely collapsed, defective, poorly viable kernel	167, 429
En1		enhancer, enhancer: transposable element (equivalent to *Spm*); autonomous, regulates I (=*dSpm*) transposition (e.g. at *g2-m* = *pg-m* = *pg14-m*)	458, 459
end1		early nodulin homolog1, (was *uaz227*) cDNA to endosperm mRNA, very strong homology to soybean early nodulin mRNA	237
eno1	9.03	enolase1, cDNA clone pZm245 complements enolase mutant in *E. coli*, encodes enolase	307
enp1	6.01	endopeptidase1, electrophoretic mobility; null allele is known; monomeric, encodes endopeptidase	368
esp5		embryo specific protein5, (was *Emb5*); cDNA clone, embryo specific, ABA responsive, deduced amino acid sequence very hydrophilic and gly/glu-rich	637
et1	3.09+-0.01	etched1, pitted, scarred endosperm, virescent seedling; plastid membranes altered	229, 577
et2	2.03+-0.02	etched2, endosperm etched; seedlings off-white albino, with occasional greening of leaf tips	590
ets1		*ets*-family transcription factor homolog *csu110*, (was *csu110*) cDNA to leaf mRNA, multiple copies, partial sequence homologous to human *Ets*-related transcription factor, may encode? *ets*-family transcription factor	28, 29
f1	1.09	fine stripe1, virescent seedling, fine white stripes on base and margin of older leaves	128, 321, 322
fae1		fasciated ear1, small, rounded ears branched at their tips	540
fas1		fascicled ear1, in *Fas1*, repeated dichotomous branching in floral meristems	624
fbr1		few-branched1, dominant *Fbr1* plants have tassel reduced to 0-3 branches; bract replaces next-to-bottom branch	407
Fcu		factor Cuna: controlling element of *r1-cu*	213
fdx1		ferredoxin1, chloroplast ferredoxin, light induced, N-terminal amino acid sequence of mature protein, cDNA sequence, encodes ferredoxin	231
fdx3		ferredoxin3, ubiquitous, cDNA clone, gene specific probe, amino acid sequence, encodes ferredoxin	231
fdx5		ferredoxin5, leaf protein, cDNA clone, distinct amino acid sequence compared to other ferredoxins, gene specific probe, encodes ferredoxin	231
fer1		ferritin1, iron induced, cDNA sequences and expression pattern indicate two genes, *fer1*, *fer2* (Lobreaux et al 1992), encodes ferritin	327
fer2		ferritin homolog2, iron induced, cDNA sequences, differential expression indicate 2 genes(Lobreaux et al. 1992), may encode? ferritin	327

SYMBOL	BIN	NAME, PHENOTYPE	REF
fgs1		ferredoxin-dependent glutamate synthase1, deduced amino acid sequence homologous with *E. coli* NADHP-glutamate synthase, single copy by Southern blot analysis, encodes glutamate synthase, ferredoxin dependent	509
fl1	2.05	floury endosperm1, (was *o4*) endosperm opaque, soft; dosage effect with *fl1*-ref allele, but *o4* allele is recessive	234
fl2	4.03+-0.01	floury2, endosperm opaque, soft; dosage effect	166, 397
fl3	8.05+-0.01	floury3, endosperm opaque, soft; dosage effect	335, 398
ftr1		ferredoxin-thioredoxin homolog1, cDNA sequence, may encode? ferredoxin-thioredoxin reductase, chloroplast	343a
g1		golden plant1, seedling and plant with distinctive golden yellow cast; stub of cut seedling displays golden vs. green distinguishably	155, 157
g2	3.01	golden plant2, (was *g5*, *pg14*, *v19*, *pg-m*) pggolden pale-green, weak plants; sheaths whitish yellow-green; *pg-m* of Peterson is mutable allele carrying *En*	260, 459
g6	9.03	golden plant6, dominant *G6* plants golden; lighter yellowish sheaths	405
ga1	4.01+-0.01	gametophyte factor1, (was *ga9*) *Ga1* pollen grains are competitively superior to *ga1* on *Ga1* silks; *Ga1-S* super-gametophyte	274
ga10	5.07+-0.07	gametophyte factor10, *Ga10* pollen grains competitively superior to *ga10*; map position inconsistent with *ga2*	212
ga2	5.07	gametophyte factor2, *Ga2* pollen grains are competitively superior to *ga2*	73
ga7	3.1	gametophyte factor7, *ga7* pollen from heterozygotes is only 10-15% functional regardless of silk genotype	483
ga8	9.03	gametophyte factor8, *Ga8* pollen grains are competitively superior to *ga8* on *Ga8* silks	527
gbp1		GTP-binding protein homolog1, (was *csu108*) cDNA to leaf mRNA, partial sequence homologous to canine GTP-binding protein RAB5, may encode? GTP binding protein	285
gcb1		GC binding protein 1, binds to anaerobic responsive element (ARE) of *Adh1* promoter	439
gdh1	1.13	glutamic dehydrogenase1, electrophoretic mobility; null allele is known (cold sensitivity); intra/interlocus hybrid bands occur, encodes glutamic dehydrogenase	471
gdh2		glutamic dehydrogenase2, electrophoretic mobility; intralocus hybrid bands occur, encodes glutamic dehydrogenase	214
geb1		glucan endo-1,3-beta-glucosidase homolog1, cDNA sequence homologous to 1,3-beta glucanase, may encode? glucan endo-1,3-beta-glucosidase	646a
gl1	7.02	glossy1, cuticle wax altered; leaf surface bright, water adheres	233, 305
gl2	2.03+-0.01	glossy2, like *gl1*, but surface wax is all rice-grain-type particles	46, 233
gl3	4.08	glossy3, like *gl1*, but surface wax has all rice-grain-type particles	233
gl4	4.06	glossy4, (was *gl16*) like *gl1*, but surface wax has reduced number of star-type particles	46, 566
gl5	4.03+-0.01	glossy5, like *gl1*; duplicate factor with *gl20*. Double homozygote phenotypically like *gl1*, but cuticle wax consists of large elongated particles.	46, 166, 566, 571
gl6	3.05	glossy6, like *gl1*, but seedling leaf surface bright green instead of bluish	166, 566
gl7		glossy7, (was *gl12*) like *gl1*	166, 566
gl8	5.09+-0.01	glossy8, (was *gl10*) like *gl1*; cuticle wax in rice-grain-type particles	166, 566
gl9	3.07+-0.03	glossy9, expression poor	166, 566
gl11	2.04+-0.01	glossy11, like *gl1*; abnormal seedling morphology, sometimes viviparous	46
gl13	4.09+-0.03	glossy 13, glossy leaf	574
gl14	2.06+-0.05	glossy14, like *gl1*; duplicate factor with *gl24*; expressed late	10
gl15	9.04	glossy15, glossy leaf surface expressed after 3rd leaf	10
gl17	5.06+-0.01	glossy17, like *gl1*, but semi-dwarf with necrotic crossbands on leaves	487
gl18	8.03+-0.02	glossy18, like *gl1*; expression poor	10, 573
gl19	3.02+-0.02	glossy19, like *gl1*; barren plant with no ear or tassel	416
gl20		glossy20, like *gl1* (duplicate factor with *gl5*)	571
gl21		glossy21, like *gl1*, duplicate factor with *gl22*	416
gl22		glossy22, like *gl1*, duplicate factor with *gl21*	408
gl24		glossy24, like *gl1*; duplicate factor with *gl14*, best at 4-leaf stage	573
glb1	1.1	globulin1, (was *prot1*) electrophoretic mobility; null allele is known; embryo protein, encodes globulin, 63,000 kDa	300, 531
glb2		globulin2, presence-absence, encodes globulin, 45,000 kDa	300
gln2		glutamine synthetase2, cytosolic, root specific, gene specific cDNA probe, 6-member nuclear gene family, encodes glutamate--ammonia ligase, cytosol	510
gln3		glutamine synthetase3, cytosolic, minor species, specific to young seedlings, gene specific cDNA probe, 6-member nuclear gene family, encodes glutamate--ammonia ligase, cytosol	510, 558
gln4		glutamine synthetase4, cytosolic, major species in both root and leaf, gene specific cDNA probes, 6-member nuclear gene family, encodes glutamate--ammonia ligase, cytosol	510, 558
gln5		glutamine synthetase5, cytosolic, major species in both leaf and root, gene specific cDNA probe, 6-member nuclear gene family, encodes glutamate--ammonia ligase, cytosol	510
gln6		glutamine synthetase6, cytosolic, a major species in root, gene specific cDNA probe, 6-member nuclear gene family, encodes glutamate--ammonia ligase, cytosol	510, 558

SYMBOL	BIN	NAME, PHENOTYPE	REF
gln7		glutamine synthetase7, chloroplast, gene specific cDNA probe, 6-member nuclear gene family, encodes glutamate--ammonia ligase, chloroplast	558
glu1		beta glucosidase1, electrophoretic mobility; cytosolic; dimeric; intralocus hybrid bands occur, encodes beta glucosidase	279, 472
gn1	2.11	gnarley1, dominant (*Gn1*) characterized by reduced internodal length, sinuously curving culm, lack of distinct boundary between blade and sheath, extra silks	180
got1	3.07+-0.01	glutamate-oxaloacetate transaminase1, electrophoretic mobility; null allele is known; glyoxysomal; dimeric; intralocus hybrid bands occur, encodes aspartate aminotransferase	279, 517
got2	5.12	glutamate-oxaloacetate transaminase2, electrophoretic mobility; null allele is known; plastidial; dimeric; intralocus hybrid bands occur, encodes aspartate aminotransferase	216
got3	5.06+-0.01	glutamate-oxaloacetic transaminase3, electrophoretic mobility; null allele is known; mitochondrial; dimeric; intralocus hybrid bands occur, encodes aspartate aminotransferase	216
gpa1	10.06	glyceraldehyde-3-phosphate dehydrogenase1, chloroplastic, A subunit, encodes glyceraldehyde-3-phosphate dehydrogenase (NADP+) (phosphorylating)	65, 506
gpb1		glyceraldehyde phosphate dehydrogenase *B1*, (was *csu152*) cDNA to leaf mRNA, strong homology to Arabidopsis GapB, may encode? glyceraldehyde-3-phosphate dehydrogenase (NADP+) (phosphorylating)	285, 489
gpc1	4.04	glyceraldehyde-3-phosphate dehydrogenase1, 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	65, 345, 506
gpc2	6	glyceraldehyde-3-phosphate dehydrogenase2, 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	506
gpc3	4.04	glyceraldehyde-3-phosphate dehydrogenase3, cytosolic, C subunit 3; coding sequence homology to *gpc4*, unique 3' untranslated region, encodes glyceraldehyde-3-phosphate dehydrogenase C, cytosolic	506
gpc4	5.07	glyceraldehyde-3-phosphate dehydrogenase4, C subunit, electrophoretic mobility, coding sequence homology to gpc3, unique 3' untranslated region, encodes glyceraldehyde-3-phosphate dehydrogenase C, cytosolic	507
grf1		G-box regulatory factor1, G-box binding factor, with bZIP motif, encodes G-box binding factor14	134a
grp1		1, protein with high glycine content and repetitive glycine stretches; putative cell wall components, encodes glycine-rich protein	135
grt1	5.1+-0.04	green tip1, pale yellow seedling with green first leaf tip; lethal	416
grx1		glutaredoxin homolog1, (was *csu40*) cDNA to leaf mRNA, strong homology to E. coli glutaredoxin, may encode? glutaredoxin	28, 29
gs1	1.12	green stripe1, grayish green stripes between vascular bundles on leaves; tissue wilts	157, 160, 381
gs2	2.04+-0.01	green stripe2, like *gs1*, but pale green stripes; no wilting	166, 565, 566
gs3	6.07+-0.04	green stripe3, like *gs2* but much smaller plant	229, 416
gs4		green stripe4, dominant *Gs4* plants are like *gs1* but smaller plant	409
gsr1	1.02	glutathione reductase1, (was *csu111*) single copy; cDNA to leaf mRNA, very strong homology to pea glutathione reductase, encodes glutathione reductase	28, 29
gss1		starch synthase homolog1, (was *uaz218*) cDNA to endosperm mRNA, nearly identical to pea starch synthase isoform II, may encode? starch synthase	236
gst1		glutathione-S-transferase1, presence-absence of isozyme bands with the highest activity, between inbred lines; members of a family of polymorphic bands, encodes glutathione S-transferase	513
gt1	1.08+-0.08	grassy tillers1, numerous basal branches; vegetatively totipotent in combination with *id1* and factors for perennialism	536, 537
h1	3.03	soft starch1, endosperm opaque, starchy (like floury), recessive to horny	393
hca1		histocompatibility antigen homolog1, (was *uaz199*) cDNA to endosperm mRNA, homologous to human histocompatibility antigen, may encode? glycoprotein	236
hcf1	2.08+-0.03	high chlorophyll fluorescence1, affects NADP+ oxidoreductase; green seedling	317, 375
hcf2	1.12+-0.04	high chlorophyll fluorescence2, missing cytochrome bf complex; yellow-green seedling	375
hcf3	1.04	high chlorophyll fluorescence3, (was *hcf9*) missing PSII thylakoid membrane core complex; green seedling	375
hcf4		high chlorophyll fluorescence4, affects CO2 fixation; green seedling	377
hcf5	6.02+-0.02	high chlorophyll fluorescence5, affects PSII reaction; green seedling	376
hcf6	1.04+-0.03	high chlorophyll fluorescence6, missing cytochrome bf complex; green seedling	317
hcf7	1.12+-0.04	high chlorophyll fluorescence 7, defective processing of 16S rRNA. Pigmentation near normal due to normal accumulation of light harvesting complexes. Deficient in many thylakoid membrane proteins.	23, 25
hcf11		high chlorophyll fluorescence11, pale green leaves, deficient in CO2 fixation, often lethal at 3- to 5-leaf stage	380
hcf12	1.12+-0.04	high chlorophyll fluorescence12, green seedling	317

SYMBOL	BIN	NAME, PHENOTYPE	REF
hcf13	1.12+-0.04	high chlorophyll fluorescence13, affects CO2 fixation; green seedling	317
hcf15	2.08+-0.03	high chlorophyll fluorescence15, affects photophosphorylation; yellow-green seedling, may survive	317
hcf18	5.1+-0.04	high chlorophyll fluorescence18, major loss of PSI; other thylakoid complexes reduced; yellow-green seedling	376
hcf19	3.07+-0.03	high chlorophyll fluorescence19, affects PSII thylakoid membrane core complex; green/yellow-green seedling	317
hcf21	5.1+-0.04	high chlorophyll fluorescence21, affects CO2 fixation, Rubisco; green seedling	376, 377
hcf23	4.03+-0.02	high chlorophyll fluorescence23, affects photophosphorylation; yellow-green seedling, may survive	317
hcf26	6.02+-0.02	high chlorophyll fluorescence26, affects electron transport; yellow-green, viable seedling	317
hcf28		high chlorophyll fluorescence28, affects CO2 fixation; green seedling	376
hcf31	1.04+-0.03	high chlorophyll fluorescence31, missing chlorophyll a/b binding protein; yellow-green seedling	376
hcf34	6.07+-0.04	high chlorophyll fluorescence34, affects photophosphorylation; yellow-green seedling	317
hcf36	6.07+-0.04	high chlorophyll fluorescence36, affects electron transport; green seedling	376
hcf38	5.1+-0.04	high chlorophyll fluorescence38, affects cytochrome bf complex, alpha and beta components of CF1; green seedling	317
hcf41	1.12+-0.04	high chlorophyll fluorescence41, affects PSII thylakoid membrane core complex; green seedling	317
hcf42	9.06+-0.03	high chlorophyll fluorescence42, affects Rubisco; green/yellow-green seedling	377
hcf43	5.1+-0.04	high chlorophyll fluorescence43, yellow-green leaves, deficient in all thylakoid polypeptides except for the antenna complexes	376, 379
hcf44	1.12+-0.04	high chlorophyll fluorescence44, affects PSI membrane core complex; pale-green seedling, lethal	377
hcf45		high chlorophyll fluorescence45, CO2-fixation reduced 90%, normal levels of RUBISCO protein	377, 380
hcf46	3.07+-0.03	high chlorophyll fluorescence46, ultraviolet light red fluorescence	317
hcf47		high chlorophyll fluorescence47, affects cytochromes; yellow-green seedling	376
hcf48	6.07+-0.04	high chlorophyll fluorescence48, affects electron transport; yellow-green seedling	376
hcf49		high chlorophyll fluorescence49, PSI mutant, not allelic to *hcf44* or *hcf50*	377, 378
hcf50	1.12+-0.04	high chlorophyll fluorescence50, missing PSI thylakoid membrane core complex; seedling slightly pale green	377
hcf60		high chlorophyll fluorescence60, green to pale green seedling leaves, deficiency in photosystem I	378
hcf101	7.05+-0.02	high chlorophyll fluorescence101, affects PSI thylakoid membrane core complex	376
hcf102	8.07+-0.04	high chlorophyll fluorescence102, affects cytochrome bf complex	115, 378
hcf103	7.05+-0.02	high chlorophyll fluorescence103, (was *hcf114*) reduced photosystem II activity due to absence of plastoquinone (PQ-9)	115
hcf104	7.05+-0.02	high chlorophyll fluorescence104, photosystem I-deficient	114, 115
hcf106	2.06	high chlorophyll fluorescence106, affects PSI, PSII, cytochrome bf, encodes chloroplast thylakoid membrane protein	344
hcf108	5.01+-0.01	high chlorophyll fluorescence108, ATPase-deficient	115
hcf113	9.02+-0.02	high chlorophyll fluorescence113, multiple effects; yellow-green seedlings	114
hcf120		high chlorophyll fluorescence120, cytochrome bf and photosystem II deficient	602
hcf316		high chlorophyll fluorescence316, affects chlorophyll a/b binding protein; yellow-green seedling	376
hcf323	6.02+-0.02	high chlorophyll fluorescence323, affects photophosphorylation, coupling factor; green seedling	376
hcf408	6.07+-0.04	high chlorophyll fluorescence408, affects chlorophyll a/b binding protein; yellow-green seedling	376
hex1	3.02+-0.01	hexokinase1, electrophoretic mobility; null allele is known; cytosolic; monomeric, encodes hexokinase	628, 630
hex2	6.05	hexokinase2, electrophoretic mobility; null allele is known; cytosolic; monomeric, encodes hexokinase	629, 630
hfi1		corn-activated Hageman factor inhibitor1, cDNA clone corresponds to partial amino acid sequence; expression in yeast confirms product inhibits trypsin, encodes corn(activated) Hageman factor inhibitor	625
his1		histone H1 gene family, cDNA sequence, encodes histone I	480
his2b1		histone2b1, cDNA to mRNA from 8 day seedlings, protein reacts with antibodies for histone H2B, encodes histone 2B	268
his2b2		histone2b2, cDNA to mRNA from seedling, protein reacts with histone2B antibodies, encodes histone 2B	268
his3		histone H3 family, 60-80 copies/diploid genome, homologous sites on several chromosomes, subfamilies H3C2, H3C3, H3C4, encodes histone 3	91
his4		histone4 family, histone H4 family; 100-120 copies/diploid genome (Chaubet et al 1986), homologous sites on most chromosomes, subfamilies H4C7, H4C14, encodes histone 4	91, 92

SYMBOL	BIN	NAME, PHENOTYPE	REF
hm1	1.09	*Helminthosporium carbonum* susceptibility1, disease lesions vs. yellowish flecks (resistant) on leaves with *Cochliobolus carbonum* race 1, encodes NADPH HC-toxin reductase	269, 610
hm2	9.05+-0.01	*Helminthosporium carbonum* susceptibility2, Dominant *Hm2* plants resistant to *Cochliobolus carbonum*. Like *Hm1*; masked by *Hm1*	403
hox1	8.05	homeobox1, protein product binds to *sh1* promoter (feedback control element), is found in nuclei, encodes HOX1 protein	43
hox2	6.08	homeobox2, possibly = koln1B; similar to *hox1*, but sequence predicts not allelic to *hox1*, encodes HOX2 protein	43
hpt1		hygromycin phosphotransferase1, transgenic chimeric gene, single dominant locus, coding region origin E. coli introduced by particle bombardment, encodes hygromycin-B kinase	622
hrg1	2.05	hydroxyproline rich glycoprotein1, cDNA, genomic clones, peptide sequence, single site (Southern analysis), accumulates in dividing cells, preferentially in provascular cells, encodes hydroxyproline-rich glycoprotein	583
hs1	7	hairy sheath1, dominant *Hs1* plants have abundant hairs on leaf sheath throughout development	601
hsf1	5.12+-0.01	hairy sheath frayed1, dominant *Hsf1* plants have pubescent sheaths and leaf margins; liguled enations at leaf margins	47, 48
hsk1		high-sulfur keratin homolog1, (was *uaz144*) cDNA to endosperm mRNA, homologous to high-sulfur keratin, encodes high sulfur keratin homolog	236
hsp1		heat shock protein1, genomic clones, single copy (Southern blots), transcribed (Northern blots), transgenic (petunia) expression, encodes HSP70	501
hsp18a		18 kda heat shock protein18a, cDNA sequence, pollen specific, encodes 18 kDa heat shock protein	16
hsp18c		heat shock protein18c, cDNA, genomic clones, unique sequence, in vitro translated protein immunoreacts with anti-maize-HSP18 antibodies, encodes 18 kDa heat shock protein	217
hsp18f		heat shock protein18f, cDNA clone, unique sequence, in vitro translation product immunoreacts with maize-HSP18 antibodies, encodes 18 kDa heat shock protein	
hsp26	1.1	heat shock protein26, (was *umc195*) cDNA, single mRNA species induced by heat shock, in vitro HSP26 imported by isolated chloroplasts, cross-reacts with anti-pea-chloroplast-HSP21 antibodies, encodes heat shock protein 26	434
hsp60		heat shock protein60, cDNA sequence homologous to *hsp60* family, encodes mitochondrial chaperonin *hsp60*	469
ht1	2.09	*Helminthosporium turcicum* resistance1, dominant *Ht1* plants resistant to *Exserohilum turcicum*	244, 245
ht2	8.06	*Helminthosporium turcicum* resistance2, dominant *Ht2* plants resistant to *Exserohilum turcicum*	247
ht3		Helminthosporium turcicum resistance3, (from *Tripsacum floridanum*); dominant *Ht3* plants resistant to *Exserohilum turcicum*	248, 249
htm1		*Exserohilum turcicum* Mayorbela resistance1, dominant *Htm1* plants resistant	490
htn1	8.08	*Helminthosporium turcicum* resistanceN1, formerly HtN. Dominant *Htn1* plants resistant to *Exserohilum turcicum*	546
hupm1	10.03	hupm/hypb protein family homolog *csu103*, (was *csu103*) cDNA to leaf mRNA, homologous to hupm/hypb protein family, gene specific probe, may encode? hupm/hypb protein	29
hvp1		human viral protein homolog1, cDNA to endosperm mRNA, homologous to Epstein-Barr virus transcription activator, may encode? transcription factor	237
hyp1		hybrid proline-rich protein1, genomic sequence, embryo-specific expression; deduced amino acid sequence shows two domains: proline-rich with PPYV and PPTPRPS elements and hydrophobic, cysteine-rich domain, encodes hybrid proline-rich protein	275
I-R		inhibitor of *R*, excision is responsible for *R1*-st stippling; transposed element modifies stippling level	367
id1	1.1	indeterminate growth1, requires extended growth and short days for flowering; vegetatively totipotent with *gt1* and factors for perennialism	537, 548
idh1	8.07	isocitrate dehydrogenase1, electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur, encodes isocitrate dehydrogenase	216
idh2	6.1	isocitrate dehydrogenase2, electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur, encodes isocitrate dehydrogenase	216
ig1	3.06	indeterminate gametophyte1, low male fertility, polyembryony, heterofertilization, polyploidy, androgenesis (male and female affected)	293
ij1	7.03	iojap striping1, many variable white stripes and margin patterns on leaves (compare *cm1*); conditions chloroplast defects that are cytoplasmically inherited	259
ij2	1.13+-0.01	iojap striping2, like *ij1*; chloroplast inheritance unknown	416
in1	7.01	intensifier1, intensifies aleurone anthocyanin pigments; *In1-D* dominant dilute	182
Ins1	9.04	insertion located upstream of *bz1-R*; up to 50 copies in genome (Southerns)	477
Ins2	9.04	insertion2: 447 bp element upstream of *bz1-R*	477
Inv		inversion, general symbol for inversion of a segment of chromosome	
Irma		Irma receptor element, reduced *En*-related element; requires both *En* and *Med* for excision	395

SYMBOL	BIN	NAME, PHENOTYPE	REF
is1		cupulate interspace1, space between the apex of the cupule and the glume cushion above; trait characteristic of teosinte	190
isp1		iron-sulfur protein1, cDNA, nuclear-encoded mitochondrial Rieske iron-sulfur protein, functional analysis in yeast, encodes Rieske iron-sulfur protein, mitochondrial	252
isr1		inhibitor of striate1 (a.k.a. *Ej1*), dominant *Isr1* plants have reduced expression of *sr2* and other leaf-striping factors	294
j1	8.09+-0.01	japonica striping1, white stripes on leaf and sheath; not often expressed in seedling	157
j2	4.08+-0.01	japonica striping2, extreme white striping of leaves, etc.	166
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	354, 450
K10		knob, heterochromatic appendage on long arm of chromosome 10; neocentric activity distorts segregation of linked genes	330, 581
K3L	3.07	knob, heterochromatic structure	132
K9S	9	Heterochromatic knob on 9S, heterochromatic knob on 9S, found in some strains and not in others	355
kas1		beta-keto acyl synthase homolog1, partially sequenced cDNA to endosperm mRNA, homologous to *Streptomyces glaucescens* beta-keto acyl synthase, may encode? beta-keto acyl synthase homolog1	237
kn1	1.11	knotted1, dominant *Kn1* plants have localized proliferation of tissue at vascular bundles on leaf	69
knox3	1.11	homeobox gene3, very similar in sequence and expression pattern to *kn1*	556
knox4	2.11	knotted-like homeobox4, genomic clone identified by homology to *kn1* homeobox, gene specific probe; possibly identical to *gn1*	180
l1		luteus1, yellow pigment in white tissue of specific chlorophyll mutants *w1*, *w2*, *j1*, *ij1*, others	121, 320, 321
l3		luteus3, lethal yellow seedling	121, 265
l4		luteus4, lethal yellow seedling	121, 265
l6	9.02	luteus6, like *l4*	166, 175
l7	9.03	luteus7, yellow seedling and plant; lethal	121, 175
l10	6.02+-0.01	luteus10, like *l4*; fails to convert protochlorophyllide to chlorophyllide	495
l11	6.02+-0.02	luteus11, yellow seedling with green leaf tips; lethal	19
l12	6.02+-0.01	luteus12, like *l11*	121
l13		luteus13, dark yellow, lethal seedling; fails to convert protoporphyrin IX to Mg-protoporphyrin	346, 416
l15	6.03+-0.01	luteus15, like *l4*, lethal yellow seedling	499
l16	1.04+-0.03	luteus16, like *l4*; leaves bleach to paler yellow in patches	416
l17	1.12+-0.04	luteus17, like *l4*; leaves with lighter yellow crossbands	416
l18	2.08+-0.03	luteus18, like *l4*	416
l19		luteus19, like *l4*. Reference allele found in M2 from treatment with ethyl methanesulfonate, but mutable with duplicate factor transmission, suggesting a 2-unit system of transposon activity	416
la1	4.03	lazy plant1, prostrate growth habit	266
lbl1		leaf bladeless1, leaf blade reduced to absent; low temperature enhances expression	374
lc1		red leaf color1, dominant *Lc1* confers anthocyanin in coleoptile, nodes, auricle, leaf blade, etc.; (compare *Sn1*), encodes LC1	51, 143
lcs1		thylakoid membrane polypeptide1, electrophoretic mobility	386
lct1		thylakoid membrane polypeptide1, electrophoretic mobility	386
lct2		thylakoid membrane polypeptide2, presence-absence	386
les1	2.04+-0.01	lesion1, dominant *Les1* plants have large necrotic lesions resembling disease lesions formed by fungal infections on susceptible lines	241, 417, 422
les2	1.01+-0.01	lesion2, dominant *Les2* plants have small white lesions resembling disease lesions formed by fungal infections on resistant lines	241, 417
les3		lesion3, like *les1*; large, elliptical, necrotic lesions	15
les4	2.09+-0.02	lesion4, dominant *Les4* plants have late expression of large necrotic lesions on leaf blade and sheath	241, 421
les5	1.04+-0.03	lesion5, like *les2*	241, 421
les6		lesion6, like *les4*, but with many small to medium, irregular, mottled spots	241, 421
les7		lesion7, dominant *Les7* plants have late expression of small chlorotic lesions	241, 421
les8	9.03	lesion8, dominant *Les8* plants have late expression of small, pale green lesions	47, 48, 241
les9	7.02+-0.01	lesion9, dominant *Les9* plants have late expression of small necrotic lesions	241
les10	2.06	lesion10, like *Les1*; numerous small, round, necrotic lesions	242, 619
les11	2.08+-0.03	lesion11, like *les1*	412
les12		lesion12, dominant *Les12* plants have many small to medium, chlorotic to necrotic lesions on the leaf blade beginning at 5 leaf stage	412
les13	6.05+-0.05	lesion13, dominant *Les13* plants have frequent small to medium necrotic spots on leaf blade, sheath and culm, appearing at the 5 leaf stage	412
les14	3.07+-0.03	lesion14, dominant *Les14* plants have 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	412

SYMBOL	BIN	NAME, PHENOTYPE	REF
les15	2.05+-0.01	lesion15, dominant *Les15* plants are tiny and yellowish green, with many small chlorotic and necrotic lesions on speckled yellow green leaf blade background that looks like iron deficiency symptoms	412
les16		lesion16, dominant *Les16* plants are pale green and develop small chlorotic lesions on the leaf blade just before flowering	412
les17	3.07+-0.03	lesion17, dominant *Les17* plants have 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	412
les18	2.04+-0.01	lesion18, dominant *Les18*, leaf lesions; map location distinct from *les11*	420
les19	2.07+-0.02	lesion19, dominant *Les19*, leaf lesions; location near that of les10 but much different in expression	420
lfy1		leafy1, dominant *Lfy1* plants have increased number of leaves above ear	535
lg1	2.02+-0.01	liguleless1, ligule and auricle missing; leaves upright, enveloping	59, 155, 156
lg2	3.07+-0.01	liguleless2, like *lg1*, less extreme	59
lg3	3.04	liguleless3, dominant *Lg3* plants lack ligule; leaves upright, broad, often concave and pleated	452
lg4	8.06+-0.01	liguleless4, dominant *Lg4* plants lack ligule and auricle but show vestiges sporadically in blade	181
lhcb1	3.09	light harvesting chlorophyll a/b binding protein1, gene-specific cDNA probe; low expression in bundle sheath cells, encodes chlorophyll a/b binding protein candidate	538, 644
lhcb2	7.03+-0.02	light harvesting chlorophyll a/b binding protein2, gene specific cDNA probe; expressed in dark	538, 596, 644
lhcb3	8.05	light harvesting chlorophyll a/b binding protein3, probed by clone provided by L. Bogorad, encodes light-harvesting chlorophyll a/b binding protein	644
lhcb48		light-harvesting chlorophyll a/b48, unique genomic sequence; promoter is light regulated in tobacco and maize leaf mesophyll protoplasts, encodes light-harvesting chlorophyll a/b binding protein	297a
lhcbm7		cDNA sequence distinct from other light harvesting chlorophyll polypeptides; two copies by Southern blot analysis;, encodes light-harvesting chlorophyll a/b binding protein	349
li1		lineate leaves1, fine, white striations on basal half of mature leaves	113
lld1		lethal dwarf1, dominant *Lld1* is small plant with up to 3 short fleshy leaves that glisten in the sunlight. Found as single seedlings and distorted half-plant chimeras in M1 from mutagenesis; no progeny produced	429
lls1	1.04+-0.03	lethal leaf spot1, chlorotic-necrotic lesions resembling *Helminthosporium carbonum* infection	241, 611
ln1	6.05+-0.05	linoleic acid1, lower ratio of oleate to linoleate in kernel	125
lo2	9.03	lethal ovule2, ovules containing *lo2* gametophyte abort; embryo sac development stops at 2 to 4-nucleate stage	400
loc1		low oil content in kernel1, associated with albino seedlings	466
lop1		lo1 pl allergen homolog1, cDNA sequence homologous to allergen Lo1 pl, may encode? allergen Lo1 pl	66
lox1		lipoxygenase1, (was *csu160*) cDNA to leaf mRNA, strong homology to *Arabidopsis* lipoxygenase, encodes lipoxygenase	28
lp1	4.06+-0.06	lethal pollen1, *lp1* pollen fails in competition with *Lp1*	396
ltf1		lysr transcription factor homolog1, (was *uaz275*) cDNA to endosperm mRNA, homologous to lysr family of transcription regulators, may encode? lysr transcription factor	236
lty1		light yellow endosperm1, reduced color; heterozygote advantage; induced by mutagens in Oh43	141
lty2		light yellow endosperm2, reduced color; heterozygote advantage; induced by EMS in Oh43	141
lu1	5.05+-0.01	lutescent1, pale yellow green leaves with *lu2*	42, 544
lu2		lutescent2, yellow green leaves with *lu1*	544
lw1	1.11	lemon white1, white seedling, pale yellow endosperm	121, 608
lw2	5.09+-0.01	lemon white2, like *lw1*	121, 608
lw3	5.1+-0.01	lemon white3, like *lw1*; duplicate factor with *lw4*	608
lw4	4.05+-0.01	lemon white4, like *lw1*; duplicate factor with *lw3*	608
lxm1	3.06	lax midrib1, dominant *Lxm1* plants have leaves with wide, flat, flexible midrib	48, 406
map1		microtubule associated protein homolog1, (was *csu21*) cDNA to leaf mRNA, homologous to mouse microtubule associated protein, MAP2, may encode? microtubule calmodulin binding protein	28
MARZadh1		matrix associated region, near *adh1*, DNA region at 5' end of *adh1*, distal to the promoter region with high affinity for the nuclear matrix, prepared from nuclei of young maize seedlings	17
mc1		mucronate1, dominant *Mc1* kernels have opaque endosperm	512
mch1		maize CRY1 homolog1, ribosomal protein gene family (cDNA probe)	312
mch2		maize CRY1 homolog2, ribosomal protein gene family (cDNA probe)	312
mct1		modifier of *cox2* transcripts1, changes transcripts of mitochondrial gene	117
mde1		mouse DNA EBV homolog1, partially sequenced cDNA to endosperm mRNA, homology to mouse homolog to Epstein-Barr virus IR3 repeat	237
mdh1	8.04	malate dehydrogenase1, electrophoretic mobility; null allele is known; mitochondrial; dimeric; intra/interlocus hybrid bands occur, encodes malate dehydrogenase, mito.	431

SYMBOL	BIN	NAME, PHENOTYPE	REF
mdh2	6.1	malate dehydrogenase2, electrophoretic mobility; null allele is known; mitochondrial; dimeric; intra/interlocus hybrid bands occur, encodes malate dehydrogenase, mito.	279, 431
mdh3	3.08	malate dehydrogenase3, electrophoretic mobility; null allele is known; mitochondrial; dimeric; intra/interlocus hybrid bands occur, encodes malate dehydrogenase, mito.	431
mdh4	1.1	malate dehydrogenase4, electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur, encodes malate dehydrogenase, cytosol	90, 285, 431
mdh5	5.04	malate dehydrogenase5, electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur, encodes malate dehydrogenase, cytosol	431
mdh6		malate dehydrogenase6, putative chloroplast enzyme, cDNA sequence, may encode? malate dehydrogenase (NADP+), chloroplast	369
mdm1	6.01	maize dwarf mosaic virus resistance1, dominant *Mdm1* plants resistant	366
MDMV-cp		maize dwarf mosaic coat protein, confers resistance to strains MDMV-A and MDMV-B, encodes maize dwarf mosaic virus coat protein	394
mdr1	4.09+-0.03	maternal derepression of *R1*, *R1 R1 r1* aleurone mottled if *mdr1 mdr1 Mdr1*, solid color if *Mdr1 Mdr1* -	295
me1	3.08	NADP malic enzyme1, electrophoretic mobility; null allele is known; tetrameric, encodes malate dehydrogenase (oxaloacetate decarboxylating) (NADP+)	216
me3	3.02	NADP malic enzyme3, cDNA clone homologous to malic enzymes, putative plastid transit peptide, single copy, encodes malate dehydrogenase (oxaloacetate decarboxylating) (NADP+)	90, 285, 505
Med		Mediator of Irma excision, lacks suppressor or mutator function but is required with *En* for excision of reduced *En*-like Irma element	395
mei1		meiosis1, male sterile with dominant *Mei1*, chromatin hyper-condensed, chromosomes sticky in metaphase I and anaphase I	205, 211
mep1	5.101+-0.04	modifier of embryo protein1, affects quantities of *Glb1* protein forms	531
mfs14		male flower specific14, cDNA sequenced, associated with early microsporogenesis	646
mfs18		male flower specific18, cDNA sequence, associated with tassel glume vascular bundles	646
mg1		miniature germ1, germ 1/4 to 1/3 of normal; viable	308
mgs1		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	591
mgs2	4.09	male gametophyte-specific2, cDNA with pectin lyase homology; *bnl-mgs2* (cDNA probe Zmc58), may encode? pectin lyase	79
mi1	1.08+-0.08	midget plant1, small plant	166, 451
mmm1	1.1	modifier of mitochondrial malate dehydrogenases1, mobilities	431
mn1	2.08	miniature seed1, small, somewhat defective kernel, fully viable; invertase reduced	333
mn2	7.04+-0.04	miniature seed2, small kernel, loose pericarp; extremely defective but will germinate	309
mn3	6.01	miniature seed3, small kernel, etched/pitted endosperm; viable	586
mn4		miniature seed4, smaller, dented, viable kernels; double mutant with *cp3* has orange pericarp	426
mnb1		DNA-binding protein MNB1, cDNA sequence, Southern blots indicate a multigene family whose members have highly homologous N-terminal basic domain; sequence very distinct from *mnb2*, encodes DNA-binding protein MNB1a	646b
mnb2		DNA binding protein MNB2, cDNA sequence, Southern blots indicate small gene family, encodes DNA-binding protein MNB1b	646b
Mod		modifier: inactive *Spm* element, enhances excisions elicited by active *Spm*	363
monosomic		aneuploid individual with one or more entire chromosomes missing from an otherwise diploid complement	
Mp		modulator, modulator of pericarp: transposable factor affecting *P1* locus; parallel to *Ac-Ds*	63
Mpi1		Max Planck Institute1, transposable element, 10-15 copies in the genome	635
mpl1	1.11	miniplant1, dominant *Mpl1* plants are andromonoecious, intermediate dwarf (compare *D8*, possible allele); not responsive to gibberellins	228
Mr	9.03	mutator of *R1-m*: transposable factor	86, 438
Mrh	9.05+-0.01	Mutator of Rhoades, mutator: controlling element of *a1-mrh*	437, 488
mrp1		MRP homolog1, cDNA to endosperm mRNA, homologous to *E. coli* MRP	237
ms1	6.03+-0.01	male sterile1, anthers shriveled, not usually exserted; affected at microspore vacuolation	183, 551
ms2	9.04+-0.01	male sterile2, like *ms1*; affected between vacuolation and pore formation	173, 175
ms3	3.05+-0.05	male sterile3, anthers shrivelled; not usually exserted.	173, 175
ms5	5.06+-0.01	male sterile5, anthers not exserted; affected at microspore mitosis	34
ms7	7.02+-0.01	male sterile7, like *ms2*; tapetal cell dysfunction	34
ms8	8.07+-0.01	male sterile8, pollen mother cells degenerate	34
ms9	1.04	male sterile9, breakdown of pollen mother cells	34
ms10		male sterile10, like *ms5*; affected at microspore vacuolation	34
ms11		male sterile11, like *ms5*; affected at microspore mitosis	34
ms12	1.08+-0.08	male sterile12, like *ms1*; affected at microspore vacuolation	34
ms13	5.03+-0.03	male sterile13, like *ms5*; affected at microspore vacuolation	34
ms14	1.06+-0.01	male sterile14, like *ms5*; affected at microspore mitosis	34

SYMBOL	BIN	NAME, PHENOTYPE	REF
ms17	1.02+-0.01	male sterile17, like *ms1*; affected variably in meiosis	163
ms20		male sterile20, degeneration obvious by mid-vacuolated microspore stage	175
ms21	6.05+-0.05	male sterile21, pollen grains developing in presence of dominant *Ms21* are defective and nonfunctional if *sks1*, normal if Sks1	315, 526
ms22		male sterile22, pollen mother cells degenerate	581, 634
ms23	3.07+-0.03	male sterile23, pollen mother cells degenerate	581, 634
ms24		male sterile24, like *ms1*; affected in microspore mitosis	634
ms28	1.02+-0.02	male sterile28, anaphase I disturbed, spindle persists	206, 211
ms41	4.08+-0.04	male sterile41, dominant *Ms41* plants male sterile	428
ms42	5.03+-0.03	Ms42 plants male sterile; penetrance varies	4
ms43	8.07+-0.04	male sterile43, anaphase I impaired	205, 211
ms44	4.08	male sterile44, dominant *Ms44* plants male sterile	2, 3
msc1	1.12+-0.04	mosaic1, dominant *Msc1* aleurone mosaic for anthocyanin color	428
msc2	5.03+-0.03	mosaic2, dominant *Msc2* aleurone mosaic for anthocyanin color	428
msr1		macrophage scavenger receptor homolog1, cDNA to endosperm mRNA, homologous to human macrophage scavenger receptor type II	237
mst1		modifier of *R1-st1*, dominant *Mst1* reduces mutability of *R1*-st	14
mta1		mouse transplantation antigen homolog1, (was *uaz208*) cDNA to endosperm mRNA, very strong homology to Arabidopsis homolog of a mouse transplantation antigen, may encode? glycoprotein	236
mtl1		metallothionein homolog1, genomic clone, transcriptional and translation start sites mapped, Northern blots, similar to other class-I metallothioneins, may encode? metallothionein	124
Mu1		Mutator1, mutator: freely transposable element; *Mu1* designates element isolated from *Adh1-S3034*	474, 498
Mu4		mutator4: elements with terminal inverted repeats similar to *Mu1*	599
Mu5		Mutator5: element with inverted terminal repeats similar to *Mu1*	599
Mu8		Mutator8: 1.4 kbp element within *wx1-mum5*; terminal inverted repeats similar to *Mu1*	178
MuDR		regulator of Mutator activity, genetically, the unit responsible for transposition of Mutator elements (*MuR1*, *MuA2*, and *Mu9* are equivalent elements in this respect, subsumed into *MuDR* - Mutator Don Robertson)	238, 474
Mut	2.03+-0.01	mutator: controlling element for *bz1-m-rh*	488
mv1	3.05+-0.05	resistance to maize mosaic virus I ("corn stripe")1, dominant confers resistance to maize mosaic virus I ("corn stripe")	52
Mx		mobile element induced by X-rays, element found at *bz1-x3m* and elsewhere in the genome; *Bz1* restriction fragments correlated (insertion and reversion)	389
myg1		maternal yellow-green1, like *hcf2* except maternally inherited and yellow-green; induced in Mutator background	391
na1	3.07+-0.01	nana plant1, short, erect dwarf; no response to gibberellins	256, 319
na2	5.04+-0.01	nana plant2, like *na1*	451
nac1		NaCl stress protein1, (was *uaz250*) cDNA to endosperm mRNA, very strong homology to salt-stress peptide of wheat grass, may encode? salt stress protein	236
nbp1	7.02+-0.01	nucleic acid binding protein1, genomic and cDNA clones; product is imported in vitro into chloroplasts; expressed only in leaf, encodes nucleic acid-binding protein	116
NCS1		nonchromosomal stripe1, maternally inherited light green leaf striping	545
NCS2		nonchromosomal stripe2, maternally inherited pale green and depressed striping; mitochondrial	103
NCS3		nonchromosomal stripe3, maternally inherited striations, distorted plants; mitochondrial	103
NCS4		nonchromosomal stripe4, maternally inherited defective striations in cms-S (RD) revertant strain. Looks like NCS3	432
NCS5		nonchromosomal stripe5, maternally inherited stunted growth, yellow stripes, aborted kernels; mitochondrial cytochrome oxidase subunit2 (*cox2*) alteration	432a
NCS6		nonchromosomal stripe6, maternally inherited stunted growth, yellow stripes, aborted kernels; mitochondrial cytochrome oxidase subunit2 (*cox2*) alteration	312a
nec1	8.03+-0.02	necrotic1, chlorotic seedling that stays rolled, wilts and dies	347
nec2	1.04+-0.01	necrotic2, green seedling develops necrotic lesions at 2-3 leaf stage; lethal	8
nec3	5.06+-0.01	necrotic3, seedling emerges with tightly rolled leaves that turn brown and die without unrolling; manually unrolled leaves tan with dark brown crossbands	404
nec4	2.03+-0.01	necrotic4, seedling yellow, leaf tips necrotic; lethal	243
nec5	4.09+-0.03	necrotic5, pale green seedling becoming necrotic; dark brown exudate; lethal	416
nec6	5.06+-0.01	necrotic6, like *nec3*	416
nec7	5.1+-0.04	necrotic7, lighter green seedling becoming necrotic in crossbands	416
nl1		narrow leaf1, leaf blade narrow, some white streaks	166
nl2	5.04+-0.01	narrow leaf2, dominant, leaves narrow and distorted	409, 422
nld1		narrow leaf dwarf1, small compact plant with narrow rolled leaves that are bleached pale green, especially along the midrib.	429

SYMBOL	BIN	NAME, PHENOTYPE	REF
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)	80, 218
nnr2		nitrate reductase2, partial cDNA from seedling roots homologous to nitrate reductase, encodes NAD(P)H:nitrate reductase	329
nnr3		nitrate reductase(NADH)3, scutellum cDNA, cyt b domain functional in *E. coli*, encodes nitrate reductase (NADH)	80
NOR	6.01	nucleolar organizer, nucleolus organizer: encodes ribosomal RNA's, encodes rRNA18S	357
ns1		narrow sheath1, plant brachytic; leaf sheath and blade taper, blade widens toward tip; husks narrowed, ears exposed	153
nsf1		nicosulfuron susceptible1, recessive susceptibility to nicosulfuron (Accent) herbicide	282
ntm9		neurotoxin M9 homolog, (was *csu129*) cDNA to leaf mRNA, homologous to scorpion neurotoxin M9, may encode? sodium channel inhibitor	29
o1	4.06+-0.01	opaque endosperm1, endosperm starch soft, opaque	166, 397, 552
o2	7.01	opaque endosperm2, like *o1*; high lysine content; regulates b-32 protein (see pro1); reduced lysine degradation (lysine-ketoglutaric reductase), encodes *o2* protein	166, 397, 552
o5	7.02	opaque endosperm5, like *o1*; virescent to yellow or white seedlings	493
o7		opaque endosperm7, like *o1*; high lysine content	335, 383
o8	2.05	opaque8, endosperm opaque, higher in lysine	220
o9		opaque endosperm9, crown opaque and light in color, frequently with a cavity; base or abgerminal side of kernel often corneous	399
o10	1.12+-0.04	opaque endosperm10, like *o1*	399
o11		opaque endosperm11, thin, opaque, somewhat shrunken kernels with greyish cast	399
o13		opaque endosperm13, opaque, etched kernels with rim of corneous starch on abgerminal side	399
o14	6.07+-0.04	opaque kernel14, large opaque kernel with mostly floury starch except for a small amount of corneous starch near the base of the abgerminal side; normal green seedling develops yellow striped appearance and is slow in growth	413
obf1	1.06+-0.01	octopine synthase binding factor1, encodes protein with bZIP motif that binds to transcriptional enhancer sequences (ocs-elements); gene specific probe; map location unclear, encodes octopine synthase binding factor	547
obf2	9.04+-0.01	octopine synthase binding factor2, encodes protein with bZIP motif that binds to transcriptional enhancer sequences (ocs-elements), may encode? octopine synthase binding factor	547
oec33	6.02	oxygen evolving complex, 33kDa subunit, (was umc172) cDNA identified by hybrid-selection, in vitro translation and immunoprecipitation with antisera against spinach OEC33, encodes oxygen evolving complex, 33 kDa subunit	539
og1		old gold stripe1, dominant *Og1* plants have variable bright yellow stripes on leaf blade	326
ole1		oleosin1, major protein from lipid bodies, cDNA clone, encodes oleosin 16kDa	613
ole2		oleosin2, cDNA and genomic sequence, encodes oleosin, 18 kDa	476
omt1		O-methyltransferase1, genomic and cDNA clones; sequence; single site (Southern analysis); transgenic expression in *E. coli*, encodes caffeate O-methyltransferase	109
ora2		orange endosperm2, color modified; plant vigor reduced in recessives, heterozygote advantage; mutagen-induced in Oh43	140
ora3		orange endosperm3, color modified; heterozygote advantage; induced by EMS in Oh43	141
oro1	6.02+-0.02	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	346
oro2		orobanche2, like *oro1*	346
orom1		orobanche modifier1, dominant *Orom1* partially corrects chlorophyll loss in *oro1*	346
orp1	4.04	orange pericarp1, duplicate factor with *orp2*; pericarp orange over *orp1 orp2* kernels; lethal, tryptophan auxotroph, encodes tryptophan synthase B subunit	418, 642
orp2		orange pericarp2, duplicate factor with *orp2*, encodes tryptophan synthase B subunit	418
os1	2.03+-0.02	opaque-endosperm, small germ1, opaque crown; kernel larger, lighter color; viable; reduced oil content	560, 561
oy1		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	174
P		plant color component at *R1*, anthocyanin pigmentation in seedling leaf tip, coleoptile, anthers, encodes *P* (of *r1*) encoded protein	142, 578, 579
p1	1.04	pericarp color1, dominant *P1* confers red pigment in cob and pericarp; tissue-specific allele variations	7, 154, 328
pal1		phenylalanine ammonia lyase candidate1, (was *csu156*) cDNA to leaf mRNA homologous to rice phenylalanine ammonia lyase, may encode? phenylalanine ammonia lyase	285
pam1		plural abnormalities of meiosis1, desynchronized meiotic divisions and premeiotic mitosis; male sterile, incompletely female sterile	205, 210
pam2		plural abnormalities of meiosis2, like *pam1*	205, 207
pb4	6.01	piebald leaves4, like *pb1*	130

SYMBOL	BIN	NAME, PHENOTYPE	REF
pbp1		promoter binding protein1, cDNA clones, Southwestern analysis supports binding to *pep1* promoter, light induced, encodes *pep1*-promoter binding protein	283
pck1		phosphoenolpyruvate carboxykinase homolog1, (was *csu145*) cDNA to leaf mRNA, low copy, homologous to yeast phosphoenolpyruvate carboxykinase, may encode? phosphoenolpyruvate carboxykinase	28, 29
pd1	3.07+-0.03	paired rows1, single vs. paired pistillate spikelets; quantitative, one of a family of loci differentiating maize vs. teosinte	310
pdc1	8.05	pyruvate decarboxylase1, cDNA and genomic clone, encodes pyruvate decarboxylase	286, 456
pdc2	8.03	pyruvate decarboxylase2, cDNA available; gene-specific probe, encodes pyruvate decarboxylase	456
pdc3	1.03	pyruvate decarboxylase3, cDNA sequence, gene-specific probe, encodes pyruvate decarboxylase	456
pdf1		thylakoid membrane polypeptide1, dominant Pdf1 confers increase in electrophoretic mobility	385
pdk2	8.05+-1	pyruvate, orthophosphate dikinase2, cDNA, genomic and peptide sequences; gene-specific cDNA, p-pPPDK4; cytosolic or plastidic, dependent on transcript processing, encodes pyruvate, orthophosphate dikinase	202
pe1		perennialism1, vegetatively totipotent in combinations with *gt1* and *id1*	536, 537
pep1	9.04	phosphoenolpyruvate carboxylase1, cytosolic C4 isozyme; single copy, similar to C3-PEPCase genes; cDNA complements *E. coli* mutant; genomic and partial amino acid sequences compare; map location in conflict, encodes phosphoenolpyruvate carboxylase	252a, 257a
pep4	7.02+-0.01	phosphoenolpyruvate carboxylase4, cDNA for anaplerotic C3 isozyme; gene specific cDNA probe;, encodes phosphoenolpyruvate carboxylase	284
pet1	8.07+-0.04	photosynthetic electron transport1, high leaf chlorophyll fluorescence, pale green, lacks cytochrome bf, reduced PSII at higher intensity light	25
pet2		photosynthetic electron transport2, lacks cytochrome bf polypeptides	25
pet3		photosynthetic electron transport3, lacks cytochrome bf polypeptides; likely allelic to *hcf6*	25
pet4		photosynthetic electron transport4, lacks cytochrome bf polypeptides	25
pet5		photosynthetic electron transport5, lacks cytochrome bf polypeptides	25
pg12	9.04	pale green12, duplicate factor with *pg11*	484
pg13		pale green13, seedling light yellowish green; stunted growth	41, 543
pg15	1.04+-0.03	pale green15, seedling light yellowish green; bleaches to near white in patches; lethal	416
pg16	1.12+-0.04	pale green16, seedling light yellowish green	416
pgd1	6.01	6-phosphogluconate dehydrogenase1, electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur, encodes 6-phosphogluconate dehydrogenase	216
pgd2	3.05	6-phosphogluconate dehydrogenase2, electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur, encodes 6-phosphogluconate dehydrogenase	216
pgl1		exopolygalacturonase1, cDNA clone nearly identical but distinct from other pgl reading frames of inbred Mo17, sequence match to N-terminal sequence of maize exopolygalacturonase, encodes exopolygalacturonase	22, 434a
pgl2		polygalacturonase2, cDNA and genomic clones, sequence nearly identical to pgl1; one of 10-12 member gene family, encodes exopolygalacturonase	22
pgl3		polygalacturonase3, cDNA and genomic clones; one of 10-12 member gene family, encodes exopolygalacturonase	22
pgl4		exopolygalacturonase4, cDNA sequence; distinct from others isolated from same inbred; one of 10-12 member gene family, encodes exopolygalacturonase	434a
pgl6		exopolygalacturonase6, genomic sequence homologous to pollen-specific cDNA, significant homology to tomato-ripening enzyme; one of 10-12 member gene family; promoter active in transgenic tobacco, encodes exopolygalacturonase	6a
pgl7		exopolygalacturonase7, genomic clone, open reading framese nearly identical to *pgl1* but distinct 3' non-coding sequence, encodes exopolygalacturonase	22
pgl8		exopolygalacturonase8, genomic clone, open reading frame nearly identical to *pgl1* but distinct 3' non-coding sequence; mRNA product confirmed by PCR, encodes exopolygalacturonase	22
pgm1	1.12	phosphoglucomutase1, electrophoretic mobility; null allele is known; cytosolic; monomeric, encodes phosphoglucomutase (glucose-cofactor)	216
pgm2	5.03	phosphoglucomutase2, electrophoretic mobility; null allele is known; cytosolic; monomeric, encodes phosphoglucomutase (glucose-cofactor)	216
pgp1		P-glycoprotein homolog1, (was *csu138*) cDNA to leaf mRNA, homologous to *Arabidopsis* P-glycoprotein, may encode? P-glycoprotein	28
ph1	4	pith abscission1, cob disarticulation; quantitative, one of a family of loci differentiating maize vs. teosinte	191
phi1	1.12	phosphohexose isomerase1, electrophoretic mobility; null allele is known; cytosolic; dimeric; intralocus hybrid bands occur, encodes glucose-6-phosphate isomerase	216
pho1		phosphate regulatory homolog1, partially sequenced cDNA to endosperm mRNA, homology to yeast pho80 gene, may encode? transcription factor	237

SYMBOL	BIN	NAME, PHENOTYPE	REF
php1		chloroplast phosphoprotein1, isozyme; phosphorylated thylakoid protein. Chloroplast phosphoprotein polymorphic in COxTx, encodes chloroplast phosphoprotein	79
phy1		phytochrome1, sequence, encodes phytochrome A	96
pi1		pistillate florets1, duplicate factor with *pi2*; secondary florets develop ("Country Gentlemen" or "Shoe Peg" expression) in *pi1 pi2* ears; quantitative character	253
pi2		pistillate florets2, duplicate factor with *pi1*	253
pl1	6.04+-0.01	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 transcriptional activator (myb) protein	160, 161
plt1		phospholipid transfer protein homolog1, amino acid sequence, deduced from cDNA, homologous to phospholipid transfer proteins, may encode? phospholipid transfer protein	603
pm1	3.05	pale midrib1, midrib and adjacent tissue lighter green; reduced plant vigor	60
pmg1		1, 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	221
pn1	7.06+-0.01	papyrescent glumes1, dominant *Pn1* plants have long, thin papery glumes in ear and, less obviously, in tassel	193
po1	6.01	polymitotic1, (was *ms4*, *ms6*) repeats 2nd meiotic division in male and female	31, 32
pop1		putative organelle permease1, (*uaz282*) cDNA to endosperm mRNA, homologous to yeast putative mitochondrial carrier protein, may encode? organellar permease	236
pox1		fowlpox viral protein homolog1, cDNA to endosperm mRNA; homologous to fowlpox virus major core protein P4B	237
ppg1	5.1+-0.04	pale pale green1, light pale green seedling with white crossbands that become necrotic, spreading to the rest of the leaf causing lethality	416
ppi1		peptidyl-prolyl isomerase1, cDNA homologous to tomato peptidyl-prolyl cis-trans isomerase, may encode? peptidyl-prolyl cis-trans isomerase	195
pr1	5.08+-0.01	red aleurone1, changes purple aleurone to red; encodes flavonoid 3'-hydroxylase	149
pre1		premature senescence1, senescence begins at least 2 weeks prior to anthesis, spreading from bottom to top of plant; occasionally sheds viable pollen	57
prf1		profilin homolog1, deduced amino acid sequence from cDNA shares 76-85% identity with two other plant profilins; 3-6 member multigene family; gene specific probe, may encode? profilin	580a
prf2		profilin homolog2, amino acid sequence, deduced from cDNA, shares 76-85% identity with two other plant profilins; 3-6 member gene family; gene specific probe, may encode? profilin	580a
prf3		Profilin homolog3, amino acid sequence deduced from cDNA shares 76-85% identity with two other plant profilins, 3-6 member gene family; gene specific probe, may encode? profilin	580a
prg1	5.1+-0.04	pitted rough germless1, small pitted rough endosperm, usually germless; seed with larger embryo will produce small striated seedlings	520
prh1	4.06	ser/thr protein phosphatase1, PCR clone from root mRNA; expressed in E. coli as active kinase; 4-8 copies by Southern analyses; gene specific probe, encodes serine/threonine specific protein phosphatase	557
prh2		protein phosphatase homolog2, (was *uaz244*) cDNA to endosperm mRNA, strong homology to a yeast open reading frame, homologous to human 61K transforming protein phosphatase, PP2A, may encode? protein phosphatase	236
pro1	8.05+-0.01	proline responding1, (allele *o6*) crumpled opaque kernel; green-striped lethal seedling; responds to proline in culture	196
prp1		pathogenesis-related protein1, cDNA clone, single copy, deduced protein product is basic (vs. acidic), normally accumulates during germination, not induced by mercuric chloride, encodes PRP1	84
prp2	2.05	pathogenesis-related protein homolog2, (was *csu133*) cDNA to leaf mRNA, homologous to kidney bean pathogenesis-related protein, PIR S14730, gene specific probe, may encode? pathogenesis-related protein	29
prr1		putidaredoxin reductase homolog1, (was *uaz204*) cDNA to endosperm mRNA, strong homology to NADH-putidaredoxin reductase, may encode? putidaredoxin reductase	237
ps1	5.06	pink scutellum1, (was *vp7*, *lyc1*) some alleles viviparous; endosperm and scutellum pink, seedling white with pink flush	492, 564
psa1		photosystemI1, lacks photosystem I core complex polypeptides	25
psa2		photosystemI2, lacks photosystem I core complex polypeptides	25
psa3		photosystemI3, lacks photosystem I core complex polypeptides	25
psa4		photosystemI4, lacks photosystem I core complex polypeptides	25
psa5		photosystemI5, (was csu18) cDNA to leaf mRNA, strong homology to barley sequence photosystem I subunit N, encodes PSI, subunit N	28
psa6		photosystem I reaction center6, (was *csu67*) cDNA to leaf mRNA, strong homology to barley chloroplast psaK sequence, encodes photosystem I, subunit K	28
psb1	8.07+-0.04	photosystem II1, lacks PSII core complex; pale seedling; mutable (Mu-induced)	25, 617
psb2		photosystemII2, lacks photosystem II core complex polypeptides	25

SYMBOL	BIN	NAME, PHENOTYPE	REF
psei1		cystatin1, cDNA, isolated protein inhibits papain, developing endosperm, encodes cysteine proteinase inhibitor	1
pt1	6.06	polytypic ear1, dominant *Pt1* plants have proliferation of irregular growth on ear and tassel	402
ptc1		proteasome C9 homolog1, (was *uaz237*) cDNA to endosperm mRNA, very strong homology to proteasome subunit C9 of several species, may encode? proteasome (endopeptidase) component C9	236
ptd1	1.1	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	521, 522
ptd2	7.05+-0.02	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	521, 522
ptk1		protein kinase homolog1, cDNA, genomic clones homologous to serine/threonine protein kinases (one domain) and to *Brassica* self-incompatibility locus glycoprotein (a second domain), may encode? receptor-like serine/threonine protein kinase	620
px1	2.08+-0.03	peroxidase1, (was *prx1*) electrophoretic mobility; null allele is known; monomeric, encodes peroxidase	55, 226, 227
px2		peroxidase2, electrophoretic mobility; monomeric, pollen specific, encodes peroxidase	55, 336
px3	7.06+-0.01	peroxidase3, electrophoretic mobility; monomeric, encodes peroxidase	54, 336
px4		peroxidase4, electrophoretic mobility; null allele is known; monomeric, encodes peroxidase	55, 336
px5		peroxidase5, presence-absence, encodes peroxidase	55, 336
px6		peroxidase6, presence-absence, encodes peroxidase	55, 336
px7		peroxidase7, electrophoretic mobility; null allele is known; monomeric, encodes peroxidase	54, 336
px8		peroxidase8, electrophoretic mobility; monomeric, encodes peroxidase	54, 55
px9		peroxidase9, electrophoretic mobility; null allele is known; monomeric, encodes peroxidase	54, 55
py1	6.06	pigmy plant1, leaves short, pointed; fine white streaks	597
py2	1.15+-0.01	pigmy plant2, like *py1*	416
pyd1	9	pale yellow seedling; deficiency for short terminal segment of chromosome arm, lethal; complements *yg2* but not *wd1*	358
r1		colored1, regulates anthocyanin pathway; dominant *R1* (*S* element) confers function in aleurone; dominants represented by *R1-r* or *r1-r* (*P* element) confer function in anthers, leaf tip, brace roots, etc., encodes *R1* (myc) protein	149
ra1	7.02	ramosa1, ear and tassel many-branched; tassel branches taper to tip	34, 200
ra2	3.03	ramosa2, tassel many-branched, upright, not conical like *ra1*; irregular kernel placement	61, 166, 433
ra3	4.06+-0.06	ramosa3, branched inflorescence	453
rab17	6.06	responsive to abscisic acid17, cDNA sequence agrees with amino acid sequence, encodes glycine-rich protein(RAB17)	102
rab28		abscisic acid-responsive28, cDNA and genomic clones, inducible by ABA in embryos and young leaves; induced by water-stress in leaves; homologous to cotton LeaD-34	465
rab30	1.01	responsive to abscisic acid30, cDNA elicited by ABA, encodes abscisic acid responsive protein30	465
rap1		retinoblastoma-associated protein homolog1, cDNA to endosperm mRNA, homologous to human retinoblastoma-associated protein, RB1, may encode? cell cycle protein	237
rBg		receptor of *Bg*, responds to *Bg*	511
rcm1	7.01+-0.01	rectifier1, dominant *Rcm1* restores miniature seed of teosinte cytoplasm to normal	6, 117
rcm2		rectifier2, dominant *Rcm2* weakly restores miniature seed of teosinte cytoplasm to normal	6, 117
rcm3		rectifier3, dominant *Rcm3* restores miniature seed of teosinte cytoplasm to normal; from *Z diploperennis*	6
rcu		receptor of *Fcu*, responds to *Fcu*	213
rcy:Mu7		receptor of *Cy*, first described for *bz1* allele, *bz1-rcy*	525
rd1	1.12	reduced plant1, semi-dwarf plant; possible allelism with *bv2*	401
rd2	6.07+-0.04	reduced plant2, like *rd1*, but not as extreme	204
rd3	3.05+-0.01	reduced plant3, like *rd1*; anthocyanin interactions	351
rDNA18S	6.01	NOR (nucleolus organizer) component, encodes rRNA18S	278
rDNA25S	6.01	NOR (nucleolar organizer) component, encodes rRNA25S	278
rDNA5.8S	6.01	NOR (nucleolus organizer) component, encodes rRNA5.8S	278
rDNA5S	2.09	cluster consisting of several thousand repeated genes, encodes rRNA5S	582, 647
rDt		receptor of Dotted, transposable element excised by action of *Dt*	562
ref1	3.02+-0.02	reduced floury endosperm1, small, reduced endosperm with dull, floury appearance; low frequency of expression; approx. 20% will germinate	520
ren1	5.09	reduced endosperm1, small seed with reduced, opaque endosperm, usually lethal; seed with larger embryos produce fertile plants	521, 522
ren2	7.05	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	258, 521, 522

SYMBOL	BIN	NAME, PHENOTYPE	REF
ren3		reduced endosperm3, reduced endosperm, partially filled to empty pericarp; small germ or germless; larger seed produce fertile plants	520, 522
rf1	3.05	restorer of fertility1, dominant *Rf1* restores fertility to cms-T; complementary to *Rf2*	146, 272
rf2	9.03+-0.01	restorer of fertility2, see *rf1*	146, 148
rf3	2.11+-0.01	restorer of fertility3, dominant *Rf3* restores fertility to cms-S	70, 148
rf4	8	restorer of fertility4, dominant *Rf4* restores fertility to cms-C; complementary with *Rf5* and *Rf6*	219, 475
rf5		restorer of fertility5, dominant *Rf5* restores fertility to cms-C; complementary with *Rf4* and *Rf6*	277, 614
rf6		restorer of fertility6, dominant *Rf6* restores fertility to cms-C; complementary with *Rf4* and *Rf5*	277, 614
rf7		restorer of fertility7, dominant *Rf7* partially restores fertility to cms-Y	475
rfz1		rat frizzled homolog1, cDNA to endosperm mRNA, homologous to rat homolog of Drosophila polarity gene frizzled	237
rg1	3.05	ragged leaves1, dominant *Rg1* plants develop defective tissue between veins of older leaves, causing holes and tearing	64
rgd1	6.01+-0.01	ragged seedling1, seedling leaves narrow, thread-like, have difficulty in emerging	299
rgh1	8.1+-0.01	rough kernel1, small floury kernel with rough and pitted surface and nonviable embryos	415
rgo1		reversed germ orientation1, embryo faces base of ear; variable frequency, maternal trait	508
rhm1	6	resistance to *Helminthosporium maydis*1, chlorotic-lesion reaction with *Cochliobolus heterostrophus* (=*H. maydis*) race O	553
ri1	4.01+-0.01	rind abscission1, dominant *Ri1* plants have cob disarticulation; quantitative, one of a family of loci differentiating maize vs. teosinte	191
ring 9S		ring carrying *Wd1*, *Yg2*, and *C1-I*; frequent losses recognizable in endosperm in presence of *C1*, in plants if *wd1* or *yg2*	
Ring chromosome		chromosome with a centromere and no ends--i.e., arm segments are attached to form a closed ring	
rip1	8.05	ribosome-inactivating protein1, electrophoretic mobility, abundant 32kD endosperm protein (b32 protein), cytosolic, inactivates rabbit (not maize or wheat) ribosomes, encodes ribosome inactivating protein	196, 621
ris1	5.06	iron-sulfur protein1, one of two very similar cDNAs recovered by antiserum screen from B73 seedling leaf RNA, both transcribed in leaf tissue, encodes Rieske iron-sulfur protein, chloroplastic	24
ris2	4.09	iron-sulfur protein2, one of two very similar cDNAs for chloroplastic iron-sulfur protein, encodes Rieske iron-sulfur protein, chloroplastic	24
rlc1		rindless culm1, upper internodes lack rind in longitudinal bands	94
rld1	9.08	rolled leaf1, in dominant *Rld1* plants, leaves are tightly rolled and tend to be entangled; ligular flaps on abaxial surface of leaf; resembles *Ce1*	47, 48
rli1		rough lineate1, lineate-like streaks of protruding tissue on leaf blade which produce a rough texture	429
rMrh		receptor of *Mrh*, responds to *Mrh*	488
rMut		receptor of *Mut*, responds to *Mut*	488
rMx		responder to *Mx*, non-autonomous element responding to *Mx*	389
rnp1		chloroplast RNA binding protein homolog1, (was *csu17*) cDNA to leaf mRNA, strong homology to tobacco nuclear encoded chloroplast RNA binding proteins, encodes chloroplast RNA binding protein	29
rp1		resistance to *Puccinia sorghi*1, dominant *Rp1* resistant	338, 339
rp3	3.05	resistance to *Puccinia sorghi*3, dominant *Rp3* resistant	246, 636
rp4	4.01+-0.01	resistance to *Puccinia sorghi*4, dominant *Rp4* resistant	224, 636
rp5		resistance to *Puccinia sorghi*5, dominant *Rp5* resistant	224, 515
rp6		resistance to *Puccinia sorghi*6, dominant *Rp6* resistant	224, 636
rpl10		ribosomal protein L10 homolog, (was *uaz198*) partially sequenced cDNA to endosperm mRNA, homologous to yeast acidic ribosomal protein, rpl10E, may encode? ribosomal protein L10e	236
rpl19		ribosomal protein L19 homolog, (was *csu36*) cDNA to leaf mRNA, homologous to rat ribosomal protein L19, low copy number, may encode? ribosomal protein L19	285, 489
rpl5		ribosomal protein L5 homolog, (was *uaz189*) cDNA to endosperm mRNA, very strong homology to cytoplasmic ribosomal proteins L5, encodes ribosomal protein L5	236
rpo1		RNA polymerase II homolog1, (was *csu150*) cDNA to leaf mRNA, highly homologous to yeast RBP2, RNA polymerase, may encode? RNA polymerase	285
rpp9		resistance to *Puccinia polysora* and *Puccinia sorghi*9, dominant *Rpp9* resistant	609
rps8		ribosomal protein S8 homolog, (was *csu34*) multiple copies, cDNA to leaf mRNA, sequence homologous to rat ribosomal protein S8, GenBank X06423, may encode? ribosomal protein S8	285
rps11		ribosomal protein S11, cDNA sequenced; homology to rps11; two bands hybridize in Southerns; encodes ribosomal protein S11	313b
rps22		ribosomal protein S22 homolog, (was *csu28*) multiple copies; cDNA to leaf mRNA, homologous to *Xenopus* ribosomal protein S22, may encode? ribosomal protein S22	285
rs1	7	rough sheath1, dominant *Rs1* plants have extreme ligule disorganization.	297
rs2	1.06+-0.01	rough sheath2, short, zigzag plants with warty, distorted sheaths and leaves	297

SYMBOL	BIN	NAME, PHENOTYPE	REF
rs4	7.01+-0.01	rough sheath4, dominant *Rs4* plants have rough leaf sheaths; vascular bundles enlarged	409
rt1	3.04+-0.01	rootless1, secondary roots few or absent	262
rth1	1.12+-0.04	roothair defective1, roothairs do not elongate fully in *rth1* homozygotes.	429, 626
rth2	5.1+-0.04	roothair defective2, like *rth1*	429, 626
rth3	1.04+-0.03	roothair defective3, like *rth1*, but "stocking cap" roothair initials under electron microscope	429, 626
ruq		receptor of *Uq*, element mediated by *Uq*	186
S		seed color component at *R1*, anthocyanin pigmentation in aleurone; (see also cms-S), encodes *S* (of *r1*) encoded protein	142, 578
sad1		shikimate dehydrogenase1, electrophoretic mobility; plastidial; monomeric, encodes shikimate dehydrogenase	627
sar1		SAR homolog1, (was *uaz151*) cDNA to leaf mRNA, strong homology to *Arabidopsis* sar1 homolog, encodes GTP-binding protein, SAR1 homolog	236
sbd1	6.07+-0.04	sunburned1, sun-exposed leaves greyish-waxy	194, 410
sbe2		starch branching enzyme2, amino acid sequence, deduced from cDNA sequence, has 71% homology to pea sbeI and 52% to maize sbeI, encodes starch branching enzyme II	177
sci1		subtilisin-chymotrypsin inhibitor homolog1, cDNA to mRNA from germinating embryo infected with *Fusarium moniliforme*, may encode? subtilisin-chymotrypsin inhibitor	118
sdh1		sorbitol dehydrogenase homolog1, (was *uaz152*) cDNA to endosperm mRNA, strong homology to sorbitol dehydrogenases, encodes sorbitol dehydrogenase	236
sdw1	8.05+-0.01	semi-dwarf plant1, dominant *Sdw1* plants have shortened internodes, erect leaves	47
sdw2	3.06+-0.01	semi-dwarf2, short plant, 1/3-1/2 normal height, with normal green erect leaves; does not respond to gibberellins; no anthers in ear	412
se1	4.1+-0.01	sugary-enhancer1, high sugar content with *su1*; light yellow endosperm; freely wrinkled in III677a	176
sen1	3.05+-0.05	soft endosperm1, duplicate factor with sen2; endosperm soft, opaque	584
sen2	7.04+-0.04	soft endosperm2, duplicate factor with *sen1*	584
sen3	1.08+-0.08	soft endosperm3, duplicate factor with *sen4*; like *sen1*	584
sen4		soft endosperm4, duplicate factor with *sen3*	584
sen5	2.06+-0.05	soft endosperm5, duplicate factor with *sen6*; like *sen1*	584
sen6	5.07+-0.07	soft endosperm6, duplicate factor with *sen5*	584
sft1		small flint type1, ears on sft1 plants produce only small flint endosperms; +/*sft* ears are normal	562
sg1		string cob1, dominant Sg1 plants have reduced pedicels	189
sh1	9.02	shrunken1, inflated endosperm collapses on drying, forming smoothly indented kernels; sucrose synthase-1 of endosperm (compare *css1*); homotetramer, encodes sucrose synthase	255
sh2	3.09	shrunken2, inflated, transparent, sweet kernels collapse on drying, becoming angular and brittle; endosperm ADPG pyrophosphorylase subunit (compare *bt2*), encodes ADP glucose pyrophosphorylase	340
sh4	5.09+-0.01	shrunken4, collapsed, chalky endosperm	607
sh5	5.05+-0.01	shrunken5, sides of kernel collapsed	351, 571
sh6	7.01+-0.01	shrunken pale green6, shrunken, opaque, normal size kernel; pale green virescent seedling that greens slowly and is usually lethal. Like *sh1* in kernel phenotype.	413, 588
si1	6.02+-0.01	silky1, (was *ts8*, *ms-si*) multiple silks in ear; sterile tassel with silks	183
sip1		stress-induced protein1, cDNA sequence homologous to thaumatin-like protein, encodes thaumatin-like protein	185
sk1	2.04+-0.01	silkless ears1, pistils abort, no silks	271
sks1	2.06+-0.01	suppressor of KYS sterility1, pollen grains developing in presence of dominant *Ms21* are defective and nonfunctional if *sks1*, normal if Sks1	315, 526
sl1	7.04	slashed leaves1, leaves slit longitudinally by necrotic streaks. Plants are weaker than normal, but produce pollen and ears.	233
sm1	6.06	salmon silks1, silks salmon color with *P1-RR*, brown in *P1-WW*	7
sn1		scutellar node color1, anthocyanin in coleoptile, nodes, auricle, leaf blade, etc. (compare *Lc1*)	197, 198
sod1		superoxide dismutase1, electrophoretic mobility; plastidial; Cu-Zn dimeric; intralocus hybrid bands occur, encodes superoxide dismutase, chloroplastidic	27
sod2	7.05	superoxide dismutase2, electrophoretic mobility; cytosolic; Cu-Zn dimeric, encodes superoxide dismutase, cytosolic	27, 82, 83
sod3		superoxide dismutase3, electrophoretic mobility; mitochondrial; Mn tetrameric; intralocus hybrid bands occur; cDNA complements yeast mutant;, encodes superoxide dismutase, mitochondrial	27
sod4	1.04	superoxide dismutase4, electrophoretic mobility; cytosolic; Cu-Zn dimeric; intralocus hybrid bands occur; two similar sequences X17564 (sod4), X17565(sod4A), encodes superoxide dismutase, cytosolic	27
sos1	4.02+-0.01	Suppressor of sessile spikelets1, dominant *Sos1* from teosinte suppresses sessile spikelets in ear primordia and in tassel, unlike teosinte ears, where the pedicellate, not the sessile spikelet is lacking	138

SYMBOL	BIN	NAME, PHENOTYPE	REF
spc1	3.06+-0.01	speckled1, dominant *Spc1* plants display brown speckling on leaves and sheath at flowering; supporting tissues weak	422
spc2	1.12+-0.04	speckled2, green seedling with light green speckles	416
spc3	3.07+-0.03	speckled3, green seedling with dark and light green speckles	416
Spm		Suppressor-Mutator, suppressor-mutator: autonomous transposable element (equivalent to *En*); regulates *dSpm* (=I) transposition and function at *a1-m1*, *a1-m2*, *bz1-m13*, etc., encodes TnpA	361, 362
sps1	8.08	sucrose phosphate synthase1, cDNA encodes a 1068 amino acid leaf protein; transgenic (*E. coli*) directs sucrose phosphate synthesis, encodes sucrose-phosphate synthase	641
spt1	2.08+-0.03	spotted1, pale green, weak seedlings with dark green spots	416
spt2	4.03+-0.03	spotted2, like *spt1*	416
sr1	1.02	striate leaves1, many white striations or stripes on leaves	68, 166
sr2		striate leaves2, white stripes on blade and sheath of upper leaves	267
sr3		striate leaves3, virescent seedling and striate to striped plant	203
sr4	6.07+-0.04	striate leaves4, seedlings pale luteus, later leaves white-striped	408
srp1		signal recognition particle RNA, gene family, encodes 7SL RNA	81
ssu1	4.08	ribulose bisphosphate carboxylase small subunit1, probed by clone for ribulose bisphosphate carboxylase small subunit provided by W. C. Taylor, encodes ribulose bisphosphate carboxylase	66a
ssu2	2.06	ribulose bisphosphate carboxylase small subunit2, probed locus, encodes ribulose bisphosphate carboxylase	66a
st1	4.03+-0.01	sticky chromosome1, small plant, striate leaves, pitted kernels resulting from sticky chromosomes; *st1-e* heightened by high temperature	35
stAc		stabilized Activator, RFLP locus; name from P. Chomet (unpublished)	78
stm1		stolon tip maize homolog1, (was *csu6*) cDNA to leaf mRNA, strong homology to potato stolon tip protein sequence, less homologous to yeast guanylyltransferase, may encode? guanylyltransferase	29
stp1	8.05	sugar transport homolog1, (was *csu142*) cDNA to leaf mRNA, strong homology to yeast plasma membrane sugar transport protein, may encode? sugar transport protein	29
su1	4.04+-0.01	sugary1, endosperm wrinkled and translucent when dry; the sweet corn gene - recessive is sweet at milk stage; starch debranching enzyme I absent in developing endosperm, phytoglycogen but no debranching enzyme in germinating seeds; *su1-am*	
su2	6.06+-0.01	sugary2, endosperm glassy, translucent, sometimes wrinkled, may encode? starch branching enzyme	175
su3		sugary3, endosperm glassy, smoother than *su1*	585
sup1		suppressor1, dominant *Sup1* modifies *o2* kernels to semi-transparent	348
sus2	9.04	sucrose synthase2, (was *sus1*, *css1*) sucrose synthase-2 of embryo and other tissues, compare *sh1*; location on 9L confirmed by B-A translocations, encodes sucrose synthase	352
sut1		sucrose transport1, anthocyanin accumulates in a non-clonal pattern at tip and margins of leaves soon after emergence from whorl; reduced plant height	57
sy1		yellow scutellum1, tissue yellow in recessive rather than white	564
T		translocation, general symbol for exchange of parts (usually reciprocal) between non-homologous chromosomes	
ta1		transaminase1, electrophoretic mobility; dimeric; intralocus hybrid bands occur; possibly = *got1*	336
tan1	6.09+-0.02	tangled1, alters patterns of cell growth, division and differentiation throughout the plant; irregular cell shapes	429, 555
tau1		tau protein homolog1, (was *csu64*) cDNA to leaf mRNA, homologous to brain specific 14-3-3 protein, tau chain A, may encode? activator of tyrosine and tryptophan hydroxylases	90, 285
tb1	1.11	teosinte branched1, many tillers; ear branches tassel-like	76
tbp2	5.03	TATA-binding protein2, cDNA clone; Genbank annotation encodes TBP that can function in yeast and maps to chromosome 5 near *pgm2*, encodes TATA box binding protein	618
td1	5.06+-0.01	thick tassel dwarf1, plants shortened, tassel dense	12
te1	3.05	terminal ear1, stalked ear appendages at tip; varying to infolded ears	350
tga1	4.04	teosinte glume architecture1, glumes indurated, erect, long, boat-shaped; factor transferred from teosinte	137
tha1		thylakoid assembly1, reduced polypeptides of photosystem II, photosystem I, cytochrome bf; normal coupling factor, normal RUBISCO; missing polypeptides appear to be synthesized normally	25
tha2		thylakoid assembly2, reduced polypeptides of cytochrome bf, photosystems I and II, coupling factor; missing polypeptides appear to be synthesized normally	25
thc1		thiocarbamate sensitive1, sensitive to Eradicane	460
thp1	7.05	thiol protease homolog1, (was *csu5*) cDNA to leaf mRNA, homologous to *Vigna mungo* sulfhydryl-endopeptidase; single copy, may encode? thiol protease	90, 285
tiny fragment 9	9.02+-0.01	centric fragment that carries *Sh1*, *Bz1*, and X component	364

SYMBOL	BIN	NAME, PHENOTYPE	REF
tl		trait was previously symbolized as a gene, *tl1*, but inheritance is complex and irregular; associated with aneuploidy	371
tlr1	1.12+-0.04	tillered1, dominant *Tlr1* plants show extreme tillering	428
tlr2		tillering2, dominant *Tlr2* plants show 2-3 tillers per plant	280
tls1	1.12+-0.04	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	5
Tourist		family of transposable elements, 1-50k copies in genome, average length 133 bp	71
tp1	7.03	teopod1, dominant *Tp1* plants have many tillers, narrow leaves, many small partially podded ears, tassel simple	325
tp2		teopod2, like *tp1*	457
tp3	3.03+-0.01	teopod3, semi-dominant that increases tillering and decreases number of lateral tassel branches; originally identified by J. Beckett, may be allele of *Cg1*	38
tpase		transposase of *Ac*, required for transposition of *Ac*, encodes TPASE	303, 304
tpe1		thin pericarp1, reduced cell number in pericarp (from *Coroica*)	192
tpi1	7.04+-0.01	triose phosphate isomerase1, electrophoretic mobility; plastidial; dimeric; intra/interlocus hybrids occur with *Tpi2*, encodes triose phosphate isomerase (plastidial)	631
tpi2	2.07+-0.01	triose phosphate isomerase2, electrophoretic mobility; plastidial; dimeric; intra/interlocus hybrids occur with *Tpi1*, encodes triose phosphate isomerase (plastidial)	631
tpi3	8.02+-0.01	triose phosphate isomerase3, electrophoretic mobility; cytosolic; dimeric; intra/interlocus hybrids occur with *Tpi4* & *Tpi5*; encodes triose phosphate isomerase (cytosolic)	631
tpi4	3.04	triose phosphate isomerase4, electrophoretic mobility; cytosolic; dimeric; intra/interlocus hybrids occur with *Tpi3* & *Tpi5*; encodes triose phosphate isomerase (cytosolic)	628, 631
tpi5	8.09	triose phosphate isomerase5, electrophoretic mobility; cytosolic; dimeric intra/interlocus hybrids occur with *Tpi3* & *Tpi4*, encodes triose phosphate isomerase (cytosolic)	631
tpk1		tousled protein kinase1, (was *uaz130*) cDNA to endosperm mRNA, very strong homology to *Arabidopsis* protein kinase, TOUSLED, encodes protein kinase, tousled homolog	236
tpm1		thylakoid peptide modifier1, dominant decrease in electrophoretic mobility	384
tr1	2.02+-0.02	two-ranked ear1, distichous vs. decussate phyllotaxy in ear axis; quantitative, one of a family of loci differentiating maize vs. teosinte	310
trAc9705		transposed Activator sequence, probed sequence in Burr et al. (MNL 65:109), location on 1S from data of Diane Burgess, DNAP	79
trisomic		normal chromosome complement plus an additional chromosome	353
trn1	9.05+-0.04	torn1, dominant *Trn1* plants have chlorotic and adherent leaf tissues on later leaves, which become green and healthy after sunlight exposure but are torn	414
tru1		tassels replace upper ears1, upper ear branches tassel-like, tillers bear ears	540
ts1	2.05	tassel seed1, tassel pistillate and pendant; if removed, small ear with irregular kernel placement develops	159
ts2	1.04	tassel seed2, like *ts1*, but tassel branches variably pistillate and staminate; sequence homologous to short chain alcohol dehydrogenases, encodes TS2 protein	159
ts3	1.1	tassel seed3, Dominant *Ts3*, tassel with large sections of either pistillate or staminate flowers in tandem; some pollen.	433, 462
ts4	3.05+-0.01	tassel seed4, tassel compact silky mass, upright, with pistillate and staminate florets; ear silky and proliferated	433, 462
ts5	4.03	tassel seed5, dominant *Ts5* tassels are upright with scattered, short silks; branches mostly pistillate toward the base	164
ts6	1.13+-0.01	tassel seed6, dominant *Ts6* tassels are pistillate to mixed, compact; ear with irregular kernel placement	433
tsc1		tar spot complex1, dominant *Tsc1* confers resistance to tar spot complex	85
tsh1		tassel sheath1, leaves develop at base of tassel branches and spikelet pairs	56
tu1	4.07	tunicate1, dominant *Tu1* ears develop long glumes enclosing individual kernels; tassels develop large, coarse glumes and sex reversal; both inflorescences become grossly vegetative and sterile in homozygotes	111, 112
tua1	1.11	alpha tubulin1, mRNA expressed primarily in roots; member of tandem repeat (see *tua2*), encodes alpha tubulin	387
tua2	1.11	alpha tubulin2, member of tandem repeat (see *tua1*), separated by 1.5 kbp, near *adh1*, preferentially expressed in radicles, root tips and coleoptiles; 6 alpha tubulin genes identified, encodes alpha tubulin	387, 615
tua3		alpha tubulin3, alpha tubulin family; mRNA expressed in all dividing cells examined, encodes alpha tubulin	388
tua4		alpha tubulin4, belongs to alpha tubulin subfamily I, with *tua1* and *tua2*; gene specific cDNA probe, encodes alpha tubulin	615
tua5		alpha tubulin5, alpha tubulin subfamily II with *tua6*; gene specific cDNA probe, encodes alpha tubulin	615
tua6		alpha tubulin6, alpha tubulin subfamily II, gene specific cDNA probe, encodes alpha tubulin	615

SYMBOL	BIN	NAME, PHENOTYPE	REF
tub1		beta tubulin1, genomic clones sequenced; gene-specific probe (by Southern blot) hybridizes to a single transcript size, encodes beta tubulin	254
tub2		beta tubulin2, cDNA sequenced; single copy (Southern blots), encodes beta tubulin	254
tub3		beta tubulin3, cDNA sequence, gene specific probe (Southern blots, sequence), encodes beta tubulin	502
tub4		beta tubulin4, cDNA sequence, gene specific probe (Southerns, sequence), encodes beta tubulin	502
tub5		beta tubulin5, cDNA sequence, gene specific probe (Southern blots, sequence), encodes beta tubulin	502
Ty1		Ty1-copia group retrotransposon candidate, partial genomic sequence homologous to reverse transcriptase, may encode? reverse transcriptase	238a
U5snRNA		U5snRNA, Identity based on homology to *Arabidopsis* clones; genomic clones have distinct 5'-sequence, encodes U5snRNA	313a
ub1		unbranched1, tassel with one spike	427
ubf9		genomic sequence, hybridizing mRNA expressed during cell division and/or cell growth; multiple copies in genome, encodes ribosomal protein 27A	93
ubi1		ubiquitin1, genomic sequence, 7 contiguous direct ubiquitin repeats; transcript specific probe; promoter active in monocots, not in tobacco, encodes polyubiquitin	97
ubi2		ubiquitin2, genomic sequence encodes 7 contiguous ubiquitin monomers; transcript specific probe, encodes polyubiquitin	97
uce1		ubiquitin conjugating enzyme1, (was *uaz102*) cDNA to endosperm mRNA, very strong homology to plant ubiquitin conjugating enzymes, encodes ubiquitin conjugating enzyme	236
ufo1		unstable factor for orange1, dominant *Ufo1* plants have orange color in anthers, silks, and most other plant parts in presence of *P1-WR* or *P1-RR*; growth retarded	595
ugp1		UDP-glucose pyrophosphorylase1, (was *uaz194*) cDNA to endosperm mRNA; matches potato UDP-glucose pyrophosphorylase, encodes UDP-glucose pyrophosphorylase	236
ugu1		UTP-glucoseP uridyltranferase homolog, cDNA to endosperm mRNA, homologous to slime mold UTP--glucose-1-P uridylyltransferase, may encode? UTP-- glucose-1-phosphate uridyltransferase	237
umc217(gfu)	1.03	*umc217(gfu)*, cDNA clone pZmL1032 (cultivar Berkeley Fast) hybridizes to mRNA with very late anaerobic accumulation	455
Uq1		ubiquitous: controlling element mediating *a1-ruq*; *ruq-st* (receptor element), *ruq31*, *ruq66* (receptor elements)	186
Uq2		ubiquitous: one of 5 newly activated *Uq* elements unlinked to *Uq1*	445
Uq3		ubiquitous: one of five newly activated *Uq* elements not linked to *Uq1*	445
Uq4		ubiquitous: one of five newly activated *Uq* elements unlinked to *Uq1*	445
Uq5		ubiquitous: one of five newly activated *Uq* elements not linked to *Uq1*	445
Uq6		ubiquitous: one of five newly activated *Uq* elements not linked to *Uq1*	445
v1	9.04+-0.01	virescent1, yellowish white seedling, greens rapidly; low temperature accentuates	128
v2	5.13+-0.01	virescent2, like *v1*, but greens slowly; low temperature accentuates	128, 157
v3	5.06+-0.01	virescent3, light yellow seedling, greens rapidly; low temperature accentuates	128
v4	2.06	virescent4, like *v2*	128
v5	7.02+-0.01	virescent5, like *v1*, but older leaves have white stripes	128
v8	4.07	virescent8, like *v2*; lethal	129
v12	5.1+-0.01	virescent12, like *v3*	463
v13		virescent13, first leaf with green tip; greens slowly	463
v16	8.07+-0.02	virescent16, like *v2*; deficiency of chloroplastic 16S and 23S rRNA	463
v17	4.06+-0.06	virescent17, like *v1*, but greening from base to tip	463
v18		virescent18, like *v1*	463
v21	8.06+-0.02	virescent1, grainy virescent, greening from tips and margins inward	39
v22	1.1	virescent22, like *v1*	12
v23	4.04	virescent23, like *v1*	12
v24	2.08+-0.03	virescent24, like *v1*, except not cold-sensitive and developmentally conditional high chlorophyll fluorescence attributable to premature assembly of the light harvesting complexes.	416
v25	1.04+-0.03	virescent25, greenish white seedling; greens from base upward	416
v26	2.03+-0.02	virescent26, yellowish white seedling with green leaf tip and midrib	416
v27	7.05+-0.02	virescent27, virescent with many small yellow green streaks; husks and culm whitish at flowering	416
v28	9.01	virescent28, like *v1*, greens slowly	416
v29		virescent29, grainy virescent	416
v29		virescent29, Greens rapidly in longitudinal streaks on leaf blade, changing to green with light green streaks. Expression heightened by cool temperatures (20-25C).	416
v30	9.06+-0.01	virescent30, like *v1*, but more yellow	104
v31	9.01	virescent1, grainy, light green seedling; small green plant with longitudinal white stripes	229
va1	7.03+-0.01	variable sterile1, variable male and female fertility; cytokinesis fails in anaphase I	33

SYMBOL	BIN	NAME, PHENOTYPE	REF
vg1	1.09	vestigial glume1, dominant *Vg1* glumes very small, cob and anthers exposed; upper leaves have scant ligules	567
vp1	3.05	viviparous1, embryo fails to become dormant, viable if transplanted; some alleles dormant; chlorophyll and carotenoids unaffected; anthocyanins in aleurone suppressed	169, 172, 323, 341
vp2	5.06	viviparous2, embryo fails to become dormant; white endosperm, white seedling; anthocyanins unaffected	169, 172
vp5	1.01+-0.01	viviparous5, like *vp2*	491
vp8	1.13+-0.01	viviparous8, embryo fails to become dormant; chlorophyll and carotenoids unaffected; small, pointed-leaf seedlings	491a
vp9	7.02+-0.01	viviparous9, (was *y7, z1*) like *vp2*; *vp9-4889* dormant, pale aleurone, pale green seedling	491a
vp10		viviparous10, yellow endosperm, colored aleurone, green seedlings, adherent	554
vpp1		vacuolar proton pump homolog1, low copy, cDNA to leaf mRNA, strong homology to yeast vacuolar H(+)-ATPase 54 kda subunit, may encode? pyrophosphate-energized proton pump, vacuolar	28, 29
vsi1		variable short internodes1, clusters of 2-4 short internodes, predominantly at base of plant but varies in location; temperature sensitive	144
vsp1		vegetative-specific protein homolog1, (was *uaz246*) cDNA to endosperm mRNA, very strong homology to slime mold vegetative specific protein H7, may encode? vegetative-specific protein	236
vsr1		virescent striped1, dominant *Vsr1* seedlings virescent, greening to white and yellow striped plant	428
w1	6.09+-0.01	white1, white seedling (yellow with *l1*), germinates normally; plastid transcripts variously aberrant	155, 156, 324
w2		white2, white seedling (yellow with *l1*); endosperm pitted and spotted (allele *dek21*); plastid DNA content decreased	324
w3	2.07+-0.01	white3, like *vp2*; *w3-8686* pale endosperm, pale green seedling in dim light	127, 324
w11	9.03	white11, like *w1*	129
w14	6.09+-0.01	white14, like *w1*	121
w15	6.01+-0.01	white15, like *w1*; fails to convert protochlorophyllide to chlorophyllide	121
w16	7.02+-0.01	white16, like *w1*	390
w17	7	white17, like *w1*	390
w18	1.13+-0.01	white seedling18, like *w1*; pale green streaks in some backgrounds	408
w19	3.09	white19, white plant tissue; identified in plants carrying the *a1-x1* deficiency, forming albino chimeras on loss of ring3 carrying *A1-b Sh2*	415, 429, 580
wc1	9.07+-0.01	white cap1, dominant *Wc1* kernels have pale yellow endosperm if *Y1* (pearly white with *y1*); whiteness is emphasized in soft-starch crowns	302
wd1	9	white seedling, deficiency for distal half of first chromomere of short arm; does not complement *pyd1, v28, v31, yg2*	358
wgs1	5.1+-0.04	white green sectors1, white seedling with green sectors	416
whp1	2.1	white pollen1, duplicate factor with *c2* for pollen color and for anthocyanins; encodes chalcone synthase	107
wi1	6.02+-0.01	wilted1, chronic wilting, leaves not as cool as normal; delayed differentiation of metaxylem vessels	468
wi2	3.05+-0.05	wilted2, in dominant *Wi2* plants, top leaves wilt under moisture/temperature stress	407
wi3		wilted3, Like *wi2* but slightly yellowish green.	409
wip1		wound induced protein1, wounding-induced transcript, cDNA clone (601bp) sequenced; homologous to Bowman-Birk proteinase inhibitors, may encode? Bowman-Birk proteinase inhibitor	503
wlu1	3.08+-0.02	white luteus1, pale yellow seedling; lethal	416
wlu2	7.05+-0.02	white luteus2, like *wlu1*	416
wlu3	8.06+-0.02	white luteus3, like *wlu1*	416
wlu4	9.06+-0.03	white luteus4, like *wlu1*	416
wlu5	1.08+-0.01	white luteus5, like *wlu1*	408
wrk1	3.04+-0.01	wrinkled kernel1, dominant *Wrk1* kernels small and wrinkled	428
wrp1	2.08+-0.03	wrinkled plant1, dominant *Wrp1* plants dwarf, leaves and culm longitudinally corrugated; dosage effect	49
ws1		white sheath1, light yellow leaf sheaths; duplicate factor with *ws2*	289
ws2		white sheath2, see *ws1*	289
ws3	2.01+-0.01	white sheath3, white leaf sheath, culm, husks	482
ws4	1.04+-0.03	white sheath4, dominant *Ws4* seedlings and plants lighter green in sheaths	407
wsm1	6.01+-0.01	wheat streak mosaic virus resistance1, dominant resistance	366a
wsm2	3.04+-0.01	wheat streak mosaic virus resistance2, Dominant *Wsm2* like *Wsm1*	365
wsm3		wheat streak mosaic virus resistance3, Partial resistance; semidominant; *Wsm3* plants express delayed WSMV-induced symptoms of dispersed, isolated spots and rings	365
wsp		weak streaked plant, maternally inherited reduced plants	67, 147

SYMBOL	BIN	NAME, PHENOTYPE	REF
wt1	2.05	white tip1, tip of first leaf white and blunt	576
wt2	4.03+-0.03	white tip2, seedling with white leaf tip and crossbands on first 2 leaves	416
wusl1005(gfu)	5.05	wusl1005(gfu), cDNA clone (cultivar Berkeley Fast); continuous anaerobic accumulation of mRNA through 72 h	79, 455
wx1	9.03	waxy1, amylopectin (stained red by iodine) replaces amylose (blue staining) in endosperm and pollen; extensive allelic series, encodes NDP-glucose-starch glucosyltransferase, starch granule-bound	71, 110
wyg1	7.01+-0.01	white yellow green seedling1, whitish, light yellow-green seedling	390
y1	6.01	white1, reduced carotenoid pigments in endosperm; some alleles affect chlorophyll in seedlings (e.g. *y1-8549*), encodes phytoene synthase	119
y10	3.07+-0.01	pale yellow10, pale endosperm; white seedling, lethal	492
y11		pale yellow11, pale endosperm; green seedling	570
y12		pale yellow12, like *y11*	570
y3	2.01+-0.01	pale yellow3, compare *al1*, of which *y3* is evidently an allele.	454
y8	7.01	pale yellow8, pale endosperm	264
y9		pale yellow9, pale endosperm, slightly viviparous; green to pale green seedlings and plants	494, 497
yd2	3.06+-0.01	yellow dwarf2, yellow dwarf seedling, lethal	496
yg1	5.13+-0.01	yellow-green1, yellow-green seedling and plant	170
yg2	9	yellow-green2, like *yg1*; complements *pyd1* but not *wd1*	261
ypt1		ypt homolog1, cDNAs obtained by homology to GTP-binding domain of ras-protein family and mouse ypt protein, encodes ras protein family homolog	443
ys1	5.1+-0.01	yellow stripe1, yellow tissue between leaf veins, reflects iron deficiency symptoms	30
ys2	1.04+-0.03	yellow stripe2, yellow tissue between leaf veins	467
ys3	3.05	yellow stripe3, like *ys1*	643
ysk1	4.04	yellow streaked1, dominant *Ysk1* plants have longitudinal yellow streaks in top 3rd of mature leaves	422
zag1	6.06	zea agamous homolog1, amino acid sequence, deduced from cDNA to influorescence mRNA, 61% identical to Arabidopsis floral homeotic gene protein AG1, may encode? transcription factor	524
zag2	3.05	Zea agamous homolog2, amino acid sequence, deduced from cDNA sequence, has 49% identity to *Arabidopsis*, floral homeotic gene product, may encode? floral transcription factor	524
zap1		zea apetala homolog1, (was *csu137*) low copy number, cDNA to mRNA, nearly identical to *Arabidopsis* floral homeotic gene, *ap1*, may encode? transcription factor, flowering	28, 29
zb1		zebra crossbands1, yellowish crossbands on older leaves	126
zb2		zebra crossbands2, crossbands on seedling leaves	593
zb3	5.13+-0.01	zebra crossbands3, yellowish crossbands on older leaves	131, 166
zb4	1.02	zebra crossbands4, regularly spaced crossbands on earlier leaves; enhanced by cool temperatures	232
zb6	4.06+-0.01	zebra crossbands6, regularly spaced crossbands on earlier leaves; enhanced by cool temperatures	575
zb7	1.11	zebra crossbands7, lighter green crossbands on seedlings; glossy	416
zb8	9.03	zebra crossbands8, yellow-green crossbands on older leaves; strong anthocyanin expression in leaf tip and blade	422, 428
zbr1	4.08	maize beta repeat homolog1, (was *csu166*) cDNA to leaf mRNA homologous to insect giant secretory protein beta repeat, may encode? secretory protein	29
Zeon1		zein retrotransposon; 1k copies of LTR-related sequences, 3-400 copies of internal sequence	251
zl1	1.04	zygotic lethal1, homozygous recessive zygotes do not develop; detected by extreme distortion of ratios for *p1* (1.5 units away). Authentic stocks are no longer available.	163, 164a
zn1		zebra necrotic1, necrotic tissue appears between veins in transverse leaf bands on half-grown or older plants	250
zn2		zebra necrotic2, like *zn1*	201
zp		zein polypeptide, designator for genes encoding zein, encodes zein	559, 560
zp15	6.01	zein protein, 15kDa15, high methionine; genomic blot indicates one or two copies, encodes 15-kDa zein (beta zein)	450a
zp19/22		19/22-kDa zein protein gene family, the major zein gene family, includes subfamilies A20, A30, B49, B59, encodes zein-1 (alpha zein)	77a
zp19/22cluster 1		alpha zein protein cluster1, 56 kb cluster of five alpha-zein, subfamily 4 (SF4, aka: B49; 22A,22B,22C; z1C, with same transcriptional orientation; includes eight repetitive DNA's; only one zein sequence does not have an early, in-frame	326a
zp27		27-kDa zein protein, proline rich; least abundant of zeins in endosperm, encodes 27-kDa zein (gamma zein)	622a
zp27cluster		27kDa zein protein cluster, tandem genes encoding 27k-Da zein, some lines have only one gene in this region, encodes 27-kDa zein (gamma zein)	123a
zpg1		zebra-stripe pale green1, chlorophyll modifications; heterozygote advantage; induced by EMS in Oh43	141

SYMBOL	BIN	NAME, PHENOTYPE	REF
zpl1	4	zein polypeptidesL11, Zp1La - Zp1Lf complex, encodes zein	638
zpl1a	4	zein protein 1a, zein protein characterized by electrophoretic mobility on isoelectric focusing gels, encodes zein	638
zpl1b	4	zein protein 1b, zein protein characterized by electrophoretic mobility on isoelectric focusing gels, encodes zein	638
zpl1c	4	zein protein 1c, zein protein characterized by electrophoretic mobility on isoelectric focusing gels, encodes zein	638
zpl1d	4	zein protein 1d, zein protein characterized by electrophoretic mobility on isoelectric focusing gels, encodes zein	638
zpl1e	4	zein protein 1e, zein protein characterized by electrophoretic mobility on isoelectric focusing gels, encodes zein	638
zpl1f	4.01	zein protein 1f, zein protein characterized by electrophoretic mobility on isoelectric focusing gels, encodes zein	638
zpl2a	4.04	zein polypeptidesL2a, zein protein characterized by electrophoretic mobility on isoelectric focusing gels. Maps near orp1, encodes zein	638
zpl2b	7.02	zein polypeptidesL2b, zein protein characterized by electrophoretic mobility on isoelectric focusing gels, encodes zein	638
zpl3a	4.04+-0.01	zein polypeptidesL3a, zein protein characterized by electrophoretic mobility on isoelectric focusing gels. Near *bnl17.13c*, encodes zein	638
zpr10/(22)	4.01+-0.01	zein-protein regulator, elevation of 10kD zein	44
zrp3		zea root protein3, cDNA expressed only in roots, within a distinct subset of cortical cells, the inner three to four cell layers, most strongly in the tip and 1 cm back, encodes cortical cell protein	269a
zrp4	4.06+-0.06	Zea root preferential4, cDNA, 1.4kb, preferentially accumulates in roots of young plants, may encode? O-methyl transferase	235
zug1		zea UGA3 homolog1, (was *csu9*) cDNA to leaf mRNA, homologous to yeast transcriptional activator, UGA3, may encode? transcription factor	29

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646. Wright, S. et al. 1993. Plant J 3:41-49 *(mfs14, mfs18)*
646a. Wu, S. and Kriz, A.L. 1992. GenBank/EMBL/DDBJ, nucleic acid sequence database *(geb1)*
646b. Yanagisawa, S. and Izui, K. 1993. J Biol Chem 268:16028-16036 *(mnb1, mnb2)*
647. Zaitlin, D. et al. 1992. MNL 66:70-71 *(rDNA5S)*

TABLE OF NEW RANDOM-CDNA GENELIST CANDIDATES:
SEQUENCES AVAILABLE IN GENBANK, DBEST AND WITH KNOWN HOMOLOGY

This year, several hundred sequences, representing randomly selected cDNA's from leaf and endosperm libraries were submitted both to GenBank and the Expressed Sequence Tag database (dbEST). The sequences were submitted by the laboratories of Chris Baysdorfer (CSU identified), Tim Helentjaris and Rob Ferl (UAZ identified). The dbEST performs BLASTX searches and stores the sequences and the homology search results in a file currently accessible by e-mail. The Maize Genome Database, MaizeDB can access most GenBank sequences, at GenBank, by a simple 'click' for WWW browsers (see database section for details). Currently GenBank only provides WWW connections to the latest full release. When dbEST establishes a WWW connection, MaizeDB will 'hot link' to the dbEST files. The cDNA's serve as probes for RFLP mapping at Tucson, AZ (electronic bulletin board, grasses@net.bio.net; Helentjaris, T. et al., 1994 MNL 68) and Columbia, MO. (Chao, S. et al. 1994 TAG in press). The sequence information has been used to construct PCR mapping probes (example, bnl17.23(pal)). The table below only includes sequences that have been submitted to GenBank and, additionally, have homology recognition or probe unique map sites. Map information, including map scores, is being entered into MaizeDB and, thereby, is available as soon as entered.

NOMENCLATURE and CRITERIA for GENE DESIGNATION: If a cDNA has any homology, however weak, to an entry in the sequence databases, it has been named based on that homology, using the maize nomenclature guidelines. When the best homolog is another random cDNA with unknown function, but there is also homology to a characterized sequence, the name is based on the characterized sequence. If the homology is to a gene previously identified in maize, it is named with the mnemonic of the first reported gene, immediately followed by '*' and then the probe name. If the laboratory submitting the sequence has not yet assigned a probe name for mapping, the GenBank identifier has been temporarily assigned. MaizeDB stores all synonyms.

PROBED SITES: Corresponding probed site(s) retain the laboratory probe name, with any gene designation attached as a parenthetic suffix. Multiple sites for a given probe are distinguished with a letter (a,b,c,etc) immediately following the probe name and preceding any parenthetic suffix. For example, the names of two sites, probed by uaz218, which encodes a new starch synthase gene, *gss1*, would be named *uaz218a(gss1)*; *uaz218b(gss1)*. until evidence is adduced that one or both sites actually encode this product

GENELIST STATUS: Genes for which a clearly distinct, defined sequence is indicated by surveys or by mapping were placed on the genelist. Some 79 have been placed on the gene list.

PROBED SITES or GENES?: In some cases, a single, unique site could be distinguished on RFLP patterns and these probed sites are considered to be the site that encodes the cDNA. Otherwise, the probed sites are considered candidate locations for the gene.

HOMOLOGY	NO MAP DATA	UNIQUE SITE	MULTIPLE or UNCLEAR
none	gene function unknown *csu9(gfu)*	genelist, gene, function unknown *csu173(gfu)*	gene, function unknown *csu11(gfu)*
hit, new	genelist *rfz1*	probed site = gene genelist *cdc48*	probed sites are candidates genelist *ltf1*
hit, not new	potential allele of known gene *glu*uazT14748*	Allele:If map data not in conflict; name as allele of known gene *mdh4* Genelist: If maps to a clearly defined, new location	possible allele of known gene *pdk*uaz153*

GENELIST CANDIDATES and DESIGNATED GENES with CANDIDATE PRODUCTS
(sequences with no BLASTX homology not listed unless mapped to gene location; please refer to dbEST for updated homology records and the BLASTX search results)

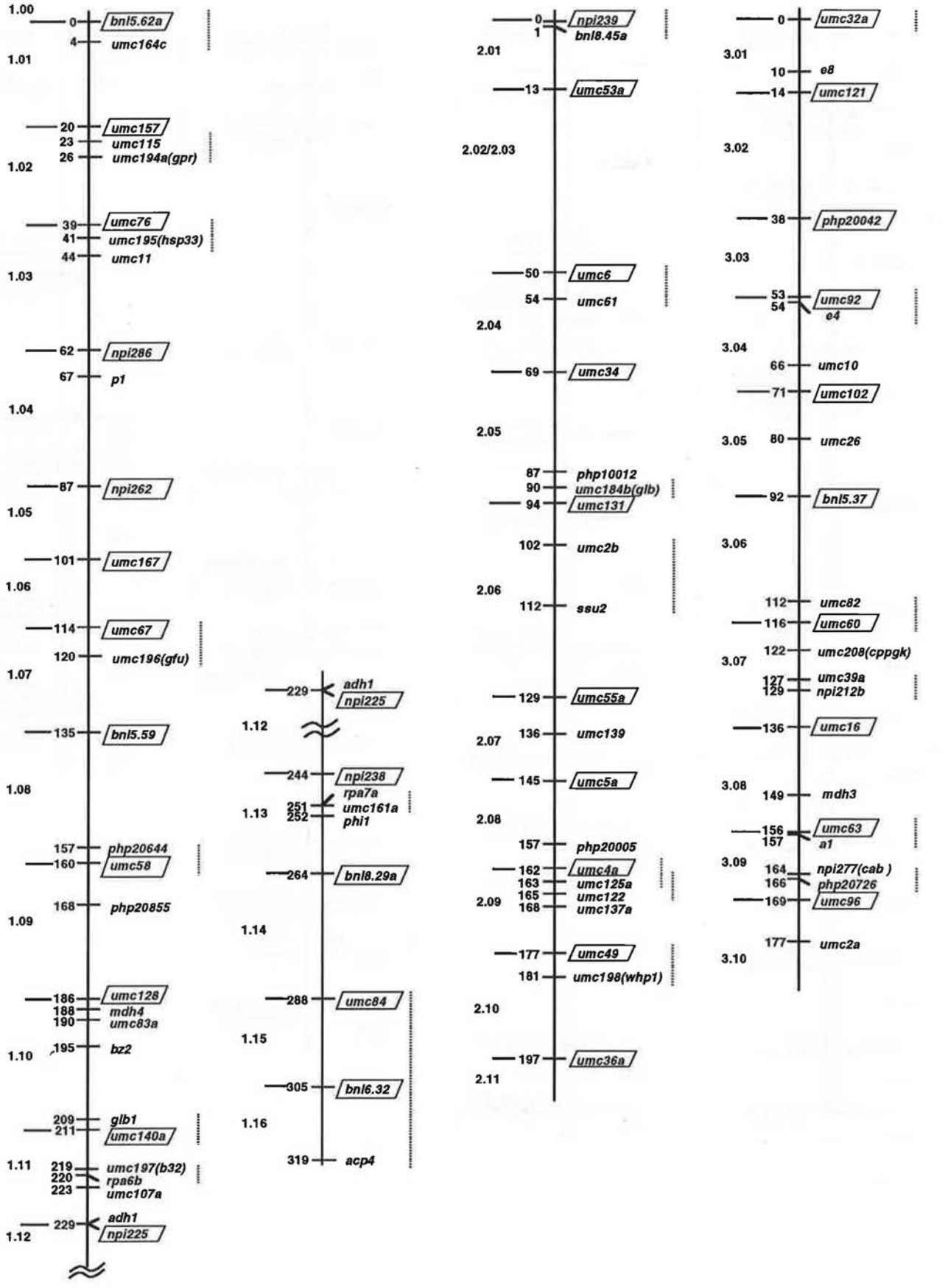
PROBE	GENE SYMBOL	GENBANK	UAZ CLONE ID	POS (bin)[a]	HOMOLOGOUS PRODUCT
csu5	*thp1*	M95060		7.05	protease: thiol protease
csu6	*stm1*	T12528, T12529		+	potato stolon tip protein
csu9	*zug1*	T12534			transcription factor, yeast UGA3
csu12	*cin*csu12*	M95061		+	reverse transcriptase
csu13	*h1*csu13*	T12655, T12656			histone 1
csu17	*rnp1*	T12657, T12658		+	RNA binding protein, chloroplast
csu18	*psa5*	T12659, T12660		+	photosystem I, subunit N
csu19	*cld1*	T12661, T12662		+	cold-regulated protein
csu21	*map1*	T12663			calmodulin binding protein, microtubule
csu25	*cyp1*	T12664		+	cytochrome P450
csu27	*bcl1*	T12665		+	cell cycle protein, CDC10
csu28	*rps22*	M95062			ribosomal protein S22
csu30	*atp1*	M95063		3.05	ATPase: vacuolar, proteolipid
csu34	*rps8*	M95064		+	ribosomal protein S8
csu36	*rpl19*	M95065		+	ribosomal protein L19
csu37	*vpp1*	T12671		+	ATPase, vacuolar
csu39	*csu39(gfu)*	T12673		4.09	
csu40	*grx1*	T12674		+	glutaredoxin
csu43	*csu43(gfu)*	T12675		9.03	

csu56	*ohp*csu56*	T12687		+	transcription factor, o2 heterodimerizing protein
csu63	*cdj1*	T12693			chaperone DNA J
csu64	*tau1*	M95066		+	activator of tyrosine and tryptophan hydroxylases
csu65	*ans1*	M95067		+	anthranilate synthase
csu66	*lhcb*csu66*	M95068			light-harvesting CAB protein
csu67	*psa6*	T12694, T12695		+	photosystem I, subunit K
csu68	*ant*csu68*	T12696, T12697		+	permease: adenine nucleotide translocase
csu70	*csu70(gfu)*	T12698		6.01	
csu74	*fdx*csu74*	T12699			ferredoxin
csu77	*mdh4*	M95069			malate dehydrogenase
csu96	*psei*csu96*	M95070		+	protease inhibitor: cysteine protease inhibitor
csu102	*lhcb*csu102*	T12718			light-harvesting CAB protein
csu103	*hupm1*	T12719		+	hupm/hypb protein
csu108	*gbp1*	M95071		+	GTP binding protein
csu110	*ets1*	T12722		+	transcription factor, ets-family
csu111	*gsr1*	T12723		1.02	glutathione reductase
csu116	*elf1*	M95072		+	elongation factor, 1-alpha subunit
csu117	*lhcb*csu117*	T12725			light-harvesting CAB protein
csu125	*cah1*	M95073			carbonic anhydrase
csu129	*ntm9*	T12726, T12727		+	sodium channel inhibitor
csu133	*prp2*	T12729		2.05	pathogenesis-related protein
csu136	*plt*csu136*	M95074			phospholipid transfer protein
csu137	*zap1*	T12732, T12733		+	transcription factor, flowering
csu138	*pgp1*	T12734, T12735			glycoprotein: P-glycoprotein
csu142	*stp1*	T12737		8.05	permease: sugar transport protein
csu145	*pck1*	T12738		+	phosphoenolpyruvate carboxykinase
csu146	*cdc48*	T12739		6.02	cell cycle protein, CDC48
csu148	*csu148(gfu)*	T12741			calnexin
csu149	*sca*csu149*	T12742		5	short chain alcohol dehydrogenase
csu150	*rpo1*	M95075		+	RNA polymerase
csu152	*gpb1*	M95076			glyceraldehyde 3-phosphate dHase B (NADP+)(phosphoryl- ating)
csu154	*eif5*	T12744		+	elongation initiation, factor 5
csu156	*pal1*	M95077		+	phenylalanine ammonia lyase
csu158	*eno*csu158*	T14753		9.03	enolase
csu160	*lox1*	T12746		+	lipoxygenase
csu166	*zbr1*	T12749		4.08	secretory protein
csu169	*mtl*csu169*	T12751			metallothionein
csu173	*csu173(gfu)*	T12754		5.07	
uaz#	*agp*uazT14743*	T14743	05c04h06	+	ADP glucose pyrophosphorylase
uaz#	*bvp2*	T14732	05c04f07		glycoprotein
uaz#	*chs1*	T14695, T14696	05c04f07		chitin synthase
uaz#	*glu*uazT14748*	T14748	05c01c02		beta-glucosidase
uaz#	*gpc*uazT14761*	T14761	05c01h01		glyceraldehyde 3-phosphate dHase
uaz#	*his4*uazT14749*	T14749	05c01c03		histone 4
uaz#	*hvp1*	T14771	05c02b04		transcription factor, human virus
uaz#	*mde1*	T14784	05c02d07		mouse, viral protein homolog
uaz#	*mrp1*	T14770	05c02b03		E. coli., MRP
uaz#	*msr1*	T14702, T14703	05c04d02		macrophage scavenger protein
uaz#	*pdk*uazT14754*	T14754	05c01c10		pyruvate, orthophos- phate dikinase
uaz#	*pho1*	T14680, T14681	05c04b08		transcription factor, phosphate metabolism
uaz#	*plt*uazT14763*	T14673	5c02a01	+	phospholipid transfer protein
uaz#	*pox1*	T14744	05c04h07		fowlpox virus, core protein
uaz#	*rap1*	T14652	05c01b09		cell cycle protein, retinoblastoma
uaz#	*rfz1*	T14765	05c02a04		tissue polarity protein
uaz#	*rps11*T14795*	T14795	05c02f12		ribosomal protein S11
uaz#	*sus*uazT14713*	T14713	05c04d10		sucrose synthase
uaz#	*ugu*uazT14742*	T14742	05c04h05		UTP-- glucose-1- phosphate uridyl- transferase
uaz#	*ugu1*	T14728	05c04e10		UTP-- glucose-1- phosphate uridyl- transferase
uaz#	*zag*uaz231*	T14687, T14688	05c04c02		transcription factor, flowering
uaz#	*zp19/22**	T14705, T14706	05c04d05		zein-1 (alpha zein)
uaz#	*zp19/22**	T14726	05c04e08		zein-1 (alpha zein)
uaz#	*zp19/22**	T14733	05c04f08		zein-1 (alpha zein)
uaz5	*zp19/22*uaz5*	T14767	05c02a08	+	zein-1 (alpha zein)
uaz102	*uce1*	T14794	05c02f11		ubiquitin conjugating enzyme
uaz128	*zp19/22**	T14751	05c01c05		zein-1 (alpha zein)

uaz130	*tpk1*	T14658, T14659	05c04a03	+	protein kinase, tousled homolog
uaz131	*dts1*	T14746	05c01b12	+	aspartyl-tRNA synthetase, alpha-2 subunit
uaz144	*hsk1*	T14673	05c04b04	+	high sulfur keratin homolog
uaz151	*sar1*	T14655	05c03g12	+	GTP-binding protein, SAR1 homolog
uaz152	*sdh1*	T14656	05c04a01	+	sorbitol dehydrogenase
uaz153	*pdk*uaz153*	T14657	05c04a02	+	pyruvate, orthophosphate dikinase
uaz154	*sus*uaz154*	T14660, T14661	05c04a07		sucrose synthase
uaz155	*ant*uaz155*	T14665	05c04a11	+	permease: adenine nucleotide translocase
uaz157	*rpl19*uaz157*	T14670, T14671	05c04b03	+	ribosomal protein L19
uaz158	*alt*uaz158*	T14674, T14675	05c04b05	+	alanine amino transferase
uaz161	*elf2*	T14689, T14690	05c04c04	+	elongation factor, 1-gamma
uaz169	*zp15*uaz169*	T14764	05c02a02		zein: 15 kDa zein (beta zein)
uaz171	*hsp18*uaz171*	T14788	05c02d11	+	heat shock protein, 18kDa
uaz188	*pdk*uaz188*	T14704	05c04d04		pyruvate, orthophosphate dikinase
uaz189	*rpl5*	T14714,T14715	05c04d11	+	ribosomal protein L5
uaz190	*gpc*uaz190*	T14755	05c01c11	+	glyceraldehyde 3-phosphate dHase C, cytosolic
uaz193	*rip*uaz193*	T14729	05c04f01	+	ribosome-inactivating protein
uaz194	*ugp1*	T14797	05c02h07	+	UDP-glucose pyrophos-horylase
uaz196	*pdk*uaz196*	T14738	05c04g07		pyruvate, orthophosphate dikinase
uaz198	*rpl10*	T14756	05c01d03	+	ribosomal protein L10e
uaz199	*hca1*	T14693	05c04c07		glycoprotein
uaz204	*prr1*	T14721, T14722	05c04e05		putidaredoxin reductase
uaz206	*uce*uaz206*	T14707, T14708	05c04d06	+	ubiquitin conjugating enzyme
uaz207	*bvp1*	T14709, T14710	05c04d07	+	transcription factor, bovine virus
uaz208	*mta1*	T14711, T14712	05c04d09	+	glycoprotein
uaz210	*hsp18*uaz210*	T14730	05c04f02	+	heat shock protein, 18 kDa
uaz216	*myb*uaz216*	T14694	05c04c08	+	transcription factor: myb protein
uaz218	*gss1*	T14684	05c04b10		starch synthase
uaz219	*hsp70*uaz219*	T14741	05c04h04	+	heat shock protein, 70kDa
uaz220	*elf*uaz220*	T14798	05c03c06	+	elongation factor, 1-alpha subunit
uaz227	*end1*	T14666, T14667	05c04a12	+	root meristem protein
uaz228	*his2b*(uaz228)*	T14716	05c04d12	+	histone 2B
uaz229	*sbe*uaz229*	T14653, T14654	05c03g08	+	starch branching enzyme II
uaz230	*abt1*	T14678	05c04b07	+	permease
uaz232	*sci*uaz232*	T14663	05c04a08	+	protease inhibitor: subtilisin-chymo-rypsin inhibitor
uaz237	*ptc1*	T14766	05c02a05	+	protease: proteasome (endopeptidase) component C9
uaz238	*ppi*uaz238*	T14778	05c02c04	+	peptidyl-prolyl cis-trans isomerase
uaz241	*prh2*	T14676, T14677	05c04b06	+	protein phosphatase
uaz242	*clp1*	T14785	05c02d08	+	protease: Clp ATP-dependent protease, chloroplast
uaz243	*atp*uaz243*	T14725	05c04e07	+	ATP synthase beta chain, mitochondrial
uaz244	*prh*uaz244*	T14691, T14692	05c04c05	+	protein phosphatase: serine/threonine
uaz246	*vsp1*	T14752	05c01c06	+	vegetative-specific protein
uaz248	*his3*uaz248*	T14800	05c03h09	+	histone 3
uaz249	*ubf9*uaz249*	T14781	05c02d01	+	ribosomal protein S27A
uaz250	*nac1*	T14760	+5c01g10		salt stress protein
uaz275	*ltf1*	T14772	05c02b05		transcription factor, lysr
uaz280	*vpp*uaz280*	T14790	05c02e08	+	ATPase, vacuolar
uaz282	*pop1*	T14791	05c02f05	+	permease: organellar permease
uaz285	*tpase*uaz285*	T14723, T14724	05c04e06	+	Ac transposase
uaz288	*ppi*uaz288*	T14750	05c01c04	+	peptidyl-prolyl cis-trans isomerase

[a]A '+' signifies that map information for probed sites is available per Helentjaris et al., MNL68 and Chao et al., in press.

Mary Polacco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23 umc115
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1.02
39 umc76
41 umc195(hsp33)
44 umc11
1.03
62 npi286
67 p1
1.04
87 npi262
1.05
101 umc167
1.06
114 umc67
120 umc196(gfu)
1.07
135 bnl5.59
1.08
157 php20644
160 umc58
168 php20855
1.09
186 umc128
188 mdh4
190 umc83a
1.10
195 bz2
209 glb1
211 umc140a
1.11
219 umc197(b32)
220 rpa6b
223 umc107a
229 adh1
1.12 npi225
229 adh1
npi225
1.12
244 npi238
rpa7a
251 umc161a
1.13 252 phi1
264 bnl8.29a
1.14
288 umc84
1.15
305 bnl6.32
1.16
319 acp4
0 npi239
1 bnl8.45a
2.01
13 umc53a
2.02/2.03
50 umc6
54 umc61
2.04
69 umc34
2.05
87 php10012
90 umc184b(glb)
94 umc131
102 umc2b
2.06
112 ssu2
129 umc55a
2.07
136 umc139
145 umc5a
2.08
157 php20005
162 umc4a
163 umc125a
165 umc122
2.09 168 umc137a
177 umc49
181 umc198(whp1)
2.10
197 umc36a
2.11
0 umc32a
3.01
10 e8
14 umc121
3.02
38 php20042
3.03
53 umc92
54 e4
3.04
66 umc10
71 umc102
3.05 80 umc26
92 bnl5.37
3.06
112 umc82
116 umc60
122 umc208(cppgk)
3.07
127 umc39a
129 npi212b
136 umc16
3.08
149 mdh3
156 umc63
157 a1
3.09
164 npi277(cab)
166 php20726
169 umc96
177 umc2a
3.10


0 php20725
4.01
14 umc31a
15 umc87a
19 adh2
4.02
28 bnl5.46
4.03/4.04
58 gpc1
65 umc201(nr)
66 umc42a
69 umc47
4.05/4.06
105 prh1
107 umc66a
110 umc19
4.07
126 umc133a
133 umc15a
4.08
139 ssu1
150 umc52
4.09
170 php20608
4.10
188 umc111
4.11
200 bnl8.23
4.12
0 bnl6.25
5.01
7 rpa7b
11 umc147a
5.02/5.03
39 pgm2
42 rpa6a
49 umc27a
umc186b(Bs1)
5.04
55 umc166a
60 umc43
5.05
76 umc 40
5.06
83 bnl7.71
92 bnl5.71
5.07
117 umc126a
119 umc54
5.08
123 umc51
125 umc141
5.09
137 umc209(prk)
150 umc108
5.10
160 umc68
5.11/5.12
191 umc104b
5.13
196 rpa3
204 php10017
5.14
0 gpc2
2 umc85
6.01
10 pgd1
16 enp1
17 umc59
18 oec33
6.02/6.03
58 umc65
6.04/6.05
82 umc204(bz1)
86 pl1
99 umc180(pep)
104 umc21
6.06
113 rab17
116 umc46
121 umc173b(pdk)
6.07
133 umc38a
6.08
152 umc132
6.09
163 umc62
170 idh2
6.10
172 mdh2
179 umc134a
6.11
0 php20581b
7.00
9 npi400a
7.01/7.02
41 umc136
44 npi112
45 umc116
7.03
71 umc110a
7.04
78 umc125b
79 bnl7.61
80 bnl14.07
e1
7.05
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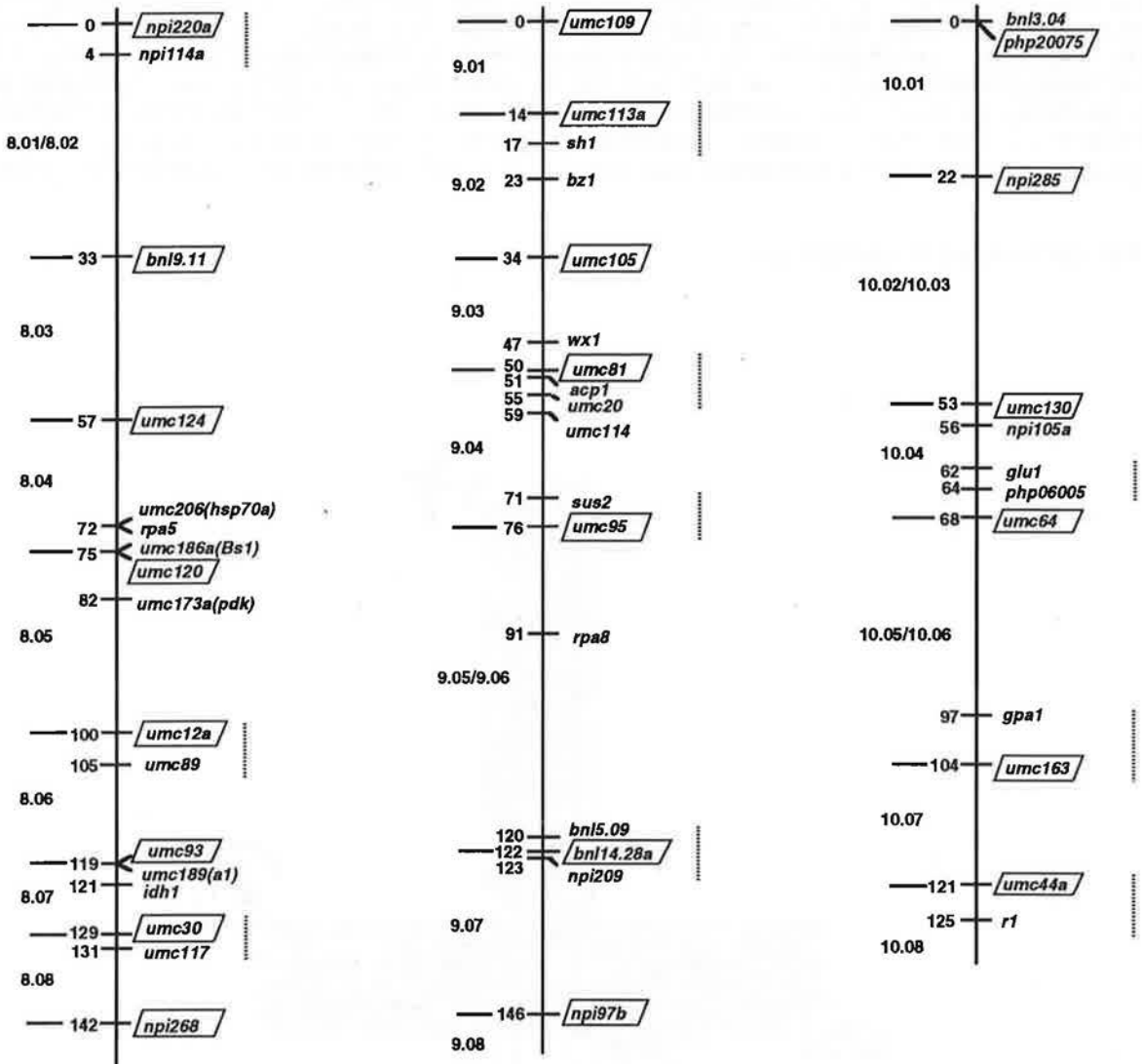
107 php20690
109 bnl16.06
umc45
7.06
122 npi611
123 php20020
125 umc168
129 umc35
7.07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8.03
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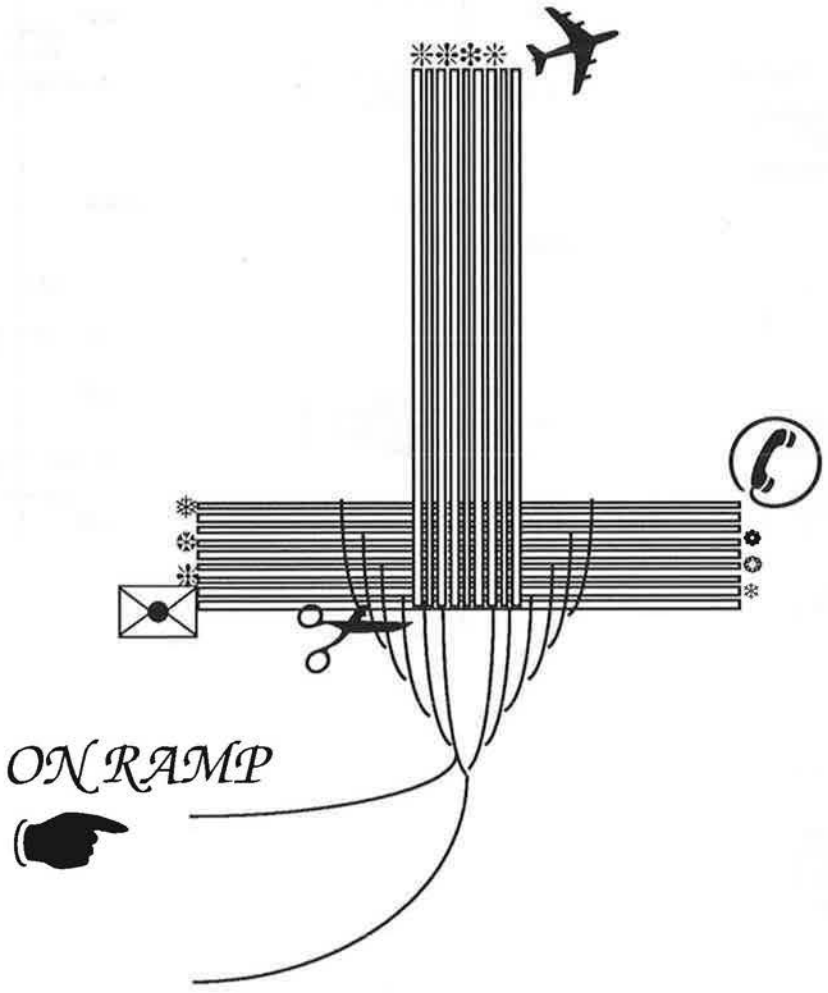
8.04
72 umc206(hsp70a)
rpa5
75 umc186a(Bs1)
umc120
82 umc173a(pdk)
8.05
100 umc12a
105 umc89
8.06
119 umc93
umc189(a1)
8.07 121 idh1
129 umc30
131 umc117
8.08
142 npi268
8.09
165 npi414
8.10
175 npi438b
umc7
8.11
183 npi107
0 umc109
9.01
14 umc113a
17 sh1
9.02 23 bz1
34 umc105
9.03
47 wx1
50 umc81
51 acp1
55 umc20
59 umc114
9.04
71 sus2
76 umc95
91 rpa8
9.05/9.06
120 bnl5.09
122 bnl14.28a
123 npi209
9.07
146 npi97b
9.08
0 bnl3.04
php20075
10.01
22 npi285
10.02/10.03
53 umc130
56 npi105a
10.04
62 glu1
64 php06005
68 umc64
10.05/10.06
97 gpa1
104 umc163
10.07
121 umc44a
125 r1
10.08

MOLECULAR MAP BASED ON TXCM AND COXTX RECOMBINANT INBRED FAMILIES

This map, for which most of the allele distributions have been contributed by other investigators, combines segregation data in two recombinant inbred populations (Burr et al. Genetics 118:519-526, 1988). These data were subjected to MapMaker 3.0. We first developed framework maps for each chromosome. The minimum value for entering the framework was a LOD of 2.0. These loci appear in bold. Loci not separated by recombination are shown on the same line. Two-point map distances in cM are shown. Additional loci shown in normal type are linked to the nearest framework marker with a LOD of 3.0 or greater. They are shown with the two-point distances separating them from the nearest framework marker. Semicolons were used when there were too many markers to fit on one line. Estimated positions for the centromeres are indicated by heavy bars. This work was funded in part by the Maize Genome Database Project.

Eileen C. Matz, Frances A. Burr, and Benjamin Burr

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bnl(tas1C)
12.4
bnl8.05 1.1 cdo1081A 1.3 bnl5.62 3.3 umc94A
7.2
uaz104
5.5
npi97A 2.9 ncr(cat2) 6.5 cdo20A
6.1
umc157 1.3 csic(rab30) 1.3 npi579B 2.6 npi406 3.8 uaz2A 4.1 npi415
5.6
dnap9705::Ac
2.6
npi411B
2.7
npi93B 1.2 npi109A 1.3 npi423
1.9
npi403B
0.6
uaz1 1.1 bnl9.13
4.0
umc76
3.8
cdo1387B 1.2 npi439A
2.8
npi234 umc11 npi241B 1.4 bnl10.38
3.0
bnl1.326 3.0 koln9A
4.6
wusl1032 1.8 npi448 2.9 umc8A 4.1 npi242B
5.4
npi427B umc13 1.3 wusl1120
2.7
npi286 p1 pge(phyb1) umc(hcf3) 0.6 ynh21;4.7 bnl12.06A;6.5 cdo38A;7.3 npi(sod4);9.9 cdo938A ;10.5 ncr(sod4A);14.4 rz672A
7.5
bnl2.323
14.4
npi453 1.3 npi262 2.4 rz500 8.4 bnl7.21
6.4
rz296A rz421 cdo344C uaz7 uaz8 0.6 uaz9 1.2 rz323A 1.2 uaz13 1.3 bnl1.556 1.3 uaz5 1.3 uaz6
2.5
npi304 npi598 npi214 1.4 npi401 1.4 uaz4
5.2
ucsd61A 1.3 umc67 1.4 npi279 2.8 uaz17
3.6
npi272 npi429 1.4 npi258 3.4 bnl5.59
2.5
csu60 uaz14A uaz15 1.1 dup382 3.8 cdo464A
2.5
uwo2 1.1 cdo475B 1.1 cdo595 2.2 ucsd72A 3.7 umc82B
3.2
umc58 1.1 cdo116 1.1 rz28A 1.4 umc119
9.7
npi224G wsu(nia4) 1.6 ncr(nrA) 2.6 uaz18D
13.4
uaz19C uaz20A
4.6
npi605 2.9 npi566
3.6
umc23A umc33
3.2
npi236 1.2 uaz2B
1.2
bnl17.15(bt2)
3.6
bcd207A cdo94B 1.1 bcd386A
3.6
umc128 1.1 rny(pcr1) 1.3 npi447
0.6
umc37
4.6
npi569A npi573 npi614 mdh4 1.2 npi120 1.4 npi255
1.2
umc83 0.1 dup135A 1.1 cdo98B 2.8 bnl17.06
3.1
bz2
2.2
dup103
4.7
bnl8.10 2.2 dup218B 7.4 umc27B
8.7
npi615 3.8 cdo795A 4.3 rz403
6.9
ynh20 2.5 bcd1072C 3.7 tbp1
6.3
phyA1 bnl17.21(tub) 1.1 kn1 2.7 umc107A 3.5 cdo122A 3.7 bnl17.04 5.2 bnl15.18
2.7
adh1 pge14.2 1.2 umc106A
1.0
umc72B npi98A pge(C2)
4.3
npi282B 1.1 npi407
2.5
npi581A
1.2
bnl17.18B umc147B 0.6 bnl7.25
3.8
phi1 pge5B pgeF8 uaz21A 1.3 npi75B
5.5
npi238 0.6 bnl8.08A 1.1 gdh1
7.0
bnl8.29A ucsd44A 1.8 npi241A
12.0
csh(chi1) umc84 1.1 cdo457B
11.5
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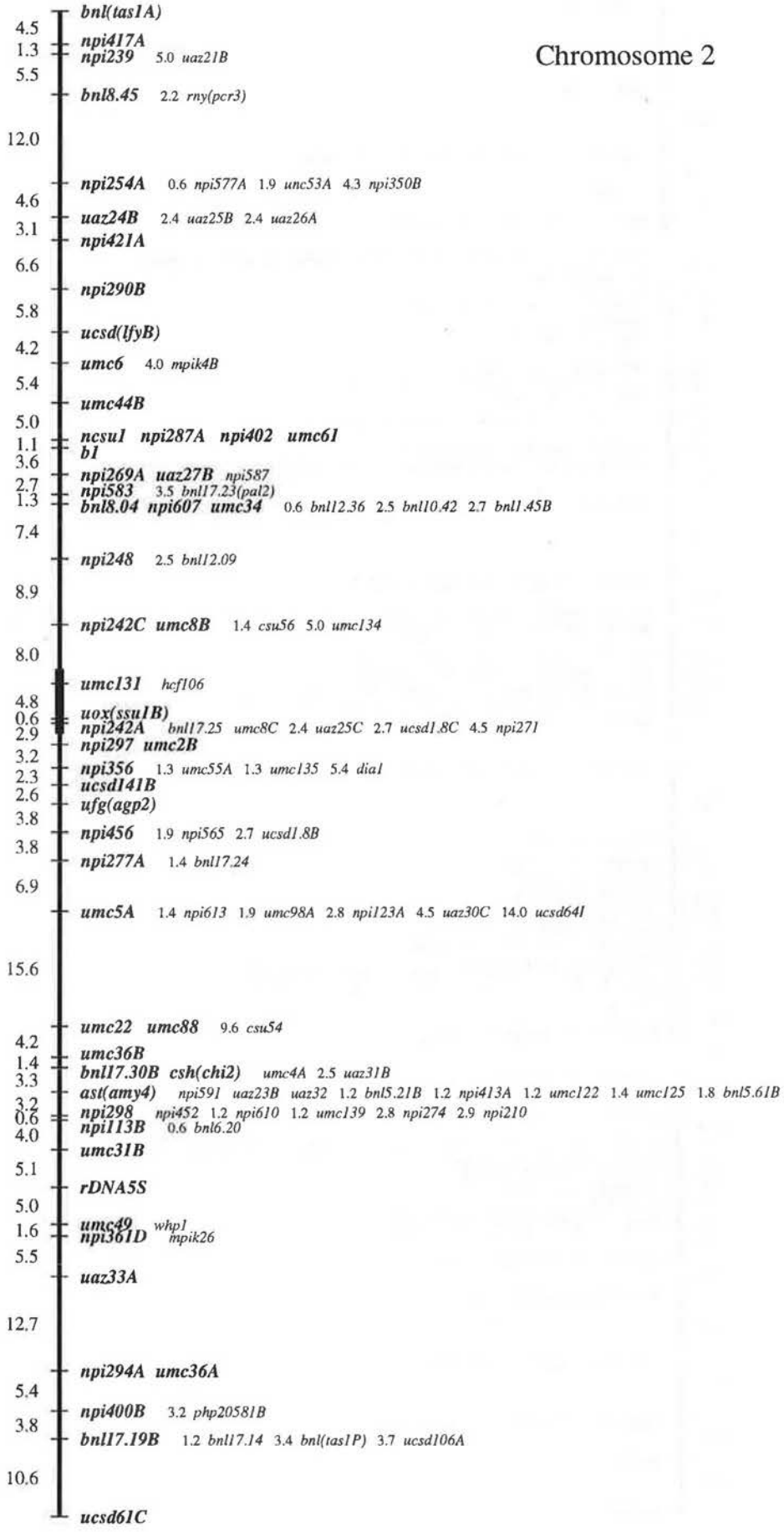
uaz22
9.9
mpik9


Chromosome 2
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1.3
npi239 5.0 uaz21B
5.5
bnl8.45 2.2 rny(pcr3)
12.0
npi254A 0.6 npi577A 1.9 unc53A 4.3 npi350B
4.6
uaz24B 2.4 uaz25B 2.4 uaz26A
3.1
npi421A
6.6
npi290B
5.8
ucsd(lfyB)
4.2
umc6 4.0 mpik4B
5.4
umc44B
5.0
ncsu1 npi287A npi402 umc61
1.1
b1
3.6
npi269A uaz27B npi587
2.7
npi583 3.5 bnl17.23(pal2)
1.3
bnl8.04 npi607 umc34 0.6 bnl12.36 2.5 bnl10.42 2.7 bnl1.45B
7.4
npi248 2.5 bnl12.09
8.9
npi242C umc8B 1.4 csu56 5.0 umc134
8.0
umc131 hcf106
4.8
uox(ssu1B)
0.6
npi242A bnl17.25 umc8C 2.4 uaz25C 2.7 ucsd1.8C 4.5 npi271
2.9
npi297 umc2B
3.2
npi356 1.3 umc55A 1.3 umc135 5.4 dia1
2.3
ucsd141B
2.6
ufg(agp2)
3.8
npi456 1.9 npi565 2.7 ucsd1.8B
3.8
npi277A 1.4 bnl17.24
6.9
umc5A 1.4 npi613 1.9 umc98A 2.8 npi123A 4.5 uaz30C 14.0 ucsd641
15.6
umc22 umc88 9.6 csu54
4.2
umc36B
1.4
bnl17.30B csh(chi2) umc4A 2.5 uaz31B
3.3
ast(amy4) npi591 uaz23B uaz32 1.2 bnl5.21B 1.2 npi413A 1.2 umc122 1.4 umc125 1.8 bnl5.61B
3.2
npi298 npi452 1.2 npi610 1.2 umc139 2.8 npi274 2.9 npi210
0.6
npi113B 0.6 bnl6.20
4.0
umc31B
5.1
rDNA5S
5.0
umc49 whp1
1.6
npi361D mpik26
5.5
uaz33A
12.7
npi294A umc36A
5.4
npi400B 3.2 php20581B
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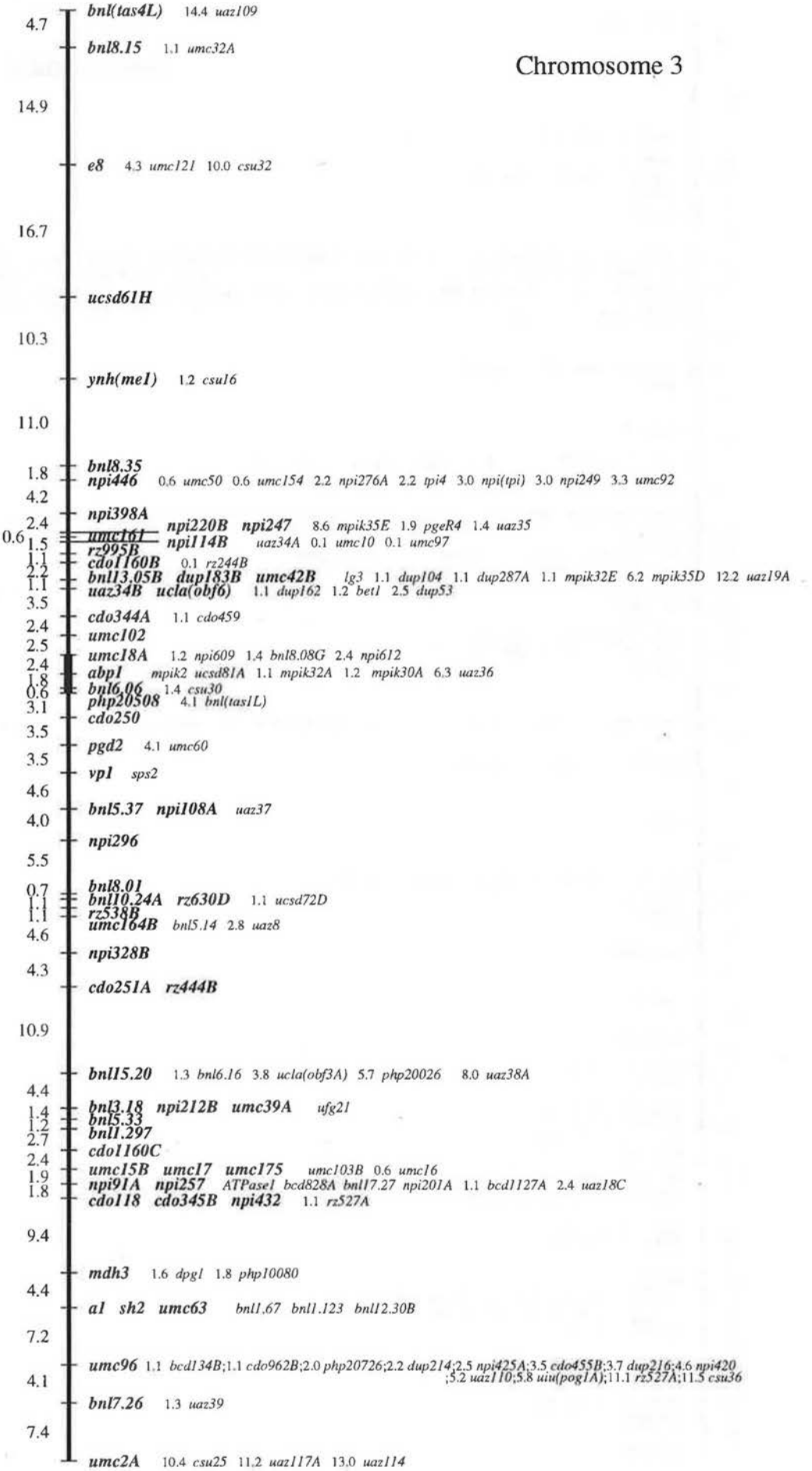
ucsd61C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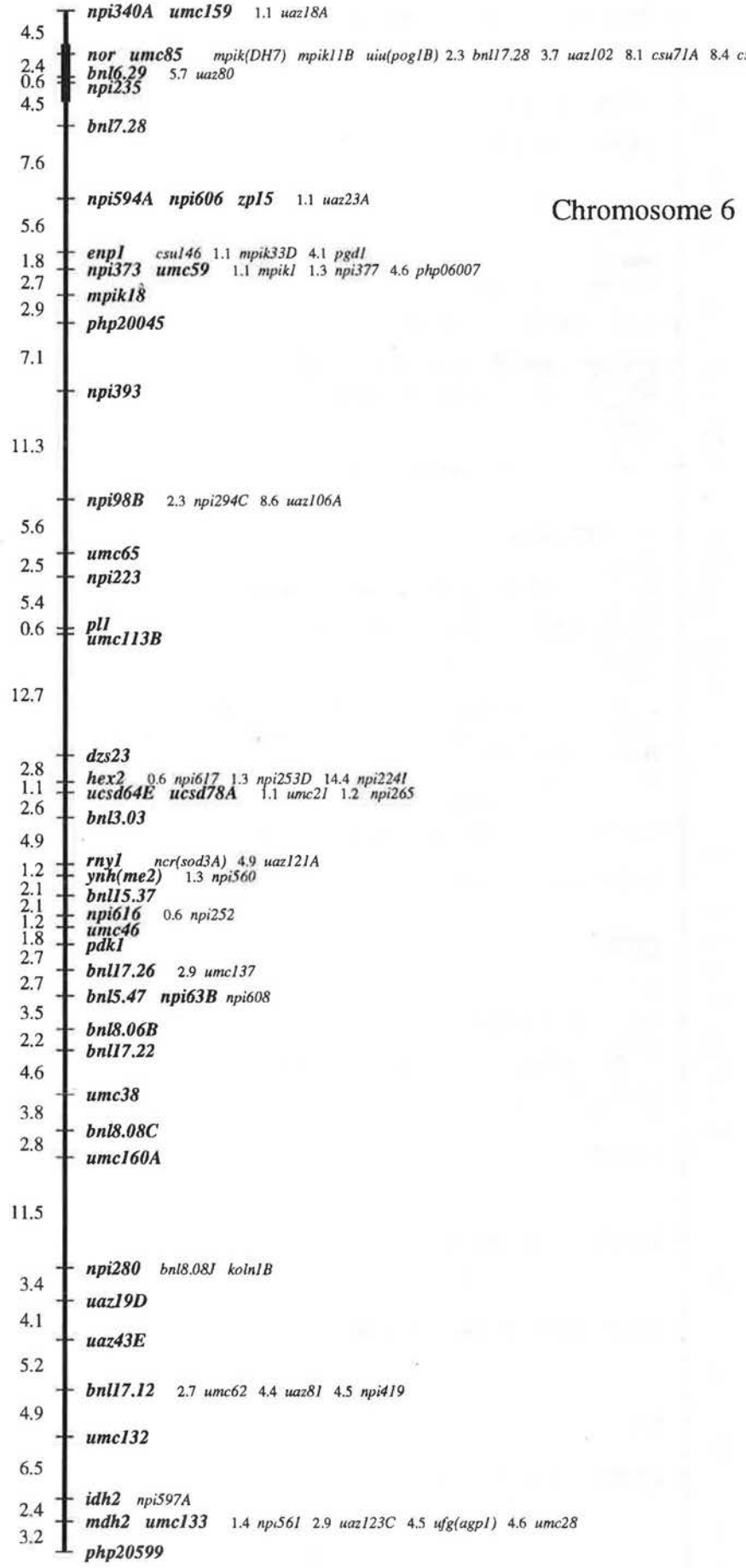
14.9
e8 4.3 umc121 10.0 csu32
16.7
ucsd61H
10.3
ynh(me1) 1.2 csu16
11.0
1.8 bnl8.35
npi446 0.6 umc50 0.6 umc154 2.2 npi276A 2.2 tpi4 3.0 npi(tpi) 3.0 npi249 3.3 umc92
4.2
npi398A
2.4 npi220B npi247 8.6 mpik35E 1.9 pgeR4 1.4 uaz35
0.6 1.5 umc161 npi114B uaz34A 0.1 umc10 0.1 umc97
1.1 rz995B
cdo1160B 0.1 rz244B
2.2 bnl13.05B dup183B umc42B lg3 1.1 dup104 1.1 dup287A 1.1 mpik32E 6.2 mpik35D 12.2 uaz19A
1.1 uaz34B ucla(obf6) 1.1 dup162 1.2 bet1 2.5 dup53
3.5
cdo344A 1.1 cdo459
2.4 umc102
2.5
umc18A 1.2 npi609 1.4 bnl8.08G 2.4 npi612
2.4 abp1 mpik2 ucsd81A 1.1 mpik32A 1.2 mpik30A 6.3 uaz36
1.8 bnl6.06 1.4 csu30
0.6 php20508 4.1 bnl(tas1L)
3.1 cdo250
3.5
pgd2 4.1 umc60
3.5 vp1 sps2
4.6
bnl5.37 npi108A uaz37
4.0 npi296
5.5
0.7 bnl8.01
1.1 bnl10.24A rz630D 1.1 ucsd72D
1.1 rz538B
umc164B bnl5.14 2.8 uaz8
4.6
npi328B
4.3
cdo251A rz444B
10.9
bnl15.20 1.3 bnl6.16 3.8 ucla(obf3A) 5.7 php20026 8.0 uaz38A
4.4
1.4 bnl3.18 npi212B umc39A ufg21
1.2 bnl5.33
2.7 bnl1.297
cdo1160C
2.4
1.9 umc15B umc17 umc175 umc103B 0.6 umc16
1.8 npi91A npi257 ATPase1 bcd828A bnl17.27 npi201A 1.1 bcd1127A 2.4 uaz18C
cdo118 cdo345B npi432 1.1 rz527A
9.4
mdh3 1.6 dpg1 1.8 php10080
4.4
a1 sh2 umc63 bnl1.67 bnl1.123 bnl12.30B
7.2
umc96 1.1 bcd134B;1.1 cdo962B;2.0 php20726;2.2 dup214;2.5 npi425A;3.5 cdo455B;3.7 dup216;4.6 npi420
4.1 ;5.2 uaz110;5.8 uiu(pog1A);11.1 rz527A;11.5 csu36
bnl7.26 1.3 uaz39
7.4
umc2A 10.4 csu25 11.2 uaz117A 13.0 uaz114


Chromosome 4
bnl(tas1E)
6.1
agr(r115)
10.1
npi294J umc123
3.9
1.1 agr(c94)
2.0 mpik5A mpik6 1.9 mpik5B
4.1 mpik7
mpik8
6.2
1.2 zpl1A dnap3,uaz50,52A,55,57A;1.1 uaz51,54;2.3 uaz14B,26B,44B,45B,46A,47B,48A,49B,D;2.4 uaz43D;3.8 uaz43B,53A
3.6 php20713 php20725 zpl1E ;4.9 uaz58A;6.5 uaz41D,42B;10.5 uaz103A
1.3 npi604A 1.3 uaz30A,63A;2.1 uaz65B;2.4 uaz41C;2.9 uaz66A,68B,69A;2.9 uaz67;3.5 zpl1B,C;4.5 uaz64B;4.9 zpl1D
1.3 zpl1F ;6.7 uaz38B;8.2 uaz103B;16.1 uaz129
bnl17.13B 1.4 uaz17B
8.0
1.8 umc31A umc87A umc55B
adh2
6.3
bnl5.46
4.6
npi386 uaz63B uaz62B 2.9 uaz30B 3.9 uaz57B 5.6 uaz53B
8.6
1.3 aco1 bnl17.13C npi574 2.5 uaz49C;2.8 uaz61B;3.0 bnl17.10;3.8 uaz41B,42A,44A,45A,46C,49A,zpl2A;4.0 uaz43A
1.3 gpc1 ;4.4 uaz69B;5.3 uaz42C,48C;5.7 zpl3A;7.1 uaz56;8.5 csu19;10.8 csu12B;11.2 uaz48B;14.9 uaz46B
1.3 npi95A
1.3 bl2 umc42A umc47 bnl17.09 wsu(nia2) 5.4 csu100
1.3 bet2 bnl15.45 dnap4 bnl12.06B,bnl15.27,npi367B,umc23B;2.2 ncr(nrB);2.3 uaz72;2.4 mpik11D,F,ucsd61J
3.1 uaz71A ;3.4 mpik12E,umc(orp1);4.8 csu81
1.4 bnl(tas3A)
1.9 dup(als1)
3.3 npi284
0.6 npi267 npi594B npi259A
1.2 bnl7.20 1.1 bnl17.23(pal3) ucsd64F 5.1 mpik11E
3.9 uaz73
7.3
umc156A umc126B 0.6 umc14 1.1 uaz47A 1.2 npi340B, npi396 2.3 ucsd64G 4.9 ucsd61I 5.1 ucsd78C
5.9
npi584 2.3 mpik3 4.1 bnl8.08H
8.2
trg1
8.2
umc19 bnl8.08E umc104A 0.1 uaz66B 2.7 umc66
2.7 bnl5.67
2.3 uaz74
4.9
wsu(nia3)
6.6
npi292
5.6
npi253B
4.2
0.7 bnl10.05 npi208A
bnl7.65
5.0
1.8 umc158 5.4 uaz122
1.8 npi300C npi570
1.9 npi270 15.5 wsu(nia1)
3.8 bnl17.05
0.6 c2
0.6 npi410 npi444 umc15A uox(ssu1A) 1.4 csu166A
3.3 umc52 1.7 pge14.1 6.7 uaz33B
5.1
1.2 ris2 2.5 uaz115
cuny9 1.2 uaz41A
5.4
mgs2A
2.7 npi203 uaz65A 1.4 uaz123B 2.0 uaz118
1.3 npi333 pge14 pge15 2.7 npi294G
2.7 npi593 2.9 uwo3
6.1
2.0 ncr(cat3) ncr(b70B)
2.7 bnl8.23 1.3 umc111
1.9 bnl15.07 0.6 npi451
csh(cdc2A)
4.8
bnl(tas1O)


Chromosome 5
bnl(tas1N) 1.4 bnl(tas2G) 3.4 bnl(tas2B)
12.4
bnl8.33 8.6 uaz75
5.6
csh(chi3) 8.4 csu149
7.7
uaz76A 2.7 csu33
5.2
npi409
3.4
bnl6.25
3.7
bnl8.29B 10.2 ucsd44B
4.9
npi75A npi579A 1.3 bnl7.24B
6.3
1.5 bnl17.18A npi581B 2.9 hcf108 4.8 umc147A
1.3 npi305A
2.9 npi282A cuny7 1.1 ucsd64A 1.1 ucsd104A
2.7 umc90
2.6 umc72A
2.9 phyA2
pgm2 2.3 umc106B 2.3 umc107B 9.9 csu10
9.4
csic(mah9) pge(B5)
3.8
1.8 tbp2
2.1 umc27A
niu2::Bs1 uaz25D uaz25E 2.3 csu150B 3.0 bnl7.56
3.6
rny(pcr2) dnap2 npi434
1.8
2.8 bnl5.02 mdh5 1.1 mpik33E 1.4 bnl5.27 1.4 umc166
1.3 bnl6.10
2.4 umc43 3.6 uaz77 5.1 csu168A
npi256
4.7
umc1 npi275 1.3 bnl6.22 2.3 wusl1005 5.1 uaz111 8.7 ncr(cat1)
3.6
bnl7.43 bnl10.06 1.3 bnl1.380 1.4 uaz132 2.4 ncr(b70A) 7.5 npi213
2.8
bnl4.36 5.9 dup(als2)
3.7
a2 1.4 uaz70A
3.4
bt1 npi(pmr15) 1.3 npi408 1.3 npi571 2.0 ris1
4.2
bnl7.71 1.6 npi53B 2.3 npi104 2.7 npi424 8.8 uaz131
7.0
koln2.1 1.4 npi449 6.3 amp3
8.5
1.5 npi295
bnl5.71
4.8
bnl10.12
4.4
npi237 2.4 bnl17.23(pal)
3.8
uaz78
2.4 umc54 umc156B umc126A 0.8 bnl5.40 3.7 npi562
4.2
umc51 2.4 ucsd72E
1.8 npi458A 9.9 csu26
6.4
php20566
12.3
bnl9.07B npi442 umc108
12.3
mpik10 umc68 4.1 npi253C 8.4 wsu(nia5)
13.3
bnl5.24
7.7
npi288A 1.4 uaz71B 6.2 got2
14.9
umc104B 4.4 php10017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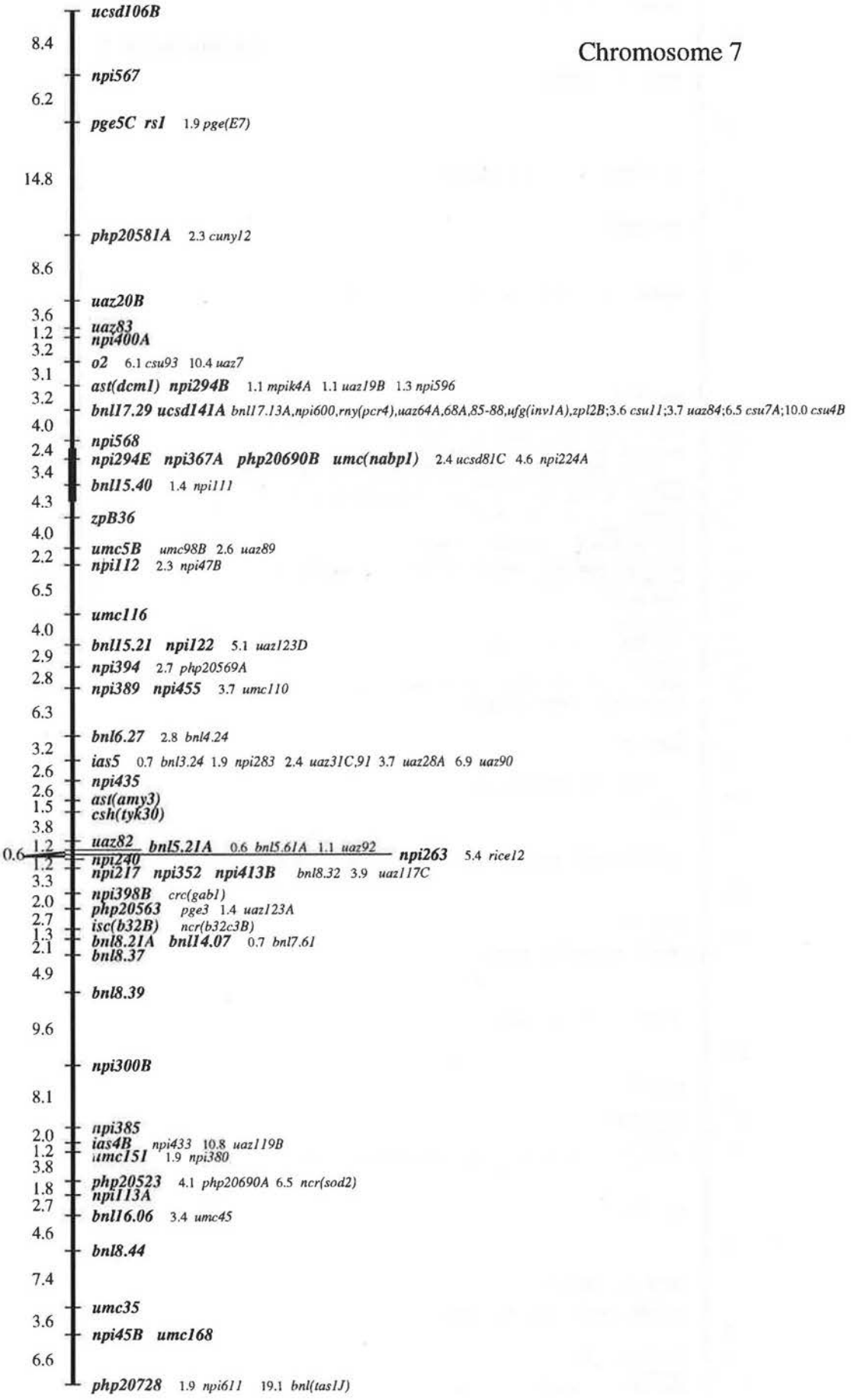
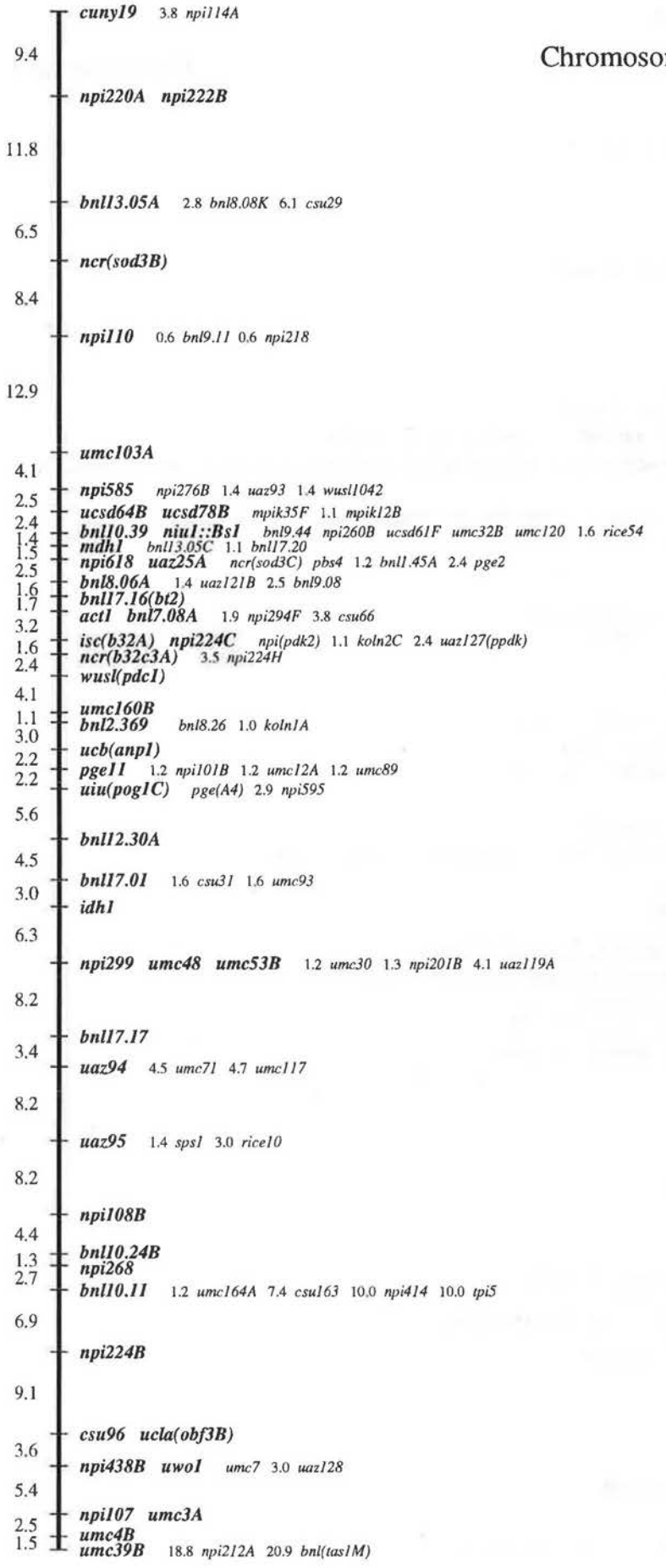
4.5
nor umc85 mpik(DH7) mpik11B uiu(pog1B) 2.3 bnl17.28 3.7 uaz102 8.1 csu71A 8.4 csu150A
2.4
bnl6.29 5.7 uaz80
0.6
npi235
4.5
bnl7.28
7.6
npi594A npi606 zp15 1.1 uaz23A
5.6
enp1 csu146 1.1 mpik33D 4.1 pgd1
1.8
npi373 umc59 1.1 mpik1 1.3 npi377 4.6 php06007
2.7
mpik18
2.9
php20045
7.1
npi393
11.3
npi98B 2.3 npi294C 8.6 uaz106A
5.6
umc65
2.5
npi223
5.4
pl1
0.6
umc113B
12.7
dzs23
2.8
hex2 0.6 npi617 1.3 npi253D 14.4 npi224I
1.1
ucsd64E ucsd78A 1.1 umc21 1.2 npi265
2.6
bnl3.03
4.9
rny1 ncr(sod3A) 4.9 uaz121A
1.2
ynh(me2) 1.3 npi560
2.1
bnl15.37
2.1
npi616 0.6 npi252
1.2
umc46
1.8
pdk1
2.7
bnl17.26 2.9 umc137
2.7
bnl5.47 npi63B npi608
3.5
bnl8.06B
2.2
bnl17.22
4.6
umc38
3.8
bnl8.08C
2.8
umc160A
11.5
npi280 bnl8.08J koln1B
3.4
uaz19D
4.1
uaz43E
5.2
bnl17.12 2.7 umc62 4.4 uaz81 4.5 npi419
4.9
umc132
6.5
idh2 npi597A
2.4
mdh2 umc133 1.4 npi561 2.9 uaz123C 4.5 ufg(agp1) 4.6 umc28
3.2
php20599

Chromosome 7




cuny19 3.8 npi114A
9.4
Chromosome 8
npi220A npi222B
11.8
bnl13.05A 2.8 bnl8.08K 6.1 csu29
6.5
ncr(sod3B)
8.4
npi110 0.6 bnl9.11 0.6 npi218
12.9
umc103A
4.1
npi585 npi276B 1.4 uaz93 1.4 wusl1042
2.5
ucsd64B ucsd78B mpik35F 1.1 mpik12B
2.4
bnl10.39 niu1::Bs1 bnl9.44 npi260B ucsd61F umc32B umc120 1.6 rice54
1.4
mdh1 bnl13.05C 1.1 bnl17.20
1.5
npi618 uaz25A ncr(sod3C) pbs4 1.2 bnl1.45A 2.4 pge2
2.5
bnl8.06A 1.4 uaz121B 2.5 bnl9.08
1.6
bnl17.16(bt2)
1.7
act1 bnl7.08A 1.9 npi294F 3.8 csu66
3.2
isc(b32A) npi224C npi(pdk2) 1.1 koln2C 2.4 uaz127(ppdk)
1.6
ncr(b32c3A) 3.5 npi224H
2.4
wusl(pdc1)
4.1
umc160B
1.1
bnl2.369 bnl8.26 1.0 koln1A
3.0
ucb(anp1)
2.2
pge11 1.2 npi101B 1.2 umc12A 1.2 umc89
2.2
uiu(pog1C) pge(A4) 2.9 npi595
5.6
bnl12.30A
4.5
bnl17.01 1.6 csu31 1.6 umc93
3.0
idh1
6.3
npi299 umc48 umc53B 1.2 umc30 1.3 npi201B 4.1 uaz119A
8.2
bnl17.17
3.4
uaz94 4.5 umc71 4.7 umc117
8.2
uaz95 1.4 sps1 3.0 rice10
8.2
npi108B
4.4
bnl10.24B
1.3
npi268
2.7
bnl10.11 1.2 umc164A 7.4 csu163 10.0 npi414 10.0 tpi5
6.9
npi224B
9.1
csu96 ucla(obf3B)
3.6
npi438B uwo1 umc7 3.0 uaz128
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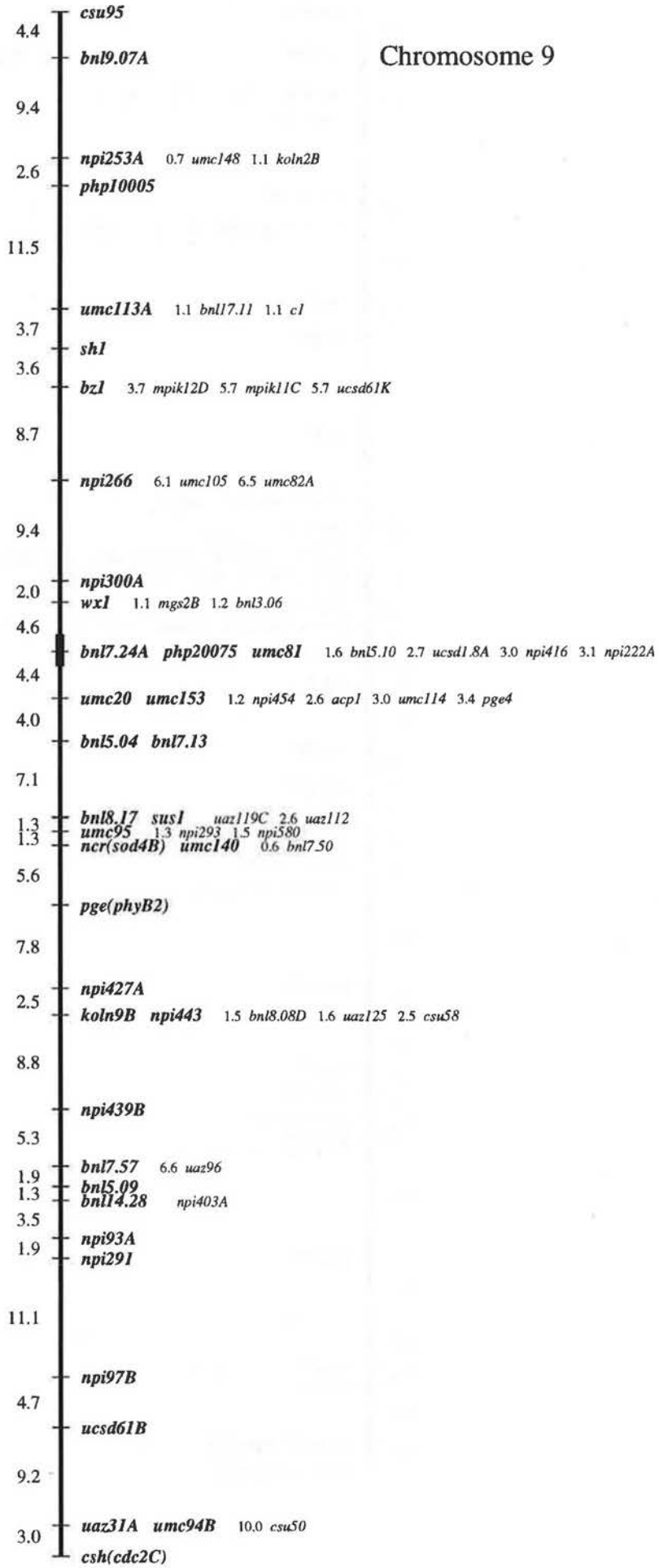
npi107 umc3A
2.5
umc4B
1.5
umc39B 18.8 npi212A 20.9 bnl(tas1M)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csu95
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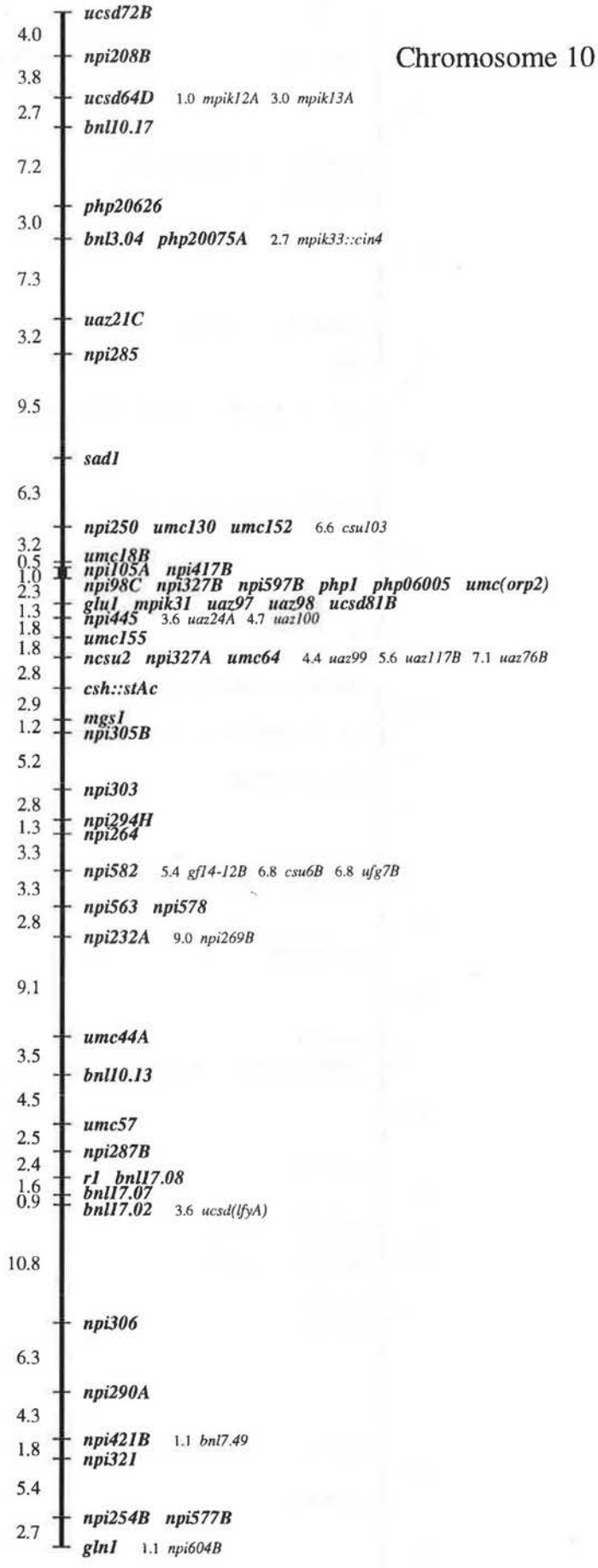
bnl9.07A
9.4
npi253A 0.7 umc148 1.1 koln2B
2.6
php10005
11.5
umc113A 1.1 bnl17.11 1.1 c1
3.7
sh1
3.6
bz1 3.7 mpik12D 5.7 mpik11C 5.7 ucsd61K
8.7
npi266 6.1 umc105 6.5 umc82A
9.4
npi300A
2.0
wx1 1.1 mgs2B 1.2 bnl3.06
4.6
bnl7.24A php20075 umc81 1.6 bnl5.10 2.7 ucsd1.8A 3.0 npi416 3.1 npi222A
4.4
umc20 umc153 1.2 npi454 2.6 acp1 3.0 umc114 3.4 pge4
4.0
bnl5.04 bnl7.13
7.1
bnl8.17 sus1 uaz119C 2.6 uaz112
1.3
umc95 1.3 npi293 1.5 npi580
1.3
ncr(sod4B) umc140 0.6 bnl7.50
5.6
pge(phyB2)
7.8
npi427A
2.5
koln9B npi443 1.5 bnl8.08D 1.6 uaz125 2.5 csu58
8.8
npi439B
5.3
bnl7.57 6.6 uaz96
1.9
bnl5.09
1.3
bnl14.28 npi403A
3.5
npi93A
1.9
npi291
11.1
npi97B
4.7
ucsd61B
9.2
uaz31A umc94B 10.0 csu50
3.0
csh(cdc2C)


Chromosome 10
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4.0
npi208B
3.8
ucsd64D 1.0 mpik12A 3.0 mpik13A
2.7
bnl10.17
7.2
php20626
3.0
bnl3.04 php20075A 2.7 mpik33::cin4
7.3
uaz21C
3.2
npi285
9.5
sad1
6.3
npi250 umc130 umc152 6.6 csu103
3.2
0.5
umc18B
1.0
npi105A npi417B
2.3
npi98C npi327B npi597B php1 php06005 umc(orp2)
1.3
glu1 mpik31 uaz97 uaz98 ucsd81B
1.8
npi445 3.6 uaz24A 4.7 uaz100
1.8
umc155
2.8
ncsu2 npi327A umc64 4.4 uaz99 5.6 uaz117B 7.1 uaz76B
2.9
csh::stAc
1.2
mgs1
npi305B
5.2
npi303
2.8
1.3
npi294H
npi264
3.3
npi582 5.4 gf14-12B 6.8 csu6B 6.8 ufg7B
3.3
npi563 npi578
2.8
npi232A 9.0 npi269B
9.1
umc44A
3.5
bnl10.13
4.5
umc57
2.5
npi287B
2.4
r1 bnl17.08
1.6
bnl17.07
0.9
bnl17.02 3.6 ucsd(lfyA)
10.8
npi306
6.3
npi290A
4.3
npi421B 1.1 bnl7.49
1.8
npi321
5.4
npi254B npi577B
2.7
gln1 1.1 npi604B

GENETIC MAP OF THE *ZEA MAYS* PLASTID CHROMOSOME

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The complete sequence of one maize plastid gene was reported in the past year. Its approximate location on the chromosome is shown on the map below, and its gene product is briefly described in the following table.

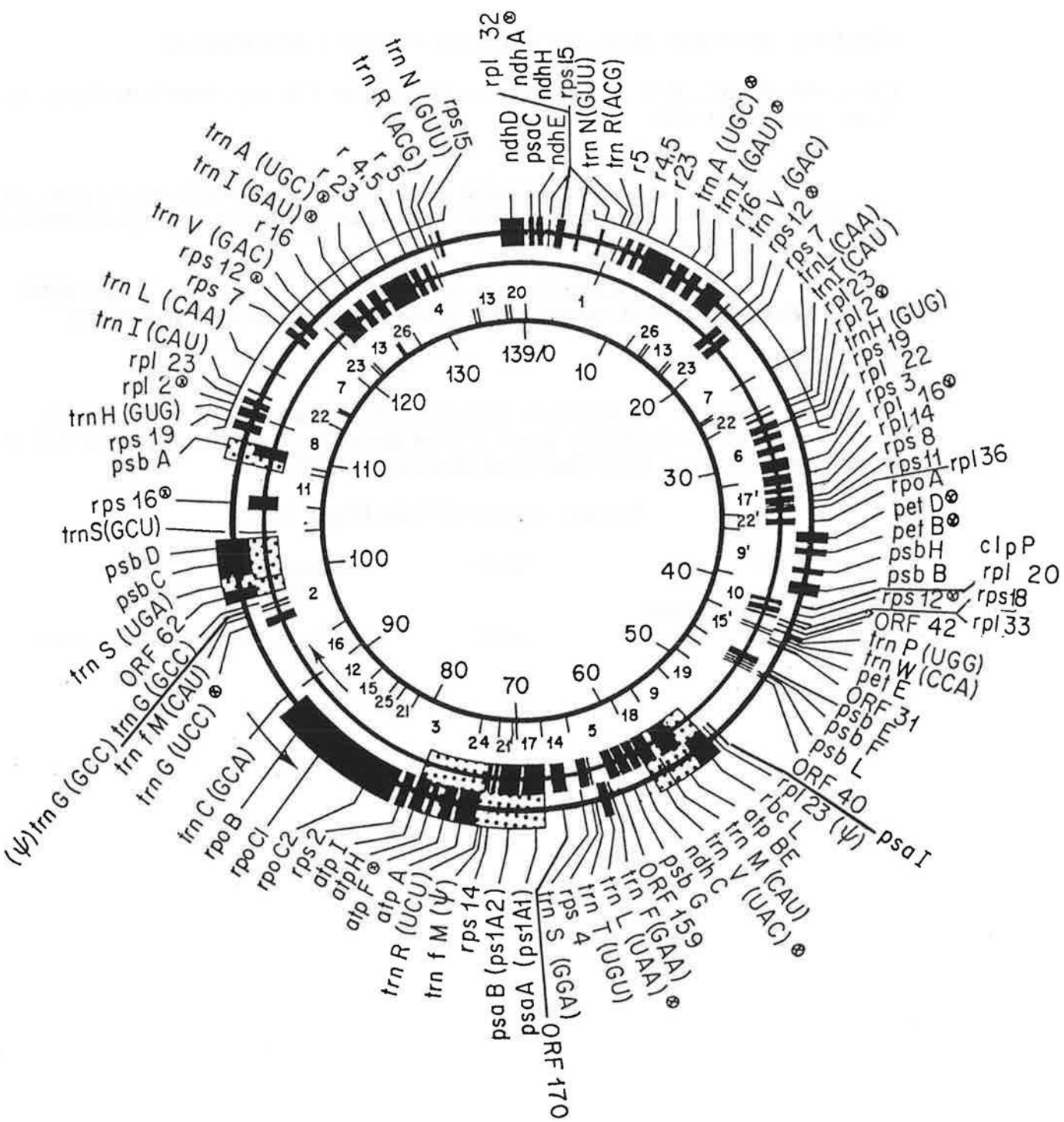
See the 1987-1993 News Letters for descriptions of other sequenced genes: **MNL 62**:148; **MNL 63**:155; **MNL 64**:164; **MNL 65**:164; **MNL 66**:160; and **MNL 67**:167.

References:

Weglöhner, Wolfgang, and Alap R. Subramanian. 1993. Nucleotide sequence of maize chloroplast *rpl32*: completing the apparent set of plastid ribosomal protein genes and their tentative operon organization. **Plant Mol. Biol. 21**: 543-548.

Recently-Reported Maize Plastid Genes

Gene Product	Gene	Reference
70S Ribosomal Proteins:		
L32	*rpl*32	Weglöhner and Subramanian, 1993


rpl 32
ndhA ⊛
ndhH
rps 15
ndhD
psaC
ndhE
trn N(GUU)
trn R(ACG)
r5
r4.5
r23
trn A (UGC) ⊛
trn I (GAU) ⊛
r16
trn V (GAC)
rps 12 ⊛
rps 7
trnL (CAA)
trn I (CAU)
rpl 23 ⊛
rpl 2 ⊛
trnH (GUG)
rps 19
rpl 22
rps 3
rpl 16 ⊛
rpl 14
rps 8
rps 11
rpl 36
rpo A
pet D ⊛
pet B ⊛
psbH
clpP
psb B
rpl 20
rps 12 ⊛
rps 18
ORF 42
rpl 33
trn P (UGG)
trn W (CCA)
pet E
ORF 31
psb N
psb T
psb L
ORF 40
rpl 23 (ψ)
psa I
rbc L
atp BE
trn M (CAU)
trn V (UAC) ⊛
psb G
ndh C
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)
trn G (UCC) ⊛
trn f M (CAU)
trn G (GCC)
(ψ)trn G (GCC)
ORF 62
trn S (UGA)
psb C
psb D
trnS(GCU)
rps 16 ⊛
psb A
rps 19
trnH (GUG)
rpl 2 ⊛
rpl 23
trn I (CAU)
trn L (CAA)
rps 7
rps 12 ⊛
trn V (GAC)
r16
trn I (GAU) ⊛
trn A (UGC) ⊛
r23
r5
trn R (ACG)
trn N (GUU)
rps 15
1
10
20
30
40
50
60
70
80
90
100
110
120
130
139/0

PHYSICAL MAPS (MASTER CHROMOSOMES) OF THREE MAIZE MITOCHONDRIAL GENOMES (Zea Mays ssp mays)

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We have identified a new type of normal maize mitochondrial genome named NA (N in A188 nuclear background) which brings the number of identified maize mitochondrial genomes to five, of which three are the cytoplasmic male sterile (cms) types: T, S and C, and of which two are the male fertile (normal) types: NA (as define above) and NB (the previously characterized genome in a B37 nuclear background).

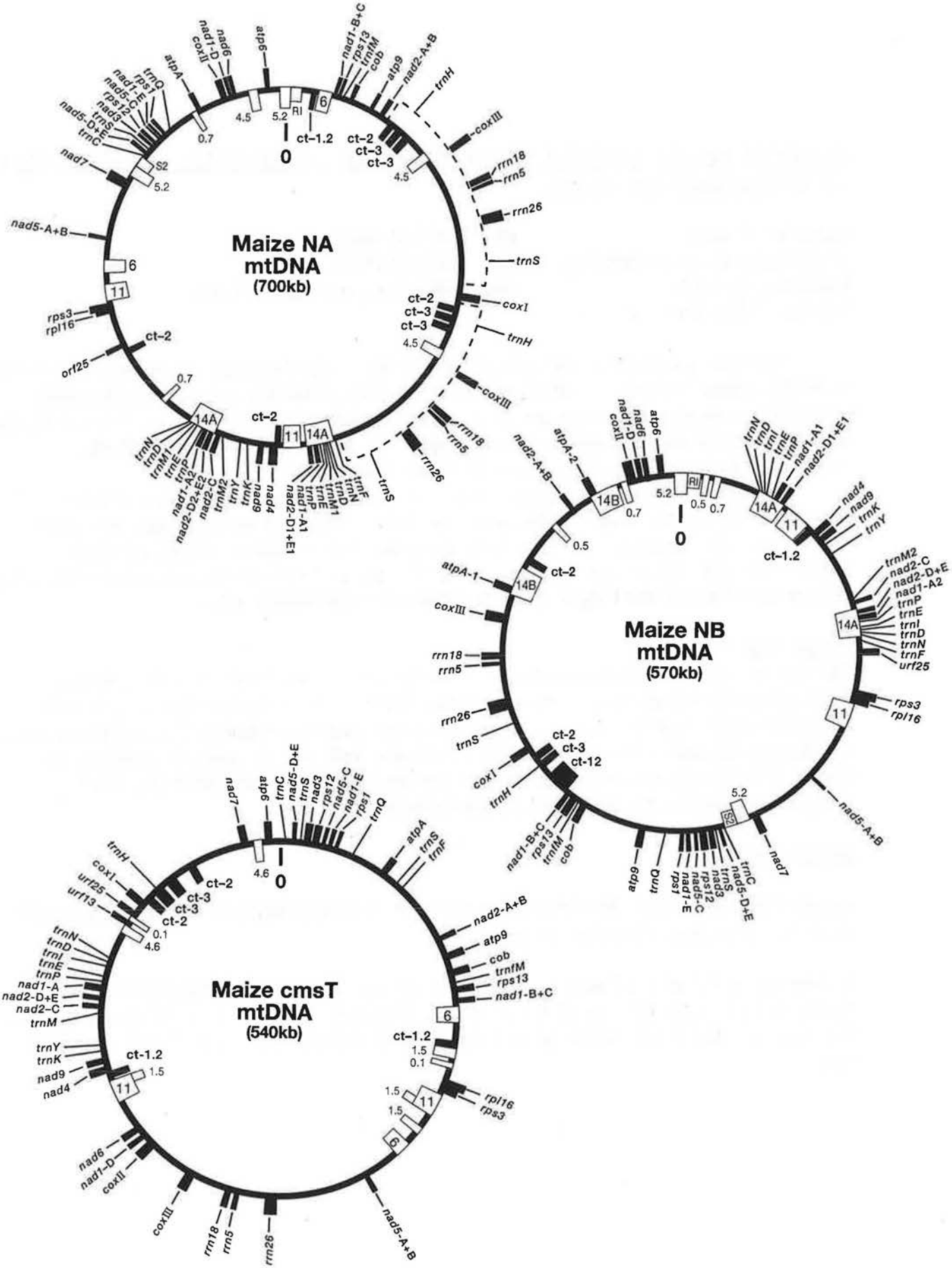
We have constructed the physical maps for NA, NB and cmsT. The restriction enzymes mapping data with BamHI, XhoI and SmaI for each of the three genomes can be find in the maize database . Details about the genome organization, the hypothetical multipartite structure and the identification of the various genes can be found in the following publications: Fauron and Casper 1994, Wolstenholme and Fauron 1994 .

Figure legend:
The size of each master chromosome is shown in parenthesis. All the known genes are indicated on the outside of each master circles. The repeats are represented by open boxes inside the circles, and the size of each (in kb) is indicated by a number. The integrated forms of plasmids R1 and S2 in maps NA and NB are also indicated by open boxes inside the circles. The black boxes inside the circles show the location of chloroplast (ct) DNA integrated sequences, the size of which are indicated in kb.

References:

Fauron CMR and Casper M (1994) A second type of normal maize mitochondrial genome: an evolutionary link. Genetics. *In press*

Wolstenholme DRW and Fauron CMR (1994) Mitochondrial genome organization. In: Advances in Cellular Biology of Plants. Vol. 2: Molecular Biology of the Mitochondria. Eds. C.S. Levings III and I.K. Vasil. Kluwer Academic Publishers. Dordrecht, The Netherlands. *In press*


Maize NA
mtDNA
(700kb)
Maize NB
mtDNA
(570kb)
Maize cmsT
mtDNA
(540kb)

IX. MaizeDB: MAIZE GENOME DATABASE

CONTEXT: The USDA Plant Genome Initiative includes 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 research grants through the Competitive Grants Program , the Initiative supports database development through the Agricultural Research Service. The Plant Genome Database was derived by "Prototype Developers" working first on maize, soybean, wheat, forest trees, and *Arabidopsis*, and is now being followed with other species. The structure is inclusive of higher plant data and is focused at the National Agricultural Library (NAL).

PRODUCTS AND PROSELYTIZING: The Gene List, Stock List, and Zealand 94 this year were derived by output from MaizeDB. Demonstrations of MaizeDB have been given at recent Maize Genetics Conferences, Plant Genome I and II, Corn Breeders School, North Central Corn Breeding Conf., Amer. Soc. Plant Phys., Arabidopsis Int. Conf., Int. Genetics Cong., and the Amer. Seed Trade Assoc. (COOPERATORS NOTE: the most enthusiastic interest in MaizeDB was at the Plant Phys. and the Arabidopsis meetings). Development and advancement has been greatly aided by comments and responses from the 1992 and 1993 demonstrations, and by experience with the implementation before and after the demos.

CONTENT: MaizeDB now includes much more information. Extensive descriptions of mutant genes have been provided to Gerry Neuffer by Cooperators, and these continue to be enhanced with new information; extensive stock information (especially inbred lines and their parentage); maps from new sources; latest map scores for the BNL RI populations and others; probe data; gene products; GenBank identifications; current literature references, and others. Requests to Cooperators for explicit mapping data are being made as needed. The database design for representing QTL data is approaching a testing phase. Phenotype and trait information is progressing toward a structured, systematic form. Rate of progress, and the breadth and depth of the database, will depend on your encouragement and on the continuity and extent of resources for the work in the future.

WHAT CAN I DO? Systematically presented data, including consistent use of terms and symbols in consistent, partitioned form, are particularly valuable for database entry. As you are describing a new variant, for example, try to identify the stage(s), body part(s), condition(s), expression, and characteristics as fully and precisely as possible. If a clone is derived and is used in mapping or in other work, assign a unique, unambiguous name to the clone and maintain that name (when needed, cite synonyms that have been used in other published work with it). Avoid terming two objects (mutants, genes, probes, clones) the same if they may turn out to be different. We will be happy to provide to you a template for systematic representation of probes and the parameters needed for molecular-marker mapping, for your use in your lab or to accompany data to be incorporated in MaizeDB.

WHERE DO I GET IT? MaizeDB can be accessed as described in the following two pages. These are simple and convenient means by which to look up and extract information. The MaizeDB Group asks for your help and input. We especially ask for your corrections, suggestions and ideas as they arise from using the database.

The MaizeDB Working Group is Ed Coe, Mary Berlyn, Pat Byrne, Georgia Davis, Denis Hancock, Stan Letovsky, Mary Polacco, and Marty Sachs.

Ye Nomenclature Standards are for all Cooperators, including thyself. Ere thee name a gene, consider ye Criteria, refer to ye Standards, and consult MaizeDB. Else.

THE MAIZE GENOME DATABASE IS AVAILABLE ON-LINE

MaizeDB, the full-capability relational Sybase database for maize genome data, is now available for on-line access. MaizeDB is part of the Plant Genome Database, which is under development at the National Agricultural Library and which has the goal of encompassing information on all plant species in a single database.

MaizeDB contains a broad array of information on maize genetics and breeding, integrated in a relational structure using Sybase software. Current categories of information include loci, alleles and other variations, gene products, stocks, maps, probes, phenotypic traits, references, and people. In the next few months we expect to add information on pests and stresses and results of quantitative trait locus studies.

Certain data you may find interesting to explore:

- Updated BNL and UMC maps.
- Current lit, attached as we identify them.
- Genbank numbers (attached to allele, locus, probe)
- Addresses of persons for clones/probes, mail, phone, etc. (We make every effort to keep these addresses current).
- Southern blot (gel) pattern, data
- Hotlinks to GenBank, SwissPROT

All interested users may access MaizeDB without charge. Our sole condition is that you inform us if you see something in either the content or format of the database that needs correction or improvement. You may leave notes directly in the Sybase version of MaizeDB (under the "/" at top left corner of database forms; see also Sybase Tutorial) or contact us as indicated on the next page.

We know there will be some things that appear chaotic or cryptic to the "naive" user, that we have lost the capacity to recognize because we have forgotten the problems or have explained them away to each other.

We continually upgrade "helps", and will incorporate suggestions from you at any time, but especially need suggestions now, in the early phases of accessibility. Is the first screen you see sufficiently clear? The second? Are the "helps" intuitively comfortable? Do you find information where you expect to?

As a "guest", you may search three data formats: gopher flat files, World Wide Web (WWW) hypertext flatfiles, or the Sybase database, MaizeDB. For guest logins, USER NAME is guest, PASSWORD is corncob. You may mail yourself data retrieved by Gopher or Sybase searches.

DATA AVAILABLE TO GOPHER SEARCHING

The MaizeDB Gopher server is updated regularly from the underlying Sybase database and allows rapid, user-friendly full text searches of most portions of the data. The Gopher access lacks the robust query and browsing capabilities of the full-featured database, but is sufficient for many purposes. If one searches for "ht1" under the Loci category, for example, Gopher will respond with a list of "documents" containing that character string; those documents can then be viewed one by one. Guidelines on formulating searches in Gopher are contained under the menu option "Gopher Information". Other menu choices include links to other Plant Genome gophers and to other biology gophers.

GETTING CONNECTED:

You may access the data using a MacIntosh, a PC or a Unix computer but you will need to be connected to the Internet. Modem connections will work, but do not support an X-windows (this is not the same as Microsoft Windows) display of the Sybase database. A major advantage of X-Windows is that you will be able to use a mouse to navigate. In the absence of X-Windows, keyboard commands may be used. The keyboard commands are relatively simple and may be obtained when you first login.

GOPHER

You may access our gopher client by any of the following:

(1) install your own gopher (client) software

(2) log on teosinte.agron.missouri.edu as described below.

(3) connect to some other gopher client.

CAUTION: If you use gopher software installed on a system with an IBM 3270 emulation (which many university mainframe systems use), the Gopher "Guest Access" option to the relational database may not function correctly.

OBTAINING GOPHER SOFTWARE

You will need the gopher client software. Software for Unix, MacIntosh, and PC computers is available without cost by anonymous ftp (file transfer protocol) from boombox.micro.umn.edu The software and help files are in the /pub/gopher directory.

For anonymous ftp to any machine supporting it, the user name is "anonymous" and the password is your email address. Downloading the software and installation is easiest if you have access to a campus computing services desk, or a systems administrator or can get help from a friendly hacker.

The gopher client may be modified so that it automatically calls up the MaizeDB gopher at teosinte.agron.missouri.edu or 128.206.11.1. Otherwise, you will probably connect to the University of Minnesota gopher; from there you may access the MaizeDB gopher from the listing of gophers that are located in Missouri (USA).

WORLD WIDE WEB CONNECTIONS

If you have access to the internet, then obtaining a WWW browser will provide you with a pleasing interface to the Maize Genome Database, as well as other WWW servers. In addition, WWW browsers are able to process information from gopher and WAIS

servers.

WWW differs from gopher in that its files are hypertext, with links to related files in the same or different databases that can be traversed at the click of a mouse button. For example, if you have called up the MaizeDB record on adh1, you can click on the product name and go to a record with additional information. From that form, you can click on the SwissPROT accession number and obtain further information regarding alcohol dehydrogenase. From there you can traverse a link to the Medlars entry of the paper that describes adh1. In the course of browsing this information, you have accessed three different databases on two continents.

For Unix with X-windows, Macintosh, or MSDOS Windows, Mosaic is the browser of choice. This may be obtained by anonymous ftp to ftp.ncsa.uiuc.edu. Be sure to obtain the client appropriate to your computer type.

For computers not capable of X Windows or the Macintosh or MSDOS windowing systems, then lynx may obtained from ftp2.cc.ukans.edu. This is a simple WWW browser that is vt100-capable, so it will work over a modem connection.

DIRECT LOG IN: GOPHER AND SYBASE

(1) If using an X-window, you need to allow teosinte (the machine or remote host where MaizeDB resides) to open a window on your machine:

- If you are on a Unix computer, type "xhost + teosinte.agron.missouri.edu"

- On a MacIntosh or PC, configure your X-Windows software to permit remote connections. If you use xterm, this is usually set automatically.

(2) Establish a telnet connection to: teosinte.agron.missouri.edu There are several ways to do this, depending on your computer and software.

On Unix machines, type "telnet teosinte.agron.missouri.edu"

Most Macintosh or MSDOS software clients will have on-line help available, and the prompts will lead you through the login process.

(3) Login as 'guest' and use the password 'corncob' A list of options will appear as follows:

1- VT100 Version of MaizeDB
2- X-Windows Version of MaizeDB
3- MaizeDB Gopher (vt100)
4- Lynx WWW Browser (vt100)
5- Help for X-Windows
6- Help for Sybase
7- Mail Sybase Tutorial to Yourself
8- Exit
Enter choice:

Type in the number of your choice, then press return. The keyboard commands required for vt100 Sybase access may be viewed (choice #6) or emailed to you (choice #7).

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Faxed information and queries may be sent to (314) 874-4063.

If you are unable to use the email addresses as given, teosinte.agron.missouri.edu is also known as 128.206.11.1.

Phone contact for help: 314-882-1722 (Denis Hancock).

X. RECENT MAIZE PUBLICATIONS

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r7. Ahmadi, M; Wiebold, WJ; Beuerlein, JE, 1993. Grain yield and mineral composition of corn as influenced by endosperm type and nitrogen. Commun Soil Sci Plant Anal 24:2409-2426.

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XI. SYMBOL INDEX

("r" refers to numbered references in the Recent Maize Publications section)

XII. AUTHOR AND NAME INDEX

("r" refers to numbered references in the Recent Maize Publications section)
(* identifies articles authored in this Newsletter)

CLONE INFORMATION SHEET (PLEASE SUPPLY FOR EACH CLONE)

CLONE DESIGNATION: ISOLATING LAB/PERSON:

IS THIS A KNOWN SEQUENCE CLONE (circle one)? Yes No GENE SYMBOL:

WHAT PRODUCT OR FUNCTION?

PRODUCT ACRONYM: EC NO.:

CLONE TYPE (genomic, cDNA, etc.):

ISOLATED FROM WHAT ORGANISM:

REFERENCE:

RESTRICTION MAP/SEQUENCE INFORMATION (please specify GENBANK, EMBL, EST, SWISSPROT NOS. if possible):

SOUTHERN BLOT INFORMATION

LINE ANALYZED	ENZYME(S) TRIED	# BANDS SEEN	APPROX. MW

CHROMOSOME ARM, IF KNOWN:

NEAREST MARKERS, IF KNOWN:

IT IS OPTIMAL FOR US TO RECEIVE A STAB (ELSE 10µg OF DRIED PLASMID WOULD BE ACCCEPTABLE).

HOST OF SUPPLIED STAB CULTURE: AMT. OF PURIFIED PLASMID:

VECTOR: MEDIA:

ENZYME(S) TO CUT OUT INSERT: INSERT SIZE:

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SPECIAL CONDITIONS NEEDED FOR PCR:

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March, 1994

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