MAIZE GENETICS COOPERATION

NEWSLETTER

69

August 15, 1995

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Charles R. Burnham

A scholar to be emulated, Dr. Charles R. Burnham displayed an enthusiasm for learning until his death on April 19, 1995. The last weeks of his life were as filled with the excitement of genetics as his entire 70 year career. He was writing a manuscript addressing important questions concerning how chromosomes pair and adding to a book called Genetics Is For Everyone. The restoration of the American Chestnut - nearly annihilated by the blight disease - had been a high priority for him since his 1972 retirement from the University of Minnesota. He founded the nonprofit American Chestnut Foundation to develop a disease resistant American Chestnut by using a breeding method not previously employed. A research farm in Virginia is dedicated to this effort.

Dr. Burnham came to the University of Minnesota in 1938. He was still making scientific contributions in 1995, operating out of his nursing home room with the assistance of many friends at the University and elsewhere. He advised almost 50 graduate students over the years. Those students are around the world and have great appreciation for the depth of understanding Dr. Burnham passed along to them. Three of his former students are members of the U.S. National Academy of Sciences and one is a member of the Royal Society of Canada. His research focused mainly on the behavior of chromosome rearrangements that occur in nature or are induced by radiation or other agents. He made important contributions on chromosome pairing, genetics of cross compatibility, pigment patterns, ancestral traits, disease resistance, and gene mapping. He worked on many plant species but mostly corn and barley. The textbook he authored in 1962, Discussions in Cytogenetics, stands today as a major contribution in teaching how inheritance patterns reflect the behavior and transmission of chromosomes.

A pioneer in plant genetics, Dr. Burnham studied with giants in the field. His Ph.D. in 1929 was under R.A. Brink at the University of Wisconsin. He was a National Research Council Fellow with R.A. Emerson at Cornell University, E.M. East at the Bussey Institution at Harvard and with E.G. Anderson at the California Institute of Technology. He also studied with L.J. Stadler at the University of Missouri and D.F. Jones at Yale. At Cornell, Burnham was a member of a group which became known as the "pioneers in corn genetics", including R.A. Emerson, M.M. Rhoades, B. McClintock, and G.W. Beadle. Dr. Burnham's first job was at West Virginia University in plant breeding and cytogenetics. In 1938, he joined what is now the Department of Agronomy and Plant Genetics of the University of Minnesota as an Associate Professor. His awards included Fellow of the American Society of Agronomy, Distinguished Service Award of Sigma Xi, and the Gamma Sigma Delta Award of Merit.

Careers are often built "On the Shoulders of Giants". Dr. Burnham always greatly admired his mentors. In the same manner, his students, colleagues, and friends believe their lives have been enriched by their association with him. He believed in students and greatly enjoyed seeing them get excited about genetics. Encouraging students in high school who have an interest in genetics was one of his goals. He established the Burnham Scholarship for Research by Pre-College Students. Contributions may be made to this scholarship (payable to University of Minnesota, c/o R.L. Phillips) or to the American Chestnut Foundation (401 Brooks Hall, P.O. Box 6057, West Virginia University, Morgantown, WV 26506).

Ronald L. Phillips
Regents' Professor
Department of Agronomy and Plant Genetics
University of Minnesota
St. Paul, MN 55108

John R. Laughnan

Dr. John R. Laughnan was 74 years old at the time of his death on July 15th, 1994. His bachelor's degree in Plant Sciences was from the University of Wisconsin in 1942, and his Ph.D. in Genetics was from the University of Missouri in 1946, under L. J. Stadler. He served on the faculty at the University of Illinois most of his career, and for brief periods at Princeton University and the University of Missouri.

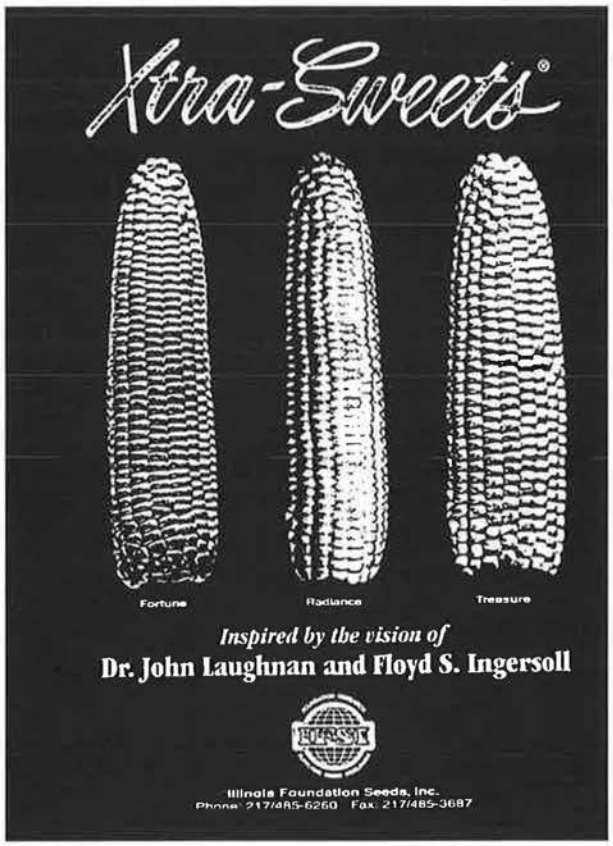
John taught introductory plant biology, genetics, and physiological genetics to thousands of graduate and undergraduate students and was advisor to, and served on the graduate committees of, many graduate students. As a teacher, he brought the pleasure of scientific insights, and the challenge of skillfully defining and attacking problems, to the learner. His former students are now geneticists who work on maize, *Drosophila*, soybean, mosquito, human, yeast, fish, and *Tetrahymena*; plant breeders; virologists; medical doctors; heads of seed companies and professional societies; lawyers; and deans and department heads at colleges and universities.

John touched the lives of many people. However, he undoubtedly touched most the lives of millions who never met him, the ones who enjoy "supersweet" sweet corn every summer. That discovery, in fact, goes back to a moment I remember when John commented to his then-graduate-student that he was looking into why *shrunken2* seeds were sweet. John's basic contributions to understanding of duplicated genes in corn and *Drosophila*, and to the genetics of cytoplasmic male sterility and its restoration, are monuments of skillful, complex, and creative experimental study. John in 1958 proposed initiation of regular informal meetings of scientists interested in corn genetics, from which the Maize Genetics Conference has grown into an international gathering of over 450 scientists each year.

Friends and colleagues may wish to join others in commemorating his legacy to scholarship, to teaching, and to stimulation of graduate and undergraduate students. To honor and remember John in a fashion that recognizes this legacy, a fund has been established to support travel to scientific meetings for graduate students in plant biology at the University of Illinois. The John R. Laughnan Fund in Plant Biology is in the form of an endowment whose earnings will be used to finance travel awards. The goal is $20,000, to ensure that the fund will be self-supporting.

In addition to my respect for John as a colleague, as his first doctoral student I am particularly conscious of all that I have personally been given and all that has been given by John to others. I encourage you to consider a gift to the John R. Laughnan Fund In Plant Biology, in remembrance of his legacy. Checks may be made out to UIF/John R. Laughnan Fund; address to University of Illinois Foundation, P.O. Box 3429, Champaign, IL 61826-9916.

E. H. Coe
Geneticist, ARS-USDA and Professor of Agronomy, Univ. of Missouri
Ph.D., Univ. of Illinois, 1954



I. FOREWORD
at last... (21 Sep 95)

We thank you for your patience. It is reassuring when, despite reason to be impatient, colleagues simply inquire about your summer's crop, or about the progress of your other activities, or whether their subscription has lapsed, and do not suggest lassitude. MNL is still an important item in libraries also, shown by followups asking about the status of their subscriptions (no doubt some library user has asked). Please accept my personal apology for the long delay in this issue, caused by a combination of illness of the key achiever of the work with an extraordinary volume and depth of material to be redacted and synthesized; we hope you will find the quality and quantity of the content here help to make up. PLEASE SEE ESPECIALLY THE FOLLOWING:

The MaizeDB section
The Probe Bank section
The new UMC Core Map and the new (60-year) synthesis of the Genetic Map, matched to it
The table of improved Core Markers
The table of cDNA identifications
The current RFLP Map from Brookhaven National Laboratory

The 'Cooperation' exists because you are a 'Cooperator' in keeping up the tradition of sharing maize genetics information with colleagues, here and in many unheralded conversations, correspondence, and shared stocks. Among my treasured paradigms are memories of how freely and enthusiastically their ideas and their stocks were shared, supplemented by helpful details, by our colleagues such as Barbara McClintock, Charles Burnham, John Laughnan, among many, and the theme continues. 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, 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. Cooperators emphasize presentation of brief technical notes, updates, mutants, segregation ratios, tables of mapping data, clones, biochemical functions, and the like. Comprehensive material and analyses are better directed to formal publication.

More and more cooperators supply notes, tables and figures in electronic form, and this greatly facilitates editing and compiling.

Gifts to the Endowment Fund for support of the Newsletter now total about $90,000. 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 are made possible only by sustained and ongoing encouragement of this work within the Agricultural Research Service. The MaizeDB project has advanced, through the efforts of Dr. Jerry Miksche, from a temporary to a regular, ongoing project; please note the section on MaizeDB, including access, content, and goals, provided by our new Curator, Dr. Mary Polacco. We urge you with our strongest enthusiasm to use, assess, and contribute to the database.

Mary Polacco ingeniously contrived and "dumped" the Gene List and reference links; Zealand 95 and reference links; the Stock List; and author and symbol indexes from MaizeDB, aided by the skillful savvy of Denis Hancock and Shirley Kowalewski. Help, advice and ideas also from my colleagues Mike McMullen, who reviewed and helped refine the whole, and Pat Byrne and Georgia Davis, who compiled, summarized, and evaluated contents, are warmly appreciated. Shirley Kowalewski, during recovery from a protracted illness, skillfully made the contents into fine form, twisted diverse electronic sources to suit and interpreted exotic scripts, structured the year's literature and indexes, and questioned quality or content, or gave creative advice, at key moments. Thanks are also given to Lou Butler for helping get the Newsletter copy edited and moving forward at critical stages. 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 70, 1996), 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 1996 Maize Genetics Conference at Pheasant Run, in St. Charles, Illinois, March 14-17, 1996, will be mailed to former attendees in November 1995; 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 1996 Maize Genetics Conference is:

Mary Alleman
Curt Hannah
Paul Sisco
Jeff Bennetzen
Tim Helentjaris
Sue Wessler
Paul Chomet (Chair)
Barbara Kloeckener
Udo Wienand

Editor Coe

ALBANY CALIFORNIA
USDA Plant Gene Expression Center

An SEM study of embryogenesis and seedling development in ABPHYL plants

--David Jackson and Sarah Hake

All grasses are characterized by a simple two-ranked or distichous phyllotaxy, where one leaf and corresponding lateral bud is initiated per node. However, there have been several reports of phyllotactic variants in maize (Greyson et al., Can. J. Bot. 56:1545-50, 1978, and references therein), some of which were heritable and were referred to as ABPHYL (for "aberrant phyllotaxy"). Plants from ABPHYL families showed a range of phyllotaxies including decussate, bijugate, spiral and distichous. We were recently provided with seed from families showing ABPHYL characteristics (a kind gift from M. Menzi, Swiss Federal Research Station). These families give rise to between 50 - 90% of plants having altered phyllotaxy, with the majority being decussate, i.e. having two leaves per node (and two ear shoots at ear nodes). These lines also give rise to a high proportion (up to 10%) of twin plants. The ABPHYL character behaves as a recessive and is penetrant in a number of inbred lines.

In order to understand how organogenesis is occurring in ABPHYL plants, we undertook an SEM study of embryo development in ABPHYL and normal sibs. The material was generated by using decussate ABPHYL plants (which had two ears at one node) and pollinating one ear with pollen from an inbred (B73) and the other with pollen from a decussate sib. Developing kernels were sampled at different times after pollination and the embryos were dissected out, fixed in 4% glutaraldehyde in PBS and dehydrated, critical point dried, coated and viewed in the SEM using standard techniques. The seedling apex samples were prepared using the replica technique developed by Williams and Green (Protoplasma 147:77-79, 1988).

In normal embryos, the shoot apical meristem is first visible as a dome-shaped bulge on the face of the scutellum, which becomes surrounded by the coleoptilar ring (Fig. 1A). The first true leaf primordium is then initiated opposite to the scutellum (Fig. 1B).

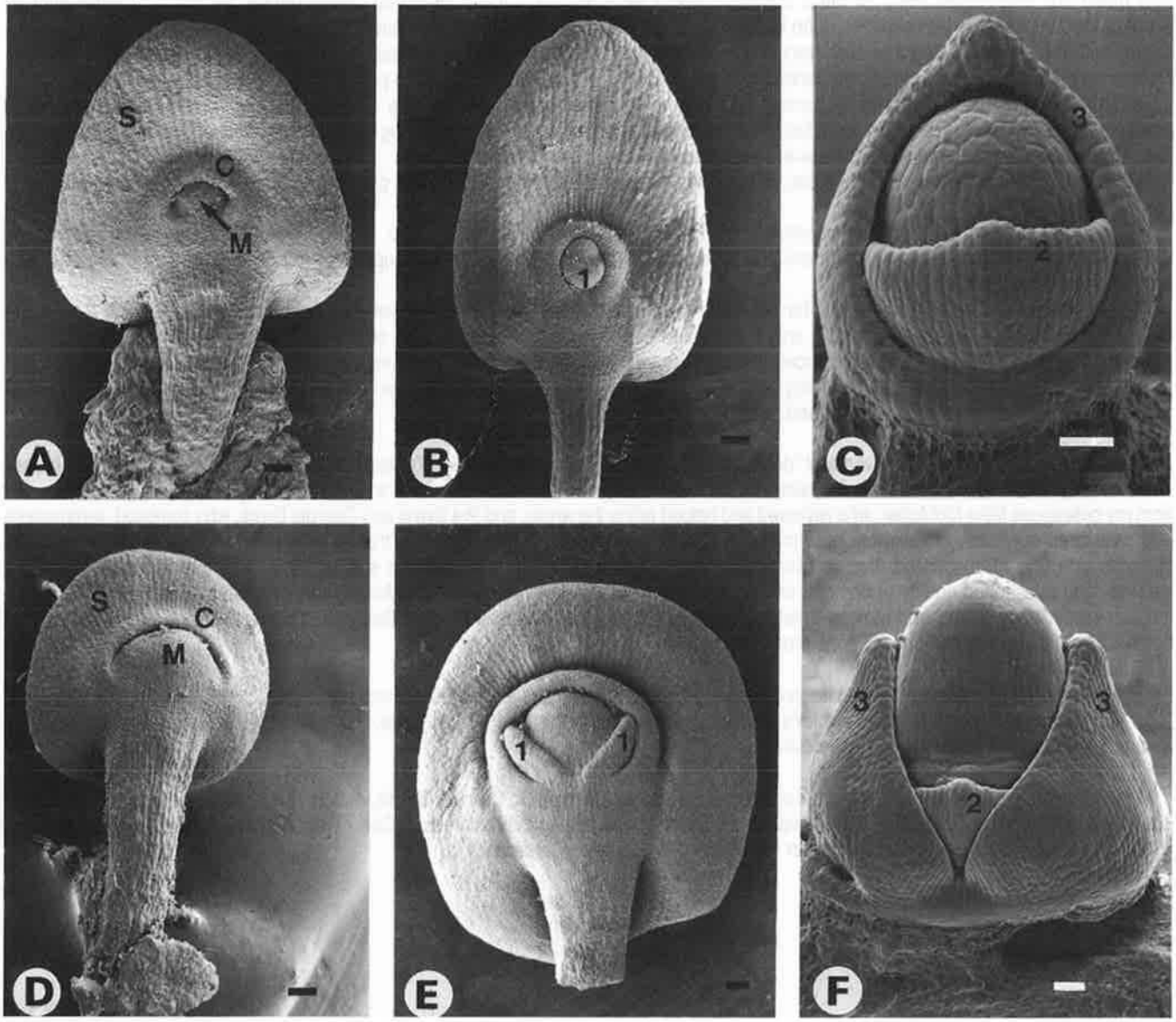


Fig. 1. SEM analysis of normal and ABPHYL embryos and seedling apices. A-C= normal, D-F=ABPHYL. A,D = coleoptilar stage, B,E = first leaf stage, C,F = 14 day seedling apex. S= scutellum, C= coleoptilar ring, M= shoot apical meristem, 1= first leaf (leaves), 2,3= plastochron 2, 3 leaves. Bar=50um.

This pattern is reiterated such that leaves are always initiated opposite to the previous leaf, as seen in the seedling apex after removal of most of the leaves (Fig. 1C).

In ABPHYL plants of the same age, the initiation of the major embryonic structures is roughly synchronous with normal sibs. The most striking aspect of ABPHYL is that the newly initiated shoot apical meristem, and the surrounding coleoptilar ring, is much larger than normal (Fig. 1D). The first true leaves are initiated as a pair perpendicular to the normal leaf axis (Fig. 1E). Of many (>100) embryos from the ABPHYL sib cross which we dissected, all showed very similar early embryo development, leading us to suggest that a consistent defect in ABPHYL is the ability to control the size of the initiating shoot meristem, and this problem leads to a certain proportion of plants having decussate phyllotaxy. In decussate ABPHYL seedling apices, the two leaves in a pair are highly synchronized (Fig. 1F).

We are continuing to characterize ABPHYL by using molecular markers for organogenetic events in the shoot apical meristem (Jackson et al., Development 120:405-413, 1994), and are also setting up a clonal analysis to determine how the increase in meristem size compensates for the doubling in leaf initiation events per plastochron.

AMES, IOWA
Iowa State University

A gene conditioning high oleic maize oil, *olc1*

--Allen Wright

Maize (*Zea mays* L.) oil with higher levels of oleic acid relative to linoleic acid would have greater stability. Thus high oleic acid oil is a desirable breeding objective. From EMS-treated B73 (paraffin oil pollen treatment) a mutation has been recovered that increases oleic acid concentration of the seed oil. This mutation (designated *Olc1*) has a partially dominant effect . The oleic acid concentrations of non-mutant B73, heterozygous *Olc1* and homozygous *Olc1* were approximately 27, 35 and 52%, respectively (Table 1). B-A translocations (Table 2) indicate that the *Olc1* gene is located on the long arm of chromosome 1.

Table 1. Chi square analysis of *olc1/Olc1* F2and *olc1/Olc1* x *olc1/olc1* segregations

olc1/Olc1 F2 Phenotype	Obs. (x)	Exp. (m)	$(x-m^2)/m$
Low oleic: *olc1/olc1* (Range: 24.8 - 30.1% oleic)	27	25	0.16
Intermediate oleic: *olc1/Olc1* (Range: 32.4 - 38.1% oleic)	52	50	0.08
High oleic: *Olc1/Olc1* (Range: 50.3 - 55.2% oleic)	21	25	0.64
Chi square=0.88 P=64.54%			
olc1/Olc1 X *olc1/olc1* Phenotype	Obs. (x)	Exp. (m)	$(x-m^2)/m$
Low oleic: *olc1/olc1* (Range: 25.5 - 30.1%)	36	35	0.0286
Intermediate oleic: *Olc1/olc1* (Range: 33.0 - 38.1%)	34	35	0.0286
Chi square=0.0572 P=81.27%			

Table 2. Maximum oleic acid content of B-A translocation crosses to mutant (*Olc1*) and non-mutant B73 (maximum value of at least 10 individual kernel determinations).

	oleic composition (%)	
B-A Stock	*Olc1*	Control
TB-1Sb	35.4	42.1
TB-1La	41.5	51.9
TB-2Sb	29.5	35.5
TB-2Lc	31.8	35.6
TB-3Sb	30.3	34.7
TB-3La	31.8	39.7
TB-4Sa	36.8	42.3
TB-4Lf	39.0	39.7
TB-5Sc	30.9	40.4
TB-5La	30.5	40.9
TB-6Sa	33.4	38.2
TB-6Lc	32.3	36.9
TB-7Sc	36.4	43.3
TB-7Lb	35.3	39.3
TB-8Lc	30.1	33.9
TB-9Sd	31.4	34.1
TB-9Lc	34.2	39.2
TB-10Sc	33.4	37.6
TB-10L20	30.0	35.5

Genetic mapping of two acetyl-CoA carboxylase genes

--James J. Caffrey, Eve S. Wurtele and Basil J. Nikolau

The biotin-containing enzyme acetyl-CoA carboxylase (ACCase) catalyses the formation of malonyl-CoA from acetyl-CoA. This reaction occurs in at least two spatially separate compartments. In the plastids, the reaction is the first committed step in *de novo* fatty-acid biosynthesis; in the cytosol, malonyl-CoA is required for the synthesis of a number of secondary metabolites, including flavonoids. In some non-Gramineae, two structurally distinct forms of the enzyme exist: a "multisubunit type", resembling the *E. coli* ACCase, which occurs in plastids, and a "multifunctional type" cytosolic enzyme, resembling yeast and mammalian ACCase (Sasaki et al., J. Biol. Chem. 268:25118, 1993; Alban et al., Biochem. J. 300:557, 1994). These isoforms of ACCase can be distinguished by the molecular masses of their biotin-containing subunits and by their susceptibility to the aryloxyphenoxypropionate and cyclohexanedione herbicides: the multisubunit ACCase is resistant to the herbicides, while the multifunctional ACCase is susceptible. In Gramineae such as maize, wheat, and rice, the multisubunit ACCase appears to be absent, which accounts for the susceptibility of these species to graminicides (Konishi and Sasaki, Proc. Nat'l. Acad. Sci. USA 91:3598, 1994).

In maize, two types of multifunctional ACCase can be resolved by ion-exchange chromatography, ACCase I and ACCase II. ACCase I occurs in the plastids of mesophyll cells, and is susceptible to inhibition by graminicides, while ACCase II is not in plastids and is resistant to these herbicides (Egli et al., Plant Physiol. 101:499, 1993). Furthermore, two very similar partial cDNAs coding for the multifunctional ACCase (pA3 and pA4) have been sequenced (Ashton et al., Plant Mol. Biol. 24:35, 1994). Based on the sequence of the 4.3 kb partial cDNA clone (pA3), PCR primers were designed to amplify the 873 bp fragment between positions 3035 and 3907 of the maize ACCase cDNA. The amplified DNA product was gel-purified and end-sequenced to confirm its identity.

The amplified 873 bp ACCase fragment was used as an RFLP probe to genetically map the two structural genes that code for maize ACCase. Two families of recombinant inbred plants derived from the crosses Tx303 x CO159 and T232 x CM37 (Burr et al., Genetics 118:519, 1988) were used. Southern blot analyses of *Eco*RI-digested DNA isolated from each recombinant plant re-

vealed a strongly hybridizing band that was polymorphic in both families. The segregation pattern of this polymorphic band enabled the mapping of one ACCase structural gene (*accA*) to near the centromere of chromosome 2, in the interval defined by the RFLP markers *umc131* and *uox* (*ssu1B*) (Figure 1). [Ed. note: *accA* and *accB* are considered temporary symbols pending clarification of the relationships of functions and mapsites]

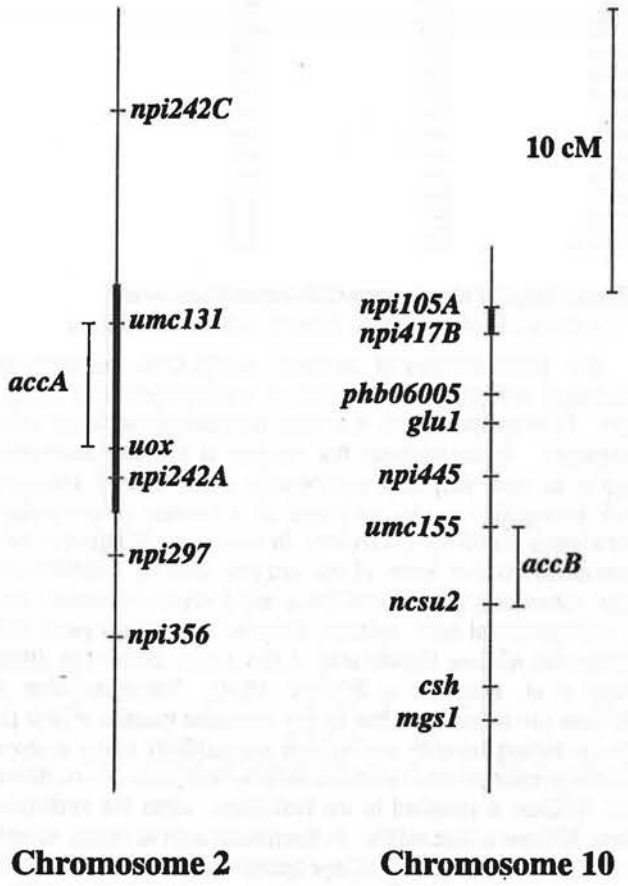


Figure 1. Genetic location of the two maize 240 kD ACCase genes on the RFLP map of the maize genome. The *accA* gene is located near the centromere on chromosome 2, in the interval defined by the RFLP loci, *umc131* and *uox*. The *accB* gene is located on the long arm of chromosome 10, in the interval defined by the RLFP loci, *umc155* and *ncsu2*. The thick bars on each map represent the genetic location of the centromeres of chromosomes 2 and 10.

In addition to the *accA* band, these blots revealed a second, less intensely hybridizing band that was polymorphic only in the T232 x CM37-derived family. The segregation pattern of this band enabled the mapping of the second ACCase structural gene (*accB*) to chromosome 10L, in the interval defined by the RFLP markers *ncsu2* and *umc155*. The position of the *accB* locus was confirmed by analyzing the segregation of a polymorphism revealed by *Eco*RV digestion of DNA isolated from the same individual recombinant plants.

The *accA* locus corresponds in position to the ACCase gene mapped by Egli et al. (MNL 68:92, 1994) to chromosome 2. A graminicide-resistance locus, termed *Acc1-S2*, has been previously mapped to 10L, 6.3 cM from *umc155* (van Dee et al., Agron. Abstracts, p.198, 1992). This resistance locus probably corresponds in position to the *accB* locus that we have mapped here. These data support the conclusion that such herbicide-tolerant mutations occur in the structural genes for ACCase. Furthermore, the data imply that the *accB* locus codes for the plastid-located, graminicide-sensitive ACCase I isoform, and the *accA* locus codes for the nonplastid-located, graminicide-insensitive ACCase II isoform.

Resistance to *Bipolaris maydis* is controlled by two genes

--Ru-Ying Chang and Peter A. Peterson

We reported unexpected results from our *rhm* tagging project, which indicated that there are two linked genes, designated *rhm1* and *rhm2*, involved in chlorotic-lesion resistance to *Bipolaris maydis* (Chang and Peterson, MNL 68:4-6, 1994). We arbitrarily set the dominant gene in the T line as *Rhm1* and that in the *Cy* line as *Rhm2*.

The two-gene model predicts that the inbred lines, which were shown (Smith and Hooker, Crop Sci. 13:330-331, 1973) to have a single, dominant gene for susceptibility to this disease have, in fact, two genes, with only one being dominant. The dominant gene in these inbred lines should recombine with one of the two dominant genes designated in this study, but not with both. A recombination test (Fig. 1) has been carried out to verify this prediction. Several inbred lines were selected from Smith and Hooker's study and crossed separately with our designated *Rhm1* and *Rhm2* lines and the F1's were testcrossed by the *rhm* tester. Summarized results from these tests are listed in Table 1.

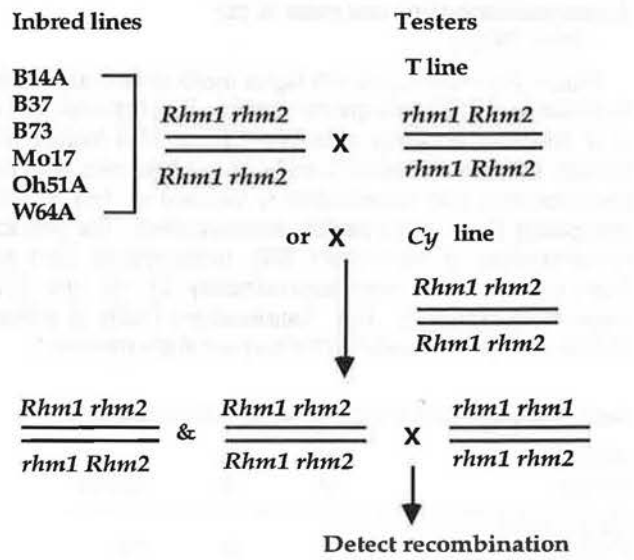


Figure 1. Test of the two-gene model, the recombination test. The *Rhm1* gene in the inbred lines of Smith and Hooker's study should recombine with the *Rhm* gene either in the T line or in the *Cy* line, but not both. Consistency with this predicted result will indicate a questionable definition of the genotypes prior to the tests.

Table 1. Results of the recombination tests. Crossing scheme is shown in Figure 1. Number of symbols (+, –, or seg., etc.) represents the number of families showing the designated event. + Recombination seen; - No recombination; seg Segregation.

Line	Cross with T line (*Rhm2*)	Cross with *Cy* line (*Rhm1*)
B14	+–	––––
B37	+++–	––––
B73	Data not available	seg. seg. seg. seg.
Mo17	seg. +	seg. seg. seg. –
Oh51A	++––	––––
W64A	––	––
c2 W22	+	–
a1-m(papu)	Data not available	–
a2bt	+	Data not available

As shown in Table 1, 5 (B14, B37, Oh51A, *c2* W22 and *a2 bt*) of the 9 lines for which data are available clearly recombined with the T line while one (W64A) did not. The lack of recombination in one line and in some progeny families of others is an indication of heterozygosity of the original T line at the *rhm2* (see later) locus rather than a paradox to our model, since the T line was developed originally by crossing two lines with different origins, followed by selfing for several generations. The results in Table 1 also clearly show that none of the tested lines recombined with the *Cy* line. This indicates that the inbred lines tested have the same dominant gene as the *Cy* line. Mo17 and B73 segregated with the *Cy* line. Tests of the original parent of B73 and one progeny family showed that our B73 line has a homozygous recessive genotype (*rhm1 rhm2//rhm1 rhm2*). A test of two progeny families showed that Mo17 has an array of genotypes at the *rhm1* locus (see later).

Since the *rhm* gene originally identified by Smith and Hooker is often referred to as *rhm1*, we now designate the dominant gene in the *Cy* line as *Rhm1* (*rhm1* for a more generic term), which is the same as the originally identified *rhm* gene, and the dominant gene in the T line as *Rhm2* (*rhm2*), which is a second gene involved in chlorotic-lesion resistance to *B. maydis*. Note that these designations are reversed from our last year's report (Chang and Peterson, MNL 68:5-6, 1994), to be consistent with the literature.

A silky-tassel (*sit*) mutant

--Ru-Ying Chang and Peter A. Peterson

In a line of the genotype, *T2-3e~a-m(papu)//N a sh2* (translocation breakpoint linked to *a-m(papu)*, a plant, designated 922036-1, showed a heavily silky tassel in the summer of 1992. The tassel was covered by very long, almost evenly distributed, silks. Pollen amount was reduced substantially and the pollen shed very poorly. The selfed top ear did not yield any seed set and thus only the outcrossed second ear was obtained. This mature ear was also covered by long silks which were difficult to remove. Normal kernels were interspersed with almost empty kernels (may be partly due to the translocation involved).

The F1 from this cross showed both normal tassels and normal ears, indicating that silky tassel is a recessive trait. F2 progeny were planted in 1993 summer. The segregation for silky tassels is listed in Table 1.

Table 1. Segregation ratios for *sit* mutants in the F2 generation of *sit* x normal or selfed progeny of a mutant plant (last row).

Entry	Silky-tasseled	Normal
1993 F2	13	111
1993g F2	3	15
1994 F2	4	13
1994 mutant selfed	3	8

The silky phenotype expressed in the F2 was less severe and showed a wide array of severity. The five light silky tassels (Table 1) showed only a few long silks. The segregation did show a clear-cut result. Deviation from 3:1 was highly significant (chi^2 = 13.936), and that from 15:1 was approaching significance (chi^2 = 3.794). Segregation in the 93 greenhouse (93g) and 94 summer approached 3:1 closely (15:3 for 93g and 11:4 for 94). However, selfed seed of plants with medium silky tassels (severely silky plants could not be successfully selfed) yielded less than 1/2 offspring with silky tassels.

The above observations show that the silky tassel phenotype is probably controlled by a single recessive gene whose expression is influenced by other factors such as environment, etc. Tests have shown that this phenotype is independent of either the *a-m(papu)* mutable allele (*En*) or the translocation feature (T2-3e) and is not dependent on the direction of the cross.

Three silky-ear mutants (*si*) were reported by Fraser in the 1930's (Fraser, J. Hered. 24:41-46, 1933). Ears of our silky tassel (*sit*) mutants showed a greater degree of severity than *si1* reported by Fraser. *sit* ears closely resemble his *si2* and *si3* mutants, though are less severe. One distinct characteristic is that our *sit* mutant showed severe silky phenotype on tassels, which was reported to be a minor characteristic for Fraser's mutants. Allelism tests and mapping experiments have not been executed.

Mutations in several *En*-containing populations

--Ru-Ying Chang and Peter A. Peterson

It was noted that some *En*-containing populations in our regular nursery plots generated mutants at notable frequencies. During the seasons between 1990 greenhouse and 1992 summer, at least 24 mutant plants were noted. The 24 mutants can be traced back to at least 5 independent origins. The number and phenotypes of the mutants and the populations in which the mutants originated are listed in Table 1. The population size included from the '92 summer nursery back through '89 summer, to which the farthest mutant is traced.

Table 1. Mutants obtained from several *En*-containing populations. 913834 and 924602 can be traced back to a common parent in one generation, while 922018, 922020 and 922021 can be traced back to a common parent in two generations. All striped mutants are considered to be 2 in calculation of mutation rates, since they have two independent origins. The 3 mutants in the last row were considered as one.

Entry	Genotype	Pop'n size	Mutant	Mut. rate
913834	T2-3d~*a-m(papu)* */a et* x *Ht B37 or* @	510	5 striped	3.92×10^{-3}
924602			3 striped	
922018			6 striped	
922020			2 striped	
922021			2 striped	
922036-1	T2-3e~*a-m(papu)/a sh* @	214	1 silky tasseled	4.67×10^{-3}
922350	T4-6(033-16) ~*c2-m1/+* x Normal or @	720	1 dwarf	1.39×10^{-3}
924832, 33	T7-9(027-9) ~*wx-844/ c sh wx* @	343	3 dwarfs	2.92×10^{-3}

All mutant phenotypes are heritable and independent of *En* activity and independent of the translocation feature. The striped-leaf mutants are being investigated using reciprocal crosses to verify whether they were generated by deficiencies as the mutants isolated by Robertson and Stinard (Genetics 115:353-361, 1987). The silky tassel mutant is reported in an accompanying report (this issue). All the dwarf mutants grow between 50 and 70 cm tall. Their leaves were compacted together, and their pollen and ears were severely affected so that no seed was obtained from crosses in both ways.

The mechanisms by which *En* enhances the generation of mutants are not clear, though Robertson and Stinard (Genetics 115:353-361, 1987) proposed several for the high mutation rates associated with their *Mutator* populations.

Evidence that deletions of the *yg2* region of the short arm of chromosome nine found in *Mutator* stocks were induced by the *Mutator* system and were not spontaneous

--Donald S. Robertson

In an excellent review of the *Mutator* system by Bennetzen, Springer, Cresse and Hendrickx (Crit. Rev. Plant Sci. 12(1/2):57-95, 1993), they discussed the evidence presented by Robertson and Stinard (Genetics 115:353-361, 1987) that the *Mutator* system induced deletions involving the *yg2* locus. Bennetzen et al. concluded that, because of the absence of a proper control population, there was insufficient evidence presented in the Genetics paper to distinguish between deletions induced by the *Mutator* system and those that might occur spontaneously. They suggested that "...a non-*Mutator* line with a similar background, would have provided such evidence" (i.e., that the deletions were *Mutator*-induced).

The control data they desired were reported by Robertson (Mol. Gen. Genet. 200:9-13, 1985, see Materials and Methods section and Table 3). In this paper, the *Mutator* and the control stocks were described for the isolation plot involved in the production of the *yg2-Mu* isolates analyzed in the Genetics paper. "The control tests were carried out in the same manner [as those for the production of *Mutator* mutants] except that standard (non-*Mu*) lines (used for propagating the *Mu* lines used in the *Mutator* tests) served as the female parent." Thus, the requirements stipulated by Bennetzen et al. for a proper control were met. The pertinent data are as follows: 125 *yg2* mutants out of a population of 779,338 from the *Mutator* population, zero *yg2* mutants from a control population of 527,041. Eighteen of these *yg2-Mu* mutants have been shown to involve deletions (Genetics 115:353-361, 1987 and Genetics 136:1143-1149, 1994). Nine of these deletions were examined cytologically, and seven of them had the terminal knob found on the short arm of chromosome nine. Due to the failure of Robertson and Stinard to repeat a complete description of both the *Mutator* and the control populations involved in the production of the *yg2-Mu* mutants in the Genetics article or to include a citation of the Molecular and General Genetics paper as a source of this information, Bennetzen et al. were not aware of the control that had been involved in these tests.

Before submitting this note to the Newsletter, I sent the first draft of it to Jeff Bennetzen for his information and criticisms. Although he thought the controls utilized in the *yg2* experiment provided a high likelihood ("90% certain") that the deletions were *Mutator*-induced, he suggested that "*Mu*-off" lines (i.e., lines that had once been active *Mutator* lines but in subsequent crosses had lost their ability to induce mutations) would serve as a better source for the control population. He felt that such lines would be more appropriate because the active *Mutator* stocks used in this test could have activated a breaker *Ds*-like activity of some other transposable element system carried by the *Mutator* stocks. The presence of other active transposable element systems in active *Mutator* stocks has been found by several workers (e.g., Patterson, et al., Genetics 127:205-220, 1991). There are, however, also difficulties with using "*Mu* off" lines as controls. Not all lines that first appear to be off are found to be so on further testing, and some such lines have been found to cycle back to an active state (Hardeman and Chandler, Dev. Genet. 10:460-472, 1989). Thus there is probably no way to be certain that any given deletion was actually induced by the *Mutator* system. It is not unreasonable to assume, however, that at least some of the 13 known deletions found in the *Mutator* lines were the result of the activity of this system. The observation that seven deletions were not terminal suggests that some were not induced by a *Ds*-like system.

Thirteen mutable *yg2* mutants were found in this experiment and all had the small wildtype sectors typical of unstable *Mutator*-induced mutations. None had the larger wildtype sectors that are characteristic of many, but not all, unstable mutants produced by other transposable element systems.

Final proof as to the origin of any given deletion awaits molecular analyses of these deletion events. A first step in such a process would probably involve the cloning of the *yg2* gene. The *Mutator*-induced mutable *yg2* mutants are available from the Maize Genetics Cooperation or from me for anyone interested in pursuing this matter further.

Evidence for orthologous QTL for plant height between maize and sorghum: verification with replicated progeny

--D. Ahnert, M.G. Pereira and M. Lee

RFLPs are being used to estimate the genetic locations and effects of QTL for plant height across generations and environments. In this note, we report the preliminary plant height QTL mapping results obtained by investigating F2:3 lines evaluated in one environment and compare the results to those obtained with F2 plants studied by Pereira and Lee (Theor. Appl. Genet. 1994). The F2:3 population was developed from a cross between Combine Kafir 60 (CK60) and PI229828. CK60 is an inbred line and a representatative of the subspecies *bicolor*. PI229828 is a wild-type representative of the *Sorghum bicolor* subspecies *drumondii*. They differ in several traits including panicle morphology, plant height, maturity, tiller production, and resistance to insects. One hundred and fifty-two F2:3 lines, derived by self-pollinating the F2 plants, were grown in a 12 x 13 rectangular lattice design of one-row plots with two replications near Ames, IA in 1994. A total of 8 traits were considered including plant height, peduncle size, panicle length, leaf length, leaf width, node number, tiller number, and stock diameter. These traits were also measured in the same location in 1993.

QTL were determined on the adjusted entry means at 111 loci by interval mapping using MAPMAKER-QTL and single-factor analysis of variance. Six independent QTL for plant height were identified in linkage groups A, B, D, E, F, and H. Individually, the QTL accounted for 11 to 32% of the phenotypic variation. The multiple QTL model accounted for 77% of the variation for plant height in this population. Additive effects ranged from 15 to 52 centimeters and the dominance effects from 5.7 to 123 centimeters. In all cases, alleles for increased plant height were derived from the tall parent (PI229828). Alleles from this parent were overdominant, except in linkage group H where the CK60 allele was partially dominant. As hypothesized by Pereira and Lee (Theor. Appl. Genet., 1994) the QTL of linkage group A, E and, H may correspond to the sorghum genetic loci *Dw3*, *Dw4* and *Dw2*, respectively (Quinby and Karper, Agron. J. 46:211-216, 1954). The remaining QTL (group B) may correspond to the *Dw1* locus or to additional loci influencing plant height not yet reported in sorghum.

The genetic locations of the QTL mapped with F2 plants coincide with the locations of the QTL mapped in the same linkage groups in the F2:3 population. Thus, the same genomic regions

affecting plant height were identified across generations. The direction of the additive and dominance effects of these QTL were the same in both generations, but the magnitude of the effects differed. F2:3 progenies had slightly higher values for additive and dominance effects. This is probably due to the fact that F2:3 progenies were evaluated on a plot mean basis, which reduces environment variation and experimental error. Evaluation based on replicated progenies may also increase the efficiency of identifying QTL with small effects. This may have contributed to the identification of the two additional QTL for plant height mapped in the F2:3 generation (linkage groups D and F).

Comparative mapping in sorghum and maize has revealed that plant height QTL of sorghum linkage groups A, E, and H may be orthologous to plant height QTL identified for maize chromosomes 1, 6, and 9, respectively (Pereira and Lee, Theor. Appl. Genet., 1994). In both the F2 and F2:3 generations, the confidence intervals obtained from the sorghum QTL are within those obtained 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. 88:7-16, 1994, for QTL on chromosomes 1 and 6).

All measured traits will be considered in further analyses for consistency of QTL detection across generations and environments.

Comparison of QTL mapping for plant height in F2:3 and F6:7 generations

--D.F. Austin and M. Lee

The first objective of our study was to locate and characterize genetic factors controlling quantitative traits in recombinant inbred (RI) progeny of an elite maize population. The second objective was to compare the RFLP linkage map and detection of quantitative trait loci (QTL) in F2:3 and RI (F6:7) lines of the same population grown at the same location in different years. With the RI population, we expect to detect QTL with smaller phenotypic effects due to increased replication of the homozygous parental marker classes. Also, the near homozygous state of RI progeny should result in a higher precision of trait measurement.

The population was derived from a cross between inbred lines Mo17 and H99, which differ for several traits, including insect resistance, kernel size, grain yield, ear length, plant height, and flowering traits. From the original cross, 186 unselected F6:7 lines were developed. The lines were grown in a 14x14 lattice design with two replications near Ames, IA in 1993. A total of 14 traits were evaluated including ear height, plant height, ear-to-tassel interval, anthesis, silk emergence, silk delay, grain yield, ear number/plant, ear length, ear diameter, cob diameter, kernel row number, kernel depth, and 300 kernel weight.

A linkage map of 101 loci (100 RFLP and 1 morphological) was generated with MAPMAKER/EXP 3.0 using the "ri self" setting. Ten well characterized linkage groups were observed with an average interval between loci of 15.4 cM. Using 150 F2:3 lines from the same population, Veldboom et al. (Theor. Appl. Gen. 88:7-16, 1994) produced a linkage map with 104 loci (103 RFLP and 1 morphological). The total map distances for the F2:3 and RI maps using the 87 common marker loci are nearly identical at 1419 cM and 1420 cM, respectively. Map order is identical except for two loci on the end of the long arm of chromosome 9 linked by 2 cM in the RI map. These loci, *npi209* and *bnl14.28*, are present in the opposite order in the F2:3 map.

Single-factor analyses of variance were conducted for all pairwise marker loci and quantitative trait combinations. Trait data consisted of adjusted entry means from the lattice analysis. A significant ($P<0.05$) contrast between the homozygous parental genotypic classes was interpreted as evidence for linkage between a QTL and a marker locus. From the total number of significant loci for a trait, a subset of loci was selected to represent the total number of QTL detected by selecting the loci with the highest significance from a cluster of closely linked significant loci. If multiple loci on the same chromosome were significant when evaluated simultaneously in a model, they were included in the subset of significant regions as distinct QTL explaining unique portions of the trait variation.

For plant height, 31 loci and 7 chromosomes were significantly associated with trait variation. Twelve QTL were detected accounting individually for 2.3 to 11.8% of the phenotypic variation. Collectively, the additive effects of the 12 QTL explained 47.2% of the phenotypic and 52.4% of the genotypic variation as determined in a multiple model. QTL on 1S, 1L (2 QTL), 2S, 2L, 4L, 5L, and 8L have alleles from Mo17 (the taller parent) contributing to increased plant height. QTL on 3L, 4S, and 7L (2 QTL) have alleles from H99 contributing to increased plant height. Also, six of the seven QTL with the largest additive effects have Mo17 contributing increased plant height, but the second largest QTL is associated with H99 alleles. The QTL with the largest R2 value, identified by *umc37* on 1L, has an additive effect of 6.7 cm, representing a difference of 13.4 cm in RI lines polymorphic at this locus.

Veldboom et al. (Theor. Appl. Gen. 88:7-16, 1994) identified five plant height QTL in the F2:3 generation of this population using interval mapping with MAPMAKER QTL. Four of the five regions were also identified with RIs. QTL were detected in both generations at *umc37* (1L) and *umc34* (2S). QTL were detected in both generations on 4S and 7L but at loci differing in location by 17 cM and 11 cM (based on F2:3 map), respectively. For all four common QTL, the parental alleles contributing to increased plant height were the same for the two generations. A QTL was detected in the F2:3 on 6L, but the region was not significantly associated with plant height variation in the F6:7. The F2:3 and F6:7 generations were both grown at the same locations, but the environmental conditions were very different in 1989 and 1993. Conditions in 1989 were the seventh driest on record while 1993 was one of the wettest growing seasons on record. Despite this difference in generations and environments, most regions associated with plant height variation in the F2:3 were also detected in the F6:7.

In the F2:3 generation, a large significant region on chromosome 1 was attributed to a single QTL identified by *umc37* with the genetic effect for increased plant height derived from Mo17. The region could not be further resolved with the F2:3 generation. This region has been resolved into three distinct QTL in the F6:7 identified by *P1*, *umc37*, and *umc86A* on the basis of evaluation in multiple models (Figure 1). Genetic effects for increased plant height for all three QTL are derived from Mo17. This suggests that the increased recombination fraction and precision of RIs allowed the separation of one large linkage group of significant loci into multiple linked QTL.

The remaining eleven traits evaluated with the RI population in 1993 have also been analyzed in the same manner. The experiment was repeated in 1994 for an additional environment. Also, the F2:3 experiment has been repeated a second year by Veldboom et

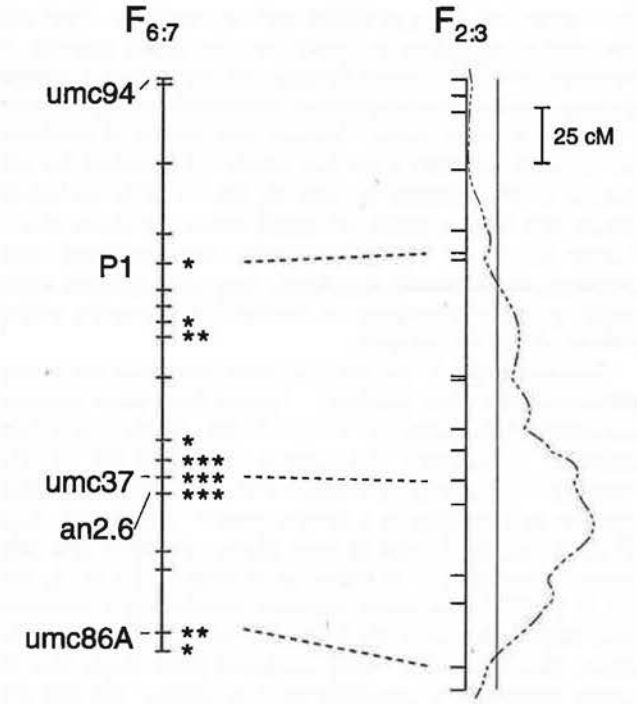


Figure 1. Comparison of plant height QTL detection on chromosome 1 in F2:3 and F6:7 generations of a single-cross population of inbreds H99 (short) and Mo17 (tall). Names of RFLP marker loci are on the left. In the F2:3, a LOD threshold of 2.0 was used. Significance levels in the F6:7 analysis are noted by *, **, and *** representing 0.05, 0.01, and 0.001, respectively.

al. (in review). Additional comparisons will be made to determine consistency of QTL detection across generations, environments, and independent samples of this population.

Ac transposon-induced recombination at the *P* locus

--Yongli Xiao and Thomas Peterson

The *P* gene encodes a Myb-homologous transcriptional regulator of flavonoid biosynthesis in the kernel pericarp, cob glumes, and other floral organs (Grotewold et al., Cell 76:543-553). Using the transposable element *Ac* as a tag, the *P* gene was cloned and found to have a unique structure: the coding sequence is flanked by two long (5.2 kb) repeats in direct orientation (Lechelt et al., Mol. Gen. Genet. 219:225-234; see Figure 1 in following report by Zhang and Peterson). We subsequently demonstrated that the *Ac* element, when inserted between the two direct repeats in the *P-ovov-1114* allele, induces a significant level of homologous recombination between the repeats (Athma and Peterson, Genetics 128:163-173). A similar enhancement of recombination has been shown to be induced by *Mu* transposons inserted in direct repeats at the maize *Knotted* locus (Lowe et al., Genetics 132:813-822).

To further characterize the mechanism of this transposon-induced recombination, we have compared the mutability of six *P* gene alleles with *Ac* insertions at different sites in the locus. Each allele was tested as a heterozygote with *P-wr*, in the same hybrid (4Co63/W23) genetic background. Plants were detasseled and allowed to pollinate with the *r-m3::Ds* reporter stock, and the mature ears were examined for the presence of colorless pericarp sectors indicative of *P* gene mutations, including deletions produced by recombination at the *P* locus. The results can be summarized briefly as follows: the three alleles with *Ac* inserted at various sites between the direct repeats of the *P* locus had twice the frequency of pericarp sectors (3.8%) as did three alleles with *Ac* insertions either within or outside the 5' direct repeat (1.9%). We have previously estimated that, for the *P-ovov-1114* allele, 80 to 90% of the colorless pericarp sectors are due to deletions generated by recombination, and 10 to 20% are due to intragenic transpositions and other mutations (Athma and Peterson, 1991). These results suggest that the ability of a transposable element to stimulate recombination between repeated sequences is significantly enhanced by insertion of the element between the repeat sequences.

Cloning and characterization of *P-ww*-12:27-3* allele

--Jianbo Zhang and Thomas Peterson

In last year's Maize Newsletter (68:10), we reported a new *P-ww* allele of the *P* gene with interesting characteristics. The *P-ww*-12:27-3* allele suppresses the orange pericarp pigmentation specified by *P-ovov-1114*, and has a very strong negative *Ac* dosage effect. To determine the structure of the *P-ww*-12:27-3* allele, a genomic library was constructed in lambda FIX II. Ten clones were isolated which hybridized to probes from both *Ac* (the internal 1.6 kb *Hin*dIII fragment) and the *P* gene (JZ001, a PCR product within *P-rr* fragment 10). Five clones (Type I) also hybridized with *P-rr* fragment 8B, while the other five clones (Type II) did not. Southern blot and PCR analysis indicated that Type I clones contain an inverted duplication of part of the *P-rr* gene, which is consistent with genomic Southern blots, and could suggest that *P-ww*-12:27-3* inhibits *P-ovov-1114* expression through an anti-sense RNA mechanism. The duplication in *P-ww*-12:27-3* is at least 10 kbp in length, beginning in *P-rr* fragment 10 and extending beyond the *Eco*RI site located 6 kbp 5' of the *P-rr* transcription start site (see Figure 1). Type II clones contain most or all of an apparently normal *Ac* element, and an unknown DNA fragment which is probably the extension of the duplication present in Type I clones. Northern blot analysis showed that the pattern of *Ac* transcription in *P-ww*-12:27-3* is different from

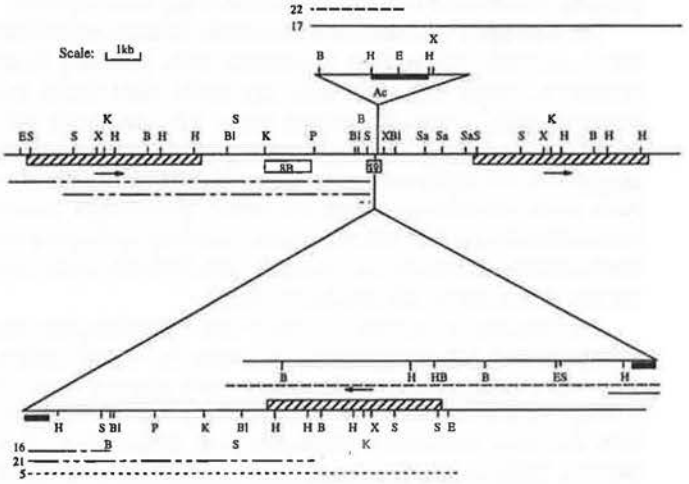


Figure 1. Restriction map of the *P-ww*-12:27-3* allele. The central map indicates the structure of the *P-rr* allele, with 5.2 kb repeats (hatched boxes) in direct orientation flanking the *P-rr* transcribed region. The *P-ww*-12:27-3* allele contains two insertions within *P-rr* fragment 10: an *Ac* element (upper triangle), and a large complex insertion (lower triangle) containing both *Ac*-homologous sequences (black boxes) and *P*-homologous sequences in inverted orientation. The different dashed lines represent different lambda clones: clones 16, 21, and 5 are Type I clones, clones 22 and 17 are Type II clones. Restriction enzyme sites are indicated as follows: B = *Bam*HI, BI = *Bgl*II, E = *Eco*RI, H = *Hin*dIII, K = *Kpn*I, P = *Pst*I, S = *Sal*I, Sa = *Sac*I, X = *Xho*I. Not all sites for *Pst*I and *Sac*I are shown.

that in *P-vv* and *P-ovov*, but the mechanism of the high negative *Ac* dosage effect of *P-ww*-12:27-3* remains to be determined.

Expression of the *P-wr* allele of the *P* gene

--Surinder Chopra and Thomas Peterson

The *P* gene encodes a Myb-homologous transcriptional regulator of the flavonoid biosynthetic genes in the pathway leading to the production of a red flavonoid pigment called phlobaphene. Alleles of the *P* gene differ strikingly with regard to the distribution of phlobaphenes in kernel pericarps and cob glumes. We have previously characterized the *P-rr* allele, which specifies red pericarps and red cob glumes, and we are now investigating the mechanism of organ-specific expression of the *P-wr* allele, which specifies colorless pericarps and red cob glumes. RNA blot and RT-PCR analyses have shown that both colorless pericarps and the red cob glumes of *P-wr* plants contain the *P-wr*-specific transcripts. However, transcripts of the genes *C2*, *Chi* and *A1*, encoding enzymes for flavonoid biosynthesis, are present at a very low level in *P-wr* pericarps between 11 to 22 DAP (days after pollination). Using PCR methods, we have amplified *P-wr*-specific cDNAs from pericarps and cob glumes. Compared to *P-rr*, the *P-wr* 3' end and thus the -COOH terminus of the protein are completely different. In contrast, the 5' regions of *P-rr* and *P-wr* are more similar, but contain several changes in the Myb-homologous DNA binding domain. One of the sequence differences in the *P-wr* protein would substitute a tyrosine residue for a conserved cysteine in helix 3 of tryptophan repeat 1 in the Myb-homologous domain; this change will be investigated as a possible cause of tissue-specific activity of the *P-wr* protein.

Genomic structure of the amplified *P-wr* locus

--Surinder Chopra, Peifen Zhang and Thomas Peterson

We have previously reported that the *P-wr* allele contains a 5- to 10-fold tandem repetition of *P* gene sequences (Athma and Peterson, MNL 65:46, 1991). Southern blot analysis demonstrated that, except for a 700 bp fragment at the 3' end of the *P-rr* gene, all other *P-rr* gene probes (Lechelt et al., Mol. Gen. Genet. 219:225-234) map within the amplified region of the *P-wr* allele (Athma, unpublished). To determine the fine structure of the *P-wr* allele, we have analyzed several genomic lambda clones isolated from a *P-wr* (W23) genomic library. All the lambda clones were overlapping, and each one contained a complete 13.3 kb unit, and varying lengths of flanking sequence representing the 5' or 3' ends of the 13.3 kb sequence (Figure 1). These results show that *P-wr* contains a tandem array of at least three units of a 13.3 kb sequence. The *P-wr* repeat structure is further supported by analysis of two YAC clones of 180 and 200 kbp provided by Keith Edwards (ICI); restriction mapping of the YAC DNAs is consistent with the map predicted by tandem repetition of the 13.3 kbp sequence. We plan to fractionate the YACs by plasmid integration and marker rescue and thereby isolate individual *P-wr* repeat units for sequence comparisons and functional tests.

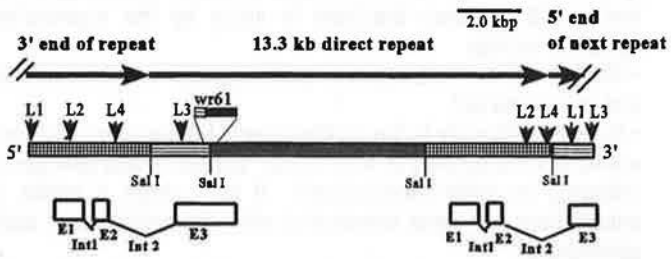


Figure 1. Restriction map of *P-wr* locus. Composite restriction map was generated from overlapping *P-wr* genomic lambda clones. Vertical arrowheads indicate endpoints of lambda clones L1, L2, L3 and L4. A full length 13.3 kb direct repeat and the 3' and 5' ends of the flanking repeats are shown as horizontal arrows. DNA fragment wr61 represents a unique sequence at the 3' end of the *P-wr* cDNA which replaces the 700 bp fragment 14 present in the *P-rr* allele. Boxes with the same filling pattern represent similar DNA sequences. Transcriptional units with exons (E1, E2, and E3) and introns (Int1 and Int2) are shown below the genomic map..

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Structural and functional elements of the 5' region of the *P-rr* gene

--Xianggan Li, Laura Tagliani, Lyudmila Sidorenko, Ben Bowen, and Thomas Peterson

The *P-rr* gene conditions the synthesis of a phlobaphene-like red pigment in mature cob glumes, pericarps, and husks. The *P-rr* gene is expressed at a relatively low level, predominantly in the female inflorescence, and at a late stage of development. These characteristics suggest that the *P-rr* promoter could be useful for directing the expression of foreign genes for pathogen resistance specifically in pericarps, silks, and cob glumes. The *P-rr* 5' region contains a Tourist-like mobile element located approximately 1 kbp 5' of the transcription start site; the same element is found at the identical site in the 5.2 kbp direct repeat 3' of the *P-rr* gene, suggesting that insertion of the Tourist-like element occurred prior to duplication of the 5.2 kbp direct repeats flanking *P-rr*.

Functional analysis of the *P-rr* promoter via transient assay gave the following results. A basal construct, containing the GUS reporter gene, *Adh1* intron, 220 bp *P-rr* untranslated leader and 240 bp upstream from the *P-rr* transcription start site, gives a low but detectable level of GUS expression when introduced via microprojectile bombardment into pericarps. Addition of the adjacent 5' 1.0 kb *P-rr* fragment containing a tRNA-homologous sequence, or a 1.2 kb *Sal*I fragment located 4.6 kb 5' of the *P-rr* transcription start site, boosts the activity of the basal construct about 10-fold. The increased expression suggests that both fragments from the *P-rr* 5' region contain enhancer elements. Because *Ac* insertions in either fragment can reduce *P-rr* expression in vivo (Moreno et al., Genetics 131:939-956), both sequences may be important for *P-rr* expression. The extended *P-rr* promoter construct containing the 1.24 kbp immediately upstream of the 5' start site directs the expression of the GUS reporter gene preferentially in pericarp compared to scutellum when the amount of DNA used is lowered to 100 pg per bombardment. We have initiated stable transformation experiments with these constructs to analyze the activity and tissue specificity of the *P-rr* promoter in transgenic maize plants.

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Development of a research proposal for integrating molecular and anthropological approaches to understanding the co-evolution of maize and human cultures

--Deborah A. Muenchrath, Peter Bretting, J. Stephen Smith, and Karen R. Adams

Agriculture is embedded in the natural ecosystem and the human social system. It is the bridge between them. Norman Borlaug, 1990

The co-evolution of maize and humans in the Americas enabled both species to flourish over millennia. Their interrelationship is reflected in the maize and maize-based cultures found from Mesoamerica through the southwestern United States. Maize seed dispersal and maintenance depends on humans, and maize productivity influences human population sizes and migrations. Thus, the development of human cultures and the evolution of maize are closely intertwined.

Ancient, as well as contemporary, maize-human relationships hold lessons pertinent for germplasm and resource stewardship. Understanding the co-evolution of maize and associated human cultures may provide critical insights for charting future agricultural and societal development. Such an understanding may be achieved through innovative research that synthesizes the knowledge and methods of various disciplines and perspectives.

To discuss and explore collaborative, interdisciplinary approaches to the study of the interrelationships between maize evolution and human cultural development, a workshop was organized. It was sponsored by Pioneer Hi-Bred International, Inc. and hosted by the USDA-ARS North Central Regional Plant Introduction Station and Iowa State University, August 29-31, 1994 at Ames, Iowa. The workshop convened thirty individuals interested in maize and human cultures, including researchers from a variety of disciplines, and specialists in cultural heritage conservation and in germplasm management. Participants came from the United States and Mexico, and were affiliated with academia, industry, governmental agencies, tribal offices, museums, and non-profit germplasm conservation organizations.

Prior to the workshop, selected participants submitted background papers which summarized issues and common research methods of their respective disciplines. These compendia briefed all participants on archaeology and ethnobotany, plant germplasm management and breeding, molecular markers and genetic characterization of maize, and evolutionary interpretation of DNA data. At the workshop, displays illustrated the various approaches. Speakers introduced the concerns, challenges, and potential contributions, as well as limitations, of the various fields:

- Hopi tribal perspectives on evolution, and migration traditions and history; and Hopi traditions about maize: Maize as the life plan (Walter Hamana, Hopi Cultural Preservation Office);
- Maize and human cultures in the ancient U.S. Southwest (Karen R. Adams, Crow Canyon Archaeological Center);
- Contemporary maize and human cultures in the U.S. Southwest (Carol Brandt, University of New Mexico); and
- Maize molecular markers (Tim Helentjaris, University of Arizona, and J. Stephen Smith, Pioneer Hi-Bred International, Inc.).

Participants toured North Central Regional Plant Introduction Station fields to view the phenotypic diversity exhibited among sixty maize landraces native to the United States and northwestern Mexico. They also visited the Station's seed storage facilities to learn more about maize germplasm management systems.

Participants gathered in two discussion groups, moderated by Drs. John Doebley and Peter Bretting. The groups' tasks were to: (1) identify areas of mutual interest across disciplines, (2) establish long-term research objectives, (3) explore approaches and technologies to address those objectives efficiently, and (4) outline specific research projects. Both groups determined that a descriptive database on modern and historic maize and an inventory of archaeological maize collections were required for future research. In addition, specific research issues, hypotheses, and appropriate technologies, materials, and sampling strategies were considered.

I. Geographic Focus. As conceived in this workshop, studies will focus initially on the maize and cultures of the southwestern United States and adjacent northwestern Mexico. This region has an extensive archaeological record, including a number of uncharred maize collections, continuity between ancient and contemporary indigenous cultures and agricultural systems, and connections with central Mexico, maize's center of origin. The project will eventually expand to other regions of the United States, particularly those encompassing the progenitor races of the highly productive Corn Belt Dents: the Northern Flint and Southern Dent races. Companion studies will trace maize phylogenies between Mexico and the United States.

II. Molecular Analysis. The criteria for identifying optimal molecular technologies emphasized that they: (1) be in the public domain, (2) be cost-effective, and (3) provide phylogenetic information. It was determined that molecular data on extant cultivars, experimentally-deteriorated specimens, and archaeobotanical specimens from ample collections should be analyzed molecularly to determine the feasibility and utility of the technologies before applying them to scarce archaeobotanical materials. Technologies for amplifying and preserving DNA from such archaeobotanical materials were discussed.

III. Archaeological Questions. Specific archaeological questions that would be more tractable to study by the approaches discussed include:

- What patterns in maize genetic diversity occur over time on local and regional levels?
- Did maize diversity in the southwestern U.S. result from several waves of introductions, or from in situ selections and divergence following an initial introduction? If there were a series of introductions, do these correspond with other evidence of trade connections?
- What is the relationship of Utah-area Fremont Dent with other southwest maize?
- When shifts in maize morphology occurred (e.g., decrease in ear row number), were there concurrent shifts in genetic patterns?
- Do maize genetic changes correspond to changes in human material culture?

• What is the relationship between the degree of maize genetic diversity and the periodic human population dispersals and aggregations observed in the ancient southwestern U.S.?
• Can human migrations be traced through the distribution patterns of specific maize genes (allele distributions)?
The answers to the preceding questions have implications for modern germplasm and environmental management, plant breeding, and genetics.

IV. Perspective of Maize-Based Cultures. A proposal to include the knowledge systems of indigenous peoples was presented by Ricardo Salvador. Dr. Salvador discussed the information about maize and human cultures that is encoded in the knowledge, languages, and traditions of Native Americans. Integrating these knowledge systems with those of the scientific community will enhance interpretations and understanding of human-maize relationships. Indigenous people should be consulted and included as an integral part of the planning and implementation of any research program examining maize evolution and human cultural development.

V. Handbook on Maize Diversity. It was proposed that the information obtained through the research program be compiled into a comprehensive handbook on the indigenous maize races of the United States. Such references are currently available on the maize of Mexico, Cuba, Peru, and other Latin American countries. The version for the U.S. maize races would describe extant and ancient maize, and their relationships to each other and to non-U.S. maize. Descriptions of extant races would include ecogeographical, ethnographic, morphological, agronomic, physiological, molecular, and elemental data. Descriptions of ancient maize races would consist of morphological and molecular characterizations, as well as information from the archaeological record regarding the associated human culture. Interpretive sections would also be included. Such a reference would be useful to many fields of study. Additionally, an electronic version might be developed, which would contain a more complete database; this could be updated more rapidly and frequently and it would aid searches for specific information. Presently, we lack sufficient data to write such a volume. The research projects under consideration would contribute much of the necessary data, so that an ultimate outcome of the research program may include a handbook on U.S. maize.

VI. Training Program. In addition to the scientific goals, a major programmatic objective is to train students in an interdisciplinary fashion. Students will be involved in each phase of the project. They will broaden their perspectives and knowledge, and develop new skills. Anthropology students will develop skills in agroecology, biology, and molecular biology; biology students will become more familiar with anthropological approaches. Core labs will be established at two or more universities for interdisciplinary training. Students will also serve as interns at other facilities as needed. A special effort will be made to include Native Americans and Hispanic students.

In the final workshop session, moderated by Deborah Muenchrath, participants convened in a single group to draft conclusions and outline research initiatives. The research program proposal that emerged provides a stepwise, multi-disciplinary approach to the study of the co-evolution of maize and human cultures that will be significant to several disciplines. This research program will address issues relevant to archaeology, genetics, germplasm management, maize evolution, and plant breeding. It will examine modern, historic, and ancient maize and cultures and their evolution and relationships through time and across geographic areas. It will uncover patterns in both the genetic diversity among maize races and the connections among maize and human cultures. It will also contribute toward the conservation of indigenous human cultures and biological resources. It will provide technical and interdisciplinary training for students of anthropology, archaeology, molecular biology, genetics, genetic resource management, and agroecology. The program also will contribute data for development of a comprehensive, cross-disciplinary database and reference handbook on maize native to the United States. Table 1 summarizes expected outcomes.

Table 1: Expected outcomes of the research program described.

Characterization of Patterns in Genetic Diversity in Time and Space	Interdisciplinary Training	Cultural Conservation Efforts
will have practical utility to:	will emphasize:	will include:
• Archaeology • Germplasm Management • Plant Breeding • Genetics • Systematics	• Anthropology • Archaeology • Molecular Biology and Genetics • Agroecology • Systematics	• Education • Establishment of community seed banks • Cultural heritage conservation

U.S. Maize Handbook

In addition to syntheses of human cultural information and maize systematic data, the handbook will include these types of descriptions and information:

Extant Cultivars	Ancient Maize	Systematic and Evolutionary Relationships
Ecogeographical Ethnographic Morphological Agronomic Physiological Molecular Elemental	Archaeological Record - provenance and date - associated human cultural groups Morphological Molecular	Among ancient and extant maize races With non-U.S. maize

While the specific organization and funding details for such a comprehensive research program remain to be developed, its scientific basis was outlined during the workshop. Important elements of the research plan were identified and a timetable drafted (Table 2).

Some initial elements of the project that are fundamental to subsequent research are already in progress, such as the development of suitable molecular technologies. An inventory of archaeological maize collections is essential for identifying the appropriate materials for morphometric and molecular characterizations. Results from initial morphometric and DNA analyses should help focus the larger, integrated, and multi-disciplinary research program.

The workshop identified six specific technical areas of research:
1) Develop microsatellite or simple sequence repeat (SSR) technology as a "fingerprinting" tool. Current elite inbred lines and segregating populations are now under analysis to develop and map SSR loci. As this technology is perfected, it will be incorporated into the proposed project.
2) Obtain morphometric and SSR characterizations of extant races of maize in the United States, beginning with southwestern materials.
3) Develop techniques for extracting DNA from archaeological maize specimens in suitable amount and of sufficient quality for SSR profiling. Evaluate the potential of conserving ancient DNA by amplifying the entire DNA extract.

Table 2: Outline of proposed research program.

Phase I Objectives
- Establish basic databases to provide framework for subsequent work.
- Obtain preliminary data to test feasibility of applying molecular technologies to archaeobotanical specimens.
- Demonstrate utility of interdisciplinary approach.

Year*	Analyses of Extant Cultivars	Analyses of Archaeological Samples	Molecular Analyses	Elemental Analyses
1-2	• Compile and summarize literature on extant cultivars • Obtain seed from existing collections: USDA/ARS NPGS Native Seeds/SEARCH CIMMYT INIFAP • Identify suitable field sites for field evaluations in the southwestern U.S. and northwestern Mexico • Obtain permission to collect seed and associated information from Native Americans† • Determine appropriate repositories for new collections	• Inventory archaeological maize collections • Obtain curator permission to analyze specimens • Compile archaeological reports corresponding to the inventoried collections and assemble a database from these reports. The database will include information on: - Provenance and Dates - Morphometrics Collect any such missing data, where possible • Assess dates of materials via absolute dating technique (e.g., AMS, TAMS)	• Develop analytical methodologies suitable for extant and archaeological maize specimens and which are available in the public domain (e.g., SSR)§ • Test methods on "artificially-degraded" maize samples (e.g., charred) • Develop methods to amplify and preserve DNA extracted from archaeological maize	• Develop methodologies suitable for extant and archaeological maize§
3-5	• Initiate field evaluations at various locations to begin germplasm characterization: - Morphology - Phenology, etc. • Collect seed and associated information: - Involve germplasm managers, agronomists, anthropologists, and local community - Collect duplicate sets, with one set remaining in the community. This will provide an opportunity for establishing community seed banks for education, and local agriculture, contributing to *in situ* conservation of cultural and genetic resources. • Increase seed, as needed	• Obtain morphometric data from extant cultivars grown in field evaluations (applying standard archaeobotanical technique) to establish a reference database and to determine genetic vs. environmental effects on morphological characters • Simulate processing and degradation (e.g., dessication, shrinkage, deterioration, burning, etc.) with materials from field evaluations to assess processing and post-depositional effects on morphological characters	• Molecular characterization of materials from field evaluations • Begin molecular characterization of archaeological specimens	• Test methods with materials from field evaluations

Phase I Outcomes and Transition to Phase II
- Establish electronic database of maize characterization data.
- Determine feasibility of applying molecular technologies to archaeobotanical specimens.
- Evaluate Phase I in light of specific hypotheses to be tested.
- Publish results of Phase I.
- Seek funding for Phase II.

Phase II Objectives
- Collect data on extant and archaeological maize to address specific hypotheses.
- Establish interdisciplinary student training program.
- Synthesize data on southwestern U.S. maize races for handbook.

Year*	Analyses of Extant Cultivars	Analyses of Archaeological Samples	Molecular Analyses	Elemental Analyses
1-2	• Continue germplasm characterizations in field evaluations	• Compile morphometric data from extant and archaeological specimens	• Molecular characterization of archaeological specimens	• Analyze archaeological maize
3-4	• Compile field evaluation data for southwestern section of U.S. maize handbook	• Compare morphometric data from extant and archaeological specimens	• Compare molecular patterns of extant and archaeological maize	

Phase II Outcomes and Transition to Phase III
- Determine evolutionary relationships among extant and archaeological maize.
- Identify patterns in maize diversity among extant and archaeological maize.
- Synthesize data to address specific hypotheses.
- Publish results.
- Evaluate effectiveness of interdisciplinary student training program.
- Seek funding for Phase III.

Phase III Objectives
- Expand programs to other regions of the United States.
- Initiate QTL mapping to investigate the genetic and physiological bases for southwestern maize adaptations to arid and high elevation environments.
- Draft section on southwestern U.S. maize for handbook.

* Each of these activities will overlap time periods. Some activities are contingent on the success or completion of others; thus, adjustments in this schedule may be necessary.

† Native American groups will be consulted regarding the appropriate ways of studying traditional cultivars and associated practices.

§ This work is already in progress and can stand independently of the rest of the research described.

4) Apply SSR technology to archaeobotanical specimens to generate data to address taxonomic, phylogenetic, and cultural questions.
5) Investigate the potential for elemental chemical analysis to match archaeobotanical specimens with their production locality.
6) Investigate the genetic and physiological bases for maize adaptation to arid and high-elevation environments using quantitative trait loci (QTL) mapping.

From this research, the following research data would emerge:

- Morphometric and DNA descriptions of extant cultivars, which would contribute to generating a new or more comprehensive understanding of the genetic variability in extant races;
- DNA descriptions of archaeobotanical specimens applicable to specific phylogenetic questions; and
- Data and methodologies for further investigation of cultural, biological, and genetic issues.

A comprehensive study will demand cooperation and coordination among many researchers and organizations to utilize resources efficiently and to analyze data effectively. During the project, responsibilities and leadership will shift as the research emphases shift. Nevertheless, a team composed of researchers with expertise from one or more of the disciplines involved will serve as a steering committee to provide coordination and continuity among the studies. The project will involve a partnership among academia, government (U.S. and Mexico), industry, and cultural and non-profit organizations.

Funding for this project will be sought in several phases. Initially, funding will be requested from private organizations and foundations to develop an inventory and database of archaeological materials, to compile relevant literature on extant maize cultivars into a database, and to develop a detailed project plan. Development of appropriate molecular and elemental analysis methodologies will be on-going and may be funded in the form of preproposals for obtaining the preliminary data necessary to demonstrate the feasibility and relevance of these approaches to the overall project. Although the components of the project have their own merit per se, multi-agency funding will be sought from NSF, DOE, and USDA to support a single, integrated research initiative. Funding to establish the student-training labs may be sought separately from other project elements.

We welcome inquiries, comments, or suggestions on the proposed research initiative. Please direct comments to Dr. Peter Bretting at USDA-ARS North Central Regional Plant Introduction Station, Agronomy Hall G214, Iowa State University, Ames, IA 50011-1010.

BALTIMORE, MARYLAND
Carnegie Institution of Washington

The role of TnpA and TnpD in transposition of *Spm*

--Ramesh Raina and Nina Fedoroff

The transcriptional and transpositional activity of the *Suppressor-mutator* (*Spm*) transposable element is subject to epigenetic regulation. There are several distinct epigenetic forms of the *Spm* element which differ in the extent of methylation at the 5'-end of the element. Active elements are unmethylated and inactive elements are methylated at the 5'-end. The 5'-end of the element consists of two regions: the 0.2 kb upstream control region (UCR) which is the element's promoter (Raina et al., Proc. Natl. Acad. Sci. USA 90:6355, 1993) and the 0.35 kb downstream control region (DCR), which is a G+C-rich untranslated leader sequence. The element-encoded proteins TnpA and TnpD are necessary and sufficient for transposition of *Spm* in tobacco cells (Masson et al., Plant Cell 3:73, 1991). TnpA is a DNA-binding protein (Gierl et al., EMBO J. 7:4045, 1988) and there are multiple copies of its 12-bp binding site located at the element's 5'- and 3'- ends. TnpA affects the epigenetic state of the *Spm* element by activating the methylated inactive *Spm* promoter (Schlappi et al., Cell 77:427, 1994). However, the role of TnpA and TnpD in the transposition of *Spm* is not understood.

The main impediment to studying TnpA and TnpD has been the difficulty of over-expressing these proteins in *E. coli*. As a consequence, earlier binding studies used either *in vitro* translated protein or a DNA-protein complex precipitated with anti-TnpA antibodies (Gierl et al., EMBO J. 7:4045, 1988; Trentmann et al., Mol. Gen. Genet. 238: 210, 1993). Here we describe successful production of soluble TnpA from *E. coli*. TnpA was cloned in the pFLAG MAC expression vector (IBI *E. coli* FLAG expression system) such that the FLAG peptide at the N-terminus and TnpA are expressed as a fusion protein. TnpA expression in *E. coli* cells was induced with IPTG. When induction conditions were optimized, more than 50% of the fusion protein was recovered in the soluble fraction (0.1mM IPTG, 25 C incubation temperature at 150 rpm). The fusion protein was then purified by binding to an antibody affinity column containing anti-FLAG antibodies and eluting with FLAG peptide. TnpA purified by this procedure had less than 10% contaminating *E. coli* proteins and was used directly for DNA binding studies.

TnpA-DNA binding studies were done using both an oligonucleotide containing two TnpA binding motifs in a tail-to-tail orientation (shown to have the highest binding affinity for TnpA; Trentmann et al., Mol. Gen. Genet. 238: 210, 1993), and the complete UCR. Binding was detected by band-mobility shift experiments. TnpA bound to oligonucleotides containing the TnpA binding motifs and the UCR, but not to the DCR sequence of the

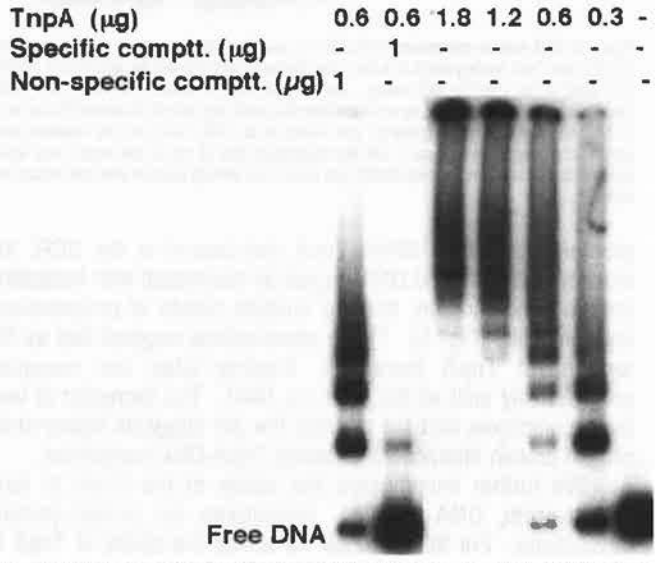

Figure 1: DNA-TnpA complexes analyzed in a mobility shift assay. An 0.2 kb UCR fragment was labeled with ^{32}P-dCTP using Klenow enzyme. Typically 0.1-0.5 ng of labeled DNA was mixed with different amounts of purified TnpA in binding buffer [10% glycerol, 150 mM NaCl, 1mM β-mercaptoethanol, 1mM EDTA, 2 mg poly(dI-dC), 300 mg/ml BSA] in 20 ml reaction volume and incubated at 25 C. The reaction mix was fractionated on a 4% high-ionic-strength polyacrylamide gel (Ausubel et al., Current Protocols in Molecular Biology, 1989).

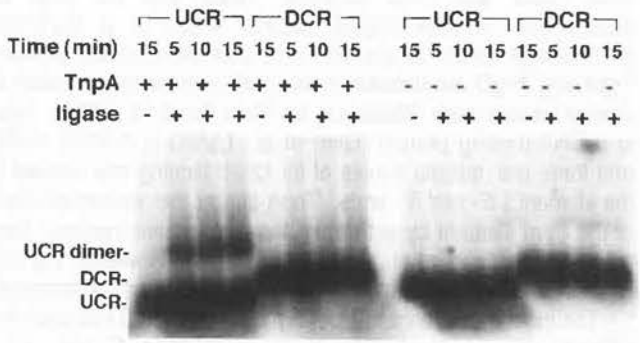

Figure 2: Ligation-mediated dimerization of blunt-ended DNA fragments. UCR (0.2 kb) and DCR (0.35 kb) fragments were labeled as described in Figure 1. The binding reactions were carried out as described in Figure 1, except that the binding buffer was supplemented with 5 mM ATP, 20 mM DTT, 4% polyvinyl alcohol at 25C for 20 min. 1 mg TnpA was used in the binding reaction where indicated. A 10 ml aliquot was withdrawn from the binding reaction and 4 units of T4 DNA ligase were added to the remaining portion. 10 ml aliquots were withdrawn at 5 min intervals and the reaction was stopped by adding Proteinase K. The entire sample was fractionated on a 1% agarose gel.

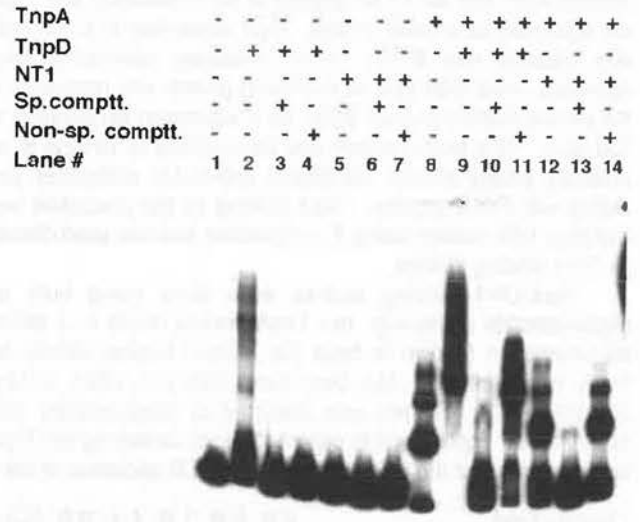

Figure 3: DNA-protein complexes analyzed in a mobility shift assay. An 0.15 kb fragment carrying two TnpA binding motifs in a tail-to-tail orientation was labeled as described in Figure 1 and used in the mobility shift assay. Extracts of tobacco control NT1 cells and TnpD-expressing cells were prepared by a modification of a previously described method (Forter et al., In: Methods in Arabidopsis Research, eds. Koncz et al., 378, 1993) and the reactions were performed as described in Figure 1, with the modification that 10 mg of cell extract was added where indicated and 4 mg of poly (dI-dC) was used in the binding reaction when cell extract was added.

promoter (control). When TnpA was bound to the UCR, the mobility of the labeled DNA fragments decreased with increasing protein concentration, forming multiple bands of progressively lower mobility (Fig. 1). These observations suggest that as the amount of TnpA increases, binding sites are occupied progressively until all the sites are filled. The formation of very large complexes that fail to enter the gel suggests higher-order protein-protein interactions between TnpA-DNA complexes.

We further investigated the ability of the TnpA to form higher-order DNA-protein complexes by protein-protein interactions. For this purpose we tested the ability of TnpA to accelerate the formation of DNA multimers in the presence of ligase as described by Miron et al. (EMBO J. 11:1205, 1992). TnpA stimulated formation of UCR dimers but not DCR dimers (Fig. 2). Since TnpA binds to the UCR, but not to the DCR, these observations suggest that TnpA mediates the formation of

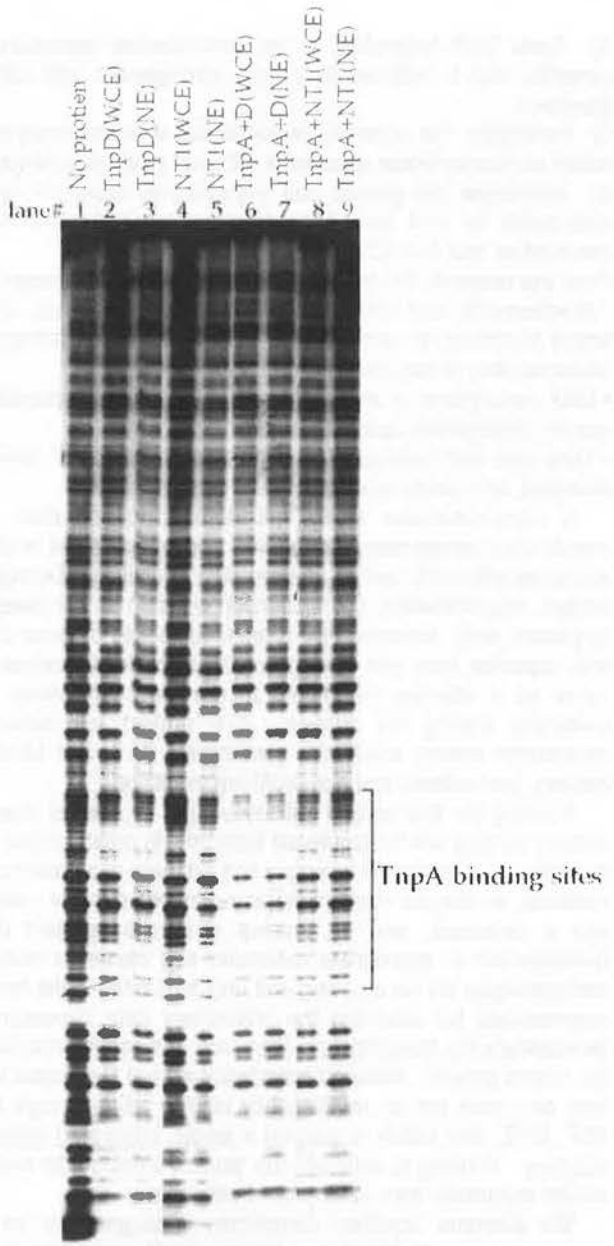

Figure 4: DNaseI footprint analysis following the binding of TnpA and TnpD to an oligonucleotide containing a TnpA binding site. DNA was labeled at one end as described in Figure 1, binding reactions were performed as described in Figure 3, and DNaseI digestion was carried out as described by Ausubel et al. (1989) in Current Protocols in Molecular Biology. Where indicated, 10 mg of whole cell extract (WCE) or 5 mg nuclear extract (NE) was used in the assay.

higher-order complexes by interaction of protein bound to different DNA molecules. Because there are TnpA binding sites at both element ends, the higher order interactions of TnpA molecules bound to each element end may serve to bring element ends together during transposition.

We next assessed the ability of TnpD to interact with the ends of the element and TnpA. Since we have not yet succeeded in expressing TnpD in *E. coli*, we used extracts of tobacco cell lines over-expressing *TnpD* cDNA from a 35S promoter as a source of TnpD, while TnpA was prepared as above. We used oligonucleotides with two TnpA binding motifs in a tail-to-tail orientation in a mobility shift assay. Extracts of the tobacco cells expressing TnpD retarded the mobility of the test oligonucleotide, while extracts of control NT1 cells did not (Fig 3.,

lanes 2 and 5). However, the mobility shift was abolished by both specific and non-specific DNA competitors, indicating that TnpD binds DNA non-specifically (lanes 3 and 4). As previously reported (Trentmann et al., Mol. Gen. Genet. 238: 210, 1993), TnpA retards the mobility of an oligonucleotide containing the double TnpA binding sites (lane 8). Addition of TnpD-containing extracts results in a supershift of the DNA-TnpA complex (lane 9). The supershift persists in the presence of non-specific competitor DNA (lane 11). Addition of specific competitor completely abolishes the shift, indicating that the supershift is due to the interaction of the TnpD with TnpA and not with the DNA (lane 10). Interaction of TnpD with TnpA is further revealed by a DNaseI footprint analysis. As reported in earlier studies (Gierl et al., EMBO 7:4045, 1988), TnpA alone does not protect its binding sites from DNaseI digestion (data not shown). However, a DNaseI footprint was obtained when the oligonucleotide was pre-incubated with both TnpA and tobacco whole cell or nuclear extracts containing TnpD (Fig 4., lanes 6 and 7). No footprint was observed either with TnpD extracts alone, or when control NT1 cell or nuclear extracts were used either alone or with TnpA (lanes 2-5, 8 and 9). Because the TnpA binding site is protected from DNaseI digestion, we infer that the footprint is due to the formation of a DNA-TnpA-TnpD complex. The fact that the footprint is obtained with TnpA and TnpD, but not TnpA alone suggests either that TnpD stabilizes the DNA-TnpA complex or that the binding of the larger protein to the DNA-TnpA complex restricts access of DNaseI to this region in some way.

Thus we have shown that TnpA binds to the end of the element, leading to increased occupation of binding sites as a function of TnpA concentration. We have also provided evidence that there are higher-order protein-protein interactions among the TnpA molecules bound to the DNA, which may be responsible for bringing the ends of the element together during transposition. Furthermore we have shown that TnpD is probably not itself a specific DNA-binding protein, but interacts with TnpA to form a DNA-TnpA-TnpD complex. Our current hypothesis is that TnpD is an endonuclease and that its cleavage site is determined at least in part by its interaction with TnpA.

Analysis of the *Spm*-encoded TnpA and TnpD proteins using single- and double-hybrid protein fusions in a plant transient assay

--Michael Schläppi and Nina Fedoroff

The *Suppressor-mutator* (*Spm*) transposable element is subject to methylation-dependent epigenetic regulation in transgenic tobacco as it is in maize (Schläppi et al., Genetics 133:1009, 1993). The *Spm* promoter alone is rapidly methylated in tobacco (Schläppi et al., Cell 77:427,1994), but only if it contains the G+C-rich first untranslated leader sequence designated the 'downstream control region' (DCR) (Banks et al., Genes Dev. 2:1364, 1988). TnpA, the element's most abundant gene product, transcriptionally activates the methylated, inactive *Spm* promoter, but represses the unmethylated promoter (Schläppi et al., Cell 77:427, 1994). TnpA-mediated reactivation correlates with promoter demethylation. The DNA-binding and protein- dimerization domains of TnpA alone (Trentmann et al., Mol. Gen. Genet. 238:201, 1993) are sufficient for repression of the unmethylated promoter. By contrast, the C-terminus of TnpA, but not its N-terminus, is required for transcription activation of the methylated promoter. Only full-length TnpA, however, effects transposition of *Spm* elements in the presence of TnpD, another *Spm*-encoded protein required for transposition (Schläppi et al., Cell 77:427, 1994; Frey et al, EMBO J. 9:4037, 1990; Masson et al., Plant Cell 3:73, 1991). These results suggested that the C-terminus of TnpA directly activates transcription from the methylated *Spm* promoter and that both the N-terminus and the C-terminus of TnpA interact with TnpD to produce a transposition-competent complex. We have investigated these questions using both GAL4-based and a novel *Spm*-promoter-based single- and double-hybrid systems in transient plant cell assays.

TnpA was tested for the presence of a transcription activation domain using TnpA-GAL4 DNA binding domain fusions (Giniger et al., Cell 40:767, 1985). The effector plasmid pPHI 1210, containing a translational fusion of the yeast GAL4 binding domain to the strong VP16 activation domain (from Herpes Simplex virus; Triezenberg et al., Genes Dev. 2:718, 1988), and the reporter plasmid DP 1446, containing a transcription fusion of GAL4 binding sites upstream of a minimal CaMV 35S promoter to a firefly luciferase gene (both kindly provided by Ben Bowen and Brad Roth, Pioneer Hi-Bred International, Inc.), were used to produce most fusion constructs. The VP16 activation domain in plasmid pPHI 1210 was replaced with different domains of TnpA to test for transcription activation potential (Unger et al., Plant Cell 5:831, 1993). As a background control, the VP16 activation domain was deleted from GAL4. Effector and reporter plasmids were co-introduced into tobacco suspension cells by microprojectile bombardment, and the results are shown in Figure 1. Compared to bluescript and GAL4/VP16 antisense control plasmids (lanes 1 and 2, respectively), the GAL4/VP16 plasmid activates the luciferase reporter gene about 100-fold (lane 3).

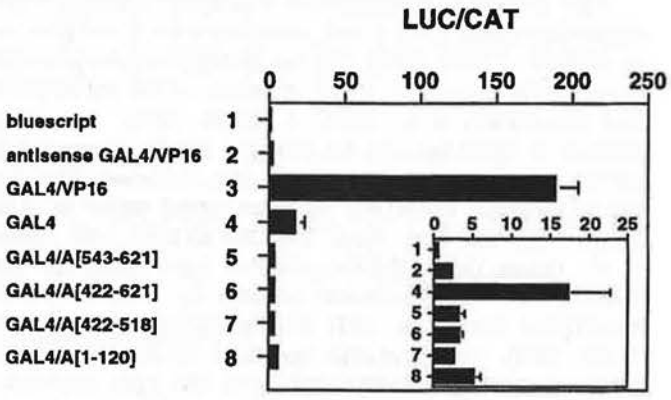

Figure 1. Analysis of putative TnpA transcription activation domains in transiently transfected tobacco cells. The strong transcription activation domain VP16 (from Herpes Simplex) in the effector plasmid pPHI 1210 was replaced with different TnpA domains from the N- or C-terminus to produce translational fusions with the GAL4 DNA-binding domain (from yeast). Each effector plasmid was co-bombarded individually into tobacco suspension cells together with the GAL4 binding site-containing luciferase (LUC) reporter plasmid DP 1446. To normalize for transfection efficiency, a chloramphenicol acetyltransferase (CAT) reporter plasmid was included, and relative promoter activity is expressed as the ratio of LUC to CAT activity. The LUC reporter plasmid is in each lane. The lanes are: Bluescript, pKS+ plasmid control (lane 1); antisense GAL4/VP16, antisense control (lane 2); GAL4/VP16, positive control (lane 3); GAL4, background control (lane 4); GAL4/A[543-621], a.a. 543 to 621of the C-terminus of TnpA fused to the GAL4 binding domain (lane 5); GAL4/A[422 -621], a.a. 422 to 621, the protein dimerization domain and the C-terminus of TnpA fused to the GAL4 binding domain (lane 6); GAL4/A[422 -518], a.a. 422 to 518, the protein dimerization domain of TnpA fused to the GAL4 binding domain (lane 7); GAL4/A[1-120], a.a. 1 to 120 of the N-terminus of TnpA fused to the GAL4 binding domain (lane 8).

The main results of the experiment were as follows:

(1) The *o2* gene of QPM is a monogenic recessive. It controls the accumulation of increased lysine in endosperms, tassels and leaves at the seedling stage. The modifier factors and genetic background affect the physical properties of maize endosperms.

(2) More asp, gly, lys, arg and less pro, glu, leu, phe were observed in the embryo as compared with the endosperm of QPM and normal. The same situation was also observed in endosperms of QPM as compared with the endosperm of normal (Tables 1 and 2).

(3) Protein contents between endosperms of QPM and endosperms of normal showed almost no difference (Table 3). The amino acid composition analysis indicates that the soluble proteins and glutelins are rich in lysine, while the zeins are almost devoid of lysine. The endosperm protein fractions of QPM show no more lysine than normal (Table 4). The endosperm of QPM contains more glutelin and less zein that normal.

(4) Twelve inbred lines of QPM, and a few heterotic hybrids of QPM, were developed.

Studies on superior new waxy, pop and semi-dent germplasm

--Mengqian Zeng, Yannan Liu, Taolan Yang and Songqing Ye

One waxy, one popcorn and three semi-dent germplasms were developed. These have been tested and used in hybrid production as new germplasm resources. Sixteen hybrids were developed. We bred these new germplasms by using inbreeding and dwarf selection from an improvement population which was bred by recurrent selection. Though each possessed its own characteristics and specific properties, all showed good disease resistance and high combining ability.

The results for disease resistance in all five new germplasms are shown in Table 1. Yinuo 303, JB, Zi24, MoA and Yi 508 all had good resistance to *Helminthosporium turcicum, H. maydis, Pythium aphanidermatum and Fusarium graminearum, Ustilago reiliana* and maize dwarf mosaic virus (MDMV).

The five new germplasms have different responses to different types of CMS (Table 2).

Table 1. The disease resistance of five new germplasms. HR = high resistance, MR = medium resistance, and R = resistance.

Germplasm	*H. turcicum*	*H. maydis*	*P. aphan. & F. gramin.*	*U. reiliana*	MDMV
Yinuo 303	HR	HR	R	R	MR
JB	HR	MR	R	R	MR
Zi24	HR	HR	R	R	MR
MoA	HR	HR	R	R	MR
Yi508	HR	HR	R	R	MR

Table 2. Fertile reaction of five germplasms to different CMS types. + = full restoration; - = maintenance.

	Cytoplasm		
Germplasm	T	S	C
Yinuo 303 waxy	-	-	-
JB popcorn	-	-	+
Zi24 semident	-	+	-
MoA semident	-	+	-
Yi508 semident	-	-	+

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Molecular analysis of *opaque2* alleles

--Hans Hartings, Nadia Lazzaroni, Vincenzo Rossi, and Mario Motto

Studies on genetic mutations that influence the accumulation of zein proteins have indicated the presence of several regulatory mechanisms controlling the expression of specific members of the zein multigene family. The *opaque2* (*o2*) gene is one of the most widely studied loci involved in these regulatory mechanisms. The *o2* mutation causes, in addition to a modification of endosperm appearance, a severe reduction (50-70%) in zein synthesis, leading to a concomitant enhancement of lysine content in the seed. A number of spontaneous *o2* mutations have been described in past years. We describe the molecular analysis of ten recessive (*o2*) alleles of independent origin [*o2-R, o2-m(r), o2-Columbian, o2-Agroceres, o2-261, o2-mh, o2-33, o2-Go2-Charentes, o2-Italian*, and *o2-Crow*].

Southern analysis performed on the *o2* alleles, with *Hin*dIII and *Eco*R1 endonucleases in combination with two molecular probes corresponding to the 5' and the 3' end of the *O2* cDNA respectively, allowed division of alleles into 6 polymorphic groups. Results are summarized in Table 1.

Table 1. Summary of Southern analyses (fragment size in kb).

o2 mutant	*Hin*dIII -probe1	*Eco*R1 -probe1	*Eco*R1 -probe2
wt normal	16.0	9.5	5.0
o2-R	12.0	7.5	5.0
o2-Italian	12.0	7.5	5.0
o2-m(r)	7.0	7.0	4.0
o2-Columbian	7.0	7.0	4.0
o2-Agroceres	7.0	7.0	4.0
o2-261	7.0	7.0	4.0
o2-mh	7.0	7.0	4.0
o2-33	16.0	7.0	12.0
o2-G	16.0	9.5	10.5
o2-Crow	16.0	9.5	5.0
o2-Charentes	17.0	5.5	10.5

Northern analysis revealed the presence of an *O2*-homologous transcript identical in size to the wild-type transcript for the *o2-R* and *o2-Crow* alleles. The cDNA of these two *o2* alleles was isolated and studied at the sequence level. Comparison of the cDNA sequence obtained for the *o2-R* allele with the published *O2* sequence revealed 17 base substitutions. Moreover, the *o2-R* sequence contained two insertions of, respectively, 2 and 7 nucleotides, 41 bp apart. Since no stop codons are present in this sequence stretch, these insertions merely give rise to a short frame-shifted stretch and consequent alteration of the deduced protein sequence in this region. Two deletions of 2 and 23 bases respectively in the central region of the *o2-R* sequence generate a second frame shift. This change in reading frame causes the premature termination of the coding sequence. As a consequence, the open reading frame of the *o2-R* gene is only 255 codons long,with a deduced polypeptide devoid of one acidic region and the basic and zipper domains. Comparison of the *o2-Crow* and wild-type *O2* cDNA sequences disclosed 27 base substitutions. In addition, the *o2-Crow* sequence contains five deletions which measure 3, 6, 120, 1, and 4 nucleotides with respect to wild-type. Immediately following the 120 bp deletion, a 24 bp stretch devoid of homology with the *O2* sequence is present in *o2-Crow*. Whilst

the first three deletions merely cause the omission of, respectively, 1, 2, and 40 residues from the deduced protein sequence, the fourth deletion generates a frame shift, which causes the premature termination of the *o2*-Crow polypeptide. As a consequence, the deduced *o2*-Crow protein is terminated after the basic domain.

The nucleotide changes observed between the *O2* and *o2-R* sequences and between the *O2* and *o2-Crow* sequences were used to estimate the average number of nucleotide substitutions per site according to Kimura's three substitution model. The average distance calculated for the *O2* and *o2-R* sequences is K = 0.0106, while *O2* and *o2-Crow* yield an average distance of K = 0.0186. Taking these data, and considering a neutral nucleotide substitution rate of 5×10^{-9} substitutions/site/year, the *o2-R* and *O2* alleles should have diverged from a common sequence approximately 1 million years ago. In a similar manner, *O2* and *o2-Crow* diverged approximately 1.86 million years ago.

Distribution of sequences related to the *Bg* transposable element of maize in *Zea*

--H. Hartings, N. Lazzaroni, V. Rossi, and M. Motto

The *Bg rbg* transposable element system was first detected when studying a case of somatic instability at the *opaque2* (*o2*) locus. It was demonstrated that the instability encountered was due to the presence of a receptor element (*rbg*) at the *o2* locus. The responsive recessive allele, called *o2-m(r)*, could revert somatically to wild-type in the presence of a regulatory element termed *Bg*. One striking feature of the *Bg rbg* transposable element system, reported by genetic analyses, is its apparent widespread occurrence in natural maize populations. A search for the presence of *Bg* elements, by their ability to induce instability of the *o2-m(r)* allele, revealed active autonomous elements in maize populations from distinct geographical areas (Montanelli et al., Mol. Gen. Genet. 197:209, 1984).

We have assessed thirty-four accessions from *Zea* for the presence of *Bg*. *Bg*-like sequences, identified as hybridizing bands on Southern blots, were visualized in all *Zea* accessions and were present in approximately equal numbers in teosinte and maize. Analysis of 16 inbred lines with the *Eco*RI and *Hin*dIII restriction enzymes using three molecular probes, covering 75% of the *Bg* transposable element, revealed an average of 8.7 ± 2.1 *Bg*-like sequences per maize genome. Teosinte accessions and maize races, analyzed with *Eco*R1 and *Hin*dIII in combination with a centrally located *Bg* probe, disclosed an average of 12.5 ± 4.3 and 10.4 ± 3.3 homologous sequences, respectively. An average of 9.5 $\pm$ 3.1 *Bg*-like sequences were present among the 34 *Zea* accessions analyzed.

All *Zea* accessions displayed a restriction fragment characteristic for a full-length *Bg* element upon cleavage with *Bst*EII. Southern analyses performed with methylation-sensitive enzymes demonstrated that these *Bg*-like sequences are methylated, although present in unmethylated DNA regions. Hence, it can be deduced that autonomous *Bg* elements are not uncommon among *Zea* accessions, while active elements are.

A comparison of the restriction patterns of related inbred lines revealed numerous common hybridizing fragments. An index of molecular similarity (MS), computed as the ratio of the number of bands common to two hybrids over the total band number for those two hybrids, was used to determine the degree of similarity between pairs of inbred lines.

MS values were computed for the LSC inbred lines H99, Lo924 (Mo17 x H99[2]) and Mo17. Considering the pedigree of Lo924, a higher similarity of this line with H99 than with Mo17 was expected. However, a comparison of the hybridization patterns reveals a high MS value (95.9) for Lo924 and Mo17. On the contrary, Lo924 and H99 exhibit an MS value of 58.8, while H99 and Mo17 reveal an MS value of 54.0. Comparison of the restriction patterns obtained for inbred lines C103, Va17, and Va22 (Va17 x C103[2]), revealed an intermediate pattern for Va22. In fact, Va22 and Va17 exhibit an MS of 82.6, a value only slightly higher than that calculated for Va22 and C103 (73.3), while C103 and Va17 reveal an MS value of 58.2. These MS data are in good agreement with earlier findings, which indicate the prominence of Mo17 germplasm in Lo924 despite its pedigree and a low association between Va22 and C103 (Ajmone-Marsan et al., Euphytica 60:139, 1992).

Analysis of nitrogen partitioning in IHP and ILP strains

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

The regulation of C and N supply to developing kernels by the vegetative portions of the plant is a subject of great interest because it is responsible for the accumulation of starch, protein and corresponding increase in dry weight of the kernels. Accordingly, in order to clarify the metabolic factors regulating the relative content of starch and protein in the seed we focused our attention on two strains, Illinois High Protein (IHP) and Illinois Low Protein (ILP), which differ greatly in their ability to accumulate proteins and starch in the endosperm.

Studies of IHP and ILP previously reported by our laboratory suggested that the accumulation of endosperm protein is affected by the amount of N substrates supplied to the kernel. We believe additional information about the processes involved in N metabolism will prove useful in elucidating the physiological bases involved in N utilization in maize plants.

Plants of IHP and ILP strains were grown under field conditions at Bergamo with different levels of N fertilizer (0, 75, and 150 Kg/ha N), to study the accumulation and the partitioning of N in different plant parts. At two dates, i.e. silking stage and black layer maturity (BLM), 3 whole plants (total above ground parts) from each of 4 replications from each N level, were separated into leaves, stalks, and ears, which were further divided into husks, cobs, and grains at the second harvest and analyzed for total N content. Plants of the IHP and ILP strains showed a different behavior in response to the increasing rate of N fertilizer application. In particular, it was noted that plants from the IHP strain had a higher N uptake and N redistribution efficiency than plants from the ILP strain. For the IHP strain, measurements show that increasing the rate of N fertilizer application is accompanied by an increase in N concentration in the whole plant at silking and by an increase of N concentration in the kernel at BLM. The ILP strain responded to N application only at the higher level of N application. Data at BLM revealed that for ILP plants the N concentration of the whole plant, with the exception of grain, was similar to the N concentration observed at the silking stage. These results suggested that the ILP kernel phenotype is apparently due to a reduced ability of N relocation from the plant to the developing grains.

A second field experiment was conducted to document the partitioning of ^{15}N applied to IHP and ILP strains, and to follow

the pattern of subsequent remobilization of this N during plant development. For each genotype 340 mg of labeled N (as $K^{15}NO_3$, 14.4 atom % excess) was applied to individual plants in the two central rows of each subplot.

Plants were harvested at 5 stages: 14 days before pollination (-14 DAP), at flowering (0 DAP), 14-28 DAP, and at BLM. The plants were divided into 8 parts; the ^{15}N concentration was determined with a mass spectrometer (Tracer Mass Europa Scientific) connected to the N-analyzer (Carlo Erba 1500-NA). During ear development the IHP strain showed a higher efficiency in relocating the ^{15}N to the ear than the ILP strain (Fig. 1). In the other plant parts during the grain filling period, a greater decrease of ^{15}N concentration was evident in the IHP strain than in ILP, particularly in the lower leaves (Fig. 2) and in the lower stalks (Fig. 3). The ILP strain showed a decrease in the percentage of ^{15}N distribution in the lower leaves until 42 DAP, in the lower stalks until 28 DAP, and a concomitant increase of ^{15}N in the ear until 28 DAP. These results suggest that the ILP phenotype depends on a lower ability of N remobilization during the late stage of grain development.

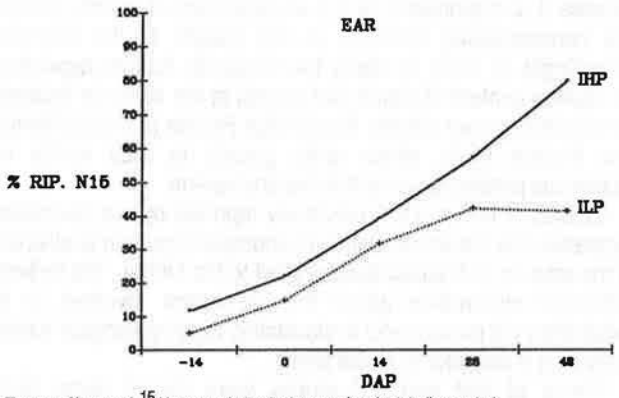

Figure 1. Change of ^{15}N accumulation in the ear after the labeling period.

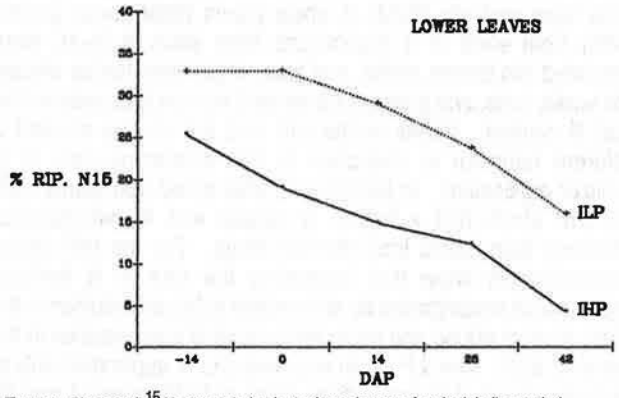

Figure 2. Change of ^{15}N accumulation in the lower leaves after the labeling period.

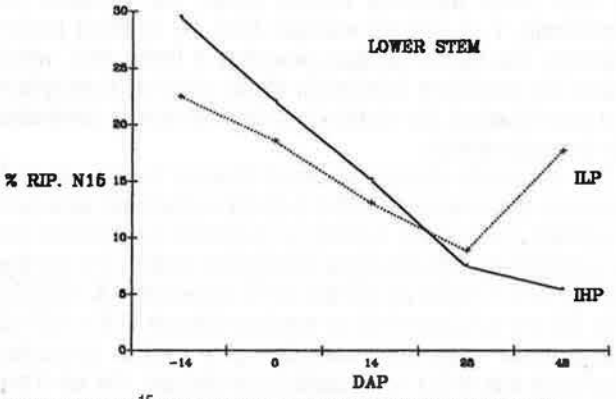

Figure 3. Change of ^{15}N accumulation in the lower stems after the labeling period.

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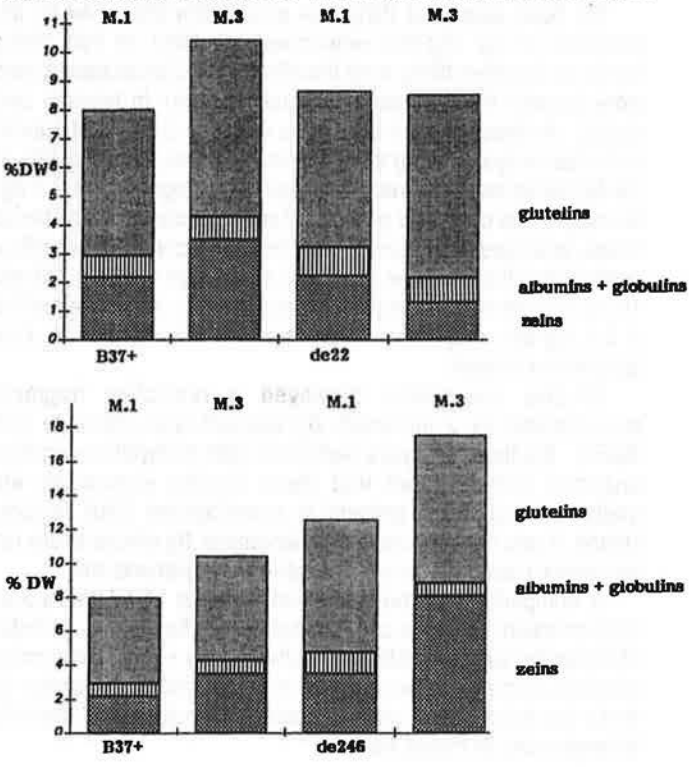
Genetic dissection of protein synthesis control in endosperm

--E. Rizzi, C. Balconi, D. Bosio, L. Nembrini, F. Salamini,* M. Motto and M. Maddaloni

With the aim of improving knowledge of protein synthesis control in endosperm and to define genic functions mediating the state of metabolism on transcriptional regulation of endosperm-specific genes, our collection of *defective endosperm* (*de*) mutants, introgressed into a B37 background, has been subjected to analyses. *de* endosperms, either grown *in vivo* or cultured on artificial media containing different levels of nitrogen (N), were fractionated for their protein content (zeins, albumins + globulins, glutelins and residue N). The data obtained indicate that differences exist between B37 and its *de* version and between *de*



Manzocchi et al. (Maydica 35:199, 1980), although some differences exist between the data reported in the cited paper and ours. Such discrepancies may reflect the different analytical methodologies adopted and/or the environmental variabilities among years. In order to standardize the conditions for growth and to evaluate the response of *de* mutants to different N nutrition, endosperms of B37+ and its *de* versions were grown *in vitro* on media containing different levels of N (media 1 and 3 as described in Balconi et al., The Plant Journal 3:325, 1991). Here we report the data only for B37, B37 *de*-246*, and B37 *de*-22*. As can be seen in the graphical representations, differences can be seen relative to the utilization of organic and inorganic N for the protein synthesis. At present we also have data indicating that, in addition to quantitative differences, the presence of qualitative differences are detected in zein and in albumin plus globulin fractions. Studies are in progress to define variations of starch and soluble sugars at quantitative and qualitative levels.

The transcriptional regulator OPAQUE2 and the nitrogen supply interact in regulating the overall protein synthesis in endosperm

--G. Donini, M. Maddaloni, C. Balconi, E. Rizzi, P. Gallusci*, F. Forlani, L. Stasse and M. Motto

The OPAQUE2 (O2) protein is a bZIP class transcription factor involved in the regulation of endosperm proteins including the 22-kD zein storage proteins. In this study we have focused our attention on the relationship between O2 and genes encoding enzymes presumed to have pivotal roles in nitrogen (N) metabolism. The genes tested were for glutamine synthetase 1-2 (GS1-2), acetolactate synthetase (ALS), and pyruvate ortophosphate dikinase (PPDK) which all proved to be sensitive to the presence of a functional *O2* allele. Particular attention has been given to the regulatory relationship between O2 and PPDK because the latter is suspected to be an enzyme that, in its cytosolic form, is responsible for carbon (C) partitioning between starch and proteins. Data were obtained indicating that O2 regulates the cyPPDK1 gene directly and specifically. The results support the hypothesis that O2, besides being an activator of 22 kD zein genes, plays a role in controlling the overall N metabolism, thus mimicking the role that the yeast transcriptional activator GCN4 plays in the general control. Finally, it is reported that the N supply to the endosperm interacts with O2, regulating the transcription of the genes for ALS, GS1-2, PPDK and O2 itself. On the basis of the data obtained, a model is presented in which two levels of control of endospermic protein synthesis are active: a first level of control exerted by *O2*, while a second level is driven by the overall state of the metabolism, possibly via regulators different from O2. Moreover, the two regulative mechanisms are interconnected in that O2 influences genes which are fluxing C and N compounds while the N supply influences the regulation of *O2* transcription.

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Enhanced protection against fungal attack by regulated expression of the b-32 gene in transgenic plants

--F. Forlani, L. Corazza, B. Balmas, G. Donini, G. Rotino, M. Motto and M. Maddaloni

Plants use a variety of defensive mechanisms against infections by insects, viruses, bacteria, and fungi. Several protective proteins and secondary metabolites are synthesized either constitutively or in an inducible fashion upon pathogen attack. Among these antimicrobial molecules, the ribosome inactivating proteins (RIPs) are produced sometimes in large amounts by species widely distributed throughout the plant kingdom. RIPs are severe inhibitors of eukaryotic ribosomes by enzymatic cleaving of a particular N-glycosyl bond in the 28S rRNA, so that the elongation factor 2 cannot properly bind to the ribosomes. RIPs are classified into two major classes. Type 1 ribosome-inactivating proteins, such as those of wheat and barley, consists of a single catalytic peptide (MW 30-32 kDa). Type 2 ribosome-inactivating proteins, such as ricin and abrin, are heterodimers composed of subunits A and B joined by a disulfide bond. The A chain is similar in size and enzymatic activity to type 1 RIPs. The B chain is a docking peptide promoting the transport of the holoprotein into the cell. The B chain increases the toxicity of type 2 RIPs to eukaryotic cells by a factor up to 10^6 in respect to type 1 RIPs.

In maize endosperm a cytosolic albumin with a molecular weight of 32 kDa, termed b-32, is synthesized in temporal and quantitative coordination with the deposition of storage proteins. The synthesis of b-32 is under the control of the regulatory genes *Opaque2* (*O2*) and *Opaque6* (*O6*), as the mutant alleles of these loci prevent its accumulation. While it has been clearly demonstrated that the protein product of the *O2* locus is a strong transcriptional activator of the b-32 gene promoter, the relation between the b-32 and the *O6* gene is not yet clarified.

In a previous paper we have noted that the DNA sequence of the b-32.66 cDNA is in part homologous to the protein-synthesis inhibitor II from dehusked barley grain (Lohmer et al., EMBO J. 10:617-624, 1991). Fifty-eight amino acids are conserved in a stretch of 173 residues. When the positions from amino acid 80 to amino acid 110 are considered, the identity reaches the value of 50%; of the remaining amino acid residues, 40% are similar.

We have produced transgenic tobacco plants with a construct (pwun-B32) expressing the b-32 gene under control of a promoter inducible by fungal attack. Some of the transgenics show enhanced resistance to *Rhizoctonia solani* AG4. The construct pwun-B32 was also used to transform *Solanum tuberosum* and *Solanum integrifolium* (wild aubergine). At present several kanamicin-resistant plants of both species have been obtained and we have preliminary results showing that the large majority of transgenic potato plants, under wounding conditions, produce a peptide immunoreactive to anti-b-32 antibodies.

Such a test has not been done for *S. integrifolium*. In the future we will challenge transgenic *S. tuberosum* and *S. integrifolium* plants with specific fungal strains to test if they have increased resistance as well.

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Fruitful EMS mutagenesis: search for suppressors of *lg1* and discovery of an *adh1* trans-acting mutant

--Lisa Harper, Barbara Kloeckener-Gruissem, and Michael Freeling

EMS mutagenesis of pollen, rather than of kernels, permits the recovery of high numbers of independent new mutations. We followed the method of Dr. Gerry Neuffer (Mutagenesis, The Maize Handbook, Freeling and Walbot, Eds, Springer-Verlag New York, 1994, pp. 212-219) to generate a mutagenized seed stock to search for second site suppressors of *lg1* and trans-acting mutants affecting *adh1* expression. We found that 0.1% to 0.2% EMS resulted in approximately 50% pollen death as monitored by germination on agarose media (see below for recipe). Germination of mutagenized pollen was monitored before and after actual pollinations. Germination on agarose media proved to correlate with seed set, and was an important step in predicting the success of the experiment.

Pollen from plants homozygous for *lg1-R* was EMS-treated and crossed to *lg1-R* testers. 6725 kernels on 242 ears were generated from 515 crosses. These M1 seeds were planted, observed, pollen tested, and self pollinated. Twenty-five kernels from each of 1520 M2 families were screened for *lg1* revertants. Of 5 families segregating 25% wild-type, no wild-type individuals were found to give 100% *lg1* progeny upon test crossing to *lg1*. Thus, a simple recessive suppressor was not found. We discovered that the *lg1* reference allele contains a large deletion; hence, a wild-type can possibly be explained by gene conversion, the reactivation of a pseudogene, the co-optation of a similar gene from another pathway, or contamination. A second suppressor screen is currently being set up with a partially functional *lg1* allele.

The spectrum of plant and kernel phenotypes found in the M2 screen was similar to what Neuffer has reported.

We screened a subset of M1 plants (4000) for deficient ADH1 enzyme activity in pollen, thereby avoiding the screening of 25 x 4000 M2 plants. Pollen was frozen, dialyzed, and stained by ethanol-dependent NBT reduction. Eight independent mutants were recovered which lack ADH1 enzyme activity. A cis-trans test was performed by crossing heterozygous M1 plants to a tester with an electrophoretically distinguishable *adh1* allele. Pollen from progeny was tested for ADH1 enzyme activity by *in situ* and starch gel staining. As expected, the *in situ* staining showed that the pollen of half of the progeny segregated 1:1 for ADH1 activity and no activity. If the new mutation acts in cis, plants heterozygous for *adh1* deficiency are expected to show only the tester allele homodimer on starch gel staining. If the new mutation acts in trans, those pollen samples are expected to show both the tester and mutant ADH1 homodimers. According to this cis-trans test, one of us (BKG) recovered the first *adh1* trans-acting mutant and 5 new cis-acting mutants. The test has not yet been completed on two of the new mutants.

While screening for the absence of ADH1 activity we were also able to score partial enzyme activity. In this initial screen, approximately 6% of the M1 plants produced pollen segregating 1:1 for various levels of reduced activity. In addition, we discovered that 4.6% of all M1 plants generated pollen that segregated 1:1 for small pollen grains, which occasionally correlated with changes in the ADH1 phenotype. Pollen abortion was found in 2.8% of M1s.

Although cloning of EMS induced alleles is not easy, the mutation frequency is at least one order of magnitude higher than that of *Mutator*-induced mutants in our lab (our standard *Mu*-active line, called mum9, has about 5 *MuDR* elements). This high frequency, and the easy monitoring of mutagenized pollen death to predict the success of the experiment, makes EMS an especially good mutagen for two types of experiments: the recovery of low-activity alleles, and the identification of new classes of mutants such as suppressors. As shown for *adh1*, partial phenotypes are observed quite frequently (6%) which most likely reflects the nature of the molecular lesion. With such low-activity alleles, new genes with multiple functions can be discovered.

The pollen germination medium was essential in this experiment. This recipe was modified by Steve Modena and Ed Coe from: F.S. Cook and D.B. Walden, Can. J. Botany 43:779, 1965.

To prepare the plates:
(Note: This medium is not sterile. Empirically, autoclaved OR FILTER-STERILIZED sucrose in this media results in very low pollen germination.)

1. Make three solutions: A: 3.0g $CaCl_2 \cdot 2H_2O$ in 100 ml; B: 1.0g H_3BO_3 in 100 ml; C. 30g sucrose in 100 ml.
2. Add 0.7g agarose to 50 ml water.
3. Heat to a boil in the microwave to melt the agarose.
4. Transfer to a cold stirrer and add: 1 ml solution A; 1 ml solution B; 50 ml solution C.
5. Quickly pipette 8-10 mls to each of 10 petri plates.
6. Let cool with lids cocked half off.
7. Replace lids when cool. Plates can be stored inverted at room temp for a few days or at 4C for a month or so. Before using plates, make sure there is absolutely no water condensation on them. This will instantly burst pollen.

To use the plates:

1. Sprinkle fresh pollen, or brush pollen in oil onto room temperature plates.
2. Allow at least 15 minutes for the pollen to begin germination. For monitoring death over time, pollen can be applied at appropriate time points, and scored all at once some time later. Plates can even be left at room temp overnight and scored the next day.
3. Observe through a dissecting scope at 20X. Plate can be illuminated from below on a clear background or from above on a black background.
4. If desired, 10 ml of 50:30:10:10 sucrose:water:EtOH:HOAc can be applied to the plates after one hour. This will kill and fix the pollen, allowing clear viewing of the field due to removal of refraction from the meniscus surrounding each grain.

Multiple-ligule (*Mlg*-1*) is probably an allele of *lg3*

--Lisa Harper, Mike Scanlon, and Michael Freeling

Multiple-ligule (*Mlg*-1*) is a dominant mutant that arose in a *Mutator* field. The initial phenotype was a ligule composed of multiple rows of ligule giving a "fuzzy" appearance. After 4 generations of introgression into Mo17, B73, and W23, it has 100% penetrance and is uniformly expressive. The phenotype is different in the different inbreds, and the ligule is not as multiplied as it was before introgression. In Mo17, the ligule over

the midrib is usually not continuous, but where it is present, it occurs in multiple "tufts". These tufts also appear randomly for several inches distal into the midrib region. The leaf also appears to have sheath extending into the blade at the midrib in much the same way as *Lg3-O*. The blade is bent outward at a greater angle than wild-type. The auricle and ligule on either side of this broadened midrib appear to be normal. This is in contrast to the phenotype in B73, where, in addition to the normal ligule, there are longitudinal ligule rows following lateral veins in the auricle only. In W23, the ligule is usually continuous and multiple around the midrib.

Mlg-1* has been localized by T-*wx* translocation mapping. *Mlg*-1* heterozygotes were crosses to a T-*wx* series and *Mlg* progeny were testcrossed to *wx*. *Mlg*-1* was found to be linked to both 3S and 3L translocations:

F1 Genotype	Wx-Mlg (P)	Wx-mlg (R)	wx-Mlg (R)	wx-mlg (P)
wx T3-9(8447)/ *Mlg*	92	45	19	133
wx T3-9(8562)/ *Mlg*	102	31	7	128

DNA was made from 10 *Mlg/mlg* and 10 *mlg/mlg* sibs. A polymorphism was found when samples were restricted with *Hin*dIII, and blots probed with a DNA fragment specific to *lg3*. No recombinants were found between the *Mlg* phenotype and the RFLP, suggesting close linkage, and probable allelism between *lg3* and "*mlg*".

Mapping the *narrow sheath* duplicate factor pair

--Mike Scanlon and Michael Freeling

The narrow leaf and plant stature mutant *narrow sheath* (*ns*) was identified by Elsing and Albertsen as a single factor trait which sometimes displayed non-Mendelian patterns of inheritance (MNL 66:49-50, 1992). Subsequent analyses have determined that, in the original inbred line in which the mutant was isolated, the *ns* phenotype is inherited as a single recessive factor trait (Table 1). However, in the inbred lines Mo17 and B73, and in our laboratory *Mutator*-active inbred stock, the *ns* trait displays duplicate factor inheritance (Table 2). In order to locate the *ns* duplicate factor pair to chromosome arm using the B-A translocation series (Kindiger and Beckett, MNL 60:43, 1986), plants heterozygous for each of the dual factors were crossed as

Table 1. Number of progeny obtained from self of normal plants generated from *ns* x *ns* / + cross in original stock obtained from Pioneer Hi-Bred International.

Self #	Mutant No.	Normal No.	Total No..	Mutants (%)
1	24	62	86	27.9
2	14	40	54	25.9
3	7	29	36	19.4
4	48	150	198	24.2
5	8	25	33	24.2
6	12	21	33	36.4
7	22	78	100	22.2
Sum total	135	406	541	**24.9**

Fits a 3:1 ratio

Table 2. Combined sum total of number of progeny obtained from selfs of *ns*/Mo17 and *ns*/B73 plants.

F1 genotype	Mutant	Normal	Total	Mutants (%)
ns/Mo17	66	849	915	7.21
ns/B73	68	1153	1221	5.57
Combined sum total	134	2002	2136	**6.27**

$X^2 = 0.001$ Fits a 15:1 ratio

Table 3. Summary of *ns1* and *ns2* TB mapping results

TB parent	Hypoploid F1 plants (#)*	F1 cross	Total *ns* progeny (#)	Total progeny (#)	*ns* progeny (%)
1S	3	*ns* backcross	8	28	28.5
1L	2	*ns* backcross	26	111	23.4
3S	6	*ns* backcross	93	210	**45.7**
3L	8	*ns* backcross	97	410	23.7
4S	2	self	7	114	6.14
4S	2	*ns* backcross	13	81	16.3
4L	3	*ns* backcross	52	105	**49.5**
5S	9	*ns* backcross	93	409	22.7
5L	3	*ns* backcross	33	154	21.4
6S	2	*ns* backcross	13	70	18.5
6L	6	*ns* backcross	59	275	21.5
7S	3	*ns* backcross	35	157	22.3
7L	7	*ns* backcross	55	227	24.2
8L	3	*ns* backcross	38	174	21.8
9S	2	self	6	100	6.0
9S	5	*ns* backcross	29	146	19.9
9L	1	*ns* backcross	6	30	20.0
10S	3	*ns* backcross	35	160	21.9
10L	4	*ns* backcross	52	249	20.9

* only hypoploids which segregated *ns* mutant progeny included in table

female onto hyperploid heterozygotes. Hypoploid plants were selected among the F1 progeny on the basis of 50% pollen semi-sterility, and plant phenotype (where applicable). The F1 hypoploids were either self-pollinated or backcrossed to *ns* mutants. The ratios of *ns* vs. non-mutant seedlings scored in the progeny of these crosses are presented in Table 3. The data indicate that the *ns* duplicate factors are located on chromosome arms 3S (*ns1*) and 4L (*ns2*). Molecular markers are now being used to RFLP map the *ns* loci to more precise locations.

The identification of a *liguleless2* genomic clone

--Justine Walsh and Michael Freeling

lg2 mutants are recessive and map to 3L-101. As homozygotes they remove ligule and auricle in the lower leaves of the plant. In subsequent leaves, ligule and auricle begin to appear at the leaf margins and, in later leaves, spread toward the midvein. The ligule and auricle are also often displaced. Thus, this mutation appears to affect the blade-sheath boundary as well as the formation of ligule and auricle. Mosaic analysis has shown that the *Lg2+* product acts non-cell-autonomously (Harper, MNL 67:17, 1993).

lg2-R (reference) arose spontaneously and was described by Brink (J. Hered. 24:325, 1933). Seven putative *Mutator*-induced alleles have been recovered in this lab. *lg2-2757* appeared in a screen of families of selfed *Mutator*-active plants. *lg2-902* was recovered from a directed *Mutator* tag. We were able to increase the number of alleles to seven by identifying five more *lg2* alleles (*lg2-219, -228, -229.1, -229.2* and *-278*) in a directed *Mutator* tag completed this past summer.

We found a *Mu8* homologous element that cosegregated with the *lg2-2757* allele. The element was present in 43 *lg2-2757/lg2-R* individuals and absent in 36 *+/lg2-R* individuals. Using a *Mu8* probe, a 5.5 kb *Eco*R1 genomic clone was identified from a bacteriophage library (Fig. 1). A 1.2 kb *Eco*R1/*Bam*HI probe, representing non-*Mu8* DNA, was used to probe genomic

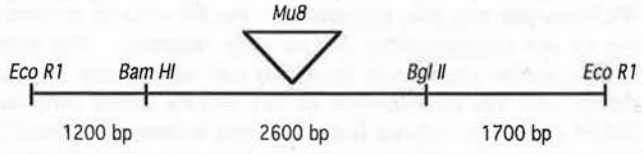

Fig 1: Restriction map of 5.5 kb *EcoR1 liguleless 2* genomic clone.

DNA cut with an enzyme that does not cut in *Mu8*. In *lg2-2757* individuals, this probe hybridizes to the same restriction fragment as *Mu8*.

Genomic DNA of the other six putative *Mutator*-induced alleles and their respective progenitor alleles was also hybridized with the *Eco*R1/*Bam*HI probe. DNA differences were detected between the new *lg2* alleles and their progenitor wildtype alleles. Initial results show that *lg2-902, -228, -229.1, -229.2* and *-278* carry some form of rearrangement or insertion and that *lg2-219* is a deletion; thus, *lg2* is cloned.

New alleles of *Rough sheath1*

--Richard Schneeberger and Michael Freeling

Rs1-0 is a dominant neomorph which causes severe developmental abnormalities of the ligule, sheath and blade due to ectopic expression of the *Rs1* homeodomain protein in the leaf (Genetics 136:295). We report the description of three new probable *Rs1* alleles, *Rs*- mu64*, *Rs*-muC3* and *Rs*-mu1429* in addition to one new *Rough sheath* mutation, *Rs*-mu173* , that is unlinked to the *Rs1* locus. All four mutants were originally identified in our *Mutator* selfing blocks and in unrelated directed tagging experiments. Each new isolate was crossed into B73, W23 and Mo17 inbred lines for three generations to obtain good expression and penetrance. All four isolates express well in the B73 background and to a lesser extent in W23 and Mo17. All four mutants closely resemble the *Rs1-O* phenotype, with expression seen on most leaves starting on the third to fifth leaf and terminating near and sometimes including the flag leaf.

Table 1. Linkage data for four *Rs** mutations.

F1	Total Observed	Normal (No.)	Recombination (%)
*Rs1- Z4 /Rs-*muC3*	190	1	0.5±0.5
*Rs1-Z4 /Rs- *mu64*	398	1	0.25±0.25
*Rs1-Z4 /Rs-*mu1429*	198	0	0
*Rs1-Z4 /Rs-*mu173*	249	114	46±3

In order to determine if these mutations are linked to the *Rs1* locus the new isolates were crossed to a *Rs1* tester and the resultant double mutants outcrossed to normal plants. The tester was a homozygous *Rs1-Z4* B73 inbred stock which expresses *Rs1* phenotype at the seedling stage. Double mutants (e.g., *Rs1-Z4/ /Rs*-muC3*) were selected in the F1 progeny and outcrossed as females by B73. The F2 progeny were scored for *Rs1* and normal phenotypes. The data shown in Table 1 indicate strong linkage of *Rs*-muC3*, *Rs*-mu64* , and *Rs*-mu1429* to *Rs1-Z4.* The cross to *Rs*-mu173* showed normal segregants in the F2, indicating that this mutation is unlinked to the *Rs1* locus. RFLP analysis using the cloned *rs1* gene was performed on 10 mutant and 10 normal individuals from families segregating 1:1 for each of the linked mutations to provide further evidence that the linked *Rs** mutations are alleles of the *Rs1* gene. In each case a *rs1* RFLP was identified that absolutely cosegregated with the mutant phenotype in five enzyme surveys (data not shown). A similar RFLP analysis was also performed on the *Rs*-mu173* mutation and no *rs1* cosegregating RFLPs were detected. The tight linkage, similar phenotypes (including lack of a mutant dosage effect), and the identification of *rs1* RFLPs linked with the mutant phenotpye indicate that these new isolates are probably alleles of *Rs1*. Therefore we will refer to them as *Rs1-C3, Rs1-64*, and *Rs1-1429* . We are currently mapping the *Rs*-mu173* mutation to determine if it represents a dominant mutation of another member of the maize homeobox gene family.

Four point linkage data for *o2, v5, Rs4* and *gl1*

--Richard Schneeberger and Michael Freeling

Rough sheath4 (*Rs4*) is an EMS-induced, dominant mutation which produces abnormally large vascular bundles in the leaf sheath (Neuffer, MNL 64:51, 1990). The gene was initially placed near the centromere of chromosome 7 with waxy reciprocal translocations (Neuffer, MNL 64:51, 1990). We report the results of four point linkage mapping to further define the position of *Rough sheath4.* A standard test cross was used to generate the mapping population as indicated in Table 1.

Table 1. Four point linkage data for *o2 v5 Rs4 gl1*.

F1: *+ + Rs4 +/o2 v5 + gl1*

		Recombinants						
	Par.	R1	R2	R3	R1+2	R2+3	R1+3	R1+2+3
	174	10	18	4	0	5	2	0
	203	5	17	3	1	2	0	0
Totals	377	15	35	7	1	7	2	0
% recombination			Region 1: 4.1± 1.0					
			Region 2: 9.9± 1.4					
			Region 3: 3.6± 0.9					

Approximately 480 kernels were separated into opaque and translucent classes and planted in our San Jose nursery. Seedlings were screened for the *gl1* phenotype 4 weeks after planting. The *virescent5* and *Rough sheath4* phenotypes were scored at the time of tassel emergence. The data are consistent with the T-*wx* mapping data and place *Rs4* approximately 10 map units from *v5* and 3.6 map units proximal to *gl1* on chromosome 7L very close to *ra1*. The map positions obtained for *o2*, *v5* and *gl1* are consistent with the current locations except that the *o2* to *v5* distance obtained in this experiment is 4 map units compared to 8 on the most current MNL map. The suggested map revision is:

```
---o2----------v5------------------Rs4---gl1---
  20           24                  33    36
```

Map locations of Liguleless*-347 and Liguleless*-9167

--Gary J. Muehlbauer, Mike Scanlon and Michael Freeling

Liguleless-347* (*Lg*-347*) is a dominant mutation recovered in a *Mutator* field by Steve Briggs at Pioneer Hi-Bred International Inc. This mutation causes the ligular and basal blade regions near the midrib to be sheath-like, suggesting a transformation of auricle and blade into sheath. Using the *waxy* reciprocal translocation stocks we mapped *Lg*-347* to translocations in the short and long arm of chromosome 3 (Table). DNA gel blot analysis of a segregating *Lg*-347* family hybridized to a *liguleless3* (*lg3*) probe (chromosome 3S location; Chi et al., MNL 68:16, 1994) showed no recombinants, indicating that *Lg*-347* is located on 3S linked to and possibly allelic to *lg3*.

Liguleless-9167* (*Lg*-9167*) is a spontaneous dominant mutation recovered by Drew Schwartz at the University of Indiana. This mutation also causes the ligular and basal blade regions near the midrib to be sheath-like, suggesting a transformation of auricle and blade into sheath. Using the *waxy* reciprocal translocation stocks we mapped *Lg*-9167* to a translocation in the long arm of chromosome 2 (Table). The map location of this mutation is on the same chromosome arm as *Gnarly1*

(*Gn1*; Foster and Hake, MNL 68:2, 1994), indicating that *Lg*-9167* may be allelic to *Gn1*.

F1 genotype	*Wx-Lg* (P)	*Wx-lg* (R)	*wx-Lg* (R)	*wx-lg* (P)
Lg-347* / T3-9(8447) *wx*	106	7	15	108
Lg-347* / T3-9(8562) *wx*	60	14	3	89
Lg-9167* / T2-9d *wx*	158	55	38	158

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Cytotoxic effects of two herbicides on meiosis

--M. Kumar, M. Prasad, H. Kumar and S.K.T. Nasar

Two commonly used herbicides, Pendimethaline and Oxyfluorfen, were evaluated for their cytotoxicity in cv. Suwan. Seed soak treatments were given for 6 hr and 12 hr with aqueous solutions of these herbicides with concentrations 5, 25 and 50 ppm. Seeds were then thoroughly washed, and planted in the field. Germination percentage was recorded. Young emerging tassels were fixed at flowering stage in aceto-alcohol fixative for 48 hr and then standard aceto-carmine squash preparations of anthers were prepared to score the frequency of dividing cells and various abnormalities (Table 1).

Marked inhibition of germination was observed in the case of both herbicide treatments. Dose-dependent inhibition of meiosis as indicated by decline in meiotic index was observed. Pendimethaline was more injurious to cell division in terms of frequency of dividing cells (95.8% - 87.1%) and abnormalities (4.2% - 12.9%) than Oxyfluorfen, which showed a higher meiotic index (97.5% - 93.2%) and less abnormality (2.5% - 6.7%). Pollen sterility also was much higher in the case of Pendimethaline treatment (1.29% - 7.32%) than Oxyfluorfen (0.57% - 3.16%). Pendimethaline treatment induced stickiness, multivalents and unequal separation of chromosomes, while Oxyfluorfen induced only laggards and stickiness.

Table 1. Cytotoxic effects (in percent) of herbicides in pollen mother cells.

Herbicide	Conc. ppm.	Tmt. (h)	Germ-ination	Meiotic index	Abnormal PMCs	Pollen sterility	Meiosis I	Meiosis II	Meta-I	Meta-II
Control	0	-	97.0	99.6	0.4	0.54	56.8	42.8	14.5	15.3
Oxyfluorfen	5	6	95.0	97.5	2.5	0.57	67.0	30.5	22.5	18.0
		12	80.0	97.0	3.0	2.20	69.8	27.2	28.6	14.9
	25	6	90.0	96.5	3.5	1.63	63.5	33.0	27.5	22.9
		12	65.0	94.9	5.1	2.5	61.7	33.2	29.0	20.3
	50	6	54.5	95.3	4.7	2.10	65.3	30.0	28.8	12.9
		12	36.4	93.2	6.7	3.16	84.7	8.5	48.5	1.2
Pendimethaline	5	6	95.2	95.8	4.2	1.29	68.5	27.3	31.5	15.5
		12	80.9	93.9	6.3	1.47	67.0	26.7	25.8	13.7
	25	6	85.0	91.7	8.3	2.19	66.8	24.9	35.0	15.5
		12	56.5	91.3	8.7	5.42	65.0	26.3	32.1	15.6
	50	6	51.0	90.3	9.7	6.12	64.0	26.3	32.8	17.2
		12	35.0	87.1	12.9	7.32	62.5	24.7	26.0	15.7

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A "β-glucosidase aggregating factor" (BGAF) is present in "null" genotypes

--Neval H. Erturk and Asim Esen

Maize ß-glucosidase catalyzes the hydrolysis of DIMBOA-glucoside, an abundant physiological substrate whose aglycone is believed to be involved in the defense of young plant parts against pests. The enzyme occurs as large aggregates in some maize inbreds. Such inbreds were originally thought to have a null allele at the enzyme locus. However the immunological and biochemical studies in our laboratory showed that these inbreds have enzyme activity and an immunoreactive enzyme monomer. We have recently discovered that "null" genotypes have a factor, ß-glucosidase aggregating factor (BGAF), which causes the aggregation and poor solubility of the enzyme in such genotypes. The existence and activity of BGAF were demonstrated as follows: (1) Equal amounts of 4- to 5-day-old frozen, etiolated shoots from a normal (K55) and a "null" (H95) genotype were mixed, homogenized, and extracted together and the enzyme activity in the supernatant was assayed. It was observed that activity in the supernatant fluid of these mixed extractions was decreased to a level barely detectable in spectrophotometric assays (data not shown), instead of being equal to the arithmetic mean of the H95 and K55 extracts made separately. (2) K55 and H95 shoots were homogenized separately and the homogenates were mixed in different ratios and extracted together, and assayed for enzyme activity. The results showed that the enzyme activity in supernatants decreased linearly as the amount of the H95 homogenate in the mixture increased (Fig. 1). (3) Shoot extracts of K55 and H95 were mixed and then assayed; the activity in the mixture was the arithmetic mean of that found in each of the two extracts. However, when the mixtures were incubated overnight and the activity was assayed in supernatants after the centrifugation, a reduction in enzyme activity was observed as the amount of the H95 extract in the mixture increased (Fig. 2). Moreover, the activity lost from the supernatant upon centrifugation was recovered quantitatively from the pellet by extraction with a buffer containing 0.5% SDS (Fig. 2). (4) The pellets of the H95 ("null") homogenates were suspended with the K55 supernatant of known activity level and incubated for 30 min., and activity was assayed in the K55 supernatant before and after incubation. Data showed that enzyme activity in the K55 supernatant decreased as the amount of the H95 pellet used for incubation increased. All these data suggest that a substance (i.e.

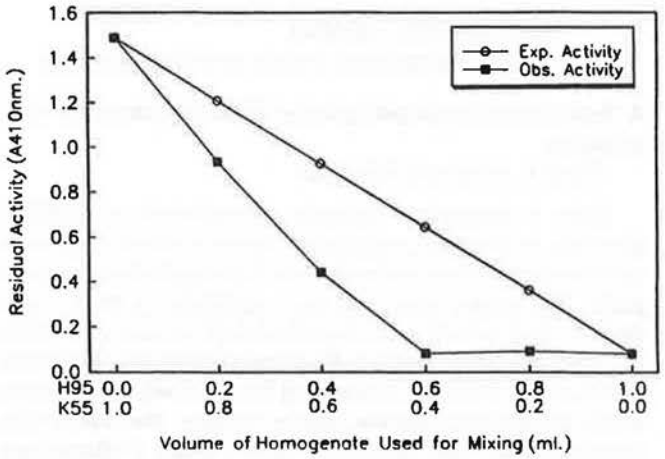

Fig. 1. β-glucosidase activity in supernatant after mixing homogenates of K55 (normal) and H95 ("null") genotypes. In all cases (including Figs. 2-4) enzyme activity was assayed after dilution of extracts 30 to 50 times in 50 mM citrate-100 mM phosphate buffer, pH 5.5. Seventy µl of the diluted enzyme was incubated with 70 µl of 5 mM p-nitrophenyl-β-D-glucose (pNPG) in wells of a microtiter plate at room temperature (24-25°C) for 5 mins. The reaction was stopped with 70 µl 0.4 M sodium carbonate and the absorbance of the pNP produced was measured at 410 nm in a microtiter plate reader. All assays were performed in quadruduplicate. The date shown in each graph were from assays done under identical conditions. Abbreviations. exp., expected; obs., observed; cent., centrifugation; spec., specific.

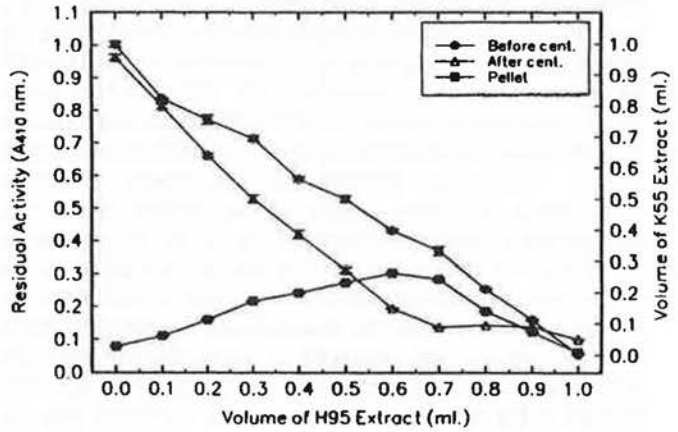

Fig. 2. β-glucosidase activity in mixtures K55 (normal) and H95 ("null") extracts

BGAF) is present in "null" inbreds and it interacts with ß-glucosidase and causes its aggregation. BGAF is present both in the supernatant and pellet fractions of homogenates from "null" genotypes.

In order to study the effect of pH on the extractability and activity of ß-glucosidase and BGAF, freeze-dried K55 (normal) and H95 ("null") whole shoot powders were extracted in separate tubes four times with the following buffers of indicated pH: sodium citrate (pH 3), sodium acetate (pH 4 and 5), MES (pH 6), HEPES (pH 7), Tris-HCl (pH 8), CHES (pH 9), and sodium carbonate-bicarbonate (pH 10 and 11). All extractions were made on ice for 30 min each. Four extracts of each genotype made with the same buffer were saved separately for assays. Because the first and second extracts contained about 90-95% of the total extractable activity at a given pH, they were pooled (1:1 volume ratio) and reassayed for enzyme activity and protein so that the activity extracted at each pH could also be expressed as specific activity. The results (Figs. 3-4) show that the amount of enzyme and protein extractable increased with pH, being lowest or negligible at pH 3 and highest at pH 11, increasing in the pH range 4 to 11. When the highest total extractable activity at pH 11 is

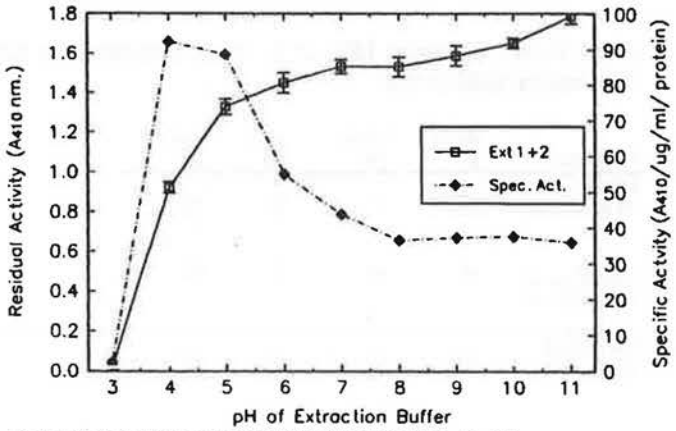

Fig. 3. Effect of pH on extractability of β-glucosidase (K55, normal)

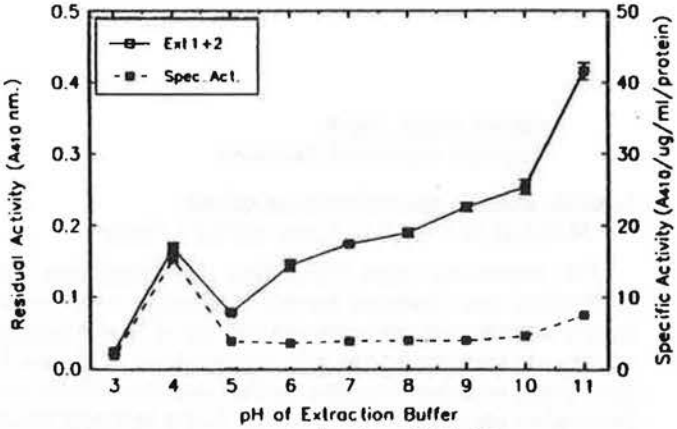

Fig. 4. Effect of pH on extractability of β-glucosidase (H95, "null")

set as 100%, relative extractabilities were 52% at pH 4 and increased from 74% at pH 6 to 93% at pH 10 in the case of K55 (Fig. 3). However, in terms of specific activity (expressed here as A410 units or the absorbance of pNP produced/mg protein), the pH 4 and 5 extracts had the highest activities (92 and 89 units, respectively) and decreased from 55 at pH 6 to 36 at pH 11 in the case of the K55 extracts (Fig. 3). As for the H95 ('null') extracts, the following striking differences were observed in comparison to those of K55: (1) The amount of total extractable ß-glucosidase activity but not protein was about 3 to 20 times lower, depending on pH, resulting in drastic decreases (3.5 to 15 units/mg protein) in specific activities (Fig. 4). (2) The pool of the first two extracts contained a lower percentage (80 and 67%, respectively) of the total extractable activity while the corresponding values ranged from 97 to 89% in the pH range 4 to 9 in K55 (Fig. 3). (3) Most surprisingly, extractability of the enzyme increased with pH only in the pH range 7 to 11 (Fig. 4). Relative extractabilities in the pH range 7 to 10 increased from 42 to 61%, in stark contrast to the corresponding values of 86 to 93% obtained for K55 in the same pH range. The real surprise was the decrease of relative extractability to 19% at pH 5 and to 35% at pH 6 after 40% at pH 4 (Fig. 4). The most plausible interpretation of these results is that BGAF is solely responsible for the differences between K55 and H95. It appears that BGAF activity is highest at and around pH 5, and it decreases as pH increases from 7 to 11. It seems that at pH 10 to 11, either BGAF is gradually solubilized and extracted or inactivated as exposure to high pH continues, as would be the case with repeated extractions. Studies focusing on isolation and further characterization of BGAF are in progress.

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An interesting case of inhibition of leaf development by X-irradiation

--M. M. Johri and Ed Coe

While tracing the progression of internode differentiation after germination, an interesting case of irradiation damage was observed. Seeds heterozygous for anthocyanin (*b, pl*) and chlorophyll markers (*wd*) were germinated in peat pots and allowed to develop for 8 and 13 days after sowing (DAS). The entire seedling was x-rayed (1,500 R at the meristem level) and promptly planted in the field. Between 60 and 80% of the seedlings survived and developed into mature plants which, although morphologically similar in size and node number to unirradiated plants, showed some radiation-induced damage. The culm diameter and Apparent Cell Number (ACN) were reduced in the 7th and 8th internodes. These observations suggest that irradiation alters the fitness of cells. Those losing dominant markers seem to proliferate more rapidly than the neighboring cells (NB) and consequently give rise to wider sectors. At the same internode the NB cells seem to be less fit to divide and expand, otherwise it is difficult to explain reduced culm diameter.

A single instance of a leaf primordium failing to develop into the leaf was found. Among 82 plants developing from seedlings irradiated 13 DAS, one appeared to have missed the 12th leaf and two consecutive leaves were present on the same side of the plant (Fig. 1A). An examination of the internode above the 11th node showed the presence of a protuberance at the site corresponding to the midrib of the 12th leaf (Fig. 1B). Whether the protuberance represents the aborted midrib or the axillary bud related to node 13 is difficult to decide. Since a sector for the loss of *Pl* extended from the protuberance basally to node 11, it is likely to represent an aborted midrib. It appears the leaf primordium just after initiation is very susceptible to radiation damage. The absence of leaf and axillary bud was accompanied by a reduction of internode length. The length of the 12th internode was reduced by 33% and of the 13th by 66%. It is possible that a developing leaf or an axillary bud provides hormonal factors for the elongation of adjacent internodes.

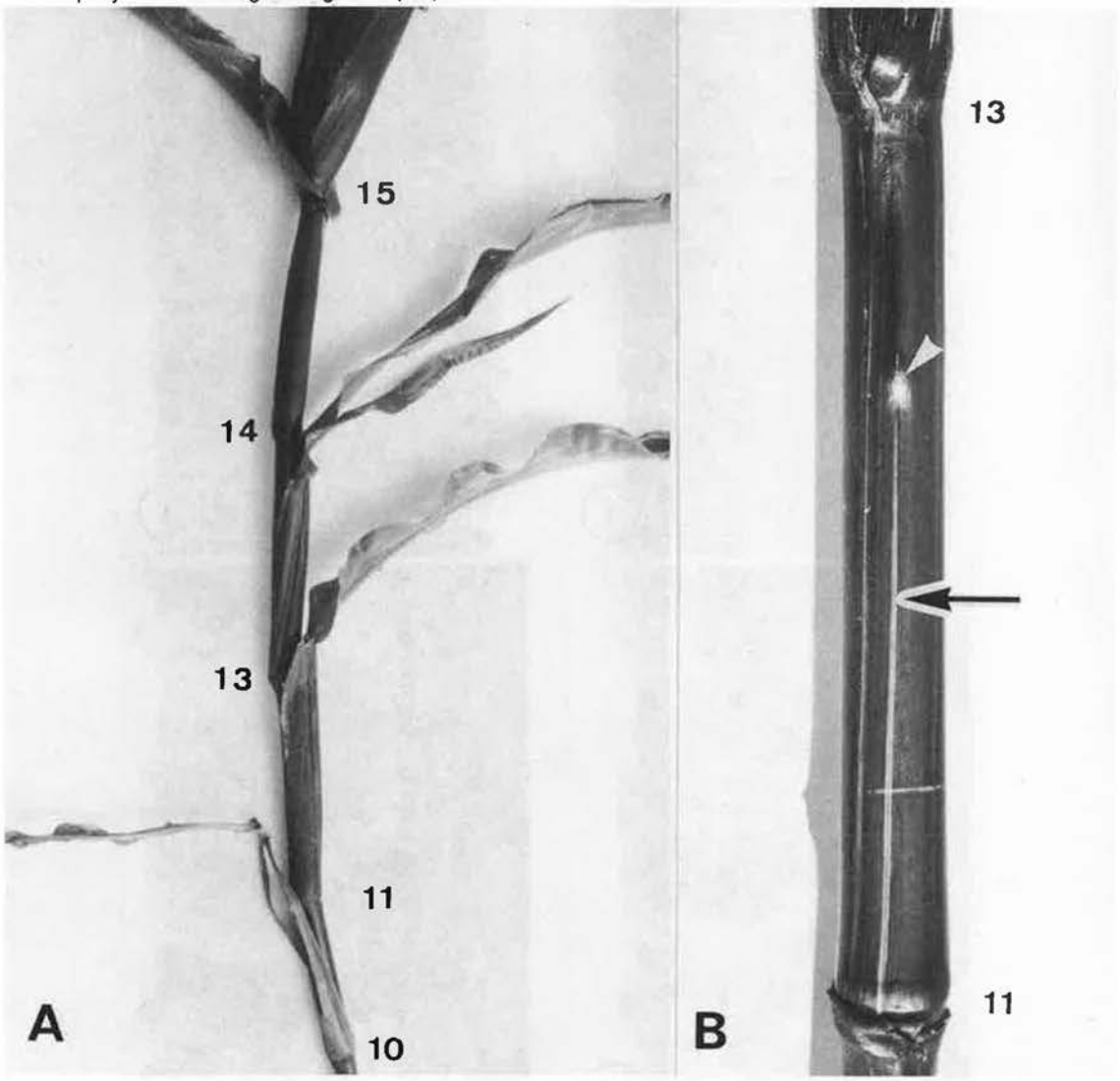

Fig. 1. A. Nodes 10-15 in a plant from a seedling irradiated 13 DAS. Note the two consecutive leaves on the same side of the plant. B. Removal of leaves shows the presence of a protuberance (white pointer) at the location of 12th leaf. A sector for the loss of *Pl* (black arrow) extends from the protuberance to node 11. Numbers 10-15 refer to respective nodes.

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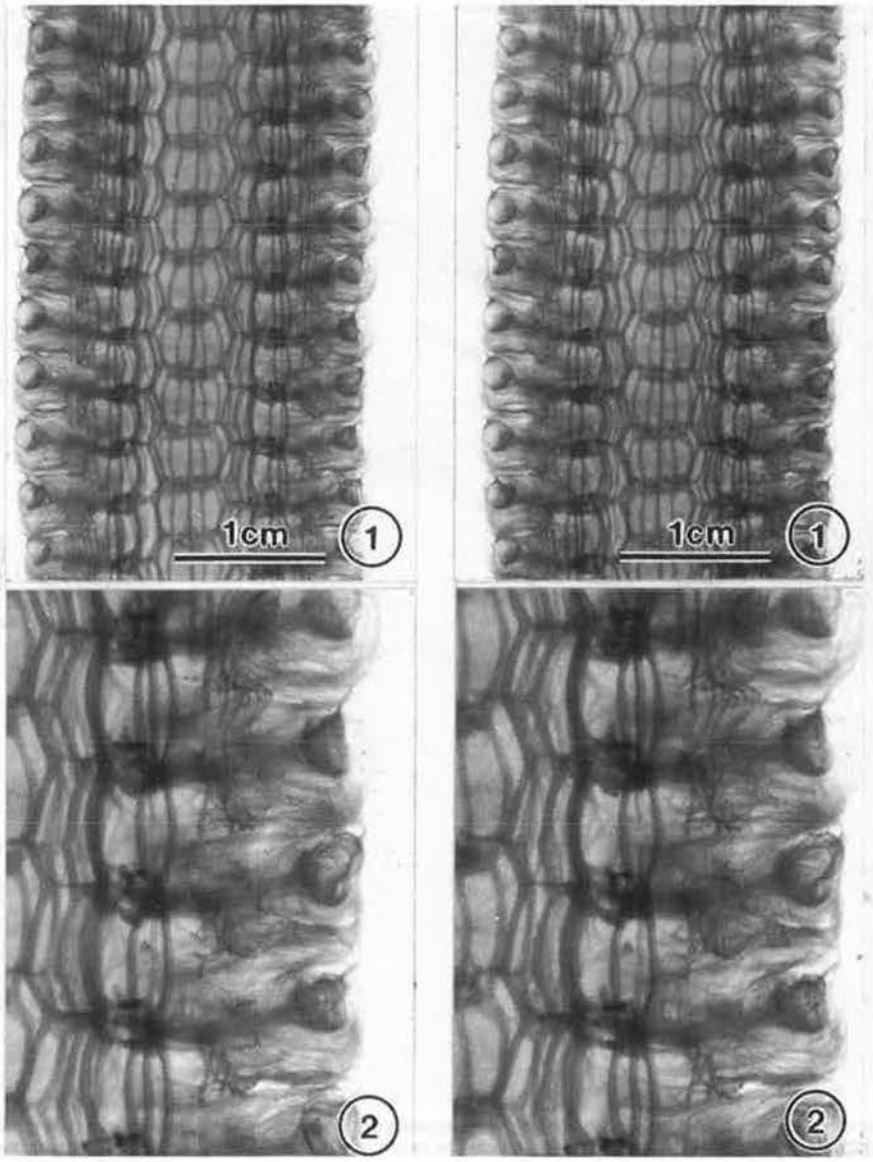
A method for studying vascular bundles in 3D
--Ping-chin Cheng

To study the arrangement of vascular bundles in a chunky specimen is a difficult task; frequently it relies on time consuming reconstruction of serial sections. Here we report a method which can be used for stereoscopic viewing of vascular bundles in bulky specimens (as large as a longitudinally bisected ear). The specimen preparation method described here can also be used for confocal microscopy.

The procedure involves fixing tissues in a 3:1 EtOH/acetic acid fixative. The tissue is left in the fixative until it is completely decolored. For a large piece of tissue, it may be necessary to make a few changes of fresh fixative. This process may take days to weeks. Then the tissue is degassed in an air-tight glass jar filled with gas-depleted water; degassed water is obtained by cooling down boiling water in a completely filled bottle capped with an air-tight cover. This degas step is essential for tissues which contain trapped air bubbles. For example, mature maize stem generally has a whitish appearance as a result of strong light scattering from the large number of air pockets. By immersing the air-containing tissue in a jar of degassed water, the trapped air can be slowly dissolved away, and finally the tissue is completely waterlogged. This process is best to be carried out in a closed jar which is topped to its rim with gas-depleted water in a 4C refrigerator. Although this degas process can be carried out at room temperature, the lower temperature enables a larger amount of gases to be dissolved in the bathing water, and hence is faster in removing the trapped air bubbles from the tissue. A small crystal of thymol should be added to the bathing water to prevent the possible growth of mold or bacteria.

When the tissue is completely free of air bubbles, the tissue is then soaked in a 0.5% (w/v) sodium metabisulfite solution. The length of this treatment ranges from an hour to several days depending on the size of the tissue. Then the tissue is transferred to a diluted Schiff reagent (prepared by adding 0.1g basic fuchsin to 85ml water with 1.9g sodium metabisulfite and 15ml 1N HCL; shaken for two hours and then decolorized with 200mg activated charcoal) at 4C, the staining time ranging from an hour to several days. It is important to use a jar with an air-tight cover



to avoid the loss of SO2 from the solution. For large pieces of tissue, a standard canning jar works well. After staining, the tissue is differentiated in a sodium metabisulfite solution (0.5% w/v) for many changes. Complete removal of unbound dye at this step is essential to ensure the final preparation has the lowest background staining. Then the tissue is dehydrated through an EtOH series followed by several changes of absolute EtOH, 1:1 EtOH/methyl salicylate and finally cleared in several changes of pure methyl salicylate. To ensure a high degree of transparency in the final preparation, complete removal of tissue water by absolute EtOH is essential. The result is a highly transparent specimen with vascular bundles stained in purple.

The stereogram (a stereo-image pair) is one of the most commonly used methods to present 3D structures. A stereogram can be obtained from a specimen by taking photographs of a specimen at two different viewing angles which simulate the appropriate parallax of the human eyes. On the other hand, a stereogram can be generated by a computer from a volumetric data set obtained by a laser scanning confocal microscope or tomographic system. The angle between the two images of a stereogram can be determined by a simple formula (Hudson and Makin, J. Phy. E. Sci. Inst., 3: 311, 1970). To photograph the specimen, a panchromatic film such as Kodak T-max 100 can be used. A green filter (Tiffen 58) will provide excellent contrast for a thin specimen (such as a leaf blade). However, for a bulky specimen (e.g. longitudinal section of an ear or a stem) where background staining is relatively high, the use of a red filter (Kodak Wratten 25 or Tiffen 25) is recommended. The specimen should be completely immersed in methyl salicylate in a flat-bottom glass container. An evenly illuminated light-box can be used as the light source. For microscopic tissues, confocal microscopy operated in epi-fluorescent mode using the 488nm Ar laser line as the excitation wavelength is ideal. A 550nm long-pass filter is used for the fluorescent detector. Serially cut optical sections obtained by the confocal microscope can then be used for generating the stereogram in a computer. For better stereo perception, it is important to avoid orienting fiber-like structures (such as vascular bundles in a stem) parallel to the base of the stereogram. Figure 1 shows a stereogram of a longitudinal slice of an ear inflorescence (var. Golden Beauty). Figure 2 is a higher magnification view showing vascular bundles associated with spikelets.

[Ed. note: With the original prints, I have been able to 'experience' a stereo image by crossed-eyes tripling and concentration on the middle image.]

In addition to simple stereoscopic viewing, the specimen preparation method mentioned above opens up possibilities of using optical tomography and multidimensional image analysis for true 3D reconstruction and interactive visualization of the vascular bundles on a computer. This work was supported by a grant from the National Science Council of the Republic of China (NSC 83-0211-B-001-001) during the author's sabbatical leave at the Institute of Botany, Academia Sinica, Taipei.

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Somaclonal variation-induced mutation in an aluminum tolerant inbred line

--Sergio T. Sibov, Marilia Gaspar, David H. Moon, Marcio J. da Silva, Laura M. M. Ottoboni and Anete P. de Souza

Somaclonal variation-induced multiple mutations were observed in progenies of an R0 plant regenerated from type I calli of the aluminum tolerant inbred line Cat-100-6. After five generations of self pollination, 14 progenies were tested for aluminum (Al) sensitivity in a nutrient solution containing 6 mg/l of Al. All of the 14 somaclones tested were sensitive to Al as defined by root tip morphology and relative growth values. The most Al-sensitive progeny, S1587-17, was selected for further study.

To study the effect of aluminum on Cat-100-6 and S1587-17 roots, seeds were germinated at 30 C between layers of moist filter paper. After 3 days, the primary root lengths were measured and the seedlings were transferred to polystyrene holders, which were then floated on an appropriate volume of an aerated nutrient solution. Aluminum, in the form of $AlK_3(SO_4)_3$, was added to the nutrient solution at a concentration of 6 mg/l of Al and seedlings were removed at various exposure times (0, 1, 2, 4, and 6 days). Fig. 1 shows the progressive destruction of normal root tip morphology in the sensitive plant with time of exposure to

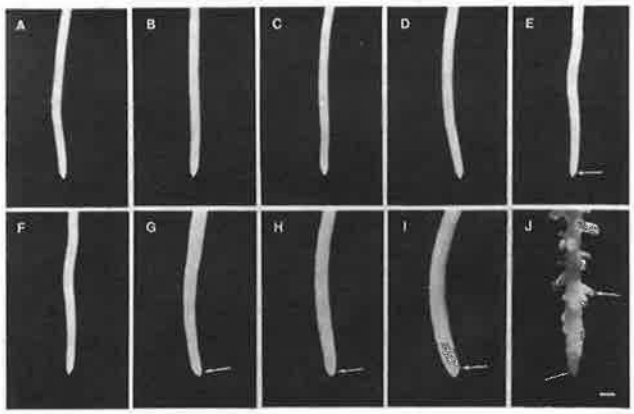

Figure 1. Effects of Al, after 0, 1, 2, 4, 6 d exposure, on the roots of tolerant Cat-100-6 (A to E) and sensitive S1587-17 (F to J) plants. Plants were grown in nutrient solution in the presence of 6 mg/l Al. The arrow in pictures G to J indicates the progressive destruction of the root tip, and in J the appearance of lateral root meristems.

Table 1. Segregation of aluminum tolerant (*Alm-*) and aluminum sensitive (*alm alm*) plants among F2 and BC progenies and their recurrent parents.

	Genotype			
Progenies	*Alm-*	*alm alm*	Total	X^{2a}
Parents				
Cat-100-6	44	0	44	
S1587-17	0	48	48	
F1				
Cat-100-6	56	0	56	
F2	174	56	230	0.051NS
BC1				
Cat-100-6 x (Cat-100-6xS1587-17)	60	0	60	
BC2				
(Cat-100-6xS1587-17) x S1587-17	47	45	92	0.43 NS
S1587-17x (Cat-100-6xS1587-17)	72	64	136	0.47 NS

[a] X^2 analysis based on (#) 3:1 or 1:1 segregation frequency.

Al (Fig. 1F-J) and the lack of effect on the tolerant plant root tip (Fig. 1A-E). After 1 day of exposure to Al, the sensitive root tip (Fig. 1G) showed signs of swelling and by 2 days the root tip was clearly swollen (Fig. 1H). The root tip structure was drastically changed after 4 days of exposure (Fig. 1I); after 6 days (Fig. 1J) the root tip was completely lost leaving a blunt swollen end and lateral root growth had commenced very close to the root apex. In contrast, the tolerant root tip showed no symptoms of Al toxicity over the same time period (Fig. 1A-E).

The genetics of Al tolerance was investigated in a F2 population obtained from Cat-100-6 x S1587-17 and a BC2 population obtained by crossing the F1 with S1587-17. The analysis indicated that the Al tolerance of Cat-100-6 is determined by a single dominant gene, as tolerant and sensitive plants segregate in typical Mendelian ratios (Table 1). [ed. note: The symbol *alm1* is applied to this locus in place of *al*, as posed by the author, inasmuch as *al* refers to albescent; *als* and *alt* are also previously assigned.]

The effect of phytotoxic levels of aluminum on sensitive and tolerant primary roots

--David H. Moon, Marcio J. da Silva, Anete P. de Souza and Laura M. M. Ottoboni

Seeds from the aluminum tolerant inbred line Cat-100-6 and from the aluminum sensitive somaclonal variant S1587-17 were germinated at 30°C and after 3 days were transferred to an aerated nutrient solution containing aluminum in the form of $AlK_3(SO_4)_3$ at a concentration of 6 mg/l. Seedlings were removed at different exposure times (0, 1, 2, 4 and 6 days) and the primary roots were fixed and embedded in paraffin. For comparison, 10 mm root sections were taken from the sensitive and tolerant plants and stained with toluidine blue and hematoxylin.

Histological sections (Fig. 1) show the effect of aluminum at the tissue level. At 0 days, both tolerant (Fig. 1A) and sensitive (Fig. 1F) plants presented normal morphology, with the tolerant plants continuing to present normal root apex morphology after 6 days exposure to aluminum (Fig. 1B-E). Figs. 1D and 1E show abscission of cap cells from the tolerant root apex, which is indicative of actively growing roots. After 1 day of exposure to aluminum the sensitive plant root (Fig. 1G) showed an increase in cap volume; after 2 days of exposure the epidermis and the cortex were severely damaged, and the root cap cells exhibited grossly swollen cells (Fig. 1H); after 4 days the peripheral cap and central cap were completely destroyed and only the cap meristem, although severely damaged, could be observed (Fig. 1I). After 6 days of aluminum exposure (Fig. 1J) all components of the meristematic zone were completely destroyed, and secondary root meristems appeared in the elongation region.

Aluminum infiltration in the roots was detected by hematoxylin staining of the primary root sections. The sensitive plant showed a gradual accumulation of aluminum within the root tissue with time. After 1 day of exposure the staining was confined to the root exterior; after 2 days the nuclei of the root cells were stained, indicating the presence of aluminum even within the nucleus of the affected cells; after 4 and 6 days of exposure there was a more intense staining within the root indicating massive infiltration of aluminum into the root tissue. The tolerant root sections showed very little staining after 1 day and minimal staining over the rest of the time period.

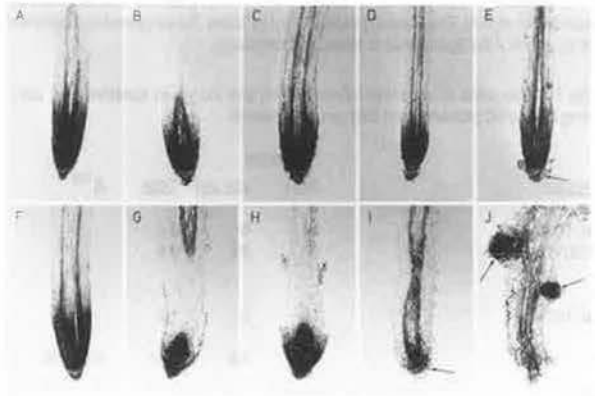

Figure 1. Toluidine blue stained maize primary roots sectioned through the elongation zone from tolerant Cat-100-6 (A to E) and sensitive S1587-17 (F to J) plants. Controls without Al (A and F); 6 mg/l Al for 1 day (B and G); 6 mg/l Al for 2 days (C and H); 6 mg/l Al for 4 days (D and I); 6 mg/l Al for 6 days (E and J). The arrow in (E) indicates cap cells abscission, a signal of actively growing root; in (I) indicates complete destruction of root cap and in (J), the appearance of lateral root meristems after disruption of root cap. Sections were photographed with bright field illumination with a magnification factor of x80.

Analysis of somaclonal variation-induced zein mutation

--Marilia Gaspar, Sergio T. Sibov, Anete P. de Souza and Laura M. M. Ottoboni

The occurrence of somaclonal variation in plants regenerated from calli established from inbred line Cat-100-6 was observed. One out over two thousand R0 regenerated plants, designated somaclone S1587, presented increased growth rate, tillering and high proliferation capacity. R1 to R5 progenies, obtained by self pollination of S1587, presented a high mutational frequency, giving rise to endosperm mutants, defective seedling and variegated leaves. Most of the mutations were determined by single recessive genes and presented unstable Mendelian segregation along the successive self pollinated generations. However, some mutations were stable.

Molecular analysis of somaclone S1587 was carried out for one of its progeny, designated S1587-17, for the zein genes. Zein proteins were extracted from S1587-17 and Cat-100-6 endosperms and submitted to isoelectric focusing (IEF) analysis. The IEF gels showed that several alterations have occurred in the somaclone S1587-17 zein profile. Since each IEF band corresponds to a single structural gene, it can be concluded that at least 6 zein genes (for proteins Z1, Z2, Z3, Z4, Z5 and Z6) were altered in the somaclone. 2D equivalent analysis of the different zein IEF bands of the two genotypes indicated that five corresponded to the 22 kDa zeins and one to the 19 kDa zeins. To investigate the segregation of the 22 kDa and 19 kDa zein genes, zein was extracted from 112 F2 (Cat-100-6xS1587-17) seeds, and

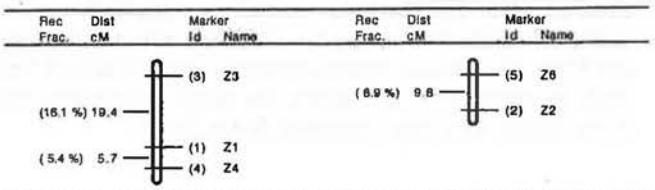

Figure 1. Zein proteins from one hundred twelve F2 seeds were analyzed by IEF in order to detect linkage between the Mendelian segregating zein polypeptides: Z1, Z2, Z3, Z4 and Z6. Linkage was determined, using Mapmaker (Lander et al., Genomics 1:174-181, 1987) version 2.0, on an Apple Macintosh computer. Markers were initially assigned to groups using the group command, and loci were mapped with the "three-point" and compare command. A minimum LOD of 3.0 and a maximum recombination frequency of 0.4 was used to verify the linkage orders. The genes for Z2 and Z6 are linked and mapped far away from Z3, Z1 and Z4.

subjected to IEF. Preliminary results indicated that five of the altered zein genes were segregating in a typical Mendelian ratio. Linkage analysis of the segregating zein genes indicated that they are grouped in two clusters (Fig. 1). Southern analysis of the S1587-17 and Cat-100-6 genomic DNA digested with *Eco*RI using a 22 kDa and a 19 kDa zein cDNA as probes revealed, as expected, several alterations in the organization of the zein genes, with the disappearance, appearance and amplification of bands in the somaclone. Since 5 out of the 6 altered zein bands in the somaclone belonged to the 22 kDa zein genes, and considering that these gene are mainly located on maize chromosome four, experiments using RFLP probes from chromosome four are underway to confirm the localization of the affected zein genes.

Phenotypic analysis of the latente gene in a segregating F2 population

--Anete P. de Souza, Joel I. Fahl, Claudia T. Guimarães, Laura M. M. Ottoboni, Waldenar Naspolini Filho, Joaquim A. Machado and Paulo Arruda

Michoacan maize presents elevated drought resistance. Under water stress Michoacan plants reduce growth rate, which is restored quickly in the presence of water. This behavior is genetically determined and was designated latente (Miranda et al., MNL 56: 28-30, 1982). The latente gene (*lte1*) was previously mapped on maize chromosome 2S, 11-49 region (Miranda et al., MNL 56: 28-30, 1982). The phenotype used for mapping was based on physiological studies of plants presenting the latente character. Latente plants present a more efficient mechanism for stomatal closure resulting in a reduced loss of water. This parameter can be measured in the field by using a porometer.

In order to study the effect of the latente characteristic in tropical maize, Michoacan plants were crossed to tropical commercial inbred lines. The segregating population was submitted to water stress conditions and the vapor diffusion resistance (VDR) was measured. A total of 73 F2 plants were analyzed. During the water stress period the F2 plants showed a retardation in their development with respect to the control. VDR of individual F2 and control plants was measured. The absolute VDR values for each F2 plant were subtracted from the VDR value of control plants. Approximately 80% of the F2 plants presented higher VDR values when compared to the control plants. In contrast to the preliminary genetic studies, where latente was considered a monogenic trait (Miranda et al., MNL 56: 28-30, 1982; Miranda et al., MNL 65: 45, 1991), our F2 data presented normal distribution, suggesting a typical quantitative trait. Probably, this normal distribution is due in part to great environmental influence over the phenotype.

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Studies on perennial maize

--Y.C. Ting

In the spring of this year, I received a package of maize kernels of both diploid and tetraploid varieties from Dr. D.L. Shaver, Cornnuts, Inc., Salinas, CA. I was very excited about these acquisitions because I have tried several times without success to transform annual maize into perennial through teosinte introgression. The failure to achieve my goal was partly due to the severe cold in the Boston area. Now the gift from Dr. Shaver gave me another chance to do experiments with maize perennialism. Table 1 depicts the results.

Table 1. Characteristics of perennial maize.

Characteristic	2n	4n
Earliness in germination	10 days	7 days
Tiller proliferation	7.6(6)*	2.4 (10)*
Ovule fertility	82% (381)**	95% (535)**
Anthocyanin gene (*b1*)	No	Yes
Ga gene	No	Yes
Propagule regeneration	No	20% (10)*

*Numbers in parenthesis indicate total number of plants tested.
**Numbers in parenthesis indicate number of ovules counted.

The kernels of the two varieties of perennial maize were sown in pots in the greenhouse in early January. It took seven days for the tetraploids to germinate and 10 days for the diploids. When the plants were about 10 weeks old, with well-developed tillers, their main stalks (culms) were cut off at a point above the soil surface. After cutting, the tillers continued to grow vigorously. As soon as the plants had reached 20 weeks old, they were transplanted to the field; this was in late May. Ten tetraploids and six diploids were maintained for observations. However, no new tillers of these plants generated in the field.

On average, the number of tillers per plant varied in diploids *vs.* tetraploids when counts were made at the 25-week stage. There were eight tillers for diploids, and only two for tetraploids. Since tillering is a required characteristic for maize perennialism, attention was focused on it. In addition, ovule fertility of these plants was also examined. It was found that 95 percent of the ovules of tetraploids set well-developed kernels upon selfing, while only 82 percent of the ovules of diploids produced kernels.

Regarding synthesis of anthocyanin pigments, the tetraploids were heterozygous for the *b1* gene located on chromosome 2. They segregated into purple and green plants in the selfed progeny, while the diploids did not. In order to have triploid perennial maize, the above two varieties were hybridized with the tetraploids as female parent. Nineteen ears of the tetraploids were pollinated with pollen from three different diploids. Only three ears bore kernels. Five presumptive triploid kernels were obtained. It seems that the tetraploids might possess a gametophyte factor in chromosome 4, but not the diploids.

Another required character for maize perennialism is the growth or regeneration of propagules into tillers. This was also investigated in the greenhouse. The procedure was to decapitate the seedlings at various stages of growth. Results showed that when decapitations were applied to the four-week-old plants, no growth of the propagules was observed, even though a drop of cytobeinin, 6 BAP (6-benzyl-aminopurine, 5 mg/l), was dripped on the surface of cuttings. However, when the plants were six weeks old, 20 percent of the decapitated plants of tetraploids grew into plantlets using the same treatment (details are not provided here). In contrast, no response was found for the diploid plants. On average, 10 plants were employed in this test for each of the two ploidy levels.

Before frost came, all the above healthy field-grown plants were moved to the greenhouse. If their growth, or regeneration of propagules, is continued next year, it would prove that the perennialism is present in these plants.

Perennial maize is of great importance in crop improvement and production. If its basic genetics, particularly at the molecular

level, is fully explored, new directions in maize farming will be established in the near future. For instance, like sugar cane, once it is planted, continued harvests can be reaped for several years.

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Does P protein require a partner, as C1 protein does?
--Erich Grotewold

The *P* gene encodes a myb-domain protein, P, required for the transcriptional regulation of a subset of the flavonoid biosynthetic genes regulated by C1 (Grotewold et al., Cell 76:543, 1994), including *C2*, *Chi1* and *A1*. C1, however, does not regulate transcription by itself and requires a member of the *R* or *B* gene families (encoding proteins with bHLH domains) for its activity. In agreement with the genetic evidence, P does not require R protein for the regulation of *A1*. Either P protein alone, or C1 protein (in the presence of R/B) are able to activate efficiently a minimal 35S promoter containing the high-affinity P binding sites identified in the *A1* promoter, when transiently expressed in embryonic callus or BMS cells (Grotewold et al., 1994). In addition to these P-binding sites, other *A1*-promoter elements are required for normal regulation of *A1* by P and by C1 proteins (Grotewold et al., 1994; Tuerck and Fromm, Plant Cell 6:1655, 1994), yet those promoter elements are not recognized by P in vitro.

P and C1/Pl1 share over 83% identity in their Myb-domains, the region responsible for the interaction between C1 and R (Goff et al., Genes Deve. 6:864, 1992). Does P require a partner, as C1 does? I decided to approach this problem by investigating the possibility that P would function in a heterologous system, and for that purpose I have chosen yeast. I introduced a reporter *LacZ* gene downstream of a dimeric high-affinity P-binding site (with a *cyc* TATA box) into the yeast chromosome. When P was expressed in yeast cells carrying this reporter construct, a very efficient activation of the reporter was obtained (over 130-fold). There was no activation when the plasmid carrying the P cDNA was absent (1-fold), nor when the P-binding sites were replaced by a dimer of a sequence to which P does not bind in vitro.

These observations are a strong indication that P is sufficient to activate transcription from promoters carrying high-affinity P-binding sites. These results are not in contradiction with the observation that other promoter elements are important for the regulation by P in plant cells. Yet, they suggest that for promoters carrying high-affinity P-binding sites, there is no need to invoke a P partner.

Not all P-regulated genes have P-binding sites. In fact, I haven't been able to identify sequences to which P binds in vitro in the promoter of the *Chi1* gene, which is dependent on the presence of P for its expression in the pericarp (Grotewold and Peterson, Mol. Gen. Genet. 242:1, 1994). This could possibly mean that P regulates this promoter through a different mechanism, which could be similar to the mechanism by which *A1* is regulated in the absence of high-affinity P-binding sites (Grotewold et al., 1994; Tuerck and Fromm, 1994). These alternative ways of regulation by P could involve the existence of accessory factors that recruit P to promoter elements, or that change the DNA-binding specificity of P.

The lojap (*lj*) protein is associated with 50S chloroplast ribosomal subunits
--Chang-deok Han and Rob Martienssen

Plants homozygous for the *iojap* (*ij*) mutation have patterned white stripes on their leaves (Jenkins, J.Hered. 15:467-472, 1924), and give rise to albino seedlings in their maternal progeny when pollinated by wild-type plants (Rhoades, Cold Spring Harbor Symp. 11:202-207, 1946). The degree and extent of striping, and the maternal transmission of the *ij*-affected plastids, are largely dependent on genetic background (Jenkins, 1924; Coe, Thompson and Walbot, Am. J. Bot 75:634-644, 1988). The *lj* gene was cloned by transposon tagging, but the sequence of the IJ protein showed no homology with proteins in current databases (Han , Coe and Martienssen, EMBO J. 11:4037-4046, 1992). To further characterize the role of the IJ protein in chloroplast and leaf development, antibodies were raised against recombinant IJ fusion proteins. Western blotting and immunolocalization have revealed that IJ is a chloroplast protein found in most cell types of immature leaves (not shown).

On the basis of ribosomal RNA profiles of mutant and wild-type plants, Walbot and Coe (Proc. Natl. Acad. Sci. 76:2760-2764, 1979) proposed that *iojap* plants suffered a programmed loss of plastid ribosomes that could account for the maternal transmission of *ij*-affected plastids. We therefore analyzed plastid subfractions to determine whether IJ was associated with plastid ribosomes or other macromolecular structures. In chloroplast lysates, most of the protein was found in high speed pellets, but it was found in the soluble fraction in the presence of 20mM EDTA. This suggested that the IJ protein was associated with an EDTA-sensitive complex. Whole seedling lysates were further fractionated by pelleting through a sucrose cushion, followed by sucrose gradient sedimentation in the presence of non-ionic detergents (polysome gradients). Sedimentation profiles were determined by uv absorption (not shown) and by western blotting (Fig. 1) using anti-IJ and anti-50S ribosome antibodies (from R. Mache). The IJ protein was found in the same fractions as the 50S ribosomal proteins under these conditions (Fig. 1).

Direct association of IJ with 50S subunits was examined by immunoprecipitation with affinity purified anti-IJ antibodies. Immunoprecipitates were fractionated by SDS PAGE and western blots were probed sequentially with anti-IJ, anti-50S ribosomal proteins and control anti-LHCPII (light-harvesting complex membrane protein) antisera (Figure 2). IJ protein was quantitatively precipitated by increasing amounts of anti-IJ antibody (first panel). 50S ribosomal proteins were also specifically recovered in the anti-IJ immuno-precipitates (second panel, I.P.), although most of the proteins remained in solution (second panel sup.). LHCPII proteins were not recovered in the precipitates (third panel), demonstrating that the co-immunoprecipitation was specific, and did not reflect contamination with residual membrane-bound ribosomes. We are currently determining which ribosomal proteins are specifically bound to IJ.

Association with 50S ribosomal subunits suggests a role for the IJ protein in the regulation of plastid translation, and supports the model first proposed by Walbot and Coe for the programmed loss of plastid ribosomes in ij mutants: Some ribosomal proteins are encoded by plastid genes, so that loss of plastid translation due to homozygous nuclear mutation could lead

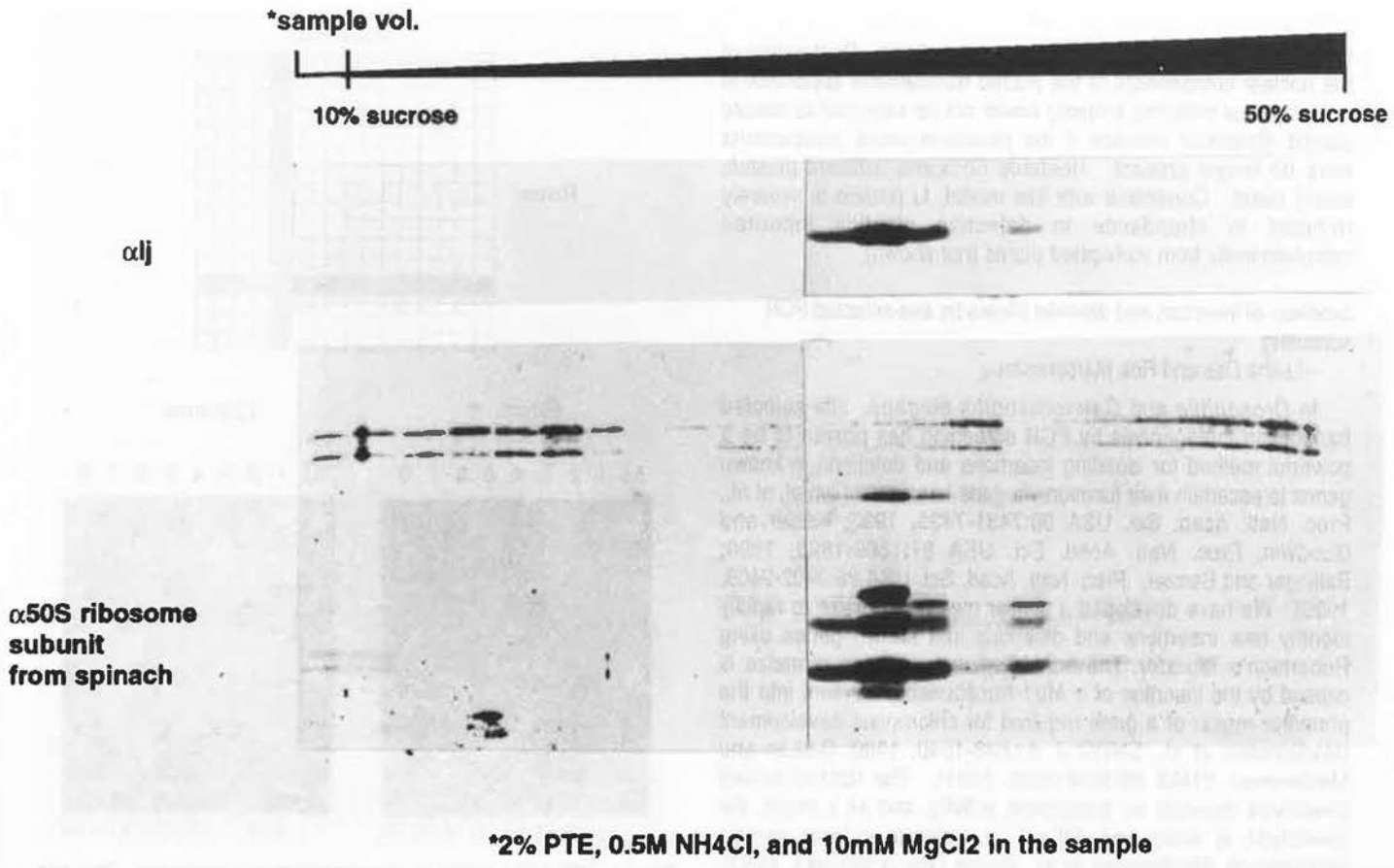

Figure 1. Western blots of IJ and 50S ribosomal proteins fractionated by sucrose gradient sedimentation. Plastid lysates were pelleted through 0.75M sucrose cushions, resuspended in 0.5M NH_4Cl and 2% PTE, and were loaded onto a 10-50% sucrose gradient. The gradient was centrifuged at 150,000g for 16hrs, and fractions were collected. Proteins from each fraction were analyzed by SDS-PAGE, and by Western blotting using either affinity purified anti-IJ antibody (top panel) or anti-spinach 50S plastid ribosomal protein antibodies (lower panel; from R. Mache) respectively.

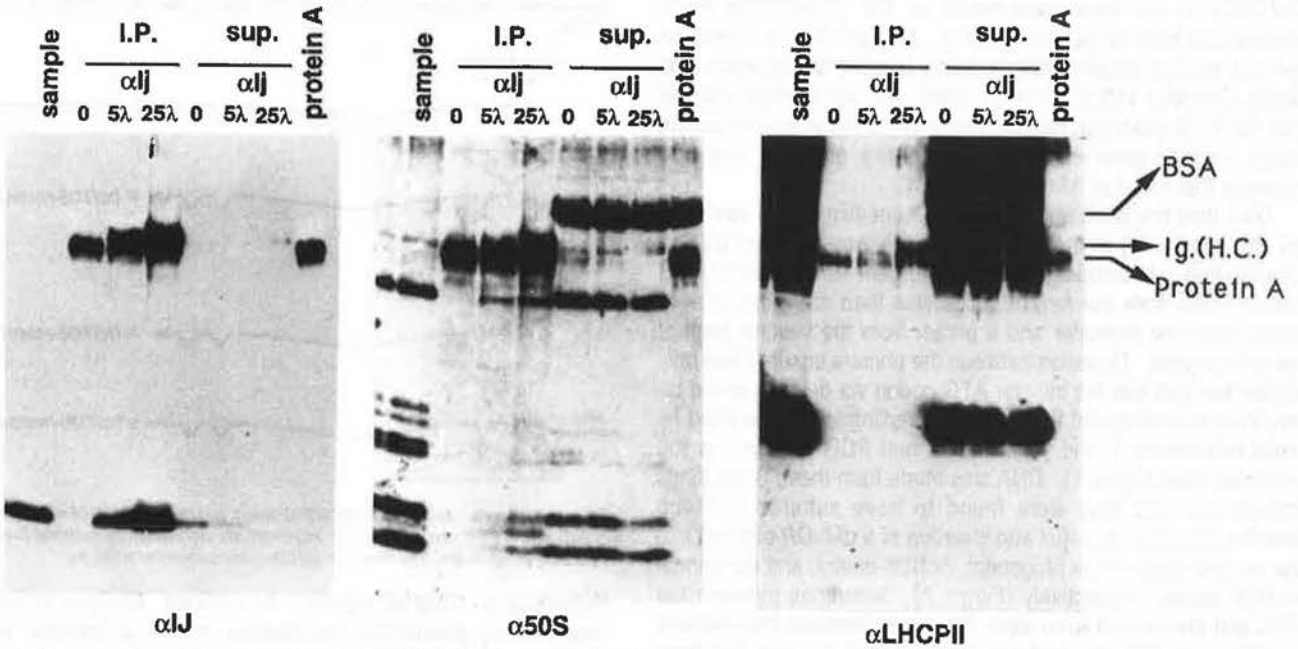

Figure 2. IP-westerns using anti-IJ antibodies. Samples were pelleted through sucrose cushions (Figure 1) were resuspended in 50mM Tris pH8, 5mM $MgCl_2$, 20mM KCl, and were used for immuno-precipitation with affinity-purified antibodies. The immuno-precipitates and supernatants were analyzed to detect IJ protein, 50S ribosomal proteins, and LHCPII membrane proteins, respectively, by sequential probing of the same blot. Lane 1, sample before i.p.; Lanes 2, 3 and 4, i.p. with 0, 5 and 25 microlitres of anti-IJ antibody; Lanes 5, 6, and 7, supernatants from immuno-precipitates loaded in lanes 2, 3 and 4, diluted 5-fold relative to the precipitates. The last lane was loaded with protein A beads alone.

to the loss of plastid-encoded ribosomal proteins. Restoration of the nuclear components of the plastid translational apparatus in heterozygous maternal progeny would not be expected to restore plastid ribosomal function if the plastid-encoded components were no longer present. Heritable ribosome-deficient plastids would result. Consistent with this model, IJ protein is severely reduced in abundance in defective plastids inherited cytoplasmically from variegated plants (not shown).

Isolation of insertion and deletion alleles by site-selected PCR screening

--Lekha Das and Rob Martienssen

In *Drosophila* and *Caenorhabditis elegans*, site-selected transposon mutagenesis by PCR screening has proven to be a powerful method for isolating insertions and deletions in known genes to ascertain their function via gene knockouts (Zwaal, et al., Proc. Natl. Acad. Sci. USA 90:7431-7435, 1993; Kaiser and Goodwin, Proc. Natl. Acad. Sci. USA 87:1686-1690, 1990; Ballinger and Benzer, Proc. Natl. Acad. Sci. USA 86:9402-9406, 1989). We have developed a similar method in maize to rapidly identify new insertions and deletions into known genes using Robertson's *Mutator*. The *hcf106-mum1* mutation in maize is caused by the insertion of a *Mu1* transposable element into the promoter region of a gene required for chloroplast development (Martienssen et al., EMBO J. 8:1633-1640, 1989; Barkan and Martienssen, PNAS 88:3502-3506, 1991). The *hcf106* mutant phenotype depends on transposon activity, and as a result, the phenotype is leaky and difficult to maintain in new genetic backgrounds (Martienssen et al., Genes Dev. 4:331-343, 1990). In order to obtain stable alleles, we used a PCR screening strategy to select plants that had suffered a deletion flanking the *Mu1* element at the *hcf106* locus. 1500 seedlings from the cross *hcf106*/+ x +/+ were germinated in the greenhouse in 2-dimensional grids to facilitate pooling. *Mu* activity was monitored using a second independent mutation (*Les28*; Martienssen and Baron, Genetics 136:1157-1170, 1994), and calculations showed that the 1500 seedlings represented a collection of about 350 *Mu* active *hcf106* gametes, 350 *Mu*-active + gametes and 800 gametes that had lost *Mutator* activity.

DNA was prepared from pools of 32 seedlings from each row (x) and column (y) of the grids. The DNA was digested with an enzyme that cuts uniquely in the *hcf106* gene at the initiator ATG codon. DNA from the *hcf106* locus was then amplified using a primer from the promoter and a primer from the second exon of the *hcf106* gene. Digestion between the primers ensured that only alleles that had lost the initiator ATG codon via deletion would be amplified in subsequent PCR. Three seedlings were identified by cross-referencing x and y pools that had PCR products of the predicted size (Figure 1). DNA was made from these 3 seedlings individually, and they were found to have suffered a 244bp deletion, insertion of a *Mu1* and insertion of a *dMuDR* element into the second intron of the progenitor, *hcf106-mum1*, and the normal *Hcf106* alleles, respectively (Figure 2). Insertions between the ATG and the second exon were recovered because they allowed amplification with the primers used. Insertions and deletions affecting the 360bp target region between the ATG and the second primer were recovered at a frequency of 1 in 350 gametes. Outcrossing and self-pollination revealed that two of these new alleles (the deletion and one of the insertions) conferred stable mutant phenotypes when homozygous, even in the absence of

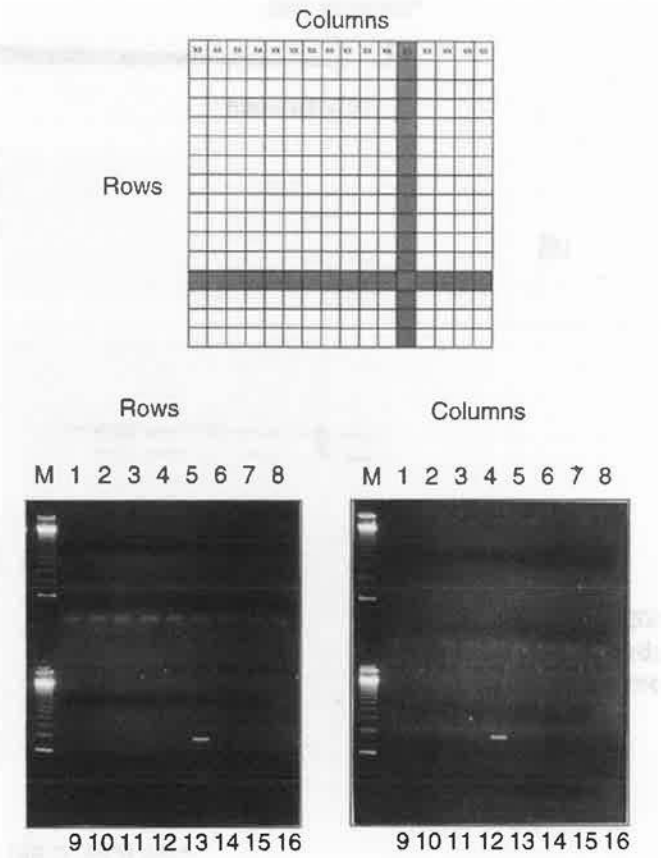

Figure 1. PCR pooling strategy for site-selected transposon mutagenesis. Seed were germinated in a 16 x 16 grid, with 2 seedlings at each position in the grid as shown. DNA was extracted from pools of 32 seedlings from each row and each column, and amplified using primers from the *Mu1* element and the second exon of the *Hcf106* gene. Each lane of each gel was loaded with PCR products from individual rows and columns. In this example, the same derivative allele was amplified in one row and one column, and thus identified by a uniquo address.

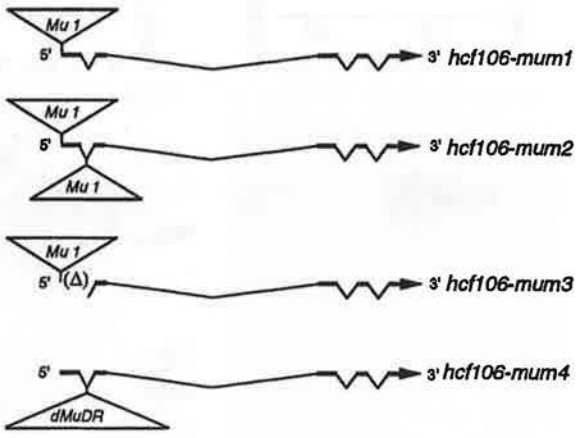

Figure 2. Schematic maps of progenitor *hcf106-mum1* and derivative *hcf106-mum2, hcf106-mum3,* and *hcf106-mum4* alleles. Exon sequences are represented by horizontal lines, while introns are shown as dips. The deletion in *hcf106-mum3* is shown as a triangle.

Robertson's *Mutator* activity. In contrast, *Mutator* activity is required for phenotypic expression of the progenitor allele, *hcf106-mum1*, in homozygous seedlings (Martienssen et al., Genes Dev. 4:331-343, 1990).

This method provides a powerful way to identify deletion (null) derivative alleles in maize, as well as new insertions in maize genes without phenotypic selection: First, insertions are recovered in a

gene of known sequence by PCR screening. In a second generation, deletions can be recovered in screens using the first insertion as the starting point. New alleles are recovered in heterozygotes, so that even if the deletion alleles are lethal, they can still be propagated and studied. Sequencing of the PCR products can be used to precisely define the derivative alleles before their phenotype is known, so that this technique is ideal for studying genes with lethal phenotypes, or those for which only dominant, gain-of-function alleles are known.

Transposon tagging of the *indeterminate* gene

--Joseph Colasanti and Venkatesan Sundaresan

The *indeterminate* (*id1*) mutation of maize causes a prolonged vegetative phase of development that results in an increase in the number of leaves and a late flowering phenotype. In a previous issue of the Maize News Letter (MNL 66:30) we reported the isolation of a new *indeterminate* mutation, *id**, and suggested that this mutation was the result of a *Ds2* transposon insertion. Since then we have shown that *id** is allelic to two known *id1* mutations, *id1-R* (from the Maize Coop) and *id1-Compeigne* (courtesy of Ben Burr, Brookhaven); we designate this new allele as *id1-CSH*. The *id1-CSH* allele was crossed into several different backgrounds and molecular analysis was performed on these plants to determine if a *Ds2* element segregated with the *id* phenotype.

A clone containing a 100 bp DNA fragment specific to *Ds2* (gift of Sarah Hake, U.C. Berkeley) was used to probe Southern blots. In over 120 progeny analysed, a 4.2 kb *Sac*I fragment was found that was always present in plants that carried the *id1-CSH* allele, and was absent in plants that did not carry it. This fragment was cloned into a pUC-based plasmid, pLITMUS29, and restriction mapped. The *Ds2* element was inserted 165 bp from one end of the 4.2 kb fragment (the "right flank") and 2.9 kb from the other end ("left flank"). When part of the left flank DNA was used to re-probe the original Southern blots, a single 4.2 kb *Sac*I fragment was found in *id1-CSH* homozygous plants and a 2.9 kb *Sac*I fragment was found in plants that did not have the *id1-CSH* allele; heterozygous plants had both bands. Interestingly, when left or right flank DNA was used to probe *Sac*I cut DNA from *id-R* plants, no band was visible. This suggests that the *id1-R* allele might be a deletion of part or all of the *Id1* gene. Further, the *id1-Compeigne* allele, when probed with the flanking DNA, exhibited a pattern which suggests that an insertion of approximately 3 kb exists in the left flank region. Taken together, these results strongly suggest that the *id1-CSH* mutation is the result of a *Ds2* insertion.

Sequence analysis of the regions immediately flanking the *Ds2* element revealed that the transposon is inserted into a putative open reading frame (ORF) of at least 84 amino acids. Preliminary RNA blot analysis using a DNA fragment containing part of the ORF region as a probe showed that it hybridized to a relatively abundant transcript of approximately 2.0 to 2.2 kb in polyA+ RNA from shoot apex tissue, and, to a much lesser extent, in mature leaf tissue. Very little hybridization was detected in seedling RNA, and no transcript was detected in RNA from roots. Comparison of the ORF sequence to known sequences in the database showed that it has similarities to "zinc-finger" DNA binding proteins from several species, including humans, *Drosophila* and *Xenopus*. This is further suggestive evidence that, if the ORF is a part of the *Id* coding sequence, the Id protein might function as a regulator of the vegetative-to-reproductive transition.

Isolation of a recombinant between *id1-CSH* and *bz2-m2*

--Joseph Colasanti and Venkatesan Sundaresan

In MNL 66:30 we reported the isolation of a new allele of the *indeterminate* (*id1*) mutation on chromosome 1. Subsequent genetic and molecular data have demonstrated that this mutation was caused by the excision of a *Ds2* element located in the nearby *bz2-m2* allele, and its re-insertion into the normal *Id1* gene (see previous report). The new *id1* allele, *id1-CSH*, is therefore linked to the restored *Bz2* gene that resulted from the germinal excision of the *Ds2* element. Purple kernels from this family, when planted and selfed, usually carried the *id1-CSH* allele (greater than 95% of the time), suggesting that *id1* is closely linked to *Bz2*. The distance between *id1* and *Bz2* was suspected of being somewhere between 1 and 10 cM, with *id1* proximal to the centromere, although, to our knowledge, precise mapping has not been carried out. The experiments described here to obtain a recombinant between these two loci show that the recombination distance is closer to the smaller value.

Maize plants that are homozygous for the *id1* mutation flower very late (or not at all) and they often exhibit aberrant floral development; i.e., the ears and tassels have marked vegetative characteristics. Whereas plants that are homozygous for the *id1-R* allele (from the Maize Coop) flower uniformly but very late in the season, *id1-CSH* homozygous plants have a wide range of flowering times, ranging from several weeks after normal sibs (i.e. mid to late August) to showing no tassel emergence even at the end of the season (mid-November). We wanted to see if the variation in severity of the *id1-CSH* phenotype was correlated with the presence of an *Ac* element; i.e., does somatic excision of *Ds2* from *id*-1* restore *Id1* function and result in plants with intermediate phenotypes?

To test this idea, a set of crosses was performed to create a maize line in which the *id1-CSH* allele was linked to the *bz2-m2* allele. Plants heterozygous for *id1-CSH* (*id1-CSH Bz2/Id1 def(an1..bz2)-6923* were grown in the autumn greenhouse of 1991 and crossed to *Id1 bz2-m2/Id1 bz2-m2* plants (MNL 66:30 describes how the original *id1-CSH* allele was isolated). Homozygous *id1* mutant plants cannot be crossed easily because of their late and unpredictable flowering time, and because they rarely produce viable ears. Because *id1-CSH* and *Bz2* are closely linked, purple kernels from the F1 were presumably *id1-CSH Bz2/Id1 bz2-m2*; this was confirmed by selfing. These kernels were planted in the winter greenhouse of 1992 and crossed to *Id1 bz2/Id1 bz2* plants; the *bz2* allele of these latter plants is the standard allele available from the Maize Coop (127A- *bz2 zb7 bm2*). To screen for crossovers, 400 spotted kernels from this cross were selected and planted in the 1992 summer field. Most of these kernels were *Id1 bz2-m2/Id1 bz2*, however, some of the progeny should be *id1-CSH bz2-m2/Id1 bz2* when a crossover occurs between the *id1-CSH* and *Bz2* loci. Of the 400 plants, 346 were selfed and 20 spotted kernels from each ear were planted in the 1993 summer field to screen for segregation of *id* mutants. Four families out of the 346 screened segregated *id* plants, suggesting that the genetic distance between *id1* and *Bz2* is approximately 1 cM (1.2 cM from these data).

In a parallel experiment, a crossover between the standard *id1-R* allele and the *bz2* allele was isolated (the seeds for this experiment were provided by John Larkin, via Scott Poethig.) Basically a similar scheme to the one described above was used; i.e., the *id1* allele linked to *Bz2* was crossed to *bz2*-carrying plants and

recombinations between *id1* and *bz2* were screened by selfing. In the 1994 summer field, 139 families (20 bronze kernels from each ear) were screened for the segregation of *id* plants. Two out of 139 *id*-segregating families were found, which suggests that the distance between *id1* and *bz2* is approximately 1.4 cM. This is in agreement with the recombination data obtained for the *id1-CSH* and *bz2-m2.* alleles. Further, this suggests that the *Ds2* elements located in *id1-CSH* and *bz2-m2* do not have a major effect on recombination between these two loci.

id1-CSH mutants flower earlier in the presence of *Ac*

--Joseph Colasanti and Venkatesan Sundaresan

With the *id1-CSH* allele closely linked to the *bz2-m2* allele, anthocyanin sectors in the aleurone can be used as a marker for *Ac* activity. The *Ac* element used in these experiments is located on chromosome 9 (Kelly Dawe, personal communication), and therefore segregates independently of the *id1-CSH bz2-m2* loci. We wanted to determine if the *Ac* activity, which causes a variegated phenotype in the aleurone by somatic excision of the *Ds2* element from the *bz2-m2* allele, has a similar effect on the *id1-CSH* phenotype; i.e., does somatic excision of the *Ds2* element from the *id1-CSH* allele restore *Id* function such that the flowering time for the *id1-CSH* mutants is reduced by *Ac*?

As described in the above report, 4 out of 336 families segregated *id1-CSH* mutant plants. Since only spotted kernels were planted, all *id* plants in each of the four families should be homozygous for the *id1-CSH* and *bz2-m2* alleles, whereas the normal siblings (i.e., in this background, those that flower 9 to 11 weeks after planting) should be *id1-CSH bz2-m2/Id1bz2* (except in rare cases where recombination or germinal excision results in an *Id1 bz2-m2* constitution). The spotted kernels indicate that each plant should also carry at least one *Ac* element. Normal siblings from each family were selfed and out-crossed to an *Id1 bz2/Id1 bz2* tester in the 1993 summer field. The frequency of spotted and bronze kernels on each ear was analyzed to determine the number of *Ac* elements. Ears derived from selfed plants with one *Ac* element segregated spotted and bronze kernels at a frequency of 9/16 and 7/16, respectively, and the same plant outcrossed to a *bz2* tester had a spotted kernel to bronze kernel ratio of 1/4 to 3/4.

To compare flowering time of *id1* mutants in the presence and absence of *Ac* activity, spotted and bronze kernels from each ear of 10 different families with a single unlinked *Ac* element were planted in the 1994 summer field. As expected, 1/3 of the spotted kernels from these ears segregated *id* mutant plants. The other 2/3 had normal flowering times and should be heterozygous plants; i.e., *id1-CSHbz2-m2/Id1 bz2*. The bronze kernels of the selfed progeny of *id1-CSHbz2-m2/Id1 bz2, Ac/-* heterozygous plants represent 7/16 of the total kernels on each ear. Of these *bz* kernels, 1/4 (4/16) should be homozygous for the tester allele, *Id1 bz2/Id1 bz2*. The other 3/16 represent kernels that carry the *id1-CSH bz2-m2* allele, but have no *Ac* element and therefore are not spotted. Of these kernels, 1/3 should also be homozygous for *id1-CSH bz2-m2*, and therefore express the *indeterminate* mutant phenotype. Therefore, of the bronze kernels planted, 1/7 should segregate *id* mutants. Overall, the experiment involved comparing the flowering time of *id* plants grown from spotted kernels with that of *id* plants grown from bronze kernels. These were compared to the flowering time of normal siblings found in both populations.

All kernels were planted in the third week of May, 1994. Normal plants from both spotted and bronze kernels usually made between 11 and 13 leaves, and all shed pollen sometime between the third week of July and the first week of August when they were approximately 9 to 11 weeks old. From the 10 rows of spotted kernels planted, 47 clearly *indeterminate* plants were segregating; i.e., none of these had emerging ears or tassels by the time their normal siblings had shed out, and they all had more than 13 visible leaves. The 10 rows of bronze kernels yielded 17 *indeterminate* plants. These also showed no signs of flowering by the time their normal sibs had shed out, and they appeared to grow more slowly than the spotted kernel *id* plants; i.e., they had fewer leaves and they were shorter. By the first week of September, at about 17 weeks after planting, some of the spotted kernel *id* plants had tassels emerging and several had leafy ear shoots, whereas the bronze kernel *id* plants showed no signs of flowering. In mid-September, three of the spotted kernel *id* plants were shedding pollen and silking; these were either selfed or crossed to each other. These plants had between 15 and 17 leaves. Approximately half of the *id* plants from spotted kernels were showing tassels at this time, while the bronze kernel *id* plants continued to produce leaves but no flowers.

By mid-October, when the plants were about 21 weeks old, all of the spotted kernel *id* plants, with the exception of three plants in one family (see below), had either shed out or had tassels emerging, and all had from 17 to more than 20 visible leaves. Most of these did not make ears, and the few ears that did emerge had marked vegetative characteristics. All of the *id* plants from bronze kernels, however, continued to produce leaves, and they had neither tassels nor ears. These plants continued to grow vegetatively and make approximately 18 to 25 leaves until the first killing frost on November 13, 1994 when they were 25 weeks old. Several of these plants were dissected and found to have tassel primordia ranging from 0.5 to 1.0 cm in length. By comparison, several *id1-R* plants that were planted at the same time in a nearby field were dissected and found to have similar size tassel primordia; none of these *id* plants produced ears.

Since spotted kernels indicate the presence of *Ac*, it appears that *id1-CSH* plants that had an *Ac* element in the background flowered significantly earlier than plants from the same family that did not have an *Ac* element. In fact, the latter plants did not flower at all, and they made only tiny tassel primordia by the time they were six months old at season's end. This is what one would expect if the *id1-CSH* allele had a *Ds2* element inserted into it which responded to *Ac* transposase; i.e., *Ac* induces the somatic excision of the *Ds2* element, occasionally producing sectors of normal tissue which can synthesize functional *Id1* gene product. It is interesting to note that mosaic plants were not seen in the *id1-CSH* plants that flowered earlier; i.e., sectors of normal tissue juxtaposed to mutant tissue were not apparent. Instead the plants had an intermediate phenotype. This might suggest that the *Id1* gene product does not act cell autonomously, and that it is a diffusible substance or that it mediates the production of a diffusible product. On the other hand, it is difficult to predict what a plant composed of sectors of *indeterminate* and normal tissue would look like. Clonal analysis experiments are currently underway to address this question.

Finally, as mentioned above, of the 47 *id* plants derived from spotted kernels, all flowered before the end of the season except 3. These plants originated from the same family and, in general,

resembled *id1-CSH* plants derived from bronze kernels in terms of growth rate, leaf number and inability to flower. DNA was prepared from each of these *id* plants and Southern blotting was performed using *Ds2*-flanking DNA as a probe. Unlike all *id1-CSH* plants examined so far, which contain a single 4.2 kb *Sac*I fragment, all three of these plants had a 2.9 kb *Sac*I band; i.e. similar in size to the *Sac*I band found in plants with a normal *Id1* allele (see above report). Primers flanking the *Ds2* insertion of *id1-CSH* alleles were used to amplify and clone this region from one of the 3 mutants. Sequence analysis revealed that this plant differed from normal plants in this region by having a 5 bp insertion. This sequence appears to be the remnants of a target site duplication, initially caused by the *Ds2* insertion. In effect, a stable allele of *id1* was created by the imprecise excision of *Ds2*. The 5 bp DNA insertion causes a frame shift in the alleged open reading frame of the Id protein (see above report). As observed, this new *indeterminate* allele (termed *id1-X*) should not respond to the presence of an *Ac* element. This finding provides additional evidence that the earlier flowering phenotype of *Ac*-containing *id1-CSH* plants is the result of somatic excision of the *Ds2* element during development.

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Structural characterization and phylogenetic evaluation of two pairs of MADS-box genes

--Günter Theißen, Tim Strater, Achim Fischer and Heinz Saedler

We have cloned and sequenced two MADS-box genes and the partial cDNA of a third one (Theißen et al., Gene, in press). One of the genomic clones was identified as *zag2* (*Zea AGAMOUS-like 2*), while the other, termed *zmm1* (*Zea mays MADS1*) has a very similar structure and the potential to encode a protein which shares 94% sequence identity with the putative *zag2* gene product. The cDNA reveals considerable similarity to *zag1* (Schmidt et al., Plant Cell 5:729-737, 1993) and the respective gene was termed *zmm2*. Remarkably, at least in the region downstream of the MADS-domain, *zmm2* is much more similar to AGAMOUS from *Arabidopsis thaliana* than *zag1*. Phylogenetic evaluation of the sequence information, as well as chromosomal localization, both suggest that we have identified two pairs of *AGAMOUS*-like MADS-box genes which were created during duplication of chromosomal segments or complete chromosomes.

Genomic organization of MADS-box genes

--Achim Fischer, Nikolai Baum, Heinz Saedler and Günter Theißen

We have developed a linker PCR procedure for preparing repetitive DNA-free probes from genomic clones, which is especially efficient for members of gene families. Using this procedure as well as standard methods to prepare hybridization probes, chromosomal map positions of MADS-box genes were determined in recombinant inbred lines. It appeared that MADS-box genes are strongly dispersed throughout the maize genome. While there is evidence that this genomic distribution is representative for plant MADS-box genes in general, the following two other observations seem to be *Zea* specific. First, at least one subfamily of MADS-box genes contains line specific gene versions, which are present in some maize lines at certain chromosomal positions, but are absent there in other lines. The members of that subfamily resemble transposable elements in many aspects. Second, the duplicate character of the maize genome, as established with different marker systems, is also revealed by the finding of pairs of highly related MADS-box genes which are accompanied by other duplicated markers. These findings have far-reaching implications for an understanding of the genomic organization of MADS-box genes and the evolution of the MADS-box gene family.

Differential expression of MADS-box genes

--Achim Fischer, Heinz Saedler and Günter Theißen

We have developed a novel protocol for efficient expression analysis of multigene families. The method enables a reliable and detailed high resolution expression analysis of known gene family members as well as the identification and characterization of new ones. We have used this technique to analyze differential expression of MADS-box genes in male and female inflorescences of maize. Six different MADS-box genes could be identified, among them four that had been described before, namely *zag1*, *zag2* (Schmidt et al., Plant Cell 5:729-737), *zmm1* and *zmm2* (Theißen et al., Gene, in press). According to Northern experiments as well as studies using our new protocol, *zag1* is more strongly expressed in female inflorescences, whereas *zmm2* is preferentially expressed in the tassel. *zag2* and *zmm1* expression is restricted to the female inflorescence. *zmm3* and *zmm7* are new genes identified by use of our technique, with *zmm3* being preferentially expressed in the male inflorescence, and *zmm7* being female-specific (see also Cacharrón et al., this issue). We suggest that our approach not only opens a major avenue towards expression analysis of gene families in general, but, applied to MADS-box genes, may help to answer old questions about the molecular basis of sex determination, monoecy/dioecy and certain flower mutations.

Expression patterns of MADS-box genes as studied by *in situ* hybridization

--Jorge Cacharrón, Achim Fischer, Heinz Saedler and Günter Theißen

During recent years, MADS-box genes have been shown to control inflorescence and flower development in dicotyledonous plant species. This let us assume that they may play a similar role in monocots like maize. Therefore, we have taken advantage of cDNA clones to study the expression patterns of several MADS-box genes in maize (*Zea mays* ssp. *mays*, T232) and teosinte (*Zea mays* ssp. *parviglumis*, Balsas) by *in situ* hybridization experiments. Here the results for three of the MADS-box genes, termed *zmm6*, *zmm7* and *zmm8*, are reported.

Interestingly, in the case of *zmm6*, differential expression between maize and teosinte was found. The gene shows the earliest expression, out of the analyzed ones, starting at the stage when spikelet-pair-primordia bifurcate into developing pedicellate and sessile spikelets. At this stage, expression of *zmm6* is restricted to one of each spikelet-primordium of a pair. This pattern is visible in male and female inflorescences of maize, while expression of *zmm6* in teosinte is only present in the male inflorescence. We could not detect any expression at corresponding developmental stages in female teosinte inflorescences. Further hybridization experiments with female

maize inflorescences showed that *zmm6* is also expressed in later stages during spikelet development, but then it is present in both the sessile and the pedicellate spikelet in stamen and gynoecium. According to the observed expression pattern one can assume a possible role for *zmm6*. Though we do not know yet whether initial expression is located in the pedicellate or the sessile spikelet, the expression correlates with the presence of the pedicellate spikelet which is aborted at an early stage during development of the female teosinte inflorescence. Thus *zmm6* could have acted, e.g., as a "reactivator" of the pedicellate spikelet during the transition from teosinte to maize. Furthermore, in investigations of Doebley and co-workers (PNAS 87:9888-9892, 1990) mapping with RI lines showed that *zmm6* is located on chromosome 1, in a region that was significantly associated with the morphological trait PEDS (pedicellate spikelet score). This supports the functional role of *zmm6* mentioned above. Further experiments are now being carried out to test this hypothesis.

In both the female inflorescences of teosinte and maize, *zmm8* is expressed in the undifferentiated flower meristem of each spikelet, and during development expression becomes located to the stamen and gynoecium as well as palea and lemma of the upper flower. *zmm8* is not expressed in the lower flower of each spikelet. A comparison of the putative protein sequence downstream of the MADS-domain between *zmm8* and *OsMADS1* of rice (Chung et al., PMB 26:657-665, 1994), showed 76% sequence identity. The distant relationship between maize and rice, together with the high sequence identity between both genes, makes it conceivable that *zmm8* is the ortholog of *OsMADS1*. The expression pattern of *zmm8* closely resembles that reported for *OsMADS1*, with one remarkable difference: we observed expression also in the stamen primordia of the teosinte ear, while a corresponding expression pattern has not been reported for *OsMADS1*. Maybe this is due to differences between unisexual and perfect flower development, respectively. For example, expression of a *zmm8/OsMADS1*-like gene could be incompatible with proper stamen formation. Thus, the gene has to be suppressed in the perfect rice flowers, but not in the female teosinte and maize flower, where stamen primordia are aborted. This hypothesis could be tested by studying the expression of *zmm8* in the maize tassel.

zmm7, in contrast to the other analyzed MADS-box genes, shows the latest onset of expression. In the female maize inflorescence, expression starts at a stage when nearly all flower organs are differentiated. It is located in the developing gynoecial ridge, and later overall in the developing silk. Further *in situ* hybridization experiments will show whether expression of *zmm7* is absent in the male inflorescence, as one would expect. Such a result would suggest that *zmm7* function is restricted to silk development. Chromosomal localization of this gene and analysis of silkless-mutants should also give more insights.

Ac transposase binds in vitro to the *Ac/Ds* terminal inverted repeats

-- Heinz-Albert Becker and Reinhard Kunze

In an earlier publication we have reported that an *Ac* transposase (TPase) protein, overexpressed in insect cells, does not bind in vitro to the terminal inverted repeats (TIRs) of *Ac/Ds* (Kunze et al., EMBO J. 8:3177-3185, 1989). During the last year we have continued the DNA-binding studies with bacterially expressed and renatured TPase derivative lacking the amino-terminal 102 amino acids, TPase(103-807), and we investigated the influence of different incubation buffer compositions on the DNA-binding properties of the TPase. In addition, we could increase the sensitivity of the gel retardation assays by using a PhosphorImager for detection.

These modifications enabled us to detect a weak interaction of the TPase protein with the TIRs. This interaction is sequence-specific, as the mutated TIR sequence of the stable *Ac18* element (Hehl and Baker, Mol. Gen. Genet. 217:53-59, 1989) is not recognized. The TPase/TIR-complexes have electrophoretic mobility similar to the complexes between TPase and the subterminal AAACGG motifs, i.e. they migrate as a diffuse band in agarose gels. However, the binding reactions differ: The TPase protein binds the subterminal AAACGG motifs with much higher affinity than the TIRs.

The *Ac* TIR sequence (C/TAGGGATGAAA) does not contain an A/TCG motif, which has been identified as the essential core sequence responsible for TPase binding to the subterminal AAACGG motifs (Becker and Kunze, MNL 68:21-22, 1994). We have therefore initiated experiments to determine whether the TPase has separate domains for binding to the TIRs and the subterminal motifs. We have obtained preliminary results indicating that the TPase-derivative TPase(103-465/R191H/H193R), which does not bind to the AAACGG motifs (Feldmar and Kunze, EMBO J. 10:4003-4010, 1991), has also lost the ability to bind to the TIRs. Accordingly, the basic TPase residues 191 and 193 are involved in recognition of the TIRs and the subterminal AAACGG motifs.

Aggregation of *Ac* transposase in transgenic tobacco

-- Iris Kornacker and Reinhard Kunze

We succeeded in visualizing aggregates of the *Ac* transposase (TPase) by immunofluorescence in transgenic tobacco protoplasts. Protoplasts were prepared from tobacco plants carrying the full length *Ac* TPase gene and a truncated TPase gene lacking 102 amino acids from the amino terminus, respectively, under control of the cauliflower mosaic virus 35S promoter. Protoplasts were centrifuged onto polylysine-precoated microscopic slides. After fixation and alcohol-extraction, they were stained by the indirect immunofluorescence procedure described by Heinlein et al. (Plant J. 5:705-714, 1994).

In protoplasts containing the full length TPase gene, the protein is detected in the nuclei in the form of many, rod-like aggregates, similar to those that were observed with a very low frequency in maize. In protoplasts expressing the truncated TPase derivative, in addition to nuclear complexes large bodies of coalesced TPase are detected in the cytoplasm. We conclude from these results that the full length TPase and the truncated TPase, which is lacking one of its three nuclear localization signals (Boehm et al., Plant J., in press, 1995), both exhibit very similar properties as in transiently transfected *Petunia hybrida* protoplasts. Individual batches of protoplasts always display immunofluorescence signals of very similar intensity and uniformly shaped aggregates. In several independent transformants the immunofluorescence signal (supposedly reflecting the amount of TPase) is always higher in plants homozygous for the transgene than in heterozygotes. The age of the plant material (between 6 and 19 weeks) used for protoplast preparation apparently has no influence on amount or shape of the aggregates.

Scofield et al. (Cell 75:507-517, 1993) have recently shown

that high levels of *Ac* TPase inhibit transposition in transgenic tobacco plants. Our results indicate that the TPase forms large aggregates, and in *Petunia* similar aggregates are supposed to be transpositionally inactive (Heinlein et al., Plant J. 5:705-714, 1994). Though there is only indirect evidence for the inactivity of the TPase aggregates and we cannot exclude a specific function of them during transposition, it is conceivable that the TPase aggregates are responsible for the effects described by Scofield et al.

Analysis of the methylation state of the transposase binding sites in the termini of *Ac*

-- Lihua Wang, Manfred Heinlein, Peter Starlinger and Reinhard Kunze

The DNA binding-affinity of the *Ac* transposase (TPase) protein to the subterminal AAACGG target motifs may be increased or decreased by C-methylation on one or the other of the two DNA strands. This differential recognition of hemimethylated target motifs by TPase could provide a mechanism for the coupling of the transposition reaction to replication.

A prerequisite for such a mechanism is that the TPase target sites are methylated in vivo. This can only be tested by genomic sequencing as the AAACGG motifs are not accessible by restriction endonucleases. We have adopted the recently developed bisulfite genomic sequencing procedure, during which all non-methylated cytosine residues are deaminated into uracil, whereas 5-methyl-cytosines are not converted. This method allows the sequence determination of many individual genomic DNA molecules (Frommer et al., PNAS 89:1827-1831, 1992).

We have begun to investigate the methylation status of the *Ac* termini in the *wx-m9* allele. Genomic DNA was extracted from dry kernels with one *wx-m9::Ac* dose in the endosperm. As a control for the reaction conditions, plasmid pJAC (which contains the *Ac* element from the *wx-m7::Ac* allele) was grown either in a *dcm+* or *dcm- E. coli* strain and mixed in single copy gene-concentration to the genomic maize DNA. Four pairs of primers were designed to amplify the *Ac* 5´-end. Primer pair 1 will give rise to a PCR band with unreacted genomic *wx-m9* DNA. Pair 2 will produce a PCR band with unreacted pJAC. Primer pair 3 was designed to yield a PCR product with bisulfite-reacted genomic *wx-m9* DNA, and pair 4 will produce an amplification product exclusively on bisulfite-reacted pJAC DNA.

We have meanwhile established appropriate bisulfite reaction conditions. PCR products of the expected lengths were obtained using the bisulfite reacted genomic DNA/pJAC-mixture as template and the two primer pairs specific for bisulfite reacted DNA, whereas no products were obtained with the primers specific for unreacted DNA.

The PCR products were cloned into pUC19 and sequenced. The pJAC DNA served as a control for the reaction conditions. As expected, all cytosine residues in the *dcm-* pJAC DNA had been converted, whereas in *dcm+* pJAC DNA the two cytosines located in dcm-recognition sites were not converted.

The bisulfite-converted upper DNA strand of the *Ac* 5´-end could also be amplified in one step and the resulting PCR band was cloned into pUC19. We have obtained preliminary data indicating that the degree of methylation of individual *Ac* molecules at the 5´-end differs. About half of all *Ac* molecules contain no methylated cytosines in the 5´-terminal 250 nucleotides. The other half is methylated to varying extents. In the majority of these molecules, more than 50% of the cytosines between *Ac* positions 26 - 90 are methylated. However, only very few, if any at all, of the more internally located cytosines are methylated. This predominantly unmethylated region encompasses the most prominent blocks of AAACGG-repeats, the TPase binding sites. We are presently extending our studies by analysing the methylation states of the lower DNA strand at the *Ac* 5´-end and both DNA strands at the 3´-end.

Transposable elements *Bg* (*Zea mays*) and *Tag1* (*Arabidopsis thaliana*) encode protein sequences with homology to *Ac*-like transposases

-- Lutz Essers and Reinhard Kunze

The maize elements *Ac/Ds* are members of a widely distributed class of eukaryotic transposable elements which are characterized by a non-transcriptional transposition mechanism, terminal inverted repeats and the creation of target site duplications at their insertion sites. To date, a group of 15 transposable elements or transposable element-like sequences have been discovered whose terminal inverted repeats have sequences similar to *Ac/Ds*.

In addition, five of these elements contain internal sequences (putatively) encoding similar transposases. This so-called hAT family includes the plant elements *Ac* from *Zea mays* (Kunze et al., EMBO J. 6:1555-1563, 1987), *Tam3* from *Antirrhinum majus* (Hehl et al., Plant Mol. Biol. 16:369-371, 1991) and *Pac1* from *Pennisetum glaucum* (MacRae et al., Genetica 92:77-89, 1994), and the insect elements *hobo* from *Drosophila melanogaster* (Calvi et al., Cell 66:465-471, 1991) and *Hermes* from *Musca domestica* (Warren et al., Genet. Res., in press, 1995).

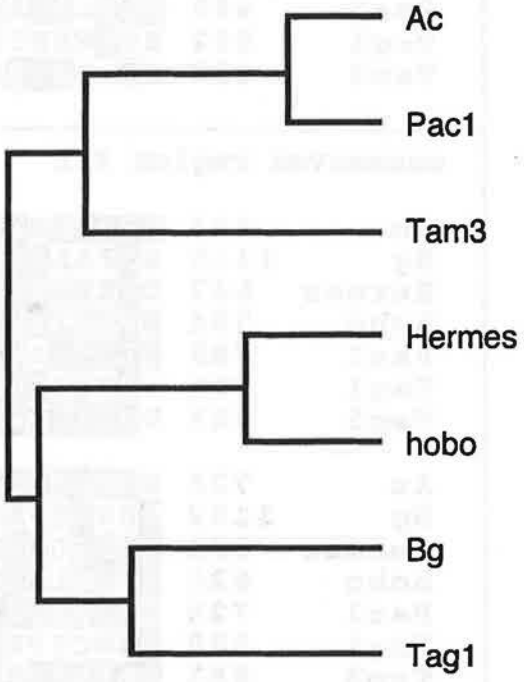

Figure 2. Calculated evolutionary relationship between different *Ac*-like transposases. The calculation was done with the GCG program "pileup", using all three conserved regions shown in Figure 1.

By homology searches we have determined that the autonomous element *Bg* from *Zea mays* (Hartings et al., Maydica 36:355-359, 1991) and the element *Tag1* from *Arabidopsis thaliana* (Tsay et al., Science 260:342-344, 1993) encode amino acid sequences with strong homology to the hAT family (Figure 1). As was described for the other members, the highest degree of conservation is observed within three separate segments, termed conserved regions I, II and III. This suggests that the *Bg* and *Tag1* transposable elements are also members of the hAT family. Two other DNAs potentially encoding proteins with a weak homology to the hAT transposases were isolated from barley genomic DNA (Chernyshev et al., Genetika 24:1338-1344, 1988) and rice cDNA (dbj RICC100171, frame +2). Moreover, by the polymerase chain reaction two sequences with homologies to conserved region I were detected in the tobacco budworm (*Heliothis virescens*) and the corn earworm (*Helicoverpa zea*) (DeVault et al., Biochem. Biophys. Res. Comm. 203:169-175, 1994). Accordingly, *Ac* transposase-related proteins appear to be widely distributed in many different species.

Finally we want to point out one interesting fact: The deduced amino acid sequence from the maize *Bg* element is more similiar to that from the *Arabidopsis thaliana* element *Tag1* than to the maize *Ac* transposase (Figure 2). Furthermore, the putative coding regions of plant transposons *Bg* and *Tag1* are more closely related to those of the insect elements *hobo* and *Hermes* than to the other plant transposons *Ac*, *Pac1* and *Tam3*. The existence of a family of related (hypothetical) transposase proteins, which are required for the mobilization of transposable elements, in very distantly

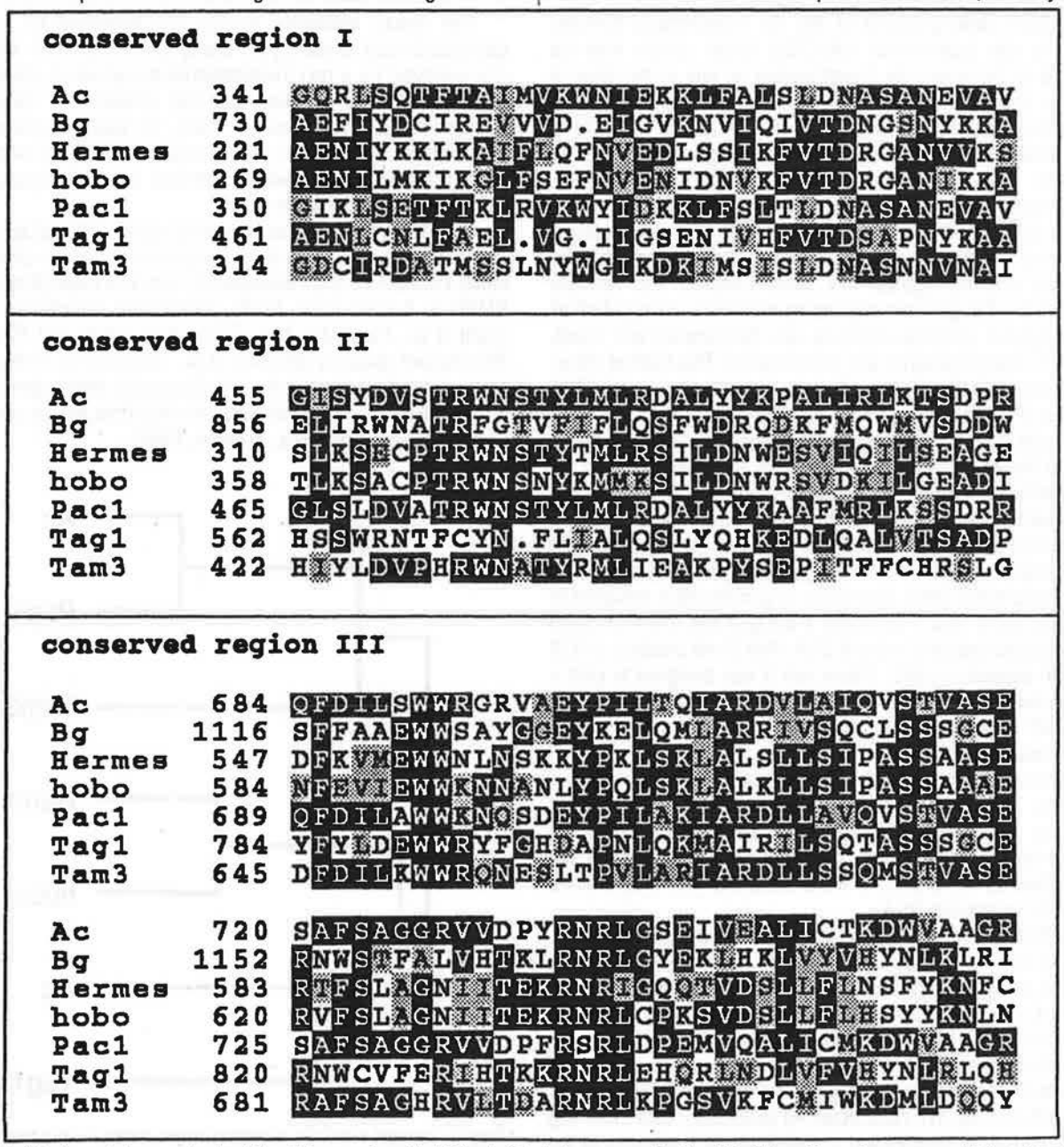

Figure 1. Multiple alignment of conserved regions I, II and III from members of the hAT family of *Ac*-like transposases. For *Bg* and *Tag1* the position numbers correspond to the codon number of the full GenBank sequence entry. The expressed frames are for *Bg* in region I frame +2 and in region II and III frame +3. All amino acids of *Tag1* are coded in frame +3. Identical amino acids are shown inverted, amino acids similar to them are shaded grey.

related species could indicate the possibility of a spreading of these elements by horizontal gene transfer. This mechanism of distribution has been suggested for the *P* element (Houck et al., Science 253:1125-1129, 1991) and *mariner* (Robertson, Nature 362:241-245, 1993).

Analysis of *Ac* transposase oligomerisation using the two hybrid system

-- Lutz Essers and Reinhard Kunze

Our present model of the *Ac* transposition process proposes that the transposase (TPase) protein binds as an oligomer to internal DNA sites near the termini of the transposon. We assume that the TPase is responsible for bringing the two *Ac* ends and possibly also the integration site in close proximity during transposition, forming a "transposom".

Two previous observations indicate the occurrence of protein-protein interactions between *Ac* TPase units. 1) By *in situ* immunofluorescence staining of transfected *Petunia* protoplasts large nuclear TPase aggregates were detected. The observation that certain amino acid substitutions in the TPase protein appear to be aggregation-deficient indicates that aggregation is an intrinsic property of the TPase. 2) A number of transposition- and DNA binding-deficient TPase mutants act as dominant inhibitors of transposition when coexpressed with a functional TPase protein.

We have begun using the yeast "two hybrid system" (Fields and Sternglanz, Trends Genet. 10:286-292, 1994) to localize the TPase protein-protein interaction domains. The two hybrid system is based on transcription activation of the lacZ gene by binding of the GAL4 protein to the upstream activating sequences. The N-terminal, DNA-binding domain of GAL4 may be separated from the C-terminal transcription activation domain. By fusing these domains separately with other proteins, ß-galactosidase expression is activated if the fusion proteins interact.

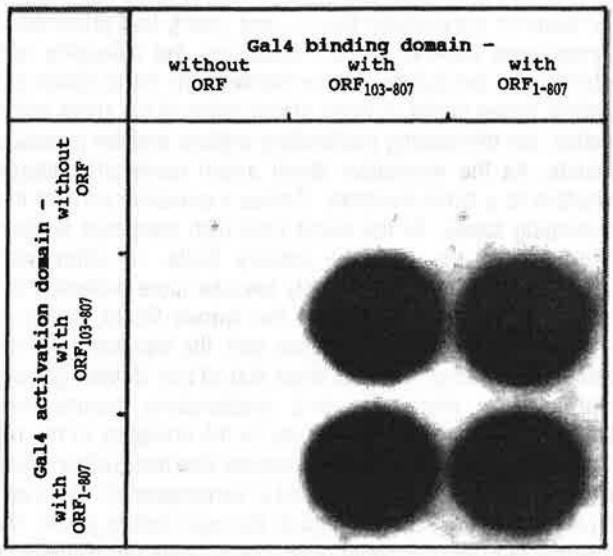

Figure 1. The figure shows the ß-galactosidase activities of yeast cells transformed with different combinations of the two hybrid plasmids. Negative controls are tests in which the TPase open-reading frame (ORF) is not present in both plasmids. All cells were grown in liquid suspension culture and aliquots were filtered through a defined area of a nylon membrane. Subsequently the cells on the filter were cracked in liquid nitrogen and incubated with X-GAL at 37 C.

The full length *Ac* TPase protein (TPase1-807) and the transpositionally active deletion derivative TPase103-807 were fused to each GAL4 domain of the standard two hybrid vectors pGAD424 and pGBT9, and transformed into yeast cells. Our results demonstrate that the wildtype *Ac* TPase protein can directly interact with itself as well as with the truncated TPase starting at codon 103 (Figure 1).

We will continue these experiments with deleted *Ac* TPase segments to localize the interactive domain(s). Although the results are not yet complete we are confident to have identified a small C-terminal TPase fragment which can still interact with the TPase protein starting at codon 103.

Suppressor activity of *Ac* transposase in transfected *Petunia* cells

-- Lutz Essers and Reinhard Kunze

The non-autonomous 0.4 kb *Ds1* element has, in contrast to most other *Ds* elements, only few AAACGG-like TPase binding motifs. Therefore *Ds1* seems to be an ideal subject for investigating their influence on the excision rate. The element was cloned in both directions between the 1'-promotor and the GUS-gene. Although *Ds1* contains several closed reading frames in both orientations, GUS-expression was obtained in transfected *Petunia* protoplasts. In further experiments promotor activities of *Ds1* were found in both orientations. However, these activities are too weak to explain the GUS-activity. We consider it more likely that the element may be read-through transcribed and spliced out, as was described for *Ds1* in maize.

We determined that the above-mentioned GUS expression was reduced to about 50% when an *Ac* transposase (TPase) expression plasmid was cotransfected into the cells. A possible explanation for this phenomenon could be a suppression effect of TPase binding to its target DNA, similar to the action of the *En/Spm*-encoded TnpA-protein (Grant et al., EMBO J. 9:2029-2035, 1990). Consistently, if the *Ds1*-element was substituted by a 16mer of the AAACGG-motif, GUS expression was reduced to about 30%. A transpositionally defective, but DNA binding-positive TPase-derivative has retained the suppressor activity, whereas mutations in the DNA-binding region abolish the suppressor effect. These findings suggest that the TPase recognizes in vivo the same DNA sequences as the renatured TPase of bacterial origin in vitro, and that the binding of the TPase to the DNA is strong enough to interfere with read-through transcription.

Expression of the *Ac* transposase in fission yeast

-- Patricia Wolff and Reinhard Kunze

To investigate if the maize element *Activator* (*Ac*) can transpose in fission yeast (*Schizosaccharomyces pombe*), we have constructed shuttle vectors containing a potentially autonomous *Ac* derivative inserted between a promoter and a selectable marker gene.

Two major modifications were introduced into *Ac*. In order to ensure expression of the *Ac* transposase gene in *S. pombe*, we replaced the *Ac* promoter and leader sequence with the fission yeast *nmt1*-promoter. This promoter can be repressed by growing the cells in thiamine-containing medium, which could be of advantage in case that expression of the *Ac* transposase at high levels turns out to be harmful for the cells. In addition, to

circumvent potential splicing problems in fission yeast, the genomic transposase coding region of *Ac* was replaced by the corresponding cDNA sequence (ORFa).

This modified *Ac* was inserted between the cauliflower mosaic virus 35S promoter and the NPTII gene, which are known to be functional in fission yeast. The final constructs contain the *ura4* gene as selectable marker and were transformed into the *S. pombe* strain h- ura4-D18. Excision of the modified *Ac* should be detectable by G418-resistance of the cells.

By Northern analysis of the transformed cells, three ORFa-homologous transcript bands were detected. The strongest signal appeared as a rather broad band about 2.8 kb in length, which is the expected size for a polyadenylated, full length ORFa transcript. In addition, two distinct but less intense bands, 1.9 kb and 3.4 kb in length, hybridized with the ORFa probe. These RNAs are presumably derived by transcriptional read-through of the *Ac* polyadenylation signal as they hybridize with *Ac* sequences 3′ to this site. The transcription initiation sites of the transcripts have not been determined.

On Western blots several protein bands are recognized by an anti-transposase-serum in total protein extracts of the transformed fission yeast cells. The largest, very weak band has the same electrophoretic mobility as the wild-type transposase. The major fraction of ORFa-derived proteins apparently consists of aberrantly processed transposase molecules.

After plating transformed yeast cells on G418-containing medium we have recovered several G418-resistant colonies. However, a simple excision of the modified *Ac* had not occurred in any of the reisolated plasmids, but rather recombinational deletions between short, directly repeated sequences were found.

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Functional redundancy of the homeobox genes *ZmHox1a* and *ZmHox1b*

--Bärbel Überlacker, Claudia Mehlem and Wolfgang Werr

ZmHox1a and *ZmHox1b* (*Zea mays* homeobox) are a pair of highly related homeobox genes encoding proteins with 87% identity on the amino acid level (91% similarity). The genes are located on chromosomes 8 (8L-17) and 6 (6L-118), respectively, within larger chromosomal regions known to be duplicated in the maize genome. No phenotypic mutant maps close to either of these positions. All Northern and *in situ* hybridization experiments performed so far show very similar transcription patterns of both genes, and the *ZmHox1a* and *ZmHox1b* mRNAs are generally restricted to meristematic and proliferating cells of the maize plant.

To gain insight into possible biological functions of each *ZmHox1* gene we ectopically expressed the *ZmHox1a* and *1b* coding frames behind the ω leader sequence of TMV under the control of the CaMV 35S promoter. The regenerated *ZmHox1a* (42) and *ZmHox1b* (137) transgenic tobacco plants show identical and heritable alterations in the development of the vegetative plant body and the flower. The phenotypic changes fall into three different classes: 1) A reduction in size. 2) An outgrowth of few to all axillary buds. The adventitious shoots often overgrow the primary shoot which indicates reduced apical dominance. 3) Alterations of the flower development in the third and fourth whorl. The stamens are homeotically transformed to petaloid organs, the stigma is changed in shape, the position of the ovaries is displaced towards the stigma and the number of ovules is significantly reduced. In severely affected plants a secondary flower develops inside the primary gynoecium. While each phenotypic alteration may vary from weak to strong, the grade of severity is highly correlated between the three classes ($P < 0.001$ in χ^2-tests). The ectopic expression of the maize *ZmHox1* homeobox gene products therefore affects different phases of the tobacco life cycle, which coincides with the transcription in different types of meristems in the maize plant.

The identical phenotypes of transgenic *ZmHox1a/1b* tobacco plants are presumably due to the 91% amino acid similarity shared between both gene products. It remains unclear why the expression patterns and the gene function of both *ZmHox1* genes have been highly conserved during evolution after gene duplication, while the individual genes in other pairs of transcriptional regulators (e.g. *C/P1* or *R/B*) acquired very distinct transcription patterns after duplication. The similar functions of *ZmHox1a* and *ZmHox1b* implied by transgenic tobacco experiments indicate a functional redundancy in the maize genome, which may explain the lack of corresponding mutants.

Transcription of the *ZmHox* homeobox genes is activated early in embryogenesis and is abundant in meristematic tissues

--Bettina Klinge and Wolfgang Werr

Four different *ZmHox* genes have been analyzed in their spatial expression pattern during development of the plant. The *ZmHox1a/1b* and *ZmHox2a/2b* gene pairs encode putative plant transcription factors and their transcripts belong to a class of rare mRNAs. The spatial expression pattern of the four homeobox genes was found to be very similar in all tested organs. Expression of the *ZmHox1* and *2* genes is activated very early in development and restricted to the embryo proper four days after pollination. After establishment of the root/shoot axis, *ZmHox* transcripts are prevalent in the embryonic root and shoot apical meristems and are found in provascular tissues and young leaf primordia. No signals were observed in the scutellum, the coleoptile or the coleorhiza of the embryo. In the seedling, the transcription of the *ZmHox* genes marks all kinds of meristems of the shoot and root system, the descending proliferating regions and the provascular strands. As the vegetative shoot apical meristem undergoes transition to a floral meristem, *ZmHox* expression persists in the developing tassel. At the same time high transcript levels are detectable in the initiating axillary buds. In differentiated inflorescences *ZmHox* transcripts become more restricted to the nucellar region and the silk of the female flower, and to the innermost layer of the sporangium wall, the tapetum cells of the developing anthers. The data show that all four *ZmHox* genes are expressed in meristems and proliferating tissues where developmental decisions contribute to the ontogeny of the plant. The overall expression pattern suggests that the *ZmHox* class of homeobox genes might be involved in transcriptional control during development from the embryonic to the reproductive phase, which is supported with the pleiotropic effects observed in transgenic tobacco plants.

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Location and designation of 3 EMS-induced dominant mutations
--M.G. Neuffer and Dan England

D10 (*D*-2428*) on chromosome 2L

A dominant EMS-induced dwarf mutant was located to chromosome 2L using the *wx*-marked reciprocal translocation method. This small, erect-leaf dwarf has leaves of proportionally normal width, slender stalk, and shortened internodes. It is not andromonoecious, with a small ear, and just a fair tassel that sheds good pollen. Tillers with a tassel seed expression are often present. The data showing linkage with *wx* T2-9d are presented below. Tests of several other chromosomes were negative.

Wx D	*Wx/+*	*wx D*	*wx/+*	Rec % SE
114	27	12	126	.14 +/- .02

Les20 (*Les*-2457*) on chromosome 1L

A dominant EMS-induced small chlorotic spot lesion mutant was tested for location to chromosome with the full set of *wx*-marked translocations. Linkage data with *wx* T1-9c and *wx* T1-9(4995) indicate location on the proximal one-third of 1L. Tests of several other chromosomes were negative.

Marker	*Wx Les*	*Wx/+*	*wx Les*	*wx/+*	Rec % SE
T1-9c	28	10	9	35	.23 +/- .05
T1-9(4995)	36	3	3	43	.07 +/- .03

Wi4 (*Wi*-2445*) on chromosome 5

A dominant EMS-induced chronically wilted mutant was shown, as indicated by the following data, to be linked with *wx* T5-9c. Other chromosome tests were negative.

Wx Wi	*Wx/+*	*wx Wi*	*wx/+*	Rec % SE
39	9	14	29	.25 +/- .046

Induced mutations with confirmed locations
--M.G. Neuffer and Dan England

Below are listed 458 recessive mutations with confirmed locations, with a brief notation indicating how they were located (if located by another investigator, the investigator's name is noted in parenthesis). The mutants are ordered by chromosome, then alphabetically by lab symbol. Of these, 226 have been given gene symbols in a previous report, but 232 have not been designated and this constitutes the first report for nearly all this group. We are in the process of collecting samples of each to send to the Coop, but samples can also be obtained directly from us. All these mutations have been listed in the maize database, and we have therefore listed our lab symbols as they appear there.

Lab Symbol	Gene	How confirmed
Chromosome 1S		
ad-N605B*		TB-1Sb
blh-N487C*		*wx* T1-9a
blh-N495B*		TB-1Sb
bnk-N1519C*		TB-1Sb
clf-N1348*	*dek1-1348*	TB-1Sb
clf-N1394*	*dek1-1394*	TB-1Sb
clf-N1401*	*dek1-1401*	TB-1Sb
clf-N792*	*dek1*	TB-1Sb
clf-N928A*	*dek1-928A*	TB-1Sb
clf-N971*	*dek1-971*	TB-1Sb
cp-N1009*		TB-1Sb
cp-N1078B*		TB-1Sb
cp-N1308A*		TB-1Sb
cp-N1393A*		TB-1Sb
cp-N1399A*		TB-1Sb
cp-N628*		TB-1Sb
cp-N991*		TB-1Sb
dcr-N1176B*		TB-1Sb
de-N1057B*		TB-1Sb
de-N1142*		TB-1Sb
de-N1162*		TB-1Sb
de-N1345B*		TB-1Sb
de-N1390A*		TB-1Sb
et-N617*		TB-1Sb
et-N745*		TB-1Sb
fl-N1208A*		TB-1Sb
gm-N1390C*		TB-1Sb
hcf-N1242B*	*hcf9*	*hcf3;* TB-1Sb
hcf-N1268B*	*hcf31*	TB-1Sb
hcf-N228B*	*hcf6*	TB-1Sb
hcf-N846B*	*hcf3*	TB-1Sb
l-N515*	*l16*	TB-1Sb
les-N501B*	*lls1-501B*	allelism
pg-N484A*		TB-1Sb
pg-N484B*		TB-1Sb
pg-N526C*		TB-1Sb
pg-N619*		TB-1Sb
ppg-N340B*	*pg15*	TB-1Sb
ptd-N923*		TB-1Sb
sh-N1322A*	*dek32*	TB-1Sb
smk-N1057A*		TB-1Sb
vp-N1136B*		TB-1Sb
vp-N1492*	*vp5-1492*	allelism (Burr)
vp-N1495*	*vp5-1495*	allelism (Burr)
vp-N1496*	*vp5-1496*	allelism (Burr)
vp-N2211*	*vp5-2211*	allelism (Burr)
vp-N81*	*vp5-81*	allelism
wl-N650A*		TB-1Sb
Chromosome 1L		
ad-N613B*		TB-1La
bl-N43*		TB-1La
cl-N801*		TB-1La
cp-N1113A*	*dek22*	TB-1La
cp-N1311C*		TB-1La
cp-N888A*	*cp3*	TB-1La hypo
cp-N918A*		TB-1La
d-N1352B*		TB-1La
d-N1883*		*bz2* linkage
d-N1895*		*d*-1883* allelism
d-N454A*		TB-1La
de-N1310B*		TB-1La
de-N1420*		TB-1La
de-N978*		TB-1La
dnt-N1185A*		TB-1La
dsc-N1315A*	*dek2*	TB-1La
et-N1001A*		TB-1La
gm-N1303*		TB-1La
hcf-N1097B*	*hcf13*	TB-1La
hcf-N1251*	*hcf12*	TB-1La
hcf-N1275C*	*hcf41*	TB-1La
hcf-N1278B*	*hcf44*	TB-1Lc
hcf-N1481*	*hcf50*	TB-1Lc
hcf-N506C*	*hcf2*	TB-1La
id-NA0972*	*id1-972*	allelism
idd-N2286A*	*idd1*	*an1, id1* allele
ij-N8*	*ij2-8*	TB-1La
l-N544*	*l17*	TB-1La
nec-N360B*	*hcf4*	TB-1Lc
pg-N1822A*		TB-1La
pg-N219*	*pg16*	TB-1La
py-N521A*	*py2-521A*	TB-1La
smp-N706A*		TB-1La
spc-N262A*	*spc2*	TB-1La
v-N1806*		TB-1La
v-N245*		TB-1La
v-N55*		TB-1La
w-N1890*		TB-1La
w-N495A*	*w18*	TB-1La
w-N547A*		TB-1La
w-N571C*	*w18-571C*	TB-1La
wl-N1831*		TB-1Lc
wl-N266A*	*wtu5*	TB-1La

wl-N47*		TB-1La
wl-N56*		TB-1La
wl-N60*		TB-1La
wl-N709B*		TB-1La
zb-N101*	*zb7*	TB-1La
Chromosome 2S		
cp-N1076A*		TB-3La-2S6270
cp-N1319A*		TB-2Sa
d-N155B*		TB-2Sa
d-N208B*		TB-3La-2S6270
dcr-N1233A*		TB-2Sa
de-N1122A*		TB-3La-2S6270
fl-N1426*		TB-3La-2S6270
gm-N1289*	*dek3*	TB-3La-2S6270
gm-N1312*		TB-2Sa
nec-N1119B*		TB-2Sa
nec-N516B*	*nec4*	TB-3La-2S6270
o-N1189A*		TB-3La-2S6270
ptc-N2284B*		TB-2Sa hypoploid
ptd-N901A*		TB-2Sa
v-N453A*	*v26*	TB-3La-2S6270
v-N605A*	*v26-605A*	TB-3La-2S6270
ws-N2357*	*ws3-2357*	allelism
wt-N136A*		TB-2Sa
wt-N178C*	*wt1-178C*	allelism
wt-N472A*	*wt1-472A*	TB-3La-2S6270
wl-N666B*	*wt1-666B*	TB-3La-2S6270
Chromosome 2L		
clf-N1024A*	*dek4*	TB-1Sb-2L4464
cp-N1225B*		*de*-660C* allelism
dcr-N1428*	*dek23*	TB-1Sb-2L4464
de-N1175*		TB-1Sb-2L4464
de-N660C*		TB-1Sb-2L4464
et-N1078A*		TB-1Sb-2L4464
fl-N1287*		TB-1Sb-2L4464
fl-N1414*	*dek16*	TB-1Sb-2L4464
hcf-N1253A*	*hcf15*	TB-1Sb-2L4464
hcf-N490B*	*hcf1*	TB-1Sb-2L4464
l-N1940*	*l18*	TB-1Sb-2L4464
mn-N1120A*		TB-1Sb-2L4464
o-N1195A*		TB-1Sb-2L4464
spt-N464*	*spt1*	TB-1Sb-2L4464
spt-N579B*		TB-1Sb-2L4464
v-N350*	*v24-350*	TB-1Sb-2L4464
v-N424*	*v24*	TB-1Sb-2L4464
v-N576A*	*v24-576A*	TB-1Sb-2L4464
v-N588A*	*v24-588A*	TB-1Sb-2L4464
w-N332*		TB-1Sb-2L4464
w-N346*		TB-1Sb-2L4464
w-N77*		TB-1Sb-2L4464
Chromosome 3S		
cp-N1283*	*dek24*	TB-3Sb
d-N339*	*d1-339*	TB-3Sb
d-N446*	*d1-446*	TB-3Sb
dcr-N1053A*		TB-3Sb
et-N1339A*	*dek5-1339A*	TB-3Sb
gl-N169*	*gl19*	TB-3Sb
hcf-N1257B*	*hcf19B*	TB-3Sb
rgh-N1112*		TB-3Sb
sh-N874A*	*dek5*	TB-3Sb
si-N1323*		TB-3Sb
wl-N2*	*cl1-2*	TB-3Sb
Chromosome 3L		
a-N796*	*a1-796*	TB-3La
bif-N2354*	*bif2*	TB-3La hypoploid
bz-N1649*	*a1-1649*	allelism
cp-N1379A*		TB-3La
cp-N1436A*		TB-3La
cp-N330D*	*dek17*	TB-3La
crp-N2207*		TB-3La
d-N210*		TB-3La hypoploid
d-N282*		TB-3La
de-N1126A*		TB-3La
de-N1166*		TB-3La
de-N932*		TB-3La
et-N1322C*		TB-3La
gl-N352A*		TB-3La
gl-N672B*		TB-3La
gm-N1311B*		TB-3La
hcf-N1280B*	*hcf46*	TB-3La
nec-N720C*		TB-3La
nj-N1695*	*vp1-1695*	allelism
p-N1703*	*a1-1703*	allelism
pa-N1687*	*vp1-1687*	allelism
rgh-N1060*		TB-3La
rgh-N802*		TB-3La
sh-N2307*	*sh2-2307*	allelism
sh-N2340*	*sh2-2340*	allelism
sh-N391B*	*sh2-391B*	allelism
sh-N627D*	*dek6*	TB-3La
smk-N1168A*		TB-3La
smp-N1324B*		TB-3La
spc-N553C*	*spc3*	TB-3La
su-N748A*		TB-3La
v-N1886*		TB-3La
wl-N28*	*wlu1*	TB-3La
wl-N4*		TB-3La
Chromosome 4S		
cb-N719A*		TB-4Sa
cp-N1313*		TB-4Sa
de-N929*		TB-4Sa
et-N788*	*dek11*	TB-4Sa
hcf-N1261A*	*hcf23*	TB-4Sa
la-N2020*	*la1-2020*	allelism
nec-N562*		TB-4Sa
nec-N673B*		TB-4Sa
o-N1119A*		TB-4Sa
o-N1228*		TB-4Sa
o-N1244A*		TB-4Sa
orp-N1186A*	*orp1*	TB-4Sa hypoploid
pg-N1881*		TB-4Sa
pg-N673A*		TB-4Sa
sh-N1105B*		TB-4Sa
sh-N1167A*	*dek25*	TB-4Sa
sh-N1324A*		TB-4Sa
sh-N1519B*		TB-4Sa
smp-N156A*		TB-4Sa
spt-N1269A*	*spt2*	TB-4Sa
su-N1161A*	*su1-1161A*	allelism
su-N1968*	*su1-1968*	allelism
su-N1994*	*su1-1994*	allelism
su-N211C*	*dek7*	TB-4Sa
su-N2313*	*su1-2313*	allelism
su-N2314*	*su1-2314*	allelism
su-N851*	*su1-851*	allelism
su-N852*	*su1-852*	allelism
su-N896A*	*su1-896A*	allelism
su-N912*	*dek7-912*	TB-4Sa
su-N959*	*su1-959*	TB-4Sa
wst-N413A*		TB-4Sa
wt-N10*	*wt2*	TB-4Sa
Chromosome 4L		
cl-N1632*	*c2-1632*	allelism
cl-N1640*	*c2-1640*	allelism
cl-N1670*	*c2-1670*	allelism
cl-N1682*	*c2-1682*	allelism
cl-N795*		TB-4Lf
cp-N1176A*	*dek10*	TB-4Lf
dcr-N1005A*	*dek8-1005A*	TB-4Lf
dcr-N1156*	*dek8*	TB-4Lf
nec-N1487*		TB-4Lf
nec-N193*		TB-4Lf
nec-N642*	*nec5*	TB-4Lf
o-N1243*	*o1-1243*	TB-4Lf
o-N1478A*	*o1-1478A*	allelism
o-N1502*	*o1-1502*	allelism
pa-N1637*	*c2-1637*	allelism
pa-N1680*	*c2-1680*	allelism
ptd-N1130*	*dek31*	TB-4Lf
rgh-N1105A*		TB-4Lf
v-N378A*		TB-4Lf
wl-N311B*		TB-4Lf
Chromosome 5S		
ad-N664*		TB-5Sc
cl-N818A*		TB-5Sc
cp-N1430*		TB-5Sc
cp-N931*	*dek18*	TB-5Sc
dcr-N925A*		TB-1La-5S8041
de-N1002A*		TB-5Sc
fl-N1333B*		TB-5Sc
gl-N681A*		TB-5Sc
l-N1838*		TB-5Sc
nec-N409*	*nec3*	TB-1La-5S8041
nec-N493*	*nec6*	linkage

pa-N1634*	*anl1-1634*	allelism
pa-N330C*	*anl1-330C*	TB-5Sc
sh-N1992*	*sh5-1992*	allelism
smk-N1529*		TB-5Sc
vp-N80*	*vp7-80*	allelism
vp-N83*	*vp2-83*	allelism
wl-N44*		TB-5Sc
Chromosome 5L		
bt-N2308*	*bt1-2308*	allelism
bt-N2309*	*bt1-2309*	allelism
cp-N1275A*		TB-5La
cp-N1331*	*dek26*	TB-5La
cp-N1369*		TB-5La
cp-N1380A*	*dek27*	TB-5La
cp-N1385*		TB-5La
cp-N863A*		TB-5La
cp-N935*		TB-5La
crp-N1365*	*dek9*	TB-5La
de-N1196*		TB-5La
dnj-N1534*		TB-5La
fl-N1145A*		TB-5La
fl-N1299*	*dek33*	TB-5La
gl-N166A*	*gl8-166A*	TB-5La
grt-N1308B*	*grt1*	TB-5La
hcf-N1256*	*hcf18*	TB-5La
hcf-N1259A*	*hcf21*	TB-5La
hcf-N1273*	*hcf38*	TB-5La
hcf-N1277B*	*hcf43*	TB-5La
mn-N1536*		TB-5La
nec-N756B*	*nec7*	TB-5La
o-N1065A*		TB-5La
pg-N296A*		*v*-735* allelism
pg-N408C*		TB-5La
pg-N71A*		*v*-735* allelism
ppg-N199*	*ppg1*	TB-5La
pr-N1515A*	*pr1-1515A*	allelism
pr-N1527A*	*pr1-1527A*	allelism
pr-N1621*	*pr1-1621*	allelism
pr-N1629*	*pr1-1629*	allelism
pr-N1631*	*pr1-1631*	allelism
pr-N1638*	*pr1-1638*	allelism
pr-N1644*	*pr1-1644*	allelism
pr-N1646*	*pr1-1646*	allelism
pr-N850*		TB-5La
sct-N206B*	*wgs1*	TB-5La
sh-N2220*	*sh4-2220*	allelism
sh-N2310*	*bt1-2310*	allelism
sh-N797A*	*bt1-797A*	allelism
smk-N1160*		TB-5La
sms-N146C*		TB-5La
v-N26*		TB-5La
v-N473B*		*v*-735* allelism
v-N735*		TB-5La
vp-N1499*	*sh4-1499*	allelism
w-N1126B*		TB-5La
w-N1834*	*pr1-1834*	allelism
w-N1868*	*lw2-1868*	allelism
w-N21A*		TB-5La
w-N22*		TB-5La
ys-N755A*	*ys1-755A*	allelism
zn-N571D*		TB-5La
Chromosome 6S		
hcf-N1263C*	*hcf26*	TB-6Sa
hcf-N510C*	*hcf5*	TB-6Sa
o-N1307A*	*dek28*	TB-6Sa
Chromosome 6L		
de-N1400*		TB-6Lc
gs-N268*	*gs3*	TB-6Lc
hcf-N1269C*	*hcf34*	TB-6Lc
hcf-N1271B*	*hcf36*	TB-6Lc
hcf-N1282C*	*hcf48*	TB-6Lc
l-N113*		TB-6Lc
l-N612B*		TB-6Lc
l-N62*		TB-6Lc
o-N1296A*	*dek19*	TB-6Lc
o-N1320A*		TB-6Lc
o-N1368*		TB-6Lc
o-N1384A*		TB-6Lc
o-N924*	*o14*	TB-6Lc
pg-N1885*		TB-6Lc
ptd-N1425A*		TB-6Lc
sh-N1320B*		TB-6Lc
smp-N272A*		TB-6Lc
stp-N65A*	*sr4*	(J. Beckett)
v-N634A*		TB-6Lc
v-N69A*		TB-6Lc
w-N278A*		TB-6Lb
w-N335*		TB-6Lc
wl-N217A*		TB-6Lc
wl-N358A*		TB-6Lc
wl-N362B*		TB-6Lc
wxl-N2292*	*sbd1*	TB-6Lc hypoploid
Chromosome 7S		
cp-N1294*		TB-7Sc
de-N1136A*		TB-7Sc
o-N2225*	*o2-2225*	allelism
o-NA0696*	*o2-A696*	allelism
o-NA0697*	*o2-A697*	allelism
sh-N1295*	*sh6-1295*	TB-7Sc
vp-N1493*	*vp9-1493*	allelism
vp-N1494*	*vp9-1494*	allelism
vp-N1497*	*vp9-1497*	allelism
vp-N2213*	*vp9-2213*	allelism
Chromosome 7L		
bd-N2355*	*bd1-2355*	allelism
cp-N1104B*		TB-7Lb
cp-N1417*		TB-7Lb
cp-N76B*	*o5-76B*	TB-7Lb
de-N1177A*		TB-7Lb
et-N1332*		TB-7Lb
gl-N1845*		TB-7Lb
gl-N212*	*gl1-212*	TB-7Lb
gl-N269*	*gl1-269*	TB-7Lb
o-N1241*	*o5-1241*	TB-7Lb
o-N1298*		TB-7Lb
o-N1310A*		TB-7Lb
o-N874B*	*o5-874B*	allelism
sh-N1341*		TB-7Lb
smp-N586B*		TB-7Lb
spc-N357A*		TB-7Lb
v-N413C*	*v27-413C*	TB-7Lb
v-N53B*	*v27-53B*	TB-7Lb
v-N590A*	*v27*	TB-7Lb
wl-N543A*	*wlu2*	TB-7Lb
wl-N629A*		TB-7Lb
Chromosome 8L		
blh-N2359*		TB-8Lc hypoploid
cp-N1387A*	*dek29*	TB-8Lc
cp-N1392A*	*dek20*	TB-8Lc
crp-N1058*	*pro1-1058*	TB-8Lc
crp-N1121A*	*pro1-1121*	TB-8Lc
crp-N1429A*		TB-8Lc
crp-N1528*	*pro1-1528*	TB-8Lc
crp-N1533*	*pro1-1533*	TB-8Lc
de-N1386A*	*emp3*	TB-8Lc
fl-N1163*		TB-8Lc
rgh-N1154A*	*pro1-1154A*	TB-8Lc
rgh-N1285*	*rgh1*	TB-8Lc
sh-N1530*	*pro1-1530*	allelism
v-N25*	*v21*	TB-8Lc
v-N29*		TB-8Lc
v-N358C*		hypoploid test
v-N779A*		TB-8La
v-N7B*		*v*-826* allelism
v-N826*		TB-8La
wl-N203A*	*wlu3*	TB-8La
Chromosome 9S		
bz-N1648*	*bz1-1648*	allelism
cl-N1623*	*c1-1623*	allelism
cl-N1625*	*c1-1625*	allelism
cp-N1054*	*dek12-1054*	TB-9Sb
cp-N1092A*		TB-9Sb
cp-N873*	*dek12*	TB-9Sb
d-N660B*	*d3-660B*	allelism
sh-N2221*	*sh1-2221*	allelism
sh-N399A*		TB-9Sb
v-N27*	*v28*	TB-9Sb
v-N585*	*v28-585*	TB-9Sb
v-N610*	*v28-610*	TB-9Sb
v-N697*	*v28-697*	TB-9Sb
v-N828*	*v31*	TB-9Sb
v-N829A*		TB-9Sb
vp-N1498*	*sh1-1498*	allelism
w-N1854*		TB-9Sb

w-N1865*		TB-9Sb
w-N627B*		TB-9Sb
wl-N1803*		TB-9Sb
wl-N1857*		TB-9Sb
wx-N1050A*	*wx1-1050A*	allelism
wx-N1240A*	*wx1-1240A*	allelism
Chromosome 9L		
cp-N1381*		TB-9Lc
dcr-N1409*		TB-9Lc
dsc-N749*		TB-9Lc
et-N357C*		TB-9Lc
fl-N1391*	*dek30*	TB-9Lc
gm-N1319B*		TB-9Lc
hcf-N1276B*	*hcf42*	TB-9Lc
o-N744*	*dek13*	TB-9Lc
pg-N660A*		TB-9Lc
v-N1871*		TB-9Lc
v-N53A*		TB-9Lc
v-N806C*		TB-9Lc
wl-N41A*	*wlu4*	TB-9Lc
Chromosome 10S		
ad-N590C*		TB-10Sc
ad-N647*		TB-10Sc
cp-N1435*	*dek14*	TB-10Sc
gl-N478B*	*gl21*	TB-10Sc
hcf-N1281C*	*hcf47*	TB-10Sc
ij-N504A*		TB-10Sc
l-N425*	*l19*	TB-10Sc
o-N1046*		TB-10Sc
rgh-N1524*		TB-10Sc
Chromosome 10L		
ad-N377B*		TB-10Lb
bf-N185A*	*bf2-185A*	allelism
cl-N1624*	*r1-1624*	allelism
cl-N1627*	*r1-1627*	allelism
cl-N1630*	*r1-1630*	allelism
cl-N1676*	*r1-1676*	allelism
cl-N1694*	*r1-1694*	allelism
cp-N1427A*	*dek15*	TB-10L19
hcf-N1265A*	*hcf28*	TB-10La
l-N1879*		TB-10L20
l-N1908*		TB-10L20
l-N195*		TB-10L20
l-N31*		TB-10L20
l-N392A*		TD-10L20
l-N59A*	*l13-59A*	TB-10L20
msc-N1330*	*w2-dek21*	TB-10L20
o-N1422*		TB-10L20
orp-N1186B*	*orp2*	link to *R*
rgh-N799A*		TB-10L19
v-N114A*		TB-10L20
v-N354B*		TB-10L20
v-N418*	*v29*	TB-10L20
v-N470A*		TB-10L20
w-N24*		TB-10L20
ws-N1877*		TB-10L20

Map location of *anthocyanin3*

--Dawn Robinett, Ed Coe, and Karen Cone

The *anthocyanin3* (*a3*) gene is a recessive intensifier of pigmentation in vegetative tissues of the plant. Previous work by Styles and Coe (J. Heredity, 77:389-393, 1986) established that *a3* maps on chromosome 3 near *anthocyaninless1*(*a1*). To determine the precise map location of *a3*, we undertook a molecular segregation analysis. An F2 population segregating for *a3* was planted and scored for the intense plant pigmentation characteristic of *a3*. DNA prepared from 26 individual intensely pigmented plants was digested with restriction enzymes, blotted and hybridized with probes for *a1* and the nearby distal marker, *umc96*. The following data were obtained:

probe	# chromosomes tested	# crossovers	recombination frequency (%)
a1	52	5	7.7
umc96	52	3	5.8

We conclude that *a3* maps between *a1* and *umc96*. These data effectively expand the interval between *a1* and *umc96* from the previously reported value (BNL map, May 1993) of 9 map units to approximately 13 map units.

Differential methylation of *Pl-Bh* appears to be restricted to cytosines in CG or CNG sequences

-- Michael G. Muszynski and Karen C. Cone

Pl-Bh is hypermethylated at specific *Msp*I/*Hpa*II sites 3' of the coding region compared to *Pl* (Figure 8, Cocciolone and Cone, Genetics 135:575-588, 1993). The *Msp*I/*Hpa*II restriction site is CCGG. *Msp*I is sensitive to methylation at the internal cytosine, while *Hpa*II is sensitive to methylation at either cytosine. Both cytosines are located within either CG or CNG sequences, which are the preferred targets for DNA methylation in plants. Methylation of cytosines outside of these target sequences has been reported for transgenes in petunia (P. Meyer et al., EMBO J. 13:2084-2088, 1994). This observation prompted us to assay for differential cytosine methylation outside of the canonical CG or CNG sequences in *Pl* and *Pl-Bh*. The restriction enzymes *Alu*I (AGCT), *Sau*3AI (GATC) and *Sau*96I (GGNCC), which are all sensitive to cytosine methylation, were used. The restriction sites assayed are located 3' of the coding region and none have a cytosine in a CG or CNG context. Digestion with these enzymes was complete for both *Pl* and *Pl-Bh* . This indicates that there are no methylated cytosines at these sites in *Pl* and *Pl-Bh*. Therefore, *Pl-Bh* appears to only be differentially methylated within CG or CNG sequences. Methods exist for detecting every methylated cytosine of a target sequence (S. Clark et al., NAR 22:2990-2997, 1994). We are currently adapting these methods for use on maize DNA to detect and quantitate methylation at individual cytosines in *Pl* and *Pl-Bh*.

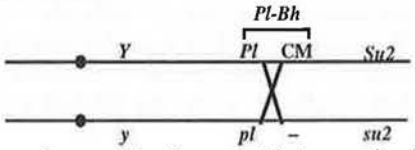
Recombination at *Pl-Bh*

-- Michael G. Muszynski and Karen C. Cone

Pl-Bh is an unusual allele of the anthocyanin regulatory gene *Pl*. Whereas *Pl* pigments vegetative tissues a uniform purple and does not affect kernel pigmentation, *Pl-Bh* pigments both vegetative tissues and kernels in a variegated and irregular manner (Cocciolone and Cone, Genetics 135:575-588, 1993). *Pl* and *Pl-Bh* have essentially identical nucleotide sequences in almost 7 kb of DNA including flanking and coding regions (O. Hoekenga, MNL this issue). *Pl-Bh* does differ from *Pl* by being hypermethylated at specific *Msp*I/*Hpa*II sites 3' of the coding region. The hypermethylation of *Pl-Bh* is correlated with altered *Pl* gene expression in husks. The mutation responsible for both the altered expression and the hypermethylation is considered *cis*-linked because it always co-segregates with the *Pl* locus. The following model explains how increased methylation may lead to the blotched phenotype. We envision an altered chromosome structure (Center of Modification or CM), located at a *cis*-linked site some distance from the gene, where a DNA methylation signal initiates. This signal is propagated stochastically along the chromosome toward *Pl-Bh*. In some cell lineages, the methylating signal moves through *Pl-Bh*, the gene becomes hypermethylated, and expression is altered. In other cell lineages, the signal does not reach *Pl-Bh*, the gene is not hypermethylated, and expression is normal.

The model predicts that *Pl* linked to CM yields the blotchy phenotype. In an effort to genetically define the CM, we attempted to separate it from the *Pl-Bh* sequence by recombina-

tion. Our rationale was that uncoupling the essentially normal *Pl* sequence from the CM by recombination should result in normal *Pl* expression. The following cross was set up and both proximal and distal morphological and RFLP markers were used to verify recombination.



X y pl – su2

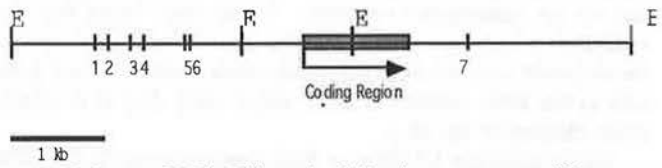
A recombination event between the *Pl* sequence and the CM should result in normal *Pl* expression. A plant carrying this recombinant chromosome would be recognized as having uniform purple pigmentation as opposed to sun-red (*pl*) or blotchy (*Pl-Bh*) pigmentation. Approximately 20,000 plants were screened during the past two summer pollinating seasons. No purple recombinant plants were found. This indicates a genetic distance of < 0.01 cM between the CM and the *Pl* locus. Alternatively, the CM may act as a suppressor of recombination such that crossovers between the CM and *Pl* locus may not be recoverable.

Ten nucleotide differences exist between *Pl* and *Pl-Bh* within a 7 kb region

--Owen A. Hoekenga and Karen C. Cone

Pl and its allele *Pl-Bh* are regulatory genes in the anthocyanin biosynthetic pathway. *Pl* conditions uniform purple pigment in the vegetative parts of the plant. *Pl-Bh* conditions pigment in the vegetative parts of the plant, as well as in the aleurone of the kernel. This pigmentation is not uniform, but variegated. Thus, relative to *Pl*, *Pl-Bh* has both a gain of function — increased tissue specificity — and a loss of function — variegated expression. We assumed that this striking difference in phenotypes would be mirrored by an equally striking difference in DNA sequence. Initial studies did not confirm this hypothesis; *Pl-Bh* has a single nucleotide insertion, approximately 630 bp downstream of the stop codon, relative to *Pl* (Cocciolone and Cone, *Genetics* 135:575, 1993). To extend the sequence analysis, I sequenced an additional 2.5 kb region upstream of the gene. Within this region, there are nine nucleotide differences. Over the 7 kb sequenced to date, there are ten nucleotide differences between *Pl* and *Pl-Bh*, as summarized below.

Restriction map of *Pl* and *Pl-Bh* showing location of differences. E, EcoRI; B, BamHI.



Relative to *Pl-Bh*, *Pl* has the following sequence differences: single nucleotide deletions at sites 1, 4, and 7; single nucleotide insertions at sites 3 and 5; two single nucleotide insertions, two nucleotides apart, at site 6; two cytosine to thymine transitions and a single nucleotide insertion within a twenty-five nucleotide span at site 2. We do not yet know if these ten nucleotide differences are responsible for the difference in phenotype observed between *Pl* and *Pl-Bh*. We are investigating this possibility as well as alternative hypotheses to explain the observed differences between *Pl* and *Pl-Bh* (see also Muszynski and Cone, MNL this issue).

Multi-target directed (targeted) approach for transposon tagging

--Guri Johal and D. Gruis

Transposon tagging is the most efficient method for cloning genes in maize that are known only by their mutant phenotypes. Dozens of genes have been cloned by this method over the last 10-12 years. However, the success rate of this method has been depressingly low. In addition, the method requires several years for completion, and is very labor- and resource-intensive. As a consequence, transposon tagging has been considered more of an adventure than a method to rely upon for routine isolation of genes.

One reason for these drawbacks is that often a single locus is used as a target in a tagging project. Typically, a tester stock, homozygous recessive for a gene of interest, is either developed or acquired from the maize Co-op or a fellow scientist and is crossed mostly as a pollen parent to a stock carrying the transposons. Since only a single locus (target) is present in the homozygous tester, mutations involving only that locus are uncovered in the F1, unless the mutational event generates an allele that shows dominance over the wild-type allele. Such dominant mutations are however very rare, at least 100 times less frequent, compared to recessive mutations.

In order to increase the utility of transposon tagging we asked if it would be possible to tag more than one gene in one mutagenesis experiment. We found that such a strategy is possible. In a stock showing transposable element activity, transposons can and probably do insert into genes throughout the genome, and the gametes produced by this stock may contain the entire set of tagged genes. However, a mutation will be generated, or more appropriately recovered, in a gene for which the gametes coming from the tester parent are also defective. If these gametes are mutant for more than one locus, plants showing mutant phenotypes for all of these loci can be obtained in a single experiment. We proved this contention in a tagging experiment where *Mutator* active stocks were crossed with the Mangelsdorf's tester. This multiple gene tester is homozygous recessive at ten different loci, one on each of the ten chromosomes. An F1 progeny of 60,000 was planted last summer in Columbia and mutant plants showing phenotypes for each of the ten recessive loci were recovered. All of these mutants are available upon request.

The frequency of mutation, however, was vastly different at different loci. Compared to more than 15 mutants for both *brown midrib2* (*bm2*) and *japonica1* (*j1*), only one *golden1* and two *liguleless1* (*lg1*) mutants were found in this progeny. Most of our *Mutator* stocks were acquired from Steve Briggs (Pioneer Hi-Bred Int'l Inc.) and Pat Schnable (Iowa State University), and the progeny of each stock was kept separate from each other. Judging from mutations at the *bm2*, *j1* and *a1* loci, it was realized that mutation frequency of these loci was stock-dependent. Some stocks were more efficient, 10-20-fold compared to others, in generating mutations at the *bm2* locus, and similar stock-dependent mutation efficiency was also evident for the *a1* and *j1* loci. Presently, it is unclear why different *Mutator* stocks differ in their ability to cause mutations at different loci, but it is possibly the result of uneven distribution of *Mu* elements on individual chromosomes in different stocks. A clustering of *Mu* elements has been reported (Ingels et al., J. Heredity 83:114, 1992). Although not shown for *Mutator* yet, transposition to linked sites is one mechanism that can cause clustering of *Mu* elements and also the bias among stocks for generating mutations at different loci in the

genome.

In summary, the genetic scheme described above can allow the tagging and cloning of multiple genes in a single project and can change transposon tagging from an adventure into a highly rewarding venture. Although we used a tester that was homozygous recessive for multiple loci, a tester heterozygous for many genes will also work. The only drawback with a heterozygous tester is that half of the tagged mutant alleles will not be recovered. However, if we can identify chromosome specific *Mutator* stocks that can produce mutations at a frequency of $1x10^{-4}$, losing half of the tagged gametes will have an insignificant effect on the success of the tagging project.

PCR-based strategies to speed up cloning after tagging with *Mutator*

--Guri Johal and John Gray

The *Mutator* (*Mu*) transposable element system is the most efficient system for tagging genes in maize. This is largely due to the transposition of hundreds of *Mu* elements that are present in active *Mutator* lines. Although the high copy number increases the chances of getting an insertional mutation, it also is the root cause of a significant problem, i.e., to determine which of hundreds of *Mu* elements in the mutant has caused the mutation. The limited resolution of ordinary agarose gels makes it impossible to identify a *Mu* element that cosegregates with the mutant allele, unless the number of background *Mu* elements is decreased. This is usually done by outcrossing the mutant a few times with an inbred that lacks *Mutator* activity. During these crosses, *Mu* elements unlinked with the mutation are segregated out. This however, takes a couple of seasons, and sometimes it also leads to the suppression of the mutant phenotype making the cosegregation analysis impossible. Another problem with the *Mutator* system is that although nine different types of *Mu* elements have been identified, the mutation of interest may be caused by a *Mu* element that has not been identified yet.

A couple of PCR-based approaches have been conceived to simplify and speed up the cloning process once a mutant or even a mutant sector is obtained during a *Mutator* tagging project. These strategies are based on employing a Mu-TIR primer (preferably from the end sequence) as an anchor primer. The other primer can be used randomly or ligated onto the other end by a method devised for the ligation mediated PCR (Mueller and Wold, Science, 246:780, 1989). Following PCR, a polymorphism will be sought between the products derived from DNA extracted from tissue (sector or pooled plants) that harbors the mutation and tissue (sector or pooled plants) that lacks the mutation. One problem that can affect the success of this approach is that maize plants, including *Mu* inactive stocks or inbreds, contain hundreds of TIR's. Thus, hundreds of products may be amplified, confounding their resolution even on a sequencing gel. To alleviate this problem, 4-16 different types of the anchor primers can be made by introducing one to two random nucleotides on the 3' end of the TIR sequence, and different anchor primers may be used singly or in combinations in different reactions to impose selectivity on the PCR products that can be amplified. Although it will increase the number of reactions, it can dramatically reduce the number of PCR bands in a given reaction, making them resolvable, and thereby increasing the chances of identifying a polymorphic product.

This procedure can conceivably also be used at the transcript level. When performed on RNA, the procedure may not even require multiple species of the Mu-TIR anchor primer, and RNA ligase can be used to ligate a specific primer at the other end of the cDNA (as in the 5' RACE technique). The instability of the mutant transcript can be a problem, however. In *Mu*-induced mutations that show suppressible properties, a modification of this procedure, in which two anchor primers, one coming from the Mu-TIR and the other containing poly T, may be even more specific, and therefore may be more successful, in the identification and cloning of a tagged gene.

Does maize endosperm undergo apoptosis?

--Guri Johal and M. Mouw

In maize, like all monocots, the endosperm is the tissue where food reserves are stored. Developmentally, it is fated or programmed to be cannabilized by the seedling during the first 2-3 weeks of growth following germination. Thus, the death of endosperm is a desirable event, but exactly when this death initiates and how it progresses is not known. In animals, one of the hallmarks of cells or tissues that undergo such programmed cell death, often termed apoptosis, is the degradation of chromatin into nucleosome-sized fragments. These degradation products can be visualized as a DNA ladder of ~180 bp by electrophoresis on agarose gels.

In a preliminary experiment designed to study the fate of DNA of maize endosperm, we found that most of the DNA is degraded in a mature kernel. In a B73 inbred, this degradation first becomes obvious during the 5th week after pollination, and by the time endosperm is seven weeks old, most of the DNA is degraded. However, we have been unable to detect intranucleosomal fragmentation of chromatin so far. Although in our experiment degradation was not seen till the 5th week after pollination, an allele-specific loss of some RFLP markers, which may represent chromosomal foci where DNA degradation first initiates, was observed as early as 18 days after pollination by Yerk et al. (MNL 67:103, 1993).

Observations on the effect of light on the progression of *lethal leaf-spot* lesions

--P.S. Close, J. Gray and G. Johal

We examined the effect of light on lesion development in the *lethal leaf spot1* (*lls1*) mutation of maize. Precedent for this approach was established by Craig Echt (MNL 60:49, 1986), who reported that light is necessary for the initiation of *Les1* lesions but not for subsequent necrosis. It was also found that the relationship between photosynthetic activity and *Les1* lesion development was not straightforward since lesions formed quite well in the white sectors of *wd1*, ring-9 (*Wd, C-I*) or *j1* plants (Dave Hoisington loc. cit.).

Using aluminium foil strips or thick paper covering to eliminate or reduce light immanent on the leaf surface, we found that *lls1* lesions do not initiate in the dark and also that existing lesions do not propagate outwards. Lesions continue to form and propagate on upper and lower leaf parts that remain open to light. Lesions that are initiated due to wounding were also found to require light for continued propagation.

Using plexiglass filters of varying transmittance values we also found that wavelengths between 650 and 700 nm were required for lesion formation, and other wavelengths were not adequate.

This region of the light spectrum spans the action spectra of both phytochrome-mediated processes and the 700 nm photosynthetic reaction center. We cannot at present determine the relative contribution of these mechanisms to the lesion development process.

Using an *lls1-ij1* double mutant we examined the requirement for a functional chloroplast in lesion formation. In contrast to *Les1* type lesions *lls1* lesions were found to initiate only in the pale or dark green sectors of these plants but failed to develop in the albino sectors. Interestingly, it appears that a lesion initiated in a green sector separated by a narrow albino sector from a second green sector can "traverse" the albino sector and continue to propagate in the second green sector.

These results are consistent with the hypothesis that free radical generation is part of the process that leads to lesion initiation and/or propagation in the *lls1* mutant plant. Both wounding and photosynthetic activity may contribute to free radical generation but the ability to dismute active radicals may be diminished in *lls1* plants. Studies with free radical scavengers and free radical generators as well as the creation of other double mutants are being conducted to further investigate this phenomenon.

Anatomical stalk changes associated with selection for rind penetrometer resistance

--Oscar Heredia-Díaz

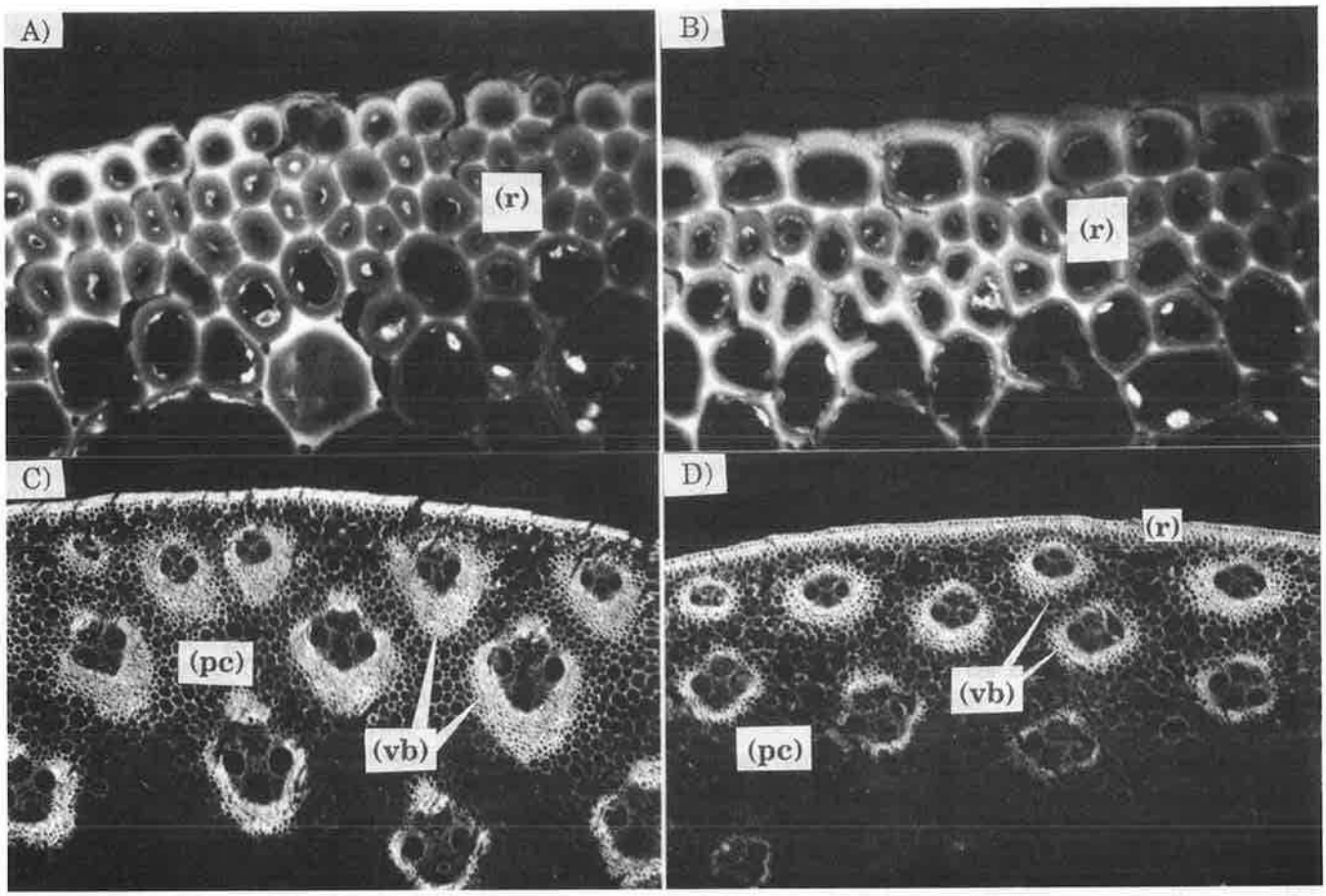
Hunter and Dalbey (Am. J. Bot. 24:492-494, 1937) studied histological structures in maize looking for accurate indicators of the relative degree of stalk breakage in the field. Since Sibale et al. (Maydica 37:111-114, 1992) compared the mechanical force gage (manual), vs. a modified-digital force gage (electronic) rind penetrometer, studies at the University of Missouri (Masole, M. Sc. thesis, 1993; Chumo, Ph. D. thesis, 1993; and Alsirt, M. Sc. thesis, 1993), have shown that the Missouri-modified electronic rind penetrometer was easier, more accurate, and more consistent in ranking genotypes (CVs were 7.5, 10.4, and 14.8%, respectively, for the three thesis studies). The benefit of using the rind penetrometer is greater because it is a non-destructive method; therefore, selection can be done before anthesis and the selected plants can then be intermated in the same season. This allows for completion of one cycle of selection per season in a recurrent selection program.

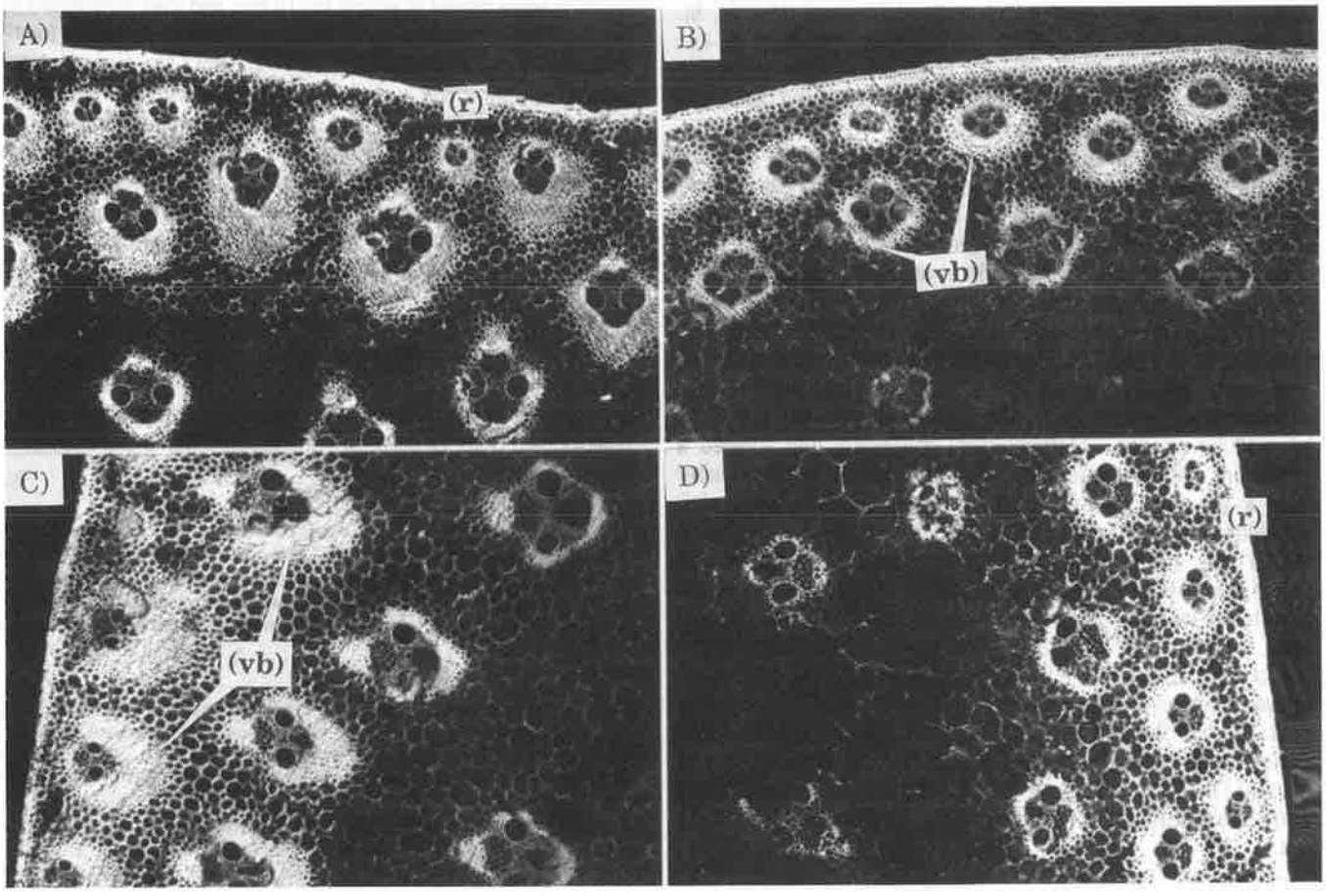
In maize, stiff-stalked genotypes have been characterized by thick bundle sheaths, thick walled, sub-epidermal sclerenchyma, and more angular, close-fitting cells. Weak stalks were characterized by thin walls, narrow bundle sheaths, and large intercellular spaces. Morphological characters that showed a consistent relationship to stalk strength were low bundle number mm^{-2} in the rind, high percentage of sheath $bundle^{-1}$, large stalk diameter, and wide lignified zone (Magee, Iowa State Coll. J. Sci. 22:257-268, 1948). The extent of lignification beyond the rind and the number of vascular bundles within this lignified area were correlated with lodging resistance in maize (McRostie and MacLachlan, I. Sci. Agr. 22:307-313, 1941). Zuber and Grogan (Crop Sci. 1:378-380, 1961) found a significant correlation between mechanical strength and rind thickness. The number of vascular bundles was lower, the thickness of the sclerified parenchyma was greater, and thickness of bundle sheaths was greater in strong-stalked as compared to weak-stalked genotypes (Kálmán et al., Acta Biol. Szeged 21:[1-4],35-40, 1975).

The objective of this project was to identify changes in stalk anatomy in response to bi-directional selection for rind penetrometer resistance. Stalk sections of a representative plant from each extreme of selection, C_6 high (RPR=19.9 load-kg) and C_6 low (RPR=2.9 load-kg), were obtained from the middle portion of the internode used for selection for RPR. Stalks were cut into about 5-mm thick cross sections and fixed in a formaldehyde-alcohol-acetic acid (FAA) solution (50 mL 95% EtOH, 5 mL glacial acetic acid, 10 mL 37-40% formaldehyde, and 35 mL dH_2O). Sections were fixed for 48 h, taken out of FAA, and washed in 70% and 50% EtOH for 1 h each. After washing, sections were put in Hepes (N-[2-Hydroxyethyl] piperazine-N'-[2-ethanesulfonic acid], Sigma Co., St. Louis, MO) buffer for 3 to 4 h, changing the buffer twice during this time, and leaving the samples in Hepes buffer for long-term storage. Hepes buffer keeps samples at physiological pH (7.2 to 8.2), better preserving microstructures. Attempts were made to obtain thin sections (10 to 20 mm) using a Cryo-Stat microtome (Jung Frigocut 2800N, Leica Instruments GmbH, Nussloch, Germany). The Cryo-Stat microtome requires small samples mounted in a plastic polymer Histo-Prep (2-phenoxyethanol, polyoxypropylene-polyoxyethylene block copolymer, Fisher Sci., Fair Lawn, NJ) embedding media and frozen at temperatures below 0°C. Cryo-sectioning is a fast procedure designed to obtain numerous sections in a relatively short period of time. Unfortunately, the different consistency of tissues, specifically those from C_6 high, made it impossible to obtain tear-free sections. Alternatively, samples embedded in paraffin and sectioned at 10 µm with a manual microtome were successfully obtained for both cycles. To eliminate paraffin from sections once they were mounted on the glass microscope slide, sections were placed in xylene for 10 min and then rehydrated by placing them in two washes of 100% EtOH and 95% EtOH, for 10 min twice each, and once each in 70% dH_2O and Hepes. Non-stained preparations were used for observations under the confocal microscope. To identify patterns of lignin accumulation, a stain specific for lignin developed by Conn (Biological Stains, 4th ed., The Williams and Wilkins Co., Baltimore, MD., 1953) was used. The applicability of this stain is for general plant tissues, especially meristematic tissue, and it is designed to show chromatin, spindles, cellulose, and lignified walls. The staining procedure involved: (i) removal of paraffin from sections, (ii) staining for 30 to 50 min in 1% aqueous safranin-o, (iii) rinsing in dH_2O, (iv) counterstaining with 0.2% fast green in EtOH for 1 to 1.5 min, and (v) rinsing in absolute EtOH. Stained sections were permanently fixed on the slide by saturating with polymer Mowial (Calbiochem Corp, La Jolla, CA) and covered with a glass cover.

Fig. 1 (following page, top) Confocal micrographs of stalk cross sections of C_6 HRP and C_6 LRP. Compare: A) Rind of C_6 HRP (60X); B) Rind of C_6 LRP (60X); note more layers of rind parenchyma cells and thicker interlumen region in A as compared to B; C) C_6 HRP (4X); and D) C_6 LRP (4X); note more angular shape and larger inward portion of vascular bundle sheaths of B as compared to C. Rind = (r); parenchyma cell region = (pc); and vascular bundle-sheaths = (vb).

Fig. 2 (following page, bottom). Confocal micrograph comparison of stalk cross sections of C_6 HRP (A and C) and C_6 LRP (B and D) (4X). Note the more angular shape and larger inward portion of vascular bundle sheaths of A and C compared to B and D. Also note that the parenchyma cell region (pc) in C extends further inward. Rind = (r); parenchyma cell region = (pc); and vascular bundle-sheaths = (vb).


A)
(r)
B)
(r)
C)
(pc)
(vb)
D)
(r)
(vb)
(pc)


A)
(r)
B)
(vb)
C)
(vb)
D)
(r)

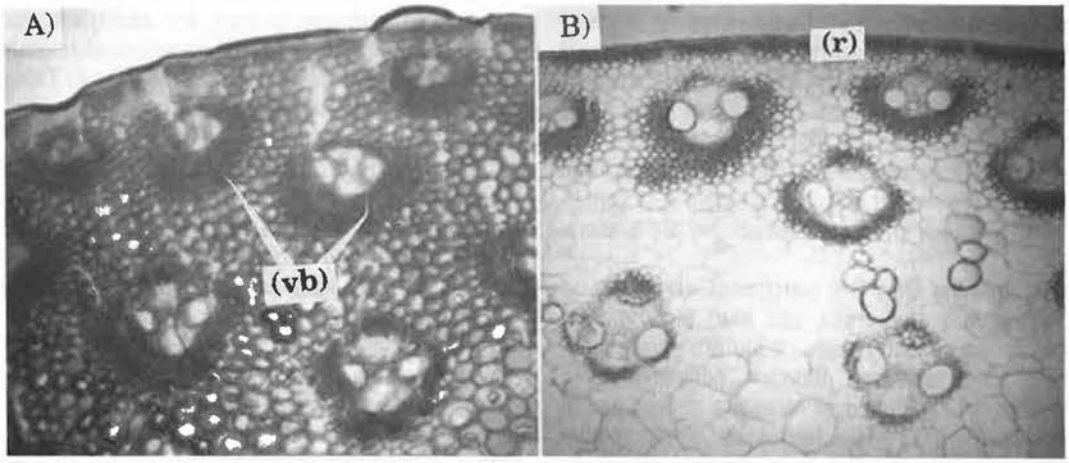


Fig. 3. Light micrographs comparing staining for lignin in stalk crosssections of C_6 HRP (A) and C_6 LRP (B) (4X). Rind (r), parenchyma cell region (pc) and vascular bundle-sheaths (vb) are shown. Note a more general retaining of the stain and more heavily stained vascular bundle sheaths in A as compared to B. Stain also concentrated on the vascular bundle-sheaths shown in Figs. 1 and 2.

Fixed slides were observed under a MRC-600 (BIO RAD, Cambridge, MA) laser scanning confocal microscope and digitized with a Nikon (Optiphot, Nikon Corp., Tokyo, Japan) video camera at the University of Missouri-Columbia Molecular Cytology Core facility. Taking advantage of tissue autofluorescence, images from non-stained preparations of C_6 high and C_6 low were captured in a Macintosh Quadra (Model 840 AV, Apple Computer, Inc., Cupertino, CA) computer and dye-sublimation-printed on thermal graphic paper. Preparations stained with safranin-o were observed under a bright-field light microscope and images were captured by an Image-1/AT (Fryer Co., St. Louis, MO) image analysis system. Final printing was also done on a dye-sublimation printer (Codonics, Inc., Akron, OH).

Sectioning the C_6 low stalk material was easier than C_6 high, apparently because stalks from C_6 low have more similar rind and pith tissue consistency, whereas the C_6 high stalks have two different tissue consistencies with the rind being much tougher than the pith. The main differences between sections of C_6 low vs. C_6 high as observed under the confocal microscope were that in C_6 high: i) the rind, mainly composed of parenchyma cells, was denser, and perhaps had one or two extra layers of cells; ii) the parenchyma cell walls were thicker; iii) a more angular shape of vascular bundle sheath region, defined by a greater density of cell layers forming the sheath; and iv) the inward portion of the rind vascular bundle sheaths (sclerenchyma cells) was larger (Fig. 1). Stalks from C_6 high have a parenchyma cell region (in which the rind vascular bundles are contained) that extends further towards the center of the stalk than in C_6 low stalks (Fig. 2). These findings agree with those previously reported by Magee (1948), Kálmán et al. (1975), Berzonsky et al. (Crop Sci. 26:482-488, 1986), although the mechanisms that produced such changes may be different. No apparent difference in vascular bundle numbers between C_6 low and C_6 high was observed, which contrasts with the findings in MoSQA and MoSQB, whose vascular bundle numbers were greater in the high than in the low selections (Chang et al., Crop Sci. 16:621-625, 1976; and Berzonsky et al., 1986). However, the density of vascular bundles area^{-1} was greater in the C_6 high stalks because the stalk diameter was smaller in C_6 high than in C_6 low.

In an attempt to identify differences in lignification, cross-sections of C_6 low and C_6 high were stained for lignin using o-safranin and fast green. This particular stain is designed to show cellulose and lignified structures in a red color. Histological sections of the internode used in selection for RPR showed differences in accumulation of lignin around the vascular bundles of plants from the extremes of selection (C_6 low vs. C_6 high; Fig. 3). It is likely that the differential in lignin accumulation is a correlated response to selection, rather than a direct response. The staining patterns agreed with vascular bundle sheath patterns shown in Figs. 1 and 2. Rind vascular bundle sheaths in C_6 high were more heavily stained than those from C_6 low, indicating a greater lignification of cell walls in the C_6 high stalks.

Characterization of C_6 high agrees with results reported by Chang et al. (1976) and McRostie and MacLachlan (1941). Chang et al. (1976) indicated that MoSQB, selected for high stalk crushing strength, had more heavily lignified vascular bundles, whereas McRostie and MacLachlan indicated that lodging-resistant lines had considerably more lignification within the rind than lodging-susceptible lines.

A possible mechanism by which plants selected for high RPR achieve stalk strength is through the condensation of the rind parenchyma cell layers and rind vascular bundle sheath cell layers, and thickening of the cell wall, along with lignification of such structures. The condensation of stalk structural components may be reflected in findings by Masole (1993), who showed increase of rind thickness and reduction of stalk diameter in response to selection for high RPR.

A proposal for developing chromosome arm-specific *Mutator* stocks for transposon tagging

--Kevin D. Simcox

The goal of this proposal is to develop a method of selecting for active *Mutator* (*Mu*) lines which contain *Mu* elements on a specific chromosome arm. The working hypothesis is that the development of selected chromosome arm stocks would enhance the frequency of recovery of *Mu* tagged alleles in a specific genomic region. The method for selecting chromosome arm-specific *Mutator* stocks will involve crossing *Mu* stocks by the series of B-A translocations. Translocation heterozygotes of each B-A stock will be crossed onto a diverse set of *Mu* lines to uncover recessive mutations present on a particular chromosome arm. In

turn, pollen from the *Mu* plants will be outcrossed onto a standard inbred line. The *Mu* x B-A crosses will be screened either in the field or the sandbench for recessive mutations. *Mu* x inbred crosses involving *Mu* plants found to carry recessive mutations on a particular chromosome arm will then be used in a targeted approach to tag known loci located on that chromosome arm. *Mu* plants selected using B-A translocations (directed selection) will be compared to randomly selected *Mu* plants (random selection) to determine if directed selection enhances the recovery of mutations at defined loci.

Ears harvested from the *Mu* x B-A testcrosses will initially be screened for recessive kernel traits and then planted in a sandbench to screen for seedling traits. If the *Mu* parent plant was heterozygous for a recessive mutation, pollination by a translocation heterozygote will uncover recessive phenotypes in frequencies less than 15%. The frequency of hypoploids recovered will depend upon the B-A translocation. Seedlings expressing the recessive phenotype should be hypoploid. Heterozygous mutations in the *Mu* parent plant can be recovered either in the *Mu* x inbred outcross, or by crossing pollen from the hypoploid onto B73 to recover diploid progeny, heterozygous for the mutation.

Novel mutations will appear as single events uncovered by the hypoploid chromosome. Recovery of interesting seedling mutations will depend, of course, on the pleiotropic effect of the mutation on seedling viability. Novel mutations which survive can be recovered from hypoploid plants by outcrossing to B73. RFLP or PCR analysis could be used to verify that novel mutations are hypoploid individuals, not due to *Mu*-induced deletions. Another type of mutation that will be encountered is a forward somatic mutation. Forward somatic mutations are typically manifested as colorless or yellow longitudinal stripes in the leaves. Since somatic mutations occur during cell division in the leaf meristem region, these mutations can not be recovered unless the sector includes the tassel. However, somatic sectors may indicate the presence of cis-linked *Mu* elements.

If selection of *Mu* lines using B-A translocations enhances the recovery of chromosome arm-specific mutations then the next step would be to develop "enhanced" *Mu* lines for each chromosome arm. Two different routes can be taken at this point: 1) *Mu* lines could be developed from a single selected *Mu* plant, or 2) a population could be developed by sib crosses between different selected *Mu* plants.

A limited set of *Mu* x B-A testcrosses were made in the summer 1994 nursery to get an idea of the types and frequency of phenotypes that might be recovered in the sandbench. A number of single mutation events were identified in the sandbench. These were mainly mutable albino or luteus phenotypes. Several cases of somatic sectors were found which could either represent a hypoploid or a forward somatic mutation of a heterozygote.

Clustering of disease resistance loci in the maize genome

--Michael D. McMullen and Kevin D. Simcox

Prior to the advent of RFLP mapping, there was a surprising lack of disease resistance genes that had been placed onto the maize genetic linkage map. A substantial number of resistance genes and QTLs to different plant pathogens and pests have been mapped in the last five years. When the various resistance genes and QTLs are placed onto the linkage map it is evident that resistance genes are clustered in the genome. These clusters of disease resistance loci include not only genes conferring resistance to a single pathogen or pest, but also genes for resistance to unrelated organisms. The bin locations of resistance genes and QTLs reported in the literature are shown in Table 1. There are six different regions that contain resistance genes or QTLs for more than one organism.

The region around bin 1.04, near the centromere on chromosome 1, contains loci for resistance to maize streak virus (*msv1*) and *Cochliobolus carbonum* race 1 (*hm1*). QTLs for northern corn leaf blight (NCLB), *Erwinia stewartii*, gray leaf spot (GLS), *Gibberella zeae*, and 2nd brood European corn borer (ECB) also map in or near this region.

The cluster on chromosome 3 includes loci for resistance to maize mosaic virus (*mv1*), WSMV (*wsm2*), and *Puccinia sorghi* (*rp3*). These genes map near *umc10* in the centromeric region of chromosome 3. QTL loci for resistance to *G. zeae* and ECB map to bins just distal to this region.

The short arm of chromosome 4 contains a single gene for resistance to *P. sorghi* (*rp4*) and QTLs for resistance to NCLB, GLS, *G. zeae*, and 1st brood ECB. This region is particularly

Table 1. Chromosomal bin location of disease resistance loci and QTLs affecting plant response to pathogens and pests. An asterisk (*) is used to designate a bin containing a QTL.[1]

	MDMV	SCLB	WSMV	MMV	*P.s.*	*P.p.*	*E.s.*
1S							1.04-1.06*
1L							
2S							
2L							
3			3.04	3.04	3.04		
4S					4.01*		
4L							
5							
6	6.01	6.01	6.01				
7							
8							
9S							
9L							
10S					10.01	10.01	
10L			10.05				

	GLS	*G.z.*	ECB	NCLB	MSV	*C.c.*
1S	1.04*		1.01*	1.02-1.05*	1.04	
1L		1.07*	1.07*			1.06
2S	2.04*		2.02*			
2L			2.08*	2.08		
3		3.06*	3.05*	3.08*		
4S	4.02*	4.01-				
4.04*		4.01*				
4L	4.07*					
5		5.04*		5.01*		
6						
7			7.04*	7.03*		
8	8.06*		8.04*	8.04*		
8.06						
9S			9.01*			
9L						9.04*
10S						
10L	10.05*	10.05*	10.04*			

[1]MDMV = Maize Dwarf Mosaic Virus (McMullen and Louie, Mol. Plant-Microbe Interact. 2:309, 1989); SCLB = Southern Corn Leaf Blight (Zaitlin et al., Genome 36:555, 1993); WSMV = Wheat Streak Mosaic Virus (McMullen et al., Mol. Plant-Microbe Interact. 7:708, 1994); MMV = Maize Mosaic Virus (Ming et al., MNL this issue); *P.s.* = *Puccina sorghi* (MNL 68:177); *P.p.* = *Puccina polysora* (MNL 68:177); *E. s.* = *Erwinia stewartii* (Ming et al., MNL this issue); MSV = Maize Streak Virus (Kyetere et al., MNL this issue); NCLB = Northern Corn Leaf Blight (Freymark et al., 87:537, 1993); GLS = Gray Leaf Spot (Bubeck et al., Crop Sci 33:838, 1993); *G.z.* = *Gibberella zeae* (Pe et al., MGG 241:11, 1993); ECB = European Corn Borer (Beavis et al.,Crop Sci 34:882, 1994; Schön et al., Heredity 70:648, 1993); *C.c.* = *Cochliobolus carbonum* race 1 (MNL 68:167).

interesting since *bx1* maps to this region. The *bx1* locus is involved in the accumulation of DIMBOA, a pre-formed anti-microbial compound. The presence of DIMBOA has been shown to be correlated with reduced NCLB lesion size (Couture et al., Physiol. Plant Path. 1:515, 1971; Freymark et al., Theor. Appl. Genet. 87:537, 1993). The NCLB, GLS, *G. zeae*, and ECB QTLs mapping to this region might be associated with regulation of DIMBOA accumulation.

On chromosome 6, three resistance genes map to bin 6.01; maize dwarf mosaic virus (*mdm1*), WSMV (*wsm1*), and resistance to southern corn leaf blight (*rhm1*). This region on chromosome 6 also contains the NOR, which has been shown to co-segregate with *mdm1* (Simcox et al., MNL 67:117, 1993; Simcox et al., Theor. Appl. Genet. 90:341-346, 1995).

A cluster on the long arm of chromosome 8 includes *Ht2* and *Htn1* (bin 8.06), and QTLs for resistance to NCLB and GLS. The QTL for NCLB located on the long arm of chromosome 3 (3.08) might be related to the NCLB QTL on chromosome 8. Chromosomes 3 and 8 share extensive duplicated sequences, especially in the region of bins 3.08 and 8.06 (Simcox and Bennetzen, Phytopathology 83:1326, 1993).

The other significant cluster is on the long arm of chromosome 10. Resistance to WSMV (*wsm3*) and QTLs for resistance to GLS, *G. zeae*, and ECB have been mapped near *umc44*.

It is interesting to speculate if there is a functional significance underlying clustering of disease resistance genes or if clustering is a consequence of genome organization. It is possible that these clusters represent gene families involved in signal transduction, or some other step in the cascade of events leading to an incompatible interaction between the host and the pathogen or pest. On the other hand, the clusters of disease resistance genes might represent ancestral arrangements of either disease resistance genes or genes which were recruited during the evolution of the pathosystem. It is interesting to note that the clusters present on chromosomes 1 and 3 are in the centromere region, and that the cluster on chromosome 6 is tightly linked to the NOR, which suppresses recombination. Over the evolution of maize, sequences mapping to regions of reduced recombination potential might be maintained as a linkage block. It will be interesting to determine if disease resistance genes map to syntenic regions of other grass species.

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Quantitative genetic analysis of loci controlling synthesis of maysin, a corn earworm resistance factor, in maize silks

--P.F. Byrne, M.D. McMullen, M.E. Snook, T. Musket, N.W. Widstrom, B.R. Wiseman

The antibiosis type of resistance to the corn earworm (CEW) *Helicoverpa zea* (Boddie) in maize is due primarily to the concentration of maysin and related flavone glycosides in the silk. Although there are a number of clues about the genetic control of CEW resistance and maysin synthesis (Byrne et al., MNL68:35, 1994; Widstrom and Snook, Plant Breeding 112:120-126, 1994; Wiseman and Bondari, J. Econ. Entomol. 85:293-298, 1992), understanding of the inheritance of maysin content is incomplete.

Because maysin synthesis occurs as a branch of the flavonoid metabolic pathway, our study sought to relate variation in maysin concentration to loci of that well-characterized pathway. One major section of the pathway, which controls the synthesis of C-glycosyl flavones (including maysin) and phlobaphenes (responsible for red cob and pericarp pigments), is regulated by the *p1* locus (Styles and Ceska, Maydica 34:227-237, 1989). That locus also affects the silk-browning trait, whereby silks of some genotypes turn brown after wounding and others do not change color (Levings and Stuber, Genetics 69:491-198, 1971). The other section of the pathway leads to flavonols and 3-hydroxy anthocyanins and is regulated by the coordinate action of either *r1* + *c1* or *b1* + *pl1* in a tissue-specific manner (Coe et al., In G.F. Sprague and J.W. Dudley, Corn and Corn improvement, Am. Soc. Agron., 1988; Dooner et al., Ann. Rev. Genet. 189:136-141, 1991). Although the two parts of the pathway require common intermediates up to the flavanone branch point, each portion appears to be independently regulated, at least in pericarp tissue (Grotewold et al., Cell 76: 543-553, 1994; Styles and Ceska, Maydica 34:227-237, 1989).

Our study sought to identify and estimate the contribution of loci that affect maysin synthesis in the population (GT114 x GT119)F_2, derived from a cross of a high- by a low-maysin parent.

Maysin concentrations in silks of 285 F_2 plants were determined by reversed-phase HPLC (Snook et al., J. Chromatogr. 477:439-447, 1989) at the USDA-ARS Phytochemical Research Unit, Athens, Georgia. RFLP genotypes at loci encoding flavonoid pathway enzymes or linked marker loci were determined for the same plants. Single-factor analysis of variance was first used to detect significant associations between maysin concentration and genotypic classes at individual RFLP loci, based on a comparison-wise error rate of 0.05. Then, significant loci from the individual locus analyses and significant digenic epistatic interaction terms were included in multiple-locus models. The "best" model was determined to be that which explained the greatest proportion of the phenotypic variance (i.e., had the highest R^2 value) and in which all terms were significant at the 0.05 significance level.

The *p1* locus had by far the largest effect on maysin concentration, accounting for 58% of the total phenotypic variance (Table 1). Significant loci unlinked to *p1* included *umc207* and *umc113* (both closely linked to *c1* and *bz1*), *r1*, and *a1*, which was marginally significant. We are uncertain which of the genes *c1* or *bz1* influenced maysin concentration. We plan to differentiate between these loci by testcrossing the parental lines to *c1* and *bz1* tester stocks and observing aleurone color development. We are not able to test the *c1* locus directly because the parental lines were not polymorphic for the *c1* probe with the eight enzymes tested.

The "best" multiple-locus model accounted for 71.1% of the phenotypic variance for maysin concentration. In addition to *p1*, the model included the *c1* - *bz1* region and epistatic interactions of *p1* with *c1* - *bz1*, *umc166b* , and *r1*. *umc166b* was scored as a second locus which appeared in the analysis of *umc166a*, a marker flanking *a2* on chromosome 5. Our data place *umc166b* on chromosome 1 about 40 cM from *p1* toward the distal end of the long arm, but we are uncertain what function that locus or a linked locus might have.

Table 1. Results of single-factor analysis of variance examining the association of maysin concentration with genotype classes at 16 flavonoid pathway loci or closely linked markers.

Marker (locus)	Linkage group	Significance †	$R^{2§}$	N	Source of higher allele
Significant loci:					
umc185 (*p1*)	1	<0.001	0.580	284	GT114
npi286 (near *p1*)	1	<0.001	0.574	276	GT114
umc166b‡	1	<0.001	0.148	283	GT114
umc207 (near *c1*, *bz1*)	9	<0.001	0.067	278	GT119
umc113 (near *c1*, *bz1*)	9	<0.001	0.063	284	GT119
umc182 (*r1*)	10	0.005	0.038	275	GT114
umc44 (near *r1*)	10	0.002	0.044	282	GT114
umc189 (*a1*)	3	0.025	0.026	278	GT114
umc63 (near *a1*)	3	0.039	0.023	285	GT114
Non-significant loci:					
umc181 (*bz2*)	1	0.402	0.007	282	
csu164 (near *bz2*)	1	0.337	0.008	285	
chi (*chi1*)	1	0.134	0.014	285	
umc84 (near *chi1*)	1	0.207	0.011	281	
umc198 (*whp1*)	2	0.552	0.007	175	
csu64 (near *whp1*)	2	0.419	0.006	273	
umc6 (near *b1*)	2	0.200	0.011	285	
umc34 (near *b1*)	2	0.146	0.014	283	
umc4 (near *chi2*)	2	0.604	0.004	284	
npi239 (near *F3H*¶)	2	0.800	0.002	284	
umc198 (*c2*)	4	0.211	0.017	185	
npi270 (near *c2*)	4	0.522	0.005	284	
umc166a (near *a2*)	5	0.251	0.010	284	
chi (*chi3*)	5	0.649	0.005	173	
npi409 (near *chi3*)	5	0.162	0.013	273	
umc21 (near *sm1*)	6	0.568	0.004	278	
csu13 (near *in1*)	7	0.742	0.002	284	
c1	9	non-polymorphic			

† *F*-test *P*-value.

§ Portion of the phenotypic variance accounted for by genotypic classes at a locus.

‡ Approximately 40 cM to the right of *p1*.

¶ Locus encoding flavanone-3-hydroxylase (L. Taylor, personal communication).

Mean maysin concentrations for genotypic classes indicate a variety of types of gene action (data not shown). The effect of *p1* is strikingly additive, while the GT114 allele at *umc207* appears dominant for low maysin concentration. In their interactions with *p1*, both *umc166b* and *r1* affect maysin concentration only when *p1* is homozygous dominant for the high-maysin allele; however, they differ in that the effect of *umc166b* is significant in the heterozygote, and the effect of *r1* is in the GT114 homozygous class.

The importance of *p1* in our analyses was predicted, as the concentration of maysin and its analogs is known to be associated with the *p1*-controlled silk-browning trait (Byrne et al., MNL68:35, 1994), and the parents used in our study have contrasting silk-browning phenotypes. The two findings of this study we find most interesting are:

(1) The involvement of loci from the *r1/c1* controlled portion of the pathway in the expression of a trait predominantly controlled by *p1*. Although the contribution of *r1* to maysin concentration was small, that of the *c1* - *bz1* region was highly significant, accounting for an additional 9% of the phenotypic variation (as an individual locus and in interaction with *p1*) after *p1* was already in the model. This contrasts with indications of independent regulation of the two parts of the pathway in pericarp tissue (Grotewold et al., Cell 76: 543-553, 1994; Styles and Ceska, Maydica 34:227-237, 1989). However, in coleoptile tissue, Styles and Ceska (Can. J. Genet. Cytol. 23:691-704, 1981) reported that *p1* affected concentrations of some 3-glucosylated flavonols (in the *r1/c1* portion), and that alleles at *bz1* (also in the *r1/c1* portion) affected flavone concentrations, although the significance of that difference was not clear.

(2) The importance of regulatory loci (*p1*, *r1*, possibly *c1*), as opposed to structural loci, in the expression of these chemical concentration traits. The only known structural loci having significant associations were *a1* (which was marginally significant in the maysin single-factor analysis and eliminated in the multiple-locus model) and possibly *bz1*. One explanation for this result is that the parental lines may not have segregated for functionally distinct alleles at the structural loci evaluated, while they did segregate at *p1* and *r1* (based on our observations of cob and brace root color). Another, more speculative explanation for our results is that changes at regulatory loci are inherently more important than changes at structural loci in determining end-product concentration. Biochemical flux-enzyme theory offers support for this explanation; in complex pathways, substantial reductions in activity of any one structural enzyme are likely to have only small effects on flux through the system (Kacser and Burns, Genetics 97: 639-666, 1980).

Will regulatory loci be equally important in other populations and for other quantitative traits? We plan to test that hypothesis for maysin concentration and for CEW larval growth response by analyzing several additional populations, including one segregating for suspected functional differences at a structural locus (*c2*), and another segregating for an apparent dominant inhibitor allele at a regulatory locus (*c1*).

DURHAM, NORTH CAROLINA

Morphological differences in pollen grains of *Zea diploperennis*, *Tripsacum dactyloides*, *Tripsacum-diploperennis* hybrids and maize

--M. Eubanks

The exine, i.e., outer layer, of the wall of a pollen grain in *Zea mays* L. and its wild relatives, teosinte and *Tripsacum*, has distinctive sculpturing of simple raised projections called spinules. Scanning electron microscopic characterization of spinule patterns and density has been used in palynological studies to identify modern and fossil maize pollen (Tsukada and Rowley 1964; Irwin and Barghoorn 1965; Banerjee and Barghoorn 1972; Grant 1972). In *Zea*, the spinules are more or less uniformly distributed over the surface of the pollen grain, whereas in *Tripsacum*, spinules are consistently clumped together and this forms a distinctive reticulate pattern.

Scanning electron microscopy was employed for investigation of pollen spinule patterns and density among *Tripsacum dactyloides* L., *Zea diploperennis* Iltis, Doebley and Guzman, Tripsacorn (*T. dactyloides* X *Z. diploperennis*), Sun Dance (*Z. diploperennis* X *T. dactyloides*), *Zea mays*, and an F2 maize *diploperennis* hybrid. Dry pollen grains were mounted, coated with gold-palladium alloy for conductivity, then photographed with high resolution SEM. Spinules were more or less evenly distributed in diploperennis, maize, and all of the hybrid plants, with the exception of Tripsacorn, which shows some clumping of spinules but not the degree of distinctive reticulation that occurs in *Tripsacum*. Spinule density was determined by counting the number of spinules in an area of 100 m^2. See Table 1 for results.

Findings show Tripsacorn spinule density of 6.4/m^2 is greater than the diploperennis male parent and less than the *Tripsacum*

Table 1. Spinule density in *Zea*, *Tripsacum* and *Tripsacum-Zea* hybrids.

Type	No. spinules/100 μ^2	No. spinules/ μ^2
Tripsacum dactyloides	1,144	11.2
Zea diploperennis	466	4.6
Tripsacorn	650	6.4
Sun Dance	409	4.0
Zea mays	584	5.7
Maize X *diploperennis*	541	5.3

female parent as expected; thus spinule density is intermediate between both parents. Spinule density in Sun Dance is 4.0/m^2. It is lower than the diploperennis female parent; significantly lower than the Tripsacum male parent, and lower than Tripsacorn, the reciprocal hybrid between the same two parents. Pollen spinule density is evidently a characteristic that is distinctive between the different hybrids Tripsacorn and Sun Dance. Maize spinule density is 5.7/m^2. The maize-diploperennis hybrid at 5.3/m^2 is intermediate between both its parents. Additional SEM study is being carried out to investigate potential significance of these findings for pollen identification.

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Mapping results: nuclear mutations affecting chloroplast biogenesis

--Alice Barkan and Laura Roy

***psb1* maps to chromosome 6L.** *psb1* is a *Mu*-induced mutation that causes the specific loss of photosystem II from the thylakoid membrane. A tentative map position on 8L was reported previously and is now known to be incorrect. This past summer we unambiguously mapped *psb1* to 6L. Crosses of six *psb1*/+ plants by TB-6Lc resulted in progeny segregating pale green seedlings lacking photosystem II (specifically). In this summer's crop, crosses with other B-A stocks (including those that unmask 8L) did not unmask any mutant phenotypes.

***psb2* maps to chromosome 5L.** *psb2* is a *Mu*-induced mutation that causes the specific loss of photosystem II from the thylakoid membrane. Crosses of two *psb2*/+ plants by TB-5La resulted in the segregation of pale green seedlings lacking photosystem II. Crosses of +/+ plants by the same B-A plants did not result in mutant progeny, nor did crosses of *psb2*/+ by other B-A stocks. Therefore, *psb2* maps to the long arm of chromosome 5.

That *psb1* and *psb2* are not allelic was confirmed by the results of allelism tests, in which the two mutations complemented one another. *psb1* and *psb2* do not map to the same chromosome arms as two previously mapped nuclear mutations that also cause the specific loss of photosystem II (*hcf3* and *hcf19*). Therefore, there are at least 4 nuclear genes required specifically for the biogenesis of photosystem II.

***crp2* maps to chromosome 5S.** *crp2* (chloroplast RNA processing 2) is a *Mu*-induced mutation that causes defects in the metabolism of the chloroplast *psaI*, *petA*, and *psbB* mRNAs (unpublished results). Progeny of 14 *crp2*/+ plants crossed by TB-5Sc segregated small, yellow green plants with hypoploid morphology, and exhibiting the protein and RNA defects typical of *crp2*. Crosses of +/+ plants with the same B-A plants did not result in mutant progeny, nor did crosses of *crp2*/+ with other B-A translocation stocks. Therefore, the *crp2* locus maps to the short arm of chromosome 5.

FORT COLLINS, COLORADO
Colorado State University

Localization of the Pex1 protein to the pollen tube wall

-- Amy Rubinstein, Anne Broadwater and Patricia Bedinger

Very little is known about the genes required for pollination in self-compatible species, such as maize, or about the mechanism of the rapid growth of the maize pollen tube. As a first step toward identifying some of these genes, our lab isolated several genes expressed specifically in pollen. Two of the clones isolated contained a very distinctive repeat motif: ser-pro4. Prolines found in this motif are usually hydroxylated. Hydroxyproline is an unusual amino acid that occurs in only a few groups of extracellular matrix proteins, including collagen and elastin in animals and hydroxyproline rich glycoproteins (HRGPs) in plants. One major class of HRGPs, the extensins, are characterized by the ser-pro4 motif that we found in our clones. Thus, we named the genes *Pex1* and *Pex2* (pollen, extensin-like).

Finding genes like these in pollen was an exciting discovery for a number of reasons. First, although extensin-like genes have been found to be specifically expressed in the pistil, none had been found in pollen. These are the first pollen-specific genes to be found with the canonical extensin repeat motif. Secondly, the discovery of these genes also marked the first time a classical extensin repeat motif had been found in any maize gene.

We isolated and sequenced a complete genomic clone representing *Pex1*. We discovered that the gene actually contained two distinct domains: one contained the ser-pro4 repeats and was presumed to have a rod-like structure. The other domain, at the N terminus, contained no repeated motifs. This structure was reminiscent of the sexual agglutinins of *Chlamydomonas*, which mediate the recognition between the plus and minus mating types of *Chlamydomonas*. We would now like to investigate the possibility that the pollen Pex proteins interact with a partner molecule in silk in a manner analogous to mating in *Chlamydomonas*.

Southern analysis confirmed that there are two copies of this gene in the maize genome. From Northern analysis, we determined that the *Pex* genes are expressed specifically in pollen. In addition, we found that the *Pex* genes are not expressed in early stages of pollen development and are present at a low level in starch-filled immature pollen. Only upon pollen maturation were high levels of *Pex* messages detected. This work, including the cloning and characterization of the *Pex* genes, has been accepted for publication in the Proceedings of the National Academy of Sciences, USA.

To more directly study the protein encoded by *Pex1*, and to provide clues about its function, a fusion protein was constructed using a piece of the *Pex* gene and the *trpE* gene of *E. coli*. Antibodies raised against this fusion protein recognized two proteins in pollen extracts just over 200 kD in size, as well as larger proteins in the stacking gel. In agreement with Northern analysis, these proteins were not present in seedling, endosperm, ear, silk, root, leaf or wounded leaf extracts. We also found no Pex protein in meiotic tassel or young microspore extracts. In starch-filled immature pollen, the Pex antibodies recognized the >200 kD proteins, but did not recognize any proteins in the stacking gel. Because deglycosylation with trifluoromethanesulfonic acid (TFMS) removed the proteins in the stacking gel, we are hypothesizing that they represent the glycosylated form of the

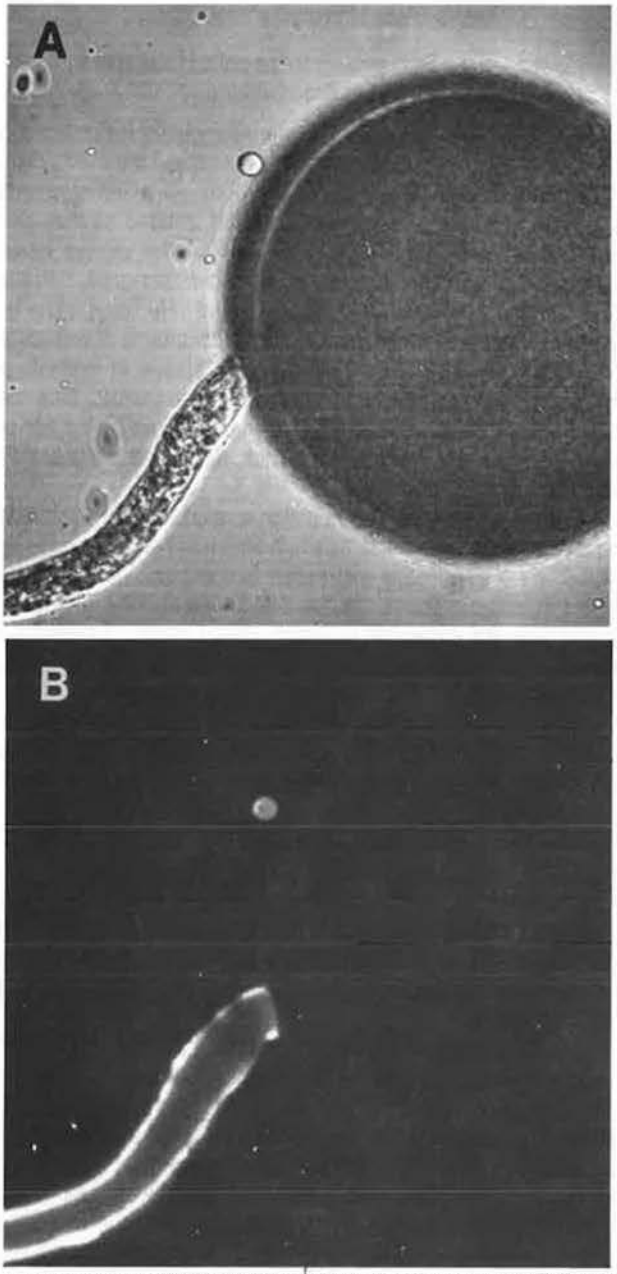

Figure 1. Localization of Pex1 to the pollen tube wall with the Pex1 antibody and a fluorescent secondary antibody using a confocal light microscope. A. transmitted image B. fluorescent image.

Pex proteins. Our results suggest that the glycosylation of the Pex proteins occurs during very late stages of pollen development.

We have also used the Pex antiserum in immunolocalization experiments with *in vitro* germinated pollen. Utilizing a FITC-labeled secondary antibody and with the aid of a confocal microscope, we were able to determine that the Pex proteins are localized to the pollen tube wall. This result is consistent with a function for the Pex proteins during the process of pollination.

In addition, we carried out several experiments to determine the nature of the association of the Pex proteins with the pollen tube wall. The extensins, for example, are known to be covalently crosslinked to the walls of vegetative cells. To test for such tight association, we treated pollen tubes with 1% SDS, 1M NaCl and 8M LiCl. None of these agents eliminated the Pex signal. These results indicate that the Pex protein is tightly bound (possibly covalently) to the pollen tube wall. This is consistent with the properties of known extensins, which cannot be extracted from cell walls with SDS or high salt. Treatment of the pollen tubes with Proteinase K caused the Pex signal to disappear almost entirely. This indicates that the substance being recognized by the antibody is indeed a protein.

We envision three possible functions for the protein product of the *Pex* genes. It could be a structural component of either the pollen or pollen tube walls, necessary for providing structural support during the extremely rapid growth of the pollen tube. It may be involved in the initial recognition between pollen and silk at the point of pollen tube germination, or it may mediate cell-cell signaling during pollen tube growth. It is also possible that the Pex proteins are involved in all of these processes.

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New *shrunken2* mutants derived from an *Ac/Ds* stock with totally unexpected properties

--J.R. Shaw, D. R. McCarty and L. C. Hannah

In an attempt to use *Ds* as a highly efficient mutagen of the *shrunken2* locus, we have uncovered an interesting and unexpected set of mutants. In brief, an analysis of 13 newly–derived mutants reveals that all are homozygous lethal, all are transmitted through the male parent with 50% expected frequency, and those monitored are associated with loss of function of the closely linked *A1* gene. However, none is a deletion for the *Sh2* locus. A brief summary of the results is given below.

Initially 44 revertants from the *Ds*–containing allele *sh2–m1* were collected. This mutant is described in Giroux et al., Proc. Natl. Acad. Sci. 91:12150, 1994. Revertants were made homozygous and approximately 1/2 of these were placed in a detasselling block and pollinated by a *sh2* mutant, *sh2–R* in the sweet corn 'Florida Stay Sweet'. This mutant contains a very large insertion in the 5' end of the gene (Shaw and Hannah, unpublished). Approximately 150,000 kernels from each revertant were monitored for the occurrence of new *sh2* mutants. One and only one revertant, *Rev25,* was found to produce new *sh2* mutants at very high frequencies (several per thousand seed). While the vast majority of the resulting mutant seed did not germinate, we did find about 25 new, heritable mutants. Most of these were analyzed as described below.

Plants derived from mutant seed of the detasselling block were self–pollinated and crossed onto wild-type plants. Seeds of the cross were planted, numbered, selfed and crossed onto 'Florida Stay Sweet'. Leaf samples were also taken and Southern analysis was used to identify plants containing the mutant *sh2–R* allele. The large 5' insertion in this mutant leads to diagnostic restriction fragments. A more complicated crossing scheme was used in those cases in which the initial outcross to wild-type was not performed.

Unexpectedly, selfed seed from the heterozygotes above did not include typical *sh2* seed. Rather, these ears contained paper–thin seeds composed almost entirely of pericarp. In cases of excellent seed set, these aborted seeds were seen only after shelling. That these plants were *sh2* heterozygotes however,

was seen in the test cross. Typical *shrunken2* seed were found in the cross to 'Stay Sweet', albeit at frequencies of approximately 25%. Of the approximately 25 new mutants initially isolated, we failed to identify any that behaved as typical *sh2* mutants (the standard mutant phenotype and normal transmission through the male). In those families not exhibiting the lethal seed phenotype, Southern analysis showed that all plants contained the *sh2–R* allele. Additional plants from these families will be analyzed in the future; however, we cannot exclude the possibility that the new mutant in each of these families totally lacks male transmission.

Homozygous lethality and reduced male transmission are hallmarks of mutants of this region of chromosome three that are thought to be deletions. Three X–ray induced mutants, *a-x1*, *a-x2* and *a-x3* are known. These lack *A1* and *Sh2* function and are associated with either reduced or no male transmission. These mutants are also homozygous lethal and presumably lack a gene necessary for normal seed development. These mutants have been interpreted as deletions for that portion of chromosome 3 that contains at least these three genes.

In an attempt to extend the analysis of the new mutants and their apparent similarity with the *a-x* series of mutants, we monitored a number of the newly created, homozygous lethal *sh2* mutants for *A1* function. The revertant, *Rev25*, from which most but not all of the newly–created *sh2* mutants were derived, contains a functional *A1* allele. However, crossing of a number of the new *sh2* mutants derived from *Rev25* to an *a1* tester showed that these mutants lacked *A1* function. We conclude that the mutation that gave rise to the loss of *Sh2* function also abolished *A1* function as well as the gene needed for seed development. In this regard, these mutants are identical to the *a-x* series of mutants.

Surprisingly, while these mutants appear from more classical types of analyses to be deletions, *Sh2* sequences are still present in these mutants. Because the new mutants are homozygous lethal, paired samples (wild-type/new mutant and *sh2–R*/new mutant) were analyzed on Southern blots. The large 5' insertion in *sh2–R* gave rise to unique 5' fragments, and RFLPs 3' to the gene were used to monitor variation in that region. Of the 13 new mutants analyzed, all contained 5' *Sh2* fragments. Clearly, these are not deletions for this gene.

Probing of the 3' portion of the gene proved less straightforward. A unique fragment was found in 12 of the 13 mutants. This fragment was of a different mobility than that found in *sh2–R*, *Rev25* or in any of the wild-type alleles analyzed. While differing from these other alleles, the 3' fragment was indistinguishable in mobility from all 12 mutants. Eleven of the 12 mutants arose from *Rev25* while the 12th mutant was derived from another revertant, *Rev31*. Two other features are interesting about the 3' portion of *Sh2* in the new mutants. First, the fragment is always of less intensity than that of the corresponding fragment found in the heterozygote. This is not the case with the 5' fragment. Secondly, the 3' fragment was present in the heterozygote of the new mutant *sh2–M15* with *sh2–R* but was absent in the paired wild-type/*sh2–M15* sample. Thus it appears that this fragment is genetically unstable. The results from the paired samples involving *sh2–M15* may suggest that the fragment is meiotically separable from the *sh2* locus. However, since the DNA used in those blots revealing lesser intensity of this band was always derived from single plants, it would appear that this DNA maybe mitotically unstable.

The data to date show that while the 5' portion of *Sh2* is always present in these mutants and is of the intensity expected, in comparison to the paired allele, this is not the case with the 3' portion of the gene. This fragment is always of less–than–expected intensity and is missing in some plants. The point delimiting these regions has not been identified although it must lie within or close to the 3' portion of intron 13 of the gene. Intron 13 is the largest within *Sh2* (1.8 kbp). Surprisingly, probing of Southerns with this intron and selected subclones derived from it reveals that these sequences are very highly repetitive in the maize genome. Whether this fact is related to the mutant formation is currently unknown.

The transposable element *Ds* may not be involved in the creation of these new mutants. We also analyzed a new mutant derived from a wild-type allele of uncertain origin. The properties of this new mutant -- homozygous lethality, reduced male transmission, reduced intensity of the 3' fragment -- are indistinguishable from those of the mutants described above. We (Clancy, unpublished) sequenced selected portions of the wild-type allele and can detect no evidence that this allele is related to the progenitor of *sh2–m1*. Therefore, we have no evidence that *Ds* is involved in the creation of these new *sh2* mutants.

These data are relevant for a number of reasons. First they call into question the reliability of using reduced male transmission and the loss of function of more than one gene as definitive criteria for deletions. In our case, we repeatedly isolated *sh2* mutants that behaved like deletions by these two criteria, yet the *shrunken2* gene sequences are still present. We are currently examining *a-x1* and *a-x2* at the molecular level to determine if any *Sh2* sequences are present in these stocks. The data also point out a problem of using a closely linked transposable element to efficiently isolate new mutants of a particular gene. We screened approximately 25 revertants of a *Ds*–induced mutation and obtained no evidence that any could produce new mutants via *Ds* reinsertion.

The mechanism giving rise to these new mutants is clearly not presently understood. One possibility that explains all of the unexpected observations is the following: sequences within intron 13 of *Sh2* pair with homologous sequences that lie distal to the *A1* locus and the gene required for normal seed development. Recombination then occurs, giving rise to a linear chromosome and a ring containing all sequences lying between these two homologous sequences. Following recombination, the 5' portion of *Sh2* would remain on chromosome 3 whereas the 3' portion as well as *A1* and the seed development gene would be found on the ring. This model is in agreement with the orientation of *A1* and the transcriptional unit of *Sh2*, as recently demonstrated by Schnable and colleagues, explains the inferred somatic instability (instability of ring chromosomes) of the 3' but not the 5' portion of *Sh2*, exploits the repetitiveness of the sequences found within intron 13 of *Sh2*, and explains the fact that this mutation is recurrent and independent of the transposable element *Ds*. It is currently under test.

The phenotype exhibited by these new *sh2* mutants, paper–thin seed composed almost entirely of pericarp, is common in maize genetics. With this letter, we ask for wild-type seed from selfed ears segregating for this phenotype. In return, we will determine whether these lack *Sh2* function and, in doing so, gain some insight into the frequency of this event in maize.

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Correction of allelism results for *am1* and *am2*
--William F. Sheridan and Inna N. Golubovskaya

We have been studying the genetic regulation of the initiation of meiosis and the early events of prophase I. The *ameiotic1* (*am1*) gene appears to function at a minimum from some point during premeiotic interphase until the early zygotene phase of meiosis (I. Golubovskaya et al., Genetics 135:1151-1166). In addition to the reference mutant *am1* allele (M. Rhoades MNL 30:38-42, 1956), we are studying the *am1-pra1* allele isolated following N-nitroso-N-methylurea treatment of seed (I.N. Golubovskaya et al., Genetika 28(3):137-146, 1992) and also two new alleles, *am1-485* and *am1-489*, isolated from *Mutator* stocks during our screen for new transposon-tagged meiotic mutations.

A new meiotic mutation termed *ameiotic2* (*am2*) was reported (C. Curtis and G. Doyle, J. Heredity 82:158-163, 1991) and seed samples were kindly provided to us by Greg Doyle. We conducted two sets of allelism tests between *am1* alleles and the *am2* mutation. In both sets of tests the *am2* mutation failed to complement *am1* as evidenced by male sterile plants. Cytological examination of microsporocytes from these plants revealed a typical ameiotic phenotype. The *am2* mutation is therefore a new allele of the *am1* locus and we now refer to it by the designation *am1-2*.

Location of *mac1* on chromosome arm 10S
--William F. Sheridan and Inna N. Golubovskaya

We have isolated a new recessive mutation that appears to control the switch of the ovule hypodermal cells from the vegetative to the sporogenous developmental pathway. Whereas in normal ovules there is a single archesporial cell, in mutant ovules there are usually several archesporial cells that undergo a normal meiosis. Homozygous mutant plants are partially female fertile but are completely male sterile. This mutation had a laboratory designation of *lar*-487* but we now designate it with the gene symbol *multiple archesporial cells1* (*mac1*). Crosses of *mac1/mac1* plants as female by pollen from TB-10Sc yielded progeny that segregated 26 fertile plants and 15 male sterile plants. This locus is therefore on chromosome arm 10S.

HOMESTEAD, FLORIDA
Holden Farms
RALEIGH, NORTH CAROLINA
North Carolina State University

Death of Karalee Holden, a maize genetics pioneer in winter nursery work in Florida
--Anne S. Erickson and Major M. Goodman

Karalee Holden was the field manager responsible for planting, pollinating, and harvesting much of the Florida winter nursery crop for university and USDA geneticists and plant breeders from 1954-1982, while working for the Ag Alumni Seed Improvement Association (affiliated with Purdue University). For many geneticists and plant breeders, Karalee was the person most responsible for the success of their Florida winter nurseries.

Karalee died on June 4, 1993, at age 61, after a second round with ovarian cancer. She is survived by her parents, sisters, and by her partner in Holden Farms (telephone 305-247-6876), Anne Erickson, who is carrying on the family avocado services business and the wholesale hibiscus nursery she and Karalee started after leaving Ag Alumni in 1983.

HONOLULU, HAWAII
University of Hawaii

Near isogenic lines of tropical inbred Hi27
--James L. Brewbaker

A series of marker genes, largely derived directly from Maize Genetics Coop, were backcrossed over the past three decades into inbred Hi27. An initial report of this program was presented in MGCN 46:33-37, 1972. The inbred Hi27 was selfed 7 times in Hawaii from CM104 (India), an inbred released in 1964 after 13 sib and 1 self generations in India from the Colombian flint, Amarillo Theobromina (unpublished 1964 report, Cooperative Maize Improvement Scheme, Rockefeller Foundation). It has wide tropical adaptability and pest resistance, and enters several important tropical hybrids (Kim et al, Proc. Corn and Sorghum Res. Conf. 29:118-133, 1974). Our studies suggest Hi27 to be homozygous for *A1 A2 b Bz C-I Pl pr r Y p-ww* (some NILs are *p-wr*); also for *Ht1*, *Mv1* and *Rp1-d* backcrossed into it in Hawaii. Several linkages with QTLs are apparent in these NILs, as the parents are very different; i.e., Corn Belt dent vs. a tropical flint that is high-eared, daylength-sensitive, and has high general resistance to many diseases and pests of maize (Brewbaker, Logrono and Kim, Hawaii Inst. Tropical Agric. Res. Ser. 62, 1989).

Gene	Location	# BCs	Phenotype
a1	3L-149.0	4	colorless aleurone, green or brown plant
ad1	1L-108	7	adherent, tassel branches adhere
ae1	5L-57	1	amylose extender, high amylose endosperm
b1	2S-49	8	colored plant; *B* (on *A Pl*) boosts stalk color
ba1	3L-102	3	barren stalk
bd1	7L-109	5	branched silkless; ear is silkless, branched
bf1	9L-137	5	blue fluorescent
bif1	8-0	2	barren inflorescence (*Bif1*); few spikelets
bk1	9L-82	6	brittle-stalk; brittle leaves, tassels, etc.
bm1	5S-41	5	brown midrib
bm2	1L-161	5	*brown midrib2*
bm3	4S-(55)	5	*brown midrib3*; low stalk lignin
bm4	9L-141	1	similar to *bm1*
br1	1L-81	5	brachytic; not GA-responsive
br2	1L-(64)	6	*brachytic2*; 35% reduction
br3	5-(-)	1	like *br1*
bt1	5L-42	6	brittle endosperm (carries *a2*)
bt2	4S-67	6	brittle endosperm; encodes ADPG pyroPP
bz1	9S-31	5	bronze aleurone (modifies purple)
c1	9S-26	5	colored aleurone
cg1	3S-35	7	corn grass; *Cg1* plants grassy
ch1	2L-155	8	chocolate pericarp; *Ch1* plants tan pericarp
cr1	3S-26	6	crinkly leaf; plant dwarfed
ct1	8-(22)	1	compact plant; ear forked
d1	3S-44	3	dwarf; broad leaves; GA responsive
Dt1	9S-(-)	4	dotted aleurone; regulates *A1* locus, is *a1/a1*
du1	10L-31	5	dull endosperm; branching enzyme
el1	8L-(-)	6	elongate (collapsed seed, unreduced gametes)
et1	3L-161	5	etched endosperm; virescent (lethal) seedling
f1	1L-86	4	fine stripe; virescent seedling
fl1	2S-68	7	floury endosperm
fl2	4S-39	8	*floury2* endosperm; high lysine
g1	10L-50	7	golden plant; distinctive gold stalk
gl1	7L-36	4	glossy; cuticle wax altered, water adheres
gl2	2S-30	4	*glossy2*, like *gl1*
gs1	1L-135	1	green stripe, complementary to *gs2*
gs2	2S-54	1	green stripe, complementary to *gs1*
gt1	1S-(55)	4	grassy tiller; long flag leaves
h1	3S-(49)	3	horny (soft) starch
ij1	7L-52	4	iojap stripe; chloroplast defects, maternal
in1	7S-20	4	intensifies color (aleurone) on *A C R Pr*
j1	8L-80	7	japonica stripe

kn1	1L-133	3	knotted; *Kn1`* with proliferations
la1	4S-36	6	lazy; prostrate plant
lc1	10L-68	7	red leaf (*Lc1* is presumed; origin uncertain)
lg1	2S-11	6	liguleless; ligule and auricle missing
lg2	3L-101	4	*liguleless2*; less extreme than *lg1*
lg3	3-65	7	*liguleless3*; *Lg3* liguleless, leaves concave
li1	10L-(33)	1	lineate; fine, white stripes on basal leaves
lw1	1L-(134)	5	lemon-white; white seedling, pale yellow seed
mn1	2S-(68)	6	miniature seed
ms8	8L-66	6	*male sterile8*; anthers not exserted
ms10	10L-(45)	5	*male sterile10;* anthers not exserted
mv1	3-(80)	6	maize mosaic virus; *Mv* resistant
na1	3L-113	6	nana; dwarfed c. 90%, not GA responsive
na2	5S-(17)	6	*nana2*; like na1
o1	4L-98	6	opaque endosperm
o2	7S-16	6	*opaque2*endosperm; high lysine
og1	10S-19	9	old gold stripe; *Og1* striped gold/white
oy1	10S-15	6	oil yellow; seedling yellow, stunted
p-rr	1S-26	7	*p1* locus; pericarp color; red cob, seed
p-rw	1S-26	7	*p1* locus; red cob, colorless pericarp
p-vv	1S-26	10	*p1* locus; variegated pericarp
pg11	6L-38	1	*pale green11*; duplicate locus of *pg12*
pg12	9-61	1	see *pg11*
pl1	6L-49	3	purple plant; *Pl* purple (on *A B*)
pm1	3L-(73)	6	pale midrib; plant reduced in vigor
pn1	7L-112	7	papyrescent glumes; *Prl*ong papery glumes
px3	7L-(112)	2	*peroxidase3*; anodal, many plant tissues
py1	6L-69	6	pigmy; dwarfed, leaves thin, streaked
r-g	10L-64	3	colorless aleurone & plant on *B Pl*
R	10L-64	4	(*R* allele) colored aleurone & plant
R-nj	10L-64	6	*R-nj*, navajo (blotched) red aleurone
ra2	3S-49	5	*ramosa2*; tassel branched, ear irregular
rf1	3S-(62)	8	restorer; *Rf* restores T cytoplasm
rf4	8-(0)	10	restorer; *Rf4* restores C cytoplasm
rg1	3L-67	10	ragged leaf; *Rg1* with torn leaves, dwarfed
rp1	10S-3	5	race-specific resistance (*Rp1-d*) to *P.sorghi*
rt1	3S-(60)	2	rootless; secondary roots few or absent
sh1	9S-29	5	shrunken endosperm; encodes sucrose syn.
sh2	3L-149.	6	*shrunken2* endosperm; encodes ADPG PP
sk1	2S-56	4	silkless; pistils abort
sl1	7L-50	6	slashed; leaves split lengthwise
sr1	1S-0	5	striate leaf; white streaks, semi-dwarfed
su1	4S-66	6	sugary; debranching enzyme (two origins)
su2	6L-58	2	*sugary2* endosperm; starchy, glassy
tp1	7L-46	7	teopod; *Tp* ith simple tassel, podded ears
ts2	1S-24	5	*tassel seed2*; tassel silky, ear compact
ts4	3L-73	5	*tassel seed4*; tassel compact, silky
ts5	4S-53	6	*tassel seed5*; *Ts5* tassels with some silks
ts6	1L-158	2	*tassel seed6*; *Ts6* tassels silky, compact
tu1	4L-101	10	tunicate; *Tu1* kernels in long glumes
v2	5L-107	1	virescent; yellow-white, greens slowly
v4	2L-83	5	*virescent4*; greens slowly (carries fl)
v5	7S-24	6	*virescent5*; older leaves with white stripes
v16	8L-52	6	*virescent16*; like *v2*; segr *ms*(8?)
vg1	1L-85	1	vestigial glumes; *Vg* glumes short, no pollen
vp5	1S-1	3	viviparous; white endosperm & seedling
w3	2L-111	1	white endosperm and seedling, some vivipary
ws3	2S-0	5	white sheath; whitened husks, culm
wx1	9S-56	6	waxy endosperm; also pollen with amylopectin
y1	6L-17	6	pale yellow endosperm; often white
yg2	9S-7	4	*yellow green2*; seedling, plant yellow, dwarf
zn1	10L-29	5	zebra necrotic; lvs. banded, necrotic tissue

Translocations and Cytoplasms:

T4-6b	10	
T4-10f	10	
T9-10a	9	
cms-C	female-transmitted, restored by *Rf4* (and others)	
cms-T	female transmitted, restored by *Rf1 Rf2*	

New Mutants under Study:

blo	5	blotched leaves, pale stripes; from Hawaiian Sugar
bst	3	brown stripe; one of *ys* (yellow stripe) types?
lll	4	leaf-fleck; dominant; like *les1*, larger lesions (segr. opaque and pale yellow)
si1	4	Coop's "*ms-si*" male-sterile; silky ears
sky	3	skinny plant; gross dwarf, like *nl1* (narrow leaf)
tlb	5	tip leaf burn; maybe like *bu1* (leaf burn)
zbc	4	zebra leaf (Caribbean Composite origin)
zbr	2	red zebra; plants with reddish zebra-marked leaves
"y8"	4	pale yellow endosperm, similar to *y8* (7S-18), or *y10*; linked to *o2*

Multiple Genetic Marker Stocks

a Dl, A B Pl C R, A C R Pr, ae wx, wx bk2, br f, C sh bz wx, A et C R, la su, Og A B Pl, o2 y

Recombinant inbred lines from tropical x temperate single crosses

--Hyeon Gui Moon and James L. Brewbaker

A series of 1068 RILs (recombinant inbred lines) have been created by selfing 6 generations from 9 single crosses of elite maize inbreds. The single crosses involve both tropical and temperate parents. The temperate parents included B73 (Iowa), DB544 (S. Korea), Fla2BT73 (Florida) and Hi31 (Hawaii, ex Iowa inbred B68). The tropical parents included Hi34 (Hawaii, ex Antigua), Ki14 (Thailand), Narino330 (Philippines, ex. Colombia), Tx601 (Texas), Tzi4 (Nigeria, ex Guatemala x Nigeria) and Tzi17 (Nigeria). At least 20 sublines were derived from each of the 9 parents by selfing, for use in establishing expected normal distributions for QTLs (Brewbaker, Quantitative Genetics on a Spreadsheet, 1994).

All crosses were made to exploit genetic differences in disease and pest resistance (Kim, Brewbaker and Hallauer, Proc. Corn and Sorghum Res. Conf. 43:194-226, 1988). The RIL families are simply designated A through J. Set G has been subjected to extensive RFLP mapping, as noted by Ming et al. in an accompanying article.

High-priority QTLs that distinguish the parents of the 9 sets of RILs are indicated below. Dentiness and daylength susceptibility segregated in almost all sets.

(A) Hi34 x Tx601: *Sphacelotheca reilana* = head smut (HEAD), *Spodoptera frugiperda* = fall armyworm (FAW), Ostrinia stalk borers (OST), plant height (PH), ear height (EH), kernel weight (KW).

(B) B73 x Narino330: Maize Dwarf Mosaic Virus (MDMV), Maize Mosaic Virus (MMV), Downy mildew (DM), *Puccinia polysora* rust (POLY), *Exserohilum turcicum* blight (TURC), OST, maturity, PH, EH, internode length (INT), tassel branches.

(C) B73 x Tx601: MDMV, DM, *Fusarium moniliforme* kernel rot (FUS), FAW, EH, leaf number (LNO), INT.

(D) B73 x Tzi4: DM, Maize Streak Virus (MSV), POLY, *Puccinia sorghi* rust(SORG), TURC, FAW, OST, maturity, seed color, LNO, INT, *Striga helmonthica* = witchweed (STRIGA).

(E) DB544 x Fla2BT73: Black Streak Dwarf Virus (BSDV), Bacterial Leaf Blight (BLB), *Helminthosporium (Bipolaris) maydis* (MAYD), STRIGA, maturity, PH, EH, LNO, tip leaf burn, tassel branches.

(G) Hi31 x Ki14: MDMV, MMV, DM, Maize Stunt Mycoplasm (STUNT), *Curvularia* (CURV), POLY, *Xanthomonas stewartii* = Bacterial (Stewart's) wilt (WILT), MAYDIS, *Ustilago maydis* (SMUT), TURC, DS, pericarp thickness (PT), staygreen (SG), tassel type, KW.

(H) Hi34 x Tzi4: MMV, MSV, DM, SORG, root lodging, seed color, INT.

(I) Hi34 x Tzi17: MMV, MSV, SMUT, SORG, TURC, Stalk rot, OST, STRIGA, seed color, SHK, tassel length, KW.

(J) Tx601 x Tzi4: MMV, SMUT, SORG, TURC, OST, seed color, PH.

Evaluations conducted since 1993 suggest that the following QTLs are associated with monogenic variation among the parents (H.G. Moon, PhD thesis, U. Hawaii, 1995): common and polysora rusts, bacterial leaf blight, Stewart's bacterial wilt, *maydis* and *turcicum* blights, MMV and MSV viruses, and "Staygreen" (probable tolerance to *Fusarium moniliforme*). Digenic variations have been suggested for pericarp thickness and tolerance to European corn borer and to *Striga* (witchweed). Where desirable

and possible, closely linked molecular markers of these QTLs are being identified to serve as "flags", hopefully to accelerate genetic gains in maize.

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RFLP mapping of maize mosaic virus resistance gene

--R. Ming, M.D. McMullen, J.L. Brewbaker, R.C. Pratt, H.G. Moon, T. Musket, and D.T. Kyetere

Maize mosaic virus (MMV), transmitted by the leafhopper *Peregrinus maidis* (Ashmead), causes a common disease of maize in many tropical and subtropical countries. Ninety-one recombinant inbred lines (RILs), derived from the cross Hi31 (a B68 conversion) and Ki14 (a Thai inbred), were used for MMV resistance gene mapping. Previously, a resistance gene in Hi31, tracing back to Caribbean flints (Brewbaker and Aquilizan, Crop Sci. 5:412-415, 1965), was identified and named *Mv* (Brewbaker, Ohio So. Coop. Series Bull. 247:145-151, 1981). The RILs were screened for the disease under a natural infection in Hawaii in the summer of 1994 in a field where susceptible corn was planted successively for a year to increase the virus. The first 10 plants from each line were rated for disease response in two replications using a 1-9 scale (1=no symptom, 9=severe symptom). Twenty sub-lines each were grown of resistant parent Hi31 (mean 1.10, range 1.0 to 2.0)and susceptible parent Ki14 (mean 6.15, range 5.0 to 7.0). The disease rating for the RILs ranged from 1.0 to 7.0.

Based on the phenotypic scores, 28 highly susceptible RILs were chosen for the pooled-sampling approach. DNA from the parents, RILs, and the susceptible pool was digested by eight restriction enzymes: *Eco*RI, *Hin*dIII, *Eco*RV, *Bam*HI, *Dra*I, *Xba*I, *Bgl*II and *Sac*I. One hundred sixty-three DNA probes distributed throughout the 10 chromosomes of maize were used to screen parents and the susceptible pool. One hundred fourteen informative probes were used for analysis of the RILs. Since duplicated loci were exhibited from 13 probes, a total of 127 loci were genotyped for these RILs.

Initial evidence for the *Mv* map position was obtained from the pooled-sampling approach, as probes on chromosome 3 near the centromere were biased to the susceptible parent allele. The SAS/GLM procedure was used to determine the correlations between RFLP markers and the MMV resistance (Table 1).

Table 1. The loci significantly associated with MMV resistance from single factor analysis of variance.

Marker	Chrom.	Position*	R^2	Probabil.
umc121	3	18	0.0615	0.0206
csu16	3	40	0.1983	0.0031
php20042	3	45	0.2409	0.0020
umc102	3	78	0.3202	0.0001
csu30	3	83	0.3768	0.0001
php20508	3	84 **	0.4169	0.0001
umc26	3	86	0.4014	0.0001
bnl5.37	3	98	0.1491	0.0003

*the map position was based on the UMC 1993 RFLP map.
**estimated map position based on bnl RFLP map.

Loci on chromosome 3 near the centromere (*umc102, csu30, php20508, umc26*) showed the greatest effects, indicating a major MMV resistance gene was located in this region. This gene is present in the resistant parent Hi31 and apparently traces back to the Argentine parent used in conferring common rust resistance into B14. The results confirmed Brewbaker's suggestion from near-isogenic conversions that *mv* is linked to *lg2* and *na1* on chromosome 3 (Brewbaker, Proc. Annu. Corn Sorghum Res. Conf. 29:118-133, 1974). A number of RFLP probes on chromosomes 4, 7, and 9 were marginally significant for MMV resistance. Possible QTLs for MMV resistance on these chromosomes will need to be confirmed with larger populations.

We conclude that resistance to MMV in B68 and Caribbean flints involves a major QTL on chromosome 3 linked to RFLP marker *php20508*. This gene is probably the previously described gene *mv*.

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RFLP mapping of genes conferring resistance to *Erwinia stewartii*

--R. Ming, M.D. McMullen, J.L. Brewbaker, H.G. Moon, T. Musket, and R. Holley

QTLs for resistance to Stewart's bacterial wilt (*E. stewartii*) were mapped using recombinant inbred lines derived from Hi31 X Ki14 (see accompanying article). Ninety-six RILs and ten sub-lines of each parent had been planted at Henderson, Kentucky, by Dr. Randall Holley of Northrup-King Co., for Stewart's wilt resistance evaluation under natural infection. The bacterium *E. stewartii* overwinters in mature corn flea beetles. In the disease nursery at Henderson, beetle populations have been high and the field was under severe Stewart's wilt pressure last year. Among the 96 RILs planted at Henderson, 25 were eliminated due to herbicide damage (Beacon). The remaining 71 RILs were scored for Stewart's wilt severity in two replications using a 1-9 scale (1=no symptom, 9=severe symptom). The sub-lines of resistant parent Ki14 and susceptible parent Hi31 averaged 1.91 and 6.50, and ranged from 1.0 to 4.0 and 4.0 to 8.0, respectively. The F1 hybrid was scored 4.08, intermediate between its parents. The RILs ranged from 1.2 to 7.9.

One hundred twenty-seven RFLP loci were analyzed with the disease data using SAS/GLM (Table 1). Markers on the short arm of chromosome 1 showed the greatest effects on conferring resistance, suggesting a major QTL in this region. Another region

Table 1. The loci significantly associated with *E. stewartii* resistance from single factor analysis of variance.

Marker	Chrom.	Position	R^2	Probabil.
umc164	1	4	0.0727	0.0286
npi262	1	84	0.1191	0.0364
umc167	1	98	0.3273	0.0002
umc67	1	111	0.1224	0.0313
bnl8.29	1	264	0.0754	0.0269
umc19	9	96	0.1927	0.0083
bnl7.50	9	101	0.1176	0.0406
npi291	9	155	0.1360	0.0227

on chromosome 9 also showed significant association with resistance. On chromosomes 2, 4, 5, 7, and 8, only single markers showed significant association with disease resistance (data not shown). From previous studies, few loci have been suggested to control Stewart's bacteria wilt resistance (Blanco, Phytopathology 69:849-853, 1979).

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A test of pollen competition using TB-9Sb

--Wayne Carlson

Beckett (J. Hered. 73:29, 1982) tested male transmission of the B-translocations, TB-1La and TB-1Lc. The translocations were in the heterozygous condition: 1, 1-B, B-1. An approximate 1:1 ratio is expected for the normal vs. the translocation gamete, with other gametes being inviable or seldom transmitted. However, Beckett found that the translocation gamete was transmitted at a rate of 56-59%. This rate may be an underestimate of the translocation class. Beckett's approach was to cross the B-1 translocations into a high nondisjunction inbred background, so that most translocation gametes would show nondisjunction. Subsequently, the rate of nondisjunction was measured as an indicator of transmission of the translocations. This method will miss translocation gametes that lack nondisjunction, albeit a small number. The pollen competition effect of TB-1La and TB-1Lc was also minimized by missing the duplication gamete (1 B-1). It was counted in the normal (no nondisjunction) category. This group should be small, but may have been transmitted to some extent. Consequently, the true value for pollen competition is probably greater than that found by Beckett.

The findings for TB-1La and TB-1Lc are not consistent with published data on TB-9Sb heterozygotes. The *Wx* marker on TB-9Sb was used to detect transmission of the translocation gamete (Carlson, Maize Breeding and Genetics, p. 754, Table 4, 1978). The *Wx* marker is on the 9-B and is extremely close to the translocation breakpoint. It is unaffected by nondisjunction, since that phenomenon is confined to the B-9 chromosome. As a result, it marks all translocation gametes and does not need nondisjunction as an identifier. The data should, therefore, be very accurate in determining any advantage of the translocation gamete over normal. The table cited above contains data on male transmission of standard TB-9Sb (last entry in table). The TB-9Sb heterozygote gave 50% *Wx* transmission, indicating no competitve advantage to B-containing pollen.

One reason for the discrepancy in data between the B-1 translocations and the B-9 translocation could be survival of the duplicate gamete, A B-A. This gamete is ignored in both tests and becomes part of the "normal" gamete class. It is possible that transmission of the 9 B-9 duplicate gamete is much more frequent than that of either 1 B-1 gamete, due to a smaller amount of gene duplication. The result would be to reduce the apparent size of the 9-B B-9 gamete class.

A test was devised in which the duplicate 9 B-9 gamete could be identified and removed from the data. The heterozygote was marked as follows: 9 (*C sh wx*) 9-B (*Wx*) B-9 (*c Sh*). In a cross as a male parent to a *c sh wx* tester, the 9 B-9 gamete should give the phenotype, *C Sh wx*. While this phenotype could be produced by a crossover chromosome 9, it would require a double crossover on the B-9, including one crossover in the short *C-Sh* interval. Therefore, the *C Sh wx* class should consist almost entirely of the 9 B-9 class. Another consideration in identifying 9 B-9 gametes is the possibility that some crossover gametes are missed by identifying only the *C Sh wx* class. The crossover gametes, 9 (*C sh wx*) B-9 (*C sh*) and 9 (*c Sh wx*) B-9 (*c Sh*), would not be identified. These types occur by a crossover somewhere between *C* and the translocation breakpoint on the B-9. The rate of crossing over in this region can be estimated for normal (chromosome 9) gametes by measuring the frequency of *c wx* kernels per total *wx*. The total rate for all crosses is 1013 per 5788 or 17.5%. If the same rate occurs in 9 B-9 gametes, 17.5% of this gamete class would not be found. However, the noncrossover 9 B-9 (*C Sh wx*) class is small, and a change in it of 17.5% would not significantly affect the calculations.

Below are given data on the number of *Wx* and *wx* kernels produced in crosses of *c c sh sh wx wx* x9 (*C sh wx*) 9-B (*Wx*) B-9 (*c Sh*). Three ears were classified for each male parent.

Removal of the 9 B-9 class from the data increased the percent of *Wx* very little. The pollen competitive advantage of TB-9Sb is 53.3% (or 53.5% with an adjustment for crossing over). This value is below the 56 to 59% values found by Beckett for the B-1 translocations, despite the fact that every effort has been made to maximize the measurement of the TB-9Sb gamete class. The data do show an approximate 3.5% competitive advantage by TB-9Sb pollen over normal 9. However, one of the genes used to measure the phenomenon of pollen competition might account for the competitive advantage. The *Wx* allele transmits through the pollen at a slightly higher rate than the *wx* allele (Coe et al., Corn and Corn Improvement, p. 198, 1988). Consequently, the linkage of the 9-B B-9 gamete to *Wx* could, by itself, account for the advantage of TB-9Sb chromosomes over the normal 9. The data, therefore, show either a weak or a nonexistent pollen competitive effect of TB-9Sb.

The discrepancy between these results and those of Beckett could be explained in a number of ways. Perhaps certain genetic backgrounds allow pollen competition effects by the B chromosome and others do not. Alternately, a gametophyte effect of genes on chromosome 1 may have produced excess transmission of the B-1 translocation gametes (Beckett, 1982). Other ideas can be invoked, such as a differential effect of meiotic loss on TB-9Sb which cancels the pollen competition effect (Carlson and Roseman, Genetics 131: 211, 1992). However, all that can be concluded is that more work is needed in different genetic backgrounds and with different translocations to determine the significance of the pollen competition effect of B translocations.

Male Parent	Classification (*C Sh wx* in parentheses)		%*Wx*	% *Wx* after removing *C Sh wx*
	Wx	wx		
9528-1	554	535(28)	50.9%	52.2%
9528-2	452	477(27)	48.7	50.1
9528-3	573	541(23)	51.4	52.5
9528-5	563	552(23)	50.5	51.6
9528-6	558	509(29)	52.3	53.8
9528-7	614	558(30)	52.4	53.8
9528-8	626	480(11)	56.6	57.2
9528-9	663	571(10)	53.7	54.2
9528-10	608	512(20)	54.3	55.3
9528-11	594	562(22)	51.4	52.4
9528-13	526	491(9)	51.7	52.2
	6331	5788(232)	52.2%	53.3%

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The synthesis of two low molecular weight heat shock proteins correlates with the growth rate of maize lines at enhanced temperature

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The response of plants to the influence of high temperature is closely related to the synthesis of specific proteins called heat shock proteins (HSPs) (Lindquist and Craig, Annu. Rev. Genet. 22:631-677, 1988; Vierling, Annu. Rev. Plant Physiol. Plant Mol. Biol. 42:579-620, 1991, for review). The adaptive role of HSP synthesis is well known and the physiological functions of a number of proteins are fairly well studied. The role of low molecular weight (LMW) HSPs of plants remains less studied at present. The diversity of this group of proteins makes it particularly difficult to determine their functions.

Seedlings of inbred lines were used as a model system to give a preliminary determination of LMW HSPs in order to examine them at length. The growth response of young plants to high temperature indicates the ability of the line to adapt to experimental conditions. The investigations performed showed a relation between the synthesis of two LMW HSPs and the ability of the line to enhance the rate of growth at high temperature.

The following inbred lines were used in the study: A34, A334, A166, L, Ma21, H513, S54, SV56, Oh43, HY WS, W32, V158, A344, Ma23C, A340, A295, A347, A375, A641, PLS61 and W834 (kindly provided by Krasnodar Research Institute of Agriculture, Russia). The adaptive ability of the seedlings and the set of low molecular heat shock proteins were studied. Three-day-old seedlings were transferred to a growth chamber on a medium containing Knopp nutrient solution. The first group of plants was kept at 28°C and the second one at 38°C. The relative humidity was 60%. The shoot length was measured in 30 plants of each group every day starting from the fifth day of germination. The measurements were performed up to the stage of the appearance of the fifth leaf, i.e. 13-15 days from the emergence of seedlings. The curve of changes in the above-ground part of the seedlings in both groups was of an S-shaped form during the experiment. The coefficients were calculated in the equation of linear regression for curves showing the change in the length of the above-ground part of the seedlings. In this case, the coefficient of regression is the average growth rate of plants of each line at 28°C or 38°C. To calculate the coefficients of regression (R_1 at 28°C and R_2 at 38°C), the middle part of the curve, an almost linear one (from the 6th to the 11th day of germination), was used. The ratio of the coefficient of regression at 38°C to that at 28°C shows changes in the growth rate under the effect of high temperature and the ability of plants to adapt to it. This ratio was called the coefficient of thermo-adaptation (C_t). Average coefficients of regression and coefficients of variation were calculated from the sampling of coefficients of regression at 28°C and 38°C in the lines studied.

Table 1. The growth rate of varied maize lines at control (28°C) and high (38°C) temperature, indicated by regression coefficients (R_1 and R_2) and thermoadaptation coefficient (C_t) which is R_1/R_2.

Line	28°C R_1	38°C R_2	C_t
A334	29.45 ± 2.01	38.55 ± 2.55	1.31
A347	27.9 ± 2.27	34.69 ± 2.40	1.24
V158	25.09 ± 0.97	30.14 ± 0.99	1.20
A34	21.1 ± 1.10	25.0 ± 0.67	1.18
L	22.77 ± 1.55	26.4 ± 0.66	1.16
SV56	28.1 ± 1.36	31.85 ± 1.44	1.13
W32	24.46 ± 0.06	27.2 ± 0.84	1.11
HyWS	22.1 ± 0.92	22.4 ± 0.80	1.08
A641	29.43 ± 1.78	31.51 ± 1.04	1.07
Oh43	20.99 ± 1.26	22.4 ± 0.89	1.07
A166	30.29 ± 2.60	31.9 ± 1.04	1.05
A344	26.8 ± 1.30	26.47 ± 0.78	0.99
S54	30.2 ± 1.65	29.4 ± 1.19	0.97
PLS61	34.15 ± 1.30	32.9 ± 0.95	0.96
A295	27.2 ± 1.10	25.6 ± 1.00	0.94
Ma23C	26.89 ± 0.90	24.88 ± 1.32	0.93
A375	28.29 ± 1.19	25.04 ± 0.64	0.89
W834	22.75 ± 1.03	19.5 ± 0.64	0.86
A340	27.2 ± 1.57	23.2 ± 1.02	0.85
H513	29.29 ± 1.43	24.77 ± 1.16	0.85
Ma21	26.8 ± 1.70	20.6 ± 1.60	0.77

The temperature of 38°C appears to be critical for seedlings. At hyperthermia the growth rate of the seedlings increases, decreases, or remains unchanged (Table 1). Thus, lines can be distinguished by varying responses (according to the growth rate) to the high temperature effect. The average value of the coefficient of regression did not change greatly at the high temperature: 26.73 at 28°C and 27.35 at 38°C. However, the coefficient of variation increased from 12.76 at 28°C to 17.95 at 38°C. This increase resulted from varying changes in the growth rate in different genotypes under conditions of high temperatures. The ability of the seedlings to grow at 38°C, and hence the adaptive potentials of the given genotype are expressed by the coefficient of thermo-adaptation, C_t (R_2/R_1 ratio). This coefficient varied from 0.77 (Ma21 line) to 1.31 (A334 line).

To analyze the set of LMW HSPs, eight lines were selected. Three of them showed various degrees of inhibition of growth at high temperature: Ma21 (C_t=0.77), H513 (C_t=0.85) and S54 (C_t=0.97). In other lines the C_t increased in different degrees: A166 (C_t=1.05), SV56 (C_t=1.13), L (C_t=1.16), A34 (C_t=1.18) and A334 (C_t=1.31). The 3-day-old etiolated seedlings of selected lines were incubated in the presence of ^{35}S-methionine for three hrs at 28°C (control) and 41°C (heat shock). The labeled proteins were used in electrophoresis according to Laemmli (Nature 227:680-685, 1970). Radiographs of the gels were analyzed using a densitometer.

All lines were found to synthesize the heat shock proteins at hyperthermia, though their number and the intensity of the synthesis varied. Table 2 includes all polypeptides with molecular

Table 2. The synthesis of low molecular weight heat shock proteins by varied maize lines (Ct is given just beneath each maize line). The seedlings were incubated at 27 C (c) and 41 C (h) for 3 h. The labeled proteins were separated by SDS-PAGE. Radiographs of the gels were analyzed on a densitometer. The synthesis was on high (h), medium (m) and low (l) level, or not detected (-).

Mr	A334		L		A34		SV56		A166		S54		H513		Ma21	
	1.31		1.16		1.18		1.13		1.05		0.97		0.85		0.77	
kDa	c	h	c	h	c	h	c	h	c	h	c	h	c	h	c	h
32.5	-	l	-	m	l	m	l	m	-	h	l	m	-	m	-	m
31	l	m	l	m	m	m	m	m	l	m	-	-	l	m	l	m
30	l	l	l	l	l	l	-	-	l	m	l	l	m	l	l	l
29	-	l	-	l	l	m	l	m	-	l	-	-	-	-	-	-
28	l	h	l	h	m	h	l	h	l	m	m	h	-	h	l	m
27	-	m	m	h	-	m	-	m	m	m	-	m	m	h	m	m
25	-	m	m	h	-	m	m	h	-	m	l	h	-	h	-	l
23	-	l	m	h	-	l	-	-	-	m	-	-	l	m	m	m
22.5	-	-	-	l	-	-	-	-	-	-	-	-	-	-	-	-
22	l	l	-	l	l	m	m	m	-	m	l	m	-	-	-	-
21.5	m	-	m	m	m	m	-	-	l	m	m	m	m	m	m	-
20	-	m	-	m	-	m	-	m	l	l	-	m	-	m	-	-
19.5	m	h	m	h	m	h	m	h	m	h	m	h	m	h	m	h
19	l	h	-	h	l	m	l	m	-	l	-	h	-	m	-	h
18	l	m	m	m	l	m	l	m	m	m	m	l	m	m	l	h
16	m	m	m	m	m	m	m	m	-	m	m	l	m	l	-	-
13	m	m	m	m	m	m	-	-	-	m	m	-	m	l	l	-
9	l	l	l	m	l	l	-	-	l	l	-	m	l	l	l	l

weight (MW) from 9 to 32 kDa if the synthesis of this polypeptide was enhanced at increased temperature in at least a single line studied.

The diversity of the LMW HSPs observed is rather high. Eighteen groups of polypeptides with varying MW were obtained. To facilitate the analysis, the polypeptides were separated into major, i.e. being actively synthesized, and minor ones with poor label incorporation. The major groups were composed of HSPs with MW 32.5, 31, 28, 27, 25, 19.5, 19 and 18 kDa. Other polypeptides belong to the minor groups.

A number of major HSPs with MW 32.5, 28, 27, 25, 19.5 and 19 kDa were synthesized in all lines studied. Only the S54 line failed to synthesize two major HSPs with MW 31 and 18 kDa (Table 2). Looking for a possible relation between the coefficient of thermo-adaptation and the HSP synthesis, attention was paid to the minor HSPs with MW 29 and 16 kDa. The 29 HSP was synthesized only in lines growing faster at the high temperature than at the control temperature. The protein with MW 16 kDa was synthesized in lines H513 and S54 at the control temperature but was greatly inhibited by heat shock. This protein was completely absent in the Ma21 line with the lowest C_t. The synthesis of this polypeptide increased or was maintained at the same level only in lines with the coefficient of thermo-adaptation greater than one unit. The data suggest a relation between the synthesis of HSPs 29 and 16 and the adaptation processes in young plants at high temperature.

The mechanism of the effect of the HSPs 29 and 16 on the capacity of maize to grow intensively at enhanced temperatures is obscure. It may be related to the effect of these proteins on the effective function of the mitochondria at high temperatures. The association of some LMW HSPs with seedling mitochondria has been shown upon heat shock in vivo. In this case the HSPs 29 and 16 were associated with the outer membrane of these organelles (Borovskii and Vojnikov, Russian J. Plant Physiol. 40:596-598, 1993). Furthermore, it is known that the association of LMW HSPs with mitochondria contributes to the oxidative phosphorylation abilities of isolated mitochondria in vitro (Chou et al., Plant Physiol. 89:617-621, 1989; Vojnikov et al., Plant Physiol. (Life Sci. Adv.) 8:1-4, 1989). Recently the strong thermo-stabilization influence of plant HSPs with MW 15-18 kDa on soluble and membrane associated proteins has been reported (Jinn et al., Plant Cell Physiol. 34: 1055-1062, 1993). The molecular mechanism of these proteins' action may be related to chaperone, since a chaperone function has been demonstrated in vitro for a mammalian LMW HSPs (Jacob et al., J. Biol. Chem. 268:1517-1520, 1993).

The redox control of genetic functions in isolated mitochondria

--Yuri M. Konstantinov, Galina N. Lutsenko and Vladimir A. Podsosonny

The problem of regulation of genetic functions of plant mitochondria in response to change of metabolic conditions in the cells and the whole organism remains poorly understood. The hypothesis has recently been advanced on the redox control of gene expression and functions of genomes of chloroplasts and mitochondria (Allen, J. Theor. Biol. 165:609-631, 1993). According to the hypothesis, two types of regulatory protein factors (so called "redox sensors" and "redox response regulators") form the two-component regulatory system. This system may place expression of mitochondrial and chloroplast genes under redox regulatory control (Allen, 1993). The aim of the present work was to study a possible redox control of the mitochondrial genome functions in isolated mitochondria. The kinetics of the RNA and DNA syntheses in mitochondria has been examined in the presence of potassium ferricyanide, an oxidising agent, and sodium dithionite, a reducing agent. In addition, the effect of particular inhibitors of protein kinases and phosphatases on the activity of nucleic acid synthesis in mitochondria has been examined, since hypothetical redox sensors and redox response regulators perform their regulatory functions in the form of phosphorylated protein molecules (Allen, 1993) and serve as a target of protein kinase and protein phosphatase action.

The mitochondria were isolated from 3-day-old etiolated maize seedlings (hybrid VIR42 MV) by a standard method of differential centrifugation. Mitochondrial protein was determined by the Lowry method. DNA synthesis in mitochondria was determined according to the method of Schegget and Borst (Biochim. Biophys. Acta 95:235-248, 1971) with the use of 32P-dATP with specific radioactivity 185 PBq mol-1. The conditions for measuring RNA synthesis in mitochondria were essentially similar to those described elsewhere (Carlson et al. Curr. Genet. 11:151, 1986). The kinetics of the mtRNA sysnthesis was recorded by 32P-UTP with specific activity > 74 pBq mol-1. The kinetic data were obtained from at least 3 to 4 experiments.

The observed specific changes of the transcription of the mitochondrial genome following the addition of ferricyanide and dithionite to mitochondria (Table 1) suggest the existence of the redox control of template functions of the mitochondrial genome. A significant activation of the transcription was observed in the presence of ferricyanide, while the addition of dithionite was followed by the inhibition of the RNA synthesis in mitochondria.

The changes in redox conditions in the mitochondrial system caused by the addition of ferricyanide or dithionite were also followed by changes in the activity of the DNA synthesis in these organelles (Table 2). Furthermore, a significant decrease in the DNA synthesis activity was observed in the presence of both oxidising and reducing agents, which was more prominent in the case of dithionite. The effect of the repression of the DNA synthesis was promoted in the presence of sodium fluoride, an inhibitor of protein phosphatases.

The effect of sodium fluoride on the level of the DNA and RNA syntheses in mitochondria (Tables 2 and 3) can presumably be accounted for by dependence of the activity of redox regulatory phosphoprotein factor(s) (Allen, 1993) on their phosphorylation state. Such influence of sodium fluoride on the genetic processes in mitochondria (activation of transcription and repression of the DNA synthesis activity) can presumably result from the competitive interrelationships between RNA and DNA syntheses at the level of genetic templates under the redox control, as well as from prevailing of transcription over replication under a redox potential decrease.

Table 1. The effect of potassium ferricyanide and sodium dithionite on kinetics of RNA synthesis in mitochondria of maize hybrid VIR42 MV.

Conditions	Incorporation of 32P-UTP into acid insoluble mitochondrial fraction, counts/min mg protein			
	5 min	10 min	15 min	20 min
Control	6810	8889	16300	37463
Ferricyanide (5 mM)	9247	12041	19809	41245
Dithionite (5 mM)	5020	6712	15014	31486

Table 2. The effect of potassium ferricyanide and sodium dithionite on kinetics of DNA synthesis in mitochondria of maize hybrid VIR42 MV.

	Incorporation of 32P-dATP into acid insoluble mitochondrial fraction, counts/min mg protein			
Conditions	5 min	10 min	15 min	20 min
Control	21825	31791		41668
Ferricyanide(5mM)	17043	21090	27951	31103
Dithionite(5mM)	11033	13750	22721	26053
NaF(40mM)	20830	21623	26593	30016
Ferricyanide+NaF	8611	17115	25105	27826
Dithionite+NaF	6213	12106	13526	19243

Table 3. The effect of protein phosphatase inhibitor sodium fluoride on kinetics of RNA synthesis in mitochondria of maize hybrid VIR42 MV.

	Incorporation of 32P-UTP into acid insoluble mitochondrial fraction, counts/min mg protein			
Conditions	5 min	10 min	15 min	20 min
Control	5865	6832	10926	20908
NaF (40 mM)	6807	12359	12740	30441

The kinetics of RNA and DNA syntheses in mitochondria in the presence of different concentrations (10, 25, 50, 100 and 200 nM) of staurosporine, an inhibitor of protein kinases, showed an increase in the activity of transcription and a decrease in the activity of DNA synthesis (data not shown). The changes in kinetic parameters of nucleic acid synthesis correlated with the staurosporine concentrations used. The results of the inhibitor analysis performed with sodium fluoride and staurosporine suggest the existence of at least two phosphoproteins regulating the transcription and replication in mitochondria, which affects differently the activity of these processes. It could not be ruled out that these proteins perform their regulatory action by binding with the corresponding polymerase complex.

As a whole, the changes in transcription and DNA synthesis under the influence of reducing and oxidising agents in isolated mitochondria suggest that the system of the redox control of genetic functions of mitochondria may also exist under in vivo conditions.

The reciprocal dependence of RNA and DNA syntheses in mitochondria of different genotypes

--Y.M. Konstantinov, A.S. Mashnenkov, G.N. Lutcenko and V.A. Podsosonny

The study of the relationships between transcription and replication of DNA in mitochondria (mtDNA) of different genotypes can provide valuable information on functional organization of the mitochondrial genome of maize. Possible relationships between the transcription and replication in the cytoplasmic organelles of eukaryotes can be implied from the data obtained with vertebrate mitochondria. It is known that the initiation of transcription of the L-strand and the transcriptional activation of the H-strand replication originate from the same DNA site (Clayton, Annu. Rev. Cell. Biol. 7:453-478, 1991). RNA-polymerase has been found in yeast mitochondria, that is likely to provide the synthesis of RNA primer in order to replicate mtDNA (Christianson and Rabinowitz, J. Biol. Chem. 258:14025-14033, 1983). Besides, there is no evidence on the relationship between activities of RNA- and DNA-synthesizing systems in mitochondria of different corn genotypes. Therefore, the purpose of this work was to study the kinetics of the synthesis of RNA and DNA in the isolated mitochondria of different inbreds and hybrids of maize.

In our experiments, the mitochondria used were immediately isolated from the coleoptiles of 3-day-old etiolated seedlings of inbred lines: GK26zakT, W64A, B84zakT, Mo17TV, B73zakT and hybrids: BEKKE LLO, Dneprovsky 320 MV, Odessky 44,

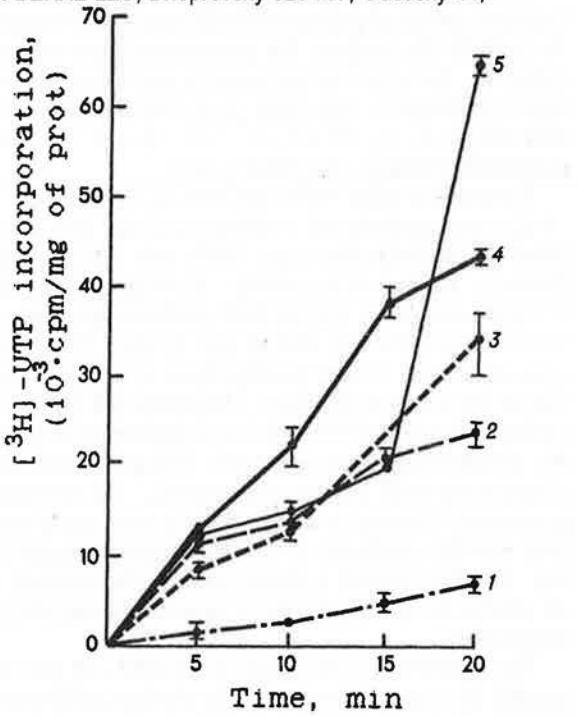

Fig. 1. Kinetics of RNA synthesis in isolated mitochondria of different maize lines. 1, GK26; 2, W64A; 3, B94zakT; 4, Mo17TB; 5, B73zakT.

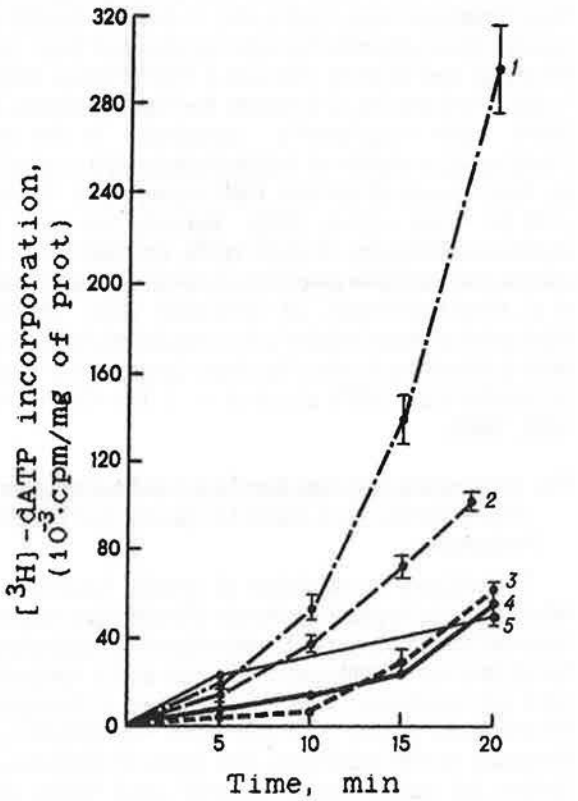

Fig. 2. Kinetics of DNA synthesis in isolated mitochondria of maize lines. Designations are as in Fig. 1.

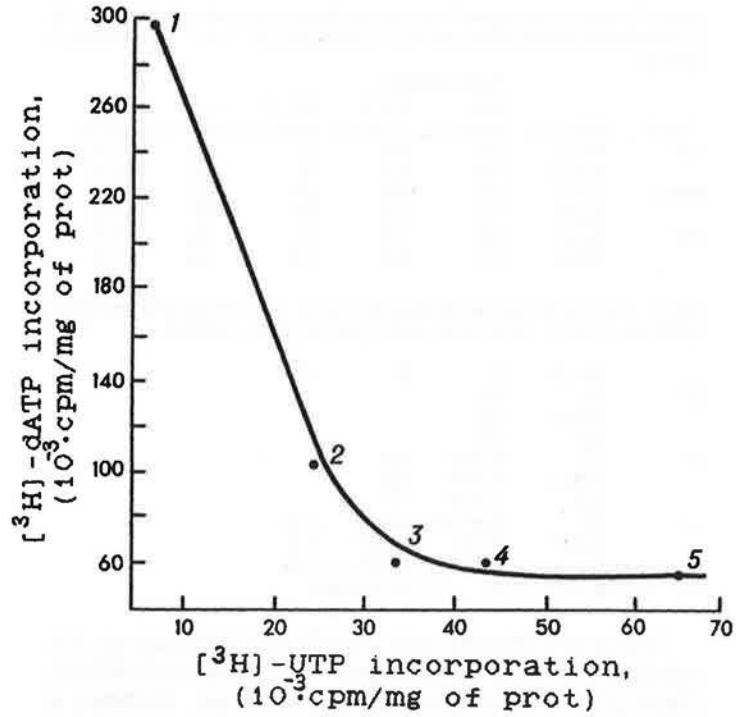

Fig. 3. Dependence between RNA and DNA syntheses in mitochondria of different maize lines. The organelles were exposed to labeled precursors for 20 min. Designations are as in Fig. 1.

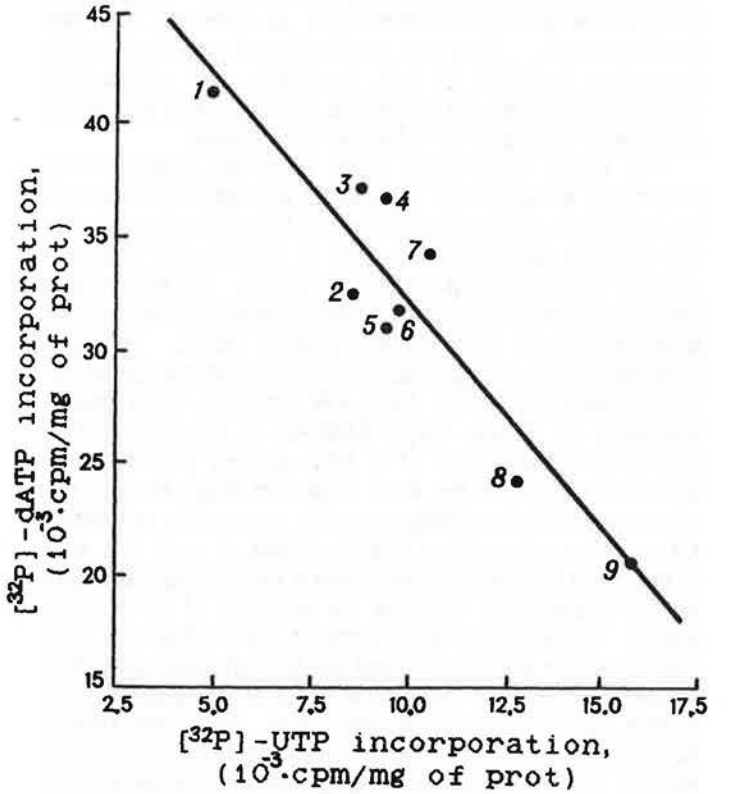

Fig. 4. Dependence between RNA and DNA syntheses in mitochondria of different maize hybrids. 1, BEKKE LLO; 2, Kollektivny 244MV; 3, Krasnodarsky 303ATV; 4, Zherebkovsky 86MV; 5, Odessky 44; 6, Rossijsky 1; 7, Dneprovsky 320 MV; 8, VIR 46MV; 9, Krasnodarsky 362ATV.

Kollectivny 244MV, Rossijsky 1, VIR 46MV, Krasnodarsky 362ATV, Krasnodarsky 303 ATV and Zherebkovsky 86MV. The mitochondria were isolated by a standard method of differential centrifugation. Protein was determined by the Lowry method. DNA synthesis was measured in mitochondria according to the method of Schegget and Borst (Biochim. Biophys. Acta 95:235-248, 1971) with the use of ^{32}P-dATP (specific radioactivity was > 111 Pbq mol^{-1}) or ^{3}H-dATP (980 Tbq mol^{-1}). The conditions for measuring RNA synthesis in mitochondria were essentially similar to those described elsewhere (Carlson et al., Curr. Genet. 11:151, 1986). The kinetics of the RNA synthesis was registered by ^{32}P-UTP (specific activity was > 74 Pbq mol^{-1}) or ^{3}H-UTP (specific activity was 550 Tbq mol^{-1}). The kinetic data were obtained from at least 3-4 experiments.

The kinetics of the RNA and DNA syntheses was recorded in the mitochondria of five maize lines within 20 min intervals, and showed that each inbred line had its particular relationships to the activities of the template process of nucleic acid synthesis. Figs. 1 and 2 show low DNA synthesis in mitochondria of maize lines B73zakT, Mo17TV and B84zakT, which possess a high transcriptional activity of mtDNA. Moreover, the mitochondria of GK26zakT lines, which synthesize mtRNA at a much lower rate, show a greater DNA-synthesizing rate. Only line W64A is somewhere in the intermediate position according to the rate of the mitochondrial RNA and DNA syntheses. Thus, we can conclude that in the genetic system of mitochondria of the maize genotypes studied there are reciprocal relations between the rate of RNA and DNA syntheses. In each line, this relation can be followed from the apparent prevalence (except for the W64A line) of one of the two types of template activity of mtDNA. Figure 3 shows the relationships between the characteristics of the mitochondrial systems of nucleic acid syntheses in all maize lines studied. A negative correlation between the activities of these systems can be seen. The coefficient of correlation calculated by the standard formula is -0.99.

A negative correlation between activities of mtRNA and mtDNA syntheses in organello (coefficient of correlation is -0.92) was also revealed for nucleic acids synthesis in mitochondria of corn hybrids (Figs. 3, 4).

The molecular mechanism responsible for genotype-specific relations of the activities of RNA- and DNA-synthesizing systems of maize mitochondria is still unknown. Neither is its biological significance in the genetic system of these organelles and the cell as a whole. However, it can be assumed that the reciprocal relationships between the synthesis of the two classes of the main genetic macromolecules in mitochondria depend on the size of the corresponding pools of nucleic acid precursors (ribonucleoside triphosphates and desoxyribonucleoside triphosphates), which are formed involving ribonucleotide reductase (EC 1.17.4.1). This enzyme catalyzes the direct recovery of ribonucleoside di- or triphosphates into corresponding desoxy derivatives and provides the DNA synthesis with the necessary pool of deoxyribonucleoside triphosphates (Reichard and Ehrenberg, Science 221:514-519, 1983). In this case, there are favorable conditions for a highly efficient synthesis of mtDNA in genotypes with a high activity of mitochondrial ribonucleotide reductase. Conversely, in genotypes with a low mitochondrial ribonucleotide reductase activity a large-sized pool of ribonucleoside triphosphates contributes to a high activity of the synthesis of the mitochondrial RNA.

In analyzing the results of the present study, we should bear in

mind that the prevalence of a single kind of template activity of DNA in maize mitochondria is somehow related to inter-genotype differences, both in the degree of polyploidy of the mitochondrial genome and in the total content of mitochondria in the cell.

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Knob DNA in maize: its relationship with combining ability for yield and maturity in different environments

--Sajjad R. Chughtai, J. Crossa*, H.I. Javed, M. Aslam and M. Hussain

Heterochromatic DNA in maize and its close relatives in the Maydeae is present in the form of highly condensed knobs at fixed locations on the chromosomes. The racial composition and geographic distribution of knobs is not random. Different sets of knobs are present in different races of maize. The high knob races are restricted to the lowlands while low knob races are restricted to the highland areas. Also, the knob frequency decreases with increasing latitude. Apparently, the amount of knob heterochromatin is related to the growing season and thus to the adaptation of maize to its environment.

Crossa et al. (Crop. Sci. 30:1182-1190, 1990) published extensive data on combining ability among 25 Mexican races of maize. The hybrids among these races were developed and evaluated at three different altitudes (1300, 1800 and 2249 meters) in Mexico during the early sixties. In collaboration with Dr. J. Crossa from CIMMYT, Mexico, we have been analyzing these data with respect to the knob composition of races and their hybrids based on knob data published by McClintock et al. (Chromosome constitution of races of maize: Collegio de Postgraduados, Chapingo, Mexico, 1981). Earlier (MNL 67:49-50, 1993), we concluded that depending on the environment, the number of homozygous or heterozygous knobs determined the hybrid performance. Now some more data along with days to pollen shedding are presented to corroborate our earlier conclusion regarding a relationship between knob DNA and combining ability for yield and maturity. It is very important to note that the relationship between knob DNA and combining ability for yield and maturity largely depends on the environment. At low altitude, both the top 50 and bottom 50 hybrids took about 78 days to mid-flowering. The main difference was in the number of knobs. The top 50 and bottom 50 hybrids on the average had 7.8 and 2.44 homozygous, and 3.46 and 5.56 heterozygous knobs, respectively. At intermediate elevation, the top 50 hybrids, on the average, were 5 days later in pollen shedding than the bottom 50. Regarding their knob composition, the top 50 had 6.84 homozygous and 3.66 heterozygous knobs compared with 3.86 homozygous and 5.04 heterozygous knobs in the bottom 50 hybrids. At high altitude, the top 50 and bottom 50 hybrids took 91 and 105 days to pollen shedding, respectively, and had 4.06 and 8.26 homozygous, and 3.64 and 5.58 heterozygous knobs, respectively. At high altitudes, the yield increased with increase in the number of heterozygous knobs though the maturity was reduced. The top 50 hybrids have a higher number of ears per plant than the respective bottom 50 hybrids at all elevations (Table 1).

Table 1. Number of knobs, days to flower, ears per plant and grain yield in the top and bottom 50 hybrids among Mexican races grown at low (1300 m), medium (1800 m) and high (2249 m) altitudes.

		Number of Knobs				
Altitude	Rank	Homo-zygous	Hetero-zygous	Days to flower	Ears/ plant	Yield (T/ha)
Low	Top 50	7.82	3.46	78	1.30	5.86
	Bottom	2.44	5.56	77	1.08	2.65
Medium	Top 50	6.84	3.66	88	1.62	7.63
	Bottom	3.86	5.04	83	1.51	3.65
High	Top 50	4.06	5.58	91	1.66	5.84
	Bottom	8.26	3.64	106	1.12	1.65

Table 2. Correlation between days to flower (DF), ears per plant (EP) and grain yield (GY) in hybrids grown at low (1300 m), medium (1800 m) and high (2249 m) altitudes.

	Altitude	DF	EP	GY
DF	Low	1.00		
	Medium	1.00		
	High	1.00		
EP	Low	-0.495**	1.00	
	Medium	-0.215**	1.00	
	High	-0.510**	1.00	
GY	Low	-0.067 NS	0.270**	1.00
	Medium	-0.138*	0.194**	1.00
	High	-0.363**	0.561**	1.00

NS = nonsignificant; * = significant; ** = highly significant

Correlations between yield, maturity and prolificacy on 300 hybrids and 25 parental races grown at three different altitudes (Table 2) elucidate some interesting relationships. Prolificacy is negatively but highly significantly correlated with maturity at all sites. In other words, earlier maturing hybrids have a highly significant probability of producing a second ear. Maturity and yield show different relationships at different altitudes (environments). They are positively but non-significantly correlated (r=0.067) at low, positively and significantly correlated (r=0.138) at intermediate, but negatively and highly significantly correlated (r=-0.363) at high altitude.

Our earlier explanation (MNL 67:49-50, 1993) of the relationship between knob composition and hybrid performance was that in the temperate environments, knob heterozygotes (like the knobless genotypes) are early in maturity and thus well adapted to the local agroclimatic conditions. We thought that knob homozygosity delayed plant development in cooler climates probably through the cis-acting position effect. The data presented in this communication strongly support this contention. We (Maydica 32:171-187, 1987; S.R. Chughtai, Ph.D. Thesis, University of Illinois, 1988; SABRAO J. 21:21-26, 1989) proposed the existence of a DNA binding protein which specifically binds to the knob DNA and thus changes the expression of the bracketing genes by a spreading or position effect. Our observation that treatment of root tips with Quinacrine (a DNA binding agent) inhibits knob condensation even at low temperatures indicates the presence of the putative protein. Quinacrine probably prevents the binding of a knob specific protein which recognized the A-T rich stretches (GAAT or GAAAAT) in knob DNA. The effect of this putative DNA binding protein is temperature dependent. Since knob DNA is highly condensed at low temperatures, the spreading or position effect is pronounced at high altitude (temperate environments) but not at the low altitude (warmer climates). In such climates, knob homozygotes are better adapted than the knob heterozygotes though they have similar maturities. Whatever the explanation may be, it is important that knob composition of hybrids (as well as landraces and varieties) of maize apparently

plays an important role in their adaptation to the environmental conditions. Consideration of this factor in breeding of maize for various target environments and purposes will ensure success more than random crossing efforts and selection. For example, breeding for cold tolerance must ensure knob heterozygosity and early maturity by choosing one of the parents with low knob number and early maturity. In other words, the early maturity of the knobless or low-knob genotypes can be coupled with the high yield of the late-maturity high-knob genotypes thus breaking the linkage between yield and maturity.

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Pioneer Hi-Bred Int.

Atlas of duplicated sequences

-- Tim Helentjaris

It was noted early on in the development of maize molecular markers that RFLP probes commonly detected two or more unlinked loci in the genome (Helentjaris et al. Genetics 118:353-363, 1988). It became evident even in these studies that these duplications extended well beyond those segments represented by the probe sequences themselves, perhaps in some cases encompassing whole chromosome arms. I have recently scanned the maize literature to compile all of the relevant data on duplicated sequences and am including my most recent version of that table here.

It should be realized that the location of many of these duplications can only be approximated to a chromosomal bin, as denoted in the Missouri RFLP map, due to the many differing studies from which the data were extracted and the lack of suitable numbers of landmark loci in some of these. Nevertheless, it can be obviously seen from this table that earlier assumptions about the extent of shared regions of sequence similarity between maize chromosomes are not only confirmed but increased considerably. The following regions evidently share extensive similarity:

1S with 9L
1L with 5S
2S with 10L
2L with 7
3 with 8
4S with 10S?
4L with 5L
terminal segments of 6 with central regions of 8
central region of 6 with 9S

These relationships are not complete in that there are many loci which appear not to fit these general patterns. Aberrant loci located near the telomeric regions are often more than just duplicated and are found in both telomeric and centromeric regions of several chromosomes. Large clusters of aberrant loci are found in the centromeric regions of all chromosomes and seem to share similarity with many other chromosomes. These two results suggest that chromosome evolution primarily occurs by the breakage and reunion of chromosomes at either their centromeric or telomeric regions, with evidence of these past events still obviously evident as "relicts" in the centromeric regions. Aberrant loci in the interior regions of arms represent in some cases transposable sequences and in others duplication events that predate the general duplication of the maize genome (i.e. alcohol dehydrogenase or sucrose synthase). The basis for other aberrant loci in these regions is still not clear.

Examination of DNA sequences both within and around genes located in these duplicated regions demonstrates that only the transcribed and their immediately surrounding sequences have maintained enough similarity to still be recognizable as sharing a common origin. Sequences found outside expressed genes appear to have diverged rapidly and are unable to detect simple duplications elsewhere in the genome. Consequently one might predict that any "random" genomic probe which detects a duplication in maize essentially represents a genic sequence, as might have been expected from the hypomethylated origin of many such clones. Examination of this hypothesis by D. Grant and myself with RFLP probes confirms that DNA sequences from many probes, selected for their *Pst*-digestibility within genomic DNA, exhibit reasonable matches with genic entries in GenBank. Given the extensive nature of these duplicated regions in the maize genome, one should probably presume that any expressed sequence is duplicated elsewhere in the genome until proven otherwise. The utility of this supposition is especially appropriate when cloning any gene, whereby the investigator should examine the corresponding region of the genome for the presence of such a duplicate gene or mutation. In my examinations of many newly isolated sequences, I have often identified corresponding sequences within predictable duplication patterns, sometimes matching them with similar mutations. In most cases examined so far, both members of a duplication appear to still be expressed, with some instances of duplicate inheritance and others where the pattern of expression has diverged enough that distinctive mutations can now be observed by the interruption of either gene. Consequently the generation of these duplicate genes has obviously contributed to the overall development of maize as a species, and its implications should be considered by both maize geneticists and breeders in their research.

Reverse genetics for maize

-- Bob Meeley and Steve Briggs

Of the scientific tools available to researchers studying gene function in organisms from other kingdoms, the ability to selectively knock out a gene is paramount. There are many examples where directed mutagenesis can be carried out simply to disrupt or otherwise recover mutations within a specific gene of interest. Accurate interpretation of the mutant phenotype most nearly describes the role of the normal gene. For plant biologists, elucidation of gene function has equal value and priority, but in the absence of needed mutants, it currently requires the use of more cumbersome techniques such as anti-sense or co-suppression, which depend upon the generation of transformants. Transformation is not a limitation for some dicot species, but transgenic methods suffer from a lack of uniformity of suppression and/or a lack of specificity for a single target gene. We have produced a new tool that will alleviate some of the difficulty in studying gene function in plants, specifically, a reverse-genetics system developed for maize. This resource consists of a collection of genomic DNAs from approximately 40,000 F1 plants produced by crossing *Mutator*-active lines* with inbred/hybrid lines (in effect, a library of the transposon-mutagenized genome), coupled with a collection of the F2 seed from each plant. *Mu* insertion alleles for a known DNA sequence can be selected from the DNA collection using PCR: a primer specific for the gene of interest is paired with a primer specific for the terminal-inverted-repeat (TIR) region of *Mu* elements. Amplification of the target gene can occur only if an allele exists in

Bin	Marker	Dup on 1?	Dup on 2?	Dup on 3?	Dup on 4?	Dup on 5?	Dup on 6?	Dup on 7?	Dup on 8?	Dup on 9?	Dup on 10?
1.01	bnl5.62		2.03				6.10-6.11			9.04	
1.01	umc164			3.07					8.09		
1.01	uaz2(gfu)	1.09									
1.01	uaz260(rpl5)			3.05							
1.01	umc94									9.09	
1.01	npi97,npi99	1.12								9.09	
1.02	npi579					5.02-5.03					
1.02	npi209									9.07	
1.02	agr294									9.06	
1.02	pds(vp5,etc.)									9.04	
1.02	umc194(gpr)									9.05	
1.02	npi404,npi403									9.06	
1.03	umc8		2.05-2.06								
1.03	uaz264(hsp)									9.05-9.06	
1.03	npi439,npi440							7.05			
1.03	npi241	1.14									
1.03	koln9									9.05-9.06	
1.03	npi109,npi335,npi273,npi358		2.06	3.04				7.04			
1.03	npi243,npi242,npi244		2.05-2.06								
1.03	agr92									9.05-9.06	
1.04	npi428,npi427									9.05-9.06	
1.04	phyB									9.05-9.06	
1.04	bnl12.06				4.04-4.05						
1.04	uaz304(hon)									9.05-9.06	
1.04	uaz266(nad)									9.05-9.06	
1.04	sod4									9.05-9.06	
1.04	uaz248(his3)					5.07					
1.04	agr153									9.05-9.06	
1.05	bnl7.21									9.05-9.06	
1.05	npi205,npi301,npi332,npi102,npi62						6.07	7L	8L		
1.05	uaz17(gfu)				4.01						
1.05	uaz21(gfu)	1.12	2.00								10.01
1.05	uaz331(tau)		2.04	3.05							
1.05	uaz274(gfu)		2.08								
1.06	obf1,2									9.04	
1.07	adpg		2.08	3.09	4.03-4.04		6.10-6.11		8.04		10.02
1.07	npi96,npi95,npi316				4.03-4.04, 4.05-4.06	5.11		7.02			
1.07	npi84,npi36				4.08						
1.07	ucsd61		2.11	3.02	4.05-4.06				8.04	9.03, 9.09	
1.08	umc82									9.04	
1.08	uaz14(gfu)									9.07-9.08	
1.08	uaz249(rps27a)			3.04					8.02, 8.10		
1.08	uaz285(tpase)					5.06			8.04-8.05		
1.08	ucsd72			3.06		5.07-5.08					10.00
1.09	npi224			3.04			6.05	7.01-7.02	8.05, 8.09		
1.09	bnl5.02					5.04					
1.09	hm1,hm2?									9.04	
1.09	amp					5.07					
1.09	ncr(nr)				4.03-4.04						
1.09	umc23				4.03-4.04						
1.09	npi246,npi245,npi340		2S				6.00				
1.09	uaz228(his2b)		2.07		4.05-4.06						10.04
1.09	uaz18(gfu)		2.06	3.07			6.00				
1.09	uaz138(gfu)					5.07			8.07-8.08		
1.09	uaz20(gfu)							7.01-7.02			
1.09	uaz19(gfu)			3.04			6.08	7.01-7.02			
1.09	uaz2(gfu)	1.01									
1.09	agr83				4.03						
1.09	npi441,npi387,npi388						6.01	7.01			
1.09	uaz205(hsp)					5.02-5.03					

1.09	pcr		2.00			5.04		7.01-7.02			
1.09	umc50			3.03							
1.10	umc83					5.03-5.04					
1.10	mdh4,mdh5					5.04					
1.10	csu71(cab)						6.10				
1.10	csu64(tau)		2.10								
1.10	umc184(glb1)		2.05								
1.10	bnl8.10								8.01		
1.10	npi569					5.02-5.03					
1.10	agr278					5.02-5.03					
1.10	agr362					5.02-5.03					
1.10	agr235					5.04					
1.10	umc27					5.03-5.04					
1.10	rpa5					5.04					
1.10	agr238					5.04					
1.11	umc140									9.06	
1.11	uaz268(gfu)					5.13		7.01-7.02			
1.11	rpa6					5.02-5.03					
1.11	umc107					5.02-5.03					
1.11	d8,d9					5.03					
1.11	uaz167					5.02-5.03					
1.11	phyA					5.02-5.03					
1.11	umc106					5.02-5.03					
1.11	adh1,adh2				4.02						
1.11	npi99,npi97									9.09	
1.12	npi225,npi98						6.02-6.03			9.09	10.04-10.05
1.12	msc1,msc2					5.03					
1.12	csu137(MADS)					5.02-5.03					
1.12	uaz130(ptk)				4.05-4.06	5.02-5.03					
1.12	umc72					5.02-5.03					
1.12	npi357,npi282(phyC)					5.02-5.03					
1.12	npi581					5.02-5.03					
1.12	pgm1,pgm2					5.02-5.03					
1.12	umc66					5.04					
1.12	umc147					5.01-5.02					
1.12	bnl17.18					5.02-5.03					
1.12	uaz166(gfu)					5.02-5.03					
1.12	uaz21(gfu)	1.05	2.00								10.01
1.12	npi75,npi226					5.01					
1.12	rpa7					5.01					
1.13	bnl8.08			3.05	4.07		6.08		8.01	9.05-9.06	
1.14	bnl8.29					5.01					
1.14	ucsd44					5.01					
1.14	npi241	1.03									
1.14	npi82,npi32		2.11								
1.14	bm2,bm1					5.06					
1.14	agr103					5.00					
1.14	agr669					5.00					
1.15	flp6(RNAb)					5.11-5.12	6.09				
1.15	csu7(gfu)							7.01-7.02			
1.16	npi361,npi294,npi359		2.03, 2.10-2.11		4.00, 4.11-4.12		6.02-6.03	7.01-7.02	8.04		10.05-10.06
2.00	php20568										10.00
2.00	pcr	1.09				5.04		7.01-7.02			
2.00	npi417										10.04
2.00	uaz21(gfu)	1.05, 1.12									10.01
2.01	npi254										10.08
2.01	uaz24(gfu)										10.04
2.01	npi350										10.08
2.01-2.02	npi577										10.08
2.02-2.03	umc53								8.08		
2.02-2.03	npi421										10.08
2.02-2.03	npi290										10.08

2.02-2.03	uaz49(zp19/22)				4.00						
2.02-2.03	uaz113(aaa)										10.08
2.02-2.03	uaz318(mb)										10.08
2.02-2.03	uaz288(gfu)			3.05							
2.02-2.03	uaz251(rps11)			3.07			6.08		8.02-8.03		10.05-10.07
2.02-2.03	npi320,npi321										10.08
2.02-2.03	npi294,npi359,npi361	1.16	2.10-2.11		4.00, 4.12		6.02-6.03	7.01-7.02	8.04		10.05-10.06
2.03	bnl5.62	1.01					6.10-6.11			9.05-6	
2.03	agr168										10.08
2.04	mpik4							7.01-7.02			
2.04	umc44										10.08
2.04	npi287,npi436										10.08
2.04	agr113										10.08
2.04	csu12(Cin4)				4.05-4.06						10.01
2.04	uaz106(gfu)						6.02-6.03				
2.04	uaz331(tau)	1.05-1.06		3.05							
2.04	tpi		2.08	3.03		5.11-5.12		7.04-5	8.02-8.03, 8.09		
2.04	uaz27(gfu)							7.01-7.02			
2.04	b,r										10.08
2.04	npi269										10.08
2.04	agr167										10.08
2.04	bnl1.45								8.04		
2.05	bnl10.42						6.02-6.03				
2.05	uaz295(orf)			3.03		5.07					
2.05	umc134						6.10-6.11				
2.05	umc8	1.03	2.06								
2.05	npi242,npi243,npi244	1.03	2.06								
2.05	uaz344(est)							7.01-7.02			
2.05	mn1,invertase							7.01-7.02			
2.05	umc184(glb)	1.10									
2.05	gf14-12										10.05-10.06
2.05	csu6(gfu)										10.04
2.05	oec				4.00, 4.05-4.06	5.02-5.03	6.02-6.03	7.03			
2.06	npi242,npi243,npi244	1.03	2.05								
2.06	uaz236(strs)									9.04	
2.06	uaz49(zp19/22)				4.01						
2.06	umc2	?		3.10							
2.06	ssu				4.08						
2.06	ucsd141							7.01-7.02			
2.06	npi11,npi317				4.00						
2.06	uaz265(sbe)						6.02-6.03				
2.06	agr216										10.08
2.06	npi340,npi245,npi246	1.09					6.00				
2.06	umc8	1.03	2.05				6.00				
2.06	uaz25(MCP)					5.02-5.03			8.02-8.03		
2.06	uaz28(gfu)							7.01-2			
2.06	uaz18(gfu)	1.09		3.07							
2.06	npi273,npi109,npi335,npi358	1.03		3.04				7.04			
2.06	npi49,npi44							7.01-7.02			
2.06-2.07	umc55				4.02						
2.06-2.07	uaz297(zpl17)						6.06		8.03		
2.06-2.07	ask2,ask1							7.01			
2.07	npi405,npi277							7.02-7.03			
2.07	npi4,npi5							7.04			
2.07	g-zein							7.02			
2.07	adpg	1.07		3.09	4.03-4.04		6.10-6.11		8.04		10.02
2.07	uaz269(kri)						6.01, 6.10		8.01		
2.07	uaz228(his2b)	1.09			4.05-4.06						10.04
2.07-2.08	umc5							7.03			
2.08	umc98							7.03			
2.08	alr1,alr2							7.03			

2.08	npi123,npi47							7.02-7.03			
2.08	tpi		2.04	3.04		5.11-5.12		7.04-7.05	8.02-8.03		
2.08	npi337,npi45							7.06			
2.08	npi221,npi216							7.02-7.03			
2.08	npi(a1)			3.09		5.11-5.12		7.02-7.03	8.06-8.07		
2.08	uaz31(gfu)							7.04		9.07-9.08	
2.08	uaz274(est)	1.05									
2.08	agr333							7.04			
2.08	agr267							7.05			
2.08-2.09	umc4								8.11		
2.09	php20569							7.04			
2.09	uaz23(gfu)						6.01				
2.09	umc125							7.04			
2.09	bnl5.21							7.04			
2.09	bnl5.61							7.04-7.05			
2.09	umc137						6.07				
2.09	npi413							7.05			
2.09	npi46,npi35							7.05			
2.09	uaz241(orf)							7.05			
2.09	uaz315(tua)							7.04			
2.09	npi118,npi113							7.05			
2.09	agr111							7.05			
2.09	bnl8.21							7.05			
2.09	umc31				4.01-4.02						
2.09	umc150						6.07		8.04		
2.09	umc36		2.10-2.11								
2.09	npi278							7.05			
2.09	npi210,npi59							7.05-7.06			
2.10	whp1,c2				4.08						
2.10	uaz298(pdsl)				4.02						
2.10	csu64(tau)	1.10									
2.10	uaz33(gfu)				4.05-4.06						
2.10	csu166(gfu)				4.05-4.06						
2.10-2.11	npi361	1.16	2.03		4.00, 4.11-4.12		6.02-6.03	7.01-7.02	8.04		10.05-10.06
2.10-2.11	umc36		2.09								
2.11	php20581							7.00			
2.11	npi400							7.00-7.01			
2.11	npi32,npi82	1.14									
2.11	ucsd106							7.00			
2.11	ucsd61	1.07		3.02	4.05-4.06				8.04	9.03, 9.09	
3.00-3.01	umc32								8.03-8.04		
3.00-3.01	npi224	1.09		3.04			6.05	7.01-7.02	8.05, 8.09		
3.00-3.01	agr116								8.04		
3.02	ucsd61	1.07	2.11		4.05-4.06				8.04	9.03, 9.09	
3.02-3.03	me1,me2						6.06				
3.02-3.03	hex1,hex2						6.06				
3.03	npi249,npi341,npi360		2.00			5.00					
3.03	npi219,npi218								8.01		
3.03	uaz164(gfu)			3.08		5.07			8.06		
3.03	uaz295(orf)		2.05			5.07					
3.03	umc50	1.09									
3.03	npi364,npi276								8.03		
3.03	tpi		2.04, 2.08			5.11-5.12		7.04-7.05	8.02-8.03, 8.09		
3.03	npi398							7.00	8.00		
3.03	npi220								8.00-8.01		
3.03	mpik35			3.04					8.03		
3.03-3.04	npi89,npi14									9.04-9.06	
3.04	umc42				4.04-4.05						
3.04	npi379,npi378										10.04
3.04	uaz339(glu)										10.04
3.04	bnl13.05								8.01, 8.03		

3.04	uaz19(gfu)	1.09					6.08	7.01-7.02			
3.04	uaz300(mtl)						6.01				
3.04	uaz249(rps27a)	1.08							8.02-8.03, 8.10		
3.04	lg3,lg4?								8.06		
3.04	npi202,npi114								8.01		
3.04	mpik32			3.05							
3.04	mpik35			3.03					8.03		
3.04	npi70,npi69								8.09		
3.04	npi358,npi335,npi273,npi109	1.03	2.06					7.04			
3.05	umc18										10.04
3.05	uaz331(tau)	1.05-1.06	2.04								
3.05	bnl8.08	1.13			4.07		6.08		8.01		9.05-9.06
3.05	zag2,zag1						6.06				
3.05	uaz288(gfu)		2.02-2.03								
3.05	pgd1,pgd2						6.01				
3.05	ucsd81							7.01-7.02			10.04
3.05	mpik32			3.04							
3.05	uaz189(rpl5)	1.01									
3.05	npi108,npi204								8.08		
3.06	agr514								8.05		
3.06	npi52,npi308								8.00		
3.06	agr184								8.07-8.08		
3.06	sdw2,sdw1?								8.05		
3.06	umc165								8.04		
3.06	agr274								8.08		
3.06	bnl10.24								8.08		
3.06	ucsd72	1.08		3.06		5.07-5.08					10.00
3.07	umc47				4.05						
3.07	umc164	1.00									
3.07	npi328								8.08-8.09		
3.07	obf3								8.10		
3.07	uaz38(gfu)				4.01						
3.07	uaz218(strs)				4.05-4.06						
3.07	bif2,bif1?								8.03		
3.07	uaz251(rps11)		2.02-2.03				6.08		8.02-8.03		10.05-10.07
3.07	uaz18(gfu)	1.09	2.06				6.00				
3.07	umc3								8.11		
3.07	umc39								8.11		
3.07	npi212								8.11		
3.07	agr50								8.06		
3.07	agr144								8.08		
3.07	uaz176(gfu)								8.07-8.08		
3.07-3.08	umc15				4.08						
3.08	npi201,npi3								8.05		
3.08	umc103								8.02		
3.08	uaz243(atp)						6.08		8.02-8.03		
3.08	npi382								8.00		
3.08	mdh3,mdh2,mdh1						6.10		8.03-8.04		
3.08	uaz164(gfu)			3.03		5.07			8.06		
3.09	cab							7.05	8.05		
3.09	bnl12.30								8.06		
3.09	npi(a1)		2.08			5.11-5.12		7.02-7.03	8.06-8.07		
3.09	sh2,bt2				4.04						
3.09	pog1						6.01		8.06		
3.09	agr568								8.04		
3.10	umc2		2.06								
3.10	csu36				4.05-4.08	5.05					
3.10	npi425,npi426								8.07-8.08		
3.10	uaz117(gfu)							7.04			10.04
3.10	agr43										10.00
3.10	uaz309(cht)										10.00
3L	uaz161(eif)				4.08-4.12		6.02-6.03			9.04	

4.00	oec		2.05		4.05-4.06			7.03			
4.00	rp4,rp1,rp5,rp6,rp9										10.01
4.00	uaz49(zp19/22)		2.02-2.03								
4.00	npi361,npi294,npi359	1.16	2.03, 2.10-2.11		4.11-4.12		6.02-6.03	7.01-7.02	8.04		10.05-10.06
4.00-4.01	zpl1(zp19/22)		2.06		4.07						
4.00	uaz280(pps)									9.07	
4.01	bnl17.13				4.03-4.04			7.01-7.02			
4.01	npi604										10.08
4.01	uaz38(gfu)			3.06							
4.01	uaz65(gfu)				4.08						
4.01	uaz68(gfu)							7.01-7.02			
4.01	uaz17(gfu)	1.05									
4.01	uaz69(gfu)				4.03-4.04						
4.01	uaz70(gfu)					5.07					
4.01-4.02	umc31		2.09								
4.01-4.02	umc87		2.00								
4.01-4.02	umc55		2.06-2.07								
4.02	uaz298(pdsl)		2.10								
4.02	adh1,adh2	1.11-1.12									
4.03	agr83	1.09									
4.03-4.04	npi316,npi95,npi96				4.05-4.06			7.02-7.03			
4.03-4.04	npi6,npi7						6.00				
4.03-4.04	npi383,npi384					5.04-5.05					
4.03-4.04	bnl17.13				4.01			7.01-7.02			
4.03-4.04	zpl2							7.01-7.02			
4.03-4.04	uaz69(gfu)				4.01						
4.03-4.04	mpik11				4.04-4.05		6.01			9.03	
4.03-4.04	uaz310(gbp)						6.00				
4.03-4.04	bt2,sh2			3.09							
4.03-4.04	ncr(nr)	1.09				5.07					
4.03-4.04	uaz71(gfu)					5.11-5.12					
4.03-4.04	gapd					5.07					10.05-10.06
4.03-4.04	mpik12								8.03	9.02	10.00
4.03-4.04	orp1,orp2							7.01-7.02			10.05-10.06
4.03-4.04	als					5.07					
4.03-4.04	umc23	1.09									
4.04-4.05	umc42			3.04							
4.04-4.05	csu12(Cin4)		2.04								10.01
4.04-4.05	npi95,npi96,npi316	1.05			4.03-4.04						
4.04-4.05	npi594						6.01				
4.04-4.05	bnl12.06	1.04									
4.04-4.05	npi367,npi30		2.00					7.02-7.03			
4.04-4.05	umc47			3.07							
4.04-4.05	npi394,npi395							7.03			
4.04-4.05	lw4,lw5?					5.01					
4.04-4.05	ucsd64				4.05-4.06	5.02-5.03	6.06		8.03		10.00
4.04-4.05	mpik11				4.03-4.04		6.01			9.03	
4.05-4.06	umc156					5.07					
4.05-4.06	umc126					5.07-5.08					
4.05-4.06	npi340,npi245,npi246		2.06								
4.05-4.06	ucsd64				4.04-4.05	5.02-5.03	6.06		8.03		10.00
4.05-4.06	ucsd61	1.07	2.11	3.02					8.04	9.03, 9.09	
4.05-4.06	ucsd78						6.06		8.03		
4.05-4.06	uaz230(gfu)						6.10				
4.05-4.06	uaz130(ptk)	1.12				5.02-5.03					
4.05-4.06	uaz228(his2b)	1.09	2.07								10.04
4.05-4.06	oec		2.05		4.00	5.02-5.03		7.03			
4.05-4.06	uaz144(atp)					5.09					
4.05-4.06	csu36(rpl5)			3.10		5.05					
4.05-4.06	uaz218(strs)			3.07							
4.06-4.07	umc66	1.12				5.04					
4.07	bnl8.08	1.13		3.05			6.08		8.01	9.05-9.06	

4.07	bnl10.17										10.00
4.07	umc104					5.12-5.13					
4.07-4.08	umc133						6.10				
4.07-4.08	uaz252(ptk)								8.03		
4.08	npi253					5.10	6.04-6.05			9.01	
4.08	uaz123(gfu)						6.09	7.03			
4.08	uaz65(gfu)				4.01						
4.08	uaz33(gfu)		2.10								
4.08	npi208,npi366										10.00
4.08	npi292,npi363					5.11-5.12					
4.08	umc15			3.07-3.08							
4.08	npi300,npi431							7.05		9.05-9.06	
4.08	c2,whp		2.10								
4.08	csu166(gfu)		2.10								
4.08	ssu		2.06								
4.08	agr563					5.11-5.12					
4.08	npi36,npi84	1.06-1.07									
4.08-4.09	npi450,npi424					5.06					
4.08-4.09	uaz279(gfu)					5.07					
4.08-4.09	npi371,npi422,npi372								8.03		10.00
4.08-4.09	mgs2									9.05-9.06	
4.08-4.09	uaz175(gfu)										10.04
4.09-4.10	npi203,npi116					5.04-5.05					
4.10-4.12	npi361,npi294,npi359	1.16	2.03, 2.10-2.11		4.00		6.02-6.03	7.01-7.02	8.04		10.05-10.06
4.10	uaz161(eif)			3L			6.02-6.03			9.04	
4.10	ncr(B70)					5.05					
4.10	agr1002					5.06					
4.10	agr248					5.06					
4.11	uaz279(gfu)					5.06					
4.12	csh(cdc2)									9.09	
5.00	agr259	1L?									
5.00	umc68					5.10-5.11					
5.00	uaz76(gfu)										10.05-10.06
5.00	agr699	1.16									
5.00	agr103	1.16									
5.01	rpa7	1.12									
5.01	bnl8.29	1.14									
5.01	ucsd44	1.14									
5.01	bnl7.24									9.04	
5.01	npi75,npi226	1.12									
5.01	npi579	1.02									
5.01-5.02	umc147	1.12									
5.02-5.03	npi305,npi307,npi437						6L				10.05-10.06
5.02-5.03	uaz166(gfu)	1.12									
5.02-5.03	bnl17.18	1.12									
5.02-5.03	npi282,npi357	1.12									
5.02-5.03	npi581	1.12									
5.02-5.03	csu137(MADS)	1.12									
5.02-5.03	uaz130(ptk)	1.12			4.05-4.06						
5.02-5.03	ucsd64				4.04-4.05, 4.05-4.06		6.06		8.03		10.00
5.02-5.03	umc72	1.12									
5.02-5.03	MSC2,1	1.12									
5.02-5.03	phyA	1.11									
5.02-5.03	csu150(rpo)						6.01				
5.02-5.03	oec		2.05		4.00, 4.05-4.06			7.03			
5.02-5.03	umc107	1.11									
5.02-5.03	rpa6	1.11									
5.02-5.03	umc106	1.11									
5.02-5.03	uaz167(gfu)	1.11									
5.02-5.03	uaz215(odo)					5.09					

5.02-5.03	uaz345(zta)					5.11-5.12					
5.02-5.03	pgm1,pgm2	1.12									
5.02-5.03	d9,d8	1.11									
5.02-5.03	uaz25(mcp)		2.06						8.02-8.03		
5.02-5.03	agr362	1.10									
5.02-5.03	agr278	1.10									
5.02-5.03	niu								8.03-8.04		
5.02-5.03	uaz205(hsp)	1.09									
5.03-5.04	umc27(gfu)	1.10									
5.03-5.04	umc83	1.10									
5.04	umc186(Bs1)								8.04-8.05		
5.04	mdh5,mdh4	1.10									
5.04	uaz111(gfu)					5.06					
5.04	mpik33						6.01				
5.04	agr235	1.10									
5.04	pcr	1.09	2.00					7.01-7.02			
5.04	umc66	1.12			4.05-4.06						
5.04	umc166	1.00									
5.04	rpa5	1.10									
5.04	agr238	1.10									
5.04	bnl5.02	1.09									
5.04-5.05	npi116,npi203				4.09-4.10						
5.04-5.05	npi384,npi383				4.03-4.04						
5.05	ncr(B70)				4.10						
5.05	uaz299(ptk)						6.01				
5.05	bnl14.28									9.07-9.08	
5.05	csu36(rpl19)			3.10	4.05-4.08						
5.06	npi53,npi302						6.00				
5.06	npi424,npi450				4.08-4.09						
5.06	agr248				4.10						
5.06	agr100?				4.10						
5.06	uaz111(gfu)					5.06					
5.06	uaz285(tpase)	1.08							8.04-8.05		
5.06-5.07	amp	1.09									
5.07	als				4.03-4.04						
5.07	uaz70(gfu)				4.01						
5.07	uaz295(orf)		2.05	3.03							
5.07	uaz279(gfu)				4.11						
5.07	bnl10.12								8.08		
5.07	gapd				4.03-4.04						10.05-10.06
5.07	uaz248(his3)	1.04									
5.07	uaz164(gfu)			3.03, 3.08					8.06		
5.07	uaz138(gfu)	1.09							8.07-8.08		
5.07	umc156				4.05-4.06						
5.07-5.08	umc126				4.05-4.06						
5.07-5.08	ucsd72	1.08		3.06							10.00
5.08-5.09	npi458,npi461										10.08
5.09	npi115,npi312,npi329		2.00								10.00
5.09	uaz215(odo)					5.02-5.03					
5.09	uaz144(atp)				4.05-4.07						
5.09-5.10	npi362,npi293,npi322	1.00								9.04	
5.09-5.10	npi313,npi316							7.01-7.02			
5.10	npi253				4.08		6.04-6.05			9.01	
5.10	bnl9.07									9.01	
5.10	lw3,lw4?				4.05						
5.10-5.11	umc68					5.00					
5.11-5.12	npi(a1)		2.08	3.09				7.02-7.03	8.06-8.07		
5.11-5.12	uaz345(zta)					5.02-5.03					
5.11-5.12	agr563				4.08						
5.11-5.12	uaz240(nbp)	1.15					6.09				
5.11-5.12	tpi		2.04, 2.08	3.03				7.04-7.05	8.02-8.03		
5.11-5.12	npi363,npi292				4.08						
5.11-5.12	uaz71(gfu)				4.05-4.06						

5.12-5.13	umc104				4.07						
5.13	uaz268(gfu)	1.11						7.01-7.02			
6.00	npi2,npi1								8.08-8.09		
6.00	npi101,npi103								8.06		
6.00	npi7,npi6				4.03-4.04						
6.00	npi245,npi340,npi246	1.09	2.06								
6.00	uaz18(gfu)	1.09	2.06	3.07							
6.00	uaz310(gbp)				4.03-4.04						
6.01	npi387,npi388,npi441	1.09						7.01			
6.01	mpik11				4.03-4.04, 4.04-4.05					9.03	
6.01	pog1			3.09					8.06		
6.01	uaz299(ptk)					5.05					
6.01	uaz300(mtl)			3.04							
6.01	uaz269(kri)		2.07				6.10		8.01		
6.01	uaz258(gfu)								8.04-8.05		
6.01	csu71(cab)	1.10									
6.01	csu150(rpo)					5.02-5.03					
6.01	pgd1,pgd2			3.05							
6.01	npi594				4.04-4.05						
6.01	uaz23(gfu)		2.09								
6.01	uaz233(act)							7.04	8.04-8.05		
6.01	mpik33					5.04					
6.01	uaz197(ptk)						6.01				
6.01	uaz237(prc)									9.02	
6.02-6.03	bnl10.42		2.05								
6.02-6.03	uaz265(sbe1)		2.06								
6.02-6.03	oec		2.05		4.00, 4.05-4.06	5.02-5.03		7.03			
6.02-6.03	npi98,npi225	1.12								9.09	10.04-10.05
6.02-6.03	uaz106(gfu)		2.04								
6.02-6.03	uaz220(eif)						6.06	7.04	8.00		
6.02-6.03	uaz161(eif)			3L	4.10					9.04	
6.02-6.03	npi361	1.16	2.03, 2.10-2.11		4.00, 4.12		6.02-6.03	7.01-7.02	8.04		10.05-10.06
6.02-6.03	umc70									9.01	
6.02-6.03	umc113									9.01-9.02	
6.04-6.05	bz1									9.01-9.02	
6.04-6.05	pl,c1									9.01-9.02	
6.04-6.05	npi253				4.08	5.10				9.01	
6.04-6.05	npi224	1.09		3.04			6.05	7.01-7.02	8.05, 8.09		
6.04-6.05	agr118									9.01-9.02	
6.04-6.05	pep-c									9.04	
6.06	dzs23,dzs10									9.04	
6.06	uaz244(prh)								8.03		
6.06	uaz297(rpl17)		2.07						8.03		
6.06	uaz220(eif)						6.02-6.03	7.04	8.00		
6.06	zag1,zag2			3.05							
6.06	ucsd78				4.05-4.06				8.03		
6.06	ucsd64				4.04-4.05, 4.05-4.06	5.02-5.03			8.03		10.00
6.06	sod3								8.018.04		
6.06	uaz121(gfu)								8.03		
6.06	me2,me1			3.02-3.03							
6.07	pdk								8.05		
6.07	umc137		2.09								
6.07	bnl5.47								8.04		
6.07	bnl8.06								8.04		
6.07	umc150		2.09						8.04		
6.07	npi205,npi301,npi332,npi102,npi62	1.07						7L	8L		
6.07-6.08	umc38								8.06		
6.08	umc160								8.05		
6.08	bnl8.08	1.13		3.05	4.07				8.01	9.05-9.06	

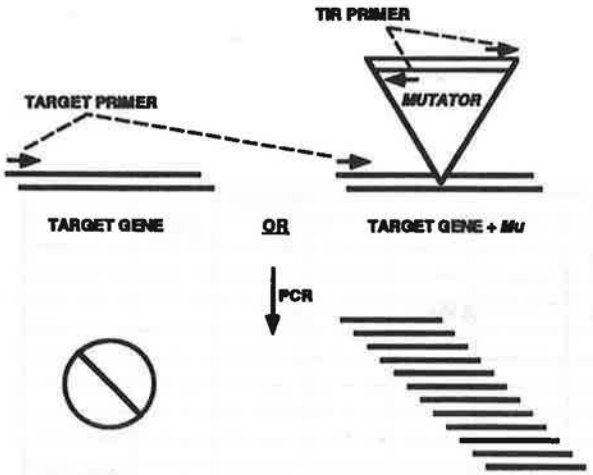
6.08	koln1								8.05		
6.08	uaz19(gfu)	1.09		3.04				7.01-7.02			
6.08	uaz333(gfu)								8.04		
6.08	uaz251(rps11)		2.02-2.03	3.07					8.02-8.03		10.05-10.07
6.08	uaz243(atp)			3.08					8.02-8.03		
6.10	idh2,idh1								8.07		
6.10	npi597										10.05-10.06
6.10	mdh2,mdh3,mdh1			3.08					8.03-8.04		
6.10	umc133				4.07-4.08						
6.10-6.11	umc134		2.05								
6.10-6.11	bnl5.62	1.01	2.03							9.04	
6.10-6.11	uaz123(gfu)				4.08			7.03			
6.10-6.11	uaz269(kri)		2.07				6.01		8.01		
6.10-6.11	uaz230(gfu)				4.05-4.06						
6.10-6.11	adpg	1.07	2.08	3.09	4.03-4.04				8.04		10.02
6.11	npi9,npi206								8.08		
6.11	npi260,npi261								8.03-8.04		
6.11	uaz240(nbp)	1.15				5.11-5.12					
7.00	ucsd106		2.11								
7.00	php20581		2.11								
7.00-7.01	npi400		2.11								
7.00-7.01	npi388,npi387,npi441	1.09					6.01				
7.01-7.02	npi361,npi294,npi359	1.16	2.03, 2.10-2.11		4.00, 4.11-4.12		6.02-6.03	7.01-7.02	8.04		10.05-10.06
7.01-7.02	mpik4		2.04								
7.01-7.02	uaz20(gfu)	1.09									
7.01-7.02	uaz27(gfu)		2.04								
7.01-7.02	uaz19(gfu)	1.09		3.04			6.08				
7.01-7.02	ucsd141		2.06								
7.01-7.02	invertase,mn1		2.05								
7.01-7.02	uaz344(est)		2.05								
7.01-7.02	uaz268(gfu)	1.11				5.13					
7.01-7.02	npi30,npi367		2.00		4.04-4.05						
7.01-7.02	zpl2				4.03-4.04						
7.01-7.02	pcr	1.09	2.00			5.04					
7.01-7.02	npi44,npi49		2.06								
7.01-7.02	bnl17.13				4.01, 4.03-4.04						
7.01-7.02	uaz68(gfu)				4.01						
7.01-7.02	ask1,ask2		2.07								
7.01-7.02	csu7(gfu)	1.15									
7.01-7.02	csu4(gfu)	2L									
7.01-7.02	orp1,orp2				4.03-4.04						10.05-10.06
7.01-7.02	ucsd81			3.05							10.04
7.01-7.02	npi316,npi313					5.09-5.10					
7.01-7.02	npi(a1)		2.08	3.09		5.11-5.12			8.06-8.07		
7.01-7.02	npi277,npi405		2.07								
7.01-7.02	npi47,npi123		2.08								
7.01-7.02	php20690							7.05			
7.01-7.02	npi224	1.09		3.04			6.05		8.05, 8.09		
7.01-7.02	g-zein		2.07								
7.03	npi394,npi395				4.04-4.05						
7.03	oec		2.05		4.00, 4.05-4.06	5.02-5.03	6.02-6.03				
7.03	agr267		2.08								
7.03	alr		2.08								
7.03	npi216,npi221		2.08								
7.03	umc5		2.07-2.08								
7.03	umc98		2.08								
7.03	agr265		2.00								
7.03-7.04	uaz123(gfu)				4.08		6.10				
7.04	agr333		2.08								
7.04	agr111		2.09								

7.04	umc125		2.09								
7.04	php20569		2.09								
7.04	uaz31(gfu)		2.08							9.07-9.08	
7.04	uaz28(gfu)		2.06								
7.04	npi5,npi4		2.07								
7.04	npi440,npi439	1.03									
7.04-7.05	bnl5.21		2.09								
7.04-7.05	bnl5.61		2.09								
7.04-7.05	tpi		2.04, 2.08	3.03		5.11-5.12			8.02-8.03, 8.09		
7.04-7.05	npi413		2.09								
7.04-7.05	uaz315(tua)		2.09								
7.04-7.05	uaz233(act)						6.01		8.04-8.05		
7.04-7.05	uaz220(elf)						6.02-6.03, 6.06		8.00		
7.04-7.05	uaz117(gfu)			3.10							10.04
7.05	npi335,npi358,npi273,npi109	1.03	2.06	3.04							
7.05	npi398			3.03							
7.05	cab			3.09					8.05		
7.05	npi431,npi300				4.08					9.04	
7.05	bnl8.21		2.09								
7.05	B32								8.05		
7.05	npi59,npi210		2.09								
7.05	uaz241(orf)		2.09								
7.05	uaz119(rps6)								8.07	9.04	
7.05	php20690							7.02			
7.05	npi35,npi46		2.09								
7.05	npi113,npi118		2.09								
7.05	npi278		2.09								
7.06	npi45,npi337		2.08								
8.01	npi220			3.03							
8.01	npi114,npi202			3.04							
8.01	npi222									9.04	
8.01	bnl8.08	1.13		3.05	4.07		6.08			9.05-9.06	
8.01	bnl13.05			3.04					8.03-8.04		
8.01	sod3						6.06		8.04		
8.01	npi218,npi219			3.03							
8.01	bnl8.10	1.10									
8.02	umc103			3.08							
8.02-8.03	tpi		2.04, 2.08	3.03		5.11-5.12		7.04-7.05	8.09		
8.03	npi276,npi364			3.03							
8.03	bif1,bif2			3.07							
8.03	ucsd78				4.04-4.06		6.06				
8.03	ucsd64				4.04-4.05, 4.05-4.06	5.02-5.03	6.06				10.00
8.03	mpik35			3.04							
8.03	mpik12				4.03-4.04					9.02	10.00
8.03-8.04	bnl13.05			3.04					8.01		
8.03-8.04	mdh1,mdh3,mdh2			3.08			6.10				
8.04	agr116			3.02							
8.04	umc32			3.00-3.01							
8.04	bnl5.47						6.07				
8.04	bnl8.06						6.07				
8.04	bnl1.45		2.04								
8.04	umc150		2.09				6.07				
8.04	npi206,npi9						6.11				
8.04	npi260,npi261						6.11				
8.04	ucsd61	1.07	2.11	3.02	4.05-4.06					9.03, 9.09	
8.04	sod3						6.06		8.01		
8.04	uaz25(mcp)		2.06			5.02-5.03					
8.04	uaz244(prh)						6.06				
8.04	uaz269(kri)		2.07				6.01, 6.10				
8.04	uaz297(rpl17)		2.07				6.06				

8.04	uaz249(rps27a)	1.08		3.04					8.10		
8.04	uaz121(gfu)						6.06				
8.04	npi361,npi294,npi359	1.16	2.03, 2.10-2.11		4.00, 4.11-4.12		6.02-6.03	7.01-7.02			10.05-10.06
8.04	uaz251(rps11)		2.02-2.03	3.07			6.08				10.05-10.07
8.04	uaz243(atp)			3.08			6.08				
8.04	uaz252(ptk)				4.07-4.08						
8.04	uaz333(gfu)						6.08				
8.04	niu					5.03					
8.04	adpg	1.07	2.08	3.09	4.03-4.04		6.10-6.11				10.02
8.04	uaz233(act)						6.01	7.04			
8.04	bnl17.08										10.08
8.04-8.05	umc186					5.04					
8.04-8.05	npi372,npi371,npi422				4.08-4.09						10.00
8.04-8.05	agr569			3.09							
8.04-8.05	uaz285(tpase)	1.08				5.06					
8.05	B32							7.05			
8.05	npi224	1.09		3.04			6.05	7.01-7.02	8.09		
8.05	pdk						6.07				
8.05	uaz258(gfu)						6.01				
8.05	koln2									9.02	
8.05	cab			3.09				7.05			
8.05	umc160						6.08				
8.05	koln1						6.08				
8.05	sdw1,sdw2?			3.06							
8.05	umc165			3.06							
8.05	agr514			3.06							
8.06	agr50			3.06							
8.06	lg4,lg3?			3.04							
8.06	agr274			3.06							
8.06	agr144			3.07							
8.06	npi101,npi103						6.00				
8.06	npi1,npi2						6.00				
8.06	pog1			3.09			6.01				
8.06	uaz164(gfu)			3.03, 3.08		5.07					
8.06	bnl12.30			3.09							
8.06	umc38						6.07-6.08				
8.06-8.07	npi(a1)		2.08	3.09		5.11-5.12		7.02-7.03			
8.07	uaz138(gfu)	1.09				5.07					
8.07	idh1,idh2						6.10				
8.07-8.08	agr184			3.06							
8.07-8.08	uaz176(gfu)			3.07							
8.08	npi3,npi201			3.08							
8.08	uaz119(rps6)							7.05		9.04	
8.08	npi426,npi425			3.10							
8.08	bnl10.24			3.06							
8.08	umc53		2.02-2.03								
8.08-8.09	npi69,npi70			3.04							
8.08-8.09	bnl10.12					5.07					
8.08-8.09	npi204,npi108			3.05							
8.08-8.09	npi328			3.07							
8.09	umc164	1.00									
8.09	npi224	1.09		3.04			6.05	7.01-7.02	8.05		
8.09	uaz174(gfu)				4L						
8.09	tpi		2.04, 2.08	3.03		5.11-5.12		7.04-7.05	8.02-8.03		
8.10	obf3			3.07							
8.10-8.11	uaz279(rps27a)	1.08		3.04					8.04		
8.11	umc3			3.07							
8.11	umc4		2.08-2.09								
8.11	umc39			3.07							
8.11	npi212			3.07							
9.01	umc70						6.02-6.03			9.01	
9.01	bnl9.07					5.10					

9.01	koln2								8.05		
9.01	npi253,npi325,npi324,npi343				4.08	5.10	6.04-6.05				10.00
9.01	agr118						6.05				
9.01-9.02	umc113						6.02-6.03				
9.02	c1,pl						6.04-6.05				
9.02	uaz237(prc)						6.01				
9.02	sh1,css									9.04-9.06	
9.02	bz1						6.04-6.05				
9.02	agr255										10.08
9.02	mpik12				4.03-4.04				8.03		10.00
9.03	dzs10,dzs23						6.06				
9.03	mpik11				4.03-4.04, 4.04-4.05		6.01				
9.03	ucsd61	1.07	2.11	3.02	4.05-4.06				8.04	9.09	
9.04	umc82	1.08									
9.04	obf2,obf1?	1.06									
9.05-9.06	uaz161(eif)			3L	4.10		6.02-6.03				
9.05-9.06	npi416,npi418						6.00				
9.05-9.06	npi300,npi431				4.08			7.05			
9.05-9.06	mgs2				4.08-4.09						
9.05-9.06	uaz119(rps6)							7.05	8.08		
9.05-9.06	umc194B(gpr)	1.02									
9.05-9.06	npi222								8.01		
9.05-9.06	uaz236(strs)		2.06								
9.05-9.06	uaz304(hon)	1.04									
9.05-9.06	php20075										10.01
9.05-9.06	bnl7.24					5.01					
9.05-9.06	css,sh1									9.02	
9.05-9.06	sod4	1.04									
9.05-9.06	hm2,hm1?	1.09									
9.05-9.06	uaz266(nad)	1.04									
9.05-9.06	phyB	1.04									
9.05-9.06	uaz264(hsp)	1.03									
9.05-9.06	bnl5.62	1.01	2.03				6.10-6.11				
9.05-9.06	npi293,npi322,npi362	1.00				5.09-5.10					
9.06	umc140	1.11									
9.06	npi427,npi428	1.04									
9.06	bnl8.08	1.13		3.05	4.07		6.08		8.01		
9.06	koln9	1.03									
9.06	agr153	1.04									
9.06	agr92	1.03-1.04									
9.06	npi439,npi440							7.04			
9.06	bnl7.21	1.05									
9.06	npi14,npi89			3.03-3.04							
9.06-9.07	npi403,npi404	1.02									
9.06-9.07	agr294	1.01									
9.07-9.08	npi209,npi93	1.02									
9.07-9.08	bnl14.28					5.05					
9.07-9.08	uaz14(gfu)	1.08									
9.08-9.09	npi97,npi99	1.01									
9.09	ucsd61	1.07	2.11	3.02	4.05-4.06				8.04	9.03	
9.09	uaz31(gfu)		2.08					7.04			
9.09	umc94	1.01									
9.09	npi98,npi225	1.12					6.02-6.03				10.04-10.05
9.09	csh(cdc2)				4.12						
10.00	agr43			3.10							
10.00	ucsd72	1.08		3.06		5.07-5.08					
10.00	npi366,npi208				4.08						
10.00	ucsd64				4.04-4.05, 4.05-4.06	5.02-5.03	6.06		8.03		
10.00	mpik12				4.03-4.04				8.03	9.02	
10.00	uaz309(cht)			3.10							
10.00	bnl10.17				4.07						

10.00	npi422,npi372,npi371				4.08-4.09				8.04-8.05		
10.00-10.01	PIO20075									9.05-9.06	
10.01	Cin4		2.04		4.05-4.06						
10.01	rp4,rp1,rp5,rp6,rp9				4.00						
10.02	adpg	1.07	2.08	3.09	4.03-4.04		6.10-6.11		8.04		
10.02	uaz21(gfu)	1.05, 1.12	2.00								
10.04	umc18			3.05							
10.04	npi417		2.00								
10.04	npi378,npi379			3.04							
10.04	uaz339(glu)			3.04							
10.04	uaz340(glu)										10.05-10.06
10.04	ucsd81			3.05				7.01-7.02			
10.04	npi445										10.00
10.04	uaz24(gfu)		2.01								
10.04	uaz228(his2b)	1.09	2.07		4.05-4.06						
10.04	csu6(gfu)		2.05								
10.04	npi327,npi381	1.00									
10.04	npi597						6.10				
10.05-10.06	orp1,orp2				4.03-4.04			7.02			
10.05-10.06	gf14-12		2.05								
10.05-10.06	uaz117(gfu)			3.10							
10.05-10.06	uaz175(gfu)				4.08						
10.05-10.06	uaz76(gfu)					5.00					
10.05-10.06	npi98,npi225	1.12					6.02-6.03			9.09	
10.05-10.06	npi22,npi365		2.00								
10.05-10.06	npi437,npi305,npi307					5.02-5.03	6.00				
10.05-10.06	uaz251(rps11)		2.02-2.03	3.07			6.08		8.02-8.03		
10.05-10.06	uaz340(glu)										10.04
10.05-10.06	npi361	1.16	2.03, 2.10		4.00, 4.12		6.02-6.03	7.01-7.02	8.04		10.05-10.06
10.05-10.06	gapd				4.03-4.04	5.07					
10.05-10.06	npi269		2.04								
10.07-10.08	umc44		2.04								
10.08	r,b		2.04								
10.08	bnl17.08								8.04		
10.08	npi461,npi458					5.08-5.09					
10.08	npi436,npi287		2.04								
10.08	agr168		2.03								
10.08	agr216		2.06								
10.08	agr167		2.04								
10.08	agr255									9.02	
10.08	agr113		2.04								
10.08	bnl7.49										10.00
10.08	uaz318(nbp)		2.02-2.03								
10.08	npi421		2.02-2.03								
10.08	npi290		2.02-2.03								
10.08	uaz113(aaa)		2.02-2.03								
10.08	npi254		2.01								
10.08	npi577		2.01-2.02								
10.08	npi321,npi320		2.02								
10.08	npi350		2.01								
10.08	php20568		2.00								
10.08	npi604				4.01						



which a *Mu* element has inserted in or near the gene of interest (see illustration). Prospective insertion alleles are identified by successive rounds of PCR/DNA dot blot hybridization (using a probe specific for the target gene); first on DNA pools, subsequently on DNA from individuals. Once individuals are identified, kernels from the corresponding F2 seed packet can be allocated for phenotypic analysis of specific mutant alleles. To illustrate the utility of such a system, brief examples of its application are useful:

1) *To knock out genes.* Though many DNA sequences for genes are known (be it from random sequencing, cloning by homology, reverse genetics from peptide sequence, or transposon tagging), mutations in specific genes can be difficult to isolate (e.g. lethal when homozygous, phenotype unknown, masked by a redundant function). From this collection, which has proven to be very rich in mutations, *Mu* insertion alleles can be selected for any gene *whose sequence is known.* Though not all insertion alleles will be mutagenic, due to the position or orientation of the *Mu* insertion, precise knowledge of the gene's structure can be used to selectively target exons, or a particular region of the sequence, in order to maximize the potential for recovering mutagenic insertions. Phenotypic analysis and the propagation of insertion alleles proceeds from the F_2 seed, which segregates the mutant and wild-type alleles. Lethal mutations can be discerned by the inability to recover homozygotes, yet heterozygotes can be used to propagate the allele. Gene families or redundant functions can be targeted independently, and insertion alleles combined genetically to observe the net phenotypic effect of stacked mutations. Tissue-specific or conditionally expressed genes, once cloned, can be selected for disruption, facilitating the mutational analysis of specific processes in physiology and development.

2) *A rapid means to confirm the identity of a cloned gene.* This reverse genetics technology was used to confirm the identity of a candidate clone for the maize *an1* gene (Bensen et al., Plant Cell 7; in press). The original clone was isolated by traditional transposon tagging: a screen for dwarf mutants in *Mu* F_2 families, co-segregation analysis of *Mu* elements with the mutant phenotype, isolation of flanking DNA, recovery of a wild-type clone from a cDNA and/or genomic library. With only a single transposon-tagged allele for this gene, the necessary confirmation (that the cloned sequence was indeed *an1*) was potentially arduous. However, a reverse genetics screen, using PCR primers specific for the candidate clone and the *Mu*-TIR primer, uncovered several new *Mu* alleles of the cloned sequence. Analysis of the F_2 progeny for each new allele revealed the expected dwarf phenotype. The clone was thus confirmed as *an1*; the technology made this type of confirmation possible in weeks, rather than the months or even years necessary to tag additional mutants, or to screen for revertant progeny from the existing mutant.

The main point to be made about this technology is that it is a powerful tool for the study of gene function in plants, specifically maize. Researchers are invited to learn more about this tool, including the opportunity to make use of it. Of greatest importance are the project-specific requirements that must be met before a mutant request is considered formal. This includes development of control alleles and thorough testing of PCR primers. Complex projects, such as those concerning mutagenesis of gene families, naturally require more rigorous planning. For more information, please contact Bob Meeley at the project's e-mail address (TUS@phibred.com).

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Analysis of a palindromic DNA sequence motif

--John C. Prostko and Tom Quayle

A recent analysis of the structure of zein genes and their flanking regions uncovered an unusual cluster of palindromic DNA sequences located at approximately two kilobase pairs upstream of a zein gene. Four nearly identical copies of this structure, each a GC-rich 17- to 21-base pair perfect palindrome, are contained in a stretch of only 142 base pairs. These four maize palindromic units (MPUs) are separated from each other by precise integral turns of the B-form DNA helix. Additionally, they are relatively abundant in the genome of maize, as shown by Southern blot analysis (Quayle et al. Gene 80:249-258, 1989) Furthermore, the MPUs are specifically bound by nuclear proteins in a tissue- and developmental-specific manner (Quayle, unpublished results).

A more accurate copy number determination and cloning of additional MPUs has been accomplished using a primary genomic library of maize in lambda phage EMBL3. We have recently screened this library via DNA-DNA hybridization on nitrocellulose membrane plaque lifts, and have isolated 32 MPU-containing phages. The minimum copy number for the MPUs was estimated at 30,000, based on the ratio of hybridization positive plaques to the total number of plaques screened, the average insert size, and the haploid genome of *Zea mays*, approximately 2.7×10^9 bp (K. Arumuganathan et al., Plant Mol. Bio. Rep. 9:208-218, 1991).

We have cloned and sequenced two new MPUs from maize and a recent report to GenBank shows a single MPU at approximately 2500 base pairs upstream from the *Cat3* (catalase) gene (also of maize) (M.L. Abler and J.G. Scandalios, GenBank sequence entry MZECAT3GN). Interestingly, this is at about the same upstream location of the MPUs found in the zein gene pMS2, and both genes are highly regulated, being expressed only in germinating aleurone and developing endosperm tissues respectively. We have recently been informed of a cluster of four MPUs located about 3,000 bp upstream of the *Knotted-1* gene of maize (J. Mathern and S. Hake,

```
CLONE-D-1
GAATTCACCGGCGCGTACGGCTATAATTCACCGGACT  GTCCGGTGTGCACCGGATTGTCCGGTGAGCCAACGG
CLONE D-2                              -------------------------
TGTGGCCGAGCCAACGGCTAGAAGGGGGCACCGGACT  GTCCGGTGTGCACCGGACATGTCCGGCCAACGGCT
CAT3                                   --------------------
GAGAGTGAACAGTGCATGTTCGGTGCGGCACCGGACT  GTCCGGTGCCCCAAGATGTCAGAGCTCCAACGATCG
PMS2-4                                 ----------------------
CGTTGGCGCACCGGACACTGTCCGGTGCCACCGGACA  GTCCGGTGCCCCCTCCCGACCGTTGGCTCAGCCACG
PMS2-1                                 ------------------
GGCAATTCTTGCCTTCTGTCGCCTGGCGCACCGGACA  GTCCGGTGCACCACCGGACACTGTCCGGTGCGGATT
PMS2-2                                 ---------  ---------
GCCTGGCGCACCGGACAGTCCGGTGCACCACCGGACACTGTCCGGTGCGGATTTCCTTCCTTATTTGGCGAAGCG
PMS2-3                                 --------------------
ACCGTTGGCAGCCTTGGAGCCGTTGGCGCACCGGACACTGTCCGGTGCCACCGGACAGTCCGGTGCCCCCTCCCG
```

Figure 1. Comparison of the 7 known perfect MPUs. The palindromes are underlined. The center base of each palindrome is double underlined. The dashed lines between the palindromes represent perfect homology between corresponding MPUs.

```
New Seq.   PECFWSEAVNTRCHTINRVYLHRLLKKTSYELLTGXXPNVSYFRVFGSKCYILVKKGRNSKFAPKAVEGFLLGYDSNTKAYRVSOSSGW
           *  W *A  T  *  NR        *    * G  P     RVFG  **         K  *        GY    K YR
Z.m. rt    PATYWAEALHTATHLLNRLPSKAVSHPTPHFALYGTTPSYDHLRVFGCACYPNTSATAPHKLSPRSTRCLFLGYSPDHKGYRCLDLTSH
           *  W* A *T*  * NR *              *G  P    *RVFG*  *         K  * *    * GY *  K YR *
V.c. rt    PSRGWALAMHTANYLRNRSPVHGLKGTPFERF-YGQKPDVSDLRVFGCVAYIRVPDHQRRKIDPVSMKGVFVGYEPESKAYRVLMPDGR
           W* A  T* ***NR*    *             G  P *  *RVFG  *         K            GY    K YR      *
D.m. rtro  GEALWAEAINTAVYLRNRSTSRALQSKTPMEEWTGKIPAVSHLRVFGAIAVALDKGVHKGKFESKGKEYRMIGYSIAAKGYRLFDKEKR
            * W *A *T     NR     * **  *  *G??P **  RVFG     * *     K*  *  *    GY    K YR
New Seq.   PECFWSEAVNTRCHTINRVYLHRLLKKTSYELLTGXXPNVSYFRVFGSKCYILVKKGRNSKFAPKAVEGFLLGYDSNTKAYRVSOSSGW
           *  W  A  T     NR    * * *        G  P **  RVFG    ** *      K  *     *  GY    K*YR*     *
V.c. rt    PSRGWALAMHTANYLRNRSPVHGLKGTPFERF-YGQKPDVSDLRVFGCVAYIRVPDHQRRKIDPVSMKGVFVGYEPESKAYRVLMPDGR

D.m. rtro  GEALWAEAINTAVYLRNRSTSRALQSKTPMEEWTGKIPAVSHLRVFGAIAVALDKGVHKGKFESKGKEYRMIGYSIAAKGYRLFDKEKR
           W**A  T*  * NR  * *    **      G  P   **RVFG              K          GY*   K*YR  *
Z.m. rt    PATYWAEALHTATHLLNRLPSKAVSHPTPHFALYGTTPSYDHLRVFGCACYPNTSATAPHKLSPRSTRCLFLGYSPDHKGYRCLDLTSH
```

Percent identity between the four sequences

New Seq.	to	D.m.	36%	Z.m.	to	V.c.	36%
New Seq.	to	V.c.	35%	Z.m.	to	D.m.	34%
New Seq.	to	Z.m.	28%	D.m.	to	V.c.	31%

Figure 2. Comparison of 4 coding regions from *Zea mays*, *Drosophila*, and *Volvox*. 17 of 89 amino acids (19%) are identical between the sequences.

personal communication). Comparison of the first seven known perfect MPUs shows an almost perfect identity for the palindrome region (Figure 1), but not for their flanking regions, indicating that the MPU itself is the repetitive element. The MPU region of the *Knotted-1* gene, however, has near perfect identity to the MPU region of pMS2.

Preliminary sequence data obtained from partial sequencing of one of the MPU-containing lambda clones reveals considerable homology to several potential reverse transcriptases. Figure 2 compares our sequence to reverse transcriptases from *Zea mays* (Z.m. rt) (D.F. Voytas, et al., PIR Release 41.0 accession number S27768), Drosophila (D.m. rtro) (F. Fourcade-Peronnet, et al., PIR Release 41.0 accession number S00954), and Volvox (W.c. rt) (A. Lindauer, et al., PIR Release 41.0 accession number S32437). Note that the amino acids conserved between the three reverse transcriptases are also conserved in our recently sequenced open reading frame. Further work is currently in progress to identify additional new genes adjacent to MPUs, and to indicate the function of this new DNA motif.

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Influence of the *Y11* and *Y12* genes on carotenoid biosynthesis

--Diane Janick-Buckner and Brent Buckner

The yellow color of maize endosperm is due to the synthesis and accumulation of carotenoids. Several genes have been identified that are involved in the carotenoid biosynthetic pathway. Plants that are homozygous for the recessive allele of either *y11* or *y12* exhibit lemon yellow kernels as opposed to normal yellow kernels (MNL 61:96, 1987). The influence of the *Y11* and *Y12* genes on the carotenoid biosynthetic pathway in endosperm was investigated in this study.

Carotenoids from the endosperm of sibling lemon yellow and yellow kernels were extracted and analyzed from four separate ears in triplicate. Kernels were obtained from ears produced by self pollinating plants that were heterozygous for the recessive allele of either *y11* or *y12*. The carotenoids in the extract were then separated and quantified by using high performance liquid chromatography (HPLC) while monitoring at 450 nm. The distribution of carotenoids was similar in all endosperm extracts analyzed. The major carotenoids present in both yellow and lemon yellow endosperm were lutein/zeaxanthin, a xanthophyll monoester, alpha-carotene, ß-carotene, alpha-cryptoxanthin and ß-cryptoxanthin. However, the quantity of some carotenoids was significantly reduced in lemon yellow endosperm in both homozygous recessive *y11* and *y12* endosperm. Xanthophylls were affected to a greater extent than were carotenes.

When carotenoid extract from homozygous recessive *y12* endosperm was analyzed on an HPLC equipped with a photodiode array detector, it was evident that a large quantity of zeta-carotene was present. Only a very small quantity of this

carotenoid was present in carotenoid extract prepared from sibling yellow endosperm. Thus, it appears that homozygous recessive *y12* endosperm has a reduced ability to convert zeta-carotene to neurosporene, and consequently accumulates zeta-carotene. We have also observed that under field conditions the leaves of homozygous recessive *y12* plants display a diurnal pattern, suggesting that this allele could be temperature sensitive and also influence carotenoid biosynthesis in the leaf. In addition, homozygous recessive *y12* kernels exhibit a low frequency of vivipary, suggesting that this gene influences kernel abscisic acid levels.

Allele-specific length polymorphisms associated with microsatellite repeat variation within the *Y1* gene

--Tara L. Phelps and Brent Buckner

Microsatellites are tandemly repeated short nucleotide sequences found in all eukaryotic and some prokaryotic genomes (Tautz et al., Nature 22:652- 656, 1986). Allele-specific length polymorphisms of microsatellites are known to exist in maize (Calderon-Urrea and Dellaporta, MNL 68:70, 1994). In barley the number of alleles of ribulose-1,5-bisphosphate carboxylase activase distinguished by microsatellite repeat variation far exceeds the number detected by analysis of other molecular markers such as chloroplast DNA, ribosomal DNA or RFLPs (Maroof et al., Proc. Natl. Acad. Sci. USA 91:5466-5470, 1994). Although little is known about the function of microsatellites within or near plant genes, their prevalence, high degree of repeat number variability and Mendelian inheritance have made them useful in genome mapping and as potential markers for plant breeding studies.

The *Y1* gene codes for phytoene synthase, an enzyme that condenses two geranyl-geranyl pyrophosphate molecules into one molecule of phytoene during the biosynthesis of carotenoids (Buckner et al., MNL 67:65, 1993). The dominant allele of *Y1* cloned by Buckner et al. (Plant Cell 2:867-876, 1990) has been sequenced (unpublished data) and shown to contain a CCA trinucleotide repeated eleven times upstream of the coding sequence. However, it is not known if this microsatellite is present in other alleles of *Y1*. The purpose of this study was to determine if the CCA microsatellite exists in other alleles of *Y1*, if it is polymorphic in length and if the number of trinucleotide repeats correlates with the expression of the *Y1* gene.

DNA was isolated from individual etiolated shoots grown for three to five days at 30C on germination paper saturated with 1 mM CaCl2. PCR reactions were performed in 50 µl with approximately 1 µg of genomic maize DNA. The reaction mix contained 1x PCR buffer (10 mM Tris-HCl, pH 8.3, 50 mM KCl, 1.5 mM MgCl2 and 0.01% gelatin), 200 µM of each dNTP, and 1.25 units of *Taq* Polymerase (Perkin Elmer Co., Norwalk, CT). The reaction mix was incubated in a HybaidTM thermal cycler (National Labnet Co., Woodbridge, NJ) according to the following parameters: 94C for 1 min, 59C for 1 min, 72C for 30 sec. After 30 cycles the samples remained at 72C for 5 min. The PCR products were resolved on 2.5% MetaphorTM agarose (FMC Bio Products, Rockland, ME) gels made in 0.09 M Tris, 0.09 M boric acid and 2 mM EDTA. PCR products to be sequenced were excised from 1.2% Seakem Low-Gelling agarose (FMC Bio Products, Rockland, ME) gels made in 40 mM Tris-acetate and 1 mM EDTA, pH 7.8 and purified using Wizard™ PCR Preps DNA Purification System (Promega, Madison, WI). Approximately 12 ng of purified PCR products were sequenced using an Applied Biosystems Inc. (ABI, Forrest City, CA) *Taq* DyeDeoxy™ Terminator Cycle Sequencing Kit as described by the manufacturer.

Electrophoresis of the PCR products indicated that allele-specific length polymorphisms exist in this region of *Y1*. In order to determine if these length polymorphisms were due to the alleles having different numbers of the CCA repeat, the PCR product of each allele analyzed was gel purified and sequenced. These sequence data (Fig.1) show that the alleles studied contain between 5 and 11 CCA repeats.

In addition to the CCA trinucleotide repeat variation, we have also identified that a TCATC sequence directly 5' of the CCA repeat is duplicated in four of the nine alleles analyzed (Fig. 1A,B,E and F). Seven of the alleles also have the sequence CCATC directly 5' of the TCATC sequence (A,B,C,D,G,H, and I). It is interesting to note that the last CCA repeat is part of a second CCATC sequence. It appears that the CCATC and TCATC sequences are imperfect repeats of each other and that together they constitute a pentanucleotide repeat. Only two of the alleles analyzed (A and B) contained both the CCATC and TCATC sequence duplicated. These two alleles also contain the trinucleotide CTG repeated 33 bp 5' of the CCA repeat.

Many alleles of *Y1* have been previously described (Buckner et al., Plant Cell 2:867-876, 1990; Robertson and Anderson, J. Hered. 52:53-60, 1961), however, the DNA sequence has been determined for only one dominant allele of *Y1* (unpublished data). Therefore, the sequence variation that distinguishes recessive and dominant alleles of *Y1* has not been described. Trinucleotide repeat expansion has been correlated with disease-associated alleles in several human diseases (Stallings, Genomics 21:116-121,

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(A) TGCTGCTGTTCTGCTGACTGGTCTCACCATCTCATCTCATCCCACCACCACCACCACCACCACCACCACCATCTT
(B) TGCTGCTGTTCTGCTGACTGGTCTCACCATCTCATCTCATCCCACCACCACCACCACCACCACCACCACCATCTT
(C) TGCTG    TTCTGCTGACTGGTCTCACCATC      TCATCCCACCACCACCACCACCACCACCACCA    TCTT
(D) TGCTG    TTCTGCTGACTGGTCTCACCATC      TCATCCCACCACCACCACCACCACCA          TCTT
(E) TGCTG    TTCTGCTGACTGGTCTCA      TCATCTCATCCCACCACCACCACCTCCA             TCTT
(F) TGCTG    TTCTGCTGACTGGTCTCA      TCATCTCATCCCACCACCACCACCA                TCTT
(G) TGCTG    TTCTGCTGACTGGTCTCACCATC      TCATCCCACCACCACCACCACCACCACCA       TCTT
(H) TGCTG    TTCTGCTGACTGGTCTCACCATC      TCATCCCACCACCACCACCACCACCACCACCA    TCTT
(I) TGCTG    TTCTGCTGACTGGTCTCACCATC      TCATCCCACCACCACCACCACCACCACCACCA    TCTT
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Fig.1. Sequence comparison of the CCA microsatellite region of the *Y1* alleles analyzed. The CCA microsatellite repeat is double underlined and the pentanucleotide and other trinucleotide repeats are single underlined. Sequence A: allele cloned from the hybrid of inbred lines Q66 and Q67 (Plant Cell 2:867-876, 1990), B: allele in inbred line H99, C: allele in inbred line M14, D: allele in inbred line B73, E: *y1-lemon yellow* allele (received from G.F. Sprague), F: *Zea luxurians* allele, G: standard recessive allele of *Y1* (Plant Cell 2:867-876, 1990), H: *y1- wmut* allele and I: *y1- 8549* allele (J. Hered. 52:53-60).

1994). However, our study has shown that no correlation exists in the number of CCA repeats within this region of the *Y1* gene and the expression patterns of the different alleles of *Y1*. For example, the sequence of this region in the dominant allele of *Y1* from the inbred line M14 is identical to that of the recessive alleles *y1-wmut* and *y1-8549* (Fig. 1). In addition, this region in the dominant allele found in inbred line B73 is identical in sequence to the standard recessive allele of *Y1* (Fig. 1). The recessive teosinte and *y1-lemon yellow* alleles have 5 and 6 CCA repeats, respectively, and both lack the CCATC repeat which is 5' of the CCA repeat in all other alleles of *Y1* analyzed. It is not clear from these data whether the number of repeats influences the expression of these two alleles.

The teosinte allele analyzed in this study was derived from *Zea luxurians*. However, it should be noted that since maize and teosinte readily hybridize, this allele could be the result of a previous introgression of a maize *Y1* allele into this accession of *Zea luxurians*. Teosinte is thought by many to be the progenitor to maize (Beadle, Sci. Am. 242:112-119, 1980; Galinat, Corn and Corn Improvement, G.F. Sprague and J.W. Dudley, eds., pp 1- 31, 1988). Because the teosinte allele has five CCA repeats it is likely that the microsatellite repeat has expanded in teosinte and may have done so before the evolutionary event that gave rise to maize. Several species and varieties of teosinte in addition to *Zea luxurians* have been described. These forms of teosinte differ in their growth pattern (annual and perennial) and ploidy. Therefore, it would be interesting to determine the extent of CCA repeat number variation at this locus in these other teosintes, and to determine if any alleles of teosinte or maize contain a single copy of this CCA.

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Testing the marker line *wx sh* carrying a mutation phenotypically resembling *Dt* (9S) for the presence in its genome of regulatory elements *Uq* and *Mrh* following pollen treatment with exogenous DNA

--M.K Haritonova, N.V. Krivov and V.N. Lysikov

The occurrence in the aleurone layer of colored dots is most likely to be due to the interaction between the dominant *Dt*-like element and a receptor allele presumably located in *a1*. The number and size of the colored sectors in the aleurone layer in different kernels of the same ear indicate that mutation events occur with different frequencies and differ from one another chronologically (McClintock, 1951; Peterson, 1966).

In order to ascertain whether one of the already known regulatory elements has been integrated into the recipient line, plants grown from colorless kernels and yielding upon selfing only colorless progeny, and the parental marker line *wx sh*, were testcrossed with the tester lines *a-dt* (*Dt*), *a-ruq* (*Uq*), and *a-mrh* (*Mrh*) kindly supplied by Prof. P.A. Peterson. The crosses were made according to the following scheme:

1. M1 with colorless aleurone x tester carrying the receptor. F1 seeds with colorless aleurone, with dotted and with self-colored aleurone.

2. Parental marker line, *wx sh*, x tester carrying the receptor. F1 seeds with colorless aleurone, with dotted and with self-colored aleurone.

The progeny of the crosses were tested according to the procedure used by Peterson and Salamini (1986).

The M2 ears showed segregation for ear color, so it was possible to select families in which all the kernels had colorless aleurone. It was assumed that these families lacked one of the active elements of the system (either receptor or regulator). Crossing the plants grown from these seeds with tester lines carrying one of the receptors will make it possible to identify maize plants which have acquired active regulatory elements. Accordingly, the presence of a regulatory element will provide an indirect proof of one of the mutator systems being involved in the expression of the *Dt*-like pattern, and the basis for discussing the possibility of this regulatory element being integrated into the genome as a result of pollen treatment with exogenous DNA.

Testcrosses revealed the presence in all lines carrying the *Cg2* and *Cg2+* alleles of some regulatory elements of all the five mutator systems examined (Table 1). Therefore, the exogeneous DNA isolated from *zb*-8 lg g12 v4 Ch/+++++ Cg2* seedlings might have carried all five regulatory elements. The recipient (the marker line *wx sh*) only has *En/+* in some plants. Families carrying alleles for the *Dt*-like pattern exhibited activation of the *Mrh* and *Uq* regulatory elements (Table 2). At present, a testcross is underway to detect active genetic elements *Dt*, *Ac*, *En* and others.

Table 1. The results of testing lines carrying *Cg2* and *Cg2+* alleles, as well as MKO1 and *wx sh* lines for presence of the regulatory elements *Dt*, *Ac*, *En*, *Uq* and *Mrh*.

	Number of ears carrying regulatory elements														
Tested lines	*DtDt*	*Dt+*	*++*	*AcAc*	*Ac+*	*++*	*EnEn*	*En+*	*++*	*UqUq*	*Uq+*	*++*	*MrhMrh*	*Mrh+*	*++*
Cg2 gs bm2 gl	2						5			4			2		
*Cg2+/Cg2+**	1	3					2	1		4			3		
*Cg2+/Cg2+***			2							4		1	4		
Cg2m-s	2						2			7			6		
br-220*	4			5			8			9	1		7		
sr-220*	6					5	8			6			9		
ys-253*	9					6	9			9			9		
ys-143*	1	1	1		9		1	4		5			6		
MKO1							4			4			5		
wx sh			2			6		1	7			9			6

*(*Cg2+-- Cg2*) *j*
**(*Cg2+-- Cg2*) 5369

Table 2. The results of analysis of ears obtained in crosses of the *wx sh dt-*7* mutant with tester lines.

Cross Combinations	Aleurone color: colorless	dotted	self-colored
*wx sh dt-*7* x *a-ruq*	13	--	5
	13	6	--
	36	102	1
	68	57	-
*wx sh dt-*7* x *a-mrh*	48	1	-
	5	2	--
	7	-	--
	16	9	--
	6	2	--

The genetic behavior of unstable alleles controlling aleurone layer color following pollen treatment with exogenous DNA

--M.K Haritonova, N.V. Krivov and V.N. Lysikov

Instability of the heterochronous mutation *Cg2*, like that of any other locus within the maize genome, is most probably due to the activity of a genetic factor whose presence can be inferred from the *Cg2* -----> *Cg2+* and *Cg2+* ----> *Cg2* mutational changes (Krivov, MNL66:53-54, 1992). One possible way of detecting such a factor is to transfer it to a homogeneous genetic background by treating pollen of the appropriate recipient with exogenous DNA isolated from maize plants carrying the *Cg2* alleles. We suspected regulatory sequences to be integrated into pollen grains far more frequently as compared with structural genes, and expected the appearance of plants with the *Cg2* phenotype or of unstable mutations at some genomic locations.

Therefore, in order to test the assumption of a genetic factor controlling the *cg2* locus instability, pollen of the marker line *wx sh* was treated with exogenous DNA isolated from plants of genotype *zb*-8 lg g12 v4 Ch/+++++ Cg2* and applied to stigmata of the same marker line *wx sh*.

The pollen was collected in the morning, separately from each tassel, by shaking manually, and immediately mixed with a buffer solution containing donor DNA. For a better penetration of the exogenous DNA into pollen grains, the DNA preparation was diluted with a mixture of 0.3M sucrose and TE buffer consisting of 10ml 0.01M nhbc HCl, pH 8.0 and 1ml 0.001M EDTA. The DNA was diluted in this buffer until its concentration reached 100 mg/ml, and pollen was admixed to it with energetic stirring to produce a paste-like mixture. Then the resulting paste was applied to the stigmata of the previously prepared ears of the recipient plants. The control plants received pollen mixed with the buffer containing no DNA.

Grain content of the control ears was lower (out of 10 ears, only one set 6 seeds) than that of DNA-treated ears (8 ears out of 10 set 2, 3, 3, 5, 6, 7, 9 and 16 seeds). This supports the assumption (Mishra et al., 1987) that the introduction of high molecular weight DNA molecules into pollen is possible without significantly reducing pollen viability.

Pollen treatment in the M1 resulted in 8 plants forming ears, two of which carried mutant kernels. These were visually detectable as colored dots in the aleurone layer against the colorless background of the kernel. The dot color and pattern are very similar to those of *Dt* on 9S. The *Dt*:colorless ratio in these ears was 10 *Dt*:3 colorless and 5 *Dt*:2 colorless, respectively. In the M2, one more plant was detected carrying an identical mutation, as indicated by the fact that its ear exhibited the same pattern of colored dots, at a ratio of 7 *Dt*:16 colorless.

It can be seen from Table 1 that, on the whole, the progeny of five plants grown from *Dt* and colorless kernels largely consists of either *Dt* kernels (4/6, 4/7, 4/8) or colorless ones (1/1, 1/2, 2/2, 3/2, 3/4, 4/3, 4/4). However, upon selfing the plants grown from colorless kernels, a large proportion of the progeny of two of these (3/3 and 3/5) had *Dt* kernels, and upon selfing the plants grown from *Dt* kernels, significant deviations from the theoretically expected 3:1 ratio were observed, either due to an excess (2/3, 3/1, 3/9, 4/2, and 5/2) or deficit (3/6, 3/7, 4/1, 4/5, and 5/3) of colorless kernels.

Table 1. Individual tests for the progeny of *Dt*-like and colorless kernels in the M2.

Plant No.	Parental Kernel Color	Color distribution in progeny: Color-less	Dt	Self-colored	Total	x2
1/1	colorless	225			225	
1/2	*Dt*		6		6	
2/1	*Dt*		26	16	42	30.5
2/2	colorless	44			44	
2/3	*Dt*		58	114	172	7.0
3/1	*Dt*		21	6	27	40.1
3/2	colorless	10			10	
3/3	colorless	19	9		28	
3/4	colorless	14			14	
3/5	colorless	9	13		22	
3/6	*Dt*	2	37	1	40	8.5
3/7	*Dt*	2	30		32	6.0
3/8	*Dt*	3	13		16	0.33
3/9	*Dt*	36	14	1	51	56.5
4/1	*Dt*	1	38		39	
4/2	*Dt*	61	11		72	137.0
4/3	colorless	38			38	
4/4	colorless	111			111	
4/5	*Dt*	2	18	3	23	3.3
4/6	*Dt*		22		22	
4/7	*Dt*		51		51	
4/8	*Dt*		10	1	11	
5/1	*Dt*	1	5		6	0.2
5/2	*Dt*	10	8		18	9.0
5/3	*Dt*	3	43		46	8.4

It is quite evident that the isolated alleles mutate at a high rate in somatic and generative cells. These mutation events are most likely to be due to genetic element transpositions within the maize genome. However, we will refrain from a detailed genetic analysis of induced instability since the scale of the testcrosses performed is still not large enough. Also, it is necessary to test this new mutator system for interaction with the existing, well-studied mutator systems, as well as to try to determine the class of the genetic element (receptor or regulator) integrated into the marker line *wx sh* genome.

The unstable mutation induction could most probably have occurred by integration of a regulator resembling the *Dt* element into the recipient genome, the allele for anthocyanin synthesis already carrying a receptor element responding to the *Dt*-like element. This resulted in some cells acquiring violet color against the colorless aleurone background as early as the M1. The receptor element is less likely to be integrated into the allele for anthocyanin synthesis in the recipient, since its integration into the allele fails to produce the dotted-like pattern observed by us in some of the recipient ears. The dominant allele expression in some aleurone cells is always associated with the receptor excision, resulting in a reversion to the wild-type allele. A simultaneous integration into the recipient genome of both the regulator and the receptor appears unlikely. Currently, all three assumptions are being tested. However, even now it is clear that the *Dt*-like element is dominant, which is indicated by the 3/8 plant segregation and by the colorless x *Dt* testcrosses performed, where a 1:1 (8 *Dt*:12 colorless) segregation ratio was observed in F1.

Location of factors influencing grain yield by means of marker genes

--M.E.Mihailov and A.A.Chernov

The objective of the present study was to establish the effect of genome zones marked by mutant and morphological loci on grain yield. Two F2 populations were developed as source material for this investigation by self-pollinating the F1 hybrids Ku123 x 2-9m and UIT757 x 2-9m. The multimarked line 2-9m carries the following mutant loci (chromosomes and co-ordinates of loci are indicated in brackets): *ws3* (2-0), *lg1* (2-11), *gl2* (2-30), *y1* (6-17), *sh1* (9-29), *wx1* (9-56). The lines Ku123 and UIT757 have a red cob, 2-9m has a white cob. We suppose that this trait is conditioned by the gene *P1* (1-26). The parental forms within each pair are divergent in three non-located factors controlling the anthocyanin contents (dominant trait) in different parts of plant: factor 1 - in aleurone, factor 2 - in anthers, factor 3 - in seedlings. The line 2-9m carries the dominant allele of factor 1 and the recessive alleles of factors 2 and 3. These factors are linked to each other; factors 1 and 2 are especially tightly linked in repulsion. Their classification is difficult in some cases, and we can't exclude the possibility that these factors correspond to the gene *R1* (10-64).

According to chi-square test the F2 segregation of all the markers does not differ from the monogenic 3:1 and 1:2:1 ratios at P<0.01. The F2 plants were selfed and supplementary pollinated to provide ears with normal grain yield. The homozygous plants retain their genotype in F3, and the heterozygotes segregate. So, we can identify the F2 plant genotype and separate the dominant homozygotes from the heterozygotes for most of the loci. This was carried out for all the markers except *P1* and factor 2. The F2 segregation for *y1*, factor 1, and factor 3 is represented by three genotypic classes: the dominant homozygotes (+/+), the heterozygotes (+/-) and the recessive homozygotes (-/-); and for *gl2*, *lg1*, *ws3*, *sh1*, *wx1* by two: +/+ and +/- (the recessive homozygotes -/- were excluded from further analysis because they may decrease the grain yield of the plants). The F2 segregation for *P1* and factor 2 was determined by the phenotype in two classes: the dominant homozygotes and the heterozygotes (+/) and the recessive homozygotes (-/-). In each of the investigated classes the grain yield was studied for the following parameters: mean and variance.

In both of the F2 hybrids (Ku123 x 2-9m and UIT757 x 2-9m) statistically significant differences (P<0.05) were observed for the *wx1* marker (Table 1). The grain yield of the heterozygous plants (+/-) was higher by 15% than that of the homozygotes (+/+). Such a phenomenon may be related to the formation of the compensation gene complex (CGC), extinguishing destructive effects of semilethals due to breeding for viability (Strunnikov, J. Com. Biol. 35: 666-677, 1974). When the selected CGC line is crossed with unselected lines, the resulting hybrids have one CGC dose with additive effects and one dose of recessive semilethals without depression action in the heterozygote. Such gene combination appears to increase the heterosis effect. Since the multimarked line 2-9m carries some mutations, the CGC foundation may be conditioned by minimizing the negative mutation action during the breeding and reproduction of this line. Therefore the results suggest that in the line 2-9m the chromosome 9 segment marked by the *wx1* locus affects the grain yield due to CGC (or its component) factor.

This chromosome 9 segment is also marked by the *sh1* locus. According to Table 1 the difference of the grain yield in relation to the *sh1* locus had the same sign as that of the *wx1* locus, but wasn't as great or as statistically significant. Probably the grain yield factor is linked more tightly to the *wx1* than the *sh1* locus.

In the F2 hybrid UIT757 x 2-9m (Table 1) statistically significant differences in grain yield were observed for factor 2 between the classes +/ and -/- (P<0.01), and for factor 3 between classes +/+ and -/- (P<0.001); and, +/- and -/- (P<0.05). The highest grain yield was found for factor 2 in the class +/ and for factor 3 in the class +/+. Since the line UIT757 has the dominant genotype +/+ for both factors, the grain yield factors appear to be located in the line UIT757 in the chromosome zones marked by factors 1 and 2. As these factors are linked, probably there is only one high yield factor in the marked zones.

Table 1. The effect of marked genome zones on grain yield (gm per plant).

Marker Alleles		n	mean	error	variance	n	mean	error	variance
ws3	+/-	100	140.0?	4.4	1892	-	-	-	-
	+/+	53	153.8?	6.4	2157	-	-	-	-
lg1	+/-	101	144.9?	4.4	1938	98	138.9?	4.8	2237
	+/+	52	144.6?	6.5	2204	30	137.8?	8.9	2391
gl2	+/-	110	143.7?	4.4	2168	86	142.0?	5.0	2107
	+/+	43	147.5?	6.2	1669	42	131.9?	7.7	2550
y1	-/-	36	152.8?	8.1	2344	31	144.5?	6.9	1489
	+/-	89	146.4?	4.6	1867	64	140.3?	6.5	2735*
	+/+	31	133.4?	7.8	1867	33	130.0?	7.9	2061
sh1	+/-	99	147.7?	4.6	2095	80	141.4?	5.5	2440
	+/+	57	141.2?	5.7	1832	48	134.2?	8.4	1962
wx1	+/-	98	151.9?	4.6	2083	74	147.2*	5.8	2470
	+/+	58	134.2?	5.4	1679	54	127.1?	5.7	1772
P1	-/-	50	143.1?	6.9	2353	35	142.9?	7.4	1936
	+/	106	146.3?	4.2	1843	93	137.1?	5.1	2391
factor 1	-/-	35	139.7?	7.6	2016	33	140.2?	7.9	2070
	+/1	86	146.0?	4.7	1860	65	143.9	6.1	2430
	+/+	35	149.2?	8.3	2382	30	125.7?	8.1	1989
factor 2	-/-	41	149.1?	7.7	2425	23	119.1?	7.2	1204
	+/	112	145.1?	4.1	1837	105	143.0**	4.8	2401
factor 3	-/-	-	-	-	-	22	119.6?	8.4	1544
	+/-	-	-	-	-	82	139.7*?	5.6	2601*
	+/+	-	-	-	-	24	152.8***	7.5	1347
Total		156	145.3	3.6	1988	128	138.7?	4.2	2256

*, **, *** The differences from the minimum value in the group are significant at P<0.05, 0.01, 0.001, respectively.

In the same F2 hybrid (Table 1) for the locus *y1*, F-test for factors 2 and 3 has shown significant differences (P<0.05) for grain yield variances between the classes. The highest variances were found in the classes +/- for factor 2 in the class +/.

The results suggest that the marked genome zones affect the grain yield and its variance. Future research is needed to ascertain the nature of the effects observed.

Interaction of *Cg2+* revertants with the cytoplasm genome and between themselves

--N. V. Krivov

It has been shown earlier that up to 50% of plants with the *Cg2* phenotype are possible in F1, if reciprocally crossed stocks isolated in the progeny of the heterochronic *Cg2* mutation carry the *Cg2+* alleles resulting from the reversion *Cg2* ---> *Cg2+*.

Moreover, in some cases such mutation events are observed even with hybridization of the pollen parent, carrier of the revertant *Cg2+* allele, on the marker stock *gs bm2* (Krivov, MNL 66:56, 1992). Not only the induction of the *Cg2+* ---> *Cg2* mutation, but the suppression of the mutant *Cg2* depends on the genotype of the cytoplasm (Krivov, MNL 68:48, 1994).

This communication analyzes the case of the *Cg2+* allele mutation from *Cg2+* ---> *Cg2* with the hybridization *Cg2+ ys*-9196 br*-220* x *Cg2+*. Both partners participating in the cross were isolated in the heterochronic *Cg2* mutation progeny; i.e. they are carriers of the revertant *Cg2+* alleles and have the wild-type phenotype. These alleles mutate to *Cg2* with a frequency of 10%-15% in the pollen parent, whereas in the maternal stock, that also contains two recessive mutations originated in the heterochronic *Cg2* mutation progeny, it is stable since during the 4 years from isolating, no plants with the *Cg2* phenotype have been discovered in its progeny.

This hybridization produced 17 maize plants possessing the *Cg2* phenotype. In 4 plants, despite the fact that the central shoot ended in a tassel, numerous side shoots were *Cg2* and only one plant was confirmed to be wildtype. Such a progeny may be expected only from parents homozygous for the *Cg2* allele, i.e., both *Cg2+* alleles have become *Cg2* alleles.

The individual analysis of the progeny of 8 hybrid plants among 22 indicates that they are all heterozygous *Cg2 /Cg2+* (Table 1). Even the plant with the wt phenotype was *Cg2 /Cg2+*. Only family No. 45 characterizes the parent plant as a putative homozygote *Cg2/Cg2* . In order to know how frequently the mutation *Cg2+* ---> *Cg2* occurs at the hybridization with such an effect when nearly 100% of the offspring have the *Cg2* phenotype, repeated reciprocal crossings were performed with the same source of the *Cg2* alleles. This time, along with the families in which *Cg2+* ---> *Cg2* occurred, the families that were carriers of stable alleles *Cg2* were taken as well. In all, 13 families were checked. Five of them produced plants with the *Cg2* phenotype, and only in one of the 5 was the share of plants with the *Cg2* phenotype significant.

The appearance of more than 50% and sometimes even 100% of plants having the *Cg2* phenotype in the *Cg2+/Cg2+* compounds in the F1 showed that the nature of these mutation transitions is quite different from the mutation events that are observed in marker loci due to *Ac-Ds* insertions and excisions or any other well-known transposition elements. Moreover, the *Cg2* mutation is caused by the cytoplasm genotype. Therefore, the instability of the heterochronic *Cg2* mutation is likely to be related to the transpositions of an episome-like genetic element. Spontaneous reversions to male fertility in maize stocks with cms-S are explained by the presence of such episomal genetic elements that may be fixed either in the cytoplasm or nucleus (Kemble et al., Nature 304:744-747, 1983). The *Cg2* ---> *Cg2+* reversions are likely to occur in the cytoplasm and not in the nucleus, as has been observed in the cytoplasmic revertants towards fertility in which rearrangement of the R-sequence takes place regardless of the nuclear genetic background (Zabala et al., MNL 66: 110, 1992). Hence, these reversions do not affect the *Cg2* locus and are pseudoreversions. At hybridization, if the *Cg2* alleles hit the corresponding cytoplasm genotype, the mutant heterochronic phenotype *Cg2* is restored.

The event of the *Cg2+* allele mutation from *Cg2+* ---> *Cg2* , described here when nearly 100% of the offspring possess the *Cg2* phenotype, indicates that the earlier stable *Cg2+* alleles, which is a contribution of the maternal partner of *Cg2+ ys*-9196 br*-220* participating in this cross, have also become mutable. When the stable allele becomes mutable, the *Cg2+* alleles may be supposed to be subject to paramutation. As has been known for a long time, these are the *r* and *b* genes of anthocyanin biosynthesis in maize that are subject to paramutation (Brink, Ann. Rev. Genet. 7:129-152, 1973; Coe, PNAS 45:828-832, 1959). However, paramutable *R* and *B* alleles dominate over their recessive alleles while the paramutable *Cg2+* allele is recessive and it can demonstrate its paramutagenic properties only on a specific type of cytoplasm.

Thus, this report proposes a new model of the unstable heterochronic *Cg2* mutation behavior. This model will be further checked during the course of tests with the stocks, carriers of *Cg2+* and *Cg2* allele derivatives.

Table 1. Identification of parent plants *Cg2 /Cg2+* or *Cg2 /Cg2* for the genotype in progeny.

Fam. No.	Phenotype of self-pollinated parent plant	Offspring phenotype wt	Cg2	Total	X^2 1:3
39	M(*Cg2*) self; stolon wt	12	21	33	2.3
40	"	12	23	35	1.6
41	"	9	21	30	0.4
42	wt	13	25	38	1.7
43	*Cg2 m*	13	17	30	5.4*
44	"	8	24	32	0
45	"	3	30	33	4.5*
46	"	9	16	25	1.6
Total		79	177	256	4.7*

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Inheritance of incompatible genomes in maize and *Tripsacum dactyloides* (2n = 72)

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

Two incompatible lines (T22 and Gk26) and a compatible one (Ig57) were used in a previously described experiment (MNL 65:72, 1991; MNL 68:51, 1994). In 1993, P1, P2, P3, F1, F2, BC1 ears were pollinated with *Tripsacum dactyloides* pollen according to the standard procedure (MNL 68:87, 1989).

Analysis of the ears and calculations of compatibility (c) were determined by the method developed in our laboratory (MNL 65:72, 1991). In the set of lines under research, genome incompatibility is governed by duplicate genes. The probable genetic formula of the lines is: A1 A1 a2 a2 - Gk26, A1 A1 A2 A2 - T22, a1 a1 a2 a2 - Ig57.

Line or hybrid	Number of ears without developed kernels c = o	with developed kernels c>o	Expected ratio	X^2
Gk26	8	0	8 : 0	0.00
T22	7	0	7 : 0	0.00
Ig57	0	13	0 : 13	0.00
Gk26 x T22	7	0	7 : 0	0.00
Ig57 x Gk26	4	0	4 : 0	0.00
BC1 (Gk26 x Ig57)	11	0	11 : 0	0.00
BC1 (T22 x Ig57)	10	0	10 : 0	0.00
BC1 (Ig57 x Gk26)	9	11	1 : 1	0.02
BC1 (Ig57 x T22)	13	4	3 : 1	0.37
F2 (Ig57 x Gk26)	17	5	3 : 1	0.06
F2 (Ig57 x T22)	19	1	15 : 1	0.05

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Amplification of the methylenetetrahydrofolate reductase gene

--Michael Benner, James Johnson, Michael Weisberg, Deb Jones and Donna Cartledge

Methylenetetrahydrofolate reductase (MTHFR) catalyzes the reduction of 5,10-methylenetetrahydrofolate to 5-methyltetrahydrofolate, which in turn donates a methyl group for the conversion of homocysteine to methionine. Genes encoding this enzyme have been isolated from *E. coli*, *S. typhimurium*, *Saccharomyces cerevisiae* and, most recently, humans. Alignment of prokaryotic and eukaryotic amino acid sequences reveals several areas of homology; we hypothesized that these regions of homology also exist in higher plants. We have designed degenerate oligonucleotide primers that result in the successful amplification of a putative maize MTHFR gene.

Degenerate primers corresponding to the +76 to +95 region and the +338 to +356 regions of the *E. coli metf* sequence were synthesized. The amino acids encoded by these regions correspond in position to those found in the N-terminal 40 kDa domain of the human enzyme. The predicted length of the *E. coli metf* gene fragment amplified by these primers is 281 bp; the predicted length of the amplified human gene sequence is 293 bp.

Utilization of the above primers in PCR reactions containing maize genomic DNA results in the amplification of a single major fragment of approximately 270 bp. When the maize amplification product is radiolabeled and used to probe genomic DNA, a single major band is evident. We are currently isolating a full-length clone for subsequent sequence and expression analyses. The availability of a maize MTHFR clone will facilitate the investigation of methionine production in higher plants.

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Gene effects in flints–evolutive cycle, plant and ear traits

--M. B. Aulicino

In Argentina and other countries, hybrids with early ripening are usually sown in areas with short growth seasons. In the last few years they have also been cultivated in other areas. In Europe, they have been widely used in places with extreme climates as a principal resource or as a second crop using late sowing throughout the autumn-summer period. In Argentina precocious or semiprecocious maizes are being cultivated successfully in marginal areas, but also in traditional areas because of their present operative and commercial advantages, especially the semiprecocious ones. Gaspé, native to Canada, presents little agronomic value but is extremely precocious.

The aim of this paper is to determine the genic effects present in Gaspé crosses with red Argentine flint inbred lines to apply this information to the orientation of breeding and selection plans. With this aim in mind, inbreds H38, P21, P465 and P1338 from INTA Pergamino and CFE from the IFSC were used as "female" parents and "Gaspé" as pollinator. For each of the 5 combinations the F1's, F2's and the backcrosses by both progenitors were obtained. Cycle, plant and ear traits such as: days to tasseling (DT), ear insertion height (EIH), number of kernels per row (NKR), number of rows per ear (NRE) and ear length (EL) were evaluated. The adjustment to the additive-dominance model (Mather and Jinks, 1977) was tried (Table 1). For the crosses which did not fit the model the simple genic effects and interaction effects were tested by the six parameters model (Table 2). Gaspé was the dominant progenitor

Table 1. Mean estimates of 3 gene effects for the five crosses and chi-square values (x^2).

	EIH					DT				
	CFE	P1338	H38	P465	P21	CFE	P1338	H38	P465	P21
[m]	35.6	37.7	28.7	26.8	28.0	0.7	60.3	61.6	60.2	62.3
[d]	26.8*	29.7*	9.6*	18.7*	20.6*	15.1*	14.3*	15.6*	14.2*	16.7*
[h]	-4.0*	1.9NS	1.6NS	4.9*	-0.2NS	-1.0*	-1.8*	-3.3*	-2.9*	-6.7*
x^2	*	*	*	*	*	*	NS	*	*	*

	NRE					NKR				
	CFE	P1338	H38	P465	P21	CFE	P1338	H38	P465	P21
[m]	11.2	10.4	10.2	11.3	9.9	16.9	13.7	15.8	13.1	11.3
[d]	2.1*	1.7*	1.5*	1.8*	0.4*	7.1*	4.4*	6.4*	4.1*	2.1*
[h]	-.4NS	0.6*	0.8*	0.3NS	1.1*	2.9*	4.7*	4.4*	5.5*	7.2*
x^2	NS	NS	*	*	*	*	NS	NS	NS	NS

	EL				
	CFE	P1338	H38	P465	P21
[m]	12.2	12.8	12.6	11.7	10.5
[d]	1.2*	2.4*	1.8*	1.4*	0.1NS
[h]	-.8NS	-.7NS	0.5NS	1.0NS	1.5*
x^2	NS	NS	*	*	*

* Significant at 5% or 1 % probability level
NS Not significant

Table 2. Means estimate of the six gene effects.

	EIH					DT			
	CFE	P1338	H38	P465	P21	CFE	H38	P465	P21
[m]	23.3	27.6	73.0	25.4	31.2	66.3	77.0	5.4	55.1
[d]	27.5*	19.9*	29.9*	17.4*	19.8*	14.9*	16.4*	14.4*	19.5*
[h]	6.4NS	-6.7NS	-91.4*	.1NS	-22.3NS	-16.1*	-41.8*	17.2*	.8NS
[i]	13.7NS	1.7NS	-33.6*	1.5NS	-1.9NS	-5.4NS	-14.8*	9.8*	1.3*
[j]	3.3NS	-.4NS	3.8NS	14.3*	13.4*	3.3*	-4.8*	-1.4NS	-8.8*
[l]	9.5NS	13.3NS	62.8*	11.2NS	23.4*	1.0*	23.2*	-1.0*	.8NS

	NRE			NKR	EL
	H38	P465	P21	CFE	P1338
[m]	14.4	9.8	12.7	16.9	11.3
[d]	1.5*	1.8*	0.4NS	7.4*	2.6*
[h]	-10.8*	6.4NS	-3.3NS	-0.7NS	-0.7NS
[i]	-3.8*	1.1NS	-3.2*	-0.1NS	2.0NS
[j]	-0.3NS	-0.1NS	0.4NS	-9.5*	-1.3NS
[l]	7.8*	-5.1*	1.3NS	4.0NS	2.3NS

* Significant at 5% or 1 % probability level
NS Not significant

for the trait (DT), as the negative values of the genic effect [h] demonstrate. For all the ear traits and EIH, the inbreds were dominant with the exception of CFE for the traits EIH, NRE, and EL; P1338 for EL and P21 for EIH. The crosses with P1338 and CFE adjust perfectly to the three parameters model, assuring a simple inheritance for most of the traits studied, with the exception of the trait DT for CFE. As Gaspé was designated as P2 in the model, the positive value of [h] determined that the inbreds are the carriers of most of the alleles which increase the traits studied. As the male parent "Gaspé" is common to all crosses, the differences in the results are evidently determined by a differential genetic behaviour of the inbreds. For the traits EIH, DT and NRE, the crosses with H38 and P21 presented the majority of the significant simple and interaction effects. The additive effects greatly surpassed the dominant ones for the cycle, plant and some ear traits, assuring an effective advance in conventional methods of selection. Overdominance is revealed for some ear traits, especially for the crosses with P21.

A synthetic based on these materials could be used in a breeding program to obtain precocious materials with flint characteristics.

Embryo inviability in crosses of tetraploid (2n=40) X diploid (2n=20) can be overcome by embryo rescue

--María Dina García and María del Carmen Molina

In many unsuccessful crosses, fertilization and early embryo development occurs but some irregular events subsequently take place, mainly the failure of the endosperm to develop properly, resulting in embryo abortion and seed collapse (Raghavan V., In "Plant Cell, Tissue and Organ Culture", J. Reinert and Y.P.S. Bajaj, eds., p. 375, Springer-Verlag, Berlin, 1977). Embryo culture can be used to overcome this problem.

The aim of this work was to obtain triploid plants (2n=30) from crosses between tetraploids (2n=40) and diploids (2n=20) by embryo rescue.

Immature embryos were obtained from two crosses: inbred line N103A (2n=40) x cv. Colorado Klein (2n=20), and inbred line N104A (2n=40) x cv. Ever Green (2n=20). N103A and N104A were kindly supplied by Dr. E. B. Patterson in 1992.

Plants were grown in the greenhouse during spring. Caryopses were harvested 12, 13, 17 and 22 days after pollination (DAP) and surface sterilized. Embryos were aseptically excised, transferred to 10 ml culture medium and incubated at 28-30°C in 16 h photoperiod.

Embryos were cultured on the basic medium (García et al., 1991 MNL 65:76-77, 1991) supplemented with different concentrations of plant growth regulators (Table 1).

Caryopses collected at 22 DAP from N103A x Colorado Klein crosses looked shrivelled because of defective endosperm development. The embryos were excised from these caryopses at 1.8 to 2.2 mm length and cultured on A, B, C and D media. Of the embryos 48% showed coleoptilar growth but only 12% exhibited radicle development. One month after culture initiation, plantlets looked very weak and a few of them gave rise to adventitious roots (36%); subsequently no roots developed on rooting medium (basic medium + 1 $mg.L^{-1}$ alpha-naphtaleneacetic acid). After two months of culture, 64% of the plantlets had died in vitro. The remainder were potted but they didn't survive.

Caryopses from N103A x Colorado Klein and N104A x Ever Green crosses, harvested at 12 and 13 DAP respectively, showed

Table 1. Culture media used for plating immature triploid embryos.

Culture media	Plant growth regulators ($mg.L^{-1}$) Picloran	Kinetin	2,4-D	IBA
A	-	-	-	-
B	0.05	-	-	-
C	0.05	0.05	-	-
D	-	0.05	-	-
E	0.10	0.05	-	-
F	-	0.05	0.10	-
G	-	0.05	-	0.10
H	-	-	-	0.10

Abbreviations: 2,4-D = 2,4-Dichlorophenoxyacetic acid. IBA = Indole-3-butyric acid.

normal appearance. Embryo length ranged from 0.6 to 1.5 mm for both crosses. Caryopses from N103A x Colorado Klein collected at 17 DAP showed defective endosperm development and looked shrivelled. The embryo lengths were from 1.5 to 2.3 mm. These embryos were excised and transferred on E, F, G and H media (Table 1). During the first week of culture 100% of the embryos germinated without deformities on each culture medium. Plants showed narrow and slighty curly leaves and a few adventitious roots after 20 days of culture. Plants were potted and transferred to the greenhouse about 30 or 40 days after culture initiation. Few flowering plants were recovered (8%). These plants flowered from 90 to 170 days after culture initiation and gave morphologically normal tassels and ears but no seeds were obtained from self pollinated plants. Mature plant height varied from 40 cm to 130 cm.

Pollen viability ranged from 11% to 55% and cytogenetic analysis revealed a chromosome number 2n=30. At diakinesis and metaphase I chromosomes formed 10iii in 10% of the cells. The rest of the cells had different numbers of i, ii and iii. Cells at pachynema showed two nucleolar organizers and one or more persistent nucleoli at metaphase and anaphase. In 40% of the cells analysed at anaphase 15 chromosomes migrated towards each pole and in the remaining 60% different numbers of chromosomes migrated towards each pole. Abnormalities like lagging chromosomes, chromatid bridges or micronuclei were not observed.

In conclusion, caryopses from tetraploid (2n=40) x diploid (2n=20) crosses showed apparently normal embryos but endosperm growth failures. These caryopses were inviable, but mature plants (2n=30) were obtained from the embryos excised and cultured on nutrient media, although in vitro development of triploid embryos showed some abnormalities compared with the diploid ones under the same conditions (García et al., MNL 65:76-77, 1991; Van Lammeren, Acta Bot. Neerl. 37(1):49-61 1988).

Meiotic behaviour and DNA content in five races native to Bolivia

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Sixty-eight individuals from maize from Bolivia were studied. These individuals are from 5 races which represent different altitudinal strata. This fact let us compare differences in frequency of B chromosomes and total DNA content variation in different ecogeographical conditions.

The individuals from races of high altitudes (3200m), Tuimuru, Pisankalla and Jampe Tongo have a higher frequency of individuals with B chromosomes than those from lower altitudes (races Duro Amazónico and Blando Cureño, 200 m).

Individuals with 2n=20+1B (Table 1) have 10ii+1i in all studied cells (Fig. 1A). In 80% of the dyads (T1) studied the B chromosome is included in the nucleus. Individuals with 2 B

chromosomes have 10ii+1iiB in 80% of the studied cells (Fig. 1B). In 5% of them the B is not included in the nucleus. Thus, the meiotic behaviour insures the inclusion of B chromosomes in the nucleus in most of the studied cells.

Table 1.

		Races from 3200m			Races from 200m	
	0B	1B	2B	3B	0B	1B
No.	16	6	5	3	34	4
%	53	20	17	10	89.5	10.5
DNA content range (pg)	5.1-6.6	5.2-6.5	6.3	5.3-6.4	5.1-7.5	5.6-6.3
No.	9	5	1	3	23	3

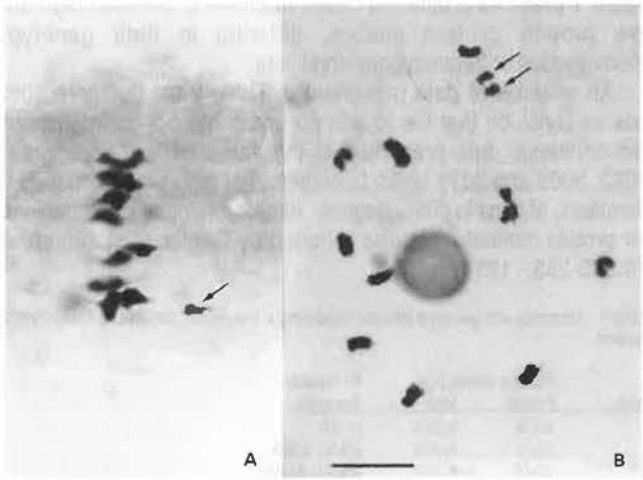

Figure 1. Race Pisankalla. a) Metaphase in individual with one B (10ii+1iB); B) diakinesis in individual with 2 B(10ii+1iiB). Bar=10µm.

The DNA content (2C, pg) was measured using the scanning methods with a Zeiss Universal Microspectrophotometer (UMSP 30), at a wavelength of 570nm. The method (Feulgen stain) was according to Tito et al. (TAB 83:58-64, 1991). The differences in DNA content were tested by an analysis of variance and the comparisons between means by using Scheffe's method. Several authors have found that there is a positive correlation among nuclear DNA content variation and several cellular parameters (chromosome length or volume, heterochromatin, banding pattern, satellite DNA, etc.). Variation has also been found in the nuclear DNA with respect to geographical distribution. Bennett (Jones & Brandham eds., Curr. Chrom. Research p. 151-158, 1976) concluded that there was a DNA amount-latitude cline. A high positive correlation between the northern limit of cultivation of several grasses and DNA amount per diploid genome was found. Rayburn et al. (Am. J. Bot. 72:1610-1617, 1985) observed an opposite cline in *Zea mays* ssp. *mays*.

With respect to altitude, Rayburn (Evol. Trends Plants 4:53-57, 1990) described in 12 maize populations from SW U.S. a significant negative correlation between altitude and genome size. These results are in agreement with the negative correlation observed between knob number and altitude in Mexican maize populations.

However, Rayburn and Auger (TAG 79:470-474, 1990) found that U.S.A. populations at 1500m have larger genome size than populations from lower altitudes. Low genome size appears to be a characteristic of maize from elevations above 1800 m (Rayburn 1990 ibid.). These data indicate that adaptation in maize to altitude, with respect to genome size, is very complex.

The variation in nuclear DNA content described could be due to intrachromosomal DNA or supernumerary chromosomal variation. As described above, races from high altitudes have a higher frequency of B's that those of lower altitudes.

In order to analyze DNA content variation independently of B's, we first analyzed DNA content of individuals with 2n=20 chromosomes. Analysis of variance showed that differences between races from higher and lower altitudes were nonsignificant (F=14.96, alpha=0.05). The only significant differences found were between two individuals, one from race Duro Amazónico (2C=7.51 pg) and the other from race Blando Cruceño (2C=5.13pg).

The variation in total DNA content of individuals with B chromosomes (1-3 B's) was within the range of individuals without B's. No significant correlation between B chromosome number and nuclear DNA content was observed. This suggests that intrapopulational variation in the amount of A chromosomal DNA (due to differences in knob number, heterochromatin amount, repeat sequences interspersed with nonrepetitive DNA, etc.) masks the variation due to number and size of B chromosomes.

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Alteration in the meiotic nucleolar cycle in individuals

--E. Quintela Fernandez and L. Poggio

The most common type of nucleolar behaviour is the "dispersive nucleolus," i.e., the nucleolus dispersing during prophase and reappearing at telophase. In some organisms, Risueño and Medina (Rev. Biol. Cel. 7:1-162, 1976) described a "semipersistent nucleolus" in mitosis. In this case, the nucleolar material remains in the cytoplasm during anaphase and telophase. In most organisms the nucleolus at meiosis is not a visible organelle from diakinesis onwards. The analysis of the meiotic behaviour of individuals belonging to several Bolivian races of maize reveals that the nucleolus initiated the dispersion at prometaphase I but persisted as a discrete structure during metaphase I and anaphase I. However, its size decreased with the progression of meiosis (Fig. 1A). At metaphase the nucleolus was usually located in one of the poles (Fig. 1A). At the end of anaphase I prenucleolar bodies appeared and the nucleolar reorganization occurred in the normal way. The nucleolar behaviour during meiosis II was normal.

This phenomenon was observed in all the studied anthers of some individuals of the races Blando Cruceño and Tuimurú. In other individuals of these and other races (Pisankalla, Jampe Tongo and Duro Amazónico) the "semipersistent nucleolus" was detected only in some of the anthers.

To study this phenomenon young anthers were fixed in 3 parts of absolute alcohol: 1 glacial acetic acid. The squash of anthers was made in a drop of acetic haematoxylin. Feulgen staining was done as indicated in Tito et al. (TAG 83:58-64, 1991) and nucleolar silver staining was carried out according to the technique used by Lacadena et al. (1984).

The absence of DNA in the "semipersistent nucleolus" was indicated by its failure to stain after Feulgen treatment. The presence of proteins was indicated by its positive silver staining (Fig. 1B). These results support the nucleolar nature of this body, although it was not possible to discriminate whether the persistent material belong to the granular or fibrillar component.

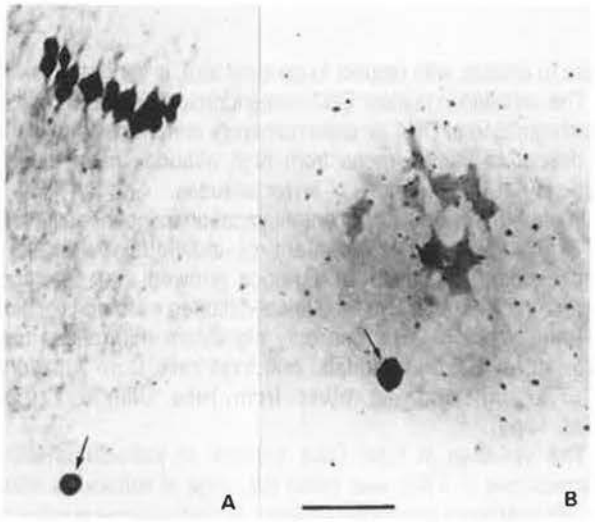

Figure 1. Metaphases I, with semipersistent nucleolus. A) Haematoxylin stain; B) Silver stain. Bar = 10 µm.

A structure called "nucleolar body" was described in several accessions of *Bromus* and *Zea* (Walters). This would not be the case in the individuals here studied because in most of the cells only one normal nucleolus was observed until diakinesis and additional nucleolar-like bodies were not detected. The observation of more than 50 cells suggests that the dispersion of the nucleolar material, in this case, is very slow and persists during meiosis I.

In Bolivian races the presence of B chromosomes is frequent. It is interesting to point out that the atypical behaviour of the nucleolus described is not related to the presence of B chromosomes. From a total of 36 studied individuals (growing in the same conditions), 8 presented "semipersistent nucleolus" (6 without B's, 1 with one B and 1 with 2 B's). In general there are no differences in the nucleolar behaviour among individuals with and without B's.

The "semipersistent nucleolus" was also observed in several individuals of alloplasmic lines (lines of maize with cytoplasm of *Zea mays* ssp. *mexicana*, Poggio et al., in prep.).

Inheritance of protein content in the endosperm

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

During 1993 and 1994, 105 different pedigrees of maize were evaluated for their protein content in the endosperm. A sample constituted of 30 to 60 grains was taken from each pedigree. The pericarp and the embryo were removed from each individual grain, then the endosperm was defatted, air-dried and milled to 60 mesh. Proceeding in this way, samples of each pedigree were prepared and the protein content for each was determined following the classic procedures of Kjeldahl method (A.O.A.C., 1965 and 1981).

The endosperm texture of these maizes is variable: some of them are flint, others are *opaque2* and many of them are *waxy*. Thirty-seven pedigrees were selected as they had more than 10% protein content in the endosperm (see Table 3). These materials will be employed in future breeding plans.

The pedigrees analyzed could be classified as follows: a) inbred lines (Sn); b) F1 hybrids; c) F2 hybrids; d) First Backcross (BC1); e) populations; f) first sib generation (SI1); g) first self-pollinated generation (S1) and h) second self-pollinated generation (S2).

The different F1 and F2 hybrids, as well as the BC1's, were obtained through different types of crossings such as:

INBRED LINE X INBRED LINE

INBRED LINE X POPULATION and its reciprocals

POPULATION X POPULATION

The data obtained from the several analyses confirm the hypothesis developed by Magoja (Com. Inv. UNLZ 3(6), 1980) in which a model useful to explain the inheritance mechanism for protein content was presented. This hypothesis was stated supposing crossing between inbred lines, but our results demonstrate that it is also suitable for crossing between heterozygotes.

Let a/a/a or a/a/A be the genotypes for low protein content and A/A/A or A/A/a the genotypes for high protein content. Table 1 presents 8 different cases of crossing between high and low protein content maizes, differing in their genotype (homozygous or heterozygous condition).

An analysis of data presented in Table 2 for Pedigree 3063 lets us conclude that the lowest protein content is overdominant. Nevertheless, data presented in the same table for Pedigrees 3062, 3065 and 3075 seem to confirm that high protein content is dominant, at least in some degree. Similar inheritance mechanisms for protein content were also reported by Genter et al. (Agron. J. 49:283-285, 1957).

Table 1. Crossings and genotype structure supposing a: low protein content and A: high protein content.

Case	Parents Genotypes Female	Male	F1 Possible Genotypes
1	a/a/a	A/A/A	a/a/A
2	a/a/a	A/A/a	a/a/a; a/a/A
3	a/a/A	A/A/A	a/a/A; A/A/A
4	a/a/A	A/A/a	a/a/a;; a/a/A; A/Aa; A/A/A
5	A/A/A	a/a/a	A/A/a
6	A/A/A	a/a/A	A/A/A; A/A/a
7	A/A/a	a/a/a	A/A/a; a/a/a
8	A/A/a	a/a/A	A/A/A; A/Aa; a/aA; a/a/a

Table 2. Protein content (in percent) for different types of crossings according to the model presented in Table 1.

Case	Parent Female	Male	F1 Hybrid
1	ZN6 (9.7%)	H38 (10.2%)	93-3063 (8.8%)
3	SCV2 (8.1%)	ZN6 (9.7%)	93-3062 (10.4%)
4	SCV1 x A255 (9.3%)	SCV1 (10.8%)	93-3074 (10.1%)
4	SCV1 x A255 (9.3%)	FW (11.4%)	93-3075 (13.8%)
5	H38 (10.2%)	ZN6 (9.7%)	93-3065 (11.1%)
6	CM1 (12.3%)	WEM (8.7%)	93-3066 (11.4%)
8	SCV1 (10.8%)	SCV1 x A255 (9.3%)	93-3073 (10.7%)

Observing Table 2 in its whole and according to the genotypes presented in Table 1, it may be concluded that, in addition to the dominance or overdominance observed, the genotype of the female parent of the crossing strongly conditions the protein content of the results observed for the reciprocal hybrids 3063 and 3065.

In many experiments of selection, negative correlations between yield (bu/a) and endosperm protein content were observed. For example, when high yielding plants are selected, the protein content is reduced and this fact could explain the generalized low protein content of commercial hybrids. Jugenheimer (Corn, J. Wiley & Sons, NY, 1976) analyzed in 145 maize inbreds the previously mentioned correlation and found a value of r = -0.522.

According to the previous paragraph in complete agreement with Bathia (Euphytica 24:789-794, 1975) it would not be advantageous to select separately by yield or protein content. The most effective way of selection would be considering

Table 3. Percentage of protein content and endosperm texture of the 37 pedigrees selected from the whole analyzed.

Generation	Pedigree	Protein Content %	Endosperm Texture
So	3012	10.49	waxy
	3013	10.80	waxy
S2	3023A/E1	11.90	waxy
F1	3024	10.06	flint:waxy
F2	3024A	10.49	flint:waxy
	3024S	10.60	flint:waxy
	3075	13.76	flint:waxy
BC1	3074	10.07	flint:waxy
S1	3014A	10.16	waxy
F1	3080	13.63	flint:waxy
	3084	10.05	flint:waxy
	3062	10.42	flint:waxy
F2	3079	12.60	flint:waxy
BC1	3072	10.93	flint:waxy
So	3003S	10.05	waxy
	3018	12.48	waxy
S1	3003A	12.47	waxy
F1	3066	11.38	waxy
So	3004	11.38	waxy
S2	3022A	10.27	waxy
SI1	3021S	11.38	waxy
F2	3077	11.96	flint:waxy
So	3008S	10.82	flint
S1	3030A	10.27	flint
	3031A	10.28	flint
SI1	3031S	11.27	flint
	3040S	10.28	flint
F1	3071	10.28	flint
Sn	3041	11.19	flint
	3042	10.49	flint
	3046	12.92	flint
	3048	11.38	flint
	3049	10.16	flint
	3083	10.28	opaque2
	3086	10.16	opaque2
F1	3064	10.93	flint
	3065	11.15	flint

simultaneously both traits (yield and protein content), which means to evaluate yield in protein content/grown surface (Kg protein/acre).

Variation in the transmission of B chromosomes, 0B x 1B and 1B x 0B crosses

--M. Rosato, A.M. Chiavarino, M.J. Puertas, C.A. Naranjo, and L. Poggio

Longley (J. Agr. Res. 35:769-784, 1927) first described that B chromosomes (B's) are present in unselected races of maize. We have found that B polymorphisms are common in unselected local populations of maize from Argentina and Bolivia. The B's can be maintained by their mechanisms of "drive," which consist in suppression of meiotic loss and nondisjunction at the second pollen grain mitosis (Roman, Genetics 32:391-409, 1947; Carlson and Roseman, Genetics 131:211-223, 1992), together with preferential fertilization of the sperm nucleus carrying the B's (Roman, Proc. Natl. Acad. Sci. USA 34:36-42, 1948; Carlson, Genetics 62:543-554, 1969) and higher competitive ability of pollen grains carrying the B's (Beckett, J. Hered. 73:29-34, 1982).

The nonMendelian behavior of B's produces a variation in their transmission. The genetic control of this variation was demonstrated in *Myrmeleotettix maculatus*, *Pseudococcus affinis*, *Secale cereale* and *Aegilops speltoides* (Romera et al., Heredity 66:61-65, 1991; Cebriá et al., Amer. J. Bot. 81:1502-1507, 1994; Jiménez et al., 1995).

We are currently investigating whether such a genetic control of B transmission occurs in maize. The present work reports the analysis of the variation of the rate of B transmission in 1B x 0B and 0B x 1B crosses.

A large sample of 145 individuals of the Pisingallo race (from N.W. Argentine) were screened for B number in the root tips. The seeds were grown on wet filter paper, pretreated with 0.02M 8-hydroxyquinoline for 3 hours and fixed with ethanol:acetic, 3:1. They were stained with 2% hematoxyline and ferric citrate as mordant. From this sample we selected 0B and 1B individuals, and made 20 crosses female 1B x male 0B and 20 crosses female 0B x male 1B. The progeny were collected plant by plant and at least 25 individuals per spike were similarly screened for B number. With these results, the mean number of B's transmitted in each cross was calculated.

In the 0B x 1B crosses we found a large range of variation in the mean number of B's transmitted (0.17 - 1, the average mean being 0.52 ± 0.26). The differences among crosses were due to the proportion of 2B vs. 0B progeny because most plants of the progenies showed 0B or 2B, 1B plants being rarely found. This indicates that nondisjunction occurred in nearly 100% of the cases. On the other hand, this also indicates that the mechanism of nondisjunction occurs irrespective of the mean number of B's transmitted. Therefore, both processes seem to be independently controlled.

In the 1B x 0B crosses the range of variation was smaller (0.31 - 0.58, the average mean being 0.47 ± 0.08). The plants of the progenies showed either 0B or 1B, indicating the lack of nondisjunction on the female side (Fig. 1).

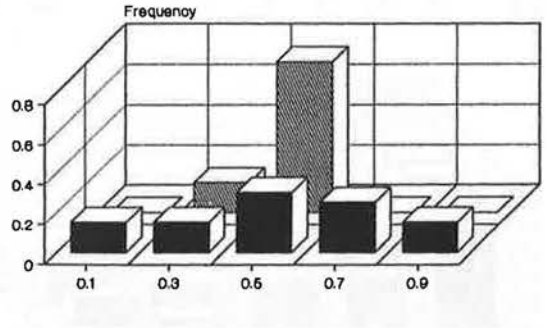

Figure 1. B chromosomes transmitted to the progeny.

If the B's were not lost during meiosis, and nondisjunction occurred in all cases at second pollen grain mitosis, the expected frequency of B's transmitted to the progeny in 0B x 1B crosses would be 25%. Figure 1 shows that much higher frequencies (50%) were observed in our experiment. This indicates the occurrence of preferential fertilization by the sperm nucleus carrying 2B (Roman, Proc. Nat'l. Acad. Sci. USA 34:36-42, 1948).

In some 0B x 1B crosses the frequency of transmission was significantly lower (8%). Therefore, there is a variation in the frequency of B transmission. It is possible that preferential fertilization does not always occur.

Our results show a variation in the rate of transmission which can be related to a genetic control on the preferential fertilization, or other processes of male and female gametogenesis.

The differences between the variation in B transmission rate

on the male and the female are remarkable. In our opinion this strongly suggests a genetic control of the variation, because if the variation were due only to environmental causes, the expected variations would be the same in both sexes.

The polymorphism for genes controlling the B transmission rate may explain the differences in B frequency in different populations of maize.

B chromosome polymorphism in N.W. Argentine populations

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Supernumerary chromosomes (B's) are one of the most frequent forms of naturally occurring chromosomal polymorphisms. These polymorphisms are common in maize populations, however there are few studies at population level. In native races of maize from Argentina there are no studies analyzing the frequency of polymorphism due to B chromosomes.

In the present work the frequency of B chromosomes and the average mean number of B's per individual belonging to six populations from the races Amarillo de Ocho, Capia Rosado, Capia Blanco, Altiplano, Pisingallo is analysed (Table 1, Fig. 1).

Table 1.

Population	0B	1B	2B	3B	4-7B	Percent Ind. w/B	B/Ind	Num.
Amarillo de Ocho	45	31	11	-	-	55	0.9	64
Altiplano	50	18	9	9	14	50	1.18	22
Capia Rosado	52	36	8	-	4	48	0.8	25
Capia Blanco	44	21	20	3	12	56	1.2	34
Pisingallo (I)	54	35	8	3	-	46	0.6	37
Pisingallo (II)	56	28	13	2	1	44	0.62	78

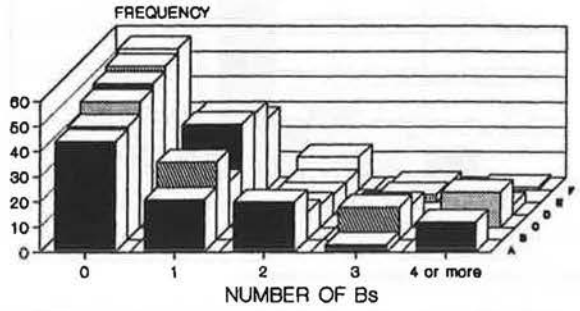

Figure 1. Frequency of B chromosomes among populations.

In all cases the chromosome counts were made in root tips pretreated with 0.02M 8-hydroxyquinoline for 3 hours. They were fixed in ethanol:acetic (3:1), and stained by 2% acetic hematoxylin. The populational frequency of B's was obtained from 182 individuals (22 to 78 in each population, see Table 1). The meiotic behaviour studies were made in male inflorescences fixed and stained in the same form as described above.

The B frequency was found to range from 44% to 56% in the studied populations (% individuals with B's) (Table 1). The G test indicates that there is a significant difference in the frequency of B's among some of the populations. Moreover the frequency of individuals with different doses of B's varies among the studied populations (Table 1, Fig. 1).

The analysis of meiotic behaviour of individuals with different doses of B's during the male gametogenesis indicates that B's are not lost during meiosis. This fact would contribute to the maintenance of the described B's polymorphisms.

The interpopulational variation described could be due to different selection pressures in each of the populations and/or polytypism for genes affecting the B transmission to the next generation.

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Agronomic traits variation in a hybrid among lines with mobile elements

--Luis B. Mazoti and Ana M. Broccoli

Mobile elements are useful in inducing genetic diversity. Such effects have evolutionary and breeding implications. Their distribution in important breeding lines suggests that they play a role in the endeavor to create better crops (Peterson, P. Breed. Rev. 4:81-121, 1986). We propose to investigate if variation in quantitative traits may be found by evaluating a hybrid derived from a mutational *Ds-Ac* type system and if such a variation is associated with mutation in aleurone layer color. The visible effect gene marker is *C1*, that controls anthocyanin production (colored aleurone) which by *Ds-Ac* action induces mutation to *C1-I* (colorless aleurone) (Mazoti MNL 40:62, 1966).

The female parent line had purple aleurone. We used seven pedigrees of the same S8 line derived from the *Ds-Ac* system as pollinator, and separated colorless kernel progenies (mutant) from purple ones (normal). Hybrid materials were evaluated over three years at the Instituto Fitotecnico de Santa Catalina by ear yield components in a Complete Randomized Design. ANOVA and Krushkali-Wallis analyses were used, considering the variable pollinator aleurone color as the main effect.

Descendants of colorless aleurone progenitors (mutants) showed greater yield potential than those of their siblings from purple aleurone parents. In the first and third year, excluding ear length, there were significant differences ($P = 0.05$) in favor of the colorless group. In the second year, with severe water stress, the traits ear diameter and hundred-grain weight gave statistical differences ($P = 0.05$) showing superiority of the purple group, so showing inverse behavior. This opens up the debate on the major aptitude for colorless genotype yield, demanding superior environmental quality to demonstrate its potential.

The variance values differ from group to group, suggesting a greater potential variation in the non-marker mutation purple group. An unexpected genetic component of variance appears among the simple hybrid combinations. Agronomic traits like yield aptitude and water stress response vary from group to group, showing distinct biological strategies under different environmental conditions.

These results suggest that this genetic material could generate diversity in traits of agronomic interest that can be useful in plant breeding programs.

Effect of plant density on yield and quality of forage maize

--L. Bertoia, R. Burak and A. Nivio

During 1990/91, 91/92 and 92/93 field trials were carried out in the boroughs of San Vicente and Cañuelas, Buenos Aires

Province to evaluate the effect of 3 plant densities (60, 80 and 100 thousand pl/ha) on the yield of dry matter from ear (EDMY) and stover (SDMY), on plant height (ALT), and on the percentage of the morphological composition of the plant (% of ears or harvest index (HI)). Among 16 hybrids per trial for this work, information from 5 hybrids that were repeated for 3 years was taken into account: Cargill Record 160, Cargill Semiden 5, Funk's Tronador, Morgan 506 and Pioneer 3456.

The planting was on typical argiudol soil. The design used was random blocks with 4 replications and a factorial arrangement of 5 hybrids x 3 densities x 3 environments. ANOVA was carried out using a mixed model and the Tukey test (5%) for multiple comparisons. The obtained values demonstrate that the increase in density produced a significant increase in the total DM production, giving means of 19270, 20830 and 22710 kg/ha for 60, 80 and 100 thousand pl/ha respectively. The analysis of the results permitted the adjustment to a first grade lineal response of the TDMY to a plant density within the evaluated interval. The trait EDMY showed significant differences between the values of 60 and 100 thousand pl/ha (10070, 9335 and 8703 for 100, 80 and 60 thousand pl/ha). In the analysis of HI (EDMY/TDMY) significant differences due to the effect of plant density were not recorded: 0.4438, 0.4424 and 0.4362 for 60, 80 and 1000 pl/ha respectively. Comparing hybrids, significant differences were seen only between Cargill R160 (47.86%) vs. Cargill SD5 (43.57%), Pioneer 3452 (42.36%) and Morgan 506 (41.76%); for Funk's Tronador (44.81%) significant differences with the rest of the participants were not shown. Effect of density upon the height of the plants was not observed (range: 2.37 to 2.35 m).

In analysing the environmental effect significant differences appear for ALT and HI. The interaction hybrids x environment was significant for the traits EDMY, SDMY and TDMY, demanding an analysis of these variables per environment.

The results obtained indicate that all the hybrids produced an increase of TDMY with greater densities without modifying the morphological composition of the plant. The characteristics of the environment produced a differential response of the evaluated materials.

In agreement with these results, the plant density used in the area of influence (60 thousand pl/ha), reaching approximately 50 thousand plants per ha at harvest is lower than the adequate amount for hybrids commonly used in silage production.

In conjunction with the analysis of the effect of the plant density upon the DMY undertaken in the first part, its effects upon 3 very important parameters for establishing the quality of maize destined for silage were also assessed in a lab: concentration of soluble carbohydrates,% protein and digestibility of the DM from ears and stover in the same 5 commercial hybrids.

Soluble carbohydrates--This is considered fundamental to reach adequate conservation of the pierced material quickly. The concentration of soluble carbohydrates of DM coming from the whole plant was not affected by the three densities evaluated (19.8%, 19.23% and 19.07% for 60, 80 and 100 thousand pl/ha respectively). Significant differences were found for the five analysed hybrids (range: 19.73% to 21.12%). Also differences were produced between the evaluated environments (range: 18.02% to 20.99%). The analysis of the plant components separately showed that in the case of ears and stover the density did not affect concentration of soluble carbohydrates (range: 26.06% to 26.89% for ears and 13.54% to 13.63% for stover). It was found that in both cases the interaction of hybrids and environment was significant.

Protein--The concentration of proteins in whole plants did not show differences between hybrids or densities. On the other hand, modifications due to environmental effects were produced. The interactions of hybrids x enviroments, hybrids x densities and densities x environments were not significant. On analyzing the components of the plant significant differences were found only for environmental effect on stover (range: 4.40% to 5.57%) and on ears (range: 6.52% to 7.28%). In the later there were also differences between hybrids (range: 6.68% to 7.25%).

Digestibility--The digestibility of the ear was affected by the density. Significant differences were found between 100 thousand pl/ha (78.77%) vs. 80 thousand pl/ha (81.20%) and 60 thousand pl/ha (80.82%). In the stover fraction significant differences were not found in the percentage of digestibility due to density. In both components the interaction of hybrids x environment was significant. The digestibility of the DM of the whole plant was not affected by the increase in density (66.44%, 67.87% and 68.06% for 100, 80 and 60 thousand pl/ha respectively).

The yield in digestible DM of the whole plant is the product of the total DM yield by the digestibility. The statistical analysis of this trait showed significant differences among the five hybrids evaluated (rank: 14,080 to 15,630 kg/ha); on the other hand, the density of 100,000 pl/ha produced a yield of 16,010 kg/ha of digestible DM, a value which significantly surpasses those attained by the density of 80 thousand pl/ha (14,650 kg/ha) and 60 thousand pl/ha (13,870 kg/ha). In accordance with these results and those obtained in the first part of this work, the greater densities employed produced significant increases in digestible DM, without varying the concentration of soluble carbohydrates and proteins of the different fractions which make up the plant.

Forage production in Argentine land races

--R. Burak, L. Bertoia and C. López

The increase in demand for dairy products, mainly due to the new MERCOSUR, has led to the need to incorporate high technology in the dairy farm, with the aim of improving productivity. The use of maize silage is standard practice, because it allows the harvested forage to be used strategically in times when grass production is low. The scarcity of improved maize forage in the Argentine market shows the need to develop plans for investigation in this area. Following this criterion, the aim of our work was to find out about forage behavior of maize land races and its crosses, and the detection of materials for starting a breeding program.

Eight (1 to 8) land races from the germplasm bank of INTA Pergamino, their 28 crosses and 4 checks, which have shown to be good dry matter producers (Morgan 369, Dekalb 4F37, Funk's Tilcara and Cargill Semiden 5) were evaluated. The experimental design used was alpha lattice 8x5 with 4 replications. The planting was carried out in Esteban Echeverría and Cañuelas locations, which form part of the Buenos Aires milk-belt. The variables analysed were: Total dry matter yield (TDMY), ear dry matter yield (EDMY) and stover dry matter yield (SDMY). The averages adjusted by lattice for the land races and crosses of EDMY and SDMY were analysed following Gardner and Eberhart's model II for diallel crosses. In the ANOVA, an error probability ($P < 0.05$) was used.

The combined ANOVA (Table 1) detected significance for genotypes in TDMY, EDMY and SDMY, for populations in SDMY and for their interactions with locations in all the studied variables.

Taking EDMY into account, the heterosis parameter and its components average heterosis, varietal heterosis and specific heterosis are significant, while their respective interaction with locations is not. The sum of squares of heterosis was the most important source of variation. The crosses with major EDMY were 2x7 and 2x4, showing high percentages of high parent heterosis (52% and 31% respectively) and similar values to the check hybrids.

Table 1:. ANOVA for a diallel set of eight land races.

S.V.	D.F.	M.S.		
		TDMY	EDMY	SDMY
Genotypes	35	*	*	*
Populations	7	*	ns	*
Heterosis	28	*	*	ns
Average Het.	1	*	*	ns
Variety Het.	7	ns	*	ns
Specific Het.	20	ns	*	ns
Gen.* Loc.	35	*	*	*
Pop.* Loc.	7	*	*	*
Het.* Loc.	28	ns	ns	*
Av.Het.* Loc.	1	ns	ns	ns
Var.Het.* Loc.	7	ns	ns	ns
Spec.Het.* Loc.	20	ns	ns	ns
Combined Error	202			

*Indicates significance at the 5% level of probability.
ns Indicates no significance.

For SDMY the heterosis parameter did not show significance in the combined analysis or in the individual one corresponding to Esteban Echeverría, while in Cañuelas the three heterotic components turned out to be significant. In both locations the sum of squares of populations was the most important source of variation. The crosses with the best SDMY were 1x3 and 1x4. The crosses 2x7, 2x4 and 1x3 showed the greatest TDMY after taking the average of the yield in both locations.

The heterosis expressed by some crosses, with yields in certain cases greater than that of the checks, indicates the possibility of deriving inbreds of their progenitor land races. That will depend upon these results being repeated in future evaluations.

Forage characterization of Argentine commercial hybrids

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In the last few years the use of the whole maize plant as fodder has spread throughout Argentina. Due to the lack of selected materials specifically bred for this purpose, grain hybrids are often used. Our aim was to differentiate commercial hybrids through traits related to aptitude for forage production.

The evaluated traits were plant height (PH), weight of ear (WE), weight of stover (WS),% protein in stover (PS) and in ear (PE),% soluble carbohydrates in stover (SCS) and ear (SCE),% digestibility in stover (DS) and ear (DE) in five commercial maize hybrids: Cargill R160 (R160), Semiden 5 (SD5), Funk's Tronador (TRON), Morgan 506 (M506) and Pioneer 3452 (P3452) for a three year period in three environments. The dendrogram obtained from a similitude matrix, using the Manhattan distance coefficient and the UPGMA technique groups R160 with M506 and SD5 with TRON. The four make up a subgroup which is clearly divided from P3452. The analysis of principal components established that PH (0.92), and SD (0.93) are the traits with the greatest discriminatory value in the first component, which explains 46.25% of total variation. The second component explains 23.48%, PS and WS being the most important characters. The third explains 16.87% where WE (0.96) is the principal character. The first component separates out P3452, the hybrid which possesses the highest values of PH and DS, from the rest. The second component groups R160 with M506 and separates them from P3452, TRON and SD5. The first two present the highest PS values. The third component puts M506 at one extreme and R160 at the opposite extreme, this being the hybrid which shows the greatest ear yield. The biochemical characters of the stover, relevant to forage corn, show a higher discriminatory value in the first and second components. In the third component the ear yield, fundamental for grain maize, is the greatest. The analysis concludes that P3452 is the hybrid with the best response to the forage idiotype and the rest of group, principally R160, to the grain idiotype.

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Map locations of three 18 kDa hsps

--D. Maillet, C. Richardson and D.B. Walden

The heat shock proteins (hsps) of maize are encoded by multigene families. The DNA sequences of three 18 kDa hsps and the induction of the small hsp family have been investigated in maize inbred Oh43 (Atkinson et al., Developmental Genetics 14:15-26, 1993). As part of our studies on the 18 kDa hsp gene family DNA probes which are specific for the 3' regions of three different 18 kDa hsp have been assigned map locations using two recombinant inbred (RI) families (T232 x CM37 and Tx303 x CO159). cMHSP18-1-1, cMHSP18-3-3, and cMHSP 18-9-3 (Atkinson et al., 1993) have been designated as *uwo9*, *uwo11*, and *uwo10*, and placed on the RI linkage map at positions 9L085, 8S056, and 3S049 respectively. The placement of *uwo10* is consistent with other RI mapping data (Helentjaris et al., MNL 68:101-104, 1994), which also indicate that there is an 18 kDa hsp on 3S at a similar location.

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Twin seedlings

--Gurmit Singh and S.S. Gill

During germination trials in the laboratory on cv. Partap-I, while sorting out normal and abnormal seedlings, some seedlings were encountered which had two coleoptiles (Figure 1). In the literature, nothing was found on seedlings with two coleoptiles in maize. In rice, seedlings containing two coleoptiles were designated as "twin seedlings" by Professor Yuan Long, Director of the Hunan Hybrid Rice Research Centre in Changsha, China. The frequency of the twin seedlings was 0.25 percent. The experiment was repeated many times, and each time the twin seedlings were planted in a pot but none grew beyond 10-15 days. Rice embryo sac analysis indicated that most twin seedlings arose from fertilized two-egg nuclei or multiple egg nuclei and cleavage embryos. Yuan reported adventitious embryos in the embryo sac

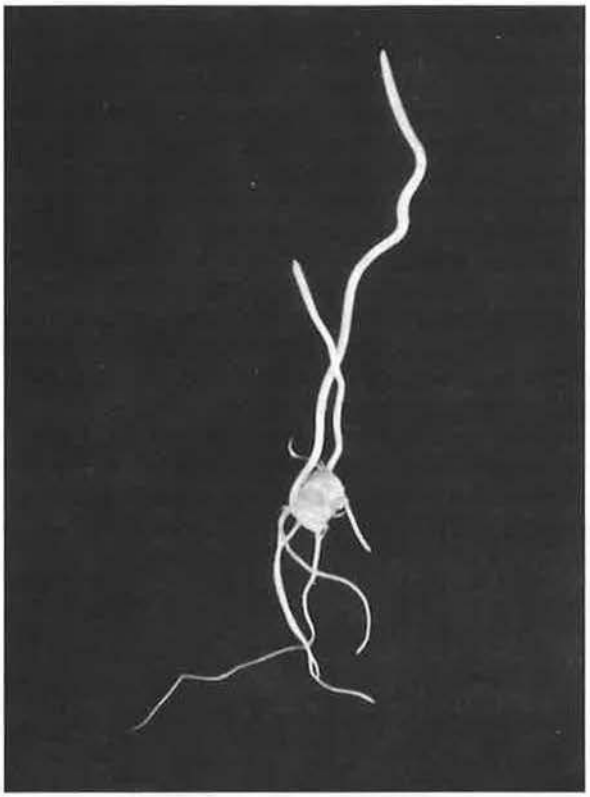


Figure 1. Seedling with two coleoptiles.

with frequencies of 2.6 to 5.1 percent, which originated from the nucellar cells.

According to Yuan these twin seedlings indicate apomixis in plants. Apomixis or asexual reproduction is a genetic tool to develop true-breeding hybrids with permanently fixed heterosis. This facultative apomixis could be used by (1) using some chemicals that can selectively kill fertilized eggs but not the adventitious embryos; or (2) transferring the long dormancy character to such twin seedlings and sowing F1 hybrid seeds before the alleviation of dormancy.

During a search for the twin seedlings trait at IRRI, seven rices (of 452 screened so far, from India, China and Guyana) exhibited this trait with a frequency of 0.14 to 0.51 percent. One rice line from China also possessed triple seedlings at the rate of 0.67 percent. The frequency of twin seedlings can be increased by germinating the seeds without shell. Nitrogen in the germination media, hydrogen peroxide treatment and germinating the seeds at optimum temperature also enhanced this trait.

However, the significance of the twin seedlings in maize has to be realized. Work on the establishment of the twin seedlings characteristic is in progress.

MADISON, WISCONSIN
University of Wisconsin

The origin of bicolor sweet corn as told by Oscar Pearson on 11 November 1994

--W.F. Tracy

Dr. Oscar Pearson was breeding sweet corn for Eastern States Farmers Exchange in Westfield, Massachusetts. He was working closely with D.F. Jones of the Connecticut Agricultural Experiment Station at New Haven.

Jones had a field corn breeder friend at Purdue who liked sweet corn. Jones crossed P39 (yellow endosperm) by Luther Hill (white endosperm) and gave it to his friend at Purdue. This was about 1946 or 1947.

Pearson thought this hybrid was "bland, not very tasty."

Meanwhile at Eastern States, Pearson had taken a white Crosby from northern New Hampshire and converted it to yellow endosperm. The new line had "good solid texture it didn't shrivel too much. It just dimpled."

He then made a line cross, P39 x Yellow Crosby (yellow endosperm) and crossed this to Luther Hill (white endosperm). It was considerably sweeter than P39 x Luther Hill. Pearson believes that he got some modifiers for high sugar levels from the yellow Crosby. This cross, (P39 x Yellow Crosby) x Luther Hill, was named `Butter and Sugar' and it was released about 1950.

Two modifiers reduce the level of expression of *Corngrass1* (*Cg1*)

--Bruce G. Abedon and W.F. Tracy

Corngrass1 (*Cg1*) has a grasslike appearance when highly expressed due to retention of juvenile traits in distal vegetative organs. Variation in *Cg1* expression has been attributed to modifying loci as well as instability at the *Cg1* locus. Analysis of segregation ratios for high and low expression *Cg1* segregants in 11 families and three genetic backgrounds indicates the presence of two modifiers that reduce the level of *Cg1* expression.

Eleven families segregating for *Cg1* were created in two inbred (De811 and Mo17) and one hybrid (B73xMo17) backgrounds. Two, four, and five families were evaluated for De811, Mo17, and B73 x Mo17, respectively. The number of backcross generations was three for De811 and four for Mo17 backgrounds. The hybrid was made by crossing B73 to *Cg1* plants that were the result of three generations of backcrossing to Mo17. The source of *Cg1* was the Maize Genetics Cooperation stock 310D. Selfed *Cg1* plants were used to initiate the backcross program in De811 and Mo17. The final number of plants per family varied from 29 to 178.

After anthesis, the number of *Cg1* and normal segregants was determined for each *Cg1* family. *Cg1* plants were divided into two phenotypic classes (high and low expression) for each background. For De811 and B73 x Mo17 backgrounds, *Cg1* plants were classified as highly expressed if they had narrow leaves, multiple tillers (>1), and inflorescences that were more than 50% vegetative. In the Mo17 background, *Cg1* expression is normally more reduced than in other backgrounds. Consequently, in this background, highly expressed *Cg1* plants had narrow to intermediate width leaves, few tillers (<1), and inflorescences that ranged from 50% vegetative to fully reproductive but with only one central spike. In all backgrounds, plants with low expression had slightly reduced to normal width leaves, few tillers (<1), slightly reduced to normal inflorescences, and juvenile epicuticular wax sectors occurring on a greater number of transition leaves than inbred or hybrid recurrent parent controls. This last criterion reduced the chance of near-normal *Cg1* plants being misclassified as normal.

All data were analyzed by chi-square analysis. In order to determine if segregation was occurring normally in each *Cg1* family, we tested a 1:1 segregation ratio for *Cg1*/+ and +/+ progeny. In order to test and distinguish between one and two gene models for modifying loci, we tested 1:1, 3:1, and 1:3

segregation ratios for *Cg1* high and low expression classes. Contingency chi-square tests were also performed for the combined data from all *Cg1* families.

Segregation of *Cg1*/+ and +/+ progeny fit a 1:1 ratio for each *Cg1* family analyzed individually as well as for the combined data among families (Table 1). In addition, the contingency chi-square for this segregation was not significant (Table 1), indicating homogeneity among families. These results indicate that segregation occurred normally in the *Cg1* families used in this study.

Table 1. Segregation of *Cg1*/+ and +/+ progeny in 11 families and 3 genetic backgrounds.

Genetic background	Family	# of plants *Cg1*/+	# of plants +/+	1:1 segregation Estimated[a] X^2
B73 x Mo17	A	19	13	1.13
	B	32	35	0.13
	C	12	17	0.86
	D	22	13	2.31
De811	E	85	93	0.36
	F	55	55	0.00
Mo17	G	26	33	0.83
	H	28	19	1.72
	I	24	31	0.89
	J	32	24	1.14
	K	25	30	0.45
Total		360	363	0.01
				9.83[b]

[a] X^2 at P 0.05, 1df= 3.84; P 0.01= 6.64

[b] Contingency X^2 (at P 0.05, 10df= 18.31; P 0.01= 23.21)

The contingency chi-square test for segregation of high and low *Cg1* classes was significant (p<0.01), indicating non-homogeneity among families (Table 2). Consequently, segregation ratios for high and low *Cg1* expression classes were tested on an individual family basis.

All families fit hypothesized segregation ratios (Table 2). Families A, E, G, and H fit a 1:1 segregation ratio while families B, C, D, F, I, J, and K fit a 1:3 segregation ratio. In order to determine if the non-significant deviation from hypothesized ratios was due to sampling error resulting from small sample size in some families, data from families fitting a 1:1 segregation ratio (families A, E, G, and H) were pooled to form Group 1 and data from families fitting a 1:3 segregation ratio (families B, C, D, F, I, J, and K) were pooled to form Group 2. Group 1 and Group 2 fit a 1:1 and 1:3 segregation ratio, respectively. Since all families were derived from the same *Cg1* source, these results indicate that all families segregated in hypothesized ratios.

Table 2. Segregation of high and low *Cg1* expression in 11 families and 3 genetic backgrounds.

Genetic background	Family	# of plants Low expression	# of plants High Expression	Segregation Estimated[a] X^2 1:1	1:3	3:1
B73 x Mo17	A	10	9	0.05	7.74	5.07
	B	9	23	6.13	0.17	37.50
	C	2	10	5.33	0.44	21.78
	D	6	16	4.54	0.06	26.73
De811	E	40	45	0.29	22.06	35.39
	F	13	42	15.29	0.05	77.39
Mo17	G	14	12	0.15	11.54	6.21
	H	16	12	0.57	15.43	4.76
	I	5	19	8.17	0.22	37.56
	J	10	22	4.50	0.67	32.67
	K	7	18	4.84	0.12	29.45
Group 1[c]		80	78	0.03	55.37	50.03
Group 2[d]		52	150	47.54	0.06	261.39
						26.13[b]

[a] X^2 at P 0.05, 1 df = 3.84; P 0.01 = 6.64

[b] Contingency X^2 (at P 0.05, 10 df = 18.31; P 0.01 = 23.21)

[c] Pooled data from families A, E, G, and H

[d] Pooled data from families B, C, D, F, I, J, and K

The results from families B, C, D, F, I, J, and K indicate the presence of two unlinked, dominant, modifying genes that together reduce *Cg1* expression. Data from families A, E, G, and H indicate the presence of one dominant modifying locus. Since all families were derived from the same source of *Cg1*, one might conclude that the 1:1 segregation ratio for families A, E, G, and H, resulted because one of the modifiers in families B, C, D, F, I, J, and K was fixed while the other was segregating. Due to the genetic structure of these families, we feel that self progenies are required to confirm the results of this experiment. These progenies are being generated in the greenhouse.

MANHATTAN, KANSAS
Kansas State University

Additional loci for rust resistance

--Scot Hulbert and Jeff Drake

Common rust resistance genes have been mapped to three genomic regions in maize: The *Rp1* area on 10S, which includes genes designated *Rp5* and *Rp6* and spans two or more cM; the *Rp3* locus on chromosome 3, and the *Rp4* locus on 4S. We have recently identified a gene from the hybrid 'Golden King' which segregates independently of these three areas.

Golden King was determined to carry two dominant genes for rust resistance (Hulbert *et al.* Plant Dis. 75:1130, 1991). One of these was phenotypically identical to *Rp1-A* and mapped to the *Rp1* locus. The other provided resistance to a unique spectrum of rust pathotypes and segregated independently of the *Rp1* genes. We have now determined that this gene also segregates independently of the *Rp3* and *Rp4* loci and propose the *Rp7* designation. Test crosses of the following F_1's with *Rp7* segregated as two independent genes:

Rp3-F X *Rp7*	216:65 (res.:susc., pathotype KS1) X^2 3:1 = 0.52, P > .25
Rp3-C X *Rp7*	141:52 (pathotype KS1) X^2 3:1 = 0.39, P > .50
Rp4-B X *Rp7*	103:32 (pathotype OH1) X^2 3:1 = 0.12, P > .50

An F2 family from the *Rp4-B* X *Rp7* cross also segregated as expected for two dominant genes: 169 resistant: 8 susceptible to pathotype OH1; X^2 15:1 = 0.90, P > .25. Since the *Rp7* locus segregated independently of the *Rp1*, *Rp3* and *Rp4* loci, it represents a fourth locus controlling dominant resistance to common rust in maize.

The *Rp7* gene confers high levels of resistance to ten of the 12 rust pathotypes in our collection. It does not provide resistance to pathotype HI1 from Hawaii, or KS2 from Kansas. In resistant reactions, most fungal infections result in small (usually < 1 mm) necrotic spots. Some limited sporulation occurs in some environments, and the pustules are usually surrounded by necrotic rings.

We have also identified a simply inherited resistance from the line CG13 which was obtained from A. L. Hooker's stocks at the

University of Illinois. Preliminary analyses of crosses with this line have indicated a unique inheritance for this resistance. F2 progeny from the cross CG13 X H95 segregated 125 resistant:136 susceptible to rust pathotype IN1. The level of resistance in the resistant seedlings was very high, so scoring was unambiguous. Instead of the expected 3:1 or 1:3 ratios expected for single dominant or recessive genes, the segregation fit a 1:1 ratio (X^2 = 0.46, P>.25). F3 families from 17 susceptible F2 individuals bred true for susceptibility. F3 families from eleven of the resistant F2 individuals all segregated for resistance. Analysis of an additional 23 random F3 families also failed to identify any that were homozygous resistant; eleven families were completely susceptible and 13 segregated for resistance. Segregation of resistance in F3 families was similar to that of the F2, closely fitting a 1:1 ratio. The combined ratio for 22 F3 families that segregated for resistance was 662:681 resistant: susceptible. Four of the resistant F2 plants were also backcrossed to the susceptible H95 parent. Each of these backcross families also segregated 1:1, resistant:susceptible (combined ratio = 132:140). The inheritance model we will test is that of a single locus where only the heterozygote confers resistance.

The resistance from the CG13 line is effective against only two of the rust pathotypes in our collection and is, therefore, unlikely to provide effective resistance in the field. It provides a very high level of resistance to pathotype IN1, and an intermediate level of resistance to IA1.

Association of a lesion mimic phenotype with certain *Rp1* gene combinations

--Gongshe Hu and Scot Hulbert

The complex nature of the *Rp1* rust resistance locus allows certain combinations of *Rp1* genes to be recombined into the *cis* configuration (Saxena and Hooker, P.N.A.S. 61:1300, 1968; Hu and Hulbert, Genome 37:742, 1994). We have constructed a number of different recombinants carrying two or three tightly linked *Rp* genes. Such 'compound' *Rp1* genes can now be manipulated together as single genes. Two different recombinants (*Rp1-DJ4* and *Rp1-DJ46*) were identified which carried both *Rp1-J* and *Rp1-D*. Progeny from these recombinants, which were segregating for the compound genes, also segregated for a mild lesion-mimic phenotype. None of the parental lines showed the phenotype. In addition, all of the plants in the segregating families with noticeable necrotic or chlorotic spots carried the compound gene. Several lesion-mimics have previously been identified which are thought to have resulted from mutation or recombination events at *Rp1* (Pryor, TIG 3:157, 1987; Hu et al. unpublished). It is, therefore, possible that certain combinations of 'wild type' *Rp1* genes may contribute to a lesion-mimic phenotype. The experiment reported here was meant to determine the relationship between the *Rp1-DJ* resistances and the lesion-mimic phenotype.

The *Rp1-DJ46* and *Rp1-DJ4* compound genes were crossed twice to three inbred lines, H95, A188 and W23. Plants carrying the *Rp1* genes were then self-fertilized and the resulting BC1 F2 families were planted in the field. Individual plants in each family were self-fertilized, at which time they were rated for lesion mimic phenotypes. Ratings for individual plants are based on the percentage of leaf area on the lower leaves that was covered by chlorotic or necrotic spots (Table 1); for instance, '0' indicates no spots and '2' indicates 20% of the area of the lower leaves was covered by lesions. The *Rp1* genotypes of the individual plants were subsequently determined by progeny testing. Plants designated 'RR' or 'Rr' in Table 1 carry the compound gene while those designated 'rr' are homozygous for the *rp1* allele of the recurrent parent.

Table 1. Distribution and severity of lesion-mimic phenotypes in BC1F2 families segregating for *Rp1-J* + *Rp1-D* recombinants in different genetic backgrounds.

Les	*Rp1-DJ46* in three genetic backgrounds			*Rp1-DJ4* in two backgrounds	
Rating	A188	H95	W23	A188	H95
10	---	---	---	3 RR, 3 Rr	---
9	1 RR, 1 Rr	1 RR	----	2 RR, 1Rr	---
8	1 Rr	1 RR	----	4 Rr	2 RR
7	1 RR	---	----	1 Rr	1 RR, 1Rr
6	1 Rr	2 RR	----	---	1 Rr
5	1 RR	---	----	1 Rr, 1rr	1 Rr
4	3 Rr	1 Rr	1 Rr	1 rr	1 Rr, 1 rr
3	2 RR, 2 Rr	1 Rr	2 Rr	---	1 RR, 1 Rr, 1 rr
2	2 Rr, 2 rr	1 Rr	2 Rr	2 rr	2 RR, 4 Rr, 1 rr
1	1 Rr, 1 rr	6 Rr, 1 rr	3 RR, 1 Rr, 2 rr	---	---
0	4 Rr, 4 rr	1 RR, 6 Rr, 1 rr	2 RR, 4 Rr, 2 rr	2 rr	4 Rr, 3 rr

Examination of the data reveals two points. First, there is an apparent correlation between the *Rp1-DJ* compound gene and a lesion-mimic phenotype. In the A188 and H95 backgrounds, 28 out of a total of 96 plants were rated as '6' or higher and all these carried the *Rp1-DJ* compound gene. Second, the expression of the phenotype is dependent on other genetic factors besides *Rp1*. This is indicated by individuals that carry the *Rp1* compound gene but do not show the phenotype in each of the families. It is also apparent when comparing the severity of phenotypes in the different genetic backgrounds. The expression of the lesion-mimic phenotype is very weak in the W23 background as compared to the A188 or H95 background.

MEXICO CITY, MEXICO
CIMMYT

Old maize and ancient DNA

--Robert Bird, Natasha Bohorova, Diego Gonzalez de León and David Hoisington

CIMMYT's Applied Biotechnology Laboratories have begun a multifaceted project to study the relationships between 600 year old maize from the north coast of Peru and a wide range of traditional races from Peru and elsewhere. ARQUEOBIOS, an organization dedicated to studying archaeological remains of biological materials, associated with the Universidad Nacional de Trujillo, is in charge of coordinating the field work. The ears, kernels and cobs come from habitation and ceremonial sites of the Chimú empire. We plan to use various methods to determine molecular diversity within the ancient maize and to compare this diversity to that in modern maize. The actual methods will depend upon the quantity and quality of the DNA extracted from the ancient embryos. One can predict that no modern maize will prove to be very close to this, but that 8-10-rowed Sabanero Harinoso of the nearby highlands, or Mochero of the same area of the coast will be the closest. According to predictions based on archaeological patterns, the race Cuzco may relate more to Chimú flours than to more geographically proximal races.

A second facet of the study is to see whether there is any sign of life in this ancient material, evidenced by reaction to vital staining, by functioning physiological systems or, just maybe, by cell division. Central to such an effort is the fine-tuning of embryo

rescue techniques. We are using as test material some reserve seed from earlier generations of accessions in the CIMMYT Maize Germplasm Bank which has been saved for just such studies. We expect to get revival of at least a significant percentage of seed in samples that have low germination -- certain combinations of nutrients, hormones, light and temperature are promising. Hopefully the results will show that one can retrieve germplasm without resort to tissue culture, an inefficient procedure.

Netzahualcoyotl teosinte: a new population near Texcoco

--Robert McK. Bird

Sometime in the last four decades, teosinte appeared a few kilometers southwest of Texcoco (state of Mexico, Mexico). Today it is a flourishing, though limited, population creating problems for local farmers. I first saw it last summer while traveling the new Texcoco-Mexico City highway across what used to be Lake Texcoco -- it was abundant along the flanking fence for about two kilometers. Since then during weekend outings I have collected it at many places in the area from Colonia Netzahualcoyotl (a.k.a. Boyeros) north to San Andrés Riva Palacio.

Plants of this teosinte are 1.0-3.0 m tall and have 3-9 ear branches; rarely is there a tiller or branch low on the stem. The uppermost ear branch usually is at the penultimate leaf-bearing node. The next-to-last ear branches vary greatly -- 2-95 cm long. The longer branches have terminal tassels; these and the main tassel vary in the abscission of their rachids, 40-100%. Ear rachids from one typical plant range 5.4-8.0 mm long, 2.5-3.9 mm wide and 2.9-5.2 mm thick (rachid weight: 40-108 mg). In the population ear rachid weights vary considerably: the range of means for 4 plants with especially small rachids is 64-75 mg, while large-rachid plants range 124-136 mg.

There is some evidence of mutual introgression with maize. I and friends have collected some very small ears, each with a balance of teosinte and maize cob traits. There are some earlets on teosinte-like plants with two spikelets per segment, reduced abscission, and/or thickened cob rachises. Associated maize ears are often quite small with small, flinty kernels.

There is one collection of Netzahualcoyotl teosinte in the CIMMYT Maize Germplasm Bank (accession 13588), from San Felipe, collected by H. G. Wilkes and T. A. Kato Y. in 1992. J. Jesús Sánchez G. and Lorenzo Ordaz S. (*El Teocintle en Mexico*, 1987) cite two collections from this population in the INIFAP (Mexican national agricultural) germplasm bank, one made by them and one by John Doebley. All have been classified as part of the Chalco race, but some plants and ear segments seem too small for that race. I will place several samples of seed in the INIFAP and CIMMYT germplasm banks.

I have found scattered plants on the shoulder of the Texcoco-Vera Cruz highway and in the center of Texcoco. Wilkes, Sánchez and Ordaz say that teosinte in the Valley of Mexico occurs exclusively in maize fields, but in several places Netzahualcoyotl teosinte is found on the shoulders of roads and the banks of irrigation ditches in competition with grass and large broadleaf weeds. In some of the small ejido corn fields with teosinte there is five times as much teosinte as maize, making for poor and difficult harvests. Some corn is cut as forage but most is cut later for fodder; teosinte is often included. Because most of the resulting manure is placed on fields, teosinte seed are being spread over an ever wider area. Informants know of this cycle and add that irrigation water is a factor. Several informants, including Sr. Guadalupe Guevara (retired *maestro* of CIMMYT's Maize Germplasm Bank), have told me that teosinte appeared in their fields within the last 8-40 years. Perhaps it came in a cow brought from Chalco or Michoacán, maybe with purchased fodder. Maybe it spread from an observation plot at one of the local agricultural institutions.

Sequencing to test models of maize domestication

-- Robert McK. Bird

Fifteen years ago (MNL 53:53-54, 1979; Biotropica 12:30-41, 1980) I proposed the outline of what can be called the Intersectional Introgression Model, that (1) early gardeners in Mexico began cultivating a wild maize with miniature maize-like ears, a form of which was found in caves near Tehuacán, SE of Mexico City, (2) the early domesticate was spread to SE Guatemala and by chance hybridized with and was introgressed by *Zea luxurians*, and (3) various allelic combinations in the hybrid swarm allowed both the rapid cultural selection of widely varying maize races and the natural selection of weedy populations which became Mexican annual teosinte (MAT). Tehuacan Early Domesticated (TED) differed from the wild form perhaps only by having more adherent kernels. The oldest date measured directly on TED cobs is 2750 B.C., 2300 years more recent than originally estimated (uncalibrated; Long et al., Radiocarbon 31:1035-1040, 1989), so the hybridization of (2) may have occurred shortly before 2000 B.C., later than I earlier proposed because of the new dates. Wilkes presented a very similar model, also in 1979, but he proposed that the introgression came from *Z. diploperennis*. Archaeobotanical evidence closely fits the model: the oldest well dated maize samples from North, Central and South America are all fully distinct morphologically from teosinte, and there are still no archaeological specimens of teosinte or maize-teosinte hybrids which date securely before 1100 B.C. (a few teosinte rachids and some Mexican cobs which appear to be introgressed by teosinte and which are thought to be earlier need to be directly radiocarbon dated). The traits which maize and MAT share are also explained -- both derive from one hybrid swarm.

Since 1976 several well-known models have been defined wherein no maize was present until man selected mutant or transmutant forms of MAT, the latter without basic genetic change. Now that John Doebley, Adrian Stec and associates are isolating key genes responsible for some of the morphological differences between maize and teosinte, we are closer to being able to choose among evolutionary models by using nucleotide sequencing to define quantitatively the differences between individual key alleles of species in section *Luxuriantes* and of various maizes and Mexican annual teosintes in section *Zea*.

If maize were a domesticated version of MAT, with *no* introgression from *Z. luxurians*, the nucleotide sequences of alleles of the key differentiating loci within sect. *Zea* should reflect somewhat over five millennia of evolution since domestication. For four such loci, *a* to *d*, alleles should fit a pattern like *a1 b1 c1 d1, a1 b1 c1 d3, a2 b2 c2 d2, a2 b2 c2 d4*, etc. in maize, and *a3 b3 c3 d3, a4 b4 c4 d4, a4 b4 c1 d3*, etc. in MAT. The numerals indicate two maize and two teosinte lineages out of many, as well as some recent introgression between maize and MAT (e.g., *a1 b1 c1 d3*). The ancestor of domesticated maize and present MAT would have had alleles *a0 b0 c0 d0*. Alleles in sect. *Luxuriantes* would be separated by several millions of years of evolution from all comparable alleles in sect. *Zea*.

On the other hand, if the more complex Intersectional Introgression Model is correct, many alleles at the key loci in the MAT races and some of those in present-day maize should have been introgressed from *Z. luxurians*. Such alleles (capitalized) would now reveal a pattern like *A5 B5 C5 D5* in *Z. luxurians*, *a3 B7 C7 d3, a4 B8 C8 D8*, etc. in MAT, and *a1 b1 c1 d1, a1 b1 c1 D6, A8 b2 c2 d1*, etc. in maize. The 5, 6, 7 and 8 indicate lineages of *Z. luxurians* alleles which evolved relatively independently after the hybridization, as in maize of highland South America and northeastern U.S. or in teosinte of west Mexico. There may even be some *A5 B5 C5 d1* teosinte somewhere in Central America, a maize-introgressed *Z. luxurians*. Remember that *Z. diploperennis* might substitute for *Z. luxurians* in these discussions.

There could be small sets of *Z. luxurians* alleles in most maize, those alleles which had been "teosinte-negative" in the teosinte and selected in the hybrid progeny because they contributed positively to early maize races. Doebley and Stec (Genetics 134:566, 1993) found some alleles with unexpected effects which might be examples -- e.g., the teosinte allele at a QTL which they locate on arm 7L has a positive effect on CUPR, the number of cupules per rank. Such alleles could explain much about the explosive evolution of maize since 1100 B.C. with extraordinary increases in the size and number of various organs. Moreover, observations by Donald Robertson (MNL 57:6, 1983) and others, that progeny from the maize x *Z. luxurians* cross demonstrate a notable increase in mutation rate, suggest that activation of transposons by the proposed wide hybridization could have increased the potential for novel traits. Perhaps footprints left during various episodes of transposon insertion/exsertion will be revealed by sequencing, and these could help define phylogeny.

The Intersectional Introgression Model provides a parsimonious explanation of Sarah Hake's 1980 results using DNA hybridization (MNL 56:90-92, 1982). In full-genome hybridizations involving *Tripsacum laxum*, U.S. dent inbreds W64A and B37, Ladyfinger popcorn (LP) and several teosintes, she found a divergence of 8.8% (% divergence = °T_m celsius) between W64A and *T. laxum* (probably a reflection of many millions of years of separation), 3.7-4.2% divergence between maize (LP and 2 inbreds) and sect. *Luxuriantes* (*Z. diploperennis* and *Z. luxurians*), and 2.7-3.3% divergence between Balsas teosinte and maize (B37 and LP). The 2.7-3.3% is *not* well explained if maize and MAT separated only several thousand years ago -- perhaps 0.002-0.008% would be expected. If Balsas teosinte contains many *Z. luxurians* alleles, the 2.7-3.3% figures make sense, although Balsas-*Z. luxurians* divergence is 3.6%. Maize (W64A and B37)-Chalco teosinte divergence was only 0.9%, perhaps reflecting the long-term introgression between maize and teosinte that many think has been occurring in the Chalco area; Balsas-Chalco divergence was 0.2%.

MILAN, ITALY
University of Milan

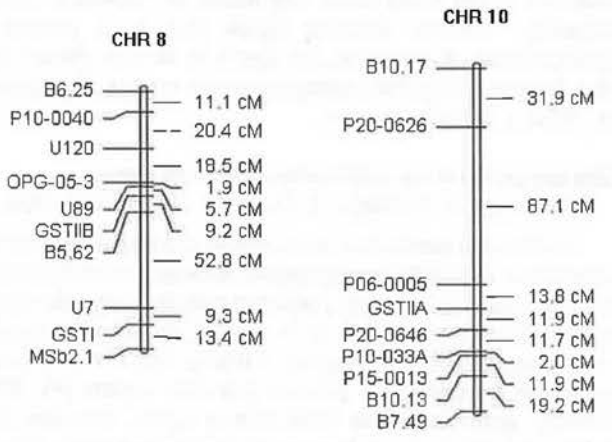
Molecular analysis and mapping of gst genes

--L.Rossini, C.Frova, M.E.Pè and M.Sari Gorla

The glutathione(GSH)-glutathione-S-transferase(GST) system is a detoxification system capable of inactivating many toxic molecules. It is present in a wide variety of organisms, from mammals to insects and plants, in which it is involved in conferring tolerance to different classes of herbicides, including s-triazines, acetanilides and thiocarbamates. In maize, three gst genes (*gst1*, gstII, gstIII) (Shah et al., Plant Mol Biol 6:203-211, 1986; Moore et al. Nucleic Acids Res. 14:7277-7235,1986; A. Greenland, personal communication) have been identified and sequenced. [Ed. note: Temporary symbols pending clarifications of gene relationships.] They belong to a gene family and show extensive sequence homology at both DNA and protein levels. They encode for four isozymes which are active as dimers: GSTI, GSTIII and GSTIV are homodimers of 29, 26 and 27 kDa subunits respectively, while GSTII is a heterodimer of 29 (same as GSTI) and 27 (same as GSTIV) kDa subunits.

We found evidence that the number of maize gst genes and isozymes is higher, some isoforms being tissue-specific while others are expressed in most tissues. In particular, the two major bands detectable in electrophoretic enzyme assay (native PAGE) are expressed in roots, leaves and scutella. By screening numerous maize genotypes we have identified two inbred lines, B37 and B83, lacking these two bands in all tissues. Analysis of null/+ and null/null F1s indicated that the null mutations are recessive and allelic in the two inbreds. In order to identify to which of the characterized GST isoforms these bands correspond, we have carried out Northern experiments to analyze the transcription pattern of gstI and gstII in roots of normal and of the two null lines. As specific probes we have used a cDNA clone of gstII (kindly provided by A. Greenland) and a probe including the first exon of gstI synthesized via PCR on the basis of the published sequence information. gstII transcript was detected in all genotypes, while gstI mRNA was absent in the null lines. These data indicate that a single gene, gstI, controls the expression of the two isoforms. We tentatively identify them as GSTI (29 kDa homodimer) and GSTII (29/27 heterodimer).

The genomic organization of the two genes was investigated by Southern analysis: *gst1* is a single copy gene, while the hybridization pattern of gstII suggests the presence of a duplicated gene. We have mapped these genes by RFLP analysis using an F2 population of 149 individuals, previously characterized for 110 molecular markers. *gst1* was placed on the long arm of chromosome 8, while the two putative gstII loci, gstIIA and gstIIB, were mapped on chromosome 8 (70cM from *gst1*) and on chromosome 10 respectively (Figure).



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Molecular characterization of *o2-T*: a mutant allele at the *opaque2* locus

--B. Lazzari, P. Ciceri, R. Carzaniga, A. Genga, F. Faoro and A. Viotti

Several wild-type and mutant alleles of the *opaque2* gene have been characterized at the DNA, RNA and protein levels (Bernard et al., Plant Mol. Biol. 24:949-959, 1994); one of them, referred to as *o2-T* and present in the W64A genetic background, has been further investigated by sequence analysis. This allele derives from the spontaneous mutation of the *O2-wl* allele of the W64A wild-type line. Both the wild-type and mutant alleles have been amplified by RT-PCR starting from total RNA of immature endosperm at 15 days after pollination. The amplified fragments have been cloned in pBluescript (Stratagene) and sequenced.

The sequence analysis of these two clones shows 99% homology in the 5' end of the sequences, but a deletion in the mutated allele causes a frame shift which leads to a stop codon, in accordance with data obtained by sequence analysis of the corresponding genomic regions of both *W64A+* and *o2-T*. The deduced truncated protein has a molecular mass of 24kD and an isoelectric point of 4.37 (data obtained by computer analysis). This protein could correspond to the 40 kD polypeptide detected by Western blot analysis, as the real and apparent MW of the O2 protein on denaturing gels do not correspond, due to the presence of particular amino acid sequences in the N-terminus of the O2 polypeptides which could alter the normal correlation between the relative mobility and the molecular mass (Bernard et al., Plant Mol. Biol. 24:949-959, 1994; Liang et al., Electrophoresis 13:346-353, 1992). The predicted isoelectric point is in accordance with experimental data, as the o2-T polypeptide analyzed by IEF migrates as a single band with a pI of about 4.3.

Electron microscopy examination of immunogold labeled sections of maize endosperm shows that the *o2-T* polypeptide is located in the cytoplasm, while the wild-type is 90% in the nucleus. It is important to note that the truncated protein lacks the basic domain structure that, in the case of Opaque-2 protein, has a double function of DNA binding and nuclear localization. The basic domain, in fact, contains the NLS B bipartite structure, which seems to be the major factor responsible for Opaque-2 nuclear targeting. Another targeting signal, NLS A, is present in Opaque-2 and o2-T proteins, but seems to be less efficient than NLS B in redirecting the polypeptide to the nucleus (Varagona et al., Plant J. 5:207-214, 1994).

Zein expression is not restricted to endosperm tissue

--A. Genga, R. Carzaniga, B. Lazzari, F. Faoro and A. Viotti

Several zein genes have been cloned and sequenced and the comparison of their 5' flanking regions showed a common feature: the presence of a distal and a proximal dual promoter with respect to the ATG codon, indicated as P1 and P2, respectively. Analysis of the expression in transgenic *Petunia hybrida* of chimeric constructs containing the different promoter regions (P1, P2 or P1+P2), upstream of the GUS coding region, revealed GUS activity not only in the endosperm of transformed seeds, thereby retaining their original tissue specificity, but also in anthers, particularly in the tapetal cell layers (Quattrocchio et al., Plant Mol. Biol. 15:81-93, 1990). This observation prompted the authors to investigate in more detail the situation in maize inflorescences, where Northern blot analysis revealed the presence of a 1.9 kb mRNA, indicating a preferential transcription from the distal promoter P1 (Quattrocchio et al., ibid.).

In order to detect even low levels of zein gene expression in tissues other than endosperm, total RNA was extracted from male inflorescences of the line A69Y at different developmental stages, reverse transcribed using oligo-dT primer and McMuLV reverse transcriptase, and amplified by PCR using primers corresponding to consensus sequences derived from a comparative analysis of the 5' and 3' regions of about 10 different structural zein genes. Before reverse transcription total RNA was treated with RNase-free DNase, to remove any possible contaminant trace of genomic DNA present in the RNA sample. Subsequent Southern blot analysis, using as probes a mixture of sequences coding for different light zein (ZL) chains, showed the presence of amplification products, confirming that transcripts for this zein class are present in developing tassels. The same analysis on transcripts corresponding to the heavy zein (ZH) chains is being performed at present.

To assess for the presence of zein polypeptides, Western blot analysis was carried out with anti-zein sera on protein extracts obtained from developing male inflorescences. Our results indicate that polypeptides belonging to both classes, ZH and ZL, are synthesized in these organs, even if in much smaller amounts than in endosperm. Their presence has also been confirmed by immunogold detection on anther sections containing pollen grains at different developmental stages. Zein polypeptides were localized in the ER of the inner layer of the tapetum.

In order to determine if the expression of zein genes in male inflorescences is regulated in a manner similar to or different from endosperm, the first step was to investigate whether the regulatory gene *opaque2* is also expressed in this tissue. This was done by RT-PCR on total RNA, previously treated with RNase-free DNase, using two sequences internal to the *O2* coding region as primers. Amplification products were analyzed by Southern blot with the entire coding sequence as probe. The expected length of the amplified sequences is 680 bp for products obtained from mRNA molecules and 1027 bp for those derived from genomic DNA, as three introns are present in this region. Only the expected smaller fragment was amplified when DNase-treated RNA samples were used, thereby indicating that not only structural zein genes, but also the regulatory *O2* gene, are expressed in male inflorescences.

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Chromosome characterization of the race Jala of maize

--A. Lòpez R., T.A. Kato Y. and F. Castillo G.

Jala is one of the most astonishing types of maize because of its ear size. Its geographic distribution is restricted to the small valley of Jala, located in the southern part of the state of Nayarit in Mexico; however, we do not know how variable it is across the valley regarding its chromosome knob constitution, since there was only information on six plants from a single collection: NAY72 (McClintock et al., Colegio de Postgraduados, Chapingo, Mexico, 1981). Based on two types of available collections, those recently

collected (1988) by the Colegio de Postgraduados, and the old ones conserved in the CIMMYT germplasm bank which were collected between 1944 and 1968, a cytological study was carried out with two objectives: 1) to determine whether different populations of the race are relatively uniform or not; and 2) to determine if the race has changed its chromosome knob constitution with time.

Six accessions of the race Jala were studied: three collected by the Colegio de Postgraduados in 1988 (CP88-2, CP88-5 and CP88-6) from three different farmers in the valley, and three from the CIMMYT germplasm bank (NAY 6, NAY 53 and NAY 208) originally collected in the same place in 1944, 1952 and 1968, respectively. The chromosome knob constitution of 20 plants per accession was determined by examining pachytene meiotic microsporocytic cells with the usual propionic carmine squash method for 21 knob positions. Knob frequency, weighted by knob size, was subjected to principal component and cluster analyses by SAS (1985) procedures.

The cumulative variance accounted by the first two principal components was 65% and the dispersion of the characterized accessions determined by them is shown in Figure 1. An

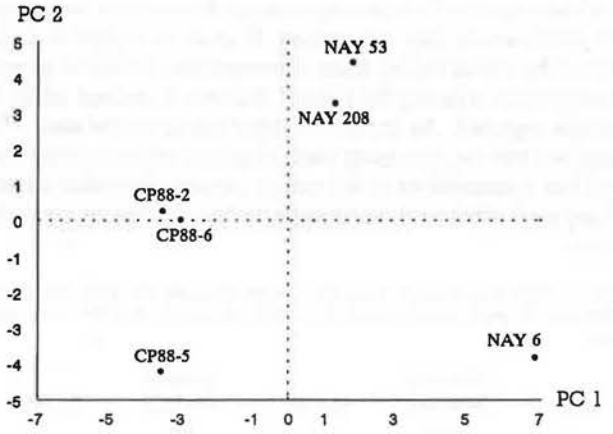

Figure 1. Dispersion of six maize Jala accessions, on the plane defined by the first two principal components, based on the weighted frequencies of four knob sizes in 21 positions in the chromosomes, and the presence of supernumerary elements.

outstanding feature is the close grouping of the CP88 collections far from the NAY collections, of which NAY 6 is the most distant. This dispersion is due mainly to the higher positive values in the eigenvector of the first principal component, given by the frequencies of medium knobs in $1S_2$, $2S_1$, $2L_1$, $4L_1$, $6L_2$ and $7L_1$, of the small ones in $8L_2$ and 7S, of the large knobs in $2S_1$ and $5L_1$ and frequencies of the abnormal 10 chromosomes, all of them with higher frequency in the NAY collections, especially in NAY 6. The dispersion is also due to the high negative values in the eigenvector of this principal component associated with the frequencies of the small knobs in $2S_1$, $3L_1$, $5L_1$ and $6L_3$, of the large ones in $2L_1$ and $4L_1$, and of the B chromosomes, with higher frequencies in the CP88 collections. The second principal component provides distances that separate two collections from the third one in each of the groups CP88 and NAY, given by the higher negative values in the eigenvector, which are associated with the frequencies of large knobs in $1S_2$, $7L_1$, $8L_1$ and 9S, of the medium knob in 7S and of the small one in $1L_1$. The previously described dispersion is in close agreement with the dendrogram obtained from the cluster analysis shown in Figure 2. These results provide evidence that in

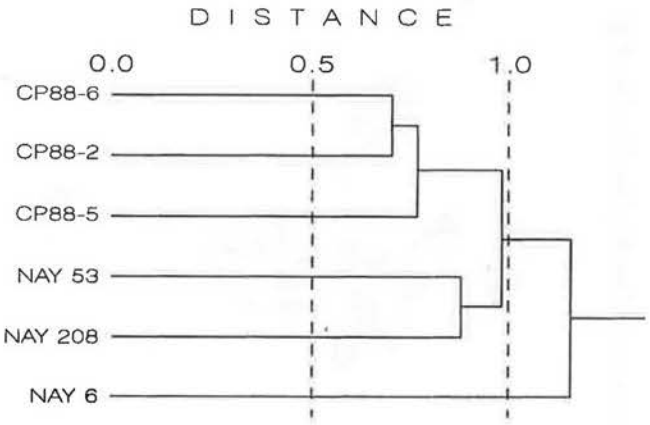

Figure 2. Dendrogram obtained from the cluster analysis of weighted chromosome knob frequencies of six maize Jala accessions.

spite of the restricted distribution, this race has some degree of variation among populations within the limits of the small valley of Jala.

If each of the four knob sizes in the 21 positions in the chromosomes of maize is considered as one allele in 21 corresponding loci, the average genetic diversity per collection was calculated, with the results shown in Table 1.

Table 1. Values of genetic diversity for six collections of the race Jala of maize, obtained from the frequencies of four knob sizes in 21 chromosome positions.

Collection	Procedence	Date of Collection	Genetic Diversity	Average by Origin
CP88-5	CP	1988	0.3485	
CP88-6	CP	1988	0.3524	0.3566
CP88-2	CP	1988	0.3672	
NAY 6	CIMMYT	1944	0.2897	
NAY 53	CIMMYT	1952	0.3216	0.318
NAY 208	CIMMYT	1968	0.3439	

$D = 1-(1/m)\sum_l \sum_u p_{lu}2$; where $l = 1$ to 21 loci and $u = 1$ to r alleles; m = 21 x 4

It can be observed that the values obtained for the CIMMYT collections are lower than those for the CP88 collections and of the former the lowest is for NAY 6 (collected in 1944), followed by NAY 53 (1952) and NAY 208 (1968). The lower diversity could be attributed to phenomena as those independently mentioned by Salhuana and by Crossa (CIMMYT Proc. Global Maize Germplasm Workshop, 29-38 and 159-154, 1988) of genetic drift that may occur during the seed increases by the germplasm banks due to the small size of seed samples used. Other factors that might have contributed to this differentiation are the occurrence of physical or biotic environmental modifications in the region during the time considered, the introgression of genes from introduced new varieties, the reduction in the crop farming area of this race, or because of changes in the selection criteria made by new generations of farmers.

Additionally, the chromosome constitution of the race Jala was analyzed with respect to 21 Mexican races whose data were taken from McClintock et al. (Colegio de Postgraduados, Chapingo, Mexico, 517 p., 1981) with the aim of ascertaining the relations among those 22 races, based on the same statistical methods. Race Jala (JL) was placed near the Tuxpeño complex: Tuxpeño, Vandeño and Celaya (TX, VA, CE) as shown in Figure 3, due mainly to the high frequencies of medium knobs in the long arms of chromosomes 2, 3, 4, 5, 7, 8, 9.

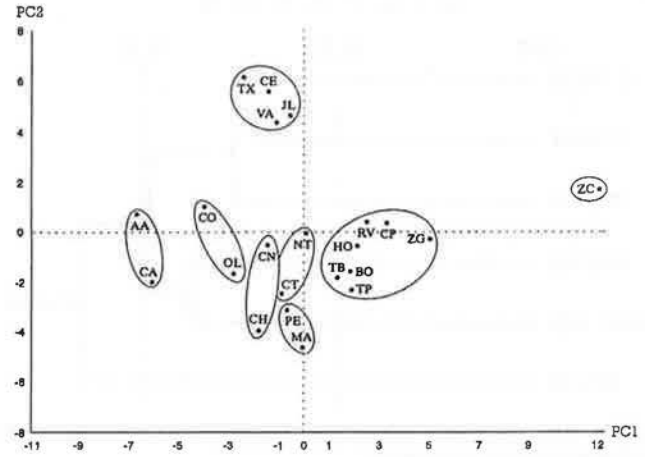

Figure 3. Clustering of 22 Mexican maize races in the plane determined by the first two PC, from the analysis of the weighted frequencies of four knob sizes in 21 chromosome positions, and those of supernumerary elements.

Chromosome knobs and chiasma frequency

--T. Angel Kato Y.

There is no information about the variation in chiasma frequency among and within populations of maize. So, an exploratory study was done by analyzing cytological material already in existence in the laboratory. It is known that chromosome knobs influence genetic recombination and special chromosomes (abnormal 10 and B type) affect both the genetic recombination and the chiasma frequency (Kikudome, Genetics 44:815-831, 1959; Rhoades and Dempsey, Genetics 53:989-1020, 1966; Ward, Can. J. Genet. Cytol. 18:479-484, 1976).

Cytological materials of six collections were selected: three with high and three with low knob content belonging to three races: Cacahuacintle (Mexico 7 and one of unknown origin); Chalqueño (Mexico 208) and Conico (Guanajuato 30, Mexico 3 and Oaxaca 377). Of these collections, the one of Chalqueño and two of Conico (Guanajuato 30 and Oaxaca 377) have many knobs (an average of 30.2% of the known knob positions), and the other three possess few knobs (6.6%). Guanajuato 30 was known to have plants with abnormal chromosome 10 and B chromosomes. These collections had been grown in the experimental field of the Colegio de Postgraduados located in Montecillo, State of Mexico during the years of 1990, 1991 and 1993.

Data on chiasma frequency were obtained from 40 meiotic metaphase I cells from anthers of at least two flowers for each plant, using the traditional propionic-carmine squash technique. The number of plants analyzed varied for the different collections (Table 1). Total and terminal chiasmata were registered for each bivalent in every cell; by differences between these data the interstitial chiasma frequencies were obtained.

The mean total, interstitial and terminal chiasma frequencies between the two groups of collections were not statistically different; however, among and within the collections these frequencies were significantly different. These results seem to indicate, first, there is not any general effect of chromosome knobs on the formation of chiasmata and if there is any influence, probably this is related to the local chiasmata distribution on specific bivalents or chromosome arms where the knobs are localized; second, it seems that different plants have distinct chiasma formation capacity, depending on their particular genotype (Table 1).

Table 1. Mean total chiasma frequencies per plant in six maize collections.

	Many knobs			Few Knobs		
Plt.	Mex. 208	Oax. 377	Gto. 30	Mex. 3	Mex. 7	Cac. O.D.
1	20.07	20.50	22.07	19.90	21.40	20.35
2	19.87	20.17	21.87	19.85	21.32	20.25
3	19.77	20.02	21.30	19.72	20.65	20.07
4	19.70	20.00	21.07	19.67	20.47	20.02
5	19.67	19.85	20.92	19.57	20.30	19.87
6	19.62	19.55	20.92	19.42	20.07	19.87
7	19.45	19.55	20.90	19.25	20.07	19.72
8	19.45	19.47	20.87	19.25	20.05	19.62
9	19.40	19.42	20.60	19.15	19.30	19.55
10	18.87	19.27	20.37	19.10	19.15	19.52
11	18.72	19.15	20.15	18.77	19.10	19.50
12	18.55	19.15	19.97	18.47	19.07	19.42
13	18.27	19.07	19.92	16.72	19.00	19.20
14	18.37	19.77			18.57	18.82
15	18.35	19.67				18.72
16		19.47				18.70
17		19.37				18.45
18						17.42

The collection that showed the highest mean total chiasma frequency was Guanajuato 30. In this collection two plants were found having a heterozygous abnormal chromosome 10, and one plant with 1B and three with 2B chromosomes. Since it is known that these special chromosomes increase the chiasma frequency of the plants where they are present, it would be logical to expect that all the plants having these chromosomes should be grouped among those showing the highest chiasma frequency within the sample analyzed. As shown in Table 2 this is not the case. This suggests that the increasing effect of special chromosomes has a limit that is established by the specific chiasma formation capacity of any plant which, in turn, depends on its genotype, as concluded above.

Table 2. Relationship between mean total chiasma frequency per plant and abnormal chromosome 10 and B chromosomes in the collection Guanajuato 30 of the race Conico of maize.

Plant	Mean Total Chiasmata/Plt.	Stat. Dif.[1]	Abnormal Chrom. 10	B Chrom.
1	22.07	a	Het.	
2	21.87	ab		2B
3	21.30	abc		
4	21.07	bcd		
5	20.92	bcde	Het.	
6	20.92	bcde		2B
7	20.90	bcdef		
8	20.87	cdef		
9	20.60	cdefg		
10	20.37	defgh		
11	20.15	defghi	2B	
12	19.97	efghi	1B	
13	19.92	fghi		
14	19.77	ghi		
15	19.67	ghi		
16	19.47	hi		
17	19.37	i		

[1]Tukey test $P < 0.05$

When the correlations were calculated between the total and the interstitial chiasma frequencies in each of the six collections analyzed, it was found that the three collections with high knob frequencies also showed high r values (0.93 in Oaxaca 377, 0.81 in Guanajuato 30 and 0.73 in Mexico 208). In those with few knobs, these values were much lower (Cacahuacintle of unknown origin, 0.61, Mexico 3, 0.58 and Mexico 7, 0.48). One problem in these results is that not all the collections were planted in the same year and field plot, so they grew under different environmental conditions and probably are not completely comparable. However,

there are two cases that, separately, seem to show the aforementioned relationship to be valid since the collections comprising each case were grown in the same year and in contiguous plots: 1) Mexico 208 with many knobs, and Mexico 3 and 7 with few knobs, grown in 1991; and, 2) Guanajuato 30 with many knobs, and Cacahuacintle, of unknown origin, with few knobs, planted in 1993 (Table 3).

Table 3. Correlations between total chiasma frequencies and the corresponding interstitial and terminal ones in different maize collections.

Planting		Chiasma Frequency Correlation Total Interstitial		Total Terminal	
Year	Collection	r	r^2	r	r^2
1990	Oaxaca 377	0.93	0.86	-0.85	0.73
1993	Guanajuato 30	0.81	0.66	0.57	0.33
1991	Mexico 208	0.73	0.53	0.02	0.0004
1991	Mexico 3	0.58	0.34	0.36	0.13
1991	Mexico 7	0.48	0.23	0.10	0.01
1993	Cacahuacintle (unknown origin)	0.61	0.37	-0.30	0.09

In spite of these limitations, a tentative hypothesis was developed for explaining these results: the chromosome knobs, depending on their size, interfere to a certain degree with the chiasma terminalization, probably by slowing down the process. It is known that the chromosome knob heterochromatin is of the constitutive type that usually appears compacted in all cellular stages. Possibly this characteristic of the knobs causes the interference of chiasma terminalization by presenting some resistance for the homologous chromatids united by a chiasma to become "free" and permit their separation.

Even though the described data and their interpretations might be interesting, there is a need for further well-planned experiments that provide more acceptable evidence to strengthen the proposed ideas.

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Salt resistance of plants selected in vitro

--S.N.Larina, Yu.I.Dolgykh and Z.B.Shamina

Cell cultures initiated from immature embryos of A188 were cultivated on MS growth medium with 1% NaCl. Viable salt-resistant calli were selected during 6 subcultures.

Part of the salt-resistant embryogenic calli was used for plant regeneration. Regenerated plants had decreased viability and fertility. After 4 subcultures, the regeneration ability of callus has been lost.

Subculturing on NaCl 1%	1	2	3	4
No. regenerants in vitro	18	23	9	4
No. of regenerants in soil	4	8	3	4
No. of fertile regenerants	4	0	1	3

The progeny of 8 regenerated plants were tested for salt resistance in hydroponic chamber. The solution for plant cultivation was based on MS salts with addition of NaCl 0.5%. Results are shown in Fig.1. Number of subcultures is shown in parentheses.

Among R1 plants, the best germination and viability was in regenerants from 1 subculture. Germination in 0.5% NaCl was 100%. Viability was considerably better than that of the A188 line. Increasing the number of subcultures did not lead to improved salt resistance of regenerated plants.

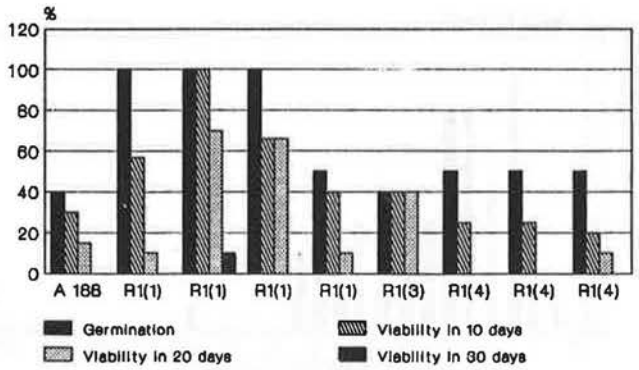

Figure 1. Viability of R1 plants on NaCl 0.5%.

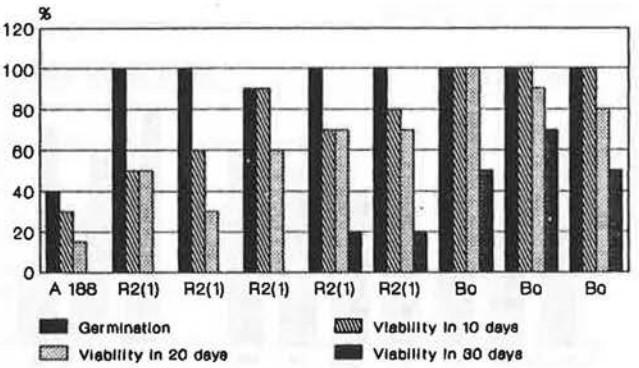

Figure 2. Viability of R2, BC plants on NaCl 0.5%.

Seeds from one of the R1(1) regenerants were used for obtaining R2 plants and backcrosses of R1 X A188. Salt resistance was analysed. The results are represented in Fig. 2. All the seeds germinated 2 - 2.5 times better than the control line. Backcrosses have demonstrated the best results.

Development of drought-resistant plants via tissue culture

--Yu. I. Dolgykh and S.N.Larina

The effectiveness of cell selection in vitro for development of drought-resistant plants was evaluated. Embryogenic callus was initiated from immature embryos of F1 hybrids *Chi31* x *Cateto S.G.* and *Chi31* x *Tuxpeno Norteno*, which had been produced by Dr. V.S. Shcherbak (Krasnodar Research Institute of Agriculture). The tolerant cell lines were isolated on MS medium supplemented with 25% PEG (6000), and plants were regenerated from the selected calli.

The drought-tolerance of regenerated plants (R0) and their progeny after self-pollination (R1 and R2) were tested using physiological methods. Since resistance to dessication and thermoresistance are the important components of drought resistance, the water retaining ability under drying and the rate of leaf damage under high temperature (50C) were determined. The water-retaining ability had been shown to correlate with drought tolerance (McCaig and Romagosa, Crop Sci., 31:1583,1991).

The R0 regenerants demonstrated less average water loss under drying than the initial plants. Plants with high and average water-retaining ability were prevalent among regenerants, and the most sensitive forms were eliminated (Fig.1). R1 and R2 were

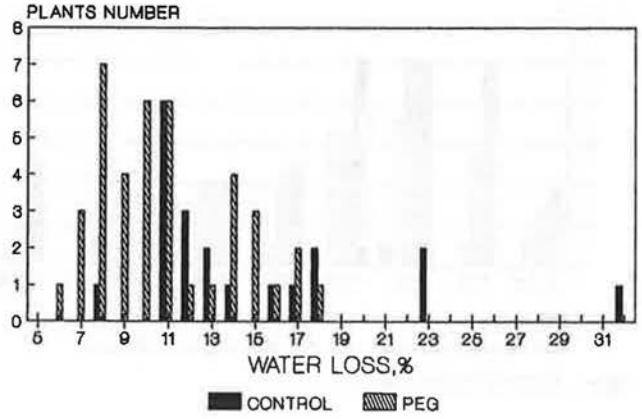

Figure 1. Water loss under drought.

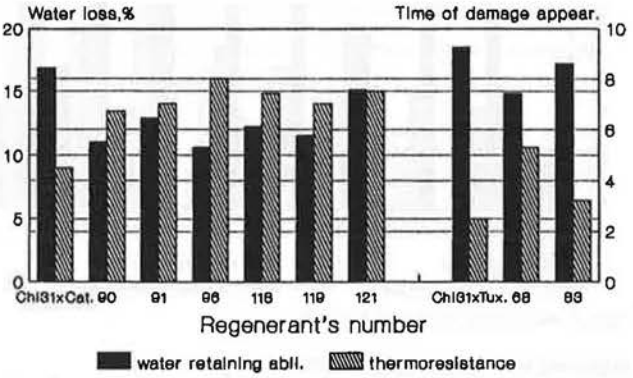

Figure 2. Water retaining ability and thermoresistance of R1 plants at tasseling.

tested under drought at the 8 and 10-11 leaf stages, and during tasseling, 20-30 plants from the progeny of every regenerant were analysed. Average thermoresistance of the selected plants exceeded control values in all families of regenerants through all stages of ontogenesis. The water-retaining ability of R1 was increased in comparison with nonselected plants only during tasseling (Fig.2). Three best families analysed in R2 also demonstrated increased water-retaining ability and thermoresistance. Limits of variability of these traits among selected plants differed from controls (Table).

Plants	Water loss, %	Time damage appears, minutes
Chi31 x Cateto	11.8 - 29.5	5 - 15
R 2 # 90	13.1 - 23.1	9 - 26
R # 91	7.6 - 16.6	9 - 25
R # 119	7.3 - 13.4	4 - 24

While silking of the initial plants was delayed under drought, shedding and silking of the selected regenerants took place simultaneously.

Our results reveal that tissue culture of maize can be used for selection of drought-resistant plants.

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More about associations of the growth-regulating genes

--Emil Khavkin and Ed Coe

Previously (MNL 68:61, 1994) we have hypothesized that genes for plant growth and development are associated into functionally significant clusters. Sixteen initially delineated clusters, 10-30 cM long (2 to 3 bins of the molecular map), comprise almost 90% of already mapped growth genes. The majority of recently reported QTLs for plant architecture, growth pattern and yield (Ajmone-Marsan et al., Maydica 39:133, 1994; Beavis et al., TAG 83:141,1991; Doebley and Stec, Genetics 134:559,1993; Edwards et al., TAG 83:765, 1992; Higginbotham, MNL 65:65, 1991; Stuber et al., Genetics 132:823, 1992; Zehr et al., TAG 83:903, 1992), growth and differentiation *in vitro* (Armstrong et al., TAG 84:755, 1992; Cowen et al., TAG 84:720, 1992; Wan et al., TAG 85:360,1992), pollen growth (Sari-Gorla et al., Heredity 69:423, 1992) and ABA accumulation (Quarrie et al., Russ. J. Plant Physiol. 41:565,1994) map within these clusters. We consider this evidence a corroboration to the cluster model.

It is no wonder that QTLs for plant height precisely match naked eye polymorphisms (NEPs) for reduced plant stature. More impressive are those map segments where the closely mapped NEPs expressing various growth and developmental manifestations coincide with QTLs for growth traits that are widely different. QTLs co-mapping with NEPs do not always correspond by their apparent functions.

These cases are exemplified by bin 1.11 (*d8, kn, phy1* and *tb1)* and QTLs for plant height, architecture and yield; bin 1.12/13 (*ij2, py2, rd1, tls1, tlr1, ts6,* and *vp8)* and QTLs for embryogenesis in callus culture, plant height, ear and tassel development and days to pollen; bin 2.06 (*ba2* and *ts1)* and QTLs for plant height, leaf length, ear architecture and embryogenesis *in vitro*; bin 3.05 (*abp1, d2, rd3, rg1, te1, tp3, ts4* and *vp1)* and QTLs for pollen growth, plant growth and architecture; bin 3.06/07 (*ba1, bif1, lg2, na1, les14, les17* and *yd2*) and QTLs for embryogenesis *in vitro,* days to pollen and the numerous parameters for plant architecture and yield substantiated by reports from three laboratories; bin 4.03-4.04 (*la1, orp1, st1, tga1* and *ts5)* and QTLs for pollen germinability and many traits of plant shoot and inflorescence architecture; bin 5.06/07 (*br3, bv1, gl17, nec3, nec6, td1, vp2* and *vp7*) and QTLs for pollen growth, plant architecture, yield and days to tassel; bin 9.03/04 (*baf1, d3, gl15* and *les8*) and QTLs for embryogenesis *in vitro,* pollen growth, plant height and yield, and ABA synthesis. In these cases, the choice between polygenic interactions or monogenic pleiotropic expression of physiologically linked traits will provide a prospective means to dissect the complex traits and to search for key genes working as developmental switches.

Several additional clusters can be presumed that combine the mapping positions for single NEPs and for several molecular probes, co-segregating with growth-related QTLs. The most promising locations are bin 2.01 (*lg1* and QTLs for plant height, ear length and days to pollen); bin 4.07/08 (*tu1, nec5* and numerous QTLs for plant architecture, growth and yield reported from four laboratories); bin 6.01/02 (*rgd1, si1, wi1* and QTLs for

embryo differentiation in callus culture, pollen tube growth, days to tassel and days to pollen, plant height and architecture).

Two map segments devoid of NEPs for growth and development contain the major QTLs for embryogenic callus initiation, plant regeneration, plant yield and apparently ABA synthesis (bin 9.05/06); and for plant growth, days to pollen and plant yield (bin 10.07/08). Using the hybridization probes for the already known growth genes to map these segments of the genome could help reveal the genes manifesting these quantitative traits.

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Distribution of haploids on the ear

--K.R. Sarkar, B.M. Prasanna and P. Gayen

In a controlled pollination with silk trimming, the ovules at the bottom of the ear have longer silk length than those at the tip and the pollen tubes have to traverse a longer distance. Hence, there should be greater likelihood of getting haploids at the bottom half of the ear. In a survey of over 161 ears obtained from *ACR* x *C-I*, a haploidy-inducer line cross, this expectation was not realized. On the contrary, the tip half showed a haploid frequency of 7.49 percent (11739 kernels) as against 3.77 percent (11345) kernels in the bottom half. In a further study of the same cross, an additional 122 ears were longitudinally divided into three zones, top, middle and bottom, and the haploid cases were scored (Table 1). The results were interesting. While the top third of the ears

Table 1. Distribution of haploids in ears from *ACR* x *C-I* crosses.

		Haploid (%)		
ACR culture	No. Ears	Top	Middle	Bottom
6732	14	10.23	6.93	3.54
6733	9	14.93	10.93	7.04
6735	15	9.65	3.97	3.71
6736	15	8.94	4.91	4.16
6738	8	7.36	5.14	4.05
6744 D	8	6.78	2.72	2.12
Subtotal	69	9.69	5.76	3.94
6666	12	5.95	2.63	5.01
6729	18	8.25	5.12	6.39
6734	17	5.92	3.46	4.39
6744 E.4	6	8.80	2.01	3.08
Subtotal	53	6.90	3.68	5.04
Total	122	8.52	4.91	4.40

always yielded the highest frequency (8.52%), there was no substantial difference between the middle (4.91%) and the bottom thirds (4.40%). However, some families showed higher frequencies at the bottom portion while others had more haploids in the middle zone. The same trend was also observed when one common male was used to cross on seven female cultures. The reasons behind such occurrences are not immediately clear; perhaps the sequence of fertilizations of ovules on the ear tip and differential silk factors are responsible for this uneven spatial distribution of haploidy cases on the cob. This observation will, however, help in further studies on the mechanism of the origin of haploids.

Cytomixis in monoploids

--P. Gayen and K.R. Sarkar

Cytomixis is no longer a fixational artifact (Basaviah and Murty, Cytologia 52:69-74, 1987). This phenomenon has been reported mainly in pollen mother cells of chromosomally imbalanced plants in a number of crop species (Nettancourt and Grant, Cytologia 29:191-195, 1964). In maize, cytomixis was reported in pollen mother cells of a triploid and its hyperploid progeny (McClintock, Genetics 16:175, 1929) and in a trisomic for chromosome 5 (Premchandran et al., Curr. Sci. 57:681-682, 1988).

We observed extensive cytomixis in two haploid plants, with 83% and 51% cells being involved in cytoplasmic connections and chromatin transfer. These plants were earlier treated with 0.06% colchicine at seedling stage (6-7 day old) by the cotton wad method, but doubling of chromosome number did not occur. Cell clusters having two to six cells were more common. The highest number of cells involved in a cluster was 48. In large clusters, cells with no chromosome (→) to as many as 27 chromosomes (⇒) were found to be present (Fig. 1). The number of cell-to-cell cytoplasmic connections ranged from one to four. In some cases a single cell was seen to be connected with five to six nearby cells (see figure).

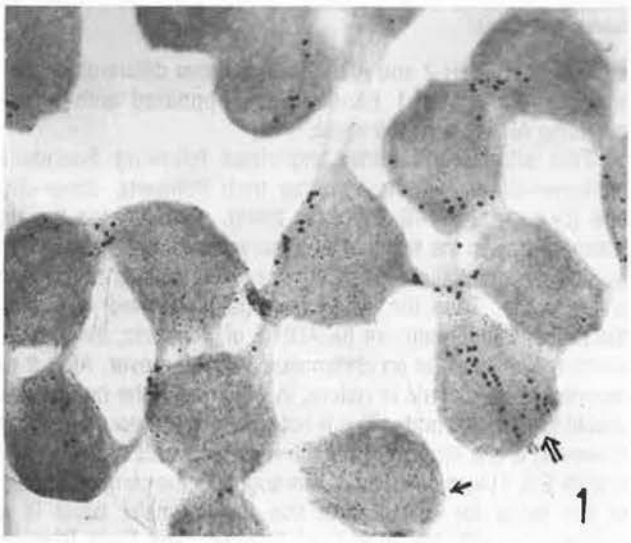

Fig. 1. Part of a large cluster showing: cell with no chromosome (→), cell with 27 chromosomes (⇒) and multiple cytoplasmic connections.

Alcohol dehydrogenase isozyme variants

--Jashir K. Madan, P. Gayen and K.R. Sarkar

ADH isozyme patterns were analyzed in scutellar tissues from maize seeds soaked 12-60 h, comprising 120 diploids carrying *A C R* markers and 42 haploids derived from the same stock. Tris-soluble samples were run in an alkaline starch gel system applying 20 mA current for 4 h.

All haploids and diploids homozygous for ADH showed either a fast moving densely stained ADH-2 band along with the fast moving faintly stained ADH-1 band or a slow moving ADH-2 with the slow moving ADH-1 band (nomenclature according to Scandalios, Biochem. Genet. 3:37-79, 1969). Heterozygous diploids, on the other hand, showed a dimeric nature for ADH-2 giving three clear bands (fast moving, intermediate and slow moving in 1:2:1 intensity), and a monomeric nature for ADH-1 giving only two bands (fast moving and slow moving in 1:1 intensity) (Fig. 1). In none of the cases, out of a total of 162 seeds

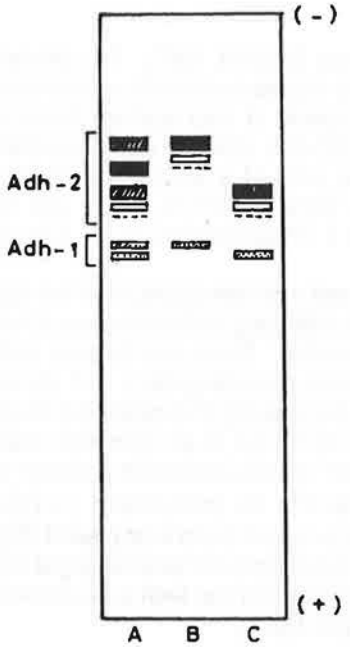

Figure 1. Schematic representation of ADH isozyme variants in scutellum of maize. A = diploid, heterozygous for both the loci; B, C = diploid, homozygous for slow moving and fast moving bands for both the loci.

analyzed, did ADH-2 and ADH-1 bands move differentially, i.e., a slow-migrating ADH-1 band always appeared with a slow-migrating ADH-2 and vice versa.

This situation is better explained following Scandalios' monomer-dimer hypothesis rather than Schwartz' dimer-dimer one (Schwartz, PNAS 56:1431, 1966). Considering the tight linkage between the two loci as observed in the present study as well as by Scandalios, these appear to be located on the same chromosome. Thus, the ADH-1 band (faintly stained) observed in the present study can not be ADH-2 of Schwartz, the locus for which is known to be on chromosome 4. Moreover, ADH-2 was reported to be dimeric in nature, in which case the heterozygote should have three bands. This is not observed in even a single case. However, a low intensity band co-migrating with ADH-2 (anodal to both S & F) was observed. This suggests the compound nature of the locus for ADH-2, but this low intensity band is not synonymous with the ADH-C of Schwartz and Endo (Genetics 53:709-715, 1966).

The pattern of bands was very consistent over a period of 12 to 60 h of soaking. No significant difference in band intensity was observed either at different periods of soaking or between haploids and diploids as measured by densitometry.

Thus, it needs to be confirmed further whether ADH-2 and ADH-1 in the present study are determined by two separate (linked) loci on chromosome 1 producing dimeric and monomeric complexes.

Presence of active *Ubiquitous* in a 'high haploidy' *ACR* line

-- B.M. Prasanna and K.R. Sarkar

A 'high haploidy' *ACR* line was screened for the presence of three mobile element systems--*Uq*, *Mrh* and *Cy*. This *ACR* line is characterized by its high haploid induction frequencies when used as a female parent in crosses with the 'Stock 6' *C-I* male lines. For instance, in Hyderabad winter 1993-94, the haploid frequencies ranged from 0.07 to 20.43% (2397/56640 haploids; av. 4.23%) out of a total of 115 plants tested. In Delhi monsoon 1994, the same line yielded 155 haploids out of 3759 total colorless kernels (av. 4.12%) (unpublished results from our laboratory).

About 6-8 plants each of the *ACR* line were crossed as female parent with the MERL (Mobile Element Receptor Line) series: *a-ruq*, *a-mrh* and *bz-rcy*. The resultant F2 (colored/mottled) plants were selfed and the F2 progeny were evaluated for the presence/absence of mutability of the alleles of the MERL series (Table 1).

Table 1. Classification of progeny kernels following self-pollination of *ACR*/MERL plants.

Origin	No. Ears.	Cl	Spotted*	cl	Total K.
ACR/a-ruq	27	4174	1275	726	6175
ACR/a-mrh	8	954	2**	352	1308
ACR/bz-rcy	14	3364	0	1026	4390

*'Spotted' refers to the characteristic mutabilily pattern of the element system
**One-spot *Mrh* kernels

Uq mutability pattern was observed in each of the 27 ears scored, clearly indicating that at least one active copy of the element is present in the *ACR* line. On the other hand, one-spot kernels for *Mrh* could be observed in only two of the eight ears scored. The limited sample size does not allow us to conclude about the frequency and nature of *Mrh* element activity, whereas absence of *Cycler* mutability in the 14 plants tested suggests the lack of *Cy* in the material tested. It shall be interesting to analyze the role of the active *Uq* element, if any, in the haploid occurrence of this high haploidy *ACR* line in crosses with the *C-I* inducer males in subsequent seasons.

The transposable element in *R-mb:cc* - Composition and test for homology with some two-element systems

-- B.M. Prasanna and K.R. Sarkar

The newly discovered *R-mb:cc*, a mutable allele derived from the *R-marbled* (*R-mb*) genetic stock in our laboratory (MNL 67:87-88, 1993; MNL 68:63-64, 1994) is further studied to: (i) genetically analyze the nature of the element system operating at this allele; and (ii) test the homology, if any, of the element in *R-mb:cc* with the known element systems in maize.

(i) Composition of the element system in *R-mb:cc*: The *R-mb:cc* allele frequently gives rise to completely colorless somatic revertants as well as kernels with very light striping (1-2 concentric colored stripes/rings). Kernels of the former category gave rise to some germinal revertants with completely colorless expression; a majority of the 'colorless' category as well as the 'very light striped *R-mb:cc*', on progeny testing, yield a high percentage of colorless kernels with 1-2% very light striped kernels. This is in stark contrast to the segregation profiles of medium/heavily striped kernel categories, where the frequency of occurrence of colorless/very light striped kernels is substantially low (1-2%). As per the procedure to verify the composition of the element system (one/two-element system), we crossed various categories of *R-mb:cc* with *ACR* lines and the F1 progeny (colored/mottled) were selfed. If the transposable element system in *R-mb:cc* is a two-element system, the F2 progeny should segregate in a 12:3:1 (colored: mb:cc: colorless) ratio; segregation in a 3:1 (colored: mb:cc) ratio would indicate a one-element system. Results from this study are presented in Table 1.

Table 1. Segregation profiles in the F2 progeny obtained by selfing *ACR/R-mb:cc* plants.

Culture	Origin*	No. Ears	F2 progeny segregation				No. of ears fitting	
			Cl	mb:cc	cl	Total	3:1	12:3:1
6903	*A C R* x cc1	8	1595	630	266	2491	4	1
6904	*A C R* x cc2	4	599	334	156	1089	-	-
6905	*A C R* x cc2	2	269	112	99	480	-	-
6906	*A C R* x cc2	6	1489	437	84	2010	6	3
	Subtotal	12	2357	883	339	3579	6	3
6907	*A C R* x cc3	1	158	42	17	217	1	1
6908	*A C R* x cc3	5	907	381	117	1405	2	2
6909	*A C R* x cc3	12	2090	966	991	4047	-	-
6910	*A C R* x cc4	7	1231	445	226	1902	1	1
6911	*A C R* x cc4	2	383	134	71	588	1	-
	Subtotal	27	4769	1968	1422	8159	5	4
6912	*A C R* x cc6	3	478	222	27	727	1	1
6913	*A C R* x cc6	5	812	561	169	1542	-	-
	Subtotal	8	1290	783	186	2269	1	1
Total	55	10011	4264	2223	16498	16	9	

*cc1 = near colorless/very light striped *R-mb:cc*; cc2 = light striped; cc3 and cc4 = medium striped; cc6 = heavy striped

It can be observed that the F2 progeny defy either 3:1 or 12:3:1 segregations. For computing the 3:1 ratio, the mb:cc and colorless categories were pooled, on the basis that the colorless kernel category originated due to the transposable element activity in *R-mb:cc*. Also, wherever the segregation pattern fitted a 12:3:1 ratio, the 3:1 segregation for colored:mb:cc is naturally followed, but not vice versa, as is evident from Table 1. Significant also was the occurrence of ears where the segregation of colored:mb:cc fitted a ratio of 1:1. One plausible reason for the above observations may be the presence of the regulator component of the *R-mb:cc* element system in the *ACR* lines (6186, 6187 and 6188) used in crossing with *R-mb:cc*. Additional tests are being carried out to verify the composition of the element system in *R-mb:cc*.

(ii) Test for homology with other transposable element systems: *R-mb:cc* was also tested for its ability to induce mutability of heterologous responding alleles of some two-element transposon systems, the MERL series of which were generously provided by Ellen Dempsey, Peter Peterson, F. Salamini and G.F. Sprague. The crossing scheme adopted was as per the standard procedures. Plants homozygous for *R-mb:cc* with an active element were crossed by plants containing the appropriate responding allele (MERL) of the other element system but lacking the regulator element of that system. The resultant F1s were selfed or testcrossed and the F2/BC progeny were screened to detect mutability of the MERL allele. The results obtained are presented in the following table:

	En	*Uq*	*Mrh*	*Cy*	*Bg*
R-mc:cc/a-m(r)	(-)				
R-mb:cc/a-ruq		(+)			
R-mb:cc/c-ruq		(+)			
R-mb:cc/a-mrh			(-)		
R-mb:cc/bz-rcy				(-)	
R-mb:cc/o2-m(r)					(-)

Out of the five transposable element systems tested, only *Uq* (*Ubiquitous*) was found in the *R-mb:cc* genetic stock. Absence of *En* (*Enhancer*), *Mrh* (*Mutator* of Rhoades), *Cy* (*Cycler*) and *Bg* (*Bergamo*) clearly rules out the possibility of the regulatory elements of these systems in triggering the mutability pattern of *R-mb:cc*. On the other hand, presence of *Uq* does not necessarily provide evidence for its involvement. In the case of *Uq*, mutability characteristic of *Uq* indicated by + was found in all the 8 plants tested with the *a-ruq* allele, whereas mutability for *c-ruq* was found in 3/5 plants for which testing was carried out. The presence of *Uq* in only a few of the lines that carry the *R-mb:cc* phenotype suggests that *Uq* may not be an integral component of the *R-mb:cc* allele. Also, the presence of *Uq* in a 'near colorless' line from *R-mb:cc* suggests that *Uq* cannot substitute for the transposable element system in *R-mb:cc*. Additional tests are now being carried out to ascertain if there is any interaction of *R-mb:cc* with the components of *r-cu/Fcu* and *Ac/Ds* transposable element systems.

Paramutagenic property of *R-mb:cc*

-- B.M. Prasanna and K.R. Sarkar

The *R-mb:cc* allele, characterized by the presence of concentric colored stripes/rings on a colorless aleurone background, was genetically analyzed to determine the paramutagenic property, if any, in crosses with *R-r:standard* lines. The *R-marbled* (*R-mb*) allele, the parental allele of *R-mb:cc*, was recorded to display a paramutagenic behavior in association with *R-r* lines (Brink and Weyers, Proc. Natl. Acad. Sci. USA, 1053-1059, 1957), similar to that of its 'sister' pattern allele, *R-st*. Surprisingly, subsequent workers interested in the phenomenon of paramutation in maize paid less attention to the *R-mb* allele, in comparison with the *R-st* allele; consequently, literature on the paramutagenic property of *R-mb* is almost negligible. Although the phenotype of *R-mb:cc* is distinctly different from that of *R-mb*, we were interested in finding out if the derivative retains the paramutagenic property.

Various categories of *R-mb:cc* -- near colorless, light striped, medium striped and heavy striped -- were crossed with *R-r:standard* lines and the resultant progeny were selfed/sibbed. For comparison, the same *R-r:standard* lines were also crossed with the *R-mb* stock. Results from the F2 and subsequent generation (data not shown) clearly indicated the paramutagenic nature of *R-mb:cc*, in causing a reduction in the anthocyanin pigmentation intensity of the *R/R* kernels. To assess the reduction in pigmentation intensity, the resulting ears were assessed by a standard set of *R* kernels. Observation of paramutation in the progeny kernels aided us in concluding that *R-mb:cc*, possibly a 'change in state' from *R-mb*, retains the paramutagenic property despite a distinctly different variegation pattern. Interesting, perhaps, is the occurrence of modified *R/R* progeny kernels where a few sectors of mottling (with reduced pigmentation) were present in concentric circles of broken lines, resembling the characteristic variegation pattern of *R-mb:cc* (with solid, concentric circles/stripes originating from the germinal region). Progeny testing of such kernels ruled out the presence of the *R-mb:cc* allele. Although the reasons behind such occurrences are not clear, speculations can be made about the possible links between DNA methylation, transposable elements and paramutation.

Though the significance of paramutation in higher plants is now

increasingly realized, the exact mechanisms behind this puzzling phenomenon continue to elude geneticists. Intensive efforts by maize researchers are needed to provide clues to better understand this interesting phenomenon.

Search for the presence of active transposons in Indian maize germplasm and assorted genetic testers

-- B.M. Prasanna

The pervasiveness of genetically active mobile elements, such as *Uq* and *Mrh*, in maize breeding material as well as lines native to Mexico and South America, and the significance of such activity in maize breeding material, has been emphasized earlier by maize workers. Keeping this in view, a program is being carried out to screen the available Indian germplasm for the presence of active mobile elements. The material includes populations being used for the development of inbred lines, CM lines (Coordinated Maize inbreds, developed by the All India Coordinated Maize Improvement Project, redesignated as Directorate of Maize Research), assorted genetic stocks and a few entries from the North Eastern Himalayan (NEH) maize collection.

The crossing scheme to uncover the active regulatory elements in populations/lines/inbreds, as per standard procedures, involved crosses with the Mobile Element Receptor Lines (MERL), such as *c-ruq*, *a-ruq*, *a-mrh*, *a-m(r)*, *bz-rcy* and *o2-m(r)*. The colored F1s from crosses of lines x MERL were handled by selfing or backcrossing to the same MERL or a recessive line to uncover the specific recessive used with MERL. The exposure of any MERL to an active regulatory element triggers a spotting pattern in the resultant progeny. Salient results from this study are presented in Table 1.

The number of plants analyzed for each of the entries in this assay exceeded fifteen, except in cases of 1681 (for *c-ruq*) and CM 105 (for *a-ruq*) to obtain a 0.95 probability of detecting an element if it is present at a frequency of at least 0.1 (Sedcole, Crop. Sci. 17:667-668, 1977).

Table 1. Test for presence of active mobile elements in Indian maize germplasm and assorted genetic testers: (+) /(-) indicate presence/absence of active element in the material tested.

Maize line/population	MERL	Corresponding mobile element Uq	Mrh	En	Cy	Bg
Germplasm/Breeding Material						
M-18, NEH (Meghalaya strain) [1]	*c-ruq*	(-)				
S-38, NEH (Sikkim strain) [1]	*a-ruq*	(-)				
S-54, NEH germplasm (Sikkim strain) [1]	*bz-rcy*				(-)	
	a-mrh		(-)			
1672 Population [2]	*a-ruq*	(-)				
	a-mrh		(-)			
	a-m(r)			(-)		
	o2-m(r)					(-)
1681 Population [2]	*c-ruq*	(+)				
	bz-rcy				(-)	
	a-m(r)			(-)		
	o2-m(r)					(-)
CM 111, Inbred [2]	*c-ruq*	(+)				
CM 105, Inbred [2]	*a-ruq*	(-)				
Genetic Stocks						
g r-g, genetic tester [1]	*a-ruq*	(+)				
	a-mrh		(-)			
cl sh1 wx A1 A2 C2 R, genetic tester [3]	*c-ruq*	(+)				
Chr. 3L genetic tesler [3]	*bz-rcy*				(-)	
Chr. 7S genetic tester [3]	*c-ruq*	(-)				

[1] Source: Maize Genetics Section, IARI
[2] Source: Directorate of Maize Research
[3] Source: Maize Genetics Coop., USA

The investigation affirms the pervasiveness of the *Ubiquitous* element in maize germplasm in diverse geographic regions. Although the manner of conscious selection for homozygosity and uniformity should quickly eliminate active mobile elements from the inbred lines, it is surprising to find the presence of an active *Uq* in one of the two inbreds tested, CM 111, a commonly used inbred line in the Indian maize breeding programs.

The present study utilized only five of the many genetically characterized transposable element systems in maize and also a limited number of entries from the vast NEH germplasm collection. The manner of testing precluded the possibility of ascertaining whether individual plants that showed presence/absence of mutability for a specific element system such as *Uq* harbor any other known or unknown mobile elements. Also, further tests are needed to find out whether the mutability pattern uncovered in material such as CM 111 (for *Uq*) is due to the activation of the resident *Uq* or due to a change at the receptor element. The study is being continued, involving more Indian maize breeding material and NEH lines, to investigate the presence of active mobile elements and their influence in breeding programs.

A new cytotype in *Coix gigantea*

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

The genus *Coix* (tribe Maydeae, family Gramineae) is an Asiatic wild relative of maize. Spontaneous occurrence of aneuploidy and structural heterozygosity (Gayen, et al., MNL 68:68, 1994) is not a new event in this genus. *Coix gigantea* is one of the five species

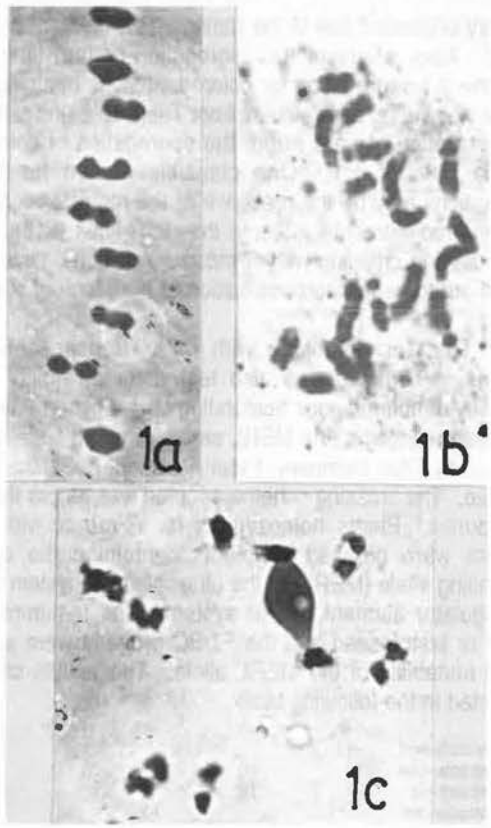

Fig. 1. (a) Nine bivalents in metaphase I; (b) Eighteen chromosomes in somatic metaphase; (c) Nine bivalents in diakinesis with two bivalents attached to the nucleolus.

known to be present in this genus. Cytologically this species is well known, with three cytotypes (2n=10, 2n=20 and 2n=40) on the basic number x=5 (Celarier, Cytologia 22:160-183, 1957; Goldblatt, Index to Plant Chromosome Numbers, 1984). Recently, a new stable cytotype with n=6 and 2n=12 has been reported in *C. gigantea* (Christopher and Jacob, Cytologia 56:265-268, 1991).

In a naturally occurring population of *Coix* from Talegaon, Pune (Coix-28), we observed chromosome number 2n=18 with more than 95% pollen fertility in all the 23 plants tested. The plants were meiotically checked for chromosome number and meiotic abnormalities, if any. In diakinesis and metaphase I (Fig. 1a), 9 bivalents were clearly observed. There was no meiotic abnormality at any stage of meiosis. Somatic chromosome number was also consistently found to be 2n=18 in all the root tips examined (Fig. 1b). Two pairs of chromosomes had secondary constrictions in mitotic metaphase. In diakinesis (Fig. 1c) two bivalents were attached to the nucleolus. Out of a total of 483 cells studied in diplotene and diakinesis in 23 plants, no additional nucleoli were observed in any cell.

Pollen viability under Delhi conditions

--B.M. Prasanna

Interesting observations on extended viability of pollen under specific conditions were made in MNL by Barnes and Walbot (MNL 67:106) and Burnham (MNL 67:101). I have attempted to ascertain the viability of maize pollen under Delhi conditions. The pollinating season in Delhi is characterized by medium to very high humidity (60-90%) with field temperatures ranging 32-40 C during the peak pollination period (last fortnight of August to first fortnight of September), with frequent occurrence of mild to moderate showers due to the southeast monsoon. Pollen viability under these conditions was analyzed by two methods: (i) pollinations in vivo of selected inbred lines and genetic testers; and (ii) in vitro germinability of pollen collected from the field.

(i) in vivo: Two sets of material were used for this purpose: (a) Two inbred lines (6914, an unreleased line, and MCU 508, a released line) carrying *y* (white endosperm) marker and CM 111, an inbred line with *Y* (yellow endosperm) as a male parent; (b) homozygous *R-mb:cc* lines as female lines in crosses with lines carrying *R-Navajo* (*R-nj*) marker in homozygous condition. In both cases, the female lines were detasseled, pollen was collected from the male parents at different timings of the day (8:00 AM, 11:00 AM, 3:00 PM and 6:00 PM) and fractionated in a glassine bag to exclude debris, and generous amounts of the sieved pollen were used for pollinations on at least 5-6 detasseled plants each (with similar silk age) for each treatment (day timing). Since *R-nj* displays full penetrance when transmitted through the male parent in crosses with *R-mb:cc* (unlike *R-mb:cc* which shows a drastic reduction in penetrance and expressivity when used as a male parent), *R-mb:cc* x *R-nj* crosses were expected to yield kernels with either nj+mb:cc or Navajo phenotypic expressions.

The results obtained (data not shown) from pollinations done in the last week of August indicated that the pollen of CM 111 retained a considerable amount of viability till the evening hours even on days with considerably high mid-day temperatures (35-40 C). This viability was further extended on days (August 28 and 29) which coincided with showers during the normal pollination period, since pollinations carried out even at 6:00 PM resulted in good seed set (with 281 av. kernels/ear vs. 368 av. kernels/ear control for pollinations done at 8:00 - 8:30 AM). In the case of *R-mb:cc* x *R-nj* crosses, extended pollen viability was observed on rainy days. However, on the normal days (clear sky with high temperatures and humidity), the average seed set was almost negligible when pollinations were done during the evening hours. The presence of the Navajo marker in the male parent aided in excluding the possibility of contamination.

(ii) in vitro pollen germination ability: Pollen germinability of CM 111 and MCU 508 was evaluated to ascertain the extent of germination in pollen collected at regular intervals (8:00 AM, 11:00 AM, 2:00 PM and 5:00 PM) from the field-grown plants. Although both CM 111 and MCU 508 germinate well *in vitro*, under normal conditions, in Walden's *in vitro* pollen germination medium, pollen germinability cannot be considered as an accurate measurement of the capacity to bring about fertilization. Therefore, this assay has been used only to supplement the observations made in vivo. The *in vitro* study was carried out on three successive days in the first week of September. Pollen germinability at different day timings differed significantly in the two inbred lines. A comparison of the percent change in pollen germination at successive timings (in comparison with the observation at 8:00 AM, when the pollen is expected to be fresh and mostly viable) revealed that a considerable amount of viable pollen can be obtained even at 2:00 PM and 5:00 PM in the case of CM 111, unlike MCU 508. The decline in pollen germination in MCU 508 was also sharper than CM 111. The above observation suggests that under certain climatic conditions, maize lines might show considerable differences with respect to pollen viability. Definitive reasons for this are difficult to offer. Probably, a combination of factors such as the genetic constitution of the line, tassel form (compact or lax) and, plausibly, anthocyanin coloration in plant parts may have a role to play.

Disease reaction of wild relatives of maize to *Erwinia* stalk rot and *Helminthosporium* leaf blight

--R.C. Sharma, M.M. Payak and J.K.S. Sachan

Erwinia stalk rot (ESR) caused by *E. chrysanthemi* p.v. *zeae* is a major disease, particularly in the plains and hilly regions of India. The disease occurs when the plants are about 40 days old. As the disease causes soft rot, the affected plants die prematurely.

Table 1. Reaction of *Coix*, *Trilobachne*, *Chionachne* and teosinte to *Erwinia chrysanthemi* p.v. *zeae* and *Helminthosporium maydis* of maize under artificial disease inoculations.

Pedigree	Origin	ESR (%)	*H. maydis* (1-5*)
Cultivated Coix-7	Nagaland	20.0	1.0
Cultivated Coix-8	Nagaland	80.0	1.0
Cultivated Coix-9	Nagaland	60.0	1.0
Cultivated Coix-11	Nagaland	20.0	1.5
Cultivated Coix-13	Nagaland	40.0	2.0
Cultivated Coix-14	Nagaland	60.0	1.0
Wild Coix-24	Matheran	0.0	1.0
Wild Coix-25	Pune	0.0	1.0
Wild Coix-28	Talegaon	0.0	1.0
Wild Coix-29	Dombivili	0.0	2.0
Wild Coix-30	Purander	0.0	1.0
Wild Coix-33	Almora	0.0	2.0
Trilobachne-1	Dang Forest	0.0	1.0
Trilobachne-2	Matheran	0.0	1.0
Chionachne-1	Western Ghats	0.0	1.0
Chionachne-3	Pune	0.0	1.0
Chionachne-5	Kolhapur	0.0	1.0
Teosinte-1	Guatemala	60.0	1.0
Teosinte-2	Mexico	100.0	1.0
Teosinte-3	Mexico	100.0	2.0
Teosinte-4	Mexico	60.0	2.5

*1 = no disease; 5 = maximum disease

Leaf blight caused by *Helminthosporium maydis* is the most widely distributed disease of maize not only in India but in all the tropical and subtropical regions of the world. Various collections of *Coix*, *Chionachne*, *Trilobachne* and teosinte were evaluated for these two diseases under conditions of artificial inoculations at I.A.R.I. farms during summer.

The data obtained were not only interesting but useful. All the collections of *Trilobachne* and *Chionachne* were highly resistant to both the diseases tested. Among *Coix* collections, cultivated ones showed a high degree of susceptibility to ESR, but wild Coix-25 (2n=10) showed immunity to both the diseases. In the case of teosinte also, susceptibility reaction to ESR was observed (Table 1). The information may serve a useful purpose for transfer of disease resistance traits to cultivated maize.

NORMAL, ILLINOIS
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A study of the progeny of monosomic-10 plants

--R. Lane, D. F. Weber, and M. C. Schneerman

Sears (Chromosoma 4:535-550, 1952) reported that univalent chromosomes frequently misdivide during meiosis in wheat producing telocentric chromosomes. We have been analysing progeny of maize monosomics (which have a univalent chromosome in each meiotic cell) to determine if chromosomal variants might also be recovered from them.

We (Teissonniere, Weber, and Schneerman, MNL 68:70, 1994) previously studied progeny of monosomic-4 maize plants. Monosomic-4 plants that had *su* on their single chromosome 4 were crossed by a *Su/Su* stock producing F1s that were *Su/su*. These F1s were reciprocally testcrossed. 15 of 49 F1s testcrossed as male parents produced ratios that were significantly different from a 1:1 ratio. Thirteen of these had more sugary than non-sugary kernels and 2 had fewer sugary than non-sugary kernels. However, none of these 49 F1s testcrossed as female parents had ratios that differed significantly from a 1:1 ratio. The fact that these differences were observed when the F1 progeny were testcrossed as males but not as females is consistent with the possibility that chromosomal abnormalities might have been present in these plants.

Here we report the results of crosses involving monosomic-10 maize plants utilizing markers on both arms of this chromosome. The monosomic-10 plants were produced utilizing the *r-X1* system and selected as described by Weber (Maize Handbook, ed. M. Freeling and V. Walbot, pp. 350-358, 1994). We crossed monosomic-10 plants that were *Oy Bf2* (in 10S and 10L respectively) as male parents by diploid *oy bf2* female parents, and the F1s were testcrossed in the summer of 1993. Approximately 100 testcross progeny of each of 38 different F1 plants were analyzed, and 6 of the 38 testcrosses had ratios that were significantly different from a 1:1 ratio for one or both of the marker mutants as determined by a Chi-square test. The ratios in these six crosses are given below (* = significantly different from a 1:1 ratio at $p \leq 0.05$).

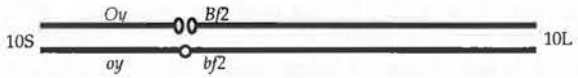
Plant	# *Oy*	# *oy*	χ^2	# *Bf2*	# *bf2*	χ^2
93-532-3	59	39	4.08*	57	41	2.61
93-532-5	58	38	4.17*	58	38	4.17*
93-532-11	63	36	7.36*	64	35	8.50*
93-533	62	37	6.31*	52	47	0.25
93-534-1	59	37	5.04*	58	38	4.17*
93-534-2	56	42	2.00	59	39	4.08*

We also crossed monosomic-10 plants that were *Oy Bf2* as female parents by diploid *oy bf2* male parents, and the F1s were reciprocally testcrossed in a 1991-1992 winter nursery. 45 crosses where the F1s were testcrossed as male parents have been classified, and 8 of the testcrosses had ratios that were significantly different from a 1:1 ratio for one or both of the marker mutants. Unfortunately, only a small number of progeny were available from some of these crosses. The ratios for these crosses that deviated significantly from a 1:1 ratio are given below:

Plant	# *Oy*	# *oy*	χ^2	# *Bf2*	# *bf2*	χ^2
H91-401-2	10	11	0.05	16	5	5.76*
H91-401-8	53	46	0.50	62	37	6.31*
H91-402-1	37	29	0.97	42	24	4.91*
H91-403-10	49	49	0.00	39	59	4.08*
H91-405-12	9	7	2.78	8	1	5.44*
H91-409-1	42	22	6.25*	41	23	5.06*
H91-409-3	43	30	2.32	45	28	3.96*
H91-415-6	51	33	3.86*	51	33	3.86*

These have not been corrected for small sample sizes, so deviations from a 1:1 ratio in plants 401-2 and 405-12 may be due to small sample sizes.

The reasons for these deviations from a 1:1 ratio are not known. If the univalent chromosome 10 misdivided during anaphase I of meiosis producing telocentric chromosomes for both arms, the F1 dihybrids would have telocentrics for both arms of chromosome 10 from the monosomic with dominant alleles of *Oy* and *Bf2*, and a normal chromosome 10 from the tester parent with recessive alleles of both loci as diagrammed below:



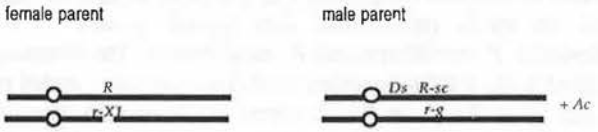
When a plant of this type is testcrossed, the telocentric chromosomes might be lost during meiosis in a portion of the cells, and one might expect that more than half of the viable gametes would contain recessive alleles for one or both loci. Because nearly all of the crosses where ratios deviated significantly from a 1:1 ratio had an excess of dominants for one or both loci, we believe that it is unlikely that these plants contain telocentrics for chromosome 10.

Another possibility is that the exceptional F1 progeny could have been trisomic for chromosome 10 (the monosomic contributed 2 chromosome 10s with dominant markers and the tester contributed one 10 with recessive markers). When such a plant is testcrossed as a male parent, a ratio of 2 dominants to one recessive would be obtained for the marker mutants (because haploid pollen grains almost always outcompete disomic pollen grains). The testcross ratios where the F1s were testcrossed as male parents could be accounted for if in this way. However, a trisomic of this type (with two dominants and one recessive) testcrossed as a female parent is expected to give a ratio of approximately 5 dominants to 1 recessive, and the testcross ratios where the F1s were testcrossed as female parents did not give such ratios. We therefore believe that the aberrant ratios where the F1s were testcrossed as female parents were not due to trisomy.

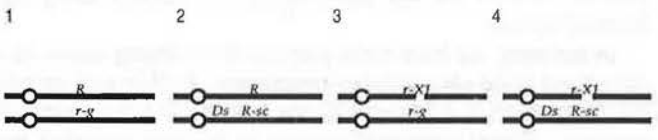
Endosperm and plant phenotypes of *r-X1* deficiency hemizygotes
--D. Weber and M. Schneerman

The *r-X1* deficiency is an X-ray induced submicroscopic deficiency that includes the *r* locus on chromosome 10. The deficiency is not transmitted through male gametes; however, it is transmitted with high efficiency through female gametes. If *R/r-X1* plants (which are heterozygous for the deficiency) are testcrossed as female parents, about 55-60% of the kernels are colored (*R/r*) and 40-45% are colorless (*r/r-X1*) deficiency-bearing kernels. Plants germinated from the *R/r* kernels from the above cross are invariably diploid while those germinated from *r/r-X1* kernels include 10-18% monosomics and 10-18% trisomics. The remaining plants are mostly diploids; however, multiply aneuploid plants and plants with deficiencies are also produced (reviewed in Weber, Maize Handbook ed. M. Freeling and V. Walbot, pp. 350-358, 1994). Thus, aneuploids are only produced from embryo sacs that contain the *r-X1* deficiency. Clearly, a factor is present on chromosome 10 that is deleted in the *r-X1* deficiency that is necessary for normal chromosomal disjunction during one or more of the embryo sac divisions. In the current study, we wanted to examine the effect of the *r-X1* deficiency in the hemizygous condition in endosperm and embryo cells to determine if additional genetic factors could be assigned to the *r-X1* deficiency.

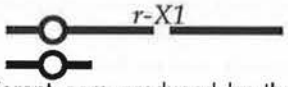
Production of endosperm sectors that are hemizygous for the *r-X1* deficiency. Jerry Neuffer, University of Missouri, generously provided us with a stock that had a chromosome-breaking *Ds* that was proximal to the *R* locus (between the *R* locus and the centromere) on chromosome 10. Neuffer selfed an *Ac*-containing *r-g/R-sc Ds4* plant and selected mottled kernels which were sent to us. These mottled kernels were produced when the *Ds* breaks the *R-sc*-bearing chromosome proximal to the *R* locus, the other homolog contains *r-g*, and *Ac* is present. In other words, the mottled kernels should have the same genotype as the original selfed *Ac*-containing *r-g/R-sc Ds4* plant. The number of *Ac* elements that were present is not known. We crossed these as male parents on *R/r-X1* plants in the inbred W22. This cross is diagrammed below (the *r-X1* deficiency is shown as a gap in the chromosome):



Kernels with the following four types of endosperms are expected (some have an *Ac*) (for simplicity, only one of the two identical maternally contributed chromosomes is shown):



Kernel types 1 and 2 are colored because they have a dominant allele of the *R* locus and kernel type 3 is colorless because it has one recessive *r-g* allele and a deficiency that includes the *r* locus. In kernel type 4, the *Ds* causes breaks in 10L proximal to the *R* locus if an *Ac* is present. Thus, the endosperms of these kernels should contain cells with the genetic constitution indicated above and some cells that are hemizygous for the *r-X1* deficiency as diagrammed here:



Several different ears produced by the above cross each contained kernels with three different endosperm phenotypes. Type I kernels had purple normal endosperm, type II kernels had colorless normal endosperm, and type III kernels had purple etched endosperm. Type III kernels were usually somewhat smaller than the other two kernel types. We conclude that type I kernels had genotypes 1, 2, or 4 with no *Ac*, type II kernels had genotype 3, and type III kernels had genotype 4 with an *Ac*. Thus, it appears that kernel type III had a mixture of viable and inviable (or at least very slow-growing) cells to produce the etched phenotype, and we conclude that cells hemizygous for the *r-X1* deficiency are inviable, and that a gene necessary for survival of endosperm cells has been lost in the segment that is deleted in the *r-X1* deficiency.

Production of leaf sectors that are hemizygous for the *r-X1* deficiency. We planted type I (purple, non-etched) and type III (purple, etched) kernels in our 1994 summer nursery. Leaves of plants grown from type I kernels were completely normal (no sectors); however, leaves of plants grown from type III kernels had numerous small sectors on them that were lighter green in color. Cells in the lighter green sectors appeared to be normal in size because the leaf surface was not altered in areas where a sector was present. No necrotic or albino areas were detected on plants grown from type III kernels. From these observations, we conclude that a locus is deleted in the *r-X1* deficiency that is required for normal chlorophyll pigmentation in leaf cells; however, no locus required for leaf cell viability is deleted in the *r-X1* deficiency. We had hoped to obtain large sectors on leaves of these plants that might be used for subtractive hybridization directed towards the cloning of genetic loci in the *r-X1* deficiency; however, all of the sectors observed to date were extremely narrow (one or a few cells wide).

We conclude from this study that a locus required for endosperm viability and a locus required for normal chlorophyll pigmentation in leaves are deleted in the *r-X1* deficiency in addition to the locus that is required for normal chromosome disjunction. We cannot be certain that the leaf and endosperm effects are caused by two different loci; however, this would seem likely.

An excellent pocket microscope for classifying maize pollen
--Dave Weber and Marti Schneerman

We lost all of our pocket microscopes (as well as numerous other items) when our field building burned to the ground in October, 1993. We examined pocket microscopes from several vendors prior to purchasing replacement scopes. We found one we prefer for pollen classification in the field.

We have always used pocket microscopes from Fisher, and they have been satisfactory. We compared 50X Peak Pocket microscopes (stock number C39,117) from Edmund Scientific Company, 101 E. Gloucester Pike, Barrington, NJ 08007-1380 (phone 609-573-6250) with pocket microscopes from several vendors, and we feel that the Peak scopes are far superior for pollen classification. They have a much brighter image and better

resolution than other scopes we have examined. The Peak scopes are very light, and small (about the size of a Sharpie pen), and they have a pen clip so they can be easily carried in a shirt pocket. We were concerned that they might get moisture or other material in them because they are a sealed unit and cannot be cleaned out. However, we have now used these scopes in two winter nurseries and one summer nursery, and no detectable foreign material or moisture has entered the scopes. In fact, the Fisher scopes (in our hands) needed to be disassembled and the lenses cleaned every few days if they were used intensively. Although the Peak scopes have a smaller field of view than the Fisher scopes, the field is reasonable for pollen classification.

Thus, the Peak scopes give a crisp bright image, are small and light, and never need to be disassembled and cleaned. We highly recommend these scopes for examining pollen in the field.

OTTAWA, CANADA
Agriculture Canada

Phytochemical mechanisms of resistance to ear rots

--D. J. Bergvinson and L.M. Reid

Recent studies have shown that both the kernel and silk possess phytochemical resistance mechanisms to *Fusarium graminearum*. Kernel resistance appears to consist of both physical resistance in the form of cell wall bound phenolic acids, and toxins in the form of soluble secondary metabolites. Silk resistance was initially thought to involve flavonoid biosynthesis in response to infection (Reid et al., Can. J. Bot. 70:1697-1702, 1992) but we have now identified resistant inbreds and hybrids with a contiguous wax layer covering the silk that is thicker than that found in susceptible genotypes.

During the past year we have been able to verify our field observations from last year's field season. Last summer we used scanning and transmission electron microscopy to see the morphological differences in the silk between resistant (CO272) and susceptible (CO266, CO265) lines. Compared to susceptible lines, CO272 had a visibly thicker wax coating on the silk as well as showing little or no checking in the silk.

During the winter we extracted the silk wax to both quantify and characterize the wax load of CO272 and susceptible lines in a time-study experiment. We found CO272 to have four major wax constituents that ranged from 2 to 5 times the levels found in susceptible lines. In addition, we found the wax levels in CO272 to increase from the day of silk emergence to 8 days post-emergence and then drop off, while susceptible lines tended to peak at 4 days (Fig. 1). This trend is interesting from the field screening perspective, as the best spread in disease severity ratings is achieved when the silk channel inoculation is applied between 6 and 8 days post-silking (Reid et al. 1992).

Not only does wax load change over time but it also changes along the profile of the silk. Most genotypes tend to have the greatest wax load at the point where the silk emerges from the husk. In the case of CO272 the wax load is high along the entire length of the silk. We have also screened 66 Ontario commercial hybrids for wax load and compared this to field ratings. The most resistant hybrid (Pride K127) had the highest wax load of all the hybrids screened. However, unlike CO272, the commercial hybrid only had high levels of wax at the husk collar and the rest of the silk had levels comparable to more susceptible hybrids. This study to

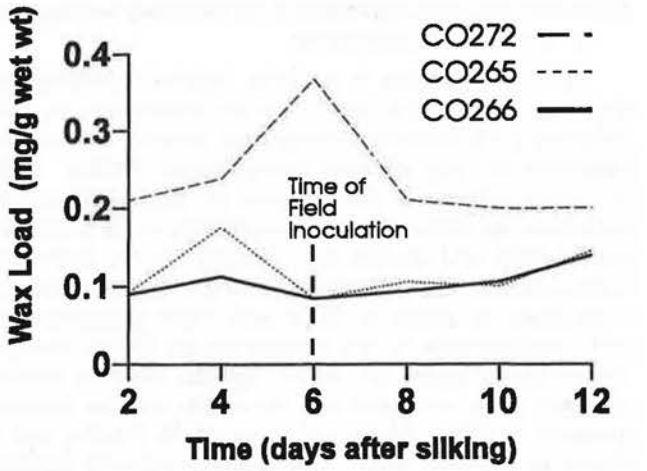


Figure 1. Time profile of silk wax load on resistant (CO272) and susceptible (CO265 and CO266) corn lines.

date has shown that wax load is at least a component, if not the main component, in silk resistance and that timings of sampling and positioning along the silk channel are important considerations when studying this resistance mechanism. We have now identified the major wax constituents to be simple hydrocarbons (C25-C31).

Last summer we conducted a time study for kernel resistance using our resistant Agriculture Canada inbred CO325 and a susceptible inbred CO266. We observed that kernel inoculation with *F. graminearum* resulted in the progressive spread to approximately 8 kernels (4 were wounded initially) in 5 days for both lines. However, by 9 days the spread had stopped for CO325 while the infection continued to spread in CO266 until the cob was completely infected by 28 days post-inoculation. We are presently doing phytochemical analysis on the kernels which were collected at 1, 3, 5, 7, 9, 11, 14, 21, 28, 35 days post-inoculation. Kernels have been fractionated into two fractions: pericarp/aleurone and germ/endosperm. Differences in soluble and cell-wall-bound phytochemicals over time and between lines will hopefully provide further insights into kernel resistance mechanisms.

During the winter we have developed a liquid bioassay procedure to test pure compounds that are found in corn for their effects on spore germination and hyphal growth of *F. graminearum*, *F. moniliforme* and *F. subglutinans*. The bioassay consists of 2 mL of Bilay's medium (1.5% sucrose) being added to 5 mL test tubes along with 10,000 spores and the test compound. After four days of periodic shaking, the contents of the tube are homogenized and the optical density recorded. Using this procedure we have found phenolic acids such as ferulic and p-coumaric acid to have an LC_{50} of approximately 200 ppm, while the most toxic compounds included salicylic and cinnamic acid. The phenolic amines did not demonstrate any toxicity using this bioassay system.

In summary, we have made progress in identifying waxes as a component in the silk resistance mechanism. Further work on how this mechanism is influenced by the environment is currently in progress. Kernel resistance appears to be most important two weeks after inoculation, and our efforts this season have focused on intensive sampling at this stage in ear development in hopes of seeing more dramatic differences between resistant and susceptible genotypes. We also have a bioassay system in place

for testing compounds found in corn for their effects on spore germination and hyphal growth of the major *Fusarium* spp. from Ontario.

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The revealing and selection of high-protein sources of maize
--V.E. Micu, Eugenia Partas and A.I. Rotari

This research began in 1974. In the first stage (1974-80) the protein content of 1,572 populations and samples was determined by the Kjeldahl semi-micromethod. In the investigated collection, including large diversity in geographical and genetic origin, morphology, growing period and other traits, samples with high protein content (12-15%) were revealed, the majority of them among local flint populations from Moldova.

In the second stage, the individual selection of high-protein plants in the best population was conducted and the results for four of them (K-44, K-466, K-553, K-1081) are presented in Table 1. Annually, 10-20 plants were self-pollinated and analyzed for protein content. Though the volume is limited, nevertheless each of the four populations reveals large genetic diversity among plants in protein content. Using long-term selection, progenies with protein content of more than 23-24% have resulted, significantly exceeding the initial level. It is necessary to note that the best progenies, with 23-24% protein, were obtained in population K-466, which had the highest initial protein content (Table 1) and highest variation. After 12 years of selection, genetic variability of protein content is not settled, as shown by the 1992 data.

Table 1. The protein content in self-pollinated progenies of the last 10 cycles of selection (% in dry matter).

Cycles of Selection & Year	K-44 lim	K-466 lim	K-553 lim	K1081 lim
Initial Level	12.12-12.75	13.25-15.69	12.25-13.75	14.44-14.91
3 - 1983	17.08-20.09	15.88-20.69	13.72-18.41	15.64-19.25
4 - 1984	17.06-19.00	14.68-23.35	16.37-19.25	15.88-21.66
5 - 1985	15.52-19.37	15.16-19.85	17.44-19.61	12.51-15.88
6 - 1986	13.96-19.12	12.27-21.30	15.88-16.60	12.27-16.00
7 - 1987	15.06-19.61	14.19-22.65	17.69-18.08	15.64-17.69
8 - 1988	16.97-21.17	14.80-20.21	17.08-19.13	13.83-18.53
9 - 1989	17.20-21.39	15.61-24.27	17.31-20.45	16.36-18.78
10- 1990	18.24-20.02	17.88-23.86	18.81-19.27	17.05-19.50
11- 1991	18.79-21.02	18.03-23.71	17.15-20.18	15.40-18.26
12- 1992	16.03-17.71	17.37-21.53	15.67-17.88	16.60-17.05
Populations limit	12.12-21.39	12.27-24.27	12.25-20.45	12.27-21.66
No. of Analyzed Progenies	159	626	60	68

The third stage of work began in 1987 and includes the cross of the four families highest in protein, self-pollination of hybrids, and selection in segregating progenies. Protein content in 1989-93 was determined by Near-Infrared method mod. 4500. In the F1 in all cases the protein content decreased considerably (Table 2), but the best progenies of the F2-F5 generation exceeded the best of the parental component by a considerable extent, except the hybrids with K-466. In these, the protein content of cross progenies was 21.05%, and the best selections from the F2-F5 had lower protein contents.

Nevertheless, even with these exceptions, the results testify to: (1) the presence of various genetic factors in different high protein sources; (2) the possibility of concentration of genetic factors favorably affecting the protein content from different sources into one. This allows us to expand the genetic base for further selection of a high-protein source. The work will be continued in this direction in order to obtain a source of high protein unrelated to IHP.

Table 2. The protein content of crossed families (1987), their best progenies (1988-92) and their F1-F5 (% in dry matter).

Sources & Hybrids	1987	1988	1989	1990	1991	1992
K-44 female	17.93	19.45	19.05	19.10	19.95	17.75
K-1081 male	16.40	16.26	17.60	18.40	17.35	17.48
K-44 x K-1081		15.83	21.30	23.31	23.40	21.95
K-1081 female	16.10	16.40	16.21	17.60	18.40	17.35
K-553 male	16.00	17.93	18.30	18.20	18.55	18.90
K-1081 x K-553		14.49	16.60	19.55	20.40	21.10
K-1081 female	15.04	16.38	16.10	17.58	18.35	17.35
K-466 male	21.05	18.03	18.14	20.40	20.87	20.48
K-1081 x K-466		15.52	19.85	18.90	21.45	20.58

PISCATAWAY, NEW JERSEY
Rutgers University

Stocks carrying mapped *Ac* elements available from the Co-op
-- Hugo K. Dooner

Several recently developed *Ac*-carrying stocks have been deposited with the Stock Center. These stocks carry *Ac* elements that have been mapped to chromosomes or chromosome arms and, therefore, constitute potentially useful starting materials for transposon tagging experiments.

Last summer, seed was generated from 24 stocks carrying different *Ac*s that transposed from *bz-m2(Ac)* in 9S to non-9 sites in the genome (Dooner et al., Genetics 136:261-279, 1994). Many of these *Ac*s are in chromosomes or chromosome arms for which no mutable alleles carrying *Ac* are known. Each transposed *Ac* (*trAc*) was re-extracted from the particular translocation with which it originally showed linkage and its linkage to *wx*, the translocation breakpoint marker, was verified. A fair supply of seed for each *trAc* (from 30 to 50 semisterile ears) has been sent to the Stock Center. The Stock Center needs to regrow these stocks and assign them new numbers, but is prepared to handle some seed requests in 1995.

The seeds deposited with the Co-op derive from the particular translocation heterozygote in the *wx* translocation series that showed linkage between *wx* (the *T* breakpoint) and *Ac*. The *bz-m Wx* seeds are segregants from crosses of the type

Bz wx-T +
bz Wx-N Ac x *bz-m2(DI) wx* (no *Ac*)

and should therefore be *bz Wx/ bz-m2(DI) wx ; Ac/+*, i.e., they should not carry the translocation and should not be semisterile. Also, barring undetectable double crossovers, they should carry the W22 parental contributions for both the *bz Wx 9S* segment and the segment in the non-*9* chromosome extending from the position of the translocation breakpoint to that of the *trAc*.

Caveat. Since *Ac* does not reside in a reporter gene, a secondary transposition from its original location would not be easily detected. Therefore, some precautions need to be taken in propagating the stocks. Here are two suggested ways of proceeding.

(1) Amplify the stock by selfing the *bz Wx/ bz-m2(DI) wx; Ac/+* plants. Then, pool the spotted seeds from several ears for use as *Ac*-carrying parents in the large scale tagging crosses the

following generation. This procedure minimizes the problem of secondary transpositions by avoiding the bottleneck of using one or two plants, but results in plants carrying variable doses of *Ac*. It is the "quick and dirty" way.

(2) The safest procedure is to maintain the stock by crossing the *bz Wx/ bz-m2(DI) wx; Ac/+* plants to the particular *wx* (*Bz*) translocation (available from the Stock Center) that showed linkage to the *trAc* in question. Then, take *Wx* segregants from the outcross and testcross them to the reporter stock *bz-m2(DI) wx* to confirm the *Wx-Ac* linkage. Keep ears showing linkage (most of the bronze seeds have spots) and discard the rare cases where a secondary transposition to an unlinked site has taken place (half of the bronze seeds have spots, half don't). This approach is more laborious and time consuming, but it gives one the added confidence that the individuals that one is using in the large scale tagging experiment descend from a plant that, in fact, had *Ac* in the targeted chromosome.

Table 1 lists the *trAc* stocks available, pertinent mapping information, and the chromosomal location of each *trAc*.

Table 1. List of transposed *Ac* maize stocks.

		Translocation showing linkage				
trAc	Recent pedigree number	Translocation	Breakpt. in non *9*	*Ac-wx* (cM) [1]	Other information	Most likely location
8178	C4301-3	2-9b	2S.18	5		*2S*
	C4304-6	2-9c	2S.49	15		
	C4307-9	2-9d	2L.83	38		
8163	C4310-11	3-9(8447)	3S.44	12		*3S*
	C4312-13	3-9c	3L.09	30		
8183	C4314-15	3-9(8447)	3S.44	6		*3*
	C4316-17	3-9c	3L.09	3		
8200	C4318-19	4-9g	4S.27	3	*Ac-su*: 4cM	*4*
		4-9(5657)	4L.33	4		
6076 *	C4320	5-9a	5L.69	31	*Ac-pr*: 12cM	*5L*
	C4321	5-9c	5S.07	4		
8175	C4322-23	5-9c	5S.07	10	*Ac-pr*: 35cM	*5S*
		5-9a	5L.69	U		
8193	C4324-27	5-9c	5S.07	4	*Ac-pr*: U	*5S*
		5-9a	5L.69	U		
8179	C4328-29	5-9a	5L.69	7	*Ac-pr*:14cM	*5L*
		5-9c	5S.07	21		
8181	C4332-33	5-9a	5L.69	10	*Ac-pr*: 41 cM	*5L*
		5-9c	5S.07	U		
8186	C4334-35	5-9a	5L.69	9	*Ac-pr*:34cM	*5L*
		5-9c	5S.07	33		
8196	C4338-39	5-9a	5L.69	7	*Ac-pr*:35cM	*5L*
		5-9c	5S.07	U		
6062	C4340-41	6-9b	6L.10	20		*6L*
6063	C4342-43	6-9b	6L.10	7		*6*
8172	C4344-45	6-9b	6L.10	16		*6L*
8184	C4346-47	6-9b	6L.10	3		*6*
8161	C4348-49	7-9(4363)	7 cent	13		*7*
		7-9a	7L.63	U		
8173	C4350-51	7-9(4363)	7 cent	1		*7L*
	C4352-53	7-9a	7L.63	29		
8190	C4354-55	7-9(4363)	7 cent	5		*7*
		7-9a	7L.63	U		
8194	C4356-57	7-9(4363)	7 cent	3		*7*
		7-9a	7L.63	U		
8185	C4358-59	7-9a	7L.63	3		*7L*
		7-9(4363)	7 cent	U		
8162 *	C4360-61	8-9d	8L.09	14		*8*
		8-9[6673]	8L.35	U		
8182	C4364-65	8-9d	8L.09	6		*8L*
	C4366-67	8-9[6673]	8L.35	1		
6059	C4368-69	9-10b	10S.40	24		*10S*
	C4370-71	9-10(8630)	10L.37	U		
8180	C4372-73	9-10b	10S.40	11		*10*
	C4374-75	9-10(8630)	10L.37	18		

[1] U = unlinked

* *trAc* is borne on a *wx* chromosome. The *Ac-wx* distance was determined directly from a *Wx* reciprocal translocation for *6076* and indirectly from the *Ac-bz* distance in a *wx* reciprocal translocation for *8162*.

RALEIGH, NORTH CAROLINA
North Carolina State University

Comparative recombination distances for maize inbreds, wide crosses and interspecific hybrids

--Claire G. Williams, C.W. Stuber and Major M. Goodman

Variability in recombination rates for maize was first reported by Bregger (Amer. Nat. 52:57-61, 1918). More recently, this has been confirmed using molecular markers (Tulsieram et al., TAG 84:65-72,1992; Fatmi et al., TAG 86:859-866, 1993). Several chromosomal and genic mechanisms for recombination rates are known in maize: supernumerary chromosomes, chromosomal rearrangements such as inversions or translocations, transposable elements, and modifier genes. A fifth mechanism, cryptic structural differentiation (CSD), inhibits recombination between chromosomes from genetically distant parents. Reduced recombination or "linkage drag" due to CSD is considered a deterrent to using exotic maize germplasm in U.S. maize breeding programs (Lonnquist, Ann. Corn Sorg. Ind. Res. Conf. Proc. 29:102-117, 1974).

The objective of this study is to survey "linkage drag" for a wide range of maize and teosinte races. Testcross pedigrees were constructed for a wide sample of maize and teosinte races using chromosome 1L (Chr1L) rare-allele stocks to ensure balanced polymorphism at all loci. The Chr1L stock is an admixture of U.S. and exotic maize germplasm. Data from eight linked isozymes on Chr1L were used to compare genetic variability in recombination rate among maize and teosinte races.

All exotic x Chr1L maize testcrosses showed enhanced recombination relative to domestic x Chr1L testcrosses. No linkage drag was apparent. Variability in recombination rate was also unrelated to genetic distance between parents; relic indigenous maize isolates did not have more or less recombination than more widespread exotic races. Segregation ratios, when distorted, showed a random pattern with respect to origin. Distorted ratios biased recombination distance only with respect to slight shifts in allele frequencies. Segregation distortion did not account for recombination shrinkage.

The four teosinte x maize testcrosses (Central Plateau, Guatemala teosinte, Balsas teostinte and *Zea diploperennis*) showed an opposing trend. Recombinant genotypes were largely absent, resulting in a severe deficiency of heterozygotes. Selective elimination of teosinte recombinants appears to have occurred due to a small inversion common to all three teosinte species. These inversions resulted in severe genetic map "shrinkage" and segregation distortion at all loci tested on Chr1L.

Desynaptic (*dy*) mutant reduces crossover recovery on Chromosome 1L

--Claire G. Williams, C.W. Stuber and Major M. Goodman

Desynaptic is a meiotic mutant first reported by Nelson and Clary (J. Hered. 43:205-210, 1952). Cytogenetical analysis shows *dy* mutants do not display orderly disjunction during anaphase I, after crossing-over has occurred (Maguire, Chromosoma 65:173-183, 1978). Mutant plants can be phenotyped easily by a reduced proportion (65-75%) of normal pollen and this can be verified with pachytene analysis. Cytogenetic analysis of *asynaptic*, another maize meiotic mutant, did not corroborate marker-based approaches to recovering meiotic products so we tested the hypothesis that *dy* is a gene

which modifies recombination frequencies.

Mutant *dy* plants from stock verified by Maguire et al. (Genome 34:879-887, 1991) were crossed to a chromosome 1L rare-allele stock and a single F1 parent was selfed to produced 200 F2 plants segregating for *dy*. Mutant and wild-type F2 parents which were segregating for marker loci were then selfed to produced F3 offspring. Linkage distances were compared between a mutant *dy/dy* F2 family and a wild-type *Dy/dy* F2 family composed of 100 offspring each.

Preliminary data show that the wild-type offspring had a total map distance comparable to our previous study (72.3 cM for the *amp1-gdh1* interval on chromosome 1L) but the *dy/dy* mutant offspring showed a total of 46.9 cM for the same interval.

Evidence for independent early and late developmental programs during juvenile epidermal differentiation

--Stephen Moose

Leaves 1 through 7 in most maize inbred lines exhibit juvenile traits. The juvenile phase is most easily identified by the expression of leaf epidermal traits such as bluish, highly hydrophobic waxes and the absence of leaf hairs. Using these traits as well as toluidine blue-O staining and cell wall morphology as markers, I have observed that all juvenile leaves have similar phenotypes and therefore appear developmentally equivalent with respect to the juvenile-to-adult phase transition. The phenotypes of *glossy15* (*gl15*) mutations, however, do not support this hypothesis. In *gl15* mutant seedlings, adult traits are observed beginning with leaf 3 but do not affect the juvenile character of leaves 1 and 2. This phenotype could reflect the possibility that the available *gl15* mutant alleles are hypomorphic rather than null mutations, where *Gl15* gene function is reduced but not absent; or it may represent the effects of an additional, independently regulated juvenile epidermal program which only operates in leaves 1 and 2 (Evans et al., Development 120: 1971-1981, 1994; Moose and Sisco, Plant Cell 6: 1343-1355, 1994).

In this report, I present three new findings which support the hypothesis that the juvenile epidermal phenotypes in leaves 1 through 7 represent the composite effects of two similar, but independently regulated developmental programs. These programs appear to act in distinct spatial/temporal domains which overlap at leaf 3. The early juvenile program operates in leaves 1 - 3, while the late juvenile program acts in leaves 3 through 7 and is regulated by *Gl15*.

Phenotypic null alleles of *gl15*: I have examined the phenotypes of seven different *gl15* mutant alleles, none of which affect the juvenile traits in leaves 1 and 2. The origin of four of these *gl15* alleles is known. The unstable *gl15-m1* allele is the result of a 2.1-kb *dSpm* insertion and therefore probably conditions a null phenotype. This hypothesis is supported by the observation that *gl15-m1* did not exhibit dosage effects expected of a hypomorphic allele. Hypoploid *gl15-m1* plants generated by crossing *gl15-m1* to a TB-9Lc stock had leaf epidermal phenotypes which were qualitatively similar to their *gl15-m1* parents. I have also identified three independent stable derivative alleles from *gl15-m1*. The molecular features of these alleles were examined using a cloned portion of the *gl15-m1* gene as a probe. One derivative allele from *gl15-m1* appears to have sustained a 600-bp deletion within the *dSpm* element, which has led to a stabilized insertion. Even if this *dSpm* is inserted in a non-coding region of the *Gl15* gene, it is unlikely that this allele would encode a functional *Gl15* protein. Therefore this *gl15-stable* allele is probably a phenotypic null.

Cg1 extends the effects of the early juvenile program: Figure 1 shows a typical leaf 3 from *gl15-m1* plants. Revertant juvenile wax sectors can be partially obscured by the diffuse pattern of transition waxes often found at the tip of leaf 3, which is similar to that observed in leaf 6 of normal plants. This result suggests that the deposition of these apparently similar juvenile waxes in leaf 3 is regulated independently. The diffuse waxes appear to be produced by the action of the early juvenile program, while the underlying sector depends on *Gl15* function. In *Cg1/+, gl15-m1* plants, revertant sectors in leaves 3 - 8 are also obscured by a diffuse layer of juvenile waxes (Fig. 1). These waxes are not present in subsequent leaves which exhibit defined juvenile and adult sectors. This observation indicates that the effects of the early juvenile program may be extended through leaf 8 in the presence of *Cg1*.

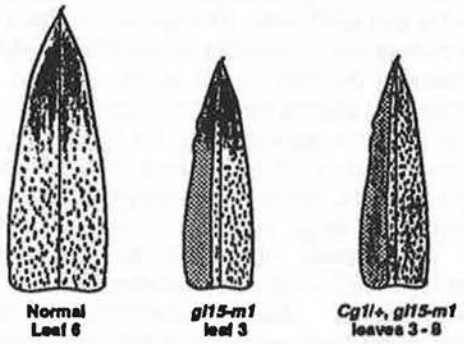


Figure 1: *Cg1* Extends the Effects of the Early Juvenile Program. Schematic drawings of representative leaf phenotypes are shown. A single large *Gl15*-revertant sector expressing juvenile waxes is within the left half of each *gl15-m1* leaf. Epidermal tissues expressing juvenile waxes appear grey, adult waxes white. Stipples indicate the presence of epidermal hairs.

Adult trait expression in revertant juvenile sectors from *gl15-m1*: Microscopic examination of *gl15-m1* plants revealed two types of revertant sectors, those which express only juvenile traits and others which express both juvenile and adult traits. The phenotypes of the two types of sectors suggested that the reactivation of *Gl15* and juvenile traits during leaf development was not always capable of suppressing adult cellular differentiation, and that the timing of the reversion event was important. A possible explanation for these sector phenotypes is that the early reactivation of *Gl15* during leaf development can suppress adult traits, but later transposition events cannot suppress adult differentiation which has been previously initiated. Upon further examination of many more sectors, contradictions to this hypothesis were observed. I have identified large revertant sectors from leaves 4 and 5 which were coincident in both the abaxial and adaxial epidermis and expressed juvenile waxes, but also possessed epidermal hairs. These sectors represent early transposition events since they affect a large number of cells within the leaf, yet both juvenile and adult traits are present. Closer examination of these sectors revealed that the two different sector phenotypes were distributed with a defined polarity. These sectors showed only juvenile traits near the tip of the leaf, but both juvenile and adult traits were present near the base of the leaf. These observations argued that the presence of adult traits was influenced by the relative position of the sector from the base of the shoot.

The effects of leaf position on the presence of adult traits

within *gl15-m1* revertant juvenile sectors were further examined by determining where epidermal hairs appeared in adjacent mutant and revertant sectors which extended the entire length of the leaf blade. Such sectors were expected to be derived from early transposition events, and all cells within these sectors should share a common lineage. At least 10 sectors were selected from each of leaves 3 through 5 of *gl15-m1* plants backcrossed three times into the W64A inbred background. A schematic representation of these observations appears in Figure 2. No epidermal hairs were observed in leaf 2 or revertant sectors from leaf 3. These tissues showed a complete juvenile wax phenotype. *gl15* mutant sectors from leaf 3 expressed epidermal hairs throughout their length except at the leaf tip, where they were absent. As described earlier, the tip of leaf 3 still shows some juvenile wax expression, and the absence of hairs was correlated with the presence of juvenile wax. In leaves 4 and 5, *gl15* mutant sectors expressed epidermal hairs throughout their entire length. However, adjacent revertant sectors from these same leaves were found to express both juvenile and adult traits. The epidermal hairs within these revertant sectors were distributed with a defined polarity. Hairs were present at the base of the sector, and their frequency gradually declined towards the leaf tip. Leaf 4 sectors expressed epidermal hairs for approximately the basal quarter of the sector's length, while in leaf 5 hairs were present in the basal half of a sector. By leaf 6, sectors expressing juvenile waxes had hairs along their entire length like *gl15* mutant sectors (data not shown). These results demonstrate that the appearance of epidermal hairs within a *gl15-m1* revertant sector is dependent upon leaf position. Adult differentiation appears to be suppressed in revertant sectors along the entire length of leaf 3 and progressively less in leaves 4 and 5.

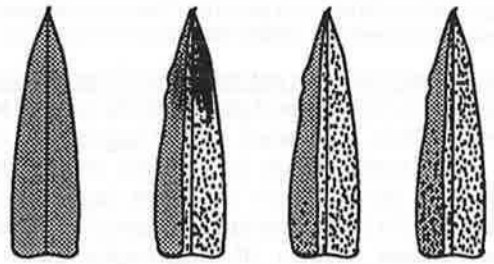


Figure 2: Wax and Epidermal Hair Phenotypes of *gl15-m1* Leaf Sectors. Schematic drawings of representative leaf phenotypes are shown. A single large *Gl15*-revertant sector expressing juvenile waxes is within the left half of each *gl15-m1* leaf. Epidermal tissues expressing juvenile waxes appear grey, adult waxes white. Stipples indicate the presence of epidermal hairs. Note how epidermal hairs are progressively distributed towards the leaf tip in revertant sectors from successive leaves.

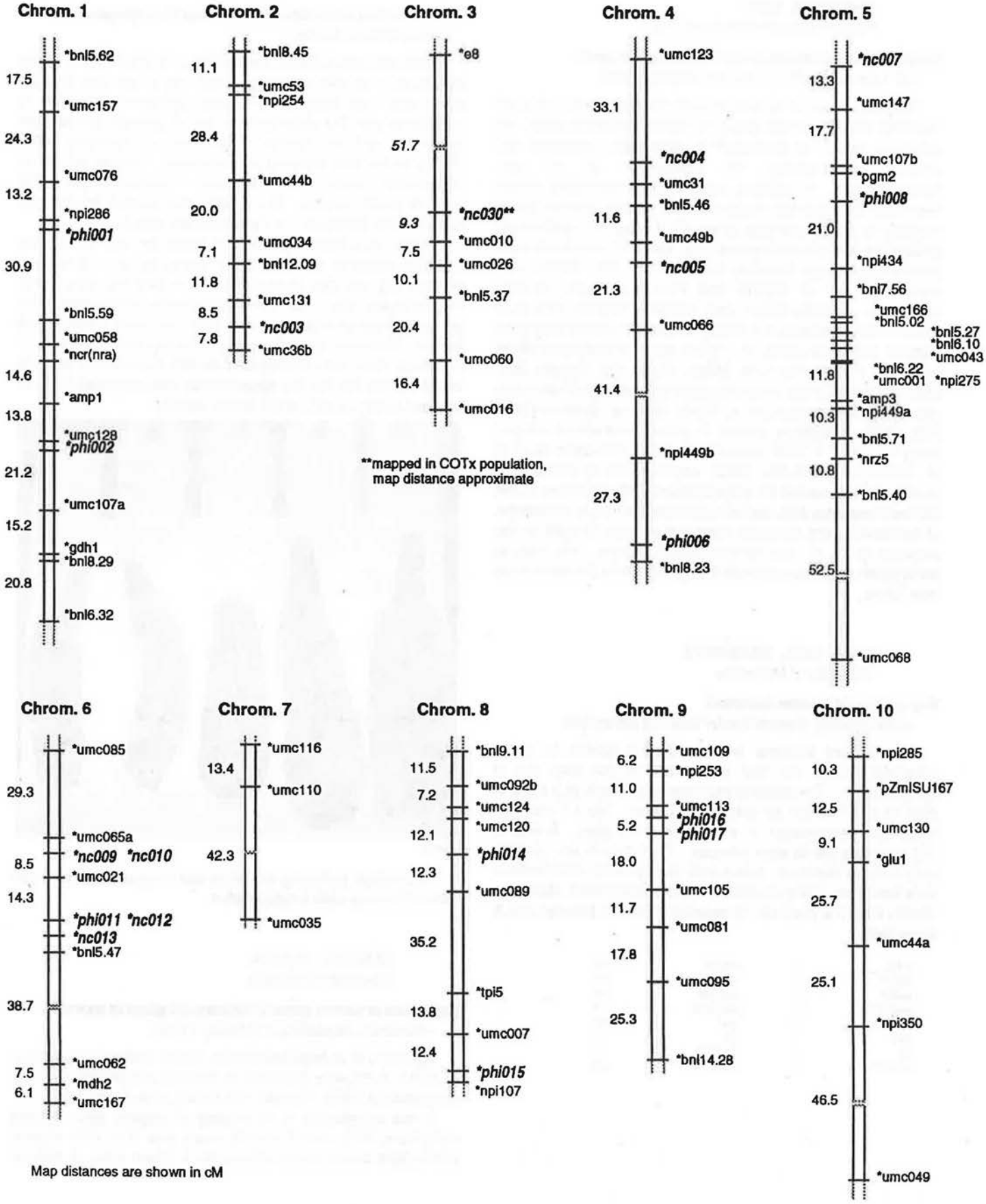
The proposed early juvenile program clearly conditions juvenile epidermal phenotypes in leaves 1 and 2. Therefore, it might be expected to influence the differentiation of other leaf cells which were present at the same time during development. In mature W64A embryos, cells which will become the upper portions of leaves 4 and 5 and the majority of leaves 1, 2, and 3 are all present and may therefore be considered developmentally equivalent. Adult traits in these leaf tissues are completely absent and their suppression is independent of *Gl15* activity. These observations provide support for the existence of an early juvenile program. The distribution of epidermal hairs within revertant sectors from leaves 4 and 5 of *gl15-m1* plants suggests that the action of the early juvenile program is restricted to a defined spatial/temporal domain at the base of the developing shoot, and that somatic reactivation of *Gl15* only suppresses adult traits when the early program is also active. The remainder of juvenile leaf epidermal cell differentiation is independent from the early juvenile program and is controlled by *Gl15*. It is still not clear why adult traits are completely suppressed in leaves 4 - 6 of normal plants but do appear within *Gl15*-active somatic sectors. Such phenotypes suggest that *Gl15* directly activates juvenile traits, but only plays an indirect role in the suppression of adult epidermal cell differentiation.

Seed survival after very early harvesting

--Major M. Goodman, Sheila D. Goodman and Dianne Beattie

During the summer of 1994, one of our isozyme transplants (7451-3) was injured by earworm or armyworm so that the tassel was destroyed. The plant was a rare favorable recombinant between *Sad1* and *Glu1* on chromosome 10, so it was used as a female. The top ear developed slowly, so three pollinations were made within 2 days on ears 2, 3, and 4, which silked virtually simultaneously. Ear 1 was left covered with a shoot bag. One or 2 days later, when ear #1 did silk, a pollination was set up to repeat the most important pollination (this particular one had already been made twice). After collecting pollen from 7481-6 (for the third time), it was obvious that 7451-3 was rapidly dying (from the base to the top of the plant) from an *Erwinia* bacterial stalk rot from irrigation water, and there was no point in making a fourth pollination on a dying plant. Knowing that the three possibly successful pollinations had been made for only a few days, it seemed impossible to salvage them, since we were approaching peak pollination period, and there was no time to arrange for embryo culture. In a half-hearted attempt to rescue something, the three pollinated earshoots were cut off at their points of attachment to the stalk and carried home that night. There they were trimmed a bit at the base, placed in small jars of water (with a bit of sugar added to each jar), and placed under a cool-white fluorescent light (used during the winter season for houseplants). The "developing" ears themselves were not immersed, but the stems were well covered with water. The water/sugar solution was changed several times as it became discolored, and the earshanks were trimmed a bit further. After the husks covering the ears acquired mold, the ears were husked. Ear #3 had rotted and was discarded. The naked ears, #2 and #4 (still greenish and damp) were left to dry under the lights. Ear #2 (7451-3 x 7481-6) had only 11 kernels; ear #4 (7451-3 x 7441-4) had only 3. All of these seeds were subsequently "planted" in the isozyme lab in September, using standard protocols (treated with Thiram 50 WP, wrapped in germination paper, dampened, placed in plastic boxes, incubated for 6 days at 35 C), except that the seeds were additionally surface sterilized in 3% H_2O_2 for 5 minutes before "planting." After clipping off bits of the coleoptiles, the seedlings were transplanted to 3-inch square peat pots, taken first to the greenhouse, then trucked to Homestead, Florida and transplanted there. All seeds and plants survived. Plant growth was normal. In emergency situations, it may sometimes be possible to salvage even less than seven-day-old kernels with crude and easily arranged methods.

Location of currently mapped Simple Sequence Repeats (in *italics*)



Map distances are shown in cM

RALEIGH, NORTH CAROLINA
USDA-ARS
JOHNSTON, IOWA
Pioneer Hi-Bred International, Inc.

Simple Sequence Repeats in maize - a progress report

--M. Lynn Senior, Emily Chin and Stephen Smith

Work has begun in earnest towards the development of a set of publicly available Simple Sequence Repeat markers in maize. An extensive search of Genbank® in early 1994 uncovered 105 sequences containing 190 repeats of di-, tri- and tetranucleotides. In addition, several repeat-containing clones have been extracted and sequenced from a maize genomic library enriched for the dinucleotide repeat AG (Library was synthesized by Ben Burr, Brookhaven National Labs, Upton NY and clones were provided by Linkage Genetics, Salt Lake City, UT). Primer pairs were made for all repeats that have acceptable flanking sequences. Of those primer pairs screened thus far, 35% have shown some polymorphism in maize. To date, 17 of these have been mapped using 192 plants of a B73 x Mo17 recombinant inbred population (Provided by C.W. Stuber, USDA-ARS, Raleigh, NC). The map shown on the preceding page was generated from these data using Mapmanager 2.5 (K. Manly, Mammal. Genome 4:303-313, 1993). In addition, another 11 primer pairs will be mapped using the Cm37 X T232 recombinant inbred population (Burr et al., Genetics 118:519-526, 1988). Approximately 60 primer pairs remain to be screened for polymorphism. Although most of the Genbank sequence data has been exhausted, we are in the process of synthesizing and screening additional genomic libraries for the presence of di-, tri- and tetra-nucleotide repeats. We hope to make primer sequences available through the Maize Database in the near future.

SAINT PAUL, MINNESOTA
University of Minnesota

Map position of *teosinte branched1*

--John Doebley, Pamela vander Weil and Adrian Stec

We mapped *teosinte branched1* (*tb1*) relative to seven molecular marker loci that are near it on the long arm of chromosome 1. The mapping population was based on a cross of W22 by a *tb1-ref/tb1-ref* homozygous plant. The F1 from this cross was backcrossed to a *tb1-ref/tb1-ref* plant. A total of 232 backcross plants were assayed. The *tb1* stock was given to us by Charles Burnham. Locus order and recombination fractions were determined using computer program MAPMAKER Macintosh version 2.0. The map with recombination fraction between loci is shown below:

rz403	--	*bnl8.10*	0.0
bnl8.10	--	*npi615*	2.2
npi615	--	*umc140*	3.5
umc140	--	*bcd1072c*	1.7
bcd1072c	--	*mu4*	0.0
mu4	--	*tb1*	1.7
tb1	---	*umc107*	1.3
umc107	--	*bnl 15.18*	0.0

SAINT PAUL, MINNESOTA

Xenia effect in a cross between BSSS and *Zea diploperennis*

--Lawrence A. Carlson

About ten ears of BSSS were selfed in St. Paul, MN when the first dozen or so silks appeared. Then one or two days later the same ears were pollinated with *Zea diploperennis* pollen. In pollinations with *Zea diploperennis* I cut off the husk almost down to the ear, split the balance of the husk and sprinkled pollen directly on the silks attached to the kernels. I usually get 100 to 200 crossed seeds per ear this way. I sometimes get a few airborne pollen crosses. This is how I first noticed the dramatic xenia effect in the maize x *Zea diploperennis* crosses.

Figure 1 is a photo of a BSSS ear selfed, two ears selfed early and then pollinated with *Zea diploperennis* pollen, and two ears crossed only with *Zea diploperennis*. The ears that were selfed, then pollinated later, show dramatically smaller kernels toward the tip, while those pollinated on the same day show uniform small kernels. Several of the ears pictured have not been shelled as of December 1994, but a few adjacent kernels picked out of the back would indicate that the *Zea diploperennis* crossed kernels have an adjusted weight of 60% of the BSSS kernels.

Figure 1.

This multiple pollinating technique can no doubt be refined and evolved for many uses in cytogenetics.

SARATOV, RUSSIA
University of Saratov

Expression of nuclear genes in different cytoplasmic sources

--Alexandra Zavalishina and Valery Tyrnov

By means of androgenesis in vivo, brown marker isogenic lines (*a B Pl C R Pr*) were produced in different cytoplasmic sources: Wisconsin 23 (W23), Kinelsky 103 (K103), cms-T, and cms-S.

It was ascertained: 1) in progeny of isogenic lines carrying cytoplasms W23, cms-T, cms-S, every year 3 to 60% atypical plants (light brown, green with tracks of brown color on stem or

tassel) appeared for 8 tested generations, whereas in progeny of isogenic lines, carrying the cytoplasm K103, all plants were typical brown (for 12 generations); 2) if atypical plants were crossed by normal ones, only atypical plants appeared in progeny (for 6 generations). When these atypical plants were crossed by a line with gene *A*, the whole spectrum of color (sun red, pale purple, sun purple, pale purple with some dark spots) appeared in progeny. This shows that dominant genes *B Pl* in atypical plants manifest themselves as recessive ones; 3) in reciprocal crosses between typical and atypical plants, irrespective of cytoplasm source in the maternal plant, only atypical descendents arose in F1. F2 also consists of atypical plants. It is difficult to give an explanation to this fact. The phenomenon of occurrence of atypical plants we observed also in brown marker isogenic lines in cytoplasms cms-S,-T,-C, produced by means of backcrosses. After 10 backcrosses only atypical plants arose. At the same time the same method led to production of atypical isogenic line in teosinte cytoplasm. Thus, cytoplasmic-nuclear interactions continue to act in many generations. We don't exclude the possibility that it affects a wider spectrum of characters and has, probably, breeding and evolutionary effects.

Cytological expression of *ig* mutation in megagametophyte

--N. Enaleeva, O. Otkalo and V. Tyrnov

The cytological effect of the *ig* gene in a homozygous condition (in an embryo marker line background) was investigated. The technique of isolation of total megagametophytes (MGs) at early coenocytic and mature developmental stages was used. Different abnormalities of MG structure (55-98% of plants) were observed. The range of abnormality types was found to be wider than in the original *ig* W23 line, described by Lin (1978, 1981). The comparison of abnormality patterns, manifested at different stages of MG development, resulted in the conclusion that the *ig* gene expression occurs at the beginning of the postmeiotic MG developmental program. The major effect of the *ig* mutation seems to consist in disturbance of some cytoskeleton functions, relating to bipolar nuclei distribution during the coenocytic phase. It is very likely that in MG-genesis, the formation of a large central vacuole after the first mitotic division is suppressed or reduced. As a result, the nuclei are not arranged at the opposite ends of the coenocyte, but are situated at random. Because of the abnormal nuclei position, disturbance of the subsequent chain of events happens. In particular, the number of mitotic cycles increases, the nuclei are scattered or are in contact with each other, and the cytokinesis and following cell differentiation is irregular. The cell types depend on nuclear position at the moment of differentiation. In some cases the MG developmental program is blocked at different stages and MG nuclei start to degenerate. It may be assumed, therefore, that the *Ig* gene apparently controls only one key event of MG-genesis, and its mutation into *ig* gives rise to a disbalance of this developmental program, resulting in plural cytological patterns.

Utilization of haploids for producing of mutants

--Valery Tyrnov

In our laboratory, the technology for matroclinal haploid production in great numbers was developed (Apomixis Newsletter, 1992, 6:6). Haploids were tested during 5 years of experiments as the objects for mutation breeding. Ionizing radiation and chemical mutagens were used. The apical meristem of seedlings, dry seeds with haploid embryos, ears and seeds of maternal forms were influenced. Haploids, in comparison with diploids, are 2-3 times more sensitive to irradiation (as to inhibition and lethal effects). However, the level of chromosomal aberrations in haploids was 30-90% lower. Perhaps it is conditioned by more extensive damage of haploid cells and their non-participation in mitotic cycles. All haploids after mutagenic treatments were changed. Diploids had no changes. Diploid self-pollinated mutant progeny, obtained from haploids, frequently were heterozygous, if apexes or even seeds with haploid embryos were influenced. Probably, this is connected with the appearance of different mutations in ears and panicles. The best variant for production of homozygous mutants from haploids consisted in treatment by mutagens of initial diploid maternal forms. The mutant forms with changed vegetation time, morphology, and color were obtained in different lines. In Mangelsdorf's tester, in addition, mutants for certain genes were produced. The appearence of white and light green stripes on leaves was a typical phenomenon after mutagenic treatment of seedlings and seeds with haploid embryos. It may be used as the test for estimation of different factors on mutagenic activity and also in ecological investigations of surrounding pollution.

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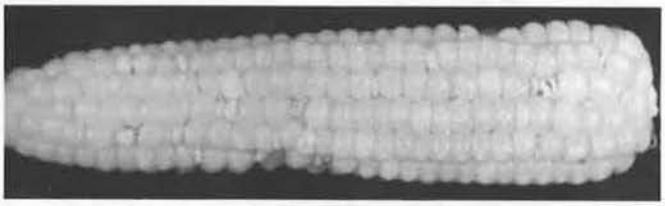
A source with a high percentage of haploid seeds in popcorn

--Toma Dankov, Miglena Kruleva and Zlatka Bojilova

It is known that popcorn ordinarily does not accept for pollination the pollen of the other maize subspecies. During the autumn of 1993 in the sowing area of a population of white popcorn (pearl type), under conditions of free pollination with pollen mainly from horse dent subspecies, two ears with high percentage of xenia and haploid seeds were found (Figure). From a total of 711 seeds (weak and wrinkled) 30 were haploid, or 2.37%. The haploidy was cytologically confirmed.



During 1994 most of the haploid seeds were sown in the field. One part of them did not germinate, another part died at the 5-6th leaf, and only 2 plants reached the flowering stage. The female fertility was expressed by the presence of 6-9 silklets on the small ears, but the tassels were fully sterile. From the normal seeds of those two ears 64 plants were grown. In the freely pollinated ears, about 15% haploid seeds were established.

It is interesting to note that the haploid seeds do not have horny but floury endosperm, which gives us reason to think that it concerns the androgenic but not matroclinous haploidy. The investigations continue mainly in that direction.

Pollinating the silk of an ear with five marked pollen components with a possibility to differentiate the seeds

--Toma Dankov, Miglena Kruleva, Zlatka Bojilova

Determining the genotype of a given maize plant, used as a

pollinator on defined female testers, is reached quickly and easy by dividing the pollen. This, however, is realized slowly if the plant is male sterile and can be used only as a mother component. Some possibilities in that direction are given by the availability of more than one ear on a plant (Dankov, MNL, 47:6-7, 1973). Up to now the pollination of the one ear from two different pollinators by dividing the silk was used. Greater possibilities in that direction are proposed by the great number of markers on the embryo, endosperm and seedlings.

Since 1988 we have been working on the creation and utilization of such markers in connection with haploidy. In 1993 and 1994 we realized the pollination of 3-4 ears from A632 cms-C and B37 cms-C lines with 5 pollinators for each ear - self pollen (without marker), colored embryo, embryo-endosperm marker and protruding embryo. The silk was divided into 5 parts by lightly binding with thread (Fig. 1), after which a cardboard separator was used to pollinate each part separately (Fig. 2). For one ear we obtained 30-50 seeds, which we distinguished by a dominantly inherited marker of the pollinator. Only about 3% of the seeds remained undefined, possibly connected with an embryo-endosperm marker or the heterofertilization.

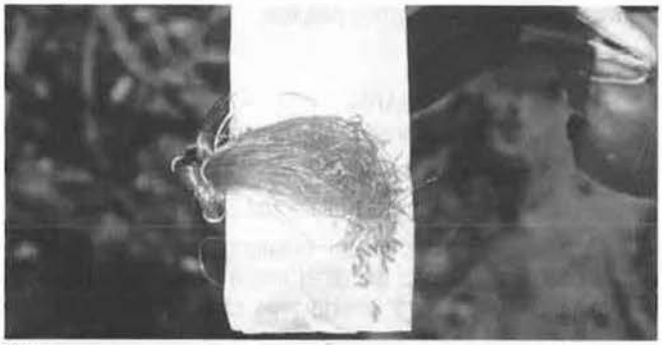


Figure 1.

Figure 2.

The transfer of markers continues on the following C-type sterility testers:

N rf4 rf4 Rf5 Rf5 Rf6 Rf6 (endosperm marker)
N Rf4 Rf4 rf5 rf5 Rf6 Rf6 (coloured embryo)
N Rf4 Rf4 Rf5 Rf5 rf6 rf6 (protruded embryo)

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Stay-green characteristics and characters related to stay-green in inbred lines

--Keun Jin Choi, Hong Seok Lee, Moon Sup Chin, Keun Yong Park, Seon Woo Cha and Seung Eui Park

Stay-green characteristic is one of the very important characters for silage and grain corn varieties, providing longer duration of filling period for high seed yield and longer duration of harvesting time for silage. This experiment was carried out to find out the stay-green characteristics, and characters related to stay-green, in 40 inbreds selected from Korea and other countries. Each inbred differed in maturity period, leaf number and leaf area. The mean number of days from silking to ear leaf senescence (DELS) of 40 inbreds was 34.4 days, the mean fresh leaf number was 9.54, the mean fresh leaf area was 2,522 cm2, and the mean chlorophyll content in silking stage was 0.073mg˙/2.8cm2. The inbreds were classified into four groups based on the peroxidase band pattern in the seed. DELS for the four groups were 27.3, 30.1, 37.5 and 41.2 days, respectively. The characteristics related to stay-green were fresh leaf number, fresh leaf area, chlorophyll content and DELS. Their correlation coefficients with the stay-green score were -0.992**, -0.977**, -0.984** and -0.974**, respectively. The stay-green inbreds had high photosynthetic activities and also had high protein and lipid contents, but metabolism of crude fiber and neutral detergent fiber (NDF) in leaves was low. The stay-green inbreds showed disease resistance to several diseases such as *Helminthosporium turcicum*, maize dwarf mosaic virus (MDMV), black streaked dwarf virus (BSDV) and stalk rot, and had especially great resistance to *Helminthosporium maydis*.

Heterosis and heritability of stay-green characters

--Keun Jin Choi, Moon Sup Chin, Keun Yong Park, Hong Seok Lee, Jong Ho Seo and Deuk Young Song

Since we determined stay-green characteristics and characters related to stay-green in inbred lines, to find out the heterosis and heritability of stay-green characters we crossed three inbred lines, Ga209, Hi39 and KS75 in a half diallel and got the three crosses of F1 and F2 selfed from the F1. We planted the three parent lines, and three F1 and F2 generations, and estimated the heterosis, heritability of stay-green characters, days from silking to ear leaf senescence (DELS), fresh leaf number, fresh leaf area and chlorophyll content.

High heterosis was expressed in DELS of the F1 and even of the F2 population in Hi39/KS75. A high positive heterosis of fresh leaf number, fresh leaf area and chlorophyll content was observed in the F1 but a negative heterosis in the F2 populations except for Hi39/KS75. Inbreeding depression in the fresh leaf number, fresh leaf area and chlorophyll content appeared remarkably associated with the progress of growth, especially from 30 to 52 days after silking (DAS) in an April 23 planting, but Hi39/KS75 had low inbreeding depression in DELS compared to the other two F2 populations. Among the characteristics related to stay-green, the fresh leaf number showed the highest heritability. The other characteristics, except the days from silking to maturity, also exhibited relatively high heritability. Hi39/KS75 showed very high heritability irrespective of planting dates. The stay-green score was found to be significantly

correlated with the fresh leaf number, fresh leaf area and chlorophyll content at 52 DAS. A multiple regression analysis with the F2 populations revealed that the fresh leaf number was mostly accountable for the variation in DELS and stay-green score, but less accountable at the later growing stage.

Segregation of stay-green characters in an F2 population

--Keun Jin Choi, Moon Sup Chin, Keun Yong Park, Seon Lim Kim, Tae Wock Chung and Hong Seok Lee

After reporting on the stay-green character and its heterosis and heritability, we tried to find out the segregation pattern of stay green characteristics in an F2 generation. We investigated the segregation frequencies of stay-green score, DELS and fresh leaf number in F2 generations of about 150~200 plants with parents and F1 hybrids planted at the Crops Experiment Station field in 1992.

The frequency distribution of stay-green scores and DELS generally had a simple bi-modal pattern irrespective of planting dates, and the pattern of stay-green scores shifted to higher levels in later stages. Hi39/KS75 had the longest DELS and it showed highest stay-green characteristics at 67 DAS. KS75, which was selected at our Crops Experiment Station in 1988, was considered a useful material in breeding for stay-green lines. The distribution patterns of F2 plants of Ga209/Hi39 and Ga209/KS75 were highly characterized as related to stay-green at 37 DAS, but shifted to lower levels at 52 DAS and maintained up to 83 DAS. In the progenies of the three crosses, the F2 from Hi39/KS75 showed a high stay-green score, DELS and fresh leaf number from early days after silking. It was possible to obtain higher stay-green lines in progenies of the cross of Hi39/KS75 than that of parents.

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Rindless culm (*rlc*) gene derived from Dangjin inbred

--Heebong Lee, E.H. Coe, Georgia Davis and Bongho Choe

This study was carried out to identify the location of the gene for rindless culm (*rlc*), which was derived from Dangjin, a Korean local maize line selfed six generations at the Genetic and Breeding Lab, Coll. of Agri., Chungnam Nat'l. Univ. (Korean J. Breed. 24:42-47, 1992). Materials used were F2s from crosses of waxy translocations with *rlc*. F2 kernels were separated into waxy and normal seed, and these were grown in 1994 at Columbia, Missouri. The expression is shown in Figure 1; the plants could be distinguished for rindless culm and normal after tasseling.

Table 1. Rindless and normal segregations from waxy translocations crossed with Dangjin rindless inbred.

	waxy		normal		
Translocation	rlc	+	rlc	+	χ^2*
wx T1-9(8389)	6	20	23	34	0.469
wx T4-9b	2	3	2	11	0.242
wx T4-9g	9	23	9	26	0.366×10^{-8}
wx T4-9(5657)	2	17	4	12	0.466
wx T5-9a	9	7	14	13	1.35×10^{-2}
wx T6-9b	5	26	2	35	1.031
wx T7-9(4363)	8	32	11	58	0.076
wx T8-9d	12	22	15	25	2.101×10^{-3}
wx T8-9(6673)	15	17	14	16	0.057
wx T9-10b	0	13	17	17	8.134***

rlc to normal plant (+) ratio under the assumption of *i:j*
***significantly deviant at p = 0.001

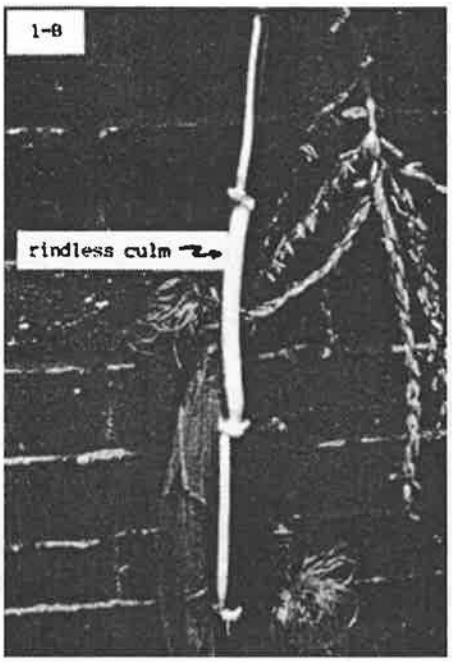

Figure 1. Rindless culm plant after tasseling. Variable fractions of the culm in the upper few internodes display absence of rind, extending from node to node in sector-like fashion but increasing in degree.

Chi-square analysis of the waxy translocation tests indicated that the rindless culm gene is linked to T9-10b (Table 1). All 13 plants from waxy seeds displayed normal culms, while non-waxy seeds showed segregation of normal and rindless culms. This places the *rlc* gene near T9-10b, which has a breakpoint at 0.4 on the short arm of chromosome 10.

Gene for multi-tillers and ears

--Heebong Lee, E.H. Coe, Georgia Davis and Bongho Choe

Since 1980 authors Lee and Choe have studied the prospects for development of maize hybrids with multi-tillers and ears. Toward the breeding of a new type of maize hybrid with multiple tillers and ears, a few inbred lines with multi-tillers and ears were developed: IK1, IK2, IK3, IK4 (MNL 68:100, 1994), Waesungri, and PI213749 inbred.

The tillering traits in these lines were determined to be controlled by a single dominant gene in studies with each line. Only the tillering gene in IK1 has been evaluated for chromosome location (Kang et al., Korean J. Breed. 23:285-289, 1992). To determine the location of additional tillering genes, the PI213749 inbred was crossed with a non-tillered inbred, KV1IK1, which has been selfed for six generations, and an F2 was produced for RFLP mapping.

F2 individuals were grown in the field in 1994 at Columbia, Missouri. Plants were rated for tillers as follows: 0, 1, 2, and 3 or more tillers. Leaf tissue was collected from individual F2 plants and parents. After freeze drying, genomic DNA was extracted using the CTAB method (UMC Maize RFLP Laboratory Manual 1995, p. 34). DNA was digested with *Eco*RI, *Hin*dIII and *Dra*I. DNA of parental lines, 0-tiller and 3+ tiller individuals was loaded onto 0.8% agarose gels and electrophoresed. DNA was transferred to Magnacharge membrane according to standard

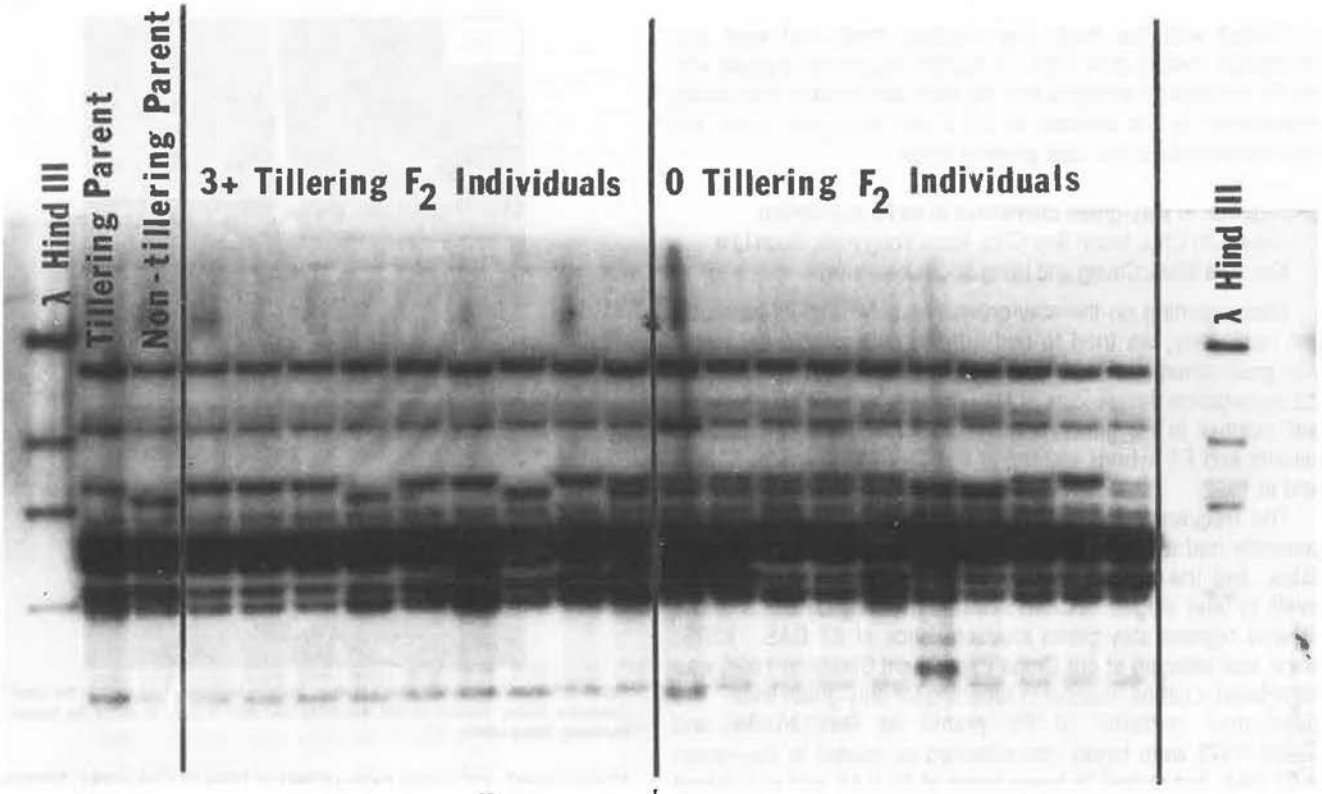

Figure 1. Southern blot of *Eco*RI-digested genomic DNA hybridized with ^{32}P-dCTP labelled *umc76* probe.

laboratory protocols. Hybridization using dCTP^{32} oligolabelled probes from the Maize Core Marker Set indicated that tillering is linked to *umc76* on the short of chromosome 1 (Fig. 1). Additional probings to establish whether the locus is proximal or distal to *umc76* are in progress. Future testing will be needed to determine whether the gene from PI213749 and *tlr1* (of Neuffer) are allelic.

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opaque15, a soft endosperm mutation that reduces gamma-zein content and has the properties of a defective _opaque2_ modifier

--Joanne Dannenhoffer, Gloverson Moro, Dwight Bostwick, Etti Or, Ronald Burnett and Brian Larkins

We have recently characterized (MNL this issue) a novel, opaque mutation that reduces synthesis of gamma zein in the endosperm. The mutation causes a two- to three-fold reduction in accumulation of the 27 kDa-gamma-zein mRNA and protein, but there is no pronounced effect on the other classes of zein proteins, or on non-zein proteins. *o15* has the properties of a defective *opaque2* modifier. In contrast to *o15*, *o2* modifier genes are associated with a two- to three-fold increase in the synthesis of the 27-kDa gamma zein mRNA and protein. There are two gamma-zein genes, *A* and *B*. Transcripts of the two genes were distinguished using a PCR- based assay, and the mutation alters the ratio of mRNAs encoded by the genes; *o15* causes a preferential reduction in the level of the *A* gene transcript. *o2* modifier genes are associated with an increase in the proportion of the *A* gene transcript. Using a combination of bulk-segregant analysis and RFLP mapping, the chromosome location of *o15* was determined to be on the distal end of chromosome 7L, coincident with the location of an *o2* modifier. Protein bodies in *o15* endosperm are comparable in size to those in *O15*, however, there are fewer protein bodies per cross-sectional area.

o15 acts as a single, recessive gene and kernels that are homozygous for *o15* do not germinate when planted in soil or on moist filter paper. To analyze this mutation we used kernels from heterozygous ears. In an effort to grow homozygous *o15* plants to use for further work on this mutation, we found that seedlings develop if we imbibe seeds in sterile water and germinate them on MS media. This indicates that the inability to germinate is a nutritional deficiency.

Our original analysis of the population of EMS-induced opaque mutants from Dr. M.G. Neuffer identified another mutant with reduced amounts of gamma-zein protein. We conducted an allelism test between *o15* and this second mutant. Detection of opaque seeds in the F1 indicates that they are allelic. We are now mapping the second mutation to confirm its identification.

A mutant alpha-zein in _floury2_ endosperm

--Craig E. Coleman and Brian A. Larkins

The *floury2* (*fl2*) mutation is characterized by a soft, starchy kernel that is highly susceptible to mechanical damage and pathogen infestation. The mutation is expressed semi-dominantly and is associated with elevated levels of BiP, an ER resident chaperone protein. Zein accumulation is generally reduced and the protein body development is severely affected. The mutation maps to 4S within a cluster of alpha-zein genes. We have cloned an alpha-zein gene that is tightly linked to the *fl2* locus and encodes a polypeptide that accumulates as a precursor. Failure to process the protein is due to a defect in the signal peptide that targets the molecule to the ER. Specifically, there is an alanine to

valine substitution at the C-terminus, or -1 position, of the signal peptide. The product of this gene bears homology to the 22-kDa alpha-zeins, but has an apparent molecular weight of 24-kDa on SDS-polyacrylamide gels. In an analysis of a backcross population the 24-kDa alpha-zein was detected in 64 *fl2*/+/+ kernels but was absent in 64 +/+/+ kernels. The gene was cloned as a segment of a 7.8 kb *Sst*I RFLP identified by bulked segregant analysis of an F2 population using a 22-kDa alpha-zein gene probe. Linkage of this RFLP to the *fl2* locus was assessed by Southern analysis, showing that the band was absent in 79 normal progeny of an F2 population. The deduced amino acid sequence of the gene contained in the RFLP matches perfectly with the first 45 amino acids of the N-terminus of the 24-kDa alpha-zein protein. Isolation and N-terminal sequencing of the protein were done by Dr. Jeff Gillikin in Dr. Becky Boston's lab at North Carolina State University, Raleigh, North Carolina.

Failure to cleave the signal peptide from the 24-kDa alpha-zein would be expected to result in the attachment of the protein to the lumenal surface of the ER membrane, and subsequent disruption of protein translocation. Such a perturbation may explain the elevation in BiP accumulation and the abnormal protein body development. The fact that there has been only one *fl2* allele identified, and that it is expressed semi-dominantly, suggests that it is a unique, gain-of-function mutation. This concept is consistent with the defect in the signal peptide of the 24-kDa alpha-zein protein arising from a unique point mutation. Epistasis of the zein regulatory gene *O2* to *fl2* can be explained by the presence in the promoter region of the 24-kDa alpha-zein gene of the 5'-TCCACGTAGA-3' motif common to genes regulated by the *O2* transcriptional activator. Taken together, these data provide compelling evidence that the 24-kDa alpha-zein gene is responsible for the *fl2* phenotype.

Genetic mapping of *opaque2* modifier genes

--Maurício A. Lopes, Glóverson L. Moro and Brian Larkins

Although the *opaque2* (*o2*) mutation causes an increase in the lysine content of the grain, the pleiotropic effects of the mutation prevented its widespread agronomic utilization. The soft, chalky endosperm of *o2* kernels is less dense and highly susceptible to insect and mechanical damage. The identification of genes with the ability to overcome these negative effects of the mutation, while maintaining its high nutritional value, provided new hope for the development of commercially attractive high lysine genotypes. These genes, designated *o2* modifiers, were subsequently used by breeders at the International Maize and Wheat Improvement Center (CIMMYT, Mexico) and University of Natal (South Africa) to develop modified *o2* genotypes called Quality Protein Maize (QPM). QPMs are *o2* varieties with normal, vitreous appearance and hardness, but with an enhanced percentage of lysine.

QPMs have not reached widespread commercial utilization, partially because the mechanisms controlling the modification of *o2* endosperm are not fully understood. Our lab has been working on the biochemical, genetic and molecular characterization of the modification and a considerable amount of information regarding this process is now available. The formation of vitreous endosperm is highly correlated with an increased accumulation of the 27-kD γ-zein, suggesting the involvement of this protein in the endosperm modification. However, the precise mechanism by which this happens is unclear. The modifiers, which act in a semi-dominant fashion, seem to affect γ-zein expression through a post-transcriptional mechanism. Although *o2* modifiers were considered to be genetically complex, segregation analysis suggests that there are probably no more than two or three loci with a major effect.

To determine the genetic map location of the modifier loci, we used bulked segregant analysis to identify linked RFLPs. Segregating progenies from crosses of W64A *o2* and W22 *o2* by Pool 33 QPM were analyzed. Approximately 90 RFLP markers covering the entire genome were used in this study. A single locus contributing to the phenotypic variation was identified in each cross. For W22*o2* x Pool 33 QPM a modifier locus was mapped near the telomere of chromosome 7L. For W64A*o2* x Pool 33 QPM, a second modifier locus was mapped at the 27-kD γ-zein locus, near the centromere on 7L.

The role of γ-zein locus in endosperm modification is unclear. The 27-kD γ-zein locus may contain one (Ra) or two (A and B) genes. Analysis of 44 recombinant inbred lines developed from the cross between W64A*o2* (Ra allele) x Pool 33 (AB allele) shows that modification is associated with the presence of the AB allele. Similar analysis of 37 recombinant inbred lines between W22*o2* x Pool 33 (both AB allele) showed that some inbreds with modified phenotype carry the AB allele from W22*o2*. This suggests that the allele of Pool 33 per se is not necessary for modification. Sequence analysis of A and B genes from *o2* and modified *o2* genotypes showed very few nucleotide differences in either the promoter or coding sequences.

We are extending the sequence analysis of the γ-zein locus to identify *cis*-acting regulatory elements. We are also extending the mapping analysis of *o2* modifier genes using QPM lines from South Africa. These materials were developed independently from those at CIMMYT, and they may help further resolve loci involved in endosperm modification.

Transposon-tagging of the *dwarf3* gene, which controls a cytochrome P450-mediated step early in the biosynthesis of gibberellins

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The sessile nature of higher plants requires that they use environmental signals, in addition to developmental signals, to alter their growth and development to respond appropriately to environmental conditions or stresses. Phytohormones are thought to play a primary role in transmitting the appropriate information within the plant. Gibberellins (GAs) are isoprenoid phytohormones required for normal growth and development in higher plants and have been proposed to be signals in multiple processes such as germination, juvenile to adult transitions, vernalization and flowering. Mutants that are deficient in GA-biosynthesis have been isolated in a number of plant species and have the characteristic features of recessive dwarfing and can be normalized by the addition of GA.

The *d3* (*dwarf3*) gene of maize is blocked in an early step of the GA-biosynthesis pathway before the biosynthesis of GA_{12}-aldehyde and after the biosynthesis of *ent*-kaurene. We have transposon-tagged the *d3* locus using Robertson's Mutator. The mutant allele *d3-2(Mu)* had a linked *Mu8* element. The flanking DNA was cloned and shown to be very tightly linked to the *d3* locus by mapping in a high resolution population developed by selecting

for recombination between *d3* and the linked genetic markers *wx* and *gl15*. In this work a total of 754 chromosomes have been examined for recombination between the cloned fragment and *d3*, and none have been observed, indicating tight linkage of the clone to the *d3* locus. To identify the nature of the *d3* gene product cDNAs were isolated from a light-grown seedling library and a vegetative meristem library. Database comparisons revealed that the predicted *D3* protein had significant sequence similarity to cytochrome P450s. It has the highly conserved cytochrome P450 signature sequence (FXXGXXXCXG). The cysteine of the signature sequence is involved in binding heme Fe.

To unambiguously establish the identity of the *d3* gene, a second mutant allele of *d3* (*d3-4*) was cloned and characterized. Maize *d3-4* has a novel 327 bp insertion element, named *Sleepy*, in an exon. The *Sleepy* insertion is flanked by five bp direct repeats. Analysis of *D3* mRNA expression by RT-PCR showed that the *D3* transcript is expressed in roots, developing leaves and the vegetative meristem. *D3* mRNA was observed to be reduced in two mutants, *d3-2(Mu)* and *d3-5*. Other recently identified steps in the pathway in combination with maize *d3* will now allow a molecular approach to GA-biosynthesis. It will be necessary to determine the developmental sites of GA-biosynthesis and how GA-biosynthesis enzymes are regulated in order to formulate insightful models of how plants use this phytohormone to regulate their growth and development. Additionally it is interesting to note that a number of quantitative trait loci for maize height have been mapped near genes involved in GA-biosynthesis and reception. In particular, allelic variation at the *d3* locus has been proposed to be the basis of a quantitative trait locus that has been defined for a naturally occurring height variant in maize (Touzet et al. submitted).

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Expression of *o2* modifiers in normal maize genotypes

--Glóverson L. Moro, Maurício A. Lopes, Bruce Hamaker and Brian Larkins

The effects of *opaque2* (*o2*) modifier genes are well described in Quality Protein Maize (QPM). However, all the available information describes the effects of these genes in *o2* backgrounds. Very little is known about the kind of biochemical and physical changes *o2* modifier genes cause in wild-type (*O2*) endosperm. In order to address these questions, we used a backcross scheme to develop genotypes containing *o2* modifiers and the *O2* allele. The W64A line was used as source of the normal *Opaque2* allele and Pool 34 QPM was used as recurrent parent and source of modifier genes. After each round of backcrossing, we visually selected against the *o2* allele. Selected BC5 plants went through three generations of selfing to eliminate the *o2* mutation. Ultimately, the presence of the *O2* allele in BC5-S3 plants was confirmed by Southern analysis with an *O2* cDNA clone. The 13 BC5-S3 plants homozygous for the *O2* allele were selfed and their kernels (BC5-S4) were analyzed.

As observed in QPMs, modified normal kernels show a high content of γ-zein, but this is not necessarily followed by an increase in kernel density or hardness. There was no statistical difference in kernel density among the different inbreds and the two original parents (Pool 34 QPM and W64A). Hardness, as measured by the Stenvert test, showed a broad range of variation and was weakly correlated with γ-zein content ($r = 0.40$, $P = 0.098$). However, based on the range of Stenvert values (19.5 to 28.4), all the inbreds tested would be considered to have a very hard endosperm. Therefore, it seems that there is a threshold of density and hardness in normal endosperm above which the effects of γ-zein are not evident.

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Tests of male sterile mutants

--Earl Patterson

Many nuclear male sterile genes have been assembled over a long period of years. I am indebted to several geneticists for submitting seed sample sources of male sterile types that occurred unexpectedly in their research plantings.

Upon receipt, each accession was tested initially to determine whether the sterility was cytoplasmically transmitted. For this purpose, the standard commercial inbred line WF9 has been useful since its nuclear genotype is nonrestoring for each of the cytoplasmic male sterile types *cms-C*, *cms-T* and *cms-S*. The procedure followed was to pollinate male sterile plants by WF9. If the progeny were male sterile, there was presumptive evidence that the sterility was cytoplasmically transmitted. Further tests were then conducted using diagnostic lines whose genotypes of nuclear restorer genes differentiate among *cms-C*, *cms-T* and *cms-S* cytoplasms.

These screening tests were conducted in order to eliminate cytoplasmic male steriles from further study. Screening with additional diagnostic lines provided evidence consistent with assignment to *cms-T* (4 sources) or *cms-S* (9 sources). Further confirmations of these assignments by tests of cytoplasmic components were not made. Tested sources are listed in Table 1.

Table 1. Cytoplasmic male sterile sources.

cms-S	*cms-T*
RJL 5399	RJL 5209
USSR 2-27	68-571
USSR 29-54	Golden Glow (Palmer) (PI)
PI 213787	Manwiller (PI)
PI 214199	
PI 217219	
PI 262489	
PI 262500	
PI 267212	

PI sources represent numbered Plant Introduction accessions of maize varieties, or named varieties of PI accessions. PI accessions are propagated at the Regional Plant Introduction Station, Ames, Iowa. Seed samples are available from Ames upon request. PI262500 carried both cytoplasmic and nuclear male sterility.

All instances of male sterility with an apparent nuclear mode of inheritance were further confirmed by male transmission. The procedure was as follows. Male sterile plants that occurred in plantings of accessions were pollinated by WF9. F1 plants were self-pollinated and crossed as male parents on one or more standard commercial inbred lines free of nuclear male sterile alleles and carrying normal (non-sterile) cytoplasm. From selfing

of F1 plants, F2 progenies were grown to confirm the segregation of male sterility. Progenies were also grown from the pollination of standard commercial lines by the same F1 plants. Several plants in each family were self-pollinated and progenies grown the following generation; it is expected that half of these progenies will segregate for male sterility due to recessive nuclear male sterile alleles that have been pollen-transmitted.

Table 2. Nuclear male sterile gene sources.

Previous Designation	Information from Tests	New Designation
ACCO 1752	= *ms10*	*ms10-6001*
Alex 21096	= *ms2*	*ms2-6002*
Alex 78165		*ms*-6003*
Alex 78193	Group 1 (7L)	*ms*-6004*
Bear 1	= *ms14*	*ms14-6005*
Bear 2	Group 6 (9L)	*ms*-6006*
Bear 3	= *ms7*	*ms7-6007*
Bear 4	Group 3 (3L)	*ms3-6008*
Bear 5	Group 3 (3L)	*ms3-6009*
Bear 6	Group 1 (7L)	*ms*-6010*
Bear 7	Group 2 (9L)	*ms*-6011*
Bear 8	= *ms2*	*ms2-6012*
Bear 10	Group 1 (7L)	*ms*-6013*
Bear 11	Group 1 (7L)	*ms*-6014*
Holden 4439	= Holden 4442	*ms*-6015*
Holden 4442	= Holden 4439	*ms*-6016*
Holden 7469	= *si1-at*	*si1-at-6017*
Hooker 3879	Group 2 (9L)	*ms*-6018*
Hooker 5345	Group 7 (2L)	*ms*-6019*
Hooker 5472	Group 3 (3L)	*ms3-6020*
Hooker 8472	Group 4 (9L)	*ms*-6021*
Hooker 8508	Group 4 (9L)	*ms*-6022*
RJL (Lambert) M1		*ms*-6023*
RJL (Lambert) M11	Group 7 (2L)	*ms*-6024*
RJL (Lambert) M19		*ms*-6025*
RJL (Lambert) M37		*ms*-6026*
RJL (Lambert) M59	Group 2 (9L)	*ms*-6027*
RJL (Lambert) M70		*ms*-6028*
RJL (Lambert) M71	Group 7 (2L)	*ms*-6029*
RJL (Lambert) M72	= *si1-at*	*si1-at-6030*
RJL (Lambert) B76	Group 2 (9L)	*ms*-6031*
RJL (Lambert) 1015	= *ms9*	*ms9-6032*
RJL (Lambert) 1871		*ms*-6033*
RJL (Lambert) 5602	Group 8 (1S)	*ms*-6034*
RJL (Lambert) 7029	= *ms10*	*ms10-6035*
(RJL) PI 245138		*ms*-6036*
(RJL) PI 267218	= *ms9*	*ms9-6037*
(RJL) PI 262500	Group 7 (2L)	*ms*-6038*
JRL (Laughnan) 303		*ms*-6039*
JRL (Laughnan) 305		*ms*-6040*
JRL (Laughnan) 308	Group 7 (2L)	*ms*-6041*
JRL (Laughnan) 1623	= *ms9*	*ms9-6042*
JRL (Laughnan) 1638	Group 3 (3L)	*ms3-6043*
Sprague 75-6	Group 8 (1S)	*ms*-6044*
Sprague 091		*ms*-6045*
Sprague 111	Group 4 (9L)	*ms*-6046*
Sprague 345	Group 4 (9L)	*ms*-6047*
Sprague 391		*ms*-6048*
Sprague 398		*ms*-6049*
COOP 56-112	= *ms1*	*ms1-6050*
COOP *r1-g wx1*	= *ms11*	*ms11-6051*
COOP *adh-ms* W23		*ms*-6052*
Recent acquisitions:		
JRL 1086		*ms*-6053*
JRL 1930		*ms*-6054*
JRL 2138		*ms*-6055*
Sprague 0007		*ms*-6056*
Sprague 0035		*ms*-6057*
Sprague 0239		*ms*-6058*
Sprague 0268		*ms*-6059*
Sprague 0406		*ms*-6060*
Sprague 0683		*ms*-6061*
Sprague 0686		*ms*-6062*
Sprague 0851		*ms*-6063*
Steffensen 0709		*ms*-6064*
COOP 0574		*ms*-6065*
COOP 1699		*ms*-6066*

The information included in Table 2 is intended to serve several purposes. In the first column are indicated the previous temporary laboratory designations of male sterile gene stocks. In the last column the proposed new designations are indicated, carrying a 4-digit laboratory number in order to conform to maize terminology rules. Since limited distribution of some of these stocks has been made to a few geneticists under the old designations, the listing of old and new designations will permit them to relate their stocks to the new symbols.

The new temporary symbols consist initially of a generic symbol for the category of the trait; here the symbol *ms* has been chosen. An asterisk denotes a temporary gene designation and the 4-digit laboratory number differentiates a specific male sterile gene source from others. There is no assurance even that the *ms* symbol will be valid in future since the mutant allele may prove to be allelic to a locus named by some other symbol (e.g., *as1* or *si1*). Once a particular gene in this collection has been shown to be allelic to a symbolized locus, then that permanent symbol is used, followed by a dash and retention of the 4-digit laboratory designation to distinguish the allele itself. In the event the new gene source is not allelic to any currently symbolized gene, a new gene locus symbol may be assigned. The first gene symbol in the listing (*ms10-6001*) indicates that the male sterile gene originally carried as ACCO 1752 proved to be allelic to *ms10* and is now designated as allele 6001 at that locus. The new symbol *si1-at-6017* is an example of an instance in which the generic symbol (ms) was replaced when allelism with *si1* was found.

The middle column in Table 2 indicates positive outcomes of allelism tests with symbolized loci, or assignments to groups based on allelism with each other and common location within identified chromosome segments.

All the mutant male sterile alleles reported here have shown a recessive expression. As indicated in Table 2, some of them have been assigned to one of eight groups, mostly on evidence that the male sterile alleles have been "uncovered" (are present in hemizygous condition, ms/-) in plants hypoploid for a B-A translocation. Members of the same group are allelic with each other.

Evidence for the different groups is as follows:

Group 1 (7L)--members of this group are uncovered in plants hypoploid for TB-7Lb. They apparently are not allelic to *ms7* and there is no clear indication of allelism with *va1*. Included in this group are *ms*-6004*, *ms*-6010*, *ms*-6013* and *ms*-6014*.

Group 2 (proximal 9L)--Uncovered by hypoploid TB-9Lc and linked to hypoploid TB-9Sb. Not allelic to *ms2* nor to male steriles in Groups 4 or 6. Included in this group are *ms*-6011*, *ms*-6018*, *ms*-6027* and *ms*-6031*.

Group 3 (3L)--Uncovered by hypoploids of the compound TB-3La-2S6270. Allelic to *ms3*, which is the basis for assignment to the long arm of chromosome 3. The locus of *ms3* is thus within the segment bounded by 3L.1 (the interchange point of TB-3La) and 3L6, the chromosome 3 interchange point of T2-3 6270. Included in this group are *ms3-6008*, *ms3-6009*, *ms3-6020* and *ms3-6043*.

Group 4 (distal 9L)--Uncovered by hypoploid TB-9Lc and hypoploid TB-9La. Not allelic to male steriles in Groups 2 or 6. Alleles at this locus show about 10 percent recombination with *Bf1*. Included in this group are *ms*-6021*, *ms*-6022*, *ms*-6046*

and *ms*-6047*.

Group 5 (1S)--Allelic to *ms9*. Uncovered by hypoploid TB-1Sb and hypoploid TB-1Sb-2L4464. The locus of *ms9* is thus within the segment bounded by 1S.05 and 1S.53. Included in this group are *ms9-6032, ms9-6037* and *ms9-6042*.

Group 6 (distal 9L)--Uncovered by hypoploid TB-9Lc and hypoploid TB-9La. Not allelic to male steriles in Groups 2 or 4. Included as an allele at this locus is *ms*-6006*.

Group 7 (distal 2L)--Uncovered by hypoploid TB-1Sb-2L4464, but not by hypoploid TB-1Sb. Assignment to 2L also confirmed by the fact that alleles at this locus show about 15 percent recombination with *Ch1*. They are also uncovered in duplicate-deficient plants derived from T2-5f and T2-8(8376), that are deficient for terminal segments of the long arm of chromosome 2. Included in this group are *ms*-6019, ms*-6024, ms*-6029, ms*-6038* and *ms*-6041*.

Group 8 (1S)--Uncovered by TB-1Sb and hypoploid TB-1Sb-2L4464. The male sterile locus is thus within the segment bounded by 1S.05 and 1S.53. Tests for allelism with *as1*, *ms9*, *ms12*, *ms14* and *ms17* all have appeared to be negative. Included in this group are *ms*-6034* and *ms*-6044*.

Included in this report is a listing of 79 male sterile sources. Of these, 13 have shown a cytoplasmic mode of inheritance. Of the nuclear gene sources assembled more than ten years ago, 17 appear to be alleles of male sterile genes with permanent symbols, 20 have been located to chromosome, but not shown to be alleles of male sterile loci with permanent gene symbols and 15 are unlocated to chromosome and lacking unambiguous indication of allelism with male sterile gene loci with permanent gene symbols. An additional 14 sources are recent acquisitions that are being perpetuated and evaluated, but which have not been subjected to a significant amount of testing. In general, allelism testing of various sources has not included tests with male sterile genes assigned permanent symbols during the past ten years.

In the course of allelism testing, repeats of test plantings in different seasons have frequently exhibited erratic and differing results. This result has occurred even when the same seed source has been planted in different seasons. The ambiguous results have been mainly of two types: (1) different results observed from allelism test crossing made in reciprocal directions, and (2) in families containing male sterile plants, the occurrence of such plants in frequencies often lower than expected from a cross yielding a positive allelism result; it is as if many plants homozygous for a male sterile allele had expressed male fertility. The same result could be produced by the action of gametophyte factors operating in conjunction with linked male sterile gene loci. This possibility does not seem at all likely, however, since the number of male sterile gene loci in the genome showing aberrant ratios would require postulation of numerous instances of linkage with gametophyte factors showing fortuitous linkage. The widespread distortion of ratios might also occur if alleles at male sterile gene loci might themselves act as gametophyte factors under some conditions. This might particularly be true of mutant alleles arising from insertion or removal of transposable elements. Whatever the actual reason, there is a strong suggestion that the expression of male sterility may be strongly modified by background genotypes and/or environmental influences. It is noteworthy that Marc Albertsen, in his plantings of some of these male sterile sources, has experienced the same kinds of anomalous and ambiguous results (personal communication).

The expression of male sterility appears to be more consistent when recessive male sterile alleles are present in hemizygous condition rather than as homozygotes. The preferred strategy, then, may be to locate male sterile genes to specific chromosome regions by hemizygosity in hypoploid B-A translocation plants or by linkage to the deficient segment in hypoploid plants. As a second stage, allelism would need to be tested only by direct crosses among male sterile genes located in the same general chromosome regions.

Numerous repeats of tests have been conducted in order to obtain results deemed suitable for a convincing assessment of the question of allelism. As a result, it is expected that virtually all conclusions of allelism reported here will be validated by future investigations. However, if anyone requests seed samples of male sterile sources reported to represent alleles at a particular locus with a view to testing for allelic differences, it would be only prudent to reconfirm that the sources are indeed allelic before embarking on further labor-intensive or expensive investigations. It should also be pointed out that two or more allelic male sterile sources submitted by the same person have a higher probability of representing identical alleles than if submitted by different people, since alleles unknowingly transmitted through different lineages or plantings at one laboratory may frequently be detected separately, yet trace back to a common pedigree source in an earlier generation.

Relationship between genome size and germination under cold conditions in Southwestern US maize populations

-- M. Afzal, D. P. Biradar, and A. Lane Rayburn

Several laboratories including ours have documented extensive variation in maize genome size. Genome size has been correlated with latitude (Laurie and Bennett, Heredity 55:307-313, 1985; Rayburn et al., Am. J. Bot. 72:1610-1617, 1985; Tito et al., TAG 83:58-64, 1991), altitude (Rayburn and Auger, TAG 79:470-474, 1990; Rayburn, Evol. Trends Plants 4:53-57, 1990), effective growing season (Bullock and Rayburn, Maydica 36:247-250, 1991), flowering time (Rayburn et al., Plant Breed. 112:318-322, 1994), and overall agronomic performance (Biradar et al., TAG 88:557-560, 1994).

With respect to cold tolerance, McMurphy and Rayburn (Plant Breed. 106:190-195, 1991) noted no clear trend in genome size. This study concentrated on populations which had been artificially selected for cold tolerance. The purpose of this study was to determine if, under natural selection, a correlation between cold tolerance and genome size exists.

Lines used in this study were open-pollinated Southwestern US Indian maize populations. The populations were obtained from North Central Regional Plant Introduction Station at Ames, Iowa. Nine of these populations originated in New Mexico and fourteen populations were from Arizona. Five seeds from each of these populations were sown in two replications. The pots were then kept in a cold chamber at a constant temperature of 11.5 C. The number of kernels germinated was recorded. In order to insure that the kernels used were of good quality, germination was also performed at 32 C. The relative percentage cold germination was calculated by dividing the percent germination at 11.5 C by the percent germination at 32 C.

Nuclear DNA content was determined according to the procedure of Rayburn et al., (J. Exp. Bot. 40:1179-1185, 1989). Nuclear DNA content of the populations was expressed with

respect to A619 (= 100.00 arbitrary units) which was used as an external standard. Correlation analysis was carried out by using the relative cold germination percentage and the estimated genome sizes of the populations used in this study.

A wide variation in both of these parameters was observed among the populations studied. Genome size ranged from 95.5 to 116.2 AU, while the corrected germination percentage ranged from 20.0 to 100.0. A significant negative correlation (R = -0.44; p = 0.04) was observed between genome size and germination percentage of all the populations studied (Fig.1).

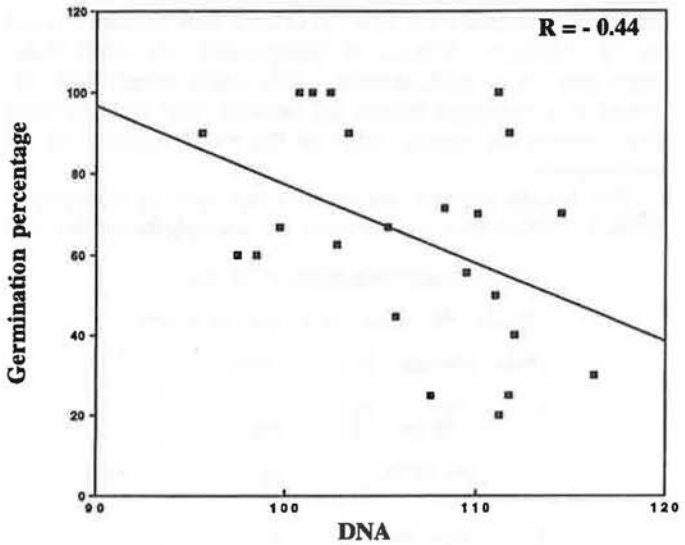

Figure 1. Correlations between DNA content and cold germination percentage of different populations.

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d2 allelic to *d3*?

--Philip Stinard

Maize COOP notes from the 1970's indicate that the COOP's accessions of *d2* (chromosome 3) and *d3* (chromosome 9) were found to be allelic. Both stocks trace back to the first season that maize genetic stocks were grown for the COOP in Cornell in 1935 and therefore the original accessions were most likely allelic as well. We suspect that the problem is with the *d2* stock, because both stocks show tight coupling of the dwarf to *wx1* on chromosome 9, which is what one would expect of *d3*. *d2* should show linkage to chromosome 3 markers. Attempts have been made to obtain independent sources of *d2* (most recently an accession obtained from Nicholas Harberd), but so far they have proven to be allelic to *d3* as well. It is possible that the original placement of *d2* to chromosome 3 was erroneous and that the original *d2* mutant was allelic to *d3* all along. The other possibility is that the stocks were confused before they were sent to the COOP for propagation. We are in the process of checking other sources of *d2* for allelism with *d3*. If anyone has additional sources of *d2* that do not trace back to the Maize COOP, we would appreciate receiving them and will test them for allelism as well.

Two-point linkage data for *gl1 Bn1* on chromosome 7

--Philip Stinard

This report summarizes the results of a two-point linkage study of *gl1* and *Bn1* on the long arm of chromosome 7. The test was set up as a repulsion backcross as follows: (*y1 gl1 bn1 / y1 Gl1 Bn1*) X *y1 gl1 bn1* (the test was done in a *y1* background in order to facilitate scoring of *Bn1*). The kernels from this cross were separated on the basis of *Bn1* vs. *bn1* phenotype and separately planted and grown to seedling stage in sand benches. The seedlings were scored for *gl1*. The results of this test are presented here. The *gl1* - *Bn1* distance of 27.8 centimorgans is reasonably consistent with the distance presented on the current linkage map (35 centimorgans).

Reg.	Phenotype	No.
0	*gl1* +	449
	+ *Bn1*	463
1	*gl1 Bn1*	165
	+ +	186
Total		1263

% recombination *gl1* - *Bn1* = 27.8 ± 1.3

gl13 recovered from Maize COOP stocks

--Philip Stinard

gl13, once thought to be lost, has been recovered from stocks at the Maize COOP Stock Center. The mutant is referred to by Anderson in MNL 29:5-6 (1955; the reference to *gl13* in the 1993 and 1994 MNL gene lists is a mistaken reference to a *gl3* allele). Mutant *gl13* seedlings express a good glossy phenotype, although the seedlings may be somewhat twisted and necrotic. Seedlings that survive give rise to small plants with sterile tassels and tiny ears that do not set seed under Illinois conditions. Anderson's unpublished notes indicate possible location of *gl13* to chromosome 5 based on F2 data showing linkage (repulsion) of *gl13* with *wx1* in the *wx1*-marked translocation T5-9c (5S.07, 9L.10). Recently increased stocks of *gl13* are now available from the Maize COOP Stock Center.

The dominant pale endosperm factor *Iy1* is allelic to *Wc1*

--Philip Stinard

The Maize COOP has been maintaining a dominant pale endosperm factor named *Iy1* (*Inhibitor-of-Y1*, McWhirter, MNL 41:175, 1967). Because the mutant *White cap1* (*Wc1*) has a similar phenotype, an allelism test between the two mutants was conducted. A homozygous *Y1 Wc1* line was crossed by a homozygous *Y1 Iy1* line, and the F1 was backcrossed by a homozygous *Y1 wc1* line. All of the backcross progeny kernels had the *Wc1* phenotype, indicating either allelism, or very tight linkage between the two mutants. Since the number of progeny kernels was large (n = 4,458), it is likely that the two mutants are allelic. Since *Wc1* has precedence in the literature, the *Iy1* allele should be renamed *Wc1-Iy1*.

Isolation of a new allele of *Bn1*

--Philip Stinard

When a white endosperm *y1* line is crossed to a *Y1* line and the F1 is selfed, it is very common to find a pale yellow kernel phenotype segregating 3:1 pale yellow : white among the *y1* class

of the F2. The genetics of this phenomenon has not been reported upon. Because the phenotype of the pale yellow kernels is similar to that of *Bn1* (*Brown aleurone1*, accumulation of yellow pigment in the aleurone layer, most easily seen on white endosperm backgrounds), an allelism test was conducted between one of these unknown pale yellow aleurone factors and *Bn1*. The particular pale yellow aleurone allele chosen for this test, *Bn*-PSS*, came from a *rgd1 y1* stock that had been crossed to our M14/L317 standard lines a few generations previously, followed by several generations of selfing, and was now homozygous for pale yellow aleurone on *y1* endosperm. The homozygous *y1 Bn*-PSS* line was crossed to the Maize COOP's *y1 Bn1* line, and the F1 was backcrossed by a homozygous *y1 bn1* line. All of the kernels on the backcross ears were pale yellow, indicating either allelism or tight linkage of the two mutants. Since the number of kernels on the ears was rather large (n = 3,799), the mutants are most likely allelic. *Bn*-PSS* has been redesignated *Bn1-PSS*.

When working with *y1* and other endosperm carotenoid mutants, it is desirable to have a genetic background that is free of factors that obscure the mutant phenotype. The pale yellow aleurone color present in *Bn1* mutant kernels can sometimes mask the mutant phenotype of certain carotenoid mutants, such as *y9*, that are only slightly paler than the nonmutant yellow endosperm phenotype. Thus, the COOP is undertaking a survey of some of its more commonly used inbred lines to determine which ones carry *Bn1*. The lines under study are A632, A636, B73, B77, B79, H99, M14, Mo17, Oh43, Q66, Q67, W22, and W23. The inbred lines were crossed by *y1 Bn1*, and the F1's will be crossed by a *y1 bn1* and the ears scored for *Bn1* segregation vs. *Bn1* homozygosity. The results should be ready in time for next year's Maize Newsletter.

pb1 is allelic to *y1*

--Philip Stinard

The mutant *pb1* is a temperature-sensitive mutant that produces patches of lighter color on its leaves when grown at high temperatures (35 C). The COOP's stocks of *pb1* all show complete linkage of *pb1* with *y1*. Because there exist temperature-sensitive alleles at the *y1* locus that produce phenotypes similar to that of *pb1* (Robertson and Anderson, J. Hered. 52:53-60, 1961), allelism tests were conducted between *pb1* and the temperature-sensitive *y1* allele *y1-wmut* (as well as with an unplaced temperature-sensitive piebald mutant, *pb*-Burnham*). Crosses were made between *pb1* and *pb*-Burnham* as well as between *y1-wmut* and *pb*-Burnham*. (No crosses were made directly between *pb1* and *y1-wmut*.) Kernels from these crosses were planted in flats of soil and grown in a 35 C growth chamber. The resulting seedlings all showed the high temperature piebald phenotype. Nonmutant control kernels produced green seedlings under the same conditions. Thus, these three mutants are allelic. We have renamed the *pb1* and *pb*-Burnham* alleles *y1-pb1* and *y1-pbBurnham*, respectively.

sh5 is allelic to *bt1*

--Philip Stinard

The two mutants *sh5* and *bt1* have similar phenotypes (shrunken/collapsed endosperms) and map to the same region of chromosome 5. To test the relationship between these two mutants, plants homozygous for *sh5* were crossed to plants heterozygous for *bt1*. In all instances, the progeny segregated 1:1 for plump : shrunken kernels, indicating allelism of these two mutants. Since *bt1* has precedence over *sh5*, the name *bt1-sh5* is proposed for the *sh5* allele.

Three-point linkage data for *Wc1 Bf1 bm4* on 9L

--Philip Stinard

The question of the order of the genes *Bf1* and *bm4* at the tip of the long arm of chromosome 9 is an important one for those studying deletions and other cytological phenomena on 9L. To date, the placement of the order of *Bf1* and *bm4* has been based on the compilation of two-point linkage data. No direct three-point tests have been reported. This report summarizes the results of a three-point linkage test between *Wc1*, *Bf1*, and *bm4* that confirms the current order on the maize linkage map of chromosome 9.

The linkage test was set up as a testcross as indicated in Table 1. Kernels from the testcross ear were planted in the field and the resulting plants were self-pollinated. Mature plants were scored for *bm4* within a few days after pollination. The self-pollinated ears were scored for *Wc1*. Approximately 40 kernels from each ear were grown to seedling stage in a sand bench, and the seedlings were scored for *Bf1* using a UV light. The following linkage relationships were established (distances are given in centimorgans): *Wc1* - 10.9 - *Bf1* - 4.4 - *bm4*. These data confirm the order of these three genes on chromosome 9, but the distance between *Wc1* and *Bf1* is much less than that reported on the current linkage map (30 centimorgans, based on the data of Burnham, MNL 33:74, 1959). The data of Coe (MNL 38:109, 1964 and MNL 61:47, 1987), which give a *Wc1* - *Bf1* distance of 16 centimorgans, are more consistent with the data presented in this report.

Table 1. Three-point linkage data for *Wc1 Bf1 bm4*.

Testcross: (*Wc1 bf1 Bm4* / *wc1 Bf1 bm4*) X *wc1 bf1 bm4*

Region	Phenotype	No.	Totals
0	*Wc1* + +	160	
	+ *Bf1 bm4*	130	290
1	*Wc1 Bf1 bm4*		15
	+ + +	20	35
2	*Wc1* + *bm4*		7
	+ *Bf1* +	6	13
1+2	*Wc1 Bf1* +	1	
	+ + *bm4*	1	2
Total			340

% recombination *wc1* - *bf1* = 10.9 ± 1.7
% recombination *bf1* - *bm4* = 4.4 ± 1.1

ws2 is allelic to *g1*

--Philip Stinard

The mutants known as the duplicate factor pair *ws1 ws2* (*white-sheath1 white-sheath2*) have a long and confusing history. Two separate mutants known as *ws1* and *ws2*, each with distinct phenotypes, were maintained by the Maize Genetics COOP Stock Center for many years. Confusion probably arose when it was found that *ws1* showed possible duplicate factor (15:1) inheritance. That, combined with the fact that both mutants arose

from the same maize variety (Kempton, J. Hered. 12:224-226, 1921), led to the labelling of both stocks as the duplicate factors '*ws1 ws2*.' 1982 was the last time that the original *ws1* line maintained by the COOP was propagated. Some time after that, it was discarded and only the *ws2* line was kept.

A note made in a field book in 1963 indicated that *ws2* was allelic to *g1*. To test this possible allelism, *ws2* plants were crossed to *g1* plants in the COOP's 1994 summer nursery. Progeny of this cross grown to seedling stage in the sand bench did indeed produce a golden phenotype, indicating allelism. The *ws2* allele has been renamed *g1-ws2*.

Although the COOP's original accession of *ws1* was lost, it may have been rediscovered among the COOP's unplaced white sheath mutant collection. One stock, which has the name *ws1 ws2-Pawnee*, has a phenotype identical to that described by Kempton for *ws1*. Furthermore, the variety of corn in which his mutant arose was named 'Pawnee.' The COOP is conducting further tests to determine whether *ws1 ws2-Pawnee* shows duplicate factor inheritance.

y3 is allelic to *al1*

--Philip Stinard

y3 and *al1* are pale yellow endosperm/albescent seedling mutants that map to the same region of the short arm of chromosome 2 (Perry and Sprague, J. Amer. Soc. Agron. 28:990-996, 1936). For many years they have been listed as separate, tightly linked loci, with *y3* conditioning the pale yellow endosperm phenotype and *al1* conditioning the albescent seedling phenotype. However, these two traits have never been unambiguously separated by crossing over, and the possibility remains that *y3/al1* is a single gene with pleiotropic effects. In order to investigate this possibility, crosses were made in the Maize COOP's summer 1994 nursery between two sources of *y3* (tracing back to G. F. Sprague and E. A. Graner, respectively) and an *al1* allele (*al1-Brawn*; Robertson, J. Hered. 66:67-74, 1975) obtained from D. S. Robertson. All crosses between *y3* and *al1-Brawn* produced pale yellow endosperm kernels which when planted in the sand bench gave rise to albescent seedlings. The likelihood of both the *y3* and the *al1-Brawn* stocks independently carrying the same two tightly linked mutants is very low. Thus, the two mutants are almost certainly alleles at the same locus. Since *al1* has precedence in the literature, it is suggested that the gene symbol *al1* be retained for the locus, and that *y3* be named *al1-y3*.

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Selection for anther culture ability

--V.H. Beaumont and J.M. Widholm

Anther culture ability is the ability of a genotype to induce microspore division and embryo formation from microspores , once the anthers are plated in appropriate conditions. This trait is strongly genotype-dependent in maize with most genotypes being unresponsive (Petolino and Jones, Crop Sci. 26:1072-1074, 1986). It is generally explained with a few dominant genes (Theor. Appl. Genet. 80:459-464, 1990). The F1 hybrid Pa91 x FR16 was previously found to be responsive and some F2 plants from this cross gave high yields in our anther culture system. Therefore, we started a selection program with the progeny.

All the anthers plated came from donor plants grown in the field (Champaign, Illinois) during the summers 1992 to 1994. The anther culture protocol has been described elsewhere (Beaumont et al., MNL66:114-115, 1992). The variables E/100A (number of embryos produced for 100 anthers plated) and % Resp.P (percentage of plants producing at least one embryo) were recorded. Because of the non-normality of the distributions, we chose non-parametric tests to analyse the data: the Wilcoxon (= Mann-Whitney) test was used to analyse E/100A and the χ^2 test was used to analyse %RESP.P.

The number of plants and anthers plated are given in Table 1. In 1992, the percentage of responding plants was not recorded for F1 hybrids. The F2 generation was anther cultured in 1992 but we did not select in this generation since the tassels were used for anther culture and the plants could not be self-pollinated. 43 F3 families were produced in 1992 and evaluated for their anther culture ability in 1993. Five F3 families were selected for their anther culture ability (marked F3Sel). Two plants in each selected F3 family were selfed, giving rise to ten F4 subfamilies (marked 5 x 2 in the table). All F4 families from selected F3's were anther cultured in 1994. Results from the best F4 (F4Sel) are given in Table 1.

Table 1: Selection for anther culture ability from the cross Pa91 x FR16 (PF). Geno: genotype, E/100A: number of embryos for 100 anthers plated, %RESP. P: percentage of responding plants, ND: not determined.

Geno	Families	Plants	Anthers	E/100A	%RESP.P
Plated in 1992:					
FR16	-	4	270	5.9	
Pa91	-	5	300	1.7	
PF F1	-	ND	1050	13.9	ND
PF F2	-	51	2520	12.6	74%
Plated in 1993:					
FR16	-	4	240	1.7	
Pa91	-	5	300	0.3	
PF F3	43	108	5430	3.4	33%
F3Sel	5	10	540	16.1	70%
HFI		70	2100	27.3	66%
Plated in 1994:					
FR16	-	1	30	3.3	
Pa91	-	3	90	0	
PF F4	5 x 2	67	3420	7.3	36%
F4Sel	1 (#6B)	9	480	31.2	56%
HFI	-	62	1860	28.0	34%

This progeny was anther cultured during three different years. Therefore, we had to compare the results to a common control. The parents FR16 and Pa91 do not produce enough embryos to be evaluated precisely. The F1 hybrid could have been a good control but the lack of seeds did not allow us to plate anthers in 1993. We chose to compare the results obtained to the genotype HFI (haplodiploid from anther culture of H99 x FR16) during the corresponding year. Since HFI might not react the same way as the PA91 x FR16 progeny, the comparisons obtained must be interpreted cautiously (Fig. 1).

The yields of the 5 F4 families are higher than their F3, for the E/100A ($P < 0.1$) and %RESP.P ($P < 0.001$). Altogether, the F4 families gave 36% responding plants (Table 1), but all families produced embryos (100% responding F4 families). Thus, the

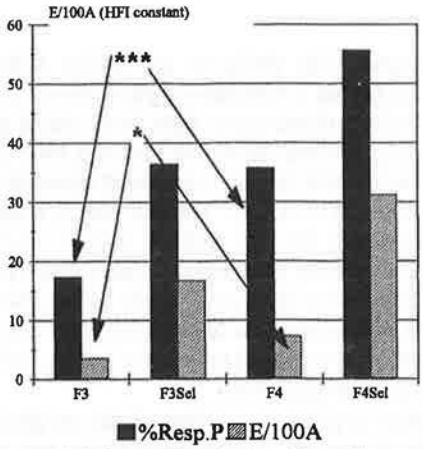

Figure 1: Anther culture ability of successive generations. The results are corrected assuming HFI to be constant over the years. Sel: selected families (see Table 1). The results obtained in the F3 generation were compared to those obtained in the F4, with the Wilcoxon test for the variable E/100A and the χ^2 test for the variable %RESP.P. Significant differences are noted. *:P < 0.1; **: P < 0.01; ***: P < 0.001.

selected F3's represent a good starting material for further selection. The genotype PFF4#6B seems very interesting since its number of embryos (E/100A) and percentage of responding plants (%RESP.P) are higher than HFI for the same year. These results demonstrate that breeding for anther culture ability is possible. We will continue this program and also cross HFI with PFF4#6B for further selection.

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Temperature sensitivity in ornamental corn and kale

--Walton C. Galinat

The temperature-sensitive chlorophyll and carotenoid factors of corn, especially *al* (snow white due to lack of protection by carotene), *cl1* (chr. 3) and its modifier, *clm* (chr. 8) have counterparts in the ornamental cabbage-kales, which have the advantage over corn in continuing to grow vigorously at temperatures in the 30's just above freezing (and are edible and beautiful outdoors all winter). For a listing of about 225 such genes in corn, see Coe et al. 1988, pages 172-176 of Sprague & Dudley, Corn and Corn Improvement. As the synthesis of chlorophyll fails due to temperatures below 55 F, the synthesis of anthocyanin increases in both corn (*A, B, Pl, R*) and its unnamed counterpart mutants in cabbage-kale. Thus, for greatest beauty in both japonica (*j*) striped corn plants (termed Rainbow Corn by Luther Burbank) and ornamental cabbage-kale grown for display purposes, planting should be delayed until early July -- so that plant growth will still be active as the temperature drops towards autumn. The ornamental cabbage-kale will remain beautiful through most of the winter as the only color in an otherwise bleak landscape. In the best selected material, a central disk of scarlet red over albino is framed by a green margin. Leaf types vary from flat (cabbage), crinkle (kale) to lace (deeply cut) of endive type.

A return to the wild high-density conditions

--Walton C. Galinat

Teosinte, as the wild progenitor of maize, was adapted for survival under the stress of high population density. The competition for space and sunshine came from other teosinte plants as well as other species of wild plants. The survival of teosinte required adaptation to population stress.

During domestication, the developing maize changed its adaptation to low density conditions resulting from slash and burn clearing of land before planting and weed removal afterwards. In a few cases (Oloton), the competition for sunshine was won by evolving giant stalks that would tower up over small trees and shrubs. Water was necessary sometimes from irrigation or near seepage areas at the base of mesas in the desert. When intercropping maize with other food plants such as beans and squash, the maize was spread out in space or time so that there was no competition between canopies for sunshine. In the several hundreds of races of maize, including modern varieties of sweet corn, in almost every case the adaptation is to low density conditions.

During the last ten or more years, plant breeding for adaptation to crowding resulting from new technologies for high density planting simulates a return to the wild, high-density conditions of 8,000 years ago. The adaption now comes from smaller erect leaves, and slightly reduced plants and ears together with greatly reduced tassels. This is the new industrial maize of high density monoculture that is flooding the world market with enormous yields of cheap feed corn.

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Detection of high levels of polymorphism among dent and popcorn inbred lines using Inter-Simple Sequence Repeat (ISSR) amplification technique

--R.V. Kantety, X. Zeng, J.L. Bennetzen, B.E. Zehr

Nineteen elite popcorn inbred lines and eight dent corn inbred lines were used in a study to investigate the degree of polymorphism resulting from a novel DNA marker technique, Inter-Simple Sequence Repeat (ISSR) amplification (Zietkiewicz et al., Genomics 20:176-183, 1994). ISSR involves the use of designed primers to anchor a subset of tandem di-, tri-, or tetra-nucleotide repeats in the genome, resulting in amplification of the DNA sequence between two repeat units. This technique combines the advantages of RAPDs (Random Amplified Polymorphic DNA) (Welsh and McClelland, Nucleic Acids Res. 18:7213-7218, 1990; Williams et al., Nucleic Acids Res. 18:6531-6535, 1990), i.e. not requiring prior knowledge of specific genomic sequences, with that of SINE-morphs (Short Interspersed repetitive Elements) (Kaukinen and Varvio, Nucleic Acids Res. 20:2955-2958, 1992), i.e. the ability to target multiple genomic loci in a single polymerase chain reaction (PCR).

Each PCR contained a total reaction volume of 20 µL; with 10 mM Tris-HCl (pH 8.3), 50 mM KCl, 1.5 mM $MgCl_2$, 0.01% Gelatin, 0.01% Triton-X-100, 125 µM dNTPs, 1.25 µM primer DNA, 1 µCi ^{32}P dCTP (3000 Ci/mmol; 10 mCi/ml), 2.5 U Taq DNA polymerase, 0.1 mM cresol red dye, and 25 ng template DNA. PCR conditions were ordered as follows: first step denaturation at 94C for 90 s; then 35 cycles of *i)* 30 s at 94C (denaturation), *ii)* 45 s at 45C (annealing), *iii)* 90 s at 72C (extension); and a final extension for 5 min. at 72C. Six-percent polyacrylamide gels (acrylamide:N,N'-methylenebisacrylamide, 30:1) were used to separate PCR products under non-denaturing conditions, with 3 M urea, in 1X TBE buffer, cast in 0.04 x 38 x 50 cm dimension, and

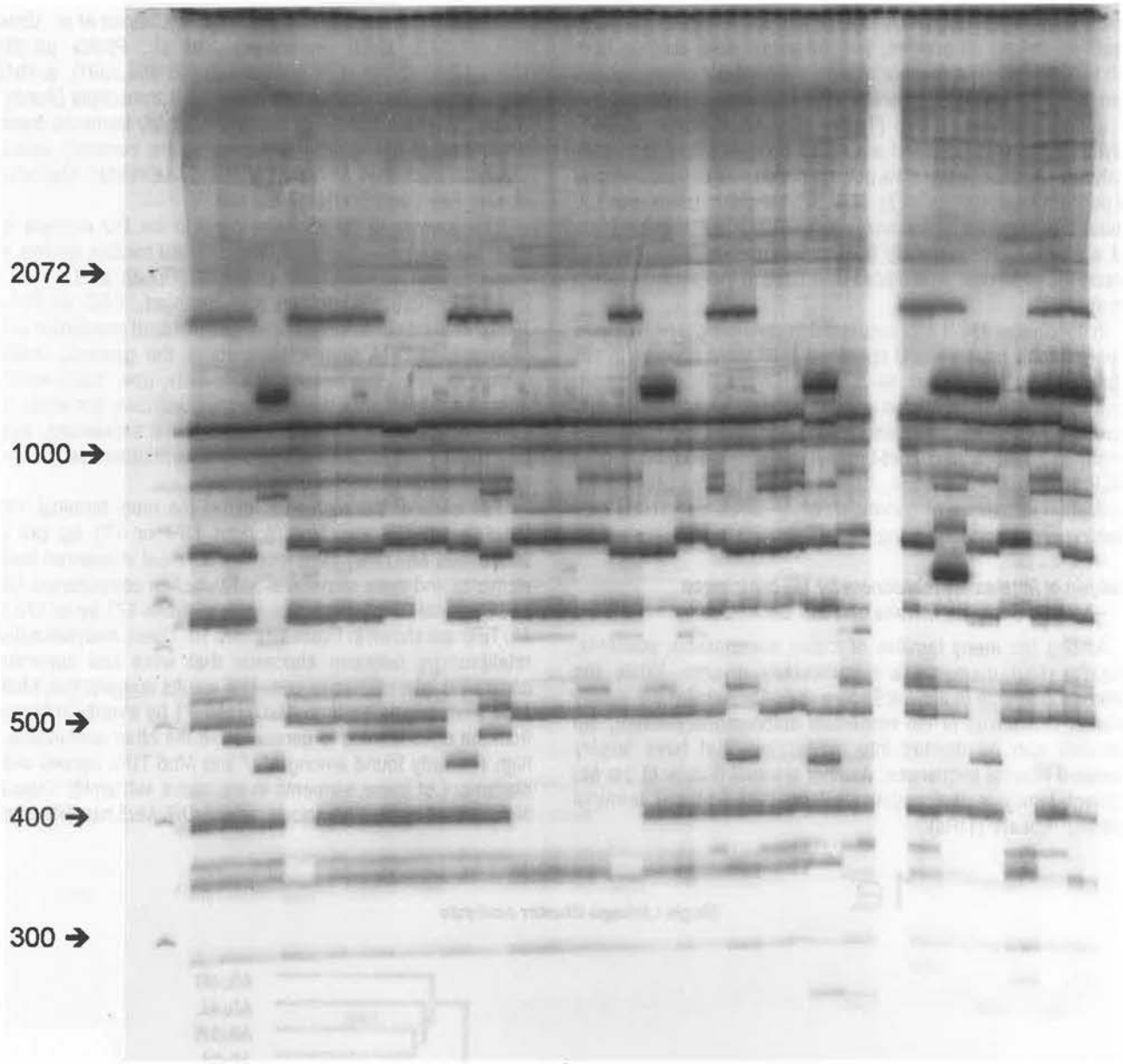


Figure 1. Example of ISSR autoradiograph using primer $(CA)_6$ RY. Lanes represent (left to right) nineteen popcorn and eight dent corn inbred lines. For each inbred, separate DNA samples were run in adjacent lanes. Two blank lanes were included after samples from the second dent corn line. Exposure of X-ray film was for 12 h without an intensifying screen.

electrophoresed at 1000 V for 2 to 3 h. Gels were dried at 80C under vacuum and exposed to X-ray film for 8 to 12 h at room temperature.

Ten primers were designed based on di- $[(CA)_n, (GT)_n]$ and tri- $[(AGC)_n, (GCT)_n]$ nucleotide tandem repeats (Table 1). Primers with the same tandem repeat were differentiated by one or two bases at their 3' end, in order to amplify sequences between tandem repeats and to achieve a degree of specificity for these unique regions. Primers were obtained from both Integrated DNA Technologies Inc. and the Purdue DNA synthesis laboratory.

For each inbred line, two DNA samples derived from separate extractions were used in each PCR run (see Figure 1). Only bands which were consistent across separate DNA extractions were considered as reproducible, and thus scored. Repeated PCR runs involving separate DNA extractions indicated that > 98% of ISSR

Table 1. Summary of the primer structure and polymorphism obtained using Inter-Simple Sequence Repeat (ISSR) amplification.*

Primer Sequence	Total Number Bands	Number Bands Polymorphic	Percentage Polymorphism
$(CA)_6$ **R**	58	57	98
$(CA)_6$ **RY**	42	40	95
$(CA)_6$ **RG**	47	46	98
$(GT)_6$ **YR**	63	59	94
$(GT)_6$ **AY**	10	9	90
$(GCT)_4$ **Y**	47	45	96
$(AGC)_4$ **Y**	63	58	92
$(AGC)_4$ **GR**	37	36	97
$(AGC)_4$ **GY**	46	44	96
$(AGC)_4$ **AY**	42	40	95
Average	45	43	95

*R = purine and Y = pyrimidine in primer sequence notation. Percentage polymorphism indicates at least one difference in band expression across the combined set of pop- and dentcorn inbred lines.

bands were reproducible. A total of 455 scoreable bands were identified among all primers, with fragment sizes ranging from 100 to 3000 bp. The number of bands scored per primer ranged from 10 to 63, giving an average of ~ 45 markers (band positive or null)/primer/inbred line (Table 1). An average of 95% polymorphism was detected among all pop- and dent corn lines analyzed. Approximately 80% polymorphism was detected among 19 popcorn lines, compared to 50% polymorphism using over 100 probes with three restriction enzymes each in RFLP analysis (data not shown). Approximately 95% polymorphism was detected among the eight dent corn inbred lines used in this study (data not shown).

In conclusion, ISSR was successful in generating a high degree of polymorphic markers, and appeared to be more effective in this regard when compared to RFLP analysis using popcorn germplasm. ISSR provided a high number of scoreable and reproducible markers per primer. Also, the ISSR technique was simple to perform and cost-time efficient when compared to RFLP technique. Therefore, ISSR appears to hold promise for application in studies of genetic diversity, DNA marker-assisted breeding and genomic mapping.

Analysis of *Mu* element relatedness by TIR comparison

--Antonio Costa de Oliveira and Jeff Bennetzen

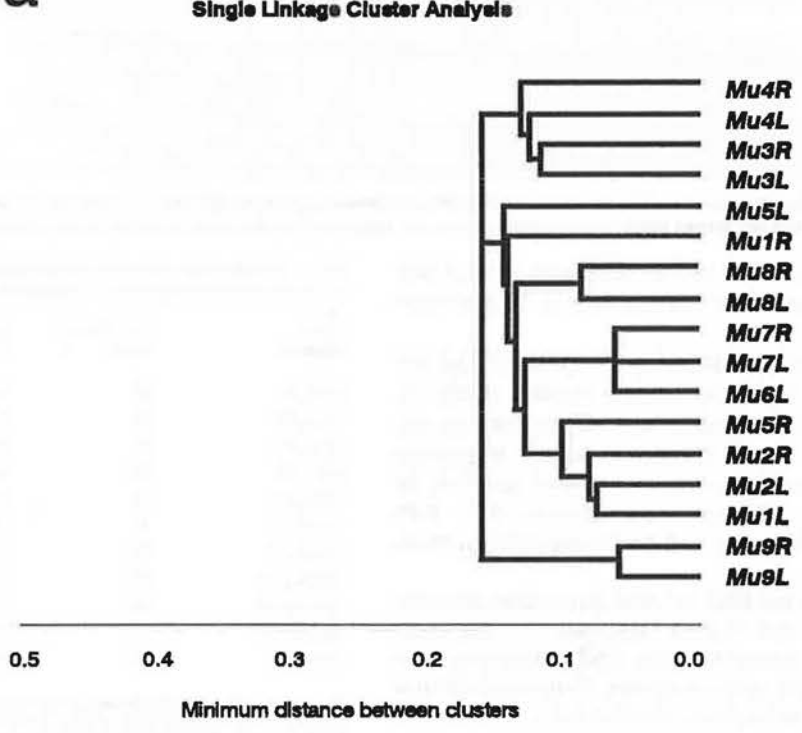
Among the many families of maize transposable elements, *Mutator* (*Mu*) elements are particularly diverse. While the defective elements of the *En*(*Spm*) and *Ac/Ds* families are mostly deletion derivatives of the respective autonomous element, *Mu* elements can be divided into subfamilies that have largely unrelated internal sequences. Another unusual feature of the *Mu* element family is their relatively long (185-514 bp) terminal inverted repeats (TIRs).

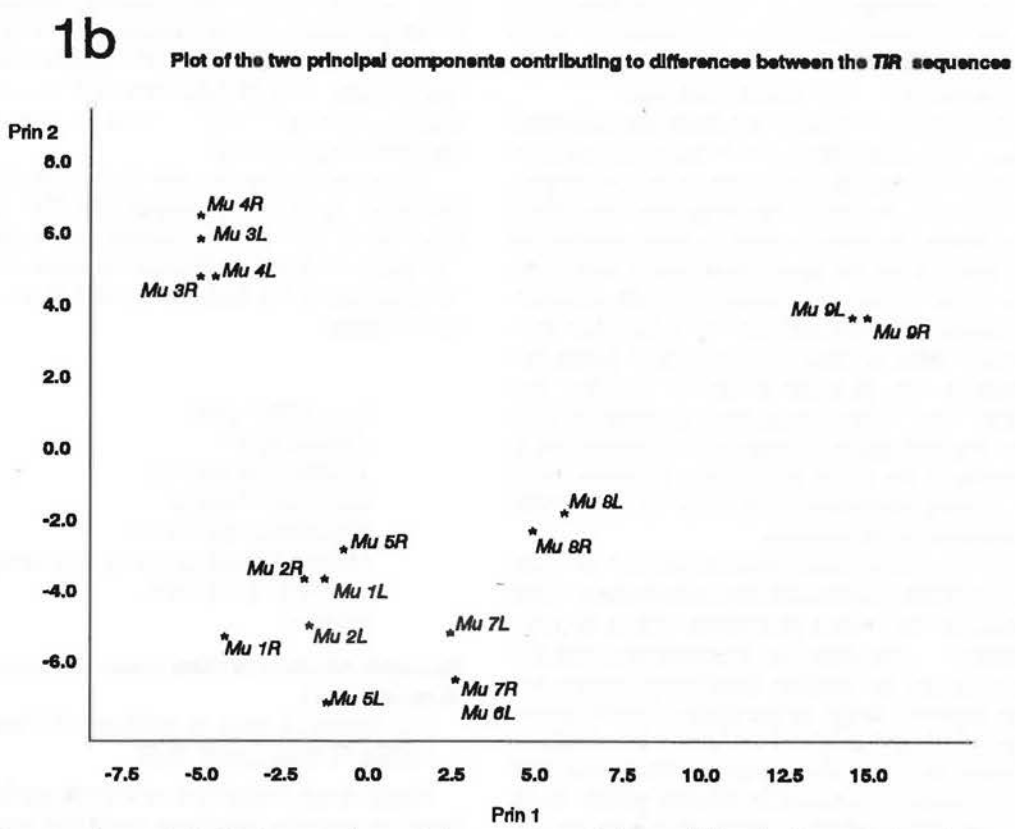
The autonomous *Mu* element, *MuDR* (Chomet et al., Genetics 129:261-270, 1991; Hershberger et al., PNAS 88:10198-10202,1991; Qin et al., Genetics 129:845-854,1991), is 4942 bp long and encodes at least two convergent transcripts (Benito and Walbot, Maydica, 1994). Six subfamilies of *Mu* elements, based on similarities of the internal sequences, are currently accepted: *Mu1/Mu2*, *Mu3*, *Mu4*, *Mu6/Mu7*, *Mu8* and *MuDR/Mu5* (Bennetzen et al., Crit Rev Plant Sci 12:57-95, 1993).

The sources of the diversity found in the *Mu* element family have not been fully identified, and different models on how these elements evolved have been proposed (Talbert et al., J Mol Evol 29:28-39, 1989; Bennetzen and Springer, TAG 87:657-667, 1994). At least some internal changes involved acquisition of large segments of DNA from other parts of the genome, making it difficult to determine exactly what events, how many events, or what order of events have occurred. Because the ends of the elements are more conserved than internal sequences, differing primarily by single base substitutions, a relatedness analysis of this element component seemed feasible.

For each of the elements, either the most-terminal 172 bp (*Mu6* 's left TIR and *Mu3* 's right TIR) or 171 bp (all other sequenced *Mu* TIRs) were found to be most conserved between elements, and these sequences were used for comparisons. Cluster and Principal Components analyses of these 171 bp or 172 bp of *Mu* TIRs are shown in Figures 1a and 1b. These analyses indicated relationships between elements that were not apparent by comparing internal sequences. The results suggest that *Mu3* and *Mu4* were originated from *MuDR* (*Mu9*) by events independent from the ones leading to generation of the other subfamilies. The high similarity found among *Mu7* and *Mu6* TIRs agrees with the placement of these elements in the same subfamily. Despite its high level of internal homology with *MuDR*, *Mu5* has TIRs that are

1a





no more closely related to those of *MuDR* than are those of the other defective *Mu* elements. This raises the possibility that the *Mu1/Mu2* subfamily (for instance) may have been derived relatively recently from *Mu5*, and not directly from *MuDR*. The isolation of *Mu8* from the other elements suggests that this element could have been originated directly from the autonomous *MuDR*, or from a *MuDR*-like element. As previously noted by many investigators, the two ends of a single element are usually most similar, implying that they evolve in a concerted manner possibly involving conversion across a cruciform structure.

The isolation of *MuDR* from the other elements suggests that either the intermediate defective elements are not present in the currently-existing maize populations or that such elements have not yet been discovered. Comparison of the degree of dissimilarity between TIRs of *MuDR* and of its defective relatives to the degree of sequence variation for the silent (i.e., intron and third position) sequences of *adh1* (Gaut and Clegg, PNAS 90:5095-5099, 1993) suggests that *MuDR* diverged from its known defective elements less than ten million years ago. Since homologues of *Mu* elements have been found in species that diverged from maize more than 10^7 years ago, this suggests either horizontal transmission of these elements, concerted evolution of unlinked *Mu* element ends, or the loss/extinction (or a lack of detection to date) of defective *Mu* elements that are less closely related to *MuDR*. However, the application of molecular clock analysis to a transposable element (which may evolve more rapidly, by self-mutagenesis, or less rapidly, via concerted evolution, than standard maize genes) may be relatively inaccurate. The high dissimilarity between the TIRs of *MuDR* and other *Mu* TIRs has been suggested by Benito and Walbot (Maydica, 1994) to be a consequence of selection for promoter activity in the *MuDR* TIRs. Alternatively or additionally, there may have been selection against promoter function in the non-autonomous elements.

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Opaque mutations from *Mutator* self populations

--Wenpeng Yang, P. SanMiguel, P. Stinard, D. Robertson and J. Bennetzen

Over the course of many years of testing *Mutator* populations for mutagenic activity, many opaque mutations have been found to segregate on selfcross ears. We have begun the genetic analysis of fifty such ears, segregating for presumably independent opaque mutations. Mutations in *opaque1* (*o1*), *opaque2* (*o2*) and *opaque6* (*o6*) have been found by allelism tests with standard tester alleles. Candidate *opaque5* (*o5*) and *opaque7* (*o7*) mutations have also been conditionally identified.

Eleven of the fifty mutant lines produced opaque seeds that either did not germination (six lines) or germinated and died shortly thereafter (five lines). In this latter class were two of the *o6* mutations. We observed in agreement with previous reports (Gavazzi et al., Theor. Appl. Gen. 46:339-345, 1975; Ma et al., Cereal Chem. 52:412-419, 1975; Manzocchi et al., Theor. Appl. Gen. 72:778-781, 1986) that the homozygous seedlings dies shortly after the emergence of the first or second leaf. One of the *o6* mutant lines (#3142) was exceptional, however; opaque

seed yielded viable seedlings that matured to produce adult plants. In our summer nursery at Purdue, the field-grown *o6/o6* seedlings from mutant #3142 first produced yellowish leaves that slowly greened from the veins out as the leaf aged.

The three *o6* mutations identified in this study were subjected to further analysis. Opaque seeds from the homozygous-viable *o6* mutant were planted in small pots and grown in the greenhouse. After 27 days of growth, the *o6/o6* seedlings from line #3142 had produced six leaves; the lower three leaves were yellowish but green near the veins, while the upper three leaves were more uniformly yellow. After 53 days, nine leaves had been produced; the upper four leaves were yellowish and the bottom five were withered and dead. After 80 days, all of the *o6/o6* plants had produced thirteen leaves, of which all but the top four had withered and died. Two of these plants were sacrificed for DNA preparation, and the third yielded a reasonable quantity of viable pollen. The viability of the #3142 *o6* mutation suggests that it may be due to a partial inactivation of the locus, or to instability (e.g., somatic reversion) of the mutation.

Two of the *o6* mutations were backcrossed two or more generations to a B73/Mo17 hybrid and were subsequently tested for cosegregation of the mutant phenotype with a *Mutator* transposable element. At the same time, the putative parental and mutant lines were scored for Southern hybridization pattern with three restriction fragment length polymorphism (RFLP) probes linked to *o6*: *bnl17.08*, *bnl19.08* and *bnl19.44*. The Southern hybridization results indicated that mutations #3142 and 5117 were induced on chromatids derived from the Q67 parent. A *Mu* element that cosegregated with the opaque phenotype was not found in the two tested families, #3142 and #5117, using several different restriction enzymes and *Mu1*, *Mu2*, *Mu3*, *Mu4*, *Mu5*, *Mu7*, *Mu8 and MuA (MuDR)* probes. Hence, either these mutations were not induced by *Mu* element insertion into *o6* or the responsible element was not detected in the Southern analysis. This latter possibility could have two origins: either the *Mu* element responsible is of a subfamily that has not yet been identified or the causative *Mu* band was obscured by other bands in the Southern blots analyzed.

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Tlr1 may be allelic to *tb1*

--Patricia S. Springer and J.L. Bennetzen

Crosses were performed to determine if there is an interaction between *Tillered1* (*Tlr1*) and *teosinte branched1* (*tb1*). *Tlr1* is a dominant mutation reported to confer excessive tillering in the heterozygous state (Neuffer et al., MNL 61:50-51, 1987). In the W23/L317 background supplied by the Maize Genetics Coop, this phenotypic effect is not severe. *Tlr1* had previously been mapped to the long arm of Chromosome 1. The recessive mutation *tb1* causes excessive tillering and the conversion of ear shoots into vegetative branches (Burnham, MNL 35:87, 1961). Schnable recently reported (MNL 66:5, 1992), and we have also observed, that *tb1* acts as a semi-dominant allele for increased tiller number in some environments.

Self pollination of a *Tlr1/+* plant resulted in F2 progeny that segregated for the teosinte branched phenotype, including ear shoots converted into vegetative tassels. A cross between *Tlr1/+* and *tb1/tb1* plants resulted in 15 teosinte branched and 25 wild-type progeny. The deviation from a 1:1 ratio may be due to the poor penetrance of the teosinte branched phenotype (our unpublished observations).

These results, together with the fact that *Tlr1* and *tb1* map to the same region of 1L, suggest that *Tlr1* and *tb1* are allelic. Alternatively, *Tlr1* and *tb1* may be semi-dominant mutations in different genes that both condition the same phenotype and, when heterozygous in the same plant, provide an additive effect that mimics allelism.

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Monogenic tolerance to maize streak virus maps to the short arm of chromosome 1

--D. Kyetere, R. Ming, M. McMullen, R. Pratt, J. Brewbaker, T. Musket, K. Pixley, and H. Moon

Maize streak, incited by maize streak geminivirus (MSV), is an important economic disease of maize that occurs in most African countries. Previous genetic studies have reported monogenic (Storey and Howland, Ann. Appl. Biol. 59:429-436, 1967; Soto et al., Ann. Appl. Biol. 100:539-546, 1982) or multigenic inheritance (Kim, Crop Sci. 29:890-894, 1989) of resistance to infection by MSV.

In this study, recombinant inbred lines (RILs) derived from a cross of the tropical inbreds Tzi4 (MSV tolerant) and Hi34 (MSV susceptible) were evaluated for MSV tolerance using controlled leafhopper (*Cicadulina mbila*, Naude) infestation protocols slightly modified from Leuschner (IITA Res. Briefs 1(2):4-6, 1980). Experiments were conducted at two locations: one glasshouse experiment at Namulonge, Uganda, and two field experiments at the CIMMYT Mid-altitude Maize Research Station in Harare, Zimbabwe. A rating scale of 1 (very few streaks or widely spaced spots) to 5 (severe, systemic streaking and chlorosis) based on the protocol described by Soto et al. (Ann. Appl. Biol. 100:539-546, 1982) was used to assess disease severity of RILs, parental sublines, and the F1 planted in a lattice design with two replications.

Seventy-one RFLP markers provided informative polymorphisms in a total of 87 RILs. The association between marker genotype and MSV tolerance was determined using three analytical approaches: single factor analysis of variance (SFAOV, PROC GLM, SAS), and two interval mapping procedures described by Knapp and Bridges (J. Hered. 81:234-235, 1990) and Lander and Botstein (Genetics 121:185-199, 1989). The recombinant inbred lines were considered as an 'F2 intercross' population for MAPMAKER/EXP 3.0 analysis.

Results of analysis of variance indicated phenotypic tolerance scores of the parents, F1 and RILs were consistent across locations. The F1's reaction was intermediate in the Namulonge

experiment and similar to the tolerant parent, TZi4, in the two Harare experiments, suggesting a degree of dominance is associated with MSV tolerance in TZi4.

Results from SFAOV and the two interval mapping approaches showed a significant association of MSV tolerance with four linked RFLP markers, *bnl12.06*, *npi262*, *umc167*, and *umc67*, found on the short arm of chromosome 1. An R^2 of 0.74 was observed for the association between *npi262* and MSV mean tolerance score of four ratings (Table 1). The two interval mapping approaches identified the same chromosome 1 region with individual intervals accounting for as much as 90% of the variation in MSV tolerance (Table 2). Analysis using Knapp and Bridges (J. Hered. 81:234-235, 1990) resulted in significance levels of less than 10^{-17} associated with intervals in the region. Qualitative

Table1. Results from single factor analysis of variance showing loci significantly associated with MSV tolerance (mean tolerance value based on scores based on four rating intervals) on chromosome 1 in three experiments.

	Namulonge		Harare (1)		Harare (2)	
Locus[1]	Prob.	R^2	Prob.	R^2	Prob.	R^2
bnl12.06	0.009	0.11	0.0001	0.32	0.0001	0.34
npi262	0.0001	0.43	0.0001	0.58	0.0001	0.74
umc167	0.0001	0.32	0.0001	0.46	0.0001	0.56
umc67	0.0001	0.23	0.0001	0.32	0.0001	0.37
php20855	0.009	0.09	0.010	0.09	0.002	0.12
bnl8.29	0.004	0.10	N.S.	---	0.020	0.06

[1]Loci listed in relative order on chromosome 1 (Coe, MNL 1993).
Prob. = significant probability level.
N.S. = not significant. R^2 = the fraction of phenotypic variation associated with genotypes at respective loci.

Table 2. Results from MAPMAKER/QTL analysis showing marker intervals on chromosome 1 detecting significant variation, peak LOD and percent variation (% var.) for MSV tolerance.

	Namulonge		Harare	exp 1	Harare	exp 2
Locus	LOD[b]	% var.[c]	LOD	% var.	LOD	% var.
bnl5.62-bnl12.06	N.S.	--	15.57	90.0	20.99	91.9
bnl12.06-npi262	10.93	52.9	22.33	89.0	27.16	83.6
np1 262-umc167	11.44	56.3	18.93	76.6	26.60	83.5
umc167-umc67	7.96	35.9	10.19	43.5	15.70	72.5
umc67-php20855	N.S.	--	8.25	65.0	10.26	65.0

[a] Loci in intervals listed in relative order on chromosome 1 (Coe, MNL 1993).
[b] Maximum LOD between each interval.
[c] Percent of phenotypic variation explained by respective loci (=R^2).
N.S. = not significant.

analysis of data from the final (fourth) rating also was performed. Recombinant inbred lines were classified as tolerant if they were more tolerant, or not significantly less tolerant than, Tzi4; or susceptible if significantly less tolerant than Tzi4. These data fit a chi-square goodness of fit test for a 1:1 Mendelian ratio, further indicating presence of a single gene. Multipoint linkage analysis mapped this gene, designated *msv1*, between *bnl12.06* and *npi262* at a genetic distance of 10 cM from *bnl12.06* and 3 cM from *npi262*.

WOOSTER, OHIO
The College of Wooster
LONDON, ONTARIO
The University of Western Ontario

Light induced accumulation of a polyubiquitin-encoding RNA in plumules of five day seedlings

--Robert A. Bouchard, Priti Krishna and David B. Walden

It has previously been shown that RNA accumulation from specific members of the gene families of the small heat-shock protein (shsp) genes and the *hsp83* genes can also be induced by light in the cotyledons of *Pharbitis nil* (Krishna, Felsheim, Larkin and Das, Plant Physiol. 100: 1772-1779, 1992; Felsheim and Das, Plant Physiol. 100: 1764-1771, 1992). We have now detected light-induced accumulation of RNA detected by a gene-specific probe for the maize polyubiquitin encoding gene *uwo6* (MubC1), already shown to be a heat-shock gene, in plumules of five-day, dark-grown maize seedlings.

Seeds were surface-sterilized and the seedlings were grown in the dark on sterile wet filter paper in foil-wrapped pans in a 25 C incubator for five days. On day five, sampling was done as follows: 0-time control seedling plumules were removed and immediately placed in liquid nitrogen; heat shock seedlings were placed at 41 C in the dark for 1 hour, then plumules were harvested and placed in liquid nitrogen; pans containing time course (30, 60, 105, and 150 minute) seedlings were unwrapped and covered with transparent plastic, then returned to the 25 C incubator for the designated times with the lights on, after which plumules were harvested and placed in liquid nitrogen. Whole-cell RNA was prepared and RNA-dots were probed with a PCR-generated fragment specific for maize polyubiquitin clone MubC1. As previously reported (Liu, Walden, and Atkinson, MNL 67:72, 1993), RNA detected by this probe is up-regulated under heat shock; in the present experiments, accumulation under heat shock was over 300% of the level seen in control plumules. RNA up-regulation to 140% of the level found in the control was detectable after 30 minutes of light-induction, and RNA accumulation reached a level comparable to that seen under heat shock by 150 minutes. In contrast, little or no detectable light-induced accumulation was observed using probes specific for transcripts of two maize small heat-shock genes: *uwo9* (Mhsp18-1) and *uwo10* (Mhsp18-3), ruling out a general stress response. We are currently examining light-induced accumulation of RNAs for the additional maize ubiquitin genes and other heat-shock genes for which we have specific probes in plumule and radicle RNA.

Heat-shock inducibility of RNA from a small heat shock protein encoding gene is independent of its developmental induction

--Robert A. Bouchard and David B. Walden

We have previously shown that RNA accumulation from specific members of the maize gene family comprising small heat-shock protein (shsp) genes is developmentally modulated in a gene-specific manner within tissues containing developing microsporocytes (Bouchard, Frappier, Liu, Raizada, Atkinson, and Walden, Maydica 38:135-144, 1993). Transcripts detected by the probes specific for two such shsp genes, *uwo9* (Mhsp18-1) and *uwo11*(Mhsp18-9), show developmental induction in anthers or spikelets containing late-prophase through meiotic division microsporocytes relative to somatic tissue controls. However, transcripts detected by the probe specific for shsp gene *uwo10* (Mhsp18-3), also derived from a cDNA clone recovered from the heat-shocked somatic tissue cDNA library, show no developmental induction relative to control somatic tissues in anthers or spikelets containing late-prophase through meiotic division microsporocytes, or in tissues harboring microsporocytes at any other stage of development. This observation suggested two possibilities to us. Either the product of shsp gene *uwo10* remains uninduced in microsporogenous tissues because a gene-specific site or inducer for developmental induction is lacking, or this

particular shsp gene is repressed or modified in these tissues so that it can no longer respond to induction when the other shsp gene does.

To differentiate between these possibilities, we compared the levels of *uwo10* RNA seen in control and heat-shocked microsporogenous tissues, as well as in somatic tissues. The tissues used were: isolated staged anthers containing late prophase microsporocytes (late prophase anthers), spikelets containing early and late prophase anthers (prophase spikelets), the immature inner leaves surrounding the source tassels (tassel sheaths), and five day old plumules (plumules). RNA was isolated from control and heat-shocked samples and RNA-dots were probed using a gene-specific subclone of 18-3. The observed elevations in heat-shocked versus control samples were as follows: in late prophase anthers, >570%; in prophase spikelets, 550%; in tassel sheaths, >700%; in five day plumules, >500%. Thus, the *uwo10* gene retains the same capacity for heat shock induction in late prophase anthers and spikelets as in somatic tissues, despite the fact that the microsporogenous tissues show no developmental accumulation of this shsp RNA at stages when the products of other shsp genes show developmental accumulation. These results favor the hypothesis that a gene-specific site or inducer for developmental induction is lacking for gene *uwo10* in microsporogenous tissues.

WUHAN, CHINA
Huazhong Agricultural University

Allozyme polymorphism and relationships to quantitative traits: diversity of 10 local varieties

--Z.X. Zhang, Y.L. Zheng, J.S. Li and J.L. Liu

The North Carolina II mating design was used to evaluate the genetic variation of 10 local varieties (Lanhuazao, Wuxiumi13, Wuxiyumi14, Roulihuang, Xiaomeihuang, Zhuxiyumi, Yinxinhuang, Jingzihuang, Qiyunhuong and Hongtianhua) collected from the Three Gorges area, and four exotic synthetics (BSSSC9, Lancaster, Suwon2, and Meihuang9). The results showed there was abundant additive genetic variability within local varieties. The estimation of combining ability revealed that the value of GCA effects on studied traits was significantly different among the fourteen materials. Lanhuazao, Xiaomeihuang, Zhuxiyumi, Wuxiyumi14 and Roulihuang had positive GCA effects in yield and its component factors. BSSSC9 and Meihuang9 also showed higher GCA effects in most of the quantitative traits, and were exotic elite germplasm. Ten local varieties and four exotic populations were assayed for allozyme variations at 16 enzyme marker loci. Extensive allozyme variability was observed in all materials studied. No locus was monomorphic over all materials. Each of the loci *adh1*, *amy1*, *cox2*, *cox3*, *e1*, *e8*, *got1*, *got2*, *mdh2*, *px4*, *sod1*, and *sod4* expressed two allozymes. Both *acp1* and *mdh1* had three allozymes present, and *acp4* had four allozymes present. Within local varieties and exotic populations, 1.17 and 1.79 alleles per locus were present, respectively. *Got2-6* was present in the local varieties but was not discovered in exotic populations; *E8+4.5*, *Mdh2+5.5* and *Sod1+B* were observed in four exotic populations but were not observed in local varieties. The proportion of polymorphic loci of local varieties ranged from 50% to 68.75%, the mean value was 63.13%, and the average expected heterozygosity was 0.1919. MRD (Modified Rogers' Distance) based on 16 loci revealed the genetic differences among local varieties (0.148 - 0.391) and between local varieties and exotic populations (0.110 - 0.444). Based on combining ability, heterotic patterns and allozyme variation of the ten local varieties, a suggestion about how to form the basic population which could be used for population improvement has been brought forward. The correlation coefficient between MRD based on per se isozyme loci and quantitative traits of hybrids showed that a chromosome fragment near *acp4* and *adh1* on 1L is associated with yield per plant directly or indirectly. But there were no relationships between MRD based on 16 allozyme loci and SCA, and between SGD (Specific Genetic Distance) and SCA of yield and other quantitative traits. The results indicated allozyme marker loci were of limited usefulness in predicting heterosis in maize.

III. USING MAIZE IN K-12 CLASSROOMS

This is our second year for the Education Section in our newsletter. Last year we had five submissions. We have eight submissions this year--more than a 50% increase! What is very exciting is that ideas in last year's submissions helped to stimulate some of the current submissions. Thanks to everyone who submitted their ideas and activities!

Your ideas are needed for future editions. If you have visited classrooms, worked with teachers, or in any other way used maize in the classroom, your colleagues would love to hear about it. Also, if you make use of ideas submitted in the newsletter, we would love to hear about it. Vicki Chandler will act as the coordinator to assemble the Education Section of the newsletter. Your ideas or use reports can be submitted via email, FAX, diskette, or old-fashioned mail. It would be best to get the information by the end of December, beginning of January.

Vicki Chandler is also attempting to keep an updated mailing list of all persons interested in maize in K-12 education. If you did not receive a mailing Fall 1994, and would like to be added to the list, please send Vicki Chandler your complete address, telephone, FAX, and email.

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Maize for kindergarten students

--Caron Burnette, Kindergarten Teacher, Suwanee Elementary School, Gwinnett County, Georgia

Each year prior to Thanksgiving we study the Native Americans who lived in the United States. In our study we learn about various tribes, where they were located, and their culture. The main groups of Native Americans we learn about are: Northwestern, Southwestern, Plains, and Eastern Woodland. We learn how the land, climate, and resources influenced the culture in which the Native Americans lived. During our study we discover that maize is an important food for some of the Native American people. In order for my students to gain a better understanding of what maize is and the work that was done to grind it for meal, I provide them with ears of dried maize and an authentic stone grinder and grinding stone in one of my centers. The children push the kernels of maize off the cob into a basket, put a handful of the kernels on the stone grinder, and then try to grind the kernels with the grinding stone. This kinesthetic activity enables the children to experience what maize is and how maize was prepared by some of the Native American people.

Also, at the Science Table, there are decorative gourds, squash, Indian corn and hybrid corn for the children to explore and compare. After the children have had ample opportunity to explore these specimens, we discuss the observations they had made about the various types of maize, squash, and gourds. Comparative charts are made, and their observations are recorded by the teacher on chart paper.

Maize genetics labs for high school/college classes

--Dick Kowles, Biology Department, St. Mary's College of Minnesota, Winona, MN 55987-1399

I have used maize in a couple of ways in my teaching at St. Mary's College, and I would like to share this with others. I teach the introductory genetics course to biology majors which are comprised of mostly sophomores. I have used maize materials in two of my laboratory activities. These activities may be appropriate for an upper level high school genetics course as well.

In the initial lab of the course, I use sugary vs. normal kernels as a 3:1 demonstration and a review of goodness of fit tests. Then I assign exercises for their analysis and lab reports that include the following:

(1) Ears depicting a 9:3:3:1 ratio with sugary vs. normal and colored vs. colorless. Students are asked to test the data for independent assortment and to subdivide Chi square if appropriate.

(2) Seedlings that are the result of F2 selfs of plants heterozygous for two different virescent mutations (usually v4 and v17) are available in flats. Expectations are a 9:7 ratio. Students are asked to calculate the ratio, use goodness of fit tests, and deduce a feasible mode of inheritance and parental genotypes. It is a neat test in which to determine whether students can discern between a 1:1 ratio and a 9:7 ratio.

(3) Students also plant kernels in pots that test a backcross involving *sugary1*, *lazy*, and *striate1*. I obtain the seed by making backcrosses of *Su/su La/la Sr/sr* x *su/su la/la sr/sr* during the summer months. Using these ears, students remove all the kernels from two or more rows on the ear and plant the sugary and normal kernels in a separate series of pots. After they germinate into small seedlings, they place the pots on their sides in a horizontal position at the edge of a greenhouse bench. In a short time, normal and lazy seedlings can be discerned. Seedlings can now be scored for all three traits. This exercise is then a three-point test for the students to analyze, with *sugary1* and *lazy* being linked and *striate1* being independent of the other two traits.

In another exercise, maize microsporocytes are used to study the cytological aspects of meiosis. Students make their own preparations with propionic carmine staining procedures. All meiotic stages are identified as a class effort. A video system attached to a research-grade microscope is used to display and discuss the good preparations. Heterozygous translocations are sometimes inserted into the students' material to check their observation abilities. Their discovery of rings leads to pairing discussions.

Mitochondrial mysteries

--Christine Chase, Horticulture Sciences Department, University of Florida; and
--Susan Gabay-Laughnan, Department of Plant Biology, University of Illinois

This one-hour demonstration has been given by the Chase lab for numerous groups of high school and junior high school students. Only small pieces of equipment are required, and the demonstration has also been taken to local classrooms. The demonstration begins with a 20-minute slide show and discussion. The following points are made:

1. Many important genetic discoveries relevant to all organisms were first made studying plants (Mendel and McClintock, for example). Plants are wonderful organisms for genetic investigations!

2. The plant cell contains, among other organelles, a nucleus, mitochondria and chloroplasts. The functions of these organelles are briefly discussed.

3. While much of the genetic information (DNA) resides in the nucleus and is inherited according to Mendel's laws, some genetic information resides in the mitochondria and chloroplasts. This information is usually inherited from the maternal parent.

4. The expression of genetic information in the nucleus, mitochondria, and chloroplasts must be coordinated somehow.

5. Cytoplasmic male sterility (CMS) systems are good tools to study coordination among different genomes. The mitochondria encode this maternally inherited failure to produce functional pollen, but nuclear restorer genes can suppress or overcome the mitochondrial genotype, resulting in a male-fertile plant.

6. An experiment that we used to identify regions of the mitochondrial genome involved in CMS is described. The experiment involves the following concepts:

a. CMS plants sometimes undergo a mitochondrial mutation to male fertility. We call these mutants fertile revertants. We can use this genetic change to help us identify parts of the mitochondrial DNA that may be responsible for the CMS trait. We do this by comparing the mitochondrial DNA from CMS plants and the fertile revertants.

b. Mitochondria can be purified from other cell components by differential centrifugation. DNA can then be released and purified from these mitochondria.

c. Restriction endonucleases and agarose gel electrophoresis can be used to compare the mitochondrial DNAs from CMS plants and fertile revertants. After these techniques are explained, photos of the restriction fragment patterns are passed around to see who can identify fragment differences. Fragments present in the CMS plants but not in the fertile revertants are candidates for further study.

The remainder of the hour is spent in the lab, demonstrating the various components of the experiment. We do the following:

1. Grind some etiolated seedlings in mitochondrial extraction buffer in a blender, explaining that this breaks open cells to release all of the component parts.

2. Large components (nuclei, plastids, starch grains) are removed by a low speed centrifugation (1,000 x g for 10 min). The supernatant is centrifuged at 10,000 x g for 10 min to pellet smaller components. The resulting pellet is a crude preparation of mitochondria. These spins can be accomplished in a variable speed microfuge.

3. While spins are in progress, we demonstrate agarose gel electrophoresis. We usually have three gels prepared. One (still in the casting tray with slot former) is passed around so that students can see how the gel is formed and what it feels like. A second is set up in an electrophoresis unit with buffer. Student volunteers can load pre-prepared samples of bromphenol blue and xylene cyanol in 20% glycerol. The two dyes are quickly separated during a 100 volt run, so students can observe the separation of molecules according to size. The third gel has been run, stained with ethidium bromide, and set up under a plexiglass shield on a transilluminator. We usually run molecular weight standards. These provide a striking pattern that is easy for the students to observe when the illuminator is turned on.

Susan has generously provided seeds of CMS-S maize and a cytoplasmic revertant to fertility for this work. In addition, she suggests the use of the RU male-fertile cytoplasm. These three cytoplasms are distinguished by different mitochondrial episome components. The CMS-S line carries the S1 and S2 episomes, the revertant lacks episomes and the RU line carries episomes of different molecular weight. The episomes are present in high copy number and can be readily visualized on ethidium bromide-stained gels of undigested, crude mitochondrial DNA preparations. This provides a simple and striking demonstration of cytoplasmic genetic differences (and separation of DNA molecules by size). We have plans to increase seeds of these materials and to make some reciprocal crosses, which could be used to demonstrate maternal inheritance of the mitochondrial plasmid DNAs. We'd be happy to provide seeds and further assistance to anyone interested in demonstrating mitochondrial mysteries.

Balanced lethal, epistasis, linkage

--Charles R. Burnham

In MNL 67:101, 1993, I reported the *y1 + l10/y1 w15 +* balanced lethal. The crossover stock *Y1 + +/y1 w15 l10* segregates 3 green:1 albino with an occasional luteus seedling. The linkage is so close that at least 75 seeds must be grown to obtain one luteus seedling (*l10* is at 6L-19, *w15* at 6L-13). A better combination should be *w15* at 6L-13 with *l15* at 6L-30. The white seeds from the cross of *Y1 +/y1 w15 x Y1 +/y1 l15* will be the balanced lethal combination. To establish the crossover, cross those plants with a *y1 y1* stock. Most of the selfed ears will segregate either luteus or white seedlings. They can be offered to those teaching elementary genetics. The frequency of luteus seedlings from selfs of the *Y1 + +/y1 w15 l15* crossover for this cross should be high enough to make a good demonstration.

The *la1* -11- *su1* -15- *gl4* stock is good for a 3-point backcross linkage demonstration. Plant the *Su* and *su* seeds separately. When the plants are 6-8 inches high, turn the containers on their sides. Only the normal, *La* plants will bend up.

Incorporating corn in K-12 education

--W.F. Tracy, Madison, WI

For the second year I taught a one week summer course on the biology, history, and sociology of corn (MNL 68:116). Eighteen elementary and middle school teachers took the course. The design of the course is for me to provide basic information on corn and its place in our society and the teachers develop activities and a "web." The web is a concept used by elementary educators in which a subject such as corn is put at the center of the web and all

curricular areas, science, history, music, language, etc. are related to the topic. Corn is obviously ideal for this concept.

This year we will be planting corn gardens at local elementary schools, to teach the life cycle and very basic genetic principles. The plan is for fourth graders to plant the corn in the spring and make predictions. When they return in the fall they will harvest the corn and test their hypothesis. For this to be successful we will need teachers that are very dedicated, and around most of the summer.

I am preparing a manual for the course with scientific, social, and economic information. The manual will also discuss classroom corn culture and activities and experiments. If any of you have activities or other things that would be useful for such a manual I would be happy to hear from you. If they are included in the manual I would certainly credit the source. Along with the manual I hope to develop very simple "kits" consisting of segregating ears and seeds that will segregate for seedling traits.

School children: lost in the maize

--Jeena Tharp and Torbert Rocheford, Departments of Education/Agronomy, University of Illinois, Champaign-Urbana

The following is written by Jeena Tharp who was raised on a farm in Illinois, is a senior majoring in education, and is an undergraduate worker in the Rocheford laboratory. Jeena is also working with her father to produce a video on growing corn on a farm. They have completed portions covering harvesting and moving the grain to storage, and expect to complete the planting and growing portion this summer. The video will be available for distribution in late 1995.

As a student in elementary education, I volunteered to go with Dr. Rocheford to his son's second-grade classroom to give a brief introduction to maize and to determine the background knowledge of the students. It was stunning to hear that in such a large farming area very few of the students knew much about this important crop.

We started the lesson by asking questions that gave us an idea of their vocabulary and pre-existing knowledge. I then read a few pages from The Story of Corn by Peter Limburg, and later gave them pictures to color. Next we showed and discussed the parts of a mature plant and gave each of them an ear of corn which they shucked and silked. This led to a discussion of the properties of maize, such as the various types (popcorn, sweet corn, field corn), colors, sizes, how it grows, and its many uses. In closing the lesson we gave each student a dish of soil with his/her name on it and seeds to plant and watch grow.

Overall, I feel that the lesson went well. The students were very interested and excited. The students really liked husking the corn and being able to take an ear of corn home. However, there are a few points to remember when visiting a classroom of any age group.

- Take concrete things for the students to touch and observe.
- Be aware of the timing when giving students handouts and manipulatives. You can lose their attention very quickly.
- When doing demonstrations, be sure to do a few that the students are capable of and allowed to do.
- Have the cups of soil already prepared and have a sunny, warm place that is easily accessible to the students.
- Include personal stories.
- Select students to be helpers.

Other ideas and extensions:

- Take a field trip to a farm where maize is grown.
- Take a field trip to a factory or plant that uses maize.
- Make or watch a video of a process that maize might go through.
- Do a lesson on different food products made from maize. Grind corn into cornmeal and make different types of food.
- Do a unit on different uses of corn. Have the students make charts and bulletin boards.
- Tie maize to Thanksgiving and do a lesson on the history of maize.

I encourage everyone to get involved in the schools. Most teachers and students like to have the people of the community and professionals visit the classrooms to share their knowledge about special topics. Students of all ages enjoy this and will usually give more of their attention to the guest than to the teacher because it is something different. The teachers are very helpful in preparing the class for the guest. They can also be helpful in giving the guest suggestions for activities to go along with the topic. I really think that students should be given the opportunity to learn more about their environment and possibly their options for the future.

Activities useful for kindergarten and first-grade classes

--Alan L. Kriz, Discovery Research, DEKALB Genetics Corp., Mystic, Connecticut

I've gone into my older daughter's classrooms the past two years, each time with a different slant toward corn as an interesting, fun, and important organism.

Illustration of Mendel's First Law:

Although the concept of segregation of alleles might seem too complex for kindergartners, the kids remember aspects of the experiment a year later. What I tried to stress here was variation in maize and how to do an experiment. I first explained that genes control a lot of things about us and all living things, and there are different forms of genes that basically do the same thing but with some different effects. I illustrated this fact by referring to eye color, and explained that my daughter Becky and I both have blue eyes, but that Becky's sister has brown eyes like her mother. I explained that each of us had two forms of any gene, one from our mother and one from our father. I then told them that corn has different forms of genes as well, and that one such gene controls plant color (I used an albino seedling trait for this): the "green gene" (wild-type allele) makes the plant green, and the "white gene" makes the plant white. After explaining that the white gene is a mutant (yes, kind of like the turtles) and that two white genes are necessary to make the plant white, I went on to tell them that the different forms of the genes get shuffled up and some corn seeds get two green genes, some one green and one white gene, and others two white genes. I illustrated this by placing green and white beads into small test tubes, and had them tell me which would give a green plant and which could give a white plant -- most students picked up on this right away. I then had each student come up and, with their eyes closed, pick two beads out of a bag containing an equal number of green and white beads and place them into a tube in a tube rack. We then separated the tubes into green-green, green-white, and white-white classes, grouped the green-green and green-white together since these would all give green plants, and counted the individuals. We recorded this

observation on the board (16 green:4 white), then made a prediction of what would happen if we planted 20 kernels from an ear containing "shuffled up" green and white genes, and wrote this on the board as a prediction. We proceeded to plant out the twenty kernels; they promised to water the flat, and I told them I would be back at the end of the next week to help count the plants. When we counted up the green and white plants, we observed 13 green and 5 white plants, and I think I convinced them that 13 is pretty close to 16 and that 5 is pretty close to 4, without getting into ratios. The kids had a grand time at this, and continued to talk about the experiment at home -- favorable comments were received from several parents about this demonstration.

Corn as a Commodity:

I went into Becky's first-grade class this past fall to talk about the importance of corn and its uses. From raiding the pantry at home I was able to come up with a couple of shopping bags of items that contain corn, and showed them that corn can be used for everything from trash bags to Life-Savers. I also took in some corn-starch-based packing peanuts as well as some styrofoam ones, and explained that corn is a renewable resource and that we could make all of the styrofoam peanuts we want as long as we could grow enough corn. I also showed them how the starch peanuts dissolved in water, while the styrofoam ones did not, and asked which would be better for the environment. Kids this age are very green-minded and they really appreciated this demonstration. Finally, I went to the large U.S. map in the classroom and showed them where the majority of the corn is grown in this country (they got a kick out of the term "Corn Belt"), and told them that the amount of corn grown this past year was equivalent to 35 Connecticuts. Each student then came up to get his or her souvenir -- an ear of colored corn in a pollinating bag.

I found the kids very receptive to both presentations; they asked lots of questions (particularly during the latter), and in conversations I've had with Becky's friends I can see that they remember some of the key concepts regarding corn utilization. Some of them only refer to me as "Dr. Corn".

Corn for third graders

--Pat Byrne, USDA-ARS, Columbia, MO

The hardest part about talking to my son's third grade class was deciding which of the many possible topics on maize to cover in a one-hour time-slot. I finally settled on these three areas:

Origin and diversity of maize. The poster "Indian Corn of the Americas" was a perfect visual aid for this, as it includes photos of teosinte, pod corn, an array of colorful landraces, and modern varieties. A big hit was the gray mass of "huitlacoche" (corn smut), which elicited incredulous groans when I explained that it's eaten as a delicacy in Mexico.

Biology and reproduction. Many of the kids had heard terms like tassel, silks, and husks, but couldn't match the names with plant parts very well. Ideas on how the reproductive process works were also pretty wild. So I went through the pollination /fertilization/seed development cycle. To illustrate plant defense mechanisms, we had a husking contest with tight- vs. loose-husked ears.

Corn utilization. The day before I talked, the teacher assigned as homework that each student should look around the house, read ingredients labels, and list as many things as they could that contain some form of corn. The class of 20 came up with 85 different items, including some that were new to me: marshmallows, cherry licorice, puppy chow, goldfish food, rice krispies, and sonic boom (whatever that is). We had a vote on their favorite way to eat corn; fresh, sweet corn narrowly edged out popcorn, with breakfast cereal, cornbread, and canned corn receiving one or two votes each.

For a finale, each kid got a couple of colorful ears, which they sketched the next day as an art project.

One of the main factors in the success of this venture was a teacher who had trained her class to be good listeners, and was willing to do pre- and post-activities to involve the kids more and make my talk more than an isolated event.

Corn display for museum day

--Carol Rivin, Dept. of Botany and Plant Pathology, Oregon State University, Corvallis, OR

The biological sciences departments at OSU sponsor regular "Museum Days" when they set up museum-type displays and activities that are attended by children from the local schools. Last year, I was asked to do one on maize. I used some of the ideas from Sue Wessler's newsletter contribution, and I got some wonderful materials from Bill Tracy. I tried to emphasize two things: 1) the role of indigenous people in the history of maize agriculture, and 2) the huge variety of foods and products that use components of the maize plant.

My display was not going to be staffed most of the time, so I designed it to have only a small amount of text and a self-explanatory activity. The physical format was a walk-thru with three parts: I had an entrance wall with some posters showing the origin of maize, and maize agriculture and the general uses and products from corn plants. This was followed by a display case area showing some of the varieties of maize and origins of Corn Belt maize. Many of the materials in this section were very graciously provided to me by Bill Tracy. The centerpiece of the display was a large table heaped with household items of all kinds and a sign asking each student to try to identify those items that contained maize products. At either end of the table I put a box of pencils and check-off sheets that listed each item in the display. The items ranged from the obvious (tortillas) to the obscure (a corn plastic pen). I tried to include a lot of things they would be very familiar with like breakfast cereals and soft drinks. The kids were encouraged to look at labels and see what things listed corn syrup, dextrose, cornstarch, etc. On an exit table, I put a small packet of corn seeds for each child to take home. Inside the packet I put instructions for planting and a note telling them that all the items on the display table contained corn products.

It was a successful display and mostly easy to mothball for future use or to bring into individual classrooms. I did have a little problem with theft of small, tempting items from my display table (small toys and candy), so when I repeated it I just used empty boxes or wrappers stuffed with cotton.

Growth curve with maize seedlings

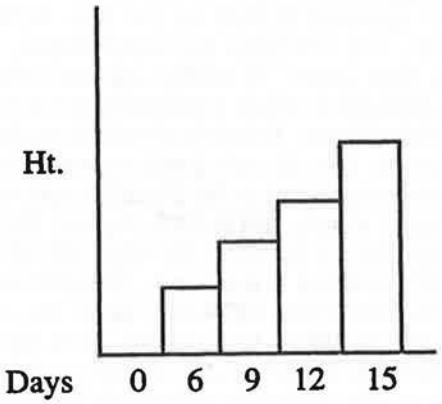
--Mary Berlyn, Yale University

Here's a pre-arithmetic growth curve for really young ones. It worked great with my own kids as preschoolers and it would be good for the kindergarten, first-grade set. It requires only corn kernels of any well-growing stock, construction paper and

scissors, and yields a quite handsome growth curve. It does not require fine motor skills or ability to read subdivisions on a ruler or draw. Corn is good because it grows straight and tall. Individuals (or, less preferably, groups of 2 or 3) could each produce a graph.

- Plant the kernel.
- On a large piece of construction paper, draw a y axis for Ht., and an x axis for Days.
- From construction paper of a different color, cut long strips of width on the order of an inch or a couple of cm.
- When the seedling emerges and at regular intervals, the child holds the long strip, one end touching the soil, next to the axis of the plant and cuts the strip right at the top of the shoot.
- The strip is pasted to the graph, labelling the Day below it.
- Three days (or other chosen interval) later, a second strip is used to measure and is pasted next to the first, and Day is recorded. And so on.

Result:



Even very young children can do every bit of the measuring and recording themselves. Not rocket science and not genetics, but a fun-to-make and instructive bar graph.

NEWSLETTER OF THE UNITED STATES GERMPLASM ENHANCEMENT-MAIZE PROJECT

Volume 1 Issue 1, August 1994

Interest high in U.S. G.E.M. project

Interest was very high at the meetings held in December (1993) and February (1994) presenting the U.S. Germplasm Enhancement -Maize project proposal, **U.S. G.E.M.** (public-private cooperative project to enhance the **LAMP** germplasm) to the public and private sectors of the plant breeding community. 50+ attended the meeting held at the ASTA meeting Dec. 9, in Chicago, and at the NCR-167 meeting, Feb. 15, Bettendorf, IA, another 25 or so were added to the list of those interested in the project. Bolstered by this interest, the ASTA has taken the proposal to Capitol Hill and to the Agriculture Subcommittees of both the House and Senate. Other legislators and key aides have also been given the proposal and they expressed interest and support. The proposal asks for funding of a USDA-ARS enhancement project under the direction of Dr. Linda Pollak, USDA-ARS, Ames IA, to enhance identified temperate accessions, and for the establishment of an entirely new program in the south or south-east to adapt identified tropical and sub-tropical accessions to day-length sensitivity for future adaptation and enhancement in the southern and central cornbelt. Evaluation for value-added traits will also be an important part of the project.

A Technical Advisory Committee, **U.S. G.E.M. TAC**, has been formed, as directed by the ASTA, and work has progressed on working out a protocol, cooperator agreements and release policies for the program. These documents were sent this month to the cooperator companies and U.S. Public Corn Breeders. By the first **TAC** meeting, June 1 in Kansas City, MO, at least 29 private companies had pledged "in-kind" support, i.e., space in summer and winter nurseries and yield test plots, for the project. 12,300 summer and winter nursery rows and 7500 yield test plots have been pledged so far. This is enough support to begin enhancement of the temperate accessions identified by the USDA-ARS/Pioneer Hi-Bred Latin American Maize Project, (**LAMP**). Initial crosses will be made this winter and backcrosses next summer for what should become the single most comprehensive germplasm enhancement effort ever in maize.

U.S. G.E.M. project addresses short and long term needs of corn industry

The protocol developed by the Technical Advisory Committee of **U.S. G.E.M.** project (Dr. Wilfredo Salhuana, **Pioneer Hi-Bred**, Chair; Dr. Linda Pollak, **USDA-ARS**, Coordinator; Dr. Kevin Montgomery, **Golden Harvest**; Dr. Jim Parks, **Wyffels Hybrids**; Dr. David Harper, **Holden's**; Dr. Blaine Johnson, **University of Nebraska**; Dr. Randy Holley, **Northrup King**; and Dr. Doug Tiffany, **Pioneer Hi-Bred**) is a product of years of experience in working with exotic materials and attempting to bring them into commercial utilization.

The 8 plant breeders on the committee have developed a protocol that includes short, medium and long range breeding plans to incorporate temperate and tropical accessions into the corn breeding programs of at least 29 companies and University maize breeding and research programs. Crosses of selected accessions will be made with proprietary germplasm and in many cases 3-way or back crosses will also be made in order to adapt the germplasm to the central corn belt. These breeding populations will be released to the public and private breeding community. Private companies will continue proprietary efforts in the crosses made with their own lines. Per se and test cross yield testing will identify winning S2's. Each company will release to the North Central Region Plant Introduction Station (**NCRPIS**) a synthetic of the best S2's identified in their yield tests. As public funding becomes available to work on the tropical accessions and tropical hybrids and lines, the project should provide the corn breeding industry with new and unique germplasm to meet the needs of farmers and end users far into the next century.

Volume 1 Issue 2, December 1994

Congress approves $500,000 for U.S. G.E.M.

Congress approved $500,00 for the **U.S. G.E.M** project in September. The Committee Conference Report, published on Sept. 20, 1994, stated: "In addition, the conference agreement provides $500,000 to initiate a national program to enhance the corn germplasm base. Scientists are concerned that the current narrow genetic base for corn greatly increases vulnerability to unforeseen pest problems for the $16 billion corn crop."According to Dr. Charles Murphy, **USDA-ARS**, National Program Leader, the appropriation is a line item in the budget and will be divided up after a 10% overhead is subtracted. $300,000 will go into the CRIS of Dr. Linda Pollak, **USDA-ARS**, Ames, Iowa and $150,000 to Marty Carson, **USDA-ARS,** Raleigh, North Carolina. About 35% of the $300,000 will be used to support public cooperators. This appropriation represents a major step in procuring the funding needed for the **U.S. G.E.M.** project as proposed by the **ASTA** appointed committee. The original proposal called for $950,000 to fully fund the project on a yearly basis. Many supporters sent letters to Senators and/or Representatives in support of the **U.S. G.E.M.** proposal earlier this year. In particular, **Senators Dale Bumpers** and **Thomas Harkins**, and Representatives **Richard Durbin** and **Neal Smith** were instrumental in getting the $500,000 in the 1995 budget, and deserve a "Thank You."

U.S. G.E.M. Day well attended

U. S. G.E.M. held its first Field day on Sept. 22, 1994 at the Iowa State University Agronomy and Agricultural Engineering Research Farm near Ames, IA. The Field Day was publicized in the Des Moines Register, which also sent a reporter to cover the activities at the field day. About 65 people attended, including Rodney Williams, **Iowa Corn Growers**, Bob Mustell, **National Corn Growers Association**, and Don Gingerich, **special assistant to Congressman Jim Lightfoot**. Drs. Dwayne Buxton, Research Leader of the **USDA-ARS** Field Crops Research Unit and Ron Cantrell, Head, **ISU Dept. of Agronomy**, welcomed the guests. All cooperators present were introduced and short talks were given by some of the ISU cooperators who are using **LAMP** germplasm. Larry Johnson, working on wet milling traits, Pam White, studying starch functionality and Craig Abel, developing insect resistance, all gave short presentations of their work. Several commercial corn breeders commented on how good the Corn Belt by exotic

crosses looked in terms of standability, general plant health and ear size.

TAC expands

The third meeting of the Technical Advisory Committee was on Sept. 22 after the field day. The agreements to utilize proprietary lines in breeding crosses were reviewed. Additional value-added trait studies were identified and the protocol was further revised. Members of the **TAC** present at the **ASA** meetings in Seattle met on Nov. 15 and were informed that the number of companies willing to sign the agreement now total 21 and number of public cooperators interested in the project totaled 19. The **TAC** met again in Chicago during the **ASTA** convention on Dec. 6. The impact of this partial funding of the project was discussed. It was decided that **TAC** membership would be for a 3 year period before rolling over membership to other interested parties. The committee also agreed to add representation from the value-added trait sector, as well as adding a representative with an entomology background. The committee felt these individuals would enable the project to address a broader range of germplasm issues.

Data begin to come in

Lab data from some of the initial screenings done by cooperators have begun to come in. **United AgriSeeds** has performed NIR calibrations to interpret composition analyses of protein and oil made on 1000 S1 lines of 73 temperate **LAMP** accessions. **Hoegemeyer Hybrids** evaluated the S1 lines for resistance to Corn Lethal Necrosis, CLN, finding 8 non-USA accessions that had 25% or more S1 lines with commercial levels of tolerance.

Progress to date

Initial crosses are being made this winter by 21 commercial companies utilizing their proprietary inbreds in making breeding crosses to the top 5% of the temperate **LAMP** accessions. A second cross will be made next summer such that each 3-way will contain 75% elite Corn Belt material, not more than 50% from one company, and 25% **LAMP** accession. These crosses, both the 50% and 75%, will be topcrossed in the winter of 1995 and tested in the summer of 1996. The winners will go on to be selfed in 1996/97.

RELEASE POLICY

The **TAC** felt that some sort of release agreement needed to be in place between the **U.S. G.E.M.** Project and the interested public and private institutions. This agreement would concern the release of breeding material, breeding lines and finished products. Since "finished products" were a long way off, and still somewhat hard to define, release policies for breeding material and finished lines were targeted. The rationale for establishing these policies was twofold. First, it was felt that the **U.S. G.E.M.** project ought to maintain control over the materials in the early stages of development to insure that the protocols are followed and that the quality and integrity of the material is maintained through release to **NCRPIS**. Second, it was felt that the public and private institutions who have offered to participate in the project by offering "in-kind-support", defined as "contributing in the breeding effort", ought to be designated as "cooperators" and thereby have immediate access to the breeding crosses and data. Public and private institutions who did not offer "in-kind-support" but wanted access to breeding crosses and data would not be denied access, but would have to get the material after release to **NCRPIS.**

Private "cooperators" have a vested interest in the breeding crosses because of the use of their proprietary lines and because of the in-kind-support they have offered. All S2's developed will be considered proprietary material of the company doing the selfing and will not be released. A synthetic of recombined S2's will, however, be released to the **NCRPIS** in year 5, following yield testing of the S2 test crosses.

Public "cooperators" have a vested interest in the breeding crosses because of the in-kind-support they have offered. Public S3 lines will be released to **NCRPIS** in year 5, following yield testing of the S2 test crosses. In order to maintain quality of the material, it was felt that the only restriction on a public cooperator, was to not redistribute it without the coordinator's permission.

Another reason for the release policies was that the **U.S. G.E.M.** project ought to receive appropriate recognition for the development of the breeding material and coordination of the project. This recognition is important for both the future funding of the project and the career advancement of the scientists involved. The recognition would be via the joint official release statement of the S2 synthetics and S3 lines to the **NCRPIS** by the **U.S. G.E.M.** project and the cooperator involved as published in Crop Science.

In summary then, signing an agreement and becoming a "Private Cooperator" means you agree to contribute to the breeding effort by utilizing your resources in crossing proprietary material to **LAMP** accessions, participate in cooperative yield testing of the testcrosses, releasing the breeding crosses, relevant data and an S2 synthetic to the coordinator. Becoming a "Public Cooperator" means that you agree to contribute to the breeding effort by utilizing some of your resources in the breeding effort following the creation of breeding crosses, releasing S3 lines to the coordinator and not redistributing material without the coordinator's permission. Cooperators may also be involved in screening germplasm and developing lines for disease and insect resistance and value added traits. Not signing the agreement means that you have free access to all material released to the **NCRPIS.**

Please send your comments and suggestions regarding this newsletter to: Dr. G. Douglas Tiffany, Pioneer Hi-Bred, Int., RR 8, Box 113A, Mankato, MN 56001; Phone: (507)625-3252, FAX: (507)625-6446; E-Mail: TIFFANYD@PHIBRED.COM, or COMPUSERVE: 72303,2077.

FOR MORE INFORMATION ON THE U.S. G.E.M. PROJECT, CONTACT: DR. LINDA POLLAK, USDA/ARS, DEPT. OF AGRONOMY, IOWA STATE UNIVERSITY, AMES, IA 50011 PHONE: 515/294-7831, FAX: 515/294-9359

V. NATIONAL CORN INITIATIVE

"The future of the corn industry is written in the genetic code"

Executive Summary

The Critical Technologies Partnership, the National Corn Growers Association, private sector agribusinesses, and trade associations request that $20 million be appropriated annually for five years, continuing with $10 million annually for an additional five years, to identify the genes and genome of corn that impact specific needs and potentials of the agricultural market.

Economic Impact

Corn is an economic powerhouse for the U.S. -- the production, processing, transport and sale of corn is a critical source of jobs and economic strength for the entire nation -- reaching far beyond the Corn Belt. Though produced on only 24% of our cultivated land, corn generates $20 billion in farm value and over $4 billion in exports annually.

U.S. corn usage is approximately 62% for livestock feed for meat (beef, pork, poultry, and cultured fish) and milk, 3% for human food, 15% for industrial raw material, and 20% for export grain.

In addition to being an important food source for humans and livestock, corn is the raw material for over 3,500 value-added products, ranging from sweeteners and paper finishing to fuel ethanol and pharmaceuticals.

New Value-Added Products

We must enhance our knowledge about the structure, organization, and functions of the corn genes to ensure and expand our ability to grow, process, and export this vital natural resource and its products. This is a proposal for a concerted effort to derive a resource of genetic information that will allow us to overcome yield ceilings and quality limitations and open new product development opportunities.

Technology Leadership

A timely effort will maintain the role of corn in the U.S. economy and will ensure U.S. leadership in world agriculture, including availability of important biotechnology, informatics, patents, and materials necessary for maintaining our international competitive position.

The Scientific Coordinating Committee of the Plant Genome Program has encouraged initiation of a corn genome project. Many individual federal, state and private sector scientists, and leaders in industries involved in seed production, biotechnology, crop management, feeding, processing, and export of corn and corn products stress the desirability of addressing a single commodity like corn, which is genetically representative of all cereal species, to leverage research dollars. This will increase the cost effectiveness of locating genes, defining their functions, and enhancing production efficiency, quality, and value of all the cereal crops.

The National Corn Initiative

Corn, a crop native to America, is an economic powerhouse for the U.S. The most important crop species in the U.S. economy, corn generates $20 billion a year in farm value. Last year, U.S. farmers planted over 73 million acres of corn -- 23.5% of all U.S. crops. The production, feeding, processing, transport and sale of corn is a critical source of jobs and economic strength for the entire nation -- reaching far beyond the Corn Belt. Besides being an important food source for humans and livestock, 15% of corn grain is the raw material for over 3,500 important products -- ranging from fuel ethanol to pharmaceuticals. Each time corn is processed and used as an ingredient or raw material in consumer products, corn increases in overall economic value -- supporting and generating new jobs and increased income for consumer product manufacturers, transportation services, distributors, retailers, and their financial institutions and suppliers. Studies suggest that if the U.S. exported just 10% more agricultural products as processed products rather than in raw form, we could create 100,000 new jobs, increase personal income by more than $2 billion, and increase gross output by an additional $5 billion.

The goal of the National Corn Initiative is to improve profitability and sustainability in the corn industry. Using genomic technology, the genetic blueprint and function of the estimated 40,000 genes of corn will be determined. This is an applied, directed research program conducted by geneticists and molecular biologists in universities and private sector research laboratories. With this information, solutions to longstanding constraints in the corn industry (agronomic performance, damage by second generation European corn borer and other stalk borers, corn rootworm, and grain quality at harvest and in storage) will be possible.

Impact on the Corn Industry and the Nation

The National Corn Initiative has the potential to deliver many important benefits to the corn industry and the nation's economy and security.

Protect U.S. interests and access to important biotechnology and gene patents and foster cooperation with the scientific communities of other nations

Significantly reduce crop losses and reliance on pesticides through improved biological methods to control and alleviate serious industrial threats and targeted pests

Improve yields and reduce crop losses caused by adverse environmental conditions such as heat, drought, and salt

Contribute to a 20% increase in production efficiency within 10 years, for a conservative $4 billion in added farm value per year. Applying the tools and knowledge gained from the National Corn Initiative, the development rate of new hybrids and the overall productivity of the farming process can be increased -- satisfying a need and aggressively exploiting an opportunity to increase production to meet U.S. and expanding world demand. To produce the 1994 corn crop with 1950 technology would require over 210,985,000 acres. Acquisition of this additional acreage would include marginal land that would have to be cultivated at enormous cost to the environment. This would also make corn and value-added corn products, especially meat, more expensive for consumers.

Year	National Corn Acreage	National Average Yields (bu/acre)	Total National Production (bu)
1950	83,302,000	37.6	3,131,009,000
1994	2,917,000	138.6	10,103,030,000

Develop new ways to overcome problems with grain storage and transport

Develop tailored hybrids with valuable specialty starches, oils and protein content

Provide a stepping stone to similar advances with other cereal crops

Infrastructure and Administration

Based on a five-year track record, the Plant Genome Office of the Agricultural Research Service will manage the National Corn Initiative. The Plant Genome Office will be responsible for ensuring that basic research is strongly linked to the needs of breeders and end-users.

The coordinating committee will be the primary forum for individuals and institutions of diverse backgrounds and interests to define and prioritize agribusiness needs and wants. Based on the results of this collaboration, focused competitive programs will be published for private companies, universities, joint ventures, and ARS laboratories to submit research proposals for funding consideration. By using programs to manage groups of projects that complement and reinforce each other, the National Corn Initiative can have the greatest possible impact on technology and the economy.

Strategies

Organize and sustain a program of mission-linked, market-driven, vertically integrated maize genome research from the seed producer through the farm gate to desired outcomes in the corn industry.

Strengthen and provide continuity for the existing infrastructure of trained and experienced people, laboratories, facilities, equipment, and support services required for isolating, sequencing, and mapping the genes of corn and maintaining stocks of genes and probes for distribution to the research community.

Assess, or generate, and document variations in traits through a collaboration of public and private laboratories and breeders, seeking variants for essentially every gene where a recognizable function or visible effect can be defined.

Develop a Maize Database containing information on genetics, uses, economics, breeding, biochemistry, and biotechnology as a readily accessible, professionally managed, electronic repository for information on the genetics of corn, accessible on the information superhighway.

Develop intellectual property policies as specific projects require.

The National Corn Initiative will harness creativity and take advantage of the many resources already in place. In biotechnology, the USDA-Agricultural Research Service (both the corn genome program and commodity research projects), cooperating universities, private industry, and plant biotechnology laboratories have created the institutional capacity, and well-trained personnel, to isolate, identify, and map corn genes. The existing Maize Genome Database is an efficient, readily-used informatics structure accessible through the information superhighway. This is coupled with expert plant breeding and testing networks, including cooperation with the vigorously responsive seed industry, by which genetic knowledge can be translated into adapted hybrids with desired characteristics.

New Products

New information must be derived on the genetics of starch and other grain properties for:

new specialty starches

enhanced starch extractability for improved wet milling efficiency and ethanol production

increased starch content for greater productivity and energy source for livestock

enhanced grain quality and reduction of mold and mycotoxins for improved storage

designed protein content, quality, and digestibility for feeding livestock -- value added meat

absorbable phosphorous for improved livestock intake and waste management.

Current efforts leveraging corn's role in industry include characterizations of diverse strains from the Latin American Maize Project (LAMP), and an infusion of select germplasm from LAMP into elite U.S. strains in the Germplasm Enhancement of Maize (GEM) project. These projects are drawing forth remarkable ranges of diversity for yield potential, grain quality, protein content and qualities, starch content and qualities, and potentials for resistance to pests and stresses, all in need of efficient methods for genetic characterizations and analyses.

An example of a problem that would greatly benefit from understanding the genetic makeup of corn is aflatoxin, a fungus generated carcinogen. Aflatoxin is one of the most serious

problems in stored grain, causing millions of dollars in losses annually. Genetic knowledge, plant breeding, biotechnology, and engineering together can begin to design the eradication of this costly problem.

Waxy corn is an example of an application that was derived from a particular genetic quality of the species. It was studied purely as a genetic quality of the grain until the property of gelling starch was realized. This "specialized" corn, grown on 500,000 acres, is now a $135 million contributor to foods and industrial products ranging from pudding to adhesives.

"Supersweet" corn was recognized by serendipity just 40 years ago during basic genetic studies. This specialized corn accounts for a $108 million market annually from Florida, alone.

Challenges Facing U.S. Agriculture

Clearly, we must vigorously protect and expand our ability to grow, process, and export this vital national resource and increase our ability to compete in global markets for corn and corn products. Currently, the U.S. enjoys many advantages in corn production and exports because of our productive soils, favorable climate, excellent transportation system, and sophisticated agricultural technology. However, our historical advantage is eroding as competing nations rapidly improve their technology and management skills. We now dominate 60% of the corn export market, but we only grow 35.3% of the World's corn. Many other nations have the capacity to overtake the U.S. in this important market in coming years -- not only in the production of unprocessed corn, but also in the manufacture and sale of value-added corn-based products -- creating a challenge to U.S. economic strength and innovative leadership.

The Environment

To compete in the global corn market, the U.S. must continually strive to efficiently and economically improve corn production capabilities -- to maximize yield and combat serious threats from disease, pests, and climate changes -- without harming the environment and without creating an unmanageable surplus. Modern biotechnology holds the key to achieving this goal. Recent technological developments in plant gene sequencing and mapping have opened the door for a dramatic acceleration in agricultural production control -- not just for corn but for other crop species as well.

Current Breeding Procedures

For thousands of years, agriculturalists have used cross breeding to produce healthier, more desirable, higher yielding crops, and to fight plant disease, pests, and adverse climatic conditions. A most noteworthy fact is that there is a steady increase in corn yields (3.8% per year based on 1960 yields; 2 bushels per acre per year), attributable primarily to hybrid corn breeding. To date, technology development has been focused on plant breeding and the application of proven methodology to the breeding process. These techniques applied to corn continue to advance production efficiency and improve the response of hybrids to specific environmental occurrences. However, these methods are time consuming and labor intensive, requiring large scale selection and testing work in the seed industry to derive the desired morphology, physiological characteristics, resistances, and other properties.

By combining today's analytical techniques for characterization of grain and fodder with enhanced genetic knowledge and tools, corn breeders will be able to change the pace at which hybrids with desired traits and improvements are developed and commercialized -- from 10-20 years currently to 5-6 years (a conservative estimate) -- thereby giving U.S. farmers a competitive advantage in world markets.

PROGRAM AND FUNDING

Organization and Planning

Director and Steering Committee establish guidance of the program
(primarily by teleconferencing, Internet, and fax)
(aided by two administrative aids)
Evaluate and define priorities, strategies, targets, timelines, and distributions of tasks

Initiate Coordinating Committees as working groups for applications

Time required: 1 year
Estimated cost: $100,000 per year

Tools and Knowledge Development

Isolate, sequence, define, and map 40,000 genes

Implement infrastructure for preservation and distribution of cloned DNA

Maintain and enhance information dissemination and bridge to information on other crops

Relate the functions and the map positions to genes in the other cereals

Time required: 5 years
Estimated cost: $10 million per year

Functions and Applications Development

Define the full range of traits to be analyzed and mapped

Relate functions of the genes to biochemical and developmental pathways

Relate the traits to the traits of other cereals, including mutual applications

Determine and develop the program according to production and market needs

Time required: 10 years
Estimated cost: $10 million per year concurrent with the 5 year period described above

May 11, 1995 Letter Requesting Input From Maize Cooperators

Dear Maize Cooperator:

As you are most probably aware, a National Corn Initiative is under development and is being sponsored by the Critical Technologies Partnership. This letter seeks your help and input in creating an organization that can articulate and prioritize specific research programs with the genes and genome of corn that will support industry's efforts to produce innovative and cost effective corn production and corn derivative products.

The National Corn Initiative is a collaborative effort between industry, academia, research institutions, and government to leverage limited resources in support of advanced research that will have commercial impact on the U.S. economy. The Critical Technologies Partnership, a regional program whose mission is to identify technology strengths and transform these strengths into programs that will promote economic growth and prosperity, sponsored the collaboration and facilitated the initial planning process. It became clear very early on in our discussions that this program had implications beyond the midwest, so we began to seek the input and participation from others in the agricultural community. The core group championing the National Corn Initiative includes: Monsanto Company, Mycogen Corporation, Pioneer Hi-Bred International, National Corn Growers Association, University of Illinois - Urbana-Champaign, University of Missouri - Columbia, and the U.S. Department of Agriculture/Agricultural Research Service. We also receive very valuable input from the Anheuser Busch Companies, Ralston Purina, American Seed Trade Association, and other organizations, as well as from numerous individual scientists in both the public and private sector.

We envision an organization that will include input and participation from a broad cross section of the agricultural community. The Coordinating Committee, composed of representatives from industry, trade associations, and research institutions, will oversee, review, evaluate, and initiate goal setting, benchmarks, and priorities for the Initiative.

NCI DIRECTOR
--> Coordinating Committee Representatives
Industry(4) Trade Associations (2) Scientists (4)

-->Special Topic Workshops, Industry/Scientists to define and advise on research needs impacting production; feed uses/livestock; processing; specific end use applications:

Elements of research to include (but not limited to):

- tissue-specific expression
- agronomic trait analysis
- plant development and properties
- grain development and properties
- gene expression

To ensure that the organization is a dynamic group representing the diverse interests of basic and applied research we are proposing that one chair in each of the designated segments (industry, trade associations, scientists) of the Coordinating Committee be rotated every two years to include a new member.

The Special Topic Workshops will identify and advise the Coordinating Committee on those areas of research that are significant to both our understanding of the corn genome and which have the potential for increasing the competitiveness of U.S. farmers and associated downstream industries.

To achieve our goal of maintaining our competitiveness in global agricultural markets, it is essential that people, such as yourself, volunteer to participate on the Coordinating Committee and/ or the Special Topics Workshops. If you are unable to be an active member at this time, we would appreciate your suggestions and recommendations of colleagues that you feel best represent your interests and those of your organization or institution.

To keep everyone up to date on the development of the National Corn Initiative we have set up a home page on the Internet. It is located at the World Wide Web address: http://www.agron.missouri.edu/NCI.html

Thank you for your time and support of the National Corn Initiative. Please mail your thoughts and suggestions to me at 121 South Meramec, St. Louis, Missouri 63105, or fax 314-889-7666, or e-mail lauriep@aix.cait.wustl.edu , or call me at 314-889-3433.

Sincerely,

Laurie Peterfreund
Technology Development Coordinator, Critical Technologies Partnership

WORKSHOPS ON GOAL-SETTING, PRIORITIES, AND BENCHMARKS
(SOME SUGGESTED CONSIDERATIONS)

4-5 scientists/specialists for each area?
What will be done first?
How soon?
Then what?
Expected outcome?

1. Tissue-specific expression of genes and other sequences:
   a. Libraries from specific tissues
   b. ESTs vs. complete sequences vs. genomics
   c. Repetitive sequences/genomic distribution/species specificity
   d. Priorities on types of sequences
   e. Computer homology searches
   f. Informatics - connectivity, communication, accessibility

2. Mapping and probe handling:
   a. Mapping probed sequences
   b. Banking and distribution of probes
   c. Integration of morphological/physiological mutations
   d. Use of specialized stocks (monosomic/addition lines, hypoploids)
   e. Segment libraries, large genomic inserts (YACs, BACs)
   f. Comparative genomics to other cereals
   g. Informatics - data resource, analysis, representation

3. Agronomic trait genetic analysis and breeding methodologies:
   a. Basis of selection effectiveness in populations
   b. Transfer of exotic germplasm
   c. PCR (etc.)-based selection/mapping
   d. QTL mapping
   e. Identification of candidate genes
   f. Isolation of QTL sequences
   g. Informatics - data and means for creative exploitation

4. Grain development and properties
   a. Value added - modified constituent qualities
   b. Value added - modified contents
   c. Innovations in contents - new products
   d. Efficiency, structure, resistances
   e. Storage, shipping, and processing quality
   f. Informatics - data and means for creative exploration

5. Plant development and properties
   a. Value added - modified constituent qualities
   b. Value added - modified contents
   c. Innovations in contents - new products
   d. Efficiency, structure, resistances
   e. Informatics - data and means for creative exploration

6. Correlating genes and phenotypes:
   a. Gene machine to identify mutant phenotype/sequence associations, and review public vs. private role
   b. Testing functionality of sequences via transgenics/complementation
   c. Biochemical pathway genetic dissection
   d. Informatics - data and means for creative exploration

7. Gene expression processes:
   a. Transcription factors, motifs, etc.
   b. Tissue, stage, and condition-specific regulators/promoters
   c. Role of modification/methylation
   d. Informatics - biochemistry, enzymology, metabolism, regulation

8. Bioinformatics:
   a. Map data, analysis, representation
   b. Sequence and mapping data connectivity, communication, accessibility
   c. Resource inventories and images
   d. Trait information - data and means for creative exploration and exploitation
   e. Biochemistry, enzymology, metabolism, regulation
   f. Germplasm characterization

9. Socio-economic analyses [management level]
   a. Product priorities
   b. Sustainability
   c. Public acceptance
   d. Risk analysis
   e. Property ownership
   f. International impact
   g. Economic impact

VI. ADDRESS LIST

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This year's Catalog of Stocks of the Maize Genetics Cooperation • Stock Center has been revised extensively following a complete inventory of our collection. Some gene combinations have been discontinued either because of poor vigor, problems in pedigrees, difficulty in scoring the traits in combination (e. g. *brachytic1 anther-ear1*), or because the genes in question are so far apart on the chromosome (>50 centimorgans) that they are virtually unlinked. These stocks will sent to the National Seed Storage Laboratory in Fort Collins within the next couple of years and will still be available upon special request. Check with us for availability. All single mutant genes and alleles will continue to be maintained. Nothing that is unique will be discarded, although some mutant stocks may have been removed from this year's list due to poor supply and will be relisted after they have been increased.

Several categories of stocks have been discontinued, and others have been added. The exotics and the popcorns have been discontinued because they represent germplasm lines (as opposed to genetic stocks) that are better maintained by the North Central Regional Plant Introduction Station in Ames, Iowa. The Stock Center's collection of these lines has been sent to Ames, and will be supplied by them (contact Mark Millard, Maize Curator, USDA-ARS-NCRPIS, Agronomy Building, Iowa State University, Ames IA, 50011, 515-292-6502 [voice], 515-292-6690 [fax], nc7mm@sol.ars-grin.gov [internet]).

The Black Mexican Sweet Corn lines with and without B chromosomes will continue to be listed, maintained and supplied by us as B chromosome lines. A new category of stock called "Toolkits" has been created to include stocks that do not represent a particular linkage group, but which are useful in genetic research. Examples include the lines of Chuck Armstrong that produce good type II embryogenic callus cultures and Bryan Kindiger's *ig1* stocks that are useful in producing inbred lines in different cytoplasmic backgrounds.

3,089 seed samples were supplied in response to 314 requests during 1994. Of these, a total of 63 requests were received from 23 foreign countries. 331 more samples were distributed this year than last year, making a new annual record. Approximately half of our requests were received by e-mail.

While the summer weather was dryer than optimal, supplemental irrigation helped us achieve a fantastic harvest. About 5 acres of nursery were grown. Good increases were obtained of numerous stocks that were in low supply and of stocks from the collections of Marcus Rhoades and Donald Robertson. A thorough inventory was made of the stock drawers, and mutants previously thought to be lost (e. g. *gl13*, *v13*, *oro2*, *frz1*, *l3*, and *v7*) were recovered. A systematic increase was made of old translocation stocks, and new *wx1*-linked translocations from several sources were added to the Stock Center collection. Special plantings were made of several categories of stocks, including massive plantings of hundreds of unplaced mutants. Some tests for allelism were made within groups, and pedigree errors were corrected. We had a very successful winter nursery at the USDA facility in Puerto Rico last year and have recently sent seeds there again for this year's winter nursery.

Greenhouse sandbench plantings were made to determine or confirm genotypes relative to seedling traits. Field plantings were also grown to develop pedigree information with regard to mature plant traits. Such information is used to determine or verify genetic constitutions of sources used to perpetuate stocks and supply seed requests. We are hoping that funds for new greenhouse space become available in the near future, so that mutant stocks that do not do well under field conditions can be grown and seedling tests can be done more efficiently.

We have obtained stocks from the collection of Barbara McClintock and are in the process of obtaining stocks from the collections of Charles Burnham and Walton Galinat. We recently received a collection of *wx1-*, *y1-*, *o2-*, or *su1*-linked translocations from William Findley. We also recently received a collection of transposed *Ac* stocks, that will be useful in transposon tagging, from Hugo Dooner (see his report elsewhere in this issue of MNL). A large collection of cytoplasmic stocks in different inbred backgrounds was received from Margaret Smith of Cornell. We are entering data on the newly acquired stocks of McClintock, Rhoades, and Robertson into the computer, and expect to have this information available electronically in the near future. Because of the size of these collections, it is not possible for us to list the individual stocks in their entirety in the Maize Newsletter, although we will supply information on our holdings on request.

Accepting stock requests via e-mail (our internet address is maize@uiuc.edu) has been very successful (approximately half of our requests were received that way in 1994). We recently set up a WWW server that now allows us to receive requests over the 'Web' from users with software such as NCSA Mosaic or Netscape (our home page URL is http://www.uiuc.edu/ph/www/maize). We are continuing to enter data into our internal database. In addition to information about our stocks, we also have reprint collections of M.M. Rhoades, G.F. Sprague and E.G. Anderson. Information about these reprints is accessible from our growing database.

We have been continuing our collaboration with Ed Coe's efforts in building a Maize Genome database. This is part of the Plant Genome Database (PGD) program centered at the National Agricultural Library. Information about our stocks is presently in MaizeDB (and therefore also with the PGD at NAL and also with GRIN) allowing users access to information about available maize genetic stocks. 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.*

Our hopes are that 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. This has now been accomplished in 'WWW' format.

Marty Sachs	Philip Stinard	Janet Day	Earl Patterson
Director	Curator	Research Specialist	Co-director

CHROMOSOME 1

101A sr1 zb4 P1-ww
101B sr1 P1-wr
101C sr1 P1-ww
101D sr1 P1-rr
101F sr1 ts2 P1-rr
103D vp5
103E zb4 ms17 P1-ww
105A zb4 P1-ww
105C zb4 P1-ww br1
105E ms17 P1-wr
105F ms17 P1-ww
106A zb4 P1-ww bm2
106B ts2 P1-rr
106C ts2 P1-ww bm2
107A P1-cr
107B P1-rr
107D P1-cw
107E P1-mm
107F P1-vv::Ac
107G P1-or
107H P1-ww
109B P1-rr an1 gs1 bm2
109D P1-rr ad1 bm2
109E P1-wr br1 f1
110A P1-wr an1 Kn1 bm2
110D P1-wr an1 bm2
110E P1-wr ad1 bm2
110F P1-wr br1 Vg1
110G P1-wr br1 f1 gs1 bm2
110H P1-wr br1 f1 bm2
110K P1-wr br1
111G P1-wr rs2
112E as1
112H P1-ww br1
112K an1 gs1 bm2
113C br1 f1
113E br1 f1 Kn1
113K hm1; hm2
113L Hm1; hm2
114C br1 bm2
114D Vg1
114F br2 hm1
114G br2; hm1 hm2
115C v22-8983
115D bz2-m::Ds; A1 A2 C1 Pr1 R1
116A bz2-m::Ds; A1 A2 Ac C1 Pr1 R1
116C an1 bm2
116D an1-bz2-6923; A1 A2 Bz1 C1 C2 Pr1 R1
116G an1
116I bz2 gs1 Ts6 bm2; A1 A2 Bz1 C1 C2 R1
117A br2
117D tb1
117DA tb1-8963
117E Kn1
118B Kn1 bm2
118C lw1
118I Ts6 bm2
119A Adh1+1S; Adh2-1P
119B vp8
119C gs1
119D gs1 bm2
119E Ts6
119F bm2
119H Adh1-FkF(gamma)25; Adh2+N
120A id1
120B nec2
120C ms9
120D ms12
120E v22-055-4 bm2
120F Mpl1-Sisco
120G Mpl1-Freeling
121A ms14
121B mi1-8043
121C D8
121D lls1
121E ty*-8446
121G ct2
121H bz2-m::Ds lls1; A1 A2 Bz1 C1 C2 R1
122A TB-1La (1L.20; B)
122B TB-1Sb (1S.05; B)
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
124L w*-6474
125A Les2-N845A
125B Mpl1-Jenkins
126A bz2 gs1 bm2; A1 A2 Bz1 C1 C2 R1
126F o13
126G P1-vv::Ac bz2-m::Ds; A1 A2 Bz1 C1 C2 R1 TB-1Sb (1S.05; B)
127A bz2 zb7-N101 bm2
127B dek1-N792
127C dek2-N1315A
127D dek22-N1113A
127E f1
127G Tlr1-N1590
127I gt1
128A ij2-N8
128B l16-N515
128C l17-N544
128D pg15-N340B
128E pg16-N219
128F v25-N17
129A w18
129B wlu5-N266A
129C zb7-N101
130A o10-N1356

CHROMOSOME 2

201F ws3 lg1 gl2 b1
203B al1
203D al1 lg1
203G y3 gl2
205A al1 lg1 gl2 b1
205B lg1
205C lg1 gl2
205F al1 lg1 gl2 B1
205G al1 gl2 B1
206A lg1 gl2 B1
208B lg1 gl2 B1 sk1
208C lg1 gl2 B1 sk1 v4
208D lg1 gl2 B1 v4
208E lg1 gl2 b1
208H gl2
209E lg1 gl2 b1 sk1
211A lg1 gl2 b1 fl1
211H gl2 wt1
212B lg1 gl2 b1 fl1 v4
212D lg1 gl2 b1 v4
213B lg1 gl2 wt1
213F lg1 B1-V Ch1
213H lg1 gl2 B1-V
214C d5
214D B1 gl11
214E B1 ts1
214J B1 sk1
214L lg1 gl2 mn1
215A gl14
215B gl11
215C wt1
215E fl1
215G fl1 v4
215H wt1 gl14
216A fl1 v4 Ch1
216D fl1 w3
216E fl1 v4 w3
216G fl1 v4 w3 Ch1
217A ts1
217B v4
217G v4 Ch1
217H ba2 v4
218A w3
218C w3 Ch1
218D Ht1
218E ba2
218G B1+Peru; A1 A2 C1 C2 r1-r
218H w3-8686
218I w3-86GN12
219A B1+Peru; A1 A2 C1 C2 r1-g
219B b1; A1 A2 C1 C2 r1-g
219C Ch1
219G B1+Bolivia; A1 A2 C1 C2 r1-g
220A Les1-N843
220B ws3 lg1 gl2; Alien Addition T2-Tripsacum
220F os1
221B B1 gs2
221C Ch1 wlv1
221G wlv1
222A TB-1Sb-2L4464 (B; 1S.05; 1S.53; 2L.28)
222B TB-3La-2S6270 (B; 3L.10; 3L.60; 2S.46)
223A Trisomic 2
224A w*-4670
224B v*-5537
224H whp1; A1 A2 C1 c2 R1
224I ws3-7752
224J ijmos*-7335
224K glnec*-8495
224L ws3-8949
224M ws3-8991
225A TB-3La-2L7285
225B TB-1Sb-2Lc (1S.05; 2L.33)
227A dek3-N1289
227B dek4-N1024A
227C dek16-N1414
227D dek23-N1428
227E Les4-N1375
227I nec4-N516B
228A l18-N1940
228B spt1-N464
228C v26-N453A
228E B1-Bh
229A Ch1 rf3
229B v24-N424
229C w3 Ch1 rf3

CHROMOSOME 3

301A cr1
302A d1-6016
302B d1 rt1
302E d1-tall
303F g2
303G g2 d1
304A d1 ys3
304F d1 Lg3-O ys3
304G Lg3-O Rg1
304I d1 h1
305A d1 Lg3-O
305B d1 Lg3-O gl6
305D d1 Rg1
305J d1 Lg3-O h1
305K d1 cl1; Clm1-4
306B d1 gl6
306D d1 Rg1 ts4
307A d1 pm1
307C pm1
308B d1 ts4
308E ra2
309D ra2 Rg1 lg2
310C ra2 lg2
310D Cg1
310E ra2 ys3
310G ra2 y10
311A cl1
311B cl1; Clm1-2
311C cl1; Clm1-3
311E rt1
311F ys3
311G Lg3-O ys3
312D Lg3-O
313A gl6
313D ms3
313E Lg3-O gl6
314A gl6 lg2 A1; A2 C1 C2 R1
314C gl6 lg2 a1-m et1; A2 C1 C2 Dt1 R1
314E Lg3-O pm1
314F Rg1 gl6 lg2
314G gl6 lg2
315B Rg1 gl6
315C Rg1
315D A1-b(P415)
315H gl6 a1-m; A2 C1 C2 dt1 R1
316A ts4
316H gl6 lg2 a1 et1; A2 C1 C2 R1
316I gl6 lg2 a1 et1; A2 C1 C2 Dt1 R1
317F gl6 ts4 lg2
318A lg1
318B ba1
318C y10-7748
319A lg2 A1-b(P415) et1; A2 C1 C2 Dt1 R1
319C lg2 a1-m et1; A2 C1 dt1 R1
319D lg2 a1-m et1; A2 C1 Dt1 R1
319F lg2 a1-st et1; A2 C1 C2 Dt1 R1
320A lg2
320C lg2 na1
320F A1 sh2; A2 b1 C1 pl1 R1
320H lg2 a1; A2 C1 C2 R1
321A A1-d31; A2 C1 R1
321B lg2 a1; A2 C1 C2 dt1 R1
321C lg2 A1-b(P415) et1; A2 C1 C2 dt1 R1
321D a1-m4; A2 C1 C2 R1
321E a1-rUq; A2 C1 C2 R1
322A A1-d31 sh2; A2 C1 dt1 R1
322B A1-d31 sh2; A2 C1 Dt1 R1
322F a1-m; A2 b1 C1 dt1 pl1 R1
322G a1; A2 C1 C2 R1
323A a1-m; A2 C1 Dt1 R1
323E a1-m et1; A2 C1 C2 Dt1 R1
324A a1-st; A2 C1 Dt1 R1
324B a1-st sh2; A2 C1 C2 Dt1 R1
324E a1-st et1; A2 C1 Dt1 R1
324G a1-st; A2 C1 dt1 R1
324H a1 et1; A2 C1 C2 dt1 R1
325A a1-p et1; A2 C1 dt1 R1
325B a1-p et1; A2 B1 C1 Dt1 Pl1 R1
325C a1-x1
325D a1-x3
325E A1 Ga7; A2 C1 C2 R1
325G a3
325I a1-p; A2 C1 C2 Dt1 R1
325J a1-p; A2 C1 Pr1 R1
325K a1-m3 sh2-m1::Ds; A2 Ac C1 C2 R1
326A sh2
326B vp1
326C Rp3
326D te1
327A TB-3La (3L.10; B)
327B TB-3Sb (3S.50; B)
327C TB-3Lc
327D TB-3Ld
328A Trisomic 3
329A v*-9003
329B v*-8623
329C w*-022-15
329D yd2
329E w*-8336
329F yg*-W23
329G w*-062-3
329H v*-8609
329HA v*-8959
329HB yel*-8630
329HC yel*-5787

329I pg2
329Z T3-B(La); T3-B(Sb)
330A h1
330C d1 Lg3-O h1
330G a1-mrh; A2 C1 C2 Mrh R1
330H A1-b(P415) Ring 3; A2 C1 C2 R1
330I a1-Mum2; A2 C1 C2 MuDR R1
330J a1-Mum2; A2 C1 C2 R1
330K a1 sh2; A2 C1 C2 dt1 R1
331A TB-1La-3L5267
331B TB-1La-3L4759-3
331C TB-1La-3L5242 (1L.2; 3L.65)
331E TB-3Lf
331F TB-3Lg
331H TB-3Li
331I TB-3Lj
332B dek5
332C dek24-N1283
332D Wrk1-N1020
332G dek6-N627D
332H dek17-N330D
332I Lxm1-N1600
332J ms23
332L brn1
332M Spc1-N1376
332N wlu1-N28
332P g2 brn1
332Q brn1 cr1
332R brn1 ra2 lg2

CHROMOSOME 4

401A Rp4
401B Ga1
401C Ga1 su1
401D Ga1-S
401F Ga1-S C2; A1 A2 C1 R1
401I ga1 su1
401J Ga1-M
401K Ga1-S su1
402A st1
402C fl2 st1
402D Ts5
403A Ts5 fl2
403B Ts5 su1
403F Ts5 su1 o1
403G Ts5 o1
405B la1
405D la1 su1 gl3
405G la1 su1 gl4
406C fl2
406D fl2 su1
407B fl2 su1 bm3
407D su1
407E su1-am
407F su1-am; du1
408B su1 bm3
408C su1 zb6
408E bm3
408J su1 ra3
408K su1 se1
409A su1 zb6 Tu1
410D su1 zb6 gl3
411A su1 gl4 j2
411B su1 gl4 o1
411F su1 gl7 v17
412C su1 gl3
412E su1 j2 gl3
412G su1 gl4 Tu1
413A su1 o1
413B su1 gl4
413D su1 C2-Idf1(Active-1); A1 A2 C1 R1
413F su1 de*-414E
413G v23 Su1 gl3
414A bt2
414B gl4
414C gl4 o1
414E de*-414E
414F bm3 gl4
415A j2
416A Tu1
416B Tu1-I(1st)
416C Tu1-I(2nd)
416D Tu1-d
416E Tu1-md
416F Tu1 gl3
417A j2 gl3
417B v8
417C gl3
417D o1 gl3
418A gl3 dp1
418B c2; A1 A2 C1 R1
418D C2-Idf1(Active-1); A1 A2 C1 R1
418E dp1
418F o1
418G v17
419A v23
419D o1 c2; A1 A2 C1 R1
419E gl7
419F Dt6 gl3; a1-m A2 C1 R1
419H c2-m1::Spm; A1 A2 C1 R1
420A su1 Dt4; a1-m A2 C1 R1
420B TB-9Sb-4L6504
420C nec*-rd
420D yel*-8457
420F dp*-4301-43
420G w*-9005
420H C2 Dt4; a1-m A2 C1 R1
420I TB-9Sb-4L6222 (B; 9S.40; 9S.68; 4L.03)
421A TB-4Sa (4S.25; B)
421B TB-1La-4L4692
421C TB-7Lb-4L4698
422A Trisomic 4
423A TB-4Lb
423B TB-4Lc
423C TB-4Ld
423D TB-4Le
423E TB-4Lf (4L.18; B)
427A cp2-o12
427AA cp2-dek7
427AB cp2
427B dek25-N1167A
427C Ysk1-N844
427D orp1-N1186A; orp2-N1186B
427E dek8-N1156A
427F dek10-N1176A
427G Ms41-N1995
427H dek31-N1130
427I Sos1-ref
428A gl5 Su1; gl20
428C nec5-N642A
428D spt2-N1269A
428F lw4; Lw3
428G bx1
428H gl5 su1; gl20

CHROMOSOME 5

501A am1 a2; A1 C1 R1
501B lu1
501D ms13
501E gl17
501G gl17 a2; A1 C1 C2 R1
501I am1
502A gl17 a2 bt1-R v2; A1 C1 R1
502B A2 ps1-vp7 pr1; A1 C1 R1
502D A2 bm1 pr1; A1 C1 R1
502F Nl2-N1445
504A A2 bt1-R pr1; A1 C1 R1
504C A2 bm1 pr1 zb3; A1 C1 R1
505B A2 pr1 ys1; A1 C1 R1
505C A2 bt1-R pr1 ga*-Rhoades; A1 C1 R1
506A A2 v3 pr1; A1 C1 R1
506B A2 pr1; A1 C1 R1
506C A2 pr1 v2; A1 C1 R1
506D na2 A2 pr1; A1 C1 R1
506F A2 pr1 v12; A1 C1 R1
506L A2 pr1 br3; A1 C1 R1
507A a2; A1 C1 R1
507B a2 bm1 bt1-R bv1 pr1; A1 C1 C2 R1
507F a2 bm1 bt1-R ga*-Rhoades; A1 C1 C2 R1
507G a2 bm1 bt1-R; A1 C1 C2 R1
508A a2 bm1 bt1-R pr1; A1 C1 C2 R1
508C a2 bt1-R bv1 pr1; A1 C1 R1
508F a2 bm1 pr1 ys1; A1 C1 R1
510A a2 bm1 pr1 v2; A1 C1 R1
510G a2 bm1 pr1 eg1; A1 C1 R1
511C a2 bt1-R pr1; A1 C1 R1
511F a2 bt1-R Pr1; A1 C1 C2 R1
511H a2 bt1-R; A1 C1 C2 R1
512B a2 v3 pr1; A1 C1 R1
512C a2 bt1-R pr1 ga*-Rhoades; A1 C1 R1
513A a2 pr1; A1 C1 R1
513C a2 pr1 v2; A1 C1 R1
513D A2 pr1 sh4; A1 C1 C2 R1
513E a2 pr1 v12; A1 C1 R1
515A vp2
515C ps1-vp7
515D bm1
516B bt1-R
516C ms5
516D td1 ae1
516G A2 bm1 pr1 yg1; A1 C1 R1
517A v3
517B ae1
517E ae1 pr1 gl8
518A sh4
518B gl8
518C na2
518D lw2
519A ys1
519B eg1
519C v2
519D yg1
519E A2 pr1 yg1; A1 C1 R1
519F A2 pr1 gl8; 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-N409
521B Nec*-3-9c
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
521M zb*-5588
521N lnec*-5931
522A TB-5La (5L.10; B)
522B TB-5Lb
522C TB-5Sc (5S.30; B)
527A dek18-N931A
527B dek9-N1365
527C dek26-N1331
527D dek27-N1380A
527E grt1
527F nec7-N756B
527I ppg1-N199A
527J nec6-N493
528A Hsf1-N1595
528B wgs1-N206B
528C anl1-N1643
528D TB-1La-5S8041 (B; 1L.20; 1L.80; 5S.10)

CHROMOSOME 6

601C rgd1 y1
601D rgd1 Y1
601F po1-ms6 y1 pl1
602A po1-ms6 y1 wi1
602C y1
602J y1-w-mut
602K y1-gbl
602L y1-pb
603A y1 l10
603B y1 l11-4120
603C l12 y1
603CA l12-4920 y1
603D w15-8896 y1
603H mn3-1184 y1
604D y1 l15
604F y1 si1-mssi
604H y1 ms1
604I Y1 ms1
605A y1 wi1 Pl1
605C y1 pg11; pg12 wx1
605E Y1 wi1 Pl1
605F Y1 wi1 pl1
606A Y1 pg11; pg12 Wx1
606B y1 pg11; pg12 wx1
606C Y1 pg11; pg12 wx1
606E y1 pl1
606F y1 Pl1
606I y1 pg11 su2; pg12 Wx1
607A y1 Pl1-Bh1; A1 A2 c1 R1 sh1 wx1
607C y1 su2
607E y1 pl1 su2 v7
607H y1 Pl1-Bh1; A1 A2 c1 C2 R1 sh1 Wx1
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
610I Y1 Pl1 su2 v7
611A Pl1 sm1; P1-rr
611D Pt1
611E Y1 pl1 w1
611I sm1 py1; P1-rr
611K Y1 Pl1 w1
611L w1; l1
612A w14
612B po1-ms6
612C I*-4923
612D oro1
612I py1
612J w14-8657
612K w14-8050
612L w14-6853
612M w14-025-12
612N w14-1-7(4302-31)
613A 2NOR; A1 a2 bm1 C1 pr1 R1 v2
613D vms*-8522
613F w14-8613
613H pg11-6853; pg12-6853
613L w*-8954
613M yel*-039-13
613N yel*-7285
613P yel*-8631
613T pg11-6656; pg12-6656
613U wh*-8624
614A TB-6Lb
614B TB-6Sa (6S.50; B)
614C TB-6Lc (6L.11; B)
615A Trisomic 6
627A dek28-N1307A
627B dek19-N1296A
627C vp*-5111
627D hcf26
627E Dt2; a1-m A2 C1 C2 R1 TB-6Lc

CHROMOSOME 7

701B ln1-D
701C ln1-D gl1
701D o2
701F Hs1
702A v5 o5
702B o2 v5 ra1 gl1
703A o2 v5 gl1
703D o2 ra1 gl1
703J Rs1-O
703K Rs1-Z
704A o2 ra1 gl1 ij1
704B o2 ra1 gl1 sl1
704C o2 v5 gl1 sl1
705A o2 gl1
705B o2 gl1 sl1
705D o2 bd1

706A o2 sl1
707A y8 v5 gl1
707B in1; A1 A2 C1 pr1 R1
707C in1 gl1; A1 A2 C1 C2 pr1 R1
707D v5
707E vp9
707F y8 gl1
707G in1 gl1; A1 A2 C1 C2 Pr1 R1
708A ra1
708G y8
709A gl1
709C gl1-m
710A gl1 Tp1
710B gl1 mn2
710E o5 gl1
710H ms7 gl1 Tp1
711A Tp1
711B ij1-ref::Ds
711G ts*-br
712A ms7
712B ms7 gl1
712D ij1 bd1
713A Bn1
713E Bn1 bd1
714A Pn1
714B o5
714D va1
714H gl1 va1 Bn1
715A Dt3; a1-m A2 C1 R1
715C gl1 Dt3; a1-m A2 C1 R1
715D in1 Dt3; a1-m A2 C1 pr1 R1
716A v*-8647
716B yel*-7748
716F Les9-N2008
717A TB-7Lb (7L.30; B)
718A Trisomic 7
719A TB-7Sc (7S.08; B)
720A Dt3; a1-m TB-7Lb
727A dek11-N788
727B wlu2-N543A
727E gl1-cgl
727G Rs1-O o2 v5 ra1 gl1
728A Px3-6
728B ptd2-Mu3193
728C cp1
728D sh6-8601
728E sh6-N1295

CHROMOSOME 8

801A gl18-gl23
801B v16
801C v16 j1
801D v16 ms8 j1
801I yel*-024-5
803A ms8
803B nec1-025-4
803F nec1-7748
804A v21-A552
804B dp*-8925
804D wh*-053-4
804E w*-017-4
804F w*-034-16
804G w*-8635
804H w*-8963
805A fl3
805D fl3 ms8 j1
805E el1
805G ms8 j1
806A TB-8La (8L.70; B)
806B TB-8Lb
807A Trisomic 8
808A ct1
809A TB-8Lc (8L.24; B)
810A v16 j1; l1
810B j1
810C gl18 v21-A552 j1
827A dek20-N1392A
827B dek29
827C Bif1-N1440
827D Sdw1-N1592
827E Clt1-N985
827J wlu3-N203A
827K pro1
827L pro1-Tracy

CHROMOSOME 9

901B yg2 C1 sh1 bz1; A1 A2 C2 R1
901C yg2 C1 sh1 bz1 wx1; A1 A2 C2 R1
901E yg2 C1 bz1 wx1; A1 A2 R1
901H yg2 C1 Bz1; A1 A2 C2 R1
902A yg2 c1 sh1 bz1 wx1; A1 A2 R1
902B yg2 c1 sh1 wx1; A1 A2 R1
902C yg2 c1 sh1 wx1 gl15; A1 A2 R1
903A C1 sh1 bz1; A1 A2 R1
903B C1 sh1 bz1 wx1; A1 A2 R1
903D C1-l sh1 bz1 wx1; A1 A2 R1
904B C1 sh1; A1 A2 R1
904D C1 wx1 ar1; A1 A2 R1
904F C1 sh1 bz1 gl15 bm4; A1 A2 C2 R1
905A C1 sh1 wx1 K9S-l; A1 A2 C2 R1
905C C1 bz1 Wx1; A1 A2 R1
905D C1 sh1 wx1 K9S-l; A1 A2 C2 K10 R1
905E C1 sh1 wx1 v1; A1 A2 C2 R1
905H c1 sh1 wx1; A1 A2 b1 C2 R1-sc:m2
906A C1 wx1; A1 A2 C2 Dsl Pr1 R1 y1
906B C1 wx1; A1 A2 C2 Dsl pr1 R1 Y1
906C C1-l Wx1; A1 A2 C2 Dsl R1
906D C1-l; A1 A2 C2 R1
907A C1 wx1; A1 A2 C2 R1
907E C1-l wx1; A1 A2 C2 R1 y1
907G c1-p; A1 A2 B1-b C2 pl1 R1
907H c1-n; A1 A2 b1 C2 pl1 R1
908A C1 Wx1 ar1 da1; A1 A2 C2 R1
908D C1 wx1 gl15; A1 A2 C2 R1
908F C1 wx1 da1; A1 A2 C2 R1
908H C1 wx1; A1 A2 C2 R1 y1
909A C1 wx1 Bf1; A1 A2 C2 R1
909B c1 bz1 wx1; A1 A2 C2 R1
909C c1 sh1 bz1 wx1; A1 A2 C2 R1 y1
909D c1 sh1 wx1; A1 A2 C2 R1
909E c1 sh1 wx1 v1; A1 A2 C2 R1
909F c1 sh1 wx1 gl15; A1 A2 C2 R1
909J c1 sh1 bz1 gl15 bm4; A1 A2 C2 R1
910B c1 sh1 wx1 gl15 Bf1; A1 A2 C2 R1
910D c1; A1 A2 C2 R1
910G C1 sh1-bz1-x2 Wx1; A1 A2 C2 R1
910H C1 sh1-bz1-x3; A1 A2 C2 R1
911A c1 wx1; A1 A2 C2 R1 y1
911B c1 wx1 v1; A1 A2 C2 R1
911C c1 wx1 gl15; A1 A2 C2 R1
912A sh1
912B sh1 wx1 v1
912E lo2
913C sh1 l7
913D sh1 l6
914A wx1 d3-N660B
914K Wc1-ly; Y1
915A wx1
915B wx1-a
915C w11
916A wx1 v1
916C wx1 bk2
917A wx1 Bf1
917C v1
917D ms2
917E gl15
917F d3-N660B
918A gl15 Bf1
918B gl15 bm4
918C bk2 Wc1
918D Wc1
918F Wx1 Bf1
918G Wc1-Wh Bf1 bm4
918K bk2 v30-8587
919A bm4
919B Bf1 bm4
919C l6
919D l7
919G l6; l1
920A yel*-034-16
920B w*-4889
920C w*-8889
920E w*-8950
920F w*-9000
920G Df3; Tp3-9
920H pg12-yg8322; pg11
920L ygzb*-5588
920M wnl*-034-5
920N pyd1
921A TB-9La
921B TB-9Sb (9S.40; B)
921C TB-9Lc (9L.10; B)
922A Trisomic 9
922B Wc1; TB-9Lc
922C C1-l; TB-9Sb
922D TB-9Sd (9S.18; B)
923A wx1-a
923B wx1-B
923C wx1-B1
923D wx1-B2::TouristA
923E wx1-B3::Ac
923F wx1-B4::Ds2
923G wx1-B6
923H wx1-B7
923I wx1-B8
923J wx1-BL2
923K wx1-BL3
923L wx1-C
923M wx1-C1
923N wx1-C2
923O wx1-C3
923P wx1-C4
923Q wx1-C31
923R wx1-C34
923S wx1-F
923T wx1-90
923U wx1-H
923V wx1-H21
923W wx1-I
923X wx1-J
923Y wx1-M
923Z wx1-M1
923ZA wx1-M6R
923ZB wx1-M6NR
923ZC wx1-M8
923ZD wx1-P60
923ZE wx1-R
923ZF wx1-Stonor
924A C1 C1-l wd1 Wd1+ Ring 9S; A1 A2 C2 R1
924B C1-l Ring 9S; A1 A2 C2 R1
924C yg2
924D wd1
927A dek12-N873
927B dek13-N744
927C dek30-N1391
927D Les8-N2005
927E Zb8-N1443
927H C1 Dt7; a1-r A2 C2 R1
927I G6-N1585
927J wx1 dt7; a1-r
928A v28-N27
928B wlu4-N41A
928G c1-m5::Spm wx1-m8::Spm-l8; A1 A2 C2 R1
928H wx1-m7::Ac7
928I C1 bz1-mut::Mut; A1 A2 Bz2 C2 R1
929A TB-9 isochromosome Type 1
929B TB-9 isochromosome Type 2
929C T9-B(La); T9-B(Sb)
929D TB-9 isochromosome (original)
929E Dp9
929F T9-B (La + Sb)
929G T9-8(4453); TB-9Sb
929H T9-3(6722); TB-9Sb
929I TB-9Sb-1866
929J TB-9Sb-1852
929K TB-9Sb-2150
929L TB-9Sb-14
929M TB-9Sb-2010

CHROMOSOME 10

X01A oy1
X01B oy1 R1; A1 A2 C1
X01C oy1 bf2
X01E oy1 bf2 R1; A1 A2 C1
X02C oy1 zn1 R1; A1 A2 C1 C2
X02E oy1 du1 r1; A1 A2 C1 C2
X02G oy1 zn1
X03A sr3
X03B Og1
X03E oy1 y9
X04A Og1 du1 R1; A1 A2 C1
X04B ms11
X04D bf2
X05A zn1 bf2
X06C nl1 g1 R1; A1 A2 C1 C2
X06F bf2 R1 sr2; A1 A2 C1 C2
X07C y9
X07D nl1
X09B li1 g1 R1; A1 A2 C1 C2
X09F ms10
X09G li1 g1 r1; A1 A2 C1 C2
X10A du1
X10D du1 g1 r1; A1 A2 C1 C2
X10F zn1
X10G du1 v18
X11A zn1 g1
X11C zn1 g1 r1; A1 A2 C1 C2
X11F g1 r1; A1 A2 C1 C2
X11H zn1 R1-r; A1 A2 C1 C2
X11I Tp2 g1 R1 sr2; A1 A2 C1 C2
X12A g1 r1 sr2
X12C g1 R1-g sr2; A1 A2 C1 C2
X12E g1 R1; A1 A2 C1 C2
X13D g1 r1-r sr2; A1 A2 C1 C2
X14A r1-r lsr1-Ej; A1 A2 C1 C2
X14F r1 v18; A1 A2 C1 C2
X15B l1 r1 sr2; A1 A2 C1 C2
X15C R1-g; A1 A2 C1 C2
X15D r1-ch; A1 A2 C1 C2 Pl1
X15E Pa*-Brawn
X15F lsr1 R1-g Sr2
X15G isr1 r1-g sr2
X16B r1; A1 A2 abnormal-10 C1
X16D r1 sr2; A1 A2 C1 C2
X16F R1 K10-ll; A1 A2 C1 C2
X17A r1-g; A1 A2 C1 C2
X17B r1-r; A1 A2 C1 C2
X17C R1-mb; A1 A2 C1 C2
X17D R1-nj; A1 A2 C1 C2
X17E R1-r; A1 A2 C1 C2
X18A R1-lsk; A1 A2 C1 C2
X18C R1-st; A1 A2 C1 C2
X18D R1-sk; A1 A2 C1 C2
X18E R1-st Mst1
X18G R1-sc:m2; A1 A2 bz2 C1 C2
X18H R1-nj; A1 A2 bz2 C1 C2
X18I r1; A1 A2 C1 C2
X19B w2
X19C l1 w2
X19D o7
X19F r1 w2
X20B l1
X20C v18
X20F yel*-8721
X20H yel*-5344
X20HA yel*-8793
X20HB yg*-8962
X21A TB-10La (10L.45; B)
X21B TB-10L19 (10L.00; B)
X21C TB-10Lb
X22A TB-10Sc (10S.30; B)
X22B T1La-B-10L18
X22C TB-10Lb
X23A Trisomic 10
X24A cm1
X25A R1-sc:m2; a1-st A2 C1 C2
X25B R1-sc:m2; A1 A2 C1 c2
X25C R1-sc:m122; A1 A2 C1 C2 pr1
X25D R1-sc:m2; A1 a2 C1 C2

X25E R1-sc:m2; A1 A2 c1 C2
X25F R1-sc2; a1-st A2 C1 C2
X25G R1-sc2; A1 A2 c1 C2
X26A R1 r1-x1; A1 A2 C1 C2
X26B R1-sc:m2; A1 A2 C1 C2
X26C R1-sc122; A1 A2 C1 C2
X26D R1-sc*5691; A1 A2 C1 C2
X26E R1-sc:m2; A1 A2 C1 C2 pr1 wx1
X26F R1-sc:m2; A1 A2 C1 C2 In1-D
X26G R1-sc:m2; A1 A2 C1 c2-m2
X27A dek14-N1435
X27B dek15-N1427
X27C w2-dek21
X27D Les6-N1451
X27E gl21-N478B; gl22-N478C
X27F Vsr1-N1446
X27G Oy1-N700
X27H orp2-N1186B; orp1-N1186A
X27I I19-N425
X27J I13-N59A
X28B R1-sc:m2; a1-m1::rDt (Neuffer)
X28C R1-nj (Cudu); A1 A2 C1 C2
X28D Vsr*-N716
X28F cr4-6143
X28G R1-nj (Chase); A1 A2 C1 C2
X28H g1-ws2
X28I R1-sc:m2; a1-m1-5719::dSpm A2 C1 C2

UNPLACED GENES

U140A dv1
U140C I4
U140E I3
U140F Fas1
U140G ms22
U140H ms24
U240A Les7-N1461
U240B vp10
U240C v13
U240D o11
U340A rd3
U340B zb1
U340C zb2
U340D g1-ws2-Pawnee; ws1-Pawnee
U340E y11
U340F y12
U340G oro2
U440A o9
U440B gl13
U440C zn2
U440D ub1-76C
U440E frz1

MULTIPLE GENE

M141A A1; A2 B1 C1 C2 Pl1 Pr1 R1-g
M141D A1; A2 b1 C1 C2 pl1 R1-g
M241A A1; A2 B1 C1 C2 Pl1 Pr1 r1-g
M241C A1; A2 B1 C1 C2 Pl1 Pr1 R1-r
M341D A1; A2 B1 c1 C2 Pl1 Pr1 R1-r
M341F A1; A2 b1 C1 C2 pl1 Pr1 R1-r
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
M641D A1; A2 C1 C2 Pr1 r1 wx1 y1
M641E A1; A2 C1 C2 r1-g wx1 y1
M741A A1; A2 b1 C1 C2 pl1 Pr1 r1-g wx1
M741C Stock 6 A1; A2 B1 C1 C2 pl1 R1-r
M741F Stock 6 A1; A2 C1 C2 pl1 R1-g y1
M741G Stock 6 A1; A2 C1-I C2 pl1 R1-g wx1 y1
M841A A1; A2 C1 C2 pr1 R1 su1
M841C colored scutellum A1; A2 C1 C2 Pr1 R1
M941A A1; A2 c1 C2 Pr1 R1 wx1 y1
MX40A Mangelsdorf's tester a1 bm2 g1 gl1 j1 lg1 pr1 su1 wx1 y1
MX40D gl1; wx1 y1
MX40E gl8; 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
MX41D a1; A2 C1 C2 gl1 pr1 R1 su1 wx1 y1
MX41E a1-m1-n::dSpm; A2 C1 C2 R1 wx1-m8::Spm-I8

B-CHROMOSOME

B542A Black Mexican Sweet, B chromosomes present
B542B Black Mexican Sweet, B chromosomes absent

TETRAPLOID

N102A A1; A2 Autotetraploid B1 C1 C2 Pl1 Pr1 R1
N102C a1-m; A2 Autotetraploid C1 C2 Dt1 R1
N102D A1; A2 Autotetraploid C1 C2 R1
N102E Autotetraploid; B chromosomes present
N102EA Autotetraploid; B chromosomes present
N102F A1; a2 Autotetraploid C1 C2 R1
N103A Autotetraploid; P1-rr
N103B Autotetraploid; P1-vv::Ac
N103C Autotetraploid; P1-ww
N103D Autotetraploid; P1-wr
N103E Autotetraploid; P1-mm
N103F Autotetraploid; bz2
N104A Autotetraploid; su1
N104B A1; A2 Autotetraploid C1 pr1 R1
N105B Autotetraploid; wx1 y1
N105E Autotetraploid; bt1-R
N106C Autotetraploid; wx1
N106D Autotetraploid; sh1 Wx1 Y1
N107B W23 Autotetraploid
N107C Synthetic B Autotetraploid
N107D N6 Autotetraploid

CYTOPLASMIC STERILE/RESTORER

C736A R213 Rf1; rf2
C736B Ky21 Rf1; Rf2
C736C B37 rf1; Rf2
C736D N6 rf1; Rf2
C736E Tr rf1; rf2
C736F W23 rf1; Rf2
C736G B73 rf1; Rf2
C836A Wf9 cms-T; rf1 rf2
C836B N cytoplasm rf1; rf2

CYTOPLASMIC TRAIT

C337A NCS2
C337B NCS3

INVERSION

I143B Inv1c (1S0.3-1L.01)
I143C Inv1d (1L.55-1L.92)
I143D Inv1k (1L.46-1L.82)
I243A Inv2b (2S0.5-2L.15)
I243B Inv2h (2L.13-2L.51)
I343A Inv3a (3L.38-3L.95)
I343B Inv3b (3L.19-3L.72)
I343C Inv3c (3L.09-3L.81)
I344A Inv9a (9S0.7-9L.9)
I443A Inv4b (4S0.1-4L.12)
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 Inv6d (6S0.7-6L.33)
I743C Inv6(3712) (6S0.7-6L.63)
I843A Inv6e (6S0.8-6L.32)
I943A Inv7f (7L.17-7L.61)
I943B Inv7(8540) (7L.12-7L.92)
I943C Inv7(3717) (7S0.3-7L.3)
IX43A Inv8a (8S0.3-8L.15)
IX43B Inv9b (9S.05-9L.87)

RECIPROCAL TRANSLOCATION (wx1 and Wx1 marked)

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; 9S.07); 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; 9S.07); 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
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 (5S.07; 9L.1); Wx1
Wx34B T5-9(4817) (5L.06; 9S.07); Wx1
Wx34C T4-9b (4L.9; 9L.29); Wx1
Wx35A T5-9(8386) (5L.87; 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; 9S.07); 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

TOOLKIT

T318AA lg1 ig1; R1-nj TB-3Ld
T318AB cms-L; ig1 R1-nj
T318AC cms-MY; ig1 R1-nj
T318AD cms-ME; ig1 R1-nj
T318AE cms-S; ig1 R1-nj
T318AF cms-SD; ig1 R1-nj
T318AG cms-VG; ig1 R1-nj
T318AH cms-CA; ig1 R1-nj
T318AI cms-C; ig1 R1-nj
T318AJ cms-Q; ig1 R1-nj
T940A Hi-II Parent A (for producing embryogenic callus cultures)
T940B Hi-II Parent B (for producing embryogenic callus cultures)
T940C Hi-II A x B (for producing embryogenic callus cultures)

VIII. 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 been detected 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. Newly detected maize genes that have been previously identified in other plant species should be named where appropriate (see the last paragraph in Section 2) with reference to the list of generic names compiled by the Commission on Plant Gene Nomenclature.

When designating homozygous genotypes with two or more unlinked genes, the genes are separated by semicolons, eg. *a1;a2;c1;c2;r.* If linked, the genes are separated by spaces, e.g. *C1 sh1 bz1 Wx1*. Heterozygous genotypes should be written with a slash separating the sets of linked genes, e.g. *C1 Bz1/c1 bz1*. If the genes are unlinked, the proper designation is *Sh2/sh2; Bt2/bt2*.

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. However, if two laboratories are targeting the same locus in mutagenesis experiments, they should consult before naming their new alleles to avoid giving the same designation to different alleles. 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 April 4, 1995 by the Nomenclature Subcommittee.

William Beavis
Mary Berlyn
Benjamin Burr
Vicki Chandler
Ed Coe
Christiane Fauron
Oliver Nelson
Mary Polacco
Steve Rodermel
Marty Sachs
Carolyn Wetzel

IX. Maize Genome Database, a USDA-ARS Plant Genome Database

http://www.agron.missouri.edu/

The Maize Genome Database or MaizeDB is curated as a Sybase database, at the University of Missouri-Columbia, and provides user-friendly, Internet access to the maize genome and the biology of maize. August 1995 records included 240 genetic maps; 6227 mapped loci; 3498 map data entries, both recombination and map score data; 2335 probes; 1985 genetic/cytogenetic stocks; 19,821 locus variations, including 2800 mutants from the Neuffer collection, allozyme typing for 21 loci and 437 elite stocks; 4662 stock pedigrees; 6785 bibliographic references, indexed to genetic objects; 2430 addresses of maize researchers. Gene functionality may be queried by mutant phenotype, trait, confirmed or putative gene products, metabolic pathways, induction conditions and text descriptions of genes. Work is in progress to document quantitative trait loci. Users with Internet connections may access the data by guest login to the relational APT query forms; gopher; World Wide Web; or by file transfer of a special graphical format, ACEDB. Any data retrieved may be saved to a file on your computer(gopher, WWW) or e-mailed (gopher, WWW, relational query).

Data input is from various sources, including specially formatted, electronic lab notebooks of researchers who focus on mapping or mutant characterization; direct entry by off-site curators into the APT forms; electronic updates of pointers to external databases. These contributors are international and from academic, government , and industrial research groups. Data, especially regarding gene function and expression, are also taken from the scientific literature, both electronic and printed. The WWW and APT fill-in-the-blanks forms execute direct queries on the underlying Sybase tables, and therefore, any information entered, by off- or on-site curators, is instantly available to WWW or relational queries.

New in 1994-95 -- boxes enclose hints on retrieving information from MaizeDB using forms(WWW or APT)

1. Mapping; Maps; Data; Core(Anchor) Markers

Mappers may identify the chromosome region corresponding to their mapping data by pasting their scores into the Scores field on the MapScore (by locus) form and clicking on the Probed Site for each MapScore retrieved. When a precise match is not found, piecemeal groups representing stretches of 10 or so individuals work best. The better the match, the tighter the linkage. Sample queries--note the use of **%**, the Sybase wild-card:

For bnl maps (recombinant inbred or RI populations)
SCORES: **%2131213312**
or SCORES: **%2131213312%2212**

For umc 95 maps (immortal F2 or IF2 populations)
SCORES: **%BHHHAHHHB**
OR SCORES: **%BHHHAHHHB%AABBH**

New Maps

Mycogen (365 markers)
UMC 1995 RFLP (602 markers)--with map scores
CIMMYT (346 markers)
Mitochondrial (two normal and T-cytoplasm restriction fragment maps)

A table of core or anchor markers that define mapping bins is stored as a hypertext file on the WWW and may be accessed from the top page for MaizeDB under 'Of Interest to Maize Cooperators'. A Probe request form is also accessible from that page.

Map retrieval. Retrieve the above maps and the previously entered BNL maps from the Map form after typing within the Name, Source or Linkage Group field:

NAME: **Mycogen** to retrieve all Mycogen maps
NAME: **Mycogen%2** to retrieve Mycogen chromosome 2
NAME: **umc 95** [or NAME: **umc 95%2**] for umc 95 maps [chromosome 2 only]
SOURCE: **CIMMYT** all CIMMYT maps
LINKAGE GROUP: **mito**
NAME: **bnl** [or NAME: **bnl%2**] for bnl 1993 map [chromosome 2 only]
NAME **bins** [or NAME: **bins%2**] for bins maps [chromosome 2 only]
NAME: **%** for a list of all the maps

Retrieve Complete Map Scores, in order by chromosome and coordinate, for umc 95 maps on the WWW from the Map Score Tables form, by selecting Panel Type: **Immortal F2 .** Retrieve BNL map score panels similarly, but select Panel Type: **Recombinant Inbred.** The dynamically extracted table of map scores is currently accessible only by WWW.

2. Representation of QTL Data

Design has been completed and data entry is on-going. QTL representation will facilitate comparisons with genome information obtained from classical, biochemical and molecular genetic studies. Traits where public information is available include yield and yield components, quality attributes, such as kernel protein and starch concentrations, developmental characteristics and responses to pests and abiotic stress. A full description of how to access the data is provided in an on-line JQTL article accessible as a QTL Experiment form 'hint' on the MaizeDB WWW directory of focused query forms.

A list of QTL experiments that have been entered into the database may be retrieved from the QTL Experiment form, by typing within the name field:

Name: %

3. Allozyme variations

These data were provided in electronic form by either Charles Stuber or Alex Kahler and include both null alleles and electrophoretic variants or allozymes for some 21 loci, as surveyed across 437 maize lines.

A list of the germplasm or stocks with characterized allozymes or null alleles may be retrieved on the Stock form by typing within the 'phenotype' field :

Phenotype: **electro**
(or
Phenotype: electrophoretic mobility)
Phenotype: **null**

4. WWW; connections to 18 other databases

The MaizeDB World Wide Web server is seamlessly connected to files, including images and graphics, from other databases around the world, as though the data existed in the MaizeDB records. Free software, Mosaic or Netscape, is required to browse files on the WWW, or to store the information on your machine. For example, while browsing the MaizeDB, you may read that the function of maize gene, *dps1,* was confirmed by transgenic complementation of *E. coli* mutations in *dap1.* By clicking on *dap1,* you would retrieve the record from the *E. coli* Stock Center at Yale University. Clicking on the EC number, for gene products that are enzymes, connects to another database, ENZYME, which describes the reaction and in turn, connects to all SwissProt entries corresponding to that EC number and, also, to OMIM (On-line Mendelian Inheritance in Man).

WWW connectivity requires precise matching of records in MaizeDB to records in other databases around the world. It permits curators of distinct datasets to combine data, in a seamless fashion, without actually importing the data and maintaining updates. The ability to extract distinct formats of data from MaizeDB make it relatively easy to prepare files of matching identifiers so that external databases may use the WWW to connect to us, as occurred June 1994 with SwissProt.

MaizeDB records currently access external data in the following databases (-> indicates further connections through the external database). Generally connections are made by clicking on the 'Key' for the external database record; clicking on the database name provides access to the external database home-page. Other connections, such as gene product EC# or related genes in other databases, 'hop' directly to the external database.

<u>Sequences</u>

dbEST random cDNA's partial sequences (EST's), with periodically updated similarity (BLAST) searches; updates of connecting identifiers automatically delivered to MaizeDB
GenBank nucleotide sequences
GSDB nucleotide sequences
SwissProt protein sequences; **-> Prosite** (motifs, signature sequences); **-> MedLine**
PIR protein sequences
GenoBase nucleotide sequences, **->** other sequences submitted by same researcher; performs updated (BLAST) similarity searches on retrieved sequences (if less than 4000 bp); useful for updates on EST sequenced EST's (see dbEST above)

Genomes, Germplasm

AAtDB *Arabidopsis* Genome Database
***E. coli* Stock Center** *E. coli* genetic stocks, map; **-> ENZYME; -> GenBank**
GrainGenes Wheat, Barley, Oats, etc. Genome Database
GRIN Germplasm Resources Information Network
RiceGenes Rice Genome Database
SGB *Saccharomyces* Genome Database
Xlocus Multi-genome database

Metabolism

ENZYME reactions, comments; **-> SwissProt** (all species); **-> OMIM**
Klotho metabolic compounds database

References

J Biol Chem abstracts, articles (since April 1995)
JQTL on-line journal sponsored by Crop Science Society of America
Agricola abstracts (connection made; electronic matching tool under development).

Sequenced Locus Retrieval. A list of loci with sequences in GenBank may be retrieved from the Locus form typing within the field External Database,

EXTERNAL DATABASE: **Gen** or **GenBank**

This query may be further constrained to only loci with mutant phenotypes by additionally typing within the Phenotype field:

PHENOTYPE: **%**

Or for loci with sequences in GenBank, and with some map location, type within the map coordinates field:

VALUE: **%**

5. E-mail to cooperators. Using the WWW server, clicking on the e-mail address of a Person record will activate an e-mail form addressed to that Person.

DEVELOPERS AND CURATORS OF THE DATABASE INCLUDE: EH Coe (PI), P. Byrne, G. Davis, D. Hancock, M. Polacco (Columbia, MO) M. Berlyn (New Haven, CT), S. Letovsky (Baltimore, MD) C. Fauron (Salt Lake City, UT), S. Rodermel, C. Wetzel (Ames, IA) M. Sachs (Urbana, IL)

For technical help in accessing the database, please e-mail **db_request@teosinte.agron.missouri.edu** or contact Denis Hancock, 314-882-1722 (phone), 314-874-4063 (fax).

ACCESSING MaizeDB - WHAT IS REQUIRED.

Guest login only requires that your machine have **Internet connectivity,** direct or indirect; modem connections are supported, as are connections using **any computer, including PC, MacIntosh and UNIX.**

->guest login protocol (telnet is the UNIX command, not needed for other machine software):

telnet teosinte.agron.missouri.edu
login(user name): **guest**
password: **corncob**

Guest login provides access to:

gopher
Sybase APT forms; both X-Window and vt100 emulation users supported
Lynx, a WWW browser that does not require an X-Window
help

Guests are encouraged to type comments on the Note form of the database. While not required, leaving your e-mail address will permit us to contact you directly for further clarification or to provide help.

NOTE: Users with X-Windows (this is not the same as Microsoft Windows) software will enjoy the most user-friendly access to the APT Sybase forms for fully relational queries of the database. If connecting by modem, the X-Window will not function, and users should select the vt100 emulation.

NOTE: If using the vt100 emulation of MaizeDB/Sybase, type '**r**' while holding down the '**control**', or '**CTRL**' key to access the commands required to query or browse the database. The command utilities are described in more detail in the 'help' option that appears after login as a guest.

->gopher makes available hierarchical collections of information across the Internet. Gopher client (user) software provides easy access to all gopher data servers on the Internet. All words in a record, except commonly used words, are indexed, and thus, may be used to query records. Free gopher client software, for UNIX, PC, or MacIntosh machines, is available by anonymous ftp from boombox.umn.edu. Once installed, open server gopher.agron.missouri.edu, port 70 or use gopher to find us by location in Columbia, Missouri. On-line help is provided by the gopher software and in a file on the MaizeDB gopher server. Our gopher server supports login to the SYBASE format.

->World Wide Web (WWW) WWW is a hypermedia or interactive-file retrieval system and allows users to traverse on-line documents by clicking on hyper links - terms, icons or images that point to other related documents. Hyper links permit retrieval of any document anywhere on the Internet. Retrieved 'documents' may include text files, graphics, and videos. Users without WWW software may access the WWW-linked format of MaizeDB by selecting the Lynx option after 'guest login'. Browsing software, such as Mosaic, supports mouse capability and is available without charge by anonymous ftp from ftp.ncsa.uiuc.edu. The UNIX version, but not the MacIntosh or PC versions, requires an X-Window on the user's machine; it will require a systems administrator to install. Netscape software is similar to Mosaic and available by anonymous ftp from ftp.mcom.com. Netscape on a machine with SLIP connections to the Internet, may access the World Wide Web by modem. To access MaizeDB on the WWW, use the MaizeDB **WWW address,** otherwise known as **URL** or uniform resource locator:

http://www.agron.missouri.edu

The WWW formatted data are dynamically extracted from the most current version of the database, which is continuously updated.

-> ACEDB format This is a special, stand-alone, graphical format and requires a UNIX machine. The database may be retrieved by anonymous ftp from the National Agricultural Library, **probe.nalusda.gov** in directory **pub/maizedb** and in file macedist950414.tar.Z. ACEDB formatted information is periodically extracted from MaizeDB. It does not support the robust, fill-in-the-blanks form queries of the Sybase database, accessible by the guest login service and by WWW.

ANONYMOUS FTP requires that the user have ftp or file transfer software to connect to another machine. Once connected, login as '**anonymous**' and use **your e-mail address** as the password. If using a UNIX machine, type: **cd pub/maizedb** and to transfer the database, type **binary** and, on a separate line, type **get macedist950414.tar.Z**.

HISTORY OF MAIZEDB DESIGN: SOME LANDMARKS

Fall 1991 First prototype MaizeDB operational. Some 24,000 records created the first 6 months, largely from data summaries in the Maize Genetics Cooperation Newsletter (MNL) volume 65.

Dec 1992 First public access to the data: a gopher server established. First Gopher access was 100-200 transactions/month, and steadily grew over the first 2 years to over 5,000 transactions/month.

March 1993
- o Maize Gene List, MNL vol 67 pp 134-15, extracted from MaizeDB
- o Version 2 of MaizeDB design implemented

March 30, 1993 40,055 records in the database

June 1993
ACEDB formatted data extracted from MaizeDB

August 1993 Tool developed for electronic reference loading, with matching to authors and previously loaded reference, from PC or MacIntosh reference manager formats. This tool has been used for loading various reference sets, including those of MNL or in Mutants of Maize (in press 1995).

Fall 1993 MaizeDB placed on World Wide Web

Dec 31, 1993 Guest login to MaizeDB(SYBASE) established that permits access by modem or other connection on the Internet, and by any machine with telnet software.

Jan 3, 1994 61,544 records in the database

Winter 1994
- o MaizeDB records connected to external databases on the World Wide Web; growth of WWW access has steadily increased from 200 transactions/month to over 20,000 WWW transactions per month.
- o WWW connections made to external databases from within individual MaizeDB records

March 1994 Genetic indexing of 1993 references extracted from the MaizeDB, published as hardcopy (Zealand) in MNL, vol 68. pp 148-153. Information was indexed to chromosome, gene or allele and trait.

June 1994 SwissProt connects to MaizeDB using a file extracted from MaizeDB per specifications of SwissProt curators; updates of connectors exchanged regularly.

December 1994 QTL forms implemented; first data entered.

Jan 1, 1995 96,900 records in the database

January 1995 Permanent curator position established.

X. MAIZE PROBE BANK

CLONES FOR DISTRIBUTION FROM THE UMC RFLP LABORATORY

The following is a summary of DNA clones that are currently available for distribution as stabs from the UMC RFLP Laboratory.

Clone Set	Abbreviation	Number Distributable
Asgrow	asg	85
Brookhaven National Lab.	bnl	109
California State University	csu	784
Tripsacum-UMC	tda	20
Mycogen Plant Genetics	agr	413
Pioneer Hi-Bred International	php	161
Pioneer Hi-Bred International	npi (#'s greater than 100)	236
University of Arizona	uaz (5C & 6C)	1632
University of Missouri	umc	238

Please note that within these categories of clones we may not have or are not permitted to distribute all clones of that designation. We do not distribute npi clones with numbers less than 100, nor certain umc clones that were sent to us with restrictions. We distribute specific defined-function clones on a case-by-case basis. We do not have uaz clones in the 1C, 2C, 3C and 7C series, and are unable to provide these at this time. To be certain that a particular clone is available please check the Available From line on the probe form of the maize database--clones available from the UMC RFLP Laboratory will show T. Musket, who is the clone distribution coordinator. Clicking on T. Musket will give address and e-mail information. The easiest method to request clones is to use the probe request form directly from the maize database WWW homepage URL=http://teosinte.agron.missouri.edu. Please be certain to enter information in each field on the request form to aid our processing of your request. If you are unable to use the World Wide Web, send your request to Theresa A. Musket (address, phone and email in this Newsletter; FAX is 314-874-4063). Please provide your name, full mailing address, and email address if available.

Over the last two years the number of clones for which we are responsible for distribution has grown from a few hundred to greater than three thousand. Our resources, both personnel and financial, for maintenance and distribution of clones are very limited. Please request only those clones necessary for your experiments. Because of concurrent research commitments we have been having difficulty answering clone requests as rapidly as we (and you) would like. Please request clones as far in advance of need as possible.

We thank Asgrow Seeds, Mycogen Plant Genetics and Pioneer Hi-Bred International for their generous donations of probes to the maize research community. The cooperation of individuals in making defined-function clones available is equally appreciated.

RFLP ACRONYMS IN USE

agr Agrigenetics
asg Asgrow Seed
ast Academica Sinica, Taiwan
bcd barley cDNA, Cornell University
bnl Brookhaven National Laboratory
cdo oat leaf cDNA, Cornell University
crc Carlsberg Research Center
csh Cold Spring Harbor
csic Centro de Investigacion y Desarrollo, Barcelona
csu California State University, Hayward
cuny City University of New York
dnap DNA Plant Technologie Corp
dup Dupont
gii Genetics Institute Inc.
ias Iowa State University
isc Ist Sper Cereal
koln University of Koln
mpik Max-Planck-Institute, Koln
ncr North Carolina Raleigh
ncsu North Carolina State University
niu Northern Illinois University
npi Native Plants Incorporated
pge Plant Gene Expression Center
php Pioneer Hi-Bred International
psu Penn State University
rg rice genomic, Cornell University
rny Rockefeller University
rpa Rhone Poulenc
rz rice cDNA, Cornell University
std Stanford University
uaz University of Arizona
ucb University of California-Berkley
ucla University of California-Los Angeles
ucr University of California-Riverside
ucsd University of California, San Diego
ufg University of Florida-Gainesville
uiu University of Illinois-Urbana
umc University of Missouri-Columbia
umn University of Minnesota
uox University of Oxford
uwo University of Western Ontario
wsu Washington State University
wusl Washington University, St. Louis
ynh Yale University

XI. GENE LIST AND WORKING MAPS

GENELIST: A table of the defined gene loci of maize, extracted from the Maize Genome Database (MaizeDB - see section IX), follows. The table includes the symbol for the locus; the location in 'bins' as described below; the locus name with a brief phenotypic description; and references to first reports or publications central to the designation of the locus. These publications are prefixed with "g" for genelist. 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.

Leaps: The genelist is dynamic in MaizeDB, and through it an increasingly refined and consistent production and expansion of the list continues. New mapped loci identified by directly visible mutations, and new mapped loci defined by probing with sequences from clones with specifically targeted functions, contribute most to the growth. There is a growing volume of cDNA sequences for which a defined function can be identified, and the result is a conspicuous expansion of our knowledge resource. Nearly two-thirds have been mapped or have been placed to linkage group.

Bounds (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. We make an effort to reflect the evidence faithfully, and try to be conservative in designating a gene before its time, but in at least a few instances the temptation to designate a gene may have swept criteria aside because of its significance. In other words, the Genelist may sometimes include or exclude inappropriately, and we invite your communications. Several hundred candidate-function clones await evidence - see the next paragraph.

Bins: The bin locations of genes are given as one number whenever localization appeared to be sufficiently firm. In cases where one or more bins were prospects, the range of possible bins is given. The assignments should be considered tentative, and are based on reading *from* the new map after they were drawn, rather than *to* the map (see below). The bin boundaries and bin identifications were substantially changed in a number of instances in the new Core Map, as described further in the map section.

Finds: In need of ideas or possible projects? See the table from the cDNA project, provided by Tim Helentjaris, that follows the genelist. Note especially, however, that the bin assignments in this table are based on 1994 rather than 1995 bins.

Nomenclature: The Standard for Maize Nomenclature is reprinted in the preceding section.

Ed Coe, Denis Hancock, Shirley Kowalewski and Mary Polacco

RFLP AND GENETIC MAPS: The current UMC Maize RFLP Map, containing more than 600 loci, is cut into left and right portions (note the *arrows* at the point of surgery) and is accompanied by a Genetic Working Map, containing more than 660 loci, presented *as a matched map on a parallel line* to the right of the RFLP Map.

The **RFLP Map** shows the current core markers in boxes - these are loci defined with a revised core probe set available from the lab upon request (**see section X for information on how to request** the core probe set, or probes for additional loci listed on this map, through MaizeDB on the World Wide Web or by other routes). Most of the probes are publicly available from the University of Missouri-Columbia RFLP Laboratory. Availability is listed in the Maize Genome Database (http://www.agron.missouri.edu).

Bold numbers in boxes to the left of the map are *bin numbers* for each chromosome. The *core marker* is the first marker for the bin below it. Numbers to the far left of the map at the end of the extended lines represent map positions (cumulative distances from the most distal marker on the short arm) to each core marker. Numbers to the left of the map next to the line represent interval distances between adjacent markers. Interval distances are shown to aid in interpretation of map order certainty, as described below.

Core marker choice: Core markers were chosen according to the following criteria. Markers that had simple fragment patterns and were distributed along the chromosome every 20 to 30 cM were selected as potential core markers. In addition, markers that would be more amenable to sequencing (less than 1200 bp in size) were chosen where possible. Markers that were not among the previous set identified by Gardiner et al., 1993, were screened against A619, A632, B73, Mo17, CO159, and Tx303 using *Eco*RI, *Hin*dIII, *Eco*RV, *Bam*HI, *Dra*I, *Xba*I, *Bgl*II, and *Sst*I to determine whether they were polymorphic enough to be designated as core markers. Several substitutions were made due to low levels of polymorphism or high fragment pattern complexity. Core marker sequences will be made available as soon as possible, through the Maize Genome Database and in published form. An information table for the Core Probe Set follows the Gene List.

Methods and RFLP Map Construction: The mapping population consists of 54 immortalized F2 individuals from a cross of Tx303 x CO159. Hybridizations and washing procedures were conducted according to the protocols given in the University of Missouri RFLP Laboratory Manual (copies are available by writing the UMC RFLP Lab or by request from musket@teosinte.agron.missouri.edu). All hybridizations were carried out using ^{32}P oligolabelled probes. All probes were screened on CO159 and Tx303 using *Eco*RI, *Hin*dIII, *Eco*RV, *Bam*HI, *Dra*I, *Xba*I, *Bgl*II, and *Sst*I. The enzyme with the best fragment separation between the two lines was chosen for mapping. If multiple copies were evident more than one enzyme was used to map where necessary. All autoradiograms were scored independently by two readers, who required concurrence on clarity of the readings and on the scores themselves. Readings with more than 3 missing data points were discarded. Linkage groups were constructed using MAPMAKER for MacIntosh, Version 2 on a Centris 660AV.

The marker data for 9 of the 10 chromosomes were analyzed by two-point analysis using the *group* function at LOD 5, theta 0.38. Markers for chromosome 4 were analyzed at LOD 4, theta 0.45. Initial marker order for each chromosome was determined using the *first order* command. The marker order for the first order maps was examined by the *ripple* procedure. Suggested changes in marker order indicated by the *ripple* analysis with improved map likelihood scores were examined by using the *compare* command on four or five markers to include the nearest marker flanking *ripple*-identified marker triplets. The marker order from *compare* analysis with the best total map likelihood score was set for each region. Once all the *compare* analyses were completed the marker order for each chromosome was rechecked by repeating the *ripple* analysis.

Two different analyses were conducted to examine marker placement and distance between markers. The *drop marker* analysis was performed to identify markers causing localized distortions in marker distance. Autoradiograms were reexamined for markers where the *drop marker* analysis indicated deletion of that marker would result in shortening the chromosome by 12 cM or greater. This analysis identified three markers that were removed from the map. In addition to the drop marker analysis, the mapscores and marker orders were loaded into MAPMAKER for DOS, Version 3, and double crossovers were identified with the *genotypes* routine. Autoradiograms were examined for markers

with two or more double crossovers within a short interval. This analysis resulted in modified scores for three markers with mis-entered data. Loci that MAPMAKER indicated were separated by distances of 0.0 or 0.1 cM were due to missing data points and their positions were merged to a single position. Individual chromosomes were redrawn using MacDraw II.

Map order certainty:

Distances of <0.9 are the result of missing data and adjacent marker orders may be reversed.

Distances of 0.9 are the result of one recombinant individual between adjacent markers. Relative order is not defendable at LOD 3.

Distances between 0.9 and 1.8 are generally not defendable at LOD 3.

All distances that are exact multiples of 0.9 represent incremental changes in the number of cross-overs between adjacent markers. Any distance that falls between these values is the result of missing data and/or presence/absence scoring of a marker.

Marker orders with adjacent marker distances between 0.9 and 5.0 cM located near "gaps" are often not defendable at LOD 3.

The **Genetic Working Map** accompanying the RFLP map is a "next-generation map"; it has been totally reviewed and redrawn as follows: Using the framework of the RFLP Map, genes placed on that map are placed on the parallel lines, at the coordinate locations defined in the RFLP Map. Genes on the current Brookhaven National Laboratory map (presented in succeeding pages), and genes mapped with RFLP markers in other experimental populations are placed by relations with markers in common or are approximated in map position by relating markers from maps to maps where necessary. *Coordinates marked with asterisk (*)* are defined in this fashion. Other genes are placed, insofar as possible, on the map line by review of past data, from 1935 to date. The *bins* in the Genelist are defined from map location.

Please note that distances are NOT consistently adjusted to Haldane centiMorgans in the maps - these are *Working Maps* designed to be refined and revised with new data and better methods, including adjustments with mapping functions and yet-unavailable analytical tools.

The contributions of Mary Berlyn and Stan Letovsky in advance-testing analytical tools for mapping, which have contributed to chromosome-1 mapping decisions directly and have helped set criteria for intelligent decisions with all of the chromosomes, are appreciated.

Ed Coe, Georgia Davis, Mike McMullen, Theresa Musket 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	176
a2	5.04	anthocyaninless2, like *a1*, but red pericarp with *P1-RR*, encodes *A2*	297
a3	3.08	anthocyanin3, recessive intensifier of expression of *R1* and *B1* in plant tissue, encodes a3-product	371
aaa1		adenosylmethionine aminotransferase1, endosperm cDNA 5C01E09 (*uaz113*) similar to bacterial biotin synthesis enzyme, may encode S-adenosylmethionine--8-amino-7-oxononanoate transaminase	264
aap1		acylaminoacyl-peptidase1, endosperm cDNA 5C09A06, similar to acylaminoacyl-peptidase, may encode acylaminoacyl-peptidase	264
abp1	3.05	auxin binding protein1, putative auxin receptor, single band in Southerns, endoplasmic reticulum and plasma membrane locales (aka *axr1*, *aux311*), encodes auxin binding protein	691
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, ABP4	610
abp5		auxin binding protein homolog5, genomic sequence, promoter-reporter gene fusion functional in maize protoplasts, may encode auxin binding protein, ABP5	610
abt1		ATP-binding transport protein homolog1, endosperm cDNA 5C04B07 (*uaz230*), similar to membrane carrier proteins, may encode membrane permease	263
Ac		Activator, autonomous transposable element, regulates *Ds* transposition and dissociation; *Ac9* is element isolated from *wx1-m9*, encodes TPASE, putative *Ac* transposase	63, 411
Ac2		Activator2, similar to *Ac*, but one dose engenders no excisions, and higher doses show exponential increases	146, 148
acc1	10.04	acetyl-coenzyme A carboxylase1, tissue-culture selected resistance to cyclohexanedione (e.g., sethoxydim) and aryloxy phenoxypropionate (e.g., haloxyfop) herbicides (probed site *accB* on chromosome 10 may correspond), encodes acetyl-coenzyme A carboxylase I	513
aco1	4.04	aconitase1, electrophoretic mobility; monomeric, encodes aconitate hydratase	718
aco2		aconitase2, electrophoretic mobility, encodes aconitate hydratase	718
aco3		aconitase3, electrophoretic mobility, encodes aconitate hydratase	718
aco4		aconitase4, electrophoretic mobility; monomeric, encodes aconitate hydratase	718
acp1	9.03	acid phosphatase1, electrophoretic mobility; cytosolic; dimeric, encodes acid phosphatase	167, 168, 236
acp2		acid phosphatase2, electrophoretic mobility; dimeric, encodes acid phosphatase	168, 236, 315
acp4	1.11	acid phosphatase4, electrophoretic mobility; monomeric, encodes acid phosphatase	315
acpt1		acyl carrier protein1, acyl carrier protein (acp) cDNA, encodes 121 aa polypeptide, contains transit peptide sequence, encodes acyl carrier protein	647
ad1	1.08	adherent1, seedling leaves, tassel branches, and occasionally top leaves adhere	325
ad2		adherent2, upper leaves and tassel tend to adhere and fuse; seedling and juvenile stages normal	487
adh1	1.10	alcohol dehydrogenase1, electrophoretic mobility; null alleles are known; dimeric; intra/interlocus hybrid bands occur), encodes alcohol dehydrogenase	602, 608

adh2	4.03		alcohol dehydrogenase2, electrophoretic mobility; null alleles are known; dimeric; intra/interlocus hybrid bands occur, encodes alcohol dehydrogenase	608
adk1	6.00		adenylate kinase1, electrophoretic mobility; plastidial, encodes adenylate kinase	723
adr1			alcohol dehydrogenase regulator1, recessive (in strain R6-67) sustains higher levels of scutellar ADH vs. usual decline (in W64A) during germination	344
ae1	5.05		amylose extender1, glassy, tarnished endosperm; high amylose content; application: highly linear starch is used for food films, fibers, and other industrial purposes, encodes 1,4-alpha-glucan branching enzyme	703
afd1	6.01	6.08	absence of first division1, male and female sterility; failure of synapsis, anaphase I equatorial	227
agp1	6.07	6.08	ADP glucose pyrophosphorylase1, leaf or embryo cDNA similar to *bt2* sequence, but distinct 3'-noncoding sequence and Southern blot pattern, encodes ADP glucose pyrophosphosphorylase, leaf	534
agp2	2.06		ADP glucose pyrophosphorylase2, embryo cDNA that hybridizes to endosperm transcripts, distinct from *sh2* and *bt2*, encodes ADP glucose pyrophosphosphorylase, leaf	222
agt1			agravitropic1, primary root unresponsive to gravity	160
ahh1			adenosyl homocysteine hydrolase1, endosperm cDNA 5C01H11 (*uaz145*) similar to plant activated methyl cycle enzyme, encodes S-adenosyl-L-homocysteine hydrolase	264
aip1			auxin induced protein homolog1, leaf cDNA *csu190*, similar to auxin-induced protein, may encode auxin-induced protein	31
akh1	4.05		aspartate kinase-homoserine dehydrogenase1, cDNA 77% homologous to carrot threonine-sensitive AK-HSDH bifunctional enzyme, encodes aspartate kinase homoserine dehydrogenase	445
akh2	2.06		aspartate kinase homoserine dehydrogenase2, cDNA sequence 75% homologous to carrot threonine sensitive AK-HSDH bifunctional enzyme, encodes aspartate kinase homoserine dehydrogenase	445
al1	2.01		albescent plant1, variably cross-banded to white leaves, pale yellow endosperm, some alleles viviparous; *y3* is allelic	527
ald1	8.06		aldolase1, cytosolic aldolase; cDNA and genomic clones; Southern blots give single or double band; promoter functional in transient expression assay, encodes aldolase	252, 322
alh1	1.11		histone la, (was H1a); electrophoretic mobility, encodes histone la	675
alp1			aluminum-induced protein homolog1, leaf cDNA *csu359* similar to wheat aluminum-induced protein, GenBank accession L28008, may encode aluminum-induced protein	31
alpha			*a1* locus component (see beta), determines reduced aleurone and plant color, brown pericarp	354
als1	4.05		acetolactate synthase1, sensitive to imidazolinone herbicides; acetohydroxyacid synthase has altered herbicide inhibition kinetics, encodes acetohydroxyacid synthase	14, 490
als2	5.03	5.04	acetolactate synthase2, sensitive to imidazolinone herbicides; acetohydroxyacid synthase has altered herbicide inhibition kinetics, encodes acetohydroxyacid synthase	14, 490
alt1			L-alanine:2-oxoglutarate aminotransferase1, electrophoretic mobility; *alt1* and *alt2* interact to form heterodimers, encodes L-alanine:2-oxoglutarate aminotransferase	711
alt2			L-alanine:2-oxoglutarate aminotransferase2, electrophoretic mobility; *alt1* and *alt2* interact to form heterodimers, encodes L-alanine:2-oxoglutarate aminotransferase	711
alt3			L-alanine:2-oxoglutarate aminotransferase3, electrophoretic mobility, encodes L-alanine:2-oxoglutarate aminotransferase	711
am1	5.03		ameiotic1, male and female sterility - anaphase I equatorial; alleles *am1-pra1*(was *pra1*), *am1-2* (was *am2*)	508, 559
amp1	1.07		aminopeptidase1, electrophoretic mobility; cytosolic; monomeric, encodes aminopeptidase	503
amp2	1.06	1.07	aminopeptidase2, electrophoretic mobility; monomeric, encodes aminopeptidase	503
amp3	5.04		aminopeptidase3, electrophoretic mobility; monomeric, encodes aminopeptidase	503
amp4			aminopeptidase4, electrophoretic mobility; monomeric, encodes aminopeptidase	503
amy1			alpha amylase1, electrophoretic mobility; monomeric; possibly identical to *amy3*, encodes alpha amylase	102
amy2	5.03	5.04	beta amylase2, electrophoretic mobility; monomeric, encodes beta amylase	101
amy3	2.07		alpha amylase L25805, aleurone cDNA, 94% similarity to rice alpha amylase; possibly identical to *amy1*, encodes alpha amylase	744
an1	1.08		anther ear1, andromonoecious dwarf, intermediate stature; few tassel branches; responds to gibberellins; *an1-6923* includes deletion of *Bz2+*, encodes AN1	170, 173
anl1	5.03		anthocyaninless lethal1, Colorless aleurone; small kernels; embryo inviable	120
ans1			anthranilate synthase homolog1, leaf cDNA csu65 sequence similar to yeast TRP3 gene, may encode anthranilate synthase	321
ant1			adenine nucleotide translocator1, leaf cDNA, open reading frame encodes 40,519 Da polypeptide; single site (5L, MNL 67) contradicted by two sites probed in Tropical Maize F2's by p-csuh26, may encode adenine nucleotide translocator, mitochondrial	22
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	27
aph1			aphid resistance1, recessive resistance	55, 99

app1			abnormal phragmoplast formation1, phragmoplasts in microsporocytes abnormal, cytokinesis disorganized (aka *aph1*)	662
ar1	9.03		argentia1, virescent seedling, greens rapidly; husk leaf tips striped	186
ars1			autonomously replicating sequence1, replicates in yeast; 11,000 copies in maize	47
ars2			autonomously replicating sequence2, replicates in yeast; 10,000 copies in maize	47
ars3			autonomously replicating sequence3, replicates in yeast; 28,000 copies in maize	47
as1	1.05		asynaptic1, synaptic failure in male and female	32
ask1	7.01	7.02	aspartate kinase1, lysine-threonine resistance in cultures and seedlings, increased threonine in kernels, altered kinetics of aspartate kinase, encodes aspartate kinase	151
ask2	2.06		aspartate kinase2, lysine-threonine resistance, encodes aspartate kinase	151
asp1			absence of meiotic spindle1, meiosis normal up to diakinesis; spindle absent, telophases contain 3-10 nuclei	684
asr1	4.01		absence of seminal roots1, dominant *Asr1* seedlings lack seminal roots	434
atn1			anaerobic tolerant null1, enhances survival of ADH-null under anoxia	357
atp1	3.05		ATPase1, leaf cDNA csu30 is identical to *Avena sativa* vacuolar ATPase subunit, encodes proteolipid, vacuolar ATPase	321
atp2			ATP synthase2, cDNA clone, encodes ATP synthase beta chain, mitochondrial	732
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	267
ats1	8.00	8.09	atrazine susceptible1, lacks glutathione S-transferase	246
B chromosome			supernumerary chromosome; occurs naturally in many maize and teosinte populations	545
B-A translocation			interchange between a B chromosome and a member of the basic (A) set of chromosomes	577
b1	2.03		colored plant1, dominant *B1* plants have anthocyanin in major plant tissues; some alleles affect aleurone and embryo color; regulates flavonoid enzymes, encodes myc protein of *B1*	179
ba1	3.06		barren stalk1, ear shoots and most tassel branches and spikelets absent	268
ba2	2.04	2.05	barren stalk2, like *ba1*, but tassel with bundled branches having peduncles fused to the culm at the base	268
ba3			barren stalk3, no ear produced	510, 511
baf1	9.02		barren stalk fastigiate1, ear shoots often absent, or distorted and fused to culm at base; tassel branches erect, bundled, fused to culm at base	116
bcl1			B cell lymphoma homolog1, leaf cDNA csu27, similar to human lymphoma protein, may encode cell cycle protein CDC10	30
bd1	7.06		branched silkless1, ear silkless, branched at base; tassel proliferated, bushy	328
ben1			bentazon resistance1, dominant resistance	197
bet1	3.04		glycinebetaine1, salt tolerance, glycinebetaine levels, recessive bet1 blocks choline oxidation, may encode choline monooxygenase	360
beta			*a1* locus component (see alpha), determines aleurone and plant color, red pericarp	354
bf1	9.07		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	688
bf2	10.04		blue fluorescent2, similar to *Bf1* in expression; shows earlier, stronger seedling fluorescence	11
Bg			Bergamo, transposable element; regulatory element mediating *o2-mr*	587
bif1	8.02	8.03	barren inflorescence1, dominant *Bif1* plants have ear and tassel with many fewer spikelets, bare rachis appendages	465
bif2	3.05	3.09	barren inflorescence2, recessive few-branched tassel, normally no pollen shed; variable expression on ear with 0-2 spikelets produced at each floral node	59
bip1			BiP homolog1, cDNA clone, protein body, putative molecular chaperone of hsp70 family, encodes maize BiP	53
bk2	9.04	9.05	brittle stalk2, brittle plant parts after 4-leaf stage	351
blh1	1.02	1.05	bleached1, dominant *Blh1* plants have pale green midveins and base in upper leaves	479
bm1	5.04		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	185
bm2	1.11		brown midrib2, like *bm1*	78
bm3	4.04	4.05	brown midrib3, like *bm1*; has lowered activity of catechol O-methyl transferase; application: modified lignin has improved digestibility in ruminants; silage corn with *bm3* is in production. However, *bm3* results in weaker stalks, reduced stover and grain yield, and in some cases reduced maturity; these effects appear genotype-specific., encodes caffeate O-methyltransferase	79, 171, 338
bm4	9.07	9.08	brown midrib4, like *bm1*	81
bn1	7.03		brown aleurone1, yellowish brown aleurone color	342
br1	1.07		brachytic1, short internodes, short plant; no response to gibberellins	324, 327
br2	1.06		brachytic2, like *br1*; *mi1* allelic, and preceded (1935), but *br2* retained due to long usage	359
br3	5.00	5.09	brachytic3, like *br1*	635

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 (aka *sbe1*), may encode starch branching enzyme I	19
brn1	3.01		brown aleurone1, brown kernel, brown embryo; seedling lethal	573
Bs-1			Barley stripe mosaic virus, transposable element, retrovirus-like; 1-5 copies in genome, internal sequence has fragments similar to plasma membrane [H+]-ATPase, encodes *BS1*, putative ORF1 product	305
bs1			barren sterile1, Plant weak, with little or no tassel and usually with only a vestige of pistillate inflorescence, shank, husks	425, 733
bsd1			bundle sheath defective1, pale green seedling lethal, reduced Rubisco and NADP-malic enzyme, small bundle sheath plastids	349
bt1	5.04		brittle endosperm1, mature kernel collapsed, angular, often translucent and brittle (alleles *sh3*, *sh5*), may encode amyloplast adenylate translocator	390, 724
bt2	4.04		brittle endosperm2, like *bt1*; endosperm ADPG pyrophosphorylase subunit (compare *sh2*), encodes ADP glucose pyrophosphorylase, 55 kDa subunit	171, 689
btn1			brittle node1, tassel breakage in B73 inbred line	317
bu1			leaf burn1, leaves show burning, sometimes horizontal bands, accentuated by high temperature	209
bv1	5.04	5.05	brevis plant1, short internodes, short plant	362
bv2			brevis plant2, plant height 50-70% of normal; possible allelism with *rd1*	528
bvp1			bovine virus protein homolog1, endosperm cDNA 5C04D07 (*uaz207*), similar to a bovine virus protein, may encode transcription factor	263
bvp2			bovine virus protein homolog2, endosperm cDNA 5C04F07, similar to bovine virus glycoprotein, may encode glycoprotein	264
bx1	4.01		benzoxazinless1, cyclic hydroxamates (blue color in etiolated c oleoptile or crushed root tip with FeCl3), which inhibit *Ostrinia nubilalis* and *Helminthosporium turcicum*, present in *Bx1* roots, absent in *bx1*	132
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	558
bz2	1.08		bronze2, like *bz1*; anthers not fluorescent; *an1-6923* mutation includes deletion for *Bz2*; potential function flavonoid acylation, glycosylation, transport, or deposition, encodes BZ2 product	498, 499
c1	9.01		colored aleurone1, *C1* colored; *C1-I* dominant colorless; *c1* colorless, includes *c1-p* (pigment inducible by light at germination), *c1-n* (not inducible), encodes myb protein of *C1*	165
c2	4.08		colorless2, colorless aleurone, reduced plant and cob color; *C2-Idf* dominant inhibitor; duplicate factor with whp1 for pollen color and for anthocyanins, encodes chalcone synthase	62
cah1			carbonic anhydrase homolog, leaf cDNA *csu125* sequence similar to pea carbonic anhydrase, may encode carbonic anhydrase	321
cal1			calmodulin homolog1, cDNA sequence identical to plant calmodulin consensus sequence, encodes calmodulin	244, 245
car1	1.00	1.05	catalase regulator1, dominant *Car1* determines increased enzyme activity level, encodes CAR1 product	594
cat1	5.03		catalase1, electrophoretic mobility; cytosolic/glyoxysomal; tetrameric; intra/interlocus hybrid bands occur, encodes catalase	42, 606
cat2	1.01		catalase2, electrophoretic mobility; null allele is known; cytosolic/glyoxysomal; tetrameric; intra/interlocus hybrid bands occur, encodes catalase	591
cat3	4.10		catalase3, electrophoretic mobility; null allele is known; mitochondrial; tetrameric; intralocus hybrid bands occur, encodes catalase	593
cbp1			calmodulin binding protein1, partial cDNA clone from a root tip expression library; fusion protein binds calmodulin; 1-2 copies estimated, encodes calmodulin binding protein	548
cdc2			cell division control protein2 homolog, cDNA sequence homologous to cdc2/cdc28 subfamily of serine/threonine protein kinases, may encode serine/threonine protein kinase	121
cdc48	6.02		cell division protein48 homolog, single map site, leaf cDNA *csu146*, 5'-sequence similar to an *E. coli* cell division protein, encodes cell cycle protein CDC48	29
cdh1			cinnamyl alcohol dehydrogenase1, electrophoretic mobility, encodes cinnamyl alcohol dehydrogenase	203
cdj1			chaperone DNA J homolog1, leaf cDNA csu63, deduced amino acid sequence nearly identical to chaperone DNA J, multiple copies, may encode chaperone DNA J	29
cdpk1			calcium dependent protein kinase1, endosperm cDNA 5C04G11, similar to plant calcium dependent protein kinases, may encode calcium dependent protein kinase	264
ce1			curled entangled1, dominant *Ce1* plants have rolled leaves that tend to be entangled	108, 514
cfl2			complementary to *fl2*, recessive female gives floury with *fl2* pollen; heterozygous female gives normal phenotype	505, 506
cfr1	1.00	1.05	coupling factor reduction1, chloroplast ATP synthase affected; seedlings pale green and greatly reduced in vigor	166
cg1	3.02		corngrass1, semidominant *Cg1* plants have narrow leaves, extreme tillering	634
cg2	3.02		corngrass2, dominant *Cg2* plants have narrow leaves, high tillering; mutable	382
cgl1			*Colletotrichum graminicola* resistance1, dominant *Cgl1* plants are resistant	21

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	2.09		chocolate pericarp1, dominant *Ch1* ears have tan to dark brown pericarp and cob	8
chi1	1.11		chalcone flavanone isomerase1, genomic sequence, gene-specific probe, transcript defined by S1-nuclease protection analysis, encodes chalcone flavanone isomerase	247
chs1			chitin synthase homolog1, endosperm cDNA 5C04C09, similar to chitin synthase of Candida albicans, may encode chitin synthase	264
cif1			cross-incompatibility(female)1, cross-incompatibility when homozygous *cif1* female is crossed with male homozygous recessive for *cim1* and *cim2*	546
cim1			cross-incompatibility(male)1, reduced seed set when male parent is homozygous recessive *cim1* and *cim2* and female parent is homozygous recessive *cif1*	546
cim2			cross-incompatibility(male)2, reduced seed set when male parent is homozygous recessive *cim1* and *cim2* and female parent is homozygous recessive *cif1*	546
Cin			Cinteotl, repetitive sequences dispersed in the genome	248
ck2			casein kinase2, partial cDNA has three regions of identity to all other known casein kinase 2 alpha subunit genes, encodes casein kinase	152
cl1	3.04		chlorophyll1, white to green seedlings, depending upon alleles of modifier *clm1*; pale yellow endosperm	183
cld1			cold regulated protein homolog1, leaf cDNA csu19, similar to barley cold-regulated protein, may encode cold-regulated protein	29
clh1			histone lc1, electrophoretic mobility, encodes histone lc	675
clm1	8.00	8.09	modifier of *cl1*, dominant *Clm1* alleles confer greening in *cl1* seedlings but do not restore endosperm carotenoids	183
clp1			CLP protease1, endosperm cDNA 5C02D08 similar to chloroplast Clp ATP-dependent protease, encodes Clp ATP-dependent protease, chloroplast	263
clt1	8.04		clumped tassel1, dominant *Clt1* plants have variable dwarfing, developmental anomalies	219, 461
clx1			calnexin homolog1, low copy number, leaf cDNA *csu148*, similar to *Arabidopsis* calnexin, may encode calnexin	29
cm1	10.06		chloroplast modifier1, white or yellow stripes on leaves (compare *ij1*); conditions chloroplast modifications that are maternally inherited	677
cms-C			cytoplasmic male sterility, female-transmitted male sterility, C type; restored by *Rf4*	40
cms-S			cytoplasmic male sterility, female-transmitted male sterility, S type; restored by *Rf3*	310, 313
cms-T			cytoplasmic male sterility, female-transmitted male sterility, Texas type; restored by *Rf1 Rf2*	310, 313
cms-Y			cytoplasmic male sterility, female-transmitted male sterility, Y type; partially restored by *Rf7*	134
cp1	7.02		collapsed1, endosperm collapsed and partially defective	378, 379
cp2	4.04		crumpled2, shrunken sugary endosperm; white seedling with green stripes (aka *dek7*, *o12*)	466
cp3	1.05	1.06	collapsed kernel3, variably collapsed floury non-pigmented nonviable kernel; double mutant combination with *mn4* has orange pericarp	618
cps1	1.06	1.12	chloroplast protein synthesis1, reduced levels of RUBISCO and all thylakoid membrane complexes; unaltered chloroplast mRNA; decreased chloroplast polysomes	26
cps2	6.01	6.08	chloroplast protein synthesis2, 20-fold reduced RUBISCO, 2-fold reduced thylakoid polypeptides, decreased chloroplast polysomes (aka *hcf133*)	25
cr1	3.02		crinkly leaves1, plant short; leaves broad, crinkled, foreshortened	180
cr4	10.01	10.02	crinkly leaf4, crinkly seedling leaves; plants short with rough, extremely crinkly leaves and club tassel; aleurone mosaic	668, 671
crp1	7.02	7.06	chloroplast RNA processing1, fails to accumulate monocistronic petB and petD mRNA's; lacks cytochrome f/b6 and photosystem I polypeptides (aka *hcf111*, *hcf136*)	25, 127
crp2	5.00	5.04	chloroplast RNA processing2, fails to degrade group II introns in chloroplast (aka *hcf143*); *Mu*-induced	25
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*	335
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*	335
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*	335
csp1			white spot1, tiny to medium elliptical, nearly transparent spots, scattered on leaf blade beginning at 8-leaf stage	332
ct1	8.01	8.02	compact plant1, semi-dwarf plant, ear furcated	451
ct2	1.01	1.05	compact plant2, semi-dwarf plant with club tassel	224
cta1			chitinase *a1*, cDNA sequence corresponds to peptide sequence of maize 28kDa chitinase A, encodes chitinase	287

ctb1			chitinase *B1*, cDNA sequence corresponds to protein sequence of maize chitinase B, encodes chitinase	287
ctn1			caltractin1, endosperm cDNA 5C06B08, similar to algal caltractin, encodes caltractin	264
cto1			cob turned out1, ear inverted to a sheet or tube, kernels internally placed; variable expression	589, 699
cx1	10.04		catechol oxidase1, electrophoretic mobility; null allele is known; monomeric; no hybrid bands, encodes catechol oxidase	536, 537
Cy			Cycler, regulatory element mediating *bz1-rcy*	601
cyc1			cyclin1, classI; sense RNA functions in *Xenopus* oocytes, encodes cyclin	551
cyc2			cyclin2, class II; sense RNA functions in *Xenopus* oocytes, encodes cyclin	551
cyc3			cyclin3, classIII; sense RNA functions in *Xenopus* oocytes, encodes cyclin	551
cyc4			cyclin4, Class I; sense RNA functions in *Xenopus* oocytes, encodes cyclin	551
cyp1			cytochrome P450 homolog1, leaf cDNA csu25, similar to rat cytochrome P450, may encode cytochrome P450	29
d1	3.02		dwarf plant1, andromonoecious, short, compact plants; responds to gibberellins; *d1-t* intermediate in height, encodes D1	173
d2			dwarf plant2, like *d1*, encodes D2	680
d3	9.03		dwarf plant3, like *d1*, encodes D3 P450 heme thiolate protein	143
d5	2.02		dwarf plant5, like *d1*, encodes D5	680
d8	1.10		dwarf plant8, dominant *D8* plants resemble *d1*; not responsive to gibberellins; (compare *Mpl1*, probable allele), encodes D8	525
d9	5.02		dwarf plant9, dominant *D9* plants semidwarf with broad, dark green leaves; not andromonoecious, not responsive to gibberellins	481, 483
d10	2.08	2.09	dwarf plant10, From EMS pollen treatment, *D10* is a tiny dwarf with fair tassel; tillers with tassel seed expression; not andromonoecious; leaves proportionally normal and stalk slender; shortened internodes	489
da1	9.00	9.08	dilute aleurone1, aleurone color diluted	190
dap1	5.08		dappled aleurone1, dominant *Dap1* kernels show patches of normal and abnormal aleurone cells; effect with colored aleurone is conspicuous	668
dar1			monodehydroascorbate reductase homolog1, leaf cDNA *csu196* sequence similar to cucumber monodehydroascorbate reductase (NADH), may encode monodehydroascorbate reductase (NADH)	31
Def			deficiency, general symbol for a loss of a chromosome segment	407
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*	638
dek1	1.03		defective kernel1, germless; colorless (mosaic) aleurone; floury white endosperm; anthocyanins and carotenoids absent; cultured embryos not obtained (aka *clf1*, *gay1*)	466, 467, 470
dek2	1.06	1.12	defective kernel2, discolored, scarred endosperm; lethal; cultured embryos green	466, 467, 470
dek3	2.00	2.04	defective kernel3, germless; cultured embryos white with green stripe	466, 467, 470
dek4	2.05	2.10	defective kernel4, germless; floury endosperm; cultured embryos green, narrow leaved	466, 467
dek5	3.00	3.04	defective kernel5, shrunken endosperm; white seedling with green stripes	466, 467, 618
dek6	3.07		defective kernel6, shrunken endosperm; lethal; cultured embryos normal	466, 467, 470
dek8	4.06	4.11	defective kernel8, shrunken endosperm; lethal; cultured embryos green, small	466, 467, 618
dek9	5.05	5.09	defective kernel9, crumpled endosperm; lethal; anthocyanins and carotenoids reduced; cultured embryos not obtained	466, 467
dek10	4.06	4.11	defective kernel10, collapsed endosperm; lethal; cultured embryos green, curled, stubby	466, 467, 618
dek11	4.00	4.05	defective kernel11, etched endosperm; lethal; cultured embryos white with green stripes	466, 467, 618
dek12	9.00	9.03	defective kernel12, collapsed endosperm; lethal; cultured embryos green, narrow-leaved, curled	466, 467, 470
dek13	9.03	9.08	defective kernel13, defective opaque endosperm; lethal; cultured embryos pale green with green stripes	466, 467, 618
dek14	10.00	10.03	defective kernel14, collapsed endosperm; lethal; cultured embryos yellow-green	466, 467, 618
dek15	10.04	10.07	defective kernel15, collapsed floury endosperm; lethal; cultured embryos green	466, 467, 618
dek16	2.05	2.10	defective kernel16, small, floury, nonviable kernel; cultured immature embryos give slender green plants	466, 619
dek17	3.05	3.09	defective kernel17, collapsed endosperm; lethal; cultured embryos not obtained	470, 619
dek18	5.00	5.04	defective kernel18, collapsed endosperm; lethal; cultured embryos green, narrow-leaved	618, 619
dek19	6.01	6.08	defective kernel19, collapsed opaque endosperm; lethal; cultured embryos green	470, 619
dek20	8.04	8.09	defective kernel20, collapsed endosperm; lethal; cultured embryos green	618, 619
dek22	1.06	1.12	defective kernel22, collapsed endosperm; lethal; cultured embryos not obtained	113, 618, 620
dek23	2.05	2.10	defective kernel23, defective crown; lethal; cultured embryos not obtained	113, 466, 620
dek24	3.00	3.04	defective kernel24, collapsed endosperm; lethal; cultured embryos normal	470, 620
dek25	4.02		defective kernel25, shrunken endosperm; lethal; cultured embryos normal	618, 620
dek26	5.05	5.09	defective kernel26, collapsed endosperm; lethal; cultured embryos normal	470, 620
dek27	5.05	5.09	defective kernel27, collapsed endosperm; lethal; cultured embryos green	618, 620
dek28	6.00	6.01	defective kernel28, opaque endosperm	620

dek29	8.04	8.09	defective kernel29, collapsed endosperm; viable; cultured embryos green, narrow-leaved	618, 620
dek30	9.03	9.08	defective kernel30, floury endosperm; lethal; cultured embryos green, narrow-leaved	620
dek31	4.07		defective kernel31, pitted endosperm; lethal	621
dek32	1.00	1.05	defective kernel32, large shrunken floury nonviable kernel	485
dek33	5.04	5.05	defective kernel33, opaque, floury, dented, wrinkled kernel with floury endosperm; occasionally viviparous	470, 485
dep1	6.00	6.08	defective pistils1, female florets have abnormal structure; the ovaries form two or more short defective pistils that do not function	422
des17	8.04	8.09	defective seedling17, reduced height, partial suppression of primary root growth, contorted leaves, lethal	207
dHbr			defective Heartbreaker, element similar to Tourist	303
dhn1	6.05		responsive to abscisic acid17, protein induced by abscisic acid; cDNA sequence agrees with amino acid sequence (aka *dhn3*, *rab17*, *rny1*), encodes dehydrin, DHN1	115
dia1	2.06		diaphorase1, electrophoretic mobility; cytosolic; monomeric, encodes dihydrolipoamide dehydrogenase	718
dia2	1.11		diaphorase 2, electrophoretic mobility; cytosolic; dimeric, encodes dihydrolipoamide dehydrogenase	718
dib1			dichotomously branched1, main axis branches into two normal tops, most often at node 4-8 but variable; associated with aneuploidy	423, 424, 425
dlf1			delayed flowering1, tall late plant with additional nodes and leaves at flowering; no apparent response to day length	488
dp1	4.09		distal pale1, seedling leaf tip virescent	13
dps1			dihydrodipicolinate synthase1, cDNA complements *E. coli* mutant (AT997dapA-) blocked in DHPS (aka *dhp1*), encodes dihydrodipicolinate synthase	206
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*	410, 411
Ds-r			*Ds* element equipped with bacterial plasmid sequences to permit rescue from the plant genome	578
dsc1	4.00	4.05	discolored kernel1, crumpled, discolored, germless lethal	290, 597
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.	410
dSpm			defective *Spm*, designator for non-autonomous transposable elements that respond to *Spm*	599
dsy1			desynaptic1, male and female sterility; synapsis and chiasmata occur, followed by separation after pachytene	228
dsy2			desynaptic2, like *dsy1*	226
dsy3			desynaptic3, like *dsy1*	232
dsy4			desynaptic4, like *dsy1*	232
Dt1	9.00		Dotted1, regulates controlling element rDt at *A1*; responding *a1-m* alleles express colored dots on colorless kernels and purple sectors on brown plants, encodes Dt transposase	554
Dt2	6.04		Dotted2, like *Dt1*	500
Dt3	7.05	7.06	Dotted3, like *Dt1*, but expression variable	500
Dt4	4.00	4.11	Dotted4, like *Dt1*, but dots chiefly on crown of kernel	155
Dt5	9.00		Dotted5, like *Dt1*	155
Dt6	4.05		Dotted6, like *Dt1*	654
dts1			aspartyl-tRNA synthetase1, endosperm cDNA 5C01B12 (*uaz131*), similar to rat aspartyl-tRNA synthetase subunit, encodes aspartyl-tRNA synthetase, alpha-2 subunit	264
du1	10.04		dull endosperm1, glassy, tarnished endosperm; affects soluble starch synthase and branching enzyme IIa	171, 391
dv1			divergent spindle1, chromosomes unoriented at metaphase I; partial male and female sterility	111, 112
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	450
dzr1	4.02		zein-protein regulator, elevation of 10kD zein	46, 105
dzs10	9.03		delta zein10, [was *Zps10/(22)*]; high (22.5%) methionine 10kD zein; RFLP (probe 10kZ-1), encodes 10-kDa zein (delta zein)	46
dzs23	6.04		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	681
e1	7.04		esterase1, electrophoretic mobility; null allele is known; dimeric; intralocus hybrid bands occur, encodes esterase	606
e2			esterase 2, presence-absence only, encodes esterase	603
e3	3.05	3.09	esterase3, electrophoretic mobility; dimeric; intralocus hybrid bands occur, encodes esterase	607
e4	3.04		esterase4, electrophoretic mobility; null allele is known; monomeric, encodes esterase	257
e5(I)			esterase, electrophoretic mobility, duplicate factor with E5(II), encodes esterase	385
e5(II)			esterase, electrophoretic mobility; duplicate factor with E5(I), encodes esterase	385
e6			esterase6, presence-absence only, encodes esterase	385

e7			esterase7, presence-absence only, encodes esterase	385
e8	3.01		esterase8, electrophoretic mobility; null allele is known; dimeric; intralocus hybrid bands occur, encodes esterase	385
e9			esterase9, electrophoretic mobility; null allele is known, encodes esterase	385
e10			esterase 10, electrophoretic mobility, encodes esterase	385
ech1			enoyl-CoA hydratase1, leaf cDNA 6C06C04 (*uaz348*) similar to animal fatty acid beta-oxidation enzyme, may encode enoyl-CoA hydratase	264
eg1	5.06	5.08	expanded glumes1, glumes open at right angle	82
eif5			elongation initiation factor 5, leaf cDNA *csu154*, similar to yeast and rat translation initiation factor, may encode elongation initiation factor 5	29
el1	8.04	8.09	elongate1, chromosomes uncoiled during meiotic metaphase and anaphase in male and female; frequent unreduced gametes	559
elf1			elongation factor1, multiple copies; leaf cDNA *csu116*, partial sequence identical to a tomato elongation factor, encodes elongation factor 1-alpha subunit	321, 561
elf2			elongation factor2, endosperm cDNA 5C04C04 (*uaz161*), similar to Trypanosoma translation factor, may encode elongation factor 1-gamma	263
emb1	1.00	1.05	embryo specific1, (was *emb*-8531*) embryo development blocked at late proembryo to early transition stage	114
emb2	9.00	9.03	embryo specific2, (was *emb*-8522*) embryo development blocked at the transition stage	114
emb3	4.06	4.11	embryo specific3, (was *emb*-8547*) embryo development blocked at transition stage; suspensor bulbous; embryo proper enlarged	114
emb4	1.00	1.05	embryo specific4, (was *emb*-8502*; *emb*-8503*) embryo development blocked at late transition stage	114
emb5	2.06	2.10	embryo specific5, (was *emb*-8518*) embryo development blocked at transition to early coleoptilar stage	114
emb6	4.06	4.11	embryo specific6, (was *emb*-8540*) embryo development blocked at coleoptilar stage	114
emb7	1.00	1.05	embryo specific7, (was *emb*-8519*; *emb*-8520*) embryo development blocked at the transition stage through stage 2	114
emb8	4.06	4.11	embryo specific8, (was *emb*-8514*) embryo development blocked at coleoptilar stage or later	114
emb9	3.05	3.09	embryo specific9, (was *emb*-8521*) embryo development blocked at the transition stage or later	114
emb10	1.00	1.05	embryo specific10, (was *emb*-8545*) embryo development blocked during elaboration of embryonic structures (stage 4)	114
emb11	4.06	4.11	embryo specific11, (was *emb*-8513*) embryo development blocked from stage 3 to stage 6; small embryo	114
emp1	1.00	1.05	empty pericarp1, germless, unfilled kernel	597
emp2	2.06		empty pericarp2, germless, unfilled kernel	597
emp3	8.09		empty pericarp3, small, extremely collapsed, defective, poorly viable kernel	466
En1			Enhancer, transposable element (equivalent to *Spm*); autonomous, regulates transposition of I (=*dSpm*), e.g. at *g2-m* = pg-m = *pg14-m*, encodes TnpA	522, 523
end1			early nodulin homolog1, endosperm cDNA 5C04A12 (*uaz227*), similar to soybean early nodulin, may encode root meristem protein	263
eno1	9.02		enolase1, cDNA clone pZm245 complements enolase mutant in *E. coli*; leaf cDNA *csu158* probes same site, encodes enolase	345
eno2	1.05		enolase2, constitutive cDNA pENO2 , encodes enolase	346
enp1	6.02		endopeptidase1, electrophoretic mobility; null allele is known; monomeric, encodes endopeptidase	420
esp5			embryo specific protein5, (was *Emb5*); cDNA clone, embryo specific, ABA responsive, deduced amino acid sequence very hydrophilic and gly/glu-rich (aka *emb5*)	729
et1	3.09		etched1, pitted, scarred endosperm, virescent seedling; plastid membranes altered	661
et2	2.00	2.04	etched2, endosperm etched; seedlings off-white albino, with occasional greening of leaf tips	669
ets1			ets-family transcription factor homolog1, leaf cDNA *csu110*, multiple copies, similar to human Ets-related transcription factor, may encode ets-family transcription factor	29
f1	1.07		fine stripe1, virescent seedling, fine white stripes on base and margin of older leaves	366, 367
fab1			fatty acid biosynthesis1, endosperm cDNA 2C01H08 (uaz99) similar to fatty acid biosynthesis enzyme, may encode 3-oxoacyl-[acyl carrier protein] reductase	264
fae1			fasciated ear1, small, rounded ears branched at their tips	621
fas1			fascicled ear1, in *Fas1*, repeated dichotomous branching in floral meristems	712
fbr1			few-branched1, dominant *Fbr1* plants have tassel reduced to 0-3 branches; bract replaces next-to-bottom branch	479
Fcu			Factor Cunha, controlling element of *r1-cu*	234
fdx1			ferredoxin1, chloroplast ferredoxin, light induced, N-terminal amino acid sequence of mature protein, cDNA sequence, encodes ferredoxin	258
fdx3			ferredoxin3, ubiquitous, cDNA clone, gene specific probe, amino acid sequence, encodes ferredoxin	258, 398
fdx5			ferredoxin5, leaf protein, cDNA clone, distinct amino acid sequence compared to other ferredoxins, gene specific probe (aka *pfd5*), encodes ferredoxin	258

fer1			ferritin1, iron induced, cDNA sequences and expression pattern indicate two genes, *fer1*, *fer2* (Lobreaux et al 1992), encodes ferritin	373
fer2			ferritin homolog2, iron induced, cDNA sequences, differential expression indicates 2 genes, may encode ferritin	373
fgs1			ferredoxin-dependent glutamate synthase1, deduced amino acid sequence homologous with *E. coli* NADPH-glutamate synthase, single copy by Southern blot analysis, encodes glutamate synthase, ferredoxin dependent	584
fl1	2.04		floury endosperm1, (was *o4*) endosperm opaque, soft; dosage effect with fl1-ref allele, but *o4* allele is recessive	260
fl2	4.04		floury2, endosperm opaque, soft; dosage effect; encodes an abnormal 24kDa alpha-zein as deduced from clone	171, 447, 454
fl3	8.04		floury3, endosperm opaque, soft; dosage effect	383, 455
fnr1			ferredoxin NADP reductase1, single or low copy; root, nitrate induced cDNA; deduced amino acid sequence >90% similar to rice FNR; produces an active FNR product in *E. coli*, encodes ferredoxin--NADP(+) reductase	560
ftr1			ferredoxin-thioredoxin homolog1, cDNA sequence, may encode ferredoxin-thioredoxin reductase, chloroplast	392
g1	10.05		golden plant1, seedling and plant with distinctive golden yellow cast; stub of cut seedling displays golden vs. green distinguishably (aka *ws2*)	173, 175
g2	3.00		golden plant2, (was *g5*, *pg14*, *v19*, pg-m) golden pale-green, weak plants; sheaths whitish yellow-green; pg-m of Peterson is mutable allele carrying *En*	294, 523
g6	9.01	9.02	golden plant6, dominant *G6* plants golden; lighter yellowish sheaths	477
ga1	4.02		gametophyte factor1, (was *ga9*) *Ga1* pollen grains are competitively superior to *ga1* on *Ga1* silks; *Ga1*-S super-gametophyte	307
ga2	5.05		gametophyte factor2, *Ga2* pollen grains are competitively superior to *ga2*	80
ga7	3.09		gametophyte factor7, *ga7* pollen from heterozygotes is only 10-15% functional regardless of silk genotype	556
ga8	9.02		gametophyte factor8, *Ga8* pollen grains are competitively superior to *ga8* on *Ga8* silks	605
ga10	5.00	5.09	gametophyte factor10, *Ga10* pollen grains competitively superior to *ga10*; map position inconsistent with *ga2*	233
gbp1			GTP-binding protein homolog1, leaf cDNA *csu108*, partial sequence similar to *Arabidopsis* GTP binding protein, RHA1, may encode GTP binding protein	321
gbp2			GTP binding protein2, cDNA sequence similar animal G protein beta-subunits; single copy, may encode GTP binding protein, beta subunit	714
gcb1			GC binding protein 1, binds to anaerobic responsive element (ARE) of *Adh1* promoter	502
gdh1	1.11		glutamic dehydrogenase1, electrophoretic mobility; null allele is known (cold sensitivity); intra/interlocus hybrid bands occur, encodes glutamic dehydrogenase	538
gdh2	10.03		glutamic dehydrogenase2, electrophoretic mobility; intralocus hybrid bands occur, encodes glutamic dehydrogenase	235
geb1			glucan endo-1,3-beta-glucosidase homolog1, cDNA sequence homologous to 1,3-beta glucanase, may encode glucan endo-1,3-beta-glucosidase	742
gl1	7.02		glossy1, cuticle wax altered; leaf surface bright, water adheres, may encode phospholipid transfer protein	342
gl2	2.02		glossy2, like *gl1*, but surface wax is all rice-grain-type particles	48, 259
gl3	4.08		glossy3, like *gl1*, but surface wax has all rice-grain-type particles	259
gl4	4.06		glossy4, (was *gl16*) like *gl1*, but surface wax has reduced number of star-type particles	48, 652
gl5	4.05		glossy5, like *gl1*; duplicate factor with *gl20*; double homozygote phenotypically like *gl1*, but cuticle wax consists of large elongated particles.	48, 171, 652, 656
gl6	3.04		glossy6, like *gl1*, but seedling leaf surface bright green instead of bluish	171, 652
gl7			glossy7, (was *gl12*) like *gl1*	171, 652
gl8	5.05		glossy8, (was *gl10*) like *gl1*; cuticle wax in rice-grain-type particles, may encode beta-ketoacyl CoA reductase	171, 652
gl9	3.05	3.09	glossy9, expression poor	171, 652
gl11	2.03		glossy11, like *gl1*; abnormal seedling morphology, sometimes viviparous	48
gl13			glossy13, necrotic glossy leaf; plants that survive to maturity are sterile (both male and female) and smaller than nonmutant sibs.	12
gl14	2.00	2.10	glossy14, like *gl1*; expressed late (*gl24* allelic)	12
gl15	9.03		glossy15, glossy leaf surface expressed after 3rd leaf (aka *gl4*)	12
gl17	5.04		glossy17, like *gl1*, but semi-dwarf with necrotic crossbands on leaves	552
gl18	8.04	8.09	glossy18, like *gl1*; expression poor (aka *gl23*)	12
gl19	3.00	3.04	glossy19, like *gl1*; barren plant with no ear or tassel	458
gl20			glossy20, like *gl1*; duplicate factor with *gl5* (aka *gl5-2*)	656
gl21	10.00	10.03	glossy21, like *gl1*; duplicate factor with *gl22*	458
gl22			glossy22, like *gl1*; duplicate factor with *gl21*	480
gl24			glossy24, like *gl1*; duplicate factor with *gl14*, best at 4-leaf stage	657

glb1	1.09		globulin1, (was *prot1*) electrophoretic mobility; null allele is known; embryo protein, encodes globulin, 63,000 kDa	337, 609
glb2			globulin2, presence-absence, encodes globulin, 45,000 kDa	337
gln1	10.07		glutamine synthetase1, chloroplast, gene specific cDNA probe, 6-member nuclear gene family, encodes glutamate--ammonia ligase, chloroplast	641
gln2			glutamine synthetase2, cytosolic, root specific, gene specific cDNA probe, 6-member nuclear gene family, encodes glutamate--ammonia ligase, cytosol	364, 585
gln3			glutamine synthetase3, cytosolic, minor species, specific to young seedlings, gene specific cDNA probe, 6-member nuclear gene family, encodes glutamate--ammonia ligase, cytosol	585, 641
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	585, 641
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	585
gln6			glutamine synthetase6, cytosolic, a major species in root, gene specific cDNA probe, 6-member nuclear gene family, encodes glutamate--ammonia ligase, cytosol	585, 641
glu1	10.03		beta glucosidase1, electrophoretic mobility; plastidic; dimeric; intralocus hybrid bands occur, encodes beta glucosidase	539
gn1	2.10		gnarley1, dominant (*Gn1*) characterized by reduced internodal length, sinuously curving culm, lack of distinct boundary between blade and sheath, extra silks	198
gos1			GOS2 homolog, endosperm cDNA 5C09H04, similar to rice GOS2 protein, may encode translation factor, SUI1 family	264
got1	3.08		glutamate-oxaloacetate transaminase1, electrophoretic mobility; null allele is known; glyoxysomal; dimeric; intralocus hybrid bands occur, encodes aspartate aminotransferase	592
got2	5.08		glutamate-oxaloacetate transaminase2, electrophoretic mobility; null allele is known; plastidial; dimeric; intralocus hybrid bands occur, encodes aspartate aminotransferase	237
got3	5.03	5.04	glutamate-oxaloacetic transaminase3, electrophoretic mobility; null allele is known; mitochondrial; dimeric; intralocus hybrid bands occur, encodes aspartate aminotransferase	237
gpa1	10.04		glyceraldehyde-3-phosphate dehydrogenase1, chloroplastic, A subunit; two pseudogenes have been identified, and leaf cDNA *csu140* hybridizes to 2 other bands, encodes glyceraldehyde-3-phosphate dehydrogenase (NADP+) (phosphorylating)	68
gpb1			glyceraldehyde phosphate dehydrogenase *B1*, leaf cDNA *csu152* similar to *Arabidopsis GapB*, may encode glyceraldehyde-3-phosphate dehydrogenase (NADP+) (phosphorylating)	321
gpc1	4.05		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	68, 394, 581
gpc2	6.00	6.01	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	581
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	581
gpc4	5.05		glyceraldehyde-3-phosphate dehydrogenase4, C subunit, electrophoretic mobility, coding sequence homology to *gpc3*, unique 3' untranslated region, encodes glyceraldehyde-3-phosphate dehydrogenase C, cytosolic	582
gpdh1			glucose-6-phosphate dehydrogenase1, leaf cDNA *csu350*, 5' sequence similar to plant glucose-6-phosphate dehydrogenase, encodes glucose-6-phosphate dehydrogenase	31
gpn1			glyceraldehyde-3-phosphate deHaseN1, cDNA sequence, encodes glyceraldehyde-3-phosphate dehydrogenase (NADP+) (nonphosphorylating)	250
grf1			G-box regulatory factor1, cDNA sequence; protein co-immunoprecipitates with G-box binding complex but does not bind to DNA;low copy number, encodes G-box regulatory factor14	149
grp1			glycine-rich protein1, protein with high glycine content and repetitive glycine stretches; putative cell wall components, encodes glycine-rich protein	150
grt1	5.05	5.09	green tip1, pale yellow seedling with green first leaf tip; lethal	458
grx1			glutaredoxin homolog1, leaf cDNA csu40, similar to *E. coli* glutaredoxin, may encode glutaredoxin	29
gs1	1.10		green stripe1, grayish green stripes between vascular bundles on leaves; tissue wilts	178, 433
gs2	2.03	2.04	green stripe2, like *gs1*, but pale green stripes; no wilting	171, 651, 652
gs3	6.02	6.08	green stripe3, like *gs2* but much smaller plant	458
gs4	10.04	10.05	green stripe4, dominant *Gs4* plants are like *gs1* but smaller plant	481
gsr1	1.02		glutathione reductase1, single copy leaf cDNA *csu111*, similar to pea glutathione reductase, encodes glutathione reductase	29
gss1			starch synthase homolog1, endosperm cDNA 5C04B10 (*uaz218*), similar to pea starch synthase isoform II, may encode starch synthase	263
gst1	8.08		glutathione-S-transferase1, presence-absence of isozyme bands, between inbred lines, members of a family of polymorphic bands; transcripts correlate with dimer isoforms; single copy in Southerns, encodes glutathione S-transferase	588

gst3			glutathione transferase3, cDNA sequence, correspondence to amino terminal of maize GSTIII, low or single copy, transgenic expression in *E. coli* results in enzyme activity similar to maize GSTIII enzyme, encodes glutathione S-transferase	441
gt1	1.00	1.12	grassy tillers1, numerous basal branches; vegetatively totipotent in combination with *id1* and factors for perennialism	614
gzs1			*Gibberella zea* silk mediated resistance1, dominant resistance to *Gibberella zea* (ear rot) through the silk	549, 550
hl	3.02	3.03	soft starch1, endosperm opaque, starchy (like floury), recessive to horny	446
hca1			histocompatibility antigen homolog1, endosperm cDNA 5C04C07 (*uaz199*), similar to human histocompatibility antigen, may encode glycoprotein	263
hcf1	2.05	2.10	high chlorophyll fluorescence1, affects NADP+ oxidoreductase; green seedling	426
hcf2	1.06	1.08	high chlorophyll fluorescence2, missing cytochrome bf complex; yellow-green seedling	426
hcf3	1.03		high chlorophyll fluorescence3, (aka *hcf9*) missing PSII thylakoid membrane core complex; green seedling	426
hcf4	1.06	1.12	high chlorophyll fluorescence4, affects CO_2 fixation; green seedling	428
hcf5	6.00	6.01	high chlorophyll fluorescence5, affects PSII reaction; green seedling	427
hcf6	1.00	1.05	high chlorophyll fluorescence6, missing cytochrome bf complex; green seedling	361
hcf7	1.06	1.12	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.	25
hcf11			high chlorophyll fluorescence11, pale green leaves, deficient in CO_2 fixation, often lethal at 3- to 5-leaf stage	429
hcf12	1.06	1.12	high chlorophyll fluorescence12, green seedling	361
hcf13	1.07		high chlorophyll fluorescence13, affects CO_2 fixation; green seedling	361
hcf15	2.05	2.10	high chlorophyll fluorescence15, affects photophosphorylation; yellow-green seedling, may survive	361
hcf18	5.05	5.09	high chlorophyll fluorescence18, major loss of PSI; other thylakoid complexes reduced; yellow-green seedling (possibly allel ic to *hcf43*)	427
hcf19	3.05	3.09	high chlorophyll fluorescence19, affects PSII thylakoid membrane core complex; green/yellow-green seedling	361
hcf21	5.05	5.09	high chlorophyll fluorescence21, affects CO_2 fixation, Rubisco; green seedling	427, 428
hcf23	4.00	4.05	high chlorophyll fluorescence23, affects photophosphorylation; yellow-green seedling, may survive	361
hcf26	6.00	6.01	high chlorophyll fluorescence26, affects electron transport; yellow-green, viable seedling	361
hcf28	10.05	10.06	high chlorophyll fluorescence28, affects CO_2 fixation; green seedling	427
hcf31	1.00	1.05	high chlorophyll fluorescence31, missing chlorophyll a/b binding protein; yellow-green seedling	427
hcf34	6.01	6.08	high chlorophyll fluorescence34, affects photophosphorylation; yellow-green seedling	361
hcf36	6.01	6.08	high chlorophyll fluorescence36, affects electron transport; green seedling	427
hcf38	5.05	5.09	high chlorophyll fluorescence38, affects cytochrome bf complex, alpha and beta components of CF1; green seedling	361
hcf41	1.06	1.12	high chlorophyll fluorescence41, affects PSII thylakoid membrane core complex; green seedling	361
hcf42	9.03	9.08	high chlorophyll fluorescence42, affects Rubisco; green/yellow-green seedling	428
hcf43	5.05	5.09	high chlorophyll fluorescence43, yellow-green leaves, deficient in all thylakoid polypeptides except for the antenna complexes (possibly allelic to *hcf18*)	427, 432
hcf44	1.06	1.12	high chlorophyll fluorescence44, affects PSI membrane core complex; pale-green seedling, lethal	428
hcf45			high chlorophyll fluorescence45, CO_2-fixation reduced 90%, normal levels of RUBISCO protein	429
hcf46	3.05	3.09	high chlorophyll fluorescence46, ultraviolet light red fluorescence	361
hcf47	10.00	10.03	high chlorophyll fluorescence47, affects cytochromes; yellow-green seedling	427
hcf48	6.01	6.08	high chlorophyll fluorescence48, affects electron transport; yellow-green seedling	427
hcf49			high chlorophyll fluorescence49, PSI mutant, not allelic to *hcf44* or *hcf50*	431
hcf50	1.06	1.12	high chlorophyll fluorescence50, missing PSI thylakoid membrane core complex; seedling slightly pale green	428
hcf60			high chlorophyll fluorescence60, green to pale green seedling leaves, deficiency in photosystem I	431
hcf101	7.00	7.06	high chlorophyll fluorescence101, affects PSI thylakoid membrane core complex	427
hcf102	8.04	8.09	high chlorophyll fluorescence102, affects cytochrome bf complex	431
hcf103	7.00	7.06	high chlorophyll fluorescence103, (was *hcf114*) reduced photosystem II activity due to absence of plastoquinone (PQ-9)	127
hcf104	7.00	7.06	high chlorophyll fluorescence104, photosystem I-deficient	127
hcf106	2.05		high chlorophyll fluorescence106, affects PSI, PSII, cytochrome bf, encodes *HCF106* thylakoid membrane protein	393
hcf108	5.01		high chlorophyll fluorescence108, ATPase-deficient	127
hcf113	9.00	9.03	high chlorophyll fluorescence113, multiple effects; yellow-green seedlings	126
hcf120			high chlorophyll fluorescence120, cytochrome bf and photosystem II deficient	686
hcf316	10.00	10.03	high chlorophyll fluorescence316, affects chlorophyll a/b binding protein; yellow-green seedling	427

hcf323	6.00	6.01	high chlorophyll fluorescence323, affects photophosphorylation, coupling factor; green seedling	427
hcf408	6.01	6.08	high chlorophyll fluorescence408, affects chlorophyll a/b binding protein; yellow-green seedling	427
hex1	3.02	3.03	hexokinase1, electrophoretic mobility; null allele is known; cytosolic; monomeric, encodes hexokinase	719, 721
hex2	6.04	6.05	hexokinase2, electrophoretic mobility; null allele is known; cytosolic; monomeric, encodes hexokinase	720, 721
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	715
hhp1	10.02		hupm/hypb protein family homolog1, leaf cDNA *csu103*, 5'-sequence similar to hupm/hypb protein family, gene specific probe, may encode hupm/hypb protein	29
his1			histone I, histone H1 gene family, cDNA sequence, encodes histone I	547
his2a1			histone2A1, cDNA from unfertilized ovule, similar to histone2A, encodes histone 2A	52
his2b1			histone2b1, cDNA to mRNA from 8 day seedlings, protein reacts with antibodies for histone H2B, encodes histone 2B	301
his2b2			histone2b2, cDNA to mRNA from seedlings, protein reacts with histone2B antibodies, encodes histone 2B	301
his2b3			histone 2B3, genomic sequence selected with his2B cDNA probe, encodes histone 2B	302
his2b4			histone 2B4, genomic sequence selected with his2B cDNA probe, encodes histone 2B	302
his3			histone3, histone H3 family, 60-80 copies/diploid genome, homologous sites on several chromosomes, subfamilies H3C2, H3C3, H3C4, encodes histone 3	103
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	103, 104
hm1	1.06		*Helminthosporium carbonum* susceptibility1, disease lesions vs. yellowish flecks (resistant) on leaves with *Cochliobolus carbonum* race 1, encodes NADPH HC-toxin reductase	696
hm2	9.04		*Helminthosporium carbonum* susceptibility2, dominant *Hm2* plants resistant to *Cochliobolus carbonum*. Like *Hm1*; masked by *Hm1*	453
hox1	8.04		homeobox1, protein product binds to *sh1* promoter (feedback control element), is found in nuclei, encodes *hox1* protein	45
hox2	6.06		homeobox2, possibly = *koln1B*; similar to *hox1*, but sequence predicts not allelic to *hox1*, encodes *hox2* protein	45
hpt1			hygromycin phosphotransferase1, transgenic chimeric gene, single dominant locus, coding region origin *E. coli* introduced by particle bombardment, encodes hygromycin-B kinase	709
hrg1	2.04		hydroxyproline rich glycoprotein1, cDNA, genomic clones, peptide sequence, single site (Southern analysis), accumulates in dividing cells, preferentially in provascular cells, encodes hydroxyproline-rich glycoprotein	665, 666
hs1	7.00	7.01	hairy sheath1, dominant *Hs1* plants have abundant hairs on leaf sheath throughout development	685
hsf1	5.06		hairy sheath frayed1, dominant *Hsf1* plants have pubescent sheaths and leaf margins; liguled enations at leaf margins	49
hsk1			high-sulfur keratin homolog1, endosperm cDNA 5C04B04 (*uaz144*), similar to high-sulfur keratin, encodes high sulfur keratin homolog	263
hsp1			heat shock protein1, genomic clones, single copy (Southern blots), transcribed (Northern blots), transgenic (petunia) expression, encodes HSP70	574
hsp18a	9.05		18 kda heat shock protein18a, induced by heat shock specifically in anthers or spikelets containing meitotic microsporocytes, encodes 18 kDa heat shock protein, cytoplasmic	17
hsp18c	8.03		heat shock protein18c, induced by heat shock specifically in anthers or spikelets containing meiotic microsporocytes, encodes 18 kDa heat shock protein, cytoplasmic	238
hsp18f	3.03		heat shock protein18f, induction by heat shock is developmentally constitutive, encodes 18 kDa heat shock protein, cytoplasmic	238
hsp26	1.03		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	495
hsp60			heat shock protein60, cDNA sequence homologous to hsp60 family, encodes mitochondrial chaperonin hsp60	533
ht1	2.08		*Helminthosporium turcicum* resistance1, dominant *Ht1* plants resistant to *Exserohilum turcicum*	273, 274
ht2	8.05		*Helminthosporium turcicum* resistance2, dominant *Ht2* plants resistant to *Exserohilum turcicum*	276
ht3			*Helminthosporium turcicum* resistance3, (from *Tripsacum floridanum*); dominant *Ht3* plants resistant to *Exserohilum turcicum*	272, 277
htm1			*Exserohilum turcicum* Mayorbela resistance1, dominant *Htm1* plants resistant	562
htn1	8.06		*Helminthosporium turcicum* resistanceN1, formerly *HtN*; dominant *Htn1* plants resistant to *Exserohilum turcicum*	627
htz2			high tryptophan zein2, recessive increased in tryptophan content (.36 to .40 vs. .15 to .17%) in zein	622
htz3			high tryptophan zein3, recessive increased tryptophan content in zein	622
hvp1			human viral protein homolog1, endosperm cDNA 5C02B04, similar to a virus transcription activator, may encode transcription factor	264

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	311
I-R			Inhibitor of *R*, excision is responsible for *R1-st* stippling; transposed element modifies stippling level	419
iaglu1			indol-3-ylacetyl glucosyl transferase1, single or low copy cDNA sequence matches N-terminal polypeptide sequence of purified maize indol-3-ylacetyl glucosyl transferase and, in transformed *E. coli*, produces a catalytically active protein, encodes (uridine 5'-diphosphate-glucose:indol-3-ylacetyl)-beta-D-glucosyl transferase	682
ibp1			initiator binding protein1, cDNA sequence encodes protein binding to the shrunken promoter; duplicate loci distinguished by gene-specific sequences, both transcribed, encodes shrunken initiator-binding protein	381
ibp2	1.00		initiator-binding protein2, cDNA sequence selected as encoding protein binding to the shrunken promoter; duplicate loci distinguished by gene-specific sequences, both transcribed, encodes shrunken initiator-binding protein	381
id1	1.08		indeterminate growth1, requires extended growth and short days for flowering; vegetatively totipotent with *gt1* and factors for perennialism	633
idh1	8.06		isocitrate dehydrogenase1, electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur, encodes isocitrate dehydrogenase	237
idh2	6.07		isocitrate dehydrogenase2, electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur, encodes isocitrate dehydrogenase	237
ig1	3.06		indeterminate gametophyte1, low male fertility, polyembryony, heterofertilization, polyploidy, androgenesis (male and female affected)	331
ij1	7.03		iojap striping1, many variable white stripes and margin patterns on leaves (compare *cm1*); conditions chloroplast defects that are cytoplasmically inherited, encodes IJ1	293
ij2	1.11	1.12	iojap striping2, like *ij1*; chloroplast inheritance unknown	458
in1	7.02		intensifier1, intensifies aleurone anthocyanin pigments; *In1-D* dominant dilute	200
Ins1			insertion1, located upstream of *bz1-R*; up to 50 copies in genome (Southerns)	544
Ins2			insertion2, insertion2: 447 bp element upstream of *bz1-R*	544
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	449
is1			cupulate interspace1, space between the apex of the cupule and the glume cushion above; trait characteristic of teosinte	211
isp1			iron-sulfur protein1, cDNA, nuclear-encoded mitochondrial Rieske iron-sulfur protein, functional analysis in yeast, encodes Rieske iron-sulfur protein, mitochondrial	280
isr1	10.06		inhibitor of striate1, (a.k.a. *Ej1*), dominant *Isr1* plants have reduced expression of *sr2* and other leaf-striping factors	329
j1	8.08		japonica striping1, white stripes on leaf and sheath; not often expressed in seedling	175
j2	4.08		japonica striping2, extreme white striping of leaves, etc.	171
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	405, 515
K3L	3.07		knob on 3L, heterochromatic structure found in most strains, varies in size	147
K9S	9.00		knob on 9S, heterochromatic terminal knob found in some strains and not in others	406
K10	10.07		abnormal10, heterochromatic alternative end of long arm of chromosome 10 found in some strains; neocentric activity distorts segregation of knobs and of genes linked to them	376
kn1	1.10		knotted1, dominant *Kn1* plants have localized proliferation of tissue at vascular bundles on leaf	73
knox3	1.10		knotted related homeobox3, shoot meristem and developing stem specific, very similar in sequence and expression pattern to *rs1*, *kn1* and *knox8*	638
knox4	2.10		knotted-like homeobox4, genomic clone identified by homology to *kn1* homeobox, gene specific probe; possibly identical to *gn1*	198
knox8			knotted related homeobox8, cDNA, shoot meristem and developing stem specific, similar in sequence and expression pattern to *kn1*	289
kri1			ketol-acid reductoisomerase1, endosperm cDNA 5C05H04 (*uaz269*) similar to plant branched chain amino acid synthesis enzyme, may encode ketol-acid reductoisomerase	264
l1	10.06		luteus1, yellow pigment in white tissue of specific chlorophyll mutants *w1*, *w2*, *j1*, *ij1*, others	365, 366
l3			luteus3, lethal yellow seedling	291
l4			luteus4, lethal yellow seedling	291
l6	9.02		luteus6, like *l4*	171, 190
l7	9.02		luteus7, yellow seedling and plant; lethal	190
l10	6.01	6.02	luteus10, like *l4*; fails to convert protochlorophyllide to chlorophyllide	568
l11	6.03		luteus11, yellow seedling with green leaf tips; lethal	20
l12	6.01	6.02	luteus12, like l11	133
l13	10.07		luteus13, dark yellow, lethal seedling; fails to convert protoporphyrin IX to Mg-protoporphyrin	396, 458
l15	6.02		luteus15, like *l4*; lethal yellow seedling	572
l16	1.00	1.05	luteus16, like *l4*; leaves bleach to paler yellow in patches	458
l17	1.06	1.12	luteus17, like *l4*; leaves with lighter yellow crossbands	458

l18	2.06	2.10	luteus18, like *l4*	458
l19	10.00	10.03	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	458
la1	4.03	4.04	lazy plant1, prostrate growth habit	292
lbl1			leaf bladeless1, leaf blade reduced to absent; low temperature enhances expression	430
lc1	10.06		red leaf color1, dominant *Lc1* confers anthocyanin in coleoptile, nodes, auricle, leaf blade, etc.; (compare Sn1), encodes LC1	54, 159
lcs1			thylakoid membrane polypeptide1, electrophoretic mobility	438
lct1			thylakoid membrane polypeptide1, electrophoretic mobility	438
lct2			thylakoid membrane polypeptide2, presence-absence	438
les1	2.04		lesion1, dominant *Les1* plants have large necrotic lesions resembling disease lesions formed by fungal infections on susceptible lines	459, 474
les2	1.02		lesion2, dominant *Les2* plants have small white lesions resembling disease lesions formed by fungal infections on resistant lines	459, 474
les3	10.00	10.07	lesion3, like *les1*; large, elliptical, necrotic lesions	15
les4	2.05	2.10	lesion4, dominant *Les4* plants have late expression of large necrotic lesions on leaf blade and sheath	270, 464
les5	1.00	1.05	lesion5, like *les2*	270, 464
les6	10.00	10.03	lesion6, like *les4*, but with many small to medium, irregular, mottled spots	270, 464
les7			lesion7, dominant *Les7* plants have late expression of small chlorotic lesions	270, 464
les8	9.03		lesion8, dominant *Les8* plants have late expression of small, pale green lesions	49, 270
les9	7.02		lesion9, dominant *Les9* plants have late expression of small necrotic lesions	270
les10	2.05	2.06	lesion10, like *Les1*; numerous small, round, necrotic lesions	271, 706
les11	2.02		lesion11, like *les1*	464, 484
les12	10.00	10.07	lesion12, dominant *Les12* plants have many small to medium, chlorotic to necrotic lesions on the leaf blade beginning at 5 leaf stage	464, 484
les13	6.00	6.02	lesion13, dominant *Les13* plants have frequent small to medium necrotic spots on leaf blade, sheath and culm, appearing at the 5 leaf stage	270, 484
les14	3.00	3.09	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	270, 484
les15	2.04		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	484
les16	10.00	10.03	lesion16, dominant *Les16* plants are pale green and develop small chlorotic lesions on the leaf blade just before flowering	484
les17	3.00	3.09	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	270, 484
les18	2.03	2.04	lesion18, dominant *Les18*, leaf lesions; map location distinct from *les11*	462
les19	2.06	2.07	lesion19, dominant *Les19*, leaf lesions; location near that of *les10* but much different in expression	462
les20	1.05	1.08	lesion20, dominant *Les20* plants have small chlorotic spot lesions	489
lfy1			leafy1, dominant *Lfy1* plants have increased number of leaves above ear	612
lg1	2.02		liguleless1, ligule and auricle missing; leaves upright, enveloping	173, 174
lg2	3.06		liguleless2, like *lg1*, less extreme	65
lg3	3.04		liguleless3, dominant *Lg3* plants lack ligule; leaves upright, broad, often concave and pleated	518
lg4	8.05		liguleless4, dominant *Lg4* plants lack ligule and auricle but show vestiges sporadically in blade	199
lhcb1	3.09		light harvesting chlorophyll a/b binding protein1, gene-specific cDNA probe; low expression in bundle sheath cells (aka *cab1*), encodes chlorophyll a/b binding protein candidate	615, 739
lhcb2	7.04		light harvesting chlorophyll a/b binding protein2, gene specific cDNA probe; expressed in dark (aka *cab1*, *cab2*)	615, 679, 739
lhcb3	8.03		light harvesting chlorophyll a/b binding protein3, probed by clone provided by L. Bogorad (aka *cab4*), encodes light-harvesting chlorophyll a/b binding protein	615
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	334
lhcbm7			light harvesting complex mesophyll7, cDNA sequence distinct from other light harvesting chlorophyll polypeptides; two copies by Southern blot analysis (aka *cab-m7*), encodes light-harvesting chlorophyll a/b binding protein	399
li1	10.04		lineate leaves1, fine, white striations on basal half of mature leaves	122
lld1			lethal dwarf1, dominant *Lld1* plants small 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	472
lls1	1.01	1.03	lethal leaf spot1, chlorotic-necrotic lesions resembling *Helminthosporium carbonum* infection	697
ln1	6.00	6.02	linoleic acid1, lower ratio of oleate to linoleate in kernel	138

lo2	9.02		lethal ovule2, ovules containing *lo2* gametophyte abort; embryo sac development stops at 2 to 4-nucleate stage	450
loc1			low oil content in kernel1, associated with albino seedlings	530
lop1			*lo1* pl allergen homolog1, cDNA sequence homologous to allergen *Lo1* pl, encodes LOP1, putative pollen allergen	69
lox1			lipoxygenase1, leaf cDNA *csu160*, similar to *Arabidopsis* lipoxygenase, encodes lipoxygenase	30
lp1	4.00	4.11	lethal pollen1, *lp1* pollen fails in competition with *Lp1*	457
ltf1			lysr transcription factor homolog1, endosperm cDNA 5C02B05 (*uaz275*), similar to lysr family of transcription regulators, may encode lysr transcription factor	263
lty1			light yellow endosperm1, reduced color; heterozygote advantage; induced by mutagens in Oh43	157
lty2			light yellow endosperm2, reduced color; heterozygote advantage; induced by EMS in Oh43	157
lu1	5.03	5.04	lutescent1, pale yellow green leaves with *lu2*	44, 624
lu2			lutescent2, yellow green leaves with *lu1*	625
lw1	1.10		lemon white1, white seedling, pale yellow endosperm	693
lw2	5.05		lemon white2, like *lw1*	693
lw3	5.06		lemon white3, like *lw1*; duplicate factor with *lw4*	693
lw4	4.06		lemon white4, like *lw1*; duplicate factor with *lw3*	693
lxm1	3.06		lax midrib1, dominant *Lxm1* plants have leaves with wide, flat, flexible midrib	49, 478
mac1	10.00	10.03	multiple archesporial cells1, (was *lar*-487*) several archesporial cells undergo normal meiosis; plants partially female sterile but completely male sterile	617
magellan			magellan, low copy number, *Ty3*/gypsy-like retrotransposon isolated from *wx1-M* and found in all *Zea* species and in *Tripsacum andersonii*	540, 725
map1			microtubule associated protein homolog1, leaf cDNA csu21, similar to mouse microtubule associated protein, MAP2, may encode microtubule calmodulin binding protein	30
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	18
mas1			malate synthase1, cDNA isolated by antibody screen, encodes malate synthase	504
mc1			mucronate1, dominant *Mc1* kernels have opaque endosperm	586
mch1			maize CRY1 homolog1, ribosomal protein gene family (cDNA probe), encodes 40S ribosomal protein S14, cytoplasmic	352
mch2			maize CRY1 homolog2, ribosomal protein gene family (cDNA probe), encodes 40S ribosomal protein S14, cytoplasmic	352
mct1			modifier of *cox2* transcripts1, changes transcripts of mitochondrial gene	129
mde1			mouse DNA EBV homolog1, endosperm cDNA 5C02D07, similar to mouse homolog to Epstein-Barr virus IR3 repeat	264
mdh1	8.03		malate dehydrogenase1, electrophoretic mobility; null allele is known; mitochondrial; dimeric; intra/interlocus hybrid bands occur, encodes malate dehydrogenase	491
mdh2	6.07		malate dehydrogenase2, electrophoretic mobility; null allele is known; mitochondrial; dimeric; intra/interlocus hybrid bands occur, encodes malate dehydrogenase	491
mdh3	3.08		malate dehydrogenase3, electrophoretic mobility; null allele is known; mitochondrial; dimeric; intra/interlocus hybrid bands occur, encodes malate dehydrogenase	491
mdh4	1.08		malate dehydrogenase4, electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur; probed by csu77, encodes malate dehydrogenase	321, 491
mdh5	5.03		malate dehydrogenase5, electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur, encodes malate dehydrogenase	491
mdh6			malate dehydrogenase6, putative chloroplast enzyme, cDNA sequence, may encode malate dehydrogenase (NADP+)	421
mdm1	6.01		maize dwarf mosaic virus resistance1, dominant *Mdm1* plants resistant	416
MDMV-cp			maize dwarf mosaic coat protein, confers resistance to strains MDMV-A and MDMV-B, encodes maize dwarf mosaic virus coat protein	448
mdr1	4.06	4.11	maternal derepression of *R1*, *R1 R1 r1* aleurone mottled if *mdr1 mdr1 Mdr1*, solid color if *Mdr1 Mdr1 mdrr1*	330
me1	3.08		NADP malic enzyme1, electrophoretic mobility; null allele is known; tetrameric, encodes malate dehydrogenase (oxaloacetate decarboxylating) (NADP+)	237
me3	3.03		NADP malic enzyme3, leaf cDNA csu16 similar to malic enzymes, putative plastid transit peptide, single copy or major band, encodes malate dehydrogenase (oxaloacetate decarboxylating) (NADP+)	321, 579
Med			Mediator of *Irma* excision, lacks suppressor or mutator function but is required with *En* for excision of reduced *En*-like *Irma* element	449
mei1			meiosis1, male sterile with dominant *Mei1*, chromatin hyper-condensed, chromosomes sticky in metaphase I and anaphase I	230, 231
mep1	5.05		modifier of embryo protein1, affects quantities of *Glb1* protein forms	609
mfs14			male flower specific14, cDNA sequenced, associated with early microsporogenesis, encodes MFS14	741

mfs18			male flower specific18, cDNA sequence, associated with tassel glume vascular bundles, encodes MFS18 protein precursor	741
mg1			miniature germ1, germ 1/4 to 1/3 of normal; viable	347
mgs1	10.04		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	674
mgs2	4.09		male gametophyte-specific2, pollen-specific cDNA with pectate lyase homology, may encode pectate lyase	86, 694
mha1	2.09		membrane H(+)-ATPase1, cDNA and genomic sequences similar to plant plasma membrane proton-translocating ATPase, major band in Southerns; partly contained in *Bs-1* retroelement, may encode H(+)-ATPase, plasma membrane--fragment	77, 299, 306, 509
mip1			major intrinsic membrane protein homolog1, leaf cDNA *csu177*, similar to rice nodulin, may encode membrane permease	31
mmm1	1.08		modifier of mitochondrial malate dehydrogenases1, mobilities of the mitochondrial isozymes encoded by *Mdh1*, *Mdh2* and *Mdh3* are slightly increased in *mmm1* homozygotes	491
mn1	2.04		miniature seed1, small, somewhat defective kernel, fully viable; invertase reduced	380
mn2	7.00	7.06	miniature seed2, small kernel, loose pericarp; extremely defective but will germinate	348
mn3	6.01		miniature seed3, small kernel, etched/pitted endosperm; viable	670
mn4			miniature seed4, smaller, dented, viable kernels; double mutant with *cp3* has orange pericarp	468
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	743
mnb2			DNA binding protein MNB2, cDNA sequence, Southern blots indicate small gene family, encodes DNA-binding protein MNB1b	743
Mod			Modifier, inactive *Spm* element, enhances excisions elicited by active *Spm*	414
monosomic			aneuploid individual with one or more entire chromosomes missing from an otherwise diploid complement	
Mp			Modulator of pericarp, transposable factor originally found at *P1-vv*; parallel to *Ac-Ds*	63
Mpi1			Max Planck Institut1, transposable element, 10-15 copies in the genome	727
mpl1	1.10		miniplant1, dominant *Mpl1* plants are andromonoecious, intermediate dwarf (compare *D8*, possible allele); not responsive to gibberellins	255
mpu			maize palindromic unit, family of sequences occurring upstream of zein gene(s), *cat3*, and *kn1*, with potential homology to reverse transcriptases; 17- to 21- bp perfect palindromes, copy number 30,000	535
Mr	9.02		Mutator of *R1-m*, transposable factor	96, 501
Mrh	9.04		Mutator Rhoades, controlling element of *a1-mrh*	553
mrp1			MRP homolog1, endosperm cDNA 5C02B03, similar to *E. coli* MRP	264
ms1	6.02		male sterile1, anthers shriveled, not usually exserted; affected at microspore vacuolation	202, 631
ms2	9.03		male sterile2, like *ms1*; affected between vacuolation and pore formation	188, 190
ms3	3.06	3.07	male sterile3, anthers shrivelled; not usually exserted.	188, 190
ms5	5.04	5.05	male sterile5, anthers not exserted; affected at microspore mitosis	37
ms7	7.02		male sterile7, like *ms2*; tapetal cell dysfunction	37
ms8	8.07	8.08	male sterile8, pollen mother cells degenerate	37
ms9	1.01	1.05	male sterile9, breakdown of pollen mother cells	37
ms10	10.04	10.05	male sterile10, like *ms5*; affected at microspore vacuolation	37
ms11	10.04		male sterile11, like *ms5*; affected at microspore mitosis	37
ms12	1.00	1.12	male sterile12, like *ms1*; affected at microspore vacuolation	37
ms13	5.00	5.04	male sterile13, like *ms5*; affected at microspore vacuolation	37
ms14	1.05		male sterile14, like *ms5*; affected at microspore mitosis	37
ms17	1.03		male sterile17, like *ms1*; affected variably in meiosis	181
ms20			male sterile20, degeneration obvious by mid-vacuolated microspore stage	190
ms21	6.00	6.08	male sterile21, pollen grains developing in presence of dominant *Ms21* are defective and nonfunctional if *sks1*, normal if *Sks1*	358, 604
ms22			male sterile22, pollen mother cells degenerate	726
ms23	3.05	3.09	male sterile23, pollen mother cells degenerate	726
ms24			male sterile24, like *ms1*; affected in microspore mitosis	726
ms28	1.00	1.05	male sterile28, anaphase I disturbed, spindle persists	230
ms41	4.08		male sterile41, dominant *Ms41* plants male sterile	471
ms42	5.00	5.01	male sterile42, dominant *Ms42* plants male sterile; penetrance varies	4
ms43	8.04	8.09	male sterile43, anaphase I impaired (aka *ms29*)	230, 231
ms44	4.08		male sterile44, dominant *Ms44* plants male sterile	2, 3
msc1	1.06	1.12	mosaic1, dominant *Msc1* aleurone mosaic for anthocyanin color	471
msc2	5.00	5.04	mosaic2, dominant *Msc2* aleurone mosaic for anthocyanin color	471
msr1			macrophage scavenger receptor homolog1, endosperm cDNA 5C04D02, similar to human macrophage scavenger receptor	264
mst1	10.06		modifier of *R1-st1*, dominant *Mst1* reduces mutability of *R1-st*	16
msv1	1.04		maize streak virus tolerance1, dominant *Msv1* plants tolerant	343

mta1			mouse transplantation antigen homolog1, endosperm cDNA 5C04D09 (*uaz208*), similar to *Arabidopsis* homologue of a mouse transplantation antigen, may encode glycoprotein	263
mtl1			metallothionein homolog1, genomic clone, transcriptional and translation start sites mapped, Northern blots, similar to other class-I metallothioneins, may encode metallothionein	137
Mu1			Mutator1, freely transposable element; *Mu1* designates element isolated from *Adh1*-S3034	571
Mu4			Mutator4, elements with terminal inverted repeats similar to *Mu1*	683
Mu5			Mutator5, element with inverted terminal repeats similar to *Mu1*	683
Mu8			Mutator8, 1.4 kbp element within wx1-mum5; terminal inverted repeats similar to *Mu1*	196
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), encodes MURA, MURB	265, 541
Mut	2.02		Mutator Rhoades, controlling element for *bz1-m-rh*; distinct from *Mrh*	553
mv1	3.05		resistance to maize mosaic virus I ("corn stripe")1, dominant confers resistance to maize mosaic virus I ("corn stripe")	58
Mx			Mobile element induced by X-rays, element found at *bz1-x3m* and elsewhere in the genome; *Bz1* restriction fragments correlated (insertion and reversion)	442
myg1			maternal yellow-green1, like *hcf2* except maternally inherited and yellow-green; induced in Mutator background	444
na1	3.06		nana plant1, short, erect dwarf; no response to gibberellins	286, 363
na2	5.03		nana plant2, like na1	517
nac1			NaCl stress protein1, endosperm cDNA 5C01G10 (*uaz250*), similar to wheat salt-stress peptide, may encode salt stress protein	263
nad1			NADH ubiquinone oxidoreductase1, endosperm cDNA 5C05D10 (*uaz266*), similar to a NADH-ubiquinone oxidoreductase subunit, encodes NADH:ubiquinone oxidoreductase, 29.9 kDa subunit family	264
nbp1	7.02		nucleic acid binding protein1, genomic and cDNA clones; product is imported in vitro into chloroplasts; expressed only in leaf, encodes nucleic acid-binding protein	128
nc			North Carolina, descriptor for SSR loci	611
NCS1			nonchromosomal stripe1, maternally inherited light green leaf striping	626
NCS2			nonchromosomal stripe2, maternally inherited pale green and depressed striping; mitochondrial *nad4-nad7* fusion	118
NCS3			nonchromosomal stripe3, maternally inherited striations, distorted plants; mitochondrial	118
NCS4			nonchromosomal stripe4, maternally inherited defective striations in cms-S (RD) revertant strain; looks like *NCS3*	492
NCS5			nonchromosomal stripe5, maternally inherited stunted growth, yellow stripes, aborted kernels; mitochondrial cytochrome oxidase subunit 2 (*cox2*) alteration	493
NCS6			nonchromosomal stripe6, maternally inherited stunted growth, yellow stripes, aborted kernels; mitochondrial cytochrome oxidase subunit 2 (*cox2*) alteration	353
nec1	8.04	8.05	necrotic1, chlorotic seedling that stays rolled, wilts and dies	395
nec2	1.04		necrotic2, green seedling develops necrotic lesions at 2-3 leaf stage; lethal	10
nec3	5.04		necrotic3, seedling emerges with tightly rolled leaves that turn brown and die without unrolling; manually unrolled leaves are tan with dark brown crossbands	475
nec4	2.02	2.04	necrotic4, seedling yellow, leaf tips necrotic; lethal	269
nec5	4.06	4.11	necrotic5, pale green seedling becoming necrotic; dark brown exudate; lethal	458
nec6	5.04		necrotic6, like *nec3*	458
nec7	5.05	5.09	necrotic7, lighter green seedling becoming necrotic in crossbands	458
nl1	10.04		narrow leaf1, leaf blade narrow, some white streaks	171
nl2	5.03		narrow leaf2, dominant *Nl2*, leaves narrow and distorted (aka *Rgd2*)	465
nld1			narrow leaf dwarf1, small, compact plant with narrow, rolled leaves that are bleached pale green, especially along the midrib	488
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)	89, 239
nnr2			nitrate reductase2, partial cDNA from seedling root cDNA similar to nitrate reductase, encodes NAD(P)H:nitrate reductase	375
nnr3			nitrate reductase(NADH)3, scutellum cDNA, cyt b domain functional in *E. coli*, encodes nitrate reductase (NADH)	89
NOR	6.01		nucleolar organizer, nucleolus organizer: encodes ribosomal RNA's, encodes rRNA18S	408
ns1	3.00	3.04	narrow sheath1, plant brachytic; leaf sheath and blade taper, blade widens toward tip; husks narrowed, ears exposed	169
ns2	4.06	4.11	narrow sheath2, duplicate factor with *ns1*	595
nsf1			nicosulfuron susceptible1, recessive susceptibility to nicosulfuron (Accent) herbicide	318
ntm9			neurotoxin M9 homolog, leaf cDNA *csu129*, similar to scorpion neurotoxin M9, may encode sodium channel inhibitor	29
o1	4.07		opaque endosperm1, endosperm starch soft, opaque	171, 454, 632
o2	7.01		opaque endosperm2, like *o1*; high lysine content; regulates b-32 protein (see *rip1*); reduced lysine degradation (lysine-ketoglutaric reductase), encodes O2 protein	171, 454, 632

o5	7.02		opaque endosperm5, like *o1*; virescent to yellow or white seedlings	566
o7	10.06		opaque endosperm7, like *o1*; high lysine content	435
o8	2.04		opaque8, endosperm opaque, higher in lysine	241
o9			opaque endosperm9, crown opaque and light in color, frequently with a cavity; base or abgerminal side of kernel often corneous	456
o10			opaque endosperm10, like *o1*	456
o11			opaque endosperm11, thin, opaque, somewhat shrunken kernels with greyish cast	456
o13			opaque endosperm13, opaque, etched kernels with rim of corneous starch on abgerminal side	456
o14	6.02	6.08	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	466, 485
o15	7.02	7.06	opaque15, reduces 27kDa gamma zein mRNA and protein; germinates only with supplemented media	135
obf1	1.05		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	630
obf2	9.03		octopine synthase binding factor2, encodes protein with bZIP motif that binds to transcriptional enhancer sequences (*ocs*-elements), may encode octopine synthase binding factor	630
oec33	6.01		oxygen evolving complex, 33kDa subunit, cDNA identified by hybrid-selection, in vitro translation and immunoprecipitation with antisera against spinach OEC33 (aka *umc172*) encodes oxygen evolving complex, 33 kDa subunit	616
og1	10.02	10.03	old gold stripe1, dominant *Og1* plants have variable bright yellow stripes on leaf blade	371
olc1	1.06	1.12	oleic acid content1, high oleic acid in recessive (52% vs. 27% in B73; 35% in heterozygote)	736, 737
ole1			oleosin1, major protein from lipid bodies, cDNA clone, encodes oleosin, 16kDa	700
ole2			oleosin2, embyro lipid body protein, peptide, cDNA and genomic sequence, encodes oleosin, 18 kDa	543
ora2			orange endosperm2, color modified; plant vigor reduced in recessives, heterozygote advantage; mutagen-induced in Oh43	156
ora3			orange endosperm3, color modified; heterozygote advantage; induced by EMS in Oh43	157
oro1	6.00	6.05	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	396
oro2			orobanche2, like *oro1*	396
orom1			orobanche modifier1, dominant *Orom1* partially corrects chlorophyll loss in *oro1*	396
orp1	4.05		orange pericarp1, duplicate factor with *orp2*; pericarp orange over *orp1 orp2* kernels; lethal, tryptophan auxotroph, encodes tryptophan synthase B subunit	460, 735
orp2	10.04		orange pericarp2, duplicate factor with *orp1*, encodes tryptophan synthase B subunit	460
os1	2.00	2.04	opaque-endosperm, small germ1, opaque crown; kernel larger, lighter color; viable; reduced oil content	644, 645
oy1	10.02		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	189
P			plant color component at *R1*, anthocyanin pigmentation in seedling leaf tip, coleoptile, anthers, encodes myc protein P (of *r1*)	158, 658, 659
p1	1.03		pericarp color1, dominant *P1* confers red pigment in cob and pericarp; tissue-specific allele variations, encodes P1, 44kDa Myb homolog	172, 374
pal1			phenylalanine ammonia lyase1, leaf cDNA *csu156* similar to rice phenylalanine ammonia lyase, encodes phenylalanine ammonia lyase	321
pam1			plural abnormalities of meiosis1, desynchronized meiotic divisions and premeiotic mitosis; male sterile, incompletely female sterile	229
pam2			plural abnormalities of meiosis2, like *pam1*	226
pb4	6.02		piebald leaves4, like *pb1*	144
pbp1			promoter binding protein1, cDNA clones, Southwestern analysis supports binding to *pep1* promoter, light induced, encodes *pep1*-promoter binding protein	319
pck1			phosphoenolpyruvate carboxykinase homolog1, leaf cDNA *csu145*, low copy, similar to yeast phosphoenolpyruvate carboxykinase, may encode phosphoenolpyruvate carboxykinase	29
pd1			paired rows1, single vs. paired pistillate spikelets; quantitative, one of a family of loci differentiating maize vs. teosinte	350
pdc1	8.04		pyruvate decarboxylase1, cDNA and genomic clone, encodes pyruvate decarboxylase	323, 520
pdc2	8.02		pyruvate decarboxylase2, cDNA available; gene-specific probe, encodes pyruvate decarboxylase	520
pdc3	1.03		pyruvate decarboxylase3, cDNA sequence, gene-specific probe, encodes pyruvate decarboxylase	520
pdf1			thylakoid membrane polypeptide1, dominant *Pdf1* confers increase in electrophoretic mobility	437
pdi1			protein disulfide isomerase homolog, endosperm cDNA 5C05G07 similar to protein disulfide isomerase, may encode protein disulfide isomerase	264

pdk1			pyruvate, orthophosphate dikinase1, cDNA, genomic and peptide sequences; cytosolic or plastidic, dependent on transcript processing, encodes pyruvate, orthophosphate dikinase	223
pe1			perennialism1, vegetatively totipotent in combinations with *gt1* and *id1*	613, 614
pep1	9.03		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 data conflict, encodes phosphoenolpyruvate carboxylase	281, 288
pep4	7.02		phosphoenolpyruvate carboxylase4, cDNA for anaplerotic C3 isozyme; gene specific cDNA probe;, encodes phosphoenolpyruvate carboxylase	320
pet1	8.04	8.09	photosynthetic electron transport1, high leaf chlorophyll fluorescence, pale green, lacks cytochrome bf, reduced PSII at higher intensity light (aka *hcf121*)	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
pex1			pollen extensin-like1, clone specifies repeat motif ser-pro4, encodes hydroxyproline-rich glycoprotein	580
pg2	3.04		pale green2, seedling pale green, classification difficult; plants weak	142
pg11	6.02		pale green11, duplicate factor with *pg12*; seedling light yellowish green; mature plant pale and vigorous	557
pg12	9.03		pale green12, duplicate factor with *pg11*	557
pg13			pale green13, seedling light yellowish green; stunted growth	43, 623
pg15	1.00	1.05	pale green15, seedling light yellowish green; bleaches to near white in patches; lethal	458
pg16	1.06	1.12	pale green16, seedling light yellowish green	458
pgd1	6.01		6-phosphogluconate dehydrogenase1, electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur, encodes 6-phosphogluconate dehydrogenase	237
pgd2	3.05		6-phosphogluconate dehydrogenase2, electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur, encodes 6-phosphogluconate dehydrogenase	237
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	23, 496
pgl2			polygalacturonase2, cDNA and genomic clones, sequence nearly identical to *pgl1*; one of 10-12 member gene family, encodes exopolygalacturonase	23, 496
pgl3			polygalacturonase3, cDNA and genomic clones; one of 10-12 member gene family, encodes exopolygalacturonase	23, 496
pgl4			exopolygalacturonase4, cDNA sequence; distinct from others isolated from same inbred; one of 10-12 member gene family, encodes exopolygalacturonase	496
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	7
pgl7			exopolygalacturonase7, genomic clone, open reading framese nearly identical to *pgl1* but distinct 3' non-coding sequence, encodes exopolygalacturonase	23
pgl8			exopolygalacturonase8, genomic clone, open reading frame nearly identical to *pgl1* but distinct 3' non-coding sequence; mRNA product confirmed by PCR, encodes exopolygalacturonase	23
pgm1	1.09		phosphoglucomutase1, electrophoretic mobility; null allele is known; cytosolic; monomeric, encodes phosphoglucomutase (glucose-cofactor)	237
pgm2	5.02		phosphoglucomutase2, electrophoretic mobility; null allele is known; cytosolic; monomeric, encodes phosphoglucomutase (glucose-cofactor)	237
pgp1			P-glycoprotein homolog1, leaf cDNA *csu138* similar to *Arabidopsis* P-glycoprotein, may encode P-glycoprotein	30
ph1	4.01		pith abscission1, cob disarticulation; quantitative, one of a family of loci differentiating maize vs. teosinte	212
phi			Pioneer Hi-Bred International, designator for SSR loci	611
phi1	1.10		phosphohexose isomerase1, electrophoretic mobility; null allele is known; cytosolic; dimeric; intralocus hybrid bands occur. cDNA clone, encodes glucose-6-phosphate isomerase	237
pho1			phosphate regulatory homolog1, endosperm cDNA 5C04B08, similar to yeast *pho80* gene, may encode transcription factor	264
php1	10.04		chloroplast phosphoprotein1, isozyme; phosphorylated thylakoid protein, polymorphic in COxTx, encodes chloroplast phosphoprotein	86
phy1			phytochrome1, sequence, encodes phytochrome A	109
pi1			pistillate florets1, duplicate factor with *pi2*; secondary florets develop ("Country Gentlemen" or "Shoe Peg" expression) in *pi1 pi2* ears; quantitative character	282
pi2			pistillate florets2, duplicate factor with *pi1*	282
pki1			protein kinase inhibitor1, endosperm cDNA sequence, contains conserved Zn++ binding motif of yeast protein kinase C inhibitor, may encode protein kinase C inhibitor	629

pl1	6.04		purple plant1, *Pl1* plant tissues have light-independent pigment, *pl1* blue light-dependent; *Pl1-Bh1*, colored patches in *c1* aleurone and in plant; transcriptional activator for flavonoid genes, encodes myb protein of *Pl1*	178, 179
ploc1			plastocyanin homolog1, leaf cDNA *csu257*, similar to plastocyanin, may encode plastocyanin	31
plt1			phospholipid transfer protein homolog1, amino acid sequence, deduced from coleoptile cDNA, homologous to phospholipid transfer proteins, may encode phospholipid transfer protein	687
pm1	3.05		pale midrib1, midrib and adjacent tissue lighter green; reduced plant vigor	66
pmg1			phosphoglycerate mutase1, cDNA sequence corresponds to sequence of purified protein; genomic sequence; amino acid sequence similar to alkaline phosphatases (yeast, *E. coli*, human), encodes phosphoglycerate mutase, cofactor independent	242
pn1	7.06		papyrescent glumes1, dominant *Pn1* plants have long, thin papery glumes in ear and, less obviously, in tassel	208
po1	6.01		polymitotic1, (aka *ms4, ms6*) repeats 2nd meiotic division in male and female	34, 35
pop1			putative organelle permease1, endosperm cDNA 5C02F05 (uaz 282), similar to yeast putative mitochondrial carrier protein, may encode organellar permease	263
pox1			fowlpox viral protein homolog1, endosperm cDNA 5C04H07, similar to virus major core protein	264
ppg1	5.05	5.09	pale pale green1, light pale green seedling with white crossbands that become necrotic, spreading to the rest of the leaf causing lethality	458
ppi1			peptidyl-prolyl isomerase1, cDNA homologous to tomato peptidyl-prolyl cis-trans isomerase, may encode peptidyl-prolyl cis-trans isomerase	214
pr1	5.05		red aleurone1, changes purple aleurone to red; encodes flavonoid 3'-hydroxylase	165
prc1			proteasome C9 homolog1, endosperm cDNA 5C02A05 (*uaz237*), similar to proteasome subunit, may encode proteasome (endopeptidase) component C9	263
pre1			premature senescence1, senescence begins at least 2 weeks prior to anthesis, spreading from bottom to top of plant; occasionally sheds viable pollen	61
prem1			pollen retroelement maize1, retroelement-like, 10000 copies/haploid genome, pollen	695
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, PRF1	663
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, PRF2	663
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, PRF3	663
prg1	5.05	5.06	pitted rough germless1, small pitted rough endosperm, usually germless; seed with larger embryo will produce small striated seedlings	596
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	640
prh2			protein phosphatase homolog2, endosperm cDNA 5C04B06 (*uaz244*), similar to human cell transforming protein, may encode protein phosphatase	263
prl1			protease PrlC candidate1, endosperm cDNA 2C01C07, similar to bacterial oligopeptidase A, may encode oligopeptidase	264
pro1	8.04		proline responding1, (allele *o6*) crumpled opaque kernel; green-striped lethal seedling; responds to proline in culture	215
prp1			pathogenesis-related protein1, cDNA clone, single copy, deduced protein product is basic (vs. acidic), normally accumulates during germination,induced by fungal elicitors but not wounding, encodes PRP1	93
prp2	2.04		pathogenesis-related protein homolog2, leaf cDNA *csu133*, single copy, 5'-sequence similar to kidney bean pathogenesis-related protein, PIR S14730, may encode pathogenesis-related protein	29
prr1			putidaredoxin reductase homolog1, endosperm cDNA 5C04E05 (*uaz204*), similar to NADH-putidaredoxin reductase, may encode putidaredoxin reductase	264
ps1	5.04		pink scutellum1, (aka *vp7*, *lyc1*) some alleles viviparous; endosperm and scutellum pink, seedling white with pink flush	650
psa1			photosystemI1, lacks photosystem I core complex polypeptides	25, 497
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, leaf cDNA csu18, similar to barley photosystem I subunit, encodes PSI, subunit N	30
psa6			photosystem I reaction center6, leaf cDNA csu67, similar to barley photosystem I subunit, encodes photosystem I, subunit K	30
psb1	6.01	6.08	photosystem II1, (was *hcf134*) lacks PSII core complex; pale seedling; mutable (*Mu*-induced)	25, 704
psb2	5.05	5.09	photosystemII2, lacks photosystem II core complex polypeptides; *Mu*-induced	25
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	452

ptd1	1.09		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	597, 598
ptd2	7.05	7.06	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	597, 598
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), encodes receptor-like serine/threonine protein kinase	707
ptk2			protein kinase homolog2, endosperm cDNA 5C02A07, similar to *Arabidopsis* tyr-ser-thr protein kinase, may encode tyr-ser-thr protein kinase	264
px1	2.05	2.10	peroxidase1, (was *prx1*) electrophoretic mobility; null alleles occur; monomeric; found in leaf and elongating tissues but not in anther, embryo, endosperm, anthers, pollen, encodes peroxidase	253, 254
px2			peroxidase2, electrophoretic mobility; monomeric, pollen specific, encodes peroxidase	384
px3	7.05	7.06	peroxidase3, electrophoretic mobility; monomeric, encodes peroxidase	384
px4			peroxidase4, electrophoretic mobility; null allele is known; monomeric, encodes peroxidase	384
px5			peroxidase5, presence-absence, encodes peroxidase	384
px6			peroxidase6, presence-absence, encodes peroxidase	384
px7			peroxidase7, electrophoretic mobility; null allele is known; monomeric, encodes peroxidase	384
px8			peroxidase8, electrophoretic mobility; monomeric, encodes peroxidase	56, 57
px9			peroxidase9, electrophoretic mobility; null allele is known; monomeric, encodes peroxidase	56, 57
py1	6.07		pigmy plant1, leaves short, pointed; fine white streaks	680
py2	1.11	1.12	pigmy plant2, like *py1*	458
pyd1	9.00		pale yellow deficiency1, seedling yellow; deficiency for short terminal segment of chromosome arm, lethal; complements *yg2* and *v28* but not *wd1*	409
r1	10.06		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 myc protein of S (of *r1*)	165
ra1	7.02		ramosa1, ear and tassel many-branched; tassel branches taper to tip	37, 220
ra2	3.02	3.03	ramosa2, tassel many-branched, upright, not conical like *ra1*; irregular kernel placement	67, 171, 494
ra3	4.00	4.11	ramosa3, branched inflorescence	519
rab28			abscisic acid-responsive28, cDNA and genomic clones, inducible by ABA in embryos and young leaves; induced by water-stress in leaves; similar to cotton LeaD-34	529
rab30	1.01	1.02	responsive to abscisic acid30, cDNA elicited by ABA, encodes abscisic acid responsive protein30	529
rap1			retinoblastoma-associated protein homolog1, endosperm cDNA 5C01B09, similar to human retinoblastoma-associated protein, may encode cell cycle protein	264
rBg			receptor of *Bg*, responds to *Bg*	587
rcm1	7.02		rectifier1, dominant *Rcm1* restores miniature seed of teosinte cytoplasm to normal	6
rcm2			rectifier2, dominant *Rcm2* weakly restores miniature seed of teosinte cytoplasm to normal	6
rcm3			rectifier3, dominant *Rcm3* restores miniature seed of teosinte cytoplasm to normal; from *Z. diploperennis*	6
rcu			receptor of *Fcu*, responds to *Fcu*	234
rcy:Mu7			Receptor of *Cy*, first described for *bz1* allele, *bz1-rcy*	601
rd1	1.11		reduced plant1, semi-dwarf plant; possible allelism with *bv2*	451
rd2	6.00	6.08	reduced plant2, like *rd1*, but not as extreme	225
rd3	3.06		reduced plant3, like *rd1*; anthocyanin interactions	401
rDNA5S	2.08		5S ribosomal RNA, cluster consisting of several thousand repeated genes, encodes rRNA5S	664, 746
rDNA5.8S	6.00	6.01	5.8S ribosomal RNA, NOR (nucleolus organizer) component, encodes rRNA5.8S	314
rDNA18S	6.00	6.01	18S ribosomal RNA, NOR (nucleolus organizer) component, encodes rRNA18S	314
rDNA25S	6.00	6.01	25S ribosomal RNA, NOR (nucleolar organizer) component, encodes rRNA25S	314
rDt			receptor of Dotted, designator for nonautonomous transposable element found in certain alleles at *a1*, excised by action of *Dt1*, etc.	646
rea1	3.06		red embryonic axis1, red embryonic axis, wilted seedling, some vivipary	218
ref1	3.04		reduced floury endosperm1, small, reduced endosperm with dull, floury appearance; low frequency of expression; approx. 20% will germinate	596
ren1	5.06	5.07	reduced endosperm1, small seed with reduced, opaque endosperm, usually lethal; seed with larger embryos produce fertile plants	597, 598
ren2	7.04		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	290, 597, 598
ren3	10.07		reduced endosperm3, reduced endosperm, partially filled to empty pericarp; small germ or germless; larger seed produce fertile plants	598
rf1	3.04		restorer of fertility1, dominant *Rf1* restores fertility to cms-T; complementary to *Rf2*	309
rf2	9.04		restorer of fertility2, see *rf1*, may encode aldehyde dehydrogenase	162, 164
rf3	2.09		restorer of fertility3, dominant *Rf3* restores fertility to cms-S	74
rf4	8.00		restorer of fertility4, dominant *Rf4* restores fertility to cms-C; complementary with *Rf5* and *Rf6*	240

rf5			restorer of fertility5, dominant *Rf5* restores fertility to cms-C; complementary with *Rf4* and *Rf6*	312, 701
rf6			restorer of fertility6, dominant *Rf6* restores fertility to cms-C; complementary with *Rf4* and *Rf5*	312, 701
rf7			restorer of fertility7, dominant *Rf7* partially restores fertility to cms-Y	542
rfa1			replication factor A homolog1, endosperm cDNA 5C09D05, similar to yeast DNA binding protein, may encode replication factor-A	264
rfz1			rat frizzled homolog1, endosperm cDNA 5C02A04, similar to rat homologue of *Drosophila* polarity gene frizzled, may encode tissue polarity protein	264
rg1	3.04		ragged leaves1, dominant *Rg1* plants develop defective tissue between veins of older leaves, causing holes and tearing	64
rgd1	6.01		ragged seedling1, seedling leaves narrow, thread-like, have difficulty in emerging	336
rgh1	8.09		rough kernel1, small floury kernel with rough and pitted surface and nonviable embryos	486
rgo1			reversed germ orientation1, embryo faces base of ear; variable frequency, maternal trait	583
rhm1	6.00	6.01	resistance to *Helminthosporium maydis*1, chlorotic-lesion reaction with *Cochliobolus heterostrophus* (=*Bipolaris maydis*, *H. maydis*) race O	636
rhm2	6.00	6.01	resistance to *Bipolaris maydis*2, double recessive with *rhm1* resistant	97, 98
ri1	4.01		rind abscission1, dominant *Ri1* plants have cob disarticulation; quantitative, one of a family of loci differentiating maize vs. teosinte	212
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	8.04		ribosome-inactivating protein1, electrophoretic mobility, abundant 32kD endosperm protein (b32 protein), cytosolic, inactivates rabbit (not maize or wheat) ribosomes, encodes ribosome inactivating protein	215, 708
ris1	5.04		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	9.02	9.02	rindless culm1, upper internodes lack rind in longitudinal bands	107
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*	49
rli1			rough lineate1, lineate-like streaks of protruding tissue on leaf blade which produce a rough texture	472
rMrh			Receptor of *Mrh*, responds to *Mrh*	553
rMut			receptor of *Mut*, responds to *Mut*	553
rMx			Responder to *Mx*, non-autonomous element responding to *Mx*	442
rnp1			chloroplast RNA binding protein1, leaf cDNA csu17, similar to RNA binding proteins, encodes chloroplast RNA binding protein	29
rp1	10.01		resistance to *Puccinia sorghi*1, dominant *Rp1* resistant	386, 387
rp3	3.04		resistance to *Puccinia sorghi*3, dominant *Rp3* resistant	275, 728
rp4	4.01		resistance to *Puccinia sorghi*4, dominant *Rp4* resistant	251, 728
rp5	10.01		resistance to *Puccinia sorghi*5, dominant *Rp5* resistant	251, 590
rp6	10.01		resistance to *Puccinia sorghi*6, dominant *Rp6* resistant	251, 728
rp7			resistance to *Puccinia sorghi*7, dominant *Rp7* resistant	283
rpa2			acidic ribosomal protein2, endosperm cDNA 5C05D03, similar to acidic ribosomal protein, may encode 60S ribosomal protein P2, cytoplasmic	264
rph1			RNase PH homolog, endosperm cDNA 5C06F11 similar to *E. coli* tRNA processing enzyme, may encode RNase PH	264
rpl5			ribosomal protein L5 homolog, endosperm cDNA 5C05D11 (*uaz189*), similar to ribosomal protein, encodes ribosomal protein L5	263
rpl7			ribosomal protein L7, leaf cDNA *csu201*, similar to ribosomal protein, may encode 60S ribosomal protein L7	264
rpl10			ribosomal protein L10 homolog, endosperm cDNA 5C01D03 (*uaz198*), similar to yeast acidic ribosomal protein, may encode ribosomal protein L10e	264
rpl12			ribosomal protein L12, endosperm cDNA 5C05D11 similar to mammalian and yeast ribosomal protein, may encode 60S ribosomal protein L12, cytoplasmic	264
rpl13			ribosomal protein L13A homolog, endosperm cDNA 5C03G09, similar to rodent ribosomal protein, may encode 60S ribosomal protein L13A	321
rpl17			ribosomal protein L17, endosperm cDNA 5C05G01, similar to barley ribosomal protein, encodes 60S ribosomal protein L17	264
rpl19			ribosomal protein L19 homolog, leaf cDNA csu36, similar to rat ribosomal protein, low copy number, may encode ribosomal protein L19	263
rpl30			ribosomal protein L30, endosperm cDNA 5C02F01 similar to yeast ribosomal protein gene, may encode 60S ribosomal protein L30, cytoplasmic	31

rpo1			RNA polymerase II homolog1, leaf cDNA *csu150*, similar to yeast RBP2, RNA polymerase, may encode RNA polymerase	321
rpp9	10.01		resistance to *Puccinia polysora* and *Puccinia sorghi*9, dominant *Rpp9* resistant	698
rps2			ribosomal proteinS2 mitochondrial homolog, leaf cDNA *csu191* similar to Marchantia mitochondrial ribosomal protein, may encode ribosomal protein S2, mitochondrial	264
rps5			ribosomal protein S5, endosperm cDNA 5C06A11 similar to ribosomal protein, may encode 40S ribosomal protein S5, cytoplasmic	321
rps8			ribosomal protein S8 homolog, multiple copies; leaf cDNA csu34 similar to ribosomal protein, encodes 40S ribosomal protein S8, cytoplasmic	264
rps11	10.06		ribosomal protein S11, cDNA sequenced; homology to *rps11*; two bands hybridize in Southerns, encodes ribosomal protein S11, cytoplasmic	31
rps22			ribosomal protein S22 homolog, multiple copies; leaf cDNA csu28 similar to *Xenopus* ribosomal protein, may encode ribosomal protein S22	321
rps24			ribosomal protein S24, endosperm cDNA 5C01E10, similar to animal ribosomal protein, may encode 40S ribosomal protein S24, cytoplasmic	356
rs1	7.00		rough sheath1, shoot meristem and developing stem specific; dominant *Rs1* plants have extreme ligule disorganization (aka *kn2*)	333
rs2	1.05		rough sheath2, short, zigzag plants with warty, distorted sheaths and leaves	333
rs4	7.02		rough sheath4, dominant *Rs4* plants have rough leaf sheaths; vascular bundles enlarged	481
rt1	3.04		rootless1, secondary roots few or absent	296
rth1	1.06	1.12	roothair defective1, roothairs do not elongate in *rth1* homozygotes; plants are dwarfed and appear nutrient deficient	716, 717
rth2	5.05	5.09	roothair defective2, root hairs elongate 1/5-1/4 of normal; plants are vigorous and show little nutritional deficiency	716, 717
rth3	1.02	1.05	roothair defective3, like *rth1*, but "stocking cap" roothair initials under electron microscope	716, 717
ruq			receptor of *Uq*, element mediated by *Uq*	205
S	10.06		seed color component at *R1*, anthocyanin pigmentation in aleurone; (see also cms-S), encodes myc protein of S (of *r1*)	158, 658
sad1	10.02		shikimate dehydrogenase1, electrophoretic mobility; plastidial; monomeric, encodes shikimate dehydrogenase	718
sar1			SAR homolog1, endosperm cDNA 5C03G12 (*uaz151*), similar to *Arabidopsis sar1* homologue, encodes GTP-binding protein, *SAR1* homolog	263
sbd1	6.01	6.08	sunburned1, sun-exposed leaves greyish-waxy	482
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	195
sci1			subtilisin-chymotrypsin inhibitor homolog1, cDNA from germinating embryo infected with *Fusarium moniliforme*; similar to subtilisin-chymotrypsin inhibitor, may encode subtilisin-chymotrypsin inhibitor	130
sdh1			sorbitol dehydrogenase homolog1, endosperm cDNA 5C04A01 (*uaz152*), similar to sorbitol dehydrogenases, encodes sorbitol dehydrogenase	263
sdw1	8.04		semi-dwarf plant1, dominant *Sdw1* plants have shortened internodes, erect leaves and tassel branches	50
sdw2	3.00	3.09	semi-dwarf2, dominant *Sdw2*, short plant, 1/3-1/2 normal height, with normal green erect leaves; does not respond to gibberellins; no anthers in ear	484
se1			sugary-enhancer1, high sugar content with *su1*; light yellow endosperm; freely wrinkled in Ill677a	192
sen1	3.00	3.09	soft endosperm1, duplicate factor with *sen2*; endosperm soft, opaque	667
sen2	7.00	7.06	soft endosperm2, duplicate factor with *sen1*	667
sen3	1.00	1.12	soft endosperm3, duplicate factor with *sen4*; like *sen1*	667
sen4			soft endosperm4, duplicate factor with *sen3*	667
sen5	2.00	2.10	soft endosperm5, duplicate factor with *sen6*; like *sen1*	667
sen6	5.00	5.09	soft endosperm6, duplicate factor with *sen5*	667
sfb1			SF1 binding protein candidate1, endosperm cDNA 5C09C08 similar to spinach transcription factor, may encode DNA binding protein S1FA	264
sft1			small flint type1, ears on *sft1* plants produce only small flint endosperms; +/sft ears are normal	646
sg1			string cob1, dominant *Sg1* plants have reduced pedicels	210
sh1	9.01		shrunken1, inflated endosperm collapses on drying, forming smoothly indented kernels; sucrose synthase-1 of endosperm (compare *sus2*); homotetramer, encodes sucrose synthase	285
sh2	3.09		shrunken2, inflated, transparent, sweet kernels collapse on drying, becoming angular and brittle; endosperm ADPG pyrophosphorylase subunit (compare *bt2*); application: "supersweet" sweet corn - kernels sugar-sweet for long period, encodes ADP glucose pyrophosphorylase, 60kDa subunit	388
sh4	5.05		shrunken4, collapsed, chalky endosperm	692
sh6	7.00	7.02	shrunken pale green6, shrunken, opaque, normal size kernel, like *sh1*; pale green virescent seedling that greens slowly and is usually lethal	485, 673
sht1			susceptible to *H. turcicum*1, *Sht1* is a dominant inhibitor of *Ht2*, *Ht3*, and *Htn1* (but not of *Ht1*) in inbred lines related to B14	95, 628

si1	6.02		silky1, (aka *ts8, ms-si*) multiple silks in ear; sterile tassel with silks	201
sin1			sin homolog1, endosperm cDNA 5C01E06, similar to yeast SIN3, may encode transcription factor	264
sip1			stress-induced protein1, cDNA sequence homologous to thaumatin-like protein, encodes thaumatin-like protein	204
sk1	2.03	2.04	silkless ears1, pistils abort, no silks	308
sks1	2.04	2.05	suppressor of KYS sterility1, pollen grains developing in presence of dominant *Ms21* are defective and nonfunctional if *sks1*, normal if *Sks1*	358, 604
sl1	7.03		slashed leaves1, leaves slit longitudinally by necrotic streaks. Plants are weaker than normal, but produce pollen and ears.	259
sm1	6.04	6.05	salmon silks1, silks salmon color with *P1-RR*, brown in *P1-WW*	9
sn1	10.06		scutellar node color1, anthocyanin in coleoptile, nodes, auricle, leaf blade, etc. (compare *Lc1*), encodes SN1	216, 217
sod1			superoxide dismutase1, electrophoretic mobility; plastidial; Cu-Zn dimeric; intralocus hybrid bands occur, encodes superoxide dismutase	28
sod2	7.05		superoxide dismutase2, electrophoretic mobility; cytosolic; Cu-Zn dimeric, encodes superoxide dismutase	91, 92
sod3			superoxide dismutase3, electrophoretic mobility; mitochondrial; Mn tetrameric; intralocus hybrid bands occur; cDNA complements yeast mutant;, encodes superoxide dismutase	28
sod4	1.03		superoxide dismutase4, electrophoretic mobility; cytosolic; Cu-Zn dimeric; intralocus hybrid bands occur; two similar sequences X17564 (sod4), X17565(sod4A), encodes superoxide dismutase	28
sos1	4.02		Suppressor of sessile spikelets1, dominant *Sos1* suppresses sessile spikelets in ear primordia and in tassel, reduced tassel branching. Contrasts to teosinte ears, where the pedicellate, not the sessile spikelet is lacking	154
spc1	3.06		speckled1, dominant *Spc1* plants display brown speckling on leaves and sheath at flowering; supporting tissues weak	464, 465
spc2	1.06	1.12	speckled2, green seedling with light green speckles	458
spc3	3.05	3.09	speckled3, green seedling with dark and light green speckles	458
Spm			Suppressor-mutator, autonomous transposable element (equivalent *En*); regulates dSpm (=I) transposition and function at *a1-m1*, *a1-m2*, *bz1-m13*, etc.; encodes TnpA, its positive and negative regulator and elicitor of transposition	412, 413
sps1	8.06		sucrose phosphate synthase1, cDNA encodes a 1068 amino acid leaf protein; transgenic (*E. coli*) directs sucrose phosphate synthesis, encodes sucrose-phosphate synthase	734
spt1	2.05	2.10	spotted1, pale green, weak seedlings with dark green spots	458, 473
spt2	4.00	4.05	spotted2, like *spt1*; plants very weak to inviable.	458
sr1	1.02		striate leaves1, many white striations or stripes on leaves	72, 171
sr2	10.07		striate leaves2, white stripes on blade and sheath of upper leaves	300
sr3	10.01		striate leaves3, virescent seedling and striate to striped plant	224
sr4	6.00	6.08	striate leaves4, seedlings pale luteus, later leaves white-striped	480
srp1			signal recognition particle RNA, gene family, encodes 7SL RNA	90
ssu1	4.08		ribulose bisphosphate carboxylase small subunit1, probed locus, may encode ribulose-1,5-bisphosphate carboxylase, small subunit	70, 713, 740
ssu2	2.05		ribulose bisphosphate carboxylase small subunit2, probed locus, may encode ribulose-1,5-bisphosphate carboxylase, small subunit	70, 713, 740
st1	4.04		sticky chromosome1, small plant, striate leaves, pitted kernels resulting from sticky chromosomes; *st1-e* heightened by high temperature	38
stAc	10.04		stabilized Activator, RFLP locus; name from P. Chomet(unpublished)	85
stm1			stolon tip maize homolog1, leaf cDNA csu6, similar to potato stolon tip protein sequence, may encode S-adenosylmethione decarboxylase	29
Stowaway			Stowaway, like Tourist, a small inverted repeat element but with distinct sequence, associated with coding sequence of both monocots and dicots	76
stp1	8.03		sugar transport homolog1, leaf cDNA *csu142* similar to yeast plasma membrane sugar transport protein, may encode sugar transport protein	29
su1	4.05		sugary1, endosperm wrinkled and translucent when dry; starch debranching enzyme I absent in developing endosperm, phytoglycogen but no debranching enzyme in germinating seeds; *su1-am* sugary-amylaceous; *su1-st* recessive starchy; application: the "sweet corn" gene - recessive kernels are creamy-sweet at milk stage, encodes starch debranching enzyme I	131
su2	6.04	6.05	sugary2, endosperm glassy, translucent, sometimes wrinkled, may encode starch branching enzyme	190
su3	4.05		sugary3, endosperm glassy, smoother than *su1*	672
sup1			suppressor1, dominant *Sup1* modifies *o2* kernels to semi-transparent	397
sur1			sulfonylurea herbicide response1, recessive sensitivity to sulfonylurea herbicides	243
sus1	9.04		sucrose synthase2, (was *sus2*, *css1*) sucrose synthase-2 of embryo and other tissues, compare *sh1*; location on 9L confirmed by B-A translocations, encodes sucrose synthase	403
sut1			sucrose transport1, anthocyanin accumulates in a non-clonal pattern at tip and margins of leaves soon after emergence from whorl; reduced plant height	61

sy1			yellow scutellum1, tissue yellow in recessive rather than white	650
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*	384
tan1	6.04	6.08	tangled1, alters patterns of cell growth, division and differentiation throughout the plant; irregular cell shapes	639
tasBF266			telomere associated sequence BF266, subtelomeric sequence; multiple copies include non-telomere locations	87
tasBF267			telomere associated sequence BF267, subtelomeric sequence, muliple copies including non-telomeric	87
tasBF268			telomere associated sequence BF268, subtelomeric sequence, multiple copies, including non-telomeric	87
tau1			tau protein homolog1, leaf cDNA csu64 similar to brain specific 14-3-3 protein, tau chain A., may encode activator of tyrosine and tryptophan hydroxylases	100, 321
tb1	1.09		teosinte branched1, many tillers; ear branches tassel-like	83
tbp1	1.09		TATA-binding protein1, Maize TATA-binding protein component of TFIID transcription initiation factor, encodes TATA box binding protein	249, 705
tbp2	5.02	5.03	TATA-binding protein2, cDNA clone; encodes a TBP that functions in yeast; maps to chromosome 5 near *pgm2*, encodes TATA box binding protein	249
td1	5.04		thick tassel dwarf1, plants shortened, tassel dense	13
te1	3.05		terminal ear1, stalked ear appendages at tip; varying to infolded ears, encodes TE1	400
tga1	4.05		teosinte glume architecture1, glumes indurated, erect, long, boat-shaped; factor transferred from teosinte	153
tgd1			dTDP-glucose dehydratase homolog1, leaf cDNA *csu219*, 5' sequence similar to Streptomyces dTDP-glucose dehydratase, may encode dTDP-glucose 4,5-deydratase	31
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	524
thp1	7.04		thiol protease homolog1, leaf cDNA csu5 similar to *Vigna mungo* sulfhydryl-endopeptidase; single copy, may encode thiol protease	100, 321
tiny fragment 9	9.00	9.02	centric fragment that carries *Sh1*, *Bz1*, and X component	415
tl			tasselless1, trait was previously symbolized as a gene, *tl1*, but inheritance is complex and irregular; associated with aneuploidy	425
tlr1	1.06	1.12	tillered1, semi-dominant, *Tlr1/Tlr1* plants show extreme tillering; *Tlr1/tlr1* show some tillering.	471
tlr2			tillering2, dominant *Tlr2* plants show 2-3 tillers per plant	316
tls1	1.09	1.12	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			Tourist, family of transposable elements, 1-50k copies in genome, average length 133 bp	75, 745
tp1	7.03		teopod1, dominant *Tp1* plants have many tillers, narrow leaves, many small partially podded ears, tassel simple	370
tp2	10.04	10.05	teopod2, like *tp1*	521
tp3	3.03		teopod3, semi-dominant that increases tillering and decreases number of lateral tassel branches; originally identified by J. Beckett, may be allele of *Cg1*	41
tpase			transposase of *Ac*, required for transposition of *Ac*, encodes TPASE, putative *Ac* transposase	340, 341
tpe1			thin pericarp1, reduced cell number in pericarp (from Coroica)	213
tpi1	7.03		triose phosphate isomerase1, electrophoretic mobility; plastidial; dimeric; intra/interlocus hybrids occur with *Tpi2*, encodes triose phosphate isomerase (plastidial)	722
tpi2	2.06		triose phosphate isomerase2, electrophoretic mobility; plastidial; dimeric; intra/interlocus hybrids occur with *Tpi1*, encodes triose phosphate isomerase (plastidial)	722
tpi3	8.02		triose phosphate isomerase3, electrophoretic mobility; cytosolic; dimeric; intra/interlocus hybrids occur with *Tpi4* & *Tpi5*, encodes triose phosphate isomerase (cytosolic)	722
tpi4	3.03		triose phosphate isomerase4, electrophoretic mobility; cytosolic; dimeric; intra/interlocus hybrids occur with *Tpi3* & *Tpi5*, encodes triose phosphate isomerase (cytosolic)	719, 722
tpi5	8.07	8.08	triose phosphate isomerase5, electrophoretic mobility; cytosolic; dimeric intra/interlocus hybrids occur with *Tpi3* & *Tpi4*, encodes triose phosphate isomerase (cytosolic)	722
tpk1			tousled protein kinase1, endosperm cDNA 5C04A03 (*uaz130*),similar to *Arabidopsis* protein kinase, TOUSLED, encodes Lea Group 3 proteinMLG3	263
tpm1			thylakoid peptide modifier1, dominant decrease in electrophoretic mobility	436
tpt1			triose phosphate translocator1, cDNA sequence similar to plant triosephospate/phosphate translocators, encodes triose phosphate/phosphate translocator	194
tr1	2.02	2.03	two-ranked ear1, distichous vs. decussate phyllotaxy in ear axis; quantitative, one of a family of loci differentiating maize vs. teosinte (cp. RANK of Doebley)	350

trAc9705			transposed Activator sequence, probed sequence in Burr et al. (MNL 65:109), location on 1S from data of Diane Burgess, DNAP	86
trg1	4.00		trigonelline1, recesssive deficiency in trigonelline and in SAM:nicotinic acid N methyl transferase (Rhodes, D), may encode SAM:nicotinic acid N methyl transferase	402
trh1			thioredoxin H homolog1, endosperm cDNA 5c05b02 similar to plant and yeast thioredoxin, may encode thioredoxin H	264
trisomic			normal chromosome complement plus an additional chromosome	404
trn1	9.03	9.04	torn1, dominant Trn1 plants have chlorotic and adherent leaf tissues on later leaves, which become green and healthy after sunlight exposure but are torn	487
tru1			tassels replace upper ears1, upper ear branches tassel-like, tillers bear ears	621
ts1	2.04	2.05	tassel seed1, tassel pistillate and pendant; if removed, small ear with irregular kernel placement develops	177
ts2	1.03		tassel seed2, like *ts1*, but tassel branches variably pistillate and staminate; sequence homologous to short chain alcohol dehydrogenases, encodes TS2 protein	177
ts3	1.09		tassel seed3, dominant *Ts3* tassel has large sections of either pistillate or staminate flowers in tandem; some pollen	526
ts4	3.04	3.05	tassel seed4, tassel compact silky mass, upright, with pistillate and staminate florets; ear silky and proliferated; possibly = *zag2*	526
ts5	4.03		tassel seed5, dominant *Ts5* tassels are upright with scattered, short silks; branches mostly pistillate toward the base	182
ts6	1.11		tassel seed6, dominant *Ts6* tassels are pistillate to mixed, compact; ear with irregular kernel placement	494
tsc1			tar spot complex1, dominant *Tsc1* confers resistance to tar spot complex	94
tsh1			tassel sheath1, leaves develop at base of tassel branches and spikelet pairs	60
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	124, 125
tua1	1.10		alpha tubulin1, mRNA expressed primarily in root tips and pollen; member of tandem repeat (see *tua2*), encodes alpha tubulin	439
tua2	1.10		alpha tubulin2, member of tandem repeat (see *tua1*), separated by 1.5 kbp, near *adh1*, preferentially expressed in radicles, root tips and coleoptiles; encodes alpha tubulin	439, 702
tua3	5.01		alpha tubulin3, alpha tubulin family; mRNA expressed in all dividing cells examined, encodes alpha tubulin	440
tua4			alpha tubulin4, belongs to alpha tubulin subfamily I, with *tua1* and *tua2*; gene specific cDNA probe, encodes alpha tubulin	702
tua5	2.08		alpha tubulin5, alpha tubulin subfamily II with *tua6*; gene specific cDNA probe, encodes alpha tubulin	702
tua6			alpha tubulin6, alpha tubulin subfamily II, gene specific cDNA probe, encodes alpha tubulin	702
tub1	1.01		beta tubulin1, genomic clones sequenced; gene-specific probe (by Southern blot) hybridizes to a single transcript size, encodes beta tubulin	284
tub2	8.04		beta tubulin2, cDNA sequenced; single copy (Southern blots), encodes beta tubulin	284
tub3			beta tubulin3, cDNA sequence, gene specific probe (Southern blots, sequence), encodes beta tubulin	575
tub4	5.03		beta tubulin4, cDNA sequence, gene specific probe (Southerns, sequence), encodes beta tubulin	575
tub5			beta tubulin5, cDNA sequence, gene specific probe (Southern blots, sequence), encodes beta tubulin	575
Ty1			Ty1-copia group retrotransposon candidate, partial genomic sequence homologous to reverse transcriptase, may encode reverse transcriptase	266
U5snRNA			U5snRNA, identity based on homology to *Arabidopsis* clones; genomic clones have distinct 5'-sequence, encodes U5snRNA	355
ub1			unbranched1, tassel with one spike	469
ubf9			ubiquitin fusion protein9, genomic sequence, hybridizing mRNA expressed during cell division and/or cell growth; multiple copies in genome, encodes ribosomal protein 27A, cytoplasmic	106
ubi1			ubiquitin1, genomic sequence, 7 contiguous direct ubiquitin repeats; transcript specific probe; promoter active in monocots, not in tobacco, encodes polyubiquitin	110
ubi2			ubiquitin2, genomic sequence encodes 7 contiguous ubiquitin monomers; transcript specific probe, encodes polyubiquitin	110
uce1			ubiquitin conjugating enzyme1, endosperm cDNA 2C06C11 (*uaz102*), similar to plant ubiquitin conjugating enzymes, encodes ubiquitin conjugating enzyme	263
ufo1	10.01	0.01	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	678
ugp1			UDP-glucose pyrophosphorylase1, endosperm cDNA 5C02H07 (*uaz194*), similar to potato UDP-glucose pyrophosphorylase, encodes UDP-glucose pyrophosphorylase	263
ugu1			UTP-glucoseP uridyltranferase homolog, endosperm cDNA 5C04E10, similar to slime mold UTP--glucose-1-P uridylyltransferase, may encode UTP-- glucose-1-phosphate uridyltransferase	264

Uq1			Ubiquitous, controlling element mediating *a1-ruq*; *ruq-st*, *ruq31*, and *ruq66* are receptor elements	205
Uq2			Ubiquitous2, one of 5 newly activated *Uq* elements unlinked to *Uq1*	512
Uq3			Ubiquitous3, one of five newly activated *Uq* elements not linked to *Uq1*	512
Uq4			Ubiquitous4, one of five newly activated *Uq* elements unlinked to *Uq1*	512
Uq5			Ubiquitous5, one of five newly activated *Uq* elements not linked to *Uq1*	512
Uq6			Ubiquitous6, one of five newly activated *Uq* elements not linked to *Uq1*	512
uro1			urmodulin homolog1, endosperm cDNA 5C06D10 similar to rodent uromodulin, may encode transcription factor	264
v1	9.03		virescent1, yellowish white seedling, greens rapidly; low temperature accentuates	141
v2	5.08	5.09	virescent2, like *v1*, but greens slowly; low temperature accentuates	175
v3	5.04		virescent3, light yellow seedling, greens rapidly; low temperature accentuates	141
v4	2.05		virescent4, like *v2*	141
v5	7.02		virescent5, like *v1*, but older leaves have white stripes	141
v8	4.06	4.07	virescent8, like *v2*; lethal	143
v12	5.05	5.06	virescent12, like *v3*	527
v13			virescent13, first leaf with green tip; greens slowly	527
v16	8.07		virescent16, like *v2*; deficiency of chloroplastic 16S and 23S rRNA	527
v17	4.00	4.11	virescent17, like *v1*, but greening from base to tip	527
v18	10.04	10.07	virescent18, like *v1*	527
v21	8.06	8.07	virescent21, grainy virescent, greening from tips and margins inward	39
v22	1.08		virescent22, like *v1*	13
v23	4.05		virescent23, like *v1*	13
v24	2.05	2.10	virescent24, like *v1*, except not cold-sensitive and developmentally conditional high chlorophyll fluorescence attributable to premature assembly of the light harvesting complexes.	458
v25			virescent25, greenish white seedling; greens from base upward	458
v26	2.00	2.04	virescent26, yellowish white seedling with green leaf tip and midrib	458
v27	7.02	7.06	virescent27, virescent with many small yellow green streaks; husks and culm whitish at flowering	458
v28	9.00		virescent28, like *v1*, greens slowly	458
v29	10.04	10.07	virescent29, Greens rapidly in longitudinal streaks on leaf blade, changing to green with light green streaks. Expression heightened by cool temperatures (20-25C).	458
v30	9.05		virescent30, like *v1*, but more yellow	119
v31	9.02		virescent31, grainy, light green seedling; small green plant with longitudinal white stripes	256
va1	7.03		variable sterile1, variable male and female fertility; cytokinesis fails in anaphase I	36
vg1	1.07		vestigial glume1, dominant *Vg1* glumes very small, cob and anthers exposed; upper leaves have scant ligules	653
vp1	3.05		viviparous1, embryo fails to become dormant, viable if transplanted; some alleles dormant; chlorophyll and carotenoids unaffected; anthocyanins in aleurone suppressed (aka *vp4*), encodes VP1, dormancy regulatory protein	187, 368, 389
vp2	5.04		viviparous2, embryo fails to become dormant; white endosperm, white seedling; anthocyanins unaffected, encodes phytoene C-11,12 desaturase, subunit A	184, 187
vp5	1.02		viviparous5, like *vp2*, encodes phytoene C-11,12 desaturase, subunit B	563
vp8	1.11		viviparous8, embryo fails to become dormant; chlorophyll and carotenoids unaffected; small, pointed-leaf seedlings	564
vp9	7.02		viviparous9, (was *y7*, *z1*) like *vp2*; *vp9*-4889 dormant, pale aleurone, pale green seedling, encodes zeta-carotene C-7,8 desaturase, subunit A	564
vp10			viviparous10, yellow endosperm, colored aleurone, green seedlings, adherent	637
vpp1			vacuolar proton pump homolog1, low copy leaf cDNA csu37, similar to yeast ATPase subunit, encodes vacuolar (H+)-ATPase, 54 kDa	29
vsi1			variable short internodes1, clusters of 2-4 short internodes, predominantly at base of plant but varies in location; temperature sensitive	161
vsp1			vegetative-specific protein homolog1, endosperm cDNA 5C01C06 (*uaz246*), similar to slime mold vegetative protein, may encode vegetative-specific protein	263
vsr1	10.04	10.07	virescent striped1, dominant *Vsr1* seedlings virescent, greening to white and yellow striped plant	471, 476
w1	6.05		white1, white seedling (yellow with *l1*), germinates normally; plastid transcripts variously aberrant	173, 174, 366
w2	10.06		white2, white seedling (yellow with *l1*); endosperm pitted and spotted (allele *dek21*); plastid DNA content decreased	369
w3	2.06		white3, like *vp2*; *w3*-8686 pale endosperm, pale green seedling in dim light, may encode phytofluene 11'-12' dehydrogenase	140, 369
w11	9.02		white11, like *w1*	143
w14	6.05		white14, like *w1*	133
w15	6.01		white15, like *w1*; fails to convert protochlorophyllide to chlorophyllide	133
w16	7.02		white16, like *w1*	443
w17	7.01		white17, like *w1*	443
w18	1.10		white seedling18, like *w1*; pale green streaks in some backgrounds	480

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*	486, 660
wc1	9.05	9.06	white cap1, dominant *Wc1* kernels have pale yellow endosperm if *Y1* (pearly white with *y1*); whiteness is emphasized in soft-starch crowns	339
wd1	9.00		white deficiency1, white seedling, deficiency for distal half of first chromomere of short arm; does not complement *pyd1*, *v28*, *yg2*	409
wgs1	5.05	5.09	white green sectors1, white seedling with green sectors	458
whp1	2.09		white pollen1, duplicate factor with *c2* for pollen color and for anthocyanins, encodes chalcone synthase	117
wi1	6.01	6.02	wilted1, chronic wilting, leaves not as cool as normal; delayed differentiation of metaxylem vessels	532
wi2	3.00	3.09	wilted2, in dominant *Wi2* plants, top leaves wilt under moisture/temperature stress	479
wi3			wilted3, Like *wi2* but slightly yellowish green.	481
wi4	5.02	5.06	wilted4, dominant *Wi4* plants are chronically wilted	489
wip1			wound induced protein1, wounding-induced transcript, cDNA clone (601bp) sequenced; homologous to Bowman-Birk proteinase inhibitors, may encode Bowman-Birk proteinase inhibitor	576
wip2			wound inducible protein2, endosperm cDNA 5C05B11 (*uaz293*), similar to basic wound-induced bean protein, may encode wound inducible protein--basic	264
wlu1	3.05	3.09	white luteus1, pale yellow seedling; lethal	458
wlu2	7.02	7.06	white luteus2, like *wlu1*	458
wlu3	8.04	8.09	white luteus3, like *wlu1*	458
wlu4	9.03	9.08	white luteus4, like *wlu1*	458
wlu5	1.07		white luteus5, like *wlu1*	480
wlv1	2.05		white leaf-virescent1, pale green virescent seedling	463
wrk1	3.04		wrinkled kernel1, dominant *Wrk1* kernels small and wrinkled	471
wrp1	2.04	2.05	wrinkled plant1, dominant *Wrp1* plants dwarf, leaves and culm longitudinally corrugated; dosage effect	51
ws1			white sheath1, light yellow leaf sheaths; duplicate factor with *g1* (*ws2*).	326
ws3	2.01		white sheath3, white leaf sheath, culm, husks	555
ws4	1.00	1.05	white sheath4, dominant *Ws4* seedlings and plants lighter green in sheaths	479
wsm1	6.01		wheat streak mosaic virus resistance1, dominant *Wsm1*, major gene for resistance; may be allelic to *Mdm1*	417
wsm2	3.05		wheat streak mosaic virus resistance2, dominant *Wsm2* like *Wsm1*	418
wsm3	10.05		wheat streak mosaic virus resistance3, partial resistance, semidominant; *Wsm3* plants express delayed WSMV-induced symptoms of dispersed, isolated spots and rings	418
wsp			weak streaked plant, maternally inherited reduced plants	71, 163
wt1	2.04		white tip1, tip of first leaf white and blunt	649
wt2	4.00	4.05	white tip2, seedling with white leaf tip and crossbands on first 2 leaves	458
wx1	9.03		waxy1, amylopectin (stained red by iodine) replaces amylose (blue staining) in endosperm and pollen; extensive allelic series; application: highly branched starch has gelling properties, used for food gels, adhesives, and other industrial purposes, encodes NDP-glucose-starch glucosyltransferase, starch granule-bound	123
wyg1	7.03		white yellow green seedling1, whitish, light yellow-green seedling	443
y1	6.01		white1, reduced carotenoid pigments in endosperm; some alleles affect chlorophyll in seedlings (e.g. *y1-8549*, *y1-pb*), encodes phytoene synthase	131
y8	7.01		pale yellow8, pale endosperm	298
y9	10.03	10.04	pale yellow9, pale endosperm, slightly viviparous; green to pale green seedlings and plants, may encode zeta-carotene C-7,8 desaturase	565
y10	3.07		pale yellow10, pale endosperm; white seedling, lethal	655
y11			pale yellow11, pale endosperm; green seedling	655
y12			pale yellow12, like *y11*	567, 570
yd2	3.06		yellow dwarf2, yellow dwarf seedling, lethal	569
yg1	5.07		yellow-green1, yellow-green seedling and plant	185
yg2	9.00		yellow-green2, like *yg1*; complements *pyd1* but not *wd1*	295
ypt1			ypt homolog1, cDNAs obtained by homology to GTP-binding domain of ras-protein family and mouse ypt protein, encodes YPT1 ras protein family homolog	507
ypt2			ypt homolog2, cDNA obtained by homology to GTP-binding domain of ras-protein family and mouse ypt protein, encodes YPT2 ras protein family homolog	507
ys1	5.05	5.06	yellow stripe1, yellow tissue between leaf veins, reflects iron deficiency symptoms; defect in Fe-phytosiderophores, may encode phytosiderophore	33
ys2	1.00	1.05	yellow stripe2, yellow tissue between leaf veins	531
ys3	3.05		yellow stripe3, yellow tissue between leaf veins, corrected by ferrous iron supplementation	738
ysk1	4.04		yellow streaked1, dominant *Ysk1* plants have longitudinal yellow streaks in top 3rd of mature leaves	465, 476

zadc1			amino deoxychorismate synthesis homolog1, leaf cDNA *csu329*, similar to bacterial para-aminobenzoate synthase glutamine amidotransferase subunit, may encode p-aminobenzoate synthase glutamine amidotransferase, CII	31
zag1	6.06		*Zea* AGAMOUS homolog1, amino acid sequence, deduced from cDNA to inflorescence mRNA, 61% identical to *Arabidopsis* floral homeotic gene protein AG1; expression strong in female inflorescence; possibly *pt1*, may encode transcription factor(MADS box)	600
zag2	3.04	3.05	*Zea* AGAMOUS homolog2, amino acid sequence, deduced from cDNA sequence, has 49% identity to *Arabidopsis*, floral homeotic gene product; expression restricted to female inflorescence; possibly *ts4*, may encode floral transcription factor(MADS box)	600
zap1			*zea* apetala homolog1, low copy number; leaf cDNA *csu137* similar to *Arabidopsis* floral homeotic gene, *ap1*, may encode transcription factor, flowering	29
zb1			zebra crossbands1, yellowish crossbands on older leaves	139
zb2			zebra crossbands2, crossbands on seedling leaves	676
zb3	5.08	5.09	zebra crossbands3, yellowish crossbands on older leaves	145, 171
zb4	1.03		zebra crossbands4, regularly spaced crossbands on earlier leaves; enhanced by cool temperatures	261
zb6	4.06		zebra crossbands6, regularly spaced crossbands on earlier leaves; enhanced by cool temperatures	648
zb7	1.09		zebra crossbands7, lighter green crossbands on seedlings; glossy	458
zb8	9.01	9.02	zebra crossbands8, in dominant *Zb8* plants, yellow-green crossbands on older leaves; strong anthocyanin expression in leaf tip and blade	465, 471
zbr1	4.08		maize beta repeat homolog1, leaf cDNA *csu166*, similar to insect giant secretory protein beta repeat, may encode secretory protein	29
zcef1			*Zea* CEFD homolog1, leaf cDNA *csu188*, similar to *Streptomyces clavuligerus* protein, may encode pyridoxal-phosphate-dependent aminotransferase	31
Zeon1			Zein retrotransposon, 1k copies of LTR-related sequences, 3-400 copies of internal sequence	279
zmh1			*Zea* mouse H58 homolog1, endosperm cDNA 5C06F05 similar to conserved vertebrate embryogenesis protein, may encode vacuolar protein sorting PEP8 homolog	264
zmm1			*Zea mays* MADS1, genomic sequence whose putative protein has 94% identity with that of *zag2*; this pair of MADS box genes, and *zmm2* with *zag1*, may reflect duplications; expression restricted to female inflorescence	690
zmm2			*Zea mays* MADS2, cDNA similar to *zag1* , and to AGAMOUS of *Arabidopsis*; *zmm2* and *zag1*, like *zmm1* and *zag2*, are possibly duplications phylogenetically; expression preferentially in the tassel	690
zmm3			*Zea mays* MADS3, identified by Northerns; expressed preferentially in male inflorescence	193
zmm6			*Zea mays* MADS6, identified by cDNA hybridization in situ; early expression in male and female inflorescences, differentially in one member of a spikelet-primordium pair	88
zmm7			*Zea mays* MADS7, identified by Northern analysis; expression female-specific, late onset	193
zmm8			*Zea mays* MADS8, identified by cDNA hybridization in situ; expression in undifferentiated floral meristem and becomes localized in stamen and gynoecium, palea and lemma of the upper flower of pairs	88
zms1			*Zea* male sterile protein homologue1, leaf cDNA 6C06C01, similar to *Arabidopsis MS2*, may encode male sterile protein	264
zn1	10.04		zebra necrotic1, necrotic tissue appears between veins in transverse leaf bands on half-grown or older plants	278
zn2			zebra necrotic2, like *zn1*	221
znod1			*Zea* nodulation homologue1, leaf cDNA 6C06E02, similar to alfalfa early nodulation protein ENOD8	264
zp			zein polypeptide, designator for genes encoding zein, encodes zein	642, 643
zp15	6.01		zein protein, 15kDa15, high methionine; genomic blot indicates one or two copies, encodes 15-kDa zein (beta zein)	516
zp19/22			19/22-kDa zein protein gene family, the major zein gene family, includes subfamilies A20, A30, B49, B59; also characterized as containing two molecular weight subfamilies 19 kDa and 22 kDa, encodes zein-1 (alpha zein)	84
zp19/22cluster1			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 stop codon, may encode zein	372
zp27			27-kDa zein protein, proline rich; least abundant of zeins in endosperm, not clear if part of zp27cluster, encodes 27-kDa zein (gamma zein)	710
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)	136
zpB36	7.02		27kD zein protein36, modifier of opaque endosperm from Pool 33 QPM, may encode zein	377
zpc326			plasmacytoma 326 homolog, endosperm cDNA 5C03G07, similar to human H326 and to murine plasmacytoma protein, PC326	264
zpg1			zebra-stripe pale green1, chlorophyll modifications; heterozygote advantage; induced by EMS in Oh43	157
zpl1			zein polypeptidesL1, Zp1La - Zp1Lf complex, encodes zein	730, 731

zpl1a	4.01	4.02	zein protein 1a, zein protein characterized by electrophoretic mobility on isoelectric focusing gels, encodes zein	730, 731
zpl1b	4.02		zein protein 1b, zein protein characterized by electrophoretic mobility on isoelectric focusing gels, encodes zein	730, 731
zpl1c	4.02		zein protein 1c, zein protein characterized by electrophoretic mobility on isoelectric focusing gels, encodes zein	730, 731
zpl1d	4.02		zein protein 1d, zein protein characterized by electrophoretic mobility on isoelectric focusing gels, encodes zein	730, 731
zpl1e	4.01	4.02	zein protein 1e, zein protein characterized by electrophoretic mobility on isoelectric focusing gels, encodes zein	730, 731
zpl1f	4.02		zein protein 1f, zein protein characterized by electrophoretic mobility on isoelectric focusing gels, encodes zein	730, 731
zpl2a	4.04		zein polypeptidesL2a, zein protein characterized by electrophoretic mobility on isoelectric focusing gels. Maps near *orp1*, encodes zein	730, 731
zpl2b	7.01	7.02	zein polypeptidesL2b, zein protein characterized by electrophoretic mobility on isoelectric focusing gels, encodes zein	730, 731
zpl3a	4.04		zein polypeptidesL3a, zein protein characterized by electrophoretic mobility on isoelectric focusing gels, encodes zein	730, 731
zqm1			*Zea* QM1 homolog, cDNA sequence homologous to human tumor suppressor QM1 protein, encodes QM protein homolog	191
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	304
zrp4	4.00	4.11	*Zea* root preferential4, cDNA, 1.4kb, preferentially accumulates in roots of young plants, may encode O-methyl transferase	262
zta1			*Zea* TAT binding protein homolog1, endosperm cDNA 5C01A07, similar to TAT binding protein, may encode TAT binding protein	264
zug1			*Zea* UGA3 homolog1, leaf cDNA csu9, similar to yeast transcriptional activator, UGA3, may encode transcription factor	29

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g736. Wright, AD. 1995. Maydica 40:85-88 *(olc1)*
g737. Wright, AD. 1995. MNL 69:3 *(olc1)*
g738. Wright, JE. 1961. MNL 35:111 *(ys3)*
g739. Wright, S and Helentjaris, T. 1988. MNL 62:104 *(lhcb1, lhcb2)*
g740. Wright, S et al. 1987. MNL 61:89-90 *(ssu1, ssu2)*
g741. Wright, S et al. 1993. Plant J 3:41-49 *(mfs14, mfs18)*
g742. Wu,S. and Kriz,A.L. 1992. GenBank/EMBL/DDBJ, nucleic acid sequence database *(geb1)*
g743. Yanagisawa, S and Izui, K. 1993. J Biol Chem 268:16028-16036 *(mnb1, mnb2)*
g744. Young, TE; Demason, DA; Close, TJ. 1994. Plant Physiol 105:759-760 *(amy3)*
g745. Zack, CD et al. 1986. Maydica 31:5-16 *(Tourist)*
g746. Zaitlin, D et al. 1992. MNL 66:70-71 *(rDNA5S)*

UMC 1995 Core Probe Set Information Sheet

Probe Name	Locus Name on UMC 95 Map	Chr.	Bin No.	Type	Insert Size (bp)	Enzyme	In Old Core Set
p-tub1	*tub1*	1	1.01	genomic	158	*EcoRI/HindIII*	no
p-umc157	*umc157*	1	1.02	genomic	1220	*PstI*	yes
p-umc76	*umc76*	1	1.03	genomic	760	*PstI*	yes
p-asg45	*asg45*	1	1.04	genomic	350	*PstI*	no
p-csu3	*csu3(gfu)*	1	1.05	cDNA	1200	*EcoRI/XhoI*	no
p-umc67	*umc67*	1	1.06	genomic	650	*PstI*	yes
p-asg62	*asg62*	1	1.07	genomic	500	*PstI*	no
p-umc128	*umc128*	1	1.08	genomic	740	*PstI*	yes
p-csu164	*csu164*	1	1.09	cDNA	700	*EcoRI/XhoI*	no
p-umc107	*umc107a*	1	1.10	genomic	1090	*PstI*	no
p-umc161	*umc161a*	1	1.11	genomic	700	*PstI*	no
p-bnl6.32	*bnl6.32*	1	1.12	genomic	2250	*PstI*	yes
p-bnl8.45	*bnl8.45a*	2	2.01	genomic	2100	*PstI*	no
p-umc53	*umc53a*	2	2.02	genomic	640	*PstI*	yes
p-umc6	*umc6*	2	2.03	genomic	590	*PstI*	yes
p-umc34	*umc34*	2	2.04	genomic	970	*PstI*	yes
p-umc131	*umc131*	2	2.05	genomic	810	*PstI*	yes
p-umc255	*umc255*	2	2.06	genomic	1050	*PstI*	no
p-umc5	*umc5a*	2	2.07	genomic	850	*PstI*	yes
p-asg20	*asg20*	2	2.08	genomic	550	*PstI*	no
p-umc49	*umc49a*	2	2.09	genomic	630	*PstI*	yes
p-php20581	*php20581b*	2	2.10	genomic	1400	*PstI*	no
p-umc32	*umc32a*	3	3.01	genomic	990	*PstI*	yes
p-csu32	*csu32*	3	3.02	cDNA	500	*EcoRI/XhoI*	no
p-asg24	*asg24*	3	3.03	genomic	550	*PstI*	no
p-asg48	*asg48*	3	3.04	genomic	1600	*PstI*	no
p-umc102	*umc102*	3	3.05	genomic	1010	*PstI*	yes
p-bnl5.37	*bnl5.37*	3	3.06	genomic	2300	*PstI*	yes
p-bnl6.16	*bnl6.16*	3	3.07	genomic	2450	*PstI*	no
p-umc17	*umc17*	3	3.08	genomic	850	*PstI*	no
p-umc63	*umc63*	3	3.09	genomic	620	*PstI*	yes
p-csu25	*csu25a*	3	3.10	cDNA	1100	*EcoRI/XhoI*	no
p-agr r115	*agrr115*	4	4.01	cDNA	600	*EcoRI/HindIII*	no
p-php20725	*php20725a*	4	4.02	genomic	1650	*PstI*	yes
p-umc31	*umc31a*	4	4.03	genomic	550	*PstI*	yes
p-npi386	*npi386*	4	4.04	genomic	1200	*PstI*	no
p-agr r37	*agrr37*	4	4.05	cDNA	300	*EcoRI/HindIII*	no
p-umc156	*umc156a*	4	4.06	genomic	570	*PstI*	no
p-umc66	*umc66a*	4	4.07	genomic	1020	*PstI*	no
p-umc127	*umc127c*	4	4.08	genomic	1210	*PstI*	no
p-umc52	*umc52*	4	4.09	genomic	780	*PstI*	yes
p-php20608	*php20608*	4	4.10	genomic	1500	*PstI*	yes
p-umc169	*umc169*	4	4.11	genomic	670	*PstI*	no
p-npi409	*npi409*	5	5.01	genomic	660	*PstI*	no
p-umc90	*umc90*	5	5.02	genomic	1240	*PstI*	no
p-tub4	*tub4*	5	5.03	cDNA	200	*EcoRI/HindIII*	no
p-bnl4.36	*bnl4.36*	5	5.04	genomic	2300	*PstI*	no
p-csu93	*csu93b*	5	5.05	cDNA	800	*EcoRI/XhoI*	no
p-umc126	*umc126a*	5	5.06	genomic	670	*PstI*	yes
p-umc108	*umc108*	5	5.07	genomic	970	*PstI*	yes
p-bnl5.24	*bnl5.24*	5	5.08	genomic	2500	*PstI*	no
p-php10017	*php10017*	5	5.09	genomic	470	*PstI*	yes
p-umc85	*umc85*	6	6.01	genomic	600	*PstI*	yes
p-umc59	*umc59*	6	6.02	genomic	930	*PstI*	yes
p-npi393	*npi393*	6	6.03	genomic	1200	*PstI*	no
p-umc65	*umc65a*	6	6.04	genomic	650	*PstI*	yes

p-umc21	*umc21*	6	6.05	genomic	1050	*Pst*I	yes
p-umc38	*umc38a*	6	6.06	genomic	1010	*Pst*I	yes
p-umc132	*umc132a*	6	6.07	genomic	500	*Pst*I	yes
p-asg7	*asg7*	6	6.08	genomic	550	*Pst*I	no
p-asg8	*asg8*	7	7.01	genomic	500	*Pst*I	no
p-asg34	*asg34a*	7	7.02	genomic	1350	*Pst*I	no
p-asg49	*asg49*	7	7.03	genomic	400	*Pst*I	no
p-umc254	*umc254*	7	7.04	genomic	1050	*Pst*I	no
p-umc245	*umc245*	7	7.05	genomic	700	*Pst*I	no
p-umc168	*umc168*	7	7.06	genomic	1080	*Pst*I	no
p-npi220	*npi220a*	8	8.01	genomic	400	*Hin*dIII	yes
p-bnl9.11	*bnl9.11*	8	8.02	genomic	2400	*Pst*I	no
p-umc124	*umc124*	8	8.03	genomic	1160	*Pst*I	yes
p-bnl7.08	*bnl7.08a*	8	8.04	genomic	2300	*Pst*I	no
p-bnl2.369	*bnl2.369*	8	8.05	cDNA	700	*Eco*RI	no
p-csu31	*csu31*	8	8.06	cDNA	800	*Pst*I	no
p-npi268	*npi268*	8	8.07	genomic	670	*Pst*I	no
p-npi414	*npi414*	8	8.08	genomic	870	*Pst*I	yes
p-agr r21	*agrr21*	8	8.09	cDNA	900	*Eco*RI/*Hin*dIII	no
p-umc109	*umc109*	9	9.01	genomic	840	*Pst*I	yes
p-umc192	*bz1*	9	9.02	genomic	1800	*Eco*RI/*Hin*dIII	no
p-umc25	*wx1*	9	9.03	cDNA	2300	*Eco*RI	no
p-csu147	*csu147*	9	9.04	cDNA	800	*Eco*RI/*Xho*I	no
p-umc95	*umc95*	9	9.05	genomic	680	*Pst*I	yes
p-csu61	*csu61*	9	9.06	cDNA	500	*Eco*RI/*Xho*I	no
p-asg12	*asg12*	9	9.07	genomic	700	*Pst*I	no
p-csu54	*csu54b*	9	9.08	cDNA	1400	*Eco*RI/*Xho*I	no
p-php20075	*php20075a*	10	10.01	genomic	1400	*Pst*I	yes
p-npi285	*npi285*	10	10.02	genomic	970	*Pst*I	yes
p-umc130	*umc130*	10	10.03	genomic	640	*Pst*I	yes
p-umc64	*umc64*	10	10.04	genomic	710	*Pst*I	yes
p-umc163	*umc163*	10	10.05	genomic	410	*Pst*I	yes
p-umc44	*umc44a*	10	10.06	genomic	800	*Pst*I	no
p-bnl7.49	*bnl7.49a*	10	10.07	genomic	2100	*Pst*I	no

Clone Identification by Sequence Similarity and Mapping Results

I. The lab designations for all clones with putative sequence identities are listed in the first column. Those with a designation of rsp, spf, or beginning with "2" or "5" were isolated from endosperm libraries. Those beginning with "6" originated from an etiolated seedling library. Those denoted "csu" were previously isolated from a mature vegetative tissue library (obtained from Stratagene) and sequenced by Chris Baysdorfer at California State University-Hayward; many have since been mapped at the University of Arizona.

II. In the second column, the GenBank accession number assigned to this sequence is reported when available.

III. In the third column, the most significant similarity found through comparison of our sequence with the GenBank database is reported. Comparison is examined at the amino acid similarity level by the subroutine BLASTX and if not successful in identifying a match, subsequently at the nucleic acid level by BLASTN. Strong similarities at the amino acid level are usually indicated by a BLASTX score of 180 or better. Weaker matches are usually indicated by a suffix of "homolog" in this field. Alternatively if protein motif similarities are determined through the use of the BLOCKS program, then the term "motifs" is usually added as a suffix in this section.

IV. In the fourth column, the highest BLASTX score is reported for the similarities detected (suffixed by "X"). If this score is preceded by ">", this indicates that multiple regions of similarity were detected which often reflect a much higher level of significance than indicated by the highest BLASTX score for individual blocks. If the score is followed by "N", this score represents a BLASTN score. Alternatively, "%" reflects the amount of identity between the two sequences.

V. In the fifth column, the accession numbers from GenBank, dbEST, PIR, or SwissProt of the closest match are reported. If the identity was established at the level of protein motifs through the use of the BLOCKS routine, this is indicated.

VI. In the sixth column, we report an indication from colony screening as to whether the sequence is abundantly (Abnd) or rarely expressed and if the former, in what tissues (S=seedling, E=endosperm).

VII. In the seventh column, an indication of the type of pattern in a Southern hybridization to maize genomic DNA is reported as being either a "Complex" pattern (more than three prominent fragments observed) or "Simple" (three or less).

VIII. In the eighth column are given the UAZ map designations for those clones which have been mapped recombinationally in the Brookhaven RI lines. Mnemonics are given for those clones which exhibit some extensive homology to an entry in GenBank. "gfu" refers to a clone that has been mapped but for which sequence information is not yet available or which did not detect any significant homology within GenBank..

IX. The last column reports the chromosomal locations detected by this mapping. Here we use the "Binning" as developed by the maize group at Missouri, to define chromosomal locations along each chromosome. Bins were assigned relative to the 1993-1994 University of Missouri-Columbia Maize RFLP map [ED. NOTE: Please see the 1995 UMC map - several bins and bin boundaries have been modified as a result of refinements in scoring and analysis].

The Maize cDNA Project is the result of a collaboration between groups at three institutions:

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Clones with Significant Indications of Function through Sequence Analysis

			Similar Sequence					
I* Clone Designation	II GenBank Access. #	III Putative Gene Product	IV Score	V Access #	VI Express. Pattern	VII Genomic Complexity	VIII Locus Name	IX Genomic Location
2C01H10 5C05F10 6C02G05	T18751 T15301 T18435	triosephosphate isomerase - maize	562X	A25501	Abnd/S	Complex	uaz93(tpi) uaz296(tpi)	2.04,3.04, 8.02-8.03
csu142	T12737	sugar transport protein homolog - yeast	96X	S30811		Simple	csu142(sut)	2c
rsp12,rsp64 5C04A07,5C04D10, 5C07G02,5C11D03	T14662 T14713 T23326 T25261	sucrose synthase - maize	564X	L01626	Abnd/E	Simple	uaz154(sh1)	9.02
5C01G05	T18269	glucosamine-fructose-6-phosphate aminotransferase [1]	410X	P14742	Rare	Complex	uaz309(cht)	3.10,10.00
6C04B04,6C06C10	T15330	b-glucosidase - maize	662X	X74217	Abnd/S	Simple	uaz339(glu)	3.04,10.04
6C01G09	T20392	b-glucosidase - maize	85%	X74217	Abnd/S	Complex	uaz340(glu)	10.04,10.05-10.06
5C04B10	T14684	soluble starch synthase - rice	184X	D16202	Rare	Complex	uaz218(strs)	3.07,4.05-4.06
5C04H03 5C10A05	T18721 T25209	*waxy* gene- maize	1609N	X03935	Rare	?		

[1] 1st enzyme in chitin synthesis

5C04H06,5C06D08 5C10G07	T18724 T14743 T25240	ADPG pyrophosphorylase (*sh2*) - maize	520X	S48563	Abnd/E	-	-	1,3L,4,5
5C06E05 5C07B08	T18304 T18637	ADPG pyrophosphorylase (embryonic) - potato	664X	S18238	Rare	Complex	agp1	6.11
5C02H07 6C01B06,6C02D08	T14797 T20333 T18408	UDPG pyrophosphorylase - potato rice dbEST	546X 707N	P19595 D24990	Rare	Simple	uaz194(udp)	2.08
5C05F05	T15298	starch branching enzyme I - maize	587X	JT0968	Rare	Simple	uaz265(sbe1)	2.06,6.02-6.03
5C03G08 5C09B11	T14653 T14654 T23384	starch branching enzyme 2 - maize	523X	L08065	Rare	Simple	uaz229(sbe2)	3.02
rsp33		a-glucan phosphorylase - potato	361X	S15531	-			
Rice10		fructose-biphosphate aldolase - rice	--	--	-	Simple	uaz291(fda)	8.08
5C07H03 5C08C04 5C11D06	T23335 T23349 T25264	alcohol dehydrogenase 1 - maize rice	230X 537N	A00342 X16296	Rare			1.11-1.12
csu149	T12742	short chain alcohol dehydrogenase (*ts2* homolog)			-	Simple	csu149(sca	5.00
5C04A01	T14656	sorbitol dehydrogenase similarity - human	129X	Q00796	Rare	Simple	uaz152(sdh)	9.07-9.08
2C02D11 5C01C11, 5C01F03, 5C01H01, 5C02C11, 5C02E03, 5C04E09, 5C05F03, 5C09G12 5C10H04, 5C11A01 6C01H03, 6C02B05 csu140, csu152	T14755 T14761 T14789 T14727 T15296 T20366 T23281 T18391 T18686 T23408 T25245 T25250 M95076	glyceraldehyde-3-phosphate dehydrogenase - maize rice dbEST *Arabidopsis* dbEST	583X 1327N 1293N	PQ0178 D16096 M64116	Rare	Simple	uaz73(gpa) uaz190(gpa) uaz261(gpa) uaz271(gpa)	4.03-4.04,5.07
5C10H08	T25248	anthranilate synthase 1 - *Arabidopsis*[2]	537X	JQ1684	Rare	?		
csu65	M95067	anthranilate synthase 2 - yeast[3]	58%	P00937	-	-		
6C02A09	T18386	a-ketoglutarate dehydrogenase - yeast	>225X	P20967	Abnd/E	Simple	uaz215(odo)	5.02-5.03,5.09
5C03E12, 5C06C08, 5C06D11, 5C10G06	T18293 T18302 T23305 T25239	a-ketoglutarate dehydrogenase - human rice dbEST	>179X 820N	Q02218 D23067	Rare	Complex	?	
5C04E05, 5C07D08, 5C07F11	T14721 T14722 T18645 T23324	monodehydroascorbate reductase - *Cucumis*	110X	D26932	Rare	Simple	uaz204(dar)	5.09
csu324		citrate synthase - pig	67%	M21197	-	-		
5C01C07, 5C09B01 csu158	T14753 T23378	enolase - maize rice dbEST *Arabidopsis* dbEST	319X 784N 751N	X55981 D21305 T14216	Rare	Simple	csu158(eno1)	9.03
5C03H06, 5C05H04, 5C08G09	T15312 T23313 T23369	ketol-acid reductase isomerase - *Arabidopsis* rice dbEST	538X 355X	X68150 36767	Abnd/E	Complex	uaz269(kri)	2.07,6.01, 6.10,8.04
csu198	T18783	malate dehydrogenase - pig	69%	M29463	-	-		
csu77	M95069	malate dehydrogenase - maize	61%	M95069	-	Simple	csu77(mdh)	1.10
csu16		NADP malic enzyme - maize	100%	J05130	-	Simple	csu16(me)	3.02,6
csu262	T18824	6-phosphoglucanate dehydrogenase - algae	66%	X58719	-	-		
5C06D06, 5C09B06	T18298 T23382	pyruvate dehydrogenase E1 complex - human	316X	S13825	Rare	Simple	uaz301(pyrd)	1.08
5C04A04,5C04C06, 5C06G09, 5C07E01, 5C09B04, 5C09C05 5C11B03, csu155	T18315 T18678 T18648 T23380 T23390 T25252	pyruvate phosphate dikinase - maize	534X	M58656	Abnd/E	Simple	uaz127, uaz172, uaz183, uaz188, uaz196, uaz319 (pdk)	6.07,8.05
5C05D10, 5C09E04, 5C10C04	T15339 T23401 T25216	NADH-ubiquinone oxidoreductase - cow *Arabidopsis* dbEST	169X >77	P80266 14420	Rare	Complex	uaz266(nad)	1.04,9.05-9.06
6C02C04	T15328	catalase1 - maize	212X	P18122	Abnd/ES	Simple	uaz360(cat1)	5.05
rsp27		catalase2 - maize	166N	X54819	-			1s
6C02C06, 6C06C07	T18399	catalase3 - maize	272X	L05934	Abnd/S	Simple	mitoch?	4L
6C02D10	T18410	peroxidase (lignin form enz) - rice	120X	S22087	Rare	Simple	uaz235(px)	2.06
csu182	T18842	superoxide dismutatase - maize	98%	X17564	-			

[2] component I catalyzes the formation of anthranilate using ammonia rather than glutamine. pathway: first step in biosynthesis of tryptophan. subcellular location: chloroplast (probable)

[3] catalyzes the formation of anthranilate. pathway: first step in biosynthesis of tryptophan and of pyocyanin

6C02E12, 6C02F07 csu160	T18420 T18426 T12746	lipoxygenase L-2 - rice	159X	J03211	Abnd/S	Complex	uaz225(lox)	7.04
6C06B11	T15323	cytochrome P450 hydroxylase - *S.melongena*	143X	X70981	Abnd/S	Simple	uaz338(p450)	7.01-7.02
csu25	T12664	cytochrome P450 similarity - rat	88X	P33274	-	Simple	csu25(p450)	3.10
5C05B02	T15273	thioredoxin - *Arabidopsis* rice dbEST	200X 175X	P29448 37996	Rare	Simple	?	
csu111	T12723	glutathione reductase - pea	223X	P27456	-	-		
csu44		glutathione-S-transferase - maize	100%	X04455	-	-		
csu40	T12674	[4] Glutaredoxin similarity - *E. coli* *Arabidopsis* dbEST	74% 298N	P00277 T13672	-	Simple		
Rice12		glutamic dehydrogenase - rice	--		-	Complex	uaz292(gdh)	7.04
rsp13, rsp145 5C09H06	T23410	aspartate amino transferase - rice	410X	S18891	Rare	?		
5C04B05, 5C07G11	T14674 T14675 T23332	alanine transaminase - millet	671X	S28429	Rare	Simple	uaz158(alt)	5.05
5C03D02	T23295	[5] glutamate 1-semialdehyde aminotransferase rice dbEST	100X 530N	P30268 D24532	Abnd/ES	?		
csu194	T18851	methionine synthase - *E. coli*	60%	M87625	-			
6C01G01	T20390	S-adenosyl methionine synthase - rice	78%	Z26867	Abnd/S	Complex	?	
5C01E09	T18260	adenosylmethion-8-amino-7-oxononanoate [6] aminotransferase - bacteria	>167X	P22805	Rare	Complex	uaz113(aaa)	2.02-2.03, 10.08
5C01H11	T18277	[7] s-adenosyl-l-homocysteine hydrolase rice dbEST *Arabidopsis* dbEST	539X 492X 195X	D16138 37567 14494	Rare	Simple	uaz145(ahh)	4.03-4.04
csu156	M95077	phenylalanine ammonia lyase - rice	91%	X16099	-	Complex	csu156(pal)	2s,4s,5L
5C01C04, 5C02C04, 5C08F11	T14750 T14778 T23368	[8] peptidyl-prolyl c-t isomerase - maize rice dbEST	500X 1092N	P21569 D16134	Abnd/E	Simple	uaz238(ppi)	5.07
5C05G07, 5C06F04, 5C08A08, 5C11B04 6C01F05 fplo	T15306 T18307 T20387 T23340 T25253	[9] protein disulfur isomerase - barley *Arabidopsis* dbEST rice dbEST	475X 217X 114X	L33252 34988 37958	Rare	Simple	uaz298(pdi) uaz239(pdi)	2.10,4.02
5C05C09, 5C10E02	T15280 T25224	[10] hypothetical 42.8kd protein - yeast	221X	P32615	Rare	Complex	uaz295(orf)	2.05,3.03, 5.07
5C01E01 6C01B08	T18661 ?	ubiquitin precursor - maize rice dbEST	307X 848N	S04863 D25067	Abnd/E	Simple	uaz247(ubi)	4.11
5C08E11	T23365	ubiquitin precursor - sunflower *Arabidopsis* rice	310X 308X 305X	S17435 S01425 P50380	Rare	?		
csu204	T18788	ubiquitin carrier protein - wheat	73%	M74077	-	-		
2C06C11 5C02F11, 5C04D06 5C05D12, 5C07E09	T14794 T14707 T14708 T15289 T18767 T23320	ubiquitin-conjugating enzyme E2 - wheat	611X	P16577	Rare	Simple	uaz102(uce) uaz206(uce)	6.01
csu269 spf1, spf11		[11] nucleoside diphosphate kinase - mouse	191X	M65037	-	Simple	uaz31(ndk) uaz10(ndk)	1.05
spf4		nucleoside diphosphate kinase - *Spinacia*	241X	S24165	-	Simple	uaz91(ndk)	7.04
csu269	T18831	nucleoside diphosphate kinase - rice	83%	D16292	-	-		
5C10C03	T25215	[12] Acyl-CoA binding protein - *Brassica* *Arabidopsis* dbEST rice dbEST	229X 483N 529N	X77134 T04081 D28303	Rare	?		
2C02C10	T18756	acyl carrier protein - maize	170N	X57956	-	Simple	uaz6(acpt)	3.05

[4] function: the disulfide bond functions as an electron carrier in the glutathione-dependent synthesis of deoxyribonucleotides by the enzyme ribonucleotide reductase. in addition, it is also involved in reducing some disulfides in a coupled system with glutathione reductase

[5] catalytic activity: n2-acetyl-l-ornithine + 2-oxoglutarate = n-acetyl-l-glutamate 5-semialdehyde + l-glutamate. the reaction catalyzed by acoat is highly reversible. moreover this enzyme may transaminate ornithine. pathway: fourth step in arginine biosynthesis. cofactor: pyridoxal phosphate. subcellular location: mitochondrial matrix. similarity: belongs to class-iii of pyridoxal-phosphate-dependent aminotransferases

[6] pathway: bioconversion of pimelate into dethiobiotin

[7] This enzyme forms an antagonist of methylation and is thought to be utilized in its control.

[8] involved in folding of proteins

[9] catalytic activity: rearrangement of both intrachain & interchaindisulfide bonds in proteins to form the native structures. subcellular location: lumen of endoplasmic reticulum

[10] encoding serine hydroxymethyltransferases, whose inactivation is required to render yeast auxotrophic for glycine

[11] while this enzyme is involved in the transfer of phosphates from ATP to other nucleotide diphosphates, there is also an interesting association with tumor cells

[12] function: binds medium- and long-chain acyl-CoA esters with very high affinity and may function as an intracellular carrier of acyl-CoA esters.

2C01H08	T18750	acyl carrier protein reductase - *Cuphea* *Arabidopsis* dbEST	>255X >112X	S19832 33679	-	Simple	uaz99(acpr)	10.04
5C02D03,5C02H03	T23282 T23286	colinephosphate cytidylyltransferase - rat[13] *Arabidopsis* dbEST rice dbEST	107X 83X 103X	P19836 49925 D28176	Rare	?		
csu265	T18827	cycloartenol synthase - *Arabidopsis*[14]	66%	U025550	-	-		
6C06C04	T15326	enoyl-CoA hydratase - rat	95X	P07896	Abnd/S	Simple	uaz348(ech)	8.05
6C06A05, 6C06A06 6C06B04	T15316 T15317 T15318	mevalonate kinase similarity - rat	72X	P17256	Abnd/ES	Simple	uaz337(mvk)	6.08,8.04
5C03E07, 5C06C02	T23301 T18289	oleosin KD18 - maize	703N	J05212	?	?		
5C02A01 csu136, csu205	T14763 M95074	phospholipid transfer protein - maize	222X	J04176	Rare	Simple	csu136(plt)	10s
5C03D06	T23297	d(24)-sterol C-methyltransferase - yeast[15] rice dbEST	115X 104X	P25087 D24567	Rare	?		
csu125	M95073	chloroplast carbonic anhydrase - pea	59%	X52558	-	-		
6C02A04	T18381	CP29 (CAB-binding protein homolog?) *Arabidopsis*	>180X	X71878	Rare	Simple	uaz200(cab)	7.04
csu66	M95068	Chlorophyll A/B-binding protein - rice	74%	D00642	-	Complex	csu66(cab)	8s
csu71		Chlorophyll A/B-binding protein - maize	99%	X14794	-	Complex	csu71(cab)	1L
csu102	T12718	Chlorophyll A/B-binding protein - rice	93%	X13909	-	Complex	-	
csu117	T12725	Chlorophyll A/B-binding protein - *Arabidopsis*	72%	M63931	-	-		
csu224	T18804	Chlorophyll A/B-binding protein - maize	90%	X55892	-	-		
csu229	T18809	16kd O2 evolving factor - *Spinacia*	62%	X05512	-	-		
csu74	T12699	ferredoxin - maize	95%	M73828	-	Simple	?	
5C03F05	T23307	sulfite reductase similarity - cyanobacteria	73X	P30008	Abnd/S	?		
6C01D01	T20381	PEP carboxylase - maize	99%	X15238	Abnd/ES	?		
csu18, csu233, csu237, csu267	T12659 T12660 T18812 T18816 T18829	photosystem 1 subunit - barley	66%	X66428	-	-		
csu67	T12694 T12695	photosystem K-1 - barley	63%	L12707	-	-		
csu185, csu257	T18823	plastocyanin - barley	65%	Y00704	-	-		
6C02C05	T18398	RUBISCO, lg subunit - maize	330X	P00874	Abnd/S	Simple	-	Chloroplast
5C11D08	T25266	mitochondrial adenylate kinase - rat[16]	108X	JQ1945	Rare	?		
5C04B04,5C05A03	T14672 T14673 T15269	ATP synthase a mitochondrial similarity - yeast	73X	P07251	Rare	Simple	uaz144(atp) uaz254(atp)	4.05-4.07,5.09
5C04E07	T14725	ATP synthase b mitochondrial - maize Arabidopsis dbEST rice dbEST	655X 527X >242X	P19023 46770 37494	Rare	Complex	uaz243(atp)	3.08,6.08, 8.04
csu26		ATP/ADP mitochondrial translocator - maize	100%	X02842	-	Simple	csu26(amt)	5.08
5C04A11	T14665	*Bt1* precursor - maize	183X	P29518	Rare	Complex	uaz155(bt1)	10.04
2C01F03	T18745	*Bt1*-similarity?, MCP similarity? - maize	93X	mzebtia	-	Complex	uaz25(mcp)	2.06,5.02- 5.03,8.04
5C02F05, 5C02H10	T14791 T18669	mitochondrial carrier protein - yeast	115X	P32331	Rare	Complex	uaz282(mcp)	1.06
csu68	T12696 T12697	ADP,ATP carrier protein. precurser - potato	>98	S17917	-	Simple	-	
6C02E07	T18416	ATPase vacuolar, nuclear binding subunit - barley	323X	L11862	Abnd/E	Complex	uaz223(atp)	9.04
csu30	M95063	ATPase vacuolar, proteineolipid subunit	85%	M73232	-	Simple	csu30(atp)	3.05
csu37	T12671	ATPase vacuolar, 54-kDa subunit - yeast	89X	D13916	-	Simple	-	
csu220	T18800	possible pyrophosphatase? - rice	91%	D15382	-	-	-	
5C02E08,5C05B01	T14790 T15272	PPase, vacuolar membrane proton pump - *Arabidopsis*	>265X	P31414	Rare	Complex	uaz280(pps)	4.00,9.07
5C06E01,5C07D01	T18303 T18643	voltage dependent anion channel (porin) - maize[17]	429X	X77733	Rare	Simple	uaz312(por)	2.06
5C05F04	T15297	cytochrome c & c1 heme lyase motifs rice dbEST	-- 314X	BLOCKS 36333	Rare	Simple	uaz262(gfu)	2.06
5C08B12	T23346	40s ribosomal protein S3 - *Xenopus* *Arabidopsis* dbEST	194X 485N	P02350 T21989	Rare	-	-	
6C01B02	T20332	40s ribosomal protein S4 - rice	76%	D23071	Rare	-	-	

[13] function: controls phosphatidylcholine synthesis. catalytic activity: ctp + choline phosphate = pyrophosphate + cdp choline. enzyme regulation: by phosphorylation. subcellular location: it can interconvert between an inactive cytosolic form and an active membrane-bound form. subunit: homodimer. the cytidyltransferase may interacts with membranes through its amphipathic helix

[14] Isolation of a gene encoding cycloartenol synthase by functional expression in a yeast mutant lacking lanosterol synthase using a chromatographic screen

[15] function: methyltransferase required for ergosterol synthesis

[16] catalytic activity: GTP + AMP = GDP + ADP. subcellular location: mitochondrial matrix.

[17] function: forms a channel through the cell membrane, that allows diffusion from small hydrophilic molecules. subcellular location: plasma membrane or outer membrane of mitochondria

5C06A11	T18283	40s ribosomal protein S5 - rat	495X	P24050	Rare	Simple	uaz308(rps5)	8.02
5C01A12		40s ribosomal protein S6 homolog - tobacco	108X	S25550	Abnd/E	Complex	uaz119(rps6)	7.05,8.08, 9.05-9.06
5C01A09, 5C04G09, csu34	T18658 T18712 T18713 M95064	40s ribosomal protein S8 homolog - human	>242X	P09058	Rare	Simple	uaz115(rps8)	4.08-4.09
5C02F12 6C02A02	T14795	40s ribosomal protein S11 - maize	333X	P25460	Abnd/S	Complex	uaz251(rps11)	2.02-2.03, 3.07,6.08, 8.04, 10.05-10.07
5C08C03	T23348	40s ribosomal protein S12 - rat rice dbEST	216X 966N	M18547 D23869	Rare	-		
5C01H07	T18275	40s ribosomal protein S14 - maize	438X	P19951	Rare	-		
2C01G06	T18747	40s ribosomal protein S21 - yeast	>70X	P05764	-	Complex	-	
csu28	M95062	40s ribosomal protein S22 - *Xenopus*	72%	M34706	-	-		
5C01E10, 5C06G08	T18261 T18314	40s ribosomal protein S24 - human	175X	P16632	Rare	Complex	-	
5C09A02	T23373	40s ribosomal protein S27 - rat rice dbEST	331X 841N	P24051 D23139	Rare	-		
5C02D01, 5C06G06	T14781 T18312	40s ribosomal protein S27A - maize	325X	JS0657	Rare	Complex	uaz249(rps27a)	1.08,3.04, 8.04,8.10
5C01A05	T18654	40s ribosomal protein S28 - rat	232X	P25112	Rare	Simple	uaz146(rps28)	1.01
5C03H03, 5C04D11, 5C05D07, 5C09E10, 5C09G08, 5C11D05	T14714 T14715 T15285 T23312 T23403 T23406 T25263	60s ribosomal protein L5 (5s rRNA-binding protein)-yeast rice *Arabidopsis*	263X 442N 405N	P15125 S39486 Z17506	Rare	Simple	uaz189(rpl5) uaz260(rpl5)	1.01,3.05
rsp81		60s ribosomal protein L7 - human	220X	R5HU7	-	-		2,10
5C01D03, 5C01F07 5C01H05, 5C02H06, 5C04F09	T14756 T14796 T18266 T18273 T18699	60s ribosomal protein L10e homolog - yeast	87X	P15826	Rare	Complex	uaz198(rpl10)	3.10
5C05D11, 5C07G06	T15288 T23329	60s ribosomal protein L12 - human *Arabidopsis* rice dbEST	268X 142X 553N	P30050 S29452 D15738	Rare	-		
5C02E11, 5C03E11, 5C03G09, 5C04D09	T14711 T14712 T23283 T23304	p23 tumor specific transpl Ag (L13) - human[18] *Arabidopsis* rice dbEST	387X 381X 934N	A44367 S37230 D24830	Rare	Simple	uaz208(tta) uaz126(tta)	1.12
csu245 5C09E05	T23402	60s ribosomal protein L14 - *Xenopus* *Arabidopsis* dbEST	179X 455N	X05025 T21082	Rare	-		
5C05G01	T15303	60s ribosomal protein L17 - barley	308X	S35101	Rare	Complex	uaz297(rpl17)	2.07,6.06, 8.03
5C04B03, 5C04G01, 5C06C05 csu36	T14670 T14671 T14735 T18291 M96065	60s ribosomal protein L19 - human	495X	P14118	Rare	Simple	csu36(rpl19) uaz157(rpl19) uaz213(rpl19)	3.10,4.05-4.08 5.05
5C01A04	T18653	60s ribosomal protein L24 homolog - tobacco	62X	M87838	Rare	Complex	uaz270(rpl24)	4.08-4.09
5C02F01, 5C02F07	T23284 T23285	60s ribosomal protein L30A - yeast *Arabidopsis* rice dbEST	184X 928N 865N	P04449 Z26463 D23828	Rare	Complex	-	
5C05D03, 5C06C03 5C10D04, 6C01D02	T15284 T18290 T20340 T25220	60s ribosomal protein P2 - yeast rice dbEST	222X 846N	P08094 D22651	Rare	Complex	uaz294(lap2)	10.08
csu236	T18815	translation initiation factor 2 - bacteria	54%	X04399	-	-		
6C02E11	T18419	translation initiation factor eif2 - human	160X	L19161	Abnd/S	Simple	uaz224(if2)	7.04
6C01A08	T20330	translation initiation factor 4F - wheat	>84X	M95747	Abnd/SE	Simple	?	
csu175, csu203 5C06C07, 5C07H01, 5C09D04	T18787 T18836 T18292 T23333 T23399	translation initiation factor 5A - tobacco *Arabidopsis* rice dbEST	354X 365X 998N	P24921 S31362 D24702	Abnd/E	Complex	uaz303(if5)	7.04
5C03C06, 5C04H09, 5C08H09, 5C10G09 csu116, csu226 rsp35, rsp37	T14798 T14745 T18806 T18726 T23372 T25242 M95072	elongation factor 1a - *Arabidopsis*	611X	P17786	Rare	Complex	uaz220(eif)	6.02-6.03,6.06, 7.04,8

[18] while denoted as a tumor antigen, this is in fact a one amino acid mutation of a ribosomal protein L13 type

5C04C04	T14690 T14689	elongation factor 1g - *Arabidopsis* rice dbEST	215X 684N	L17307 D22778	Rare	Complex	uaz161(eif)	3L,4.10, 6.02-6.033,9.05-9.06
5C10C06	T25217	elongation factor 1g - *Arabidopsis* rice	405X 405X 501N	S36462 S36462 D15948	Rare	?		
5C10A11	T25212	elongation factor Tu (Ct) - *Arabidopsis*	231X	P17745	Rare	?		
5C03H10,5C10H03 csu63	T12693 T23314 T25244	chaperone DNAJ homolog - *Allium* *Arabidopsis*	584X 481X	S42031 S35588	Abnd/ES	?	csu63(cdj)	?
csu52 rsp30		chaperone DNAJ similarity, nuclear envelope protein - yeast	60%	X56560	-	-	uaz109(cdj)	3.01
6C01A04, 6C02E06	T20328 T18415	chaperonin 10kd chloroplast - pea	>276X	Q02073	Abnd/ES	Simple	uaz222(cpn10)	4.07
6C01D12	T20345	chaperonin (RUBISCO-binding protein, b-subunit) - rape *Arabidopsis* dbEST	317X 501N	P21241 Z18404	Abnd/ES	?		
5C01H04, 5C02D08 5C06H12, 5C08D04, 5C10A02	T18272 T14785 T18326 T23356 T25208	[19] CD4B (chloroplast ATP-binding chaperonin) - tomato *Arabidopsis* ERD1 gene rice dbEST	596X 461N 957N	P31542 D17582 D29692	Rare	Simple	uaz242(chp)	10.04
5C03E09	T23302	CLP-TRYBB (chloroplast ATP-binding chaperonin?) - trypanosome	82X	P31543	Abnd/E	?		
csu209 5C09H04	T18792 T23409	[20] GOS2 - rice	201X	P33278	-	-		
5C06G10	T18316	[21] annexin (synexin) - *Dictyostelium*	134X	P24639	Rare	Simple	uaz320(anx)	6.01
5C02D02, 5C04G12, 5C05A12, 5C07E12, 5C09D07	T14782 T14740 T15271 T23321 T23396	protein prenyltransferase motifs		BLOCKS	Rare	Simple	uaz255(gfu) uaz277(gfu)	3.02
5C09H10	T23411	[22] Sec61 b-subunit - *Arabidopsis*	118X	Z26753	Rare	?		
2C07F04	T18781	[23] signal recognition part recept a subunit- dog	152X	A24570	-	Simple	uaz8(spr)	3.06-3.07
5C08B07	T23344	[24] signal recognition part recept 9kd protein - *C. elegans*	179X	P34642	Rare	?		
5C01B12, 5C09C01, 5C09C07	T14746 T23387 T23391	aspartyl-tRNA synthetase a chain - rat	429X	P15178	Rare	Complex	uaz132(dts)	5.05-5.06
6C02G11	T18439	seryl-tRNA synthetase homolog - yeast	99X	P07284	Abnd/ES	Complex	uaz236(strs)	2.06,9.04
2C01B05	T18731 T18732	19kd a zein 19A2 - maize	>355X	S15656	Abnd/E	Complex	uaz49(zp19/22)	2.02-2.03,4.01
2C06H03 spf19, spf24	T18774	19kd a zein 19A20 - maize	539X	zizm2	Abnd/E	Complex	uaz68(zp19/22)	4.01
2C01B06	T18733	19kd a zein 19A30 - maize	228X	S21970	Abnd/E	Complex	uaz49(zp19/22)	4.01
2C06H04	T18775	19kd a zein 19AB1 - maize	363X	S03417	Abnd/E	-		
5C01H08, 5C04H01 5C10F09,	T14762 T18717 T18718 T25235	19kd a zein 19C2 - maize	622X	P06677	Abnd/E	Complex	-	
5C03B07, 5C10F12	T25206 T25237	19kd a zein CLO - maize	317X	P06675	Abnd/E	Complex	uaz219(zp19/22)	4.01
5C02A08, 5C06F03 spf6, spf28	T14767 T18306	19kd a zein 19D1 - maize	563X	P06678	Abnd/E	Simple	uaz5(zp19/22) uaz272(zp19/22)	1.05
5C03G02, 5C11E08 spf20	T18673 T25269	19kd a zein PMS2 - maize	434X	P24450	Abnd/E	Complex	uaz149(zp19/22)	4.01
2C06D05	T18768	19kd a zein ZG31A - maize	>190X	S21965	Abnd/E	Complex	-	
5C04D05	T14706	22kd a zein pML1 - maize	315X	X14334	Abnd/E	Complex	-	
5C03B06, 5C04F08 5C10H10,5C11C04	T18672 T14733 T25249 T25258	22kd a zein PZ22.1 - maize	>299X	P04700	Abnd/E	Complex	uaz185(zp19/22)	4.01

[19] function: required for the retention of lumenal endoplasmic reticulum proteins, affects glycoprotein processing in the golgi apparatus. subcellular location: integral membrane protein

[20] function: additional factor that functions in concert with eif-2 and the initiator tRNA in directing the ribosome to the proper start site of translation

[21] promotes membrane fusion and is involved in exocytosis

[22] function: appears to play a crucial role in the insertion of secretory and membrane polypeptides into the ER. it is required for assembly of membrane and secretory proteins and is essential for cell growth. it interacts with other membrane proteins required for protein translocation

[23] integral membrane protein ensures the correct targeting of the nascent secretory proteins to the endoplasmic reticulum membrane system

[24] function: signal-recognition-particle assembly has a crucial role in targeting secretory proteins to the rough endoplasmic reticulum membrane. srp9 together with srp14 and the alu portion of the srp RNA, constitutes the elongation arrest domain of srp. the complex of srp9 and srp14 is required for srp RNA binding (by similarity)

5C02C05, 5C02C08	T14751 T14780	22kd a zein PZ22.3 - maize	559X	P04698	Abnd/E	Complex	-	
spf10 5C10E04	T25226	22kd azein ZA1 - maize	569X	B22831	Abnd/E	-		
2C07D02 5C02A02, 5C02A03, 5C04E12, 5C04G06 spf8	T14764 T18780 T18689 T18708 T18662	16kd b zein ZC1 - maize	>523X	P06673	Abnd/E	Complex	uaz169(zp16)	6.01
5C05D05, 5C07F10	T23316 T23323	10kd d-zein - maize	407X	JA0086	Abnd/E	Simple	-	6.06,9.04
rsp80, rsp92		g zein - maize	>217X	zmzm19	Abnd/E	Simple	-	7s
g zein		g zein - maize				Simple	uaz327(gz)	2.07
6C01C02	T20336	g zein genomic region similarity - maize	207N	X58197	Rare	Simple	?	
5C04B07	T14678 T14679	glutenin similarity - wheat rice dbEST	>73X 712N	A30843 D21910	Rare	Simple	uaz230(glu)	4.05-4.06,6.10
5C07G01	T23325	napin gene - *Brassica napus*	577N	M64633	Rare	?		
5C09A06	T23374	[25] acylamino acid-releasing enzyme - pig	106N	D00524	Rare	?		
csu223	T18803	[26] cystatin - maize	90%	D10622	-	-		
5C04A08, 5C09B03, 5C09C03 rsp96, rsp111	T14663 T23379 T23389	chymotrypsin inhibitor - barley	292X	S37493	Rare	Simple	uaz232(sci)	2.09
6C02H08	T18444	lysosomal proteinease similarity - wheat	?		Rare	Simple	uaz234(pros)	2.06
5C07B07 6C01C07	T18636 T20337	[27] mitochondrial processing protease - rat	458X	B48529	Rare	Complex	?	
2C01C07	T18736	[28] oligopeptidase A - bacteria	>102X	A42298	-	Simple	uaz100(prc)	10.02-10.03
5C01H12	T18278	proteasome C5 complex - human	288X	P20618	Rare	Simple	uaz226(prc)	1L
5C02A05, 5C09B05	T14766 T23381	proteasome C9 complex - human rice dbEST	316X 349N	P25789 D22304	Rare	Simple	uaz237(prc)	6.01,9.02
csu5	M95060	[29] thiol protease - rice bean	65%	X15732	-	Simple	thp1	7.03
csu96	M95070	thiol protease inhibitor	81%	M29259	-	Simple	csu96(thp)	9
5C03B04 rsp11, rsp21	T18671	Trypsin/Factor 7A inhibitor - maize	378X	P01088	Abnd/E	Smear	-	
5C04F01	T14729	[30] b32 protein RIP3 - maize (*rip1*)	613X	P25891	Abnd/E	Simple	uaz193(rip)	8.04-8.05
5C04B01, 5C07A12, 5C09D11, 5C11D07	T18677 T18631 T23398 T25265	b32 protein RIP9 - maize	474X	P25892	Abnd/E	Simple	uaz156(rip)	3.08
5C06F11, 5C06H09	T18321 T18324	[31] ribonculease PH homolog- *E. coli* rice dbEST	94X 933N	P03842 D23232	Rare	Simple	uaz313(rns)	8.03
5C05E10	T15294	metallothionein I similarity - castor bean *Arabidopsis* dbEST	158X 672N	P30564 T04702	Rare	Complex	uaz300(mtl)	3.04,6.01
csu169, csu206	T12751 T18790	metallothionein homolog - maize	511N	S57628	-	-		
6C02F11	T18429	actin AC1 - carrot rice dbEST	170X 451N	J01238 D25019	Abnd/S	Complex	uaz233(act)	8.04-8.05,6.01 7.04
5C01H03,5C08E04	T18271 T23361	actin 97 - potato *Arabidopsis* rice	622X 1241N 1233N	P30171 M20016 X15863	Rare	Complex	-	
5C10D06	T25221	[32] clathrin coat assembly protein - *C. elegans*	204X	P35602	Rare	?		
5C06B09	T18287	[33] profilin - maize	346X	P35081	Rare	Simple	uaz305(prof)	6.01

[25] function: this enzyme catalyzes the hydrolysis of the amino- terminal peptide bond of an N-acetylated peptide to generate an N-acetylated aa and a peptide with a free amino-terminus. it preferentially cleaves off ac-ala, ac-met and ac-ser

[26] cysteine proteinase inhibitor, developmental stage: reaches a maximum 2 weeks after flowering and then decreases gradually. similarity: shows high similarity to cystatins; belongs to the phytocystatin subfamily

[27] function: the mitochondrial processing protease cleaves presequences from mitochondrial protein precursors. function: this is a component of the ubiquinol-cytochrome c reductase complex (complex iii or cytochrome b-c1 complex), which is part of the mitochondrial respiratory chain. this protein may mediate formation of the complex between cytochromes c and c1. subunit: the mitochondrial processing peptidase is composed of two subunits, the catalytic subunit and the processing enhancing protein. subcellular location: mitochondrial matrix. similarity: belongs to the insulinase family of proteases

[28] similar to zinc metalloproteinases, may play a specific role in the degradation of signal peptides after they are released from precursor forms of secreted proteins

[29] function: thought to be involved in the hydrolysis of stored seed proteins

[30] endosperm ribosome inactivating protein, catalytic activity: endohydrolysis of the n-glycosidic bond at one specific adenosine on the 28s rRNA

[31] removes nucleotide residues following the -CCA terminus of tRNA and adds nucleotides to the ends of RNA molecules

[32] function: component of the adaptor complexes which link clathrin to receptors in coated vesicles. clathrin-associated protein complexes are believed to interact with the cytoplasmic tails of membrane proteins, leading to their selection and concentration. ap47 is a subunit of the plasma membrane adaptor. subcellular location: component of the coat surrounding the cytoplasmic face of coated vesicles located at the golgi complex.

[33] antagonist of actin polymerization

5C11B05 6C01F07, 6C04E06 csu272	T20355 T25254	a-tubulin 1 - maize *Arabidopsis*	99% 1232N	X15704 M84697	Abnd/SE	Simple	uaz177(tua)	2.00
6C02D05, 6C03A07 6C06C08, 6C06D02	T18407 T15329 T15332 T25274	a-tubulin 3 - maize	558X	S28429 P22275	Abnd/E	Complex	uaz201(tua3)	5.01
6C06B09	T15321	a-tubulin 6 - maize	584X	P33627	Abnd/E	Complex	uaz315(tua)	2.09,7.04
csu148	T12741	calnexin - *Arabidopsis* [34]	192X	P29402	-	-		
5C06B08	T18286	caltractin - *Scherffeli* [35]	440X	X69220	Rare	Simple	uaz322(cal)	7.05
5C06D10	T18301	uromodulin (fibrillin) - rat [36]	105X	P27590	Rare	Simple	uaz304(hon)	1.04,9.04
6C02C10	T18403	Ser-Lys-rich protein	--	-	Abnd/S	Simple	?	
5C04B11, 5C05A01	T14685 T14686 T15267	Ala-repeat protein	--	-	Rare	Simple	uaz159(glu)	5.02-5.03
6C02B04	T18390	Lys-Glu-rich protein	--	-	Rare	Simple	uaz202(glu)	8.04
5C02D07	T14784	Arg-Pro-rich protein	--	-	Rare	Simple	uaz342(glu)	9.05-9.06
6C02F01	T18421	Pro-Pro-rich protein (extensin-like)	--	-	Abnd/E	Complex	-	
6C02F03	T18423	Pro-Val-rich protein (extensin-like)	--	-	Abnd/E	Simple	uaz192(glu)	7.05-7.06
5C05D01	T15283	hydroxyproline rich(Ser,Pro) protein - rice	83X	S20500	Rare	Simple	uaz341(glu)	4.05-4.06
5C07C09	T18649	glycine-rich cell wall protein homolog - rice	101	P29834	Rare	Smear	-	
6C02D09	T18409	glycine-rich protein - tomato	>68X	X55691	Rare	Smear	-	
csu190	T18848	auxin-induced mRNA - tobacco	82%	X56267	-	-		
5C05E12	T15295	ABA-induced, stress, ripening protein - tomato rice dbEST	94X 418N	L08255 D23985	Rare	Simple	?	
csu108	M95071	GTP-binding protein [37]	77%	M35520	-	Simple	csu108(gbp)	5s
csu234	T18813	ras-related GTP-binding protein - rice	64%	D17779	-	-		
5C03G12, 5C07G05	T14655 T23328	GTP-binding protein SAR1 - *Arabidopsis* [38] rice dbEST	354X 258N	Q01474 D21890	Rare	Simple	uaz151(gbp)	1.09
5C04D03	T18680	GTP-binding protein, developmentally regulated - mouse [39] *Arabidopsis* dbEST	283X 289N	D10715 T14006	Rare	Simple	uaz245(gbp)	7.05
5C10C02	T25214	GTP-binding protein YPTM2 - maize [40] *Arabidopsis*	458X 412X	Q05737 P28188	Rare	?		
5C01G12	T18270	guanine nuclear-binding protein b subunit - yeast	119X	S19487	Rare	Complex	uaz310(gbp)	4.03-4.04,6.00
5C06B04	T18285	guanine nuclear-binding protein b subunit - yeast	97X	S19487	Rare	Simple	uaz306(gbp)	9.02
5C05C03	T15277	guanine nuclear-binding protein b subunit - yeast rice dbEST	97X 175X	S19487 36095	Rare	Simple	uaz257(gbp)	4.05-4.06
5C08C12	T23353	G-protein b subunit - *Arabidopsis* dbEST rice dbEST	119X 594N	Z29843 D23414	Rare	?		
5C04C02	T14687	TDR3 homeotic (MADS) - tomato rice dbEST	188X 411N	X60756 D22380	Rare	Complex	uaz231(mad)	9.07-9.08
csu137	T12732 T12733	MADS box (*agamous*) - *Arabidopsis*	70%	X53579	-	Simple	csu137(mad)	1.12,5.02-5.03
5C09A09	T23377	protein kinase (*CTR1*) - *Arabidopsis* [41]	204X	L08789	Rare	?		
5C03D08, 5C04A03	T14658 T14659 T23298	protein kinase (*Tousled*) - *Arabidopsis*	380X	L23985	Rare	Complex	uaz130(tpk)	1.12,4.05-4.06 5.02-5.03
5C04G11, 5C07B02	T18714 T18715 T18633	Ca-dependent protein kinase - *Arabidopsis*	319X	L14771	Rare	Complex	uaz197(cdpk)	6.01,6.01
csu231	T18811	protein kinase - rat [42]	60%	M16112	-	-		
csu252	T18821	MAP protein kinase - tobacco [43]	80%	X69971	-	-		

[34] function: calnexin is part of a complex whose function is to bind Ca(2+) to the er membrane and thereby regulate the retention of ER resident proteins

[35] function: this calcium-binding protein is found in the basal body complexes (the functional homologue of the centrosome in animal cell). in mitotic cells it is specifically associated with the poles of the mitotic spindles at the sites of the duplicated basal body complexes

[36] function. not known. may play a role in regulating the circulating activity of cytokines as it binds to il-1, il-2 and tnf with high affinity

[37] function: protein transport. probably involved in vesicular traffic (by similarity).

[38] RAS-like protein involved in protein transport; endoplasmic reticulum; golgi stack.

[39] predominantly expressed in the embryo and down-regulated during development

[40] function: protein transport. probably involved in vesicular traffic (by similarity). similarity: to ras proteins. belongs to ypt1 sub-family.

[41] *CTR1*, a negative regulator of the ethylene response pathway in *Arabidopsis*, encodes a member of the Raf family of protein kinases

[42] Ca^{++}-dependent, CAM dependent

[43] similarity: belongs to the cdc2/cdc28 subfamily of ser/thr protein kinases. strongest similarity with other map kinases

5C08E10	T23364	[44] MAP kinase (STE7) - yeast *Arabidopsis* rice dbEST	175X >61X 364N	P06784 P25859 D24368	Rare	?		
5C02A07	T18663	protein kinase (tyr-ser-thr) *Arabidopsis* rice dbEST	225X 135X	L07248 38402	Rare	Simple	uaz252(ptk)	4.07-4.08,8.04
5C05H12,5C09D08	T15315 T23397	protein kinase (tyr) homolog - pig	73X	Q00655	Rare	Simple	uaz299(ptk)	5.05,6.01
5C08B04	T23342	14-3-3 homolog - maize	510X	Q01526	Rare	Simple	ufg7(tau)	2.05,10.05-10.06
csu64	M95066	[45] 14-3-3 homolog - human	72%	J03868	-	Simple	csu64(tau)	1.10,2.10
5C07A05	T18627	[46] 14-3-3 homolog - tomato rice dbEST *Arabidopsis* dbEST	429X 955N 833N	L29150 D24218 L09109	Rare	Complex	uaz331(tau)	1.06,2s,3.05
5C04C05	T14691	phosphoprotein phosphatase - *Drosophila*	218X	S29396	Rare	Complex	uaz244(prh)	6.06,8.03
5C06G06?	T18312?	transcription activator - virus	206X	P03209	Rare	?		
5C04C08, 5C06D02 5C11E10	T14694 T25270	*myb* transfer protein similarity - virus	81X	A28013	Rare	Complex	uaz216(gfu)	4.05-4.06
5C07C04	T18641	[47] nucleolar transcription factor similarity - rat	>65X	P25978	Rare	Simple	uaz335(ntf)	4.05-4.06
5C03F11	T23308	[48] transcription factor BTF3a - human	99X	JC1235	Rare	?		
5C09C08	T23392	[49] transcription factor s1Fa - spinach rice dbEST *Arabidopsis* dbEST	246X 666N 484N	X79543 D22962 T20524	Rare	?		
5C04D07	T14709 T14710	transcription factor, Zn finger similarity - virus rice dbEST	96X 619N	P27426 D24735	Rare	Simple	uaz207(tfz)	2.05
csu150	M95075	RNA Polymerase II - yeast	73%	M15693	-	Simple	csu150(rpo)	5.02-5.03,6.01
csu17	T12657 T12658	[50] chloroplast 31kd ribonucleoprotein - tobacco	60%	X53942	-	Simple	csu17(rnp)	2.09
5C02D05, 5C11F03 6C01H06	T14783 T25271 T20367	U1 small nuclear ribonucleoprotein similarity	>39X	M18465	Rare	Simple	uaz346(gfu?)	10.08
5C07C06	T18642	[51] RNA binding protein (splicing) - *Drosophila* rice dbEST	>184 276N	L04929 D24389	Rare	?		
fpl6		RNA-binding protein - maize			-	Complex	uaz240(rnb)	1.15,5.11-5.12 6.11
5C04G05, 5C05C12	T14737 T15282	RNA-binding protein (GAP-associated tyr phosphoprotein) - human *Arabidopsis* dbEST	>82X 315X	M88108 49463	Rare	Simple	uaz214(rnb) uaz259(rnb)	5.00
5C02G05 6C01E07, 6C02G01, 6C02G12 csu195, csu208 , csu215	T18431 T20349 T18440 T18666 T18791 T18852 T18796	glycine-rich protein, ABA - and H_2O-inducible, RNA [52] binding - maize rice dbEST *Arabidopsis* gene	133X 219N 103X	P10979 D23787 Q03250	Abnd/ES	Simple	?	
6C02F05	T18425	[53] DNA-binding protein - *Drosophila*	73X	P13469	Abnd/ES	Simple	uaz318(nbp)	2.02-2.03, 10.08
5C08B05	T23343	DNA-binding protein - maize	215N	X66076	Rare	?		
6C04B07, 6C06D05	T15333 T25277	[54] DNA-binding protein similarity - wheat	77X	P25032	Abnd/S	Simple	uaz179(nbp)	2.06

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[44] function: serine/threonine protein kinase required for cell-type-specific transcription and signal transduction in yeast. it is thought that it is phosphorylated by the ste11 protein kinase and that it can phosphorylate the fus3 and or kss1 kinases. enzyme regulation: phosphorylated at multiple sites in response to pheromone. pathway: response to pheromone-induced signal. similarity: with the conserved catalytic domains of ser/thr-protein kinases. belongs to the map kinase kinase family

[45] 14-3-3 homolog, function: activates tyrosine and tryptophan hydrolyses in the presence of ca(2+)/calmodulin-dependent protein kinase II, strongly activates protein kinase c

[46] this protein is similar to the 14-3-3 protein homolog from human brain which is also identified in plants as being an inhibitor of protein kinases

[47] function: ubf recognizes the ribosomal rna gene promoter and activates transcription mediated by rna polymerase i through cooperative interactions with the species-specific factor sl1. it binds specifically to the upstream control element. subunit: ubf consists of 2 polypeptides of 94 and 97 kd, encoded by closely related genes. subcellular location: nuclear. similarity: contains five hmg boxes

[48] function: general transcription factor. BTF3 can form a stable complex with RNA polymerase II. required for the initiation of transcription

[49] An unusual small DNA-binding protein specifically recognizes a negative element within the spinach rps1 promoter

[50] function: could be involved in splicing and/or processing of chloroplast RNA's

[51] function: may be involved in RNA processing in relation with cellular proliferation and/or maturation. subcellular location: nuclear.

[52] similar to RNA-binding proteins

[53] function: its capacity to bind DNA and protein(s), and its differential expression during development may suggest a role in the regulation of gene expression during *Drosophila* development. subcellular location: nuclear. ptm: phosphorylated

[54] function: interacts specifically with the 8-bp sequence 5'-cacgtggc-3'in the abscisic acid response element (abare). subcellular location: nuclear. similarity: to other bzip proteins

2C06G06 5C05F12, 5C05G04, 5C06B02	T15305 T15302 T18284 T18772	CAAT box-binding protein - maize	285X	zmnfyb	Rare	Simple	uaz7(caat)	7.01-7.02
5C06A10	T18282	G-Box binding factor similarity - *Dictyostelium*	90X	L29705	Rare	?		
5C09C10	T23393	[55] Drome Matern Tudor protein similarity? - *Drosphila* *Arabidopsis* dbEST	>72X >277X	P25823 21180	Rare	?		
csu56	T12687	[56] leucine zipper transcript regulator- maize	94X	L00623	-	Complex	csu56(ohp)	2.05
5C01G03	T18267	[57] OCS-element binding factor similarity rice dbEST	83X 582N	P24068 D23215	Rare	Simple	?	
6C01F12	T20358	[58] TAT-binding homog1 - yeast rice dbEST	242X 168X	P33297 36651	Abnd/ES	Simple	uaz345(zta)	5.02-5.03,5.11-5.12
5C01A07,5C07G04	T18656 T23327	[59] TAT-binding homolog 2 - yeast	172X	P33298	Rare	Simple	uaz118(zta)	4.08
5C01E06	T18258	[60] Paired amphipathic helix protein - yeast *Arabidopsis* dbEST	105X 516N	P22579 T04565	Rare	Simple	?	
5C05C11, 5C05H07	T15281 T15313	TATA-binding protein - maize *Arabidopsis* dbEST	220X 146X	S32622 49207	Rare	Simple	?	
6C02C08	T18401	histone H2A - pea	95X	P25470	Abnd/ES	Complex	uaz221(his2a)	7.04
5C04D12, 5C09G02 5C10F10, 6C06C02 csu285	T14716 T15325 T18835 T23405 T25236	histone H2B - maize rice dbEST *Arabidopsis* dbEST	300X 658N 169X	P30756 D15709 S30619	Rare	Complex	uaz228(his2b)	1.09.2.07, 4.05-4.06, 10.04
5C03H09, 5C05D06, 5C08D05	T14800 T23317 T23357	histone H3 - *Arabidopsis* rice dbEST	>494X 943N	S24346 D15959	Abnd/ES	Simple	uaz248(his3)	1.04,5.07
5C02C03	T14777	histone H4 - maize	233X	A25642	Rare	Simple	?	
Rice54		Sm-D nuclear antigen - rice	--		-	-	uaz290(sdag)	8.03
csu146	T12739	[61] CDC48 - yeast	60%	X56956	-	Simple	csu146(cdc48)	6.01
6C01G11	T20363	ATPase (CDC48) - yeast	67%	L14616	Rare	?		
5C08F01	T23366	nucleolin (topoisomerase I) - frog	110X	S32644	Rare	?		
5C09D05	T23395	[62] replication factor-A protein 1 - yeast	>83X	P22336	Rare	?		
5C11E05	T25268	[63] AC15 activator 140kd subunit - human	112X	P35251				
5C08D02	T23354	[64] DNA Polymerase I - *Bacillus*	112X	D12982	Rare	?		
5C10D10	T25298	[65] RAD16, DNA repair - yeast	117X	P36617	Rare	?		
6C06D10	T15336	[66] pG1 protein (transforming) - human	159X	A44803	Rare	?	chloroplast?	
6C02C02	T18395	[67] retinoblastoma associated protein - human	132X	P28749	Abnd/SE	Simple	uaz191(rap)	2.09
5C04A12	T14666 T14667	[68] early nodulin - soybean rice dbEST	230X 760N	D13506 D15238	Rare	Simple	uaz227(end)	6.01
6C06E02	T15338	early nodulin 8 - soybean rice dbEST	95X 309N	L18899 D24693	Rare	?		

[55] function: required during oogenesis for the formation of primordial germ cells and for normal abdominal segmentation. developmental stage: expressed throughout the life cycle. the tud mRNA accumulates within the posterior region of the developing oocyte during the early to middle stages of oogenesis.

[56] strong but short homology to both opaque2 and CPRF-2, which is a constitutively-expressed DNA-binding protein involved in light responsiveness

[57] function: may contribute to developmentally specific patterns of gene expression. binds specifically to ocs-element. ocs-elements are transcriptional enhancer found in the promoters of several plant genes. similarity: to other bzip proteins

[58] transcriptional repressor, CDC48-like

[59] similarity: belongs to the cdc48/pas1/sec18 family

[60] function: negative transcriptional regulator, represses the HO gene expression in daughter cells resulting from mitotic division. subcellular location: nuclear, possibly linked to centromere. similarity: sin3 contains 4 paired amphipathic helix motifs

[61] function: may have a role in spindle pole proliferation. subcellular location: bound loosely to components of the microsomal fraction

[62] function: binds single-stranded DNA. stimulates the activity of a cognate strand exchange protein (sep1). it cooperates with T-ag and DNA topoisomerase I to unwind template DNA containing the simian virus 40 origin of DNA replication

[63] function: the elongation of primed DNA templates by DNA polymerase delta and epsilon requires the action of the accessory proteins pcna and activator 1. the 140 subunit binds to the primer-template junction.

[64] DNA-directed DNA polymerase; DNA replication; DNA repair; hydrolase; exonuclease; DNA-binding

[65] involved in nucleotide excision repair of DNA damaged with uv light, bulky adducts, or cross-linking agents. endonuclease that specifically degrades single-stranded DNA (by similarity).

[66] induced in transformed cells

[67] function: may have a function in cell cycle regulation. p107 exists in both phosphorylated & unphosphorylated forms

[68] root meristem protein

csu177	T18838	nodulin 26 similarity - rice dbEST	70%?	D17443	-	-		
5C03C09	T25207	early nodulin - soybean rice dbEST	199X 640N	S34801 D15436	Rare	Simple	uaz122(end)	4.05-4.06
5C01C06	T14752	vegetative specific protein[69] - *Dictyostelium* rice dbEST *Arabidopsis* dbEST	115X 821N 814N	P14327 D15817 Z29019	Abnd/E	Simple	uaz246(vsp)	4.05-4.06
5C04E06,5C06H10	T15305 T18322	Ac transposase similarity - maize	>152X	X01380	Rare	Complex	uaz285(tpase)	1.08,5.06 8.04-8.05
csu12	M95061	CIN4 retroelement similarity - maize	67%	Y00086	-	Complex	csu12(cin4)	2.04,4.05-4.06
6C02E02,6C06C01	T18413 T15324	male sterile similarity MS2 - *Arabidopsis*	154X	X73652	Abnd/E	Simple	uaz195(ms)	4.05-4.06
csu19	T12661 T12662	cold regulated gene[70] - barley	83%	M60733	-	Simple	csu19(cld)	4.03-4.04
2C02A04 csu192, csu206	T18753	iron deficiency-induced gene - barley	136X	bylids3	-	Simple	uaz80(fei)	6.00
5C05G11	T15309	17.4kd heat shock - rice *Arabidopsis* dbEST	183X 84X	P31673 33680	Rare	Simple	uaz264(hsp)	1.03,9.05-9.06
5C02D11	T14788	17.6kd heat shock chloroplast - cheno rice dbEST	182X 568N	P11890 D15924	Rare	Simple	uaz171(hsp)	4.05-4.06
5C04F02,5C04H02	T14730 T18719 T18720	18kd heat shock - maize	119X	P24631	Abnd/E	Simple	uaz210(hsp)	3.02
5C04D01	T14700 T14701	70kd heat shock mitochondrial[71] - *Phaseolus* rice dbEST	654X 1335N	Q01899 D15740	Rare	Complex	uaz205(hsp)	1.09,5.02-5.03
6C01E09	T20351	70kd heat shock - carrot rice dbEST	153X 333N	P26791 D22078	Abnd/S	Complex	?	
csu179 5C08E07	T18839 T23363	70kd heat shock - maize *Arabidopsis* dbEST rice dbEST	369X 344X 897N	P11143 P22953 D22323	Rare	?	?	
5C04F03, 5C04H04	T18692 T18693 T14741	80kd heat shock chloroplast - spinach	549X	M99565	Rare	Simple	uaz211(hsp) uaz219(hsp)	5.02-5.03
5C07B01	T18632	82kd heat shock protein[72] - *Arabidopsis* rice dbEST	431X 899N	A48426 Z11920	Rare	Simple	uaz332(hsp)	6.01
csu274	T18833	83kd heat shock - rice	90%	Z11920	-	-		
5C01G10, 5C09C12	T14760 T23394	low temperature-specific gene - barley or salt stress induced hydroph - wheatgrass	189X	Z25537	Abnd/ES	Simple	uaz250(str)	10.04
5C10E03	T25225	glycine-rich protein ua3[73] - tomato	119X	S14984	Rare	?		
5C04C03, 5C04F07 csu133	T18697 T12729 T14688	pathogenesis related protein PR2[74] - bean	>84	S14730	Rare	Complex	uaz283(prp)	2.05
5C05B11	T15276	wound induced basic protein - *Phaseolus* rice dbEST	102X 326N	JS0731 D15315	Rare	Simple	uaz293(wip)	4.03-4.04
rsp75		wound-induced protein inhibitor similarity - tomato	61X	A34359	-	-		
5C07B03, 5C08A02 5C10F08,	T18634 T23338 T25234	hypothetic protein FMR1[75] similarity - human rice dbEST	>84 845N	A39530 D25209	Rare	Simple	uaz317(fmr)	1.01
5C06F05	T18308	essential embryonic gene - chicken rice dbEST	139X 487N	B44882 D15882	Rare	Simple	?	
5C01F05, 5C05H02, 5C06A01	T18264 T18279	ABC1 gene similarity - yeast	82X	Z28353	Rare	Simple	uaz263(abc)	4.07

[69] this protein is expressed in growing cells and deactivated upon the initiation of development

[70] contains several arginine residues in close proximity which may be involved in protein RNA interactions

[71] DNAK-like chaperonin

[72] function: molecular chaperone. has ATPase activity (by similarity). similarity: belongs to the heat shock protein hsp90 family

[73] extensin-like cDNAs with expression in response to wounding

[74] chitin synthase?

[75] This sequence has homology to a proposed protein found at the human Fragile X site.

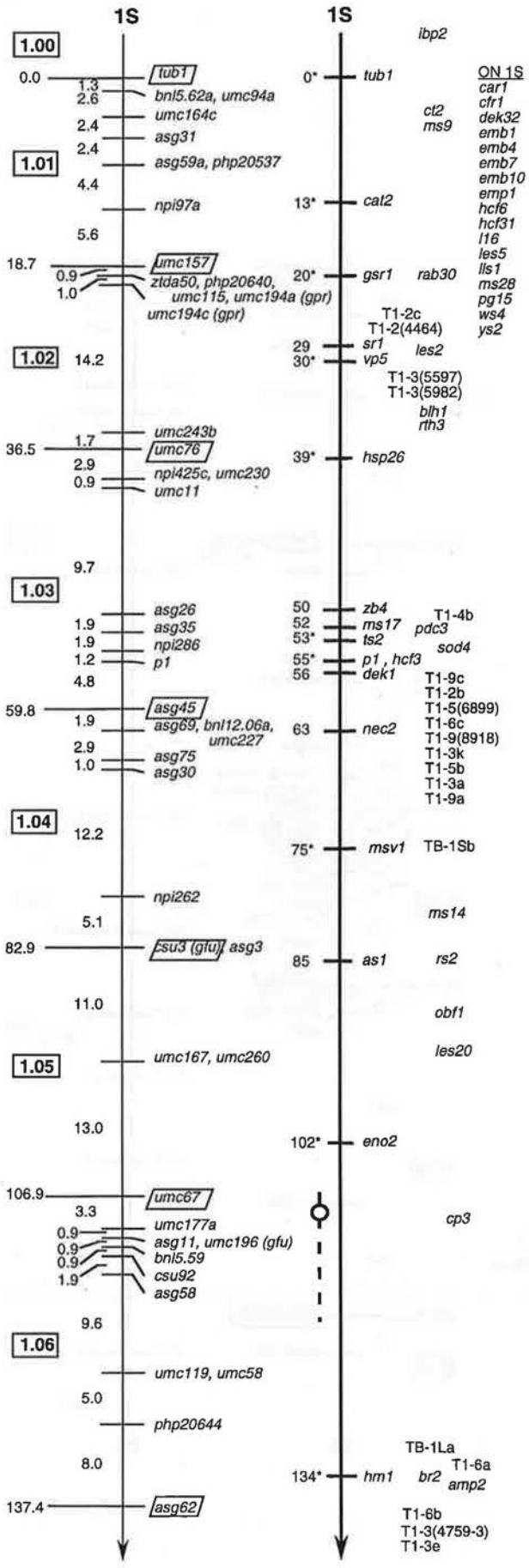
The following are clones which have been mapped but where sequence information is either not yet available or does not indicate a significant similarity:

			Similar Sequence					
I* Clone Designation	II GenBank Access. #	III Putative Gene Product	IV Score	V Access #	VI Express. Pattern	VII Genomic Complexity	VIII Locus Name	IX Genomic Location
1C06C04							uaz36(gfu)	2.05
1C07A11						Simple	uaz82(gfu)	7.04
2C01A03	T18729 T18730	a-zein by seq. but not hybrid?				Simple	uaz34(gfu?)	2.04
2C01A05						Simple	uaz79(gfu)	5.07
2C01B08	T18734	a-zein by hybrid but not seq.?				Complex	uaz66(gfu?)	4.01,4.07
2C01B12						Simple	uaz89(gfu)	7.03
2C01C05	T18735	no significant similarity				Complex	uaz43(gfu?)	6.08
2C01C09	T18737	no significant similarity				Simple	uaz29(gfu?)	2.06
2C01C11	T18738	extensive vector seq.?				Simple	uaz98(gfu?)	10.04
2C01D12	T18739 T18740	extensive vector seq.?				Simple	uaz3(gfu?)	1.03
2C01E03						Complex	uaz49(gfu)	2.01
2C01E11	T18743	no significant similarity				Simple	uaz74(gfu?)	4.08
2C01E12		a-zein?				Complex	uaz48(gfu)	4.05-4.06
2C01F01						Complex	uaz70(gfu)	4.01,5.07
2C01F02	T18744	no significant similarity				Simple	uaz24(gfu?)	2.01,10.04
2C01F04	T18746	no significant similarity				Complex	uaz18(gfu?)	1.09,2.06, 3.07,6.00
2C01F05						Complex	uaz33(gfu)	2.10,4.05-4.06
2C01F07	?					Complex	uaz35(gfu?)	3.04
2C01F08						Complex	uaz90(gfu)	7.03
2C01F10						Complex	uaz28(gfu)	2.06,7.03-7.04
2C01F11						Simple	uaz23(gfu)	2.09,6.01
2C01G08	?					Simple	uaz72(gfu?)	4.05-4.06
2C01H06	T18748	no significant similarity					uaz2(gfu?)	2.09
2C01H09						Simple	uaz015(gfu)	1.05
2C02A02						Simple	uaz97(gfu)	10.04
2C02A03	T18752	no significant similarity				Simple	uaz27(gfu?)	2.04,7.01-7.02
2C02B02	T18754	no significant similarity				Simple	uaz95(gfu?)	8.07-8.08
2C02B04	T18755	no significant similarity				Simple	uaz20(gfu?)	1.09,7.01-7.02
2C02B05	?					Simple	uaz2(gfu?)	1.01,1.09
2C02B10						Complex	uaz19(gfu)	1.09,3.04, 6.08,7.01-7.02
2C02F02						Complex	uaz14(gfu)	9.07-9.08
2C02F12						Complex	uaz38(gfu)	3.06,4.01
2C02G02	?						uaz4(gfu?)	7.07-7.08
2C02G12		a-zein by hybrid?				Complex	uaz069(gfu)	4.01,4.03-4.04
2C02H02	T18759	no significant similarity				Complex	uaz17(gfu?)	1.05,4.01
2C02H06	T18760 T18761	a-zein by hybrid but not seq.?				Complex	uaz41(gfu?)	4.01,4.05-4.06
2C02H07						Complex	uaz21(gfu)	1.05,1.12, 2.00,10.01
2C02H08	T18762	several rice dbESTs including:		C0962A C1533 1A		Simple	uaz77(gfu?)	5.06
2C03A07						Simple	uaz37(gfu)	3.05
2C03C09						Complex	uaz68(gfu)	4.01,7.01-7.02
2C03C11						Simple	uaz78(gfu)	5.07
2C03D01						Simple	uaz92(gfu)	7.04
2C03D02						Simple	uaz84(gfu)	7.01-7.02
2C03G04	T18763 T18764	no significant similarity				Simple	uaz96(gfu?)	9.07-9.08
2C03G07	T23310	no significant similarity				Simple	uaz83(gfu)	7.01-7.02
2C03H04						Simple	uaz71(gfu)	4.05-4.06, 5.11-5.12
2C04B10						Simple	uaz4(gfu)	1.04
2C04C03						Simple	uaz1(gfu)	1.02
2C04D01						Simple	uaz81(gfu)	6.08
2C04E02						Simple	uaz22(gfu)	1.15
2C04H05							uaz75(gfu)	5.00
2C04F10						Simple	uaz39gfu)	3.09
2C04G04	T18765	a-zein by hybrid but not seq?				Complex	uaz65(gfu?)	4.01,4.08
2C05A02		a-zein by hybrid?				Complex	uaz7(gfu)	4.01
2C05C05						Simple	uaz40(gfu)	9.07-9.08
3C01A08							uaz110(gfu)	3.09
3C01B04							uaz111(gfu)	5.04,5.06
3C01C03	?						uaz112(gfu?)	9.05-9.06
5C01A01					Rare	Simple	uaz116(gfu)	10.02-10.03

5C01A06,5C015E06	T18655	*Arabidopsis* dbEST	>49	14637	Rare	Complex	uaz117(gfu?)	3.10,7.04 10.04
5C01C05	T14751	a-zein by seq but not hybrid?			Rare	Simple	uaz128(gfu?)	8.10
5C01C08					Abnd/ES	Simple	uaz147(gfu)	7.04-7.05
5C01G07					Rare	Complex	uaz114(gfu)	3.09
5C01H02					Abnd/E	Simple	uaz168(gfu)	5.07
5C01D11, 5C02A09 5C04G03,5C05A02	T14736 T14768 T15268 T14759	no significant similarity			Rare	Simple	uaz273(gfu?) uaz276(gfu?) uaz253(gfu?)	1.05
5C02A11	T18664	rice dbEST	480N	D16016	Rare	Complex	uaz274(rest)	1.05,2.08
5C02B05	T14772	human ORF HUMRSC399-1	77X	D13642	Rare	Simple	uaz275(hest)	5.05
5C02B08	T14774	no significant similarity			Rare	Complex	uaz286(gfu?)	1.09
5C02D10	T14787	no significant similarity			Rare	Simple	uaz170(gfu?)	4.05-4.06
5C02E07	T25205	no significant similarity			Rare	Simple	uaz148(gfu)	9.07-9.08
5C02G02	T18665	no significant similarity			Rare	Complex	uaz288(gfu?)	2.02-2.03,3.05
5C02G08	T18667	ORF - fungi	>82X	P09379	Rare	Simple	uaz281(yorf)	4.05-4.06
5C03C07					Rare	Simple	uaz120(gfu)	1.02
5C03C08, 5C06H11, 5C08E01	T18325 ? T23293	no significant similarity			Rare	Simple	uaz121(gfu)	6.06,8.04
5C03D03		rice dbEST	355X	37832	Abund/E	?		
5C03E06, 5C04B06, 5C09B07,	T14677 T23300 T23383	yeast cDNA YBL0507	235X	Z23261	Rare	Complex	uaz241(yest)	2.09,7.05
5C04E11					Rare	Complex	uaz173(gfu)	7.01-7.02
5C03G07	T23310	H326, ubiq expressed gene - human	143X	U06631	Rare	Complex	uaz123(gfu)	4.08,6.10 7.03-7.04
5C03H11	T18674	rice dbEST	>396N	D15619	Abnd/ES	Simple	uaz186(rest)	5.05
5C04A02	T14657	PPDK seq similarity?			Rare	Simple	uaz153(gfu?)	10.02-10.03
5C04A05	?	no significant similarity			?	?	uaz131(gfu?)	?
5C04B09	T14682 T14683	no significant similarity			Rare	Complex	uaz287(gfu?)	6.04-6.05
5C04B11, 5C05A01	T14685 T14686	no significant similarity			Rare	Simple	uaz159(gfu?)	5.02-5.03
5C04C01	T23315	no significant similarity			Rare	Simple	uaz160(gfu)	6.04-6.05
5C04C07, 5C08D03	T14692 T14693	no significant similarity			Rare	Simple	uaz199(gfu?)	7.04
5C04C09	T14695 T14696	no significant similarity			Rare	Complex	uaz289(gfu?)	9.02
5C04C10					Rare	Simple	uaz203(gfu)	1.05
5C02B03, 5C04C12 5C10E06	T14699 T25228	no significant similarity			Rare	Simple	uaz187(gfu?)	7.01-7.02
5C04D02	T14702 T14703	no significant similarity			Rare	Complex	uaz279(gfu?)	4.08,5.07
5C04D07	T14709 T14710	rice dbEST		D24735 D23733	Rare	Simple	uaz207(gfu?)	2.05
5C01F06, 5C04E03	T14717 T14718	*Arabidopsis* dbEST	178X	40286	Rare	Simple	uaz209(gfu?)	6.06
5C04F03	T18692 T18693	no significant similarity			Rare	Simple	uaz211(gfu?)	5.02-5.03
5C04F06, 5C05G10 5C10F02	T14731 T25231	yeast ORF YBL026	111X	Z35787	Rare	Simple	uaz212(gfu?)	4.03-4.04
5C04F09	T18699	rice dbEST		D23229	Rare	Simple	uaz271(gfu?)	3.10
5C04H05, 5C04E10 5C04H07, 5C09G11	T14742 T14728 T14744 T23407	no significant similarity			Rare	Simple	uaz278(gfu?)	7.05
5C05B06	T15275	no significant similarity			Rare	Simple	uaz256(gfu?)	6.08
5C05C08	T15279	no significant similarity			Rare	Simple	uaz258(gfu?)	6.01,8.05
5C05D02	T14782	no significant similarity			Rare	Complex	uaz267(gfu?)	1.02
5C05D08, 5C10E12	T15286 T25230	*Arabidopsis* dbEST	187X	48449	Rare	?		
5C05E08	T15293	rice dbEST *Arabidopsis* dbEST	298X 269X	38193 21098	Rare	?		
5C05F04	T15297	rice dbEST		D22938	Rare	Simple	uaz262(gfu?)	2.06
5C01E08, 5C04A09, 5C05G02	T15304 T14664	no significant similarity			Rare	Simple	uaz349(gfu?)	4.05-4.06
5C05F08	T15300	*Arabidopsis* dbEST	130X	52289	Rare	?		
5C05H10, 5C07H02	T15314 T23334	*Arabidopsis* dbEST	>42X	33669	Rare	Complex	uaz268(gfu?)	1.11,5.13 7.01-7.02
5C06A03	T18280	no significant similarity			Rare	Simple	uaz307(gfu?)	8.01
5C06C11	T18295	no significant similarity			Rare	Simple	uaz302(gfu?)	2.06
5C06E09	T18305	no significant similarity			Rare	Simple	uaz311(gfu?)	1.05-1.06
5C06G02, 5C08E06	T18309 T23362	no significant similarity			Rare	Simple	uaz314(gfu?)	1.02

5C06G11	T18317	no significant similarity			Rare	Simple	uaz316(gfu?)	1.02
5C07A01					Rare	Simple	uaz330(gfu)	6.01
5C07B03	T18634	no significant similarity			Rare	Simple	uaz317(gfu?)	9.09
5C07B04	T18635	no significant similarity			Rare	Simple	uaz323(gfu?)	?
5C07B09	T18638	no significant similarity			Rare	Simple	uaz333(gfu?)	8.08,?
5C07B10					Rare	Simple	uaz325(gfu)	5.05
5C07B12	?	no significant similarity			Rare	Simple	uaz343(gfu?)	4.07
5C07C01	T18640	no significant similarity			Rare	Simple	uaz326(gfu?)	1.04
5C07C02					Rare	Simple	uaz334(gfu)	2.08
5C07C05					Rare	Simple	uaz328(gfu)	8.08
5C07C11					Rare	Simple	uaz336(gfu)	6.01
5C07H06	T23336	human dbEST	>41X	27225	Rare	?		
5C10A12	T25213	*Arabidopsis* dbEST rice dbEST	505N 415N	T21425 D23534	Rare	?		
5C10D01	T25219	rice dbEST	548N	D15308	Rare	?		
5C10D12	T25222	rice dbEST	400N	D23153	Rare	?		
5C10F02	T25231	yeast ORF	111X	Z35787	Rare	?		
5C11B12	T25256	*Arabidopsis* dbEST	560N	T21425	Rare	?		
6C01B07	T20375	no significant similarity			Rare	Simple	uaz174(gfu?)	4L,8.09
6C01B09	T20376	no significant similarity			Rare	Complex	uaz175(gfu?)	4.08,10.04
6C01C11	T20380	rice cDNA	80%	D24362	Abnd/SE	Simple	uaz344(rest)	2.05,7.01-7.02
6C01D08	T20397	no significant similarity			Abnd/S	Simple	uaz140(gfu?)	2.09
6C01E05					Rare	Simple	uaz125(gfu)	9.05-9.06
6C01E10					Rare	Complex	uaz176(gfu)	3.07,8.07-8.08
6C01F02	T20353	no significant similarity			Abnd/S	Simple	uaz162(gfu?)	6.01
6C01F06	T20354	no significant similarity			Abnd/S	Complex	uaz164(gfu?)	3.03,3.08, 5.07,8.06
6C01G04	T20360	*Arabidopsis* dbEST	70%	Z24464	Abnd/SE	?		
6C01G05	T20391	no significant similarity			Rare	Complex	uaz138(gfu?)	1.09,5.07 8.07
6C01H07	T20395	rice dbEST	83%	D23915	Abnd/SE	?		
6C02F05	T18425	no significant similarity			Rare	Simple	uaz318(gfu?)	2.02-2.03,10.08
6C03A07	T25274	*Arabidopsis* dbEST rice dbEST	524X 1117N	P29511 Z11931	Abnd/SE	Complex	uaz163(gfu)	5.02-5.03
6C03A08					Abnd/SE	Complex	uaz134(gfu)	5.01
6C03G05					Abnd/S	Simple	uaz135(gfu)	2.06
6C04B04	T25275	no significant similarity			Abnd/S	Simple	uaz178(gfu)	10.02-10.03
6C04B05	T25276	no significant similarity			Rare	Simple	uaz165(gfu)	8.04-8.05
6C04B11					Abnd/SE	Simple	uaz166(gfu)	1.12,5.02-5.03
6C04C04	T25278	no significant similarity			Abnd/SE	Simple	uaz143(gfu)	7.01-7.02
6C04C05					Abnd/SE	Complex	uaz180(gfu)	4.02
6C04C06					Abnd/SE	Simple	uaz142(gfu)	4.05-4.06
6C04C08					Rare	Simple	uaz181(gfu)	2.06
6C04D10					Abnd/S	Simple	uaz137(gfu)	4.05-4.06
6C04E01					Abnd/SE	Simple	uaz139(gfu)	1.03
6C04E06	T25279	no significant similarity			Abnd/SE	Simple	uaz167(gfu)	1.11,5.02-5.03
6C04F04					Abnd/E	Simple	uaz141(gfu?)	9.03
6C06A03	T15340	*C. elegans* ORF	108X	L14745	Abnd/S	Simple	uaz136(cest)	7.03
6C06A10					Rare	Simple	uaz133(gfu)	3.09
6C06C06	T15327	*E. coli* ORF	125X	P22564	Rare	Complex	-	
csu3	T12525 T12526	no significant similarity			-	Simple	csu3(gfu?)	1.05
csu4	T12527	no significant similarity			-	Simple	csu4(gfu?)	2.06
csu6	T12528 T12529	rice dbEST		D22140	-	Complex	csu6(gfu?)	10.04
csu7	T12530 T12531	rice dbEST		D24964 D24860 D24862	-	Complex	csu7(gfu?)	1.15,7.01-7.02
csu9	T12534	no significant similarity			-	Simple	csu9(gfu?)	2.10
csu10	T12535 T12536	no significant similarity			-	Simple	csu10(gfu?)	5.02-5.03
csu11	T12653 T12654	no significant similarity			-	Simple	csu11(gfu?)	7.01-7.02
csu29	T12666	no significant similarity			-	Simple	csu29(gfu?)	8.02
csu31	T12667 T12668	no significant similarity			-	Simple	csu31(gfu?)	8.07-8.08
csu32	T12669	*Arabidopsis* dbEST		27017	-	Simple	csu32(gfu?)	3.01
csu33	T12670	no significant similarity			-	Complex	csu33(gfu?)	5.01
csu46	T12676	rice dbEST		D24411	-	Simple	csu46(gfu?)	2.06
csu48	T12677 T12678	*Arabidopsis* dbEST	70%	Z17560	-	Complex	csu48(gfu?)	5.02-5.03
csu49	T12679 T12680	no significant similarity			-	Simple	csu49(gfu?)	2.06
csu50	?	no significant similarity			-	Simple	csu50(gfu?)	9.07-9.08
csu54	T12684	no significant similarity			-	Simple	csu54(gfu?)	2.08

csu58	T12688	no significant similarity			-	Simple	csu58(gfu?)	9.05-9.06
csu60	T12690	no significant similarity			-	Simple	csu60(gfu?)	1.05
csu1	T12705	no significant similarity			-	Complex	csu01(gfu?)	4.05-4.06
csu93	T12714	no significant similarity			-	Simple	csu93(gfu?)	7.01-7.02
csu95	T12716	rice dbEST		D15970	-	Simple	csu95(gfu?)	9.01
csu100	T12717	no significant similarity			-	Complex	csu100(gfu?)	4.05-4.06
csu103	T12719	rice dbEST	65%	D24020	-	Simple	csu103(gfu?)	10.02-10.03
csu147	T12740	no significant similarity			-	Complex	csu147(gfu?)	2.06
csu163	T12747	no significant similarity			-	Simple	csu163(gfu?)	8.09
csu166	T12749	no significant similarity			-	Complex	csu166(gfu?)	2.10,4.05-4.06
csu168	?	no significant similarity			-	Simple	csu168(gfu?)	5.05
csu171	T12752	rice dbEST	84%	D15402	-	-		
csu217	T18798	*Arabidopsis* dbEST	66%	T04175	-	-		
csu222	T18802	rice dbEST	83%	D26538	-	-		
csu244	T18817	rice dbEST	85%	D23592	-	-		
csu245	T18818	*Arabidopsis* dbEST	66%	Z17549	-	-		
csu249	T18819	rice dbEST	80%	D22191	-	-		
csu268	T18830	rice dbEST	67%	D15599	-	-		
rsp31		no significant similarity			-	Simple	uaz105(gfu?)	1.05
rsp48		no significant similarity				Complex	uaz048(gfu?)	1.01
rsp54		no significant similarity			-	Complex	uaz107(gfu?)	4.01
rsp84		no significant similarity			-	Complex	uaz106(gfu?)	2.04,6.02-6.03
spf16		no significant similarity			-	Complex	uaz31(gfu?)	2.08,7.04 9.07-9.08
spf29		no significant similarity			-	Simple	uaz11(gfu?)	1.04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4.4 npi97a
5.6
18.7 umc157
0.9 ztda50, php20640, umc115, umc194a (gpr)
1.0 umc194c (gpr)
1.02
14.2
1.7 umc243b
36.5 umc76
2.9 npi425c, umc230
0.9 umc11
9.7
1.03
asg26
1.9 asg35
1.9 npi286
1.2 p1
4.8
59.8 asg45
1.9 asg69, bnl12.06a, umc227
2.9 asg75
1.0 asg30
1.04
12.2
npi262
5.1
82.9 csu3 (gfu) asg3
11.0
1.05
umc167, umc260
13.0
106.9 umc67
3.3 umc177a
0.9 asg11, umc196 (gfu)
0.9 bnl5.59
0.9 csu92
1.9 asg58
9.6
1.06
umc119, umc58
5.0
php20644
8.0
137.4 asg62
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ON 1S
car1
cfr1
ct2 dek32
ms9 emb1
emb4
emb7
emb10
13* cat2 emp1
hcf6
hcf31
l16
les5
20* gsr1 rab30 lls1
ms28
pg15
T1-2c ws4
T1-2(4464) ys2
29 sr1 les2
30* vp5
T1-3(5597)
T1-3(5982)
blh1
rth3
39* hsp26
50 zb4 T1-4b
52 ms17 pdc3
53* ts2 sod4
55* p1 , hcf3
56 dek1 T1-9c
T1-2b
T1-5(6899)
63 nec2 T1-6c
T1-9(8918)
T1-3k
T1-5b
T1-3a
T1-9a
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ms14
85 as1 rs2
obf1
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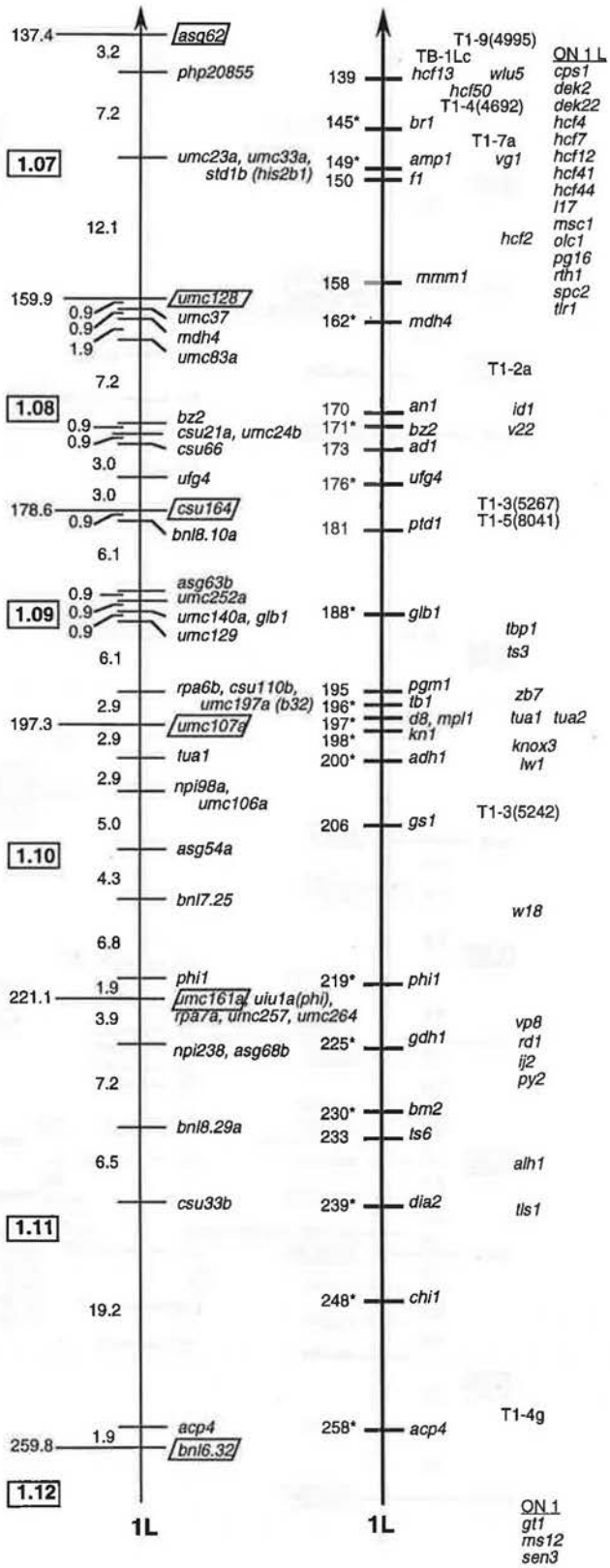
T1-3e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0.9 mdh4
1.9 umc83a
7.2
1.08
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0.9 csu66
3.0 ufg4
3.0
178.6 csu164
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6.1
asg63b
0.9 umc252a
1.09
0.9 umc140a, glb1
0.9 umc129
6.1
rpa6b, csu110b, umc197a (b32)
2.9
197.3 umc107a
2.9 tua1
2.9 npi98a, umc106a
5.0
1.10
asg54a
4.3
bnl7.25
6.8
1.9 phi1
221.1 umc161a, uiu1a(phi), rpa7a, umc257, umc264
3.9
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7.2
bnl8.29a
6.5
csu33b
1.11
19.2
1.9 acp4
259.8 bnl6.32
1.12
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ON 1 L
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hcf50 dek2
T1-4(4692)
145* br1 dek22
T1-7a hcf4
149* amp1 vg1 hcf7
150 f1 hcf12
hcf41
hcf44
l17
hcf2 msc1
olc1
158 mmm1 pg16
rth1
162* mdh4 spc2
tlr1
T1-2a
170 an1 id1
171* bz2 v22
173 ad1
176* ufg4
T1-3(5267)
181 ptd1 T1-5(8041)
188* glb1
tbp1
ts3
195 pgm1 zb7
196* tb1
197* d8, mpl1 tua1 tua2
198* kn1 knox3
200* adh1 lw1
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206 gs1
w18
219* phi1
vp8
225* gdh1 rd1
ij2
py2
230* bm2
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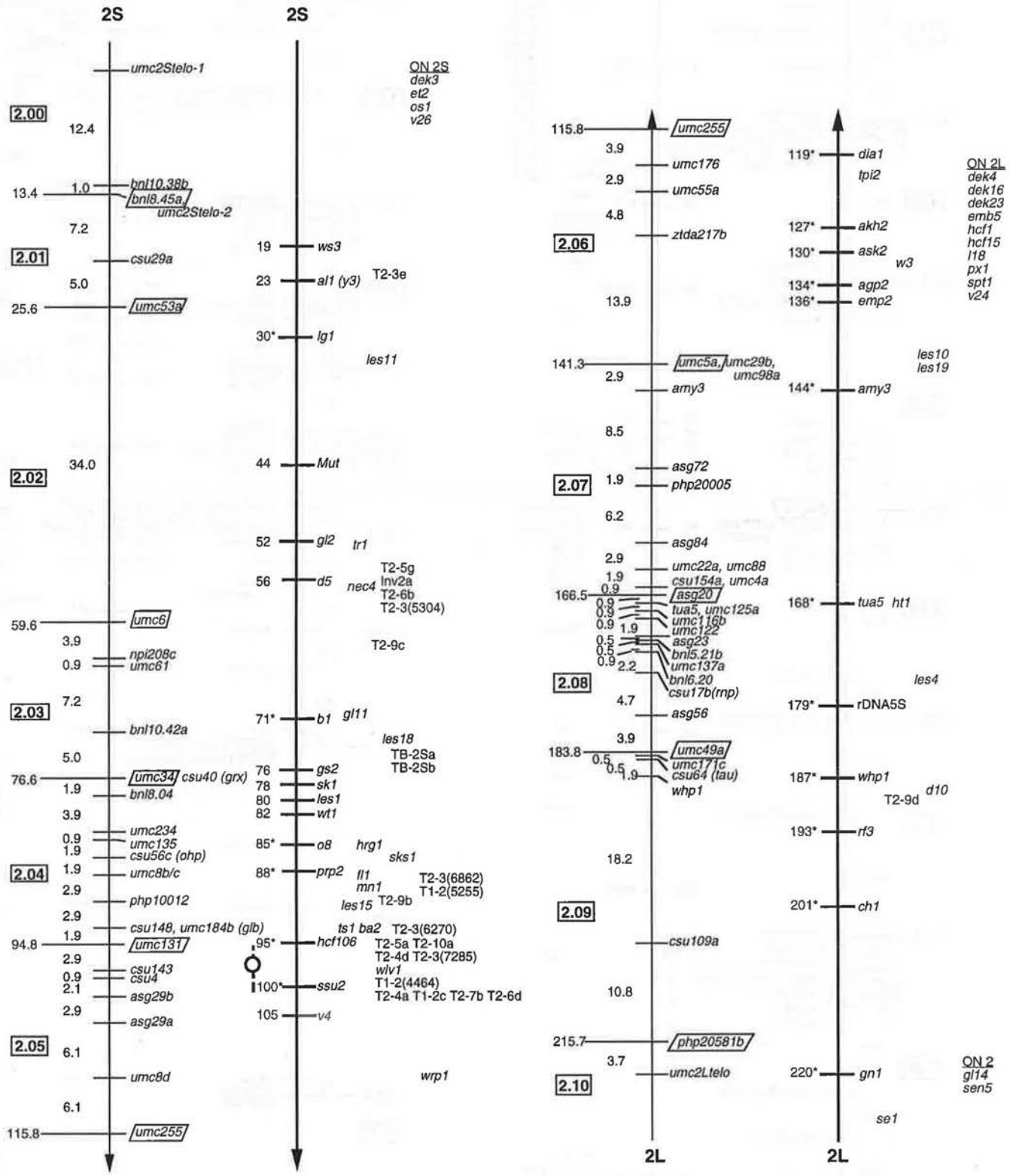
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3.9
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0.9
umc135
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csu56c (ohp)
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umc8b/c
2.9
php10012
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csu148, umc184b (glb)
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94.8
umc131
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csu143
0.9
csu4
2.1
asg29b
2.9
asg29a
2.05
6.1
umc8d
6.1
115.8
umc255
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ON 2S
dek3
et2
os1
v26
19
ws3
23
al1 (y3)
T2-3e
30*
lg1
les11
44
Mut
52
gl2
tr1
T2-5g
56
d5
nec4
Inv2a
T2-6b
T2-3(5304)
T2-9c
71*
b1
gl11
les18
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TB-2Sb
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gs2
78
sk1
80
les1
82
wt1
85*
o8
hrg1
sks1
88*
prp2
ll1
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mn1
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les15
T2-9b
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T2-4d T2-3(7285)
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ssu2
T1-2(4464)
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v4
wrp1
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umc255
3.9
umc176
2.9
umc55a
4.8
2.06
ztda217b
13.9
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umc98a
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amy3
8.5
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2.07
1.9
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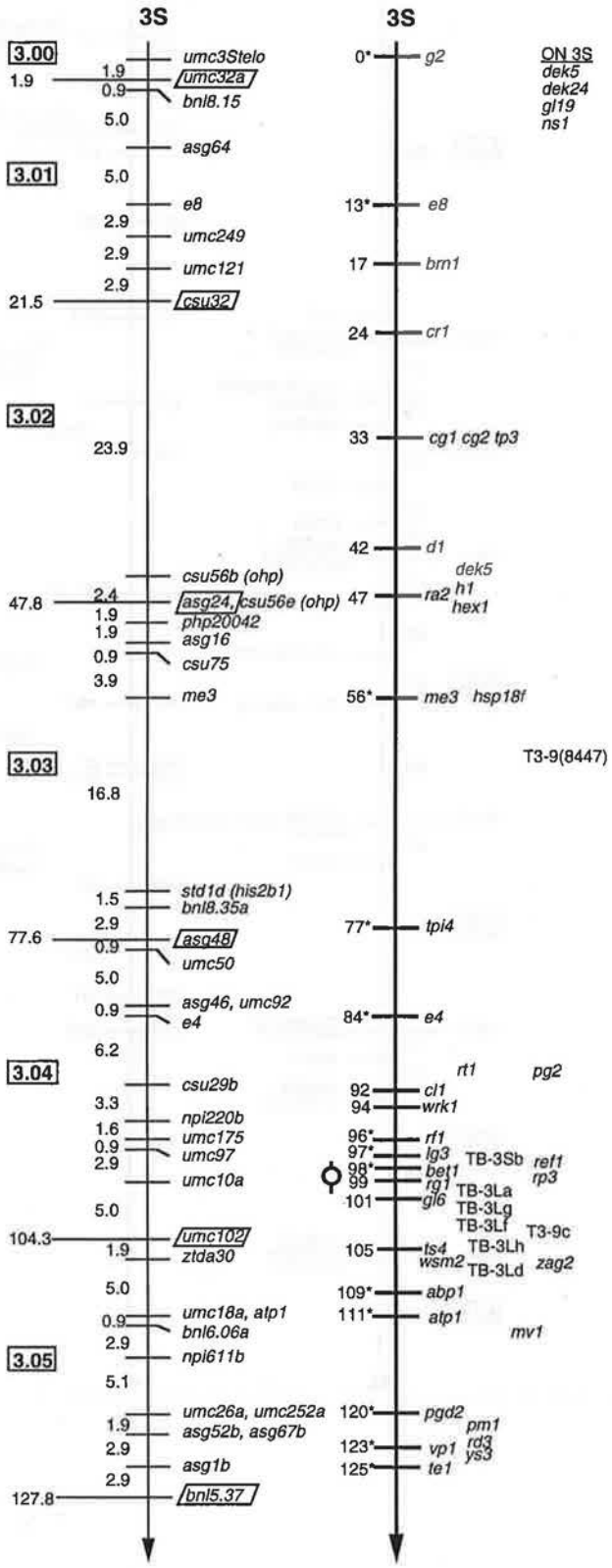
6.2
asg84
2.9
umc22a, umc88
1.9
csu154a, umc4a
0.9
166.5
asg20
0.9
tua5, umc125a
0.9
umc116b
0.9
1.9
umc122
0.5
asg23
0.5
bnl5.21b
0.9
2.2
umc137a
bnl6.20
2.08
csu17b(rnp)
4.7
asg56
3.9
183.8
umc49a
0.5
umc171c
0.5
1.9
csu64 (tau)
whp1
18.2
2.09
csu109a
10.8
215.7
php20581b
3.7
2.10
umc2Ltelo
2L
119*
dia1
tpi2
ON 2L
dek4
dek16
dek23
emb5
hcf1
hcf15
l18
px1
spt1
v24
127*
akh2
130*
ask2
w3
134*
agp2
136*
emp2
les10
les19
144*
amy3
168*
tua5
ht1
les4
179*
rDNA5S
187*
whp1
d10
T2-9d
193*
rf3
201*
ch1
220*
gn1
ON 2
gl14
sen5
se1
2L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3S
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umc3Stelo
1.9
umc32a
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bnl8.15
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asg64
3.01
5.0
e8
2.9
umc249
2.9
umc121
2.9
21.5
csu32
3.02
23.9
csu56b (ohp)
2.4
47.8
asg24, csu56e (ohp)
1.9
php20042
1.9
asg16
0.9
csu75
3.9
me3
3.03
16.8
1.5
std1d (his2b1)
bnl8.35a
2.9
77.6
asg48
0.9
umc50
5.0
0.9
asg46, umc92
e4
6.2
3.04
csu29b
3.3
npi220b
1.6
umc175
0.9
umc97
2.9
umc10a
5.0
104.3
umc102
1.9
ztda30
5.0
0.9
umc18a, atp1
bnl6.06a
2.9
3.05
npi611b
5.1
umc26a, umc252a
1.9
asg52b, asg67b
2.9
asg1b
2.9
127.8
bnl5.37
0*
g2
ON 3S
dek5
dek24
gl19
ns1
13*
e8
17
brn1
24
cr1
33
cg1 cg2 tp3
42
d1
dek5
47
ra2
h1
hex1
56*
me3
hsp18f
T3-9(8447)
77*
tpi4
84*
e4
rt1
pg2
92
cl1
94
wrk1
96*
rf1
97*
lg3
TB-3Sb
98*
bet1
ref1
99
rg1
rp3
101
gl6
TB-3La
TB-3Lg
TB-3Lf
T3-9c
105
ts4
TB-3Lh
wsm2
TB-3Ld
zag2
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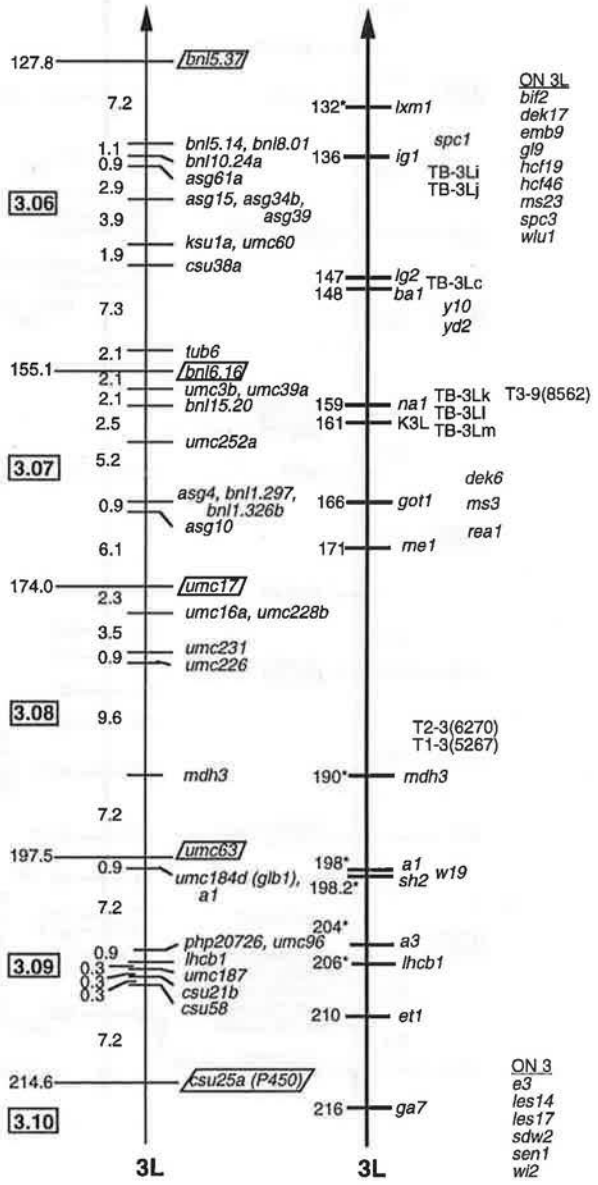
pm1
123*
vp1
rd3
ys3
125*
te1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bnl10.24a
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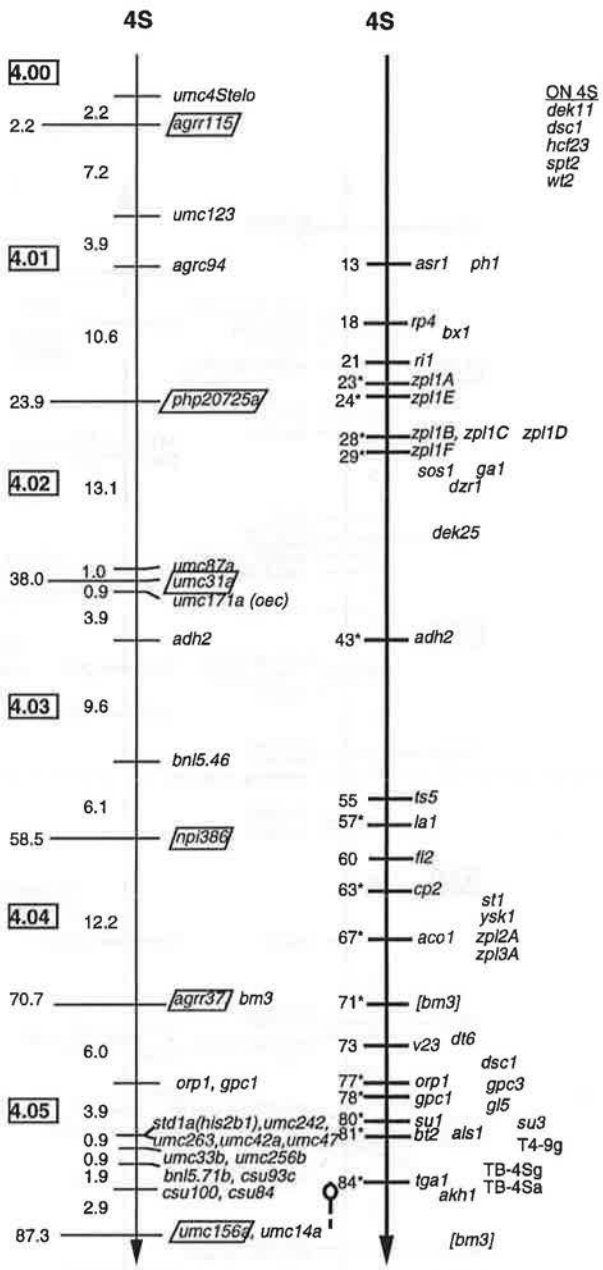
3.06
asg15, asg34b,
asg39
3.9
1.9
ksu1a, umc60
csu38a
7.3
2.1
tub6
155.1
bnl6.16
2.1
umc3b, umc39a
2.1
bnl15.20
2.5
umc252a
5.2
3.07
asg4, bnl1.297,
0.9
bnl1.326b
asg10
6.1
174.0
umc17
2.3
umc16a, umc228b
3.5
umc231
0.9
umc226
3.08
9.6
mdh3
7.2
197.5
umc63
0.9
umc184d (glb1),
a1
7.2
0.9
php20726, umc96
3.09
0.3
lhcb1
0.3
umc187
0.3
csu21b
0.3
csu58
7.2
214.6
csu25a (P450)
3.10
3L
132*
lxm1
ON 3L
bif2
dek17
emb9
gl9
hcf19
hcf46
ms23
spc3
wlu1
spc1
136
ig1
TB-3Li
TB-3Lj
147
lg2
TB-3Lc
148
ba1
y10
yd2
159
na1
TB-3Lk
T3-9(8562)
161
K3L
TB-3Ll
TB-3Lm
dek6
166
got1
ms3
rea1
171
me1
T2-3(6270)
T1-3(5267)
190*
mdh3
198*
a1
w19
198.2*
sh2
204*
a3
206*
lhcb1
210
et1
ON 3
e3
les14
les17
sdw2
sen1
wl2
216
ga7
3L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4S
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umc4Stelo
2.2
2.2
agrr115
7.2
umc123
3.9
4.01
agrc94
10.6
23.9
php20725a
4.02
13.1
1.0
umc87a
38.0
umc31a
0.9
umc171a (oec)
3.9
adh2
4.03
9.6
bnl5.46
6.1
58.5
npi386
4.04
12.2
70.7
agrr37
bm3
6.0
orp1, gpc1
4.05
3.9
std1a(his2b1),umc242,
0.9
umc263,umc42a,umc47
0.9
umc33b, umc256b
1.9
bnl5.71b, csu93c
csu100, csu84
2.9
87.3
umc156a, umc14a
ON 4S
dek11
dsc1
hcf23
spt2
wt2
13
asr1
ph1
18
rp4
bx1
21
ri1
23*
zpl1A
24*
zpl1E
28*
zpl1B, zpl1C
zpl1D
29*
zpl1F
sos1
ga1
dzr1
dek25
43*
adh2
55
ts5
57*
la1
60
fl2
63*
cp2
st1
ysk1
67*
aco1
zpl2A
zpl3A
71*
[bm3]
73
v23
dt6
dsc1
77*
orp1
gpc3
78*
gpc1
gl5
80*
su1
su3
81*
bt2
als1
T4-9g
TB-4Sg
84*
tga1
TB-4Sa
akh1
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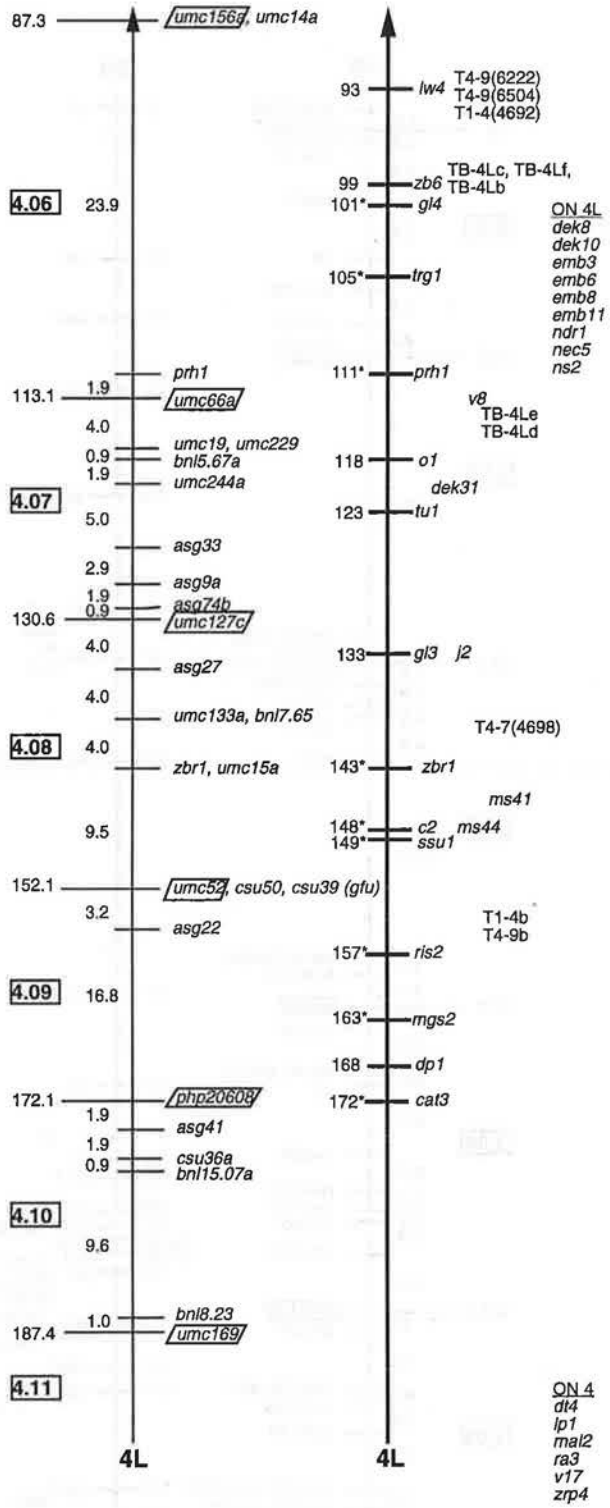
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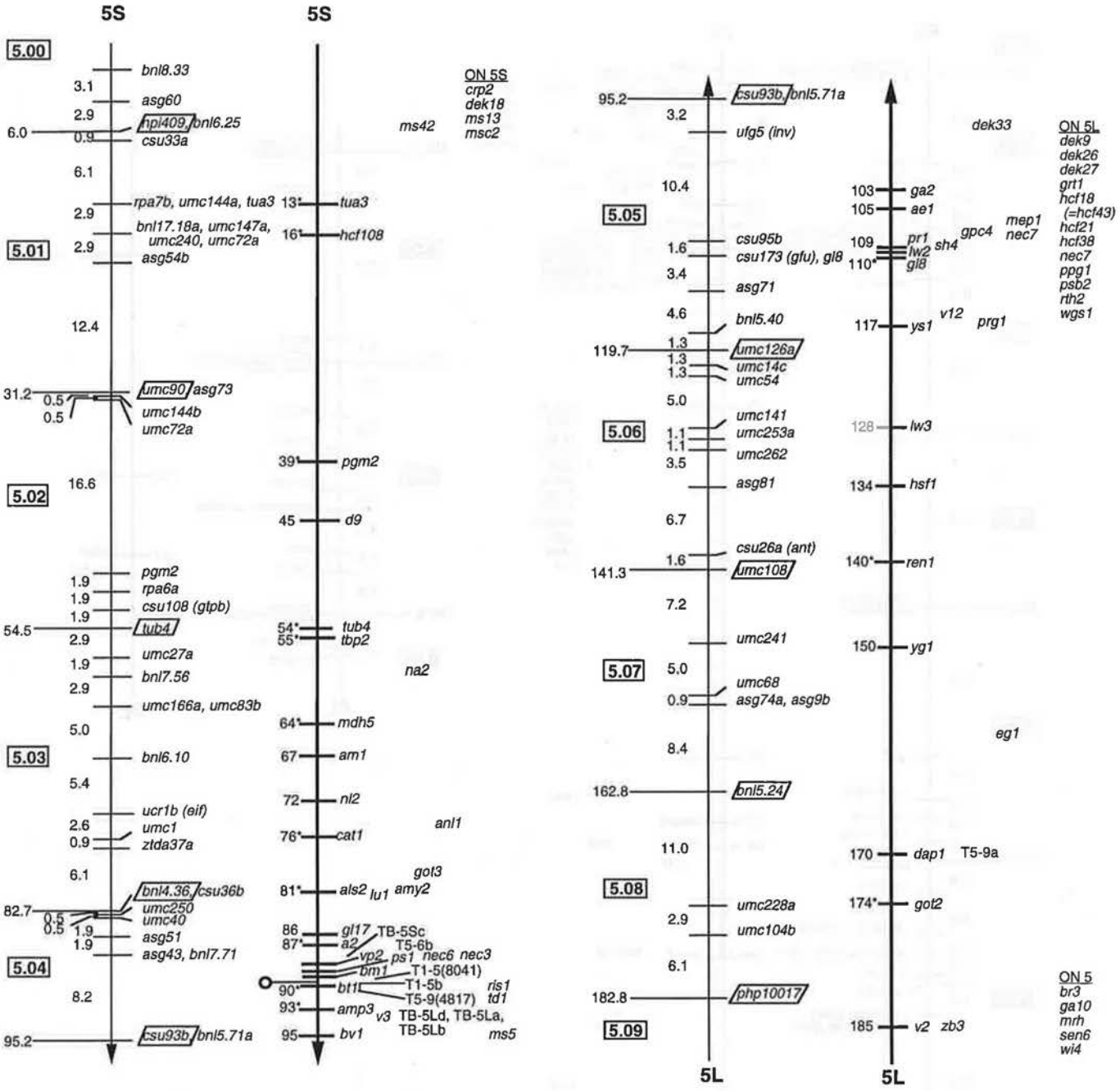
umc156a, umc14a
4.06
23.9
prh1
113.1
1.9
umc66a
4.0
umc19, umc229
0.9
bnl5.67a
1.9
umc244a
4.07
5.0
asg33
2.9
asg9a
1.9
asg74b
0.9
130.6
umc127c
4.0
asg27
4.0
umc133a, bnl7.65
4.08
4.0
zbr1, umc15a
9.5
152.1
umc52, csu50, csu39 (gfu)
3.2
asg22
4.09
16.8
172.1
php20608
1.9
asg41
1.9
csu36a
0.9
bnl15.07a
4.10
9.6
1.0
bnl8.23
187.4
umc169
4.11
4L
93
lw4
T4-9(6222)
T4-9(6504)
T1-4(4692)
99
zb6
TB-4Lc, TB-4Lf,
TB-4Lb
101*
gl4
ON 4L
dek8
dek10
emb3
105*
trg1
emb6
emb8
emb11
ndr1
nec5
ns2
111*
prh1
v8
TB-4Le
TB-4Ld
118
o1
dek31
123
tu1
133
gl3
j2
T4-7(4698)
143*
zbr1
ms41
148*
c2
ms44
149*
ssu1
157*
ris2
T1-4b
T4-9b
163*
mgs2
168
dp1
172*
cat3
4L
ON 4
dt4
lp1
mal2
ra3
v17
zrp4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6.1
rpa7b, umc144a, tua3
2.9
bnl17.18a, umc147a,
umc240, umc72a
5.01
2.9
asg54b
12.4
31.2
umc90, asg73
0.5
0.5
umc144b
umc72a
16.6
5.02
pgm2
1.9
rpa6a
1.9
csu108 (gtpb)
1.9
54.5
tub4
2.9
umc27a
1.9
bnl7.56
2.9
umc166a, umc83b
5.0
5.03
bnl6.10
5.4
ucr1b (eif)
2.6
umc1
0.9
ztda37a
6.1
82.7
bnl4.36, csu36b
0.5
umc250
0.5
umc40
1.9
1.9
asg51
asg43, bnl7.71
5.04
8.2
95.2
csu93b, bnl5.71a
13
tua3
16
hcf108
39
pgm2
45
d9
54
tub4
55
tbp2
64
mdh5
67
am1
72
nl2
76
cat1
81
als2
lu1
amy2
86
gl17
TB-5Sc
87
a2
T5-6b
vp2
ps1
nec6
nec3
bm1
T1-5(8041)
90
bt1
T1-5b
ris1
T5-9(4817)
td1
93
amp3
v3
TB-5Ld, TB-5La,
TB-5Lb
95
bv1
ms5
ON 5S
crp2
dek18
ms13
msc2
ms42
na2
anl1
got3
amy2
5L
95.2
csu93b, bnl5.71a
3.2
ufg5 (inv)
10.4
5.05
1.6
csu95b
csu173 (gfu), gl8
3.4
asg71
4.6
bnl5.40
1.3
119.7
umc126a
1.3
umc14c
1.3
umc54
5.0
5.06
umc141
1.1
umc253a
1.1
umc262
3.5
asg81
6.7
1.6
csu26a (ant)
141.3
umc108
7.2
umc241
5.07
5.0
umc68
0.9
asg74a, asg9b
8.4
162.8
bnl5.24
11.0
5.08
umc228a
2.9
umc104b
6.1
182.8
php10017
5.09
5L
dek33
103
ga2
105
ae1
mep1
gpc4
nec7
109
pr1
sh4
lw2
110
gl8
117
ys1
v12
prg1
128
lw3
134
hsf1
140
ren1
150
yg1
eg1
170
dap1
T5-9a
174
got2
185
v2
zb3
ON 5L
dek9
dek26
dek27
grt1
hcf18
(=hcf43)
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rth2
wgs1
ON 5
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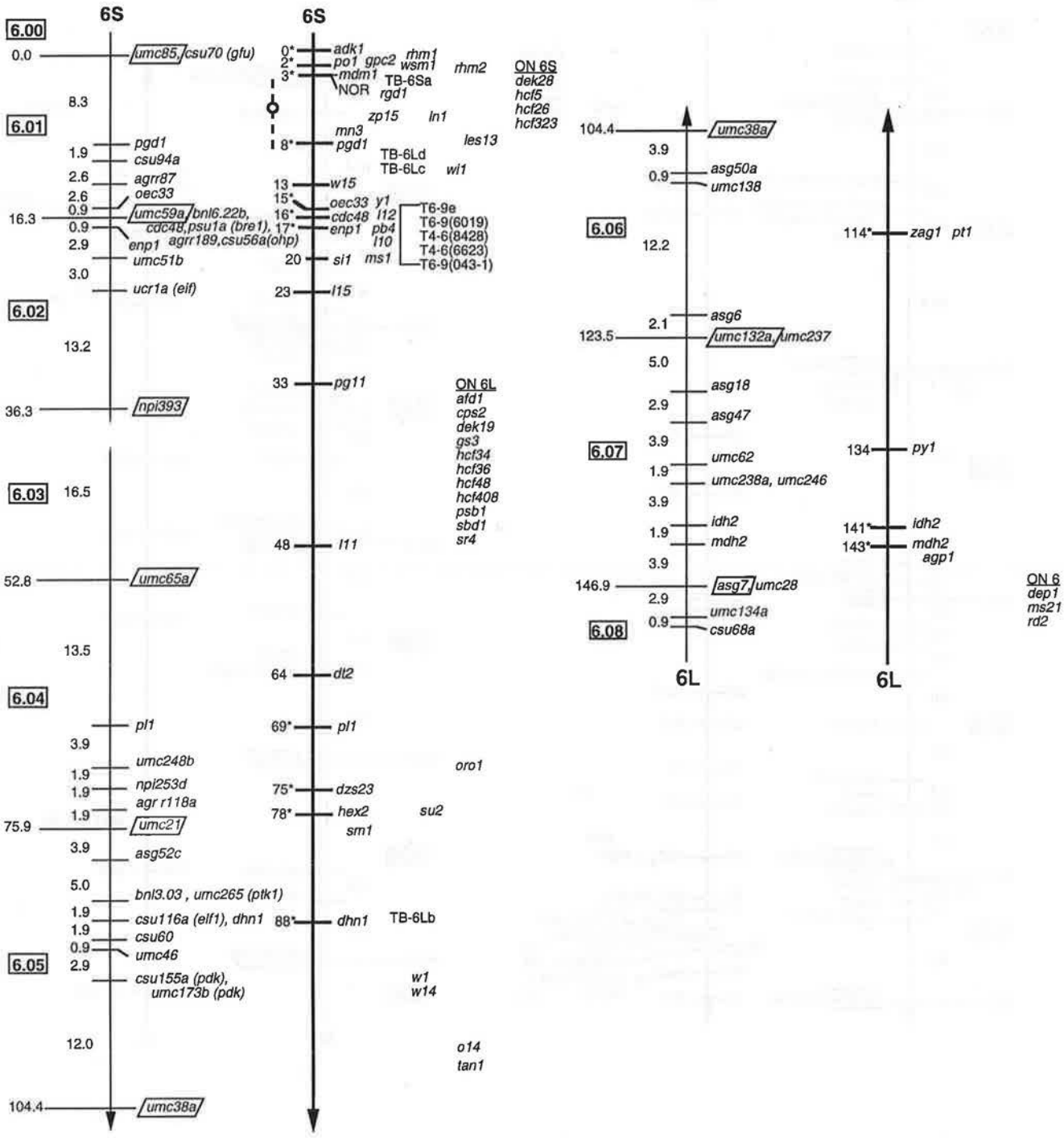
ga10
mrh
sen6
wi4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6L
6L
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6.02
6.03
6.04
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6.06
6.07
6.08
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pgd1
csu94a
agrr87
oec33
umc59a, bnl6.22b,
cdc48,psu1a (bre1),
agrr189,csu56a(ohp)
enp1
umc51b
ucr1a (eif)
npi393
umc65a
pl1
umc248b
npi253d
agr r118a
umc21
asg52c
bnl3.03 , umc265 (ptk1)
csu116a (elf1), dhn1
csu60
umc46
csu155a (pdk),
umc173b (pdk)
umc38a
adk1
po1 gpc2 rhm1
mdm1 wsm1 rhm2
NOR TB-6Sa
rgd1
zp15 ln1
mn3
pgd1 les13
TB-6Ld
TB-6Lc wl1
w15
oec33 y1
cdc48 l12
enp1 pb4
l10
si1 ms1
T6-9e
T6-9(6019)
T4-6(8428)
T4-6(6623)
T6-9(043-1)
l15
pg11
l11
dt2
pl1
dzs23
hex2 su2
sm1
dhn1 TB-6Lb
w1
w14
o14
tan1
ON 6S
dek28
hcf5
hcf26
hcf323
ON 6L
afd1
cps2
dek19
gs3
hcf34
hcf36
hcf48
hcf408
psb1
sbd1
sr4
oro1
umc38a
asg50a
umc138
asg6
umc132a, umc237
asg18
asg47
umc62
umc238a, umc246
idh2
mdh2
asg7, umc28
umc134a
csu68a
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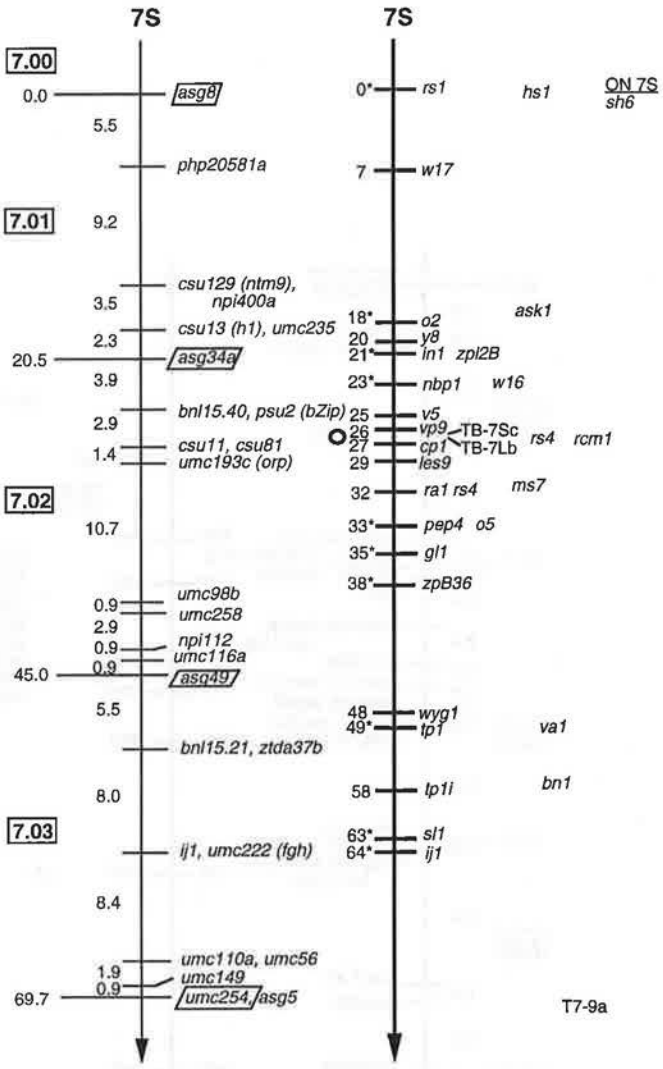
py1
idh2
mdh2
agp1
ON 6
dep1
ms21
rd2


7S
7S
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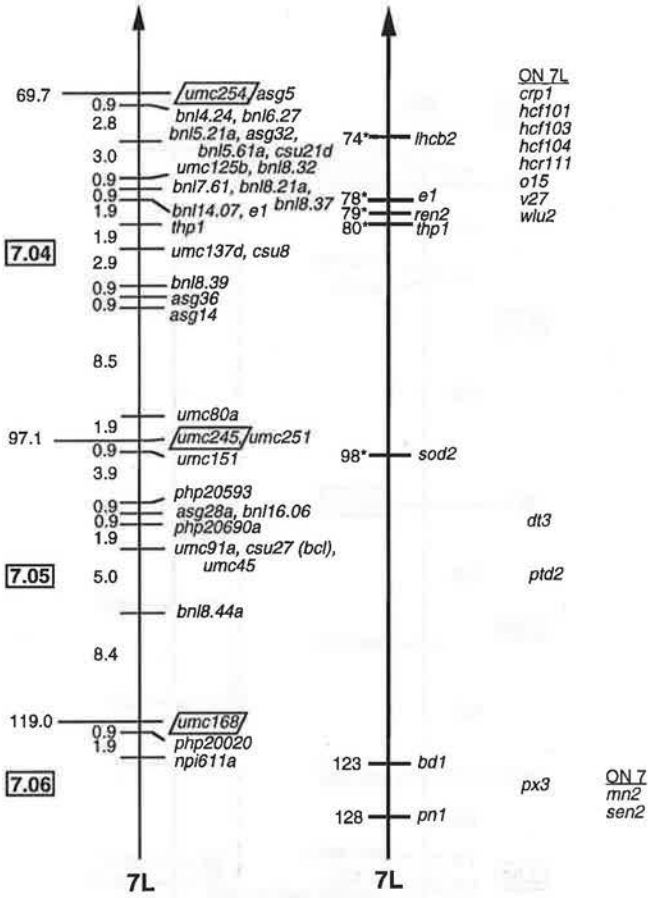
5.5
php20581a
7.01
9.2
3.5 csu129 (ntm9), npi400a
2.3 csu13 (h1), umc235
20.5 asg34a
3.9
2.9 bnl15.40, psu2 (bZip)
1.4 csu11, csu81
umc193c (orp)
7.02
10.7
0.9 umc98b
2.9 umc258
0.9 npi112
0.9 umc116a
45.0 asg49
5.5
bnl15.21, ztda37b
8.0
7.03
ij1, umc222 (fgh)
8.4
1.9 umc110a, umc56
0.9 umc149
69.7 umc254, asg5
0* rs1 hs1
ON 7S
sh6
7 w17
18* o2 ask1
20 y8
21* In1 zpl2B
23* nbp1 w16
25 v5
26 vp9 TB-7Sc
27 cp1 TB-7Lb rs4 rcm1
29 les9
32 ra1 rs4 ms7
33* pep4 o5
35* gl1
38* zpB36
48 wyg1
49* tp1 va1
58 tp1i bn1
63* sl1
64* ij1
T7-9a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2.8 bnl5.21a, asg32, bnl5.61a, csu21d
3.0 umc125b, bnl8.32
0.9 bnl7.61, bnl8.21a, bnl8.37
0.9 bnl14.07, e1
1.9 thp1
1.9 umc137d, csu8
7.04
2.9
0.9 bnl8.39
0.9 asg36
asg14
8.5
1.9 umc80a
97.1 umc245, umc251
0.9 umc151
3.9
php20593
0.9 asg28a, bnl16.06
0.9 php20690a
1.9 umc91a, csu27 (bcl), umc45
7.05 5.0
bnl8.44a
8.4
119.0 umc168
0.9 php20020
1.9 npi611a
7.06
7L
74* lhcb2
78* e1
79* ren2
80* thp1
98* sod2
123 bd1
128 pn1
7L
ON 7L
crp1
hcf101
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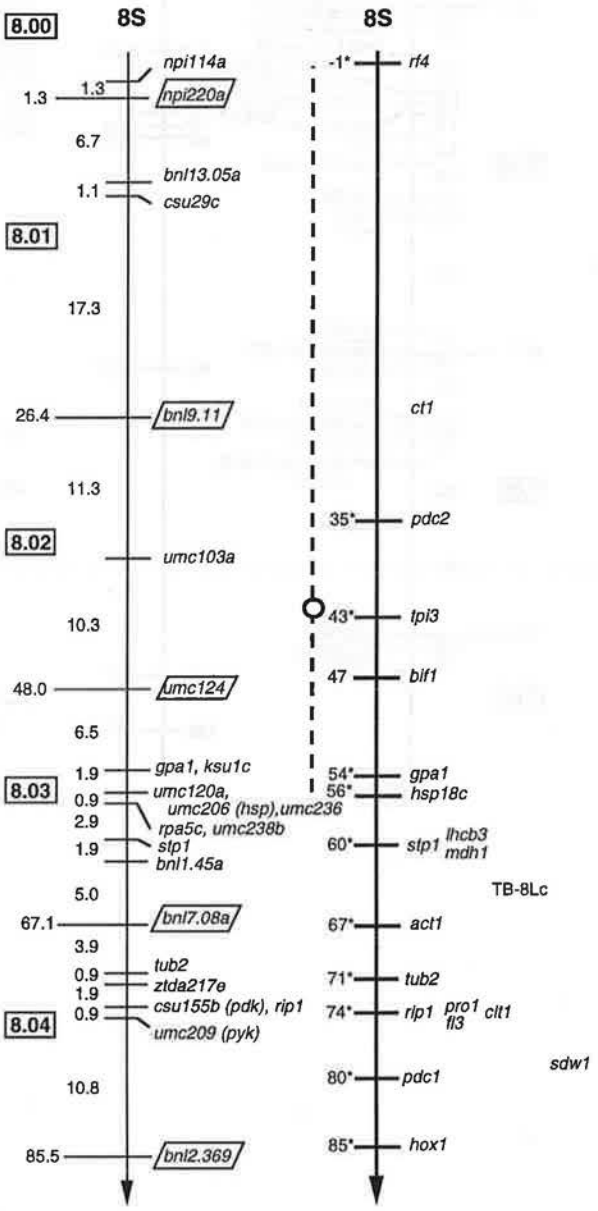
hcf104
hcr111
o15
v27
wlu2
dt3
ptd2
px3
ON 7
mn2
sen2


8.00
8S
8S
npi114a
1.3
1.3
npi220a
-1*
rf4
6.7
bnl13.05a
1.1
csu29c
8.01
17.3
26.4
bnl9.11
ct1
11.3
35*
pdc2
8.02
umc103a
10.3
43*
tpi3
48.0
umc124
47
bif1
6.5
1.9
gpa1, ksu1c
54*
gpa1
8.03
0.9
umc120a,
umc206 (hsp),umc236
56*
hsp18c
2.9
rpa5c, umc238b
60*
stp1
lhcb3
mdh1
1.9
stp1
bnl1.45a
TB-8Lc
5.0
67.1
bnl7.08a
67*
act1
3.9
0.9
tub2
71*
tub2
1.9
ztda217e
0.9
csu155b (pdk), rip1
74*
rip1
pro1
fl3
clt1
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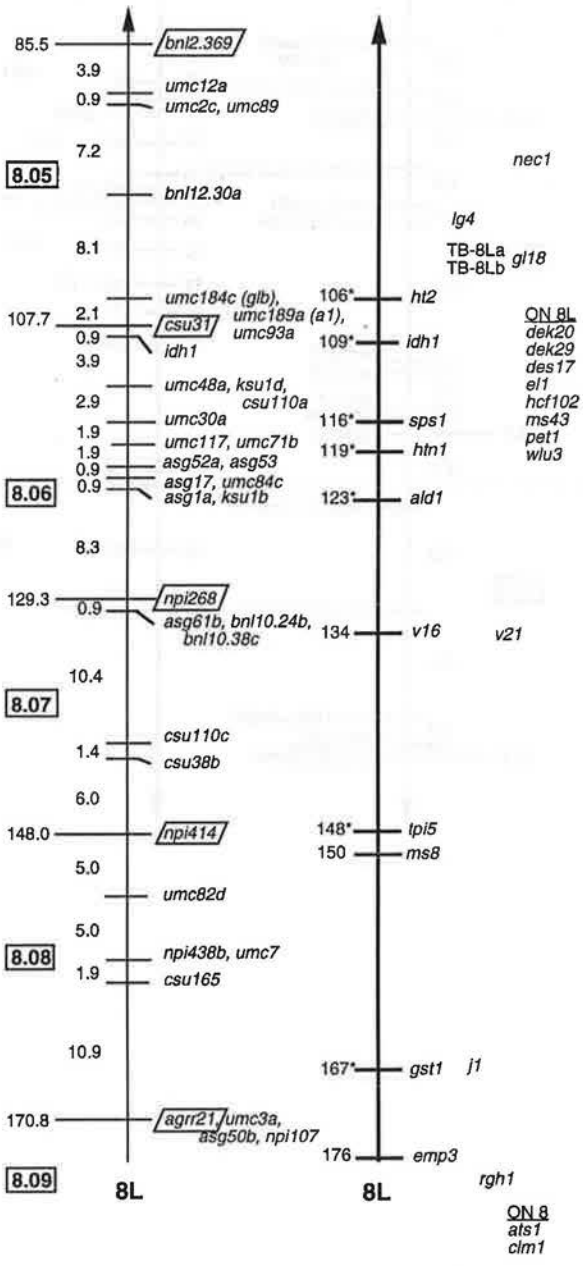
umc209 (pyk)
sdw1
10.8
80*
pdc1
85.5
bnl2.369
85*
hox1


85.5
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3.9
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0.9
umc2c, umc89
7.2
nec1
8.05
bnl12.30a
lg4
8.1
TB-8La
TB-8Lb
gl18
umc184c (glb),
106*
ht2
107.7
2.1
csu31
umc189a (a1),
umc93a
ON 8L
dek20
dek29
des17
el1
hcf102
ms43
pet1
wlu3
0.9
idh1
109*
idh1
3.9
umc48a, ksu1d,
csu110a
2.9
umc30a
116*
sps1
1.9
umc117, umc71b
119*
htn1
1.9
asg52a, asg53
0.9
asg17, umc84c
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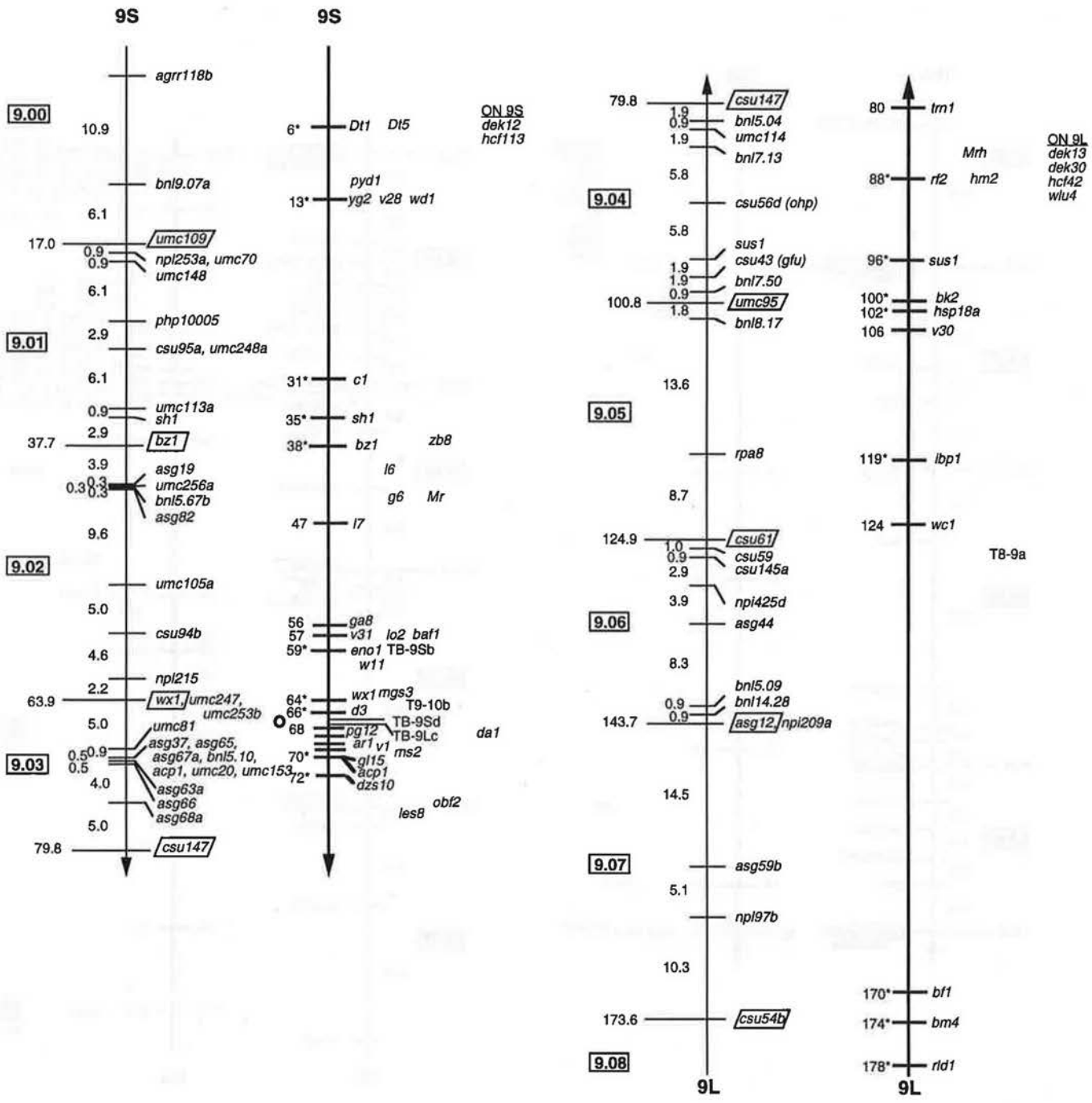
asg1a, ksu1b
8.06
123*
ald1
8.3
129.3
npi268
0.9
asg61b, bnl10.24b,
bnl10.38c
134
v16
v21
10.4
8.07
csu110c
1.4
csu38b
6.0
148.0
npi414
148*
tpi5
150
ms8
5.0
umc82d
5.0
8.08
npi438b, umc7
1.9
csu165
10.9
167*
gst1
j1
170.8
agrr21,
umc3a,
asg50b, npi107
176
emp3
rgh1
8.09
8L
8L
ON 8
ats1
clm1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37.7
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0.3
asg19
umc256a
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bnl5.67b
asg82
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umc105a
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csu94b
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63.9
wx1, umc247, umc253b
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9.03
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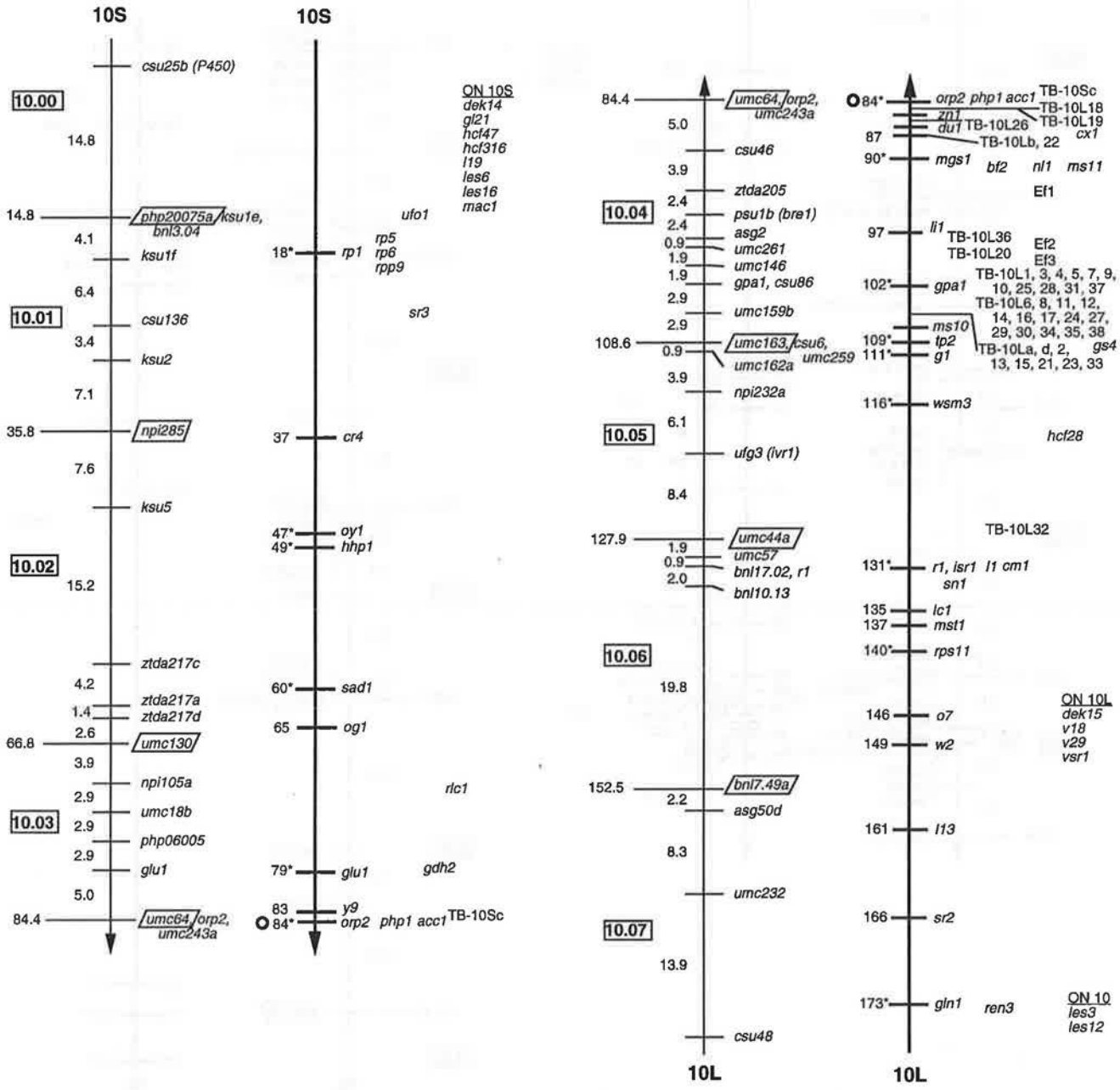
asg63a
asg66
asg68a
5.0
79.8
csu147
6*
Dt1 Dt5
ON 9S
dek12
hcf113
pyd1
13*
yg2 v28 wd1
31*
c1
35*
sh1
38*
bz1
zb8
l6
g6 Mr
47
l7
56
ga8
57
v31 lo2 baf1
59*
eno1 TB-9Sb
w11
64*
wx1 mgs3
66*
d3
T9-10b
68
TB-9Sd
pg12
TB-9Lc
ar1 v1 rms2
70*
gl15
72*
acp1
dzs10
obf2
les8
da1
79.8
csu147
1.9
0.9
bnl5.04
1.9
umc114
bnl7.13
5.8
9.04
csu56d (ohp)
5.8
sus1
1.9
csu43 (gfu)
1.9
bnl7.50
0.9
100.8
umc95
1.8
bnl8.17
13.6
9.05
rpa8
8.7
124.9
csu61
1.0
0.9
csu59
csu145a
2.9
3.9
npi425d
9.06
asg44
8.3
bnl5.09
0.9
bnl14.28
0.9
143.7
asg12, npi209a
14.5
9.07
asg59b
5.1
npi97b
10.3
173.6
csu54b
9.08
9L
80
tm1
Mrh
ON 9L
dek13
dek30
hcf42
wlu4
88*
rf2 hm2
96*
sus1
100*
bk2
102*
hsp18a
106
v30
119*
ibp1
124
wc1
T8-9a
170*
bf1
174*
bm4
178*
rld1
9L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6.4
10.01
csu136
3.4
ksu2
7.1
35.8
npi285
7.6
ksu5
10.02
15.2
ztda217c
4.2
ztda217a
1.4
ztda217d
2.6
66.8
umc130
3.9
npi105a
2.9
10.03
umc18b
2.9
php06005
2.9
glu1
5.0
84.4
umc64/orp2, umc243a
ON 10S
dek14
gl21
hcf47
hcf316
l19
les6
les16
mac1
ufo1
18*
rp1
rp5
rp6
rpp9
sr3
37
cr4
47*
oy1
49*
hhp1
60*
sad1
65
og1
rlc1
79*
glu1
gdh2
83
y9
84*
orp2
php1 acc1
TB-10Sc
84.4
umc64/orp2, umc243a
5.0
csu46
3.9
ztda205
2.4
10.04
psu1b (bre1)
2.4
asg2
0.9
umc261
1.9
umc146
1.9
gpa1, csu86
2.9
umc159b
2.9
108.6
umc163/csu6, umc259
0.9
umc162a
3.9
npi232a
6.1
10.05
ufg3 (ivr1)
8.4
127.9
umc44a
1.9
umc57
0.9
bnl17.02, r1
2.0
bnl10.13
10.06
19.8
152.5
bnl7.49a
2.2
asg50d
8.3
umc232
10.07
13.9
csu48
10L
84*
orp2 php1 acc1
TB-10Sc
zn1
TB-10L18
du1
TB-10L26
TB-10L19
cx1
87
TB-10Lb, 22
90*
mgs1
bf2
nl1
ms11
Ef1
97
li1
TB-10L36
TB-10L20
Ef2
Ef3
102*
gpa1
TB-10L1, 3, 4, 5, 7, 9, 10, 25, 28, 31, 37
TB-10L6, 8, 11, 12, 14, 16, 17, 24, 27, 29, 30, 34, 35, 38
ms10
109*
tp2
gs4
111*
g1
TB-10La, d, 2, 13, 15, 21, 23, 33
116*
wsm3
hcf28
TB-10L32
131*
r1, isr1
l1 cm1
sn1
135
lc1
137
mst1
140*
rps11
ON 10L
dek15
v18
v29
vsr1
146
o7
149
w2
161
l13
166
sr2
173*
gln1
ren3
ON 10
les3
les12
10L

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 and Inbred. 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 by the U.S. Department of Energy Division of Energy Biosciences and the Maize Genome Database Project.

To obtain postscript files of the March 15, 1995 BNL RI maps:

ftp to bnlux1.bnl.gov (130.199.128.1)
login as "anonymous"
for password, type in your name
cd maize
mget c*.ps
quit

These files must be printed on legal (35.3 ~ 21.4 cm) sheets.

Eileen C. Matz, Frances A. Burr, and Benjamin Burr, Brookhaven National Laboratory

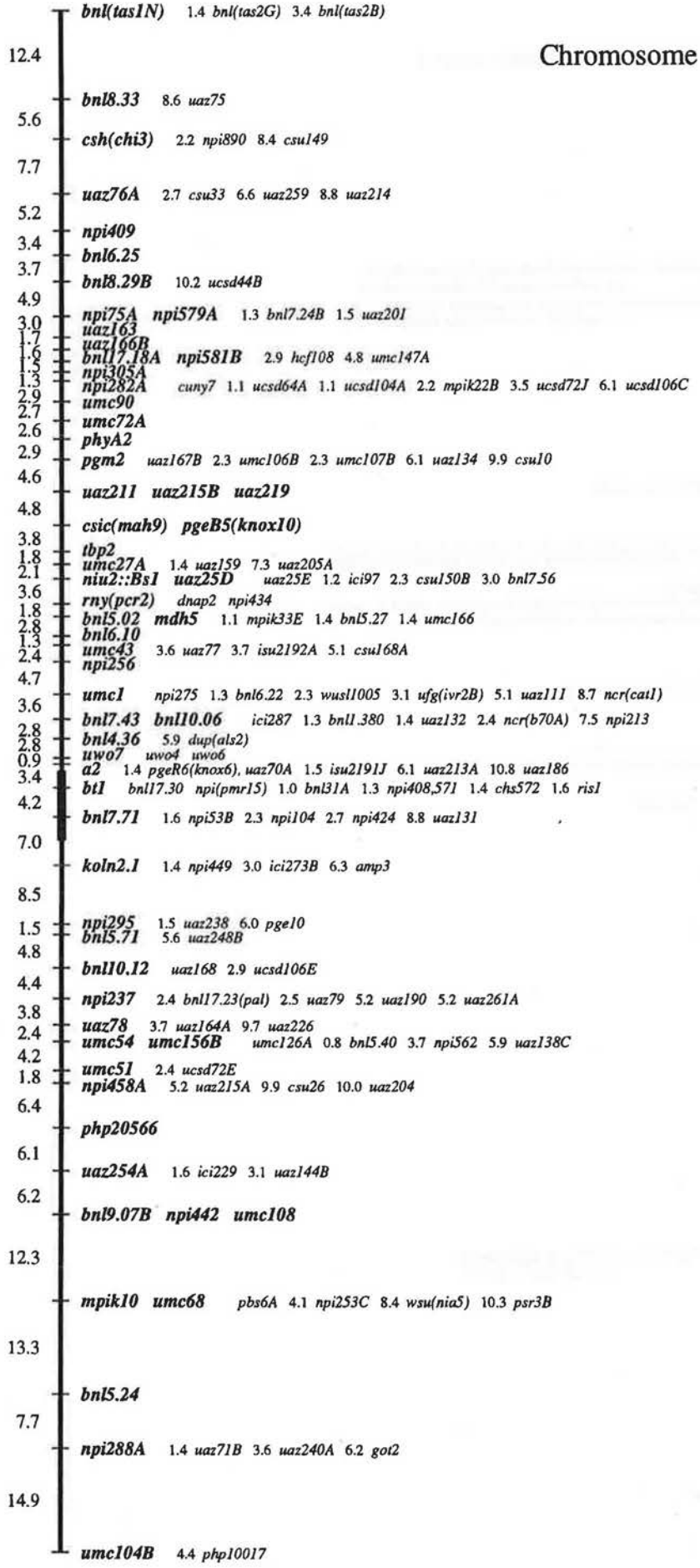
Chromosome 1

Chromosome 2

Chromosome 3

Chromosome 4

Chromosome 5



Chromosome 6
npi340A umc159 1.1 uaz18A 7.4 bnl16l
4.5
nor umc85 mpik(DH7),mpik11B,uiu(pog1B);2.3 bnl17.28;3.6 uaz150,197B;3.7 uaz102;8.1 csu71A;8.4 csu150A
2.4
bnl6.29 cdo580H tug2 1.4 uaz269C 3.0 uaz258A 4.0 isu1410B 5.7 uaz80
0.6
npi235 2.9 isu2232H
4.5
bnl7.28
7.6
npi594A npi606 zp15 1.1 uaz23A ;1.2 isu1774A ;1.5 isu1410J ;2.4 mwg645B, uaz233B ;3.4 bcd98F
1.2
uaz169 ;2.5 uaz233D ;3.5 uaz237B ;5.0 uaz197A ;7.0 pbs8
4.4
enp1 csu146 1.0 y1 1.1 mpik33D 4.1 pgd1 9.1 uaz227
1.8
npi373 umc59 1.0 bnl28(sbe1) bnl107 1.1 mpik1 1.3 npi377 1.5 uaz162 4.6 php06007
2.7
mpik18
2.9
php20045
7.1
npi393
11.3
npi98B 2.3 npi294C 8.6 uaz106A
5.6
umc65 4.7 uaz161A
2.5
npi223
5.4
0.6
pll 7.6 tug8
umc113B
5.3
uaz160
5.4
dzs23
2.8
hex2 0.6 npi617 1.2 psr108 1.3 npi253D 1.4 tug6 2.5 ici96, uaz244A 2.7 pic2A 2.8 tug7 14.4 npi224l
1.1
ucsd64E ucsd78A 1.1 ucsd72l, 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 pge20 1.8 uaz220
2.1
npi616 0.6 npi252
1.2
umc46 0.6 dup400(pac)
1.8
pdk1 1.1 chs562, uaz183A 1.2 uaz172B
2.7
bnl17.26 2.9 umc137
2.7
bnl5.47 npi63B npi608 7.2 uaz209
3.5
bnl8.06B
2.2
bnl17.22 1.4 isu1410l
4.6
umc38
3.8
bnl8.08C
2.8
umc160A 1.2 uaz256
11.5
npi280 bnl8.08J koln1B
3.4
uaz19D
4.1
uaz43E 7.3 uaz251D
0.7
uaz243B
4.5
bnl17.12 2.6 ufg(vp2274B) 2.7 umc62 4.4 uaz81 4.5 npi419
4.9
umc132
6.5
idh2 npi597A 3.0 uaz269D
2.4
mdh2 umc133 1.4 npi561 1.9 uaz229 2.9 uaz123C 4.5 ufg(agp1) 4.6 umc28
3.2
php20599 15.8 uaz240B

Chromosome 7

Chromosome 8

cuny19 ***pbs6C*** 2.6 *pbs13D* 3.8 *npi114A*

9.4

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* 3.8 *isu1410A*

12.9

umc103A

4.1

npi585 *npi276B* 1.4 *uaz93* 1.4 *wusl1042* 2.7 *bnl21*

2.5

ucsd64B ***ucsd78B*** *mpik12B,15D,E,16C,17C,24,35F* *uwo11* 4.0 *bcd98B* 8.4 *mpik17D*

2.4

bnl10.39 ***niu1::Bs1*** *bnl9.44,ici286A,npi260B,uaz249C,269A,ucsd61F,umc32B,120;*1.6*uaz290(SDAg);*1.8*ici277*

1.4

mdh1 *bnl13.05C* 1.1 *bnl17.20* 1.2 *isu1719C, 2191B* 2.4 *uaz243C,251A,252B*

1.5

npi618 ***uaz25A*** *tug1* *ncr(sod3C)* *pbs4* 1.2 *bnl1.45A, uaz244B* 1.4 *pge21* 2.4 *pge2* 3.1 *isu2191H*

2.5

bnl8.06A 1.4 *uaz121B* 2.5 *bnl9.08*

1.6

bnl17.16(bt2)

1.7

act1 ***bnl7.08A*** 1.0 *bnl24A* 1.3 *ucsd106H* 1.4 *uaz202* 1.9 *npi294F* 3.8 *csu66*

3.2

isc(b32A) ***npi224C*** *npi(pdk2);*1.1 *koln2C;*2.2 *uaz188;*2.4 *uaz127(ppdk),172A,183B;*2.5 *uaz147*

1.6

ncr(b32c3A) 3.5 *npi224H* ;3.6 *uaz165,193;*3.7 *uaz196*

2.4

wusl(pdc1)

4.1

umc160B 2.9 *mwg645K*

1.1

bnl2.369 *bnl8.26* 1.0 *koln1A* 2.2 *ici222*

3.0

ucb(anp1) 1.5 *cdo708*

2.2

pge11(knox11) *bnl162* *cdo580A* *dgg9A* *uaz233A* 1.2 *npi101B* 1.2 *umc12A* 1.2 *umc89*

2.2

uiu(pog1C) *pgeA4(knox5)* 1.2 *mwg645A* 2.9 *npi595* 3.0 *dgg9H*

5.6

bnl12.30A 4.0 *pic6B*

4.5

bnl17.01 1.6 *csu31* 1.6 *umc93* 1.7 *uaz138B* 5.0 *uaz164B*

3.0

idh1

6.3

npi299 ***umc48*** ***umc53B*** 1.0 *ici95;*1.2 *isu1774B, umc30;*1.3 *npi201B;*1.5 *uaz176A;*2.5 *pbs6B;*4.1 *uaz119A;*4.6 *tum1*

8.2

bnl17.17 1.5 *pbs9A*

3.4

uaz94 4.5 *umc71* 4.7 *umc117*

8.2

uaz95 1.4 *sps1* 3.0 *uaz291(F-bA)*

8.2

npi108B

4.4

bnl10.24B

1.3

npi268

2.7

bnl10.11 1.2 *umc164A* 4.9 *uaz174* 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*

5.4

npi107 ***umc3A***

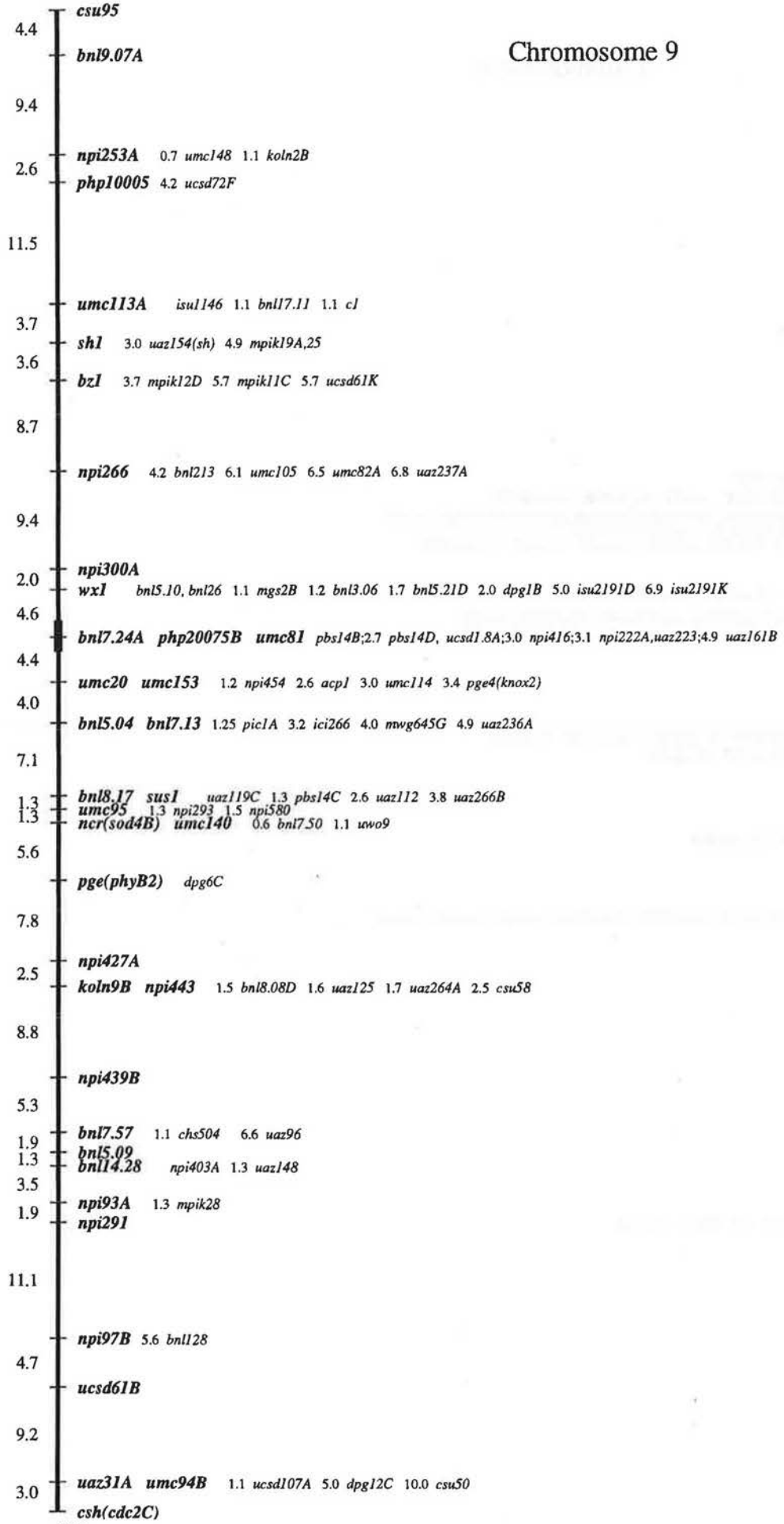
2.5

umc4B 1.3 *bcd98E*

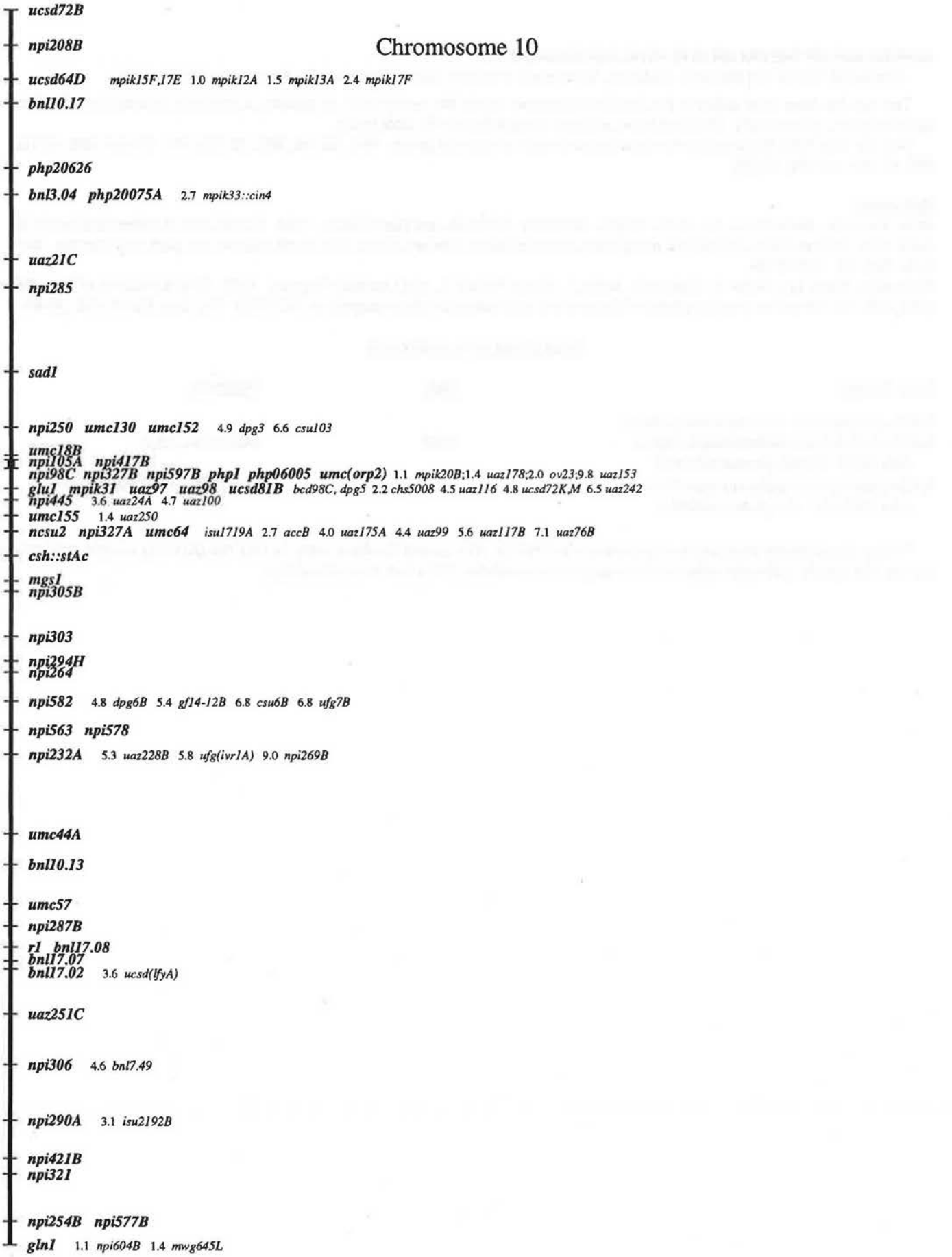
1.5

umc39B 18.8 *npi212A* 20.9 *bnl(tas1M)*

Chromosome 9



Chromosome 10



GENETIC MAP OF THE *ZEA MAYS* PLASTID CHROMOSOME

--Carolyn M. Wetzel and Steven R. Rodermel, Department of Botany, Iowa State University, Ames, Iowa, 50011-1020

Two new loci have been added to the plastid chromosome map in this year's report. In addition, a previously characterized ORF has been assigned a gene identity. Details of these additions are described in the table below.

See the 1987-1994 News Letters for descriptions of other sequenced genes: MNL 62:148; MNL 63:155; MNL 64:164; MNL 66:160; MNL 67:167; and MNL 68:209.

References:

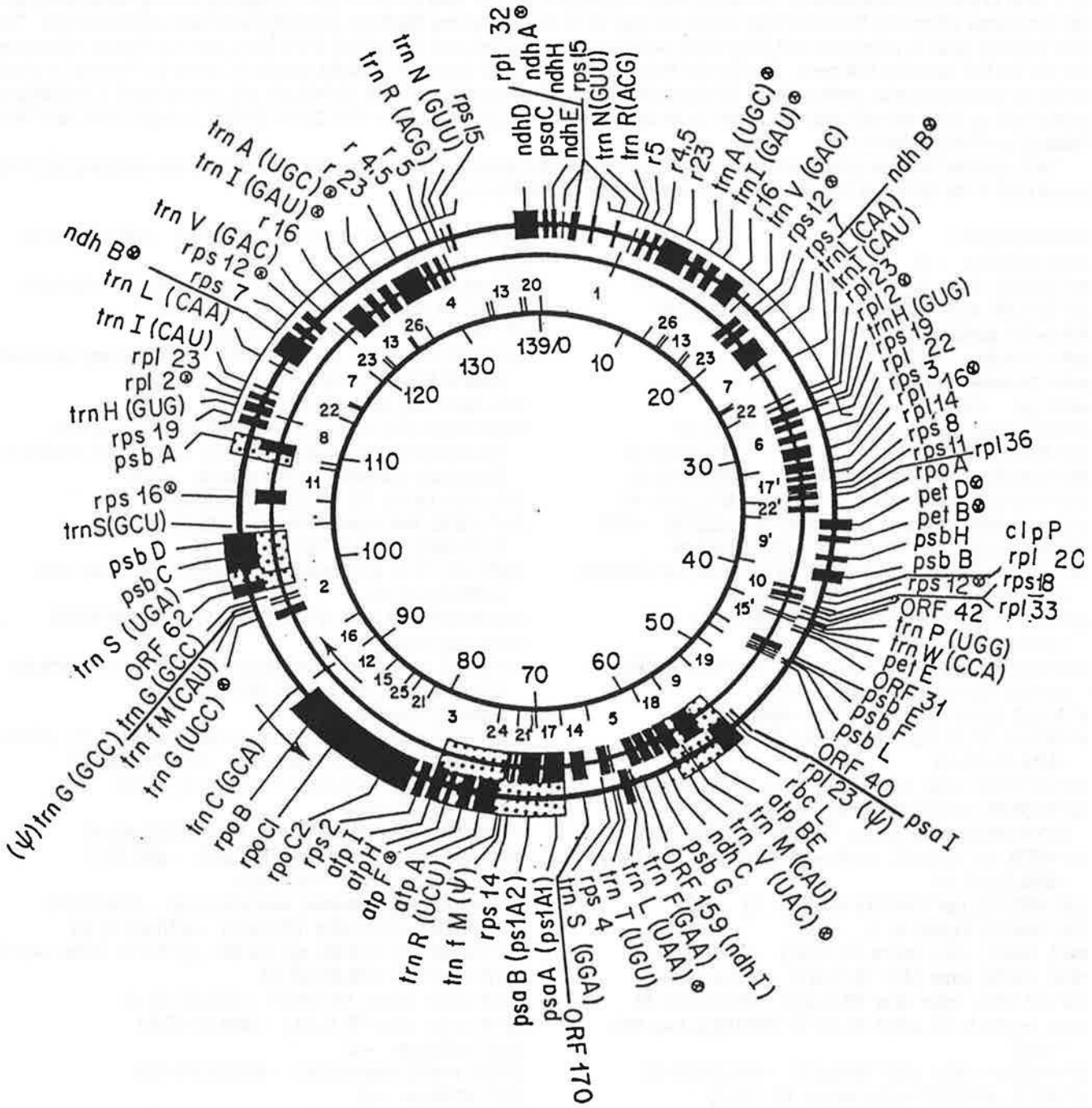
Maier, Rainer M., Neckermann, Kai, Hoch, Brigitte, Akhmedov, Norvus B., and Hans Kössel. 1992. Identification of editing positions in the *ndhB* transcript from maize chloroplasts reveals sequence similarities between editing sites of chloroplasts and plant mitochondria. Nucl. Acids Res. 20: 6189-6194.

Steinmüller, Klaus, Ley, Arthur C., Steinmetz, Andre A., Sayre, Richard T., and Lawrence Bogorad. 1989. Characterization of the *ndhC-psbG-ORF157/159* operon of maize plastid DNA and of the cyanobacterium *Synechocystis* sp. PCC6803. Mol. Gen. Genet. 216: 60-69.

Table of New Gene Assignments

Gene Product	Gene	Reference
NADH-plastoquinone oxidoreductase proteins:		
NADH-plastoquinone oxidoreductase chain 2 (aka NADH-dehydrogenase subunit 2)	*ndhB*	Maier et al., 1992
NADH-plastoquinone oxidoreductase chain I (aka NADH-dehydrogenase subunit I)	*ndhI*	Steinmüller et al., 1989[a]

[a] locus was originally described in this reference as ORF159. The current GenBank entry for ORF159 (X17438) assigns *ndhI* as its identity. No specific publication related to this assignment is available, to the best of our knowledge.


rpl 32
ndh A ⊛
ndh H
rps 15
ndh D
psa C
ndh E
trn N (GUU)
trn R (ACG)
r5
r4.5
r23
trn A (UGC) ⊛
trn I (GAU) ⊛
r16
trn V (GAC)
rps 12 ⊛
rps 7
ndh B ⊛
trn L (CAA)
trn I (CAU)
rpl 23
rpl 2 ⊛
trn H (GUG)
rps 19
rpl 22
rps 3
rpl 16 ⊛
rpl 14
rps 8
rps 11
rpl 36
rpo A
pet D ⊛
pet B ⊛
psb H
clp P
psb B
rpl 20
rps 12 ⊛
rps 18
ORF 42
rpl 33
trn P (UGG)
trn W (CCA)
pet E
ORF 31
psb N
psb T
psb L
ORF 40
rpl 23 (ψ)
psa I
rbc L
atp BE
trn M (CAU)
trn V (UAC) ⊛
ndh C
psb G
ORF 159 (ndh I)
trn F (GAA) ⊛
trn L (UAA) ⊛
trn T (UGU)
rps 4
trn S (GGA)
ORF 170
psa A (ps1A1)
psa B (ps1A2)
trn f M (ψ)
rps 14
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
trn S (GCU)
rps 16 ⊛
psb A
trn H (GUG)
rps 19
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
r4.5
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

XII. ZEALAND 1995

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 ('first report'); mapping; cloning; sequencing; and trait inheritance information that have been 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 in the back of this issue 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: orthology --r3

ad-N605B:* order before TB-1Sb(1) --MNL69:43-46

ad-N613B:* order after TB-1La(1) --MNL69:43-46

Adh1+Cm: evolution --r74

adh1: evolution --r339 r711

adh1: promoter --r239 r371 r433

adh1: yac --r635

bl-N43:* order after TB-1La(1) --MNL69:43-46

blh-N487C:* linkage with T1-9a(1) *wx1* --MNL69:43-46

blh-N495B:* order before TB-1Sb(1) --MNL69:43-46

bnk-N1519C:* order before TB-1Sb(1) --MNL69:43-46

chi1: map note, sequence, first report; Chi1+Z22760 --r274

cl-N801:* order after TB-1La(1) --MNL69:43-46

cp-N628, cp*-N991, cp*-N1078B, cp*-N1393A, cp*-N1399A:* order before TB-1Sb(1) --MNL69:43-46

cp-N918A, cp*-N1311C:* order after TB-1La(1) --MNL69:43-46

csu3, csu12b, csu20(lhcb), csu61, csu92, csu110b, csu134a, csu145c, csu164, map location --r102

d-N1883* shows linkage with bz2 --MNL69:43-46

d-N454A, d*-N1352B:* order after TB-1La(1) --MNL69:43-46

dcr-N1176B:* order before TB-1Sb(1) --MNL69:43-46

de-N1057B, de*-N1142, de*-N1162, de*-N1345B, de*-N1390A:* order before TB-1Sb(1) --MNL69:43-46

de-N978, de*-N1310B, de*-N1420:* order after TB-1La(1) --MNL69:43-46

dek-MS2115, dek*-MS8319* located to 1S --r597

dek-MS6214* located to 1L --r597

dek1, dek32: order before TB-1Sb(1) --MNL69:43-46

dek2, dek22: order after TB-1La(1) --MNL69:43-46

dnt-N1185A:* order after TB-1La(1) --MNL69:43-46

emp1 located to 1S; *emp1* -31.57- T1-9(8918)(1), map data --r597

et-N1001A:* order after TB-1La(1) --MNL69:43-46

et-N617, et*-N745:* order before TB-1Sb(1) --MNL69:43-46

fl-N1208A, fl*-N1308A:* order before TB-1Sb(1) --MNL69:43-46

gm-N1303:* order after TB-1La(1) --MNL69:43-46

gm-N1390C:* order before TB-1Sb(1) --MNL69:43-46

gsr1: umc157 -3.8- *gsr1* -0.9- *umc115* -2.8- *umc194c(gpr)* --r102

hcf2, hcf12, hcf13, hcf41: order after TB-1La(1) --MNL69:43-46

hcf3, hcf6, hcf31: order before TB-1Sb(1) --MNL69:43-46

hcf4, hcf44, hcf50: order after TB-1Lc(1) --MNL69:43-46

ibp2: sequence --r408

id1: sequence, structure; *id1* -1- *bz2*, map data; *id1-Compeigne*; *id1-CSH*, clone isolation --MNL69:35

ij2: order after TB-1La(1) --MNL69:43-46

Inv1h(left) breakpoint at 0.089 differs from previously reported value of 0.071 --r415

isu6, isu18, map location --r691

kernel weight QTL: flanking markers *bnl5.59* and *umc23a* in Reventador maize/parviglumis teosinte; *umc157* and *umc37b* in Reventador maize/parviglumis teosinte --r169

l16: order before TB-1Sb(1) --MNL69:43-46

les-J2552*, first report: distal to T1-9c(1) but not T1-9(4995); linked to *lls1* --r321

les20 -23- T1-9c *wx1*; *les20* -7- T1-9(4995) *wx1*, map data --MNL69:43-46

maysin QTL near *p1* in GT114/GT119 F2 --MNL69:53-54

mdh4, map location --r102

ms-6034*, first report: order before TB-1Sb(1); complements *as1, ms9, ms12, ms14, ms17*; allele *ms*-6044* --MNL69:126-128

ms9: order before TB-1Sb(1) and after T1-2(4464)(1); alleles *ms9-6032, ms9-6037, ms9-6042* --MNL69:126-128

msv1, first report: *bnl12.06a* -10- *msv1* -3- *npi262* --MNL69:136-137

o-N1009:* order before TB-1Sb(1) --MNL69:43-46

olc1, first report: order after TB-1La(1) --MNL69:3

P1-wr, restriction map --MNL69:9

P1-ww-12:27-3*: sequence, restriction map --MNL69:8-9

pg-N1822A:* order after TB-1La(1) --MNL69:43-46

pg-N484A, pg*-N484B, pg*-N526C, pg*-N619:* order before TB-1Sb(1) --MNL69:43-46

pg15: order before TB-1Sb(1) --MNL69:43-46

pg16: order after TB-1La(1) --MNL69:43-46

pgm1: orthology --r3

phi001, phi002, map location --MNL69:119-120

phi1: orthology --r3

photosynthesis QTL linked to *bnl5.62a* --r87

plant height QTL near *umc37a* & *an2.6* --MNL69:7-8

ptd-N923:* order before TB-1Sb(1) --MNL69:43-46

ptd1, bz2 -10- *ptd1, ptd1* -17- *kn1, ptd1* -25- *bm2*, map data; *ptd1-Mu1568* --r597

py2: order after TB-1La(1) --MNL69:43-46

rth1: order after TB-1La(1) --r715

rth3: order before T1-2(4464)(1) and T1-2c(2) --r715

smk-N1057A:* order before TB-1Sb(1) --MNL69:43-46

smp-N706A:* order after TB-1La(1) --MNL69:43-46

spc2: order after TB-1La(1) --MNL69:43-46
Stewart's wilt QTL near *umc167* in Hi31/Ki14 RIs --MNL69:60-61
tb1: evolution,; *umc140* -1.7- *bcd1072c* -1.7- *tb1* -1.3- *umc107* -0- *bnl15.18* --MNL69:120
tbp1: map location --r695
ts2: orthology --r56
tua1: promoter --r566
v-N55, v*-N245, v*-N1806:* order after TB-1La(1) --MNL69:43-46
vp-N1136B:* order before TB-1Sb(1) --MNL69:43-46
w-N547A, w*-N1890:* order after TB-1La(1) --MNL69:43-46
w18: order after TB-1La(1) --MNL69:43-46
wl-N1831:* order after TB-1Lc(1) --MNL69:43-46
wl-N47, wl*-N56, wl*-N60, wl*-N709B:* order after TB-1La(1) --MNL69:43-46
wlu5: order after TB-1La(1) --MNL69:43-46
wt-N650A:* order before TB-1Sb(1) --MNL69:43-46
zb7: order after TB-1La(1) --MNL69:43-46

CHROMOSOME 2
accA: npi242C umc131 accA -1.5- *uox(ssu1b) npi242a npi297 npi356* --MNL69:3-4
agp2: first report, map location, clone isolation --r253
Amy-L25805+Oh43*: sequence --r743
amy3: first report --r743
b1: evolution --r540
ch1: orthology --r3
cp-N1076A:* order before T2-3(6270)(2) --MNL69:43-46
cp-N1225B:* map data --MNL69:43-46
cp-N1319A:* order before TB-2Sa(2) --MNL69:43-46
csh(chi2): map note --r274
csu4, csu29a, csu40(grx), csu64(tau), csu109a, csu148, csu154a: map location --r102
d-N155B:* order before TB-2Sa(2) --MNL69:43-46
d-N208B:* order before T2-3(6270)(2) --MNL69:43-46
d10 -14- T2-9d *wx1*, map data --MNL69:43
d5: orthology --r3
de-N660C:* order aft er TB-1Sb-2L4464(2); cp*-N1225B allelic --MNL69:43-46
dcr-N1233A:* order before TB-2Sa(2) --MNL69:43-46
de-N1122A:* order before T2-3(6270)(2) --MNL69:43-46
de-N1175:* order after T1-2(4464)(2) --MNL69:43-46
dek-MS1365* located to 2L --r597
dek-MS2159* -20- T2-9d(2), map data --r597
dek-MS2444, dek*-PIC, dek*-PIE* located to 2L --r597
dek3: order before TB-3La-2S6270(2) --MNL69:43-46
dek4: ,*dek16, dek23:* order after TB-1Sb-2L4464(2)--MNL69:43-46
emp2 -19- *w3; emp2* -21- T2-9d(2); *fl1* -46- *v4* -8- *emp2* -47- *ch1*, map data; allele *emp2-Mu1047* --r597
et-N1078A:* order after T1-2(4464)(2) --MNL69:43-46
fl-N1287:* order after T1-2(4464)(2) --MNL69:43-46
fl-N1426:* order before TB-3La-2S6270(2) --MNL69:43-46
fl1: orthology --r3
gm-N1312:* order before TB-2Sa(2) --MNL69:43-46
hcf106-mum1::Mu1, hcf106-mum2::Mu1, hcf106-mum3::Mu1, hcf106-mum4, structure --MNL69:34-35
hcf1: order after T1-2(4464)(2) --MNL69:43-46
hcf15: order after T1-2(4464)(2) --MNL69:43-46
isu7: map location --r691
kernel weight QTL: flanking markers *umc34* and *umc131* in Chapalote maize/mexicana teosinte; *umc49a* and *umc36a* in Reventador maize/parviglumis teosinte --r169
l18: order after T1-2(4464)(2) --MNL69:43-46
Lg-9167* -23- T2-9d *wx1*, map data --MNL69:24-25
lg1: orthology --r3
Mha1+D3L, Mha1+W22: sequence --r76
mha1 approximately at same location as *umc36a* and *npi294a* --r319
mha1: sequence --r74 r76 r319 r319 r486
mn-N1120A:* order after T1-2(4464)(2) --MNL69:43-46
ms-6019* -15- *ch1*; between T2-5f(2) and T2-8(8376)(2), per duplicate-deficient plants; order after T1-2(4464)(2); alleles *ms*-6024, ms*-6029, ms*-6038, ms*-6041* --MNL69:126-128
nc003: map location --MNL69:119-120
nec-N1119B:* order before TB-2Sa(2) --MNL69:43-46
nec4: order before TB-3La-2S6270(2) --MNL69:43-46
o-N1189A:* order before TB-3La-2S6270(2) --MNL69:43-46
o-N1195A:* order after T1-2(4464)(2) --MNL69:43-46
os1: genelist --r632
prp2: umc34 - prp2 - php10012 --r102
ptc-N2284B* after TB-2Sa, linked to TB-2Sa in hypoploid --MNL69:43-46
ptd-N901A:* order before TB-2Sa(2) --MNL69:43-46
px1: orthology --r3
spt-N579B:* order after T1-2(4464)(2) --MNL69:43-46
spt1: order after TB-1Sb-2L4464(2) --MNL69:43-46
trAc8178 -5- T2-9b(2); *trAc8178* -15- T2-9c(2); *trAc8178* -38- T2-9d(2) --MNL69:123-124
v24: order after TB-1Sb-2L4464(2) --MNL69:43-46
v26: order before TB-3La-2S6270(2) --MNL69:43-46
w-N77, w*-N332, w*-N346*: order after T1-2(4464)(2) --MNL69:43-46
wt-N136A:* order before TB-2Sa(2) --MNL69:43-46
wt1: order before TB-3La-2S6270(2) --MNL69:43-46

CHROMOSOME 3
a1-sh2 spacer, restriction map --r119
a1: promoter --r273
a3 -8.7- *a1; a3* -6.5- *umc96*, map data --MNL69:46
atp1: map location --r102
brn1 -14.1- *g2; brn1* -36- *cl1; brn1* -21.5- *d1; brn1* -6.7- *cr1; brn1* -26.7- ra2; *brn1* order before TB-3Sb; *brn1* -19- *d1* -21- *lg3*, map data --r597
cp-N1379A, cp*-N1436A:* order after TB-3La(3) --MNL69:43-46
crp-N2207:* order after TB-3La(3) --MNL69:43-46
csu25a(P450), csu29b, csu32, csu38a, csu56b(ohp), csu58, csu96, csu154b: map location --r102
d-N282:* order after TB-3La(3) --MNL69:43-46
dcr-N1053A:* order before TB-3Sb(3) --MNL69:43-46
de-N932, de*-N1126A, de*-N1166:* order after TB-3La(3) --MNL69:43-46
dek17, dek24: order before TB-3Sb(3) --MNL69:43-46
dek5: order before TB-3Sb(3), *brn1* -22- *dek5* -23- *lg3*, map data; allele *dek5-MS33* --r597
dek6: order after TB-3La(3) --MNL69:43-46

e3, e4, e8: orthology --r3
et-N1322C:* order after TB-3La(3) --MNL69:43-46
gl-N352A, gl*-N672B:* order after TB-3La(3) --MNL69:43-46
gl19: order before TB-3Sb(3) --MNL69:43-46
gl6: umc92a -7- (*umc10a,umc102)* -1- *gl6* -1- *bnl6.06* -11- *bnl5.37a*, map data --r729
gm-N1311B:* order after TB-3La(3) --MNL69:43-46
got1: orthology --r3
hcf19: data conflict re placement with TB-3Sb vs. TB-3La --r443
hcf46: order after TB-3La(3) --MNL69:43-46
hex1: orthology --r3
hsp18f: map note --MNL69:96
ILS-1: sequence, first report --r10
isu1: map location --r691
lg2-2757::Mu8, lg2-MF219, lg2-MF228, lg2-MF229.1, lg2-MF229.2, lg2-MF278, lg2-MF902: clone isolation, restriction map --MNL69:23-24
lg3 -22- T3-9(8447) *wx1*; lg3 -14- T3-9(8562) *wx1*, map data --MNL69:22-23
lg3-Mlg: origin --MNL69:22-23
maysin QTL near *a1* in GT114/GT119 F2 --MNL69:53-54
mdh3: orthology --r3
me1: orthology --r3
me3: map location --r102 MNL69:126-128
ms3: order after TB-3La(3), before T2-3(6270)(3); alleles *ms3-6008, ms3-6009, ms3-6020, ms3-6043* --MNL69:126-128
mv1 linked to php20508 in Hi31/Ki14 RIs, map data --MNL69:60
nc030: map location --MNL69:119-120
nec-N720C:* order after TB-3La(3) --MNL69:43-46
ns1: after before TB-3Sb--MNL69:23
rea1: first report, order before T2-3(6270)(3) --r242
ref1 -30- *cl1*; T3-9c(3) -30- *ref1*, map data; allele *ref1-Mu1185* --r597
rf1 -1- *umc97* -5- *umc102* -1- (*rg1,bnl6.06a)*, map data; *umc50* -21- *rf1* -6- *umc97* -1- *umc102* -5- *bnl6.06a* -12- *bnl5.37a*, map data; *umc50* -11- *rf1* -1- (*umc97,umc10,umc102)* -1- *bnl6.06* -8.3- *bnl5.37a* -9- *bnl5.37b* (duplication of *bnl5.37a*, specific to R213 inbred), map data --r729
rgh-N802, rgh*-N1060:* order after TB-3La(3) --MNL69:43-46
rgh-N1112:* order before TB-3Sb(3) --MNL69:43-46
Sh2+ILP76::ILS-1: sequence --r10
sh2-M15: first report --MNL69:56-57
Sh2-Rev25, Sh2-Rev31: origin --MNL69:56-57
si-N1323:* order before TB-3Sb(3) --MNL69:43-46
smk-N1168A:* order after TB-3La(3) --MNL69:43-46
smp-N1324B:* order after TB-3La(3) --MNL69:43-46
spc3: order after TB-3La(3) --MNL69:43-46
su-N748A:* order after TB-3La(3) --MNL69:43-46
tpi4: orthology --r3
trAc8163 -30- T3-9c(3); *trAc8163* -12- T3-9(8447)(3), map data --MNL69:123-124 r173
trAc8183 -3- T3-9c(3); *trAc8183* -6- T3-9(8447)(3), map note --MNL69:123-124
*Tub*L10633+B73*: sequence --r693
v-N1886:* order after TB-3La(3) --MNL69:43-46
w19: orthology --r3
wl-N4*: order after TB-3La(3) --MNL69:43-46
wlu1: order after TB-3La(3) --MNL69:43-46
wsm2: bnl.8.35 -4.3- *umc93* -7.0- (*umc10a,umc97,umc102)* -1.7- *wsm2* -1.7- (*bnl6.06,umc18)* -16.8- *bnl5.37* --r435
zag2: sequence, phylogenetic analysis --MNL69:37

CHROMOSOME 4
adh2: orthology --r3
adh2: promoter --r493
bt2: map location --r691
Cat3+W64A: TouristA element inserted 5',,sequence --r74
cb-N719A:* order before TB-4Sa(4) --MNL69:43-46
cl-N795:* order after TB-4Lf(4) --MNL69:43-46
cp-N1313:* order before TB-4Sa(4) --MNL69:43-46
cp2(=dek7): order before TB-4Sa(4); *cp2* -7.1- *fl2; cp2* -21- *su1* -10- *gl3*, map data; allele *cp2-MS2608* --r597 MNL69:43-46
csu26b(ant), csu36a, csu84, csu91a, csu100: map location --r102
de-N929:* order before TB-4Sa(4) --MNL69:43-46
dek10: order after TB-4Lf(4) --MNL69:43-46
dek11: order before TB-4Sa(4) --MNL69:43-46
dek25: order before TB-4Sa(4); *dek25* -25.8- *fl2, dek25* -44.7- *su1*, map data; allele *dek25-MS2410* --r597 MNL69:43-46
dek31: order after TB-4Lf(4); *dek31* -18.4- *c2, dek31* -19.9- *gl3, su1* -21- *gl4* -22- *dek31*, map data; allele *dek31-MS2689* --r597 MNL69:43-46
dek8: order after TB-4Lf(4) --MNL69:43-46
dsc1: su1 -3- *dsc1* -1- *bm3* OR *su1* -3- *bm3* -1- *dsc1*, map data; allele *dsc1-Mu2058* --r597
dzr1: genelist; order after TB-4Sa; *dzr1* -16- *adh2*, map data --r105
fl2: clone isolation --MNL69:124-125
gpc1: origin --r340
hcf23: order before TB-4Sa(4) --MNL69:43-46
kernel weight QTL: flanking markers *bnl5.46* and *umc42a* in Reventador maize/parviglumis teosinte; *umc42a* and *umc66a* in Chapalote maize/mexicana teosinte --r169
Mgs2+Zm58.1: sequence --r677
mgs2: genelist; *umc52 - mgs2 - npi116a* (aka *npi203)* map note --r677
nc004, nc005: map location --MNL69:119-120
nec-N193, nec*-N1487:* order after TB-4Lf(4) --MNL69:43-46
nec-N562, nec*-N673B:* order before TB-4Sa(4) --MNL69:43-46
nec5: order after TB-4Lf(4) --MNL69:43-46
ns2: order after TB-4Lf(4) --MNL69:23
o-N1119A, o*-N1228, o*-N1244A:* order before TB-4Sa(4) --MNL69:43-46
pg-N1881, pg*-N673A:* order before TB-4Sa(4) --MNL69:43-46
phi006: map location --MNL69:119-120
Prh1+RDS91: TouristD inserted 5', position,sequence --r74
rgh-N1105A:* order after TB-4Lf(4) --MNL69:43-46
sh-N1105B, sh*-N1324A, sh*-N1519B:* order before TB-4Sa(4) --MNL69:43-46
smp-N156A:* order before TB-4Sa(4) --MNL69:43-46
spt2: order before TB-4Sa(4) --MNL69:43-46

Stewart's wilt QTL near *umc19* in Hi31/Ki14 RIs, map data --MNL69:60-61
su3 -6- T4-9g(4), map data; allele su3-5081 --r597
tga1: evolution --r104 r438
trAc8200 -4- *su1*; *trAc8200* -3- T4-9g(4); *trAc8200* -4- T4-9(5657)(4), map data --r173 MNL69:123-124
v-N378A:* order after TB-4Lf(4) --MNL69:43-46
wl-N311B:* order after TB-4Lf(4) --MNL69:43-46
wst-N413A:* order before TB-4Sa(4) --MNL69:43-46
wt2: order before TB-4Sa(4) --MNL69:43-46
zbr1: map location --r102

CHROMOSOME 5
ad-N664:* order before TB-5Sc(5) --MNL69:43-46
Ae1-5180::Mu1: restriction map --r643
am1-485, am1-489: first report --MNL69:58
anl1: order before TB-5Sc(5) --MNL69:43-46
bt1-N2310: map note --MNL69:43-46
bt1: map location --r691
cl-N818A:* order before TB-5Sc(5) --MNL69:43-46
cp-N863A, cp*-N935, cp*-N1275A, cp*-N1369, cp*-N1385:* order after TB-5La(5) --MNL69:43-46
cp-N1430:* order before TB-5Sc(5) --MNL69:43-46
crp2: order before TB-5Sc(5) --MNL69:55
csh(chi3): map note --r274
csu26a(ant), csu33a, csu36b, csu108(gtpb), csu134b, csu134c, csu137, csu173(gfu): map location --r102
d*-6: map note --r242
dcr-N925A:* order before T1-5(8041)(5) --MNL69:43-46
de-N1002A:* order before TB-5Sc(5) --MNL69:43-46
de-N1196:* order after TB-5La(5) --MNL69:43-46
dek-MS1182, dek*-MS2146, dek*-NS8070, dek*-PIO:* located to 5L --r597
dek18: order before TB-5Sc(5) --MNL69:43-46
dek9, dek26, dek27, dek33: order after TB-5La(5) --MNL69:43-46
dnj-N1534:* order after TB-5La(5) --MNL69:43-46
fl-N1145A:* order after TB-5La(5) --MNL69:43-46
fl-N1333B:* order before TB-5Sc(5) --MNL69:43-46
gl-N681A:* order before TB-5Sc(5) --MNL69:43-46
gl8: order after TB-5La(5) --MNL69:43-46
gpc4: bnl5.71 - gpc4 - umc126 --r583
gpc4: origin --r340
grt1: order after TB-5La(5) --MNL69:43-46
hcf108: order before TB-5Sc(5) --r443
hcf18, hcf21, hcf38, hcf43: order after TB-5La(5) --MNL69:43-46
kernel weight QTL: flanking markers *bnl15.40* and *umc110a* in Chapalote maize/mexicana teosinte
l-N1838:* order before TB-5Sc(5) --MNL69:43-46
lw2-N1868: map note --MNL69:43-46
mn-N1536:* order after TB-5La(5) --MNL69:43-46
nc007: map location --MNL69:119-120
nec3: order before T1-5(8041)(5) --MNL69:43-46
nec7: order after TB-5La(5) --MNL69:43-46
nrz5: map location --MNL69:119-120
o-N1065A:* order after TB-5La(5) --MNL69:43-46
pg-N408C:* order after TB-5La(5) --MNL69:43-46
phi008: map location --MNL69:119-120
photosynthesis QTL linked to *bnl5.40* --r87
ppg1: order after TB-5La(5) --MNL69:43-46
pr-N850:* order after TB-5La(5) --MNL69:43-46
prg1 -13- *ae1; prg1* -21- T5-9a(5), map data; allele *prg1-Mu8186* --r597
psb2: order after TB-5La(5) --MNL69:55
ren1: pr1 -24- *ren1* -31- *v2,; ae1* -10- *pr1* -9- *gl8* -*19* -ren1, map data; allele *ren1-Mu807* --r597 r691
rth2: genelist; order after TB-5La(5) --r715
sca-csu149:* map location --r102
smk-N1160:* order after TB-5La(5) --MNL69:43-46
smk-N1529:* order before TB-5Sc(5) --MNL69:43-46
sms-N146C:* order after TB-5La(5) --MNL69:43-46
tbp2: map note, sequence --r695
trAc6076 -12- *pr1*; *trAc6076* -4- T5-9c(5); *trAc6076* -31- T5-9a(5) --MNL69:123-124 r173
trAc8175 -35- *pr1*; *trAc8175* -10- T5-9c(5); *trAc8175* -50- T5-9a(5) --MNL69:123-124 r173
trAc8179 -14- *pr1*; *trAc8179* -7- T5-9a(5); *trAc8179* -21- T5-9c(5) --MNL69:123-124 r173
trAc8181 -41- *pr1*; *trAc8181* -10- T5-9a(5); *trAc8181* -50- T5-9c(5) --MNL69:123-124 r173
trAc8186 -34- *pr1*; *trAc8186* -9- T5-9a(5); *trAc8186* -33- T5-9c(5) --MNL69:123-124 r173
trAc8193 -50- *pr1*; *trAc8193* -4- T5-9c(5); *trAc8193* -50- T5-9a(5) --MNL69:123-124 r173
trAc8196 -35- *pr1*; *trAc8196* -7- T5-9a(5); *trAc8196* -50- T5-9c(5) --MNL69:123-124 r173
v-N26, v*-N735*: order after TB-5La(5) --MNL69:43-46
w-N21A, w*-N22, w*-N1126B*: order after TB-5La(5) --MNL69:43-46
wgs1: order after TB-5La(5) --MNL69:43-46
wi4 -25- T5-9c *wx1*, map data --MNL69:43
wl-N44:* order before TB-5Sc(5) --MNL69:43-46
wusl1005(gfu): genelist --r507
zn-N571D:* order after TB-5La(5) --MNL69:43-46

CHROMOSOME 6
agp1: map location --r691
agp1: sequence, first report --r534
cdc48: map location --r102
csu16b, csu56a(ohp), csu60, csu68a, csu70(gfu), csu94a, csu116a(elf1), csu155a(pdk): map location --r102
d-9*: map note --r242
de-N1400:* order after TB-6Lc(6) --MNL69:43-46
dek-MS1104: y1* -40- *su2* -44- *dek*-MS1104*, map data --r597
dek19: order after TB-6Lc(6) --MNL69:43-46
dek28: order before TB-6Sa(6) --MNL69:43-46
enp1: orthology --r3
gpc2 -2- *wx1* --r583
gs3: order after TB-6Lc(6) --MNL69:43-46
hcf34, hcf36, hcf48: order after TB-6Lc(6) --MNL69:43-46
hcf5, hcf26: order before TB-6Sa(6) --MNL69:43-46
IGS: sequence, evolution --r656
l-N62, l*-N113, l*-N612B:* order after TB-6Lc(6) --MNL69:43-46
mn3 -2- *y1* -8- *l15; mn3* -4- *w15* -3- *y1*, map data; allele *mn3-1184* --r597
nc009, nc010, nc012, nc013: map location --MNL69:119-120
o-N1320A, o*-N1368, o*-N1384A:* order after TB-6Lc(6)

--MNL69:43-46
o14: order after TB-6Lc(6) --MNL69:43-46
pg-N1885:* order after TB-6Lc(6) --MNL69:43-46
phi011: map location --MNL69:119-120
photosynthesis QTL linked to *bnl6.29a* --r87
Pl1+Rhoades, Pl1+Tx303, Pl1-Bh1: sequence --r127 r128
psb1: order before TB-6Lb(6) --MNL69:55
ptd-N1425A:* order after TB-6Lc(6) --MNL69:43-46
sh-N1320B:* order after TB-6Lc(6) --MNL69:43-46
smp-N272A:* order after TB-6Lc(6) --MNL69:43-46
trAc6062 -20- T6-9b(6) --MNL69:123-124 r173
trAc6063 -7- T6-9b(6) --MNL69:123-124 r173
trAc8172 -16- T6-9b(6) --MNL69:123-124 r173
trAc8184 -3- T6-9b(6) --MNL69:123-124 r173
v-N69A, v*-N634A:* order after TB-6Lc(6) --MNL69:43-46
w-N278A:* order after TB-6Lb(6) --MNL69:43-46
w-N335:* order after TB-6Lc(6) --MNL69:43-46
wl-N217A, wl*-N358A, wl*-N362B:* order after TB-6Lc(6) --MNL69:43-46
wsm1: jcl270 -6.1- *npi245* -1.7- *umc85* -2.5- *wsm1* -0.8- *(NOR,npi235)* -0.8- *npi101c* -2.5- *umc59a* -17.8- *umc65* --r435
y1-8549, Y1-B73, Y1-H99, y1-lem, Y1-M14, Y1-Q66/Q67, y1-w-mut: sequence, microsatellite --MNL69:84-85
zag1: phylogenetic analysis --MNL69:37

CHROMOSOME 7
bn1 -27.8- *gl1*; *Bn1-PSS* allele --MNL69:129-130
cp-N1104B, cp*-N1417:* order after TB-7Lb(7) --MNL69:43-46
cp-N1294:* order before TB-7Sc(7) --MNL69:43-46
crp1: map note --r30
csu8, csu11, csu13(h1), csu27(bcl), csu81, csu129(ntm9), csu154c: map location --r102
de-N1136A:* order before TB-7Sc(7) --MNL69:43-46
de-N1177A:* order after TB-7Lb(7) --MNL69:43-46
dek-MS2082* -34.7- T7-9(4363)(7), map data --r597
dek-MS5153* located to 7L --r597
et-N1332:* order after TB-7Lb(7) --MNL69:43-46
gl-N1845:* order after TB-7Lb(7) --MNL69:43-46
hcf101, hcf103, hcf104: order after TB-7Lb(7) --r443
kernel weight QTL: flanking markers *bnl15.40* and *umc110a* in Chapalote maize/mexicana teosinte --r169
m82 -28.2- *php20581a* -36.3- *bnl8.39* -6.5- *bnl15.40* -18.5- *umc116* -22.5- *umc110* -28.5- *bnl14.07* -53.6- *bnl8.44* -8.3- *umc35* --r6
ms-6004:* order after TB-7Lb(7); complements *ms7, va1*; alleles *ms*-6010, ms*-6013, ms*-6014* --MNL69:126-128
o-N1298, o*-N1310A:* order after TB-7Lb(7) --MNL69:43-46
o15: first report --MNL69:124
o2-Crow, o2-R: sequence, evolution --MNL69:18-19
o2-m55::Ac: restriction map --r440
o2-T: derived from *O2-wl*, sequence --MNL69:102
o2: sequence:amplification primers --r440
photosynthesis QTL linked to *bnl7.61* --r87
ptd2: ij1 -47- *ptd2*, map data; *ptd2-Mu3193* allele --r597
ren2: ij1 -33- *ren2; y8* -19- *gl1* -37- *ren2*, map data; allele *ren2-Mu326* --r597
rs1 -25.5- *o2* -16.3- *gl1; rs1* -26.6- *o2; rs1* -5.7- *npi400a* -19.2- *php20581a* -12.7- *o2*; *ucsd106b* -8- *rs1* -14- *npi400a* -16- *php20581a* -2- *o2*; *hs1* unlinked to *rs1* or *o2*; *rs1* and *ers1* are unlinked --r41
rs4: o2 -4.1- *v5* -9.7- *rs4* -3.6- gl1, map data --MNL69:24b
sh-N1341:* order after TB-7Lb(7) --MNL69:43-46
sh6: order before TB-7Sc(7) --MNL69:43-46
smp-N586B:* order after TB-7Lb(7) --MNL69:43-46
spc-N357A:* order after TB-7Lb(7) --MNL69:43-46
thp1: genelist --r102
trAc8161 -50- T7-9a(7); *trAc8161* -13- T7-9(4363)(7) --MNL69:123-124 r173
trAc8173 -29- T7-9a(7); *trAc8173* -1- T7-9(4363)(7) --MNL69:123-124 r173
trAc8185 -3- T7-9a(7); *trAc8185* -50- T7-9(4363)(7) --MNL69:123-124 r173
trAc8190 -50- T7-9a(7); *trAc8190* -5- T7-9(4363)(7) --MNL69:123-124 r173
trAc8194 -50- T7-9a(7); *trAc8194* -3- T7-9(4363)(7) --MNL69:123-124 r173
v27: order after TB-7Lb(7) --MNL69:43-46
vp9-N2213 --MNL69:43-46
wl-N629A:* order after TB-7Lb(7) --MNL69:43-46
wlu2: order after TB-7Lb(7) --MNL69:43-46
zpB36: map location --MNL69:125

CHROMOSOME 8
ald1: umc117 - ald1 - npi268 --r583
crp-N1429A:* order after TB-8Lc(8) --MNL69:43-46
csu29c, csu31, csu110a, csu110c, csu155b(pdk), csu165: map location --r102
dek20, dek29: order after TB-8Lc(8) --MNL69:43-46
emp3: order after TB-8Lc(8) --MNL69:43-46
fl-N1163:* order after TB-8Lc(8) --MNL69:43-46
fl3: orthology --r3
gst1: umc7 -9.3- *gst1* -13.4- *MSb2.1* --MNL69:101
gstIIB: cent8 - *umc89* -5.7- *gstIIB* --MNL69:101
hcf102: order after TB-8Lc(8) --r443
hsp18c: map note --MNL69:96
htn1 -15.4- *v16* -46- *j1; umc48* -11.8- [*umc30a, umc117]* -0.8- *htn1*; *ht2* -10- *htn1* map data; *ht2* not allelic to *htn1* --r620
idh1: orthology --r3
phi014, phi015: map location --MNL69:119-120
photosynthesis QTL linked to *bnl2.369, bnl9.11* --r87
pro1 -36.4- *j1*, map data; allele *pro1-MS5132* --r597
rgh1: order after TB-8Lc(8) --MNL69:43-46
stp1: map location --r102
trAc8162 -14- T8-9d(8); *trAc8162* -50- T8-9(6673)(8) --MNL69:123-124 r173
trAc8182 -6- T8-9d(8); *trAc8182* -1- T8-9(6673)(8) --MNL69:123-124 r173
v-N29, v*-N779A, v*-N826*: order after TB-8Lc(8) --MNL69:43-46
v21: order after TB-8Lc(8) --MNL69:43-46
wlu3: order after TB-8La(8) --MNL69:43-46

CHROMOSOME 9
bz1-m13CS17::dSpm: first report --r542
bz1: orthology --r3
C1+W22(LC), c1, C1-I, c1-m1::Ds, c1-m2::Ds, c1-n, c1-p, C1-S: promoter, sequence --r127 r598

cp-N1092A:* order before TB-9Sb(9) --MNL69:43-46
cp-N1381:* order after TB-9Lc(9) --MNL69:43-46
csu12a, csu43(gfu), csu54b, csu59, csu93a, csu94b, csu95a, csu145a, csu145b, csu147: map location -- r102
d3, d3-2(Mu): clone isolation --MNL69:125-126
dcr-N1409:* order after TB-9Lc(9) --MNL69:43-46
dek12: order before TB-9Sb(9) --MNL69:43-46
dek13, dek30: order after TB-9Lc(9) --MNL69:43-46
dsc-N749:* order after TB-9Lc(9) --MNL69:43-46
dzs10: sequence --r705
eno1: umc105 - csu94b - eno1 - wx1 --r102 r507
et-N357C:* order after TB-9Lc(9) --MNL69:43-46
gm-N1319B:* order after TB-9Lc(9) --MNL69:43-46
hcf42: order after TB-9Lc(9) --MNL69:43-46
hsp18a: map note --MNL69:96
ibp1: first report --r408
kernel weight QTL: flanking markers *umc95* and *bnl14.28a* in Reventador maize/parviglumis teosinte --r169
maysin QTL near *bz1, c1* in GT114/GT119 F2 --MNL69:53-54
Mgs3+Zm58.2: sequence --r677
mgs3: first report, *mgs3* -1- *wx1* --r677
ms-6006:* order after TB-9La(9); complements *ms*-6011, ms*-6021* --MNL69:126-128
ms-6011:* order after TB-9Lc(9); complements *ms2, ms*-6021, ms*-6006;* alleles *ms*-6018, ms*-6027, ms*-6031* --MNL69:126-128
ms-6021:* order after TB-9La(9); complements *ms*-6011, ms*-6006;* alleles *ms*-6022, ms*-6046, ms*-6047* --MNL69:126-128
pg-N660A:* order after TB-9Lc(9) --MNL69:43-46
phi016, phi017: map location --MNL69:119-120
rf2-8122: clone isolation --r604
rf2-m8110: first report --r604
rf2: wx1 -5- *umc153* -10- *rf2* -8- *sus1* -1- *umc95; wx1* -2- (rf2,*bnl5.10,umc153)* -5- *sus1* -2- *umc95,* map data --r729
sh-N399A:* order before TB-9Sb(9) --MNL69:43-46
Sh1+Black Mexican: *TouristA* inserted into intron,sequence --r74
sh1: promoter --r670
sus1: sequence --r307
trAc8156 -3.7- *wx1*, map data --r173
trAc8157 -25- *wx1*, map data --r173
trAc8166 -13- *wx1*, map data --r173
trAc8167 -10- *wx1*, map data --r173
trAc8168 -19- *wx1*, map data --r173
trAc8169 -11- *wx1*, map data --r173
trAc8176 -12- *wx1*, map data --r173
trAc8187 -9- *wx1*, map data --r173
trAc8191 -1- *wx1*, map data --r173
trAc8198 -18- *wx1*, map data --r173
v-N53A, v*-N806C, v*-N1871:* order after TB-9Lc(9) --MNL69:43-46
v-N829A:* order before TB-9Sb(9) --MNL69:43-46
v28, v31: order before TB-9Sb(9) --MNL69:43-46
w-N627B, w*-N1854, w*-N1865:* order before TB-9Sb(9) --MNL69:43-46
wc1 -10.9- *bf1* -4.4- bm4, map data --MNL69:130b
wl-N1803, wl*-N1857:* order before TB-9Sb(9) --MNL69:43-46
wlu4: order after TB-9Lc(9) --MNL69:43-46
wx1-M: sequence --r539
wx1: orthology --r3

CHROMOSOME 10
acc1: npi445 - umc155 - (ncsu2 -2.7- *acc1) - csh::stAc - mgs1* --MNL69:3-4
ad-N377B:* order after TB-10Lb(10) --MNL69:43-46
ad-N590C, ad*-N647:* order before TB-10Sc(10) --MNL69:43-46
csu6, csu46, csu86, csu136, csu140, : map location --r102
dek-MS2181, dek*-MS2425* located to 10S --r597
dek14: order before TB-10Sc(10) --MNL69:43-46
dek15: order after TB-10L19(10) --MNL69:43-46
gl21: order before TB-10Sc(10) --MNL69:43-46
gpa1: origin --r340
gstIIA: php06005 -13.8- *gstIIA* -11.9- *php20646* --MNL69:101
hcf28: order after TB-10La(10) --MNL69:43-46
hcf47: order before TB-10Sc(10) --MNL69:43-46
ij-N504A:* order before TB-10Sc(10) --MNL69:43-46
isu5: map location --r691
l-N31, l*-N195, l*-N392A, l*-N1879, l*-N1908:* order after TB-10L20(10) --MNL69:43-46
l13: order after TB-10L20(10) --MNL69:43-46
l19: order before TB-10Sc(10) --MNL69:43-46
lc1: evolution --r540
mac1: order before TB-10Sc(10) --MNL69:58b
maysin QTL near *r1* in GT114/GT119 F2 --MNL69:53-54
o-N1046:* order before TB-10Sc(10) --MNL69:43-46
o-N1422:* order after TB-10L20(10) --MNL69:43-46
pZmISU167: map location --MNL69:119-120
r1: evolution --r540
ren3: o7 -31- *ren3; r1* -54- *ren3; ren3* -31- T9-10b(10); allele *ren3-Mu1339* --r597
rgh-N1524:* order before TB-10Sc(10) --MNL69:43-46
rgh-N799A:* order after TB-10L19(10) --MNL69:43-46
rlc1 -0- T9-10b(10) *wx1*, map data --MNL69:123
Rp1-DJ4, Rp1-DJ46: compound recombinants --MNL69:99
trAc6059 -24- T9-10b(10); *trAc6059* -50- T9-10(8630)(10) --MNL69:123-124 r173
trAc8180 -11- T9-10b(10); *trAc8180* -18- T9-10(8630)(10) --MNL69:123-124 r173
v-N114A, v*-N354B, v*-N470A:* order after TB-10L20(10) --MNL69:43-46
v29: order after TB-10L20(10) --MNL69:43-46
w-N24:* order after TB-10L20(10) --MNL69:43-46
w2: order after TB-10L20(10) --MNL69:43-46
wsm3: umc155 -14.3- *umc163* -7.1- *wsm3* -8.1- *umc44* -0.8- *umc57* -20.1- *bnl7.49* -23.2- *bnl10.13* --r435

UNPLACED & CLONES
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bsd1: first report --r380
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car30, car757: first report --r14
cbp1, cbp2: first report; Cbp1+MeritL01496, Cbp2+MeritL01497 sequence --r557
cdpk2: first report; *Cdpk2+211D* sequence, restriction map --r194

chn-L00973, chn*-L16798:* first report, sequence --r738
cyc1, cyc2, cyc3, cyc4: first report; *Cyc1+B73, Cyc2+B73, Cyc3+B73 Cyc4+B73,* sequence --r564
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des-GG11, des*-GG21, des*-GG22, des*-GG23:* first report --r242
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hsp70-X73473, hsp70*-X73474*: sequence --r38
htz2, htz3: genelist --r619
iaglu1: first report; *Iaglu1+W64A* sequence --r662
knox8: first report --r313
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Maize1, Maize2: first report, sequence --r700
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Stowaway: first report --r75
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coxI(mtNA), coxII(mtNA), coxIII(mtNA): map location --r206
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rrn5(mtT): map location --r206
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XIII. RECENT MAIZE PUBLICATIONS

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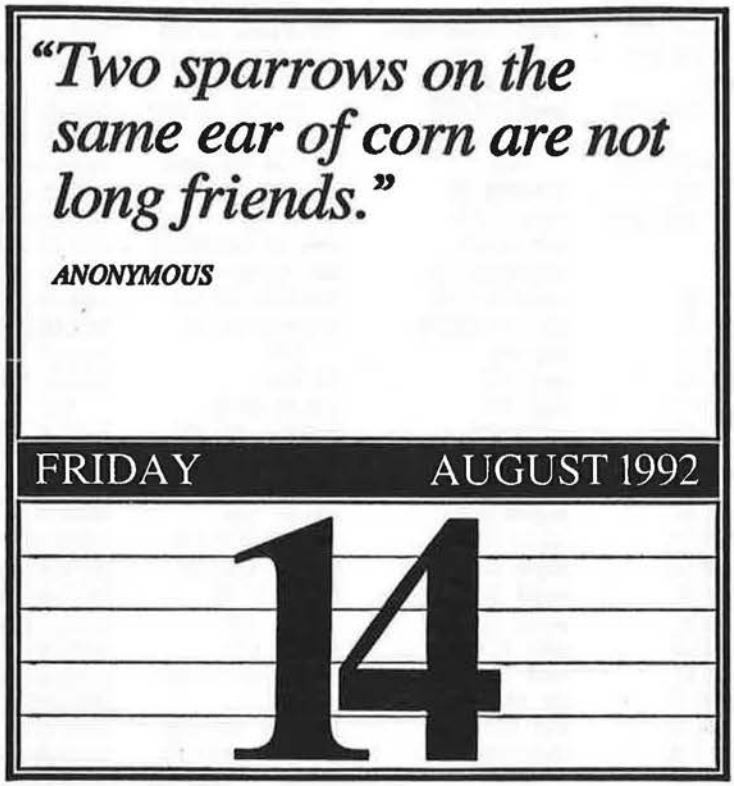

XIV. SYMBOL INDEX

("r" refers to numbered references in the Recent Maize Publications section)

XV. AUTHOR AND NAME INDEX

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From The American Farmer, vol. IX, No. 2, p. 12, March 30, 1827

ON THE SELECTION OF SEED CORN
J. S. Skinner, Esq. [editor]

Dear Sir, *Wake Forest, N.C., Jan. 15, 1827*

I have endeavoured, in conformity to your request, to find the communication I some years ago made to the newspapers, of Joseph Cooper's method of selecting seed corn; but in vain though I have searched several files of papers, and a series of almanacks, in which I remember to have seen it. It was my intention upon this failure, to have furnished you with what my memory retained of the information Mr. Cooper gave me, but it has somehow been delayed to this time, when happening to see in the 2d volume of the American Farmer, a letter from John Taylor, of Caroline, to George W. Jeffreys, recommending a mode of selection that was pointedly condemned by Mr. Cooper's, I have been reminded of what I owe to one who has laid the whole agricultural community under so many obligations. As these conflicting opinions are sustained by perhaps equal authority, it appears to me *now*, as it may have done to you *before*, that it is important both should be known, in order that the fact, now held doubtful, may be subjected to further experiment, and settled by other and uniform testimony.

In a tour to the north, about the year 1810, I paid a visit to the celebrated New Jersey farmer, Joseph Cooper, who then resided on the banks of the Delaware, opposite to Philadelphia. In a walk over his fields, I remarked what I thought the great proportion of ears of corn to the stalks on which they grew; and asked Mr. C. if my impression was correct--if his corn was not unusually prolific? He answered me in the affirmative, and proceeded to inform me how it had attained that quality. It was owing to his selecting for seed the ears from stalks which produced two or more each, while the corn was standing in the field. He illustrated the effects of his peculiar mode of selection by an anecdote. A Judge Pennington (I think that was the name) had made the same observation that I had, and asked for some of his *prolific sort of corn* for seed. Mr. Cooper supplied him. After a few years, the Judge applied for more, saying the seed had degenerated and quite run out, and though at first unusually productive, it was now no better than corn of the common sort. Mr. C. asked if he had observed his injunctions as to the method of saving seed. He said he remembered none; but his method had been, when the corn lay in a heap, to selected the largest and fairest ears. Mr. C. assured him that this was the worst selection he could have made, for these large and fair ears were produced singly upon a stalk, and would infallibly produce stalks bearing no more, and of course an inferior crop. The Judge was once more furnished with seed, upon a promise to select in the prescribed manner, and no complaints were afterwards made of its degeneracy.

What the estimated advantage of Mr. Cooper's mode of selection was, I do not exactly recollect; I only remember the impression that it was more moderate than I had expected, considering the importance he seemed to attach to it. I think he might have calculated, that in the many years he had pursued the plan, his crops had from this cause been improved about 12 or 15 per cent.

If what I recollect of Mr. Cooper's practice and opinions shall appear adapted to promote rational and successful inquiry or to the establishment of a useful fact, you are at liberty to make such use of this as you may think proper; and I shall be gratified that I have been able, in this imperfect manner, and at this protracted period, to furnish an acceptable answer to your inquiry.

With great respect,
I am
Your obedient servant,
CALVIN JONES

From the American Farmer, vol. XIII, No. 5, p. 39, April 15, 1831

THE FARMER.

BALTIMORE, FRIDAY, APRIL 15, 1831

IMPROVEMENT OF CORN.--The Editor of the American Farmer [Gideon B. Smith], has been several years in the habit of improving corn by *crossing* different varieties with decided advantage If he has a variety with small ears, which he deems good in other respects, he plants it in the rows with another kind with large ears, that flowers at the same time; and, at the time of the tassels appearing, carefully cuts away the male flowers (or tassels) of the large eared kind. By this operation, large ears are produced of the small eared kind. There are some kinds of early corn, which, though excellent in other respects for green corn, are very much injured by the coloring matter of their red cobs. This he attempted to remedy last summer by transferring the corn from the red to the white cob in the same way, and he thinks with success. He planted some of the red cob Tuskarora,--which he thinks the best early green corn, in the rows with the largest eared white cob sugar corn he could find, about half and half. As the tassels of the sugar corn made their appearance, he carefully cut them away, leaving the whole to be supplied by the pollen from the tassels or male flowers, of the red cob Tuskarora. the result was, he had the Tuskarora corn on the white cob of the sugar corn, as he desired. From his experiments, the Editor concludes, that any variety of corn may at pleasure, thus be transferred to the cob of any other variety that flowers at the same time; and that if a large eared kind can be found that flowers at the proper time, the smallest eared kind may be made to produce large ears by the above process. He has not extended his experiments to the improvement of the cob of field corn; but, has no doubt, that, by the same process, the thick cob of some kinds may be improved. Suppose the thick cob kind were planted in the row with some other that usually has a small cob, and the tassels of the latter cut off as above directed, would not the desired variety of corn be obtained on the small cob?

From the Evening and Morning Star, Independence, MO, vol. 1, No. 6, p. 48, November, 1832

WORLDLY MATTER
INDIAN CORN.
SINGULAR FACT.

IN the course of some experiments made by the editor of the American Farmer, for the purpose of improving Indian corn last

year, he impregnated the pistils (silk) of the large white Tuscarora with the pollen from the tassels of the golden Sioux. The result was a perfect hybrid between the two. The grain being of a pure brimstone color, of the size and form of the Tuskarora, and like that with eight rows on the cob. It was a most beautiful variety of corn; partaking of all the good qualities of both, without the disadvantage of the large cob and small grain of the golden Sioux. We planted this corn last spring; the stalks were very dwarfish, resembling those of the Sioux, and the corn very early fit for use. It is now ripe, however, and on examining it a day or two since we find that the two original colors have separated, and instead of the brimstone color, we have on every ear grains of the bright yellow Sioux, and the pure white Tuskarora; but the quality of the corn is evidently superior to either of the original parents, although the colors have resumed their original tints. This is, to us, a singular circumstance, and one which we are unable to account for. The only thing analogous to it we have read of, is the proposition advanced by an able writer some time since in the columns of the Farmer, that the offspring of cross breeds of animals, would instead of partaking of the mixt character of their immediate parents, assume that of one or the other of their original progenitors. How far this proposition may hold good with animals we do not know, but it certainly appears to be the case in the vegetable world, at least so far as the fact above stated warrants the formation of an opinion. There is a good deal of difficulty in reconciling the above fact with the law of nature, which requires two parents for the production of every organized being, animal or vegetable If the two kinds of corn which were combined in the hybrids have become again distinct varieties, they are each of them the produce of but one parent the Tuskarora is the produce of a female parent exclusively, and the Sioux that of a male parent; for it must be recollected there was no male Tuskarora nor female Sioux present, either during the origin of the hybrid last year or the subsequent culture and separation of varieties this year. Yet we know, that if we deprive the corn of either the male or female flowers, (tassel or silk,) there will be no corn formed on the cob. How then are we to account for the present fact of the separation of the two varieties? It was this difficulty that made us doubt the correctness of the proposition relative to cross breeds of animals above referred to, and although we have the fact before us in the case of the corn, we are still compelled to doubt its general application. We do not think that each variety has resumed all its original characters; one of them we know it has not--the size of the Sioux grain is larger than the original, and there are but eight rows on the cob; in these respects retaining the hybrid character derived from the Tuskarora; but then the original color and flintiness of the grain is resumed; the Tuskarora has resumed its original character entirely, with the exception of the soft flowerly quality of the grain, the flintiness of the hybrid derived from the Sioux parent is retained. As the Tuskarora was the female parent of the hybrid the number of rows and the size of the grain would of course be like those of that variety, and hence the presence of those characters in the present separated varieties. We should be glad to receive an explanation of this circumstance from some of our practiced naturalists.

[Ed. Note: Thanks to Gerry Neuffer for locating and extracting these items.)

This is an informal newsletter by which working research information on the genetics and cytogenetics of maize is shared. The information and data are shared by Cooperators with the understanding that they will not be used in publications without their specific consent.

Notes for the 1996 Maize Genetics Cooperation Newsletter need to be in the editor's hands by January 1. Be concise, not formal, but include specific data, tables, observations and methods. A double-spaced, letter-quality copy of your text is needed. Please follow the simple style used in this issue (title; authors; use minimal citations in text but list full citations of references). Whenever possible send an electronic version on 3-1/2 or 5-1/4 floppy disk, identifying the operating system (e.g., MS-DOS) and the word processor (e.g., Microsoft Word). Figures, charts and tables should be compact and camera-ready, and provided in electronic form if possible. Please use tabs instead of spaces to separate columns in tables. Send your submissions to E. H. Coe, Jr., 210 Curtis Hall, University of Missouri, Columbia, MO 65211; email: ed@teosinte.agron.Missouri.edu. Submission by email is acceptable, but not preferred.

Subscription information is provided on the form included in this issue, or can be requested from the editor (address and email above).

Author and Name Indexes (and see **MaizeDB**)	
Nos. 3 through 43	Appendix to MNL 44, 1970 (copies available)
Nos. 44 through 50	MNL 50
Nos. 51 to date	Annual in each issue
Symbol Indexes (and see **MaizeDB**)	
Nos. 12 through 35	Appendix to MNL 36, 1962 (copies available)
Nos. 36 through 53	MNL 53
Nos. 54 to date	Annual in each issue
Stock Catalogs	
Marker Stocks	In this issue and **MaizeDB**
Translocations	MNL 55 and **MaizeDB**
Rules of Nomenclature (1995)	In this issue and **MaizeDB**
Cytogenetic Working Maps	MNL 52:129-145; 59:159; 60:149 and **MaizeDB**
Gene List	In this issue and **MaizeDB**
Clone List	MNL 65:106; 65:145, this issue and **MaizeDB**
Working Linkage Maps	In this issue and **MaizeDB**
Plastid Genetic Map	In this issue and **MaizeDB**
Mitochondrial Genetic Maps	MNL 68:211-218 and **MaizeDB**

Cooperators (that means you) need the Stock Center.
The Stock Center needs Cooperators (this means you) to:

(1) Send stocks of new factors you have reported in this Newsletter or in publications, and stocks of new combinations, to the collection.

(2) Inform the Stock Center on your experience with materials received from the collection.

(3) Acknowledge the source of the stocks for research when you publish, and advice or help you have received in development of your research project.

MaizeDB needs Cooperators (this means you) to:

(1) Look at the entries in **MaizeDB** (see section IX in this Newsletter) for "your favorite genes" and send refinements and updates to maryp@teosinte.agron.missouri.edu.

(2) Compile and provide mapping data in full, including counts by phenotypic class for each member of the progeny or map scores for molecular markers; parents and type of cross; recombination percentage and standard error.

(3) Probe or primer information per the information sheet in the back of this issue; fingerprint data indicating enzyme and fragment sizes and defining mapped as well as unmapped fragments.

Cooperators, Clone Home! Each functionally defined clone enhances the map, and mapping information enhances further exploration of the function. Your clone is wanted; please see the information sheet in the back of this issue.