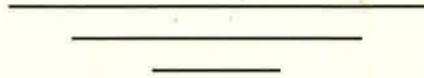


MAIZE GENETICS COOPERATION

NEWSLETTER

72



April 15, 1998

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Word from Joyce Bork, Chair, Science Dept., Webster University: Ginny Harrison died in November after a valiant battle with cancer. The University will soon be naming the biology lab the Ginny Harrison Biology Lab, and in their new building, which should be ready in five to seven years, her name will be on a new lab. A memorial fund has been established at Webster University, to receive gifts in Ginny's name toward new equipment in the Science Dept., at the following address:

(For the Ginny Harrison Fund)

*Webster University
Development Office
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I. FOREWORD	1
II. REPORTS FROM COOPERATORS	2
ALBANY, CALIFORNIA and BERKELEY, CALIFORNIA	
Mapping of the dominant <i>Fasciated</i> locus and generation of revertants --Jackson, D, Hake, S	2
<i>barren inflorescence2</i> is required for the formation of branch meristems --McSteen, PCM, Hake, S	2
<i>ifa1</i> maps to chromosome 1S --Laudencia-Chingcuanco, D, Hake, S	3
AMES, IOWA	
Mutator-induced anomalous alleles of the <i>blue fluorescent1 (bft)</i> locus in maize --Robertson, DS	3
A search for interchromosomal mitotic recombination in maize --Peterson, T	7
BARNUM, MINNESOTA	
A quick way to overview LAMP data --Kutka, FJ	8
BARNUM, MINNESOTA and BIG TIMBER, MONTANA	
Another look at Mandan maize --Kutka, FJ, Christensen, D	8
BEIJING, CHINA	
Identification of maize inbred lines and validation of genetic relationship among maize inbred lines using RAPD markers --Zhang, CL, Sun, ZL, Jin, DM, Sun, SM, Guo, BT, Wang, B	9
Breeding and genetic analysis on the multiplasmic lines of maize (<i>Zea mays</i> L.). I. Breeding and morphological observations --Zeng, M, Liu, S, Yang, T, Liu, Y, Li, S	10
Breeding and genetic analysis on the multiplasmic lines of maize (<i>Zea mays</i> L.). II. Transmission electron microscope observations on mitochondria --Li, J, Zeng, M, Liu, Y, Li, S	11
Breeding and genetic analysis on the multiplasmic lines in maize (<i>Zea mays</i> L.). III. Electron microscope observations on starch grains in the endosperm of maize seed --Li, J, Zeng, M, Li, S and Liu, Y	13
BEIJING, CHINA	
Discovery and primary study on photoperiod-sensitive male sterility in maize --Zhou, H, Deng, Y, Tian, Z	14
BEIJING, CHINA	
Chemical mutagenesis of <i>y1</i> mutants in corn --Zhao, YL, Song, TM, Ma, HP	15
BERGAMO, ITALY	
Chromosome endoreduplication in endosperm cells of IHP and ILP maize strains grown in vitro --Cavallini, A, Bosio, D, Reali, A, Natali, L, Balconi, C, Motto, M	16
Biochemical characterization of "high-lysine" endosperm mutants --Bosio, D, Balconi, C, Motto, M	16
Identification of a putative histone deacetylase RPD3-like gene from maize that complements a yeast <i>RPD3</i> -null mutant --Rossi, V, Hartings, H, Motto, M	17
Comparative analysis of genetic similarity among maize inbred lines detected by RFLPs, RAPDs, SSRs, and AFLPs --Pejic, I, Ajmone-Marsan, P, Morgante, M, Kozumplick, V, Castiglioni, P, Taramino, G, Motto, M	18
Genetic diversity and its relationship to hybrid performance in maize as revealed by RFLP and AFLP markers --Ajmone-Marsan, P, Castiglioni, P, Fusari, F, Kuiper, M, Motto, M	19
Maize transformation with <i>Agrobacterium tumefaciens</i> --Lupotto, E, Reali, A, Passera, S, Chan, M-T	20
BERKELEY, CALIFORNIA	
<i>reverse germ orientation 1</i> mutants make three florets per spikelet --Kaplinsky, NK, Freeling, M	22
BLACKSBURG, VIRGINIA	
DIMBOA-glu concentration in different taxa of teosinte --Shahid, M, Esen, A	23
The difference between two β -glucosidase allozymes at the amino acid sequence level --Shahid, M, Bandaranayake, H, Esen, A	24
Polymorphism of β -glucosidase isozymes Glu1 and Glu2 in maize (<i>Zea mays</i> L.) --Shahid, M, Esen, A	24
CAMPINAS, SAO PAULO, BRASIL	
Preliminary cytological studies on a maize strain carrying the <i>latent1</i> supergene --Pierozzi, NI, Miranda, LEC de, Miranda, LT de	25
CHESTNUT HILL, MASSACHUSETTS	
Continued studies on the genic stability of the progeny of maize anther culture-derived microspore plants --Ting, YC, Nguyen, DQ	26
COLOGNE, GERMANY	
A confocal laser scanning microscopy study of 4 embryo specific mutants --Elster, R, Bommert, P, Werr, W	26
COLUMBIA, MISSOURI and ATHENS, GEORGIA	
Functional variants of a maysin QTL - - - Is there a difference? --Lee, EA, Byrne, PF, McMullen, MD, Snook, ME, Coe, EH	27
DNIEPROPETROVSK, UKRAINE	
Effect of metholachlor on pollen germination of different maize genotypes --Satarova, TN	28
DULUTH, MINNESOTA	
Isolated meiocyte culture of polyploid microspores allows direct observation of abnormal cell division --Wolfe, KW, Liu, Q	28
Microfilament distribution in the maize male sterile mutants <i>ms2</i> and polyploid --Wolfe, KW, Liu, Q	29
FREIBURG, GERMANY	
Isolation of the new necrotic root mutant <i>brt1</i> --Hochholdinger, F, Feix, G	29
Isolation of the new root mutant <i>slr1</i> affecting lateral root formation --Hochholdinger, F, Park, WJ, Feix, G	30
Tiller formation in Gaspé Flint is not affected by the <i>rtcs</i> mutation --Hochholdinger, F, Feix, G	30
HAMBURG, GERMANY	
Gene expression during fertilization and zygote development --Dresselhaus, T, Cordts, S, Heuer, S, Bantin, J, Lörz, H, Kranz, E	31

Isolation and characterization of genes which are strongly down-regulated after fertilization --Cordts, S, Kranz, E, Brettschneider, R, Lörz, H, Dresselhaus, T.....	31
Expression analysis of MADS box genes in pollen, egg cells and zygotes --Heuer, S, Lörz, H, Dresselhaus, T.....	32
IBARAKI, JAPAN	
Nitrous oxide (N ₂ O) is effective for chromosome counting in maize --Kato, A.....	32
IRKUTSK, RUSSIA	
Differential redox regulation by glutathione of translation in isolated mitochondria --Konstantinov, YM, Subota, IY, Arziev, AS.....	33
A leucine motif in the amino acid sequence of mitochondrial ribosomal protein S13: possible involvement in protein oligomerization --Konstantinov, YM, Arziev, AS.....	33
JOHNSTON, IOWA	
Molecular analysis of T0 plants transformed by <i>Agrobacterium</i> and comparison of <i>Agrobacterium</i> -mediated transformation with bombardment transformation in maize --Zhao, ZY, Gu, W, Cai, T, Tagliani, LA, Hondred, D, Bond, D, Krell, S, Rudert, ML, Bruce, WB, Pierce, DA.....	34
New chromosome 10S male-sterile mutant: <i>ms29</i> --Trimnell, MR, Fox, TW, Albertsen, MC.....	37
New chromosome 2L male-sterile mutants <i>ms30</i> and <i>ms31</i> --Trimnell, MR, Fox, TW, Albertsen, MC.....	38
JOHNSTON, IOWA and FORT COLLINS, COLORADO and URBANA, ILLINOIS and GRAND FORKS, NORTH DAKOTA	
Two new alleles of the male-sterile mutant <i>ms45</i> --Trimnell, MR, Bedinger, P, Patterson, E, Sheridan, W, Albertsen, MC.....	38
Are there too many genes in maize? --Helentjaris, T, Briggs, K.....	39
KISHINEV, MOLDOVA	
Experimental mutagenesis of corn --Lysikov, VN.....	40
Chromosomal abnormalities in maize regenerants in R1 --Kravchenko OA, Lysikov, VN, Palii, AF.....	43
Dependence of some characters of the maize male gametophyte on gamma radiation and genotype --Kravchenko, OA.....	44
Possibilities for identifying major genes controlling quantitative traits using maternal haploids of maize --Chalyk, ST, Chebotar, OD, Bylich, VG, Chernomorets, AA.....	44
A new mutator system? --Krivov, NV.....	45
Tests for allelism between <i>ys</i> mutants differing in origin --Krivov, NV, Lysikov, VN.....	45
The upset of meiosis at maize pollination with high dose gamma-irradiated pollen --Romanova, IM.....	46
On the possible mechanism of 6-mercaptopurine and laser radiation-induced DNA breaks and chromosome aberrations in <i>Z. mays</i> --Burlilov, VK, Paschenko, VM, Lysikov, VN, Basova, IA.....	46
LACOMBE, ALBERTA, CANADA	
Xenia assisted reciprocal recurrent selection in maize --Wolfe, RI.....	47
LLAVALLOL, ARGENTINA	
Early waxy and high quality protein maize inbreds: study of fungal diseases --Corcuera, VR, Sandoval, MC.....	48
<i>Zea mays</i> inbreds resistant to different populations of <i>Ustilago maydis</i> (DC) Corda --Sandoval, MC, Corcuera, VR.....	49
General combining ability of flint, waxy and dent maize early inbreds --Corcuera, VR, Sandoval, MC, Naranjo, CA.....	49
Studies to determine the degree of resistance to <i>Ustilago maydis</i> D.C. Corda in wild and cultivated species of the genus <i>Zea</i> --Astiz Gassó, MM, Molina, MdelC.....	50
The male transmission rate of B-chromosomes is controlled by the A-chromosomes in maize --Chiavarino, AM, Rosato, M, Rosi, P, Poggio, L, Naranjo, CA.....	52
Nuclear DNA amount and doses of B-chromosomes in native populations of maize --Rosato, M, Chiavarino, AM, Naranjo, CA, Poggio, L.....	53
Heterochromatic bands and doses of B-chromosomes in native populations of maize --Rosato, M, Chiavarino, AM, Poggio, L, Naranjo, CA.....	53
Genomic in situ hybridization (GISH) technique for determining the origin of the paired and unpaired chromosomes in the 2n= 30 hybrid <i>Zea perennis</i> x <i>Zea luxurians</i> --Poggio, L, Confalonieri, V, Comas, C, Gonzalez, G, Naranjo, CA.....	54
LOMAS DE ZAMORA, ARGENTINA	
Forage aptitude in maize populations --Torrecillas, M, Bertoia, LM.....	55
LONDON, ONTARIO	
The thermal induction of HSP 18 mRNA in <i>Zea mays</i> callus --Friedberg, JN, Yang, Z, Walden, DB.....	56
The induction of mRNA from 18 kDa HSP genes in maize lateral roots --Yang, Z, Greyson, RI, Bouchard, RA, Walden, DB.....	56
Nucleolar fusion in different maize cell-types --Maillet, DS, Quinn, CE, Friedberg, JN, Walden, DB.....	56
MILAN, ITALY	
The <i>fused leaves</i> mutant in maize is altered in the embryo and juvenile leaf morphology --Frascarolo, P, Landoni, M, Dolfini, S, Gavazzi, G, Consonni, G.....	58
MILAN, ITALY	
Maize mutants defective in embryogenesis --Giulini, AP, Busti, E, Consonni, G, Dolfini, AS, Furini, A, MacCabe, AP, Gavazzi, G.....	58
MOSCOW, RUSSIA and COLUMBIA, MISSOURI	
The major quantitative trait loci for plant stature, development and yield are general manifestations of developmental gene clusters --Khavkin, E, Coe, EH.....	60
NEW HAVEN, CONNECTICUT	
<i>Rough sheath2</i> encodes a MYB-domain protein that negatively regulates meristem-specific KNOTTED1-like homeobox proteins in the leaf --Timmermans, M, Nelson, T.....	66
<i>midribless1 (mrl1)</i> is required for the correct timing of vascular initiation and the coordination of subsequent developmental events in the maize leaf --Jankovsky, JP, Nelson, T.....	67

NOVOSIBIRSK, RUSSIA	
Perspectives of developing apomixis in maize --Sokolov, VA, Lukina, LA, Khatypova, IV	67
RALEIGH, NORTH CAROLINA	
Partial isolation and characterization of the maize alternative oxidase gene --Polidoros, AN, Mylona, PV, Scandalios, JG	68
ST. PAUL, MINNESOTA	
Spontaneous activation of transposable elements following an interracial cross in maize --Vladutu, CI, Phillips, RL	70
SAINT PAUL, MINNESOTA and BOLOGNA, ITALY	
Bulked segregant analysis confirms the importance of the region near <i>umc89a</i> for days to pollen shed in maize --Tuberosa, R, Salvi, S, Phillips, RL	71
Mapping QTLs for ABA concentration in leaves of a maize cross segregating for anthesis date --Tuberosa, R, Parentoni, S, Kim, TS, Sanguineti, MC, Phillips, RL	72
SARATOV, RUSSIA	
Apomixis and endosperm development --Tyrnov, VS	73
Nuclear-plasmic interaction and instability of the maize genome --Zavalishina, AS, Tyrnov, VS	74
SOFIA, BULGARIA	
Maize immature embryo culture --Todorova, L, Kruleva, M, Krapchev, B, Nedev, T	76
TAICHUNG, TAIWAN	
Map position of the centromere on chromosomes 1 and 9 --Lin, B-Y, Chang, S-J	76
Chromosome rearrangements associated with B-A translocations --Lin, B-Y, Chang, S-J	77
Using B-A translocations to isolate AFLPs on the proximal half of chromosome 10 --Cheng, Y-M, Lin, B-Y	77
Two RFLPs included in the <i>r-X1</i> deletion --Peng, S-F, Lin, B-Y	78
URBANA, ILLINOIS	
Variation for ABA concentration in tassels from H99xFR16 and H99xPa91 F4 families --Wassom, JJ, Mei, C, Rocheford, TR, Widholm, JM	78
URBANA, ILINOIS	
Three-point linkage data for <i>su1 bm3 gl7</i> on 4S --Stinard, P	79
Results of TB tests of symbolized unplaced mutants --Stinard, P, Jackson, JD	79
Linkage tests of Non-waxy (<i>Waxy1</i>) reciprocal translocations involving chromosome 9 at the MGCSC --Jackson, JD, Stinard, P	79
<i>zb1</i> and <i>zb2</i> stocks at the Stock Center are allelic to <i>zb3</i> --Stinard, P, Sachs, MM	81
Linkage tests of <i>waxy1</i> marked reciprocal translocations at the MGCSC --Jackson, JD, Stinard, P	81
WALTHAM, MASSACHUSETTS	
Supermaize research --Galinat, WC	82
Plant - animal symbiosis --Galinat, WC	83
Maize genetics ten centuries before Mendel, as suggested by Indian art --Galinat, WC	83
WOODWARD, OKLAHOMA	
Improvement of anther culture response of apomictic maize- <i>Tripsacum</i> hybrids --Li, YG, Bai, DP, Kindiger, B	84
Plant regeneration from somatic culture of apomictic maize- <i>Tripsacum</i> hybrids --Li, YG, Kindiger, B	84
WOOSTER, OHIO and LONDON, ONTARIO	
Quantitation of 18kDa HSP mRNA accumulation in metal-ion insulted maize seedling radicles and plumules --Bouchard, RA, Yang, Z, Walden, DB	85
ZHENGZHOU, CHINA and WUHAN, CHINA	
Polymorphisms of synthesized populations from Chinese and exotic populations at 17 isozyme loci --Tie, SG, Zheng YL, Liu, JL	86
III. ADDRESS LIST	87
IV. MAIZE GENETICS COOPERATION STOCK CENTER	108
V. MAIZE DATABASE	116
VI. MAIZE PROBE BANK	117
VII. WORKING MAPS	118
VIII. MAIZE GENETICS COOPERATION - 1929	129
IX. SYMBOL INDEX	131
X. AUTHOR INDEX	133

Remembering

C. H. Li
(new translation Li Jingxiong)

Virginia Harrison

Cooperators

I. FOREWORD

The Notes in this Newsletter are voluntarily shared "Conversations among Cooperators." This is not a refereed journal -- the data and ideas here are not published but are presented with the understanding that they will not be used in publications without specific consent of the authors. Cooperators provide brief technical notes, updates, mutant descriptions, segregation ratios, tables of mapping data, developmental and anatomical information and techniques, clones, biochemical functions, and the like. Comprehensive material and analyses are better directed to formal publication. Maize Cooperators have the tradition of sharing information with colleagues, not only in MNL but also in many unheralded conversations, correspondence, and shared stocks and clones. By sharing our research information, we contribute to the advancement of biology and to the power of shared technical knowledge.

Beginning in 1997, MNL became a Virtual Hotletter and Linkletter! Notes submitted at any time go verbatim into MaizeDB as received, flagged as future items for the next issue (<http://www.agron.missouri.edu/mnl/73/>). We progressively incorporate figures and tables as electronic images, and we link the articles to database objects for user access and for the development of summaries and syntheses such as the Genelist, Maps, and Indexes. In parallel, redacting of copy (editing and formatting) in preparation for the press proceeds by desktop processing. After the deadline passed, the print version of this issue, simply containing the Notes received to that date; the Address List; Stock Center Report and Stock List; Maize Database; Probe Bank; and UMC 1998 Molecular Marker Map, has been finalized and sent to the press. Indexes to Symbols and to Authors and Names cited in this MNL issue are included. Assembly of portions of MNL that represent syntheses of information (e.g., Gene List, Genetic Maps, Zealand, Recent Maize Publications) will be done periodically insofar as possible, but on a separate schedule from MNL. Syntheses will also be present in MaizeDB, where they can be viewed or printed by MaizeDB users.

A new item of correspondence from 1929, found during the research of Lee Kass (see MNL 71, page iv), is reproduced on pages 130-131 in this issue.

Gifts to the Endowment Fund for support of the Newsletter have grown to well over \$125,000. 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. Gifts to the Endowment Fund are very much appreciated, to assure that costs of production are met, but more importantly to underwrite distribution to deserving institutions, libraries and individuals. A bequest from Ginny Harrison to the Endowment Fund furthers our emphasis on teaching and education initiatives in MNL and on the net.

The continuity and support necessary for collecting genetic and molecular information from the literature and from individual contributions; evaluating; and preparing gene lists, maps, and similar syntheses, is made possible by the USDA - Agricultural Research Service through the MaizeDB program, of which Mary Polacco is Curator. We urge you with strongest enthusiasm to use, assess, and contribute to the database.

Shirley Kowalewski again refined and redacted the copy; cursed diverse electronic sources and exotic scripts or performed hand entry; structured indexes; questioned quality or content; and gave the editor a quality technician's creative advice. Beth Bennett contributed with diligence and precision to many tasks, including library and literature work, composition, and checking of accuracy and completeness. My colleagues Mary Polacco and Mike McMullen have never yet refused to give invaluable advice and encouragement. At University Printing Services, Yvonne Ball and the printshop staff again efficiently ensured the job was done promptly and well.

Information about the 1999 Maize Genetics Conference, at Grand Geneva Lodge, Lake Geneva, Wisconsin, March 11-14, 1999, is on the MaizeDB Web, <http://www.agron.missouri.edu>, and information packets will be mailed to former attendees in November 1998; others may request the mailing by providing their address to Coe. The program and abstracts are prepared by Bill Sheridan; back copies are available from Coe. Electronic submission and "Webification" of abstracts by Mary Polacco, started in 1996, will be used for the 1999 Conference. The Steering Committee for the 1999 Maize Genetics Conference is:

Becky Boston	Ben Bowen	Kelly Dawe
Al Kriz	Jane Langdale	Mike McMullen
Neelima Sinha	Julie Vogel, Co-Chair	Cliff Weil, Co-Chair
Ex officio: Karen Cone, Treasurer	Marty Sachs, local coordinator	

Preparing notes for the next issue (Number 73, 1999)?
SEND YOUR ITEMS ANYTIME; NOW IS YOUR BEST TIME.
See details inside the back cover.

Your clone can be mapped, and deposited in the Maize Probe Bank. Please see the Clone Information Sheet in the back of this issue, or see <http://www.agron.missouri.edu/Coop/clonesheet96.html>.

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Editor Coe

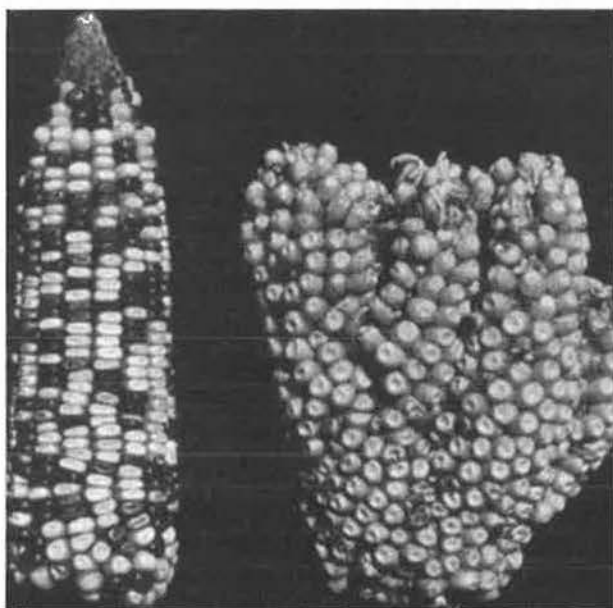
ALBANY, CALIFORNIA
Plant Gene Expression Center
BERKELEY, CALIFORNIA
University of California

Mapping of the dominant *Fasciated* locus and generation of revertants

--Jackson, D*, Hake, S

*Current address: Cold Spring Harbor Lab., PO Box 100,
Cold Spring Harbor, NY 11724

The dominant *Fasciated* (*Fas1*) mutation causes enlargement and splitting of the primary inflorescence apical meristems. *Fas1* plants have huge branched ears and split central tassel branches (Figure, normal ear on left, *Fas1* ear on right). The ontogeny of *Fas1* inflorescences has been characterized in detail by Orr and co-workers (Orr, AR, Haas, G and Sundberg, MD, *AJB* 84: 723-34, 1997).



We mapped *Fas1* using the standard set of *waxy* reciprocal translocations. *Fas1* plants (*Wx1*) were crossed to each translocation tester and F1 *Fas1* plants were crossed by *wx1* testers. Analysis of the F2 progeny from several different translocation crosses indicated that *Fas1* was in repulsion to *wx1*, suggesting that *Fas1* is on chromosome 9. To confirm this we crossed *Fas1* by *Rolled*, and outcrossed the double mutants to standard inbreds. In the F2, we found that *Fas1* was in repulsion to *Rld1*, (38 +/+; 89 *Fas*+; 85 +/*Rld*; 27 *Rld*/*Fas* individuals) indicating a map distance of approximately 27 cM between *Rld1* and *Fas1*. Since *Rld1* is close to the distal tip of 9L, and we know that *Fas1* is linked to *wx1*, it is most likely that *Fas1* is proximal to *Rld1* and we tentatively place it at 9L-110+/-.

In an attempt to characterize the molecular lesion in the *Fas1* locus, we screened for Mutator induced revertants. *Mu* active, homozygous *Fas1* plants were crossed by normal pollen from the W23 inbred line, and we screened for plants with normal ears and tassels in the F1 generation. In a small pilot screen we recovered five normal plants in a population of only seven thousand plants, a frequency much higher than expected for a targeted *Mu* insertion. Since *Fas1* plants were used as the female parent in the initial

crosses it is unlikely that the normal plants were the result of contamination. Test crossing of the putative revertants gave plants with normal inflorescences, and we did not see any new recessive phenotypes segregating in the self progeny.

The high frequency of reversion of the *Fas1* locus is similar to the frequency observed for revertants of the dominant *Knotted1-O* allele. *Kn1-O* is caused by a tandem duplication and many of the revertants resulted from loss of the duplicated copy rather than transposon insertion (Veit, B et al., *Genetics* 125:623-31, 1990). If a similar phenomenon is causing reversion of the dominant *Fas1* phenotype, it may not be possible to isolate the *Fas1* gene by this strategy.

barren inflorescence2 is required for the formation of branch meristems

--McSteen, PCM, Hake, S

barren inflorescence2 (*bif2*) mutants are characterized by a reduction in the number of spikelets in the tassel and ear and a reduction in the number of lateral branches on the tassel (Fig. 1B). An EMS allele, *bif2-2354*, and a *Mu*-induced allele, *bif2-47330*, have previously been described (Briggs, S and Johal, G, *MNL* 66:51 1992; Neuffer, MG and Briggs, S, *MNL* 68:28, 1994). We describe two new alleles, isolated from lines containing active *Mutator* (*Mu*) elements, which were kindly given to us by Paul Chomet and Guri Johal. The mutants fail to complement *bif2* and hence are designated, *bif2-1606* and *bif2-70*. The new alleles have a more severe phenotype than *bif2-2354*. Usually, a thin bare unbranched rachis (inflorescence stem) forms in place of the tassel (Fig. 1C). Spikelets that form infrequently are usually single instead of paired and have elongated pedicels. Occasionally, one or two lateral branches, with a limited number of spikelets, are produced. The ear is similarly affected: ear shoots form but when the husk leaves are opened, only a bare rachis is present (not shown). A few florets may be found at the base of the ear or in small patches on the rachis. In addition, the ear is almost always fasciated. In contrast to the dramatic effect on inflorescence development, vegetative development appears normal. We propose that *bif2* is required for the formation of branches and spikelets. Infrequent branch and spikelet formation may be due to redundancy with other *barren inflorescence* loci.

Scanning electron microscopy was used to determine why *bif2-1606* mutants do not produce branches and spikelets. The first step in normal inflorescence development is the formation of branch meristems by the inflorescence meristem (Fig. 1D). Branch meristems at the base of the tassel reiterate the main spike to produce long branches. Branch meristems on the main spike and the lateral branches produce two spikelet meristems, each of which produces two floret meristems (Fig. 1D). In *bif2-1606* inflorescences, no branch meristems were produced by the inflorescence meristem (Fig. 1E). Ripples or undulations on the surface of the rachis may represent the suppressed bract primordia which normally subtend branch meristems. Therefore, we propose that *bif2* mutants do not produce branches and spikelets because they are blocked in inflorescence development before the production of branch meristems.

This conclusion was confirmed by RNA in situ hybridization using *kn1* as a marker for meristematic tissue. In normal inflorescences, *kn1* is expressed in the inflorescence, branch, spikelet and floret meristems (Jackson, D et al., *Development* 120:405-413, 1994; Fig. 1F). In *bif2-1606* inflorescences, *kn1*

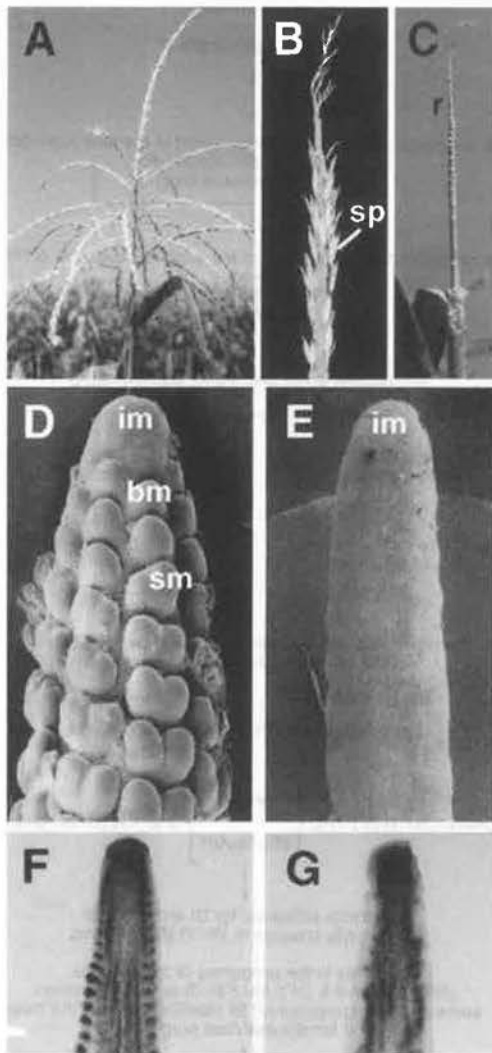


Figure 1. A) Normal tassel. B) *bif2-2354* tassel with no branches and a reduced number of spikelets on the main spike. C) *bif2-1606* tassel with no branches or spikelets. D) SEM of a normal tassel. E) SEM of a *bif2-1606* tassel showing that branch meristems are not produced. F) RNA *in-situ* hybridization showing *kn1* expression (blue) in a normal ear. G) RNA *in-situ* hybridization of *kn1* expression in a *bif2-1606* ear. Abbreviations: sp, spikelet, r, rachis, im, inflorescence meristem, bm, branch meristem, sm, spikelet meristem.

was expressed in the inflorescence meristem but was not expressed on the flanks of the inflorescence meristem where branch meristems initiate (Fig. 1G). Similar results were obtained with *bif2-70*. This confirms that *bif2* mutants do not produce branch meristems. Whether *bif2* is required for the initiation or the maintenance of branch meristems is currently being investigated.

Our results show that *bif2* plays a fundamental role in formation of branch meristems which is the first step in the production of florets. To determine the molecular mechanism by which it acts we are currently attempting to clone *bif2* by transposon tagging.

ifa1 maps to chromosome 1S

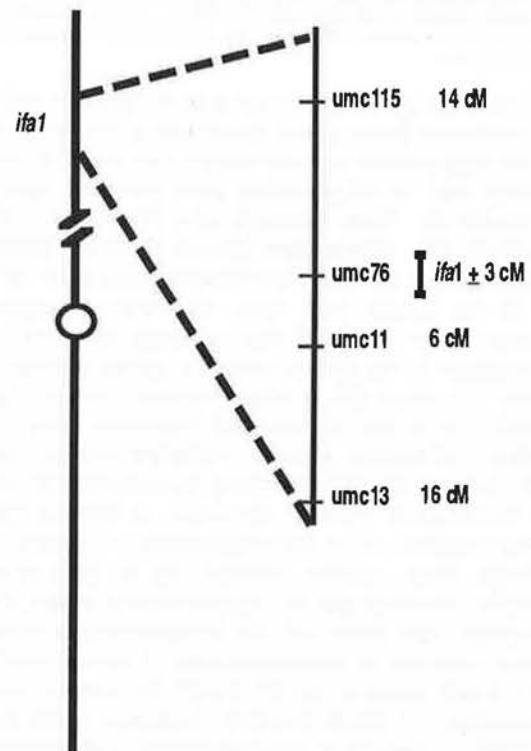
--Laudencia-Chinguanco, D, Hake, S

indeterminate floral apex1 (ifa1, a.k.a. nuc1) is a recessive mutation that perturbs floral and inflorescence meristems

(MNL71:2). Mutant plants often develop extra spikelets per branch in the main inflorescence and more than two flowers form per spikelet. Further, an inflorescence-like structure often develops from the center of the carpel (in ear florets) and at the center of the stamens (in tassel florets). We interpret this phenotype to suggest that *ifa1* is required in the suppression or negative regulation of meristem indeterminacy.

To determine the chromosome location of *ifa1*, we crossed homozygous plants as male to the standard set of *waxy*-marked reciprocal translocation lines. Non-*waxy* and *waxy* kernels from the ears of selfed F1 plants were planted and scored for *ifa1* phenotype. Only the F2 progenies of the cross between *ifa1* and T1-9c showed evidence of linkage (repulsion): 0/34 *ifa1* in *waxy* plants and 26/78 *ifa1* in non-*waxy* plants. This segregation distortion suggests that *ifa1* is on the short arm of chromosome 1.

Several RFLP markers were used to further define the location of *ifa1* on chromosome 1S. DNA from 18 plants showing the mutant phenotype was extracted and analyzed for polymorphism. The figure shows the calculated linkage map of



these markers with reference to *ifa1*. No recombination was observed between *umc76* and *ifa1* out of 36 chromosomes examined.

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Mutator-induced anomalous alleles of the blue fluorescent1 (*bf1*) locus in maize

--Robertson, DS

The original blue fluorescent1 (*Bf1-R*) mutant allele was described by Teas and Anderson (Proc. Nat. Acad. Sci. USA 37:645, 1951). This mutant accumulates the β -glucoside of

anthranilic acid, anthranilic acid and an unknown compound due to altered inhibition of anthranilic acid metabolism (Singh & Widholm, Biochem. Genet. 13:357, 1975). Homozygous mutant seedlings, and the anthers of plants homozygous or heterozygous for the mutant allele, fluoresce blue under ultraviolet light. Mutant alleles have been designated *Bf1*, and normal alleles *bf1* (see Table 1 for the description of symbols used in this note to designate the alleles and phenotypes involving this locus.)

Table 1. Description of symbols used to designate alleles and phenotypes involving the blue fluorescent 1 locus.

<i>bf1</i>	Symbol designation for this locus ^a
<i>bf1</i>	Nonmutant allele at this locus ^a
<i>Bf1-R</i>	Mutant reference allele
<i>Bf1-Mu</i>	Any mutant allele induced by the <i>Mutator</i> system
<i>Bf1-Mu-044-3</i>	<i>Mutator</i> -induced allele described in this note
<i>Bf1-Mu-544-3</i>	<i>Mutator</i> -induced allele described in this note
<i>Bf1</i>	Symbol used to designate the blue fluorescent phenotype as expressed in seedlings and/or anthers of plants homozygous or heterozygous for a mutant allele
<i>bf</i>	Symbol used to designate the nonmutant phenotype

^aBecause the original symbol chosen for the mutant allele at this locus was *Bf1*, the nonmutant allele must be designated by *bf1*, which is also the symbol for the locus. The reader will be able to determine from the context in which this symbol is used whether it represents the locus or the nonmutant allele.

From seeds produced in an isolation plot (see schematic in Fig. 1) in which the female parent plants were active *Mutator* plants and the male parents were homozygous for the *Bf1-R* allele, 214 seedlings with the *Bf* phenotype were recovered (genotype = *Bf1-Mu/Bf1-R*). These seedlings were transferred to the field. At maturity their anthers were checked for pollen abortion and the *Bf* phenotype and they were then pollinated by *bf1 bf1* plants. Half of the kernels from these ears were expected to be heterozygous for the *Bf1-R* allele (genotype *Bf1-R/bf1*) and half heterozygous for the *Bf1-Mu* allele (an isolate, genotype = *Bf1-Mu/bf1*). For about 50% of *Mutator*-induced mutants generally, mutability can be used to distinguish the induced mutant from the standard mutant allele. However, mutability could not be utilized for this purpose with *Bf1-Mu* mutants because the intensity of the blue fluorescence made it impossible to observe the small revertant sectors, which are characteristic of *Mutator*-induced mutants. Thus, another criterion had to be employed to distinguish the plants with the *Mutator*-induced mutant alleles (or aberrations) from those with the standard mutant allele. The criterion used was to screen progenies of self-pollinated plants from the outcrosses of the *Bf1-Mu/Bf1-R* plants as females to homozygous *bf1* stocks (resulting genotypes = *Bf1-Mu/bf1* & *Bf1-R/bf1*) for absence or reduced numbers of *Bf* seedlings.

The following are some of the events that might be responsible for such a phenomenon and possible observations that would be expected if the events occurred: 1) Induced deletions involving the *bf1* locus, which were not male transmissible. Plants heterozygous for such deletions when outcrossed as males to *Bf1-R* tester plants would have no *Bf* seedlings in the outcross progenies but 50% or less *Bf* seedlings would be expected when the plants were crossed as females to the same tester stocks [Putative *Mutator*-induced deletions involving the *yg2* locus that were not male transmissible were found by Robertson and Stinard (Genetics 115:353, 1987)]. Also pollen abortion might be expected in plants heterozygous for such deletions [See Robertson (Genome 37:433, 1996) for the description of a *Bf1-Mu* deletion characterized by the segregation of abortive pollen grains]. 2) The event, when homozygous, could result in the failure of the endosperm to

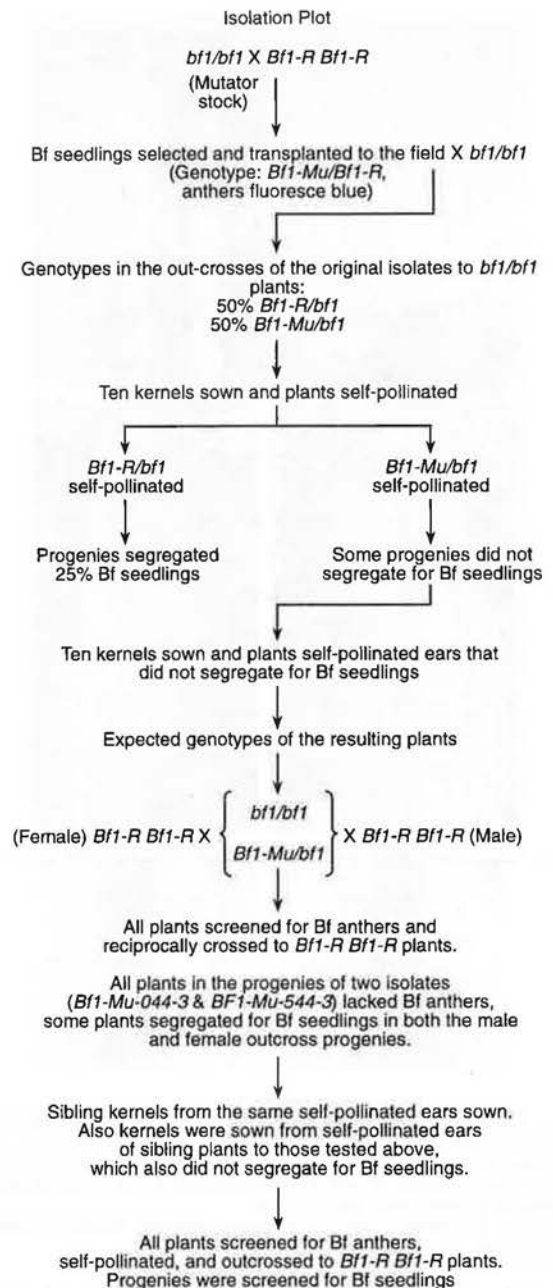


Figure 1. Schematic diagram of the method used to produce and analyze the *Mutator*-induced *Bf1-Mu* mutants described in this study.

develop. If this occurred, self-pollinated ears of heterozygous plants would not have normal seed set. 3) The homozygous event could interfere with the development of the germ. Such an event would result in the segregation of germless kernels on the ears of self-pollinated heterozygous plants. 4) The event could affect the ability of homozygous kernels to germinate. If germination was blocked completely self-pollinated heterozygous ears would produce no *Bf* seedlings. If germination was not always affected or if it was just delayed, a less than expected number of *Bf* seedlings might be expected in some self progenies. 5) The mutation event could affect the viability of the female or male gametophyte. If the male gametophyte was involved, reduced transmission of the mutant allele would be expected in male

outcrosses. If the female gametophyte was affected, reduced seed set would be expected.

Ten kernels from each of the outcrosses of 100 of the *Bf1-Mu/Bf1-R* heterozygotes were sown and the resulting plants (genotypes = *Bf1-Mu/bf1* & *Bf1-R/bf1*) self-pollinated. In 38 of the outcross progenies tested, one or more of the progenies of the self-pollinated plants did not segregate for any Bf seedlings. To determine which of the 5 possible events described above was responsible for the absence of Bf seedlings, ten sibling kernels from each of these 38 families were sown and the resulting plants were screened for Bf anthers. This screening involved the systematic collection of the central spikes of all plants with freshly extruded anthers, arranged in a carrier that would allow the determination of the plant of origin for each. The anthers were screened in a dark room under ultraviolet light, and a washer was placed firmly on each spike with fluorescent anthers so that they could be recognized in normal light. All plants were screened for pollen sterility and, whether or not they had Bf anthers, they were reciprocally crossed to plants homozygous for the *Bf1-R* mutant allele. The seedlings from the progeny of the reciprocal crosses were then screened for the Bf phenotype (see Fig. 1).

In families of two of the 38 isolates, *Bf1-Mu-044-3* and *Bf1-Mu-544-3*, all the plants lacked Bf anthers but some of the plants segregated for Bf seedlings in both the female and male outcross progenies to the homozygous *Bf1-R* tester stocks (Table 2). The

Table 2. Segregation of Bf seedlings in reciprocal crosses to homozygous *Bf1-R* plants of *Bf1-Mu* isolates that lacked Bf anthers.^a

Family and plant number	Female outcross progenies				Male outcross progenies			
	bf	Bf	Total	%Bf	bf	Bf	Total	%Bf
<i>Bf1-Mu-044-3</i>								
5003-1	36	6	42	14.29**	38	10	48	20.83**
-2	--	--	--	----	36	19	55	34.54*
-3	24	14	38	36.84 ^{ns}	44	4	48	8.33(**)
-6	40	8	48	16.67**	45	6	51	11.76(**)
TOTAL	100	28	128	21.86**	163	39	202	19.31**
Contingency chi square = 0.4978 ^{ns}								
<i>Bf1-Mu-544-3</i>								
5033-1 ^{bc}	18	16	34	47.06 ^{ns}	22	19	41	46.34 ^{ns}
-4	20	19	39	48.72 ^{ns}	30	25	55	45.45 ^{ns}
-5 ^{bc}	0	32	32	100.00	(1) ^d	38	38	100.00
-7 (3) ^d	0	0	0	----	0	24	24	100.00
-8	18	24	42	57.14 ^{ns}	19	16	35	45.71 ^{ns}
-9	--	--	--	----	25	11	36	30.56*
TOTAL	56	59	115	51.30 ^{ns}	96	71	167	42.51 ^{ns}
(Minus #5 & #7)								
Contingency chi square = 2.4859 ^{ns}								

- *,** The chi square tests for deviations from a 1:1 ratio were significant at the 5% and 1% levels, respectively.
- (**) The chi square tests for significant deviation from a 1:1 ratio were not dependable because one class had fewer than 5 individuals.
- ^{ns} Chi square tests for deviations from a 1:1 ratio in the outcrosses or the contingency tests were not significant.
- ^a All plants carrying the *Bf1-Mu* alleles in this table had normal pollen. For *Bf1-Mu-044-3*, six plants were crossed, four plants were heterozygous for the mutant allele and two plants were homozygous for the nonmutant allele. For *Bf1-Mu-544-3*, nine plants were crossed, two plants were homozygous for the mutant allele, four plants were heterozygous and three plants were homozygous for the nonmutant allele.
- ^b The ears of these plants were semi-sterile. The ears of all other plants had normal seed set.
- ^c These plants had a few anthers with a single small fluorescent spot.
- ^d These plants probably are contaminants.

Bf1-Mu-544-3 and the *Bf1-Mu-044-3* isolates differed in that both female and male outcross progenies of all but 2 *Bf1-Mu-544-3* plants (5033-5 & 7) segregated for 50% Bf seedlings,

whereas most of the crosses of *Bf1-Mu-044-3* had significantly less than 50% Bf seedlings in the progenies of both the male and female outcrosses. There was not a significant difference in the percentages of Bf seedlings in the female and male outcross progenies of this mutant (Table 2). These two isolates also differed in two other respects. While no outcross progenies of *Bf1-Mu-044-3* had 100% Bf seedlings, two plants of *Bf1-Mu-544-3* (5033-5 & 7) had only Bf seedlings in their outcross progenies and thus were homozygous for the mutant allele. These observations along with the lack of pollen abortion (see Tables 2 & 3) rule out deletions as an explanation for the lack of Bf seedlings in the original self progenies. The *Bf1-Mu-544-3* isolate also differed from *Bf1-Mu-044-3*, in that the former isolate had two plants (5033-1 & 5) with an occasional anther with a small discrete fluorescent spot. Such spots were never observed in any plants of *Bf1-Mu-044-3*. The discreteness of these spots suggests that the anthranilic acid in this mutant is cell limited and not diffusible. Similar sharp boundaries between revertant nonfluorescent and mutant fluorescent sectors were found by Bender and Fink (Cell 83:725, 1995) in an analysis of the *pai* mutant of *Arabidopsis*, which accumulates anthranilate derivatives and exhibited blue fluorescence similar to *Bf1-Mu-044-3* and *Bf1-Mu-544-3*.

To determine if these two isolates retained the same anther phenotype under the different environmental conditions of a second growing season and to ascertain the frequency of homozygous mutant seedlings, if they occurred, sibling kernels to those that produced the families tested in Table 2 were sown. In the family that produced the self-pollinated ear that was the source of the plants for tests of mutant *Bf1-Mu-544-3* in Table 2, two sibling plants also had no Bf seedlings in the progenies of their self-pollinated ears. Plants from the kernels of the self-pollinated ears of these two plants were also included in these tests (Table 3). None of the tested families in Table 3 had any plants with totally fluorescent anthers. Plants 6 and 7 in family 1013 of mutant *Bf1-Mu-544-3*, however, had an occasional anther with a single small discrete fluorescent spot. The total Bf seedlings in the progenies of self-pollinated heterozygous plants of both isolates were significantly less than 25%. Although 11 out of the 31 progenies had a 3:1 ratio or a ratio close to 3:1, the numbers of Bf seedlings in most progenies were so few that the tests for significance were not very reliable. Thirteen of the progenies had significantly less than 25% Bf seedlings. Nine of these latter progenies had 2 or less Bf seedlings, including five that had no mutant seedlings. The lower than expected 25% mutant seedlings in the progenies of self-pollinated plants is not surprising because the ears chosen to provide the kernels for the plants involved in these tests did not segregate for any Bf seedlings. Although the original seedling tests that were made, by which the ears were chosen for further testing, consisted of about 50 seedlings, the results from the selfs in Table 3 suggest that the ears selected for the tests did carry some kernels that were homozygous for the *Bf-Mu* alleles.

The results from Tables 2 and 3 for *Bf1-Mu-044-3* suggest that the lack or infrequent occurrence of Bf seedlings in progenies of selfs could be the result of kernels with homozygous mutant germs that did not germinate or germinated at less than the expected frequency. If homozygous mutant kernels did germinate, the Bf seedlings they produced did not go on to produce mature plants. This latter conclusion is confirmed by the observation that

Table 3. Self and outcross tests of mutants *Bf1-Mu-044-3* and *Bf1-Mu-544-3*, which lack the Bf phenotype in the anthers but retain the seedling Bf phenotype.^a

Family and plant number	Progenies from self-pollination				Male outcross progenies			
	bf	Bf	Total	%Bf	bf	Bf	Total	%Bf
<i>Bf1-Mu-044-3</i> (Tests using sibling kernels to those that produced the population of this mutant tested in Table 2)								
1016-4	24	6	30	20.00 ^{ns}	3	32	35	91.43 ^{**}
-5	7	7	14	50.00*	12	13	25	52.00 ^{ns}
-6	21	0	21	0.00	20	25	45	55.56 ^{ns}
-8	--	1	1	-----	17	21	38	55.26 ^{ns}
1017-1	5	0	5	0.00	28	10	38	26.32 ^{**}
-3c	21	1	22	4.55(*)	23	4	27	14.81(**)
-4	24	0	24	0.00	24	17	41	41.46 ^{ns}
-5	33	4	37	10.81(*)	31	11	42	26.19 ^{**}
-6	--	--	-----	-----	42	8	50	16.00 ^{**}
-8	23	4	27	14.81(ns)	19	16	35	45.71 ^{ns}
-9	18	2	20	10.00(ns)	21	21	42	50.00 ^{ns}
TOTAL	176	25	201	12.44 ^{**}	240	178	418	42.58 ^{**}
<i>Bf1-Mu-544-3</i> (Tests using sibling kernels to those that produced the progeny of the mutant tested in Table 2).								
1010-1 ¹	29	3	32	9.38(*)	20	24	22	54.54 ^{ns}
-3 ¹	(13) 9	9	22	40.91	0	43	43	100.00
-5 ¹	22	3	25	12.00(ns)	23	23	46	50.00 ^{ns}
-8 ¹	22	3	25	12.00(ns)	22	16	48	54.17 ^{ns}
-9	--	--	-----	-----	0	36	36	100.00
1011-1d ¹	24	6	30	20.00 ^{ns}	15	21	36	58.33 ^{ns}
-2b ¹	27	4	31	12.90(ns)	16	17	33	51.52 ^{ns}
-4 ¹	0	29	29	100.00	0	37	37	100.00
-5	0	22	22	100.00	0	27	27	100.00
-6	0	8	8	100.00	0	39	39	100.00
-7	0	28	28	100.00	0	49	49	100.00
-8 ¹	21	3	24	12.59(ns)	25	20	45	44.44 ^{ns}
-9	23	4	27	14.81(ns)	22	21	43	48.84 ^{ns}
TOTAL	168	26	194	13.40 ^{**}	143	152	295	51.53 ^{ns}
(Minus 1010-3,9 & 1011-4,5,6,7)								
The kernels for the plants in the following two families were from self-pollinated ears of two sibling plants to that which provided the kernels for both families 1010 and 1011.								
1013-1 ¹	34	0	34	0.00	23	13	36	36.11 ^{ns}
-2	25	0	25	0.00	30	12	42	28.57 ^{**}
-5	40	1 ^e	41	2.44(**)	32	10	42	23.81 ^{**}
-6f	38	1	39	2.56(**)	23	26	49	53.06 ^{ns}
-7f	0	53 ^e	53	100.00	1 ^h	61	62	98.39
TOTAL	137	2	139	1.44(**)	108	61	169	36.09 ^{**}
(Minus #7)								
1014-3	36	5	41	12.20 ^{ns}	28	8	36	22.22 ^{**}
-5 ¹	13	4	17	23.53(ns)	22	11	33	33.33 ^{ns}
-6	38	3	41	7.32(**)	32	5	37	13.51 ^{**}
TOTAL	87	12	99	12.12 ^{**}	82	24	106	22.64 ^{**}

** The chi square tests for significant deviations from a 3:1 ratio in the progenies of the selfs or 1:1 ratio in the outcross progenies were significant at the 1% level.

ns Chi square tests for significant deviations from a 3:1 ratio in the progenies of the selfs or a 1:1 ratio in the progenies of the outcrosses were not significant.

(*), (***) The chi square tests for significant deviations from a 3:1 in the progenies of the selfs or a deviation from a 1:1 ratio in the outcross progenies were not dependable because one class had fewer than 5 individuals.

a All plants in this table carrying the *Bf1-Mu* alleles had normal pollen. Of the total of 17 plants involved in the tests of *Bf1-Mu-044-3*, 11 were heterozygous for the mutant allele and six were homozygous for the nonmutant allele. Of the total of 30 plants involved in the tests of *Bf1-Mu-544-3*, seven were homozygous for the mutant allele, 14 were heterozygous for the mutant allele and nine were homozygous for the nonmutant allele.

b The self-pollinated ears of these plants were semisterile. The ears of all other plants had normal seed set.

c The kernels for this family came from the same ear as those for family 1016.

d The kernels for this family came from the same ear as those for family 1010.

e These seedlings were very weakly fluorescent. These two plants had a few anthers with a single small discrete Bf spot. Most anthers, however did not show any fluorescence.

f These plants were very sickly and were difficult to classify. They were classified as possible nonmutants. In light of only Bf seedlings in the outcross progeny they probably were really weakly-fluorescent seedlings.

g This seedling is probably the result of a nonmutant contaminant pollen grain.

i The self-pollinated ears of these plants segregated for germless kernels. The ears of all other plants had only kernels with normal appearing germs.

no mature plants were produced that were homozygous for the mutant allele in either Tables 2 or 3. Alternatively, the results from Table 2 suggest that the gametes with the mutant allele were transmitted in less than the expected frequency through both the male and female. Such transmission deficiencies also would result in less than the expected frequency of mutant seedlings in the progeny of self-pollinated plants. However, in the male outcrosses of this mutant in Table 3, 50% Bf seedlings were observed in about half of the seedling tests of the outcross progenies. The remaining plants had mutant percentages that approximated those observed for the male outcross in Table 2. Interestingly, however, most of the plants with 50% Bf seedlings in the male outcrosses, which had a sufficiently large number of seedlings in the self progenies for a good test of significance, had fewer than the expected mutant seedlings. These results could be due to a reduced transmission of the mutant allele because the female gametophytes with the mutant allele are lethal, or because the homozygous mutant zygote is lethal or because homozygous mutant germs do not germinate or, if they do, germination is delayed. Lethality of the female gametophyte and failure of homozygous mutant kernels to develop are ruled out because there is no consistent evidence of reduced seed set on self-pollinated ears (see Table 3). Zygotic lethality is not likely because it would result in germless kernels which were not consistently found to be associated with the transmission of these mutant alleles (see Table 3). Thus it would seem that reduced viability of homozygous mutant germs or the delayed germination of kernels with such germs is the likely explanation for the less-than-expected Bf seedlings in these self progenies.

Data from plants derived from the original self-pollinated ear of mutant *Bf1-Mu-544-3* suggest that the allele is transmitted in expected frequencies when heterozygous plants are used in both male and female outcrosses (Table 2). This is also observed to be true in the male outcrosses of plants from sibling kernels of the same ear in Table 3 (families 1010 & 1011). However, heterozygous plants from sibling self-pollinated ears to the original one used in tests of this mutant (Table 2) exhibit a significant deficiency of mutant seedlings in male outcross progenies in the self and outcross tests of Table 3 (Families 1013 & 1014). The results from the latter two families are similar to those observed for the self and outcross tests of *Bf1-Mu-044-3*.

There is one outstanding difference between the results observed for these two mutants in these two sets of crosses (Tables 2 & 3). No homozygous *Bf1-Mu* plants are observed in the *Bf1-Mu-044-3* families, while there are nine homozygous *Bf1-Mu-544-3* plants in Tables 2 and 3. A total of 39 plants with this mutant allele were involved in the tests recorded in these two tables. Besides the nine mutant homozygotes, there were 12 plants homozygous for the nonmutant (*bfl*) allele and 18 heterozygotes. The ratio of these genotypes does not differ significantly from the 1:2:1 ratio expected if the mutant allele had normal transmission and if homozygous mutant plants were not lethal. If homozygous mutant plants were observed in these tests, why the lack of Bf seedlings in the progeny of the original self-pollinated ears and the deficiency of Bf seedlings in the progenies of self-pollinated plants derived from these original ears (Table 3)? Chance exclusion of Bf seedlings in the original seedling test of the self progenies of the three plants used in the test of this mutant seems very unlikely in light of the additional results from tests of sibling plants produced by kernels from the same ears

(Tables 2 & 3). In the original seedling tests of this mutant, the seedlings were screened with ultraviolet light when most of the seedlings were at about the three leaf stage. If the homozygous *Bf1-Mu-544-3* kernels were much slower germinating than the heterozygous or homozygous nonmutant kernels, Bf seedlings may not have emerged by the time these samples were screened, but would have perhaps emerged if the seedlings had been allowed to grow for a longer period of time before they were discarded. This also might explain the lower than expected percentages of Bf seedlings in the self progenies of all three populations of this mutant (Table 3, families 1010, 1011 - 13.40%, 1013 - 1.44% & 1014 - 12.12%). The seedlings screened in these latter tests came from the next generation of self-pollination and thus were not screened at the same time or under exactly the same conditions and perhaps had grown slightly longer before screening than the seedlings of the original self progenies, which may account for the presence of a few Bf seedlings in these latter tests (Table 3). If once the Bf seedlings emerge they are capable of developing into mature plants, the genotypes of mature homozygous nonmutant, heterozygous and homozygous mutant plants would be expected in the observed ratio.

The presence of a few Bf seedlings in the self progenies of *Bf1-Mu-044-3* (Table 3) may also be the result of slow germination of the homozygous kernels. This mutant differs, however, from *Bf1-Mu-544-3* in that homozygous *Bf1-Mu-044-3* plants are not observed. *Bf1-Mu-044-3* could be a more severe mutation, which when homozygous produces some Bf seedlings that die before the mutant plants can reach maturity.

The results from the tests of *Bf1-Mu-544-3* differ from those of *Bf1-Mu-044-3* in another way. Four *Bf1-Mu-544-3* plants (Table 2 - 5033-1 & 5, Table 3 - 1013-6 & 7) had a unique anther phenotype. In the tassels of these plants, a few anthers were observed with a single small discrete Bf spot. These spots could possibly represent sectors of cells in which the *Mutator* element has transposed within the gene to a site that allows the mutant allele to be expressed in the tissue of the anther. Alternatively, the element may have transposed from the gene leaving a footprint that altered the expression of the mutant allele in such a way that it is expressed in the anthers as well as in the seedlings.

Of the 38 *Bf1-Mu* isolates that have been involved in this study only two lacked expression of the mutant phenotype in the anthers. Such a phenotype could result if these two alleles lack fluorescence in the anthers because they are insertion mutants in which the inserted *Mu* element is susceptible to suppression (modification) resulting from methylation of the inserted element. Martienssen, et al. (Genes Dev. 4:331, 1990) and Martienssen and Baron (Genetics 126:1157, 1994) describe a pattern of modification of *Mutator*-induced mutants that occurs progressively as the plant develops. Frequently the plants carrying the mutants they studied would have tassels in which the mutant phenotype was completely suppressed (nonmutant). If this phenomenon was responsible for the phenotype of these two *Bf1-Mu* mutants, it has so far been observed to occur in 100% of the heterozygous and homozygous mutant plants tested. Although over 1000 Bf seedlings were observed in these tests, not one had any sectoring in the later developing seedling leaves that might be expected if modification was occurring. Martienssen and Baron (1994) found that once a suppressible *Mutator*-induced mutant allele is modified it is not usually reactivated unless crossed to an active *Mutator*

stock. Because the *Bf1-R* tester stocks used in the outcrosses in Tables 2 and 3 do not carry an active *MuDSR* element, no Bf seedlings would be expected in these outcrosses. However, only Bf seedlings were observed when homozygous *Bf-Mu* plants were outcrossed as males to *Bf1-R* tester plants. Likewise, the segregation of Bf seedlings when heterozygous plants were crossed to the same tester stocks is not expected if modification is occurring. These observations suggest that modification is not responsible for the unusual phenotype of the *Bf1-Mu* alleles. However, the possibility that some as yet not understood form of modification is responsible for the loss of the expression of blue fluorescence in the anthers of plants with these mutant alleles can not be eliminated as a possible explanation for their unexpected phenotypic expression.

These mutant stocks are potentially useful for the following kinds of studies: 1) Cloning the *bfl* gene. 2) Studying the regulation of a gene that is expressed as a recessive in some tissues (seedlings) and as a dominant in other tissues (anthers). 3) The possible role of modification in the unique phenotype of these alleles.

Stocks of these and the other *Bf1-Mu* mutants are available upon request.

A search for interchromosomal mitotic recombination in maize

--Peterson, T

There are very few reports of spontaneous mitotic recombination in plants (e.g. Carlson, 1974; Ashley, 1978). We have previously identified deletions of the maize *P* gene which are thought to occur via premeiotic intrachromosomal recombination between 5.2 kbp direct repeats which flank the *P-rr* gene (Athma and Peterson, 1991). Deletions between direct repeats at the *Knotted* locus have also been reported (Lowe, Mathern and Hake, 1992).

To detect interchromosomal mitotic recombination, we crossed together two *P* gene alleles which should produce twinned sectors after reciprocal mitotic recombination. The *P-wr* allele specifies colorless pericarp and red cob glumes, while the *P-oo-13:255A-10* allele specifies orange pericarp and orange cob glumes. The *P-oo-13:255A-10* allele is essentially a weak-acting *P-rr* allele due to a 6 bp (2 codon) insertion in exon 1 of *P-rr* left as a footprint following excision of the transposable element *Ac*. Plants of genotype *P-wr/P-oo* were grown in an isolation field, detasseled, and allowed to pollinate with a *P-ww* pollen donor. The progeny ears were inspected for sectors of altered kernel pericarp pigmentation. In particular, twinned sectors of red and white pericarp should be visible against the orange pericarp specified by the *P-oo* allele. Among approximately 400 progeny ears, two distinct and unambiguous red/white twinned sectors were found. These twinned sectors could have arisen by mitotic crossing over of chromosome 1 homologs in the four-strand stage between the *P* locus and the centromere, followed by appropriate segregation of chromatids to the two daughter cells. These daughter cells would produce adjacent cell clones, with one clone carrying two doses of *P-oo* (gives red pericarp) twinned with a clone carrying two doses of *P-wr* (gives white pericarp). Unfortunately, both twinned sectors are very small (approximately 1 mm in width) and unlikely to be transmitted to the egg due to their location on the abgerminal side of the kernel. However, molecular testing may be possible if sufficient DNA can be obtained from twinned sectors for PCR analysis.

The frequency of spontaneous mitotic recombination is quite low (2 visible twinned sectors from 400 ears). It would be interesting to test whether external or endogenous agents (e.g. radiation or transposable element activity) can increase the frequency to levels useful for genetic experiments such as mosaic analysis.

BARNUM, MINNESOTA
Booming Native Plants

A quick way to overview LAMP data
--Kutka, FJ

With my northerly location, on the edge of frigid Lake Superior's cooling effects, I have found maize cultivation rather challenging. "Early" inbreds rarely fully mature and the classic New England flints and Wisconsin 25 dent are not fully dependable either. To remedy this I read what I could about maize in its latitudinal extremes and requested a copy of the data from the Latin American Maize Project (LAMP). I wanted to see what there was to know about the races Northern Flint (NF) from North America and Araucano (AR) from central Chile.

Unfortunately there are only a few dozen accessions of NF left, but there are many dozens of accessions of Araucano and other central Chilean races. To deal with this incredible mountain of

data I searched the data base for US and Chilean races that silked in less than 65 days. This gave me a look at all materials that had any chance of maturing here. Then I sorted the resulting files by race and had a nice spreadsheet for NF and AR. To make sense of all of this information I plotted grain production by days to silk for each race (Figs. 1 and 2). This clearly showed the most productive accessions for each, which follow a line of increasing production from early to later silking. This quickly reduced the many dozens of accessions to a very few most promising ones. From here it is easy to compare these for other characteristics and choose the best for a first round of trials. Given that LAMP did not choose to do further work on the early maize, this is a good first pass at these materials for anyone who might be interested.

BARNUM, MINNESOTA
Booming Native Plants
BIG TIMBER, MONTANA
Painted Mountain Corn

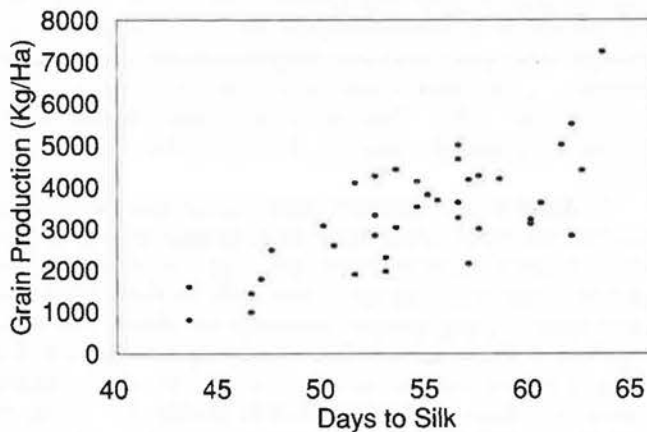
Another look at Mandan maize
--Kutka, FJ; Christensen, D

The Mandan Indians brought maize to the Great Plains region at least 350 years ago (G. F. Will, *Corn for the Northwest*, Butler Publishing, 1930). Mandan maize ("MM") spread to the Rockies and into Canada, farther from the equator than maize had been grown. European settlers depended on it until the 1930's and said it rarely failed to produce a crop (G. F. Will, 1930; D. Christensen, *Painted mountain: a new survival strain*, Seed Savers Exchange, 1989). Reports cited by G. F. Will state that MM would germinate and grow at low temperatures (5-10 F as opposed to 12-15 F for dents), growing nearly anywhere across the plains. Because it also tolerates intense summer heat it may be more versatile than highland or lowland germplasm (H. R. Lafitte and G. O. Edmeades, *Field Crops Research* 49:231-247, 1997).

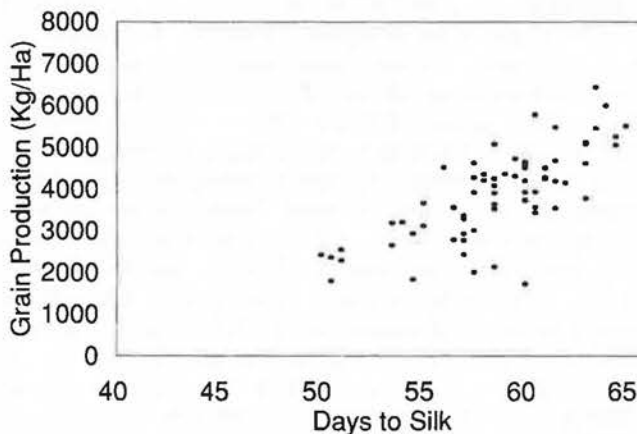
MM is shorter, with narrower stalks, lower ears and fewer roots than cornbelt dents. Ears are usually long and narrow for early dry-down to beat frosts. This form allows for early silking and a high harvest index, similar to the types in CIMMYT's drought program (G. O. Edmeades et al., in M. J. Kropff et al. (eds.), *Applications of Systems Approaches at the Field Level*, pp. 63-78, 1997). MM also tolerates drought. Normally plants grow to 1.8 m, ears are long (17-30 cm), and tillers with ears develop. In drought ears are short (10-13 cm), tillers rare, and plants short (1-1.2 m) (Will, 1930).

Over 26 years have been spent synthesizing and reselecting Mandan/Canadian Plains/Desert Highland varieties under the stress conditions of Montana to create the elite OP "Painted Mountain" (D. Christensen, 1989). "PM" silks in 43-47 days and ears range from 15-30 cm. At Big Timber, MT cornbelt dent makes ears 3 cm long whereas PM makes ears 15-20 cm long (Christensen, 1989). In an unreplicated trial in May 1997, we planted in cold soils (6 F, 5 cm deep, 20 seeds per variety). PM showed 30% emergence after four days of daytime solid temperatures above 10 F, 22 days after planting. This was equaled only by CIMMYT Pop. 900 (diverse tropical highland) and Mandan Blue flint, surpassing CMT 939011 (20%), CIMMYT Pop. 901 (10%) (both Mexican highland), and all eastern flints (0-5%) and corn belt OP's (5%) which we tried. PM, like most MM landraces, is of a hand harvest type at this point, but greatly

Northern Flint



Araucano



improved.

Mandan germplasm has also been used in breeding programs by states and provinces of the Great Plains (M. McDonald, Lethbridge, Alberta, Pers. Comm.). It represents a drought tolerant, flexible, and very productive source of earliness and cold tolerance. This germplasm deserves wider consideration; to that end Painted Mountain will be tested in Nicaragua, Peru, and Bolivia in 1998. Other stress test locations are requested.

BEIJING, CHINA

Academia Sinica, Institute of Genetics

Identification of maize inbred lines and validation of genetic relationship among maize inbred lines using RAPD markers

--Zhang, CL; Sun, ZL; Jin, DM; Sun, SM; Guo, BT; Wang, B

The objective of our study was to identify twelve maize inbred lines and validate their genetic relationship. RAPD analysis of these inbred lines' genomic DNAs was used to generate genetic fingerprintings. Two hundred and twenty arbitrary, ten-mer primers were screened to amplify all the genotypes. Thirteen of those were selected for the study because they gave reproducible, polymorphic DNA amplification patterns. The experimental results were as follows:

1. Inbred line identification: Nine primers were used to identify all the inbred lines. Four of them gave unique banding patterns for five inbred lines (Table 1). OPA08 differentiates Wenhuaug-31413 (Fig. 1), OPA10 distinguishes 488, OPC06 characterizes 8112. OPH12 is the most useful primer because it can identify Mo17 and H21 simultaneously (Fig. 2).

Table 1. Molecular markers for the identification of five maize inbred lines.

Inbred line	Primer	Primer sequence	Difference	M. W. of marker
Wenhuaug-31413	OPA08	GTGACGTAGG	absent	1.2kb
488	OPA10	GTGATCGCAG	present	0.8kb
8112	OPC06	GAACGGACTC	present	0.9kb
Mo17	OPH12	ACGCGCATGT	absent	0.7kb, 0.8kb
H21	OPH12	ACGCGCATGT	absent	0.7kb, 1.0kb

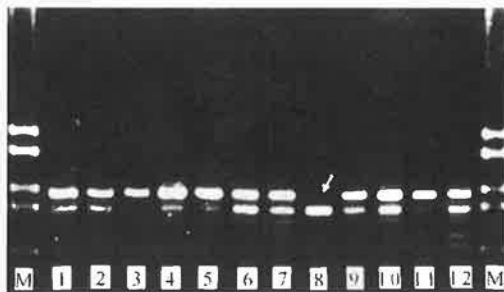


Figure 1. Amplification products generated from genomic DNAs of twelve maize inbred lines using arbitrary primer OPA08. The arrow indicates Wenhuaug-31413's unique RAPD marker. Template DNAs in different lanes were as follows: 1) 502-196; 2) Huangzao-4; 3) 478; 4) Mo17; 5) H21; 6) 107; 7) 488; 8) Wenhuaug-31413; 9) 340; 10) 515; 11) 8112; 12) 502. M represents DNA molecular weight marker VI.

Five combinations of 2-3 primers produced unique banding patterns for other inbred lines when data from each primer were analyzed in groups. OPC06/OPH12, OPF05/OPH12 and OPE12/OPH12 differentiate Huangzao-4, 515 and 340, respectively; both OPC06/OPN11 and OPA10/OPC07/OPG12 can distinguish two inbred lines, the former 512-196 and 512, the latter 478 and 107 (Table 2).

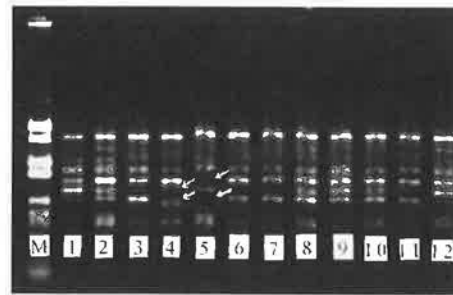


Figure 2. Amplification products generated from genomic DNAs of twelve maize inbred lines using arbitrary primer OPH12. The pair of arrows on the left indicate Mo17's unique RAPD markers and the pair of arrows on the right indicate H21's unique RAPD markers. Template DNAs in different lanes were as follows: 1) 502-196; 2) Huangzao-4; 3) 478; 4) Mo17; 5) H21; 6) 107; 7) 488; 8) Wenhuaug-31413; 9) 340; 10) 515; 11) 8112; 12) 502. M represents DNA molecular weight marker VI.

Table 2. Combinations of primers and their identified inbred lines.

Combination of primers	Inbred line	Combination of primers	Inbred line
OPC06/OPH12	Huangzao-4	OPN11/OPC06	502, 502-196
OPF05/OPH12	515	OPA10/OPC07/OPG12	478, 107
OPE12/OPH12	340		

2. Genetic relationship: Twelve primers were selected to validate the potential of RAPDs for detecting genetic variability among maize inbred lines. In order to quantify the level of polymorphism observed, Nei's estimates of similarity were used to generate a similarity matrix. All of the inbred lines fell in the range of 0.65 to 1.00 like the case in tomato and wheat. This indicates that only little polymorphism exists at their DNA level and it may be due to self-pollinating.

A dendrogram was constructed from the similarity index values (Fig. 3). Three clusters can be observed, which are further

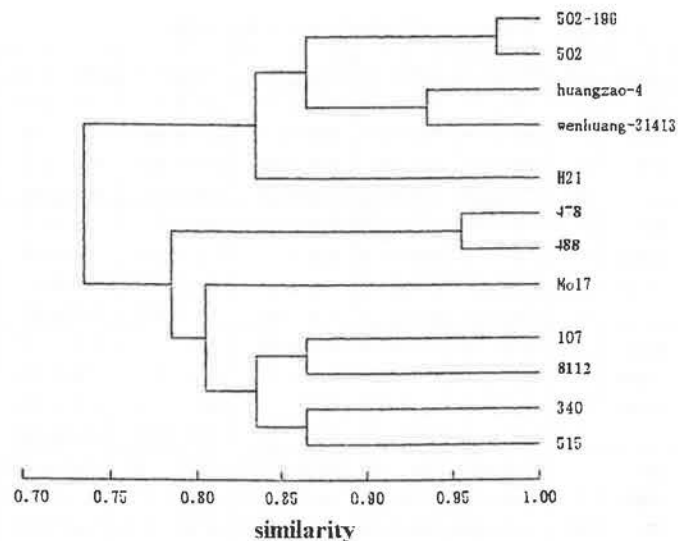


Figure 3. Dendrogram constructed from the similarity index values for different maize inbred lines with average linkage cluster analysis (UPGMA).

divided into subgroups. The first cluster consists of Huangzao-4 and four inbred lines whose parent is Huangzao-4; 502-196 and 502 form the first subgroup at 0.98 of similarity since 502-196 is an improved material of 502; Huangzao-4 and Wenhuaug-31413 form the second subgroup; H21 forms the third subgroup because its other parent is an American inbred line. The second cluster in-

cludes two inbred lines, 478 and 488, which came from an F2 plant. The five remaining inbred lines form a separate cluster for their different origin; Mo17, a well-known American inbred line, forms the first subgroup; 107 and 8112, which also came from the United States, form the second subgroup; 340 and 515 form the third subgroup, 340 is a native inbred line whose parents are Chinese local varieties and 515 has American and Chinese genealogy. This showed that genetic relationship established in the study conforms to the pedigree of the twelve maize inbred lines.

In summary, our study provides information on the molecular basis of the polymorphism detected by RAPD markers in maize. It demonstrates that RAPD assay can identify maize inbred lines and validate genetic relationship among maize inbred lines.

Breeding and genetic analysis on the multiplasmic lines of maize (*Zea mays* L.). I. Breeding and morphological observations

--Zeng, M, Liu, S, Yang, T, Liu, Y, Li, S

A group of multiplasmic lines with Mo17 nucleus, including (Fli)Mo17 (original flint cytoplasm), (su1)Mo17, (sh2)Mo17, (bt1)Mo17, (Pop)Mo17, (wx)Mo17, (Teo)Mo17, (cms-T)Mo17, (cms-S)Mo17, (cms-C)Mo17, and (cms-21A)Mo17 etc., were

developed. After crossing, backcrossing was carried out for 24 generations by Mo17 line. The degrees of homo-nucleus of Mo17 had reached 99.99999%. The group of multiplasmic lines could be distinguished by biochemical markers for specific isozymes (or soluble protein, glutamine) bands (or zymograms) (see MNL 71:5-6). The analytic experiments indicated there were almost no differences in morphological characters among these eleven lines, but they were highly different in quantitative characters. Six ear characters in all multiplasmic lines and control (check) (Fli)Mo17 are shown in Table 1. The analysis of variances has shown that the difference in row number per ear and 100grain weight between (su1)Mo17 etc. and control (Fli)Mo17 is not significant, but highly significant in ear length, grain number per row, grain weight per ear and weight per ear. In addition, agronomic characters for F1 (multiplasmic line x Huangzhao si) are shown in Table 2.

F-test demonstrates there are highly significant differences in all experimental characters of all lines, except ear diameter, and highly significant differences also in variety X environment, except ear diameter. This means that the experimental characters are markedly influenced by genetic factors and environment. The ear diameter may be influenced by genetic factors, environment and other factors. See Table 3.

Table 1. Agronomic characters for the multiplasmic line.

Sample, Item	(Fli)Mo17	(su1)Mo17	(sh2)Mo17	(bt1)Mo17	(Pop)Mo17	(wx)Mo17	(Teo)Mo17	(T)Mo17	(S)Mo17	(C)Mo17	(21A)Mo17
ear length (cm)	13.17	13.60	16.60	18.40	15.80	16.20	14.40	18.40	16.67	15.00	16.00
row number per ear	11.33	11.20	11.20	12.00	12.40	12.00	13.20	11.60	12.33	12.00	11.86
grain number per row	25.67	21.80	33.00	37.00	29.20	29.00	28.00	36.00	36.00	26.00	27.14
ear weight (g)	55.00	58.00	77.60	77.60	78.80	79.60	72.80	82.40	54.67	40.00	64.00
grain weight per ear (g)	48.00	19.20	64.00	60.80	64.40	66.80	59.60	66.80	40.00	32.00	52.57
100 grain weight (g)	21.33	25.20	24.00	19.20	24.80	25.20	24.80	22.00	15.33	14.00	21.71

Table 2. Agronomic characters for F1 (multiplasmic line x Huangzhao si).

character	plot	(Fli)Mo17 ×	(su1)Mo17 ×	(sh2)Mo17 ×	(bt1)Mo17 ×	(Pop)Mo17 ×	(wx)Mo17 ×	(Teo)Mo17 ×	(T)Mo17 ×	(S)Mo17 ×	(C)Mo17 ×	(21A)Mo17 ×
ear length (cm)	I	21.00 ± 1.83	19.75 ± 0.56	19.33 ± 1.51	20.33 ± 0.37	20.17 ± 0.90	19.25 ± 0.99	20.00 ± 1.29	19.92 ± 0.73	19.17 ± 1.34	21.00 ± 0.82	18.67 ± 0.75
	II	19.67 ± 1.37	20.67 ± 1.37	18.83 ± 1.07	18.67 ± 0.80	19.00 ± 0.82	19.50 ± 0.50	20.42 ± 0.73	19.92 ± 0.73	19.42 ± 0.93	19.02 ± 0.75	19.83 ± 1.34
	III	19.50 ± 0.76	20.17 ± 0.90	20.83 ± 0.69	20.17 ± 1.03	19.92 ± 0.73	19.00 ± 0.50	21.08 ± 0.73	20.67 ± 0.75	16.83 ± 0.90	20.92 ± 0.61	19.58 ± 0.84
	IV	19.75 ± 0.99	20.50 ± 1.26	19.50 ± 0.96	20.08 ± 0.61	20.00 ± 1.63	19.92 ± 0.45	19.50 ± 0.76	20.83 ± 0.75	19.83 ± 0.69	20.00 ± 0.82	20.33 ± 0.90
row number per ear	I	14.67 ± 0.94	15.00 ± 1.00	15.30 ± 1.49	14.00 ± 0	13.67 ± 0.82	14.33 ± 1.37	14.00 ± 1.15	14.67 ± 0.94	16.33 ± 0.75	16.00 ± 0	14.67 ± 0.94
	II	15.00 ± 1.00	15.00 ± 1.00	16.33 ± 1.37	17.33 ± 0.94	15.00 ± 1.00	14.67 ± 0.94	14.00 ± 0	14.67 ± 0.94	15.67 ± 1.37	15.20 ± 1.20	14.33 ± 1.80
	III	15.67 ± 0.75	14.67 ± 1.49	14.67 ± 0.94	15.33 ± 0.94	15.00 ± 1.53	16.00 ± 0	14.33 ± 0.75	15.67 ± 1.51	15.00 ± 1.00	14.33 ± 0.75	14.67 ± 0.94
	IV	15.67 ± 1.37	15.00 ± 1.00	16.00 ± 1.63	17.00 ± 1.53	14.67 ± 0.94	15.00 ± 1.53	15.67 ± 1.37	16.67 ± 1.49	15.67 ± 1.37	15.00 ± 1.53	12.67 ± 0.94
grain number per row	I	46.50 ± 3.04	46.17 ± 3.18	47.30 ± 4.82	46.00 ± 2.94	52.00 ± 2.77	46.17 ± 1.21	44.00 ± 3.21	48.83 ± 3.44	43.33 ± 3.04	49.17 ± 2.41	41.67 ± 1.11
	II	43.83 ± 3.67	43.33 ± 2.13	45.00 ± 2.52	45.00 ± 2.89	37.33 ± 2.56	44.33 ± 1.80	47.83 ± 1.35	44.33 ± 2.43	47.00 ± 3.74	44.50 ± 2.17	44.17 ± 3.02
	III	38.17 ± 2.41	44.00 ± 0.82	43.17 ± 2.85	47.83 ± 3.89	45.83 ± 3.29	45.00 ± 1.53	43.83 ± 2.41	39.33 ± 2.92	37.33 ± 1.97	43.83 ± 2.03	40.17 ± 3.89
	IV	38.83 ± 1.57	42.00 ± 2.89	37.83 ± 3.44	47.17 ± 1.86	44.67 ± 1.89	39.33 ± 4.96	40.00 ± 2.89	41.00 ± 2.94	38.17 ± 1.07	43.00 ± 3.74	41.67 ± 2.49
ear weight (g)	I	170.33 ± 13.91	166.17 ± 8.09	186.00 ± 16.44	191.33 ± 2.49	196.67 ± 11.76	163.33 ± 15.69	159.00 ± 15.32	201.50 ± 13.50	203.00 ± 16.51	214.00 ± 19.04	170.33 ± 11.84
	II	177.00 ± 13.24	176.00 ± 16.00	161.17 ± 11.81	182.67 ± 13.83	162.50 ± 15.10	161.83 ± 10.30	200.50 ± 8.04	181.00 ± 13.75	180.17 ± 15.53	198.20 ± 6.21	178.67 ± 14.16
	III	156.33 ± 11.38	179.83 ± 13.25	179.50 ± 14.17	187.67 ± 17.75	179.17 ± 14.25	173.67 ± 12.94	174.33 ± 13.91	190.00 ± 15.10	148.33 ± 13.02	182.3 ± 17.73	190.67 ± 16.52
	IV	171.83 ± 15.41	186.83 ± 13.04	158.83 ± 12.71	208.33 ± 8.03	181.67 ± 19.02	157.67 ± 11.69	175.00 ± 14.63	194.00 ± 14.56	182.00 ± 4.76	190.7 ± 13.40	166.05 ± 11.10
grain weight per ear (g)	I	152.83 ± 14.47	149.17 ± 9.94	169.00 ± 16.55	174.00 ± 2.24	179.67 ± 10.26	146.33 ± 16.20	140.00 ± 12.25	185.33 ± 14.07	186.17 ± 16.91	196.83 ± 19.89	152.17 ± 12.43
	II	159.33 ± 12.47	156.67 ± 17.05	145.00 ± 11.93	165.67 ± 14.79	147.67 ± 15.63	143.83 ± 9.10	183.33 ± 10.18	164.33 ± 14.34	162.33 ± 16.24	182.40 ± 5.54	161.67 ± 15.93
	III	140.00 ± 12.06	161.67 ± 10.09	163.67 ± 14.02	169.83 ± 17.98	163.33 ± 13.73	157.83 ± 12.50	158.17 ± 13.86	171.83 ± 14.89	133.00 ± 12.61	166.00 ± 17.54	173.67 ± 16.21
	IV	156.17 ± 14.77	170.67 ± 12.20	142.83 ± 12.82	192.17 ± 8.91	163.17 ± 17.92	141.50 ± 12.19	158.17 ± 14.39	177.50 ± 14.09	164.33 ± 7.32	172.50 ± 12.65	150.50 ± 11.37

Table 2 continued next page.

character	plot	(Fli)Mo17 ×	(sul)Mo17 ×	(su2) Mo17 ×	(bt1) Mo17 ×	(pop) Mo17 ×	(wx) Mo17 ×	(Teo)Mo17 ×	(T) Mo17 ×	(S) Mo17 ×	(C) Mo17 ×	(21A) Mo17 ×
100 grain weight (g)	I	22.50 ± 1.61	21.67 ± 1.49	23.50 ± 1.61	27.17 ± 1.57	25.33 ± 1.49	22.17 ± 1.77	22.33 ± 1.49	26.17 ± 1.46	26.33 ± 1.37	25.00 ± 1.41	25.00 ± 1.41
	II	24.50 ± 1.89	24.17 ± 1.86	20.00 ± 1.00	21.33 ± 1.37	26.50 ± 2.06	22.00 ± 0	27.50 ± 1.75	25.33 ± 1.60	22.33 ± 1.60	26.67 ± 1.345	26.00 ± 2.08
	III	23.30 ± 1.11	25.33 ± 1.49	26.00 ± 1.63	23.17 ± 1.07	24.00 ± 1.73	22.50 ± 1.71	25.80 ± 2.23	28.50 ± 1.89	23.83 ± 2.67	26.50 ± 1.71	29.67 ± 1.80
	IV	25.67 ± 1.11	27.33 ± 0.47	24.00 ± 2.31	24.17 ± 1.07	25.00 ± 1.91	24.33 ± 1.37	25.33 ± 1.49	26.17 ± 1.95	27.67 ± 1.47	27.00 ± 1.53	28.50 ± 0.76
ear diameter (cm)	I	4.91 ± 0.02	4.92 ± 0.02	4.94 ± 0.02	4.93 ± 0.03	4.92 ± 0.02	4.93 ± 0.04	4.91 ± 0.02	4.93 ± 0.03	5.00 ± 0	4.95 ± 0.04	4.93 ± 0.04
	II	4.93 ± 0.03	4.92 ± 0.02	4.90 ± 0.00	4.93 ± 0.03	4.95 ± 0.03	4.91 ± 0.07	4.93 ± 0.04	4.93 ± 0.04	4.93 ± 0.04	4.95 ± 0.04	4.91 ± 0.03
	III	4.95 ± 0.03	4.93 ± 0.04	4.94 ± 0.04	4.97 ± 0.04	4.93 ± 0.04	4.97 ± 0.04	4.95 ± 0.03	4.95 ± 0.04	4.93 ± 0.04	4.97 ± 0.04	4.92 ± 0.02
	IV	4.94 ± 0.04	4.97 ± 0.04	4.99 ± 0.02	4.99 ± 0.02	4.95 ± 0.04	4.93 ± 0.05	4.98 ± 0.03	5.00 ± 0	4.98 ± 0.03	5.00 ± 0	4.94 ± 0.04
height of ear site (cm)	I	86.40 ± 5.63	91.00 ± 4.66	89.50 ± 3.61	99.63 ± 5.27	104.13 ± 6.15	93.22 ± 3.94	94.00 ± 5.20	94.88 ± 2.52	89.13 ± 4.78	86.40 ± 2.56	100.29 ± 4.13
	II	99.75 ± 3.70	89.50 ± 4.64	90.88 ± 3.89	101.50 ± 3.12	95.25 ± 5.70	89.38 ± 3.50	96.00 ± 5.02	91.63 ± 4.39	98.13 ± 5.11	95.00 ± 4.50	96.75 ± 4.58
	III	90.00 ± 4.69	96.00 ± 4.03	84.50 ± 4.75	101.50 ± 2.24	85.13 ± 7.30	91.38 ± 5.07	94.75 ± 5.91	99.75 ± 3.73	91.13 ± 5.30	93.25 ± 4.15	96.75 ± 5.09
	IV	95.38 ± 2.34	99.00 ± 1.80	89.50 ± 1.94	105.38 ± 6.32	92.88 ± 6.27	99.63 ± 3.16	107.50 ± 7.53	99.00 ± 2.60	89.25 ± 3.19	100.00 ± 2.83	96.75 ± 5.56
plant height (cm)	I	214.10 ± 3.41	222.88 ± 2.37	212.88 ± 2.67	222.88 ± 3.10	222.63 ± 2.69	223.11 ± 4.07	215.63 ± 1.93	214.25 ± 3.42	224.38 ± 2.39	214.90 ± 3.09	216.00 ± 1.69
	II	227.50 ± 1.32	211.75 ± 2.59	223.75 ± 3.38	224.38 ± 3.24	204.13 ± 2.89	214.75 ± 3.31	224.63 ± 3.20	202.38 ± 3.12	225.88 ± 2.93	211.63 ± 9.00	224.25 ± 3.63
	III	215.50 ± 3.08	214.00 ± 3.46	203.50 ± 3.32	225.13 ± 2.98	205.13 ± 4.78	216.00 ± 3.67	215.25 ± 4.09	214.00 ± 3.35	214.75 ± 3.63	241.88 ± 3.95	213.25 ± 3.42
	IV	227.00 ± 1.32	223.50 ± 3.00	215.25 ± 3.11	226.63 ± 3.00	224.63 ± 3.16	225.63 ± 3.04	224.38 ± 3.57	224.50 ± 3.71	213.25 ± 3.31	225.13 ± 3.62	214.50 ± 3.20

Table 3. Analysis of variances on agronomic characters for F1 (multiplasmic line x Huangzhao si).

Breeding and genetic analysis on the multiplasmic lines of maize (Zea mays L.). II. Transmission electron microscope observations on mitochondria

--Li, J, Zeng, M, Liu, Y, Li, S

In our previous paper it was described that the multiplasmic lines were successfully bred, biochemical markers were found, and morphological characters were described. In this paper, results of electron microscope observations of mitochondria are reported.

Materials used in this experiment were yellow young shoots for 11 of multiplasmic lines. The tip of yellow young shoots was taken as sample.

The transmission electron microscope observation on mitochondria in young shoots demonstrates that there is no significant difference in the structure of mitochondrial outer membrane, inner membrane, cristae and matrix among these eleven inbreds. The difference in size between (su1) Mo17 etc. and the control (Fli) Mo17 is not significant, but significant in terms of the number of mitochondria per cell. This means that the cytoplasm affects the growth of mitochondria. F-test demonstrates there is no significant difference in size of mitochondria among these eleven inbreds, either, while the difference in number of mitochondria per cell is highly significant, which is to say that the degree of nucleo-cytoplasmic interaction in different inbreds is not the same. See Tables 1-4.

Other appearances were also observed (Plate I).

Item	var.	envir.	Repl. of internal envir.	var. x Envir.	Error	Total	
ear length	df	10	3	20	30	200	263
	ss	51.15	7.14	27.68	104.58	220.61	411.16
	ms	5.12	2.38	1.38	3.49	1.10	1.56
	F	4.65**	2.16	1.25	3.17**		
raw number per ear	df	10	3	20	30	200	263
	ss	71.13	13.23	35.59	118.81	298.24	537.00
	ms	7.11	4.41	1.78	3.96	1.49	2.04
	F	4.77**	2.96*	1.19	2.66**		
grain number per row	df	10	3	20	30	200	263
	ss	568.52	1002.05	223.55	1486.36	1903.12	5183.6
	ms	56.85	334.02	11.18	49.55	9.52	19.71
	F	5.97**	35.09**	1.17	5.20**		
ear diameter	df	10	3	20	30	200	263
	ss	0.16	0.46	1.96	0.199	22.59	25.37
	ms	0.016	0.153	0.098	0.0066	0.113	0.096
	F	0.14	1.35	0.87	0.058		
ear weight	df	10	3	20	30	200	263
	ss	19421.9	1979.75	7284.94	29882.3	86378.7	144947.6
	ms	1942.19	659.92	364.25	996.08	431.89	551.13
	F	4.50**	1.53	0.84	2.31**		
100 grain weight per ear	df	10	3	20	30	200	263
	ss	23021.1	1819.80	7861.56	27821.2	71909.3	132433.0
	ms	2302.11	606.60	393.08	927.37	359.55	503.55
	F	6.40**	1.69	1.09	2.58**		
100 grain weight	df	10	3	20	30	200	263
	ss	463.00	132.79	54.21	567.54	472.46	1690.0
	ms	46.30	44.26	2.71	18.92	2.36	6.43
	F	19.62**	18.75**	1.15	8.02**		
plant height	df	10	3	20	30	200	263
	ss	3396.4	1229.02	3771.89	16603.1	26033.6	51034.0
	ms	339.64	409.67	188.59	553.44	130.1726.02	194.05
	F	2.61**	3.15*	1.45	4.25**		
height of ear site	df	10	3	20	30	200	263
	ss	2630.81	805.90	487.02	4219.56	5203.8	13347.1
	ms	263.08	268.63	24.35	140.65	26.02	50.75
	F	10.11**	10.32**	0.94	5.41**		

Table 1. t-test in mitochondrion size (unit: μm) between (su1)Mo17 etc. and (Fli)Mo17.

	(Fli) Mo17	(sul) Mo17	(su2) Mo17	(bt1) Mo17	(Pop) Mo17	(wx) Mo17	(Teo) Mo17	(T) Mo17	(S) Mo17	(C) Mo17	(21A) Mo17
x	0.684	0.663	0.797	0.669	0.628	0.700	0.658	0.764	0.782	0.746	0.783
n	20	20	20	20	20	20	20	20	20	20	20
s ²	1.422	0.452	3.660	1.115	2.212	1.286	0.463	2.031	0.994	0.541	1.761
t		0.430	1.420	0.270	0.831	0.269	0.538	1.221	1.790	1.252	157

Table 2. F-test in mitochondrion size among 11 maize lines.

	SS	DF	MS	F
treat interval	$SS_a=28.4362$	10	$MS_a=2.8436$	$\frac{MS_a}{MS_e} = 0.2672$
treat internal	$SS_e=106.3851$	220	$MS_e=10.6385$	
total	$SS_t=131.2213$	230		

$$F_{0.05}=1.910$$

$$F_{0.01}=2.472$$

Table 3. t-test in number of mitochondrion per a cell between (su1)Mo17 etc. and (Fli)Mo17.

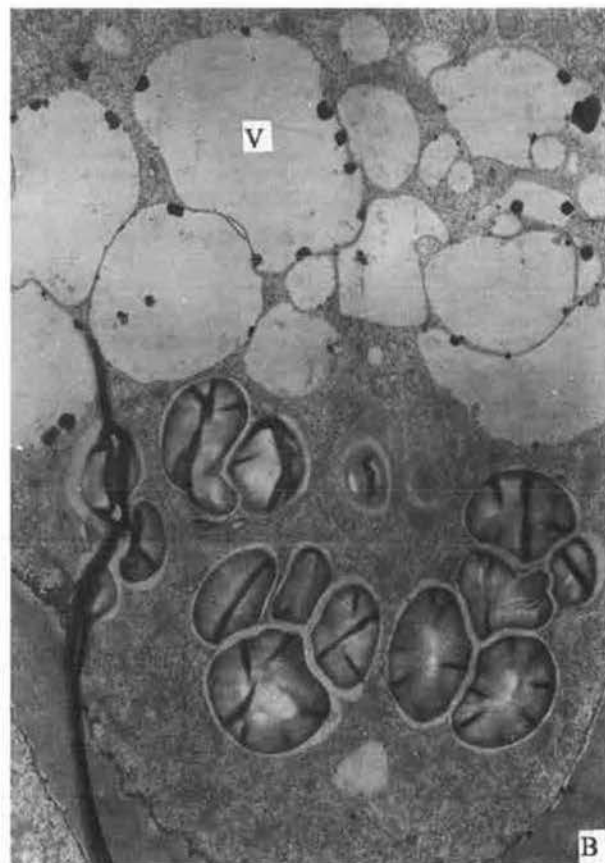
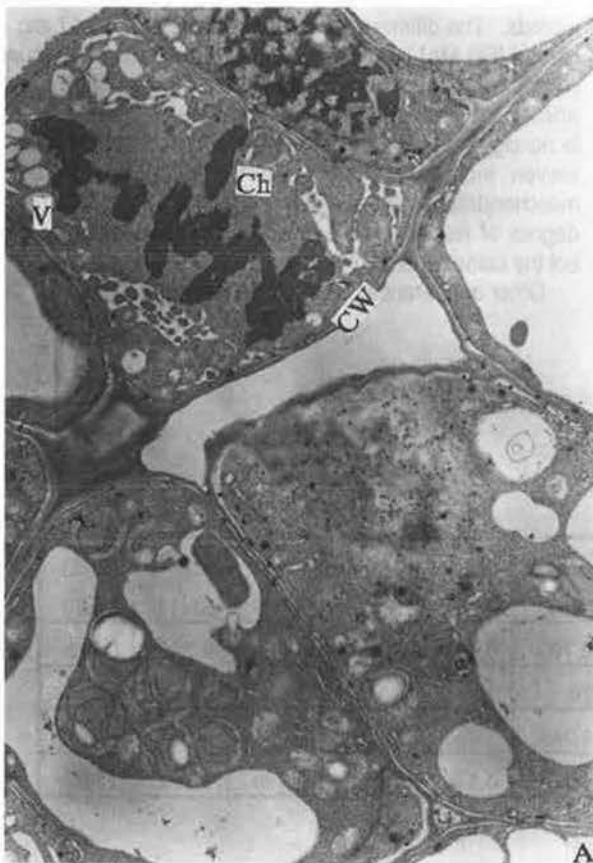
	(Fli) Mo17	(sul) Mo17	(su2) Mo17	(bt1) Mo17	(Pop) Mo17	(wx) Mo17	(Teo) Mo17	(T) Mo17	(S) Mo17	(C) Mo17	(21A) Mo17
x	22.50	21.2	30.6	24.6	44.0	20.40	37.8	33.7	26.1	23.2	40.7
n	20	20	20	20	20	20	20	20	20	20	20
s ²	1.83	3.15	12.26	4.325	31.22	3.51	20.22	16.10	8.71	2.62	27.35
t		1.949	6.824	2.828	22.809	2.932	12.928	7.427	6.022	2.015	22.615
degree of significant			**	*	**	*	**	**	**		**

Table 4. F-test in number of mitochondrion per a cell among 11 maize lines.

	SS	DF	MS	F
treat interval	$SS_a=8616.57$	10	$MS_a=2.8436$	$\frac{MS_a}{MS_e} = 50.10^{**}$
treat internal	$SS_e=1702.60$	220	$MS_e=10.6385$	
total	$SS_t=10319.77$	230		

$$F_{0.05}=1.910$$

$$F_{0.01}=2.472$$



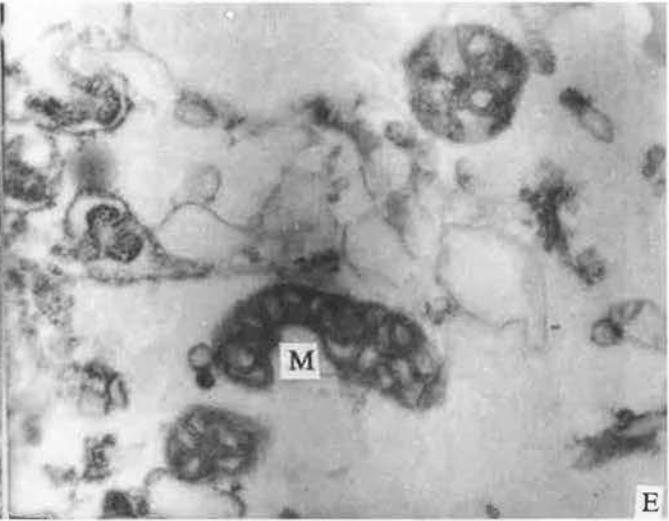
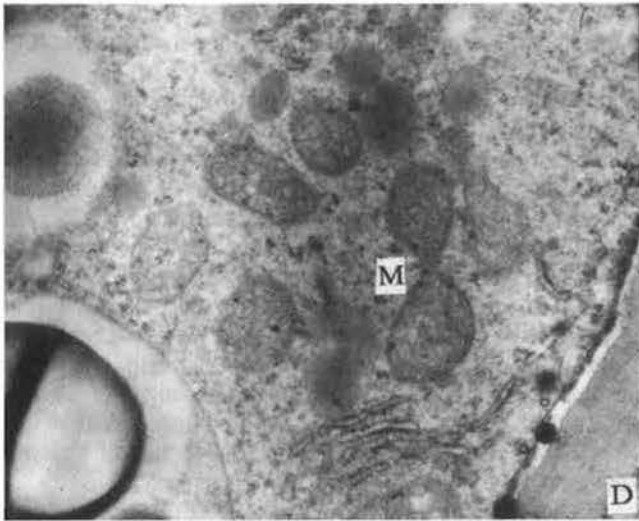
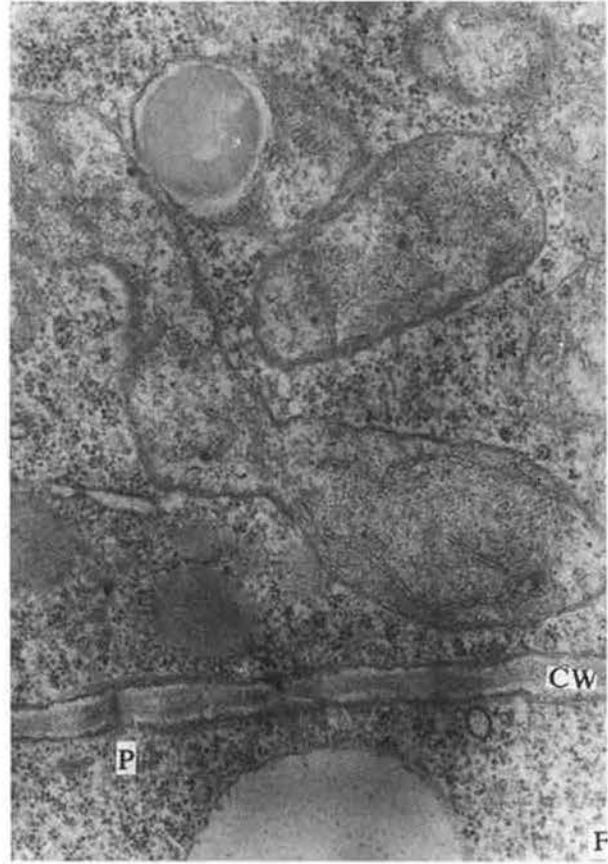
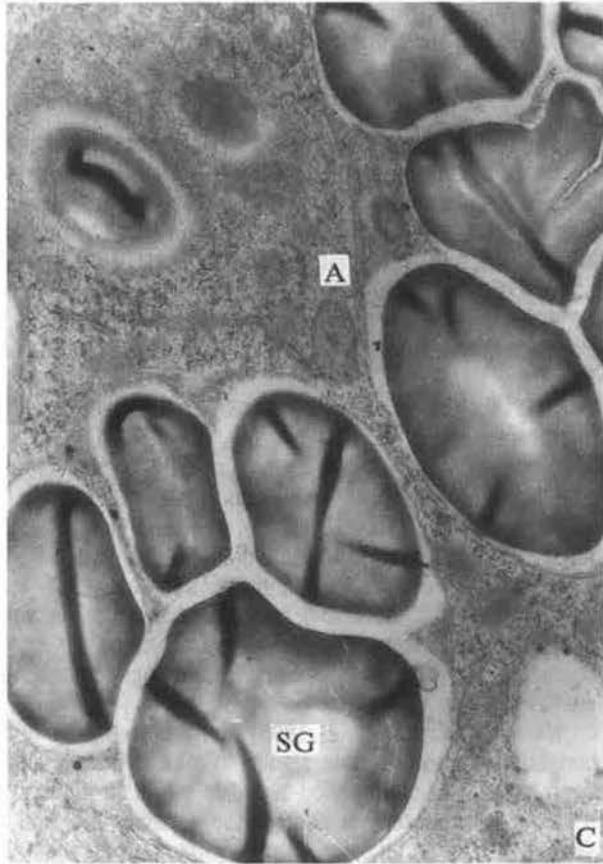


Plate I. A. A cell in mid-stage of dividing. B. Starch grains in a cell. C. Starch grains in an amyloplast. D. A mitochondrion just dividing. E. A dividing mitochondrion. F. Plasmodesmata. [Note: A-Amyloplast, CW-Cell Wall, Ch-Chromosome, M-Mitochondrion, P-Plasmodesmata, SG-Starch Grain, V-Vacuole. (X3000-25,000)]

Breeding and genetic analysis on the multiplasmic lines in maize (*Zea mays* L.). III. Electron microscope observations on starch grains in the endosperm of maize seed

--Li, J, Zeng, M, Li, S and Liu, Y

In this paper, starch grains in the endosperm of mature seed in multiplasmic maize are studied by using scanning electron

microscope. Observations on endosperm starch grains show that different cytoplasms have different effects on the characters of starch grains. The starch grains of three sweet corn cytoplasmic lines (*su1*, *sh2*, *bt1*) are mainly spherical and packed tightly, which means they have some degree of similarity, while the starch grains of four cytoplasmic male sterile inbreds (T, S, C, 21A) are mostly

irregular and packed loosely except (T)Mo17, whose starch grains are tightly packed. The average diameter of starch grains of these eleven inbreds ranges from 9.78 μ m to 14.69 μ m, the smallest starch grain is in (T)Mo17 and the largest is in (21A)Mo17, 9.78 μ m and 14.69 μ m in diameter, respectively. See Table 1 and Plate I.

BEIJING, CHINA
Chinese Academy of Agricultural Science

Discovery and primary study on photoperiod-sensitive male sterility in maize

--Zhou, H; Deng, Y; Tian, Z

Table 1. Morphological characters of endosperm starch grains in multiplasmic maize.

Item name of line	morphological character of starch grains	arranging pattern of starch grains	mean diameter (μ m)
(Fli)Mo17	polyhedral or irregularly spherical	dissimilar in size, tightly packed	9.92
(su1)Mo17	spherical, smooth	similar in size, tightly packed	10.16
(sh2)Mo17	irregularly spherical, more matrix protein	similar in size, tightly packed	10.23
(bt1)Mo17	irregularly spherical, more matrix protein	dissimilar in size, loosely packed	9.83
(Pop)Mo17	spherical, small granules attached	similar in size, tightly packed	12.78*
(wx)Mo17	spherical or ellipsoidal more matrix protein	dissimilar in size, tightly packed	12.57*
(Teo)Mo17	spherical or ellipsoidal less matrix protein	dissimilar in size tightly packed	12.60*
(T)Mo17	awl-shaped, spherical or ellipsoidal	dissimilar in size tightly packed	9.78
(S)Mo17	spherical, smooth	dissimilar in size tightly packed	12.15
(C)Mo17	spherical or irregular	dissimilar in size, tightly packed	12.35
(21A)Mo17	polyhedral, rough	similar in size, loosely packed	14.69**

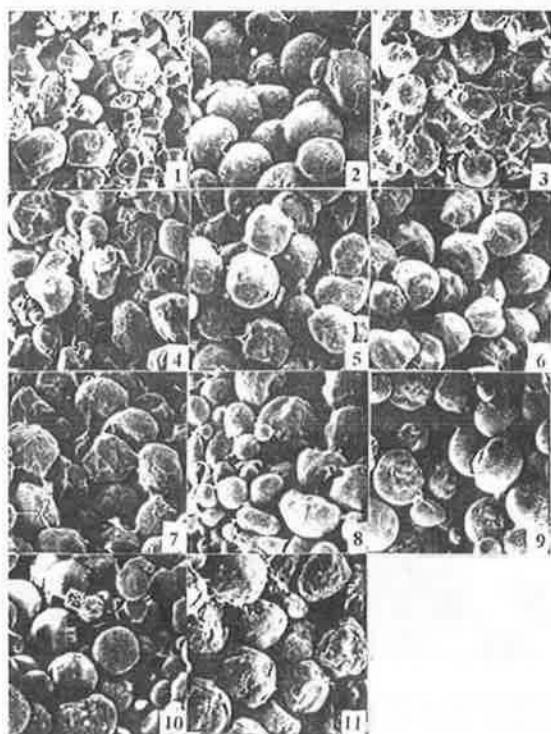


Plate I. Structure of starch grains for maize mature seed. [Note: (x1000). 1. (Fli)Mo17, 2. (su1)Mo17, 3. (sh2)Mo17, 4. (bt1)Mo17, 5. (pop)Mo17, 6. (wx)Mo17, 7. (Teo)Mo17, 8. (T)Mo17, 9. (S)Mo17, 10. (C)Mo17, 11. (21A)Mo17]

We also explored the relationship between the characters of starch grains and the property and quality of seeds and some questions associated with mitochondrion and amyloplast. All these will become a basis for the further research in multiplasmic maize.

The photoperiod-sensitive male sterility in maize first reported here was discovered by us in 1995. Over 3 years and 3 sites, we found that expression of fertility in maize line CA507 was not stable and had some correlation with the photoperiod. When planted in short-photoperiod conditions, it expressed male sterility. Otherwise it was male fertile under long-photoperiod conditions. The threshold of light length may be 14-15 hours. Preliminary study indicated that over 2 recessive genes underlaid this trait.

Since the first photoperiod-sensitive male sterility in rice was discovered by Shi (Hubei Agric. Sci. 7:1-3, 1981), many photoperiod-sensitive types of male sterility in other crops have been found one after another, such as in barley (Wang and Cheng, Hubei Agric. Sci. 11:14-16, 1991), in wheat (He et al., Hunan Agric. Sci. 5:1-3, 1992). Photoperiod-sensitive male sterility has a unique role in hybrid seed production, especially for self-pollinated crops. More and more facts support the view that photoperiod and temperature co-act in male sterility. Generally, if photoperiod acts dominantly we call it photoperiod-sensitive male sterility and vice versa.

He et al. (Crops 2:1-2, 1995) first reported their finding of temperature-sensitive male sterility in maize in 1995. We have been looking for photoperiod-sensitive maize for several years, because we believe that photoperiod sensitive material should exist in maize as in other crops. Photoperiod-sensitive male sterility should be more stable than temperature-sensitive because photoperiod compared with temperature is more steady and predictable among different years, and therefore has a great advantage in practical application.

In the winter of 1994, we found in Nanbin farm, Sanya city, Hainan province that all the 38 plants of inbred line CA507 displayed male sterility. The anthers of the inbreds didn't shed and had no normal pollen in them but a few irregular grains which could not be dyed to dark-blue color when treated with KI. All the plants displayed complete male sterility. From then on, we have conducted a series of studies on this character.

CA507, and F1s of CA507 crossed with other five normal inbreds, were planted in Beijing and in Sanya respectively in 1994 and 1995. CA507 was grown both in long and in short photoperiod conditions in Wuhan University in 1996. Three F2s and one BC1 were planted in Sanya and Beijing in 1996 for the evaluation of gene acting mode. The fertility of pollen was determined through KI dyeing under microscope.

Seeds from the same selfed ear of CA507 were grown both in Sanya (latitude 18.3 N) and in Beijing (latitude 40 N) (see details in Table 1). While all the 38 plants displayed complete male sterility in Sanya, only 10 plants were male sterile and the other 10 plants were male fertile in Beijing. In order to detect whether this fertility difference resulted from the difference of photoperiod or temperature between the two locations, we arranged another experiment in Wuhan University under precisely controlled environment (Table 2) and collected the related temperature data of the three locations during the growing season (Fig. 1, 2, 3).

Table 1. Fertility statistics of CA507 under different photoperiods in Sanya and Beijing (1994-1995).

Location	Total plants	Fertile	Sterile	Light hours/day
94Sanya	38	0	38	11-12
95Beijing	20	10	10	14.5-15.5

Two sets of CA507 seeds were grown respectively in controlled culture rooms with 11 and 17.5 light hours per day in Wuhan University in 1996. Among the 19 plants treated with 11 light hours per day, 18 plants displayed male sterility and 1 plant displayed partial male fertility in a few tassel branches. However, under the 17.5 light hours condition, all the 18 tested plants were male fertile.

Table 2. Reaction of CA507 to different photoperiod in controlled conditions in Wuhan (1996).

Light-hours	Total plants	Fertile	Partial fertile	Sterile
11	19	0	1	18
17.5	18	18	0	0

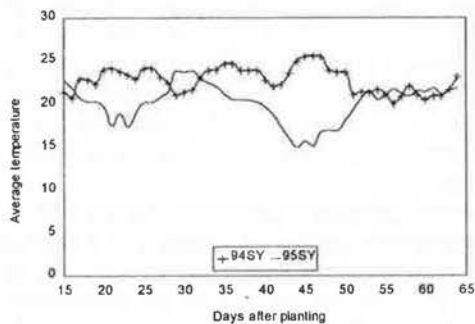


Figure 1. Comparison of temperatures between 1994 and 1995 in Sanya.

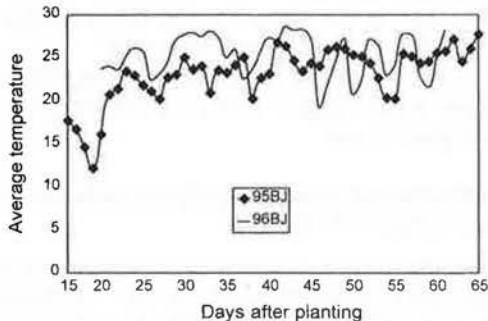


Figure 2. Comparison of temperatures between 1994 and 1995 in Beijing.

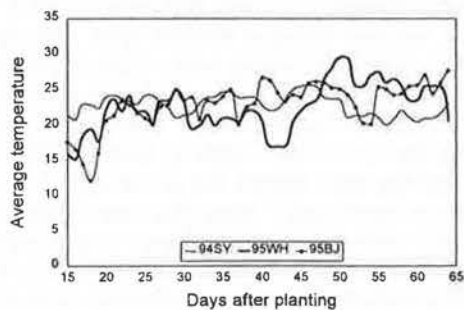


Figure 3. Comparison of temperatures among Sanya, Wuhan and Beijing.

From Figure 1, though the temperature difference between 1994 and 1995 was remarkable in Sanya during 20-25 days and 40-50 days after planting, the fertility of the test plants didn't

change much. Temperature didn't vary much between 1995 and 1996 in Beijing (Fig. 2) and between Beijing and Wuhan in 1995 (Fig. 3). From the results of our experiments in the 3 sites, we could conclude that the main factor affecting fertility was photoperiod, not temperature.

In summary, we have the following primary results: 1) CA507 displayed male sterility in short photoperiod conditions and male fertility in long photoperiod conditions, which indicated that CA507's fertility was photoperiod-sensitive; 2) the 14.5-15.5 light hours in Beijing was perhaps the threshold for fertility transition of CA507, which accounted for the coexistence of sterile and fertile type of CA507 when grown in Beijing. If we extended the light time to 17.5 hours per day, all the test plants displayed male fertility.

In Table 3, all the 304 F1 plants in the first 4 crosses showed male fertility both in Beijing and in Sanya, but 3 partial fertile plants in the 17 plants in cross CA507 x Duo4-3 in Sanya. This indicated that CA507's photoperiod-sensitivity may be controlled by recessive genes. But we used just 5 inbreds to test it; this needs further verification with precise genetic design.

Table 3. Phenotypes in F1 (1995-1996).

Crosses	Sanya			Beijing	
	TP	FP	PFP	TP	FP
CA507 x Huangzhao4	37	37	0	51	51
CA507 x 1007	38	38	0	70	70
CA507 x Zhi26-5	37	37	0	68	68
CA507 x 1006	29	29	0	46	46
CA507 x Duo4-3	17	14	3	69	69

Notes: TP=total plants, FP=fertile plants, PFP=partial fertile plants

In F2 and BC1 (see detail in Table 4), we found that all the F2 plants of CA507 x Huangzhao4 and CA507 x 1007 were male fertile both in Beijing and in Sanya. However, there were 1 male sterile plant in 20 plants of F2 (CA507 x 1006) and 1 in 17 plants of BC1 (CA507 x (CA507 x 1007)). We reckoned that over 2 pairs of genes controlled CA507's sensitivity to photoperiod. But the population was not big enough to identify the genetic mode precisely. Further study should be done on this.

Table 4. Phenotypes in F2 and backcross (1996).

Crosses	Generation	Sanya			Beijing	
		TP	FP	SP	TP	FP
(CA507 x Huangzhao4)	F2	27	27	0	38	38
(CA507 x 1007)	F2	29	29	0	27	27
(CA507 x 1006)	F2	20	19	1		
CA507 x (CA507 x 1007)	BC1	17	16	1		

Note: TP=total plants, FP=fertile plants, SP=sterile plants

Through several years study, we obtained the following primary results: 1) CA507's fertility was photoperiod-sensitive; 2) 14.5-15.5 light hours was perhaps the fertility/sterility transition period for CA507; 3) over 2 pairs of recessive genes control CA507's character of photoperiod sensitivity; 4) the genetic mechanism of this phenomenon still needs further study.

BEIJING, CHINA
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Chemical mutagenesis of *y1* mutants in corn

--Zhao, YL; Song, TM; Ma, HP

Paraffin oil with ethyl methanesulfonate (EMS) was used for

treating mature pollen of corn inbred line Huangzao 4, and an allele test of white endosperm mutants from M2 with *y1* gene stock was made to verify whether new mutants were allelic to the *y1* gene. The results are summarized in the following table.

Table 1. Mutation rates of the *y1* gene under different EMS levels.

EMS treat. levels(%)	M2 ear no.	Mutant no.	Induced mutation rate(%)
0.250	9	0	0
0.200	19	0	0
0.167	532	80	15.04
0.143	68	0	0
0.125	289	0	0
0.111	209	11	5.26
0.100	334	4	1.20
0.091	223	1	0.45
0.083	214	1	0.47
Total of EMS treat.	1896	9	5.11
Paraffin oil	238	0	0
No treat.	50	0	0
Total of non-EMS treat.	288	0	0

From 1896 EMS treated M2 ears 97 *y1* mutants were found. The average *y1* mutation rate was 5.11% under 9 different EMS treatment levels. No *y1* mutants were found among the 288 non-EMS treated M2 ears under paraffin oil and no treatment. 0.167% EMS in paraffin oil was the most effective in inducing *y1* mutants. At this concentration, 80 *y1* mutants were found among 532 M2 ears, and the induced mutation rate was 15.04%. The second was 0.111% EMS level, with induced mutation rate of 5.26%. The mutation rates of 0.100%, 0.091% and 0.083% EMS treatment were 1.20%, 0.45% and 0.47%, respectively. It was obvious that induced mutation rates of the *y1* gene greatly varied with the different EMS treatment concentrations, and mutation rates, in general, increased geometrically with the increase of EMS levels to some extent. 0.167% EMS in paraffin oil could be used as a recommendation for inducing *y1* mutants.

BERGAMO, ITALY

Istituto Sperimentale per la Cerealicoltura

Chromosome endoreduplication in endosperm cells of IHP and ILP maize strains grown in vitro

--Cavallini, A, Bosio, D, Reali, A, Natali, L*, Balconi, C, Motto, M
*Università di Pisa - Dipartimento di Biologia delle Piante
Agrarie, Sezione di Genetica

An important feature of cell differentiation in maize endosperm is nuclear enlargement through chromosome endoreduplication (Duncan and Ross, J. Heredity 41:259-268, 1950). This process may be related to many differentiation events, such as protein and starch synthesis and storage, accumulation of nucleotides, enzyme activation, and hormone synthesis (D'Amato, Caryologia 42:183-211, 1989), and consists of subsequent cycles of DNA replication without entering mitosis until high ploidy levels are attained. Variations in chromosome endoreduplication frequency in endosperm parenchyma have been described among maize populations (Kowles and Phillips, Int. Rev. Cytol. 112:97-136, 1988): in particular, different mean ploidy levels were reported in the endosperm of the strains used in these experiments, IHP (36.4C) and ILP (25.8C) (Cavallini et al., Protoplasma 189:156-162, 1995). To establish if an in vitro culture method may mimic the development of maize endosperm in vivo, we analyzed zein and starch content and chromosome

endoreduplication in IHP and ILP strains, excised at 4 or 9 days after pollination (DAP) and cultured until 25 DAP. Ears of IHP and ILP maize strains were harvested at 4 and 9 DAP and were cut into blocks containing 10 kernels per block as described by Gengenbach (Planta 134:91-93, 1977). The blocks were cultured on agar media containing salts as described in Nitsch and Nitsch (Science 163: 85-87, 1969), 150 g/l sucrose and 0 or 4 g/l glutamine. For endoreduplication analysis, after fixation in ethanol/acetic acid 3:1 (v/v), the endosperms at 25 DAP were macerated in 5% pectinase and squashed; slides were then stained by Feulgen reaction and DNA content of nuclei was measured cytophotometrically.

At 25 DAP, both IHP and ILP kernels explanted at 4 or 9 DAP and grown in vitro with or without glutamine, showed a phenotype similar to that observed in kernels grown to maturity in field conditions; in particular at 4 DAP the ILP strain showed, in both media, a reduced accumulation of the zein fraction and a higher starch content in comparison to the IHP strain. IHP showed a higher zein content (%/d.w.) in both media than ILP (4.18% vs. 1.87% without glutamine; 3.22% vs. 1.60% with glutamine in the media); on the other hand the ILP strain showed a higher starch content (%/d.w.) in both media than IHP (40.00% vs. 28.00% without glutamine; 40.50% vs. 28.43% with glutamine in the media).

For endoreduplication analysis we observed that mean ploidy level in endosperms cultured after excision at 4 DAP was similar to that found in vivo: IHP showed a higher ploidy level than ILP (34.08C (0.71 vs. 24.18C (0.54 for endosperm cultured without glutamine, 30.89C (0.92 vs. 24.97C (0.72 for those cultured in 4 g/l glutamine). Variability in chromosome endoreduplication was observed in endosperms excised at 9 DAP, probably related to the higher level of differentiation in these organs compared to those excised after 4 DAP.

In summary the results of this research suggest that the in vitro culture method tested mimics the development of maize endosperm grown in vivo.

Biochemical characterization of "high-lysine" endosperm mutants

--Bosio, D, Balconi, C, Motto, M

The storage proteins of maize are a subject of interest from the standpoints of grain quality and nutrition. Of these proteins, the zeins of the endosperm have received the greatest attention from biochemists, molecular biologists, and geneticists. These attentions are primarily due to the high proportion of zeins in the total protein complement and their deficiency in certain essential amino acids, the most limiting of which is lysine. Zein synthesis is affected by a number of genes, some of which are defined by mutant alleles. All mutants confer an opaque phenotype to the endosperm and the development of "high lysine" maize was predicted on the use of these mutations, because they inhibit zein synthesis and therefore, elevate the percentage of lysine in the grain. Some of these mutant genes have been mapped and their effect on zein synthesis described (Motto et al., Oxford Survey Plant Mol. Cell Biol. 6:87-114, 1989). Our research is devoted to a biochemical analysis of the opaque endosperm mutants, *opaque1* (*o1*), *opaque5* (*o5*), *opaque9* (*o9*), *opaque11* (*o11*), *floury1* (*fl1*) and *floury3* (*fl3*) through a characterization by SDS and IEF electrophoresis analysis of zein accumulation in the endosperm during development.

The inbred lines A69y +, A69y *o1*, A69y *o2-m(r)+Bg*, A69y

o5, *A69y o9*, *A69y o11*, *A69y fl1* and *A69y fl3* were grown at the Institute of Cereal Research, Section of Bergamo, Italy. Ear samples were harvested at 15 and 25 days after pollination (DAP), and at maturity. The seed samples harvested at 15 and 25 DAP were frozen in liquid nitrogen and the seeds stored at -20 C and an aliquot at -80 C. For the chemical analyses endosperms without embryos were used after freeze drying for 48 h.

The procedures used for analyses are described in Balconi et al. (Plant J. 3(2):325-334, 1993). Samples of endosperm, freeze-dried and ground in a mortar, were analyzed for total nitrogen (N) content on an automated N analyzer (NA-1500 Carlo Erba®). Proteins were fractionated and total protein percentage (percent N*6.4) was calculated by subtracting from the value of total N content the value of non-protein-N (NPN). Albumin plus globulin and zein fractions were analyzed by SDS gel electrophoresis. Quantification of the proportion of zein components within the total zein fraction was accomplished by scanning the SDS gels with a Personal Densitometer (Molecular Dynamics™). Data were analyzed by analysis of variance procedures and means were compared using the protected least significant difference (LSD) test, at the 0.05 probability level.

The endosperm mutants *o2-m(r)+Bg*, *o11*, and *fl3*, in comparison to wild-type endosperms, show a higher dry weight content throughout the sampling stages (mg/endosperm), and in particular at maturity. The results of total protein content (mg/endosperm) in wild-type and mutant endosperms during development and at maturity indicated that *o2-m(r)+Bg* and *fl3* mutant endosperms showed a lower endosperm total protein content, with respect to the wild-type endosperms, and followed the same trend as observed for the dry weight accumulation.

The values of protein fractions as a percentage of total protein, in wild-type and mutant endosperms, showed that for all genotypes the zein fraction increases during development, whereas, albumin plus globulin and glutelin fractions decrease. For all genotypes at all developmental stages, SDS-PAGE analysis of alcohol-soluble proteins showed the presence of polypeptides with a molecular weight corresponding to all known alcohol-soluble polypeptides that are commonly referred to as zeins.

Zein partitioning into zein families, derived by densitometer tracings of the SDS-PAGE showed, as expected, that the *o2* mutation causes a suppression of a specific zein family, the 22 kDa zein component; in addition at 15 DAP the 25 kDa zein component was very abundant in comparison with the wild-type. On the other hand, in the *o5* endosperm mutant, at both developmental stages, a reduced 28 kDa zein component was observed. In all genotypes the 20 kDa zein component (which can be resolved into two bands) was the most abundant. For the other mutants, no specific effect on the accumulation of zein components was observed.

Interestingly, in addition to the *o2-m(r)+Bg*, the SDS-PAGE analysis of albumin plus globulin protein fraction of wild-type and mutant at the various sampling stages showed the absence of the *b32* protein in *fl3* mutant at maturity stage. In the *fl3* mutant the absence, at all stages of development, of a protein band with molecular weight of 14 kDa, was also noted, which was present in the wild-type and other endosperm mutants at 25 DAP and at maturity. Studies are in progress to analyze in more detail these and other protein bands with different expression in wild-type and mutant endosperm kernels.

Identification of a putative histone deacetylase RPD3-like gene from maize that complements a yeast *rpd3*-null mutant

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One important mechanism for the dynamic alteration of chromatin structure is the acetylation and deacetylation of histones, which is affected by two enzyme activities, histone acetyltransferase (HAT) and histone deacetylase (HD). Biochemical studies have revealed a correlation between the level of histone acetylation and deacetylation with transcriptional activity and repression respectively. It is thought that the acetylation of nucleosomal histones induces an open chromatin conformation, which allows the transcription machinery access to promoters. Particularly, the central role of the RPD3 yeast-homologous histone deacetylase in many transcriptional systems was described in a wide variety of fungi and animals. These data provide evidence that histone acetylation and deacetylation are fundamental regulatory mechanisms governing cell proliferation and differentiation (for reviews see Wade and Wolffe, Curr. Biol. 7:82-84, 1997; Wolffe, Nature 387:16-17, 1997). In the germinating embryo of *Zea mays* three HATs and four HDs have been identified (Lechner et al., Biochim. Biophys. Acta 1296:181-188, 1997); in addition, a cDNA encoding for a HD2 chromatin-bound deacetylase with no RPD3 homology was described (Lusser et al., Science 277:88-91 1997).

We have recently reported the isolation and characterisation of a maize cDNA encoding a RPD3-homologous histone deacetylase (*zmRPD3*), the first to be cloned and analysed from a plant. This cDNA was identified, for the ability to weakly restore the His⁺ phenotype in a *gcn4 - bas1* yeast strain screened with a maize 10 DAP endosperm cDNA library. β galactosidase assays were performed showing that the *zmRPD3* can transactivate the yeast *HIS4* promoter in a yeast expression vector. Database analysis of this 2047 bp insert revealed an open translational reading frame of 1539 bases, extending from the first ATG located at nucleotide position 141 until a TAG stop codon at position 1679. The cDNA encodes a protein of 513 amino acid residues with a predicted molecular weight of 57.6 kDa. Analysis of the deduced amino acid sequence revealed that the protein is very similar to the yeast RPD3 protein (55.4% identity; 74.3% similarity) and other related sequences identified in a variety of organisms.

Because the amino acid sequence homology of the maize RPD3-encoded protein suggests that this protein belongs to the RPD3-family, we have examined whether the maize gene is functionally homologous to the yeast gene by expressing the maize RPD3-like protein in a yeast strain carrying a mutation in the *RPD3* gene. The restoration of resistance at sublethal doses of cycloheximide has been observed. Moreover, a *zmRPD3* transformed *rpd3 - trk1* yeast strain failed to grow in a low salt medium as expected for a RPD3 - *trk1* phenotype (Vidal and Gaber, Mol. Cell. Biol. 11:6317-6327, 1991). Hence, *zmRPD3* can functionally complement a null *rpd3* yeast mutation for at least two different phenotypic traits.

Analyses of the expression of the *zmRPD3* transcript showed that the gene probe hybridised with the mRNA extracted from leaf, coleoptile, root, and endosperm maize tissues. The results of genomic Southern analyses revealed a moderately complex hybridisation pattern including both strongly and more weakly hybridising bands. This suggests the presence of more than one copy of the *zmRPD3* sequence and/or other closely related sequences in the maize genome. These copies may possess less

sequence homology at the N-terminus than at the C-terminal region. These data are in agreement with earlier results indicating the presence in maize of at least four different proteins having histone deacetylase activity (Lechner et al., *Biochim. Biophys. Acta* 1296:181-188, 1997). The 5'-terminal probe was employed in Southern blotting experiments using a segregating population from the cross B73xA7. Analysis of polymorphic DNA fragments obtained with *Bam*HI and scoring in 107 F3 lines enabled us to localise one copy of the maize *RPD3*-like gene to the short arm of chromosome 5 near the centromeric region between the *umc1* and *bn15.71* molecular markers, with relative genetic distances of 21.1 and 14.1 cM, respectively.

Phylogenetic analysis, using *RPD3* homologous nucleotide sequences present in sequence databanks, clustered the *RPD3*-like mammalian sequences *HDAC1* and *HDAC2* (Taunton et al., *Science* 272:408-411, 1996; Yang et al., *Proc. Natl. Acad. Sci. USA* 93:12845-12850) in two evolutionarily different groups. The *RPD3*-like sequence of *Zea mays* as well as the *RPD3*-homologous sequences of other species, clustered in the *HDAC1* group. The only exception was the *RPD3* like sequence of *X. laevis* that cannot be included in any of these two groups. Analysis of evolutionary rate using relative rate tests suggest that the two groups likely evolved at different rates. The relative rate results reflect the distribution of *RPD3*-like sequences into two clusters as pointed out by Neighbour-joining cluster analysis. However, the *HD* sequence of *D. melanogaster*, included in *HDAC1* group, showed a higher evolutionary rate. Molecular and phylogenetic data gathered in this work support the hypothesis that polymorphic forms of *RPD3*-homologous sequences with significant evolutionary differences are present in maize.

Comparative analysis of genetic similarity among maize inbred lines detected by RFLPs, RAPDs, SSRs, and AFLPs

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Information about germplasm diversity and the relationships among elite breeding materials has a significant impact in the improvement of crop plants. DNA-based fingerprinting technologies have been proven useful in genetic similarity studies. Among them, RFLP was the first and is still the most commonly used in the estimation of genetic diversity in plant species. The recently developed PCR-based marker techniques, which include RAPDs, SSRs and AFLPs, are playing an increasingly important role in this type of investigation. Here we report a direct comparison of DNA based techniques in reference to their informativeness and applicability for genetic diversity study using

a set of 33 maize inbred lines.

The inbreds were surveyed for polymorphism with the four different marker systems. All of the molecular markers used in this study were able to uniquely fingerprint each of the inbred lines. The total number of assays ranged from only 6 primer combinations for AFLPs to 94 probe/enzyme combinations for RFLPs. The total number of polymorphic bands identified ranged from 90 for RAPDs to 255 for RFLPs. An average number of 4.8 alleles per locus with an average effective number of 3.2 alleles per locus ranging from 1.2 to 6.5 could be distinguished for each probe/enzyme combination using RFLPs. This value increased to 6.8 with SSRs, with an average number of effective alleles of 4.4 per locus, ranging from 1.1 to 6.6, while for RAPDs and AFLPs these values were lower (1.6 for both). This was reflected also in lower expected heterozygosity values. Overall the highest assay efficiency index was observed for AFLPs (45.7) and the lowest for RFLPs (3.2). RAPDs and SSRs (5.8 and 4.2, respectively) were comparable to RFLPs. In particular, for AFLPs the high assay efficiency index is due to the simultaneous detection of several polymorphic bands in a multiplex amplification per single reaction.

The genetic similarity trees produced from each marker system showed that inbreds were ordered in the four trees, as expected, into major groups of lines derived from BSSS and LSC, although discrepancies in forming subgroups within the major groups were observed as well as in the clustering of inbred lines of miscellaneous origins. Considering the BSSS-related lines the topology of each tree is unique with some evident similarity: the clustering of B14, B37 and B73 types is in general fully conserved across the four trees. On the LSC side cluster of C103 and Mo17 related lines were consistently reported for all methods with the exception of Va22, derived from C103, that for all methods was aggregated with lines of different origins. The Oh43 related lines (Oh43 and A619) were grouped with Lancaster only by RAPDs and AFLPs, while SSRs and RFLPs clustered these with BSSS lines; Oh43 is usually considered a Lancaster type. It is also interesting to note that similarly to the RAPD-based trees, the clustering based on AFLP data produced a tree with a relatively narrow range of similarity values between the more related and the more distant pairs of inbreds. However, all the main clusters within the set of inbreds herein studied were confirmed by clustering based on AFLP data. Four pairs of most similar inbreds (Lo932, Lo944, B14A, CM109, Lo916, Lo999, H55, H96) were clustered together as in the SSR tree, and in two cases (A619, Oh43, and H55, H96) as in RFLP and RAPD trees.

The estimates of correlation coefficients (*r*'s) among available coancestry coefficients (*f*'s) and genetic similarity (GS) data obtained from the four molecular marker systems showed that all *r*'s were highly significant ($P < 0.01$). It is worth noting that RAPDs showed the lowest correlation ($r = 0.40$) with *f* values, while AFLPs showed the highest value ($r = 0.62$). The *r*'s among similarity data obtained with the different molecular marker techniques were also significant. Correlation coefficients of RAPD marker data with those obtained using other marker systems were lower than those among similarity estimates based on AFLPs, RFLPs, and SSRs. The extent to which similarity values were correlated varied considerably across the whole data set. When the whole set of pairwise data (528) was divided in two groups (according to the arithmetic mean of the observed GS range based on RFLP data), "more similar" lines ($GS > 0.37$) and

"less similar" lines ($GS < 0.37$), it became apparent that genetic similarity estimated by different marker systems was mainly correlated only among similar lines, while the relationships among dissimilar lines were low and not significant. The GS values plotted against the estimate of coancestry value based on pedigree data followed the same pattern.

The cophenetic correlation coefficients provided for each marker system indicate the extent to which the clustering of genotypes depicted in the trees accurately represents the estimates of genetic similarity between inbreds obtained with that marker system. Overall the cophenetic coefficients were medium to high, with the RFLP (0.84), and AFLP (0.83) data resulting in the highest correlations and the RAPD (0.72) assay producing the lowest correlation.

All methods could clearly distinguish all 33 inbred lines, although the SSR data provided the highest level of discrimination between any pair of inbreds. In general, the grouping agreed with pedigree information of the lines, although some discrepancies were observed. In particular genetic similarities based on AFLP data had the highest correlation with pedigree data while those based on RAPDs had the lowest one.

In order to determine the sampling variance of genetic similarities calculated from different molecular marker data sets, bootstrap analysis with declining number of bands was performed. The relationships between number of bands and sampling variance of genetic similarity among all pairs of inbred lines for the four molecular techniques indicated that the standard deviation of the estimate was no longer significantly reduced when more than 150 bands were analysed because of a decreasing slope of the curve.

In conclusion the results of this study within a set of maize inbred lines, and the comparison between the methods employed, indicated that, with the exception of RAPDs, the other DNA markers provide consistent information for germplasm identification and pedigree validation. We have shown that SSR and AFLP profiling technologies can be good candidates to replace RFLP markers in genetic similarity estimates and variety description, and that they have comparable accuracy in grouping inbred lines selected by pedigree. They are generally much simpler to apply and more sensitive than the traditional morphological and biochemical methods or the RFLP-based fingerprinting techniques because they are more efficient in detecting polymorphism; yet they are generally correlated with RFLP analysis. A major advantage of the SSR and AFLP methods is that they can be automated and so have great potential in large-scale population genetics and plant breeding. While SSRs, thanks to their multiallelism and codominance, appear to be especially suited for the analysis of outcrossing heterozygous individuals, AFLPs, thanks to their high multiplex ratio, offer a distinctive advantage when genome coverage is a major issue due to the presence of linkage disequilibrium such as in inbred lines and breeding materials.

Genetic diversity and its relationship to hybrid performance in maize as revealed by RFLP and AFLP markers

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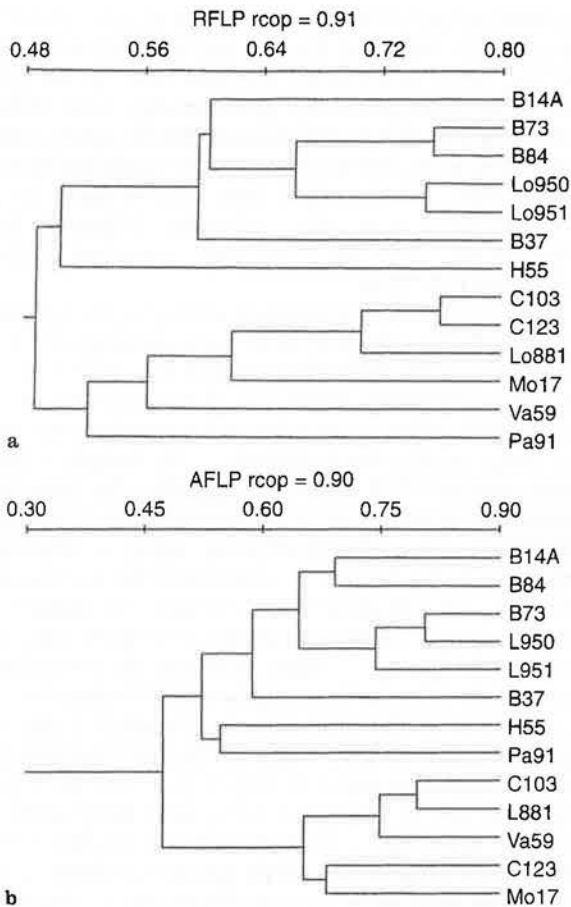
In maize, the prediction of hybrid performance is of considerable importance and has attracted large interest over the years. Recently, genetic linkage maps based on molecular markers

have been constructed in this crop (Coe et al., MNL 69:191-267, 1995), with the hope that they will provide effective means for predicting hybrid performance and heterosis. In the present study we surveyed genetic divergence among 13 inbred lines of maize using DNA markers and assessed the relationship between genetic distance and hybrid performance in a diallel set of crosses between them. The parental lines were assayed for DNA polymorphism using 135 restriction fragment length polymorphisms (RFLPs) and 209 amplified fragment polymorphisms (AFLPs).

A total of 508 RFLP bands were detected when considering 13 inbreds tested with all probe-enzyme combinations. Of the 149 probe-enzyme combinations used in this study, 135 (91%) revealed polymorphism across the 13 inbreds assayed. The majority (72%) of the polymorphic probe-enzyme combinations gave single-banded RFLP patterns. The remaining yielded multiple-banded RFLP patterns, suggesting the presence of repeated binding sequences in the genome for the corresponding DNA clones. The number of RFLP variants per probe-enzyme combination ranged from 2 to 5 in the former case and from 2 to 9 in the latter case, with an average of 3.31 and 4.97, respectively. For AFLP analysis, a total of six primer combinations was used to assay the 13 inbreds. These permitted the production of approximately 500 selectively amplified DNA fragments ranging in size from 60 to 600 nucleotides and the identification of 209 polymorphic markers. On average 30-120 distinguishable bands were observed after selective amplification with each primer combination, and an average of 34.8 of these AFLP bands were found to be polymorphic among lines with a range from 19 to 52. These results suggest that AFLPs are able to detect a larger number of polymorphisms in a more efficient way in comparison to RFLPs, due to the much higher number of loci assayed in a single multiplex PCR reaction.

Estimate of genetic distances (GDs) between lines from BSSS, LSC and miscellaneous heterotic groups calculated with RFLP and AFLP markers gave almost identical mean GD values (45.9 versus 46.2). Moreover, GDs based on AFLP data had a similar range (19.5 - 63.7) to the range of GDs calculated from RFLP data (24.1 - 59.9). For both molecular markers the subset means for GDs, were, as expected, significantly greater for combinations of lines of different origin (51.8 and 50.7) than for the BSSS x BSSS (34.7 and 36.4) and LSC x LSC (31.1 and 37.2).

The dendrograms from UPGMA cluster analyses (Rohlf, New York Exeter Publ., 1989) of GDs based on RFLP and AFLP data are presented in Figure 1. Both have a high cophenetic coefficient ($r = 0.91$ and 0.90 respectively for RFLPs and AFLPs) and therefore show a good fit with GD values. Clustering based on RFLP data resulted in two major groups (Fig. 1a). One main cluster was comprised of lines derived from or related to BSSS along with H55, while the other was composed of 5 LSC related inbreds along with Pa91. The AFLP-based dendrogram assigned the 13 inbreds to three major groups (Fig. 1b): i) the BSSS-related lines; ii) H55 along with Pa91; and iii) the Lancaster lines. In addition, when compared to the RFLP-based dendrogram, discrepancies in forming subgroups within the major groups were noted. Thus, in the dendrograms obtained from cluster analysis, all lines with defined affiliation to one of the heterotic groups were assigned to the respective main clusters. These results suggested that RFLP and AFLP data clearly separated lines from



the BSSS and LSC heterotic groups and detected pedigree relationship among inbreds.

A particular use of genetic markers is the prediction of hybrid performance. These results are consistent with experimental results of previous studies and quantitative genetic expectations.

The estimates of simple correlations (r) of GDs with F1 performance for grain yield (F1P) and SGD with SCA effects are presented in Table 1. The correlation coefficients of GDs

Table 1. Simple correlations of genetic distance (GD) and specific genetic distance (SGD) based on RFLP and AFLP data respectively, with F1 performance (F1P) and specific combining ability (SCA) of grain yield for all crosses and in different subsets of maize crosses.

Variables	Crosses(n)			
	All (78)	BSSS x BSSS (15)	LSC x LSC (10)	Unrelated lines (53)
<i>F1P</i>				
GD-RFLP	0.36**	0.31	0.28	-0.08
GD-AFLP	0.51**	0.47	0.30	0.23
<i>SCA</i>				
SGD-RFLP	0.65**	0.72**	0.27	0.38**
SGD-AFLP	0.72**	0.81**	0.66*	0.47**

*, ** Significant at 0.05 and 0.01 probability levels, respectively

calculated for RFLP and AFLP data with grain yields for the entire set of 78 hybrids were highly significant ($P < 0.01$) but only of moderate size. The r value was 0.36 for the GD based on RFLPs and 0.51 for the GD based on AFLPs. By contrast, for both classes of molecular markers a lack of relationship was noted between these two variables in the three subsets of crosses. It must be emphasized however, that the results obtained from the BSSS x BSSS and LSC x LSC group of crosses should be interpreted with caution because they are based on a small number of crosses. Estimates of r values between specific genetic distances (SGDs) and specific combining ability (SCA) effects were, with both class of markers, for all crosses and individual subsets of crosses positive and in general significant. In particular, a high correlation between the two variables was obtained for the entire set of 78 crosses (0.65 and 0.72), and in the BSSS x BSSS subset for both classes of molecular markers (0.72 and 0.81), whereas in the LSC x LSC subset a relatively high correlation (0.66) was reported only for AFLPs. In addition, significant correlations, although of moderate size, were found also in the subset of unrelated lines (0.38 and 0.47) for RFLP and AFLP, respectively. Finally, it was worth noting that correlation between GD and SGD calculated from AFLP data with F1P and SCA effects were higher than those based on RFLPs.

In summary, results from this study suggest that molecular marker based analysis, and in particular AFLP technology, offers a reliable and effective means of assessing genetic variation and of studying relationships among currently and historically important maize inbred lines. This may provide an alternative way for predicting performance and heterosis of maize hybrids. In particular, correlations between AFLP markers and SCA estimates may have a practical utility in predicting hybrid performance.

Maize transformation with *Agrobacterium tumefaciens*

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The application of *Agrobacterium*-mediated transformation to monocotyledonous species, such as rice and maize, has been recently reported. The main characteristics of the *Agrobacterium* system in these species, as for dicotyledonous species, are: i) high frequency of transformation; ii) proper integration of the foreign gene into the host genome; iii) low copy number of the gene inserted, resulting in most cases in a correct expression of the transgene itself. A limitation of the system is represented by the strict interaction between the genotype of the plant and the *Agrobacterium* strain, and the need to identify and to supplement specific signal molecules for the *vir* genes induction during the co-cultivation period (acetosyringone or sinapinic acid).

Although not being natural hosts for *Agrobacterium tumefaciens*, monocotyledonous species seem to be in some instances susceptible to the infection. Studies on *Agrobacterium* infection of maize were first reported by Grimsley et al. (Nature 325:177-179,1987) and Gould et al. (Plant Physiol. 95:426-434,1991), but the first evidences of the possibility of application of *Agrobacterium*-mediated transformation of cereal species comes from the work of Chan (Chan et al., Plant Cell Physiol. 33:577-583,1992) and Hiei (Hiei et al., Plant J. 6:271-282, 1994), who first obtained transgenic rice plants by means of

transformation of immature embryos with *Agrobacterium tumefaciens*. Most recently, the technique has been successfully applied to maize, and transgenic maize plants obtained at high frequency (Ishida et al., Nature Biotechnol. 14:745-750,1996). Ishida and co-workers report on the efficient transformation of maize inbred A188, and of some crosses between A188 and other inbreds.

Due to the ease of handling the *Agrobacterium*-mediated transformation, it would be advantageous to identify key parameters which allow the methodology to be applied to agronomically relevant genotypes, such as important inbred lines utilized in programmes of maize breeding. The present work was developed with the aim to extend the methodology of transformation with *Agrobacterium tumefaciens* to maize genotypes of relevant agronomical importance and, within these genotypes, to identify key parameters needed for transformation, and subsequent tissue culture and regeneration of transgenic plants.

To this purpose, eleven Lo inbred lines of maize, bred by our Institute, have been considered for *Agrobacterium*-mediated transformation. Among these, four were previously tested in vitro for tissue culture response and plant regeneration: Lo1054, Lo1056, Lo951 and Lo881 (Locatelli et al., MNL.66:17, 1992). Results obtained were compared with two reference genotypes: A188 and B73.

Five *Agrobacterium* strains, characterized by different chromosomal backgrounds and Ti-plasmid content, were chosen, all carrying in a binary system the chimeric construct -INTGUS-driven by the CaMV 35S promoter, for early detection of transformation events with the histochemical staining of the tissues for B-glucuronidase expression. The *Agrobacterium* strains, indicated as: C58c1(pGUSINT), Agt121(pGUSINT), EHA101(pMTCA23GUSINT), EHA105(pGUSINT), EHA105(pMT1) and LBA4404(pTOK233) were previously described in detail (Chan et al., Plant Cell Physiol. 33:577, 1992; Smith, RH and Hood, EE, Crop Sci. 35:301, 1995; Hiei et al., Plant J. 6: 271-282, 1994).

Maize plants were grown in the field during summer seasons 1996 and 1997, hand pollinated, and immature embryos dissected at 12 DAP, explanted on N6I medium (Lupotto, E, Lusardi, MC, Maydica 33:163, 1988). For transformation, immature embryos and primary embryogenic calli were co-cultivated with *Agrobacterium*. The procedure adopted followed the protocol described by Hiei et al. (1994). For the infection, bacteria were grown in AB medium supplemented with each strain's proper antibiotic selection, for three days. The bacteria were collected and diluted in low pH (pH 5.2) infection medium (LSinf) at high density (O.D. 1-1.2) in the presence of 100 uM AS. Explanted embryos were immersed in LSinf, vortexed at maximal speed 20 seconds, and incubated 10 minutes. Blotted dry embryos were subsequently co-cultivated for three days on LSD1.5, 100uM AS medium, transferred on LSD1.5 plus 250 mg/l cefotaxime (or 200 mg/l timentin) for 2 days, and tested for infection with histochemical GUS assay, or cultured for embryogenic callus induction.

For the experiments of timing, in the case of infection of A188 with *Agrobacterium* strains 2 and 6, embryos were explanted at 11, 12, 13, 14, and 15 DAP. Callus induction frequency was measured as % embryos giving an embryogenic callus. The extent of infection was rated from 0 (not infected) to 5 (heavily

infected). The frequency of transformation was recorded as % embryos exhibiting dark blue areas.

Preliminary experiments showed that a microbombardment performed with the particle gun device, improved the experiments in creating microwounding in the scutellum, thus enhancing the extent of infected tissues. In the case of pre-bombardment, 25 embryos were arranged with the scutellum exposed on the surface of culture medium and bombarded at 900 PSI, at a distance of 5 cm from the stopping screen, in 26 in Hg vacuum, with 1 um diameter gold particles, according to the customer instructions for PDS 1000/He device.

The efficiency of transformation was evaluated for each genotype, considering the interaction maize X *Agrobacterium* strain, the medium used for infection and co-cultivation, the use of elicitors (sinapinic acid and acetosyringone), and the age of the immature embryos. Each inbred line considered was also evaluated for tissue culture response, in order to choose the best combination *Agrobacterium* strain X responding inbred for routine transformation procedures.

The results obtained showed that *Agrobacterium* strains C58c1(pGUSINT) and EHA101(pMTCA23GUSINT) were most effective in the attachment to the maize genotypes A188, Lo 1095, Lo1061, Lo951 and Lo1056 (rated 4-5); effective, though at a lesser extent, were genotypes Lo1054, Lo881 and B73 (rated 3). No infection was detected in the cases of genotypes Lo1074, which was in any case not attached by any of the tested strains, as well as Lo1023. The *Agrobacterium* strain LBA4404(pTOK233) was effective on A188, B73 and Lo1061. No attachment in any of the genotypes tested was detected for strains EHA105(pGUSINT) and EHA105(pMT1). The supplement of 100 uM acetosyringone in the LSinf and LSD1.5 co-cultivation medium was beneficial for *Agrobacterium* attachment.

Considering the inbred A188 as reference genotype, and *Agrobacterium* strains C58c1(pGUSINT) and EHA101(pMTCA23GUSINT), an experiment of timing for infection was performed with the aim to identify the proper stage of embryo development for maximal attachment. Embryos were explanted every 24 hours from day 11 to day 15 after pollination, days 11 and 12 being the optimal stage for tissue culture and regeneration (87 and 94% respectively for day 11 and 12). Interestingly, this stage also resulted in the highest rate of infection (76 and 90% respectively for day 11 and 12).

A first set of experiments were performed according to the main protocol of Ishida et al. (1996), where dissected embryos are vortexed in the presence of the bacteria. In this case, some wounding occurs in the scutellar region, where the *Agrobacterium* attaches. However, in this case, bacteria preferentially attached to the ventral part of the embryo, in the region of the embryo axis. In order to facilitate *Agrobacterium* infection of the embryogenic-competent scutellar cells, we adopted the strategy of microwounding by means of microbombardment of the scutella with gold particles. In this case, the areas of infection were significantly enlarged. It was subsequently shown that the joint technologies (*Agrobacterium* plus microwounding) did not alter the subsequent capability of the infected tissue in further development.

The data obtained from the work allow drawing the following considerations. In maize, immature embryos and embryogenic primary calli derived from them can effectively be infected with an array of *Agrobacterium tumefaciens* strains, by means of a co-

cultivation procedure which includes preculture of the *Agrobacterium* in a minimal medium, and the addition of acetosyringone 100 μ M in the co-cultivation medium. Regarding the phase of infection and transformation, histologically detected with GUS assay, several genotypes have been revealed to be susceptible, and among these are several agronomically interesting inbred lines. This fact validates the approach and extends the results published by Ishida et al. (1996), who suggested that only A188 and A188-derived crosses could be infected. There was in any case clearly a strong genotypic interaction between the maize genotype and the *Agrobacterium* strain. Unwounded immature embryos offer to *Agrobacterium*, as preferential attachment site, the embryo axis, which is not useful for further callus induction and plant regeneration. In this case, the infection occurs in non-competent tissues for embryogenesis. This problem can be overcome by means of microwounding of the scutellar surface with particle gun mediated- bombardment with gold particles (PDS 1000/He Bio Rad device). Primary embryogenic calli also offer an opportune target tissue and can be infected at high rate with the same procedure. Utilizing embryogenic calli as target, we had to face problems of tissue necrosis and difficulty in callus growth recovery. The problem is on the way to being solved with the use of antioxidants in the culture medium, and the use of timentin instead of cefotaxime as antibiotic for *Agrobacterium* elimination. In our experiments, particle bombardment previous to infection increased the percentage of the *Agrobacterium*-infected embryos and increased the extent of the infected areas at the surface of the scutellum. Finally, the combination of acetosyringone as elicitor of virulence and the preculture of the bacteria in minimal medium, allowed the effective infection of a broad range of genotypes of agronomical interest.

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reverse germ orientation 1 mutants make three florets per spikelet

--Kaplinsky, NK, Freeling, M

Reverse germ orientation 1 - R (rgo1 -R) is a previously described mutant where the embryo faces the base of the ear, the reverse of the normal kernel orientation (Sachan and Sarkar, MNL 52:119-120, 1978). Similar recessive phenotypes have been explained by a reversal of normal development; the lower floret of a spikelet develops as opposed to the upper floret (reviewed in Jackson, MNL 70:66, 1996). It appears that the reverse germ phenotype is actually due to the development of one extra floret per spikelet (pers. comm. G. Chuck and R. Kerstetter). In the tassel, three florets and nine anthers are often, but not always found in *rgo1* plants (Fig. 1). Mutant ears elaborate three florets per spikelet, as opposed to two in wild type (Fig. 2). This suggests that *rgo1* may be involved in regulating the transition of spikelet meristems to floret meristems.

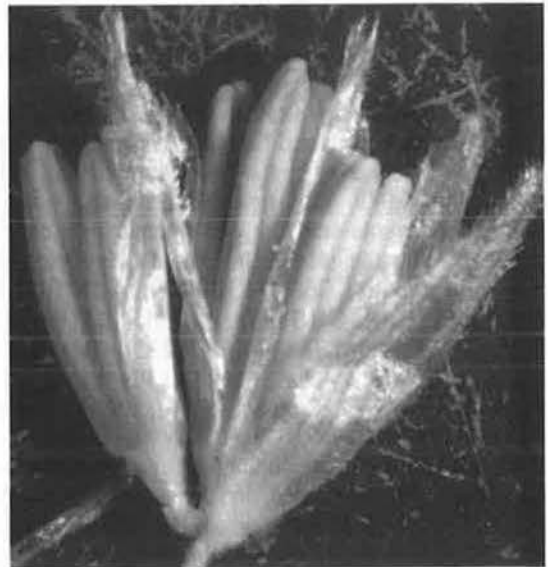


Figure 1. *rgo1* tassel spikelet showing the development of three florets, producing a total of nine anthers per spikelet.

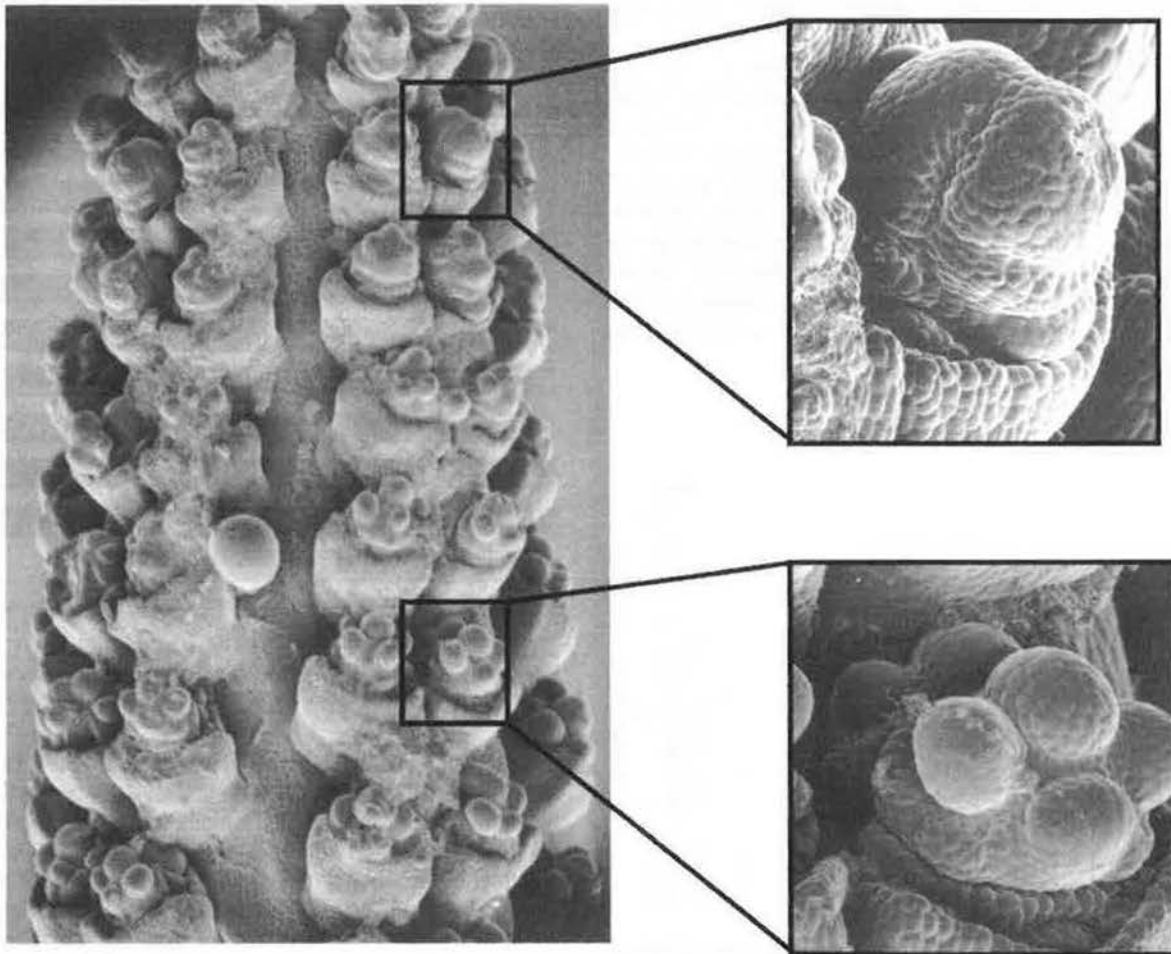


Figure 2. SEM of developing *rgo1* ear. The left panel shows the ear. The top right panel shows three floret meristems developing on one spikelet. The lower right panel shows the third floret developing, with the anther primordia reversed relative to their normal orientation.

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DIMBOA-glu concentration in different taxa of teosinte

--Shahid, M, Esen, A

Hydroxamic acids, Hxs (4-hydroxy-1,4-benzoxazin-3-ones), including 2, 4-hydroxy-7-methoxy-2H-1,4-benzoxazin-3(4H)-one (DIMBOA), play an important role in the defense of certain cereals against bacteria, fungi and insects (Niemeyer, *Phytochemistry* 27:3349-3358, 1988). They have been reported in rye, wheat and maize, where their levels have been shown to have a direct correlation with resistance against different pests (Niemeyer, *Phytochemistry* 27:3349-3358, 1988). DIMBOA and other Hxs occur in plants as 2- β -O-D-glucoside; in this form they are not toxic. DIMBOA-glu and β -glucosidase in intact plant tissues do not interact due to their presence in different subcompartments of the cell. Upon plant tissue disruption, β -glucosidase and DIMBOA-glu come into contact; the enzyme acts on DIMBOA-glu releasing DIMBOA, which is toxic to the organisms that attack the plant.

DIMBOA-glu is found in all parts of maize seedlings; its concentration in leaves is higher than in any other part/organ. In

vitro assays show that DIMBOA-glu is a substrate of maize β -glucosidase (Babcock and Esen, *Plant Sci.* 101:31-39, 1994; Cuevas et al., *Phytochemistry* 31:2609-2612, 1992). Thus, the enzyme has an important role in the activation of plant defense against various pests. In order to investigate whether or not teosinte, the postulated progenitor of maize, has DIMBOA-glu in its leaves, four of its taxa viz., *Z. mays* ssp. *parviglumis*; *Z. mays* ssp. *mexicana*; *Z. mays* ssp. *huehuetenangensis*, and *Z. diploperennis* were analyzed. The same material was also analyzed for β -glucosidase activity.

For DIMBOA-glu extraction, three leaves from three different plants of each taxon were ground to a powder in a chilled mortar and suspended in methanol (weight in gram : volume in ml ratio = 1:3). The homogenate was centrifuged at 14,000 rpm for 5 min in a Beckman microcentrifuge, and the supernatant was used for DIMBOA-glu analysis by HPLC (Waters 450). Ten μ l of the supernatant was fractionated on a C18 column (particle size 5 μ m; dimensions 150 x 4.6 mm) at room temperature using the mobile phase of 59 H₂O : 40 MeOH : 1 HOAc and the flow rate of 1 ml min⁻¹.

Elution of the Hx-glu was detected by absorbance measurement at 262 nm. The elution time for DIMBOA-glu was 5.78 min. The β -glucosidase activity was assayed using p-Nitrophenyl β -D-glucopyranoside (pNPG) as a substrate as

described by Esen and Cokmus (Biochem. Genet. 28:319-336, 1989).

The data indicate that all four teosinte taxa contain the hydroxamic acid DIMBOA-glu (Figure 1), although its level varies from one taxon to the other. The highest concentration of DIMBOA-glu was found in *Z. diploperennis* and the lowest in *Z. mays* ssp. *mexicana*, which is almost the same as in the maize inbred K55 (*Zea mays* ssp. *mays*). These results show that the DIMBOA-glu concentration in *Z. diploperennis* is more than double its concentration in the maize inbred K55. As for the β -glucosidase activity in teosinte leaves, the highest activity was measured in *Zea mays* ssp. *mexicana*, while *Zea mays* ssp. *huehuetenangensis* exhibited the lowest activity (Figure 1).

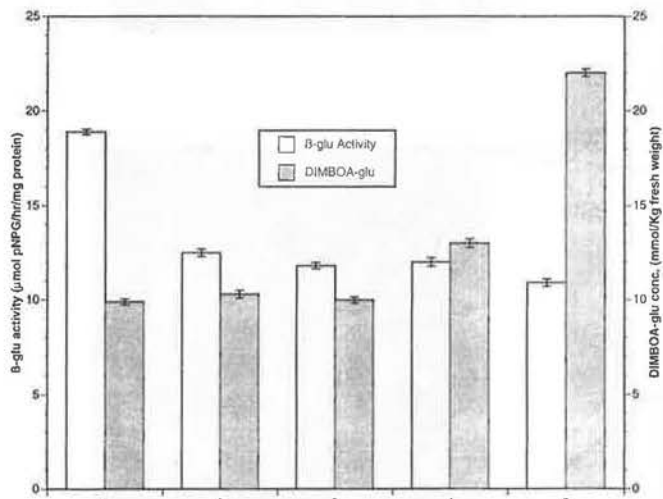


Figure 1. β -glucosidase activity and DIMBOA-glu concentration in the leaves of 15-day old light-grown maize and 4 taxa of teosinte. 1, *Z. mays* ssp. *mays*; 2, *Z. mays* ssp. *parviglumis*; 3, *Z. mays* ssp. *mexicana*; 4, *Z. mays* ssp. *huehuetenangensis*; and 5, *Z. diploperennis*.

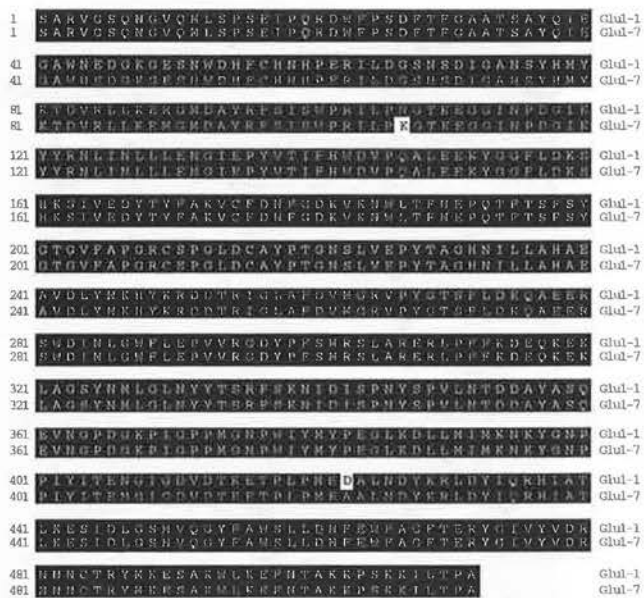
The presence of DIMBOA-glu and β -glucosidase in teosintes suggests that the defense mechanism in the teosinte taxa is similar to that in maize and other cereals.

The difference between two β -glucosidase allozymes at the amino acid sequence level

--Shahid, M, Bandaranayake, H, Esen, A

Analysis of maize and its close relative teosinte reveals that there are, at least, 28 allozymes of the β -glucosidase isozyme Glu1. Of these 28 allozymes, only 8 were found in the common maize inbreds when studies comprising 406 (C.W. Stuber and M.M. Goodman, MNL 56:127-132, 1982) and 363 lines (Kahler et al., MNL 58:32-38, 1984) were conducted. We investigated the two commonly found Glu1 allozymes, Glu1-1 and Glu1-7 at the nucleotide and amino acid sequence levels, in order to determine the molecular basis of electrophoretic mobility differences between these allozymes in native gels.

The comparison of the two allozymes at the protein level reveals that they differ with respect to 2 amino acid substitutions: N vs. K at position 107 and D vs. A at position 423 (Figure 1). Proteins move in a native gel primarily according to their net charge if the pore size in the gel is not restrictive. Thus allozyme Glu1-1 (OH7B) moves faster than allozyme Glu1-7 (K55) in an alkaline native gel because the former has two additional negative charges due to the loss of a K and gain of a D in



Decoration 'Decoration #1': Shade (with solid black) residues that match the consensus exactly.

Figure 1. Alignment of the mature proteins of Glu1-1 and Glu1-7 allozymes at the amino acid sequence level. The difference between the two protein sequences involves 2 amino acid substitutions; N to K at position 107 and D to A at position 423.

compensation to the latter. However, there are 12 different allozymes between these Glu1-1 and Glu1-7 allozymes that can be distinguished based on their electrophoretic mobility. This suggests that the charge difference may not fully explain the electrophoretic mobility differences among the Glu1 allozymes. Some of the mobility differences may result from changes in solvent accessibility and pK of the ionizable groups or compactness of the native structure depending upon the positions of both charged and noncharged amino acids.

Polymorphism of β -glucosidase isozymes Glu1 and Glu2 in maize (*Zea mays* L.)

--Shahid, M, Esen, A

Maize (*Zea mays* L.) β -glucosidase has two isoforms (Glu1 and Glu2). Glu1 is found in all parts of 15 day-old maize plants except in leaves, where the second isoform Glu2 is exclusively present. Glu1 is considered to be the most polymorphic enzyme, having, at least, 28 allozymes (Stuber and Goodman, Crop. Sci. 12:737-740, 1982). To investigate whether or not the Glu2 isozyme is also polymorphic like Glu1, seven maize inbreds, each homozygous for a different Glu1 allozyme, were analyzed for their Glu2 allozyme profile.

For the visualization of Glu1 allozymes, mesocotyls of 15-day-old light-grown seedlings were used, while the leaves (blades) of the same seedlings were used for the Glu2 allozyme studies. The plant parts were harvested on ice and immediately frozen and stored at -70 C until use. Mesocotyls and leaf blades were ground to a powder in a chilled mortar and extracted with 50 mM HEPES buffer, pH 7.0/14 mM 2-ME at a ratio of 1 g plant material to 3 ml buffer. The homogenate was kept on ice for 1h and then centrifuged at 14,000 rpm at 4 C for 30 min in a Beckman microcentrifuge. The supernatant was loaded onto a 6% native gel for electrophoresis and β -glucosidase zymogram development.

Figure 1 A shows the position of different Glu1 allozymes in a

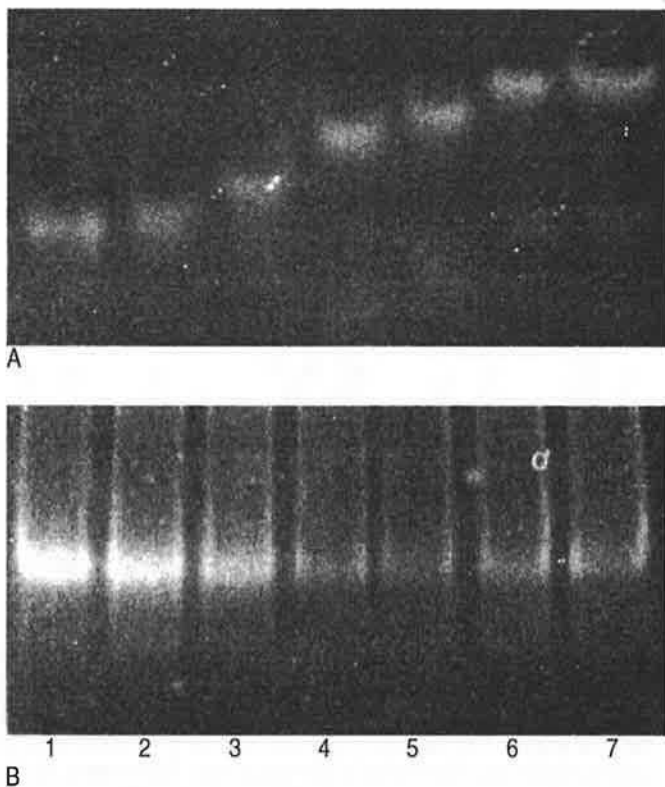


Figure 1. A. Differential mobility of Glu1 allozymes in 6% native gel. The enzyme extracts were made from the mesocotyls of 15 day-old seedlings. B. Mobility of Glu2 allozymes in 6% native gel. The enzyme extracts were made from the leaves of 15 day-old seedlings. 1, OH7B (Glu1-1); 2, H25 (Glu1-2); 3, CO109 (Glu1-3); 4, F44 (Glu1-6); 5, K55 (Glu1-7); 6, CI44 (Glu1-9); and 7, W182 (Glu1-10). β -glucosidase activity was detected by incubating the gel in 1 mM solution of 4-methylumbelliferyl β -D-glucopyranoside.

6% native gel. It is obvious from the zymogram that Glu1-1 is the fastest allozyme, while Glu1-10 is the slowest and the other allozymes are in between them. The zymogram of Glu2 (Figure 1 B) indicates that this isozyme does not have any allozymic variation in these inbreds. The lack of allozymes of the Glu2 isozyme in maize suggests that mutations at the *glu2* gene may be deleterious. This does not seem to be the case for the Glu1 isozyme which has about 28 different allozymes. The physiological and adaptive significance of extensive polymorphism at the *glu1* gene and lack of polymorphism at the *glu2* gene remains to elucidated.

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Preliminary cytological studies on a maize strain carrying the *latent1* supergene

--Pierozzi, NI, Miranda, LEC de, Miranda, LT de*
*deceased

The *latent1* gene complex (*ltp - lte - lsc*) has been studied and mapped in the short arm of chromosome 2 of maize by Miranda and Miranda (MNL 62:39, 1988) and Miranda et al. (MNL 58:48-50, 1984; MNL 60:29, 1986; MNL 61:27-29, 1987; MNL 64:35-36, 1990). This complex has been referred as a supergene and is involved in resistance against drought, heat, and frost. The cellular basis is protoplasmatic resistance by hydrophobic proteins and by sulfhydryl-disulfide bonds. The *latent1* supergene was first discovered in a Michoacan race, and it is

formed mainly by *Fas - Flt - Lsc - ltp - lte* and *B* genes. All these genes are brought together and are involved in an efficient stomatal and heat control mechanism associated with anthocyanin pigment presence. According to Miranda et al.'s hypothesis (MNL 67: 21-24, 1993) the latent complex is maintained together because: (1) the distance between these genes is so small; and (2) the complex is near a knob in the short arm of chromosome 2 (2S) in such a way that crossing over is reduced or not formed inside the complex, favoring the supergene's existence. In a breeding program developed in IAC, some directed crossing involving races such as Tuxpeños and Catetos with the *latent1* donor Michoacan race has been done. Cateto was an autochthonous Brazilian race similar to the Coastal Tropical Flints (McClintock et al., Colegio de Postgraduados, Chapingo, 1981). It has some resistance factors such as aluminum tolerance mapped on chromosome 10, but does not have *latent1* supergene (Miranda et al. MNL 58:38-46, 1984).

Preliminary cytological analyses were done in two stocks of Cateto Assis Brasil sub-race, native to the Brazilian southern region, as part of the breeding program. One of these stocks, named Ip 48-5-3, was obtained by 3 cycles of selfing. It was considered as the "control", because it does not have the *latent1* complex in chromosome 2. The other stock was the Ip 48-5-3 *lte-1* "isoline" which was obtained through crossing Ip 48-5-3 with a *latent1* stock derived from Michoacan race 21 compuesto 1-104 germplasm (IAC Maya latente). Both inbred lines were field tested in different experiments to detect the presence of the *latent1* character.

Seeds of both Cateto Ip 48-5-3 "control" and Ip 48-5-3 *lte-1* were germinated in moist filter paper at 26 C. Roots were collected, treated in para-dichlorobenzene saturated solution (p-DB) for 3 hours at 16 C, fixed in Carnoy 3:1 solution (ethyl alcohol p.a. and acetic acid p.a., respectively) and stored at -20 C until the cytological preparations were done. Softening of tissues was done by treating the roots with an equal enzymatic solution mixture of pectinase 20% and cellulase 2%. We employed the C-band procedure as described by Pierozzi and Jung-Mendaçoli (Cytologia 62:80-91, 1997) for metaphase chromosome characterization. C-banding technique was chosen because there is a good correspondence between pachytene knobs and mitotic metaphase C-bands, as demonstrated by Aguiar-Perecin and Vosa (Heredity, 54:37-42, 1985).

Preliminary data have shown that this variant of C-bands was suitable for visualizing knob heterochromatin. Ip 48-5-3 Cateto control has a C-band only in chromosomes 3, 6 and 7 and none in the short arm of chromosome 2 (Figure 1A). Isoline Ip 48-5-3 *lte-1*, which carries the *latent1* supergene, has C-bands in chromosomes 3, 6, 7 and 8 and also in the 2S position (Figure 1B). Knob heterochromatin number revealed by C-bands in the control stock differs from that obtained by McClintock et al. (Colegio de Postgraduados, Chapingo, 1981) for some Cateto samples. These authors found a higher knob number than that observed here and almost all chromosomes have knobs. It is possible that this Cateto sub-race was not plotted in that work.

The involvement of knob heterochromatin in the establishment of supergenes was first proposed by Kato (Colegio de Postgraduados, PhD thesis, 1976). Heterochromatin DNA has some properties of recombination inhibition near it, as cytologically demonstrated by authors such as Torrezan and Pagliarini (Caryologia 48:247-253 1995). In maize evolutionary history, knobs were set in some chromosome sites. Recombination events

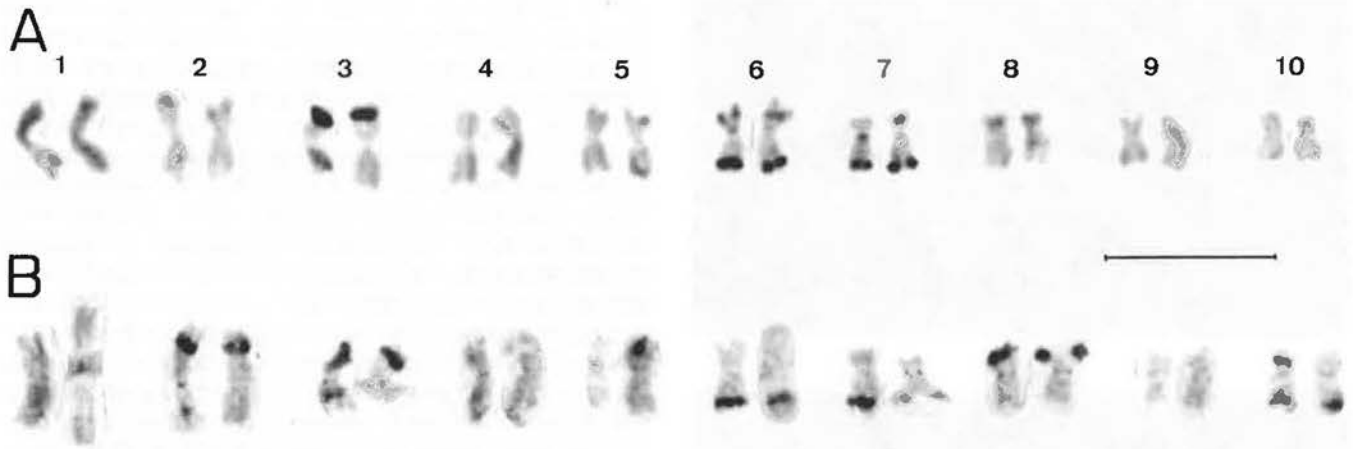


Figure 1. C-band karyotype of two stocks of Zea mays. A) Ip 48-5-3 considered as control with no C-band in chromosome 2 short arm. B) Isoline Ip 48-5-3-*lte-1*, carrying latent-1 supergene, with C-band in chromosome 2 short arm. In this isolate, one chromosome of pair number 1 was stretched. Bar = μm .

were probably reduced in their neighbourhood and in some cases this event may have favored supergene development which just conferred different adaptive values to the races (Kato, 1976). So, in this way the 2S knob, as visualized by C-banding technique in the isolate Ip 48-5-3-*lte-1*, must have helped the development of the *latent1* supergene, as hypothesized by Miranda et al. (MNL67:21-24, 1993).

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Continued studies on the genic stability of the progeny of maize anther culture-derived microspore plants

--Ting, YC, Nguyen, DQ

In the last summer, about 200 kernels of a self-pollinated maize ear derived from anther culture of KH-13 were planted in the field. The objective of the experiment was further examining genic stability of this inbred. Before last summer, this line had persistently shown instability in its selfed progeny.

The kernels were randomly picked from a fully filled ear borne on a healthy and dark green plant. The kernels appeared normal. They germinated readily. However, when the plants were three weeks old, some of them began to show symptoms of slow growth. One week later, the slow growth plants started to express other abnormalities, such as yellow-green leaves, and lazy-growth habit by prostrating on the ground. In late July, the results of this experiment were under total evaluation. It was found that 70 plants had reached adult stage with flowering and seed-setting, while 18 of them were defective in height and leaf-chlorophyll content. These defective plants also had yellow-green leaves. Their tassels were small and no anthesis was observed. Their stalks were barren. In addition, the lazy plants were also sterile.

The above abnormal plants were presumably mutations. Due to their sterility, there was no way to test this hypothesis. The ratio between normal plants and abnormal ones did not fit Mendelian expectation. It is assumed that the original parental plant had a silent *Ac* element which was activated by anther culture per se. IN consequence of this, this inbred became highly mutable.

In parallel with the above, another experiment was conducted on a second inbred which was derived from maize Dan-Sun 91 by

anther culture. This inbred line was a product of continued selfings of the original doubled-haploid. Over 120 kernels were employed and sown in the field. Seedlings and adult plants of these plantings were vigorous in growth. Their leaf chlorophyll appeared normal in content. Their stalks were strong and the stalk diameter appeared, on average, greater than that of the parental plant. This structure may account for their resistance of lodging which was discovered a few years ago. There were, on an average, two ears borne on each of these plants. The ears were small in size, but uniform in appearance. No abnormalities of any kind were observed. In other words, this inbred line was genically stable. Thus, it may allow for practical application, such as hybrid seed production, without any needed testing.

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A confocal laser scanning microscopy study of 4 embryo specific mutants

--Elster, R, Bommert, P, Werr, W

Embryo specific (emb) mutants in maize are specifically blocked in embryogenesis whereas endosperm development - unlike in *defective kernel* mutants - is not obviously affected. Phenotypic characterization in a large collection of 51 *emb* mutants was accomplished, so far, either in fully mature (40 to 60 days after pollination (DAP)) and fresh dissected kernels or in rehydrated seeds. A wide range of promising developmental blocks, interfering with early and late stages in embryo development, could be identified (Sheridan and Clark, Plant J. 3:347-358, 1993; Clark and Sheridan, Plant Cell 3:935-951).

In order to have a more detailed view of the aberrant mutant embryo morphology, we have analyzed a subset of this mutant collection by use of a confocal laser scanning microscope (CLSM). Mutant embryos from four different lines, *emb5* (former *emb**-8518), *emb9* (former *emb**-8521), *emb**-8537, and *emb**-8542, harvested between 8 DAP and 20 DAP, were fixed, stained with propidium iodide and analyzed according to Running et al. (Methods in Cell Biol. 49:217-229, 1995). Compared to their wild type siblings all 4 mutants show retarded development already at 10 DAP and are easily distinguishable at this stage. However, at

15 DAP the specific blocks become more evident: *emb5* and *emb9* are blocked at a late proembryo/ early transition stage (see Figure 1) and fail to establish a radial asymmetry as wild type siblings do by induction of a shoot meristem (SM) and a scutellum. Mutants of this type are unable to overcome the block and even at later stages in kernel development the embryos still reside in a late proembryo or early transition stage. Cells of *emb5* and *emb9* mutant embryos are frequently highly vacuolized, indicating a terminal differentiation.

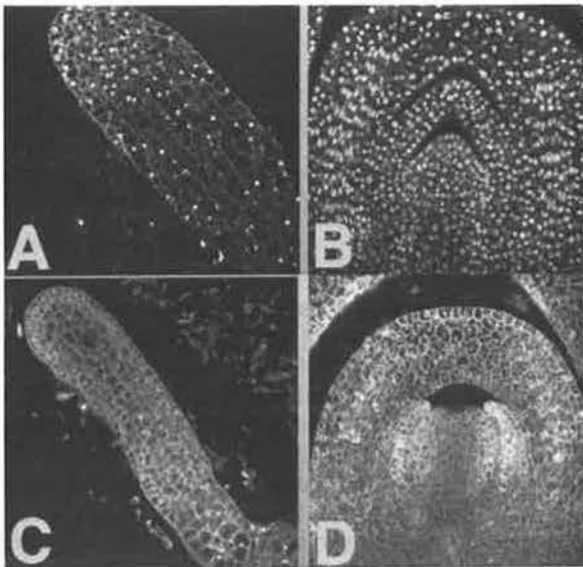


Figure 1. 1A shows a homozygote *emb5* embryo 15 DAP which is blocked in an early transition stage whereas wild type siblings are already at leaf stage 1 (seen in 1B). 1C and 1D show the corresponding pictures for *emb9*.

In contrast, mutant embryos from lines *emb*8537* and *emb*8542* show induction of a scutellum and organization of a region of cells resembling a SM are visible. At least for *emb*8537* we never found evidence for the formation of a coleoptile or embryonic leaves. Some *emb*8542* embryos analyzed at later stages, however, showed hyperplasia-like outgrowth and further embryonic tissues were formed, leading to structures which lack major plant organs, as roots or shoots are not produced.

Presently we are in the process of refining our CLSM data with an in situ analysis of mutant embryos at different developmental stages after pollination. By use of specific markers, commonly associated with the existence of a SM, we will try to establish whether the formation and function of this meristem in our mutants is affected also in molecular terms.

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Functional variants of a maysin QTL --- Is there a difference?

--Lee, EA, Byrne, PF, McMullen, MD, Snook, ME, Coe, EH

Genetic variation is essential to detect QTLs, but is it as simple as that? The P1 myb-like transcription factor encoded by the *p1* locus is responsible for activating C-glycosyl flavone, 3-deoxyanthocyanin, and phlobaphene synthesis in pericarp, cob, and

silk tissue (Grotewold et al., PNAS U.S.A., 88:4587-4591, 1991; Grotewold et al., Cell, 76:543-553, 1994; Byrne et al., PNAS U.S.A., 93:8820-8825, 1996). Because of its control over C-glycosyl flavone synthesis, *p1* is a major QTL for maysin synthesis and corn earworm (*Helicoverpa zea* [Boddie]) antibiosis (Byrne et al., 1996; Byrne et al., J. Econ. Entom., 90:1039-1045, 1997). There are five allelic variants of *p1* that are distinguished by their tissue specificity: *p1-rrb*, *p1-rww*, *p1-wrb*, *p1-wwb*, and *p1-www* (Peterson, Genetics 126:469-476, 1990; Chopra et al., Plant Cell, 8:1149-1158, 1996; Coe, MNL, 59:40, 1985; Coe and Han, MNL, 60:55, 1986; McMullen unpublished data). Three of the variants direct flavone synthesis in silk tissue: *p1-rrb* (red pericarp, red cob, browning silks), *p1-wrb* (colorless pericarp, red cob, browning silks) and *p1-wwb* (colorless pericarp, white cob, browning silks). Silk browning is due to oxidation of accumulated flavones following tissue damage (Levings and Stuber, Genetics 69:491-498, 1971). Regardless of pericarp expression, *p1* alleles that confer cob glume pigmentation also confer silk browning. During development, cell layers that give rise to glume tissues are believed to give rise to silk tissues (Emerson, Genetics 2:1-34, 1916; Peterson, Genetics 126:469-476, 1990; McMullen personal observations). While almost all white-cobbed lines lack flavones in silk tissue, a number of exceptions occur (Coe, 1985; Coe and Han, 1986). One exception is NC7A (*p1-wwb* allele), which lacks both pericarp and cob glume expression, but still confers silk browning.

Is a *p1-wwb* allele as effective as a *p1-wrb* allele in directing maysin synthesis? Are the antibiotic properties of *p1-wwb* silks equivalent to those of *p1-wrb* silks? We examined these questions in an F2 population developed from a cross between the inbred lines GT114 and NC7A. NC7A has a *p1-wwb* allele and GT114 has a *p1-wrb* allele. Both inbred lines accumulate appreciable amounts of maysin. Silk tissue was collected from the 316 individual F2 plants and maysin levels were determined as described in Byrne et al. 1996. Plants were selfed to produce F2:3 families. Ninety F2:3 families were grown out in 2-row plots for silk tissue collection to determine maysin content and antibiotic properties as described in Byrne et al. 1997. F2 individuals were genotyped molecularly using a *p1* RFLP probe and 86 other molecular markers (10 linkage groups with a total map length of 1414 cM). Single factor analysis of variance did not detect significant ($P < 0.01$) differences among the *p1* genotypic class least-square means for F2 individual maysin concentration, F2:3 family maysin concentration, and F2:3 family larval weights (Table 1). Epistatic interactions between *p1* and the other 86 loci were also examined. Again, the *p1* region was not involved in any significant ($P < 0.001$) epistatic interactions for maysin. So, even though the *p1* alleles from the parents are different in terms of tissue specificities, in terms of maysin synthesis in silk tissues and corn earworm antibiosis they are equivalent to one another. Genetically distinct alleles at a QTL do not necessarily result in different phenotypic

Table 1. *p1* genotype least-square means of individual F2 and F2:3 family maysin concentrations and F2:3 family insect bioassay larval weights. Maysin and larval weight *p1* genotype class least-square means were not significantly ($P < 0.01$) different from one another.

<i>p1</i> genotype	F2 maysin conc. (% fresh wt.)	F2:3 maysin conc. (% fresh wt.)	F2:3 larval wt. (mg.)
<i>p1-wrb</i> / <i>p1-wrb</i>	0.39	0.24	88.7
<i>p1-wrb</i> / <i>p1-wwb</i>	0.36	0.22	105.2
<i>p1-wwb</i> / <i>p1-wwb</i>	0.33	0.14	137.7

expression in all tissues, and thus may not allow detection of the QTL. The genetic variation must be specific to the tissue in question.

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Effect of metholachlor on pollen germination of different maize genotypes

--Satarova, TN

The inclusion of microgametophyte selection in breeding programs can greatly improve the process of the creation of the initial material of maize. It is determined by such characteristics of the male gametophyte as small size, a great number of pollen grains, the possibility of analyzing without damage to the maternal organism, and also by the data on the expression of a part of sporophyte genes in the period of the gametophyte's existence. Investigations on maize gametophyte selection confirm the efficiency of such an approach. The estimation of genotypes involved in pollen tolerance to different environment factors also requires attention because the ability to form a sufficient amount of pollen, the viability of the male gametophyte and other characteristics are important breeding characters. The elaboration of the details of microgametophyte selection will permit carrying out such selection in the prospective populations at the early stages of the breeding process.

We investigated the ability of pollen grains of 5 different lines of maize to germinate on artificial nutrient medium with the herbicide metholachlor=2-ethyl-6-methyl-N-(1-methyl-2-methoxy-ethyl)-chloracetanilide (commercial mark - dual 960EC). Pollen was taken from field plants which were isolated at the beginning of flowering. The control germination medium included 150 g/l sucrose, 300 g/l calcium chloride, 100 mg/l boric acid and 6 g/l agar. The germination was carried out at a temperature of 26-28 C. The control medium was added accordingly with 10 mg/l, 50 mg/l, 100 mg/l and 500 mg/l metholachlor. The data are shown in the table.

Table. Effect of herbicide metholachlor on the germination of maize pollen grains in vitro.

Metholachlor mg/l	Percentage of pollen grains germinated				
	751	GK26	1620	DK427/501	DK4/217
0	54.78	13.22	11.50	9.83	7.75
10	42.63***	12.33	16.50*	13.17	11.63**
50	10.38***	3.78***	4.17***	2.33**	1.56***
100	0***	0.44***	0.83***	0.17***	0***
500	0***	0***	-	-	-

*, **, *** - differences from control are significant at P<0.05, P<0.01 and P<0.001 accordingly.

First of all it is necessary to remark that different genotypes differ on the percentage of pollen grain germination in control. The greatest percentage was obtained for line 751, the others did not have a very high percentage of germination, which could be connected with genotypical peculiarities and also, maybe, with the specific reaction of genotypes on the weather conditions of the year.

For line 751, which had the highest percentage of germination in the control (54.78%), all concentrations of metholachlor significantly reduced the percentage of pollen germination. For line GK26, where the percentage of germination in the control was 13.22, inhibition was observed from the concentration of 50

mg/l metholachlor. For other genotypes with a lower percentage of germination in the control the concentration 10 mg/l had a stimulating effect and a significant decline was obtained at the metholachlor concentration of 50 mg/l. A fall in the percentage of pollen germination together, 63.74-81.05% for concentration 50 mg/l and 92.78-100% for concentration 100 mg/l was found. However, not all genotypes decreased the percentage of pollen germination at the same rate. The least decreases, 63.74% and 92.78% for the two above mentioned concentrations were observed for line 1620 and the greatest, 81.05% and 100% for line 751.

DULUTH, MINNESOTA
University of Minnesota Duluth

Isolated meiocyte culture of *polymitotic* microspores allows direct observation of abnormal cell division

--Wolfe, KW, Liu, Q

Direct observation of meiotic divisions of developing maize microspores has become possible using the recently developed ADLP media (developed by Annette Chang, University of California Berkeley) for meiocyte culture. For the purpose of this study, efforts were concentrated on the development of techniques that allow the direct observation of *po* post-meiotic divisions, to yield information on the process of its post-meiotic abnormal cell cycles. Previously, no development beyond the tetrad stage has occurred in meiotically dividing cultured microspores. Here however, we report that during isolated *po* meiocyte culture, development proceeds through the tetrad stage and also through a number of subsequent cell cycles. These methods and results may be useful to the study of cell cycle regulation and the process of cell division.

Meiocytes and microspores were isolated and cultured in ADLP media. To visualize chromatin structure, some cultures included SYTO 12 Live Cell Nucleic Acid Dye (Molecular Probes, Eugene OR) and observed for a period up to 1 week. After the second meiotic division, microspores remained in the tetrad stage (Figure 1a) a short time before chromatin condensation (Figure 1b) and

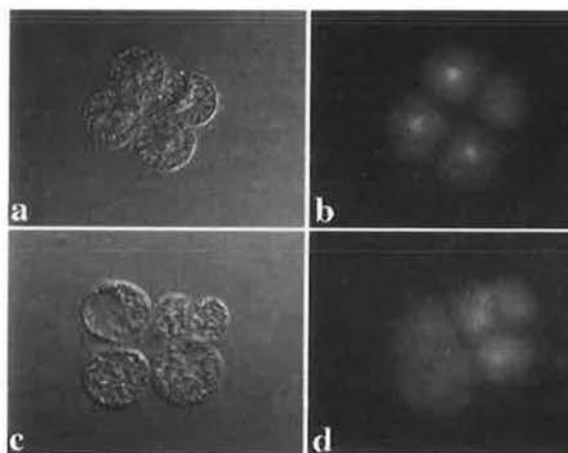


Figure 1. Isolated culture of *po* tetrads. a) a *po* microspore before post-meiotic divisions; b) chromatin is observed to condense before cells divide; c) the same cell pictures in (a), after the first post-meiotic division; d) chromatin is observable throughout post-meiotic divisions.

mutational cell division occurred (Figures 1c and 1d). The best results were obtained with cells isolated immediately following meiosis II. Cells isolated both before and during the meiotic divisions were observed to continue development through both meiotic divisions and post-meiotic divisions, although to a much lesser degree. Meiotic divisions were noted to be synchronous, in contrast to the post-meiotic divisions, which were very asynchronous.

The timing of extra cell divisions varied widely and frequently, as cells within the same tetrad were observed to divide in a very non-synchronous manner. Often one cell of a tetrad would undergo many extra cell divisions while another in the same tetrad would undergo only one or two. Chromosome condensation was always observed in cells cultured with the SYTO 12 living cell nucleic acid fluorescent dye following the second meiotic division, preceding post-meiotic divisions. Cells incubated with SYTO 12 were not observed to go through the same extent of extra cell divisions, and the timing of extra cell divisions was noticeably longer.

The culture conditions do not alter microspore development appreciably from what is witnessed during *in vivo* microsporogenesis, although more work is underway for a complete characterization of cell division timing and behavior. Results obtained so far indicate that cells proceed through the mutational cell cycles to the same extent, and the timing of the divisions appears to match those observed *in vivo*, without the influence of the surrounding somatic anther tissue.

Microfilament distribution in the maize male sterile mutants *ms2* and *polymitotic*

--Wolfe, KW, Liu, Q

The role of microfilaments in cell wall formation through the progression of microsporogenesis was explored using two maize male sterile mutants. This comparative study utilized *ms2*, a postmeiotic mutant of maize in which developing microspores are deficient in cell wall synthesis, and *po*, a mutant that develops a normal cell wall despite the occurrence of a number of postmeiotic disturbances. The first mutational abnormalities are observed to initiate at the same stage, in the tetrad stage after meiosis II, in both of these mutants. A wild-type strain was also studied as a control.

When compared to the microfilament array of similarly staged wild-type microspores (Figure 1a), the *ms2* mutants do not display a reticulate staining pattern that is consistent throughout the cytoplasm. Instead, microfilaments combine to form thick cables that display little contact with the membrane (Figure 1b), except for a small number of fine microfilaments that branch off actin cables, which are oriented parallel with the membrane. After release from the tetrad, microspores never form an ordered cortical staining pattern typical of wild-type microspores of the same stage, but instead show a more random cytoplasmic array that displays plasma membrane contact (Figure 1c). As development proceeds, the microfilaments become fewer in number as they condense into very thick microfilament cables, which extend randomly throughout the cytoplasm (Figure 1d).

Abnormalities in microfilament arrays are observed in the *po* mutant only after the tetrad stage, upon initiation of post-meiotic divisions. Following tetrad formation, the microfilament array appears quite normal, and exhibits formation of small actin patches visible in most cells (Figure 2a). Shortly after this stage,

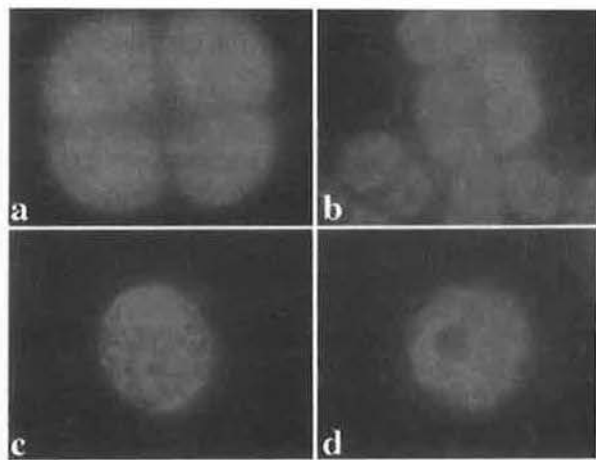


Figure 1. Wild type and *ms2* microspores stained to visualize microfilament distribution. a) a wild type tetrad showing a normal reticulate staining pattern; b) *ms2* mutant microspores in which microfilaments join to form thick cables; c) *ms2* microspore after tetrad release; d) *ms2* microspore after a longer period of development.

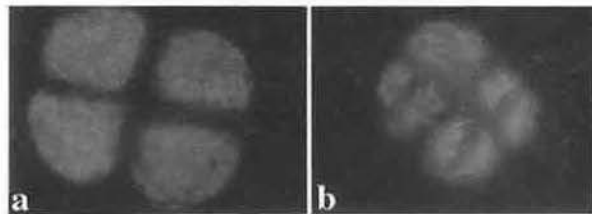


Figure 2. *polymitotic* microfilament distribution. a) microfilaments show a normal distribution before the onset of post-meiotic divisions; b) microfilaments become more disorganized as cells undergo abnormal divisions.

coinciding with the onset of abnormal post-meiotic divisions, the microfilament array appears to constrict or shrink within the cells, lending an appearance of a more disorganized microfilament array (Figure 2b). After microspore release from the tetrad, the cell wall develops to a normal thickness, despite the absence of an organized microfilament array and an intact nucleus.

An interesting correlation between the absence of cell wall formation, and an abnormal microfilament array existed during development of *ms2* mutant microspores. At similar stages wild-type and *po* microfilament arrays are observed to extend throughout the cytoplasm, and to contact the cell membrane at many points. Cortical actin patches were observed in wild-type and *po* tetrads, but were not present in *ms2* tetrads. Similar structures have been found in yeast, where the location of these actin patches has been shown to correlate with new cell wall synthesis (Gabriel and Kopecka, *Microbiology* 141:891-899, 1995; Mulholland et al., *J. Cell Biol.* 125:381-391, 1994). If the actin patches observed in wild-type and *po* represent analogous structures to those observed in yeast, the early tetrad stage could represent the time period critical to the initiation of normal cell wall formation in maize microspores.

FREIBURG, GERMANY

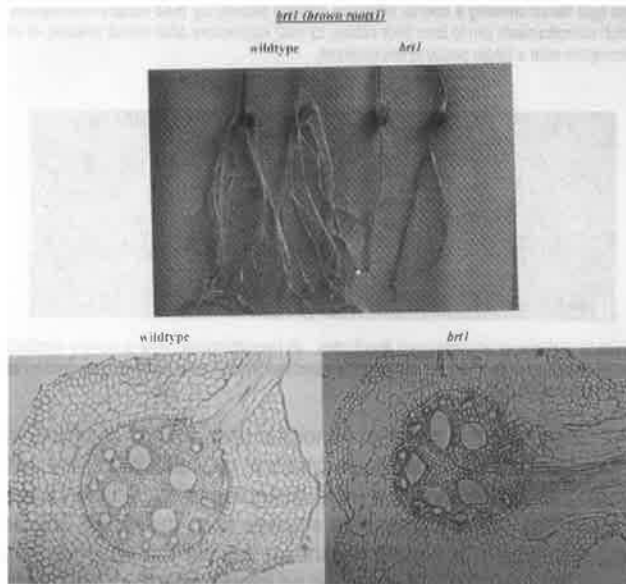
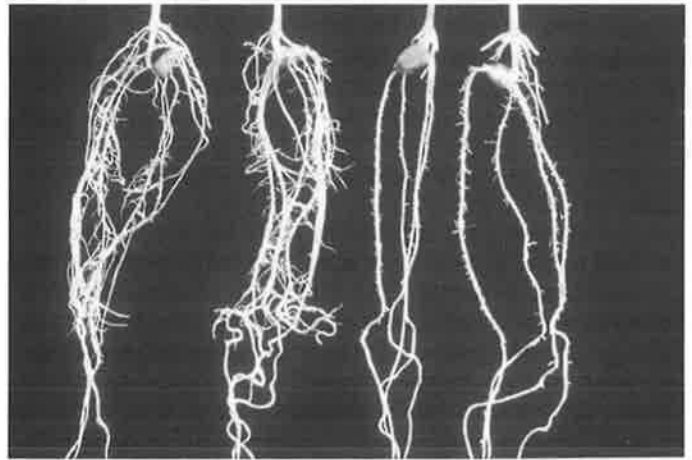
Albert-Ludwigs-University Freiburg

Isolation of the new necrotic root mutant *brt1*

--Hochholdinger, F, Feix, G

During our screening of segregating F2 families derived from

potentially mutator MU tagged material we observed seedlings with their roots turning brown approximately one week after germination. This led to a drying out of the above ground part of the growing seedlings and soon after to a dying of the affected seedlings. Histological analyses of primary root sections of the necrotic mutant seedlings (shown to be monogenic recessive and designated *brt1* for brown roots 1) revealed that the browning is restricted to the central cylinder including the vascular strands which are essential for water and nutrient uptake. A coloured picture showing the brown roots of the mutant seedlings in comparison with wild type seedlings, as well as the comparative cytological analysis, can be seen on the web version of the MNL. A possible correlation of the *brt1* locus to senescence or apoptosis makes this mutant and the search for the affected gene particularly interesting.



Isolation of the new root mutant *slr1* affecting lateral root formation

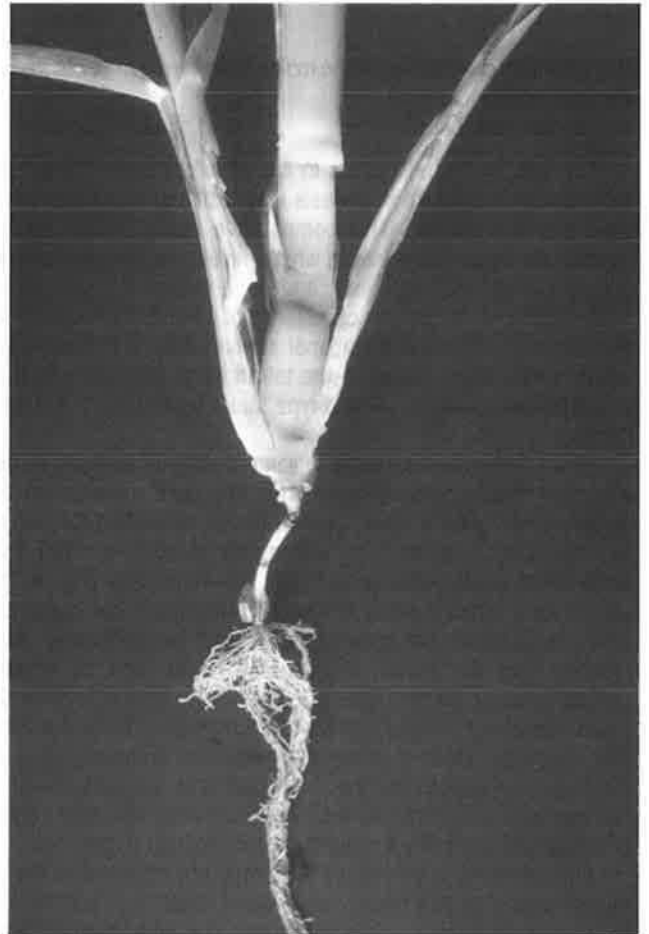
--Hochholdinger, F, Park, WJ, Feix, G

In an effort to identify mutants with deficiencies in the formation and morphogenesis of roots we identified in the segregating F2 population of mutator MU tagged material a monogenic and recessive new root mutant (designated *slr1* for short lateral roots) concerning lateral root formation. Lateral roots normally start to appear on all different root types (primary-, lateral seminal-, crown- and brace roots) at 6-7 days after root initiation. *slr1* seedlings initiate lateral root growth at normal frequency compared to wildtype seedlings of the same age, however at reduced length as shown in the Figure. This is in contrast to the previously described mutant *lrt1* (Hochholdinger and Feix, MNL 1997) which displays no lateral roots 14 days after germination. In *slr1* plants, in addition to the shortened lateral roots, the first two leaves are pale green, which represents a further trait convenient to being used in screening programs. An allele test with *lrt1* and gene isolation attempts are in progress.

Tiller formation in Gaspé Flint is not affected by the *rtcs* mutation

--Hochholdinger, F, Feix, G

Gaspé Flint plants display normally extensive tillering from the lower nodes as a dominant trait. Since crown roots emerge also from the lower nodes, a mutual influence of tiller- and crown root formation initiated from the same node would be an interesting



feature of the nodes. To test this allusion we took advantage of the root deficient mutant *rtcs* (see MNL 66:45, 1992), which lacks completely crown root formation and does not even display any microscopically visible crown root primordia (W. Hetz et al., Plant J. 10:845-857, 1996), and transferred the *rtcs* locus into Gaspé Flint. This was done by crossing the monogenic recessive *rtcs* with Gaspé Flint and analyzing the segregating F2 population for the occurrence of *rtcs* plants without tillering, which would be an indication for the influence of the *rtcs* locus on tiller formation. However, we observed only tillering *rtcs* plants (a representative example is shown in the Figure), which leads us to conclude that the molecular defect of *rtcs* plants leading to a lack of crown root formation is without influence on the tiller initiation from the same nodes.

HAMBURG, GERMANY
University of Hamburg

Gene expression during fertilization and zygote development

--Dresselhaus, T, Cordts, S, Heuer, S, Bantin, J, Lörz, H, Kranz, E

Numerous mutants have been described in maize affecting many developmental processes. Concerning sexual reproduction there are mutants available where development is disturbed e.g. in flower formation, meiosis or embryogenesis (for review see Neuffer et al., Mutants of Maize, Cold Spring Harbour Laboratory Press, 1997). Nevertheless, the fertilization process, zygote development and very early stages of embryogenesis are to our knowledge not covered by described mutants. One reason is that these processes occur deeply embedded in the maternal tissues of the ovule and it is therefore difficult to get access to specific developmental stages. This is especially a problem when the above described processes are studied not only at the cytological, but also at the molecular level.

We are following an alternative approach: the above described developmental processes have recently been transferred from in vivo to in vitro (reviewed by Kranz and Dresselhaus, Trends Plant Sci. 1:82-89, 1996) and it is now possible to get access to gametes as well as to specific stages of zygotes and early embryos. To investigate these cells at the molecular level, we have established sensitive RT-PCR methods. Gene expression of every known gene from maize can be studied at the single cell level (Richert et al., Plant Sci. 114:93-99, 1996) and representative cDNA libraries can be generated from only a few plant cells (Dresselhaus et al., Plant J. 5:605-610, 1994). Using the latter technique we have constructed a cDNA library of 128 unfertilized egg cells and a second cDNA library of 104 in vitro zygotes (18 h after fertilization; Dresselhaus et al., Plant Mol. Biol. 31:23-34, 1996). Further cDNA libraries have been generated from mature pollen and seedlings.

Using differential screening methods and gene specific probes, we have identified more than 50 genes, whose expression is up- or down-regulated after in vitro fertilization. Some genes are newly induced after fertilization. Interestingly, the majority of isolated transcripts have not been described before in maize. Some transcripts have not been described before in plants and others do not show homology to known proteins or DNAs in databases. Isolated cDNA clones encoding proteins with homology to known proteins are involved e.g. in RNA stability and

degradation, translation, DNA replication and repair, energy metabolism, protein modification and degradation. We are now analysing a few genes with interesting expression pattern in more detail, using e.g. in situ hybridization techniques, transgenic overexpression and antisense approaches as well as the gene machine to isolate *Mu*-transposon knock-outs.

Isolation and characterization of genes which are strongly down-regulated after fertilization

--Cordts, S, Kranz, E, Brettschneider, R, Lörz, H, Dresselhaus, T

The molecular characterization of the unfertilized egg cells is a topic of great importance to understand pre- and postfertilization events in higher plants more precisely. In this context it is interesting to answer the question of how much information required for further developmental processes is already deposited in the unfertilized egg cell. Since many plant cells are able to initiate embryogenesis, it is interesting to examine the difference between unfertilized egg cells and somatic cells.

With the intention to isolate genes which are specifically expressed in the egg cell, we have made a differential screening of the RT-PCR cDNA library of isolated egg cells against the RT-PCR cDNA library of in vitro zygotes (18 h after in vitro fertilization) and a conventional cDNA library of 10-day old seedlings. The result of the screening is a number of clones that are strongly down-regulated after fertilization. One group of clones does not show expression in other maize tissues and also lacks homology to known genes. A number of clones from this group belong to the same gene family, but code for different amino acid sequences. This gene family and a further clone of this group were analysed by tissue and single cell-whole mount in situ hybridisation methods (Figure 1). The expression of these clones

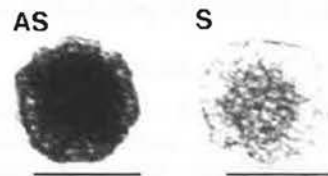


Figure 1. Whole mount in situ hybridisation of isolated egg cells with antisense (AS) and sense (S) probes of an embryo sac-specific clone (bar: 50 µm).

seems to be embryo sac-specific. A second group of isolated clones that are more strongly expressed in egg cells than in zygotes show no homology to known sequences in databases but are expressed in other maize tissues. Two of them are highly expressed in embryogenic cell cultures. The function of one of these clones (*Zmec 88*) has been analysed by transformation experiments. This clone is expressed in embryogenic suspension cells, yellow-green leaves and in uni- to bi-nucleate microspore stages. According to this expression pattern the phenotype of transgenic antisense plants in T1 and T2 - generations is partially male sterile and plants have light and transparent spots in their leaves (Figure 2).

To get more information about the functions of the above described clones, we will perform transformation experiments also with the embryo sac-specific genes and study the subcellular localization of the corresponding proteins using antibodies.

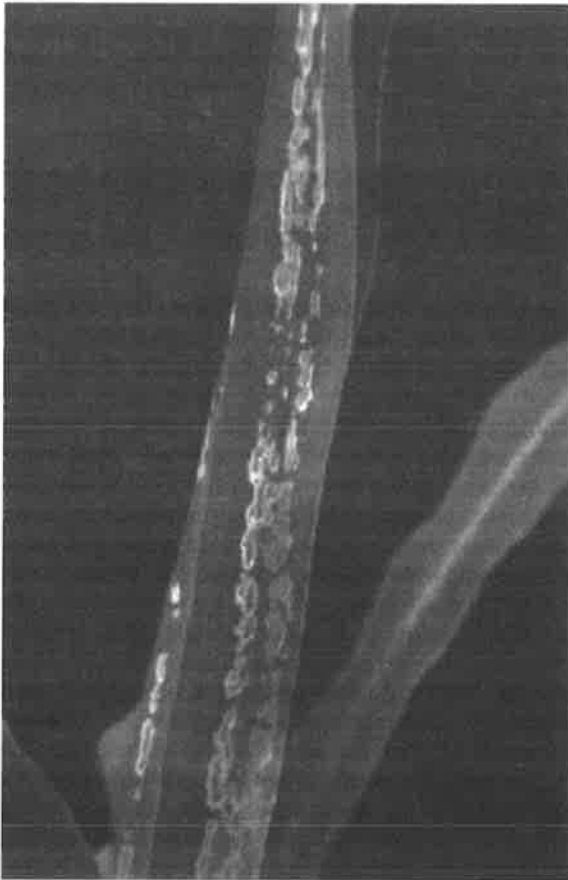


Figure 2. Phenotype of a transgenic maize plant, transformed with an antisense construct of the clone *Zmec 88* showing leaves with clear spots.

Expression analysis of MADS box genes in pollen, egg cells and zygotes

--Heuer, S, Lörz, H, Dresselhaus, T

MADS box genes represent a highly conserved gene family of transcription factors in plants. During recent years an increasing number has been isolated from different plant species. They were first identified due to their functions in flower organ development, but now it becomes obvious that MADS box genes are also expressed during embryogenesis and in vegetative organs.

To address the question of whether MADS box genes are also expressed during the fertilization process, we have screened cDNA libraries of maize egg cells, in vitro zygotes and mature pollen with different maize MADS box probes under medium stringent conditions. Homologous as well as novel MADS box genes should be detected under the conditions applied. Figure 1 shows the control experiment of the screening. Hybridization signals were obtained in the cDNA libraries of egg cells and pollen, as well as in the seedlings library, which served as a positive control. Under the conditions used no signal could be detected in the zygote library.

The subsequent plaque screening of the egg cell, zygote and pollen libraries led to the isolation of two novel MADS box genes from the pollen library and one novel MADS box gene from the egg cell library. No MADS box gene was isolated from the zygote library. Gene expression studies have shown that two MADS box

cDNA libraries MADS box probes*

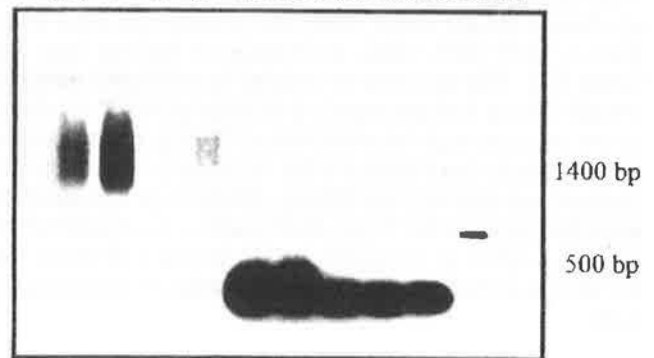


Figure 1. MADS box genes detected in cDNA libraries of egg cells (EC), mature pollen (P) and seedlings (S), but not in cDNA libraries of in vitro zygotes (Z). The libraries were screened with radiolabelled maize MADS box probes. A representative hybridization with the ZAG3/5 probe is shown. Cross-hybridization with all other MADS box probes has occurred due to the conservation of the MADS box (lane 5-10). (* For references see EMBL data bases: ZAG1: L18924; ZAG 3: L46397; ZAG5: L46398; ZEM2: X85335; ZEM3: X85336; ZMM1: X81199; ZAP1: L46400; ZAG2: X80206; ZAG4: L46399)

genes are expressed in egg cells and are completely downregulated in the zygote library. All three genes are expressed in organs other than egg cells and pollen, respectively, with an expression pattern which indicates that they are highly regulated. RNA in situ hybridizations and transformation experiments are in progress and will gain more information about these novel MADS box genes.

IBARAKI, JAPAN
Institute of Radiation Breeding

Nitrous oxide (N₂O) is effective for chromosome counting in maize

--Kato, A

Nitrous oxide is a gaseous substance and has been used for chromosome doubling in red clover. Application of nitrous oxide to the pretreatment of chromosome counting in maize gives excellent preparations with many dispersed countable chromosome figures even in tetraploid maize (Figure 1).

Maize kernels were germinated in wet vermiculite at 25 - 30 C for 2 - 3 days. The germinating seeds with root length less than 1

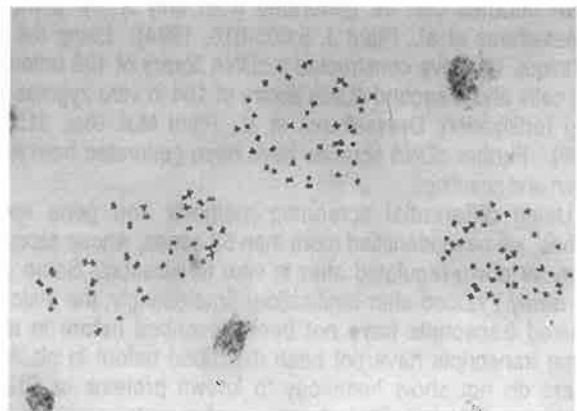


Figure 1. Chromosome figures of a tetraploid maize stock Q-28-1(4n=40).

- 2 cm were used. The excised roots were placed on wet vermiculite in a petri dish and were placed into an air-sealed ion container (10 cm inner diameter, 10 cm depth), and were treated with nitrous oxide gas (8 atm N₂O + 1 atm air) for 3 hr at room temperature (21-23 C). The pretreated roots were fixed in ice cold 90% acetic acid for ten minutes and washed with tap water for five minutes. Root tips (1-2 mm) were excised with a razor blade and hydrolyzed with enzymatic solution (1% pectolyase Y-23, Kikkoman Co., and 2% cellulase Onozuka R-10, Yakult Honsha Co. LTD., dissolved in distilled water) for 90 min at 37 C. Hydrolyzed root tips were extended on glass slide according to the method described by Kato (Biotech. Histochem. 72:249-252,1997).

Preparations with 100 - 350 countable chromosome figures were obtained in diploid maize, and about 50 countable chromosome figures are expected in tetraploid maize according to this method.

IRKUTSK, RUSSIA
Institute of Plant Physiology and Biochemistry

Differential redox regulation by glutathione of translation in isolated mitochondria

--Konstantinov, YM, Subota, IY, Arziev, AS

Recent studies have shown that transcription of some genes (so-called 'redox sensitive genes') is regulated by cellular redox status, especially by thiol-disulfide balance (Sen and Packer, FASEB Journal, 10:709-720, 1996). We have previously reported (MNL 69:63-64, 1995; MNL 70:29-30, 1996; MNL 71:40-41, 1997) on the effect of different redox conditions on mitochondrial genome expression regarding DNA, RNA and protein syntheses in organello. Significant activation of transcription and translation in mitochondria under oxidising conditions and its profound repression under reducing conditions can indicate possible multi-level redox regulation of mitochondrial gene expression.

The aim of the present work was to verify our hypothesis about possible involvement of the glutathione system in the regulation of translational activity in mitochondria.

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. Preparation of mitoplasts was performed using digitonin treatment according to Cristophe et al. (Plant Sci. Lett. 21:181-192, 1981). Protein synthesis in mitochondria and mitoplasts was measured according to the method of Bhat et al. (Biochemistry 21:2452-2460, 1982) with the use of [14C]-leucine (specific radioactivity was 1760 GBq mol⁻¹). Protein synthesis reactions in maize seedling mitochondria were highly sensitive to chloramphenicol (50 ug/ml). The kinetic data were obtained from at least 3 to 4 experiments.

The effect of reduced and oxidized forms of glutathione on the kinetics of protein synthesis in maize seedling mitochondria is shown in Table 1.

The mitochondrial translational activity is seen to increase in the presence of the oxidized form of glutathione while this process is strongly repressed when mitochondria were supplemented by the reduced form of glutathione. Thus, the redox system of such

Table 1. The effect of reduced (GSH) and oxidized (GSSG) forms of glutathione on translational activity in mitochondria of maize hybrid VIR42 MV.

Conditions	Incorporation of [14C]-leucine, % of control			
	5 min	10 min	15 min	20 min
Control	100	100	100	100
Ferricyanide (5 mM)	166	164	164	161
GSSG (5 mM)	108	101	150	145
GSH (5 mM)	65	87	84	80
GSH + GSSG	28	69	56	86

an endogenous thiol compound as glutathione affected pronouncedly the activity of the protein synthesizing system in isolated plant mitochondria.

Table 2 shows the kinetics of protein synthesis in mitochondria and mitoplasts (i.e. mitochondria without outer membrane). We used this model system to study the possible involvement of outer mitochondrial membrane components in the redox signal transduction in mitochondria. It is seen in Table 2 that mitoplasts are also capable of synthesizing the protein but with a lower rate than intact mitochondria.

Table 2. The kinetics of protein synthesis in mitochondria and mitoplasts from maize hybrid VIR42 MV.

Conditions	Incorporation of [14C]-leucine, counts/min/mg protein			
	5 min	10 min	15 min	20 min
Mitochondria	49	679	1241	1635
Mitoplasts	4	202	717	919

The data on the influence of redox conditions on the translational activity in mitoplasts are summarized in Table 3. The effects of potassium ferricyanide and sodium dithionite on the activity of protein synthesis in mitoplasts were similar to those observed for the intact mitochondria. As for the influence of GSH and GSSG on mitoplast translational activity we showed the decrease of such effects in comparison with the whole mitochondria. It is expected from the data given in Table 3 that the outer mitochondrial membrane is involved somehow in redox signal transduction in the case of the glutathione system.

Table 3. The effect of reduced and oxidized forms of glutathione on translational activity in mitoplasts from maize hybrid VIR42 MV.

Conditions	Incorporation of [14C]-leucine, % of control			
	5 min	10 min	15 min	20 min
Control	100	100	100	100
Ferricyanide	184	136	131	118
Dithionite	95	80	73	77
GSSG	140	97	107	108
GSH	129	98	73	77

As a whole, the data obtained suggests that the redox state of glutathione plays an important role in the in vivo regulation of mitochondrial translational activity in plants. We assume also that both transcription and translation factors in plant mitochondria are subject to thiol-mediated redox regulation.

A leucine motif in the amino acid sequence of mitochondrial ribosomal protein S13: possible involvement in protein oligomerization

--Konstantinov, YM, Arziev, AS

Little is known about the molecular mechanism of assembly of ribosomal protein complexes. A leucine zipper motif mediates dimerization of a number of different proteins, including a class of DNA-binding proteins (Landschulz et al., Science 240:1759-1764, 1988). The leucine zipper motif is a sequence of leucine residues spaced every seventh amino acid residue along an alpha-helix. As a result, all the leucine residues of the motif are placed

70

Z.m. msyisgarslpdeqvriastkmdgigpkkaiqlryr L gisgnikihe L tkyqidqieq Miaqdhvvhwe L
T.ae. msyisgarslpdeqvriastkmdgigpkkaiqiry L gisgnikmne L tkyqidqieq Miaqdhvvhwe L
E.s. mxyixgarslpdeqvriastkmdgigpkkaiqiry L gisxnikmne L tkyqidqieq Miaqdhvvhwe L
M.p. msyilgtlnlnsnkqvkiatrlftrigigpkkaiqvcdq L gisdtkivkk L tkyqfdqilk Mmsqnylvdse L
N.t. mlyisgarlvqdeqvriastkidgigpkkaiqvryr L gisgnikike L tkyqidqieq Migqdhvvhwe L

Z.m. krgeradier L isisryrgirhqdgsprrgqrthtnartarkqiwkgnerrlpkeqatd - 129
T.ae. krgeradier L isisryrgirhqdgsprrgqrthtnartarkqiwk - 116
E.s. krgeradier L isisryrgirhqdgsprrgqrthtnartarkooxk - 116
M.p. krviqrdir L isigcyrgfrhnaglprrgqrthtnmakterklryvsirs - 120
N.t. krgeradier L isisryrgirhqdgsprrgqrthtnartarklirk - 116

Figure 1. Leucine motif in amino acid sequence of the rps13 of maize and other plant species. One-letter code is used. Z.m., *Zea mays*; T.ae., *Triticum aestivum*; E.s., *Elymus sibiricus*; M.p., *Marchantia polymorpha*; N.t., *Nicotiana tabacum*. All amino acid sequences are from database EMBL 1993 Year October (release 36.0). Leucine and methionine residues forming motif are shown by capital letters.

along one side of the alpha-helix. According to the zipper model, the leucine side chains extending from the leucine repeat are able to interdigitate with leucine side chains of a second polypeptide which contains the same motif. Therefore, the hydrophobic surfaces of two leucine repeating sequences of the protein molecules might interact to form homo or heterodimers (Landschulz et al., Science 240:1759-1764, 1988; Kouzarides and Ziff, Nature 336:646-651, 1988; Zerial et al., EMBO J. 8:805-813, 1989). The leucine zipper model for protein dimerization is strongly supported by experimental data which show that replacement of leucine at any position of the motif destabilises dimer formation as well as the DNA-binding potential of the protein (Kouzarides and Ziff, Nature 336:646-651, 1988). Leucine zipper motifs have been reported in photosystem I reaction centre polypeptides of higher plants (Webber and Malkin, FEBS Lett. 264:1-4, 1990), glucose-transporter glycoproteins (Buckland and Wild, Nature 338:547, 1989) and voltage-gated Ca²⁺ channels (White and Weber, Nature 340:103-104, 1989). It appears reasonable to assume that the leucine zipper motif is a common mechanism for mediating protein dimer formation in a wide range of different systems when specific interactions are required.

We report here the presence of an unusual leucine motif formed by leucine repeats (-Leu-X10-Leu-) in mitochondrial ribosomal protein S13 of maize and other plant species (Figure 1). This new amino acid motif -L-X10-L-X10-M-X10-L-X10-L- is located at aa positions 37-81. The characteristic feature of the given motif in comparison with the known leucine motifs in proteins of different origin is the disruption of leucine regularity of the motif by incorporation in its composition of another hydrophobic amino acid with a long side chain, methionine, in the position 59 of the amino acid sequence. Protein secondary structure prediction by the method of Garnier (Garnier et al., J. Mol Biol. 120:97-120, 1978) showed that the region including the motif has an alpha-helical conformation (data not shown). Since leucine (including one methionine) 11 amino acids apart will appear on the same side of an alpha-helical wheel, this suggests that this new hydrophobic amino acid motif is perhaps involved in ribosomal protein-protein interaction through helix-helix association by analogy with the leucine zipper motif.

We assume also that this highly conserved amino acid motif may provide specific recognition sites during assembly of the ribosomal supramolecular complex.

JOHNSTON, IOWA
Pioneer Hi-Bred International, Inc.

Molecular analysis of T0 plants transformed by *Agrobacterium* and comparison of *Agrobacterium*-mediated transformation with bombardment transformation in maize

--Zhao, ZY, Gu, W, Cai, T, Tagliani, LA, Hondred, D, Bond, D, Krell, S, Rudert, ML, Bruce, WB, Pierce, DA

Agrobacterium-mediated transformation methods have been used principally in dicotyledonous plants. Monocotyledons are not considered to be natural hosts for *Agrobacterium*. Recently *Agrobacterium*-mediated transformation has been reported in a number of agriculturally important cereal crops, such as rice, wheat, barley and maize. Even though DNA analysis has been used in these transformation studies to confirm stable integration of T-DNA into plant genome, more complete information on the molecular analysis of the *Agrobacterium*-transformed events in monocotyledons is still not available. Those data include T-DNA integration pattern, transgene copy number, the integration of binary vector backbone DNA sequence into plant genomes, transgene expression as well as the direct comparison of *Agrobacterium*-transformed events to bombardment-generated events etc. In this current study, we use maize Hi-II line as a model system to provide the above information. Hi-II embryos were genetically transformed with LBA4404 harboring the super-binary vector created by Japan Tobacco Inc. The regenerated T0 plants were used for these analyses. Microprojectile bombardment of Hi-II embryos was used for comparison. The results indicated that *Agrobacterium* has a number of advantages over bombardment for H-II transformation.

Immature embryos of Hi-II and N6 media were used for *Agrobacterium* (LBA4404) transformation. Super binary vector pPHP10525 (Figure 1) was constructed with pSB1 and pSB11 vectors obtained from Japan Tobacco Inc. The T-DNA of pPHP10525 contained GUS and *bar* genes. The second intron of the potato ST-LS1 was inserted into the coding region of the GUS gene to prevent expression of GUS in *Agrobacterium*. Maize genomic DNA was digested with *SpeI* for GUS integration, with *XbaI* for *bar*, *spec*, *tet* and *virG* integration, and with *SphI* for PTU (plant transcription unit) of GUS and *bar*. GUS protein was assayed with GUS-Light Assay Kit (Tropix, Inc. 47 Wiggins Ave., Bedford, MA01730).

1. *Agrobacterium*-mediated Hi-II immature embryo transformation: Fresh Hi-II immature embryos were transformed with LBA4404(pPHI10525). Stably transformed calli were recovered on bialaphos-containing medium and confirmed with X-gluc staining. T0 plants were regenerated from those calli. With optimized conditions, the embryo-based transformation frequency

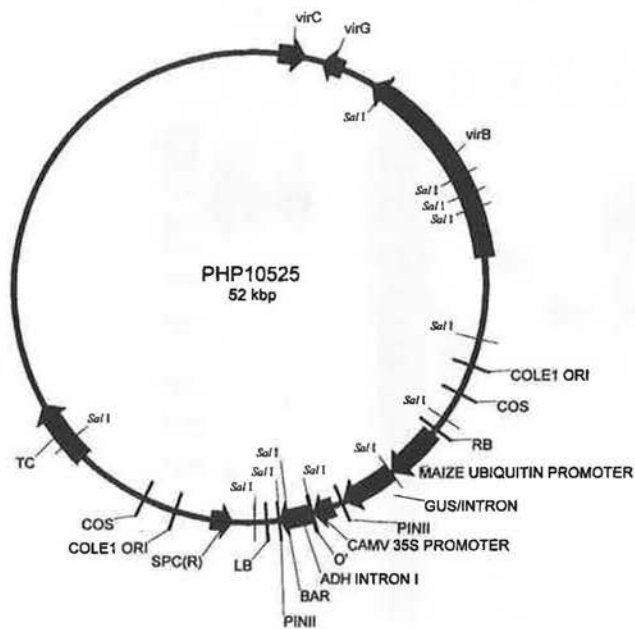


Figure 1. pPHP10525.

Table 1. *Agrobacterium* Hi-II transformation frequency.

Expt. No.	No. of immature embryos		Transformation frequency (%)
	Inoculated	GUS± event	
1	195	64	32.8
2	97	37	38.1
3	65	30	46.2
4	103	52	50.5
Sum	460	183	39.8

can be as high as 32.8 to 50.5% (Table 1).

2. Southern analysis of *Agrobacterium* transformed T0 plants: To understand the molecular characteristics of *Agrobacterium*-mediated maize transformation, T0 plants from 107 embryo-derived events were used in Southern analysis. These T0 plants were divided into two groups as follows: **Group-1:** Single plants from each of those embryo-based events that were transformed with *Agrobacterium* at 1×10^{10} , 2×10^9 , 1×10^9 , 5×10^8 , and 1×10^8 cfu/ml. The purpose of this assay is to determine transgene insertion pattern, copy number and presence or absence of backbone DNA of the binary vector as well as for verifying the effect of *Agrobacterium* concentration on these parameters. **Group-2:** 25 plants derived from 5 embryos (5 plants/embryo), where all 5 plants derived from a single embryo showed the same phenotype for the *bar* and GUS genes; 25 plants derived from another 5 embryos (5 plants/embryo), where the 5 plants showed variable phenotypes for the *bar* and GUS genes. The purpose of this assay is to identify the number of independent events generated from a single embryo. The way these events are defined is described in the following chart. Southern results are listed in Table 2 and 3 and shown in Figures 2, 3, 4 and 5.

Items	LowCopy/Simple Insertion	Multicopy/Complex Insertion
Bands in integration blots*	1-3	>3
Bands in PTU blots*	1 with proper size	>1
Copy No. for both genes	1-3	>3
Rearrangement	no	yes or no
Backbone DNA	no	yes or no

* The blots for both GUS and *bar* genes.

Table 2. Southern results of the events transformed with different concentrations of *Agrobacterium*.

Agrobac. Concn.	No. event	Low/Simple Event %	Multi/Comp Event %	Backbone Event %
1×10^8	12	8	67	4
5×10^8	24	13	54	11
1×10^9	49	30	61	19
2×10^9	14	6	43	8
1×10^{10}	8	5	63	3
Total	107	62	58	45

Table 3. Number of independent events produced from a single embryo.

Embryo No.	Number of independent events	
	5 Plants with same phenotype	5 Plants with varied phenotypes
1	1	2
2	2	2
3	3	2
4	3	3
5	5	4
Total	14	13
Average	2.8 events/embryo	2.6 events/embryo
Sum:	27 events/10 embryos	Multi-event index = 2.7

3. Comparison of *Agrobacterium* transformation with microprojectile bombardment: A number of comparisons were made for the T0 plants derived from *Agrobacterium* transformation and from bombardment transformation in Hi-II. The comparisons include transgene copy number, insertion pattern (Table 5), transgene co-expression (Table 6), and GUS gene activity (Table 7 and Figure 6 and 7). Based on these comparisons *Agrobacterium*-mediated transformation shows a number of advantages over bombardment: (1) a higher proportion of low copy/simple insertion patterns; (2) a significantly higher proportion of events with higher expression levels of GUS. In addition, *Agrobacterium* gives a significantly higher frequency of transformation, compared to our experience with bombardment (33-51% vs. 7-10% of treated embryos).

Table 4. Comparison of DNA analysis results.

Method	Event No.	Low copy/ Simple	Multicopy / Complex
Bomb*	133	8%	92%
Agro.	107	58%	42%

*Bombardment transformation with *Bt* and *pat* PTU

Table 5. Comparison of transgene co-expression.

Method	Events	GUS+ / <i>bar</i> +	GUS- / <i>bar</i> +
Bomb*	17	15 (88%)	2 (12%)
Agro	50	47 (94%)	3 (6%)

* Bombardment transformation with another inbred line.

Table 6. Comparison of GUS activity in T0 plants.

Method	Events	GUS scores		
		+++	++	+ or -
Bomb	14	6 (43%)	3 (21%)	5 (36%)
Agro	43	31 (72%)	6 (14%)	6 (14%)

GUS score +++: =>100,000 units/ug protein; ++: 10,001 to 99,999 units/ug protein; + or -: 0 to 10,000 units/ug protein

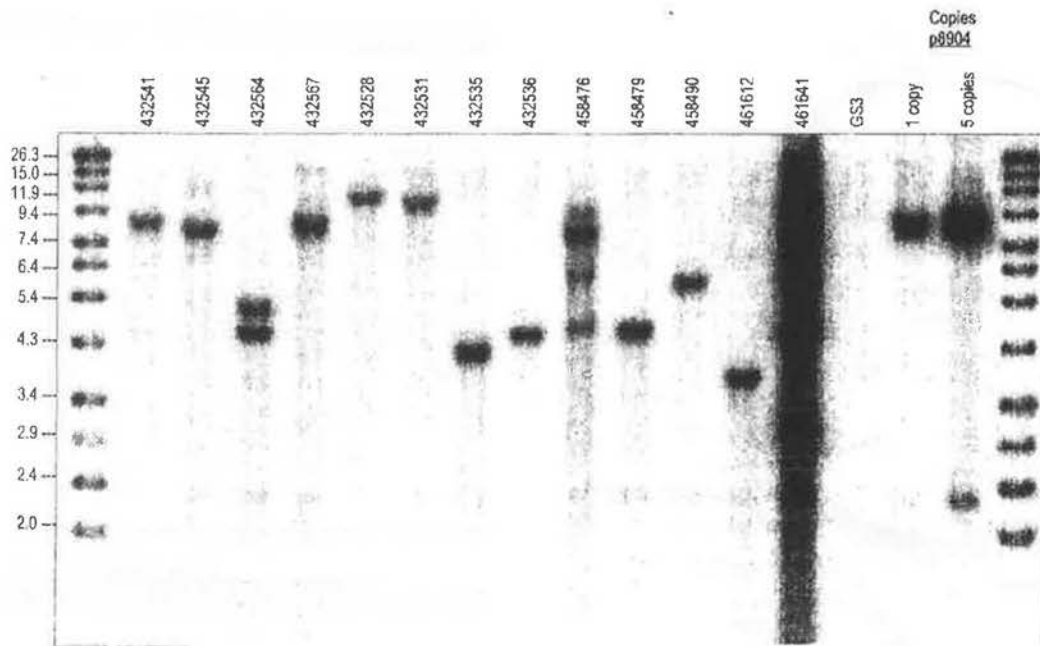


Figure 2. A Southern blot showing presence of the GUS gene.

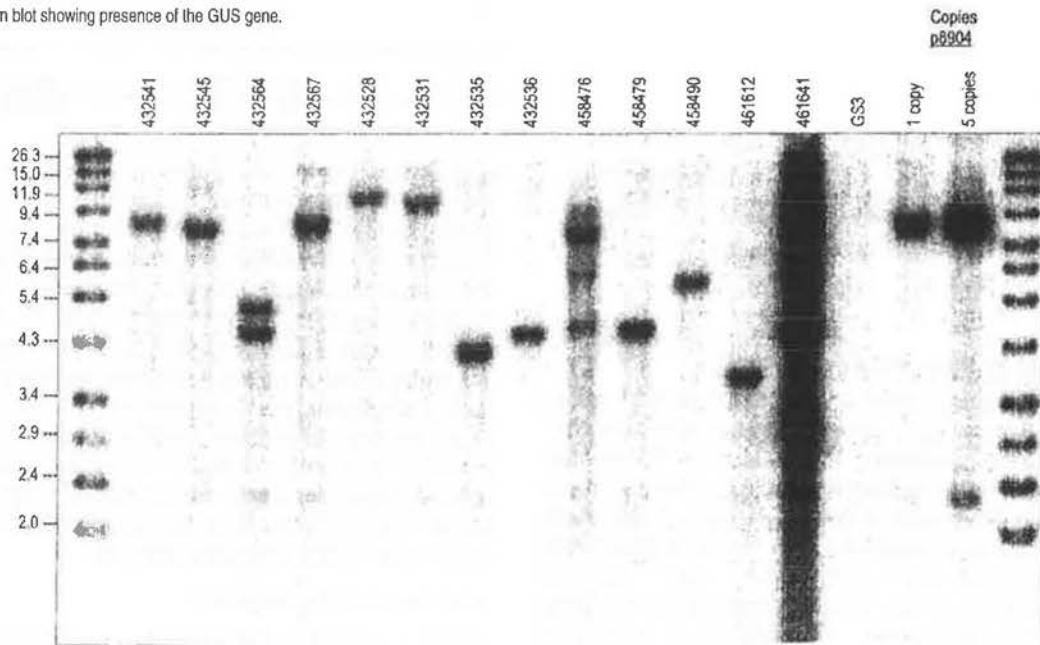


Figure 3. A Southern blot showing integrity of the GUS transcription unit (PTU).

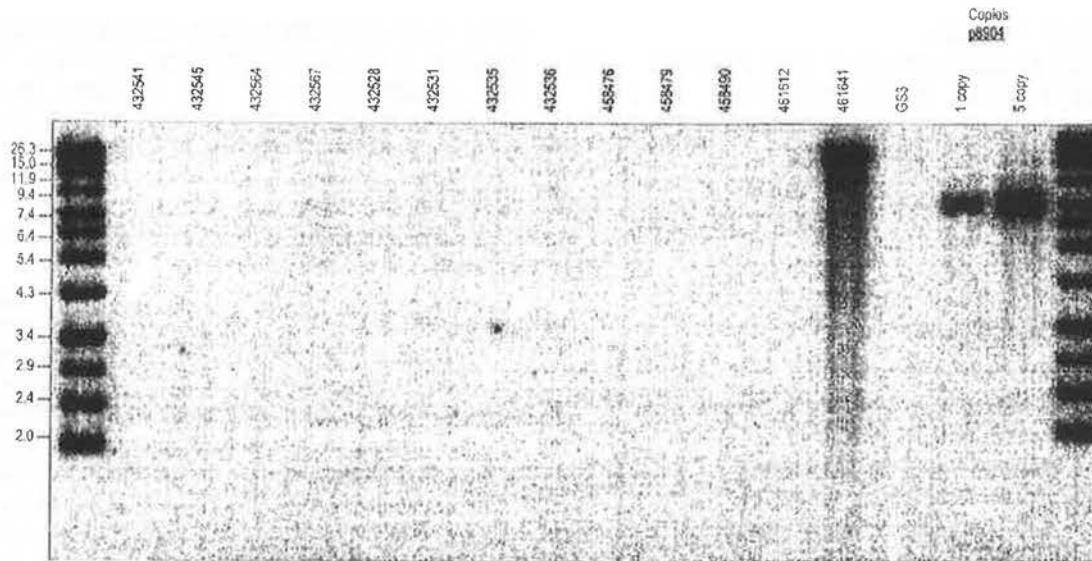


Figure 4. A Southern blot showing presence of vector backbone sequences

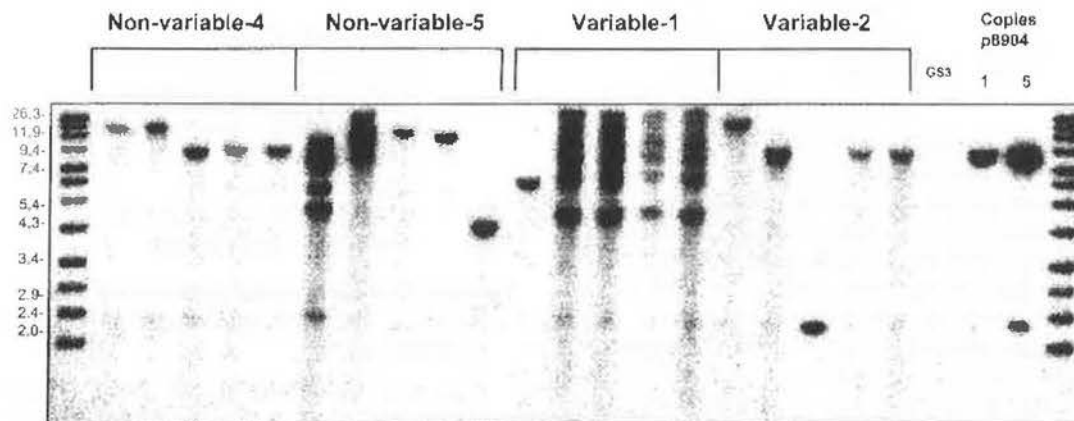


Figure 5. A Southern blot showing multiple events from the same embryo.

In summary, if transformation events with low copy and simple insertion pattern are considered as desirable events, the efficiency of producing desirable transgenic events with *Agrobacterium* would be:

39.8% (transformation frequency) x 2.7 (multi-event index) x 58% (low/simple) = 62 desirable events /100 embryos.

while the efficiency of producing desirable transgenic events with gun bombardment would be:

10% (transformation frequency) x 1.15 (multi-event index) x 8% (low/simple) = 1 desirable event /100 embryos.

New chromosome 10S male-sterile mutant: *ms29*

--Trimnell, MR, Fox, TW, Albertsen, MC

In 1984 a male-sterile mutant was identified in elite proprietary breeding material by Steve Noble in Johnston, Iowa. The male-sterile mutant was out-crossed and selfed. The F2 seed segregated as follows, indicative of a single recessive gene:

Ear Number	Fertiles	Steriles	χ^2
1	4	7	8.76**
2	11	3	0.10
3	10	4	0.10
4	11	6	0.96
5	10	4	0.10
6	12	4	--

**significant at 0.01 level

This male-sterile mutant was designated as *ms^{*}-JH84A*. We crossed *ms^{*}-JH84A* with known genetic male-steriles in 1989 through 1996 to determine allelism. Known male-steriles used in test-crosses were *ms1*, *ms3*, *ms5*, *ms7*, *ms8*, *ms9*, *ms10*, *ms11*, *ms12*, *ms13*, *ms14*, *ms17*, *ms20*, *ms22*, *ms23*, *ms24*, *ms25*, *ms26* and *ms27*. Progenies of these test-crosses were grown from 1990 through 1997. *ms^{*}-JH84A* was not found to be allelic to any of them (at least 20 plants were observed for each test-cross). In 1995, progeny from a segregating F2 ear was grown for bulk mapping the gene. Barb Hobart of our Genetic Markers Lab combined equal amounts of DNA from 26 male-fertile plants and 17 male-sterile plants grown from the F2 ear. DNA blots were constructed using bulked DNA digested with four different restriction enzymes (*Bam*HI, *Eco*RI, *Hind*III, *Eco*RV). These blots were sequentially hybridized with approximately 50 RFLP markers that were evenly spaced throughout the genome. Since *ms^{*}-JH84A* is a recessive mutation, polymorphisms were scored based on the absence of the wildtype band in the male sterile bulk lane. The marker *umc130* on the short arm of chromosome 10 gave a clear polymorphism. Linkage was confirmed by hybridizing DNA from the individuals with *umc130*. This marker segregated as either homozygous "A" allele or as heterozygotes in the male-fertile individuals, whereas all of the male-sterile individuals were

homozygous for the "B" allele.

There are two male-sterile mutations, *ms10* and *ms11*, located on the long arm of chromosome 10. We re-crossed *ms*-JH84A* with both *ms10* and *ms11* and, in each case, verified it was not allelic to either mutant. Because *ms*-JH84A* is not allelic to *ms10* or *ms11*, nor allelic to any of the unmapped known male-sterile genes, it is a new genetic male-sterile mutant. We are designating it as *ms29*.

New chromosome 2L male-sterile mutants *ms30* and *ms31*

--Trimnell, MR, Fox, TW, Albertsen, MC

Two new male-sterile mutants on chromosome 2 have recently been identified. One was observed in 1987 in Willmar, MN by Bob Rosenbrook. It was segregating in an elite proprietary breeding line. Remnant seed from the F3 line was sent to us and planted in 1988. We crossed male-sterile plants from this F3 as female with public inbreds A632 and B73. F1 plants were selfed. F2 segregations were as follows:

Genotype	Fertiles	Steriles	X2 (3:1)
A632 Ear #1	6 Fertiles	8 Steriles	7.71**
A632 Ear #2	7 Fertiles	4 Steriles	0.76
B73 Ear #1	11 Fertiles	6 Steriles	0.96
B73 Ear #2	7 Fertiles	4 Steriles	0.76

**significant at 0.01 level

We designated this male sterile as *ms*-WL87A* and maintained it in both A632 and B73. We crossed *ms*-WL87A* in 1989 through 1996 with the known genetic male steriles. Known male steriles used were *ms1*, *ms2*, *ms5*, *ms7*, *ms8*, *ms10*, *ms11*, *ms12*, *ms13*, *ms14*, *ms17*, *ms20*, *ms22*, *ms23*, *ms24*, *ms25*, *ms26* and *ms27*. Progenies of the test-crosses were grown from 1990 through 1997 (at least 20 plants per test-cross were observed). *ms*-WL87A* was not allelic with any of them. In 1995 a segregating F2 ear was grown for bulk mapping. Barb Hobart conducted the bulk mapping as previously described (see *ms29* article, this issue), except that 20 plants from each fertility class were used in the creation of the pools. Although individual DNA blots have not been run on this family, the RFLP marker *umc4* on chromosome 2L was clearly polymorphic between the two bulks, supporting the hypothesis that *ms*-WL87A* is located on chromosome 2L.

The other male-sterile mutant was observed in 1989. It was segregating in an F2 from an elite breeding cross. We designated this male sterile as *ms*-CG89D*. Male-sterile plants were out-crossed with A632 and then selfed. The resultant F2 seed segregated as follows:

Ear #	Fertiles	Steriles	X2 (3:1)
Ear #1	20 Fertiles	1 Sterile	4.59*
Ear #2	21 Fertiles	2 Steriles	3.26

*significant at 0.05 level

As these results were inconclusive for a single gene, F2 male-sterile plants were backcrossed to A632 and selfed a second time. This resulted in the following segregation, more suggestive of a single recessive gene:

Ear #	Fertiles	Steriles	X2 (3:1)
Ear #1	15 Fertiles	1 Sterile	3.00
Ear #2	10 Fertiles	5 Steriles	0.56

The F1 cross with A632 was used to make test-crosses in 1990 with known genetic male steriles. Additional test-crosses were made from 1991 through 1996. Known male-sterile mutants used were *ms1*, *ms5*, *ms7*, *ms8*, *ms9*, *ms10*, *ms11*, *ms12*, *ms13*,

ms14, *ms17*, *ms20*, *ms22*, *ms23*, *ms24*, *ms25*, *ms26* and *ms27*. Progenies of the test-crosses were grown from 1991 through 1997 (at least 20 plants per test-cross were observed). *ms*-CG89D* was not allelic with any of them. In 1993 a segregating F2 ear was grown for bulk mapping. For this mutant, Robin Tenborg, also of the Genetic Markers Lab conducted the bulk mapping. Twenty individuals from each fertility class were used in the bulked pools. The RFLP marker *umc36*, on chromosome 2L, gave a good polymorphism between the two bulked classes. DNA blots of the individual plants showed 100% linkage of the male sterile lanes with *umc36*. The wildtype individuals segregated normally.

Because *ms*-WL87A* and *ms*-CG89D* were both mapped to the long arm of chromosome 2, crosses were made between the two male-sterile mutants. The resultant crosses were grown, and no male-steriles were found (approximately 80 plants were observed). Because no male steriles have been described on 2L, and because the currently described mutants are not allelic to one another, they are new genetic male sterile mutants. We are designating *ms*-WL87A* as *ms30* and *ms*-CG89D* as *ms31*.

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Two new alleles of the male-sterile mutant *ms45*

--Trimnell, MR, Bedinger, P, Patterson, E, Sheridan, W, Albertsen, MC

Two new male-sterile mutant alleles of *ms45* have been identified. One of the alleles came from a Mutator family received from Bill Sheridan of the University of North Dakota. The other allele is a male-sterile mutant received from Earl Patterson of the University of Illinois.

Bill identified three male-sterile plants in a single Mutator-containing family. Not knowing for certain the allelic relationship of these mutants when we first received them, we designated the mutants as *ms*-BS1*, *ms*-BS2*, and *ms*-BS3*. Several crosses were made between these lines and known male-steriles to determine allelism. Because all of these were single male-sterile plants identified from the same ear row, and because of the results given below, we think that the three male-sterile plants represent the same mutation. In 1994, reciprocal test-crosses were made between *ms45*-9301* heterozygotes and *ms*-BS3* heterozygotes in our Hawaii winter nursery. The resultant progenies were grown in Johnston in 1995 and gave the following results, indicating allelism:

Female	Male	Progeny	
<i>ms45*-9301</i> Het	X <i>ms*-BS3</i> Het	40 Fertiles	6 Steriles
<i>ms*-BS3</i> Het	X <i>ms45*-9301</i> Het	29 Fertiles	11 Steriles

In 1995, test-crosses were made between *ms45*-9301* homozygotes and *ms*-BS1* heterozygotes in our Hawaii winter nursery. The resultant progenies were grown in Johnston in 1996 and gave the following results, again indicating allelism:

	Female		Male	Progeny	
Ear #1	<i>ms45⁻-9301</i> Homo	X	<i>ms⁻-BS1</i> Het	13 Fertiles	17 Steriles
Ear #2	<i>ms45⁻-9301</i> Homo	X	<i>ms⁻-BS1</i> Het	23 Fertiles	20 Steriles

Pat Bedinger at Colorado State University crossed a *ms⁻-BS1* homozygote with a *ms⁻-BS2* heterozygote in 1996. Progeny from this ear were grown in our 1996 Hawaii winter nursery and segregated 11 fertiles and 7 steriles, indicating allelism between *ms⁻-BS1* and *ms⁻-BS2*. These results confirm that all three male-steriles are allelic to *ms45*, and, because of their ear-row origin, probably represent the same mutation. They will collectively be designated as *ms45-BS1*.

The other male-sterile mutant from Earl had been designated as *ms⁻-6006*. Earl had mapped this mutant to the long arm of Chromosome 9 using B-A translocations (see MNL 69:126-128). This mutant had been crossed to *ms2* (located on 9L) and was not found to be allelic. It had not been crossed to *ms45*, however, which also is located on chromosome 9L. We made the test-crosses between *ms45⁻-9301* heterozygotes and *ms⁻-6006* heterozygotes in our 1996 Hawaii winter nursery. The resultant progenies were grown in Johnston in 1997 and gave the following results, indicating allelism:

	Female		Male	Progeny	
Ear #1	<i>ms⁻-6006</i> Het	X	<i>ms45⁻-9301</i> Het	13 Fertiles	6 Steriles
Ear #2	<i>ms⁻-6006</i> Het	X	<i>ms45⁻-9301</i> Het	18 Fertiles	6 Steriles

Microsporocyte samples of this mutant were collected at different stages of microsporogenesis. Anthers were squashed and examined by Jennifer Williams in our lab. Her descriptions of the breakdown events of *ms⁻-6006* matched observations we previously made using *ms45* material. Our new designation for this *ms45* allele is *ms45-6006*.

Are there too many genes in maize?

--Helentjaris, T, Briggs, K

With the development of genomics as a collection of technologies for the study of the genome and individual gene function, cloned and characterized copies of genes are becoming available in unprecedented numbers for many species. In particular the development of the Expressed Sequence Tag (ESTs) approach by C. Venter (Adams et al., Science 252:1651-1656, 1991) coupled with continually-decreasing costs for obtaining sequence data have provided researchers with a cheap and high-volume method for obtaining gene sequence data for literally thousands of genes in species not previously considered as model systems. In this approach, clones are randomly selected from cDNA libraries, prepared from a number of representative tissues and developmental states, and subjected to single pass sequencing, usually from the presumed 5' terminus of the original mRNA. Comparison of the resulting sequence data with entries in public databases is often able to provide a significant similarity indicative of gene function for between 40 and 60% of clones so analyzed. Given the relatively high degree of amino acid sequence conservation of genes across species and even genera, use of gene sequence data gathered by this method for expressed regions provides a method whereby homologs in your favorite species can often be identified for any published gene and function, and sometimes across very distant evolutionary gulfs. In a very real sense, this approach can be viewed as the "Rosetta Stone" for biological research in that DNA sequence data for genes can be used to "translate" an advance in knowledge in one species into

your favorite species by providing researchers with a clone for that gene. Subsequent study of that gene using the clone as a tool can then significantly accelerate the spread of knowledge from model systems to those previously thought to be intractable or impractical to many advanced techniques.

Researchers at Pioneer Hi-Bred are developing a large EST collection from maize and we have been investigating its utility in isolating and identifying maize homologs for genes first described in other species. During this process we have encountered two difficulties with this approach, the problem of isolating ESTs for rarely-expressed genes and the issue of distinguishing related gene family members from actual homologs. By virtue of the fact that mRNAs are expressed at very different rates in different tissues, developmental states, and under different environmental conditions, and given that ESTs are selected from random only from the cDNA libraries that are prepared, it is not surprising that not all genes are easily found by this approach. Nevertheless, given well over 100 cDNA libraries screened and resulting in over 100,000 entries, we were still surprised at our inability to identify maize homologs for many genes already isolated in other species, albeit usually by other methods. For instance, in looking for maize homologs for the following genes involved in the initiation of flowering which we would anticipate to be rarely-expressed and/or only limited to a small number of cells at specific developmental states,

gene name	number of ESTs
<i>constans</i>	1
<i>leafy/loricaula</i>	0
<i>terminal flower/centroradialis</i>	1
<i>luminidependens</i>	1
<i>FCA</i>	0
<i>phyB</i>	0
<i>OsMADs1</i>	12

we have only found convincing evidence for three homologs. Even looking for ESTs for genes already cloned in corn, such as *teosinte-branched*, *terminal ear*, *dwarf3*, and *purple plant1*, we have been unable to identify any with complete identity. While both subtraction and normalization strategies should exert a positive effect upon some aspects of this problem and Pioneer researchers have begun to incorporate them into their library construction protocols, this augmentation still does not address the issues of not being able to economically sample all possible tissues, developmental stages, and environmental conditions and hence may not help in the identification of ESTs for many genes in corn.

Similarly, we have at times had difficulty in distinguishing homologs for some genes first identified in other species, both for rarely-expressed genes and at times even for more abundantly-expressed gene families. Again with genes purported to play a role in the vegetative-to-flowering transition, we identified a number of possible homologs for Arabidopsis gene, *constans*, at the level of possessing significant amino acid similarity. Examination of those which still possessed similarity at the nucleotide level helped to reduce this list further but the one most likely candidate when examined further appeared to lack all of the important sequence elements identified in the Arabidopsis gene and we have been unable to confirm any strong functional relationship to it by other methods. Similar difficulties have been encountered with discerning potential homologs for *luminidependens*. In our examination of genes involved in the lignin biosynthetic pathway, we have encountered similar difficulties,

although these are expressed at much higher levels than the regulators of the flowering process. For instance, at least four and probably more homologs have been found for the 4-coumarate ligase gene, all with relatively high BLAST probabilities to previously described 4CL genes in rice, potato, and soybean. From the expression patterns of these genes, it is not clear which of these actually participate in lignin biosynthesis but may simply possess sequence elements in common with the 4CL gene and biochemical activity towards lignin precursors. Numerous genes for C-OMT, CCoA-OMT, and CAD have also been identified and their actual involvement in lignin biosynthesis has proven difficult to confirm. This phenomenon is not limited to this pathway as we have identified at least one and probably two more sucrose synthases which are expressed in addition to the previously-described *sh1* and *sus1* genes.

In summary, while the EST approach provides an economic strategy for the isolation of clones for genes identified in other species, it is not without complications. Given the large number of ESTs in our database and the portion which seem related but probably not identical to the described genes, one is confused as to just how many genes corn may actually possess? It is possible to calculate a fair estimate for the number of genes in *Arabidopsis thaliana*, from the total genome size divided by the average gene size, which seems close to the average number of genes per kilobase being found by actual genome sequencing. At this time, such a calculation in corn is meaningless given the very large genome size, primarily resulting from the amount of intervening retrotransposon-like sequences in corn. One has to wonder if corn will in fact be limited to that same general estimate even with its duplicate genome structure, given the large number of apparent homologs or related gene family members which we have already identified? It may be that corn not only has enlarged its genome by both the amplification of intervening sequences and polyploidization, but that it additionally may have created many diverged copies of genes with similar functions. Further genome sequencing and evaluation of gene function will be required to better understand this apparent conundrum.

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Experimental mutagenesis of corn

--Lysikov, VN

Studies to explore the possibilities for applying experimental mutagenesis to plant breeding in Moldova go back to the latter half of the 60s. The period coincided with the organization in the Republic of introduction of maize line varieties with a view to developing high-yielding, heterotic hybrids.

As a matter of fact, there was neither scientific nor practical experience in applying the experimental mutagenesis technique to produce new parental material of maize line varieties. This was due to difficulties in working with maize as a cross-pollinated crop and specific features of reproduction of its selfed lines. Besides, one should take into account the level of development of classical genetics at the time, i.e. strict adherence to Johanssen's theory of pure lines, resulting in a number of leading geneticists, including those working with maize, declaring the impossibility of obtaining new mutant lines of maize from its pure lines, such as American

selection lines introduced at the time by the VIR.

In view of the above, work on maize in Moldova had, as it were, to be started anew in order to consistently prove that (1) it was possible, using the experimental mutagenesis technique, to produce new mutant lines from true homozygous selfed lines of maize, (2) new mutant lines would exhibit a range of agronomic traits essential for breeding work, (3) novel mutant lines of maize, reproducing by selfing, would transmit their valuable traits to their progeny, (4) new mutant lines would, upon intercrossing with other selfed lines, e.g. with an American selection line, yield hybrids showing enhanced heterosis.

With respect to the above four points, the new mutant maize lines were clearly shown, on the basis of extensive experimental evidence, to be of considerable interest to breeders. They offer large numbers of valuable gene carriers which not only possess new traits and properties ensuring the broadening of the variability spectrum, but also speed up the development of high-yielding and highly heterotic maize hybrids resistant to adverse environments, diseases and pests.

As a result of this work, a collection of maize mutants was established comprising over 500 mutant lines. Each of these lines differed significantly from their parental stocks and possessed a number of new valuable traits useful in breeding. These mutant lines include forms with shortened growing season, drought-resistant forms, dwarfs showing high grain and silage productivity and forms exhibiting a number of other features essential for applied breeding. Concurrently and in parallel with examination of morphological and physiological traits, mutants were studied in detail with respect to quantitative traits.

Of special interest turned out to be mutant maize lines showing increased grain protein content, including those forms which exhibited high content of amino acids, such as lysine, tryptophane, methionine, etc. Sometimes, these forms contained no less and even more lysine than the forms carrying the *opaque2* (*o2*) gene. It is known that the transfer of the *o2* gene into maize lines encountered great difficulties since together with the *o2* a number of genes linked to it were being transferred which were undesirable and even harmful in terms of breeding. For example, the "*ramosa2*" (*ra2*) gene causing ear separation, and others. Mutant lines exhibiting high content of essential amino acids do not have these undesirable genes.

A collection of promising mutant lines of maize was established and a catalogue published describing and characterizing over 500 forms. Mutant lines from our collection were freely distributed to breeders in Russia, the Ukraine, Kirghizia and other regions of the former Soviet Union (FSU). This enabled plant breeders at various institutions to use the lines in their work and, more importantly, identify among them those which showed valuable traits and properties in that particular locality and to attempt their further improvement.

Thus for example, 125 mutant lines from our collection were donated to the All-Union Maize Research Institute in Dnepropetrovsk (the Ukraine). A detailed study of these under the environmental conditions of the central Ukraine enabled Prof. G. V. Grizenko to identify 21 forms resistant to root rot. Root rot resistant maize lines did not exist until that time even in the VIR collection. We could not identify these forms ourselves since in Moldova the disease was not expressed at all due to aridity.

Similar encouraging and interesting results were obtained when our mutant maize lines were used to identify forms resistant

to various maize diseases. This work was carried out by Dr. E. N. Kobeleva, a plant pathologist, at the Zhrebkovskaya experimental station (the Ukraine). She even published a monograph concerned with the problem.

A study by Z. A. Orinshtein specifically aimed at inducing, by experimental mutagenesis techniques, resistance in maize lines to boil smut (*Ustilago zaeae*), a major maize disease in Moldova, also merits special attention. Since nobody had previously succeeded in obtaining such mutations, the experiment involved maize forms highly sensitive to boil smut under normal conditions, including the GelberLand-Mais cultivar. All the work was performed with artificial infection, such that each plant was recurrently inoculated at the growing point with germinated spores of boil smut. It turned out that treatment with a combination of ionizing radiation and Rapoport's supermutagens allows maize forms to be identified which are almost completely resistant to boil smut. A test for heritability of this resistance carried out by assistant professor K. I. Kuporitskaya of the Plant Pathology Department at the Kishinev Institute of Agriculture showed that the most resistant forms (genotypes) consistently transmit boil smut resistance from generation to generation for 7 to 10 years.

Current breeding and seed-growing work aimed at producing interstrain hybrids of maize is performed on material showing cytoplasmic male sterility (CMS) thus eliminating the need to cut the tassels away from female parent plants and improving the quality of hybrid seed material. This led academician A. E. Kovarsky to explore the possibility of developing by experimental mutagenesis new mutant lines of maize exhibiting CMS. The experiments (by A. I. Konotop, Yu. S. Orlov and S. G. Byrka) very soon allowed identification of 9 mutant lines of maize of which 8 showed CMS and 1 nuclear sterility. All lines exhibiting CMS resulted from treatment with a combination of ionizing radiation and chemical super mutagens, the line showing nuclear sterility having been produced by sonication.

A subsequent test for type of sterility, which was performed using indicator lines, showed that we did generate maize lines carrying CMS, of the Moldavian type at that, the so-called S type. Among these, the mutant line derived from line VIR-49 appeared to be a very promising one, since no such line with CMS existed in previous collections. At the time, the line proved to be very useful to breeders in producing new maize hybrids. A special study of this line by A. F. Pali, one of the post-graduate students (now a professor) of the corresponding member of the Academy of Sciences of Moldova T. S. Chalyk, provided a complete support to CMS of the Moldavian (S) type having been generated by experimental mutagenesis.

Considering the fact that maize mutant lines themselves are not used directly for harvesting (except in seed production) and only interstrain hybrids are used for major plantings, an objective was set to study new mutant lines for combining ability. In other words, it was necessary to demonstrate the possibility of using mutant lines in breeding work to produce high-yielding hybrids, especially as there were no such data in the world at the time.

The formulated problem was addressed along two lines simultaneously. On the one hand, a large number of mutant lines of Moldavian selection were examined for general combining ability (GCA) by the classical topcrossing technique and on the other hand, best mutant lines in diallel crosses were studied for specific combining ability (SCA) with subsequent mathematical treatment of the results according to the method of Griffing and Khotyleva.

The experimental evidence of O. V. Blyandur and V. G. Mordvinova clearly showed that mutant lines of maize can exhibit both high GCA and high SCA.

Concurrently, the same problem was solved in somewhat different but very ingenious way by A. I. Konotop. He chose mutant lines from among classical American selfed lines of maize comprising the double interstrain hybrid VIR-42 the most common among the introduced maize lines in Moldova at the time. Derived from these lines were first simple mutant hybrids of the "Slava" type (VIR44 x mutant VIR38) female parent of the double hybrid - and the simple hybrid of the "Svetoch" type (VIR40 x mutant VIR43) - the male parent of the double hybrid. Then these simple mutant hybrids were used to produce a double hybrid of the VIR42 type (mutant Slava x mutant Svetoch).

A comparison by A. I. Konotop of the double hybrid of the VIR42 type, derived from a cross of mutant lines, and simple hybrid VIR42 showed the former to be undoubtedly superior to the latter. Thus, it was demonstrated that even a "simple assemblage" of the double interstrain hybrid VIR42 performed according to the traditional scheme but involving mutant maize lines results in the already high-yielding, highly heterotic maize hybrid showing a considerable, mathematically provable yield increase.

Thus, experimental mutagenesis was definitively shown to be a promising tool for producing increasingly high-yielding heterotic hybrids from maize lines. This was subsequently very well confirmed by work on developing new high-yielding interstrain maize hybrids which were introduced into cultivation and came to occupy vast acreages. The academician V. V. Morgun and Dr. I. G. Chuchmii (Ph.D. in agriculture) have been successfully working along these lines in the Ukraine. In Moldova, this work has been rather successfully carried out by Dr. O. V. Blyandur.

In addition to producing usual types, we have paid attention to generating large mutational changes, the so-called macromutations. In macromutations, variability usually affects a large number of important systemic characters. Identification and isolation of a macromutation has always been a very rare event. Moreover, its study encountered numerous difficulties, since not infrequently it was necessary to determine a somewhat unusual pattern of expression of characters and their inheritance which may often be complicated by incomprehensible manifestations.

Therefore, when in 1962 an original radiation macromutation, "Corngrass", was obtained following irradiation of pollen of the VIR44 maize line with gamma rays in a dose of 15 Gry, it immediately became the focus of attention. This was a very stunted (40 cm), bushy, herbaceous plant totally different from normal maize plants and, besides, completely lacking the male reproductive organs. In order to preserve this mutation, whose female reproductive organs were located in axils (two pistil filaments at each leaf), it was pollinated with a mixture of pollen from different maize lines including that from VIR38 and VIR43.

An unusually intense morphogenetic process occurred during the second and subsequent generations. Among the resulting forms, many exhibited marked, complex changes in plant morphology, such as a greatly increased number of stems, partially altered stem (even becoming geniculate in shape); altered internode positions; changed leaf morphology and phyllotaxis; significantly altered male and female reproductive organs; etc.

Many mutant plants showed a clear-cut and pronounced expression of some of the characters of putative ancestral forms

of maize - teosinte, gama grass (*Tripsacum*), and even Job's tears. Prof. F. M. Couperman of Moscow State University reported the case of simultaneous expression of characters of the above three ancestral forms of maize.

Studies of diversity of the "Corngrass" mutation progeny enabled Dr. A. N. Kravchenko to classify them morphologically. He subdivided all the forms into five categories: cultivated homozygotes, cultivated heterozygotes, teopodal forms (resembling mutations *Tp1* and *Tp2*), corngrass forms (resembling the *Cg1* mutation), and branched.

Long-term observations showed that the morphogenetic process was very intensive over 25 years, with only brown and teopodal groups making the largest contribution to diversity and serving, as it were, as a source of new forms. Furthermore, it was shown that the entire progeny of the radiation-induced macromutation "Corngrass" segregates into stable, or readily stabilized upon selfing, and unstable forms which, upon long selfing, continuously segregate not only known forms, but completely new ones as well. It was obvious that a mutation exhibiting genetic instability had been obtained.

It is only recently that the properties and features of genetically unstable mutation have begun to be studied. This was due to a number of reasons, such as: first, the previously known cases of genetic instability were discovered by chance, as spontaneous mutations; second, in the majority of cases they occurred in Protozoa rather than in plants belonging to higher organisms; third, before the molecular bases of modern genetics were developed, the very explanation of possible genetic instability, as well as description of well defined cytogenetic factors such as those by Barbara McClintock had not been accepted as serious ones and sometimes even questioned by most researchers.

It is for this reason that many experimenters, having encountered such cases in their work, exercised caution, being reluctant to waste time on studying the yet unclear phenomenon, and simply ignored them. Other researchers restricted themselves to their brief description, "registering" only what lied on the surface and required no large effort to be verified. Still others, while realizing that some new phenomena may be behind this but fearing being misunderstood by others, thought they were not in a position to undertake serious studies along these lines. And finally, there were those who did not know how to start an in-depth study of this phenomenon. The state of knowledge at the time was not high enough to enable this.

Extensive studies by Dr. N. V. Krivov, of the Institute of Genetics (Kishinev), on the pattern of behavior and expression in the progeny of traits characteristic of the Corngrass mutation using the genetic analysis technique allowed a number of important conclusions to be made. Of these, the following are worth mentioning:

1. This mutation was found to be determined by a dominant gene, designated *Cg2*, and to be nonallelic to the known American mutation *Cg1* (corngrass). It was shown to be located in the short arm of chromosome 3.

2. The expression of the *Cg2* gene is strongly dependent on modifier genes, which allows this mutation to be regarded as highly sensitive to the genetic background.

3. A specific character of the *Cg2* mutation is a peculiar discontinuity in its phenotypic expression. Thus, a proportion of plants strongly resembles the expression of macromutation of the

Tp1 and *Tp2* (*teopod1* and 2) type, another proportion closely resembling the *Cg1* macromutation. In addition, many chimeric (mosaic) plants are identified which carry, on a single plant, shoots of both mutant corngrass, or teopodal, and cultivated type.

4. A remarkable feature of this mutation is its genetic instability. Moreover, it has been found that the instability mutation can occur not only in *Cg2* ---> normal (+) but in the normal (+) ---> *Cg2* direction, i.e. regular transitions of the mutant-normal type occur. The *Cg2* --> normal (+) mutations have been shown to occur at a rate of more than 55% in homozygous *Cg2/Cg2* plants and at a rate of 15-17% in heterozygous plants. Furthermore, the frequency of the *Cg2* allele in somatic cells, measured as a proportion of mosaics among the selfed homozygotes, is of the order of 28%. It has also been found that the *Cg2* allele in phenotypically normal plants mutates with a higher frequency in somatic cells (5.5-14%) than in generative cells (2.0-2.5%).

Individual comparative analysis of the behavior of hetero- and homozygotes up to generation 6 has shown that (1) in the progeny of heterozygotes, the segregation ratio corresponds to that of the monohybrid type, i.e. 3:1, but in this case nearly always there is an excess of the normal (+) phenotype and a deficit of mutants (*Cg2*), (2) in the progeny of *Cg2/Cg2* homozygotes, solitary normal plants frequently occur. This, in turn, confirms the *Cg2* gene instability, since frequent *Cg2* ---> normal (+) reversions result in the generative tissue variegation, leading to the appearance, along with homozygous *Cg2/Cg2* cells, of the *Cg2*/normal (+) heterozygotes. And this, in turn, results, on selfing, in a proportion of plants being of the normal, (+)/(+), type.

That the above mutation is unstable is also evidenced by genotypic differences between progenies of two different ears harvested from a single plant. The same is actually indicated by high frequency of mosaic plants. And finally, this is particularly obvious from the analysis of data on the genotypes of mutant and normal shoots of the same mosaic plant.

5. Another significant feature of the *Cg2* mutation is its pronounced mutator activity. An intensive morphogenetic process is observed over 25 generations when various maize forms are crossed with this macromutation. In a number of cases, it was even possible to speak of the *Cg2* gene occasionally inducing a kind of outburst of mutator activity. In cases like these, it could even be called a "biological mutagenic factor".

In a number of elegant and carefully performed experiments, Dr. N. V. Krivov obtained six monogenic recessive chlorophyll mutations, one recessive mutation resulting in leaf necrosis, one mutation phenotypically resembling the *ramosa* (*ra*) mutation, and one mutation with abnormal development of vegetative plant organs. In addition, two mutations arose upon crossing the *Cg2* with marker lines. One of these was allelic to *Japonica* (*j*), the other being linked to markers on two chromosomes and designated as *Zebra7* (*Zb7*).

All the above facts can be very well explained in terms of insertional mutagenesis. It is by insertions of mobile genetic elements (TEs) capable of carrying a peculiar kind of "DNA genetic punctuation marks" such as promoters, enhancers, terminators, etc. that changes in gene activity and their reflection in the phenotype can be explained. This can provide explanations for multiple allelism occurring in the progeny of *Cg2*, frequent allelospecific transitions and just new mutations arising at the loci

sometimes far removed from the *Cg2* gene locus itself.

Of course, one cannot help mentioning that the *Cg2* mutation exhibits a property rarely found with insertional mutagenesis, the one under which a TE insertion results in a dominant rather than recessive mutation. However, here, as with insertional mutagenesis, the original type of phenotypic expression is partially or completely restored upon excision of the TE from the *Cg2* locus.

While classical works of B. McClintock and other authors show the genetic instability in maize to be characteristic of only those genes which determine the variegated coloration and the structure of the kernel, studies of the radiation-induced macromutation *Corngrass2* discovered in Moldova have shown for the first time that genetic instability of the *Cg2* gene is expressed at the earliest stages of zygote division and is capable of strongly affecting the structure of both vegetative (somatic) and reproductive organs resulting in an intensive morphogenetic process.

Since McClintock's experiments were substantiated by molecular techniques, it can be assumed in our case that the *Cg2* gene also possesses the property of the so-called "jumping gene", or transposon and, due to this, exhibit the features of a "genetic mutagen". It is only in this way that the "explosion" ("blast") of the morphogenetic process observed over the years can be explained. Therefore, studying specific features of the radiation-induced macromutation *Cg2* by molecular biological techniques is becoming a very promising approach. Studies like these are already under way at the Institute of Genetics, Academy of Sciences of Moldova (Kishinev).

Even if the *Cg2* gene is not assigned to the category of mobile genetic elements as a result of the studies now in progress, its role as a new, unusual mutator system in maize calls for closer attention and further studies. This is needed in order to understand the theory of evolution of the maize plant and for practical breeding involving this mutator system as a new, powerful driving force of the morphogenetic process aimed at producing new parent stocks of maize.

Concluding the description of the radiation-induced macromutation of maize, "*Corngrass2*", it should be mentioned that in 1986 the Maize Genetics Cooperation Newsletter included the *Cg2* gene (i.e. our "*corngrass*" mutation) in the list of new unlocalized genes of maize.

Another crop which proved to be a very promising one in terms of application of experimental mutagenesis techniques was the garden gladiolus, *Gladiolus hybridus* hort. Gladiolus is, in itself, a valuable and interesting flower crop to which new genetic methods were previously inapplicable. It is in this Institute that Dr. A. V. Murin launched and has been carrying out serious research on the gladiolus genetics. This work has yielded an extensive and valuable new parent material for applied breeding. Unfortunately, gladiolus has a number of features posing difficulties to obtaining new parent material by experimental mutagenesis techniques. May be it is for this reason that there has been relatively little literature on the subject. Some of the factors complicating work with gladiolus are as follows:

- 1) the complex and, probably, interspecific hybrid origin of cultivated gladiolus; although this origin of gladiolus may be regarded as its remote past, it may have occasional and unusual effects in the present as well;

- 2) the tetraploid condition which, presumably, was absolutely

necessary in the past for genome stabilization, but whose peculiar features have to be reckoned with at present;

- 3) relatively large number ($2n=60$) of small chromosomes with karyotype features scarcely studied in detail;

- 4) a complex triennial life cycle.

However, gladiolus has some properties that make it a convenient organism to work with. Thus, two modes of reproduction - sexual and vegetative - occur in this plant. This enables the experimenter to preserve and maintain in the progeny any variability present in the parents by switching from sexual to vegetative reproduction. Another equally useful property is the possibility of producing plants with chimeric tissues, since subsequent radiation-induced production of non-chimeric tissues greatly accelerates the breeding process.

A task was set to produce new valuable parent material for breeding, using advanced genetic techniques, primarily those of experimental mutagenesis and experimental recombinogenesis. Use of a set of chemical mutagens based on substances discovered and synthesized by Prof. I. A. Rapoport as well as irradiation of seeds, pollen, bulbs, and bulbils with ionizing radiation resulted in the production of extensive original material.

In doing so, highly decorative, disease and pest resistant forms showing good transportability were produced which, in addition, exhibited the degree of variability not found in the cultivated gladiolus. The entire range of forms obtained can be classified as four new types of gladioli:

- 1) fragrant gladioli having the following odors: flower-scented, citrus, carnation, rose, coffee, etc.;

- 2) remontant, i.e. capable of regrowth and of producing a second and, occasionally, even a third flower after the first one has been cut;

- 3) double-flowering, carrying flowers with a large, sometimes very large, number of spikelets;

- 4) moire flowers with petals of motley coloration which occasionally looks like an ornament or fringe.

As a result, an extensive collection of new parental stocks numbering over 6000 specimens was created. Of these, 500 forms were displayed at various exhibitions, including international ones, where they won various prizes. Two varieties were introduced into cultivation in Moldova.

Thus, application of experimental mutagenesis in Moldova showed this technique to be a promising tool both for elaboration of theoretical problems of genetics and for practical breeding.

Chromosomal abnormalities in maize regenerants in R1

--Kravchenko OA, Lysikov, VN, Palii, AF

We have previously reported (MNL 71:42-43) on the effect of gamma radiation on callus formation and somatic embryogenesis. It was found that the influence of gamma radiation was reliable and resulted in decreasing of callusogenesis and somatic embryogenesis frequency. We are now extending these studies to plant regenerants and the R1 generation. The experiment was carried out as follows: seeds of regenerants obtained from immature irradiated (4 Gr) embryos of inbred line 346 culture were germinated. Rootlet samples were fixed in 3:1 ethanol:acetic acid and standard 2% acetocarmine squash preparations were made to study and calculate various chromosomal abnormalities. Cytological analysis demonstrated that bridge and fragment formation as well as chromosome lagging was observed during anaphase and telophase of mitosis and occurred with variable

Table 1. Chromosomal abnormalities of regenerants in R1.

Types of abnormalities	Frequency, %
bridges	1.34-3.11
fragments	0.91-3.89
lagging	2.86-14.81

frequencies (Table 1). It should also be noted that the maximum lethality proved to be among regenerants with a higher chromosomal abnormality frequency, especially in early stages of ontogenesis. In addition, male gametophyte viability of regenerants that reached flowering stage was 70.73%-72.51%. The results obtained indicate that chromosomal abnormalities induced by gamma radiation appear to remain at a rather high level in culture conditions in vitro and among regenerants in R1.

Dependence of some characters of the maize male gametophyte on gamma radiation and genotype

--Kravchenko, OA

The aim of the present study was to find out whether genotype and gamma radiation affect pollen morphological and cytochemical characters. For this purpose dry seeds of inbred line 346 and its waxy counterpart were irradiated (100 Gr) before sowing. For analysis in the light scanning microscope "Morphoquant" permanent preparations of mature pollen were made using a technique developed by Kravchenko, A.N. (In: Recombinogenesis in Evolution and Breeding, Kishinev, p.264-265, 1986). Such characters as area, perimeter, eccentricity (the ratio of the biggest diameter to the smallest one), DNA quantity (in relative units) and chromatin dispersity of vegetative and generative cell nuclei were evaluated. The data were processed by two factor analysis of variance which revealed some interesting results.

Morphological characters of vegetative cell nuclei were mostly genotype-dependent and the presence of the *wx1* gene resulted in decreasing of area, perimeter, and eccentricity (Table 1). The same characters of generative cell nuclei as well as DNA quantity of vegetative cell nuclei were affected by gamma radiation factor. It should also be noted that variations with gamma ray treatment had higher indices of these characters (Table 2). DNA quantity and chromatin dispersity of generative cell nuclei proved also to be influenced by factors studied. In such a case the waxy counterpart had higher indices of these characters, in particular, chromatin dispersity was 1.5 as much. The values of cytochemical characters of generative cell nuclei affected by gamma radiation were also found to be increased. Thus, these data indicate that the effect of gamma radiation and genotype was reliable and contributed to variability of male gametophyte morphological and cytochemical characters.

Table 1. Variability of morphological and cytochemical characters in dependence of genotype.

	+/+	<i>wx1/wx1</i>
	vegetative cell nuclei	
PERIM	40.79*	39.23*
AREA	83.58**	75.49**
EXT	1.46**	1.39**
SDENS	11.90	11.11
DISP	2.66**	3.33**
	generative cell nuclei	
PERIM	19.41	18.59
AREA	14.02	13.81
EXT	3.51	2.99
SDENS	3.25***	3.66***
DISP	14.23**	21.92**

*p<0.05, **p<0.01, ***p<0.001

Table 2. Effect of gamma radiation on morphological and cytochemical characters of male gametophyte.

	control	100 Gr
	vegetative cell nuclei	
PERIM	38.7***	41.3***
AREA	75.0**	84.0**
EXT	1.43	1.43
SDENS	11.00*	12.0*
DISP	2.83	3.15
	generative cell nuclei	
PERIM	18.37**	19.63**
AREA	12.84**	14.98**
EXT	3.32	3.36
SDENS	3.31**	3.62**
DISP	16.75**	19.39**

*p<0.05, **p<0.01, ***p<0.001

Abbreviations: PERIM - perimeter, AREA - area, EXT - eccentricity, SDENS - DNA quantity, DISP - chromatin dispersity

Possibilities for identifying major genes controlling quantitative traits using maternal haploids of maize

--Chalyk, ST, Chebotar, OD, Bylich, VG, Chernomorets, AA

Identification and use of major genes controlling quantitative and qualitative traits have always been of great importance in maize breeding. The employment of diploid plants for this purpose is often fraught with difficulties. This is due to the fact that in segregating populations most of the plants exhibit heterosis and are not comparable with homozygous parental lines. To overcome these difficulties, use of maternal haploids has been suggested (Chebotar and Chalyk, 1996). Employment of matroclinal haploids makes genetic analysis much easier and helps in identifying individual major genes controlling these traits.

The objective of the present study was to explore the possibilities of using matroclinal haploids for ascertaining the number of genes controlling plant height by which maize lines 092 and A619 are differentiated. To produce maternal haploids from lines 092 and A619 and from hybrids between these (092 x A619 and A619 x 092), a haploid-inducer line was used. The resulting haploids were examined in the field. The experiments were performed during 1993, 1994 and 1996. In 1993, the environmental conditions during vegetation were favourable: good rainfall was accompanied by moderate air temperatures. The years 1994 and 1996 differed significantly from 1993: they were very hot and dry. In 1993, it was found that a proportion of haploids derived from hybrids were considerably superior in plant height to haploids produced from parental lines. The occurrence of transgressive haploids suggested that parental lines were differentiated by no less than two genes for plant height. The genes of line 092 were arbitrarily designated as A1B1 and those of line A619 as A2B2.

Of interest are transgressive haploids A1B2 and A2B1. These were superior in plant height to parental genotypes A1B1 and A2B2. If the interaction between genes A and B were additive, then transgressive haploids would occur on each side of distribution of haploids derived from parental lines. However, in the segregating population of haploids derived from hybrids no plants were found which would be inferior in height to haploids produced from parental lines. Therefore, genes A and B were assumed to show the additive x additive epistatic interaction.

Applying the method of Powers (1951), theoretical distributions of all the four genotypes in the segregating population - A1B1, A2B2, A1B2, and A2B1 - were calculated. Summing up the frequencies of these genotypes, allowing for the

fact that the proportion of each of them in the population was 25%, made it possible to calculate theoretical distributions of haploids derived from the 092 x A619 and A619 x 092 hybrids. Comparison of the observed and expected distributions showed a good agreement between the two, suggesting that lines 092 and A619 are indeed differentiated by two major genes which exhibited epistatic interactions in 1993.

In 1994, lines 092 and A619 were also found to be differentiated by two major genes concerned with plant height. However, the pattern of plant-height distribution of haploids was somewhat different from that in 1993. This altered distribution appears to have resulted from differential drought resistance of transgressive genotypes A1B2 and A2B1. One of the transgressive genotypes (this can be arbitrarily taken to be A1B2) was, as in 1993, superior in plant height to haploids derived from parental lines. However, plants of the other transgressive genotype, A2B1, shifted from the right to the left side of the distribution. It may be suggested that the epistatic interaction of genes A2 and B1 is unstable and that it is lacking completely under adverse environmental conditions. Therefore, selection of genotypes carrying precisely genes A1 and B2 may be of prime importance in maize improvement.

The severe drought of 1994 prevented us from producing enough seed for carrying out an experiment in 1995. The experiment, therefore, was continued in 1996. There was a severe drought again in the first half of the summer in 1996, resulting in the distributions of haploids being virtually the same as in 1994. Parental lines were found to be differentiated by two major genes. Transgressive genotypes A1B2 and A2B1 were distributed nearly symmetrically in relation to the haploids derived from parental lines A1B1 and A2B2. One transgressive genotype, presumably A2B1, exhibited the lowest plant height. Another transgressive genotype, probably A1B2, was on average superior in plant height to the haploids derived from parental lines. The best A1B2 plants were quite appealing as candidates for breeding. They were pollinated with pollen of diploid plants from a synthetic population, SA, which is targeted for improvement.

Unfortunately, we have no possibility to use molecular markers for mapping genes A1 and B2. Collaboration with researchers from other scientific centres will hopefully assist us in solving this problem.

In conclusion, it is hoped that the use of maternal haploids of maize will prove a useful tool in identifying major genes concerned with traits of breeding value.

In our experiment, two major genes were identified which strongly affect plant height in maize. The number of genes responsible for the trait in question showed no variation over years according to environmental conditions, but the gene interaction varied. In the "favourable" year, the genes showed additive x additive epistatic interaction, whereas the segregation frequency of haploids observed under severe drought conditions is attributable to additive gene interactions.

A new mutator system?

--Krivov, NV

Test-crossing of *sh wx* marker stock (as ear parent) with stocks isolated in the progeny of the heterochronic mutant *Cg2* resulted in *Wx-m* mosaics in the F1 among the *Wx/wx/wx* kernels. The *Wx-m* kernels had single brick-red specks in a dark blue background. The frequency of *Wx-m* kernel origination in two

experiments was 13.45×10^{-2} and 9.0×10^{-2} on average for families (MNL 1992). In F2 *Wx-m* kernels appeared with a frequency of 5.25×10^{-2} . Reliably significant deviations from the theoretically expected ratio of 3:1 were observed in some families. In total 64,168 kernels with the *Wx/wx/wx* genotype were examined, *Wx-m* kernels being revealed in 4099 of them, i.e. the frequency of appearance of these kernels averaged 6.4×10^{-2} . However, in individual families, the mutation frequency reached 29.0×10^{-2} and even higher.

The origination of *Wx-m* kernels in such a frequency and the pattern of the distribution of the mutant *wx* sectors is, as a rule, associated with the excision of the transposable element (TE), located proximally to the *Wx* locus in the chromosomes introduced from the plants tested. Therefore, the segregation of the *Wx/wx* heterozygotes is accompanied not only by the origination of *Wx-m* kernels but significant deviation from the ratio of $3Wx : 1wx$.

Saturation crossings, in which the *wx sh* marker stock was used as an ear parent, were carried out during recent years. As a result, a segment of the 9th chromosome, located in the nearest proximity to the *Wx* responsible for the appearance of *Wx-m* kernels, was introduced into the genetic background of the marker stock that did not contain complete copies of the regulatory elements (MNL 1995). Crossings with the test stocks *a-dt(Dt)*, *c-m(Ac)*, *a-m(En)*, *a-ruq(Uq)*, and *a-mrh(Mrh)* have been performed this year. The test results appeared to be negative for all five mutator systems *Dt*, *Ac*, *En*, *Uq* and *Mrh*. In other words, the sub-stock carrier of the *Wx* allele does not contain complete copies of the regulatory elements *Dt*, *Ac*, *En*, *Uq* and *Mrh* any more, but the *Wx-m* kernels continue to appear with a frequency of 10^{-2} in most families.

Tests for allelism between *ys* mutants differing in origin

--Krivov, NV, Lysikov, VN

A number of mutations, including *zb*-143*, *zn*-143*, *al*-143*, and *j*-220*, have been isolated during the genetic examination of the heterochronic mutation *Cg2* (MNL 1992). The latter is allelic to *j1* on 8L. Chlorophyll mutations, with longitudinal yellow stripes on leaves that are phenotypically similar to *ys*, are constantly appearing during reciprocal hybridization of some substocks. The tests for allelism have shown that *ys*-308*, *ys*-309*, *ys*-310*, and *ys*-253* are allelic to each other (Table 1). Both *ys*-308* and *ys*-309* are allelic to *ys3* on 3L, but *ys*-310* and *ys*-253* are not. Only one allele, *ys*-9196* from the *ys br* stock, turned out to be allelic to *ys* on 5L.

The high frequency of this mutation appearing in the genetic background of stock carriers of the *Cg2* and *Cg2+* alleles might be associated with either the activity of the mutation systems that are widely used in transposon tagging (*Ac* and *En*) or a new mutator system, the TE (transposable element) of which is located on the short arm of chromosome 9 in sites adjacent to the *Wx* locus.

Table 1. Tests for allelism between different *ys* alleles.

Mother	Father	<i>ys1</i>	<i>ys3</i>	<i>ys*-253</i>	<i>ys*-307</i>	<i>ys*-308</i>	<i>ys*-309</i>	<i>ys*-310</i>
<i>ys*-9196</i>		+	-	-	-	-	-	-
<i>ys1</i>								
<i>ys*-308</i>		-	+	+	-	*	-	-
<i>ys*-309</i>		-	+	+	-	*	*	+
<i>ys*253</i>		-	-	*	-	+	+	-
<i>ys*-310</i>		-	-	-	-	+	+	*

+ allelic; - nonallelic

The upset of meiosis at maize pollination with high dose gamma-irradiated pollen

--Romanova, IM

Induced mutagenesis is one of the promising techniques in maize breeding. Mutations can result from the pollination of plants with high dose gamma-irradiated pollen, the latter being a mutagen which provides a wide range of variegated mutations.

Cytological studies constitute an important component part in genetic breeding programs involving the development of new original forms with the aid of the methods of experimental mutagenesis. A normal course of meiosis is one of the factors which determines a stable status of the forms. Abnormal reductional divisions can lead to deviations in offspring contributing to the variability of traits in higher plants.

No studies on the indirect action of high gamma-irradiation doses via irradiated pollen have been conducted and the consequences of this treatment have not been studied in subsequent generations. Given the destabilization of meiosis in experimental plants, the effect of the irradiated pollen on morphologic process might be expected in maize.

The recipient stock P-346, and pollen of the donor marker stocks K167 *bm2*, *su1 s4 bm2 g1*, Doina, and Doina *br2* were taken as objects of the cytological studies.

The examination of meiosis in maize microsporogenesis showed that the reductional division was occurring in it, mainly, in a proper way. All stages of early (leptotene, zygotene, pachytene) and later prophase 1 are present. The normal parity of chromosomes ensures meiotic regularity. Ten bivalents, including seven of the closed type and three open, are observed, which then line up along the equatorial plate in metaphase 1 and separate normally (10 + 10) to both poles in anaphase 1.

Cytological examination of the first generation samples of stock P-346 treated with the irradiated pollen of the donor stocks K167 *bm2*, Doina, Doina *br2*, *su1 s4 bm2 g1* discovered no structural mutations. However, chlorophyll mutations from pollination of the recipient with the K167 *bm2* pollen irradiated with doses of 500, 800, and 1000 Gr, and a mutation of the barren stalk type, as well as of the golden type when the pollen was irradiated with 800 Gr suggests the existence of point mutations in the samples under study. This is proved indirectly by the absence of sterile pollen in these mutants.

A small number of second generation plants exhibited deviations from the norm in reduction division following pollination with high dose irradiated pollen K167 *bm2* in the dose range between 500 and 1000 Gr.

A relative synchrony is normal for the first meiosis in the maize anther. Some asynchrony is observed only during the second division. Treatment with irradiated pollen causes the initiation of meiotic asynchrony as soon as prophase 1. At the stage of metaphase 1 the origination of univalents, chromosome acceleration, and early chromosome divergence are observed in some cells of the form studied. Examination of meiotic anaphase 1 revealed lagging of bivalents.

The pattern of meiosis during microsporogenesis upon this treatment is as follows: in many cells, univalents were formed at the stages of metaphase, anaphase and telophase of both divisions. In most cells, they are well-discernible in diakinesis at this stage, and from one to four univalents can be scored in different meiotic cells. The number of anomalous tetrads increased to $24.31 \pm 0.86\%$ against $3.41 \pm 0.17\%$ in the control. Due to this, the

sizes and shape of the pollen grains varied strongly, the pollen appeared to be viable only in half-- $41.54 \pm 0.20\%$ in comparison with 89.72 ± 0.38 in the control.

In the control plants, meiosis mainly had a normal course, with insignificant deviations having no effect on the pollen quality. They are characterized by the regularity of tetrads, evenness of pollen grain size and high fertility.

Cytological examination of the second generation samples produced through the pollination of the stocks Doina and Doina *br2* with the high dose irradiated pollen revealed no structural changes in chromosomes except the sample produced through pollination with the Doina *br2* pollen irradiated at a dose of 300 Gr. This sample was characterized by structural changes at all stages.

At the stage of metaphase, from one to three univalents could be counted in different meiotic cells. In some cases, a delay of bivalent divergence occurred, and lagging bivalents were observed in the first and second anaphases. By the end of the meiotic division, two-nucleolus pollen was observed with a frequency of $5.01 \pm 0.41\%$, not seen in the control.

Unforeseen disorders were found in the second generation of P-346 pollinated with highly irradiated *su1 s4 bm2 g1*. The disorders started from prophase 1. Unexpectedly frequent, in comparison with the control, chromosome breaks occurred at the doses of 300, 800, 1000 Gr with a frequency of $15.8 \pm 1.52\%$, $20.34 \pm 1.81\%$, and $36.51 \pm 2.41\%$, respectively. Terminal deletions, chromosome nondisjunction, and frequent chromosome breaks at various irradiation doses were marked during diakinesis. Chromosome lagging and individual fragments were observed in metaphase 1 and anaphase 1, respectively. In the samples at the dose of 1000 Gr, along with frequent chromosome breaks, upset of nucleolus formation with a frequency of 1.83% and heteromorphic bivalents with a frequency of 0.96% were observed during diakinesis due to the different lengths of the conjugating chromosomes. These types of upsets are characteristic of meiotic mutants with disorders in spiralization and partial asynapsis.

Further development of microspores varied owing to anomalies in meiosis. Different stages of degeneration were marked in a part of microspores, the pollen fertility was quite low in the experimental plants and oscillated from 46.82% to 64.73%. In the control plants, the pollen remained highly fertile, 90.12-94.54%. The abnormal meiosis in maize suggests that pollination with the high dose irradiated pollen upsets the coordinated program of the occurrence of meiotic processes, which leads to the production of anomalous kernels or a specific level of sterility of maize plants, both male and female ones. The indirect action of the ionizing irradiation via the high dose irradiated pollen results eventually in the fixation of a considerable number of mutational changes in the genotype with aid of both point and structural mutations.

On the possible mechanism of 6-mercaptopurine and laser radiation-induced DNA breaks and chromosome aberrations in *Z mays*

--Burilkov, VK, Paschenko, VM, Lysikov, VN, Basova, IA

The combined action of sensitizers (S) and laser radiation (LR) on eukaryotic and prokaryotic DNAs has been analyzed in a number of works. The sensitizers used were ethidium bromide (EB), acridine orange (AO), and 8-methoxypsoralen (8-MOP). It has been shown that the possible mechanisms of break formation

are those of radical formation, two-quantum affine modification of DNA bases, and others.

The objective of the present study was to examine the possible mechanism of DNA breakage and of chromosome aberration induction resulting from exposure to a new sensitizer, 6-mercaptopurine (6-MP), and LR (Burilkov et al., MNL 71:44-45, 1997). We have recently identified a sensitizer which, in combination with LR, enhances the mutagenic activity of AO, EB and 8-MOP by several fold. This is 6-mercaptopurine.

We have compared cytogenetic effects of the known sensitizers, AO and EB, and of previously unused ones, such as 6-mercaptopurine (6-MP) and Cloroxine (CX), each used in combination with laser radiation (LR). The studies have shown that the highest rate of chromosome aberrations occurred when 6-MP was used as a sensitizer. This exceeded the rates of chromosome aberrations resulting from exposure to EB+LR, CX+LR, and AO+LR by factors of 1.5, 4, and 8 respectively.

It has been suggested that one possible reason for multiple chromosome aberrations resulting from exposure to 6-MP and LR may be the formation of numerous additional breaks in maize genomic DNA due to laser energy which is transferred from a sensitizer molecule to certain DNA regions. During mitosis, these breaks may be repaired or they may become chromosome aberrations. To test the proposed hypothesis, genomic DNA of maize was studied by gel electrophoresis. Electrophoretic patterns in non-denaturing conditions and break counts from densitograms have shown our hypothesis to be a plausible one.

As a next step, it was deemed necessary to study some possible causes of DNA break formation on exposure to 6-MP and LR. One possible mechanism contributing to the induction of breaks could be that of radical formation. Indeed, the EPR spectra have confirmed the presence of radicals in DNA following its exposure to 6-MP and LR and their absence under no irradiation and with irradiation but in the presence of 2-mercaptoethanol (2-ME) known as a radical quencher.

However, the mechanism of DNA break induction due to radical formation is, in this case, not the only one. This is supported by electrophoretic patterns of DNA preparations exposed to LR in the presence of various concentrations of 2-ME. It is apparent that starting with a concentration of 0.1 M 2-ME, the number of DNA breaks is not reduced but, at the same time, it is considerably higher than that in the control samples. It should be noted that no radicals were present in the EPR spectra at a concentration of 0.1 M 2-ME.

Further and more detailed studies are needed to be able to provide an answer to the question of what other mechanisms contribute to the formation of DNA breaks. Part of the answer may be various mechanisms of DNA-S complex formation. At present, mechanisms for the formation of complexes of DNA with AO, EB, and 8-MOP are known. Intercalation of molecules of the indicated sensitizers between DNA base pairs results in some physicochemical characteristics of DNA (melting temperature, absorbance maximum, and others) being altered, which is consistent with our findings. For the DNA-6-MP complex, the melting temperature differs insignificantly from that of the DNA alone, and there is no shift in the maximum value of the absorption spectrum. These findings suggest that 6-MP, while interacting with DNA, alters insignificantly its structure and conformation, since 6-MP is similar in its structure to the DNA=D4 to a great extent, with the LR wavelength, DNA breaks can be supposed to

be formed due to a direct transfer of the LR energy to certain, adenine-rich DNA regions.

In our view, the in vivo transfer of energy to the sugar-phosphate skeleton of DNA to form breaks and subsequent transformation of these into mutations can proceed according to two schemes:

- 6-MP, being homologous to adenine, forms complementary binary and ternary complexes with it in the sites of complete unwinding. LR drives the DNA-6-MP complex into an excited state which, in turn, results in DNA breaks and induces chromosome aberrations;

- during semiconservative DNA synthesis, 6-MP, being an adenine analog, can replace it at some sites, resulting in a modified DNA structure. In the sites of 6-MP localization in DNA, LR, on being absorbed by the sensitizer, induces breaks which may be repaired or may become chromosome aberrations.

In an in vitro system (DNA preparations and 6-MP), the first pathway seems to be more plausible.

LACOMBE, ALBERTA, CANADA
Field Crop Development Centre

Xenia assisted reciprocal recurrent selection in maize

--Wolfe, RI

Reciprocal recurrent selection (RRS), a breeding system in which two populations undergo repeated cycles of selection based on performance of hybrids between them, has been shown to be highly effective for improvement of a number of characteristics, including yield. However, the necessary hand crossing, record keeping, and the time generally needed to complete a full RRS cycle, limit the power of the method.

Maize, being naturally open pollinated, with the male and female portions of the plant separated, is easier than most species to cross by hand. Nevertheless, if one could retain the selective power of RRS in maize while eliminating most of the hand crossing and record keeping, and if one could shorten the RRS cycle time to one year, it should be possible to substantially improve efficiency.

This may be possible, using xenia to identify pollen source. A possible term for such a breeding method is, Xenia Assisted Reciprocal Recurrent Selection (XARRS).

There are two major forms of visible xenia in maize, aleurone color and type of endosperm. With the best choice from the species of a genetic marker from each type, it may be possible to follow pollen movement from each of two RRS populations, while maintaining effective population identity or integrity.

To make XARRS work, two populations must be set up, one homozygous, or nearly so, for a dominant aleurone color, and homozygous for a recessive shrunken endosperm. Pollen from this population must produce seed identifiable by color on the second population. This second population would be homozygous for a recessive counterpart of the colored allele in the first population, and homozygous, or nearly so, for the dominant starch-producing allele. Pollen from this population must produce normal starchy seeds on the first population.

The ideal for XARRS would be to have only one major gene of each xenia type in the breeding material under RRS, each gene with only two alleles, and all four seed types easily distinguishable. That is, there would be a color gene, with two alleles such as yellow and white, and a gene conditioning seed filling, such as normal

starch and sugary, *su*. Even if one could get such, it would probably prove to be important to select strongly against any modifiers that appear in the two populations.

These two populations would be grown together and allowed to intercross. Some of the resulting inter-population hybrid seed, recognizable as double dominants, would be grown out and tested. The rest of the seed from each plant would be kept in reserve. Information gained from testing would be used to rank the plants from which the seed was obtained. Intra-population seed from the best of the reserve plants would be used to form the next crossing block. If the crossing block were to be grown at a suitable winter increase site, one XARRS cycle could be completed per year, without any hand crossing in this part of the breeding program.

If the two marker genes, with two contrasting alleles each, function well, it would not be necessary to set the populations up with absolute, or even extremely high levels of homozygosity of the two dominant markers. Some consequent mixing between the two populations would be good for their evolution. However, if mixing produces complicated segregations of seed color, more attention would have to be paid to starting the two populations out highly homozygous for their respective dominant markers. To maintain or improve the level of homozygosity, seed of plants harvested from the crossing block having any double recessive seeds would be discarded.

Several XARRS cycles should build up considerable improvement in performance of inter-population hybrids. The world's best germplasm could be incorporated into both populations on a continuing basis.

A small back-crossing program, using the best material as recurrent parents, would be run to eliminate markers that are unacceptable for the intended use of resulting hybrid cultivars. With this being done where considered necessary, elite material from each population would be selfed to produce inbreds. Hybrids for commercial release would then be developed between the inbreds of the two populations, using standard maize breeding procedures. At some point, hopefully sooner rather than later, the best of such hybrids should begin to outperform the best commercial hybrids available from other sources.

Within an XARRS breeding program, a possible set of designations for seed types and the two populations would be:

- "Dash" for Dark-shrunken seeds, and the population designated by them

- "liSt" for light-Starchy seeds, and the population designated by them

- "DkSt" for the Dark-Starchy inter-population hybrid seed

Over the past three years I have been working on setting up genetic material for XARRS in barley, where two genetic male sterile genes, one in each of the two populations, are necessary to force out-crossing.

During this period I have also been trying to demonstrate the method in maize, where genetic male sterility is not needed. Our environment at Lacombe is not suited for commercial production of field corn. However, in sheltered locations over the past several years it has been possible to get some viable seed on the earliest maturing plants.

In 1996 I grew out some blue-starchy seeds obtained from yellow sweet corn cobs, mainly of the local early maturing cultivar Sunnyvee. These seeds had been produced from pollen of other maize plants growing nearby. I had been planning to use yellow and

white as the color markers, but found that the color contrast sometimes virtually vanished. Upon seeing the rather strong xenia effects on sweet corn of pollen, carrying a gene for a dark blue aleurone color, I decided that blue seed might contrast better with yellow than the contrast of the yellow-white combination.

This past year, 1997, I planted some of the blue-shrunken seeds from 1996 in rows adjacent to a number of yellow starchy lines, including LX511, an early maturing hybrid from Dr. Malcolm MacDonald, Lethbridge, Alberta, and some material received from Dr. Bob Hamilton at Ottawa. These were designated, 'Ble d'Inde Gaspé' flint and sugar, 'Brandon Research Centre Synthetic', and 'V360 Spanish Flint'. The Gaspé material was the earliest of any in the crossing block.

Cobs of plants from both the yellow-starchy and blue-shrunken seed planted in 1997 had some blue-starchy kernels. These were the result of inter-crossing of the two seed types. Some of these blue-starchy seeds are being grown out in Chile this winter, 1997-1998, and are to be selfed. It will be interesting to see how many, if any, of the resulting plants have a definite one-gene segregation for color.

The segregation of seed color on the plants from the blue-shrunken seed in 1997 was quite variable. It was obvious that there was more than one gene influencing color. Consequently, it is possible that the white-yellow color combination will work better. If this latter color pair is to be used, selection of the palest "white" white-starchy and the most strongly yellow yellow-shrunken seed for the XARRS crossing block might keep the color modifiers at bay.

LLAVALLOL, ARGENTINA

Instituto Fitotecnico De Santa Catalina

Early waxy and high quality protein maize inbreds: study of fungal diseases

--Corcuera, VR, Sandoval, MC

Since 1990 at the Institute a maize breeding plan to obtain high quality protein and waxy commercial hybrids useful for industrialization procedures, has been carried out. The characterization of the original populations, as well as the inbreds developed from them, through morphological, biochemical and resistance traits, is necessary and important to make a correct description and evaluation of the materials. It is interesting to detect the response of the different maize genotypes to pathogens in an early stage of their selection whilst inbreeding. In this paper, a summary about fungal diseases affecting maize in the Southern area of the Province of Bs. As. is presented.

Thirty-seven early maize inbreds were sown at the experimental field in a randomized block design with three replicates. Each pedigree was placed in a 7m² plot using a density of 71,500 plants/ha. No fungicides were applied to the seeds nor to the field. Over 1,273 individual plants were scanned for the appearance of fungal diseases. The identification was done in the field and later confirmed at the lab through isolation, culture and histology techniques.

Puccinia sorghi (Schw.) was the most important pathogen detected. It appeared in all the maize genotypes at a medium degree of infection (grade 2, rust disease scale). By decreasing order of incidence on maize plants, the following pathogens were also observed: *Kabatiella zaeae*, *Sclerospora sorghi* (Kulk),

Table 1. Percentage of diseased waxy maize inbred plants produced by 8 different fungal pathogens.

Inbred	A ¹	B ²	C ³	D ⁴	E ⁵	F ⁶	G ⁷	H ⁸
54	45.00	-	10.00	-	-	-	10.00	-
58	43.75	-	-	-	12.50	-	-	-
63	87.50	-	-	-	-	12.50	-	-
64	38.46	53.48	-	-	7.69	-	-	-
65	83.33	16.66	-	-	-	-	-	-
71	100	-	-	-	-	-	-	-
72	87.50	-	-	12.50	-	-	-	-
73	90.00	10.00	-	-	-	-	-	-
74	50.00	-	-	-	-	-	-	50.00
75	72.22	-	-	-	-	-	-	5.55
76	61.90	-	-	-	-	-	-	28.57
77	63.63	22.72	-	-	-	-	-	-
78	25.00	-	-	-	-	-	-	25.00
79	25.00	-	-	-	-	-	-	75.00
147	42.85	-	10.71	3.57	-	-	-	-
146	61.53	-	-	-	-	-	-	-
141	87.57	-	-	3.03	-	-	-	-
138	94.73	-	-	5.26	-	-	-	-
134	77.27	-	9.09	-	-	-	-	-
129	50.00	10.52	-	-	-	-	-	-
128	61.53	2.56	2.56	-	2.56	5.12	-	-
127	57.89	-	5.26	-	-	-	-	-
126	56.66	-	6.66	3.33	-	-	3.33	-
83	60.00	-	-	-	20.00	-	-	20.00
85	88.23	-	11.76	-	5.88	-	-	-
87	60.00	-	-	-	-	-	-	6.66
90	13.33	13.33	-	-	40.00	26.66	-	0.15
80	100	-	-	-	-	-	-	-
151	96.29	3.70	-	-	-	-	-	-

Ref: ¹ *P. sorghii*; ² *H. turcicum*; ³ *H. maydis*; ⁴ *U. maydis*; ⁵ *S. sorghii*; ⁶ *C. lunata*; ⁷ *K. zeae*; ⁸ *P. maydis*.

Table 2. Percentage of diseased plants produced by 8 fungal pathogens on opaque2 maize inbreds.

Inbred	A ¹	B ²	C ³	D ⁴	E ⁵	F ⁶	G ⁷	H ⁸
152	33.33	33.33	33.33	-	-	-	-	-
145	62.50	-	12.50	6.25	-	-	-	-
133	100	-	-	-	-	-	-	-
132	84.20	-	5.26	-	-	5.26	5.26	-
131	64.00	-	-	-	-	12.00	4.00	-
130	100	-	-	-	-	-	-	-

Ref: ¹ *P. sorghii*; ² *H. turcicum*; ³ *H. maydis*; ⁴ *U. maydis*; ⁵ *S. sorghii*; ⁶ *C. lunata*; ⁷ *K. zeae*; ⁸ *P. maydis*.

Physoderma maydis (Miyabe), *Ustilago maydis* (DC) Corda and *Curvularia lunata*. About *Ustilago maydis* it has to be said that produced not only the classic tumours or galls in the ears, but in 5 inbreds also promoted the appearance of male spikelets in ears and tassel-seed.

Zea mays inbreds resistant to different populations of *Ustilago maydis* (DC) Corda

--Sandoval, MC, Corcuera, VR

Ustilago maydis (Um) has produced epidemic disease in Argentina. This behaviour depended upon the maize varieties and hybrids under cultivation, as well as environmental factors and the degree of virulence and genetic stability of the different forms of the pathogen. Rosbaco (1949) pointed out that 100% of the sweet corn grown that year in our country was attacked and Godoy (1950) reported that 14% of the maize grown in Argentina was affected by this pathogen during that season. Since then, the incidence of Um was not registered, except in maize hybrids and inbreds grown at the Institute Santa Catalina since 1989.

With the single purpose of determining the behaviour of eleven maize inbreds included in a breeding plan to obtain high quality commercial hybrids useful for different industrial procedures, pathogenicity studies were carried out, using 3 populations of Um

(DC) Corda.

Eleven maize inbreds (see Table 1) were sown in pots and kept in the field. The pathogen material consisted of teliospores proceeding from spontaneous infections in Santa Catalina, Balcarce and Rio Cuarto. The teliospores were cultivated in 2% APG, and transferred to a liquid culture medium (2% CPG) and then incubated under constant agitation at 25 C until raising a concentration of 2×10^6 spores/mycelium/ml.

Table 1. Maize inbreds inoculated with different populations of Um.

Inbred	Description
3008/ 2/1	waxy
3008/2/6	waxy
3009	dent
3011	flint
3012	waxy
3012/8	waxy
3016/10/1	waxy
3016/5	waxy
3033	dent
3070	flint
3071/3	flint

Once the maize plants were at the 5 leaves stage, they were inoculated using a hypodermic syringe. The inoculation was practised by puncturing the meristematic apex (Hirschhorn, 1986) to introduce the different populations of Um. The trial consisted of 3 replicates and a tester inoculated with CPG for each inbred analyzed. A pot with 5 plants is considered a replicate.

Five days after inoculation, observations and data collection were initiated. Data obtained were analyzed through ANOVA. The determination of the degree of infection was done using the scale detailed in Table 2 (Sandoval, 1997). The statistical analysis showed no significant differences in the behaviour of the pathogen populations studied.

Table 2. Scale used to determine the pathogenicity degree of Um populations based on the host plant response.

Population	Host Reaction
Avirulent	No infection - 0
Scarcely virulent	Slight chlorosis on leaves and stems with or without anthocyanins. Preferentially on leaves - 1
Mid virulent	Severe chlorosis with necrotic spots on leaves; negative effects on development - 2
Virulent	Gall formation - 3
Very virulent	Severe attack with death of plants 6 days after infection - 4

About the degree of infection, only the inbred 3012/8 showed severe attack (grade 4). In this case the classical tumors (galls), as well as dead plants were observed. The other inbreds studied only showed slight attack (grade1) with hypersensitivity reactions. Because of this, these inbreds can be classified as resistant or immune (grade 0) to Um. This behaviour of the inbreds studied when inoculated with the three populations of Um suggests the existence of resistance in these maize genotypes, which will then be useful for the breeding plan under development.

General combining ability of flint, waxy and dent maize early inbreds

--Corcuera, VR, Sandoval, MC, Naranjo, CA

The characterization of the foundational materials as well as their inbreeding generations through the utilization of multiple descriptors is useful and important to describe and evaluate them. Nevertheless, it is not enough to select amongst different genotypes, being necessary to submit them to field trials capable

of determining the ability of yielding offspring distinguished by the high expression of their yields when they are used in hybrid combinations. This measure constitutes the general combining ability (g.c.a.) of the genotypes under study. Inbred development could be considered as a sequential process in which the worst genotypes are removed in the initial stages because of their performance or they can also be removed later based on specific combining ability tests.

During November 1996 at the Institute a 7 x 8 lattice design with 4 replicates was sown to analyze the performance of 43 single crosses between an equal number of flint, waxy and dent early inbreds developed since 1990/91 and two heterozygote testers. The crossings to obtain the F1 plants tested through this trial, were practised during the growing season 1995/96. The testers used were Z8340 and Z8543, and tests were evaluated during both seasons (1995/96 and 1996/97).

The list of participants as well as their inbreeding generation and endosperm type is shown in Table 1. The average yield for testers and each test-cross is presented in Table 2. As these values constitute a measurement of the inbreds' general combining ability (g.c.a.), all those yielding over the average of the tester used were selected. According to this, the 43 inbreds analyzed were divided into two groups, depending on the tester to which they were crossed. Inbreds showing good g.c.a. when crossed to Z 8340 are: 3011/5 (flint), 3012/6/1 (waxy), 3012/9 (waxy), 3016/7/1 (waxy), 3033 (dent), 3033/2 (dent), 3040/2 (flint), 3043/1 (flint), 3070/3 (flint), 3070/7 (flint), 3071 (flint) and 3071/3 (flint). On the other hand the inbreds denoting good g.c.a. with Z 8543 are: 3008/2/1 (flint), 3008/2/4 (flint), 3011 (flint), 3012/6/2 (waxy), 3012/8 (waxy), 3016/1 (waxy) and 3040/2 (flint). This points out that 18 inbreds of the whole analyzed, show good g.c.a., yielding more than 5,700 or 6,700 Kgs kernel/Ha depending on the tester to which they were crossed.

Yields were analyzed through ANOVA-2 (Table 3). There are statistically significant differences among blocks (replicates) and treatments (participants). There is a difference of 9.86% between the average yields of blocks 1 and 3. Blocks 2 and 4 show similar average yields.

Table 1. List of participants in the g.c.a. trial.

Nº	TC(A)	Gn	Type	Nº	TC(B)	Gn	Type
4	3016/7/1	S3	waxy	1	3008/2/	S3	flint
8	3014/3/1	S2	waxy	2	3012/	S2	waxy
9	3016/7/1	S3	flint	5	3016/5/3	S4	waxy
10	3016/5/3	S4	waxy	6	3008/2/4	S3	flint
11	3043/1	LE	waxy	7	3012/8	S3	waxy
14	3033/2	S3	waxy	12	3012/6/2	S3	dent
15	3016/5/3	S3	flint	13	3016/1	S2	waxy
16	3012/3	S2	waxy	17	3008/2/1	S3	waxy
18	3020/3	S3	waxy	25	3011	S2	flint
19	3016/1/0	S2	flint	31	3103	S4	flint
20	3012/6/1	S3	waxy	32	3104	S4	flint
21	3011/5	S2	flint	37	3071/1	S3	flint
22	3012/9	S3	dent	42	3071	S2	flint
24	3033/4	S3	flint	44	3070/3	S3	flint
26	3027	S2	flint	45	3033/2	S3	dent
29	3033	S2	wx/o2	46	3009	S2	dent
30	3033/3	S3	wx/o2	47	3013	S2	waxy
34	3070/7	S3	flint	49	3040/2	S2	flint
38	3071/3	S3	flint	50	3014	S2	waxy
39	3070/3	S3	flint				
40	3071/4	S3	flint				
43	3071	S2	flint				
48	3040/2	S2	flint				
53	3033/2	S3	dent				

3, 23, 27, 28 y 33: plots of the tester Z8543
35, 36, 41, 51 y 52: plots of the tester Z8340

Ref.: TC(A): Test-crosses with Z 8340. TC(B): Test-crosses with Z 8543. Gn: Inbreeding generation. Type: Endosperm type of the inbred tested.

Table 2. Results of the g.c.a. trial expressed as yield in Kgs/ha.

Participant	Average ²	S.D.	Variance	C.V. %
1	5,358	17.29	299.10	32.28
2	5,102	7.15	51.15	14.02
4	8,404	14.10	198.84	22.00
5	5,203	9.27	86.03	17.83
6	7,400	5.43	29.52	7.34
7	9,745	9.91	98.26	10.17
8	4,678	7.90	62.40	16.89
9	4,606	5.22	27.27	11.34
10	5,151	5.84	34.17	11.35
11	8,538	10.16	103.27	11.90
12	7,366	16.14	260.50	21.91
13	7,088	7.55	56.98	10.65
14	7,014	3.20	10.27	4.57
15	4,722	6.27	3.93	13.28
16	4,891	2.22	4.93	4.54
17	7,882	7.39	54.59	9.37
18	5,547	16.17	261.45	29.15
19	5,524	7.25	52.51	13.12
20	7,111	40.09	1606.90	65.37
21	6,434	3.90	15.23	6.07
22	5,791	12.47	155.53	21.53
24	5,254	8.67	75.14	16.50
25	7,574	4.46	19.93	5.90
26	4,533	9.38	88.04	20.70
29	6,370	10.24	104.79	16.07
30	5,168	12.16	147.78	23.52
31	6,200	17.76	315.29	27.92
33	8,628	10.80	116.76	17.43
32	5,623	15.73	247.39	18.23
34	6,361	16.81	282.72	29.90
35	9,789	16.00	255.92	25.15
37	5,340	11.30	127.53	11.54
38	6,417	11.24	148.11	21.06
39	8,730	12.17	251.65	18.96
40	6,360	15.86	95.52	18.17
42	6,281	9.77	553.48	15.36
43	6,071	23.53	43.25	37.46
44	6,176	6.58	30.58	10.83
45	6,412	5.53	2.59	8.95
46	8,790	1.61	44.24	2.51
47	5,416	6.65	307.37	7.57
48	6,571	17.53	262.68	32.27
Z 8340	5,700	5.77	33.35	10.13
Z 8543	6,700	10.82	117.12	16.15

Ref.: ²Minimum significant difference (msd): 17.52 for alpha: 0.05

Table 3. Analysis of variance for the g.c.a. trial.

Fountain	D.F.	S.S	Mid Square	F	Probability
Blocks	3	1648.19	549.398	3.49	0.0172 *
Treatments	52	34969.56	672.492	4.27	0.0000 *
Error	156	24540.71	157.312		
Non additive	1	170.88	170.877	1.09	0.2988 NS
Residual	155	24369.84	157.225		
TOTAL	211	61158.47			

General average: 63.726. Total Sum per treatments: 13509.930. Total number of plots: 212.

Coefficient of variation: 19.68%. Categorical variables: Blocks 1-4 and Treatments 1.

Studies to determine the degree of resistance to *Ustilago maydis* D.C. Corda in wild and cultivated species of the genus *Zea*

--Astiz Gassó, MM, Molina, MdelC

The purpose of the current work is to determine the degree of resistance of wild and cultivated species of the genus *Zea* to *Ustilago maydis* D.C. Corda (U.m.) which will subsequently be used in crosses to obtain new forage plants. The host materials used were the population c.v. Colorado Klein, the inbred lines SC66, B73 and E642A88 of *Zea mays* ssp. *mays* as well as clones of *Zea*

Figure 1: Reaction of Colorado Klein to 6 strains U. maydis isolates.

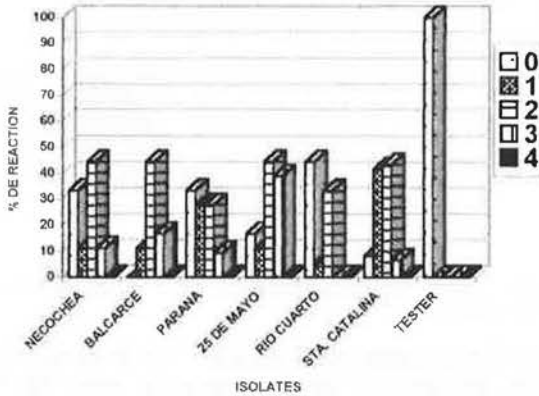


Figure 4: Reaction of E64A688 line to 6 strains U. maydis isolates

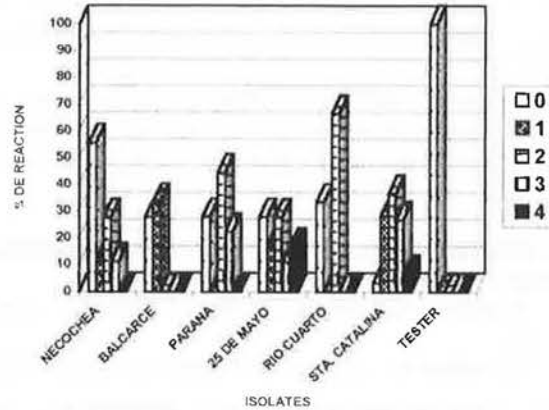


Figure 2: Reaction of SC66 line to 6 strains U. maydis isolates.

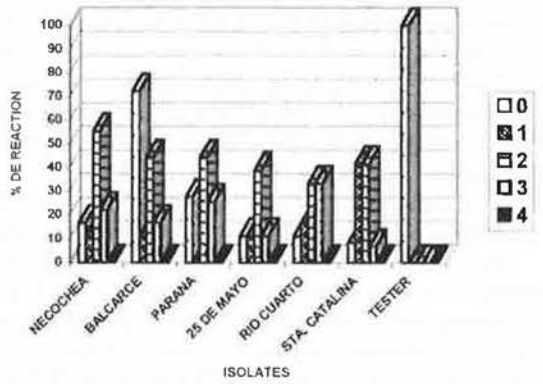


Figure 5: Reaction of Zea diploperennis to 6 strains U. maydis isolates.

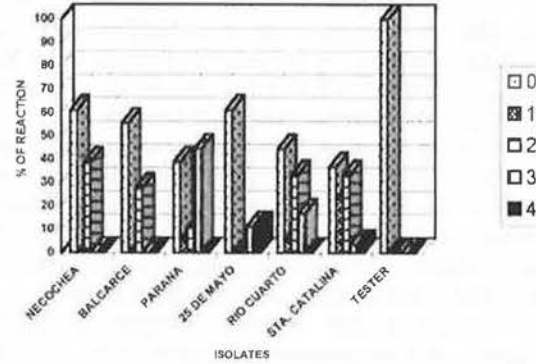


Figure 3: Reaction of B73 line to 6 strains U. maydis isolates.

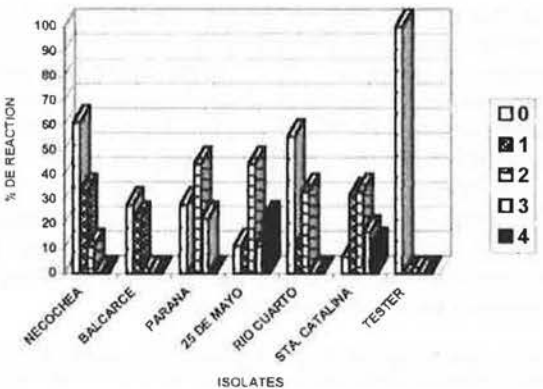
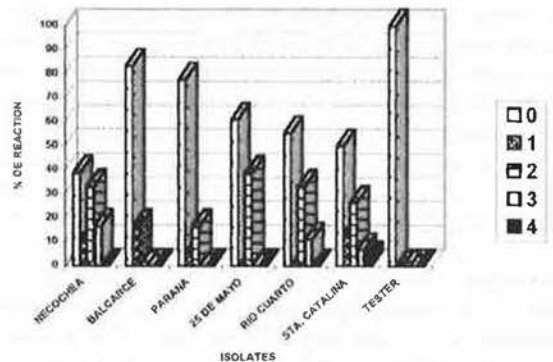


Figure 6: Reaction of Zea perennis to 6 strains U. maydis isolates.



diploperennis and *Zea perennis*. All the materials were inoculated with different of strains of U.m. isolated at Santa Catalina, Necochea, Balcarce, Paraná, 25 de Mayo, Río Cuarto. Strains were cultivated in a liquid medium of 2% C.P.G., under shaking for 8 days running at 25 C. The pathogen was inoculated using a

hypodermic syringe. The trial involved three replications and a tester (non-inoculated plants). The plants were evaluated using a reaction scale (Table 1) to determine the mean percent infection of the U.m. fungus.

Table 1. Reaction scale in hosts.

Behaviour	Host Reaction
Immune (0)	No reaction
Resistant (1)	Partial Chlorosis
Medium resistant (2)	Accent chlorosis and/or presence of stripe or anthocyanin stain.
Medium susceptible (3)	Necrosis and diminution of growth in plants
Susceptibles (4)	Formation of tumors (galls)

Wild and cultivated species of the genus *Zea* showed different reaction grades from resistant to susceptible (graphic 1-6), depending both on the *Zea* genotype or the geographic origin of U.m. strain inoculated.

The male transmission rate of B-chromosomes is controlled by the A-chromosomes in maize

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In native populations from northern Argentina we have found numerical polymorphism for accessory or B-chromosomes (Bs). It is reported that Bs are maintained in the populations due to various mechanisms of driving (Carlson and Roseman, Genetics 131: 211-223, 1992), consisting of: 1) Suppression of meiotic loss when they are in single doses, 2) Nondisjunction at the second pollen grain mitosis, 3) Preferential fertilization by the sperm nucleus carrying Bs produced after the nondisjunction process, 4) Competitive ability of B-carrying pollen grains.

One of the features of the Bs mode of inheritance is the variation in their transmission rate (TR) in such a way that in some progenies the Bs tend to be lost and in others they tend to increase in number compared to Mendelian expectation. The B TR was estimated by the ratio between the mean number of Bs transmitted to the progeny and the number of Bs of the parental plant that carried them. In previous papers we found that the frequency of B chromosomes in native races of maize varies considerably in different populations (Rosato et al. 1998, Am. J. Bot. 85(2), in press). Moreover, the existence of genotypes controlling high and low male transmission rate (male B-TR) of B chromosomes in Pisingallo race (Rosato et al., Am. J. Bot. 83(9):1107-1112, 1996) was demonstrated.

In the present work crosses were made to determine whether the genes controlling male B-TR are located on the normal chromosome set (A) or on the B-chromosomes. The experiment was carried out in two lines (high and low male B-TR lines) from the race Pisingallo (VAV 6313) which is a native population from the northwest of Argentina collected by the authors. We made four types of 0B female x 2B male crosses between and within both H (High) and L (Low) B-TR lines. Twenty two successful crosses were obtained as follows: (1) five H x H; (2) six H x L; (3) six L x H; and (4) five L x L (Figure 1). The number of Bs was determined in primary root tips of at least twenty individuals of each of the twenty two ears. The male B-TR values were compared with a two way ANOVA test. The frequencies of progeny with 2B, related to the frequencies of individuals with 0B, were compared with the Mendelian expectation (0.5) using a χ^2 test.

The mean B-TR of the progeny resulting from H x H crosses was 0.71 ± 0.04 (Table 1). The frequency of individuals with 2B was significantly higher than the Mendelian expectation ($\chi^2 = 23.04$, $p < 0.00001$). The mean B-TR of the progeny resulting

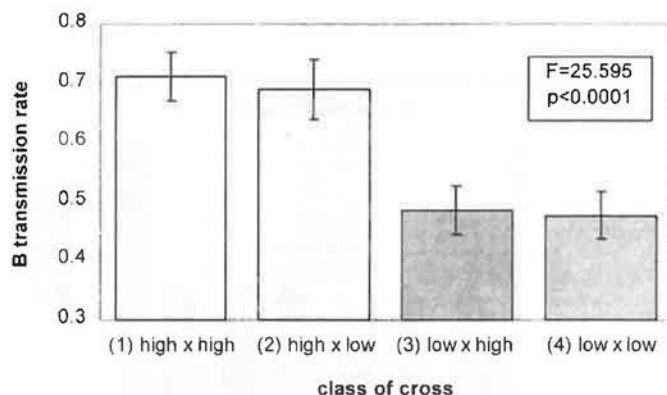


Figure 1. Types of crosses with their male B transmission rate. low: low male B-TR line, high: high male B-TR.

from L x L crosses was 0.48 ± 0.04 . The frequency of individuals with 2B did not differ significantly from the Mendelian expectation ($\chi^2 = 0.286$, $p = 0.593$).

The mean B-TR of the progeny resulting from H x L crosses was 0.70 ± 0.05 ; this value does not differ significantly from the B-TR from H x H ($p = 0.99882$). The frequency of individuals with 2B was also significantly higher than the Mendelian expectation ($\chi^2 = 21.893$, $p < 0.00001$). The mean male B-TR of the progeny resulting from L x H crosses was 0.48 ± 0.04 ; this value does not differ significantly from the B-TR from L x L ($p = 0.99906$). The frequency of individuals with 2B did not differ significantly from the Mendelian expectation ($\chi^2 = 0.39$, $p < 0.532$).

In all cases most of the individuals were 0B or 2B, the frequency of plants with 1B or 4B being very low (Table 1).

There were significant differences in the B-TR between crosses 1 and 2 vs. 3 and 4 (Table 1), when H and L B-TR female progenitors were considered ($F = 26.453$, $p < 0.0001$), whereas no significant difference was found between crosses 1 and 3 vs. 2 and 4 (Table 1) when H and L B-TR male mean B-TR progenitors were considered ($F = 0.040$, $p = 0.8436$) (Figure 1).

Our results indicate for the Pisingallo maize population (VAV 6313) that the B-TR genotype of the receptor 0B plant determines the rate by which the egg is fertilized by the sperm nucleus carrying 2Bs. Therefore, the detection of differences between sperm with and without Bs is controlled by the female progenitor genotype. Thus, some genotypes increase the frequency of fertilization by B-carrying sperm nucleus, whereas other genotypes do not. The latter result in random fertilization by the 0B or 2B sperm nucleus, leading to a Mendelian B-TR. All these results indicate that the presence of B-chromosomes determines the sperm phenotype which would be accepted by the egg cell. In addition, since in our experimental design the female progenitor has no Bs, we conclude that these controlling genes are located on the regular A chromosome set.

Table 1. Types of crosses with their male B transmission rate (male B-TR). L: low male B-TR line, H: high male B-TR (data from Chiavarino et al., Am. J. Bot. 85, 1998, accepted).

type of cross (female x male)	no. of crosses	no. of indiv. with different no. of Bs			total	mean B-TR ± SE	type of B-TR
		0B	2B	4B			
1) 0B (H) x 2B (H)	5	42	99	1	142	0.71 ± 0.04	high B-TR
2) 0B (H) x 2B (L)	6	50	109	2	161	0.70 ± 0.05	high B-TR
3) 0B (L) x 2B (H)	6	86	78	1	165	0.48 ± 0.04	Mendelian
4) 0B (L) x 2B (L)	5	66	60	0	126	0.48 ± 0.04	Mendelian

Nuclear DNA amount and doses of B-chromosomes in native populations of maize

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A large nuclear DNA variation occurs in the genus *Zea*, in such a way that DNA C value may be different among species, subspecies, populations, inbreds and open pollinated lines. Differences in the amount of heterochromatin and/or the occurrence of B-chromosomes (Bs) would be the cause of the variation in the DNA content in maize. However, the effects of different doses of B-chromosomes have been scarcely investigated.

With the aim of investigating the relationship between the DNA content and B-chromosomes, we studied the DNA amount in individuals with different doses of Bs in four Argentinian native populations of maize. The DNA content was measured using Feulgen microdensitometry (Rosato et al., Am. J. Bot. 85(2), 1998, in press). The results of total DNA content are summarized in Table 1. Differences of DNA content in these populations containing individuals with different doses of Bs (0, 1, 2, and 3B) were analyzed by means of a two way ANOVA test. This analysis indicated that neither the mean DNA content of the populations ($F=1.2397$; $p=0.3040$) nor different doses of Bs ($F=3.2259$; $p=0.0752$) differ statistically. On the other hand, only the interaction between doses of Bs and populations were highly significant ($F=4.8693$; $p=0.0001$). This means that the variation of the DNA content with the doses of Bs depends on each population.

Table 1. DNA content in individuals with different doses of Bs in four populations.

Population (Race)	Mean DNA content with different doses of Bs (X(ES) (pg) (ind. no.)			
	0B	1B	2B	3B
VAV 6485 (Blanco)	5.800±0.094 (6)	6.310±0.072 (3)	6.392±0.062 (4)	6.599±0.146 (4)
VAV 6480 (Am.grande)	6.106±0.102 (8)	6.085±0.083 (4)	6.285±0.097 (4)	6.423±0.016 (3)
VAV 6479 (Blanco)	6.409±0.086 (3)	6.074±0.050 (6)	6.375±0.070 (4)	6.622±0.068 (4)
VAV 6475 (Harinoso)	6.448±0.131 (6)	5.786±0.145 (3)	6.377±0.053 (4)	6.592±0.081 (4)

The results indicated that in the population VAV 6485, with the lowest A-DNA value (5.8 pg in individuals without Bs), the DNA content of individuals with Bs was significantly higher than that in individuals without Bs (Table 1). In the population VAV 6480, the individuals with different doses of Bs did not show any significant difference in DNA content. In the other two populations, VAV 6479 and VAV 6475 with the highest A-DNA values (6.409 pg and 6.448 pg, respectively), the DNA content in individuals without Bs was equal (VAV 6479) or even significantly higher (VAV 6475) than in individuals with 1B (6.074 pg and 5.786 pg respectively). In the latter populations the increase of DNA content with doses of Bs becomes more evident as the number of Bs increases starting from one B (Table 1).

Ayonoadu and Rees (Heredity 27:365-383, 1971) studied the genome size in individuals without Bs and with 8 Bs in the line Black Mexican sweet corn and they calculated that each B increases by about 5% the DNA content in the B-carrying individuals. In view of our results, individuals with Bs do not always have a higher DNA content than individuals without Bs. For this reason, in native populations it is impossible to predict the genome size of individuals with different doses of Bs, considering an increase of

the DNA content of 5% per each B as was determined by Ayonoadu and Rees. This fact is due to the variation in the A-DNA content within these populations which masks the increase due to the presence of Bs.

Heterochromatic bands and doses of B-chromosomes in native populations of maize

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Several studies indicated that the presence of B chromosomes is associated with the presence of knobs and with DNA content. In indigenous populations of North America, Longley (J. Agric. Res. 56:177-195, 1938) found negative association between the number of knobs and the occurrence of B chromosomes. A similar correlation was reported by Bianchi, Ghatnekar and Ghidoni (Chromosoma 14: 601-617, 1963) in Italian populations.

We found in several populations that the increase of DNA content due to the occurrence of B-chromosomes could be masked by the variation of A-DNA content (masking effect) (Rosato et al. MNL 1998, this volume, and Rosato 1997 Ph.D. thesis). With the aim of examining the cause of this masking effect, we analyzed the fluorescent chromosome banding pattern in individuals with different doses of Bs in two Argentine native populations (VAV 6475 and VAV 6485).

In the population VAV 6475, 152 individuals showed 2, 3, or 4 DAPI bands in chromosome pairs 6 and 9; these are interstitial bands on the long arms of chromosomes 6 and 9. All the studied individuals were homozygous for the DAPI+ band of chromosome 9 (+/+) (Table 1). On the contrary, the DAPI band in chromosome 6 was polymorphic, i.e. individuals were homozygous (presence or absence) and heterozygous for the DAPI band. In pair 6, the frequency of homozygotes (+/+), heterozygotes (+/-) and homozygotes (-/-) was 0.11, 0.36 and 0.53, respectively, in individuals with Bs. Otherwise in individuals without Bs, the frequency of homozygotes (+/+), heterozygotes (+/-) and homozygotes (-/-) was 0.17, 0.54 and 0.29, respectively. Assuming that chromosome 9 does not show any variation in the number of DAPI bands, the presence of this DAPI+ band was not considered in the statistical analysis. We compared the frequency of individuals with bands in pair 6 (+/+ plus +/-) in plants with and without Bs using a test for independence (Chi square). The results indicated that the presence of this band is not independent of the presence or absence of B-chromosomes in this population (Yates $X^2 = 7.11$, $p=0.0077$).

In the population VAV 6485 we analyzed 47 individuals showing DAPI bands in the same chromosome pairs (6 and 9). In pair 9, the frequency of homozygotes (+/+) and heterozygotes

Table 1. Number of individuals with DAPI+ bands in chromosome pairs 6 and 9 with different doses of Bs from populations VAV 6475 and VAV 6485.

Doses of Bs	VAV 6475						VAV 6485					
	chrom. pair 9			chrom. pair 6			chrom. pair 9			chrom. pair 6		
	+/+	+/-	-/-	+/+	+/-	-/-	+/+	+/-	-/-	+/+	+/-	-/-
0	61	---	---	10	33	18	14	2	---	---	---	3
1	37	---	---	6	16	15	6	1	---	---	---	7
2	33	---	---	2	11	20	12	---	---	---	---	6
3	19	---	---	2	6	11	8	---	---	---	---	1
4	2	---	---	---	---	2	4	---	---	---	---	1
with Bs	91	---	---	10	33	48	30	1	---	---	---	8

(+/-) was 0.97 and 0.03 in individuals with Bs, 0.88 and 0.12 in individuals without Bs. In pair 6, the frequency of heterozygotes (+/-) and homozygotes (-/-) was 0.26 and 0.74 in individuals with Bs, 0.19 and 0.81 in individuals without Bs. The frequencies of individuals with DAPI bands in pairs 9 and 6 were similar in plants with and without Bs, even though no statistical test could properly be made because of the small size of the sample.

In the population VAV 6485 there was little variation of mean number of bands between individuals with and without Bs. This is in agreement with the increase of the DNA content with the doses of Bs. On the contrary, in the population VAV 6475 the low mean number of DAPI bands in individuals with Bs could be associated with the masking effect (no detection of DNA content supplied by Bs). This fact could be associated with the existence of a negative association between heterochromatic bands and B-chromosomes to maintain an optimum total DNA content (nucleotype).

Genomic in situ hybridization (GISH) technique for determining the origin of the paired and unpaired chromosomes in the 2n= 30 hybrid *Zea perennis* x *Zea luxurians*

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In the last few years, cytological evidence was advanced to demonstrate the polyploid nature of the species belonging to the genus *Zea* (Molina and Naranjo, *Theor. Appl. Genet.*, 73:542-550, 1987; Naranjo et al., *Hereditas*, 120:241-244, 1994; Poggio et al., *Theor. Appl. Genet.*, 79:461-464, 1990; Poggio and Naranjo, *Chromosome Research*, 3 (Suppl.1): 80-81, 1995). These authors postulated five as the original basic number for maize and its related wild species (2n= 20) and that cryptic tetraploids originated by allopolyploidy from different diploid species (2n= 10) which are probably extinct nowadays. Some of the most suggestive evidence which allowed postulating this basic number stemmed from the analysis of the meiotic behavior of interspecific hybrids with 2n=30 chromosomes: *Zea perennis* x *Z. diploperennis*, *Z. perennis* x *Z. mays* spp. *mays* and *Z. perennis* x *Z. luxurians* (Molina and Naranjo, *Theor. Appl. Genet.* 73:542-550, 1987; Naranjo, et al., *Acad. Nac. Cs. Ex. Fís.Nat.*, Buenos Aires, Monografía 5:43-53, 1990; Poggio and Naranjo, *Chromosome Research* 3, Supl.1:80-81, 1995; Poggio et al., *Actas XXVII Congreso Argentino de Genética*:185, 1996). Recent molecular approaches further confirmed the allopoloid nature of maize (Moore et al., *Current Biology* 5: 737-739, 1995).

The genomic formula, proposed through cytogenetic studies, for taxa with 2n=20 is AxAx BxBx; for *Z. perennis* and hybrids with 2n=30 the formulae are ApAp A'pA'p Bp₁ Bp₁ Bp₂ Bp₂ and Ap A'p Ax Bp₁ Bp₂ Bx, respectively. In these hybrids the more frequent meiotic configuration was 5 III + 5 II + 5 I (Fig. 1A). Two hypotheses could be postulated to explain the original source of chromosomes in these configurations: 1) Bivalents are formed by allosyndetic pairing of chromosomes of *Z. perennis* and the "B" genome of any species with 2n= 20, and the univalents would correspond to *Z. perennis*, i.e ApA'pAx would constitute the 5 III, Bp₁ Bx the 5 II and Bp₂ the 5 univalents; 2) bivalents are formed by autosyndetic pairing of Bp₁ and B'p₂ genomes from *Z. perennis* and univalents would correspond to the B genome of any species with 2n= 20, i.e. ApA'pAx would constitute the 5 III, Bp₁ Bp₂ the 5

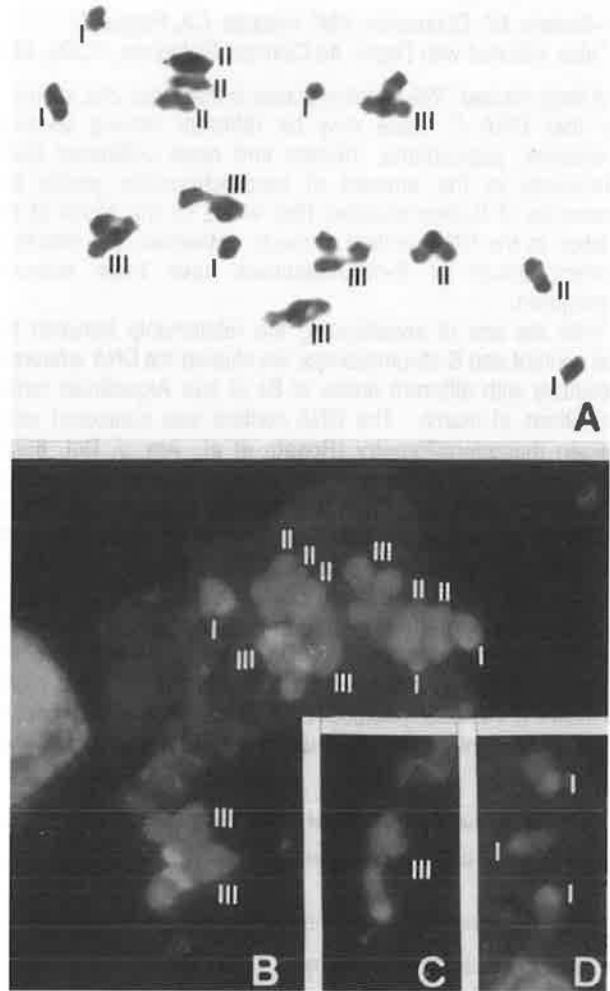


Figure 1.

II, and Bx the 5 univalents.

Genomic in situ hybridization (GISH) is a molecular cytogenetic technique which now allows chromosomes from different parents or ancestors to be distinguished by means of differential hybridization of entire genomic probes (Bennett 1995, In: Kew Chromosome Conference IV, P.E. Brandham and M.D. Bennett, eds. Royal Botanic Garden, Kew, pp. 167-183). Using this technique in hybrids it is possible to determine the genome origin of paired and unpaired chromosomes in metaphase I configurations.

Zea luxurians has positive DAPI bands that did not hybridize with *Zea perennis* probe (Poggio et al., *Actas XXVII Congreso Argentino de Genética*:185, 1996). Therefore, these chromosome regions are useful as genome markers in *Z. perennis* x *Z. luxurians* hybrids.

We present herewith the results obtained through GISH on meiotic cells of these hybrids, carried out in order to determine the origin of chromosomes forming the trivalents and univalents in the 2n= 30 *Z. perennis* x *Z. luxurians*.

Chromosome preparations: a) For meiotic configuration analysis: young panicles of the hybrids were fixed in 3:1 (absolute alcohol:glacial acetic acid). The squash of anthers was made in a drop of 2% acetic haematoxylin. b) For in situ hybridization: young panicles of the hybrid *Z. perennis* x *Z. luxurians*, growing at the

IFSC, were fixed in 3:1 (absolute alcohol: acetic acid). Fixed anthers were washed in 0.01 M citric acid-sodium citrate, pH 4.6 buffer to remove fixative, transferred to an enzyme solution containing 2 ml of 2% cellulase (Onozuka R10) and 20% liquid pectinase. The softened material was again washed in the above buffer solution. Finally, anthers were squashed onto slides in a drop of 45% acetic acid. Preparations showing well spread metaphase I cells were selected by phase contrast light microscopy. After removal of the coverslip by freezing, the slides were allowed to air dry.

DNA probes: Probes consisted of total genomic DNA, which was isolated from adult leaves of the two species *Zea perennis* and *Zea luxurians* using standard methods. Probes were labelled by random priming with digoxigenin 11-dUTP (Boehringer, Mannheim, Germany).

Fluorescent "in situ" hybridization: The technique was developed in the "Departamento de Biología Celular y Genética, Universidad de Alcalá, Alcalá de Henares, Madrid, España) and was carried out according to Cuadrado and Jouve (Genome 38(4):795-802, 1995) with minor modifications. Slide preparations were incubated in 100ug/ml DNase-free RNase in 2 X SSC for 1 hour at 37 C in a humid chamber and washed three times in 2 X SSC at room temperature for 5 min. The slides were post-fixed in fresh 4% (w/v) paraformaldehyde in water for 10 min, washed in 2 X SSC for 15 min, dehydrated in a graded ethanol series and air dried.

The hybridization mixture consisted of 50% (v/v) deionized formamide 10% (w/v) dextran sulphate, 0.1% (w/v) SDS, 0.3 mg/ml of salmon sperm DNA in 2 X SSC. Then 100 ng of digoxigenin 11-dUTP probe was added to 30 ul of hybridization mixture for each slide and denatured for 15 min at 75 C. The hybridization mixture was loaded onto the slide preparation and covered with a plastic coverslip. The slides were placed on a thermocycler machine at 75 C for 7 min (denaturation), 10 min at 45 C and 10 min at 38 C. The slides were then incubated at 37 C overnight for hybridization.

Following hybridization, coverslips were carefully floated off by placing the preparations in 2 x SSC at 42 C for 3 min and then given a stringent wash in 20% formamide in 0.1 X SSC at 42 C for 10 min. The slides were washed in 0.1 X SSC at 42 C for 5 min; 2 X SSC for 5 min at 42 C; 4 X SSC 0.2% Tween 20 at 42 C for 5 min and finally for one hour in 4 X SSC/tween 0.2% at room temperature.

To detect probes, slides were treated with sheep antidigoxigenin FITC (Fluorescein isothiocyanate). Slides were treated with 5% (w/v) BSA (bovine serum albumine) in detection buffer (4 X SSC, 0.2% (v/v) Tween-20). Then slides were incubated in a solution of the corresponding antibody in detection buffer containing 5 % BSA for one hour at 37 C and washed three times in 4 X SSC/ Tween 20 for 10 min at room temperature.

Slides were counterstained with 4'-6-diamidino-2-phenylindole (DAPI) (2 ug/ml in McIlvaine's citrate buffer, pH=7), for 10 min at room temperature and then they were mounted in antifade solution. Slides were examined with a Zeiss Axiophot epifluorescence. Photographs were taken using Fuji color super G400 color print film.

In situ hybridization was carried out on hybrid meiotic cells. Chromosomes were blocked with unlabelled *Zea perennis* genomic DNA and probed with digoxigenin labelled genomic DNA from *Zea luxurians*. This procedure allowed chromosomes from *Zea luxurians*

to be identified by mean of their telomeric specific sequences and to be distinguished from those derived from *Zea perennis*, which remained unlabelled because of the blocking procedure.

In metaphase I cells of the hybrid *Z. luxurians* x *Z. perennis* the following configurations and hybridization signals were observed: a) Univalents had a strong fluorescent signal in their telomeric regions; therefore, these chromosomes correspond to *Z. luxurians*, because they display the distal portion not blocked by *Z. perennis* DNA that bound *Z. luxurians* labelled DNA probe (Figs. 1B, D). b) Trivalents were composed by one chromosome labelled in its telomeric region - i.e. a *Z. luxurians* chromosome- and two smaller and unlabelled ones - i.e. *Z. perennis* chromosomes (Figs. 1B, C). c) Bivalents did not show any label so they correspond also to *Z. perennis* (Fig. 1B). (ed. note: see color reproductions of Figs. 1B, C, and D in MaizeDB)

These results confirm the second hypothesis previously advanced, that in these 2n= 30 hybrids the trivalents are formed by autosyndetic pairing of Ap and A'p genomes from *Zea perennis* and by allosyndetic pairing with the A genomes from the tetraploid parent *Z. luxurians*.

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Forage aptitude in maize populations

--Torrecillas, M, Bertoia, LM

Landraces without improvement and composites with few cycles of selection constitute a source of important variability to be used as a base in improvement programs, directed to the synthesis of hybrids with forage aptitude.

Four populations and two improved composites, per se and their crosses with selected inbred lines PR4 and PR10 were evaluated. These materials showed diversity in origin, cycle and plant architecture. Trials were performed in two environments located in the Buenos Aires milk belt. A complete block design with three replications was used, Morgan 369, Funk's Tilcara and Pioneer 3452 being the commercial checks. The experimental unit consisted of two 5-m rows, spaced 0.7 m apart. Combined analyses were computed to determine the effects of general combining ability (GCA) and specific combining ability (SCA).

The studied variables were Ear Dry Matter Yield (EDMY); Stover Dry Matter Yield. (SDMY); Whole Plant Dry Matter Yield (WPDMY).

In the combined analysis for SDMY and WPDMY, GCA effects in populations and composite were detected. No significant effects of GCA for the inbred lines were found. Significant effects of SCA for the variable SDMY were observed, the interaction of this effect with the environment for the same variable being significant. In two environments evaluated, the populations 1 and 4 had the greater values of GCA for SDMY and WPDMY.

The superiority shown by some crosses upon the commercial

Table 1. SDMY

Material	Mean ¹	GCA
Population 1	9.63	6.55 *
Population 2	8.92	-0.6
Population 3	7.80	-11.74
Population 4	9.61	6.30 *
Composite Pitaguá	9.08	1.08
Composite Semiflint	8.82	-1.6

Table 2. WPDMY

Material	Mean ¹	GCA
Population 1	21.08	9.52 *
Population 2	20.46	-0.85
Population 3	18.68	-14.45
Population 4	20.54	4.16 *
Composite Pitaguá	20.51	3.76 *
Composite Semiflint	19.92	-2.14

¹Yield in Tn ha⁻¹

hybrids for the variable WPDMY indicates the feasibility of using that germplasm in improvement programs directed to forage production.

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The thermal induction of HSP 18 mRNA in *Zea mays* callus

--Friedberg, JN, Yang, Z, Walden, DB

Greyson et al. (Devel. Genet. 18:244-253, 1996) have shown that the heat shock response in maize radicles and shoots is found primarily in cytoplasmically dense cells and meristematic regions. Neither the cell type or organized meristem are found in 'immature' callus. Thus to extend our observations, maize callus cultures were initiated; callus was subjected to antisense RNA in situ hybridization to determine if callus and/or cells from the callus are able to respond to thermal stress.

Callus was initiated from 16 to 21 day old embryos (inbred Ohio 43). Embryos were plated on MS basal medium containing 10⁻⁵ 2, 4-dichlorophenoxyacetic acid (2,4-D). Calli approximately 30 days old were heat shocked at 43 C for 2 hours, fixed, embedded in wax, cut in 10 micron sections and mounted on slides. The in situ hybridization was performed as described by Greyson et al. and used the antisense scMHSP 18-9-2 RNA, DIG labelled probe (Table 1, Greyson et al., 1996).

The callus cells exhibited strong expression of the HSP 18 mRNA, indicating that the tissue was responding to the heat shock. The mRNA expression appeared to be localized either in the nucleus or in the periphery of the cells. Across the callus section, mRNA expression was variable. These results indicate that the ability to respond to heat shock may be limited to cycling cells in an active nuclear cycle and that 'mature' cells lose the ability to respond to a thermal induction.

The induction of mRNA from 18 kDa HSP genes in maize lateral roots

--Yang, Z, Greyson, RI, Bouchard, RA, Walden, DB

We reported (Greyson et al, Devel. Genet. 18, 1996) that maize 18 kDa heat shock protein (HSP) mRNA increases following heat shock in the root-tip and shoot-tip. The HSP mRNA is found in specific cells. In the heat-shocked plumule, the mRNA is found mostly in the meristem, young leaves, and the vasculature of the coleoptile rather than in the more mature parenchyma cells. In the

radicles, more mRNA is found in the meristematic region (1-3mm from the tip), and in the vasculature, than in the more mature distal region (4-10mm from the tip). We proposed that meristematic cells and cytoplasmically dense cells respond to heat shock.

To test our hypothesis, maize seeds were grown in the dark for seven days to permit lateral roots to develop. The lateral roots were heat-shocked, fixed, and embedded in wax. In situ hybridization was carried out according to Greyson et al. (MNL 71, 1996), employing the probe described therein.

Strong hybridization signals were observed within the tissues of lateral roots, but no signal was found in the primary root at the site where the lateral roots initiate (Fig.1).



Figure 1. Seven day old heat-shocked maize radicle (with both primary and lateral roots) in situ hybridized with SHSP 18-9-2 antisense RNA probe.

These results confirm that in the primary root, the cells of the more mature tissues no longer respond to heat shock, but as a lateral root is initiated, the cells in the lateral root (meristematic cells) regain the ability to respond to heat shock. (This work is supported by NSERC to D.B.W.)

Nucleolar fusion in different maize cell-types

--Maillet, DS, Quinn, CE, Friedberg, JN, Walden, DB

Eukaryotic genomes contain one to several tandem arrays of rDNA which encode for ribosomal subunits usually located in the nucleolar organizing region (NOR). The region of the nucleus where transcript processing takes place has the characteristic appearance of a dark, usually spherical object, the nucleolus.

The number of nucleoli in a cell can vary from one to as many as the number of NORs encoded in the genome. The maize genome has one NOR on the short arm of chromosome 6, which can result in nuclei with one or two nucleoli. Morgan reported that of 519 premeiotic interphase cells, all had one nucleolus, and that of 508 cells in mitotic interphase 32 had two nucleoli (Morgan, Cytologia 36:669-673, 1971). It has also been shown that the number of nucleoli can be dependent on the cell type. Nucleoli are useful cytogenetic markers that allow the number of copies of chromosome 6 to be determined during interphase and prophase. Any study of nuclear organization must consider the number and size of nucleoli because nucleoli occupy space and, as a result of the possibility that multiple NORs could participate in the formation of one nucleolus, the organization of the nucleus could be altered. As part of such a study, we wanted to determine the effect that an additional NOR would have on nuclear organization.

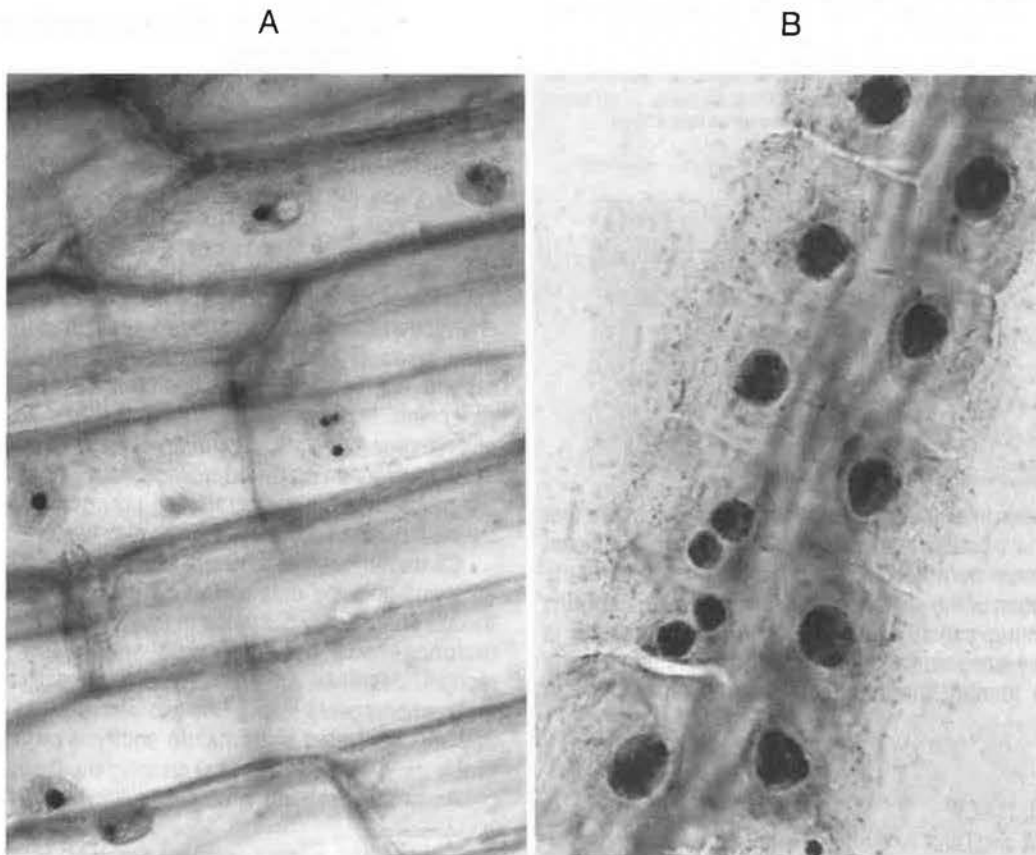


Figure 1. (a) Trisomic 6 coleoptile tissue, (b) Ohio 43 root file preparation (silver stained).

The nucleoli of maize can be detected in cell spreads with a silver staining method (Howell and Black, *Experientia* 36:1014-1015, 1980). In order to screen for trisomic 6 plants the silver staining method was applied to intact tissue (roots and the coleoptile). In addition to identifying trisomic 6 plants, the tissue preparations revealed that the order of fused/unfused nucleoli in files of cells formed a pattern. We recognized the utility of the modified protocol for any situation where large numbers of plants or cells must be screened for changes in the number or size of nucleoli. The following modified protocol can be used to quickly stain and visualize nucleoli in root and coleoptile tissue. Tapetal and callus cell spreads were also examined using the original protocol.

Tissue was fixed in 3:1 (ethanol:acetic acid) until needed for staining. The fixative was rinsed out of roots by immersion of the tissue in water for thirty minutes. Roots were digested in citrate buffer containing 4% pectinase (v/v) for 1 to 2 hours, at 37 C, then rinsed in water for ten minutes. Next, cells were spread in 3:1 fix on a microscope slide. This produces files of cells. The slide was stained with one part developer and two parts silver nitrate at 70 C (Howell and Black, *Experientia* 36:1014-1015, 1980) until nucleoli could be visualized under a light microscope. The coverslip was removed by rinsing in water and the slide was dried overnight, then mounted in premount. Fixed coleoptiles were rinsed for five minutes in water, placed on a microscope slide and cut in half. Tissue was stained as above without digestion, sealed under a cover slip and visualized under a light microscope. This allowed files of cells to be scored as above. Excess stain can be

removed from microscope slides by adding more developer at one side of the cover slip and drawing it across with a piece of filter paper. Microscope slides were sealed with nail polish and examined. Fresh callus (not fixed) was digested in citrate buffer containing 4% pectinase (v/v) and 5% cellulase (w/v), spread out on a microscope slide and stained the same way as roots. Tapetal cells were removed from fixative, placed in water for 30 minutes and then digested in 4% (v/v) pectinase and 5% cellulase (w/v) for 1 hour. After digestion cells were spread on a microscope slide, dried over night, silver stained and scored.

Files of stained coleoptile or root cells were scored sequentially for the number of nucleoli per cell. A length of 20 to 50 cells was counted per file and several slides were analyzed for each tissue type. Spread callus and tapetal cells were counted at random for the number of nucleoli per cell, as the cells could not be retained in files.

The frequency of one or two nucleoli was determined and a pattern was identified; cells containing two nucleoli are preferentially near each other in root, tapetal and coleoptile cells (refer to Table 1). Figure 1a and b show silver stained coleoptile and root cell preparations. In most cells the number of nucleoli was correlated with the size of the nucleoli: in trisomic cells there could be three small nucleoli, one small plus a medium sized nucleolus, or one large nucleolus. In the diploid cell-types the nucleus had either one medium sized nucleolus or two small nucleoli.

Genetic background and tissue-type appear to be factors affecting the frequency of fused nucleoli (see Table 1).

This phenomenon of clustered cells with the same number of

Table 1. Percentages of cells with one nucleolus (1), two nucleoli (2), or three nucleoli (3); two cells next to each other in a file of cells with two nucleoli each (22), two cells next to each other, one with three nucleoli and one with two nucleoli (23), three cells in a row all with two nucleoli (222) and four cells in a row all with two nucleoli (2222); the number of nuclei with 2 nucleoli (N2N); the total number of cells scored (n); and a ratio of cells with two nucleoli to cells with one (1:2). Abbreviations used in column one are Oh 43 (Ohio 43), Coleopt (coleoptile), Tri 6 (Trisomic 6), Mo 17 (Missouri 17), KYS (Kansas Yellow Saline), Tap (metaphase tapetal cells).

	Number of Nucleoli per Nucleus (%)			N2N	Number of Runs of Nucleoli per Cell (frequency)				n	1:2
	1	2	3		22	23	222	2222		
OH 43	87.2	12.8	-	84	18	-	2	3	659	6.8:1
Root										
Oh 43	87.3	12.7	-	123	20	-	1	1	970	6.8:1
Colept.										
Tri 6	88.0	10.0	2	25	2	4	2	0	250	8.9:1
Colept.										
Mo 17	85.0	15.0	-	38	3	-	2	0	261	5.8:1
Colept.										
KYS	82.5	17.5	-	54	6	-	0	1	308	4.7:1
Colept.										
OH 43	57.5	42.5	-	85	-	-	-	-	200	1.4:1
Tap.										
OH 43	63.0	33.0	1*	66	-	-	-	-	200	2:1
Callus										

*This observation could be the result of aneuploid cells or scoring errors.

nucleoli can be seen in all the tissue-types and strains of maize that we studied. It is possible that daughter cells retain positional memory from their parent cell leading often to similar/same nuclear organization of the short arm of chromosome 6. Similarly, cell to cell communication could be occurring that results in neighboring cells having similar numbers of nucleoli. Further study is underway to identify the frequency and mechanism(s) of nucleolar fusion.

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The fused leaves mutant in maize is altered in the embryo and juvenile leaf morphology

--Frascarolo, P, Landoni, M, Dolfini, S¹, Gavazzi, G, Consonni, G

The "fused leaves" (*fdl*) mutant was originally isolated by Dr. Jane Langdale in a genetic line carrying an active *Spm*; it behaves as a single gene recessive mutant and its phenotype is recognizable shortly after germination. In comparison to wild-type siblings, homozygous *Fdl* seedlings show a delay in germination; they also exhibit curly initial leaves that develop precociously while still enclosed in the coleoptile. Frequently, among mutant seedlings, fusion of the leaf surface is also observed: the first and the second leaves or, alternatively, the coleoptile and the first leaf are fused together. Later on in the development, the plant acquires a normal phenotype with expanded and discrete leaves. Homozygous mutant plants at four leaves stage are indistinguishable from wild type plants; they can be grown to maturity and selfed to obtain homozygous progeny seedlings.

Histological analysis reveals that fusion occurs between the epidermis of the leaves. The epidermis of the curly leaf is also altered in the cell morphology: the presence of a series of cells with larger diameter and irregular shape is observed where the fusion occurs. Development of the mutant embryo is delayed in comparison to the wild type embryo, particularly at the level of the shoot primordia. At maturity, the plumule comprises five leaf primordia whose morphology is altered, anticipating leaf morphology of the mutant seedling.

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Maize mutants defective in embryogenesis

--Giulini, AP, Busti, E¹, Consonni, G, Dolfini, AS¹, Furini, A², MacCabe, AP, Gavazzi, G

The data discussed in this note refer to the characterization of mutants with defective embryogenesis: *emb* (embryo specific) mutants, exhibiting a phenotype suggestive of specific defects in embryogenesis and *abs* (aborted seed) mutants affected in an early stage of embryo and endosperm development. Table 1 reports the list of mutants included in this study and their prominent features, while Table 2 refers to their immature embryo growth in vitro. The mutants have been generated by *Mu* or *Ac* transposon mediated mutagenesis. At the molecular level, the presence of a co-segregating polymorphism may provide a means by which the disrupted genes may be cloned.

Characteristics of *emb* and *abs* mutants: The general picture emerging from the data of Table 1 is that the *abs* mutants, even though dramatically disrupted in their development, maintain their morphogenetic potential with shoot and root primordia recognizable while *emb* mutants appear specifically blocked at an early embryogenic step. The two *abs* mutants (*abs*⁻-7065 and *abs*⁻-8075), tested as immature embryos on appropriate media (Table 2), yield callus and slow growing seedlings.

As to the *emb* mutants the following observations were obtained:

emb⁻-7182: mutant embryos are recognizable at 8 DAP as small proembryos while at 15 DAP they show an abnormally long suspensor that disappears at later stages (25 DAP) leaving an apparently blocked embryo, whose developmental arrest might be related to the abnormal suspensor development.

Histological analysis of *emb*⁻-7917 mature seeds reveals absence of embryo while the endosperm shows an abnormal aleurone consisting of two or more cell layers, particularly evident in the basal portion of the seed, probably due to alterations in the orientation of the cell division plane.

Immature *emb*⁻-7065 embryos, recovered in the selfed progeny of heterozygous *+emb* plants, can be rescued if grown on regeneration MS media where they produce small albino seedlings while normal sibs yield green seedlings. This observation suggests an involvement of the *Emb* gene in the control of components of the photosynthetic apparatus.

The *emb*⁻-7190 mutant was detected in a line obtained by selfing F1 plants heterozygous for *Ac* and *r-scm3* and originally selected by Dr. S. Dellaporta as a putative nongerminating mutant. Further analysis of F3 progeny indicated that the mutant produces primary root while lacking shoot primordia. The "shootless" character segregates in a 1 to 3 ratio indicating that it is a monogenic Mendelian trait. Lack of shoot/leaf primordia organization is observed on immature (17 DAP) embryos. Another shootless mutant in our collection, *ed*⁻-41v, analyzed by Racchi et al. (Maydica 41:271-277, 1996) has been shown to respond to the addition of kinetins to the growth medium by producing shoots while a similar mutant (*dks8*) has been studied by Dr. C. Rivin. Allelism tests between the three mutants is under way and their study should be revealing in terms of the shoot apical meristem organization.

Table 1. Characteristics of *emb* and *abs* mutants.

Mutant	Origin	Histological analysis at different DAP					Mature seed	
		10		14-18		shoot and root	end	emb
		end ⁽¹⁾	emb ⁽²⁾	end	emb			
<i>abs7065</i>	Mu	ret. ⁽³⁾	ret.	ret.	ret.	present	s.red.	ND
<i>abs8075</i>	Mu	ret.	ret.	ret.	ret.	present	s.red.	ND
<i>abs8077</i>	Mu	-	-	ret.	ret.	-	s.red.	ND
<i>emb7065</i>	Mu	-	-	ret.	ret.		normal	ND
<i>emb7191</i>	Ac	-	-	-	s.red ⁽⁴⁾	alb.sdl. ⁽⁷⁾ absent	red.	ND
<i>emb7192</i>	Ac	normal	ret.	normal	blocked	absent	normal	ND
<i>emb7182</i>	Ac	normal	ret.	normal	abn. ⁽⁶⁾	absent	normal	ND
<i>emb7190</i>	Ac	-	-	normal	abn.	root only	normal	shootless
<i>emb7917</i>	spont.	-	-	-	ND ⁽⁵⁾	absent	abn.	ND

end : endosperm

emb : embryo

ret. : retarded when compared to wild-type sibs

s.red. : slightly reduced in size

ND : not detectable

abn. : abnormal

alb. sdl. : albino seedling

Table 2 . In vitro analysis of *emb* and *abs* mutants

Mutant	Genetic background	14 DAP embryo				embryo rescue		callus production	
		size (mm)		Stage		+	m	+	m
		+	m	+	m				
<i>abs 7065</i>	W64A	3	1	1	proembryo	+++	+	+++	+
<i>abs 8075</i>	A188	4	1	1	proembryo	+++	++	+++	+++
<i>abs 8077</i>	undef.	4	.5	1	coleoptilar	+++	+(1)
<i>emb 7065</i>	A188	4	3	1	transition	+++	+(2)	+++	+++

necrotic

albino

Table 3. Mutants segregation and distribution in different ear sectors of selfed *+ /abs* and *+ /emb* plants

Mutant symbol	n	m	Segregation %	Distribution in different ear portion:			Heterogeneity (χ^2 test)
				Basal	Intermediate	Apical	
<i>abs 7065</i>	2544	677	26.6	28.2	28.0	23.5	*
<i>abs 8075</i>	1756	374	21.3**	22.1	18.9	23.1	NS
<i>abs 8077</i>	971	247	25.4	25.9	25.0	25.4	NS
<i>emb 7065</i>	1176	305	25.9	25.4	28.6	22.9	NS
<i>emb 7182</i>	1238	300	24.2	22.6	26.2	23.6	NS
<i>emb 7191</i>	1253	339	27.0	27.2	26.6	27.4	NS
<i>emb 7192</i>	2329	514	22.1	25.2	21.2	20.2	*
<i>emb 7917</i>	1545	386	24.6	24.6	24.7	25.2	NS

* Significant at the 5% level

** Significant at the 1% level

Co-segregation analysis: Co-segregation analysis has been performed on individual seedlings obtained by outcrossing heterozygous *+emb* (or *+abs*) male parents to W64A inbred females or F2 segregating progenies with the aim of identifying molecular fragments segregating with the mutant phenotype. Southern blots of restriction digests of DNA extracted from single *+/+*, *m/+* and, when possible, *m/m* seedlings, have been probed with internal *Ac* and/or *Mu* fragments. Restriction length polymorphism was observed in the case of *abs⁺-7065* (DNA digested with *EcoRI*, Southern blotted and probed with a *Mu3* fragment). This result is based on the analysis of 40 individuals obtained by crossing *+abs⁺-7065* male parents to W64A females and it was confirmed with DNA isolated from homozygous mutant seedlings recovered through immature "in vitro" rescued embryos.

A test of gametophytic selection of the mutants: An assay of the occurrence of gametophytic selection is possible if we consider the distribution of homozygous mutants at the tip and bases of selfed *+/m* heterozygous plants.

The results obtained (Table 3) refer to eight different mutants. Of the eight mutants tested only one (*abs⁺-8075*) shows a significant shortage of mutant segregation over the expected, while the remaining seven show a segregation fitting the 1:3 ratio. In two cases (*abs⁺-7065* and *emb⁺-7192*), the heterogeneity χ^2 test indicates a significantly higher proportion of mutants in the basal portion of the ear, suggesting that they exhibit a slight selective advantage over the corresponding wt allele.

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The major quantitative trait loci for plant stature, development and yield are general manifestations of developmental gene clusters

--Khavkin, E, Coe, EH

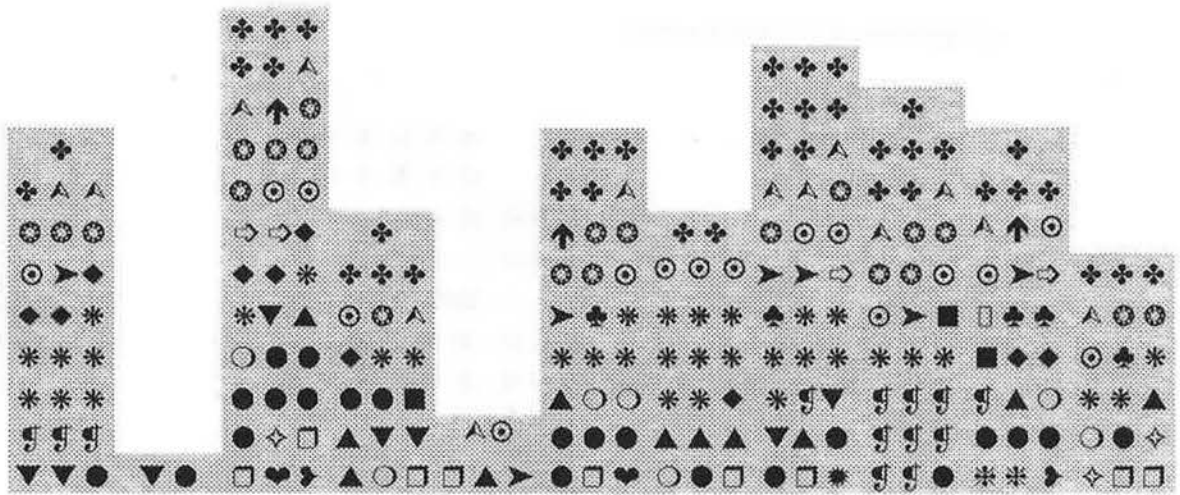
Previously (MNL 68:61, 1994; MNL 69:106, 1995; MNL 70: 42, 1996; Theor. Appl. Genet. 95: 343, 1997) we discussed functional associations of corn developmental genes. We revised the database of quantitative trait loci (QTLs) for maize plant architecture, growth and development rates in vivo and in vitro, yield and its components, and ABA accumulation and effects extracted from the current publications to conjecture QTL positions within the bins on the 1995 UMC molecular map. In this way QTLs reported by different authors were collated. Within a particular mapping population, highly diverse physiological traits were mapped by one and the same RFLP probe, whereas one and the same developmental trait was usually mapped to several positions on different chromosomes. The locations for one and the same developmental trait varied considerably in different mapping populations. When QTL distribution was followed along maize chromosomes, well-defined peaks were found. However, this pattern of QTL distribution was not specific as to putative physiological functions ascribed to particular loci. When QTLs were compared with the qualitative loci mapped by mutations and with cDNA-derived probes, such collation did not support the indirect candidate-gene approach. Taking into consideration the

mapping accuracy, QTL associations coincide with the previously defined clusters of the qualitative developmental genes (Fig. 1), particularly the homeotic genes expressing transcriptional regulators. We believe that these clusters are the functional networks of genes expressed in concert to control plant development. Apparently it is these networks of the developmental genes that are phenotypically manifested as the major QTLs for plant structure, earliness and grain yield.

Many qualitative and quantitative loci affecting corn resistance to abiotic and biotic stress: drought, phytophagous insects, viral and fungal diseases (Beavis et al., Crop Sci. 34:882, 1994; Dingerdissen et al., Mol. Breed. 2:143, 1996; Lebreton et al., J. Exp. Bot. 46:853, 1995; McMullen and Simcox, Mol. Plant-Microbe Interact. 8:811, 1995; Saghai Maroof et al., Theor. Appl. Genet. 93:539, 1996; Schon et al., Heredity 70:648, 1993; Zehr et al., MNL 68:110, 1994) map within the clusters of developmental genes. This evidence presumes that the cooperative expression of developmental clusters might mediate non-specific plant responses to unfavorable environment.

The sources of QTLs: Abler et al., Crop Sci. 31:267, 1991; Agrama and Moussa, Euphytica 91:89, 1996; Ajmone-Marsan et al., Maydica 39:133, 1994, Theor. Appl. Genet. 90:415, 1995, Maydica 41:49, 1996; Armstrong et al., Theor. Appl. Genet. 84:755, 1992; Austin and Lee, Theor. Appl. Genet. 92:817, 1996, Genome 39:957, 1996; Beaumont et al., Genome 38:968, 1995; Beavis, Proc. Annu. Corn Sorghum Ind. Res. Conf. 49:250, 1994; Beavis et al., Theor. Appl. Genet. 83:141, 1991, Crop Sci. 34:882, 1994; Berke and Rocheford, Crop Sci. 35:1542, 1995; Bohn et al., Crop Sci., 36:1352, 1996; Causse et al., Mol. Breed. 1:259, 1995; Charcosset et al., MNL 68:44, 1994; Chernov A.A. et al., MNL 68:49, 1994; CIMMYT 1994 entries to the Maize Genome Database (<http://www.agron.missouri.edu>); Cowen et al., Theor. Appl. Genet. 84:720, 1992; Doebley and Stec, Genetics 134:559, 1993; Doebley et al., J. Hered. 85:191, 1994, Genetics 141:333, 1995; Edwards et al., Theor. Appl. Genet. 83:765, 1992; Heredia-Diaz et al., Maydica 41:65, 1996; Higginbotham, MNL 65:65, 1991; Koester et al., Crop Sci. 33:1209, 1993; Kozumplik et al., Maydica 41:211, 1996; Lebreton et al., J. Exp. Bot. 46:853, 1995; Lubberstedt et al. Crop Sci. 37:921, 1997; Murigneux et al., Genome 37:970, 1994; Phillips et al., Proc. Annu. Corn Sorghum Ind. Res. Conf. 47:135, 1992; Ragot M. et al., Crop Sci. 35:1306, 1995; Rebai et al. Theor. Appl. Genet. 95:451, 1997; Ribaut et al., Theor. Appl. Genet. 92:905, 1996, Theor. Appl. Genet. 94:887, 1997; Sari-Gorla et al., Heredity 69:423, 1992, Heredity 72:332, 1994; Schon et al., Heredity 70:648, 1993, Crop Sci. 34:378, 1994; Stuber et al., Theor. Appl. Genet. 132:823, 1992; Veldboom et al., Theor. Appl. Genet. 88:7, 1994; Veldboom and Lee, Theor. Appl. Genet. 89:451, 1994, Crop Sci. 36:1310 & 1320, 1996; Wan Y., Theor. Appl. Genet. 85:360, 1992; Zehr et al., Theor. Appl. Genet. 83:903, 1992, MNL 68:110, 1994.

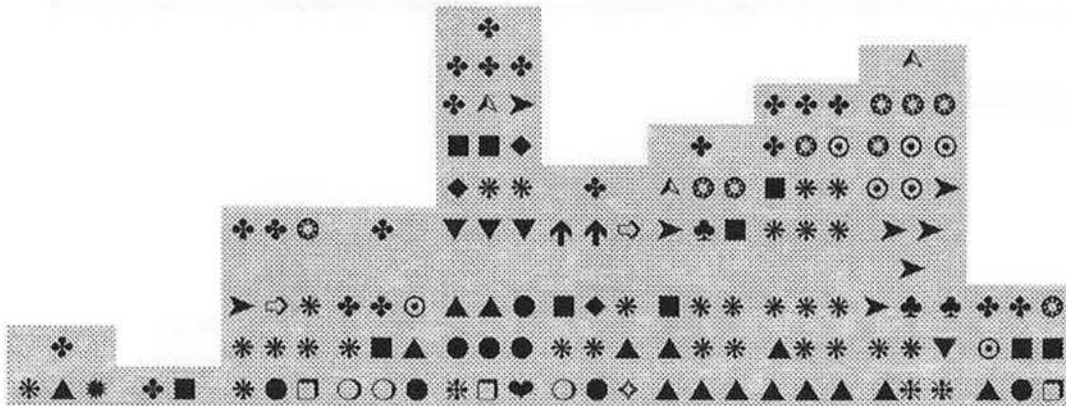
Chromosome 1 (243 probes)



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les2 vp5 *les5 nec2* *ms14* *vg1* *id1 ptd1* *kn1 tbp1* *vp8 rd1 ij2*
rth3 *ms28* *les20*
phyB1

Bins	0	1	2	3	4	5	6	7	8	9	10	11

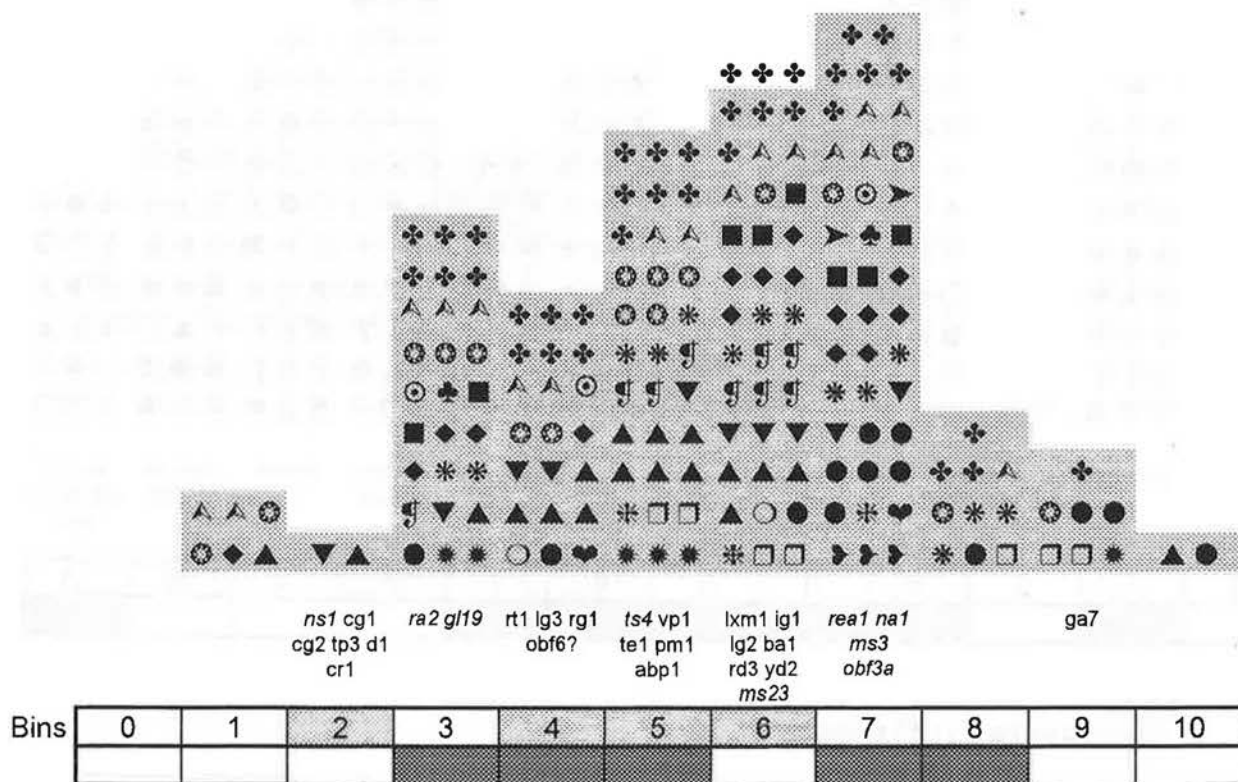
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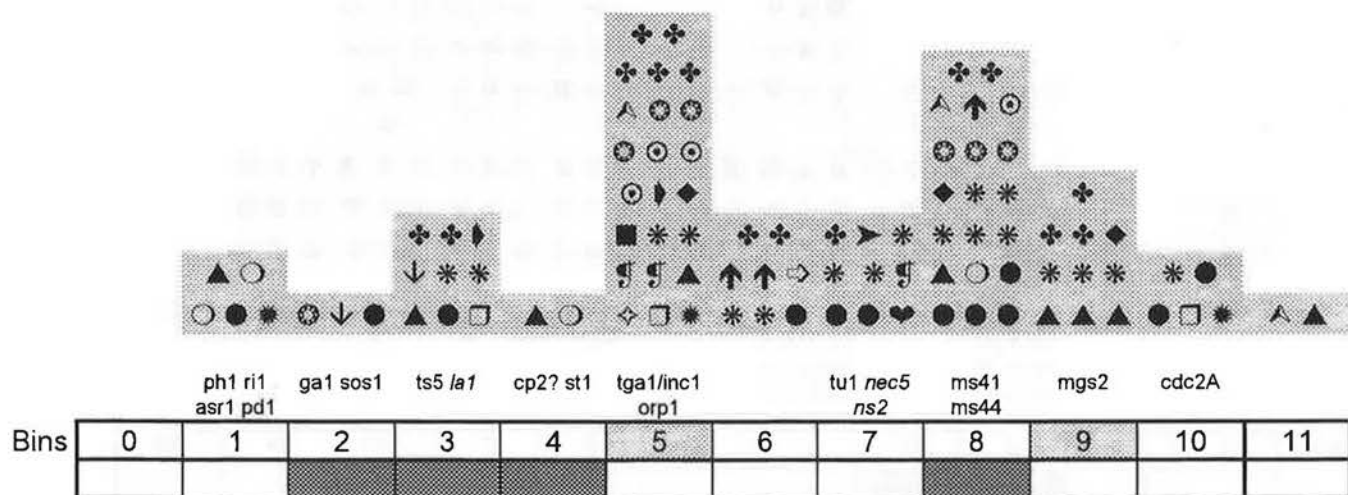
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dks8 gl2 *les1 les18* *les19* *d10*
les11 d5 *les15 ts1*
lg1 nec4 *ba2 wrp1*
tr1

Bins	0	1	2	3	4	5	6	7	8	9	10

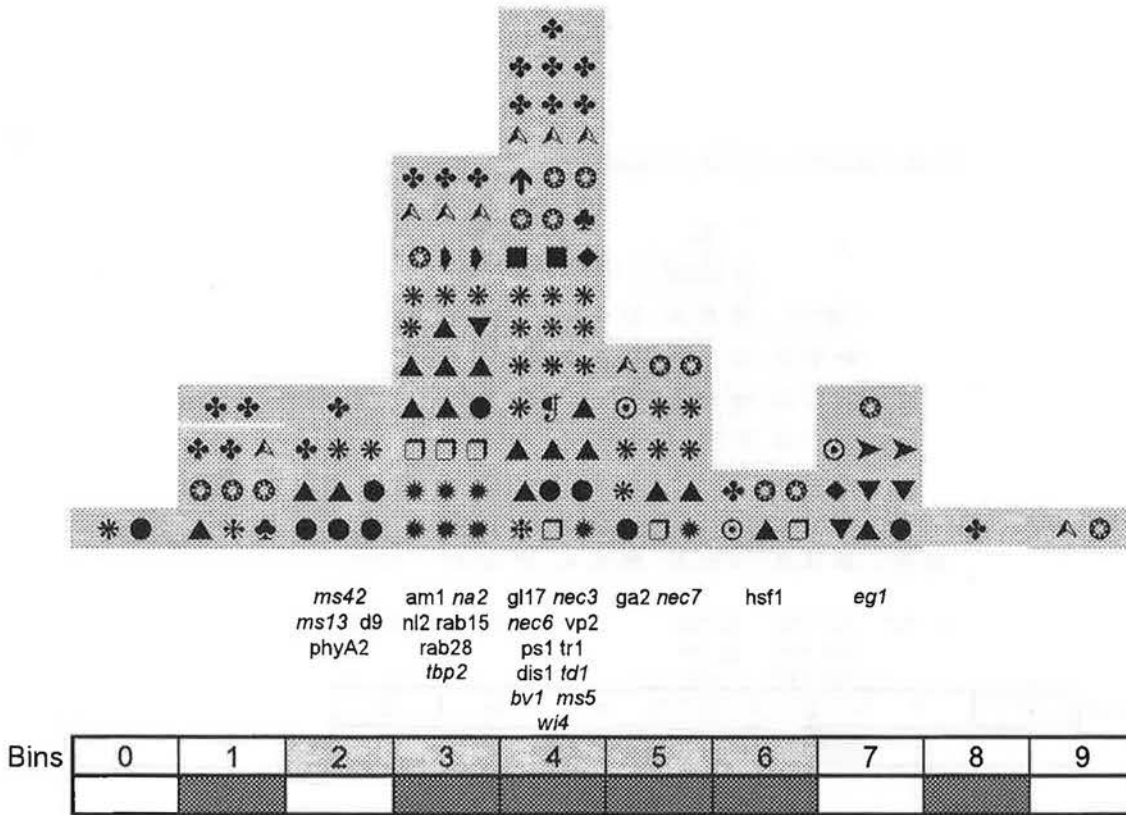
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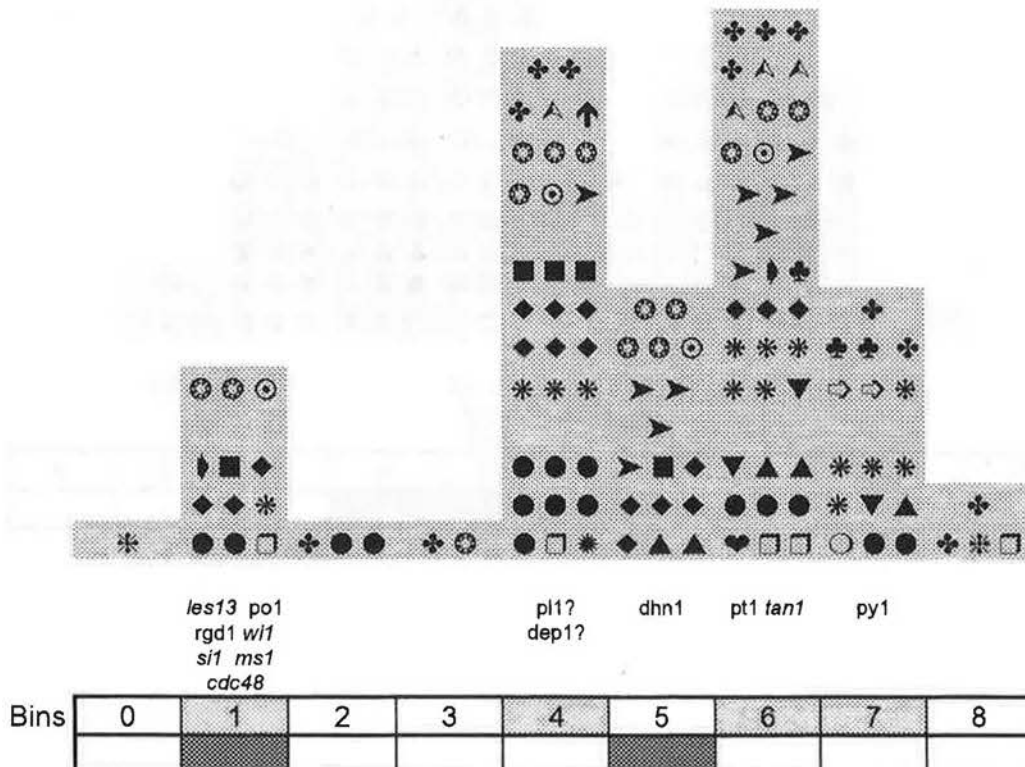
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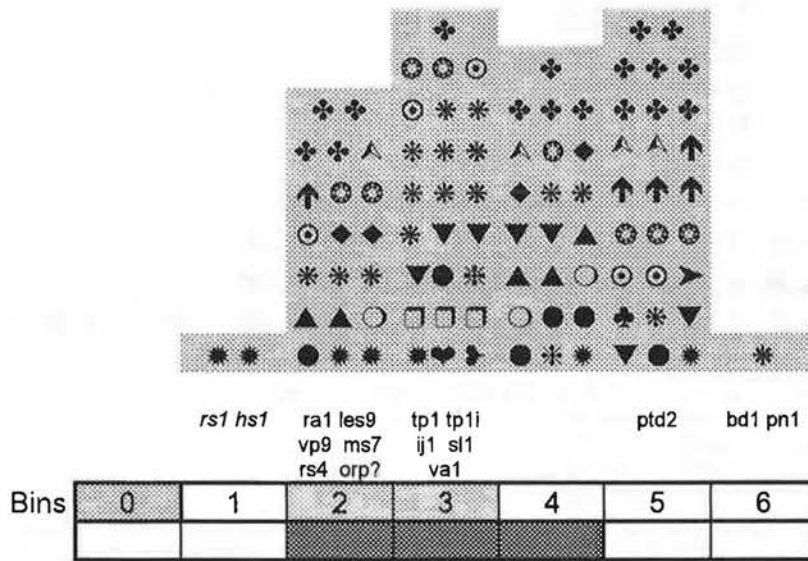
Chromosome 5 (121 probes)



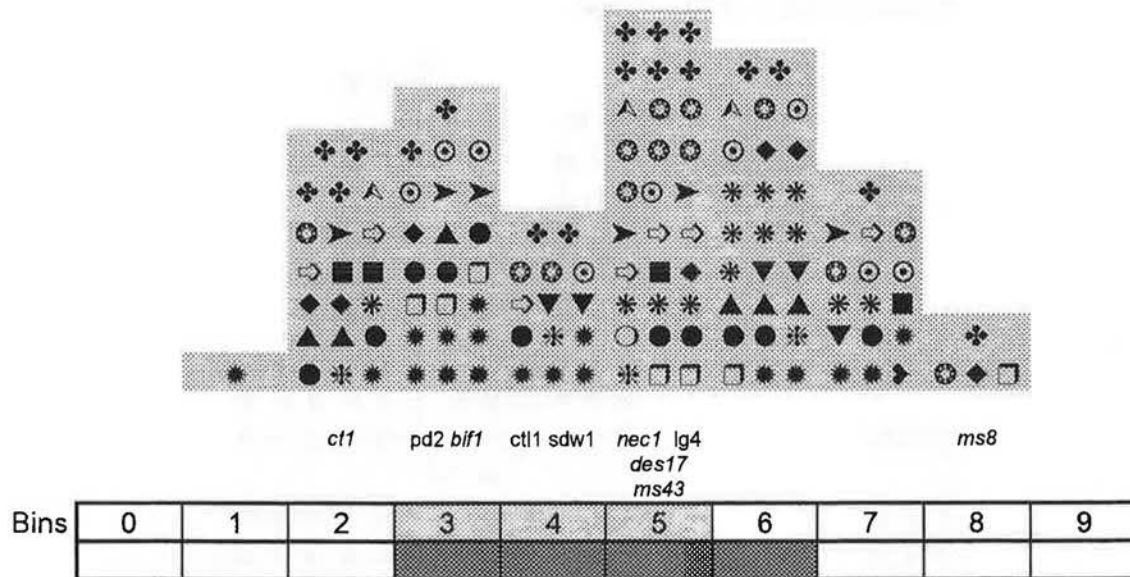
Chromosome 6 (123 probes)



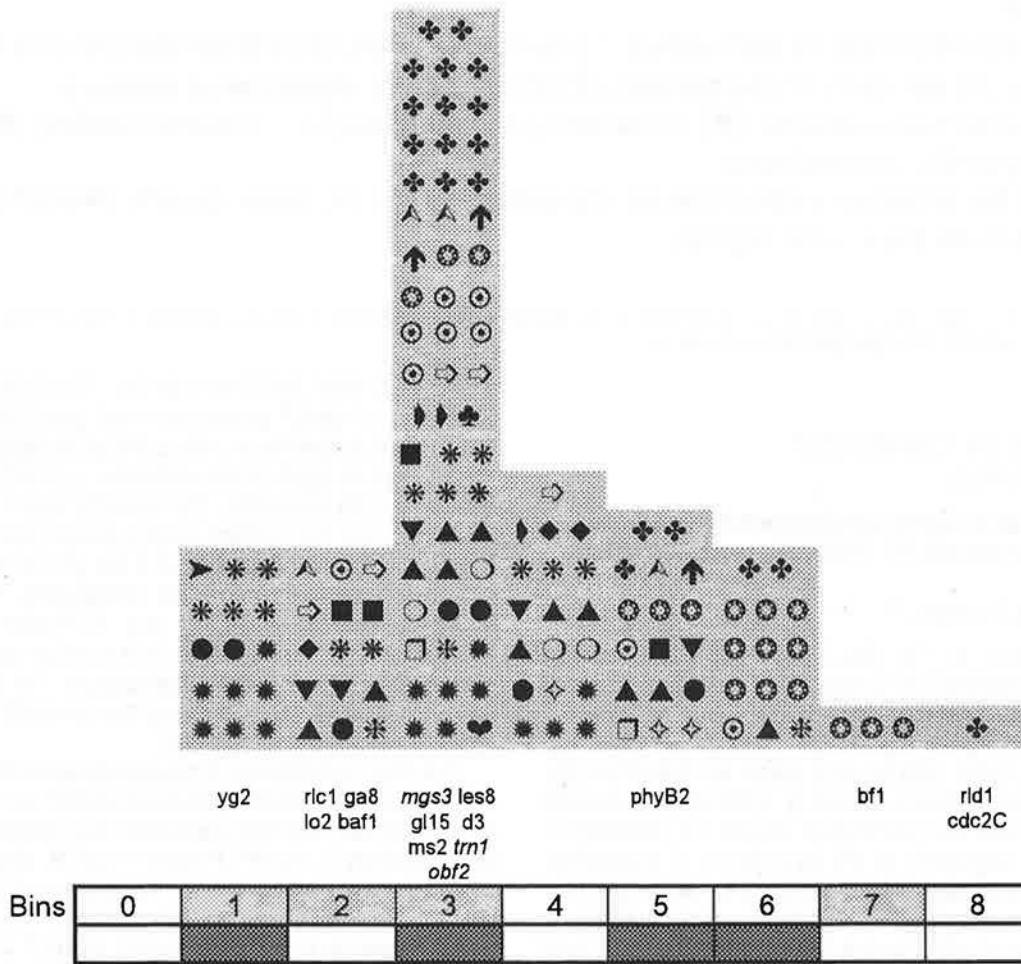
Chromosome 7 (79 probes)



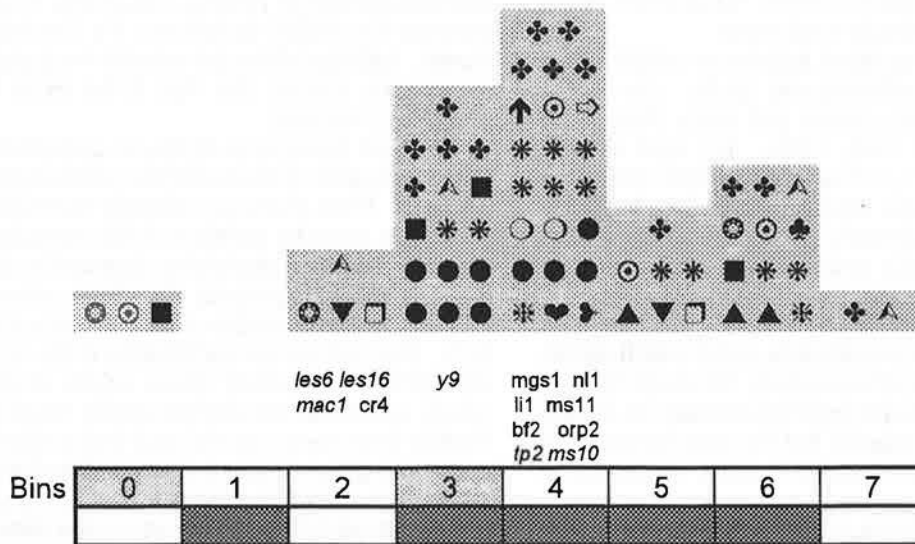
Chromosome 8 (142 probes)



Chromosome 9 (133 probes)



Chromosome 10 (72 probe)



Plant height and earliness/maturity: (♣) plant height; (♠) ear height; (♣) internode length and number; (⊙) days, heat units and growing degree days to anthesis; (⊙) days, heat units and growing degree days to silking; (♣) anthesis - silking interval; (↔) other traits for growth rate; (♣) stalk strength.

Plant body and inflorescence architecture: (□) number of tillers; (♣) leaf number; (↓) root number; (■) leaf size; (♣) ear number; (♣) ear size; (♣) other traits for inflorescence structure.

Grain yield and its components: (▼) kernel size; (▲) kernel weight; (○) kernel number; (●) grain yield; (♣) dry matter accumulation.

Other traits: (♣) immature embryo culture; (□) anther culture; (♣) pollen growth; (♥) ABA content; (♣) ABA effects on plant water regime.

Figure 1. Distribution of QTLs of plant growth and development along corn chromosomes. Shaded bins comprise the homeotic genes expressing transcription factors (the upper row) and the genes and QTLs for resistance to pathogens and phytophagous insects (the lower row).

NEW HAVEN, CONNECTICUT
Yale University

***Rough sheath2* encodes a MYB-domain protein that negatively regulates meristem-specific KNOTTED1-like homeobox proteins in the leaf**

--Timmermans, M, Nelson, T

Characterization of the *leafbladeless1* (*lbl1*) mutant phenotype suggested that *Lbl1* is required to establish adaxial cell identity in leaves and leaf-like lateral organs (Timmermans et al., MNL 71:66, 1997; Timmermans et al., manuscript in preparation). In the absence of LBL1 activity, cells obtain an abaxial identity which results in the formation of radially symmetric, abaxialized leaves. Less severe leaf phenotypes include the formation of ectopic laminar outgrowths at the boundaries of abaxialized sectors on the adaxial leaf surface, and the bifurcation of leaves. Immunolocalization using a KNOTTED1 (KN1) specific antibody on transverse sections of wild type and *lbl1* mutant apices at the level of the incipient leaf showed that the region in which cells do not accumulate KN1 protein is far smaller in *lbl1* mutant apices. Therefore, *Lbl1* or the establishment of the adaxial/abaxial axis is required for the downregulation of KN1 in the meristem, i.e. for the propagation of founder cell recruitment. Whether *lbl1* affects *kn1* expression directly or indirectly is not known.

The *Phantastica* (*phan*) mutant in *Antirrhinum* exhibits similar defects in adaxial/abaxial patterning and results in the loss of adaxial cell identity in leaves, sepals and petals (Waites and Hudson, Development 121:2143, 1995). The most extreme manifestation is the development of radially symmetric, abaxialized leaves, whereas less severely affected *phan* leaves frequently develop ectopic laminar outgrowths on the adaxial leaf surface surrounding patches of abaxial epidermis. In addition, the *phan* mutation results in the misregulation of *kn1*-like homeobox genes in *Antirrhinum* (A. Hudson, personal communication). The *Phan* gene has recently been cloned and was shown to encode a MYB-domain protein (A. Hudson, personal communication). We cloned the *Phan* homolog from maize, because the similarities between the *lbl1* and *phan* mutant phenotypes suggested that the *phan* homolog may either be *lbl1* or a gene functioning in a similar developmental pathway.

We screened a cDNA library derived from vegetative apices and young leaf primordia using the region of the *Phan* cDNA

encoding the MYB-domain as a probe. Two positive cDNAs were isolated from 1-2x10⁶ plaques screened. One cDNA clone is 1.4 kb in length and potentially encodes a full length protein. The 107 aa MYB-domain is highly conserved between PHAN in *Antirrhinum* and maize, 93% aa identity. The non-MYB region of the protein is less conserved, but contains several conserved motifs in the N-terminal portion of this domain and the C-terminal 120 aa have 68% identity between maize and *Antirrhinum*. Probes derived from the region encoding the MYB-domain hybridize predominantly to a single locus in the maize genome and less strongly to a second locus. Interestingly, the fragment of the maize *Phan* cDNA clone encoding the non-MYB region is highly repetitive within the maize genome.

The *Phan* homologs were mapped using the BNL recombinant inbred populations. Neither homolog mapped near the *lbl1* locus on chromosome 6S. The major band mapped on chromosome arm 1S in close proximity to the RFLP marker *npi598*, whereas the minor band mapped to chromosome 8L. Comparison of the RI-line-based map with the genetic map, placed the *phan* locus in close proximity to the recessive leaf mutant *rough sheath2* (*rs2*). Southern analysis using DNA from plants homozygous for the *rs2-R* allele showed that these plants carried a deletion in the *phan* locus. Similar analysis of plants homozygous for the *rs2*-90* allele, which was isolated in a directed tagging experiment with *Mutator* (R. Schneeberger and M. Freeling, unpublished results), showed the presence of a *MuDR* insertion near the C-terminus of the MYB-domain. Additional alleles are currently being analyzed, but these observations indicate that *Rs2* is the maize homolog of the *Antirrhinum Phan* gene.

Recessive mutations at *rs2* lead to a roughening of the auricle and to expression of sheath-like characters along lateral veins in the blade. These phenotypes strongly resemble the phenotypes induced by dominant mutations in the homeobox gene *Rough sheath1*. Additional phenotypes observed in *rs2* mutant plants resemble the morphological aberrations caused by dominant mutations in other homeobox genes (e.g. *Hairy sheath frayed1* and *Kn1*). The most severe manifestation of the *rs2* mutation is the development of extremely narrow leaves, which appear nearly radially symmetric and express mostly midrib-like characters. Whether these narrow leaves result from a defect in founder cell recruitment or from a failure to establish leaf identity throughout the incipient primordium remains to be determined. However, *rs2* causes cells within the leaf to maintain a less determined fate.

In situ hybridization on wild type shoot apices using the MYB-

domain encoding fragment of the *Rs2* cDNA as a probe showed the accumulation of *Rs2* transcripts throughout the P1 primordium and along the major vascular bundles in P2 to P4 primordia. *Rs2* transcripts could not be detected in the apex and in leaf founder cells. Absence of transcripts in the P0 incipient leaf is unexpected because leaf initiation is affected in severe *rs2* mutant plants. The accumulation of *Rs2* transcripts in the vasculature is consistent with the *rs2* mutant phenotype, in which only cells overlying the lateral vascular bundles adopt less determined fates. Taken together, these observations suggest a role for the MYB-domain protein encoded by *Rs2* in the negative regulation of one or more homeobox proteins in the leaf. We are currently testing this hypothesis by analyzing the expression patterns of *kn1*-like homeobox genes in shoot apices of wild type and *rs2* mutant seedlings.

***midribless1 (mrl1)* is required for the correct timing of vascular initiation and the coordination of subsequent developmental events in the maize leaf**

--Jankovsky, JP, Nelson, T

Two mutant alleles of the *mrl1* locus have been identified. *mrl1-1* was isolated from a Robertson's Mutator stock maintained by Paul Chomet and Brenda Lowe at Dekalb Plant Genetics. *mrl1-2* is a reference allele maintained by the Maize Genetics Cooperation Stock Center. RFLP linkage analysis using *mrl1-1/mrl1-1* individuals of selfed progeny from outcrosses of *mrl1-1/mrl1-1* to A158 placed the *mrl1* locus on the short arm of chromosome 2, within 1cM of *B*.

mrl1/mrl1 plants exhibit partial or total loss of the lignified midrib structure in the leaf blade. In *mrl1-1* plants the loss of midrib in the leaf blade occurs primarily in juvenile leaves and only occasionally in adult leaves. In *mrl1-2* plants, the midrib loss is more uniform among leaves, although the first adult leaves often retain a complete midrib. Only the midribs of the leaves are affected in these alleles. The husk leaves, glumes, coleorhiza and other leaf-like structures are unaffected. The auricle is affected in leaves of both alleles which show complete loss of midrib. In wild type leaves, the auricle is visible on the abaxial side of the leaf as two wedge-shaped areas of tissue above the ligule that touch at a point at the midrib. In *mrl1* leaves which have no midrib, the auricle forms an uninterrupted broad band across the leaf. The vascular pattern is also affected in *mrl1* leaves. In wild type leaves, the vascular pattern in the midrib region is distinct from that found in the rest of the leaf blade. Each pair of lateral veins in most areas of the blade is separated by 10-30 minor veins (depending on the age and specific region of the leaf). In the midrib region, each lateral vein pair is normally separated by only one or two minor veins. Affected leaves in *mrl1* mutant plants are slightly wider than their wild type siblings due to a larger number of correctly spaced minor veins between each pair of lateral veins in the mid region. The resulting vein pattern resembles the marginal blade region instead of the distinctive vein pattern in the midrib region.

The cell divisions that lead to the formation of the midrib in wild type leaves are initiated directly adaxial to the developing midvein. In *mrl1* leaves, these cell divisions are delayed. A survey of cross-sections of *mrl1* leaf primordia of varying ages suggests that there is variation in the age at which midrib initiation begins. In some *mrl1* leaves, the first divisions adaxial to the midvein will be visible midway through P3 (about half a plastochron late). In other leaves, initiation of midrib development is delayed a full

plastochron, or in cases of completely midribless leaves, delayed indefinitely. This correlates with the fact that there is a large variation in the final amount of midrib that is present in *mrl1* leaves and suggests that the longer the delay in midrib initiation, the smaller the final midrib will be in the mature leaf. These observations imply that the midribless phenotype of *mrl1* is due to a delay in the initiation of cell divisions involved in the formation of the midrib and not due to a later arrest of a normally developing midrib.

The first cell divisions of midrib formation occur directly adaxial to the midvein in wild type leaves. It is therefore possible that there may be a connection between the positioning and development of the midvein and the formation of the midrib. In wild type leaves, the midvein is initiated in the basal portion of the leaf primordium at the end of P1. In *mrl1* mutant plants, there is a delay in the initiation of midvein and of subsequently initiated flanking lateral veins. The delay ranged from one half to one plastochron, such that the midvein was initiated in mid or late P2 instead of late P1. We suggest that the variable delay in vascular initiation results in a corresponding delay in the initiation of cell divisions for midrib formation adaxial to the developing midvein in late P2 and to corresponding degrees of midribless phenotype.

NOVOSIBIRSK, RUSSIA

Institute of Cytology and Genetics

Perspectives of developing apomixis in maize

--Sokolov, VA, Lukina, LA, Khatypova, IV

Creation of apomictically reproducing maize hybrids makes it possible to obtain high yields with the fixed heterosis effect characteristic of the F1 with minimum expenditures for seed-farming. For this purpose D.F. Petrov proposed the adoption of genes controlling asexual reproduction in a wild maize relative - *Tripsacum* (Petrov, Nauka, 1964). At his laboratory hybrids of these plants with a diverse ratio of the parents' genomes and exhibiting apomictic reproduction type were obtained (Petrov et al., Genetika 3:58-71, 1968). Due to a number of negative qualities received from *Tripsacum* they could not be used in practical selection. However, in the apomictic progeny with the highest frequency plants were revealed, developing from reduced gametes either apomictically or in a sexual way. Among such plants selected were forms carrying 1, 2 or 3 chromosomes of *Tripsacum* along with 20 chromosomes of maize (Petrov et al., 1984). Later several apomictic lines of maize were reported, isolated thanks to crossover introgression into its genome of appropriate genes from *Tripsacum* chromosomes (Petrov et al., Dokl. Akad. Nauk 281:509-512, 1985; Fokina, N. I. Vavilov VOGIS 5th Congress Part 2:217-218, 1987). However by hybridization analysis these lines were shown not to carry the apomixis character (Yudin and Sokolov, Dokl. Akad. Nauk 309:219-222, 1987).

The analysis of the results obtained by 1987 led us to setting ourselves the task of establishing the role of separate *Tripsacum* chromosomes in cytogenetic control of apomixis in the hybrids. The genomic complements of the apomictic maize x *Tripsacum* hybrid forms known by now have a different number of *Tripsacum* genomes. But in all cases 18 chromosomes were the least number ensuring ability for apomictic development and at the same time carrying the negative characters of the wild parent. In this connection, as the first step in the way of apomictic maize

creation, we tried to increase the frequency of occurrence in the progeny of forms with a reduced number of *Tripsacum* chromosomes by changing a ratio of genomes of maize and *Tripsacum* in the hybrids. The report presented here contains the results of this experiment.

As initial material, 38-chromosome forms were taken, obtained earlier at our laboratory by fertilization of reduced egg-cells of 56-chromosome plants with pollen of diploid maize and including in their genomic complement two sets of maize chromosomes and one set from a tetraploid ($2n=72$) apomictic *Tripsacum* variety. These lines for a long time year after year have been producing 97 to 100% of apomictic progeny, evidencing that the haploid set of *Tripsacum* chromosomes is enough to control this character.

From our experience of work with the hybrids we knew that an increase in maize : *Tripsacum* genome ratio resulted in an increase in probability to develop progeny with chromosome reduction. So to solve our task 58-chromosome unreduced hybrids were taken, obtained by pollination of 38-chromosome plants with pollen of tetraploid maize. The genomic complement of these hybrids includes 40 maize chromosomes and 18 *Tripsacum* chromosomes, corresponding to a 4:1 ratio of the haploid sets. The high level of reduced progeny in such plants permitted us to hope for successful recovery among these of maize addition lines, where to maize genomes were added 1 to 18 *Tripsacum* chromosomes in different combinations. The total number of possible variants making 257,442 in this case would be impossible to examine cytologically. Only recovery of apomictic plants with a reduced number of *Tripsacum* chromosomes considerably simplified our task. In the progeny of the 58-chromosome unreduced hybrids we managed to isolate 39-chromosome apomictic plants in which to maize genome sets were added only 9 chromosomes of *Tripsacum*, reducing the number of combinations of variants to 511. By means of obtaining the 39-chromosome forms we managed to pass from the 56-chromosome hybrids having 36 chromosomes of *Tripsacum* in their complement to hybrids with 9 chromosomes of *Tripsacum*.

From these sufficiently stable 39-chromosome apomicts, by pollinating them with pollen of tetraploid maize, a further increase in maize : *Tripsacum* genome ratio was obtained in unreduced 59-chromosome hybrids. The frequency of such hybrid occurrence is about 0.5 %. To enhance the manifestation of the sexual reproduction character 79-chromosome hybrids (70 maize chromosomes + 9 *Tripsacum* chromosomes) were derived. From these we succeeded in developing plants with an imbalanced set of maize chromosomes (i.e. not divisible by the haploid set) and with an addition of separate chromosomes of *Tripsacum* (from 3 to 5). These plants with a high female sterility yield fertile pollen. We have so far failed to isolate apomicts among them by reason of small volume of this material. However, high tendency to sexual reproduction of the 79-chromosome hybrids producing more than 50 % of reduced progeny makes it really possible to increase this volume. Besides, as we deal with Antennaria-type apomixis due to partial meiosis with the genomic complement imbalanced in the 39-, 59-, 79-chromosome plants, crossover exchange of sites is possible between maize and *Tripsacum* chromosomes that are partially homeologous owing to their distant relationship (Koltunow, Plant Cell 5:1425-1437, 1993; Galinat, Ann. Rev. Genet. 263:1598-1600, 1971). If in such a process a chromosome participates carrying gene(s) for apomixis its transition under the control of a maize centromere is possible.

In addition to the above - mentioned 39-chromosome plants

with a high level of apomictic progeny (95 - 100 %), different 39- and 49-chromosome hybrids (30Mz + 9Tr; 40Mz + 9Tr) were developed by us. These lines were derived from the only parthenogenetic reduced hybrid originating from a 58-chromosome plant (40Mz + 18Tr). It should be noted that this plant (20Mz + 9Tr) was highly sterile and, when pollinated, it yielded one single plant with 39 chromosomes (30Mz + 9Tr), serving as the ancestor of the above lines. It should be stressed that the chromosome complement in these lines does not coincide with that in the formerly developed hybrid. For that reason they produce only 10-15% of apomictic progeny and are partially male sterile. These results confirm John Carman's hypothesis of polygenic control of apomixis and in that case the approach to the creation of apomictic maize has been revised by us about which a separate report will be made on the basis of the new experimental results (Carman, Biol. J. Linnean Soc. 61:51-94, 1997).

The work was done (partially) at the expense of the "Priority directions of genetics" grant.

RALEIGH, NORTH CAROLINA
North Carolina State University

Partial isolation and characterization of the maize alternative oxidase gene

--Polidoros, AN, Mylona, PV, Scandalios, JG

Plant mitochondria contain an alternative pathway for the transfer of electrons from reduced ubiquinone to oxygen that bypasses cytochrome c oxidase and is associated with the presence of an alternative oxidase found in the inner membrane of mitochondria from all higher plants. Recent evidence suggests that alternative oxidase has a potentially important role in protection against oxidative stress. In order to characterize the maize alternative oxidase, we amplified part of the gene with PCR, and DNA template from different maize inbred lines (Figure 1),

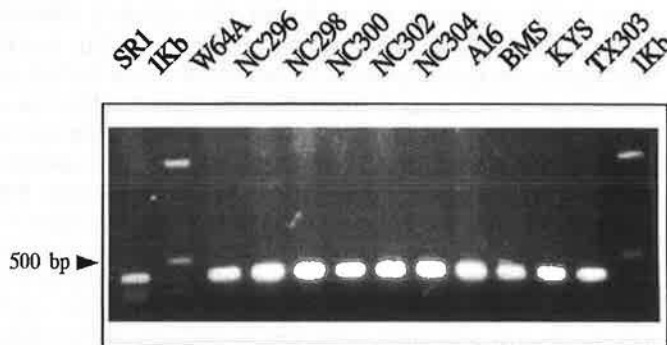


Figure 1. Electrophoretic analysis of PCR products. PCR was performed using a set of degenerate primers, which amplify part of the maize alternative oxidase gene, and template DNA from the indicated maize lines and the tobacco line SR1. PCR conditions were 3 min denaturation at 95°C followed by 35 cycles of 30sec denaturation at 95°C, 1min annealing at 58°C, and 1min extension at 75°C. The last extension step was for 5 min to ensure completion of the reaction. A single band of fairly similar size and intensity can be observed.

using degenerate primers designed from two highly conserved regions of alternative oxidase from other plant species. The degenerate primer specific to the 5' conserved region was 5'-GCIATGATGYTIGARACIGTRCG-3' and corresponds to the conserved amino acid sequence AMMLETVA. The degenerate primer specific to the 3' end of the gene was 5'-RTCICGRTGRTGWGCCTCRT-3' and its complementary strand

A

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S. guttatum GCGATGATGCTGGAGACGGTGGCGCGGTGCCGGGCATGGTGGGCGGGTACTCCTCCAC
Z. mays GCGATGATGCTGGAGACGGTGGCGCGGTGCCGGGCATGGTGGGCGGCATGCTCCTCCAC

S. guttatum CTC AAGTCCCTCCGCCGCTTCGAGCACAGCGGGGTTGGATCAGGGCCCTCCTGGAGGAG
Z. mays CTC GGCCTCACTCCGCCGCTTCGAGCACAGCGGGGTTGGATC G G G C C T C T G G A G G A G

S. guttatum GCCGAGAACGAGCGGATGCACCTGATGACCTTCATGGAGTGGCGCAGCCCGGTGGTAC
Z. mays GCCGAGAACGAGCGGATGCACCTGATGACCTTCATGGAGTGGCGCAGCCCGGTGGTAC

S. guttatum GAGCGGGCGCTGGTGTGGCGGTGCAGGGGGTCTTCTCAACGCCTACTTCTGGGGTAC
Z. mays GAGCGCGCGCTGGTGTGGCGGTGCAGGGGGTCTTCTCAACGCCTACTTCTGGGGTAC

S. guttatum CTGCTCTCCCCAAGTTCGCCACCCGGTTGTGGGCTACCTGGAGGAGGAGGCCATCCAC
Z. mays CT G G T T C C C C C A A G T T C G C C C A C C C G G T T G T G G G C T A C C T G G A G G A G G A G G C C A T C C A C

S. guttatum TCCTACACCGAGTTCCTCAAGGACATCGACAGTGGGGCCATCCAGGACTGCCCGCCCGG
Z. mays TCCTACACCGAGTTCCTCAAGGACATCGACAGTGGGGCCATCCAGGACTGCCCGCCCGG

S. guttatum GCCATCGCCCTGGACTACTGGCGCTGCCGCAGGGCTCCACCTGCGCGACGTCGTGACC
Z. mays GCCATCGCCCTGGACTACTGGCGCTGCCGCAGGGCTCCACCTGCGCGACGTCGTGACC

S. guttatum GTCGTCGCGCGAGACGAGGCACACCAC
Z. mays GTCGTCGCGCGAGACGAGGCACACCAC

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B

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S. guttatum AMMLETVAAVPGMVGGVLLHLKSLRRFEHSGGWIRALLEEAENERMHLMTFMEVAQPRWY
Z. mays AMMLETVAAVPGMVGGVLLHLKSLRRFEHSGGWIRALLEEAENERMHLMTFMEVAQPRWY

S. guttatum ERALVLA VQGVFFNAYFLGYLLSPKFAHRVVGYLEEEAIHSYTEFLKDI DSGAIQDCPAP
Z. mays ERALVLA VQGVFFNAYFLGYLLSPKFAHRVVGYLEEEAIHSYTEFLKDI L E A G K I E K R P A P

S. guttatum AIALDYWRLPQGSTLRDVTVVRADEAHH
Z. mays AIALDYWRLPQNSTLRDVTVVRADEAHH

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Figure 2. Alignment of the alternative oxidase nucleotide sequence (A) and amino acid sequence (B) from *S. guttatum* and the maize NC300 PCR product. The maize nucleotide sequence has been submitted to the GenBank and accession number assignment is AF040566. Different nucleotides and amino acids are highlighted in the maize sequences.

encodes the conserved amino acid sequence DEAHHRD. PCR products of the expected size (450bp in the tobacco SR1 control) were observed on an agarose gel with all the templates tested (Figure 1). The PCR bands were subcloned and these of the maize lines NC300 (Goodman, M., Proc. Annu. Corn Sorghum Ind. Res. Conf. 47:47-64, 1992), W64A, A16 and the tobacco line SR1 were sequenced. The sequence derived from the tobacco line was identical with the respective region of the alternative oxidase sequence from SR1 (X79768) in the GenBank. The sequence derived from the maize line W64A had no similarity with other alternative oxidase sequences and may represent a pseudogene or misprimed amplification. The sequences from NC300 and A16 were very similar and that of NC300 was selected for further analysis. The NC300 PCR product had 81.8% identity with the respective nucleotide sequence of *S. guttatum* (M60330), and 87.2% amino acid identity, as was revealed by sequence alignments (Figure 2). Using the cloned fragment of the maize alternative oxidase gene derived from the line NC300 as probe, we performed RFLP analysis with four inbred maize lines. Results indicated that the gene is polymorphic among the genotypes examined (data not shown).

Alternative oxidase in other organisms is inducible by salicylic acid (SA). Thus, we examined if the maize alternative oxidase gene is also inducible by SA. We treated 10 dpi W64A and NC300 maize seedlings with 1 and 5 mM SA, either hydroponically or with vacuum infiltration. After 24 h treatment we examined the effects on alternative oxidase transcripts. The results showed that multiple transcripts were observed and each genotype responded differently to SA (Figure 3). In all cases

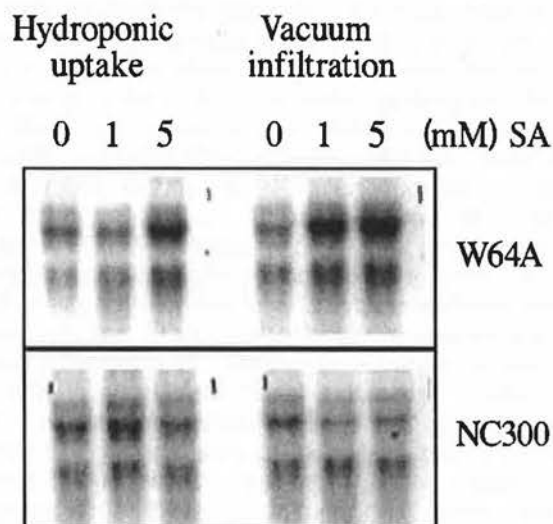


Figure 3. Alternative oxidase inducibility with SA. Northern analysis of RNA (20µg) isolated from 10 dpi W64A and NC300 maize seedlings treated with 1 and 5 mM SA either hydroponically or with vacuum infiltration. The NC300 derived alternative oxidase probe was used in this experiment. Note the 3 bands clearly detected with the probe. The upper band is fairly constant in all the lanes. The middle band is the most intense and corresponds to the expected size of the alternative oxidase transcript. Different induction is observed between the two lines.

three bands were observed. The upper band was weak and did not seem to alter with treatments. The middle and lower bands were clearly responding, with the responses of the middle one more prominent. The middle band corresponds to the expected alternative oxidase transcript size if the maize transcript is

similar with that of other plants. The W64A alternative oxidase transcripts were highly increased in the vacuum infiltrated seedlings with both 1 and 5 mM SA, when in the hydroponic treatment they increased only in the higher SA concentration. However the NC300 alternative oxidase transcripts increased only in the 1 mM SA hydroponic treatment. In the vacuum infiltration treatments the middle band seemed to decrease. Using the PCR-derived maize alternative oxidase probe we plan to isolate the cDNA and genomic DNA of the gene and characterize its responses to oxidative stress.

ST. PAUL, MINNESOTA
University of Minnesota

Spontaneous activation of transposable elements following an interracial cross in maize

--Vladutu, CI, Phillips, RL

In order to initiate a QTL analysis for several traits, we developed a Gaspé Flint (GF) x N28 F2 population. The F2 plants, all wild type, were selfed and the resulting F3 families were planted in 1996 in two replications in the field. Among the 99 F3 families, 6 segregated for obvious plant mutations: branched-silkless-like, chronic wilting, yellow-green, pigmy-like, male-sterility, white-sheath-like and stunted plants. Another F3 family gave rise to an andromonoecious dwarf. Two other putative mutations (earless and disease mimic) need further confirmation. The branched-silkless-like and the chronic wilting mutants segregated within the same F3 family. Two of the mutations showed somatic instability; some of the white-sheath-like mutants had dark green stripes in the internodes, leaf sheath and blade and one of the plants of the F3 family segregating for male-sterility had a chimeric tassel with fertile and sterile branches. In another F3 family, one plant had a chimeric tassel for anther pigmentation. Within each of the families segregating for plant mutations, several plants were selfed and occasionally outcrossed. Within the families that did not segregate for any obvious plant mutation, only one or two plants were selfed. Upon selfing, in four other families, F3 plants were found to segregate for pale yellow (*y*), floury (*fl*), brittle (*bt*) and shrunken (*sh*)-like kernels. Some of the *fl*-like kernels showed patches of wild type endosperm. In the family with the andromonoecious dwarf, one F3 plant was found to segregate for viviparous (*vp*) kernels. F3 plants within several other families segregated for different types of defective kernels. Eighteen GF x N28 F3:4 families were grown in the field in 1997. Five additional mutations arose among the 18 families. Four mutations (albino, yellow-stripe, narrow-leaf and knotted) appeared within four different F3:4 families of previously identified mutants; a zebra-crossband mutation occurred in one of the six F3:4 families derived from F3 families that in 1996 did not segregate for visible mutations. In 1997, in addition to white-sheath-like, male-sterile and *fl*-like, two other mutations, (yellow-green and narrow-leaf) showed somatic instability.

Considering that all F2 plants and F3 kernels were wild type, and that somatic instability occurred with some of the mutations, the high rate of forward mutations (resembling the behavior of Robertson's *Mu* stock) is likely the result of the activation of transposable elements (TEs) in the F1 plant. Since the retroelements are not expected to excise and thus induce reversion to the wild phenotype, and no excision events have been

so far reported for MITEs, the unstable mutations, at least, are probably caused by DNA-elements. It seems that the putative TE activity that occurred in the F1 plant was maintained in some of the F2 plants. The fact that no two F3 families segregated for the same mutation suggests that the TE insertional activity occurred late in the development of the GF x N28 F1 plant. Accurate estimates of mutation rates can not be computed due to the small size of the F3 population. However, among 300 N28 x N28E (N28E is an early backcross derivative of N28, having two chromosomal segments retained from GF) F3 families grown in 1993 in the same location, no mutation had been identified. If the N28 x N28E F3 population would have had one F3 family segregating for a mutation, the proportion (~7%) of GF x N28 F3 families segregating for a different plant mutation would be at least 20 times higher.

In the summer of 1997, several of the mutants were outcrossed with different inbred lines and complementation tests were performed for some of the kernel mutations and the branched-silkless-like mutation (the testers were kindly provided by the Maize Stock Center). The complementation tests revealed that the *sh*-like, *bt*-like and *vp* mutations had occurred in the *Sh1*, *Bt2* and *Vp1* genes respectively, all of which have been cloned (Werr et al., EMBO J. 4:1373-1380, 1985; Bae et al., Maydica 35:317-322, 1990; and McCarty et al., Plant Cell 1:523-532, 1989, respectively). The result of the complementation test involving branched-silkless-like mutation will be apparent next summer (1998).

In the summer of 1997, somatic reversion assays were initiated, using testers (generously provided by the Maize Stock Center and P.A. Peterson) for *Ac*, *Spm*, *Mu*, *Mrh* and *Dt* activity, having the corresponding defective elements in anthocyanin genes (*A1*, *A2*, *Bz1*, *Bz2* and *C1*) expressed in the aleurone layer. Mutability assayed by somatic reversion and mutagenicity do not always correlate. However, since the events leading to mutagenicity appeared to occur in the F1, we are testing for the existence of autonomous element activity in the GF x N28 F1 compared to the parents (GF and N28). In order to avoid potential confusion between the variegation caused by TE excisions with the mottling effect caused by the imprinting of *R* alleles transmitted through the male, the transposon testers were used as the female parent. Since both GF and N28 are homozygous wild-type for *A1*, *Bz1* and *Bz2*, the testcrosses for *Dt*, *Mrh* and *Mu* activity have to be carried on for one more generation (i.e. the hybrids between the testers and the tested material have to be backcrossed, as males, onto the testers as females). Negative results (no reversions) for the three genotypes have been obtained with the *Ac* testers. *Spm* activity in half of the kernels was detected in a testcross with GF but not with GF x N28 F1 and N28. This result indicates segregation for an active *Spm* element within GF. GF is not an inbred line but a cultivar obtained through mass selection. Thus, it is expected to be heterogeneous/heterozygous, and signs of genetic instability (segregation for *su1*, *y1* and *lg*) within GF had been noticed in the past (Vladutu, MSC, 1996). However, since no *Spm* activity occurred in testcrosses with GF x N28 F1, it is unlikely that *Spm* activity had induced the burst of mutations in the GF x N28 progenies.

The RFLP and the phenotypic data provide compelling evidence against the possibility of potential contamination with transposon stocks as the source of genetic instability in the GF x N28 progenies. Also, limited cytological analysis of the

microsporocytes from the GF x N28 F1 plants has not detected structural heterozygosity (such as inversions or translocations) that could have triggered chromosome breakage and subsequently activated TEs. Thus, the "genomic shock" that activated TEs in GF x N28 progenies was probably due to heterozygosity per se at yet unknown host loci.

GF is a Northern Flint open-pollinated population. N28 is a Corn Belt Dent line. Since the parental cultivars belong to historically and economically important maize races and have been used in recent breeding programs, this case has relevance in terms of the induction of de novo genetic variability in maize improvement/evolution. The high rate of forward mutations that have occurred in progenies of the above cross shows that the high level of mutagenic activity is not an exclusive attribute of Robertson's *Mu* stock, and suggests that heterozygosity per se at yet unidentified host loci can induce the activation of silent TEs. Since complementation tests have shown that putative TE insertions have occurred in *Sh1*, *Bt2* and *Vp1* genes, all of which have been cloned, the identification and characterization of the activated TEs may be straightforward.

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Bulked segregant analysis confirms the importance of the region near *umc89a* for days to pollen shed in maize

--Tuberosa, R, Salvi, S, Phillips, RL

The moderate to high heritability of the number of days from planting to pollen shed (DPS) in maize makes it an interesting candidate for a molecular marker analysis of the quantitative trait loci (QTLs) underlying its expression. Previous work carried out at the University of Minnesota identified QTLs for DPS on chromosomes 1, 2, 3, 5, 7, and 8 (Kim, PhD thesis, University of Minnesota, 1992; Phillips et al., Proc. 47 Annu. Corn Sorghum Ind. Res. Conf., pp 135-150, 1992; Parentoni, MS thesis, University of Minnesota, 1993). The most important QTL for DPS was on chr. 8L in the region near *umc89a*. Surveying a number of unrelated crosses will increase the chances of identifying major QTLs representing valuable targets for gene cloning. Bulk segregant analysis (BSA; Michelmore et al., PNAS 88:9828-9832, 1991) offers an interesting opportunity to investigate specific QTLs in populations for which genetic maps are not available. In this case, RFLP probes tagging QTLs in one population can be tested in different populations segregating for the same trait. Our objective was to use BSA in order to verify whether the chromosomal regions influencing DPS in the work carried out by Phillips and collaborators at the University of Minnesota were also involved in the control of DPS in three F2 populations segregating for DPS. In particular, we wanted to verify the role of the QTL for DPS revealed by *umc89*.

Three F2 populations (Mt42 x NC254, A679 x NC254, and A86-9 x R225) were evaluated at the St. Paul experimental location of the Department of Agronomy and Plant Genetics, University of Minnesota. The number of F2 plants evaluated was 520 in Mt42 x NC254, 350 in A679 x NC254, and 280 in A86-9 x R225. All F2 plants were scored for DPS and selfed. Among the plants which set seed in each F2 population, the 15 earliest and the

15 latest in DPS were selected. The corresponding F3 progenies were scored for DPS at the experimental location of the Department of Agronomy, University of Bologna. The DNA bulks were obtained by bulking equal amounts of the DNA of the 15 earliest and the 15 latest F2 plants. The DNA of each bulk and that of the parent lines was restricted with two to three restriction enzymes (*Bam*HI, *Eco*RI, *Eco*RV, and *Hind*III). Filters were probed with 14 genomic clones (*bnl6.22*, *bnl6.25*, *umc6*, *umc11*, *umc27*, *umc30*, *umc35*, *umc51*, *umc54*, *umc89*, *umc90*, *umc113*, *umc114*, and *umc124*) which showed RFLPs associated with QTLs for DPS in the work of Kim (PhD thesis, University of Minnesota, 1992). Filters were also probed with 24 clones (*bnl5.40*, *bnl5.71*, *bnl15.21*, *csu4*, *csu6*, *csu13*, *csu29*, *csu46*, *csu61*, *csu86*, *csu94*, *csu109*, *csu133*, *csu148*, *umc15*, *umc31*, *umc59*, *umc84*, *umc93*, *umc104*, *umc119*, *umc128*, *umc139*, and *umc161*) mapping to regions not represented by the previous group of clones.

Among the probes which showed polymorphism between parent lines of each cross, marked differences in intensity between the bands of the early- and late-maturity bulks were revealed by *csu61*, *umc11*, *umc15*, *umc27*, *umc30*, *umc51*, *umc54*, and *umc89* (Table 1). These eight probes were all but two (*csu61* and *umc15*) among those reported by Kim (PhD thesis, University of Minnesota, 1992) to be linked to QTLs for DPS in maize crosses different from those herein tested. Differences in band intensity of early and late bulks were not evidenced by any of the remaining probes. A key factor when using BSA for quantitative traits is the correct scoring of the phenotypes. In our study, DNA pools were formed according to the data for DPS of the F2 plants. The major discrepancies between the phenotypic classification of the F2 plants and their F3 progenies involved two

Table 1. Probe-enzyme combinations with marked differences in the intensity of the polymorphic bands between the bulked DNAs of the F2 plants which were classified as early and late in DPS in each cross. The combinations positive to BSA have been underlined.

Probe	Chr.	Mt42 x NC254	A679 x NC254	A86-9 x R225
<i>umc11</i>	1	<u><i>Bam</i>HI</u>	<u><i>Hind</i>III</u>	<u><i>Bam</i>HI</u>
<i>csu61</i>	1	N.P.	N.P.	<u><i>Eco</i>RI</u>
<i>umc15</i>	4	<u><i>Eco</i>RI</u>	N.P.	N.P.
<i>umc27</i>	5	<u><i>Hind</i>III</u>	<i>Eco</i> RV	<u><i>Hind</i>III</u>
<i>umc54</i>	5	<i>Eco</i> RI	N.P.	<u><i>Eco</i>RI-<i>Hind</i>III</u>
<i>umc51</i>	5	<i>Eco</i> RI	<i>Eco</i> RI	<u><i>Eco</i>RI</u>
<i>umc89</i>	8	<u><i>Dra</i>I</u>	<u><i>Eco</i>RI</u>	<u><i>Eco</i>RI</u>
<i>umc30</i>	8	<u><i>Dra</i>I</u>	<u><i>Eco</i>RI</u>	<u><i>Dra</i>I-<i>Eco</i>RI</u>

N.P.: probe-enzyme combination not polymorphic between parent lines.

A679 x NC254 F3 progenies which were found to be slightly earlier than the average of the early and late F3 progenies, although they derived from F2 plants classified as late. Despite this, Southern analysis with *umc89* showed a marked difference in the intensity of the polymorphism between the bulks.

Our results indicate that BSA using RFLPs may be utilized to identify and/or confirm the role of QTLs when previous information is available on their localization in unrelated populations. Five QTLs controlling DPS in the crosses surveyed by Kim (1992) were likely involved in the control of DPS in at least one of the three crosses herein investigated. Due to the duplicate nature of the maize genome, some of the probes negative to BSA and linked to QTLs for DPS in the work of Kim (PhD thesis, University of Minnesota, 1992), may have revealed RFLPs mapping in regions other than those investigated in his work; in such a case, the presence of a QTL for DPS near the duplicate locus is less likely. *umc11*, *umc30*, and *umc89* revealed differences between the

bulks in two crosses. So far, *umc89a* has been linked to QTLs for DPS in several maize crosses (Abler et al., *Crop Sci.* 31:267-274, 1991; Phillips et al., *Proc. 47 Annu. Corn Sorghum Ind. Res. Conf.*, pp 135-150, 1992; Zehr et al., *TAG* 33:903-911, 1992; Stuber et al., *Genetics* 132:823-839, 1992; Koester et al., *Crop Sci.* 33:1209-1216, 1993; Lin et al., *Genetics* 141:391-411, 1995; Ragot et al., *Crop Sci.* 35:1306-1315, 1995). Genes affecting flowering time are usually divided into two broad categories: i) genes responding to environmental cues, such as temperature and photoperiod, and ii) genes affecting flowering per se, independent of the influence of environmental variables. Although these two types of genes may not be mutually exclusive, the QTL region near *umc89a* likely contains a gene(s) affecting flowering per se. Coding sequences belonging to the category of flowering genes influenced by the growth environment (e.g. photoperiod) are less likely to maintain their effect over a wide range of environments and genetic backgrounds.

Mapping QTLs for ABA concentration in leaves of a maize cross segregating for anthesis date

--Tuberosa, R, Parentoni, S, Kim, TS, Sanguineti, MC, Phillips, RL

The number of days from planting to pollen shed (DPS) is often of critical importance for optimizing crop yields in different environments. The transition from the vegetative to the reproductive phase involves the integration of many environmental and developmental factors. In maize, experimental evidence suggests that genes for plant growth and development are associated into functionally significant clusters (Khavkin and Coe, *TAG* 93:343-352, 1997). As an example, the 5.06-5.07 region on maize chr. 5 contains structural genes (*vp2* and *vp7*) involved in the synthesis of abscisic acid (ABA) as well as QTLs (quantitative trait loci) for DPS, plant architecture, and pollen growth (Khavkin and Coe, *MNL* 68:61-62, 1996). A physiological interpretation for a pleiotropic effect on morpho-physiological traits could be attributed to the presence of one or more QTLs controlling the level of particular plant growth regulators (PGRs) influencing the rate of plant development. Little is known about the effects on DPS of naturally-occurring variation in the endogenous levels of PGRs. The overlap of QTLs of different traits provides evidence for a genetic correlation, either by linkage or pleiotropy, among such traits. The objective of this research was to evaluate 151 F3 families of a cross segregating for DPS to identify QTLs for L-ABA and to investigate their overlap with QTLs for DPS evidenced in the same cross by Parentoni (MS thesis, University of Minnesota, 1993).

F3 families of the cross A662 (early flowering) x B73 (late flowering) were evaluated using a 12 x 13 rectangular lattice replicated twice in trials conducted at Rosemount and Waseca, Minnesota. The parent lines and three B73 backcross-derived lines (A679, A680, and A681) were also included. Leaf samples were collected approximately at stages 2 and 3 according to Hanway's scale (*Agr. J.* 55:487-492, 1963). The concentration of unconjugated ABA was determined using an ABA-specific monoclonal antibody (Quarrie et al., *Planta* 173:330-339, 1988). The analysis of variance was carried out separately for each one of the four "growth stage x location" (sampling) combinations. Because the lattice design was found more effective than the randomized complete block design, means were adjusted accordingly before checking for normality in their distribution.

The linkage map was obtained by Kim (PhD thesis, University of Minnesota, 1992) using 63 *Pst*I RFLP probes. For QTL analysis, the statistical package MAPMAKER-QTL was utilized.

The ANOVA for L-ABA evidenced significant ($P < 0.01$) differences due to genotypes, growth stages, locations, and their interactions. Due to the significance of the second order interaction, QTL analysis was carried out on each individual sampling. In total, seven unlinked QTLs influenced (LOD > 2.0) L-ABA in at least one of the four samplings. Table 1 reports the main characteristics of the QTLs for L-ABA. In Rosemount, the number of QTLs evidenced at stages 2 and 3 did not vary, although different QTLs were revealed. For L-ABA-2, two QTLs were identified on chr. 3 and 6. The LOD score peak (LOD = 2.47) on chr. 3 was between *umc154* and *umc10a*, while the LOD peak (2.54) on chr. 6 was between *umc59a* and *bnl3.03*. In Rosemount, L-ABA-3 was significantly influenced by QTLs on chr. 5 and 8. LOD scores (2.16 and 2.96, respectively) peaked on chr. 5 between *umc54* and *umc108* and on chr. 8 between *umc16b* and *umc7*. A considerable portion (35.8%) of phenotypic variation among F3 families was accounted for by the QTL on chr. 8. In Waseca, the only region with significant effects on L-ABA-2 was on chr. 2, near *umc131*, while four regions significantly affected L-ABA-3. These four QTLs were localized on chr. 1 (near *npi234*), chr. 2 (between *umc5* and *umc88*), chr. 5 (between *umc54* and *umc108*, and chr. 8 (between *umc16* and *umc7*). The R^2 value varied from 10.3 to 34.1%. The support intervals of the two QTLs on chr. 5 and 8 overlapped with those of the QTLs in the same regions which showed significant effects on L-ABA-3 in Rosemount. With the exception of the first sampling in Rosemount, the alleles increasing L-ABA were those of B73, the late parent line of the cross. In both locations, the absolute effect of allelic substitution was of greater magnitude at stage 3. This, in turn, was paralleled by a substantial increase from stage 2 to stage 3 in the percentage of phenotypic variation for L-ABA that was accounted for by each QTL. Other authors have reported that different QTLs control L-ABA at subsequent growth stages in maize (Lebreton et al., *J. Exp. Bot.* 46:853-865, 1995; Sanguineti et al., *Maydica* 41:1-11, 1996). Another important factor which could influence QTLs for L-ABA at subsequent growth stages is the level of water stress experienced by the plants in the time-period prior to sampling. It is worth mentioning that the QTL which was evidenced in Waseca for L-ABA-2 between markers *umc131* and *umc5* on chr. 2, was mapped very close to the position reported for a major QTL influencing L-ABA in two different maize populations (Lebreton et al., *J. Exp. Bot.* 46:853-865 1995; Landi et al., *Proc. XVII Conf on Genetics, Biotechnology and Breeding of Maize and Sorghum, Thessaloniki, 65-70, 1997*). It is likely that other QTLs for L-ABA went undetected due to the low marker density of some regions (eight

Table 1. Main characteristics of the QTLs for leaf ABA concentration in 151 (A662 x B73) F3 families sampled in Rosemount and Waseca at growth stages 2 and 3.

Sampling	Chrom.	Flanking markers	LOD	U ⁽¹⁾	R ²
Rosemount-2	3	<i>umc154-umc10a</i>	2.47	-1.45	10.2
Rosemount-2	6	<i>umc59a-bnl3.03</i>	2.54	-5.13	33.2
Rosemount-3	5	<i>umc54-umc108</i>	2.16	4.18	7.7
Rosemount-3	8	<i>umc16b-umc7</i>	2.96	18.16	35.8
Waseca-2	2	<i>umc131-umc5</i>	3.42	5.58	19.3
Waseca-3	1	<i>npi234-umc23A</i>	4.14	5.11	34.1
Waseca-3	2	<i>umc5-umc88</i>	3.73	16.04	21.9
Waseca-3	5	<i>umc54-umc108</i>	2.26	5.62	10.3
Waseca-3	8	<i>umc16b-umc7</i>	2.37	13.85	17.8

(1): effect (in ng ABA/g d.w.) of substituting a B73 allele for an A662 allele.

intervals between adjacent markers were longer than 40 cM) and also in consideration that we estimate that our map covers ca. 75% of the maize genome.

The only overlap between support intervals of the QTLs for DPS and the QTLs for L-ABA consistent in both locations occurred on chr. 5. In this case, the QTL for DPS peaked near *umc54*, while the QTL peak for L-ABA-3 was localized 16 cM away from *umc54*. The allele increasing L-ABA was contributed by B73, which also contributed the allele delaying flowering at the nearby QTL for maturity. In Rosemount, an overlap between QTLs for L-ABA-2 and DPS was evidenced on chr. 3, near *umc154*. The presence of QTLs for L-ABA and DPS was also evidenced on chr. 8. However, in this case, the peaks of the two QTLs were ca. 50 cM apart. These results provide little evidence supporting an association, either by pleiotropy and/or linkage, between QTLs for L-ABA and QTLs for DPS in maize under field conditions. Accordingly, the correlations between L-ABA and DPS were negligible in magnitude in all four samplings (r from 0.08 to 0.18).

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Apomixis and endosperm development

--Tyrnov, VS

The problem of apomixis is of great economic importance. At present efforts have been on experimental production of apomictic forms with traditional genetical-breeding methods as well as methods of molecular biology and genetic engineering. As a rule, insufficient attention is given to endosperm and often to the problem of double fertilization as a whole.

On the practical side, the ideal form of apomixis is an autonomous one, when both embryo and endosperm develop without fertilization, and pollen is not necessary at all. Autonomy not only contributes to fixation of heterosis and other unique characters, but it also decreases the dependence of seed productivity on influence of unfavourable temperature, drought, rain and other abiotic factors during the process of pollination. In addition, it decreases dependence on insect-pollinators. It can not be excluded, that, in perspective, maize genes can be transferred by methods of molecular biology to other cultures, including insect-pollinating ones.

Unfortunately, in maize reliable examples of absolutely autonomous formation of embryo and endosperm and, as a consequence, seeds are unknown. At the same time in maize, in all probability, pseudogamy, where the embryo is apomictic, but the endosperm can develop only after fertilization, is distributed widely (Tyrnov and Selivanov, pp. 124-134 in *Apomixis and its use in breeding*, Moscow, "Kolos", 1976. In Russian).

By using the method of genetical marking we found repeatedly diploid plants of the maternal type, grown from kernels, the endosperm of which had all marker genes of the paternal form. We examined more than 10 million genetically marked kernels and seedlings. The frequency of occurrence of maternal type diploids in different materials was in the range 0.01-0.001%.

Pseudogamy is also a characteristic of haploids (reduced apomicts) produced experimentally. We analyzed more than 20,000 maize haploids, arising in marker crosses. They all had hybrid endosperm, possessing characters (genes) of a parental form. Our investigations showed that it was a result of single

fertilization of the central cell (Enaleeva et al., Dokl. Biol. Sci. 353:225-226, 1997). Among a low number of non-marked kernels haploids were not discovered. In all probability, the origin of such kernels is from pollen contamination.

We also described lines of maize (Tyrnov, MNL, 71:73-74, 1997; Enaleeva and Tyrnov, MNL, 71:74-75, 1997) having haploid parthenogenesis with a frequency up to 100%. At initial developmental stages, according to embryological data, autonomous apomixis takes place, i.e. the development of embryo and endosperm proceed without pollination. However, endospermogenesis does not go to the end. Haploid plants develop only in that case, if endosperm is formed by fertilization. And so, haploid parthenogenetic embryos can develop normally without fertilization, but endosperm can not.

Pseudogamy is also characteristic of the apomictic maize relative *Tripsacum*, which is regarded by many scientists as a donor of apomixis genes.

If anyone takes a look at the problem of pseudogamy from evolutionary positions, he can conclude that the evolutionary process leads first to apomictization of embryo, but not endosperm. Since endospermal function is limited, specific and short-timed, anyone would think that the contrary phenomenon must be observed - in the first turn apomictization of endosperm, but not embryo. It is probable there is a profound sense in such "unlogical evolution". We suppose that one of the important functions of endosperm connects with keeping the stability of sexual reproduction.

The analysis of evolutionary preconditions, experimental data and logical constructions allows some conclusions (Tyrnov, pp. 112-114 in *Gametic and zygotic selection*, Kishinev, 1987. In Russian).

1. Embryo development without fertilization is a rather frequent phenomenon. It is, probably, a constant component of the plant reproduction system. This puts under a threat reproduction by sexual means, that, in absolute expression, is not always worth while or useful for onto- and phylogenesis. At the same time it is the precondition for successful artificial selection of forms with increased tendency to apomixis.

2. A high level of spontaneous apomictization has led to creation of a binary system (embryo - endosperm), securing conservation of stability for a sexual reproduction system. Apomictic endosperm, as a rule, has genome content that does not allow it to develop normally. Undeveloped endosperm leads to the ruin of the embryo and, as a consequence, to the elimination of forms with an apomictic tendency.

What is the genomic content of endosperm? In diploids, it has, as a rule, 3n ploidy. Even in polyploids (4n, 6n and etc.) the ploidy is divisible by 3. When the endosperm develops without fertilization, it must have "n" or "2n" ploidy (2n or 4n in tetraploids) in connection with fusion of polar nuclei.

The possibility of endosperm development with another ploidy than 3n was discovered in crosses 2n x 4n and 4n x 2n, producing endosperm with 4n and 5n ploidy. Our data and those of other investigators show, that tetraploid endosperm, as a rule, is not able to develop, and pentaploid endosperm has various degrees of completeness.

This situation can be extrapolated on apomictic forms. In embryo sacs, potentially able to undergo haploid parthenogenesis, fusion of polar nuclei gives 2n ploidy. In case of non-reduction each of the polar nuclei has 2n ploidy and their fusion gives 4n. Thus, in all cases, the nuclei are formed with ploidy leading to degeneration

of endosperm. Fertilization gives origin to development of endosperm with 3n and 5n ploidy. The last can be rather developed, and an apomictic embryo has a chance to survive. Maybe that is just what contributes to pseudogamy and makes the passage to autonomous apomixis difficult.

Can the mentioned limitation be overcome anyhow? Probably, yes. Autonomous apomicts occur, although rarely, in nature in different species. Almost all apomicts are polyploids, including triploids. It is possible that increased endosperm ploidy can contribute satisfactory development. It can not be excluded, that the work on experimental production of apomicts must be carried out on a triploid level, since endosperm will have optimal ploidy - 3n or 6n. We examine this hypothesis now. In addition, species are known, in which only one of two polar nuclei is fertilized, and their endosperm is, possibly, diploid. The regularities of endospermogenesis in such species merit special investigation.

We also draw attention to one circumstance. By unreduced pseudogamous apomixis the endosperm will have, most probably, 5n (in diploids). At the same time very puny kernels are formed. And so, instead of heterosis, significant reduction of seed productivity can take place. We observed this phenomenon in model experiments with tetraploids of apomictic maize line AT-1. Is any progress possible in this field? It can not be excluded. We pollinated the common tetraploids (non-apomictic) by pollen of the different lines and hybrids. Theoretically, expected ploidy of the endosperm must be 5n. We observed various degrees of endosperm development: from forms like glumes to half of normal. Single kernels look like the ordinary small kernels located usually at the top of the ear. It points to the possibility of increasing, by selection, the tendency to formation of full-formed endosperm with atypical ploidy.

The given data speak about necessity to examine the regularities, concerning the role of endosperm in the reproduction system, since it is difficult to reckon on success in production of apomictic forms, even if anyone will be able to isolate and to clone apomixis genes.

Nuclear-plasmic interaction and instability of the maize genome

--Zavalishina, AS, Tyrnov, VS

Regularities of variability, constancy and instability of the genome, being the basis of formation and speciation, have not been investigated to a full degree yet. The role of cytoplasm in these processes has been examined still less. As a rule, the methods of reciprocal or backcrosses are used for investigation of a nuclear-plasmic interaction. In these cases the cytoplasm of hybrid derivatives combines with the nucleus, in which the nuclear genomes of both parents are present completely or partially. Each of them can influence phenotypical manifestation of genes. The method of androgenesis *in vivo* allows exclusion in offspring of the nuclear genome of the maternal parent. The present paper reports the results of investigation of phenotypic manifestation of nucleus-cytoplasm interaction in alloplasmic androgenic maize lines on plant colour.

As androgen-inducing lines, WF9-T, WF9-S, and W23, inducing ability of which is conditioned by the gene *ig*, as well as the line AT-T, whose inducing ability has another nature, and the line HPL-1, were used. Line HPL-1 was produced from matroclinous haploid Kinelsky 103, and so a cytoplasm of the line HPL-1 was first marked as K-103 (Zavalishina and Tyrnov, MNL 69:120-121). Three inbred lines were used as nuclear donors:

brown marker (BM), brown marker-96 (BM-96) and brown marker Saratovsky (BMS). All three lines have nuclear genes *a B Pl R*, conditioning brown colour of plants. The plants of lines BM, BM-96, BMS are very uniform in color and in quantitative signs. The inheritance of these signs was determined as a result of many years of observation. Line BM-96 was examined for 5 years, BM and BMS for about 20 years.

First, we produced androgenic haploids with the nucleus of one line (nuclear donor) and the cytoplasm of another (androgen-inducing). Haploids, as a rule, are mini-copies of the nuclear donor plants and preserve all their signs, including typical brown colour. However, on WF9-cytoplasm sometimes BM androgenic haploids were formed with changed colour - light-brown and almost green. Haploids were partially female fertile and, being pollinated by the line-nuclear donor, gave from 1 to 15 and more normal kernels with diploid embryos. The progeny of one haploid is an alloplasmic androgenic line, differing from line-donor by cytoplasm. Such a line, if it is characterized by CMS, can be constantly preserved in pure condition when pollinated by the line-nuclear donor.

Androgenic diploids were produced spontaneously on cytoplasm of HPL-1: there were 2n-2n twins with BM nuclear genome and also monoembryonic androgenic diploids with nuclear genome BM-96. Phenotypically these diploid androgens conformed completely to the nuclear donor plants. After self-pollination they also gave pure lines.

As a result, alloplasmic lines with nucleus BM were produced on cytoplasm WF9-T, WF9-S, W23, AT-T; with nucleus BMS on cytoplasm W23, AT-T, HPL-1; with nucleus BM-96 on cytoplasm HPL-1.

Now, we apply this to analysis of plant colour inheritance.

All androgenic haploids and their progenies were crossed to nuclear line donors. By this, in progenies of androgenic haploids, segregation of plant colour took place. In addition to typical brown plants, light-brown, sunlight brown (tun) and green plants were discovered. Distribution of plants by colour was rather relative, since among light-brown plants there were as many intensely coloured ones, as less. Analogously, among sun light brown (tun) plants colour was more manifest in some, and only weak traces of brown colour on the husks and tassel glumes in others. There were not even traces of brown colour in green plants. Sometimes there were plants which can be related to any group only with difficulty: for example, a few plants had light-brown tassels, but green culms, husks, sheaths, and in one plant all organs were brown, but culm, hidden in sheaths, was green.

Change of plant colour is accompanied by change of cob colour. The cobs of light brown plants were fawn-coloured, and cobs of sun-light brown and green plants were white.

Segregation of plant colour in progeny of typical androgenic brown plants appeared by crossing them to brown plant nuclear donors. For example: androgenic haploid BMS with cytoplasm AT-T had brown colour. By crossing it to nuclear donor 6 kernels were set. Four brown and 2 light brown plants were grown from them. In the progeny of one brown plant, when it was crossed to brown nuclear donor, 13 of 60 plants were brown, 21 were light brown, 12 were sun-light brown (tun) and 14 were green. By crossing light brown androgenic plants to brown nuclear donor, in progeny there were light brown, sun-light brown and green plants. Brown plants were not discovered at all.

For example: light brown x brown gave 4 light brown, 22 sun-light brown, and 60 green. From sun-light brown by the nuclear

donor, only sun-light brown and green plants were formed, and sometimes only green ones.

For example: sun-light brown x brown gave 1 sun-light brown and 26 green.

In all these crosses quantitative correlation of the plants differing by colour was different. And progeny of green androgenic plants, when they were pollinated by brown nuclear donor, during a number of generations only green plants appeared, which sometimes became dark green.

Analogous segregation was observed in progeny of alloplasmic lines with fertile cytoplasm by self-pollinating. Self-pollination of androgenic green plants led to only green plants.

In addition, progenies from reciprocal crosses of androgenic green plants to brown plants of nuclear donor were analyzed. In F1 only green plants appeared. The same was observed in F2 of these crosses. When androgenic diploids BMS on cytoplasm HPL-1 were self-pollinated, in progenies (in both members of twins) deviation in manifestation of plant colour was not discovered during 15 generations: all plants were brown. In self-pollinated progeny of BM-96 on the same HPL-1 cytoplasm half of the plants had a typical brown colour, and another half were green. Green plant colour was preserved by self-pollination in following generations. The progenies of the brown plants were not investigated.

It has to be noted that colour changes in progeny of androgenic plants happened in the same direction (from brown to green), but manifested in a different manner. Sometimes they manifested slowly during a number of generations, which looks like the result of accumulation of gene-modifiers. In other cases changes happened unevenly, and green colour manifested already in the next generation, which is analogous to mutation. Earlier we tried by the backcross method to transfer the inbred line BM on cytoplasm of lines with S-, T- and C- types of CMS. After two-three backcrosses uniform progeny were produced, consisting of brown plants only. However, following further backcrosses plants with light brown and green colour appeared. In spite of our attempts to select as maternal parents the most brown plants, only green plants appeared in progeny.

All the foregoing speaks about non-casual character of brown colour changes in the plants of alloplasmic lines. It is known that in maize a number of series of multiple alleles take part in determination of plant colour (Emerson, Cornell. Univ. Agr. Exp. Sta. 39:1-156, 1921; Coe and Neuffer, In Sprague (ed): Corn and Corn Improvement: 111-223, 1977; Coe, The Maize Handbook: 279-281, 199).

We made an attempt to control the presence of recessive alleles of genes *B* and *Pl* in plants with changed colour by the method of analysing crosses. We used a line having the genes *A b pl R-nj Cudu*. BM and BMS plants (nuclear donors) were crossed to this line. In F1 all plants had intense dark-purple colour. That colour corresponded to genotype *A B Pl R*.

Androgenic plants with brown, light brown, sun-light brown and green colour were also pollinated by the line with genes *A b pl R-nj Cudu*. In F1 of these combinations we observed, correspondingly, purple, light-purple, sun-purple, sun-light purple plants.

In addition to the described plants in F1 plants with other colours appeared: a) by pollinating androgenic BMS plants on cytoplasm AT-T purple plants appeared, the leaves of which at the top of husks were green; b) by pollinating brown plants with green stem purple plants appeared with free designs on purple

colour of husks, sheaths, tassels, and culm inside sheaths was green; c) by pollinating green plants the most various plant colour progenies arose: sun-dilute purple, sun-red purple, sun dilute purple with dark anthocyanic spots on culm or with purple stripes along culm, as well as lilac-pink and, at last, plants with very weak colour, almost green; d) by pollinating androgenic BMS on cytoplasm HPL-1 purple plants arose, the culms of which inside of sheaths had granite colour.

From all the foregoing it follows that transfer of dominant genes *B* and *Pl* into another cytoplasm provokes a change of plant colour, that phenotypically looks like the result of the presence of a series of multiple alleles and the recessive gene *pl*. The character of segregation, differing from the Mendelian one, is analogous to that described earlier in maize by other authors (Coe, Genetics 53:1035-63, 1966; Brink, Ann. Rev. Genet. 7:129-152, 1973) and was attributed to paramutations, the cause of the origin of which is not known yet.

Since in our experiment we used pure material, and the method of androgenesis in vivo allowed transfer of the same genome into the different cytoplasm, the single origin of paramutagenicity could be cytoplasm. We can suggest a scheme for the foregoing process. Under the influence of new cytoplasm the dominant gene becomes paramutable, transferring to the condition of one of its recessive alleles and acquiring by that the character of paramutagenicity. Under its action allele *B*, being brought by the nuclear donor, acquires the same characters.

Since the effect of paramutagenicity was observed, practically, in all combinations, excluding BMS on cytoplasm HPL-1, we can speak about the universality of this manifestation of nucleus-cytoplasm interaction.

Does a gene return to the previous functional condition by transfer to the initial cytoplasm?

To check this, we crossed plants of line-nuclear donor to androgenic green plants. All F1 plants had green colour. Then we again pollinated plants of the nuclear donor by F1 pollen. In the progeny of this cross all plants were green. After the second backcross among 50 plants two light brown plants with brown tassels were discovered.

This speaks of the possibility of restitution of functional activity of nuclear genes. By that the process of restitution proceeds gradually like its change under the influence of new cytoplasm. Thus, changes of genes as a result of paramutations are rather stable, and genes preserve phenotypical effect even being returned into their own cytoplasm. And only with the course of time do they return to their initial condition. In our report we considered only changes of colour manifestation. It can not be excluded that other genes, determining the quantitative traits, are under the same or analogous dependence on cytoplasmic factors. Coming from this suggestion, any cross can give new combinations of nuclear genome and cytoplasm, and their interaction can be manifested in the following generation in the form of spontaneous mutations, gene-modifiers, paramutations, mobile genetical elements, etc.

It can be supposed, that it is a cause of significant variability of characters and, in the end, a cause of line variation and aging. In all probability, this has to be taken into account in production of new sorts, since formation of some characters can not proceed according to simple schemes for ordinary nuclear or cytoplasmic inheritance.

Maize immature embryo culture

--Todorova, L, Kruleva, M, Krapchev, B, Nedev, T

For development of maize tissue cultures it is important to find a flexible genotype among a pool with agronomic value. In our investigation the influence of the following factors: genotype, embryo size and plant hormone (Dicamba) was studied. Immature embryos for initiation were taken from field donors (selfed ears) during the summer of 1997 and were planted according to Bohorova (Maydica 40:275-281). The experiments included 8 representatives (see Table 1). The studied embryos were 0.5 - 3.0 mm in size. For each genotype at least 3 plants, 50 embryos per ear, were examined. These values were averaged and Student's criterion was used for statistical analysis of the results.

Genotype A619 possesses good plant regeneration capability (Table 1). The observed differences in the regeneration capacity

Table 1. The influence of genotype on embryogenic formation and plant regeneration.

Genotype	Embryogenic calli %	Plant regeneration %
A619	28.97***	20.00*
A344	16.92	-
A95/84	22.77	-
M320	11.08	-
Bup 116	4.83	-
W401	10.98	-
163 x A654	13.79	21.05
163 x W19	1.42	-

***p < 0.001

*p < 0.05

suggested presence of a gene or block of genes with possibility to control the expression of embryogenic callus initiation.

The optimal embryo size for callus formation was determined to be 1.0 - 2.0 mm. It was found in our experiments that Dicamba was one of the best growth regulators for initiation and growth of regenerable maize calli. Similar conclusions were made by other authors (Bohorova, Hoisington, D, Theor. Appl. Genet. 92 :163-169, 1996).

Finally, an appropriate set of conditions for production of regenerants from A619, important for breeding programs, is proposed.

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Map position of the centromere on chromosomes 1 and 9

--Lin, B-Y, Chang, S-J

B-A translocations with the most proximal breakpoints on both arms of chromosomes 1 and 9 were used to map the centromeric position on RFLP maps. The mapping strategy is based on the fact that the centromere is located between the breakpoints of the two most proximal translocations: one in the short arm and the other in the long arm of the same chromosome. The RFLP marker closest to but distal to the translocation breakpoint on both arms defines the map region of the centromere.

The marker position in relation to translocation breakpoints is determined by the terminal deficiencies generated by B-A translocations. Like the B chromosome, one of the two B-A translocation chromosomes, termed B-A, undergoes nondisjunction

at the second pollen mitosis. This process generates two sperm: one with two B-As (hyperploid) and the other without any B-A (hypoploid). Upon fertilization with an egg carrying the normal chromosome complement, the latter sperm results in a hypoploid embryo deficient for the paternal copy of the chromosome arm distal to the translocation breakpoint. In other words, the hypoploid embryo associated with the terminally deficient chromosome arm is employed to generate DNA for RFLP analysis. For simplification of the RFLP analysis, both parents of the hypoploid progeny are in two different inbred backgrounds: the maternal parent is B73 and the paternal parent is L289. The RFLP analysis is done by Southern hybridization of the hypoploid DNA with each marker whose position in reference to the breakpoint is determined by the presence (proximal) or absence (distal) of the paternal signal.

Table 1 gives the mapping result of 10 RFLP markers on chromosome 1 by hypoploids of TB-1Sb and TB-1La. Three markers (*asg45*, *csu3*, and *umc167*) exhibit no paternal signal on the hypoploid of TB-1Sb, and six others (*umc177a*, *bnl5.59*, *umc119*, *umc58*, *asg62*, and *bnl6.32*) give no paternal signal on the hypoploid of TB-1La. The paternal signal of one marker (*umc67*) is absent on the hypoploid of TB-1Sb and TB-1La, and none of the 10 markers have the paternal signal on the hypoploids of both translocations, indicating that they are not located in the region between the breakpoints of the two translocations, including the centromere. Disregarding *umc67*, current data map the centromere to the *umc167-umc177a* region, an interval of about 14 map units according to the map of Davis et al. (MNL 70:123-132, 1996). On the other hand, the fact that Matz et al. (MNL 69:247-256, 1995) mapped *umc67* to the short arm of chromosome 1, places the centromere in the *umc67-umc177a* region that spans less than one map unit on the map of Davis et al. (1996). On the other hand, since the 1-B chromosome of TB-1Sb is associated with a rearrangement, and *umc67*, located near the end of the rearranged region, is deleted by multiple breakages during the formation of the translocation (Lin and Chang, MNL, this volume). In other words, *umc67* is located on the long arm of chromosome 1, most proximal to the breakpoint of TB-1Sb. As a consequence, the centromere is located between *umc167* and *umc67*, a region of 13 map units.

Table 1. Mapping 10 RFLP markers on chromosome 1 by hypoploids of TB-1Sb and TB-1La. (+, presence and -, absence of the paternal signal.)

RFLP markers	Hypoploids	
	TB-1Sb	TB-1La
<i>asg45</i>	-	+
<i>csu3</i>	-	+
<i>umc167</i>	-	+
<i>umc67</i>	-	-
<i>umc177a</i>	(+)*	-
<i>bnl5.59</i>	+	-
<i>umc119</i>	+	-
<i>umc58</i>	+	-
<i>asg62</i>	+	-
<i>bnl6.32</i>	+	-

*presence of the non-L289 paternal signal (see following article).

Table 2 shows the results of mapping 12 RFLP markers by hypoploids of TB-9Sd and TB-9Lc. The paternal signal of five markers (*umc109*, *umc113a*, *bnl3.06*, *umc247*, and *umc81*) is absent on the hypoploid DNA of TB-9Sd, and that of six others (*umc20*, *bnl5.04*, *umc114*, *bnl8.17*, *umc95*, and *csu54b*) is not present on the hypoploid of TB-9Lc. The marker (*bnl5.10*) has no

Table 2. RFLP analysis of 12 markers on chromosome 9 by hypoploids of TB-9Sd and TB-9Lc. (+, presence and -, absence of the paternal signal.)

RFLP markers	Hypoploids	
	TB-9Sd	TB-9Lc
<i>umc109</i>	-	+
<i>umc113a</i>	-	+
<i>bnl3.06</i>	-	(+)*
<i>umc247</i>	-	(+)*
<i>umc81</i>	-	(+)*
<i>bnl5.10</i>	-	-
<i>umc20</i>	+	-
<i>bnl5.04</i>	(+)*	-
<i>umc114</i>	(+)*	-
<i>bnl8.17</i>	(+)*	-
<i>umc95</i>	(+)*	-
<i>csu54b</i>	+	-

*presence of the non-L289 paternal signal.

paternal signal on the hypoploid of TB-9Sd and TB-9Lc, and none of the markers have the paternal signal on the hypoploids of both translocations, implying all 12 markers are distal to the breakpoints of the two translocations. Without consideration of *bnl5.10*, current data place the centromere in the *umc81-umc20* region, the length of which is about 2.5 map units in the Davis et al. map (1996) or 4 map units in the Burr et al. map (1995). Since *bnl5.10* is located at the proximal end of a rearrangement on the 9-B chromosome of TB-9Lc, it was deleted by multiple breakages which occurred during the formation of the translocation (Lin and Chang, MNL, this volume). Accordingly, *bnl5.10* is proximal to *umc81*, and the centromere is located in the *bnl5.10-umc20* interval whose length can not be estimated because of the uncertain map position of *bnl5.10*. Burr et al. (1995) placed *bnl5.10* on the short arm of chromosome 9, and Davis et al. (1996) mapped it to a position distal to *umc20*, which is on the long arm according to the data of this study.

Chromosome rearrangements associated with B-A translocations

--Lin, B-Y, Chang, S-J

During the RFLP analysis of hypoploids of four B-A translocations, unexpected results were observed. The hypoploids--identified by their short stature in comparison to the normal sibs, 50% pollen sterility, and chromosome counting--were produced after crossing B73 with pollen of the B-A translocation-carrying L289. The hypoploid gives or lacks the paternal L289 signal following hybridization with RFLP markers, depending on the markers being proximal or distal, respectively, to the breakpoint of the relevant B-A translocations. Most markers show the expected signal pattern on hypoploids, but some do not.

The unexpected results are associated with the hypoploids of three translocations (TB-1Sb, TB-9Sd, and TB-9Lc), exhibiting two different paternal signals: L289 or non-L289. For example, the hypoploid of TB-1Sd, following hybridization with *umc177a*, gave two signals: B73 and non-L289 (Tables 1 and 2; Lin and Chang, MNL, this volume). The L289 signal is not present. The non-L289 signal is paternal in origin, since it is absent in B73 and in the hybrid between B73 and L289 included in the same analysis. *umc177a* is the only marker showing the paternal non-L289 signal; other markers flanking *umc177a* exhibit the expected L289 signal. The same situation was observed in the hypoploids of TB-9Sd and TB-9Lc. In the case of the hypoploids of TB-9Sd, four markers (*bnl5.04*, *umc114*, *bnl8.17*, and *umc95*) on 9L, show the paternal non-L289 signal. For the hypoploid of TB-9Lc, three markers (*bnl3.06*, *umc247*, and *umc81*) behave similarly. The size of the

paternal non-L289 signals is variable: three are larger than the L289 signal, and five others are smaller.

The simplest explanation for the presence of the paternal non-L289 signal is the existence of a chromosome rearrangement--most likely an inversion--on the A-B chromosome of the three B-A translocations. This explanation is consistent with the fact that markers giving the paternal non-L289 signals are linked together: Four markers of this nature on 9L, spanning over a region of about 19 map units, are closely linked; and three others on 9S, covering about 15 map units, are likewise linked. The affected regions are not interrupted by markers that show the L289 signal.

Also consistent with the explanation is the existence of markers which lack the paternal signal (L289 or non-L289) on hypoploids of the two B-A translocations involving the two arms of the same chromosome. For example, the affected region on the 9-B chromosome is closely associated with *bnl5.10*, which is deficient for the paternal signal of the hypoploids of TB-9Sd and TB-9Lc (see Table 1; Lin and Chang, MNL, this volume). Since the former carries the deficiency of 9S and the latter the deficiency of 9L, the paternal signal is expected to be absent on only one of the two hypoploids. The fact that it is also absent on the second hypoploid suggests the occurrence of a complex rearrangement that leads to the formation of a chromosome deletion covering the *bnl5.10* locus. In addition, since the locus is linked to the three markers on 9S that give the paternal non-L289 signal--no marker giving the L289 signal is located between them, these four markers must be involved in the same rearrangement: *bnl5.10* is deleted by an additional breakage before ligation. Accordingly, *bnl5.10* is closely linked and proximal to *umc81*, the most proximal marker of the rearrangement. By the same rationale, *umc67* and *umc177a* are involved in the same rearrangement, and the former, being proximal to the latter, has been deleted from the rearranged region by a similar event.

Using B-A translocations to isolate AFLPs on the proximal half of chromosome 10

--Cheng, Y-M, Lin, B-Y

Use of two B-A translocations associated with the same chromosome arm but with different break positions, provides a method for isolation of molecular markers in a specific A-chromosome region. A B-A translocation, when crossed as staminate plant with a pistillate plant carrying the normal chromosome complement, produces a hypoploid whose A-B chromosome is deficient for the portion of the A chromosome distal to the breakpoint. The extent of the deficiency associated with an A-B chromosome is dependent on the breakpoint of a translocation. The A-B chromosome from a proximal translocation has a long deficiency, and that from a distal translocation has a short one. The two deficiencies cover a region in common--the region distal to the breakpoint of the distal translocation. They are different in the region between the breakpoints of the two translocations; the region is absent in the former, but it is present in the latter. Therefore, a molecular marker whose paternal signal appears on the first hypoploid but not on the second, is located in the region delimited by the two breakpoints. TB-10L19 and TB-10L32 were used to define the region on the long arm of chromosome 10 (10L) for isolation of AFLPs. Hypoploids from the two translocations were produced by crossing the two translocation-carrying inbred W22s as male onto inbred B73. TB-10L19 breaks very proximally to the 10th centromere (Lin,

MNL 48:182-184), and its 10-B chromosome is deficient for almost the entire 10L. TB-10L32 has a breakpoint between *g1* and *r* on 10L (Lin, MNL 48:182-184), and its 10-B chromosome is deficient for about the proximal half of 10L. Thus, the two 10-B chromosomes are different in the deletion region: the proximal half of 10L is deleted from the first 10-B chromosome, but it is not deleted from the second. Accordingly, an AFLP signal appearing on the hypoploid of TB-10L32, but not on the hypoploid of TB-10L19, is located on the proximal half of 10L. 47 AFLPs of this nature were identified in this study. These AFLPs are located on 10L but not on the B chromosome for the following reasons: TB-10L19 breaks in the proximal one fifth of the distal heterochromatic region on the B chromosome (Lin, Genetics 92:931-945), and TB-10L32 breaks in the proximal one fourth of the same region (Lin, unpublished); therefore, the 10-B(19) carries more distal B heterochromatic region than the 10-B(32) does. If an AFLP were located on the B chromosome, it would be present on the hypoploid of TB-10L19, but not on that of TB-10L32. None of the 47 AFLPs behave in this pattern; thus, these 47 AFLPs must be 10L-specific.

Two RFLPs included in the *r-X1* deletion

--Peng, S-F, Lin, B-Y

Physical mapping of RFLPs on the long arm of chromosome 10 (10L) by terminal deficiencies (TDs) induced by the *r-X1* deletion reveals a difference between the physical and genetic maps (Lin et al., Mol. Gen. Genet. 256:509-516, 1997). In the former, *bnl10.13* and *bnl17.02* are distal to *bnl7.49*, but, in the latter, they are proximal. This difference can be due to an incorrect placement of the first two RFLPs on either the genetic map or the physical one.

These two possibilities can be distinguished by studying the *r-X1* deletion according to the following rationale: the physical position of *bnl10.13* and *bnl17.02* has been determined by two TDs on 10L generated by the *r-X1* deletion. The two RFLPs fail to give the maternal signal on the two TDs, but *bnl7.49* does, indicating the former being distal to the latter. Alternatively, since the two TDs carry a terminal deficiency as well as the *r-X1* deletion, *bnl10.13* and *bnl17.02* may be located in the latter. In other words, their maternal signal being absent on the two TDs is the result of their being deleted from the *r-X1* deletion, not from the terminal deficiency. This supposition can be substantiated by studying a 10L that carries the *r-X1* deletion but not a terminal deficiency, that is, the *r-X1*-carrying chromosome 10. To do this, the *r-X1/R-r* (W22) was pollinated by an *r-g* tester, and the resulting colorless kernels, carrying the *r-X1* deletion, are compared with the colored ones, carrying a normal chromosome 10. The maternal signal of *bnl10.13* and *bnl17.02* was not observed in the former, but it was in the latter, indicating that the two RFLPs are indeed located in the *r-X1* deletion. As a consequence, the physical map of 10L is consistent with its genetic map.

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Variation for ABA concentration in tassels from H99xFR16 and H99xPa91 F4 families

--Wassom, JJ, Mei, C, Rocheford, TR, Widholm, JM

F2-derived F4 families were developed as a part of a continuing anther culture project in this lab. There is a great deal

of variation for responsiveness of maize anthers as explants for tissue culture and we have been investigating genetic and physiological factors significant to this variation. Much of our work has utilized various genotypes originating from crosses of H99, Pa91, and FR16. Previous studies in our lab have indicated genetic variation at the gametophyte and sporophyte level associated with F1 and F2 plants from crosses of these inbreds (Wan, Rocheford, and Widholm, TAG 85:360-365, 1992; Beaumont, Rocheford, and Widholm, Genome 38:968-975, 1995). Therefore, F2-derived F4 families were developed to continue the anther culture studies.

In 1995 the F4 families were grown in a nursery at Urbana, IL. There was one row for each family and approximately 20 plants in each row. The nursery was irrigated when needed. Tassels were harvested for anther culture when microspores were at the late uninucleate to early binucleate stage. Tassels were then cold-treated for 14 d at 8 C in preparation for anther culture. At this time unused parts of the tassels were frozen and stored at -70 C for several weeks before lyophilizing the tassels and assaying florets of the tassels for ABA concentration. Leaf ABA was also measured. Leaf samples from three plants of each family were

Table 1. Tassel ABA concentrations of F4 families.

LSD grouping*	ABA, ng/g dry wt	N	F4 family	Pedigree
A	1201.5	2	PF34a	Pa91xFR16
B A	962.0	2	HP15b	H99xPa91
B C	729.6	5	PF44a	Pa91xFR16
B C D	672.0	6	PF21a	Pa91xFR16
B C D	671.7	3	PF16a	Pa91xFR16
BEC D	628.0	2	HP67	H99xPa91
FEC D	582.5	4	HP8a	H99xPa91
GFEC D	563.3	4	PF20a	Pa91xFR16
GFEC DH	546.0	1	HP14	H99xPa91
GFEC IDH	518.0	1	PF36	Pa91xFR16
GFEC IDH	517.0	4	PF24a	Pa91xFR16
GFEC IDH	508.0	1	HP24a	H99xPa91
GFEJCIDH	475.0	1	PF12a	Pa91xFR16
GFEJCIDH	432.2	6	PF31a	Pa91xFR16
GFEJCIDH	431.0	2	PF43a	Pa91xFR16
GFEJCIDH	405.0	3	PF27	Pa91xFR16
GFEJCIDH	402.0	2	HP36	H99xPa91
GFEJCIDH	384.8	4	PF13a	Pa91xFR16
GFEJ IDH	378.0	5	HP13	H99xPa91
GFEJ IDH	373.0	1	HP44a	H99xPa91
GFEJ IDH	363.0	2	HP76-19	H99xPa91
GFEJ IDH	360.8	4	PF1a	Pa91xFR16
GFEJ IDH	343.8	4	PF26	Pa91xFR16
GFEJ IDH	336.0	1	HP42a	H99xPa91
GFEJ IDH	334.0	3	PF15	Pa91xFR16
GFEJ IDH	334.0	6	HP2	H99xPa91
GFEJ IDH	327.5	2	HP31a	H99xPa91
GFEJ IDH	326.3	4	PF45a	Pa91xFR16
GFEJ IH	320.7	9	PF10	Pa91xFR16
GFEJ IH	317.0	3	HP77-20	H99xPa91
GFEJ IH	313.0	4	PF32a	Pa91xFR16
GFEJ IH	312.5	2	PF6a	Pa91xFR16
GFEJ IH	310.0	1	HP32b	H99xPa91
GFEJ IH	300.0	2	HP6a	H99xPa91
GFEJ IH	290.5	2	PF37	Pa91xFR16
GF J IH	277.0	1	HP43a	H99xPa91
GF J IH	268.5	2	HP35	H99xPa91
GF J IH	268.0	5	PF56	Pa91xFR16
GF J IH	262.0	1	HP7	H99xPa91
GF J IH	253.5	2	PF40a	Pa91xFR16
GF J IH	245.0	1	PF35	Pa91xFR16
G J IH	233.0	2	HP32a	H99xPa91
J IH	206.0	1	HP3a	H99xPa91
J I	198.0	4	HP75-18	H99xPa91
J I	196.0	2	HP33a	H99xPa91
J	150.0	1	PF4a	Pa91xFR16
J	150.0	1	PF8a	Pa91xFR16
J	140.0	1	HP28b	H99xPa91

*Means with the same letter are not significantly different according to LSD (P=0.05).

bulked. There was only one replicate of each family for leaf ABA. The ABA was measured by radioimmunoassay by the method of Quarrie et al. (Planta 173:330-339, 1988).

There was significant variation for ABA concentration in tassels of the tested F4 families ($P < 0.01$). Mean concentrations of ABA ranged from 140 to 1201 ng/g dry weight, with continuous variation between the extremes (Table 1). Leaf ABA concentration ranged from 16 to 108 ng/g fresh weight. Leaf samples were not replicated, so it is not known whether there was significant variation among families. When about half the tassels had been assayed for ABA, a correlation analysis was performed which showed essentially no correlation of ABA concentration in whole florets or leaves with production of embryoids by cultured anthers. Tassel ABA was not correlated with leaf ABA. At this point the ABA assays were discontinued. Nevertheless, it is apparent that these families differ significantly for ABA. Parental inbreds were not assayed.

We wish to acknowledge the performance of ABA assays by David Driver and the use of the lab facilities of Dr. Marty Sachs (Univ. of Ill.). The ABA antibody was provided by S. A. Quarrie.

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Three-point linkage data for *su1* *bm3* *gl7* on 4S

--Stinard, P

The results of a three-point linkage test for *su1*, *bm3*, and *gl7* on chromosome 4 are presented in Table 1. The linkage test was set up as a modified backcross as indicated in Table 1. Kernels from the backcross ears were planted in the field and the resulting plants were scored for *bm3*. The plants were self-

Table 1. Three-point linkage data for *su1* - *bm3* - *gl7*. Testcross: *Su1* *bm3* *Gl7* X (*su1* *bm3* *gl7* / *Su1* *Bm3* *Gl7*).

Reg.	Phenotype	No.	Totals
0	<i>su1</i> <i>bm3</i> <i>gl7</i> + + +	198 218	416
1	<i>su1</i> + + + <i>bm3</i> <i>gl7</i>	12 6	18
2	<i>su1</i> <i>bm3</i> + + + <i>gl7</i>	5 5	10
1+2	<i>su1</i> + <i>gl7</i> + <i>bm3</i> +	1 2	3

% recombination *su1*-*bm3* = 4.7 +/- 1.0

% recombination *bm3*-*gl7* = 2.9 +/- 0.8

% recombination *su1*-*gl7* = 7.6 +/- 1.3

pollinated, and the self-pollinated ears were scored for the presence of *su1*. Kernel samples from each self-pollinated ear were planted in the sand bench, and seedlings grown from these kernels were scored for *gl7* and *v17*. (Although *v17* was included in this linkage test, the data for *v17* linkage were not tabulated since no crossovers were observed between *gl7* and *v17*. We conclude that *v17* is tightly linked to *gl7*, and the population size used in this study was not large enough to detect crossovers.) The following linkage relationship was established: *su1* - 4.7 - *bm3* - 2.9 - *gl7*. Last year, we reported that *gl7* and *v17* are uncovered by TB-4Sa (MNL 71:83). Beckett reported in 1975

that *bm3* is not uncovered by TB-4Sa (MNL 49:132). Coe recently reported in the Maize Database (ID#: 108126) that Beckett's data is ambiguous and that no determination can be made from the data whether *bm3* is uncovered by TB-4Sa. Since our data place *bm3* between *su1* and *gl7*, we predict that *bm3* will be uncovered by TB-4Sa. This test is in progress.

Results of TB tests of symbolized unplaced mutants

--Stinard, P, Jackson, JD

Most of the symbolized mutants (i. e. mutants which have been assigned a permanent gene symbol) in the Maize Genetics Cooperation • Stock Center collection have known chromosomal locations. However, a small set of symbolized mutants were either never placed to chromosome, or had conflicting mapping data. This past spring, we selected a subset of the symbolized unplaced mutants, and placed them in our summer crossing nursery to be crossed by a comprehensive set of B-A translocations (TB's). The mutants for which we obtained positive results are

Table 1. Results of TB tests of symbolized unplaced mutants.

Mutant	Arm-locating TB Cross	Number of Positive Tests/Total Number of Crosses with this TB	Mutants on Same Chromosome Arm with Similar Phenotype	Note
<i>l3</i>	TB-6Lc	1/4	<i>l10</i> , <i>l12</i> , <i>l15</i>	
<i>l4</i>	TB-7Sc	1/2	<i>w17</i>	1
<i>ora2</i>	TB-1Sb-2L(4464)	2/4		
<i>pb4</i>	TB-5La	2/2	<i>gr11</i> , <i>ppg1</i>	2
<i>v13</i>	TB-5Sc	2/2		
<i>vp10</i>	TB-10L(19)	2/3	<i>vp13</i>	
<i>vp12</i>	TB-5La	2/4	<i>w2</i>	3

1. All Coop stocks of *l4* have an albino seedling phenotype, not a luteous phenotype.

2. Previously reported on 6L. This author found some stocks previously thought to be *pb4* were in fact *pb1*, an allele of *y1*, located on 6L. Perhaps this is where the mapping confusion arose.

3. Previously reported on 6L (Araujo et al., Brazilian J. Genet. 20:71-74, 1997).

summarized in Table 1. Additional crosses with linkage markers will be made to confirm chromosome arm placement, and allelism tests will be conducted with mutants with similar phenotype located on the same chromosome arm.

Linkage tests of Non-waxy (*Waxy1*) reciprocal translocations involving chromosome 9 at the MGCSC

--Jackson, JD, Stinard, P

Approximately 1 acre each year is devoted to the propagation of the large collection of A-A translocation stocks. In this collection is a series of *Waxy1*-linked translocations that are used for mapping unplaced mutants. Each translocation is maintained in separate M14 and W23 inbred backgrounds which are crossed together to produce vigorous F1's to fill seed requests. Over the years, pedigree and classification problems arose during the propagation of these stocks. We have been able to sort through the problem ones, and we can now supply good sources proven by linkage tests to include the correct translocated chromosomes. Additional translocation stocks will be tested as time allows. Below is a summary of those we have completed testing.

Table 1. Wx1 T3-9(8562) (3L.65; 9L.22).

- A) The M14 sources showed linkage of wx1 with a1.
 B) The W23 sources showed linkage of wx1 with a1.

A) 2 point linkage data for a1-Wx1 T3-9(8562)
 Testcross: [A1 Wx1 T3-9(8562) x a1 wx1 N] x a1 wx1 N

source:93W-1423-1 ^M14

Region	Phenotype	No.	Totals
0	+ Wx	675	
	a wx	656	1331
1	a Wx	174	
	+ wx	174	348

% recombination a1-Wx1=2.1±.3

B) 2 point linkage data for a1-Wx1 T3-9(8562)
 Testcross: [A1 Wx1 T3-9(8562) x a1 wx1 N] x a1 wx1 N

source:93W-1425-2 ^W23

Region	Phenotype	No.	Totals
0	+ Wx	748	
	a wx	607	1355
1	a Wx	193	
	+ wx	201	394

% recombination a1-Wx1=2.3±.3

Table 2. Wx1 T4-9g (4S.27; 9L.27).

- A) Some F1's showed linkage of wx1 with su1.
 B) Some of the M14 sources showed linkage of wx1 with su1.
 C) Some of the W23 sources showed linkage of wx1 with su1.

A) 2 point linkage data for su1-Wx1 T4-9g
 Testcross: [Su1 Wx1 T4-9g x su1 wx1 N] x su1 wx1 N

source:87-888 x 990-8 F1 of ^M14 x ^W23

Region	Phenotype	No.	Totals
0	+ Wx	1725	
	su wx	1074	2799
1	su Wx	43	
	+ wx	92	135

% recombination su1-Wx1=4.6±.3

source:87-889 x 990-8 F1 of ^M14 x ^W23

Region	Phenotype	No.	Totals
0	+ Wx	2339	
	su wx	1807	4146
1	su Wx	100	
	+ wx	74	174

% recombination su1-Wx1=4.0±.3

B) 2 point linkage data for su1-Wx1 T4-9g
 Testcross: [Su1 Wx1 T4-9g x su1 wx1 N] x su1 wx1 N

source:82-215-5^M14

Region	Phenotype	No.	Totals
0	+ Wx	1544	
	su wx	1212	2756
1	su Wx	30	
	+ wx	48	78

% recombination su1-Wx1=2.8±.3

source:84-15-2^M14

Region	Phenotype	No.	Totals
0	+ Wx	290	
	su wx	234	524
1	su Wx	6	
	+ wx	5	11

% recombination su1-Wx1=2.1±.6

source:84-15-4^M14

Region	Phenotype	No.	Totals
0	+ Wx	1123	
	su wx	896	2019
1	su Wx	35	
	+ wx	56	91

% recombination su1-Wx1=4.3±.4

source:79-105-5^M14

Region	Phenotype	No.	Totals
0	+ Wx	870	
	su wx	718	1588
1	su Wx	28	
	+ wx	28	56

% recombination su1-Wx1=3.4±.4

C) 2 point linkage data for su1-Wx1 T4-9g
 Testcross: [Su1 Wx1 T4-9g x su1 wx1 N] x su1 wx1 N

source:93W-1443-3^W23

Region	Phenotype	No.	Totals
0	+ Wx	275	
	su wx	217	492
1	su Wx	10	
	+ wx	15	25

% recombination su1-Wx1=4.8±.9

Table 3. Wx1 T5-9c (5S.07; 9L.10).

- A) The M14 source showed linkage of wx1 with a2.
 B) The W23 source showed no linkage of wx1 with a2.

A) 2 point linkage data for a2-Wx1 T5-9c
 Testcross: [A2 Wx1 T5-9c x a2 wx1 N] x a2 wx1 N

source:82-142-3 ^M14

Region	Phenotype	No.	Totals
0	+ Wx	1545	
	a wx	1359	2904
1	a Wx	65	
	+ wx	66	131

% recombination a2-Wx1=4.3±.4

Table 4. Wx1 T5-9(4817) (5L.06; 9S.07).

- A) The M14 source showed linkage of wx1 with a2.
 B) Some W23 sources showed linkage of wx1 with a2.

A) 2 point linkage data for a2-Wx1 T5-9(4817)
 Testcross: [A2 Wx1 T5-9(4817) x a2 wx1 N] x a2 wx1 N

source:93W-1445-3 ^M14

Region	Phenotype	No.	Totals
0	+ Wx	343	
	a wx	406	749
1	a Wx	15	
	+ wx	19	34

% recombination a2-Wx1=4.3±.7

source:93W-1445-3 ^M14

Region	Phenotype	No.	Totals
0	+ Wx	912	
	a wx	886	1798
1	a Wx	65	
	+ wx	47	112

% recombination a2-Wx1=5.9±.5

B) 2 point linkage data for a2-Wx1 T5-9(4817)
 Testcross: [A2 Wx1 T5-9(4817) x a2 wx1 N] x a2 wx1 N

source:84-07-8 ^W23

Region	Phenotype	No.	Totals
0	+ Wx	1796	
	a wx	1696	3492
1	a Wx	96	
	+ wx	97	193

% recombination a2-Wx1=5.2±.4

source:85-019-1 ^W23

Region	Phenotype	No.	Totals
0	+ Wx	487	
	a wx	489	976
1	a Wx	11	
	+ wx	16	27

% recombination a2-Wx1=2.7±.5

Table 5. *Wx1* T9-10(8630) (9S.28; 10L.37).

- A) All of the M14 sources showed no linkage of *wx1* with either *r1* or *g1*.
 B) Some of the W23 sources showed linkage of *wx1* with *r1* & *g1*:

B) 2 point linkage data for *r1-Wx1* T9-10(8630)

Testcross: [*r1 Wx1* T9-10(8630) x *R1 wx1 N*] x *r1 wx1 N*

source:93W-1459-5^W23

Region	Phenotype	No.	Totals
0	<i>r Wx</i>	1187	
	+ <i>wx</i>	992	2179
1	<i>r wx</i>	157	
	+ <i>Wx</i>	377	534

% recombination *r1-Wx1*=19.7±.7

B) 2 point linkage data for *g1-Wx1* T9-10(8630)

Testcross: [*G1 Wx1* T9-10(8630) x *g1 wx1 N*] x *g1 wx1 N*
 source:93W-1459-5^W23

Region	Phenotype	No.	Totals
0	+ <i>Wx</i>	761	
	<i>g wx</i>	755	1516
1	<i>g Wx</i>	47	
	+ <i>wx</i>	25	72

% recombination *g1-Wx1*=4.5±.5

zb1* and *zb2* stocks at the Stock Center are allelic to *zb3

--Stinard, P, Sachs, MM

The *zb1* and *zb2* stocks maintained at the Stock Center seem to be allelic to *zb3* (Karen Cone, personal communication; we are also in the process of confirming this result). This may be due to a mix-up in these stocks that might have occurred when they were received by the Stock Center in 1943. If anyone has independently-maintained lines of *zb1* and *zb2* that do not trace back to the Stock Center, or were obtained from the Stock Center prior to 1943, we would appreciate receiving them so that we can test them against *zb3* and add them to the Stock Center's collection. Our address is:

Maize Genetics Cooperation • Stock Center
 S-123 Turner Hall
 1102 S. Goodwin Ave.
 Urbana, IL 61801

Linkage tests of *waxy1* marked reciprocal translocations at the MGCSC

--Jackson, JD, Stinard, P

In the collection of A-A translocation stocks maintained at MGCSC is a series of *waxy1*-linked translocations that are used for mapping unplaced mutants. Also new *wx1*-linked translocations are being introduced into this series and are in a conversion program to convert each translocation to the inbred backgrounds M14 and W23. These inbreds are then crossed together to produce vigorous F1's to fill seed requests. Over the years, pedigree and classification problems arose during the propagation of these stocks. We have been able to sort through the problem ones, and we can now supply good sources proven by linkage tests to include the correct translocated chromosomes. Additional translocation stocks will be tested as time allows. Below is a summary of those we have completed testing. Additional pedigree information on bad sources is available should anyone want to check on sources supplied to them by the Stock Center.

Table 1. *wx1* T3-9(8562) (3L.65; 9L.22) (COOP source).

- A) The M14 source showed no linkage of *wx1* with *a1*.
 B) The W23 source showed linkage of *wx1* with *a1*.

B) 2 point linkage data for *a1-wx1* T3-9(8562)

Testcross: [*A1 wx1* T3-9(8562) x *a1 Wx1 N*] x *a1 wx1 N*

source:82-087-3 ^W23

Region	Phenotype	No.	Totals
0	<i>a Wx</i>	2292	
	+ <i>wx</i>	2019	4311
1	+ <i>Wx</i>	816	
	<i>a wx</i>	857	1673

% recombination *a1-Wx1*=2.8±.2

Table 2. *wx1* T3-9(8562) (new source from D. Robertson).

2 point linkage data for *a1-Wx1* T3-9(8562)

Testcross: [*A1 wx1* T3-9(8562) x *a1 Wx1 N*] x *a1 wx1 N*

source:93W-1477-6

Region	Phenotype	No.	Totals
0	<i>a Wx</i>	1328	
	+ <i>wx</i>	906	2234
1	+ <i>Wx</i>	458	
	<i>a wx</i>	397	855

% recombination *a1-Wx1*=2.8±.3

Table 3. *wx1* T5-9c (5S.07; 9L.10).

- A) The M14 source showed no linkage of *wx1* with *a2*.
 B) The W23 source showed linkage of *wx1* with *a2*.

B) 2 point linkage data for *a2-wx1* T5-9c

Testcross: [*A2 wx1* T5-9c x *a2 Wx1 N*] x *a2 wx1 N*

source:92-411-1 ^W23

Region	Phenotype	No.	Totals
0	<i>a Wx</i>	1270	
	+ <i>wx</i>	1101	2371
1	+ <i>Wx</i>	142	
	<i>a wx</i>	74	216

% recombination *a2-Wx1*=8.3±.5

Table 4. *wx1* T5-9(8854) (5S.33; 9S.36).

This source showed no linkage of *wx1* with *a2*.

Table 5. *wx1* T5-9(022-11) (5S.30; 9L.27).

2 point linkage data for *a2-wx1* T5-9(022-11)

Testcross: [*A2 wx1* T5-9(022-11) x *a2 Wx1 N*] x *a2 wx1 N*

source:93-2128-1-

Region	Phenotype	No.	Totals
0	<i>a Wx</i>	1978	
	+ <i>wx</i>	1686	3664
1	+ <i>Wx</i>	91	
	<i>a wx</i>	53	144

% recombination *a2-Wx1*=3.8±.3

Table 6. *wx1* T9-10b (9S.13; 10S.40).

- A) The M14 sources showed linkage of *wx1* with *bf2*.
 B) The W23 sources showed linkage of *wx1* with *bf2*.

A) 2 point linkage data for *bf2-wx1* T9-10b

Testcross: [*Bf2 wx1* T9-10b x *bf2 Wx1 N*] x *bf2 wx1 N*

source:82-116-1^M14

Region	Phenotype	No.	Totals
0	<i>bf Wx</i>	334	
	+ <i>wx</i>	312	646
1	+ <i>Wx</i>	46	
	<i>bf wx</i>	19	65

% recombination *r1-Wx1*=9.1±1.1

B)2 point linkage data for *b12-wx1* T9-10b
 Testcross: [*B12 wx1* T9-10b x *b12 Wx1 N*] x *b12 wx1 N*
 source:82-117-1^W23

Region	Phenotype	No.	Totals
0	<i>b1 Wx</i>	812	
	+ <i>wx</i>	892	1704
1	+ <i>Wx</i>	40	
	<i>b1 wx</i>	39	79

% recombination *r1-Wx1*=4.4±.5

Table 7. *wx1* T1 9-10(059-10) (9S.31; 10L.53).

2 point linkage data for *r1-wx1* T9-10(059-10)
 Testcross: [*r1 wx1* T9-10(059-10) x *R1 Wx1 N*] x *r1 wx1 N*
 source:93-2069-1

Region	Phenotype	No.	Totals
0	+ <i>Wx</i>	165	
	<i>r wx</i>	362	527
1	<i>r Wx</i>	6	
	+ <i>wx</i>	37	43

% recombination *r1-Wx1*=7.5±1.1

Table 8. *wx1* T1 9-10(8630) (9S.28; 10L.37).

All of the *wx1* T9-10(8630) sources showed no linkage of *wx1* with either *r1* or *g1*.
 A new potential crossover has been identified.

WALTHAM, MASSACHUSETTS
 University of Massachusetts

Supermaize research

--Galinat, WC

The U.S. Cornbelt maize as well as European maize is undergoing an adaptation in plant and ear architecture that allows the plant to grow and produce a good ear despite the stress of extreme crowding. During the last 20 years, the concentration rate and drought tolerance have increased dramatically together with a decrease in row width to about 15 inches. Neither man nor

horse can now enter a field of mature maize but there is no problem with the combine harvester. The goal of this practice is to saturate the field with tightly packed maize plants in order that virtually all of the solar radiation is intercepted by the maize and its energy is chemically fixed by photosynthesis and translocated into storage as carbohydrates and other foods in grain on ears. The plant and ear should be designed for maximum energy storage and minimum energy wastage to barren ears, excess vegetation, weeds, insects and diseases. To achieve this goal, the necessary genes must be assembled by recombination. Shading from tassels should be reduced in the farmers' crop field and yet the seedsman must have adequate pollen production by the male rows to fertilize production of hybrid seed in crossing fields with three or four times as many female rows as male rows. Ideally, the solar energy intercepting canopy should be close to and just above the storage facility, the ear with kernels. The leaves from adjacent plants should not significantly overlap and shade each other in a lethal competition for solar energy.

All of these problems may be resolved to various degrees with the assemblage of a family of yield enhancing genes. The ultimate construction will be a supermaize of the future (Table 1).

The changes in the architecture of the maize plant suggested by use of the genes listed above are going to work as an extension

Table 1. A family of genes leading to supermaize.

Trait name	Gene	Problem solved
unbranched tassel	<i>ub</i>	Reduces shading of leaves by tassels
tassel ramosa	<i>ra-D</i>	Restores good pollen production to male rows in crossing field hybrid seed production
eye stabber	<i>es</i>	Upright short leaves for tolerance to crowding
umbrella	<i>umb</i>	Radial whorl of leaves, the solar panel, above ear
broad leaf	<i>Bl</i>	Wider leaves intercept more solar energy
long ear	<i>Le</i>	Larger energy storage facility



Figure 1. Three forward looking farmer-breeders from three different ages.

- 1-1, The first Americans had gardens of selected teosinte.
- 2, Most secondary branches were condensed into fascicles of ears.
- 3, Apical dominance of the uppermost ear with recombination of four ranking and paired female spikelets concentrated the energy into an eight-rowed ear of maize.
- 4, Year 1620. The Northern Flints received by the Pilgrims from the American Indians.
- 5,-6, Years 1950 to 1990. The modern farmer with hybrid maize, tractor and harvesting machines increases the density of plant populations.
- 7, Supermaize of the future.

of the evolutionary trends already present in Cornbelt maize for adaptation to increasingly higher density plant populations. They build upon the present background foundations by enhancing the elite changes already established. They do the same thing - only better. Their use is based on common knowledge of how the plant works and how it is leading toward a supermaize for the future (Fig. 1).

The making of supermaize, like the wisdom of evolution along a pathway toward adaptation to a certain environment, depends on the recombination of genes which cooperatively serve the adaptive purpose. Under natural selection, the encounter of such cooperating genes is due to random chance and so progress is usually slow. Under domestication, it is the human mind which finds and brings together by recombination those genes which cooperate to serve mankind's purposes of abundant food and/or beauty and progress is usually rapid. It is hoped that humans will have the necessary wisdom to direct maize evolution along a domestic pathway that will serve human survival, civilization, peace and democracy.

Plant - animal symbiosis

--Galinat, WC

In last year's Maize Newsletter (71:85-86, 1997) I had an item on the O₂ - CO₂ cycle in relation to trait technology, to which I now want to add my drawing with a descriptive caption that completes the story of plant - animal symbiosis (Fig. 2).

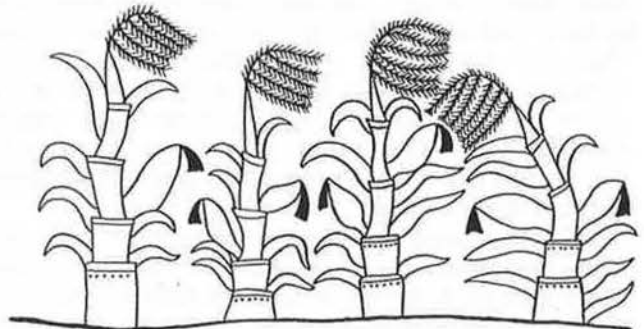


Figure 2. Plant - animal symbiosis. Complementation between the hemoglobin of animals and the chlorophyll of plants allows symbiotic survival of both, as the O₂ waste of plant photosynthesis is essential to animal respiration and growth while the animal wastes of CO₂, NH₃ and CH₄ are important to plants. Because animals lack chlorophyll, they must obtain all of their food, directly or indirectly, from plants.

Maize genetics ten centuries before Mendel, as suggested by Indian art

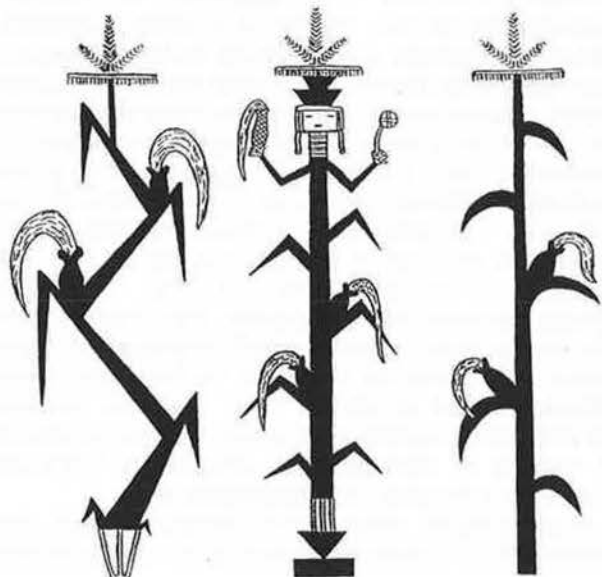
--Galinat, WC

Plate 1. Mendelian Ratios. Here reproduced from a pot of the Peruvian Mochica culture, which flourished in the Chicama Valley during the early part of the Christian Era, are four maize plants along a single ground line (from Lehmann and Doering, The Art of Old Peru, Ethnological Inst. of the Ethnographical Museum, Berlin, 1924.) They are drawn in a realistic manner of Rembrandt as if intended to represent real inherited differences rather than a suggestive design of Picasso. The ratios of the different plant types are consistent with the possibility that these first



Americans were familiar with the principles of Mendelian inheritance some ten centuries before Mendel. There are two independent 3:1 type F₂ ratios. From left to right, there are three erect plants to one semi-lazy, and then from right to left there are three of the two-eared plants to one of the one-eared type (which is much larger than the ears from two-eared plants). There is also a 1:1 backcross ratio for adventitious brace root development: 2 single node : 2 double node.

Plate 2. A bonding relationship between the American Indians and maize is apparent. This dedication could be the incentive that resulted in pre-Mendelian maize genetics. The closeness of the Indian-maize relationship is expressed in the Picasso-type sandpainting designs made by the Navajo people and others from the American Southwest (Newcomb and Richard, Sandpaintings



of the Navajo Shoot Chant, J. J. Augustine Pub., New York, 1946). Their identification with maize and wisdom about its pollination is revealed in the form of "Maize People" in the sandpaintings. The people have roots instead of legs and their arms replace an upper pair of leaves. They carry a ripe ear with silks in their right hand and in the left, a small circular object on a long line representing a pollen grain on a silk. The wisdom of this comes from the regal human-like head directing that pollen must move from the tassel to the silks of the ear in order to allow seed development. Portrayed on either side of the maize person are two different plant habits and all three designs have different leaf types. Some of these differences in plant and leaf design are due to the insensitivity of the sandpainting technique. With their hands occupied holding colored sand, placement of a trickle of dry pigments down on the desert floor in a pattern came from the large joints of the elbow and shoulder, resulting in broad curvatures and abrupt sharp changes in direction such as the stylized work of Picasso. They could not make realistic photographic-type illustrations of real biological differences by delicate finger control over line placement, as by the best artists of pre-photographic days.

At first the sand paintings were made from memorized designs for temporary use in chant ceremonies. Each time they had to be made anew by trickling dry pigments down onto the desert floor. Eventually the designs became preserved in photographs and sketches made from actual sandpaintings.

WOODWARD, OKLAHOMA

Southern Plains Range Research Station, USDA-ARS

Improvement of anther culture response of apomictic maize-*Tripsacum* hybrids

--Li, YG, Bai, DP, Kindiger, B

The gene(s) controlling apomictic reproduction in *Tripsacum* have been assigned to the long arm of chromosome 16. In addition, the long arm of *Tripsacum* chromosome 16 (Tr16L) has been translocated to the long arm of maize chromosome 6 (Mz6L) generating a Mz6L-Tr16L translocation stock (Kindiger et al., Genome 39:1133-1141, 1997). The stock carries 29 intact maize chromosomes, the Mz6L-Tr16L translocation, and 8 additional *Tripsacum* chromosomes. The form of apomictic development in *Tripsacum dactyloides* is characterized as being diplosporous pseudogamy of the *Antennaria* type which results in the complete omission of normal meiosis. This omission of meiosis precludes the reduction of the genome in the megaspore mother cells and therefore produces unreduced 2n egg cells. The genomic constitution of apomictic hybrids can be altered either by a rare event of 2n + n mating or the generation of rarely occurring polyploids (Kindiger et al., Genetica 92:197-201, 1994, Kindiger et al., Crop Sci. 36:1108-1113, 1996). An alternative approach can involve microspore culture. The microspore mother cells undergo normal meiosis during microsporogenesis and the resultant microspores are viable until the first meiotic division (Kindiger, Genome 36:989-997, 1996). Therefore microspore and anther culture methods could provide an alternative approach for reducing or eliminating the unnecessary *Tripsacum* chromosome in the Mz6L-Tr16L translocation stock.

A total of 24,000 anthers from 80 individual apomictic plants carrying Mz6L-Tr16L translocation were grown in the green house

and were cultured on both solid and liquid YP basal medium containing 0.1 mg/l 2, 4, 5-triiodobenzoic acid (TIBA) in a growth chamber at 28 C in the summer of 1996. All tassels were pretreated for 12 to 14 days at 8 C. Enlargement in a few anthers was observed after two months of culture. The poor response indicate a lack of genetic components in the Mz6L-Tr16L stock for response to anther culture. The translocation stock was, therefore, crossed to the DH139/29 maize line to improve the culture responses of this stock. The DH139/29 maize line was bred for high response to microspore culture and is protected under U.S. Patent (#5306864). The cross was again achieved through 2n + n mating and the resultant hybrids carry 39 intact maize chromosomes, the Mz6L-Tr16L translocation, and 8 additional *Tripsacum* chromosomes. A total of 45 hybrid seed were harvested. Two thirds of the hybrid seed were planted in the greenhouse and one third were planted in field nursery during the spring 1997. Forty-two plants were successfully established. About 16,000 anthers from the 42 plants growing in both greenhouse and field were cultured as described above in 1996 and tassels were again pretreated for 12 to 14 days at 8 C. Callus was obtained after two months from two of the anthers cultured. However, these calli grew poorly and failed to regenerate when transferred to hormone free YP regeneration medium. This preliminary study indicates that the addition of 10 more maize chromosomes from the DH139/29 anther culture responsive line to the Mz6L-Tr16L translocation stock can make improvement in callus formation from anthers of the apomictic translocation stock.

Plant regeneration from somatic culture of apomictic maize-*Tripsacum* hybrids

--Li, YG, Kindiger, B

In an attempt to transfer apomixis from *Tripsacum* to maize using classical backcross- hybridization methods, a series of apomictic maize-*Tripsacum* hybrids have been generated. The *Tripsacum* chromosome number in these hybrids has been reduced to 9 from 36 in the original crosses between tetraploid maize (2n = 4x = 40) and tetraploid *Tripsacum* (2n=4x=72) (Kindiger et al., Crop Sci. 36:1108-1113, 1996). Recently, the long arm of *Tripsacum* chromosome 16 (Tr16L), which carries the apomixis gene(s), has been transferred to the long arm of maize chromosome 6 (Mz6L) via a Mz6L-Tr16L translocation (Kindiger et al., Genome 39:1133-1141, 1997). The stock carries 29 intact maize chromosomes, the Mz6L-Tr16L translocation, and 8 additional *Tripsacum* chromosomes. Since normal meiotic events are omitted during apomictic development, the genetic constitution of the apomictic hybrids can be changed only by sexual polyploidization or via rarely occurring polyhaploids (Kindiger et al., Genetica 92:197-201, 1994). Consequently, further reduction of the *Tripsacum* chromosome complement in this stock will be extremely difficult. In an attempt to circumvent this situation, tissue culture techniques were developed for this stock to facilitate chromosome manipulation.

Young unemerged inflorescences from field grown plants were taken in the early boot stage when the inflorescences were 2-3 cm long. The outer leaves were removed and the inner leaves were surface sterilized by misting the leaves several times with 70% ethanol. The remaining inner leaves were removed aseptically to expose the young intact inflorescence. The clean inflorescence was then cut into 2-3 mm pieces and placed on the callus induction

medium with 1 inflorescence per plate. Immature embryos from greenhouse grown plants were also used as explants to initiate culture. Immature embryos were collected 2 weeks after pollination when the embryos were about 2 mm in length. The caryopses were removed from the spikelets, surface sterilized for 5 minutes in 10% commercial bleach plus Tween 80 and rinsed three times in sterile distilled water. The embryos were then aseptically removed from the caryopses and placed onto the callus induction medium. The callus induction and maintenance medium for both inflorescences and immature embryo cultures was N6 basal salt containing 1.0 mg/l thiamine HCl, 0.5 mg/l each of pyridoxine HCl and nicotinic acid, 2.88 g/l L-proline, 10 μ M silver nitrate and 1.0 mg/l 2,4-D. Plates were incubated at 25 C in the dark for callus induction. Calli emerged easily from both inflorescences and immature embryos after three weeks culture and these calli were subcultured on the same medium for two more weeks. They then were transferred to hormone free N6 regeneration medium and incubated at 28 C under 16/8 hours (dark/light) photoperiod. One plant was regenerated from one of the 40 plates initiated from inflorescences after two months culture on regeneration medium. No plant was obtained from calli derived from immature embryos. These results indicate that genetic components controlling regeneration in this Mz6L-Tr16L translocation stock do exist and regeneration frequency in the stock can be improved by further manipulation of culture medium and protocol. Since somatic chromosome numbers do change with duration of culture, cell culture may play a role in the reduction and/or elimination of the extra *Tripsacum* chromosomes in these maize-*Tripsacum* hybrids.

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Quantitation of 18kDa HSP mRNA accumulation in metal-ion insulted maize seedling radicles and plumules

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In a study employing in situ techniques (Yang and Walden, MNL 71:55, 1997), we previously reported that maize root tips insulted with the heavy metals Cd and Zn accumulate mRNA for 18kDa HSPs in the epidermal and cortex regions of the root tip. These tissues are distinct from the active meristems and immature vascular bundles where these messages accumulate during heat shock induction (Greyson et al., 1996. Devel. Genet. 18:244-253). We now report preliminary results of quantitative RNA-Dot experiments in which RNA from metal-ion insulted radicles and plumules was screened with a general ORF probe for 18kDa HSP mRNAs and also with gene-specific probes for three members of the 18kDa HSP gene family.

Seedling growth, heat shock (HS), and heavy metal-ion insults were performed exactly as reported in the note cited above, while RNA extraction, filter-binding, preparation of labeled DNA probes, hybridization conditions, scanning, and quantitation were as described in Bouchard et al. (Maydica 38:135-144, 1993). The probes were Mhsp18-9-2, a subclone containing the ORF of clone Mhsp 18-9 (map designation *uwo11*), which is a common probe for mRNAs from all members of the maize shsp gene family, and subclones Mhsp18-9-3, Mhsp18-1-1, and Mhsp18-3-3, which

are gene-specific 3'-UTR regions for the three shsp family members with map designations *uwo11*, *uwo9*, and *uwo10* respectively. The results, normalized to the signals seen in the untreated control for each probe set to 100%, are shown in Tables 1 and 2.

Table 1. Radicles.

Treatment Probe	C	HS	H ₂ O	Zn	Cd	K	Na	Cu
18-9-2 (ORF)	100	2209	154	880	984	138	126	381
18-9-3 (<i>uwo11</i>)	100	11967	100	3257	4853	100	100	893
18-1-1 (<i>uwo9</i>)	100	14150	100	1285	900	395	395	615
18-3-3 (<i>uwo10</i>)	100	2302	269	201	166	137	106	111

Table 2. Plumules.

Treatment Probe	C	HS	H ₂ O	Zn	Cd	K	Na	Cu
18-9-2 (ORF)	100	1850	98	421	658	75	96	138
18-9-3 (<i>uwo11</i>)	100	18210	100	1550	4865	100	100	250
18-1-1 (<i>uwo9</i>)	100	11475	100	1905	1325	100	100	510
18-3-3 (<i>uwo10</i>)	100	1301	94	102	135	112	161	87

These results, though still preliminary, appear both to confirm and extend our earlier in situ study in which the ORF probe alone was used. Induction of overall 18kDa mRNA accumulation due to insults to radicles with the divalent heavy metal-ions Zn and Cd relative to both controls and monovalent ions is evident with the ORF probe, just as seen in the in situ experiments, and a similar pattern is observed in RNA from plumules. In addition, there appears to be a more modest but consistent induction of mRNA accumulation with Cu. However, some interesting differences emerge when the results seen with the specific gene probes are compared with the 18 kDa common ORF probe and with one another. The overall level of 18 kDa mRNA accumulation under heavy metal-ion insult observed with the ORF probe is highest in the presence of Cd. This is also the case for the RNA of 18-9; indeed, the relative level of induction appears even higher. In the case of 18-1, however, the level induced by Zn seems to be higher than Cd in both radicles and plumules. The most intriguing results are seen for the RNA of 18-3, which appears to show little or no consistent induction by the divalent heavy metal-ion insults when compared with either the control and H₂O treatments or the monovalent heavy metal-ion insults.

The observations reported here suggest that the response to heavy metal-ion insult may exhibit specific differences among various members of the 18 kDa HSP gene family. This is consistent with observations we have previously reported for the developmentally modulated expression of these genes in the growing anthers and spikelets of maize (Bouchard et al., Maydica 38: 135-144, 1993). Interestingly, 18-3 shows no induction either from developmental cues or heavy metal-ion insults. This suggests that while the products of some members of the 18 kDa HSP gene family function in both heat stress and other contexts, the activities of others may be confined to heat stress conditions.

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Polymorphisms of synthesized populations from Chinese and exotic populations at 17 isozyme loci

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In order to create new germplasm for recurrent selection, we used elite southwestern China local varieties Lanhuazhao (L) and Wuxi (W) with exotic germplasm Lancaster (L), BSSSR (B), Mohuangjiu (M), and Suwan2 (S) to synthesize 4 new C0 populations, LBM, LLS, WBM, and WLS. The six populations among LBM, LLS, WBM, WLS and U.S. BSSSR and BS16 were analysed for isozyme variation at 17 enzyme marker loci for 8 isozymes. A random sample of seedlings growing at 25 C for 5 days from each population was assayed by horizontal potato starch (SIGMA Co.) gel using the method of Stuber et al. (1988). Mo17 and Oh43 were included as standards on each gel to aid gel reading. The method of denoting alleles (allozymes) and genotypes for locus has been described in detail by Stuber et al. (1988) and Kahler (1983). Gene diversity or heterozygosity was calculated for populations for each locus as follows: $H=1-\sum (f_i)^2$ where f_i is the frequency of the i th allele. The numbers of alleles detected for the 17 isozyme loci assayed in the 6 populations are given in Table 1. A total of 47 alleles was observed in 17 loci among populations assayed, with an average of 2.76 alleles per locus. The distribution of the alleles among the loci and populations is not uniform. The percentage of polymorphic loci ranged from 64.7 to 76.5. These data suggested that the 4 C0 populations from China were rich in allelic diversity as compared to U.S. BSSSR and BS16. For six populations, gene diversity (heterozygosity) for 17 isozyme loci is presented in Table 2. The total heterozygosities among each locus and population ranged from 0 to 66. The total heterozygosities of 17 loci among 6 populations ranged from 22.6 to 29.0. The results showed that the 4 C0 populations and BS16 and BSSSR maintain rich isozyme variation and also provide a review that the genetic bases of maize synthesized populations maintain abundant and plentiful genetic variation.

Table 1. Allele number at 17 isozyme loci in 6 populations.

Loci	WBM	LBM	LLS	WLS	BSSSR	BS16
adh1	2	2	2	2	2	2
cat3	2	2	2	2	1	2
e1	2	3	3	2	3	2
e2	1	2	1	2	2	1
e8	2	3	2	3	3	3
glu1	2	5	3	2	3	4
got1	1	1	1	1	1	1
got2	1	1	3	2	1	3
got3	1	1	1	1	1	1
mdh1	3	3	3	2	3	2
mdh2	4	3	3	3	2	3
mdh3	2	2	1	1	2	2
mdh5	2	2	2	2	2	2
mmm1	1	1	1	1	1	1
pgd1	3	3	5	3	3	4
pgd2	4	3	4	3	3	3
phi1	2	2	2	1	1	2
X	2.059	2.294	2.294	1.941	2.000	2.235
Polymorphic loci (%)	70.6	76.5	70.6	70.6	64.7	76.5

Table 2. Gene heterozygosities (gene diversity) for 17 isozyme loci of 6 maize populations.

Loci	WBM	LBM	LLS	WLS	BSSSR	BS16
adh1	39	25	21	10	60	7
cat3	18	9	32	32	0	4
e1	40	61	57	44	60	40
e3	0	42	0	12	28	0
e8	37	48	32	41	19	61
glu1	28	61	12	18	63	27
got1	0	0	0	0	0	0
got2	0	0	12	37	0	43
got3	0	0	0	0	0	0
mdh1	44	20	37	4	36	32
mdh2	47	54	66	57	39	56
mdh3	24	19	0	0	27	18
mdh5	41	20	29	6	45	30
mmm	0	0	0	0	0	0
pgd1	58	57	64	60	55	58
pgd2	66	52	63	66	46	58
phi1	11	6	18	0	15	0
H	26.6	27.9	26.1	22.4	29.0	26.5

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Science Watch (New York Times)

Wasps to the Rescue

[S]cientists have discovered another ingenious partnership between plants and animals. To fight beet armyworm caterpillars, corn plants release a "cocktail" of airborne chemicals that attract parasitic wasps. The wasps, natural enemies of the caterpillar, home in on the scent and deposit their eggs on the enemy's body. When the wasp larvae hatch, they devour the caterpillar.

There are two puzzles here. Just what prompts the corn plant to send out its signal? Dr. H.T. Alborn of the Federal Department of Agriculture Agricultural Research Service in Gainesville, Fla., and five colleagues found that damage to the plant was not enough to set off the alarm. In the current issue of the journal *Science*, they identified the triggering substance as a compound in the caterpillar's saliva called volicitin.

Which leads to puzzle No. 2: Why would the caterpillar secrete a substance that betrays it unto death? It is speculated that volicitin might play some digestive or hormonal role. But nature has not yet divulged the secret.

(Alborn, H.T.; Turlings, T.C.J.; Jones, T.H.; Stenhalen, G.; Loughrin, J.H.; Tumlinson, J.H. 1997. An elicitor of plant volatiles from beet armyworm oral secretion. *Science* 276:945-949.)

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IV. MAIZE GENETICS COOPERATION STOCK CENTER



Maize Genetics Cooperation • Stock Center

USDA/ARS/MWA - Plant Physiology and Genetics Research Unit

&

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2010 seed samples have been supplied in response to 273 requests, for 1997. Of these, a total of 65 requests were received from 19 foreign countries. Approximately three fourths of our requests were received by electronic mail or through our order form on the World-Wide Web.

We rearranged the stock list, giving trisomics and B-A Translocation stocks their own categories. We hope that this will help cooperators find stocks of interest.

Approximately 5 acres of nursery were grown this summer. Despite cool weather in May slowing germination in our first planting, growing conditions during the rest of the season were nearly optimal and with irrigation we obtained good increases of most stocks.

Special plantings were made of several categories of stocks:

1. Approximately 1.5 acres was devoted to the vast mutant collection of Gerry Neuffer with special attention also given to the collection of mutants that we have obtained from Donald Robertson.

2. Plantings were also made from donated stocks from the collections of James Birchler (marked B-A translocation stocks), Ed Coe (various genetic stocks), Susan Gabay-Laughnan (male-sterile cytoplasms and restorers), Jerry Kermicle (*R1* alleles), Bryan Kindiger (apomictic maize/*Tripsacum* hybrids - being grown in a winter greenhouse), Michael McMullen (Brink pericarp color collection), Donald Miles (high chlorophyll fluorescence mutants), Gerry Neuffer (EMS-induced mutants), and others. We expect to receive additional accessions of stocks from maize geneticists within the upcoming year.

3. We also made a special planting of characterized unplaced mutants to map to chromosome arm using B-A translocations. We were able to place the mutants *piebald4*, *virescent13*, *viviparous10*, and *viviparous12* with a high degree of certainty, and made tentative placements of *luteus3*, *luteus4*, and *orobanche2* (see MNL article on our TB mapping results). We plan to continue this mapping project next summer.

4. Approximately 1 acre each year is devoted to the propagation of the large collection of A-A translocation stocks. In this collection is a series of *waxy1*-linked translocations that are used for mapping unplaced mutants. Over the years, pedigree and classification problems arose during the propagation of these stocks. We were able to sort through the problem ones, and we can now supply good sources proven by linkage tests to include the correct translocated chromosomes. Additional translocation stocks will be tested as time allows.

Since the USDA discontinued its winter nursery program at Isabela, Puerto Rico, we grew last year's winter nursery at the Illinois Crop Improvement Association's facilities in Juana Díaz, Puerto Rico. The crop performed well there, probably better than at Isabela, and we received excellent increases. We plan to continue growing our winter nurseries at this location.

We sent backups of the stocks in our main collection to the National Seed Storage Laboratory (NSSL). With the help of Julie Fleming, this information was incorporated into GRIN.

We would like to make a special plea for cooperators to donate mutants that they have worked on, to the Stock Center in a timely manner. What often happens is that people publish on these mutants, and then forget about them. Seeds sit around at room temperature and lose viability, or get eaten by insects and mice, or even get discarded. These mutants are lost forever, and the research that was done on them can never be replicated or followed up. We recently received the only known existing seeds of the mutant *ageotropic1* (*agt1*). The seeds did not grow when they were field planted, and we had to make heroic efforts to germinate them under pampered conditions and transplant them to the field. Fortunately, with only about five percent germination even under these conditions, we were able to recover the mutant. However, we could have just as easily lost it. Please, as soon as you have published on your mutant, send seeds to the Stock Center. Do this now, before you forget.

Marty Sachs
Director

Philip Stinard
Curator

Janet Day Jackson
Senior Research Specialist

CATALOG OF STOCKS

CHROMOSOME 1

101A sr1 zb4 p1-ww
 101B sr1 P1-wr
 101C sr1 p1-ww
 101D sr1 P1-rr
 101F sr1 ts2 P1-rr
 102A Ws4-N1589
 102D Blh1-N1593
 102F ms28
 103D vp5
 103DA vp5-DR3076
 103DB vp5-86GN4
 103DC vp5-86GN3
 103DD vp5-86GN6
 103DE vp5-86GN11
 103DF vp5-Mumm#1
 103DG vp5-N81
 103E zb4 ms17 p1-ww
 104F ms*-6034
 104G ms*-6044
 105A zb4 p1-ww
 105B zb4 P1-wr
 105C zb4 p1-ww br1
 105E ms17 P1-wr
 105F ms17 p1-ww
 106B ts2 P1-rr
 107A P1-cr
 107B P1-rr
 107C P1-rw
 107D P1-cw
 107E P1-mm
 107F P1-vv::Ac
 107G P1-or
 107H p1-ww
 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
 110H P1-wr br1 f1 bm2
 110K P1-wr br1
 111F Les20-N2457
 111G rs2
 111H Les5-N1449
 112B p1-ww br1 f1 bm2
 112E as1
 112H p1-ww br1
 112I p1-ww br1 gs1 bm2
 113B rd1
 113BA rd1-Wasnok
 113C br1 f1
 113E br1 f1 Kn1
 113K hm1; hm2
 113L Hm1; hm2
 114C br1 bm2
 114D Vg1
 114F br2 hm1; Hm2
 114G br2 hm1; hm2
 115C v22-8983
 115CA v22-055-4
 115J bz2-m::Ds; A1 A2 C1 C2 Pr1 R1
 116A bz2-m::Ds; A1 A2 Ac C1 C2 Pr1 R1
 116C an1 bm2
 116D an1-bz2-6923; A1 A2 Bz1 C1 C2 Pr1 R1
 116G an1
 116GA an1-93W1189
 116I bz2 gs1 bm2 Ts6; A1 A2 Bz1 C1 C2 R1
 117A br2
 117D tb1
 117DA tb1-8963
 117E Kn1
 118B Kn1 bm2
 118C lw1
 118CA lw1-3108
 118J Adh1-3F1124r53
 118K Adh1-1S5657; Adh2-33

118L Adh1-3F1124::Mu3
 118M Adh1-3F1124r17
 118N Adh1-1L14H; su1
 118O Adh1-Cm
 118P Adh1-FCm
 118Q Adh1-Ct
 119A Adh1-1S; Adh2-1P
 119B vp8
 119C gs1
 119D gs1 bm2
 119E Ts6
 119F bm2
 119H Adh1-FkF(gamma)25; Adh2-N
 119J Adh1-Fm335::Ds1
 119K Adh1-Fm335RV1
 119L Adh1-2F11::Ds2
 119M Adh1-1F725
 120A id1
 120B nec2
 120C ms9
 120CA ms9-6032
 120CB ms9-6037
 120CC ms9-6042
 120D ms12
 120E v22-055-4 bm2
 120F Mpl1-Sisco
 120G Mpl1-Freeling
 121A ms14
 121AA ms14-6005
 121B br2-mi8043
 121C D8
 121D lls1
 121DA lls1-N501B
 121E ty*-8446
 121G ct2
 121GA ct2-rd3
 124A v*-5688
 124B j*-5828
 124C w*-8345
 124CA w*-013-3
 124CB w*-8245
 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
 125C hcf13-N1097B
 125D hcf41-N1275C
 125E hcf50-N1481
 126A bz2 gs1 bm2; A1 A2 Bz1 C1 C2 R1
 126B id1-N2286A
 126C dek1-N928A
 126D dek1-N971
 126E dek32-N1322A
 126F o13
 126H P1-vv::Ac bz2-m::Ds
 126I P1-vv::Ac
 126J P1-ww-1112
 126K P1-ovov-1114
 126L P1-rr-4B2
 126M P1-vv-5145
 126N dek1-N1348
 126O dek1-N1394
 126P dek1-N1401
 127A bz2 zb7-N101 bm2
 127B dek1-N792
 127C dek2-N1315A
 127D dek22-N1113A
 127E fl
 127F Msc1-N791A
 127G Tir1-N1590
 127I gt1
 128A ij2-N8
 128B l16-N515

128C l17-N544
 128D pg15-N340B
 128E pg16-N219
 128F v25-N17
 128G py2-N521A
 128H spc2-N262A
 129A w18
 129AA w18-571C
 129B wlu5-N266A
 129C zb7-N101
 129D emp1-R
 129E ptd1-MS1568
 129F dek*-MS2115
 129G dek*-MS6214
 130A o10-N1356

CHROMOSOME 2

201F ws3 lg1 gl2 b1
 203B al1
 203BA al1-Brawn
 203BB al1-y3
 203D al1 lg1
 203G al1-y3 gl2
 205A al1 lg1 gl2
 205B lg1
 205C lg1 gl2
 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-Salamini
 209E lg1 gl2 b1 sk1
 209I gl2-Parker's Flint
 210E gl2-PI3050-3
 210F gl2-PI200291
 210G gl2-PI239114
 210H gl2-PI251009
 210I gl2-PI251885
 210J gl2-PI251930
 210K gl2-PI262474
 210L gl2-PI262493
 210M gl2-PI267186
 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
 214B lg1 b1 gs2
 214C d5
 214D gl11 B1
 214E B1 ts1
 214J sk1
 214L lg1 gl2 mn1
 215A gl14
 215B gl11
 215C wt1
 215CA wt1-N472A
 215CB wt1-N666B
 215CC wt1-N178C
 215D mn1
 215E fl1
 215EA fl1-o4
 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
 217I Les10-NA607
 217J Les11-N1438
 217K Les15-N2007
 217L Les18-N2441

217M Les19-N2450
 217N cpc1-N2284B
 218A w3
 218C w3 Ch1
 218D Ht1-GE440
 218DA Ht1-Ladyfinger
 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
 219D Ht1 Ch1
 219G B1-Bolivia-706B; A1 A2 C1 C2 r1-g
 219H B1-Bolivia; A1 A2 C1 C2 Pr1 R1 r1-g
 219I B1-I; A1 A2 C1 C2 Pr1-Rhoades r1-r
 219J B1-I; A1 A2 C1 C2 Pr1-Rhoades r1-g
 219K B1-S; pl1-McClintock R1-g
 219L B1-S; pl1-McClintock R1-r
 220A Les1-N843
 220B ws3 lg1 gl2; Alien Addition T2-Tripsacum
 220D hcf15-N1253A
 220F os1
 221A gs2
 221C wlv1-N1860 Ch1
 221G wlv1-N1860
 224A w*-4670
 224AB w*-017-14-A
 224B v*-5537
 224H whp1; A1 A2 C1 c2 R1
 224I ws3-7752
 224J jimos*-7335
 224K glnec*-8495
 224L ws3-8949
 224M ws3-8991
 224N ws3-8945
 226A ws3-N2357
 227A dek3-N1289
 227B dek4-N1024A
 227C dek16-N1414
 227D dek23-N1428
 227E Les4-N1375
 227I nec4-N516B
 227K et2-2352
 227L et2-91g6290-26
 228A l18-N1940
 228B spt1-N464
 228C v26-N453A
 228CA v26-N605A
 228E B1-Bh
 228F ms*-6019
 228G ms*-6024
 228H ms*-6029
 228I ms*-6038
 228J ms*-6041
 229A rf3 Ch1
 229B v24-N424
 229BA v24-N576A
 229BB v24-N588A
 229BC v24-N350
 229C w3 rf3 Ch1
 229E emp2-MS1047
 229F dek*-MS1365
 229G dek*-MS4160
 229J dek*-PIE

CHROMOSOME 3

301A cr1
 301B bif2-N2354
 302A d1-6016
 302AA d1-N446
 302AB d1-N339
 302B d1 rt1
 302E d1-tall

303A d1 rt1 Lg3-O
303F g2
303FA g2-pg14::l
303FB g2-v19
303FD g2-56-3040-14
303FE g2-59-2097
303FF g2-94-1478
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
305K d1 cl1; Clm1-4
306D d1 Rg1 ts4
306F ref1-MS1185
307A Sdw2-N1991
307C pm1
308B d1 ts4
308E ra2
308F ra2 Rg1
309A a1-m3::Ds Sh2
309B a1-m1-5718::dSpm
309C a1-m1-5719A1::dSpm
309D a1-m1-5719A1::dSpm; Mod Pr1
309E a1 Sh2; Spm-w
309F a1-m2-8417::dSpm
309G a1-m2(os)-o1
309H a1-m2-7991A-o2
309I a1-m2-7995::dSpm
309J a1-m2-7977B::dSpm
309K a1-m2-8012A-p1
309L a1 Sh2; Spm-s
309N a1-m2-7995B
309O a1-m1-5996-4::dSpm
309P a1-m1-5719A1::dSpm; Spm-i
309Q a1-m5::Spm-w; Spm-s
309S a1-m2-8411A::Spm-w Sh2
309T a1-m2-7981B6::Spm-w
309U a1-m2-8409::Spm-i
309V a1-m5::Spm-w Sh2
309W a1-m2-8011::Spm-w Sh2
309X a1 Sh2; Spm-w-8745
309Y a1 Sh2; Spm-i
309Z a1-m1-5720-o2
310C ra2 lg2
310D Cg1
311A cl1
311AA cl1-N2
311B cl1; Clm1-2
311BA cl1-7716; Clm1-2
311C cl1; Clm1-3
311D cl1-p; Clm1-4
311E rt1
311F ys3
311G Lg3-O ys3
312A Les14-N2004
312B Les17-N2345
312D Lg3-O
312G brn1-R
312H g2 brn1-R
312I brn1-R cr1
312J brn1-R ra2 lg2
312K brn1-Nelson
312L brn1-3071
312M ms23
313A gl6
313AA gl6-gl7
313D ms3
313DA ms3-6008
313DB ms3-6009
313DC ms3-6043
313DD ms3-6020
314A gl6 lg2 A1; A2 C1 C2 R1
314C gl6 lg2 a1-m et1; A2 C1 C2 Dt1 R1
314F Rg1 gl6 lg2
314G gl6 lg2
315B Rg1 gl6
315C Rg1
315D A1-b(P415); A2 C1 C2 R1

315I A1-m2(os)-p1
315J A1-m2(os)-r2
315K a1-m2-7991A-o1
315L a1-m2-7991A-p2
315M a1-m2-7991A-p3
315N a1-m2-7991A-p4
315O a1-m2-7991A-p4b
315P a1-m2-7991A-p5
315Q a1-m2-8010A-o2
315R A1-m3-r1a sh2-m1::Ds
315S a1-m5-o1
315T a1-m5-o2
315U A1-m5-r1
315V A1-m5-r4
315W A1-m5-r5
316A ts4
316B a1-N796
316C dek5-N1339A
317F gl6 ts4 lg2
317I a1-m1-5996-4m::dSpm; Spm
317J a1-m2::Spm-s; Spm-w
317K a1-m2-7991A::Spm-s
317L a1-m2-8004::dSpm
317M a1-m2-8010A::Spm-s
317N a1-m2-8011::Spm-w
317O a1-m2-8012A
317P a1-m2-8147
317Q a1-m2-8167::dSpm
317R a1-m2-8414C
317S a1-m2-8549C
317T a1-m5::Spm-w Sh2
317U a1-m5::Spm-w sh2-1
317V a1-m1-OS::Spm
317W a1-m1-5720::Spm
317X a1-m1-6078::dSpm
317Y a1-m2-8409-2
317Z A1 def-1260
318A ig1
318B ba1
318C y10-7748
318D hcf19-N1257A
318E sh2-N391B
318F sh2-N2340
318G na1
318H vp1-Mc#2
318I y10-8624
319A lg2 A1-b(P415) et1; A2 C1 C2 Dt1 R1
319C lg2 a1-m et1; A2 C1 C2 dt1 R1
319D lg2 a1-m et1; A2 C1 C2 Dt1 R1
319F lg2 a1-st et1; A2 C1 C2 Dt1 R1
319G lg2 a1-st et1; dt1
320A lg2
320C lg2 na1
320E et1
320F A1 sh2; A2 b1 C1 C2 pl1 R1
320K sh2-94-1001-11
320L sh2-94-1001-58
320M sh2-94-1001-1003
320N a3-Styles; B1-b Pl1-Rhoades r1-g
320O a3-Styles; B1-b Pl1-Rhoades R1-nj
321A A1-d31; A2 C1 C2 R1
321B lg2 a1; A2 C1 C2 dt1 R1
321C lg2 A1-b(P415) et1; A2 C1 C2 dt1 R1
321D a1-m4::Ds; A2 C1 C2 R1
321E a1-rUq; A2 C1 C2 R1
321F a1-Mum1; A2 C1 C2 R1
321G a1-Mum2; A2 C1 C2 MuDR R1
321H a1-Mum3; A2 C1 C2 R1
321I a1-Mum4; A2 C1 C2 R1
321J a1-Mum5; A2 C1 C2 R1
322A A1-d31 sh2; A2 C1 C2 dt1 R1
322B A1-d31 sh2; A2 C1 C2 Dt1 R1
322C A1-Mum3-Rev; A2 C1 C2 R1
322F a1-m; A2 b1 C1 dt1 pl1 R1
322J et1-27
322K et1-34
322L et1-2162
322M et1-2320
322N et1-2424

322P et1-3191
322Q et1-3328
322S et1-84-6013
322T et1-88g-9733
322U et1-89-90-1572
323A a1-m; A2 C1 C2 Dt1 R1
323D a1-m sh2; A2 C1 C2 Dt1 R1
323E a1-m et1; A2 C1 C2 Dt1 R1
323G a1-m1::rDt (Neuffer); A2 C1 C2 Dt1 R1
323H a1-st; A2 C1 C2 dt1 Mrh R1
323I a1-m1::rDt (Neuffer); A2 C1 C2 dt1 R1
324A a1-st; A2 C1 C2 Dt1 R1
324B a1-st sh2; A2 C1 C2 Dt1 R1
324E a1-st et1; A2 C1 C2 Dt1 R1
324G a1-st; A2 C1 C2 dt1 R1
324H a1 et1; A2 C1 C2 dt1 R1
324I a1-st et1; A2 C1 C2 dt1 R1
324J a1-sh2-del-Robertson; A2 C1 C2 R1
324K a1-Mus1; A2 C1 C2 R1
324L a1-Mus2; A2 C1 C2 R1
324M a1-Mus3
324N a1-Mus4
325A a1-p et1; A2 C1 C2 dt1 R1
325B a1-p et1; A2 B1 C1 C2 Dt1 Pl1 R1
325C a1-x1; A2 C1 C2 R1
325D a1-x3; A2 C1 C2 R1
325E A1 ga7; A2 C1 C2 R1
325G a3
325I a1-p; A2 C1 C2 Dt1 R1
325J a1-p; A2 C1 C2 Pr1 R1
325K a1-m3::Ds sh2-m1::Ds; A2 Ac C1 C2 R1
326A sh2-Elmore
326B vp1
326BA vp1-Mum3
326BC vp1-86N6
326BD vp1-86GN14
326BF vp1-86GN19
326BG vp1-Mum2
326BH vp1-Mum1::Mu
326C Rp3
326D te1-1
326DA te1-Forester
326DB te1-Grogan
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
329I pg2
329K yel*-8630
329L yel*-5787
330A 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
330L a1-mrh; A2 C1 C2 R1
332B dek5-N874A
332C dek24-N1283
332D Wrk1-N1020
332F gl19-N169
332G dek6-N627D
332H dek17-N330D
332I Lxm1-N1600
332M Spc1-N1376
332N wlu1-N28
332S Mv1
333A dek5-25
333AA dek5-MS33

CHROMOSOME 4

401A Rp4-a
401B Ga1
401C Ga1 su1
401D Ga1-S
401E Ga1-S; y1
401I ga1 su1
401J Ga1-M
401K Ga1-S su1
402A st1
402D Ts5
403A Ts5 fl2
403B Ts5 su1
405B la1-Pl239110
405BA la1-Funk:1087
405BC la1-N2020
405D la1-R su1 gl3
405G la1-R su1 gl4
406C fl2
406CA fl2-DR9234
406D fl2 su1
407D su1
407DA su1-N86
407DB su1-N2316
407DC su1-BKG489-13
407DD su1-Pl
407DE su1-R2412
407DF su1-N896A
407DG su1-N1161A
407DJ su1-N959
407DK su1-N1968
407E su1-am
407F su1-am; du1
408B bm3-Burnham su1
408C su1 zb6
408E bm3-91598-3
408J su1 ra3
408K su1; se1
409A su1 zb6 Tu1
410D su1 zb6 gl3
411A su1 gl4 j2
411B su1 gl4 o1
411F gl7 su1 v17
412C su1 gl3
412E su1 j2 gl3
412G su1 gl4 Tu1
413A su1 o1
413B su1 gl4
413D su1 C2-ldf1(Active-1); A1 A2 C1 R1
413F su1 de*-414E
413G v23 Su1 gl3; bm*-COOP
414A bt2
414AA bt2-Williams
414AB bt2-60-158
414AC bt2-9626
414B gl4
414BA gl4-Stadler
414BB gl4-gl16
414C gl4 o1
414E de*-414E
415A j2
415B o1-N1243
416A Tu1-A158
416B Tu1-l(1st)
416C Tu1-l(2nd)
416D Tu1-d
416E Tu1-md
416F Tu1 gl3
417A j2 gl3
417B v8
417C gl3
417D o1 gl3
417G j2 gl3 c2; A1 A2 C1 R1
418A gl3 dp1
418B c2; A1 A2 C1 R1
418D C2-ldf1(Active-1); A1 A2 C1 R1
418E dp1
418F o1
418G v17
419A v23-8914
419E gl7

419F D16 gl3 C2; a1-m A2 C1 R1
419H c2-m1::Spm; A1 A2 C1 R1
419L c2-m2::dSpm c2-m3::Mpi1
419J c2-Mum1
419K c2-m2::dSpm; Spm-s
420A su1 D14 C2; a1-m A2 C1 R1
420C nec*-rd
420CA nec*-016-15
420D yel*-8957
420F dp*-4301-43
420G w*-9005
420H D14 C2; a1-m A2 C1 R1
424C gl3-64-4
424D gl3-56-3120-2
424E gl3-56-3129-27
424G gl3-PI183683
424H gl3-PI251928
424I gl3-PI251938
424J gl3-PI254858
424K gl3-PI267180
424L gl3-PI267219
424M gl3-PI-311517
424N gl3-15
426A Gl5 Su1; gl20
427A cp2-o12
427AA cp2-N211C
427AB cp2-N1875A
427AC cp2-MS2608
427AD cp2-N912
427B dek25-N1167A
427C Ysk1-N844
427D orp1-N1186A; orp2-N1186B
427E dek8-N1156
427F dek10-N1176A
427G Ms41-N1995
427H dek31-N1130
427I Sosl-ref
428A gl5 Su1; gl20
428C nec5-N642
428D spt2-N1269A
428E wl2-N10
428F lw4; Lw3
428G bx1
428H gl5 su1; gl20
428L dsc1-MS2058

CHROMOSOME 5

501A am1 a2; A1 C1 C2 R1
501B lu1
501D ms13
501E gl17
501G gl17 a2; A1 C1 C2 R1
501I am1
502B A2 ps1-Sprague pr1; A1 C1 C2 R1
502C D9-N2319
502D A2 bm1 pr1; A1 C1 C2 R1
502E Ms42-N2082
502F Ni2-N1445
502G A2 ga10; Bt1
503A A2 bm1 pr1 yst1; A1 C1 C2 R1
504A A2 bt1 pr1; A1 C1 C2 R1
504C A2 bm1 pr1 zb1; A1 C1 C2 R1
504E A2 bt1; A1 C1 C2 R1
505B A2 pr1 yst1; A1 C1 C2 R1
505C A2 bt1 pr1 ga*-Rhoades; A1 C1 C2 R1
505D pr1-N1515A
505E pr1-N1527A
506A A2 v3 pr1; A1 C1 C2 R1
506B A2 pr1; A1 C1 C2 R1
506C A2 pr1 v2; A1 C1 C2 R1
506D na2 A2 pr1; A1 C1 C2 R1
506F A2 pr1 v12; A1 C1 C2 R1
506L A2 br3 pr1; A1 C1 C2 R1
507A a2; A1 C1 C2 R1
507AA a2-Mus2; A1 C1 C2 R1
507AB a2-Mus3; A1 C1 C2 R1
507AC a2-Mus1; A1 C1 C2 R1
507F a2 bm1 bt1 ga*-Rhoades; A1 C1 C2 R1
507G a2 bm1 bt1; A1 C1 C2 R1

507H A2 bt1 pr1; A1 C1 C2 R1
507I a2-m4::Ds; wx1-m7::Ac7
508A a2 bm1 bt1 pr1; A1 C1 C2 R1
508C a2 bm1 bt1 bv1 pr1; A1 C1 C2 R1
508F a2 bm1 pr1 yst1; A1 C1 C2 R1
508H a2-Mum1
508I a2-Mum2
508J a2-Mum3
508K a2-Mum4
508L bv1 pr1
509G a2-m1::dSpm; Bt1
509H a2-m1(l)::dSpm(class II)
509I pr1-m1
509J a2-m1::dSpm pr1-m2
509K a2-m1(ps)
509L a2-m1::dSpm; Spm-s
509M a2-m5::dSpm
509N A2-m1(os)-r1
510A a2 bm1 pr1 v2; A1 C1 C2 R1
510G a2 bm1 pr1 eg1; A1 C1 C2 R1
511C a2 bt1 pr1; A1 C1 C2 R1
511F a2 bt1 Pr1; A1 C1 C2 R1
511H a2 bt1; A1 C1 C2 R1
512C a2 bt1 pr1 ga*-Rhoades; A1 C1 C2 R1
512D vp2-N1136B
512E W14-N2445A
512F pb4
512G gl8-N166A
512H v13
512I lw2-vp12
513A a2 pr1; A1 C1 C2 R1
513C a2 pr1 v2; A1 C1 C2 R1
513D A2 pr1 sh4; A1 C1 C2 R1
513E a2 pr1 v12; A1 C1 C2 R1
514A a2 bm1 pr1; A1 C1 C2 R1
514B ae1-PS1
514C ae1-PS2
514D ae1-PS3
514E ae1-PS4
514F ae1-PS5
514G ae1-PS6
514H ae1-PS7
514I ae1-PS8
514J ae1-PS9
514K ae1-PS10
514L ae1-PS11
514M Ae1-5180-r4
515A vp2
515AA vp2-DR5180
515AB a2 vp2-green mosaic; A1 C1 C2 R1
515C ps1-Sprague
515CA ps1-8776
515CB ps1-881565-2M
515CC ps1-N80
515D bm1
515F bt1-N2308
515G bt1-N2309
516B bt1-R
516BA bt1-Elmore
516BB bt1-C103
516BC bt1-Singleton
516BD bt1-sh3
516BE bt1-sh5
516BH bt1-6-783-7
516BI bt1-Vineyard
516BJ bt1-T
516BL bt1-3040
516BM bt1-N797A
516C ms5
516D td1 ae1
516G A2 bm1 pr1 yg1; A1 C1 C2 R1
517A v3
517AB v3-8982
517B ae1
517BA ae1-EMS
517BB ae1-PS12
517BC ae1-PS13
517BD ae1-PS14
517BE ae1-PS15
517BF ae1-PS16

517E ae1 pr1 gl8
518A sh4
518AA sh4-Rhoades
518AB sh4-o9
518B gl8-Salamini
518BA gl8-R
518BB gl8-6:COOP
518BC gl8-6:Salamini
518BD gl8-10:COOP
518BE gl8-PI180167
518C na2
518D lw2
519A ys1
519AA ys1-W23
519AB ys1-5344
519AC ys1-N755A
519AD ys1-74-1924-1
519B eg1
519C v2
519D yg1
519E A2 pr1 yg1; A1 C1 C2 R1
519F A2 pr1 gl8; A1 C1 C2 R1
519H zb1
520A hcf38-N1273
520B v12
520C br3
520F A2 Dap1; A1 C1 C2 R1
520G A2 pr1 Dap1; A1 C1 C2 R1
520H Dap1-2
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
521J yg*-8951
521K lw3; lw4
521L w*-021-7
521N Inec*-5931
521NA Inec*-8549
521P lw3; Lw4
527A dek18-N931A
527B dek9-N1365
527C dek26-N1331
527D dek27-N1380A
527E grt1-N1308B
527F nec7-N756B
527G dek33-N1299
527H Msc2-N1124B
527I ppg1-N199
527J nec6-N493
528A Hsf1-N1595
528B wgs1-N206B
528C anl1-N1643
528CA anl1-330C
528E prg1-MS8186
528F ren1-MS807
528H dek*-MS2146
528I dek*-MS1182

CHROMOSOME 6

601C rgd1 y1
601F po1-ms6 y1 pl1
601H rhm1 rgd1 y1
601I rhm1 y1 l11
601J Wsm1 Mdm1; Wsm2 Wsm3
601K wsm1 mdm1; wsm2 Wsm3
601L Mdm1 y1
602A po1-ms6 wi1 y1
602C y1
602D rhm1 Y1
602J y1-w-mut
602K y1-gbl
602L y1-pb1
602M y1-8549
602N y1-Caspar
602O y1-0317
603A y1 l10
603AA y1 l10-1359

603B y1 l11-4120
603C y1 l12-4920
603D w15-8896 y1
603H mn3-1184 y1
604D y1 l15
604F y1 si1-mssi
604FA y1 si1-ts8
604H y1 ms1
604HA y1 ms1-Robertson
604I y1 ms1
604IA ms1-6050
605A wi1 y1
605C y1 pg11; pg12 Wx1
605E wi1 Y1 Pl1
605F wi1 Y1 pl1
606A y1 pg11-4484; pg12-4484 Wx1
606AA pg11-8925; pg12-8925
606AB pg11-48-040-8; pg12-48-040-8
606AC pg11-8563; pg12-8563
606AD pg11-8322; pg12-8322
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 C2 R1 sh1 wx1
607C y1 su2
607E y1 pl1 su2 v7
607H y1 Pl1-Bh1; A1 A2 c1 C2 R1 sh1 Wx1
607I y1 Pl1-Bh1; A1 A2 c1 C2 R1 sh1 skb1 wx1
608A gs3-N268
608B Y1 l12
608C sbd1-N2292
608D Les13-N2003
608F y1 pl1 w1
608G Y1 l11
609D Y1 su2
609DA Y1 su2-89-1273
609DB su2-PS1
609DC su2-PS2
609F ms1-Albertsen
610B D12 Pl1; a1-m A2 C1 C2 R1
610C pl1 sm1; P1-rr
610F Y1 pl1 su2 v7
610G hcf34-N1269C
610H Y1 D12 pl1; a1-m A2 C1 C2 R1
610I hcf36-N1271B
610J hcf48-N1282C
610K hcf26-N1263C
611A Pl1 sm1; P1-rr
611D Pt1
611E Y1 pl1 w1
611EA w1-7366
611I sm1 tan1-py1; P1-rr
611K Y1 Pl1 w1
611L w1; l1
611M afd1
611N sr4-N65A
611O o14-N924
612A w14
612B po1
612BA po1-ms6
612C l*-4923
612D oro1
612DA oro1-6474
612I tan1-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 C2 pr1 R1 v2
613D vms*-8522
613F w14-8613
613I tus*-5267
613J gm*-6372
613L w*-8954

613M yel*-039-13
613N yel*-7285
613P yel*-8631
613T pg11-8656; pg12-6656
627A dek28-N1307A
627B dek19-N1296A
627C vp*-5111
627D hcf26
627G dek*-MS1104; l*-1104

CHROMOSOME 7

701B In1-D
701D o2
701E o2-Mum1
701F Hs1
702A o2 v5
702B o2 v5 ra1 gl1
702I In1-Brawn
703A o2 v5 gl1
703D o2 ra1 gl1
703J Rs1-O
703JA Rs1-1025::Mu6/7
703K Rs1-Z
704B o2 ra1 gl1 sl1
704C o2-NA696
704D o2-NA697
705A o2 gl1
705B o2 gl1 sl1
705D o2 bd1
706A o2 sl1
707A y8 v5 gl1
707B In1; A1 A2 C1 C2 pr1 R1
707C In1 gl1; A1 A2 C1 C2 pr1 R1
707D v5
707E vp9
707EA vp9-3111
707EB vp9-86GN9
707EC vp9-86GN15
707F y8 gl1
707G In1 gl1; A1 A2 C1 C2 Pr1 R1
708A ra1
708G y8
709A gl1
709AA gl1-56-3013-20
709AB gl1-56-3122-7
709AC gl1-PI183644
709AD gl1-PI218043
709AE gl1-PI251652
709AF gl1-PI257507
709AG gl1-lstra
709AH gl1-BMS
709AI gl1-7L
709AJ gl1-9::COOP
709AK gl1-N212
709AL gl1-N269
709C gl1-m
710A gl1 Tp1
710B gl1 mn2
710E o5 gl1
710I gl1 Bn1
711A Tp1
711B ij1-ref::Ds
711G ts*-br
712A ms7
712AA ms7-6007
712B ms7 gl1
713A Bn1
713E Bn1 bd1
713H Bn1 ij1
713I bd1 Pn1
714A Pn1
714B o5
714BA o5-PS3038
714BC o5-N874B
714C o5-N1241
714D va1
715A Dt3; a1-m A2 C1 C2 R1
715C gl1 Dt3; a1-m A2 C1 C2 R1
716A v*-8647
716B yel*-7748
716C dlif1-N2389A
716F Les9-N2008

727A dek11-N788
727B wlu2-N543A
727D v27-N590A
727DA v27-N53B
727DB v27-N413C
727E gl1-cgl
727F Rs4-N1606
727G Rs1-O o2 v5 ra1 gl1
727H ms*-6004
727I ms*-6010
727J ms*-6013
727K ms*-6014
728A Px3-6
728B ptd2-MS3193
728C cp1
728D sh6-8601
728E sh6-N1295
728F ren2-NS326
728G dek*-MS2082
728H dek*-MS5153

CHROMOSOME 8

801A gl18-g
801B v16
801I yel*-024-5
801K v16 ms8
802A rgh1-N1285
802B emp3-N1386A
802G ms43
802H gl18-PI262473
802I gl18-PI262490
803A ms8
803B nec1-025-4
803D gl18-g ms8
803F nec1-7748
803G nec1-6697
804A v21-A552
804B dp*-8925
804C tb*-poey1013
804D wh*-053-4
804E w*-017-14-B
804F w*-034-16
804G w*-8635
804H w*-8963
805A fl3
805C gl18-g v21-A552
805E el1
805G ms8 j1
808A ct1
808B Lg4-O
808C Htn1
810A v16 j1; l1
810B j1
827A dek20-N1392A
827B dek29-N1387A
827C Bif1-N1440
827D Sdw1-N1592
827E Clt1-N985
827F pro1-N1058
827G pro1-N1121A
827H pro1-N1528
827I pro1-N1533
827J wlu3-N203A
827K pro1
827L pro1-Tracy
828A ats1
828C pro1-N1154A
828D pro1-NA342

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 C2 R1
901H yg2 C1 Bz1; A1 A2 C2 R1
901I yg2 C1 sh1 Bz1 wx1 K9S-l; A1 A2 C2 R1
902A yg2 c1 sh1 bz1 wx1; A1 A2 C2 R1
902B yg2 c1 sh1 wx1; A1 A2 C2 R1
902C yg2 c1 sh1 wx1 gl15; A1 A2 C2 R1

902D yg2 c1 sh1 Bz1 wx1 K9S-s; A1 A2 C2 R1
903A C1 sh1 bz1; A1 A2 C2 R1
903B C1 sh1 bz1 wx1; A1 A2 C2 R1
903D C1-l sh1 bz1 wx1; A1 A2 C2 R1
904B C1 sh1; A1 A2 C2 R1
904D C1 wx1 ar1; A1 A2 C2 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 C2 R1
905D C1 sh1 wx1 K9S-l; A1 A2 C2 R1
905E C1 sh1 wx1 v1; A1 A2 C2 R1
905G C1 bz1 wx1; A1 A2 C2 R1
905H c1 sh1 wx1; A1 A2 b1 C2 R1-scsm2
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
906G C1-l Sh1 Bz1 Wx1; Dsl
906H C1 Sh1 bz1 wx1; Ac
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
907I C1-S wx1; A1 A2 C2 R1
908A C1 wx1 da1 ar1; A1 A2 C2 R1
908B C1 wx1 v1; A1 A2 C2 R1
908D C1 wx1 gl15; A1 A2 C2 R1
908F C1 wx1 da1; A1 A2 C2 R1
909A C1 wx1 Bf1-ref; 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
910B c1 sh1 wx1 gl15 Bf1-ref; 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
910I sh1-bb1981 bz1-m4::Ds
910IA sh1-bb1981 bz1-m4::Ds; Ac
910L yg2-str
911A c1 wx1; A1 A2 C2 R1
911B c1 wx1 v1; A1 A2 C2 R1
911C c1 wx1 gl15; A1 A2 C2 R1
912A sh1
912AA sh1-1746
912AB sh1-9026-11
912AD sh1-60-155
912AF sh1-4020
912AG sh1-9552
912AH sh1-9626
912AI sh1-3017
912AJ sh1-6
912B sh1 wx1 v1
912E lo2
912H lo2 wx1
913C sh1 l7
913D sh1 l6
913E baf1
913F yg2-Mum1
913G yg2-Mum2
913H yg2-Mum3
913I yg2-Mum4
913J yg2-Mum5
913K yg2-Mum6
913L yg2-Mum7
913M yg2-Mum8
913N yg2-Mum9
913O yg2-DR83-106-3
913P yg2-DR83-106-5
914A wx1 d3-COOP
914K Wc1-ly; Y1
914L bz1-Mus1
914M bz1-Mus2
914N bz1-Mus3
914O bz1-Mus5
914Q bz1-Mus7

914R bz1-Mus10
915A wx1
915B wx1-a
915C w11
915D wx1-N1050A
915E wx1-Alexander
915F wx1-N1240A
916A wx1 v1
916C wx1 bk2
916E wx1 v1 gl15
916H v31-N828
917A wx1 Bf1-ref
917C v1
917D ms2
917DA ms2-6002
917DB ms2-6012
917E gl15-Sprague
917EA gl15-Lambert
917F d3-COOP
917FA d3-d2
917FB d3-015-12
917FC d3-072-7
917FD d3-8054
917FF d3-d2-Harberd
917FG d3-d2-Phillips
917FH d3-N660B
918A gl15 Bf1-ref
918B gl15 bm4
918C bk2 Wc1
918D Wc1
918F Wx1 Bf1-ref
918G Wc1 Bf1-ref bm4
918GA Wc1-Wh Bf1-ref bm4
918K bk2 v30
918L wx1 Wc1
919A bm4
919B Bf1-ref bm4
919C l6
919D l7
919G l6; l1
919I Bf1-DR-046-1
919J bz1-Mum9; MuDR
919K bz1-Mum4::Mu1
919L bz1-Mum1
919M bz1-Mum2
919N bz1-Mum3
919O bz1-Mum5
919P bz1-Mum6
919Q bz1-Mum7
919R bz1-Mum8
919S bz1-Mum9
919T bz1-Mum10
919U bz1-Mum11
919V bz1-Mum12
919W bz1-Mum15
919X bz1-Mum16
919Y bz1-Mum18
920A yel*-034-16
920B w*-4889
920C w*-8889
920E w*-8950
920F w*-9000
920G Tp3L-9SRhoades
920L ygzB*-5588
920M wnl*-034-5
920N pyd1
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 Wd1 wd1 C1 C1-I Ring 9S; A1
A2 C2 R1
924B C1-I Ring 9S; A1 A2 C2 R1
924C yg2
924D wd1
924E wd1 C1 sh1 bz1
924F C1 Sh1 sh1 Bz1 bz1 wx1 tiny
fragment 9
924G C1-I Bz1; Ac Dsl
924H c1 sh1 bz1 wx1; Ac
925A bz1-m1::Ds wx1-m9::Ac
925B wx1-m9::Ds; Ac
925C bz1-m2::Ac
925D Wx1-m9r1
925E bz1-m2(DII)::Ds wx1-m6::Ds
925F C1 sh1 bz1 wx1-m8::Spm-l8
925H bz1-m2(DI)::Ds wx1; R1-sc
925I c1-m2::Ds Wx1; Ac
925J c1-m858::dSpm wx1
925K c1-m1::Ds
926A sh1-m5933::Ds
926B Sh1-r3(5933)
926C Sh1-r6(5933)
926D Sh1-r7(5933)
926E Sh1-r8(5933)
926F Sh1-r9(5933)
926G Sh1-r10(5933)
926H Sh1-r11(5933)
926I sh1-m6233::Ds
926J Sh1-r1(6233)
926K Sh1-r2(6233)
926L C1-I sh1-m6258::Ds
926M Sh1-m6258-r1
926N Sh1-r6795-1
926O bz1-m5::Ac
926P Bz1-wm::Ds1
926Q Bz1-m1-p
926R Bz1-m2-r1
926S Bz1-m2(DII)-r1
926T Bz1-m2(DII)-r2
926U Bz1-m2(DII)-r3
926V sh1-bb1981 Bz1-m4-p1
926W sh1-bb1981 Bz1-m4-r6851
926X sh1-bb1981 Bz1-m4-r7840B
926Y sh1-bb1981 Bz1-m4-r8332
926Z Bz1-m5-p1
926ZA Bz1-m5-r1
926ZB Bz1-m5-r2
927A dek12-N873
927B dek13-N744
927C dek30-N1391
927D Les8-N2005
927E Zb8-N1443
927H C1 D17; a1-r A2 C2 R1
927I G6-N1585
927K Rld1-N1990
927L Rld1-N1441
928A v28-N27
928AA v28-N585
928AB v28-N697
928AC v28-N610
928B wlu4-N41A
928C ms20
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

928J C1 bz1-(r)d; A1 A2 C2 R1
928K C1 Sh1 bz1-s; A1 A2 C2 Mut R1
928L ms*-6006
928M ms*-6011
928N ms*-6018
928O ms*-6021
928P ms*-6022
928Q ms*-6027
928R ms*-6031
928S ms*-6046
928T ms*-6047
929E Dp9
930A wx1-Mum1
930B wx1-Mum2
930C wx1-Mum3
930D wx1-Mum4
930E wx1-Mum5
930F wx1-Mum6
930G wx1-Mum7
930H wx1-Mum8
930I wx1-Mum9
930J wx1-Mum10
930K wx1-Mum11
930L wx1-Mus16
930M wx1-Mus181
930N wx1-Mus215
931A Wx1-m5::Ds
931B wx1-m6::Ds
931C wx1-m6-o1
931D Wx1-m7-i1
931E Wx1-m8-r10
931F Wx1-m9-r3
931G Wx1-m9-r4

CHROMOSOME 10

X01A oy1-Anderson
X01AA oy1-yg
X01AB oy1-8923
X01B oy1 R1; A1 A2 C1 C2
X01C oy1 bf2
X01E oy1 bf2 R1; A1 A2 C1 C2
X02C oy1 zn1 R1; A1 A2 C1 C2
X02E oy1 du1 r1; A1 A2 C1 C2
X02G oy1 zn1
X02H Oy1-N1459
X02I Oy1-N1538
X02J Oy1-N1583
X02K Oy1-N1588
X02L Oy1-N1989
X03A sr3
X03B Og1
X03D Og1 R1; A1 A2 C1 C2
X03E oy1 y9
X04A Og1 du1 R1; A1 A2 C1 C2
X04B ms11
X04BA ms11-6051
X04D bf2
X04DA bf2-N185A
X05B Gs4-N1439
X05E bf2 sr2
X06A bf2 r1 sr2; A1 A2 C1 C2
X06C nl1 g1 R1; A1 A2 C1 C2
X06F bf2 R1 sr2; A1 A2 C1 C2
X07A nl1 g1 r1; A1 A2 C1 C2
X07C y9
X07CA y9-y12
X07D nl1
X08A vp10
X08B vp10-86GN5
X08C vp10-TX8552
X08F li1
X08FA li1-IL90-243Tco
X09B li1 g1 R1; A1 A2 C1 C2
X09EA g1-g4
X09EB g1-56-3004-24
X09EC g1-1-7(X-55-16)
X09ED g1-68-609-13
X09EE g1-ws2
X09EF g1-PI262473
X09F ms10
X09FA ms10-6001
X09FB ms10-6035

X09G li1 g1 r1; A1 A2 C1 C2
X10A du1
X10AA du1-PS1
X10AB du1-PS2
X10AC du1-PS3
X10AD du1-PS6
X10AE du1-PS4
X10AF du1-PS5
X10D du1 g1 r1; A1 A2 C1 C2
X10F zn1
X10FA zn1-N25
X10G du1 v18
X11A zn1 g1
X11D Tp2 g1 r1; A1 A2 C1 C2
X11E g1 R1 sr2; A1 A2 C1 C2
X11F g1 r1; A1 A2 C1 C2
X11H zn1 R1-r; A1 A2 C1 C2
X11I Tp2 g1 sr2
X12A g1 r1 sr2; A1 A2 C1 C2
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
X13E g1 r1-ch; A1 A2 C1 C2 wx1
X14A r1-r lsr1-Ej; A1 A2 C1 C2
X14E r1; A1 A2 C1 C2 wx1
X14F v18 r1; A1 A2 C1 C2
X14I r1-m3::Ds
X15B li1 r1 sr2; A1 A2 C1 C2
X15C R1-g; A1 A2 C1 C2
X15D r1-ch; A1 A2 C1 C2
X15F lsr1 R1-g sr2
X15G lsr1 r1-g sr2
X15H lsr1 R1-r:PI302369
X15I lsr1 R1-nj Mst1
X16B r1; A1 A2 abnormal-10 C1 C2
X16C R1-ch; A1 A2 C1 C2 P11
X16D r1 sr2; A1 A2 C1 C2
X16E r1 K10-II; A1 A2 C1 C2
X16F R1 K10-II; 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
X18B R1-sk.nc-2; A1 A2 C1 C2
X18C R1-st; A1 A2 C1 C2
X18D R1-sk; A1 A2 C1 C2
X18E R1-st Mst1
X18G R1-scm2; A1 A2 bz2 C1 C2
X18H R1-nj; A1 A2 bz2 C1 C2
X18I r1; A1 A2 C1 C2
X19A R1-sc:124
X19B w2
X19BA w2-Burnham
X19BB w2-2221
X19C li1 w2
X19D o7
X19F r1 w2
X19G r1-n19 Lc1; b1
X19H r1-g:ø Lc1; b1
X20B li1
X20C v18
X20F yel*-8721
X20H yel*-5344
X20HA yel*-8793
X20HB yg*-8962
X20HC yel*-8454
X24A cm1
X24B lep*-8691
X25A R1-scm2; a1-st A2 C1 C2
X25B R1-scm2; A1 A2 C1 C2
X25C R1-sc:122; A1 A2 C1 C2 pr1
X25D R1-scm2; A1 a2 C1 C2
X25E R1-scm2; A1 A2 c1 C2
X26A R1 r1-X1; A1 A2 C1 C2
X26B R1-scm2; A1 A2 C1 C2
X26C R1-sc:122; A1 A2 C1 C2
X26D R1-sc:5691; A1 A2 C1 C2
X26E R1-scm2; A1 A2 C1 C2 pr1 wx1
X26F R1-scm2; A1 A2 C1 C2 ln1-D
X26G R1-scm2; A1 A2 C1 c2-
m2::dSpm

X26H R1-scm2; A1 A2 C1 C2 wx1
X27A dek14-N1435
X27B dek15-N1427A
X27C w2-N1330
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
X27K v29-N418
X27L Les12-N1453
X28B R1-scm2; a1-m1::rD1 (Neuffer)
X28C R1-nj:Cudu; A1 A2 C1 C2
X28D Vsr*-N716
X28E Les3
X28F cr4-6143
X28G R1-nj:Chase; A1 A2 C1 C2
X28J R1-scm2; a1-m1-5719::dSpm A2
C1 C2
X28J R1-scm2; A1 A2 bz1 C1 C2
X28K ren3-MS1339
X29B dek*-MS2181

UNPLACED GENES

U140C I4
U140E I3
U140F Fas1
U140G ms22
U140H ms24
U240A Les7-N1461
U240D o11
U340D ws1-COOP ws2-COOP
U340DA ws1-Pawnee ws2-Pawnee
U340E y11
U340G oro2
U340H oro4
U440B gl13
U440C zn2
U440D ub1-76C
U440E frz1
U440F mg1-Sprague
U540A dv1
U540B dyl
U640A dsy1-Doyle
U640B dsy1-Russian
U640C pam1
U640D pam2
U640E ada1
U640F atm1 Adh1-1S5657
U740A abs1-PI254851
U740B y2
U740C lty1
U740D lty2
U740E lty3
U740F pi1 pi2
U740G Fbr1-N1602
U740H ad2-N2356A
U840A csp1-NA1173
U840B rli1-N2302A
U840C mrl1-IHO
U840D Les21-N1442
U840E zb3

MULTIPLE GENE

M141A A1 A2 B1 C1 C2 P11 Pr1 R1-g
M141AA A1 A2 B1 C1 C2 P11-Rhoades
Pr1 R1-g
M141B A1 A2 B1 C1 C2 pl1 Pr1 R1-g
M141D A1 A2 b1 C1 C2 pl1 R1-g
M241A A1 A2 B1 C1 C2 P11 Pr1 R1-g
M241C A1 A2 B1 C1 C2 P11 Pr1 R1-r
M241D A1 A2 b1 C1 C2 P11-Rhoades
r1-g
M340A A1 A2 B1 c1 C2 pl1 Pr1 R1-g
M340C A1 A2 b1 c1 C2 pl1 Pr1 R1-g
M341B A1 A2 B1 C1 C2 pl1 Pr1 R1-r
M341C A1 A2 b1 C1 C2 P11 Pr1 R1-r
M341CA A1 A2 b1 C1 C2 P11-Rhoades
Pr1 R1-r

M341D A1 A2 B1 c1 C2 Pl1 Pr1 R1-r
M341F A1 A2 b1 C1 C2 pl1 Pr1 R1-r
M441B A1 A2 B1 C1 C2 pl1 Pr1 R1-r
wx1
M441D A1 A2 B1 C1 C2 Pl1 Pr1 r1-r
M441F A1 A2 b1 C1 C2 pl1 Pr1 R1-g
wx1
M541B Oh43*ACR A1 A2 b1 C1 C2
pl1 Pr1 R1-g
M541F a1 A2 C1 C2 R1-nj
M541G A1 a2 C1 C2 R1-nj
M541H A1 A2 c1 C2 R1-nj
M541I A1 A2 C1-C2 R1-nj
M541J A1 A2 C1 c2 R1-nj
M541K A1 A2 C1 C2-ldf1(Active-1)
R1-nj
M541L A1 A2 bz1 C1 C2 Pr1 R1-nj
M541M A1 A2 Bz1 C1 C2 pr1 R1-nj
M541N A1 A2 C1 C2 gl1 in1 R1-nj
M541O A1 A2 C1 C2 In1-D R1-nj
M641C A1 A2 b1 C1 C2 pl1 Pr1 R1-r
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-C2 pl1
R1-g wx1 y1
M741H Stock 6; A1 A2 B1 C1 C2 Pl1
R1-nj
M741I Stock 6; A1 A2 C1 C2 R1
M841A A1 A2 C1 C2 pr1 R1 su1
M841B f1 wx1
M841C v4 wx1
M841E v2 wx1
M941A A1 A2 c1 C2 Pr1 R1 wx1 y1
MX40A Mangelsdorf's tester; a1 bm2
g1 gl1 j1 lg1 pr1 su1 wx1 y1
MX40C a1 D11 gl2 lg1 wt1
MX40D gl1 wx1 y1
MX40E gl8-R 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-l8

B-CHROMOSOME

B542A Black Mexican Sweet, B
chromosomes present
B542B Black Mexican Sweet, B
chromosomes absent

TRISOMIC

123A trisomic 1
223A trisomic 2
328A trisomic 3
422A trisomic 4
523A trisomic 5
615A trisomic 6
718A trisomic 7
807A trisomic 8
922A trisomic 9
X23A trisomic 10

TETRAPLOID

N102A Autotetraploid; A1 A2 B1 C1
C2 Pl1 Pr1 R1
N102D Autotetraploid; A1 A2 C1 C2
R1
N102E Autotetraploid; B chromosomes
present

N102EA Autotetraploid; B
chromosomes present
N102F Autotetraploid; A1 a2 C1 C2
R1
N103A Autotetraploid; P1-rr
N103B Autotetraploid; P1-vv::Ac
N103C Autotetraploid; P1-ww
N103D Autotetraploid; P1-wr
N103E Autotetraploid; P1-mm
N104A Autotetraploid; su1
N104B Autotetraploid; A1 A2 C1 C2
pr1 R1
N105B Autotetraploid; wx1 y1
N105D Autotetraploid; A1 a2 bt1 C1
C2 R1
N105E Autotetraploid; bt1
N106C Autotetraploid; wx1
N107B Autotetraploid; W23
N107C Autotetraploid; Synthetic B
N107D Autotetraploid; N6

**CYTOPLASMIC
STERILE/RESTORER**

C736A R213 (N); mito-N Rf1 rf2
C736AB R213 (T) Sterile; cms-T Rf1
rf2
C736B Ky21 (N); mito-N Rf1 Rf2 Rf3
RfC
C736C B37 (N); mito-N rf1 Rf2 rf3
rfC
C736CA B37 (T) Sterile; cms-T rf1
Rf2
C736CB B37 (T) Restored; cms-T
Rf1 Rf2
C736E Tr (N); mito-N Rf3 rfC rT
C736EA Tr (S) Restored; cms-S Rf3
rfC rT
C736F W23 (N); mito-N rf1 Rf2 rf3
RfC
C736FA W23 (N); mito-N rf1 Rf2 rf3
RfC
C736G B73 (N); mito-N rf1 Rf2 rf3
rfC
C736H L317 (N); mito-N rf3 RfC rT
C836A Wf9 (T) Sterile; cms-T rf1
rf2
C836B Wf9 (N); mito-N rf1 Rf2 rf3
rfC
C836C Wf9 (T) Restored; cms-T Rf1
Rf2 rf3 rfC
C836D Wf9 (S) Sterile; cms-S rf1
rf2 rf3 rfC
C836F Mo17 (N); mito-N rf1 Rf2 rf3
rfC
C836G Mo17 (C) Sterile; cms-C rf1
Rf2 rf3 rfC
C836H Mo17 (S) Sterile; cms-S rf1
Rf2 rf3 rfC
C936D K55 (N); mito-N Rf1 Rf2 rf3
RfC
C936DA K55 (N); mito-N Rf1 Rf2
rf3 RfC
C936F N6 (N); mito-N rf1 Rf2 rf3
RfC
C936FA N6 (N); mito-N rf1 Rf2 rf3
RfC
C936G N6 (T) Sterile; cms-T rf1 Rf2
Rf2
C936H N6 (T) Restored; cms-T Rf1
Rf2
C936I SK2 (N); mito-N rf1 Rf2 rf3
rfC
C936J SK2 (T) Sterile; cms-T rf1
Rf2
C936K SK2 (T) Restored; cms-T Rf1
Rf2
C936M 38-11 (N); mito-N rf1 Rf2 rf3
rfC

CYTOPLASMIC TRAIT

C337A NCS2
C337B NCS3

TOOLKIT

T318AA TB-3Ld Ig1; Ig1R1-nj
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)
T940D KYS (for chromosome
observations in pachytene
microsporocytes)

T3307A trAc8178; T2-9b (2S.18;
9L.22) wx1
T3307B trAc8178; T2-9c (2S.49;
9S.33) wx1
T3307C trAc8178; T2-9d (2L.83;
9L.27) wx1
T3307D trAc8163; T3-9(8447)
(3S.44; 9L.14) wx1
T3307E trAc8163; T3-9c (3L.09;
9L.12) wx1
T3307F trAc8183; T3-9(8447)
(3S.44; 9L.14) wx1
T3307G trAc8183; T3-9c (3L.09;
9L.12) wx1
T3308A trAc8200; T4-9g (4S.27;
9L.27) wx1
T3308B trAc6076; T5-9a (5L.69;
9S.17) wx1
T3308C trAc6076; T5-9c (5S.07;
9L.10) wx1
T3308D trAc8175; T5-9c (5S.07;
9L.10) wx1
T3308E trAc8193; T5-9c (5S.07;
9L.10) wx1
T3308F trAc8179; T5-9a (5L.69;
9S.17) wx1
T3308G trAc8181; T5-9a (5L.69;
9S.17) wx1
T3308H trAc8186; T5-9a (5L.69;
9S.17) wx1
T3309A trAc8196; T5-9a (5L.69;
9S.17) wx1
T3309B trAc6062; T6-9b (6L.10;
9S.37) wx1
T3309C trAc6063; T6-9b (6L.10;
9S.37) wx1
T3309D trAc8172; T6-9b (6L.10;
9S.37) wx1
T3309E trAc8184; T6-9b (6L.10;
9S.37) wx1
T3310A trAc8161; T7-9(4363)
(7ctr; 9ctr) wx1
T3310B trAc8173; T7-9(4363)
(7ctr; 9ctr) wx1
T3310C trAc8173; T7-9a (7L.63;
9S.07) wx1
T3310D trAc8190; T7-9(4363)
(7ctr; 9ctr) wx1
T3310E trAc8194; T7-9(4363)
(7ctr; 9ctr) wx1
T3310F trAc8185; T7-9a (7L.63;
9S.07) wx1
T3311A trAc8162; T8-9d (8L.09;
9S.16) wx1
T3311B trAc8182; T8-9d (8L.09;
9S.16) wx1
T3311C trAc8182; T8-9(6673)
(8L.35; 9S.31) wx1
T3311D trAc6059; T9-10b (9S.13;
10S.40) wx1
T3311E trAc6059; T9-10(8630)
(9S.28; 10L.37) wx1

T3311F trAc8180; T9-10b (9S.13;
10S.40) wx1
T3311G trAc8180; T9-10(8630)
(9S.28; 10L.37) wx1
T3312A Ds-1S1 P1-vv::Ac Dek1
T3312B Ds-1S2 P1-vv::Ac Dek1
T3312D Ds-1S4 P1-vv::Ac Dek1
T3312E Ds-1L1 P1-vv::Ac Bz2
T3312F Ds-1L3 Bz2; Ac
T3312G Ds-2S1 B1-Peru; P1-vv::Ac
T3312I Ds-2S3 B1-Peru; P1-vv::Ac
T3312J Ds-2S4; P1-vv::Ac
T3312L Ds-3L1 A1 Sh2; P1-vv::Ac
T3312M Ds-3L2 A1 Sh2; P1-vv::Ac
T3312O Ds-4L1 C2; P1-vv::Ac
T3312P Ds-4L3 C2; P1-vv::Ac
T3312Q Ds-4L4 C2; P1-vv::Ac
T3312S Ds-4L6 C2; P1-vv::Ac
T3312T Ds-4L7 C2; P1-vv::Ac
T3312U Ds-5L1 A2 Pr1 Bt1; P1-
vv::Ac
T3312V Ds-5S1 A2 Pr1 Bt1; P1-
vv::Ac
T3312W Ds-5S2 A2 Pr1 Bt1; P1-
vv::Ac
T3312Y Ds-9S1 C1-I wx1; Ac
T3312Z Ds-10L2 R1-sc; P1-vv::Ac

**B-A TRANSLOCATION (BASIC
SET)**

122A TB-1La
122B TB-1Sb
222A TB-1Sb-2L4464
222B TB-3La-2S6270
327A TB-3La
327B TB-3Sb
421A TB-4Sa
423E TB-4Lf
522A TB-5La
522C TB-5Sc
614B TB-6Sa
614C TB-6Lc
717A TB-7Lb
719A TB-7Sc
809A TB-8Lc
922B Wc1; TB-9Lc
922D TB-9Sd
X21B TB-10L19
X22A TB-10Sc

B-A TRANSLOCATION (OTHERS)

122C P1-wr; R1-nj TB-1Lc Y1
126G P1-vv::Ac bz2-m::Ds; A1 A2 Bz1
C1 C2 R1 TB-1Sb
221I B1-Peru; TB-2Sa
221J TB-2Sb
225A TB-3La-2L7285 (3L.39;
2L.26)
225B TB-1Sb-2Lc (1S.77; 1S.05)
320P TB-1La-3Le (3L.45; 1L.58)
320Q TB-5La-3L(1)
320R TB-5La-3L(2)
320S TB-5La-3L(3)
327C TB-3Lc
327D TB-3Ld
329Z T3-B(La); T3-B(Sb)
331A TB-1La-3L5267
331B TB-1La-3L4759-3
331C TB-1La-3L5242 (3L.65; 1L.90)
331E TB-3Lf
331F TB-3Lg
331G TB-3Lh
331H TB-3Li
331I TB-3Lj
331J TB-3Lk
331K TB-3Li
331L TB-3Lm
420B TB-9Sb-4L6504
420I TB-9Sb-4L6222
421B TB-1La-4L4692
421C TB-7Lb-4L4698

423A TB-4Lb
 423B TB-4Lc
 423C TB-4Ld
 423D TB-4Le
 425A TB-4Sg
 425B TB-4Lh
 425C TB-4Li
 428I Di6; TB-4Sa
 522B TB-5Lb
 522D TB-5Ld
 528D TB-1La-5S8041
 614A TB-6Lb
 627E Di2; a1-m A2 C1 C2 R1 TB-6Lc
 720A Di3; a1-m1::rDt (Neuffer) TB-7Lb
 806A TB-8La
 806B TB-8Lb
 921A TB-9La
 921B TB-9Sb
 921C TB-9Lc
 922C C1-I; TB-9Sb
 929A IsoB9-9 isochromosome Type 1
 929B IsoB9-9 isochromosome Type 2
 929C T9-B(La); T9-B(Sb)
 929D IsoB9-9 isochromosome (original)
 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
 X21A TB-10La
 X21C TB-10Ld
 X22B T1La-B-10L18
 X22C TB-10Lb
 X30A TB-10L1
 X30B TB-10L2
 X30C TB-10L3
 X30D TB-10L4
 X30E TB-10L5
 X30F TB-10L6
 X30G TB-10L7
 X31A TB-10L8
 X31B TB-10L9
 X31C TB-10L10
 X31D TB-10L11
 X31E TB-10L12
 X31G TB-10L14
 X31H TB-10L15
 X31I TB-10L16
 X31J TB-10L17
 X32A TB-10L18
 X32C TB-10L20
 X32D TB-10L21
 X32E TB-10L22
 X32F TB-10L23
 X32G TB-10L24
 X32H TB-10L25
 X32I TB-10L26
 X32J TB-10L27
 X32K TB-10L28
 X33A TB-10L29
 X33B TB-10L30
 X33C TB-10L31
 X33D TB-10L32
 X33E TB-10L33
 X33F TB-10L34
 X33G TB-10L35
 X33H TB-10L36
 X34A TB-10L37
 X34B TB-10L38

INVERSION

I143B Inv1c (1S.30-1L.01)
 I143C Inv1d (1L.55-1L.92)
 I143D Inv1k (1L.46-1L.82)
 I243A Inv2b (2S.06-2L.05)
 I243B Inv2h (2L.13-2L.51)
 I343A Inv3a (3L.38-3L.95)

I343B Inv3b (3L.21-3L.70)
 I343C Inv3c (3L.05-3L.95)
 I344A Inv9a (9S.69-9L.90)
 I443A Inv4b (4S.10-4L.12)
 I443B Inv4c (4S.89-4L.62)
 I444A Inv2a (2S.69-2L.80)
 I543A Inv4e (4L.16-4L.81)
 I743A Inv5(8623) (5S.67-5L.69)
 I743B Inv6d (6S.70-6L.33)
 I743C Inv6(3712) (6S.76-6L.63)
 I843A Inv6e (6S.80-6L.32)
 I943A Inv7f (7L.17-7L.61)
 I943B Inv7(8540) (7L.12-7L.92)
 I943C Inv7(3717) (7S.32-7L.30)
 IX43A Inv8a (8S.30-8L.15)
 IX43B Inv9b (9S.05-9L.87)

RECIPROCAL TRANSLOCATION
 {wx1 and Wx1 marked}

wx01A T1-9c (1S.47; 9L.22); wx1
 wx01B T1-9(5622) (1L.10; 9L.12); wx1
 wx02A T1-9(4995) (1L.19; 9S.20); wx1
 wx02AA T1-9(4995) (1L.19; 9S.20); wx1
 wx03A T1-9(8389) (1L.74; 9L.13); wx1
 wx04A T2-9c (2S.48; 9S.33); wx1
 wx05A T2-9b (2S.17; 9L.22); wx1
 wx06A T2-9d (2L.83; 9L.270); wx1
 wx07A T3-9(8447) (3S.44; 9L.14); wx1
 wx08A T3-9c (3L.09; 9L.12); wx1
 wx09A T3-9(8562) (3L.65; 9L.22); wx1
 wx10A T4-9e (4S.53; 9L.26); wx1
 wx11A T4-9g (4S.27; 9L.27); wx1
 wx12A T4-9(5657) (4L.33; 9S.25); wx1
 wx13A T4-9b (4L.90; 9L.29); wx1
 wx14B T5-9(022-11) (5S.29; 9L.27); wx1
 wx15A T5-9(4817) (5L.06; 9S.07); wx1
 wx16A T5-9d (5L.14; 9L.10); wx1
 wx17A T5-9a (5L.69; 9S.17); wx1
 wx18A T6-9(4778) (6S.80; 9L.30); wx1
 wx19A T6-9a (6S.79; 9L.40); wx1
 wx20A T6-9b (6L.10; 9S.37); wx1 y1
 wx21A T6-9(4505) (6L.13; 9ctr); wx1
 wx22A T7-9(4363) (7ctr; 9ctr); wx1
 wx23A T7-9a (7L.63; 9S.07); wx1
 wx24A T8-9d (8L.09; 9S.16); wx1
 wx25A T8-9(6673) (8L.35; 9S.31); wx1
 wx26B T9-10(059-10) (10L.53; 9S.31); wx1
 wx28A T5-9(8386) (5L.87; 9S.13); wx1
 Wx30A T1-9c (1S.47; 9L.22); Wx1
 Wx30B T1-9(4995) (1L.19; 9S.20); Wx1
 Wx30C T1-9(8389) (1L.749; 9L.13); Wx1
 Wx31A T2-9c (2S.48; 9S.33); Wx1
 Wx31B T2-9b (2S.17; 9L.22); Wx1
 Wx32A T3-9(8447) (3S.44; 9L.14); Wx1
 Wx32B T3-9(8562) (3L.65; 9L.22); Wx1
 Wx32C T3-9c (3L.09; 9L.12); Wx1
 Wx33A T4-9e (4S.53; 9L.26); Wx1
 Wx33B T4-9(5657) (4L.33; 9S.25); Wx1
 Wx33C T4-9g (4S.27; 9L.27); Wx1
 Wx34B T5-9(4817) (5L.06; 9S.07); Wx1
 Wx34C T4-9b (4L.90; 9L.29); Wx1
 Wx35A T5-9(8386) (5L.87; 9S.13);

Wx1
 Wx35B T5-9a (5L.69; 9S.17); Wx1
 Wx35C T5-9d (5L.14; 9L.10); Wx1
 Wx36A T6-9(4778) (6S.80; 9L.30); Wx1
 Wx37A T6-9(8768) (6L.89; 9S.60); Wx1
 Wx37B T7-9(4363) (7ctr; 9ctr); Wx1
 Wx37C T6-9(4505) (6L.13; 9ctr); Wx1
 Wx38A T7-9a (7L.63; 9S.07); Wx1
 Wx38B T8-9d (8L.09; 9S.16); Wx1
 Wx38C T8-9(6673) (8L.35; 9S.31); Wx1
 Wx39A T9-10(8630) (9S.28; 10L.37); Wx1
 Wx39B T9-10b (10S.40; 9S.13); Wx1

V. MAIZE GENOME DATABASE
<http://www.agron.missouri.edu/>

MaizeDB currently presents 15,515 loci, including quantitative trait loci and gene candidates; 14,460 references; 4,071 colleague addresses; 489 traits; 2,789 genetic stocks; 5,449 elite germplasm pedigrees; 21,399 variations with 3803 images, and 771 phenotypes; 1055 (putative) gene products; 62,783 links to 41 external databases from 13,582 records; GRIN, SwissProt and GenBank (Entrez format) link reciprocally to MaizeDB pages. The WWW page 'What's New' provides an historical listing of data and design changes, with hypertext links to database records. Since Jan 1995 there have been 2,913,414 accesses, with 202,824 in Mar 1997, more than double last year. A more detailed usage analysis is accessible from the database homepage.

The Maize Genetics Newsletter (MNL), years 1992-current, is newly on-line, linked to MaizeDB authors and other objects. Links to new external databases were made: MedLine (1921 links), EcoCyc(298), AGIS-EC#(297). Medline provides access to published abstracts, related articles and GenBank records; EcoCyc accesses *E. coli* metabolic pathway data and links to the Coli Genetic Stock Center; and AGIS-EC# accesses plant metabolic pathway and gene product data.

Of the loci with map locations, 1270 are genes, 4133 are probed sites, and 651 are QTL; 93 hybridize to clones from various Triticeae species; 126 to rice. Of the 1804 EST's or partially sequenced cDNA's in maize, 669 have been placed on the BNL or the UMC maps. Of the mapped sites using clones from other species, there are 93 marked by probes from the Triticeae and 181 with rice probes.

Accessing the data: WWW access modes include (a) 4 styles of precision query forms, including one that provides user-specified subsets of Mapmaker formatted data; (b) full text query; (c) browser access to special lists, computed regularly, which dynamically extract data from MaizeDB. These special lists include map-indexed lists of genes, probes, microsatellite primers, genetic stocks, mutant images; cooperators; current literature; new data. Most of the browsing lists are accessible from the 'Of Interest to Maize Cooperators' page.

In addition to various WWW routes, MaizeDB supports access by gopher, ACEDB and ad hoc SQL. There is guest Internet login by modem to all routes, including access by clients without gopher or WWW software on their computers.

The fastest way to become acquainted with how MaizeDB represents information in areas of your interest is to use the full-text query option, prior to utilizing the all-attributes precision query pages. Detailed help is available on-line. Please email us at db_request@teosinte.agron.missouri.edu, for help, suggestions, and anything related.

Nomenclature Issues

Following is a proposed criterion key for designating and naming cDNAs and their genes. Please respond to MaizeDB, at db_request@teosinte.agron.missouri.edu, with any comments about the proposal.

Principle: Whenever appropriate, seek a mnemonic in Mendel, check MaizeDB for prior usage and for the same map location or potential equivalence, assign a new mnemonic (e.g., *mnm1*) if justified, and request MaizeDB (db_request@teosinte.agron.missouri.edu) to reserve the mnemonic. Mendel may be accessed from the MaizeDB home page, under 'Of Interest to Maize Cooperators', and then the Commission for Plant Gene Nomenclature (CPGN) (<http://jii06.jic.bbrsc.ac.uk>).

For a sequenced cDNA, synopsis of the nomenclature syntax:

[*mnemonic*, gene or institution][*integer*][*letter*, if multiple site][*mnemonic or gene family number* in parentheses]

Before Mapping, use the anonymous acronym of the institution or laboratory with a sequential number, e.g. *int###*.

After Mapping,

Single Copy: If probability/score for the sequence indicates this is a cDNA from a new gene with defined function, assign a new mnemonic (e.g., *mnm1*), and request MaizeDB to reserve it and to maintain the *int###* as a synonym. If this is a cDNA homologous to a named gene mapping at a different location, designate a new, homologous gene (e.g., *mnm2*).

Multiple Copy, with independent evidence indicating that the map site is uniquely the site for the cDNA: Assign a mnemonic. For other mapped sites, use *int###b(mnm)*, etc.

Multiple Copy, with no independent evidence of uniqueness, append a mnemonic in parentheses -- e.g. *int###(mnm)* -- thus, *csu123(abc)*. In the absence of a mnemonic, include the Mendel Family number, in (). These numbers will soon be made available in the public Mendel database.

Mary Polacco

VI. MAIZE PROBE BANK

DNA clones in the public bank and distribution center at the UMC Maize RFLP Lab now total ~ 6700, of which we distribute more than 4900 (Table 1). This includes the large sets of sequenced cDNAs generated by Chris Baysdorfer, California State University - Hayward (designated csu), and by Tim Helentjaris (designated uaz or 5C, 6C, 7C), which represent the largest collection of candidate genes publicly available for maize. Over the last five years the UMC Maize Probe Bank has filled more than 820 requests for more than 24,000 clones to 31 countries, including 87 sets of the UMC Core Marker probes, which have become the standard landmark probe set for mapping genes, phenotypes and QTLs in maize.

The activities of the RFLP Lab and the Probe Bank are meaningful as they contribute to the research needs of the community. The easiest avenue to request clones is through the "PROBE Requests" form in MaizeDB, <http://www.agron.missouri.edu>, or by email to musket@teosinte.agron.missouri.edu.

To contribute clones for distribution or to be mapped onto the UMC Maize Genetic Map contact Theresa Musket at the email listed above and she will inform you of the information and materials needed.

These activities are supported by USDA-ARS CRIS projects "Genetic Mechanisms and Molecular Genetic Resources for Corn" and "Maize Genome Database"; and by FAO/IAEA Coordinated Research Program, "Molecular Markers for Maize, Rye and Rice".

Table 1. Clones maintained by UMC RFLP Lab and Probe Bank.

Clone Set	Abbreviation	Type of Clone	Total Number in Set	Distribution by UMC
Asgrow	asg	maize genomic	85	Yes
Brookhaven National Laboratories	bnl	maize genomic	109	Yes
Brookhaven National Laboratories	bnl	maize cDNA	12	Yes
California State University-Hayward	csu	maize cDNA	1197	Yes
Contributed Clones		mostly maize	295	90 Yes, 205 No
Cornell University	bcd, cdo, rg, rz	barley, oat, rice	151	No
Iowa State University	isu	maize cDNA	136	Yes
LIMAGRAIN	lim	maize genomic	113	Yes
Mycogen Plant Sciences	agr	maize cDNA	413	Yes
Northrup King	npi	maize	30	No
Pioneer Hi-Bred International	php	maize genomic	161	Yes
Pioneer Hi-Bred International	npi	maize both	236	Yes
Rice Genome Research Program	rgp	rice	371	No
Texas A&M University	txs	sorghum genomic	145	No
University of Arizona	uaz 5C, 6C, 7C	maize cDNA	1920	Yes
University of Hohenheim	Ch6S	maize genomic	339	No
University of Minnesota	umn	oat	29	No
University of Missouri	umc	maize both	249	237 Yes, 12 No
University of Missouri-Rice	pOs	rice genomic	18	Yes
University of Missouri-Rye	ScG	rye genomic	171	Yes
University of Missouri-Tripsacum	tda	tripsacum genomic	476	20 Yes, 456 No
Total Number of Clones:			6694	
Total Distributable Clones:			4906	

Theresa Musket, Mary Polacco, Mike McMullen, and Ed Coe

TWO BULLETINS ABOUT THE DOG

I.

Following note was received from a Fish person:

the dog's name was found on a
fishing trip, underwent
horizontal transfer, can
be found hob-knobbing
at a new location.

(the reward is a hank of clothesline, toward future fishing for larger games and toward keeping the dog running).

II.

Received documentation from Day that calls for reopening play; investigations are underway, dog's name is subject to re-assay.

VII. UMC 1998 MOLECULAR MARKER MAP OF MAIZE ESTs, Sequenced Core Markers, and NonMaize Probes

We present here a map containing 1736 loci, as a resource to further efforts for gene discovery and for understanding of developmental and metabolic processes necessary to advance fully the potentials of maize. This map is densely populated with loci probed by sequenced clones, especially with ESTs.

Our immortalized F2 maize population of 54 individuals, derived from Tx303 x CO159 (Gardiner et al., *Genetics* 134:917-930, 1993) was used. The average marker density for the map is one marker per centimorgan. The loci consist of 1121 (65%) probed by cDNAs, 546 (31%) probed by genomics, and the remainder isozymes and simple sequence repeats. Sequence information is available for the probes for 56% of the distinct loci. Of the distinct loci, 38% are associated with probes that can be assigned putative functions. Eighty percent of the homologies are with genes from plants, with *Zea mays*, *Arabidopsis thaliana*, and *Oryza sativa* having the highest number of matches. The homologies represent a diversity of biological processes, including signal transduction, cell cycle regulation, carbon metabolism, floral and leaf development, stress response, and fatty acid metabolism, among others.

Ninety core markers were chosen for low copy number, high polymorphism rate, and even spacing throughout the genome; improved probes relative to those in Gardiner et al. (1993) were selected when appropriate. Sequence data are available for 84 of the core markers. These markers are the boundaries for the maize bins, which are used to organize data from different mapping populations and types of experiments including those involving cytogenetic stocks, mutants, or quantitative trait loci on a common framework known as the bin map. Currently, the bin map contains 5,179 loci including those presented on this map. Core marker sets are available through the probe request hotlink in the Maize Genome Database (<http://www.agron.missouri.edu>).

The map contains 221 loci probed by barley, oat, wheat, sorghum, tripsacum, or rice clones. These loci provide points of alignment with map data from other grass species, particularly rice, which has extensive map and sequence information available. The map, scores, sequence, and probe information are currently available through the Maize Genome Database. This information is updated as new homologies and map information become available.

The additional expressed sequence tagged sites mapped here, together with the core markers, and the loci mapped by nonmaize probes, are a tool and resource for research and gene discovery in maize and other cereal grains.

Methodology

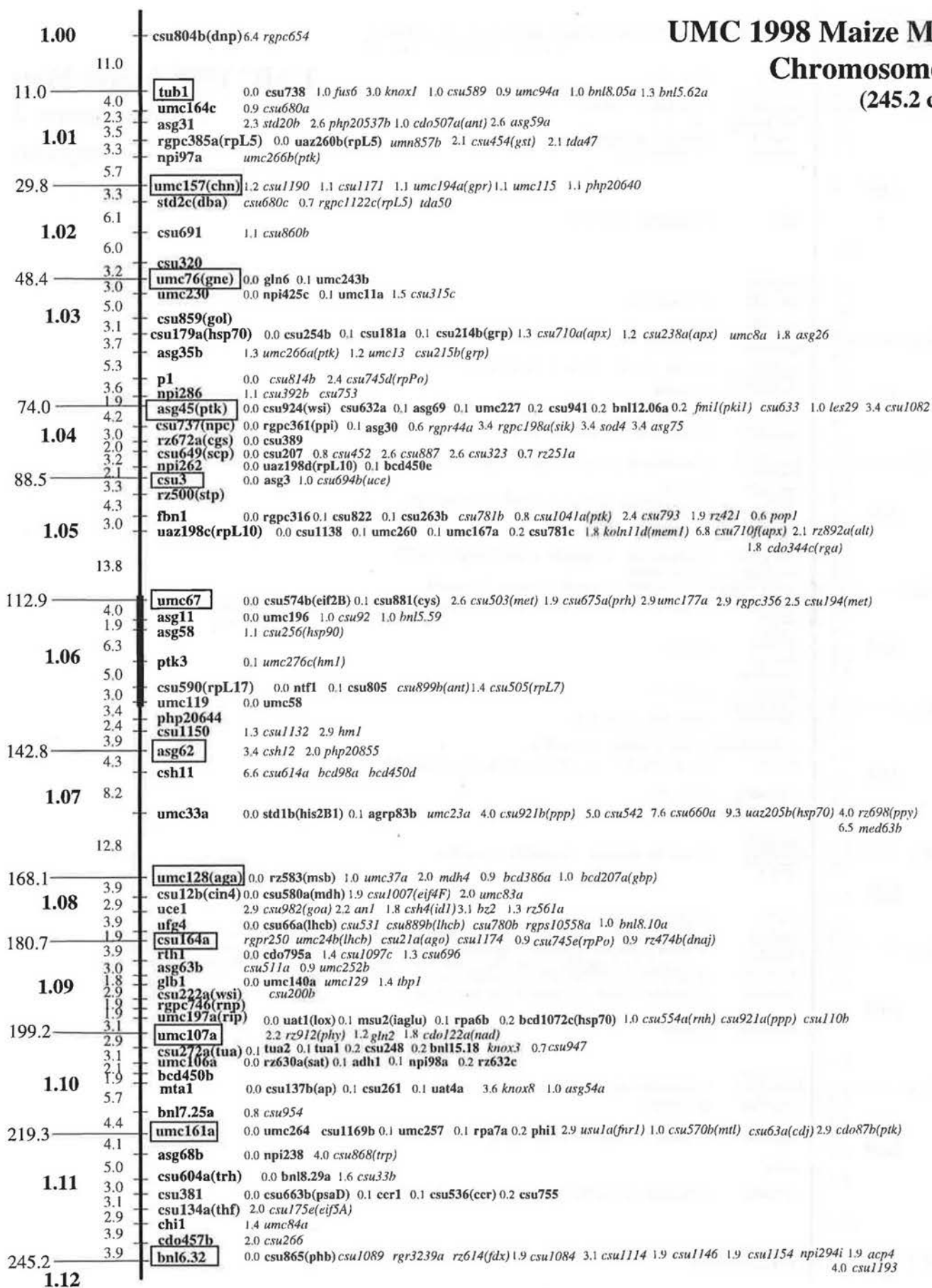
DNA preparation, Southern hybridizations, sequencing, and autoradiography were conducted according to standard laboratory procedures. SSR protocol is a modification of the method of Lynn Senior. Final concentrations of reaction components are as follows: Perkin Elmer Buffer 1X; MgCl₂ 2.5 mM; dATP, dCTP, dGTP, dTTP 0.1 mM each, SSR primers-forward and reverse 50 ng each (Research Genetics Inc, Huntsville, AL); AmpliTaq Gold™ polymerase 0.3 units (Perkin Elmer Corporation, Norwalk, CT); genomic DNA 50 ng; sterile water to a total volume of 15 ul. All thermocycling was performed in a 96-well thin-walled microtiter style plate (Costar 6509) with an oil overlay in an Amplitron II™ thermocycler (Barnstead/ThermoLyne Corporation, Dubuque, IA). The cycling profile included a preliminary eight to ten minute dwell at 95C to activate the polymerase. This was followed by two cycles of 1 min at 95C, 1 minute at 65C, and 90 seconds at 72C. Subsequently, single cycles of a one degree decrement for the annealing temperature until an annealing temperature of 55C was achieved. The final phase of amplification included twenty-nine additional cycles at the 55C annealing temperature. Following amplification, PCR products were resolved in a 3.5% small fragment resolution (SFR) agarose (Amresco Inc., Solon, OH), 1X TBE gel containing approximately 2 ug/ml ethidium bromide.

Map construction -- Chromosomes were constructed using MAPMAKER for UNIX, Version 3 on a Sun SPARC Server 1000. The 10 maize chromosomes were defined with the "make chromosomes" function and the 90 core markers were anchored to chromosomes. Initial framework orders were assigned for the core markers for each chromosome. The remaining markers were attached to linkage groups with the "assign" command. Additional markers were added to the framework, first at LOD3 then at LOD2, 10-15 markers at a time with the "build" command. Remaining markers assigned to each linkage group were positioned relative to the framework loci with the "place" command. A few of the placed markers were then added to the framework using the "together" command and the remaining markers positioned against the new framework with the "place" command. Marker loci with more than three double cross-overs based on the "genotype" function were deleted. Chromosome maps were drawn to postscript files, exported, translated to Macintosh format and edited with Adobe Illustrator 6.0.1.

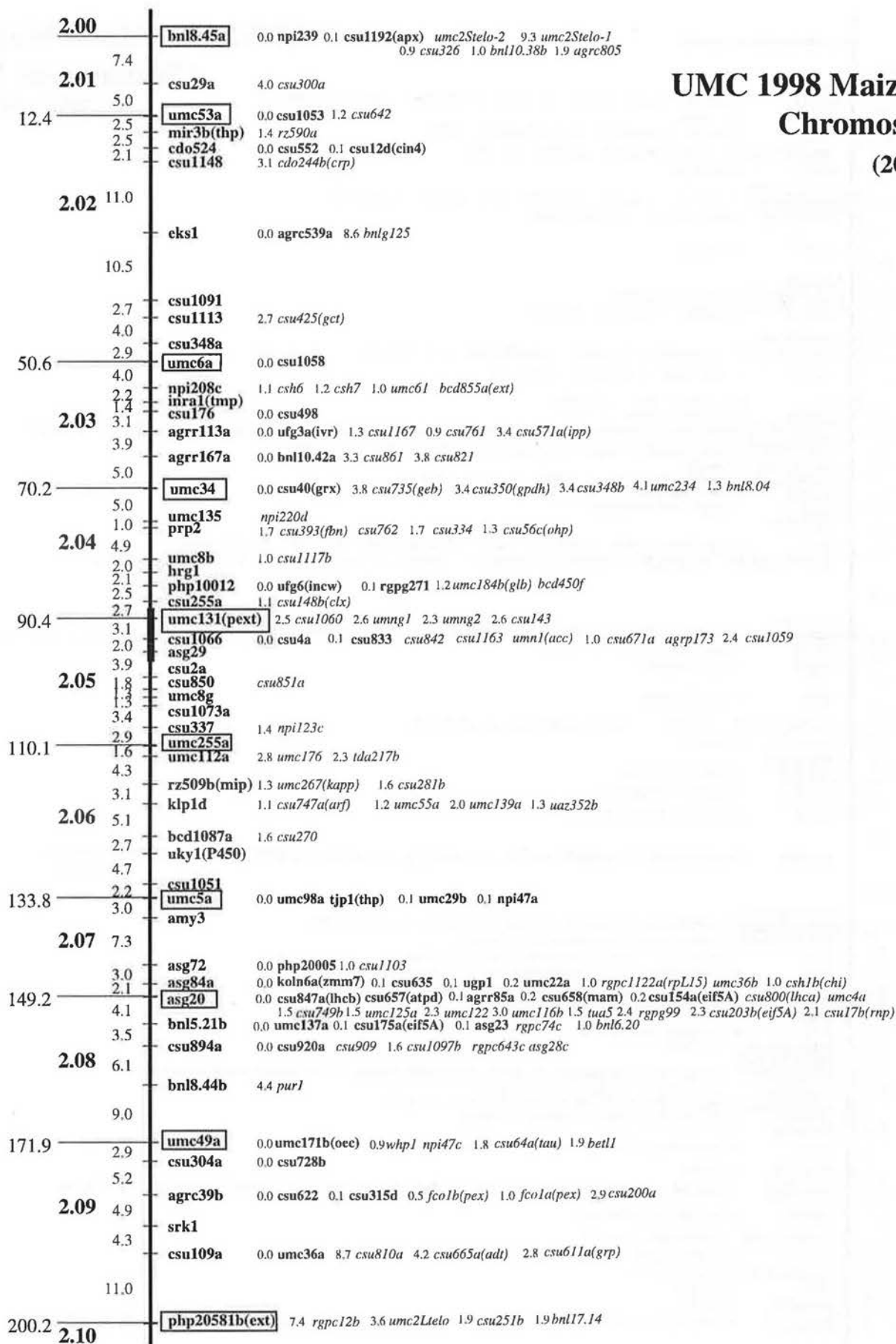
Homologies—Individual investigators provided homology information when submitting each cDNA or genomic clone for mapping. All non-core clones from genomic or cDNA library mapping were analyzed for homology using the NCBI blast server or the dbEST neighbors algorithm (Altschul et al., 1990, www.ncbi.nlm.nih.gov/BLAST; Boguski et al., 1993, www.ncbi.nlm.nih.gov/irx/dbST/dbest_query.html; www.ncbi.nlm.nih.gov/Entrez/entrezhelp.html#Special). Identity was declared at values greater than 10⁻⁸ for BLASTX against dbEST, and 10⁻¹⁰ of BLASTX against the nonredundant (nr) nucleotide or peptide database or using the neighbors algorithm. Mnemonics were derived for the associated loci according to the maize nomenclature guidelines. Core marker sequences were analyzed using the email version of BLAST 1.4.11 (Altschul et al., 1990, blast@ncbi.nlm.nih.gov). Homology searches against the NCBI nr and dbEST databases were made using, respectively, the BLASTX and TBLASTX algorithms. Matches were declared at P(N) < 0.01. In some cases, although a gene name is given, it was clear that the reported match is only to a motif, not to the entire gene.

G. Davis, M. McMullen, M. Polacco, D. Grant, T. Musket, C. Baysdorfer, M. Staebell, G. Xu, L. Koster, K. Houchins, S. Melia-Hancock, and E. H. Coe

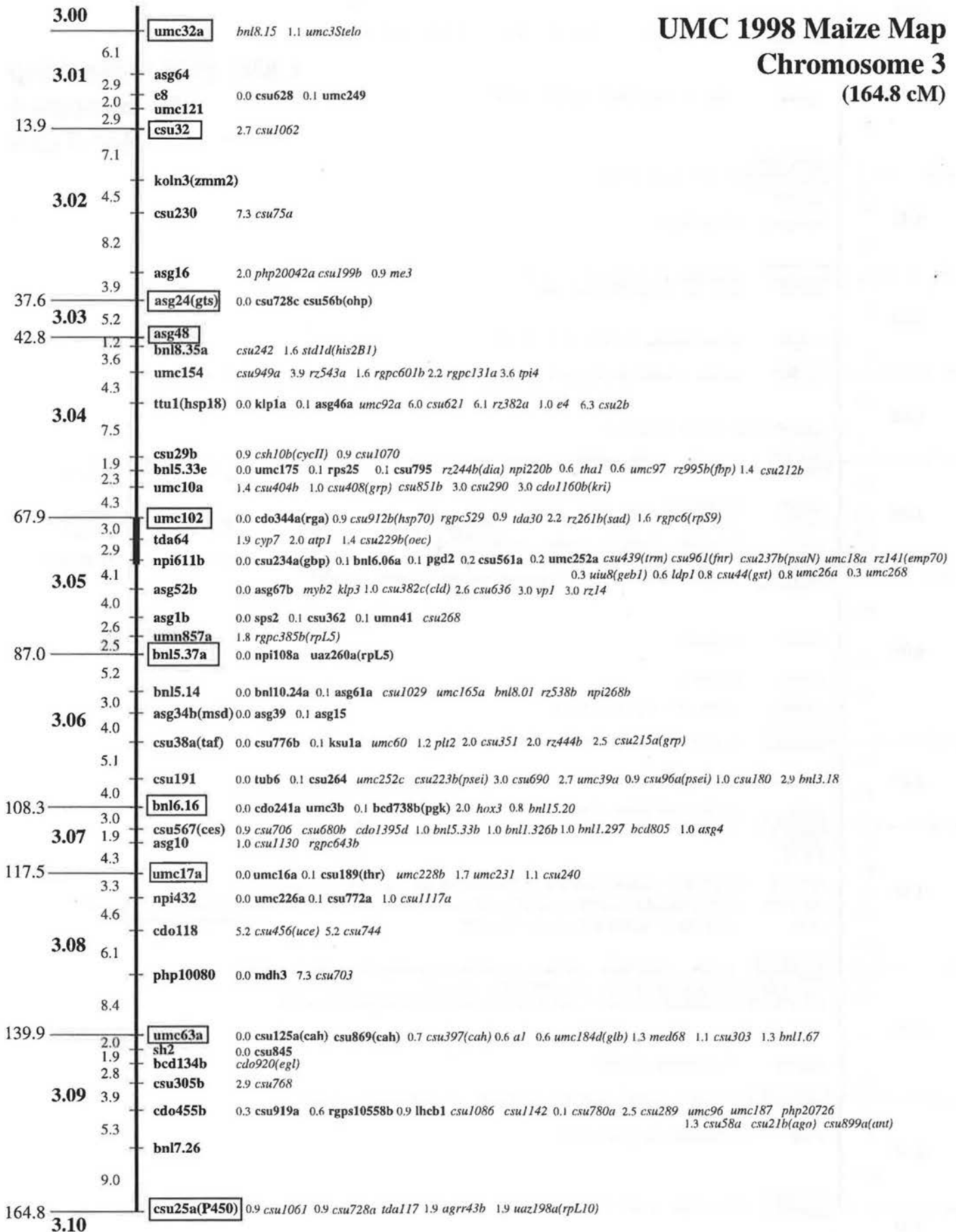
UMC 1998 Maize Map Chromosome 1 (245.2 cM)



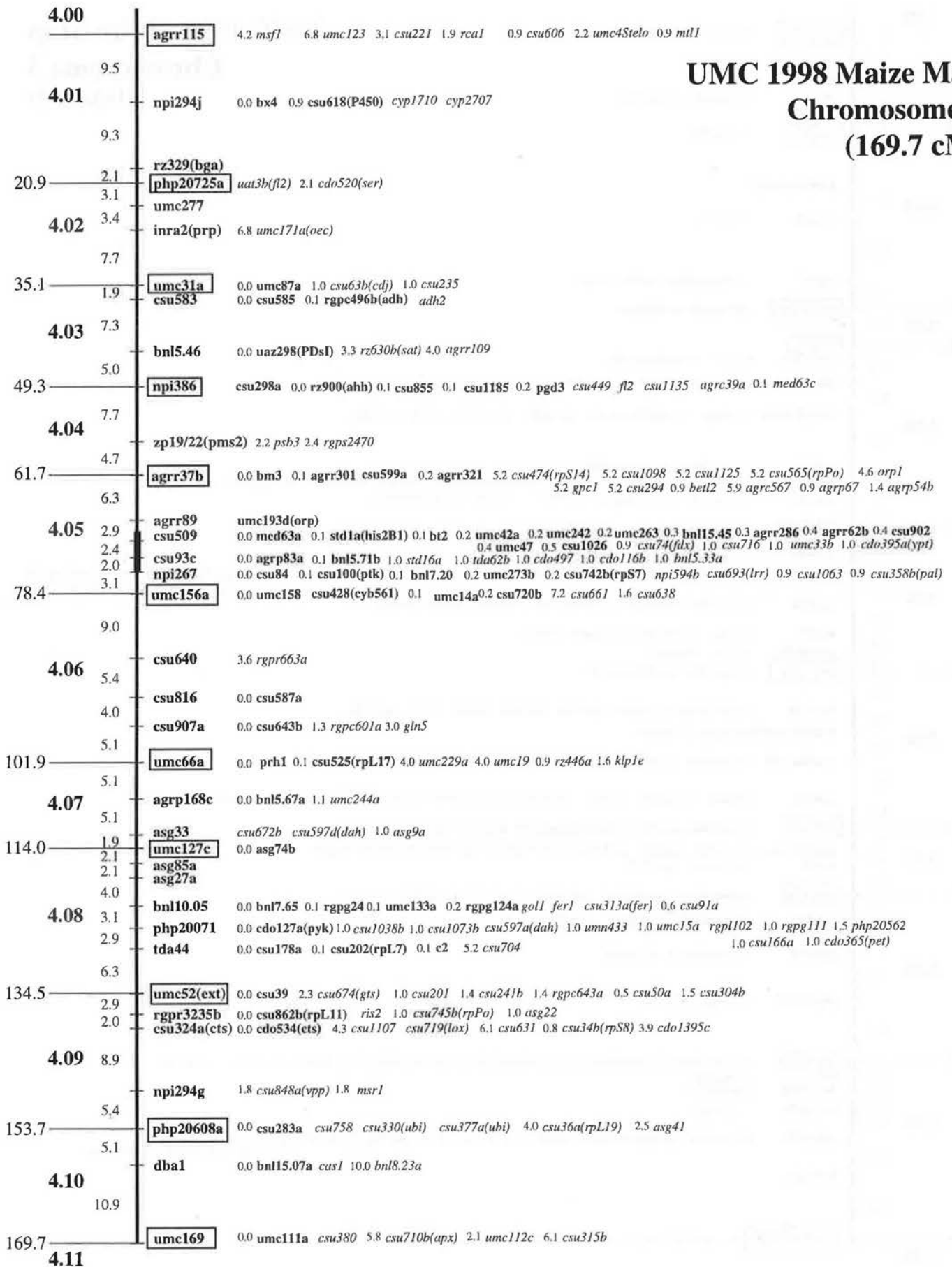
UMC 1998 Maize Map Chromosome 2 (200.2 cM)



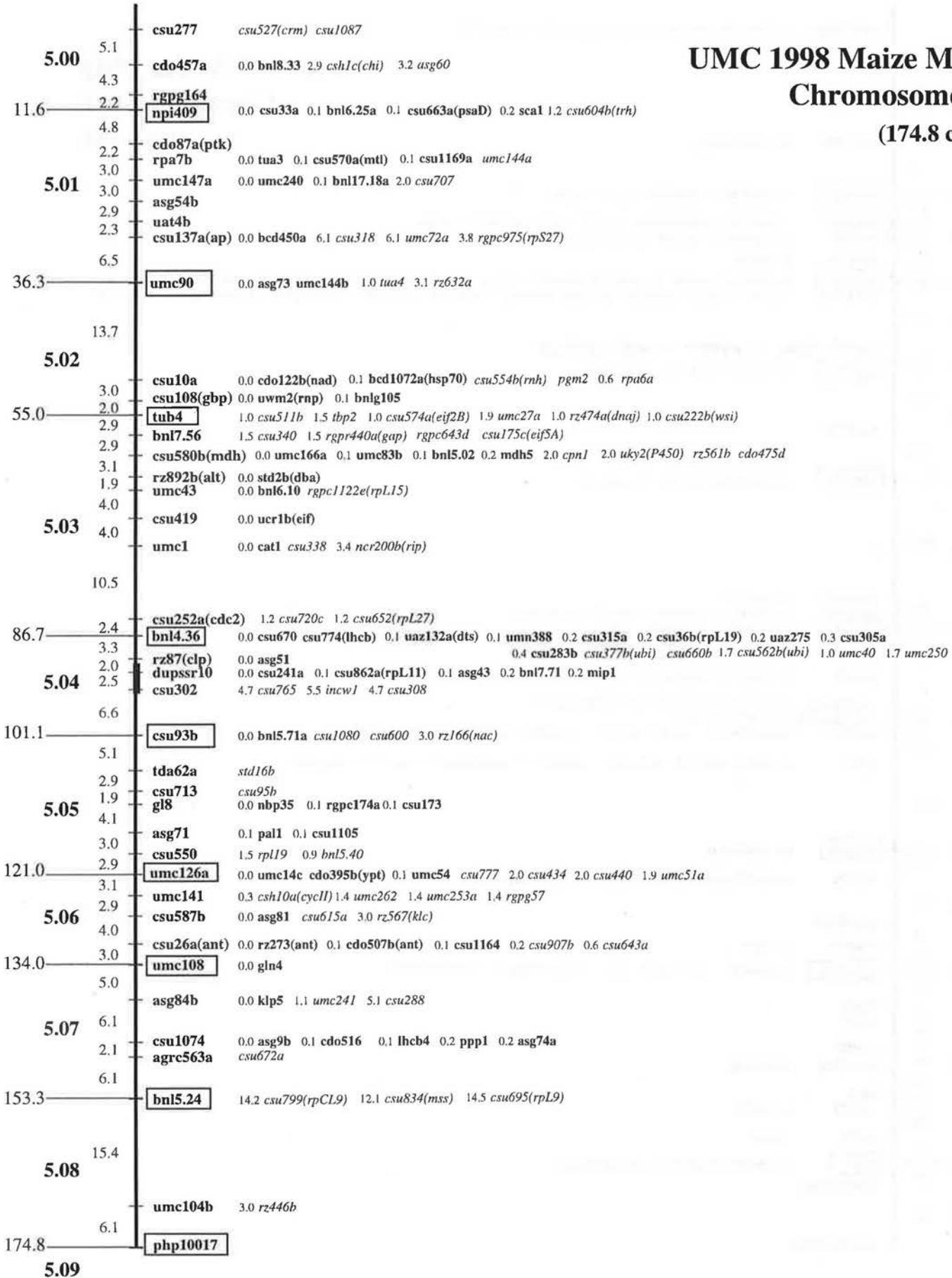
UMC 1998 Maize Map Chromosome 3 (164.8 cM)



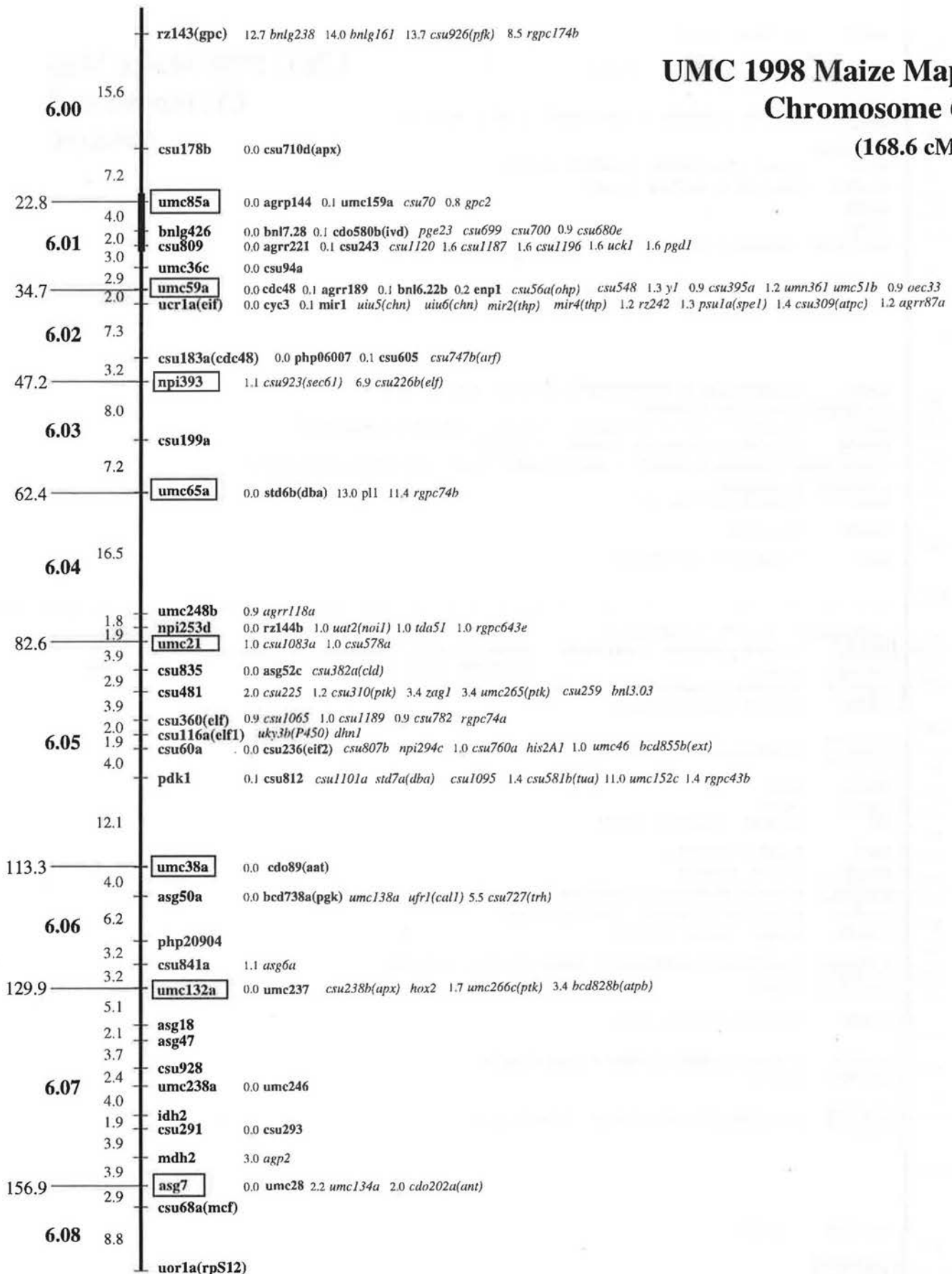
UMC 1998 Maize Map Chromosome 4 (169.7 cM)



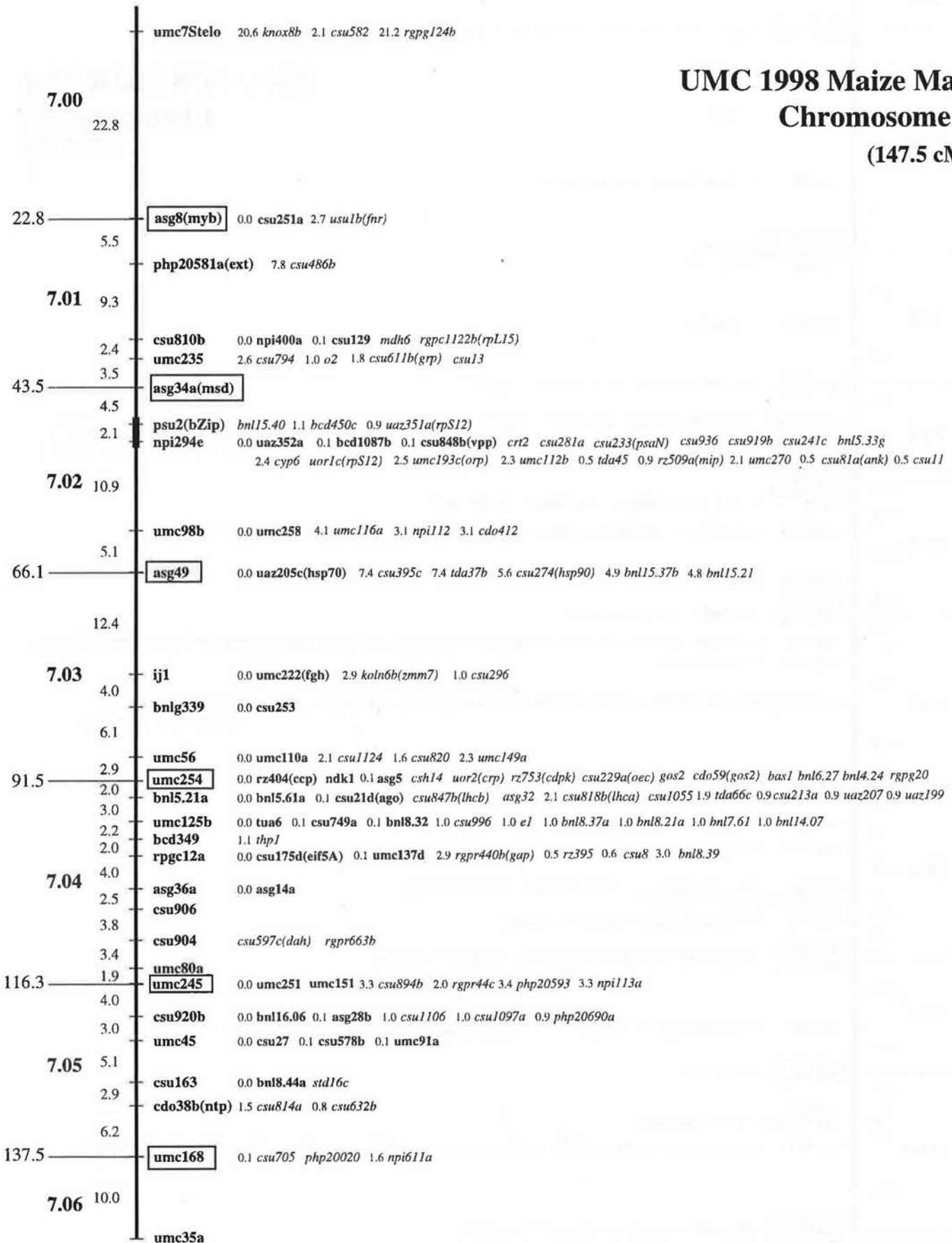
UMC 1998 Maize Map Chromosome 5 (174.8 cM)



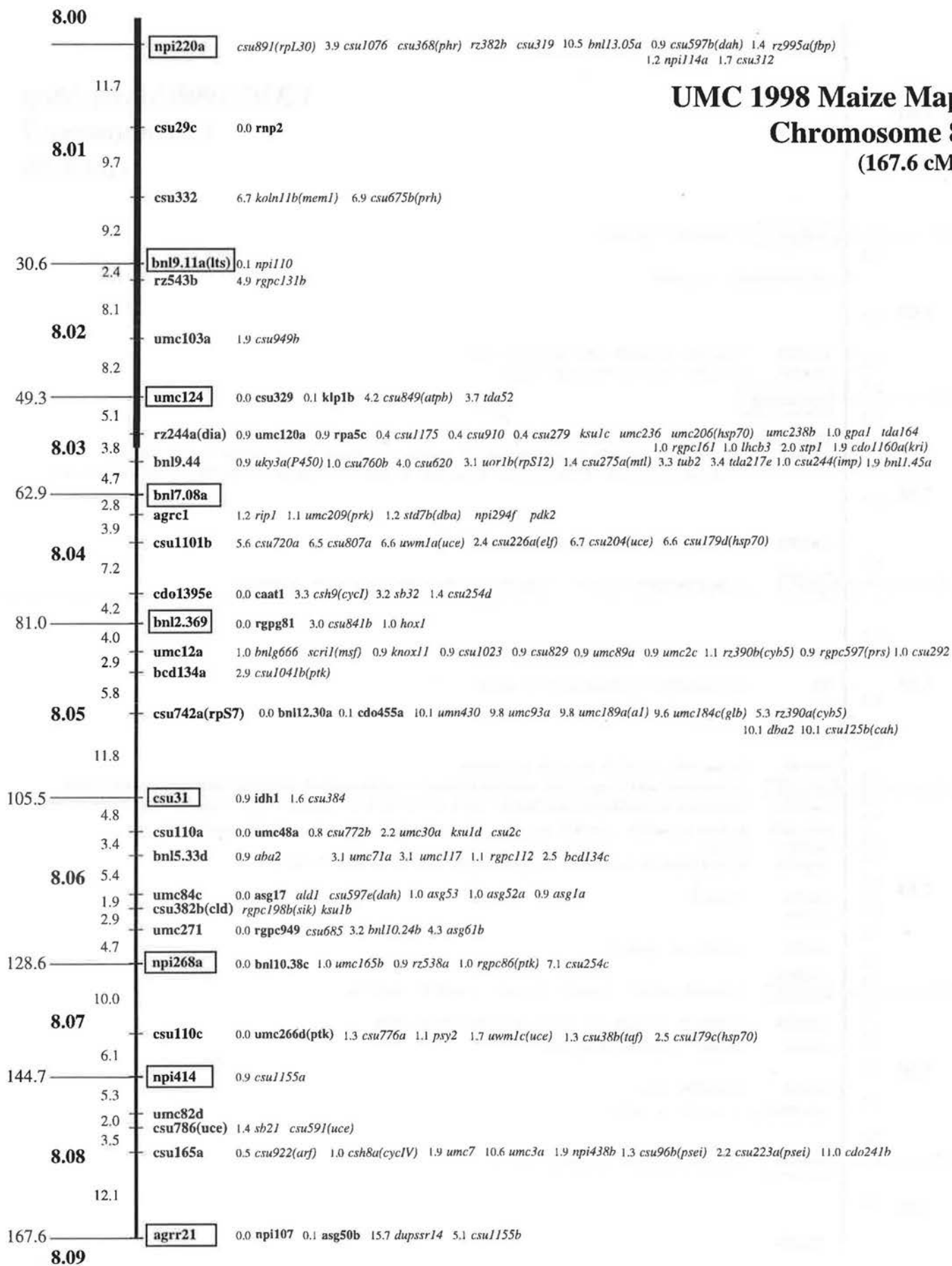
UMC 1998 Maize Map Chromosome 6 (168.6 cM)



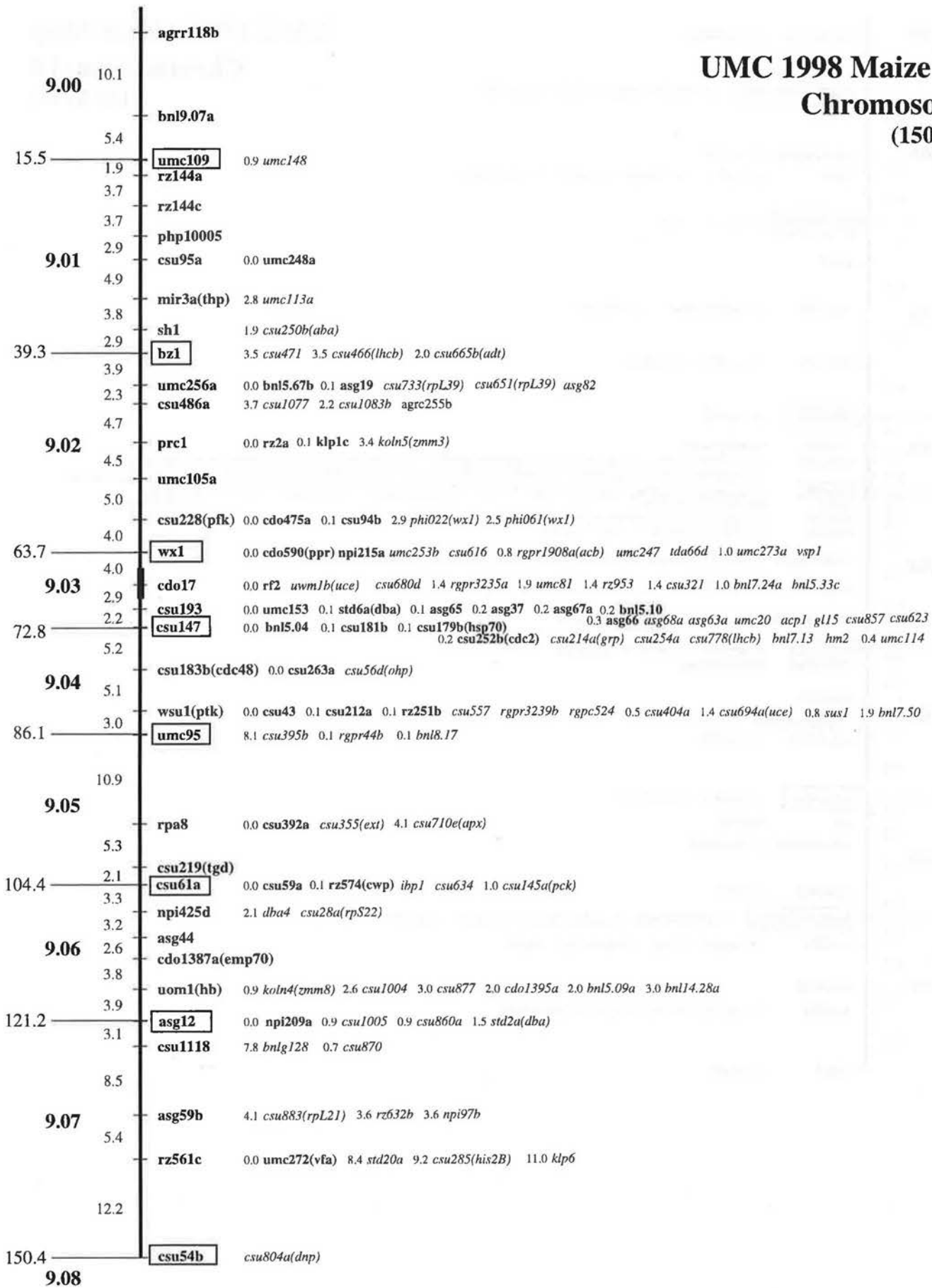
UMC 1998 Maize Map Chromosome 7 (147.5 cM)



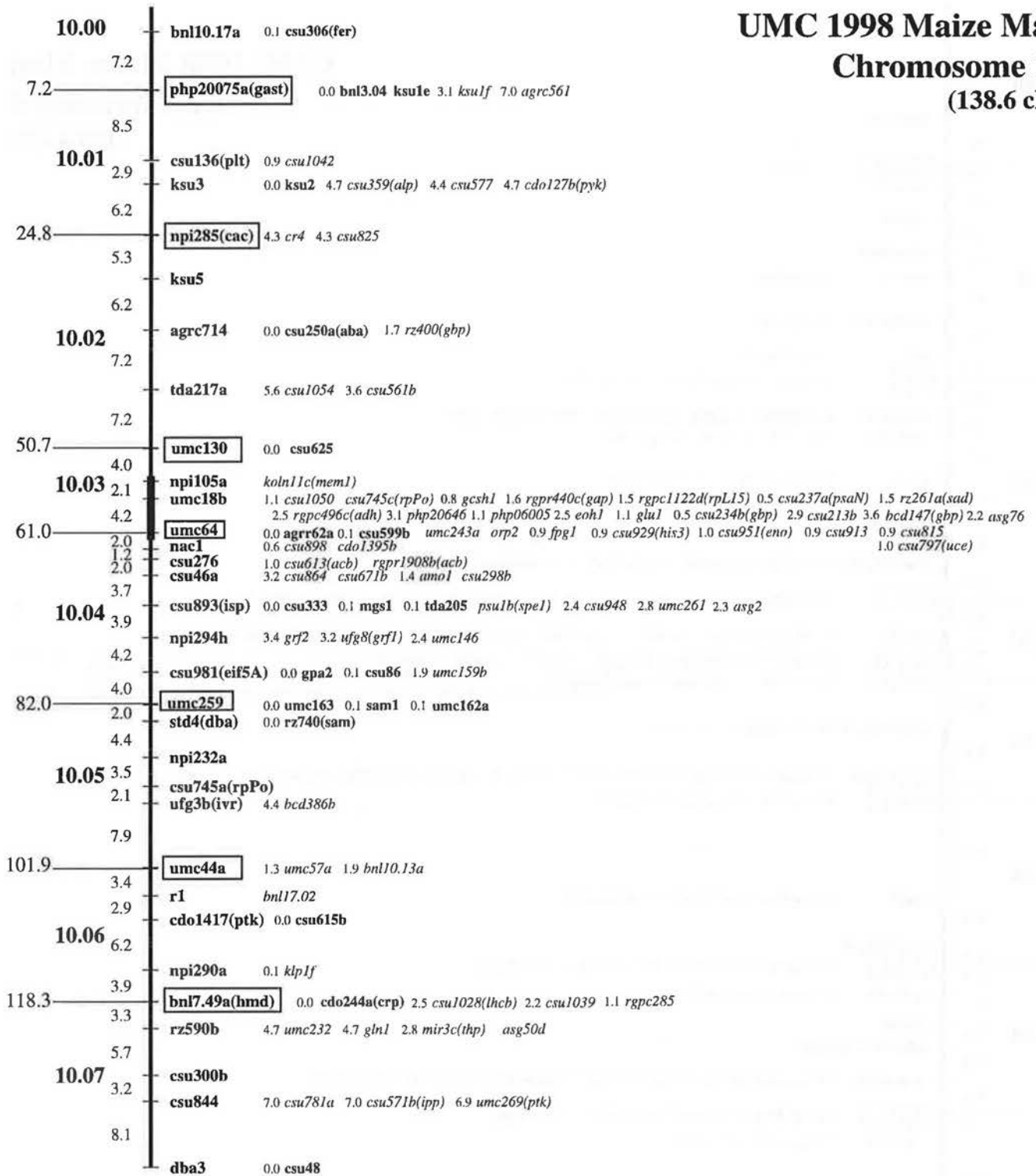
UMC 1998 Maize Map Chromosome 8 (167.6 cM)



UMC 1998 Maize Map Chromosome 9 (150.4 cM)



UMC 1998 Maize Map Chromosome 10 (138.6 cM)



VIII. MAIZE GENETICS COOPERATION - 1929

NEW YORK STATE COLLEGE OF AGRICULTURE
CORNELL UNIVERSITY
ITHACA, N. Y.

DEPARTMENT OF PLANT BREEDING

Nov. 23, 1929

Prof. R. A. Brink,
Madison, Wis.

Dear Sir :

In discussing the proposed revision of the mimeographed summary of linkage in maize, Professor Emerson suggested that I look after assembling the unpublished data that students of maize genetics might be willing to contribute. In a letter sent out with the summary prepared last winter, Professor Emerson wrote as follows:

"I hope that each of you, whether or not you attended the New York meeting, will send me such relevant data as you have not yet published, showing either linkage or independent inheritance. In so far as you have data ready for publication, I prefer to receive a copy of your manuscript, but shall be glad to have also records which you are not ready to publish, if you care to send them. I agree not to publish any such data without your consent and in any case to give proper credit. Any records sent, however, should be with the understanding that I am at liberty to use them in an early revision of the mimeographed sheets for distribution to other workers, pending the publication of the general linkage paper which I have been threatening to bring out for some years now.

I indicated at New York that the records were too incomplete to warrant publication now, a fact made strikingly obvious by the rainbows on the maps. The distribution of the data in mimeographed form should serve temporarily the needs of those actively studying maize genetics; and others can wait. The co-ordination of effort agreed to in New York should go far toward straightening out many of the question marks in the next year or two."

We should very much like to have these data available in the next few months, say by February 1 at the latest.

We are planning to make several minor improvements in the summary. We feel that it would be of considerable help to maize geneticists to have available a

summary of the known genetic factors in maize including those of which there is no published account. The advantages of such a summary list are obvious. We are therefore asking whether you would be willing to send us a list of the mendelian factors on which you have not published, giving the following information:

1. Name and symbol of the factor.
2. Whether dominant or recessive.
3. A very brief description of the character including any known interaction with other factors.
4. Linkage relations if known or suspected.

Suggestions as to how the summary might be made more useful to corn geneticists will be greatly appreciated. We shall also appreciate your soliciting the cooperation of graduate students and others working on corn of whom we may not know.

Sincerely,

G. W. Beadle

G. W. Beadle

GWB:G

all Var.

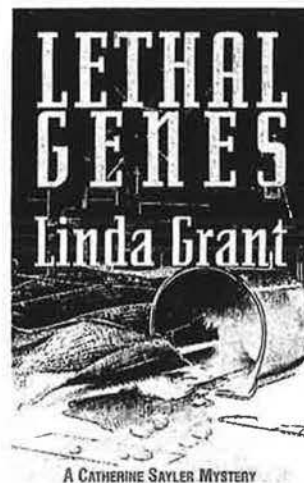
IX. SYMBOL INDEX

a1 70 74 80 81	bz1 70	Fas 25	lg2 62	MuDSR 7	po1 28 29 63
a1-m(En) 45	bz2 70	fas1 2	lg3 62	Mz6L-Tr16L 84	ppg1 79
a1-ruq(Uq) 45	c1 70	fdl1 58	lg4 64	na1 62	ps1 63
a2 70 80 81	c1-m(Ac) 45	fl1 16	li1 65	na2 63	pt1 63
abp1 62	cat3 86	fl3 16	lls1 61	nec1 64	ptd1 61
abph1 61	cdc2A 62	Flt 25	lo2 65	nec2 61	ptd2 64
abs*-7065 58	cdc2C 65	g1 46 78 81	lrt1 30	nec3 63	py1 63
abs*-8075 58	cdc48 63	ga1 62	Lsc 25	nec4 61	py2 61
abs*-8077 59	cg1 42 62	ga2 63	lte1 25	nec5 62	r1 70 74 78 81
Ac 7 45 58 70	cg2 42 45 62	ga7 62	ltp 25	nec6 63	R1-nj:Cudu 75
ad1 61	ck2 61	ga8 65	lw2 79	nec7 63	r1-scm3 58
adh1 86	cms-21A 10 11 13	gl2 61	lxm1 62	nl1 65	r1-X1 78
al*-143 45	cms-C 10 11 13 75	gl7 79	mac1 65	nl2 63	ra-D 82
al1 61	cms-S 10 11 13 41 75	gl11 61	mdh1 86	NOR 56	ra1 64
am1 63	cms-T 10 11 13 75	gl15 65	mdh2 86	npi234 72	ra2 40 62
an1 61	cp2 62	gl17 63	mdh3 86	npi598 66	rab15 63
ai as mrh(Mrh) 45	cr1 62	gl19 62	mdh5 86	ns1 62	rab28 63
as1 61	cr4 65	glu1 24 86	mgs1 65	ns2 62	rab30 61
asg45 76	csu3 76	glu2 24	mgs2 62	nuc1 3	rd1 61
asg62 76	csu4 71	gn1 61	mgs3 65	o1 16	rd3 62
asr1 62	csu6 71	got1 86	mmm1 86	o2 40	rea1 62
B-chr 52 53	csu13 71	got2 86	mpl1 61	o2-m(r)+Bg 16	rg1 62
b1 25 74	csu29 71	got3 86	Mrh 45 70	o5 17	rgd1 63
b32 17	csu46 71	grt1 79	mr1 67	o9 16	rgo1-R 22
ba1 62	csu54b 76	hs1 64	ms*-6006 39	o11 16	ri1 62
ba2 61	csu61 71	hsf1 63 66	ms*-BS1 38	obf2 65	rip1 17
baf1 65	csu86 71	hsp18 56 85	ms*-BS2 38	obf3a 62	rlc1 65
bar 34	csu94 71	id1 61	ms*-BS3 38	obf6 62	rlt1 2 65
bd1 64	csu109 71	ifa1 3	ms*-CG89D 38	OPA08 9	rp3 17
bf1 65	csu133 71	ig1 62	ms*-JH84A 37	OPA10 9	rs1 64
Bf1-Mu 4	csu148 71	ij1 64	ms*-WL87A 38	OPC06 9	rs2 61 66
Bf1-Mu-044-3 4	ct1 64	ij2 61	ms1 37 38 63	OPC07 9	rs2*-90 66
Bf1-Mu-544-3 4	ct2 61	inc1 62	ms2 29 38 39 65	OPE12 9	rs4 64
Bf1-R 3	ctl1 64	j*-220 45	ms3 37 62	OPF05 9	rt1 62
bf2 65 81	d1 62	j1 42	ms5 37 38 63	OPG12 9	rtcs1 30
bif1 64	d3 65	K2S 25	ms7 37 38 64	OPH12 9	rth3 61
bif2-473CC 2	d5 61	K6L 53	ms8 37 38 64	OPN11 9	s4 46
bif2-70 2	d8 61	K9S 53	ms9 37 38 61	oro2 79	sdw1 64
bif2-1606 2	d9 63	kn1 61 66	ms10 37 38 65	orp 64	sh1 45 70
bif2-2354 2	d10 61	Kn1-0 2	ms11 37 38 65	orp1 62	sh2 10 13
Bl 82	des17 64	l3 79	ms12 37 38	orp2 65	si1 63
bm2 46	dhn1 63	l4 79	ms13 37 38 63	P1-oo-13:255A-10 7	sk1 61
bm3 79	dis1 63	l10 79	ms14 37 38 61	P1-rr 7	sl1 64
bnl3.03 72	dks8 58 61	l12 79	ms17 37 38 61	p1-rrb 27	slr1 30
bnl3.06 76 77	Dt 45 70	l15 79	ms20 37 38	p1-rww 27	sos1 62
bnl5.04 76 77	e1 86	la1 62	ms22 37 38	P1-wr 7	spec 34
bnl5.10 76 77	e2 86	lbl1 66	ms23 37 38 62	p1-wrb 27	Spm 70
bnl5.40 71	e8 86	Le 82	ms24 37 38	P1-ww 7	st1 62
bnl5.59 76	ed*-41v 58	les1 61	ms25 37 38	p1-wwb 27	su1 10 11 13 46 48 70
bnl5.71 71	eg1 63	les2 61	ms26 37 38	p1-www 27	79 80
bnl6.22 71	emb*-7065 58	les4 61	ms27 37 38	pb1 79	su2 11
bnl6.25 71	emb*-7182 58 59	les5 61	ms28 61	pb4 79	T1-2(4464) 79
bnl6.32 76	emb*-7190 58	les6 65	ms29 37 38	pd1 62	T1-9c 3
bnl7.49 78	emb*-7191 59	les8 65	ms30 38	pd2 64	T3-9(8562) 80 81
bnl8.17 76 77	emb*-7192 59	les9 64	ms31 38	pgd1 86	T4-9g 80
bnl10.13 78	emb*-7917 58	les10 61	ms41 62	pgd2 86	T5-9(4817) 80
bnl15.21 71	emb*-8518 26	les11 61	ms42 63	ph1 62	T5-9(8854) 81
bnl17.02 78	emb*-8521 26	les13 63	ms43 64	phi1 86	T5-9c 80 81
br1 61	emb*-8537 26	les15 61	ms44 62	phyA2 63	T9-10(059-10) 82
br2 46 61	emb*-8542 26	les16 65	ms45 38	phyB1 61	T9-10(8630) 81
br3 45	emb5 26	les18 61	ms45*-9301 38	phyB2 65	T9-10b 81
brt1 29	emb9 26	les19 61	ms45-6006 39	pl1 63 74	tan1 63
bt1 10 11 13	En 45	les20 61	Mu 2 58 70	pm1 62	TB-1La 76
bt2 70	es 82	lg1 61 70	MuDR 66	pn1 64	TB-1Sb 76 77

TB-1Sb-2L(4464) 79	umc113a 76
TB-4Sa 79	umc114 71 76 77
TB-5La 79	umc115 3
TB-5Sc 79	umc119 71 76
TB-6Lc 79	umc124 71
TB-7Sc 79	umc128 71
TB-9Lc 76 77	umc130 37
TB-9Sd 76 77	umc131 72
TB-10L19 77 79	umc139 71
TB-10L32 77	umc154 72
tb1 61	umc161 71
tbp1 61 64	umc167 76
tbp2 63	umc177a 76 77
td1bv1 63	umc247 76 77
te1 62	Uq 45
tet 34	uwo9 85
tga1 62	uwo10 85
tls1 61	uwo11 85
tp1 42	v13 79
tp2 42 65	v17 79
tp3 62	va1 64
tpi1 64	vg1 61
tr1 61 63	virG 34
Tr16L 84	vp1 62 70
trn1 65	vp2 63 72
ts1 61	vp5 61
ts2 61	vp7 72
ts3 61	vp8 61
ts4 62	vp9 64
ts5 62	vp10 79
ts6 61	vp12 79
tu1 62	vp13 79
ub1 82	w17 79
umb 82	wi1 63
umc4 38	wi4 63
umc5 72	wrp1 61
umc6 71	wx1 2 3 10 11 13 45
umc7 72	49 80 81
umc10a 72	Wx1-m 45
umc11 371	y1 15 70 79
umc13 3	y3 61
umc15 71	y9 65
umc16b 72	yd2 62
umc20 76	yg2 4 65
umc27 71	ys*-253 45
umc30 71	ys*-308 45
umc31 71	ys*-309 45
umc35 71	ys*-310 45
umc51 71	ys*-9196 45
umc54 71 72	ys1 45
umc58 76	ys3 45
umc59 71	ZAG1 32
umc59a 72	zag1 32
umc67 76 77	ZAG2 32
umc76 3	ZAG3 32
umc81 76 77	ZAG4 32
umc84 71	ZAG5 32
umc88 72	ZAP1 32
umc89a 71	zb*-143 45
umc90 71	zb1 81
umc93 71	zb2 81
umc95 76 77	zb3 81
umc104 71	zb7 42
umc108 72	zem1 32
umc109 76	ZEM2 32
umc113 71	ZEM3 32

ZMM1 32
zmm1 32
zn*-143 45

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X. AUTHOR INDEX

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- | | | | | |
|---|---|--|---|---|
| <p>Abler, BS 60 72
 Adams, MD 39
 Agrama, HAS 60
 Aguiar-Perecin, MLRde
 25
 Ajmone-Marsan, P 18*
 19* 60
 Albertsen, MC 37* 38*
 Anderson, EG 3
 Armstrong, CL 60
 Arziev, AS 33*
 Ashley 7
 Astiz Gassó, MM 50*
 Athma, P 7
 Austin, DF 60
 Ayonoadu, U 53
 Babcock, GD 23
 Bae, JM 70
 Bai, DP 84*
 Balconi, C 16* 17
 Ball, Y 1
 Bandaranayake, H 24*
 Bantín, J 31*
 Baron, A 7
 Basova, IA 46*
 Baysdorfer, C 117
 Beaumont, V 60 78
 Beavis, WD 60
 Beckett, JB 79
 Bedinger, P 38* 39
 Bender, J 5
 Bennett, B 1
 Bennett, MD 54
 Berke, TG 60
 Bertoia, LM 55*
 Bhat, NK 33
 Bianchi, A 53
 Birchler, J 108
 Black, DA 57
 Blyandur, OV 41
 Bohn, M 60
 Bohorova, NE 76
 Bommert, P 26*
 Bond, D 34*
 Bosio, D 16*
 Boston, R 1
 Bouchard, RA 56* 85*
 Bowen, B 1
 Brettschneider, R 31*
 Briggs, K 39*
 Briggs, S 2
 Brink, R 75
 Bruce, WB 34*
 Buckland, R 34
 Burilkov, VK 46* 47
 Busti, E 58*
 Bylich, VG 44*
 Byrka, SG 41
 Byrne, P 27
 Byrne, PF 27*
 Cai, T 34*
 Carlson, W 7 52
 Carman, J 68</p> | <p>Castiglioni, P 18* 19*
 Causse, M 60
 Cavallini, A 16*
 Chalyk, ST 41 44*
 Chan, M-T 20*
 Chang, A 28 76 77
 Chang, S-J 76* 77*
 Charcosset, A 60
 Chebotar, OD 44*
 Cheng 14
 Cheng, Y-M 77*
 Chernomorets, AA 44*
 Chernov, AA 60
 Chiavarino, AM 52* 53*
 Chomet, P 2 67
 Chopra, S 27
 Christensen, D 8*
 Chuchmii, IG 41
 Chuck, G 22
 Clark, J 26
 Coe, EH 1* 19 27* 60*
 72 75 108 117*
 Cokmus, C 24
 Comas, C 54*
 Cone, K 1 81
 Confalonieri, V 54*
 Consonni, G 58*
 Corcuera, VR 48* 49*
 Cordts, S 31*
 Couperman, FM 42
 Cowen, N 60
 Cristophe, T 33
 Cuadrado, A 55
 Cuevas, L 23
 D'Amato, F 16
 Davis, G 76
 Dawe, K 1
 Dellaporta, S 58
 Deng, Y 14*
 Doebley, JF 60
 Doering, H 83
 Dolfini, AS 58*
 Dresselhaus, T 31* 32*
 Driver, D 79
 Duncan, RE 16
 Edmeades, GO 8
 Edwards, M 60
 Elster, R 26*
 Emerson, RA 27 75
 Enaleeva, NC 73
 Esen, A 23* 24*
 Feix, G 29* 30*
 Fink, GR 5
 Fokina, ES 67
 Fox, TW 37* 38*
 Frascarolo, P 58*
 Freeling, M 22* 66
 Friedberg, JN 56*
 Furini, A 58*
 Fusari, F 19*
 Gabay-Laughnan, S
 108
 Gaber, RF 17</p> | <p>Gabriel, M 29
 Galinat, WC 68 82* 83*
 Garnier, P 34
 Gavazzi, G 58*
 Gengenbach, BG 16
 Ghatnekar, MV 53
 Ghidon, A 53
 Giulini, AP 58*
 Gonzalez, G 54*
 GoodmanMM 24 69
 Gould, J 20
 Greyson, RI 56* 85
 Griffing, B 41
 Grimsley, N 20
 Grizenko, GV 40
 Grotewold, E 27
 Gu, W 34*
 Guo, BT 9*
 Haas, G 2
 Hake, S 2* 3* 7
 Han, C 27
 Hanway, JJ 72
 Harrison, G 1
 Hartings, H17*
 He 14
 Helentjaris, T 39* 117
 Heredia-Diaz, O 60
 Hetz, W 31
 Heuer, S 31* 32*
 Hiei, Y 20
 Higginbotham, JW 60
 Hirschhorn 49
 Hobart, B 37 38
 Hochholding, F 29*
 30*
 Hoisington, D 76
 Hondred, D 34*
 Hood, EE 21
 Howell, WM 57
 Hudson, A 66
 Ishida, Y 21 22
 Jackson, D 2*
 Jackson, JD 22 79* 81*
 108*
 Jankovsky, JP 67*
 Jin, DM 9*
 Johal, G 2
 Jouve, N 55
 Jung-Mendaçolli 25
 Kaplinsky, NK 22*
 Kato, A 32*
 Kato, TA 25
 Kermicle, J 108
 Kerstetter, R 22
 Khatypova, IV 67*
 Khavkin, E 60* 72
 Khotyleva, L 41
 Kim, TS 71 72*
 Kindiger, B 84* 108
 Kobeleva, EN 41
 Koester, RP 60 72
 Koltunow 68
 Konotop, AI 41</p> | <p>Konstantinov, YM 33*
 Kopecka, M 29
 Kouzarides, T 34
 Kovarsky, AE 41
 Kowalewski, S 1
 Kowles, R 16
 Kozumplik, V 18* 60
 Kranz, E 31*
 Krapchev, B 76*
 Kravchenko, AN 42 44
 Kravchenko OA 43*
 44*
 Krell, S 34*
 Krivov, NV 42 45*
 Kriz, A 1
 Kruleva, M 76*
 Kuiper, M 19*
 Kuporitskaya, KI 41
 Kutka, FJ 8*
 Lafitte, HR 8
 Landi, P 72
 Landoni, M 58*
 Landschulz, WH 33 34
 Langdale, J 1 58
 Laudencia-
 Chingcuanco, D 3*
 Lebreton, C 60 72
 Lechner, T 17 18
 Lee, EA 27*
 Lee, M 60
 Lehmann, W 83
 Levings, CS 27
 Li, J 11* 13*
 Li, S 10* 11* 13*
 Li, YG 84*
 Lin, B-Y 76* 77* 78*
 Lin, YR 72
 Liu, JL 86*
 Liu, Q 28* 29*
 Liu, S 10*
 Liu, Y 10* 11* 13*
 Locatelli, F 21
 Longley, AE 53
 Lörz, H 31* 32*
 Lowe, B 7 67
 Lubberstedt, T 60
 Lukina, LA 67*
 Lupotto, E 20* 21
 Lusardi, MC 21
 Lusser, A 17
 Lysikov, VN 40* 43* 45*
 46*
 Ma, HP 15*
 MacCabe, AP 58*
 MacDonald, M 48
 Maillot, DS 56*
 Malkin, R 34
 Martienssen, R 7
 Mathern, J 7
 Matz, E 76 77
 McCarty, DR 70
 McClintock, B 25 42 43
 McDonald, M 9</p> | <p>McMullen, MD 1 27* 60
 108 117*
 McSteen, PCM 2*
 Mei, C 78*
 Mendel, G 83
 Michelmores, RW 71
 Miles, D 108
 Miranda, LEC de 25*
 Miranda, LT de 25*
 Molina, MdelC 50* 54
 Moore, G 54
 Mordvinova, VG 41
 Morgan, DT 56
 Morgante, M 18*
 Morgun, VV 41
 Motto, M 16* 17* 18*
 19*
 Moussa, ME 60
 Mulholland, J 29
 Murigneux, A 60
 Murin, AV 43
 Musket, T 117*
 Mylona, PV 68*
 Naranjo, CA 49* 52*
 53* 54*
 Natali, L 16*
 Nedev, T 76*
 Nelson, T 66* 67*
 Neuffer, MG 2 31 75
 108
 Newcomb 83
 Nguyen, DQ 26*
 Niemeyer, HM 23
 Nitsch, C 16
 Nitsch, JP 16
 Noble, S 37
 Orinshtein, ZA 41
 Orlov, YS 41
 Orr, AR 2
 Packer, L 33
 Pagliarini, MS 25
 Pali, AF 41 43*
 Parentoni, SN 71 72*
 Park, WJ 30*
 Paschenko, VM 46*
 Passera, S 20*
 Patterson, E 38*
 Pejic, I 18*
 Peng, S-F 78*
 Peterson, TA 7* 27 70
 Petrov, DF 67
 Phillips, RL 16 60 70*
 71* 72*
 Pierce, DA 34*
 Pierozzi, NI 25*
 Poggio, L 52* 53* 54*
 Polacco, M 1 116* 117*
 Polidoros, AN 68*
 Powers, L 44
 Quarrie, SA 72 79
 Quinn, CE 56*
 Racchi, ML 58
 Ragot, M 60 72</p> |
|---|---|--|---|---|

Rapoport, IA 41 43	Sachan, JKS 22	Sokolov, VA 67*	Torrezan, R 25	White 34
Reali, A 16* 20*	Sachs, MM 1 79 81*	Song, TM 15*	Trimnell, MR 37* 38*	Widholm, JM 4 78*
Rebai, A 60	108*	Stec, A 60	Tuberosa, R 71* 72*	Wild, F 34
Rees, H 53	Saghai Maroof, MA 60	Stinard, P 4 79* 81*	Tyrnov, VS 73* 74*	Will, GF 8
Rembrandt van Rijn 83	Salvi, S 71*	108*	Veit, B 2	Williams, J 39
Ribaut, J 60	Sandoval, MC 48* 49*	Stuber, CW 24 27 60	Veldboom, LR 60	Wolfe, KW 28* 29*
Richard 83	Sanguinetti, MC 72*	72 86	Venter, C 39	Wolfe, RI 47*
Richert, J 31	Sari-Gorla, M 60	Subota, IY 33*	Vidal, M 17	Wolfe, AP 17
Rivin, C 58	Sarkar, KR 22	Sun, SM 9*	Vladutu, CI 70*	Yang, T 10*
Robertson, DS 3* 4 67	Satarova, TN 28*	Sun, ZL 9*	Vogel, J 1	Yang, Z 56* 85*
108	Scandalios, JG 68*	Sundberg, MD 2	Vosa 25	Yudin, BF 67
Rocheferd, TR 60 78*	Schon, CC 60	Tagliani, LA 34*	Wade, PA 17	Zavalishina, AS 74*
Rohlf 19	Selivanov, AS 73	Taramino, G 18*	Waites 66	Zehr, BE 60 72
Romanova, IM 46*	Sen, CK 33	Taunton, J 18	Walden, DB 56* 85*	Zeng, M 10* 11* 13*
Rosato, M 52* 53*	Shahid, M 23* 24*	Teas, HJ 3	Wan, Y 60 78	Zerial, M 34
Roseman, RR 52	Sheridan, WF 1 26 38*	Tenborg, R 38	Wang 14	Zhang, CL 9*
Rosenbrook, B 38	Shi 14	Tian, Z 14*	Wang, B 9*	Zhao, YL 15*
Rosi, P 52*	Simcox, K 60	Tie, SG 86*	Wassom, JJ 78*	Zhao, ZY 34*
Ross, JG 16	Singh, M 4	Timmermans, M 66*	Webber, AN 34	Zheng YL 86*
Rossi, V17*	Sinha, N 1	Ting, YC 26*	Weber 34	Zhou, H 14*
Rudert, ML 34*	Smith, RH 21	Todorova, L 76*	Weil, C 1	Ziff, E 34
Running 26	Snook, ME 27*	Torrecillas, M 55*	Werr, W 26* 70	

MOM and pop corn

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"DON'T BE LIKE HIM!" 1958. BY K. IVANOV

Don't be like him!

*At every meeting he is swearing and
complaining that the corn doesn't
grow well*

*He came home and blushed in embar-
rassment because in his daughter's
garden the corn was growing just fine!
Corn will grow where people like to work
hard!*

a final note

NOTES & NEWS

Buzz Westfall • St. Louis County Executive

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A KERNEL OF GOOD NEWS

In 1995, the Critical Technologies Partnership -- a project of St. Louis County Economic Council and Civic Progress -- formed a collaboration among industry, academia and the National Corn Growers Association (NCGA) to develop a plan that focused on identifying specific genes that control the traits of corn which influence farming efficiencies and economies -- maximizing yields, minimizing environmental impact and improving nutritional value. Acting on these ideas, the NCGA has broadened the collaboration and achieved recent Congressional approval of \$40 million to the National Science Foundation for funding the Plant Genome Initiative. The initiative will focus on deciphering the genetics of corn and other economically significant crops to keep the U.S. competitive in the global marketplace. ■



A BI-MONTHLY PUBLICATION OF
ST. LOUIS COUNTY ECONOMIC COUNCIL

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The mission of St. Louis County Economic Council is to facilitate the development of long-term, diversified business and employment opportunities that will lead to increased wealth and well-being for the citizens, businesses, and institutions of St. Louis County and the St. Louis region.

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Univ Of Mo - Columbia 210 Curtis Hall
Columbia, MO 65211

developments



April, 1998

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The Notes in this Newsletter are cooperatively shared information. The data and ideas here are not published, but are presented with the understanding that they will not be used in publications without specific consent of the authors

Notes for the 1999 Maize Genetics Cooperation Newsletter need to be in the editor's hands before 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, and an electronic version. Please follow the simple style used in this issue (city /institution title /--authors; tab paragraphs; give citations with authors' initials --e.g., Maizer, BA et al., J Hered 35:35, 1995, or supply a bibliography). Figures, charts and tables should be compact and camera-ready, and supplied in hard copy accompanied by electronic form (jpg or gif) if possible. To separate columns in tables, please tab instead of using spaces for quality tabulations on the web. Send your MNL Notes, hard copy and electronic, anytime; they will go on the Web verbatim, and will be printed in the annual issue. Electronically, either (1) attach to an email addressed to me; (2) send by FTP to teosinte.agron.missouri.edu (pub/mnl_subunit director; see MaizeDB for details), and alert us with an email to ed@teosinte.agron.missouri.edu; or (3) send a diskette. By all means forward a double-spaced hard copy of the text, with the figures. Send to:

Ed Coe
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Columbia, MO 65211

SEND YOUR ITEMS ANYTIME; NOW IS YOUR BEST TIME

Author and Name Indexes (and see **MaizeDB**)

Nos. 3 through 43
Nos. 44 through 50
Nos. 51 to date

Appendix to MNL 44, 1970 (copies available)
MNL 50:157
Annual in each issue

Symbol Indexes (and see **MaizeDB**)

Nos. 12 through 35
Nos. 36 through 53
Nos. 54 to date

Appendix to MNL 36, 1962 (copies available)
MNL 53:153
Annual in each issue

Stock Catalogs

Marker Stocks
Translocations

In this issue and **MaizeDB**
MNL 55:140 and **MaizeDB**

Rules of Nomenclature (1995)

MNL69:182 and **MaizeDB**

Cytogenetic Working Maps

MNL 52:129-145; 59:159; 60:149 and **MaizeDB**

Gene List

MNL69:191; 70:99 and **MaizeDB**

Clone List

MNL 65:106; 65:145; 69:232 and **MaizeDB**

Working Linkage Maps

MNL69:191; 70:118; 71; and **MaizeDB**

Plastid Genetic Map

MNL 69:268 and **MaizeDB**

Mitochondrial Genetic Maps

MNL 70:133 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 the ordered array of map scores for molecular markers or counts by phenotypic classes; 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 <http://www.agron.missouri.edu/Coop/clonesheet96.html>, or the Clone Information Sheet in the back of this issue. EC

