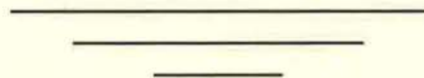
# MAIZE GENETICS COOPERATION

# NEWSLETTER

# 73



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Robert I. Brawn
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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/). 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 has 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, is 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.

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; pulled together 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, processing of figures, 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 42d Maize Genetics Conference, at Couer d'Alene, Idaho, March, 2000, will be on the MaizeDB Web, **http://www.agron.missouri.edu,** and information packets will be mailed to former attendees in November 1999; others may request the mailing by providing their address to Coe. The program and abstracts are prepared from electronic submissions; back copies are available from Coe, or see MaizeDB. Electronic submission, "Webification", and printing of abstracts will be done by Mary Polacco. The Steering Committee for the 2000 Maize Genetics Conference is:

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**Preparing notes for the next issue (Number 74, 2000)?**
**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/mapit.html.**

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Editor Coe

ALBANY, CALIFORNIA
Plant Gene Expression Center
COLD SPRING HARBOR, NEW YORK
Cold Spring Harbor Lab

**The genetics of ear fasciation in maize**
--Jackson, D, Hake, S

Fasciation, from the Latin, fascis, meaning bundle, is a process that describes variations in plant form resulting from abnormal growth of part of the plant body. Usually fasciation is recognized by an enlargement and flattening of the stem, resulting from proliferation at the shoot apex. In principle, this could occur by a number of mechanisms operating in the central or peripheral zone of the shoot apical meristem:

(i) by an increase in the rate of cell division in the central and/or peripheral zone, or

(ii) by a delay in the transition from central to peripheral zone identity, resulting in an accumulation of central zone cells, or

(iii) by a delay in the incorporation of peripheral zone cells into primordia, resulting in an enlargement of the peripheral zone.

Heritable ear fasciation in maize has been known for a long time (Weatherwax, 1935, The American Midland Naturalist 16:1-71). Several years ago we reported a new mutant of maize called *fasciated ear* (*fae*) (Hake and Veit, MNL 62:2, 1988). Mutant plants had enlarged and branched ears resulting from fasciation of the ear inflorescence meristem. Following introgression into the B73 inbred line, we noticed that the phenotype was modified and the ear branches were smaller and coming from the base of the ear. This phenotype resembled the ears of *ramosa3* (*ra3*) plants, and crossing these mutants together showed that they fail to complement. Subsequent F2 analysis indicated that *fae* and *ra3* are indeed allelic, and we have therefore renamed this allele *ra3-fae1*.

I (DJ) am characterizing new recessive fasciated ear mutants, preliminarily named *fae2* and *fae3,* that were gifts from Paul Chomet (Dekalb Plant Genetics) and maize cooperators in Krasnodar, Russia, respectively (Fig. 1). In addition, the *compact plant2* (*ct2*) mutation from the Maize Stock Center has severely fasciated ears as well as a thick tassel phenotype. A second isolate of *ct2*; *ct2-rd3*, from the Stock Center, also shows severely fasciated ears.

The *compact plant1* mutant is also described in the Maize Newsletter gene list as having furcated (branched) ears, but in my experience *ct1* plants have normal ears, and their description may have been confused with *ct2*. Phenotypic and molecular characterization of the fasciated ear mutants is in progress.

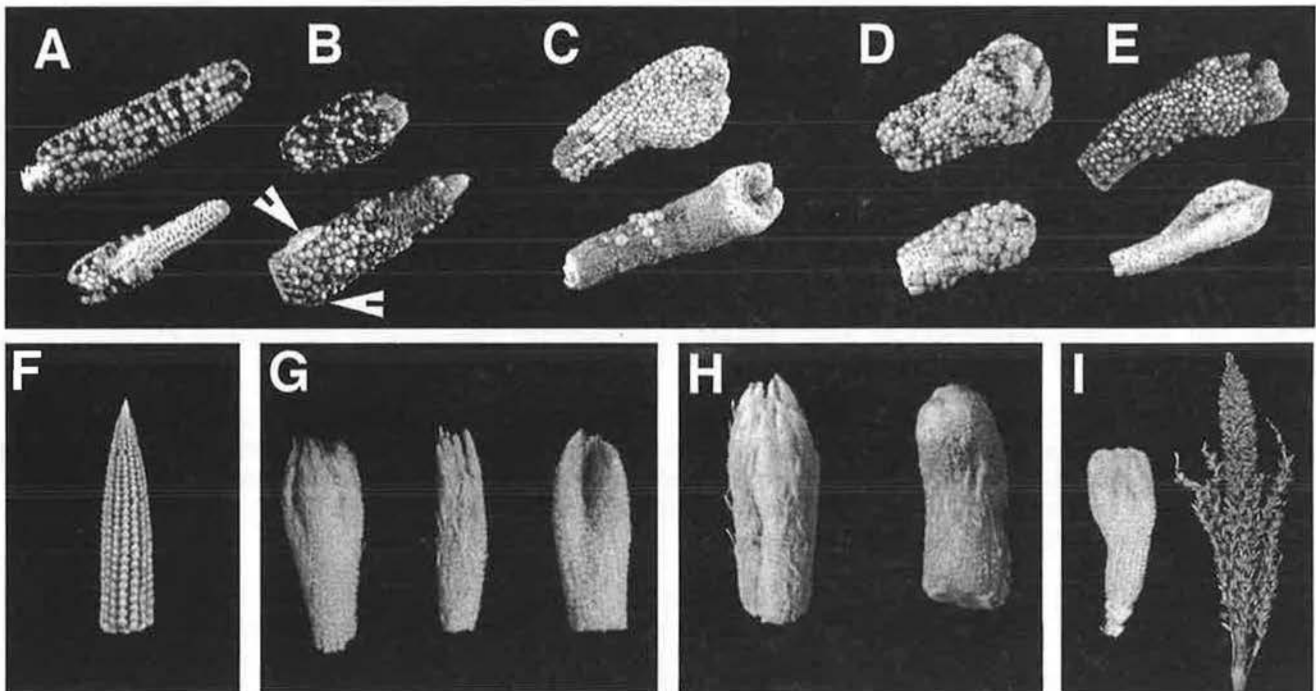


**Figure 1. Fasciated ear mutants of maize.**

A. Normal mature ears showing straight rows of kernels. The ears taper towards the tip (to the right of this ear).
B. *ra3-fae1* mutant ears. The upper ear is fasciated and the tip is broad and flattened and the rows of kernels are irregular. The ear below from a different family does not show the fasciated phenotype but has branches at the base (arrows). These are axillary inflorescence branches that sometimes bear kernels.
C. *fae2* mutant ears. The upper ear shows broadening and flattening of the tip of the ear and the rows of kernels are irregular. The lower ear has been sparsely pollinated and shows the cob structure, typically this mutation shows line fasciation where the apex is flattened and extended into a curving line.
D. *fae3* mutant ears. The tip of these ears is broad and branched, and typically undergoes splitting and ring fasciation to form an ear that is hollow on the inside.
E. *ct2* mutant ears. These ears show flattening and broadening of the tip, somewhat similar to *fae2*. The lower ear is not pollinated and shows the distorted shape of the cob.
F. Immature normal ear, with the silks removed to show the narrow tapering tip and the regular rows of florets.
G. Immature *fae2* ears, showing flattening and branching of the tip of the ear.
H. Immature *fae3* mutant ears, in this case the tips of the ear are broader in all directions rather than being flattened.
I. Immature *ct2* ear showing severe enlargement and flattening at the tip. This mutation also causes broadening of the central spike of the tassel, shown to the right.

ALBANY, CALIFORNIA
USDA-ARS, Plant Gene Expression Center
URBANA, ILLINOIS
University of Illinois

**Wab (*Wavy auricles in blades*), a dominant leaf mutation located on chromosome 2L**

--Hake, S, Hester, H, Wassom, J, Widholm, J, Rocheford, T

We have characterized and mapped a new dominant leaf mutation that arose spontaneously in an in vitro androgenesis tissue culture derived line. Normal maize leaves have a blade portion that is broad and flat, a sheath portion that wraps around the culm, and a ligule and auricle located at the junction of blade and sheath. The ligule is an epidermally-derived fringe and auricles are the two pale green, wedge-shaped tissues located just above the ligule. The *Wab* (*Wavy auricles in blade*) mutation is characterized by auricle tissue that is found in the blade portion of the leaf. Most often the extra auricle is adjacent to the normal auricle, spreading distally, other times the extra auricle is surrounded by normal leaf blade tissue (Figure 1). *Wab* leaf blades have a more pronounced horizontal posture, possibly due to the extra auricle tissue. Leaf blades are also narrower. We measured leaf widths for 40 mutants and normal siblings in a population that was back-crossed to B73 three times. *Wab* leaves #13 and #14 (counting from the base of the plant) were 59% and 57% of the wild-type width. We have also noted that there are one to three more leaves in *Wab* mutants that normal siblings. Occasionally, leaves are found that are very narrow and have no ligule at all, otherwise the normal ligule is not affected.

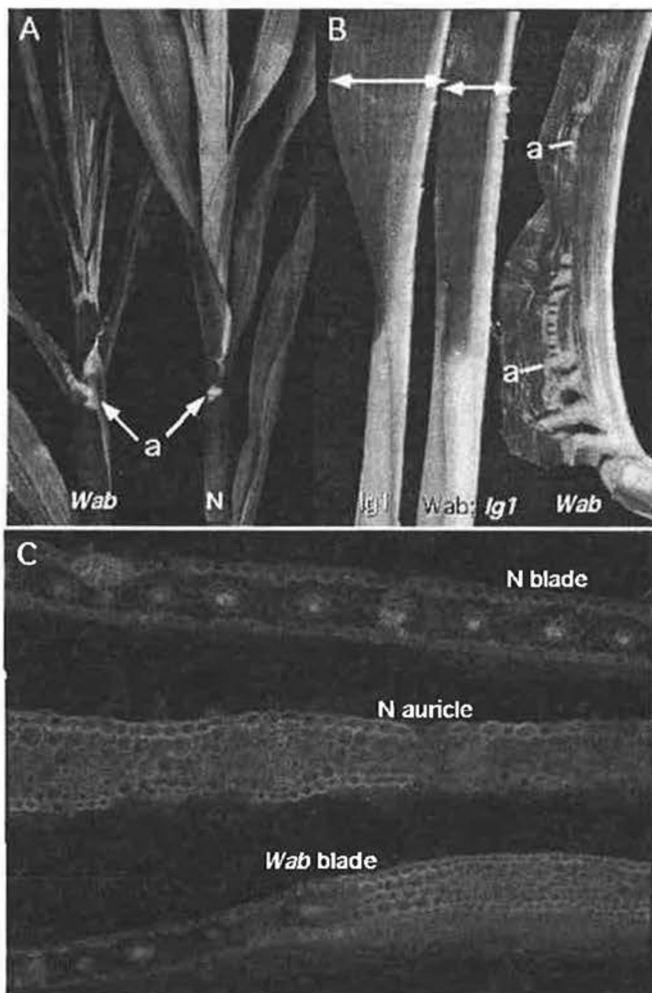


Figure 1. A. *Wab* and a normal sibling are compared in a colored background in which the auricles are white and the blade and sheath red. The auricle tissue extends up the leaf margin in the *Wab* individual (a = auricle). B. Adaxial surface of half leaves of *lg1*, *Wab*, and a *lg1*; *Wab* double mutant are compared. Compare the leaf widths (arrows). C. Hand-sections of normal and *Wab* leaves were examined under the fluorescent scope. Portions of the *Wab* blade resemble the normal auricle in histology.

The phenotype is fully penetrant and severe in B73. The phenotype is less severe in A619 and Mo17. A preliminary examination of a small population suggests that homozygotes are not obviously different than heterozygotes. The phenotype is most striking in adult plants, but can be scored as early as leaf #4 by the presence of slightly translucent margins and narrower leaves.

We crossed *Wab* mutants into a *liguleless1* (*lg1*) mutant background to determine the effect on the ectopic auricle. *lg1* mutants lack a ligule and auricle, and have very upright leaves (Sylvester et al., Development 110:985 1990). The normal and ectopic auricle of *Wab* mutants disappeared in the *lg1* background and the leaves were more narrow than the leaves of *lg1* single mutants (Figure 1). The margins of the *Wab;lg1* mutant at the sheath/blade border were membranous.

We used waxy translocation stocks to map the mutation to chromosome 2. Linkage was found with both T2-9b and T2-9d. Further mapping of a population back-crossed twice into B73 showed tight linkage with *umc98a*. One recombinant was found in a total of 32 giving an approximate map position of 3 cM from *umc98a*.

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**Characterization of the maize *P-wr* allele using reverse genetics**

--Dinges, J, Chopra, S, Walters, L, Meeley, R, Peterson, T

The maize *P* locus encodes a *Myb*-homologous regulator of red phlobaphene pigment biosynthesis in pericarp, cob glumes, and other floral tissues (Grotewold et al., Cell 76:543-553, 1994). The *P-wr* allele of the *P* gene specifies the accumulation of pigment in the cob, but not the pericarp (Styles and Ceska, Maydica 34:227-237, 1989). *P-wr* consists of a multi-copy gene complex with approximately six tandem repeats aligned in a head-to-tail arrangement (Figure 1A) (Chopra et al., Molecular and General Genetics 260:372-380, 1998).

In order to gain a greater understanding of the tissue specificity associated with this allele, induced mutations were identified using the Trait Utility System for Corn (TUSC) developed by Pioneer Hi-Bred International, Inc. (Meeley and Briggs, MNL 69:67,82; Bensen et al., Plant Cell 7:75-84, 1995). TUSC is a reverse genetics tool based on PCR and the *Mutator* transposable element family. Using several *P-wr* specific primers, we have detected 13 unique *Mu* insertion events within a 1.4 kilobase region of *P-wr* including a portion of the promoter, exon 1, intron 1, exon 2, and part of intron 2 (Figure 1C). Screening for insertion events in the 3' region of *P-wr* is continuing.

We have not yet identified which of the six copies of *P-wr* contain the *Mu* element insertions. There are a number of sequence

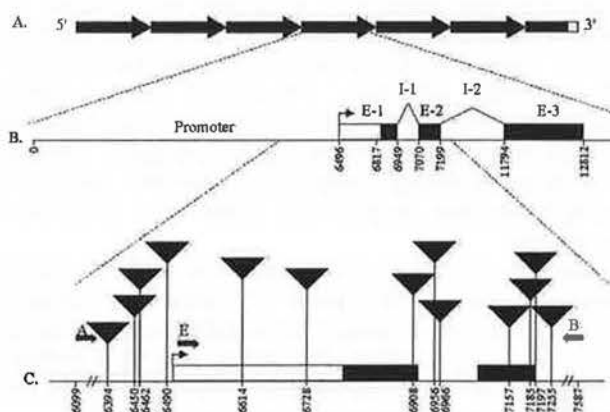


Figure 1. A. The *P-wr* multi-copy gene complex. Each 12.6 kb repeat is indicated by the arrows. The 6.4 kb truncated copy lying at the 3' end is represented by the dark box. The 157 base pairs of chromosomal DNA flanking one *Mu* insertion is indicated as the putative 3' end of the *P-wr* complex (open box). B. Enlargement of a single *P-wr* copy showing the exon/intron structure. The dark arrow shows the transcription start site, the open box indicates the untranslated leader sequence, and the black boxes indicate the coding regions. C. Enlargement of the area screened for *Mutator* insertions using TUSC. The triangles represent *Mu* element insertion locations, with the nucleotide position indicated below. The positions of PCR primers A, B, and E used for TUSC pool screening are shown by the large arrows.

polymorphisms in the *P-wr* genomic sequences, both among the segments flanking the 13 different *Mu* insertion clones, and in comparison to the previously determined genomic and cDNA sequences of *P-wr* from inbred line W23 (Chopra et al., Plant Cell 8:1149-1158, 1996). These polymorphisms include a twenty-eight base pair direct duplication, several smaller (3 – 6 bp) direct duplications, and numerous single base pair changes, insertions, and deletions. Many of the polymorphisms are found on more than one independent clone, ruling out their origin from PCR or sequencing errors. These polymorphisms could be due to differences between the several *P-wr* alleles that were used in the TUSC population, or to sequence differences among the six gene copies of the *P-wr* complex. The latter situation would facilitate the mapping of *Mu* insertions to a particular gene copy.

An unexpected outcome of the TUSC approach is the possible detection of the 3' end of the *P-wr* gene complex. The junction between the *P-wr* complex and the flanking chromosomal DNA has eluded previous conventional cloning attempts. One of the TUSC clones contains a *Mu* insertion located 157 bp 3' of a truncated *P-wr* sequence. This truncated *P-wr* copy contains the promoter region, but none of the coding sequence. The 157 base pairs between the truncation of *P-wr* and the *Mu* element have no homology to the known *P-wr* sequence, nor any relevant database hits. PCR experiments on genomic DNA have confirmed the presence of this copy, but a positive Southern blot confirmation has yet to be completed.

The phenotypes of the *Mu* insertion alleles should help to identify which copy or copies of the *P-wr* complex are functional. Gain of *P* function in pericarp and/or loss of function in cob glumes are particularly striking phenotypes that may result from transposable element insertions into the *P-wr* gene. The genetic transmission and/or phenotypes resulting from many of these *Mutator* insertion events are yet to be characterized, and one season of outcrosses may be necessary to make the plants more vigorous. However, one interesting case was identified in the summer of 1998. A plant with a gain in *P* pericarp function has been identified as an ear with a red sector. The ear was derived from a family that contains a *Mu* insertion in the 5' untranslated leader sequence (position 6728). The accumulation of pigment in the pericarp may be a result of the phenomenon of *Mu* suppression, wherein transcription begins from an outward-reading promoter present in the TIR of the *Mu* element (Barkan and Martienssen, Proc. Natl. Acad. Sci. USA 88:3502-3506, 1991). Further testing will be required to establish the mechanism of this ectopic *P-wr* expression in pericarp. Nonetheless, this result indicates that the *P-wr* gene product can function in pericarp when it is expressed there. Analysis of this and other novel phenotypes arising from the mutant plants should provide key insights into the tissue specificity associated with *P-wr*.

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**The ultrastructure of developmental anthers of genic male sterile *ms10* in maize**

--Zhou, H, Sun, R

Anther development of *ms10* was studied by transmission electron microscopy. The results indicated that the ultrastructure of the male sterile line was different from that of the fertile line. ms microspores could not continue their development after tetrads. Faces of microspores and inside faces of tapetal cells did not form "U body", but formed many filiform structures (see photo). From this time on, microspore development stopped, and almost all matter in cells degraded and caused sterility. This filiform structure was not reported before. We conducted two years of observation using Huangzao4(ms10), and the results were the same.

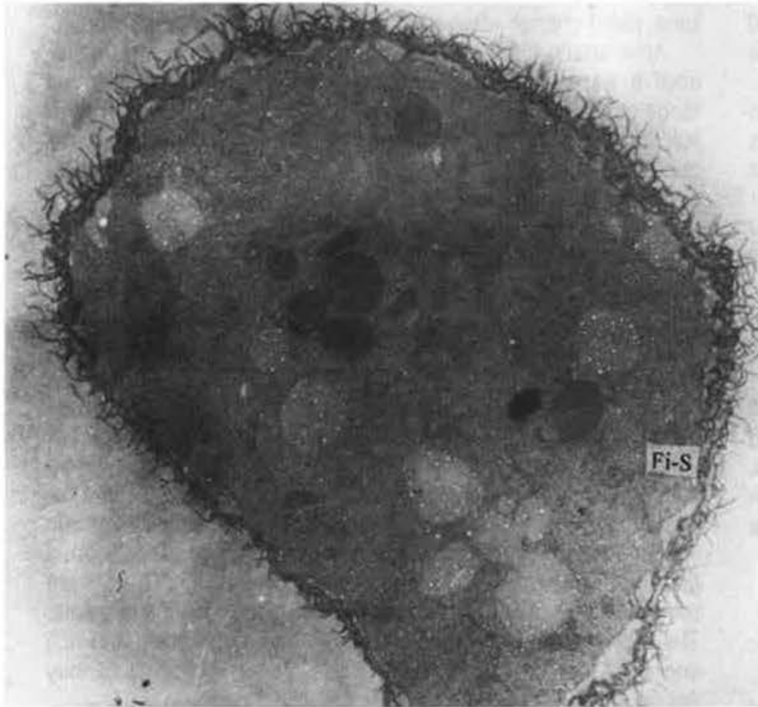



Figure. Huangzao4(ms10) microspores, filiform structure (Fi-S) on the faces.

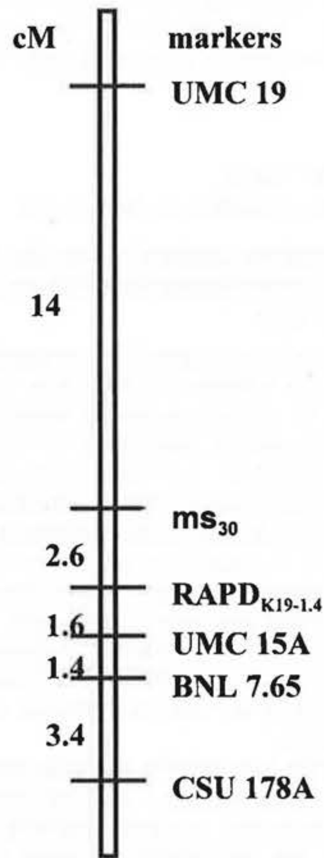
**Molecular mapping of a male sterile gene (*ms30*) in maize**
--Liang, Y, Zhou, H, Jiang, W

Professor Li Jingxiong found a male sterile gene (his designation is msx) and located it on maize chromosome 4 with B-A translocations (unpublished data). Dr. Zhou Hongsheng (1997) studied microspore development and found that pollen in Msx msx was normal, but that the msx msx anther was abnormal from the uninucleate stage. They thought it was a new male sterile gene and temporarily named it *ms30* in the book "Biology of Male Sterility in Maize" (Li Jingxiong et al., 1998). Professor Li gave some seeds to M. C. Albertsen who tested the gene and published a paper in MNL71. In his paper, Dr. Albertsen designated the gene as *ms*-li89*. In last year's MNL72, Dr. Albertsen has designated an ms mutant *ms*-WL87A* on 2L as *ms30* (see MNL72:38). So, we have the same name for different mutants. How to resolve the problem?

We mapped *ms30* by RFLP and RAPD markers in this report. A BC1 population derived from 6960 (*ms30 ms30*) X Zhonghuang17 (*Ms30 Ms30*) and a sibling population SIB5(((2603SuSu *ms30 ms30*/2611susu *Ms30 Ms30*)F2)SIB5) were employed as map populations for RFLP analysis, while the BC1 was used for RAPD analysis. Eighteen probes on maize chromosome 4 and BSA analysis were used to screen with RFLPs, 278 10-mer random primers and BSA analysis were employed to identify RAPDs. By using JoinMap software, linkage as well as genetic distance between *ms30* and markers were obtained. As a result, *ms30* was mapped on chromosome 4. The main results were as follows:

1. Observing anthers at the flowering period, we found that 71 plants were sterile while 64 plants were fertile among 135 individuals in the SIB5, and 64 plants were sterile while 57 plants were fertile among 120 individuals in the BC1. Thus the ratio of

Map of $ms_{30}$



sterility to fertility conformed to the expected ratio 1:1.

2. RFLP analysis on the SIB5 population showed that *ms30* was tightly linked with two RFLP loci *umc15a* and *umc66a* on maize 4L, the recombination value was 5.9% and 14.8% respectively.

3. In the BC1 population, polymorphism was detected between parents and between two bulked DNA pools by the probes *umc66a, umc19, umc15a, bnl7.65, csu178a* and *csu91a*. While *umc19, umc15a, bnl7.65, csu178a* were used to conduct further analysis on BC1 individuals. Results showed that *ms30* was tightly linked to them, the genetic distance was *umc19*-14cM-*Ms30/ms30* -4.2cM-*umc15a*-1.4cM-*bnl7.65*-3.4cM-*csu178a* (see Figure).

4. In RAPD analysis, a RAPD marker ($RAPD_{K19-1.4}$) was found to be tightly linked to *ms30*. The genetic distance was 2.6cM.

Results from RFLP analysis on two different segregating populations strongly supported that *ms30* was on maize 4L, which was in accordance with the previous study by the B-A system. Because two known male sterile genes on chromosome 4 are dominant, therefore the recessive male sterile gene *ms30* is a new ms gene.

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**Effects of space induced variations in maize (*Zea mays* L.). I. Influence of space on photosynthetic pigment and chloroplast**

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

Dried seeds of plants carried by recoverable satellites to near-earth orbit are subjected to the effects of manifold space mutagens, such as microgravity, cosmic radiation, high vacuum, etc., and variations emerge among the progenies of plants grown from the seeds. So far, the experiments in this field in China and abroad mainly concentrated on morphological effects, physiological and biochemical mechanisms of induced variations, and selection of mutants, while reports about changes of plant chloroplast ultrastructure and photosynthetic pigments were very few, and no information about maize (*Zea mays* L.) was found. The objective of this experiment was to study changes of chloroplast ultrastructure and photosynthetic pigment contents of maize leaves after space flight, and try to elucidate the cytological and cytogenetical bases of the changes.

**Plant materials and satellite carrying treatments.** Dried seeds of two maize inbred lines, YiZi24 and Yi141, were divided into two parts, one of which was used as ground control, the other was carried by the Recoverable Scientific Exploration and Technological Experimentation Satellite of China, launched on October 20, 1996 and returned to earth after 15 days. The satellite conditions were: flight altitude, Perigee 175±5 km, Apogee 235±5 km; period around the earth, 90 min, total time of space flight, 360 h; temperature in the biocabin, 10~20 C; vacuum degree, $10^{-3}$~$10^{-6}$ Pa; gravity level, $10^{-3}$~$10^{-5}$ G; total dose of radiation (15 d), 1.92 mGy.

**Effects of space flight on photosynthetic pigment contents in maize leaves.** Determination of absorption spectra of acetone extracts of maize leaves revealed that the absorption curves of space-flight treated were quite similar to those of their corresponding ground controls at the same time of sampling, only that the absorbance values of the former were slightly lower near 663 nm and 645 nm (maximum absorption peaks of chlorophyll a and b, respectively). This showed that their light-absorption characteristics didn't change after space flight.

After space flight, the contents of chlorophyll a and chlorophyll b were significantly reduced, and the relative reduction range of chloroplayll b far exceeded that of chlorophyll a. The result suggested that the former was more sensitive to space mutagens. As shown in Table 1 (A, B), there was a substantial decrease of chlorophyll (a+b) contents after space flight that lowered the total amount of their photosynthetic pigments (Ca+b+k), except for that of YiZi24 60 days after sowing for its carotenoid contents were far higher than those of ground control.

**Effects of space flight on chloroplast ultrastructure of maize leaves** Electron micrographs of chloroplasts from ground controls showed the shape of a typical convex lens composed of alternately arranged grana and stroma lamellae. The double outer envelopes and intima systems were observed clearly. The thylakoid membranes in grana and stromata were rich in number and arranged in an orderly fashion. Each granum disc was stacked with several to more than twenty thylakoids which made the section of granum discs approximating a rectangular shape. There were plenty of ribosomes and some osmiophilic globules in the stromata. The fact that the chloroplasts had integrated structure, and rich and clear photosynthetic membrane systems, indicate that they possess a powerful photosynthetic capacity.

After space flight, a series of chloroplast configuration changes, such as strip shape, circular shape, and irregular amoeba shape, etc, were often observed. The main changes in the internal structure of chloroplasts were incompletely developed systems of grana and stroma lamellae, a marked decrease in the number of grana per chloroplast and thylakoids per granum, a notch or notches on the envelope membrane, and blurring of granum lamellae. Twisted and disorderly arranged stroma lamellae were often arranged in a radiating fashion with centers of obscure granum discs , or arranged in a parallel manner without stacking into granum discs. Swollen thylakoids were seen, resulting in an increase of intrathylakoid space (see Fig. 1, Fig. 2).

The most obvious difference between bundle sheath chloroplasts of space-flown plants and ground control was that the former possessed a far greater number of starch grains. However, further studies are needed to make sure whether or not it was caused by microgravity as some authors postulated.

**Table IA Effects of space flight on photosynthetic pigment contents of maize leaves(hg/ml)**

| groups | | chlorophyll Ca | | | chlorophyll Cb | | | chorophyllC(a+b) | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | ck | sp | Change of sp relative to ck(%) | ck | sp | Change of sp relative to ck(%) | ck | sp | Change of sp relative to ck(%) |
| Yil41. | I * | 41.127 | 37.745 | -8.2 | 21.739 | 16.391 | -24.6 | 62.866 | 54.136 | -13.9 |
| | II | 40.397 | 39.414 | -2.4 | 19.517 | 17.308 | -11.3 | 59.896 | 56.722 | -5.3 |
| | III | 41.482 | 41.983 | 1.2 | 49.432 | 29.552 | -40.2 | 90.914 | 71.535 | -21.3 |
| | IV | 41.431 | 41.344 | -0.2 | 34.860 | 28.150 | -19.2 | 76.292 | 69.494 | -8.9 |
| Yizi24 | I | 35.087 | 36.212 | -25.3 | 16.581 | 10.242 | -38.2 | 51.668 | 36.454 | -29.4 |
| | II | 37.747 | 37.192 | -1.5 | 19.172 | 15.411 | -19.6 | 56.919 | 52.630 | -7.5 |
| | III | 41.621 | 40.336 | -3.1 | 28.809 | 21.439 | -25.6 | 70.430 | 61.805 | -12.2 |
| | IV | 38.493 | 40.230 | 4.5 | 58.333 | 23.165 | -60.3 | 96.826 | 63.395 | -34.5 |

* I, II, III, IV are the photosynthetic pigment contents of maize leaves from plants 13days after germination in laboratory and 60,70,and 90 days after sown in the field, respectively. ck: ground control; sp: space treatment

**Table IB Effects of space flight on photosynthetic pigment contents of maize leaves (hg/ml)**

| groups | | CK | | | Ca+b+k | | | Ca/Cb | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | ck | sp | Change of sp relative to ck(%) | ck | sp | Change of sp relative to ck(%) | ck | sp | Change of sp relative to ck(%) |
| Yil41 | I * | 7.208 | 9.381 | 30.1 | 70.074 | 63.517 | -9.4 | 1.892 | 2.303 | 21.7 |
| | II | 1.758 | 2.881 | 63.9 | 61.654 | 59.603 | -3.3 | 2.067 | 2.277 | 10.2 |
| | III | 2.609 | 1.503 | -4.04 | 93.523 | 73.038 | -21.9 | 0.839 | 1.421 | 69.4 |
| | IV | 2.644 | 3.056 | 15.6 | 78.936 | 72.550 | -8.1 | 1.195 | 1.469 | 22.9 |
| Yizi24 | I | 4.754 | 4.707 | 1.0 | 56.422 | 41.161 | -27.0 | 2.116 | 2.559 | 20.9 |
| | II | 3.459 | 17.205 | 397.4 | 60.378 | 69.835 | 15.7 | 1.969 | 2.413 | 22.5 |
| | III | 1.171 | 3.503 | 199.2 | 71.601 | 65.308 | -8.8 | 1.445 | 1.881 | 30.2 |
| | IV | 3.231 | 2.064 | -36.1 | 100.05 | 65.459 | -34.5 | 0.660 | 0.591 | -10.5 |

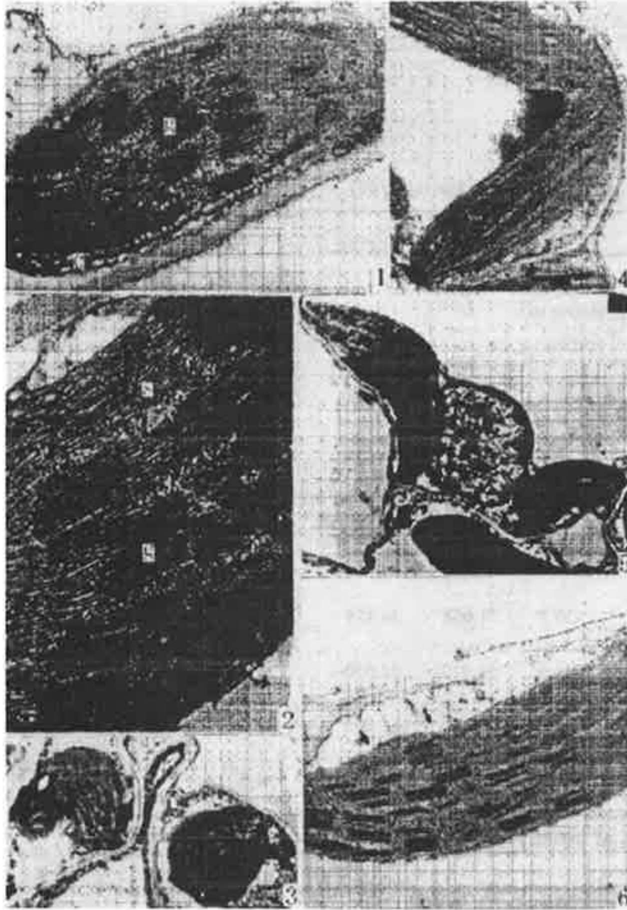


Figure 1. Chloroplasts of maize mesophyll cells from ground control, showing more granum discs and thylakoid membranes arranged in orderly fashion. (1) X 30000 X 0.4; (2) X 49500 X 0.4; (3) ~ (6) Chloroplasts of maize mesophyll cells after space flight. (3) Showing changes of chloroplast contours X 7500 X 0.4; (4) Showing grana lamellae decreased in number, X 9900 X 0.4; (5) Showing changes of chloroplast contours, X 5000 X 0.4; (6) Showing notch of chloroplast envelope.

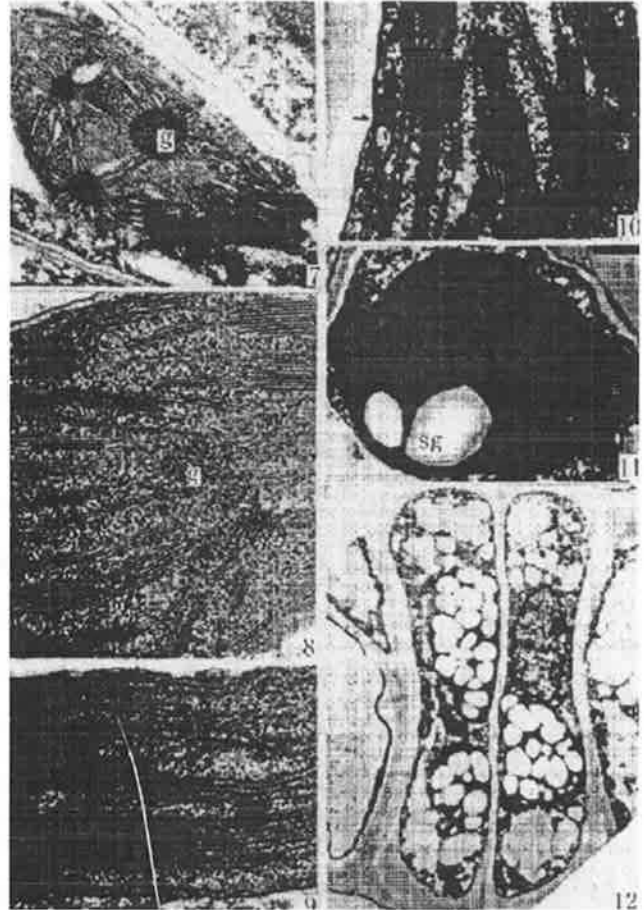
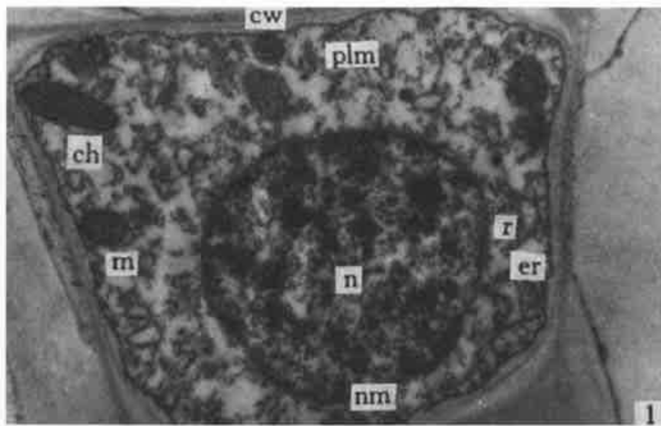


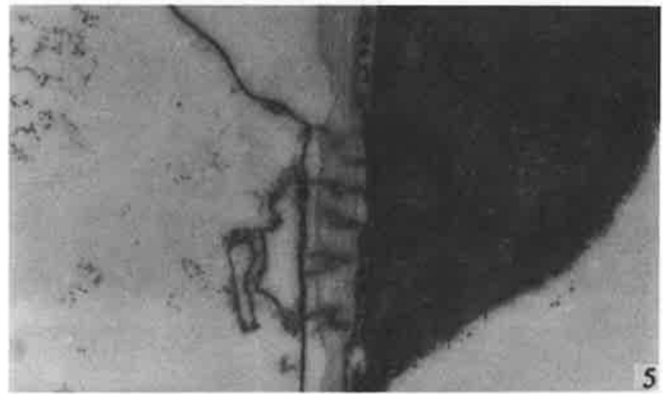
Figure 2. Chloroplasts of maize mesophyll cells after space flight. (7), (8) Showing thylakoids arranged in a radiating fashion, with centers of obscure grana discs, (7) X 24000 X 0.4, (8) X 49500 X 0.4; (9) Showing thylakoids arranged parallelly, no grana discs, X 49500 X 0.4; (10) Showing swelling of thylakoids and notch of envelope membrane (arrow), X 49500 X 0.4; (11) of corn bundle sheath cells from ground control, X 13000 X 0.4; (12) cells of corn bundle sheath cells after space flight, showing more starch.

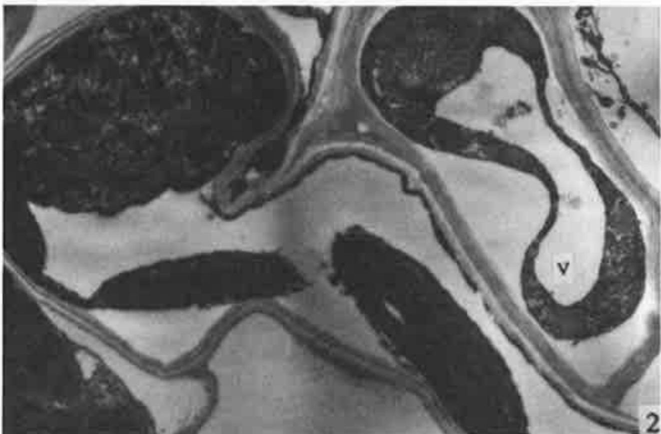
**Effects of space-induced variations in maize (*Zea mays* L.). II Influence of space conditions on ultrastructure of leaves**

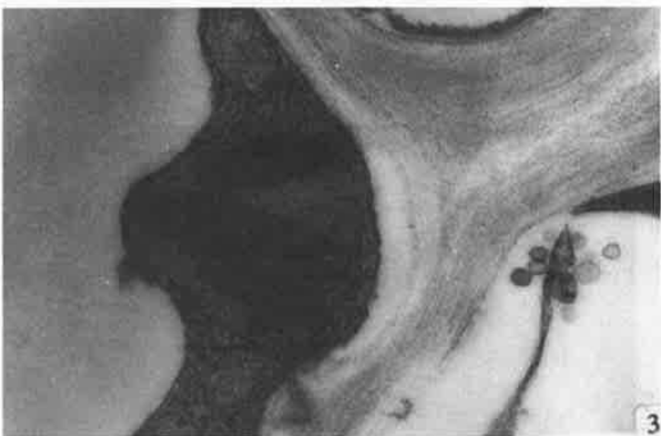
--Li, S, Zeng, M, Liu, M, Liu, Y

Seven maize inbred lines, Yi 0-4-1, Yizi24, Yi141, Yt183, Yt185, 311 and 8112, were used as experimental materials. Dried seeds of maize were carried by recoverable satellite under space conditions of 175 km to 253 km from sea level. Various changes of maize leaves were observed, including young plants, plasmolysis, expansion of vacuoles and cell walls, proliferation of plasmodesmata, variations of grana disc and stroma thylakoids, breadth of nuclear membranes, increase in number and enlargement of endoplasm reticulums and variations of othe endomembranes.(see Plates I-II).







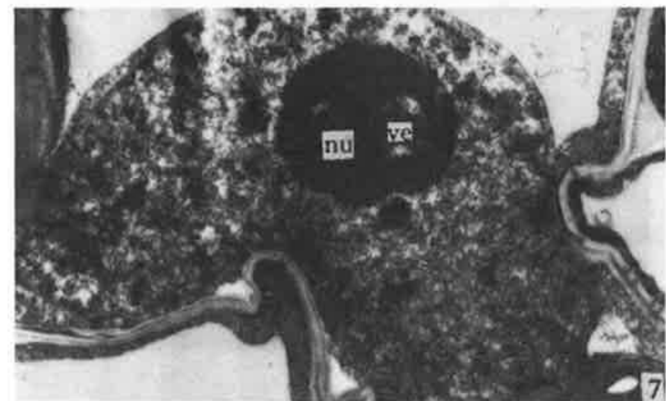
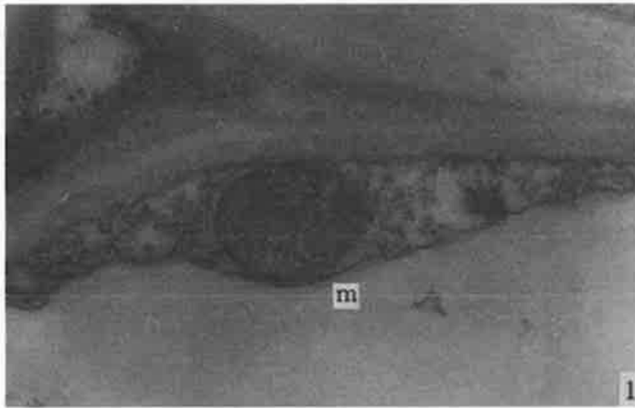
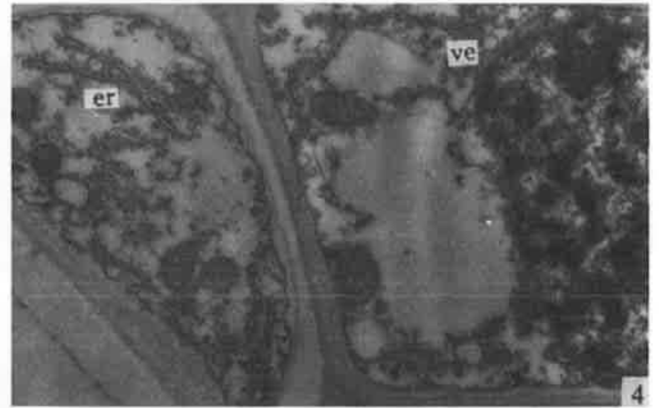
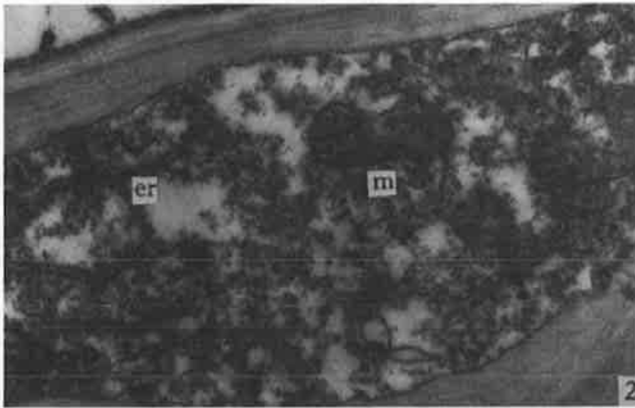
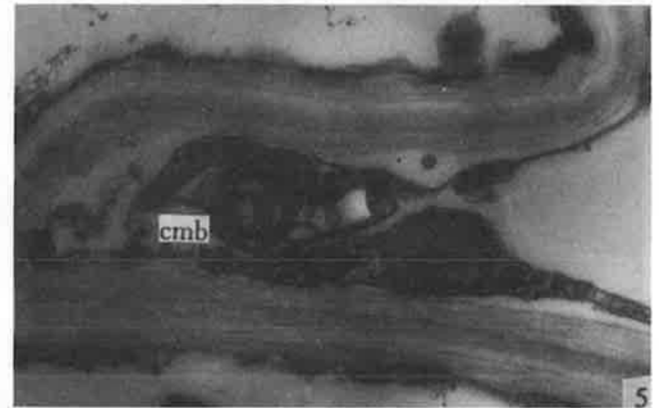



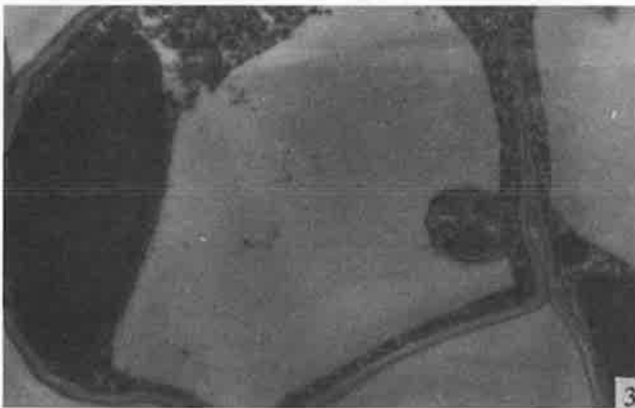

Plate I. 1, Electron micrograph of corn leaf cells from ground control (X10000). 2-7, Electron micrographs of corn leaf cells after space flight. 2, Plasmolysis, cell walls out of shape and big central vacuoles (X9900). 3, Thickened cell walls and 3 uncertain objects in a cell (X26000). 4, Rich plasmodesmata between cells, some mitochondria around the plasmodesmata and plasmodesmata swollen in one end (arrow) (X30000). 5, Plasmodesmata branched in one end (X30000). 6, Changes of nucleus shape and concentric membrane bodies (X30000). 7, Breach of nuclear membrane and nucleolus vacuoles (X12450).











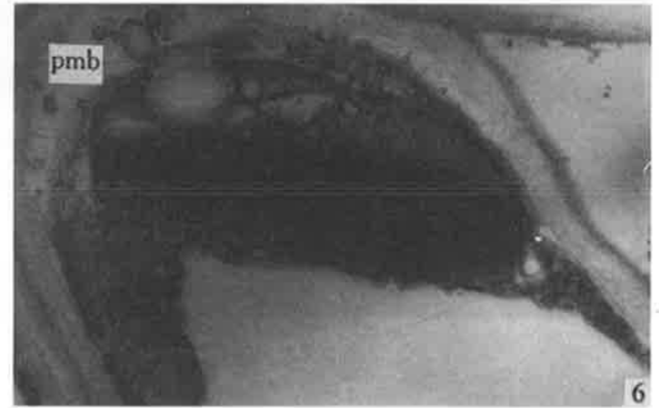



Plate II. 1, Mitochondria from ground control cells (X49500). 2-6, Electron micrographs of corn leaf cells after space flight. 2, Swollen endoplasmic reticulums and dividing mitochondria (X30000). 3, Curly endomembrane and concentric membrane body (X12450). 4, Swollen and circular endoplasmic reticulums. Note some vesicles nearby (X1500). 5, Concentric membrane body (X39000). 6, Paramutal body (39000).

**Genetic analysis of QPM**

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

In our previous paper (MNL, 69: 17-18), we described analyses of the endosperm lysine accumulation controlled by the *o2* gene and breeding for High Quality Protein Maize (QPM). The purpose of this paper is to present the results of genetic analysis on QPM and its controls (soft endosperm). Three QPM inbred lines (T.T/02, Yi041/02 and Yi140/02), two normal opaque-2 inbred lines (Yi040, opaque-2) and ten normal maize inbred lines (I1278C, Mo17, Zi24, 525, Dwarf C103, Hai7-1, Dwarf 15-1, Duo 229, Bolicui and Ji B) were used as experimental materials. The results

Table 1 Grain separation rate of the cross progeny ($F_2$, $BC_1$) in QPM and normal maize

| Cross combination and treatment | Number of the grain | | | Expect rate | $x^2$ | P |
|---|---|---|---|---|---|---|
| | Total | Transparent | Semi-opague or opague | | | |
| (I1278C × T.T/02)$F_2$ | 297 | 229 | 68 | 3 : 1 | 1.2468 | 0.30-0.20 |
| (I1278C × T.T/02)$BC_1$* | 393 | 202 | 191 | 1 : 1 | 0.3664 | 0.60-0.50 |
| (MO17 × T.T/02)$F_2$ | 306 | 235 | 71 | 3 : 1 | 0.8504 | 0.50-0.40 |
| (MO17 × T.T/02)$BC_1$ | 385 | 194 | 191 | 1 : 1 | 0.0936 | 0.80-0.70 |
| (T.T/02 × Zi24)$F_2$ | 300 | 206 | 94 | 3 : 1 | 0.7350 | 0.50-0.40 |
| (T.T/02 × Zi24)$BC_1$ | 406 | 208 | 198 | 1 : 1 | 0.3547 | 0.60-0.50 |
| (525 × Yi041/02) $F_2$ | 589 | 452 | 137 | 3 : 1 | 1.0969 | 0.30-0.20 |
| (525 × Yi041/02) $BC_1$ | 397 | 204 | 193 | 1 : 1 | 0.4937 | 0.50 |
| (DwarfC103 × Yi041/02) $F_2$ | 508 | 387 | 121 | 3 : 1 | 0.8504 | 0.40-0.30 |
| (DwarfC103 × Yi041/02) $BC_1$ | 391 | 200 | 191 | 1 : 1 | 0.3683 | 0.60-0.50 |
| (Yi041/02 × Hai $_{7-1}$) $F_2$ | 605 | 458 | 147 | 3 : 1 | 0.2207 | 0.70-0.60 |
| (Yi041/02 × Hai $_{7-1}$) $BC_1$ | 399 | 206 | 193 | 1 : 1 | 0.4912 | 0.50 |
| (Yi040/02 × Dwarf $_{15-1}$) $F_2$ | 603 | 454 | 157 | 3 : 1 | 0.5654 | 0.50-0.40 |
| (Yi040/02 × Dwarf $_{15-1}$) $BC_1$ | 405 | 206 | 199 | 1 : 1 | 0.3545 | 0.60-0.50 |
| (Duo 229 × Yi140/02) $F_2$ | 587 | 446 | 141 | 3 : 1 | 0.2672 | 0.60-0.50 |
| (Duo 229 × Yi140/02) $BC_1$ | 404 | 206 | 198 | 1 : 1 | 0.2475 | 0.70-0.60 |
| (Bolicui × opague-2) $F_2$ | 308 | 234 | 74 | 3 : 1 | 0.2771 | 0.70-0.60 |
| (Bolicui × opague-2) $BC_1$ | 389 | 198 | 191 | 1 : 1 | 0.2571 | 0.70-0.60 |
| (Ji B × opague-2) $F_2$ | 298 | 228 | 70 | 3 : 1 | 0.4454 | 0.60-0.50 |
| (Ji B × opague-2) $BC_1$ | 397 | 202 | 195 | 1 : 1 | 0.2519 | 0.70-0.60 |

* Used o2 line as backcross parent

showed that the *o2* gene of QPM is a monogenic recessive gene the same as normal opaque-2 (see Table 1). It controls the accumulation of increased lysine in endosperms, tassels and leaves at the seedling stage. The chromatogram of two-dimensional electrophoresis indicated the effects of the *o2* gene and its modifiers on the synthesis of zeins. In Mo17 and Mo17/02 endosperms the synthesis of 27KD, 22KD, 20KD and 15 KD Zein was severely suppressed. In QPM and opaque-2 endosperms little difference existed.

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**Genetic relationship among maize genotypes based on embryo protein markers**

--Mladenovic-Drinic, S, Demic, G, Konstantinov, K

By developing molecular genetics methods, based on PCR technology or not, the genome could be dissected at the level of DNA and different types of DNA molecular markers are available for comprehensive evaluation of the relationship among maize (*Zea mays* L.) inbred lines, maize hybrids and parental inbred lines at the level of DNA. Biochemical processes leading to nuclear gene expression in eukaryotic cells are going on at the level of chromatin. At this stage nuclear DNA and other nuclear components are organised in the nucleus in a highly ordered way to achieve a perfect synchronisation and accuracy of the various structural/functional processes occurring in a differentiated cell. DNA markers are good tools for the determination of genotype polymorphism at the level of DNA and identification of genotypes based on DNA polymorphism. The question is which part of DNA polymorphism, determined by DNA markers, corresponds to functional genome polymorphism. A good example is when several physiologically different traits of plant development are mapped by one and the same molecular probe or when one and the same developmental trait is mapped to several widely distant loci (Khavkin and Coe, MNL70:42-45, 1996) and especially in the case when protein overexpression is posttranscriptionally regulated. A particularly important question arises when investigation of the biochemical background of the heterotic effect is under way. Somatic cells of dry embryo tissue after inbred line crossing, as well as all somatic cells during development of the F1 plant, carry chromatin of both parental lines. The highest heterotic effect is expressed in the somatic tissue of the F1 plant generation. Therefore we set up an experiment to answer the question: Could dry embryo salt soluble proteins be informative genetic markers in the investigation of ge-

netic distance, genotype identification and genetic background of heterotic effect as well? In this paper we are presenting results on genetic relationships among inbred lines based on embryo salt soluble proteins.

Analysed maize genotypes, including both Corn Belt public lines and inbreds created at the Maize Research Institute "Zemun Polje" (MRI) are listed in Table 1.

Table 1. Genetic background of analysed inbred lines

| | |
|---|---|
| B73 | BSSS C5 |
| B73 Sbms | B73 rec. |
| B73M | B73 rec. |
| B84 | BSSS C7 |
| ZPL326 | BSSS, MRI developed germplasm |
| ZPL219 | A662, B73 |
| A632 | (Mt42xB14) $B14^3$ |
| ZPL362 | A619, A632 |
| ZPL2217 | BSSS |
| ZPL385 | MRI developed germplasm with exotic component |
| Mo17 | CI 187-2 x C103 |
| ZPL412 | Yu pop x Mo17 |
| ZPL395 | (Yu pop x Mo17)Mo17 |
| F2 | Pop Lacaune |
| F7 | Pop Lacaune |

Salt soluble proteins were isolated and analysed as reported previously (Drinic et al. MNL 70:71-72, 1996; Konstantinov et al. MNL70:72-73, 1996; Mladenovic-Drinic and Konstantinov, In "Genetics and exploitation of heterosis in crops", Int. Symp. Mexico City, Mexico, 1997, pp. 94 - 95). Association among 15 inbred lines analysed has been determined from cluster analysis based on protein marker data. The UPGMA clustering method was used for hierarchical clustering and the necessary computations were performed using the NTSYS-pc program.

The protein based dendrogram for 15 analysed inbred lines, consisting of three major groups, is presented in Fig. 1. The first group (Cluster 1) consists of inbred lines derived from or related to BSSS germplasm which clustered in the protein study; the second group (Cluster 2) belonging to Lancaster germplasm inbreds clustered in the protein study; and the third group (Cluster 3) is two public French inbred lines, F2 and F7 (Fig. 1).

It is obvious from subdivisions that further break outs are very largely according to pedigree background. Within the BSSS group, B73 and B84, derived from advanced cycles of the recurrent selection program of Iowa Stiff Stalk Synthetic, clustered separately from sterile versions of B73 genotype (B73 Sbms) and B73M. The third subdivision consists of A632 with ZPL362. Inbreds ZPL326 and ZPL2217 were loosely aggregated with B73 and A632 related lines. Within the Lancaster group of inbreds association in two subdivisions of related lines has been obtained; lines ZPL385 and Mo17 clustering together in one subgroup, while ZPL395 and its progenitor ZPL412 were in another.

Grouping of inbreds revealed by the present analysis generally agreed with the pedigrees of these lines. There is a major split between Stiff Stalk and non-Stiff stalk pedigreed inbreds. Therefore results presented support our suggestion that embryo salt soluble proteins provided association of inbred lines that largely concur with expectations based upon pedigree data, and are good candidates for discrimination between maize genotypes.

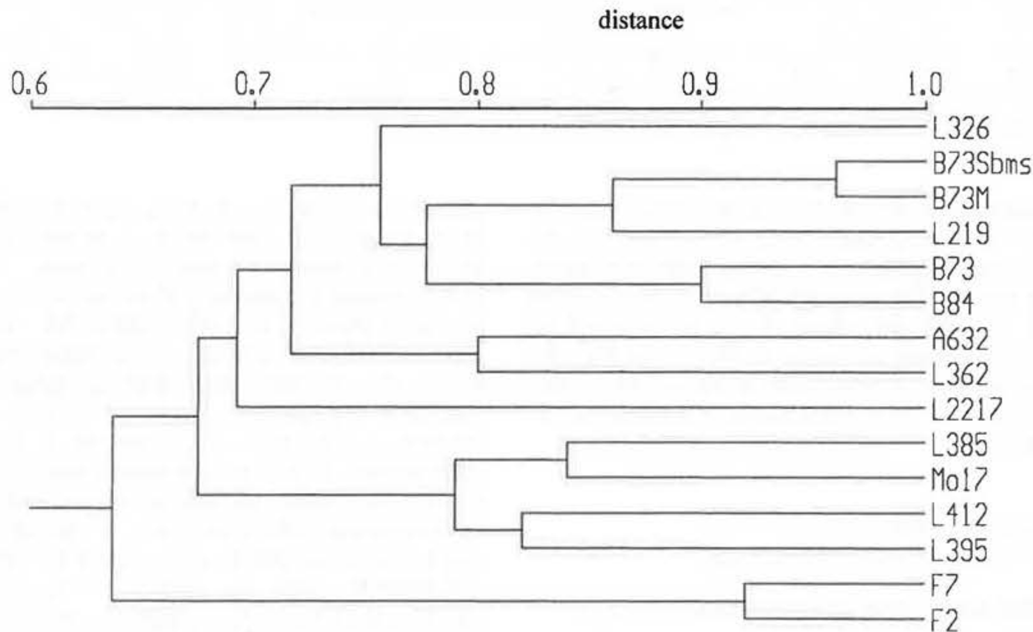


Figure 1. Dendrogram for fifteen maize inbred lines based on cluster analysis (UPGMA) of genetic distances from embryo salt soluble protein data.

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Istituto Sperimentale per la Cerealicoltura

**Polymorphism, distribution, and segregation of *Eco*RI and *Pst*I based AFLP markers in a molecular linkage map in maize**

--Ajmone Marsan, P, Castiglioni, P, Ferrarini, M, van Wiik[1], R, Motto, M

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Extensive genome mapping based on DNA restriction fragment length polymorphism (RFLP) markers has been accomplished in many crop species. These maps and their associated technology have been used successfully for a number of applications in genetic research and breeding, including gene tagging, evolutionary studies, marker-aided selection, and analysis of quantitative trait loci (QTLs). However, these maps and their associated QTL analyses are expensive and time-consuming technologies and may not provide detailed coverage throughout the genome.

The development of the polymerase chain reaction (PCR) has expanded the repertoire and efficiency of DNA marker systems, which include the AFLP method (Vos et al., Nucleic Acids Res. 23: 4407-4414, 1995). The advantage of AFLP assay over other DNA marker techniques includes the detection of a large number of polymorphisms from a single PCR reaction, within a very short period of time, and the requirement for small amounts of DNA, thus reducing expenses and expediting the construction of high-density linkage maps. Accordingly, with the aim of exploiting AFLP markers in a maize genome mapping program, we used the AFLP technique in order to generate and map AFLP markers using an F2 mapping population, and to investigate their inheritance and distribution associated with the use of enzyme combinations differing in sensitivity to DNA methylation.

Two-hundred-twenty-nine F3 progenies, each tracing back to an individual F2 plant, derived by crossing the maize inbred lines B73 and A7, were used. This population has been described previously to construct an RFLP linkage map (Ajmone-Marsan et al., Theor. Appl. Genet. 90:415-424, 1995). The protocol adopted for the generation of AFLP markers was essentially the same as that described by Vos et al. (1995). DNA isolated from each F3 family was digested with an *Eco*RI/*Mse*I or *Pst*I/*Mse*I enzyme combination (ECs).

In this study we were able to detect 1568 visible bands and map 246 AFLP markers covering 2057 cM. Five gaps larger than 30 cM remained. Therefore, the efficiency of generating AFLP markers appears substantially higher relative to RFLP mapping in the same population, and the speed at which they can be generated shows a great potential for application in marker-assisted breeding. The appropriate selection of primer combinations (PC) that generate high levels of polymorphism with markers well distributed over the genome plays a crucial role. We have also observed that some primer combinations produced as many as 19 polymorphic markers distributed over as many as 9 chromosomes.

The majority of AFLP markers (89.1%) followed Mendelian segregation. They showed allelic frequencies in agreement with expectation, and were unambiguously placed on the linkage groups (72.4%). The addition of a large number of AFLP markers to the map did not disturb the original order and relative distance of the previously mapped RFLP markers. In the experiment here reported, the assay of a relatively large number of mapping progenies, the high level of informativeness of codominant scored AFLP markers and the rejection of markers with unexpected behaviour, have probably minimised the map inflation; typing errors have been credited to be in part responsible for map extension.

By adding AFLP markers, we generated a map which is 440 cM longer than the map generated with RFLP markers alone. The increase of the total map length was mainly caused by the addition of markers to telomeric regions, where RFLP markers were poorly represented. The current study indicated that *Pst*I/*Mse*I PCs were more efficient in detecting polymorphism than *Eco*RI/*Mse*I primers. In addition, *Pst*I AFLP markers are more randomly distributed across chromosomes and chromosome regions, while *Eco*RI AFLP markers clustered mainly on centromeric regions and on chromosome 1. Specific regions were observed, in which only markers produced with either *Pst*I/*Mse*I or *Eco*RI/*Mse*I restriction enzyme combination were located (i.e. 1S, 2S, 5L, 7S and 7L). As the amplification products generated by the *Eco*RI/*Mse*I AFLP technique may contain repetitive sequences, there is a higher probability of identifying *Eco*RI/*Mse*I AFLP markers than *Pst*I/*Mse*I AFLP markers and RFLPs in highly repetitive regions near the centromeres. This may be a plausible explanation for the stronger clustering of *Eco*RI-based AFLP markers in the centromeres.

The more random distribution of *Pst*I-based AFLP markers on the genetic map reported here may reflect a preferential localisation of the markers in the hypomethylated telomeric regions of the chromosomes. There is considerable evidence that hypomethylated regions of the maize genome are associated with genes (Bennetzen et al., Genome 37:565-576, 1994) and that recombination occurs primarily within genes, or perhaps unique sequences, and rarely in intergenic regions (Dooner and Martinez-Ferez, The Plant Cell 9:1633-1646, 1997, and references therein).

**Role of Opaque-2 in the shikimate pathway in maize endosperm**

--Maddaloni, M*, Donini, G, Thompson, R[1], Motto, M

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During our research programs devoted to understanding the regulatory mechanisms responsible for zein gene expression in maize endosperms, we have shown that the role of transcriptional activator Opaque-2 (O2) is not only confined in controlling the expression of the 22-kDa zein gene family (Motto et al., in B.A. Larkins and I.K. Vasil eds., Cellular and Molecular Biology of Plant Seed Development, pp. 479-522, 1997). In fact, the O2 protein can transiently activate the expression of its own promoter (Lohmer et al., EMBO J. 10:617-624, 1991) and regulates directly or indirectly a number of other, non storage protein genes. These include *b-32*, encoding a type I ribosome-inactivating protein (Maddaloni et al., J. Genet & Breed. 45: 377-380, 1991), and *b-70*, encoding a heat shock protein 70 analogue, possibly acting as a chaperonin during protein body formation (Marocco et al., Plant Cell 3:507, 515, 1991). O2 also regulates the enzyme levels of LKR (lysine-ketoglutarate reductase) (Brochetto-Braga et al., Plant Physiol. 98:1139-1147, 1992), ASK1 (aspartate kinase1) (Azevedo et al., Plant Physiol. 108:103, 1995), and cyPPDK1 (cytosolic orthophosphate dikinase1) (Maddaloni et al., Mol. Gen. Genet. 250:647-654, 1996).

To assess the degrees to which changes in transketolase

(TKS) protein synthesis in maize endosperms are reflected at the mRNA level, the relative abundance of TKS transcripts was examined by Northern blot analysis. Poly(A)+ mRNA was isolated from wild-type and *o2* endosperms harvested at 15 days after pollination, size fractionated and immobilized onto membrane filters. The filters were hybridized and washed under stringent conditions with cDNA probes for the Tks gene from sorghum, provided by P. Westhoff (Heinrich-Heine Universität, Dusseldof, Germany). The results clearly demonstrated that TKS mRNA is more abundant (4-5 fold) in wild-type endosperms than in the *o2* endosperms. The O2 protein is, therefore, indeed involved in the regulation of TKS gene expression in maize endosperm. The same filters were checked for uniformity of loading by rehybridizing with a specific probe for the ubiquitin gene which, in our hands, is not affected by the *o2* mutation. This observation is of particular interest due to the fact that PPDK and TSK enzymatic activities are involved in the synthesis of phosphoenolpyruvate and erythrose 4-phosphate, respectively; these metabolites are the direct precursors of heptulosonate 7-phosphate, the first compound in the shikimate pathway. This pathway is currently receiving a great deal of interest due to its cardinal importance in the metabolic fluxes in the plant (reviewed in Weaver and Herrmann, Trends in Plant Science 9:346-3351, 1997). Similarly to PPDK, TSK mRNA is also modulated by the nitrogen compounds present in the growing media.

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### *Mu* suppressible *Liguleless3* alleles produce altered transcripts

--Girard, L, Freeling, M

*Lg3-0* is a semi-dominant, neomorphic mutation that transforms regions of leaf blade, auricle and ligule into sheath. The *Liguleless3* gene is a member of the *knox* class 1 family of homeobox genes and the dominant alleles which define it are due to ectopic expression of the gene in the leaf (Muehlbauer et al., Plant Physiology in press).

Screens for *Mutator* induced revertants of *Lg3-0* resulted in the isolation of numerous partial and complete revertants in which the *Lg3-0* phenotype is either reduced in severity or eliminated entirely. A subset of these revertant alleles were determined to be *Mu* suppressible. *Mu* suppression is when the phenotype caused by the *Mu* insertion is dependent on *Mu* activity such that in the absence of *Mu* activity, the phenotype reverts to that of the progenitor. In this context, when *Mu* is active the *Lg3* suppressible alleles appear wild type and when *Mu* is inactive the plants appear mutant.

The *Lg3-0r331*, *Lg3-0r422*, and *Lg3-0r1021* alleles are *Mu* suppressible revertants of *Lg3-0*. Each is caused by the insertion of a *Mu* element into the same site in the 5'UTR. We used Northern analysis to make an initial assessment of how these alleles might deal with a large, cumbersome insertion into their transcribed region. We found that the transcripts produced by these alleles are significantly shorter than those of wild type as well as its progenitor, *Lg3-0*.

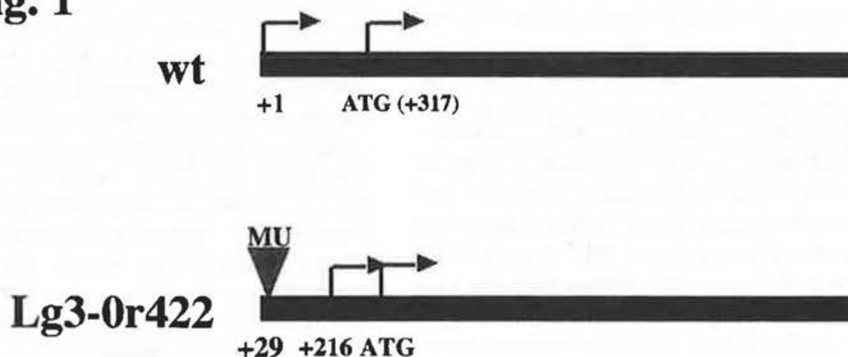
In order to characterize these aberrant transcripts further, we used RACE (Rapid Amplification of cDNA Ends ) to clone the cDNA corresponding to the *Lg3-0r422* transcript. We found that the transcripts produced by this allele are being initiated approximately 187 base pairs downstream relative to wild type (Fig. 1).

The *Lg3-0r331*, *Lg3-0r422*, and *Lg3-0r1021* alleles produce a transcript that is much smaller than that of wild type or its progenitor. Since all three of these alleles are *Mu* suppressible, we initially thought that they might be behaving in a manner similar to the suppressible *hcf106*, in which a *Mu* element inserted into the 5'UTR functions as an outward reading promoter. Instead, characterization of the *Lg3-0r422* cDNA revealed that in this allele, transcription is initiated much further downstream than the site of *Mu* insertion, as if the element were able to redirect the start of transcription. One possibility is that the insertion causes the adoption of a secondary structure in the region which is prohibitive to transcription initiation at the correct site.

### Dosage analysis of the *Vestigial glume1-R* phenotype

--Jesaitis, L, Freeling, M

The *Vestigial glume1-R (Vg1-R)* dominant mutant exhibits several reduced structures including the ligule, sheath margin, and tassel and ear glumes. Our previous studies (unpublished) suggest that the affected structures develop initially normally and that aberrant cell death, occurring late in development, is involved in tissue diminution. To gain insight into the mechanism by which cells die in *Vg1-R* plants, we examined the effect of wild type gene dosage (*vg1+*) on the mutant phenotype. We generated a wild type gene dosage series by crossing pollen containing the B-A translocation TB-1La to *Vg1-R*/+ ears. TB-1La carried *vg1+* as well as the enzymatic marker *Adh1-C* (generously provided by Dr.



James Birchler). *Vg1-R* females carried *Adh1-F*. Progeny genotypes were determined by starch gel analysis of ADH activity from scutellum. Hypoploids were identified by a single *Adh1-F* band, euploids and hyperploids by three bands, *Adh-F* and *C* homodimers and an F/C heterodimer. The relative ratios of homo to heterodimer bands allowed us to distinguish euploids from hyperploids.

No difference in mutant phenotype severity was observed between the three classes. Ligule, sheath margin, and male and female glumes were reduced to the same extent in hypoploids, euploids, and hyperploids. If the *Vg1-R* phenotype resulted from a loss of function in a gene for which two functional copies are required, we would expect to observe a normal phenotype in plants containing two copies of *vg1+* in addition to the mutant *Vg1-R* allele. This was not the case, suggesting that cell death in *Vg1-R* plants results from a gain in gene function. Further support for this conclusion comes from the observation that wild type hypoploid siblings containing only one functional *vg1+* gene did not display the *Vg1-R* phenotype. Cell death in the mutant is not attributable to overproduction of the wild type gene product since we did not detect an increase in mutant phenotype severity with increasing wild type gene dosage. Increasing *vg1+* dose also didn't mitigate the mutant phenotype, suggesting that *Vg1-R* does not interfere with normal wild type gene function. The lack of difference observed between the three ploidy classes suggests that the *Vg1-R* phenotype is insensitive to the dose of the wild type allele and thus results from either altered gene function or expression pattern.

### Characterization of epidermal-pattern mutants: macrohairless

--Nelson, J, Freeling, M

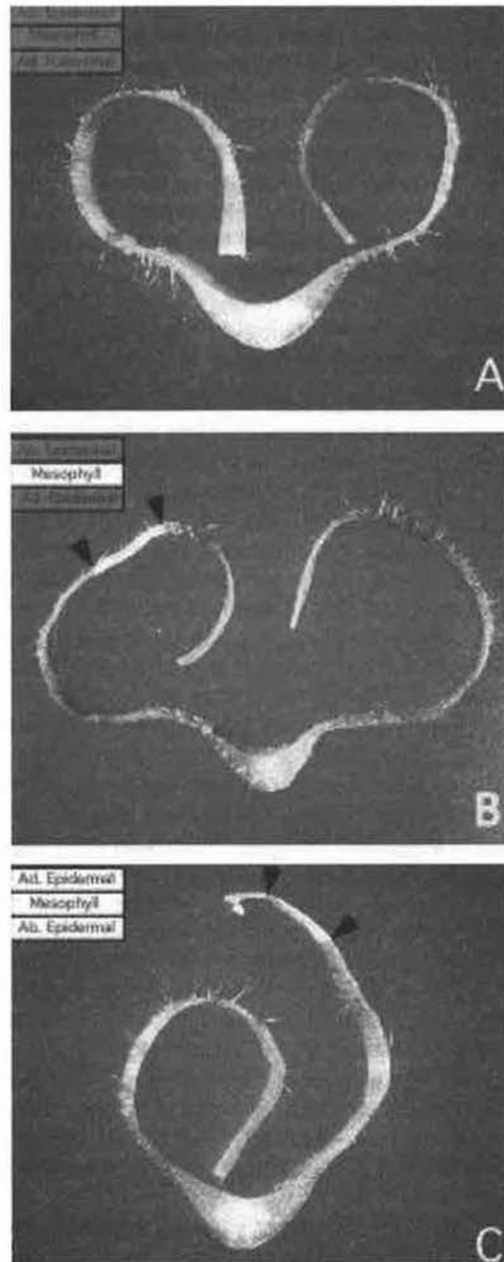
Macrohairless (*mhl1-R*) is an epidermal-patterning mutant first identified in a *Mu*-active background. The phenotype appears to be controlled by a single recessive gene. The original mutant displayed "complete" loss of macrohairs on the adaxial epidermal surface. After three generations of introgression to W22 and W23, *mhl* shows 100% penetrance yet reduced expressivity. The introgressed mutant shows a partial loss of macrohairs. In each of the inbreds there was a varying degree of macrohair loss. In a family segregating for *mhl/mhl* and *mhl/+* there was an average of 1.7 macrohairs/$cm^2$ in the macrohairless plants as compared to an average of 37.7 macrohairs/$cm^2$ in the heterozygous normal siblings.

Macrohairless has been preliminarily mapped to 4L. Homozygous macrohairless mutants were outcrossed by a series of B-A translocations covering 18 of the 20 arms. Crosses by TB-4L uncovered the partial loss of macrohair phenotype associated with the introgressed mutant. None of the B-A translocations uncovered a phenotype displaying a complete loss of macrohairs. This mapping result will be confirmed by growouts of the above *mhl/* TB-4L test crossed to *mhl/mhl*.

### Genetic and histological analysis of Rolled

--Nelson, J, Lane, B, Freeling, M

Rolled (*Rld*) is a semi-dominant mutation in which dorsiventral (adaxial-abaxial) polarity is switched with the morphological outcome of leaves which roll inward, toward the midvein. These adaxial-abaxial switches involve the epidermal and ground tissues, excluding the vascular bundles which retain wildtype polarity. The




Mosaic Analysis. Transverse sections of leaves of *wlu4, rld1+/Wlu+, Rld1-O* genotype in A, B and C. White sectors indicate removal of the dominant mutant allele *Rld1-O* from partiicular tissue layers of leaf. A) Leaf with no white sectors. Inset tissue-layer drawing shows this. Note the inversion of the abaxial and adaxial epidermal layers, characteristic of *Rld* mutants. B) White sector, indicated by arrow-heads, marks the loss of *Rld1-O*. Close examination showed epidermis guard cells were still green, as shown by inset. This sector had typical *Rld1-O* polarity. C) *Rld1-O*.

extent of the epidermal phenotype includes presence of ligule tissue on the abaxial blade/sheath boundary, switching of abaxial and adaxial epidermal cell types: the expression of characteristically adaxial cell types such as macrohairs and bulliform cells on the abaxial surface, and the lack of them on the adaxial surface. The internal phenes of the Rolled mutation include abnormal patterning of the hypodermal schlerenchyma which might create or reinforce a structural imbalance resulting in the curvature of the leaf blade.

In addition, a disruption in the formation of transverse and intermediate veins in regions of the blade and sheath is frequently seen; this results in a pale stripe at the blade/sheath boundary.

Mosaic analysis was done to identify the focus of action of the mutant gene product ROLLED. We used the *Rld1-0* allele. This mosaic analysis of ROLLED was set up by crossing pollen from *white luteus4* (*wlu4*) heterozygotes onto *Rld*/+ ears. The progeny of this cross were imbibed and then exposed to x-irradiation. This generated white wild-type sectors (*wlu*,+/--) in green Rolled (*wlu*,+/+,*Rld*) background. Preliminary results of mosaic mapping suggest that ROLLED acting in the epidermis only is sufficient to switch the dorsiventral polarity.

**New mutants with aberrant ligule/auricle formation**
--Kloeckener-Gruissem, B, Freeling, M

The maize leaf develops three morphologically distinct domains: i. the sheath which wraps around the stem, ii. the blade which angles off the stem and serves as the major photosynthetically active region and iii. the region that separates those two domains, occupied by the triangular shaped auricles, which act as a hinge for the blade and the ligule, a fringe-like epidermal structure that grows out of the leaf's plane. So far, two genes have been identified by recessive mutants that play an essential role in the proper development of the ligule/auricle region, namely *liguleless1* (*lg1*) and *liguleless2* (*lg2*). For both genes, many mutant alleles have been isolated. Certainly, other genes must be involved in the development of the ligule/auricle region. Past screens for liguleless mutants focused on the altered, upright stature of the plants, due to the lack of the auricles, and hence it was easy to miss any mutants in which only the ligule, but not the auricle is affected. Therefore we decided to investigate the ligule structure on every plant while screening mutagenized families. Although screening of transposon *Mutator* (*Mu*) families facilitates the isolation of the mutated gene, the frequency with which new *Mu*- induced mutations occur might be about ten fold lower than that of chemically induced mutations. Therefore, we chose to screen ethylmethanesulfonate (EMS) mutagenized material. Using the method of EMS treatment of pollen, Jay Hollick and Vicki Chandler, University of Oregon, Eugene, generated highly mutagenic seed stocks. They generously shared 840 M2 families with us. We grew 30 kernels from each of these families in the summer of 1998 in San Jose, California, and screened for families segregating plants with deficient ligules or auricles.

Here we report the isolation of two novel mutants that seem to affect exclusively the ligule/auricle region. We found one family that segregated (2/15) plants with a phenotype similar to that of *lg1* mutant plants, but also with several important differences. Like *lg1*, mutant plants showed the upright leaf stature, due to the lack of auricles. Closer examination of the ligule/auricle region revealed a novel phenotype. On each leaf, a varying number of small patches of sectors with normal appearing ligules were found. The size of the sector can range from less than one to five millimeters. If a larger sector is located at or near the leaf margin, the development of an auricle can be recognized. Whether the other ligule sectors were also developing auricles requires Scanning Electron Microscopy (SEM) analysis. The exact pattern of the sectors did not appear on sequential leaves, suggesting that they are not of meristematic nature. Another novel aspect of this phenotype is in the location of sector development. Sectors can be found irregularly spaced over a distance of up to 2 cm in the proximo-distal dimension of mature, juvenile leaves. The existence of such broad "line" suggests that the normal function responsible for defining the fine line at which the ligule normally develops is deficient. This is in contrast to revertant sectors of the *lg1-m1* allele (*Ac*-induced) (Moreno et al. 1997, Genes & Development 11, 616.), where all sectors, also irregularly spaced, lie on a sharply drawn line from margin to midrib. The novel liguleless phenotype described here suggests that the mutant might not be another allele of *lg1* or *lg2*. We performed complementation tests by crossing pollen from the new liguleless mutant plants to homozygous *lg1* as well as *lg2* plants. Progeny was scored for a liguleless phenotype at the seedling stage. The *lg2* mutant complemented the new mutant, suggesting that they are not allelic. The progeny of the *lg1* cross did not yield a clear result: 50% of the seedlings were normal while the other half showed the phenotype of the new mutant. These results suggest two points: *lg1* can complement the new mutant but interactions exist between the new mutant and *lg1*. We also examined the progeny of the new mutant crossed to inbred lines and all were normal, suggesting the original mutant plants were not heterozygous for a dominant allele. We are in the process of obtaining progeny from self-pollinations.

We identified another family segregating a ligule phenotype. In 2 out of 15 plants, a ligule formed on the abaxial surface of the leaf. A normal ligule/auricle developed at its normal place on the adaxial side of the leaf. The abnormal, abaxial ligule does not span the entire width of the leaf but is rather restricted to the region at the tip of the triangular-shaped auricle on both sides of the leaf, near the midrib. Just proximal to the ligule fringe, reaching a few millimeters into the sheath, one can recognize a small region of cells of shiny appearance, which is clearly distinct from the neighboring sheath cells. Similarly, distal to the fringe there extends a sector of distinct appearance into the blade. The length of such sectors varies over 5 to 10 cm. Whether such sectors share characteristic features of normal auricle, blade, or sheath will be determined by SEM analysis. Possibly this mutant is deficient in the identification of polarity with respect to the ab- and ad-axial side of the leaf as well as the proximo-distal dimension. The formation of this abaxial ligule is sensitive to the development of the plant: only leaves 5 through 9 are affected.

We are continuing the analysis of both mutants by introgression into various genetic backgrounds as well as the construction of double mutants with other mutants that affect the ligule/auricle region.

**The toxicity of MuDR in *E. coli* is mediated by the dnaY gene**
--Walker, N, Freeling, M

The dnaY gene in *E. coli* was originally identified in a screen for mutants defective in DNA replication. It has since been shown (Garcia et al., Cell 45:453-9, 1986) that this gene encodes the cognate tRNA for the AGA and AGG arginine codons. It was therefore renamed ArgU. The ArgU tRNA is present at very low levels, and is in fact one of the rarest tRNA's in *E. coli*. Brinkmann et al. (Gene 85:109-14, 1989) found that attempts to express heterologous genes containing AGA or AGG codons led to plasmid instability and decreased cell viability. Spanjaard et al. (Nucleic Acids Res. 18:5031-6, 1990) demonstrated that tandem AGA or AGG arginine codons caused frameshifts in the expressed protein when present in an mRNA. This "toxicity", and these frameshifts, could be alleviated by the overexpression of the ArgU gene.

*MuDR*, the autonomous element of the Mutator family of trans-

## murA coding region

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ATG GAC TTG ACG CCC AGT TTC AAT TCG CTA GAC TCC AAC GGC ATT CCC AAC TCC CCC GAT GTA GAT CCG GCA TTG
GGC GAA ACA GGT GGC AGT GAG GGA CTT CAG AAG ATT GAT GGG GAA TCA CAA CTG GAC TGG GAT TCG ATT ATA GTA
TCA GAT GTA TTG GAT GAT GAA GGC AGA GTA CAA GTA CCT ACC GAA AAT GAG ATA TAT TTT AAT CTT GGA CTC AAT
AAA GGG GAT GAG GCT GCC AAT AAT AGG TTT TCT GGC AGT GGT ACA AAT TGT CAT GCA CAG GGA AGT TTG GAT ACG
GAC AAC GAA GAT CAC CAT GCT GAT CAG CCT TGT CAA GAC TAC ATT CCA GAT GAA AAG AGG GTG GTG TAT AAT AGG
ATG AAT CCT TCT ATG CAG CCA GGT TGT TTG TTT CCT AAC ATG AAA GAA TTT AGG ATT GCT ATG CGA CAG TAT GCA
ATA AAA CAT GAG TTC GAG CTT GGA ATT GAA GTT ACT TCG ACA ACA AGA TAC GTT GGA TAC TGT AAG GGT GGT GAT
TGC CCG TGG AGG ATC TAT GCA CGT GAA GAG AAG AAA GGA TTG CCT ACT ATT GTG GTA GCT GTA CTA GAT GAT GTT
                            triplet
CAC ACT TGC ACA TCT AGT GGA AGG AGG AGG ACT ACT ACG CCA ACT TGT GGT TGG GTC GCA TTC CAC GCT AAA CCC
TTG CTC ATG AAG AAA CCA CAA ATG GGT GCT AAA GAG TTA CAA CAA ACA CTA CAG ACA ACT CAT AAT GTC ACT ATT
                                                                                    HindIII
GGG TAT GAT ACA GTT TGG AAA GGG AAA GAG AAG GCT TTG AGA GAG CTG TAT GGA TCT TGG GAG GAA AGC TTC CAG
CTC TTG TAC TCT TGG AAG GAG GCT GTA ATT GCA GTC ATG CCC GAT AGT GTG ATT GAG ATT GAT GTT ATT TTG GAA
GAT GGG AAG TAC TAT TTT AGT CGA TTC TTT TGT GCC TTT GGT CCA TGC ATA TCT GGG TTC CGA GAT GGG TGC AGA
CCT TAT CTT AGT GTG GAC TCG ACA GCA TTG AAC GGT AGA TGG AAC GGA CAT CTT GCA TCT GCT ACT GGT GTA GAT
GGC CAC AAT TGG ATG TAC CCA GTA TGT TTT GGT TTT TTC CAA GCT GAG ACA GTT GAC AAT TGG ATT TGG TTC ATG
AAA CAG CTC AAA AAG GTT GTG GGT GAC ATG ACA CTA CTT GCT ATA TGT TCA GAT GCA CAA AAA GGG CTG ATG CAT
                                        A
GCT GTC AAT GAG GTA TTT CCG TAT GCT GAG AGA AGA GAA TGC TTC AGA CAC TTA ATG GGT AAC TAT GTG AAA CAC
                                                        B
CAT GCT GGG TCA GAG CAC ATG TAT CCA GCA GCA AGG GCC TAT AGG AGA GAT GTA TTT GAA CAC CAT GTT AGC AAG
GTC AGA AAT GTT CAC AAG ATT GCT GAG TAC TTA GAC CAA CAC CAC AAA TTC CTT TGG TAC AGG AGT GGT TTC AAC
                                                                                           BglII
AAA GAT ATC AAA TGT GAT TAC ATC ACA AAT AAC ATG GCT GAG GTT TAT AAT AAC TGG GTT AAA GAC CAC AAA GAT
CTT CCT GTG TGT GAT TTG GCT GAG AAA ATT AGG GAG ATG ACA ATG GAA CTG TTT CAT CGT AGG CGA AGG ATT GGT
    HindIII
CAT AAG CTT CAT GGT ATT ATT TTG CCA TCT GTC TTA GCG ATA CTA AAG GCT CGG ACT AGA GGG TTG GGC CAC TTG
TCC ATT GTA AAA TGT GAC AAC TAC ATG GCA GAG GTA CGA GAC AGC ACT AAT TGT ATG ACT AAA CAT GTC GTG AAT
                                                                                            IleX
GCA GAA CTG AAA CAG TGT TCT TGT GAG GAA TGG CAA CAC ACT GGG AAA CCG TGT CAA CAT GGT CTA GCC CTA ATT
ATT GCC CAA GAT TCC AGA GAT GTA GGT ATG GAA AAT TTT GTT GAC GAT TAT TAC TCT ACT GAA AGA TTC AAG ATA
        XbaI C
GCA TAT TCT AGA AGG GTG GAA CCA ATT GGT GAT CGT TCG TTT TGG CCA TCA GTT GAT TTC GCC AGT GGA GTG TTT
                D                                                           XhoI
GCA CCA ATA GCT AGA AGA GGT CTT GGA AGA CAA CGA AAA AAT AGA ATT AAA AGC TGT CTC GAG GGT GGG AGT GCT
AGA AAT AAA AGT ACC AAC GAA AAT GAG AAA ACG AAA AAG CGA CTC AAA AGG CAA TAC ACT TGT CCT AAT TGT GGT
GAA TTG GGA CAC CGC CAA TCT AGC TAC AAG TGC CCT TTG AAT GGG ACA AAA AAA AGG AAA AGG AAA CCA CGG ATA
                    BamHI
AAC ACC ACA AAA AAT TGG ATC CCT AAA GAG CTT CGG ACT TCT TCA CAG AAT GTA CCA GTA CAG CCA GAC GTA GCA
GAG GAA GTC ACT GAA CAA GAG CTA GAA GAT CCA CAG CCA GAG ACA GAA CAA TTG GGT CTT GCA CTC TTC CAG CCG
TTG GGT GCA CAA ATC ACT GAA CAA GAG GCC GAT GAA CCA GCC GAG CAA GCT CCA CCT GCT TCT CCA CCA CCG ACA
AGG AAA TGG CTA GTG AAG AAA ATC ACC CCC AAG AAA AGA CTG AGG ATT AGT GCT CAG CAG AAG CAG TAT TAA
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**Fig 1**: murA coding region showing AGA/AGG arginine codons and the ATT isoleucine doublet. The AGG triplet is labled as such, and the AGA/AGG doublets are labled A through D. The ATT doublet is labled IleX. Several restriction sites have been included as landmarks.

posable elements has proven extremely refractory to manipulation in *E. coli* (Gutierrez et al., Genetics 149:329-346, 1998). Examination of the murA gene, the presumptive transposase, revealed a preponderance of AGA and AGG arginine codons. The murA mRNA has more than 40 AGA or AGG codons, including a triplet AGG and 4 doublets (Fig. 1). It also has a doublet of the cognate codon for the rare IleX tRNA. Overexpression of the ArgU tRNA allows the stable maintenance of murA clones in *E. coli*, but it does not allow high-level expression of the gene product. Preliminary mutagenesis experiments to alter the AGA and AGG codons to codons more commonly used in *E. coli* have been successful (Fig. 2).

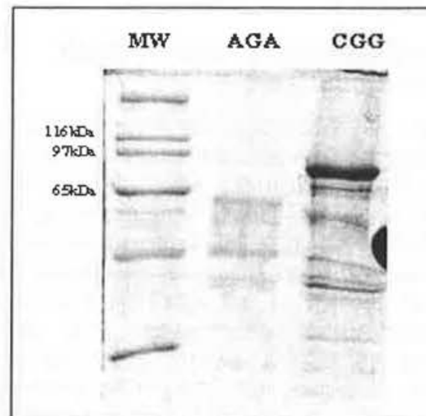


**Fig 2**: 700aa fragment of murA fused to GST and expressed in E.coli. Both lanes where co-transformed with the ArgU tRNA. The construct in lane three has had the AGG triplet (see fig 1) mutagenized to a CGG triplet. The expected size for a full length product is 109kDa, the observed size of ≈75kDa is probably due to termination at the AGA doublet labled A in figure 1 (predicted size 72kDa).

BROOKINGS, SOUTH DAKOTA
South Dakota State University

**Identity and location of *v26***

--Whalen, RH

The recessive mutant *virescent26* was obtained by Neuffer and Beckett (MNL 61:50, 1987), who originally designated it as *v-453A*. They placed it on 2S since it was uncovered by TB-3La-2S(6270) but not by TB-3La.

Linkage tests with other 2S markers were made to map *v26*. The source of our *v26* stock was Coop. 89-582-1. Seedlings are strongly yellowish-white, and green slowly from tip to base without becoming grainy. Culms, leaf sheaths, and husks are whitish, although the MNL gene lists and description in Mutants of Maize (Neuffer, Coe and Wessler, 1997) do not mention this. Some variability in expression was noted among our progenies for these traits at anthesis, however. Viability is normal and ears are obtained if adjacent normal sibs are removed early. The F2 repulsion data were as follows:

| Marker | + + | + m | v + | v m | Total | $\chi^2$ indep. (1 d.f.) |
|---|---|---|---|---|---|---|
| *fl1-o4* | 302 | 132 | 105 | 39 | 578 | 0.576 |
| *gl11* | 195 | 54 | 70 | 13 | 332 | 1.402 |
| *gl2* | 691 | 262 | 283 | 19 | 1255 | 59.317 |
| *lg1* | 496 | 215 | 221 | 3 | 935 | 79.569 |

These data place *v26* about 27 cM distal to *gl2* and 12 cM distal to *lg1*, which is very near *ws3*. Because of this location and the similarity of culm, sheath and husk phenotypes of *v26* to *ws3*, we tested the two mutants for allelism. Surprisingly, the *ws3* stock obtained from the Maize Coop. also had virescent seedlings, although this is not stated in the MNL gene lists or in Mutants of Maize. Crosses between these two mutants proved them to be allelic. The symbol *ws3* has priority.

BUFFALO, NEW YORK
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LONDON, ONTARIO
University of Western Ontario

**Three-dimensional visualization of meiosis I in Ohio43**

--Lee, TC, Walden, DB, Cheng, PC

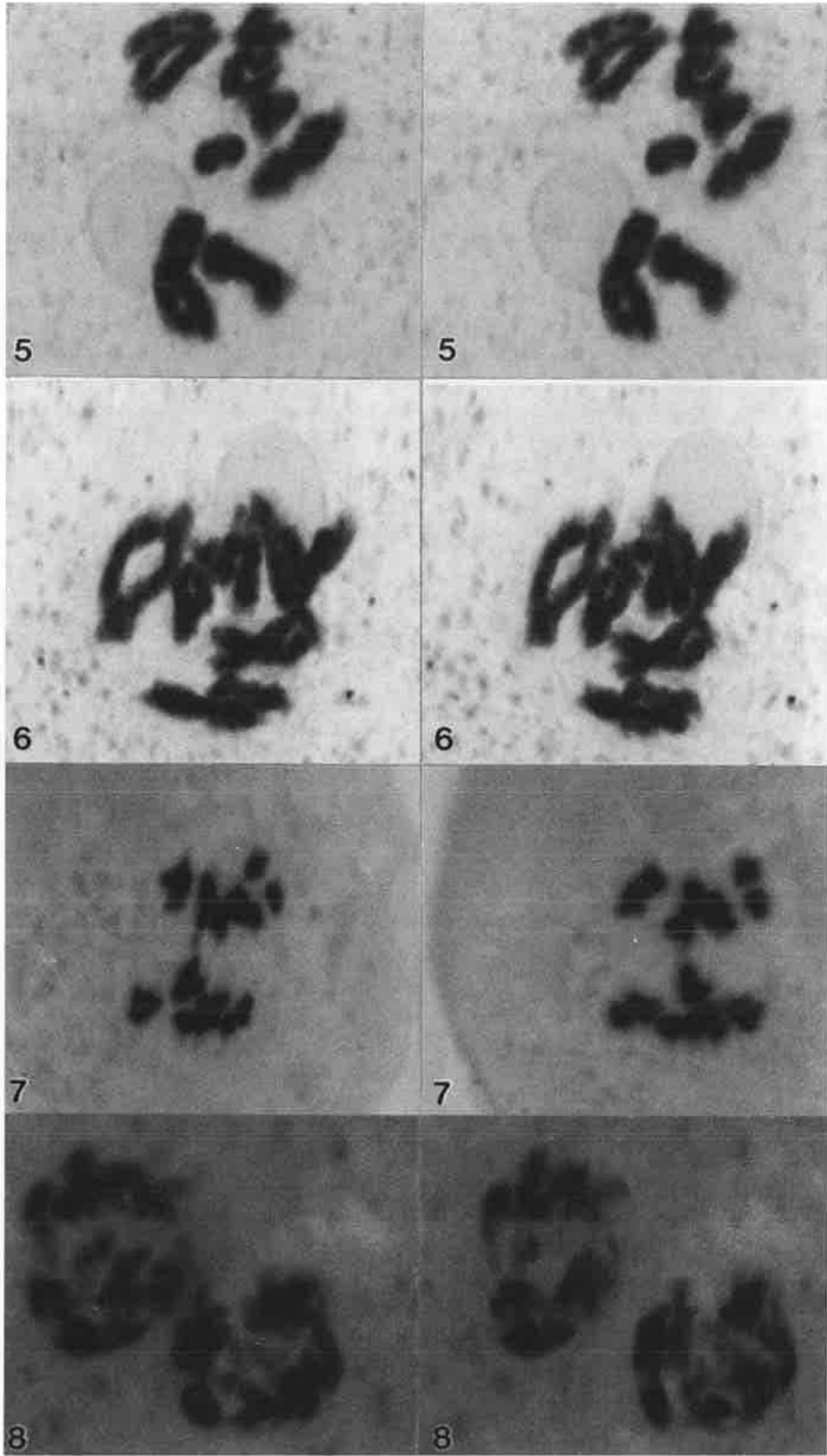
Visualization is interdependent with gene mapping and DNA sequencing, because spatial relationships are important to understand function (Brakenhoff et al., Nature 317:748-749, 1985; Dempsey, The Maize Handbook, Springer-Verlag, 432-441, 1994). The ability to visualize the chromosomes, especially in three-dimensional (3D) space, has great benefits for the better understanding of a gene's function. For these reasons, a better technique is needed to study the chromosomes in 3D. Studying the stages of meiosis I serves as an excellent opportunity to evaluate the capability of 3D imaging techniques. The loosening of the chromosomes in meiosis I exemplify the advantages of 3D imaging.

Tassel inflorescence of Ohio43 inbred were collected from the field station at the University of Western Ontario in London, Ontario, Canada, during the summer of 1998. Tassels at the proper developmental stages were fixed in 3:1 (95%ETOH: acetic acid) solution and stored in mason jars at room temperature. The anthers were treated with the Feulgen-method and cleared in methyl salicylate (winter green oil).

An Olympus GB200 laser scanning microscope equipped with a PlanApo-60X oil immersion objective (NA=1.4) and high precision piezo stage was used in this study. To observe the fluorescence of Feulgen-stained specimens, the 488nm emission line of Ar ion laser was used as the excitation light and >520nm fluorescent emission was detected. Series of optical sections (1024 x 768, 8 bits) were obtained at an interval of 0.1μm. The resulting images were processed through a deconvolution algorithm (Holmes and Liu, Biomedical Visualization, VCH, 283-327, 1992). AutoDeblur™ software (AutoQuant, Watervliet, New York) running a no-neighbor deconvolution algorithm was executed by an Intel Pentium® II based computer operating at 300MHz with 352MB RAM.

For 3D reconstruction, the raw image was reduced to 512x384 in 8 bits format by a binning method to overcome computer hardware and software limitations. Rendering by maximum intensity volumetric method was performed using the VayTek™ Voxblast software. Stereographs were generated using projections from various vantage points.

To ensure better printing contrast, all images are presented in reverse contrast. A stereoviewer can be used to view the sterograph images. Figure 1 is a stereograph of a premeiotic interphase nucleus. Figure 2 shows the lepotene stage. Figure 3 is in the zygotene stage, showing the chromosome mass aggregated to form a "cap" on the nucleolus. Figure 4 is in the pachytene stage. Notice the attachment of chromosome #6 to the nucleolus (No) through the nucleolus organization region (NOR). This nucleolus attachment provides an important feature for the identification of chromosome #6. Figure 5 represents chromosome structures in late diplotene. Figure 6 represents diakinesis (notice the chiasmata), Figure 7 is anaphase I (notice the anaphase bridges, which are the threads stretching across the metaphase plate, and the spindle fibers are also visible). The decrease in the size of the nucleolus throughout the images of prophase I and its disappearance in Figure 7 of anaphase are also noticeable. Figure 8 is late telophase I.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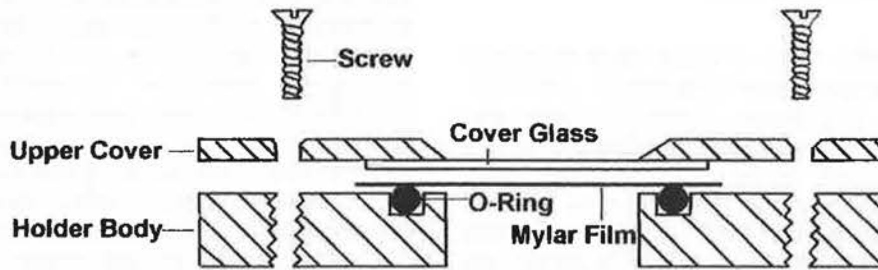
BUFFALO, NEW YORK
Williamsville North High School
Williamsville East High School
State University of New York

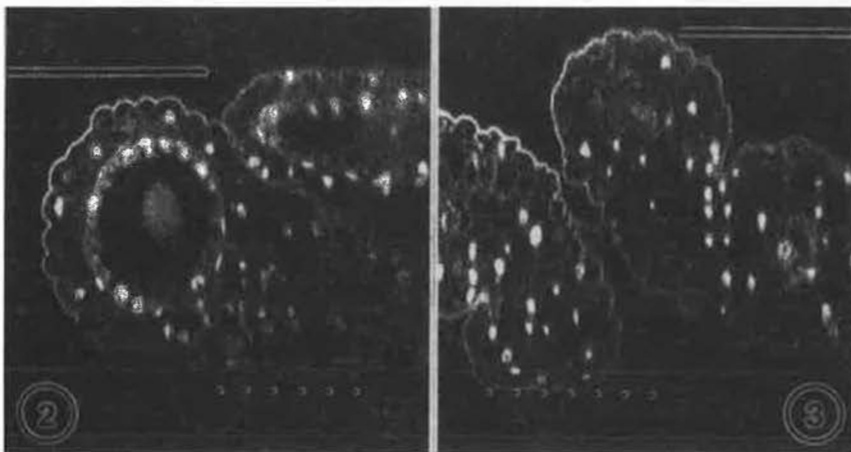
**A specimen holder for confocal and multi-photon fluorescent microscopy**

--Lee, TC, Cheng, WY, Cheng, PC

Confocal microscopy and multi-photon fluorescent microscopy provide unsuppressed capabilities of optical sectioning. Therefore, volumetric data sets can be obtained by sequentially scanning successive levels in a specimen. The quality of this sequential imaging method requires a stable specimen over the period of data acquisition. We have re-designed our previous specimen chamber (Cheng et al., Multi-dimensional Microscopy. Springer, 339-380, 1993) and fabricated a new chamber which allows significantly better specimen holding capabilities to handle maize pollen grains and small embryos.

The specimen chamber consists of two aluminum blocks (upper cover and holder body; Figure 1). The opening of the cover plate is tapered to allow for easy exchange of different objective lenses using a revolving nosepiece. The opening of the holder body allows for the possibility of viewing the specimen in conventional wide-field mode and also enables the scanning of samples in transmitting mode. Specimens are held between a cover glass and a flexible plastic film (e.g. Mylar or acetic cellulose film). An O-ring is installed to provide a clamping force for the cover slip and supporting film and also prevents mounting medium leakage. The




Cover glass ————

Mylar Film · · · · · · ·

flexible plastic film conforms to the curvature of the specimen, resulting in a firm holding force on the specimen.

The holding force experienced by the specimen depends on the stiffness of the supporting film. Therefore, it is important to select a suitable supporting film for a specific sample. Figure 2 and Figure 3 show the effect of two different supporting films for holding maize anthers. Figure 2 is an x-z scan of an anther held by a sheet of acetic cellulose (dotted line, 70µm in thickness) and Figure 3 shows a x-z scan of an anther held by a sheet of Mylar film (dotted line, 10µm, SaranWrap®). The solid line on both figures indicates the surface of cover glass. Note the acetic cellulose film was too stiff and caused a noticeable tissue deformation. The specimen chamber described above is suitable for holding small maize tissue for confocal and multiphoton fluorescent microscopy.

CHESTNUT HILL, MASSACHUSETTS
Boston College

**Regeneration of plantlets in diploid maize**
--Ting, YC, Tran, L

In the summer of 1998, about 10 diploid maize plants were grown in the field. This maize strain came from Corn-Nuts Inc., California as a gift of Dr. D. L. Shaver. It grew in the Boston area as a semiperennial because it failed to regrow from the previous root stock in the second year. During most of the growth period, all of these plants grew vigorously. However, in the middle of September, it was noticed that one of the plants was shorter than the rest. The short plant was apparently slow in growth, with shortened internodes and thick leaf laminae. At the end of September, a male inflorescence developed normally, but only a few antheses appeared. Shortly afterward, some plantlets generated from the spikelets. In the second week of October, five of the plantlets were removed and planted in the greenhouse. They stayed green and alive for nearly two months. Then they discontinued growth. In 1946, Singleton (J. Hered. 37:61-64) reported plantlet generations of a diploid sweet corn. His attempt to grow the plantlets upon removal from the original plant was unsuccessful. He further found that the characteristic of plantlet generation was possibly attributed to a recessive gene, *id*. The reversion of floral meristem to juvenile shoot meristem as was observed in the present study may also be due to a recessive gene. For developmental genetics, it may be a valuable material of investigation.

COLOGNE, GERMANY
Max-Planck-Institut für Züchtungsforschung, Abteilung Salamini

**Components of the maize GCN5/ADA2 coactivator complex**
--Becker, H-A, Riehl, M, Santandrea, G, Serna, A, Thompson, RD

The bZIP-type transcriptional activator *opaque2* (*o2*) has been shown by a number of groups to be a major regulator of storage protein expression in maize endosperm. The availability of a heterologous protoplast trans-activation assay enabled us to map the region of Opaque-2 which functions as a major activation domain, i.e., is needed for Opaque-2 protein to activate expression from a target promoter. Similarities between the acidic activation domain sequence of Opaque-2, and several other plant transcription factors with that of the yeast bZIP factor GCN-4 led us to speculate that the mechanism of acidic domain-mediated activation in yeast and maize would be similar (current models for transcriptional activation invoke adaptor complexes which mediate between transcription factors binding to upstream activating sequences and the RNA-polII located at the transcription start). To get more insight into the detailed mechanism of transcriptional activation in the developing endosperm, we have begun to isolate central components of the yeast GCN5/ADA2 complex from maize. The histone acetyltransferase (HAT) clone zmGCN5 was isolated by 2-Hybrid screening of a maize cDNA library using a GAL4-ADA2 fusion protein as bait construct. The bait ADA2-sequence information was derived from heterologous plant EST information. The full length zmGCN5 encodes a 515 amino acid protein which possesses 59% similarity and 49% identity to yeast GCN5 over the C-terminal 2/3 of the sequence, including the catalytic domain, ADA2-interaction domain and a bromodomain. A single gene copy of zmGCN5 is present in the maize genome. A 2.2 kb mRNA is detected in the endosperm at all stages of maize karyopsis development analyzed so far, and elsewhere in the plant, predominantly in mRNA from actively dividing cells. The protein was expressed as a GST-fusion in *E. coli*, and the *E coli*-derived zmGCN5 was shown to have HAT activity on core histones in vitro. HAT enzymes are found as components of multiprotein complexes in yeast and other eukaryotes. In the best characterised system, yeast, at least 4 different complexes have been identified, the simplest of which contains, in addition to GCN5, ADA2 and ADA3. Although histone acetyltransferase activity is retained by isolated HAT enzymes in vitro with core histones as a substrate, the supplementary proteins are required for the acetylation of histones in nucleosomes in vitro, and putatively, in vivo too. Recently, we have isolated a putative partner of zmGCN5, zmADA2, from a maize cDNA library by heterologous probe screening. However, we have yet to find an ADA3 homologue. Approaches to unravel the role of both proteins in transcriptional activation and attempts to isolate further components of adaptor complexes are in progress.

COLUMBIA, MISSOURI
USDA-ARS and University of Missouri

**Allelism of *v28* stock with *yg2***
--Coe, EH

My stock of *v28*, obtained from M.G. Neuffer (27:1121-4@), aka v*-N27, does not complement *yg2*. By oversight, I had not previously conducted an allelism test. This mutant complements *pyd1* but does not complement any known *wd1* mutation. Its phenotype is like that of *yg2*, varying with background, like *yg2-ref*. The designation of the *v28* mutation should be *yg2-N27*, based on its original designation as *v*-N27* (aka *v*-27*, aka *v28)*. Our paper on physical and genetic mapping of terminal deficiencies in 9S (Chao et al., Genetics 143:1785-1794, 1996) reported that the map site of *v28* was not separated from that of *yg2* by RFLP markers or by *wd1* deficiencies, and the results of the allelism test are consistent with those results. Unfortunately, this reduces the number of mutants by which deficiencies in 9S might be discriminated. Isolate *v*-N697*, which is allelic to *v*-N27* (tests by M.T. Chang), has not been tested directly against *yg2*, but presumably it is allelic. Both stocks have been provided to the Stock Center (per D. England). The *wd1* deficiencies reported in the 1996 pa-

per will be made available at the Stock Center.

### Tests of seedling mutants on 9S

--Coe, EH

Results of some complementation tests of seedling chlorophyll mutants are shown in Table 1. Direct tests of *v*-N1893* showing it is not allelic to *yg2*, combined with results with *wd1*, indicate that this mutant is an allele of *pyd1* (i.e., *pyd1-N1893*). This mutant arose in a *bz2-m* experiment -- if it should prove not to be a deficiency, it is a genic mutation in the region that *pyd1* deficiencies on 9S uncover. Tests of *w*-N1865*, which does have a white phenotype, indicate it is a *wd1*-like deficiency. Tests of *w*-N1854*, with a white to near-white phenotype, also suggest it may be a *wd1*-like deficiency, but in the absence of tests on *pyd1* this remains uncertain. Both of the latter mutants arose in an *a1-m* experiment. I appreciate the help of Dan England in selecting these mutants, and in advising on their origin and pedigree.

Table 1. Complementation tests among chlorophyll mutants on 9S. Phenotypic designations are virescent (v), pale yellow (py), yellow-green (yg), and white (w). Tester stocks were homozygous *yg2 yg2*, and stocks segregating for heterozygosity for *v28*, *pyd1*, *v31*, or *wd1*. Numbers with + marks represent the number of non-complementing tests in which the mutant was known to be present in the pollen parent.

| Mutant | Phenotype | *yg2* | *v28* | *pyd1* | *v31* | *wd1* |
|---|---|---|---|---|---|---|
| *v*-N1893* | v>py>w | 3+ | 8+ | py (3 tests) | 5+ | w (1 test) |
| *w*-N1854* | py?>w | yg | yg | - | 2+ | w (1 test) |
| *w*-N1865* | w | yg | yg | py (1 test) | 3+ | w |

COLUMBIA, MISSOURI
University of Missouri
URBANA, ILLINOIS
USDA/ARS/MWA
Maize Genetics Cooperation • Stock Center

### Allelic relationships among the zebra mutants in the Coop's *zb1*, *zb2*, and *zb3* stocks

--Szalma, S, Stinard, PS, Cone, KC

Several of the zebra crossbands (zb) mutants in maize exhibit a similar phenotype in which the leaves display transverse bands of normal green tissue alternating with yellowish, frequently grainy, bands of tissue deficient in chlorophyll. To begin to explore the genetic interaction among this class of zb mutants, we intercrossed the Coop's *zb1*, *zb2*, and *zb3* stocks with the goal of producing double zb mutants. Surprisingly, in the F1 progeny of *zb1* x *zb2*, all plants had a zebra phenotype, suggesting that these two mutants are alleles of the same gene. Two sets of F1 progeny were obtained by crossing *zb1* and *zb2* to two distinct *zb3* stocks. The progeny from crosses of *zb1* and *zb2* to one *zb3* parent (Coop Stock 504C) displayed a zebra phenotype, whereas the progeny from crosses of *zb1* and *zb2* to a second *zb3* parent (Coop Stock 519G) displayed a normal phenotype. To help sort out the relationship among these zb mutants, we created separate F2 populations segregating for *zb1*, *zb2*, or *zb3* (504C). Molecular segregation analysis was performed using an RFLP marker from chromosome 5L, the reported location of *zb3*. The results showed that *zb1*, *zb2* and *zb3* (504C) all map on 5L, suggesting that all are allelic. To help resolve these discrepancies, pedigrees of the zb stocks maintained by the Coop were reviewed. The *zb1* (U340B) and the *zb3* (519G) mutants, both originally obtained from Punnett's and Burnham's collections, appear to be bona fide. However, pedigree searches reveal that the *zb3* stock maintained as 504C really carries *zb1*, not *zb3*, and that this *zb1* allele is identical to the *zb1* allele in U340B. The identity of *zb2* is still a mystery. It was originally described as a seedling zebra (Stroman, GN. 1924. Genetics 9:493-512), but the mutant phenotype of the Coop's *zb2* stock (U340C) is evident in the mature plant (like *zb1*) and not in the seedling. A pedigree search of the Coop's *zb2* stocks gives no reason to suspect a recent stock mix-up. The *zb2* stock was propagated from an intercross between separate *zb2* stocks obtained from Eyster and Burnham. The F1 cross of these two sources produced zebra plants, indicating allelism. Also, notes taken when the Burnham stock was first grown by the Coop in 1943 reveal that this *zb2* stock was a mature plant zebra, not a seedling zebra. Therefore, if the *zb1* and *zb2* stocks were mixed up, the mix-up occurred before these mutants came into the Coop's hands, and had to have been present in both the Eyster and Burnham sources. Based on our allelism test and mapping results, we conclude that the zb in the U340C *zb2* stock is at least allelic (and maybe identical) to *zb1*. To reflect the allelism between the Coop's stocks of *zb1* and *zb2*, the *zb1* allele in the *zb2* stock has been renamed *zb1-2*

In summary, we conclude that *zb1* maps to the long arm of chromosome 5. *zb3*, as found in stock 519G, represents a separate gene whose map location is unassigned. TB tests are in progress in order to place *zb3* to chromosome arm. All three distinct stocks, *zb1*, *zb1-2*, and *zb3*, have been assigned new stock numbers (519H, 519I, and U840E, respectively) to reflect their chromosomal locations.

COLUMBUS, OHIO
Ohio State University

### Ectopic expression of P and R+C1 induces few new proteins

--Grotewold, E

The ectopic expression of the P and R+C1 regulators of maize flavonoid biosynthesis in cultured BMS cells induces the accumulation of distinct classes of flavonoid and phenylpropanoid compounds (Grotewold et al., Plant Cell 10:721-740, 1998). To gain insight into how many genes might be regulated by P or R and C1, proteins were radiolabeled with $^{35}$S-methionine in vivo and separated by 2-D polyacrylamide gel electrophoresis (PAGE). Only a small number of new proteins that were not found in untransformed cells were detected by this method in either the P- or R/C1-expressing cell-lines (Fig. 1). This number is of the order of magnitude expected, if the sole functions of P and R/C1 are to induce de novo some or all of the enzymes and trafficking components required for 3-deoxy and 3-hydroxy flavonoid accumulation, respectively. Significantly, there were no proteins on these gels that obviously were down-regulated by either P or R/C1. Two proteins in the 40 kD region (black arrows in Fig. 1B-C) that appear to be induced by either P or R/C1 could correspond with chalcone synthase encoded by *c2* (MW 43 kD) and flavanone/flavanonol reductase encoded by *a1* (MW 40 kD). We have not confirmed, however, if these enzymes co-migrate electrophoretically with the proteins indicated. The identity of two proteins that were strongly expressed only in R/C1-expressing cells (indicated by white arrows on Fig. 1B) would merit further study, because neither appears to correspond in molecular weight or pI to any of the proteins encoded by the

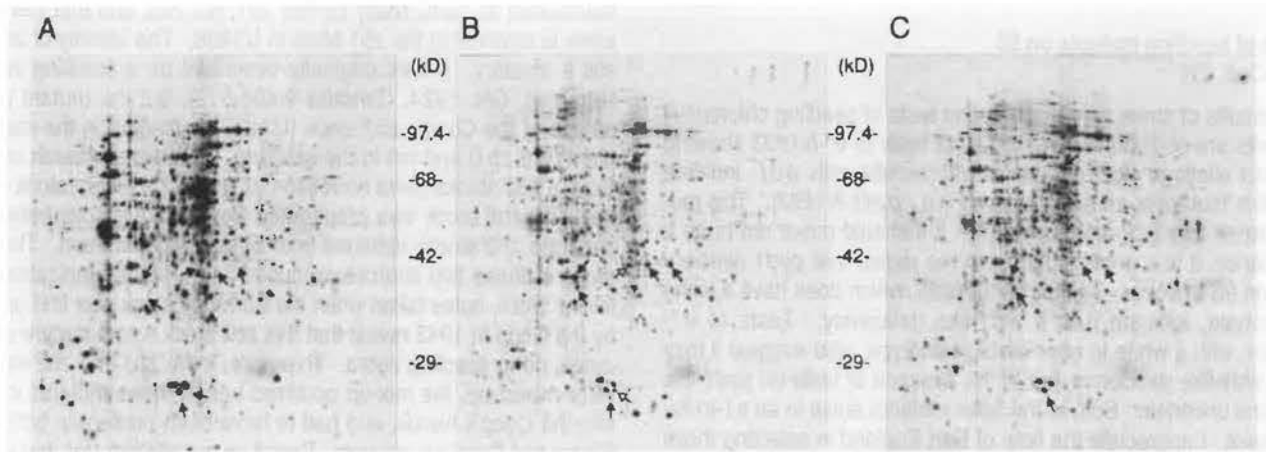



Figure 1. Pattern of $^{35}$S-labeled protein accumulation in BMS callus cells transformed with 35S::R+C1 (B), 35S::P (C); or un-transformed. Arrows in B) and C) indicate dots absent in the control (A) line. The position of those dots in the un-transformed lines is indicated by an arrow. Empty arrows in B) correspond to dots only seen in the R+C1 lines.

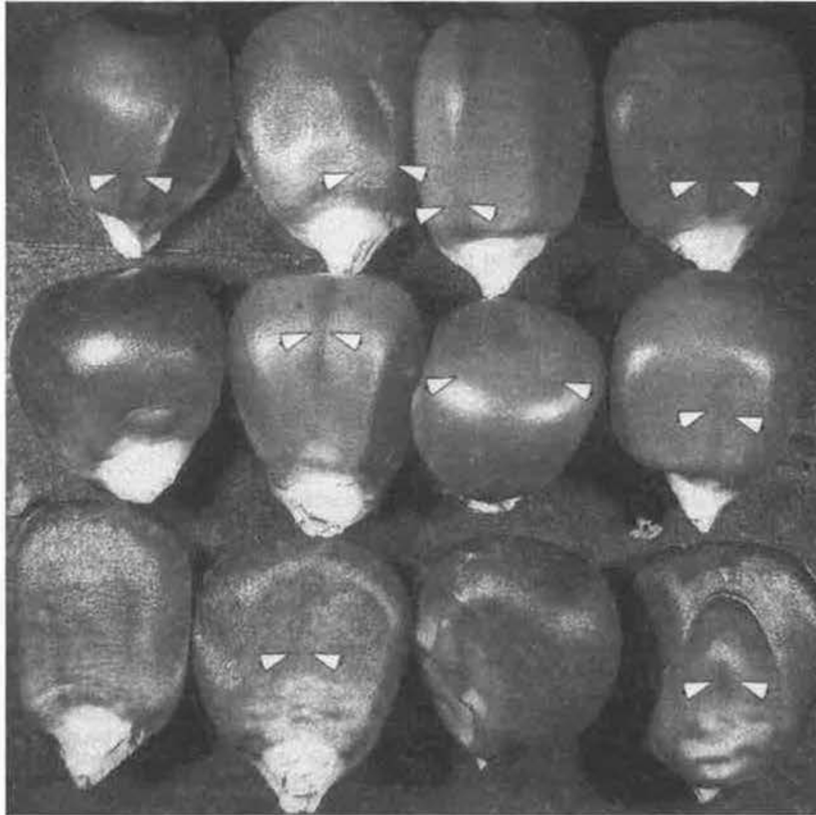
R/C1-regulated genes *f3h*, *a2*, *bz1* or *bz2*. No proteins specifically induced by P were confidently identified by 2D-PAGE, despite the observation that the overexpression of P has some dramatic effects on maize and *Arabidopsis* plants (Rabinowicz et al., MNL 71:21-22, 1997). The formation of compounds controlled by P, including *C*-glycosyl flavones, luteoforol and fluorescent molecules of unknown nature (Grotewold et al., Plant Cell 10:721-740, 1998) may require constitutively expressed proteins. Alternatively, P could induce proteins not detected by this method, for example proteins without methionine or of same MW as pre-existing proteins.

**Aleurone and pericarp pigmentation in the *a1-mum2* allele**

Wang, H, Grotewold, E

The Myb-domain proteins P and C1 control the accumulation of the phlobaphene pigments in the pericarp and cob glumes, and of the anthocyanin pigments in the aleurone, respectively. Recent studies have shown that P and C1 bind to identical sites in the promoter of the *a1* flavonoid biosynthetic gene. The *a1* promoter has a modular structure with proximal $^{ha}$PBS (high affinity P binding sites) and more distal $^{la}$PBS (low affinity P binding sites) (Sainz et al, Plant Cell 9:611-625, 1997). The *a1-mum2* allele harbors a *Mu1* insertion in between these two PBS, in an element that has been found to be conserved in the promoters of other flavonoid biosynthetic genes (Tuerck, J.A. and Fromm, M.E., Plant Cell 6:1655-1663, 1994; Lesnick, M.L. and Chandler, V.L., Plant Physiol. 117:437-445, 1998). In the presence of *Mutator* activity, *a1-mum2* alleles provide densely spotted aleurones on a colorless background (Fig. 1). To investigate the pattern of pericarp variegation provided by the excision of *Mutator*, *a1-mum2* lines were crossed to *P-rr* stocks. In the absence of *a1* gene function, *P-rr* pericarps accumulate a characteristic brown pigment. Seeds derived from plants of the genotype *P-rr/P-wr a1-mum2/a1* were investigated after self pollination or after crossing to *a1/a1* pollen (26A:4-5 x 13-8) for aleurone spotting and pericarp variegation. While aleurone spotting is evident in most kernels (Fig. 1A, B), the expected red pericarp variegated sectors are rare, usually very fine and are present at a much lower frequency than the spots in the aleurone (Fig. 1A, B). The high spotting frequency in the aleurone, and the presence of a large number of spotted aleurones in each ear are indicative of the presence of *Mutator* activity during ear development. The low frequency at which red revertant somatic sectors appear in the pericarp could indicate either that *Mutator* does not transpose with high frequency in the pericarp, or that the *cis*-acting regulatory elements in which *Mu1* is inserted in the *a1-mum2* allele are much more important for the regulation of *a1* by P than by C1. Understanding the reason for the low frequency of variegation in the pericarp may expose a property of *Mutator* unexpected from previous studies, or reveal a fundamental difference in the way the P and C1 Myb-domain transcription factors control the expression of a gene common to the two main branches of maize flavonoid biosynthesis.

A



B

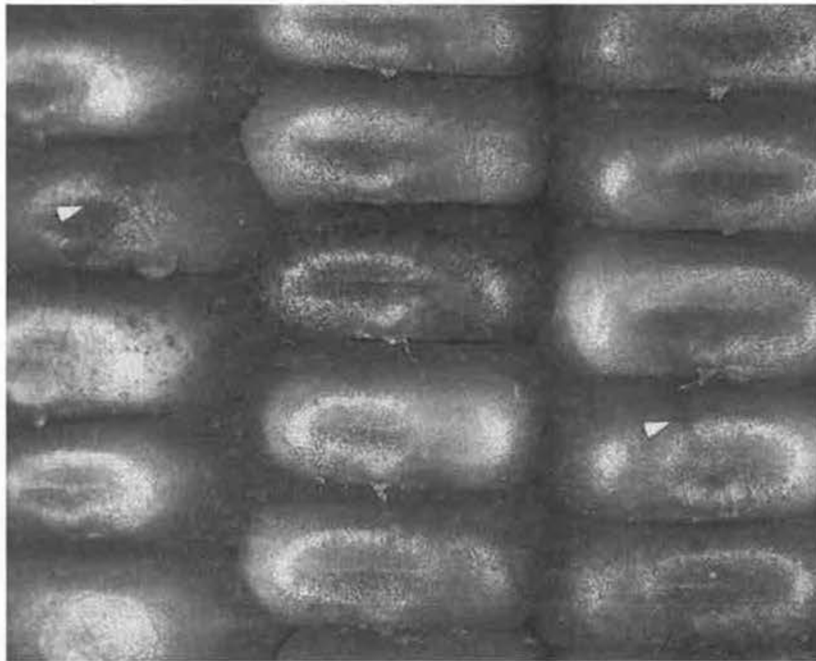


Figure 1. Pericarp and aleurone pigmentation in the *a1-mum2* allele. **A)** Kernels derived from selfing *P-rr/P-wr a1-mum2/a1* plants (26A:4-6@) were selected to illustrate the variety of red revertant sectors found in the pericarp (arrows indicate the width of sectors). **B)** A sector of an ear derived from the cross *P-rr/P-wr a1-mum2/a1* x *a1/a1* (26A:4-5 x 13-8) shows the low frequency of red variegated pericarps. Arrows indicate two such sectors.

## Diversification of the *R2R3 Myb* gene family and the segmental allotetraploid origin of the maize genome

--Braun, EL, Grotewold, E

The maize genome is thought to have arisen by the reversion of an ancient polyploid to disomic inheritance (reviewed by White and Doebley, Trends Genet. 14:327-332, 1998). Comparisons of divergence times for specific duplicated loci in maize indicate that they exhibit two different coalescent times, corresponding to divergence times of approximately 11.4 mya (million years ago) and 20.5 mya, suggesting that the ancestor of maize was a segmental allotetraploid (Gaut and Doebley, PNAS 94:6809-6814, 1997). The maize genome duplication is expected to result in the doubling of any gene families present prior to duplication event, suggesting that surveys of large gene families are likely to reveal a signature of the segmental allotetraploid origin of the maize genome.

As a part of a larger survey of the *R2R3 Myb* gene family in maize (Rabinowicz et al., submitted for publication), we examined recent *Myb* gene duplications in maize. This survey of *R2R3 Myb* genes was accomplished by using RT-PCR (reverse transcriptase-polymerase chain reaction) to amplify a short segment of the *Myb* genes using a pair of degenerate primers corresponding to the conserved DNA recognition helices. The RNA used for RT-PCR was prepared from seedlings and various tissues of maize plants under normal growth conditions. Analysis of these segments is complicated by their limited length (averaging 129 bp) and their biased codon usage (mean GC content of third codon positions is 90%). These factors result in high variance of individual distance estimates and cause most commonly used methods of estimating synonymous distances to significantly underestimate the number of substitutions when highly divergent sequences are compared. However, analysis of the data can reveal general patterns, such as the presence of *Myb* genes that originated during the maize genome duplication. In fact, one of the gene pairs analyzed by Gaut and Doebley (PNAS 94:6809-6814, 1997) corresponds to the *Myb* genes encoded by *C1* and *Pl*, indicating that *Myb* genes were duplicated during the allotetraploid origin of the maize genome. Among the 44 recently duplicated *Myb* genes identified (Table 1), we found 10 groups of Myb sequences that correspond to *Myb* genes that are likely to have undergone duplication during the allotetraploid origin of the maize genome. However, we also found indications of additional recent gene duplications and complex patterns of evolution for *Myb* genes in maize.

Five groups of recently duplicated *R2R3 Myb* genes have three or more member sequences, clearly indicating the existence of recent *Myb* gene duplications that do not reflect the maize genome duplication. The largest group of recently duplicated *Myb* genes identified by this study, group 2 (Table 1), has four additional members based upon the unweighted maximum parsimony (MP) estimate of phylogeny for the *Myb* genes of maize obtained using amino acid sequences (Rabinowicz et al., submitted for publication). These sequences may correspond to genes that have diverged at a higher rate than other *Myb* genes, although it is important to note that any accelerated divergence must have occurred at synonymous sites. Indeed, these results suggests that there may be currently unappreciated sources of rate variation at synonymous sites in the maize genome. This rate variation probably does not reflect differences in codon usage, since all four of the divergent sequences exhibit biased codon usage (third codon position GC content ranges from 81.4% to 86%). However, additional sources of rate variation may include factors such as gene conversion resulting in slower than expected divergence between specific *Myb* genes or differences in the rate at which synonymous mutations accumulate in genes with different chromosomal locations (such as that noted for mammals by Wolfe et al., Nature 337:283-285, 1989).

Table 1. Recently duplicated *R2R3 Myb* genes identified in maize [a].

| Group | Sequences | Ks [b] | Divergence (mya) [c] |
|---|---|---|---|
| 1 | *Pl*, *C1* | 0.0597 | 4.6 |
| 2 [d] | *P*, IP20, 1C1, IQ68, 1H48, 2H67 | 0.4233 | 32.6 |
| 3 | IF17, IQ32 | 0.1850 | 14.2 |
| 4 | 1C4 [e], IP59 [e] | 0.1752 | 13.5 |
| 5 | 3H101, IP126, IP39 | 0.0996 | 7.7 |
| 6 | 4H48, IF41, IM66 | 0.2172 | 16.7 |
| 7 | IM16, IP29 | 0.1408 | 10.9 |
| 8 | IP122, IP156 | 0.2171 | 16.7 |
| 9 | HX30, IP148 | 0.0653 | 5.0 |
| 10 | IF50, IP26 | 0.0309 | 2.4 |
| 11 [f] | IM61 [e], IQ26 | 0.2029 | 15.6 |
| 12 | IF45, IP119 | 0.0630 | 4.8 |
| 13 | 1C18 [e], IF55 [e] | 0.0951 | 7.3 |
| 14 | 1H9 [e], IM65 [e], IP47 [e] | 0.2619 | 20.1 |
| 15 | IP45, IP71, IP74 | 0.3710 | 28.5 |
| 16 | IF13, IF14 | 0.1010 | 7.8 |
| 17 [g] | IP19, IP34 | 0.0000 | 0.0 |
| 18 | IP102, IP124 | 0.1375 | 10.6 |

[a] Recent duplications were identified by screening the Myb sequences for those with uncorrected synonymous distances lesser than 0.3 and uncorrected nonsynonymous distances lesser than 0.1.

[b] Ks (synonymous distance) of the most divergent comparison. Synonymous distances were calculated by MEGA 1.01 (computer program available from the Institute of Molecular Evolutionary Genetics at the Pennsylvania State University, University Park, PA) using the method of Nei and Gojobori (Mol. Biol. Evol. 3: 418-426, 1986) with the Jukes-Cantor correction for multiple hits. This method will produce underestimates of the synonymous distance for more ancient duplications, due to the codon bias in this dataset. However, the underestimation will be fairly modest for the divergence times considered in this table.

[c] Divergence time in millions of years before present calculated by assuming that synonymous mutations accumulate at an average rate of $6.5 \times 10^{-9}$ substitutions per synonymous site per year (see Gaut et al. PNAS 93: 10274-10279, 1996). The sampling variance of individual distance estimates indicates that the coefficient of variation for specific divergence times ranges from approximately 30% to 50%.

[d] Four additional sequences (1C42, IF25, IF35, IM44) belong to this group based upon the unweighted maximum parsimony estimate of phylogeny. They may represent rapidly evolving sequences.

[e] These sequences exhibit less extreme codon bias, with less than 80% GC in third codon positions.

[f] Two additional sequences (IP49, IP108) belong to this group based upon the unweighted maximum parsimony estimate of phylogeny. They may represent rapidly evolving sequences.

[g] These sequences exhibit 3 nonsynonymous differences in the sequenced region.

A total of 26 Myb sequences corresponding to 13 recently duplicated pairs of genes were identified among the 82 *R2R3 Myb* genes sequenced as a part of the survey performed by Rabinowicz et al. (submitted for publication), including the *Myb* genes encoded by *C1* and *Pl*. At least 10 of these pairs are likely to reflect duplications that occurred during the maize genome duplication. Two of these groups of sequences, groups 10 and 17 (Table 1) appear to represent very recent divergences that may have occurred after the allotetraploid origin of the maize genome. One pair of sequences, group 11, corresponds to a gene clade that has two additional members based upon the MP estimate of maize *Myb* gene phylogeny (Rabinowicz et al., submitted for publication). Like the additional sequences that appear to belong to group 2, these sequences may correspond to genes that have diverged at a higher rate than other *Myb* genes. The degree of codon bias for the divergent sequences does exhibit some variation from that observed for other *Myb* genes, since IP49 is less biased (third codon position GC content is 69.8%) while IP108 is highly biased (third codon position GC content is 97.7%). However, the absence of a consistent pattern suggests that the divergence of these genes does not reflect their differences in codon bias.

The basis for the maintenance of duplicated genes in organisms

has been the subject of substantial debate, since duplicated genes are predicted to exhibit functional redundancy. One possibility is that duplicated genes rapidly establish different patterns of expression, making both genes subject to selection because their activity is necessary in different tissues. In fact, different patterns of gene expression have been noted for duplicated *Myb* genes, such as the duplicated *C1* and *Pl* genes of maize (Cone et al., Plant Cell 5:1795-1805, 1993). A similar situation was evident for two additional pairs of *Myb* genes corresponding to groups 7 and 12 from Table 1. However, the remaining pairs exhibit at least some overlap in their expression patterns and two groups (group 9 and group 17 from Table 1) exhibit complete overlap in their expression patterns. Although these data cannot exclude the possibility that subtle differences in expression patterns exist for some of these gene pairs, they are not consistent with the hypothesis that patterns of expression are often altered following gene duplications. Instead, they suggest that patterns of gene expression may exhibit some degree of conservation, at least over relatively short evolutionary time scales.

The *R2R3 Myb* gene sequences obtained by Rabinowicz et al. (submitted for publication) provide evidence for the existence of duplicated *Myb* genes in maize that reflect the segmental allotetraploid origin of the maize genome. However, they also provide evidence for additional gene duplications that cannot be explained by the maize genome duplication as well as evidence for unappreciated sources of rate variation at synonymous positions in a subset of maize *Myb* gene sequences. The availability of these short segments from *Myb* genes will facilitate future work, such as obtaining full length cDNAs to determine the similarity between their carboxyl-terminal and mapping of these genes to firmly establish that their origin reflects the segmental allotetraploid origin of the maize genome. Furthermore, the recently duplicated Myb genes identified in this study suggest the existence of 18 or fewer gene duplications associated with the duplication of the maize genome, which is substantially lower than that expected if the sampling of maize *Myb* genes is complete. The excess of *Myb* genes without closely related paralogues in this dataset suggests that the sampling of *Myb* genes in maize remains incomplete. Alternatively, duplicated loci that were not detected by this study may have been lost, may not be expressed at detectable levels under the growth conditions examined, or may be obscured by rate differences. Regardless of the specific explanations, it is clear that the *Myb* genes present in maize have undergone many recent duplications and that the biological basis for these duplications is relatively complex.

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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 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 by data on the expression of a part of sporophyte genes in the period of gametophyte existence. The investigations on maize gametophyte selection confirm the efficiency of such an approach. The estimation of effects of genotypes on pollen tolerance to different environmental 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 details of microgametophyte selection will permit carrying out such selection in prospective populations in 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-methoxiethyl)-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 26-28 C. To the control medium was added 10 mg/l, 50 mg/l, 100 mg/l and 500 mg/l metholachlor. The data are shown in the table.

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

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

| Treatment | Percentage of pollen grains germinated | | | | |
|---|---|---|---|---|---|
| | 751 | Ul26 | 1620 | Al427/501 | Al4/217 |
| Control | 54.78 | 13.22 | 11.50 | 9.83 | 7.75 |
| 10mg/l | 42.63*** | 12.3316.50 * | 13.17 | 11.63** | |
| 50mg/l | 10.38*** | 3.78*** | 4.17*** | 2.33** | 1.56*** |
| 100mg/l | 0*** | 0.44*** | 0.83*** | 0.17*** | 0*** |
| 500mg/l | 0*** | 0*** | - | - | - |

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

As 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 Ul26, where the percentage of germination in the control was 13.22, the effect of inhibition was observed at a concentration of 50 mg/l metholachlor. For other genotypes with a lower percentage of germination in the control the concentration 10mg/l had a stimulating effect and a significant decline was obtained at the metholachlor concentration 50mg/l. The fall in the percent of pollen germination put together for genotypes was 63.74-81.05% for concentration 50 mg/l and 92.78-100% for concentration 100 mg/l. However, the percentage of pollen germination for all genotypes did not decrease at the same rate. The smallest 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.

**Anatomical analysis of maize androgenic structures**

--Satarova, TN

The late embryoidogenesis in maize anther culture and the composition of mature embryoids provides further regeneration and secondary callusogenesis. We observed that the majority of embryoids in the moment of their departure from an anther had the organs typical for mature zygotic embryos, such as scutellum

and root and shoot points of growth, but often they were abnormally altered. In the scutellum a 1-2-layer epidermis with very little starch was seen. In some cases it was modified into multilayer epidermal tissue, or nipple-shaped epidermis with a large amount of starch. The subepidermal layer usually developed into multicellular tissue and differed with superproduction of starch. The filling of its cells with plenty of starch grains, often huge, led to different cellular abnormalities, such as the crushing of the nucleus with growing starch grains, the appearance of nuclei of irregular form, the formation of 2-nuclear cells due to disruption of the cell wall and fusion of two nuclei. In parenchyma of the central part of the scutellum great starch accumulation was also observed but nuclei here were normal, and cells were not compact. In the central part of the scutellum sometimes single, extremely large cells with nuclei of irregular shape occurred. We observed also abnormalities of root and shoot points of growth leading to the decline of regeneration ability, such as the absence of meristematic zone in the well-formed root eminence or stem bud; the development of callus on the coleoptile; or the squeezing of the stem bud with neighboring embryoids, which did not allow it to emerge normally. These abnormalities partially explain the fact that the number of embryoids which had given the seedlings by emergence was considerably lower than the number of embryoids transferred. Thus, for genotypes B14xWf9, B14xAnd44 and And44xDK201 the regeneration frequencies were correspondingly 24.14, 40.54 and 54.55%. The critical size of embryoids at the time of their departure from an anther also apparently had great value. If the length of embryoid had been less than 3mm, only 22.95% were able to undergo the regeneration of seedlings. If it had been from 3mm to 6mm and more, the regeneration frequency increased to 36.31%.

**The results of studies of morphogenetic potential of maize inbreds with different length of vegetative period**

--Piralov, GR

It is known that inbreds of maize are discriminated by the rate of callus growth, frequency of totipotent callus formation, plant regeneration and duration of callus cultivation in undifferentiated state. These differences were explained by genotypic features of inbreds (Duncan D.R. et al., Planta, 165:322-332, 1985). Without any doubt in the basic rules of genetic factors in the control of the morphogenetical potential of maize, we attempted to consider this problem from another point of view and compared the peculiarities of callusogenesis and regeneration in maize inbreds in connection with the length of their vegetative period. The preliminary study (Piralov et al., Bull.Inst.Grain Farming, 1997, N4) carried out with 9 early-late (length of vegetative period 90-105 days) and middle-late (115-130 days) maize inbreds revealed higher morphological potential in the middle-late inbreds. In the present experiment we compared morphogenetic potential of early-late inbred DK2/66 and middle - late inbred DK675.

The calli were induced from immature embryos (1.0-2.0 mm) on medium containing the inorganic nutrients of N6 and MS media, vitamins of D medium (Duncan et al.,1985), L-proline (690 mg/l), myo-inositol (100 mg/l), casein hydrolysate (100 mg/l), silver nitrate (10 mg/l), sucrose (20 mg/l), 2,4-D (1 mg/l), agar (7 g/l). The explants were grown in the dark at 25-27 C and transferred on fresh medium every 15 days. For differentiation and regeneration, calli were transferred on media either with 0.1 mg/l 2,4-D or without hormone.

Summary data for both media are given in the table. As shown in this table, the frequency of totipotent callus formation and regeneration was higher in DK675. In particular, the frequency of regeneration on media with different levels of hormone exceeded inbred DK2/66 by 14-20 times. In addition, if the differentiation and regeneration process in inbred DK2/66 is restricted by primordia and small seedlings (up to 1 sm) formation in DK675 in the same period of time plantlets are formed about 5-10 sm and higher. Some of these plantlets were grown to maturity, selfed or sibbed and their seeds were collected for field evaluation. Regenerants of the early-late inbred were not obtained. We can support calli of DK2/66 in culture only 3-4 months, while DK675 calli kept about 5 years without any loss of regenerative potential.

Table. The frequency of totipotent callus formation and regeneration of inbreds DK2/66 and DK675.

| | | Frequency of Regeneration | | | |
|---|---|---|---|---|---|
| Inbred | Frequency of totipotent callus formation (%) | Number of calli | Aging of calli (days) | 2,4-D content in medium (mg/l) | Number of plantlets/callus |
| D_2/66 | 54.8 a | 338 | 29-36 | 1.25 | 0.1 a |
| -"- | | 59 | 46-52 | 0.1 | 1.4 a |
| -"- | | 54 | 94 | 0 | 0.4 a |
| D_675 | 61.6 b | 355 | 29-38 | 1.25 | 1.5 b |
| -"- | | 20 | 53 | 0.1 | 14.3 b |
| -"- | | 84 | 87 | 0 | 8.5 b |

Thus, if the morphogenetic potential of inbreds is evaluated with the combination of such properties as frequency of totipotent callus formation, quantitative and qualitative indexes of regeneration, rate of callus growth and duration of callus growth without loss of regenerative ability, we can draw the conclusion that the middle-late inbred possesses much higher morphogenetical potential than the early-late ones. The explanation of the differences in morphogenetic potentials among inbreds with different length of vegetative period should be searched for in genetic and physiological peculiarities of inbreds, in the distinctions among the inbreds in the pool of endogenous hormones and in the activity of enzymes, that are metabolizing the hormones.

The question about relations between the length of the vegetative period of maize inbreds and their morphogenetical potentials may be interesting for the creation of a selection system in vitro for the length of the vegetative period.

**Five-year old embryogenenic callus culture of maize inbred DK675**

--Piralov, GR, Abraimova, OE

For the obtaining of plant somaclonal variants the application of long-term embryogenic callus culture is preferable, because large numbers of genetic mutations during cultivation are accumulated. In this report the obtaining, stabilization and several peculiarities of five-year old embryogenic callus culture of maize inbred DK675 are described. In the spring of 1993 the culture was induced from immature embryos (length 1.0-2.0 mm) on the medium containing the inorganic nutrients of N6 medium, vitamins of D medium (Duncan D.R. et al., Planta, 165: 322-332, 1985), L-proline (690 mg/l), myo-inositol (100 mg/l), casein hydrolisate (100 mg/l), silver nitrate (10 mg/l), sucrose (20 mg/l), 2,4-D (1 mg/l), agar (7 g/l). The explants were grown in the dark at 25-27 C and were transferred on fresh medium every 12-20 days.

The frequency of embryogenic callus formation was 63.0-77.5%. The embryoids were observed on the callus surface at 10-

14 days after induction as globular structures, situated either one by one or in groups. The first 60 days calli were grown on medium with initial content of 2,4-D. Under such conditions the signs of differentiation and regeneration were observed on the tissue surface. Therefore after 2 months of cultivation the level of 2,4-D was elevated to 1.25 mg/l, and after the next 2 months it was increased to 1.45 mg/l. Under these conditions the callus has been growing all the following period. The callus produced plantlets very intensively on media with 0.1 mg/l 2,4-D and without hormone.

The callus proliferated more actively at the spring-summer period of growth than at the autumn-winter one. In addition, it showed better growth when callus pieces were placed very densely, near each other.

To the end of the growing cycle (20 days) the callus included white compact scutellum-like bodies, embryoids, primordia and numerous sectors of friable undifferentiated nonmucilagenous tissue. It collapsed easily into small pieces by slight mechanical influence or self-voluntarily. These pieces were used for propagation of the callus. In the spring of 1997 we twice determined the rate of growth of culture. The fresh weight of the callus increased about 2 times for the 20 days of growing.

As the result of propagation and growing we obtained a large amount of callus material which has been growing about five years without loss of regeneration capacity. The material is unique because it resulted from one embryo and is of interest for the researching of somaclonal variability, cell selection and genetic engineering.

**Examination of the direct method of obtaining embryogenic cell suspensions of maize**

--Piralov, GR, Abraimova, OE

The creation of cell selection technology requires reproducible methods of obtaining embryogenic cell suspensions for a wide range of genotypes. At present such cell suspensions of maize are obtained by the indirect method, based on the disintegration in liquid medium of friable embryogenic callus of type II. In this report the results of examination of the direct method of obtaining cell suspensions of maize is described (Mezentsev A.V. et al., Dokl. VASHNIL, 9:3-5, 1987). Cultures were induced from seedling pieces of F1 hybrid Slavutich3 in N6 medium with L-asparagine (400 mg/l), L-proline (1200 mg/l), 2,4-D (4-6 mg/l) and were incubated in the dark at 27-28 C on a gyratory shaker (120 rpm). The process of cell suspension formation was going on in the 2 stages. In the first stage, which continued about 20 days, the heterogenous culture with single cells and groups (2-5 cells) was formed. The first cells were elongated as a rule, and were observed approximately at 5-6 days of growing, while the first oval to round small cells were found at 8-10 days. At 4-5 days of cultivation the liquid medium in vessels became very mucilagenous and therefore it was substituted every 3-4 days.

At day 20 of cultivation the density of cultures was about 1 million cells/ml. Their viability determined by the method of Widholm (Widholm J.M., Stain Technol., 47:189-194, 1972) was 50-60 %. The cultures were well dispersed and contained single cells and groups (2-10 cells). After 3 weeks, the old medium was discarded and cultures were transferred to new vessels. To these vessels 15 ml of fresh medium was added.

In the second stage the growth rate of suspensions increased and resulted predominantly in cultures of oval to round small cells with dense cytoplasm. We selected several sublines with a high rate of growth from the culture. The sublines were maintained by subculturing every 7 days on initial medium (5-7 ml inoculum on 15 ml fresh medium). Their growth subordinated to the exponential law with doubling time about 48-72 h in log-phase. In the cultures on the medium for differentiation (2,4-D - 0.1 mg/l) we observed somatic embryoids. Regeneration potential of these cultures is being researched now.

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**Field trial to test a natural resource for corn rootworm resistance**

--Eubanks, MW, Riedell, W

T. F. Branson (Ann. Entomol. Soc. Amer. 64:861-863, 1971) reported that *Tripsacum dactyloides* is resistant to corn rootworm. A bridging mechanism for moving *Tripsacum* genes into corn has been achieved through wide cross hybrids between *Tripsacum dactyloides* and *Zea diploperennis* (M. W. Eubanks, Econ. Bot. 49:172-182, 1995). Efficacy of this genetic bridge for conferring natural rootworm resistance to corn has been demonstrated through a series of insect bioassays (M. W. Eubanks Amer. J. Bot. (suppl.):84:116, 1997; MNL 70:22-23, 1996; MNL 68:40-41, 1994). Under the auspices of NSF grant no. 9801386, a trial testing efficacy of the rootworm resistance under field conditions trait in crosses between *Tripsacum-Z. diploperennis* and corn was conducted at the USDA Northern Grain Insects Research Laboratory in Brookings, SD, during the summer of 1998.

Twenty to thirty-six seeds of each of the corn inbreds B73 and W64A, and four hybrid lines [B73 X Tripsacorn, B73 X (W64A X Sun Star), W64A X Sun Star, and W64A X Tripsacorn] were planted in a 65 ft X 45 ft plot in rows spaced 40 inches apart in May, 1998. The plot consisted of three test rows surrounded by buffer rows planted in NK4242. The first test row contained B73 and W64A; the middle row was planted in [B73 X (W64A X Sun Star)], and the third row was planted in (W64A X Sun Star) and (W64A X Tripsacorn). The plot was fertilized with 50-35-35 NPK at a rate of 215 lbs/acre on May 8, then planted on May 14. The test rows were hand planted one seed per hill to a depth of 2-2.5 inches at 9-inch plant spacing and infested with an approximate total of 400,000 Western corn rootworm eggs at a rate of 1400 eggs per foot in the three test rows. A biophenometer was placed in the soil to a depth of four inches. All rows were cultivated with a single row blue on May 28 and June 22. On July 16, all plants were dug from the test plots, and the roots were washed with a pressure washer. Then the plants were refrigerated until the roots were rated on July 27.

The roots were scored using the 1-6 Iowa rating scale (Hills and Peters ): 1 = no damage or only a few minor feeding scars; 2 = feeding scars evident, but no roots eaten off to within 1.5 inches of the plant; 3 = several roots eaten off to within 1.5 inches of the plant, but never the equivalent of an entire node of roots destroyed; 4 = one node of roots completely destroyed; 5 = two nodes of roots completely destroyed; 6 = three nodes of roots completely destroyed. Plants that have a root rating of 1 or 2 are resistant. The results are presented in Table 1.

Table 1. Root ratings of 1998 rootworm resistance field trials

| Line Tested | No. of plants | Root Ratings 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|---|---|
| B73 | 27 | 0 | 0 | 11 | 9 | 4 | 3 |
| W64A | 28 | 0 | 0 | 11 | 13 | 2 | 2 |
| B73 X Tripsacorn | 19 | 0 | 1 | 1 | 2 | 4 | 11 |
| B73 X (W64A X Sun Star) | 16 | 0 | 4 | 6 | 4 | 1 | 1 |
| W64A X Sun Star | 12 | 3 | 3 | 5 | 0 | 0 | 1 |
| W64A X Tripsacorn | 30 | 1 | 11 | 6 | 8 | 4 | 0 |

Compared to the maize inbreds which had root ratings ranging from 3 to 6, at least one resistant plant with a root rating of 1 or 2 was recovered in all of the hybrid lines tested. Lines that demonstrated greatest resistance were (W64A X Sun Star) and (W64A X Tripsacorn). The field test confirmed that the natural corn rootworm resistance trait observed in growth chamber bioassays is also expressed under field conditions.

### Growth chamber bioassays to test a natural resource for corn rootworm resistance

--Eubanks, MW

T. F. Branson (Ann. Entomol. Soc. Amer. 64:861-863, 1971) reported that *Tripsacum dactyloides* is resistant to corn rootworm. A bridging mechanism for moving *Tripsacum* genes into corn has been achieved through wide cross hybrids between *Tripsacum dactyloides* and *Zea diploperennis* (M. W. Eubanks, Econ. Bot. 49:172-182, 1995). Efficacy of this genetic bridge for conferring natural rootworm resistance to corn has been demonstrated through a series of insect bioassays (M. W. Eubanks Amer. J. Bot. (suppl.):84:116, 1997; MNL 70:22-23, 1996; MNL 68:40-41, 1994). Results of insect bioassays conducted in 1998 as part of a recurrent selection program for development of isogenic corn lines that have natural rootworm resistance are reported here. The work is supported by NSF grant no. 9801386.

Thirteen *Tripsacum-Z. diploperennis* hybrid lines and two corn inbreds have been tested in growth chamber bioassays at the Duke University Phytotron. Included were three hybrid lines, Sun Dance, Sun Star, and 20A (a hybrid not previously tested for rootworm resistance); two corn inbred lines B73 and W64A, and ten corn X *Tripsacum-Z. diploperennis* hybrid lines including 97-1 X 97-5, its reciprocal cross and backcrosses of this line to 97-5 and Tripsacorn; 97-1 X 97-3; W64A X Tripsacorn and a backcross of this line to Tripsacorn; B73 X Tripsacorn and a backcross of this line to Tripsacorn, and T33 X Tripsacorn. The research design was four replicates in a randomized block. Plants were grown in 4.5-inch diameter pots with nylon cloth covering the bottom of the pots to prevent larval escape out the holes in the bottom of the pots. Out of 482 plants, 438 germinated. Eighteen of the 438 were albino seedlings and died. Of the remaining 420 plants, 303 were infested with newly hatched Western corn rootworm larvae at three weeks post germination. In replicate one, each plant was infested with 100 larvae; in replicates two and three, treatment consisted of 70 larvae per plant, and in the fourth replicate each plant was infested with 50 larvae. At the end of three weeks after infestation, all plants were harvested. The roots were carefully washed, then scored using the 1-6 Iowa rating scale (Hills and Peters ): 1 = no damage or only a few minor feeding scars; 2 = feeding scars evident, but no roots eaten off to within 1.5 inches of the plant; 3 = several roots eaten off to within 1.5 inches of the plant, but never the

Table 1. Root Ratings in NSF Phase II Insect Bioassay #1

| Line | Treated | Controls | Root Ratings 1 | 2 | 3 | 4 | 5 | 6 | Plants repotted |
|---|---|---|---|---|---|---|---|---|---|
| **Tripsacum-diploperennis $F_2$ hybrid lines** | | | | | | | | | |
| Sun Dance | 9 | 4 | 0 | 0 | 0 | 9 | 0 | 0 | |
| 20A | 8 | 4 | 0 | 0 | 4 | 4 | 0 | 0 | |
| Sun Star | 8 | 4 | 0 | 0 | 2 | 5 | 1 | 0 | |
| **Corn $F_3$ and $F_5$ hybrid progeny** | | | | | | | | | |
| W64AXSS#25 | 20 | 8 | 0 | 0 | 6 | 14 | 0 | 0 | |
| 97-7 | 19 | 8 | 0 | 0 | 1 | 18 | 0 | 0 | |
| 97-1 X 97-5 | 35 | 8 | 3 | 4 | 10 | 18 | 0 | 0 | 2 |
| 97-5 X 97-1 | 32 | 8 | 0 | 2 | 6 | 23 | 1 | 0 | |
| 97-5 X 97-1 BC1 | 23 | 8 | 0 | 2 | 7 | 14 | 0 | 0 | |
| (97-5 X 97-1)XTC | 22 | 8 | 0 | 4 | 5 | 13 | 0 | 0 | |
| 97-1 X 97-3 | 21 | 7 | 0 | 1 | 5 | 15 | 0 | 0 | |
| NC64TC | 21 | 7 | 0 | 3 | 6 | 12 | 0 | 0 | |
| NC64TC BC3 | 10 | 2 | 3 | 0 | 1 | 6 | 0 | 0 | 1 |
| B73 X TC#38 | 16 | 8 | 2 | 3 | 2 | 9 | 0 | 0 | 2 |
| B73 X TC#22 | 13 | 0 | 2 | 0 | 6 | 5 | 0 | 0 | 2 |
| B73 X TC#34 | 14 | 8 | 3 | 0 | 5 | 6 | 0 | 0 | 3 |
| 73TC X TC | 6 | 3 | 4 | 0 | 1 | 1 | 0 | 0 | 2 |
| T33 X TC | 12 | 8 | 0 | 1 | 3 | 8 | 0 | 0 | |
| **Corn inbred lines** | | | | | | | | | |
| B73 | 6 | 6 | 0 | 0 | 1 | 5 | 0 | 0 | |
| W64A | 7 | 8 | 0 | 0 | 0 | 7 | 0 | 0 | |

equivalent of an entire node of roots destroyed; 4 = one node of roots completely destroyed; 5 = two nodes of roots completely destroyed; 6 = three nodes of roots completely destroyed. Plants that have a root rating of 1 or 2 are resistant. Any live larvae were counted and placed in 1 dram glass vials containing 95% ETOH that were labeled by individual plant. After scoring the roots, twelve resistant plants with a root rating of one (highest level of resistance) were repotted in 10-inch diameter pots and transferred to the greenhouse for selfing to increase seed of resistant lines and for cross pollinating with corn to advance the recurrent selection breeding program to develop rootworm resistant corn lines. Leaf tissue from these plants was also sampled for DNA analysis. Families with highest level of resistance identified in this bioassay are (97-1 X 97-5), NC64TC, and (B73 X TC). The root rating results are reported in Table 1.

### Comparative analysis of the genomes of *Zea* and *Tripsacum*

--Eubanks, MW

To test the hypothesis that hybridization between a wild *Zea* and *Tripsacum* (P.C. Mangelsdorf and R.G. Reeves, Texas Exp. Sta. Bull. 574, 1939; M.W. Eubanks, Econ. Bot 49:172-182 1995; M.W. Eubanks, Theor. Appl. Genet. 94:707-712, 1997) was the pivotal event in the origin of maize, DNA fingerprinting of Eastern gamagrass (*Tripsacum dactyloides*), perennial teosinte, (*Zea diplopererennis*), three species of annual teosinte (*Zea mays* ssp. *parviglumis*, *Zea mays* ssp. *mexicana* and *Zea luxurians*), three ancient indigenous races of maize (Nal Tel, Chapalote, and Pollo), and a modern maize inbred line (W64A) was conducted (see Table 1). The basic operating assumption of this experiment is that if maize

Table 1. Taxa Assayed by DNA Fingerprinting

| Taxa | Accession/Source | Provenance |
|---|---|---|
| *Tripsacum dactyloides* (2n=36) | DHT 62-237/Nat. Germplasm Repository, Miami, FL | Eastern U.S. |
| *Tripsacum dactyloides* (2n=72) | Indiana Univ., Bloomington, IN | Eastern U.S. |
| *Zea diploperennis* | Iltis et al. #1250/H.H. Iltis, Univ. WI-Madison | Jalisco, Mexico |
| *Zea mays* ssp. *parviglumis* | P.I. 331785, USDA (NCRPIS), Ames, IA | Mexico |
| *Zea mays* ssp. *mexicana* | P.I. 566683, USDA (NCRPIS), Ames, IA | Mexico |
| *Zea luxurians* | P.I. 306615, USDA (NCRPIS), Ames, IA | Guatemala |
| Chapalote | M.M. Goodman, NCSU, Raleigh, NC | Mexico |
| Nal Tel | M.M. Goodman, NCSU, Raleigh, NC | Mexico |
| Pollo | M.M. Goodman, NCSU, Raleigh, NC | Mexico |
| W64A | J. G. Coors, Univ. WI-Madison | U.S. |

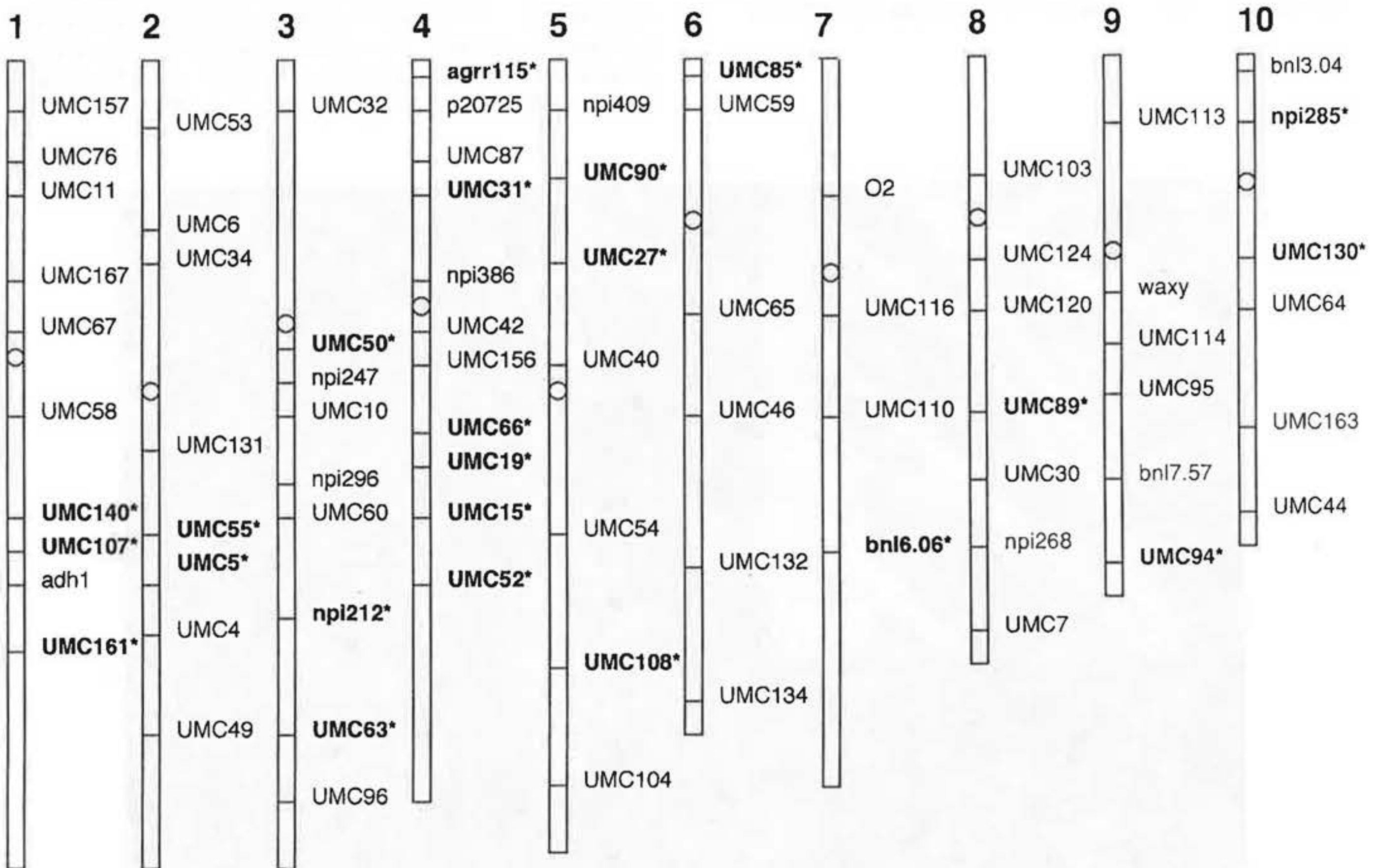



Figure 1. RFLP markers in *Tripsacum-Zea* genome comparison. * indicates alleles shared by maize and *Tripsacum* not present in teosinte.

arose via intergeneric hybridization between *Tripsacum* and a wild *Zea*, alleles shared by *Tripsacum* and maize that are not present in any of the teosintes should be present. To the contrary, if the phylogeny of maize is monophyletic with descent traced directly from one of the annual teosintes (G.W. Beadle, J. Hered. 30:245-247, 1939; W.C. Galinat, Ann. Rev. Genet. 5:447-478, 1971; H.H. Iltis, Science 222:886-893, 1983; J. F. Doebley, Econ. Bot. 44 (suppl):6-27, 1990), any alleles shared by maize and *Tripsacum* should also be found in the ancestral teosinte.

Approximately 20 grams of leaf tissue were harvested from five or more individuals of each taxa and shipped frozen on dry ice to Linkage Genetics, Inc., Salt Lake City, Utah, for DNA analysis. Methods for isolation, restriction enzyme digestion, electrophoresis, Southern transfer, labelling, and hybridization with restriction fragment length polymorphism probes are described by T. Helentjaris et al. (Pl. Mol. Biol. 5:761-769, 1985; Theor. Appl. Genet. 72:761-769, 1986). The restriction enzymes employed to digest the total bulked genomic DNA of each taxa were *Eco*RI, *Hin*dIII and *Bam*HI. Proximal locations of the seventy-four molecular markers that have been mapped to the ten linkage groups of

maize are indicated in Figure 1. Plant material was grown from seed in a greenhouse or obtained from clonal materials.

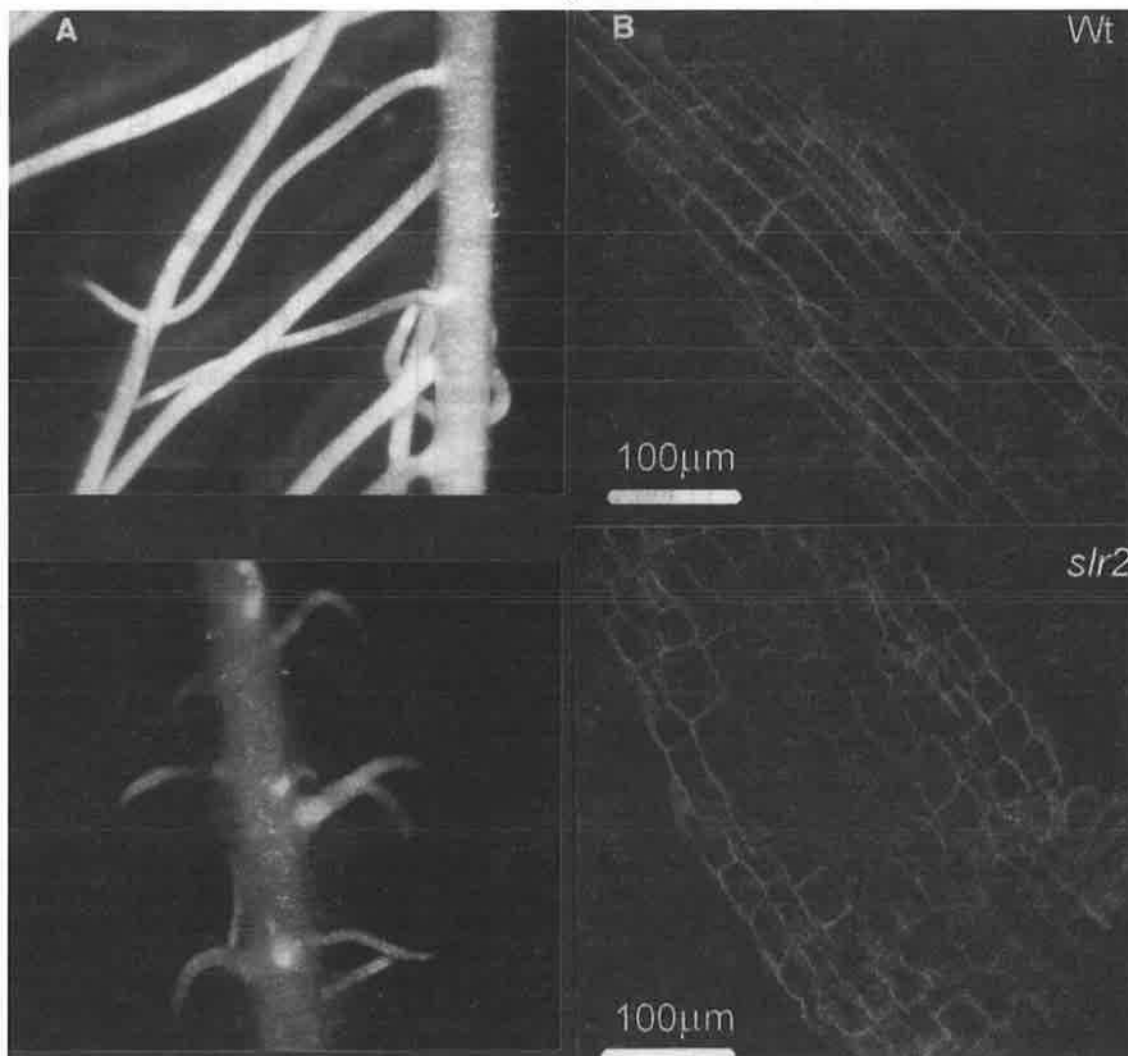
Analysis of the autoradiographs revealed twenty-one loci (indicated by asterisk in Fig. 1) where *Tripsacum* shares alleles with one or more of the maize lines that are not present in any of the teosintes tested. These preliminary results lend support to the hypothesis that the evolutionary history of maize is reticulate with descent via hybridization between *Zea* and *Tripsacum*. An alternative explanation for these results could be lineage sorting of alleles from a common ancestor. This seems unlikely, however, because the frequency of >28% alleles shared between *Tripsacum* and maize that are not found in teosinte is higher than would be expected to explain these results by lineage sorting . Expanded sampling of more taxa with more probes is needed to rigorously test the hybrid origin of maize hypothesis. This research was supported by National Science Foundation grant no. 9660146.

FREIBURG, GERMANY
Albert-Ludwigs-University

**The newly isolated root mutant *slr2* is affected in lateral root elongation**

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

The genetic control of lateral root formation has been studied in great detail in the dicotyledonous plant *Arabidopsis*, but in contrast very little is known about its genetic basis in monocotyledonous plants like maize. The only mutants isolated so far in maize are *lrt1*, which is transiently defective in lateral root initiation during early postembryogenic root growth (Plant J. 16:247-255, 1998), and *slr1*, which forms only short lateral roots at the young seedling stage (MNL 72:30, 1998). We now report the isolation of *slr2*, which is very similar in its phenotype to *slr1* and leads to the formation of short lateral roots as a result of an impaired cell elongation mechanism. This is demonstrated in the Figure which shows in part A a close-up view of a wt primary root (upper part) in comparison with a *slr2* primary root with shorter lateral roots,

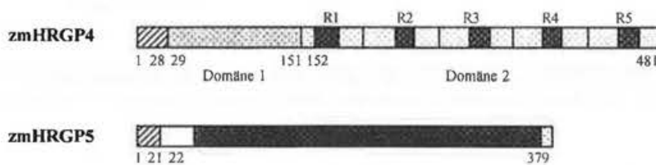



A

and in part B, a confocal laser scanning microscopic picture of relevant tissue depicting the cell size differences. The formation of short lateral roots is specific for the early growth periods of the primary- and lateral seminal roots. Later phases of these roots, as well as lateral root formation on crown roots, are not affected. *Slr2* does not show the pale yellow appearance of the first two leaves typical for *slr1*. The mutant *slr2* is not allelic to *slr1* and *slr1/slr2* double mutants display a non-additive phenotype, indicating an interaction of the two loci. We are now in the process of performing allelism tests of *slr1* and *slr2* with further tentative mutants recently isolated in our lab which are defective in lateral root growth and morphology.

**Structure of two new types of proline-rich proteins**
--Wulff, D, Feix, G

Proline-rich proteins are among the major protein components of plant cell walls, and the different gene families identified so far display cell type specific expression patterns. So far, three different proline rich putative cell wall proteins have been described in maize. We now report the identification of two additional proteins, tentatively designated HRGP4 and HRGP5, the amino acid sequence of which has been deduced in the case of HRGP4 from the nucleic acid sequence of a cDNA isolated from a leaf cDNA-library, and in the case of HRGP5 from the sequence of a gene isolated from a (fixII genomic library. A schematic representation of the structure of the two isolates is given in the Figure. Both protein structures display at their amino-terminus an export specific

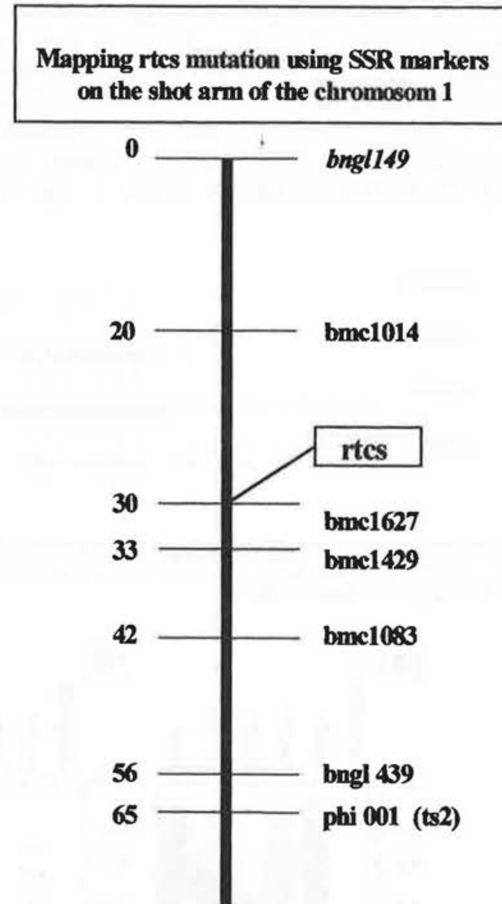



signal sequence which correlates often with the transport of the proteins into the cell wall. The second domain of HRGP4 from amino acid 152 to 481 is composed of 5 very similar proline-rich repeats consisting largely of proline, lysine and histidine. The central part of these repeats (indicated by the stippled boxes) is almost identical with the main part of the second proline rich protein (dark stippled part) which contains the amino acid sequence block proline-glutamate-proline-lysine repeated 45 times. A particular role of the new proteins in specific cell wall complexes is anticipated and currently under investigation.

FREIBURG, GERMANY
Albert-Ludwigs-University Freiburg
RASTATT, GERMANY
Südwestsaat

**Mapping of the root specific *rtcs* locus with the help of microsatellites**
--Krebs, O, Feix, G, Beaumont, V, Schwall, M

The monogenic root mutant *rtcs* is completely deficient in the formation of all nodal roots (lateral seminal-, crown- and brace roots). The defect of the mutant is effective very early in root initiation since no primordia formation for the affected root types can be detected microscopically in relevant tissue slices. This loss of function feature of *rtcs* is of particular interest, and work towards the isolation of the affected gene is underway. The mutant locus had previously been mapped to the short arm of chromosome 1 with the help of a cosegregating RAPD marker (Hetz, W. et al., Plant J. 10:845-847, 1996). We have now performed a more extended mapping analysis by working with microsatellites specific for chromosome 1, which have been found to be polymorphic for *rtcs* plants in the F2 mapping population of A632 x rtcs. The outcome of this work is summarized in the map segment of chromosome 1 shown in the Figure. It can be seen from the marker posi-




tions that *rtcs* is located 10 cM from *bmc1014* and 3 cM from *bmc1083* while *bmc1627* has shown no recombination with *rtcs* (130 plants tested). This result is now used as a basis for a fine mapping of *rtcs* with the AFLP procedure.

HAMBURG, GERMANY
University of Hamburg

**Fertilisation regulated ribosomal protein genes contain a $(GCC)_n$ motif in their 5' UTRs**

--Dresselhaus, T, Lörz, H

From cDNA libraries of unfertilised maize egg cells (Dresselhaus, T et al., Plant J 5:605-610, 1994) and in vitro zygotes, 18 h after gamete fusion (Dresselhaus, T et al., Plant Mol Biol 31:23-34, 1996), we have isolated more than 50 genes, whose expression is up- or downregulated after in vitro fertilisation. Among the isolated cDNAs were four encoding novel ribosomal proteins (r-proteins) from maize: ZmRPS21A, ZmRPS21B, ZmRPL39 and ZmRPP0, respectively (Accession No. X98656, Y09636, X95458 and Y07959). Relatively high transcript amounts of *ZmrpS21*A, *ZmrpL39* and *ZmrpP0* were detected in unfertilised egg cells. After *in vitro* fertilisation (IVF) transcript amounts of *ZmrpS21*A and *ZmrpL39* even increased, while transcript amounts of *ZmrpP0* strongly decreased (Figure 1). Transcript for ZmRPS21B was not detected in egg cells and zygotes, whereas this is the predominant transcript of the *rpS21* gene family in other tissues of maize (data not shown). The homologous ribosomal genes (r-genes) from rice are regulated in a coordinated fashion during the somatic cell cycle and are strongly induced during G1 phase of the cell cycle (data not shown).

The two subunits of eukaryotic ribosomes contain more than 80 different r-proteins. Assembly of ribosomes in active nucleoli was shown to be regulated by coordinated gene expression in yeast (Mager WH and Planta RJ, Biochem Biophys Acta 949:1-15, 1988). In animals, this is different and translational controls are predominant (e.g. Aloni R et al., Mol Cell Biol 12:2203-2212, 1992). Coordinated translational control of r-protein mRNA suggests that common regulatory elements are involved. The mRNA encoding r-protein L32 from murine spp. redistributes from untranslated subribosomal particles into polysomes after mitogenic activation of quiescent cells (Kaspar RL et al., J Biol Chem 267:508-514, 1992). The regulatory element for translation was identified in the 5' UTR as a conserved polypyrimidine element. 5' UTRs of ribosomal P-proteins were

Figure 1. Fertilisation-regulated r-genes of maize contain a $(GCC)_n$-motif in their 5' UTR. The 5' UTRs of the four r-genes presented here are aligned. GCC-triplets are numbered and printed in bold italics. The start codons are indicated with +1. The expression of the r-genes 18 h after IVF is indicated as follows: 0: expressed at undetectable levels in egg cells and zygotes, ++: strongly up-regulated and --: strongly down-regulated after IVF.

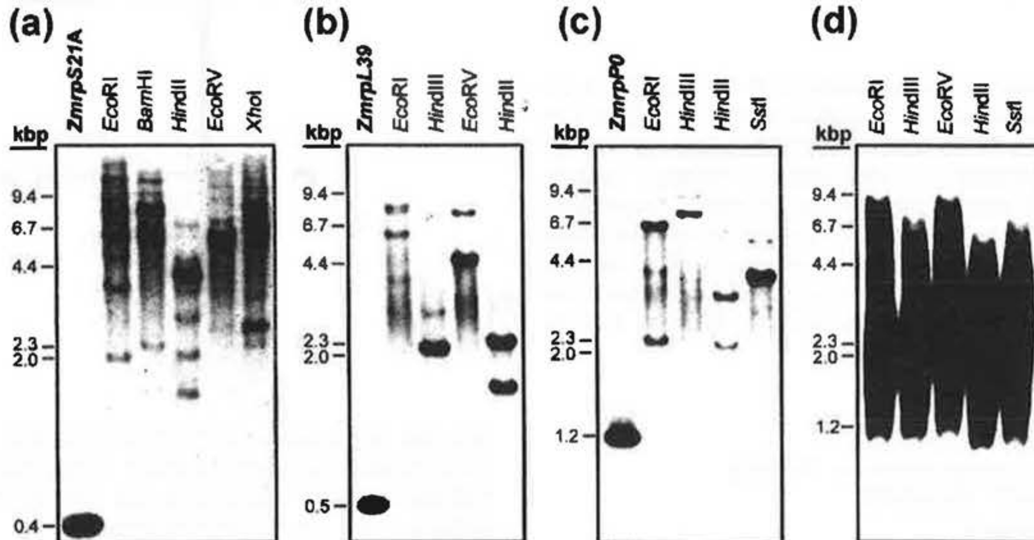



Figure 2. Genomic organisation of maize r-genes *ZmrpS21* A/B, *ZmrpL39*, *ZmrpP0* and the $(GCC)_n$-motif. Genomic DNA was extracted from the maize inbred line A188. Each lane contains 10 pg cDNA of the clones indicated, or 10 µg genomic DNA digested with the enzymes shown above each lane. DNAs were separated in 0.8% agarose gels, blotted and hybridised with probes as follows: the ORFs of *ZmrpS21*A (a) and *ZmrpL39* (b) were used as hybridisation probes, respectively, the full length cDNA of *ZmrpP0* was used as probe in (c) and in (d) either the full length cDNAs of *ZmrpS21* A and *ZmrpL39*, respectively, or the PCR amplified $(GCC)_8$-motif of *ZmrpL39* was used as probe for hybridisation.

shown to influence both gene expression and translation efficiency (Bermejo B et al., J Biol Chem 269:3968-3975, 1994). We have identified repetitive GCC triplets in the 5' UTRs of transcripts for maize r-proteins that are stored at relatively high amounts in unfertilised egg cells (*ZmrpS21*A, *ZmrpL39*, *ZmrpP0*) and which are absent in the transcripts for ZmRPS21B, of which no transcript was detected in egg cells and zygotes (Figure 1). As shown by Southern blot analysis, these repetitive triplets are very abundant in the genome of maize (Figure 2d). GCC triplets have been identified also in the human genome on chromosomes X, 11 and 16. They are polymorphic at the FRAXF site on chromosome X, where 6-29 triplets have been found (Parrish JE et al., Nat Genet 8:229-235, 1994). Extensive regions of GCC triplets were also found at the FRAXA site on chromosome X and it was shown that large triplet boxes exclude nucleosomes (Wang Y-H et al., J Mol Biol 263:511-516, 1996). In plants, GCC triplets have been described in the promoter of ethylene-induced genes. Expression is mediated by a $A(GCC)_2$ *cis*-sequence element (Sato F et al., Plant Cell Physiol 37:249-255, 1996). In tobacco, gene expression was induced after virus infection mediated by an inverted GCC motif ($TAAGA(GCC)_2$; Livne B et al., Plant Sci 130:159-169, 1997). Whether the $(GCC)_n$-motif decribed in this paper is needed for nucleosome exclusion, gene activation or repression, transcript processing and stability, or selective mRNA translation after fertilisation remains to be determined.

Southern blot analyses further indicated that the investigated r-genes occur as small gene families in the maize genome. We have digested genomic DNA of the maize inbred line A188 with at least four different enzymes that do not cut within the cDNA sequences. Nevertheless, due to introns within all corresponding genomic sequences (data not shown), the precise number of genes and pseudogenes cannot be predicted. In Figure 2a, seven (*Eco*RV) to ten (*Eco*RI, *Bam*HI) bands of different signal intensities are visible after hybridization with a *ZmrpS21*A probe. Hybridisation with the ORF of *ZmrpL39* resulted in two (*Hind*III) to four (*Eco*RI) bands (Figure 2b). A similar result was obtained with *ZmrpP0*, where two (*Ssf*I) to five (*Eco*RI, *Hind*II) bands are visible (Figure 2c). From these data we suggest that there are probably at least two r-genes of the *rpL39* and *rpP0* families in the maize genome. R-genes of the *rpS21* family are more abundant and at least six genes can be expected. When the full length cDNAs of *ZmrpS21A* or *ZmrpL39* were used as probes, a smear ranging from 1.2 to 9.4 kbp was obtained (Figure 2d). The same result was achieved, when only the $(GCC)_8$-motif of *ZmrpL39* was used as hybridisation probe. Shorter exposure times of this filter revealed numerous bands (data not shown). Whether these signals are originating from repetitive $(GCC)_n$-motifs in the maize genome or from unspecific hybridisation to GC-rich sequences has to await further experimentation, e.g. sequencing of the maize genome.

**Molecular analysis of *In-D*; a semi-dominant allele of the anthocyanin regulatory gene *Intensifier***

--Scheffler, B[1], Rojek, R, Pusch, I, Wienand, U

[1]University, MS, USDA-ARS-NPURU, PO Box 8048, 38677

The *C2* and *White-pollen* (*Whp*) loci both encode for chalcone synthase (CHS). In the aleurone tissue, the *Intensifier* locus (*In*) regulates the production of CHS coming from the *Whp* locus. Kernels that are *in/in* allow CHS to be produced from *Whp* locus, while CHS production from *Whp* is inhibited in *In*/(*In* or *in*) kernels. Franken, P, U Niesbach-Klosgen, U Weydemann, L Marechal-Drouard, H Saedler, U Wienand (1991, EMBO J. 10:2605-2612) investigated the expression of the *Whp* locus and the results from their study indicated that *In* might regulate, directly or indirectly, *Whp* at the post-transcriptional level. Burr, FA, B Burr, BE Scheffler, M Blewitt, U Wienand, EC Matz (1996, The Plant Cell 8:1249-1259) have cloned and characterized *In*. The results from this study show that *In* encodes for a myc-related protein, thus indicating *In* may act as a suppressor of transcription. In order to elicit the regulatory function of *In*, we began an investigation of *In-D*. *In-D* is a semi-dominant mutation of *In* that inhibits overall production of anthocyanins in the aleurone tissue.

Genomic Southern analysis of *In-D* shows that there are minor differences within the coding region of *In* and *In-D*. Sequence analysis of a lambda *In-D* genomic clone uncovered significant differences between the two loci in introns 2 and 6. Intron 6 of *In-D* is1000 bp smaller than *In*'s intron.

A large number of cDNAs were isolated from a lambda Zap cDNA library, constructed from mRNA isolated from the aleurone of 30 DAP *In-D* kernels. Sequence analysis of these clones shows, as in the case of *In*, that *In-D* exhibits missplicing. The missplicing patterns between the two alleles are very similar except for intron 2. *In-D* also exhibits premature polyadenylation in intron 2. One half of the cDNA clones showed high homology to the *In-D* genomic clone, but there were significant differences to indicate that they did not originate from this copy of *In-D*. Upon further genomic DNA analysis of *In-D* and segregating populations from *In-D*/*In* and *In-D*/*in*, it appears that *In-D* consists of two complete, but structurally different, copies of the wild-type allele. Upon sequence analysis of this second copy major differences between the duplicated copies in introns 3, 5, 6 and 7 were detected.

Northern and Western analysis demonstrate that *In-D* is expressed at significantly higher levels when compared to *In*, and that total CHS production is inhibited or delayed.

**Isolation of two new CACTA transposable elements from anthocyanin genes in maize**

--Techen, N, Borchert, L, Scheffler, BE, Wienand, U

Transposable element (TE) specific features are terminal inverted repeats and a target site duplication upon integration. Elements containing identical inverted repeats have been grouped into families. The *En/Spm* element from maize (Pereira, A, H Cuypers, A Gierl, Z Schwartz-Sommer, H Saedler. 1986. EMBO J. 5:835-841; Masson, P, R. Surosky, JA Kingsbury, N Federoff, 1987. Genetics 177:117-137) belongs to the so called CACTA family of elements. The characteristics of the CACTA family, which have been described from different species, is the CACTA terminal inverted repeat and a 3 bp target site duplication upon integration. Other elements of this family are Tam1 from *Antirrhinum majus* (Nacken, WKF, R Piotrawiak, H Saedler, H. Sommer. 1991. Mol. Gen Genet. 228:201-208), Tgm1 from soybean (Rhodes, PR, LO Vodkin. 1985. Proc. Natl. Acad. Sci. USA 82:493-497), and Pis1 from pea (Shirsa, AH. 1988. Mol. Gen. Genet. 212:129-133). We have found two further such elements in maize through the molecular analysis of anthocyanin genes. These new CACTA elements have been identified through the analysis of the *In* (*Intensifier*) gene and the *C2* (Chalcone synthase) mutant *C2-Idf* (*Inhibitor diffuse*). In Intron 6 of *In* a 948 bp long CACTA element and a 3 bp target site duplication have been identified by

comparing the sequences of the alleles *In* and *In-D* (*Intensifier dilute*). The highly repetitive element contains several direct and indirect repeats, and palindromic sequences. The analysis of this transposable element showed a region of 450 bp with a high AT content (69%). Polyadenylated homologous transcripts of this element have been found in young seedlings, roots, embryos and tassels. Six such transcripts were isolated from a cDNA library and sequenced. The sequences are highly conserved (78-88%) and vary between 109 bp and 153 bp in length. Part of the CACTA element (about 500 bp) is also inserted in the *C2, Whp*, and *C1* genes. Insignificant homology was detected when compared to the ILS-1 TE (Alrefai, R, B Orozco, T Rocheford. 1994. Plant Physiol 106:803-804). So far, no regulatory function of this element could be detected. Upon sequence analysis of the *C2-Idf* allele another member of the CACTA element family was discovered. It is integrated into the promoter of one of the three known "*C2* copies" present in the mutant *C2-Idf*. This element is 1165 bp in length.

HONOLULU, HAWAII
University of Hawaii

**Molecular mapping of QTLs conferring resistance to *Sphacelotheca reiliana* (Kühn) Clint**
--Lu, XW, Brewbaker, JL

Head smut (also known as tassel smut) of corn, which is caused by the fungus (*Sphacelotheca reiliana* (Kühn) Clint), has been occasionally a serious disease in North America, Mexico, Australia, New Zealand, S. Africa and Europe. One hundred recombinant inbred lines (RILs), derived from the cross Hi34 (Hawaii inbred) and TZi17 (Nigeria inbred), were used for mapping quantitative trait loci conferring resistance to head smut. Field evaluation for resistance to head smut was carried out by Dr. Nowell in Greytown, South Africa, under natural infection during the 1994 crop cycle. The percentage of smutted plants of each plot was rated on a 1-9 scale (1 = no symptom, 9 = severe symptom). The experimental design was an 11 x 11 double lattice design with two replications. The 121 entries include ten sub-lines each from the resistant parent TZi17 (mean 2.4, range 1 to 4.5) and the susceptible parent Hi34 (mean 5.8, range 1.4 to 8.4), one F1 hybrid (mean 5.5, range 4.5 to 6.5), and one hundred RILs (mean 3.9, range 1 to 9).

One hundred and sixteen RFLP and four SSR markers, well distributed throughout the 10 chromosomes of maize, were used for analysis of RILs under Dr. M. Khairallah at CIMMYT. The constructed linkage map had a total length of 2060 cm and an average spacing of 18.7 cm between markers. Identification of marker loci linked to *S. reiliana* resistance was conducted using single factor analysis of variance. Marker loci on chromosomes 1, 2, 9, 10 carried genes influencing resistance (Table 1). The most influential region for resistance to *S. reiliana* was located in the proximal portion of chromosome 1, where RFLP marker *asg30* showed the highest F value (F=10.24). Two linked markers, *umc167* and *asg75*, were also associated highly with resistance to *S. reiliana*. The data suggest a major QTL for resistance to *S. reiliana* on the short arm of chromosome 1 closely linked to marker *asg30*.

A LOD score of 4.2 was set as the genome-wise threshold value at P<0.05 for identifying putative QTL based on the 1000 permutation test from QTL Cartographer. A scan of all ten chromosomes using composite interval mapping by QTL Cartographer revealed only one peak. This was on the short arm of chromosome 1 close to the marker *asg30*, with LOD score of 5.3 that accounted for 10.6% of phenotypic variation for head smut. The map position was in bin1.04, about 4 cM from marker *asg30* and 16 cM from *umc157*. The data all support the contention that a single major gene, here designated as *spr1*, confers resistance to *S. reiliana*..

Table 1. Loci significantly associated with resistance to corn head smut from single-factor analysis of variance

| Locus | Chromosome bin[a] | F (1, n-2) | Probability (F) |
|---|---|---|---|
| *asg75* | 1.03 | 5.327 | 0.023 |
| *asg30* | 1.04 | 10.168 | 0.002 |
| *umc167* | 1.05 | 4.591 | 0.035 |
| *umc50* | 3.04 | 4.349 | 0.040 |
| *phi022* | 9.03 | 5.115 | 0.026 |
| *csu25b* | 10.00 | 7.408 | 0.008 |

[a]From Maize Genome Database (http://www.agron.missouri.edu)

Seven pairwise interactions (P<0.01) for resistance to *S. reiliana* were detected. All of the three most significant pairwise interactions (P<0.001) involved one marker in the long arm of chromosome 7. The mixture model, including the resistance allele *spr1* and all the three interactions with markers of chromosome 7, could explain up to 60% phenotypic variation (SAS GLM).

**Genetics of resistance in maize to the corn leaf aphid (Homoptera: Aphididae)**
--Lu, XW, Brewbaker, JL

Two sources of resistance to the corn leaf aphid, *Rhopalosiphum maidis* (Fitch) were verified in Hawaii and are the subject of the present genetic study. The aphid has long been investigated as a pest of preferred hosts like maize (*Zea mays* L.), barley (*Hordeum vulgare* L.) and sorghum (*Sorghum bicolor* (L.) Moench). The species is parthenogenetic and viviparous, and it serves as a host of MDMV virus diseases.

Two visual rating methods were adopted in the present study at Waimanalo Experiment Station of the University of Hawaii for the evaluation of resistance to corn leaf aphids. The first rating was based on the degree of infestation in whorl during tassel emergence. The second rating was based on the degree of aphid infestation on ears covered with shootbags about 14 days after pollination. Both rating methods were based on a 1 to 5 scale (1 = no aphids and 5 = severe)

Sub-lines 71 and 72 of commercial inbred Hi38 (*bt1* supersweet) have repeatedly shown no aphid development since resistance was discovered in Nov. 1997. Generation mean analysis was conducted on 6 generations [P1 (Hi38-71, resistant); P2 (G24, susceptible); F1; F2; BC1 and BC2] to determine the type of gene action involved in Hi38-71. Means and their SE for parental, F1, F2, and backcross generations are summarized in Table 1 from the two rating methods. Genetics of resistance is shown to be monogenic and recessive in Hi38-71. Chang and Brewbaker also reported a recessive allele, *aph*, for resistance to corn leaf aphids in AA8sh2 based on generation mean and diallel analyses (MNL 48: 37-38; MNL 50: 31-32). AA8sh2 is among the ancestry of Hi38-71.

High tolerance to the aphids was also observed in inbred Hi34, parents of several sets of RILs at University of Hawaii (MNL 69: 59-60). Molecular markers were used to map the resistance loci to corn leaf aphid in a set of 100 RILs derived from the cross of Hi34 (Resistant) and TZi17 (Susceptible). The two parents dif-

Table 1. The corn leaf aphid ratings for parents Hi38-71 (P1), and G23 (P2), $F_1$, $F_2$, and backcross (B1, B2) generations.

| Generations | Tassels | Ears | Average |
|---|---|---|---|
| P1 | 1.37 ± 0.24 | 1.13 ± 0.20 | 1.25 ± 0.15 |
| P2 | 3.58 ± 1.81 | 3.11 ± 1.10 | 3.34 ± 0.97 |
| F1 | 2.70 ± 1.35 | 3.47 ± 1.69 | 3.10 ± 0.79 |
| F2 | 2.57 ± 0.85 | 2.52 ± 1.47 | 2.55 ± 0.65 |
| B1 | 1.60 ± 0.25 | 2.00 ± 1.50 | 1.80 ± 0.50 |
| B2 | 3.00 ± 0.91 | 2.96 ± 1.29 | 2.98 ± 0.59 |

fered significantly ($P<0.01$) for the resistance to corn leaf aphids in both rating methods. The tassel and ear ratings for corn leaf aphids of Hi34 were 2.36 and 2.54, for TZi17 were 3.72 and 3.76, and for the F1 hybrids (Hi34 x TZi17) were 3.67 and 4.27 respectively. The correlation coefficient between the means of tassel rating and ear rating for the RILs was highly significant ($P<0.01$). The tassel and ear ratings were combined for the identification of QTLs conferring resistance to corn leaf aphids. Composite interval mapping of 100 RILs by 120 marker loci confirmed the presence of a major recessive resistant gene with a LOD peak as 8.2 near the marker *bnl12.09* on the short arm of chromosome 2. This gene explained about 14.3% phenotypic variation for resistance to the corn leaf aphid in the RIL, and may trace back to Antigua 2D from which Hi34 was bred. This gene is presently labeled as *aph2*, and studies are underway to determine its relationship to resistant allele in Hi38-71.

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### Localizing a proximal site on the B chromosome controlling preferential fertilization

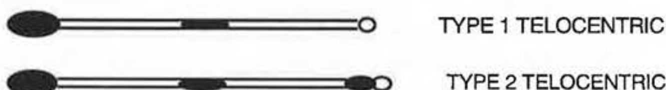
--Carlson, WR

Previous work in this laboratory focussed on localizing B chromosome sites that control nondisjunction. Selection of mutants was helped by a screening technique with the translocation, TB-9Sb. In this procedure, a high nondisjunction genetic background was utilized and the relatively few kernels that showed a lack of nondisjunction for the B-9 were selected as potential mutants of nondisjunction (Carlson, Chromosoma 42: 127, 1973). A reasonable percent of kernels that lacked nondisjunction in the initial screen were found to be "mutants". The mutants turned out to be spontaneous deletions of different regions of the B chromosome, either on the B-9 or the 9-B. This method (in combination with the work of several other laboratories utilizing different techniques) helped localize sites on the B chromosome that were required for nondisjunction.

Selecting in the same way for mutants of preferential fertilization is not feasible. The problem is that there is no way to enrich the population studied for mutants. Instead, every plant in a population must be testcrossed to detect a lack of preferential fertilization. The number of crosses required is prohibitive. An alternate approach is to analyze existing deletions, found in the nondisjunction screen, for a lack of preferential fertilization. It seems likely that some of the deletions should overlap with sites controlling preferential fertilization. The fact that these deletions do not permit nondisjunction presents a problem, since nondisjunction is necessary for a measurement of preferential fertilization. A systematic study of these deletions, therefore, seems impossible. However, in one case the problem can be sidestepped.

A derivative of the B-9 chromosome of TB-9Sb was isolated in 1970 (Carlson, Chromosoma 30: 356). This derivative was shown to be a pseudoisochromosome, which presumably lacks the B short arm (if such an arm exists). The two arms of the pseudoisochromosome differ in the proximal region. One arm lacks the B centric heterochromatin and is incomplete. The other arm appears to be complete (Carlson and Chou, Genetics 97: 379, 1981).

The pseudoisochromosome (also called the original iso) misdivides frequently to give rise to telocentrics. The telocentrics lacking centric heterochromatin are called type 1 telocentrics, while the ones which have centric heterochromatin are called type 2.




Isochromosomes can also be recovered from the telocentrics by misdivision. For the purposes of this report, there are only a few types of chromosomes: the original iso, type 1 telos and isos and type 2 telos and isos. Studies by Kaszas and Birchler (Embo J. 15: 5246, 1996) use a different nomenclature for these chromosomes to indicate the number of misdivision events that each chromosome has undergone.

The type 2 telos are capable of high rates of nondisjunction and can be tested, without difficulty, for preferential fertilization. They show an excess of nondisjunctional kernels in which the B-9 telos are present in the embryo. In other words, they show preferential fertilization. For example, five ears were classified in a cross of *yg/+ bz bz* x 9-B 9-B type 2 telo B-9. The male parent is hemizygous for the B-9. All its viable meiotic products are: 9-B B-9. Nondisjunction is seen as *bz* kernels that give *Yg* seedlings and *Bz* kernels that give *yg* seedlings. The five ears in this cross produced 863 *Yg bz* kernels and 206 *yg Bz* kernels. The predicted ratio of *Yg bz* to *yg Bz* is 2:1 for random fertilization, rather than 1:1, since the tester is heterozygous for *yg/+* and cuts the frequency of *yg Bz* kernels in half. The observed ratio was 4.2:1.0. This ratio would be even greater if the rate of germination were taken into account, since the *bz* class germinated at a rate of 89.5% while the *Bz* class germinated at a rate of 97.8%. (This variation is due to linkage of the detrimental *sh* phenotype with *bz*). The estimated rate of preferential fertilization in the cross, taking germination rates into account, is 69.6%. This is fairly typical for standard TB-9Sb crosses (Carlson, Genetics 62: 543, 1969) and suggests no impairment in functioning of the preferential fertilization system.

The type 1 telos are not so easily tested for preferential fertilization. They are incapable of nondisjunction, due to deletion of the centric heterochromatin. Therefore, the *Yg bz* and *yg Bz* classes do not ordinarily exist in crosses with the telocentric. However, this barrier to testing preferential fertilization can be overcome. It was shown recently that the type 1 telocentric is unstable when present in the hemizygous condition. Crosses of a *yg yg bz bz* tester as female to plants with 9-B 9-B type 1 telo B-9 produced significant numbers of *Yg bz* and *yg Bz* phenotypes. These resulted from misdivision of the centromere rather than nondisjunction (Carlson, MNL 70: 28, 1996). The hemizygote, therefore, can be used in a test of preferential fertilization.

Instability of the type 1 telocentric apparently arises in meiosis, through misdivision of the unpaired chromosome. Misdivision produces an unstable telocentric. If the unstable telocentric is transmitted to the second pollen mitosis, it can undergo a pseudo-nondisjunctional process. The telocentric can misdivide and produce an isochromosome that migrates to one pole. This type of division is similar to nondisjunction, in that both chromatids go to one pole. One of the resultant sperm has a B-type chromosome while the other does not. As a result, preferential fertilization can occur. Cytological results from a cross with the hemizygous telocentric showed that 0-iso disjunction was the main source of *Yg bz* kernels (Carlson, MNL 70: 28, 1996). A comparison of *Yg bz* and *yg Bz* kernels from this cross could give a rate of preferential fertilization for the cross. However, "nondisjunction" occurred at a low frequency, with only 29 *bz* kernels out of 597 (4.9%). Also, many of the *bz* kernels were *yg* in seedling phenotype, indicating 0-0 disjunction and B-9 loss at the second pollen mitosis, rather than misdivision. The *yg bz* class must be discarded in a test of preferential fertilization. Consequently, other crosses were examined to see if more useful genetic data could be obtained.

Perhaps coincidentally, a different method for producing unpaired type 1 telos gave a higher frequency of *Yg bz* and *yg Bz* kernel types. In this procedure, the telocentric is combined with T8-9(4453) and segregation is used to produce the constitution: 9-B 9-8 telo B-9. The B-9 is unpaired in this construct and undergoes frequent misdivision. A cross of *yg yg bz bz* x 9-B (*Wx*) 9-8 (*wx*) telo B-9 was made, and *Wx* progeny selected. Selection of *Wx* removes 9-8 from the data and utilizes only the 9-B B-9 meiotic product, making this cross comparable to the previous one. Classification gave a relatively high rate of *bz* kernels. Eight ears were classified. Among 941 *Wx* kernels, 77 were *bz* (8.1%). These kernels were grown in the field and testcrossed to analyze their constitution. The *Bz* kernels were classified on a sand bench for *Yg* vs. *yg*. Among 799 *Bz* kernels, 111 were *yg*. If we assume that all the *bz* kernels gave *Yg* seedlings (see later), the data show a lack of preferential fertilization because the *Yg bz* frequency is less than that for *yg Bz*. The rate of preferential fertilization calculated from these data is actually below 50%, suggesting a reversal of preferential fertilization. A calculation of preferential fertilization can be made by a) assuming that all *bz* kernels are *Yg* and b) correcting the *yg Bz* data for the rate of germination which is 94.7%. Preferential fertilization is then 77 divided by 77 + 117 = 40%.

The *bz* kernels from the cross were grown in the field, but unfortunately they were not classified at the seedling stage for *Yg* vs. *yg*. Therefore, the possibility of 0-0 disjunction (*yg bz* phenotype) and B-9 loss could not be tested. However, similar crosses with plants of the same genetic parentage showed no B-9 loss among 25 germinated from *bz* kernels. In any case, the existence of a *yg bz* class would only reduce the rate of preferential fertilization further, because this class would be subtracted from total *bz*. The conclusion is that deletion of the centric heterochromatin, or a closely adjacent region, on type 1 telocentrics, blocks preferential fertilization. A proximal region on the B chromosome is needed, therefore, for functioning of the preferential fertilization system.

**Analyzing crosses involving misdivision**

--Carlson, WR

There are peculiarities in crosses that utilize an unpaired type 1 telocentric for studies of preferential fertilization. For example, the rate of *Yg bz* + *yg Bz* kernel types is much lower (20% in the cross analyzed above) than the usual nondisjunctional rates (50-100%). This is not unexpected, since misdivision is a different process from nondisjunction. Another oddity is the fact that a considerable number of kernels in these crosses are variegated for endosperm phenotype (*Bz/bz*). In the cross above, using 9-B 9-8 telo B-9 plants, 65/941 *Wx* kernels were variegated ( 6.9%). When these were germinated, 54 green (*Yg*), 6 yellow (*yg*) and 3 variegated (*Yg/yg*) plants were found. Two did not germinate. The predominance of a stable (green) seedling phenotype suggests an explanation for the variegated kernels. Most likely, the source of endosperm variegation was usually an unstable telocentric, produced by misdivision at meiosis. The telocentric was unstable due to the absence of a telomere at its centric end. It divided equationally at the second pollen mitosis to send an unstable telocentric into each sperm cell. The daughter telocentrics were unstable in the endosperm but stable in the plant, because telomeric healing only occurs in the plant (McClintock, Proc. Natl. Acad. Sci. USA 25: 405, 1939). Two other classes in the cross, *Yg bz* and *yg Bz*, can also be explained by misdivision. The *Yg bz* kernels were analyzed by growing *bz* kernels in the field and testcrossing them, as female parents, to *bz bz*. Testcross data can be used to determine the number of B-9 chromosomes in a plant (Robertson, Genetics 55: 433, 1967). The testcrosses showed that 35 out of 39 plants tested were hyperploid. That is, they had two chromosomes carrying *Bz*. The remaining plants were heterozygotes, with one *Bz*-carrying chromosome. It is believed, although not yet tested, that the two chromosomes in the hyperploid plants were telocentrics. They probably resulted from two unstable telos migrating to one pole and undergoing "healing" to form two stable telos. This type of disjunction (0-2) is distinct from the 0-iso disjunction found earlier for the type 1 telocentric (Carlson, MNL 70: 218, 1996). The difference between crosses that produced 0-2 disjunction and ones that produced 0-iso disjunction may be attributable to genetic background.

It seems very likely that the *yg Bz* kernels from the 9-B 9-8 telo B-9 crosses also resulted from 0-2 disjunction. The *yg Bz* kernels should have received the reciprocal fertilization of the *Yg bz* kernels, with two unstable telos entering the endosperm. Why was the endosperm of *yg Bz* kernels not variegated? Unlike the embryo, the endosperm lacks a system of telomeric healing that could stabilize the telocentrics. The explanation may be that two unstable telocentrics in the same cell readily fuse with each other to produce a stable isochromosome. A stable endosperm phenotype may, therefore, be attributable to fusion at the centromere of two unstable chromosomes.

One final consideration is whether the *Yg bz* and *yg Bz* phenotypes could have been produced by chromosomal instability after fertilization, rather than through a dissimilarity of sperm before fertilization. If an unstable telocentric is produced at meiosis and is transmitted to both sperm of a pollen grain, could the chromosomes be unstable after fertilization? If so, calculations of preferential fertilization would be invalid, since the various recessive phenotypes (*yg Bz*, *Yg bz* and *yg bz*) would result from postfertilization loss rather than an effect on the fertilization process. This idea seems unlikely for two reasons. Considering the *Yg bz* kernels, two telos were found in the embryo in most cases. If B-9 loss accounted for this phenotype, there should be only one telo delivered to both the endosperm and embryo. Second, the varie-

gated kernels that were found in these crosses gave primarily *Yg* seedlings, as noted above. If an unstable telocentric was delivered to both endosperm and embryo in these cases, there is little evidence of telo loss or instability in the seedling.

**Reversal of preferential fertilization**
--Carlson, WR

A surprising finding has emerged from studies of preferential fertilization. In two different crosses, the elimination of preferential fertilization has been accompanied by an apparent reversal of the fertilization pattern. The first study involved a tester stock which acted through the female to block preferential fertilization by B-containing sperm. Crosses of the type *c c sh sh wx wx gl15 gl15* x hyperploid TB-9Sb lacked preferential fertilization. The rate of preferential fertilization ranged from 42 to 45% (Carlson, Genetics 62: 543, 1969, Table 5) rather than the random value of 50%. The values from 1969 are not statistically analyzable for a significant deviation from 50%, since they involve some data manipulation. However, a reversal of fertilization pattern was also found in the cross discussed above with the type 1 telocentric (40% preferential fertilization).

In a third case, elimination of preferential fertilization led to random fertilization, rather than a reversal of pattern. This was found when the B-9 chromosome was present in plants that also carried several standard B chromosomes. In this case, most pollen should contain at least one B-type chromosome in each sperm cell, due to random segregation of the chromosomes following nondisjunction. The rate of preferential fertilization by the B-9 was 52% (Carlson, Genetics 62: 543, 1969, Table 2), very close to a random value. The difference between the first two experiments and the last one can be explained on the basis of activity vs. nonactivity of the system for preferential fertilization. The experiment with *c sh wx gl15* tester involved inactivation of the system by the female parent. The experiment with the type 1 telo also produced inactivation of the system, this time by deletion of a proximal region of the B. However, the experiment which combined TB-9Sb with standard B chromosomes utilized an active system, which functioned in both sperm of the pollen. Therefore, competition between sperm occurred equally and the result was random fertilization.

In sum, inactivation of the preferential fertilization system appears to produce a reversal of preferential fertilization. The reversal could be due to 1) a non-specific effect of excess chromatin on fertilization, or 2) a specific effect of the B chromosome.

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**Changes of DNA-topoisomerase I activity from maize mitochondria under the influence of redox conditions**
--Konstantinov, YM, Tarasenko, VI

Little is known about the regulation of type I DNA-topoisomerase (topo I) activity in plant mitochondria. We have previously reported (MNL 69:63-64, 1995; MNL 70:29-30, 1996; MNL 71:40-41, 1997; MNL 72:33, 1998) on the effect of different redox conditions (including the redox system of glutathione) on mitochondrial genome expression regarding DNA, RNA and protein synthesis in organello. Significant activation of transcription and translation in mitochondria under oxidising conditions and its profound repression under reducing conditions can indicate the existence of a special mechanism of redox regulation of genetic functions in plant mitochondria.

The aim of the present work was to verify our hypothesis that mitochondrial topo I is a redox regulatory enzyme which is regulated by the glutathione system via the reduction/oxidation of a critical disulfide bridge(s). Three-day-old etiolated maize seedlings of hybrid VIR42 MV were used for mitochondria isolation. The method of topo I purification was the same as described in our note from this issue. Topoisomerase activity was measured as previously described (Tarasenko and Konstantinov, Biopolymers and Cell 14:111-116, 1998).

The relaxation activity of mitochondrial topo I has been studied under model changes of redox conditions created by the addition of potassium ferricyanide and oxidised glutathione (GSSG) as oxidising agents, and sodium dithionite and reduced glutathione (GSH) as reducing agents. The addition of both oxidising and reducing agents causes the changes in the activity of topo I from mitochondria (Figures 1-3) but the effects of redox agents depend strongly on the stage of enzyme purification. Figure 1

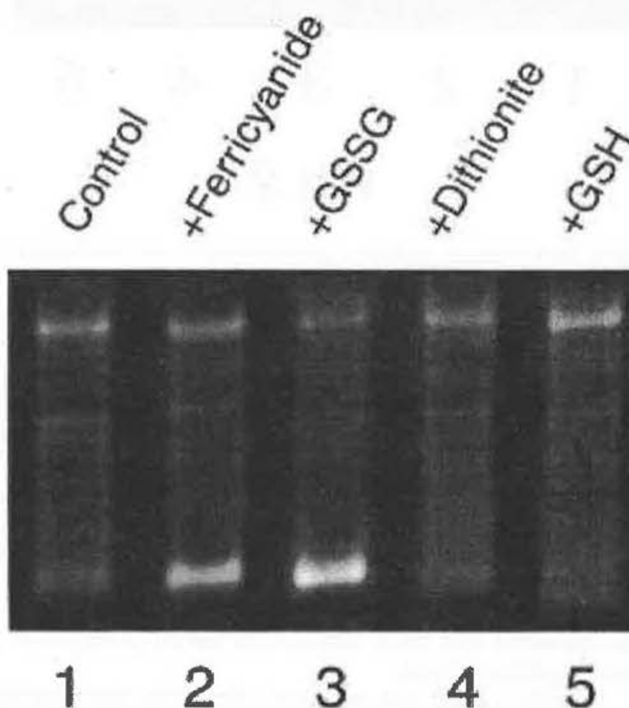


Figure 1. The effect of redox conditions on topoisomerase activity after ammonium sulfate fractionation.

shows an activation of topo I in the presence of dithionite or GSH and its significant repression following the addition of ferricyanide or GSSG after the stage of ammonium fractionation. As regards the effects of these agents on topo I activity after the stage of chromatography on the column with DEAE-Toyopearl ("Toyosoda", Japan), the effects were observed just for oxidised and reduced forms of glutathione (Figure 2). Studying of the

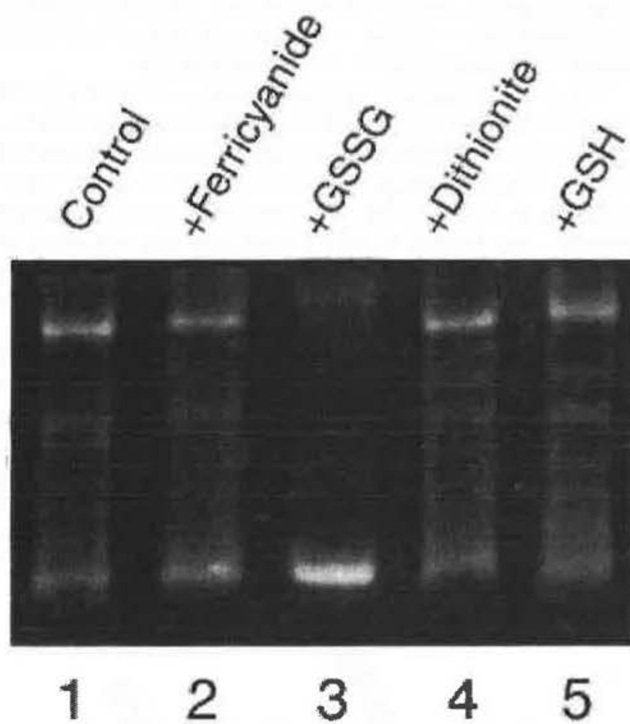


Figure 2. The effect of redox conditions on topoisomerase activity after chromatography on DEAE-Toyopearl.

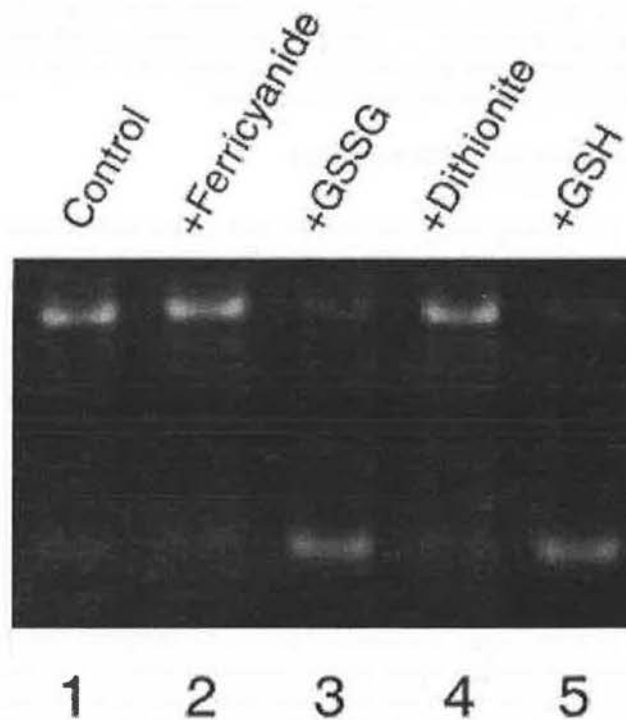


Figure 3. The effect of redox conditions on topoisomerase activity after chromatography on dsDNA-cellulose ("Pharmacia", USA).

redox agents effects on the topo I activity after the final stage of enzyme purification (Figure 3) revealed that both GSSG and GSH caused inhibition of relaxation activity. As a whole, the results of these experiments show the dependence of mitochondrial topo I activity on redox conditions. These results also suggest the existence in mitochondria of special regulatory enzymes involved in reduction/oxidation of cysteine residues in the topo I protein molecule. Figure 4 shows the location of cysteine residues in the amino acid sequence of plant topo I potentially involved in redox regulation of topo I activity.

The data presented demonstrate that type I DNA-topoisomerase from maize mitochondria has the properties of a redox regulatory enzyme.

Financial support from the INTAS (Project Number 98-0522) is acknowledged.

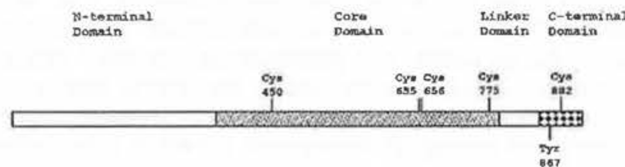


Fig.4

Figure 4. The domain structure of the plant type I DNA-topoisomerase. The scheme was made on the basis of data of Balestrazzi et al., Gene 183:183-190, 1996. Cys - cysteine, Tyr - "active site" tyrosine. Numbers designate amino acid positions.

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**Effects of different types of inhibitors on mitochondrial DNA-topoisomerase I**

--Konstantinov, YM, Tarasenko, VI, Grokhovsky, SL, Sukhanova, AS, Zhuze, AL

Molecular biological and genetic evidence clearly indicates that topoisomerases are important, often essential, cellular enzymes involved in nearly all aspects of DNA structure and metabolism (for review see Berger, Biochim. Biophys. Acta 1400:3-18, 1998). We have previously described (MNL 71:39-40, 1997) some characteristics of type I DNA-topoisomerase (topo I) from maize

mitochondria. The objective of the present study was to investigate the effects of different types of inhibitors on mitochondrial DNA-topoisomerase I activity.

The mitochondria were isolated from 3-day-old etiolated maize seedlings of hybrid VIR42 MV by a standard method of differential centrifugation. Mitochondrial protein was determined by the Lowry method. The purification of topo I from isolated mitochondria included the stages of organelle solubilization, ammonium sulfate fractionation, chromatography on a column with DEAE-Toyopearl, and chromatography on a column with ds-DNA-cellulose as previously described (Tarasenko and Konstantinov, Biopolymers and Cell 14:111-116, 1998). We have studied the effect of some inhibitors currently used to characterize type I DNA-topoisomerase activities from different sources (Figure 1).

It is well known that the topo I inhibitors belong to the ligands with powerful antimicrobial and antitumor activity. The best known of these agents is plant alkaloid camptothecin, which specifically affects the rejoining step of the topo I reaction and therefore stabilizes the covalent protein-DNA cleavable complex. Therefore, camptothecin selectively kills cells in the S phase of the cell cycle because of topo I dysfunction during DNA replication (Chen and Liu, Annu Rev Pharmacol Toxicol 34:191-218, 1994). In our experiments camptothecin does not have any influence on the mitochondrial topo I activity in concentrations of 10 uM to 1 mM. Therefore there is a striking difference in sensitivity to camptothecin between topo I of maize mitochondria and the analogous enzyme from mammalian organelles.

A number of DNA non-intercalating ligands are known to bind to the minor groove of DNA with AT specificity (Zimmer and Wahnert, Prog. Biophys. Mol. Biol. 47:31-112, 1986). These agents are able to inhibit the cleavage activity of eukaryotic topo I (Chen et al., Proc. Natl. Acad. Sci. USA 90:8131-8135, 1993). In this work we investigated the effect of such minor groove-binding agents on topo I activity from mitochondria. Among these types of topo I inhibitors we used such well-known ligands as Hoechst dye 33258, distamycin A, netropsin, and ligands from a new series of high sequence-specific dimeric derivatives of netropsin and distamycin A: bis-netropsins (bis-Nts) and bis-distamycins (bis-Dsts).

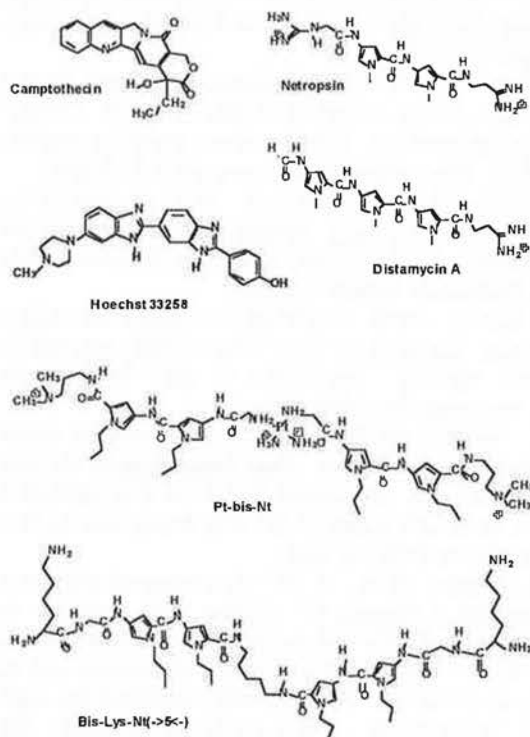


Figure 1. Chemical structures of type I DNA-topoisomerase inhibitors used in this work.

Bisbenzimidazole Hoechst 33258 is a cell permeable, adenine-thymine binding fluorescent dye used to stain DNA during evaluation of the cell cycle, induction of apoptosis by various ligands and cell viability by flow cytometry. It is known that this dye inhibits topo I activity in vitro, like camptothecin. In our experiments Hoechst 33258 is shown to cause a 50% decrease of mitochondrial topo I activity at a concentration of 5 uM. Distamycin A also inhibits topo I (a two-fold decrease of activity at 1.2 uM). It was unexpected, but bis-Dst had lower inhibition activity in comparison with the parent compound distamycin A and caused 50% inhibition of topo I only at 40 uM.

We have also studied the effect of such derivatives of netropsin as bis-Lys-Nt(->5<-) and Pt-bis-Nt which caused a two-fold decrease of topo I activity at concentrations of 15 uM and 3.75 uM, respectively.

We conclude that type I DNA-topoisomerase activity from maize mitochondria is rather sensitive to the action of inhibitors currently used for studying the catalytic mechanism of topo I reaction. These agents also have wide clinical significance due to their efficacy as antimicrobial and antitumor agents. We propose that type I DNA-topoisomerase from maize mitochondria may serve as a convenient model system in a search and testing of new ligands with the highest antitumor and antiviral activities.

Acknowledgements: We would like to thank Dr. N.V.Dorofeev for help with the figure preparation. Financial support from the INTAS (Project Number 98-0522) is acknowledged.

## ITHACA, NEW YORK
## Cornell University

**Formerly restricted interview with Barbara McClintock, now available at Cornell University Archives.**

--Kass, LB, Provine, WB

In 1980, Professor William B. Provine of Cornell University and Paul Sisco, then a graduate student in Cornell's Department of Plant Breeding, conducted a day-long confidential interview with Dr. Barbara McClintock in her laboratory at Cold Spring Harbor Laboratory, Long Island, New York. The interview (Provine and Sisco 1980) was recorded on four cassette tapes, and transcribed by two professional transcribers. It was deposited in the Cornell Archives and, at McClintock's request, classified as restricted until after her death.

The year after McClintock died, Provine invited Lee Kass to collaborate on conducting research for an intellectual biography of Barbara McClintock. Provine gave Kass permission to obtain copies of the transcripts from the Cornell Archives. These served as a basis for further investigations leading to documented materials for the biography. Provine and Kass secured funding from the National Science Foundation to support the research for this project (see MNL 71, page iv).

In 1996 Kass and Provine shared the transcribed interview with Nathaniel Comfort, who also used it for guidance with his

Ph.D. dissertation on McClintock's controlling elements (Comfort 1997). At the 1997 meeting of the Historical Section of the Botanical Society of America, Kass and Provine (1997) reported initial results from their investigations.

Recently Provine and Sisco's interview with McClintock has been unrestricted and is now available to scholars at the Division of Rare and Manuscript Collections, Cornell University Library.

References:

Comfort, N. 1997. *Breakage, Fusion, Bridge. The Discovery and Reception of Barbara McClintock's Controlling Elements.* Ph.D. Dissertation. State University of New York at Stony Brook, Stony Brook, NY.

Kass, L. B. and W. B. Provine. 1997. Genetics in the roaring 20's: The influence of Cornell's professors and curriculum on Barbara McClintock's development as a cytogeneticist. *American Journal of Botany* 84(6, Supplement):123.

Provine, W. B. and P. Sisco. 1980. Interview with Barbara McClintock. August 28, 1980. Cold Spring Harbor Laboratory, Cold Spring Harbor, Long Island, New York. Division of Rare and Manuscript Collections, Carl A. Kroch Library, Cornell University Library, Ithaca, New York.

**Current List Of Barbara McClintock's Publications**

-- Kass, LB

Introduction:

In 1987, Moore edited and reprinted a collection of Barbara McClintock's papers for the Great Books in Experimental Biology Series. McClintock's publications relevant to the discovery and characterization of transposable elements are reprinted in that work. The volume also includes a list of McClintock's published papers under the heading "Numbered List of Publications" (Moore 1987). In researching McClintock's work for a biography that I am writing in collaboration with William B. Provine, I examined the journals, symposia, etc., where all of McClintock's papers appear. In the course of my research I found 14 additional contributions. I subsequently compiled a chronological list of all known contributions published by McClintock, which I now wish to share with Maize Cooperators.

My list updates and amends Moore's (1987) published list and brings the total number of publications to 88. I annotated citations to include, when available, dates when the papers were received, and the month they appeared in print. In some cases the publication date and inclusive pages were revised to reflect accurately these citations. This may be important for scholars who do not have direct access to the publications and might request them on interlibrary-loan. I would be pleased to hear from anyone who may know of any additional references that should be included in this list.

I gratefully acknowledge Dr. Edward Coe for his support with this project. I would be thankful to anyone who can provide missing dates or additional relevant corrections. I may be contacted at the L. H. Bailey Hortorium, Cornell University, Ithaca, NY 14853 or by email at lbk7@cornell.edu.

Reference cited:

Moore, John A. (ed.) 1987. The Discovery and Characterization of Transposable Elements. The Collected Papers of Barbara McClintock. Great Books in Experimental Biology, Garland Publishing Co. New York.

ANNOTATED CHRONOLOGICAL LIST OF THE PUBLICATIONS OF BARBARA MCCLINTOCK
(Prepared by Lee B. Kass)

Note: This list uses dates of publication as referenced by McClintock; i.e., the 1951 Cold Spring Harbor Symposia on Quantitative Biology is cited here as 1951, although the copyright date for the symposium is 1952. Additional pertinent information is enclosed in brackets.

*Appears in Moore's (1987) "Numbered list of publications," pgs. xiii-xv. I add month of publication and submission dates in brackets. I list publications chronologically and add letters following dates for more than one publication in the same year. I add subheadings following titles for Carnegie Institution of Washington Year Book reports, and complete titles for other publications. Inclusive years for Carnegie Year Book reports are in brackets.

**Appears in Moore (1987) and edited for accuracy; i.e., titles, page numbers, or dates corrected.

No Star = additions to "Numbered list of publications" (Moore 1987).

McClintock, Barbara. 1925. A Resume of Cytological Investigations of the Cereals with Particular Reference to Wheat. Ithaca, NY. 52 pgs. plus 25 unnumbered pgs. of tables and bibliographies. [Thesis M. A. Cornell University. A literature review; no original research. Acknowledges Prof. L. W. Sharp.]

*Randolph, L. F. and B. McClintock. 1926. Polyploidy in Zea mays L. American Naturalist LX (666) [Jan./Feb. 1926, received – no date given]: 99-102.

McClintock, Barbara. 1927. A Cytological and Genetical Study of Triploid Maize. Cornell University, Ithaca, New York. 104 pgs. plus 39 unnumbered pgs. of tables, plates, and bibliographies. [Thesis Ph.D. Acknowledges L.W. Sharp and A.C. Fraser.]

*Beadle, G. W. and Barbara McClintock. 1928. A genic disturbance of meiosis in Zea mays. Science 68 (1766) [2 November 1928, received - no date given]: 433. [This became George Beadle's dissertation research project.]

*McClintock, Barbara. 1929a. A cytological and genetical study of triploid maize. Genetics 14 (2) [11 March 1929, received 11 July 1928]: 180-222. [Publication of 1927 Ph.D. thesis. Genetics was issued bimonthly at this time.]

*McClintock, Barbara. 1929b. A method for making aceto-carmin[e] smears permanent. Stain Technology IV (2) [April 1929, received - no date given]: 53-56. [In this publication carmine is incorrectly spelled in the title, throughout the text, and in the citation to Belling 1926.]

*McClintock, Barbara. 1929c. A 2N-1 chromosomal chimera in maize. Journal of Heredity XX (5) [May 1929, received - no date given]: 218. [McClintock annotated the reprint she sent to T. H. Morgan indicating that only one photograph was intended to be published. She apparently submitted two exposures with the intent that the best one would be printed. The citation to Blakeslee and Belling Science, 55, is incorrect; the year, 1924, is missing, and the volume number should be 60

(LX) not 55.]

*McClintock, Barbara. 1929d. Chromosome morphology in Zea mays. Science 69 (1798) [14 June 1929, submitted - no date given]: 629. [The first published ideogram of Zea chromosomes. The chromosomes were identified in the "first division in the microspore" (Mitosis) not at pachytene of Meiosis I as described by some text book authors. The citation for McClintock Genetics, 14, is incomplete. The year, 1929, is missing.]

McClintock, Barbara and Henry E. Hill. 1929e [ABSTRACT]. The cytological identification of the chromosomes associated with the 'R-golden' and 'B-liguleless' linkage groups in Zea mays. Anatomical Record 44 (3) [25 December 1929]: 291. [The paper was "read by title" at the Joint Genetics Sections of the American Society of Zoologists and the Botanical Society of America, held with the AAAS, Des Moines, and Ames, Iowa, December 1929 - January 1930. Resulting manuscript submitted March 1930, and published one year later in Genetics 16: 175-190, March 1931. See McClintock 1933a (pg. 209) for correction of B-lg linkage group association with Chromosome 2 not Chromosome 4.]

*McClintock, Barbara. 1930a. A cytological demonstration of the location of an interchange between two non-homologous chromosomes of Zea mays. Proceedings of the National Academy of Sciences 16 (12) [15 December 1930, communicated 6 November 1930]: 791-796.

McClintock, Barbara. 1930b [ABSTRACT]. A cytological demonstration of the location of an interchange between two non-homologous chromosomes of Zea mays. Anatomical Record 47 (3) [25 December 1930]: 380. [Paper presented on 30 December 1930, at the Joint Genetics Sections of the American Society of Zoologists and the Botanical Society of America, held with the AAAS, Cleveland, Ohio, December 1930 - January 1931. Two weeks prior to these meetings, the results were published in PNAS 16: 791-796, December 1930.]

*McClintock, Barbara and Henry E. Hill. 1931. The cytological identification of the chromosome associated with the R-G linkage group in Zea mays. Genetics 16 (2) [16 March 1931, received 1 March 1930]: 175-190.

*McClintock, Barbara. 1931a. The order of the genes C, Sh, and Wx in Zea mays with reference to a cytologically known point in the chromosome. Proceedings of the National Academy of Sciences 17 (8) [15 August 1931, communicated 7 July 1931]: 485-491. [Communicated the same date and issued as one reprint with Creighton and McClintock 1931. The results reported in McClintock 1931a are necessary for an understanding of Creighton and McClintock 1931, which follows directly in the Journal. These papers were intended to be read together. McClintock 1931a ends with the following statement: "It was desired to present briefly the evidence at this time, since it lends valuable support to the argument in the paper which follows." Creighton & McClintock, 1931 state: "In the preceding paper it was shown that the knobbed chromosome carries the genes for colored aleurone" etc. Unfortunately the "preceding paper" (McClintock 1931a) is neither cited nor referenced.]

*Creighton, Harriet B. and Barbara McClintock. 1931. A correlation of cytological and genetical crossing-over in Zea mays. Proceedings of the National Academy of Sciences 17 (8) [15 August 1931, communicated 7 July 1931]: 492-497. [Communicated the same date and issued as one reprint with McClintock 1931a; see annotation for McClintock 1931a.]

*McClintock, Barbara. 1931b. Cytological observations of deficiencies involving known genes, translocations and an inversion in Zea mays. Missouri Agricultural Experiment Station Research Bulletin 163 [December, authorized 23 December 1931]: 1-30. [McClintock NRC Fellow at Missouri and Cal Tech, investigation conducted at Missouri beginning June 1, 1931; L. J. Stadler suggested the problem and furnished all the material in the growing state.]

McClintock, Barbara. 1932a [ABSTRACT]. Cytological observations in Zea on the intimate association of non-homologous parts of chromosomes in the mid-prophase of meiosis and its relation to diakinesis configurations. Proceedings of the International Congress of Genetics II [24-31 August 1932, preface dated 26 July 1932]: 126-128. [McClintock NRC Fellow at Cal Tech with E.G. Anderson. This paper was presented at the 6th International Congress of Genetics as a Sectional Paper in the session titled "Cytology I, Saturday August 27." McClintock presented paper number 6 of 11 papers. Resulting manuscript submitted in April 1933 and published in ZZMA 19:191-237, September 1933.]

Creighton, Harriet B. and Barbara McClintock. 1932 [EXHIBIT]. Cytological evidence for 4-strand crossing over in Zea mays. Proceedings of the International Congress of Genetics II [24-31 August 1932, preface dated 26 July 1932]: 392. [This was an exhibit that was part of the section on "General Cytology" in the "General Exhibits." The section was organized by Ralph E. Cleland.]

*McClintock, Barbara. 1932b. A correlation of ring-shaped chromosomes with variegation in Zea mays. Proceedings of the National Academy of Sciences 18 (12) [15 December 1932, communicated 2 November 1932]: 677-681. [McClintock NRC Fellow at Missouri with L. J. Stadler; her address is given as U of Missouri; Contribution from Dept of Field Crops, Missouri Agricultural Experiment Station Journal Series No. 355.]

**McClintock, Barbara. 1933a. The association of non-homologous parts of chromosomes in the mid-prophase of meiosis in Zea mays, with 51 figures in the text and plates VII-XII. Zeitschrift fur Zellforschung und microskopische Anatomie 19 (2) [22 September 1933, received 21 April 1933]: 191-237. [McClintock NRC Fellow in the biological Sciences, University of Missouri with L. J. Stadler and California Institute of Technology with E. G. Anderson; investigations conducted at Missouri and at Cal Tech.]

McClintock, Barbara. 1933b. News Items from Ithaca: 11. Brown midrib$_1$ ... . Maize Genetics Cooperation News Letter 4 [18 December 1933]: 2.

McClintock, Barbara. 1933c. News Items from Ithaca: 12. A new narrow leafed character is linked with $a_1$. Maize Genetics Cooperation News Letter 4 [18 December 1933]: 2.

**McClintock, Barbara. 1934. The relation of a particular chromosomal element to the development of nucleoli in Zea mays with 21 figures in the text and plates VIII-XIV. Zeitschrift fur Zellforschung und microskopische Anatomie 21(2) [23 June 1934, received 2 March 1934]: 294-328. [McClintock NRC Fellow in the biological sciences, California Institute of Technology with E. G. Anderson; investigation conducted at Cal Tech. Paper written while McClintock was a Guggenheim Fellow in Berlin and Freiburg, Germany and submitted just prior to leaving Germany.]

*Creighton, Harriet B. and Barbara McClintock. 1935. The correlation of cytological and genetical crossing-over in Zea mays. A corroboration. Proceedings of the National Academy of Sciences 21 (3) [15 March 1935, communicated 9 February 1935]: 148-150. [Written while McClintock was a research assistant in the Department of Plant Breeding, Cornell University (address Botany Department).]

*Rhoades, Marcus M. and Barbara McClintock. 1935. The cytogenetics of maize. Botanical Review. 1 (8) [August 1935, received - no date given]: 292-325. [Written while McClintock was a research assistant in the Department of Plant Breeding, Cornell University.]

McClintock, Barbara. 1936. Cornell University, Ithaca, N.Y. – 8. Mosaic plants in part heterozygous and in part homozygous for a chromosome 5 deficiency. 9. Several inversions ... chromosome 9 ... and chromosome 4, ... detected and isolated by Creighton and [McClintock]. 10. Disjunction studies on interchanges show that sister spindle fiber regions do not separate in I, .... Maize Genetics Cooperation News Letter 10 [4 March 1936]: 5-6.

**McClintock, Barbara. 1937. [ABSTRACT] The production of maize plants mosaic for homozygous deficiencies: Simulation of the $bm_1$ phenotype through loss of the $Bm_1$ locus. [In Abstracts of papers presented at the 1936 meetings of the Genetics Society of America, M. Demerec, Secretary.] Genetics 22 (1) [January 1937, presented 29 December 1936]: 200. [Investigations funded by the Rockefeller Foundation and conducted in Department of Plant Breeding, Cornell University; McClintock's address - Cornell University. In September 1936, McClintock left Cornell to begin her Assistant Professor appointment at U of Missouri. Results reported are part of a manuscript submitted February 1938 and published in Genetics 23: 315-376, July 1938. Note subheadings for sections V and VI in published paper are exactly the same as title of this abstract.]

**McClintock, Barbara. 1938a. [ABSTRACT] A method for detecting potential mutations of a specific chromosomal region. [In Abstracts of papers presented at the 1937 meetings of the Genetics Society of America] Genetics 23 (1) [January 1938, presented 28 December 1937]: 159. [McClintock Assistant Professor of Botany at U of Missouri; results reported here were based on investigations funded by the Rockefeller Foundation and previously conducted in Department of Plant Breeding, Cornell University.]

*McClintock, Barbara. 1938b. The production of homozygous deficient tissues with mutant characteristics by means of the aberrant mitotic behavior of ring-shaped chromosomes. Genetics 23 (4) [July 1938, received 25 February 1938]: 315-376. [Most of work undertaken at Cornell with aid of grant from the Rockefeller Foundation; original material supplied by L. J. Stadler.]

*McClintock, Barbara. 1938c. The fusion of broken ends of sister half-chromatids following breakage at meiotic anaphase. Missouri Agricultural Experiment Station Research Bulletin 290 [July 1938, authorized 12 July 1938]: 1-48. [Continuation of investigations begun at Cornell University between 1934-1936; cites McClintock 1938b.]

*McClintock, Barbara. 1939. The behavior in successive nuclear divisions of a chromosome broken at meiosis. Proceedings of the National Academy of Sciences 25 (8) [15 August 1939, communicated 7 July 1939]: 405-416.

[1940 NO PUBLICATIONS]

*McClintock, Barbara. 1941a. The stability of broken ends of chromosomes in Zea mays. Genetics 26 (2) [March 1941, received 27 November 1940]: 234-282. [Paper published just prior to McClintock's academic leave (1941-1942).]

*McClintock, Barbara. 1941b. The association of mutants with homozygous deficiencies in Zea mays. Genetics 26 (5) [September 1941, received 3 May 1941]: 542-571. [Both the journal article and reprints are dated inaccurately as September 1940.]

**McClintock, Barbara. 1941c [Issued December 1941, Symposium held June 1941]. Spontaneous alterations in chromosome size and form in Zea mays. pp. 72-80. In Genes and Chromosomes - Structure and Organization. Cold Spring Harbor Symposia on Quantitative Biology Volume IX [June 1941, Issued December 1941]. Katherine S. Brehme ed. The Biological Laboratory, Cold Spring Harbor, Long Island, New York. [McClintock was appointed guest investigator for academic year 1941-42, Department of Botany, Columbia University. During the summer of 1941, and from December 1941 through December 1942, McClintock was also guest investigator, Carnegie Institution of Washington, Department of Genetics, Cold Spring Harbor. McClintock resigned from University of Missouri effective August 1942.]

**McClintock, Barbara. 1942a. The fusion of broken ends of chromosomes following nuclear fusion. Proceedings of the National Academy of Sciences 28 (11) [15 November 1942, communicated 22 September 1942]: 458-463.

*McClintock, Barbara. 1942b [1 July 1941 - 30 June 1942]. Maize genetics: The behavior of "unsaturated" broken ends of chromosomes. Phenotypic effects of homozygous deficiencies of distal segments of the short arm of chromosome 9. Carnegie Institution of Washington Year Book No. 41 [Issued 18 December 1942, submitted June 1942]: 181-186. [In the text McClintock cites her work as "McClintock 1941; see bibliography." The reprints do not include the bibliography, which lists three McClintock publications (1941a, b, & c, published in March, September, & December 1941, respectively.]

*McClintock, Barbara. 1943 [1 July 1942 - 30 June 1943]. Maize genetics: Studies with broken chromosomes. Tests of the amount of crossing over that may occur within small segments of a chromosome. Deficiency mutations: Progressive deficiency as a cause of allelic series. Carnegie Institution of Washington Year Book No. 42 [Issued 7 December 1943, submitted June 1943]: 148-152. [McClintock was permanently appointed to the staff of Carnegie Institution of Washington, Department of Genetics, Cold Spring Harbor, in 1943.]

**McClintock, Barbara. 1944a. Carnegie Institution of Washington, Department of Genetics, Cold Spring Harber [sic], Long Island, N.Y. [This report is untitled in the MGCNL. This is a report on deficiencies in Chromosome 9]. Maize Genetics Cooperation News Letter. 18 [31 January 1944, submitted 1943]: 24-26. [The report concludes, " ... the chromosomal breakage mechanism is a "mutation" inducing process which "induces" the same mutant time and again." Moore (1987) cites title as: "Breakage-fusion-bridge cycle induced deficiencies in the short arm of chromosome 9." However, the term "Breakage-fusion-bridge cycle" is not used in this report.]

*McClintock, Barbara. 1944b. The relation of homozygous deficiencies to mutations and allelic series in maize. Genetics 29 (5) [Sept. 1944, received 8 Feb. 1944]: 478-502.

*McClintock, Barbara. 1944c [1 July 1943 - 30 June 1944]. Maize genetics: Completion of the study of the allelic relations of deficient mutants. The chromosome-breakage mechanism as a means of producing directed mutations. Continuation of the chromatid type of breakage-fusion-bridge cycle in the sporophytic tissues. Homozygous deficiency as a cause of mutation in maize. Carnegie Institution of Washington Year Book No. 43 [Issued 15 December 1944, submitted June 1944]: 127-135. [The text cites McClintock 1938, and McClintock 1941, but no references are listed.]

*McClintock, Barbara. 1945a. Neurospora. I. Preliminary observations of the chromosomes of Neurospora crassa. American Journal of Botany 32 (10) [December 1945, issued 14 January 1946, received 28 August 1945]: 671-678.

*McClintock, Barbara. 1945b [1 July 1944 – 30 June 1945]. Cytogenetic studies of maize and Neurospora: Induction of mutations in the short arm of chromosome 9 in maize. Preliminary studies of the chromosomes of the fungus Neurospora crassa. Carnegie Institution of Washington Year Book No. 44 [Issued 14 December 1945, submitted June 1945]: 108-112.

*McClintock, Barbara. 1946 [1 July 1945 – 30 June 1946]. Maize genetics: Continuation of the study of the induction of new mutants in chromosome 9. Modification of mutant expression following chromosomal translocation. The unexpected appearance of a number of unstable mutants. Carnegie Institution of Washington Year Book No. 45 [Issued 13 December 1946, submitted June 1946]: 176-186.]

*McClintock, Barbara. 1947 [1 July 1946 – 30 June 1947]. Cytogenetic studies of maize and Neurospora: The mutable Ds locus in maize. Continuation of studies of the chromosomes of Neurospora crassa. Carnegie Institution of Washington Year Book No. 46 [Issued 12 December 1947, submitted June 1947]: 146-152.

*McClintock, Barbara. 1948 [1 July 1947 – 30 June 1948]. Mutable loci in maize: Nature of the Ac action. The mutable c loci. The mutable wx loci. Conclusions. Carnegie Institution of Washington Year Book No. 47 [Issued 10 December 1948, submitted June 1948]: 155-169.

*McClintock, Barbara. 1949 [1 July 1948 – 30 June 1949]. Mutable loci in maize: The mechanism of transposition of the Ds Locus. The origin of Ac-controlled mutable loci. Transposition of the Ac locus. The action of Ac on the mutable loci it controls. Mutable loci $c^{m-2}$ and $wx^{m-1}$. Conclusions. Carnegie Institution of Washington Year Book No. 48 [Issued 9 December 1949, submitted June 1949]: 142-154.

*McClintock, Barbara. 1950a. The origin and behavior of mutable loci in maize. Proceedings of the National Academy of Sciences. 36 (6) [15 June 1950, communicated 8 April 1950]: 344-355.

*McClintock, Barbara. 1950b [1 July 1949 – 30 June 1950]. Mutable loci in maize: Mode of detection of transpositions of Ds. Events occurring at the Ds locus. The mechanism of transposition of Ds. Transposition and change in action of Ac. Consideration of the chromosome materials responsible for the origin and behavior of mutable loci. Carnegie Institution of Washington Year Book No. 49 [Issued 15 December 1950, submitted June 1950]: 157-167.

*McClintock, Barbara. 1951a [1 July 1950 – 30 June 1951]. Mutable loci in maize. Carnegie Institution of Washington Year Book No. 50 [Issued 14 December 1951, submitted June 1951]: 174-181.

**McClintock, Barbara. 1951b [C. 1952, Symposium held June 1951]. Chromosome organization and genic expression. Pgs. 13-47. In Genes and Mutations, Cold Spring Harbor Symposia on Quantitative Biology, Volume XVI [7-15 June 1951]. Katherine Brehme Warren ed. The Biological Laboratory, Cold Spring Harbor, Long Island, New York. [Copyright 1952. This reference has been cited as 1951 or 1952- see Citation Index. Moore (1987) lists it as 1951. McClintock cites it as 1951. Carnegie Year Book No. 51, Department of Genetics Bibliography, lists it as McClintock 1951.]

*McClintock, Barbara. 1952. [1 July 1951 – 30 June 1952]. Mutable loci in maize: Origins of instability at the $A_1$ and $A_2$ loci. Instability of $Sh_1$ action induced by Ds. Summary. Carnegie Institution of Washington Year Book No. 51 [Issued 12 December 1952, submitted June 1952]: 212-219.

*McClintock, Barbara. 1953a. Induction of instability at selected loci in maize. Genetics 38 (6) [November 1953, issued 20 January 1954, received 14 April 1953]: 579-599.

**McClintock, Barbara. 1953b [1 July 1952 – 30 June 1953]. Mutations in maize: Origin of the mutants. Change in action of genes located to the right of Ds. Comparison between $Sh_1$ mutants. Change in action of genes located to the left of Ds. Meiotic segregation and mutation. Carnegie Institution of Washington Year Book No. 52 [Issued 11 December 1953, submitted June 1953]: 227-237. [Listed in Moore (1987) as a 1954 publication.]

*McClintock, Barbara. 1954 [1 July 1953 – 30 June 1954]. Mutations in maize and chromosomal aberrations in Neurospora: Mutations in maize. Chromosome aberrations in Neurospora. Carnegie Institution of Washington Year Book No. 53 [Issued 10 December 1954, submitted June 1954]: 254-260.

*McClintock, Barbara. 1955a. Carnegie Institution of Washington, Department of Genetics, Cold Spring Harbor, Long Island, N.Y. 1. Spread of mutational change along the chromosome. 2. A case of Ac-induced instability at the bronze locus in chromosome 9. 3. Transposition sequences of Ac. 4. A suppressor-mutator system of control of gene action and mutational change. 5. System responsible for mutations at $a_1{}^{m-2}$. Maize Genetics Cooperation News Letter 29 [17 March 1955]: 9-13.

*McClintock, Barbara. 1955b [1 July 1954 – 1955]. Controlled mutation in maize: The $a_1{}^{m-1}$-Spm system of control of gene action and mutation. Continued studies of the mode of operation of the controlling elements Ds and Ac. Carnegie Institution of Washington Year Book No. 54 [Issued 9 December 1955, submitted June 1955]: 245-255.

**McClintock, Barbara. 1956a [Issued Feb. 1956, Symposium held 15-17 June 1955]. Intranuclear systems controlling gene action and mutation. pp. 58-74. In Mutation, Brookhaven Symposia in Biology, No. 8. Biology Department, Brookhaven National Laboratory, Upton, NY. [No editor listed for this volume. R.C. King, Symposium Chairman. Cited in Moore (1987) as "Issued 1956" but listed chronologically with the 1955 publications. Listed in Carnegie Year Book No. 55 Bibliography as a 1956 publication.]

**McClintock, Barbara. 1956b. Carnegie Institution of Washington, Department of Genetics, Cold Spring Harbor, Long Island, N.Y. 1. Further study of the $a_1$ $^{m-1}$-Spm system. 2. Further study of Ac control of mutation at the bronze locus in chromosome 9. 3. Degree of spread of mutation along the chromosome induced by Ds. 4. Studies of instability of chromosome behavior of components of a modified chromosome 9. Maize Genetics Cooperation News Letter 30 [15 March 1956]: 12-20. [In Moore (1987) the number 9 is missing following the last word of descriptive subtitle. This deletion is also transcribed in Buckner's (1997) bio-biography of McClintock.]
*McClintock, Barbara. 1956c [1 July 1955 - 1 June 1956]. Mutation in maize: Ac control of mutation at the bronze locus in chromosome 9. Control of gene action by a non-transposing Ds element. Continued examination of the $a_1$ $^{m-1}$-Spm system of control of gene action. Changes in chromosome organization and gene expression produced by a structurally modified chromosome 9. Carnegie Institution of Washington Year Book No. 55 [Issued 14 December 1956, submitted June 1956]: 323-332.
**McClintock, Barbara. 1956d [C.1957, Symposium held June 1956]. Controlling elements and the gene. pp. 197-216. In Genetic Mechanisms: Structure and Function, Cold Spring Harbor Symposia on Quantitative Biology, Volume XXI [4-12 June 1956]. K. B. Warren ed. The Biological Laboratory, Cold Spring Harbor, Long Island, New York. [Listed in Carnegie Year Book 56 Bibliography as McClintock 1956.]
*McClintock, Barbara. 1957a. Carnegie Institution of Washington, Department of Genetics, Cold Spring Harbor, Long Island, N.Y. 1. Continued study of stability of location of Spm. 2. Continued study of a structurally modified chromosome 9. Maize Genetics Cooperation News Letter 31 [15 March 1957]: 31-39.
*McClintock, Barbara. 1957b [1 July 1956 – 30 June 1957]. Genetic and cytological studies of maize: Types of Spm elements. A modifier element within the Spm system. The relation between $a_1$ $^{m-1}$ and $a_1$ $^{m-2}$. Aberrant behavior of a fragment chromosome. Carnegie Institution of Washington Year Book 56 [Issued 9 December 1957, submitted June 1957]: 393-401.
*McClintock, Barbara. 1958 [1 July 1957 – 30 June 1958]. The suppressor-mutator system of control of gene action in maize: The mode of operation of the Spm element. A modifier element in the $a_1$ $^{m-1}$-Spm system. Continued investigation of transposition of Spm. Carnegie Institution of Washington Year Book 57 [Issued 19 December, submitted June 1958]: 415-429.
Moreno, Ulises, Alexander Grobman, and Barbara McClintock. 1959a. Escuela Nacional de Agricultura, La Molina, Lima, Peru: 5. Study of chromosome morphology of races of maize in Peru. Maize Genetics Cooperation News Letter 33 [1 April 1959]: 27-28.
*McClintock, Barbara. 1959b [1 July 1958 – 30 June 1959]. Genetic and cytological studies of maize: Further studies of the Spm system. Chromosome constitutions of some South American races of maize. Carnegie Institution of Washington Year Book 58 [Issued 14 December 1959, submitted June 1959]: 452-456.
*McClintock, Barbara. 1960 [1 July 1959 – 30 June 1960]. Chromosome constitutions of Mexican and Guatemalan races of maize: General Conclusions. Carnegie Institution of Washington Year Book 59 [Issued 12 December 1960, submitted June 1960]: 461-472. [Milislav Demerec, Director, Department of Genetics, retired 30 June 1960. He was succeeded by Berwind Kaufman.]
*McClintock, Barbara. 1961a. Some parallels between gene control systems in maize and in bacteria. American Naturalist XCV (884) [Sept.-Oct. 1961, received-no date given]: 265-277.
*McClintock, Barbara. 1961b [1 July 1960 – 30 June 1961]. Further studies of the supppressor-mutator system of control of gene action in maize: Control of $a_1$ $^{m-2}$ by the Spm system. A third inception of control of gene action at the $A_1$ locus by the Spm system. Control of gene action at the locus of Wx by the Spm system. Control of reversals in Spm activity phase. Nonrandom selection of genes coming under the control of the Spm system. Carnegie Institution of Washington Year Book 60 [Issued 11 December 1961, submitted June 1961]: 469-476. [Berwind P. Kaufman, Acting Director, Department of Genetics.]
*McClintock, Barbara. 1962 [1 July 1961 – 30 June 1962]. Topographical relations between elements of control systems in maize: Origin from $a_1$ $^{m-5}$ of a two-element control system. Analysis of $a_1$ $^{m-2}$. The derivatives of bz$^{m-2}$. Carnegie Institution of Washington Year Book 61 [Issued 10 December 1962, submitted June 1962]: 448-461. [Annual Report of the Director of the Department of Genetics: "as this report goes to press the Department is being terminated" (pg. 438). Berwind P. Kaufman, Director, retired on 30 June 1962. Subsequently, McClintock's reports are published in the Annual Report of the Director (Alfred D. Hershey), Genetics Research Unit, Carnegie Institution of Washington. The Unit replaced the former Department of Genetics, active at Cold Spring Harbor from November 1, 1920 to June 30, 1962.]
*McClintock, Barbara. 1963 [1 July 1962 – 30 June 1963]. Further studies of gene-control systems in maize: Modified states of $a_1$ $^{m-2}$. Extension of Spm control of gene action. Further studies of topographical relations of elements of a control system. Carnegie Institution of Washington Year Book 62 [Issued 9 December 1963, submitted June 1963]: 486-493.
**McClintock, Barbara. 1964 [1 July 1963 – 30 June 1964]. Aspects of gene regulation in maize: Parameters of regulation of gene action by the Spm system. Cyclical change in phase of activity of Ac (Activator). Carnegie Institution of Washington Year Book 63 [Issued December 1964, submitted June 1964]: 592-601, plus 2 plates and 2 plate legends. [Cited in Moore (1987) as 592-602.]
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**Changing a duplicated designation for two different male-sterile mutations**

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

A change in gene designations is being made to accommodate the assignment of the same male-sterile mutant designation to two unrelated mutants. In 1998, Trimnell, et al. (MNL 72) assigned a series of mutant designations from *ms29* through *ms31* to describe three new male-sterile mutations. These designations were chosen to "fill-in" some of the numerical gaps in male-sterile mutant designations that have developed over the years. In MNL 72, the gene designation *ms30* was used to describe a mutant previously identified as *ms*-WL87A* that mapped to chromosome 2L. After MNL 72 was published, MCA was notified that a 1998 book entitled "Biology of Male Sterility in Maize" (published in Chinese) included reference to a *ms30* assigned to a male-sterile mutant found by Professor Li Jingxiong that we (MNL 70:30) had given a temporary designation of *ms*-Li89*, pending further mapping and allelism crosses. Unfortunately, this resulted in the same designation for two different male-sterile mutants. To alleviate this problem, I propose that the *ms30* designation continue to be used to describe the Chinese mutant, putting the use of *ms30* in line with what is published in the "Biology of Male Sterility in Maize". Subsequently, this means that *ms*-WL87A* will no longer have any connection to *ms30*. We propose that its new designation be *ms38*, with a reference allele designation of *ms38-WL87A*. (Please note that the original description of *ms*-WL87A* is given in MNL 72:38.) All of this demonstrates the need to coordinate the assignment of new male-sterile mutant designations to avoid further duplications or random numerical designations.

**New chromosome 3L male-sterile mutant *ms37***

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

In 1991 in Johnston, IA, Steve Briggs grew out several hundred rows of Robertson's Mutator families. We observed these families and found over 350 families segregating for male-sterile mutations. One of these families we designated as *ms*-SB177*.

In Johnston during the summer of 1992, we grew remnant seed of *ms*-SB177*. The family again segregated for male sterility, and crosses with inbreds A632 and B73 were made. These crosses were selfed in our Hawaii winter nursery that year, and the A632 segregating ears were grown in our Johnston nursery in 1993. The segregations for the original seed and resulting F2 ears are shown:

| Genotype | Fertiles | Steriles | X2 (3:1) |
|---|---|---|---|
| Original | 21 Fertiles | 14 Steriles | 4.20 |
| A632 Ear #13 | 73 Fertiles | 19 Steriles | 0.93 |
| A632 Ear #14 | 57 Fertiles | 14 Steriles | 1.06 |
| A632 Ear #8 | 52 Fertiles | 23 Steriles | 1.28 |

In our 1995 Johnston nursery, segregating rows of *ms*-SB177* were grown and leaf samples were taken for chromosome mapping. Bulk mapping was run as described in MNL 72:37 except that 19 male-fertile and 20 male-sterile plants were used for the DNA pools. Two RFLP markers on chromosome 3L, *php10-080* and *umc63*, were polymorphic between the two bulks. DNA blots of male-sterile individuals were hybridized with the *php10-080* marker. Four recombinant alleles out of 40 alleles total were detected, indicating that the *ms*-SB177* gene is linked to *php10-080* on chromosome 3L.

Testcrosses were made between *ms*-SB177* and the known male-sterile mutants located on Chromosome 3 (*ms3* and *ms23*) as well as with the unmapped male steriles *ms24* and *ms27*. At least 40 plants were observed for each test-cross, and all test-cross progeny were found to be fertile, indicating *ms*-SB177* was not allelic. We are designating *ms*-SB177* as the reference allele for a new male-sterile mutant, *ms37*.

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**New chromosome 2L male-sterile mutant *ms33* and alleles**

--Trimnell, MR, Patterson, E, Fox, TW, Bedinger, P, Albertsen, MC

Three new male-sterile mutants located on chromosome 2L have recently been identified. All three were found independently of one another.

Earl Patterson of the University of Illinois identified a male-sterile mutation he designated as *ms*-6019*. Earl discovered that other unknown male-steriles he had found were allelic to *ms*-6019*. They were *ms*-6024*, *ms*-6029*, *ms*-6038* and *ms*-6041*. He found that these male-steriles mapped to the long arm of Chromosome 2 by using B-A translocations (see MNL 69:126-128).

Pat Bedinger of Colorado State University found a male sterile she identified as *ms*-Stan1*. In the winter of 1996, Pat provided us with segregating seed to map this unknown male-sterile mutant. The seed was grown in our Hawaii winter nursery and leaf samples were taken for mapping. Bulk mapping was performed as described in MNL 72:37 except that 17 male-sterile and 20 male-fertile plants were used in the creation of the bulks. The RFLP markers *umc36* and *php20-581b*, on the long arm of chromosome 2, were both polymorphic between the two pools. Hybridization of these markers against DNA from the male-sterile individual plants revealed four recombinants for the *php20-581b* probe and one recombinant for *umc36*. This data indicates that *ms*-Stan1* maps on chromosome 2L near the marker *umc36*.

In 1990, Don Morrow at our Garden City, KS Research Center identified a proprietary inbred line segregating for a male-sterile mutation that we named *ms*-GC89A*. He selfed the fertile plants in the row and sent us seed from 12 individual ears, as well as remnant seed from the original segregating ear. We grew one row of all 13 ears in 1992 in our Johnston summer nursery and found that 9 of the 13 rows segregated for male sterility.

| Genotype | Fertiles | Steriles | X2(3:1) |
|---|---|---|---|
| *ms*-GC89A* | 12 | 3 | 0.20 |
| *ms*-GC89A* (8 ears-1990 source) | 108 | 30 | 0.78 |

In the 1995 Hawaii winter nursery, segregating rows of *ms*-GC89A* were grown, and leaf samples were taken for chromosome mapping. Bulk mapping was done using 19 male-sterile and 20 male-fertile plants for the DNA pools. Again, *umc36* showed a polymorphism between the two phenotypic classes and also was found to be 100% linked to the trait when DNA blots from male-sterile individuals were run. Hybridization of the marker *bnl17.14*, also on chromosome 2L, gave 6 recombinants on the male-sterile individual plant DNA blot.

Because all three of these male-sterile mutants mapped to Chromosome 2, testcrosses were made among them to determine if they were allelic to one another. These testcrosses were grown in our Johnston, IA, nursery in 1998. The testcrosses and allelism results are listed below:

| | Female | Male | Progeny | |
|---|---|---|---|---|
| Ear #1 | *ms*-GC89A* Hom | *ms*-6019* Het | 12 Fertiles | 10 Steriles |
| Ear #1 | *ms*-6019* Hom | *ms*-GC89A* Het | 3 Fertiles | 1Sterile |
| Ear #1 | *ms*-GC89A* Hom | *ms*-Stan1* Het | 14 Fertiles | 10 Steriles |
| Ear #1 | *ms*-Stan1* Het | *ms*-GC89A* Het | 22 Fertiles | 8 Steriles |
| Ear #1 | *ms*-Stan1* Het | *ms*-6019* Het | 26 Fertiles | 7 Steriles |
| Ear #1 | *ms*-6019* Hom | *ms*-Stan1* Het | 21 Fertiles | 14 Steriles |

These male steriles also were crossed with the known male steriles located on Chromosome 2 (*ms30, ms31, ms32* (new from Pat Bedinger)), as well as *ms24* and *ms27* (both currently unmapped), and were found not to be allelic.

Since Earl's male-sterile lines were more than likely found before Pat's or ours, we would like to designate *ms*-6019* as the reference allele for a new male-sterile mutant, *ms33*. The other new alleles will be designated as follows: *ms33-6024, ms33-6029, ms33-6038, ms33-6041, ms33-Stan1* and *ms33-GC89A*.

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**New chromosome 7L male-sterile mutant *ms34***

--Trimnell, MR, Patterson, E, Albertsen, MC

Earl Patterson of the University of Illinois sent us several male-sterile mutants that he had found over the years. We have been looking at these for the past few years to further characterize them. One of these Earl had identified as *ms*-6004*. He had found that this male-sterile was located on Chromosome 7L by using B-A translocations. He also found that several other unknown male-steriles were allelic to *ms*-6004*. They were *ms*-6010, ms*-6013* and *ms*-6014*. These male steriles were not allelic to *ms7* or *va1* (see MNL 69:126-128).

After receiving these male sterile mutants, we made reciprocal test-crosses (when possible) between *ms*-6013* and *ms22* (located on chromosome 7S) and the unmapped known male-steriles *ms24* and *ms27*. Progenies of the test-crosses were grown from 1997-1998, and all fertile plants were observed (at least 40 plants per test-cross were observed), indicating that *ms*-6013* is not allelic to *ms22, ms24* or *ms27*. We believe this is a new male-sterile on chromosome 7L, and we are designating *ms*-6004* as the reference allele for a new designation, *ms34*. The alleles described here will be identified as *ms34-6010, ms34-6013* and *ms34-6014*.

**New chromosome 9L male-sterile mutants *ms35* and *ms36***

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

Two new male-sterile mutants on chromosome 9 have recently been identified. Earl Patterson of the University of Illinois found one of these male-steriles and identified it as *ms*-6011*. He determined that several other unknown male-sterile mutants were allelic to *ms*-6011*. They were *ms*-6018, ms*-6027* and *ms*-6031*. By using B-A translocations, he found that they mapped to the long arm of chromosome 9. He also determined that they were not allelic to *ms2* (see MNL 69:126-128).

MRT made testcrosses between *ms*-6031* and the other known male-sterile mutants on chromosome 9 (*ms25* and *ms45*). *ms*-6031* also was crossed with *ms27*, while *ms*-6011* was testcrossed to *ms24* (both unmapped male-sterile mutants). A minimum of 40 plants was grown from each testcross. Fertile plants were observed in all testcrosses indicating that they were not allelic to any known male-sterile mutants.

MCA found the other male-sterile mutant in 1985 in a row of Country Gentleman sweetcorn in our Johnston, IA, nursery. He selfed fertile sib plants and sib pollinated the male-sterile plant that he found. The selfed ears were grown in 1986 in our Johnston nursery where they segregated for male sterility, and where crosses to male-sterile plants were made with B73. In 1993 we re-discovered this male sterile in our inventory, named it *ms*-MS85A* and planted it in our summer nursery. We selfed the F1 B73 crosses and grew the F2 seed in our 1994 Johnston nursery, separating non-sugary and sugary kernels. Segregations for the original selfed ears and the B73 crosses are shown below:

| Genotype | Fertiles | Steriles | X2(3:1) |
|---|---|---|---|
| Country Gentleman Ear #5 | 18 Fertiles | 4 Steriles | 0.55 |
| Country Gentleman Ear #10 | 9 Fertiles | 1 Sterile | 1.20 |
| B73 Non-sugary F2 | 32 Fertiles | 4 Steriles | 3.70 |
| B73 Sugary F2 | 28 Fertiles | 4 Steriles | 2.67 |

In our 1995 Hawaii winter nursery, leaf samples were taken for chromosome mapping. TWF performed bulk mapping as described in MNL 72:37 except that 20 male-fertile and 17 male-sterile plants were used in the creation of the DNA pools. Markers *bnl5.09* and *bnl14.28* on chromosome 9L were both polymorphic between the two pools. Hybridization of these probes to DNA blots of the male-sterile individuals revealed four and one recombinant individuals, respectively, indicating that *ms*-MS85A* is linked to both RFLP markers on chromosome 9L.

We made reciprocal test-crosses (when possible) of *ms*-MS85A* to the other known male-sterile mutants on chromosome 9 (*ms2, ms25, ms45*), as well as to the unmapped male-sterile mutants *ms24* and *ms27*. A minimum of 40 plants was grown of each test-cross. All of the resultant progeny showed fertile plants, indicating that *ms*-MS85A* was not allelic to any of the known male-sterile mutants. Because Earl's Group 2(9L) male steriles (see MNL 69:126-128) also mapped to chromosome 9, reciprocal testcrosses were made. In this case we used *ms*-6031* to cross with *ms*-MS85A*. A minimum of 40 progeny from both crosses were grown producing all fertile plants, indicating that the two male-sterile mutants were not allelic to one another. Because neither of these male-steriles are allelic to any known male steriles,

nor to each other, we believe they are new genetic male-sterile mutants. We are designating *ms*-6011* as the reference allele for *ms35*, a new male-sterile mutant. Its alleles described here will be known as *ms35-6018*, *ms35-6027* and *ms35-6031*. *ms*-MS85A* will be designated as the reference allele for a new male-sterile mutant identified as *ms36*.

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**A monogenic factor causing lemon colour of aleurone in maize grains**
--Mihailov, ME, Chernov, AA

A monogenic factor responsible for lemon coloration of aleurone (often with greenish shade) has been detected. This coloration is masked with yellow endosperm, but it is seen with the naked eye in white endosperm due to *y1* (genotype *y1 y1 y1*). The colour intensity depends on gene dose, a recessive state causes colourless aleurone. The lemon shade is easily observed at harvest, but disappears during drying on the ear side turned to the sun.

This factor (named *Lm1*) was found in the F3 progeny of Ku123 x 2-9m hybrid. A dominant allele originates from Ku123 line, a recessive one is from 2-9m. Mendelian inheritance points out the monogenic inheritance.

Sowing an F3 family heterogeneous for this trait with further self-pollinating resulted in three types of ear:

1) aleurone of all grains is lemon (8 ears);

2) aleurone of some grains is lemon and aleurone of the others is colourless (19 ears);

3) aleurone of all grains is colourless (6 ears).

From types 1 and 3 *Lm1* and *lm1* lines with white endosperm were selected. From these lines four F2 ears were produced, with lemon/white grain ratio 269:89, 239:82, 183:67, 157:62. The classification was satisfactory, only 2-5 grains per ear were doubtful.

The *Lm1*-factor remains unlocated. Linkage with the loci of chromosomes 2 (*ws3*, *lg1*, *gl2*), 6 (*y1*), 9 (*sh1*, *wx1*), or 10 (*R1*) was not detected.

**The development and refinement of maize mutagenesis techniques in Moldova**
--Lysikov, VN

Early development of maize mutagenesis techniques in Moldova was based on the use of conventional methods employing ionizing radiation. The most common procedure was to expose dry maize kernels to X-ray or gamma radiation. Preference was given to gamma radiation since many types of gamma-ray sources (gamma-ray guns) appeared at the time. The development of new methods of exposure to radiation occasionally depended on specific features of certain gamma-ray units.

The majority of gamma-ray sources were charged with radioactive cobalt 60 ($^{60}$Co) and provided hard gamma radiation. The half-life of cobalt 60 is 5.3 years, the gamma radiation flux energy being 1.71 MeV. In recent years, a proportion of commercially available gamma-ray sources have been charged with a cesium 137 ($^{137}$Cs) isotope producing a relatively softer gamma radiation. Use of cesium 137 yielded better results since, unlike cobalt 60, its half-life was longer (some 30 years), making frequent recharges unnecessary. Admittedly, the energy of beta rays emitted by cesium 137 was lower (0.51 MeV).

Our early work largely relied on a comparatively small and low-power unit, GUT- $^{60}$Co-50-1, designed for gammascopy of hardware. Its capacity was some 100 Gy/h. The effective irradiated surface was about 10cm in diameter, i.e. a Petri dish with kernels could be placed in it.

Initially, only dry kernels were irradiated to produce mutations. By this time, the mutagenic irradiation dose was already known for maize kernels (100 to 150 Gy). This was within the so-called critical dose whose lethal values ranged from 50 to 60% (LD - 50-60). This irradiation dose consistently induced heritable changes, i.e. mutations. The absolute number of the resulting mutants was, however, low, about 1 to 5%. Such a low rate of mutations, of which far from all could be classed as favourable mutations of any interest to the breeder, could hardly be a satisfactory one. Therefore, the immediate task was to increase the quantity of irradiated material and, hence, the output of mutant forms. However, the small size of the irradiated spot and the comparatively long period of time required to produce a mutagenic dose (10 to 15 h per sample) were a major obstacle to increasing the amount of irradiated material. These difficulties could be overcome in one of two ways: (1) by purchasing a more sophisticated and powerful gamma-ray source, or (2) by developing new methods for irradiating seeds. In due course, we did purchase more powerful gamma-ray sources: GUBE - $^{60}$Co-4000-1 and RHM -_-20.

In order to save time, new methods of irradiation were proposed, carefully studied and widely applied.

Among the first irradiation methods to be proposed was that of irradiating water-soaked, rather than dry, maize kernels. In doing so, the mutagenic dose was halved, thus reducing the irradiation time per sample by one half and leaving the rate and range of mutations practically unchanged. Subsequently it was demonstrated that by irradiating not only soaked but also slightly sprouted seeds, the required mutagenic dose could be further reduced by a factor of the order of 2, with the rate and range of mutations remaining virtually unchanged.

Concurrently, a method was developed of irradiating not only water-soaked seeds but also seeds presoaked in solutions containing growth-promoting substances, chemical mutagens, amino acids, etc.

It is essentially the need for irradiating larger samples (and, hence, quantities of plant material) while using the same comparatively low-capacity gamma-ray source of the GUM - $^{60}$Co-50-1 type that led us to propose the maize pollen irradiation technique. This technique offered a number of advantages among which the following are worth noting: (1) the considerably extended productive time of the gamma-ray source: the seeds were irradiated prior to sowing, i.e. in spring, and pollen in summer, during maize anthesis, (2) a significantly reduced (by a factor of 2 to 3) required mutagenic dose for irradiating pollen, down to 20 to 30 Gy, and (3) the dramatically increased possibilities for irradiating greater numbers of pollen grains which, being much smaller in size than maize kernels, could be placed in larger numbers in a Petri dish in the gamma-irradiated spot.

It should also be noted that the pollen irradiation technique had another important implication to the geneticist. The sperm in a pollen grain is actually a single cell whereas maize kernels are multicellular structures. Each embryo cell can have a different function. Therefore, the possibility of a mutation occurring in a unicellular

structure, such as the sperm, is much larger than in a multicellular one, such as the embryo. In an embryo, there is no way of knowing in which cell a new growth will occur and whether this particular cell will be directly involved in the formation of a new, i.e. mutant, organism.

For the work on maize pollen irradiation to be successful, a special procedure had to be developed and strictly adhered to. In essence, the procedure was as follows: (1) the plants chosen to be studied in the field were, prior to the study, isolated, i.e. the ear and the tassel were each covered with an isolating parchment bag, (2) the plants and both isolating parchment bags were assigned the same number, so that during pollination the pollen could be applied to its own female generative organs, i.e. stigmata, (3) the pollen was harvested in the field in the morning, immediately delivered to the gamma-ray source and, without delay, irradiated, so that the irradiated pollen could be delivered back to the field and applied to its own stigmata before noon. This is a very important prerequisite since, in Moldova, pollination of maize is generally ineffective after 12 o'clock in the noon when insolation is increased and the air becomes very hot. Selfing is to be strictly ensured.

Before long, the maize pollen irradiation technique was modified. In essence, the modofication consisted in that it became common practice to irradiate immature pollen, right in the tassels, rather than the harvested mature pollen. To this end, 4 to 5 days before heading, the tassels were detached from maize plants by an abrupt movement of the hand and immediately placed in a jar with water. Meiosis was found to be already completed in immature tassels, but tetrads continued to be formed. It was demonstrated, also experimentally, that for irradiating pollen in immature tassels the mutagenic dose could be reduced to 10-11 Gy (1000-1100 r.). This allowed the mutagenic dose to be achieved in immature tassels as soon as within an hour even with our low-power gamma-ray source.

Following pollination, the immature tassels were again placed in jars with water. Each tassel was covered with a parchment "isolator" and assigned the same number as the respective plant in the field. The female generative organs (stigmata) in a plant were also covered with parchment "isolators" in advance, to prevent the foreign pollen from alighting on them. The irradiated tassels were held in jars with water for 4 to 5 days to allow the pollen to reach maturity. Then, for the pollen to shed readily, the covered tassels were exposed to the bright sunlight for a few minutes. Thereupon, the pollen was taken to the field and applied to the respective (same number) plant, ensuring strict selfing. This technique gained wide recognition and was extensively used in various experiments.

Interestingly, it is the technique of immature tassel irradiation that was used as the basis for another, also promising technique, that of incorporating radioactive isotopes in maize pollen grains. For incorporation in pollen grains, solutions of radioactive isotopes of phosphorus 32 ($^{32}P$) and sulfur 35 ($^{35}S$) were employed. Precisely these isotopes were used because both phosphorus and sulfur were present in sufficiently large amounts in generative organs of maize. Another reason for choosing these particular isotopes was that there are significant differences between them. Thus, $^{32}P$ produces rather hard beta radiation of the order of 1.7 MeV, whereas sulfur, on the contrary, yields soft beta radiation of the order of 0.13 MeV.

In essence, the above technique was as follows: immature maize tassels, previously covered with parchment "isolators" (bags) and properly numbered, were placed in solutions of radioactive substances rather than being placed in water. In doing so, preference was given to solutions in which radioactive isotopes were contained in a rather mobile form. Most commonly, these were orthophosphoric acid for $^{32}P$, and $Na_2{}^{35}SO_4$ and other solutions for $^{35}S$.

The immature tassels were held in radioactive solutions for 4 to 5 days. Then the pollen was harvested from them and applied to its own stigmata. The incorporation of radioactive isotopes, e.g. of $^{32}P$, in tassels was detected, using a Geiger-Muller counter, as soon as 15 minutes later. The applied pollen was also examined for the presence of radioactive isotopes using the appropriate instruments. While the radioactive isotopes finding their way into pollen grains were relatively few in number, their irradiation of pollen from within the pollen grains was quite appreciable and the efficiency of the method proved to be very high. This was probably due to the fact that the half-life of $^{32}P$ is 14.3 days and that of $^{35}S$, 81 days, i.e. what actually occurred here was a long exposure to either hard or soft beta radiation.

The mutations resulting from incorporation of radioactive isotopes in pollen grains were quite unique: they exhibited an unusual variation range and, in a number of cases, many mutants with economic traits were identified. Thus, cytoplasmic male sterility mutations were obtained which were of high potential economic value in the maize seed industry.

Another technique of maize seed or pollen irradiation was that of combining low (stimulative) and high (mutagenic) radiation doses. This technique was developed with a view to increasing the relatively low viability of the mutants produced. Moreover, allowance was made for the fact that the irradiated object (seed or pollen grain) was composed of morphologically, physiologically and genetically heterogeneous tissues exhibiting differential responses to various low and high radiation doses. Two versions of the technique were studied: the direct, under which a low (stimulative) dose was applied first to be followed by a high (mutagenic) dose, and the reverse, where a high (mutagenic) dose was applied followed by a low (stimulative) radiation dose.

An important feature of this technique was variation in the timing of low and high dose application. This time interval - a peculiar kind of relaxation - varied from one maize cultivar to another, ranging from 5 min to 1 h in "best" treatments. In most cases, the best results were obtained with the direct method of irradiation combined with the relaxation time around 5 min.

Somewhat unique among the maize irradiation techniques is that of irradiating maize pollen with high (lethal) and superhigh (superlethal) doses. This technique was developed as a result of ingenious studies of Prof. V.S. Syomin (Moldova Institute of Viticulture and Fruit Growing) on interspecific hybrids of fruit crops and came to be known as Pandey method. It is apparent that high (lethal) and superhigh (superlethal) radiation doses all but kill maize pollen making it inviable, i.e. incapable of pollination and fertilization. Very high doses of the order of several hundred and even thousand Gy affect the genetic basis of pollen, i.e. DNA. They actually cause fragmentation of the DNA into stretches of varying length, or oligonucleotides. This peculiar fragmented DNA is incapable of growth within pollen tubes on maize stigmata and, consequently, can not reach the site of fertilization and participate in the fertilization process.

That is the reason why a special procedure was developed for induced delivery of these stretches of DNA (oligonucleotides) to the site of fertilization by means of a peculiar "vector". Acting as

such a vector can be non-irradiated own pollen which readily germinates on maize stigmata. While germinating, this pollen entraps and carries along individual oligonucleotide stretches, occasionally so long that they contain genetic information corresponding to intact genes and, of course, a large number of small stretches carrying little genetic information. Our studies employed marker lines with a set of specific genes. In mixing irradiated donor pollen (containing fragmented DNA) with non-irradiated recipient pollen, incorporated at times are individual small DNA fragments and at other times larger oligonucleotide stretches carrying intact genes. A peculiar process of parallel involvement of these fragments in fertilization occurs. Furthermore, it has been demonstrated experimentally that in this case two processes occur simultaneously and in parallel: transfer of individual genes and experimental mutagenesis. Transfer of intact genes is controlled by using marker lines. The occurrence of new mutations, i.e. mutagenesis is attributable to small DNA fragments acting as mutagenic agents.

Two more maize irradiation techniques are currently being studied with a view to experimental production of new mutations. One involves irradiation of embryos isolated from maize endosperm, the other of female generative organs on plants growing in the field.

As is known, the maize embryo is diploid, whereas the endosperm is triploid. Since very early in its development the diploid embryo depends on the triploid endosperm for its nourishment, the latter may have a pronounced effect on the developing organism. In order to reduce or even eliminate the influence of the triploid endosperm, the embryo was isolated prior to or immediately after irradiation.

The isolated irradiated embryo is placed on a specially selected nutrient medium and grown according to the in vitro tissue culture procedure. From the resulting callus, regenerants are removed which are initially also grown on an artificial medium and then transplanted to pots with soil.

Already early results suggest increased somatic variability and even previously unobserved traits, such as expansion of the embryo scutellum, in some genotypes.

The other novel irradiation technique, that of irradiating female generative organs on a plant growing in the field, became feasible with the purchase of a portable X-ray apparatus, REIS-1. This instrument can be carried in a briefcase and is a battery- or mains-operated unit. It is mounted on a tripod in the field right in front of the chosen plant at the height corresponding to the position of the developing female generative organs (ears) of the maize plant. The apparatus is capable of autonomous operation. The X-ray beam produced by the apparatus is narrow and can be focused on a particular spot. The dose is determined based on the irradiation time.

**The first experiment studying the possibility for elimination of the damaging effect of ionizing irradiation during maize radiation mutagenesis**

--Ikhim, IG, Scorpan, VG, Lysikhov, VN

It is well known that the percentage of mutations appearing during maize radiation mutagenesis is comparatively low due to high elimination of seedlings and young plants in the first generation. It is an explanation relating to comparatively high mutation doses--varying within the range of so called crucial i.e. $LD_{50-60}$ doses.

For increasing maize radiation mutation yields and expansion of their variability spectrum an experiment was carried out for studying the elimination or decreasing of γ-ray damaging effects during irradiation of dry maize seeds.

For obtaining the contrasting effect the paternal form of "Début" hybrid bred by S. T. Chalyk and distributed over districts in Moldova was used, which possessed a slightly lower viability as well as higher radiosensivity. For these aims the routinely used ($LD_{50-60}$) dose of ionizing irradiation was not taken but a slightly higher dose was used instead in the range of 60-70% LD. The maize dry seeds were irradiated by RHM-γ-20 installation with $^{60}Co$ isotope.

For eliminating the $^{60}Co$ γ-ray damaging effect the decision was made to use a number of chemical and physical factors of impact. As the chemical factors 12 substances were utilized: biologically active substances, growth hormones, biostimulators, antioxidants etc. Temperature, U.V. laser hydroelectrolytic products – acidic and basic, i.e. the anodic and cathodic fractions, electromagnetic field of electric current with the industrial frequency and SHF served as factors of physical impact.

Before sowing the maize irradiated seeds were imbibed in solutions of the chemical substances in conventionally used doses and treated with the physical factors also in plant stimulating doses. It is worth noting in addition that LGI-21 U.V. laser on molecular nitrogen was used in the treatment with laser.

The seeds treated in such a way were sown immediately in a field in three-fold replications. In the next field the non-irradiated control was sown as well as the γ-irradiated control but with no treatments with chemical or physical factors.

The results of the experiment were determined by counting initial and total appearance of seedlings in all the treatments. Then during the first month of growing plant counting was carried out weekly since it was in this period that the $^{60}Co$ γ-ray negative damaging effect occurred and great elimination of plants weakened by the irradiation happened to take place.

Immediately after seedling appearance it became clear that the real protective effect was shown only by a single chemical substance and two physical factors. But the really highest protection was provided by only one biological stimulator known in our country as "Crossing" ( a patent of the Institute of Plant Physiology AS MR – professor Atimoshae M.V.). The electromagnetic field of electric current with the industrial frequency gave a weaker but still real protection and the effect of SHF was still slightly weaker.

The effect of "Crossing" was remarkable not only due to the fact that the number of seedlings provided by it was 2.5-fold greater, but it caused the least percentage of plants to perish in the first period of their growth. Therefore a 6-fold greater number of plants was maintained in the treatment with "Crossing" than in the irradiated control. It is also of interest to note such a peculiarity: the total number of plants maintained after the "Crossing" treatment was very close to the plant number in the irradiation – free control.

As was mentioned above the effect of the two physical factors electromagnetic field of current with the industrial frequency and SHF were weaker than that of "Crossing" but were still rather significant. Thus the effect of these factors caused a 2-fold greater appearance of early seedlings than in the irradiated control. This difference was slightly less but still significant for the

total seedlings.

After elimination of the weakened plants during the first month of their life the electromagnetic field of current with the industrial frequency maintained a 2-fold greater number of plants and the one by SHF was 1.5-fold greater than in the irradiated control.

Though these results are obtained for the first time and demand a thorough verification and a more precise definition, already at present they witness that elimination of the $\gamma$-ray damaging effect during maize radiation mutagenesis seems to be quite possible.

**The influence of homozygosis in some segments of the maize genome on recombination frequency in the neighbouring ones**
--Chernov, AA, Mihailov, ME

The influence of the marker loci of chromosomes 1 (*P1*), 2 (*ws3, lg1, gl2*), 6 (*y1*), 9 (*c1, sh1, wx1*), 10 (*R1*) on recombination frequency (rf) in *ws3-lg1-gl2* and *c1-sh1-wx1* segments was studied. The Ku123 x 2-9m hybrid heterozygous for these loci was used. On 227 F2 plants self-pollinated F3 ears were produced. F3 families were used to estimate genotype of F2 plants for marker loci, and in the suitable F3 families rf values were scored.

Only four significant effects were detected: the influence of the *gl2* locus on rf(*ws3-lg1*), *ws3* locus on rf(*lg1-gl2*), *wx1* locus on rf(*c1-sh*) and *c1* locus on rf(*sh1-wx1*) (see Table 1). The table data show that homozygosis for a marker locus leads to increasing rf value in neighbouring chromosome segments. It may be supposed that meiotic crossing over is suppressed in heterozygous chromosome areas, and the level of this suppression depends on the size of the heterozygous interval (the larger the heterozygous interval the greater the level of suppression).

Table 1. Recombination values (mean rf% ± SE) in the different genotypic classes of F2 (Ku123 x 2-9m).

| Segment | Genotype | Number of F3 families analysed | Mean rf, % | The proportion of the factor in total dispersion |
|---|---|---|---|---|
| | *gl2*+/+ | 6 | 20.0±3.3 | |
| *ws3-lg1* | *gl2*+/- | 64 | 12.0±0.5 | 36.6% *** |
| | *gl2*-/- | 10 | 21.5±2.1 | |
| | total | 80 | 13.8±0.7 | |
| | *ws3*+/+ | 5 | 25.8±1.5 | |
| *lg1-gl2* | *ws3*+/- | 64 | 20.8±0.6 | 6.9% * |
| | *ws3*-/- | 2 | 19.0±1.9 | |
| | total | 71 | 21.1±0.6 | |
| | *wx1*+/+ | 13 | 7.4±0.6 | |
| *c1-sh1* | *wx1*+/- | 66 | 5.6±0.3 | 33.0% *** |
| | *wx1*-/- | 8 | 11.8±0.7 | |
| | total | 87 | 6.4±0.3 | |
| | *c1*+/+ | 5 | 28.3±3.2 | |
| *sh1-wx1* | *c1*+/- | 65 | 21.6±0.6 | 10.1% ** |
| | total | 87 | 22.2±0.6 | |

1) * P<0.05, ** P<0.01, *** P<0.001.
2) genotype for *c1* locus in some cases could not be detected because of the interaction with *R1* locus.

**Effect of high temperature on male gametophyte viability of waxy maize**
--Kravchenko, OA, Kravchenko, AN

The aim of our investigation was to study the male gametophyte reaction at the pollen germination and pollen tube growth stages in high temperature stress condititons. The 346 and 502 inbred lines, and their waxy counterparts, as well as their MR3 progeny (obtained from irradiated immature embryos in vitro), were taken as the experimental material. Fresh collected pollen from each genotype was subjected to high (+35 C) temperature treatment for 20 minutes. After that pollen was cultivated on the nutrient medium (developed by Cook F.S., Walden D.B., Can J. Bot. 43:779-786, 1965) for 3 hours at 24 C to determine pollen viability.

As a result, a significant variability of male gametophyte viability among plants of MR3 generation was found. In general, the high temperature treatment resulted in decreasing of maize pollen viability. However, in some cases we have also observed increasing of viability. The reliable effect of genotype and temperature was revealed by two factor analysis of variance (Table 1). The results obtained indicated that pollen viability of MR3 progeny of inbred lines 346 and 502 was more genotype-dependent than that of their waxy counterparts. As the results also show, pollen viability of MR3 progeny of inbred line 502wx1 depends mostly on the temperature factor. The same character of the other waxy counterpart proved to be the most genotype and temperature interaction dependent.

Table 1. Genotype and temperature effect (%) on male gametophyte viability.

| MR3 progeny of inbred lines: | Factors | | |
|---|---|---|---|
| | genotype | temperature | their interaction |
| 346 | 73.44*** | 7.47*** | 16.61*** |
| 346wx1 | 47.76*** | 2.91*** | 56.19*** |
| 502 | 81.72*** | 4.34*** | 9.76*** |
| 502wx1 | 49.78*** | 52.62*** | 22.12*** |

***-P<0.001

**Creating new haploid-inducing lines of maize**
--Chalyk, ST (Cealic, ST)

In a previous publication it was shown that crossing two haploid-inducing lines, KMS and ZMS, resulted in the production of transgressive genotypes whose haploid-inducing capacity was more then two times higher than that of the parental lines (Chalyk et al., MNL 68:47, 1994). It was suggested that the parental lines KMS and ZMS differed from each other by two genes controlling haploid-inducing capacity. Transgressive genotypes carry both genes and are capable of inducing 7 to 9% of haploids.

The objective of our subsequent work was to produce new homozygous haploid-inducing lines which would be superior to the parental lines KMS and ZMS in haploid-inducing ability and would carry marker genes *A1*; *B1*; *C1*; *R-nj* and *Pl1* allowing haploids to be identified both at the stage of mature seeds and at the stage of vegetating plants.

The parental line ZMS carries marker genes *A1*; *C1*; *R-nj*, which enable haploids to be identified at the stage of mature seeds by the anthocyanin coloration of the top of the endosperm and lack of coloration in the embryo. The parental line KMS carries genes *a1*; *B1*; *Pl1* which allow haploids to be identified at the stage of 3 to 5-day-old seedlings by the lack of anthocyanin coloration in roots (Tyrnow and Zavalishina, DAN 276:735-738, 1984). Both of the haploid inducers, ZMS and KMS, were created on the base of Coe's line, Stock 6 (Coe 1959).

We worked with the progeny resulting from selfing of the KMS x ZMS cross. Selfed in each generation were only those plants which exhibited intense anthocyanin coloration. Upon harvesting, the ears were examined for the presence of *R-nj*. Up to generation F8, individual plants were evaluated for two major

traits: (1) haploid-inducing capacity, and (2) intensity of the embryo, endosperm and whole plant coloration. In F2 and F3, the haploid inducing capacity was measured by the number of haploids induced within the lines. Starting with F4, the haploid inducing capacity was evaluated by applying pollen from the selfed plants to diverse genetic material: lines, hybrids, synthetic populations.

In 1998, fourteen lines of the F8 progeny were sown in the field. Their perfect uniformity suggested that all of them were highly homozygous. Subsequent evaluation was, therefore, performed for a line as a whole rather than individual plants. For estimating the haploid-inducing capacity, a mixture of pollen from several plants of the evaluated inducer line was taken. Used as a maternal parent were two inbred lines, MK01y and A619, and a cross between them, MK01y x A619. The results obtained for the best five new haploid inducers and parental lines ZMS and KMS are presented in Table 1.

Table 1. Frequency of seeds with haploid embryo resulting from pollination with pollen from maternal haploid inducers.

| Haploid inducer | Average % of haploids | Maternal genotype | | | | | |
|---|---|---|---|---|---|---|---|
| | | MK01y | | A619 | | MK01y x A619 | |
| | | Total seeds | % haploids | Total seeds | % haploids | Total seeds | % haploids |
| ZMS | 2.3 | - | - | 877 | 2.7 | 1819 | 2.0 |
| KMS | 2.0 | 375 | 2.4 | - | - | 1040 | 1.9 |
| MHI-1 | 5.5 | 623 | 4.9 | 1078 | 5.1 | 646 | 6.8 |
| MHI-2 | 6.1 | 532 | 7.0 | 1234 | 5.9 | 516 | 5.4 |
| MHI-3 | 6.7 | 569 | 11.1 | 1481 | 5.3 | 1547 | 6.4 |
| MHI-5 | 6.5 | 771 | 9.2 | 1445 | 6.9 | 3072 | 5.6 |
| MHI-8 | 6.7 | 867 | 7.4 | 1346 | 7.7 | 2036 | 5.8 |
| An average over 5 MHI lines | 6.5 | | 8.3 | | 6.2 | | 5.9 |

New haploid inducers, MHI (Moldovian Haploid Inducer), were superior to the parental lines ZMS and KMS. Five MHI lines induced an average of 6.5% haploids over three maternal genotypes. The percentages of haploids induced by them were approximately equal, ranging from 5.5% to 6.7%. This was almost three times higher than the frequency of haploids induced by initial haploid-inducing lines, ZMS and KMS. Weak expression of marker genes in MK01y x ZMS and A619 x KMS did not allow us to select haploids from this material, while we had no problem with expression of the marker system when our new haploid-inducing lines were applied. The new inducers did not differ from one another phenotypically: the plants were dark violet; the *R-nj* gene was clearly expressed in the seeds.

The maternal genotype had a valuable effect on the frequency of haploids produced. The highest percentage of haploids was obtained with line MK01y - an average of 8.3%. In line A619, an average output of haploids was 6.2%. The MK01y x A619 hybrid yielded the lowest percentage of haploids - 5.9%.

We suggest that one of the factors determining the proportion of haploids of a particular maternal genotype is the coinciding or differential periods of flowering of the maternal genotype and the haploid-inducing line. In our experiment, the MK01y x A619 hybrid started silking 5 days before pollen shedding in haploid-inducing lines, whereas in line MK01y the silking coincided with mass pollen shedding in haploid inducers. The line A619 started silking 2-3 days before pollen shedding in haploid-inducing lines.

Previously, it has been established that the frequency of occurrence of haploids is not the same in different portions of the ear (Chase, Amer. J. Botany 35:117-167, 1969). The highest proportion of haploids occurred in the top portion of the ear where the egg cells are the youngest. We also measured the frequency of haploids in three ear parts: top, middle and bottom. The results are presented in Table 2.

Table 2. The frequency of seeds with haploid embryo occurring in different portions of the ear.

| Maternal genotype | No. of ears | Top of ear | | Middle of ear | | Bottom of ear | |
|---|---|---|---|---|---|---|---|
| | | Total seeds | % haploids | Total seeds | % haploids | Total seeds | % haploids |
| MK01y | 13 | 837 | 15.5 | 1060 | 6.6 | 933 | 6.5 |
| A619 | 78 | 4834 | 9.1 | 4821 | 4.9 | 4638 | 3.4 |
| MK01y x A619 | 20 | 2645 | 9.6 | 2651 | 3.3 | 2403 | 3.3 |
| Average | | | 9.9 | | 4.6 | | 3.8 |

Our results strongly support the conclusions reached by S.Chase about thirty years ago. The young egg cells in the top portion of the ear are more capable of developing without fertilization and of giving rise to a haploid embryo than the older egg cells in the bottom of the ear.

Involved in the above analysis were all the ears of line A619 resulting from crosses with haploid inducers and some ears of line MK01y and the MK01y x A619 hybrid. As averaged over three maternal genotypes, the frequency of haploids in the top portion of the ear was 9.9%. This is almost three times the frequency of haploids in the bottom of the ear and more than twice that in the middle portion. The results in Table 2 and those obtained by S.Chase allow the supposition that the proper timing of pollination of ears may increase the output of haploids.

From the results obtained in our experiment it may be concluded that new inducers of maternal haploids have been created which allow haploids to be produced in large numbers from almost any genotype of maize.

**Use of maternal haploids for improving maize inbred lines**

--Chalyk, ST (Cealic, ST)

Spontaneous recurrent mutations result in each maize material, exhibiting a certain amount of harmful mutant genes. Most new mutations are recessive and are slow to be eliminated. They represent so called genetic load (King and Stanfield, 1997). Maize lines, populations and hybrids become less vigorous due to this load.

Cleaning of a breeding material from harmful spontaneous mutations is very desirable. However, elimination of the genetic load is difficult because most of the harmful mutations are of recessive nature. Their appearance is masked by normal homologous alleles in diploid plants. Use of maize haploid plants can be an efficient means for clearing of a breeding material. A possibility of obtaining maize maternal haploids in mass quantity was shown in numerous works. Very important for a breeding program is the fact that the maize maternal haploids are quite viable. They grow normally during the vegetative period. Maternal haploids are usually male sterile. However, some of their female flowers are fertile and function normally. If pollinating an ear from a maize haploid plant with pollen taken from a diploid plant the ear usually forms kernels between several and several dozen. This property of the maize maternal haploid plants allows involving them in a breeding process

without doubling a chromosome number.

In our experiment, maternal haploid plants were used to improve two maize lines, 092 and Rf7. Haploids were produced from these lines using a haploid-inducing line. The resulting haploid plants were grown in a field plot. During flowering, well developed healthy haploid plants were pollinated with pollen from diploid plants of the initial line. The seeds produced and the plants grown from them have been designated as the C1 progeny. Well-developed haploid plants are assumed to be free from mutant genes which reduce viability. The use of such plants allows the frequency of harmful genes to be drastically reduced. Therefore, the procedure in our work, in which a line passes through the haploid sporophyte ridding itself of harmful genes, is termed a haploid filter. Thus the C1 progeny is a progeny which has passed through the haploid filter once.

The next year haploids were produced from the C1 progeny. These were grown in an experimental plot and pollinated with pollen harvested from diploid C1 plants. In this way, seeds representing the C2 progeny were produced. That is, a progeny was obtained which had passed through the haploid filter twice. The initial lines, 092 and Rf7, were reproduced per se.

With the aim of comparison initial lines were grown in the field, designated as 092(C0) and Rf7(C0), and their progenies which had passed through the haploid filter once and twice, respectively: 092(C1), 092(C2) and Rf7(C1), Rf7(C2). Plant height is the most informative indicator of plant vigor during early growth of the plant. Therefore, plant growth dynamics was followed during the experiment. Presented in this paper are the results of two measurements. The first one was done at the 6-7 leaves stage (d1) and the last measurement was done after flowering when the plants completed their growth (d6).

To produce seeds for the next cycle of selection, most ears of the plants under study were hand pollinated. Therefore ear measurements and yield determinations were not performed.

The lines studied and their progenies - C0, C1 and C2 - were grown in adjacent experimental plots. The area of the plot was 15 square meters and the density of planting was 60 plants per plot.

The diploid initial lines studied (C0) and their progenies which passed through the haploid filter (C1 and C2) have been found to differ considerably in their growth rate during early developmental stages. Taking line 092 as an example, it can be shown that the tallest plants during early growth were those of C2 (Table 1). The height of these was 49.8±0.9 cm where that of the initial line 092 was only 37.8±0.9 cm. During early growth, the plant height difference was 12 cm, suggesting that the C2 plants exhibited a higher growth rate than those of the initial line (C0).

Table 1. Plant height values for diploid plants of lines 092 and Rf7 and their C1 and C2 progenies.

| | Plant height, cm | | | |
|---|---|---|---|---|
| Genotype | d1 | Differences from initial line | d2 | Differences from initial line |
| 092 (C0) | 37.8±0.9 | | 163.7±1.0 | |
| 092 (C1) | 43.0±0.7 | 5.2*** | 164.8 | 1.1 |
| 092 (C2) | 49.8±0.9 | 12.0*** | 171.1±0.9 | 7.4*** |
| Rf7 (C0) | 53.8±1.3 | | 171.6±1.3 | |
| Rf7 (C1) | 56.5±1.3 | 2.7 | 177.7±1.1 | 6.1*** |
| Rf7 (C2) | 58.9±1.3 | 5.1** | 176.3±1.4 | 4.7*** |

*,**,*** Differences from initial line are significant at 5%, 1% and 0.1%

The differences observed between C0 and C2 plants presumably reflect the efficiency of application of the haploid filter for improving the line. It can be assumed that the frequency of harmful genes reducing viability is indeed lower in C2 than in the parental line. The C1 progeny showed an intermediate plant height value between the initial line and C2. The height of C1 plants was 43.0±0.7 cm taller than 092 plants. By the end of the growth season, the differences between the line studied and its C1 and C2 progenies decreased while remaining significant. The height of C2 plants at the end of the growth season was 171.1±0.9 cm, or 7.4 cm taller than the parental line 092.

The same tendency was observed in line Rf7 (Table 1). At the beginning of the growth season, the tallest plants were those of C2. Their height was 58.9±1.3 cm. This was 5.1 cm taller than the plants of the initial line Rf7. The C1 progeny showed intermediate values, being 2.7 cm taller than the initial line. That is, applying the haploid filter resulted, here too, in the plants of this line showing higher rates of growth during early development. This tendency continued throughout the growth season, although by the end of the season the differences leveled off. Thus, at the end of the growth season, C2 plants measured 176.3±1.4 cm in height, being 4.7 cm taller than the plants of initial line Rf7. At the end of the growth season, C1 plants were taller than C2 plants, the difference being insignificant and attributable to random factors.

The results from lines Rf7 and 092 are evidence in favor of an assumption that using selection at the haploid sporophyte level appears to be a powerful tool for improving breeding material.

A useful indicator to the value of a genotype may be the haploids obtained from this genotype. Therefore, besides comparisons of diploid plants of initial lines and their C1 and C2 progenies, haploid plants obtained from initial lines and their C1 and C2 progenies were also used for comparisons.

Plant height measurements of haploid plants are presented in Table 2. It has been found that the general tendency observed in diploid plants persists in haploid plants. At the beginning of the growth season, haploids from C1 and C2 progenies were taller than the plants of initial lines 092 and Rf7.

Table 2. Plant height values for haploid plants of lines 092 and Rf7 and their C1 and C2 progenies

| | Plant height, cm | | | |
|---|---|---|---|---|
| Genotype of haploid plants | d1 | Differences from haploids of initial line | d6 | Differences from haploids of initial line |
| n092 (C0) | 25.1±1.2 | | 105.4±2.6 | |
| n092 (C1) | 30.0±0.8 | 4.9** | 108.5±1.6 | 3.1 |
| n092 (C2) | 28.4±1.0 | 3.3* | 105.6±3.3 | 0.2 |
| nRf7 (C0) | 35.9±1.3 | | 108.3±1.4 | |
| nRf7 (C1) | 39.7±1.3 | 3.8* | 110.2±2.0 | 1.9 |
| nRf7 (C2) | 39.3±2.3 | 3.4 | 109.1±3.4 | 0.8 |

*,** Differences from haploid of initial line are significant at 5% and 1%

It is worth noting that in this case, unlike that of diploid plants, the C1 progeny did not show intermediate plant height values. Haploids of progenies C1 and C2 of line Rf7 were practically of the same height at the beginning of the growth season. In line 092, haploids from the C1 progeny were even taller than those from C2. This is attributable to the fact that in our experiment haploids obtained from C1 passed through a haploid filter twice. We suppose this was quite sufficient to rid the lines under study of harmful genes reducing plant viability. Therefore, haploid plants from progenies C1 and C2 were almost uniform in height. In diploid plants, the C1

progeny resulted from pollination of haploid plants with pollen harvested from initial lines. It is conceivable that with pollen from parental lines, part of a genetic load found its way into the C1 progeny. Therefore, in diploid plants the C1 progeny exhibited plant height values intermediate between the parental line and the C2 progeny which had passed through the haploid sporophyte twice. By the end of the growth season, the differences between the parental lines and their C1 and C2 progenies became less pronounced. However, in some cases they continued to be significant.

It may be concluded that in ridding inbred lines of harmful recessive genes the use of maternal haploid plants appears to be a tool that may be of interest to the breeder.

**Using maternal haploid plants in recurrent selection in maize**
--Chalyk, ST (Cealic, ST), Rotarenco, VA

We suggest using maternal haploid plants for increasing the efficiency of recurrent selection in maize. Maternal haploids exhibit a fair degree of viability. Partial fertility of their ears enables the selected haploids to be involved in the breeding process. Selection of haploid plants carrying useful genes is much easier as compared with diploid plants. Expressed in haploids are all the alleles: both dominant and recessive. Dominance and overdominance are completely lacking. This facilitates selection for genes with additive and epistatic effects.

Segregation of maternal haploids differs significantly from that of diploids. In haploids, it corresponds to the segregation of gametes. The frequency of useful gene combinations is much higher in haploids than in diploids. Natural selection is an important factor in improving diverse material in breeding programs involving haploids. Haploid plants carrying harmful genes either die at various stages of embryo and seedling formation or are rather stunted and sterile.

In our work maternal haploid plants were used to improve two synthetic populations of maize: SA and SP. These are populations of dent maize carrying 50% each of germplasm of North-American inbred lines. Maternal haploids were produced in a space-protected nursery. The synthetic population and the line inducing maternal haploids were sown in alternate rows, two rows of the former per row of the latter. Before flowering, tassels were removed from the plants of the synthetic population. Pollination with pollen from the inducer line occurred spontaneously, by wind. The resulting seeds were selected for the expression of gene *R-nj*. The putative haploids were those kernels in which the *R-nj* gene was expressed on the endosperm but was lacking in the embryo.

The resulting seeds with haploid embryos were sown in the field using an ordinary seeder. Each year, no less than 2000 haploid plants obtained from the improved synthetic population were grown. The haploid plants produced from the initial synthetic population were pollinated with a mixture of pollen harvested from diploid plants of the same synthetic population. Both the largest and medium-sized haploid plants were pollinated. No less than 200 to 300 haploid plants were involved in pollination. Selection was for ear size in haploids. 20 to 30 of the largest ears showing no sign of any disease were selected. The seeds harvested from selected ears were the product of the first cycle of selection (C1). As was shown before, they contain a normal diploid embryo.

The next year the bulk of the C1 seeds was sown in a space-protected nursery to produce haploids. The remaining C1 seeds were stored in a refrigerator. The next season the haploids produced from C1 and C1 seeds stored in a refrigerator were sown in the experimental field. At flowering, C1 haploids were pollinated with a mixture of pollen harvested from diploid C1 plants. Upon selection of ears, the resulting seeds were the product of the second cycle of selection (C2). Thus, a two-step scheme of recurrent selection was employed: the first step involved producing haploids, the second growing the haploids, pollinating them with a mixture of pollen from diploid plants of the same cycle of selection, and selecting haploids exhibiting traits valuable for breeding. The above procedure for improving synthetic populations has been termed haploid sib recurrent selection. A total of two cycles of the haploid sib recurrent selection was carried out for each synthetic population.

In 1998, diploids of the initial synthetic populations and of the first and second cycles of selection, C0, C1 and C2, were planted in the field. The plot area was 30 square meters. The planting density was 60,000 plants per hectare. 180 plants were planted for each cycle of selection of each synthetic population. The primary objective in 1998 was to propagate the seeds of both synthetics and their C1 and C2 cycles. Concurrently with seed multiplication, various plant and ear traits were being measured. For seed multiplication, half the plants in each treatment were used. The other half were open pollinated. Productivity and seeds per plant were only measured in plants whose ears had been open pollinated. The other traits were measured on all the plants in each treatment. The results allow some rather interesting conclusions to be drawn.

Application of recurrent selection resulted in a significant increase in productivity in both synthetic populations. The initial

Table 1. Ear trait values for the initial synthetic populations SP and SA and two cycles of haploid sib recurrent selection.

| Ear trait | Population | Mean | Gain per cycle, % | Coefficient of variation, % |
|---|---|---|---|---|
| Yield, g/plant | SP C0 | 53.9±3.5 | | 58.6 |
| | SP C1 | 67.0±4.5* | | 60.3 |
| | SP C2 | 65.7±3.6* | 11.0 | 49.2 |
| | SA C0 | 56.0±3.1 | | 46.8 |
| | SA C1 | 60.4±3.6 | | 49.8 |
| | SA C2 | 74.6±4.4*** | 16.7 | 49.8 |
| Ear length, cm | SP C0 | 12.8±0.3 | | 23.8 |
| | SP C1 | 15.2±0.3*** | | 18.6 |
| | SP C2 | 15.2±0.3*** | 9.2 | 20.5 |
| | SA C0 | 13.5±0.3 | | 19.9 |
| | SA C1 | 14.0±0.4 | | 22.1 |
| | SA C2 | 14.8±0.4* | 4.5 | 20.9 |
| Ear diameter, cm | SP C0 | 3.77±0.05 | | 12.2 |
| | SP C1 | 4.05±0.05*** | | 10.08 |
| | SP C2 | 4.02±0.03*** | 5.7 | 8.54 |
| | SA C0 | 3.72±0.05 | | 13.65 |
| | SA C1 | 3.77±0.05 | | 12.31 |
| | SA C2 | 3.98±0.04*** | 3.5 | 8.69 |
| Seeds per plant, No. | SP C0 | 232.4±12.8 | | 49.5 |
| | SP C1 | 263.7±14.8 | | 50.3 |
| | SP C2 | 275.8±13.7* | 9.4 | 44.4 |
| | SA C0 | 262.9±15.3 | | 48.6 |
| | SA C1 | 275.9±14.5 | | 44.0 |
| | SA C2 | 329.1±17.5** | 12.6 | 44.6 |
| Weight of 1000 kernels, g | SP C0 | 228.2±5.1 | | 19.9 |
| | SP C1 | 256.1±5.7*** | | 20.0 |
| | SP C2 | 241.4±5.5 | 5.8 | 20.4 |
| | SA C0 | 220.7±7.6 | | 28.8 |
| | SA C1 | 216.1±4.6 | | 17.9 |
| | SA C2 | 223.4±4.0 | 1.2 | 15.2 |

*,**,*** Differences from initial synthetic population are significant at 5%, 1% and 0.1%

synthetic population SP exhibited a yield of 53.9 g of seeds per plant, with the mean yield of the second cycle of selection (SP C2) being 65.7 g/plant (Table 1). The gain per cycle of selection amounted to 11.0%. This is a fairly large gain per cycle of selection. However, the gain was even larger in the SA synthetic population. In the initial synthetic population SA, the yield was 56.0 g/plant whereas in plants from the second cycle of selection (SA C2) it was 74.6 g/plant. The gain per cycle of selection was as high as 16.7%.

Selection of haploid plants for ear size resulted in larger ears in diploid plants of synthetic populations. Thus, for example, the ear length and diameter in synthetics of the second cycle of selection were significantly larger than those in the initial synthetics. The number of seeds per plant was significantly increased (Table 1).

Interestingly, selection of haploid plants for larger ear size resulted in larger plant size of diploids (Table 2). For instance, the mean plant height was 144.8 cm in the original synthetic population SP, and 171.2 cm in plants from the second cycle of selection (SP C2). The gain per cycle of selection was 9.3%. In the SA synthetic population, the gain per cycle of selection for plant height was 12.1%. Besides plant height, some other traits showed an increase, such as ear height, leaf length and, to a somewhat lesser degree, the number of leaves per plant.

Table 2. Plant trait values for the initial synthetic populations SP and SA and two cycles of haploid sib recurrent selection.

| Plant traits | Population | Mean | Gain per cycle, % | Coefficient of variation, % |
|---|---|---|---|---|
| Plant height, cm | SP C0 | 144.8±2.0 | | 17.2 |
| | SP C1 | 171.2±1.5*** | | 11.0 |
| | SP C2 | 171.8±1.6*** | 9.3 | 11.6 |
| | SA C0 | 148.7±1.6 | | 13.2 |
| | SA C1 | 164.2±1.4*** | | 10.3 |
| | SA C2 | 184.7±2.0*** | 12.1 | 22.5 |
| Ear height, cm | SP C0 | 42.9±0.9 | | 25.8 |
| | SP C0 | 56.0±0.9*** | | 18.7 |
| | SP C0 | 51.5±1.0*** | 10.0 | 22.8 |
| | SA C0 | 41.9±0.7 | | 20.2 |
| | SA C1 | 44.2±0.6* | | 17.7 |
| | SA C2 | 55.7±1.0*** | 16.5 | 21.8 |
| Leaf length, cm | SP C0 | 62.2±0.7 | | 14.6 |
| | SP C1 | 73.3±0.7*** | | 12.3 |
| | SP C2 | 72.9±0.5*** | 8.5 | 8.8 |
| | SA C0 | 65.0±0.6 | | 11.2 |
| | SA C1 | 66.2±0.6 | | 10.4 |
| | SA C2 | 69.4±0.6*** | 3.4 | 10.5 |
| Leaf number | SP C0 | 15.9±0.1 | | 8.7 |
| | SP C1 | 17.3±0.1*** | | 5.5 |
| | SP C2 | 16.3±0.1** | 1.2 | 6.6 |
| | SA C0 | 15.9±0.1 | | 6.0 |
| | SA C1 | 15.3±0.1*** | | 5.6 |
| | SA C2 | 16.9±0.1*** | 3.0 | 5.2 |

*,**,*** Differences from initial synthetic population are significant at 5%, 1% and 0.1%

We suggest that the high efficiency of haploid sib recurrent selection was due to two factors: (1) natural selection of haploid plants; this eliminated harmful genes from the synthetic populations, and (2) artificial selection of haploids; haploid plants made it possible to select useful genes with additive and epistatic effects. Genes with additive and epistatic effects form a basis for the improvement of populations (Hallauer, Quantitative genetics in maize breeding, 1986). A combination of the above factors resulted in a large gain per cycle of selection.

An important factor in improving synthetic populations is maintaining population genetic variability during selection. The coefficient of variation can provide a general idea about the way the population genetic variability changes. In the synthetic population SA, the coefficient of variation for yield showed little change. If anything, it exhibited some tendency to increase. The genetic variation presumably did not decrease during the first two cycles of selection, thus making it possible to expect a large gain per selection cycle for subsequent cycles as well. In the synthetic population SP, the phenotypic variation for yield decreased during the second cycle of selection, C2.

We expect the gain per cycle of selection to be considerably decreased after 4 to 5 cycles of selection because of reduced genetic variability. Therefore, new germplasm is planned to be introduced into synthetic populations starting with selection cycle 4. Work is underway to select donors to be used for improving synthetic populations SA and SP. Our experience shows that a useful means in fulfilling this task can again be provided by haploid plants which are a good indicator of the presence of useful genes in the material studied.

The above suggests that maternal haploid plants can be a useful tool for the corn breeder whose work is aimed at improving synthetic populations of maize.

**Studies on postradiation regeneration at initial ontogenetic stages in radiation mutagenesis of maize**

--Ikhim, YG, Scorpan, VG, Lysikov, VN

The most complete pattern of the action of ionizing irradiation during the whole period of plant ontogeny can be obtained in field experiments, however, they do not allow the investigation of the effect of γ-radiation and postradiation treatments at the initial, most important stages of plant development.

Postradiation treatment with stimulating factors is aimed at activating the repair system, which must increase the survival ability through somatic protection of initial cells maintaining the critical weight. In the course of postradiation regeneration, biologically active substances contribute to the recovery of tissues, including meristems that might serve as a test-system for estimating the degree of repair process activation. Compensation of phytohormonal insufficiency induced by radiation should also be defined in the action of exogenous biologically active substances.

Laboratory experiments were carried out to study the possibility of eliminating the damaging action of γ-radiation during radiation mutagenesis in two types of maize: 1. a radiation sensitive line – 19-3-3 ( S. T. Chalyk's breeding ); 2. radiation resistant hybrid Moldavskiy 401 ( bred at the Research Institute "Porumbeni" ). Air dried seeds were exposed to a sublethal γ-radiation dose followed by a treatment with biologically active substances "Crossing" and "Phytostim". Also, seeds were exposed to electrical field (EF) currents at industrial frequency and super-high frequency (SHF) for postradiation treatments.

The table summarizes the measurement data on the length of maize roots and coleoptiles at 8 days after the γ-radiation and postradiation treatment. Inhibition and termination of root and coleoptile growth were observed to occur in the treatments under study, in the treated control to a larger extent, accompanied by practically complete absence of meristem cell division. The data prove that the joint action on seeds leads to a partial elimination of the damaging action of γ-radiation. The difference is most reliable in the treatments with "Crossing" and SHF, even at the sig-

Table 1. The influence of postradiation treatments on the growth of maize seedlings.

| TREATMENT | ROOTLETS | | COLEOPTILES | |
|---|---|---|---|---|
| | $x \pm S_x$, sm | % to the control | $x \pm S_x$, sm | % to the control |
| Control | 8.1±0.53 | 100 | 2.9±0.27 | 100 |
| Radiated control | 4.5±0.66** | 55.6(100) | 1.8±0.19** | 62.1(100) |
| Radiation + Crossing | 7.0±0.61** | 86.4(+55.6) | 2.2±0.13 | 75.9(+22.2) |
| Radiation + Phytostim | 6.7±0.64* | 82.7(+48.8) | 1.9±0.15 | 65.5(+5.5) |
| | | | | |
| Control | 7.4±1.36 | 100 | 3.7±0.51 | 100 |
| Radiated control | 3.9±0.51* | 52.7(100) | 3.8±0.57 | 102.7(100) |
| Radiation + EF | 6.0±0.73* | 81.1(+53.8) | 3.9±0.41 | 105.4(+2.6) |
| Radiation + SHF | 7.9±0.72** | 106.7(+102.5) | 4.8±0.54 | 129.7(+26.3) |

The difference with control is significant, * - $p<0.05$, ** - $p<0.01$.

nificance level of 1%. In these treatments, resumption of root growth due to regeneration of the initial meristem was observed at 7–8 days. Part of the plants survived due to regeneration of secondary meristem. Similar phenomena, but less contrasting, were observed in the experiment involving hybrid seeds where the degree of seed hybridity was effective. The postradiation treatment with the chemical stimulator "Crossing" appeared to be the most effective.

The process observed in the postradiation regeneration is supposed to be as follows: Regeneration from the secondary meristems resulting from the redifferentiation of particular cells, as well as the currently carried out cytological studies, indicated an increase in repopulation in the group of initial cells, i.e., the exogenous biologically active factors contributed to the regeneration of damaged cells, accounting for the reduction of their elimination.

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### Difficulties connected with utilization of the haploid method in male-sterile counterpart production of corn lines

--Zabirova, ER; Shatskaya, OA; Shcherbak, VS

For several years we have carried out experiments on practical utilization of parental (androgenetic) haploidy in corn breeding programs. Up to now male-sterile counterparts production through androgenesis has not been widely employed, because of several difficulties connected with the conversion procedure.

The production of sterile counterparts by the haploid method is possible only for inbreds which are maintainers of sterility (cms). At present it is known that 91% of inbreds maintain cmsS, and only 68% maintain cmsC (Frankovskaya et al., 1995). We have no possibility to avoid this difficulty, and the inbreds that restore fertility are transferred into sterile cytoplasm by use of the extensive backcrossing method.

The second difficulty is connected with genetic markers that are used for paternal haploid induction. Unfortunately they are not good enough for practical use in corn breeding. They were created on the basis of different cms types and include the *ig* gene, which increases the paternal haploidy frequency up to 2% (Kermicle, 1969) or even 9% (Kindiger, 1992). Difficulties in practical use of these markers depend on their low productivity, because of small ears with a large quantity of defective kernels. Besides, low expression of the gene *R-nj* makes it difficult to select haploid kernels. To overcome this inconvenience special crosses are carried out between samples with different doses of the *ig* gene and different coloration expression (MNL 71:45). It is essential to have a marker with such coloration expression which gives a chance to identify haploid kernels without difficulties. Their haploid frequency has to be not less than 0.1%. We believe this aim will be achieved.

The third difficulty was quite unexpected. An unusual phenomenon was found in the sterile counterpart of the Kr714S inbred, which was obtained through paternal haploidy (cytoplasm S was taken from marker cmsS *ig/+ R-nj*, nucleus from original inbred Kr714). Crossing of Kr714S x P502RS was done to determine the restoring of S sterility in Kr714S. Several ears with colored and colorless kernels were found among hybrid F1 progeny. This means that although the phenotype of Kr714S was identical with Kr714 (besides sterility), some part of the female (marker) genetic material was involved in its genotype. Similar facts were reported by other authors (Chalyk, 1970; Tyrnov, 1984). This phenomenon has wrecked our notion that the paternal haploid appears as a result of male parent sperm getting into female cytoplasm and the embryo developing without female genetic material. The case of Kr714S was not the only one. The same thing was observed in inbred Kr82S. That's why we have to do some additional crosses to be completely sure of the identity of the line and its sterile counterpart.

While overcoming all difficulties during the last 5 years we obtained 71 paternal haploid plants, which formed normal kernels after pollination with the corresponding line—maintainer of sterility paternal haploidy frequency was varied from 0.03% to 0.14%. As a result of this research we produced sterile counterparts (cmsS or cmsC) for 16 corn lines.

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### *Zea* seedling reaction to inoculation with *Ustilago maydis* (DC) Corda

Astiz Gassó, MM, Molina, MC

*Ustilago maydis* (**Um**) is a Basidiomycete that promotes the development of galls in *Zea*, the relation with the host being necessary to fulfill its life cycle. Damage produced in maize plants by the presence of corn stunt are: chlorosis, seedling death, tassel seed and tumors in leaves, stems, ears and tassel. At first, it was thought that **Um** attacked only *Z. mays* and *Z. mexicana*, but later it was verified that **Um** also attacks *Z. perennis, Z. diploperennis, Z. parviglumis, Z. luxurians* and their hybrids with the grown species.

Until 1964, corn stunt did not have any incidence at the IFSC. But in that year, a *Z. perennis* clone from Jalisco (Mexico) was introduced and later on *Z. mexicana, Z. parviglumis, Z. luxurians* and *Z. diploperennis* were also grown and hybridized to *Z. mays*. As the hybrids are grown at the field as well as in the greenhouse, vegetative plants are available during all the year. The pathogen multiplies on these plants with a corresponding increase in the number of spores disseminated by air and in the soil. This fact is the main cause by which 1 to 10% of all *Zea* species and hybrids are attacked, depending on environmental conditions favouring pathogen development.

In this paper the results of analyzing the response of *Z. mays, Z. perennis* and *Z. diploperennis* seedlings when they are inoculated

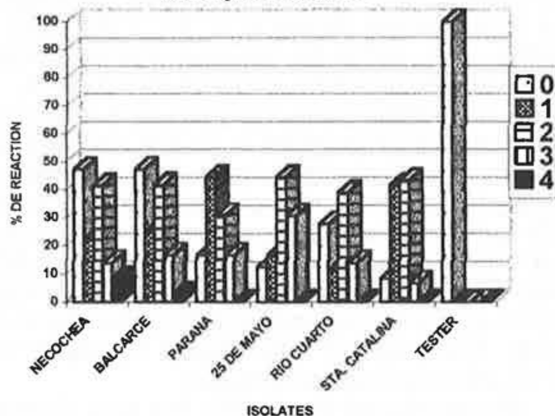


Figure 1: Reaction of Colorado Klein to 6 strains *U. maydis isolates.*

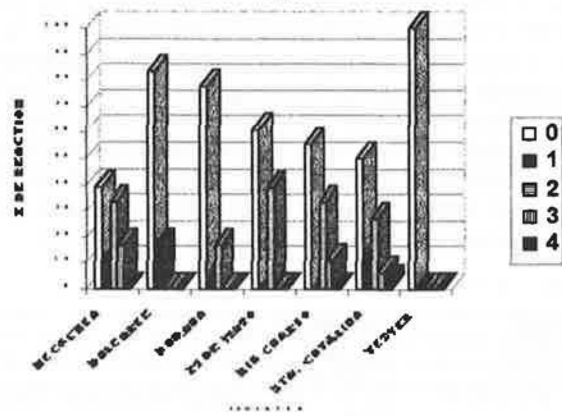


Figure 2: Reaction of Zea perennis to 6 strains U. maydis isolates.

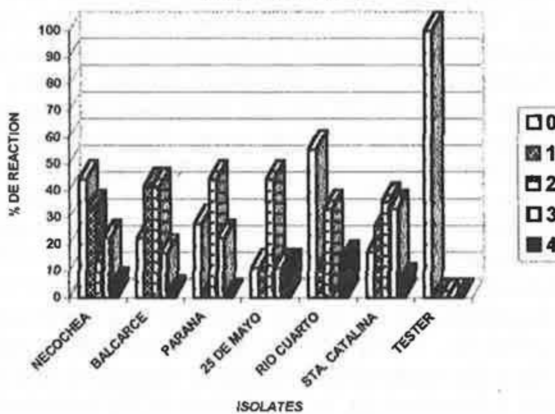


Figure 3: Reaction of B73 line to 6 strains U. maydis isolates.

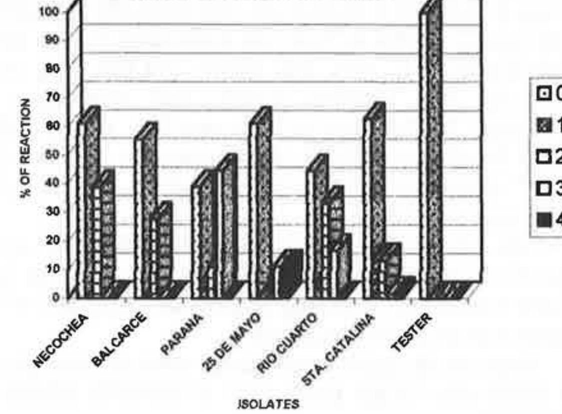


Figure 4: Reaction of Zea diploperennis to 6 strains U. maydis isolates.

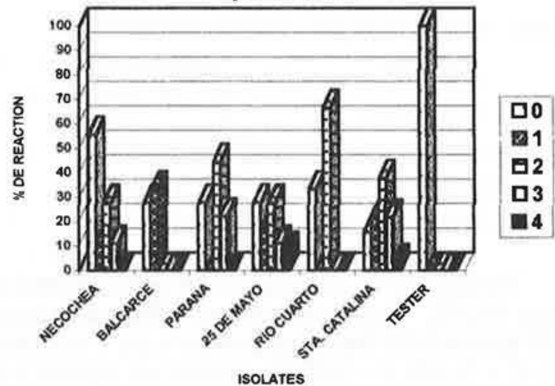


Figure 5: Reaction of E64A688 line to 6 strains U. maydis isolates

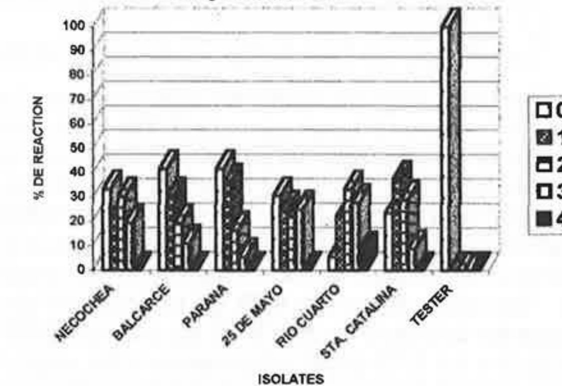


Figure 6: Reaction of SC66 line to 6 strains U. maydis isolates.

with 6 populations of **Um** are presented. This was done with the purpose of determining resistance of the species and/or inbreds to **Um.**

The host materials used were the population cv "Colorado Klein", the inbreds SC66, B73, E624A688 of *Z. mays* as well as clones of *Z. perennis* and *Z. diploperennis*. 1296 plants were inoculated during two years running (1997-98) with different strains of **Um** isolated from the province of Buenos Aires (Sta. Catalina,

Balcarce, Necochea and 25 de Mayo), the province of Entre Ríos (Paraná) and the province of Córdoba (Río Cuarto). These strains were cultivated in a liquid medium of 2% C.P.G under shaking during 8 days running at 25 C ± 2. The pathogen was inoculated by puncturing the base of the seedlings with an hypodermic syringe and the sporidial suspension was then forced up into the leaf whorl. In many previous works, this method was really successful in producing disease galls in seedlings. The trial involved three replications and a tester (non-treated plants). The plants were evaluated using a reaction scale to determine the mean percentage of infection with **Um** (Table 1). First symptoms in seedlings were observed 3 to 4 days after inoculations and gall development occurred 7 to 8 days after the treatment.

Table 1. Reaction scale in hosts.

| Behaviour | Host Reaction |
|---|---|
| 0= Immune | No reaction. |
| 1= Resistant | Partial chlorosis. |
| 2= Medium resistant | Accent chlorosis and/or presence of stripe or anthocyanin stain. |
| 3= Medium susceptibly | Necrosis and disminution of growth in plant. |
| 4= Susceptibly | Formation of tumors (galls) |

The behaviour of the host when inoculated with 6 populations of **Um** is analyzed in Figures 1-6. The hosts that reacted forming galls (grade 4) were cv Colorado Klein: Necochea (8.34%) and Balcarce (2.78%); B73: Río Cuarto (14.15%), 25 de Mayo (11.11%), Sta. Catalina (5.84%) and Balcarce (1.04%); E642A688: 25 de Mayo (8.33%) and Sta. Catalina (3.34%); SC66: Rio Cuarto (4.55%); *Z. perennis*: Sta. Catalina (1.67%) *and Z. diploperennis*: 25 de Mayo (13.89%), Paraná (2.78) and Sta. Catalina (1.67%).

From this analysis, it can be concluded that the wild and grown species of the genus *Zea* reacted in different ways (tolerant and/or resistant to moderately susceptible) depending on the geographic origin of **Um** populations.

These results might be considered when selecting germplasm to obtain new forage plants from interspecific hybrids of the genus *Zea*.

**B-chromosomes in female progenitors do not affect the male B-transmission rate in maize**

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In previous works we reported the isolation of lines of high and low B-chromosomes transmission rate (B-TR). This fact demonstrates the presence of genes controlling B-TR by both male and female sides (Rosato et al., Am. J. Bot. 83(9): 1107-1112, 1996). Recently, we made f.0B x m.2Bs crosses within and between high and low B-TR lines. We found that the resulting male B-TR depends on high or low B-TR line which the 0B female parent belongs to. This means that a high B-TR will be obtained if the female progenitor belongs to the high B-TR line (or a low B-TR if it belongs to a low B-TR group) independently of the origin of pollen (high or low B-TR lines). We concluded that B-TR is genetically controlled by the female parent without Bs, and these genes are located in the regular A chromosome set (Chiavarino et al., Am. J. Bot. 85(11): 1581-1585, 1998).

These results drove us to ask the following questions: Could B-chromosomes in the female progenitor affect the action of genes controlling the male B-TR? If so, do Bs enhance or repress the action of these genes? With the aim of evaluating these questions we crossed f.2Bs x m.2Bs and f.0B x m.2Bs (as control cross) only within high and low B-TR lines. The starting native population for selection was VAV 6313, race Pisingallo, with a high frequency of B-chromosomes (Rosato et al., Am. J. Bot. 85(2): 168-174, 1998). Lines used for this experiment suffered four generations of selection for high (0.82) and low (0.48) male B-TR.

Eight successful crosses per each type of cross (a total of 32) were obtained and classified as: (1) f.0B x m.2Bs (both high B-TR line -control-), (2) f.2Bs x m.2Bs (high B-TR line), (3) f.0B x m.2Bs (low B-TR line, -control-), and (4) f.2Bs x m.2Bs (low B-TR line). The dose of Bs was determined in primary root tips of 19 to 30 individuals of each ear obtained to calculate B-TRs. The root tips were pretreated with 0.002 mol/L 8-hydroxyquinoline for 3 h at 20 C-22 C and subsequently fixed in ethanol:acetic acid (3:1). Finally, root tips were squashed in propionic hematoxylin (2%) with ferric citrate as a mordant.

We calculated the male B-TR in both types of crosses as the quotient between the mean number of Bs transmitted to the progeny by the male progenitor and the dose of Bs of the male progenitor (2Bs in all cases). The transmission of 2Bs in the female progenitor was mostly Mendelian, resulting in 1B in each egg cell. The male B-TR of the resulting f.2Bs x m.2Bs crosses was calculated considering only Bs transmitted by the male progenitor. Therefore if the progeny presents 1B and 3Bs, then the male progenitor transmitted 0B and 2Bs respectively and the female progenitor transmitted 1B in both cases. Few plants had 0B, 2Bs or 4Bs, their frequencies being 0.02, 0.02, and 0.04 respectively (Table 1). These data are explained by a failure in the formation of bivalents in the female progenitor. The result is two B-univalents that may migrate to the same pole. Plants with 0B may be formed since 2Bs migrate to the nonfunctional pole (nonfunctional megaspore) and the egg cell is fertilized by a sperm nucleus (SN) without Bs. Plants with 2Bs may be the result of migration of 2Bs to the functional pole (functional megaspore) and fertilization with a SN without Bs, or the migration of 2Bs to the nonfunctional pole and fertilization by a SN with 2Bs. In this case, it is impossible to determine whether Bs were transmitted by the male or female parent. Finally, plants with 4Bs may be formed if 2Bs migrate to the functional pole and are fertilized with a SN with 2Bs.

Table 1. Type of crosses with their mean B-transmission rate (B-TR).

| Type of cross (female x male) | # of crosses | # of individuals with different doses of Bs | | | | | Mean male B-TR |
|---|---|---|---|---|---|---|---|
| | | 0B | 1B | 2Bs | 3Bs | 4Bs | |
| 1) 0B x 2Bs (high B-TR line) | 8 | 56 | --- | 141 | --- | --- | 0.710 |
| 2) 2Bs x 2Bs (high B-TR line) | 8 | 2 | 62 | 6 | 132 | 8 | 0.692 |
| 3) 0B x 2Bs (low B-TR line) | 8 | 107 | 1 | 97 | --- | --- | 0.470 |
| 4) 2Bs x 2Bs (low B-TR line) | 8 | 6 | 96 | 3 | 86 | 7 | 0.475 |

The high and low mean male B-TR were compared with a two-way ANOVA. There were significant differences between high B-TR and low B-TR lines (type of cross: 1 and 2 vs. 3 and 4; Figure 1) ($F = 31.463$, $P < 0.00001$), whereas no significant differences were found either between type of cross ($F = 0.051$, $P = 0.8231$), or interaction ($F = 0.094$, $P = 0.7609$) (Table 1, Figure 1). The results of this work indicate that in the low B-TR line, in fact, the B-TR is Mendelian (B-TR $\approx 0.5$), Bs being randomly transmitted to the progeny. High B-TR lines show a strong drive (B-TR > 0.5), irrespective of the presence of one B chromosome in the egg

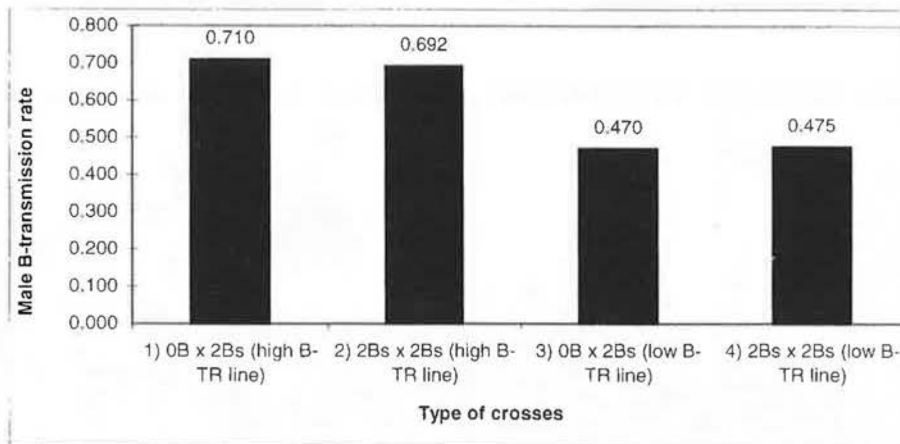


Figure 1. Mean male B-transmission rate of the different types of crosses, F=31.463, p<0.00001.

cell. In view of this, we dismiss the possibility that the presence of B-chromosomes in the egg cell affects the function of genes controlling male B-TR. Otherwise, Rosato et al. (1996) determined, by crosses f.1B x m.0B, that female B-TR is also genetically controlled on the female side. They also obtained two lines of high and low female B-TR. Considering these results, we concluded that there would be an equilibrium that keeps the frequency of individuals with Bs in the population. This hypothetical equilibrium would be reached by a balance among "anti-B" genes, causing a reduction in female B-TR, and "pro-B" genes, causing an increase in male B-TR, but controlled by the female progenitor genotype in both cases.

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**Cytogenetic studies in hybrids between *Zea mays* and *Tripsacum dactyloides***

--Molina, MC, García, MD

Hybridization was attempted between *Zea mays* and *Tripsacum dactyloides* with the purpose of studying chromosome affinity between both species and the possibility of introgressing some characters from the wild species to maize. Crossings between *Z. mays* (2n=20-40) and *T. dactyloides* (2n=72) showed post-zygotic incompatibility. Between 12 to 17 days after pollination, embryos aborted because of endosperm collapse. Reciprocal crosses induced parthenogenesis in *T. dactyloides.*

Embryo rescue and induction of organogenesis and somatic embryogenesis allowed us to obtain 5 hybrid plants with 2n=46 (ZT46) and 188 plants with 2n=56 (ZT56), from crosses between *Zea mays* 2n=20 (Zm20) x *T. dactyloides* and *Z. mays* 2n=40 (Zm40) x *T. dactyloides,* respectively (Fig. 1a and b).

The most frequent meiotic configurations in parents and hybrids were:

Zm20 = 10 II
Zm40 = 10 IV or 9 IV + 2 II
Td = 26 II + 5 IV or 24 II + 6 IV.
ZT46 = 18 II + 10 I or 16 II + 14 I.
ZT56 = 28 II or 26 II + 4 I.

In the hybrid ZT46, homoeologous chromosomes of *T. dactyloides* paired amongst them whilst those of Zm20 remained as univalents. Pairing between the chromosomes of both species was exceptionally observed, building one or two trivalents. Anaphase was not regular, with lagging chromosomes and a different chromosome number at each pole. Pollen fertility was determined with Lugol and ranged from 0 to 60%. No viable seeds have been obtained to date.

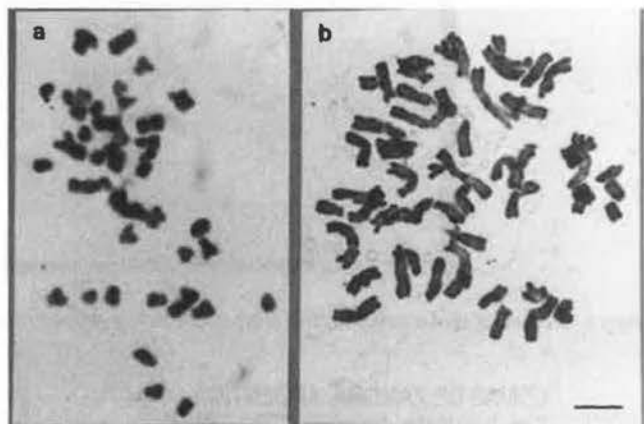


Figure 1. Karyotypes of the hybrids between Zm20, Zm40 and *T. dactyloides*. a- ZT26 (2n=46), b- ZT56 (2n=56).

In the hybrid ZT56, homoeologous chromosomes of *T. dactyloides* and Zm40 homologous ones paired amongst themselves, with the most frequent meiotic configurations 28 II (Fig. 2a) or 26 II + 4 I. The average of meiotic configurations in ZT56 was 0.61 I + 25.16 II + 1.29 IV. In some cases, tetravalents were the result of chromosome pairing between both species (Fig. 2b). Exceptionally, chromosomes paired into hexavalents (average/cell = 0,02%) or they stuck, building multivalents (Fig. 2c).

80% of anaphases were regular and in the remaining 20% lagging chromosomes were observed (Fig 2d) as well as inversion bridges. The pollen was completely sterile. Viable seed was obtained from the 50% of the plants pollinated with Zm20, Zm40, *T. dactyloides* or *Z. perennis.* The plants originating from these seeds had a chromosome number 2n=56. Apparently they originated by apomixis.

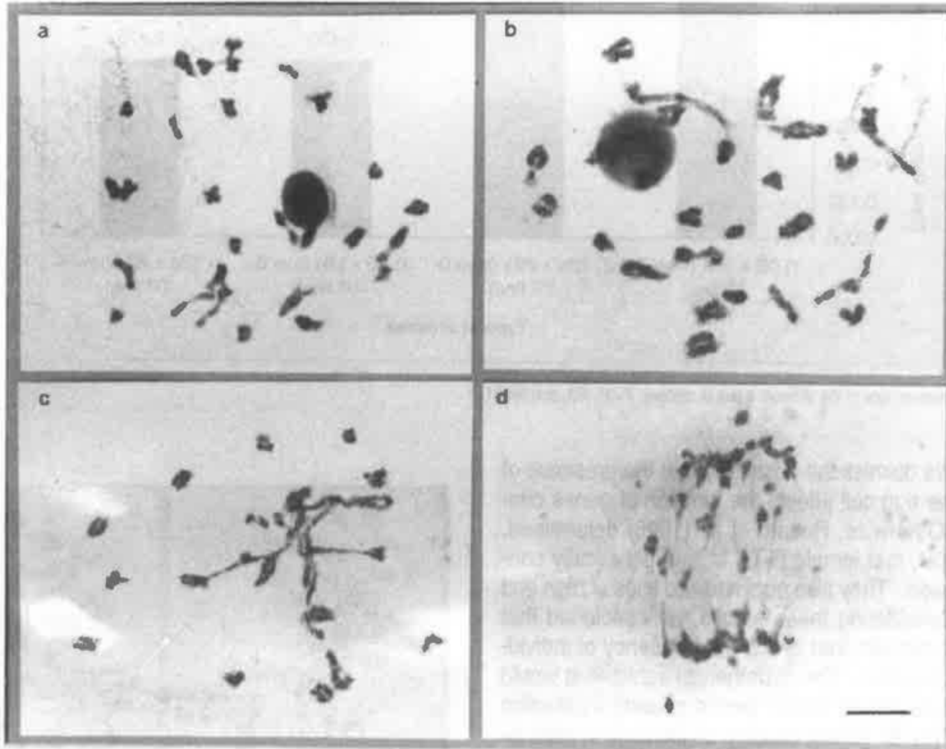


Figure 2. Meiotic configuration of the hybrids ZT56. a- 28 II; b- 26 II + 1 IV; c- sticking chromosomes; d- Anaphase with set lagging chromosomes.

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**Phytosanitary behaviour of different maize inbreds to *Ustilago maydis* (DC) Corda and *Puccinia sorghi* (Schw.)**

--Corcuera, VR*, Sandoval, MC
*Technician of the CIC.

During the growing seasons 1996/97 and 1997/98, seventy-eight maize inbreds were evaluated for their response to the attack of the fungal pathogens responsible for corn smut and maize rust. These inbreds are being developed and tested to obtain commercial hybrids with high protein or starch content and quality. In the period mentioned above, data from all the plants belonging to different pedigrees were taken to determine percentage of attack and placement of the tumors produced by *Ustilago maydis* (DC) Corda (Figure 1). Only 7.95% were attacked by this pathogen. This figure is lower than the average registered at the Institute (13.33%). Considering only the diseased plants, 53% of them showed tumors in leaves and the stems, 36% showed ear tumors and only 10% denoted tumors in ears and anthers as well as the presence of female flowers in the tassel (tassel-seed) and the development of structures similar to anthers in the apex of the ears. This phenomenon could be attributed to the production of hormones or metabolites by the pathogen which would promote the hormonal alteration of the host.

78% of all the genotypes studied showed rust attack in grade 2 and 3 (Figure 2). Chlorotic spotting phenotype was observed in 63% of these materials (Figure 3). 51% of them also showed the classic pustules and only 13% of the pedigrees revealed chlorotic spotting phenotype as unique sympton.

Figure 1. Tumors of corn smut.

Table 1 shows the results obtained for corn stunt and maize rust attack in 14 inbreds selected from the whole analyzed by their upper agronomic traits. These inbreds were also measured during both growing seasons. An increase of *Puccinia sorghi* attack was registered during the second season. This could be due to the favourable temperature and humidity conditions which helped teleospore germination. Three of these inbreds were irradiated with 150 Gy at a Gamma-cell ($Co^{60}$). The irradiated inbred 3089 showed scarce attack by *P. sorghi* if compared to the original genotype. Almost no changes were appreciated in the inbred 3074 when irradiated and the degree of attack became

Figure 2. Maize rust, classic symptom.

higher in the irradiated 3078/5.

Recessive mutations can be obtained using ionizing radiation. Dominant mutants are rarely developed by this method. The interaction between the original background of the inbreds and the radiation dose and type are responsible for the different behaviour of the plants subjected to the same treatment.

Not only does specific resistance to *P. sorghi* exist, but there is also general resistance controlled by multiple genes (Gingera et al., 1995), which appears both in adult plants and seedlings as small and isolated pustules that constitute the so-called "chlorotic spotting phenotype". Many times these symptons were confused with a degree of susceptibility to rust, but they really constitute a resistance form. Ears from plants denoting this phenotype were already selected to continue improving resistance to maize rust in the materials employed in this breeding

Table 1. Maize rust and corn smut incidence on 14 inbreds analyzed during 1996/97 and 1997/98.

| INBRED | PUCCINIA SORGHI | USTILAGO MAYDIS |
|---|---|---|
| 3003 A | 45.0 | 0.0 |
| B | 56.5 | 0.0 |
| 3002 A | 43.7 | 12.5 |
| B | 100.0 | 0.0 |
| 3012/7 A | 38.4 | 7.7 |
| B | 50.0 | 0.0 |
| 3016/7 A | 50.0 | 0.0 |
| B | 25.0 | 0.0 |
| 3016/1 A | 72.2 | 0.0 |
| B | 25.0 | 0.0 |
| 3022 A | 60.0 | 0.0 |
| B | 62.5 | 0.0 |
| 3024 A | 64.0 | 0.0 |
| B | 55.9 | 0.0 |
| 3078/1 A | 50.0 | 0.0 |
| B | 86.5 | 0.0 |
| 3078/2 A | 57.8 | 0.0 |
| B | 50.0 | 0.0 |
| 3072 A | 56.6 | 0.0 |
| B | 83.9 | 6.5 |
| 3102 A | 62.5 | 6.2 |
| B | 80.9 | 0.0 |
| 3074 A | 94.7 | 0.0 |
| B | 68.7 | 0.0 |
| C | 61.5 | 2.6 |
| 3078/5 A | 61.5 | 2.5 |
| B | 44.5 | 0.0 |
| C | 84.2 | 10.5 |
| 3089 A | 64.0 | 0.0 |
| B | 94.2 | 0.0 |
| C | 27.8 | 0.0 |

**A:** data belonging to growing season 1996/97, **B:** data belonging to growing season 1997/98, **C:** data belonging to irradiated plants measured during 1997/98.

Figure 3. Chlorotic-spotting phenotype.

## Electrophoretic studies on maize inbreds with different endosperm texture

--Corcuera, VR, Naranjo, CA

With the purpose of going deep into the study of endosperm proteins in high protein content maize inbreds developed at the Institute since 1990, electrophoretic studies were started. A modified version of SDS-Page electrophoresis technique was used to analyze 9 inbreds classified as flint, opaque2 and waxy. The gel was prepared using acrylamide 35% w/v, NN-methylene-bis-acrylamide 2% w/v. Both reagents were adjusted to ph 8.8 (1M Tris pH 8.8) for the running gel and HP 6.8 (1M Tris HP 6.8) for the stacking gel. The molecular weight marker used contained: phosphorolidase b (MW 97,000), carbonic anhydrase (MW 29,000), bovine albumin (MW 66,000) and egg albumin (MW 45,000). Figure 1 shows the electrophoretic pattern of the

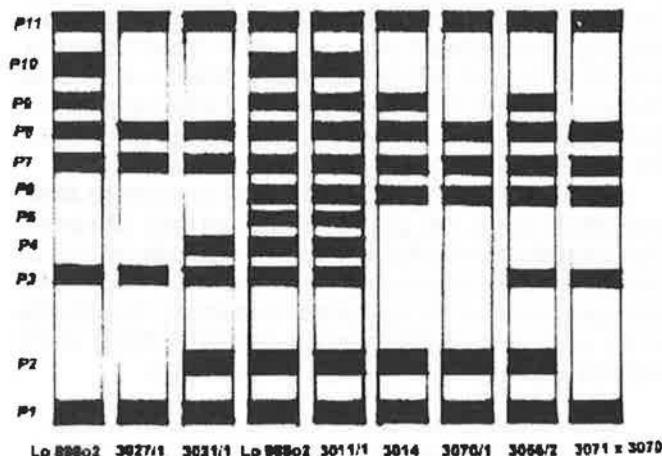

Figure 1. Electrophoretic patterns for 2 endosperm proteins observed in 9 maize inbreds.

materials analyzed. Bands 1, 7 and 8 are present in all the genotypes, whilst band 10 only appears in opaque2 maizes. Using NTSYS programme, the affinity degree amongst the genotypes studied was calculated using the protein bands observed in the electrophoretic pattern as the traits to analyze the gel. Figure 2 shows the phenogram obtained according to which the genotypes divide into 2 groups at the similarity level of about 0.20. Group 1 comprises flint and opaque2 inbreds whilst waxy maizes are placed

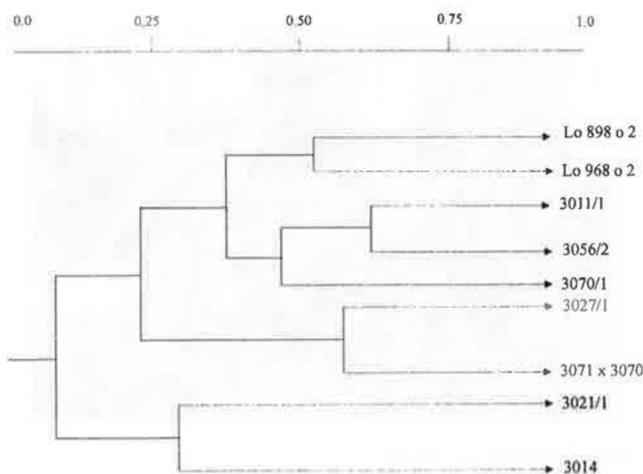


Figure 2. Phenogram belonging to 9 maize inbreds analyzed by their endosperm protein electrophoretic pattern (CCC: 0.725).

in Group 2. Group 1 also divides at the similarity level 0.30 into two subgroups: one comprises high quality protein maizes (opaque2) and the other embraces normal type maize (flint). The phenogram results agree 100% with the data registered in the genealogical records since 1990.

**Structural and histological study of somatic maize embryos**
--Galián, LR, Cárdenas, CM

Callus begun from in vitro culture of immature embryos from two alloplasmic lines of maize was examined histologically to observe the influence of exogenous 2-4-D hormone concentration, saccharose concentration, and age of the embryos cultivated, on the induction of somatic embryos.

Meristem apex was observed in the callogenic mass only when the callogenic mass originates through scutellar development induced by the combination of 1 mg.L-1 of 2-4-D and 3% saccharose with embryos of up to ten days after pollination. At the same time examination of the callus through MEB shows an organised globular structure with isodiametric cells characteristic of the first stages of the embryogenic axis from scutellum.

In contrast, callus originating under other combinations shows meristematic points that give rise to aberrant roots afterwards. The observations prove that somatic embryogenesis can only be induced in the first stages of in vitro culture under the influence of exogenous auxin in the appropriate concentration. Later change in the auxin concentration provokes the manifestation of the totipotentiality of the cells that were induced before.


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**Plant regeneration of maize-*Tripsacum* hybrids from organogenic or embryogenic long-term callus cultures**
--García, MD, Carmen Molina, MC

Embryo rescue has been used to produce maize-*Tripsacum* hybrids (Farquharson L.I., 1957. Heredity 48:295-299). Nevertheless the number of hybrids obtained could be increased through the induction of long-term embryogenic callus cultures from the rescued embryos (Furini A. and Jewell C., 1995. Maydica 40:205-210). The purpose of this work was to obtain long-term organogenic or embryogenic callus cultures to regenerate a high number of plants from maize-*Tripsacum* crosses. The genotypes used were *Zea mays* L. cv. Colorado Klein x cv. Ever Green (Zm20, 2n=20); inbred N107B (Zm40, 2n=40) from the Maize Genetics Coop. Stock Center, Urbana, Illinois, USA and *Tripsacum dactyloides* (2n=72).

Plants were grown in the greenhouse during the spring of 1995, 1996 and 1997. Crosses were made by removing the husk leaves and shortening the silks immediately before the pollen was applied (Mangelsdorf, 1974). The ears were sprayed with a solution of 40 $\mu$molL$^{-1}$ 2,4-dichlorophenoxyacetic acid (2,4-D) to improve caryopsis development. They were harvested 12 to 14 days after pollination (dap), caryopses were disinfected with 2.5% sodium hypochlorite, embryos were excised and plated on culture medium and incubated at 28 C-30 C with a 16 h photoperiod. Embryos were cultured on García et al. basic medium (García et al., 1992. Revista de la Fac. de Agron. UNLP 68:15-25) supplemented with the following combinations of 2,4-D and 6-benzylaminopurine (BA)(in $\mu$molL$^{-1}$): 0-0 (A); 4-0 (B); 1,3-1,3 (C); 2,6-1,3 (D). Callus cultures were subcultured monthly in medium B. Shoots and somatic embryos were subcultured on medium A for plant regeneration. The chromosome number was determined in the root tips, which were pre-treated with 8-hydroxyquinoline and fixed in ethanol-acetic acid (3:1) solution and stained with ferric haematoxylin or Feulgen reactive.

The results differed according to the maize used as female parent:

Hybrid ZT46 (Zm20 x *T. dactyloides*) Caryopses were turgid and embryos were 0.5 mm or less (globular and transition stage) 12 dap. Table 1 observations were made 50 days after culture initiation. Organogenic callus induction was significantly higher in those media containing both plant growth regulators (2,4-D + BA), but plant regeneration and later transplanting were not efficient. Only 5 plants, 3 to 5 cm height with 1 to 3 adventitious roots, were regenerated but they all died during the transplanting period.

Nevertheless plants with normal root development were regenerated from callus cultures growing on medium with 2,4-D as the only growth regulator. The 6.25 % of the embryos plated on this medium gave rise to callus able to regenerate plants by organogenesis and somatic embryogenesis for a 12 month period. Eighteen plants are growing in the greenhouse. All of them showed a chromosome number of 2n = 46.

Hybrid ZT56 (ZM40 x *T. dactyloides*) Caryopses were turgid and embryos were 1.5 mm length (scutellar stage) at 12 to 14 dap. Embryogenic callus arose from 100% of the embryos and they regenerated plants during 24 months after plating. Some of the somatic embryos and organogenic callus were transferred to media A since the first month and up to date they gave rise to 188 vigorous plants with many adventitious roots, 77 of which are growing in the greenhouse. A chromosome number 2n=56 was determined in the regenerated plants.

Hybrid phenotype Both hybrids showed some similar vegetative characteristics: they are tillering plants with *Tripsacum*-like leaves and short rhizomes. Plant height ranged from 0.5 to 1 m in ZT46 and 1 to 2.9 m in ZT56. Hybrid ZT56 plants placed in the greenhouse in October 1997 started flowering by December. The

Table 1. Frequency of organogenesis or somatic embryogenesis from ZT46 hybrid embryos.

| Culture medium | Responses (%) | | | | Number of embryos | Number of regenerated plants |
|---|---|---|---|---|---|---|
| | Germination | Organogenesis/ embryogenesis | Callus | Without growth | | |
| A | 0 | 0 | 50 | 50 | 8 | 0 |
| B | 0 | 18.7 a | 81.3 | 0 | 16 | 21 |
| C | 9.1 | 63.6 b | 27.3 | 0 | 22 | 0 |
| D | 7.7 | 61.5 b | 23.1 | 7.7 | 26 | 5 |

Numbers followed by the same letter do not differ statistically according to Brant and Snedecor test (Cochran W.G., Cox G.M., 1965. Diseños Experimentales pp 128-132. Ed. Trillas, S.A., México)

lateral inflorescences were distich ears, intermediate between maize and *Tripsacum*. The apical inflorescences were tassels with 3 to 5 lateral branches. Although the pollen was sterile, viable seeds were obtained from April 1998 to June and from September onwards.

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**The expression and localization of HSP 18 mRNA in *Zea mays* L. callus**

--Friedberg, JN, Walden, DB

Previously, Greyson et al. (Devel. Genet. 18:244-253, 1996) and Yang et al. (MNL 72:56. 1998.) have demonstrated through in situ hybridization that the heat shock response in maize seedlings is localized to meristematic regions of primary roots, lateral roots and shoots. These findings raise questions about the nature of this localized response. To address this question we employed the use of in vitro callus cultures (Friedberg et al., MNL 72:56. 1998) to determine if the heat shock response requires an organized meristem or if actively cycling cells can respond. Furthermore, we characterized the heat shock response in not just the callus but in roots that were induced to regenerate from callus.

To assess the heat shock response in maize callus we employed the use of the antisense in situ hybridization, using the scMHSP 18-9-2 RNA, DIG labeled probe (Table 1, Greyson et al., 1996). All callus and root tissues were heat shocked at 43 C for two hours, fixed and embedded in wax blocks. Ten and 15 micron sections were prepared and mounted on glass cover slips in preparation for in situ hybridizations. All in situ hybridizations were performed according to Greyson et al., 1996. Callus was initiated from immature embryos (inbred Oh 43) and subjected to in situ hybridizations after 30, 60, 90 or 120 day intervals. At each of these intervals, several additional calli were transplanted to root induction media. Once root induction was evident (approximately 5-10mm), the roots were also subjected to in situ hybridizations with the aforementioned probe.

Analysis of callus in situ hybridizations revealed a significant level of HSP 18 mRNA expression in callus 30, 60, 90 or 120 days old in response to heat shock (Figure 2). The experiment was replicated eight times and there appeared to be no significant difference in HSP 18 mRNA expression between the intervals tested (Figure 1). Analysis of in situ hybridizations on the callus-induced roots exhibited positive HSP 18 mRNA expression, which was localized to cells in the meristematic region. Observations of heat shocked callus sections revealed that mRNA expression was localized to the nucleus and the periphery of the cells (Figure 2, E to H) and not throughout the entire cell as is seen in meristematic tissue (Yang, 1997, MSc Thesis The University of Western Ontario, London, Ontario). Although it could not be observed directly, the mRNA localization in callus might be a result of cells with large vacuoles, possibly restricting the mRNA to the periphery and the nuclear region. Furthermore, it is possible that some of the cytoplasmic mRNA could have been lost due to the rupturing of cells during sectioning.

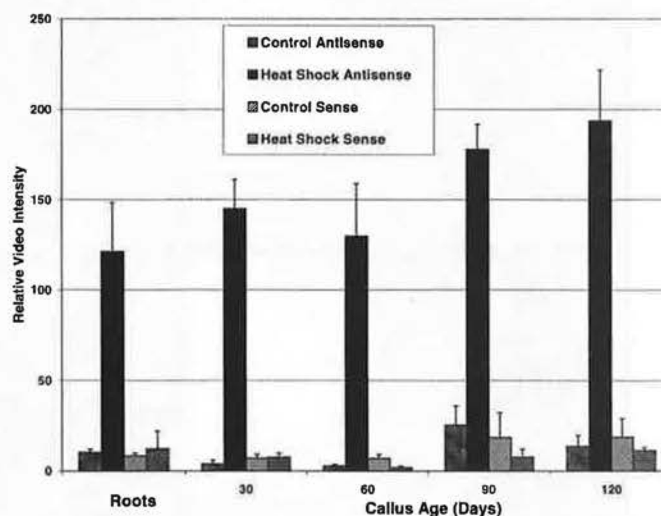


Figure 1. Densitometric readings of DIG stained in situ hybridization sections using the scMHSP 18-9-2 antisense and sense probes. A) Unadjusted data averaged from the relative video intensity of callus sections (30, 60, 90 and 120 days old) and root (induced from callus) sections. The data were drawn from control and heat shock sections hybridized with the antisense probe, and control and heat shock slides hybridized with sense probe. B) The average relative video intensity representing the net antisense heat shock response (control data subtracted from heat shock data) for each time interval and roots (induced from callus). There is not a significant difference among the time intervals of the callus response nor is the callus response significantly different from that measured in the roots. Error bars = the standard deviation; sample size (n) = 5.

The roots that regenerated from callus cultures appeared to possess the cell anatomy of embryo-derived roots and when heat shocked, a characteristic heat shock response. As the roots elongated and the cells differentiated the heat shock response diminished in the mature cells but was present in the meristematic regions. It appeared that the regeneration process did not affect the functioning of organized tissue.

It was observed that across the four time intervals the heat

shock response remained at a constant level indicating that the cells were still alive and active. This provides further evidence that cells in callus culture have the ability to remain alive beyond the life span of an embryo-derived plant and may have the ability to remain active indefinitely.

The callus appeared to expand outward from the edges and not from the center. Analysis of callus sections revealed that cells throughout the callus have the ability to respond to heat shock. This may indicate that the cells inside the callus are actually dividing and the callus increased from both the outside and the inside. Alternative possibilities are that the cells were not dividing but retain the ability to express the heat shock response.

The probe used in these hybridization experiments was specific to the ORF of HSP 18 as confirmed by Northern blot hybridization. However, in maize there are three different HSP 18 genes (cMHSP 18-1, cMHSP 18-3 and cMHSP 18-9) that code for different HSP 18 mRNAs that share considerable homology in their ORFs (Goping et al., 1991, Plant Molec. Bio. 16:699-711; Atkinson *et al.*, 1993, Dev. Genet. 14:15-26). Further, it is known now that there are multiple translation start sites yielding up to eight distinct proteins from these three genes (Frappier et al., 1998, Genet. 148:471-477). Thus, it is not known which HSP 18 mRNAs are expressed in heat shocked callus. Further studies using probes specific for the 3' and 5' untranslated regions should be employed to determine which of the HSP 18 mRNAs are expressed in heat shocked callus.

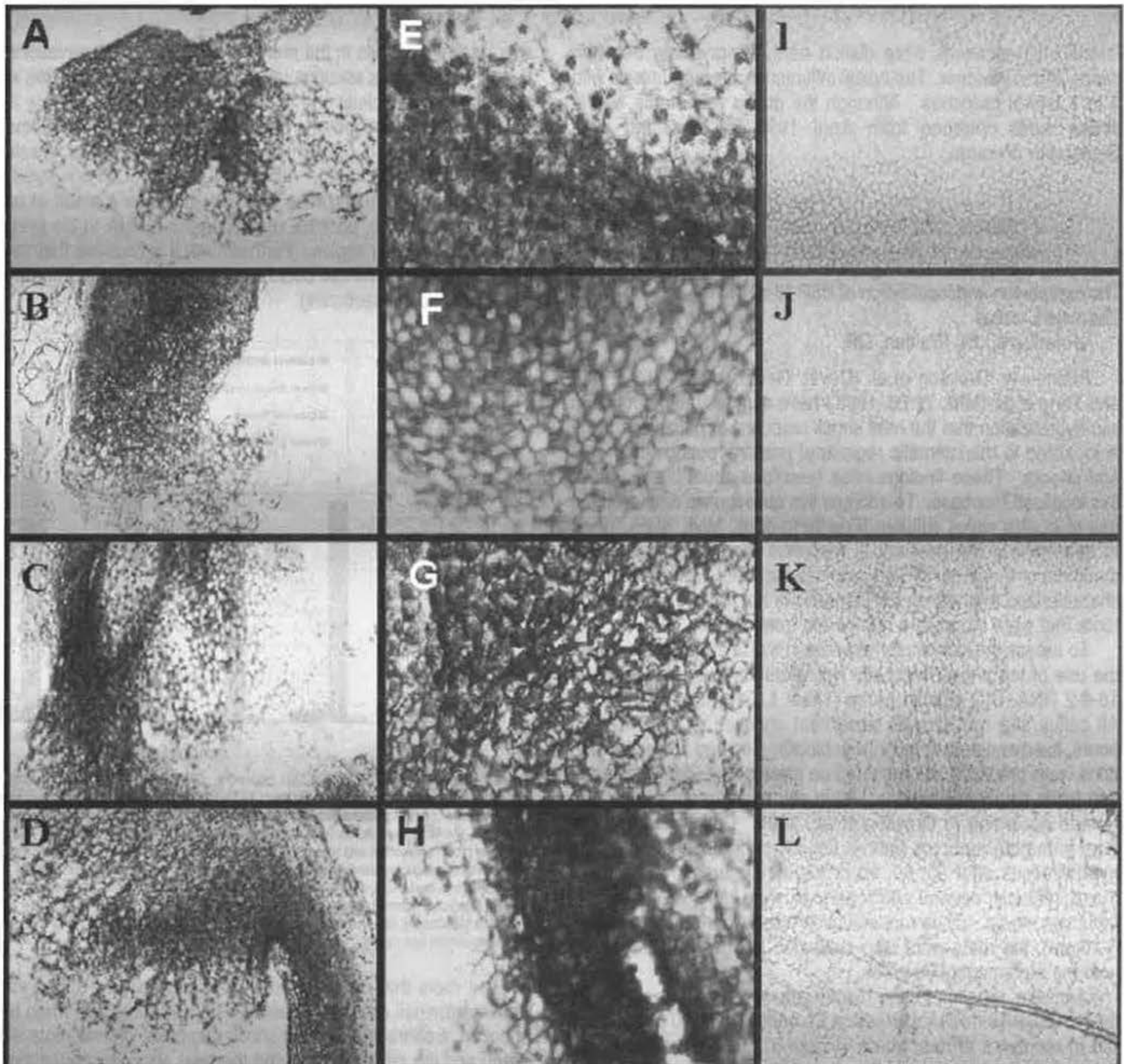


Figure 2. Reproduction of digital photographs of representative in situ hybridizations of 30, 60, 90 and 120 day old heat shock and control callus sections probed with the scMHSP 18-9-2 antisense RNA probe. A&E) 30 day old callus heat shocked at 43 C for two hours. B&F) 60 day old callus heat shocked at 43 C for two hours. C&G) 90 day old callus heat shocked at 43 C for two hours. D&H) 120 day old callus heat shocked at 43 C for two hours. I to L are control callus from 30, 60, 90 and 120 day old callus respectively. Notice the increase of HSP 18 mRNA (dark regions) in the heat shock callus (A to H) relative to the controls (I to L). Pictures A to D and I to L were taken at 100x magnification. Pictures E to H were taken at 400x magnification.

The evidence that callus can express a heat shock response indicates that the ability to respond to heat shock is not only a characteristic of meristematic tissue but a characteristic of any cells in an active nuclear cycle. Further support can be seen in the ability of secondary roots to express a strong heat shock response even though they emerge from mature cells that cannot respond to heat shock (Yang, 1997).

**Additional NORs and the number of nucleoli per coleoptile cell in maize**

--Quinn, C, Maillet, DS, Walden, DB

As part of an ongoing study of the nucleoli of maize we have examined the effect of the presence of additional NORs. In the coleoptile of diploid lines of maize, which have one NOR on chromosome 6, most coleoptile cells (approximately 80 to 90%) Maillet et al., MNL 72:56-58,1998) have one nucleolus, the rest have two nucleoli. In a cultivar which has four NORs per nucleus it should be possible for the cells to have one to four nucleoli. We have counted the number of nucleoli that are present in coleoptile cells from two lines that have four NORs in somatic cells, a tetraploid line of maize and the diploid cultivar 2NOR, in order to determine the effect of additional NORs on the number of nucleoli. The tetraploid has four copies of chromosome 6 and therefore four NORs; the cultivar 2 NOR has two NORs on each of the two copies of chromosome 6, which can be observed in an acetocarmine preparation of cells in early pachytene.

Our observations of 1064 tetraploid (uwo-52509) coleoptile cells confirm that one to four nucleoli can be present . It was expected that the linear arrangement of NORs would alter the ratio of nuclei per cell compared to the tetraploid. Examination of 800 coleoptile cells from the cultivar 2NOR revealed that only one or two nuclei were present per cell. In some cases there was one nucleolus plus one micronucleolus (Table 1).

Table 1. Percentage of nucleoli per coleptile cell of a 2NOR and a tetraploid cultivar.

| | # Nucleoli/ cell | | | | |
|---|---|---|---|---|---|
| | 1 | 1* | 2 | 3 | 4 |
| Tetraploid (n =1064) | 74.6 | 0 | 19.2 | 5.5 | 0.7 |
| 2NOR (n =800) | 90.8 | 6.7 | 2.5 | 0 | 0 |

* = nuclei with 1 nucleolus and one micronucleolus

The observation of one to four nucleoli in the tetraploid demonstrates that it is possible for four NORs on separate chromosomes to form nucleoli. However in the stock 2NOR where the four NORs are in pairs they do not form three or four nucleoli. It is possible that the additional NOR on chromosome six of the 2NOR cultivar is not functional or that one of the NORs can suppress the other, perhaps as a result of their proximity or relative size.

**Morphological changes in coleoptile nucleoli after heat shock**

--Maillet, DS, Walden, DB

Our earlier study (Maillet et al. MNL 72:56-58,1998) on nucleoli has been extended to include an assessment of changes induced by specific environmental insults. There are several reports which describe changes in nucleoli during heat shock in terms of the loss of structure or the redistribution of specific proteins. Observation of intact silver stained nucleoli within epidermal cells of coleoptiles that were heat shocked 2, 4, or 6 hours (shifted from 27 C to 42 C) revealed a characteristic series of changes in nucleolar morphology. Although there is considerable variation in the response of cells from different regions of the tissue, at each time point most of the nucleoli in an area of the tissue would be at approximately the same stage. This variability may be the result of the mechanisms that control the heat shock response. Cells must receive a signal, either internal or external, to initiate transcription and translation, some heat shock proteins must move to the location where they act, and it is unlikely that all cells are equally responsive to heat shock. After two hours of HS the nucleoli had developed protrusions on their surfaces. The number of these structures corresponded to the number of NORs present in the genome. Cells from a diploid cultivar of maize (Ohio 43) had one or two protrusions. Since the nucleoli are viewed from one angle, the protrusions may not always be visible. Figure 1 shows heat shocked (two hours) Ohio 43 nuclei with one (a) and two (a insert) nucleoli. After four hours the many nucleoli had swollen and a furrow was present often in the middle of the nucleolus (Figure 1b). Often when two protrusions are observed in a diploid heat shocked cell they are at opposite ends of the nucleolus. Six hours after the initiation of HS many of the nucleoli appear to have been divided into two to four masses (Figures 1c and d).

In order to test the hypothesis that the protrusions are the sites where the NORs are attached to nucleoli, heat shocked coleoptile cells were examined in a cultivar that has additional NORs. The cultivar 2NOR has two NORs that are visible during early pachytene (Figure 1e), thus it would be expected that one to four nucleoli could be present (see the companion article for the number of nucleoli) in coleoptile cells, and that there would be one to four protrusions present after two hours of heat shock. Coleoptile cells that were heat shocked for two hours had one to four protrusions present on or near the surface of the nucleoli as expected, indicating that the number of protrusions are related to the number of NORs. In a very few nuclei a chromatin fiber could be seen connecting the micronucleolus to one of the protrusions on the nucleolus (Figure 1f).

These observations indicate that NORs can be made more distinct by a two hour heat shock and that the changes in nucleolar morphology appear to follow a characteristic series of stages.

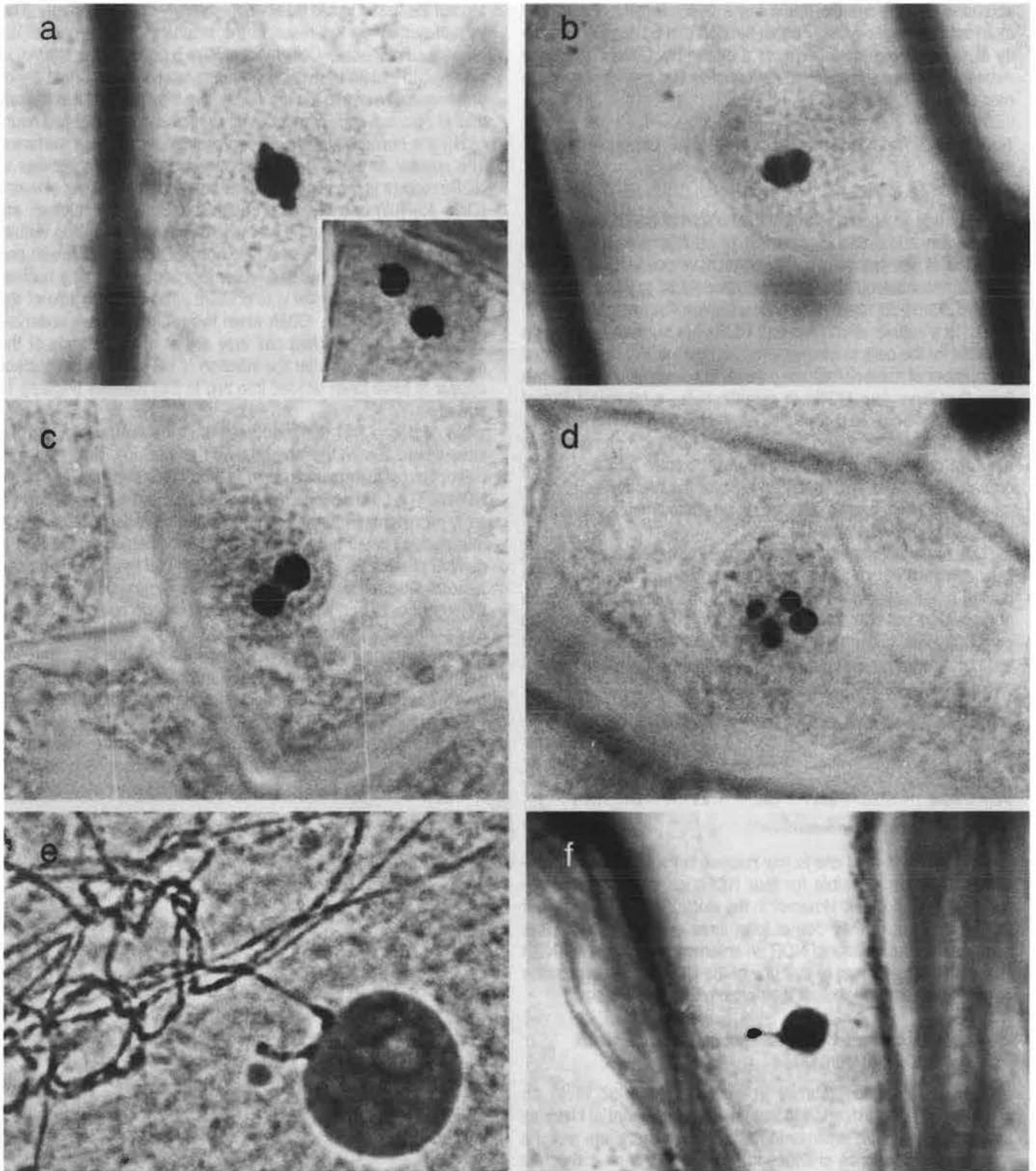


Figure 1. Stages of changes in nucleolar morphology (a to d), early pachytene in the 2 NOR cultivar (e), and a micronucleolus attached to a nucleolus in the cultivar 2 NOR (f).

MILAN, ITALY
University of Milan

**Shoot meristemless (*sml*) mutant in maize**
--Pilu, R, Mencarelli, M, Tamagnone, L, Consonni, G, Gavazzi, G

The shoot meristemless (*sml*) phenotype was selected as a putative non germinating mutant in a line obtained by selfing F1 plants heterozygous for *Ac* and *r-scm3*. This mutant (originally named *emb 7190* as previously reported in MNL 72:58 ), shows up at germination, since primary root protrusion is not associated with shoot emergence. Other shootless mutants (*ed-41v* and *dks8)* in our collection, that have been studied by Dr. C. Rivin, are not allelic to *sml*.

Longitudinal sections of immature (16 DAP) *sml* mutant embryos show a normal root primordium while a shoot meristem is not recognizable. In its place several meristematic-like cells are found intermingled with nonmeristematic ones, suggesting that the mutant is unable to recruit meristematic cells into the orderly structure of a SAM (Figure 1).

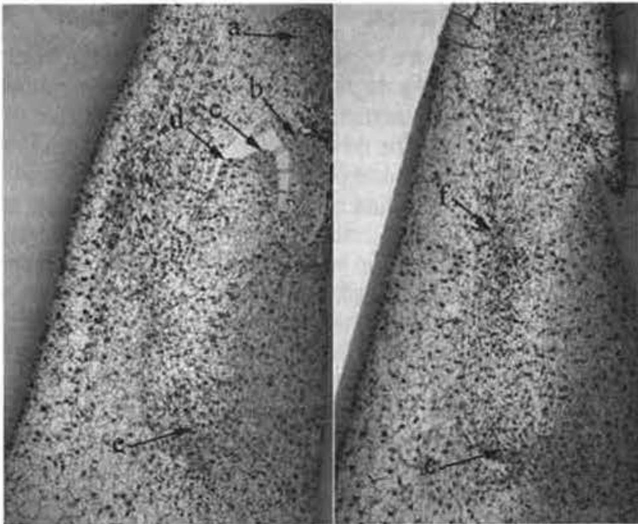


Figure 1. Longitudinal sections of wt and sml embryos at 16 DAP stained with toluidine blue. Note the presence of putative meristematic cells in the area where the shoot apical meristem (SAM) should be. It seems as if these meristematic cells are not recruited into the orderly structure of the SAM with its characteristic zonation. a) coleoptile, b) first leaf, c) shoot apical meristem, d) second leaf bulge, e) root meristem, f) meristematic-like cells.

In its original background (W22) the mutant segregates 3:1. If heterozygous plants are outcrossed to A344 or A188 inbred lines and the F1 is selfed, the F2 consists of normal (wt), distorted growth (dgr) and shoot meristemless (sml) seedlings in a 12:3:1 ratio.

Seedlings with distorted growth are characterized by abnormal growth of the coleoptile and internodes and, in some cases, by the appearance of two or more shoot apexes or of a root replacing the shoot. During development leaves do not expand and plants do not elongate. Mature plants exhibit a bushy growth habit (Figure 2).

The data reported in Table 1 are best interpreted by assuming that the sml phenotype results from the interactions of two recessive factors: *sml* and *dgr* (*distorted growth*). The distorted growth phenotype (Figure 2) is attributable to a partial

Figure 2. Distorted growth (DGR) phenotype at maturity.

recovery of the sml phenotype, possibly exerted by the *Dgr* allele on the *sml/sml* genotype. Presence of one functional dose of *Dgr* allows bypass of the meristematic block induced by *sml* by promoting some shoot morphogenesis even though without allowing total recovery. The statistical analysis ($\chi^2$ test) performed on the data shown in Table 1 confirms the hypothesis of a recessive epistatic interaction.

Table1. Results of F2 segregations obtained in selfed progeny of the mutant outcrossed to different inbred lines.

| Inbred | wt | dgr | Sml |
|---|---|---|---|
| A188 | 430 | - | 136 |
| A344 | 108 | 40 | 10 |
| B73 | 282 | 64 | 20 |

The *sml* gene has been mapped on chromosome 10L and experiments to delimit its location more accurately are in progress.

MOSCOW, RUSSIA
Institute of Agricultural Biotechnology

**A188 inbred and its somaclones do not differ in the lengths of amplified fragments of the anionic peroxidase gene *ZmAP1***
--Zabrodina, MV, Karyagina, AS, Khavkin, EE, Shilov, IA

Previously (MNL 70:42, 1996) we reported that two A188 isoperoxidases: Px9, which is predominantly expressed in roots, and the root-specific Px12, were manifest in the leaves of several A188 somaclones. The disrupted tissue-specific expression was inherited; however, (A188 x somaclone R27) F2 plants did not segregate for the newly established staining pattern. We presumed that somaclonal variation affects trans-control of peroxidase expression rather than the coding sequences of *px9* and *px12*.

Teichmann et al. (Eur. J. Biochem. 247:826-832, 1997) cloned *ZmAP1* coding for an anionic peroxidase, which was active predominantly in roots, the mesocotyl, and the coleoptile, whereas no *ZmAP1* expression was found in the primary leaf of maize seedlings. By its expression pattern, *ZmAP1* resembles *px9* and/or *px12*.

We compared the amplification products of *ZmAP1* cDNA and

genomic DNA isolated from the leaves of A188 inbred and its two somaclones, R27 (4th seed generation) and R105 (2nd seed generation). Dr. T. Teichmann (Max-Delbrück-Laboratorium, Köln, FRG) kindly provided the *ZmAP1* clone (Genbank accession Y13905). Two pairs of primers were constructed to flank two functionally important regions corresponding to 165-316 and 843-1043 nucleotides of *ZmAP1* cDNA sequence.

When genomic DNAs from three genotypes were amplified with these primer pairs, the lengths of DNA fragments were similar to those produced by cDNA amplification. It follows that somaclonal variation produced no sizable inserts into two regions of *ZmAP1*.

**Direct amplification of the conserved sequences of MADS-box genes in A188 inbred and two of its somaclones**

--Zabrodina, MV, Karyagina, AS, Khavkin, EE

Somaclones of the A188 inbred manifest heritable modifications in inflorescence architecture, including the decreased number of kernel rows per ear and the increased numbers of tassel branches and kernels per row (Dolgykh Y.I., this MNL issue). The mapping positions for these traits often collate with the loci for homeotic genes (Khavkin and Coe, MNL 72:60, 1998). It is presumable that somaclonal variation directly affects homeotic genes in control of inflorescence development.

DNA fragments produced by amplifying genomic DNAs and cDNAs were compared by size. Genomic DNA was extracted from the seedling leaves of A188 inbred and its somaclones, R27 and R105. Degenerated primers for amplifying MADS-box region of MADS-box genes were constructed using the consensus for *zag1*--*zag5*, *zmm1*, *zmm2*, and *zap1* (Genbank accessions L18924, X80206, L46397, L46399, L46398, X81199, X81200, L46400). Degenerate primers for amplifying K-box sequence were designed separately for three subgroups of MADS-box genes, *agamous*, *agamouslike6*, and *apetala1*. The clones of *zag1*, *zmm2*, *zag5* and *zap1a*, kindly provided by Drs. R. J. Schmidt and B. A. Ambrose (University of California-San Diego), served as the references.

Amplification of the MADS-box sequence in leaf and clone DNAs produced a single DNA band of predicted size. Amplification of the K-box sequences of *agamous*, *agamouslike6*, and *apetala1* genes in leaf DNA produced correspondingly four, two, and one DNA bands, some similar in size to the products of clone amplification and some considerably larger. The size of the products of clone amplification matched the sizes predicted from the published sequences.

Each DNA band observed could comprise more than one product of amplification of related sequences. It is, however, clear that A188 and somaclones did not differ in the size of any of the DNA fragments produced by amplifying leaf DNA with each particular pair of primers. Apparently, no substantial inserts into the conserved regions of MADS-box genes resulted from somaclonal variation.

**High level of variability among the plants regenerated from callus of inbred A188**

--Dolgykh, YI

About 200 somaclones have been regenerated from scutellar callus of inbred A188 after cultivation in vitro during eight months. The viability of the regenerated plants was low: only 11.3% of somaclones reached maturity. Most regenerated plants differed in several traits from the initial line. About 40% of somaclones manifested developmental abnormalities, and 13% were sterile. Six self-pollinated regenerated plants with normal development were chosen randomly, and their developmental patterns were analyzed through three seed generations. The inherited variations of quantitative traits included decrease in plant height, the node number, the number of kernel rows per ear and increase in the length of the fourth leaf from the top of the plant, the number of tassel branches, and the number of kernels in a row. The time to anthesis in all somaclones was one or two weeks shorter as compared to A188. Purple-colored kernels were observed in the R1 and R2 progenies of three regenerated plants. The level of expression of this trait was variable. Two somaclones differed from A188 in 10-20% lower capacity for embryogenic callus induction in immature embryos. Two to seven patterns were inherited simultaneously in each somaclone. The future experiments will elucidate whether these variations resulted from several independent mutations or from one pleiotropic mutation.

**The content of hormones in the embryos of inbreds competent and incompetent for morphogenesis**

--Dolgykh, YI, Zhdanova, NE, Pustovoitova, TN

Maize genotypes are known to differ in their capacity for morphogenesis in vitro. The degree of competence for morphogenesis can depend on the concentration of endogenous phytohormones in the cells of explants. The contents of free auxin (IAA), cytokinins (Z and ZR) and abscisic acid (ABA) were compared by immunoenzyme assay in (1) immature embryos of regenerable (A188) and recalcitrant (Gk26) lines isolated at the developmental stage, which was optimal for callus induction, and (2) mature and immature A188 embryos, which differed in their morphogenetic capacities. The analysis showed that the frequency of embryogenic callus formation was related to the concentration of hormones in the explant cells (Table1). Lower level of auxin and cytokinins and higher concentration of abscisic acid characterized the tissues with a competence for somatic embryogenesis. To temporarily decrease the auxin content in the isolated immature Gk26 embryos, one batch of the embryos was incubated with IAA-oxidase co-factors *p*-coumaric acid and 2,4-dichlorophenol. The second batch of Gk26 embryos was treated with exogenous ABA to elevate the concentration of the endogenous hormone. The effect of the treatment depended on the concentration of reagents. The artificial change in the balance of endogenous hormones resulted in a 3-10-times higher frequency of embryogenic callus formation by the explants (Table 2).

Table 1. The content of endogenous hormones in maize embryos (ng/g fresh weight)

| Line | Frequency of embryonic callus induction, % | IAA | ABA | Z+ZR |
|---|---|---|---|---|
| A188, immature embryos | 60-80 | 129.5±25.7 | 9092±2.3 | 15.7±1.0 |
| A188, mature embryos | 25-35 | 8.3±3.5 | 419.3±35.3 | 20.7±1.7 |
| Gk26, immature embryos | 2-4 | 3000.0 ±451.8 | 283.2±17.3 | 65.8±12.4 |

Table 2. The frequency of explants forming embryogenic callus.

| Untreated | *p*-Coumaric acid, $5 \times 10^{-4}$ M | 2,4-dichlorophenol, $10^{-5}$ M | ABA, $2 \times 10^{-5}$ M |
|---|---|---|---|
| 2.4+1.4 | 12.2±3.4 | 13.9±3.2 | 20.9±4.4 |

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Ludwig-Maximilians-University

**Correlation of *Ac/Ds* element transposition with DNA methylation and replication**

--Ros, F, Kunze, R

Genetic data suggest that *Ac* transposes in maize during replication. Based on the observation that *Ac* transposase (TPase) binds in vitro selectively to hemimethylated DNA and that the TPase binding sites of *Ac/Ds* are methylated in vivo, we have proposed a model that explains the association of transposition with replication, and the chromatid selectivity of transposition as a consequence of different *Ac/Ds* methylation states (Wang, L et al., Plant Cell 8:747-758, 1996).

To validate this model, we have begun to investigate the effect of replication on *Ac/Ds* transposition in the petunia protoplast test system. We constructed two reporter plasmids. In pNT150MiniDs-f1DIR (Fig. 1a) a 700bp long Mini-*Ds* element blocks the expression of the GUS gene. Plasmid pNT150MiniDs-f1DIR/Rep (Fig. 1b) is identical to pNT150MiniDs-f1DIR, except that the 1'-Pr./LUC gene fragment has been replaced by a viral replicon from the Tomato Yellow Leaf Curl Virus (TYLCV) (Kheyr-Pour, A et al., Nucleic Acids Res. 19:6763-6769, 1992).

To determine the transposition activity, each reporter plasmid was co-transfected together with a TPase expression plasmid into ca. $10^6$ petunia protoplasts. The TPase protein enables the *Ds* element to transpose, resulting in reversion of the GUS gene. After transfection, protoplasts are cultured for approximately 30 hr in 5 ml rich medium. Subsequently, 200 µl of the protoplasts are spread and fixed on nitrocellulose membranes, and stained for GUS activity. The number of blue protoplasts is taken as an approximation of the *Ds* excision frequency.

According to our preliminary results, in the presence of the TYLCV replicon the number of blue protoplasts is 2 to 3 times higher than without it (Table 1). Moreover, the staining intensity of the protoplasts is significantly higher after transformation with the replicon-containing plasmid. To determine whether this effect is merely the result of the altered sequence composition of the pNT150MiniDs-f1DIR/Rep plasmid, or if the replication activity of the TYLCV replicon is responsible for the apparent higher transposition frequency, we performed several tests.

(1) Petunia protoplasts were transfected with reporter plasmids isolated from $Dam^{+}$-$Dcm^{+}$ host strains. Control experiments using plasmids from $dam^{-}dcm^{-}$ strains demonstrated that Dam-Dcm methylation does not affect the transposition efficiency of the *Ds* element in Petunia protoplasts. We determined the GATC methylation state of pNT150MiniDs-f1DIR and pNT150MiniDs-f1DIR/Rep isolated from a $Dam^{+}$-$Dcm^{+}$ strain after transfection into protoplasts. The restriction enzyme *Mbo*I exclusively digests DNA containing unmethylated adenine residues within its recognition sequence, GATC. Therefore, plasmid DNA is initially resistant to *Mbo*I digestion, whereas after a few replication cycles in Petunia cells, the plasmid becomes sensitive to *Mbo*I digestion. Total DNA was isolated from protoplasts 2, 4 and 6 days after transfection and was digested with *Mbo*I and a second, methylation-insensitive restriction enzyme that linearizes both plasmids. Southern blot hybridization analysis using a 1024 bp *Mbo*I fragment of the two plasmids as a probe showed that *Mbo*I digestion released the 1024 bp fragment in increasing amounts at 2, 4, and 6 days after transfection in pNT150MiniDs-f1DIR/Rep transfected protoplasts. However, this fragment was not detected in pNT150MiniDs-f1DIR transfected protoplasts. This result shows that only the plasmid containing the viral replicon becomes *Mbo*I-sensitive in transfected protoplasts, indicating that pNT150MiniDs-f1DIR/Rep actively replicates in the cells, but not pNT150MiniDs-f1DIR. These results suggest that DNA replication improves transposition of the *Ds* element, independently from the methylation status of its DNA.

(2) Our model predicts that fully methylated *Ds* should be unable to transpose in the absence of replication. After replication, supposedly only one of the two hemimethylated daughter elements is transposition competent. To test this model, we have treated pNT150MiniDs-f1DIR and pNT150MiniDs-f1DIR/Rep plasmids with *Sss*I methylase that methylates all cytosine residues to $^{5m}C$ within the double-stranded dinucleotide recognition sequence 5'...CG...3'. After the reaction all CpG dinucleotides in the plasmids, including the *Ds* element, are methylated. We have obtained preliminary results after transfection of protoplasts with C-methylated and unmethylated reporter plasmids and subsequent GUS staining (Table 1):

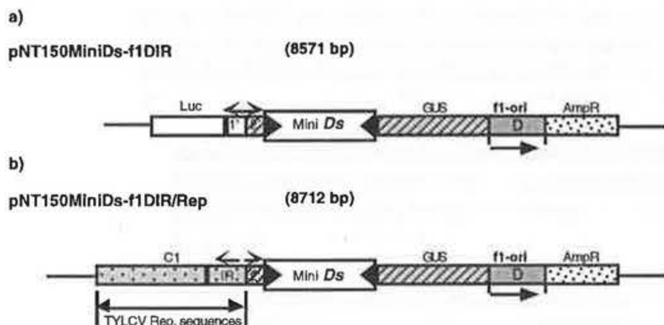


Figure 1. **a)** pNT150MiniDs-f1DIR construct. **b)** pNT150MiniDs-f1DIR/REP construct. **Luc**: Luciferase gene, **f1-ori**: origin of replication of M13 bacteriophage, with **D** is just indicated the orientation of the f1 replication origin, **2'**: GUS gene promoter, **1'**: Luciferase gene promoter, **MiniDs**: 700-bp Ds element, **GUS**: GUS gene, **AmpR**: ampicillin resistance, **IR**: region containing the promoter and the DNA binding sites for the viral C1 protein, **C1**: viral protein responsible for the replication function.

Table 1. Number of GUS positive protoplasts after transfection with C-methylated and unmethylated reporter plasmids.

| Plasmid | No. of blue protoplasts |
|---|---|
| pNT150MiniDs-f1DIR | 170 |
| pNT150MiniDs-f1DIR/Rep | 450 |
| pNT150MiniDs-f1DIR-*Met* | 2 |
| pNT150MiniDs-f1DIR/Rep-*Met* | 230 |

The frequency of GUS-positive protoplasts with the non-replicating, fully C-methylated plasmid is almost zero, whereas the replicating plasmid gives rise to normal frequencies. The lack of GUS-positive protoplasts with pNT150MiniDs-f1DIR-*Met* can be explained by one of two mechanisms. Either, in accordance with our model the fully methylated *Ds* is unable to transpose in the absence of replication and thus the GUS gene remains blocked, or alternatively, the *Ds* can excise, but due to the methylation the 2'-promoter persists in an inactive state. Experiments are under way to distinguish between these mechanisms. Preliminary (yet unconfirmed) PCR data indicate that the *Ds* excision frequency is significantly reduced (more than one order of magnitude) in pNT150MiniDs-f1DIR-*Met* compared to pNT150MiniDs-f1DIR/Rep-*Met*.

We have begun to investigate the effects of hemimethylation

on *Ds* transposition. Two pNT150MiniDs-f1DIR/Rep DNAs have been produced in which either one or the other DNA strand in the *Ds* element are CpG methylated, whereas outside *Ds* the plasmid is unmethylated. In the very first transfection experiment with these two DNAs, the *Ds* excision frequencies differed significantly. If these data can be confirmed, they strongly support our model.

### Isolation of a second MutS-homolog from *Zea mays* named Mus2

--Horwath, M, Kunze, R

The DNA mismatch repair system (MMR) is highly conserved among different organisms like *E. coli* or *H. sapiens*. In *E. coli* the MMR system consists of three main proteins, MutH, MutS and MutL. The MutS protein is responsible for the recognition and binding to mispaired nucleotides and small single strand DNA loops. In eukaryotes the mismatch repair machinery is much more complex. For example, in *S. cerevisiae* six different MutS (MSH) and 4 MutL (MLH) homologs have been found. In yeast and human it has been demonstrated that MSH2 forms heterodimers with MSH6 (involved in repair of basepair mismatches and 1 nucleotide

**MSH2 homologous proteins**

```
arathMSH2     VTGPNMGGKSTFIRQVGVIVLMAQVGSFVPCDKASISIRDCIFARVGAGDCQLRGVSTFM
Mus1          ITGPNMGGKSTFIRQVGVNVLMAQVGSFVPCDQASISVRDCIFARVGAGDCQLHGVSTFM
hMSH2         ITGPNMGGKSTYIRQTGVIVLMAQIGCFVPCESAEVSIVDCILARVGAGDSQLKGVSTFM
yMSH-2        ITGPNMGGKSTYIRQVGVISLMAQIGCFVPCEEAEIAIVDAILCRVGAGDSQLKGVSTFM
              :**********:***.**   ****:*.****:.*.::: *.*:.******.**:******

arathMSH2     QEMLETASILKGASDKSLIIIDELGRGTSTYDGFGLAWAICEHLVQVKRAPTLFATHFHE
Mus1          QEMLETASILKGASDKSLIIIDELGRGTSTYDGFGLAWAICEHLMEVTRAPTLFATHFHE
hMSH2         AEMLETASILRSATKDSLIIIDELGRGTSTYDGFGLAWAISEYIATKIGAFCMFATHFHE
yMSH-2        VEILETASILKNASKNSLIIVDELGRGTSTYDGFGLAWAIAEHIASKIGCFALFATHFHE
               *:*******:.*:..****:********************.*::      .  :*******

arathMSH2     LTALAQANSEVSG--NTVGVANFHVSAHIDTESRK-------LTMLYKVEPGACDQSFGI
Mus1          LTALAHRNDDEHQHISDIGVANYHVGAHIDPLSRK-------LTMLYKVEPGACDQSFGI
hMSH2         LTALANQIPTVNN---------LHVTALTTEET---------LTMLYQVKKGVCDQSFGI
yMSH-2        LTELSEKLPNVKN---------MHVVAHIEKNLKEQKHDDEDITLLYKVEPGISDQSFGI
              ** *:.                  ** *                :*:**:*: * .******

arathMSH2     HVAEFANFPESVVALAREKAAELEDFSPSSMIIN--NEESGKRKSREDDPDEVSRGAERA
Mus1          HVAEFANFPEAVVALAKSKAAELEDFSTTPTFSDDLKDEVGSKRKRVFSPDDITRGAARA
hMSH2         HVAELANFPKHVIECAKQKALELEEFQYIGESQGYDIMEPAAKKCYLER-E---QGEKII
yMSH-2        HVAEVVQFPEKIVKMAKRKANELDDLKTNNEDLK--KAKLSLQEVNEGNIRLKALLKEWI
              ****..:**: ::  *: ** **:::.                : . :.
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**MSH6 homologous proteins**

```
Mus2          RFALLLTGPNMGGKSTIMRATCLAVVLAQLGCYVPCTSCELTLADSIFTRLGATDRIMTG
hMSH6         AYCVLVTGPNMGGKSTLMRQAGLLAVMAQMGCYVPAEVCRLTPIDRVFTRLGASDRIMSG
yMSH6         PRLGLLTGANAAGKSTILRMACIAVIMAQMGCYVPCESAVLTPIDRIMTRLGANDNIMQG
                  *:**.* .****::* : : .::**:*****.  . ::  * ::*****.*.** *

Mus2          ESTFLVECTETASVLQKATVDSLVLLDELGRGTSTFDGYAIAYAVFRHLVERVRCRQLFA
hMSH6         ESTFFVELSETASILMHATAHSLVLVDELGRGTATFDGTAIANAVVKELAETIKCRTLFS
yMSH6         KSTFFVELAETKKILDMATNRSLLVVDELGRGGSSSDGFAIAESVLHHVATHIQSLGFFA
              :***:** :** .:*  **  **:::****** :: ** *** :*.:.:.  ::.  :*:

Mus2          THYHSLTKEFASQPHVSLQHMACMFKPRSDGNGQ---KELTFLYRLTSGACPESYGLQVA
hMSH6         THYHSLVEDYSQNVAVRLGHMACMVENECEDPSQ---ETITFLYKFIKGACPKSYGFNAA
yMSH6         THYGTLASSFKHHPQVRPLKMSILV----DEATR----NVTFLYKMLEGQSEGSFGMHVA
              *** .*...: : *  :*: .   :  :   :.***:: .* .  *:*::.*

Mus2          AMAGIPKSIVEKASVAGQ--------VMRAKIAGNFKSSEQRAEFSTLHEEWLRAALAVS
hMSH6         RLANLPEEVIQKGHRKAR--------EFEKMNQS---------------LRLFREVCLAS
yMSH6         SMCGISKEIIDNAQIAADNLEHTSRLVKERDLAANNLNGEVVSVPGGLQSDFVRIAYGDG
              :..:.:.:::..  .          .                  .:    .
```

Figure 1. Alignment of the highly conserved C-terminal regions of MSH2 and MSH6 homologous proteins. The maize homologs Mus1 and Mus2 are underlined. The putative nucleotide binding regions are shown in bold letters, the putative helix-turn-helix motif is underlined. atMSH - *Arabidopsis thaliana*: MSH2 (3914056); hMSH - *Homo sapiens*: MSH2 (1171032), MSH6 (1082386); yMSH - *Saccharomyces cerevisiae*: MSH2 (2506880), MSH6 (3024187); mus1 and mus2 - *Zea mays* (Genbank accession numbers are shown in parentheses)

loops) and MSH3 (involved in repair of small single strand DNA loops).

As in all known MutS homologous proteins certain amino acids in the C-terminal part are highly conserved, we were able to search for homologous sequences by RT-PCR with degenerate oligonucleotides. Using this approach, we isolated two MutS-homologous cDNAs, *mus1* and *mus2*. Sequence comparisons with MutS-homologs from bacteria, yeast and mammals revealed that *mus1* is most closely related to MSH2 from yeast (MNL 71, pp. 63-64, Figure 1) and *Arabidopsis*, whereas *mus2* is most similar to MSH6 genes. By Southern blotting we found that *mus2* is – like *mus1* – a single copy gene. The *mus2*-probe detects on a Northern blot a very weakly expressed transcript with a length of approximately 4.1 kb. With the same probe we isolated a 2.9 kb cDNA clone lacking the 5'- and 3'-ends from a maize seedling cDNA library (kindly supplied by Monika Frey, Technical University of Munich). The sequence analysis of a genomic Lambda clone suggests that the coding region is at least 3.5 kb long and codes for a putative protein of 1185 amino acids. The 5'- and 3'-ends of the cDNA have to be confirmed yet.

Figure 1 shows the alignment of the most highly conserved regions of MSH2 and MSH6 proteins. It turns out that the sequences of the MSH2 proteins are more conserved over the region of the four putative nucleotide binding domains, and especially in the helix-turn-helix motif.

In collaboration with Monika Frey (Technical University of Munich) we RFLP-mapped the *mus2* gene by using a recombinant inbred population (Burr et al., Genetics 118:519-526,1988; Burr and Burr, TIG 7: 55-60, 1991). *Mus2* maps on chromosome 3S.

With the aim of investigating the biochemical properties of the maize Mus1 and Mus2 proteins, we began to establish an overexpression system in *E. coli*. By optimization of the expression and purification conditions we were able to produce Mus1 in a soluble form. However, in gel shift experiments with DNA probes containing a mismatch this protein exhibits no mismatch specific binding activity.

In analogy to the properties of MSH2 and MSH6 proteins in yeast and mammals (Palombo et al., Science 268:1912-1914, 1995; Iaccarino et al., EMBO J. 17: 2677-2686, 1998), it seems possible that Mus1 is only functional as a heterodimer with MSH3 or MSH6 proteins.

To this end we have first hints for an interaction between Mus1 and Mus2, as the latter can only be overexpressed in *E. coli* by co-expressing Mus1. This implies some kind of stabilization of Mus2 by Mus1.

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**Accumulation of satellites in hybrids of maize with *Tripsacum***
--Sokolov, VA, Khatypova, IV

Earlier in a maize-*Tripsacum* hybrid line a chromosome with two satellites on both ends was discovered (Fig. 1). In maize the nucleolus-organizing chromosome is chromosome 6 and in *Tripsacum* chromosome 16 (Kindiger et al., Genome, 39:1133-1141, 1996). Proceeding from the morphology of the two-satellite chromosome and the lack of *Tripsacum* chromosome 16 in the newly-arisen line, we came to the conclusion that it was formed by a transfer of the

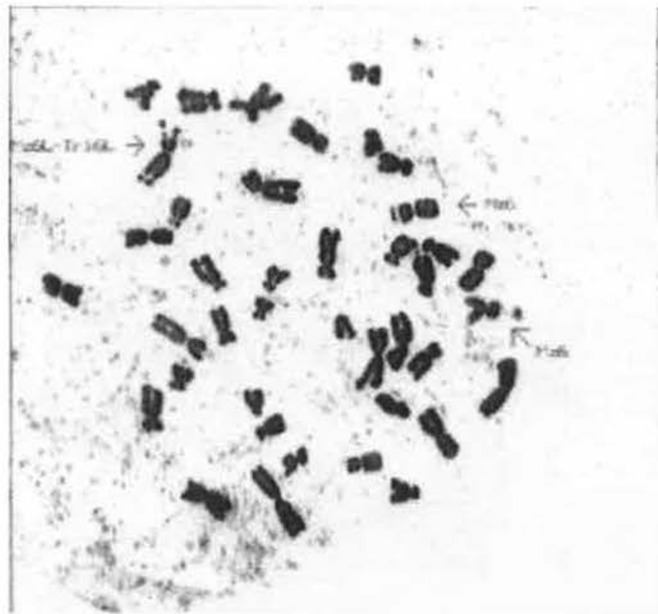

Figure 1.

long arm of the nucleolus-organizing gamagrass chromosome to the long arm of maize chromosome 6. The appearance of a chromosome with two satellites may be a result of meiotic crossing-over of these chromosome kinds due to available homology (Galinat, Evolution, 27:644-655, 1973; Blakey, Ph.D. dissertation, No.9412465, University of Missouri, Columbia, 1993).

In previously published results associations of maize chromosomes with *Tripsacum* chromosomes and mutual translocations were shown in meiosis (Maguire, Evolution, 15:394-400, 1961; de Wet and Harlan, Genetics, 78:493-502, 1974). From our data crossover exchanges between nucleolus-organizing chromosomes are also frequent enough and we found their independent emergence in three lines. Perhaps the repetition of the translocations observed by us is analogous to those described in hybrids of closely related sunflower species (Riesebery et al. Science, 272:741-745, 1996).

In 1997 in one plant from a line carrying a reconstructed chromosome 6MzL-16TrL we found a third satellite (Fig. 2, 3). As seen from the figures the extra (third) satellite immediately adjoins the "maize" satellite in the reconstructed chromosome. Such a phenomenon, though with two satellites only, was observed in progeny of maize-*Tripsacum* hybrids obtained by J. R. Harlan. The additional satellite next to that usual for chromosome 6 was found by B. Kindiger and S. A. Modena (MNL, 58:67, 1984).

We do not have experimental results explaining the mechanism of formation of the additional satellite. One can only assume a specific duplication of the nucleolus-organizing region and the satellite, or unequal crossing-over as a result of complexity of the reconstructed chromosome. Another possible explanation may be reactivation of the nucleolus-organizing region that remained quiet if adopting the hypothesis of maize tetraploid nature and the formation of chromosome 6 from two satellite chromosomes (Leitch and Bennett, Trends Plant Sci., 2(12):470-476, 1997). Other explanations are possible, however, for their adoption special experiments are needed.

For the present it is obvious that the "duplication" of the nucleolus-organizing and satellite regions has some repetition in the

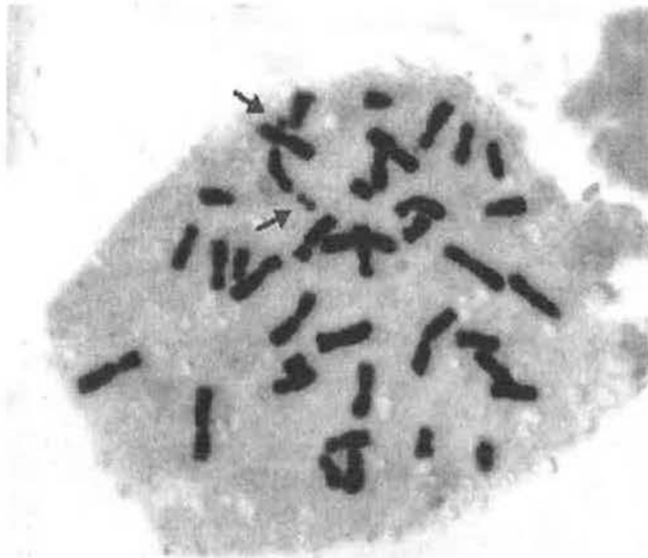

Figure 2.

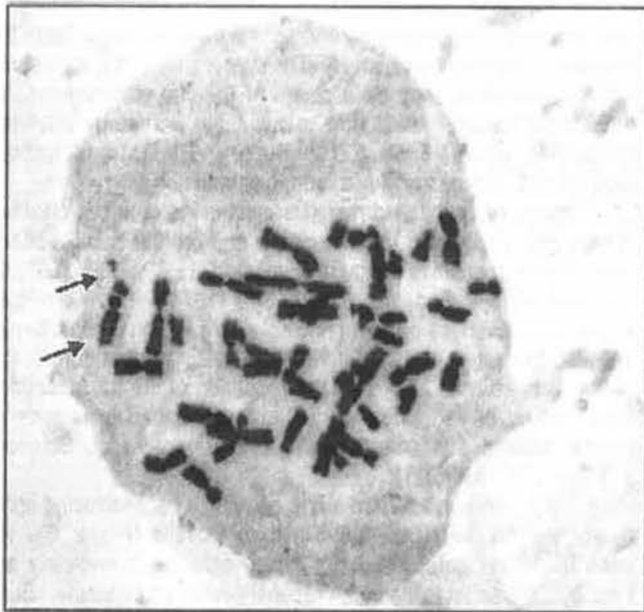

Figure 3.

progeny of maize-*Tripsacum* hybrids and that the material from Russia carries a larger amount of satellites. The latter is in complete accordance with the high activity of their space research from the moment of the launching of the first satellite in 1957.

Acknowledgement: We are deeply thankful to Chet Dewald and B. Kindiger for their help in preparation of the report in question.

**Inactivation of the imprinting effects in maize-*Tripsacum* hybrids**
--Sokolov, VA, Khatypova, IV

One of the main reasons for "incrossability" of different ploidy plants of one and the same species or close species is seed abortion conditioned by endosperm non-development. This phenomenon has been known for more than 70 years and for a long time hypotheses explaining it have been reduced to three possibilities:

1. deviation from the 2 : 3 : 2 ratio between numbers of maternal tissue endosperm and embryo genomes respectively (Muntzing, A, Hereditas 14:27- 42, 1930);
2. deviation from the 3 : 2 ratio of endosperm genomes to that of embryo (Watkin, AE, J. Genetics 25:125-162, 1932);
3. deviation from the 2 : 3 ratio of number of genomes in maternal cells to that of endosperm (Valentine, DM, Proc. 8 Intern. Botany Congress, Paris, p.170, 1954).

However as experimental methods of genetics progressed it was getting more and more obvious that the reasons for the anomalous development of endosperm are in its own genotypic structure (Johnston, SA et al., Theor. Appl. Genet. 57:5-11, 1980).

Classic experiments by Lin and the hypothesis formulated by him of parental imprinting of genomes in maize became the turning-point in our understanding of genetic mechanisms controlling the normal morphogenesis of endosperm (Lin, B-Y, Genetics 100:475-486, 1982; Genetics 107:103-115, 1984). It lies in the fact that values of expression of developmental genes introduced by maternal and paternal genomes are unequal. Therein any deviation from the ratio of two maternal genomes (2m) : one paternal (1p) leads to imbalance and seed abortion.

By now, thanks to Birchler and other researchers' works, we understand that besides imprinting, endosperm development is also influenced by other factors, but to all appearances imprinting is the main factor among them (Birchler, JA, Ann. Rev. Genet. 27:181-204, 1993).

Our interest in this phenomenon has to do with work on transfer of apomixis (pseudogamous type diplospory) from *Tripsacum* to maize where we met with a problem of seed size in apomictic intergeneric hybrids and with high sterility partly associated with ovary death in early stages of development. In a work published by Daniel Grimanelli with co-workers (Sex. Plant Reprod. 10:279-282, 1997) absence of imprinting effect in crossing diploid and tetraploid *Tripsacum* species (*Poaceae*) was shown. The same was also evidenced by the results obtained at our laboratory earlier when no dosage effects were observed in apomictic $B_{III}$-hybrids of maize with *Tripsacum* with 2 to 9 maize genomes accumulated (Petrov, DF et al., Apomixis and its role in evolution and breeding, New Delhi, India, 9-73, 1984; Kindiger, B and Sokolov, VA, Trends in Agronomy, 1:75-94, 1997).

The material for this research was progeny of an F1 hybrid obtained in 1964 from crossing tetraploid maize with *Tripsacum* having a genome formula 2n=56 (20Mz + 36Tr). As a result of backcrossing, numerous $B_{II}$ and $B_{III}$-hybrids with a different ratio between complete parental genomes or between complete maize genomes and *Tripsacum* subgenomes or separate chromosomes were derived. In this case the term "subgenome" was given by us to a stable group of 9 chromosomes of the wild parent (obtained in one of the $B_{II}$-hybrids) the carriers of which have been steadily exhibiting for 20 years the same apomixis as the hybrids with a complete genome of 18 chromosomes. The pedigrees of these hybrids were published earlier (Sokolov, VA et al., Russian Genetics 34:499-506, 1998).

The exception is a line with 63 chromosomes developed from backcrossing a 29-chromosome $B_{II}$-hybrid (20Mz + 9Tr) in one of its generations. This $B_{II}$-hybrid pedigree will be reported by us in a separate publication later. Here we'll only mention that the 63-chromosome twins originated from a 43-chromosome plant as a result of fertilization by tetraploid pollinator of unreduced egg cell ($B_{III}$-hybrid) (see Fig. 1). From these twin plants after polli-

nation with tetraploid some 50 filled but small size seeds - subnormal by Lin's terminology - were obtained by us. Unfortunately only part of them were viable and all plants obtained from them were $B_{II}$-hybrids with 50 maize chromosomes and an addition of 3 (about 50% - Fig. 2) to 1 or 2 *Tripsacum* chromosomes. They were not totally female fertile and so only part of them produced seeds, and only when pollinated with tetraploid. The cytogenetic structure of this generation has not been analysed yet.

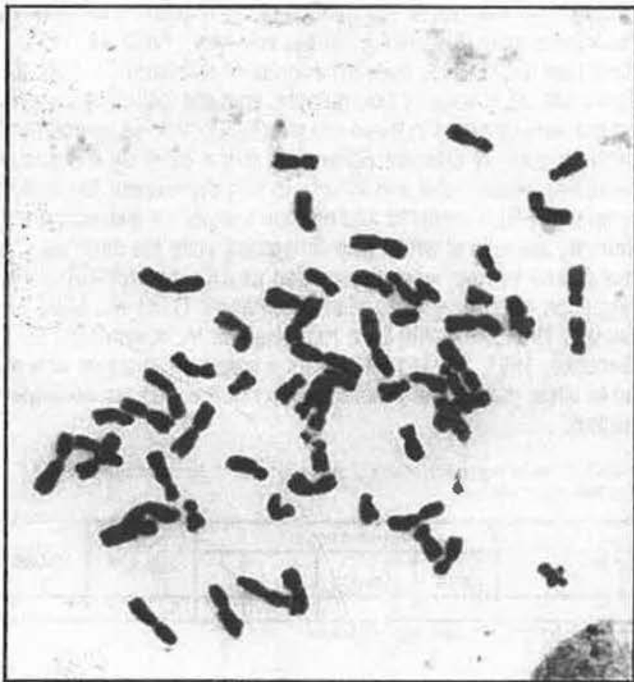

Figure 1. The $B_{III}$-hybrid 2n=63 (60Mz + 3Tr).

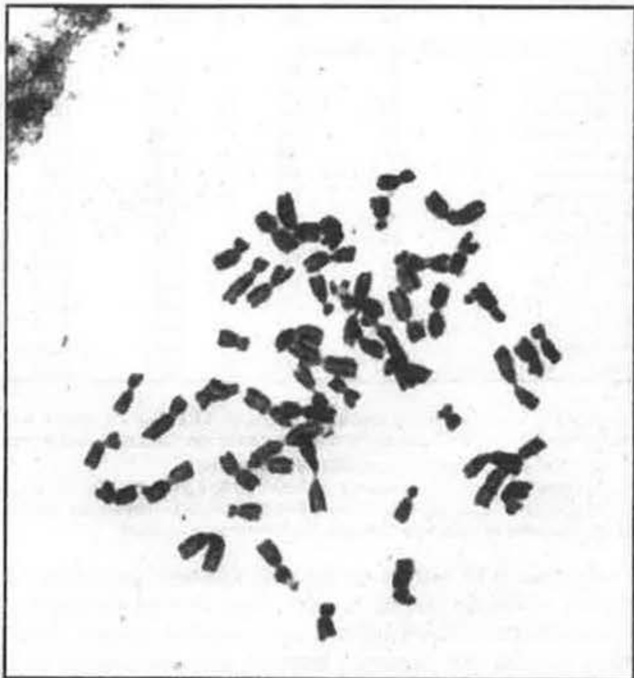

Figure 2. The $B_{II}$-hybrid from 2n=63, 2n=53 (50Mz + 3Tr).

Apomixis in gamagrass and, therefore, in the hybrids is pseudogamous diplospory of the *Antennaria*-type. So for the kernel to develop normally fertilization of the polar nucleus is needed. The results of pollination with tetraploids (pollinator 2n=4x) and diploids (pollinator 2n=2x) are reflected in the ratios between maternal and paternal genomes presented in the table. The relative seed size, we put down in the column "normal" of the table, in Fig. 3 is designated by numbers 1 and 2, the "subnormal" size is indicated by number 3.

Table.

| Genotypes of apomictic line | Ratio between genomes in endosperm pollinated with | | Seed size | |
|---|---|---|---|---|
| | tetraploid | diploid | Normal | Subnormal |
| 38 20Mz + 18Tr | 4m : 2p | 4m : 1p | + | |
| 48 30Mz + 18Tr | 6m : 2p | 6m : 1p | + | |
| 58 40Mz + 18Tr | 8m : 2p | 8m : 1p | + | |
| 39 30Mz + 9Tr | 6m : 2p | 6m : 1p | + | |
| 49 40Mz + 9Tr | 8m : 2p | 8m : 1p | + | |
| 59 50Mz + 9Tr | 10m : 2p | ? | + | |
| 79 70Mz + 9Tr | 14m : 2p | ? | + | |
| 78 60Mz + 2 x 9Tr | 12m : 2p | ? | | + |
| 96 60Mz + 2 x 18Tr* | 12m : 2p | - | - | - |
| 63 60Mz + 3Tr** | 8m : 2p | ? | | + |
| 53 50Mz + 3Tr | 6m : 2p | ? | | + |

*A dwarf plant does not eject silk, does not set seeds

**Nonreduction without parthenogenesis

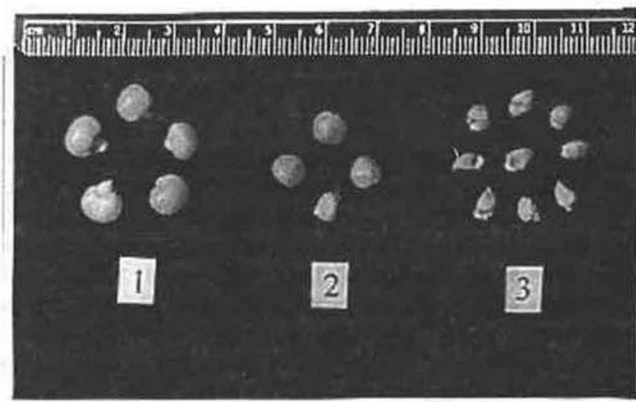


Figure 3. The relative seed size: 1, 2n=39 (30Mz + 9Tr); 2, the offspring of 2n=51 (50Mz + 1Tr); 3, 2n=53 (50Mz + 3Tr).

As follows from the results presented in the table the varying of the ratio between maize genomes of maternal and paternal origin within a wide range after pollination with tetraploid (from 4m : 2p to 14m : 2p) as well as diploid (from 4m : 1p to 8m : 1p) does not lead to imprinting manifestation, and both endosperm and embryo develop normally producing viable seeds. In cases when the lines were not pollinated with diploid, and so we lacked appropriate results from this manipulation, this fact is marked as "?" in the table. There's no doubt that this is observed by reason of the presence in the hybrids of a complete *Tripsacum* genome or its part of 9 chromosomes in which case "normal" endosperm develops. With three *Tripsacum* chromosomes available in the $B_{III}$-hybrid (2n=63) and then in its reduced progeny (the $B_{II}$-hybrid 2n=53), abnormally small "subnormal" endosperm is formed.

With a very low frequency plants with genome duplications (2n=78; 2 x 30Mz + 2 x 9Tr and 2n=96; 2 x 30Mz + 2 x 18Tr) are encountered. They appear to be a result of lack of one single megaspore division in rare cases of meiotic diplospory in *Tripsacum*. The plant having 96 chromosomes developed abnor-

mally - it was dwarf, with a very little ear, did not eject silk and produced no progeny. From the 78-chromosome plant not differing in phenotype from the 39-chromosome sibs, with its sufficiently high sterility, we managed to obtain seeds and these were subnormal in size.

Thus, based on the results presented, one may conclude that the availability of *Tripsacum* chromosomes in some way inhibits imprinting expression in the hybrids. And it's quite obvious that for its total suppression some critical number of *Tripsacum* chromosomes is needed. In this stage of the research we can assert the validity of this conclusion for 9 chromosomes.

The development of the subnormal size grains obtained from the 78-chromosome plants may be explained by imbalance between maize genomes and twice the set of 9 *Tripsacum* chromosomes. It need be noted that despite twice the chromosomes these plants are male sterile, moreover the development of their anthers is disturbed and meiosis does not occur in the anthers.

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**Effect of components of the *Bg-rbg* system of transposable elements on reversion frequency of the mutable allele *o2-m(r)* and the evolutionary role of transposable elements**

--Koterniak, VV

Reversion of the mutable allele *o2-m(r)* (formed by an insertion of the receptor element *rbg* in the *o2* locus) to normal *O2* occurs in the presence of the regulatory element *Bg* as a result of *rbg* excision from the *o2* locus, and phenotypically is expressed in restoration of the normal (vitreous) endosperm structure. In cases when such a reversion takes place during gametophyte development or at the early stages of endosperm development it leads to formation of kernels which are indistinguishable from normals: whole endosperm revertants (WER).

*Changing frequency of reversions by selection.* The frequency of reversion of *o2-m(r)* in our initial source of instability designated as 3449*o2*, derived from selfed generations of a simple hybrid of the *o2-m(r)/o2-m(r) +Bg/+Bg* x *O2/O2 Bg/Bg* genotype, can be significantly and rapidly changed by ear selection. Disruptive selection was started in 1990 from ear 89-2911-1 (F5 kernels of this ear contained 8.0 % of WER), and carried out for two generations of selfing. Ears were obtained with WER frequencies from 0.8 to 53.7%, i. e. with gametic frequency equal to 0.3 and 17.9%, respectively (Koterniak V. V., Russian Journal of Genetics, 1995, 31:950-954). The lines with low and high frequency of WER formation were designated respectively as 3449 *o2-lf Bg-lf* and 3449 *o2-hf Bg-hf* (or LFWER and HFWER). In the following it will be shown that the additional specification of *o2-m(r)* alleles and *Bg* elements is justified.

In 1993-1997 selection for WER frequency was looser since we did not try to obtain HFWER families with WER frequency significantly higher than 50% (in this case it would be difficult to distinguish ears homozygous and heterozygous for *o2-m(r)*). We also did not try to obtain LFWER lines with WER frequency equal to zero to have the possibility of studying normal descendants of these lines. Thus for planting in 1997 ears were selected with gametic frequencies of reversion of the receptive alleles within the limits of 0.18 - 0.30 and 14.64 - 17.00 % for *o2-lf* and *o2-hf* respectively.

*Reversion frequency of the mutable o2-m(r) allele is determined by its state.* To determine the effect of selection on the state of responsive allele *o2-m(r)* we analyzed the F1 kernels obtained in 1997 by crossing an *o2-R Bg* line (courtesy of F. Salamini) with LFWER and HFWER lines lacking the regulatory element as a result of its loss or inactivation (Table 1). The possibility of the loss or inactivation of the regulatory element was reported earlier (Salamini F. et al., Heredity, 1982, 49: 111-115; Koterniak V.V., 1995, Russian Journal of Genetics 31: 950-954). Since the *o2-R* allele is not mutable, somatic instability observed on the ears obtained in these crosses is conditioned by interaction of the regulatory element *Bg* present in the *o2-R Bg* line and the receptive alleles *o2-lf* and *o2-hf*. In this experiment besides the normal (WER), variegated and opaque kernels we also counted the kernels, one side of which was variegated while the other was normal (these kernels were designated as 1/2n or 1/2WER). Their formation was reported earlier (Montanelli C. et al., Mol. Gen. Genet., 1984, 197: 209-218; Koterniak V. V., Karaivanov G. P., Genetica, 1991, 27: 1814-1819) as a result of reversion of a mutable allele during first mitotic division of the primary endosperm nucleus.

Table 1. F1 kernel segregation on ears of crosses of an *o2-R Bg* tester with 3449 *o2-lf +Bg* and 3449 *o2-hf +Bg* lines.

| Ear No | Number of kernels + | | | | WER, %++ | 1/2WER,% |
|---|---|---|---|---|---|---|
| | n (WER) | 1/2n (1/2WER) | v | o | | |
| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
| *o2-R o2-R Bg Bg* x *o2-lf o2-lf +Bg +Bg* crosses | | | | | | |
| 9258p252/ *o2-lf* | - | 3 | 129 | 4 | 0 | 2.27 |
| 9264p253/ *o2-lf* | - | 2 | 107 | 3 | 0 | 1.83 |
| (9266/*o2-lf*)-1 | - | 10 | 193 | 1 | 0 | 4.93 |
| (9266/*o2-lf*)-2 | - | 2 | 61 | - | 0 | 3.17 |
| 9267/*o2-lf* | - | | 60 | - | 0 | 0 |
| 9269/*o2-lf* | 1 | 4 | 105 | 3 | 0.91 | 3.64 |
| Total | 1 | 21 | 655 | 11 | 0.15** | 3.10*** |
| *o2-R o2-R Bg Bg* x *o2-hf o2-hf +Bg +Bg* crosses | | | | | | |
| 9258/*o2-hf* | 1 | 10 | 89 | 3 | 1.00 | 10.00 |
| 9258p12/ *o2-hf* | 1 | 9 | 109 | - | 0.84 | 7.56 |
| 9258p13/ *o2-hf* | 9 | 39 | 149 | 11 | 4.57 | 19.80 |
| 9263p269/ *o2-hf* | 3 | 22 | 100 | - | 2.40 | 17.60 |
| 9263/*o2-hf* | 1 | 31 | 129 | - | 0.62 | 19.25 |
| (9264/*o2-hf*)-1 | 3 | 17 | 153 | - | 1.73 | 9.83 |
| (9264/*o2-hf*)-2 | 2 | 22 | 121 | 6 | 1.38 | 15.17 |
| (9266/*o2-hf*)-1 | 5 | 17 | 133 | - | 3.23 | 10.97 |
| (9266/*o2-hf*)-2 | 2 | 22 | 100 | - | 1.61 | 17.74 |
| (9267/*o2-hf*)-1 | 1 | 24 | 131 | 2 | 0.64 | 15.38 |
| (9267/*o2-hf*)-2 | 2 | 17 | 114 | - | 1.50 | 12.78 |
| 9269/*o2-hf* | 2 | 6 | 85 | - | 2.15 | 6.45 |
| (9270/ *o2-hf*)-1 | 4 | 20 | 103 | 9 | 3.15 | 15.75 |
| (9270/*o2-hf*)-2 | 3 | 31 | 88 | 1 | 2.46 | 25.41 |
| Total | 39 | 287 | 1604 | 32 | 2.02 | 14.87 |

+ n (WER), v, o - normal (whole endosperm revertants), variegated and opaque kernels respectively. 1/2n (1/2 WER) - kernels 1/2 of which is normal and the other half is variegated.

++ - % of WER was calculated without considering opaque kernels.

**, *** - significance of differences between *o2-R o2-R Bg Bg* x *o2-lf o2-lf +Bg +Bg* and *o2-R o2-R Bg Bg* x *o2-hf o2-hf +Bg +Bg* crosses at P=0.01 and P=0.001, respectively. Here and in the following tables significance of differences was determined by t-criterion.

Analysis of F1 kernels on the ears obtained by crossing *o2-R Bg* with *o2-lf +Bg* and *o2-hf +Bg* lines showed significant differences both in WER frequency and in 1/2WER content. Since in these crosses the regulatory element was represented by the standard *Bg* but not by the regulatory elements existing in the lines undergoing disruptive selection for WER frequency, it is

possible to make the conclusion that frequency of formation of WER is conditioned by the receptive alleles *o2-lf* and *o2-hf* present respectively at LFWER and HFWER lines. This means that selection for WER frequency resulted in the changes in the state of the initial *o2-m(r)* allele. The change in state phenomenon is known for different systems of transposable elements and for the *o2-m(r)* allele and was reported earlier by Montanelli C. et al (1984) under selection for a different pattern of kernel variegation.

Data obtained also demonstrate that some ears have small numbers of opaque kernels (Table 1). Their presence can be explained by the loss or inactivation of the regulatory element, the possibility of which was mentioned above. It is interesting to note that in some cases we observed kernels 1/2 of which were variegated and the other half opaque, showing that this loss or inactivation can take place at the same stages of endosperm development at which the 1/2WER formation occurs.

Differences in reversion frequencies of receptive alleles contained in LFWER and HFWER lines seem not to be connected with presence or absence of other genes (modifiers). This conclusion can be reached by comparing gametic frequencies of mutable alleles obtained when analyzing selfed ears of the hybrids from the crosses of LFWER and HFWER lines with the *opaque2* and normal testers carrying alleles which did not pass through selection for WER frequency (Table 2). The testers were homozygous for *o2-m(r)* or *O2* alleles and did not contain regulatory element *Bg.*

Table 2. Results of kernel segregation on ears obtained by selfing the hybrids of 502 *O2 +Bg* and 502 *o2-m(r) +Bg* testers with 3449 *o2-lf Bg-lf* and 3449 *o2-hf Bg-hf* lines (1997 year data).

| Genotype | Number of ears | Number of kernels | | | Gametic frequency (gf),%* |
|---|---|---|---|---|---|
| | | n | v | o | |
| (*O2/o2-lf Bg-lf/+Bg*)@ | 16 | 5442 | 1227 | 364 | 4.00a |
| (*o2-m(r)/o2-lf Bg-lf/+Bg* )@ | 9 | 294 | 2693 | 893 | 4.92a |
| (*O2/o2-hf Bg-hf/+Bg*) @ | 11 | 3595 | 530 | 232 | 17.88b |
| (*o2-m(r)/o2-hf Bg-hf/+Bg*) @ | 13 | 900 | 2510 | 954 | 13.20c |

* - a common letter at the means indicates insignificance of the differences between them (P=0.05).

In Table 2 total data of kernel segregation are presented on selfed ears of crosses of LFWER and HFWER lines with normal and *o2-m(r)* testers of 502 line background. Here for analysis ears were used with a segregation ratio of normal and variegated kernels to opaque ones not significantly different from 3:1 (for descendants of *o2-m(r) +Bg* tester) or from 15:1 (for descendants of *O2 +Bg* tester). These ratios indicate ears that were obtained by selfing plants heterozygous for one copy of the regulatory element, and allow calculating gametic frequencies (gf, %) by the formula gf=100n/(2(n+v)) for descendants of the *o2-m(r) +Bg* tester, and by the formula gf=100(n-12/15(n+v))/(6/15(n+v)) for descendants of the *O2 +Bg* tester, where n and v are the number of the normal and variegated kernels respectively.

In the F2 generation of the crosses of LFWER lines with *O2 +Bg* tester the number of WER was lower, though insignificantly, as compared with selfed progenies of the crosses of the same lines with the *o2-m(r) +Bg* tester. This can be explained by the fact that in the case of *o2-m(r) +Bg* tester a portion of gametes which took part in WER formation carried alleles (brought in by the 502 *o2-m(r) +Bg* tester) not changed by selection (i. e. with a higher frequency of receptive allele reversion). Accordingly in the F2 generation of the crosses of HFWER lines with *O2 +Bg* tester the number of WER was significantly higher as compared with selfed progenies of the same lines with *o2-m(r) +Bg* tester. This also can be explained by the fact that in the case of *o2-m(r) +Bg* tester a portion of gametes which took part in WER formation carried alleles (brought in by the 502 *o2-m(r) +Bg* tester) not changed by selection (i. e. with a lower frequency of receptive allele reversion).

In case a high frequency of WER was determined by modifier genes not linked with the *o2* locus, it was expected that the selfed progeny of the *O2 /o2-hf Bg-hf/+Bg* crosses would have lower WER frequency in comparison with the same trait of selfed HFWER lines. However, the calculated genetic frequency of WER on the selfed *O2 /o2-hf Bg-hf/+Bg* crosses (17.88%, see table 2), not only was not lower than the gametic frequency of WER on selfed HFWER ears (in 1997 its value was equal to 11.40%), but even exceeded the latter (probably due to genotypic differences between HFWER lines and their crosses with 502 *O2 +Bg* line).

*Effect of the dose of the regulatory element* Bg *on frequency of reversion.* Expression of mutability at the *o2* locus is not connected with dosage effects for the standard *Bg* (Montanelli C. et al., 1984). However, a regulatory element (*Bg-7b3*) was described, one dose of which was insufficient for *rbg* excision (Motto M. et al., Maydica, 1989, 34: 107-122).

Analysis of the crosses obtained with participation of LFWER and HFWER lines permitted us to make some conclusions about the effect of selection for WER frequency on activity of regulatory elements *Bg-lf* and *Bg-hf,* expressed in effects of different doses of these elements on reversion frequencies of the mutable *o2* alleles studied.

To test the effect of different doses of the regulatory elements in LFWER and HFWER lines we made crosses of these lines with *opaque2* testers which contained the receptive alleles and lacked the regulatory element (Table 3). By comparing the WER frequency on selfed ears of LFWER and HFWER lines with that on ears obtained from crossing these lines (taken as male parent) with *opaque2* tester it was possible to compare the effect of the regulatory element in 3 and 1 doses respectively. By comparing the frequency of WER on the ears obtained from crossing LFWER and HFWER lines (used as male parent) with *o2-m(r) +Bg* tester with the frequency of WER on ears of reciprocal crosses, we compared the effect of regulatory element in 1 and 2 doses respectively. In this analysis only exactly reciprocal ears were included. Accordingly in studying effects of 1 and 3 gene doses only exactly paired ears were used. Besides the *o2-m(r) +Bg* tester which contained the *o2* allele not exposed to selection (502 *o2-m(r) +Bg* line), a 3449 *o2-hf +Bg* line (i.e. the HFWER line lacked regulatory element *Bg*) was used as a tester. Under selfing, this line was characterized by stable opaque endosperm structure.

Data obtained showed significant differences between LFWER and HFWER lines in the dependence of a receptive allele reversion frequency on the dose of the regulatory element. Different doses of the regulatory element *Bg-lf* present in LFWER lines did not significantly affect frequency of WER formation. Insignificant differences in WER frequency in the crosses of varied doses of this regulatory element were observed both for each year separately and in comparison between the years in which crosses with varied composition of receptive alleles were studied (in Table 3 significance of differences in WER frequency between the years is not shown). This indicates that by the dosage effect

Table 3. Kernel segregation on ears of selfed LFWER and HFWER lines and their crosses with 502 *o2-m(r) +Bg* and 3449 *o2-hf* +Bg testers (1994-1997 years data).

| Selfing or crossing | Bg dose | No. of ears | Number of kernels | | | Gametic frequency of WER, %+ | Gametic frequency of "o", % |
|---|---|---|---|---|---|---|---|
| | | | n (WER) | v | o | | |
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| 1994 | | | | | | | |
| 502 *o2-m(r) +Bg* x LFWER | 1 | 12 | 15 | 2054 | 7 | 0.72a++ | 0.34 |
| LFWER@ | 3 | 12 | 70 | 2384 | - | 0.95ab | - |
| 502 *o2-m(r) +Bg* x HFWER | 1 | 16 | 77 | 4002 | 39 | 1.89b | 0.95 |
| HFWER@ | 3 | 16 | 1232 | 2255 | - | 11.78c | - |
| 1995 | | | | | | | |
| 3449 *o2-hf +Bg* x LFWER | 1 | 11 | 12 | 1443 | - | 0.89a | - |
| LFWER@ | 3 | 11 | 55 | 1866 | - | 0.95a | - |
| 3449 *o2-hf +Bg* x HFWER | 1 | 13 | 61 | 1768 | 13 | 3.34b | 0.71 |
| HFWER@ | 3 | 13 | 937 | 1595 | - | 12.34c | - |
| 1997 | | | | | | | |
| 502 *o2-m(r) +Bg* x LFWER | 1 | 14 | 32 | 2947 | 7 | 1.07a | 0.23 |
| LFWER x 502 *o2-m(r) +Bg* | 2 | 14 | 59 | 3042 | - | 0.95a | - |
| 502 *o2-m(r) +Bg* x HFWER | 1 | 10 | 62 | 2249 | 13 | 2.68b | 0.56 |
| HFWER x 502 *o2-m(r) +Bg* | 2 | 10 | 677 | 1641 | 31 | 14.60c | 0.81+++ |

+- % of WER was calculated without considering opaque kernels.

++ - - a common letter at the means indicates insignificance of the differences between them for each year separately (P=0.05).

+++ - for HFWER x 502*o2-m(r) +Bg* ears calculation was performed by the formula gf=100(o/2(n+v+o).

for *Bg-lf* resembles standard *Bg*. We also note the resemblance between the receptive allele *o2-lf* of LFWER lines and the receptive allele *o2-m(r)* of the standard source of *Bg*: the mean gametic frequency of reversion in endosperm tissue is 0.86% for the first (see Table 4) and 0.78 for the latter (Salamini F., Cold Spring Harbor Symp. Quant. Bio., 1981, 45: 467-476).

In contrast with the *Bg-lf* element, frequency of WER formation depends strongly and positively on the dose of *Bg-hf,* the regulatory element present in HFWER lines. Thus on the ears obtained by the crosses of HFWER lines with 502*o2-m(r) +Bg* tester, 2 and 3 doses of *Bg-hf* conditioned a WER frequency respectively 5.4 and 6.2 times higher in comparison with that observed under 1 dose of the same regulatory element.

Results of the crossing of LFWER and HFWER lines with 3449 *o2-hf +Bg* tester were analogous to the results obtained by crossing with 502 *o2-m(r)+Bg* tester. Moreover, results of crossing with the 3449 *o2-hf +Bg* tester showed that observed differences in number of WER are conditioned not by the dose of the receptive allele (or by dose of the receptor element) but by the dose of the regulatory element. Thus the gametic frequency of reversion of the mutable allele *o2-hf* which leads to formation of the WER on the selfed ears of 3449 *o2-hf Bg-hf* lines (3 doses of *o2-hf* and 3 doses of *Bg-hf*) was equal to 12.34%, which is 3.7 times higher than the gametic frequency of reversion of *o2-hf* on the ears obtained by crossing 3449 *o2-hf +Bg* (female parent) with 3449 *o2-hf Bg-hf* (3 doses of *o2-hf* and 1 dose of *Bg-hf*).

It is interesting to note that in progenies of lines with high WER frequency in comparison with the progenies of lines with low WER, the frequency of emergence of derivatives lacking the active regulatory element *Bg* was also higher. Data in Table 3 show that the frequency of formation of opaque kernels (as a result of the loss or inactivation of the regulatory element) in the crosses of HFWER lines with the *o2-m(r) +Bg* tester was higher than in the crosses of LFWER lines with the same tester. The frequency of ears which were heterozygous for the regulatory element *Bg* was also higher in HFWER lines. Thus from 114 ears of HFWER lines and 95 ears of LFWER lines studied in 1992-1996 we found 4 heterozygous ears of HFWER lines and 1 heterozygous ear of LFWER lines, giving gametic frequency of their formation equal to 1.75 and 0.53 respectively. Among the causes which determined the enhanced formation of derivatives lacking the regulatory element at the HFWER lines could be: i) changes in *Bg-hf* which enhance frequency of its inactivation, and ii) higher frequency of the loss of *Bg-hf,* e.g. as a result of its nonreplicative transposition. Since the receptor element *rbg* is a deletion derivative of the regulatory element *Bg* which lost its ability for autonomous transposition (Hartings H. et al., MNL, 1992, 66: 20-21), we can assume that the product of the regulatory element *Bg-hf* not only increases the transposition of the receptor element but also the transposition of the regulatory element itself. This in turn increased the possibility of the loss of the latter as a result of nonreplicative transposition.

Proceeding from the above it is possible to conclude that disruptive selection for WER frequency resulted in changes of state of both the receptive allele and the regulatory element. Change in state of the initial receptive allele *o2-m(r)* led to new receptive alleles *o2-lf* and *o2-hf* which determine low and high frequency of *rbg* excision respectively. Change in state of the regulatory element *Bg* resulted in change of its ability to induce excision of the receptive element from the *o2* locus, expressed by dependence of the reversion frequency or the mutable alleles on the dose of the regulatory element. Frequency of *rbg* excision depends weakly and insignificantly on the dose of *Bg-lf.* In contrast with this, the frequency of *rbg* excision is characterized by strong positive dependence on the dose of *Bg-hf.*

Rapid derivation (during two generations of selfing) of the changed forms of transposable elements as a result of disruptive selection for WER frequency shows existence of significant heterozygosity in the state of the transposable elements in our initial source of instability used for selection. Since parental lines of this source were characterized by a high level of inbreeding and were homozygous for the receptive allele (the genotype of the female parent was *o2-m(r)/o2-m(r) +Bg/+Bg*), or for regulatory element (the genotype of the male parent was *O2/O2 Bg/Bg*), it is possible to conclude that the indicated heterogeneity is the result of inherent instability of transposable elements. Among the causes which determined this heterogeneity and led to the changes of the transposable elements could be self-inflicted intra-element deletions and changes in their pattern of methylation (Schiefelbein J. W. et al., Proc. Natl. Acad. Sci. USA, 1985, 82: 4783-4787; Schwarz-Sommer Zs. et al., EMBO J., 1985, 4: 2439-2443; Schwartz D., Dennis E., Mol. Gen. Genet., 1986, 205: 476-482).

*Expressivity of some quantitative traits in lines with high and low frequency of reversion.* For evaluation of the effect of disruptive selection for WER frequency on quantitative traits we studied some quantitative traits of LFWER and HFWER lines: kernel weight, volume and density; number of kernels per ear; number of leaves on the main stalk and the length of the period from emergence of seedlings to flowering of male inflorescences (Tables 4 and 5).

Kernel weight, density and volume was determined on samples of 50 variegated kernels taken from the seed remnants of the selfed ears studied in 1992-1996. Kernel volume was determined by liquid (purified petrol) displacement. For determination of the number of leaves and date of flowering, the LFWER and HFWER families were planted in 1997 in two terms with an interval of 6 days. To facilitate leaf counting the fifth and tenth leaves were marked.

Data in Table 4 show that on average gametic frequency of WER in HFWER lines was 12.6 times higher than in LFWER lines. Kernel weight and volume in lines with a high frequency of WER was more than 30% higher in comparison with lines with a low frequency. Kernel density also was higher in HFWER lines though the differences in this trait were expressed less clearly and on average were equal to only 2%. Number of kernels on the ears of LFWER and HFWER lines was approximately the same.

Table 4. Expressivity of some quantitative traits in lines with low (LFWER) and high (HFWER) frequency of WER formation.

| Year | Line | Number of ears | WER, % | WER gametic frequency, % | 50 kernel weight, g | 50 kernel volume, $cm^3$ | Kernel density, $g/cm^3$ | Number of kernels per ear |
|---|---|---|---|---|---|---|---|---|
| 1992 | LFWER | 3 | 1.09* | 0.36* | 5.19 | 4.10 | 1.262 | 263.7 |
| | HFWER | 2 | 48.10 | 16.03 | 5.53 | 4.30 | 1.284 | 278.5 |
| 1993 | LFWER | 27 | 2.22* | 0.74* | 4.89* | 3.84* | 1.269* | 220.1 |
| | HFWER | 11 | 30.19 | 10.06 | 6.71 | 5.16 | 1.302 | 256.2 |
| 1994 | LFWER | 12 | 3.39* | 1.13* | 5.28* | 4.16* | 1.271* | 220.5 |
| | HFWER | 17 | 33.41. | 11.14 | 6.77 | 5.20 | 1.303 | 220.1 |
| 1995 | LFWER | 12 | 2.95* | 0.98* | 5.19* | 4.03* | 1.290 | 192.1 |
| | HFWER | 13 | 33.29 | 11.10 | 7.20 | 5.34 | 1.298 | 177.0 |
| 1996 | LFWER | 4 | 2.52* | 0.84* | 5.64* | 4.45* | 1.268 | 113.3 |
| | HFWER | 6 | 27.41 | 9.14 | 7.17 | 5.60 | 1.281 | 134.5 |
| Total | LFWER | 58 | 2.57* | 0.86* | 5.10* | 4.00* | 1.273* | 209.3 |
| | HFWER | 49 | 32.52 | 10.84 | 6.87 | 5.29 | 1.298 | 208.7 |
| HFWER as % of LFWER | | | 1265.4 | 1260.5 | 134.7 | 132.3 | 102.0 | 99.7 |

* - significance of the differences between LFWER and HFWER lines (P(0.05)

Lines with a high frequency of WER had more leaves and were characterized by later flowering of the male inflorescences (Table 5).

It is well known that the *o2* gene significantly affects kernel weight and the mutant forms are characterized by reduced kernel weight. The effect of the *o2* gene on kernel volume is less definitive and depending on the material used can also be significant (see for example Arnold J. M. et al., Crop Sci., 1977, 17: 362-366; 421-425). Proceeding from this we can assume that different alleles of the *o2* gene, i.e. *o2-lf* and *o2-hf,* influence kernel weight and volume differently, contributing to existing differences on these traits between LFWER and HFWER lines.

Table 5. Number of leaves and length of period from emergence to flowering in lines with low (LFWER) and high (HFWER) frequency of WER formation.

| Lines | Number of plants | Number of leaves per plant | Number of days to flowering |
|---|---|---|---|
| First term planting | | | |
| LFWER | 17 | 12.9*** | 57.6* |
| HFWER | 19 | 14.0 | 58.9 |
| Second term planting | | | |
| LFWER | 69 | 12.6*** | 56.4*** |
| HFWER | 74 | 13.3 | 59.3 |

* ,*** - significance of the differences between LFWER and HFWER lines at P=0.05 and P=0.001 respectively.

Influence of *o2-lf* and *o2-hf* on other quantitative traits also can not be excluded if it is presupposed that the product of the *o2* gene, being the strong transcriptional activator of the *b-32* gene (Lohmer S. et al., EMBO J., 1991, 10:617-624), also takes part in regulation of activity of other genes. In this connection it is necessary to mention the report of Genga A. et al. (MNL, 1995, 69:102), in which it was established that both the structural zein genes and the regulatory *o2* gene are expressed not only in the endosperm but also in male inflorescences.

Besides the indirect influence of the transposable elements *Bg* and *rbg* on quantitative traits studied (via the *o2* gene), the possibility also exists of their direct involvement in the expression of these traits if the genes which participate in the formation of quantitative traits have insertions of *Bg* and *rbg* elements. In case of the changes in state of the transposable elements (e. g. as a result of selection) they will change the activity of these genes and as a result of this the expression of quantitative traits.

*About the evolutionary role of transposable elements.* Summing up the data obtained it is possible to conclude that disruptive selection for WER frequency resulted in changes of state both of the receptive allele and of the regulatory element which affected the frequency of WER formation. Disruptive selection also resulted in changes of some quantitative traits of the lines obtained, changes which at least partially can be connected to the state of transposable elements.

We can assume that analogous phemomena exist in the case of other genes (in the first place regulatory genes) controlled by different systems of transposable elements. This means that transposable elements in genomes of the organisms which constitute a certain population permit the factors exerting selection pressure on this population to cause significant and rapid changes of the genomes of the organisms. The basis of these changes is the instability of transposable elements, which determine the formation of new states of regulatory and receptor elements, i.e. their heterogeneity. The consequence of heterogeneity of transposable elements is, on the one hand, significant diversity in the expression of the genes under their control and, on the other hand, the possibility of rapid genetic changes in case a selection factor is present in the medium.

Earlier it was proposed that transposable elements are generators of genetic diversity in life-threatening stress conditions (McClintock, Science, 1984, 226: 792-801). Stress conditions lead to the activation of cryptic regulatory elements which in turn result in a high level of excision and transposition of receptor elements. The main source of genetic variability in this scenario is the changes in nucleotide sequences of coding and regulatory regions resulting from imprecise excision of receptor elements (see review of Wessler, Science, 1988, 242: 399-405).

Undoubtedly stress conditions enhance genetic variability of organisms. However, available data show that the formation of new states of transposable elements, as a result of their inherent instability, causes significant genetic diversity and gives sufficient material for evolutionary changes of organisms even in the absence of stress factors. The necessary condition for such changes is the presence of a selection factor affecting the traits whose expression is controlled by transposable elements. In this scenario the main source of genetic variability is the changes of transposable elements and changes in state of mutable alleles caused by the action of the transposable elements.

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**The occurrence of haploids on the second ears of parthenogenetic lines**

--Smolkina, YV, Tyrnov, VS

Methods vary for the generation of seed from parthenogenetic lines. These include hybridization, backcrosses and self-pollination. Sometimes it is necessary to have the material for embryological analysis or for culture in vitro. Generally, these techniques concern only one individual. In such cases it would be advisable to

use the second ears. The use of the second ear makes it possible to either reject the individual or accept the individual following an identification of its frequency of parthenogenesis. In performing this method, it is possible to obtain information regarding the frequency of parthenogenesis on the different ears of the same plant.

We have examined the parthenogenetic maize lines AT-1 and AT-3, described earlier (Tyrnov, MNL 71:73-74, 1997), as well as their analogies, having the cytoplasms of M, S and C type.

The parameters investigated are as follows:

1) the simultaneous pollination of the 1st and 2nd ears by the different dates of appearance in their silks - 1 and 3-4 days;

2) the pollination of the second ear 3-4 days after pollination of the first one.

The parameter was used to answer the question about stimulation or inhibition of parthenogenesis in the second ear by embryological processes of the first.

The conclusion from this research is as follows:

1) On both ears the kernels with parthenogenetic haploid embryos can form with the same frequency. The difference in frequencies of parthenogenesis is within the limits of the values as recorded before by the delay of pollination. This variation is inevitable because of the difference in flowering and timing of ear development between the first and second ear. Consequently, the frequency of parthenogenesis in the second ear can be lower. From this research, we have found that the second ear can be successfully used in breeding, taking into account that their frequency of parthenogenesis can be lower than in the first ones.

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**Colchicine-induced chromosome doubling of maternal haploids with in vitro culture**

--Nedev, T, Gadeva, P, Kraptchev, B, Kruleva, M

The possibility in maize for chromosome doubling from maternal haploid derived dry seeds was examined. Dry seeds were plated on germination medium with different colchicine concentrations and were stored for different durations. Seeds were rinsed with sterile water after treatment and transferred onto medium without colchicine to promote germination. Colchicine (Ferak, Art., No 535122) from 250 mg/l to 600 mg/l was used for chromosome doubling. A filter-sterilized aqueous colchicine solution was added to the colchicine supplemented induction medium. In Experiment 1, colchicine (250 mg/l) treatment lasted for 168 h, in Experiment 2 (600 mg/l) it lasted for 8 and 16 h. Genotypes A (Ig1IRL-93-18/8-6 h) and B (Ig1IRL-93-18/8-6 x A654) were used. The ploidy status of the obtained plants was determined by chromosome counting of germinated root tip cells. Compared to Exp. 1, Exp. 2 treatment induced a greater response. The maternal plants obtained showed diploid, tetraploid and hexaploid cells at root meristem level. Considering the total number of cells produced, high levels of colchicine (Exp. 2) were very effective. A suitable combination was: for genotype A - 600 mg/l, 16 h, 100 % doubled haploids; for genotype B - 600 mg/l, 8 h, 87.8 % doubled haploids and different variants of chimeras. It was surprising that a decrease in colchicine (Exp. 1) significantly affected the aptitude for obtaining cells with different ploidy levels - tetraploid, hexaploid and chimeras for genotype A to totally depressed development of seeds for genotype B.

In summary, the results from this study suggest that optimum colchicine treatment of maternal haploid dry seeds appeared to be genotype dependent.

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**Red Bulgarian sweet corn**

--Dankov, T, Kruleva, M, Krapchev, B, Nedev, T

In 1988 we crossed sweet corn x embryo marker created by us with the purpose of obtaining sweet corn maternal haploids. In segregation of the cross obtained we found single kernels with red colored sweet corn endosperm. In observation of generations with red kernels during 8 years we succeeded in stabilizing this character (see Figures 1 and 2). For 2 years we kept the population of red sweet corn by intracultivar pollination under ear bags.

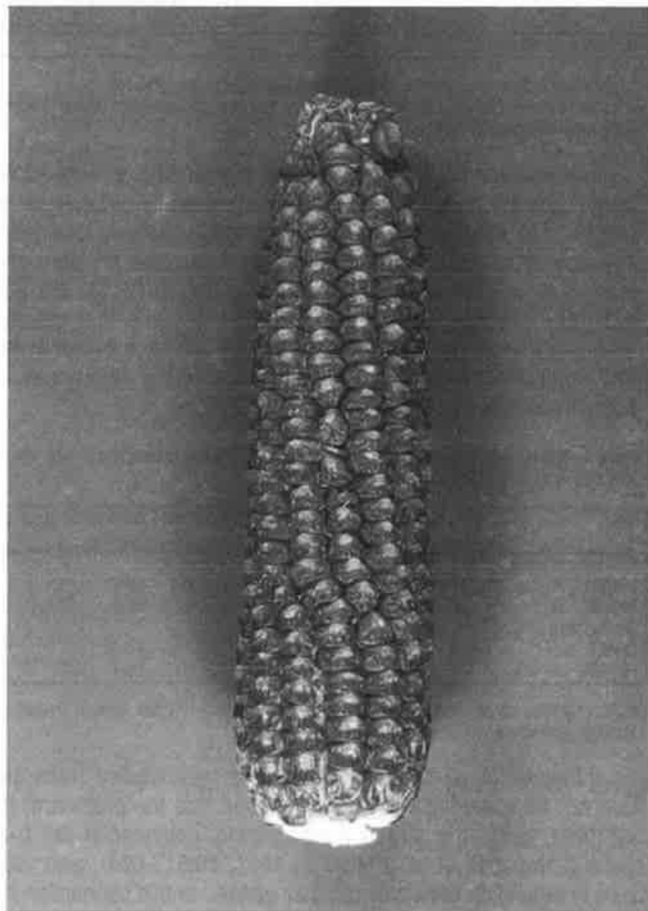

Figure 1. Ear of red Bulgarian sweet corn.

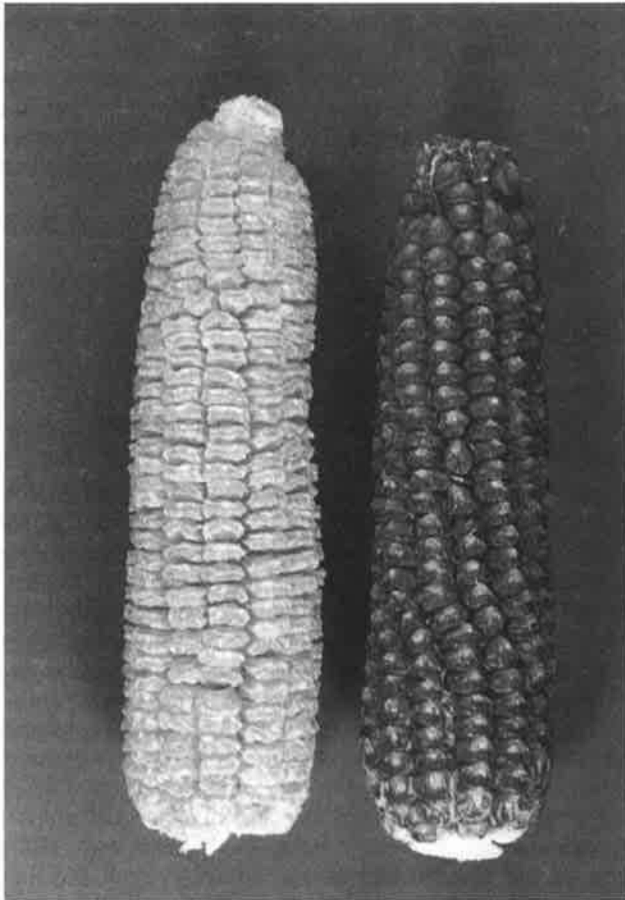

Figure 2. Ears of: left - sweet corn; right – red Bulgarian sweet corn.

Based on 25 individual plants of red sweet corn, a series of traits of agronomic importance were measured. The results obtained are given in Table 1.

Table 1. Plant and ear traits of red Bulgarian sweet corn.

| Character | Mean ± SD |
|---|---|
| Plant height (cm) | 143.4 ± 0.9 |
| Tassel length (cm) | 30.8 ± 0.6 |
| Leaf number | 9.4 ± 0.1 |
| Length of the nearest to the ear leaf (cm) | 68.8 ± 0.8 |
| Width of the nearest to the ear leaf (cm) | 6.7 ± 0.1 |
| Ear insertion height (cm) | 28.8 ± 0.9 |
| Ear length (cm) | 16.9 ± 0.4 |
| Ear diameter (cm) | 4.0 ± 0.06 |
| Number of kernel rows | 14.0 – 16.0 |
| Kernel number per row | 22.8 ± 1.1 |
| Weight of 1000 kernels (g) | 225.9 ± 5.4 |

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**Retroviral envelope gene sequences: Are they widespread in both monocots and dicots?**

--MacRae, AF, Nadimpalli, R

Infectious long terminal repeat (LTR) retrotransposable elements such as the animal retroviruses were once thought to be nonexistent in higher plants. Such infectious LTR retroelements (capable of moving from cell to cell) have now been postulated to exist in some higher plants. This postulated existence of infectious LTR retroelements is based on retroelement-mediated transduction of a maize cellular gene (Bureau, T, White, S, and Wessler, S, Cell 77: 479-480, 1994; Jin, Y-K, and Bennetzen, JL, Plant Cell 6: 1177-1186, 1994), and on the recent findings of conserved DNA and amino acid sequences similar to envelope (*env*) glycoprotein gene sequences of retroviruses as found within *Arabidopsis thaliana Tat1* retroelements (Wright, DA, and Voytas, DF, Genetics 149: 703-715, 1998), and within *Glycine max SIRE-1* retroelements (Laten, HM, Majumdar, A, and Gaucher, EA, PNAS 95: 6897-6902, 1998).

In July 1996, we obtained preliminary experimental evidence by dot blot hybridization that sequences similar to the human *HIV-1* strain ADA envelope [*env*] gene exist in 7 higher plant species (6 dicots and 1 monocot) that were tested.

Two hybridizations were performed sequentially, both on the same Zeta probe GT nylon membrane (BioRad). Approximately 1 microgram of denatured genomic DNA from the following higher plant species was applied to the membrane: *Zea mays, Brassica rapa, Arabidopsis thaliana, Nicotiana tabacum, Pisum sativum, Lupinus texensis*, and *Phaseolus lunatas*. Then approximately 50 nanograms of denatured DNA from the following control DNAs was applied to the membrane: the probe *HIV-1 env* 2.7 kb gene fragment (one positive control), and pUC 18 plasmid DNA and lambda-*Hin*dIII DNA (two negative controls).

In the first hybridization (Figure 1), the dot blot was first hybridized at 35 C in aqueous hybridization solution (Ausubel et al., Curr. Prot. Mol. Biol., 1989, Wiley & Sons) overnight, with a 2.7kb *HIV-1 env* gene *Kpn*I fragment obtained in 1996 from Dr. Lee Ratner of Washington University Medical School, St. Louis, Missouri (Westervelt, P, Gendelman, HE, and Ratner, L, PNAS 88: 3097-3101, 1991). This 2.7 kb *Kpn*I fragment, which was radioactively labeled with $^{32}P$, contains almost all of the human *HIV-1 env* gene and a small portion of the *nef* gene (L. Ratner, personal communication). After hybridization and washing, the radioactive dot blot was exposed to X-ray film for 3 days (Figure 1).

In the second hybridization (Figure 2), to ensure that the positive dot blot hybridization found in the 7 plant species above was due to hybridization to the envelope gene alone, and not to the *nef* gene, we next probed the stripped dot blot from above with a 580 bp *Bgl*II fragment isolated from within the *HIV-1* envelope gene (without any *nef* gene sequences) (Westervelt, P, Gendelman, HE, and Ratner, L, PNAS 88: 3097-3101, 1991). Once no radioactive signal was detectable on the dot blot after the previous, 2.7kb *HIV-1 env* probe hybridization, as confirmed by X-ray film exposure, we rehybridized it with the 580 bp *Bgl*II *HIV-1* envelope probe at room temperature (25 C) for 3 days in

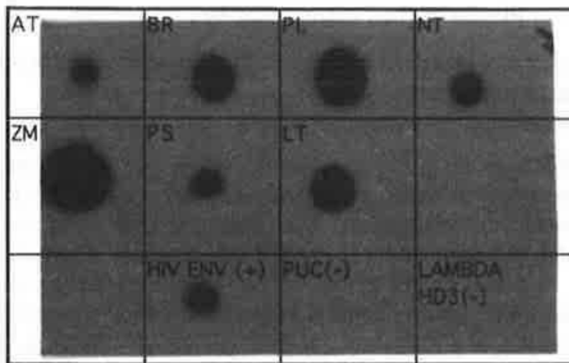


**Figure 1.** Dot blot hybridization to 7 plant species: AT=Arabidopsis thaliana; BR=Brassica rapa; PL= Phaseolus lunatas; NT= Nicotiana tabacum; ZM= Zea mays; PS=Pisum sativum; LT=Lupinus texensis. Positive control (+) is HIV-1 env fragment; two negative controls (-) are pUC and lambda HindIII DNA. The probe was a 2.7 kb KpnI HIV-1 env fragment.

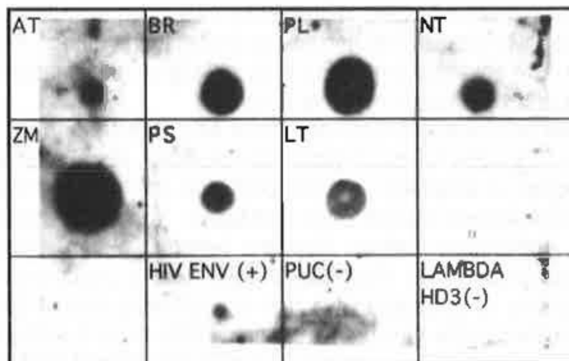


**Figure 2.** The same dot blot and control DNAs as above. Stripped and reprobed with the 580 bp BglII HIV-1 env fragment.

aqueous hybridization solution. Following hybridization and washing, 1-day and 2-day X-ray film exposures were made. The 2-day film exposure is shown in Figure 2.

Post-hybridization washes were the same for both hybridizations, as follows: 2X SSC, 0.1% SDS, 5 min., 25 C, twice; 0.2X SSC, 0.1% SDS, 5 min., 25 C, twice; 0.2X SSC, 0.1% SDS, 15 min., 42 C, twice; 0.1X SSC, 0.1% SDS, 15 min., 60 C, twice; followed by a 2X SSC rinse.

The results in terms of presence of signals resulting from hybridization were the same for both hybridizations that were performed. As shown in Figures 1 and 2, hybridization was detected in all 7 plant species, as well as in the positive control DNA. No hybridization was detected in either of the two negative control DNAs.

Our finding in 1996 as reported here, along with other findings published in 1998 (Wright, DA, and Voytas, DF, Genetics 149: 703-715, 1998; Laten, HM, Majumdar, A, and Gaucher, EA, PNAS 95: 6897-6902, 1998), encourages us to continue our research in the following directions:

1. Identification of a wider variety of plants in which these envelope-like sequences may be present.
2. Determination of whether these sequences in plants have evolved via a vertical or horizontal transmission, and/or perhaps via horizontal transfer from animal pollinators or herbivores.
3. Determination of whether these sequences are, indeed, parts of intact and fully functional retroviruses within higher plant species.
4. If such functional, infectious retroviruses do exist in plants, attempting to understand what role(s) they may play in gene transfer and in the evolution of higher plants in general.

Acknowledgements: We wish to thank Thuy Nguyen, Jessica Golby and Anne Hall for valuable laboratory assistance.

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**The structure of *teosinte branched1*: a progress report**
--Doebley, J, Stec, A

The *teosinte branched1* (*tb1*) gene has been cloned (Doebley, Stec and Hubbard, 1997, Nature 386: 485-488), but its complete structure is not known. Because some evolutionary analyses of nucleotide variation in *tb1* require a knowledge of the location of transcribed vs. non-transcribed regions, we are analyzing the structure of this gene. Here, we report our current evidence on the structure of *tb1*. This preliminary evidence was obtained by RT-PCR and by comparison of genomic and cDNA sequences.

The longest known *tb1* cDNA is 1306 bp excluding the polyA tail. On northern blots, *tb1* hybridizes to a message that is between 1.4 and 1.6 kb in length. Thus, the known 1306 bp cDNA plus a 100 bp polyA tail would be sufficient to account for the 1.4 kb message seen on northern blots. The genomic and cDNA sequences are fully colinear without any intervening sequences (introns). Thus, *tb1* may be an intronless gene and the known cDNA could be full length. One concern with this interpretation is that the genomic sequence immediately upstream of the 5' end of the cDNA lacks an obvious TATA-box. Thus, if this interpretation is correct, *tb1* would belong to a class of genes that lack a TATA-box.

Approximately 840 bp upstream of the 5' end of the cDNA, there is a short open reading frame (ORF) of about 100 bp with an adjacent TATA-box-like motif in the genomic sequence (Fig. 1). To test whether this TATA-box-like motif might be part of the *tb1* promoter, we employed RT-PCR using one primer (JD105: gaagaccaactcatctgacc) located in the 100 bp ORF and another (JD82: ccgatctggtagctgagg) within the region covered by the cDNA (Fig. 1). RT-PCR yielded a 196 bp product corresponding to the genomic sequence that flanks a 795 bp intron. The intron is bound by the conserved donor-acceptor (gt...ag) splicing sites. Thus, the *tb1* transcript can include the small 100 bp ORF and the TATA-box-like motif just upstream of it may form part of the promoter. If transcription begins about 35 bp downstream of the putative TATA-box, then the predicted length of the transcript (after removal of the intron) would be about 1304 bp plus the polyA tail. This length is long enough to account for the 1.4 to 1.6 kb message seen on northern blots. There are two difficulties with this model. (1) The first ATG, which is only 10 bp from the putative transcription start site, is not in the expected reading frame and would produce a polypeptide of only two amino acids in length. The second ATG, however, is in the expected reading frame. (2) The known cDNA clone extends into the intron, and one

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        .........|.........|.........|.........|.........|.........|.........|.........|
   1 ttctataaccgctactgcttattgtcattatgcgacttggaagacattttcttcctactgaaaggcggtctgtttttgt
                                                                                 *
  81 gttgtcgagagtgtgatgggtaaccatagttaataatgcactggatctatcactactcatacaggtcccataTGCCTAAT
 616 AATGTTGTGAAGACCAACTCATCTGACCACATCTGTCCCTACCATGCTTGTACACCACACTACATACATCACTCATCACT
 241 GGTCCTTCGTTTCGgtaccctcctcccacaatgttcaatgtatatactaatagttctcaaataaattcctgtggatgtta
 321 caaaaacccacggtctttggtttcctgaagaagtatttcatggaggcgcgcacgtccatcgtactgcgtcctgcagctat
 401 ggccgcccccatctggccaataaatgtactaggtcacttgtagccaatagcgtttcaacatgcacacagcttttcccccca
 481 atagtgcaggtccttgtattctcctccctctccctcacctcaaatctcatccacacgaacaggcggcacggcagtattcc
 561 tccacagccctcctctctataagatggcacagccctctcaggtaggggcgagtgtctcactctcacatagtaaaaaaaa
 641 aaaaacgcccccaaggttcttaagcacaattctctagctatcttggtctcctacacagcctatgcacatgagcccatgcc
 721 tctcctctccttgcgcctgcatagagaggtggtatgatcacctggaaagttttaactctctctctctctctctctct
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        +
 961 ggggccttggagtcccatcagtaaagcacatgtttcctttctgtgattcctcaagccccatggacttaccgctttaccaa
1041 caactgcagCTAAGCCCGTCTTCCCCAAAGACGGACCAATCCAGCAGCTTCTACTGCTACCCATGCTCCCCTCCCTTCGC
1121 CGCCGCCGACGCCAGCTTTCCCCTCAGCTACCAGATCGGTAGTGCCGCGGCCGCCGACGCCACCCCTCCACAAGCCGTGA
1201 TCAACTCGCCGGACCTGCCGGTGCAGGCGCTGATGGACCACGCGCCGGCGCCGGCTACAGAGCTGGGCGCCTGCGCCAGT
1281 GGTGCAGAAGGATCCGGCGCCAGCCTCGACAGGGCGGCTGCCGCGGCGAGGAAAGACCGGCACAGCAAGATATGCACCGC
1361 CGGCGGGATGAGGGACCGCCGGATGCGGCTCTCCCTTGACGTCGCGCGCAAATTCTTCGCGCTGCAGGACATGCTTGGCT
1441 TCGACAAGGCAAGCAAGACGGTACAGTGGCTCCTCAACACGTCCAAGTCCGCCATCCAGGAGATCATGGCCGACGACGCG
1521 TCTTCGGAGTGCGTGGAGGACGGCTCCAGCAGCCTCTCCGTCGACGGCAAGCACAACCCGGCAGAGCAGCTGGGAGGAGG
1601 AGGAGATCAGAAGCCCAAGGGTAATTGCCGCGGCGAGGGGAAGAAGCCGGCCAAGGCAAGTAAAGCGGCGGCCACCCCGA
1681 AGCCGCCAAGAAAATCGGCCAATAACGCACACCAGGTCCCCGACAAGGAGACGAGGGCGAAAGCGAGGGAGAGGGCGAGG
1761 GAGCGGACCAAGGAGAAGCACCGGATGCGCTGGGTAAAGCTTGCTTCAGCAATTGACGTGGAGGCGGCGGCTGCCTCGGG
1841 GCCGAGCGACAGGCCGAGCTCGAACAATTTGAGCCACCACTCATCGTTGTCCATGAACATGCCGTGTGCTGCCGCTGAAT
1921 TGGAGGAGAGGGAGAGGTGTTCATCAGCTCTCAGCAATAGATCAGCAGGTAGGATGCAAGAAATCACAGGGGCGAGCGAC
2001 GTGGTCCTGGGCTTTGGCAACGGAGGAGGAGGATACGGCGACGGCGGCGGCAACTACTACTGCCAAGAGCAATGGGAACT
2081 CGGTGGAGTCGTCTTTCAGCAGAACTCACGCTTCTACTGAACACTACGGGCGCACTAGGTACTAGAACTACTCTTTCGAC
2161 TTACATCTATCTCCTTTCCCTCAACGTGAGCTTCTCAATAATTTGCTGTCTTAATCTATGCGTGTGTTTCTCTTTCTAGA
                                                                     !
2241 CTTCGTAATTGGCTGTGTGACGATGAACTAAGTTTGGTCATCGCATGATGATGTATTATAGCTagctagcatgcactgtg
2321 gcgttgattcaataatggaattaatcggtgtcgtcgatttggtgatttccgaactgaatctctgtgatga
        .........|.........|.........|.........|.........|.........|.........|.........|
```

Figure 1. Composite nucleotide sequence of *tb1* from cDNA and genomic sequences. Putative exons (upper case), an intron (lower case), intron splicing sites (underlined), TATA box-like motif (double underlined), primers used in RT-PCR (arrows, black boxes), 5' (+) and 3' (!) ends of the cDNA, start and stop codons (gray boxes) are shown. For evolutionary analyses of nucleotide variation, the gene was partitioned into the 5' non-transcribed and transcribed regions at 35 bp downstream of the TATA-box-like motif (*).

would have to infer that this cDNA was derived from an unprocessed message.

While the exact structure of *tb1* is uncertain, the RT-PCR result establishes that the transcribed region of the gene can include the 100 bp ORF which is 35 bp downstream of the TATA-box-like motif. Thus, for the purpose of our evolutionary analysis of nucleotide variation in *tb1*, we have used the position (*) 35 bp downstream of the TATA-box-like motif as the point of division between the 5' non-transcribed region and the transcript. Additional experiments are under way to resolve the full structure of this gene.

**Identification of a recessive semi-dwarf mutation**

--Olsen, MS, Phillips, RL

We have identified a recessive semi-dwarf occurring as a spontaneous mutation in the Mo17 genetic background. These plants are approximately half as tall as normal Mo17 plants but have the same number of nodes. Semi-dwarf plants have erect leaves and tassel branches and exhibit a significant silk delay. Semi-dwarf plants were used as male parents in crosses with normal sibs as well as wild-type Mo17 and B73 plants. Additionally, four normal sibs were self-pollinated in order to progeny-test the heterozygous class. In crosses with wild-type Mo17 and B73 plants, no semi-dwarf plants were recovered among 60 F1 progeny of each cross. Two of the four self-pollinated sibs were homozygous wild-type and did not show segregation. Progeny of the remaining two self-pollinated sibs exhibited 3 wild-type : 1 semi-dwarf segregation (Table 1). Two test-crosses of semi-dwarf plants with heterozygous sibs segregated 1 wild-type : 1 semi-dwarf (Table 2).

Table 1. Segregation ratios of self-pollinated heterozygous plants.

| | Progeny classes | | | | |
|---|---|---|---|---|---|
| Plant | Wild-type | Semi-dwarf | Ratio tested | $\chi^2$ | $P$ |
| 1 | 20 | 3 | 3:1 | 1.754 | 0.185 |
| 2 | 20 | 9 | 3:1 | 0.563 | 0.453 |
| Combined | 40 | 12 | 3:1 | 0.103 | 0.748 |

Table 2. Segregation ratios of homozygous semi-dwarf x heterozygote test-crosses.

| | Progeny classes | | | | |
|---|---|---|---|---|---|
| Plant | Wild-type | Semi-dwarf | Ratio tested | $\chi^2$ | $P$ |
| 1 | 30 | 27 | 1:1 | 0.158 | 0.691 |
| 2 | 27 | 24 | 1:1 | 0.176 | 0.675 |
| Combined | 57 | 51 | 1:1 | 0.333 | 0.563 |

Although complementation tests with known semi-dwarf mutants have not been performed, both *sdw1* and *sdw2* exhibit dominant behavior (Neuffer, M.G., E.H. Coe, S.R. Wessler, Mutants of Maize, p 283, 1997).

**A chromosome 9 region containing the *dzs10* allele from B37LTI increases whole kernel methionine level in the inbred line A679**

--Olsen, MS, Graham, GI, Phillips, RL

A mutant line with increased lysine levels designated B37LTI (lysine-threonine inhibited) was developed through EMS mutagenesis of B37 seed (Muenchrath, D.A. and R.L. Phillips, Crop Sci. 33:1095-1099, 1993). The region of chromosome 9 encompassing the high-methionine 10 kD structural gene, *dzs10*, from B37LTI has been introgressed into A619, A632, A679 and A682 using RFLP markers. Within the A679 and A682 genetic backgrounds, two independent BC1 plants were used to develop heterozygous BC3 individuals. From these heterozygous BC3 plants, seven BC3S2 ears homozygous for the donor parent allele (DP) and seven BC3S2 ears homozygous for the recurrent parent allele (RP) were derived. Identical backcross strategies were utilized in the A619 and A632 genetic backgrounds; however in the A619 background, BC2S2 ears were analyzed, while in the A632 background, BC1S2 ears were used.

From each ear, approximately 50 kernels were ground to pass a 1 mm screen. Total protein was extracted from 35 mg meal with 1.7 mL of a 1% SDS, 0.0125 M Na borate, 2% β-mercaptoethanol, pH 10 solution (Wallace, J.C., M.A. Lopes, E. Paiva, and B.A. Larkins, Plant Physiol. 92:191-196, 1990). Norleucine was used as an internal standard. Following extraction, samples were centrifuged at 16000 x g for 15 min and 7.5 µL protein extract from each sample was placed in a 6 x 50 mm glass tube. Samples were dried under vacuum and 50 µL 6 N HCl was added to each tube. Thirteen sample tubes were placed together in a Waters hydrolysis vial. Hydrolysis vials were alternately evacuated and flushed with $N_2$ four times and sealed prior to hydrolysis at 150 C for 1 h. Following hydrolysis, samples were allowed to cool and then dried under vacuum.

Derivitization, separation and quantitation of amino acids followed procedures by Knect and Chang (Knect, R. and J. Chang, Anal. Chem., 58:2375-2379, 1986). Dried hydrolysate was resuspended in 32 µL 50 mM $NaHCO_3$. Thirty microliters of resuspended hydrolysate was transferred to a 12 x 32 mm HPLC vial (Chrom Tech). Derivitization was accomplished by adding 60 µL Dabsyl-Cl solution (1.3 mg Dabsyl Cl / 1 mL acetonitrile) and heating at 70 C until all precipitates had dissolved. Following derivitization, 410 µL of a 1:1 95% ethanol : 50 mM $Na_2HPO_4 \cdot 7H_2O$ pH 7.5 solution was added. Amino acid separation and quantitation were achieved using High Performance Liquid Chromatography (HPLC). All HPLC columns used were Spherisorb S5ODS2 analytical columns (Chrom Tech). Derivitized amino acids were detected at 436 nm.

Plants were considered completely random in the field. A randomized complete block design was employed during laboratory analysis to control vial-to-vial variability during acid hydrolysis of proteins prior to HPLC quantitation of amino acid levels. The randomized complete block design had seven replications with eight treatments. All four of the recurrent parents and the donor parent (B37LTI) were included in four of the replications. Analysis of variance was performed for each of the amino acids measured both as a percent of the total recovered amino acids and as a percent of the dry meal. Within each genetic background, contrasts were used to compare backcross-derived plants homozygous for the B37LTI chromosome 9 region with backcross-derived plants homozygous for the recurrent parent chromosome 9 region, recurrent parent plants, and B37LTI plants.

In the A679 genetic background, backcross-derived individuals carrying the introgressed segment (A679+DP) were significantly higher in methionine than backcross-derived A679 individuals not carrying the B37LTI chromosome 9 region (A679-DP) or A679 individuals on a percent of total recovered amino acid basis (Table 1). A679+DP ears were 49.6% higher in methionine than A679-DP ears and 43.8% higher in methionine than A679. A679+DP exhibited a significant 63.8% increase in methionine over A679-DP on a dry matter basis (Table 2). Similar increases in methionine attributed to the B37LTI chromosome 9 introgression were not observed in the other three genetic backgrounds.

A considerable change in the amino acid profile of A679 was associated with the introgression of the B37LTI chromosome 9

Table 1. Comparison of backcross-derived plants homozygous for the B37LTI chromosome 9 segment (+DP), backcross-derived plants homozygous for the recurrent parent chromosome 9 segment (-DP), recurrent parent plants (RP) and B37LTI for methionine levels on a percent of total recovered amino acids basis.

| | mg methionine/100mg total recovered amino acids | | | |
|---|---|---|---|---|
| | +DP | -DP | RP | B37LTI |
| A679 | 1.87 | 1.25** | 1.30* | 2.24 |
| A682 | 1.67 | 1.59 | 1.16 | 2.24* |
| A619 | 1.87 | 1.89 | 1.71 | 2.24 |
| A632 | 1.91 | 1.88 | 1.91 | 2.24 |

*,** Significantly different from A679+DP at the $\alpha$=0.05 and 0.01 levels respectively.

Table 2. Comparison of backcross-derived plants homozygous for the B37LTI chromosome 9 segment (+DP), backcross-derived plants homozygous for the recurrent parent chromosome 9 segment (-DP), recurrent parent plants (RP) and B37LTI for methionine levels on a dry matter basis.

| | mg methionine/g dry meal | | | |
|---|---|---|---|---|
| | +DP | -DP | RP | B37LTI |
| A679 | 1.54 | 0.94* | 1.07 | 2.14 |
| A682 | 1.58 | 1.50 | 1.21 | 2.14 |
| A619 | 1.70 | 1.66 | 1.58 | 2.14 |
| A632 | 2.05 | 1.82 | 1.91 | 2.14 |

*Significantly different from A679+DP at the $\alpha$=0.05 level.

segment. There was a relatively large and highly significant increase in methionine associated with the B37LTI chromosome 9 segment. A679 was the only genetic background in which a methionine increase was contributed by the introgressed segment. This change in methionine could be attributed to the replacement of the A679 *dzs10* allele with the B37LTI *dzs10* allele. The *dzs10* gene is a structural gene encoding a storage protein which is 23% methionine (Kirihara, J.A., J.B. Petri, and J.W. Messing., Gene 71:359-370, 1988). Interference of normal expression of this gene in A679 may contribute to the low level of methionine in this inbred line.

TAICHUNG, TAIWAN
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**Physical mapping of AFLPs and RFLPs by B-10L translocations**
--Cheng, Y-M, Lin, B-Y

Hypoploids of 34 B-10L translocations (Lin, MNL 48:182-184, 1974) were used to map seventeen AFLPs located on the proximal half of 10L (Cheng and Lin, MNL 72:77-78, 1998). The hypoploids were produced by crossing the translocations (W22) as male with B73. The position of marker loci was determined by the "uncovering" analysis of each locus on the hypoploid DNAs. Figure 1 shows the order of 17 AFLP loci relative to the breakpoints of 34 B-10L translocations. Also integrated into the map are the locations of five RFLPs analyzed in conjunction with 17 AFLPs as well as that of six morphological genes (*zn*, *du1*, *bf2*, *li*, *g1* and *r*). Thirty four B-10L translocations divide 17 AFLPs into six groups, five of which include only a single AFLP and the other one more than ten. This map differs from that of Neuffer et al. (in Mutants of maize, 1997) in that the order of *csu613* and *umc259* is reversed between the two maps, and so is the order of *csu276* and *umc259.*

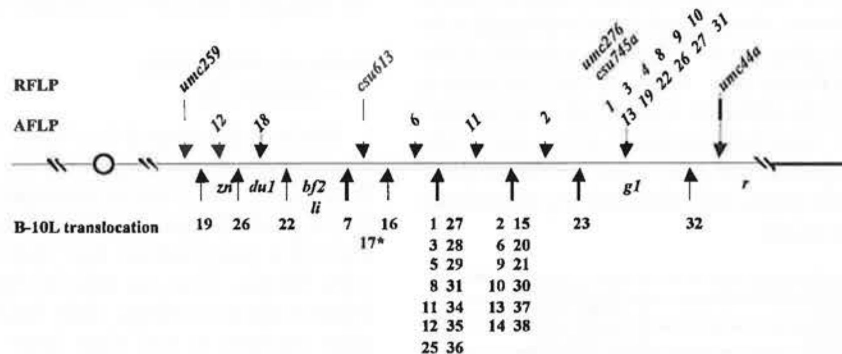


Figure 1. A map including 17 AFLPs, 5 RFLPs and 34 B-10L translocations.

URBANA, ILLINOIS
Maize Genetics Cooperation • Stock Center

**Allelism testing of green stripe stocks in Maize COOP Stock Center collection**

--Jackson, JD

This report summarizes allele testing of green stripe mutations characterized by phenotype only in the Maize COOP Stock Center collection. Some of these mutations have been found in other COOP stocks and some have been sent in by cooperators over the years. Crosses were made between plants known to be heterozygous for the *gs** and plants homozygous for *gs1* or *gs2*. In most cases plants were scored at the seedling stage and again at maturity. Proposed new designations have been assigned to those alleles for which positive results were obtained. These stocks have been increased and placed on the 1999 stocklist. It is expected that with further sorting of mutations characterized by phenotype only, additional green stripe mutants will be discovered and allele tested.

| previous designation | allelism test with *gs1* | allelism test with *gs2* | new designation |
|---|---|---|---|
| *gs*-PI228173 (68-M60)* | positive | negative | *gs1-PI228173* |
| *gs*-PI 262495* | positive | negative | *gs1-PI 262495* |
| *gs*-0229* | negative | positive | *gs2-0229* |

**Allelism testing of lazy stocks in Maize COOP Stock Center collection**

--Jackson, JD

This report summarizes allele testing of lazy mutations characterized by phenotype only in the Maize COOP Stock Center collection. Some of these mutations have been found in other COOP stocks and some have been sent in by cooperators over the years. Crosses were made between plants known to be heterozygous for the *la** and plants homozygous for *la1*. Plants were scored at maturity in both Illinois and Puerto Rico. Proposed new designations have been assigned to those alleles for which positive results were obtained. These stocks have been increased and placed on the 1999 stocklist. It is expected that with further sorting of mutations characterized by phenotype only, additional lazy mutants will be discovered and allele tested.

| previous designation | allelism test with *la1* | new designation |
|---|---|---|
| *la*-PI1842843* | positive | *la1-PI1842843* |
| *la*-Funks 1087* | positive | *la1-Funk:1087* |
| *la*-Funks 2232* | positive | *la1-Funk:2232* |
| *la*-N2276B* | positive | *la1-N2276B* |

**Recovery of dormant *vp9* allele**

--Jackson, JD

"Botany 100" is a trait I brought from Dr. John Laughnan's lab at the University of Illinois. It is a stock in W23 nuclear background that is recessive for a pale yellow endosperm that gives albino seedlings. He used it in the Botany 100 labs for showing 3:1 Mendelian ratios in seedlings. By planting given numbers of yellow and pale seeds you can easily show a 3:1 ratio of green to albino seedlings. This stock is very useful for teaching and we have supplied it to several people for such purposes. I wanted to further characterize this trait, so it could be properly added to the Stock Center's collection.

Dr. Laughnan's stock traces back to an albino trait in (EP) cytoplasm that he received from Jerry Kermicle in 1973. Also in 1973 Kermicle and Lonquist described a recessive white endosperm trait linked to the *Rcm* gene (Kermicle J. L. and J.H. Lonquist, MNL 47: 209-211, 1973). Later, Jim Allen stated that the white endosperm trait is allelic to *vp9* (D. Robertson, personal communication) and went on to show linkage of *Rcm1* with *vp9* on the short arm of chromosome 7 (Allen,J.O. et al, Maydica 34:277-290, 1989). Previously Don Robertson had reported on a *y7-Wisconsin #2* that turned out to be an allele of *vp9* (Robertson, D., J. Hered. 66:67-74, 1975). This allele seemed to consistently produce almost all dormant seeds. This sounded like the stock from Dr. Laughnan, and after reading the Maydica article I thought this might be the same trait.

We have maintained Dr. Laughnan's stock for years by self pollination and selection for ears segregating the pale endosperm trait. Unable to obtain the original sources or their pedigrees, allelism crosses were made between "Botany 100" and the COOP's *vp9-R* and *vp5*. "Botany 100" heterozygotes were crossed as females by plants that were also self-pollinated to confirm the presence of *vp5* or *vp9-R*. Five such crosses with *vp5* were negative. However, seven crosses with *vp9-R* confirmed that the "Botany 100" stock we now maintain is allelic to *vp9-R*. The allele now called *vp9-Bot100* has been added to the Stock Center's collection and is available for teaching and research purposes.

The *vp9-Bot100* is a dormant allele. On *vp9-R* self-pollinated ears the pales are viviparous. In comparison, the pale kernels on the *vp9-Bot100* stock are never viviparous and the ears of *vp9-Bot 100* crossed by *vp9-R* are predominantly dormant with only rare viviparous kernels. The pale kernels on *vp9-R* and *vp9-Bot 100* have a greenish almost fluorescent cast to them. Crosses were done to determine if *Bn1* was present in the stock to give the greenish color . Tests were negative in that the greenish color did not disappear and seems to be associated with the trait.

**Recovery of *lw1-6474***

--Jackson, JD

While sorting through the Stock Center collection of mutants characterized by phenotype only, an albino (*w*-6474*) was found that is tightly linked to pale endosperm color on ears segregating 3:1 for yellow vs. pale. The Stock Center has also been maintaining a *w*-6474* (stock number 124L) that had been placed to 1L by a 1976 TB test. This one also segregates for pale yellow kernels linked to albino seedlings. Both stocks have been allele tested to other mutants in this class (pale yellow endosperm, albino seedlings) with positive results for *lw1*. The *lw1-6474* allele has been given stock number 118CD.

Don Robertson had previously reported a *lw1-6474* allele (Robertson, J. Hered. 66:67-74, 1975). This is probably the same allele. The COOP's source was E. G. Anderson 1967-2111-3 ⊗.

**Additional 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 to-

gether 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. Below is a summary of additional translocation stocks we have completed testing.

**Table 1. *Wx1* T2-9b F1** (2S.18; 9L.22)

**A)** The F1 source showed linkage of *wx1* with *v4*:

2 point linkage data for *v4-Wx1* T2-9b
Testcross: [ *V4 Wx1* T2-9b x *v4 wx1* N] x *v4 wx1* N

source:87-966 x 964 F1 of ^M14 x ^W23

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | + Wx | 1629 | |
| | v wx | 1342 | 2971 |
| 1 | v Wx | 205 | |
| | + wx | 175 | 380 |

% recombination *v4-Wx1* = 11.3±.5

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**Table 2. *Wx1* T5-9d** (5L.14; 9L.10)

**A)** The M14 sources showed linkage of *wx1* with *gl8*:

2 point linkage data for *gl8-Wx1* T5-9d
Testcross: [ *Gl8 Wx1* T5-9d x *gl8 wx1* N] x *gl8 wx1* N

source:93W-1449 ^M14

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | + Wx | 58 | |
| | gl wx | 42 | 100 |
| 1 | gl Wx | 15 | |
| | + wx | 24 | 39 |

% recombination *gl8-Wx1* = 28.1±3.8

source:94-1867-1 ^M14

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | + Wx | 497 | |
| | gl wx | 492 | 989 |
| 1 | gl Wx | 129 | |
| | + wx | 131 | 260 |

% recombination *gl8-Wx1* = 20.8±1.1

source:94-1869-1 ^M14

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | + Wx | 1332 | |
| | gl wx | 1282 | 2614 |
| 1 | gl Wx | 331 | |
| | + wx | 272 | 603 |

% recombination *gl8-Wx1* = 18.7±.7

**B)** The W23 sources showed no linkage of *wx1* with *gl8*.

---

**Table 3. *Wx1* T7-9(4363)** (7ctr.; 9ctr.)

**A)** Some M14 sources showed linkage of *wx1* with *gl1*:

2 point linkage data for *gl1-Wx1* T7-9(4363)
Testcross: [ *gl1 Wx1* T7-9(4363) x *Gl1 wx1* N] x *gl1 wx1* N

source:94-1874 ^M14

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | gl Wx | 98 | |
| | + wx | 101 | 199 |
| 1 | + Wx | 13 | |
| | gl wx | 8 | 21 |

% recombination *gl1-Wx1*=9.5±2.0

source:95-889-2 ^M14

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | gl Wx | 931 | |
| | + wx | 942 | 1873 |
| 1 | + Wx | 49 | |
| | gl wx | 35 | 84 |

% recombination *gl1-Wx1*=4.3±.5

source:76-2062-1 ^M14

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | gl Wx | 428 | |
| | + wx | 434 | 862 |
| 1 | + Wx | 36 | |
| | gl wx | 12 | 48 |

% recombination *gl1-Wx1*=5.3±.7

**B)** Some W23 sources showed linkage of *wx1* with *gl1*:

2 point linkage data for *gl1-Wx1* T7-9(4363)
Testcross: [ *gl1 Wx1* T7-9(4363) x *Gl1 wx1* N] x *gl1 wx1* N

source:87-1029 ^W23

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | gl Wx | 663 | |
| | + wx | 731 | 1394 |
| 1 | + Wx | 19 | |
| | gl wx | 12 | 31 |

% recombination *gl1-Wx1*=2.2±.4

source:94-1876 ^W23

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | gl Wx | 612 | |
| | + wx | 622 | 1234 |
| 1 | + Wx | 38 | |
| | gl wx | 18 | 56 |

% recombination *gl1-Wx1*=4.3±.6

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**Table 4. *Wx1* T9-10(8630)** (9S.28; 10L.37)

**A)** Unconverted source showed linkage of *wx1* with *r1*:

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:72-3342-2

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | r Wx | 935 | |
| | + wx | 850 | 1785 |
| 1 | r wx | 120 | |
| | + Wx | 174 | 294 |

% recombination *r1- Wx1*=14.1 ±.8

**B)** Additional W23 sources showed linkage of *wx1* with *r1*:

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:87-1077 ^W23

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | r Wx | 1445 | |
| | + wx | 1085 | 2530 |
| 1 | r wx | 172 | |
| | + Wx | 250 | 422 |

% recombination *r1- Wx1*=14.3±.6

source:93W-1459-5 ^W23

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | r Wx | 1052 | |
| | + wx | 736 | 1788 |
| 1 | r wx | 117 | |
| | + Wx | 192 | 309 |

% recombination *r1- Wx1*=14.7±.8

source:93W-1459-6 ^W23

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | r Wx | 1213 | |
| | + wx | 910 | 2123 |
| 1 | r wx | 136 | |
| | + Wx | 248 | 384 |

% recombination *r1*- *Wx1*=15.3±.7

## Additional 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 additional translocation stocks we have completed testing. Additional pedigree information on bad sources is available should anyone want to check on sources supplied to them previously by the Stock Center.

**Table 1. *wx1* T2-9b** (2S.18; 9L.22)

**A)** The M14 source showed no linkage of *wx1* with *v4*.

**B)** The W23 source showed linkage of *wx1* with *v4:*

2 point linkage data for *v4-wx1* T2-9b
Testcross: [*V4 wx1* T2-9b x *v4 Wx1* N] x *v4 wx1* N

source:82-082-2 ^W23

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | v Wx | 1087 | |
| | + wx | 994 | 2081 |
| 1 | + Wx | 225 | |
| | v wx | 141 | 366 |

% recombination *v4-Wx1*=15.0±.7

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**Table 2. *wx1* T5-9a** (5L.69; 9S.17)

**A)** The M14 source showed linkage of *wx1* with *v2:*

2 point linkage data for *v2-wx1* T5-9a
Testcross: [*V2 wx1* T5-9a x *v2 Wx1* N] x *v2 wx1* N

source:92H-475-2 ^M14

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | v Wx | 501 | |
| | + wx | 571 | 1072 |
| 1 | + Wx | 57 | |
| | v wx | 48 | 105 |

% recombination *v2-Wx1*=8.9±.8

**B)** The W23 source showed linkage of *wx1* with *v2:*

2 point linkage data for *v2-wx1* T5-9a
Testcross: [*V2 wx1* T5-9a x *v2 Wx1* N] x *v2 wx1* N

source:92H-474-2 ^W23

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | v Wx | 578 | |
| | + wx | 677 | 1255 |
| 1 | + Wx | 57 | |
| | v wx | 45 | 102 |

% recombination *v2-Wx1*=7.5±.7

---

**Table 3. *wx1* T6-9b** (6L.10; 9S.37)

**A)** The M14 sources showed linkage of *wx1* with *y1:*

2 point linkage data for *y1-wx1* T6-9b
Testcross: [*Y1 wx1* T6-9b x *y1 Wx1* N] x *y1 wx1* N

source:88-976 x 977 ^M14

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | y Wx | 2160 | |
| | + wx | 1219 | 3379 |
| 1 | + Wx | 77 | |
| | y wx | 43 | 120 |

% recombination *y1-Wx1*=3.4±.3

2 point linkage data for *y1-wx1* T6-9b
Testcross: [*y1 wx1* T6-9b x *Y1 Wx1* N] x *y1 wx1* N

source:87-1711-3 ^M14

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | + Wx | 1755 | |
| | y wx | 1181 | 2936 |
| 1 | y Wx | 25 | |
| | + wx | 19 | 44 |

% recombination *y1-Wx1*=1.5±.2

**B)** The W23 sources showed linkage of *wx1* with *y1:*

2 point linkage data for *y1-wx1* T6-9b
Testcross: [*y1 wx1* T6-9b x *Y1 Wx1* N] x *y1 wx1* N

source: 94-1907-1^W23

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | + Wx | 1403 | |
| | y wx | 1093 | 2496 |
| 1 | y Wx | 29 | |
| | + wx | 29 | 58 |

% recombination *y1-Wx1*=2.3±.3

source:97-1275-1;-2;-4 ^W23

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | + Wx | 993 | |
| | y wx | 843 | 1836 |
| 1 | y Wx | 42 | |
| | + wx | 50 | 92 |

% recombination *y1-Wx1*=4.8±.5

source:88-972-974 Bulk 1 ^W23

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | + Wx | 1400 | |
| | y wx | 881 | 2281 |
| 1 | y Wx | 36 | |
| | + wx | 44 | 80 |

% recombination *y1-Wx1*=3.4±.4

---

**Table 4. *wx1* T7-9(4363)** (7ctr.; 9ctr.)

**A)** The M14 sources showed no linkage of *wx1* with *gl1*.

**B)** The W23 sources showed linkage of *wx1* with *gl1:*

2 point linkage data for *gl1-wx1* T7-9(4363)
Testcross: [*gl1 wx1* T7-9(4363) x *Gl1 Wx1* N] x *gl1 wx1* N

source:88-980 x 979 ^W23

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | + Wx | 1737 | |
| | gl wx | 1780 | 3517 |
| 1 | gl Wx | 150 | |
| | + wx | 155 | 305 |

% recombination *gl1-Wx1*=8.0±.4

source:93W-1416 ^W23

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | + Wx | 450 | |
| | gl wx | 442 | 892 |
| 1 | gl Wx | 28 | |
| | + wx | 29 | 57 |

% recombination *gl1-Wx1*=6.0±.8

---

**Table 5. *wx1* T7-9a** (7L.63.; 9S.07)

**A)** The M14 source showed linkage of *wx1* with *gl1*:

2 point linkage data for *gl1-wx1* T7-9a
Testcross: [*gl1 wx1* T7-9a x *Gl1 Wx1* N] x *gl1 wx1* N

source:92-429-2 ^M14

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | gl Wx | 832 | |
| | + wx | 768 | 1600 |
| 1 | + Wx | 127 | |
| | gl wx | 135 | 262 |

% recombination *gl1-Wx1*=14.1±.8

**B)** The W23 source showed linkage of *wx1* with *gl1*:

2 point linkage data for *gl1-wx1* T7-9a
Testcross: [*gl1 wx1* T7-9a x *Gl1 Wx1* N] x *gl1 wx1* N

source:92H-482-1 ^W23

| Region | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | gl Wx | 1120 | |
| | + wx | 1188 | 2308 |
| 1 | + Wx | 245 | |
| | gl wx | 203 | 448 |

% recombination *gl1-Wx1*=16.3±.7

## Allelism testing of zebra necrotic stocks in Maize COOP Stock Center collection

--Jackson, JD

This report summarizes allele testing of stocks of zebra necrotic mutations characterized by phenotype only in the Maize COOP Stock Center collection. Some of these mutations have been found in other COOP stocks and some have been sent in by cooperators over the years. In most cases crosses were made between known heterozygotes and homozygous *zn2* plants. Plants were scored at the seedling stage and again at maturity. Proposed new designations have been assigned to these alleles. These stocks have been increased and placed on the 1999 stocklist. During the screening of green stripe mutants, one culture was observed to have more of a zebra necrotic phenotype and upon testing was determined to be allelic to *zn2*. It is expected that with further sorting of mutations characterized by phenotype only, additional zebra necrotic mutants will be discovered and allele tested.

| previous designation | allelism test with *zn2* | allelism test with *gs1* | new designation |
|---|---|---|---|
| *zn*-PI251887 (8-m168-10)* | positive | negative | *zn2-PI251887* |
| *zn*-PI236997 (8-m87-2)* | positive | negative | *zn2-PI236997* |
| *zn*-PI239110 (m110)* | positive | negative | *zn2-PI239110* |
| *gs*-56-3012-10* | positive | negative | *zn2-56-3012-10* |

## *bm3* is uncovered by TB-4Sa

--Stinard, PS

In last year's MNL (MNL72:79), we reported the results of a 3-point linkage test for *su1*, *bm3*, and *gl7* on 4S. This data indicated that the correct order of the genes on chromosome 4 is *su1-bm3-gl7* (this is the local order--the global order can not be determined until the global order of *su1* and *bm3* are known). Since both *su1* and *gl7* are uncovered by TB-4Sa, it follows that *bm3*, which is located between these two loci, should also be uncovered by TB-4Sa. However, the only previously published data about the chromosome location of *bm3* (Beckett, JB, MNL 49:130-134, 1975) indicated that *bm3* is not uncovered by TB-4Sa. Last winter, we repeated this test, crossing TB-4Sa onto two separate *su1 bm3* stocks maintained by the Coop. This summer, we observed the progeny of these crosses, and noted that 18 out of 50 plants grown from these crosses had brown midribs. The TB-4Sa stock itself does not carry *bm3*. We conclude that *bm3* is located distal to the 4S breakpoint of TB-4Sa.

## Three-point linkage data for *gl5 fl2 su1* on 4S

--Stinard, PS

The results of a three-point linkage test for *gl5*, *fl2*, and *su1* 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 self-pollinated, and the self-pollinated ears were scored for the presence of *fl2* and *su1*. Kernel samples from each self-pollinated ear were planted in the sand bench, and seedlings grown from these kernels were scored for *gl5 gl20* (*gl20* was included in the cross because only the double mutant *gl5 gl20* expresses glossy seedlings.) The following linkage relationship was established: *gl5* - 2.8 - *fl2* - 7.6 - *su1*. These data are consistent with the *fl2* - *su1* distance (8 cM) given on the most recent genetic map of chromosome 4.

Table 1. Three-point linkage data for *gl5* - *fl2* - *su1*.

Testcross: ( *Gl5 fl2 Su1 Gl20* / *gl5 Fl2 su1 Gl20*) X *Gl5 Fl2 Su1 gl20*.

| Reg. | Phenotype | No. | Totals |
|---|---|---|---|
| 0 | + fl2 + | 168 | |
| | gl5 + su1 | 149 | 317 |
| 1 | + + su1 | 5 | |
| | gl5 fl2 + | 5 | 10 |
| 2 | + fl2 su1 | 13 | |
| | gl5 + + | 14 | 27 |
| 1+2 | + + + | 0 | |
| | gl5 fl2 su1 | 0 | 0 |

% recombination *gl5--fl2* = 2.8 +/- 0.9
% recombination *fl2--su1* = 7.6 +/- 1.4
% recombination *gl5--su1* = 10.5 +/- 1.6

## Three-point linkage data for *inr1 g1 r1* on 10L

--Stinard, PS

We report here the results of linkage tests for the locus *inr1*, which we had previously shown to be on 10L (MNL 71:84). In our previous report, the *Inr1* allele was known as *da1* since it was recovered from the Coop's *da1* stocks. However, since *da1* was originally reported to be on chromosome 9 (Emerson, Beadle, and Fraser, Cornell Univ Agric Exp Stn Mem 180, 1935), and was reported to behave in a recessive manner (Eyster, WH, J Hered 22:225, 1931), and since the locus that we are working with is on 10L and acts as a dominant inhibitor of certain *R1* alleles (manuscript in preparation), we have named the locus *inhibitor of R1* (*inr1*), and the dominant inhibitor allele *Inr1*. Whether *Inr1* is identical to *da1* may never be known for certain. We are continuing

to try to recover the recessive dilute aleurone mutant that was described by Eyster, from the Coop's *da1* stocks.

The results of a three-point linkage test for *inr1*, *g1*, and *r1* on chromosome 10 are presented in Table 1. The linkage test was set up as a series of modified backcrosses as indicated in Table 1. The kernels from the first cross (*inr1 g1 R1-g / Inr1 G1 R1-S*) were all purple since *R1-g* is not inhibited by *Inr1*. Kernels from the second cross [(*inr1 g1 R1-g / Inr1 G1 R1-S*) X *inr1 g1 r1-g*] segregated for full purple kernels and pale kernels in an approximate ratio of 6:4. Full purple kernels from this cross were grown in our summer nursery, scored for *g1*, and crossed as males onto an *Inr1 G1 R1-S* tester in order to evaluate which alleles were present at the *R1* locus, and were crossed as females by *inr1 G1 R1-S* to evaluate whether *Inr1* was present. Pale purple kernels were grown, scored for *g1*, and crossed as females by *inr1 G1 R1-S* to confirm that *Inr1* was present.

The following linkage relationship was established: *inr1* - 12.1 - *g1* - 12.1 - *r1*. These data are consistent with the *g1* - *r1* distance (14 cM) given on the most recent genetic map of chromosome 10. These data place *inr1* about 12 centimorgans proximal to *g1* on the long arm of chromosome 10.

Table 1. Three-point linkage data for *inr1* - *g1* - *r1*.

Definitions: *Inr1* = dominant inhibitor of *R1* allele.
*inr1* = non-inhibitor allele.
*R1-S* = *R1* allele susceptible to inhibition by *Inr1* (e. g. *R1-d::Catspaw*).
*R1-g* = normal *R1-g* allele, not susceptible to inhibition by *Inr1*.
*r1-g* = recessive colorless aleurone *r1-g* allele. Is colorless regardless of the allele at the inr1 locus.

Testcrosses: [(*inr1 g1 R1-g / Inr1 G1 R1-S*) X *inr1 g1 r1-g*] X *inr1 G1 R1-S* and *Inr1 G1 R1-S* X [(*inr1 g1 R1-g / Inr1 G1 R1-S*) X *inr1 g1 r1-g*].

| Reg. | Genotype | No. | Totals |
|---|---|---|---|
| 0 | *Inr1 G1 R1-S* | 43 | |
| | *inr1 g1 R1-g* | 39 | 82 |
| 1 | *Inr1 g1 R1-g* | 7 | |
| | *inr1 G1 R1-S* | 5 | 12 |
| 2 | *Inr1 G1 R1-g* | 8 | |
| | *inr1 g1 R1-S* | 4 | 12 |
| 1+2 | *Inr1 g1 R1-S* | 0 | |
| | *inr1 G1 R1-g* | 1 | 1 |

% recombination *inr1--g1* = 12.1 +/- 3.2
% recombination *g1--r1* = 12.1 +/- 3.2
% recombination *inr1--r1* = 24.3 +/- 4.1

## Miscellaneous allelism tests

--Stinard, PS

Over the past few years, we have conducted allelism tests between mutants with similar phenotype, especially if they have been found to have the same chromosome location. We report here the results of four such positive allelism tests:

Notes written on old envelopes of seeds of the mutant *opaque9* (*o9*) indicated that this mutant might be allelic to *shrunken4* (*sh4*). Crosses made between *o9* and *sh4* confirmed this result. We suggest that *o9* be renamed *sh4-o9*.

Richard Whalen reported to us the similarity in phenotype between *white sheath3* (*ws3*) and *virescent26* (*v26*), both located on chromosome 2, and both producing virescent seedlings that mature to plants with white leaf sheaths. We conducted allelism tests between the *v26* allele *v26-N453A* and the *ws3* reference allele, and found them to be allelic. We propose that *v26-N453A* be renamed *ws3-N453A*, and that the other *v26* alleles be renamed as *ws3* alleles in a corresponding manner.

A couple of years ago, we noted that the *light yellow endosperm3* (*lty3*) mutant of Dollinger produces plants with a weak albescent phenotype. We placed *lty3* in our TB mapping block this past summer, and at the same time set up allelism test crosses with *albescent1* (*al1*). We found that *lty3* is uncovered by TB-3La-2S(6270), and is indeed allelic to *al1*, which is located on 2S. We propose that *lty3* be renamed *al1-lty3*.

A small kernel, white endosperm mutant producing green seedlings was found segregating in a stock of *pitted1-Mu1568*, a Mutator-induced defective kernel mutant. This new mutant was named *y*-129E*. Subsequent allelism tests proved it to be allelic to *y1*. The mutant is now designated *y1-129E*. Tests are in progress to determine whether the small kernel phenotype is part of the *y1* phenotype, or whether it is due to a tightly linked small kernel mutation.

## Results of TB tests of unplaced mutants

--Stinard, PS, Jackson, JD

Last year, we reported the results of TB mapping crosses made to symbolized unplaced mutants in the Coop's collection (MNL72:79). This year, we repeated the arm-locating cross for all of the mutants that gave positive TB tests last year, and made full TB mapping crosses on additional unplaced mutants. The mutants for which we obtained positive results are 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.

The placement of *l4* to 7S, as reported last year, turned out to be incorrect. Out of seven additional crosses of *l4* by TB-7Sc made this year, none segregated for luteus seedlings.

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 |
|---|---|---|---|---|
| *al*-JRL* | TB-9Lc | 2/2 | *ar1, v1, v30* | |
| *l3* | TB-6Lc | 16/19 | *l10, l12, l15* | 1 |
| *les*-3F-3330* | TB-5Sc | 2/2 | | |
| *lty3* | TB-3La-2S(6270) | 2/3 | *al1* | 2, 4 |
| *oro2* | TB-1Sb-2L(4464) | 8/13 | | 1, 5 |
| *pb4* | TB-5La | 9/9 | *gm1, ppg1* | 1 |
| *v*-PI267226* | TB-5La | 2/2 | *v2, v3, v12, yg1* | |
| *v13* | TB-5Sc | 10/10 | | 1 |
| *vp10* | TB-10L(19) | 11/17 | *vp13* | 1 |
| *vp12* | TB-5La | 7/14 | *lw2* | 1, 3 |
| *y11* | TB-1Sb-2L(4464) | 2/2 | *w3* | 5 |

1. A repeat and confirmation of tests reported in MNL 72.
2. Found to be allelic to *al1*, as reported elsewhere in this MNL.
3. Found to be allelic to *lw2*, as reported elsewhere in this MNL.
4. This mutant was not found to be uncovered by TB-3La.
5. These mutants were not found to be uncovered by TB-1Sb.

## *vp12* is allelic to *lw2*

--Stinard, PS

Last year (MNL72:97), we reported that the class I viviparous mutant *vp12* (pale yellow or white [depending on the genotype at the *bn1* locus] endosperm, viviparous embryo, albino seedlings) is uncovered by TB-5La, and therefore appears to be located on the long arm of chromosome 5. We repeated this TB test last winter, and conducted allelism tests between *vp12* and the only other

known class I mutant on 5L, *lw2* (pale yellow or white endosperm, dormant embryo, albino seedlings). Both the TB crosses and the allelism tests gave positive results, confirming that *vp12* is on 5L and indicating that *vp12* is an allele of *lw2*. The mutant kernels from the allelism test crosses were pale yellow, dormant, and gave rise to albino seedlings. *lw2* had not been previously known to have viviparous alleles, but the fact that a class I viviparous mutant should be found to be allelic to a dormant class I mutant is not surprising, since other class I viviparous loci (e. g. *w3*, *vp5*, and *vp9*) have dormant alleles. Since the name *lw2* has precedence, we propose that *vp12* be renamed *lw2-vp12*.

*vp12* was previously placed to chromosome 6 by Araujo et al. using *waxy1*-marked translocations (Brazilian Journal of Genetics 20:71-74, 1997). The translocation that gave the positive chromosome 6 map location, *wx1* T6-9b, is heterozygous for the white endosperm mutant *y1* (the Coop provides a vigorous hybrid between an M14 and a W23 conversion of this translocation; the M14 version is *Y1*, but the W23 version is *y1*.) We conclude that the erroneous mapping result reported in the paper of Araujo et al. is due to the tight coupling of *y1* and *wx1* in the T6-9b stock that they used for mapping.

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**Reverse maize breeding for high density populations**
--Galinat, WC

After having repeatedly taken maize apart by isolating its key traits in a teosinte background and then hybridizing the derivatives to reconstruct maize by recombination of the key genes--and all the time fighting a battle with weeds, birds, squirrels and the irrigation system, all after 60 years of corn experience, I have concluded that maybe the Europeans have the right idea with their high density monoculture of cereals. They have: no weeds, no herbicides, no irrigation, no soil erosion and almost no problems.

The wheat breeders and corn breeders developed their cereal plants and agriculture in complete isolation from each other in two different Worlds separated for thousands of years by the Atlantic Ocean until the time of Columbus. During this period they evolved different breeding procedures because their available grasses were very different after isolation along different evolutionary pathways for millions of years. They had different forms of reproduction and inheritance. The farmer-breeders on the European side of the Ocean happened to work out a system that is more environmentally friendly and better adapted to mass-production industrial agriculture than that represented by present-day maize starting on the western side of the Atlantic. However, some modern corn-breeders have been now considering reverse breeding for adaptation to high density populations, e.g. wheat, that would be both environmentally safe and make use of the huge harvesting combines designed for wheat. They would have to retrogress maize back some 5000 years to reach the same level as present-day wheat. They have already started maize in this direction by breeding for adaptation to increases in stand density. (See my item "Canopy and yield enhancement per acre with dense populations". MNL70:67, 1996.)

The reverse breeding of the new high density maize combines certain primitive plant habit traits from teosinte with other highly evolved traits of maize--its tassels, ears and kernels that adapt to hybrid seed production and economic uses. The high density maize would still carry its C4 type of photosynthesis yielding greater productivity than wheat and still carry the yellow endosperm gene from maize for the high carotene so tragically absent in all other cereals. From wheat we might transfer the gene(s) for gluten endosperm into maize to give it the elastic cohesiveness necessary for leavened dough to rise as it captures the $CO_2$ gas. This increases the quality and lightness of baked bread. This transfer of the gluten endosperm to maize is technologically possible by the gene gun techniques. This would create the new maize as a superior daily bread that becomes the hope for survival and glory of humankind.

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**Temperature and timing of heat shock gene induction in spikelets of maize**
--Bouchard, RA, Walden, DB

We have previously reported on the localization and timing of the accumulation of small heat shock gene transcripts in active meristems and immature vascular bundles of seedling radicle and plumule during heat shock induction (Greyson et al., 1996. Developmental Genetics 18:244-253) as well as the localization of developmentally modulated and heat induced accumulation in spikelets (MNL 71:87, 1997). We now report preliminary results of quantitative RNA-Dot experiments comparing the temperature and timing of heat induction in spikelets and somatic tissues.

Relative induction temperatures were examined in spikelets from the central (microspores in uninucleate stages) and lateral (microsporocytes in prophase and division stages) branches of a tassel of Ohio43, as compared with the somatic tissue of the growing leaves taken from around the same tassel. Samples were incubated for 1 hour each at 30, 33, 35, 37, 39, or 42C in a Robbins Incubator and then snap-frozen in liquid nitrogen for later RNA isolation. RNA Dot-blots were prepared with equal amounts of RNA from these samples, from control samples frozen at harvest , and from post-control samples held at ambient temperature until all incubations were concluded. Duplicate blots were probed with 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 subclone and Mhsp18-3-3, which is a gene-specific 3'-UTR region for the shsp family member with map designation *uwo10*. Additional blots were probed for RNAs representing two distinct hsp families: a gene-specific 3'-UTR fragment for *hsp82* and an *Arabadopsis* probe for the highly-conserved hsp100 family. The results with all these probes were qualitatively identical. There is little or no hsp RNA accumulation from any of the three families in leaf samples incubated below 37C, which squares with what we reported earlier for seedlings. For spikelets, however, accumulation is at a peak in the 35C samples, and is already down somewhat in the 37C samples. Figure 1 shows results for the Mhsp18-3-3 probe which illustrate the common pattern. The first row shows the signals seen on RNA from lateral spikelets, the second central spikelets, and the third leaves taken from around the tassel.

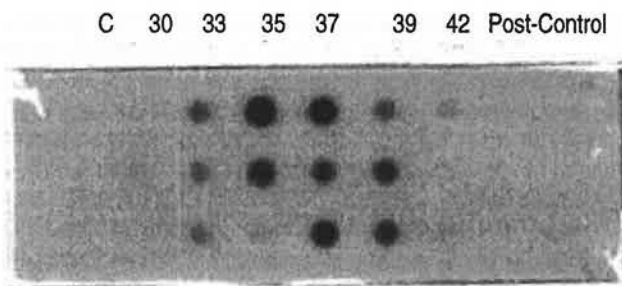


Figure 1.

We have also performed a preliminary study of the timing of shsp RNA accumulation in spikelets by freezing an initial sample of spikelets, then incubating an intact plant at heat shock temperature and freezing additional spikelet samples at intervals. In this case, we have so far probed only with the shsp18ORF and 18-3-3 probes. In both cases, the results show a very rapid response in spikelets, with substantial RNA accumulation by 30 minutes, a peak at 1 hour, and a considerable drop-off by 2 hours of heat shock. This contrasts with our earlier observations on radicles (Greyson et al, 1996), where accumulation did not peak until 2 hours. These results are illustrated by an exposure of the shspORF probing as shown in Figure 2, which displays R-Dots at full concentration (5 micrograms) on the first row and a one-fifth dilution on the second.

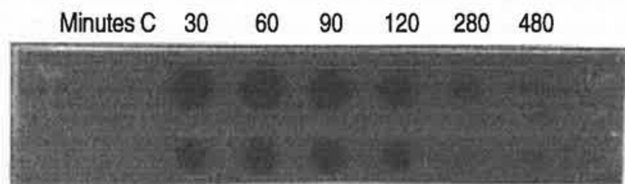


Figure 2.

Taken together, these results strongly suggest that in addition to producing some heat shock RNAs as part of their normal sequence of development, spikelets are also distinctive in responding to heat shock more rapidly and at lower temperatures than somatic tissues. The possibility for a potential role of hsps in maize male fertility thus remains a continuing focus for our ongoing research.

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**The physical location of the gene *ht1* (*Helminthosporium turcicum resistance1*) in maize (*Zea mays* L.)**

--Li, LJ; Song, YC; Yan, HM; Wang, L; Liu, LH

The disease induced by infection of *Helminthosporium turcicum* is one of the serious maize diseases and is distributed in many countries including China. It damages maize production seriously. It was reported that there are four *Helminthosporium turcicum* resistance loci, namely *ht1, ht2, ht3* and *htn1* (Zaitlin et al., 1992). The *ht1* was first identified by Hooker (1963). Recently, the genetic working map accompanying the RFLP map in maize was published (Coe, 1995). In this map, many genes including *ht1* were placed at the coordinate locations defined in the RFLP map. The gene *ht1* is 168 cM from the zero point and linked closely to *umc22* with 4.3 cM on one side and *umc122* with 1.2 cM on the other side. However, the genetic map could only show the relative locations and distances of genes or markers, but physical maps show real positions and distances of genes or markers on the chromosomes. Up to now, very little work has been reported concerning physical mapping of genes of agronomic interest in maize (Shen et al., 1987; Hong et al., 1993; Song et al., 1997; Li et al., 1997). The physical location of the gene *ht1* of maize has not been reported yet, as we know.

In situ hybridization (ISH) technique is a powerful tool to physically locate specific DNA sequences or genes directly on chromosomes. The sensitivity of in situ hybridization (ISH) techniques for humans is now down to 0.25kb (Richard et al., 1994). The ISH of small single or low copy DNA sequences in plants was more difficult than in humans. Recent developments and numerous refinements of methods including chromosome preparations and detection procedures have made it possible to detect signals of small single or low copy DNA sequences in plants (Gustafson et al., 1990; Gustafson et al., 1992; Song and Gustafson, 1995; Song et al., 1997; Li et al., 1997; Ren et al., 1997). In these studies, the signals were detected only by DAB. Recently, fluorescence in situ hybridization (FISH) has developed rapidly in plants. However, the probes used in these reports were either total genomic DNA (Mukai et al., 1993; King et al., 1994), repetitive DNAs (Griffor et al., 1991; Pederson et al., 1995; Linares et al., 1996) or large DNA fragments (Ambros et al., 1986) or BAC clones (Jiang et al., 1995). Only Dong and Quick (1994) mapped a 2.6 kb single/low copy DNA sequence on metaphase chromosomes of wheat and rye by FISH. It was demonstrated that FISH had many advantages over ISH using DAB detection method, for example, high sensitivity and higher contrast between signals and chromosomes (Jiang et al., 1994; Heiskanen et al., 1996). The FISH of DNA segments around 1 kb in size has never been reported so far in plants. The study to detect and map smaller single or low copy DNA sequences and genes by FISH is very important for physical mapping of plant genomes.

RFLP markers are very good landmarks for mapping genes. Even though the gene *ht1* has not been cloned, its physical location could be deduced by the physical position of the RFLP markers linked closely with it. In this study, we have determined the chromosome location of *ht1* by FISH of the two RFLP markers *umc22* and *umc122* linked tightly to and flanking it.

**Plant and RFLP marker materials:** Maize (*Zea mays* L.) inbred line Huang Zao 4 (Li, 1997), which derived from a native cultivar in China, was used as the tested plant material, the seeds were provided by Professor Song Jianchen, Shandong Agricultural University, Shandong Province, P.R. China. Both tested RFLP markers *umc22* and *umc122* are close to the *ht1* gene dictating reaction to *Helminthosporium turcicum* on the long arm in linkage group 2 of the classic genetic maps (Coe, 1995). They are 780 and 450 bp DNA fragment size cloned in pUC19 and kindly offered by the RFLP laboratory and probe bank of the U. S. Department of Agriculture, Agriculture Research Service, and Department of Agronomy, University of Missouri, Columbia, USA.

**Chromosome preparation:** Chromosome preparation methods were developed using the protoplast technique as described by Song et al. (1995) with some modifications. Root tips were collected from germinating seeds and treated in $\alpha$-bromonaphthalene for 2 hr at RT (room temperature), then washed in deionized water for 15 min. The root tips were fixed in freshly prepared

methanol:acetic acid (3:1) fixative overnight at 4 C, subsequently washed in deionized water for 15 min. Then the root tips were digested in 1% cellulase (Shanghai Institute of Biochemistry, Chinese Academy of Sciences) and 1% pectinase (SERVA) at 28 C for 2.5 hr. After enzymatic treatment, cells were subjected to a hypotonic treatment in water, then 2-3 root tips were squashed with forceps in 2-3 drops of fixative on a cold slide and flame dried. The prepared slides were stored at –20 C or immediately used for FISH.

**Biotin labeling DNA and in situ hybridization:** The probe was biotin-labeled with the procedure of the kit supplied by Sino-American Biotechnology Company China. After labeling reaction at 15 C for 2.5 h, labeling was stopped by adding 5 ul 0.2M EDTA (pH 8.0). Then, labeled probe was separated through a Sepharose column and evaluated by means of dot blots.

In situ hybridization was performed using the procedure described by Gustafson and Dille (1992).

**Detection:** Fluorescent signals were detected based on the procedure published by Griffor et al. (1991), Gustafson and Dille (1992) and Dong and Quick et al. (1994) with some modifications. The detection reagents came from a kit offered by Beijing Medical Academy. The procedure includes the following steps: 1) after hybridization, the coverslip was removed by dipping the slides in a 2 x SSC solution and the slides were washed in 20% formamide (in 2 x SSC) at 42 C for 10 min, 2 x SSC at 42 C for 10 min, 0.1% Triton at room temperature for 4 min and PBS at room temperature for 5 min, then the slides were drained but not dried; 2) 20 μl FITC-avidin D was added to each slide, the slides were covered with a coverslip, incubated in a humid chamber at 37 C for 30 min and the slides were washed 3 times, each for 5 min in PBS at room temperature; 3) 20 μl of biotinylated goat anti-avidin D antibody were added to the slides for 30 min at 37 C in a humid chamber, then slides were washed as above; 4) repeat step 2; 5) after washing in 2 x SSC at room temperature for 5 min and subsequently in PBS at room temperature for 5 min, 10 μl (1 μg/ml) propidium iodide in an anti-fade (10 μg/ml) solution were added to the slides, the slides were covered with a coverslip, and observed and photographed under a Zeiss fluorescence microscope.

An average of the hybridization site measurements was taken by calculating the distance from the centromere to the detection site and using that as a percentage of the arm on which the site was located. The arm ratio of the chromosome showing a detection site was also measured in order to determine on which chromosome the site was located.

Counterstained by FITC and propidium iodide (PI), the chromosomes appeared red and the signals showed yellow. The hybridization signals of both *umc22* and *umc122* were showed on 2L (the long arm of the chromosome 2) and their percentage distances from the hybridization site to the centromere were 61.78 and 63.01 respectively (Figs. 1a, c and e). The signals of *umc22* and *umc122* were also detected on 7L and the percentage distances were 47.54 and 48.85 respectively (Figs. 1b, e). In some cases the detected cells showed only one signal spot on one chromatid of one member of the detected chromosomes (Fig. 1b); but in another case, the detected cells usually showed signals on both homologous chromosomes or two sister chromatids of one member of the homologous chromosomes (Figs. 1a, c). In some detected cells, the signals from both sister chromatids were merged into a large one, just like a band (Fig. 1e). Interphase nuclei were also scored in our FISH analysis. In 100 observed interphase cells, about 60 cells showed signals. Signals were shown on 1, 2, 3, and 4 sites of different interphase nuclei respectively. Figures 1d and f showed the nuclei hybridized by *umc22* and *umc122* respectively, and each had 4 hybridization sites. The karyotypes showing the genetic and physical map of *umc22* and *umc122* were presented in Figure 2 so as to compare them.

The probes *umc22* and *umc122* both showed two different hybridization sites on 2L and 7L. It means that they are duplicated sequences. It has been demonstrated that many of the RFLP markers were duplicated at least once somewhere else in the maize genome. That maize is an allotetraploid was proved by different maize geneticists (Poggio et al., 1990; Tito et al., 1991; Molina et al. 1992). Some scientists demonstrated that the duplicated sequences of maize RFLP markers probably were relevant to properties of the allotetraploid (Weber, 1990; Dowty and Helentjaris, 1992; Song et al., 1997). It can be thought there were some homologous sequences or regions in the different original species from which maize originated even though they belonged to different genomes. Helentjaris et al. (1988) noted that the maize RFLP marker duplications were not necessarily scattered randomly over the entire chromosome, but more often appeared to cluster. Dowty and Helentjaris (1992) reported that there was also a higher order pattern to the duplication saturated regions of three different "sets" of chromosomes: 10-2-7, 5-1-9 and 3-8-6. For example, chromosome 2 shares duplications with both chromosomes 7 and 10, chromosomes 7 and 10 do not share any duplicated markers. Helentjaris (1995) further reported that the regions, 2L with 7 etc. shared extensive similarity. In this study, our results were just consistent with the above reports. The markers *umc22* and *umc122* not only share the same chromosome arms, 2L and 7L, but also have almost the same percentage distances between them (1.2 on 2L and 1.3 on 7L), even the order of these two markers and centromeres was the same on both 2L and 7L. It demonstrated that different ancestor species from which maize originated had strong conservativeness in the sequences, organization and order of markers in genomes or regions of the genomes.

The gene *ht1* (*Helminthosporium turcicum resistance1*) is linked closely to *umc22* with 4.3 cM on one side and *umc122* with 1.2 cM on the other side. Because genetic and physical maps show the same marker order, the gene *ht1* should be physically located between the hybridization sites of *umc22* and *umc122*, i.e., percentage distances 61.78 and 63.01 on 2L. Our results demonstrated that both *umc22* and *umc122* had their homologous sequences on 7L besides 2L and the order of these two markers and centromeres was the same on both 2L and 7L. Therefore, the gene *ht1* probably also had its homologous sequence between two hybridization sites of *umc22* and *umc122* on 7L. Actually, the signal spot showing the hybridization site of the marker *umc122* probably also represents the physical location of *ht1*, because each signal spot could occupy about 1-3 cM.

Our results demonstrated that FISH has a number of advantages over DAB (diaminobenzidine tetrahydrochloride) detected ISH. First, the yellow shown by the hybridization signals has high contrast with the red displayed by the chromosomes, while in the DAB detected ISH it is more difficult to distinguish the color not only between the signal and chromosomes but also between the signal spots and those contaminated by Giemsa staining. Second, for small single or low copy DNA hybridization, FISH can get higher detection rate and find more detected cells showing signals on two members or two sister chromatids of one member in the

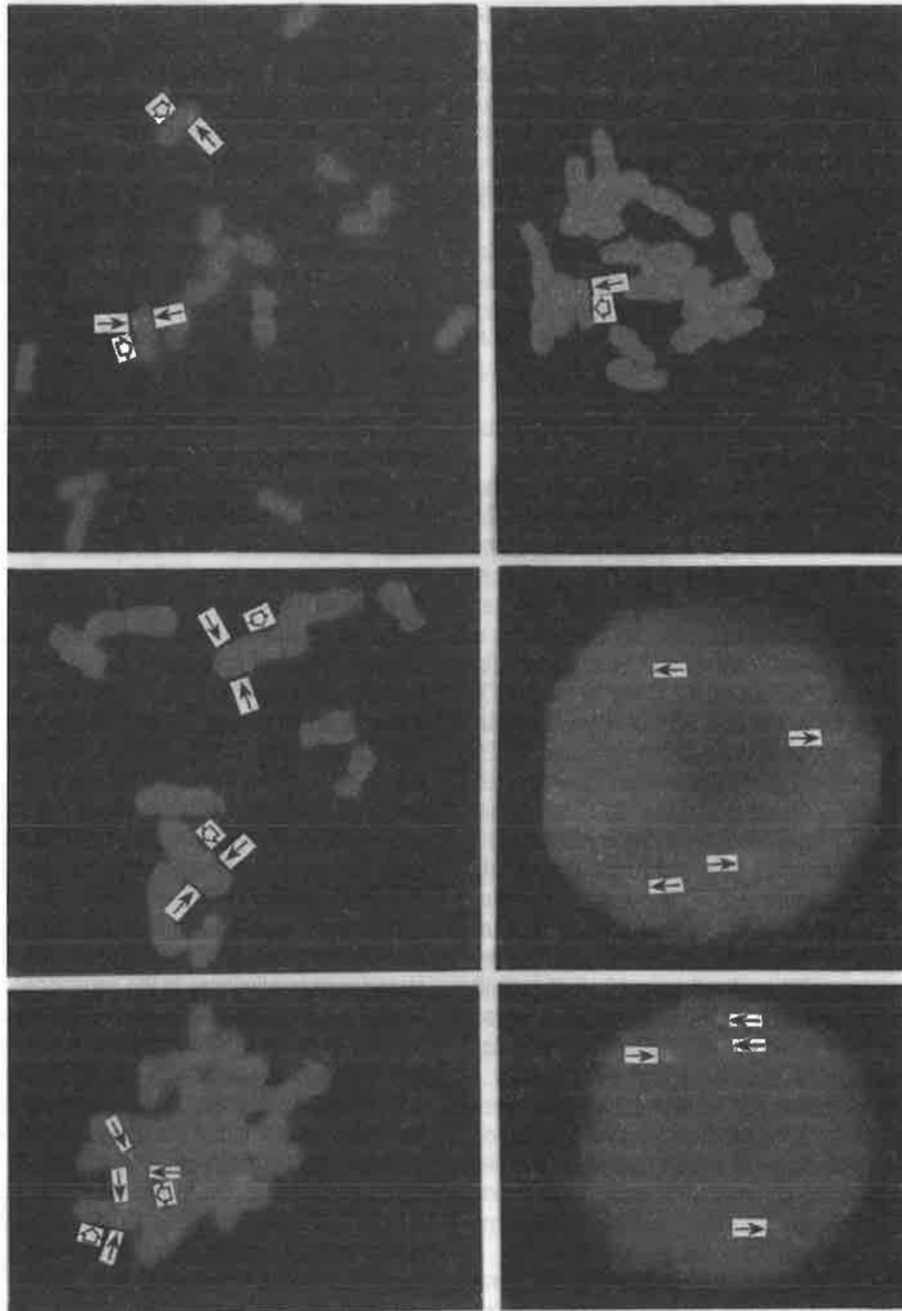

Figure 1a-f. The hybridized sites of the probes *umc22* and *umc122* on the chromosomes in maize. In all figures, the black arrows denote the hybridization signals, the white arrows denote the centromeres. a) The hybridization signal of *umc22* is in 2L. b) The hybridization signal of *umc22* is in 7L. c) The hybridization signal of *umc122* is in 2L. d) The interphase cell has four hybridization signals of *umc22*. E) The hybridization signal of *umc122* is in 2L and 7L. f) The interphase cell has four hybridization signals of *umc122*.

homologous chromosomes. In this study, their rates were about 15%, while the detection rates were only about 10%. Very few or even no cells in which the signals were detected simultaneously on two homologues or two sister chromatids could be found in DAB ISH (Gustafson et al., 1990; Gustafson et al., 1992; Song and Gustafson, 1995; Song et al., 1997; Li et al., 1997; Ren et al., 1997). Third, the markers at multiloci can be located simultaneously by multicolor FISH and it is very useful for confirming the order of closely linked genes or markers on the chromosomes. However, FISH, as a powerful tool of molecular cytogenetics, has not been reported so far for mapping of single or low copy markers around 1 kb in size in plants, not as in animals. We think the main obstacle is chromosome preparation. The protoplast chromosome preparation techniques developed by Gustafson and Dille (1992) and modified by Song and Gustafson (1995) can get rid of the cell debris hiding the chromosomes and offer more target chromosomes for combining of the probes. This will be beneficial to improving hybridization efficiency and compensating the short-

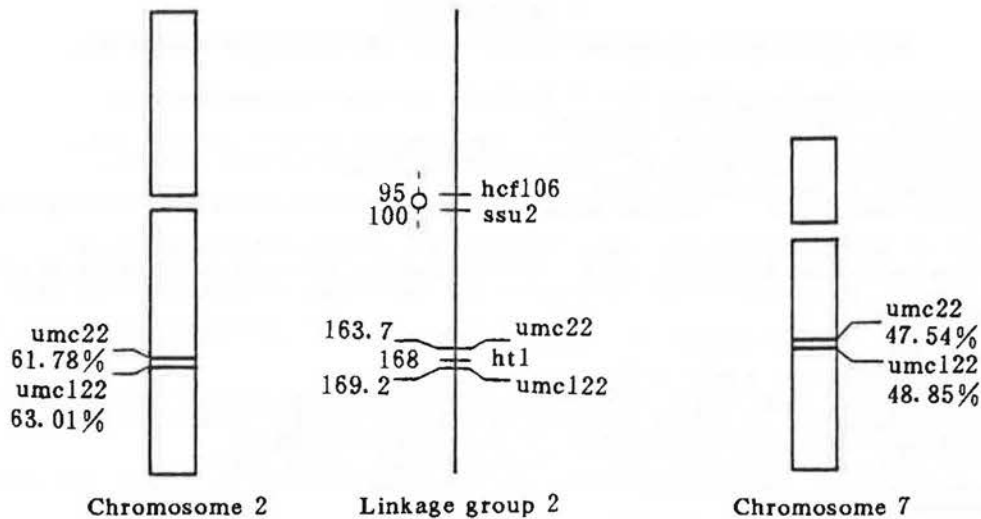


Figure 2. The idiograms of the locations of *umc22* and *umc122* markers on the chromosomes and the comparison of their sites between the chromosomes and the genetic linkage groups.

age of low detection rate for small single or low copy DNA hybridization. Therefore, one of the effective approaches to develop FISH techniques in plants is combining FISH with protoplast chromosome preparation.

In ISH studies of rice RFLP markers it was indicated that considerable variation could, and did, exist between genetic and physical maps (Gustafson and Dille, 1992; Song and Gustafson, 1995). In the genus *Triticum* it has been demonstrated that the physical distances between genes are often quite different from the genetic distances (Lukaszewski and Curtis, 1993). Heslop-Harrison (1991) thought there was often little correlation between the separation distances of markers on the genetic and physical map. However, in this study, the tested markers *umc22* and *umc122* located at the middle part of the long arm in linkage group 2 of the genetic map (Coe, 1995), according to our results are also physically located at the middle part of 2L. The genetic distance between these two markers was 5.5 cM, the percentage distance was 1.2 and the distance in both genetic and physical maps was close to each other. It demonstrates that the maize physical map is basically consistent with the genetic map for *umc22* and *umc122*. At present, the physical location of most genes or markers is still unknown. We suggest that different species and genes should not be the same for the relationship between the genetic and physical map. Although there are variations between these two types of map, the correlation between them will be displayed for more and more genes or markers as their physical mapping is developed.

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## IV. MAIZE GENETICS COOPERATION STOCK CENTER

# Maize Genetics Cooperation ◦ Stock Center

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1988 seed samples have been supplied in response to 279 requests, for 1998. Of these, a total of 63 requests were received from 20 foreign countries. Approximately three fourths of our requests were received by electronic mail or through our order form on the World-Wide Web.

With the help of Mary Polacco and Denis Hancock a new web-based query form has been developed to search for stocks of interest in our collection. This query form is available at http://www.agron.missouri.edu/cgi-bin/sybgw_mdb/mdb3/StockMGSC/query

We have also listed a new 'Phenotype Only' category of stocks. These are stocks that have been donated to the COOP over the years, and have been classified according to their mutant phenotype only. For the most part, these stocks have not as yet been allele tested, nor has their gene been located to a chromosome arm. While we expect that most of these will represent new alleles of known loci, some will represent unique, as yet undescribed loci. Over the past few years, some mutants in this class have been mapped and/or allele tested and where appropriate, the now characterized mutant stock was added to our main catalog. We are now listing all of these mutants to give cooperators that are interested in specific traits, easier access to these mutants. Many of the stocks recently donated to us by Gerry Neuffer will be placed in this class, as we grow them up.

Approximately 5 acres of nursery were grown this summer at the Crop Sciences Research & Education Center located at the University of Illinois. Despite the wet spring weather, we had good stands and obtained good increases of most stocks grown this year.

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. We have made good progress in increasing the Neuffer collection, and have almost completed our initial increase of the Robertson collection.

2. Plantings were also made from donated stocks from the collections of James Birchler (marked B-A translocation stocks), Ed Coe (various genetic stocks), Jerry Kermicle (*R1* alleles), 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 confirm the map locations of the mutants *luteus3*, *orobanche2 piebald4*, *virescent13*, *viviparous10*, and *lemon white2-vp12* with a high degree of certainty, and made a tentative placement of *y11* (see MNL article on our TB mapping results). We plan to continue this mapping project next summer.

4. We conducted allelism tests of several categories of mutants with similar phenotype or chromosome location. We found additional alleles of *albescent1*, *green stripe1*, *green stripe2*, *lazy1*, *lemon white1*, *lemon white2*, *shrunken4*, *viviparous9*, *white sheath3*, *yellow endosperm1*, and *zebra necrotic2*. In this manner, we are hoping to move stocks from our vast collection of unplaced uncharacterized mutants and integrate them into the main collection.

5. 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 are now being tested.

We continue to grow a winter nursery of 0.5 acres at the Illinois Crop Improvement Association's facilities in Juana Díaz, Puerto Rico. We had an excellent winter crop last year, and all indications are that the crop will perform well this year as well, despite the passage of Hurricane Georges over Puerto Rico. We plan to continue growing our winter nurseries at this location.

We anticipate hiring an additional Research Specialist who will be responsible for the new stocks generated by the NSF project "Maize Gene Discovery, Sequencing and Phenotypic Analysis", in which we are involved along with Virginia Walbot and others in the maize community. We anticipate that this and other projects recently funded by the NSF Plant Genome Program, will greatly enhance our collection along with greatly increasing our understanding of maize as a biological organism.

**We would like to make a special plea for cooperators to donate their genetic stocks and mutants 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. 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

**CHROMOSOME 1 MARKER**

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
104A Ts3
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
109A gs1-PI228173
109B gs1-PI262495
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
111B hcf3-N846B
111C hcf3-N1242B
111D hcf44-N1278B
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 def(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
118CB lw1-6474
118J Adh1-3F1124r53
118K Adh1-1S5657; Adh2-33
118L Adh1-3F1124::Mu3
118M Adh1-3F1124r17
118N Adh1-IL14H; 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 nec2-8147
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
125A Les2-N845A
125B Mpl1-Jenkins
125C hcf13-N1097B
125D hcf41-N1275C
125E hcf50-N1481
125F hcf2-N506C
125G hcf31-N1268B
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 f1
127F Msc1-N791A
127G Tlr1-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-N495A
129AA w18-571C
129B wlu5-N266A
129C zb7-N101
129D emp1-R
129E ptd1-MS1568
129F dek*-MS2115
129G dek*-MS6214
130A o10-N1356
130B cp3-N888A
130BA cp3-N888A; mn4-N888C
130C id1-NA972

**CHROMOSOME 2 MARKER**

201A mrl1-IHO
201F ws3 lg1 gl2 b1
203B al1
203BA al1-Brawn
203BB al1-y3
203D al1 lg1
203G al1-y3 gl2
204A al1-lty3
205A al1 lg1 gl2
205B lg1
205C lg1 gl2
205G al1 gl2 B1
206A lg1 gl2 B1
206C D10-N2428
206D Wrp1-NA1163
206E oro2
207A y11
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 gl2-3050-3
210F gl2-PI200291
210G gl2-PI239114
210H gl2-PI251009
210I gl2-PI251885
210J gl2-PI251930
210K gl2-PI262474
210L gl2-PI262493
210M gl2-PI267186
210N gl2-N718
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::Bg Ch1
213H lg1 gl2 B1-v::Bg
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
218DB Ht1
218E ba2
218G B1-Peru; A1 A2 C1 C2 r1-r
218GA 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
219F B1-Peru; A1 A2 bz2 C1 C2 r1-g
219G B1-Bolivia-706B; A1 A2 C1 C2 r1-g
219H B1-Bolivia; A1 A2 C1 C2 Pl1 Pr1 r1-g
219I B1-I; A1 A2 C1 C2 Pl1-Rhoades r1-r
219J B1-I; A1 A2 C1 C2 Pl1-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
221AA gs2-0229
221C wlv1-N1860 Ch1
221G wlv1-N1860
224B v*-5537
224H whp1; A1 A2 C1 c2 R1
224I ws3-7752
224J ijmos*-7335
224K glnec*-8495
224L ws3-8949
224M ws3-8991
224N ws3-8945
226A ws3-N2357
226B b1-m1::Ds1; A1 A2 C1 C2 r1-g
226C b1-md2::Ds1; A1 A2 C1 C2 r1-g
226D b1-Pm5; A1 A2 C1 C2 r1-g
226E b1-Perum216; A1 A2 C1 C2 r1-g
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 ws3-N453A
228CA ws3-N605A
228E B1-Bh
228F ms33-6019
228G ms33-6024

228H ms33-6029
228I ms33-6038
228J ms33-6041
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
229H dek*-MS2159
229J dek*-PIE

**CHROMOSOME 3 MARKER**

301A cr1
301B bif2-N2354
301C spc3-N553C
301D Wi2-N1540
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::I
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
309M a1-m1-5719A1::dSpm sh2
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
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
313AB gl6-N672B
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
318EA sh2-N2307
318F sh2-N2340
318G na1
318H vp1-Mc
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
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
322I et1-24
322J et1-27
322K et1-34
322L et1-2162
322M et1-2320
322N et1-2424
322O et1-2457
322P et1-3191
322Q et1-3328
322R et1-5079
322S et1-84-6013
322T et1-88g-9733
322U et1-43
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
326AA sh2-Garwood
326AB sh2-60-156
326B vp1
326BA vp1-Mum3
326BC vp1-86N6
326BD vp1-86GN14
326BE vp1-86GN18
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 MARKER**

401A Rp4-a
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-PI239110
405BA la1-Funk:1087
405BB la1-Funk:2232
405BC la1-N2020
405BD la1-N2276B
405BE la1-PI184284
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-PI
407DE su1-R2412
407DF su1-N896A
407DG su1-N1161A
407DH su1-N2313
407DI su1-N2314
407DJ su1-N959
407DK su1-N1968
407DL su1-N1994
407E su1-am
407F su1-am; du1
408B bm3-Burnham su1
408C su1 zb6
408E bm3-91598-3
408J su1 ra3
408K su1; se1
408L su1 zb6 Tu1
409A su1-st
409B su1-66
409C su1-P
409D su1-5051
409F su1-28510
409G su1-28511
409H su1-28512

409I su1-28513
409J su1-28515
409K su1-28516
409L su1-28517
409M su1-28518
409N su1-28519
409O su1-28520
409P su1-30394
409Q su1-30397
409R su1-30398
409S su1-30399
409T su1-30400
409U su1-30401
409V su1-Bn2
409W su1-A3
409X su1-4582::Mu1
409Y su1-8064
409Z su1-2401
409ZA su1-3837
409ZB su1-7110
409ZC su1-2857
409ZD su1-2859
410D su1 zb6 gl3
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-Idf1(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
414AD bt2-5288
414B gl4
414BA gl4-Stadler
414BB gl4-gl16
414BD gl4-N525A
414C gl4 o1
414E de*-414E
415A j2
415B o1-N1243
415C o1-N1478A
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
418A gl3 dp1
418B c2; A1 A2 C1 R1
418D C2-Idf1(Active-1); A1 A2 C1 R1
418E dp1
418F o1
418G v17
419A v23-8914
419E gl7
419F Dt6 gl3 C2; a1-m A2 C1 R1
419G Dt6 C2; a1-m A2 C1 R1
419H c2-m1::Spm; A1 A2 C1 R1
419I c2-m2::dSpm c2-m3::Mpi1
419J c2-Mum1
419K c2-m2::dSpm; Spm-s
420A su1 Dt4 C2; a1-m A2 C1 R1
420C nec*-rd
420CA nec*-016-15
420D yel*-8957
420F dp*-4301-43
420G w*-9005
420H Dt4 C2; a1-m A2 C1 R1
424C gl3-64-4
424D gl3-56-3120-2
424E gl3-56-3129-27
424F gl3-60-2555
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
426B gl3-PI251941
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 Sos1-ref
428A gl5 Su1; gl20
428C nec5-N642
428D spt2-N1269A
428E wt2-N10
428F lw4; Lw3
428G bx1
428H gl5 su1; gl20
428L dsc1-MS2058

**CHROMOSOME 5 MARKER**

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 Nl2-N1445
502G A2 ga10; Bt1
503A A2 bm1 pr1 ys1; A1 C1 C2 R1
503B hcf43-N1277B
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 ys1; 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 ys1; 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(II)::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
510D a2 pr1 gl8; A1 C1 C2 R1
510E a2 ae1 pr1 gl8; 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 ga*-Rhoades; A1 C1 C2 R1
511H a2 bt1; A1 C1 C2 R1
512C a2 bt1 pr1 ga*-Rhoades; A1 C1 C2 R1
512D vp2-N1136B
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
515E bt1-N1992
515F bt1-N2308
515G bt1-N2309
516B bt1-R
516BA bt1-Elmore
516BB bt1-C103
516BC bt1-Singleton
516BD bt1-sh3
516BE bt1-sh5
516BF bt1-Eldridge
516BH bt1-6-783-7
516BI bt1-Vineyard
516BJ bt1-T
516BK bt1-W187R
516BL bt1-3040
516BM bt1-N797A
516C ms5
516D td1 ae1
516DA td1-Nickerson
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
517BH ae1-Elmore
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
519I zb1-2
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 lnec*-5931
521NA lnec*-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-N1634
528CA anl1-330C
528E prg1-MS8186
528F ren1-MS807
528H dek*-MS2146
528I dek*-MS1182
529B anl1-N1645
529C anl1-N1671
529D anl1-N1685
529E anl1-N1691

**CHROMOSOME 6 MARKER**

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
602P y1-129E
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-Brawn1
604F y1 si1-mssi
604FA y1 si1-ts8
604FB y1 si1-Sam
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
605G l3
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 Dt2 Pl1; a1-m A2 C1 C2 R1
610C pl1 sm1; P1-rr
610F Y1 pl1 su2 v7
610G hcf34-N1269C
610H Y1 Dt2 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 y1; A1 a2 bm1 C1 C2 pr1 R1 v2 wx1
613D vms*-8522
613F w14-8613
613I tus*-5267
613J gm*-6372
613L w*-8954
613M yel*-039-13
613N yel*-7285
613O l*-4-6(4447)
613P yel*-8631
613T pg11-6656; pg12-6656
627A dek28-N1307A
627B dek19-N1296A
627C vp*-5111
627G dek*-MS1104; l*-1104

**CHROMOSOME 7 MARKER**

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
706B vp9-Bot100
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-R
707EA vp9-3111
707EB vp9-86GN9
707EC vp9-86GN15
707F y8 gl1
707G in1 gl1; A1 A2 C1 C2 Pr1 R1
708A ra1
708B bd1-N2355
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-Istra
709AH gl1-BMS
709AI gl1-7L
709AJ gl1-9:COOP
709AK gl1-N212
709AL gl1-N269
709AM gl1-N345B
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 dlf1-N2389A
716D dlf1-N2461
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 ms34-6004
727I ms34-6010
727J ms34-6013
727K ms34-6014
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 MARKER**

801A gl18-g
801B v16
801I yel*-024-5
801K v16 ms8
802A rgh1-N1285
802B emp3-N1386A
802C Ht2
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
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
827CA Bif1-N2001
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 MARKER**

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-Hayes; A1 A2 C2 R1
902D yg2 c1 sh1 Bz1 wx1 gl15 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-I 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 K10 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-scm2
906A C1 wx1; A1 A2 C2 Dsl Pr1 R1 y1
906B C1 wx1; A1 A2 C2 Dsl pr1 R1 Y1
906C C1-I Wx1; A1 A2 C2 Dsl R1
906D C1-I; A1 A2 C2 R1
906G C1-I Sh1 Bz1 Wx1; Dsl
906H C1 Sh1 bz1 wx1; Ac
907A C1 wx1; A1 A2 C2 R1
907E C1-I 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
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-Hayes; A1 A2 C2 R1
911D Fas1
912A sh1
912AA sh1-1746
912AB sh1-9026-11
912AC sh1-3-6(6349)
912AD sh1-60-155
912AE sh1-EMS
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

914B dek12-N1054
914K Wc1-ly; Y1
914L bz1-Mus1
914M bz1-Mus2
914N bz1-Mus3
914O bz1-Mus5
914P bz1-Mus6
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
916G Trn1-N1597
916H v31-N828
917A wx1 Bf1-ref
917C v1
917D ms2
917DA ms2-6002
917DB ms2-6012
917E gl15-Sprague
917EA gl15-Lambert
917EB gl15-KEW
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::Ds
923ZA wx1-m6R
923ZB wx1-m6NR
923ZC wx1-m8::Spm-I8
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
925C bz1-m2::Ac
925D Wx1-m9r1
925E bz1-m2(DII)::Ds wx1-m6::Ds
925F C1 sh1 bz1 wx1-m8::Spm-I8
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 Dt7; 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-I8; A1 A2 C2 R1
928H wx1-m7::Ac7
928I C1 bz1-mut::rMut; A1 A2 Bz2 C2 Mut R1
928J C1 bz1-(r)d; A1 A2 C2 R1
928K C1 Sh1 bz1-s; A1 A2 C2 Mut R1
928L ms45-6006
928M ms35-6011
928N ms35-6018
928O ms*-6021
928P ms*-6022
928Q ms35-6027
928R ms35-6031
928S ms*-6046
928T ms*-6047
929E Dp9
930A wx1-Mum1
930B wx1-Mum2
930C wx1-Mum3
930D wx1-Mum4
930E wx1-Mum5::Mu
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
931H wd1-Mus1
931I wd1-Mus2
931J wd1-Mus3
931K wd1-Mus4
931L wd1-Mus5
931M wd1-Mus6

**CHROMOSOME 10 MARKER**

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
X03F Inr1-Ref
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 g1-56-3005-24
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-sc:m3::Ds
X14J R1-nj::Ac
X14K r1-Del902
X15B l1 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 isr1 r1-g sr2
X15H isr1 R1-r:PI302369
X15I isr1 R1-nj Mst1
X16B r1; A1 A2 abnormal-10 C1 C2
X16C R1-ch; A1 A2 C1 C2 Pl1
X16CA R1-ch
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 l1 w2
X19D o7
X19F r1 w2
X19G r1-n19 Lc1; b1
X19H r1-g:e Lc1; b1
X20B l1
X20C v18
X20I R1-d:Arapaho

X20J R1-d:Catspaw
X24A cm1
X24B lep*-8691
X24C v*-8574
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 In1-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 l19-N425
X27J l13-N59A
X27K v29-N418
X27L Les12-N1453
X28B R1-scm2; a1-m1::rDt (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
X28I R1-scm2; a1-m1-5719::dSpm A2 C1 C2
X28J R1-scm2; A1 A2 bz1 C1 C2
X29A ren3-MS1339
X29B dek*-MS2181

## UNPLACED GENES

U140C l4
U140G ms22
U140H ms24
U240A Les7-N1461
U240D o11
U240E zn2
U240F zn2-PI251887
U240G zn2-PI236997
U240H zn2-PI239110
U240I zn2-56-3012-10
U340D ws1-COOP ws2-COOP
U340DA ws1-Pawnee ws2-Pawnee
U340H oro4
U440B gl13
U440C hcf49-N1480
U440D ub1-76C
U440E frz1
U440F mg1-Sprague
U540A dv1
U540B dy1
U640A dsy1-Doyle
U640B dsy1-Russian
U640C pam1
U640D pam2
U640E ada1
U640F atn1 Adh1-1S5657
U740A abs1-PI254851
U740B y2
U740C lty1
U740D lty2
U740F pi1 pi2
U740G Fbr1-N1602
U740H ad2-N2356A
U840A csp1-NA1173
U840B rli1-N2302A
U840D Les21-N1442
U840E zb3
U840F agt1
U840G Wi3-N1614
U840H nld1-N2346

## MULTIPLE GENES

M141A A1 A2 B1 C1 C2 Pl1 Pr1 R1-g
M141AA A1 A2 B1 C1 C2 Pl1-Rhoades Pr1 R1-g
M141B A1 A2 B1 C1 C2 pl1 Pr1 R1-g
M141D A1 A2 b1 C1 C2 pl1 R1-g
M142A A1 A2 b1 C1 C2 pl1 R1-r
M142B a1 A2 b1 C1 C2 pl1 R1-r
M142C A1 a2 b1 C1 C2 pl1 R1-r
M142D A1 A2 b1 bz1 C1 C2 pl1 R1-r
M142E A1 A2 b1 bz2 C1 C2 pl1 R1-r
M142F A1 A2 b1 c1-p C2 pl1 R1-r
M142G A1 A2 b1 C1-I C2 pl1 R1-r
M142H A1 A2 b1 C1 c2 pl1 R1-r
M142I A1 A2 b1 C1 C2-Idfm pl1 R1-r
M142J A1 A2 b1 C1 C2-Idf1(Active-1) pl1 R1-r
M142K A1 A2 b1 C1 C2 pl1 pr1 R1-r
M142L A1 A2 b1 C1 C2 gl1 in1 pl1 R1-r
M142M A1 A2 b1 C1 C2 In1-D pl1 R1-r
M142O C1 sh1 bz1 wx1; A1 A2 C2 R1-r
M142P c1 sh1 wx1; A1 A2 C2 R1-r
M142Q yg2 c1 sh1 wx1; A1 A2 C2 R1-g
M142R A1 A2 C1-I C2 R1-r wx1
M142S su1 c2; A1 A2 C1 R1-r
M142T A1 A2 b1 C1 C2 pl1 r1-g
M142U A1 A2 b1 C1 C2 pl1 r1-r
M142V A1 A2 C1 C2 R1-nj
M142W A1 A2 C1 C2 R1-st
M142X A1 A2 b1 C1 C2 Pl1 r1-g
M142Y A1 A2 B1 C1 C2 Pl1 r1-g
M142Z a1-st A2 b1 C1 C2 pl1 R1-scm2
M142ZA A1 a2 b1 C1 C2 pl1 R1-scm2
M142ZC A1 A2 b1 bz2 C1 C2 pl1 R1-scm2
M142ZD A1 A2 b1 c1-n C2 pl1 R1-scm2
M142ZE A1 A2 b1 c1-p C2 pl1 R1-scm2
M241A A1 A2 B1 C1 C2 Pl1 Pr1 r1-g
M241C A1 A2 B1 C1 C2 Pl1 Pr1 R1-r
M241D A1 A2 b1 C1 C2 Pl1-Rhoades r1-g
M242A A1 A2 b1 C1 c2 pl1 R1-scm2
M242B A1 A2 b1 C1 C2 pl1 pr1 R1-scm2
M242C in1 gl1; A1 A2 b1 C1 C2 pl1 R1-scm2
M242D a1 sh2; A2 b1 C1 C2 pl1 R1-scm2 wx1
M242E c1 sh1 wx1; A1 A2 b1 C2 pl1 R1-scm2
M242F su1 c2; A1 A2 b1 C1 pl1 R1-scm2
M242G A1 A2 b1 C1 C2 pl1 R1-scm2
M242H A1 A2 b1 C1 C2 pl1 r1-g
M242I A1 A2 b1 C1 C2 pl1 r1-r
M340A A1 A2 B1 c1 C2 pl1 Pr1 R1-g
M340B 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 Pl1 Pr1 R1-r
M341CA A1 A2 b1 C1 C2 Pl1-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 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-I C2 R1-nj
M541J A1 A2 C1 c2 R1-nj
M541K A1 A2 C1 C2-Idf1(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
M741B Stock 6; A1 A2 B1 C1 C2 Pl1 R1-r
M741C Stock 6; A1 A2 B1 C1 C2 pl1 R1-r
M741F Stock 6; A1 A2 C1 C2 pl1 R1-g y1
M741G Stock 6; A1 A2 C1-I C2 pl1 R1-g wx1 y1
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
M841D v2 wx1
M841F A1 A2 bz2 C1 C2 R1-scm2 wx1
M841G A1 A2 C1 c2 R1-scm2 wx1
M841I su1 wx1
M941A A1 A2 c1 C2 Pr1 R1 wx1 y1
M941B Mangelsdorf's tester; a1 bm2 g1 gl1 j1 lg1 pr1 su1 wx1 y1
M941C a1 Dt1 gl2 lg1 wt1
M941D gl1 wx1 y1
M941E gl8-R wx1 y1
MX40A A1 A2 C1 C2 P1-vv::Ac r1-sc:m3::Ds
MX40B A1 A2 Ac2 bz2-m::Ds C1 C2 R1
MX40C A1 A2 C1 C2 r1-sc:m3::Ds trAc8168
MX41A A1 A2 C1 C2 gl1 pr1 R1 wx1 y1
MX41B A1 A2 C1 C2 gl1 pr1 R1 su1 wx1 y1
MX41C a1 a2 bz1 bz2 c1 c2 pr1 r1 wx1 y1
MX41D a1 A2 C1 C2 gl1 pr1 R1 su1 wx1 y1
MX41E a1-m1-n::dSpm A2 C1 C2 R1 wx1-m8::Spm-I8

## B-CHROMOSOME

B542A_Black Mexican Sweet; B chromosomes present
B542B_Black Mexican Sweet; B chromosomes absent

## TRISOMIC

123A trisomic 1
223A trisomic 2
328A trisomic 3
422A trisomic 4
523A trisomic 5
615A trisomic 6
718A trisomic 7
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 rfT
C736EA Tr (S) Restored; cms-S Rf3 rfC rfT
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 rfT
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
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
CX36A N6 (C) Restored; cms-C rf1 Rf2 rf3 RfC
CX36B N6 (S) Sterile; cms-S rf1 Rf2 rf3 RfC
CX36C B37 (C) Sterile; cms-C rf1 Rf2 rf3 rfC
CX36D B37 (S) Sterile; cms-S rf1 Rf2 rf3 rfC

**CYTOPLASMIC TRAIT**

C337A NCS2
C337B NCS3

**TOOLKIT**

T0318AA TB-3Ld Ig1; ig1R1-nj
T0318AB cms-L; ig1 R1-nj
T0318AC cms-MY; ig1 R1-nj
T0318AD cms-ME; ig1 R1-nj
T0318AE cms-S; ig1 R1-nj
T0318AF cms-SD; ig1 R1-nj
T0318AG cms-VG; ig1 R1-nj
T0318AH cms-CA; ig1 R1-nj
T0318AI cms-C; ig1 R1-nj
T0318AJ cms-Q; ig1 R1-nj
T0940A Hi-II Parent A (for producing embryogenic callus cultures)
T0940B Hi-II Parent B (for producing embryogenic callus cultures)
T0940C Hi-II A x B (for producing embryogenic callus cultures)
T0940D 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
T3312C Ds-1S3 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 TRANSLOCATIONS (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 TRANSLOCATIONS (OTHERS)**

122C TB1-Lc
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
225B TB-1Sb-2Lc
320P TB-1La-3Le
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
331E TB-3Lf
331F TB-3Lg
331G TB-3Lh
331H TB-3Li
331I TB-3Lj
331J TB-3Lk
331K TB-3Ll
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 Dt6; TB-4Sa
522B TB-5Lb
522D TB-5Ld
528D TB-1La-5S8041
614A TB-6Lb
627E Dt2; a1-m A2 C1 C2 R1 TB-6Lc
720A Dt3; 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
TX40D P1-vv::Ac; r1-sc:m3::Ds TB-1Sb
TX40F Ac2; bz2-m::Ds TB-8Lc
TX40G a1-m; Dt1 TB-9Sd
TX40H r1-sc:m3::Ds; TB-9Lc trAc8168
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 (1.S.30; 1.L.01)
I143C Inv1d (1.L.55; 1.L.92)
I143D Inv1k (1.L.46; 1.L.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.70; 9L.90)
I443A Inv4b (4S.10; 4L.12)
I443B Inv4c (4S.89; 4L.62)
I444A Inv2a (2S.70; 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)
I943E Inv7a (7L.05; 7L.95)
IX43A Inv8a (8S.30; 8L.15)
IX43B Inv9b (9S.05; 9L.87)

**RECIPROCAL TRANSLOCATIONS (Wx1 AND wx1 MARKED)**

wx01A T1-9c (9L.22; 1.S.48); wx1
wx01B T1-9(5622) (9L.12; 1.L.10); wx1
wx02A T1-9(4995) (9S.20; 1.L.19); wx1
wx02AA T1-9(4995) (9S.20; 1.L.19); wx1
wx03A T1-9(8389) (9L.13; 1.L.74); wx1
wx04A T2-9c (9S.33; 2S.49); wx1
wx05A T2-9b (9L.22; 2S.18); wx1
wx06A T2-9d (9L.27; 2L.83); wx1
wx07A T3-9(8447) (9L.14; 3S.44); wx1
wx08A T3-9c (9L.12; 3L.09); wx1
wx09A T3-9(8562) (9L.22; 3L.65); wx1
wx10A T4-9e (9L.26; 4S.53); wx1
wx11A T4-9g (9L.27; 4S.27); wx1
wx12A T4-9(5657) (9S.25; 4L.33); wx1
wx13A T4-9b (9L.29; 4L.90); wx1
wx14A T5-9c (9L.10; 5S.07); wx1
wx14B T5-9(022-11) (9L.27; 5S.30); wx1
wx15A T5-9(4817) (9S.07; 5L.06); wx1
wx16A T5-9d (9L.10; 5L.14); wx1
wx17A T5-9a (9S.17; 5L.69); wx1
wx18A T6-9(4778) (9L.30; 6S.80); wx1
wx19A T6-9a (9L.40; 6S.79); wx1
wx19B T6-9e (9L.24; 6L.18); wx1
wx20A T6-9b (9S.37; 6L.10); wx1 y1
wx21A T6-9(4505) (9ctr.00; 6L.13); wx1
wx22A T7-9(4363) (9ctr.00; 7ctr.00); wx1
wx23A T7-9a (9S.07; 7L.63); wx1
wx24A T8-9d (9S.16; 8L.09); wx1
wx25A T8-9(6673) (9S.31; 8L.35); wx1
wx26B T9-10(059-10) (9S.31; 10L.53); wx1
wx27A T9-10b (9S.13; 10S.40); wx1
wx28A T5-9(8386) (9S.13; 5L.87); wx1
Wx30A T1-9c (9L.22; 1.S.48); Wx1
Wx30B T1-9(4995) (9S.20; 1.L.19); Wx1
Wx30C T1-9(8389) (9L.13; 1.L.74);

Wx1
Wx31A T2-9c (9S.33; 2S.49); Wx1
Wx31B T2-9b (9L.22; 2S.18); Wx1
Wx31C T2-9d (9L.27; 2L.83); Wx1
Wx32A T3-9(8447) (9L.14; 3S.44); Wx1
Wx32B T3-9(8562) (9L.22; 3L.65); Wx1
Wx32C T3-9c (9L.12; 3L.09); Wx1
Wx33A T4-9e (9L.26; 4S.53); Wx1
Wx33B T4-9(5657) (9S.25; 4L.33); Wx1
Wx33C T4-9g (9L.27; 4S.27); Wx1
Wx34A T5-9c (9L.10; 5S.07); Wx1
Wx34B T5-9(4817) (9S.07; 5L.06); Wx1
Wx34C T4-9b (9L.29; 4L.90); Wx1
Wx35A T5-9(8386) (9S.13; 5L.87); Wx1
Wx35B T5-9a (9S.17; 5L.69); Wx1
Wx35C T5-9d (9L.10; 5L.14); Wx1
Wx36A T6-9(4778) (9L.30; 6S.80); Wx1
Wx37A T6-9(8768) (9S.61; 6L.89); Wx1
Wx37B T7-9(4363) (9ctr.00; 7ctr.00); Wx1
Wx37C T6-9(4505) (9ctr.00; 6L.13); Wx1
Wx38A T7-9a (9S.07; 7L.63); Wx1
Wx38B T8-9d (9S.16; 8L.09); Wx1
Wx38C T8-9(6673) (9S.31; 8L.35); Wx1
Wx39A T9-10(8630) (9S.28; 10L.37); Wx1
Wx39B T9-10b (9S.13; 10S.40); Wx1

## PHENOTYPE ONLY

### Kernel Traits

**blotched aleurone**
Bh*-86-1381-1
Bh*-Tu*-Mumm

**brittle endosperm**
bt*-011-11
bt*-1979-14
bt*-1979-16
bt*-1982
bt*-4380
bt*-4539
bt*-4973
bt*-60-151
bt*-8101
bt*-8102
bt*-83-84-3541-1
bt*-84-4
bt*-84-5
bt*-84-5091-9
bt*-84-5257-1
bt*-84-6
bt*-85-3096-6
bt*-85-3098-15
bt*-85-3099-16
bt*-85-3372-27
bt*-87-2132-39
bt*-87-2254-2
bt*-87-88-2630-28
bt*-8804
bt*-8805
bt*-88-3177-14
bt*-88-3177-2
bt*-88-3177-7
bt*-89-1265-18
bt*-90286
bt*-A4109
bt*-Alexander
bt*-F10
bt*-F-15
bt*-F-23
bt*-F-31
bt*-F-34
bt*-F-36
bt*-F-8
bt*-Panzio
bt*-PetersonResHy
bt*-PI200197
bt*-PI251887
bt-gm*-84-5045-39
bt-gm*-85-3017-24
bt-sh*-PI251930

**brown endosperm**
brn*-1981-1
brn*-1981-2
brn*-1981-3
brn*-1981-4
brn*-84-23
brn-bt*-81-F-24
lt-brn-sm*-86-1302-37

**brown pericarp**
bp*-PI183639

**colored plumule**
Pu*-1976-RYDCO

**colorless aleurone**
cl*-86-1478-16
cl*-85-86-3559-1
cl-crown-pale-base*-85-86-3558-23
r*-86-151590-6

**defective kernel**
de*-1276
de*-1364
de*-17
De*-1976-RYDCO
de*-2080
de*-2192
de*-2424
de*-2915
de*-2919
de*-3188
de*-4309
de*-5044Hagie
de*-85-86-3567-35
de*-8505
de*-8507
de*-8508
de*-86-1472-6
de*-8808
de*-8809
de*-8811
de*-8818
de-oro*-6577
de-small*-8813
de-small*-8814
de-small*-8815
de-small*-8816
de-small*-8817
def*-8101
def*-8102
def*-8103
def*-8104
def*-8105
def*-8106
def*-8107
def*-8108
def*-8109
def*-8110
def*-8111
def*-8112
def*-8113
def*-8114
def*-8116
def*-8118
def*-8119
def*-8120
def*-8121
def*-8122
def*-8123
def*-8125
def*-8126
def*-8127
def*-8128
def*-8130
def*-8131
def*-8132
def*-8134
def*-8136
def*-8137
def*-8138
def*-8201
def*-84-22
def*-84-28
def*-84-29
def*-84-30
def*-84-31
def*-84-37
def*-84-40
def*-84-41
def*-84-45
def*-84-48
def*-84-49
def*-84-53
def*-84-54
def*-84-58
def*-84-60
dek*-1979-32
dek*-1981-1
dek*-74-0060-4
dek*-84-14
dek*-86-1496-35
dek*-8902
dek*-8903
dek*-8904
dek*-F-16
dek*-PS602
wrinkled-de*-86-1473-5
wrinkled-gm*-86-1582-32

**discolored kernel**
pig*-84-5080-18
pig-gm*-1979-51
pig-gm*-1979-52
pig-gm*-1979-9
pig-gm*-1981-A
pig-gm*-1981-B
pig-gm*-1982-3
pig-gm*-84-5078-10
pig-gm*-86-1200-3
pig-gm*-87-2275-15
pig-gm*-87-2305-22
pig-gm*-Briggs 1998-1
pig-gm*-Briggs 1998-2
pig-gm*-PI251930
ptd-dek*-1976-RYDCO
ptd-dek*-1981
small-pig-gm*-88-89-3554-44

**dull endosperm**
du*-0203

**etched endosperm**
et*-3130
et*-3576
et*-5191
et*-6-9321-1
et*-73-766-1
et*-84-5266-26
et*-84-5270-40
et*-86-1493-6
et*-8616
et*-86-3518-21
et*-87-2349-13
et*-88-89-3525-22
et*-88-89-3554-33
et*-89-90-1547-19
et*-8-M-4
et*-Mu1767
et*-Mu2349
et*-Osturana
et-de*-3526-8
et-gm*-86-1475-34
et-gm*-86-87-1742-38
et-gm*-87-2502-19
granular-o*-84-5274-30
small-et*-85-3527-29
sm-et*-85-86-3522-29

**flint kernel**
flint*-87-2126-22

**floury endosperm**
fl*-67-412
fl*-83-3386-19
fl*-84-44
fl*-8515
fl*-Mojo
fl*-N7B-65-1294
fl*-shoepeg
fl*-sucaxo
fl-cap*-1981
fl-cap*-66-519-1
fl-de*-8905
small-fl-cap*-1981

**germless**
brn-gm*-85-3315-6
brn-gm*-85-86-3587-46
brn-gm*-85-86-3595-3
brn-gm*-86-1161-5
emb*-85-3100-32
emb*-85-3378-8
gm*-1387
gm*-1979-11
gm*-1979-53
gm*-5234
gm*-6372
gm*-8510
gm*-86-1011-2
gm*-86-1013-4
gm*-86-1335-1
gm*-86-1591-7
gm*-86-87-1742-18
gm*-87-2456-9
o-gm*-84-44
pr-gm*-86-1109-1
sh-gm*-84-5045-32
sh-gm*-88-3082-4
small-dsc-gm*-95W-240
sm-o-gm*-86-1323-4
w-o-gm*-85-3135-4
w-o-gm*-86-1349-1
w-o-gm*-88-3270-10
y-gm*-85-3288-28

**glassy endosperm**
ae*-1979-1
ae*-1979-7
ae*-1981-MuT
ae*-84-7
ae*-92-1365-3
ae*-Briggs 1998-1
ae*-Mu32
ae*-Mu92-1365-3

**lemon white**
lw*-1979-45
lw*-1979-46
lw*-1981
lw*-1998-1
lw*-1998-2
lw*-1998-4
lw*-73-2548
lw*-82-1
lw*-8509
lw*-8513
lw*-8514
lw*-85-3076-28
lw*-85-3252-5
lw*-PI200203
pale-y*-83-84-3549-13
pale-y*-84-5082-33
pale-y*-84-5167-48
pale-y*-84-5288-19
pale-y*-85-3005-22
pale-y*-85-3006-30
pale-y*-85-3007-40
pale-y*-85-3010-40

pale-y*-85-3016-15
pale-y*-85-3017-31
pale-y*-85-3065-25
pale-y*-85-3069-6
pale-y*-85-3087-29

**marbled aleurone**
Dap*-3
Dap*-86-8126-2
Dap*-89-3177.0
Dap*-89-3177.5
Dap*-89-3178.3
Marbled*-Sprague

**miniature kernel**
mn*-1981-51
mn*-87-2215-17
mn*-87-2346-20
mn*-87-2347-36
mn*-88-89-3564-25
mn*-PI239110
mn*-PI245132
small-k*-97-4784-1

**mottled aleurone**
Mt*-2313
Mt*-65-2238
Mt*-Sprague

multiple aleurone layer
Mal*-Galinat
Mal*-Nelson
Mal*-PI515052

**opaque endosperm**
o*-1979-54
o*-1981-11-Fox-19
o*-1981-3-Fox-7
o*-1981-5-Fox-9
o*-1981-6-Fox-10
o*-1981-8-Fox-15
o*-1982
o*-1982-2-Fox-13
o*-2-Fox-6
o*-3015
o*-73-798-1
o*-76GH-76
o*-8129
o*-82:288-1
o*-83-3367-11
o*-83-3399-20
o*-83-3399-6
o*-83-84-3549-39
o*-84-5025-15
o*-84-5025-17
o*-84-5025-8
o*-84-5044-35
o*-84-5091-13
o*-84-5094-4
o*-84-5095-23
o*-84-5117-16
o*-84-5261-37
o*-84-5270-40
o*-84-5282-27
o*-84-5295-13
o*-84-5321-28
o*-84-5324-29
o*-84-8a
o*-85-3084-8
o*-85-3088-3
o*-85-3335-35
o*-86-87-1767-10
o*-87-2350-2
o*-97-4784-6
o*-BS20-Fox-3
o*-de*-1981-9-Fox-18
o*-Fox-12
o*-PI195245
o*-PI200285
o-dek*-6
o-gm*-83-3398-6
o-gm*-84-33
os*-2162
o-sh*-86-1297-2
o-sh*-F1979-19
pro*-Mu1
sh-o*-87-2455-7
small-o*-PI195243

**pale aleurone**
pa-Cl*-m-86-1474-39
pa-Cl*-m-86-1478-4
pale-Cl*-86-1476-14
pale-Cl*-LGC65
pale-Cl-gm*-84-5251-1

**pale crown**
pa-crown*-85-86-3558-23
Pale-Cl-mut*-87-2224-33

**pale yellow endosperm**
al*-84-5020-32
pale-endo*-73-3
pale-endo*-73-4004
pale-y*-83-3382-16
pale-y*-83-3382-18
pale-y*-83-84-3548-25
pale-y*-84-5103-16
pale-y*-84-5275-14
pale-y*-85-3016-30
pale-y*-85-3036-38
pale-y*-85-3042-7
pale-y*-85-3044-34
pale-y*-85-3134-46
pale-y*-85-3374-13
pale-y*-85-3377-2
pale-y*-85-3511-18
pale-y*-85-3562-31
pale-y*-85-86-3533-9
pale-y*-86-1151-7
pale-y*-87-2160-16
pale-y*-87-2350-2
pale-y*-87-2350-25
pale-y*-90-3220-1
pale-y*-90-3220-20
pale-y-gm*-Rsssc-77-110
pale-y-o*-84-5288-2
pale-y-o*-86-1296-27
small-y*-95-1930-2
y*-84-5272-12
y*-84-5288-1
y*-85-3041-2
y*-85-3078-41
y*-85-3087-12
y*-85-3125-7
y-sh*-86-1583-22

**red silk scar**
red-silk-scar*-MTC

**shrunken kernel**
sh*-1979-10
sh*-1982-2
sh*-2927-Mumm
sh*-2928-Mumm
sh*-83-3328-24
sh*-84-3
sh*-84-5248-20
sh*-84-5317-44
sh*-8502
sh*-8503
sh*-8506
sh*-8511
sh*-8517
sh*-85-3045-7
sh*-85-3104-27
sh*-85-3112-20
sh*-85-3375-38
sh*-86-1565-17
sh*-87-2045-25
sh*-87-2045-6
sh*-87-2050-1
sh*-87-2050-3
sh*-87-2355-29
sh*-87-2406-3
sh*-8806
sh*-8807
sh*-8906
sh*-8907
sh*-97P-29-5
sh*-Alexo1968
sh*-F-11
sh*-F-2
sh*-F-25
sh*-KERR
sh*-RJL
sh-bt*-85-3392-31
sh-de*-6607
sh-de*-RSSSC-117
sh-fl*-9180
sh-fl*-9392
sh-wx*-F-18
su-sh*-F-5

**spotted aleurone**
cl-mut*-85-86-3564-1
coarse-mutable*-86-1417-7

**sugary kernel**
su*-1979-5
su*-1979-8
su*-1981
su*-1982
su*-83-3383-21
su*-83-3383-4
su*-84-5167-6
su*-84-5267-18
su*-84-5350-2
su*-84-5350-31
su*-8501
su*-8504
su*-85-3113-11
su*-85-3133-32
su*-85-3217-10
su*-85-3436-29
su*-87-2046-27
su*-8801
su*-8802
su*-8803
su*-8908
su*-F-37
su*-MOEWS
su*-PI193430
su*-PI228183
su-sh*-Briggs-1998-1
su-sh*-F-22

**viviparous kernel**
pale-vp*-87-2286-1
pale-vp*-87-2286-18
pale-vp*-87-2286-2
pale-vp*-87-2286-25
pale-vp*-87-2286-3
pale-y*-84-5027-22
pale-y*-84-5032-21
pale-y-vp*-84-5266-5
pale-y-vp*-85-3100-31
pale-y-vp*-85-3140-15
pale-y-vp*-85-3240-5
pale-y-vp*-85-3267-6
pale-y-vp*-85-3385-34
pale-y-vp*-86-1316-27
pale-y-vp*-88-3177-14
ps*-85-3288-28
ps*-85-3492-36
ps*-85-86-3567-1
ps*-86-1105-2
ps*-86-87-1742-18
ps*-96-5032-6
ps*-Mu85-3061-21
ps*-Mu86-1105-1
vp(ps)*-86-1449-3
vp(ps)*-86-1565-17
vp*-0118
vp*-0315
vp*-2-8c
vp*-71-1367
vp*-73-30173
vp*-8101
vp*-8104
vp*-8106
vp*-8107
vp*-8108
vp*-8109
vp*-8110
vp*-8111
vp*-8112
vp*-8113
vp*-8114
vp*-8115
vp*-8116
vp*-8117
vp*-8201
vp*-8203
vp*-8204
vp*-8205
vp*-8208
vp*-8209
vp*-8210
vp*-8211
vp*-8418
vp*-8420
vp*-84-5079-29
vp*-84-5279-29
vp*-84-5315-29
vp*-85-3011-11
vp*-85-3017-9
vp*-85-3040-29
vp*-85-3042-7
vp*-85-3099-16
vp*-85-3135-4
vp*-85-3182-6
vp*-85-3339-25
vp*-85-3422-13
vp*-85-86-3567-20
vp*-86-1109-1
vp*-86-1407-15
vp*-86-1573-27
vp*-87-2146-18
vp*-87-2339-1
vp*-95-2086-1
vp*-PI 183642
vp*-PI 200204
vp*-PI 254854
vp*-PI 430482
vp*-PI185847
vp-de*-87-2406-23
vp-Y*-86-1267-31
vp-Y*-86-1361-7
w-vp*-84-5020-4
w-vp*-85-3014-6
w-vp*-85-3304-13
w-vp*-91-1859-8
w-vp*-91-2544-7
y-vp*-0730
y-vp*-1982-1
y-vp*-1982-2
y-vp*-2062-Coop
y-vp*-60-153
y-vp*-65-792
y-vp*-6961
y-vp*-73-2656
y-vp*-80-6118
y-vp*-8102
y-vp*-8103
y-vp*-8105
y-vp*-8206
y-vp*-8207
y-vp*-83-13
y-vp*-83-1A
y-vp*-83-3101-36
y-vp*-8336
y-vp*-8419
y-vp*-8512
y-vp*-85-3572-30
y-vp*-8701
y-vp*-Alexho68-195

**waxy endosperm**
wx*-0208
wx*-87-2254-2

**white cap kernel**
Wc*-1982-1
Wc*-DC

**white endosperm**
y*-1981
y*-1982-3
y*-73-2
y*-73-2262-1
y*-73-2262-2
y*-73-2394
y*-73-324-1
y*-73-4035
y*-73-426
y*-84-8b
y*-87-2201-3
y*-syn-DOCI
y*-Williams-60-154

## Seedling Traits

**albino seedling**
nlw*-85-3357-17
peach-albino-mutable*-87-2209-30
w*-005-19
w*-017-14-A
w*-017-14-B
w*-034-16
w*-037-14
w*-039-15
w*-2246 Funks
w*-3858
w*-4670
w*-4873
w*-5201
w*-5255
w*-5863
w*-6293
w*-6575
w*-7165
w*-7281
w*-78-297-3
w*-8105W Funks
w*-8129
w*-8147
w*-8201
w*-84-5205-46
w*-84-5222-30
w*-85-3359-11
w*-85-3552-25
w*-85-3559-30
w*-8549
w*-86-1078-6
w*-86-1265-30
w*-86-2222-5
w*-8630
w*-8635
w*-8637
w*-8670
w*-8963
w*-8977
w*-B-75
w*-PI184276
w*-PI201543
w*-PI228176
w*-PI228179
w*-PI232965
w*-PI232968
w*-PI232972
w*-PI239103
w*-PI239110
w*-PI251009
w*-PI251885
w*-PI251930
w*-PI251932
w*-PI254851
w*-PI267162
w*-Singleton-16
w*-Singleton-24
w*-Singleton-25
w*-Tama
wh*-053-4
wh*-89-578-6
wh*-BMS-Rhoades

**clasping leaf**
clasping-leaf*-87-2320-9

**glossy leaf**
gl*-218-1
gl*-32TaiTaiTaSarga
gl*-4339
gl*-5249
gl*-56-3023-6
gl*-56-3023-9
gl*-56-3036-7
gl*-6
gl*-60-2484-8
gl*-63-3440-8
gl*-85-3095-12
gl*-87-2215-30
gl*-97P-261-5
gl*-Bizika
gl*-gl12
gl*-LGC#117
gl*-LGC#27
gl*-Manglesdorf
gl*-Moritsa
gl*-PI184286
gl*-PI200203
gl*-PI228177
gl*-PI239101
gl*-PI239110
gl*-PI251885
gl*-PI251933
gl*-PI262474
gl*-PI262476
gl*-PI262494
gl*-PI262500
gl*-PI267203
gl*-PI267209
gl*-PI267212

**high chlorophyll fluorescence**
hcf*-88-3005-3

**luteus yellow seedling**
l*-2215
l*-84-5225-33
l*-85-3225-4
l*-85-3457-40
l*-85-3513-1
l*-85-3541-20
l*-86-1354-9

**orobanche**
oro*-85-3087-3
oro*-85-3106-41
oro*-85-3113-11
oro*-88-89-3550-32

**pale green seedling**
pas*-90-3222-13
pg*-84-5205-13
pg*-84-5234-29

**piebald leaf**
pb*-87-2442-5

red seedling leaf
red-leaf*-86-1569-7

**translucent leaf**
trans-leaf*-56-3122-7
trans-leaf*-68F-958
trans-leaf*-79-6533
trans-leaf*-PI228176

**tube leaf**
fused-leaves*-PI228170

**virescent seedling**
v*-002-17
v*-007-18
v*-022-17
v*-025-4
v*-037-5
v*-1-2(5376)
v*-1-9(5622)
v*-2-9(5257)
v*-388-Sprague
v*-4308
v*-4698
v*-5-10(5355)
v*-5287
v*-5413
v*-5575
v*-56-3012-10
v*-5828
v*-60-151
v*-60-2397-15
v*-65-1433
v*-7230
v*-7281
v*-7312
v*-74-1690-1
v*-74-1873-1
v*-74-1948-1
v*-8070
v*-8129
v*-8201
v*-8339
v*-8522
v*-8654
v*-8743
v*-8806
v*-8957
v*-8958
v*-9026
v*-Funk-84-13
v*-leng
v*-LGC-111
v*-LGC-142
v*-LGC-98
v*-pb-3019-16
v*-PI183640
v*-PI185851
v*-PI195244
v*-PI195245
v*-PI200197
v*-PI200201
v*-PI228174
v*-PI228176
v*-PI236996
v*-PI239105
v*-PI239114
v*-PI239116
v*-PI251883
v*-PI251891
v*-PI251930
v*-PI254856
v*-PI262476
v*-PI262487
v*-PI262489
v*-PI267184
v*-PI267209
v*-PI267212
v*-PI267226
v*-PI270293
v*-Pollacsek
v*-RumanianFlint
v*-Singleton-22
v*-Singleton-34

**white striped seedling**
str*-84-5222-7
str*-86-1494-27

**yellow green seedling**
yg-nec*-95-5320-7
Yg-str*-Mu

**zebra striped seedling**
zb*-89-3137-5

## Plant Traits

**absence of leaf blade**
bladeless*-87-2406-23

**barren stalk**
ba*-1447
ba*-68-679-8
ba*-74-304-12
ba*-74-369-2
ba*-PI200290
ba*-PI218135
ba*-PI239105
ba*-PI251885
ba-ub*-94-4712

**brachytic plant**
br*-2180
br*-228171
br*-78-136KEW
br*-OSIJEK-Yugoslavia
br*-PI239105
br*-Singleton#8
br*-Singleton1969-252
td*-PI262476

**brown midrib**
bm*-PI228174
bm*-PI251009
bm*-PI251893
bm*-PI251930
bm*-PI262480
bm*-PI262485
bm*-PI267186

**chromosome breaking**
Chrom-breaking*-Mu

**colored leaf**
lc*-PI239110

**crinkled leaf**
cr*-97P-111
cr*-98-1698

**defective tassel**
Tp*-54-55-Jos.
Tp*-PI213734
Tp*-T8-Jos.
Tp*-Tenn61

**dwarf plant**
d*-018-3
d*-119
d*-136-220
d*-1821
d*-2108
d*-2201
d*-2447-8
d*-3047
d*-3-eared-JC
d*-5312
d*-56-3037-23
d*-60-2428
d*-64-4156-1
d*-74-1701-5
d*-75-6071-1
d*-76-1304-9
d*-76-2186
d*-78-282-3
d*-78-286-1
d*-78-286-5
d*-87-2198-36
d*-gl11
d*-MarovacWhiteDent
d*-PI180231
d*-PI184286
d*-PI213769
d*-PI228169
d*-PI228171
d*-PI239110
d*-PI245132
d*-PI251652
d*-PI251656
d*-PI251885
d*-PI254854
d*-PI262495

d*-PI267219
d*-rosette
d*-shlf9-436-1
d*-su
d*-su2
d*-Teo
d*-ts1

**liguleless**
lg*-32TaiTaiTaSarga
lg*-56-3037-5
lg*-64-36
lg*-64-4
lg*-67-501-6
lg*-PI184281
lg*-PI200299
lg*-PI228170
lg*-PI262493
lg*-ZCXGRB

**male sterile**
ms*-6015
ms*-6026
ms*-6028
ms*-6033
ms*-6036
ms*-6039
ms*-6040
ms*-6045
ms*-6048
ms*-6049
ms*-6052
ms*-6054
ms*-6055
ms*-6057
ms*-6058
ms*-6059
ms*-6060
ms*-6061
ms*-6062
ms*-6064
ms*-6065
ms*-6066
ms*-PI217219
ms-si*-355

**multiple midrib**
multiple-midrib*-87-2406-23

**narrow leaf**
nl*-5688
nl*-PI245132

**ramosa**
ra*-412E
ra*-4889
ra*-D
ra*-PI184279
ra*-PI239103
ra*-PI262495
ra*-PI267181
ra*-PI267184

**reduced pollen fertility**
ga*-0188
ga*-0213
ga*-3615
ga*-94-764
Ga*-Yugoslavia

**tassel seed**
ts*-0174
ts*-69-Alex-MO17
ts*-Anderson
ts*-PI200203
ts*-PI267209
ts*-Sprague

**terminal ear**
te*-Galinat

## Ear Traits

**distichous ear**
distichous*-68-1227

**distorted segregation**
off-ratio*-85-3255-6
off-ratio*-86-1155-1
wx-off-ratio*-86-1110-4

**polytypic ear**
pt*-McClintock
pt*-Mu

**silky**
si*-0443
si*-0503
si*-0648
si*-8104

**tunicate**
Tu*-5090B

**unpaired rows**
up*-Shirer

## V. MAIZE GENOME DATABASE
http://www.agron.missouri.edu

MaizeDB, http://www.agron.missouri.edu, started in 1991 and maintained by the ARS, is an integrated genome resource for the maize genetics community. In Oct 1998, the Missouri Maize Project received funding from the NSF Plant Genome Program to support new extensions and enhancements of the database, with particular emphasis on user access. The implementation has three phases: (1) upgrading user capabilities and interfaces of MaizeDB with better data analysis tools and map viewers; (2) development of a new object-oriented framework; (3) extension and enhancement of the current MaizeDB. The NSF funding complements the ARS commitment to support the curation and serving of the production database on the WWW. We are pleased to introduce a new cooperator, Dr. Su-Shing Chen, Chair and Professor of Computer Science and Engineering, University of Missouri-Columbia. Dr. Chen is a co-PI on the NSF project and will be taking the lead in development of new enhancements to the database.

Users throughout the community will be selected to test and comment on new design tools. In addition, we strongly encourage all cooperators to take a moment and try looking up something of interest and inform us where you have difficulty finding something. Feel strongly encouraged to let us know about anything you would like to see changed or added. Send email to db_request@teosinte.agron.missouri.edu or use the email link on our home page.

**New Design 1998**

1. Stock query form specific for MGCSC (Maize Genetics Cooperation Stock Center). This form now retrieves only Stocks available from the Stock Center. The search options are limited to a selected group, many of which are provided in menu format, rather than 'type into the blanks'.

2. SSR (Simple Sequence Repeats or microsatellite) tabulations (browse only, real time, linked to database for details). A copy of the tabulation, as extracted April 1999, is appended.

3. Person queries by name and city only. This form is a simplified version of the Person 'all attributes' query page, and retrieves only the address and phone information. It is linked to the full MaizeDB record for other information, such as references authored. We would greatly appreciate if you would check your entry and provide any updates or corrections to us.

4. Reorganization of pages so that form queries and browse lists are listed together by category of information, in addition to the options for style of query. For example, touching on Loci/Variations accesses query forms for all attributes, a simplified Locus query form, and various browsable listings of mutant Images, the MNL Gene List and nomenclature guidelines. Touching on Stocks/Probes will lead to the Stock center and its Catalogs and various Stock searching styles which include the full text of the entire database, any attribute, and names only. The Probes section of this page leads to lists of the Core markers, the SSR tabulations (see above), probes available from the UMC RFLP laboratory, and the various searching styles: full text, any attribute, names only.

**Design Plans for the coming year.**

1. Table making. Current users of MaizeDB may access a few tables but the columns allowed are predetermined. Examples include map scores by the bin; the gene list; SSR compilations; the core marker table; the stock center catalog. Typical form searches where the user may specify attributes will retrieve a list of names only. A table-making tool, with menu derived options, would permit additional columns to be selected, for example phenotypes and SSR probes, and map coordinates.

2. Graphical mapviewer. Current map displays are text only, and do not permit custom or comparative map displays. We plan to develop a new utility, building on current WWW map display software; it will extract data in real time, and support robust query, display and print options.

3. Interface for data entry by the community. Community includes the expert curator, such as at the Stock Center, high through-put projects in maize, and Cooperators at large. Stock Center curation is provided direct access to the entire database, while the Cooperator at large, updating address information for example, might have more limited access to the database.

Mary Polacco, Curator MaizeDB

**PCR Primer Pairs for Mapped Simple Sequence Repeats (SSR) or Microsatellites**
**http://www.agron.missouri.edu/ssr.html**

There are 578 pairs for 541 loci. **Bins** are named by the chromosome number, followed by a decimal and a numeric identifier. Caveat: order within a bin may not be inferred from the table. Certain SSR from the Source, Acemaz, where they may be listed numerically only, have a prefix '*bnlg*', rather than '*bngl*', to avoid 'l'- '1' confounding. **At** refers to an RFLP, gene or gene candidate marked by the SSR. **Sources** for the data: Acemaz, http://burr.bio.bnl.gov/acemaz.html; Emily Chin while at Pioneer, now at Garst; Keith Edwards, Long Ashton, UK; Mike McMullen, Columbia MO; Lynn Senior while at NC State, now at Novartis; Graziana Taramino from Dupont.

| Bins | SSR | At | Primer Pairs | Source |
|---|---|---|---|---|
| 1 | bnlg149 | | CATCCTCCAAAAGCACTACGT<br>CAGCTGTCCGACACTTATTCTGTA | Acemaz |
| 1.00- 1.05 | mmc0092 | | GGGTGGTGGGTGGACAGTG<br>CTAAATCCACTTTCCTGTTGC | Edwards, KJ |
| 1.01 | bnlg1014 | | CACGCTGTTTCAGACAGGAA<br>CGCCTGTGATTGCACTACAC | Acemaz |
| 1.01 | bnlg1112 | | GTGAGAATCCTTCAGCGGAG<br>CTGTGGCAGATGTGGTATGG | Acemaz |
| 1.01 | bnlg1124 | | TCTTCATCTCTCTATCAAACTGACA<br>TGGCACATCCACAAGAACAT | Acemaz |
| 1.01 | bnlg1179 | | GCGATTCAGTCCGCAGTAGT<br>GTACTGAACAAACCGTGGGC | Acemaz |
| 1.01 | phi056 | *tub1* | ACGCCCAGATCTGTTCCTTCTC<br>ATGGCGGCAGGCCGATTGTT | Chin, E |
| 1.01 | phi097 | *tub1* | TGCTTCACATTCAGTCACCGTCAG<br>CCACGACAGATGATTACCGACC | Chin, E |
| 1.02 | bnlg1007 | | GATGCAATAAAGGTTGCCGT<br>ATGTGCTGTGCCTGCCTC | Acemaz |
| 1.02 | bnlg1083 | | ACAGTCTGTTGGGGAACAGG<br>CAACGCTGGTTTGTCGTTTA | Acemaz |
| 1.02 | bnlg1178 | | ACTACAGTTGAACGCCCCTG<br>GCTCATGTGCAAATGCAAGT | Acemaz |
| 1.02 | bnlg1429 | | CTCCTCGCAAGGATCTTCAC<br>AGCACCGTTTCTCGTGAGAT | Acemaz |
| 1.02 | bnlg1614 | | CCAACCCACCCAGAGGAGA<br>AGCGGGCGAGATCTTCAT | Acemaz |
| 1.02 | bnlg1627 | | CGGACGGGGGTTATTAAAAT<br>TGTGTTCGCAGAATCTCTCG | Acemaz |
| 1.02 | bnlg1803 | | GTATGCGTCGCTAGTCGTGA<br>TGTTGTCTATTGGCAACCGA | Acemaz |
| 1.02 | bnlg109 | | GCCAGCTGATGTCTGATGAACAGCACA<br>GATCGGGCCAGATTTCTCAAGTCGTCA | Acemaz |
| 1.02 | bnlg147 | | AGGAAGCTTTGGTCAAGTCTTA<br>GCTCACTCGATTTGTTGTGCTA | Acemaz |
| 1.03 | bnlg1203 | | GACCCGTCTCTCTTGAGTGC<br>GTCTGTCTGCACCCGTTTTT | Acemaz |
| 1.03 | bnlg1458 | | GAAAGGCTCGCTAGTCGCTA<br>AATTCCTATCGATCCTGGCC | Acemaz |
| 1.03 | bnlg1484 | | GTAAAAGACGACGACATTCCG<br>GACGTGCACTCCGTTTAACA | Acemaz |
| 1.03 | bnlg1866 | | CCCAGCGCATGTCAACTCT<br>CCCCGGTAATTCAGTGGATA | Acemaz |
| 1.03 | bnlg1953 | | CCTCGGAGCTCGATTTACAC<br>AACATTTAACCGCCGTCATC | Acemaz |
| 1.03 | bnlg2180 | | ACAAGGGCGTACCAACCAC<br>TGACCAGAGGCTTCCATACC | Acemaz |

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|---|---|---|---|---|
| 1.03 | bnlg2204 | | AGGCGACTTAGCTGCAGAAG<br>CGACTTTCGGTTTGGAAAAG | Acemaz |
| 1.03 | bnlg176 | | AGTTCACGTCCAGCTGAATGACAG<br>CGCGCATCGCATGCTTATCCTA | Acemaz |
| 1.03 | bnlg182 | | AGACCATATTCCAGGCTTTACAG<br>ACAACTAGCAGCAGCACAAGG | Acemaz |
| 1.03 | bnlg439 | | TTGACATCGCCATCTTGGTGACCA<br>TCTTAATGCGATCGTACGAAGTTGTGGAA | Acemaz |
| 1.03 | phi001 | *ts2* | TGACGGACGTGGATCGCTTCAC<br>AGCAGGCAGCAGGTCAGCAGCG | Chin, E |
| 1.03 | phi095 | *p1* | CCGATCGGCTTTATCACTGTTTAGC<br>ATGCACCATTCTAGCACTATAGCAACACT | Chin, E |
| 1.04 | bnlg1016 | | CCGACTGACTCGAGCTAACC<br>CCGTAACTTCCAAGAACCGA | Acemaz |
| 1.04 | bnlg1811 | | ACACAAGCCGACCAAAAAAC<br>GTAGTAGGAACGGGCGATGA | Acemaz |
| 1.04 | bnlg2238 | | TGCCACTCAAGCCTTCTTTT<br>TTCTGATTGCAGTGCAGACC | Acemaz |
| 1.05 | bnlg1832 | | GCGCCCACAACAAGTAAATT<br>CCTCATTGTAAGGGGCAGAA | Acemaz |
| 1.05 | bnlg1886 | | TCTCTCTCACATGCACGCC<br>TTTGATTTGGGGAACCAGAG | Acemaz |
| 1.05 | bnlg2086 | | CGGAACCTGCTGCAGTTAAT<br>GAGATGCAGGAATGGGAAAA | Acemaz |
| 1.05 | bnlg2295 | | CGGAGGAGTGGTTCTTGAAA<br>GGTTAGTGAAAGGGTTGCCA | Acemaz |
| 1.05 | bnlg652 | | CGCACGTCGGGAGAGAGGGAGA<br>GCCGCAAACATAGCCGCCAAAAAT | Acemaz |
| 1.06 | bnlg1023 | *bnlg1023b* | CGGACGATTGAAAAGGAAAA<br>TTGCAAGGGTCATTCGTAGT | Acemaz |
| 1.06 | bnlg1041 | | ATCATCTTCCACCTCGTTCG<br>CGCTATCTTCCCTTCCCTCT | Acemaz |
| 1.06 | bnlg1057 | | TTCACCGCCTCACATGAC<br>GCAACGCTAGCTAGCTTTG | Acemaz |
| 1.06 | bnlg1273 | | AAACACCAAACGTCACGTGG<br>GGCGACGAGATACAGGATGT | Acemaz |
| 1.06 | bnlg1556 | | ACCGACCTAAGCTATGGGCT<br>CCGGTTATAAACACAGCCGT | Acemaz |
| 1.06 | bnlg1598 | | GGCAAGATTCGGACCAGG<br>CGGTTAGGAGCAGTACGTCA | Acemaz |
| 1.06 | bnlg1908 | *bnlg1908b* | TCAGGCAGCAATGTTCAGAC<br>TGGAGTAGCTCACGTTGACG | Acemaz |
| 1.06 | bnlg2057 | | CAGCAGAACCTGTGGACAGA<br>TGCATACTTGAGGATCGGAG | Acemaz |
| 1.06 | bnlg421 | | GGGGCAAGGACTTGTCGGT<br>AGCCAGTTGCCCAGCATCT | Acemaz |
| 1.06- 1.12 | mmc0011 | | ACATTCATAATCAGCACCGAG<br>TTCAGGCCTCGTGATGACATG | Edwards, KJ |
| 1.06- 1.12 | mmc0031 | | AGATTCAGGCCTCGTGATGAC<br>CCACCGTTTTCGCGGTTGGTT | Edwards, KJ |
| 1.06 | umc1035 | | CTGGCATGATCACGCTATGTATG<br>TAACATCAGCAGGTTTGCTCATTC | McMullen, MD |
| 1.07 | bnlg1025 | | TGGTGAAGGGGAAGATGAAG<br>CCGAGACGTGACTCCTAAGC | Acemaz |
| 1.07 | bnlg1564 | | ACGGGAGAACAAAAGGAAGG<br>CTCTCCCTCACATCCGCC | Acemaz |

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|---|---|---|---|---|
| 1.07 | bnlg257 | | TCGAGAGACGAGCGTTTGAATGCT<br>GCTCTGAGGTTTTCATACGGGGTT | Acemaz |
| 1.07 | bnlg615 | | CTTCCCTCTCCCCATCTCCTTTCCAA<br>GCAACCTGTCCATTCTCACCAGAGGATT | Acemaz |
| 1.08 | bnlg1044 | | GACTCTCCAGTCTCCGTTGC<br>ACATGAAAACGAGCAATGCA | Acemaz |
| 1.08 | bnlg1629 | | GTTGGATGGAAAATTCTAGATCG<br>TTGCGTCATTACAGCAGGAG | Acemaz |
| 1.08 | bnlg1643 | | ACCACCGTCCACCTCCAC<br>ATTGACCCCGTGACCCTC | Acemaz |
| 1.08 | bnlg2228 | | GCAGCAATCGACACGAGATA<br>CTTGGATCGCACTCCGTC | Acemaz |
| 1.08 | dupssr12 | | CAGGTACTACGTGCCGTG<br>CTAGAGACAAACGAGGCTAGG | Taramino, G |
| 1.08 | mmc0041 | | AGGACTTAGAGAGGAAACGAA<br>TTTATCCTTACTTGCAGTTGC | Edwards, KJ |
| 1.08 | phi002 | *umc128* | AAAAGGCCGTCAGAGCAGAACTGA<br>GTGACCGTGCCGTTGTATCACAA | Chin, E |
| 1.08 | phi037 | *umc128* | CCCAGCTCCTGTTGTCGGCTCAGAC<br>TCCAGATCCGCCGCACCTCACGTCA | Chin, E |
| 1.08 | phi038 | *umc128* | TCAGACTCCGCCCAGCAATCATCTG<br>AGCCTAGTGCTTATCTTGAAGGCTT | Chin, E |
| 1.08 | phi039 | *umc128* | ACCGTGTCTAATGTGTCCATACGG<br>CGTTAGGAGCTGGCTAGTCTCA | Chin, E |
| 1.09 | bnlg1268 | | TCCACGGTGACTGTAGAACG<br>CACTTCCCCCAGATCATTTG | Acemaz |
| 1.09- 1.10 | bnlg1331 | | TGGTGATAACTGTCAAGCGC<br>TTGGGGCATTGGCCTATATA | Acemaz |
| 1.09- 1.10 | bnlg1502 | | AGGTCCTGGCACTAAGAGCA<br>AGAGGTGGTATGATCACCTGG | Acemaz |
| 1.09- 1.10 | bnlg1597 | *bnlg1597a* | GATAATCTCGTCTCGCCAGG<br>CATAAAAGGATGCCGACGAC | Acemaz |
| 1.09- 1.10 | bnlg1720 | | CAACCCGGATGTCTCAAGTT<br>TTCGATGCGTATGTACTCAGC | Acemaz |
| 1.09- 1.10 | bnlg100 | | TGCACGCACGGGCACTGAAC<br>TAAGACATCTATGGCCACCGGAG | Acemaz |
| 1.09 | bnlg400 | | AGCTGTGACTGTGAAGGGAAAA<br>CGTCACACCGCTGTTTCTTG | Acemaz |
| 1.09 | phi011 | *glb1* | GAGCTTCAGCAAGAGCATCCAG<br>CAACGCGATCGATGTGAGCACA | Chin, E |
| 1.09 | phi055 | *glb1* | GAGATCGTGTGCCCGCACC<br>TTCCTCCTGCTCCTCAGACGA | Chin, E |
| 1.09 | phi094 | *glb1* | AAAGAGGAGGAACGCGAAGGAC<br>TCACATCCTGGCGGTCACCA | Chin, E |
| 1.1 | bnlg1347 | | GTGGTCACGACGAAATCCTT<br>TTGCAATCACACAGGTGGTT | Acemaz |
| 1.1 | bnlg1671 | | TCACGATCAGCAAGCAATTC<br>CCCCACCAACCTTAGAGTCA | Acemaz |
| 1.11 | bnlg1055 | | GCTGGATGGCAGGTACAGAG<br>TGCAATGGAGAAGCAACAAG | Acemaz |
| 1.11 | bnlg2123 | | TGATGCAGACAAGTCCTTCG<br>ACAAATCTCACCTCTGCGCT | Acemaz |
| 1.11 | bnlg2331 | | TCTGATATCATAAAGGAGGACCG<br>GGAGCTTGCGCTTTTTAACA | Acemaz |
| 1.11 | bnlg131 | | CTCTGCGCTACCTTTCTGAGTC<br>GCGGAATCCTTGTGTTCTTG | Acemaz |

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|---|---|---|---|---|
| 1.11 | bnlg504 | | CGGCAGCTCCAGCACCGGCAT<br>AGTGTCCACATACCGCCACACACGTTT | Acemaz |
| 1.11 | phi064 | | CCGAATTGAAATAGCTGCGAGAACCT<br>ACAATGAACGGTGGTTATCAACACGC | Chin, E |
| 1.11 | phi120 | | GACTCTCACGGCGAGGTATGA<br>TGATGTCCCAGCTCTGAACTGAC | Chin, E |
| 2.00- 2.04 | mmc0063 | | ACACCCCTATCCAACATAAAT<br>TGCAAAATTAATTGATACATAG | Edwards, KJ |
| 2.01 | bnlg1092 | | TATTCTGGTCAAGTTGGGGC<br>GCTTGATCTCCAATCCTTGC | Acemaz |
| 2.01 | bnlg1338 | | GTGCAGAATGCAGGCAATAG<br>GCAAATGTTTTCACACACACG | Acemaz |
| 2.02 | bnlg1017 | | ATTGGAAGGATCTGCGTGAC<br>CAGCTGGTGGACTGCATCTA | Acemaz |
| 2.02 | bnlg1297 | | TCTCGATCGCTCCGATCTAT<br>GACTCAACTCCAAAAGGCGA | Acemaz |
| 2.02 | bnlg1302 | | GGATAATGGCATTTTTTTAAACC<br>TTGTTGGTGATAAAAGGGGC | Acemaz |
| 2.02 | bnlg1327 | | TCTCTCTCGCGTGTGTGC<br>TGGGTCTCCTTCTCCGTCTA | Acemaz |
| 2.02 | bnlg2042 | | TGTCGCGTACTCGCATTTAG<br>TTTGATTGGTGATCTCGCAG | Acemaz |
| 2.02 | bnlg2277 | | TTACGGTACCAATTCGCTCC<br>GACGACGCCATTTTCTGATT | Acemaz |
| 2.02 | bnlg125 | | GGGACAAAAGAAGAAGCAGAG<br>GAAATGGGACAGAGACAGACAAT | Acemaz |
| 2.02 | bnlg469 | *bnlg469b* | AGGGTGTACAGGTCCAAGTCCAA<br>AATGTGGGTCGTCAGCCATCAG | Acemaz |
| 2.02 | phi098 | | GAGATCACCGGCTAGTTAGAGGA<br>GTATGGTTGGGTACCCGTCTTTCTA | Chin, E |
| 2.03 | bnlg1064 | | CTGGTCCGAGATGATGGC<br>TCCATTTCTGCATCTGCAAC | Acemaz |
| 2.03 | bnlg1537 | | CTGGAAACTGTTGCCTAGCC<br>TTTCCCTTACCCCCAAACTC | Acemaz |
| 2.03 | bnlg1621 | *bnlg1621b* | CTCTTCGATCTTTAAGAGAGAGAGAG<br>ACACGAGGCACTGGTACTAACG | Acemaz |
| 2.03 | bnlg2248 | | CCACCACATCCGTTACATCA<br>ACTTTGACACCGGCGAATAC | Acemaz |
| 2.03 | bnlg469 | *bnlg469b* | AGGGTGTACAGGTCCAAGTCCAA<br>AATGTGGGTCGTCAGCCATCAG | Acemaz |
| 2.03 | bnlg381 | | TCCCTCTTGAGTGTTTATCACAAA<br>GTTTCCATGGGCAGGTGTAT | Acemaz |
| 2.03- 2.04 | umc1026 | | TCGTCGTCTCCAATCATACGTG<br>GCTACACGATACCATGGCGTTT | McMullen, MD |
| 2.04 | bnlg1018 | | CGAGGTTAGCACCGACAAAT<br>CGAGTAAATGCTCTGTGCCA | Acemaz |
| 2.04 | bnlg1175 | | ACTTGCACGGTCTCGCTTAT<br>GCACTCCATCGCTATCTTCC | Acemaz |
| 2.04 | bnlg1613 | | GGGGATGATTCCGATAGGC<br>GCGCTCTCTTTTCCCTCTCT | Acemaz |
| 2.04 | bnlg1818 | | GATGCTGGATGGAGATCGTT<br>CTTAGTATCTAATTGAACAGTTCTCTCTC | Acemaz |
| 2.04 | bnlg108 | | GCACTCACGCGCACAGGTCA<br>CGCCTGCCAAGGTACATCAC | Acemaz |
| 2.04 | bnlg166 | | GCCAACGTTTCCAGCCTGA<br>CTCCGTTTGCCCGAGTCC | Acemaz |

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| 2.04 | phi083 | *prp2* | CAAACATCAGCCAGAGACAAGGAC<br>ATTCATCGACGCGTCACAGTCTACT | Chin, E |
| 2.04- 2.05 | umc1003 | *zpu1* | AATAGATTGAATAAGACGTTGCCC<br>TGTTCCAATGCTTTTGTACCTCTA | McMullen, MD |
| 2.05 | bnlg1036 | | GGGAGTATGGTAGGGAACCC<br>AAACCCTTGGAGCATACCCT | Acemaz |
| 2.05 | bnlg1047 | *bnlg1047c* | ATGGAGATGGAGGAGAGAGAGA<br>GATGCGGCGATGGCTAA | Acemaz |
| 2.05 | bnlg1063 | *bnlg1063b* | GGAGACAACCCCGACGAC<br>GGTACCAGAGCCACAGATCC | Acemaz |
| 2.05 | bnlg1831 | | TCGCTCATTTGCATACACCT<br>TAGGAACATGCCAGCAGTTG | Acemaz |
| 2.05 | bnlg1893 | | AATCCTGTAGCGTGTGTCCC<br>TAACTGAGTTGTTGAAGGAAATTG | Acemaz |
| 2.05 | bnlg1909 | | CCTGACCCTGTTCCTGAAAA<br>GTGTGTCTGGAGCTGTTCGA | Acemaz |
| 2.05 | bnlg1914 | | ATGCAACATTTCGTGATCCA<br>GATTTTTCTAGCACTCGCGC | Acemaz |
| 2.05 | bnlg2039 | | ATTTTAGGCTCGGCATGATG<br>GCGAGATGCTTTTTAATGGG | Acemaz |
| 2.05 | bnlg2328 | *bnlg2328a* | AGCAGTGAGGAAGAAGCAGG<br>TTACCCTCCCTTGTCGTGAC | Acemaz |
| 2.05 | bnlg180 | | CTAGAGCCTTCGTCGCAGAG<br>AACGGCGGCGAGATAAAAT | Acemaz |
| 2.05 | bnlg371 | | CAACGCGAAGCAGAGATAAAA<br>TCGTCGCATGACCATAGTAGC | Acemaz |
| 2.05 | dupssr21 | | GTGCAAACTAATCCAAAGCAA<br>ATGTAGGGACAAAGGAATAAATCA | Taramino, G |
| 2.05 | nc131 | *isu89* | TTTCTTCGATCCCATGTCAC<br>TAGTGTGCTAGAACGTGCGC | Senior, L |
| 2.05 | nc132 | *isu89* | TCATCTTGCTCTGATGCTCG<br>TGTGGGGGCACGTTAATTAC | Senior, L |
| 2.05 | nc133 | *isu89* | AATCAAACACACACCTTGCG<br>GCAAGGGAATAAGGTGACGA | Senior, L |
| 2.05 | umc1020 | *pmg1* | CCTGGAGAGCCACTACAAGGAA<br>TCAGCCTGAGCTCACATCATCT | McMullen, MD |
| 2.05 | umc1028 | | CCCAGGTAAAATTCGCTAGCCT<br>GGAACAAGGAAAGCTGAATACACG | McMullen, MD |
| 2.06 | bnlg1138 | | TGCTCTAGCCGACCTCAATT<br>ATGCCTGAACCGTGATTAGG | Acemaz |
| 2.06 | bnlg1184 | | CCATCTAGAGCCGAGCAAAC<br>TTGAAGCTCCTAGATCCCGA | Acemaz |
| 2.06 | bnlg1225 | | GCAGTAGAAGAGCGAGCGAG<br>CATACGCTGTCACTGCCACT | Acemaz |
| 2.06 | bnlg1396 | | CGCATTTCTCCTGCAGTACA<br>TGCTTGAGTCGTCGAATCTG | Acemaz |
| 2.06 | bnlg1887 | | CGAACCACTGTAGGCATGTG<br>ATCATGCAGAGCAGATGCAG | Acemaz |
| 2.06 | bnlg121 | | AGTTCTACAGGCTTCTTGTCCAA<br>CTATAAAGAAGGTAACTGGTTGCTC | Acemaz |
| 2.06 | nc003 | | ACCCTTGCCTTTACTGAAACACAACAGG<br>GCACACCGTGTGGCTGGTTC | Senior, L |
| 2.07 | bnlg1045 | | TCCCCGATAGCATATCGATC<br>GTGACTTTGGGGAGTTTGGA | Acemaz |
| 2.07 | bnlg1413 | | CTGATTGGATTACATAAAGTTCAGC<br>AAGTTGATGGCTTGGACACC | Acemaz |

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| 2.07 | bnlg1633 | | GTACCTCCAGGTTTACGCCA<br>TCAACTTCTCATGCACCCAT | Acemaz |
| 2.07- 2.08 | mmc0191 | | GGTGTTCAGTGTGAAAGGTTA<br>AAGATTTCCGCAAGGTTAAAC | Edwards, KJ |
| 2.07 | mmc0271 | | CGTAATGCGTAGCAACATAG<br>CAACATCCTTTCCACCG | Edwards, KJ |
| 2.08 | bnlg1140 | | TAGGCCATATTGGCCCATG<br>AATGCCGTGGACGTAAGATC | Acemaz |
| 2.08 | bnlg1141 | | GAACTGGATTCCATCATCGG<br>AGGCTCAGCTGGCATTTAGA | Acemaz |
| 2.08 | bnlg1169 | | CTAAGCTAGACACGGCCCTG<br>GATCTCGTCCCGATTTTCAA | Acemaz |
| 2.08 | bnlg1233 | | GAACACCAGAGGAGAGTGGG<br>TTCACTTGTCCACCACTGGA | Acemaz |
| 2.08 | bnlg1258 | | GGTGAGATCGTCAGGGAAAA<br>GAGAAGGAACCTGATGCTGC | Acemaz |
| 2.08 | bnlg1267 | | AAATCTGTGCTGTGCTGTGG<br>TGTCGAGTGGTCCTACGATG | Acemaz |
| 2.08 | bnlg1316 | | CGAAACAGAGCCCAAAAGAC<br>GATCCGCGTCTAGCCCCT | Acemaz |
| 2.08 | bnlg1329 | | ATAGAATGGGATGTGGGCAA<br>TCCGATCATATCGGGAGATC | Acemaz |
| 2.08 | bnlg1335 | | GAAGGTTGCTCTTCCACTGG<br>TGGTTTGTGCAAGTGTCACC | Acemaz |
| 2.08 | bnlg1606 | | TGTCCTTGTACCAGTGCTGC<br>GCTGTTCAGGATCTTCTGCC | Acemaz |
| 2.08 | bnlg1662 | | GCACCCACATGAAGTATCCC<br>TTGTTTTTGCAGTGCCTCAG | Acemaz |
| 2.08 | bnlg1721 | | ACGACTTTCATGCCTCGTCT<br>ATTTCTTTTGCCACCTCAGC | Acemaz |
| 2.08 | bnlg1746 | | ACAGCTTTAGACTTAGACCACACG<br>GCACAAGCGAAGGTTTTCTC | Acemaz |
| 2.08 | bnlg1767 | | AATTTCACGGTAGGGACACG<br>AATCCGCGTGTTTTCATAGG | Acemaz |
| 2.08 | bnlg1908 | *bnlg1908a* | TCAGGCAGCAATGTTCAGAC<br>TGGAGTAGCTCACGTTGACG | Acemaz |
| 2.08 | bnlg1940 | | CCTTTTGTTTCAGGCCGTTA<br>CAGCAGCCTGATGATGAACA | Acemaz |
| 2.08 | bnlg2077 | | GACCAGAGGATGGGGAAATT<br>GTAGGCACATGCACATGAGG | Acemaz |
| 2.08 | bnlg2144 | | TCTGGGTGTGCTTGCTCTC<br>TGTTCTCAGCATTCCCAACA | Acemaz |
| 2.08 | bnlg198 | | GTTTGGTCTTGCTGAAAAATAAAA<br>GCTGGAGGCCTACATTATTATCTC | Acemaz |
| 2.08 | dupssr24 | | ACTGCACTGCACCTCTCTC<br>ACACAACGGCTTCTAACCTT | Taramino, G |
| 2.08 | dupssr25 | | TGTTCACTTGTCCACCACTG<br>GGAAGCACATAAACTATCTCGG | Taramino, G |
| 2.08 | phi090 | *npi298* | CTACCTATCCAAGCGATGGGGA<br>CGTGCAAATAATTCCCCGTGGGA | Chin, E |
| 2.08 | phi127 | | ATATGCATTGCCTGGAACTGGAAGGA<br>AATTCAAACACGCCTCCCGAGTGT | Chin, E |
| 2.09 | bnlg1520 | | TCCTCTTGCTCTCCATGTCC<br>ACAGCTGCGTAGCTTCTTCC | Acemaz |
| 3.00- 3.04 | mmc0022 | | AGGTGTTGTTTTTGTTCGCT<br>TGCTTGTTTAAGCTCATTATT | Edwards, KJ |

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| 3.01 | phi049 | *umc32a* | CTTCTGTTCCGCCATCCAGTATGTT<br>GATTGCGATAACATTGCGGCAAGTTGT | Chin, E |
| 3.03 | bnlg1144 | | TACTCGTCGTGTGGCGTTAG<br>AGCCGAGGCTATCTAACGGT | Acemaz |
| 3.03 | bnlg1325 | | CTAAATGCGCAGCAGTAGCA<br>TGCTCTGCAACAACTTGAGG | Acemaz |
| 3.03 | bnlg1523 | | GAGCACAGCTAGGCAAAAGG<br>CTCGCACGCTCTCTCTTCTT | Acemaz |
| 3.03 | phi036 | *umc59e* | CCGTGGAGAGACGTTTGACGT<br>TCCATCACCACTCAGAATGTCAGTGA | Chin, E |
| 3.04 | bnlg1019 | *bnlg1019a* | ACCATAGTTGGACGGACCAC<br>ACCACAACACAGACGAGCAC | Acemaz |
| 3.04 | bnlg1447 | | GAGAGGAGAGGCTGAGCTGA<br>TCCTCCCACTGAATTTCCAC | Acemaz |
| 3.04 | bnlg1452 | | CTCCTCTCCTCCACGATCAC<br>CGCAAACGATCTCTGACCTT | Acemaz |
| 3.04 | bnlg1628 | | GTAGGGTTCAAGGAGGCACA<br>CTCTCTGGTGAGCTGGCTTT | Acemaz |
| 3.04 | bnlg1638 | | CATATCTCTAGCTTCTCGTCTTCG<br>ACACCGATCGAGGAAGAATG | Acemaz |
| 3.04 | bnlg1647 | | CGTCGTCTGTGGACGTACTG<br>AGAAGCTCACAAGCCTGCTC | Acemaz |
| 3.04 | bnlg1904 | | AGGAGCATGCACTTGGTTCT<br>ACTCAACTGATGGCCGATCT | Acemaz |
| 3.04 | bnlg2047 | | CATGCATCCATCCTTTTCCT<br>ATCCATCGGCAACTACAAGC | Acemaz |
| 3.04 | bnlg2136 | | TGCTCCTTCTCGAGCACC<br>ATGGACGTACGGCAGACTCT | Acemaz |
| 3.04 | bnlg602 | | CCCGATAGCCAAGCTCTCGCCAA<br>AGCTCGTGGACCGAACAAGCCCA | Acemaz |
| 3.04 | nc030 | *tpi4* | CCCCTTGTCTTTCTTCCTCC<br>CGATTAGATTGGGGTGCG | Senior, L |
| 3.04 | phi029 | *tpi4* | TTGTCTTTCTTCCTCCACAAGCAGCGAA<br>ATTTCCAGTTGCCACCGACGAAGAACTT | Chin, E |
| 3.04 | umc1025 | | GCTCCACTTCCACCCTGATATG<br>CGCTAATGTCCCCATTGATGAT | McMullen, MD |
| 3.04 | umc1030 | | TCCAGAGAATGAGATGACAAGACG<br>CAGAATAACAGGAGATGAGACGCA | McMullen, MD |
| 3.05 | bnlg1022 | | GTGTTGTCGATCCACTCCCT<br>GCAAAGATCTGTGAGGGGAC | Acemaz |
| 3.05 | bnlg1035 | | TGCTTGCACTGTCAGGAATC<br>CAGCTCTGACACACCACACA | Acemaz |
| 3.05 | bnlg1113 | | GCTCCCAACTTTCAGAATCG<br>TTCTCCCTTTTTTTCGCAGA | Acemaz |
| 3.05 | bnlg1117 | | GGCCGGGCTCAATTTATAAT<br>CCTTCTTCAACCCTCCTTCC | Acemaz |
| 3.05 | bnlg1246 | *bnlg1246b* | CGCAGGCCGGGGAA<br>CCTGGCGCCCAACC | Acemaz |
| 3.05 | bnlg1399 | | ATTTTAGTGCCGCCGTGTAT<br>TGCATGCATTTCTTTTTTGC | Acemaz |
| 3.05 | bnlg1456 | | CTCTAGGTGGTTAAGATTAACTCATT<br>TTCATGAGGACCGTGTTGAA | Acemaz |
| 3.05 | bnlg1505 | | GAAAGACAAGGCGAAGTTGG<br>GCTTCTGAACTGGATCGGAG | Acemaz |
| 3.05 | bnlg1957 | | CTCTGCTTTCCTCGGCTTTA<br>CTCAAATCACCCGCAGCG | Acemaz |

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| 3.05 | bnlg420 | | CTTGCGCTCTCCTCCCCTT<br>GGCCAGCTCACTGCTCACT | Acemaz |
| 3.05- 3.10 | mmc0071 | | TTACGGACAAGACGCTACTAC<br>ATACGTTTCGGCCAATCTCCT | Edwards, KJ |
| 3.05 | phi053 | *umc102* | CTGCCTCTCAGATTCAGAGATTGAC<br>AACCCAACGTACTCCGGCAG | Chin, E |
| 3.05 | phi073 | *gst4* | TTACTCCTATCCACTGCGGCCTGGAC<br>GCGGCATCCCGTACAGCTTCAGA | Chin, E |
| 3.06 | bnlg1047 | *bnlg1047a* | ATGGAGATGGAGGAGAGAGAGA<br>GATGCGGCGATGGCTAA | Acemaz |
| 3.06 | bnlg1063 | *bnlg1063a* | GGAGACAACCCCGACGAC<br>GGTACCAGAGCCACAGATCC | Acemaz |
| 3.06 | bnlg1449 | | AGTCAACGTAGCTGGCGAGT<br>TTCACGACGGGTCTCTCTCT | Acemaz |
| 3.06 | bnlg1601 | | CAGACCAGAGACCATCTGCA<br>ATCGTGCGCTAGTCCAGAGT | Acemaz |
| 3.06 | bnlg1796 | | GATGTCTGCCTCCCTTGAAG<br>AAGGACCCACCACTACAACG | Acemaz |
| 3.06 | bnlg1798 | | AAGTTGGTGGTGCCAAGAAG<br>AAAAGGTCCACGTGAACAGG | Acemaz |
| 3.06 | bnlg2241 | | GTGCACACTCTCTTGCATCG<br>TAGTCAGCATCTGCCGTGTC | Acemaz |
| 3.06 | dupssr23 | | TGATCATCATAAGCACACCG<br>CCAATGTGAAGCAAGAGAGAA | Taramino, G |
| 3.07 | bnlg1160 | | AATACTGGACCACCAGGCAC<br>CGTGGGTCACCAGGAGTC | Acemaz |
| 3.07 | bnlg1605 | | TCCTGCCCCCTTTGTTTTC<br>CACCTCTGAACCCCTGTGTT | Acemaz |
| 3.07 | bnlg1779 | | CCCTTTTATATCTCAAGTGTAGAACC<br>AGAGCACCCACCACGATAAC | Acemaz |
| 3.07 | bnlg1931 | | GGGATGCTCGTAGTAGGGGT<br>ACGCACACAACAAAGAGACG | Acemaz |
| 3.07 | bnlg1951 | | CAAGCGTTCTGGTTTTCACA<br>ATTGCCGTTCTCAAAACGAG | Acemaz |
| 3.07 | bnlg197 | | GCGAGAAGAAAGCGAGCAGA<br>CGCCAAGAAGAAACACATCACA | Acemaz |
| 3.08 | bnlg1108 | | GGATTCCTTTATGACGGGGT<br>AGTAACAACCAAGGCATCGG | Acemaz |
| 3.08 | bnlg1350 | *bnlg1350a* | TGCTTCAGCGCATTAAACTG<br>TGCTCGTGTGAGTTCCTACG | Acemaz |
| 3.08 | bnlg2243 | | ATCTATCACGACGAACGGGA<br>ATCTCCCTAGCTCGCTCTCC | Acemaz |
| 3.08 | phi046 | *npi257* | ATCTCGCGAACGTGTGCAGATTCT<br>TCGATCTTTCCCGGAACTCTGAC | Chin, E |
| 3.08 | phi088 | *npi432* | GATTGCGATAAGCATTGCGGCAGTT<br>CTTCTGTTCCGCCATCCAGTATGT | Chin, E |
| 3.09 | bnlg1182 | | AGCCGAGTCAGTTCGAGGTA<br>CAGGGGCTTGAGGTGAGTTA | Acemaz |
| 3.09 | bnlg1257 | | CGGACGATCTTATGCAAACA<br>ACGGTCTGCGACAGGATATT | Acemaz |
| 3.09 | bnlg1496 | | CTGGGCAGACAGCAACAGTA<br>AGCCAAAGACATGATGGTCC | Acemaz |
| 3.09 | bnlg1536 | | CAAAAAAAAAATATGTATACGGGG<br>ATGCACGAGCTTTTGGAGTT | Acemaz |
| 3.09 | bnlg1754 | | CCATCGCTGTACACATGAGG<br>TACCCGAAGGATCTGTTTGC | Acemaz |

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| 3.09 | mmc0001 | | ATTGAGAAGATGAGAACCGTC<br>CCTACAAATGCAACAAATGCT | Edwards, KJ |
| 3.09 | phi047 | *npi425a* | GGAGATGCTCGCACTGTTCTC<br>CTCCACCCTCTTTGACATGGTATG | Chin, E |
| 3.1 | bnlg1098 | | GGCGCAGAGAGAGAAGAAAG<br>GTTGGCGCCAGTTTTTCTCT | Acemaz |
| 4 | bnlg1370 | | TATTTAATTTAGTGTGGAGCTCACG<br>CGAGGGTCAGTTGTTGCTCT | Acemaz |
| 4 | bnlg372 | | TTCACATGCCATCCTCCTATAT<br>TATCCCTCTCTGATCACGTTGG | Acemaz |
| 4.01 | bnlg1241 | | ATTCTTGACATCCATCCGGT<br>TGTTGTTTCACTCAGCGTCC | Acemaz |
| 4.01 | bnlg1318 | | TTATGTGTGCAGAACGACTCG<br>AGCATGGCAGAGAAGGTGAT | Acemaz |
| 4.01 | bnlg1434 | | TCCAAGCTGGAAGCCTTAAG<br>TCTTGTCTCCTCTCTCCCCC | Acemaz |
| 4.01 | nc135 | *umc123* | CACAAAGAGCAGCCCACTTT<br>AAGTTGCTGACATCGATCCA | Senior, L |
| 4.01 | phi072 | *mtl1* | ACCGTGCATGATTAATTTCTCCAGCCTT<br>GACAGCGCGCAAATGGATTGAACT | Chin, E |
| 4.01 | umc1011 | *mtl1* | TCTAGCTTGTGGTGGTGGTTGA<br>ACATGAGCACAAAGACTGACGC | McMullen, MD |
| 4.01 | umc1017 | *cyp3* | GAAGAGGTAAGGACGACGACGA<br>GCACCTGCAGTGAACGTCAGTA | McMullen, MD |
| 4.01 | umc1022 | *trp1* | AACAAGTTTTGTTTGACAAGCCG<br>ATGATCACCCCGTCAGCG | McMullen, MD |
| 4.03 | bnlg1126 | | GAGATCGAAGGTCATGGCAC<br>ATGGTTCCTGGTTCAGATGG | Acemaz |
| 4.03 | bnlg1162 | | CATAGCAACAAGGACCCTACG<br>CGTCCTAGTGGAACCAGGAA | Acemaz |
| 4.03 | nc004 | *adh2* | TGCGAAGAAGCAGTAGCAAA<br>TGGAGGTAGAAGACGCACG | Senior, L |
| 4.03 | phi021 | *adh2* | TTCCATTCTCGTGTTCTTGGAGTGGTCCA<br>CTTGATCACCTTTCCTGCTGTCGCCA | Chin, E |
| 4.04 | phi074 | *zp22.1* | CCCAATTGCAACAACAATCCTTGGCA<br>GTGGCTCAGTGATGGCAGAAACT | Chin, E |
| 4.04 | phi096 | *zp1* | CAACAATGTCGTCGTCGCTCTATC<br>GACGACCGTTGAAACTGGTGCTTT | Chin, E |
| 4.05 | bnlg1159 | | GTGTGCCTATCCTTCCGAGA<br>AAGGACGTCAACAACGAACC | Acemaz |
| 4.05 | bnlg1168 | | CGATAAGTTAGGGACGGCTG<br>CGCTCACTCCCTCTCTCTCT | Acemaz |
| 4.05 | bnlg1217 | | AGCTGATCTGCACGTTGTTG<br>GCAGATCCACGCCATTTAAA | Acemaz |
| 4.05 | bnlg1265 | | GGTTGTCCGTAAAGGCAAGA<br>TGTGAAGGCCAGACAGTCAG | Acemaz |
| 4.05 | bnlg1729 | | GACATCTGTGCCCAACAATG<br>CCAATCCCGACAGGTTCTGT | Acemaz |
| 4.05 | bnlg1755 | | CCTAGTAGACCTCACCGCCA<br>GGAGTTCACCGATGGCAC | Acemaz |
| 4.05- 4.06 | bnlg1937 | | AATGCTCGGTCCACAGAATC<br>AACTGGAGCCAAAAGTGGTG | Acemaz |
| 4.05 | bnlg490 | | GCCCTAGCTTGCTAATTAACTAACA<br>ACTGTAAGGGCAGTGGACCTATA | Acemaz |
| 4.05 | bnlg667 | | CGTGGATGTAAGGGGGCGCGCT<br>GGCCGCTGCTCAACACAGGCAG | Acemaz |

| 4.05 | nc005 | *gpc1* | CCTCTACTCGCCAGTCGC<br>TTTGGTCAGATTTGAGCACG | Senior, L |
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| 4.05 | phi026 | *gpc1* | TAATTCCTCGCTCCCGGATTCAGC<br>GTGCATGAGGGAGCAGCAGGTAGTG | Chin, E |
| 4.05 | phi079 | *gpc1* | TGGTGCTCGTTGCCAAATCTACGA<br>GCAGTGGTGGTTTCGAACAGACAA | Chin, E |
| 4.05 | umc1031 | | TTGGGTTCATACCTCCTAGGAACA<br>ACGTGGACAACCAGTCTATCAACA | McMullen, MD |
| 4.06 | bnlg1023 | *bnlg1023a* | CGGACGATTGAAAAGGAAAA<br>TTGCAAGGGTCATTCGTAGT | Acemaz |
| 4.06 | bnlg1621 | *bnlg1621a* | CTCTTCGATCTTTAAGAGAGAGAGAG<br>ACACGAGGCACTGGTACTAACG | Acemaz |
| 4.06 | bnlg1741 | | TGCCAAATTGCCAACCTAAT<br>TCAGACGGTGCATCTGAAAC | Acemaz |
| 4.06 | bnlg252 | | CGTTCTCCGTACAGCACAGACCAACGT<br>CTCAGATGAACTCCTCAGCAGCTGTAGCCT | Acemaz |
| 4.07 | bnlg1137 | | ATGAGCTCAGTCACACTGTAGTG<br>ACTGATGACTGGTCCATGCA | Acemaz |
| 4.07 | bnlg1189 | | CGTTACCCATTCCTGCTACG<br>CTTGCTCGTTTCCATTCCAT | Acemaz |
| 4.07 | bnlg1784 | | GCAACGATCTGTCAGACGAA<br>TTGGCATTGGTAATGGGTCT | Acemaz |
| 4.07 | bnlg1927 | | TTTTTTTGTAAGCGATCCGG<br>GATGAATCTGCGTCCGTCTT | Acemaz |
| 4.07 | bnlg2291 | | CCTCTCGATGTTCTGAAGCC<br>GTCATAACCTTGCCTCCCAA | Acemaz |
| 4.07 | dupssr34 | | TCAGTGCTTTCATTGTAACGA<br>ATAAACATCTTGCCAGCAAA | Taramino, G |
| 4.08 | bnlg1444 | | GCATGGATGGAGAAAGAGGA<br>AGACGACGAAGCTTTTGCAT | Acemaz |
| 4.08 | bnlg2162 | | GTCTGCTGCTAGTGGTGGTG<br>CACCGGCATTCGATATCTTT | Acemaz |
| 4.08 | bnlg2244 | | CAGGAAAACGAAAACCCAGA<br>CTACGCGGGTCTCATCTCAT | Acemaz |
| 4.08 | dupssr28 | | GAAGGAAGCCTTTGTTACAAGT<br>CTGGAGTGCTGGTCTTGTTAT | Taramino, G |
| 4.08 | mmc0321 | | TAATCCGAGCTGCAGAG<br>CACATGAGCTGAGCATCA | Edwards, KJ |
| 4.08 | phi066 | | CCATCCTTGAGGTGGTGTGAC<br>GAAGGAGCAGTAGCACTTGGTG | Chin, E |
| 4.08 | phi086 | | TACGTCGACGAGATCACTGGTC<br>CCACCATGATGCACCCACACT | Chin, E |
| 4.08 | phi092 | *ssu1* | GTGGGGGAGCCTACTACAGG<br>GACGAGGCCATCATCACGGT | Chin, E |
| 4.08 | phi093 | *ssu1* | AGTGCGTCAGCTTCATCGCCTACAAG<br>AGGCCATGCATGCTTGCAACAATGGATACA | Chin, E |
| 4.09- 4.10 | bnlg1019 | *bnlg1019b* | ACCATAGTTGGACGGACCAC<br>ACCACAACACAGACGAGCAC | Acemaz |
| 4.09- 4.10 | bnlg1565 | | TCGGAGACGAGGCTGAAC<br>CTGGAGACGTTTGGTGTCAA | Acemaz |
| 4.09 | bnlg292 | *bnlg292b* | TGGTAGGACCTTACAATGGGA<br>CGGGAGTACTGCTACACACGA | Acemaz |
| 4.1 | bnlg1917 | | ACCGGAACAGACGAGCTCTA<br>TTTGCTTCCAACTCACATGC | Acemaz |
| 4.11 | bnlg1337 | | TCTAGAGACGGGAAAACACAAG<br>AAAGCCGGTGGATAAGAAAA | Acemaz |

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| 4.11 | bnlg1890 | | ACCGGAACAGACGAGCTCTA<br>GTCCTGCAAAGCAACCTAGC | Acemaz |
| 4.11 | bnlg2186 | | GAGAAGTGGACACACTTCAGCA<br>TTCTATCAAGTCCTAAGGACTAATCATA | Acemaz |
| 4.11 | bnlg589 | | GGGTCGTTTAGGGAGGCACCTTTGGT<br>GCGACAGACAGACAGACAAGCGCATTGT | Acemaz |
| 4.11 | phi006 | *cat3* | AGGCGGCGTGCTGAACACCT<br>CGCTTCATCTCCCGTGACAATG | Chin, E |
| 4.11 | phi019 | *cat3* | TCCGCCTTTGTACCAATACAAGCCA<br>ATCCATCTTCAGGTAGCAGGGGT | Chin, E |
| 4.11 | phi076 | *cat3* | TTCTTCCGCGGCTTCAATTTGACC<br>GCATCAGGACCCGCAGAGTC | Chin, E |
| 5 | bnlg1006 | | GACCAGCGTGTTGATCCC<br>GGAGACCCCGACTCTCTCTC | Acemaz |
| 5.00- 5.09 | mmc0081 | | TGAAATAATTCACAGCACTCC<br>TGATAGCACAACACAGCTATG | Edwards, KJ |
| 5 | mmc0151 | | AAACCATGCATCCAACRAATG<br>AGACCCAGAGATGATTTAGG | Edwards, KJ |
| 5 | nc130 | *isu62* | GCACATGAAGATCCTGCTGA<br>TGTGGATGACGGTGATGC | Senior, L |
| 5.01 | bnlg1382 | | TTTTCTTTCAAAAATATTCAGAAGC<br>GCAGGATTTCATCGGTTGTT | Acemaz |
| 5.01 | bnlg1836 | | GGGTTGATGCAAGATGGAAC<br>AGACGAAACATACGAACGGG | Acemaz |
| 5.01 | bnlg143 | | GCACTGCCGGAGTGCCTTCT<br>ATGCCGTGATCTGTGACATCTAACC | Acemaz |
| 5.01 | nc007 | *ohp2* | ACTGTTCCACCAAACCAAGC<br>CTCCATGGAGAAGACGCG | Senior, L |
| 5.01 | phi024 | *ohp2* | ACTGTTCCACCAAACCAAGCCGAGA<br>AGTAGGGGTTGGGGATCTCCTCC | Chin, E |
| 5.02 | bnlg105 | | GACCGCCCGGGACTGTAAGT<br>AGGAAAGAAGGTGACGCGCTTTTC | Acemaz |
| 5.02 | bnlg565 | | TAAGAACGACGAACGGTAACTG<br>GCTCACTGCACGCCAACAC | Acemaz |
| 5.02 | dupssr1 | | TGTTCTCAACAACCACCG<br>CGTTTAGCGATATCATTTTCC | Taramino, G |
| 5.02 | phi113 | *ole2* | GCTCCAGGTCGGAGATGTGA<br>CACAACACATCCAGTGACCAGAGT | Chin, E |
| 5.03 | bnlg1046 | | TGAGCCGAAGCTAACCTCTC<br>GATGCAAAGGAGGTTCAGGA | Acemaz |
| 5.03 | bnlg1063 | *bnlg1063c* | GGAGACAACCCCGACGAC<br>GGTACCAGAGCCACAGATCC | Acemaz |
| 5.03 | bnlg1208 | | GCTGTGATGGTGAGACGAGA<br>GCAGGCACTACTAAAACCGC | Acemaz |
| 5.03 | bnlg1660 | | AACCAAGGTTCTTGGAGGCT<br>ACCATTGTATTTTCCTAGAGAATCG | Acemaz |
| 5.03 | bnlg1700 | | GTCACATCCATGTAGTGCACG<br>GGCACCCTTTTGAAACCTTT | Acemaz |
| 5.03 | bnlg1879 | | TGCTCTCACAAGATGGTGGA<br>CCACAGGATAAAATCGGCTG | Acemaz |
| 5.03 | bnlg1902 | | AACTACCGTCGAAGTGGTGG<br>CGCCTCTCTCTGACTTGTTG | Acemaz |
| 5.03 | bnlg557 | | TCACGGGCGTAGAGAGAGA<br>CGAAGAAACAGCAGGAGATGAC | Acemaz |
| 5.03 | phi008 | *rab15* | CGGCTACGGAGGCGGTG<br>GATGGGCCCACACATCAGTC | Chin, E |

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| 5.04 | bnlg1287 | | GCCCTACCTGTTCTGTCTCG<br>TGTCCCATACCTCAACGTGA | Acemaz |
| 5.04 | bnlg2323 | | ACCGTCTCAGCAAAATGGTC<br>CCGCCTTCACTATGGTCAAT | Acemaz |
| 5.04 | bnlg150 | | GAAAAACCCCCTCCCCATAT<br>AATGGCCGAACACAATTCAA | Acemaz |
| 5.04 | bnlg603 | | CTGAGCTGGCCCCTGTGAATGGTG<br>CGCCCTCCGCTGCGCTTCTCT | Acemaz |
| 5.04 | bnlg653 | | CGCATTGCCATGGATGAAGAACTGG<br>GCAAGCGCCTCACAAGGTATGCACA | Acemaz |
| 5.04 | dupssr10 | | AGAAAATGGTGAGGCAGG<br>TATGAAATCTGCATCTAGAAATTG | Taramino, G |
| 5.05- 5.06 | bnlg1237 | | TGGCGCGATTTTCTTCATAT<br>AAAGAGCAACCTTCAACGGA | Acemaz |
| 5.05 | bnlg1246 | *bnlg1246a* | CGCAGGCCGGGGAA<br>CCTGGCGCCCAACC | Acemaz |
| 5.05- 5.06 | bnlg278 | | CATGCATCAACGTAACTCCCT<br>CATGTCACGCGTTCCACTTG | Acemaz |
| 5.05 | mmc0282 | | CTCTTTCTTTATTTGTTCCGTT<br>GGACTACACATCACCAGCA | Edwards, KJ |
| 5.06 | bnlg1847 | | GACGCTAGAGAGAGGCGAAG<br>ATGTAACAAGAAGGCCCGTG | Acemaz |
| 5.06 | bnlg609 | | GCTCGTTCTCGCCAGTGTGCCG<br>GGCCCGAGCCATCTCTGCTGC | Acemaz |
| 5.06 | phi087 | *umc51a* | GAGAGGAGGTGTTGTTTGACACAC<br>ACAACCGGACAAGTCAGCAGATTG | Chin, E |
| 5.06 | phi100 | *umc51a* | AATCTGCTGACTTGTCCGGTTGTC<br>CCATACATATCGGCCATGATGCTC | Chin, E |
| 5.06 | phi101 | *umc51a* | TGTTCGCCGTCTAGCCTGGATT<br>TCATCAGCAACGACGACTACTCC | Chin, E |
| 5.06 | umc1019 | *umc126a* | CCAGCCATGTCTTCTCGTTCTT<br>AAACAAAGCACCATCAATTCGG | McMullen, MD |
| 5.07 | bnlg1118 | | CAGAGTTGATGAACTGAAAAAGG<br>CTCTTGCTTCCCCCCTAATC | Acemaz |
| 5.07 | bnlg1306 | | CACCTTGAAAGCATCCTCGT<br>CAAAAACAAATGGCAGCTGA | Acemaz |
| 5.07 | bnlg1346 | | CATCATGAAGCAATGAAGCC<br>CCGCGCCATTATCTAGTTGT | Acemaz |
| 5.07 | bnlg1695 | | ACCAAATCCTCATCTCGGAA<br>CAATCTCCCCAAAATCTCGA | Acemaz |
| 5.07 | bnlg1711 | | TAATCTTGGGGGGTTTAGGG<br>GACATGTCCCATTCCCATTC | Acemaz |
| 5.07 | bnlg1885 | | GACAGACGCAACTACCGAAA<br>TGTTCAATTTGATGTTCATTGC | Acemaz |
| 5.07 | bnlg2305 | | CACCTTGAAAGCATCCTCGT<br>GTATCACACCCTCTGCTGCA | Acemaz |
| 5.07 | bnlg118 | | CTTCCAGCCGCAACCCTC<br>CCAACAACGCGGACGTGA | Acemaz |
| 5.07 | phi048 | *umc108* | GCAAACCTTGCATGAACCCGATTGT<br>CAAGCGTCCAGCTCGATGATTTC | Chin, E |
| 5.07 | phi058 | *umc108* | AGGTGCTGGACACAGACTTCAAC<br>ACTGAGATCCAGGCTCCTCTTC | Chin, E |
| 5.07 | phi085 | *gln4* | CGAGACCACCATCATCTGGAAG<br>TTTGCAATCGCTTCGGGGACC | Chin, E |
| 5.07 | phi128 | *asg85b* | TTGCTCGGTATGAAGAAAATAGTCTTTCC<br>ATCTTGCAACTAGACTGAGGCAACCA | Chin, E |

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| 5.09 | bnlg386 | | CACCCTCCCTTTGCAGGTA<br>TGGTTTATCAGATAACGATTCAGC | Acemaz |
| 5.09 | bnlg389 | | GGTCACCCTCCCTTTGCAG<br>ATTGCCTACACAGTTTGATTGG | Acemaz |
| 6 | bnlg1043 | | TTTGCTCTAAGGTCCCCATG<br>CATACCCACATCCCGGATAA | Acemaz |
| 6.00- 6.01 | bnlg1597 | *bnlg1597b* | GATAATCTCGTCTCGCCAGG<br>CATAAAAGGATGCCGACGAC | Acemaz |
| 6 | bnlg1600 | | CGATCAGTGCGTGGAGAGTA<br>TAGGCATGCATTGTCCATTG | Acemaz |
| 6 | bnlg161 | *bnlg161b* | GCTTTCGTCATACACACACATTCA<br>ATGGAGCATGAGCTTGCATATTT | Acemaz |
| 6 | bnlg238 | | CTTATTGCTTTCGTCATACACACACATTCAT<br>GAGCATGAGCTTGCATATTTCTTGTGG | Acemaz |
| 6 | phi075 | *fdx1* | GGAGGAGCTCACCGGCGCATAA<br>AAAGGTTACTGGACAAATATGCGTAACTCA | Chin, E |
| 6 | phi126 | | TCCTGCTTATTGCTTTCGTCAT<br>GAGCTTGCATATTTCTTGTGGACA | Chin, E |
| 6 | umc1002 | *fdx2* | AGCTAGCTATACACCGCCAGG<br>TCAGTTTGGAACAGGGAAAAGTA | McMullen, MD |
| 6 | umc1023 | *fdx2* | CTTGTGCCACCACATGCAGTA<br>CAGTTTGGAACAGGGAAAAGTACG | McMullen, MD |
| 6.01 | bnlg1047 | *bnlg1047b* | ATGGAGATGGAGGAGAGAGAGA<br>GATGCGGCGATGGCTAA | Acemaz |
| 6.01 | bnlg1139 | | GGGGGGTTGAGAGAGAAAAA<br>ACGGCGATGATGAATTAAGC | Acemaz |
| 6.01 | bnlg1165 | | CGCTTGCATCATCTCAAGAA<br>TTCAAGTTTAGCCACCCACC | Acemaz |
| 6.01 | bnlg1188 | | ATTAAGTAAATGACTATCTAGTGTTTGTCG<br>AATAGCAAGGCATCAGCCAT | Acemaz |
| 6.01 | bnlg1246 | *bnlg1246d* | CGCAGGCCGGGGAA<br>CCTGGCGCCCAACC | Acemaz |
| 6.01 | bnlg1422 | | GACGATTAACAGGTGGGGAC<br>ATGATGCAAATGAGGCACAA | Acemaz |
| 6.01 | bnlg1432 | | AAAGCAAACAAACAATGGGC<br>TGCGTGCAGTGACATATTCA | Acemaz |
| 6.01 | bnlg1433 | | CTCAGTCCCTCCCATTTTGA<br>TTCTGGCTCAAAGGGCTAGA | Acemaz |
| 6.01 | bnlg1538 | | CAGCCGAAGACGAAGCC<br>GTGGTGAACGAACGAGCAA | Acemaz |
| 6.01 | bnlg1641 | | ATCGTAACTCGATGGTTCGC<br>TACGCTATTCAAAGCGGTCC | Acemaz |
| 6.01 | bnlg1753 | | GTAGGGGTTACAAGCGTTGC<br>GTGGACAGATGTTCACGTGG | Acemaz |
| 6.01 | bnlg1867 | | CCACCACCATCGTAGGAGTT<br>CAGTACACAGCAGGCAGCTC | Acemaz |
| 6.01 | bnlg2097 | | CCAATCCTCAGAACTAGGAGA<br>ACTGGTAGGAGCAAGCAGGA | Acemaz |
| 6.01 | bnlg2191 | | CACACAATCCCCACAAAAAA<br>CGAAACATCCAGGAAACTGC | Acemaz |
| 6.01 | bnlg107 | | GCAACTAGAAGTAGATGGCTTGTTATGG<br>CAACAACAAGTGGCTGGCTAGGGTGAA | Acemaz |
| 6.01 | bnlg249 | | CCGGTCGCAGTTAGTAGATGAT<br>TCGGCGTTGATTTCGTCAGTA | Acemaz |
| 6.01 | bnlg391 | | CAGATATCACAGCATCAGAAGATCA<br>AAAATGTAAGAACTTGTTTGGGATT | Acemaz |

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| 6.01 | bnlg426 | | TGCATTAATTAGAAGGCTATCAAA<br>GGTTTGGTGACTGGACTGACTT | Acemaz |
| 6.01 | phi077 | | GAGAAGAGGATCAGGTTCGTTCCA<br>CGCGTTGTACATCTTGCCTGCTT | Chin, E |
| 6.01 | umc1018 | *gpc2* | GAACGGATATTGGAACCTGTGC<br>GTGCACGGTGTCGTACTTGAAC | McMullen, MD |
| 6.02 | bnlg1371 | | TTGCCGATAAGAACCAAACA<br>ACGACCGGTGTGGTTACATT | Acemaz |
| 6.02 | bnlg2151 | | GGAAGCTCAGGGCTCCTAAT<br>TTAGCTGGCATGCATCATTT | Acemaz |
| 6.02 | y1SSR | *y1* | CAAGAAGAGGAGAGGCCGGA<br>TTGAGCAGGGTGGAGCACTG | Buckner, B |
| 6.04 | bnlg480 | | GACATTTCCAATGGCGGCTTTCC<br>TCTAGTTATTCCAAGCCCTGGGC | Acemaz |
| 6.04 | nc009 | *pl1* | CGAAAGTCGATCGAGAGACC<br>CCTCTCTTCACCCCTTCCTT | Senior, L |
| 6.04 | nc010 | *pl1* | TGAGCTGACGACGAGCAG<br>CATTATCTGTTCGGCCCG | Senior, L |
| 6.04 | phi031 | *pl1* | GCAACAGGTTACATGAGCTGACGA<br>CCAGCGTGCTGTTCCAGTAGTT | Chin, E |
| 6.04 | umc1014 | *pl1* | GAAAGTCGATCGAGAGACCCTG<br>CCCTCTCTTCACCCCTTCCTT | McMullen, MD |
| 6.05 | bnlg1154 | | GGGTGATCACATGGGTTAGG<br>AAATCAATGCTCCAAATCGC | Acemaz |
| 6.05 | bnlg1443 | | TACCGGAATCCTCTTTGGTG<br>TTTGACAACCTCTTCCAGGG | Acemaz |
| 6.05 | bnlg1617 | | CGTGCACGGTACAGAAAGAA<br>AGAAAGCCACGTACCCCTTT | Acemaz |
| 6.05 | bnlg1702 | | TTATCATCAAATGGAGGACACG<br>AAAGACACACGCTAATGGGC | Acemaz |
| 6.05 | bnlg1732 | | AACTTTTGGCATTGCACTGG<br>CGTAAGTGCACACGGCATTA | Acemaz |
| 6.05 | bnlg1922 | | GTCTTGGGCAGTAATCAGGC<br>TCGATCAAAGACGTTCATGC | Acemaz |
| 6.05 | bnlg2249 | | AGGATCCCCTAGCAAAAGGA<br>CCCCCTAGTTCGTTGCATAA | Acemaz |
| 6.05 | mmc0241 | | TATATCCGTGCATTTACGTTT<br>CATCGCTTGTCTGTCGA | Edwards, KJ |
| 6.05 | nc012 | *pdk1* | TAATTTAAACACCACACCACCG<br>ACACACGCCAAAGAAAAACC | Senior, L |
| 6.05 | nc013 | | AATGGTTTTGAGGATGCAGCGTGG<br>CCCCGTGATTCCCTTCAACTTTC | Senior, L |
| 6.05 | phi025 | *pdk1* | GCAACATCCTGGAGAGCCACTACAAGG<br>ACAGCCTGTTTTCCTGGACAGTGAACTC | Chin, E |
| 6.05 | phi078 | *pdk1* | CAGCACCAGACTACATGACGTGTAA<br>GGGCCGCGAGTGATGTGAGT | Chin, E |
| 6.05 | phi081 | *pdk1* | AAGGAACTGGTGAGAGGGTCCTT<br>AGCCCGATGCTCGCCATCTC | Chin, E |
| 6.05 | phi102 | *npi252* | TGAATCTAAACATAACTTATGTCTAGGTACATAG<br>CAAA<br>CCTCGGATTCCGGATTGTAAGTCA | Chin, E |
| 6.05 | phi129 | | GTCGCCATACAAGCAGAAGTCCA<br>TCCAGGATGGGTGTCTCATAAAACTC | Chin, E |
| 6.06 | bnlg345 | | CGAAGCTAGATGTAGAAAACTCTCT<br>CTTACCAACCAACACTCCCAT | Acemaz |
| 6.06 | dupssr15 | | GAAGTCGATCCATCCACC<br>GGGGTAGTGGAGATAACTAGTG | Taramino, G |

| 6.06 | phi070 | *mlg3* | GCTGAGCGATCAGTTCATCCAG<br>CCATGGCAGGGTCTCTCAAG | Chin, E |
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| 6.07- 6.08 | bnlg1136 | | TAACCGGATGAGCATCTTCC<br>CATCAGCTTCAACGAGTTCG | Acemaz |
| 6.07- 6.08 | bnlg1521 | | GTTGCATACACACCACAGACA<br>GATACCTTCCCTGCCTCACA | Acemaz |
| 6.07 | bnlg1740 | | TTTTCTCCTTGAGTTCGTTCG<br>ACAGGCAGAGCTCTCACACA | Acemaz |
| 6.07 | bnlg1759 | *bnlg1759a* | AGACGGAGTCCTCGTTTGC<br>ACCGGTTCGTACCACTCACT | Acemaz |
| 6.07 | phi123 | | GGAGACGAGGTGCTACTTCTTCAA<br>TGTGGCTGAGGCTAGGAATCTC | Chin, E |
| 6.08 | phi089 | | GAATTGGGAACCAGACCACCCAA<br>ATTTCCATGGACCATGCCTCGTG | Chin, E |
| 7 | bnlg1367 | | CGACGGCGTACAGAGAGAG<br>GGTCGCCACCCCACCT | Acemaz |
| 7.00- 7.01 | bnlg1642 | | GAATCCGTACGTTCTTCCCA<br>TTCAGCTAGCTCACGGGATT | Acemaz |
| 7 | bnlg1686 | | GAATCCGTACGTTCTTCCCA<br>TTCAGCTAGCTCACGGGATT | Acemaz |
| 7.00- 7.01 | bnlg2132 | | GGCGAGAGAGGCAAAGTTAA<br>GTCGCACAAGGGGATCAC | Acemaz |
| 7.00- 7.01 | mmc0171 | | AATCCTACTTGCTGCCAAAGC<br>CTTTGAGCTTTTTGTGTGGAC | Edwards, KJ |
| 7.01 | bnlg1200 | | CGTCCTCGTTGTTATTCCGT<br>GTTCCCTCTCTCCCTCCCTC | Acemaz |
| 7.01 | bnlg1292 | | GGCGCGCACATAGCTC<br>GCCTGGGCTGGCTTCA | Acemaz |
| 7.01 | bnlg2160 | | GAAGCAACCCATTTTCATCC<br>AGATTGGATTCCTGCCTCCT | Acemaz |
| 7.01 | phi057 | *o2* | CTCATCAGTGCCGTCGTCCAT<br>CAGTCGCAAGAAACCGTTGCC | Chin, E |
| 7.01 | phi112 | *o2* | TGCCCTGCAGGTTCACATTGAGT<br>AGGAGTACGCTTGGATGCTCTTC | Chin, E |
| 7.02 | bnlg1003 | | GACCCTCAACCGACCAGTAA<br>ATGGCCTTTTTAAAGGAGGG | Acemaz |
| 7.02 | bnlg1094 | | GTGAAGAACGATGACGCAGA<br>CAGCAACGCTCTCACATTGT | Acemaz |
| 7.02 | bnlg1164 | | AAACAGGGTGTGACAGGTCC<br>GAACGGGCAGACGCATAAG | Acemaz |
| 7.02 | bnlg1247 | | GAGCCAACGAAAGGGGTG<br>CCGCAGTCGAACCCTCTC | Acemaz |
| 7.02 | bnlg1380 | | ACAATTCGATCGAGAGCGAG<br>CCTTTCTTGCTGGTTCTTGC | Acemaz |
| 7.02 | bnlg1759 | *bnlg1759b* | AGACGGAGTCCTCGTTTGC<br>ACCGGTTCGTACCACTCACT | Acemaz |
| 7.02 | bnlg1792 | | CGGGAATGAATAAGCCAAGA<br>GCGCTCCTTCACCTTCTTTA | Acemaz |
| 7.02 | bnlg1808 | | CTTTTCTCTTCTAGTAATGAACAGTCA<br>GCATGATCGAACGAAGGC | Acemaz |
| 7.02 | bnlg2203 | | CTCCGGCGAGCCCAGAC<br>CTCGACCCATGCTCTCCTCT | Acemaz |
| 7.02 | bnlg2233 | | AGGGACAGCGAGATAAAGCA<br>GTCCCTGACAGGCGGACC | Acemaz |
| 7.02 | bnlg398 | | CGTCGGCCAACAGGGTATC<br>CTCGCACGCGGTCTTCTTC | Acemaz |

| 7.02 | bnlg657 | | TCTGAGGATGCCCAATCATGCGC<br>CGTTTCCGTTCGTCACCAGCTCG | Acemaz |
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| 7.02- 7.03 | dupssr11 | | AGGCAAGGCTTTCTTCATAC<br>CGGACGACGACTGTGTTC | Taramino, G |
| 7.02- 7.03 | dupssr9 | | GATGTCGTGTGAGTGACCTG<br>GTGTTGCTATTGCAGTGAGAC | Taramino, G |
| 7.02 | phi114 | *oec17*-<br>*Z26824* | CCGAGACCGTCAAGACCATCAA<br>AGCTCCAAACGATTCTGAACTCGC | Chin, E |
| 7.02 | umc1016 | *kpp1* | GTGATACCGGGTAATCTGGTGC<br>GATGATGGGTGATCATCGGTTC | McMullen, MD |
| 7.02 | umc1036 | | CTGCTGCTCAAGGAGATGGAGA<br>GACACACATGCACGAGCAGACT | McMullen, MD |
| 7.03 | bnlg1070 | | TTCCAGTAAGGGAGGTGCTG<br>TAAGCAACATATAGCCGGGC | Acemaz |
| 7.03 | bnlg1305 | | GCACGGGCATCAGAGAGAG<br>CATGGGTAAGTTGCTGAAAGTTT | Acemaz |
| 7.03 | bnlg1579 | | CGGTTAAAAGGAGAGGTCCC<br>GACTTCAGGCACATCTTGCA | Acemaz |
| 7.03 | bnlg339 | | CCAACCGTATCAGCATCAGC<br>GCAGAGCTCTCATCGTCTTCTT | Acemaz |
| 7.03 | bnlg434 | | GTGCAAAGGGGAGAGAGGAA<br>TCGCCGTTCTTCGCCTTAG | Acemaz |
| 7.03 | bnlg572 | | ACTGGACTGTCCTCGTGCCTA<br>CAAAAAAAGATTCGTTCGGAGTAA | Acemaz |
| 7.03 | phi091 | *npi394* | ATCTTGCTTCCATAAGATGCACTGCTCT<br>CTCAGCTTCGGTTCCTACACAGT | Chin, E |
| 7.03 | umc1015 | *php20569a* | CAGACACAAGCAGCAAAGCAAG<br>TCCGACTCCAAGAAGAGGAGAA | McMullen, MD |
| 7.04 | bnlg1161 | | GAACGGACGACGGTCGAT<br>ACCTCCACACGTCCCCAC | Acemaz |
| 7.04 | bnlg1666 | | GCTGGTAGCTTTCAGATGGC<br>TGTCCCTCCTCCAGTTTCAC | Acemaz |
| 7.04 | bnlg1805 | | GCCCGTTTGCTAAGAGAATG<br>TGTTCGAGCATTTGCTCTTG | Acemaz |
| 7.04 | bnlg2259 | | ACCATTGATTTCATGGTATTGG<br>GCGGATAATGACATTGGGTC | Acemaz |
| 7.04 | bnlg2271 | | TAAAGGAGGTGGCGGTGACT<br>GTATACCGAGCTCAGCCGAG | Acemaz |
| 7.04 | bnlg155 | | ACCGAGTAGCCGAGACACG<br>AGAGTCCTGGAGCCACATGAG | Acemaz |
| 7.04 | dupssr13 | | TCGTTCGGTCCATGAAAT<br>CAAATATCTCTCATCTTTGCTGAC | Taramino, G |
| 7.04 | umc1001 | | GCTACCCGCGGACATATAAT<br>CCATGGGTAAAAACCCTACAGTG | McMullen, MD |
| 7.04 | umc1029 | | AACACCTGCTGGATATGGATCACT<br>GGAAGAAAAATGTCGACCTGCTC | McMullen, MD |
| 7.05 | bnlg2328 | *bnlg2328b* | AGCAGTGAGGAAGAAGCAGG<br>TTACCCTCCCTTGTCGTGAC | Acemaz |
| 7.05 | bnlg469 | *bnlg469c* | AGGGTGTACAGGTCCAAGTCCAA<br>AATGTGGGTCGTCAGCCATCAG | Acemaz |
| 7.05 | phi069 | | AGACACCGCCGTGGTCGTC<br>AGTCCGGCTCCACCTCCTTC | Chin, E |
| 7.05 | phi082 | | CACAGCACAGGCAGTTCG<br>CGCGGCAAAAGATCTTGAACACCT | Chin, E |
| 7.06 | bnlg469 | *bnlg469c* | AGGGTGTACAGGTCCAAGTCCAA<br>AATGTGGGTCGTCAGCCATCAG | Acemaz |

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| 7.06 | phi045 | *umc35a* | CTACTACATGCGATCACGGACCAT<br>AACCAGTTCCAGTCTCCACTGAGT | Chin, E |
| 7.06 | phi051 | *umc35a* | GGCGAAAGCGAACGACAACAATCTT<br>CGACATCGTCAGATTATATTGCAGACCA | Chin, E |
| 7.06 | phi116 | | GCATACGGCCATGGATGGGA<br>TCCCTGCCGGGACTCCTG | Chin, E |
| 8.00- 8.01 | bnlg1252 | | GATTTTGCTTGAAGCCGAAG<br>GCTTTGCAGCACTGTCGTAG | Acemaz |
| 8.01 | bnlg1073 | | TCGATCTAAGTATTGTAAACGTACG<br>GTATTTGGAGGCGCCATAGA | Acemaz |
| 8.01- 8.02 | bnlg1194 | | GCGTTATTAAGGCAAGCTGC<br>ACGTGAAGCAGAGGATCCAT | Acemaz |
| 8.01 | bnlg2037 | | GGGTGCTCGTAGTAGGGGTT<br>CTAAGGCACACGGAGAGAGG | Acemaz |
| 8.02 | bnlg1352 | | AAACAAAGCAGAGAGCGGAA<br>CCGTCCGTCTGCTGTAAATT | Acemaz |
| 8.02 | bnlg2235 | | ATCCGGAGACACATTCTTGG<br>CTGCAAGCAACTCTCATCGA | Acemaz |
| 8.02 | bnlg2289 | | CACAAGCATGGAGGAAGACA<br>GAGGAGAAACGAAGCCACTG | Acemaz |
| 8.02 | phi119 | | GGGCTCCAGTTTTCAGTCATTGG<br>ATCTTTCGTGCGGAGGAATGGTCA | Chin, E |
| 8.02- 8.03 | umc1034 | | GTGTTTCCGTTTCGCTGATTTTAC<br>TCATCCATGTGACAGAGACGACTT | McMullen, MD |
| 8.03 | bnlg1067 | | GGCTTGCTTTTGCTTCACTT<br>CTCATCCCATTCGTTCCACT | Acemaz |
| 8.03- 8.04 | bnlg1460 | | TTTCACGGCTGGAGTGCC<br>GACTCCGCTGTTCCAGAATC | Acemaz |
| 8.03 | bnlg1834 | | AAGGTTGGGTGTTGCTATGC<br>TAGCTCTGCCACTGGACATG | Acemaz |
| 8.03 | bnlg2082 | | GACGGAAGGTGGAGCATAGA<br>ACGAACGTGATACGGGTCTC | Acemaz |
| 8.03 | bnlg669 | | GCACGCACCAGCAGTCGGCAGT<br>CGGCCTAGTGGGCATGGAGCCT | Acemaz |
| 8.03 | dupssr3 | | TTTAAAACCTCTTTATGACTTTTG<br>CTGATACCATATCCAGCATCA | Taramino, G |
| 8.03 | phi115 | *act1* | CTAGTGGGCGAACAACTGGTAAG<br>AAAGAGACCGTGTCAGGATTGCC | Chin, E |
| 8.03 | phi125 | | ACCGCCGGTGCGAGTTGAAG<br>CTTGGGATTGCCCTCATCCAC | Chin, E |
| 8.04 | bnlg1863 | | GGCGTTCGTTTTGCACTAAT<br>CGACACAGTTGACATCAGGG | Acemaz |
| 8.04 | bnlg119 | | AGGTGAGGAGAGGAAAGGTTGT<br>GCCACTCCGCATCCGAGC | Acemaz |
| 8.04 | phi014 | *rip1* | AGATGACCAGGGCCGTCAACGAC<br>CCAGCTTCACCAGCTTGCTCTTCGTG | Chin, E |
| 8.04 | phi060 | *rip1* | ACATGCAGAAGCTTGGCATCAAGG<br>GCTGAGCGATCAGTTCATCCAG | Chin, E |
| 8.04 | phi121 | | AGGAAAATGGAGCCGGTGAACCA<br>TTGGTCTGGACCAAGCACATACAC | Chin, E |
| 8.05- 8.06 | bnlg1152 | | CGCTACCGATTGTTGAATTG<br>AAAGTCGTCCGGTCAAATTG | Acemaz |
| 8.05 | bnlg1176 | | ACTCCTCAAAACCTAGGTGACA<br>CACCGATGATGGTGAGTACG | Acemaz |
| 8.05 | bnlg1246 | *bnlg1246c* | CGCAGGCCGGGGAA<br>CCTGGCGCCCAACC | Acemaz |

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|---|---|---|---|---|
| 8.05 | bnlg1446 | | GTCTAGATCTGTGGCATTCACC<br>GCTCTCTCTCTCTCTCTCTCTCC | Acemaz |
| 8.05 | bnlg1599 | | TTAAATCTTCTCCGAGGCGA<br>GCCGATCTTGAGGAAGCC | Acemaz |
| 8.05 | bnlg1651 | | GAGAGGAGAGAACCACGCAC<br>CTCGAGTCCAAGTCCACCAT | Acemaz |
| 8.05- 8.06 | bnlg1782 | | CGATGCTCCGCTAGGAATAG<br>TGTGTTGGAAATTGACCCAA | Acemaz |
| 8.05 | bnlg1812 | | CGAGAAGACTTGCGTGAACA<br>TTACGTGCGTCGTCAGAATC | Acemaz |
| 8.05 | bnlg2046 | | TTGGTGAAACGGTGAAATGA<br>CTGGTGAGCTTCACCCTCTC | Acemaz |
| 8.05 | bnlg2181 | | CCAATTCACCAATCATGCAA<br>TTGGGGTGAAGCAATGTGTA | Acemaz |
| 8.05 | bnlg162 | | ACTAGCAGCAGTAAAACCTAATAAAGGGA<br>CAAGTAGCTAGCAGTCATTTGCAGTGT | Acemaz |
| 8.05 | bnlg666 | | AAAAGGCAAGTAGCTAGCATGCATTTGCAG<br>GGCTCACGTCCGTATCCAAACCAACA | Acemaz |
| 8.06 | bnlg1031 | | AATCGGTGAGGCTTCACAAC<br>ATGCCTACCTACCACCATGC | Acemaz |
| 8.06 | bnlg1065 | | TGATGCTCGTTGCTTACCTG<br>TTGCCTCTCGTCTTCCAACT | Acemaz |
| 8.06 | bnlg1607 | | TTCGTCCAGATTTCATTCCC<br>CGTCCGTCCTTTTCTGAGAG | Acemaz |
| 8.06 | bnlg240 | | AAGAACAGAAGGCATTGATACATAA<br>TGCAGGTGTATGGGCAGCTA | Acemaz |
| 8.06- 8.07 | mmc0181 | | CTAATCACCAACCACCAACAC<br>AGTCCGTCCTCTGTCCTCGTC | Edwards, KJ |
| 8.07 | bnlg1350 | *bnlg1350b* | TGCTTCAGCGCATTAAACTG<br>TGCTCGTGTGAGTTCCTACG | Acemaz |
| 8.07 | bnlg1823 | | TGTGACTCCATACCGCACAT<br>CTCATCATGTTGTACATGGCG | Acemaz |
| 8.07 | bnlg1828 | | TTGTGTACGATGCGATCGAT<br>ACAACGGACAGGAACAGGAC | Acemaz |
| 8.08 | bnlg1056 | | ATCGTTGTTGGGTACACGGT<br>ACGGGTAGTGGTGAAGATGC | Acemaz |
| 8.09 | bnlg1131 | | TTAGTTGGGTAAACGTGCAC<br>GCATCAGGGGGTAGTTGAGA | Acemaz |
| 8.09 | dupssr14 | | AGCAGGTACCACAATGGAG<br>GTGTACATCAAGGTCCAGATTT | Taramino, G |
| 8.09 | phi015 | *gst1* | GCAACGTACCGTACCTTTCCGA<br>ACGCTGCATTCAATTACCGGGAAG | Chin, E |
| 8.09 | phi080 | *gst1* | CACCCGATGCAACTTGCGTAGA<br>TCGTCACGTTCCACGACATCAC | Chin, E |
| 9 | bnlg1272 | | ACCGAAGATGAGGTGTGACA<br>TCAGTGCAAGGGCAATTTAG | Acemaz |
| 9 | bnlg1724 | | CTGACCCAGAGCATTGTGAA<br>GATGAAGAGCTTGCAGTCCC | Acemaz |
| 9.01 | bnlg1288 | | TCGCTCCTCGGCCTATAGTA<br>GGTGGCAGACCCAAGATTTA | Acemaz |
| 9.01 | bnlg1583 | | ATCAAGCTTATCGAGAGAGAGAGAG<br>CGACGGTGGAAAGACTGC | Acemaz |
| 9.01 | bnlg1810 | | ATGCTCCTCCTCTCCTCCAT<br>GCGATGATGAGCTGCAAGTA | Acemaz |
| 9.01 | bnlg2122 | | TCATCTGGCAAAACCTAGCC<br>CTTGCCAACTTGAGGACATG | Acemaz |

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|---|---|---|---|---|
| 9.01 | phi028 | *sh1* | TCTCGCTGTCCTTCGATTAGTACGG<br>AATGCAGGCGATGGTTCTCCGGCCT | Chin, E |
| 9.01 | phi033 | *sh1* | ATCGAAATGCAGGCGATGGTTCTC<br>ATCGAGATGTTCTACGCCCTGAAGT | Chin, E |
| 9.01 | phi044 | *sh1* | TTATTGGTCCCTCTCCCGTCCCAGA<br>AGCATACCCCAATGGTCAACAGGGA | Chin, E |
| 9.01 | phi067 | | CTGCAAAGGTAAGCACTAGGATGCT<br>CATCATTGATCCGGGTGTCGCTTT | Chin, E |
| 9.01 | phi068 | | GTACACACGCTCCGACGATTAC<br>TCTTCTCCACCAGAGCCTTGTAAG | Chin, E |
| 9.02 | bnlg1082 | | AAAGATCATGGGCGTACCAG<br>CAGGAACCTGATGACCACCT | Acemaz |
| 9.02 | bnlg1372 | | AGCGGTGCTCAAATAGGAG<br>CGCCGGCTTCCCTCAC | Acemaz |
| 9.02 | bnlg1401 | | CACTCGGTTTTTGCTTAGCC<br>GTGTCGTCGAGTGCATGC | Acemaz |
| 9.02 | bnlg1913 | | TAGGGTTTACACGCGCGG<br>ATTTCGCTAAGTCTTTGGCG | Acemaz |
| 9.02 | bnlg244 | | GATGCTACTACTGGTCTAGTCCAGA<br>CTCCTCCACTCATCAGCCTTGA | Acemaz |
| 9.02 | dupssr19 | | GCTGAAGGACTAAAGAAACCG<br>CCTCCAAGGTTGGTACTGTC | Taramino, G |
| 9.02 | dupssr6 | | GATCCTACCAAAATCTTATAGGC<br>ACAGCTAGCCAAGATCTGATT | Taramino, G |
| 9.02 | phi017 | *bz1* | CGTTGGCGACCAGGGTGCGTTGGAT<br>TGCAACAGCCATTCGATCATCAAAC | Chin, E |
| 9.02 | umc1033 | | CTTCTTCGTAAAGGCATTTTGTGC<br>GTGCGGGATTCCTTAGTTTGC | McMullen, MD |
| 9.02 | umc1037 | | GTGCGCGATTCCTTAGTTTGC<br>CTTCTTCGTAAAGGCATTTTGTGC | McMullen, MD |
| 9.03- 9.04 | bnlg1626 | | TTAAATCCAGAGTGTCCCCG<br>TTCTGGATGGTTGCACACAT | Acemaz |
| 9.03 | bnlg1687 | | GGGCAGCAGGCGAGAG<br>CAACTCAGCCCAGCCAGG | Acemaz |
| 9.03- 9.04 | bnlg1688 | | TATCCCCTTTTCTCGATCCC<br>AAGCAAGCGTCGTTTTGTCT | Acemaz |
| 9.03 | bnlg1730 | | GGGTGCTCGTAGTAGGGGTT<br>AACACGTCAACAAGGGGAAG | Acemaz |
| 9.03 | bnlg469 | *bnlg469a* | AGGGTGTACAGGTCCAAGTCCAA<br>AATGTGGGTCGTCAGCCATCAG | Acemaz |
| 9.03 | bnlg127 | | CATGTATACGAGAAGCACCCTAT<br>ATCGTAACTCAGCGGTTTGTG | Acemaz |
| 9.03 | bnlg430 | | CTTACTGAGCATCTTCCTTCTCTCC<br>TCCGGTGATGCTCCAGCGAC | Acemaz |
| 9.03 | bnlg469 | *bnlg469a* | AGGGTGTACAGGTCCAAGTCCAA<br>AATGTGGGTCGTCAGCCATCAG | Acemaz |
| 9.03- 9.04 | mmc0051 | | ACGACTCTATCCCTGCCAACT<br>TCTGGTTGTGAAAGCTATCCT | Edwards, KJ |
| 9.03 | nc134 | *gl15* | CTCAGTTCTTTTCGATGGACG<br>AGTCGCCTGCAGCTAGCTAG | Senior, L |
| 9.03 | phi022 | *wx1* | TGCGCACCAGCGACTGACC<br>GCGGGCGACGCTTCCAAAC | Chin, E |
| 9.03 | phi027 | *wx1* | CACAGCACGTTGCGGATTTCTCT<br>GCGTACGTACGACGAAGACAC | Chin, E |
| 9.03 | phi061 | *wx1* | GACGTAAGCCTAGCTCTGCCAT<br>AAACAAGAACGGCGGTGCTGATTC | Chin, E |

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|---|---|---|---|---|
| 9.03 | phi065 | *pep1* | AGGGACAAATACGTGGAGACACAG<br>CGATCTGCACAAAGTGGAGTAGTC | Chin, E |
| 9.04 | bnlg1714 | | CATCATGGAGGCATATGTCG<br>ACACATTTAGACCCACCCCA | Acemaz |
| 9.04 | phi016 | *sus1* | TTCCATCATTGATCCGGGTGTCG<br>AAGGAGCAACATCCCATCCAGGAA | Chin, E |
| 9.04 | phi032 | *sus1* | CTCCAGCAAGTGATGCGTGAC<br>GACACCCGGATCAATGATGGAAC | Chin, E |
| 9.04 | phi042 | *sus1* | ATGTGGCCATCATTCAATGCTGTAGAC<br>ACACATGCAGGTGCAGCCAGA | Chin, E |
| 9.05 | bnlg1012 | | GAGTGAGCGTGCGGAGTC<br>AACAGGCCAAACTCCTCCTC | Acemaz |
| 9.05- 9.06 | bnlg1091 | | ATTCTTTCCCAGAGCAGCAA<br>TTGTGCGATTGTGTACGGTT | Acemaz |
| 9.05- 9.06 | bnlg1129 | | GAGAGTATGCTACTCGCCGC<br>GACGAGTTTGGAGTGCCATT | Acemaz |
| 9.05 | bnlg1209 | | GTCCCGGGCAGAATAATACC<br>TTCCTCCTTGAAGTGCTCGT | Acemaz |
| 9.05- 9.06 | bnlg1270 | | TAGTTAACATGAGCAAATTAACAAGA<br>TAGAAATGCAGAACCAGGGC | Acemaz |
| 9.05 | bnlg1884 | | TTCGGATGCATGTGTAACGT<br>CGGAAGTCCCATCTGTTTGT | Acemaz |
| 9.05 | phi040 | | GGGATATATGTCCCCCACAATCGT<br>GGCCCTAAGCGAAAATCTATGCTGA | Chin, E |
| 9.06 | bnlg292 | *bnlg292a* | TGGTAGGACCTTACAATGGGA<br>CGGGAGTACTGCTACACACGA | Acemaz |
| 9.07 | bnlg1191 | | AATCATGCGTAGGCGTAGCT<br>GCCAGAGGAAAAAGAAGGCT | Acemaz |
| 9.07 | bnlg1375 | | TCGACAACGAGCAACTCATC<br>CTGCAGATGGACTGGAGTCA | Acemaz |
| 9.07- 9.08 | bnlg1506 | | GCAGGCAACAACCAACAATA<br>AAAGCCTCAGAGCTTCAACG | Acemaz |
| 9.07 | bnlg1525 | | AGGAATTGCGAGTCTTCCAA<br>CAACCCCCAAAATGAACAAA | Acemaz |
| 9.07 | bnlg1588 | | TAACTTGTTGTGCAGAGAGAGAGAG<br>CCCAGAAACATCGCCAATG | Acemaz |
| 9.07 | bnlg128 | | CACCTGGAGGGACCCATTCC<br>AGGACCACAGGATCCATCATCCT | Acemaz |
| 9.07 | bnlg279 | | GCATGCGTACCTTCAAGCTA<br>TGTGTTCATCGGCAATTTTG | Acemaz |
| 9.07- 9.08 | bnlg619 | | ACCCATCCCACTTTCCACCTCCTCCT<br>GCTTTCAGCGAATACTGAATAACGCGGA | Acemaz |
| 9.07 | dupssr29 | | CAGCGAATACTGAATAACGC<br>TGTTGGATGAGCACTGAAC | Taramino, G |
| 10 | phi041 | | TTGGCTCCCAGCGCCGCAAA<br>GATCCAGAGCGATTTGACGGCA | Chin, E |
| 10 | phi117 | | ATCGGATCGGCTGCCGTCAAA<br>AGACACGACGGTGTGTCCATC | Chin, E |
| 10 | phi118 | | TTGGGATGTGATGTGAGAGCTTGCT<br>GAAAGCGGAGAGAGGGCTTCAA | Chin, E |
| 10.02 | bnlg1451 | | TGATCGATGGCTCAATCAGT<br>ATCTGGAACACCGTCGTCTC | Acemaz |
| 10.02 | phi052 | *npi285a(cac)* | CAGAATGGGACGACAAGGTCATC<br>GGGACACTTCTAGCAGGATCTGTTT | Chin, E |
| 10.02 | phi059 | | AAGCTAATTAAGGCCGGTCATCCC<br>TCCGTGTACTCGGCGGACTC | Chin, E |

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|---|---|---|---|---|
| 10.02 | phi063 | | GGCGGCGGTGCTGGTAG<br>CAGCTAGCCGCTAGATATACGCT | Chin, E |
| 10.03 | bnlg1037 | | GCATGATCACAACCACACCT<br>CCTGGCATTTGTTCAACCTT | Acemaz |
| 10.03 | bnlg1079 | | CGTACGTCGTTGCTGTCTGT<br>CAGTACGTGCAGTCCCTCCT | Acemaz |
| 10.03 | bnlg1547 | | TTGGATCAACTTACCCAGGC<br>ACATGCGTGCTACCCATACA | Acemaz |
| 10.03 | bnlg1655 | | ATTAAAATCTTGCTGATGGCG<br>TTCTGTTCCCGCCTGTACTT | Acemaz |
| 10.03 | bnlg1712 | | CTCAGGCTTCACGTGGGTTT<br>GTTACACTCCCCTGCCAAAA | Acemaz |
| 10.03 | bnlg1716 | | AAATAACCAGAACATGCCGC<br>CGCAACTTTCATCGAGTTGA | Acemaz |
| 10.03 | bnlg1762 | | GAGCTCTTGCTTGTGTGCAG<br>ATAAAAGGCCGAGCTTCTCC | Acemaz |
| 10.03 | bnlg210 | | GCCTCGCACCAAGACATAATA<br>TGCCCCATTTGAGTAGACTTC | Acemaz |
| 10.03 | bnlg640 | | TGCGGATCCAACACGGACTGTCC<br>GCAGGCTCTCCGCCCACACCTC | Acemaz |
| 10.03 | phi050 | *umc155* | TAACATGCCAGACACATACGGACAG<br>ATGGCTCTAGCGAAGCGTAGAG | Chin, E |
| 10.03 | phi054 | | AGAAAAGAGAGTGTGCAATTGTGATAGAG<br>AATGGGTGCCTCGCACCAAG | Chin, E |
| 10.04 | bnlg1074 | | CATGCTAATAGCCTACCGGG<br>TTTCCCCCTGATTCGTTATG | Acemaz |
| 10.04 | bnlg1518 | | AGCTGTACACGCAGTAGGCA<br>GGCTCTGTTAATTCGATCGC | Acemaz |
| 10.04 | bnlg1526 | | ACGAGCGAGTGGAGAATAGG<br>AGCCCAGTACGTGGGGTC | Acemaz |
| 10.04 | bnlg2336 | | GGTAGGGGAAAAAACATGCA<br>TGATAAAGTTCTCTATTTGTCTGCC | Acemaz |
| 10.04 | phi062 | *mgs1* | CCAACCCGCTAGGCTACTTCAA<br>ATGCCATGCGTTCGCTCTGTATC | Chin, E |
| 10.04 | phi071 | *hsp90** | GGAGTTCATCAGCTACCCCATCT<br>TTCTGCTTGTTGATCTGCACCCAC | Chin, E |
| 10.04 | phi084 | *nac1* | AGAAGGAATCCGATCCATCCAAGC<br>CACCCGTACTTGAGGAAAACCC | Chin, E |
| 10.05-10.06 | bnlg1028 | | AGGAAACGAACACAGCAGCT<br>TGCATAGACAAAACCGACGT | Acemaz |
| 10.05 | bnlg1185 | | CGGTCCAGGCAGGTTAATTA<br>GACTCGAGGACACCGATTTC | Acemaz |
| 10.05 | bnlg137 | | AGACAACTACCCCCACCCA<br>CCAGGTTACCGTGAAATGCT | Acemaz |
| 10.06 | bnlg1250 | | CCATATATTGCCGTGGAAGG<br>TTCTTCATGCACACAGTTGC | Acemaz |
| 10.06-10.07 | bnlg1677 | | GAGCAGAGCAGCTCCAAGAT<br>AACAAGACGGGAGACAATGG | Acemaz |
| 10.06-10.07 | bnlg2190 | | TCCTCCTTCATCCCCTTCTT<br>CCCAGTATCATTGCCCAATC | Acemaz |
| 10.06-10.07 | bnlg153 | | TCCACTGCTCCTCCACTGC<br>CACTTCAAACTGTCAAATCTCCA | Acemaz |
| 10.06 | bnlg236 | | CGCTTTGCAGTACCAGTACACAC<br>GACGACAACTGCAGAGTACCAGA | Acemaz |
| 10.06 | bnlg594 | | CGAGCGCTTTGCGAGTACCAGTACACA<br>CTGCGTGCGTCCAGCCTCCACT | Acemaz |

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| 10.06 | phi035 | *umc57a* | CGTGCAAGCAGTCCTCCCAG<br>CTCCCTGATGATGAGCTAGAAAGG | Chin, E |
| 10.07 | bnlg1360 | | TCTGCTCATCCACAACTTGC<br>AGAACGTGAAGCTGAGCGTT | Acemaz |
| 10.07 | bnlg1450 | | ACAGCTCTTCTTGGCATCGT<br>GACTTTGCTGGTCAGCTGGT | Acemaz |
| 10.07 | bnlg1839 | | AGCAGACGGAGGAAACAAGA<br>TCTCCCTCTCCCTCTTGACA | Acemaz |
| 10.07 | umc1038 | | CGTCACACTCCTCTGCCACTT<br>GAGGATTCAGAACTCGACTCGG | McMullen, MD |

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**The Greatest Gift to Humankind**

Over 5000 years ago, the **American Indian** transformed the slender two-rowed ear of teosinte into the first little nickel-sized,eight-rowed ear of maize (corn) that soon became their "staff of life". Like most diploids,maize continued to evolve rapidly under the Indian's selection. Now, on this legacy, corn breeding and agriculture have become scientific to meet the world's food needs for human growth in body, mind, and spirit.

This black and white copy for the Maize Newsletter has been adapted from my color painting,size 11" x 17".The feathers represent those of the wild turkey,a common Indian food and now of world-wide use since introduction by the Indians at the first Thanksgiving at Plymouth Colony,in 1621.Together the native Americans and the Pilgrims had an incredible idea.They thought harvest gathers more than food. It brings people together as friends to share in the joy of a good meal. Let us not forget their wisdom and humanity.

CLONE INFORMATION SHEET -- PLEASE SUPPLY FOR EACH CLONE--FORM IS ALSO AT THE FOLLOWING URL:
http://www.agron.missouri.edu/Coop/clonesheet96.html

CLONE DESIGNATION: ISOLATING LAB/PERSON:

IS THIS A KNOWN SEQUENCE CLONE (circle one)? Yes No GENBANK NO.: SWISSPROT NO.:

WHAT PRODUCT OR FUNCTION?

PRODUCT ACRONYM: EC NO.:

PROPOSED GENE NAME: CLONE TYPE (genomic, cDNA, etc.): FROM (ORGANISM):

REFERENCE:

**Restriction Map/Sequence Information (give GENBANK, EMBL, dbEST, SWISSPROT, other Nos. as appropriate):**

**SOUTHERN BLOT INFORMATION**

| LINE ANALYZED | ENZYME(S) TRIED | # BANDS SEEN | APPROX. MW |
|---|---|---|---|
| | | | |

**NORTHERN BLOT INFORMATION**

| TISSUE(S) | CONDITION(S) | # BANDS SEEN | APPROX. MW |
|---|---|---|---|
| | | | |

CHROMOSOME ARM, IF KNOWN:
NEAREST MARKERS, IF KNOWN:

***If you already have map information for this clone, please submit mapscores and mapping population information in typed or electronic format with this form for inclusion in the Maize Genome Database. New data also will be entered in MaizeDB.***

**IT IS OPTIMAL FOR US TO RECEIVE A STAB** (ELSE 10μg OF DRIED PLASMID WOULD BE ACCCEPTABLE).

HOST OF SUPPLIED STAB CULTURE: AMT. OF PURIFIED PLASMID:

VECTOR: SELECTIVE AGENT:

ENZYME(S) TO CUT OUT INSERT: INSERT SIZE:

CAN THE INSERT BY PCR'D? Yes No PRIMER SEQUENCE:

SPECIAL CONDITIONS NEEDED FOR PCR:

MAY WE FREELY DISTRIBUTE THIS CLONE NOW? Yes No
AFTER PUBLICATION OR ONE YEAR? Yes No
CONTACT PERSON REGARDING CLONE:

NAME:

ADDRESS:

PHONE: FAX:

E-MAIL:

SEND CLONES AND INFORMATION TO:

MS THERESA MUSKET
302 CURTIS HALL
UNIVERSITY OF MISSOURI
COLUMBIA, MISSOURI 65211

PHONE: 573/882-2033
FAX: 573/884-7850

EMAIL: MUSKET@teosinte.agron.missouri.edu

April, 1999

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MAIZE GENETICS COOPERATION NEWSLETTER and the MAIZE GENOME DATABASE

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| ...... | 2002 issue | 12.00 | 6.00 | .............................. |
| ...... | 2003 issue | 12.00 | 6.00 | .............................. |
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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 2000 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. Check MaizeDB for the most current information on submission of notes. Send your notes as attachments or in the body of an email addressed to coee@missouri.edu (we will acknowledge receipt, and will contact you further if necessary). You may also send by FTP to teosinte.agron.missouri.edu (pub/mnl_submit directory; see MaizeDB for details), and alert us with an email. If email is not feasible, please mail a double-spaced, letter-quality copy of your note, preferably with a disk containing the 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 electronic form (jpg or gif) if possible. To separate columns in tables, please tab instead of using spaces, to ensure quality tabulations on the web. Your MNL Notes will go on the Web verbatim promptly, and will be prepared for printing in the annual issue. Mailing address:


Ed Coe
210 Curtis Hall
University of Missouri
Columbia, MO 65211-7020


**SEND YOUR ITEMS ANYTIME; NOW IS YOUR BEST TIME**

| | |
|---|---|
| MNL 63ff. on line | MaizeDB - http://www.agron.missouri.edu |
| Author and Name Indexes (and see **MaizeDB**) | |
| Nos. 3 through 43 | Appendix to MNL 44, 1970 (copies available) |
| Nos. 44 through 50 | MNL 50:157 |
| Nos. 51 to date | Annual in each issue |
| Symbol Indexes (and see **MaizeDB**) | |
| Nos. 12 through 35 | Appendix to MNL 36, 1962 (copies available) |
| Nos. 36 through 53 | MNL 53:153 |
| Nos. 54 to date | Annual in each issue |
| Stock Catalogs | Each issue 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; 72:118 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 report 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, and advice or help you received, when you publish.

**MaizeDB** needs Cooperators (this means you) to:

(1) Look up "your favorite gene" in **MaizeDB** (see section V in this Newsletter) and send refinements and updates to maryp@teosinte.agron.missouri.edu, coee@missouri.edu, or db_request@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) Provide probe or primer information per the information sheet in the back of this issue; users also will be helped by 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. Prof L.