MAIZE GENETICS COOPERATION

NEWSLETTER

66

March 15, 1992

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

This issue is dedicated to

Marcus M. Rhoades

Original "Secretary"
for the
Maize Genetics Cooperation Newsletter

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Maize Genetics Stock Collection

Stimulator of Cooperation

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Marcus M. Rhoades, 1903-1991

The death of Dr. Marcus M. Rhoades on December 30, 1991 ended a long career devoted to the observation, study, and genetic manipulation of the maize plant. Dr. Rhoades held a unique place in the community of maize researchers. His associations with maize workers, spanning over six decades, left their mark on hundreds of individuals in all parts of the world. He will be mourned not only by his own students and close colleagues, but also by younger corn workers who treasure a few brief encounters with him and by good friends in universities across the nation whose visits to his laboratory were occasions for stimulating conversation and exchange of information.

Dr. Rhoades played an important role in the founding of the Maize Genetics Cooperation. The now legendary "cornfab" in R. A. Emerson's hotel room during the 1928 meeting of the Genetics Society of America, when the need for cooperative efforts became apparent, has been described in Rhoades's article on "The Early Years of Maize Genetics". Three and a half years later, the Maize Genetics Cooperation was formally organized; Dr. Rhoades was designated custodian of genetic stocks and also served in a secretarial capacity in the reports to corn workers that constitute the first issues of the Maize Genetics Cooperation News Letter. The tremendous excitement and creativity of that time when new discoveries were rapidly being made and the stimulus of daily interaction with fellow maize researchers such as McClintock, Beadle, Emerson, Sprague, and Burnham made his experience as a graduate student and postdoctoral experimentalist at Cornell a high point in Dr. Rhoades's life; he referred to that period as the "Golden Age of Corn Genetics". Many years later, Dr. Rhoades again became intimately involved with the Maize Genetics Cooperation when he supervised the transfer of the genetic stock collection from Ithaca to Urbana. At the same time, he took responsibility for the preparation of the annual News Letter; from 1956 to 1974, the News Letters were assembled, edited, and distributed from Urbana and, when he joined the Botany Department at Indiana University, from Bloomington. Dr. Rhoades was rightfully proud to be a member of an elite group of scientists who unselfishly shared unpublished information, exchanged stocks, and revealed new techniques in order to advance the knowledge of maize genetics.

Marcus Rhoades will be remembered in the annals of genetics as one of the giants who developed and shaped the fields of maize genetics and cytogenetics. He had the discernment to choose significant problems in his research; the continued interest in certain areas uncovered by his pioneering studies is a tribute to his perception and the keenness of his observation. The highly productive period at Cornell was followed by five years at Ames and later at Washington as a USDA geneticist. His first academic appointment was at the Associate Professor level at Columbia University. Eight years later, he transferred his laboratory and corn stocks to the University of Illinois at Urbana and, in 1958, he accepted an offer from Indiana University, where he served as Chairman of the Botany Department for ten years.

In 1981, at the age of 78, Dr. Rhoades was awarded the Thomas Hunt Morgan Medal of the Genetics Society of America in recognition of a lifetime's contribution to genetics. His first scientific paper appeared in 1931 and this News Letter carries a report of his last research efforts. Especially notable among his contributions is the study of the Dotted gene, sometimes regarded as a precursor of the work on transposable controlling elements. The induction of mutations in *a* by the *Dt* gene was thoroughly investigated with the genetic techniques available at the time. It was a remarkable system that attracted considerable attention and Dr. Rhoades often referred to the Dotted study as one of his most important contributions. Many years later, after the discovery of the *Ac-Ds* mutable system by McClintock, Earle Doerschug, a student of Rhoades, showed that *Dt*, like *Ac*, has the ability to transpose and molecular studies by Nancy Shepherd identified a *Ds*-like element inserted in the *A* locus. Work on *Dt* has continued with a developmental study by R. K. Dawe and Michael Freeling.

Another area of interest was non-Mendelian genetics. Rhoades's Ph.D. thesis on cytoplasmic inheritance of male sterility described the first instance in plants where a phenotype other than chlorophyll variegation was determined by cytoplasmic factors. This was followed by a study of plastid mutation by the iojap gene showing that constituents of the cytoplasm could be permanently modified by a nuclear gene. The publications that resulted are frequently cited in textbooks and corn workers such as Susan Gabay-Laughnan, John Laughnan, and Virginia Walbot, have continued to explore this aspect of maize genetics. A life-long study of abnormal chromosome 10 and the associated phenomena of preferential segregation and neo-centromere formation began during his years at Columbia. This example of "meiotic drive" had an observable cytological basis unlike some similar cases in *Drosophila*. In later years, Rhoades made use of a series of terminal deficiencies to elucidate the peculiar structural organization of the abnormal chromosome 10.

Dr. Rhoades had an abiding interest in chromosome mechanics and the influence of heterochromatic elements of the maize genome on such fundamental processes as recombination, meiotic segregation, and behavior of the chromosomes in the microspore divisions. The elegant study of the high-loss phenomenon

showed how interaction of B chromosomes and knobs could cause breakage and loss of chromatin during gametogenesis. All of his research studies were pursued with characteristic energy, persistence, and attention to detail. His scientific papers are models of clarity and demonstrate his masterful command of the English language. Rhoades was also an articulate and lucid lecturer; hundreds of graduate students in botany, zoology, microbiology and agronomy will attest to the relevancy and usefulness of his cytogenetics course in their later careers. Drew Schwartz, one of the first of his 26 Ph.D. students, has written: "He is remembered by his students for his friendly informality, his patient tutoring, his ready wit as well as the thoroughness and dedication with which he presented his lecture material."

As one of the leaders in the field of Genetics, Marcus Rhoades won wide recognition from scientists throughout the world. he was invited to the University of Sao Paulo, to North Carolina State College, to Cornell University, and to the Australian National University at Canberra as Visiting Professor. He was elected to the National Academy of Sciences, the American Philosophical Society and the American Academy of Arts and Sciences. In addition to the T. H. Morgan Award of the Genetics Society of America, he received a Certificate of Merit Award for outstanding contribution to American botany from the Botanical Society of America. He was made a foreign fellow of the Royal Danish Academy of Science and Letters in 1977 and, in 1982, he received an honorary Sc.D. degree from Indiana University. On his 70th and his 80th birthdays, Dr. Rhoades was honored by festschrift volumes containing publications by his students and colleagues. Dr. Rhoades served on innumerable committees, panels, and editorial boards, but he regarded his 12 years participation as a member of the Selection Committee of the Guggenheim Foundation as the most rewarding and enjoyable of these experiences.

Marcus Rhoades's modest demeanor, his enthusiasm for sports, and his easy commerce with people from all walks of life endeared him to many. It is rare to find in one individual a sense of humanity and a common touch combined with a towering intellect and an unflagging dedication to research. Dr. Rhoades possessed these qualities in plentiful measure. Despite several severe setbacks in his health, Dr. Rhoades remained active in his laboratory until his last days. He continued to make pollinations, both in the winter greenhouse and in the cornfield north of Bloomington. He accepted the depredations of old age with grace, dignity, and a truly remarkable fortitude. Throughout his life, he expressed a strong appreciation and admiration of the corn plant--as a research object, as a nutritious food, and as a delightful source of beverage. He also had a deep spiritual affinity for the plant; at his request, his ashes will be scattered in the cornfield. The words written by Dr. Rhoades in a biographical memoir for L. J. Stadler apply equally well to his own passing. "The science of genetics lost one of its most distinguished men. According to his wishes . . . no funeral services were held. Unobtrusively and quietly he passed from this world of living men. But time can never erase from the minds of his students, friends and colleagues their memories of a wise and great man."

Ellen Dempsey

Marcus Rhoades Memorial Fund

One of the world's most distinguished maize geneticists, Marcus Rhoades, passed away on December 30, 1991. A fund in Marcus' memory has been set up at Indiana University. Disbursements from this fund will be used to provide money for graduate students at Indiana who may need it for any of a variety of reasons for which grant funds are not available. Anyone wishing to make a donation in memory of Marcus should send me a check made out to the I.U. Foundation - Marcus Rhoades Memorial Fund.

Tom Blumenthal, Chairman
Department of Biolgoy
Indiana University
Bloomington, IN 47405

I. FOREWORD

Marcus Rhoades, whose memory we honor with the dedication of this issue, produced or read 65 Maize Newsletter issues in which Cooperators shared data and information. This issue of MNL; all of the 65 before it; all map compilations; all gene lists; all of the joyous challenging and sharing in seminars and conferences; and our attitude of sharing knowledge and materials; are a tribute to him. It is fortunate for us that we have been set on this path by such a Cooperator.

The working research information here is shared with the understanding that each item is unpublished and is not to be cited in publications without specific consent of the authors. By sharing our research information here, we contribute to the advancement of biology and to the power of shared technical knowledge.

Information here is in the form of "notes" and is not "published" in the sense of a refereed journal. In event a policy statement should be needed, the following suggested guidelines may ensure against misunderstanding of our Newsletter:

1) In publications, refer to MNL notes in the text, never in the bibliography. Specify "unpublished data", or "personal communication" (i.e., with the colleague's consent). The volume and page numbers might be given, as an aid to the reader.
2) When preparing your MNL notes, emphasize brief technical notes, updates, mutants, mapping data, and the like. Avoid presenting comprehensive material and analyses that are better directed to formal publication.
3) Never refer to MNL notes as "published".
4) If challenged, forward these comments as a statement of the purpose, intent, and policy of the cooperators who contribute to this Newsletter.

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

Gifts to the Endowment Fund for support of the Newsletter now total nearly $75,000. 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. Part of our regular support has been from the National Science Foundation (Integrated Mapping Grant). The continuity and support necessary for collecting genetic and molecular information, evaluating it, and preparing gene lists, maps, and similar syntheses, however, is made possible only by sustained and ongoing encouragement of this work within the Agricultural Research Service, USDA, specifically as part of my regular research project and most recently as part of the Plant Genome Initiative project for development of a database prototype for maize.

A warm acknowledgement for advice and ideas during the past year is given to my colleagues, Gerry Neuffer, Shiaoman Chao, Marty Sachs, Mary Polacco, Jim Birchler, Karen Cone, Kathy Newton, Jack Beckett, and John Walker. Their advice and encouragement is greatly appreciated.

Shirley Kowalewski this year, while coping with new twins, edited and nurtured the contents from rough into fine form, fooled the word-processor into doing what was needed, structured the year's literature, and gave creative advice at critical moments. Mary Ann Steyaert booked addresses and subscriptions through the year, carried out literature searching and verifications with efficiency and accuracy, and artfully prepared the mockup. Denis Hancock steadily and enthusiastically enhanced our computer efficiency to a higher art. Kudos for vigilance and communications are given to Evelyn Bendbow, Earl Patterson, and Dick Whalen, for their contributions of corrections to the gene list and other places. At University Printing Services, Yvonne Ball and the printshop staff again efficiently and carefully made sure that the job was done promptly and well.

For submission of notes for the next issue (Number 67, 1993), please see details inside the back cover.

If you wish to subscribe to this Newsletter please use the form in the back of this issue. Gifts to the Endowment Fund, toward our goal of $100,000, will be very much appreciated.

Ed Coe

AMES, IOWA
Iowa State University

_In_ likely has an insert

--Peter A. Peterson

Geneticists have long noticed the appearance of colored sectors involved with *C-I* in the genotype *C-I C C*. Whether those colored spots are due to the spontaneous loss of *C-I* or the occasional loss of the insert now known to be in *C-I*, has not been verified. Now with *In*, colored sectors often appear in numerous crosses of *c2 in/c2 in* x *c2 In/c2 In*. The expected phenotype from this cross is colorless. If a change occurs in *In* (to *in*?), colored (pale) sectors would appear. It will be interesting to see what the *In* gene status is when *In* is finally isolated and analyzed.

bz-m918501

--Peter A. Peterson

This is a new *bz*-mutable (*bz-m*) that arose in an isolation plot of *Bz/Bz* x *bz/bz*. The only transposon known to have been introduced into this genotype was *Uq-I159*. Preliminary segregation studies show it to be autonomous or to have two or more *Uq* elements segregating.

The pattern of this mutable is relatively coarse. This would suggest, though not yet tested, that these are non-responding stable derivatives. Especially so, in view of the large *bz* sectors in kernels that are heavily mutable.

bz-m918504

--Peter A. Peterson

As with *bz-m918501*, *bz-m918504* also arose in a cross in an isolation plot when *Uq-I159* was the only known transposon. The segregation ratio suggests that it is autonomous as all the round *bz* types are spotted in a cross of *bz-m Sh/bz sh* x *bz/bz*. The pattern is a late type of moderate frequency. There is an unexpected high frequency of completely colored types among the round class. If these represent an instance of a non-suppressible insert, then it would not be autonomous.

bz-m918505

--Peter A. Peterson

This mutable allele also arose in a cross similar to *bz-m918501* when *Uq-I159* was the only introduced transposon. Unlike the two *bz-m* previously described, this one has a relatively dark background expression with a low frequency of spots. Because this was a *bz*-spotted exception that was planted, and this plant was backcrossed by *bz sh*, the appearance of colored progeny as shown in this Table are unexpected.

The cross: *bz-m Sh/bz sh* x *bz sh/bz sh*

		Round		Shrunken	
	Colored	*bz-m*	*bz* stable	Colored	*bz*-st
918805 x 5360	72	69	3	6	63
918805t x 8551	128	96	4	6	253

The near equal frequency of colored and spotted round indicates the possibility of an insert that is not suppressible.

bz-m918516

--Peter A. Peterson

This mutable allele arose as a single kernel exception in the cross *Bz Sh/Bz sh* x *bz sh/bz sh*. The transposon in this cross was *Uq-BSSS*. The testcross segregation is suggestive of an autonomous type.

The cross: *bz-m Sh/bz sh* x *bz sh/bz sh*

		Round		Shrunken		
	Colored	*bz-m* spotted	*bz* stable	Colored	*bz* sp	*bz*-stable
918516t/8551	8	67	1	2	4	73
918516/8551	7	93	2	4	4	84

The pattern of mutability is a light background (unlike *bz-m918505*) with a moderately coarse spotting.

HPLC identification of anthocyanins in maize endosperm

--Elizabeth E. O. Caldwell and Peter A. Peterson

High performance liquid chromatography was used to analyze the anthocyanin content of pooled aleurone and starchy endosperm. Tissue was collected from mature, fully-colored, red, *C-(lineC) sh Bz wx* kernels.

Individual kernels were prepared for analysis as follows:

1. pericarp and embryo tissues removed
2. 1% HCl in water and the remaining tissue (endosperm including aleurone) ground to a slurry
3. supernatant lyophilized
4. pigments redissolved in 1% HCl in methanol

Anthocyanins were separated using a C18 reverse phase column (Lichrosorb 1ORP18, 25 x 0.5cm) and detected at 540nm. The first eluent was 15% acetic acid, 1.5% phosphoric acid, 83.5% water. The second eluent was acetonitrile. The following gradient was a modification of one published by Asen and Griesbach (J. Amer. Soc. Hort. Sci. 108:845-850, 1983):

Time	Eluent 1	Eluent 2
0 to 10 min	9%	1%
10 to 50 min	99% to 80%	1% to 20%

Individual anthocyanins were identified by comparing the unknown retention times with results from extracts of lyophilized USDA-83 petunia petals. The USDA-83 standard was provided by Dr. Robert Griesbach along with identification of its anthocyanin content.

By comparing the maize and USDA-83 extracts, three acylated anthocyanins were identified in the *C-(lineC) sh Bz wx* mature endosperm tissue. These include: peonidin-3-(caffeylrutinoside)-5-glucoside, peonidin-3-(p-coumarylrutinoside)-5-glucoside, and malvidin-3(caffeylrutinoside)-5-glucoside. Peonidin has been reported in maize aleurone but not these derivatives.

HPLC identification of flavonol-glycosides in maize endosperm

--Elizabeth E. 0. Caldwell and Peter A. Peterson

High performance liquid chromatography was used to analyze the flavonol-glycoside content of pooled aleurone and starchy endosperm. Tissue was collected from mature, fully-colored, red, *C-(lineC) sh Bz wx* kernels.

Individual kernels were prepared for analysis as follows:

1. pericarp and embryo tissues removed
2. remaining endosperm (including aleurone) ground to a slurry with water
3. supernatant lyophilized
4. pigments redissolved in methanol

Flavonol-glycosides were separated using a C18 reverse phase column (Lichrosorb 1ORP18, 25 x 0.5cm) and detected at 340nm. The aqueous eluent (Eluent 1) was 1% triethylamine in water, adjusted to pH 3.0 with phosphoric acid. The second eluent was acetonitrile. The following gradient was a modification of one published by Asen and Griesbach (J. Amer. Soc. Hort. Sci. 108:845-850, 1983):

Time	Eluent 1	Eluent 2
0 to 20 min	100% to 80%	0% to 20%
21 to 40 min	80%	20%

Individual flavonol-glycosides were identified by comparing the unknown retention times with results from extracts of lyophilized Blue Magic petunia petals. The Blue Magic standard was provided by Dr. Robert Griesbach along with the identification of its flavonol-glycoside content.

By comparing the maize and Blue Magic extracts, six flavonol-glycosides were identified in the *C-(lineC) sh Bz wx* mature endosperm tissue. These include: quercetin-3-glucoside, quercetin-3,7-diglucoside, quercetin-7-glucoside, quercetin-3-sophoroside, quercetin-3-sophoroside-7-glucoside, and quercetin-3-(caffeoylsophoroside)-7-glucoside. Of these, quercetin-3-sophoroside, quercetin-3-sophoroside-7-glucoside, and quercetin-3-(caffeoylsophoroside)-7-glucoside are identified for the first time in maize tissue.

C-m897140 shows reduced frequency of transmission through male

--Vijay R. Thatiparthi and Peter A. Peterson

A sectored kernel identified as *C-m897140* was obtained from the cross *C Sh Bz Wx/C sh bz X c sh Bz wx/c sh Bz wx* (cross 1) as a single seed exception from a TEL (transposable element laden) population carrying *Cy, Uq* and *En* transposable elements. The genotype of this kernel was *C Sh Bz Wx/c sh Bz wx*. The sectored phenotype shows somatic loss of the *Wx, Bz* and *C* genes when the appropriate tester is used. The plant from this kernel was selfed and outcrossed to a *bz-rcy* tester (cross 2). The sectored kernels from cross 2 were backcrossed both as female and male to the *bz-rcy* tester [(*C Sh Bz Wx/C Sh bz-rcy wx* X *C Sh bz-rcy wx/C Sh bz-rcy wx* (cross 3)]. The percentage of sectored kernels (breakage) among the colored class from cross 3 varies from 0% to 7.4% when used as female and 0% to 33.3% when used as a male. This variable rate is likely due to varied expression for causes still to be determined.

The outcross of the sectored kernels in cross 3 also indicated reduced frequency of transmission of chromosome 9. The percentage of colored kernels from cross 3 was close to the expected 50% when crossed as female and only 18-27% when crossed as male. The reduced frequency of transmission also coincided with a reduction in the crossing over frequency between the *Bz* and *Wx* loci (2.9% to 15.7% as compared to the normal frequency of 25%) possibly indicating the presence of an inversion (crossover suppressor) in the vicinity of the *C-m897140* mutant. Initial screening for the presence of transposable elements indicated the absence of *Uq*. Further tests are under way to confirm the presence of an inversion in the short arm of chromosome 9.

Two-point linkage data for *a2* and *Ae-5180*

--Philip S. Stinard

We report the results of a two-point linkage test between *a2* and the dominant mutant amylose-extender allele, *Ae-5180*, on chromosome 5. The testcrosses were made as indicated in Table 1.

Table 1. Two-point linkage data for *a2* and *Ae-5180*. Testcross: (*a2 Ae-5180/A2 Ae*) X *a2 a2 Ae Ae*.

Reg.	Phenotype	No.	Totals
0	++	1068	
	a2 ae	1095	2163
1	+ *ae*	262	
	a2 +	254	516

% recombination *a2--Ae-5180*=19.3±0.8

The following linkage relationship was established: *a2*-19.3-*Ae-5180*. This result is consistent with the *a2-ae* distance of 22cM reported on the 1991 maize linkage map, but shows a greater distance between *a2* and *Ae-5180* than does our previously reported data (13.1cM; MNL 62:11-13). This discrepancy may be due to differences in genetic background, or environmental influences, and demonstrates how linkage data can vary from replication to replication.

Three-point linkage data for *cr4, oy* and *y9* on 10S

--Philip S. Stinard

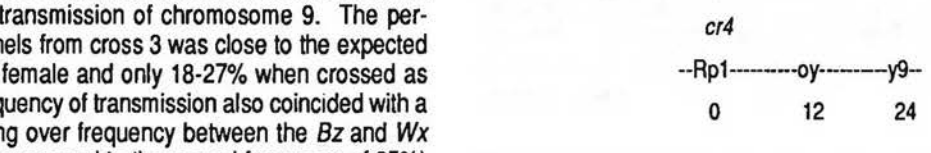
We report the results of a three-point linkage test for *cr4, oy* and *y9* on the short arm of chromosome 10 (Table 1). The linkage test was set up as a modified backcross as indicated in Table 1.

Table 1. Three-point linkage data for *cr4, oy* and *y9*. Testcross: *Cr4 Cr4 Oy Oy Y9 Y9* X (*Cr4 oy y9/cr4 Oy Y9*).

Reg.	Phenotype	No.	Totals
0	+ *oy y9*	135	
	cr + +	179	314
1	+ + +	17	
	cr oy y9	21	38
2	+ *oy* +	16	
	cr + *y9*	13	29
1+2	+ + *y9*	0	
	cr oy +	0	0

% recombination *cr4 --oy*=10.0±1.5
% recombiantion *oy--y9*=7.6±1.4

Kernels from the backcross ears were planted in the field, the resulting plants selfed, and the selfed ears scored for *y9*. Kernel samples from each ear were planted in the sand bench, and the resulting seedlings were scored for *cr4* and *oy*. The following linkage relationship was established: *cr4*-10.0-*oy*-7.6-*y9*. The distance between oy and *y9* (7.6cM) is shorter than that reported on the 1991 linkage map (12cM), but the data unequivocally place *cr4* distal to *oy* on the short arm of chromosome 10, in the vicinity of *Rp1*. The suggested map revision is:



The elongate method of generating tetraploid maize stocks, revisited

--Philip S. Stinard

D. E. Alexander, in a frequently overlooked 1957 paper (Agron. J. 49:40-43), describes a method of inducing autotetraploidy in maize using the elongate (*el*) gene. Plants homozygous for *el* produce both reduced and unreduced eggs; the 2n constitution of the unreduced eggs is probably due to the omission of the second meiotic division (Rhoades and Dempsey, Genetics 54:505-522). This

unusual meiotic behavior can be exploited in order to first transfer a mutant allele of interest from a diploid background into a diploid homozygous *el* background, followed by recovery of the mutant in a tetraploid background following pollination of the mutant *el el* line by a previously established tetraploid line. We used this method to generate tetraploid stocks carrying the dominant mutant amylose-extender allele, *Ae-5180*.

A homozygous *el* plant was pollinated by a heterozygous, diploid *Ae-5180* plant (*Ae-5180 Ae*) in our 1989 summer nursery. The resulting ear segregated for plump wildtype kernels (with *Ae Ae El el* embryos), plump glassy kernels (with *Ae-5180 Ae El el* embryos), and shriveled, defective (triploid or aneuploid embryo) kernels. Plump *Ae-5180* kernels were planted in our 1989-1990 winter nursery, and the resulting plants self-pollinated. The selfed ears segregated in a 3:1 ratio of *Ae-5180* phenotype : wildtype kernels. Among the selfed progeny kernels, one fourth would be expected to have *el el* embryos and thus give rise to plants producing unreduced (2n) eggs. Progeny kernels with *Ae-5180* phenotype were planted in our 1990 summer nursery, and the resulting plants pollinated by tetraploid maize lines obtained from the Maize Stock Center. Of 27 successful pollinations, two ears segregated for both plump (putative tetraploid embryo) and shriveled (triploid embryo) kernels, and thus must have been borne on *el el* plants. The remaining 25 ears had only shriveled (triploid embryo) kernels.

Putative tetraploid embryo kernels with *Ae-5180* phenotype were planted in our 1990-1991 winter nursery, and the resulting plants pollinated by a tetraploid W23 stock obtained from the Maize Stock Center. The four ears obtained from these crosses were scored for the number of *Ae-5180* and wildtype kernels (Table 1). Three of the four ears had ratios of *Ae-5180* : wildtype that approximated 1:1 ratios (one of these ears differed from 1:1 at the $p < 0.05$ level). The remaining ear had a 5:1 ratio of *Ae-5180* : wildtype, a classical tetraploid ratio. These ratios can be explained if the three 1:1 ears came from plants with the tetraploid genotype *Ae-5180 Ae Ae Ae* (the product of a single crossover between *Ae-5180* and the centromere during meiosis I in the *el el* parent), and the 5:1 ear came from a plant with the genotype *Ae-5180 Ae-5180 Ae Ae* (no crossover between *Ae-5180* and the centromere). We conclude that *Ae-5180* behaves as a dominant mutant allele in tetraploid as well as in diploid backgrounds.

Table 1. Counts of *Ae-5180* and wildtype kernels on ears from the cross of tetraploid plants carrying *Ae-5180* by a tetraploid W23 (wildtype) stock.

Female Parent	Ae-5180	wildtype	chi-square (ratio)
90-91-8525-1	136	162	2.268 (1:1)
-8525-4	153	115	5.388 (1:1)*
-8525-5	154	140	0.667 (1:1)
-8526-8	261	52	0.001 (5:1)

* Chi-square for 1:1 ratio significant at $p < 0.05$ level.

shrunken-6 (*sh6*), a new shrunken endosperm/virescent seedling mutant located on 7S

--Philip S. Stinard

A new recessive shrunken kernel mutant was found segregating on the selfed ear of a plant (86-87-8601-3) derived from a *y1-Mu* stock. The mutant kernels are nearly identical to *sh1* kernels in appearance. When planted in a sandbench, mutant kernels give rise to pale green seedlings that green slowly. One mutant plant was grown in the field to maturity, producing a tassel that shed poorly, as well as a small ear. Mutant seedlings from the original isolate were mutable, with small longitudinal revertant sectors typical of mutable seedling mutants derived from Mutator lines. Stable lines have since been isolated.

Plants heterozygous for this mutant, originally designated *su-sh*-8601*, were crossed by a series of B-A translocations involving 19 of the 20 maize chromosome arms. Positive tests were obtained for crosses involving TB-7Sc. The mutant *su-sh*-8601* was tested for allelism with *cp2* (collapsed endosperm/pale green sectored seedling), also located on 7S, and was found not to be allelic. Since *su-sh*-8601* appears to be unique, we have given it the designation shrunken-6 (*sh6*). Crosses to further map *sh6* with respect to 7S markers are being planned.

[ed. note: Compare note from Columbia by M. G. Neuffer; presumed allelism to be tested next season.]

sugary-3 (*su3*), a new endosperm mutant with sugary/shrunken phenotype

--Philip S. Stinard

A new recessive mutant conditioning sugary/shrunken endosperm was found segregating on the selfed ear of a plant (84-5081-3) grown from the outcross to standard of a putative Mutator-induced *wx* mutant, *wx-Mus3*. Mutant kernels have the glassy appearance of *su1*, but are smoother than *su1* kernels. Mutant kernels are also shrunken in a manner reminiscent of *sh1*. Mutant kernels give rise to normal, healthy green plants. Analysis of the starch of mutant endosperms reveals that mutant endosperms have a higher amylose content (around 40%) compared to wildtype (typically 25%; Jay-Lin Jane, Iowa State University, unpublished data). This mutant, originally designated *su-sh*-5081*, was allele tested with all mutants with similar phenotype, including *sh1*, *su1*, *su2*, *ae*, *du*, *sh2*, *bt1*, and *bt2*. All allele tests proved negative. Since the endosperm phenotype is most similar to *su1* and *su2*, *su-sh*-5081* was given the designation sugary-3 (*su3*). Although the endosperms of *su3* kernels have elevated amylose content, *su3* was not designated as *ae2* because *su3* lacks the unique phenotypic interaction that *ae* has with *wx*. Kernels homozygous for both *ae* and *wx* in the genetic backgrounds we work with are small, severely wrinkled, and translucent. Kernels homozygous for both *su3* and *wx* are shrunken like *sh1*, but have the opaque appearance of *wx*.

We attempted to locate *su3* to chromosome arm using a series of B-A translocations covering 19 of the 20 maize chromosome arms. We obtained multiple negative results for all chromosome arms tested. We next crossed *su3* to a series of *wx* marked translocations with breakpoints near the centromeres of the non-9 chromosomes. The F1's were selfed and were outcrossed to either standard *wx* testers or *su3* testers. Only selfed ears of the F1 between *su3* and *wx* T4-9g (4S.27, 9L.27) showed evidence of linkage (repulsion) between *su3* and *wx*. The outcrosses of *su3 / wx* T4-9g to *wx* and *su3* were grown in our 1991 nursery, the resulting plants self-pollinated, and the selfed ears scored for *su3* and *wx*. The data from both types of outcrosses were combined in order to calculate linkage values. The results, reported in Table 1, indicate tight linkage between *su3* and *wx* T4-9g ($p = 6.5 \pm 2.5$cM). It should be noted that the parental and crossover classes are unbalanced, with a shortage of kernels in the *wx* classes. This is most likely due to poor seed set on semisterile ears carrying the translocation, leading to the rejection of any ear that had too few kernels to be properly classified.

Table 1. Two-point linkage data for *su3-wx* T4-9g (classification for *su3* and wx only). Testcross: *Su3 Su3 wx wx* n n X (*su3 Wx* n / *Su3 wx* T4-9g) or *su3 su3 Wx Wx* n n X (*su3 Wx* n / *Su3 wx* T4-9g).

Reg.	Phenotype	No.	Totals
0	*su3* +	59	
	+ *wx*	28	87
1	*su3 wx*	1	
	++	5	6

% recombination *su3*--*wx*=6.5±2.5

Because of the evidence of linkage between *su3* and *wx* T4-9g, we conducted a three-point linkage test involving *su3* and the chromosome 4 linkage markers *su1* and *gl4*. The test was set up as a modified backcross as indicated in Table 2. Kernels from the backcross ears were planted in the field, the resulting plants self-pollinated, and the selfed ears scored for *su1* and *su3*. Kernel samples from each ear were planted in the sandbench, and the resulting seedlings were scored for *gl4*. The results (Table 2) indicate independent segregation between *su3* and *su1*, and between *su3* and *gl4*. The linkage observed between *su1* and *gl4* (p = 18.6cM) is close to that reported on the 1991 maize linkage map (15cM). Since *su3* is not uncovered by either TB-4Sa or TB-4Lf, we conclude that *su3* is probably not located on chromosome 4.

Table 2. Three-point linkage data for *su3*, *su1*, and *gl4*. Testcross: *Su3 Su3 Su1 Su1 Gl4 Gl4* X (*su3 Su1 Gl4 / Su3 su1 gl4*)

Reg.	Phenotype	No.	Totals
0	*su3* ++	38	
	+ *su1 gl*	31	69
1	*su3 su1 gl*	37	
	+++	25	62
2	*su3* + *gl*	11	
	+ *su1* +	7	18
1+2	*su3 su1* +	7	
	+ + *gl*	5	12

% recombination *su3*--*su1*=50.3±3.9
% recombination *su1*--*gl4*=18.6±3.1
% recombination *su3*--*gl4*=49.7±3.9

Further corroboration that *su3* is not located on chromosome 4 was obtained by conducting testcrosses of hypoploid plants grown from crosses of *su3* by TB-4Sa and TB-4Lf. Crosses of hypoploid plants by *su3* produced ears segregating approximately 1:1 for mutant and wildtype kernels, confirming that *su3* is not uncovered by TB-4Sa or TB-4Lf. Since *su3* is linked to *wx* T4-9g, but is not located on chromosome 4, the following possibilities remain: (1) *su3* is located on chromosome 9, or (2) our *wx* T4-9g stock (obtained from the Maize Stock Center) is misidentified and instead is a translocation stock involving chromosome 9 and a chromosome other than chromosome 4. Both of these possibilities are currently being investigated.

A new modifier of aleurone pigments

--Patrick Schnable

A modifier of aleurone pigmentation was uncovered in a cross of Black Mexican Sweet (BMS) with Vebz. BMS carries *A1*, *A2*, *C1*, *C2*, *R*, *Pr* and *su1*; it therefore has purple and sugary kernels. Vebz is a *V*ery early *b*ronze stock developed in Ames in the early 1980's from a cross of a *sh bz* line from Missouri to a colored Cornell line we called Aet. Vebz is homozygous for *A1*, *A2*, *C1*, *C2*, *R*, *pr* and *sh bz*; it therefore produces bronze shrunken kernels. As expected, the F1 between these two lines yielded purple kernels. However, the backcross of this F1 to BMS segregated 1:1 for blue-purple:greenish purple kernels. The former are expressing the BMS phenotype. Nine selfed ears on plants derived from blue-purple F1BC seeds failed to segregate greenish purple kernels. In contrast, all five selfed ears from plants derived from greenish-purple F1BC seeds segregated 3:1 greenish purple:blue-purple. These data demonstrate that the greenish purple phenotype is controlled by a dominant modifier present in the Vebz line and absent in BMS. This modifier is not *Pr*; eight out of the nine selfed F1BC ears that failed to segregate greenish purple kernels were segregating for *pr* and three of the five selfed F1BC ears that did segregate for greenish purple kernels did not carry *pr*. Classification is easiest in a BMS background.

tb may condition a semi-dominant effect on tiller number

--Patrick Schnable

Plants homozygous for *tb* (teosinte branched) are generally highly tillered and produce multiple branches that terminate in tassels. These branches occur at nodes that would normally produce ears. These effects on floral morphology are absent in heterozygotes. However, during backcrossing we noted that *tb* heterozygotes appeared to be more highly tillered than would normally be expected in given genetic backgrounds. To test *tb*'s role as a positive modifier on tiller number in heterozygotes, plants from cross 1 were scored for tiller number and selfed. Progeny tests of 17 of the resulting selfed families established which plants from the cross *tb/Tb* X *Tb/Tb* were heterozygous for *tb*. The ten *tb* heterozygotes had an average tiller number of 4.1. The seven *Tb/Tb* plants had an average tiller number of 2.3. These means are significantly different as determined by the t test. Therefore, this limited data set suggests that *tb* may be a positive modifier of tiller number even when heterozygous.

C-I-b836024: *Ac* induces chromosome breakage at the shrunken locus

--Etienne Kaszas and Peter A. Peterson

The chromosomal breaker linked to *C-I* and described as *C-I-b836024* (M. Muszynski and P. A. Peterson, MNL 64:9), has been further characterized. System tests show that the breaker is identified to the *Ac/Ds* family (observation of the BFB cycle is another confirmation). According to linkage analysis, the breaker is tightly linked to the shrunken-1 locus, or even inserted in the shrunken gene (original mutant occurred as a variegated and shrunken kernel; transposed breakers give in most cases, a low variegated round phenotype).

Segregation ratios indicate that it is an autonomous element (1 sect.:1 non-sect. from crosses to Line C or *C bz*). Therefore, the structure of the breaker could involve two tightly linked *Ac*s, or an *Ac-Ds* complex, similar to the structure of the macrotransposon described by Dooner and Belachew (1991). Crosses were made with recessive distal markers (*yg2*: yellow green and *wd1*: deletion giving rise to lethal seedlings), to detect breakage in leaf tissue. No yellow green or white sectors were observed, which is in contrast to the original *C-I Ds* in the presence of *Ac* (McClintock, 1951). This may be a case of tissue specific expression of *Ac*.

A number of derivatives have been recovered from the original breaker. These include:

1) transposed breakers showing a low pattern of mutability (smaller sectors: 1-3 a), but the BFB cycle still occurs; transposed breakers were mapped and found to be linked to the original site, proximal or distal to the shrunken locus; the segregation (1 sect.:1 non-sect.), characteristic of autonomous control, suggests

that transposition could involve the complex structure, able to induce breakage.

2) changes of state (*C-I-b912009W*: small sectors and a corresponding low mutability of the *wxm9::Ds* allele); the shrunken phenotype, together with a failure to detect any cross-over products carrying the recessive shrunken allele without the breaker, indicates that this breaker derivative did not move from its original site. Further tests are underway to identify the composition of the breaker.

Genetic mapping and allelisms of Mutator-induced defective kernel mutants - an update

--M. G. James, M. J. Scanlon, P. S. Stinard and D. S. Robertson

A large number of defective kernel mutants (*dek*s) have been generated by crosses to Robertson's Mutator for the purpose of transposon tagging. Previously, we reported on the genetic mapping of 47 *Mu*-induced *dek* mutants (MNL 65:11) and the determination of allelisms of 17 of these mutants with each other or with previously described kernel mutants (MNL 65:10). In this report, we describe the establishment of 7 additional allelisms (Table 1), provide more detailed and updated mapping information for four previously described mutants (Table 2), and assign 10 new *Mu*-induced *dek* mutants to chromosome arm (Table 3). Chromosomal placements were conducted using B-A translocations, the wx-9 series of reciprocal translocations, and allelism tests, and more definitive mapping was achieved with conventional linkage analyses using genetic markers on the appropriate chromosome.

We also report that the previous placement by B-A translocation of *dek*-326* (renamed *ren2*, MNL, this issue) to the long arm of chromosome 7 was confirmed through RFLP mapping with MAPMAKER by Lance Veldboom and Mike Lee, Iowa State University. *dek*-326* was determined to lie 3.2cM distal to *BNL14.07* and 4.9cM proximal to *BNL8.39*.

Table 1. Allelic defective kernel mutants.

Mutant designation	Allelic to:	Chromosome arm	Kernel phenotype	New designation
dek-3252*	*dek*-2058*	4S	crumpled, discolored, lethal	*dsc1-3252*
dek-43*	*et1*	3L	etched kernel	*et1-43*
dek-5133*	*sh4*	5L	shrunken: collapsed, chalky endosperm	*sh4-5133*
dek-5132*	*pro1*	8L	crumpled, opaque, lethal	*pro1-5132*
dek-NS413*	*dek*-NS326*	7L	reduced endosperm, lethal	*ren2-413*
dek-PI-AEO*	*dek1*	1S	floury endosperm, pigments absent, germless	*dek1-PI-AEO*
Dap2	*Dap1*	5L	mosaic aleurone color[a]	*Dap1-2*

[a]kernel phenotype listed is for *Dap1 Dap1* + and *Dap2 Dap2* + heterozygous endosperms, where mutant is carried through the female; *Dap1 Dap1 Dap1* or *Dap2 Dap2 Dap2* endosperms have mosaic aleurone, reduced endosperm, and are lethal; + + *Dap1* or + + *Dap2* endosperms are wildtype in appearance (MNL 62:16-17).

Table 2. Genetic mapping of *Mu*-induced defective kernel mutants.

dek mutant designation	chromosome	linkage testcross	% recombination
dek25-2410	4S	*Su1 Gl4/Su1 Gl4* x (*dek25/su1 gl4*)	- *su1*: 44 +/- 2.39 - *gl4*: unlinked
		fl2/fl2 x (*fl2/dek25*)	- *fl2*: 25 +/- 1.95[a]
dek7-2608	4S	*su1 gl4/su1 gl4* x (*su1 gl4/dek7*)	- *su1*: 8.6 +/- 2.28 - *gl4*: 26.7 +/-3.61
		fl2/fl2 x (*fl2/dek7*)	- *fl2*: 7.1 +/-2.27[b]
dek31-2689	4L	*c2/c2* x (*c2/dek31*)	- *c2*: 18.6 +/-3.91
		gl3/gl3 x (*gl3/dek31*)	- *gl3*: 19.6 +/- 3.67
		su1 gl4/su1 gl4 x (*su1 gl4/dek31*)	- *su1*: 32 +/- 4.95 - *gl4*: 22.8 +/- 4.17[c]
emp2 (*dek*-1047*)	2L	*wx/wx* x (*wx* T2-9d/ *dek*-1047*)	- *wx* T: 21.5 +/- 2.75
		W3/W3 x (*w3/dek*-1047*)	- *w3*: 18.5 +/- 2.43[d]

[a]the data suggest *dek25* - 25 - *fl2* - 19 - *su1*
[b]the data suggest *dek7* - 8.6 - *su1* - 18.1 - *gl4* and *fl2* - 7.1 - *dek 7*
[c]the data suggest *su1* - 9 - *gl4* - 23 - *dek31* - 18.5 - *c2*
[d]revised data (from MNL 65:11) suggest *emp2* - 18.5 - *w3*

Table 3. *Mu*-induced defective kernel mutants placed to chromosome arm.

1S	2L	3L	4S
dek-PI-AEO*[a]	*dek*-PI-C3*[a] *dek*-PI-E3*[a]	*dek*-43 (et1)*	*dek*-3252*
5S	5L	7L	
dek-NS807d*[b]	*ren1 (dek*-807)*[b] *dek*-PI-OI*[a] *Dap2*	*ren2-413*[b]	

[a]mutant was a gift from Steve Briggs, Pioneer Hi-bred Int'l., Inc.
[b]mutant was a gift from Nancy Shepherd, DuPont de Nemours, Inc.

Nucleotide sequence and transcript analysis of transposon *Mu-A2*, a regulator of *Mu* element transposition

--Martha G. James, Michael J. Scanlon, Minmin Qin, Donald S. Robertson and Alan M. Myers

This report characterizes *Mu-A2*, a transposon of the Mutator family known to provide transposition function to an independent *Mu* element, *Mu1*, located within the *a1-mum2* allele of the *A1* gene. A genetic element controlling *Mu1* transposition was identified in specific maize lines in which mutable and stable *a1* kernels were produced at a 1:1 ratio in crosses to standard lines (Robertson and Stinard, Dev. Genet. 10:482-506, 1989). This genetic element is termed here *Mu-A2*. Subsequent genetic analysis showed *Mu-A2* is a mobile locus and, furthermore, that its copy number can increase (Robertson and Stinard, Theor. Appl. Genet. 82: in press). These results suggested the *Mu-A2* locus represents a transposon capable of regulating *Mu1*. Other studies also identified genetic loci controlling *a1-mum2* mutability; these elements, termed *Cy* (Schnable and Peterson, Mol. Gen. Genet. 215:317-321, 1989), and *Mu-R1* (Chomet et al., Genetics 129:261-270, 1991) may be the same genetic element as *Mu-A2*.

The same strain used to define *Mu-A2* genetically was also used to identify and isolate a specific genomic DNA fragment that cosegregated with *a1-mum2* mutability (Qin and Ellingboe, Mol. Gen. Genet. 224:357-363, 1990; Qin et al., Genetics 129:845-854, 1991). An increase in the copy number of this genomic fragment correlated with an increased degree of mutability. Taken together, these data indicated the cloned fragment contains the *Mu-A2* locus. Nucleotide sequence analysis identified a *Mu* element within this fragment based on its high degree of homology to previously described *Mu* elements in the regions of the terminal inverted repeats (Qin et al., Genetics 129:845-854, 1991). The complete 4942 base pair (bp) nucleotide sequence of *Mu-A2* is contained in the EMBL sequence database (accession number X62251).

Sequence analysis of *Mu-A2* revealed two ATG-initiated open reading frames (ORF) that could encode a polypeptide of more than 100 amino acids in length. The largest of these is a 612 codon ORF (ORF612, from nt 970 to nt 2805) known from Northern hybridization analysis to be transcribed into a 3.4 kilobase (kb) mRNA, denoted transcript "L" (Qin and Ellingboe, Mol. Gen.

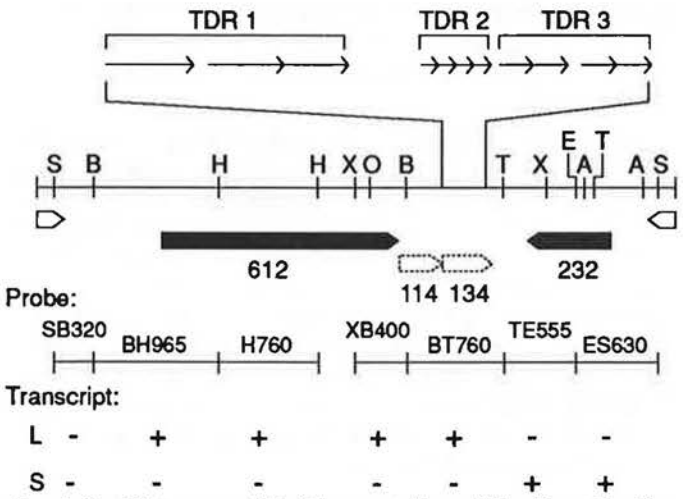


Figure 1. The 4942nt sequence of *Mu-A2* is represented by a solid line with restriction sites indicated for *Sac*I (S), *Bam*HI (B), *Xba*I (X), *Xho*I (O), *Stu*I (T), *Eco*RI (E), and *Sal*I (A). Open polygons show terminal inverted repeats, solid polygons show ORFs beginning with ATG initiation codons, and dotted polygons show ORFs lacking an ATG codon. Numbers under each ORF indicate codon length. Polygons point in the 5' to 3' direction of the element they represent. The expanded area comprises 338nt; arrows represent tandem direct repeats. All elements are drawn to scale. Hybridization probes in Northern analyses were random-primer labeled and are identified by appropriate restriction enzyme letter codes followed by numbers indicating fragment nucleotide length. Hybridization of a particular probe fragment to either transcript "L" or transcript "S" (see text) is denoted as "+" or "-", respectively.

Genet. 224:357-363, 1991; Figs. 1 and 2). This transcript is present specifically in plants derived from mutable kernels in a cross of *Mu-A2* and wildtype strains (Qin and Ellingboe, Mol. Gen. Genet. 224:357-363, 1990; this report). Two additional ORFs (ORF115, from nt 2806 to nt 3145, and ORF134, from nt 3146 to nt 3547) are located immediately downstream of ORF612 and may be present on the same 3.4kb transcript (Fig. 2). Neither ORF115 nor ORF134 contains an initiation codon; thus, their expression as protein would depend on RNA splicing or a translational mechanism that attached this coding information to ORF612. Also, RNA splicing in the region upstream of ORF612 may enable transcription to initiate from an ATG codon located 5' to this ORF, thus extending the reading frame. A second ATG-initiated ORF (ORF232, from nt 4477 to nt 3782) is transcribed on the opposite strand from ORF612 into a 900bp mRNA, denoted transcript "S" (Qin and Ellingboe, Mol. Gen. Genet. 224:357-363, 1990; Fig. 1). Neither transcript "L" nor transcript "S" specifies a polypeptide homologous to any sequence in the Protein Identification Resource database. An interesting feature of ORF134 is the clustering of three different tandem direct repeat (TDR) sequences comprised of 55nt (TDR1), 11nt (TDR2), and 27nt (TDR3) (Fig. 1). These repeats are situated so that the 3'nt of ORF115 is the 5' end of the first repeated sequence, TDR1 (Fig.1). The repeated sequences are not similar to direct repeats found in *Mu1*, *Mu1.7*, or *rcy:Mu7*.

To date, *Mu-A2*, *Mu-R1*, and *Cy* all have been shown to provide transposition function to an independent *Mu* element. Because *Mu-A2* and *Mu-R1* were isolated by analysis of the same *a1-mum2* allele, and because molecular analysis showed these two elements have identical restriction maps (Chomet et al., Genetics 129:261-270, 1991; Qin et al., Genetics 129:845-854, 1991; this report), they most likely represent the same regulatory locus. Comparison of the nucleotide sequence of *Mu-A2* with that of *Mu9*, a *Mu* element isolated from the *bz2::mu9* allele (Hershberger et al., Proc. Natl. Acad. Sci. USA 88:10198-10202, 1991) reveals the near-identity of these two elements. *Mu-A2* and *Mu9* differ in sequence at only three nucleotide positions, suggesting that these two elements are the same and implying that *Mu9* also is capable of providing transposition function to *Mu1*. Northern analyses of both *Mu-R1* and *Mu9* identified transcripts similar in size to those derived from *Mu-A2*. For reasons that are not clear, discrepancies exist in the reported sizes of the large transcript (3.4kb for *Mu-A2*; 2.9kb for *Mu9*, 2.5kb for *Mu-R1*). Differences in RNA size estimation may explain some or all of these discrepancies. Taken together, the data suggest that *Mu-A2*, *Mu-R1*, and *Mu9* are nearly identical transposons capable of the autonomous regulation of *Mu1*. The relationship of these regulatory loci to *Mu* elements other than *Mu1* remains to be discovered. However, because the family of *Mu* elements shares homology in regions of the terminal inverted repeats, it is predicted that the regulatory function of *Mu-A2* will extend to the entire transposon family.

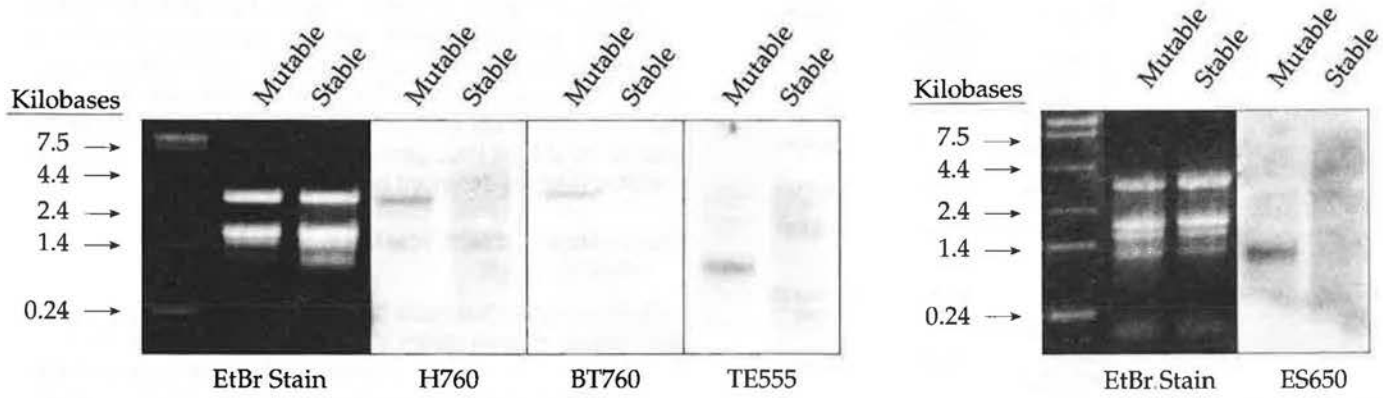


Figure 2. Transcription of *Mu-A2*. Total RNA isolated from seedlings of mutable or stable *a1-mum2* progeny in a cross showing 1:1 segregation for the mutability trait was separated by electrophoresis in denaturing agarose gels. *Mu-A2* transcripts were detected using the indicated restriction fragments (see Fig. 1) as random-primer labeled probes in Northern hybridization analyses. In each case, transcripts were detected only in RNA from mutable plants. Molecular weight standards are the 0.24-9.5Kb ladder from Gibco-BRL.

Transposon tagging the *su1* locus

--M. J. Scanlon, M. G. James, P. S. Stinard, D. S. Robertson and A. M. Myers

We are interested in transposon tagging and cloning genes involved in endosperm development. Included among the kernel mutants grown in our 1991 nursery were plants from seven putative Mutator-tagged *su1* allelic families identified by D. S. Robertson. The laboratory designations of this allelic series are given below:

*su1-Mu*2412* *su1-Mu*7110*
*su1-Mu*8064* *su1-Mu*5167*
*su1-Mu*3217* *su1-Mu*4582*
*su1-Mu*3162*

Southern blot analysis of genomic DNA derived from *su1-Mu*2412* seedlings has revealed a *Mu1*-tagged 2.2Kb *Hin*dIII fragment that cosegregates with the *su1* phenotype. This family also cosegregates a 4.0Kb *Mu1* homologous *Eco*RI fragment. The 2.2Kb *Hin*dIII genomic fragment has recently been cloned into phage lambda NM1149 and purified. Efforts are now underway to isolate genomic DNA flanking the *Mu1* insertion of this clone. Flanking DNA will then be used as a molecular probe in Northern analyses of *Su1* transcript, cloning of the wildtype cDNA, and RFLP mapping of putative QTL's for kernel size.

Seven novel defective kernel mutants from Robertson's Mutator stocks

--M. J. Scanlon, M. G. James, P. S. Stinard and D. S. Robertson

We announce the naming of seven previously undescribed putative *Mu*-induced defective kernel mutants. Each mutant was placed to chromosome arm via the use of the appropriate B-A translocation. Additional map location data has been generated for five of these mutants using either the waxy-9 series of translocations, linkage to chromosome marker genes and/or RFLP linkage data as indicated in Table 1. Each mutant has tested negative for allelism to all previously described defective kernel mutants located on its respective chromosome arm. The seven mutants are given the names *ptd1*, *ptd2*, *emp1*, *emp2*, *dsc1*, *ren1* and *ren2*.

Table 1. Seven *Mu*-induced *dek* mutants.

Name	Former designation	Phenotype	Map information	Lab of Origin
ptd1	*dek-Mu*1568*	pitted endosperm, lethal	1L; (a)	Robertson
ptd2	*dek-Mu*3193*	pitted endosperm, lethal	7L; (b)	Robertson
emp1	*dek-Mu*2045*	empty pericarp, germless	1S; (c)	Robertson
emp2	*dek-Mu*1047*	empty pericarp, germless	2L; (d)	Robertson
dsc1	*dek-Mu*2058*	discolored endosperm, germless	4S; (e)	Robertson
ren1	*dek-Mu*807*	reduced endosperm, opaque, lethal	5L; (f)	Shepherd (Du Pont)
ren2	*dek-Mu*326*	reduced endosperm, lethal	7L; (g)	Shepherd (Du Pont)

(a) linkage to *Kn1*, *bm2* and *bz2* in MNL 65:11.
(b) MNL 65:11.
(c) linkage to *wx1* T1-9(8918) in MNL 65:11.
(d) linkage to *wx1* T2-9d and linkage to *w3* in mapping note by James et al., this issue. RFLP linkage in Lee et al., MNL 65:10.
(e) MNL 65:11.
(f) RFLP linkage in Lee et al., MNL 65:10.
(g) RFLP linkage in mapping note by James et al., this issue.

A correlation between endosperm and seedling phenotypic variation in an allelic series of *et1* mutants

--M. S. Scanlon, P. S. Stinard, M. G. James and D. S. Robertson

An earlier report (MNL 64:11) described allelic variation in the endosperm phenotypes of our putative mutator-tagged alleles of the *et1* locus. Currently our study has identified 11 independently induced *et1* alleles (see mapping article by James et al., this issue), which display great variation in the degree of etching and/or shrinkage of the endosperm. In addition to the endosperm phenotype, the *et1* allele is pleiotropic and confers a virescent seedling phenotype. In order to investigate the variation in the seedling virescence of these alleles, representative Mutator-tagged *et1* endosperm variants were sandbench tested. The results are given in Table 1; the non-Mutator induced standard *et1* allele was included for the sake of comparison.

Table 1. Kernel and seedling phenotypes of putative *Mu*-induced *et1* alleles.

Mutant designation	Kernel phenotype	Degree of etching	Seedling phenotype
et standard	"normal" etched	moderate	virescent
*et1-Mu*3328* (a)	severely etched	extreme	virescence extreme, albino
*et1-Mu*2320* (a)	severely etched	extreme	virescence extreme, albino
*et1-Mu*24* (a)	mild shrunken	none to slight	mostly green, pale stripes
*et1-Mu*2457* (a)	severely shrunken	moderate	virescent
*et1-Mu*2320* (b)	small, sugary, defective	kernel is shriveled	lethal
*et1-Mu*2162* (b)	small, sugary, defective	kernel is shriveled	lethal

(a) data from heterozygotes with standard etched (*et1/et1-Mu*)
(b) data from homozygous mutant kernels (*et1-Mu/et1-Mu*)

These results indicate that the severity of the virescent phenotype may be correlated with the degree of etching and not the shrunkenness of the endosperm. Despite the extreme albino phenotype of the *et1-Mu*3328* and *et1-Mu*2320* seedlings grown from severely etched seed, these seedlings do eventually become green, and thus are truly virescent. Cox (MNL 42:52-56) reported similar results from seedlings grown from severely etched kernels. The lethality of the *et1-Mu*2320* and *et1-Mu*2162* sugary defective kernels is attributed to the absence of a germ. The small defective kernel phenotype is seen on self-pollinated ears of these two alleles only. It should be noted that variation in seedling and endosperm phenotypes is exhibited within, as well as between, *et1-Mu* allelic families. Possible causes of the phenotypic variations described herein include: variable expression of *et1* due to different Mutator element insertion sites or *Mu*-induced deletions; genetic background effects, although each of the alleles described has been outcrossed to standard Q60 and standard B70 backgrounds for at least three generations; the presence of a modifier of etched (*Met*) as described by Cox in MNL 40:39-42.

Orange pericarp contains more niacin

--Allen D. Wright

Evidence exists that niacin biosynthesis in maize is via oxidative degradation of tryptophan (Tarr and Arditti, Plant Physiol. 69:553-556). The tryptophan synthase defective mutant orange pericarp would be a useful tool to test for alternative biosynthetic routes. As a preliminary test, five kernels each of orange pericarp and its normal sib from a near isogenic Mo17 15:1 ear were analyzed for niacin by Tomas Guillarte at Johns Hopkins University using a radiometric microbial assay for niacin (Guillarte, J. Nutr. Biochem. 2:334-336). The mutant contained more niacin than its

normal sib when expressed on a per kernel basis and on a per gram basis. These results indicate that maize may be competent for niacin biosynthesis through a pathway that does not require tryptophan.

	Niacin content ± SE	
	as ug/kernel	as ug/g
o/p	4.78±0.23	16.29±0.62
N	3.93±0.77	12.91±2.41

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_vp2-5080_, a new mutable allele of _vp2_ with large revertant sectors

--Philip S. Stinard and Brent Buckner

A recessive pale yellow endosperm mutant with yellow revertant endosperm sectors was found segregating on the selfed ear of a plant (84-5080-13) grown from the outcross to standard of a putative Mutator-induced *wx* mutant, *wx-Mus2j*. When planted in the sandbench, pale yellow mutable kernels gave rise to albino seedlings with a heavy mutability pattern of large (many cells in width) and small (one to a few cells in width) longitudinal green sectors. This mutant was given the designation *wmut*-5080*.

Allele tests of *wmut*-5080* to known white endosperm/albino seedling mutants, including *al*, *cl*, *lw1*, *lw2*, *vp2*, *vp5*, *vp9*, *w3*, *y9*, and *y10*, proved negative. Crosses of *wmut*-5080* heterozygotes by a set of B-A translocations involving 19 of the 20 maize chromosome arms failed to uncover the mutant phenotype. The simpler methods of locating the mutant to chromosome having failed, *wmut*-5080* heterozygotes were crossed to a series of *wx* marked translocations with breakpoints near the centromeres of the non-9 chromosomes. The F1's were selfed and outcrossed to standard *wx* testers. Only selfed ears of the F1 between *wmut*-5080* and *wx* T5-9(4817) (5L.06, 9S.07) showed evidence of linkage (repulsion) between *wmut*-5080* and *wx*. The outcross to *wx* of one of these ears was grown in Don Robertson's 1990-91 winter nursery, the resulting plants self pollinated, and the selfed ears scored for semisterility, *wx*, and *wmut*-5080*. The results of this linkage test are reported in Table 1, and indicate tight linkage between *wmut*-5080* and T5-9(4817) (p=1.8±0.8cM).

Table 1. Three-point linkage data for *wx* T5-9(4817) *vp2-5080*. Testcross: (*wx* T5-9(4817) *Vp2 / Wx* n *vp2-5080*) X *wx wx* n n *Vp2 Vp2*

Reg.	Phenotype	No.	Totals
0	*wx* T +	126	
	+ n *vp*	135	261
1	*wx* n *vp*	2	
	+ T +	3	5
2	*wx* T *vp*	2	
	+ n +	2	4
1+2	*wx* n +	1	
	+ T *vp*	0	1

% recombination wx--T5-9(4817)=2.2±0.9
% recombination T5-9(4817)--*vp2-5080*=1.8±0.8

Elsewhere in the 1990-91 winter nursery, *wmut*-5080* was allele tested with the chromosome 5 mutants *vp2* (a repeat of an earlier allele test, this time using the Maize Stock Center's *vp2* stock) and *ps*. This time, the allele test with *vp2* proved positive. (A check of the pedigree of the *vp2* stock we had used for the earlier negative allele test revealed ambiguities that rendered its genetic identity suspect.) We have given *wmut*-5080* the designation *vp2-5080*.

During the course of propagating and mapping *vp2-5080*, we found that expression of this mutant varied widely from mutable to stable, from viviparous to dormant, from pale yellow to nearly white endosperm, and from albino seedling to pastel seedling. We have not crossed *vp2-5080* into isogenic lines to determine the cause of these variations. However, variation in dormancy and seedling phenotype has been observed in certain alleles of *vp9* and *w3* (Robertson, J. Hered. 66:67-74, 1975), and may be due to segregating genetic modifiers. Similar, or perhaps identical modifiers may be at work in *vp2-5080*.

The only other known mutable allele of *vp2*, *vp2-grmos* (Robertson, ibid.), produces large revertant sectors in both endosperm and seedling. We note the similarity in mutability pattern between *vp2-5080* and *vp2-grmos*, but it remains an open question as to whether this pattern of mutability is inherent to mutable alleles of *vp2*.

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Transcriptional reactivation of inactive *Spm* elements in the presence of *Spm-w* elements

--Vladimir Kolosha and Nina Fedoroff

The 8.3kb maize suppressor-mutator (*Spm*) transposable element comprises a single transcription unit and its primary transcript gives rise to several processed, polyadenylated mRNAs by alternative splicing (Masson et al., Genetics 177:117, 1987; Masson et al., Cell 58:755, 1989). The mRNAs potentially encode at least 4 different polypeptides, some with extensive common domains (Masson et al., 1989). Two element-encoded proteins, designated TnpA and TnpD, have been shown to be required for transposition of the element (Frey et al., EMBO J. 9:4037, 1990; Masson et al., Pl. Cell 3:73, 1991). The structure of the *tnpA* and *tnpD* transcripts is shown in Figure 1, along with the structure of the element. The 68kDa DNA-binding TnpA protein is encoded by the most abundant transcript of the element (Pereira et al., EMBO J. 5:835, 1986; Gierl et al., EMBO J. 7:4045, 1988). The TnpD protein is encoded entirely within the first intron of the *tnpA* transcript, which contains two long open reading frames (ORF1 and ORF2) and is expressed from a rare, alternatively spliced dicistronic transcript which also codes for TnpA (Masson et al.,

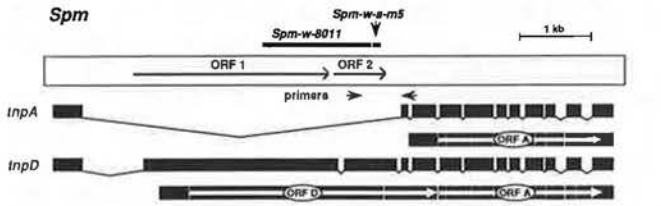


Figure 1. A diagrammatic representation of the *Spm* transposable element. The exon structure and ORFs of the two element-encoded transcripts for which functions have been established, *tnpA* and *tnpD*, are represented below the diagram. Located within the first intron of the *tnpA* transcript are two large open reading frames (ORF1 and ORF2) of which ORFD, the coding sequence for TnpD, is comprised. ORFA, the TnpA coding sequence, is encoded by the last 10 exons and is encoded in both the *tnpA* and *tnpD* transcripts. Bars just above the diagram show the location and extent of the deletions in the *Spm-w-8011* and *Spm-w-a-m5* elements. Arrows below the diagram show the location of the PCR primers.

1989). *Spm* elements are subject to both positive and negative regulation. *Spm* can be inactivated by a heritable, reversible genetic mechanism (Fedoroff and Banks, Genetics 120:559, 1988). Inactivation is correlated with increased methylation of C residues both upstream and downstream of the element's transcription start site. McClintock reported that an inactive *Spm* (*Spm-i*) element is subject to transient genetic reactivation in the presence of an active element, suggesting the existence of a positive autoregulatory gene on the element (McClintock, CIW Yrbk 57:415, 1957). Two *Spm-w* elements with deletions eliminating part of the TnpD coding sequence have been shown to retain the ability to trans-activate an inactive element genetically (Fedoroff and Banks, 1988; Fedoroff, Genetics 121:591, 1989).

We showed that *tnpA* transcripts are more abundant in plants containing both an inactive element and a trans-activating *Spm-w* element than in plants with only one of the two elements (Banks et al., Genes Dev. 2:1364, 1988). In these experiments, carried out before the element's minor, alternatively spliced transcripts had been identified and characterized (Masson et al., 1989), transcripts of the trans-activating element were not distinguished from those of the activated element. By making use of inactive and trans-activating elements which can be distinguished structurally, we show here that an *Spm-i* is transcribed in the presence of an active one. We have used two different *Spm-w* elements, both of which have internal deletions, as the trans-activating element. The structure of the standard *Spm* (*Spm-s*) and the *Spm-w* elements is shown in Figure 1. The *Spm-w-8011* element has a 1.7kb internal deletion commencing in ORF1 and ending in ORF2, while the *Spm-w-a-m5* element has a 144bp deletion near the 3'end of ORF2 (Masson et al., 1987; Masson et al., 1989). The inactive element is an *Spm-s* element inserted at a site just upstream of the *a* gene (Masson et al., 1987). The element has undergone spontaneous genetic inactivation, which we have previously shown to be associated with an increase in the extent of methylation of C residues in the immediate vicinity of the element's transcription start site (Fedoroff and Banks, 1988; Banks et al., 1988; Banks and Fedoroff, Dev. Genet. 10:425, 1989).

Several size classes of transcripts with homology to the *Spm* element can be detected in maize plants with active elements (Masson et al., 1989). The most abundant transcript encoded by the intact standard *Spm* element is the approximately 2.4kb *tnpA* transcript (Figure 2, lane 6). Plants with active elements also have larger and smaller transcripts that show homology to the element. Two size classes of large transcripts have been detected (Masson et al., 1989). From careful measurements of relative mobility, the sizes of these transcripts are presently estimated to be 6.0 and 6.7kb. The shorter of these transcripts therefore corresponds in length to the previously identified *tnpC* and *tnpD* transcripts, while the longer exceeds this length and may represent a partially processed transcript. Short transcripts with homology to the *Spm* element are also abundant in maize plants. Many short transcripts have been cloned and found to be processed transcripts of internally deleted, transposition-defective elements. Transcripts with the length expected for the previously described *tnpB* transcript are not detectable by blot hybridization (Masson et al., 1989).

None of the large-element-encoded transcripts can be detected by blot hybridization in RNA isolated from plants with an *Spm-i* element (Figure 2, lane 1), but a small amount of *tnpA* transcript can be detected in many such plants. Short *Spm*-homologous transcripts are as abundant in plants with an *Spm-i* as in those with an active element. In RNA isolated from plants containing the *Spm-w-8011* element as the only genetically active element, the two largest detectable *Spm*-homologous transcripts have estimated lengths of 5.2 and 4.4kb (Figure 2, lanes 2 and 3). These size estimates are in reasonably good agreement with the anticipated length of the large transcripts from the deleted element. The length of the *tnpA* transcript is indistinguishable between RNA preparations from plants with the *Spm-w-8011* and *Spm-s* elements. This suggests that processing of transcripts of the *Spm-w-8011* element is not affected by the deletion. The large transcripts from both the *Spm-s* and the *Spm-w-8011* elements are detectable in RNA from plants that contain both the *Spm-w-8011* element and an *Spm-i* element (Figure 2, lanes 4 and 5). The relative intensities of the bands corresponding to the transcripts of the deleted *Spm-w-8011* element and the *Spm-s* elements are approximately the same, suggesting that the large transcripts of the two elements are roughly comparable in abundance.

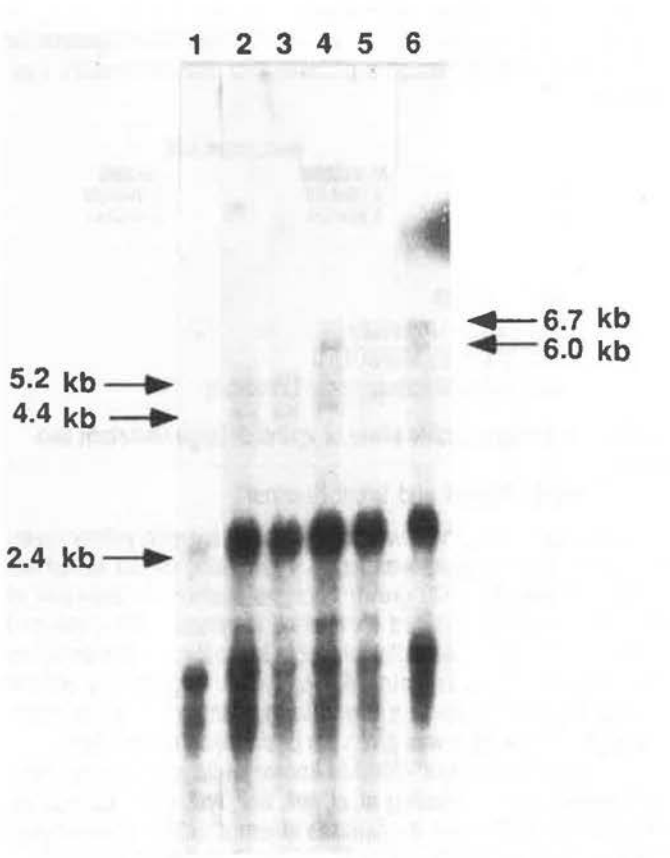


Figure 2. Northern blot hybridization analysis of polyA+ RNA isolated from plants containing an inactive element (lane 1), the *Spm-w-8011* element (lanes 2 and 3), both an inactive element and the *Spm-w-8011* element (lanes 4 and 5), and an *Spm-s* (lane 6). The blot was probed with a cloned, full-length *Spm* DNA fragment.

Similar results have been obtained with RNA from plants containing the *Spm-w-a-m5* element and an *Spm-i* element, using PCR amplification of internal cDNA fragments to distinguish between the trans-activating deleted element and the resident, standard *Spm-i* element. Primers flanked the deletion in the *Spm-w-a-m5* element in order to detect *tnpD* transcripts from the deleted and standard elements. These give rise to a 730bp DNA fragment from the completely processed *tnpD* transcript of

the standard element and a 590bp fragment from the deleted *Spm-w-a-m5* element (Figure 3A, lanes 1 and 5). Direct sequencing of the PCR-amplified fragments confirmed that they were derived from the completely processed *tnpD* transcript of the standard and deleted elements. Variable amounts of a longer, *Spm*-homologous 820bp fragment have been detected (Figure 3). The fragment was sequenced and determined to contain the third intron of the *tnpD* transcript. Because incorporation of DNAse into the reaction mixture prior to cDNA synthesis eliminated this band, we conclude that it is attributable to DNA contamination.

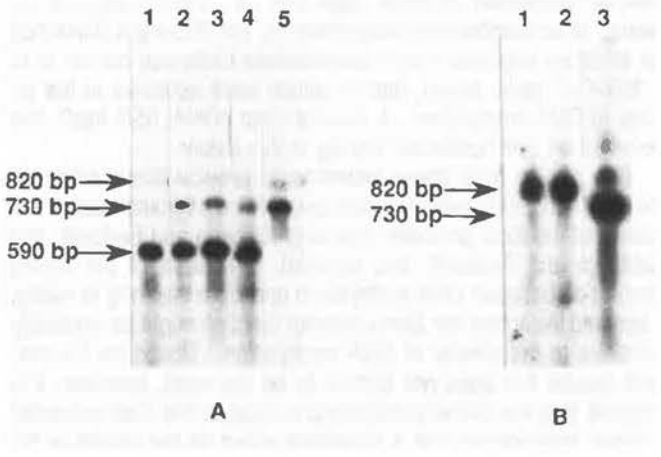


Figure 3. Southern blot hybridization analysis of fragments amplified by PCR from cDNA prepared by reverse transcription of polyA+ mRNAs. A. The RNAs were isolated from plants with an *Spm-w-a-m5* element (lane 1), both an *Spm-w-a-m5* element and an *Spm-i* element (lanes 2, 3 and 4) and an *Spm-s* element (lane 5). B. RNA was isolated from plants with an inactive *Spm* (lanes 1 and 2) or an active *Spm* (lane 3). The blot was probed with a DNA fragment homologous to ORF2.

Using the extremely sensitive PCR amplification reaction, we have detected only the 590bp fragment expected for the *tnpD* transcript of the *Spm-w-a-m5* element in plants containing this element as the only genetically active *Spm* (Figure 3A, lane 1). Moreover, the 730bp fragment expected from transcripts of the standard *Spm* element was detected in only 1 of seven different plants containing an *Spm-i* element. In RNA isolated from 6 of the 7 plants, the only fragment that could be amplified was the 820bp fragment derived from contaminating genomic DNA (Figure 3B, lanes 1 and 2). The fragment expected for the *tnpD* transcript of the standard *Spm* element was detected in the seventh plant with two different primer pairs. Since the plants used in the present experiments contained an unstably inactive element, it is likely that the element in this plant had undergone spontaneous reactivation. By contrast, plants containing both an inactive *Spm-s* and the *Spm-w-a-m5* invariably gave cDNA preparations from which both the 730bp and 590bp fragments could be amplified (Figure 3, lanes 2-4). This implies that *tnpD* transcripts of both elements are present in such plants. The 590bp fragment characteristic of the *tnpD* transcript of the *Spm-w-a-m5* element was generally more abundant in such amplification reactions than the 730bp fragment expected from the *Spm-s tnpD* transcript. But because the abundance of a given fragment among the final amplification products is influenced by its length, we cannot conclude that the transcript of the deleted element is more abundant than that of the intact element.

To obtain a rough estimate of the extent to which the inactive element had been reactivated in plants containing an *Spm-w* element, we quantified the amount of *tnpA* transcript relative to the amount of actin transcript by scanning Northern blots (Shah et al., J. Mol. Appl. Genet. 2:111, 1983). The abundance of *tnpA* transcripts in six plants with a single *Spm-i* element ranged from 2% to 30% (average of 12±10%) of its abundance in a plant containing a single *Spm-s* element. Three plants with an *Spm-w-8011* element contained an average of 35±20% as much *tnpA* transcript as plants with a standard element. The abundance of the *tnpA* transcript in RNA from plants containing a single copy of the *Spm-w-a-m5* element is 70±35% of its abundance in RNA from plants with a single *Spm-s* element. Eight pairwise comparisons were done between sibling plants containing only the *Spm-w-8011* element and both the *Spm-w-8011* element and an *Spm-i* element. The plants with both elements contained 2.8 (±0.8) times as much *tnpA* transcript as plants containing only a weak element. Similar pairwise comparisons between mRNA preparations from eight pairs of plants containing either only the *Spm-w-a-m5* element or both the *Spm-i* element and an inactive element yielded a *tnpA* transcript ratio of 1.8 (±0.5). Although the plant-to-plant variation in the abundance of element-specific transcripts is considerable, the results of these experiments suggest that the *tnpA* transcript is about as abundant in plants with an *Spm-w-a-m5* element and somewhat less abundant in plants with an *Spm-w-8011* element than in plants with a standard element. Whether this disparity is attributable to a difference in element transcription or in the stability of the transcript is not known. Since neither deletion alters the structure of the *tnpA* transcript, the effect on the abundance of the transcript may be indirect. Plants with an unstably inactive element contain low, but detectable amounts of *tnpA* transcript. The 2-3-fold greater amount of *tnpA* transcript in plants with both an inactive *Spm* element and a trans-activating *Spm-w* element suggests that the inactive element is fully active in the presence of the *Spm-w* element.

The results of present experiments provide direct evidence that transcripts of an *Spm-i* element are more abundant in plants that also contain an active element. We conclude that the capacity of an active *Spm* element to reactivate an inactive one, first inferred from genetic experiments, is detectable at the transcriptional level. We have previously reported that unstably inactive elements are more extensively methylated than active elements and we have noted that the extent of methylation is markedly lower in plants containing a trans-activating *Spm-w* element (Banks et al., 1988). These results imply an interaction between a gene product encoded by the *Spm-w* element and the inactive element. They do not permit us to distinguish between a direct transcription factor-like effect and an indirect effect mediated by a reduction in methylation.

Regulation of *Spm* promoter activity by the *Spm*-encoded *tnpA* gene product and DNA methylation

--Douglas Cook and Nina Fedoroff

The maize suppressor-mutator transposable element (*Spm*) can exist in both an active and an inactive form. Inactive *Spm* elements differ from active ones by being methylated at certain cytosine residues surrounding the element's transcription start site (Banks et al., Genes Dev. 2:1364-1380, 1988; Banks and Fedo-

roff, Dev. Genet. 10:425-437, 1989). Specifically, DNA methylation 5' of the transcription start site, in the so-called upstream control region (UCR), is correlated with element inactivity, while further DNA methylation 3' of the transcription start site, in the so-called downstream control region (DCR), is correlated with stably inactive elements. Despite the strong correlation between DNA methylation and *Spm* inactivity, it remains uncertain whether DNA methylation has a role in regulating *Spm* function. The major *Spm*-encoded gene product, TnpA, has been implicated in the reactivation of inactive elements (Banks et al., Genes Dev. 2:1364-1380, 1988), an event that is correlated with demethylation. Gierl et al. (EMBO J. 7:4045-4053, 1988) have shown that TnpA binds, in vitro, to an oligonucleotide with homology to the 12bp subterminal repeats located in the 5' UCR and in the extreme 3' end of the element, and binding affinity is reduced upon DNA methylation. Binding of TnpA to the subterminal repeats is likely responsible for the *Spm*-suppressible phenotype observed with certain *Spm* insertion alleles, since TnpA could suppress expression of a β-glucuronidase gene that had been engineered to include the TnpA binding site (Grant et al., EMBO J. 9:2029-2035, 1990). The present experiments provide direct evidence that, at high levels, TnpA can also down regulate the *Spm* promoter (see also the report by Schläppi and Fedoroff, this volume).

In this study, we employed a transient assay to define the promoter of *Spm* transcription and to examine the effect of DNA methylation and *Spm*-encoded gene products on promoter activity. The 5' end of the *Spm* element was able to drive expression of the luciferase reporter gene following introduction into tobacco cell suspensions by microprojectile bombardment. When normalized to a co-bombarded chloramphenicol acetyl transferase (CAT) internal control, luciferase activity provided a means to quantify *Spm* promoter activity.

Sequences essential for at least basal promoter activity are contained within the 5' 220bp fragment of the element, coincident with the UCR and including the proposed transcription initiation site at nucleotide 209 (Periera et al., EMBO J. 5:835-841, 1986). Inclusion of the unmethylated GC-rich first exon, which is common to all known *Spm* transcripts and contains the largest number of methylatable sites, has no effect on promoter activity. As measured in the transient expression assay, *Spm* promoter strength is weak, being roughly two to three orders of magnitude below that of the CaMV 35S promoter.

The entire UCR fragment is likely to be important for promoter activity. Deletion of the 5'-terminal inverted repeat was associated with a small but reproducible increase in promoter strength. As the extent of the deletion into the UCR increased, there was a corresponding gradual decrease in promoter activity. Deletion to within 146bp of the transcription start site eliminated half of the promoter activity, while deletion to within 50bp eliminated 90% of the measurable activity. No detectable promoter activity remained upon deletion to -35bp.

DNA methylation inhibited transient gene expression. When plasmid DNA was methylated in vitro by a CpG-specific DNA methylase, transient gene expression was inhibited by three-fold as compared to a non-methylated control. This negative effect on gene expression, however, was not specific to the *Spm* promoter since it was also observed upon methylation of a promoterless luciferase gene control and of a CaMV 35S-CAT gene fusion. In no case did DNA methylation completely eliminate *Spm* promoter activity, even when the methylated DNA contained the highly methylatable, GC-rich first exon.

We have also determined that expression of the *tnpA* cDNA from a strong promoter suppresses *Spm* promoter activity. When *Spm*-luciferase gene fusions were co-bombarded with a CaMV 35S-*tnpA* cDNA, expression from the *Spm* promoter was completely blocked, even when the 35S-*tnpA* plasmid was diluted 100-fold relative to the *Spm*-luciferase plasmid. Not surprisingly, this effect appears to be due to an interaction between TnpA and sequences within the promoter-containing UCR fragment which includes nine potential TnpA binding sites. Step-wise deletion of 5' UCR sequences removed progressively more TnpA binding sites but did not eliminate suppression by TnpA until the last subterminal repeat was removed. This result is consistent with the hypothesis that suppression of the *Spm* promoter results from an interaction between TnpA and the 12bp subterminal repeats. In co-bombardment experiments, the 35S-*tnpA* cDNA had no effect on expression of a promoterless luciferase control or of a 35S-CAT gene fusion, both of which were sensitive to the effects of DNA methylation. A second *Spm* cDNA, 35S-*tnpD*, had no effect on *Spm*-luciferase activity in this assay.

The results from these experiments provide direct evidence that *Spm*-encoded gene products and DNA methylation can affect activity of the *Spm* promoter (see also Kolosha and Fedoroff, and Schläppi and Fedoroff, this volume). Because of the strong correlation between DNA methylation and *Spm* inactivity in maize, it seemed likely that the *Spm* promoter function might be unusually sensitive to the effects of DNA methylation. Based on the present results this does not appear to be the case, however, it is possible that the extrachromosomal context of the *Spm*-promoter in these experiments had a significant effect on the results of the assay. We have shown that at high levels TnpA can down-regulate the activity of the *Spm* promoter. It is uncertain, however, whether this constitutes an autoregulatory mechanism in vivo. In the model proposed by Fedoroff et al. (MNL, this volume), we suggest that low levels of TnpA may stimulate *Spm* activity, while high levels of TnpA would inhibit transcription (also see Gierl et al., EMBO J. 7:4045-4053, 1988). Depending on the strength of the *Spm* promoter in vivo and its sensitivity to TnpA concentration, this may provide a mechanism by which to regulate TnpA protein levels and hence *Spm* function.

Promotion of early *Spm* transposition and repression of *Spm* transcription by TnpA in transgenic tobacco

--Michael Schläppi and Nina Fedoroff

The maize suppressor-mutator (*Spm*) transposable element has been transformed into tobacco cells and a visual assay for *Spm* activity using a *dSpm* cloned between the CaMV 35S promoter and the β-glucuronidase (GUS) gene has been developed (Masson and Fedoroff, PNAS 86:2219, 1989; Masson et al., Plant Cell 3:73, 1991). Callus lines exhibiting different frequencies of *Spm* excision were obtained from an initially highly active callus (Fedoroff, unpublished). This phenomenon resembles the epigenetic inactivation of *Spm* in maize, correlating with differences in methylation of sequences immediately upstream (a region termed the upstream control region or UCR) and downstream (a region termed the downstream control region or DCR) of the element's transcription start site (Banks et al., Genes Dev. 2:1364, 1988; Banks and Fedoroff, Dev. Genet. 10:425, 1989). By Southern blot analysis and copy number reconstitutions, we have selected a callus line, #8, containing 1-2 copies of *Spm* and 1 copy of the

GUS excision assay sequence. Different plantlets regenerating from callus #8 that showed different *Spm* activity were analysed with the methylation sensitive restriction enzymes *Eco*109 (to detect UCR methylation) and *Sal*I (to detect DCR methylation). Plant 8#47 exhibited very low *Spm* activity and revealed partial *Sal*I methylation in the DCR whereas plant 8#49 showed high *Spm* activity and was fully cleavable at the *Sal*I sites in the DCR (Schläppi, unpublished). DNA from both plants was fully cleavable and hence unmethylated at the *Eco*109 site in the UCR.

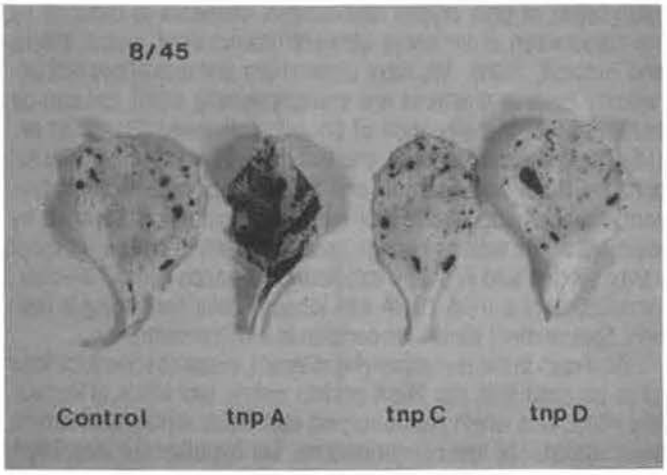


Figure 1. Histochemical visualization of *dSpm* excision events in *Spm*-containing transgenic tobacco plants retransformed with different element-encoded cDNAs driven by the CaMV 35S promoter. Excision sectors were visualized by staining with X-Gluc.

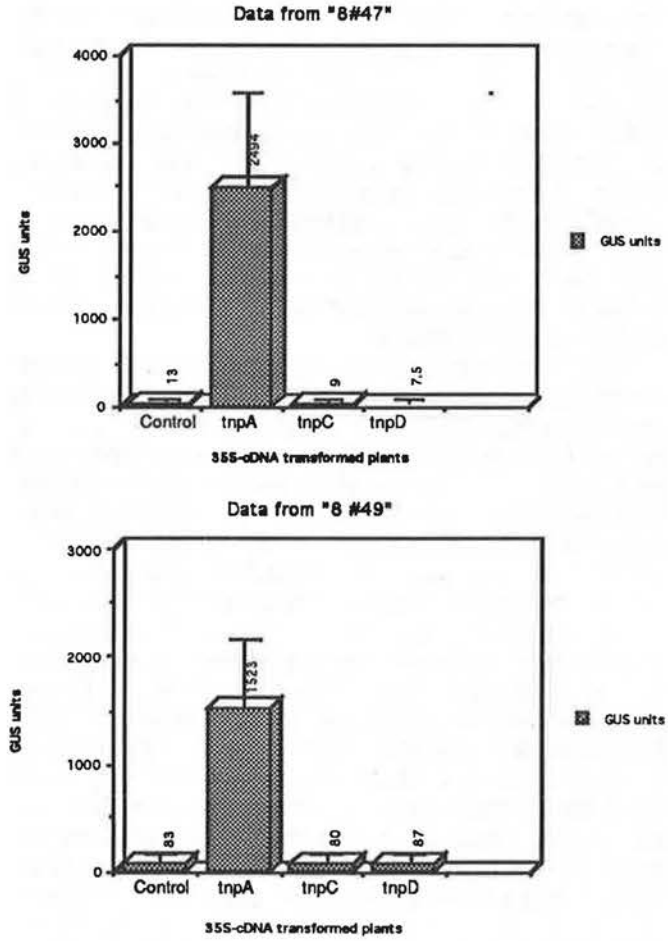


Figure 2. Quantification of β-glucuronidase (GUS) activity in *Spm*-containing transgenic tobacco plants retransformed with different *Spm*-encoded cDNAs. The *Spm* sequence of plant 8#47 was partially methylated in the DCR region, the *Spm* sequence of plant 8#49 was unmethylated. The GUS activity is given in arbitrary units per µg of measured protein.

Different *Spm* element-encoded transcripts produced by alternative splicing have been cloned recently (Masson et al., Cell 58:755, 1989). Two transcripts, designated *tnpA* and *tnpD*, have been shown to be necessary and sufficient for the element's transposition in transgenic tobacco (Frey et al., EMBO J. 9:4037, 1990; Masson et al., Plant Cell 3:73, 1991). In order to investigate the effect of different element-encoded gene products on the activity of unmethylated and partially methylated *Spm* elements in transgenic tobacco, we retransformed the previously selected plants of callus line #8 with either *tnpA*, *tnpC*, or *tnpD* cDNA under the control of the CaMV 35S promoter and the nopaline synthase terminator by *A. tumefaciens* mediated gene transfer. Control plants were retransformed by *A. tumefaciens* without any *Spm*-specific sequences. Introduction of *tnpA* into plants with either methylated or unmethylated *Spm* sequences leads to a dramatic increase of early excision events whereas *tnpC* and *tnpD* have no effect as compared to the control (see Figure 1). Control and *tnpC*- or *tnpD*-retransformed plants of the partially methylated parent plant 8#47 had roughly 10X fewer GUS sectors and 10X less GUS activity than the equivalents of the unmethylated parent plant 8#49, but similarly large sectors and GUS activities when retransformed with *tnpA* (see Figure 2). The same phenomenon was observed when plants from the callus line #8 were

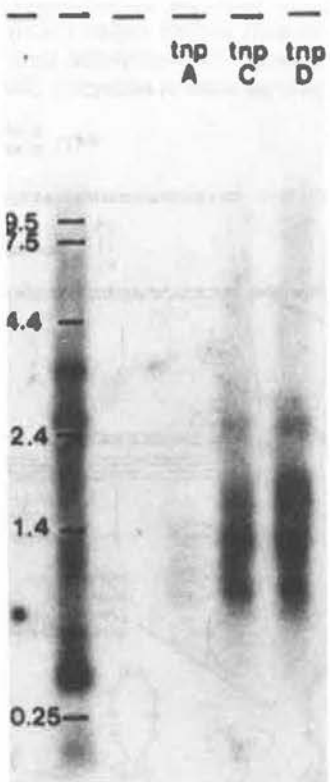


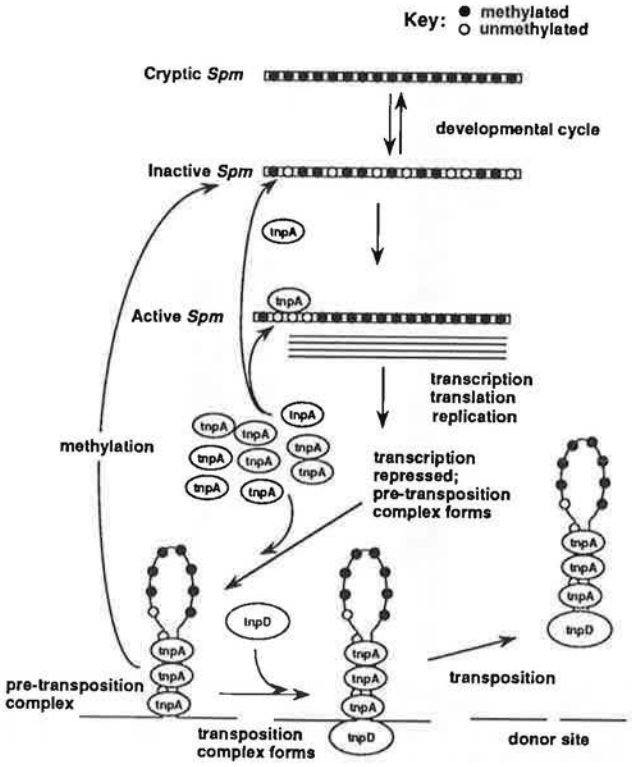
Figure 3. Northern blot hybridization analysis of polyA+ RNA isolated from *Spm*-containing transgenic tobacco plant 8#49 retransformed with either *tnpA*, *tnpC*, or *tnpD* cDNA. The blot was probed with a DNA fragment homologous to the UCR and DCR region of *Spm* that was not present in the cDNA constructs.

crossed with tobacco plants containing only *tnpA*, *tnpC*, or *tnpD* cDNA. These results suggest that the production of a certain amount of TnpA protein might be the rate-limiting step for *Spm* transposition and that partial DCR methylation has no influence on the transposition process mediated by TnpA protein in trans.

The effect of the introduced cDNAs on transcription of *Spm* in transgenic tobacco was investigated by Northern blot analysis. Over-expression of *tnpA* from the CaMV 35S promoter leads to the reduction of *Spm* transcripts to undetectable levels (see Figure 3). This preliminary result is in agreement with the repression of transient expression of 5'-*Spm*-luciferase fusions in the presence of *tnpA* cDNA (Cook and Fedoroff, this volume). This observation suggests that *Spm* transcription is repressed upon formation of the pre-transposition complex due to saturation binding of TnpA to the subterminal repeats (see also Fedoroff et al., this volume).

Regulation of the maize *Spm* transposable element: an hypothesis
--Nina Fedoroff, Michael Schläppi, Douglas Cook and Vladimir Kolosha

The accompanying diagram illustrates our present view of the manner in which *Spm* transcription and translation are regulated. In previous reports from this laboratory, we showed that inactive *Spm* elements can exist in an extremely stably inactive form which we designated cryptic and a much less stably inactive form that can be distinguished genetically by its ability to be reactivated upon introduction of an active *Spm* element (Fedoroff and Banks, Genetics 120:559, 1988; Banks et al.,Genes Dev. 2:1364, 1988; Fedoroff, Genetics 121:591, 1989). Cryptic elements are more extensively methylated within the GC-rich region of the first exon, termed the downstream control region (DCR), than are less stably inactive elements. The methylation levels of both cryptic and inactive elements are lower in embryonic DNA than in DNA of seedlings or mature plants, suggesting that there is a reduction in methylation early in development (Banks and Fedoroff, Dev. Genet. 10:425, 1989). Inactive *Spm* elements exhibit different methylation levels in different plant parts during development and methylation of both cryptic and inactive elements is reduced by the introduction of an active element (Banks et al., 1988; Banks and Fedoroff, 1989). We have shown here and elsewhere that genetically inactive elements are transcriptionally silent, but can be transcribed in the presence of an active element (Banks et al., 1988; Kolosha and Fedoroff, this volume). The accompanying reports by Cook and Fedoroff and by Schläppi and Fedoroff present evidence suggesting that the *Spm* promoter is inhibited by methylation, as well as by high levels of TnpA in both a transient assay system and in stably transformed tobacco cells. However, introduction of a *tnpA* cDNA into tobacco cells containing a resident *Spm* element stimulates excision of a *dSpm* element.



As shown in the accompanying diagram, these observations lead us to propose that the TnpA protein exerts two kinds of regulatory effect, one which promotes and one which represses element transcription. At low concentrations, we hypothesize that TnpA stimulates element expression. Whether it acts directly as a transcription factor or indirectly by preventing methylation has not yet been determined, although we have suggested that it may do both (Masson et al., Genetics 177:117, 1987; Banks et al., 1988). Nor do we know whether TnpA acts directly by binding to methylatable sites or indirectly by maintaining the element in a conformation unfavorable for methylation. Both types of interference may play a role, since the *tnpA* protein's recognition sequences are confined to the element's subterminal repetitive regions and contain methylatable C residues (Masson et al., 1987; Gierl et al., EMBO J. 7:4045, 1988), yet changes in methylation of the adjacent GC-rich DCR are also observed in reactivating elements. Gierl et al. (1988) have reported that methylation of C residues within the TnpA binding sequence reduces the affinity of the binding site for the protein, which may be an important factor in regulating element expression.

Thus it appears that element methylation is low early in development (as illustrated in the top part of the diagram), perhaps by virtue of a general suppression of DNA methylation. Remethylation and TnpA binding may both occur early in development, but the presence of TnpA protein clearly favors reduced methylation and element expression, whatever the molecular mechanism. As the concentration of TnpA increases, it may occupy progressively more of the binding sites, eventually reaching a level at which further transcription is repressed and the element ends are brought together in a complex, which we have designated the "pre-transposition complex" in the accompanying diagram (Frey et al., EMBO J., 12:4037, 1990; Masson et al., Plant Cell 3:73, 1991). The present model elaborates on previous models (Masson et al., 1987; Banks et al., 1988; Gierl et al., 1988; Frey et al., 1990; Masson et al., 1991) in postulating that the pre-transposition complex has one of two alternative fates. The first is to bind the TnpD protein, which has been shown to be required for transposition together with the TnpA protein (Frey et al., 1990; Masson et al., 1991), forming a transposition complex that leads to excision of the element from the donor site. The second is to to be remethylated and return to an inactive form. Element inactivation late in development can be monitored by the appearance of sectors on kernels of the appropriate genetic constitution (containing, for example, an *Spm*-suppressible allele and a trans-acting *Spm*) and

occurs at a high frequency (Masson et al., 1987; also unpublished observations).

We propose that the rate-limiting step in the transposition process is the formation of the pre-transposition complex for the several reasons that follow. First, we have observed that the introduction of a *tnpA* cDNA into tobacco cells with an *Spm* element increases the excision frequency, but introduction of a *tnpD* cDNA does not (Schläppi and Fedoroff, this volume). This implies that it is the TnpA protein that is rate limiting in these cells. Moreover, deletions that encroach on the subterminal repeats of the *Spm* element reduce the transposition frequency and delay it in development (Schwarz-Sommer et al.,EMBO J. 10:2439, 1985), suggesting that an element missing some of the TnpA binding sites is less efficient in forming the pre-transposition complex, perhaps requiring higher TnpA concentrations than elements with the full complement of subterminal repeats. The third and perhaps most compelling argument is a genetic one, based on the phenotypes of *Spm*-suppressible alleles. The predominant phenotype of plants containing both an *Spm* and such an *Spm*-suppressible *dSpm* insertion mutation is that characteristic of the *dSpm* mutant in the presence of an active element. This suggests that the active *Spm* element generally remains in a transcriptionally active form, because when the trans-acting *Spm* element is lost (by virtue of chromosome breakage, for example), the phenotype of the monitoring *dSpm* insertion mutation immediately exhibits the phenotype that is characteristic of it in the absence of a trans-acting element. This implies that the concentration of TnpA drops rapidly when the element is removed or inactivated and that complexes that form between TnpA and the element are not stable. Thus transcriptional inactivation of the element should be perceptible as sectors of the "no *Spm*" phenotype. As noted above, such sectors are frequent, although generally small for a fully active *Spm*, implying that inactivation is a late event in development. Moreover, they never include revertant wildtype sectors within their perimeter, which implies that *dSpm* transposition does not occur when the element is not expressed. Thus we suggest that transcriptional inhibition must either be followed immediately by transposition or rapidly result in complete inactivation of the element by virtue of the instability of the complex or the TnpA protein (or both). This, in turn, would release the element in a methylatable, TnpA-free form.

An observation made by one of us is perhaps relevant in this context (NF, unpublished). In a study conducted some time ago, it was noted that a majority of kernels containing an active *Spm* element and a monitoring *dSpm* mutation and selected for evidence of *Spm* inactivation gave plants with a transposed (inactive or reactivating) element, rather than an inactive *Spm* at its original location (these occur as well, but very infrequently). Although it cannot be ruled out that the reinsertion site is responsible for inactivation of the element, it is possible that the more frequent inactivation of newly transposed elements reflects the transcriptionally inactive structure of the transposition complex postulated here. A satisfying aspect of the present model is that it provides an explanation for the origin of the transcriptionally inactive, methylatable state as a by-product of the transposition process. Thus we perceive the transcriptional silencing of the element and the subsequent stabilization of the silent state by methylation as separate events. Moreover, it provides an explanation for the low frequency with which an active element undergoes germinal inactivation.

Note: the authors welcome your comments on this model.

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Somatic embryogenesis in S0, S1, and S2 generations of two maize populations

--Paul Pepo, Manjit S. Kang, Neeraja Kondapi and J. Ivan Dickson

Two maize populations developed in the LSU maize breeding program, viz., [(Mo17 x B73) x (L91R x Yellow Creole)] and [(Mo17 x B73) x (L331 x Yellow Creole)], hereafter referred to as L91R population and L331 population, respectively, were evaluated for their in vitro culturability and regeneration potential. The S0, S1, and S2 generations were subjected to tissue culture using the following procedures:

Seeds were surface-sterilized for 10 min. in a 0.2% aqueous mercurous chloride solution, rinsed overnight under running tap water, and re-sterilized for 5 min. in a 0.2% aqueous mercurous chloride solution, followed by several water rinses. Twenty five kernels of each generation were germinated. Aseptic seedlings were grown on a 1% agar-solidified medium containing the inorganic constituents of Murashige and Skoog (Physiol. Plant. 42:139-145, 1962), 3% sucrose, 26.7µM glycine, 4.1µM nicotinic acid, 2.4µM pyridoxine-HCl and 0.3µM thiamine-HCl.

Radicles were aseptically separated from plumules at scutellar nodes and the explants were cut into five, 2-3mm long pieces. These explants and intact, mature embryos were plated on MS medium. The pH was adjusted to 5.8 before autoclaving. Incubation was done at 26 C with 16/8 photoperiod.

The 2,4-D concentration varied between 1 and 5mg/L. After callus induction, meristematic segments were discarded; the remainder were transferred to the above culture medium for callus proliferation. To induce further differentiation, calli were subcultured on MS medium supplemented with different concentrations of 2,4-D and zeatin. Regenerated plantlets were transferred to hormone-free medium for root development.

Results (Table 1) indicated that the callus induction frequency for the two populations ranged from 4.7 to 60.9%. The highest

Table 1. Callus initiation and regeneration for L331 and L91R populations

		Callus induction (%)		Regeneration (%)	
Explant	Inbred stage	L331	L91R	L331	L91R
Radicle (R)	S0	60.9	41.1	4.75	1.51
	S1	58.0	32.0	4.45	0.0
	S2	56.0	23.9	0.0	0.0
	Mean	58.3	33.4	2.70	0.59
Plumule (P)	S0	41.9	30.1	9.94	3.05
	S1	39.0	23.9	6.65	1.26
	S2	38.9	19.0	4.43	0.0
	Mean	40.0	25.1	6.95	1.65
Embryo (E)	S0	40.0	9.8	5.00	2.17
	S1	38.2	7.2	2.06	1.20
	S2	35.8	4.7	1.05	0.0
	Mean	38.0	7.5	2.75	1.25
Mean: R+P+E	S0	50.9 (601/1181)	33.3 (228/1016)	7.03 (83/1181)	2.26 (23/1016)
	S1	47.9 (590/1232)	26.1 (233/893)	4.79 (59/1232)	0.67 (6/893)
	S2	46.9 (577/1230)	19.9 (134/673)	3.92 (25/1230)	0.0 (0/673)

frequency of callus formation was exhibited by the radicle tissue and the lowest by the embryo. In both populations, callus induction frequency decreased as amount of homozygosity increased, which suggested that callus induction was controlled primarily by dominant gene action.

Explants differed in plant regeneration percentages in both populations. A maximum number of plants was regenerated from plumule in the L331 population. No plant regeneration was noted in the S2 generation of L91R population. Results suggested that plant regeneration would be increasingly more difficult in the inbred generations and that prior to embarking on a tissue culture-based breeding program, responsive genotypes must be identified for use in a breeding program.

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Selection of somaclonal variants resistant to toxin of *Fusarium moniliforme* from a maize inbred susceptible to ear rot

--Hongsheng Zhou

Two month old callus cultures induced from a maize inbred Zhong017 susceptible to ear rot were used as experimental material to select disease resistant mutants. Pathotoxin of *Fusarium moniliforme* was put in MS2 medium from sublethal (0.01%) to completely lethal concentration (0.1%). After several months of selection, cultures 10 times more resistant than the original were obtained. The identification of resistance to ear rot of regenerate plants will be determined next year.

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The b-32 protein is a functional ribosome inactivating protein

--M. Maddaloni, S. Lohmer[1], I. Mauri[2], E. Martegani[2], F. Salamini[1], R. Thompson[1] and M. Motto
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b-32 is one of the more abundant albumins accumulated specifically in the endosperm of maize developing kernels. The gene encoding this protein is under the control of the *O2* and *O6* loci. In a recent work we have reported that the product of the *O2* locus is a strong transcriptional activator of the promoter of the *b-32* gene (Lohmer et al., EMBO J. 10:617-624, 1991).

A search for homology to available protein sequences revealed that the b-32 peptide has a significant homology to the protein-synthesis inhibitor II from barley grains (Lohmer et al., quoted). This barley protein belongs to a group of proteins whose members, collectively called ribosome inactivating proteins (RIPs), act as severe inhibitors of eukaryotic protein synthesis by enzymatically cleaving the N-glycosyl bond of a specific adenine in the 28S rRNA, such that the elongation factor 2 binds inefficiently.

b-32 is shown to be a functional RIP by the criteria of inhibition of an in vitro rabbit reticulocyte cell-free translation system, and by specific N-glycosydase activity on 28S rRNA (Maddaloni et al., J. Genet. Breed., 1991, in press). To demonstrate in vivo the RIP activity of b-32 we have transformed the yeast *Saccharomyces cerevisiae* with a plasmid expressing the b-32 coding region under the control of an inducible promoter. The results showed that i) after induction the yeast cells arrest their growth, ii) protein synthesis is severely inhibited, and iii) the 28S rRNA is modified in the expected way. In addition, we have noted that in an in vitro transient gene expression assay with tobacco protoplasts as described in Lohmer et al. (quoted), the b-32 protein potentiates the expression of a reporter gene driven by a zein promoter.

Taken together, these experiments suggest that the b-32 protein might have a dual function. The first function appears to be related to the protection of the developing endosperm and/or the germinating seedlings against pathogen attacks. This role is also supported by the empirical observations that *o2* and *o6* mutant kernels are more vulnerable than wildtype kernels to ear-rotting diseases. The second function seems to be associated with the translation machinery of the maize endosperm in enhancing zein synthesis. In conclusion, the notion that b-32 shows a RIP activity on lower eukaryotes, opens up new possibilities in using this protein as a protectant agent against fungal infections in tissues and plants different than those in which its synthesis naturally occurs.

Regulation of zein synthesis by nitrogen nutrition in cultured endosperms

--C. Balconi, E. Rizzi, M. Motto, F. Salamini and R. Thompson

The maize gene opaque-2 (*O2*) controls the expression in developing endosperm of the 22kDa zeins and of an abundant cytosolic albumin, termed b-32 (Motto et al., Oxford Surv. Plant Mol. Cell. Biol. 6:67-114, 1989). Plants homozygous for the *o2* mutation typically show a 50-70% reduction in zein, especially in the polypeptides of the 22kDa zein class. This reduction results from a lower rate of transcription of zein genes, particularly those of the 22kDa class. Available data have also suggested the importance of source supply in regulating zein synthesis in maize endosperm (Balconi et al., Plant Sci. 73:1-9, 1991). To study the effects of N nutrition upon endosperm development, normal and *o2* maize endosperms of the W22 inbred line were grown in vitro for 5 days on a solid medium containing different N amounts (Table 1). A comparison between endosperms grown to maturity in vitro and under field conditions (in vivo) was also done.

Table 1. Culture media. All media contained 0.4mg/l thiamine, 100mg/l inositol, 30g/l sucrose, 8g/l agar and salts as described in Nitsch and Nitsch (media 1-2-3) or without ammonium nitrate (media 4-5-6).

Medium	Ammonium nitrate (mg/l)	Asparagine (g/l)
1	720	-
2	720	0.02
3	720	4
4	-	-
5	-	0.02
6	-	4

Increased dry weight accumulation by cultured endosperms and increased asparagine concentrations in the medium were correlated. The same trend was observed for zein accumulation; in particular, the *o2* endosperms cultured in vitro on the medium with the highest N content reached a zein level virtually identical to that of normal endosperms grown in vitro on the same medium, and in vivo at maturity (Fig. 1). Moreover, while *o2* endosperms grown in vivo were much less efficient in accumulating the 22kDa fraction of zeins, when cultured in vitro they were capable of restoring a normal synthesis of 22kDa zein fraction. Analyses of zein and b-32 mRNA levels in the wildtype and *o2* endosperms also suggested that the expression of these RNAs is dependent upon the amounts of N supplied to the media. In conclusion, our data indicate that the zein synthesis system is subject to regulation by the levels of N

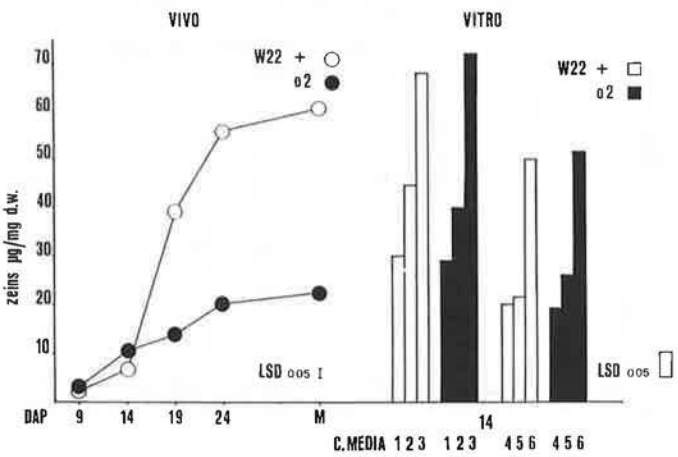

Figure 1. Zein content (ug/mg d.w.) of the W22 wildtype (○, □) and W22 mutant (○, ■) endosperms grown in vivo and in vitro.

provision, in particular asparagine, at the early stages of endosperm development. However, the short duration of endosperm culturing did not allow us to check if this feature is also maintained at later stages of development. Because zein gene expression and zein accumulation appeared to be regulated in response to amino acid supply at the level of gene transcription and/or stability of the mRNAs, this result raises the possibility that this type of metabolic control may be active at early stages of endosperm development, when N provision is not a limiting factor. One possibility is that the *O2* gene, and consequently the expression of the 22kDa zeins, may be activated only later during the phase of rapid zein synthesis (from 15 to 30 DAP) and under conditions of amino acid starvation. In the yeast *Saccharomyces cerevisiae*, it is well documented that the expression of the transcription factor *GCN4* increases under conditions of amino acid starvation (reviewed in Hinnebusch, Microbiol. Rev. 52:248-273, 1988). Alternatively, at early stages of endosperm development it can be postulated that a transcriptional activator dependent on N-availability promotes zein and b-32 gene expression. These questions are currently being addressed in our laboratory.

Analysis of the nitrogen nutritional requirements of the opaque-6 mutant

--C. Balconi, E. Rizzi, F. Salamini, R. Thompson and M. Motto

The opaque-6 (*o6*) mutation of maize, which causes seedling lethality and interferes with the synthesis of zein and b-32 proteins in the endosperm of developing kernels, has been demonstrated to be a proline requiring mutant functionally allelic to proline-1 (Manzocchi et al., Theor. Appl. Genet. 72:778-781, 1986). We have further analysed, by in vitro culture, the growth requirements of the wildtype and mutant *o6* embryos of the B37 inbred line. Mature wildtype and mutant seeds were sterilized and germinated in Petri dishes for 48h at 27 C. Embryos were then dissected and placed in test tubes containing different media, as described in Table 1. On the "basic" growth medium (without amino acid) the wildtype seedlings showed a normal phenotype, whereas the mutant seedlings exhibited the described abnormalities of leaves, reduced growth and lethality at the second leaf stage (Manzocchi et al., Theor. Appl. Genet. 72:778-781, 196). *o6* mutant embryos cultivated on basic medium (N) added with several specific amino acids (Table 1) showed a complete recovery of the

Table 1. Growth of excised embryos of mutants *o6 o6* B37 and wildtype *O6 O6* B37 on basic (salts as described in Nitsch and Nitsch, Science 163:85-87, 1969) and enriched media (basic medium added with 320mg/l amino acid). All media contained 0.4mg/l thiamine, 100mg/l inositol, 20g/l sucrose and 8g/l agar.

Growth medium	Mutant	Wildtype
Basic (N)	-	+
N + Arg (Arginine)	+	+
N + Asn (Asparagine)	+	+
N + Gly (Glycine)	+	+
N + Leu (Leucine)	+	+
N + Met (Methionine)	+	+
N + Pro (Proline)	+	+
N + Trp (Tryptophan)	+	+
N + Ala (Alanine)	-	+
N + Asp (Aspartic Acid)	-	+
N + Cys (Cystein)	-	+
N + Gln (Glutamine)	-	+
N + Glu (Glutamic Acid)	-	+
N + His (Histidine)	-	+
N + Ile (Isoleucine)	-	+
N + Lys (Lysine)	-	+
N + Phe (Phenylalanine)	-	+
N + Ser (Serine)	-	+
N + Thr (Threonine)	-	+
N + Tyr (Tyrosine)	-	+
N + Val (Valine)	-	+

normal phenotype, suggesting that this mutation is possibly related to a defect affecting not only proline biosynthesis but more generally all N-metabolism of the seed.

In vitro response to somatic embryogenesis of Lo inbred lines of maize

--F. Locatelli, M. Bertolini and E. Lupotto

Genetic transformation of maize has been recently obtained by the use of the biolistic approach applied to embryogenic regenerable suspension (Gordon-Kamm et al., Plant Cell 2:603-618, 1990). In other cereals, PEG-mediated transformations applied to regenerable protoplast systems have led to transformed regenerated plants. Also in this case, however, the establishment of embryogenic suspension cultures was the keypoint for the successful application of transformation techniques. To date, maize can be regenerated from several genotypes, but optimized friable embryogenic cultures are derived only from a few elite genotypes; among these A188 and its cross with B73 are the most widely used. In addition, other inbreds such as B79 (Lupotto and Lusardi, Maydica 33:163-177, 1988), may give origin to friable embryogenic calli, but in these cases suspension cultures have not been established.

In view of an efficient application of transformation techniques to maize cells, the identification of genotypes particularly suitable for tissue culture in giving friable cultures, suspension cultures, and regenerable protoplast systems assumes particular relevance. Furthermore, cultures initiated from agronomically suitable genotypes will facilitate breeding procedures once a new genetic trait is introduced in regenerable cells by genetic transformation.

Since the study on B79 cultures, we have been interested in establishing optimized embryogenic cultures in various maize genotypes, such as inbreds produced at the Section in Bergamo and characterized by outstanding agronomical traits.

A recent series of Lo inbred lines has been released by our Institution; each of these lines is characterized by specific traits including tolerance to pests, diseases and viruses, early vigor, and strong root apparatus (Table 1). Among the lines chosen for this investigation, six (Lo876, Lo904, Lo950, Lo951, Lo964, and Lo1054) are related to the Iowa Stiff Stalk breeding group (SSS), and four (Lo881, Lo924, Lo1056, and Lo1059) are re-

Table 1. Lo inbred lines and their characteristics.

Lo inbred lines	Some major agronomical characteristics	In vitro response[c]
Lo876[a]	stalk rot resistance, stay-green, resistance to BYDV, MDMV, and MRDV	A
Lo881[b]	high yield, stay-green, long-ear, tolerance to plant density	A
Lo904[a]	high yield, low-ear insertion, early vigor	A
Lo924[b]	high yield, stalk quality, early vigor	A
Lo950[a]	high yield, stay-green, stalk quality, tolerance to MDMV	B
Lo951[a]	high yield, stay-green, stalk quality, tolerance to MDMV	B
Lo964[a]	high yield, stay-green, low-ear insertion, stalk quality, tolerance to MDMV	A
Lo1054[a]	high yield, stay-green, stalk quality, strong root system	B
*Lo1056[b]	high yield, stay-green, stalk quality, strong root system	B
*Lo1059[b]	high yield, stay-green, stalk quality, strong root system	B

[a]related to the Iowa Stiff Stalk Group
[b]related to the Lancaster or other breeding groups
[c]classes of in vitro response based on callus induction frequency (CIF) values: A = very low, CIF <10%; B = low, CIF 10-20%; C = medium, CIF 20-50%; D = high, CIF >50%
*not yet published

lated to the Lancaster or other breeding groups (Bertolini et al., Maydica 36:87-106, 1991). These lines were evaluated per se and in crosses with A188 and B79 for their responsiveness in tissue culture. The aim of the work was to establish embryogenic cultures from each genotype, to test their regeneration capability, and to assay their responsiveness as suspension cultures.

Callus cultures were induced from the scutellum of immature embryos on N6I medium (Lupotto and Lusardi, Maydica 33:163-177, 1988) in the presence of 2mg/l 2,4-D, propagated in very dim light (200 lux) conditions on the same medium in the presence of 1mg/l 2,4-D, and regenerated on Murashige and Skoog (MS) medium devoid of hormones in the light (3000 lux). With regard to the callus induction frequency (CIF) the genotypes were grouped in four classes of in vitro responsiveness: A = very low, CIF <10% of the explanted embryos; B = low, CIF 10-20%; C = medium, CIF 20-50%; and D = high, CIF >50%. As indicated in Table 1, Lo inbreds were considered non-responding genotypes in vitro. Callus induction frequency did not exceed the second class of responsiveness (A and B) when compared to inbreds highly responding in vitro, such as A188 and B79. A second parameter evaluated at the beginning of the third subculture after embryo explant was the embryogenic callus induction (ECI), determined by scoring under dissecting microscope for the presence/absence of sectors bearing somatic embryos at the surface. ECI also includes four classes of responsiveness ranging in the same values of percentage as CIF. No embryogenic, or occasionally very few stabilized callus cultures, were developed from Lo inbreds when selfed embryos were explanted. This holds true from greenhouse and field grown donor plants in two sets of experiments. Conversely, crosses of some Lo inbreds with A188 and B79 gave interesting results regarding callus induction, somatic embryogenesis, plant regeneration, and suspension culture establishment. As summarized in Table 2, crosses of various Lo to A188 and B79 inbreds, and their reciprocals, allowed inclusion of almost each genotype in the classes C and D, thus drastically enhancing their in vitro culturability. The five Los showing some callus induction per se, grouped in class B for CIF, improved up to the highest responding class D, in crosses with A188 or with B79. In all cases friable embryogenic calli were obtained, more typically type II calli in the crosses with A188. The five less responsive Lo inbreds (grouped

Table 2. In vitro responsiveness of crosses between Lo inbreds and A188 or B79.

	Class of responsiveness of Lo selfed		Class of responsiveness from crosses							
			LoxA188		A188xLo		LoxB79		B79xLo	
Genotype	CIF	ECI	CIF	ECI	CIF	ECI	CIF	ECI	CIF	ECI
Lo881	A	0	D	D	D	D	D	C	D	D
Lo876	A	0	D	D	D	C	D	B	n.t.	n.t.
Lo904	A	0	A	C	C	C	D	B	C	D
Lo924	A	0	D	C	D	C	D	D	D	D
Lo964	A	0	C	D	C	D	D	C	n.t.	n.t.
Lo950	B	0	C	D	D	D	D	D	D	C
Lo951	B	0	C	D	D	C	D	C	D	C
Lo1054	B	0	D	D	D	D	D	D	D	D
Lo1056	B	0	D	D	D	D	D	D	n.t.	n.t.
Lo1059	B	0	D	D	D	D	D	C	n.t.	n.t.

CIF = callus induction frequency; ECI = embryogenic callus induction (evaluated over the CIF value). Classes of responsiveness: A = CIF and ECI <10%; B = CIF and ECI 10-20%; C = CIF and ECI 20-50%; D = CIF and ECI >50%.

in class A) did improve consistently their performance, except in one case. The inbred Lo904 responded very poorly when used as female parent in the cross Lo904 x A188, while response was significantly improved when it was used as pollinator in the cross A188 x Lo904. In this specific case, the cross of Lo904 with B79 also gave favorable results in culture. Although callus cultures were more hard and compact than in the cross with A188, they were nevertheless more lasting and showed higher regenerative capability.

Friable, healthy growing calli at the third subculture of some of the crosses were then challenged as suspension culture. Finely dispersed suspensions were stably obtained in the following crosses: Lo1054 x A188, Lo904 x A188, Lo951 x A188 and their reciprocals. Interestingly, suspensions were also finely established from Lo904 in cross with A188; an unexpected result because of the very poor responsiveness of the callus culture.

The efficiency of plant regeneration was determined in each genotype. Calli samples of about 1g fresh weight tissue, in triplicate, were fragmented onto MS hormone-free-medium, in the light, and subcultured every 7 days in order to stimulate maximal plant regeneration. In this respect also the various genotypes differed consistently from each other and were grouped in classes of regenerability depending on the number of regenerated complete plantlets per gram fresh weight tissue. The best responding genotypes were Lo964, Lo950, Lo951, Lo1056, and Lo1059 in combination with A188, and Lo881, Lo1056, and Lo1059 in combination with B79. Their regenerative efficiency paralleled, or even exceeded, the regenerative capability of the cross between the two best responding genotypes A188 x B79. Suspension cultures derived from the three genotypes mentioned above are currently being utilized for protoplast isolation in order to establish an efficient regenerable protoplast system.

Comparison between cluster analyses from RFLP and pedigree data of inbred lines related to Stiff Stalk Synthetic heterotic group

–P. Ajmone Marsan, C. Livini, M. M. Messmer[1], E. Melchinger[1], P. Franceschini, G. Monfredini and M. Motto
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Several criteria have been used extensively to characterize important inbred lines and hybrids of maize. During the past decade a new class of molecular markers, named restriction fragment length polymorphisms (RFLPs), have been introduced. They allow the detection of differences between genotypes at the DNA level

(Helentjaris et al., Plant Mol. Biol. 5:109-118, 1985). Moreover, RFLPs have been used to produce well populated linkage maps (Helentjaris, Trends Genet. 3:217-221, 1987; Burr et al., Genetics 118:519-526, 1988) permitting a more detailed sampling of the genome. Objectives of this study were: 1) to determine the utility of RFLPs for estimation of genetic similarities among 16 maize inbred lines from Iowa Stiff Stalk Synthetic (BSSS) heterotic group and 2) to compare genetic similarities based on molecular markers with those based on pedigree information.

Sixteen BSSS maize inbred lines (Table 1) were chosen as selected directly from the BSSS population (B37, B73, B84, N28), or derived from lines originating from BSSS (B14A, B68, CM109, Lo999, A1, A2). A5, a selection recovered from B3, is not

Table 1. Inbreds used in the analysis of RFLP and pedigree data.

Lines[a]	Background[b]
B14A	(CuzcoxB14[8])rust. res. sel.[c]
B37	BSSS(HT)C0
B68	(41.2504BxB14[3]) sel.
B73	BSSS(HT)C5
B84	BSSS(HT)C7
CM109	V3xB14[2]
Lo950	Pioneer3183
Lo951	Pioneer3183
Lo999	(B37xteosinte)xB73
N28	Stiff Stalk Synthetic
A1	50% B14
A2	50% A1
A3	Commercial hybrid
A4	Commercial hybrid
A5	B3 recovered sel.
A8	Commercial hybrid

[a]Lines designated by A1 . . . A10 are private property.
[b]Anonymous (1989), Hallauer et al. (1983), Henderson (1984), and Guarneri (1990, personal communication).
[c]Power refers to m-1, where m is the number of backcross generations to the respective parent.

a direct derivative of the previous inbreds but has Reid Yellow Dent in its pedigree. Lo950, Lo951, A3, A4, A8 were derived from commercial hybrids between BSSS and Lancaster Sure Crop lines, yet based on their breeding behaviour they were classified to the BSSS heterotic group. These 16 lines were analyzed with 149 clone-enzyme combinations for their RFLP profile according to published protocols (Livini et al., Theor. Appl. Genet., 1992, in press). For each inbred line, genomic DNA was separately digested with two restriction enzymes (*Eco*RI and *Hin*dIII). Genetic similarity between pairs of lines was calculated from molecular data according to Dice similarity coefficient (GS) (Dice, Ecology 26:297-302, 1945). The coancestry coefficient (f) (Malecot, Les mathematiques de l'hérédité, Masson et Cies, Paris, 1948) was used to calculate the genetic distance between pairs or lines from pedigree data. Pedigree information of these lines was obtained primarily from Henderson (Maize Research and Breeders' Manual, No. 10, Illinois Foundation Seeds Inc., Champaign, IL, 1984), Bertolini et al. (Maydica 36:87-106, 1991), or from maize breeders working with these materials. Collected data were subjected to cluster analysis by using the average linkage (UPGMA) method. Simple correlations (r) were calculated between GS and f values for related pairs of lines ($f>0.10$).

The dendrogram of BSSS lines obtained from cluster analysis of RFLP data is shown in Figure 1. The BSSS lines clustered in two main groups: B14A-related lines (B14A, B68, A1, CM109, A2); and a group of nine lines subdivided into three subclusters, (a) B37 and Lo999, (b) B73 and lines derived from commercial hybrids (A3, A4, Lo950, Lo951, A8), and (c) B84, A5 and N28,

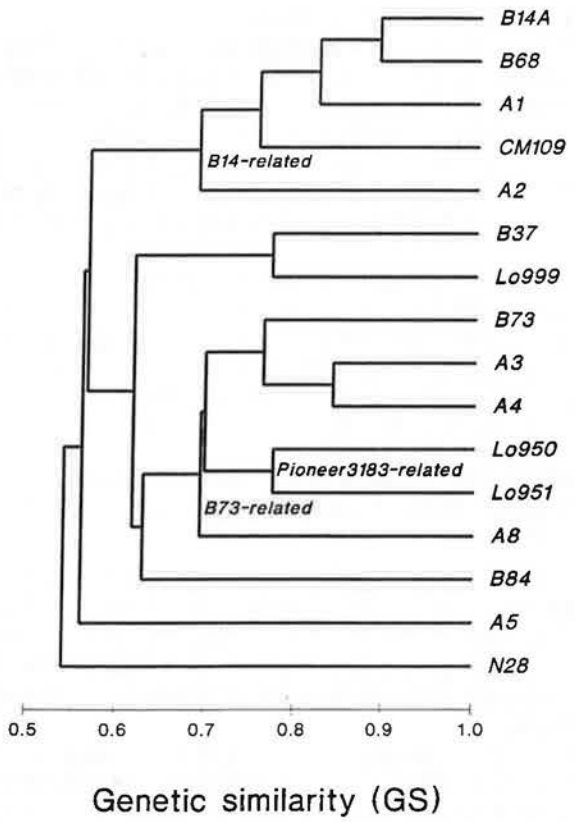

Figure 1. Association among BSSS related inbreds revealed by UPGMA cluster analysis of RFLP data.

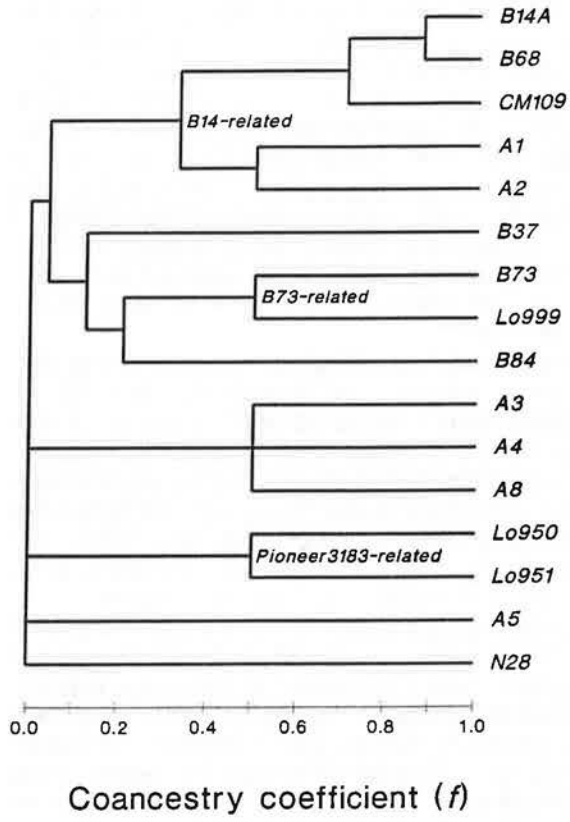

Figure 2. Association among BSSS related inbreds revealed by UPGMA cluster analysis of pedigree data.

did not cluster to any of these groups.

Cluster analyses based on pedigree (Fig. 2) and on RFLP (Fig. 1) data revealed similar associations among lines with minor discrepancies. RFLPs associated inbred lines according to known pedigrees (Table 1). Furthermore molecular analysis was able to classify inbreds derived from commercial hybrids having unavailable pedigrees. The correlation between GS and f values for 18 pairs of related (f>0.10) BSSS lines was highly significant with r=0.70.

The two indices are both valid for revealing relationships among the BSSS lines assayed. However, f values based on pedigree information explained only 50% of the variation of RFLP based GS values, suggesting that the latter offers a better tool for accurate quantification of the degree of relatedness between lines of known, uncertain or even unknown pedigree.

The b-32 protein is not encoded by the opaque-6 locus

--P. Ajmone Marsan, F. Salamini, P. Franceschini, G. Monfredini and M. Motto

Several mutations affecting the accumulation of maize seed storage proteins have been described (see Motto et al., Oxford Surv. Plant Mol. Cell. Biol. 6:87-114, 1989). Phenotypically these mutant kernels are easily recognized since they are soft and opaque in contrast to wildtype kernels that have hard, translucent endosperm.

One of these mutations, opaque-2 (*o2*), causes a partial to nearly complete elimination of the 22kDa class of zein proteins. The *o2* mutant is also unable to synthesize a number of non-zein polypeptides present in the wildtype endosperm, the most abundant of which is a 32kDa cytosolic albumin, termed b-32. Its expression during kernel development is temporarily and quantitatively coordinated with the deposition of storage proteins in endosperm tissue (Soave et al., Cell 27:403-410, 1991). The gene encoding the b-32 protein has been cloned and the complete amino acid sequence of this protein derived (Hartings et al., Plant Mol. Biol. 14:1031-1040, 1990). Furthermore, b-32 was shown to be a functional ribosome-inactivating protein and to interact with the translation machinery of the maize endosperm cell in enhancing zein synthesis (Maddaloni et al., J. Genet. Breed., in press, 1991). b-32 is coded by, or tightly linked to, the opaque-6 (*O6*) locus (Soave et al., Cell 27:403-410, 1981; Di Fonzo et al., Planta 167:587-594, 1986).

We have analysed the segregation pattern of a restriction fragment length polymorphism through Southern blots in seedlings derived from normal and *o6* kernels. In the case of identity of the two loci a perfect cosegregation was expected. Seedlings derived from kernels of B37 inbred line and from vitreous (*O6/O6* or *O6/o6*) and opaque (*o6/o6*) kernels of F2 ears in a B37 background were the sources of DNAs. A b-32 DNA insert derived from the full-length cDNA clone b-32.66 (Hartings et al., Plant Mol. Biol. 14;1031-1040, 1990) was used as a probe.

Southern hybridization of the b-32 probe to *Bam*HI digests of genomic DNA extracted from seedlings of B37 wildtype and opaque kernels revealed restriction fragment length polymorphisms. Thirty-five DNAs from the opaque seeds (*o6/o6*) were further analysed and two of them showed recombination between *O6* and *b-32* loci. Thirty-two DNAs from the vitreous seeds (*o6/o6* or *O6/o6*) were also analysed and one of them showed recombination between the two loci. Recombination frequency between *O6* and *b-32* loci, calculated according to the maximum likelihood method, was equal to 4.0±1.6%. This finding indicates that the *O6* locus does not encode the b-32 protein.

The *Bg* transposable element from *Zea mays* L. exhibits sequence homology with plant and *Drosophila* transposons

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

The controlling element system consisting of the two elements *Bg* and *rbg* (receptor of *Bg*) was originally shown to control the mutability of the *o2-m(r)* allele (Salamini, Mol. Gen. Genet. 179:497-507, 1980; Salamini, Cold Spring Harbor Symp. Quant. Biol. 45:467-476, 1981). The autonomous *Bg* element encodes functions required for transposition, while the non-autonomous *rbg* elements are deletion derivatives of the autonomous element which still bear the termini of *Bg*. *rbg* elements cannot promote their own excision, rather they transpose only in the presence of an active *Bg* element.

Both the autonomous *Bg* element and the receptor element have been isolated: the former at the waxy locus, the latter at the opaque-2 locus (Hartings et al., Mol. Gen. Genet. 227:91-96, 1991). The characterization of the elements at the sequence level has revealed a number of features. The elements generate an 8bp direct duplication of the target site upon transposition, and carry 5bp long terminal inverted repeats (CAGGG). The terminal repeats start with the nucleotides CA, like the repeats of other transposable elements, and in particular the *Ac* transposable element from maize. Similar inverted repeats, 8bp long target site duplications and the absence of a recognizable promoter sequence in the 5' part of the element are the major characteristics that *Bg* shares with the *Ac* transposable element. A direct comparison of the nucleotide sequences of the *Bg* and *Ac* elements however, did not reveal significant homologies.

Recently, Calvi et al. (Cell 66:465-471, 1991) and Feldmar and Kunze (EMBO J. 13:4003-4010, 1991) showed that the *Ac* ORFa protein sequence contains regions exhibiting homology with the deduced protein sequences of the *Tam3* transposable element from *Antirrhinum majus* and the *hobo* element from *Drosophila melanogaster*. The highest homology was found near the C-terminus of the proteins.

A computer search for coding regions, based on codon frequency statistics carried out on the entire *Bg* sequence, revealed the presence of a number of putative coding regions in the *Bg* element. These putative coding regions were screened for homology with a 57 amino acid long stretch, present in the C-terminal region of the *hobo* element, and showing high homology with the *Tam3* and

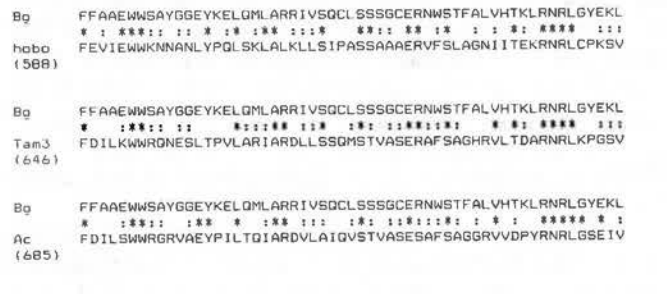

Figure 1. Alignment of *Bg*, *Ac*, *Tam3* and *hobo* amino acid sequences. * indicates identities, : indicates conservative changes. For the *Ac*, *Tam3* and *hobo* sequences, absolute positions are indicated.

Ac sequences, as indicated by Calvi and coworkers. Significant homology was found with one of the putative coding regions derived from the *Bg* sequence. This region encompasses nucleotides 3120 to 3959. Because no open reading frames can be found in the sequence 3' of position 3959, it is likely that the region showing homology is located in the C-terminal region of the putative *Bg* protein sequence. The deduced amino acid sequence of this region exhibits 31.6% of identity and 64.9% of similarity with the *Ac* sequence, 33.3% of identity and 66.6% of similarity with the *hobo* sequence, and 29.8% of identity and 66.6% of similarity with the *Tam3* sequence over a stretch of 57 amino acids (Fig. 1). No other homologies were found between the *Bg* sequence and the protein sequences of the other transposable elements.

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Physiological genetics of dominant dwarfs

--Rodney G. Winkler and Michael Freeling

D8 is a dominant gibberellin (GA) insensitive, anther-eared maize (*Zea mays* L.) dwarf mutant proposed to be involved in GA reception. In addition to *D8*, five other dominant, anther-eared maize dwarf mutants of varying severity are known. We show, by mapping and similarity of phenotype, that all six are likely alternative alleles at the *D8* locus. We define the phenotypic differences in GA response by three criteria: (a) ability to respond to exogenous GA, (b) ability to dwarf when GA synthesis is blocked, and (c) ability to respond to exogenous GA when endogenous GA synthesis is blocked. Although each allele specifies a different plant stature, from very mild to very severe dwarfism, each allele showed the same response to all three tests. Each allele is apparently saturated in its ability to respond to endogenous GA, but is able to dwarf when GA synthesis is blocked, and each allele can be returned to its "normal" dwarf phenotype when exogenous GA is applied. However, each allele sets a different maximum limit on growth. The fact that six independent alleles affect both GA responsiveness and dwarfing suggests that the wild type function of *D8* is growth retardation and that GA responsiveness and dwarfing are linked, but in a complex manner. Dominant alleles are particularly valuable in dissecting the structure and function of gene products. Molecular analysis of these six alleles should provide insight into the *D8* locus and the GA reception pathway.

Mapping *Lg3* using RFLP's

--Teresa J. Chan, John E. Fowler, and Michael Freeling

Liguleless-3 is a dominant mutation that alters the ligular region of maize. *Lg3* has been genetically mapped near the centromere of chromosome 3. However, its location on a particular arm has not yet been determined. Our intention is to map *Lg3* more accurately with respect to RFLP markers and eventually to use molecular probes to locate it on a chromosome arm.

The progeny analyzed were from an outcross of a *Lg3* heterozygote (linked to RFLP alleles designated here as "A") in a Mo17 background (linked to RFLP alleles designated here as "M"). DNA was extracted from individual plants using a miniprep method and cleaved using *Eco*RI. Southern Blot analysis of these samples using RFLP probes in the vicinity of the centromere of chromosome 3 was used to obtain the data presented in the following tables.

Table 1. Recombinant classes and number of progeny. Loci *UMC92-BNL8.35-UMC10-Lg3-NPI219*.

Progeny Type	# progeny
Parental (non-recombinant):	
A-A-A-*Lg3*-A	9
M-M-M-+-M	3
Single Recombination Classes:	
M-A-A-*Lg3*-A	1
A-M-M-+-M	0
M-M-A-*Lg3*-A	2
A-A-M-+-M	1
M-M-M-*Lg3*-A	0
A-A-A-+-M	0
M-M-M-+-A	1
A-A-A-*Lg3*-M	0
Double Recombination Classes Obtained:	
A-M-M-+-A	1
A-M-M-*Lg3*-A	1

Table 2. Additional plants assayed for recombination between loci.

Interval	# recombinants	# non-recombinants
BNL8.35-UMC10	2	21
UMC10-Lg3	0	51
Lg3-NPI219	0	4

The data presented above support the order of loci and map distances on chromosome 3 shown below. This order is different from that shown on the most recent RFLP map (Helentjaris lab, personal communication), however, it provides the fewest number of double recombinant progeny.

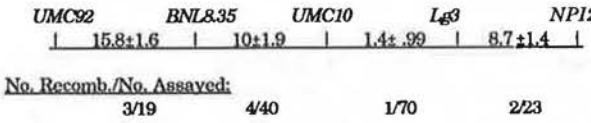

Figure 1. Genetic map predicted from data (with distances in cM).

The presence of two double-recombinant progeny is surprising, as interference would be expected to suppress these double recombinations. The high frequency of double recombination events may reflect the small sample size. However, the data could also be explained by negative interference influenced by the presence of the centromere. If this is the case, then we cannot assume that the map with the fewest number of double recombination events is necessarily correct. Further analysis of more individuals in this population as well as using plants aneuploid for the entire arm of 3S or 3L will hopefully resolve this question.

An anaerobic gene, which encodes an apparently non-glycolytic protein, shares sequence homology with *Mu1.7* and *Mu* related sequence-A

--Julie Vogel and Michael Freeling

We would like to provide further information about the characterization of an anaerobically expressed gene in maize roots, upon which our laboratory previously reported in this newsletter (MNL 63:38). Unfortunate DNA sequencing errors led to incorrect conclusions in the previous MNL report about the size, amino acid composition, and charge of the polypeptide encoded by this anaerobically expressed gene. With new, corrected DNA sequence data, we now wish to clarify the primary structural features of this gene and its predicted encoded protein.

This full-length cDNA, whose encoding gene we now denote *anp27*, hybridizes to a message of approximately 1.1kb that is in-

duced 2-3 fold in anaerobically treated seedling roots and shoots. No transcripts can be detected in any tissue of aerobically grown maize plants that we examined, and no message is present in either cold- or heat-shocked maize seedlings. Thus, expression of the *anp27* gene appears to be under specific transcriptional control; message is produced only under anaerobic conditions. This cDNA contains an open reading frame of 765 nucleotides, encoding a polypeptide of 255 amino acids with a predicted molecular mass of 27.5kDa. A cluster of proteins of this approximate size has been observed on two-dimensional gel fluorographs of proteins that have been in vivo-labelled under anaerobic conditions (unpublished data from this laboratory). We are currently attempting to identify which one of these low molecular weight polypeptides is encoded by the *anp27* gene. Although we haven't yet demonstrated, it is likely that ANP27 expression is also controlled at the level of translation, as has been seen for other maize anaerobic proteins.

The ANP27 protein appears to be both proline- (10%) and arginine- (10%) rich; yet, it does not show obvious sequence homology to known proteins rich in these amino acids. Although predicted to carry a net charge of -2, the distribution of charges within the molecule indicates two distinct charge domain regions: an acidic N-terminus (~60aa) and a highly basic middle region (~100aa). Protein sequence comparisons using current EMBL/Genbank databases reveal no obvious similarities to any other known proteins. Thus, this anaerobic protein still does not appear to represent a known glycolytic enzyme, and its identity (and function) remains unknown. Its small size and unusual primary structure are consistent with the intriguing possibility that this protein has a regulatory function during the anaerobic response.

Surprisingly, we found that the ANP27 cDNA shows high DNA sequence similarity to two other maize genomic sequences previously characterized: *Mu1.7* (Taylor and Walbot, Genetics 117:297, 1987) and *Mu* related sequence-A [denoted MRS-A by Talbert and Chandler, but which we denote herein as *MrsA*] (Talbert and Chandler, Mol. Biol. Evol. 5:519, 1988). Surprisingly, the ANP27 cDNA shares 60.8% DNA sequence identity with *MrsA*, within 716 nucleotides of overlap, and 60.7% identity with *Mu1.7*, within 606 nucleotides of overlap. This region of similarity encompasses nearly the entire protein-encoding portion of the ANP27 cDNA, but not the untranslated regions. The part of *Mu1.7* containing the homologous sequences encompasses the region from the beginning of the second internal direct repeat, through the 385bp "insertion" relative to the *Mu1* element, and ending 100bp beyond the *Mu1.7*-unique region (but before the inverted repeat end of the element). The region of sequence similarity within *MrsA* begins at the same point within the second internal direct repeat, extends contiguously through the *Mu1.7*-specific segment, and ends 200bp further downstream. Although the ANP27 cDNA is extremely [G+C]-rich (overall, it is 66% [G+C], and greater than 80% [G+C] in its middle region), the sequence similarity to the *Mu1.7* and *MrsA* elements (each ~70% [G+C] in the *Mu1.7*-unique segment) is mediated by true sequence homology, rather than just [G+C] content. From these comparisons, it is reasonable to propose that *anp27* is evolutionarily related to the *Mu1.7* element and to *MrsA*, with *anp27* having the most diverged sequence. What this reveals about genomic DNA rearrangements during maize evolution is not yet entirely clear.

The *Mu1.7*-unique segment of DNA has been shown to hybridize specifically to a 2.2-2.3kb transcript in several different (aerobic) tissues of both *Mu*-active and *Mu*-inactive lines (Taylor and Walbot, 1987; Talbert and Chandler, 1988), and this transcript is likely to be encoded by the *MrsA* locus (Talbert and Chandler). Although a cDNA for this transcript has not yet been isolated, and therefore the encoded polypeptide is not known, it is intriguing that *MrsA* might encode a product related to ANP27. The region of these *Mu* elements with sequence similarity to *anp27* neatly encompasses two oppositely oriented reading frames (ORF3 and ORF4 as per Taylor and Walbot). In fact, ORF3, when translated, is predicted to encode a polypeptide that would be ~60% (for *MrsA*) and 50% (for *Mu1.7*) conserved with the ANP27 polypeptide.

We are currently further characterizing the *anp27* gene in order to better understand its function during the anaerobic response as well as to clarify its relationship to the *Mu1.7* and *MrsA* sequences in the maize genome.

A plant genetically mosaic for chromosome 7 monosomy was derived from an *r-x1* deletion line

--Philip W. Becraft and Michael Freeling

The *r-x1* deletion causes mitotic nondisjunction during megagametophyte development. When nondisjunction occurs the egg cell receives either zero or two copies of a given chromosome. Approximately 10% of the progeny from a cross using a *r-x1* individual as a female result in monosomy for any one of the ten maize chromosomes. This phenomenon has been exploited for the purposes of mapping genes to chromosome (Weber, Maize for Biological Research, W. F. Sheridan, ed., 1982).

In an effort to map the Rough sheath-1 (*Rs1*) gene to a chromosome, a number of chromosome markers were crossed to a line containing *r-x1* and *Rs1-O*. Subsequently, Pioneer Hi-Bred, Inc. kindly mapped *Rs1* to chromosome 7S using a *Rs1* genomic clone and recombinant inbreds. To verify this and out of interest to see the phenotype of a chromosome 7 monosomic, we grew the progeny of the following cross: *Rs1-O/+*; *r-x1/R-g* x *ij/ij* (iojap; located on chromosome 7L).

Of approximately 250 F1 seedlings screened, 3 displayed white stripes typical of the iojap phenotype, indicating monosomy for chromosome 7. However on one of these the striping was limited to one half the seedling, divided at the midrib. As this plant grew, the solid green half of the plant developed a *Rs1-O* phenotype whereas the striped half did not (Fig. 1). This suggested that this plant may be genetically mosaic for chromosome 7 monosomy. To address this possibility DNA was extracted from both striped and solid green, rough sheath leaf material. The DNA was digested with *Eco*RI, electrophoresed, blotted and probed with a *Rs1* clone. Figure 2 shows that the solid green tissue was heterozygous for an RFLP at this locus, while the striped tissue only contained one allele. Therefore, the green tissue was heterozygous *rs1+/Rs1-O*; *ij/Ij+* and the striped tissue was hemizygous *rs1+/-*; *ij/-*. Furthermore, the upper band from the green tissue appears more intense than the lower band suggesting there may be two copies of that chromosome, and that the green tissue was trisomic.

These observations indicate that a mitotic nondisjunction occurred during early zygotic divisions. We are unaware of any previous reports of the *r-x1* deletion affecting sporophytic mitosis, and with only one such event it is not absolutely certain that *r-x1* caused this nondisjunction. However, since *r-x1* is known to cause mitotic nondisjunction in the megagametophyte, it seems likely that

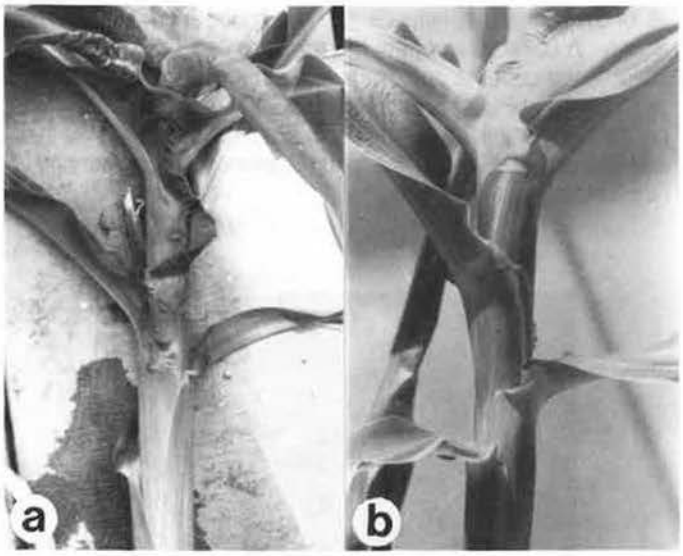

Figure 1. Two sides of the mosaic monosomic 7 plant derived from a *r-x1* line. a. The putatively trisomic half of the plant showing a solid green, rough sheath phenotype. b. The monosomic 7 half of the same plant showing white stripes and the lack of a rough sheath phenotype.

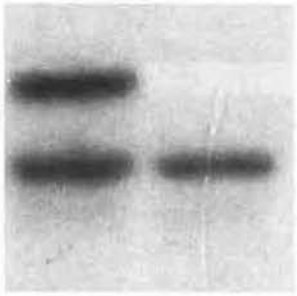

Figure 2. Autoradiograph of a southern blot of DNA extracted from the solid green, rough sheath tissue (lane A), and the striped tissue (lane B), probed with a *Rs1* clone.

it could also affect other mitoses. The rarity of this event may be because *r-x1* is normally heterozygous in the sporophyte. In the haploid megagametophyte there is no compensating wildtype allele, allowing the effect on mitosis to be more prevalent. Perhaps using B-A translocations to create hemizygous *r-x1* plants would reveal this nondisjunction phenomenon in the sporophyte as well.

RFLP linkage analysis of rough sheath-1 on chromosome 7S

--Shawndra D. Martinez, Philip W. Becraft and Michael Freeling

Rough sheath-1 (*Rs1*) was recently mapped courtesy of Pioneer Hi-Bred, Inc. to the short arm of chromosome 7 using recombinant inbreds with a *Rs1* genomic clone. To further define the location of *Rs1* on chromosome 7S, we undertook experiments to map *Rs1* using RFLPs.

The experiments were performed with a population segregating 1:1 for *Rs1-O*. Fifty-six progeny of a cross between an A643 inbred female and a *Rs1-O*/+ heterozygote in a B73 inbred background (backcrossed 4X) were scored phenotypically for *Rs1-O*. DNA was extracted from each individual and cut with either *Eco*RI, *Bam*HI, or *Hin*dIII enzyme. The gels were blotted and probed with *PIO200581B*, *NPI400* (both courtesy of Pioneer Hi-Bred, Inc.) and opaque-2 (*o2*) (courtesy of Bob Schmidt). The established order of these loci is *PIO200581B*, *NPI400*, *o2* proceeding from the telomere toward the centromere on chromosome 7S. The blots were also probed with a *Rs1* probe which verified that none of the plants were misgenotyped based on phenotype. The results of this analysis follow:

Chromosome Segment	Number of Individuals	Number of Recombinants	Map Distance (cM ± s.e.)
1. *o2 - NPI400*	55	7	12.7 ± 4.5
2. *NPI400 - PIO200581B*	52	10	19.2 ± 5.5
3. *PIO200581B - Rs1*	53	3	5.7 ± 3.2

Only two double recombinants were obtained; one involving chromosome segments 1 and 2, the other segments 2 and 3.

These data show that *Rs1* maps to the distal region of the short arm of chromosome 7, distal to *PIO200581B*. Therefore, *Rs1* defines a new zero point on the chromosome 7 RFLP map. Our data put *Rs1* approximately 37.6 map units distal to *o2*. Because the position of *o2* on the genetic map is 16, it appears likely that *Rs1* may also define a new zero position on the chromosome 7 genetic map. This position is now defined by hairy sheath (*Hs*). In previous attempts to map *Rs1-O*, *Hs* and *o2* in a three-point cross, we were unable to score the *Hs* phenotype. Until this is accomplished we cannot determine the relative positions of *Rs1* and *Hs* with certainty.

This part of chromosome 7S appears to display substantial variability in recombination frequency. In an earlier RFLP mapping experiment, 4 out of 24 individuals were recombinant between *Rs1* and *o2*, for a recombination frequency of 16.7 +/- 6.7%. The reported positions of these RFLP loci in the compiled data tables (MNL 65:145-153, 1991) would also indicate a lower recombination frequency between *PIO200581B*, *NPI400* and *o2* than was obtained in the current experiment.

Pachytene DAPI map

--R. Kelly Dawe, David A. Agard*, John W. Sedat* and W. Zacheus Cande
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Fluorescence microscopy has distinct advantages over traditional light microscopy in cytogenetic studies. Fluorescent DNA dyes (DAPI or Hoechst 33258) are simple to use, chromosomes can be viewed under conditions that preserve chromatin structure in a more life-like state, and data can be collected and analyzed in three dimensions (see Agard et al., Meth. Cell Biol. 30:353-377, 1989). We have prepared a linearized pachytene map as a first step in an effort to describe the spatial organization of chromatin during meiosis.

Nuclei from the inbred KYS were fixed with 4% paraformaldehyde in Buffer A (a buffer optimized to preserve chromatin structure, Belmot et al., J. Cell Biol. 105:77-92, 1987). A three dimensional data set was obtained by collecting serial fluorescent images at 0.25 micron intervals with a cooled charged coupled device (Fig. 1 shows a single image). The out-of-focus information was then removed using mathematical deconvolution. Figure 2 shows the effect of deconvolution on the single unprocessed image shown in Figure 1. Using the entire group of sections, the three dimensional path of each chromosome was traced and recorded. A radius of 0.8 microns was subsequently defined

around the modeled chromosome paths, and computationally straightened. The linearized chromosomes were then displayed on a flat surface (Fig. 3).

Figure 4 shows the map of cytological features previously derived by McClintock, Longley and Kato from acetocarmine staining. The chromosomes were scanned directly from The Mutants of Maize (Crop Science Society of America, p. 4, 1968) and all of the relevant comments and the knobs found in KYS are shown. A comparison of the two maps reveals that many of the major features of the acetocarmine map are evident in chromosomes fixed in Buffer A and stained with DAPI. Good examples are the chromomere on 4S and heavy chromomeres on 4L, the thick tapering end of 10S and the general prevalence of centromeric heterochromatin.

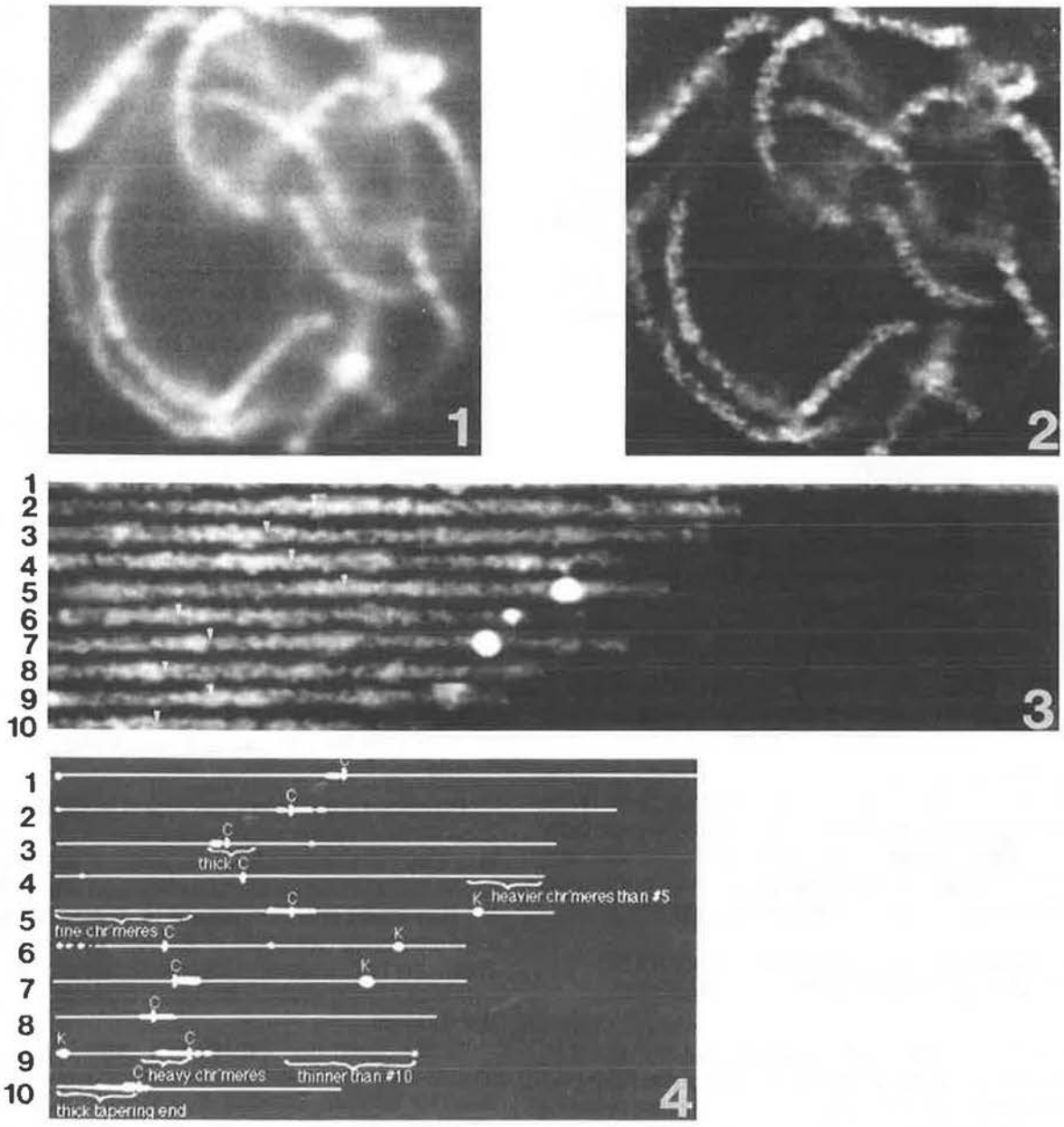


Figure 1. Fluorescent image of DAPI-stained chromosomes from the inbred KYS. This image was recorded using a 60X objective.

Figure 2. Image in Figure 1 after three dimensional deconvolution of a series of optical sections taken at 0.25 micron intervals.

Figure 3. Chromosomes straightened from the same nucleus shown in Figures 1 and 2. Chromosomes are arranged with the short arms to the left. Centromeres are noted with arrowheads. The resolution in the Z dimension of the data set is low, and this is evident in some stretches of the chromosomes (i.e. distal short arms of chromosomes 1 and 5). The bright spot on the long arm of chromosome 9 is an artifact of one chromosome passing very close to another chromosome.

Figure 4. Acetocarmine map of cytological features. The chromosomes and relevant comments were scanned directly from Mutants of Maize. As in Figure 3, chromosomes are arranged with the short arms to the left. All of the knobs except those known to occur in KYS were removed.

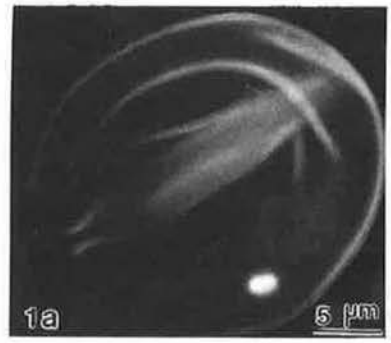
Other features of the acetocarmine map are less obvious on the DAPI map. In addition, there are differences in chromosome length, and centromeres are less pronounced using DAPI (compare to the centromeres on p3 of Mutants of Maize). Some of these discrepancies may be explained by the difference in fixation procedures. By comparing the straightened chromosomes from a number of different inbreds, we hope to develop a high resolution map of the cytological features in DAPI-stained chromosomes.

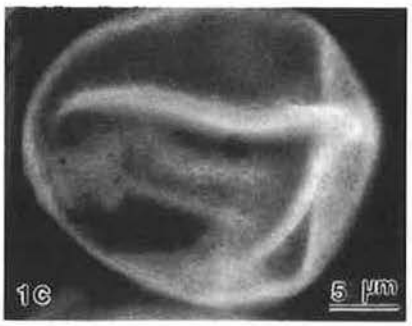
The features of cytological defects during meiotic and mitotic progression of microsporogenesis in male sterile mutants *Ms41* and *Ms44*

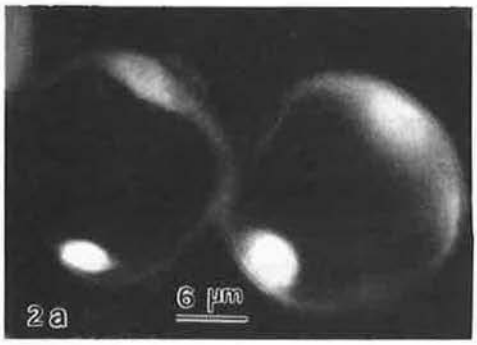
--Qinqin Liu and W. Zacheus Cande

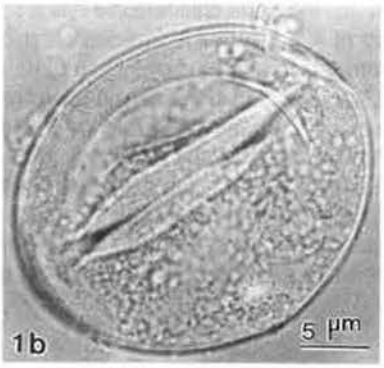
The new dominant male steriles, *Ms41* and *Ms44*, have been previously described as linked alleles on chromosome 4L (Albertsen and Neuffer, MNL 64:52). However, the cytological features of meiotic and mitotic progression during microsporogenesis in these mutants have not yet been studied. The presence of cytological defects in these mutants at different stages of microsporogenesis are identified using DAPI to stain chromatin. Normal meiotic divisions are observed in both mutants, but cytological defects occur before the first mitotic division in pollen development. These defects are correlated with nuclear and cytoplasmic disintegration.

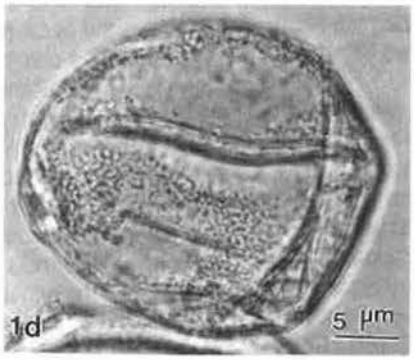
In *Ms41* (Fig. 1), the nuclei become reduced and degenerate during microspore vacuolation although normal tapetal cells and normal wall and pore development are observed during pollen maturation. The cytoplasm also breaks down at the same stage of development. In wildtype cells, vegetative and generative nuclei are observed after the first pollen mitosis (Fig. 2). Similar cytologi-

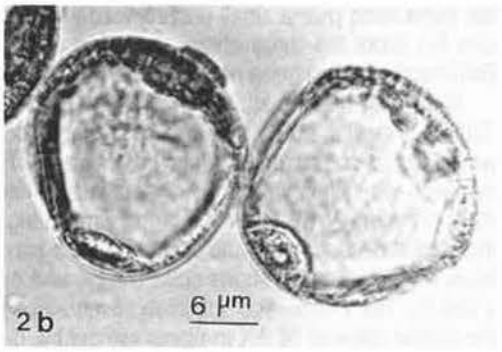












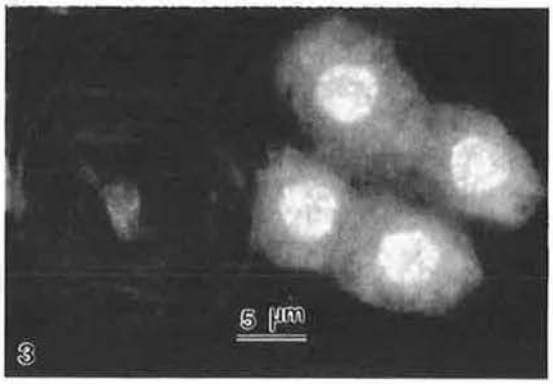


Figure 1a-d. Cytological defects before the first mitotic division in *Ms41* pollen cells. 1a: A reduced nucleus, stained by DAPI, in the pollen cell. 1b: Cytoplasm degenerated in the same cell as Figure 1a. 1c: Degenerate nucleus with diffuse chromatin staining in the pollen cell. 1d: Disintegrated cytoplasm in the same cell as Figure 1c.

Figure 2a and b. Normal cytological features after the progression of the first pollen mitosis in wildtype cells of *Ms41*. 2a: Vegetative and generative nuclei stained with DAPI. 2b: Cytoplasm with normally developed vacuoles in the center of the pollen cells.

Figure 3. Cytological defects before the first mitotic division in *Ms44* pollen cells. Nuclei become reduced and degenerate, but normal interphase nuclei in tetrads are present after meiosis.

cal defects in *Ms44* also occur before the first pollen mitosis (Fig. 3). These results suggest that mitotic progression in both mutants is blocked before the first pollen mitosis.

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Two ring chromosome derivatives containing a duplication of the *R* locus

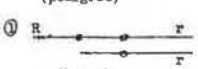
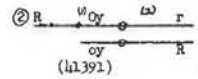
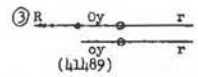
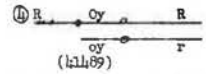
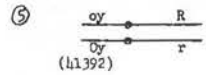
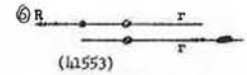
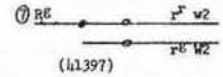
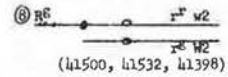
--Ellen Dempsey and M. M. Rhoades

The ring-10 chromosome of Emmerling was produced following irradiation of a K10-I chromosome when breaks occurred near the ends of 10S and 10L (Emmerling, Genetics 40, 1955). A series of ring-10 derivatives was studied by Miles (Indiana University Ph.D. Thesis, 1970). Among these was the K^m chromosome in which a long duplication, including the *R* and *G* loci as well as the three prominent chromomeres and a portion of the large knob of K10-I, was attached to the short arm of chromosome 10. In this configuration, one attribute of K10-I was lost, namely the induction of preferential segregation, although the crossover enhancement effect of K10-I was still operating. Derivatives of K^m in which the knob and three chromomeres were restored to their original orientation in 10L recovered some or most of the preferential segregation activity.

Two new derivatives arose in our stocks of ring-10; both resemble K^m in having a duplication of the *R* locus in 10S. One has the truncated knob of K10-I and the three chromomeres and was designated K^m-medium since it is shorter than K^m. The other has the same knob plus a small euchromatic tip terminating the short arm but lacks the three chromomeres; it was called K^m-short. Both chromosomes have a normal knobless 10L.

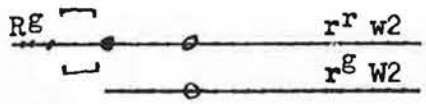
Data from genetic studies with K^m-medium are presented in Table 1. Transmission of the duplicated chromosome marked by *R* was followed in testcrosses (item 1 of Table 1). Female transmission was very slightly reduced and male transmission was about 40%. The longer K^m chromosome of Emmerling and Miles gave 40% female transmission and only 6-7% male transmission. The *R* locus in the duplication shows close linkage with *oy* in 10S (crosses 2 and 3). The 7-10% recombination observed was much less than the control value of 30.8% in plants without the duplication. Compounds having *R Oy r/oy r*, *R Oy R/oy r*, and *R Oy r/oy R* constitution were consistent in giving recombination of approximately 10% for the duplicate *R-oy* segment and about 30% for the *oy-r* region (crosses 2, 3, and 4). When K^m-medium was opposed by a K10-I carrying the *r* allele, female transmission of *R* on the duplication chromosome was considerably increased (cross 6). This result was unexpected since female testcrosses of K10-I *r*/N10 *R* heterozygotes give 70% *r* due to preferential segregation. However, the increased transmission of the duplicated chromosome in K^m-medium *R r*/K10-I *r* compounds becomes understandable when the output of single crossovers in 10S and 10L is considered. Singles in 10S give heteromorphic dyads at Anaphase I with the chromatids containing the duplicated *R* oriented toward the poles. Preferential recovery of the duplicated chromatid in the basal megaspore is expected. Single crossovers in 10L between the centromere and K10 knob would normally result in preferential segregation for the K10-I chromatid marked by *r*, but half of such chromatids also possess the duplicated *R* by virtue of the crossover so the ratio of *R:r* following singles in 10L remains 1:1.

Table 1. Crosses of K^m-medium heterozygotes.

	Heterozygote (pedigree)	Cross	Result						
①	 (41397)	♀ x r	Total 2655	% R 48.8					
		♂ x r	2469	40.7					
②	 (41391)	♀ x oy r	R Oy 525	R oy 335	r Oy 39	r oy 165	Total 1064	Recombination (1) 7.3%	(2) 31.0%
③	 (41489)	♀ x oy r	700	72	88	768	1628	9.8%	---
④	 (41489)	♀ x oy r	198	104	9	114	425	(1) and (2) 42.1%	
⑤	 (41392)	♀ x oy r	103	193	239	89	624	(1) ---	(2) 30.8%
⑥	 (41553)	♀ x r	Total 4561	% R 55.4					
⑦	 (41397)	♂ x r W2 / r -	R W 114	R w 30	r W 155	r w 55	Total 354		
⑧	 (41500, 41532, 41398)	♀ x r W2 / r W2	547	181	681	132*	1541		

* w2 classified by kernel phenotype; some r w2 escape detection so this class is underestimated.

Surprising results were observed in cross 7 of Table 1. Previous studies (Rhoades and Dempsey, Plant Genetics, UCLA Symposium, 1985) have shown that the K10-I chromosome has a segment from the distal region of 10L inserted in inverted order between the three-chromomere region and the large knob of K10-I. Three loci, *L13, O7* and *W2*, are included in this segment. In cross 7, an *r* tester hemizygous for *W2* was pollinated by a plant heterozygous for the K^m-medium chromosome with the duplicated *R*. The progeny included kernels displaying the typical *w2* phenotype (colored kernels with multiple depressed colorless sectors and colorless kernels with scarred floury-like sectors). When planted, these kernels gave white seedlings. The male parent apparently had the genotype



The *w2* plants derived from *R* kernels were unexpected since *R* should be almost completely linked with *W2* in the bracketed region. We conclude that either (1) the *W2* locus is not present in the duplication or (2) the *W2* locus is inactive in the new location. The *R* allele in the duplication retains its full activity in the seed component. The original ring and the K^m of Miles and Emmerling had the *R-r* allele (with a dominant *P* component). Our crosses suggest that the duplication in K^m-medium contains *R-g*; possibly activity of the *P* component is also suppressed in the new location. We hope to discriminate between alternatives (1) and (2) by transferring the duplicated segment back to 10L and noting whether the *W2* and *P* component of *R* regain their activity. To achieve this transfer, a rare crossover must occur which involves

pairing of the duplication with a modified 10L containing the three-chromomere region of K10-I but lacking *W2* (Df(F)). The unusual results of cross 7 have been reinforced by the finding of scarred variegated *R* kernels (called *R*-spotted) in the cross of K^m-medium heterozygotes by *r W2/r w2* pollen parents (cross 8). The proportion of scarred kernels (both *R* and *r*) on an ear is close to 25%, with roughly equal numbers in the colored and colorless classes. Again, there is no evidence of a *W2* allele in the duplicated segment.

Table 2 shows the data derived from crosses of K^m-short heterozygotes. Cross 1 illustrates the close linkage of *R* and *oy*,

Table 2. Crosses of K^m-short heterozygotes.

Heterozygote (pedigree)	Cross	Result					
		R Oy	R oy	r Oy	r oy	Total	Recomb.(1)
① R Oy / oy, r (41199)	♀ x r oy	524	47	55	532	1158	8.8%
② R, R / r (41528)	♀ x r	Total 777	% r 26.3				
③ R, R / r (41528)	♂ x r	668	25.3				
④ R, R / r (41403)	♂ x r	732	25.5				
⑤ R, r / R (41389)	♀ x r	1217	15.5				

similar to that found with K^m-medium. The percentage of *R* indicates normal female transmission of the duplicated chromosome. The duplicated region is considerably shorter than in K^m-medium and the *R* locus has been transposed to a position close to the knob. Crosses 2 and 3 show the results of male and female testcrosses when the duplicated chromosome carries two *R* alleles. The *r* kernels come only from noncrossovers and from crossovers distal to *R* in the long arm. A similar percentage of *r* kernels was obtained in cross 4 where the duplicated *R R* chromosome was opposed by K10-I carrying *r*. No preferential segregation is expected in the male testcross. In cross 5 there is a hidden *r* allele which is revealed in the progeny following a crossover anywhere between the *R* and *r* alleles on the duplicated chromosome. Thus, the *r* kernels represent one half of such crossovers and the chromosome bearing two *R* alleles (the reciprocal crossover) is not detected. Results in Table 1 with K^m-medium indicated a map distance of about 40 between the two *R* loci. If similar recombination frequencies occur in K^m-short heterozygotes, one would expect 20% *r* (crossovers) in cross 5 and 30% *r* (noncrossovers and distal crossovers) in crosses 2, 3, and 4. The lower frequencies observed for both crossover and noncrossover classes are difficult to explain.

Finally, both chromosomes were tested for their ability to induce preferential segregation in chromosome 9. The data are not extensive (see Table 3) but no preferential segregation of the *yg2* allele in chromosome 9 was observed. No change was noted due to the presence or absence of a Df(K) chromosome which carries the differential segment (three-chromomere region) but not the knob of K10-I. A frequency of 65% *yg* has been observed in similar heterozygotes carrying an intact K10-I chromosome. The lack of preferential segregation observed with the two ring chromosome derivatives is in agreement with Miles's observation that

Table 3. Tests for preferential segregation.

Heterozygote (pedigree)		Cross	Total	% yg
① $\frac{K^m\text{-med}}{Df(K)}$ $\frac{K^I 9\ yg}{K^o 9\ Yg}$	(41236)	♀ x yg	778	49.0
② $\frac{K^m\text{-med}}{N\ 10}$	" (41236)	♀ x yg	427	49.9
③ $\frac{K^m\text{-short}}{Df(K)}$	" (41247)	♀ x yg	383	49.9
④ $\frac{K^m\text{-short}}{N\ 10}$	" (41247)	♀ x yg	697	52.2

the original K^m does not induce preferential segregation. Thus, transfer of the knob to the short arm of chromosome 10 eliminates its capacity to induce preferential segregation in other chromosome pairs and may also affect the activity of genes in the duplicated segment.

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Genome mapping with non-inbred crosses using GMendel 2.0

--Craig Echt, Steven Knapp and Ben-Hui Liu

A powerful linkage analysis computer program, GMendel 2.0, has been developed which allows genome maps to be constructed from any type of diploid cross. GMendel 2.0 is unique in that it can perform multipoint linkage analysis on populations with complex genetic structures, such as those arising from an F1, F2 or backcross between highly heterozygous parents, as well as from more traditional mapping crosses, such as from an F2 from inbred parents. The general applicability of the program also allows for mapping of dominant genetic markers, such as those associated with AFLPs (amplified fragment length polymorphisms, or RAPDs), null-allele RFLPs and dominant morphological phenotypes. By way of example we describe below the construction of a linkage map of diploid alfalfa, but genome maps could be constructed from any population of similar genetic complexity.

In collaboration with Tom McCoy at Montana State U. and Tom Osborn and Kim Kidwell at the U. of Wisconsin we have constructed the first genetic linkage map of *Medicago sativa* using GMendel 2.0 (manuscript in preparation). The current map incorporates 57 RFLP and 128 RAPD markers into 8 linkage groups, corresponding to the 8 chromosomes of alfalfa, and has a total length of 1,321 recombination units. The 88 progeny we used for linkage analysis were from a backcross between diploid clones derived from cultivated alfalfa, an outcrossing tetraploid. The use of a backcross from highly heterozygous parents was necessary because cultivated alfalfa is very susceptible to inbreeding depression and inbred lines are not generally available.

A number of different segregation types can arise, depending on the type of cross, when parents heterozygous at many loci are used to generate a population for linkage analysis. Figure 1 lists all the possible parental phenotypes and their segregation genotypes generated from F1s, F2s and backcrosses. Loci in an F2 population, whether from inbred (homozygous) or heterozygous parents, have a maximum of two alleles which can segregate either as 1:2:1 or 1:3 (for dominant markers). For a backcross population from inbred parents, loci can have two alleles which segregate either as 1:1 or 1:3, while if from heterozygous parents, loci can have up to three alleles which can segregate either as 1:1, 1:3, 1:2:1, 1:1:1:1 or

GMendel 2.0 Segregation and Progeny Codes for Dominant and Codominant Markers from Crosses with Heterozygous Parents

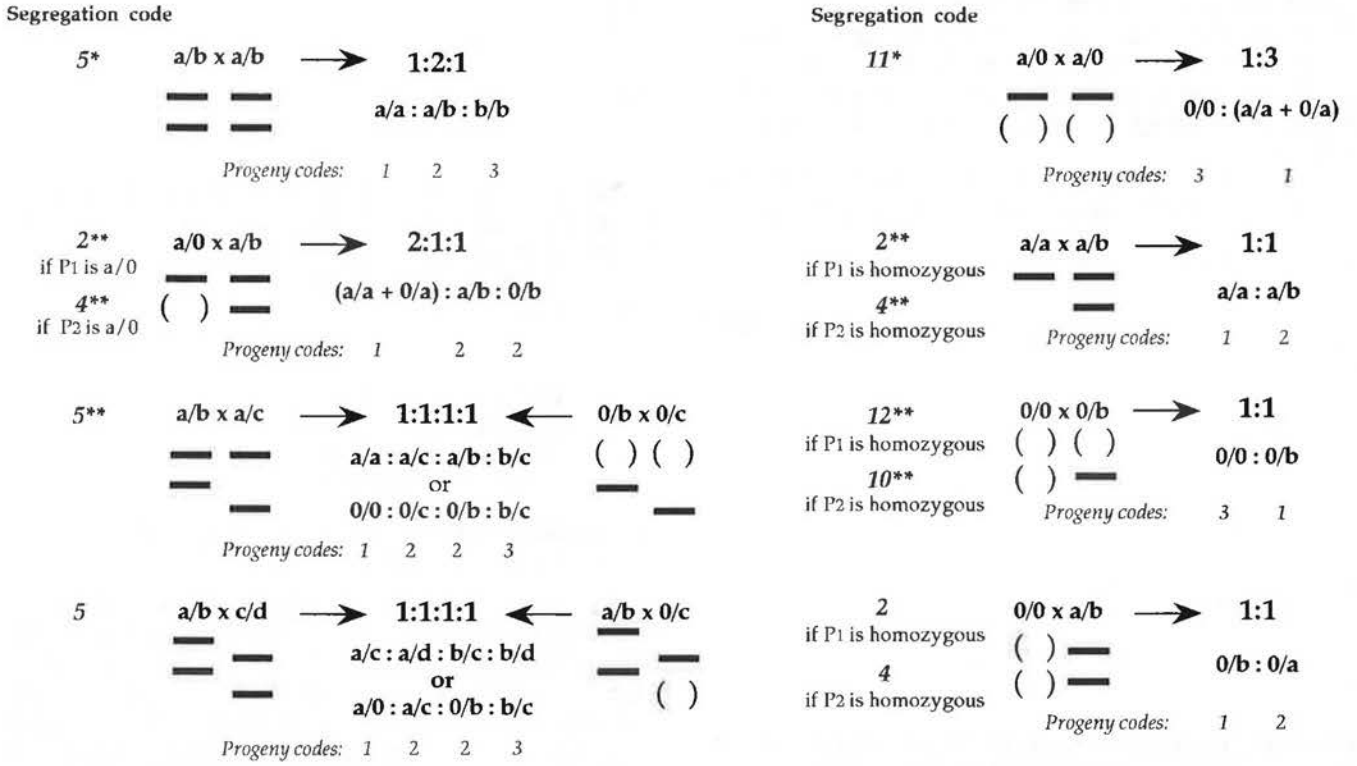

Figure 1. All possible parental DNA marker phenotypes and general segregation genotypes for single loci arising from crosses using heterozygous parents. All cases are possible from an F1 cross. The only segregation types possible from an F2 cross (self) are marked "*". Those segregation types possible from a backcross are marked both "*" and "**". In the case of a backcross P1 refers to the F1 and P2 to the recurrent backcross parent. Alleles of a locus are designated a, b, c, d or 0 (null). The segregation and progeny codes are used by the current version GMendel 2.0 for scoring parental and progeny phenotypes in the mapping database matrix. Future versions of the program will have separate codings for 1:1:1:1 and 2:1:1 segregants to allow for even greater precision of recombination and ordering estimates.

2:1:1. In an F1 population from heterozygous parents there can be up to 4 alleles per locus but the segregation classes are the same as those found from a backcross. There is, of course, no segregation expected in an F1 population from inbred parents.

Any program of general applicability which seeks to create a comprehensive linkage map from heterozygous crosses must be able to generate two-point recombination estimates from all possible matings between multiple segregation types, properly infer linkage phases and correctly order each locus with respect to all others. The main problems in accomplishing this arise from the presence of multiple alleles and multiple segregation classes, and from the inability to know linkage phases a priori due to the lack of information about the genetic structure of the parents.

Programs such as Mapmaker (Lander et al., Genomics 1:174, 1987) are designed to analyze segregation data from inbred parents only. As pointed out by Ritter et al. (Genetics 135:645, 1990), Mapmaker cannot integrate data from multiple segregation classes into one map. Ritter et al. have described a method which generates two point estimates and linkage subgroups from populations with multiple segregation types but ordering is accomplished by an ad hoc method that is not true multipoint mapping. This method was used in developing an RFLP map of diploid potato from a backcross population using heterozygous parents (Gebhardt et al., TAG 78:65, 1989).

By the use of the appropriate segregation class codes and progeny phenotype codes (Fig. 1) GMendel 2.0 generates two point maximum likelihood estimates for all pairwise matings between all loci. Linkage phases are correctly assigned based on probability rules and gene order is estimated using an advance multipoint mapping algorithm. Missing progeny data are neither estimated nor substituted and are simply excluded from the two-point estimates. As can be noted from Figure 1, the current version of GMendel ignores multiple alleles and classifies loci as segregating either 1:1, 1:2:1, or 1:3. Loci segregating 1:1:1:1 are scored as 1:2:1 segregants and loci segregating 2:1:1 are scored as 1:1 segregants (see Figure 1). Although limiting scoring to only two alleles does not take full advantage of all the genetic information present in multiallelic segregants, it does retain all the information available from two alleles and does not compromise the accuracy of the map. This approach was made necessary by program development constraints and will be altered in future versions of the program to allow for complete genetic classification of the progeny.

No a priori knowledge of linkage phase is needed since GMendel 2.0 uses simple probability rules to infer whether two loci are linked in coupling or repulsion. When the LOD score for two loci is greater than 3.0 and when a maximum likelihood estimate for coupling is used, then a recombination estimate of less than 0.30 indicates the loci are in coupling, while an estimate greater than 0.70 indicates that the loci are in repulsion. (LOD means the log of

the odds, the "odds" being the ratio of the probability that two loci are linked with a given recombination value over the probability that the two are not linked. A LOD over 3.0 means that the chances are greater that 1000:1 that the loci are linked for a given recombination estimate.) A correct repulsion recombination estimate is obtained by subtracting from 1 any coupling estimate over 0.70 (with a LOD > 3.0). This method of determining linkage phases is 100% accurate when the probability rules are met and does not require knowledge of the genotypes of the parents. Loci having recombination estimates above 0.30 are assigned to unlinked loci or to two-loci linkage groups.

Multipoint gene order is determined by GMendel 2.0 using a powerful method called the simulated annealing algorithm (SAA). The details of SAA will be presented elsewhere but, in brief, it estimates the shortest linear map, the global minimum, by simulating different gene orders for groups of loci in a progressive manner and saving only the shortest orders. A number of different ordering criteria are used in estimating minimum distances. The two main ordering criteria are the sum of adjacent recombination frequencies matrix method and the sum of adjacent 2-point LOD scores matrix method. The SAA can obtain gene orders even when non-informative matings exist between some of the loci. An example of a non-informative mating is when two loci, A and B, are each segregating 1:1 among the progeny but locus A is homozygous in one parent and locus B is homozygous in the other parent. Recombinant progeny cannot be detected from such a mating. Such matings are devoid of ordering information but gene order can be inferred from the relative order and distances of adjacent loci which do give informative matings.

The current version of GMendel 2.0 does not incorporate maximum likelihood equations for estimating recombination for loci exhibiting differential viability. However, skewed segregation ratios do not seem to have a significant effect on the recombination estimates or gene order as long as the ratios were not profoundly skewed. This was evident from our alfalfa map where 45% of the loci for which we obtained segregation data did not fall within Mendelian expectations at the $p < 0.05$ level. The high level of segregation distortion present in our mapping population results in large part from a high genetic load and can be expected within most open-pollinated or wide-cross populations. Future versions of the GMendel 2.0 will utilize maximum likelihood recombination estimates which will minimize the effects of distorted segregation ratios.

GMendel 2.0 runs under a UNIX operating system and requires a Fortran compiler. For information on obtaining a copy of the program write to Steven Knapp, Dept. of Crop and Soil Science, Oregon State Univ., Corvallis, OR 97331-3002 or send e-mail to sknapp@helix.oscs.orst.edu.

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RI Plant Manager: a microcomputer program for genetic mapping with recombinant inbred strains

--Kenneth F. Manly

RI Plant Manager is a microcomputer program for storage and analysis of genetic mapping data from recombinant inbred strains or from multilocus backcrosses. The program rapidly identifies statistically significant linkage between known loci and a newly described locus, and it facilitates rapid evaluation of alternative map orders for linked loci. The program requires a Macintosh Plus or later Macintosh model running version 5.0 or newer of the system software (including 7.0). It will run under Finder or Multifinder. Memory required depends on the size of the data files being analyzed; 2 Mb is recommended.

RI Plant Manager provides four windows for display of stored data. One is the traditional tabular display of genotypes with each row representing one locus and each column representing one strain. The program marks implied crossovers automatically. The order of loci can be changed by dragging a locus with the mouse, and when a locus is moved, the crossover marks are updated automatically. The second type of window is a graphic map with estimated map distances derived from the data. The third window shows a table of statistics for each interlocus interval, including confidence limits for the interval distance; and the fourth window displays a bibliographic reference and comments for each locus. The contents of all windows can be printed. RI Plant Manager accepts manual entry of information, but it will also import and export information from delimited text files.

RI Plant Manager is derived from RI Manager, a program written for analysis of recombinant inbred strains of mice and described in Mammalian Genome (1:123-126, 1991). RI Plant Manager, however, is written for recombinant inbred strains of plants, that is, it uses functions appropriate to self-pollinated strains to calculate map distances and confidence intervals.

The current version of RI Plant Manager, which can be licensed for $90, includes the program and a 46-page illustrated user's manual. The licensing fee, payable to Health Research, Inc., a nonprofit corporation, will be used for maintenance and further development of the program. Further information is available from Kenneth Manly, Roswell Park Cancer Institute, Buffalo, NY 14263-0001 (phone: (716) 845-3372; fax: (716) 845-8169; Internet: cammanly@ubvms.cc.buffalo.edu; bitnet: cammanly@ubvms).

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Allometric genetics X. A three point test involving a gametophyte gene (*Ga10 A2 Bt*)

--Luiz Eugenio Coelho de Miranda and Luiz Torres de Miranda

In MNL 64:34-35, using data reported by J. Gonella and P. A. Peterson with two kinds of families of backcrosses in the presence and absence of *Ga10*, we have developed a solution for the problem. In the present report a solution is presented for the case of having only one kind of family, that is, two marker factors segregating in the presence of the gametophyte factor. The original data from the authors cited are in the presence of the gametophyte factor: *A2 Bt* = 378, *A2 bt* = 7, *a2 Bt* = 117, and *a2 bt* = 253.

We have shown by allometric genetics that the calculus of the recombination of the markers is straightforward.

So the distance q between *A2* and *Bt* is directly obtained as (*A2 bt* + *a2 Bt*)/n.

With the values presented we have the sum total of *A2* = 385, *a2* = 2,270, *Bt* = 495 and *bt* = 2,160. With these values the expectations by the proposed solution are as presented in Table 1, together with the algebraic and numeric calculations.

Table 1. Data as presented by P. A. Peterson and J. Gonella in MNL 49:71-73 in their Table 1. The expectations of the segregation as presented by L. T. de Miranda and L. E. C. de Miranda in MNL 64:34-35 and subsequent algebraic steps. p is the recombination *Ga10 A2*, and q those of *A2 Bt*. α is the allometric coefficient of *Ga10*.

$$\frac{n}{2}\left[p(1+\alpha)+(1-p)(1-\alpha)\right]=385\ \underline{A2} \qquad \frac{n}{2}\left[(1-p)(1+\alpha)+p(1-\alpha)\right]=2270\underline{a2}$$

$$\frac{n}{2}\left[(p+q)(1+\alpha)+(1-p-q)(1-\alpha)\right]=495\underline{Bt} \qquad \frac{n}{2}\left[(1-p-q)(1+\alpha)+(p+q)(1-\alpha)\right]=2160\underline{bt}$$

The values obtained were p= 0.136, q=0.048 and α =0.862.
Substituting in the equations we have:

384.6 385 A2	2270.4 2270 a2
494.4 495 Bt	2160.6 2160 bt

So, the formulation is correct.
Efecting the algebric operations we have:

$$1-\alpha+2\alpha p=\frac{2}{n}\ \underline{A2} \qquad 1+\alpha-2\alpha p=\frac{2}{n}\ \underline{a2}$$

$$1-\alpha+2\alpha p+2\alpha q=\frac{2}{n}\ \underline{Bt} \qquad 1+\alpha-2\alpha p-2\alpha q=\frac{2}{n}\ \underline{bt}$$

Multiplying the first line by minus one and adding to the second we have:

$$2\alpha q=\frac{2}{n}\ (\underline{Bt}-\underline{A2}) \text{ or } -2\alpha q=\frac{2}{n}\ (2160-2270)$$

This eliminates 'p' and taking the value of α

$$\alpha=\frac{\underline{Bt}-\underline{A2}}{qn} \qquad -\alpha=\frac{\underline{a2}-\underline{bt}}{qn}$$

By allometric genetics $q=(\underline{A2}\ \underline{bt}+\underline{a2}\ \underline{Bt})\div n$
As we have the value of q with the actual numbers results

$$\alpha=\frac{495-385}{0.048\times2655}=0,863$$

The value is the same as reported in the previous work
Putting the values of α and q in the equations with p we obtain its value

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Name change for chloroplast mutator

--William C. Taylor, Mary Byrne and E. H. Coe, Jr.

Chloroplast mutator (*cm*) is a recessive mutation closely linked to *R1* that causes a distinct pattern of yellow stripes on leaves. These stripes appear to be clonal, suggesting that *cm* is active in the apical meristem and during leaf development (Thompson et al., Am. J. Bot. 70:940-950, 1983). As is the case for iojap, *cm* conditioned plastids are maternally inherited (Stroup, J. Hered. 61:131-141, 1970). Maternal inheritance suggests that the nuclear mutation causes a heritable block in chloroplast development. However, one would expect a wide array of phenotypes from a mutator, rather than the clonal pattern seen in homozygous *cm* plants. The fact that chloroplast development is blocked at the same stage in *cm* stripes (Thompson, 1983) is also not what one expects from a mutator. It is formally possible that *cm* is a mutator in a restricted sense, causing a specific mutation in the chloroplast genome at specific times in development.

In order to remove the confusion surrounding the name mutator, we propose to change the name of this mutation to chloroplast modifier. This general description is likely to be consistent with whatever the *cm* mutation turns out to be.

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Mutagenic effect of anther culture

--Y. C. Ting and K. Delorey

Individuals derived from selfing the Po (microscope-plant) plants of KH-13 via anther culture in vitro manifested not only reduced vigor but also seedling lethality. However, a few surviving plants grew into the adult stage. It was found that before tasseling the top of these plants bent downward and the flag leaves were wrapped around the young tassels (the first leaf below the tassel). As growth advanced, the tassels gradually turned upward and emerged shortly after. When the whole tassels were completely exposed the tops of the plants were still curled downward. As growth continued to advance, only a few of the florets reached anthesis stage, and their pollen shedding was generally poor. Since their pistillate florets developed normally self-pollinations of the plants were attempted. Some well-developed kernels were subsequently obtained even though the percentage of seed sets of these plants was very low compared with that of the ancestral plants.

When the progeny plants of the selfed Po plants grew into adults the curling tassels (bending downward) were again demonstrated for all of them. Hence, it was suspected that this character might represent a mutation transmitted from one generation to the next by sexual reproduction. In consequence of this, it was designated *cur*. This characteristic was not found among the progeny plants of its ancestor, and it was originated de novo during culturing.

The above curling tassel plants were subsequently crossed to a commercial inbred B73. The F1 plants from the reciprocal crosses did not show any curling tassel appearance in a population of 115 individuals. However, among the F2 plants from the selfed F1s, 11 out of a total of 49 manifested the curling tassel character. In other words, the curling tassel phenotype reappeared in a ratio of 3 (normal tassel) to 1 (curling tassel) as expected.

In another study in the last summer on the progeny plants derived from KH-13 via anther culture, it was found that they were very susceptible to lodging (by hurricane force). After the last hurricane, August, 1991, all of these progeny plants were blown down. On the other hand the F1 plants from a cross between the same progeny plants and a commercial inbred B73 were all resistant to lodging. In other words, none of them fell to the ground after the same storm. In order to know if there is any Mendelian segregation among the F1 progeny, the F1 plants were selfed. The results of this test will be available in the near future.

Negative effect of maltose on anther culture

--Y. C. Ting and K. Delorey

It was learned (personal communication) that when maltose was substituted for sucrose in barley and sorghum anther culture and regeneration of plants from calli, better results were obtained. Hence, the same procedures were followed for maize anther culture in the last two years. Up to the present, no better results were found either for anther growth or for callus regenerations.

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Isolation of new alleles of anther ear and indeterminate

--Joseph Colasanti and Venkatesan Sundaresan

The genes for anther ear (*an1*), indeterminate (*id*) and bronze-2 (*bz2*) are known to lie within 1 or 2 map units of each other on the long arm of chromosome 1. We used the mutable bronze allele *bz2-m2*, which contains a *Ds2* transposable element

insertion in the *Bz2* gene, in a series of crosses in the hopes of obtaining an insertion of the *Ds2* element into the anther ear gene.

The first set of crosses, in the summer of 1989, involved a directed cross of *bz2-m2/bz2-m2* plants carrying an active *Ac* element (a gift from Kelly Dawe, U. C. Berkeley) as the female parent with plants homozygous for the *an1 bz2* deletion (a gift from Virginia Walbot, U. Stanford) as the male parent. The *an1 bz2* plants were semi-dwarf in stature and required exogenous application of gibberellic acid (GA).

Approximately 500 ears were isolated from this cross; most of the kernels had *bz*-mutable aleurones, but there were approximately 1 to 4 completely purple kernels per ear which corresponded to germinal excision of the *Ds2* element from the *bz2* gene. These germinal revertant kernels were selected to facilitate the screening of mutants by reducing the number of plants to screen. From 1000 purple kernels planted in the summer of 1990, one semi-dwarf plant was found (#853) and subsequently selfed and outcrossed. At harvest, the ears of plant #853 had anthers typical of a GA-deficient phenotype. All other non-dwarf plants were also selfed with the intent of screening for other *Ds2*-induced recessive mutations (see below).

The progeny from the self of plant #853 were crossed to a line carrying an *an1* single gene mutation (from the Maize Coop) and also backcrossed to the *an1 bz2* deletion line. Plant #853 was found to be allelic to *an1* and the new allele tentatively designated *an1-**. In addition, in all plants examined so far, the *an1-** allele is closely linked to the *Bz2* gene, which presumably resulted from germinal excision of *Ds2* from the *bz2-m2* gene.

For molecular analysis, a 100bp fragment of the *Ds2* element was obtained from Sarah Hake (U.S.D.A., Albany) and used to screen the outcross progeny of plant #853 by Southern blotting. The use of this fragment reduces the number of bands that correspond to the *Ds2* element family and that correspond to other *Ds*-like elements. The number of *Ds2*-hybridizing bands in these progeny varied from 4 to 12, with the greatest number of bands in the selfed progeny. One 4.3kb band from a *Bam*HI digest was found to always associate with *an1-** progeny and was always absent in *an1 bz2* deletion strains. Experiments are in progress to further characterize this 4.3kb *Bam*HI fragment and to determine the effect of the presence of an active *Ac* element on the expression of the *an1-** phenotype.

In the summer of 1991 the selfed, normal progeny from the previous summer were screened for any interesting whole plant recessive mutations. In all of these families, purple and bronze kernels segregated 3:1, as expected. Bronze kernels corresponded to a homozygous *an1 bz2* deletion genotype and therefore only purple kernels were selected. Twenty purple kernels from each of 600 families were screened. Several distinct phenotypes were found (mostly dwarfs and mini-plants) but the most prominent was family #62. Fifteen of 20 kernels from family #62 grew to adult plants; of these, all but 7 plants had completely shed out by the first week of August, as did all the other plants in the other families of the screen. These seven plants continued to grow taller and exhibited other indeterminate phenotypes, such as prop root formation up to the ninth node and an increased number of internodes. Eventually, 2 of the 7 plants had formed tassels and shed pollen by the end of August; these were outcrossed to various plants in the field which were delayed in planting and were flowering at this time (courtesy of Chang-deok Han). The other 5 "indeterminate" plants did not form tassels until the middle to end of September. The normal siblings of family #62 had between 9 and 12 nodes at maturity, whereas the 7 late flowering plants had between 17 and 25 nodes at season's end and none of these 7 plants formed viable ears. Interestingly, *id/id* plants from the Maize Coop (*id-R*) that were planted at the same time in another part of the field did not flower until the end of October.

In preliminary experiments, purple kernels from family #62 that were germinated and grown under short day conditions (16 hour nights/8 hour days) for 4 or 6 weeks continued to segregate late-flowering plants; i.e. this light regime had no effect on abrogating the indeterminate phenotype, as has been reported for other *id* alleles. Experiments are in progress to determine whether this is a new *id* allele and if it is the result of a *Ds2* insertion.

_Ac_ transposition impaired by a small terminal deletion

--Thomas Peterson

A *P-vv* allele (*P-vv*-5145*) specifying extremely light variegated pericarp and cob glumes was obtained from the standard variegated *P-vv* allele. In crosses to the *Ds* tester stock *r-m3*, *P-vv*-5145* behaves as though it contains a single *Ac* element with a normal ability to induce *Ds* transposition. Southern blot analysis showed that the *Ac* element in *P-vv*-5145* is in the same position and orientation as in the progenitor *P-vv* allele.

We used PCR to amplify and sequence the termini of the *Ac* element in the *P-vv*-5145* allele. The *Ac* 5' end was identical to the progenitor *P-vv Ac*. However, the 3' sequence showed a deletion of two nucleotides: one nucleotide deleted from the 3' end of *Ac*, and one nucleotide deleted from the flanking *P* gene sequence:

	5' TIR		3' TIR	*P* gene (Intron 2)
Progenitor *P-vv*:	CAGGGATGAAA. . .	*Ac*. . .	TTTCATCCCTA	CCCGTTCGTTTTCGTT
P-vv-5145*:	CAGGGATGAAA. . .	*Ac*. . .	TTTCATCCCT	CCGTTCGTTTTCGTT
Ds (sh), Ds1 (Adh1)	TAGGGATGAAA. . .	*Ds*. . .	TTTCATCCCTA	

The deletion in *P-vv*-5145* has the effect of substituting a C for the terminal A in the 3' terminal inverted repeat of *Ac*. The terminal inverted repeat is critical for transposition, because a deletion of the last four nucleotides (CCTA) of the *Ac* 3' TIR abolishes transposition (Hehl and Baker, Mol. Gen. Genet. 217:53-59, 1989). However, *Ds* elements commonly have perfect 11 nucleotide terminal inverted repeats, while the *Ac* inverted repeat is only 10 nucleotides in length due to mismatch of the terminal nucleotides (see above). Our results demonstrate that, even though the 5' terminal nucleotide can differ between transposition-competent *Ac* and *Ds* elements, alteration of the 3' terminal nucleotide can have profound effects on *Ac* transposition.

The frequency of transposition of *Ac* in *P-vv*-5145* is not known, since germinal revertants haven't been found yet. The frequency of red revertant pericarp sectors is reduced by at least ten fold in *P-vv*-5145* relative to the progenitor *P-vv*. Because the *Ac* in *P-vv*-5145* still transactivates *Ds* elements normally, *P-vv*-5145* may be useful as an *Ac* source for experiments such as gene tagging with *Ds* elements and *Ds* breakage studies in which *Ac* transposition would lead to undesirable changes in *Ac* dosage.

I thank Michael Persans and Mary Ellen Okar for assistance.

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Molecular cloning of *ij* (iojap): Insertion of a *Ds*-like element is responsible for the *ij-ref* mutation

--Chang-deok Han, Robert A. Martienssen and Edward H. Coe

To understand the diverse and unique phenotypic expression of the iojap mutant, a transposon tagging experiment has been conducted using Robertson's Mutator (*Mu*). A new *ij* mutant was obtained from crosses of *ij-ref* (the original reference allele) to *Mu* lines. Subsequent genetic and molecular studies showed that the mutant was of a new *ij* allele (*ij-mum1*) from the *Mu* lines and contained a *Mu1* element that cosegregated with the iojap phenotype (MNL 63:65 and 64:46).

A 6.0kb *Eco*RI genomic DNA fragment containing the *Mu1* element in *ij-mum1* was cloned. Flanking DNA from the genomic clone detected a single copy fragment in the genomic DNA and a 1.3kb transcript in leaf mRNA. The patterns of transcripts hybridizing with flanking DNA from the genomic clone were correlated with the different *ij* genotypes. The level of transcripts from *ij-ref/ij-ref* was severely reduced and the size of the transcripts was slightly smaller than that from heterozygous and normal seedlings. The level of transcripts was about 50% reduced in heterozygous seedlings, compared with that in homozygous normal seedlings.

In order to verify that the cloned DNA was from the iojap locus, we took advantage of the fact that the original *ij-ref* allele described by Jenkins (1924) is unstable. Rarely, normal green sectors on pale green ('grainy') leaves have been observed in homozygous *ij-ref* plants even in an inbred background (Coe et al., 1988). In 1979 and 1983, two independent germinal revertants (*Ij-Rev*) were obtained from plants in which green sectors ran through the tassel (Coe, unpublished data). The *Ij-Rev* alleles were maintained by crossing to *ij-ref/ij-ref* plants and subsequent selfing. Normal plants (*Ij-Rev/ij-ref* or *Ij-Rev/Ij-Rev*) of selfed progeny of heterozygous *Ij-Rev/ij-ref* plants were examined by Southern hybridization, in comparison to *ij* plants carrying the *ij-ref* allele from the original plants with revertant sectors. Figure 1 is an autoradiogram of a Southern blot hybridized with a cloned

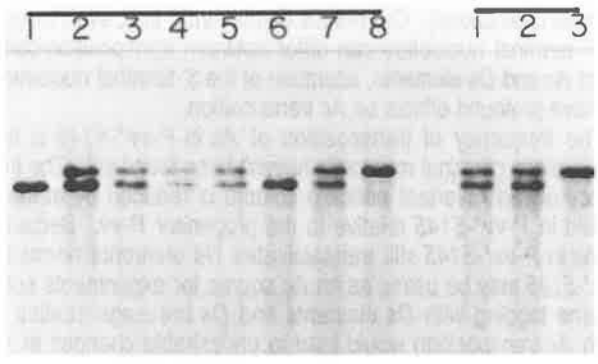

Figure 1. Southern analysis of the progeny of two independent germinal revertants (*Ij-Rev*). Genomic DNA was extracted from normal plants (*Ij-Rev/Ij-ref* or *Ij-Rev/Ij-Rev*) from the selfed progeny of *Ij-Rev/Ij-ref* plants of two families (A1-7 and B1-2) and from *ij* plants (lanes 8 and 3). The DNA samples were digested with *Bcl*I. The two independent revertants were designated A and B. The genotype of each normal plant was identified by progeny testing. Lanes A2, A3, A4, A5, A7, B1, and B2 were from heterozygous plants (*Ij-Rev/Ij-ref*). Lanes A1 and A6 were from homozygous plants (*Ij-Rev/Ij-Rev*). Flanking DNA fragment of the genomic clone from *ij-mum1* was used as a hybridization probe.

DNA fragment flanking the *Mu1* element. It shows that polymorphisms of *Bcl*I digested genomic DNA were correlated with the genotypes of the plants in two independent revertant families. Transcripts of the progeny of one of the germinal revertants were examined using the same probe (Fig. 2a). Northern blot data

Figure 2. Northern analysis of transcripts from *Ij-ref*, *Ij-Rev*, and normal (+/+) siblings. The blot of polyA+ RNA from *Ij-ref* (lane 1), *Ij-Rev* (lane 2), and normal siblings (lane 3-5) was hybridized with a flanking DNA fragment of the *ij-mum1* genomic clone (a). The following serial dilution of mRNA samples of normal siblings was loaded; 2 times higher than mRNA of *Ij-ref* and *Ij-Rev* (lane 3), equal amount (4), 2 times dilution (lane 5), 5 times dilution (6). The amount of mRNAs in each lane was reexamined by reprobing the blot with a maize actin genomic DNA (b).

showed that transcripts in the revertant seedlings accumulated up to the normal level and were of the same size as those from normal siblings. Uniform loading of polyA+ RNAs in each lane was confirmed by reprobing the blot with maize actin genomic DNA as a probe (Fig. 2b). Also, eight independent somatic revertant sectors on the leaves of homozygous *ij-ref* plants were examined by Southern hybridization, in comparison to adjacent *ij* tissues in the same leaves. Two DNA fragments were detected in genomic DNA from all the green somatic revertant sectors. One of them was of the *ij-ref* size and the other one showed the same size as seen in the germinal revertant alleles (data not shown). The adjacent *ij* tissues contained one DNA band of the *ij-ref* size.

To understand the molecular basis of the reversion event at the iojap locus, we cloned a 6kb *Eco*RI DNA fragment from the *ij-ref* allele. Figure 3 is a composite restriction map of the genomic clones of *ij-ref* and *ij-mum1*. Except for 1.5kb additional DNA located around 350bp to the left of the *Mu1* insertion site, the restriction map of *ij-ref* was identical to that of *ij-mum1* except for the *Mu1* element. Both ends of the 1.5kb DNA and the adjacent DNA were sequenced (Fig. 4a). The adjacent *ij-ref* DNA contains an 8bp direct duplication, relative to the *ij-mum1* allele. And, both ends of the 1.5kb DNA have perfect inverted repeat sequences of 16bp. Eleven base pairs from both ends of the invert repeat are almost identical to the 11bp inverted repeats of *Ac* and known *Ds* elements. Terminal and subterminal regions of both sides of the element shared around 60% sequence homology with those of *Ac* and also have two or three DNA sequences that have been shown to be an *Ac*-transposase binding site (Kunze and Starlinger, EMBO J. 11:3177-3185, 1990).

To obtain molecular proof that functional and phenotypic re-

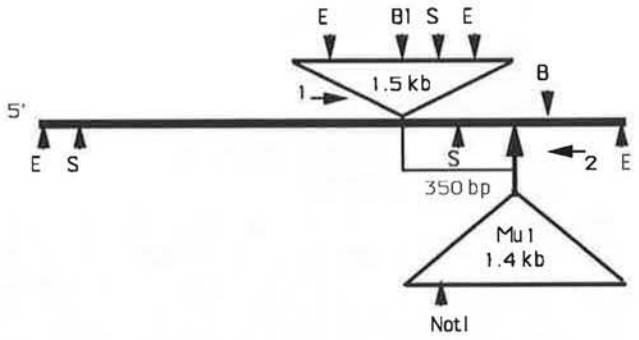

Figure 3. A composite restriction map of the genomic clones of *ij-ref* and *ij-mum1*. The restriction maps of the *ij-ref* and *ij-mum1* genomic clones were identical, except for an additional 1.5kb DNA and *Mu1* element in each clone, respectively. An additional 1.5kb DNA of the genomic clone of *ij-ref* was located 300bp to the left of the *Mu1* insertion site of *ij-mum1*. The 1.5kb DNA contained *Bgl*I, *Eco*RI, *Hin*dIII, and *Sst*I sites. The locations of primers for PCR amplification are indicated as arrows (1 and 2) at the right of the 1.5kb of *ij-ref* and at the left of the *Mu1* element of *ij-mum1*, respectively. Restriction enzyme sites are indicated as follows: B: *Bam*HI, B1: *Bgl*I, E: *Eco*RI, H: *Hin*dIII, and S: *Sst*I.

atgggaggtacaagcgccgcagtcccgagccacggcctcgcctgTAGGGGTGAAAACGGGCG

GATACGGACGGATATTAGCTCATCCCGTATCCTACCCTAATGTATTTAAAACGGATTCGGGAT

CGGATACGGATAGTTAGAAACGGGACGGATACGGATAC.....1.5 kb .....

.....GTCCCGAATCTAAAAATTACCCGTATCCGTATCCGAAAAATGGTCCGAAT

CAGTATCCGTCCGATACCGTTTCGGTACCCTACCCGTTTTCACCCCTActcgcctgcgcgccc

ccggcagccgtcactctgaatccgcgggcggc.....

	5'	3'
Ac:	CAGGGATGAAA.....	.....TTTCATCCCTA
sh-m6233, wx-m1, and Adh-Fm335:	TAGGGATGAAA.....	.....TTTCATCCCTA
1.5 kb DNA in ij:	TAGGGGTGAAAACGGG.....	.....CCCGTTTTCACCCCTA

Figure 4A. Partial DNA sequences of the 1.5kb fragment inserted in the *ij-ref* allele. The sequences of 1.5kb DNA are in capitals, while the adjacent *ij* sequences are in lower case. The italic bases indicate the 8bp direct duplication of *ij-ref*. The 16 bases of bold character in both ends of the 1.5kb DNA show the inverted repeat. DNA sequences corresponding to the *Ac* transposase binding motif are underlined. Figure 4B. Terminal inverted sequences of the 1.5kb element of *ij-ref*, *Ac* and other *Ds* elements. Sequence differences in the 1.5kb element are underlined. The data are from Pohlman et al., 1983 for *Ac*, from Sutton et al., 1984 for *Adh-Fm335*, from Weck et al., 1984 for *sh-m6233* from Wessler et al., 1986 for *wx-m1*.

covery from the genetic lesion of *ij-ref* could be due to excision of the 1.5kb element, seven independent revertant alleles were analyzed for DNA sequences around the insertion site of the 1.5kb *Ds*-like element in *ij-ref*. Two germinal revertants described above and 5 new somatic revertant sectors were used. Comparison of partial sequences of the genomic clones and cDNA of *Ij* showed that the 1.5kb element was inserted into an exon of the gene. To clone DNA of the *Ij-Rev* alleles around the insertion site, DNA of *Ij-Rev* alleles was amplified by PCR (Polymerase Chain Reaction) using primers from the left of the 1.5kb DNA and from an intron right of the *Mu1* insertion site. The PCR products were sequenced. Sequences outside the duplication region were identical in *ij-ref* and all 7 *Ij-Rev* alleles. Along with partial sequences of the genomic clones of *ij-ref*, *ij-mum1* and cDNA of *Ij*, the sequences

ij-mum1 and cDNA	ccgagccacggcctcgcctg		cgcgcccccg
ij-ref	ccgagccacggcCTCGCCTG	(Ds)	CTCGCCTGcgcgcccccg
Ij-Rev #1	ccgagccacggcCTCGCCTG		cgcgcccccg
Ij-Rev #2	ccgagccacggcCTCGCCT		TCGCCTGcgcgcccccg
Ij-Rev #4	ccgagccacggcCTCGCC		GTCGCCTGcgcgcccccg
Ij-Rev #3,5,6,7	ccgagccacggcCTCGCCTCA		GTCGCCTGcgcgcccccg

Figure 5. DNA sequences of *Ij-Rev* alleles. Genomic DNA from two germinal and 5 somatic revertants were amplified by PCR. The primers are indicated in Figure 3. Part of sequences of 7 *Ij-Rev* alleles are listed along with *ij-ref*, *ij-mum1* and cDNA of *ij*. The duplicated host DNA is underlined.

of the seven *Ij-Rev* alleles around the insertion site of *ij-ref* are shown in Figure 5. None of the PCR clones contained any sequences from the 1.5kb element. However, there were differences in the direct duplication sequences among the different alleles. Four different sequences were found from 7 independent revertants. One of the somatic revertants contained the same sequences as the wildtype, which indicated that the *Ij-Rev* allele was generated by perfect excision of the element. All the revertant alleles maintained coding regions in frame after the element was excised.

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Differential regulation of flavonoid biosynthetic genes by the *P* gene

--Erich Grotewold, Thomas Peterson, Bruce Drummond, Brad Roth and Ben Bowen

The *P* gene regulates 3-deoxy flavonoid biosynthesis in maize. The 3-deoxy flavonoid flavan-4-ol undergoes a non-enzymatic polymerization to form the red phlobaphene pigment found in pericarps and cobs carrying a *P-rr* allele (*r*ed pericarp and *r*ed cob) (Styles and Ceska, Can. J. Genet. Cytol. 19:289, 1977). *P* regulates the accumulation in the pericarp of transcripts of the *C2*, *CHI* and *A1* genes (Grotewold et al., PNAS 88:4587, 1991). *C2*, *CHI*, and *A1* encode three enzymes required for biosynthesis of both flavan-4-ol (a 3-deoxy flavonoid) and anthocyanins (which are derived from 3-hydroxy flavonoids). On the other hand, *A2*, *Bz1* and *Bz2* encode enzymes specifically required for anthocyanin biosynthesis; the *P* gene has little or no effect on the expression of these genes in the pericarp (Grotewold, unpublished).

Two alternatively spliced *P* transcripts (P-cDNA1 and P-cDNA2) encode proteins with amino terminal regions homologous to the *myb* family of transcriptional activators (Grotewold et al., PNAS 88:4587, 1991). To determine which of the cloned *P* transcripts encodes the product responsible for the regulatory function of *P*, transient expression experiments using the particle gun were performed.

When P-cDNA1 (1802bp) under the control of the 35S promoter was co-bombarded into embryonic callus cells with a luciferase gene under the control of the *A1* promoter (*P* dependent expression), a dramatic increase in luciferase activity was detected (over 200 fold). However, a very low level induction (3-6 fold) was obtained when the *A1* promoter was replaced by the *Bz1* promoter (*P* independent expression). When P-cDNA2 was used instead of P-cDNA1, there was no detectable activation of either the *A1* or *Bz2* promoters.

These experiments not only point to P-cDNA1 as the *P* encoded regulator, but also indicate important differences in the ways in which *P* and *C1*, two maize *myb* homologous genes, regulate expression of the structural genes for flavonoid biosynthesis. Whereas *C1* (a *myb* homologous regulator of anthocyanin biosynthesis) requires a member of the *R* or *B* gene families for its regulatory activity, the regulation by *P* is clearly *R/B* independent. Furthermore, *P* does not efficiently activate the *Bz1* promoter, even in the presence of *R*. However, competition experiments suggest that the product encoded by P-cDNA1 may interact with *Bz1* promoter sequences.

We tried a complementation test by shooting P-cDNA1 into pericarps carrying a *P* gene deletion. No red cells were detected, even though many red cells are found when the same pericarps were bombarded with *R+C1*. Possibly the cells bombarded with P-cDNA1 produce the colorless flavan-4-ol, but it does not polymerize into the red phlobaphene pigment because we are not able to reproduce the conditions present in a naturally maturing ear; pigment formation is thought to require specific physiological conditions of the tissue such as maturation or aging (Styles and Ceska, Maydica 34:227, 1989). The alternative possibility is that the product encoded by P-cDNA1 does not activate the *C2* and/or *CHI* genes. Experiments are in progress to test whether P-cDNA1 can activate the promoters of these genes.

We thank Dr. Heinz Saedler and Dr. Alfons Gierl for the *A2* probe and Dr. Virginia Walbot for the *Bz2* probe.

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Abscisic acid regulation of *C1* transcription

--Nam Paek, Dong G. Bai, Clint W. Magill, B. Greg Cobb and J. D. Smith

In the appropriate genetic background, all of the viviparous maize mutants except *vp1* have colored aleurone. However, kernel blocks cultured in vitro on medium containing fluridone, which inhibits phytoene desaturase, are white with colorless aleurone.

Anthocyanin synthesis in maize aleurone requires *C1* transcription (Cone et al., PNAS 83:9631-9635, 1985), but *C1* is not transcribed in the absence of *Vp1* mRNA (McCarty et al., Plant Cell 1:523-532, 1989).

We used an ACR5855$_2$ stock (*A1 A2 C1 C2 Bz1 Bz2 R Pr Vp1*) to determine if transcription of *Vp1* or *C1* was affected by abscisic acid (ABA). Ears were harvested at 5 DAP and kernel blocks were cultured on media ± fluridone. ABA levels ±SE determined from 15 DAP embryos cultured without fluridone were 138.4±35.3 pg ABA embryo^{-1} and 1.5±0.2 pg ABA embryo^{-1} when cultured with fluridone. Antisense RNA transcripts from *Vp1* cDNA and from *C1* cDNA were used to test for hybridization signals with their respective mRNA from aleurone tissues of 13, 15 and 20 DAP kernels cultured with and without fluridone.

Hybridization signals were observed for *Vp1* mRNA at all ages for kernels cultured with or without fluridone. However, *C1* mRNA hybridization signals were detected only for kernels that were cultured without fluridone.

Vp1 transcription is not affected by ABA and we have been unable to identify any association of ABA with the protein encoded by *Vp1*. Thus, ABA appears to act as an independent transcriptional factor involved in the regulation of *C1* transcription. This would explain why ACR5855$_2$ kernels cultured on medium with fluridone are colorless, but they have colored aleurone on medium containing both fluridone and ABA (Smith and Cobb, MNL 63:57-58, 1989). In cultured kernels both the intensity and time of anthocyanin expression vary with ABA levels in the media, which suggests that ABA is the effective regulator of anthocyanin expression in maize aleurone when *Vp1* and *C1* are present.

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Viviparous-10: a new viviparous mutant in maize

--J. D. Smith and M. G. Neuffer

The viviparous mutants of maize can be classified into subgroups according to their phenotypes and metabolic effects. All known viviparous mutants are recessive and their common characteristic is that embryogenesis and seedling development is a continuous process which results in premature sprouting and seedling lethality unless embryos are rescued. Our classification system is an extension of the system first proposed by Robertson (1955). Classification criteria are based on self-pollinated ears from plants heterozygous for specific mutants in an ACR5855 background (*Y; A1 A2 Bz1 Bz2 C1 C2 R Pr Vp1*).

Class I. Viviparous, yellow endosperm, green seedlings, colorless aleurone, normal carotenoids, normal abscisic acid (ABA). The only known Class I mutant is *vp1*.

Class II. Viviparous, white endosperm, albino seedlings, colored aleurone, carotenoid-deficient, reduced ABA. Mutants include *vp2*, *vp5*, *vp5*, *vp9*, *w3*, *y3* and *y9* (*y3* is temperature sensitive and sectorial, *y9* is leaky, and both have pale yellow endosperm).

Class III. Viviparous, yellow endosperm, green seedlings, colored aleurone, normal carotenoids, reduced ABA. The only previously identified Class III mutant is *vp8*.

We recently identified a new mutant which meets all of the Class III criteria described above. Numerous crosses with *vp1* and *vp8* have all given negative results, and some F2 ears from *vp1* crosses show dihybrid segregation with both colored and colorless viviparous kernels.

We propose to designate this mutant as Viviparous-10 (*vp10*). Viviparous-10 (previously *vp*-86GN5*) is one of a number of viviparous mutants induced by EMS treatment of pollen by Neuffer. Rescued embryos are viable, although we have not yet succeeded in producing seed from homozygous *vp10* plants. Seedling leaves typically fail to separate at their tips and have a rosette appearance, but growth appears to be normal after leaf tips are mechanically separated. Phenotypic differences in seedling growth patterns suggest that *vp10* precedes *vp8* in the ABA biosynthetic pathway.

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***En/Spm* as a tool for gene tagging in heterologous species**

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

An excision assay system for *En/Spm* in transgenic tobacco defined recently (Frey et al., EMBO J. 9:4037-4044) helped to identify the transposon-encoded trans-acting functions (TNPA and TNPD) necessary for excision. In this system, excision and reinsertion of a 2.2kb *dSpm* element, a natural internal deletion mutant of the autonomous element, takes place with similar characteristics as *En/Spm* transposition in maize.

This system was modified in order to tailor it for gene tagging. A 4.2kb receptor element marked by the insertion of a

DHFR gene, which confers resistance to methotrexate, was constructed. This artificial element is capable of normal excision and reinsertion in dependence of TNPA and TNPD in transgenic tobacco. New excision reporter constructs were made in which the elements are inserted in the untranslated leaders of the selectable marker genes *bar* and *Spt*. The timing and frequency of excision can be manipulated by expressing the trans-acting factors under the control of different promoters. The *bar* gene proved to be an excellent marker for the selection of germinal revertants among tobacco seeds germinating in vitro in the presence of 100mg/l L-PPT. Selection of germinals is also possible in the greenhouse by spraying seedlings with the herbicide BASTA (Hoechst AG). When TNPA and TNPD were expressed from CMV 35S promoter, the average frequency of germinal excision observed for the 2.2kb *dSpm* from a *bar* reporter was about 25%. In some plants, an early excision of the *dSpm* resulted in 100% of the gametes carrying the same reversion event. The marked receptor element has both lower somatic and germinal excision frequencies but this disadvantage is compensated for by the fact that it can be selected for reintegration.

Tobacco proved to be a suitable test system to study and manipulate *En/Spm* transposition, but since it is amphidiploid it is not a convenient species for transposon tagging. Two diploid species were chosen, *Petunia* and *Arabidopsis*, in which a working transposon tagging system for gene isolation would be desirable. Excision reporters and trans-activating constructs previously tested in tobacco were transformed into *Petunia* and *Arabidopsis* and are presently being joined by sexual crossing. These two species were also transformed with the autonomous element *En-1*. It was found to transpose in *Arabidopsis* in all the independent transformants analyzed as well as in the two subsequent generations. The frequency of somatic excision is over 50% and footprints left behind are normal. The autonomous element is capable of activating the 2.2kb *dSpm* with high frequency. Preliminary experiments show that the frequency of germinal excision of *En-1* in *Arabidopsis* is about 3%. Concerning *En-1* activity in transgenic *Petunia*, none of the four independent transformants analyzed so far showed excision.

Asymmetric ends are required for excision of *En/Spm*

--Monika Frey, Julio Reinecke, Heinz Saedler and Alfons Gierl

The correlation of genetic, molecular and biochemical data identified two kinds of cis-acting elements: the terminal inverted repeats (TIRs) and the subterminal TNPA binding motifs. It has been proposed that binding of TNPA to its motifs induces complex formation between both ends by a "zipper"-like mechanism leading in consequence to alignment of the TIRs and subsequently, after binding of TNPD to the TIRs, to the excision of the element (Frey et al., EMBO J. 9:4037-4044). One prediction of this model is that the pattern of the TNPA binding motifs at the subtermini plays a critical role in excision because altered distributions might interfere with "zipper" formation. On the other hand, alterations of the inner part of the element should only gradually influence the excision rates.

A mutational analysis of the cis-determinants was initiated in order to test the predictions mentioned above. So far two *dSpm* derivatives have been constructed and analyzed in the transgenic tobacco test system. The first is 716bp long, has intact cis-determinants and consists of 270bp of the 5' end and 446bp of the 3' end of *En-1*. This element is excised in tobacco in the presence of TNPA and TNPD as revealed by restoration of the reporter gene (*Gus* gene), Southern analysis and PCR amplification of the excision site. Excision generates the expected footprints, however, the excision frequency is reduced to about 20% compared to a native 2.2kb *dSpm* element (*Spm-l8*).

The second *dSpm* construct has two identical ends, i.e., the 446bp of the 3' end of the 2.2kb *dSpm* element are substituted by the 270bp of the 5' end. Neither histological staining for *Gus*-enzyme activity, nor Southern analysis revealed excision of this element, only with PCR excision events could be amplified. Sequence analysis of two of these PCR products displayed a striking feature. In contrast to all excision events analyzed in the tobacco system before, employing native maize *dSpm* elements or *En-1*, only "symmetrical" footprints are generated: in the one case both target site duplications (TSDs) were retained and in the other case one base pair was deleted from each TSD. Therefore, the excision frequency for the 5' end/5' end element is not only extremely low, but also the mechanism might be altered, however, excision is still dependent on TNPA and TNPD.

These results seem to confirm the prediction of the "zipper"-model that asymmetric ends are required for excision. In this respect *En/Spm* resembles *Ac* and the *P* element of *Drosophila*, which are also defective in excision when they have identical ends.

Transposition of *En/Spm* requires a bi-functional protein

--Stefan Trentmann, Monika Frey, Heinz Saedler and Alfons Gierl

The *En/Spm*-encoded TNPA protein binds to 12bp sequence motifs in the subtermini of *En/Spm* (Gierl et al., EMBO J. 7:4045-4053). TNPA is required for excision of *En/Spm* and was proposed to serve as a "glue" for the association of the element's ends (Frey et al., EMBO J. 9:4037-4044). The formation of TNPA-DNA complexes was now analyzed in more detail by gel retardation, using individual binding motifs and in vitro translated TNPA protein.

Binding of TNPA to DNA leads to the formation of two complexes: a fast migrating complex that contains one TNPA molecule and a slower migrating complex that contains two TNPA molecules which are associated by protein-protein interaction. The deletion analysis of TNPA revealed two functional domains: one for DNA binding and one for dimerization of the protein. The region between position 122 to 427 constitutes the DNA binding domain, while amino acids 428 to 542 are required for the dimerization domain of TNPA. Mutant TNPA proteins that are defective in either the DNA binding or the dimerization domain are also defective with respect to promoting excision in the transgenic tobacco test system. We therefore propose that the association of the ends of *En/Spm* occurs via the dimerization domain of TNPA proteins that are bound to the various subterminal sites at each end of *En/Spm*.

Structure and function of different *C1* alleles

--Brian Scheffler, Philipp Franken, Elvira Tapp, Andreas Schrell, Heinz Saedler and Udo Wienand

In addition to the wildtype *C1* and mutant *C1-I* allele analyzed previously (Paz-Ares et al., EMBO J. 6:3553-3558, 1987; Paz-Ares et al., EMBO J. 9:315-321, 1990) further *C1* alleles have been cloned and sequenced. Among those were the over-expressing allele *C1-S*, the light inducible allele *c1-p*, the recessive allele

c1-n, and the *Ds* element induced allele *c1-m1* (a genomic clone of *c1-m1* was kindly provided by K. Cone).

The coding region of *C1-S* and *c1-m1* is very similar to that of the wildtype *C1* allele, whereas the *c1-p* and *c1-n* alleles show several sequence differences. In the *c1-p* allele these lead to various amino acid changes and in the *c1-n* allele to a frame shift mutation. Major sequence differences between the *C1* alleles have been detected in the region 3' of the codogenic sequence. There, three deletions (455bp, 1159bp, 216bp) are present in the *c1-p* allele. The *C1-I* allele seems to have a deletion identical in size and position to the 455bp deletion present in *c1-p*.

The promoter sequences of the *c1* alleles analyzed are very homologous (up to position -600) and differ only in two short footprint like sequences (boxI and boxII) close to the putative CAAT box. Little sequence alterations (insertions and deletions) in these boxes may be correlated with the different expression patterns of the alleles. This assumption is supported by particle gun experiments. The full length *C1* promoter and deletion products thereof were fused to the luciferase gene and delivered into aleurone of germinating *c1* recessive kernels. The expression data show that sequences necessary for *C1* gene activity are present in the vicinity of boxI and boxII.

Northern experiments with mRNA isolated from maturing (30 DAP) and germinating kernels support the results of the promoter analysis and indicate different modes of regulation of *C1* and the structural genes *C2* and *A1* within the various alleles.

Analysis of functional domains of the *C1* encoded protein

--Philipp Franken, Susanne Kartzke, Peter A. Peterson, Heinz Saedler and Udo Wienand

The putative *C1* encoded protein contains two major domains: a basic one at the amino terminus with possible DNA binding capacity and an acidic domain for activation of transcription at the carboxy terminus. Functional analysis of these domains has been carried out by the investigation of revertants of transposable element induced mutants. Also in vitro constructs containing fusions of various domains of *C1* and *C1*-related cDNAs have been tested for activity.

The *En1* element insertion sites of seven *C1* mutants have been determined and revertants of four of these were further investigated. A pale revertant of one such mutant (*c1-m55437*) was analyzed and showed a deletion of a lysine in the basic domain of the putative *C1* encoded protein at amino acid position 30. This indicates that this position in the *C1* protein is of functional importance. Colorless, pale and colored revertants of the mutant *c1-m11702* have also been investigated. The *En1* element in *c1-m11702* is integrated in the acidic domain of the putative *C1* encoded protein at amino acid position 223. The pale and colorless revertants represent frame shift mutations, whereas the colored revertant contains an additional amino acid. The result of this analysis shows that sequence and charge of the acidic domain of the *C1* protein is important for its activator function.

For reconstruction of *C1* like proteins, experiments using the *C1* cDNA as well as two *C1*-related cDNAs from maize (Zm1 and Zm38; Marocco et al., MGG 216:1347-1368, 1989) have been used. Particle gun experiments with various constructs carrying different combinations of the putative DNA binding and trans-activating domains indicate flexibility of the acidic carboxy terminus. One of the *C1* homologous cDNAs and fusion constructs thereof were also capable of *A1* gene activation, but not of activation of the entire pathway.

Promoter analysis of the anthocyanin gene *A1*

--Andreas Schrell, Heinz Saedler and Udo Wienand

The *A1* gene promoter has been analyzed for functionally important sequences using particle gun experiments. The full size *A1* promoter (1758bp) as well as deletion derivatives were cloned in front of the luciferase gene. The constructs, together with cDNAs of the effector genes *C1* and *R1* were delivered into germinating kernels of the double recessive genotype *c1 r1*.

The analysis of the *A1* promoter constructs revealed that a deletion up to position -303 still has almost full size promoter activity. However, *A1* promoter activity is drastically reduced by the deletion of sequences downstream of position -214. Further fine structure analysis of putative myb and myc related sequences located in this area should show whether they represent binding sites for the *C1* and *R1* encoded proteins.

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The structure of the DNA-binding domain of the *Ac* transposase

--Siegfried Feldmar and Reinhard Kunze

The ORFa protein, the putative transposase of the maize *Ac* transposon, has been over-expressed in *E. coli*. After renaturation, the protein has DNA binding properties indistinguishable from those of the ORFa protein expressed in insect cells. In order to characterize the DNA binding domain, we have constructed several mutant ORFa proteins (Feldmar and Kunze, EMBO J. 10:4003-4010, 1991). By deletion analysis the DNA binding domain was mapped to the N-terminal third of the protein, between amino acids 136 and 270. The protein segment between amino acids 159 and 207 carries many positively charged residues (14 basic versus one acidic residue). Of these, the basic residues between 190 and 200 are essential for the DNA binding reaction, as their substitution by uncharged amino acids results in the loss of DNA binding activity, whereas a similar exchange in neighboring regions does not affect it.

The amino acid sequence in this short region is indeed crucial for DNA-recognition. At a certain position not only a basic amino acid is required, but its identity is important, too. This was shown by exchanging histidine$_{191}$ and arginine$_{193}$. The resulting mutant is completely DNA binding deficient.

Immediately downstream of the DNA binding domain a region with weak homology to a helix-loop-helix motif is located. This motif was found in some DNA binding domains of other proteins. We have constructed three mutants with partial or total deletions of the upstream and downstream helices, respectively. All three mutants have virtually the same DNA binding properties as the wildtype protein. We conclude that the potential helix-loop-helix structure is not required for DNA binding. It might, however, have a different function in vivo, since its mutation abolishes transposase function (see report from Lütticke, Courage and Kunze, this issue).

Mutational analysis of *Ac* ORFa protein in vivo

--Ralf Lütticke, Ulrike Courage and Reinhard Kunze

The development of a transient in vivo transposition assay

(Houba-Herin et al., MGG 224:17-23, 1990) enables us to test in vitro mutated *Ac* ORFa protein for a loss or reduction of transposase function. We have constructed a set of ORFa protein mutants by introducing 6bp linkers into the *MaeI* restriction sites of the *Ac* cDNA. These mutations were introduced into a plasmid which contains a 5'-terminally deleted derivative of the *Ac* cDNA behind the 2'-promoter. From this plasmid an ORFa protein derivative starting at amino acid 103 of the 807 codon ORFa and preceded by 7 out-of-frame ATGs is expressed (see also report by Becker, Lütticke and Starlinger, this issue).

Twelve mutants were created, each carrying a single 6bp linker-insertion. None of these affect the DNA binding domain of the ORFa protein. Four mutants induced *Ds* excisions from the cotransfected reporter plasmid with the same efficiency as the wildtype ORFa protein. Of these, the most N-terminal insertion behind amino acid 51 of the wildtype ORFa protein is located in the leader region, and therefore cannot alter the properties of the truncated ORFa protein. However, three insertions within the truncated ORFa, behind amino acids 623, 754 and 771 do not disturb transposase function either. These three insertions are located in protein regions with no obvious homology to the open reading frames of transposable elements *Tam3* from *Antirrhinum majus* and *Hobo* from *Drosophila*.

An insertion of two amino acids behind ORFa residue 270 destroys transposase function. This position is situated within the downstream helix of the putative helix-loop-helix motif. Although this finding is no evidence for the formation of a helix-loop-helix structure, it demonstrates that this segment is essential for the transposase (see also report of Feldmar and Kunze, this issue).

Insertions behind ORFa residues 369, 390, 445, 462, 577, 585 and 709 abolish the capacity to mobilize a *Ds* element, too. They all are located within protein regions with strong sequence homologies to the open reading frames of *Tam3* from *Antirrhinum majus* and *Hobo* from *Drosophila* (Feldmar and Kunze, EMBO J. 10:4003-4010, 1991). These results indicate that essential transposase functions different from the DNA binding reaction are located in the C-terminal two thirds of the *Ac* ORFa protein. We are presently testing if less well conserved regions of the ORFa protein are more tolerant against two amino acid insertions.

Immunohistochemical detection of the maize *Ac* protein in endosperm nuclei

--Manfred Heinlein

By using five antisera raised against different epitopes of the *Ac*-protein a signal has been obtained in endosperm nuclei of *Ac*-containing endosperm, which is absent in endosperm-nuclei devoid of *Ac*. The signal is not seen if various preimmune sera are taken as primary antibody. If an antibody is used which has been incubated with its specific antigen prior to its application the sensitivity of the detection is much reduced. The signal appears as large rod-like complexes, about 2µm long and 0.2µm wide. With decreasing *Ac*-dosage the complexes become less detectable. The visibility varies between endosperms containing different *Ac*-bearing alleles. In nuclei double-stained for DNA and *Ac* protein, the complexes in some cases appear to be perpendicularly oriented relative to DNA and thus support the assumption that the rod-like shape of the complexes might be due to binding of the protein to sites located side by side on different chromatids of endochromosomes within endopolyploid endosperm nuclei.

Further studies on the regulation of *Ac*-dependent events

--Manfred Heinlein and Peter Starlinger

Two *Ac*-dependent events usually do not occur at the same time and in the same cells (MNL 65). Therefore, the rate-limiting step of the decision, whether transposable elements *Ac/Ds* undergo excision/transposition events in the cell, is not the synthesis or activation of a trans-acting factor. However, the probability of *Ds*-excision is different at a certain time during development if different *Ac*-elements are used for trans-activation. This indicates that *Ac* elements differ in trans-activity. This is further supported by the observation that different *Ds* elements respond similarly in the presence and dose of the particular *Ac* element. In last year's report the responses of *bz-m2(DI)* in kernels carrying one or two doses of the *wx-m9 Ac* or *wx-m7 Ac* alleles, respectively, have been described (also in: Maydica 36:309). The two *Ac* elements are identical in sequence and are inserted in the same gene and in the same orientation. Hybrid F1 *wx-m7 Ac/wx-m9 Ac* plants have now been crossed to *bz-m2(DI)* and other tester plants to test whether the observed differences are due to varying genetic backgrounds of the plants used in the analysis. The postulated factors of the genetic background which should have been mixed in the hybrid should segregate independently and because of this should cause the appearance of new patterns on the tester ears. However, the known *Ac*-specific patterns appeared in the progeny. The kernels showing the particular reversion pattern in the aleurone also showed the particular *Wx* reversion pattern in the inner endosperm. The kernels also contained the appropriate *Ac* element as has been proven by PCR. The observed regulation of excision events is therefore rather due to *Ac*-specific properties than due to background effects. However, the possibility that the differences in phenotypes are due to genes closely linked to the *Ac* element cannot be excluded.

Ac elements which differ in their action in trans most likely should be differentially expressed. Comparisons between *wx-m7* and *wx-m9 Ac* did not reveal convincing evidence for differences in *Ac*-mRNA levels in seedlings. This might be different in endosperm, where the *Wx*-locus is expressed. First experiments were performed to test the influence of host-gene expression on excision. Preliminary results suggest that the *Ac* element of *bz-m2* is excised 20-30 times less often in *C-I C-I C* and *C-I C-I C-I* kernels than in *C C C* kernels as revealed by comparison of excision bands in Southern blot experiments. Since the effect is seen by excision of an *Ac* element it cannot be distinguished whether the excision of the element is influenced directly or rather indirectly through altered *Ac* expression.

Ac dosage effects are often measured by comparing patterns on progeny kernels of reciprocal crosses between *Ac*-bearing maize lines and *Ds*-bearing maize lines. The progeny of the two crosses not only differ in *Ac* dose but also in *Ds* dose. To measure the influence of *Ds* dose (not the overall *Ds* copy number, but the dose of a particular *Ds* element with visible phenotype), the reversion pattern of *wx-m9 Ds* has been compared between kernels carrying a constant dose of *bz-m2 Ac* but one, two and three doses of *wx-m9 Ds*. It appeared that the difference in reversion frequency was much stronger between two and three *Ds* than between one and two *Ds*. The two individual copies of *Ds* derived from the female are excised much less frequently than the one copy derived from the male, which suggests that probably paternal imprinting is involved. The frequency of the individual *Ds* elements in

three-*Ds* kernels in which the elements of maternal and paternal origin have been combined clearly exceeds the sum of the observed values in one- and two- *Ds* kernels. This indicates that the combination of paternally and maternally inherited elements might promote excision. Unless the *Ds9* element does encode a participating trans-acting product, one might speculate that the element derived from the male is able to complement some inhibitory properties of the element derived from the female, probably by interphase pairing. Further studies employing the *Ds* element present in the *bz-m2(DI)* allele are in progress.

The influence of promoter strength on *Ds* excision frequency in a transient excision assay

--Detlef Becker, Ralf Lütticke and Peter Starlinger

We have developed a transient petunia protoplast assay to test the biological activity of different *Ac* constructs (Houba-Herin et al., MGG 224:17-23, 1990). This assay allows the measurement of the relative transposase activity of *Ac* and derivatives of *Ac*.

The protoplasts have been cotransfected with two plasmid DNA's. One plasmid carries the β-glucuronidase (GUS) reporter gene under the control of the 1'-promoter. An insertion of a nonautonomous *Ds*-like element between the promoter and the coding sequence in this target plasmid abolishes the expression of the GUS gene. The second plasmid provides the transposase function. The excision of *Ds* leads to restoration of the reporter gene. After fixation of protoplasts on nitrocellulose filters and histochemical staining it is possible to detect β-glucuronidase activity as blue spots on the filters. The number of blue stained protoplasts is a measure of the excision frequency.

To our surprise we discovered in experiments with this transient expression system that a truncated piece of the ORFa coding sequence of *Ac* under the control of the 2'-promoter leads to a high excision frequency of the *Ds* element out of the target plasmid. As we did not do any transcription measurement, we cannot say for sure whether transcription of the *Ac*-coding sequence has been enhanced by the 2'-promoter or if the stability of the transcript has been changed by the truncation.

An analysis of transcriptional fusions using the complete ORFa coding sequence (with all introns) under the control of CaMV35S, 1'- and 2'-, *Nos*- and *Ac*-promoter leads to a low number of blue stained cells. The 2'-promoter enhanced the excision frequency 4-fold and the *Nos*-promoter 2-fold compared to the *Ac*-promoter. It seems that the number of blue stained cells reflects the strength of the different promoters.

If a truncated ORFa coding sequence with 3 out of frame ATG's upstream of ATG_{10} (codon 103 in the full length ORFa protein) is under the control of CaMV35S-, 1'- and 2'-promoter a significantly higher number of blue spots can be observed compared to the complete ORFa sequence. A lower rate of excision is obtained if the same truncated ORFa coding sequence is under the control of the *Ac*- or *Nos*-promoter. The removal of all out of frame ATG's upstream of ATG_{10} and expression under the control of the 2'-, *Nos*- or *Ac*-promoter leads to high excision frequency of the *Ds* regardless of promoter strength.

These results suggest that the maximal number of spots in a particular experiment can already be reached by the truncated ORFa coding sequence under the control of the weak *Ac*-promoter. But if the translation efficiency of the truncated ORFa coding sequence is reduced by upstream out of frame ATG's or the complete ORFa coding sequence has been used the excision frequency becomes dependent on transcription rate/promoter strength. The results suggest also that the number of excision events in a particular experiment reaches a saturation level.

The same results can be obtained using a cDNA of ORFa under the control of the 2'-promoter. The complete ORFa cDNA leads to a low number of blue spots whereas the truncated cDNA shows comparable results to the truncated genomic ORFa sequence. Further deletion of the ORFa coding sequence beyond the 10.ATG starting at amino acid 137, that is immediately behind a ten-fold repeat of the dipeptide Pro-Gln/Glu, abolishes transposase function.

The data with the complete ORFa sequence (genomic or cDNA) suggest that the N-terminal part of the ORFa protein exerts a regulatory function on transposition frequency. It is possible that the frequency of the transposition/excision rate is regulated on the protein level. This idea is supported by the following observation: a mixture of full length coding sequence and truncated coding sequence, both under the control of a 2'-promoter, leads to a number of blue stained protoplasts, which is lower than the number obtained with the truncated ORFa sequence alone. It seems that the presence of full length ORFa protein has a negative influence on the high activity of the truncated protein.

A surprising result has been observed by using different target plasmids. The presence of the luciferase gene in the vicinity of the *Ds*-like element leads to a 6-10-fold higher number of blue spots although the *Ds* elements in both plasmids are identical. We do not know yet if this result reflects a higher excision rate of *Ds* out of the luciferase containing target plasmid or if this result is due to a higher expression level of GUS-enzyme after excision of *Ds*.

Preliminary experiments indicate that the removal of the DNA sequence downstream of the *Bam*HI site (bp 181) in the *Ds* element leads to a dramatic decrease in excision rate in the presence of complete or truncated ORFa coding sequence. This result, which is different from the results obtained in a callus assay in tobacco by Li and Starlinger (PNAS 87:60446048, 1990), suggests that in *Petunia hybrida* the DNA sequence located 3' to the *Bam*HI site is important with regard to the cis-acting sequence requirements for excision. This result also shows that the quality of transposase action has not been changed by the removal of the first 102 amino acids of the ORFa protein. The tobacco results may then be due to a saturation effect of *Ac* excision under the conditions used in the callus assay.

COLUMBIA, MISSOURI
University of Missouri

Variable short internodes

--Gregory G. Doyle

Variable short internodes (*vsi*) is a recessive mutant that arose spontaneously. Its expression is quite unusual. There are clusters of short internodes generally from 2 to 4 in number which are from 2.5 to 4cm in length. The other internodes are of normal length. These clusters are usually at the base of the plant although they may be found anywhere on the plant. In a few cases two clusters of short internodes were found on the same plant. It

is possible that *vsi* is a temperature sensitive mutant and a minor cold wave in the early spring sets up this phenotypic response.

Tripsacum mapping project - progress report

--C.A. Blakey, C.L. Dewald* and E.H. Coe, Jr.
*USDA-ARS, Woodward, OK.

As of January 1992, data for the molecular map under construction in *Tripsacum dactyloides* are summarized in the following table:

	RFLP Probe DNA Source	
	Maize	*T. dactyloides*
TOTAL NUMBER OF PROBES SCREENED	215	73
Polymorphic in *T. dactyloides*	115	42
In maize (Tx303/Co159)		30
Hybridization, non-polymorphic in *T. dactyloides*	90	31
No hybridization to *T. dactyloides*	33	--
to maize	--	15
TOTAL NUMBER OF RFLP LINKAGE GROUPS of 2 or more markers	19	
Mapped markers in linkage groups	67	
Molecular	52	15
Phenotypic	--	1
Unlinked mapped markers	4	4

Instability in the *An1* component of the *idd*-2286A* mutant

--M. G. Neuffer and S. Chao

In MNL 65:52 we presented what we thought to be a simple explanation for the phenotypes found in progeny and allelism tests of the EMS induced mutants *idd*-2286A* and *id*-A972*. The preliminary observations were correct but not as consistent as indicated. It now appears that the *idd* mutant is quite variable in its dwarfing activity with some instances where homozygous *idd* individuals are distinctly short, have a dark green color and are indeterminate in growth while others are somewhat taller and have a normal green color. Some of these latter mutant plants, which were short as seedlings, grow almost normally and become determinate anther ear types that are only a little shorter than normal sibs. Furthermore 2 other EMS induced mutants, *id*-392B* and *id*-2373A*, show considerable variation in height suggesting that they also may be unstable with regard to an associated plant height trait. It is true that the *idd* mutant when crossed on *an-6923* produces anther ear plants proving allelism, but these *idd/an-6923* plants are taller and less extreme than homozygotes for the reference allele. The relationship between the anther ear and indeterminate traits on this chromosome segment are still unclear.

Location and designation of four EMS induced kernel mutants

--M. G. Neuffer

sh6, shrunken kernel, 7S (was *sh*-1295*). Shrunken opaque kernel; normal size; produces a pale green lethal seedling. Like *sh1* but more extreme.

[ed. note: Compare note from Ames by P. S. Stinard; presumed allelism is to be tested next season.]

o14, opaque kernel, 6L, distal to *Pl1* (was *o*-924*). Large opaque kernel with mostly floury starch except for a small amount of corneous starch near the base on the abgerminal side. Grows to a normal green seedling which gradually develops an iron deficiency type yellow striped appearance and is slow in growth.

dek32, defective kernel, 1S (was *shop*-1322A*). Dull opaque dented normal size non-viable kernel; endosperm floury and soft, embryo tiny, degenerate and necrotic resting in a much larger embryo cavity. Not allelic to *dek1*.

dek33, defective kernel, 5L, between *bm1* and *pr1* (was *fldt*-1299*). Opaque floury dented wrinkled non-viable kernel of nearly normal size. The endosperm is floury with little or no corneous starch. Some kernels have a patch of normal appearing tissue surrounding the silk attachment, apparent as a "Navajo"-like colored or mottled spot in the presence of the appropriate aleurone anthocyanin color genes (similar to *dek17*). The embryo is variably degenerate, with development arrested at Abbe and Stein stage 3; occasionally viviparous. Cultured immature embryos germinate to produce amorphous masses of callus and distorted shoots and roots. Linkage tests give the following order and map distances: *A2* - 7 - *bm1* - 13 - *dek* - 21 - *pr1*.

Location and designation of 8 dominant mutants from chemical mutagenesis and spontaneous origin

--M. G. Neuffer

These include 7 lesion mimics and 1 semi-dwarf which were located by crossing with the *wx* marked translocation set. The locations are given to the nearest *wx1* T breakpoint.

Les11, Lesion, 2S (was *Les*-1438*). EMS origin. Like *Les1* but slightly later in expression and less responsive to temperature and to background genotype. Located 14±3cM from *wx1* T2-9c.

Les12, Lesion, 10S (was *Les*-1453*). EMS origin. Many small to medium, chlorotic to necrotic lesions form in clusters on the leaf blade beginning at the 5 leaf (20 day) stage. These lesions rapidly coalesce to form large senescent areas that may spread over the whole leaf and cause early death in some genetic backgrounds such as Mo20W. Not linked to *R1*.

Les13, Lesion, 6L (was *Les*-2003*). EMS origin. Frequent small to medium necrotic spots on leaf blade, sheath and culm, appearing at the 5 leaf stage. Some enlarge and coalesce to form long necrotic strips along leaf veins eventually spreading and causing senescence of the whole leaf. Mutant plants are lighter green and 1/3 to 2/3 normal height. Located 11±3cM from *wx1* T6-9b.

Les14, Lesion, 3L (was *Les*-2004*). EMS origin. Many small round brown necrotic spots with light centers, some with anthocyanin halos, on leaf blade beginning at the 6 leaf stage. No reduction in height or vigor. Located 11±4cM from *wx1* T3-9(8562).

Les15, Lesion, 2S (was *Les*-2007*). EMS origin. Tiny yellowish green plants with many small chlorotic and necrotic lesions on speckled yellow green leaf blade background that looks like iron deficiency symptoms. Located 2±1cM from *wx1* T2-9b.

Les16, Lesion, 10S (was *Les*-2016*). EMS origin. Pale green plant that develops small chlorotic lesions on the leaf blade just before flowering. Located 10±3cM from *wx1* T9-10b.

Les17, Lesion, 3L (was *Les*-2345*). Spontaneous origin. Profuse small to medium chlorotic and necrotic lesions expressed at 8-10 leaf stage causing plants to have a light green color. Occasional normal green non-lesion sectors appear on leaves. Located 3±2cM from *wx1* T3-9c. In progenies segregating *Wx1* vs. *wx1* mutants rarely expressed in *wx wx* plants. Location determined by excess (over 1:1) of mutants in the *Wx wx* class for *wx1* T3-9c backcross but 1:1 for all the others.

Sdw2, Semi-dwarf, 3 (was *D*-1991*). Nitrosoguanidine ori-

gin. Short plant, 1/3-1/2 normal height, with normal green erect leaves. Does not respond to gibberellins; no anthers in ear. Location 6±3cM from *wx1* T3-9c. 8±3cM from *Lxm*.

Further characterization of the leaf development mutant, *lbl*

--Donald Miles

We have recently described an interesting developmental mutant from Mutator active lines which has a variable effect on the amount of leaf blade tissue that develops (Miles, MNL 63:66-67). We have named this locus leaf blade less (*lbl*) which describes the most extreme phenotype exhibiting complete loss of leaf blade without reduction of the midrib vascular tissue. The absence of blade tissue can vary from complete loss of blade tissue to only a 10% loss of the margins of the leaves. This variation has been classified into a series of five stages from the most extreme (stage 1) to the least affected phenotypes (stage 5). Figure 1A illustrates a mature *lbl* plant which shows the most extreme expression. Most leaves are stage 5 with no blade tissue while a few leaves express stage 3 with half leaves. Figure 1B shows a stage 3 half leaf.

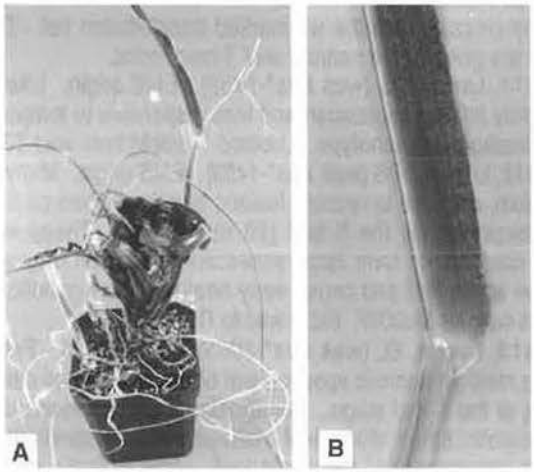


Figure 1. A, Growth of an *lbl* plant at 30 C showing the extreme expression of the mutation. B, Shows a typical stage 3 leaf with one half of a blade.

We previously suggested that there was a temperature effect on the expression of *lbl*. To test this we germinated kernels and grew seedlings for 15 days under 28 C (14hr) days and 24 C nights in growth chambers. The light intensity throughout this experiment was maintained at 300μmol m^2 s^{-1}. Under this environment, *lbl* was expressed mildly as stage 3 to 5 of leaf development. At the end of 15 days, half of the mutant seedlings were shifted to 18 C and half to 30 C and grown for 75 more days to test the temperature effect. *lbl* continued to be expressed under both growth temperatures, though the expression was much more severe when grown at 30 C. At 30 C the development was shifted more to the extreme stages 1 to 4 while at 18 C the development was less severe, most often at stages 3 to 5. Certainly the expression of *lbl* is not on or off in response to these temperature differences but its effect is ameliorated by lower than normal growth temperature.

The surprising observation made on plants grown at 30 C was that stage 4 leaves often produced a small growth of 'prop-root-like' tissue from the abaxial (lower) side of the midrib at the junction of the blades and the vascular tissue (Figure 2). Stage 4 is

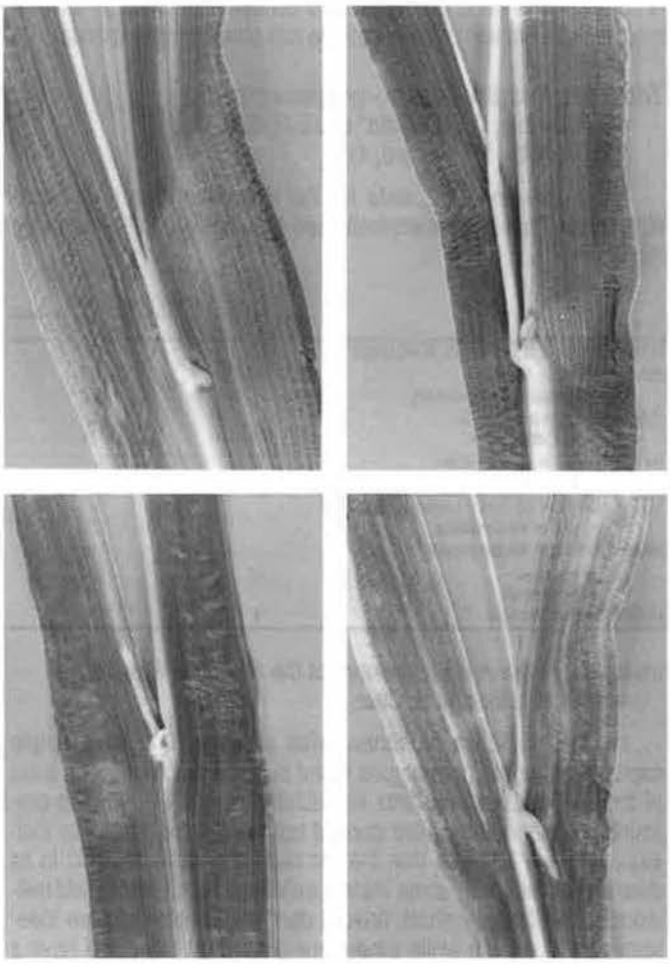


Figure 2. Growth patterns of *lbl* stage 4 leaves from different plants at 30 C. The distal end of the leaf is to the top of the photograph. This shows the typical 'prop-root-like' tissue projections on the abaxial side of the leaf at the junction of the midrib and the blades.

described as a leaf split at the tip into three structures, left and right leaf blades and central cylinder of vascular tissue which meet to form a complete leaf at about mid leaf. This 'prop-root-like' growth was 0.5 to 1.0cm in length and appeared to consist primarily of vascular tissue. It always appeared just below the junction of the leaf blades with the vascular cylinder. It extended up or down but appeared to be growing with a positive gravitropic response. The meristem responsible, the precise cellular nature and the regulation of this growth are unknown.

Identification of a putative QTL affecting ear shank length

--E. A. Lee, L. L. Darrah and E. H. Coe, Jr.

Mo17HtxB73Ht F1 hybrids containing one, two or three doses of the short arm of chromosome two were generated using a B73Ht version of TB-2Sa. While evaluating these F1's as part of another study during the summer of 1991, we noted that a dramatic difference for ear shank length existed among the three dosage classes. Ear shank length was 2.3, 10.7 and 20.2cm for the one, two and three dose classes, respectively. This direct response to chromosome arm dosage suggests that a gene(s) affecting ear shank length is located on the short arm of chromosome two.

DEFIANCE, OHIO
Defiance College

Paramutation: A system for temperature programmable transgenerational genetic change coupled to tassel determination

--Bernard C. Mikula

Paramutation, as reported by the late R. A. Brink in 1956, involved the production of a heritable change in the kernel pigment expression of the *R* gene by its allele *R-st*. When testcrossed, all *R* genes emerging from the *RR-st* heterozygote were changed. Effort has been directed toward achieving binary control of paramutated *R* gene expression by controlling early developmental conditions of seedlings with a paramutation sensitive *R* gene. In Vol. 65 of MNL, I reported that, in testcrosses, near binary control of the level of paramutation of the *R* gene was achieved in plants which as seedlings were grown for their first three weeks under 28 C or 22 C temperatures. Sib seedlings of the 1990 *RR-Ist* heterozygotes were used in 1991 to explore the developmental time interval when paramutation could be subject to temperature control. Seedlings were grown in constant light and temperature conditions until ready for floral induction. Light was provided by 14 VHO 200W daylight type fluorescent lights supplemented by 12 60W incandescent lamps. Seedlings were started 60cm from the light source and grew toward the light for the period of time held in the growth chamber. By day 10 seedlings raised in constant light at 32 C showed necrotic conditions in the youngest leaf tissues, a typical calcium deficiency symptom even though adequate calcium was present. This necrosis in the youngest leaves takes place when seedlings are ready for floral induction. Four L:D cycles (12h light:12h dark) relieved these symptoms and induced tassel formation. Floral primordia, however, were not visible until over a week later when seedlings were transplanted to field conditions. At the lower temperature, 22 C, tassel induction must take place later; six L:D cycles were applied to seedlings beginning the 16th day. At the end of each of the L:D treatment-periods plants were removed to the field to relieve crowding in limited growth chamber space. At maturity, those plants, raised as seedlings in controlled temperatures, were testcrossed to W22/W23 colorless recessives to assess the level of pigmentation of the *R* gene resulting from paramutation by the *R-Ist* allele at the two different temperatures applied to the seedlings in their first two or three weeks of development. The *R* gene used was one of four new accessions from Native Seeds/SEARCH which showed greater responsiveness to temperature than a highly inbred *R* allele long maintained in W22 background. The *R-Ist* allele was chosen because of low pigmentation in the aleurone, making it possible to assess the level of paramutation in the *R* gene by visual inspection of testcross ears. Because of space limitations, only eight seedlings for each different test could be used since crowding becomes an important factor toward the end of the two or three-week period.

In Vol. 65 of MNL the photographs of testcross ears of paramutated *R* genes from 1990 plants whose seedlings were grown at 28 C showed a high level of paramutation; that is, the kernels were nearly colorless. These highly paramutated *R* genes, when tested for heritability, were found to remain considerably lighter (left column of Fig. 1) than *R* genes from plants whose seedlings received the 22 C conditions for their first 21 days (right column of Fig. 1). In 1991, an effort was made to define a shorter developmental time interval when paramutation could be experimentally controlled. Fig. 2 shows that seedlings which received 32 C and constant light for their first 15 days have paramutant *R* expressions, when testcrossed at maturity, approaching the colorless condition as reported for the previous year. Sib seedlings grown at 22 C through 21 days, with L:D treatments for days 16-21, showed testcrosses whose *R* gene pigmentation was much more darkly mottled (Fig. 3).

Plants grown as seedlings at 22 C (Fig. 3) for the three week period showed a tassel mosaic when testcrosses from a single tassel were compared over the seven day period that pollen from a single plant is shed. The two columns of testcross ears in Figures 2 and 3 represent the first and last pollen collections from each plant. Only the first pollen collections from seedlings which received 22 C show paramutant *R* expressions in testcrosses visibly lighter than those made later in the seven-day period (Fig. 3). A total of 25 plants of the 29 plants tested which received four (days 16-19) or six (days 16-21) L:D treatments at 22 C show the tassel mosaic when earliest pollinations from a single tassel are compared with the latest pollinations.

A high level of paramutation was achieved if seedlings were held at 32 C through the 15th day. By comparison, plants maturing from seedlings which received the lower temperature of 22 C show less paramutation has taken place. These two opposing degrees of paramutation can be used as limits for assessing results of further experiments designed to define more closely the developmentally sensitive period when seedlings respond to temperature treatment. Earlier results had shown that tassel determination for inbred W22 required a minimal four-day period of L:D treatments beginning day 10 at temperatures of 25 C. At 22 C tassel determination requires a six-day period of L:D beginning day 16. Figure 4 shows all the testcrosses from four different plants whose seedlings were held in constant light for ten days at 32 C then subjected to a two day L:D period of 32 C before transfer to 22 C. A greater range of variation in paramutation from plant to plant was observed (Fig. 4) than from those seedlings held for the same period in continuous light at 32 C (Fig. 2). A variety of levels of paramutation result if two-day temperature changes are inserted during this induction period regardless of whether plants were started at the lower or higher temperature regime (Fig. 4). It may be inferred that some plants, because of differences in developmental timing, escape the brief temperature interruption and are determined to be already highly paramutated; others, in response to the brief low temperature of 22 C, will retain a low level of paramutation. Three different experiments involving such inserted two-day temperature perturbations at this floral initiation period showed that a sensitive stage in development has been identified when temperature can make a significant input into the level of plant to plant variation for paramutant *R* gene expression. The mosaicism attributed to paramutation can now be considered influenced by temperature at a critical stage of development defined by the period plants are susceptible to floral induction.

Because induction of flowering requires the imposition of a series of dark periods at different seedling ages, depending on the temperature, it was thought the light:dark (L:D) cycles were responsible for control of the level of paramutation. *R* gene expressions from plants which received constant light were lighter than those which received L:D periods. However, with higher temperatures, the greater reduction in the level of *R* expression suggested that temperature may be the more important variable. As

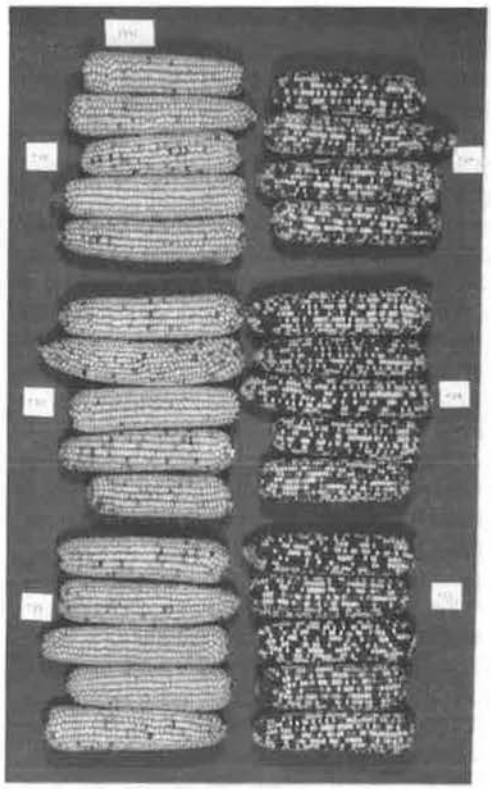


Fig. 1

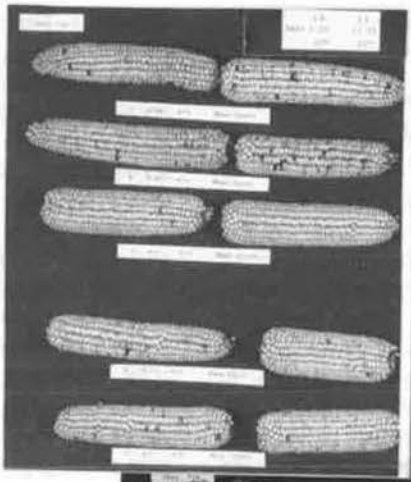


Fig. 2

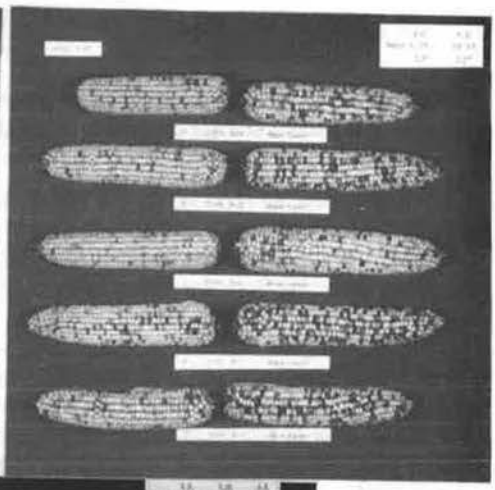


Fig. 3

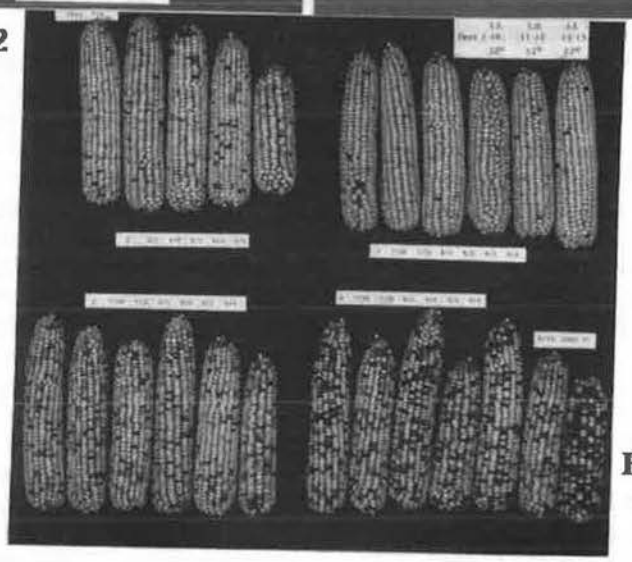


Fig. 4

seedlings grow increasingly closer to the radiant energy source in the closed system of a growth chamber, the application of dark periods results in lower plant leaf surface temperatures. From this it can be inferred that in constant light, temperatures are uniformly higher during the critical induction period hence the higher level of paramutation.

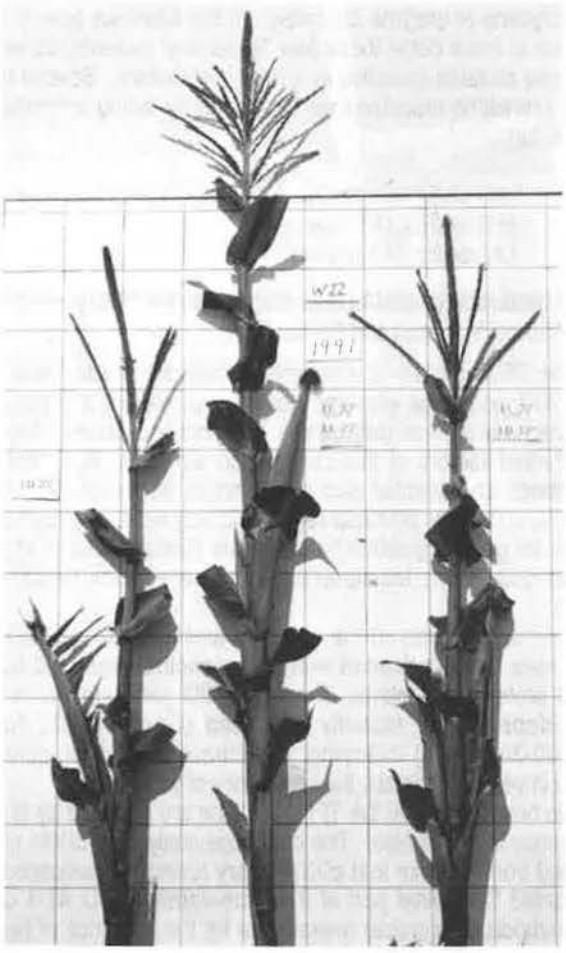
The results above show that near binary control of *R* gene expression under paramutagenic conditions can be achieved by manipulation of temperatures at a critical stage of development. A continuum of expression (heterogeneity) has been reported by the Wisconsin laboratory for the paramutant *R* alleles raised under field conditions. If paramutation can be used as a model, experimental operations describing the behavior of controlling elements in two-element systems under field conditions will require more complex operational description. The work at Wisconsin over the past 40 years, as Brink pointed out in the early 60's, shows that allelic history must be taken into account in descriptions of paramutant *R* behavior. Allelic associations in previous generations and backgrounds can influence future behavior of the *R* gene quantitatively (number of cells in the aleurone layer). Efforts designed to explore the behavior of *R* genes under paramutagenic conditions using inbred backgrounds exclusively, can now be questioned. As we try to understand paramutation can we assume the gene will behave more "appropriately" in an inbred background without some test? What test? Can we be sure the paramutant *R* has not undergone a significant change in its potential behavior in the backcross process? Of the many different alleles available, which would be most useful for a chosen experimental design? In the backcrossing process which phenotypic expressions should be chosen for the continuation of the backcrosses? If temperatures have a significant effect early in development, can we rely on field-grown material in the future, since spring field conditions are notoriously variable from day to day? Another common assumption is that gene expressions from the same tassel are uniform, but the tassel mosaics under temperature control mean that several pollinations must be made on different days to be sure of the range of variation of the *R* gene from a single plant. To make operational description even more complex, the paramutant *R* can add the changes in *R* expression resulting from the presence of each of two paramutagenic alleles (*RR-st R-st* trisomics) in a single generation, or from year to year if exposed successively to paramutagenic alleles. These many questions, though distressing in the light of Mendelian canons, are the necessary prelude to the discovery of experimental operations for dealing with controlling element (transcriptional activator?) behavior at critical developmental stages. The experimental operations and assumptions supporting the Mendelian paradigm can no longer be a reliable guide for systems showing the characteristics of the paramutation-like sys-

tems. Though evidence is still lacking for an element at the *R* locus, both McClintock and Brink considered paramutation an example of a two-element system.

Is paramutation coupled to developmental processes which determine tassel induction?

--Bernard C. Mikula

The L:D treatments above shift the corn plant from the vegetative to the flowering phase of development. Plant physiologists have programmed plant growth and development with day length since the 1920's. Fig. 5 shows the differences in plant habit



(inbred W22) which resulted from the application of the L:D treatments to seedlings. The plant on the left was raised in constant light at 22 C until day 15 then given L:D treatments at 22 C days 16-21. The plant on the right was raised in constant light until day 10 at 32 C then shifted L:D conditions at 32 C from days 11-15. Both plants look essentially alike in habit though the one from the higher temperature was determined six days earlier. The middle plant was grown for 10 days in constant light at 32 C then transferred to 22 C in continuous light from days 11-15. All plants were transplanted to field conditions at the end of their respective treatments. The developmental effects of each of the treatments can be seen in the photographs. The plant on the left had the fewest tassel branches. Larger numbers of tassel branches were found on plants given higher seedling temperatures. Four to five times as many tassel branches were found on the plants which received the higher temperature in continuous light, of which the middle plant is a typical representative. An average of three more nodes accounts for their being taller with anthesis a week later. Along with these morphological changes programmed by temperature and day length, the variation in the level of paramutation discussed above is influenced at this same developmental period.

Paramutation provides a model system where it is possible to follow incremental change in the expression of a single gene across generations. Thus, environmental programming of gene expression becomes an experimental possibility, especially since the paramutant *R* gene has been shown to have an additive memory capability from generation to generation. What molecular transducers respond to temperature and light? Where and how is the incremental memory stored from generation to generation? The answer to these questions can begin to explain how native plants have been able to respond to changing glacial boundaries across continental latitudes and altitudinal climatic boundaries. If a genetic feedback from environmental conditions exists, then it would seem appropriate that it be coupled to the mechanisms associated with control of reproductive physiology known to be entrained by day length conditions.

EUGENE, OREGON
Institute of Molecular Biology

A *Mu*-induced mutation that blocks the stable assembly of the photosystem II complex

--Rodger Voelker and Alice Barkan

hcf134 is one of twenty-seven *hcf* mutants that we have isolated from the *Mu* stocks propagated by the Hake and Freeling groups (see contribution by Barkan et al.). Its phenotype is unique among this group of mutants in that it lacks only the photosystem II (PSII) core complex; other chloroplast proteins accumulate normally. Since there are no nuclear-encoded subunits of the PSII core complex, the *Hcf134* locus appears to play an important role in controlling the expression of the chloroplast genes encoding the missing subunits, or in mediating the assembly of the complex.

We have found that *hcf134* is a recessive mutation. Mutant seedlings are slightly paler than normal, and exhibit small dark-grown sectors that presumably arise as a result of somatic excision of a transposon from the *Hcf134* gene. They are unable to photosynthesize and thus die at the three to four leaf stage, when seed reserves are exhausted.

Western analysis showed that all of the major chloroplast proteins accumulate normally except for the components of the PSII core. The products of the *psb*A, *psb*B, *psb*C, and *psb*D genes accumulate to less than 5% of wild type levels, while the *psb*E product accumulates to approximately 20% of normal levels. The mRNAs encoding these proteins are normal in size and abundance, as revealed on Northern blots. Sucrose gradient fractionation of total leaf lysates demonstrated that these mRNAs are associated with polysomes in a normal fashion, suggesting that the rate of translation is probably unaffected by the mutation. Pulse-labelling experiments in vivo and in isolated chloroplasts confirmed that the missing PSII proteins are in fact synthesized at near wildtype rates. Although the stability of these proteins has not been carefully determined, it appears that they have a half life of about 15 minutes in mutant leaves. These results reveal that the

hcf134 mutation prevents the stable assembly, rather than the synthesis of the PSII core complex.

hcf3 is an EMS-induced mutation that also exhibits the specific loss of the PSII core complex (Leto and Miles, Plant Physiol. 66: 18-24, 1980). However, we have found that *hcf134* and *hcf3* are not allelic. We have used B-A translocations to map *hcf134* to the long arm of chromosome 8.

The exact role of the *hcf134* gene product is unknown. We speculate that it may play a role in the insertion of the PSII components into the thylakoid, in their assembly into a complex, in their post-translational modification, or in prosthetic group biosynthesis.

Studies of transposon-induced nuclear mutations that affect chloroplast gene expression and assembly

--Alice Barkan, Kirsten Munck, Fenella Raymond, and Rodger Voelker

To elucidate mechanisms by which nuclear genes control chloroplast gene expression and assembly, studies are being conducted with a collection of nuclear mutants defective in chloroplast biogenesis. Twenty-seven *hcf* mutants have been recovered from the *Mu* stocks propagated by the Hake and Freeling groups in Berkeley. These mutants are slightly pale green, non-photosynthetic, and (consequently) seedling lethal. At least fifteen of these twenty-seven mutants exhibit the late somatic reversion events typical of *Mu*-induced mutations. Results of Western analyses indicate that ten mutants lack primarily a single thylakoid membrane complex: three lack specifically the chloroplast ATP synthase, one lacks PSII, four lack PSI, and two lack the cytochrome f/b6 complex. With the exception of the PSII mutant (see contribution by Voelker and Barkan), these phenotypes may result from lesions in genes encoding a structural component of the affected complex; we have, therefore, not studied them in detail. The remaining seventeen mutants are more certain to define regulatory or assembly functions since multiple photosynthetic complexes are affected. These mutations may define genes that regulate the expression of sets of nuclear or chloroplast genes, that are involved in protein targeting, or that mediate the assembly of multimeric complexes.

To begin to distinguish between these possibilities, we have analyzed the chloroplast transcripts encoding proteins that fail to accumulate, and the association of these transcripts with polysomes. Results of these studies indicate that four mutants (*hcf129*, *hcf131*, *hcf133*, and *hcf146*) exhibit reduced rates of translation initiation in the chloroplast, in that all plastid mRNAs are associated with abnormally few ribosomes. Nonetheless, the ribosomal RNAs are normal in size and abundance. The genes defined by these mutations are therefore likely to encode components of the chloroplast translation machinery.

Three mutants exhibit clear and unique alterations in chloroplast mRNA metabolism. *hcf136* is defective in the endonucleolytic cleavage event that separates the *pet*B and *pet*D coding regions of the polycistronic primary transcript. No other aberrations in RNA processing have been detected in this mutant. The *pet*B and *pet*D gene products are completely missing in *hcf136*, suggesting that the endonucleolytic cut that fails to occur is essential for generating translatable mRNAs from the primary transcript. *hcf143* contains aberrant transcripts of the *rps12* and *atpF* genes. While both of these genes contain class II introns, other genes containing class II introns give rise to normal transcripts, indicating that the defect does not extend to all class II intron-containing genes. The aberrant transcript patterns suggest that splicing still occurs in these mutants, but splicing intermediates and excised introns may overaccumulate. *hcf142* fails to accumulate the predominant *pet*A transcript. Many other transcripts accumulate normally. *hcf142* therefore has a fairly specific defect in either the transcription or stability of the *pet*A mRNA.

Finally, four mutants that fail to accumulate multiple photosynthetic complexes do not have any detectable defect in chloroplast RNA metabolism or translation (*hcf124*, *hcf137*, *hcf139*, and *hcf140*). These mutants are therefore likely to be defective in a post-translational aspect of chloroplast biogenesis, such as protein targeting or enzyme assembly. In the future we plan to characterize in more detail these four "assembly" mutants, as well as the three mutants defective in mRNA metabolism. Several of the most interesting mutations will be cloned by taking advantage of the *Mu* tag.

FREIBURG, GERMANY
University of Freiburg

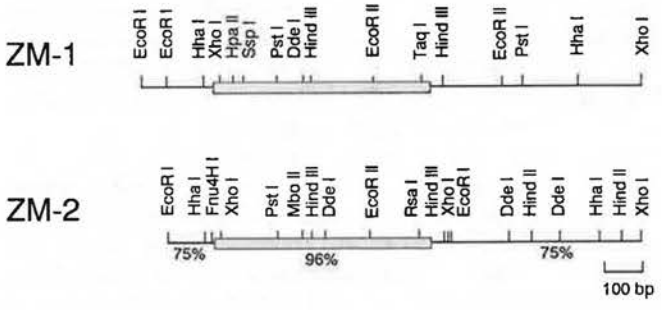
Characterization of cDNAs encoding the maize TFIID proteins

--Michael M. Haass and Günter Feix

The TFIID protein is a general transcription factor binding to the TATA sequence element located upstream of the transcription start site of most polymerase II transcribed genes. Together with further factors of this class, such as TFIIA, B, E and F, it represents an essential part of the active transcription initiation complex at the core promoter region and may serve as a continuous switch for gene regulation by activators (Meisterernst et al., Cell 66:981-993, 1991; Meisterernst and Roeder, Cell 67:557-567, 1991).

Extensive studies of the structure and function of TFIID proteins have been performed with the respective yeast and human TFIID proteins. In plants, the first TFIID gene structures from *Arabidopsis* were recently described (Gasch et al., Nature 346:390-394, 1990) indicating, in contrast to the monogenic situation in yeast or animals, the occurrence of two genes.

We now report that the TFIID proteins are encoded by at least two genes in maize also. The sequence analysis of cDNA clones isolated from a maize leaf cDNA library (using the sequence of a conserved C-terminal part of the *Arabidopsis* TFIID AT-1 cDNA as a hybridisation probe) revealed so far the presence of two different active genes (shown in the figure).



Restriction enzyme maps and schematic alignment of the cDNA clones ZM-1 and ZM-2. The boxed regions represent the ORFs. The percentage numbers indicate the homology between the cDNAs.

The maize cDNAs ZM-1 and ZM-2 display a higher overall DNA sequence homology to each other than do the *Arabidopsis* cDNAs AT-1 and AT-2. In comparison with the *Arabidopsis* TFIID clones they show about 70% homology at the DNA, and about 90% homology at the protein level.

The sequence of the N-terminal 18 amino acids of both maize TFIID proteins (as deduced from the cDNA sequences) differs from the amino acid sequence of the other known TFIID proteins, while the remaining 180 amino acids show a strong structural conservation displaying two direct repeat regions (probably involved in DNA binding), a central domain rich in basic residues (thought to be involved in protein-protein interactions) and a region similar to prokaryotic sigma factors (Hoffmann et al., Nature 346:387-390, 1990).

FREIBURG, GERMANY
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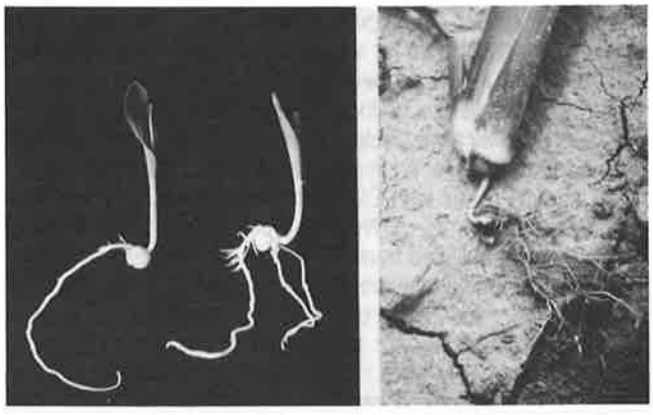
Identification of a mutant deficient in lateral root formation

--Winfried Hetz, Verena Dinger, Michael Schwall, Günter Feix and W. Gerhard Pollmer

In an attempt to generate mutants with a defect in an agronomically important trait, we crossed a flint inbred from the Hohenheim collection of breeding material with an *En* carrying dent inbred (obtained from P. Peterson, Ames). In the segregating F2 generation we recovered, among several other aberrant phenotypes, a plant with a severe deficiency in the formation of lateral roots. Genetic analysis of this plant material indicated that the observed aberrant phenotype is caused by a recessive mutation. The mutant plants (see figure) show a reduced vigour and need special care for their growth, such as intense watering and a supporting stick.



A B C

Photographs of the root system of 12 day old seedlings from the mutant (A) and wt plants (B) and of a 50 day old mutant plant (C).

The isolated mutant is supposed to be a good candidate for the identification of genes with relevance for root formation. We have, therefore, started to investigate whether the mutation is caused by the insertion of an *En* element and would hence be amenable to the isolation of the affected gene by a gene tagging procedure.

GAINESVILLE, FLORIDA
University of Florida

Mapping the restorer gene *Rf3* with RFLPs

--T. L. Kamps and C. D. Chase

Rf3 is the nuclear gene which gametophytically restores fertility to S-type cytoplasmic male sterility (cms-S) of maize. Restoration of male fertility in a gametophytic system is determined by the genotype of the pollen grain. Plants which are heterozygous for *Rf3* are semi-sterile (ca. 50% of the pollen grains are aborted). The classical *Rf3* gene has previously been mapped to 2L by genetic inversion and translocation experiments (Laughnan and Gabay, MNL 50:45-46). Identification of mapped markers closely linked to *Rf3* would more precisely define its position on 2L, facilitate transmission tests of various *Rf3* alleles, and be a significant aid in the design of cloning strategies for this gene.

Polymorphism between the male sterile inbred cms-S W182BN/CA and the restored inbred cms-S Ky21 was detected for 2L RFLP markers *NPI298*, *NPI271*, *NPI456* (Native Plants Inc.), *BNL17.14*, *BNL12.09* (B. Burr, Brookhaven National Laboratory), and *whp1* (U. Wienand, Max-Planck Institut). A BC1 population was generated from the cross cms-S W182BN/CA X (cms-S W182BN/CA X cms-S Ky21). Since only pollen grains with the restoring *Rf3* allele can effect fertilization, all progeny from this cross are expected to be semi-sterile (50% pollen abortion). Southern analysis of 47 semi-sterile BC1 plants was used to determine linkage relationships between RFLP marker loci and *Rf3*. The *Rf3* locus mapped between the proximal *whp1* and distal *BNL17.14* markers. Figure 1 shows the calculated linkage map for all markers tested.

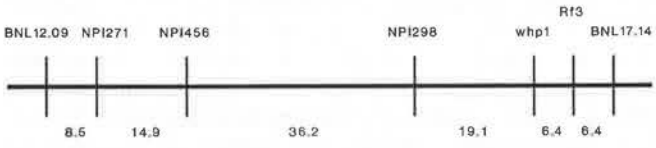


Figure 1. Map of 2L showing genetic position of the *Rf3* locus relative to RFLP markers. The recombination percentages (presented as distances in cM) were calculated with data derived from 47 BC1 plants.

Dormant, colorless alleles of *vp1*

--Christian Carson and Donald McCarty

Null mutant alleles of *vp1* produce viviparous, anthocyanin deficient kernels. The structure and expression of dormant *vp1* alleles was determined and a summary is presented in the table. Leaky expression is exhibited by the *vp1-mum3* allele; the level of anthocyanin expression is comparable to the apparent level of vivipary. Alternatively, full expression of the dormant, colorless alleles effectively promotes maturation, but not anthocyanins.

The maturation-related genes, *Glb1* and *Em*, and the anthocyanin regulatory gene, *C1*, are activated by *Vp1*. Expression of *Glb1* and *Em* mRNA is relatively normal in *vp1-McWhirter* and *vp1-c821708* embryos; and consistent with the dormant, colorless phenotype, *C1* expression is undetectable.

vp1-McWhirter mRNA is 3' truncated and the cDNA sequence encodes a C-terminal truncated protein. *vp1-Mc* protein, immunoprecipitated from in vitro translation and resolved on SDS-PAGE, is proportionately smaller than wildtype. In fact, all of the

	Phenotype				vp1 expression	
	kernels	*Gb1* *	*Em* *	*C1* *	mRNA (bases)	Protein (~M in kDa)
WT (W22)	d/p	+	+	+	2500	90/92
1 *vp1-mum3*	leaky	+	+	+	2500	90/92
2 *vp1-McWhirter*	d/c	+	+	-	2300	69/74
3 *vp1-c821708*	d/c	+	+	-	2600	66/68
4 *vp1-1695*	d/c				2600/4000	61/62/65/67
5 *vp1-a1*	d/c				3000/6000	65/68/75
6 *vp1*-Ref. (null)	v/c	-	-	-		none

d=dormant, v=vivipary, p=anthocyanin pigmented, c=colorless; leaky indicates variable none to weak vivipary and anthocyanins; + and - indicate qualitative expression detected on Northern blot; * probes for Northern blots: *Gb1*-A. Kriz, *Em*-R. Quatrano (from wheat), *C1*-K. Cone; allele donations: 1-P. Stinard, 2-H. Dooner, 3-P. Peterson, 4-M. Alleman, 5-E. Patterson; blanks are undetermined

dormant, colorless alleles encode similarly sized, apparently truncated protein. In contrast, *vp1-mum3* expresses normal sized message and protein.

We have compared *vp1-Mc* with wildtype for activation of both the *Em* and *C1* promoters during transient expression in electroporated maize protoplasts. The result is again consistent with the phenotype. In summary, *vp1-Mc* activates *Em*-GUS expression 10-20 fold, about 5% of wildtype. *C1*-GUS is activated 6 fold by wildtype, but it is not activated by *vp1-Mc*.

We conclude that the *Vp1* gene is composed of at least two distinct functional domains. The N-terminal portion of the protein, which is encoded mostly by the first exon, is sufficient in activating maturation and the *Em* promoter, while the C-terminal structure is required additionally for the activation of *C1*.

A maize invertase clone

--Karen E. Koch, Jian Xu and Donald R. McCarty

Invertase catalyzes one of two enzymatic reactions that can cleave sucrose (sucrose + H_2O → glucose + fructose). The other is mediated by the reversible sucrose synthase reaction (sucrose + UDP ↔ UDP-glucose + fructose). Sucrose breakdown is essential to growth and development of kernels and other non-photosynthetic tissues which survive on sucrose imported from phloem. Both invertase and sucrose synthase are considered critical to sucrose import, although their respective significance varies depending on the tissue and stage of development. In kernels, for example, invertase is very active in the pedicel area throughout development, where it hydrolyzes much, if not most, of the sucrose enroute into the growing grain. Invertase is also active in maize root tissues where its function may be related to sucrose import/retrieval, symbiotic relationships, or osmotic adjustment.

A cDNA clone was obtained from a maize root tip cDNA library (Clonetech, Palo Alto, CA) by probing with a tomato invertase clone from Ellen Klan and Alan Bennett (UC-Davis) (Klan and Bennett, Plant Physiol., in press). Partial sequence of the 1.2kb maize clone showed 85% homology at the amino acid level to the tomato invertase, 60% to that of carrot root, and 45% to yeast invertase. The high degree of homology to the tomato gene and the presence of other highly conserved residues in the deduced amino acid sequence suggest strongly that this clone encodes a plant invertase. Eleven other clones ranging from 1.2 to 2.5kb were subsequently isolated from the same library through homology to the initial 1.2kb maize clone.

Northern blot hybridizations to root tip RNA showed a broad band at ca. 2.5kb, corresponding to the length of the longest homologous cDNA clone. The size of this mRNA suggests a protein with a sizeable extension relative to other plant invertases. Its structure and function will be of interest.

The existence of two closely related, unlinked invertase genes in maize was indicated by Southern blot analysis of DNA from two parent lines and F2 progeny (independent assortment of two strongly hybridizing bands was evident). It is not yet known whether both genes are expressed.

GAINESVILLE, FLORIDA
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EMS-induced waxy alleles: treatment of pollen versus seed

--Ron J. Okagaki, Melanie Trull and Susan R. Wessler

The chemical mutagen ethylmethane sulfonate, EMS, is known to produce single base substitutions in a two step process. The O-6 position of guanine is first ethylated; then during DNA replication the ethylated nucleotide pairs with thymine. However, the characterization of EMS-induced mutations at the waxy (*wx*) locus indicates that EMS produces other lesions (Genetics 128:425-431, 1991). Large structural alterations were found in or near two EMS-induced *wx* alleles, *wx-1240* and *wx-1050*, by Southern blot analysis. Cloning of *wx-1240* revealed a small deletion within the gene and at least 3 additional changes upstream of the locus. Current work is focused on identifying the lesions in *wx-1050* and other EMS-induced alleles.

Genetic fine structure mapping is being employed to determine the location of the mutation within *wx-1050*. *wx-1050* was crossed with the seven *wx* alleles used to map the lesion in *wx-1240*. Pollen from the heterozygous double mutant plants has been collected, stained with iodine, and examined. Two plants from each cross were sampled, and at least 100,000 pollen grains per plant were classified. It was difficult to score the pollen, because the *wx-1050* allele is slightly leaky. Nevertheless, recombinant, *Wx*, pollen was found in heterozygotes between *wx-1050* and all *wx* alleles except *wx-M* and possibly *wx-Stonor*. This places the genetic lesion responsible for *wx-1050* in a region on the physical map bounded by *wx-B1* and *wx-Stonor*. This region will be cloned from *wx-1050* and its progenitor allele by PCR and sequenced.

EMS-induced *wx* alleles isolated by E. Amano have been characterized by Southern blot analysis. These alleles were induced by treating mature seeds with EMS. In contrast *wx-1240* and *wx-1050*, obtained from M. G. Neuffer, were generated by treating pollen. DNA was isolated from the progenitor *Wx* allele and eight mutant alleles. Samples were digested with *Sal*I, fractionated in 1% agarose gels, and blotted onto GeneScreen Plus. Filters were sequentially probed with four subclones that cover the *Wx* locus. Seven of the *wx* alleles were indistinguishable from the progenitor allele. The remaining allele had a single change that could be accounted for by the loss of a *Sal*I restriction enzyme site. Analysis of these eight alleles suggests that point mutations were produced by EMS in this collection of alleles. Genetic fine structure mapping followed by PCR and sequencing will be used to identify the lesions in several of these alleles.

The interesting observation from this work is the difference between treating mature seeds with EMS and treating pollen. A trivial explanation for this result would be that *wx-1240* and *wx-1050* represent unusual events; this will be resolved by examining more alleles induced by pollen treatment. Additional mutant alleles

obtained that were produced in this manner include a *wx* allele from M. G. Neuffer, several *c2* alleles from M. G. Neuffer and E. Coe, and five *R-catspaw* alleles from J. Kermicle. The second possibility is that pollen treatment produces complex lesions at a high frequency. This possibility deserves consideration because DNA replication, normally a necessary step for EMS mutagenesis, is delayed in pollen until after fertilization. This may give DNA repair mechanisms sufficient time to replace alkylated bases. Therefore, these mutations would be produced by another mechanism perhaps through cleavage of the DNA backbone. It is also possible that the DNA in pollen may be particularly susceptible to damage by EMS. If either of the latter two possibilities is correct, then characterizing pollen derived alleles may describe a spectrum of EMS-induced lesions produced by unanticipated mechanisms.

GRAND FORKS, NORTH DAKOTA
University of North Dakota

Isolation of meiotic mutations from active Mutator stocks

--Inna Golubovskaya and William F. Sheridan

During the summer of 1991 a search was conducted for new meiotic mutations by screening of active Mutator stocks. A total of 817 families of 20 kernels each were grown from self-pollinated ears produced on plants of known Mutator activity. The approximately 16,000 progeny plants were screened at flowering for male sterile plants. About 80 families were identified wherein one or more plants were male sterile in that their anthers failed to dehisce. The pedigree analysis of these families revealed that some traced to a common Mutator parent, and in total they formed 36 independent groups. Families of 15 kernels each from a self-pollinated ear from each of the 36 groups was planted in the greenhouse in late August and early September. During October and early November, microsporocytes were collected from all of the plants in each of the families and fixed in ethanol:glacial acetic acid (3:1 v/v) for 24 hours and then stored in 70% ethanol in the refrigerator. The microsporocytes were examined microscopically using propionocarmine staining. Examination of meiotic cells from all 36 families resulted in the identification of eight meiotic mutations. These included mutations exhibiting leptotene arrest, ameiotic, desynaptic, polymitotic, and sticky chromosome phenotypes. These materials are being grown in the winter nursery to propagate and test for heritability in preparation for their further characterization.

HARLAN, IOWA
Orsan/Wilson Hybrids, Inc.

Development of cytoplasmic male sterile lines of maize which are homozygous for indeterminate gametophyte (*ig*)

--Bryan Kindiger

Utilizing a homozygous *ig* 3 3 B-3Ld tertiary trisomic maintainer stock in a 'W23 type' background (see MNL 65:64), 9 cytoplasmic male sterile lines have been developed which are homozygous for *ig*.

The male sterile cytoplasms are cms-C, cms-S, cms-SD, cms-CA, cms-ME, cms-Vg, cms-Q, cms-L, cms-MY. All the cytoplasms have been converged (BC6+) into the background of the maintainer.

Each line is 100% male sterile due to both the sterility effects of the cytoplasm and having *ig* in a homozygous condition. Each line also carries the genetic constitution *A1 A2 C1 C2 R-nj* to obtain full expression of the *R-nj* phenotype.

Occurrence of haploids from these stocks is excellent. The frequency of derived androgenetic haploids varies between 1-9% depending on the pollen parent.

Maintenance of these lines is achieved by crossing them with the *ig ig* 3 3 B-3Ld 'maintainer' stock (MNL 65:64).

Seed of these stocks, as well as the maintainer are being supplied to the Maize Genetics Cooperation Stock Center.

IRKUTSK, USSR
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ORF2 of S mitochondrial plasmids potentially encodes a mammalian type protein kinase

--Yury M. Konstantinov, Miroslava V. Derenko, Vladislav P. Ilyin and Vladimir M. Blinov

Mitochondria of cms-S maize contain linear S1 and S2 plasmids of 6.4 and 5.4kbp (Pring et al., Proc. Natl. Acad. Sci. USA 74:2904, 1977). S1 and S2 DNA molecules have proteins covalently attached to their 5' termini that may be involved in replication of these DNAs (Kemble et al., Nucl. Acids Res. 10:8181, 1982). The S plasmids also contain a 1462bp region of nearly perfect homology, which includes the ORF2 sequence (Paillard et al., EMBO J. 4:1125, 1985). We performed a search for possible homology of protein encoded by ORF2 (327 aa) to Protein Sequence Database "SWISSPROT" (release 17) and found that this protein has sequence homology with the alpha-catalytic subunit of bovine and mouse cAMP-dependent protein kinase (EC 2.7.1.37) (Figure). The amino acid sequence of ORF2 also contains two of seven highly conserved subdomains (II and III) being present in eukaryotic protein kinases (Feng et al., FEBS Lett. 1:98, 1991). The existence of DNA-dependent protein kinases, which obviously play a significant role in the regulation of DNA transcription and replication, has been shown recently in mammalian cells (Lees-Miller et al., Mol. Cell. Biol. 10:6472, 1990; Lees-Miller et al., J. Biol.

Figure. Sequence homology of ORF2 proteins with bovine and mouse protein kinase. PK: Bovine and mouse cAMP-dependent protein kinase, alpha-catalytic subunit; ORF2: ORF2 of maize mitochondrial linear plasmids S1 and S2.

Chem. 264:17275, 1989). ORF2 is suggested to encode similar protein kinase regulating genetic functions of S1 and S2 episomes of maize through phosphorylation of appropriate proteins. In this case, one of the possible targets of the protein kinase action may be the terminal proteins attached to 5' ends of these minichromosomes.

Protection of glucose-6-phosphate dehydrogenase from heat denaturation in vitro by maize HSPs

--G. Borovsky and V. Vojnikov

It is known that the maize cell under heat stress synthesizes heat shock proteins (HSP) having a protection function. It is assumed that some HSP can prevent denaturation of proteins thus increasing resistance of the cell to heat. According to this hypothesis we performed the experiments with glucose-6-phosphate dehydrogenase (G6P-DH) in vitro. We aimed to find the effect of plant HSPs on the activity of G6P-DH after heating. The denaturation of G6P-DH was inferred from the decrease of its activity.

Proteins were extracted from 3-d-old etiolated seedlings of maize (Vojnikov et al., Plant Physiol. 33:221-225, 1986). Seedlings were grown at 27 C (CP), or heat shocked during the last 16h at 41 C (SP). We used the whole water soluble fraction of proteins precipitated by 4 volumes of cold acetone. Proteins were solubilised in 0.1M Tris-HCl buffer and were frozen in liquid nitrogen in small portions. Each portion was used immediately after melting. Bovine serum albumin (BSA) was prepared by the same procedure. G6P-DH activity was measured according to Kornberg et al. (Methods Enzymology, NY, 1955) using test-combination (Boehringer Mannheim). G6P-DH (Serva) was used in 0.000125mg/ml concentration and prepared immediately before the experiment. Heating was carried out in buffer from test-combination. G6P-DH solution was incubated for 10 min with (or without) maize proteins or BSA at 25, 35, 45 C. Then the temperature of solution was rapidly (1 min) reduced to 25 C and the activity of G6P-DH was measured. Statistical calculations were made by standard methods. The activity of G6P-DH after heating was determined in percentage from the activity at 25 C. The activity of proteins from maize was subtracted from the total. It was less than 8% of the total activity under maximal concentration (100mg/ml) of maize proteins in the mixture.

No decrease of the activity of G6P-DH and no effect of CP, SP and BSA on this activity were found after 10 min incubation at 25 C with the particular concentration of plant proteins (25mg/ml). After incubation at 35 C without any proteins, the activity of G6P-DH was 69%; after incubation with BSA - 92%; and after incubation with CP or SP - 103-104%. Following incubation at 45 C without any other proteins, the activity of G6P-DH was 10%; after incubation with BSA - 24%; with CP - 57%; with SP - 68% (Fig. 1). Using different amounts of maize proteins and BSA the percent of protected activity of G6P-DH is found to increase to 50% with increasing added protein concentration to 12.5mg/ml, and to almost 100% of the protected activity with increasing added protein concentration to 100mg/ml after incubation at 45 C (Fig. 2). The activity of G6P-DH after incubation with SP was higher than with CP by 10-12% at all concentrations except for the activity of G6P-DH close to 0 or 100% (Fig. 2).

A factor has been found among maize proteins which protects the activity of G6P-DH in vitro at high temperatures. This factor

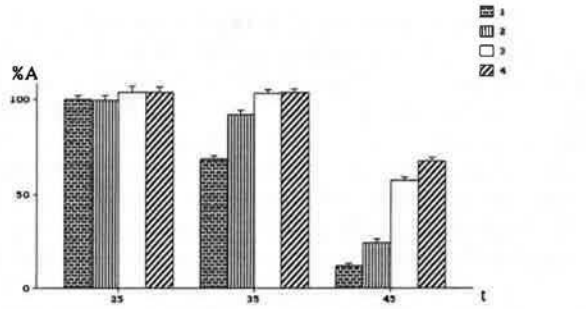

Figure 1. Activity of glucose-6-phosphate dehydrogenase after heating at different temperatures. The standard deviation is shown. % A - activity after heating; t - temperature; 1 - G6P-DH without any other proteins; 2 - G6P-DH + BSA; 3 - G6P-DH + CP; 4 - G6P-DH + SP.

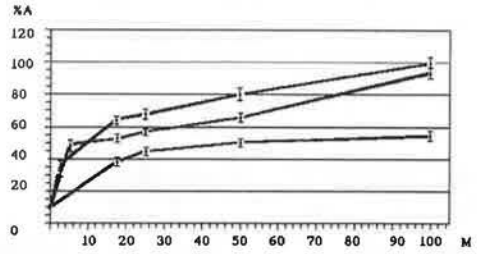

Figure 2. Dependence of activity of glucose-6-phosphate dehydrogenase after heating on concentration of added proteins. The standard deviation is shown. % A - activity after heating; M - amount of added proteins (mg/ml); upper curve - G6P + SP; medium curve - G6P-DH + CP; low curve - G6P-DH + BSA.

is assumed to be heat shock proteins (HSPs) and heat shock related proteins (HSRPs). The action of CP and SP is similar and the differences are probably related to the content of active proteins in total fraction or to the ability of the proteins with similar functions to effect protection. The synthesis of HSPs and HSRPs is known to be necessary for the cell to be alive at all temperatures (Ang and Georgopoulos, J. Bacteriol. 171:2748-2755, 1989; Werner-Washburne et al., Mol. Cell. Biol. 7:2568-2577, 1987) therefore, we believe that HSPs and HSRPs are present in our fraction of maize proteins.

It appears that some HSPs and HSRPs function on the molecular level as chaperones or molecular conformation designers (Beckmann et al., Science 248:850-857, 1990; Ostermann et al., Nature 341:125-130, 1989). The fraction of proteins precipitated only by high concentrations of ammonium sulfate serves as an anti-denaturation factor affecting proteins in vitro. It is interesting that this fraction was enriched by low molecular weight HSPs (Jinn et al., Plant Cell Physiol. 30:463-471, 1989). These facts are consistent with our hypothesis that HSPs play the main role in thermoprotection in our experiments.

The mechanisms of stabilization of proteins are unknown. On the basis of the main principles of protein denaturation (Jaenicke, Prog. Biophys. Molec. Biol. 49:117-237, 1987) we assumed that the anti-denaturation factor is connected with macromolecule at the points important for its flexibility, thus increasing the rigidity and thermostability of the molecule.

ITHACA, NEW YORK
Cornell University

A variant of C-type cytoplasmic male sterility with altered fertility restoration patterns

-- M. E. Smith and E. D. Earle

Among plants regenerated from callus cultures of the inbred

W182BN, a male sterile individual was identified. This plant was pollinated by W182BN in the normal cytoplasm background, and the resulting progeny were all fully male sterile, indicating that the plant was a cytoplasmic male sterile (cms). Subsequent crosses with known differential restorers suggested that the cytoplasm was of the C type, since it was restored to fertility when pollinated with Pa884P (a known cms-C restorer), but not when pollinated with A636, A664 or A665 (known cms-S restorers) or with NYD410 (a known cms-T restorer). In addition, progeny of this plant showed no sensitivity to *Bipolaris maydis* race T toxin, which is diagnostic for the presence of cms-T. Mitochondrial DNA analysis (carried out by D. Pring's lab at the University of Florida) showed no detectable differences between the new cytoplasm and standard C cytoplasm. The new cytoplasm has been designated CR.

Additional crosses of numerous inbred lines onto CR and two other cms-C types of cytoplasm (C and PR) were made, and fertility restoration evaluated on the F1 progeny of these crosses in either Aurora, NY or Homestead, FL. Results of the fertility restoration data (Table 1) suggest that CR varies somewhat from both C and PR cytoplasms in its fertility restoration behavior. Progeny of crosses with A632Ht, A634Ht, A635, B89, CB59G, LH145Ht, LH148, Oh51A and Va20 were more sterile in the CR cytoplasm background than in the C cytoplasm background, while the reverse was true for progeny of crosses with Ay499, CrS4HLa, FR22 and Mo17. Fewer combinations with PR cytoplasm were available, but differences in fertility restoration between CR and PR were detected. Progeny of crosses with A634Ht were more sterile in CR than PR cytoplasm, while those from CrS4HLa, Mo17 and Oh51A were more fertile in CR than PR cytoplasm.

Table 1. Male fertility ratings* of F1 crosses between different inbreds and W182BN-CR, W182BN-C and W182BN-PR. Ratings made in Aurora, NY or Homestead, FL (indicated by †).

	Fertility ratings when crossed with:		
Male parent	W812BN-CR	W182BN-C	W182BN-PR
A619	5	5	5
A632Ht	1	5	1
A634Ht	1	5	1 + 1-3
A635†	1	4	-
A641Ht†	1	1	-
Ay303E	1-3	4	1-3
Ay499	5	1 + 1-3	5
B8	1-4	4	1-3
B73†	1	1	-
B89†	1	3	-
CB59G†	1 + 4	4	-
CO192	5	4	5
CO220	5	5	5
CrS4HLa	5	1	1-3 + 5
FR22	5	1-3	4
LH59†	1	1	-
LH82†	1-4	1-4	-
LH85†	1	1	-
LH132†	1	1	-
LH145Ht†	1-3	4	-
LH146†	1-3	1-3	-
LH148†	1 + 1-3	3	-
LH149†	5	5	-
Mo17	1 + 1-3	1	1
Oh51A	1 + 1-3	1-3 + 3	1
Pa884P	5	5	5
Va20	1	5	1
W64A	5	4	5
W821BN	1	1	1

* Fertility ratings: 1=male sterile, 2=sterile anthers exserted, 3=partially fertile, 4=slightly subnormal, 5=fertile; 1-3=late breaking partial male fertility; 1-4=late breaking to slightly subnormal male fertility; + indicates segregation among the progeny for the two ratings indicated.

The plant initially identified in the field as carrying the CR cytoplasm was recorded as being derived from a normal cytoplasm culture of W182BN. Recent evidence that mitochondrial populations within a cell may be heterogeneous suggests a possible mechanism by which cms-C plants might be derived from a normal cytoplasm culture. At the time the plant carrying CR cytoplasm was regenerated, there were both normal and cms-C cultures of W182BN in the laboratory. Thus it is also possible that a labelling error occurred, and that this plant actually was derived from a culture of cms-C W182BN.

Regardless of the exact origin of this plant, it appears to differ in fertility restoration behavior from cms-C plants of W182BN from the C and PR types. Although male fertility, particularly for cms-C, varies with environment and hence might be different in Aurora and Homestead, each set of F1s with the same male parent was evaluated in one or the other environment. Thus differences in ratings between CR and the other cms-C types for a given male parent cannot be explained by environmental influence alone. Genetic differences and/or differences in genotype-by-environment interaction among the cytoplasms are indicated. Given these differences, CR may prove to be a useful source of additional cytoplasmic diversity within the cms-C group.

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Linkage between *Ms44* and *C2*

--Marc C. Albertsen and Mary R. Trimnell

We reported earlier that *Ms41* and *Ms44*, both located on chromosome 4L, were determined to be non-allelic and separated by 14±2cM (Albertsen and Neuffer, MNL 64:52). We have subsequently obtained linkage of *Ms44* with *C2*. We crossed *Ms44 C2* with a *c2* tester stock, *ms44 c2*. Male-sterile F1 plants, *Ms44 C2/ms44 c2* were backcrossed to the *c2* tester.

Progeny segregated 1:1 for purple and yellow kernels. Purple kernels were separated from yellow kernels, planted and classified for sterility. The following was obtained:

	Purple k's		Yellow k's	
	# sterile plants	# fertile plants	# sterile plants	# fertile plants
Ear row #1	17	0	0	16
#2	20	0	0	15
#3	18	0	1	17
#4	18	0	0	20
#5	20	0	0	19
#6	19	0	0	20
Totals	112	0	1	107

These results indicate that the *Ms44* locus is located virtually at the *C2* locus. We are repeating the grow-out to determine if the 1 sterile plant from the yellow kernel class was a fluke or a true recombinant. Dr. Steve Briggs suggested using *C2-Idf* to independently verify these results. The initial crosses will be accomplished in our 1991 winter nursery.

A new leaf mutant - narrow sheath (*ns1*)

--Evan Elsing and Marc C. Albertsen

A leaf-blade and plant-stature mutant (narrow sheath, *ns1*) was observed in one of our nurseries planted on Kauai, Hawaii, in

November 1990. This mutant is characterized by a brachytic-like plant with abnormal leaf-sheath and leaf-blade development. The leaf sheath tapers from its base, through the ligule, and into the leaf blade. Between the ligule and the mid-length of the leaf, the leaf may only be as wide as the midrib. The leaf blade gradually widens near mid-length of the leaf. Although the leaf never attains the width of leaves on normal sibs, it does attain a normal-like appearance. The mutant phenotype also is demonstrated in the husk leaves. As the husk leaves are equivalent to leaf sheaths, they also display width reduction. Ears that formed were exposed on the two lateral sides of the ear. Mutant plants are 30-60% shorter than normal sibs, having very short internodes. Although they appear brachytic-like, there was no allelism to *br2* (data not shown). We also made test crosses with *lg1* and *lg2*, even though we visually verified that the ligule was present. Our results (data not shown) showed no allelism.

The mutant is true breeding and is mostly controlled by a single recessive gene. Supporting data are given in Tables 1-5. Remarkably, this mutant emerged independently from two Pioneer Hi-Bred corn breeding stations at Winterville (WT), North Carolina, and Tifton (TF), Georgia, utilizing Pioneer proprietary lines. One mutant came from an F4; the other from an F6. There were no immediate parents in common, however, one grandparent was in common for three of the four inbreds involved in the original crosses. The independent occurrences of this mutant were discovered serendipitously. A source of pollen was needed to attempt a pollination on one of the mutant plants (*ns1-WT*). Pollen was obtained from a row from Tifton. We had expected to find all normal progeny from this pollination. Instead we observed a 1:1 segregation of mutant and normal plants (data not shown). When remnant seed was requested from Tifton and planted in a subsequent nursery, *ns1* plants segregated. Evidently the plant selected for pollination in November 1990 was heterozygous for *ns1*. We will therefore, refer to the Winterville mutant as *ns1-WT* and the Tifton mutant as *ns1-TF*. There is no reason, however, to believe that these mutant alleles are different from each other.

As shown in Tables 1-5, *ns1* segregates as a single recessive gene. We have some segregations, however, in which the mutant class is under-represented (e.g. Table 6). We will be growing larger numbers of progenies and testing across more genotypes to determine the true nature of *ns1* expression.

Table 1. True breeding results from *ns1-WT* self-pollination.

	# mutants	# normal	Total
Self #1	8	0	8
Self #2	9	0	9
	17	0	17

Table 2. Self of mutants from *ns1-WT* x *ns1-TF* crosses.

	# mutants	# normal	Total
Self #1	9	0	9
Self #2	5	0	5
	14	0	14

Table 3. Single recessive gene segregation from mutant plants of *ns1-WT* x *+/ns1-TF* crossed with normal sibs.

	# mutants	# normal	Total
Sib #1	9	9	18
Sib #2	10	10	20
	19	19	38

Table 4. *ns1-WT* sibbed w/ normal plants from a selfed heterozygote.

	# mutants	# normal	Total
Sib #1	0	20	20
Sib #2	8	9	17
Sib #3	9	10	19
Sib #4	4	15	19
Sib #5	0	20	20
Sib #6	2	9	11

-fits expected 1:2 segregation of a selfed heterozygote
-segregating progeny = 23 mutants:43 normals
$x^2 = 3.41$
$.10 > P > .05$
$\therefore$ fits a 3:1 segregation ratio

Table 5. Selfed progeny segregation from *ns1-WT* crossed with Pioneer inbred J65.

	# mutants	# normal	Total
Self #1	4	15	19
Self #2	3	16	19
Self #3	1	17	18
Self #4	4	14	18
Self #5	3	15	18
Self #6	4	15	19
	19	92	111

$x^2 = 3.68$
$.10 > P > .05$
$\therefore$ fits a 3:1 segregation ratio

Table 6. Selfed progeny segregations from *ns1-WT* crossed with Pioneer inbreds (G83 x W88-2).

	# mutants	# normal	Total
Self #1	1	19	20
Self #2	4	15	19
Self #3	2	17	19
Self #4	2	15	17
Self #5	3	17	20
Self #6	3	17	20
Self #7	2	18	20
	17	118	135

$x^2 = 11.08^{**}$
** significant at 1% level
$\therefore$ does not fit a 3:1 segregation ratio

We consulted several maize geneticists concerning the naming of this mutant. Suggestions included vestigial leaf, bladeless, narrow leaf, and narrow sheath. We finally decided that narrow sheath was the most descriptive, as it implies that the lower portion of the leaf also would be narrow. Our search of the literature did not find any previous descriptions of this type of mutant.

A suppressor of floral leaf development

--Steve Briggs

A recessive mutation, designated tassel-sheath (*tsh*-50330*), has been recovered from Mutator stocks. The mutation causes leaves to develop at the base of each tassel branch. The mutant plants are recognized in the field by an unbranched tassel appearance. The phenotype results from leaves that wrap around the tassel to form a sheath which encloses the basal 1/3 to 2/3 of the tassel. These tassel leaves produce no ligules or blades. Branching of the tassel is normal. Closer inspection reveals that leaves often develop at the base of tassel spikelet pairs on the rachis. These leaves also lack ligules and blades. Homologous structures are produced at the base of each cupule in the ear of mutant plants. Each ear spikelet leaf spans both spikelets in the cupule and is outside (basal to) the glumes. The ear spikelet leaves are not pigmented on *P-WR* cobs but turn purple on plants that carry *B* and the associated anthocyanin genes. The ear spikelet leaves become indurate as the ear matures. Maize may prevent the development of leaves in the inflorescence by organ-specific expression of a dominant suppressor of leaf development. The *tsh*-50330* allele ap-

pears to be a knock-out of such a suppressor.

A recessive barren-inflorescence mutation

--Steve Briggs & Guri Johal

A mutation that resembles *Bif1* has been observed in our Mutator stocks. In contrast to *Bif1*, our mutant (*bif*-47330*) appears to be fully recessive; 18 of 75 plants in an F2 family displayed the mutant phenotype. Mutants are easily spotted in the field because the tassel has few or no branches and is unusually thin, with the thinness beginning at the first node that fails to produce a leaf. The principal effect of the mutation may be to limit spikelet development. Failure to produce spikelets results in a bare or hairy ridge on the cob where a cupule would normally be. The size of these sterile sectors varies from one cupule to nearly the entire ear. The tassel is similarly modified. Tassel spikelets are typically unpaired, with only the pedicillate spikelet developing (occasionally a vestigial or normal sessile spikelet is observed). The tassel spikelet pedicels are much elongated (to about 1cm) and the spikelets themselves are about twice normal size. The florets appear normal but shed poorly or fail to shed. Occasional ears are found that show an unpaired phenotype, either as a sector or as the entire ear. Possibly the sessile spikelet in the ear is suppressed congruent with the tassel phenotype. These observations suggest that the mutation may reduce the amount of a factor that is required for the development of spikelets, with the sessile spikelet being more sensitive to this reduction than is the pedicillate spikelet.

Defenseless mutants

--Gurmukh S. Johal and Steven P. Briggs

There is no biochemical explanation for the nature of disease defense mechanisms in maize. Despite considerable research efforts, no phytoalexins have been identified. An ideal way to study this phenomenon would be with a mutant(s) in which the plant's defense is impaired. However, no such mutants have ever been isolated. The main problems are of identification and maintenance, since such mutants will be lethal and be eliminated. To our knowledge, no one has ever searched for these mutants in a systematic way.

We have now devised a genetic strategy to generate and identify such recessive mutants in maize. This approach relies on transposon mutagenesis using Robertson's Mutator. Briefly, maize plants containing Mutator are crossed with plants from an inbred line. The resulting F1 progeny are then selfed to generate F2 families. Any recessive mutation in the F1, due to insertional inactivation by *Mu*, will be expressed in the F2 and approximately 1/4th of the plants will show the mutant phenotype.

The next question is how to uncover mutations in the defense pathway. For this, we have developed a dual screening procedure. First, 48 seeds from each F2 family are planted in the field. The families in which about 1/4th of the plants fail to germinate, die as seedlings, or show root rot symptoms, are replanted in greenhouse under partially sterile conditions. Those families which now germinate and grow normally may have biochemical lesions in the defense pathway. Such families are then subjected to confirming tests.

So far, we have generated about 20,000 F2 families. Last summer, we planted about 10,000 of these in the field to identify those which were deficient in germination, emergence or plant stand. We were unable to make this selection because of constant rain and flooding shortly after planting which affected the germination of the maize crop as a whole. Out of about 46 families that were screened in the greenhouse, one segregated for plants which succumbed to a root rot disease(s) at different times after germination. These results were confirmed when 32 seeds each were planted in both the sterile and nonsterile soil-mix. Five out of 30 plants died in the nonsterile soil-mix, whereas only one out of 32 died in the sterile mix. A soil-borne root rot fungus, *Fusarium*, has been identified growing on the dead plants. Further characterization of this mutant is in progress.

The isolation, characterization, and cloning of Mutator-tagged dwarfing genes

--Robert Bensen, Guri Johal, Pat Schnable and Steve Briggs

Several andromonecious dwarfs have been isolated from Mutator lines. Allelism tests for these mutants are underway, as are tests monitoring their growth response to applied gibberellic acid (GA). One of the mutations (*an1-891339*) which causes a GA-responsive phenotype, is allelic to anther ear (*an1*). Co-segregation of *an1-891339* and a genomic digest fragment (5.7kb) containing *Mu1* has been observed. This 5.7kb fragment has been cloned. Further, a subclone (2.6kb) of this fragment which contains only flanking sequence has been isolated. The *an1* gene is flanked on either side by *id1* and *bz2* (Neuffer and Chao, MNL 65:52). Evidence supporting the prediction that the 2.6kb subclone is in or near the *an1* gene includes its absence in the *an1-6923* deletion mutant (MNL 65:130). Interestingly, the 2.6kb subclone does hybridize to DNA from the *idd*-2286A* deletion mutant (MNL 65:52). The 2.6kb subclone has been used to select maize genomic clones from a lambda-library. These genomic clones are currently being characterized.

Cloning and characterization of two nuclear genes for maize mitochondrial chaperonin cpn60

--P.S.Close[a,b], S.P. Briggs[b], B.A. Bowen[a,b], T.K. Prasad[a] and R.G. Hallberg[a,c]

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Cpn60 (hsp60) is an abundant mitochondrial protein first described in *Saccharomyces cerevisiae* and *Tetrahymena thermophila* cells (McMullin and Hallberg, Mol. Cell. Biol. 8:371-380, 1988; Reading et al., Nature 337:655-659, 1989). It belongs to a family of proteins homologous to the *E. coli* protein groEL. This family also includes the chloroplast Rubisco-binding protein. These proteins are designated 'chaperonins' because they are implicated in the folding of peptides and the assembly of oligomeric protein complexes. A cpn60 protein has been identified in *Zea mays* and found to be structurally and metabolically indistinguishable from hsp60 in yeast and *Tetrahymena* (Prasad and Hallberg, Plant Mol. Biol. 12:609-618, 1989; Prasad et al., Mol. Cell. Biol. 10:3979-3986, 1990). Protein expression is elevated after heat shock and in developing maize seedlings (Prasad and Hallberg, Plant Mol. Biol. 12:609-618, 1989). A partial cDNA clone was isolated from a lambda gt11 expression library using cpn60 antibody. This clone was used to isolate longer cDNA's.

Genomic Southerns reveal two copies of cpn60 in all maize inbreds screened. One copy is present in the *Arabidopsis* genome. Both maize genes are encoded in the nuclear genome. A maize B73

genomic library has been constructed and screened for cpn60 clones. Two major families (I and II) of overlapping clones have been identified and sequenced. These clones correspond to the two gene copies identified on genomic Southern blots. Both genes contain multiple (17) introns in identical positions. We used RFLP mapping to locate cpn60-1 to the short arm of chromosome 5 and cpn60-2 to the long arm of chromosome 1. One family of cpn60 genomic clones from *Arabidopsis thaliana* has been identified and partially sequenced. Introns are present in the *Arabidopsis* cpn60 genomic clone.

An RNA of approximately 2kb is extremely abundant and developmentally regulated in germinating seedlings. Transcript level is high in seedlings 24 hours post imbibition and decreases progressively to a lower constitutive level as seedlings age. Low levels of transcript were detected in seedling tissue up to two weeks old and mature leaf tissue. Steady state transcript levels increased significantly following heat shock treatment (3 hours, 39 C). We have isolated and sequenced two unique cDNA clones for cpn60 in maize. These correspond to Families I and II of overlapping genomic clones. The two cDNA's are 95% identical within coding regions but diverge significantly in the 3' untranslated regions. Gene specific probes are being utilized to study differential regulation of Families I and II.

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The influence of random mating on recombination among RFLP loci

--W. D. Beavis, M. Lee, D. Grant, A. R. Hallauer, T. Owens, M. Katt and D. Blair

Studies of recombination in maize have historically used backcross (testcross) or F2 progeny from bi-parental crosses of specially crafted genetic stocks. The development of molecular markers has made it possible to develop saturated linkage maps in adapted germplasm using backcross, F2 and RI progeny. Burr et al. (Genetics 118:519, 1988) have advocated the use of RI populations because there are more meioses to produce detectable recombinants among the progeny. This becomes a desirable goal as the number of genetic loci in a linkage group increases. Another approach to producing a larger percentage of detectable recombinants is to randomly mate the population from a bi-parental cross before obtaining progeny (Kempthorne, ISU Press, 1957). An advantage of random mating is that the frequency of detectable recombinants will ultimately approach 0.5 regardless of how tightly linked the loci initially appear. It is also possible to keep linkage at some level less than 0.5 by simply discontinuing random mating at some intermediate generation and deriving either F2 or RI progeny.

We have developed linkage maps using RFLP-loci and F2 progeny from randomly mated and non-randomly mated populations. Both populations were derived from a cross of B73xMO17. The non-randomly mated population, designated syn0, was obtained by selfing the original F1 and consists of 99 progeny. These progeny were further selfed to produce syn0-$F_{2:3}$ lines. The randomly mated population, designated syn5, was derived by sib-mating the original F1 for five cycles, then deriving 98 syn5-F2:3 lines as in the syn0 population. RFLP-typing of these lines was accomplished using standard molecular biology protocols. The frequency of the RFLP alleles and segregation at the loci were analyzed for deviations from expected frequencies based on Mendelian inheritance. The genetic linkage maps were constructed using MAPMAKER (Lander et al., Genomics 1:174, 1987) and a mapping protocol similar to that outlined by Landry et al. (Genome 82:543, 1991).

There was no evidence for segregation distortion at any of the loci in the syn0, but there was evidence of distorted segregation at 5 loci in the syn5 (Table 1). The frequency of the B73 allele at most (24) of the loci in the syn0 was less than 0.5, but only *umc76* was significantly less. Interestingly, the frequency of the B73 allele at most (23) of the loci in the syn5 was greater than 0.5. The frequency of the B73 allele at five loci was greater than 0.60 and at two loci it was less than 0.40. The change in frequency from the syn0 generation to the syn5 generation was almost always toward the B73 allele. However, for *bnl8.10*, *pio200518*, *bnl7.25*, and *bnl8.29*, the change was toward the MO17 allele. These loci were all linked in the syn0 generation (Figure 1). Interestingly, a plant height QTL was identified in this region using data from the syn0 population (data not shown).

Table 1. Results of segregation analyses (Chi-squared statistic) and frequencies of the B73 allele for 32 RFLP-loci in F2 progeny derived from randomly mated (syn5) and nonrandomly mated (syn0) populations of B73xMO17.

	Calculated Chi-squared values		Frequency of B73 allele	
LOCUS	syn0	syn5	syn0	syn5
umc94	4.12	5.06	0.44	0.58
bnl5.62	4.52	2.56	0.42	0.56
pio200537	4.90	7.04	0.46	0.43
pio200603	11.80	6.08	0.43	0.44
pio200689	2.98	0.92	0.47	0.47
pio200640	6.20	7.85	0.42	0.54
umc76	9.50	1.04	0.38	0.52
umc137	2.17	1.41	0.45	0.53
umc11	3.38	2.21	0.44	0.53
umc13	2.90	0.56	0.44	0.53
umc1C12	1.73	24.04	0.45	0.66
bnl7.21	2.25	14.64	0.45	0.62
umc133	2.90	8.71	0.44	0.57
pio200654	3.73	16.51	0.50	0.63
bnl5.59	5.02	8.86	0.43	0.61
pio200575	4.02	1.12	0.43	0.53
pio200674	3.04	2.54	0.44	0.55
pio200644	2.60	3.31	0.46	0.50
pio200855	3.71	0.40	0.45	0.49
umc23	5.04	4.39	0.51	0.54
umc33	3.72	0.25	0.45	0.51
pio200668	3.30	0.06	0.45	0.51
pio200661	1.50	1.05	0.47	0.51
umc128	1.43	3.20	0.46	0.49
umc50	1.38	0.10	0.53	0.49
bnl8.10	0.34	30.75	0.51	0.36
pio200518	2.60	0.91	0.52	0.47
bnl7.25	1.88	4.74	0.52	0.43
bnl8.29	6.50	10.75	0.52	0.39
umc84	0.38	18.61	0.51	0.64
pio200557	3.32	10.68	0.48	0.62
bnl6.32	4.46	8.05	0.48	0.58

The sum of the estimated recombination among adjacent pairs of RFLP loci in the syn0 population was ~ 275. This value is unadjusted for expected double recombinants. Thus, the estimated average recombination was ~ 8.8. A comparison of the linkage maps (Figure 1) revealed that there are four independent linkage groups after 5 cycles of mating. The sum of the estimated recombination among adjacent pairs of RFLP loci in the syn5 population was ~ 730 (unadjusted for expected double recombinants), or

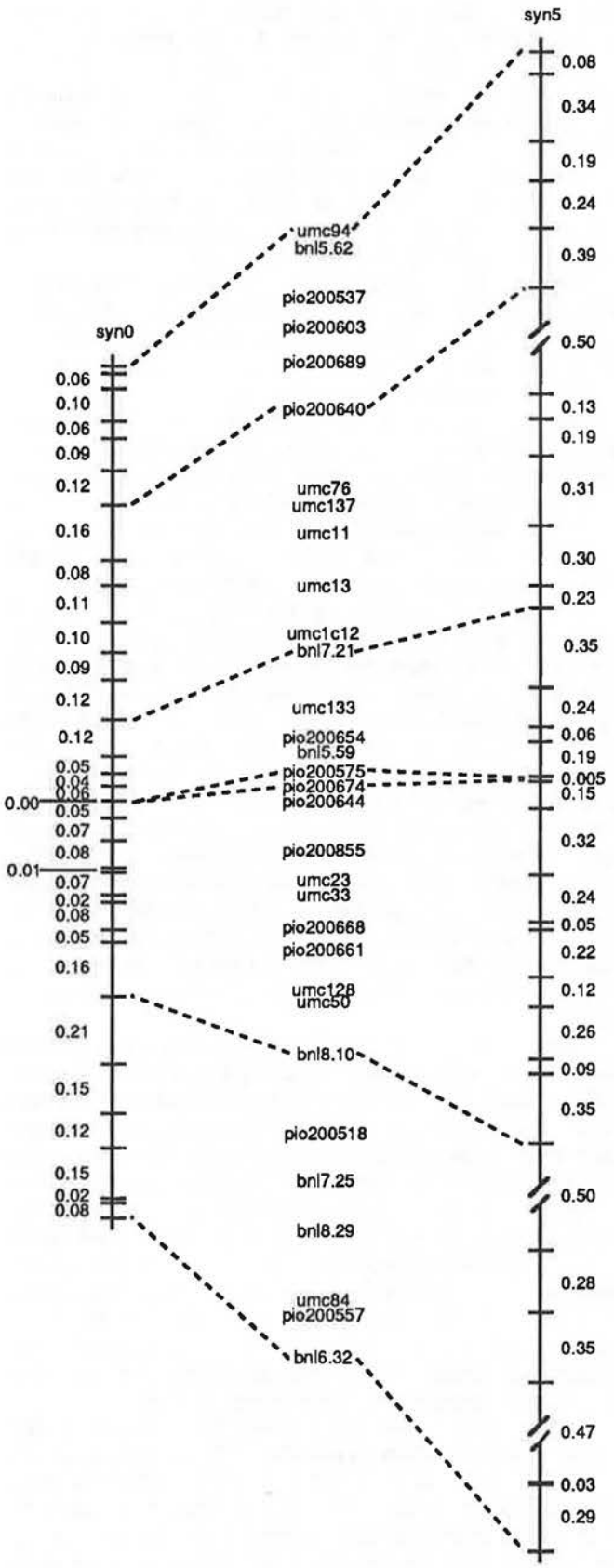

Figure 1. RFLP linkage maps of chromosome 1 based on segregation data from F2 progeny derived from an F1, (syn0) and an F1 randomly mated for five generations (syn5) of the cross B73xMO17.

an average of about 23.5. The relationship of recombination among randomly mated generations can be described

$$r_{(n)} = r_{(n-1)} + [r_{(n-1)} - 2r^2_{(n-1)}]/2, \quad (1)$$

where $r_{(n)}$ is the frequency of recombinants after n generations of random mating and $r_{(n-1)}$ is the frequency of recombinants in the prior generation. Based on the recurrence relationship, (1), and the estimated recombination in syn0, the average frequency of recombinants should have been about 35 in syn5.

The estimated recombination in the syn5 was greater than the syn0 for all pairs of adjacent loci. For many pairs of adjacent loci the observed value for $r_{(5)}$ was about what would be expected based upon the estimate from syn0 and (1). For example, the estimated recombination between *pio200661* and *pio200668* was 1.5% in the syn0. Assuming that this is the true value for this segment, then the expected amount of recombination in the syn5 would be ~10%; we observed 12%. On the other hand, there were several segments where the observed recombination in the syn5 was much less (10-20%) than expected based on the observed values in the syn0 (Figure 1). For example, there was very little increase in the number of recombinants between *umc94* and *bnl5.62*.

One of the primary objectives for generating a randomly mated population is to uncouple tight linkages. In the syn0, we were unable to detect recombinants between *pio200575* and *pio200674*. In two additional B73xMO17 populations, based on data from 112 F2's and 46 RI's, we have not been able to detect recombinants between these loci (data not shown), suggesting very tight linkage. However, in the syn5, we did detect one recombinant between these loci among the 196 sampled gametes. Uncoupling tight linkages also will be useful for QTL studies. Beavis et al. (1989 presentation to ASA, Las Vegas, NV) used simulations to show that randomly mated populations improve resolution of QTL. Patterson et al. (Genetics 124:735, 1990) advocated random mating of isogenic lines as a basis for map-based cloning of introgressed genetic loci. Finally, to be politically correct, we note that the ease with which random mating is accomplished in maize makes it an ideal organism for these types of recombination studies.

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_Wx_ locus in maize plants carrying unstable macromutation _Cg2_

--L. G. Tumanova, N. A. Vikonskaya and N. V. Krivov

Maize plants with unstable mutation *Cg2* show a high mutability at loci unlinked to *Cg2*. We observed genetic instability at the *Wx* locus of hybrid plants obtained by crossing marker line *wx sh* to line *Cg2*+143-206. The latter line carries the *Cg2*+ allele, which systematically mutates to *Cg2*. The proportion of corngrass phenotype plants resulting from such mutation events was 67.6% for the N206 family. Among the hybrid kernels of the F1 generation, the fraction of *Wx* variegated kernels ranged from 3.1% to 32.7% in various families.

Southern blot analysis of DNA from F1 plants was performed. As a *Wx* probe the 2.5kb *Eco*RI fragment of the pcWx16 plasmid containing cDNA of the *Wx* gene was used. The plasmid was kindly provided by A. Gierl. Plant DNA was digested with *Eco*RI. Among the fifteen plants examined, two samples (the 3219/7b and 3227/10a plants) whose hybridization patterns differed from

others were found. The fifteen individuals all contained a 15kb *Eco*RI hybridizing fragment. A 3.6kb *Eco*RI additional fragment hybridizing to the *Wx* probe was observed in the 3219/7b plant and two fragments, 6.6kb and 2.4kb, in the 3227/10a plant (Fig. 1).

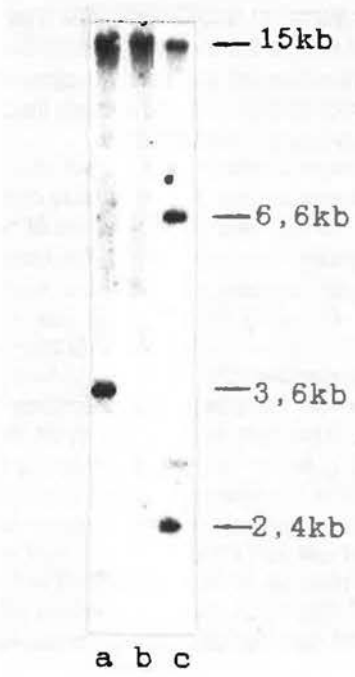

Figure 1. Southern blots on DNA from F1 plants hybridized with a *Wx* cDNA probe. DNA samples were digested with *Eco*RI, electrophoresed on 1% agarose. Lanes: a) DNA sample from the 3219/7b plant; b) DNA sample from the 3227/3a plant; c) DNA sample from the 3227/10a plant.

An ear with 185 kernels was obtained on the selfed 3227/10a plant. The segregation ratio for *Wx* kernels did not significantly deviate from 3:1. There were 119 phenotypically normal kernels, 16 variegated and 50 mutant kernels in the ear. Variegated kernels were counted in the phenotypically normal *Wx* class.

Blot hybridization analysis of DNA of F2 plants grown from variegated kernels showed the same hybridization patterns as did the analysis of DNA from the 3227/10a plant.

Thus, among analyzed hybrids from a *wx sh* X *Cg2*+143-206 cross, two plants carrying the *Wx* gene with altered structure were discovered. The altered *Wx* gene is inherited in at least one of these plants. From the hypothesis about the existence of the *Fpj* mutable system responsible for *Cg2* locus instability, one may suppose that *Wx* variegation is due to the effects of the mentioned system components.

Mutator system-controlled instability of the *Cg2* locus

--N. V. Krivov

[ed. note: this note, received after the 1991 issue was printed, overlaps with the next article but contains supplemental information and photographs.]

Morphogenetic mutants are a most suitable object for gaining insight into regulatory principles of plant development (Sawhney and Greyson, Am. J. Bot. 60:514-522, 1973; Koornneef et al., J. Hered. 74:265-272, 1983). In maize it is expedient to use semidominant mutations (*Tp1*, *Tp2*, *Tp3* and corngrass) to study mechanisms underlying organogenesis of a plant as a whole as well as that of inflorescences (Lindstrom, J. Hered. 16:135-140, 1925; Peterson, MNL 33:41, 1959; Singleton, MNL 23:7, 1949; Sheridan, Annu. Rev. Genet. 22:353-385, 1988). These mutations cause profound atavistic changes in the morphology of vegetative organs as well as transform reproductive structures into leaves (Singleton, Amer. Nat. 85:88-96, 1951; Weatherwax, J. Hered. 20:325-330, 1929). A phenotype like this has been shown to be due to a defective transition from juvenile to mature phase in shoot development (Poethig, Nature 336:82-83, 1988).

Reported here is an unusual behaviour of an unstable *Cg2* macromutation phenotypically similar, but not allelic, to *Cg1* (Fig. 1a and b), the former being located on the short arm of chromosome 3 (Lysikov et al., Sov. Genet. 20:90-99, 1984; Krivov and Lysikov, Bulletin AN SSRM 2:20-24, 1988).

Genetic analysis of the *Cg2* macromutation has shown it to be a monogenic dominant mutation which is highly sensitive to maize genetic background and gene dose. Sensitivity to genetic background is reflected in that in crosses of *Cg2* carriers with different maize lines, plants indistinguishable from normal maize plants as well as those with weak (*Cg2-w*), moderate (*Cg2-m*), and strong (*Cg2-s*) expression of the mutant phene (Fig. 1c) occur.

A study of lineages established in 1978 has revealed that the *Cg2* macromutation exhibiting 100% penetrance on the genetic background of specially selected lines mutates at a high rate in somatic and generative cells. In somatic cells, the level of mutability was assessed from the frequency of the *Cg2*-mosaic plants whose lateral shoots usually had mutant phenotypes, with the leading one being non-mutant (Fig. 1d). The *Cg2*-mosaic plants were considered normal if the majority of shoots in their bushes had normal phenotypes (shoots ending in a tassel), and mutant if most of the shoots had the corngrass phenotype. In generative cells, mutability was assessed from the occurrence of normal maize plants in homozygote progeny (*Cg2/Cg2*), with the rate of *Cg2* to *Cg2+* transition amounting to 55%. Among *Cg2+/Cg2+* homozygotes, mutation of the *Cg2+* to *Cg2* type occurred at the rates of 2-2.5% and 5.5% in generative and somatic cells respectively.

However, high mutability is not confined to the *Cg2* locus since mutant forms phenotypically resembling *ys1*, virescent seedling, striate, pericarp and cob colour, ramosa, and brachytic, have been observed among the *Cg2* progeny. Later, these mutations have been shown to be unlinked to the *Cg2* locus. To determine the chromosomal locations of newly arising mutations, carriers of these mutations were crossed with phenotypically similar mutants from the All-Union Institute of Plant Growing (St. Petersburg) collection. Tests of allelism have shown the new mutation *br* to be non-allelic with *br1*, *br2* and *br3*. The new variant *sr* is not allelic with *sr1* and *sr2*, and the new mutation *ra* is non-allelic with *ra1*. Thus, recessive mutations occurring among the *Cg2* progeny reflect the properties of mutator activity of the unstable *Cg2* locus or those of the genetic background of the lines carrying *Cg2*.

Tests of allelism have also revealed that mutations *ys*-253* and *ys*-143* are not allelic to each other, and crossing lines of non-corngrass phenotype carrying these mutations results in up to 51.5% of the F1 plants having the *Cg2* phenotype (Table 1). Tests for the ability to induce instability at the *Cg2* locus on hybridization of *Cg2+* homozygous lines carrying recessive mutations *sr*-220*, *br*-220*, *ys*-253 P-rr*, *ys*-253* and *ys*-143* have shown that *Cg2* and *Cg2*-mosaic plants occur at a rate of 44.1 to

Figure 1. Maize plants carrying mutation *Cg1*, (a) usual mutant phenotype, and (b) *m(Cg)*-mosaic phenotype. Maize plants carrying *Cg2* 3S mutation, (c) usual mutant phenotype, and (d) *m(Cg2)*-mosaic phenotype.

51.5% in combinations *ys*-253* x *ys*-143*, *ys*-143* x *ys*-253 P-rr*, and *ys*-253 P-rr* x *br*-220*. In the latter case, the appearance of *Cg2* plants entirely depended on the direction of the cross. This was indicated by the fact that not a single plant was found in the progeny of the cross with the *br*-220* line used as a female parent.

Table 1. Mutation rates at the *Cg2* locus in crosses between stable *Cg2+/Cg2+* derivatives.

Combination	No. earcorns	*Cg2+*	*Cg2-w*	*Cg2-m*	*Cg2-s*	*m(Cg2)*	*Cg2* carriers (%)	Total
Cg2+ 220 x *ys*-143*	3	141						141
ys-143* x *Cg2+* 220	2	10						10
Cg2+ 220 x *ys*-253*	2	49						49
ys-253* x *Cg2+* 220	1	8						8
br-220* x *ys*-253*	3	110				1	0.9	111
ys-253* x *br*-220*	1	96						96
br-220* x *ys*-253 P-rr*	4	101						101
ys-253 P-rr* x *br*-220*	1	18		14	3		48.6	35
br-220* x *sr*-220*	2	45						45
sr-220* x *br*-220*	3	95						95
ys-143* x *sr*-220*	2	6						6
sr-220* x *ys*-143*	3	164						164
sr-220* x *ys*-253*	2	164						164
ys-253* x *sr*-220*	1	8						8
sr-220* x *Cg2+* 220	1	146						146
ys-143* x *ys*-253 P-rr*	2	47	7	37		6	51.5	97
ys-253* x *ys*-143*	2	76	3	43		14	44.1	136
ys-143* x *br*-220*	2	90						90
br-220* x *ys*-143*	3	77						77

The same situation was observed in crossing the marker line *gs bm2* x *sr*-220* upon which the proportion of mutant plants was 51.7%. However, no mutant plants have been identified in crosses using *sr*-220* as a female parent. Mutant *Cg2* maize plants also occurred in *bm bv bt* x *ys*-253* and *j v16 ms8* x *ys*-253* crosses. A total of 5 marker lines were subjected to test. Three of these appeared to be capable of inducing, in coaction with *sr*-220*, *br*-220* and *ys*-253*, the formation of *Cg2* plants in F1. Genotypes of the lines (F2, F7, MK01, VIR-44, Co125) occurring among the recognized hybrids failed to induce mutations of the *Cg2+* to *Cg2* type. It is only in the O92 x *ys*-253 P-rr* combination that a single plant among 135 plants tested has been identified as a *Cg2* mutant.

The spontaneous appearance of mutant *Cg2* plants as well as *Cg2*-mosaic plants in crosses of phenotypically normal plants seems, like previously observed mutability (*Cg2* to *Cg2+*), to be due to a genetic factor exhibiting unique regulatory functions. According to Poethig (Nature 336:82-83, 1988), the *Tp1* mutation can have similar functions. Genes interacting with *Tp1* and *Tp2* can also be classified into this group of mutants (Poethig, MNL 62:99). Cases like these are also known to occur among other higher plants. For example, a tissue-specific regulatory protein DEF A affecting flower organogenesis has been isolated from a homeotic mutant deficiens (*defA+*) of lion's mouth. It has been suggested that regulation here is performed due to the response of this protein to general cellular signals (Sommer et al., EMBO J. 9:605-613, 1990). However, equally likely is the suggestion that the *Cg2* macromutation instability is caused by mobile elements (ME) similar to *Ac*, *En*, *Uq*, *Mu* and other elements (McClintock, Cold Spring Harbor Symp. Quant. Biol. 16:13-47, 1951; Peterson, Mol. Gen. Genet. 183:440-448, 1981; Friedemann and Peterson, Mol. Gen. Genet. 187:19-29, 1982; Robertson, Mutat. Res. 51:21-28, 1978; Saedler and Nevers, EMBO J. 4:585-590, 1985). MEs have been found in nearly all of the mutant genes studied. Thus, "pure" mutant genes seem to be non-existent (Peterson, Proc. Int. Symp. Plant Transposable Elem., 43-68, 1988). MEs are present both in the nucleus and in cytoplasm. The Robertson *Mu*-strains, for example, carry extrachromosomal *Mu1* and *Mu1.7* elements whose origin is associated with *Mu* activity. These have been suggested to be generated during *Mu* transpositions as intermediates resulting from these transpositions or as products of *Mu* excision (Sundaresan and Freeling, Proc. Natl. Acad. Sci. USA 84:4924-4928, 1987). Three groups of evidence point to the presence of MEs in the *Cg2* progeny: 1) high rate of mutation at the *Cg2* locus in generative and somatic cells; 2) induction of instability at the *Cg2* locus in crossing specially selected lines, and specific role of the cytoplasm genotypes of crossed lines; 3) high mutator activity of the *Cg2* unstable macromutation evidenced by mutants recov-

ered from the *Cg2* progeny which, despite their phenotypic similarity, are non-allelic to known mutations from the collection and to one another.

As the *Cg* corngrass phenotype is due to a defective juvenile-to-mature transition in shoot development, the *Cg2* being phenotypically similar to the *Cg1* heterochronous mutation, we have named the ME responsible for the *Cg2* locus instability *Fpj* (factor prolonged juvenile). The presence of an *Fpj* mobile element is evidenced by consistent irregularities in the time of transition from juvenile to mature (*Cg2* to *Cg2+*) and in restitution time (*Cg2+* to *Cg2*). The disruption of reciprocality in the induction of such transitions seems to be due to the presence, in some lines, of extrachromosomal *Fpj* elements similar to extrachromosomal *Mu* elements.

If, as noted above, a delay in the juvenile-to-mature transition is accompanied by a mutator effect, then introduction of an *Fpj* element into marker lines can induce mutations in marker genes as well. This hypothesis has been tested experimentally using *wx sh* x *Cg2+* and *Cg2+* x *wx sh* crosses. A total of 64,168 *Wx/wx* kernels have been examined; 4,099 out of these (i.e. 6.4%) proved to be mosaic for the *wx* locus. In some families the proportion of mosaic kernels was as high as 20-30% and higher. The high rate of mutation at the *wx* locus is an indirect evidence for the existence of an *Fpj* mobile element which appears to serve the function of switching the genetic program in *Zea mays*.

Finally, lines *sr*-220*, *br*-220*, *ys*-143*, *ys*-253* and MK01 all of which, except for MK01, carry *Fpj* have been tested for the presence of *Dt*, *Uq* and *Mrh* regulatory elements using lines having receptors at *a-m Dt*, *a-ruq*, *a-m(r)h*. It has been found that all of these lines contain *Dt*, *Uq* and *Mrh* (half or more of the kernels from a hybrid ear are mosaic), while the tester lines themselves carry no *Fpj* elements (not a single *Cg2* plant has been found). An attempt to induce the *Cg2+* to *Cg2* mutation in reciprocal cross of MK01 carrying *Dt*, *Uq* and *Mrh* regulatory elements with *sr*-220*, *br*-220*, *ys*-143* and *ys*-253* lines has been unsuccessful. The lack of interaction between *Dt*, *Uq*, *Mrh* regulatory elements and the *Fpj* system suggests that *Fpj* is not genetically identical to these families of mobile elements.

Acknowledgements: The author is very grateful to Prof. A. B. Korol for his useful criticisms in discussing the manuscript. Many thanks are also due to Prof. P. A. Peterson for kindly providing seeds of the tester lines used in this study and to Mr. G. K. Lakhman for translating the manuscript into English.

Mutation system inducing instability in the *Cg2* locus

--N. V. Krivov

The analysis of individual plants from the progeny of the *Cg2* unstable macromutation shows that this monogenic dominant mutation, exhibiting 100% penetrance in the genetic background of specially selected strains, mutates with high frequency in somatic and generative cells. The degree of the *Cg2/Cg2* homozygote instability is as high as 55% in the *Cg2+* → *Cg2* mutation, with the rate of *Cg2+* → *Cg2* mutation of *Cg2+/Cg2+* being 2 to 2.5% and 5.5% in generative and somatic cells respectively. Mutability at the *Cg2* locus can be maintained indefinitely affecting other loci unlinked to *Cg2* such as *sr*-220, br*-220, ys*-253, P-rr* and *ys*-143*. All these mutants, isolated from the *Cg2* macromutation progeny and occurring at a high frequency, were tested for allelism with phenotypically similar mutations from the VIR collection in order to determine their chromosomal locations.

Tests of allelism showed that the new mutant *br*-220* was not allelic to *br1*, *br2* and *br3*. The new variant *sr* was not allelic to *sr1* or *sr2* and the new mutation *ra* was not allelic to *ra1*. Thus it became obvious that recessive mutations isolated from the *Cg2* macromutation progeny reflect the features of mutation activity at the unstable *Cg2* locus or those of the genetic background of the strain carrying *Cg2*. Moreover, crosses between phenotypically normal maize strains carrying *Cg2+* alleles as well as *ys*-253* and *ys*-143* mutations non-allelic to one another, can produce up to 51% F1 plants having *Cg2-m* and *Cg2-w* phenotypes as well as *Cg2* mosaic plants.

Tests for the ability to induce *Cg2+* → *Cg2* mutations upon crossing strains homozygous for the *Cg2+* allele and carrying recessive mutations have revealed that *Cg2* macromutations occur at a frequency of 44.1-51.5% in *ys*-253* x *ys*-143*, *ys*-143* x *ys*-253 P-rr* and *ys*-253 P-rr* x *br*-220* combinations. In the latter case, the appearance of *Cg2* plants appears to depend on cytoplasm genotype since no *Cg2* mutant plants appeared when *br*-220* strain was used as a female parent.

The *Cg2* locus instability persisting for many generations, induction of *Cg2+* → *Cg2* mutations in following hybridization, and high frequency of recessive mutations unlinked to the *Cg2* locus all suggest the existence of a *Fpj* (factor prolonged juvenile) mutation system, similar to the well-known *Ac, Spm/En, Uq, Mrh*, etc. mutation systems. Tests of five marker strains: *wx sh, lg gl2 b v4 gs2 Ch sh/+ fl, gs bm2, bm bv bt* and *j v16 ms8* showed the latter three carried a *Fpj*, since 25-51.7% of plants from crosses of these marker strains had the corngrass phenotype. Testing F7, Co125, MK01, BIP-44, 092 strains, belonging to commercial hybrids, for the *Fpj* presence showed that among 135 plants tested a single *Cg2* mutant plant was found in the 092 x *ys*-253 P-rr* combination. Thus the newly discovered *Fpj* mutation system, while widely occurring in maize marker strains, seems to be found rarely in the strains of cultivated hybrids.

New induced heterochronic *Cg2* mutation

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

In 1962 the *Cg2* macromutation was obtained in M3 after irradiation of VIR-44 strain pollen at the rate of 1500R. Like the well-known *Cg1*, *Tp1*, *Tp2* and *Tp3* mutations, the *Cg2* macromutation has a strong pleiotropic effect and causes (1) a higher number of vegetative buds, (2) a higher number of buds producing ears, stollons, brace roots, (3) smaller leaf and internode sizes, (4) smaller ears and tassel size, and (5) transformation of reproductive structures into vegetative ones. Such phenotypic resemblance of the *Cg2* with already known macromutations leads to the conclusion that the *Cg2* is a heterochronic mutation with prolonged expression of the juvenile stage in the vegetative development programme, which is superimposed with a reproductive development programme.

As is obvious now, the *Cg2* variable expression described earlier generally falls into three discrete phenotypes: strong *Cg2-s* similar to the *Cg1* macromutation in its expression, moderate *Cg2-m* similar to *Tp1*, and weak *Cg2-w*, *Cg2* allele carriers differing from normal maize by the lack of lateral branches in a tassel. Single maize plants show *Cg2* mosaic expression when the ratio of corngrass to phenotypically normal shoots in a plant may vary strongly. Generally, a progeny is mutant following homozygote *Cg2/Cg2* self-pollination, and heterozygote self-pollination results in the segregation ratio close to 3:1.

However, an analysis of individuals in a progeny of *Cg2/Cg2* and *Cg2+/Cg2+* carried out for 9-11 generations showed that individual maize plants with normal phenotype occurred in the former, and *Cg2* carriers appeared in the latter. While the appearance of phenotypically normal plants in the first case can be explained as an error in parent plant genotype determination, such an explanation is not feasible in the second case.

In our opinion, the appearance of phenotypically normal plants following self-pollination of *Cg2/Cg2* and of individual corngrass-type plants or a small number of these upon self-pollination of *Cg2+/Cg2+* can be due to either 1) random segregation of suppressor recessive genes, which suppress expression of the *Cg2* dominant mutation in a homozygote or 2) *Cg2* gene instability. Therefore, generative cells of a plant will be mosaic due to frequent *Cg2* → *Cg2+* mutations, and both homozygous and heterozygous initial cells will occur. Such mosaicism will result in the appearance of *Cg2+/Cg2+* plants, whose number will vary (depending on the degree of mosaicism).

In fact, the appearance of mutants among the *Cg2+/Cg2+* progeny and phenotypically normal segregants in the *Cg2/Cg2* resulted from *Cg2* gene instability. Macromutation instability is also indicated by: 1) genotype differences between ear progenies from the same plant, 2) high frequency of mosaic plants, and results of the genotype analysis of normal and mutant shoots in mosaic plants, and 3) high mutation activity, evidenced by the rate and spectrum of mutations occurring in the progeny of the unstable *Cg2* macromutation.

Meiotic patterns during microsporogenesis of an unstable corngrass macromutation

--M. G. Nikolaeva and V. N. Lysikov

In order to identify mechanisms of genetic instability responsible for intense morphogenetic processes in maize, a cytological study of mutation activity of the *Cg2* gene has been undertaken. This allowed the appearance of induced chromosomal rearrangements to be monitored.

The genomic analysis (microsporogenesis analysis) has been performed on genotypes homozygous for mutant and revertant *Cg2* alleles and on unstable corngrass macromutation genotypes in maize. Various genotypes exhibit heterogeneous male inflorescence structure: normal panicle, a spikelet (prevailing in homozygotes for the mutant allele) and an accresced spikelet with a small ear. Therefore, the amount of pollen produced by different genotypes depends on the male inflorescence structure.

The most informative stage of meiosis, prophase, was studied in detail. Genotypes differing in the degree of stability have been found to be heterozygous and heteromorphic for heterochromatic knobs. This heterogeneity is more characteristic of unstable genotypes mutating from the corngrass to normal type and back. At pachytene, asynapsis has been observed in some regions of homologous chromosomes, particularly in heterozygous and unstable genotypes. These genotypes exhibited the presence of univalents (M1), irregular assortment of chromosomes during anaphase-telophase of both divisions, and, in some cases, the formation of bridges, and lagging chromosomes. A characteristic feature of meiotic prophase in pollen mother cells (PMC) of the maize corngrass macromutation is adhesion of chromosome centromeric regions and, in some cases, of heterochromatic knobs and chromosome arms, resulting in the appearance of translocation chains and rings at diakinesis-diplotene with possible recombination of genetic material and chromatin redistribution.

The above irregularities result in partial pollen sterility, with the degree of sterility being higher in genotypes homozygous for the mutant allele (*Cg2/Cg2*). The level of fertility of heterozygous genotypes depends on the genotype of the pollen parent and can range between 60% and 97%. Eventually, these irregularities can result in aneuploidy in a more distant progeny.

Having evaluated the effect of genetic instability on intrachromosomal recombination based on a cytological analysis of chiasma distribution we concluded that the *Cg2* macromutation has an inhibitory effect on the overall chiasma frequency, and on the frequency of interstitial and multiple exchanges. Heterozygous genotypes and those undergoing mutations of the type corngrass to normal phenotype are chiasma inducers, i.e. plant genotype affects variation in chiasma frequency and distribution pattern at the microsporocyte level.

The genomic analysis of maize macromutation genotypes homozygous for mutant and revertant *Cg2* alleles and of the corresponding unstable genotypes indicates the possibility of exploiting these genotypes to broaden the range of genotypic variation in maize.

Genetic transformation and mutation induced with pollen irradiated with superhigh and high γ-radiation doses

–I. M. Romanova and V. N. Lysikov

Pollen was irradiated with superhigh and high γ-radiation doses (200-1500Gr). These doses cause loss of pollen fertility due to possible DNA-fragmentation. Utilization of such fragmented pollen, for fetching from which natural pollen is used as a vector, causes mutation and transformational effects.

Marker character transfer was established: sugary endosperm (*su1*), 0.52% frequency; leaf and stem golden colour (*g1*), 4.2% out of marker pollen *su1 bm2 g1* irradiated with the dose of 800Gr on maize ovule genome in the recipient strain. Mangelsdorf tester irradiated pollen (500Gr dose) caused marker character transfer: *su1* in 1.1% frequency, brown midrib (*bm2*) 2.2%, two gene transfer 1.1% following repollination with natural non-irradiated pollen of recipient strain.

It was also found that γ-irradiated pollen (at high doses) produces specific mutagenic activity in generations following the treatment.

Chlorophyll mutations were obtained in H1 during repollination with recipient pollen of the donor strain at the doses of 500, 800 and 1000Gr. The highest percentage of chlorophyll mutation segregation was observed in M2 at the dose of 1000Gr--9.3%. This dose gave the widest variability range--25 characters.

Besides chlorophyll mutations qualitative mutations appeared in leaves, stem, endosperm, seedling colour and male and female sterility. These mutations were inherited in M2, M3 and M4 in different frequencies.

Mutations improving grain quality are the most interesting. Among them are the "sugary" mutation responsible for sugar content and the "opaque" mutation raising lysine and tryptophan content in grain. These mutations were segregating in one family in 0.15% frequency at the dose of 1000Gr.

Induced mutants having a higher number of heads are also of interest. This type of mutation was observed at doses of 300, 800 and 1000Gr, in the highest frequency at the dose of 1000Gr.

Quantitative mutations were obtained in the experiment along with qualitative mutations. The most depressing mutagenic effect

of irradiated pollen on quantitative characters was established in M1 and M2.

Maximal variability of the following characters was observed: tassel length, number of branches in a tassel and heads fully developed. The highest mutation frequency (both quantitative and qualitative ones) was obtained by pollen treatment of M2 in K-167 marker strain in the range of 500-1000Gr.

Meiotic mutations were obtained with distinctive infringement of spiralization, partial asynapsis and chromosome fragmentation following pollination of the recipient strain with pollen of the marker strain *su1 bm2 g1*. Such effect appears at the irradiation doses of 300, 800 and 1000Gr.

The results of the investigation showed the possibility of genetic variability by means of both transformational (*su1 g1 bm2 g1*) and mutational (white, yellow, *su1*, *br2*, *o2*, barren stalk, stripe, striate, lazy culm, etc.) mechanisms.

Maize mutant *Adh1* as a tool to establish cause of *Cg2* instability

--S. M. Buyukly and V. N. Lysikov

The *Cg2* macromutation induced in stock VIR-44 by γ-irradiation is unstable. At early ontogenesis stages *Cg2* mutates both from *Cg2* to + and in the reverse direction to *Cg2* (Lysikov, Krivov, and Golubovsky, Genetica 20:89-99, 1984). In order to test the hypothesis that *Cg2* instability is connected with transposition of previously unknown mobile elements an approach consisting of using the pollen selection system of alcohol dehydrogenase negative mutants and their characteristics at a molecular level was proposed.

Adh1 null mutant search was carried out by means of pollen sensitive to allyl alcohol treatment (Schwartz and Osterman, Genetics 83:63-65, 1976). About 500 plants of several derivatives carrying the *Cg2* gene were self-pollinated with pollen treated with allyl alcohol. When pollen is treated, ADH in pollen grains reacts with allyl alcohol and converts to the highly toxic acrolein. Wildtype pollen grains which contain active enzyme perish, while mutant ones which lack ADH activity survive and take part in fertilization. Ears pollinated with treated pollen had kernels from three to five in an ear and totalled 944. All the kernels were sown in a field, but only 400 plants came to the reproductive stage. Some seedlings did not germinate, some seedlings died at the 3-5 leaf stage. Screening of ADH^+/ADH^- heterozygotes was carried out in F1 by means of specific pollen staining for ADH (Freeling, Genetics 83:701-717). Wildtype pollen grains stain shiny opaque blue, but ADH^- grains are yellow-pink, translucent. Only 3 plants had both ADH^+ and ADH^- pollen grains out of a total number of 389 plants tested with pollen staining for ADH. However, the $ADH^+:ADH^-$ ratios (10:1, 2:1, 2:1) differed from the expected one (1:1). An F1 generation (120 seeds) was obtained only from one of the 3 heterozygotes. This mutant was designated *Adh1-27*.

New mutations phenotypically like yellow stripe-1 and red aleurone were discovered after self-pollination in one of the families in F2 and F3 generations of the *Adh1-27* mutant and besides they appeared simultaneously in the same plant. Earlier in F1 a mutation was found that was phenotypically similar to striate. Thus, in spite of the fact that molecular genetical investigation did not prove the above mentioned hypothesis, we assume that there are relationships between *Cg2* instability and transpositions of mobile genetical elements; supposed insertion mutant *Adh1-27* segregated and new mutations were found in generations.

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Relationship between frequency of autodiploid corn inbred occurrence and origin of initial stock

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

Methods of experimental haploidy may be used successfully in corn breeding in particular to obtain new inbreds faster. At first some promising crosses and public line populations were utilized as the initial stock to produce matroclinal haploids through gene marking. After obtaining some autodiploid inbreds and evaluating their combining ability and identifying promising crosses, these crosses were widely employed as parents to produce new haploids and autodiploids. Thus, there came an idea to use recurrent selection for combining ability together with experimental haploidy. At present the inbreds of the first, second, third and fourth cycles of recurrent selection were obtained. It is worth mentioning that from year to year the frequency of matroclinal haploid occurrence was higher in the initial stock including haploid inbreds. On an average for 8 years their frequency was 40% higher than that of the stock containing the inbreds obtained by traditional selection (Table 1).

Table 1. Relationship between the frequency of matroclinal haploid occurrence and origin of the initial stock (1982-1989).

Initial stock	Number of kernels studied	Number of haploid plants	Frequency of haploid (%)
Populations and crosses of autodiploid inbred origin	1,719,746	3,672	0.214
Populations and crosses of public line origin	2,590,156	3,853	0.153

In some hybrid combinations originating from autodiploid inbreds frequency of haploidy reached 1%. It is necessary to emphasize in the haploid plants obtained from autodiploid initial stock chromosome number doubled more easily after colchicine treatment. In certain genotypes we succeeded in 50% doubling.

Four year data showed that on an average the frequency of haploids of autodiploid origin was 2-3 times higher than that of the haploids of public line origin (Table 2).

Table 2. Relationship between the frequency of haploid diploidization and origin of initial stock.

Initial stock	Number of haploid plants treated with colchicine	Number of in breds obtained	Frequency of diploidization (%)
Populations and crosses of autodiploid inbred origin	1,988	522	26.3
Populations and crosses of public line origin	3,724	406	10.9

Thus, the use of autodiploid inbreds, their crosses and their combinations with the best inbreds and hybrids from the World Corn Collection allows increasing the number of produced haploids up to 4,000-5,000 a year and the number of new inbreds originated from them up to 500. It surely results in wider application of experimental haploidy in corn breeding.

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Genome size in annual species of *Zea*. Relation with cellular parameters and altitude

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Considerable evidence has shown correlations among genome size, cellular characters and ecological parameters in maize. Rayburn (1985) found a positive correlation between DNA content (C value), heterochromatin percentage and number of C-bands per genome and a negative correlation between nuclear DNA amount and latitude as well. Bennett (1972) concluded that "there is a relationship between nuclear DNA content and minimum generation time (MGT) in herbaceous higher plants". This would be the cause of the negative DNA-latitude correlation, because bigger nucleotypes will require longer duration and/or temperature of the growing season, and so, at high latitudes (and altitudes) selection would be operating against higher DNA contents (and, as a consequence, against longer MGT's).

Genome size has also been related to total chromosome length (TCL) and volume (TCV) (Poggio et al., 1986; Bennett, 1987; Poggio and Naranjo, 1990). Such correlation has not been tested in *Zea* yet.

Tito et al. (TAG, in press) have also found a positive correlation between C value and C+ heterochromatin content in species of *Zea*, which could be indicating that higher genome sizes are a consequence, at least in part, of the presence of intragenomic supernumerary segments.

The present investigation examines genome size (DNA content, 2C), TCL and TCV in several maize lines and races collected at different altitudes. They present different vegetative periods, and a variable number of supernumerary chromosomes (B chromosomes). The same parameters have been measured in natural populations of *Zea mays* ssp. *mexicana*, *Z. mays* ssp. *parviglumis* and the only annual species from section *Luxuriantes*, *Zea luxurians*. It is the aim of the present work to add new data and discuss the correlations among genome size, vegetative period, TCL and TCV, proportion of heterochromatin measured through C-banding technique, and altitude in the taxa analysed.

Table 1 indicates DNA content (2C), expressed in pg, TCL, TCV, and altitude for the races and lines studied.

Individuals from three populations of *Z. mays* ssp. *parviglumis* at different altitudes did not present significant differences in their genome size (2C=5.76-5.88pg; F=2.10; p<0.05). On the other hand, the analysis of several individuals from two populations belonging to *Z. mays* ssp. *mexicana* collected at 2200m and 2500m respectively yielded a significant difference (2C=7.71pg and 7.10pg respectively; F=5.14; p<0.05). It was also observed, in accordance with Rayburn and Auger (1990), that there exists a negative relationship between nuclear DNA content and altitude for populations belonging to *Z. mays* ssp. *mays*: Palomero Toluqueño, collected at 2800 meters, presented a 2C value of 5.71pg, while the Tuxpeño race, 300 meters high, showed a DNA content ranging from 6.53 to 7.15pg. Thus, correlation between genome size and altitude would be limited to *Z. mays* ssp. *mays*, and cannot be extended to the whole species. It is interesting to note in this way that in the teosintes flowering would be more strongly influenced by photoperiod than in maize.

Table 1. DNA content, total length, chromosome volume and altitude in annual species of the genus *Zea*.

Taxa	DNA (2C) pg X ± SE	Nucleus no. (replicates)	TCL (µm) X ± SE	TCV (µm3)	Altitude (m)
Section *Zea*					
Z. mays ssp. *mays*					
Gaspé Flint line	4.91*		68.23±0.21	30.10	
c-tester line	5.78±0.05	40(2)	---	---	
Black Mexican Race (0B)	5.63±0.14	20(1)	92.61**	41.64**	
Black Mexican Race (2B)	6.52±0.14	20(1)	99.98**	43.23**	
Black Mexican Race (3B)	6.96***	---	103.66±0.40	44.03	
Palomero Toluqueño Race	5.71±0.11	33(2)	---	---	2800
Tuxpeño Race (0B)	6.53±0.09	41(2)	84.97**	58.14**	300
Tuxpeño Race (4B)	7.15±0.10	20(1)	94.07±0.35	64.37	300
Z. mays ssp. *mexicana*					
K-67-1 (Chalco)	7.10±0.09	60(2)	117.67±0.76	62.18	2400
K-71-1 (Chalco)	7.71±0.11	38(2)	---	---	2200
Z. mays ssp. *parviglumis*					
K-67-5 (Balsas)	5.88±0.82	20(1)	---	---	1350
K-67-14 (Balsas)	5.76±0.80	19(1)	---	---	720
K-67-24 (Balsas)	5.85±0.79	44(3)	103.38±0.50	37.41	800
Section *Luxuriantes*					
Z. luxurians	9.83±0.13	20(1)	149.27±0.82	130.14	800

*=Data from Rayburn et al (1985). ** and *** = Expected data.
All lines are from CIMMYT, with the exception of the c-tester line (Leg. by Ing. L. B. Mazoti) and Gaspé flint (Leg. by Dr Patterson).

Total DNA content shows a significant linear positive correlation with TCL (y=3.52 + 14.58 x; r=0.8949) and with TCV (y=81.05 + 20.51 x; r=0.9506). Nuclear DNA value would be more accurately represented by TCV since this cellular parameter does not depend on the degree of chromosomal spiralization.

Rayburn et al. (1985) pointed out that in maize there is a positive correlation among genome size, C+ bands and heterochromatin percentage. This was confirmed by Tito et al. (MNL 64:71, 1990 and TAG, in press) who found a direct relation among number of C-bands in mitosis metaphase, heterochromatin percentage and nuclear DNA value. Tito et al. (l.c.) observed that C-band size is more closely related with C value than the number of C-bands per nucleus is. In the present paper a correspondence is observed between genome size and number and size of C-bands in most of the taxa. The highest proportion of heterochromatic segments is present in *Z. luxurians*, the species with the highest DNA content (2C=9.83pg). *Z. mays* ssp. *mexicana* (2C=7.10-7.65pg) shows conspicuous bands in almost every chromosome of its complement, while the c-tester line of maize (for example) has only four blocks of heterochromatin.

It is interesting to point out that the c-tester line, belonging to *Z. mays* ssp. *mays*, did not yield a DNA C value significantly different from that of *Z. mays* ssp. *parviglumis*. Nevertheless, the magnitude of the C+ segments is greater in the latter than in the former. This could indicate that heterochromatin is an important factor in the genome size variation, but it is not the only one justifying the differences in DNA content. Depending on the arrangement of the repeated DNA sequences (tandem, separated by unique sequences or moderately repeated, etc.), they could not be detected by the C-banding technique (Tito et al., TAG, in press).

Up to 34 B chromosomes have been detected in maize, with a

DNA content increment of 155% (Jones and Rees, 1982). In our study, supernumerary chromosomes increase the genome size from 10 to 16%, 5 being the maximum number observed in the Tuxpeño race from *Z. mays* ssp. *mays*.

B chromosomes present in the Black Mexican line and the Tuxpeño race were similar, and both distinguished from autosomes in morphology; they presented neither a visible centromere (being probably telocentric) nor nucleolar organizers. The positive heteropicnosis in mitotic prophase suggests that they are mostly heterochromatic. This should be confirmed through the C-banding method.

It is important to accurately assess the presence of supernumerary chromosomes and their effects on different maize lines both from basic and applied points of view. The reason for such an assertion stems from the knowledge that B chromosomes increase, in some cases, the vegetative period and cell cycle length. They can also affect recombination (through the alteration of chiasmata frequency and localization), growth rate, yield, fertility, etc. (Jones and Rees, 1982). It is thus essential to know, in order to establish predictable breeding programs, whether these observed variations are due to the presence of supernumerary chromosomes or not, and if so, if they behave according to the Mendelian laws.

Cytogenetic analysis of the hybrids *Zea mays* ssp. *mays* x *Z. mays* ssp. *parviglumis* and *Z. mays* ssp. *mays* x *Z. mays* ssp. *mexicana*

--María del Carmen Molina, Lidia Poggio and Carlos A. Naranjo

The aim of this note is to present results that support our working hypothesis of a basic chromosome number x=5 in the genus *Zea*. This has been done on the basis of the meiotic behavior of species and artificial hybrids (Molina and Naranjo, TAG 73:542-550, 1987; Naranjo et al., 1990) and from the presence of multivalents in material treated with diluted concentrations of colchicine (Poggio et al., TAG 79:461-464, 1990; Naranjo et al., MNL 65:74-75, 1991). In the present work the meiotic behavior of the hybrids *Zea mays* ssp. *mays* x *Z. mays* ssp. *parviglumis* and *Z. mays* ssp. *mays* x *Z. mays* ssp. *mexicana* are discussed.

Plant materials: 1) *Z. mays* ssp. *mays* x *Z. mays* ssp. *mexicana*: *Z. mays* ssp. *mays* = the "c-tester inbred line" introduced by Horowitz from the United States. *Z. mays* ssp. *mexicana* = Nobogame race, Leg. CIMMYT. 2) *Zea mays* ssp. *mays* x *Z. mays* ssp. *parviglumis*: *Z. mays* ssp. *mays* = the "Colorado Klein" population selected at the Instituto Fitotécnico de Santa Catalina". *Z. mays* ssp. *parviglumis* = (MO24), Leg. CIMMYT.

The hybrid *Z. mays* ssp. *mays* x *Z. mays* ssp. *mexicana* (2n=20) presented, in 81 cells analysed in diakinesis, 10 bivalents, showing a mean of closed bivalents of 9.35±0.13. Secondary association was observed and a maximum of two groups of five bivalents each at diakinesis-metaphase I were formed in 70% of the observed cells.

The hybrid *Zea mays* ssp. *mays* x *Z. mays* ssp. *parviglumis* (2n=20) presented 10ii in 98% of 271 analysed cells and the mean of closed bivalents was 7.00±2.07; the rest of the cells presented 9ii+2i. The pollen stainability was 99% and seed fertility 97%. Secondary association was observed and 46% of the studied cells presented two groups of five bivalents each.

The meiotic pairing of these two hybrids agree with the results obtained in other 2n=20 hybrids, suggesting that the subspecies have genomes homologous enough to produce normal pairing. Secondary association and the presence of two groups of five bivalents each was detected in all taxa with 2n=20. This was considered a relictual diploid condition being one of the evidences for the tetraploid origin of 2n=20 taxa (Naranjo et al., 1990).

Evaluation of essential amino acid overproducers

--Liliana H. Perini, Eduardo A. Bernatené and Carlos A. Naranjo

Different races of maize--native from Argentina--were tested as possible donors of high quality protein, with the purpose of modifying the levels of lysine and tryptophan of the endosperm in the commonmost cultivated maizes.

The evaluations were conducted in a double way. On the one hand, biochemical determinations using traditional methods to quantify the level of lysine (Beckwith et al., J. Agric. Food Chem. 23:194-198, 1975) and tryptophan (Hernández and Bates, CIMMYT, Res. Bull. Num. 13, 1969) were used. On the other hand, based on previous reports (Green and Phillips, 1974; and Rapela, 1980), the retroregulation technique was used to select overproducers of essential amino acids through the culture of mature embryos in media containing lysine plus threonine (L + T). This double method ensures a more efficient evaluation of the endosperm and the preservation of the overproducer embryos.

The native races tested were Pisingallo (originally from Catamarca) and three races from Neuquén. The primary criterion of selection of these races was the degree of endosperm hardness, using only those denoting a high percentage of flint endosperm.

The cultures were carried out according to the methods described by Rapela (1980). They showed a high number of Pisingallo individuals resistant to L + T inhibition. This resistance was shown by the normal development of the radicular zone, considering that this zone is the most affected by the L + T inhibition.

The races from Neuquén studied up to date have been demonstrated to be very sensitive to L + T inhibition. The scarce or null radicular development was reverted by the addition of methionine in the media.

The overproducer individuals were transplanted to the greenhouse 30 days after the seeding in the culture media, demonstrating good adaptation to the environmental conditions.

More about Gaspé germplasm for red flint corn improvement

--Mónica B. Aulicino and Jorge L. Magoja

In the last years, Argentina has expanded the maize cultivation areas to larger latitudes. Early hybrids must be used because of their precociousness, low thermal requirements and quick grain ripeness. They are foreign corns mainly of the dent type.

We are interested in developing precocious materials with red flint kernel type. These cultivars could better respond to Argentine production and would be adapted to the new cultivation areas.

This research started evaluating the ability of Gaspé as a precocity donor (see Aulicino and Magoja, MNL 65:40). It is necessary to know the possibility of introducing precocity from Gaspé into red flint inbreds with normal evolutive cycle.

Seven inbred lines of red flint corn were used in the present experiment: P465, P1338, H38, AD3, P21 obtained from INTA Pergamino, and DYW and CFE from Llavallol. These have been maintained by selfing and it is assumed that they are homozygous.

The inbred lines were used as female parents (P2) and Gaspé as the male (P1). Several F1's were obtained from these crossings. Likewise, other generations are derived from F1 individuals: F2 @ (by selfing) and F2 sib (by crossing "inter se" F1 individuals). The backcrosses were obtained by crossing F1's x Gaspé

(BC1) and F1's x lines (BC2).

The following traits were evaluated in parents and in the other generations: days to tassel from planting (T), plant height in cm (PH), ear insertion height in cm (EIH), stalk diameter in cm (SD), number of rows per ear (NRE), number of kernels per row (NKR), ear length in cm (EL), ear diameter in cm (ED), kernel weight per ear in g (KWE). Means (X), standard deviation (SD) and phenotypic variation coefficient (CV%) were calculated (Table 1).

Table 1. Means (X), standard deviation (SD) and coefficient of variation (CV %).

		P1	P2	F1	F2@	F2 sib	BC1	BC2
	X	40.62	68.67	54.33	57.29	56.29	49.96	62.62
T	SD	1.03	4.32	1.74	1.99	2.07	1.90	3.47
	CV%	2.54	6.29	3.20	3.47	3.68	3.80	5.55
	X	88.00	191.44	170.11	151.39	160.93	126.43	180.19
PH	SD	3.06	20.01	10.94	11.56	9.90	11.98	12.91
(cm)	CV%	3.48	10.45	6.43	7.64	6.15	9.48	7.16
	X	13.62	79.74	51.09	47.66	49.95	28.70	66.05
EIH	SD	1.70	12.52	6.97	8.32	7.52	5.62	10.36
(cm)	CV%	12.49	15.70	13.64	17.46	15.06	19.60	15.66
	X	1.35	1.42	1.41	1.35	1.43	1.30	1.36
SD	SD	0.05	1.19	0.14	0.14	0.10	0.18	0.11
(cm)	CV%	3.92	13.13	9.70	10.70	7.55	13.97	8.10
	X	9.32	12.73	11.23	11.79	11.68	10.86	12.28
NRE	SD	0.42	1.09	0.66	0.85	1.01	0.82	1.07
	CV%	4.55	8.54	5.89	7.18	8.6	7.52	8.75
	X	17.14	23.28	24.67	19.97	21.53	18.85	23.98
NKR	SD	1.90	6.44	4.38	2.36	3.59	3.88	4.54
	CV%	11.10	27.65	17.75	11.83	16.68	20.56	18.92
	X	11.24	13.51	13.61	11.7	12.61	11.40	13.16
EL	SD	0.79	2.78	1.55	1.26	1.51	1.75	1.73
(cm)	CV%	7.03	20.58	11.40	10.25	11.98	15.37	13.13
	X	3.08	3.56	3.50	3.45	3.46	3.41	3.55
ED	SD	0.08	0.15	0.16	0.21	0.18	0.18	0.22
(cm)	CV%	2.49	4.13	4.48	5.96	5.32	5.36	0.34
	X	19.70	52.94	45.62	31.88	38.09	26.78	44.62
KWE	SD	4.28	24.07	7.89	7.13	8.21	9.71	13.94
(g)	CV%	21.72	45.47	17.29	22.38	21.56	36.2	31.94

See abbreviations in the text.

Two techniques for estimating heritability were used:

a. Method of parent-offspring regression (%H): A coefficient of regression was calculated using sib means of F2's, derived from different crosses over the phenotype values of F1's. The b's calculated are the %H (Table 2).

Table 2. Heritability (%H), method of parent-offspring regression.

	T	PH (cm)	EIH (cm)	SD (cm)	EL (cm)	ED (cm)	NRE	NKR
%H	28.41	62.93	54.76	26.66	5.77	59.73	34.73	5.50

See abbreviations in the text.

b. Method of the backcrosses (h^2): This method estimates the heritability using the variances of the segregating populations (F2) and both of the backcrosses, see Warner (Agron. J. 44:427-430, 1952). It allows estimations of additive variances. The efficiency of any plant breeding program depends upon their relative magnitudes (Table 3).

Table 3. Heritability (h^2), method of the backcrosses.

	NRE	EL (cm)	ED (cm)	NKR	SD (cm)	EIH (cm)	PH (cm)
P465	<0	<0	<0	<0	0.67	0.82	0.10
H38	0.49	<0	0.58	<0	0.28	0.11	0.28
P1338	0.32	0.14	0.25	<0	<0	0.81	0.76
AD3	0.83	0.25	0.82	0.29	<0	0.99	0.2
DYW	0.53	0.39	0.37	0.58	<0	<0	<0
CFE	<0	0.54	<0	<0	0.75	<0	<0

See abbreviations in the text.

The heritability values calculated for most of the traits show that the greatest part of the variation is genetic, especially for PH, EIH and ED. Nevertheless, the traits T, SD and NRE overtook greater values than 25%.

The heritability values (h^2) calculated for NRE and EP traits in the materials derived from H38, P1338, AD3 and DYW were in agreement with the %H values found. The negative values (<0) could be explained by genetic inference of the parents on their backcrosses. All the same, the parents are capable of modifying the variance values of their backcrosses in a non-predictable manner. This fact explains the differential behavior of the inbred lines with respect to Gaspé germplasm.

Some ear and plant traits were positively correlated with earlier tasseling date (P). Furthermore, high heritability values obtained point out that selection will be effective for them. This will ensure a positive advance in future breeding.

Cadmium toxicity during germination

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

Information on the relationship between the cadmium content in an organism and the biological effects is needed (Ravera, Experientia 40:2-14, 1984). Concentration of Cd in soils varied widely. Elevated values of 10mg/kg soil or more were found.

We have studied some characters, in controlled conditions without soil, during germination of maize grains to assess the comparative behavior of the progeny and the progenitors treated with elevated amounts of Cd. We have employed two flint type inbred lines, SC66 (B) and SC75 (A), and the hybrid SC66 x SC75 (F1). Grains of similar weights (F1=B=284.5mg; A= 254.5mg) were individually put into vials with cotton-wool and 2ml of distilled water with 0; 44.5 or 89uM of Cd^{2+} (as Cl^-) and were grown at 28 C.

Some results obtained 8 days after germination were:

Table 1. Dry matter diminution. DMD = 100 (initial DW - final DW)/initial DW. Initial DW: F1 = B = 250.4 mg; A = 224 mg.

	F1	B	A
Controls	14.1	13.1	8.6
Cd 44.5uM	12.5	11.7	6.5
Cd 89.0uM	10.8	10.7	6.2

Table 2. Mean Cd concentrations (ug Cd g DW^{-1}) in seedlings. Controls: no amounts.

	F1	B	A
roots	328	351	536
aerial parts	48	43	81
grains	51	112	66
mean	142	168	211

Cadmium concentrations among the various plant parts analyzed mark the pre-eminence in the roots in relation to grains or aerial parts. Cd root concentrations were inversely related to growth rate expressed as DM diminution. Cd has affected the growth of all treatments and populations.

Waxy endosperm maizes: their response to photoperiod

--Victor R. Corcuera*, Luis B. Mazoti and Carlos A. Naranjo
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The maize plant is essentially neutral to photoperiod, though short days speed up its induction to flowering.

With the sole purpose of obtaining waxy endosperm commercial hybrids qualified for industrialization proceedings (such as WET MILLING and others), different types of waxy endosperm maizes were studied: three inbred lines, three populations and one hybrid.

The inbred lines analyzed were:

a. "c-tester" (*c sh wx*), introduced to Santa Catalina during 1937 from Cornell University, and kept under inbreeding since then.

b."CEL WAXY SMC2" and "CEL WAXY SMC3", introduced to Santa Catalina during 1990 from CIMMYT (Mexico).

The populations studied were: SCV1, SCV2 and WEM of the Instituto Fitotécnico de Santa Catalina.

Finally, the hybrid measured was FUNKS WAXY, sent to Santa Catalina from CIMMYT (Mexico) during 1990.

Three different evolutive cycle traits were measured and analyzed in all these materials: T, days to tassel (number of days computed from the date of emergence); S, days to silking; Po, days to pollen. All of these traits were measured as is recommended in the General Catalog of Maize Descriptors by IBPGR (International Board for Plant Genetic Resources).

In addition, to make the results obtained comparative amongst themselves and with others coming out from our next studies, the value of the Heat Units necessary to flower has also been calculated according to a residual method (method of the degrees/day), taking as a baseline temperature 12.8 C (approximately 55 F).

All of the measurements were practiced on the basis of individual plants (nearly 300).

As is well known, the evolutive cycle traits are closely related amongst themselves, therefore the analysis of the results can be done without discriminating each trait.

Table 1 shows the commonest statistical measurements (means, standard deviation, variability %, variance and range) for all the traits studied in each of the materials. The variability % obtained for most of the traits considered was low. This tendency is also observed in the ranges. It must be pointed out that the materials sent by CIMMYT showed a long evolutive cycle in our country. They were protoginous as well. This fact may be due to the latitude in which these maizes were grown, and consequently to the photoperiod. It is known that when maizes adapted or grown in low latitudes are moved to high latitudes, the evolutive cycle becomes longer.

Table 1. Statistical measures for evolutive cycle traits in all the materials studied.

Material		x	S.D.	Var.%	G^2	Range
	T	52.45	2.03	3.88	4.12	49-55
c-tester	S	59.10	1.59	2.70	7.29	55-60
	Po	55.35	2.60	4.70	22.09	52-60
	T	52.29	3.21	6.15	37.82	48-60
SCV1	S	55.38	2.63	4.75	22.56	52-60
	Po	54.97	3.36	6.11	11.29	51-60
	T	51.94	2.42	4.67	21.81	48-57
SCV2	S	56.36	2.40	4.27	18.23	54-60
	Po	54.73	2.45	4.48	20.07	51-58
	T	49.92	2.91	5.83	8.47	48-56
WEM	S	55.10	1.50	2.72	2.25	53-58
	Po	52.50	6.96	13.26	48.44	51-60
	T	56.29	1.81	3.21	3.28	53-60
FUNKS WAXY	S	59.86	1.34	2.25	1.79	58-61
	Po	59.04	1.55	2.63	2.40	56-60
	T	66.42	2.73	4.11	7.45	60-71
CEL WAXY SMC2	S	70.37	2.11	3.00	4.45	66-74
	Po	74.49	2.64	3.54	6.97	70-78
	T	66.24	3.49	5.27	12.18	60-70
CEL WAXY SMC3	S	70.47	1.61	2.29	2.59	68-73
	Po	74.30	2.29	3.08	5.24	71-78

Table 2 shows the existence of statistically significant differences among the maizes evaluated when their evolutive cycle traits are considered.

Table 2. Differences between means for evolutive cycle traits.

Material	T	S	Po
c-tester	52.45a*	59.10a	55.35a
SCV1	52.29a	55.38b	54.97a
SCV2	51.94a	56.36b	54.73a
WEM	49.92b	55.10b	52.50b
FUNKS WAXY	56.29c	59.86a	59.04c
CEL WAXY SMC2	66.42d	70.37c	74.49d
CEL WAXY SMC3	66.24d	70.47c	74.30d

*Individual means within a column followed by different letters are significantly different at 5% level.

Table 3 shows the Heat Units, the average number of days to silking, and a classification of each of the materials studied according to these data.

Table 3. Evolutive cycle traits, general data and classification for each material studied.

Material	S	Heat Units (C)	Cycle
c-tester	59	530.85	short
SCV1	55	494.40	short
SCV2	56	506.50	short
WEM	55	494.40	short
FUNKS WAXY	60	541.25	intermediate
CEL WAXY SMC2	70	628.40	long
CEL WAXY SMC3	70	628.40	long

S: days to silking (approach values).

The single fact that the waxy endosperm maizes show a short evolutive cycle of precocity makes them doubly interesting: 1. On the one hand commercial hybrids of waxy endosperm and short evolutive cycle could be obtained. 2. On the other hand, their precocity could be incorporated into the most cultivated maizes of our country.

The influence of the cob colour on the yields of flint and dent maizes

--Victor R. Corcuera*, Luis B. Mazoti and Carlos A. Naranjo
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During the growing season 1990/91 two bulks were created at the Instituto Fitotécnico de Santa Catalina. Each one of these bulks was composed of a mixture of seeds of different flint and dent commercial hybrids. The purpose of creating these bulks was: 1. To keep a pool of genes useful for the later development of a breeding plan, through which it is expected to obtain commercial hybrids qualified for industrialization proceedings. 2. To focus on individual plants, genes, and on their diverse combinations.

This system definitely let us keep genes and increase the genetic variability through multiple recombinations. The last is possible, as pollination is not controlled within each bulk.

The bulks were grown in two different locations of the province of Buenos Aires: 9 de Julio and Chás. The first one has soils with a medium fertility degree, whilst the second has high fertility soils. Both bulks were grown during November, using a higher density of plants in Chás (50,000 plants/Ha) than in 9 de

Julio (45,000 plants/Ha). At maturation the plants were harvested by hand, and samples were taken at random from each bulk. These samples belonged to flint and dent type in each case.

Thereafter, the samples were measured and analyzed at the laboratory through different descriptors recommended by IBPGR (International Bureau for Plant Genetic Resources). The traits/descriptors measured and evaluated in each ear were: 1. EL, ear length in cm; 2. ED, ear diameter in cm; 3. KR, number of kernel rows per ear; 4. NK, number of kernels per row; 5. EW, ear weight in g; 6. KW, kernel weight (g) per ear; and 7. %C, percentage of cob per ear.

In addition, the colour of the cob was considered for each ear measured. Then for each bulk, four groups of ears were evaluated as follows: flint type and white cob (FWC), flint type and red cob (FRC), dent type and white cob (DWC), and dent type and red cob (DRC).

Finally, the results obtained through the measurements practiced in nearly 600 ears were statistically analyzed.

Tables 1 and 2 show the commonest statistical measurements for each trait in both bulks (means, standard deviation, variability %, variance and range).

Tables 3 and 4 show the existence of some statistically significant differences amongst the average values for each trait in each bulk.

Table 1. Statistical measures for ear traits considering the coloration of the cob. (Chás bulk).

	Group	EL	ED	KR	NK	EW	KW	%C
	FWC	17.49	4.24	14.02	34.83	166.97	137.10	18.10
	FRC	16.63	4.00	13.00	33.71	148.21	120.00	19.27
X	DWC	17.62	4.56	15.20	35.20	204.00	161.00	21.22
	DRC	19.42	4.50	14.88	39.56	210.20	173.20	17.53
	FWC	1.97	0.23	1.56	4.51	34.98	31.05	3.88
	FRC	1.82	0.29	1.30	3.54	32.79	27.10	3.59
S.D.	DWC	1.51	0.38	3.03	3.27	47.22	39.11	4.09
	DRC	1.48	0.36	1.83	2.93	43.93	35.26	3.62
	FWC	3.88	0.05	2.43	20.34	1223.60	964.10	15.05
	FRC	3.31	0.08	1.69	12.53	1075.18	734.41	12.89
G^2	DWC	2.28	0.15	9.18	10.69	2229.73	1529.59	16.73
	DRC	2.19	0.13	3.35	8.58	1929.85	1243.27	13.10
	FWC	11.26	5.42	11.13	12.95	20.95	22.65	21.44
	FRC	10.95	7.25	10.00	10.50	22.12	22.58	18.63
Var %	DWC	8.57	8.34	19.93	9.29	23.15	24.29	19.27
	DRC	7.62	8.00	12.30	7.41	20.90	20.36	20.65
	FWC	11.50	3.20	12.00	16.00	90.00	60.00	10.50
	FRC	12.70	3.60	10.00	28.00	70.00	50.00	14.30
Min.	DWC	15.50	4.20	12.00	31.00	160.00	115.00	17.60
	DRC	17.00	3.80	12.00	35.00	135.00	110.00	11.80
	FWC	21.00	4.80	18.00	43.00	250.00	220.00	33.40
	FRC	19.30	4.40	14.00	39.00	200.00	160.00	28.60
Max.	DWC	19.50	5.20	18.00	40.00	280.00	220.00	28.10
	DRC	23.50	5.50	18.00	46.00	320.00	250.00	25.00

Table 2: Statistical measures for each trait considering the coloration of the cob. (9 de Julio bulk).

	Group	EL	ED	KR	NK	EW	KW	%C
	FWC	14.67	4.00	13.32	28.64	116.89	96.93	17.21
	FRC	15.44	3.89	13.36	30.89	121.38	100.42	17.34
X	DWC	15.11	4.47	15.00	30.36	126.07	100.36	20.56
	DRC	14.07	4.32	13.95	28.73	116.22	94.30	19.31
	FWC	1.75	0.26	1.62	4.53	24.82	21.37	3.85
	FRC	1.82	0.20	1.45	4.38	22.93	19.67	3.32
S.D.	DWC	1.33	0.29	1.71	4.94	34.20	28.38	3.67
	DRC	1.65	0.34	1.68	4.82	30.45	25.90	4.75
	FWC	3.06	0.07	2.62	20.52	616.03	456.68	14.82
	FRC	3.31	0.04	2.10	19.18	525.78	386.91	11.02
G^2	DWC	1.77	0.08	2.92	24.40	1169.24	805.42	13.47
	DRC	2.72	0.11	2.82	23.23	927.20	670.81	22.56
	FWC	11.93	6.50	12.16	15.82	21.23	22.05	22.37
	FRC	11.79	5.14	10.85	14.18	18.89	19.59	19.15
Var%	DWC	8.80	6.49	11.40	16.27	27.13	28.28	17.85
	DRC	11.73	7.87	12.04	16.78	26.20	27.46	24.60
	FWC	11.30	3.50	10.00	18.00	70.00	50.00	9.00
	FRC	12.00	3.60	10.00	24.00	80.00	65.00	12.00
Min.	DWC	13.50	3.90	12.00	22.00	80.00	60.00	14.30
	DRC	11.00	3.50	10.00	19.00	60.00	50.00	10.00
	FWC	20.50	5.00	18.00	42.00	220.00	150.00	29.40
	FRC	20.00	4.40	16.00	43.00	180.00	150.00	30.00
Max.	DWC	18.80	4.90	18.00	37.00	190.00	150.00	25.00
DRC	18.20	5.00	18.00	41.00	230.00	200.00	33.40	

Table 3: Differences between means for ear traits of the "Chás" bulk.

Group	EL	ED	KR	NK	EW	KW	%C
FWC	17.49a*	4.24ac	14.02a	34.83a	166.97ab	137.10a	18.10a
FRC	16.63a	4.00b	13.00b	33.71a	148.21a	120.00b	19.27a
DWC	17.62a	4.56c	15.20ab	35.20a	204.00bc	161.00ac	21.22a
DRC	19.42b	4.50c	14.88c	39.56b	210.20c	173.20c	17.53a

*Individual means within a column followed by different letters are significantly different at 5% level.

Table 4: Differences between means for ear traits of the "9 de Julio" bulk.

Group	EL	ED	KR	NK	EW	KW	%C
FWC	14.67a*	4.00a	13.32a	28.64a	116.89a	96.93a	17.21a
FRC	15.44b	3.89b	13.36a	30.89b	121.38a	100.42a	17.34a
DWC	15.11ab	4.47c	15.00b	30.36ab	126.07a	100.36a	20.56b
DRC	14.07c	4.32c	13.95c	28.73a	116.22a	94.30a	19.31b

*Individual means within a column followed by different letters are significantly different at 5% level.

From the detailed observation of the data given in the tables, it may be pointed out that:

1. In high fertility soils, dent maizes yielded more than the flints, though when one considers the low fertility situation (9 de Julio) there are not significant differences in yield between these two types (unless considering individual ears).

2. In high fertility soils, the DRC maizes were the highest yielding, and the FRC showed the lowest yields. The opposite occurs in low fertility soils.

Undoubtedly, the data and the general tendencies shown in this issue constitute an interesting fact, which will be deeply studied during the next growing seasons from this time on.

Furthermore, the multiple recombinations occurred in the field during the last growing season (90/91), and according to the results obtained, it can be pointed out that the coloration of the cob influences, although partially, the final yields.

The colour of the cob, as well as pericarp colour, is genetically determined by allelomorphs of the gene *P*. Thus, it would be convenient to study if there is some kind of association between this gene and the ones determining yield, though this is very complicated as it is a phenomenon of quantitative inheritance. If this fact could be effectively proved, the colour of the cob related to the endosperm texture might be considered in breeding plans to obtain commercial hybrids specifically developed for different areas with a different fertility degree of their soils as well.

Phenotypic and chromosomic aberrations in plants regenerated from callus

--Maria del Carmen Molina, M. D. Garcia[1] and O. Caso[2]
[1]F.I.C.A., [2]CEVEG, CONICET

Maize plant regeneration from callus gives the possibility of obtaining genetic variability such as aneuploidy, polyploidy and chromosomic deficiencies, duplications, inversions and translocations. Nutrient media, time in culture, explant and genotype affect the occurrence of these aberrations (D'Amato, Frontiers of Plant Tissue, pp. 287-295, 1978; McCoy and Phillips, Can. J. Genet. Cytol. 24:559-565, 1982; Lee and Phillips, Genome 29:109-112, 1987).

The object of this work is the phenotypic and cytogenetic study of maize plants regenerated from organogenic callus after 32 months in culture (Fig. 1).

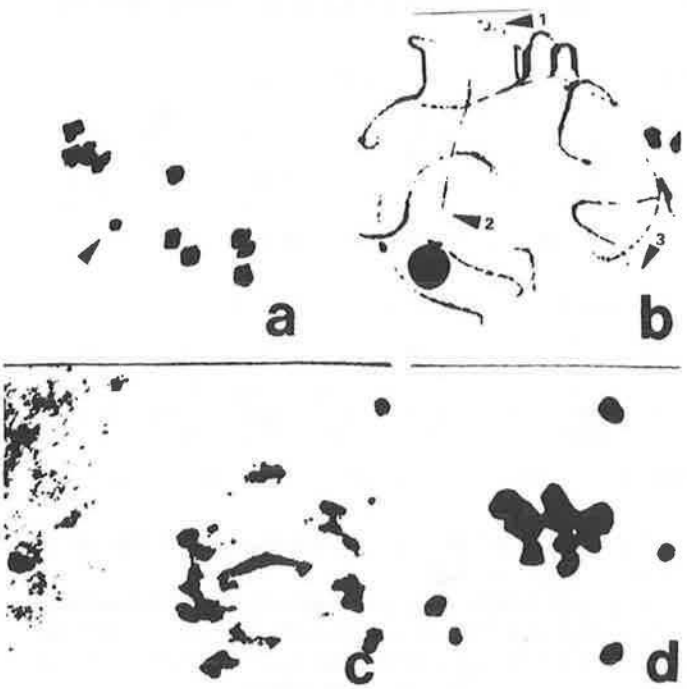


Figure 1. Meiotic chromosomes in maize plants regenerated from callus after 32 months in culture. 1a. Metaphase with an extra chromosome. 1b. Pachynema showing an extra chromosome (10 and deficiencies (2-3). 1c. Anaphase showing inversion bridges. 1d. Metaphase with 5 monovalents.

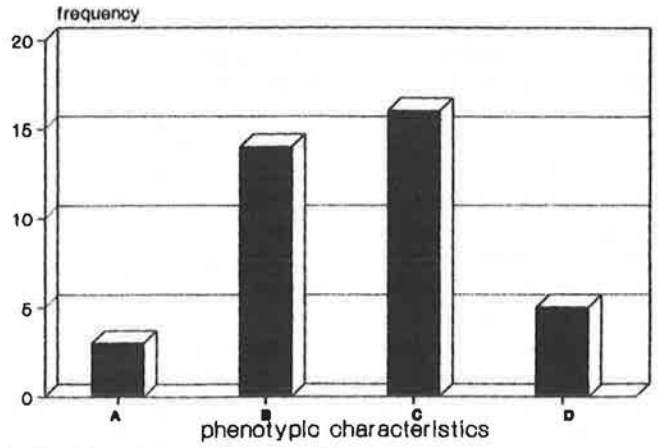
The callus was initiated from one immature embryo (1mm length) of *Zea mays* ssp. *mays* cv. Colorado Klein on nutrient medium containing 0.5mg/L 2,4-dichlorophenoxyacetic acid (2,4-D). After 4 weeks the callus was cut into small pieces and maintained by subculturing on media with 1 or 2mg/L 2,4-D every 30 days (Garcia et al., MNL 64:72-73, 1990). Shoots arose from callus on maintenance medium near the end of each subculture period. Adventitious roots arose from shoots on nutrient medium containing N6 mineral salts (Chu, Proc. Symp. Plant Tissue Cult., pp. 43-45), N6 vitamins, L-asparagine 150mg/L, 0.5 or 1mg/L naphthalenacetic acid, sucrose 5%, agar 0.8% and pH 5.8. Callus still regenerates plants after 50 months in culture.

Tassels were fixed in a solution composed by acetic acid and ethanol (1:3), then stained with acetic haematoxylin.

The cytogenetic analysis of maize plants regenerated after 12 months in culture revealed that 70% of them had normal karyotype and the remaining 30% showed some alterations (Garcia et al., MNL 64:72-73, 1990).

Fifty plants obtained from callus after 32 months of subcultures showed the following characteristics: a) normal plants with different ear insertions; b) plants of 1m height with many stems, leaves and ears; c) plants of 35-40cm height with short internodes and many leaves--these individuals are similar to shrunken mutants; d) plants of 15-30cm height, small stems, wrinkled leaves, sterile and small tassels and ears--similar to dwarf mutation.

Only 8% of the plants showed a normal phenotype, 92% showed different characteristics if compared to their progenitors (Graphic 1).



Graphic 1. Phenotypic frequency of regenerated plants.

Cytogenetic analysis revealed that 20% of the plants had a normal karyotype (2n=20), and 80% had an extra chromosome (2n=21).

The chromosomic configuration 10ii + 1i was the most frequently observed, although some plants showed a high number of monovalents, deficiencies, inversions and translocations. These phenomena have never been observed in plants regenerated from callus after 12 months in culture.

Chromosomic aberrations were not correlated with phenotypic abnormalities found in regenerated plants.

The conclusions of this work are: 1) Tissue culture in the presence of 2,4-D causes chromosomic and phenotypic aberrations in maize regenerated plants. 2) Chromosomic and phenotypic aberrations increase with the period of culture on medium containing 2,4-D.

Phenotypic abnormalities in the progeny of plants regenerated from callus

--M. D. Garcia, Maria del Carmen Molina and O. Caso

Maize plants have been regenerated from callus after 17, 32, 48 and 50 months of subcultures. This callus was obtained from one maize embryo (cv. Colorado Klein) and cultured in vitro on medium containing 2,4-D (Garcia et al., MNL 64:72-73, 1990).

Seventy percent of plants regenerated after 17 months showed normal phenotype and karyotype whilst 30% showed light phenotypic abnormalities and some chromosomic alterations.

The plants regenerated after 32 months of subcultures showed normal phenotype in 8% but the other 92% showed great somaclonal variation with different degrees of phenotypic abnormalities, including the appearance of similar traits to those produced by the genes dwarf and shrunken, which have never been observed in the original Colorado Klein population.

The meiotic study of this progeny revealed that 95% of the plants had chromosomic aberrations such as deficiencies, duplications, inversions and translocations, and 70% of the same plants had an extra chromosome.

One hundred percent of plants regenerated after 48 and 50 months of subcultures showed great phenotypic abnormalities, and the chromosomic behavior is being studied now.

Plants regenerated after 32 months of subcultures were self-pollinated or open-pollinated if pollen was sterile. The kernel color of 75% of the ears obtained mutated from yellow flint to white flint.

The analysis of the offspring of one of these ears (white flint kernel) showed great variation both for plant height and ear insertion, plants with two ears at the same node, non-branched tassels, colored anthers, albino plants and completely deformed plants.

The most dramatic and spectacular segregation was observed in the ear, because whilst the original one was white flint, the self-pollination of the offspring of it produced: 1) ears supporting yellow flint kernels, similar to the original population; 2) ears with yellow and white flint kernels; 3) ears supporting dent kernels as well as yellow and white sugary ones; 4) ears with dent and white sugary kernels in the rate 3:1; 5) ears with flint, dent and sugary kernels; 6) some ears with viviparous kernels and brittle endosperm.

It is important to point out that each plant regenerated from callus obtained from one embryo has a different phenotype and genotype, but a high number of recessive genes (not present in its parental types) appear. This means that each cell or group of cells regenerates a new maize plant with a different genetic constitution.

Lee (M.S. thesis, 1984) supposes that the high number of chromosomic breakages and the appearance of a high number of recessive genes could be due to the action of transposable elements, which would be responsible for the high genetic variation observed in those regenerated plants. This author also points out that the genome stress occurred while in vitro culture activates certain elements which behave as silent genes during the normal stage.

It could be also considered that these changes are produced by effects of the 2,4-D (a mutagenic substance), because when the culture term increases, alterations do too.

Whatever the mechanisms producing genetic variation at the callus, it is important to remark that this method could be used as a source of genetic variation, as the changes occurred have been shown to be heritable.

MADISON, WISCONSIN
Agrigenetics Company

A comparison of three Agrigenetics maize RFLP linkage maps

--Jennifer Shoemaker, David Zaitlin, Jeffrey Horn, Sandra DeMars, Jane Kirschman and Jan Pitas

The Agrigenetics RFLP linkage map of maize has undergone considerable change since it was first published four years ago (Murray et al., MNL 62:89-91, 1988). Several more cloned genes have been added, and the map has expanded with the inclusion of segregation data for an additional 78 public probes. A cytological marker, the gene cluster encoding the cytoplasmic 5S rRNA, was placed on the long arm of chromosome 2 (see Zaitlin, Steffensen and Zimmer in this issue), and a genomic *Sal*I fragment (6-1-1) that lies adjacent to a *Mu1* element (Qin and Ellingboe) has been mapped to a locus on chromosome 8. All of the 3-class RFLP probe segregation data has been reanalyzed, and we have chosen to present the map in a format that reflects the specific population in which each probe locus was actually mapped rather than as a grand composite (as was done previously). Differences between the current map and the 1988 version and discrepancies between the Agrigenetics linkage map and the maps developed at the University of Missouri and Brookhaven National Laboratories are discussed.

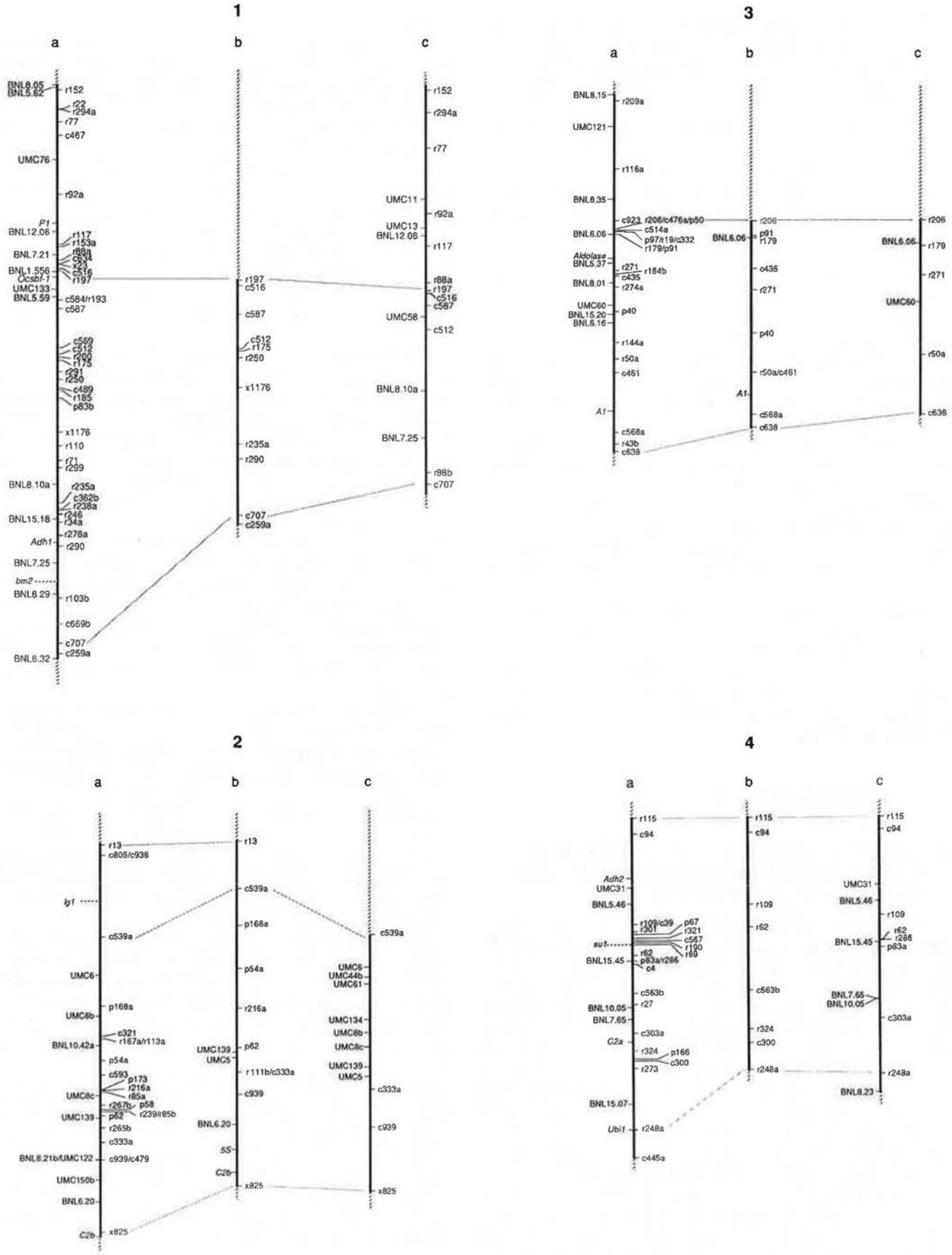
Mapping populations: Two-hundred ninety-one probes were mapped to 293 discrete loci in an F2 progeny set from A619 X Mangelsdorf's Multiple Tester (map a in the Figure). Allelic segregation data were recorded from a minimum of 88 and a maximum of 99 individuals for each probe. The six recessive phenotypic traits (from Mangelsdorf's Tester) were all scored in the field as 2-class data for 90 plants. Map b was calculated from the segregation scores for 87 probes (89 loci) in 93 F2 progeny from B68 *Ht* X B73 *Ht rhm*. In addition to the two F2 populations, we mapped 115 probes to 120 loci in a set of 200 related inbred lines (a recombinant inbred family; see Burr and Burr, Trends Genet. 17:55-60, 1991) from the cross De811 X B73 *Ht rhm*. Also, three single genes for resistance to fungal leaf diseases (*Rp1, Ht2,* and *rhm*) have been mapped in other populations and are not included here.

Molecular probes: The majority of the Agrigenetics proprietary maize RFLP probes were isolated from two specific cDNA libraries made from poly(dA)-containing maize RNA. Clones prepared from etiolated coleoptile mRNA in pSP64 (Melton et al., Nucl. Acids Res. 12:7035-7056, 1984) are given the prefix "c", while those prepared from root polysomal mRNA in pGEM-2 are designated "r" clones. A number of genomic clones are also represented. *Pst*I-derived clones ("p") are in pGEM-3Z and *Xho*I fragments inserted into the *Sal*I site of pGEM-3 are "x" clones. The pGEM plasmid vectors were obtained from Promega Corporation in Madison, WI. All of the *Pst*I-generated clones from the public laboratories, those with BNL and UMC prefixes, were recloned into the plasmid pJKKmf(-) (Kirschman and Cramer, Gene 68:163-165, 1988), which, as with the pGEM family of plasmids, enables the synthesis of a labelled RNA transcript of high specific-activity ("riboprobe") from either of two bacteriophage promoters.

Mapping program: We have developed proprietary mapping software that enables us to construct genetic linkage maps from molecular probe segregation data in virtually any population of known structure. The basic program, Surveyor, employs orthogonal contrasts to detect segregation distortion not due to linkage. If population sizes are too small (~32 individuals for F2), no linkage is calculated. Currently, Surveyor does not use orthogonal contrasts when evaluating the data from a recombinant inbred population. Thus for the map labelled c, we deleted all probes and individuals that had greater than 10% missing data; the final population size was 182 individuals.

Surveyor uses maximum likelihood methods to calculate genetic linkage. Except in the simplest cases (e.g. BC1, F2), there is no "formula" for calculating linkage based on observed frequencies; instead, linkage values are calculated by numerically maximizing complicated likelihood formulae with the aid of a high-speed computer. Once the linkage values have been calculated, the set of

Figure 1. Agrigenetics maize RFLP linkage map. The chromosomes are oriented with the short arm at the top of the page, and the hatched termini signify that the actual ends of the arms (telomeres) are undefined at present. Linkage distances (refer to the scale) are given in percent recombination. Allelic segregation data for probes scored in each population were analyzed using the computer program Surveyor. Agrigenetics probe loci are shown on the right side of the figures, and loci detected by probes from public institutions (UMC and BNL), cloned genes, isozymes and phenotypic markers are shown on the left. Isozyme and gene loci are italicized, and the positions of phenotypic markers are indicated by dotted lines. Lower case suffixes indicate that a given locus is reiterated within the genome in at least one of the three populations (*UMC44a* on 10L and *UMC44b* on 2L, for example). For each chromosome, the three independent maps have been aligned at the first locus common to all three.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UMC139
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c939/c479
UMC150b
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C2b
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p54a
r216a
UMC139
UMC5
p62
r111b/c333a
c939
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5S
C2b
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UMC6
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UMC61
UMC134
UMC6b
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c939
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b
c
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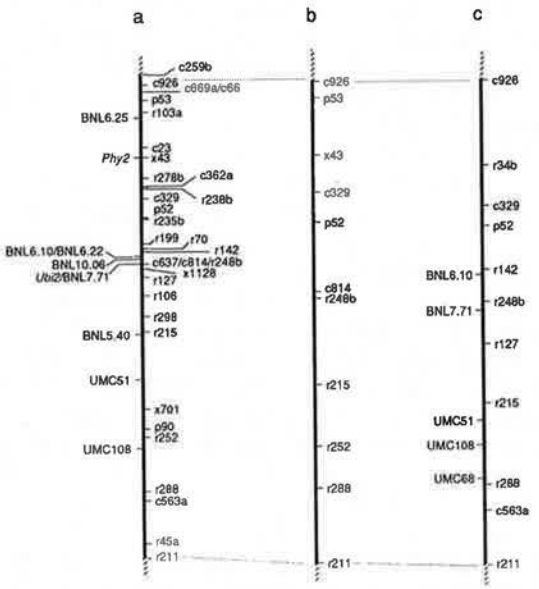
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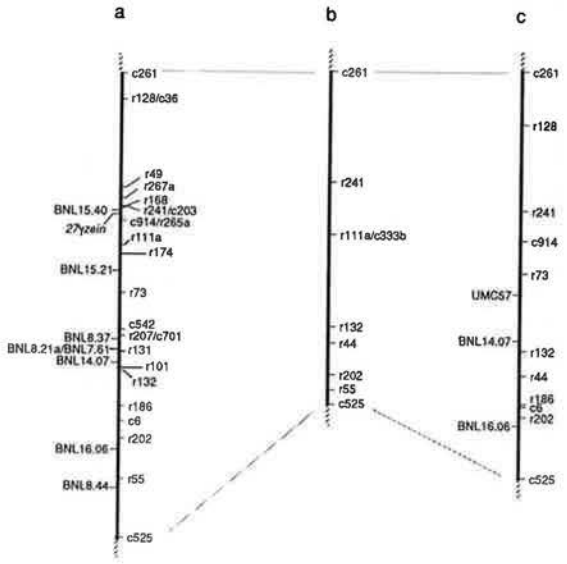
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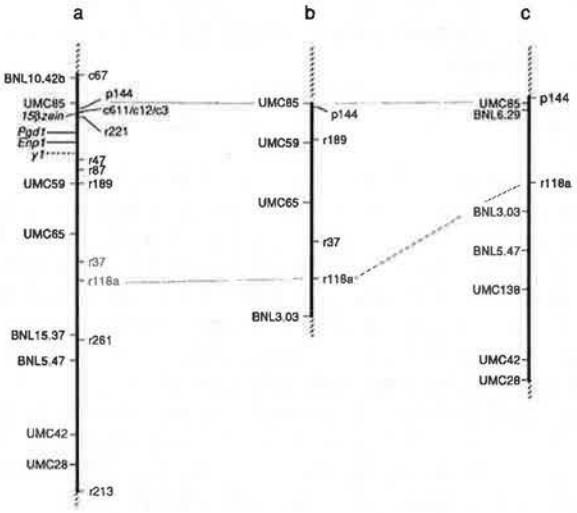
r128/c36
r49
r267a
r168
BNL15.40
r241/c203
27γzein
c914/r265a
r111a
r174
BNL15.21
r73
c542
BNL8.37
r207/c701
BNL8.21a/BNL7.61
r131
BNL14.07
r101
r132
r186
c6
BNL16.06
r202
r55
BNL8.44
c525
c261
r241
r111a/c333b
r132
r44
r202
r55
c525
c261
r128
r241
c914
r73
UMC57
BNL14.07
r132
r44
r186
c6
r202
BNL16.06
c525

6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15βzein
c611/c12/c3
Pgd1
r221
Enp1
y1
r47
r87
UMC59
r189
UMC65
r37
r118a
BNL15.37
r261
BNL5.47
UMC42
UMC28
r213
UMC85
p144
UMC59
r189
UMC65
r37
r118a
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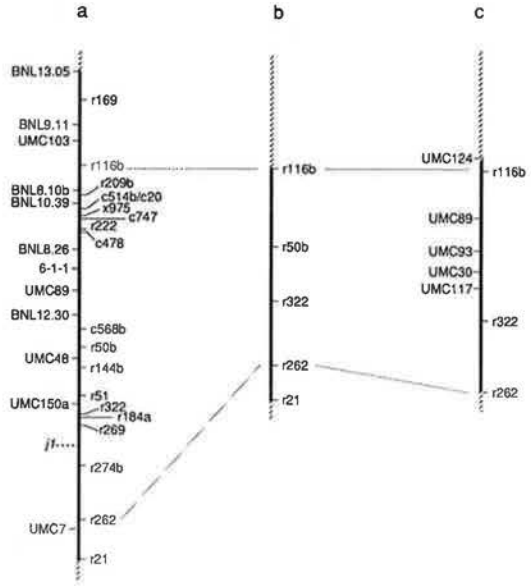
UMC138
UMC42
UMC28

8


a
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r209b
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x975
c747
r222
c478
BNL8.26
6-1-1
UMC89
BNL12.30
c568b
r50b
UMC48
r144b
r51
UMC150a
r322
r184a
r269
r274b
UMC7
r262
r21
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UMC124
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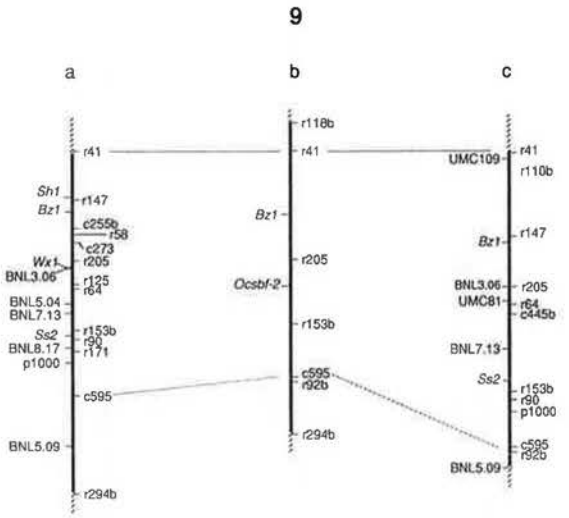
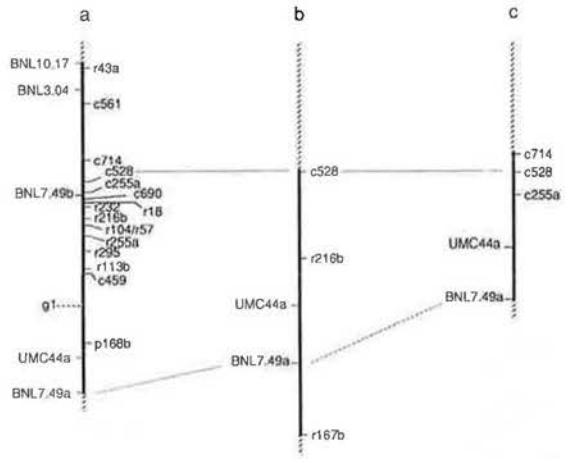
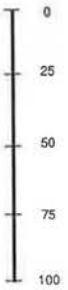
UMC30
UMC117
r322
r262







probes is partitioned into linkage groups based on a similarity relation which depends on a tolerance parameter. Early in mapping, the researcher may explicitly define this parameter; as the linkage map becomes better defined, the researcher can specify the expected number of chromosomes and have the computer calculate a suitable value for the parameter. Each linkage group is then ordered in such a way that the total variance associated with the resulting order is minimized. We use a method called "simulated annealing" to search the space of all possible partition orders in an efficient manner. This process results in a maximum likelihood order for each linkage group. Graphical images are then generated by the Agrigenetics program Drawmap and can be directly ported to a Sun workstation for manipulation in Framemaker.

Features of the linkage maps: As of January 1992, 314 individual RFLP probes, the two isoenzymes *Enp1* and *Pgd1* (*Mdh1* was removed because of the poor quality of the data), and six phenotypic markers (*bm2, lg1, su1, y1, j1,* and *g1*) have been mapped in three populations. Thirty-eight probes are from the University of Missouri, Columbia and 57 probes are from Brookhaven National Laboratories. The remaining 219 probes were developed at Agrigenetics. The maps include 16 cloned gene sequences of known identity, 11 of which are listed in Murray et al., 1988. Five genes have been added since 1988: a genomic clone of the maize aldolase gene (Dennis et al., J. Mol. Biol. 202:759-767, 1988) and two cDNA clones (OCSBF-1 and OCSBF-2) that encode protein factors that bind specifically to the octopine synthase (ocs) enhancer element of *Agrobacterium tumefaciens* Ti plasmids (Singh et al., Plant Cell 2:891-903, 1990) were all received from Liz Dennis; the maize phytochrome sequence (*Phy2* on the map) was provided by Allen Christensen (Christensen and Quail, Gene 85:381-390, 1989) and cLC46E, a cDNA clone of *C2* was from Udo Wienand (personal communication). A genomic clone specific for *O2* (opaque-2), received from R. Schmidt (Schmidt et al., Science 238:960-963, 1987), was not mapped because there was no detectable sequence polymorphism around this locus in our three mapping populations.

UMC42 and *UMC133* map to different positions as compared to the most recent UMC core RFLP map (Gardiner et al., MNL 65:54-56, 1991). We map these loci to chromosomes 6 and 1 respectively while the UMC group maps them both to chromosome 4. These discrepancies are most easily explained by the fact that the probes were mapped in populations that differed with respect to the parental inbreds and the number of individuals. We find that *UMC57* maps to chromosome 7, while the UMC group mapped it to chromosome 10 in 1989 (J. Gardiner, personal communication), although the latest UMC map does not include this locus. A comparison of the Agrigenetics maps with a maize RFLP map provided by Ben Burr in April, 1991 reveals several differences with respect to BNL loci. In addition to small inversions on chromosomes 1 and 9 involving tightly linked loci, there is a rearrangement on chromosome 7 that includes *BNL14.07, BNL7.61, BNL8.37* and *BNL8.21. BNL6.10* and *BNL6.22* are separated on chromosome 5 by at least 10 map units on the BNL map, while we find that these two probes map to one locus. On the short arm of chromosome 6 we define independent loci for *UMC85* and *BNL6.29* that are 2 map units apart (map c), while these two probes map to a single locus on the BNL map.

A comparison of the three Agrigenetics maps shows that they are essentially colinear. Except for some minor rearrangements, the locus orders are essentially preserved between map a (from

A619 X Mangelsdorf's Tester) and the composite map published in 1988. The most notable changes are the inversion on the top of chromosome 1 (*BNL8.05* is now at the end of 1S, while *c467* was the most distal on the 1988 map), and a few places where loci are very tightly linked or where probes that previously mapped to a single locus now define separate loci (*r301, r190, r89* and *c4* on 4a). These changes are due to probe additions as well as to improvements and corrections in the mapping program.

Astute observers will notice that there are occasions where all members of a reiterated pair or trio of loci are not represented on the maps. This is because an independently segregating secondary band could not be firmly placed (*r45b* and *UMC8a* are examples) or, as with *c303b* and *c539b*, too few individuals were reliably scored to calculate linkage (see above). Also, there are several instances in which a large region of a chromosome is missing and is represented by a hatched line. This indicates (i) that there were no polymorphic probes found in that region in a particular population (3b, 3c, and 10b), or (ii) that probes scored as polymorphic and previously known to map to the region did not show linkage to the other loci on the chromosome (1b).

The authors would like to acknowledge Greg Schulenberg and Cheryl Morstad for their expert technical assistance, Kim Maly, Diana Mefford and Signe Melton for help with the map drawing, and Michael Murray for his support and guidance. We also thank Jack Gardiner and Ed Coe of the University of Missouri, Columbia and Ben Burr of the Brookhaven National Laboratories for providing us with their probes.

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Linkage of a second gene for NCLB resistance to molecular markers in maize

--David Zaitlin, Sandra J. DeMars and Manju Gupta

There are presently four dominant or partially dominant genes known that individually condition resistance in maize to *Exserohilum turcicum*, the northern corn leaf blight (NCLB) pathogen. These genes, known as *Ht1, Ht2, Ht3* and *HtN*, can be readily differentiated from one another by the reaction they confer in maize to the known physiologic races of *E. turcicum*. Plants homozygous or heterozygous for either *Ht1, Ht2* or *Ht3* express a qualitative form of resistance characterized by lesions that are extensively chlorotic, rather than wilted and necrotic, and within which fungal sporulation is suppressed (Smith and White, Disease of Corn, in Corn and Corn Improvement, pp. 687-766, 1988). *HtN*, in contrast, acts quantitatively by delaying the onset of lesion development until after flowering (Gevers, Plant Dis. Rep. 59:296-299, 1975). *Ht1* was identified nearly 30 years ago in the inbred GE440 and a Peruvian popcorn introduction (Hooker, Phytopath. 53:660-662, 1963; Ullstrup, Plant Dis. Rep. 47:107-108, 1963), and has long been known to reside on the long arm of chromosome 2 at position 121 between *w3* and *Ch1*. Studies employing molecular markers have demonstrated correlations between the presence of this gene and RFLPs at several loci on 2L between resistant inbreds carrying *Ht1* (introduced through backcrossing) and the corresponding susceptible parents (Hoisington and Coe, Curr. Commun. Mol. Biol., Cold Spring Harbor: 19-24, 1989; Chyi et al., MNL 63:112, 1989). The results of a genetic study linking *Ht1* to RFLP loci on 2L can be found in Bentolila et al. (Theor. Appl. Genet. 82:393-398, 1991).

The chromosomal locations of *Ht2, Ht3* and *HtN* have not been reported, although *Ht2* (from NN14) has been shown to segregate independently of both *Ht1* (Hooker, Crop Sci. 17:132-135, 1977) and *Ht3* (Simone, Ph.D. dissertation, Univ. Ill.-Urbana, 1978; Hooker, MNL 55:87-88, 1980). While conducting a comprehensive survey of maize inbred lines, we observed RFLPs between restriction enzyme digests of genomic DNA from several near-isogenic inbred line (NIL) pairs that differed primarily at the *Ht2* locus (*Ht2/Ht2* vs. *ht2/ht2*) at two molecular marker loci on chromosome 8. The genomic clone *UMC48* detected polymorphisms with *Eco*RI, *Hin*dIII and *Eco*RV between A654 and A654 *Ht2*, Pa91 and Pa91 *Ht2*, and A619 and A619 *Ht2*. The fragments to which the probe hybridized were of a unique size in each of the *Ht2* lines; the actual size was dependent upon the particular restriction enzyme (see Figure 1). The same polymorphism was

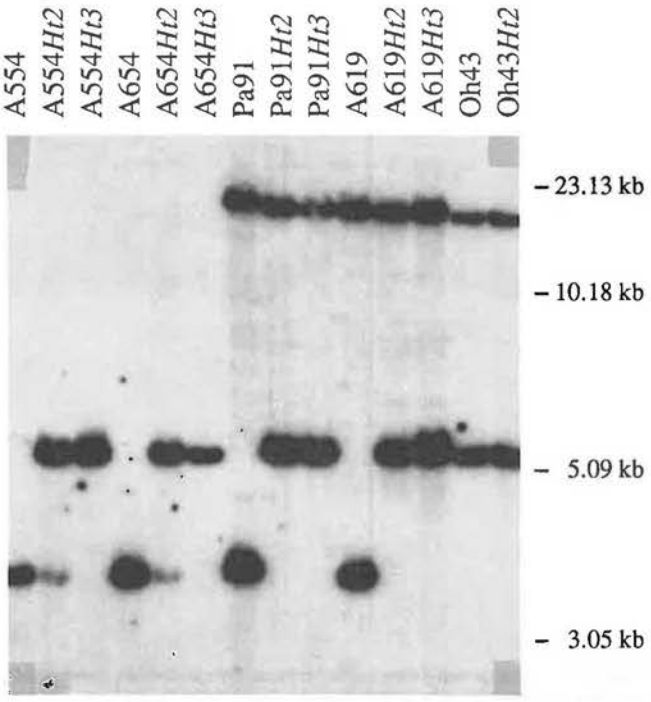

Figure 1. Hybridization of ^{32}P-labelled *UMC48* to *Hin*dIII-digested DNA from fourteen different maize inbred lines. Molecular size standard values are shown on the right side of the figure. No polymorphism was observed between Oh43 and Oh43 *Ht2* at this locus with this restriction enzyme. The high M_w band seen in Pa91, A619, Oh43 and the NCLB-resistant versions of these lines segregates independently of the other bands in our *Ht2* mapping population, but remains unmapped (D.Z., unpublished).

observed between A554 and A554 *Ht2* with *Hin*dIII and *Eco*RV, between Oh43 and Oh43 *Ht2* with *Eco*RI and *Eco*RV, and between Va26 and Va26 *Ht2* with *Eco*RV only. *UMC30*, which is closely linked to *UMC48*, showed a similar correlation with *Eco*RI between A554 *Ht2*, A654 *Ht2*, Pa91 *Ht2*, A619 *Ht2* and Va26 *Ht2* and their recurrent counterparts, and between Pa91 and Pa91 *Ht2* and A619 and A619 *Ht2* with *Hin*dIII (*Eco*RV was not used).

Approximately 375 F2 plants from the cross A619 *Ht2* X W64A were inoculated with an aqueous conidial suspension of *E. turcicum* Race 1 in the field at Madison 6 weeks after planting. Chlorotic flecking, characteristic of early *E. turcicum* infection, was observed on all plants 4-5 days later. Weather conditions in

the summer of 1991 were nearly ideal for NCLB disease symptom development, and large lesions were evident within three weeks of inoculation. The first 124 F2 plants showing clear, unambiguous symptoms of NCLB were rated and tagged (R:S=2.7:1). Genomic DNA was extracted from ear husk tissue taken from all 124 plants, digested with *Eco*RI, electrophoresed on 0.8% agarose gels and transferred to Genatran 45 nylon membrane. These mapping blots were then sequentially hybridized to twelve RFLP probes that were previously known to map to loci on chromosome 8. The allelic segregation scores for these markers (3-class) were analyzed together with the NCLB field disease ratings (2-class). Eight of the RFLP markers showed linkage to *Ht2* (significance level p=0.05). Recombination frequencies between linked loci were determined by the method of maximum likelihood (Allard, Hilgardia 24:235-278, 1956), and marker order and interlocus map distances (percent recombination) were determined using the method of minimization by simulated annealing (Metropolis et al., J. Chem. Phys. 21:1087, 1953). The linkage map is shown in Figure 2.

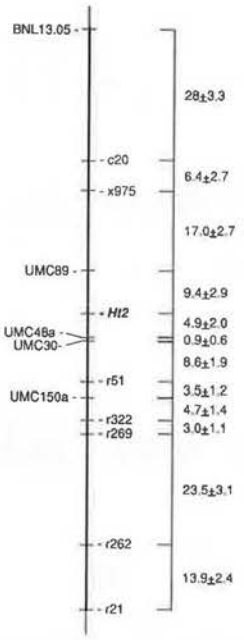

Figure 2. Genetic linkage map of chromosome 8 in maize. Linkage values are shown in percent recombination. Nomenclature for Agrigenetics probes (loci shown on the right) are: c=coleoptile cDNA clone, r=root cDNA clone, x=genomic *Xho*I clone. Probes with UMC and BNL prefixes are *Pst*I-generated maize genomic clones originating from the University of Missouri at Columbia and Brookhaven National Laboratory, respectively.

This is the first report of the genetic mapping of *Ht2* in maize. While we consider this to be a preliminary study due to the use of F2 plants in a field environment, the linkage arrangement of the RFLP markers provides a strong internal control for population structure. We find that the order and relative linkage distances agree quite closely with those determined separately in three other independent mapping populations (see Shoemaker et al., this issue). A large group of F2 progeny plants from A619 *Ht2* X W64A, including many that were used here, were advanced to the F3 generation by self-pollination. This will enable us to further test the current linkage hypothesis in a more rigorous manner next year. We have also made the necessary crosses to investigate the genetic relationship between *Ht2* and *Ht3* in several inbred backgrounds.

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Another cytological marker on the Agrigenetics maize RFLP linkage map

David Zaitlin, Dale M. Steffensen and Elizabeth A. Zimmer

In maize and its close relatives, the more than ten thousand copies of the cytoplasmic ribosomal RNA genes (18S-5.8S-26S RNAs) are organized into a large tandemly repeated cluster on chromosome 6 (Phillips et al., Stadler Symp. 15:105-118, 1983; Zimmer et al., Genetics 120:1125-1136, 1988). This structure, known as the nucleolar organizer region (NOR), is a familiar cytological feature of the short arm of chromosome 6 and is the most distal marker on 6S on several maize genetic maps based on restriction fragment length polymorphisms (RFLPs) (see Burr and Burr, Trends Genet. 7:55-60, 1991). Cytological and genetic studies (Mascia et al., MNL 54:112, 1980; Mascia et al., Gene 15:7-20, 1981; Steffensen and Patterson, Genetics 91:s123, 1979) have established that the genes encoding the 5S rRNA of maize are also clustered in a large repeating array of several thousand copies/haploid genome on the long arm of chromosome 2. The cytological site of the 5S genes was bracketed between two translocation breakpoints (0.75 to 0.82) using the translocations T2-4f (2L.75:4L.12) and T2-6(5419) (2L.82:6S.79). The 5S rRNA genes have been placed on the RFLP map of tomato at a single site on chromosome 1 (Lapitan et al., Genome 34:509-514, 1991). Here we show that sequence polymorphism exists between various maize inbred lines within or around the 5S gene cluster, and that standard RFLP mapping methodology identifies a single genetic locus on 2L between the RFLP loci *BNL6.20* and *C2b*.

The 322bp *Bam*HI 5S repeat fragment from pZm5S5 (Chen and Zimmer, Fed. Proc. 43:1557, 1984) was subcloned into pJKKmf(-) (Kirschman and Cramer, Gene 68:163-165, 1988) to enable the synthesis of a single-stranded ^{32}P-labelled RNA probe with either SP6 or T7 RNA polymerase. Initial screening of *Eco*RI digests of total DNA extracted from a group of maize inbreds revealed that the probe hybridized primarily to a diffuse region on the blots at the upper limit of resolution (>30kb in length). However, there were also eight discrete bands present of somewhat lower signal intensity, ranging between 6kb and approximately 26kb in length. Six of these bands were common to both B73 *Ht rhm* and B68 *Ht*. Bands of ~23kb and 10.3kb were unique to B73 *Ht rhm* and B68 *Ht*, respectively. The 5S RNA probe was hybridized to mapping blots containing *Eco*RI-digested samples of genomic DNA extracted from 93 F2 individuals from the cross B68 *Ht* X B73*Ht rhm*. The two diagnostic bands of ~23kb and 10.3kb were scored individually for presence vs. absence. Linkage analysis showed that the two bands mapped to the same locus (therefore they are allelic), and the autoradiograms were rescored to reflect this. The 3-class 5S rDNA data were incorporated into a large data set containing the allelic scores for 94 RFLP probes that were polymorphic in this population with either *Eco*RI or *Eco*RV. The 5S rDNA cluster showed linkage to 5 probe loci, all from chromosome 2, using the chi-square goodness-of-fit test at a significance level of p=0.05. The Agrigenetics program Surveyor was used to order the markers and minimize the map dis-

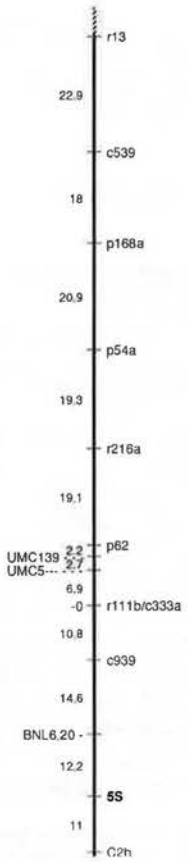

Figure 1. RFLP linkage map of chromosome 2. All maize RFLP probes were mapped in a population consisting of 93 F2 progeny from the cross B68 *Ht* X B73 *Ht rhm*. Agrigenetics probe loci are shown on the right side of the figure and loci detected by UMC and BNL probes are on the left. The figure is oriented with the short arm of the chromosome at the top (the position of the centromere was not determined). Linkage distances are given in percent recombination, and the location of the 5S rDNA gene cluster is shown in boldface.

tances. The resulting linkage map is shown in Figure 1.

Without further investigation we can only speculate as to how the accessory bands that we scored in the mapping of the 5S rDNA gene cluster are generated by *Eco*RI (a few of these bands are just visible in Figure 4 of Mascia et al., Gene 15:7-20, 1981). Because they hybridize strongly to the 5S RNA probe relative to known single-copy sequences, these bands probably represent DNA fragments that individually contain many (between 20 and 75) copies of the 5S repeat unit that arise within the cluster itself or at the junction(s) of the cluster and adjacent sequences. We find that *Eco*RI is much less sensitive to the presence of 5-methylcytosine in plant genomic DNA than are some other restriction enzymes (*Eco*RI will digest sunflower and maize DNA to completion, whereas *Pst*I and *Sal*I will not). Effects attributable to methylation have not been observed with *Eco*RI in the construction of RFLP maps using hundreds of maize DNA clones in several different populations. This would, therefore, tend to rule out such heritable chemical modification as the source of genomic polymorphism between B68 *Ht* and B73 *Ht rhm*. Examination of the nucleotide sequence of the cloned maize 5S rDNA repeat (Figure 2) reveals that there are three subsequences, all within the coding region of the 5S RNA (GAACTC at positions 41-46, GAAGTC at positions 99-104, and GCATTC at positions 113-118) that could each become recognition sites for *Eco*RI (5'-GAATTC-3') with a single base change. Gain of an *Eco*RI site (due to an actual base change, not methylation) has been observed in 10%-50% of

BamHI

5'-GGATGCGATCATACCAGCACTAAAGCACC<u>GGATCC</u>CATCA**GAACTC**CGAAGTTAAGCGTG

CTTGGGCGAGAGTAGTACTAGGATGGGTGACCTCCTGG**GAAGTC**CTCGTGTT**GCATTC**CT

TaqI

ttttttttggttttatttttgtttgttttt<u>cga</u>acaccaaaccatagtaaaaatgggtga

ccgttctcgtgttaaattacctttttatttgttttaaggggcgtttttcgggtactttgtg

cggaggaaggatgtccagtgccgtgggcgagccgagcacggaggtgtgggcataagagga

aggaggacggcaggatatgac-3'

Figure 2. Nucleotide sequence of the 322bp maize 5S rRNA repeat from pZm5S5. The 120bp coding sequence of the 5S RNA is shown in upper case letters and the sequence of the spacer region is shown in lower case. Conserved recognition sites for *Bam*HI and *Taq*I are underlined, and the three potential *Eco*RI recognition sites are emboldened. Comparisons of the maize 5S rDNA sequence with those from five other plant species reveals that the subsequences GAACTC (41-46) and GAAGTC (99-104) are conserved in the genes from wheat, flax, yellow lupine, rye and rice, while the subsequence GCATTC at positions 113-118 is present only in maize.

the repeat units of the rDNA array in some inbred lines of maize (Zimmer et al., 1988); Jupe and Zimmer, Plant Mol. Biol. 14:333-347, 1990). Conceivably, such mutational events could also have occurred within the 5S gene cluster. Mascia et al. found that a small percentage of the *Bam*HI sites located within the coding region (nucleotides 30-35) were no longer cleaved by this enzyme due to sequence divergence, although none of these variants were sequenced. As few as nine *Eco*RI sites could account for the eight bands seen on the autoradiograms (given that there is an *Eco*RI site between each end of the cluster and the adjacent sequences), although this is certainly a minimal estimate.

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Recombination between *Rp1-G* and *Rp5*

--M.A. Sudupak, K.S. Hong and S.H. Hulbert

Race specific resistance to the fungal pathogen *Puccinia sorghi* occurs at 6 or 7 identified loci in maize. Many of the resistance factors identified, however, map to one of these loci. At least 14 different resistance factors, *Rp1-A - Rp1-N*, were mapped to the *Rp1* locus on the short arm of chromosome 10 by Hooker and co-workers. Each allele has a distinct response to different races of *Puccinia sorghi*. The *Rp1* locus is flanked by the RFLP markers *bnl3.04* on the distal side and *npi422* and *npi285* on the proximal side. Recombinational analyses of *Rp1* using flanking RFLP markers has indicated that most of the *Rp1* alleles are clustered closely together (within 0.5cM) but the *Rp1-G* gene maps one to three map units distally, depending on the cross (MGG 226:377). *Rp5* also mapped about three map units distally.

In order to determine the recombinational relationship between *Rp1-G* and *Rp5*, we constructed testcross populations segregating for both genes. *Rp1-G* R168 was crossed to *Rp5* R168 and the F1 was crossed to the susceptible inbreds Oh43 and H95. These populations were then screened for susceptible individuals using the rust isolate IN1 which is avirulent on both *Rp1-G* and *Rp5* (Plant Dis. 75:1130-). Three susceptible recombinants were observed in 3450 seedlings. The distal RFLP marker, *bnl3.04* was not informative in this cross, so it could not be verified that the susceptible progeny were derived by crossing over between *Rp1-G* and *Rp5*. However, virtually all of the susceptible progeny we have isolated from other *Rp1* heterozygotes have had non-parental combinations of flanking markers. The proximal marker, *npi285*, was informative in this cross and all three susceptible progeny had the *Rp1-G* parent allele, as would be expected if

Rp1-G maps distally to *Rp5*. These data, however, should be interpreted with caution; we have observed both types of nonparental flanking markers in testcrosses of certain *Rp1* heterozygotes, presumably due to mispairing of duplicated sequences and crossing-over. It is therefore possible that when more *Rp1-G-Rp5* recombinants are analyzed the other flanking marker configuration will be observed. Furthermore, the observed frequency of recombination between *Rp1-G* and *Rp5* is about eight times lower than the frequency of instability of *Rp1-G* homozygotes observed by Pryor (MNL 61:37) and we have not yet determined the mechanism causing instability of *Rp1-G*.

The distal position of *Rp1-G* has indicated that *Rp1-G* maps to a locus separate from the other *Rp1* alleles and it appears to be either allelic to, or distal to, *Rp5*. Like the other *Rp1* area genes tested so far, *Rp1-G* is proximal to the closely flanking RFLP locus *bnl3.04*. When 130 random test cross progeny from the cross (*Rp1-G* R168 X *Rp1-I* R168) X B14 were scored for rust resistance and *bnl3.04* genotype, two recombinants were observed between *Rp1-G* and *bnl3.04*. Two other progeny were recombinant between *Rp1-I* and *Rp1-G* indicating that *Rp1-G* maps between *Rp1-I* and *bnl3.04*.

Mechanism of instability of *Rp1-J*

--M.A. Sudupak and S.H. Hulbert

Rp1 is a complex rust resistance locus with multiple alleles. Several of the 14 alleles at *Rp1* have been demonstrated to be meiotically unstable by Pryor (MNL 61:37) and Bennetzen et al. (Nature 332:369); susceptible progeny occur in testcrosses of *Rp1* homozygotes. The origin of this instability has not yet been elucidated. Possible explanations include a recombinational origin or transposable elements. The identification of RFLP loci which flank the *Rp1* locus, *npi422* and *npi285* proximally and *bnl3.04* distally, allows possible mechanisms to be tested.

To test the role of recombination in this instability, an *Rp1-J* homozygote was constructed that was heterozygous for flanking RFLP loci. This was constructed by generating two parents that were recombinant between *Rp1-J* and either the distal or proximal flanking marker. The first parent carried *Rp1-J* with the *npi285* allele exchanged for that of an *Rp1-F* R168 line. The second parent also carried *Rp1-J* but the *bnl3.04* genotype was that of an *Rp1-D* R168 line. By crossing the two parents, an F1 was made that was homozygous at *Rp1-J*, but heterozygous at marker loci on both sides of the gene. A testcross population from this F1 was screened with the *P. sorghi* isolate KS1, which is avirulent on lines carrying *Rp1-J*. Five susceptible recombinants were observed in 9750 seedlings. All five individuals had nonparental combinations of flanking markers as would be expected if the progeny were derived from an unequal crossing-over event between tandemly duplicated repeats. This model is also supported by the fact that both nonparental marker combinations were recovered. Three of the recombinants had the *npi285* allele of parent one and the *bnl3.04* allele of parent two, while the other two recombinants had the opposite combination. This would be expected if mispairing between tandemly duplicated sequences occurred in both directions.

The frequency of susceptibles (about 1/2000) indicates that *Rp1-J* is moderately unstable when compared to most *Rp1* alleles. This frequency is similar to that reported for *Rp1-B* or *Rp1-C* but less than that reported for *Rp1-G*. We are currently performing a similar experiment using *Rp1-G* homozygotes with heterozygous flanking markers. This should indicate whether instability at *Rp1-G* is also due to unequal crossing-over and determine if this is a general mechanism causing instability of resistance genes in the *Rp1-Rp5* area.

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CIMMYT

Combining ability of different versions of Tuxpeño-based maize germplasm developed at CIMMYT

--G. Srinivasan,* S. K. Vasal and F. Gonzalez C.

Tuxpeño is one of the most productive and successful Mexican races of maize (*Zea mays* L.) for the lowland tropics. CIMMYT has made use of this germplasm extensively in its breeding programs. Over the last 25 years, CIMMYT has developed and improved many Tuxpeño-based germplasms with different objectives using varied breeding methodologies, and selecting for different characters.

In this study, nine different Tuxpeño versions developed at CIMMYT were compared for their combining ability using four testers (two from Tuxpeño and two from ETO Blanco). The different Tuxpeño versions used were: 1) Population 21 (C0); 2) Population 21 (C6); 3) Population 21 (Streak Resistant); 4) Tuxpeño Sequía (C8) - (Drought Resistant version); 5) Population 21 (C2) - (Inbreeding Stress Tolerance -IST); 6) Population 21 (C1) - (Modified Reciprocal Recurrent Selection - MRRS); 7) Population 49 (C4); 8) Population 49 (Streak Resistant); and 9)

Table 1. Mean cross performance of nine Tuxpeño versions and four testers for grain yield, plant height, and days to 50% silking in two environments at Poza Rica (PR) and Tlaltizapan (TL) tested during 1991.

Tuxpeño version		Grain yield t/ha	GCA for for yield t/ha	Plant height (cm)	Days to 50% silking
Pop.21 (C0)	PR	6.60	-0.343	208	59.4
	TL	7.65	0.091	178	71.4
Pop.21 (C6)	PR	6.59	-0.354	213	58.0
	TL	7.67	0.109	186	70.6
Pop.21 (SR)	PR	6.85	-0.090	220	58.3
	TL	8.00	0.436*	190	71.7
Tux. Seq.	PR	7.49	0.545*	208	57.2
	TL	7.89	0.332	174	70.1
Pop.21 (IST)	PR	7.60	0.651**	225	59.7
	TL	7.85	0.293	191	73.1
Pop.21 (MRRS)	PR	7.13	0.182	216	59.2
	TL	8.16	0.599**	193	72.4
Pop.49 (C4)	PR	6.45	-0.497*	198	57.3
	TL	7.45	-0.107	173	68.8
Pop.49 (SR)	PR	6.73	-0.211	199	56.1
	TL	6.81	-0.752**	169	68.5
Pop.49 (IST)	PR	7.06	0.117	206	57.2
	TL	6.56	-1.000**	166	71.2
Tester-1	PR	7.44	0.493**	217	58.3
Pop.32 (Syn.)	TL	7.78	0.219	184	71.1
Tester-2	PR	6.41	-0.539**	202	57.2
Pop.32 (S.C)	TL	7.71	0.152	174	69.7
Tester-3	PR	6.83	-0.113	208	58.5
Pop.21 (Syn.)	TL	7.53	-0.026		17970.7
Tester-4	PR	7.10	0.159	214	58.2
Pop.21 (S.C)	TL	7.22	-0.345*	184	72.0
Trial mean	PR	6.82		207	58.2
	TL	7.23		176	71.1
LSD(0.05)	PR	1.29		11.2	1.51
	TL	1.08		13.6	2.39

*, ** represents significance at 0.05 and 0.01 probability levels respectively.

Population 49 (C2) (Inbreeding Stress Tolerance). Thirty-six line x tester crosses and 13 parents were evaluated in two environments at Poza Rica and Tlaltizapan, México during 1991.

For per se performance, Pop. 21 (MRRS) ranked high at Poza Rica (7.5 Mg ha^{-1}) and Pop. 21 (SR) yielded the best at Tlaltizapan (7.59 Mg ha^{-1}). For cross-performance, Pop. 21 (IST) at Poza Rica and Pop. 21 (MRRS) at Tlaltizapan ranked first, yielding 7.6 Mg ha^{-1} and 8.16 Mg ha^{-1}, respectively (Table 1). The improved cross performance of the two hybrid-oriented Tuxpeño versions was due to an increase in their ear length and 100 kernel weight. Highly significant positive general combining ability (GCA) effects for yield were recorded by Pop. 21 (IST) and Pop. 21 (MRRS) at Poza Rica and Tlaltizapan respectively (Table 1). The three versions of Pop. 49 showed negative GCA for yield primarily because they were earlier in maturity than the rest and shorter in plant height and hence relatively lower yielding than the versions of Pop. 21. However, among the three Pop. 49 versions, Pop. 49 (IST) was the highest yielder in crosses at Poza Rica and Pop. 49 (C4) was the highest yielder at Tlaltizapan.

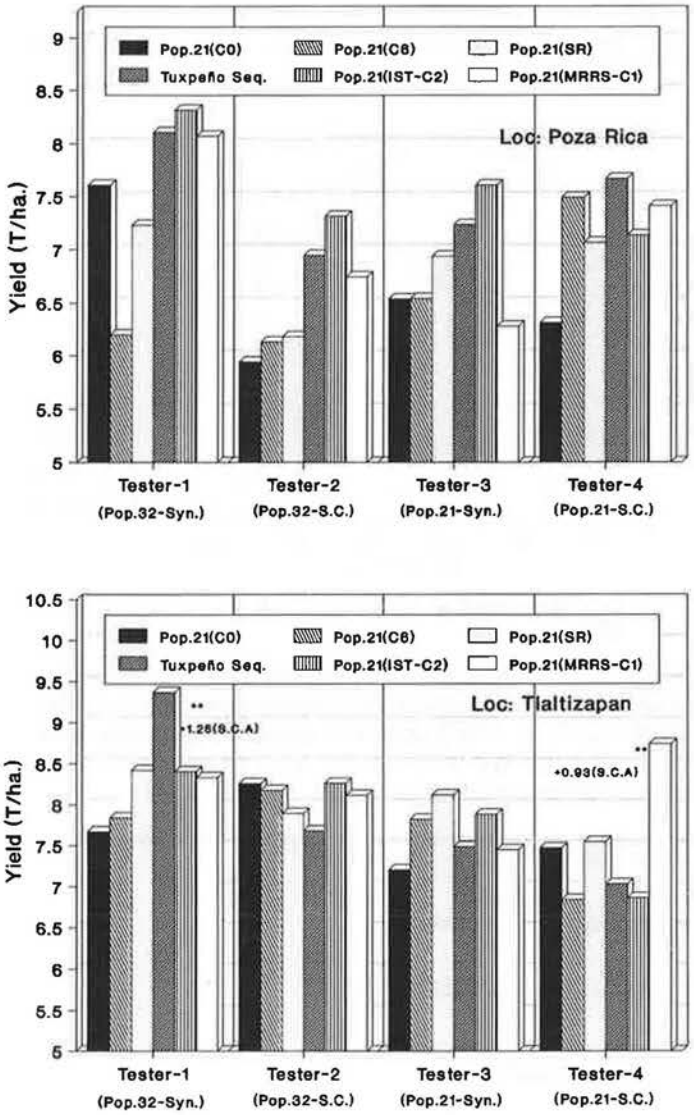

Figure 1. Grain yield (t/ha) of six Tuxpeño versions crossed to four testers.

Tuxpeño Sequía crossed to Tester 1 (Pop. 32 Syn.) was the highest yielder (9.4 Mg ha^{-1}) at Tlaltizapan. At Poza Rica, Pop. 21 (IST-C2) crossed to Tester 1 was the highest yielder (Fig. 1). Results showed that, after six cycles of improvement, although Pop. 21 showed selection gains of 1.72% per cycle for per se performance, there was little change in the cross performance. However, once the breeding methodology was changed with emphasis on hybrid development, the cross performance improved considerably showing gains of 1.4% and 2.4% per year respectively in Pop. 21 (IST) and Pop. 21 (MRRS) (Table 2). Interpopulation improvement using modified reciprocal recurrent selection (MRRS) and breeding for tolerance to inbreeding stress (IST) through S3 recurrent selection has considerably improved Tuxpeño into a more ideal germplasm for hybrid development work at CIMMYT and elsewhere.

Table 2. Cross performance of five Tuxpeño versions for grain yield averaged over two environments (Poza Rica and Tlaltizapan, México) and selection gains realized per year.

	Cross performance	
Tuxpeño versions	Yield (Mg ha^{-1})	Gain/year (%)
Pop.21 C0	7.13	
Pop.21 C6 *	7.13	0.00
Pop.21 C0	7.13	
Tuxpeño Sequía) C8 **	7.69	0.99
Pop.21 C6	7.13	
Pop.21 (IST) C2 §	7.72	1.39
Pop.21 C6	7.13	
Pop.21 (MRRS) C1§	7.64	2.40

* A cycle of selection takes two years
** A cycle of selection takes one year
§ A cycle of selection takes three years

Lodging resistant tropical maize inbred lines from Tuxpeño

-- S. K. Vasal, G. Srinivasan and F. Gonzalez C.

Root lodging due to poor and/or shallow root structure is a common problem in tropical maize growing environments. At CIMMYT, while evaluating our inbred lines for their per se performance, we accidentally discovered a very valuable source for lodging resistance in our Tuxpeño germplasm. During 1988 and 1990, we experienced a severe hurricane at our lowland tropical experimental station at Poza Rica, México during the main growing season (June-Nov.). The hurricane which hit the maize crop just before flowering practically bulldozed the entire farm. To our pleasant surprise, we identified a few patches of maize standing here and there undisturbed by the hurricane. Upon checking our pedigree, we found that they had all come from a single source going back to Population 21 Fam. 219 which we had identified in our previous studies as a good source family for producing superior inbred lines and also as possessing good combining ability.

Three inbred lines arising out of Pop. 21 Fam. 219 have recently been announced in 1991 by the Maize Program, CIMMYT for use by national programs and maize breeders worldwide. They are: 1) CML 9 (Pop. 21 C5 HC219-3-1-B-###), 2) CML 10 (Pop. 21 C5 HC219-3-2-2-3-B-2-2-###, and 3) CML 11 (Pop. 21 C5 HC219-3-2-2-3-#-7-1-B-4-1-B) ["CML" stands for CIMMYT maize line and HC in Spanish for full-sib family].

CML 9 has been extensively used as a tester line in our hybrid research program and has been found to possess excellent combining ability. These lines are characterized by good stalk quality, a vigorous root system with strong brace roots, slightly broader leaves, and a fair degree of tolerance to most of the tropical foliar diseases. CML 9 which is an early generation line is fairly high

yielding and is being used extensively in developing lodging resistant germplasm at CIMMYT. The salient characteristics of the three lines are furnished in Table 1. Small quantities of seeds of these lines are available upon request.

Table 1. Salient features of the lodging resistant lines.

Characters	CML 9	CML 10	CML 11
Maturity	Late	Late	Late
Grain color	White	White	White
Grain type	Semident	Semident	Semident
Yield (t/ha.)	4.5	2.0	2.9
Growing degree days	1126	1143	1085
Plant height (cm)	147	142	152
Ear height (cm)	69	73	77
Ear rot (1-5)*	1.9	2.3	2.2
Rust (1-5)	1.5	1.4	1.7
Maydis (1-5)	2.2	1.8	1.9

* Rating on a scale of 1-5. 1 = Resistant, 5 = Susceptible

Genetic variation and inheritance of resistance to the "Tar Spot" disease complex

--S. K. Vasal, F. Gonzalez C. and G. Srinivasan

Early work at CIMMYT for resistance to the Tar Spot Complex, incited by the fungi *Phyllachora maydis-Monographella maydis*, indicated the presence of genetic variation for different sources of resistance to the disease. Phenotypically, some resistant plants did not have any lesions, some had small lesions, and others had normal size lesions but with slow development of the disease. Genetically, the segregation observed in families of some crosses could be explained by monogenes conferring resistance to the disease, but with different gene action (Table 1). Further

Table 1. Segregation for Tar Spot disease in families of the cross (PD(MS)6 H.E. 02 x Selecc. Precoz.)-B-#-#.

	No. of plants				
Family	Res.	Susc.	Expected ratio	X^2	P (%)
1	14	16	1:1	0.133	>70
2	14	16	1:1	0.133	>70
4	20	11	3:1	1.817	>10
9	21	9	3:1	0.4	>50
10	14	15	1:1	0.035	>80
17	24	8	3:1	0	>99
26	15	17	1:1	0.125	>70
27	15	17	1:1	0.125	>70
29	16	14	1:1	0.133	>70
30	9	21	1:3	0.4	>50
40	22	8	3:1	0.044	>80

analysis of generations of resistant x susceptible crosses confirmed the presence of a single dominant gene in the resistant line Pool 22 TSR-7-B-#, and a single recessive gene in the line Pool 23 TSR-40-B-# (Table 2). Two other sources of resistance

Table 2. Segregation for Tar Spot disease in two crosses involving Pool 22 TSR x Pop.26 and Pop.25 x Pool 23 TSR

	No. of plants				
Generation	Res.	Susc.	Expected ratio	X^2	P (%)
Pool 22 TSR-7-B-#(P1) x Pop.26 C5 HC 37-1-4-B-#(P2)					
P1	42	0	1:0	-	
P2	0	42	0:1	-	
F1	40	0	1:0	-	
F2	65	17	3:1	0.796	>30
BC1	78	0	1:0		
BC2	32	48	1:1	3.2	>5
Pop. 25 C0 HC 128-2-1-B-#(P1) x Pool 23 TSR-40-B-#(P2)					
P1	35	0	1:0	-	
P2	0	39	0:1	-	
F1	0	42	0:1	-	
F2	15	66	1:3	0.796	>10

which had susceptible type lesions but slow development of disease were also crossed to susceptible parents. The development of disease over time, with scores taken at three different dates (weekly intervals), are presented in Figures 1 and 2. Generation

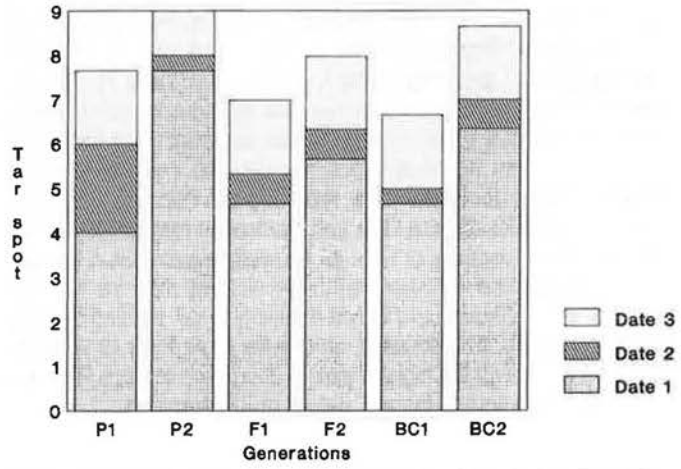

Figure 1. Progression of Tar Spot (0-10) in the cross Pool 26 TSR16-B-#(P1) x Pop. 24 C5 HC34-2-3-B-#(P2).

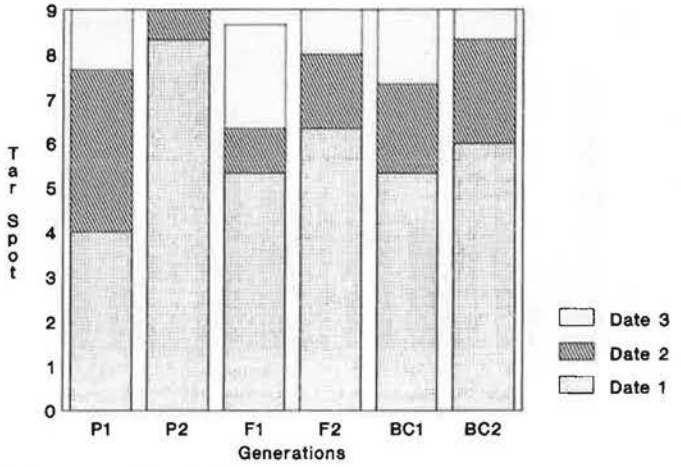

Figure 2. Progression of Tar Spot (0-10) in the cross Pool 19 TSR27-B-#(P1) x Pop. 23 CO-HCS3-31-#(P2).

Mean Analyses (GMA) for these crosses show a relatively simple genetic control of the resistance with mainly dominant and additive effects (Table 3). These and other sources of resistance have been combined to form two yellow and two white populations which will serve as sources of resistance to Tar Spot Complex.

Table 3. Genetic effects for resistance to Tar Spot disease in two crosses with resistant lines from different origins.

Mean*	Additive (A)	Dominance (D)	X^2	P (%)
Pool 19 TSR 27-B-# x Pop.23 C0 HCS3-31-#				
6.92	1.45**	-1.55*	0.41	>90
Pool 26 TSR 16-B-# x Pop.24 C5 HC34-2-3-B-#				
6.24	1.50**	-1.50*	0.51	>90

* Mean disease score ratings on a scale of 1-10.
1 = resistant, 10 = susceptible.

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C.N.R. - Istituto Biosintesi Vegetali

Recovery of callus lines from transformed protoplasts of endosperm suspension cultures

--S. Faranda and L. A. Manzocchi

Studies on the regulation of endosperm-expressed genes in maize have been hindered by the difficulties in obtaining fertile transformed plants. This has led to a renewed interest in tissue culture from maize endosperms (Lyznik and Tsai, Plant Sci. 63: 105-114, 1989; and Quayle et al., Plant Cell Rep. 9:544-548, 1991); their ability in tissue-specific synthesis suggests that, while maintaining a population of meristematic cells, endosperm cultures retain characteristics of the differentiated tissue (Felker and Goodwin, Plant Physiol. 88:1235-1239, 1988).

We have obtained stable suspension cultures from endosperm cells of A69Y maize, expressing reserve proteins (zeins), although at reduced quantitative level with respect to developing endosperms (Manzocchi, Plant Cell Rep. 9:555-558, 1991); we report experiments of protoplast transformation and recovery of callus lines.

Protoplasts were obtained from cultures in log phase with good yields (4-6 x 10^6 protoplasts per gram of packed cells). They were transformed by PEG-mediated DNA delivery with chimaeric gene constructs kindly provided by Dr. A. Viotti and containing the reporter genes GUS (Jefferson et al., EMBO J. 6: 3901-3907, 1987) or NPTII (Beck et al., Gene 19:327-336, 1982) driven by CaMV 35S RNA promoter (Pierce et al., in "Plant Gene Systems and Their Biology", A. R Liss Inc., pp. 301-310, 1987).

Different plating media and techniques were tested to obtain callus regeneration from PEG-treated protoplasts. The best results were obtained by including protoplasts in 1.2% agarose in KM8P medium (Kao and Michayluk, Planta 126:105-110, 1975) at 0.5-1 x 10^6 cells/ml immediately after transformation.

4 days after, division of 20-40% of the cells was observed; visible microcalli were distinguished 20-30 days after. Microcalli of 0.5-2mm could be picked up manually two months after plating and transferred to agar MSE medium routinely used for the growth of endosperm cultures (Manzocchi, Plant Cell Rep. 9:555-558, 1991). Plating efficiency was 0.03-0.05%.

Calli recovered from protoplasts transformed with the GUS gene were screened for GUS expression by histochemical stain two months after transformation. 13% of the calli stained blue with X-gluc reagent (Jefferson et al., EMBO J. 6:3901-3907, 1987), while no GUS expression was detected in untransformed control calli. GUS-expressing calli were successively subcultured and tested histochemically every 20 days; stable transformed phenotypes were observed in 60% of the calli 5 months after transformation.

Difficulties were found in the selection of calli from protoplasts transformed with the NPTII gene, because, as already described for cereals (Vasil et al., Biotechnology 9:743-747, 1991) endosperm cells are quite resistant to growth inhibition by kanamycin. Microcalli of 1-2mm diameter were subcultured on agar media containing 100 - 200 - 400mg/l kanamycin for 5 months, with an average survival of 25%. NPTII activity (McDonnell et al., Plant Mol. Biol. Rep. 5:380-386, 1987) was detected in 42% of the kanamycin resistant calli derived from transformed protoplasts, while no activity was found in control cultures.

Experiments are in progress to define a more effective selection procedure for the isolation of NPTII expressing cultures; Southern blot hybridization experiments are being carried out to test for NPTII and GUS coding sequences in our putative transformed callus lines.

Phenotypic expression in in vitro culture of a mutation affecting endosperm IAA content

--S. Mapelli, S. Faranda and L. A. Manzocchi

Developing and germinating maize seeds have been extensively investigated for auxin content and metabolism, and represent a widely employed model in studies on auxin physiology and biochemical pathways (Reinecke and Bandurski, in "Plant Hormones and Their Role in Plant Growth and Development", P. J. Davies, ed., M. Nijhoff Pub., pp. 24-42, 1987); a new experimental approach would be represented by the use of mutations with altered auxin content or responses.

de-B18* (from the Istituto Sperimentale per la Cerealicoltura, Stezzano, Bergamo) is a viable defective endosperm mutation of maize, monogenic, recessive, characterized by normal germination and auxin content in plant tissues, but extremely reduced levels of both free and bound indole-acetic acid (IAA) in the endosperm during the grain filling period (Torti et al., Maydica 29:335-343,1984; Theor. Appl. Genet. 72:602-605, 1986).

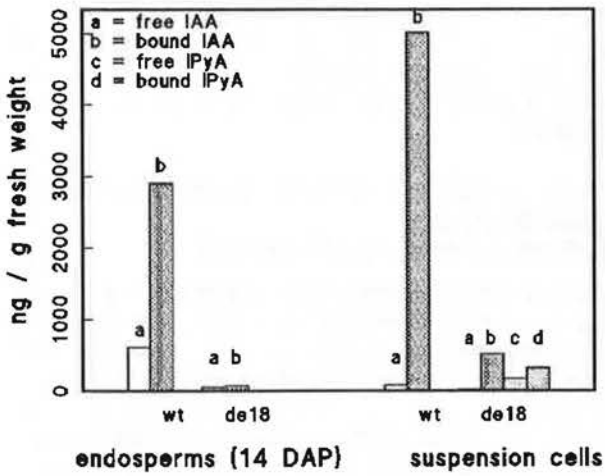
We have established cell suspension cultures derived from maize A69Y wildtype and *de*-B18* endosperms; endosperm cell cultures represent a good tool for the study of biochemical events in developing seeds, as cells do not completely de-differentiate in culture and maintain tissue-specific synthesis such as starch, anthocyanins, and zeins (rev. in Manzocchi et al., Plant Cell Rep. 7:639-643, 1989). Endosperm cultures have also been reported to express in vitro endosperm mutant phenotypes, such as waxy, amylose extender (Sarawitz and Boyer, Theor. Appl. Genet. 73:489-495, 1987) and opaque-2 (Manzocchi, Plant Cell Rep. 9:555-558, 1991).

Calli were obtained from endosperms excised from ears harvested 10 days after pollination and cultured on agar medium without auxin addition (Manzocchi et al., 1989); a good cell proliferation was obtained also for mutant endosperms, in spite of their low auxin content (Castelli, Manzocchi, and Torti, in "Plant Cell Biotechnology", M. S. S. Pais, ed., NATO ASI Series, Vol. H18, Springer-Verlag, Berlin-Heidelberg, pp. 63-67, 1988). Liquid suspension cultures were obtained and subcultured every two weeks with hormone-free medium (Manzocchi, Plant Cell Rep. 9:555-558, 1991), for 18 months; no substantial differences are evident between growth curves of wildtype and mutant cell cultures.

Cultured cells were analyzed for their content in auxins, extracted by acidified methanol. Methanol extract was partitioned against ethyl acetate for free indole compounds; indole ester-bound compounds were extracted in ethyl acetate after mild alkaline hydrolysis of water soluble residue. Analyses were carried out on reverse-phase C18 columns by a HPLC system equiped with fluorometric and electrochemical detectors.

Figure 1 reports auxin content of A69Y wt and *de*-B18* cultured cells in logarithmic growth, and of developing endosperms (14 days after pollination), as a reference.

Cultured *de*-B18* endosperm cells are characterized by a reduced content of both free and esterified IAA, with respect to wildtype cells, and they appear therefore to maintain in vitro the



expression of the mutant phenotype.

Indole-pyruvic acid (IPyA), both free and bound, is found in growing cells of *de*-B18*, and is present in detectable amounts throughout all stages of cell growth. In wildtype cells, trace amounts of IPyA are detected only in the plateau phase. The presence of IPyA in mutant cells may give an indication of alterations in the IAA pathway; IPyA has been reported to be present in maize kernels (Stowe and Thimann, Arch. Biochem. Biophys. 51:499-516, 1954); in our experiments we did not detect it in developing endosperms, either normal or mutant, but we found it in mature seeds (not reported) when biochemical pathways are slowed down or stopped.

Analysis of free substances released by the cells to culture medium confirms the different metabolism between wt and *de*-B18*. IAA is released by wildtype cells, while only IPyA is detected in culture media from mutant cells.

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Biochemical characterization of some *dek* mutants

--M. L. Racchi, M. Sturaro, S. Faranda[1] and L. A. Manzocchi[1]
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The isolation of single gene mutants defective in hormone responses or biosynthetic pathways is a powerful tool to study plant hormones and their role in growth and development. With the aim of isolating hormone mutants in maize we considered mutants affecting seed and embryo development (*dek* mutants) with altered or suppressed germination isolated by means of EMS or X ray mutagenesis (Dolfini et al., Somatic Embryogenesis, IPRA, pp. 122-132, 1985) or somaclonal variation. *dek* mutants have already greatly contributed to the understanding of genetic programs underlying embryogenesis but not all their potential has been exploited. Biochemical characterization of this class of mutants can give further important information on the differentiation process.

We report a characterization of a group of defective endosperm mutants (ed41v, ed42v, ed45v and ed47v from G. Gavazzi's collection) selected on the basis of their different seed morphology and germination capability.

A screening has been done culturing immature and mature mutant embryos as well as the normal counterpart in cytokinine enriched MS media to estimate the response to the hormones in terms of morphogenic capability. Results are presented in Table 1.

Table 1. Development of mutant embryos (mature = M and immature = I) on media without (ER0) or supplemented with cytokinines (ER1 = 0.5 mg/l BAP); (ER2 = 1mg/l kinetin).

	ER0		ER1		ER2	
Mutant	I	M	I	M	I	M
ed41v	43	33	90	0	90	0
ed42v	47	50	59	45	55	45
ed45v	23	36	35	32	19	34
ed47v	6	nd	44	nd	7	nd

*Numbers refer to embryos developing seedling structures as percent of total cultured embryos. Mutant embryos, both immature or mature, are from the same ear.

One of the mutants analyzed (ed41v) showed a positive response to cytokinines. Immature embryos cultured in the presence of 0.5mg/l BAP or 1mg/l kinetin developed into normal fertile plants. The action exerted by the hormone is specific for the embryonic phase; in fact, no response is obtained on mature embryos.

To discriminate between mutants altered mainly in the germination process and those in embryogenesis we have determined on dormant and germinating mutant and wildtype embryos a series of biochemical markers: polypeptide patterns, catalase isozymes (by zymograms on polyacrylamide gels) and malate synthase (by western blot experiments with an antibody against malate synthase). The results obtained allowed us to define *dek* mutants in different classes according to their specific developmental deficiency.

Among the mutants analyzed two are particularly interesting in this regard: ed41v and ed45v. The former, characterized by absence of shoots during germination, gains normal embryo development in the presence of cytokinines. No alterations on the germination process have been observed in terms of differences in polypeptide patterns, catalase 2 or malate synthase expression. It is a developmental mutant blocked in the differentiation process, probably due to a hormonal defect. The latter mutant does not respond to cytokinines and does not show the activation of biochemical pathways related to germination; in fact, no catalase 2 and malate synthase expression has been found. The presence in this mutant of an apparently normal embryo suggests a defect in the control of dormancy.

Spatial expression of structural and regulatory storage-protein genes

--Silvana Faccio Dolfini, Michela Landoni, Chiara Tonelli, L.Bernard[1] and A.Viotti[1]
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The cellular heterogeneity of the endosperm may be related to a differential regulation of gene expression during the development of this tissue. To elucidate this aspect, the spatial expression during the seed formation of genes encoding zeins and glutelins was investigated by in situ hybridization on wildtype lines (W64A and A69Y) and genotypes carrying mutations at loci affecting zein synthesis (*o2*, *o7*, *fl2* and *pro1-1*). Preliminarily, the lines, obtained from various sources and investigated during different harvesting periods, were characterized for their DNA restriction patterns.

The localization of storage-protein gene transcripts was visualized in serial sections of developing seeds using anti-mRNA probes transcribed from sequences representative of genes encoding heavy- (M1) and light-chain (E19 and M6) zeins and glutelins (G1) (Viotti et al., EMBO J. 4:1103, 1985; Prat et al., Gene 52:41, 1987). In normal endosperms the zein and glutelin

mRNAs are expressed in all cells, except for the aleurone layer. However, each mRNA type accumulates at a different level in the various endosperm regions, allowing recognition within the tissue of specific territories of expression for each storage-protein mRNA. The genes of the heavy-chain zeins (M1) show a higher expression in the apical region, while light-chain zein genes (E19 and M6) are more abundant on the abgerminal side. The glutelins show a greater amount of transcripts mainly in the germinal region (Fig. 1).

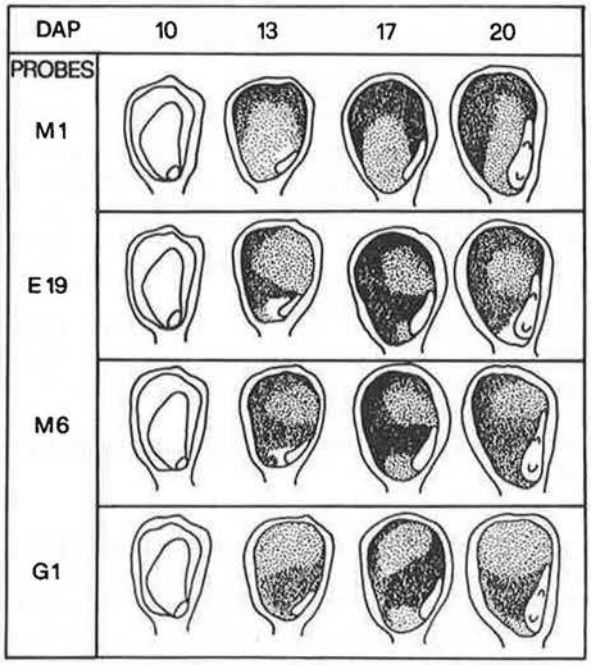

Figure 1. Accumulation of zein and glutelin mRNAs during W64A seed development.

In general, this transcript distribution clearly follows a decreasing gradient from the outer layers of the endosperm to the inner, contrasting the hypothesis that DNA amplification occurring in the central cells is correlated to a higher level of zein synthesis (Kowles and Phillips, Int. Rev. Cytol. 112:97, 1988). These spatial expression patterns, established early for each gene type, are maintained during the subsequent period of seed maturation.

The various probes were also hybridized to sections obtained from different mutants: W64A *o2*, Rossman *o2*, W22 *o7*, W64A *fl2* and W22 *pro1-1*. Each mutant reveals, in comparison with the wildtype, a specific pattern of expression, showing different and profound reductions in the accumulation of zeins and glutelins, in agreement with the hypothesis of a "regional" control of zein gene transcription and in accord with results of Northern analyses. As a consequence, the molecular constitution of protein bodies, as regards the relative proportion of heavy- and light-chain zeins, may be different in the various regions of the endosperm.

The size and abundance in the different genotypes of the transcript of the trans-acting regulatory gene opaque-2 (*O2*) was determined by Northern blotting and its cellular localization was analyzed by in situ hybridization, using as probe the *O2* cDNA (Schmidt et al., P.N.A.S. 87:46, 1990).

Results show that opaque-2 transcript is expressed and localized differently in normal and in various opaque-2 endosperms. In fact, a transcript is present over nucleus and cytoplasm in W64A and A69Y inbred lines, as well as in *o7* and *fl2* mutants (Fig. 2d). The various *o2* mutations analyzed, however, proved to be molecularly different, since a strong hybridization signal is present almost exclusively over nuclei in W64A *o2* (Fig. 2a-c), while only a few grains are visible over nucleus and cytoplasm of Rossman *o2* and A69Y *o2*. These data are supported by Northern results. In particular, the recovery of the transcript of W64A *o2*, having a molecular weight greater than the normal, suggests the absence of a post-transcriptional modification (possibly intron splicing) and correlates with its nuclear localization.

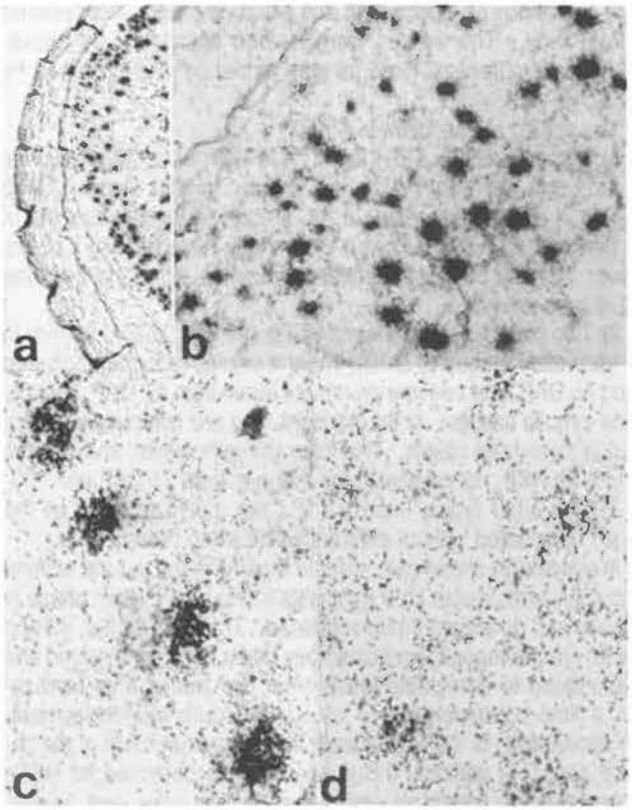

Figure 2. Hybridization of *O2* anti-mRNA probe to (a-c) W64A opaque-2 endosperm at 15 DAP (a x30, b x200, c x400) and (d) W64A endosperm at 15 DAP.

Temporal and spatial expression of HSPs in developing kernels

--Carla Frova and Graziana Taramino

In a previous note (C.Frova, MNL 63) preliminary data on HSP synthesis in post-fertilization stages (7-20 DAP) were reported. Two genotypes were analyzed and different protocols for the analysis tested. Those first data did not reveal significant differences in the HSPs induced between genotypes and developmental stages.

Here we report the results of a second analysis performed on additional developmental stages (3-42 DAP). The reciprocal F1s between two inbred lines provided the material for this analysis. The parental lines differed for the presence/absence of a low molecular weight HSP: P1 = Mo17, 17kDa+; P2 = H95, 17kDa-. The 17kDa HSP thus served as a marker to monitor the expression of the paternally derived gene in the F1 H95 x Mo17.

The procedure was optimized as follows: for very immature stages (3-14 DAP) intact kernels were excised from the cob and incubated at 25 or 41 C for three hours in distilled water containing 150µl/ml 35S methionine. For later stages (21-42 DAP) the

kernels were dissected into embryonic tissues (scutellum+embryo) and pericarp. Endosperm and aleurone were not considered in this study. The tissues were treated separately as above. After the treatment all material was extracted and analyzed by 1-D SDS PAGE.

The results are as follows:

1) In the F1 H95 x Mo17 the 17kDa HSP of paternal origin is absent up to two weeks after fertilization. In later stages (21-42 DAP) it is clearly expressed in the embryonic tissues but not in the pericarp, as expected given the entirely maternal origin of this tissue. Kernels of the reciprocal F1 express this band from the first stages analyzed.

2) Two high molecular weight HSPs (94 and 92kDa) appear in the very early stages (3 DAP), show a drastic reduction between 5 and 15 DAP, and become prominent again from 21 DAP, but only in the zygotic tissues. In the pericarp they are completely absent even in these later stages. This latter tissue however, shows a minor 35kDa HSP, not present in the embryo, from 21 DAP on.

3) The other typical maize HSPs are synthesized at all the stages considered without differences between tissues.

In conclusion, these data indicate that, as is the case in several animal systems, maize HSP synthesis in the post-zygotic phase is spatially and developmentally regulated. Three HSPs, 94, 92 and 17kDa, show temporal regulation: they begin to be synthesized and accumulated to detectable levels from two weeks after fertilization, a time corresponding to very active embryo development. The appearance of the 17kDa band indicates expression of the paternal gene. In addition, tissue specificity is observed for HSPs 94 and 92kDa, typical of embryonic tissues only, and 35kDa, apparently induced only in the pericarp.

In vivo studies of the effect of heat-shock treatments at different stages of kernel development, in particular the early ones characterized by the absence of some HSPs, are in program.

β-Glucosidase activity in seedling tissues is reduced by exposure to light

--Carla Frova

β-glucosidase is an enzyme present in several seedling and adult plant tissues. In dark grown seedlings the strongest activity was found in coleoptiles and mesocotyls (Frova, unpublished). However, accidental exposure to light resulted in a reduction of the enzymatic activity in both tissues. Here the results of a first analysis of this phenomenon are reported.

Genetic variability of the response to light. Seeds of 10 inbred lines and 3 F1s were germinated for 24h in the dark and thereafter either kept in the dark or exposed to continuous white light. Five days after germination coleoptiles and mesocotyls were collected from the seedlings, extracted and analyzed spectrophotometrically (Esen and Cockmus, Biochem. Genet. 28:319-336, 1990) for β-glucosidase activity. Exposure to light resulted in reduction of enzymatic activity in all cases in both tissues, but pronounced differences between genotypes were found. In particular, variability in reduction was larger in coleoptiles (10-85%) than in mesocotyls (25-65%).

Among the genotypes tested B77 showed the strongest response to light: 85% reduction in the coleoptile and 55% in the mesocotyl. This line was therefore used for further analyses.

Effect of light pulses. In order to test i) whether the light inhibitory effect is transient or irreversible, and ii) what seedling developmental stages are most responsive to the treatment, seeds or seedlings were exposed to white light for 24h at different times during seedling development (0-24, 24-48, 48-72, 72-96, 96-120h after imbibition) and then put back in the dark until day 8. β-Glu activity was measured each day in each treatment. The data show that light is totally ineffective when given in the first 48 hours. Subsequent pulses, 48-72, 72-96, 96-120, drastically reduce β-glucosidase activity, which in no case is restored upon resumption of the dark condition. By day 7 the enzymatic activity declines to low levels also in the dark controls in both tissues tested.

Shorter exposures, 12h pulses, yield a similar effect, although enzymatic inhibition is somewhat weaker.

Effect of different light qualities. The seedlings were germinated in the dark and exposed for 24h, between 48 and 72h after germination, to white, red, far red and blue light. In all cases an inhibitory effect was detected. No significant differences between light qualities were observed. The reduction in β-glu activity was nearly 85% in coleoptiles and 55% in mesocotyls in each treatment. These data suggest the possible implication of more than one photoreceptor in the response.

Experiments to test the effect of different light intensities in both seedling and adult plant tissues are underway, and crosses for a genetic analysis of the phenomenon have been planned.

The parental allelic ratio determines the DNA modification of the zein genes

--G. Lund and A. Viotti

The methylation state of the zein genes is presumed to correlate with the expression of these genes; i.e. the genes are heavily methylated in the embryo and demethylated in the endosperm (Bianchi and Viotti, Plant Mol. Biol. 11:203-214, 1988). We have investigated whether these genes are subjected to genomic imprinting.

Genomic DNA extracted from embryo and endosperm of the three inbred lines A69Y, W22 and W64A, and the reciprocal crosses between all three inbred lines, was digested with the methylation sensitive enzymes *Pvu*II and *Hha*I, followed by Southern blotting and hybridization to cDNA and genomic clones of the heavy- and light-chain genes zA1 and zE19.

The results show that the family of zein genes belonging to zA1 and zE19 are demethylated in the endosperm, and that the pattern of demethylation is dependent only on the contribution of the maternal genome. Furthermore, the pattern of demethylation is a conserved feature of the investigated lines, as identical patterns are observed between DNA from different maize harvests. The CpG- and CpNpG- methylation does not have a differential role as far as genomic imprinting is concerned, as identical results were obtained from the digestions of DNA using *Pvu*II and *Hha*I, which recognize CpNpG- and CpG sequences respectively.

Transformation of endosperm cell suspension cultures and immature seeds by high-velocity microprojectile bombardment

--A. Genga, L. A. Manzocchi, S. Faranda and A. Viotti

The analysis concerning the expression and regulation of tissue-specific sequences from maize has in the majority of cases been performed in heterologous systems, due to difficulties in setting up easy and routine methods of maize transformation. However, high-velocity microprojectile bombardment has recently proved to be a successful method in obtaining both transient and

stable transformation of this species.

In order to develop an experimental protocol for the study of zein gene expression, a particle gun apparatus, based on an electric device that allows controlled flow from a gas cylinder, has been constructed in our laboratory, and transformation experiments have been carried out using two different types of targets: endosperm cell suspension cultures and immature seeds.

Cell suspension cultures, established from immature endosperms of an A69Y maize line, appear to be a valuable homologous system, since these cells synthesize and accumulate zein polypeptides during their growth cycle (Manzocchi, Plant Cell Reports 9:555-558, 1991).

Immature seeds of the W22 maize line were harvested at 20 DAP and immediately subjected to bombardment. The target was represented either by the pericarp (intact seeds) or by the endosperm tissue directly exposed, after removal of the pericarp, to DNA-coated gold particles.

The target surface area was about 5 square centimeters. Cells from suspension cultures were layered on sterilized filter paper and maize seeds were placed on agar plates.

In order to optimize the conditions of the bombardment the two plasmid constructs DP74 and DP33 were used. Both plasmids contained the promoter-leader region of the 35S-CaMV fused to the 5' end of the GUS gene, and differed by the presence (DP74) or the absence (DP33) of the maize Adh1 intron (Pierce et al., Plant Gene Systems and Their Biology, Alan R. Liss, Inc., pp. 301-310, 1987). For the functional analysis of endosperm specific sequences we employed the entire promoter region (1450bp upstream to the ATG start codon) or derived fragments containing different regulatory elements of the zE19 clone, a light chain zein gene (Spena et al., J. Mol. Biol. 169:799-811, 1983).

Transient expression of the GUS activity was determined by the histochemical method (Jefferson et al., EMBO J. 6:3901-3907, 1987), usually 24h after bombardment.

The 35S-GUS constructs gave a higher number of blue spots (GUS expression unit, GEU) on entire seeds (pericarp tissue subjected to bombardment) than on the exposed endosperm tissue. The average GEU values for pericarp and endosperm tissue were about 90 and 15 per shot, respectively. The endosperm cell suspension cultures showed with the same plasmids an average GEU value of 302±111 spots per shot.

Using the zE19 construct, suspension cultured cells did not show any GUS expression. However, preliminary results of seeds bombarded with the zein E19 promoter indicate a low, but significant, GEU value (up to 20 spots) only on the exposed endosperm tissue, but not on seeds with pericarp.

Functional and molecular analysis of several mutations of the *o2* locus

--L. Bernard, P. Ciceri, B. Lazzari and A. Viotti

The maize *o2* locus codes for a DNA-binding protein which activates the transcription of a group of zein genes (Schmidt et al., Proc. Natl. Acad. Sci. 87:46-50, 1990). This protein belongs to the b-Zip family of transcriptional activators, like the jun and fos mammalian oncogenes, the yeast GCN4 and the plant TGAI and HBPI (Tabata et al., EMBO J. 10:1459-1467, 1991; Busch and Sassone-Corsi, Trends Genet. 6:36-40, 1990; Katagiri et al., Nature 340:727-730, 1989). The activation is determined by the binding to short target sequences (Lohmer et al., EMBO J. 10:617-624, 1991; Hoscheck and Schmidt, unpublished) upstream to the CAAT and the TATA boxes, which are present in almost all the high molecular weight zein genes (H) and usually absent in the low molecular weight genes (L).

The hybridization of Southern and Northern filters with *O2* probes (kindly provided by R. Schmidt) showed a high level of heterogeneity in a number of *o2* maize lines for what concerns the DNA restriction pattern and the type and level of transcription products.

The Northern results are supported by the Western analysis of total endosperm proteins (carried out with *O2* antibodies), where the different *o2* lines show the absence or the presence of *O2* modified polypeptides (Fig. 1). It has in fact been possible to

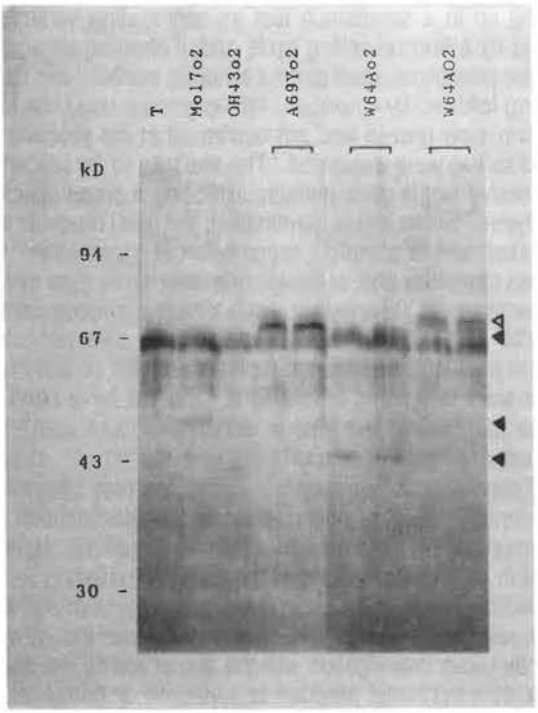

Figure 1. Western blot of several maize lines using a polyclonal *O2* antibody. T = total protein extract from tobacco leaves identifying the antibody cross-reactions bands. Δ = *O2* double band. ▲ = truncated *O2* proteins.

identify a double band in all the examined wildtype lines (W22, A69Y, NYR and W64A) at the apparent *O2* wildtype molecular weight region (68 and 69kDa), that may represent products of post-translational modification (e.g. phosphorylation or O-glycosylation) of the same protein. These two bands are always absent in the *o2* mutations (W22 *o2*, A69Y *o2*, NYR *o2* and W64A *o2*) where we can detect *O2* transcripts and notice the presence of a number of specific bands of different molecular weight which probably are truncated or unmodified *O2* polypeptides. In the two lines OH43 *o2* and Rossman *o2* that are null-transcript, there is no *O2* specific extra-band and they can be considered as negative controls.

The timing analysis of *O2* expression during seed development (at 14, 20 and 25 DAP) shows, on the basis of the same amount of total RNA or proteins, a maximum of transcription and translation at 14 DAP. Furthermore, it has been observed by Northern analysis and SDS-PAGE that the various *o2* mutations differentially affect the transcription and the product level of both the H and L zein genes.

These results suggest that the *O2* mutated proteins may

specifically affect the expression of the various subsets of the zein multigene family, according to the kind of modification they encounter.

Developmental studies on seed germination and seedling formation
--G. Gavazzi and G. Todesco

We have isolated several mutants impairing germination or early seedling development with the aim of using them as a tool to elucidate the genetic program accounting for seedling development. Some of these mutants were obtained following chemical mutagenesis, while the majority were isolated in the selfed progeny of stocks carrying *Ac* or *Mu* outcrossed to different lines. Those showing up in a sandbench test as segregating variants were retested by a second selfing cycle and, if showing up again with the same phenotype, were given a tentative symbol (*des*: defective seedling) followed by a number. This way many bona fide mutants exhibiting slow growth and not confirmed in the progeny of the second selfing were discarded. The mutants so far identified are all recessive single gene mutants exhibiting a broad spectrum of phenotypes. Some impair germination; the most dramatic ones in their effect lead to complete suppression of germination. Others suppress dormancy and, at least in one case, show clear symptoms of water stress in the seedling, while others are recognizable two days after germination or later because of the enlarged coleoptile and primary root, an abnormal (open) coleoptile or leaf morphology, or clear symptoms of dwarfism. The last have been tested for their response to exogenous gibberellins (GA4 +GA7 10^{-5}M) and crossed inter se or with known *d* mutants (*d1*, *d2*, *d3* and *d5*). The allelism test has almost been completed (see MNL 64:86-87 for preliminary results) and has led to the identification of five GA responding mutants not attributable to any of the *d* genes described in the literature. One of the mutants exhibiting an abnormal seedling development (*des17*) has been characterized, and the results are presented in the next note, while the others are presently under investigation with the aim of linking the disruption in their developmental program to metabolic or hormonal unbalance.

Characterization of *des17*
--M. Galbiati and G. Gavazzi

This mutant, recognizable at an early stage of germination, was isolated in the progeny of a *Mu* line, kindly provided by Dr. Robertson, crossed to homozygous TB-8Lc males (from Dr. Beckett). It is easily detected on the basis of reduced height, partial suppression of primary root growth, failure of the coleoptile to thoroughly enclose the emerging seedling and contorted leaf morphology. Segregation ratios, as determined by selfing +/*des17* heterozygotes, show a significant shortage of recessives over the expected one-quarter (116/754; $X^2 = 37.18$ $P < 0.001$). If germinations are performed after separating seeds originating from different sectors of the heterozygous ear, the segregation ratios observed (see Table 1) are different and best explained by assuming active expression of *des17* in the male gametophyte.

Table 1. Mutant segregation in the apical, central and basal region of selfed +/*des17* ears.

Cod. No. of selfed ear	Region	No. seedlings	Mutants (%)
6291 (9)	apical	152	15
	central	158	9
	basal	141	10
6291 (11)	apical	77	16
	central	78	10
	basal	68	7

Mutant seedlings grown in pots die within the first 60 days. The first attempts to repair the abnormal phenotype by growing homozygous embryos on media supplemented with the main plant growth regulators gave negative results, while some degree of phenotypic repair was observed by growth on mineral Murashige and Skoog (MS) media enriched with casein hydrolisate (200 mg/l), yeast extract and yeast hydrolisate (40mg/l each). When the organic components were added separately to the mineral M and S medium the results reported in Table 2 were obtained.

Table 2. Effect of the addition of different organic components to the basic (MS) medium on the growth of homozygous *des17* mutants as estimated after 7 days of culture. Ht and LA refer to average seedling height and primary root length in cm respectively. Each determination is the average of 5-10 single observations.

Organic component	Ht	LA
none	7.3	1.0
casein hydrolisate	7.8	2.1
yeast extract	6.7	2.9
yeast hydrolisate	11.0	5.2

We are presently testing the effect of single nitrogen bases on growth of detached mutant roots and embryos with the intent of correlating the *des17* phenotype with a specific metabolic requirement. The positive response of the mutant to yeast hydrolisate allows us to attempt its rescue. We succeeded in growing homozygous *des17* plants up to maturity by continuous spraying with yeast hydrolisate solution but failed to reproduce them because of their failure to develop a tassel.

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Esterase organ-specific pattern revisited
--Emil E. Khavkin and M. V. Zabrodina

Previous studies of somaclones (MNL 64:91, 1990; 65:88, 1991) led to the suggestion that several, presumably organ-specific, isozyme loci might vary more often than those expressed at all developmental stages. To test this assumption, we correlated our isozyme patterns obtained by neutral (modified Taber and Sherman) and alkaline (Davis) vertical slab PAGE to esterase loci assigned earlier upon starch gel electrophoresis.

Two cathodic esterases slowly migrating in the neutral gel (Fig. 1) were apparently the E3 and E1 isozymes described by Schwartz (PNAS 51:602, 1964). The former locus was prominent in the endosperm, weakly expressed in the scutellum and obviously absent from the embryo axis. Several public inbreds employed in this study had slow (Tx601) and fast (predominant) forms of E3 esterase. E1 esterase was most active in the endosperm, quite discernible in the axis and absent from the scutellum. There were two additional lightly (perhaps non-specifically) stained diffuse zones in cathodic zymograms: one immediately preceding E1 was found in leaves while another, with higher mobility, was characteristic of roots. These zones were not identified. The slowest zone in the alkaline gel system (Fig. 2), found only in the endosperm, might be formed by fused E1 plus E3 bands.

Several specific loci could be easily identified among anodic esterases, especially in neutral gels. The most polymorphic, the E4 series described by Harris (MNL 40:53, 1966) and Macdonald and Brewbaker (Hawaii Agr. Exp. Sta. Tech. Bull. No. 89, 1975), was very active in roots (up to 7 bands were excellently resolved in

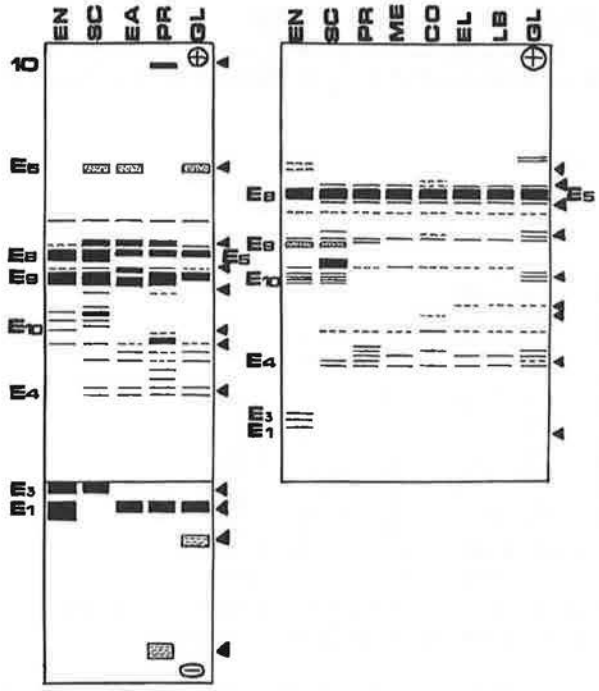

Figure 1. (left) Cathodic and anodic esterases in the neutral (modified Taber and Sherman) PAGE system. *E1* to *E10* are esterase loci and 10 is band 10 as described by Macdonald and Brewbaker (1975). EN, endosperm; SC, scutellum; EA, embryo axis; PR, primary root; GL, green leaf. Arrowheads show qualitative and quantitative organ-specific changes of esterase patterns.

Figure 2. (right) Anodic esterases in the alkaline (Davis) PAGE system. ME, mesocotyl; CO, coleoptile; EL, etiolated leaf; LB, leaf base. Other symbols as in Figure 1.

several inbreds), evident in other embryo tissues and green leaves, but completely absent from the endosperm. The quantitative changes in E4 expression in embryo tissues might at least account for some of the previously described effects of somaclonal variability. In roots at least five *E4* alleles were found (in the order of their decreasing mobility towards anode): 1) A188; 2) A239; 3) B14, C103, Tx601, Va35 and W64A; 4) B73 and Oh43; and 5) B37.

Allelic forms of E9 esterase were readily discerned in neutral gels with fast isozyme in Va35 (and maybe also in Tx601) and one or two slower allelomorphs in other inbreds. The E8 band was easily identified in alkaline gels by its position and intensive staining in all tissues, however, three allelic forms corresponding to those reported by Stuber and Goodman (USDA Agr. Res. Serv., Southern Ser., No. 16, 1983) were difficult to discriminate by their mobility. In neutral gels, E9 and E8 bands were preceded by their "twins". In several inbreds these twins were as prominent as E9 and E8 isozymes, except in the endosperm and leaves. As these bands coincided with zones singled out as presumable targets of somaclonal variation, they need further clarification. At present they might be cautiously related to the *E5* locus and 42 and 43 bands described by Macdonald and Brewbaker (1975). In alkaline gels one of these bands preceded the E9 isozyme while two other zones were more compact and adjacent to the E8 major isozyme.

Somaclonal variations were also manifest in the interval between the E4 and E8 zones, and here several minor and apparently organ-specific bands were observed. A well-resolvable series of at least four bands in alkaline and neutral gels was characteristic of endosperm and scutellum and absent from axial tissues. It might belong to the *E10* locus described by Macdonald and Brewbaker (1975), which has at least two alleles. Some bands were present only in the scutellum, while most of the electromorphs in this zone were absent from the endosperm and found, with varying staining activity, in some or all embryo tissues. Staining activity of some of the leaf-specific bands depended on greening.

Two additional organ-specific zones migrated ahead of the E8 band in the neutral gel. One of them, a diffuse band that was absent from endosperm and root but present in scutellum and shoot, might be related to the E6 esterase of Macdonald and Brewbaker (1975). A fast allelomorph was predominant and a slow variant was found in A239. Another isozyme twice as mobile as the E8 band apparently corresponded to band 10 as described by these authors in partially senescent roots. The inbreds greatly differed by activity and mobility of this isozyme: there were fast (Tx601 and WF9), intermediate (predominant) and slow (A239) variants.

S1 plasmid protein product synthesized in *E. coli* possesses a DNA polymerase activity

--Evgeny V. Kuzmin and Tatiana Yu. Scherban

S-plasmids are autonomous genetic elements found in mitochondria of cms-S maize. Their organization as linear DNA molecules with terminal inverted repeats and 5'-termini covalently attached to protein points to the similarity with viruses. This fact implies the existence of S-plasmid-encoded proteins that potentiate their replication and transcription in a specific viral-like mode. Indeed, analysis of published sequences of S-plasmids has allowed us to identify the putative protein products of two of the largest genes (ORF3 and ORF1) as a viral type DNA polymerase and a single-subunit RNA polymerase, respectively (Kuzmin and Levchenko, NAR 15:6758; Kuzmin et al., NAR 16:4177).

We have constructed recombinant plasmids where both above-mentioned S-plasmid ORFs were placed under the control of bacterial tac-promoter and SD-sequence from vector pTTQ18/19. These procedures have resulted in the addition of 10 and 12 extra codons to the N-termini of ORF3 and ORF1, respectively. Expression of both S-plasmid-specific proteins in *E. coli* has proved to be poorly reproducible and extremely strain-specific, possibly because of deleterious effects of these proteins on *E. coli* cells. Finally we have succeeded in an optimization of ORF3 expression in *E. coli* resulting in reproducible synthesis of 105kDa protein visible after Coomassie staining of total *E. coli* protein extracts. The objective of our study was to demonstrate the preservation of native features and DNA polymerase activity of the 105kDa protein synthesized by *E. coli*.

E. coli cultures harbouring plasmid pQF3 (ORF3 derivative) and plasmid pQ5 (ORF1 derivative) were processed in parallel (the latter culture served as control) by the following procedure: the cells were lysed with lysozyme, sonicated, the extract was clarified by centrifugation; DNA-binding proteins were precipitated with streptomycin sulfate, solubilized and further precipitated by ammonia sulfate (50% sat.); then the preparation was brought to 0.3M NH_4Cl and applied to Cibacron blue-agarose, the

Specific DNA polymerase activities of protein fractions eluted from blue-agarose (dpm/mkg protein):

	E. coli CA161[pQ5]	*E. coli* CA161[pQF3]
0.3M NH_4Cl (flow through)	3400	4500
0.6M NH_4Cl	500	15000
1.5M NH_4Cl	1300	24700

column was subsequently eluted with 0.6M and 1.5M NH_4Cl, and DNA polymerase activity of corresponding fractions was determined.

Electrophoretic analyses (not shown) have proved quantitative precipitation of 105kDa protein by streptomycin, its absence in 0.3M eluate and maximal presence in 1.5M eluate. It can be seen that *E. coli* cells harbouring expressible ORF3 produce a novel DNA polymerase with considerably higher affinity to Cibacron blue than DNA polymerase I of host cells. This is characteristic of α-type DNA polymerases which ORF3 putative protein belongs to according to its primary structure. Whether or not S-plasmid mRNAs are edited in maize mitochondria remains an open question. Nevertheless absence of this editing in the *E. coli* system apparently does not lead to severe changes in the ORF3 protein sequence that abolish its DNA polymerase activity. Further studies of enzymatic properties of ORF3 protein from recombinant *E. coli* and from maize mitochondria are now in progress.

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Use of tissue culture to test plant resistance to abiotic stresses

--Y. I. Dolgykh, S. N. Larina and Z. B. Shamina

The possibility of testing plant tolerance to abiotic stresses using maize tissue culture was examined.

All plant material was obtained from Dr. V. S. Sherbak (Krasnodar Research Institute of Agriculture). Seeds of four maize inbreds which differed in drought resistance were germinated in NaCl (2%) or PEG (25%) solutions, the number of germinated seeds was counted and the size of shoots and roots was measured. Leaves of 2-week-old seedlings were used to determine the ratio of physiologically bounded and free water. Embryo-produced calli of these lines were grown on Murashige-Skoog medium supplemented with 0.5-2.5% NaCl or 5-30% PEG.

Callus growth on PEG-containing media and energy of seed germination under osmotic stress were found to be similar for each of the genotypes (Fig. 1). Distribution of inbreds according to their water-bounding capacity and callus sensitivity was the same with the exception of line Gn118. This line is likely to have other mechanisms of drought resistance.

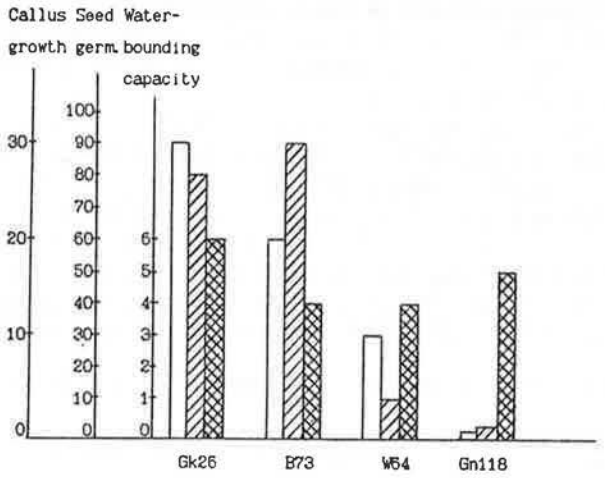

Fig. 1. Callus growth activity, energy of seed germination on PEG (% to control) and water-bounding capacity of maize inbreds.

Tested inbreds did not show significant difference in callus growth and energy of seed germination under NaCl stress (Fig. 2).

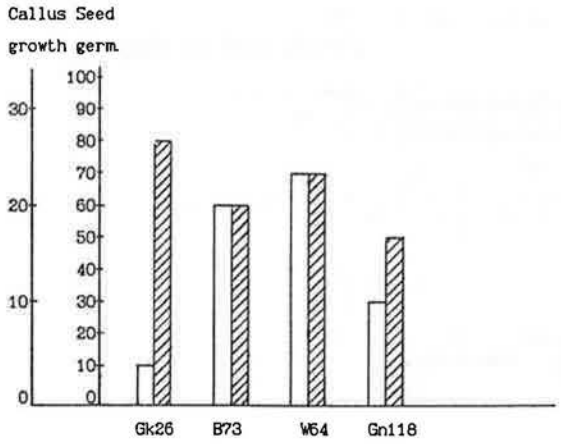

Fig. 2. Callus growth activity and energy of seed germination on NaCl (% to control) of maize inbreds.

The sensitivity of seeds and callus culture to NaCl could not be related to their sensitivity to PEG--the genotypes most tolerant to NaCl could be the most sensitive to PEG. Salt tolerance of tested inbreds is apparently determined by resistance to toxic effect of ions, but not to osmotic stress.

The fact that stress sensitivity of callus was positively correlated with stress sensitivity of intact plants allows employment of tissue culture for screening salt and drought resistant variants.

Several tolerant cell lines were selected from 8 inbreds and hybrids of maize on media supplemented with 1.5% NaCl or 25% PEG. Callus viability on NaCl medium was 20-30% and did not depend on genotype. Callus viability on PEG medium fluctuated from 7 to 76% for different genotypes. 167 PEG-tolerant plants, 69 NaCl-tolerant plants and 11 plants tolerant to both factors were regenerated from selected calli. Field resistance of these plants will be tested in 1992.

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IARI

B chromosomes and abnormal chromosome 10 (K10) in northeastern Himalayan maize

--M. Kumar and J. K. S. Sachan

The presence of B chromosomes in certain maize stocks is of worldwide distribution. B chromosomes have been found floating in the population due to non-disjunction and preferential segregation.

In contrast to the wide distribution of the abnormal chromosome 10 (K10) in different American races, it was reported to be completely absent from Asia and Europe. However, recently the presence of this abnormal chromosome 10 in higher frequency in local maize of Kashmir, India, has been reported (Jotshi and Patel, MNL 58, 1984).

Altogether, 66 maize collections from different areas of the northeastern Himalayas, including Arunanchal Pradesh, Assam, Meghalaya, Nagaland, Tripura, Sikkim, W. Bengal and Nepal, were

screened for the presence of B chromosomes and abnormal chromosome 10. Emerging tassels were fixed in a mixture of ethanol-glacial acetic acid (3:1) and were stored at 4±1 C. PMCs were analysed at pachytene for the presence of K10 and at diplotene-diakinesis for B chromosomes. Further later stages were observed for studying the behaviour of B chromosomes during metaphase I and anaphase I.

B chromosomes were found in 9 out of 66 collections studied. The collections possessing B chromosomes include two from Assam, three each from Sikkim and Nagaland and one from Tripura (Table 1). Collections from Assam (AS-59, AS-60) and Sikkim (S-21, S-24 and S-59) had 1-2 B's. Nagaland collections (N-24, N-29 and N-44) had 1-3 B's and the lone Tripura collection possessing B chromosomes (2 B's) was T-17. On the other hand, B chromosomes were found to be totally absent in collections from Arunanchal Pradesh, West Bengal, Meghalaya and Nepal (Table 1).

Table 1. Distribution of abnormal chromosome 10 (K10) and B chromosomes in NEH maize.

Region	No. of collections observed	No. of plants analysed	Total collections with K10	Total collections with B's	Total plants with B's	Average B's/plant
Assam	3	15	-	2	8	1.25
Arunanchal Pradesh	2	10	-	-	-	-
West Bengal	1	4	-	-	-	-
Meghalaya	15	73	-	-	-	-
Nagaland	13	62	1	3	9	1.33
Sikkim	18	87	-	3	11	1.18
Tripura	13	64	1	1	3	2.00

The presence of B chromosomes in Indian maize has been reported by some earlier workers also (Jotshi and Patel, 1984; Sarma and Sharma, 1986; Venkateswarlu, 1965). However, there is still only one report of B chromosomes in a maize collection from the Naga Tribe (Venkateswarlu, MNL 39, 1965).

The presence of an abnormal chromosome 10 (K10-I) was a rare event in NEH maize collections, being present in only two collections, namely N-37 and T-17 from Nagaland and Tripura respectively.

Thus, varying frequency of B chromosomes was observed in NEH maize strains, and its geographical distribution was not as limited as that of K10. This is the first report of the presence of K10 in NEH maize collections.

Lineages of 'Sikkim Primitives' (SP)

--M. Kumar and J. K. S. Sachan

Cytological characterisation of eight SP maize strains collected from different parts of the NEH region was done. These included three strains from Meghalaya (M-1, M-15, M-25), two from Tripura (T-2, T-26), two from Sikkim (S-27, S-44) and one from eastern Nepal (SP Nepal).

Pachytene analysis of the PMCs of these SP strains fixed in aceto-alcohol (3:1) was done to study the knob composition of these strains, and later on they were compared with the standard knob composition of American primitive races given by Kato (1965).

Altogether, 16 knob forming positions were identified on different chromosomes (Table 1). The minimum knob number was in the M-25 and T-26 collections, whereas T-2 from Tripura had the highest knob number (11). The most frequent knob forming positions in SP strains were 2L, 4L, 6S, 8La and 9ST. Less frequent knob forming positions were 1ST, 3ST, 6Lb and 9L. Knobs were rarely present at 2S, 3L, 5L, 6LC, 7ST, 7L and 8Lb positions. Chromosome 10 was completely knobless in all SP strains. Generally speaking, SP strains from Nepal and Sikkim possessed mostly large knobs in comparison to those of Meghalaya and Tripura.

A terminal knob on the short arm (1ST) of chromosome 1 was present in two Meghalaya collections, out of which M-1 possessed a large knob whereas M-15 had a medium sized knob at this position. An interstitial knob on the short arm of chromosome 2 at the 2S position was missing in all of the SP collections, except one large knob at this position in S-27. An interstitial knob on the long arm of chromosome 2 at the 2L position was very frequent in occurrence, being present in about 75% of the SP strains. The SP collection from Nepal possessed a very large 2La knob, whereas those of the Meghalaya and Tripura collections possessed a large to medium sized knob. A knob on the 3ST position was present in only 2 out of eight collections, namely S-44 and SP (Nepal), whereas a knob on the long arm was present in the SP (Nepal) collection only. The Nepal collection possessed a large sized knob on both arms of chromosome 3, whereas S-44, from Sikkim, possessed a medium sized knob on the short arm only.

A knob at the 4L position was present in all but M-25 SP strains. A large cylindrical knob was present in the Nepal collection, whereas a medium sized round knob was present in S-27 and S-44 from Sikkim, and T-2 and T-26 from Tripura. The Meghalaya collections M-1 and M-15 possessed a large sized 4L knob.

A medium sized 5L knob was present in T-2 only. A large or medium sized knob at the 6S position near NOR was invariably present in all the collections studied.

An interstitial knob on the long arm of chromosome 6 at the 6Lb position was present in only three collections, namely SP (Nepal), T-2 and T-26. T-2 possessed one more small knob on the 6La position also. This 6La knob was absent in all other chromosomes.

A terminal knob on the short arm of chromosome 7 at the 7ST

Table 1. Knob composition in 'Sikkim Primitives'.

		Knob positions																
Collections	Total knob no.	1ST	2S	2La	3ST	3L	4L	5L	6S	6Lb	6Lc	7ST	7L	8La	8Lb	9ST	9La
M-1	6	+	-	+	-	-	+	-	+	-	-	-	+	-	-	+	-
M-15	6	+	-	+	-	-	+	-	+	-	-	-	-	+	-	+	-
M-25	4	-	-	-	-	-	-	-	+	-	-	-	+	+	-	+	-
S-27	8	-	+	+	-	-	+	-	+	-	-	-	+	+	-	+	+
S-44	6	-	-	+	+	-	+	-	+	-	-	-	-	+	-	+	-
SP (Nepal)	8	-	-	+	+	+	+	-	+	+	-	+	-	-	-	+	-
T-2	11	-	-	+	-	-	+	+	+	+	+	-	+	+	+	+	+
T-26	4	-	-	-	-	-	+	-	+	+	-	-	-	+	-	-	-
Frequency out of 8 collections		2	1	6	2	1	7	1	8	3	1	1	4	6	1	7	2
% frequency of knobs		25.0	12.5	75.0	25.0	12.5	87.5	100.0	37.5	12.5	12.5	12.5	50.0	75.0	12.5	87.5	25.0

1, 2 . . . 9 = chromosomes; S, L = short and long arm of the chromosomes; T = terminal knob positions; b, c = knob positions on the same chromosomes; + = presence of knob; - = absence of knob

position was absent in all collections except that of a small knob in SP (Nepal). However, 50% of the collections studied possessed a knob at the 7L position. The knob at this position was mostly large, cylindrical or round.

A knob at the 8La position was present in 6 out of 8 collections studied, being absent in M-1 and SP (Nepal) collections. The Meghalaya collections had a medium sized knob at this position, whereas the Sikkim collections, as well as T-2 from Tripura, had a large knob, but T-26 possessed a small knob.

A terminal knob at the 9ST position on the short arm of chromosome 9 was present in all except T-26. This 9ST knob was mostly large cylindrical/conical, but was of medium size in S-44. The interstitial knob at the 9La position was present in two collections only, T-2 having a large sized knob whereas a medium sized knob was present in T-2.

On the basis of knob number, these SP strains can be grouped into two categories: a) low knob number group (4 knobs) comprised of M-25 and T-26, possessing common knobs at 6S and 8La, in addition to some other knobs at the 4L, 7L and 9ST positions; and b) high knob number group (6-11) comprised of M-1, M-15, S-27, S-44, SP (Nepal) and T-2. Frequent knob positions in the second group are 2L, 4L, 6S, 6Lb, ,8La, 9ST, whereas knobs are present with lower frequency on 1ST, 2S, 3ST, 3L, 5L, 6Lc, 7ST, 7L, 8Lb and 9L.

When these two SP groups are compared with those of American primitive races, the low knob number SP group appears to have its linkage from Confite Morocho of Peru and/or Palomero Toluqueno of Mexico. On the other hand, the high knob number group of SP strains possesses close similarity to the Nal-Tel-Chapalote complex.

It appears that SP maize still harbours some genes of teosinte, which were introgressed into it much before its prehistoric introduction into the NEH region. These teosinte genes appear to be responsible for much delayed flowering, prolificacy, sturdier and longer stem, and inclusion of a male spike at the tip of the female inflorescence. Such a contention gets support from the maize X teosinte crosses currently being carried out in our laboratory (Ramesha, 1991, personal communication), in which F1 hybrids show most of the characters of SP strains. Hence Beadle's assertion (e.f. Sachan and Sarkar, 1986) that SP maize can be resynthesised appears to hold true.

Knobs in northeastern Himalayan maize

--M. Kumar and J. K. S. Sachan

Knob composition of 50 maize collections from different areas of the northeastern Himalayas (NEH) was studied through pachytene analysis of PMCs. Emerging tassels of these strains were fixed in glacial acetic acid-ethanol (1:3) during 1989 and 1990 at the Division of Genetics, IARI, New Delhi. These maize collections belonged to Assam (3), Arunanchal Pradesh (2), Meghalaya (15), Nagaland (12), Sikkim (18) and Tripura (10). Pachytene analysis of PMCs was done by making temporary aceto-carmine squash preparations. Camera lucida drawings of individual chromosomes were made and these were identified on the basis of length, arm ratio, position of knobs and prominent chromomeres and pattern of centromeric heterochromatin. Knob positions were identified on the basis of standard idiogram given by Longley and Kato (1965).

Twenty-six knob forming positions have been identified altogether in different collections of NEH maize. Mean knob number, range and total knob forming positions of these collections have been shown in Table 1. Frequency of occurrence of various knobs in different regions of NEH has been shown in Table 2.

Table 1. Mean knob number, range, total knobs and common knob forming positions in NEH maize.

Region	No. of collections	Mean knob no.	Range	Total knob positions	Common knob forming position
Arunanchal Pradesh	2	8.5	7-10	11	3L,4L,6S,6Lb,8La,9ST
Assam	3	6.7	6-8	9	2La,4L,6S,6Lb, 7L,8La,9ST
Meghalaya	15	5.7	4-8	16	2La,4L,6S,6Lb, 7L,8La,9ST
Nagaland	10	7.1	5-9	18	2La,4L,6S,6Lb, 8La,9ST
Sikkim	18	5.8	4-9	18	2La,4L,6S,8La, 9ST
Tripura	10	7.3	4-11	20	2La,4L,6S,6Lb, 8La,9ST

Table 2. Frequency in percent of knbos at each knob forming position in various regions of NEH.

Knob positions	Assam	Arunanchal	Meghalaya	Nagaland	Sikkim	Tripura	Overall frequency
1Sa	-	50.00	6.70	7.68	11.10	7.68	9.20
1ST	-	-	20.00	-	-	-	4.60
1La	-	50.00	6.70	15.37	22.20	15.37	15.38
1Lb	-	-	-	-	-	7.68	1.53
2S	-	-	-	7.68	5.50	7.68	4.60
2La	100.00	50.00	60.00	84.60	55.60	76.92	69.23
2Lb	-	-	-	-	-	7.68	1.53
2LT	-	-	6.70	-	-	-	1.53
3ST	-	-	-	-	22.22	15.38	10.76
3L	-	100.00	20.00	23.07	38.90	30.77	32.07
4S	-	-	-	-	5.50	-	1.53
4L	66.66	100.00	60.00	69.23	66.70	84.61	72.30
5L	33.33	-	6.70	-	11.10	30.77	13.84
6S	100.00	100.00	100.00	100.00	100.00	100.00	100.00
6La	-	-	6.70	-	-	-	1.53
6Lb	66.60	100.00	46.70	76.92	22.22	92.30	58.46
6Lc	-	-	20.00	23.07	16.70	23.07	18.46
7ST	33.33	50.00	-	23.07	5.50	15.38	13.84
7L	100.00	50.00	46.70	61.53	33.33	46.15	47.69
8La	100.00	100.00	86.60	76.90	66.70	61.53	75.38
8Lb	-	-	6.70	37.69	5.50	7.68	12.30
9ST	66.66	100.00	80.00	84.60	77.80	92.30	84.61
9La	-	-	-	15.37	16.60	7.68	9.02
9Lb	-	-	-	7.68	-	-	1.53
10La	-	-	-	7.68	-	-	1.53
K10	-	-	-	7.68	-	7.68	3.07
Total knob position	9	11	16	18	18	20	26

1, 2 . . . 10 = chromosomes; K10 = abnormal chromosome 10; S = short arm; L = long arm; T = terminal knob position; a, b, c = knob positions on the same chromosome; + = presence of knob; - = absence of knob.

The presence of some new and unusual knob positions in NEH maize, hitherto unknown in American maize races, have been identified in the present study. These positions are 1Lb, 2Lb, 2LT and 9Lb, all having small sized knobs except the medium sized knob at the 2LT position. Knobs at these positions have also been reported in Kashmir maize (Jotshi and Patel, 1983). All of these new knob positions have varied geographical distribution in NEH. 1Lb and 2Lb knobs, present distally on the long arms of chromosomes 1 and 2, are restricted to only two collections from Tripura, namely T-7 and T-17, respectively; whereas a 2LT knob, on the terminal position of the long arm of chromosome 2, is present in the M-240 collection from Meghalaya, and a 9Lb knob in N-52 from Nagaland. It is interesting to note that knobs at both the 2La as well as the 2LT positions on the long arm of chromosome 2 have been observed in different plants of the same strain. The presence of these new knob positions in different regions sug-

gests their adaptive value and indicates the uniqueness of NEH maize.

Thus, the NEH knob complex may be comprised of frequent knob positions 2La, 4L, 6S, 6Lb, 7L, 8La and 9ST. Less frequent knobs at 1La, 3L, 5L, 6Lc, 7ST, 8L and 9L are also present in this region (Table 2). The presence of these most frequent knob positions in a majority of strains studied indicates that they were present in the original introduction into the NEH region. Fixation of these knobs with variable frequency, in a majority of the NEH strains, speaks of their adaptive value. The absence of some of these knobs in some strains can be explained on the basis of genetic drift.

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Recombinant inbred pairs (RIPs) as a more efficient RFLP mapping population

--David Weber and Tim Helentjaris

Several public and private labs currently have active RFLP mapping programs in maize. While RFLP mapping, one needs to subdivide the genome into as many compartments or "bins" as possible utilizing a fixed number of individuals in the mapping population. Two types of mapping populations are used almost exclusively for this purpose at this time: 1) F2s and 2) recombinant inbreds (RIs). Both of these populations have important virtues for mapping. In our discussions to identify the most efficient population to use for RFLP mapping, we concluded that a third type of mapping population, recombinant inbred pairs (RIPs), might be a preferable population to use for certain types of RFLP mapping because it would provide resolution that is essentially twice as great as with either F2s or RIs.

We realize that there are differences of opinion about how one should calculate the resolution of mapping using different mapping populations, and the discussion below is designed to compare the relative efficiencies of different mapping populations for developing RFLP genetic maps. Hopefully, these considerations are correct. More importantly, we hope this will stimulate discussion, and we look forward to your comments about this proposed approach.

Most RFLP mapping is carried out with DNAs from F2 plants. Numerous F2 populations are available for immediate use, and each F2 plant contains two different recombinant gametes. Furthermore, each F2 plant can also be selfed to produce a population of F3 kernels which contains all of the alleles that were present in the F2. By planting a relatively small number of these F3s and pooling the DNAs from these plants, one is able to obtain a large amount of DNA which should be enough for numerous studies. These have been described as "immortal F2s or IF2s".

One can calculate the number of "bins" that are present in a gamete produced by a F1 plant in the following way: 1) 10 bins are present without recombination because 10 chromosomes are present, 2) assuming that the maize genome is 2300 map units long, an average of 2.3 recombinational events will involve each of the 10 chromosomes; therefore, an average of 33 bins (10 + 10 x 2.3) will be found in each gamete produced by an F1 plant. Each F2 plant is formed by the fusion of two F1 gametes; therefore, an F2 plant will contain an average of 2 x 33 or 66 bins.

Mapping with recombinant inbreds in maize has been pioneered by Burr et al. (Genetics 118:519-526, 1988). Recombinant inbred lines are produced by inbreeding the progeny of an F2 for a sufficient number of generations to achieve homozygosity or near homozygosity. Recombinant inbred families, therefore, are a permanent population in which segregation is complete or nearly complete that can be used indefinitely for mapping.

One can calculate the number of "bins" that are present in a recombinant inbred plant using the assumptions given above. Both of the haploid genomes in a F2 plant would contain 33 bins and they are 50% homozygous; therefore, a F3 plant produced by a F2 would contain 33 bins + 16.5 (33/2) informative recombinants per chromosome or 49.5 bins. Each successive selfing reduces the amount of heterozygosity by half; therefore, a F4 plant would contain 33 + 16.5 + 8.25 bins, a F5 would contain 33 + 16.5 + 8.25 + 4.125 bins, F6 would contain 33 + 16.5 + 8.25 + 4.125 + 2.0625 bins etc. so that the number of bins in a recombinant inbred approaches 66.

Thus, the genome is divided into 66 bins in each F2 plant and into essentially 66 bins in each RI plant. In our discussions, we realized that by using the DNA from a pair of recombinant inbreds (RIPs) in each lane on a gel, one would have 132 bins in each sample analyzed. One could either mix DNAs from pairs of recombinant inbreds and place these DNAs in individual lanes or extract DNA from F1s between two different recombinant inbreds; the results would be the same. In this simple way, one would nearly double the number of bins. The analysis of this material could be carried out with the Mapmaker program using an appropriate correction factor.

We believe that if this simple approach were used, the resolution would be essentially doubled using a fixed number of DNA samples for mapping.

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Observations of silk resistance to *Fusarium graminearum* in germplasm with resistance to *Aspergillus flavus* and *Heliothis zeae*

--L.M. Reid, D.E. Mather and R.I. Hamilton

The ear-rotting pathogen *Fusarium graminearum* Schwabe can enter the maize ear via the silk and/or silk channel. Infection then spreads vertically from the rachis tip. Few sources of resistance to silk infection have been identified. It was hypothesized that genotypes with resistance to other pests that enter the ear via the silk might have some resistance to *F. graminearum*. Resistance to *F. graminearum* was therefore evaluated in germplasm known to be resistant to the fungal pathogen *Aspergillus flavus* Link ex. Fries and/or the corn earworm *Heliothis zeae* Boddie.

The germplasm evaluated consisted of: two *A. flavus* resistant lines, Mp313E and Tx601, and three F1 hybrids with the highly resistant inbred line CO272 and the moderately resistant inbred line F7 (Mp313E X F7, Tx601 X F7, and, Tx601 X CO272); six *H. zeae* resistant germplasm lines (GT113, GT114, GT115, GT117, GT1118, and GT119) released by Widstrom et al. (Crop Sci. 28:202, 1988); and the photo-insensitive Mexican landrace Zapalote Chico whose silks contain the flavone C-glycoside maysin

which has been associated with earworm resistance (Waiss et al., J. Econ. Entomol. 72:256-258, 1979).

Twenty plants of each inbred, hybrid, or population were inoculated by injecting 2ml of a spore suspension of *F. graminearum* (2×10^6 spores/ml) into the upper silk channel of the primary ear approximately 6 days after silk emergence. Resistance to spread of infection was evaluated in mid-October using a 7-class disease rating scale where 1= no infection, 2= 1-3%, 3= 4-10%, 4= 11-25%, 5= 26-50%, 6= 51-75%, and 7= >76% of kernels infected.

An in vitro test of detached silk tissue (Naik and Busch, Can. J. Bot. 56:1113-1117, 1978) was used to evaluate the ability of *F. graminearum* to degrade and assimilate silk of different genotypes. From each of 10 primary ears, two 1g samples of a cross-section of the silk mass were cut from the silk channel. Each sample was placed in a petri dish containing filter paper moistened with 3ml of sterile water. One sample of each pair was sprayed with 3ml of spore suspension (5×10^5 spores/ml) and the other with 3ml of sterile water. The pairs of petri dishes were incubated in a growth cabinet for 7 days at 25 C and 95% relative humidity. Samples were dried in an oven at 80 C for 48 hrs and weighed. Reduction in silk dry weight due to degradation and assimilation by the fungus was measured as the percentage difference in dry weight between the inoculated sample and the water-control sample.

Three of the *H. zeae* resistant lines (GT115, GT117, GT118), the two *A. flavus* resistant lines, and the hybrids Tx601 X F7 and Tx601 X CO272 were highly resistant (mean disease rating <3.0) (Table 1). The hybrid Mp313E X F7 and the landrace Zapalote Chico were moderately resistant (mean disease rating 3-4). The remaining three *H. zeae* resistant lines were as susceptible as the *F. graminearum* susceptible inbred CO266.

In some of the Southern U.S. genotypes, there was very little fungal degradation and assimilation of the silk tissue. There was less reduction in dry weight for silk tissue from Mp313E, Tx601, Mp313E X F7, and Zapalote Chico than for silk tissue from the highly resistant inbred CO272.

Some of the Southern U.S. genotypes evaluated warrant further study as sources of resistance to *Fusarium* ear rot. Four inbreds (GT117, GT118, Mp313E, and Tx601) had lower disease ratings than the highly resistant inbred CO272. However, only GT117 was adapted to the northern-temperate region.

Within Zapalote Chico, some plants had disease ratings of 6 or 7 but most had disease ratings of 1 or 2. This may reflect genetic segregation for resistance, and Zapalote Chico may be a more useful source of resistance than would be expected from its moderate disease rating.

Because of the high frequency of resistance observed among the genotypes evaluated, further pathological and phytochemical studies are being conducted to determine whether there is a relationship among the mechanisms against *F. graminearum*, *A. flavus*, and *H. zeae*.

Table 1. Evaluations of resistance to *F. graminearum* and days to silking of 11 inbreds, 3 hybrids, and one landrace grown at Ottawa, Canada.

	Mean Disease Rating[a]	Tissue Degradation (% Reduction in Dry Wt.)[b]	Days to Silking
GT113	5.2 ab	29.0 a	82
GT114	5.0 a	24.2 a	81
GT115	2.1 d	8.1 c	123
GT117	1.5 d	9.3 bc	89
GT118	1.1 d	9.6 b	124
GT119	5.2 a	26.3 a	84
Mp313E	1.0 d	1.7 d	124
Tx601	1.6 d	1.3 d	123
Mp313E X F7	3.1 bc	3.1 d	85
Tx601 X F7	2.9 bc	9.7 b	82
Tx601 X CO272	2.6 cd	9.2 bc	83
Zapalote Chico	3.0 b	3.5 d	82
CO272	1.9 d	7.2 c	85
F7	3.2 ab	23.9 a	69
CO266	5.0 a	32.1 a	79

[a]Based on a scale of 1-7 where 1= no infection and 7= >76% of the ear infected. Means followed by the same letter are not significantly different at the 0.15 experimentwise error rate.

[b]Means followed by the same letter are not significantly different at the 0.05 level by Duncan's multiple range test.

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Dichotomous branched mutant (*dib*)

--V. E. Micu, Eugenia C. Partas and D. G. Brinzila

It was reported earlier (MNL 54:63-64, 1980) that the two-tassel maize mutant was found in 1967 in one progeny of selfing of an open-pollinated population from Afghanistan (K2858). The stalks are dichotomously branched at nodes 4-8, and two branches with normal leaves, ears and tassels develop. Very rarely twice branched plants (with four branches and tassels) are observed. The mutant was named *dib*, dichotomous branched. The morphological description and results of preliminary genetic and cytological analyses were published. Some new data are presented in Table 1.

Table 1. Comparative traits of *dib* and normal plants.

Traits		Number of genotypes	Normal	*dib*
Plant	height, cm	11	117	112
	number of nodes to ear	11	3.1	3.4
	number of nodes above ear	11	5.9	5.8
Leaf:	length, cm	11	48.9	44.7
	width, cm	11	6.1	5.3
Ear:	length, cm	35	12.3	8.9
	number	11	1.2	1.9
Tassel:	length, cm	11	26.3	24.0
	number of flowers per tassel	42	563	491
	number of flowers per plant	42	563	982
	number of branches	11	9.1	8.2
Weight of 100 anthers, g		5	0.06	0.05
Weight of anthers from 1 tassel, g		5	2.1	1.8
Weight of anthers from 1 plant, g		5	2.1	3.5
Pollen viability, %		16	74.4	80.8

The numerous progenies which were obtained from *dib* self-, sib- and outcross-pollinated with normal were studied. It is evident from these studies that *dib* is not determined by the nuclear genes as well as the cytoplasm.

In the somatic cell of *dib* plants a variable number (from 20 to 22) of chromosomes were found that indicate aneuploidy as a cause of dichotomous branching.

During the last ten years we made efforts to obtain progenies consisting only of branched plants. The *dib* plants were selfed for several generations. From 1388 progenies studied in 1991 in three of them all plants were *dib*, in another 8 more than 90% were *dib*, and in 322 more than 50% were *dib*.

These data indicate that it is possible to obtain entirely and constant *dib* progenies and lines which can be used in breeding and in seed production.

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Mitotic instability in callus cultures of inbred lines adapted to tropical regions

--Margarida L. R. de Aguiar-Perecin and Antonio Fluminhan, Jr.

A procedure to investigate aspects of mitotic instability occurring in callus culture is presented in this report. We analyzed cultures initiated from F2 immature embryos derived from the cross between two sister lines designated 300-14-1315 (S7) and 300-14-13213 (S8), which have Type II (friable and embryogenic) culture response. These lines are derived from a flint variety (Jac Duro, Sementes Agroceres) developed in Brazil, and are homozygous for C-bands correspondent to K6L2/K6L3, K7S, K7L, K8L (L1 or L2, not determined), and K9S (references in MNL 62:100, 1988).

The cultures were incubated on MS medium containing 2mg/L 2,4-D and 20mg/L casein hydrolisate (Fluminhan and Aguiar-Perecin, Ciencia e Cultura Suppl. 43:760-761, 1991). Mitotic analysis was carried out on meristematic cells of proembryoids taken from the callus 5 days after its transferrence to fresh medium, fixed in 3:1 alcohol:glacial acetic acid and stained in Feulgen. C-banding was also employed. Proembryoids were squashed in 45% acetic acid after incubation in 2.5% pectinase. For metaphase preparation a pretreatment with 0.002M hydroxiquinoline was carried out.

The analysis of anaphase bridges showed two types of figures: i) bridges which appear to be the result of delayed segregation of sister chromatids (Fig. 1); ii) bridges (with and without bands) apparently originating from a breakage-fusion-bridge cycle initiated by broken chromosomes (Fig. 2). These observations are consistent with the interpretation that the chromosome aberrations, usually involving knobbed chromosomes, observed in regenerants are the result of chromosome breakage occurring in culture (Phillips et al., Corn and Corn Improvement, pp. 345-388, 1989).

We analyzed the frequency of bridges in some cell lines as shown in Table 1. Anaphases with 1 and 2 bridges were observed in these samples. A preliminary observation of metaphases showed

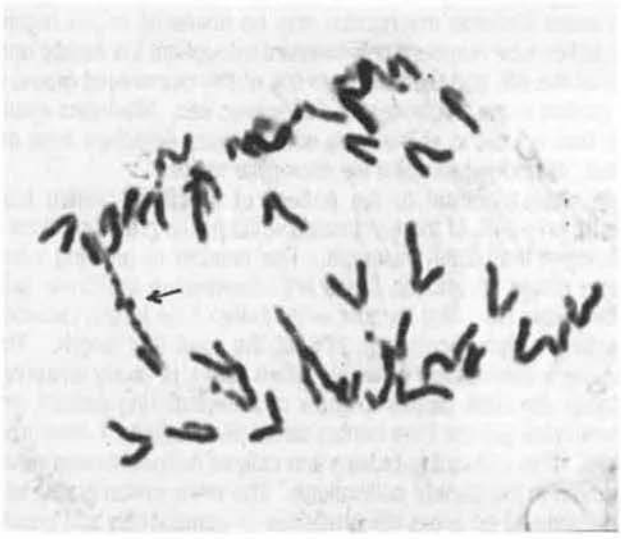

Figure 1. Feulgen stained anaphase showing one bridge apparently derived from delayed segregation of sister chromatids.

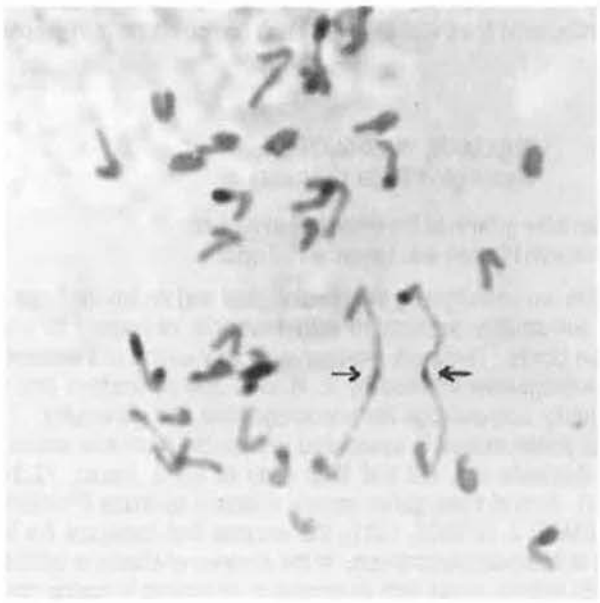

Figure 2. C-banded anaphase with two bridges involving a banded chromosome arm. They appear to be originated from previously broken chromosomes.

Table 1. Frequency of mitotic anaphases with bridges in 3 cell lines.*

			Anaphases	
Cell line designation	Weeks in culture	Normal	1 bridge	2 bridges
H	26	25	-	-
J	26	103	4	-
H	39	20	-	1
H	43	140	6	1
57	53	141	4	3

*Scored in Feulgen stained preparations.

clear alterations of the structure of chromosome 7, such as the presence of 3 bands, K7S on a subterminal chromosome position and amplification of K7L (Fig. 3). These aspects suggest that in

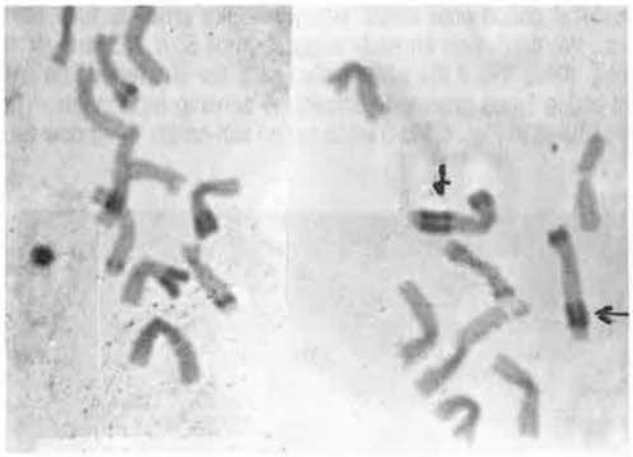

Figure 3. C-banded metaphase showing enlarged bands at K7L.

most cases this is the chromosome involved in the formation of bridges. One interesting aspect concerning the chromosomes with enlarged bands is that their pattern is not altered. If this amplification is an initial event that leads to the delay of chromatid segregation, it is an interesting point for further investigation. A

complete report on the frequency of anaphase bridges in cultures of endogamic lines with different knob compositions is in preparation.

PULLMAN, WASHINGTON
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Pollen tube growth in the white pollen mutant

--Karen Hansen and Loverine P. Taylor

We are investigating the physiological and molecular basis for the self-sterility associated with flavonoid deficiency in white pollen plants. This work represents a continuation and expansion of investigations initiated by E. H. Coe and co-workers and we gratefully acknowledge his encouragement and generosity. The white pollen mutant is associated with stable recessive alleles at the duplicate loci, *C2* and *Whp* (Coe et al., J. Hered. 72:318, 1981). Both of these genes encode chalcone synthase (Franken et al., EMBO J. 10:2605, 1991), the enzyme that catalyzes the first step in flavonoid biosynthesis. In the absence of chalcone synthase (CHS) activity, plants lack flavonoids in all tissues including pollen (Coe et al., 1981; D. Styles, unpublished data; L. P. Taylor and C. W. Nagel, in preparation).

Characterization of a similar phenomenon in petunia shows that white pollen is unable to germinate either in vivo or in vitro (Taylor and Jorgensen, J. Hered. 83, 1992). To determine if the maize white pollen mutant (*c2/c2 whp/whp*) exhibits a similar pollen phenotype, we compared in vitro germinated white pollen with an isogenic line differing by a single functional copy of CHS (*C2/c2 whp/whp*). Pollen from both the mutant and functional lines readily germinates on semi-solid media and produces tubes that are indistinguishable even after 8 hours of growth. However, in vitro pollen tube growth rates never approach the rates seen in vivo, suggesting that differences between the mutant white pollen and functional pollen may only be apparent in vivo.

Self-crosses of the white pollen mutant produce virtually no seed but the plants are female fertile and crosses of *C2/c2 whp/whp* pollen onto *c2/c2 whp/whp* silks produce fully filled ears. We confirmed an early suggestion of S. A. Modena (MNL 56:47, 1982) that if the white pollen silks are shortened, the mutant pollen tubes grow long enough to achieve fertilization. The ear pictured in Figure 1 is a white pollen self-cross. A window was cut through the husks, the silks within the window area were carefully extracted, cut to a uniform 1.5cm length, and self pollinated. Nearly full kernel set occurred in the window area.

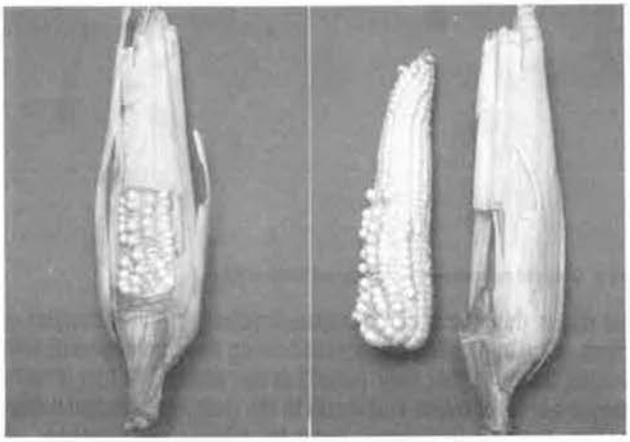

Figure 1. White pollen self-cross 45 days post pollination.

This experiment indicates that white pollen tubes can grow at least 1.5cm. To determine where pollen tube growth ceases, we examined self-crossed white pollen silks throughout their length for the presence of pollen tubes. White pollen silks were harvested 48 hrs. after pollination with either yellow *C2/c2 whp/whp* pollen (control) or white *c2/c2 whp/whp* pollen, fixed in acetic acid/formaldehyde/ethanol (1:1:8), cleared in 8N NaOH, and stained with decolorized aniline blue (0.01% in M/15 K_2HPO_4). Pollen tubes are visualized within the silks by fluorescence microscopy of the brightly stained callose deposits that are characteristic of growing pollen tubes.

Pollen grains caught by the silk hairs hydrate then germinate. The growing tube penetrates and moves into the body of the silk where it grows parallel to the vascular bundles. On a representative group of silks (17) pollen tube length was scored relative to the total silk length (Figure 2). Of the control pollinations, >98%

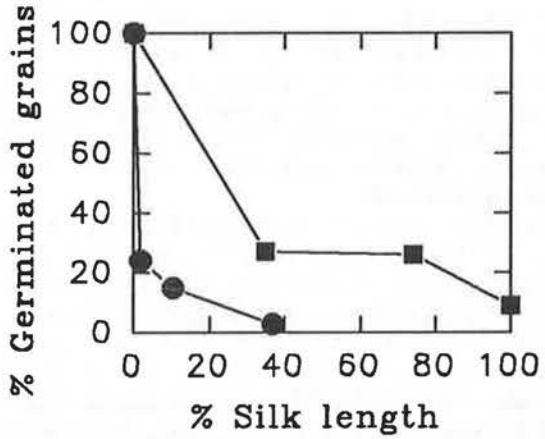

Figure 2. Pollen tube growth relative to silk length of *C2/c2 whp/whp* (■) and *c2/c2 whp/whp* (●).

of the germinated *C2/c2 whp/whp* pollen produced a tube at least 2mm in length. The dramatic decrease in the number of tubes approximately 1/3 of the distance down the silk suggests that some selective mechanism may be operating in this region. The pollen tube number stays constant throughout the middle one-third of the silk and then drops to 9% of the germinated grains in the portion of the silk closest to the embryo sac. Most silks examined had 1-3 tubes at the base where it was detached from the kernel. We did not examine the micropylar region.

In sharp contrast to the pattern of functional pollen tube growth, only 24% of the germinated white pollen grains produce a tube more than 2mm in length. The number of growing tubes rapidly drops off and no tubes are observed in the lower two-thirds of the silk. The longest white pollen tube length recorded was 5cm which represents 37% of the total silk length. The premature termination of white pollen tubes is easily observed because the tube begins to grow in a meandering pattern and accumulates greater than normal amounts of callose 1-4mm from the tip. This undulating pattern and callose deposition was never observed in the control pollinations. The white pollen grains and tubes showed no gross abnormalities in germination and growth until this point, which agrees with our in vitro germination observations. These results suggest that unlike petunia,

flavonoids may not be essential for pollen germination in maize, however, they are required for the sustained tube growth necessary to achieve fertilization.

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Callusing and rhizogenesis–response of inbreds and their hybrids

--A. Kumar, H. Kumar and S. K. T. Nasar

Some selected inbreds, Tuxp124-1, Tuxp162-1, Tuxp46-6, Tuxp237-2, Tuxp104-2, MS1DR-120, PKMS1-49, and hybrid genotypes, Tuxp162-1xTuxp237-2, Tuxp162-1xTuxp124-1, Tuxp237-2xTuxp124-1, Tuxp104-2xTuxp46-6, MS1DR-120xPKMS1-49, which were never exposed to tissue culture experiments, were subjected to callusing and rhizogenesis.

The seeds were germinated on Murashige and Skoog's (MS) basal medium. Hybrid genotypes Tuxp162-1xTuxp237-2 and MS1DR-120xPKMS1-49 showed a greater number of roots as well as longer root length than their inbreds, while one hybrid, Tuxp104-2xTuxp46-6, showed an intermediate response. For shoot growth all hybrid genotypes showed better response than their inbred genotypes except for the hybrid genotype MS1DR-120xPKMS1-49.

The MS basal medium supplemented with 2,4-dichlorophenoxyacetic acid (2,4-D), indole butyric acid (IBA), indole 3-acetic acid (IAA) and kinetin (KN) in different concentrations either singly or in combinations of two, were used for callusing and rhizogenesis. The explants used for callusing were root segments taken from seeds germinated on MS basal medium, while for rhizogenesis the callus formed on root segments was also used.

Callus formation from root segments was best achieved on the medium MS + 2,4-D (5.0mgL^{-1}) + IAA (2.0mgL^{-1}), with 88.88% of the cultures showing very good callusing response, followed by a response of 77.00% on the medium MS + 2,4-D (5.0mgL^{-1}) + IBA (2.5mgL^{-1}). The hybrid genotypes Tuxp162-1xTuxp237-2, Tuxp162-1xTuxp124-1 and Tuxp237-2xTuxp124-1 showed a response inferior to their inbreds with respect to the frequency of callus formation and callus growth. However, the other hybrid genotypes, Tuxp104-2xTuxp46-6 and MS1DR-120xPKMS1-49, showed better response than their inbreds. This was true for most of the media combinations having 2,4-D, IBA, and IAA that favoured good callus formation. The media combinations with KN showed minimum callus formation, but the calli were compact and green, with globular structures.

Rhizogenesis was achieved in many media combinations and in most of the genotypes. Roots were differentiated directly from the explant as well as through the formed callus, but the frequency of callus mediated rhizogenesis was more in most of the cases except for the media with IBA. The hybrid genotypes MS1DR-120xPKMS1-49 and Tuxp104-2xTuxp46-6 showed better response over their inbreds for the percentage of cultures showing rhizogenesis. The other hybrid genotypes were inferior to their inbreds in this aspect. However most of the hybrid genotypes showed better response than their inbreds for the number of differentiated roots per culture.

Most of the hybrids showed heterotic performance for mean root and shoot length, but some of them failed to overshoot the 'high' parent in the case of in vitro germination and thus failed to show a positive heterotic effect. For callusing and rhizogenesis also, although intergenotypic differences were observed, the hybrids did not always show positive heterotic effects. Thus the maize hybrids that were positively heterotic for yield have not always shown similar performance for tissue culture responses. This difference of performance of hybrids may be because of the different genic x environmental interactions for yield and tissue culture responses.

Ac id phosphatase isozymes in root, coleoptile and callus tissues of inbreds and their hybrids

--A. Kumar, H. Kumar, V. K. Shahi and S. K. T. Nasar

Isozymes of acid phosphatase were studied under different developmental stages, such as root and coleoptile, in vivo and at the callus stage in vitro in 5 inbreds, Tuxp162-1, Tuxp237-2, Tuxp104-2, MS1DR-120, PKMS1-49, and 4 hybrid genotypes, Tuxp162-1xTuxp237-2, Tuxp237-2xTuxp124-1, Tuxp104-2xTuxp46-6, MS1DR-120xPKMS1-49.

In all, 8 different isozymes were found in maize root, coleoptile and callus tissues. Based on their mobility towards the anode, these isozymes were numbered as A-1 to A-8 starting from the origin. These isozymes were distributed in 4 electrophoretically different zones named (i) AP-1 having isozyme band A-1, (ii) AP-2 having isozyme bands A-2 to A-4, (iii) AP-3 having isozyme bands A-5 to A-7 and (iv) AP-4 having isozyme band A-8.

Coleoptile tissues had zones AP-1, AP-2 and AP-3. The AP-4 zone was present only in callus tissues with AP-1 and AP-2 zones. Roots had only AP-2 and AP-3 zones.

Coleoptile tissues of inbreds Tuxp162-1, Tuxp237-2 and their hybrid had identical isozyme bands showing the same isozyme pattern, but they differed in the intensity of isozyme activity. Similar observations were found with inbreds MSDR1-120, PKMS1-49 and their hybrid. Hybrid Tuxp237-2xTuxp124-1 had different bands than its parent Tuxp237-2, while hybrid Tuxp104-2xTuxp46-6 showed similar bands to those shown by one of its parents, Tuxp104-2. However, the intensity of activity of isozymes was different.

The isozyme pattern of callus tissues was different than that of coleoptile tissues. Inbreds Tuxp162-1, Tuxp237-2 and their hybrid had the same bands, except for the absence of the A-8 band in the latter inbred and a difference in intensity of bands. A-8 was a new isozyme band not found in coleoptile and root tissues.

Isozyme pattern of root tissues in the inbreds MS1DR-120, PKMS1-49 and their hybrid differed in bands as well as in the intensity of identical bands.

On overall comparison, it has been found that: (i) the maximum number of isozymes was expressed in the coleoptile tissues; (ii) not only fewer isozymes were present in the calli grown in vitro, but their mobility and intensity were also markedly different; (iii) a new band, A-8, appeared in the calli of only two genotypes; and (iv) a uniform pattern of isozyme expression as was found in coleoptilar tissues was not obtained in the case of calli.

The study has shown that the genotypes under consideration were closely related with respect to acid phosphatase enzyme. In no case was a total absence of acid phosphatase enzyme observed. In coleoptiles, variation in electrophoretic mobility between parents and hybrids has not been seen. This was indicative of homozygosity at the acp loci. A marked difference in the isozyme number and mobility in calli compared to their callusing explant and nonavailability of band A-8 in in vitro tissues was due to activation

of an otherwise normally repressed locus due to genotype x tissue culture interactions.

Interphase nuclear size variations in somatic and cultured tissues

--A. Kumar, H. Kumar and S. K. T. Nasar

Root explants from some selected inbred and hybrid genotypes, Tuxp124-1, Tuxp162-1, Tuxp46-6, Tuxp237-2, Tuxp104-2, MS1DR-120, PKMS1-49, Tuxp162-1xTuxp237-2, Tuxp162-1xTuxp124-1, Tuxp237-2xTuxp124-1, Tuxp104-2xTuxp46-6, MS1DR-120xPKMS1-49, of maize were exposed to callusing in Murashige and Skoog's basal medium supplemented with 2,4-dichlorophenoxyacetic acid ($3.0mgL^{-1}$) and kinetin ($2.0mgL^{-1}$).

The diameters of interphase nuclei, reflective of nuclear volumes, of root meristem cells, and the cells of calli developed from root explants were subjected to a comparative study. The frequency distribution of differently sized interphase nuclei among root meristems and calli of different genotypes was also compared.

Among the genotypes studied, the mean nuclear diameter ranged between 67.368μ for genotype MS1DR-120 root tip cells and 8.522μ for genotype Tuxp104-2xTuxp46-6 callus tissue cells. On the whole the callus nuclei were smaller in size compared to the root cell nuclei. For the root tip nuclei, the hybrids showed larger sizes than those of their parents.

The frequency distribution of variously sized interphase nuclei gave an insight into the distribution of G1, S and G2 nuclei in analogous populations. Root meristems showed normal to near normal distribution in comparison to highly skewed to bimodal distribution in analogous callus tissues.

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Karyological studies in *Coix* and maize

--S. K. Katiyar, G. Chandel, R. Kumar* and J. K. S. Sachan*
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The study of karyotype has been realized as an aid to evolutionary significance and taxonomic classification. So far the least effort has been made to study the chromosomes of *Coix* and their comparison with *Zea* chromosomes. Therefore, the present investigation was carried out to standardize the cytological technique of mitotic chromosomes, construct the somatic karyotypes of *Coix* spp. present in the Indian subcontinent and establish chromosomal relationships within and between different species of *Coix* and with maize.

When 28 distinct collections of *Coix* from different geographical regions were studied for their somatic chromosome number, it was found that different populations of Indian origin largely fell into two major groups, i.e., (i) those with a chromosome complement of 2n=10 (*Coix aquatica*) and (ii) those with a chromosome complement of 2n=20 (*C. lacryma jobi*). Four collections showed deviations from the exact multiple of the basic number; the deviations were later found to be due to the presence of B-chromosomes in variable frequency in *C. aquatica*. Our results for different genotypes within species further revealed considerable intervarietal differences with respect to total chromosome complement, relative chromosome length, arm ratio, centromeric index and number of satellited chromosomes in *Coix*. Observations showed one pair of metacentric and four pairs of submetacentric, and four pairs of metacentric and six pairs of submetacentric chromosomes in *Coix aquatica* and *C. lacryma jobi*, respectively. *Coix aquatica* and *C. lacryma jobi* had two and five pairs of satellited chromosomes, respectively. Somatic karyotype analysis also showed that *C. aquatica* (2n=20) not only has fewer chromosomes, but also has the smallest genome at metaphase (33.25um long, with a mean chromosome length of 6.64um). The total genomic lengths of *C. lacryma jobi* and *Z. mays* were 80.82um and 87.43um, respectively.

Critical comparison of arm ratios and relative chromosome lengths of *C. aquatica* and *C. lacryma jobi* along with other morphological characteristics of chromosomes reveals that the genomes in the two species are quite different, however, the genomes of cultivated and wild *Coix* within *C. lacryma jobi* have shown a high degree of similarity. Qualitative features of maize karyotypes are relatively uniform. The karyotype of maize is symmetrical, however, it is entirely different from that of *Coix*. The chromosome number and pairs of chromosomes with secondary constriction are also different, indicating clearcut differences in the genomes of *Coix* and maize.

Somatic embryogenesis in *Coix aquatica* Roxb.

--G. Chandel and S. K. Katiyar

Protocols for the in vitro production of plants through somatic embryogenesis from cultured immature, unemerged inflorescence segments of *Coix aquatica* genotype C-21, C-27 and C-65 have been developed.

Out of various concentrations of 2,4-D (2,4-dichlorophenoxy acetic acid) and sucrose tested for callusing, N6 media with 1-2mg/l 2,4-D and 3% sucrose concentration gave good response.

Table. Mean relative chromosome length, arm ratio and centromeric index (CI) in *Coix* and maize.

	Relative Chromosome Length			Arm Ratio			Centromeric Index		
Chromosomes	*Coix aquatica* (2n=10)	*Coix lacryma jobi* (2n=20)	*Z. mays* (2n=20)	*Coix aquatica* (2n=10)	*Coix lacryma jobi* (2n=20)	*Z. mays* (2n=20)	*Coix aquatica* (2n=10)	*Coix lacryma jobi* (2n=20)	*Z. mays* (2n=20)
1	24.01	12.96	11.90	1.24	1.24	1.22	44.55	44.38	44.90
2	21.84	11.88	11.90	1.27	1.40	1.88	44.29	41.65	34.64
3	20.08	11.28	11.66	1.41	1.21	1.33	41.29	45.18	42.84
4	18.32	10.53	10.71	1.40	1.22	1.19	42.07	44.86	45.46
5	16.36	9.89	10.71	1.36	1.20	2.00	42.63	45.63	33.29
6	-	9.51	10.71	-	1.40	1.66	-	42.23	37.28
7	-	9.10	8.80	-	1.31	1.54	-	41.30	39.35
8	-	8.91	8.56	-	1.32	2.29	-	43.51	30.32
9	-	8.14	8.32	-	1.31	1.69	-	43.38	37.08
10	-	7.65	6.66	-	1.46	1.27	-	40.75	44.03

Coix aquatica - overall mean of 20 collections.
Coix lacryma jobi - overall mean of 8 collections.

The frequency of callus induction from genotype C-27 was up to 80%. Differences in callusing were found to be due to genotypes rather than the concentrations of 2,4-D. The length of the explant at the time of inoculation was also found to be critical for callusing.

Out of two different types of calluses subcultured, compact callus produced embryos when transferred to liquid MS medium with various combinations and concentrations of NAA, BAP and KIN. The maximum number of plantlets were regenerated on MS medium supplemented with 0.5mg/l BAP+0.01mg/l NAA and 3% sucrose in 25-30 days after subculturing. The combination of BAP+NAA was found to be better than the combination of KIN+NAA. Vigorous regenerated plants were transferred to sterile soil after 1-3 days of hardening treatment, kept in the glasshouse for 15-20 days, and later transferred to the field. In vitro regenerated plants showed normal flowering and seed set. This is the first time that complete reproducible in vitro regeneration protocols in *Coix aquatica* have been developed and plants taken to maturity. The seeds obtained are viable. This is a very significant step and opens up avenues for the application of plant genetic engineering methods.

Pachytene chromosome morphology of *Coix aquatica* and its comparison with *Zea* chromosomes

--S. K. Katiyar and J. K. S. Sachan

For the first time detailed cytological studies in terms of pachytene chromosome lengths, total genomic length, relative chromosome length, arm ratio and centromeric index, distribution of euchromatic and heterochromatic regions and presence or absence of heterochromatic knobs in 26 distinct collections of *Coix* spp. belonging to different geographical regions of the country were carried out, keeping in view that no systematic attempts have been made to study pachytene chromosomes of *C. aquatica* and karyotypic differences in *Coix* and *Zea* chromosomes, as both belong to the same tribe, Maydeae. Six collections of maize of both Indian and American origin, and 3 species of teosinte viz. *Z. diploperennis, Z. luxurians* and *Z. mays* ssp. *parviglumis* were also included in the study for comparison and establishing the interrelationship of *Coix* with them.

The relative chromosome lengths among 26 collections of *Coix aquatica* (2n=10) studied ranged from 13.55 to 28.28, whereas, arm ratios varied in a very narrow range of 1.07 to 2.30. The relative lengths of chromosomes 1, 2, 3, 4 and 5 of *Coix* at pachytene ranged from 22.35-36.96, 19.96-28.61, 13.68-21.37, 11.47-20.11 and 8.81-17.52, with an overall mean relative chromosome length of 28.28, 22.74, 18.59, 16.80 and 13.55, respectively. Either chromosome 1 and 2 or 1 and 3 were found to be satellited. Chromosome 1 is metacentric (CI=48.14), chromosomes 2 (CI=41.18) and 3 (CI=40.57) are submetacentrics, whereas, chromosomes 4 (CI=30.24) and 5 (CI=33.95) are acrocentrics. The essential uniformity in the gross features of the pachytene karyotype of *C. aquatica*, except for heterochromatin distribution pattern, are clearly evident by comparative karyomorphological study of different collections. There is a gradual decrease of lengths from the longest to the shortest chromosome with no abrupt change in size, which shows that the *C. aquatica* karyotype is symmetrical. The positions of heterochromatic blocks on each chromosome vary from collection to collection, however, some positions are stable while others undergo rapid changes producing polymorphism at a particular position between different collections.

The genome of *Coix aquatica* (2n=10) at pachytene is about 1.5 times longer than that of *Zea* and the chromosomes themselves are quite dissimilar in morphology to those of *Zea*. Chromosomes of *C. aquatica* are differentiated, showing differentially stained eupycnotic and heteropycnotic regions. They are also devoid of knobs. Maize and teosinte chromosomes do not exhibit similar differential staining and they possess knobs. Karyomorphological comparison (Table 1) thus does not reveal common features between the genomes of *Coix* and *Zea*, and it seems that the genomes present are quite different.

Scanning electron microscopic studies of pollen grains in the tribe Maydeae

--S. K. Katiyar and J. K. S. Sachan

To study the pollen size and surface features of exine, pollen grains of ten distinct collections of *Coix*, including both *Coix aquatica* and *C. lacryma jobi*, three collections of teosinte, including *Zea diploperennis, Z. luxurians* and *Z. mays* ssp. *parviglumis*, four collections of maize (*Zea mays* L.) and one collection each of *Chionachne koenigii* and *Trilobachne cooki* were collected and a stereocan S4-10, scanning electron microscope was used for the observations and photography.

The pollen grains of different species and genera studied were round or slightly oval in shape and uniporate. The pollen of maize was in general larger with a mean pollen grain size of 106.14um, followed by teosinte (85.11um), *Coix* (55.99um) and *Chionachne* (34.70um). Pollen grains of *Coix aquatica* had a mean diameter of

Table 1. Mean chromosome length (um), relative length (um) and arm ratio at pachytene in maize, teosinte and *Coix* (2n=10).

	Chromosome Length			Relative Chromosome Length			Arm Ratio		
Chromosome number	Maize	Teosinte	*Coix*	Maize	Teosinte	*Coix*	Maize	Teosinte	*Coix*
1	84.78	83.77	236.52	14.98	15.31	28.28	1.32	1.34	1.07
2	67.71	64.56	191.23	11.97	11.80	22.74	1.35	1.34	1.43
3	63.22	61.94	156.26	11.17	11.32	18.59	2.03	2.04	1.46
4	60.96	57.94	141.64	10.77	10.59	16.80	1.75	1.69	2.30
5	65.16	62.13	112.96	11.52	11.35	13.55	1.06	1.09	1.94
6	47.91	45.53	-	8.47	8.32	-	5.61	4.82	-
7	47.81	45.52	-	8.45	8.32	-	2.85	2.59	-
8	48.59	45.62	-	8.55	8.33	-	3.29	3.08	-
9	42.39	43.45	-	7.49	7.94	-	2.07	2.26	-
10	37.05	36.57	-	6.55	6.68	-	2.54	2.83	-
Total genomic length	565.58	547.03	838.60						
Mean chromosome length	56.55	54.70	167.72						

Maize: mean of all 6 collections studied.
Teosinte: mean of three species of teosinte studied viz., *Z. diploperennis, Z. mays* ssp. *parviglumis, Z. luxurians.*
Coix: mean of all 26 collections studied.

61.49um and were found to be larger than the pollen grains of *C. lacryma jobi* which had a mean diameter of 50.50um.

The scanning electron micrographs (SEM) of pollen of the different material studied have shown that within the genus (except *Coix*) exine sculpture was similar, but marked differences were observed at intergeneric levels. However, in *Coix* enormous variation within the genus was noticed. In maize pollen the spinules were distributed more or less uniformly all over the tectum. They did not follow any linear pattern. The pollen surface of all three species of teosinte studied was similar to maize. The spinules were less densely spaced in teosinte than in maize, however no differences were found in size and shape of the spinules in maize and teosinte and also among teosintes.

In *Coix* pollen the spinules were thinly distributed over the tectum and appeared to follow curved paths. The differences in terms of size and shape of spinules were noticed among various genotypes in the genus *Coix*. Unlike other collections, one culture of *Coix aquatica* was observed to have depressions in the tectum which formed a network over the entire surface. In *Chionachne* pollen, the spinules, which were sparse in distribution, showed a tendency to form groups of 9 to 12. In conclusion, the results of the SEM studies of the exine pattern suggest that maize is very closely related to teosinte followed by *Chionachne* and lastly *Coix*. In sparse distribution of spinules and by following the curved paths *Coix* and *Chionachne* showed some degree of closeness.

Table. Mean pollen grain size and range in maize and its relatives.

Material	Mean (in um)	Range (in um)
Maize	106.14±4.68	94.15-118.83
Teosinte	85.11±3.04	65.00-95.00
Chionachne	34.70±0.77	32.50-38.50
Coix lacryma jobi	50.50±3.16	38.00-65.00
Coix aquatica	61.49±1.77	46.00-75.00
Trilobachne	54.14±1.72	40.30-62.50

Isozyme diversity in *Zea* and related genera

--S. K. Katiyar and J. K. S. Sachan

Unaccountable numbers of genetic and biochemical studies have allowed a still closer examination of evolutionary relationships within *Zea*, but almost all of the studies were limited to American genera of Maydeae; biosystematic studies on Asiatic Maydeae have never been considered seriously.

During the present investigation the members of both American and Asiatic genera of the tribe Maydeae, comprised of 18 distinct collections of *Coix* spp. (including both *Coix aquatica* (2n=10) and *C. lacryma jobi* (2n=20)), 7 of *Chionachne*, 7 of teosinte (including *Zea mexicana, Z. diploperennis, Z. luxurians* and *Z. mays* spp. *parviglumis*) and a cultivar of maize (*Zea mays* L.) were studied for their pollen isozyme banding patterns produced by PAGE of esterase, peroxidase, malate dehydrogenase, acid phosphatase, superoxide dismutase, catalase and glutamate dehydrogenase.

The genetic distances between different collections within the genus and between the genera were calculated from the average similarity indices. The close clustering and resemblances between four *Zea mexicana* collections were noticed. Among the remaining three species, *Zea mays* ssp. *parviglumis* was found to be closer to *Z. diploperennis* than to *Z. luxurians*, *Z. mexicana* was found to be closer to maize than *Z. diploperennis* and *Z. mays* ssp. *parviglumis*. *Zea luxurians* was found to be distantly related to all the species of teosinte and maize. The results of the present study suggest that maize and teosinte are closely related, but at the same time there is a sizable dissimilarity within the teosintes. The electrophoretic movements of different isozymes in *Z. diploperennis, Z. mexicana, Z. luxurians* and *Z. mays* ssp. *parviglumis*, suggest that genetic differences among these species are greater than evident by morphology. All the *Chionachne* collections showed close resemblance to each other irrespective of their place of origin. In the same way all 18 collections of *Coix* clustered separately though the 2 species *Coix aquatica* and *C. lacryma jobi* are distinctly related.

On the basis of the intergeneric electrophoretic affinities the following similarity hierarchy was obtained

Maize	:	Teosinte	*Chionachne*	*Coix*
Teosinte	:	Maize	*Chionachne*	*Coix*
Chionachne	:	*Coix*	Teosinte	Maize
Coix	:	*Chionachne*	Teosinte	Maize

Among the tribe Maydeae genetic distance (GD) was the least (0.309) between maize and teosinte, while *Chionachne* and *Coix* were farther from both maize and teosinte. These results strongly suggest that Asiatic genera are quite distinct from the American ones. Even both members of the Asiatic Maydeae, *Coix* and *Chionachne*, were also found to be distantly related (GD=0.645) to each other.

Table. Genetic distances among different genera of the tribe Maydeae.

	Maize	Teosinte	*Chionachne*	*Coix*
Maize	0.000			
Teosinte	0.309	0.000		
Chionachne	0.781	0.740	0.000	
Coix	0.853	0.766	0.645	0.000

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A Brazilian source of *cms-S*

--Michel Ragot, Paul H. Sisco, Major M. Goodman and Charles W. Stuber

Cms-S has been discovered in Cristal MG III, a racial composite of seven Brazilian maize accessions collected by Dr. M. M. Goodman. This is the first report of cms-S in a South American race of maize. The seven accessions composing Cristal MG III are BR2307, BR2321, BR2659, BR2660, BR2674, BR2681, and BR2696. Cristal is a hard flint, sometimes white and sometimes yellow seeded. Male sterility was first noted among plants in an F2 population of a Cristal-derived inbred crossed by Mo17. To confirm cytotype, mitochondrial DNA from the Cristal-derived inbred and the original Cristal MG III composite was isolated, digested with *Bam*HI, and electrophoresed on agarose gels. *Bam*HI digestion patterns, as visualized by ethidium bromide staining, were compared with those of known cytoplasms. Both digested and undigested Cristal mtDNA showed two intense bands that co-migrated with the S1 and S2 plasmids of cms-S. Further classification into different cms-S subgroups can be done on the basis of restriction digest patterns of mtDNA (Sisco et al., TAG 71:5-19, 1986) or on the basis of presence/absence of a 1.94kb plasmid found in certain cms-S accessions (Carlson and Kemble, Plant Molec. Biol. 4:117-123, 1985). The mtDNA pattern of Cristal appeared to match the *Bam*HI pattern of the "CA subgroup" of

cms-S, but the 1.94kb plasmid was also present as shown by hybridization of the plasmid to a Southern blot of the mtDNA. The mtDNA of Cristal MG III is thus most similar to that of the "J" accession of cms-S (Beckett, Crop Sci. 11:724-727, 1971).

The finding of cms-S cytoplasm in a South American race of maize is of interest both for the origin of S cytoplasm and of the Cristal race. Previous discoveries of cms-S have pointed to an origin in Mesoamerica (Weissinger et al., Genetics 104:365-379,1983; Doebley and Sisco, MNL 63:108, 1989). Nevertheless, Levings et al. (NATO Adv. Inst. Series 31:363-372, 1983) presented evidence that the S1 molecule of cms-S could have arisen from a recombination between the R1 and R2 plasmids found in certain other South American races of maize, such as Racimo de Uva. Thus two possible explanations for cms-S in Cristal MG III are 1) cms-S was created de novo from a recombination between the R1 and R2 plasmids in South America, or 2) cms-S cytoplasm was imported to Brazil from Mesoamerica.

Reduced maps very close to *NPI238* on chromosome 1L
--Paul H. Sisco

To localize reduced on the maize RFLP map, 42 F2 plants having the reduced phenotype were analyzed. (The genotype of the original cross was *rd*-A632^5 x 4Co63). There was no recombination between reduced and *NPI238* among the 42 plants, showing that reduced is very closely linked to that marker in the distal region of chromosome 1L. Recombination data indicated that *BNL8.29A* was the next most distal and *BNL7.25* the next most proximal RFLP marker of the probes tested.

Opaque-10 maps near bronze-2 on chromosome 1L
--Paul H. Sisco

Opaque-10, an EMS-induced mutant generated by M.G. Neuffer, was mapped by him to the long arm of chromosome 1. To more precisely define the map position, we crossed *o10* to a *zb7 bm2* stock and selfed. Data from 176 F2 progeny were analyzed using Mapmaker for MacIntosh v. 1.0b (L. Proctor et al., E.I. duPont, 1990). The most likely map generated was *o10* - 34cM - *zb7* - 42 cM - *bm2*. This would place *o10* close to *bz2*. We are now crossing *o10* to *v22*, *ad1*, and other markers near *bz2*, because *bz2* itself could mask the opaque phenotype.

White luteus-5 maps near brachytic-2 on chromosome 1L
--Paul H. Sisco

White luteus-5 (formerly designated *wl*-266a*), an EMS-induced mutant generated by M.G. Neuffer, was previously reported to be loosely linked to *bz2* but not to *bm2* (MNL 63:140, 1989). In our 1991 nursery we scored 420 F2 progeny from a cross between *br2* and *wlu5*. We were not able unambiguously to score the albino lethal *wlu5* homozygotes for brachytic vs. normal phenotype, but using the ability of the Mapmaker computer program to handle missing data we were able to estimate that *wlu5* is approximately 5cM from *br2*.

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Beyond Mendel: DNA methylation in certain hybrids
--S. M. Kaeppler, G. Holland and R. L. Phillips

Heterosis, paramutation, imprinting, transposable element expression, transgene expression variation, long-range cis effects, "modifying factors"--all of these seemingly enigmatic phenomena found in maize appear to be at least partially controlled by factors beyond the bounds of Mendelian theory. In this article we will describe some preliminary results found during studies on the stability of DNA methylation through sexual generations. We will then relate these results to an allelic "cross-talk" hypothesis which suggests that these diverse observations may be controlled by a fundamental underlying mechanism, a mechanism which may have importance in understanding gene expression at the next level beyond promoters, enhancers and transcription factors.

The study involved five inbreds (B73, Mo17, P3, A679, and A682) crossed in a diallel fashion to produce 25 genotypes. An important component of the crossing strategy was that a single plant was used as a male to produce a self as well as the four respective hybrids. This provided a way to assess the methylation state of the male plant. Five seedlings from each of the 25 genotypes were pooled and DNA from these samples cut with *Hpa*II and *Hha*I; both enzymes are sensitive to CG methylation. Southern blots were probed with 26 single copy DNA sequences including sucrose synthase I, and RFLP probes from Brookhaven National Laboratories, University of Missouri-Columbia, and Native Plants, Inc. Seven of the probes showed non-parental methylation patterns in at least one of the hybrids (Table 1). In all seven cases the allele transmitted through the male was the altered allele; the hybrid made in the opposite direction contained the expected methylation pattern. An alteration occurred in all four possible hybrids only with probe *UMC54*. This indicates that in at least six of the seven cases the changes in methylation occurred after fertilization. B73 was involved as a female in all of the cases of variation; in four of the seven cases other female parents also were involved. These data indicate that there may be a post-fertilization modification of alleles, that the parental origin of the allele is important, and that this situation occurs frequently since random probes testing relatively few potentially methylated sites were used. A related situation is our analysis of tissue culture-derived lines. In these studies we have found methylation to be quite stable upon selfing but subject to change upon crossing back to the non-cultured source (unpublished).

Table 1. Sequences detecting variant methylation patterns in maize hybrids.

Probe	Hybrid(s) containing variant patterns
BNL5.09	B73 x Mo17
NPI112	B73 x P3, Mo17 x P3, A682 x P3
NPI114	B73 x P3, Mo17 x P3, A682 x P3
Sucrose Synthase I	B73 x Mo17
UMC54	B73 x P3, Mo17 x P3, A679 x P3, A682 x P3
UMC85	P3 x A679
UMC175	B73 x Mo17

This type of mechanism may have important implications regarding gene expression, but at this point we can only speculate. However, a "cross-talk" hypothesis presented by Monk (TIG 6:1120-1124, 1990) suggests that similar mechanisms may be acting in a wide range of organisms. The hypothesis states that homologous regions are "compared" after fertilization to determine the degree of genetic and epigenetic relatedness. Detection of differences may result in sequence change or epigenetic modification. Rivin (personal communication) has found that the copy number of tandem repeats in hybrids is often not the mean of the two inbreds as expected. Perhaps this cross-talk involves more than epigenetic modification and is ultimately manifested as chro-

matin or chromosome structure alteration. The finding of long-range cis effects by Schwartz (reports at 1990, 1991 Maize meetings) may imply that this mechanism occurs in a directional fashion along a chromosome and/or that homologous sequences at different chromosome positions are compared as frequently as homologous sequences at a given chromosome position.

As we try to rebuild genomes using DNA transfer technology, it will become increasingly important to understand the influence of position effects, chromatin and chromosome structure, and epigenetic modification on gene expression. In addition to at least partially explaining the phenomena listed at the beginning of this article we may find that these types of mechanisms may allow heritable ways for plants to respond to harsh environments (e.g. tissue culture, nutrient stress) without altering their basic genetic code. Outcrossing among plants could then minimize the effect of drastic changes in a single plant if that plant was uniquely affected by the stress.

Selection of a cDNA expressing lysine-insensitive dihydrodipicolinate synthase activity

--Janita M. Sellner, Robin A. Keith, Burle G. Gengenbach and David A. Somers

Dihydrodipicolinate synthase (DHPS) catalyzes the first enzymatic step specific to lysine biosynthesis. In maize, DHPS is feedback inhibited by lysine (I_{50} = 25uM). In previous studies (Frisch et al., Plant Physiol. 96:444, 1991; Frisch et al., Mol. Gen. Genet. 228:287, 1991), we purified maize DHPS to near homogeneity and isolated a corresponding full length cDNA. The maize cDNA was obtained by gene rescue in an *Escherichia coli dapA*$^-$ auxotroph that completely lacked DHPS activity. We have since used the *dapA*$^-$ strain AT997 to select for maize DHPS cDNAs with altered feedback inhibition properties.

Strain AT997 cells were transformed with the wildtype maize DHPS cDNA cloned into pUC119, mutagenized with EMS and grown on minimal medium such that growth depended on expression of maize DHPS. These cells were then subjected to selection for ability to grow in the presence of 10mM S-2-amino ethyl-L-cysteine (AEC), an analog of lysine that also inhibits maize DHPS activity. After 2 days, a number of colonies of putative AEC-resistant mutants were picked and rescreened for growth on AEC. Single cell isolates were grown and the isolated plasmids were then used to retransform auxotrophic AT997 cells to obtain clonal isolates of the mutant cDNAs.

To test whether the AEC-resistance of these colonies was conferred by a mutation in the maize DHPS gene that caused reduced feedback inhibition, DHPS activity in crude extracts was assayed in the presence of 10 to 1000uM lysine. Figure 1 shows data for two mutants that are representative of six maize DHPS cDNA mutants tested to date. In these tests, the expressed DHPS activity of the mutants was not inhibited by 1000uM lysine compared to >90% inhibition for wildtype maize DHPS at 100uM lysine. These data show a significant reduction in lysine sensitivity between the selected mutants and wildtype, and confirm that the bacterial auxotroph mutant selection protocol effectively identified lysine-resistant maize DHPS alleles. We currently are characterizing the DHPS activity from the DHPS mutant strains to determine the effect of the mutation on substrate kinetic properties of the enzyme.

The DNA sequence of both strands of the DHPS-m1 cDNA clone conferring lysine-insensitive maize DHPS activity was determined by standard methods. Oligonucleotide primers used for sequencing were based on the sequence of the wildtype maize DHPS$^+$ clone. Compared with the parental wildtype cDNA sequence (EMBL Data Bank accession no. 52850), only one nucleotide base change was present in the mutant cDNA region corresponding to the mature DHPS polypeptide. The mutation of C -> T occurred at position 497 resulting in the replacement of alanine (GCA) by valine (GTA) as shown:

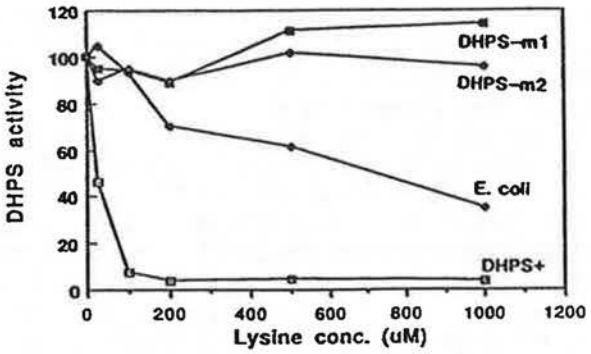

Figure 1. Effect of increasing lysine concentrations on DHPS activity from *E. coli* cells expressing maize wildtype (DHPS+) or mutant (DHPS-m1 and DHPS-m2) DHPS. The wildtype *E. coli* DHPS activity is shown for comparison.

wildtype DHPS cDNA	... 490	GTC	CAC	GCA	ACA	GAA	504 ...
		Val	His	Ala	Thr	Glu	
mutant DHPS cDNA	... 490	GTC	CAC	GTA	ACA	GAA	504 ...
		Val	His	Val	Thr	Glu	

A second maize cDNA isolate obtained after retransformation with the original plasmid also had the same mutation. These comparisons indicate that the single amino acid substitution can confer a high level of lysine insensitivity to the mutant DHPS. Additional lysine-insensitive mutants are being analyzed to fully resolve the nature of the lysine-recognition site(s) of maize DHPS.

A 233kDa subunit of acetyl-CoA carboxylase is encoded by the *Acc1* gene

--Margaret A. Egli, Burle G. Gengenbach, John W. Gronwald[1], David A. Somers and Donald L. Wyse
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Acetyl-CoA carboxylase (ACCase) is a biotinylated enzyme that catalyzes the formation of malonyl-CoA from acetyl-CoA and bicarbonate and is inhibited by several herbicides. Tissue culture selection for herbicide tolerance revealed that a partially dominant mutation in an ACCase gene (*Acc1*) of a selected maize line (S2) confers tolerance to sethoxydim and haloxyfop herbicides at both the whole plant and ACCase activity levels (Parker et al., PNAS 87:7175, 1990). Our eventual goal is to isolate the *Acc1* gene from wildtype maize to better understand its expression and regulation during development of wildtype and mutant maize and to determine its role in fatty acid biosynthesis.

We previously described the purification of a high molecular weight ACCase from maize inbred A619 and the production of ACCase antiserum (Egli et al., MNL 65:95, 1991). The possible existence of ACCase isozymes was suggested by the appearance of two high molecular weight, biotinylated polypeptides on West-

ern blots of crude extracts from inbred leaves (MNL 65:95) and by a report of multiple ACCase activities in hybrid maize (Howard and Ridley, FEBS Lett. 261:261, 1990). We now report that (i) maize leaves contain two ACCase activities that differ in subunit size and sensitivity to herbicide inhibition and (ii) the *Acc1* gene encodes a 233kDa polypeptide.

ACCase was purified from inbred B73 or mutant S2 seedling leaves by ammonium sulfate fractionation, gel filtration (S-400), affinity chromatography (Blue Sepharose), and anion exchange (Mono-Q). The ACCase activity elution profile from Mono-Q was the same for B73 and S2. Two peaks were eluted at approximately 210 (peak I) and 250mM KCl (peak II). Peak II was identical to the purified ACCase fraction that was previously used for antibody production and comprised approximately 85% of the total ACCase activity eluting from the column. Peaks I and II each contained a single biotinylated polypeptide of 225 and 233kDa, respectively. Molecular weights were estimated from 6% acrylamide gels that allowed a better separation than higher percent gels used previously. The 225kDa polypeptide was unlikely to be a proteolytic product of the 233kDa polypeptide because both were found in rapidly prepared crude extracts containing PMSF plus five other protease inhibitors.

Besides ACCase, other biotinylated carboxylases that utilize the substrates propionyl-CoA, methylcrotonyl-CoA, and pyruvate have been reported in plants. However, animal studies indicate subunit sizes of 70 to 125kDa for these carboxylases (Wurtele and Nikolau, Arch. Biochem. Biophys. 278:179, 1990). Neither peak I nor peak II contained biotinylated proteins < 200kDa or utilized methylcrotonyl-CoA as a substrate. However, both enzymes utilized propionyl-CoA approximately 50% as efficiently as acetyl-CoA.

The identity of the 233kDa ACCase polypeptide as the *Acc1* gene product was suggested by several observations. First, high concentrations of sethoxydim and haloxyfop inhibit more than 80% of ACCase activity in crude leaf extracts from wildtype but not herbicide-tolerant maize (PNAS 87:7175). Second, immunoprecipitation with antiserum to peak II ACCase decreased ACCase activity by 75% and preferentially removed the 233kDa polypeptide from the crude extract solution. Conclusive evidence that the 233kDa subunit is encoded by the *Acc1* gene was obtained by measuring the haloxyfop I_{50} values for peak I and peak II from both B73 (haloxyfop-sensitive) and S2 (haloxyfop-tolerant) maize leaves. Peak I ACCase (225kDa) was highly tolerant (I_{50} > 100uM) to haloxyfop in both genotypes and therefore was not correlated with whole plant sensitivity to the herbicide. In contrast, haloxyfop I_{50} values for peak II ACCase (233kDa) were greater in S2 (11uM) than in B73 (< 1uM), consistent with differences in ACCase inhibition previously reported for wildtype and mutant crude leaf extracts (PNAS 87:7175).

Further efforts will be directed towards sequencing and characterizing putative clones of maize ACCase obtained by antibody screening of a lambda gt11 cDNA library from A188 seedlings. Early evidence indicates that the sequence of a partial cDNA clone for maize ACCase is similar to and co-linear with animal ACCases.

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Teosinte glume architecture (*tga1*), a locus from teosinte

-- John Doebley, Jane Dorweiler and Jerry Kermicle

The glumes of the maize ear are normally (1) relatively soft, (2) oriented perpendicular to the axis of the ear, i.e. reflexed, (3) relatively short such that the palea and lemma are readily visible on a shelled cob, and (4) shovel-shaped. In contrast, the glumes of the teosinte ear are (1) highly indurated, (2) oriented parallel to the axis of the ear, i.e. erect, (3) relatively long such that they cover the palea and lemma, and (4) boat-shaped. We refer to the former complex as maize glume architecture (MGA), and, correspondingly, the latter complex as teosinte glume architecture (TGA).

In the process of transferring cross-incompatibility loci on 4S from teosinte into maize inbred W22 (Kermicle and Allen, Maydica 35:399-408, 1990), a factor producing TGA was also transferred into this inbred. In W22 background, the TGA trait is stably expressed. During six generations of backcrossing to W22, the TGA trait behaved like a single locus. In W22 background, TGA is essentially recessive to MGA. Heterozygotes are somewhat intermediate, but much more closely approach the standard W22 phenotype.

We attempted (1) to test if the factor transferred from teosinte to maize would segregate as a single locus and (2) to map its chromosomal location. First, we compared W22 to W22-TGA (W22 converted to TGA) for their genotypes at a series of molecular marker loci on chromosome 4. Molecular marker loci for which these two lines carried different alleles should lie within the chromosome segment transferred from teosinte. The two lines should possess the same allele at all marker loci outside the transferred segment. It was also known that the W22-TGA lost one of the teosinte incompatibility loci that lies 4 map units distal to *su1* during the backcrossing program. Using these two sources of information, we determined the extent of the segment of teosinte chromosome 4 transferred into W22 (Fig. 1). The segment is minimally 32 map units in length.

We also crossed W22-TGA to a *su1* tester and then backcrossed an F1 plant to W22-TGA. One hundred-seventy BC1 plants were grown and classified for glume architecture. In addition, nine molecular marker loci and *su1* were scored in the BC1 plants. Results of this experiment did not tabulate as neatly as originally envisioned. Rather than segregating into two discrete classes (TGA and MGA), a continuous bimodal distribution of glume architecture phenotypes was observed. Thus, we classified plants as either MGA, TGA or intermediate. Sixteen percent (27 plants) of the plants had an intermediate phenotype. We also classified individuals which showed either clear TGA or MGA as having moderate or strong expression of the trait (Table 1).

We considered two possible hypotheses to explain the failure of the glume architecture trait to segregate cleanly. (A) The segment transferred from teosinte contained multiple loci affecting glume architecture and the intermediate class represents individuals with some but not all of these linked loci because of cross-overs within the segment. (B) Glume architecture is largely controlled by a single major locus within the transferred segment, but differences in background (other unlinked loci) between W22

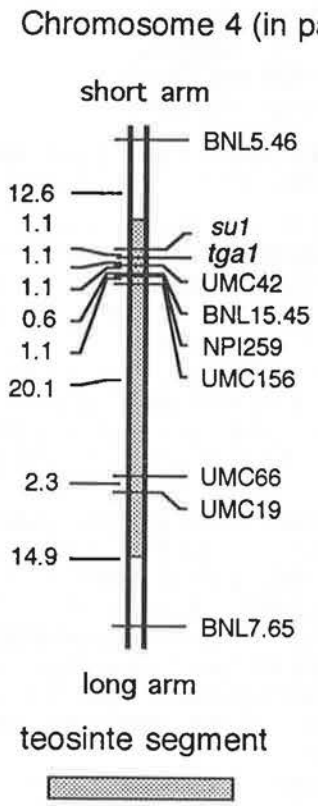

Figure 1. Linkage map of a portion of chromosome 4 showing the position of *tga1* and the marker loci used to localize it. The position of *tga1* should be considered approximate until mapped in a background in which it is stably expressed.

Table 1. Classification of BC1 plants for either teosinte (TGA) or maize (MGA) glume architecture and the degree of expression of the trait.

	Degree of Expression		
Trait	Moderate	Strong	Total
TGA	39	29	68
MGA	49	26	75

and the *su1* tester affect the expression of this putative major locus, giving rise to the intermediate phenotypes. To distinguish between these two possibilities, we examined the amount of recombination between the marker loci for plants in the intermediate phenotype and strong expression classes. If hypothesis (A) is correct then the intermediate individuals should all contain cross-overs within the chromosome segment transferred from teosinte. If hypothesis (B) is correct then the number of cross-overs in this segment should be the same for the intermediate and strong expression classes.

Examination of the data clearly supported hypothesis (B). First, ten of the 27 (44%) plants with intermediate phenotypes contained no cross-overs in the entire transferred segment from *BNL5.46* to *BNL7.65*. Thus, the intermediacy of these plants is not the result of recombination between loci in the transferred segment (hypothesis A). Second, the distribution of cross-overs within the segment in these two groups is equivalent, and the intermediate class does not show significantly greater recombination between any pair of adjacent marker loci (Table 2). Thus, there is no evidence to suggest that the intermediate phenotypes result from break-up of linked loci within the segment transferred from teosinte. Rather, it appears that unlinked factors affect the degree of expression of the glume architecture traits in our population.

Table 2. Percentage of chromosomes with cross-overs between adjacent marker loci in the full population (FP), and in the subpopulations with strongly expressed glume architecture traits (SE) and intermediate phenotypes (IP).

Marker Loci	FP	SE	IP
BNL5.46 - su1	12.6	12.7	18.5
su1 - UMC42	2.3	1.8	0.0
UMC42 - BNL15.45	1.1	0.0	3.7
BNL15.45 - NPI259	0.6	0.0	0.0
NPI259 - UMC156	1.1	1.8	0.0
UMC156 - UMC66	20.1	18.2	22.2
UMC66 - UMC19	2.3	5.5	0.0
UMC19 - BNL7.65	14.9	18.2	14.8

Based on these results, we designate a locus *tga1* (teosinte glume architecture). In W22 background, *tga1* from teosinte produces the phenotype described above as TGA. Our data indicate that *tga1* resides in the interval between *su1* and *UMC42*, approximately 1 map unit from *su1* (Fig. 1). To further investigate these results, we will map *tga1* in a population derived from a cross of W22-TGA with W22. In this population, there will be no background effects and thus all progeny should be readily classified. As discussed elsewhere (Doebley et al., PNAS 87:9888-9892, 1990), this locus on chromosome 4 is one of the key loci differentiating maize and teosinte.

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A repeated sequence derived from the *Arabidopsis* telomere is dispersed in the maize genome as detected by in situ hybridization

--Carol M. Uyeda, Manju Gupta and Dale M. Steffensen

We were interested in obtaining DNA markers adjacent to each of the twenty maize telomeres in order to define the physical ends of the chromosome arms. To accomplish this, we cloned high-molecular weight maize genomic DNA into a selective yeast artificial chromosome (YAC) vector that contains only one telomere. The other telomere would be supplied by maize chromosomes by complementation in yeast. As a result, only those yeast clones that contained both telomeres would propagate in yeast. A chromosome end-specific YAC library of 8,000 clones was generated and screened with an oligonucleotide probe $(CCCTAAA)_6$ derived from the *Arabidopsis* telomeric sequence (ATS). Positive clones were tested for *Bal31* sensitivity, which showed whether the ATS was present at the YAC terminus. The clones showing localization of ATS hybridizing sequences at the ends of the YACs were selected for further work.

To see whether the sequences in the YAC clones that hybridized to the ATS were actually maize telomeres, they would have to be subcloned and used as hybridization probes to maize chromosomes in situ. To bypass the subcloning procedure, we adapted the following strategy: total DNA of yeast strain MY602, which contained a putative telomeric YAC clone, was amplified with a single primer, 5'-d(TTTAGGG)-3', resulting in a few amplified bands. No bands were amplified with this primer from genomic DNA of the yeast strain AB1380, which did not contain any YAC. The amplified DNA from MY602 was fractionated in an agarose gel, the DNA was denatured and transferred to a nylon membrane. The blot was then probed with the ATS. The most strongly hybridizing fragments of 0.7kb and 1.5kb were isolated and used to probe a Southern blot of digested maize (A632

and B73 *Ht rhm*) and yeast (control AB1380, MY602, MY402 and MY407) genomic DNA (Figure 1). The 1.5kb fragment hybridized strongly to one band in B73 *Ht rhm* and to two bands in A632. The probe also hybridized strongly to a few fragments of MY602 DNA which were not present in the AB1380 DNA. This demonstrated that the probe sequence was not specific to yeast genomic DNA, and that it originated from maize DNA sequences cloned in the YAC in MY602. The other amplified fragment of 0.7kb produced the same patterns. The 1.5kb fragment was subcloned in a plasmid vector and the purified insert was labeled with ^{35}S-substituted dATP to a specific activity of ~1 X 10^8 dpm/ug using the Stratagene Prime-It DNA labeling kit. This labeled fragment was used as a probe for in situ hybridization.

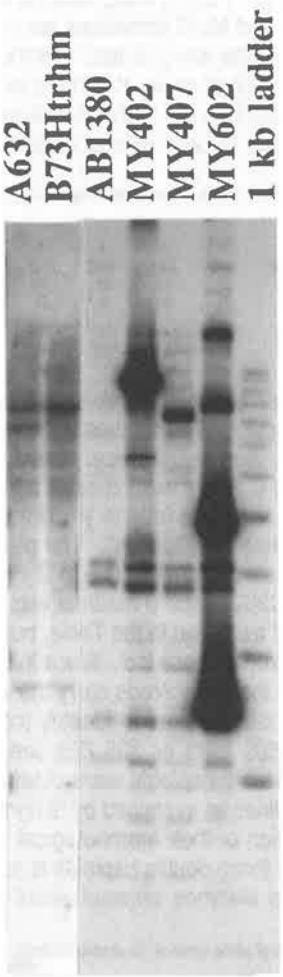

Figure 1. Hybridization of the labeled 1.5kb fragment to *Eco*RI-digested total DNA from maize and yeast. The probe came from the amplification of total DNA of yeast strain MY602 with the *Arabidopsis* telomeric repeat primer. For reference, the smaller of the two most prominent bands in lane #1 (A632) is approximately 8kb in size.

As demonstrated by in situ hybridization to maize chromosomes, the MY602 derived sequence appeared to be distributed over the maize genome with no particular localization at the telomeres. Several of the telomeres (shown in Figure 2D and 2E) were not detectably labeled, while others were. The labeled regions appeared to be randomly distributed over chromosomes and nuclei with no definitive "hot spots", as would be expected for a medium repetitive DNA sequence. The inbred line B73 was quite satisfactory for this study, especially when sporocytes were used to make

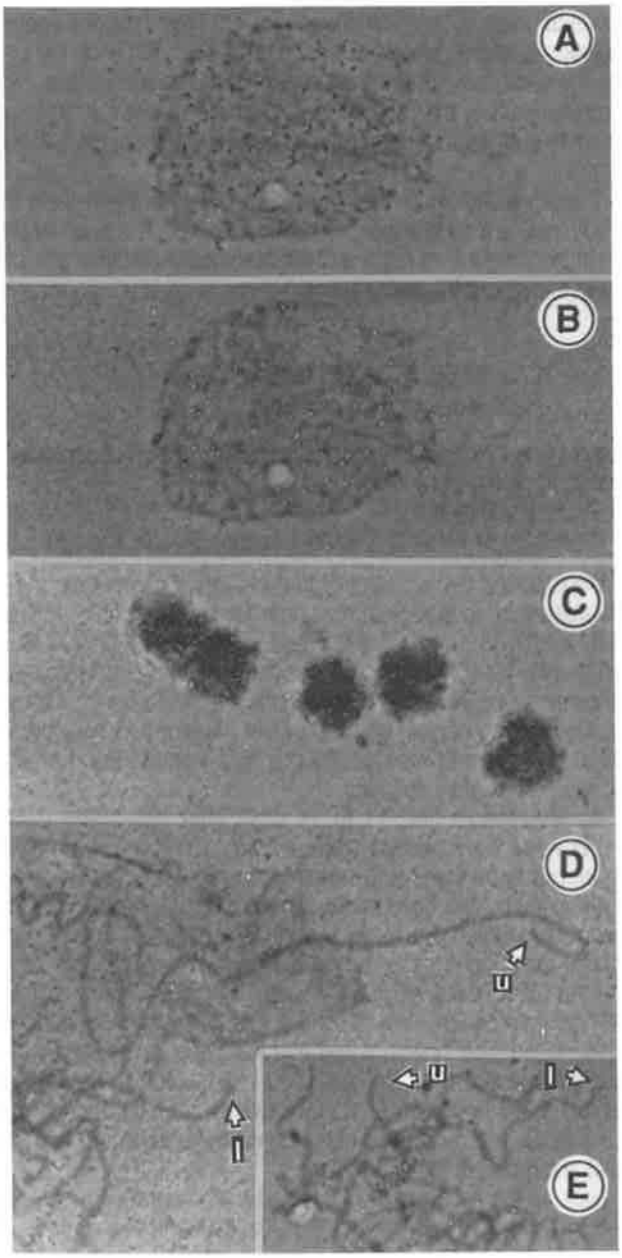

Figure 2. In situ hybridization with a ^{35}S-labeled putative maize telomere sequence: A and B are the same early meiotic nucleus photographed at two levels of focus. In A the focus is on the silver grains (black). In B the focus is on the nucleus, where the silver grains show in a refractive white color (the same in C, D, and E). Panel C shows uniformly labeled tapetal nuclei. D and E are labeled pachytene chromosomes. Unlabeled telomeres are identified as u, and labeled telomeres as l.

B73 tassels at meiotic stages were fixed in 3:1 (v/v) ethanol:acetic acid and slides of sporocytes were prepared as squashes in 45% acetic acid, frozen, dehydrated in ethanol and air dried. The chromosomal DNA on the slides was melted in 70% formamide at 70 C. The double-stranded labeled DNA probe in 50% formamide, 2X SSC and 1mg/ml *E. coli* tRNA as carrier was heated to 70 C and quick-cooled on ice. Hybridization was at 35 C. Slides were dipped in Kodak NTB2 liquid emulsion and exposed for either a week or a month. They were then stained with Giemsa and photographed under phase contrast.

slides immediately after fixation in 95% ethanol:glacial acetic acid, 3:1 (v/v). Unlike older material, freshly fixed meiotic nuclei often popped free of their cell walls. Cell walls will bind probes nonspecifically, thus obscuring the analysis. In nuclei (Figure 2A, 2B, 2C) the silver grains from the probe were distributed evenly and not at the edges where the telomeres would be attached to the nu-

clear membrane. The result shows that ^{35}S provides good enough resolution to decide whether or not the hybridization is on the long arm, the short arm, or at the telomeres of the chromosomes. Our calculations show that a specific activity of at least 1 X 10^8 dpm per ug is sufficient to detect single copy sequences of 1-2kb in length.

These results show that the 1.5kb DNA fragment from the MY602 YAC is a middle repetitive sequence, which is not only present in the subterminal regions of the chromosomes but is also dispersed throughout the maize genome.

SASKATOON, SK, CANADA
Plant Biotechnology Institute

Microsurgery of immature embryonic axis and recovery of plants through in vitro culture

--V. R. Bommineni*
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We reported the recovery of mature, fertile plants from the in vitro culture of shoot apical meristem for a variety of genotypes (MNL 63:87-88, and 64:79; Plant Cell Tissue Organ Cult. 19:225-234, 1989). In these dissections, the shoot apical meristem dome was exposed by removing the leaf primordia layers without disturbing the apical meristem cells. This report considers a culture technique to recover the plantlets from bisected embryonic axes of immature embryos.

As reported earlier, 11 to 14 days after pollination (DAP) ears of A188 were surface sterilized with 10% 'Javex' for 20-25 min, rinsed three times with sterile water, and embryos were dissected in the culture hood under the microscope. A single median longitudinal incision was made through the embryonic axis by using a sterile razor blade. The two halves of each embryo were separated and placed on MS medium with no plant hormones (MNL 63:87-88, and 64:79). In a second series, the median longitudinal incision was made in such a way that the bisected embryonic axis was kept intact at the basal surface of the scutellum (referred to as graft sections in Table 1). Two weeks after our standard in vitro culture, the recovered plantlets were transplanted into soil (pots) and grown in the glasshouse.

Data on the recovery of plants and their maturity are summarized in Table 1. No difference was noticed between explants of separated, or grafted sections. A high percentage (50%-75%) of bisected explants developed into two plants, but only 10-15% of these plants grew to maturity. Some abnormal plants were noticed from this experiment (Table 1).

Table 1. Recovery of plants from bisected longitudinal sections of immature embryonic axes of maize (A188).

	a*	b		c		d	e**
		(b1)	(b2)	(c1)	(c2)		
Grafted sections (placed together)	64	30	33(52)	35	6***(18)	9(19)	6(13)
Separated sections	79	17	61(77)	48	5****(8)	6(10)	14(24)
Control (embryos)	14	14	0	13	0	0	0

*a = number of embryos cut in half (longitudinal); b = number of plants transferred to peat pots - (b1) number of single plants, and (b2) number of paired plants (percent of paired plants - b2 x 100/a); c = number of plants matured - (c1) number of single plants, and (c2) number of paired plants (percent of paired plants - c2 x 100/b2); d = number of terminal node tassel/ear plants (percent of tassel/ear plants - d x 100/c); e = number of Abphyll plants (percent of Abphyll plants - e x 100/c).

**Origin of Abphyll ranges from 2nd to 5th node from top of the plants.

***Of the six, two are terminal node tassel/ear plants and two others are Abphyll plants.

****Of the five, two are terminal node tassel/ear plants and one other is Abphyll plant.

SLATER, IOWA
ICI Seeds, Inc.

Stock 6 induced double haploidy is random

--Ming T. Chang

An experiment was designed to generate double haploids from Stock 6. Two inbreds that were derived from Oh43 and Mo17 were crossed and the F1 seeds were used as female stock for this study. These Oh43 and Mo17 derivatives are homozygous pure inbreds as shown from the isozyme test, and their genetic constitution is homozygous *A b pl c1 r-g Y*. These two lines carry different allelic forms at isozyme loci *Mdh1*, *Mdh2* and *Idh2* as shown in Table 1. The pollen source was BC2S2 materials that were derived from crosses between Stock 6 (*A b pl C R-g y*) and *A B Pl C R-nj y* genetic stock. The F1 was backcrossed to Stock 6 (*A b pl C R-g y*) twice, then selfed twice. Seeds from the homozygous ears of *A B Pl C R-nj y* were chosen as materials for pollen source. The seeds are white (due to *y1*) flint with purple color on the crown and plumule (due to *R-nj*). The plants are dark purple on leaves, stalk, tassel and anther (due to *B Pl*). The isozyme test showed that the BC2S2 Stock 6 material was still segregating at *Adh1* and *Pgm2* loci as shown in the Table, but 90% of the individuals were already fixed at those loci. Since the Stock 6 carries the *Pgd1-22* allele and the two inbreds carry the *Pgd1-11* allele, the isozyme difference can be used to identify the origin of the haploids. A total of 600 ears or 305,778 seeds were obtained. Among those, 98 double haploids were obtained. These double haploids were pure lines as examined by isozyme markers and ear to row field evaluation of their morphological uniformity. The allelic constitutions of these double haploids is summarized in Table 2. Genetic linkage distance between *Idh2-Mdh2* is 1.1 ± 0.6 (Goodman et al., Genetics 96:697-710, 1980). Among these 98 double haploid lines, all showed tight linkage and none were derived from a crossover between *Idh2* and *Mdh2* loci on chromosome 6. It is therefore concluded that *Idh2* and *Mdh2* are tightly linked. The distributions of allelic frequency among these double haploids were *Mdh1-11* (48%) vs. *Mdh1-22* (52%), and *Mdh2-11---Idh2-22* (54%) vs. *Mdh2-22---Idh2-11* (46%). This is almost a 1 to 1

Table 1. Genetic constitution of allelic forms of isozyme loci of the male and female materials that were used to generate double haploids.

Chromosome no.	8	1	1	8	6	6	3	1	5	6
Material	*Acp1*	*Acp4*	*Adh1*	*Mdh1*	*Mdh2*	*Pgd1*	*Pgd2*	*Phi1*	*Pgm2*	*Idh2*
S6/ *R-nj*	11	22	22	22	22	22	11	22	22	22
	11	22	11	22	22	22	11	22	11	22
Oh43 derivative	11	22	22	11	11	11	11	22	22	22
Mo17 derivative	11	22	22	22	22	11	11	22	22	11

Table 2. Genetic constitution of allelic forms of the double haploids.

Double Haploids (Number)	Chromosome no.	Genetic Constitution 8	6	6	6
		Mdh1	*Pgd1*	*Mdh2*	*Idh2*
28		11	11	11	22
19		11	11	22	11
26		22	11	22	11
25		22	11	11	22

ratio due to genetically random assortment of the chromosomes. It is therefore concluded that Stock 6 induced double haploidy is a random process.

Preferential fertilization induced from Stock 6

--Ming T. Chang

A project was conducted in 1988 aimed at rapidly generating pure inbreds through double haploids. These double haploids are genetically induced by Stock 6, a line which has been reported to generate a high frequency of haploids. Two inbreds that were derived from Oh43 and Mo17 were crossed and the F1 seeds were used as female parent. The genetic constitution of the hybrid is homozygous *A b pl c1 r-g Y*. So the hybrid kernels have a deep yellow color and the plants have green leaves, sheath, tassel and anther. The male is a BC2S2 that was derived from crosses between Stock 6 (*A b pl C R-g y*) and *A B Pl C R-nj y* genetic stock. An isozyme test showed that the BC2S2 material was still segregating at *Adh1* and *Pgm2* loci (see above article), but 90% of the individuals were fixed at those loci.

A total of 600 ears (305,778 seeds) were obtained from crosses between the F1 hybrid and BC2S2 Stock 6. In theory, the seeds on the ear should all be purple crown-purple plumule (due to *R-nj*) and light yellow (due to 2 doses of *Y*). But some kernels were colorless (due to contamination or development of the unfertilized endosperm). The contaminant kernels should be colorless (due to *r-g*) and deep yellow (due to 3 doses of *Y*), and could be easily identified. Using this endosperm color screening technique, the contaminated kernels were first removed from the ear. A total of 678 contaminants (0.22%) were identified, of which 75 kernels had purple plumule color, the remaining 603 kernels had colorless plumules. These 75 kernels were the result of heterofertilization. One sperm from Stock 6 pollen fertilized the egg and one sperm from F1 pollen fertilized the polar nuclei.

Among the remaining 305,100 kernel samples, 4,471 kernels (1.5%) had colorless endosperm and colored plumule, and 3,575 kernels (1.2%) were defective (Figure 1). These 4,471 colorless kernel samples had light yellow endosperm (due to 2 doses of *Y*) and plants were purple in color. Kernel size was slightly reduced but the difference was not significant. These kernels should be derived from the development of fertilized egg with unfertilized endosperm, although the chromosome ploidy of endosperm tissue was not determined. The defective kernels were not able to germinate. The origin of these defective kernels was not known.

The remaining purple crown (due to *R-nj*) seeds were then screened for embryo plumule color. Among those seeds, we discarded 295,324 (96.8%) which had colored plumule. 1,730 (0.57%) seeds that had colorless plumule (Figure 1) were planted and at 2 to 3 leaf stage, a 0.05% colchicine solution was applied. A total of 23 mutants were obtained from these seedlings. Among those mutants were 3 albino, 7 white stripe, 1 dwarf, 4 yellow green, 5 pale green, 2 necrotic crossband, and 1 tiny seedlings. The surviving 1,506 plants were grown to maturity. Among those, 485 plants proved to be purple hybrids due to crosses between the F1 hybrid and Stock 6. However, 249 green plants did set yellow seeds (presumed to be double haploids), and the remaining 772 plants were 100% sterile (presumed to be haploids). The 772 haploid plants were further classified into two types, 760 small sterile green plants and 12 small sterile purple plants. Root tip examination from 10 plants showed that they were all haploids with 10 chromosomes. RFLP examination from another 10 plants

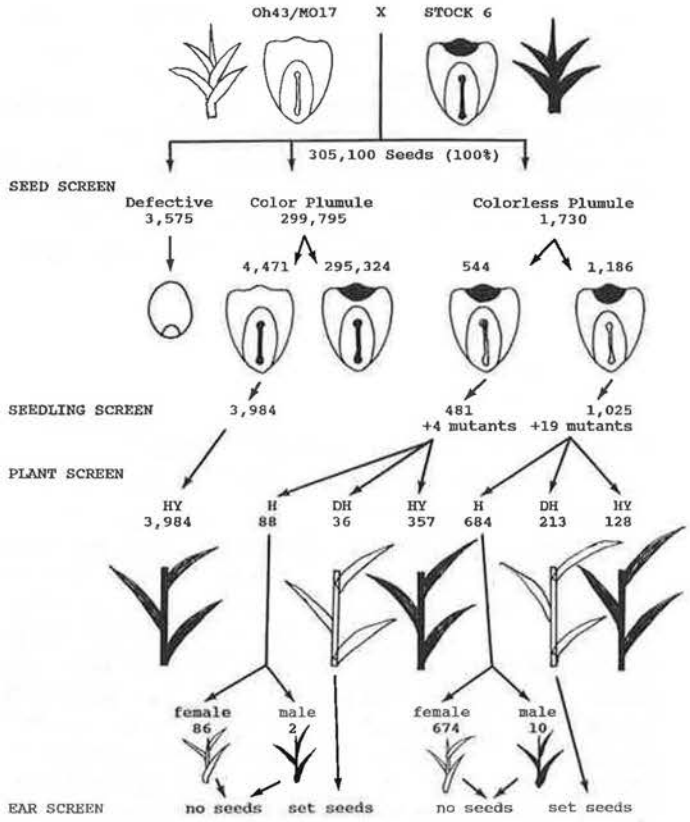

Figure 1. A schematic procedure for double haploid production using Stock 6. H = haploid, DH = double haploid, HY = hybrid.

showed a single band at all ten loci. It is therefore concluded that these small seedlings are true haploids. The parthogenetic and androgenetic ratio from Stock 6 is 760 vs. 12, or 98.45% vs. 1.55%.

The homozygosity of these 249 putative double haploid lines was examined by isozyme analysis. Ten seedlings from each line were examined for 10 different isozyme loci. Among those, 98 lines were proved to be homozygous pure inbred lines. The remaining 151 lines were segregating at 1, 2, or 3 isozyme marker loci as shown in Table 1. Results clearly showed that these plants were not derived from Stock 6. It was clear from isozyme analysis that these plants came from self-contamination. Since they had light yellow (due to *y*) and purple crown (due to *R-nj*) endosperm, it was evident that these seeds were the result of heterofertilization. The embryo was selfed and the endosperm was fertilized by Stock 6. All 249 lines were planted ear to row in 1989. Morphological uniformity was evaluated. Based on isozyme and morphological data, only 98 lines were pure inbreds and the rest were hybrids.

The number of haploids now equals 772 haploids + 98 double

Table 1. Isozyme analysis of the putative double haploids.

		Genetic Constitution			
Material	Frequency	*Mdh1*	*Mdh2*	*Pgd1*	*Pgd2*
Stock 6		22	22	22	22
Oh43 derivative		11	11	11	22
Mo17 derivative		22	22	11	11
Hybrid x Stock 6	23	11	12	11	12
	26	22	12	11	12
	25	12	11	11	22
	20	12	22	11	11
	57	12	12	11	12

haploids = 870, or 0.29% (870/305,100). That means 0.29% of the pollen grains preferentially fertilized the polar nuclei. If all of the 4,471 (1.47%) colorless endosperm colored plumule kernels are diploid embryos with unfertilized diploid endosperms, then the pollen grains must have preferentially fertilized the egg first. Thus, if lack of fertilization is the cause of haploidy, then when a single sperm goes inside the embryo sac the chance of fertilizing the egg first is 83.71%, and the chance of fertilizzing the polar nuclei first is 16.29%. It is therefore concluded that a single sperm alone will preferentially fertilize the egg.

STANFORD CALIFORNIA
Stanford University

"Pink" kernels in *bz2* testers--a connection to ABA levels?

--Virginia Walbot, Juli Nash and Maria-Ines Benito

The *bz2* tester lines we have received (in W23 or K55 backgrounds from Ed Coe; *bz2* in combination with diverse sources of novel *Adh1* alleles from Mike Freeling) or constructed (various exotic backgrounds) exhibit a sporadic expression of medium to dark pink kernel color. Typically just a few kernels per ear are affected, but in some ears most kernels are pink; some ears in the same family will have no pink color. The hue of the darkest pink kernels is annoyingly similar to dilute (poor) expression of *Bz2*. We do not have this problem with testers for any other gene of the pathway. Crosses with plants derived from *bz2* kernels selected as pink, dark bronze or the light, standard bronze color did not resolve the nature of the pink phenotype other than indicate that some lines were more prone to pink coloration. At the suggestion of J. D. Smith who paints ABA onto developing kernels to overcome vivipary, we treated *bz2* ears in summer 1991 with this hormone at a developmental stage when ABA is known to penetrate into the developing seed. Longitudinal quadrants representing four treatment conditions were created on each ear: exposure to light, light + ABA, dark + ABA, and the dark only control. We cut through the husks 12-14 dap of ears in the hybrid W23/K55 background to expose kernels on three-fourths of each ear. We put a strip of folded paper towel wetted with 10^{-4}M ABA on half of the ear; the husks were then smoothed into place over one-half of the towel and secured in place by a rubber band. The brown paper pollinating bag was replaced and nothing further done. At harvest about half (9/16) of the well-filled, treated ears had a clear sector of dark pink kernels in the ABA + light quadrant and an enhanced number of pink kernels in the dark + ABA quadrant. The other treated ears lacked such a clear demarcation of the treatment areas but had an enhanced number of pink kernels in one or more patches. Parallel treatment of *a1* ears in a similar background yielded only the expected colorless kernels. Tests are in progress in Hawaii with *r-g, c1, c2, a2, bz1,* and *bz2* testers to confirm our findings and determine whether the ABA effect is specific to *bz2*.

Our observations suggest that high levels of ABA can induce the pink phenotype in a *bz2* tester. A plausible explanation for the sporadic appearance of pink kernels would be that a variable number of kernels per ear experience sufficient stress, probably water deficit, to increase endogenous ABA to a level sufficient to cause the pink phenotype. If this model is true, then many lines of maize contain an ABA-inducible gene that can carry out BZ2 function, albeit at a very low level as pink rather than purple pigmentation results. Based on genetic criteria, *Bz2* encodes the terminal step of anthocyanin synthesis in maize. This step could be a malonyl transferase or other enzyme that decorates the glucose moiety to make a more stable molecule; the enzyme giving the pink phenotype would then be postulated to normally carry out a similar reaction on a different substrate but when induced to high levels by ABA and in the presence of substantial anthocyanin precursor, the "pink" enzyme catalyzes the BZ2 reaction. Alternatively, *Bz2* could encode a protein that guides anthocyanin into the vacuole from its site of synthesis in the cytoplasm, and the "pink" product performs a similar task for a related molecule. To date, *Bz2* shows the highest similarity to a stress-induced gene of soybean, *Gmhsp26* (Nash and Walbot, this issue), but the biochemical function of this gene is also unknown.

Early sectoring and germinal reversion in a Mutator line with the *bz1-mu1* reporter allele

--Virginia Walbot

In MNL 64:107 I reported the isolation of a line carrying the *bz1-mu1* reporter gene, an allele with a *Mu1* insertion, that exhibits two properties atypical of Mutator lines: early sectoring and an increased germinal reversion rate. The early excision stock was grown in an isolation plot in 1990, and 750 ears have been analyzed from this population thus far. Germinal reversion is ~0.1%, occurring almost exclusively as single kernel events. Only a few ear sectors have been recovered: one encompassed half the ear, and the others contained only a few kernels. The events fit a Poisson distribution suggesting that reversion events are independent. This is consistent with the hypothesis that the majority of the events occur through the pollen. This has been confirmed in 1991 by reciprocal crosses between a *bz1* deletion stock (gift of Hugo Dooner) and the *bz1-mu1* early excision line. Approximately 90% of the revertants recovered were from the pollen parent. Interestingly a similar frequency of germinal reversion (0.1%) was observed in a population of plants grown from *bz1-mu1* kernels that had a large *Bz1* sector (1/8 to 1/2 of the kernel) and from a population of kernels with the standard spotting pattern from the same parental ears. This suggests that genetic elements required for both large spotting (10^{-4} frequency of half-kernel sectors) and germinal reversion (10^{-3}) are present in kernels with either the standard or the big spot phenotype.

To test the timing of germinal reversion, *Bz1'* germinal revertants from the open-pollination experiment were planted in 1991 and pollinated by the *bz1* deletion stock; all 90 resulting ears transmitted the purple phenotype, indicating concordance between endosperm phenotype and embryo genotype. Consequently, *Mu1* excision occurs before the second mitotic division in microgametogenesis. In examining a small population of early excision *bz1-mu1* with *R-r* present, I found no sectors as large as an entire anther. In fact, the excision pattern of tiny dots appeared to be the same as found in the standard *bz1-mu1* line in both the anthers and in the plant body (*B pl*). These data suggest that reversion is very late in the sporophyte and hence typical of standard Mutator lines.

What is different about the big spot line is that excision occurs at a higher frequency during microgametogenesis and that this behavior carries over into the first few mitotic divisions in the aleurone to produce some half, quarter and eighth kernel sectors. It is noteworthy that the next size classes of sectors are one to

two orders of magnitude less frequent, until after cell division 10. In both standard and the *bz1-mu1* big spot line Mutator is "activated" at this stage, producing a high frequency (up to the percent range) of sectors containing 1 - 128 cells.

At the molecular level, both the early excision and standard *bz1-mu1* lines contain >50 copies of *Mu1* and >20 copies of *Mu9*, the putative regulatory element for the Mutator family (Hershberger et al., PNAS 88:10198, 1991). The *bz1-mu1* allele appears unchanged. Tests are in progress to determine whether early sectoring is dominant or recessive by crossing this stock to Mutator lines carrying other mutable alleles.

Early sectoring and germinal reversion in a Mutator line with the *bz2-mu2* reporter allele

--Virginia Walbot

In MNL 65:96-97 I reported the isolation and initial characterization of a Mutator line carrying the *bz2-mu2* reporter allele that had exceptionally early sectoring, many large null sectors on kernels, and a 4% frequency of germinal reversion to *Bz2*. One observation in 1990-91 was that the stock appeared to be losing Mutator activity based on an excess of *bz2* kernels and the presence of weakly active (very fine spotting pattern) kernels. Further crosses with large spotted *bz2-mu2* and *bz2-mu2/bz2* individuals indicated, however, that at least some plants were segregating a single factor required for Mutator activity. Southern blot hybridization indicates that these individuals contain only a single copy of *Mu9*, our candidate for the regulator of Mutator (Hershberger et al., 1991, and unpublished data of C. A. Warren). In addition these lines contain only one or a few copies of *Mu1*, the element in the reporter gene. The segregating *bz2-mu2* lines are identical at the genetic level to the *a1-Mum2* lines showing single copy inheritance of Mutator activity described by Robertson and Stinard (Develop. Genet. 10:282, 1989) and by Chomet et al. (Genetics 129:261, 1991; note that their line had only one *Mu1* element in it as well). Our *Mu9* probes identify a single copy of a *Mu9* element in Robertson's *a1-Mum2* line.

The correlation between a single copy of *Mu9* and the big spot phenotype is less clear, however. The *bz1-mu1* big spot stock has many copies of *Mu9* and *Mu1*. Neither Robertson and Stinard nor Chomet et al. comment on early somatic sectoring in the *a1-Mum2* lines. This feature in the *bz2-mu2* big spot line is completely atypical of standard Mutator lines and would be readily noticed with any anthocyanin reporter allele. It is possible that an additional factor--the background of our lines, the "phase" of *Mu9*, or another gene--is responsible for the early sectoring phenotype. In crosses with other Mutator lines containing diverse reporter alleles, the big spot phenotype is "dominant" if the big spot line is used as the female. In crosses in which the F1 progeny were purple and could not be scored for excision behavior, i.e. *Bz2/bz2-mu2 A2/a2-mu1*, F2 crosses to the respective testers showed retention of the big spot phenotype on many *bz2* ears in the W23 background and sporadic occurrence in the *a2* crosses in a different inbred line. Examples of the big spot phenotype transferred to lines with other reporter alleles are being grown now to test for transmission to the next generation.

As for the timing of somatic and germinal excision events, *bz2-mu2* contrasts sharply with the *bz1-mu1* big spot line (this volume). First, early excision of *bz2-mu2* is visible in the plant body with *B pl* as large stripes, tassel branches, clusters of anthers, individual anthers and in one instance a half-tassel on a tiller. The half-tassel produced just over 50% revertant progeny in a cross to *bz2*. Plants with several large sectors in the tassel also produced many germinal revertants. These observations suggest that in the *bz2-mu2* line some excision events occur in the apical meristem cells or their immediate derivatives to produce large sectors. Many small sectors are also present in the soma and in the aleurone indicating that events occur throughout development. Second, if there is a bias between the ear and tassel in producing germinal revertants it is more subtle than in the *bz1-mu1* line and much more complicated to follow as revertants do not follow a Poisson distribution.

Third, in selections of independent *Bz2'* revertants, picked as individual kernels from different *bz2* X *bz2-mu2/bz2* crosses, I found non-concordance between the endosperm phenotype (purple) and embryo genotype (*bz2* or *bz2-mu2*) 19% of the time (15 cases/79 trials). This means that about one-fifth of the germinal revertants result from events in pollen that produce sperm of two genotypes. There are several possible scenarios. Excision could occur in one sperm nucleus but not the other. If true, then because there is 4% reversion to purple by scoring the aleurone we should expect that among the kernels with a spotted aleurone, there should be a similar percentage with a *Bz2'* embryo. Such an embryo should yield a revertant plant, i.e. purple body and 50% purple progeny kernels (parent was *Bz2'/bz2* by virtue of the original cross). To date, I have recovered only two such cases, too few given the populations examined to conclude that excision in individual sperm nuclei is the only mechanism underlying non-concordance.

But consider another surprising fact: 9/15 of the non-concordant cases transmit *bz2* not *bz2-mu2*. As the pollen is already haploid, the *Bz2'* revertant allele must arise from the mutable allele. This means that in the majority of non-concordant cases and in 11% of the total population (9/79) *bz2* alleles are created in the pollen by excision or by loss of *Mu9*. The excision hypothesis could encompass cases [1] that involve "mistakes" in DNA repair occurring from a single excision event in the germinal nucleus that are repaired to *Bz2'* or *bz2* as a consequence of DNA replication prior to sperm formation and [2] a contribution from excision in individual sperm nuclei that leads to either *Bz2* or *bz2* alleles. Both scenarios predict that among the kernels with a bronze phenotype, there will be some with a *Bz2'* embryo. The number of *Bz2'* embryos among progeny with spotted and bronze-colored aleurones will be determined in a large population in 1992. The excision scenario also predicts that all of the *bz2* cases will represent *Mu1* excision events from the reporter allele; this can be tested by Southern blotting. The *Mu9* segregation, or change of phase, scenario will be tested on these same Southern blots; in this scenario the *bz2* kernels are actually *bz2-mu2* but *Mu9* is not present or is non-functional. It may also be instructive to test what fraction of the *Bz2'* derivatives also lack *Mu9*.

The 6/15 non-concordant cases that transmit *bz2-mu2* most likely represent the true, independent cases of excision in individual sperm nuclei (6/79 = 8%). This is a high percentage of events in a single cell type. Concordant cases (81%, 64/79) represent the majority of cases. These most likely represent excision events that precede pollen formation or events that occur early in gametophytic development and hence result in a single allele in the sperm. As the big spot *bz2-mu2* stock produces purple anthers and tassel sectors of all sizes, it is likely that excision events occur throughout tassel development.

Revertants and recombinants in *bz2* alleles with *Mu* insertions

--Virginia Walbot

Heterozygotes were constructed between different mutable alleles of *bz2* containing *Mu* insertions:

bz2-Mu1/bz2-mu2	both *Mu1* insertions, approx. 800bp apart
bz2-Mu1/bz2-mu3	both *Mu1* insertions, approx. 400bp apart
bz2-Mu1/bz2-Mu9	*Mu1* and *Mu9* insertions, approx. 300bp apart
bz2-mu2/bz2-mu3	both *Mu1* insertions, approx. 400bp apart
bz2-mu2/bz2-Mu9	*Mu1* and *Mu9* insertions, approx. 500bp apart

In a population of 194,600 kernels from active lines 3 purple kernels were found (1.5×10^{-5}) representing revertants, recombinants, and/or gene conversion events. Similar lines with an inactive Mutator system were screened yielding 2 purple kernels among 377,900 progeny (5.3×10^{-6}) from recombination and/or gene conversion events. From these data it appears that an active Mutator system does little to enhance intragenic recombination and/or gene conversion events. Dooner and colleagues have demonstrated a similar lack of effect of *Mu1* insertions (Dooner and Ralston, Maydica 34:333, 1990). They concluded that *Mu1* insertions act in a manner similar to point mutations, in contrast to *Ds* insertions that appear to favor the recovery of conversion events compared to intragenic recombinants (Ralston et al., Genetics 119:185, 1988; Dooner, Genetics 113:135, 1986).

Anthocyanins protect DNA from UV damage

--Ann E. Stapleton and Virginia Walbot

Solar radiation is both a source of light for photosynthesis and UV that can damage plant DNA. Maintaining the integrity of DNA is of critical importance to all organisms. Plants use both DNA repair and various shielding strategies to minimize DNA damage; relatively little is known about the biochemical basis of irradiation-induced DNA damage in plants (McLennan, DNA Replication in Plants, p. 135, 1987).

UV light is generally classified into 3 subgroups: UV-A (320-390nm), UV-B (280-320nm) and UV-C (less than 280nm). The absorption spectrum of DNA includes wavelengths from 240 to 310nm; the level of solar UV that reaches the surface of the earth is high in the UV-A region of the spectrum, decreases sharply in the UV-B range, and drops to nearly zero by 290nm (McLennan, DNA Replication in Plants, p. 135, 1987). Most studies of the effects of UV light have used wavelengths of 254nm, in the UV-C region, although this wavelength is extremely rare in sunlight.

UV-C and UV-B produce DNA damage primarily via the formation of cyclobutane pyrimidine dimers (PDs). Pyrimidine dimers are 80%-90% of the UV-light induced DNA photoproducts, with most of the rest being pyrimidine(6,4)pyrimidone (Franklin and Haseltine, Mut. Res. 165:1, 1986). The epidermis of plants absorbs 95%-99% of incoming UV light; flavonoid compounds (such as anthocyanins) and cuticular waxes are the agents of UV absorption (McLennan, DNA Replication in Plants, p. 135, 1987). Because anthocyanins and other flavonoids absorb light in the UV-B range, it is commonly suggested that these compounds shield plant DNA from damage. However, this hypothesis has not previously been tested.

We extracted anthocyanins from the purple maize line K8 (an inbred in the W23 background) which contains all the structural genes required to produce anthocyanins and the genes *B* and *P1* that confer anthocyanin expression on nearly every tissue of the plant. For An1 prep 5g husk tissue was frozen in liquid nitrogen, ground to a powder, and mixed with 1% HCl in methanol. After incubation overnight at 4 C and 3 changes of extraction buffer the final volume was 45ml; this was filtered through Whatman #1 to remove particulate matter and stored at 4 C. For An2 prep 5g husk tissue was finely chopped and extracted with 45ml 8:1:1 methanol:acetic acid:water. The resulting solution was passed over a Sephadex LH20 column to remove sugars and stored at 4 C. Quercetin (Sigma) was used from a stock solution of 10mg/ml in methanol.

Genomic DNA was prepared from BMS cell cultures and irradiated. To test the ability of flavonoids to shield DNA, anthocyanin extracts or quercetin solutions were sealed between quartz plates with beeswax for irradiations. For UV-C irradiation we used one GL-15 germicidal lamp positioned 12cm above the sample, with an output of 30 J/m^2/sec. For UV-B we used a TR302 transilluminator with filter, output rated as 8 J/m^2/sec and a simulator of solar UV-B, output rated as 1 J/m^2/sec. We specifically nicked the DNA near the pyrimidine dimers using T4 endonuclease V, which was kindly supplied by Dr. P. C. Hanawalt. DNA was treated for 15 minutes at 37 degrees in 10mM Tris pH 8.0, 100nM NaCl, 10nM EDTA, 1mg/ml BSA, 2 microliters T4 endo V (lot 25). Alkaline loading dye was added to DNA samples to denature them; DNA was size fractionated on 0.6% alkaline gels with running buffer composed of 30mM NaOH, 1mM EDTA. The gels were neutralized, stained with EtBr, photographed, and the amount of unnicked DNA quantitated by densitometry.

The anthocyanins that were isolated from purple maize husk tissue by either of the two extraction methods absorb in the UV range. When these anthocyanins (at a concentration approximately equal to 1/3 the concentration in K8 plants) are interposed between DNA and a UV source they protect the DNA from PD formation (and thus from nicking by T4 endo V) (Table 1). This protection decreases in proportion to increase in dose. Quercetin also protects DNA from damage.

Table 1. Percent of control band densitometer trace of BMS genomic DNA irradiated with UV-B solar simulator (with and without anthocyanin shielding).

	0 min	40 min					120 min			
T4endoV:	-	+	+	+	+	+	+	+	+	+
Antho:				An1	An2	Q		An1	An2	Q
Expt. 1	79±1	100	17	86	68	50	0	35	30	28
Expt. 2	104 ±4.3	100	23	83	83	56	11	69	43	21

Control (0 min) and two doses of UV-B were used with the same anthocyanin shielding (An1 and An2) and quercetin shielding (Q). Columns without An1 or An2 or Q headings were unshielded. T4 endo V was omitted in the (-) lane; two or three identical such lanes were run in each experiment to measure loading variation; the ± indicates the variation in percent in these two or three lanes. All data were normalized to the 0 min, plus T4 lane.

When sections of leaf tissue from purple and green plants are irradiated with high doses of UV-C or UV-B and the level of PDs assayed as described above, we find that the purple tissue shows less damage than green tissue. This demonstrates that anthocyanins also protect DNA from UV damage in vivo.

Endogenous genetic regulation of anthocyanin production in embryogenic callus

--John P. Bodeau and Virginia Walbot

Embryogenic ("type II") maize callus cultures are generally colorless, but calli of certain genotypes will synthesize anthocyanin either in response to light (G. Dietrich, unpublished data), or following particle-gun introduction of constitutively expressed *R* and *C1* gene-family members (Goff et al., EMBO J 9:2517). We

want to better understand the endogenous genetic regulation of anthocyanin pigmentation in embryogenic callus, with two primary goals in mind. 1) By determining which tissue-specific regulatory alleles enable pigment synthesis, we may learn which natural tissue is most closely related to embryogenic callus. 2) After we know the regulatory alleles required for anthocyanin synthesis in callus, we may initiate embryogenic lines lacking a single structural or regulatory gene, in theory allowing any of these genes to be used as a visual marker for transformation.

We began in 1989 by crossing several pigment tester lines to the inbred line A188, from which embryogenic callus is easily initiated. A188 has the regulatory genotype *r-r c1 b pl*, with wild-type alleles of the structural genes *C2, A1, A2, Bz1, Bz2*, and *Pr*. Thus it is colorless in all tissues except for pale pink anthers and red seedling leaf sheaths; embryogenic A188 callus is colorless under all growth conditions we have tested, including light and cold treatments. The alleles introgressed included *R-r, R-g, R-nj, R-scm2, r-g, B, B-peru, C1, C1-S, C1-I, Pl, c2, a1, a2, bz1, bz2*, and *an-bz2-6923*. These alleles came from a variety of backgrounds, predominantly W23 and K55, which are virtually incapable of giving rise to embryogenic callus. By a program of repeated backcrosses to A188, we have produced several genotypes having from 50% to 87.5% A188 nuclear background, from which type II embryogenic calli were initiated in August, 1991.

Lines with a higher fractional component of A188 were significantly more efficient at initiating type II embryogenic callus (Table 1). The identity of the introgressed background also affected

Table 1 Percent of immature embryos which initiated embryogenic type II callus as a function of introgressed background.

Fraction A188:	1.0	.875	.75
A188 inbred	42% (n=36)		
W23		48% (n=205)	42% (n=115)
K55		35% (n=48)	0% (n=25)

initiation efficiency: K55-containing material was relatively inefficient, while W23-containing lines were at least as efficient as pure A188. Our attempt to improve the callus-induction efficiency of desired genotypes by a backcrossing program to A188 was thus successful.

Pigment production in embryogenic calli was scored after growth for two weeks in near darkness, or after a two hour exposure to full sunlight followed by two weeks' growth under low wattage fluorescent bulbs (Table 2). Because of ongoing genetic segregation, sampling error, and relatively mild light treatments, the possibility of false negatives is quite likely, and must be considered when interpreting all observations. When it was seen, pigment was usually in the surface cells of the undifferentiated, friable mass forming the bulk of embryogenic callus. The embryoids themselves, however, were colorless, even when arising from red callus. In only one genotype (JD40), containing the *B-peru* allele, pigment was seen in the suspensor-like part of embryoids. However, calli of this genotype were unlike normal type II calli, and grew poorly.

Pigment accumulated in both dark- and light-grown calli of the genotypes *R-r C1 B Pl* (JD26, JD30) and *R-r C1 b Pl* (JD28), while light-dependent pigment accumulation occurred in both *R-r C1 B pl* (JD27) and *R-r C1 b pl* (JD29). Thus, on the basis of positive scoring of pigmented calli, we can definitely state that *B* is not required for pigment synthesis, while *Pl* is probably required to eliminate a light-requirement. Our observations match Racchi's (Plant Cell Rep. 4:184) observations in non-embryogenic

Table 2. Scoring of embryogenic callus lines initiated in 1991: genotype, background, and pigmentation phenotypes.

Cross	Genotype	%A188/ Other bkgd.	Color: dark	Color: light
Regulatory genotypes				
JD1(x)	*r-r c1 b pl*	1.00	cl	cl
JD19 x 1	*R-g/r-r C1/c1 b pl*	.50/WKN1	cl	cl
JD26.1-1(x)	*[R-r/r-r CV? Pl/? B/?]* (x)	.875/W23	Red	Red
JD26.2-2(x)	*R-r/R-r [C1/? B/? Pl/?]* (x)	.875/W23	Red	Red
JD27.2-1(x)	*pl [R-r/r-r C1/? B/?]* (x)	.875/W23	cl	Red
JD28.1-2(x)	*R-r/R-r b [C1/? Pl/?]* (x)	.875/W23	Red	Red
JD29.1-1(x)	*R-r/R-r b pl [C1/?]* (x)	.875/W23	cl	Red
JD29.1-3(x)	*b pl [R-r/r-r C1/c1]* (x)	.875/W23	cl	cl
JD30.1-1(x)	*R-r/R-r [C1/c1 Bwk/? Pl/?]* (x)	.875/K55	Red	Red
JD30.1-4(x)	*pl [R-r/r-r C1/c1 Bwk/?]* (x)	.875/K55	cl	Red
JD31.1-2(x)	*R-r/R-r b [C1/c1 Pl/?]* (x)	.875/K55	cl	cl
JD32.1-5(x)	*r-r pl [C1/c1 B/?]* (x)	.875/W23	cl	cl
JD34.2-1(x)	*r-g b pl [C1/c1]* (x)	.75/W23	cl	cl
JD35.1-1(x)	*r-g C1/C1 b pl*	.75/W23	cl	cl
JD37.1-2(x)	*R-r/R-r c1 [B/? Pl/?]* (x)	.75/W23	cl	\ \
JD40.1-3(x)	*r-? [C1/? BPeru/? Pl/?]* (x)	.75/?	cl (Red suspensors)	cl (Red suspensors)
JD40.1-6(x)	*r-? C1/C1 Bperu/Bperu pl*	75%/?	cl	\ \
JD41.1-1(x)	*b pl [R-r/? C1-S/c1]* (x)	.875/?	cl	cl
Structural genotypes				
JD42.1-3(x)	*a1 R-g/R-g b pl [C1/c1]* (x)	.875/KN1	cl	cl
JD43.1-5(x)	*a2 R-g/R-g C1/C1 b pl*	.75/?	cl	cl
JD46.2-4(x)	*bz1 R-g/R-g C1/C1 B/B [Pl/?]* (x)	.75/W23	cl (Bronze)	cl

endosperm calli of K55/W23 hybrid background. All of his material, of genotype *R-r C1*, and differing only in its *B pl* genotype, eventually made anthocyanin, but only *Pl* genotypes made it rapidly (e.g., within 30 days after culture). Thus neither *B* nor *Pl* were required in endosperm callus, but *Pl* was an important enhancer of pigmentation.

The *R-r* and *C1* requirements are more ambiguous, as we must contend with negative evidence. *C1* is probably required for callus pigmentation, as *c1 R-r B Pl* (JD37) calli were colorless. This conclusion is uncertain, however, because only two calli were scored, and these grew poorly. As for *R*, in an earlier experiment it appeared that the seed-component (*R(S)*) is not required for callus pigment synthesis, because *r-r B C1 pl* calli left for several days on a lab bench in diffuse sunlight did indeed accumulate pigment (data not shown). Under the light conditions in the current experiment, however, *r-r B C1 pl* calli (JD32) failed to make pigment in the dark or in the light. Meanwhile, the plant component of *R* (*R(P)*, or *-r*) may be required, but is not sufficient for pigmentation. *R(P)* is the only permissive regulatory allele that inbred A188 (*r-r b c1 pl*) has, and A188 calli are always colorless. Experiments are in progress to better establish the *R-r* and *C1* requirements, which will give us additional insight into the relatedness of embryogenic callus to other plant tissues.

In addition to genotypes with varied regulatory genotypes, we have successfully initiated callus lines homozygous recessive for the structural genes *a1*, *a2*, and *bz1*. All of these calli were colorless, although the exact regulatory genotypes of these lines are uncertain. Experiments are in progress to verify that these calli have permissive regulatory genotypes. If so, we will attempt to reintroduce the missing genes by particle bombardment, and visually select for pigmented, i.e. transformed, tissue.

Mu9, a candidate for the regulator of Mutator activity, encodes two head-to-head transcripts

--R. Jane Hershberger, Christine A. Warren and Virginia Walbot

Mu9 is a 4942bp *Mu* element that is a candidate for the autonomous Mutator element (Hershberger et al., Proc. Natl. Acad. Sci. USA 88:10198-10202, 1991). Based on its restriction map and the sequence of its ~215bp terminal inverted repeats (TIRs), *Mu9* appears to be similar or identical to *Mu R1* (Chomet et al., Genetics 129:261-270, 1991) and *Mu A2* (Qin et al., Genetics 129:845-854, 1991), other candidates for the autonomous element whose sequences have not yet been reported. *Mu9* encodes two transcripts of ~2.8kb (gene A) and 1kb (gene B), diagrammed in Figure 1.

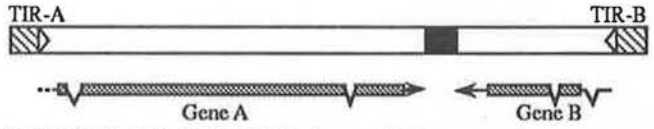

Figure 1. Structure of the *Mu9* transcripts and open reading frames

We have isolated cDNA clones covering 2.6kb of gene A; we are missing the 5' end of the gene and the initiation codon. The truncated cDNAs encode an 813 amino acid open reading frame, and in the genomic *Mu9* sequence there is an ATG in frame 10 amino acids upstream of the beginning of the cDNA clone. Gene A has at least two introns, one of 145bp near the 5' end, the other of 79bp near the 3' end. Gene B also has two short introns, of 117 and 72bp, both near its 5' end. The cDNAs we have isolated for this transcript encode a 207 amino acid polypeptide, beginning with an ATG at base 4476 (numbering according to Hershberger et al., 1991). There is an untranslated leader of 141bp that includes all of the first exon; this leader does not contain any ATG codons.

The gene B transcript begins just at the inside edge of the 215bp TIR-B, which implies that the gene B promoter is contained within the TIR. TIR-A and TIR-B are 96% similar overall, with only two differences in the first 180bp. On RNA blots, the message levels of gene A and gene B seem to be approximately equal; therefore, we postulate that TIR-A contains the promoter for gene A.

The premise that both *Mu9* TIRs contain promoters implies that transcription stop sites or strong polyadenylation signals should lie between the *Mu9* transcription units; otherwise, *Mu9* would make antisense as well as sense copies of its genes. The ~350bp region of *Mu9* that separates the two messages (black box in Figure 1) contains a series of four different direct repeats, ranging from 11 to 27bp, that are repeated two to five times each. The sequence between the termini of the cDNAs for gene A (3209) and gene B (3575) is given below, with the four types of repeats shown in different typefaces:

ACTGCTACGAACAAACACTATGAAACCTCCACC**TGTATTGGTTGTAAGACTGCT**AAG
AACAAGCCCAG **TGTATTTGTTGTAAGACTGCT** CAGTTTTAGTT
GCCAGTTCGTTGCTTCCCAGGTT CAGTTTTATTT CAGTTTTATTT CAGTTTTATTT
CAGTTTTATTC ATCAG *GCCCAGTTTCAG AGAAACAGATTGCTT*
GCCCAGTTCAGAGAAACAGATTGCTT GCAATTGTC *GCCCAGTTCAG*
AGAAACAGATTGCTT
*GCCCAGTTTCAGAGAAATA*GAGCGCAG*AAAACAGATAAAATATAACAC AAAACA-*
GATAACATATAACAC ACATGAATAACACTGAGCCATTAGTTCTTACAACCTCA

Although none of these motifs corresponds exactly to the polyadenylation consensus sequence, this region probably constitutes a series of either polyadenylation signals or transcription termination signals for the nascent RNAs. The structure of transcription termination regions is not well understood in plants, and studying this region may provide some new insight into this aspect of RNA processing.

The Bronze-2 protein has strong sequence similarity to a soybean stress protein, *Gmhsp26-A*

--Juliana Nash and Virginia Walbot

The product of the Bronze-2 (*Bz2*) gene is required last in the sequence of known anthocyanin biosynthetic structural genes (*C2 ->A1->A2->Bz1->Bz2->*), yet its function remains unknown. To look for clues that might lead us to the biochemical activity of *Bz2*, we performed nucleotide and amino acid sequence searches against the GenBank and EMBL DNA and protein sequence databases. We found that the primary amino acid sequence of the soybean *Gmhsp26-A* protein (Czarnecka et al., Mol. Cell. Biol. 8:1113-1122, 1988) is very similar to the *Bz2* protein sequence (Nash et al., Plant Cell 2:1039-1049, 1990); both encode a 26kDa gene product. The first 85 amino acids of exon 1 share a 44% identity and a 68% similarity, while there is no significant similarity between the second exons (see Figure 1). Transcripts pro

```
  2 TAGTMRVLGGEVSPFTARARLALDLRGVAYELLDEPLGPKKSDRLLAANP 51
    |.:.:::||  .|||..|..:||.|:||.|.:|:| || .||| ||  ||
  4 TQEDVKLLGIVGSPFVCRVQIALKLKGVEYKFLEENLG.NKSDLLLKYNP 52

 52 VYGKIPVLLLPDGRAICESAVIVQYIEDVARESGGAEAGSLLLPDDPYER 101
    |. |:|| ::.::.:|.|| |||:||::. ::..  ..:..   : :..|
 53 VHKKVPV.FVHNEQPIAESLVIVEYIDETWKNNPILPSDPY...QRALAR 98

102 AMHRFWTAFIDDKFWPALDAVSLAPTPGARAQAAEDTRAALSLLEEAFKD 151
      :| .. | : ....: .|.  . . . .:. |. .  . |.:  |.
 99 FWSKFIDDKIVGAVSKSVFTVDEKEREKNVEETYEALQFLENELKD..KK 146

152 RSNGRAFFSGGDAA.......PGLLDLALGCFLPALR...ACERLHGLSL 191
     .| .|  .: ||       | : ::|   ::.. :    :.: ::: |
147 FFGGEEFGLVDIAAVFIAFWIPIFQEIAGLQLFTSEKFPILYKWSQEF.L 195

192 IDASATPLLDGWSQRFAAHPAAKRVLPDTE 221
     .: . .:|..:.. ||  .|    |....
196 NHPFVHEVLPPRDPLFAYFKARYESLSASK 225
```

Figure 1. Amino acid sequence similarity between BZ2 and Gmhsp26-A proteins. Identical, very similar, and remotely similar residues are indicated by |, :, and ., respectively. Similarity calculations made in the text utilized only the very similar residues. BZ2 amino acid sequence is on the upper side of the alignment while *Gmhsp26-A* is on the lower: residue positions are noted in the margins. The intron position for *Bz2* follows residue #114 and for *Gmhsp26-A* follows residue #102. Sequence alignment was performed using GCG software (Genetics Computer Group, Madison, WI).

duced from *Bz2* and *Gmhsp26-A* are susceptible to splicing failure, and the resultant unspliced messages encode an approximately 14kDa protein in each case because of an in-frame stop codon within the intron sequence (Czarnecka et al., 1988; Nash et al., 1990). No gene sequences in the databases shared significant similarity to the *Bz2* nucleotide sequence

While the structural similarity of the first exons of these proteins is very high, the function of neither protein has been determined. The *Gmhsp26-A* protein was named a heat-shock protein because of a weak similarity to the set of small heat-shock proteins of animals, but the protein lacked any significant similarity to plant small heat-shock proteins (Czarnecka et al., 1988). Transcripts from *Gmhsp26-A* are induced as a result of many different stress conditions including, heat-shock, heavy-metal stress, oxidative stress, and ABA treatment (Czarnecka et al., Plant Mol. Biol. 3:45-58, 1984). Walbot et al. (this issue) show that *Bz2* expression may also be affected by ABA levels in kernel tissue. The expression patterns of *Bz2* have not been determined for

each of the above mentioned stress conditions yet, but we have shown that the *Bz2* gene is not a heat-shock protein gene: its transcript abundance decreases upon exposure of maize seedlings to 41 C treatments (J. Nash and V. Walbot, submitted). In fact, the heat-shock protein homology of *Gmhsp26-A* is within the second exon of this protein indicating that any related function of this soybean protein to heat-shock would be contained in a region of the protein with which *Bz2* does not share similarity. These findings, along with the observation of the similar intron-splicing behaviors of *Gmhsp26-A* and *Bz2*, are suggestive that these proteins may have been constructed by the process of exon-shuffling. Perhaps the first exon shares a function important to both of these proteins, such as metal-binding or a role in hormone-stress physiology, but the second exons are derived from different ancestral histories.

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Detection of an unfertilized polar nucleus with a fertilized egg cell
--Akio Kato

It has been known for about forty years that maize produces a few maternal haploids in its progenies (the incidence is about 0.1%) (Chase, Genetics 34:328-332, 1949) and that in the case of double fertilization, the maize embryo sac, egg cell and polar nucleus are fertilized by different sperms originating from different pollen grains. This phenomenon is called "heterofertilization" (Sarkar et al., J. Hered. 62:118-120, 1971; Robertson, J. Hered. 75:457-461, 1984). The most likely explanation for this phenomenon is that "A part of the maize pollen tubes has only one sperm cell or releases one sperm cell when the tubes enter the embryo sac." However this hypothesis had not been verified. In this paper I report data which strongly support the hypothesis. I detected an unfertilized polar nucleus with a fertilized egg cell using purple embryo marker and dual pollination.

Two normal maize lines, i.e. X18G (supplied by the Maize Genetics Cooperation Stock Center) and a sweet corn variety, Silver Honey Bantam, were used (Table 1). The X18G line carries a dominant pigment-conditioning gene *R-scm2*. The *R-scm2* gene, in conjunction with the dominant pigment-conditioning genes *A1, A2, C1, C2*, induces a deep pigmentation of the aleurone and scutellum. This color is visible in the dormant kernel. The seeds of Silver Honey Bantam are colorless due to the absence of one or more of the major dominant color genes.

Table 1. Description and genotypes of the stocks used.

Line	Genotype	Phenotype
X18G	*A1 A1 A2 A2 C1 C1 C2 C2 R-scm2 R-scm2 Y Y Sh2 Sh2*	colored scutellum, colored aleurone, yellow normal endosperm
Silver Honey Bantam	*y y sh2 sh2*	colorless scutellum, colorless aleurone, white shrunken endosperm

I put a small amount of X18G pollen on the silks of detasseled Silver Honey Bantam very carefully. Twenty four hours later, I deposited an appropriate amount of Silver Honey Bantam pollen on the same silks (dual pollination). Fourteen ears were pollinated and about 3500 seeds were obtained. Then I examined the characters of the aleurone, endosperm and scutellum (Table 2).

The seeds with colored aleurone and colored scutellum were

Table 2. Segregation of seed color after dual pollination.

Cross	colored aleurone with normal endosperm		colorless aleurone with shrunken white endosperm	
	colored scutellum	colorless scutellum	purple scutellum	colorless scutellum
Silver Honey Bantam X X18G X Silver Honey Bantam (24h later)	1602	4	5	1871

those that had been fertilized by the X18G pollen while the seeds with colorless aleurone and colorless scutellum were fertilized by the Silver Honey Bantam pollen (one or more seeds should be haploid because of the above-mentioned spontaneous haploid production). Of the four seeds with a colored aleurone and colorless scutellum, three were haploid (n=10) and one was diploid (2n=20) based on root tip chromosome counting. I planted the diploid plant and selfed it. The genotype was *Y/y, Sh2/sh2* and all the selfed seeds showed a colorless scutellum and aleurone, presumably due to contamination associated with heterofertilization between X18G and the pollen. The three haploid plants which were sterile and exhibited marked haploid features (narrow leaves, white stripes, dwarfism) were considered to be maternal haploids based on the color of the scutellum.

The presence of five seeds with colorless aleurone and colored scutellum is interesting. These findings suggest that the eggs were fertilized by the X18G sperm, while 24 hours later the polar nuclei were fertilized by the Silver Honey Bantam sperm and double fertilization occurred. Since the fertilization of maize occurs within 24 hours after pollination (Styles et al., Maydica 32:139-150, 1987), it is suggested that the pollen tube of Silver Honey Bantam which was pollinated 24 hours later could not compete with the X18G pollen tube which was pollinated before. It is unlikely that the pollen of X18G germinated slowly on the silks or the pollen tubes extended slowly to the silks. The possibility that the pollen tube of X18G reached the ovule at the same time as the pollen tube of Silver Honey Bantam and that they heterofertilized the polar nucleus and the egg cell is also remote. It is obvious that if two pollen tubes (that of X18G and Silver Honey Bantam) enter the ovule and release sperm cells at the same time, double heterofertilization may occur: namely the egg cell is fertilized by the X18G sperm cell and the polar nucleus is fertilized by the Silver Honey Bantam sperm cell. Conversely the egg cell may be fertilized by the Silver Honey Bantam sperm cell and the polar nucleus may be fertilized by the X18G sperm cell. Since the latter case was not observed in the current experiment, it is suggested that the presence of five seeds with colorless aleurone and colored scutellum indicates the occurrence of single fertilization, namely unfertilized polar nuclei with fertilized egg cells. It is reasonable to assume that unfertilized egg cells with fertilized polar nuclei may also occur, and that the eggs develop apomictically and produce haploid embryos. If this hypothesis is valid, it is suggested that the embryonic development of the unfertilized egg cell starts within 24 hours after the fertilization of the polar nucleus, as indicated by the presence of the three haploid plants in this experiment.

Heterofertilization may be involved in this phenomenon too. Some investigators state that simultaneous insertion of the pollen tube results in heterofertilization. However, simultaneous insertion is not always necessary for heterofertilization to occur. In this experiment heterofertilization occurred in spite of the wide interval of pollination.

The presence of this aberrant form of fertilization was first

reported in the maize line Stock 6 (Kato, MNL 64:109-110, 1990), which has been known to induce a high frequency of haploid stocks (the incidence of haploids is 3%) (Coe, Am. Nat. 93:381-382, 1959; Sarkar et al., Genetics 54:453-464, 1966). However, since the normal line used in this experiment exhibited the same pattern of aberrant fertilization, it is suggested that single fertilization may be widespread in maize.

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Duplicated RFLP loci are abundant in the genome

--Jean Dowty and Tim Helentjaris

Duplicated sequences in the genome are commonly revealed by RFLP detection techniques as first described by Helentjaris et al. in 1988 (Genetics 118:353-363). At that time, using over 200 single copy RFLP genomic and cDNA clones as probes, they estimated that approximately 29% of markers detected a duplicate sequence at another locus in the genome. Section V of the 1991 Maize Genetics Cooperation News Letter currently lists approximately 1,400 markers, 35% of which we estimate are duplicated at least once somewhere else in the maize genome. All ten chromosomes have a number of duplicated sequences, but as noted by Helentjaris et al., the duplications are not necessarily scattered randomly over the entire chromosome, but more often appear to cluster (Figure 1).

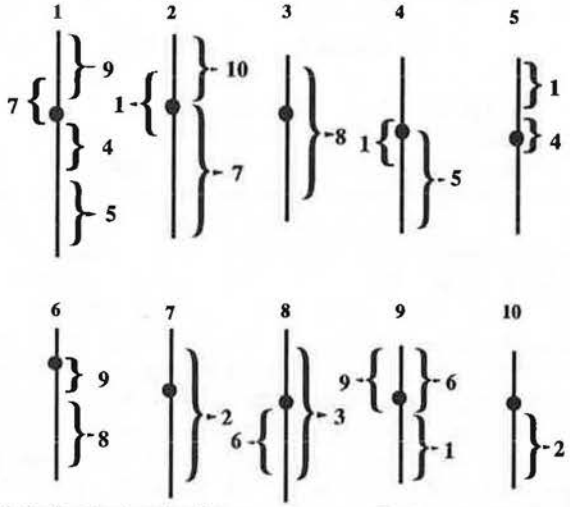

Figure 1. Duplicated regions shared between chromosomes. Brackets represent regions with a high concentration of markers which map to another chromosome (listed by number next to the bracket).

There is also a *higher order* pattern to the duplication-saturated regions of 3 different "sets" of chromosomes: 10-2-7 (Figure 2), 5-1-9, and 3-8-6. 70% of the markers on chromosome 2 are duplicated on either chromosome 10 or 7. The short arm of chromosome 2 contains all the duplications mapped to chromosome 10L, while the long arm of chromosome 2 shares all the duplications mapped to chromosome 7. Similarly, 46% of the duplications on chromosome 1 are shared with either chromosomes 5 or 9. All of the duplications shared by chromosomes 5 and 1 map to the short arm of chromosome 5 and the long arm of chromosome 1 respectively. All but two of the duplications shared by chromosomes 1 and 9 map to the short arm of chromosome 1 and the long arm of chromosome 9 respectively. 60% of the duplications on chromosome 8 are shared with chromosomes 3 and 6. While the duplications shared by chromosome 6 and 8 map to the long arm of chromosome 8, the duplications shared by chromosomes 3 and 8 map to both arms of chromosome 8. It is worth noting that while chromosome 2 shares duplications with both chromosomes 7 and 10, chromosomes 7 and 10 do not share any duplicated markers between them. Similarly, chromosomes 3 and 6 share only one marker between them, while chromosomes 5 and 9 share three markers between them.

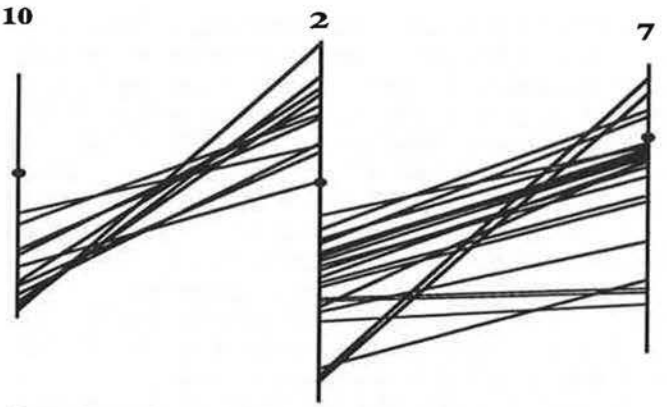

Figure 2. An example of the typical *higher order* pattern of duplicated markers within a chromosome "set" such as is seen in the chromosomes 10-2-7.

We believe this type of information will be useful to researchers who have isolated clones for specific gene interruptions originating within one of these duplicated regions. A careful perusal of the maize map in the corresponding region is probably warranted to determine if a homologous disruption is located there which might be due to a related sequence gene, as has been found for *B* and *R*, *orp1* and *orp2*, *D8* and *D9*, etc. We are also using some of the markers which are duplicated on the chromosome set 10-2-7 in maize to analyze evolutionary relationships within the *Andropogoneae* tribe by screening for duplications shared with some of the close relatives of maize such as teosinte, *Tripsacum*, and *Coix*, as well as with other genera in the *Gramineae* family.

Developing an assay for the contribution of GA-biosynthetic loci to quantitative variation in height

--Mary Ann F. Cushman and Tim Helentjaris

Results from an earlier study (Edwards et al., 1991) demonstrated that the genomic locations of several regions (QTL) controlling quantitative variation for height in F2 progenies from the cross, CO159 X Tx303, coincided with the locations of a number of dwarf mutant loci. One particular QTL was closely linked by analysis of the isozyme, *Acp1*, and the anonymous RFLP, *NPI222*, to the centromere of chromosome 9 and to a GA-biosynthetic locus, defined by the mutation, *d3*. This region accounted for 27% of the variance for plant height in this particular experiment and was also an important yield QTL. Similar results have since been observed in a number of other crosses.

The following test was devised as part of a series of studies designed to test Robertson's hypothesis (J. Theor. Biol., 1985) that moderate alleles at loci previously defined by more extreme mutant alleles may be responsible for quantitative variation; that is, they may function as QTL. This experiment has been repeated three times on inbred maize lines, twice in the greenhouse and once in the field; the latest greenhouse experiment is described herein. Given positive results from these initial experiments, our intent is

to apply this method to segregating populations to provide stronger supporting evidence for efforts to clone loci that may behave as important QTL.

The inbreds Mo17 and B73 were planted in the greenhouse in August 1991 in a randomized complete block design with four replications and two blocks per replication. In each block, four plants of each genotype were treated weekly with either 100ul of 1mg/ml GA_3 (0.1mg or $2.7X10^{-7}$ moles applied) in 40% ethanol or 100ul of 40% ethanol as a control. Height measurements for all plants were taken weekly, measuring from soil level to the tip of the longest leaf or to the top of the culm. At the end of the experiment, internode numbers were also recorded; however, this value was unaffected by GA treatment. Plant heights of the treated and untreated populations of each genotype differed significantly after a single treatment (Fig. 1, Week 2).

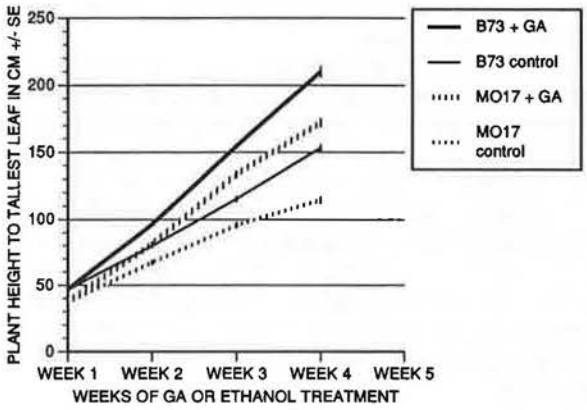

Figure 1.

To describe the response to GA treatment, an index of growth was calculated for each plant in both the control (I_C) and the treated (I_T) populations, where I = (final height - initial height)/initial height. These genotypes differed significantly for the growth index when treated with ethanol alone in the control (Mo17 I_C = 3.92±0.19 and B73 I_C = 5.51±0.14). After four weeks of treatment with GA_3, however, there was no significant difference in the growth index (Mo17 I_T = 10.20±0.43 and B73 I_T = 10.04±0.34), suggesting that these inbreds possessed the same potential for plant height once GA biosynthesis was no longer limiting. A simple measure of relative response to GA (R_{GA}) might be calculated as $R_{GA}=I_T-I_C$. These two genotypes would differ for this value (Mo17 R_{GA} = 6.20 and B73 R_{GA} = 4.53). It is possible that the difference in R_{GA} for plant height in these two inbreds might be ascribed to simple differences in GA levels, presumably due to different alleles at GA biosynthetic or degradative loci. Height in progenies from this cross can be shown to vary quantitatively, so it will be important to show whether variance in a segregating population can be precisely explained by any of the GA biosynthetic loci. If similar results (I_T values that are not significantly different, but R_{GA} that are significantly different) are obtained with other inbred lines and segregating progenies, this may provide a system in which the limitation of GA biosynthesis can be evaluated as a QTL controlling plant height in various genetic backgrounds.

These results demonstrate that these lines differ significantly in height both with and without treatments with GA_3, and for an index of growth in the absence of exogenously added GA, but not for the index of growth following GA treatment when GA is non-limiting. We conclude that it may be possible to partition the variance for plant height with and without exogenously added GA and demonstrate the presence of loci which quantitatively control endogenous GA levels and hence, plant height.

One way to address this question more precisely may be to use inbred lines into which different alleles of a region which may contribute to quantitative variation have been introgressed. These lines could be compared to their corresponding recipient lines for height variables with and without exogenously added GA. In this case, the importance of specific loci can now be assessed more precisely, because the confounding effects of other regions have been removed. In a sense, one has converted quantitative variation into qualitative variation. By introgressing a single region presumed to contain the *d3* locus into a number of genetic backgrounds, one should be able to compare the action of this new allele very precisely to the original allele present in these backgrounds. These lines should be equivalent in their ability to respond to GA, but may differ in endogenous GA levels, presumably due to the action of different GA alleles. If the paired lines (recipient and converted lines) differ in plant height without treatment but do not differ significantly after GA treatment, this would be strongly suggestive that the *d3* locus was functioning as a major QTL for plant height in these inbreds and would confirm a major component of Robertson's hypothesis. It would also demonstrate that this could be a productive strategy to confirm and clone genes that act as major QTLs for a number of important agronomic traits.

UPTON, NEW YORK
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RFLP mapping of mutants

--Eileen C. Matz, Frances A. Burr and Benjamin Burr

We are interested in correlating the RFLP map based on recombinant inbred families T232xCM37 and Tx303xCO159, with the traditional linkage map in regions of the genome where the RFLP map is not marked with common loci. We also have attempted to map two loci that had been mapped only to chromosome arms. In all cases this was done by analyzing the distribution of RFLP alleles in the homozygous mutant class from an F2 population derived from crosses between a full color stock (*B, Pl, R-r*) and the morphological mutant.

Mutant	Map position	Linked RFLP locus	Homozygous recessive class	Heterozygous class	Homozygous dominant class	% recomb.
j2	1L	*BNL7.25*	8/9	1/9	0/9	6
		BNL8.29A	9/9	0/9	0/9	0
		BNL6.32	7/9	2/9	0/9	11
oy1	10S15	*PIO200075A*	20/31	9/31	2/31	21
		PIO060005	28/31	3/31	0/31	5
py2	1L	*BNL6.32*[1]	4/6	2/6	0/6	17
sr2	10L98	*UMC44A*	16/18	2/18	0/18	6
		BNL10.13	17/20	2/20	1/20	10
		R1	17/20	3/20	0/20	8
		BNL17.02	17/20	3/20	0/20	8
		GLN1	10/18	7/18	1/18	25
gl15	9L074	*BNL5.04*	5/5	0/5	0/5	0
		BNL7.13	5/5	0/5	0/5	0

[1]Most distal probe on 1L; *py2* was unlinked with *BNL8.29A*.

Integrated RFLP map

--Benjamin Burr

The probing and scoring data obtained and compiled at

Brookhaven for the RI families from T232xCM37 and Tx303xCO159 have been confirmed, with help from Tim Helentjaris, with NPI data. The integrated data set has been analyzed together and has now been arrayed in a single map diagram.

[ed. note: Reproductions of the map, approx. 25x35 inches folded to 12.5x11.4, may be obtained from Coe, Columbia, MO. The data and coordinates are being readied for the database prototype.]

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Chromosome knob numbers of somatic cells of five inbreds

--Y. Wan and J. M. Widholm

Maize chromosome knobs consist of constitutive heterochromatin which remains condensed throughout the mitotic cell cycle. The knob compositions can be determined by pachytene analysis. However, the position of heterochromatin can be detected by mitotic chromosome C-banding techniques (Hadlaczky and Kalman, Heredity 35:371-374; Rayburn et al., Amer. J. Bot. 72:1610-1617). The pachytene knobs, for the most part, are the same heterochromatin detected by C-banding.

To rapidly evaluate the effectiveness of some antimicrotubule agents in inducing chromosome doubling of anther-derived callus (Wan et al., Theor. Appl. Genet. 81:205-211), we tried to determine the ploidy level of the callus cells by counting the knob numbers of interphase or nondividing cells. There could be at least two advantages to this approach: 1) the knobs should be visible in these cells; and 2) interphase and nondividing cells are more abundant than the cells at other stages. Although we later used flow cytometric analysis to determine the effectiveness of the chromosome doubling treatments, we did determine the knob numbers of root tip cells of five inbreds (B73, FR16, H99, Mo17 and Pa91) when we tested the approach. We felt that the approach may be useful for determining the ploidy level in some cases.

Root tips were collected from germinating seeds and were treated in cold water (4 C) for 24h and were fixed in a solution of 3:1 ethanol:glacial acetic acid for another 24h. After being stained with 1% acetocarmine and squashed on a glass slide, root tip meristem cells were examined under a light microscope. The visible heterochromatin regions in well spread interphase or nondividing cells were counted.

As shown in Figure 1, dark stained heterochromatic regions could be clearly seen in interphase root tip cells. Mo17 had 4 such regions and FR16 had 8, whereas H99, Pa91 and B73 all had 12. Since in mitotic interphase or nondividing cells it is impossible to distinguish the heterochromatic knobs from the heterochromatic nucleolus organizing region (NOR) of chromosome 6, the heterochromatic regions we observed include the 2 NORs. Thus Mo17 should have 2 knobs in the diploid somatic cells, FR16 has 6, and H99, Pa91 and B73 should have 10. Previously reported knob numbers in some inbreds were determined by pachytene analysis (Chughtai and Steffensen, Maydica 32:171-187), where the NORs could be recognized and be excluded. Also in pachytene cells the homologous chromosomes are paired and two homologous knobs appear as one. The reported knob numbers of Mo17 and B73 were 1 and 4, respectively (Chughtai and Steffensen, Maydica 32:171-187), so the 2 knobs of Mo17 we observed is consistent with the reported 1 knob in the pachytene cell. However the 10 knobs of

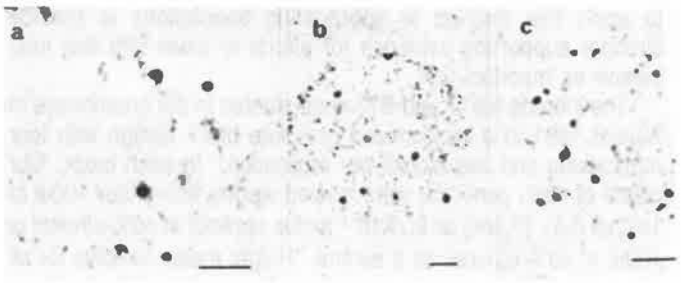


Figure 1. Heterochromatic regions including knobs and two NORs observed in the root tip meristem cells of maize inbreds. a) Mo17, 4 such regions; b) FR16, 8; c) Pa91, 12. Bar represents 5μm.

B73 we found would be 2 more than the expected number. The difference could be caused by counting 2 chromomeres as small knobs. The knob numbers of the other three inbred lines have not been presented previously.

Among these 5 inbred lines, the knobs of Mo17 were relatively larger, so were more apparent on the nuclear background (Fig. 1). The knobs of FR16 were also easily distinguished from the diffuse euchromatin (Fig. 1). We found that the knobs of B73, H99 and Pa91 are smaller compared with those of Mo17 and FR16.

The results indicate that maize heterochromatin knobs are visible in interphase or nondividing somatic cells using a simple method, especially for the strains with few or larger knobs such as Mo17 and FR16. The dark stained knobs and NORs are also visible in well spread maize callus cells using the same method, so the ploidy level of the callus cells could be determined by counting the knob number.

Globulin-1 gene expression in suspension cell cultures is dependent on exogenously supplied ABA

--Si Qing Liu, David R. Duncan and Alan L. Kriz

The most abundant protein in maize embryos is a vicilin-like storage protein encoded by the Globulin-1 (*Glb1*) gene. Expression of *Glb1* in intact embryos is regulated by ABA, and absence of *Glb1* transcripts in embryos homozygous for *vp1* mutations indicates that a functional *Vp1* gene product is required for *Glb1* expression. These observations are consistent with the presence of consensus ABA-response sequence elements located from positions -161 to -67 (relative to the transcript start site) in the *Glb1* promoter (Genetics 129:863, 1991). To further characterize the *Glb1* promoter in a functional manner, we have initiated experiments designed to identify regions of the promoter which are necessary for *Glb1* expression in cultured maize cells. For these experiments, a modified version of the promoter in which a novel *Bam*HI site was introduced at position +17 to facilitate cloning was fused to GUS and a series of 5' promoter deletions were generated. Plasmid constructs were introduced into cells of the maize P3377 suspension culture by particle bombardment. Treatment of the cells with ABA (100uM) is necessary for *Glb1*-driven GUS expression, as no GUS activity is observed with any construct in the absence of ABA. Use of the promoter deletions in this assay indicates that sequences between -86 and -358 are necessary for *Glb1* expression: high amounts of ABA-dependent expression are observed with the -358 construct, and no expression is detectable with the -86 construct, even in the presence of ABA. Experiments are in progress to further delineate the sequences required for ABA-dependent expression of the *Glb1*

gene. In addition, this assay system provides a rapid, sensitive means for analyzing promoter constructs in cells treated in different ways, such as varied ABA content and/or osmotic stress. We are currently collecting data from such experiments.

Another interesting observation obtained from these experiments concerns the accumulation of natural *Glb1*-encoded polypeptides in the P3377 cells. Immunoblot analysis of proteins extracted from these cultured cells indicates that no *Glb1* proteins accumulate in the absence of ABA, as expected, and inclusion of ABA in the culture medium results in production of *Glb1* protein. The protein product that accumulates, however, is the processing intermediate proproGLB1, and neither the mature GLB1 protein or its immediate precursor proGLB1 are detectable by this assay. It is also interesting to note that, in developing embryos, proproGLB1 is found exclusively in the endoplasmic reticulum, while the only *Glb1* proteins found in the vacuolar fraction are proGLB1 and GLB1. These results suggest that the cellular components necessary for proper transport and processing of *Glb1*-encoded polypeptides are not present in the P3377 cultured cells. Analysis of *Glb1* protein synthesis in these cells may be useful for studying mechanisms of globulin processing and transport.

The *Glb1-V* allele encodes an arginine-rich, basic seed globulin

--Alan L. Kriz, Faith C. Belanger and Cheryl A. Green

The maize *Glb1* gene, which encodes a vicilin-like embryo storage protein, is highly polymorphic, and several naturally occurring alleles have been described. The most common alleles, referred to as size alleles, encode mature polypeptides ranging in molecular mass from about Mr 60,000 to Mr 70,000, as determined by SDS-PAGE. We recently reported the nucleotide sequences of the *Glb1-L* (*Large* protein) and *Glb1-S* (*Small* protein) alleles (Genetics 129:863, 1991) and determined that the size difference between the two proteins encoded by these alleles is due to a 12 amino acid duplication in the GLB1-L protein relative to the GLB1-S protein. To further investigate the nature of allelic variation of *Glb1*, we cloned and sequenced the *Glb1-V* allele, the protein product of which has been reported by Osterman (Biochem. Genet. 26:463, 1988) to differ from the *L* and *S* proteins in the manner of protein processing. The sequence of *Glb1-V* is very similar to that of the other *Glb1* alleles, except that deletion of a single residue in the last (fifth) exon results in a frameshift mutation which dramatically alters the characteristics of the protein, as determined by deduction of the amino acid sequence from the nucleotide sequence. The most notable difference between the GLB1-V protein and the other GLB1 proteins is an effective substitution of several glutamic acid residues in the carboxy-terminal third of the polypeptide with arginine residues, due to the change in reading frame. The net result of these acidic to basic amino acid substitutions is a drastic change in the isoelectric point of the protein from about 6.5 to 12.3. Some characteristics of the different allelic forms of mature *Glb1* proteins, as deduced from nucleotide sequence information, are as follows:

	GLB1-L	GLB1-S	GLB1-V
mol. wt.	56,300	55,100	53,500
pI	6.2	6.8	12.3
# arg residues	61	59	83
# basic aa, total	100	98	115
# acidic aa, total	88	82	37

It is not apparent from the primary sequence of the GLB1-V protein how the processing of this polypeptide would differ from that of other GLB1 proteins. It is likely that the sequence changes introduced by the frameshift mutation alter the structure of the protein in such a manner as to result in altered processing. Analysis of GLB1-V protein synthesis may provide insight to the nature of globulin processing in maize embryos.

It is also interesting to note that the introduction of a large number of basic amino acid residues does not affect accumulation of the GLB1-V protein in maize embryos. One of the objectives of our laboratory is to use modified versions of the *Glb1* gene, engineered to contain a large number of lysine codons, as a means of enhancing the nutritional quality of maize grain protein. Mother Maize has shown us that the seed is capable of tolerating a *Glb1*-encoded protein with a large number of basic arginine residues, and it is likely that modified polypeptides, containing large numbers of basic lysine residues rather than arginines, will effectively be accumulated in the seed as well.

Identification of maize cDNA clones corresponding to genes encoding chitinase and β-1,3-glucanase

--Shenchuan Wu, Jack M. Widholm and Alan L. Kriz

The degradative enzymes chitinase and β-1,3-glucanase have been found to accumulate in higher plants following pathogen attack and environmental stress, which indicates their involvement in plant defense responses. These enzymes are particularly important for resistance of plants to fungal invasion, as chitin and β-1,3-glucans are major cell wall constituents of certain fungi. As an approach to identifying means for possible enhancement of fungal resistance in maize through the use of genetic engineering technology, we have isolated cDNA clones corresponding to maize genes encoding chitinases and β-1,3-glucanases. These full-length clones were obtained from a cDNA library generated from mRNA isolated from maize seedlings (inbred line Va26) that had been treated with mercuric chloride as an inducing agent. The library was screened with partial clones, corresponding to each gene, that had been generated by PCR amplification of cDNA obtained from the mercuric chloride-induced mRNA sample. Degenerate oligonucleotide primers for the PCRs were designed from published sequences of barley chitinase and β-1,3-glucanase genes. Nucleotide sequence analysis of these clones indicates that the maize and barley chitinase genes exhibit 73% amino acid identity, and that the β-1,3-glucanase sequences from the two species exhibit 63% identity at the amino acid level.

To investigate gene expression patterns, the chitinase and β-1,3-glucanase clones have been used as probes in northern blot analysis of maize RNAs from a variety of tissues and treatments. Various chemical treatments had previously been shown to result in induction of chitinase and β-1,3-glucanase expression (e.g., Plant Science 76:211, 1991). While no transcripts corresponding to either gene were present in control 7-day old seedlings, treatment with mercuric chloride resulted in rapid, high-level induction of expression of both genes. Similar responses in β-1,3-glucanase expression patterns were observed with ethephon and salicylic acid treatments, but these latter treatments had little effect on expression of chitinase genes. Neither gene was expressed in maize kernel tissues during normal seed development, but both wounding and infection with the fungal pathogen *Aspergillus flavus* resulted in accumulation of chitinase and β-1,3-glucanase transcripts. This induction was limited to aleurone tissues, with little or no expression detectable in the embryo or the starchy endosperm of kernels from wounded or inoculated ears.

These preliminary studies on expression patterns of genes involved in fungal defense mechanisms provide information with respect to the control of economically important fungal infections of maize kernels. *A. flavus* infection, for example, can result in production of the highly carcinogenic compound aflatoxin. Since aflatoxin-contaminated grain is of little value, effective control of *A. flavus* infection would have a significant impact on the grain industry. Such control may be realized through the use of genetic engineering approaches to enhance and redirect expression of chitinase and β-1,3-glucanase genes to seed tissues through the use of appropriate promoters.

Rearrangement of the "R" sequence is the only mitochondrial DNA alteration consistently associated with cms-S reversion to fertility

--Gracia Zabala, Susan Gabay-Laughnan and John R. Laughnan

The mitochondrial genome of S cytoplasmic male-sterile (cms-S) maize plants contains two linear episomes, S1 and S2. The inverted repeats (IRs) at the termini of these episomes recombine with identical IR sequences present in the main mitochondrial genome at the junction of sites designated σ - σ' and ψ - ψ' (Schardl et al., Nature 310:292-296, 1984). These recombination events result in the linearization of a high proportion of the mitochondrial genome. The resulting linear molecules have an episome at one end flanked by a σ or ψ sequence. At the other end is a 2kb sequence (R) common to σ' and ψ', including 195/208bp homology to the IRs of S1 and S2, flanked by the remainder of a σ' or ψ' sequence. The R region shows strong DNA sequence homology with the unique end of the R1 episome of the RU cytoplasm.

Southern blots of mitochondrial DNA (mtDNA) digested with restriction endonucleases and hybridized to the probe for the R sequence can give a complex pattern due to the different arrangements in which R is found, i.e., σ-R-σ', ψ-R-σ', σ-R-ψ', ψ-R-ψ', *R-σ' and *R-ψ' (where * indicates a linear end). Cytoplasmic revertants to fertility in M825, 38-11, H95 and W182BN nuclear backgrounds lose both of the S episomes and all linearized mtDNA molecules. At first, no significance was attached to the absence of *R-σ' and *R-ψ' since there is no linearization in these revertants. Revertants in the WF9 nuclear background lose neither the free S episomes nor the linearized high molecular weight mtDNA molecules. However, the R sequences at the linear chromosome ends are rearranged as are the integrated R sequences. All R sequences are rearranged in WF9 revertants. In some cases the R sequence seems to have been eliminated from the mitochondrial genome completely. Other WF9 revertants retain the R sequence in abundance but in one or more novel arrangements. The rearrangement of the R sequence at the linear chromosome ends is the only consistent change in all cms-S cytoplasmic revertants analyzed, regardless of the nuclear background in which the reversion occurred. It is not the R sequence per se that is associated with cms-S since the mitochondrial genomes of M825, 38-11, H95 and W182Bn revertant contain intact integrated R sequences. The male sterility seems to be correlated with the R sequence located at the ends of the linearized mitochondrial DNA molecules.

Expression of the R1-homologous region in mtDNA of cms-S sterile, restored and cytoplasmically revertant plants

--Gracia Zabala, Susan Gabay-Laughnan and John R. Laughnan

The molecular lesion responsible for the S-type of cytoplasmic male sterility (cms-S) in maize appears to involve sequences derived from mitochondrial episomes. All cms-S cytoplasmic revertants analyzed show mitochondrial DNA (mtDNA) rearrangements involving S1-, S2- and R1-homologous sequences. The type of rearrangement depends on the nuclear background. However, all cytoplasmic revertants analyzed to date contain rearrangements at the termini of the mitochondrial genome linearized by recombination between S episome terminal inverted repeats (IRs) and IR-homologous sequences present in the main mitochondrial genome. The termini of these linear molecules that rearrange contain a sequence, R, homologous to the unique end of the R1 episome present in RU cytoplasm. Plants with RU cytoplasm are fully fertile, however.

We have analyzed the expression of this R-homologous sequence in mitochondria of cytoplasmic revertants that arose in different nuclear backgrounds and their respective sterile progenitors. The complex transcriptional profile of the R region changes upon reversion in the seven revertants analyzed, representing four nuclear backgrounds. Although the changes in transcriptional profile vary among the different nuclear backgrounds, all seven revertants lack a 1.39kb transcript present in all cms-S mitochondria.

The nuclear restorer gene *Rf3* restores fertility to cms-S plants. Our laboratory has identified newly arisen *Rf* genes, not allelic to *Rf3*, that also restore fertility to cms-S plants. We have examined the expression of the R sequence in cms-S lines with and without a nuclear *Rf* gene in two different nuclear backgrounds, WF9 and B37. The *Rf3* gene affects expression from the R region while two of the new restorers do not. The *Rf3* gene reduces the steady-state abundance of the transcripts and alters the transcriptional profile of the R region. The 1.39kb transcript is among those transcripts of which the steady-state abundance is reduced. This effect is more pronounced in *Rf3 Rf3* homozygotes than in *Rf3 rf3* heterozygotes although transcription is not eliminated entirely. Even though the newly arisen restorer genes do not affect the R transcriptional profile, we can not rule out the possibility that they may regulate the expression of these sequences at the post-transcriptional level.

We have analyzed mitochondrial RNA from strains containing RU cytoplasm and have determined that the R1 episome, that has homology to the R sequence of cms-S, is not transcribed in the RU cytoplasm in a manner similar to the R sequence in cms-S.

Normal (N) cytoplasm mitochondria carry the R sequence (R1 homology) integrated next to one copy of the 5.27kb repeat. Houchins et al. (EMBO J. 5:27-2788, 1986) have reported a 2.85kb transcript hybridizing to a 4.13kb clone containing the R sequence in normal cytoplasm. This transcript is thought to arise within R with transcription proceeding towards and into the 5.27kb repeat. We do not detect this or any other transcript with our R probe. It is possible that transcription in cms-S takes place in the region of the R sequence not adjacent to the 5.27kb repeat in N. These results suggest that the transcriptional pattern we have observed for the R region in cms-S mitochondria is specific to the particular arrangement of the R sequence in the cms-S genome and not to the R sequence per se.

We now have three compelling results supporting the involvement of mtDNA regions harboring the R sequence in the cms-S defect. These regions undergo rearrangements in all cytoplasmic revertants analyzed, regardless of nuclear background. The transcriptional profile of the R region changes upon reversion in all re-

vertants analyzed. The *Rf3* nuclear restorer gene reduces and alters the transcriptional activity of the R region in both nuclear backgrounds studied.

We will extend our analysis of gene expression of the R region into the developmental stage when pollen dysfunction first becomes evident. Such a developmental focus is essential since cms-S restoration is gametophytic, not sporophytic as in cms-T and cms-C.

Transposition characteristics of the naturally occurring cms-S restorer in inbred line CE1

--John R. Laughnan, Susan Gabay-Laughnan and Janet M. Day

In MNL 63:121 we presented evidence for transposition of the cms-S restorer carried by inbred line CE1, here designated *Rf(CE1)*. This restorer is located in the long arm of chromosome 2 where it ordinarily gives from five to ten percent recombination with the *wx* gene in *wx* T2-9d translocation heterozygotes. The testcross: (S) *rf rf wx wx* X (S) *Rf(CE1)* N *Wx/rf* T2-9d *wx* is used to search for instances of putative transposition of *Rf(CE1)*; these are identified as male parents whose testcross ears show significantly enhanced frequencies of *wx* kernels. Since the heterozygous male parents in these testcrosses carry S-type cytoplasm, *rf* (nonrestoring) pollen grains are aborted, so only *Rf(CE1)* pollen grains function. Hence, on testcrossed ears which exhibit both *Wx* and *wx* kernels, the *Rf(CE1)-wx* recombination rate is given directly by the percentage of *wx* kernels. Pollen examination with a field microscope confirms that male parents to be testcrossed are heterozygous for both the translocation and the *Rf(CE1)* restorer.

Occasionally (S) *Rf(CE1)* N *Wx/rf* T2-9d *wx* male parents with significantly higher *Rf-wx* recombination rates are identified by this procedure. In MNL 63:121 we reported on five such putative cases of *Rf(CE1)* transposition among 33 tested plants. In another experiment involving *Rf(CE1)*, and the same procedure, there were no putative cases of transposition among 52 tested plants. In the second cycle of this experiment, however, where the 45 *Rf(CE1)* marked heterozygotes tested were offspring of 16 stable female heterozygotes of the first cycle, there were 11 cases of putative *Rf(CE1)* transposition. This pattern of low or no activity in the first generation *Rf(CE1)* heterozygotes, with enhanced *Rf-wx* recombination rates in later cycles, appears to be the pattern with other *Rf* marked heterozygotes that have been tested, including spontaneous *Rfs I, III, IV,* and *VI*. The *Rf(CE1)* experiment has been continued through the fourth cycle. New instances of change from control-rate to high-rate *Rf-wx* recombination are encountered with advancing cycles. Interestingly, high-rate parents may produce both high-rate and control-rate progeny, the latter of which, we believe, are the result of loss, by meiotic recombination, of the most distant (transposed) *Rf* element.

We have considered the possibility that the cases of testcross ears with high frequencies of *wx* kernels may not result from *Rf(CE1)* transposition, but may instead be due to occasional instances of change of S- to "N"-type cytoplasm in smaller or larger sectors of the tassels of *Rf(CE1)* heterozygous male parents. Lacking S cytoplasm, all pollen from such sectors should be functional, including the nonrecombinant *rf wx* type, and these would register as increased numbers of *wx* kernels on testcross ears. The occurrence of such sectors of a size that would explain high frequencies of *wx* kernels on these ears is not likely, since a pollen check of each male testcross parent is made to confirm that it is heterozygous for both *Rf(CE1)* and the translocation, before it is involved in crosses. Moreover, there is independent evidence indicating that the S to "N" cytoplasmic change is low in these strains. A direct test of whether this spurious transmission of *rf wx* through the male parent can account for high *wx* frequencies on testcross ears is based on the recognition that, if only *Rf(CE1)* pollen grains function, all offspring are expected to be male-fertile; if *rf wx* grains are functional because they derived from an S to "N" mutation in the tassel, they are expected to produce male-sterile offspring, since the testcross female parent is (S) *rf rf*. Table 1 summarizes data on the characteristics of *wx* progeny from (S) *rf rf wx wx* X (S) *Rf(CE1)* N *Wx/rf* T2-9d *wx* testcrosses. The tabular information is arranged according to classes of increasing frequencies of *wx* kernels on testcross ears, a random sample of whose *wx* kernels were planted to score. While it is evident that male-sterile offspring do occur, their overall frequency, 4.4% of 963 plants scored, is far too low to account for the numbers of *wx* kernels observed in column 1 classes, and there is no evidence for a correlation of frequency of male-sterile offspring with increased frequency of *wx* kernels on the ears. The 13 male-sterile plants in class 2 were distributed among 9 of the 23 progenies, and the 18 male-sterile plants in class 4 were distributed among 6 of the 7 progenies, 10 of the 18 plants occurring in a single progeny. In a study similar to this one, involving the restorers of spontaneous origin *Rfs I, III, IV,* and *VI*, among 1,924 testcross progeny scored, there were 19 (1.0%) male-sterile progeny, plus 31 (1.6%) plants with exserted anthers that could not be scored. Even if these are considered to be male-sterile, the 2.6% of male-sterile offspring can account for only a small fraction of *wx* kernels on testcross ears.

The data in Table 1 provide additional information on *Rf(CE1)* transposition. The 921 male-fertile plants were scored for presence or absence of the translocation, and these data are presented for the six classes based on the frequency of *wx* kernels on testcross ears. Since all these offspring are fertile and derive from *wx* kernels, they must represent *Rf-wx* recombinants. If the *Rf-wx* exchange occurs between T and *wx*, the recombinant strand is *Rf(CE1)* N *wx* and the resultant offspring will lack the translocation; if it occurs between *Rf* and T, the recombinant strand is

Table 1. Fertility characteristics of recombinant waxy (*wx*) progeny from (S) *rf rf wx wx* X (S) *Rf(CE1)* N *Wx/rf* T2-9d *wx* testcrosses.

wx kernels on testcross ears		No. of testcross progenies	Total no. of fertile plants	No. of plants with translocation:				Male-sterile plants	
Class	%	(*wx wx*)		Present	(%)	Absent	(%)	No.	(%)
1	0-10	14	115	60	(52)	55	(48)	1	(0.9)
2	10.1-20	23	268	152	(57)	116	(43)	13	(4.6)
3	20.1-30	3	65	49	(75)	16	(25)	0	(0.0)
4	30.1-40	7	174	155	(89)	19	(11)	18	(9.4)
5	40.1-50	9	237	218	(92)	19	(8)	8	(3.3)
6	50.1-60	2	62	54	(87)	8	(13)	2	(3.1)
TOTALS		58	921	688		233		42	(4.4)

Rf(CE1) T *wx*, and the resultant offspring carry the translocation. With *Rf(CE1)* located at its control site, about 5 to 10 map units from *wx* in the heterozygous translocation complex, the data indicate near equality (60:55) for plants with and those without the translocation. In classes with increasing frequencies of *Rf-wx* recombinant kernels on the ear, there is a progressive shift in this ratio in favor of plants with the translocation vs. plant without the translocation, as in classes 5 and 6, where *Rf-wx* recombination frequencies are at their highest, and corresponding percentages of plants with the translocation are 92% and 87%, respectively, with the difference between them not statistically significant by contingency chi-square analysis. This shift in ratio is consistent with the presence of a transposed *Rf* at a recipient site some distance from the original, or donor, site. As this distance increases the ratio of *Rf* T *wx* to *Rf* N *wx* progeny is expected to increase, and to reach its maximum when the transposed *Rf* assorts independently of *wx*, either because it is far distant from *wx* in the 2-9 heterozygous translocation complex, or is in another chromosome. The data in Table 1 support the presence of transposed cms-S *Rf(CE1)* elements; they do not provide information on whether *Rf(CE1)* is present or absent at the original site in heterozygotes with a transposed *Rf*. An accompanying article deals with this question.

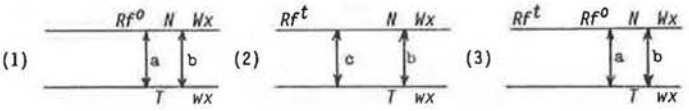
Evidence for replicative transposition of cms-S restorers

--John R. Laughnan, Susan Gabay-Laughnan and Janet M. Day

Evidence supporting the transposability of the naturally occurring restorer, *Rf(CE1)*, in inbred line CE1, was reported in MNL 63:121 and has been extended in a companion article on these pages titled "Transposition characteristics of the naturally occurring cms-S restorer in inbred line CE1".

We present here preliminary evidence that plants that carry a transposed *Rf* most often retain *Rf* at or near the original (donor) site, a situation often referred to as replicative transposition. The system used to identify instances of *Rf* transposition involves the testcross: (S) *rf rf wx wx* X (S) *Rf* N *Wx/rf* T *wx*. The female parent on the left carries S-type cytoplasm, is phenotypically male-sterile (*rf rf*) and is homozygous for the mutant waxy-1 (*wx*) allele on chromosome 9. The male parent also carries S-type cytoplasm, is heterozygous for the restorer (*Rf*) to be tested for transposition capability, is heterozygous at the waxy locus (*Wx wx*), and for a translocation (T/N) chosen to effect a suitably close linkage between the particular *Rf*-gene, at what we will call the original, or control, site, and the *wx* locus. In these studies, map distances between *Rf* and *wx* in the translocation heterozygotes are around 12 map units or less. Since the restorers of cms-S, unlike those of cms-C and cms-T, act gametophytically, *rf* (nonrestoring) pollen grains of this male parent abort, so only *Rf* pollen grains function. Therefore, *wx* is transmitted through pollen only if there is a crossover between *Rf* and *Wx* to produce a functional *Rf-wx* microspore. On testcross ears that exhibit both *Wx* (starchy) and *wx* kernels, the *Rf-wx* recombination rate is given simply by the percentage of *wx* kernels, without the need to score progeny plants for the presence (male-fertile) or absence (male-sterile) of *Rf*. Instances of putative transposition are identified as male parents whose testcross ears show significantly enhanced frequencies of *wx* kernels. Many such cases of plants with enhanced *Rf-wx* recombination have been encountered in the natural restorers *Rf(CE1)* and *Rf(Tr)*, and in the spontaneously occurring *Rfs*, *I*, *III*, *IV* and *VI*. Such higher *Rf-wx* recombination rates range from slightly above control rates, through intermediate levels, to rates exceeding 50%.

The evidence in support of replicative transposition of *Rf* genes is based on the frequency of male-fertile vs. male-sterile offspring from the cross (see Table 1 legend): (S) *Rf* N *Wx/rf* T *wx* X (N) *rf* T *wx/rf* T *wx* that is used to propagate the *Rf* heterozygotes whose testcrosses have been described above. When *Wx* kernels from this cross are planted, most of the progeny plants will be: (S) *Rf* N *Wx/rf* T *wx* heterozygotes with the same genotype as the female parent. If, in the female parent, a crossover occurs, either between *Rf* and T, or between T and *Wx*, the chosen *Wx* kernels, of either (S) *rf* N *Wx/rf* T *wx* or (S) *rf* T *Wx/rf* T *wx* genotype, will produce only male-sterile progeny. The three simple diagrams below illustrate heterozygous female parents of three possible types: (1) a single Rf^o at the original, or control site, (2) a single transposed Rf^t at a recipient site some distance from the Rf^o site, and (3) two *Rf* genes, one (Rf^o) at the original site, and the other (Rf^t) at the transposition site. The arrows indicate crossovers that can yield *Wx*-non-*Rf* male-sterile recombinant offspring.



In example (1) the cross by male parent (N) *rf* T *wx/rf* T *wx* is expected to produce from 5 to 10% *Wx*-non-*Rf* male-sterile progeny corresponding to the short map distance between Rf^o and *Wx* (crossovers a and b).

In example (2) either crossover b or c will produce *Wx*-non-*Rf* male-sterile progeny; since in this case the Rf^t-*Wx* distance is identifiable, from independent testcrosses, as significantly above the control level, and may range up to and above 50%, the frequencies of male-sterile (*Wx-non-Rf*) offspring are expected to be proportional to the Rf^t-*Wx* distance and should approach levels of 50% of offspring in some instances.

In example (3), where Rf^o and Rf^t are both present, only crossovers a and b will yield *Wx*-non-*Rf* male-sterile offspring, since a crossover between Rf^t and Rf^o results in a *Wx*-Rf^o male-fertile offspring. Thus, given a female parent that has been shown by independent testcrossing to carry a transposed Rf^t, the progeny of its cross with the (N) *rf* T *wx/rf* T *wx* male parent should exhibit a male-sterile frequency corresponding to the Rf^t-*Wx* distance if Rf^o is no longer present at the original site (nonreplicative). If, however, Rf^o is present at its original site (replicative), only control frequencies of male-sterile offspring are expected, no matter how distant Rf^t may be from Rf^o, and this holds even if Rf^t is located in a nonhomologous chromosome.

The data in Table 1 provide a test of the above expectations. In column 1 are given the genotypes of four different *Rf* heterozygotes in the study. For example, in the case of the *Rf(CE1)* N *Wx/rf* T *wx* heterozygote (row 1), there were 30 plants involved as female parents in the indicated cross. On the basis of the performance of each of these plants as male parents in testcrosses into (S) *rf rf wx wx* testers, 14 (column 3) gave relatively low *Rf-wx* recombination rates, from 3.8% to 14.8% (column 2). Columns 4-7 (row 1) deal with the distribution of male-fertile and

Table 1. Frequencies of male-sterile offspring from (S) *Rf* N *Wx/rf* T *wx** X (N) *rf* T *wx/rf* T *wx* crosses, arranged according to *Rf-wx* recombination rates in progeny of testcrosses of the female parent.

Genotype of restorer parent	From crosses whose female parents gave lower-level *Rf-wx* recombination rates in testcross						From crosses whose female parents gave higher level *Rf-wx* recombination rates in testcross					
	% *Rf-wx* recombination	No. of crosses	male-fertile progeny	male-sterile progeny	Total progeny	% male-sterile	% *Rf-wx* recombination	No. of crosses	male-fertile progeny	male-sterile progeny	Total progeny	% male-sterile
Rf(CE1) N *Wx/rf* T2-9d *wx*	3.8-14.8	14	99	8	107	7.5	16.0-45.4	16	92	10	102	9.8
Rfl N *Wx/rf* T8-9 (043-6) *wx*	3.0-11.0	13	117	6	123	4.9	24.5-33.0	7	107	3	110	2.7
Rflll N *Wx/rf* T2-9d *wx*	0.0-6.2	13	114	5	119	4.2	32.8-56.5	6	74	1	75	1.3
RfVI N *Wx/rf* T2-9d *wx*	--	--	--	--	--	--	39.4-69.5	3	43	3	46	6.5
	TOTALS	40	330	19	349	5.4		32	316	17	333	5.1

*Each of the 72 plants tested here was pollen checked to confirm that it was heterozygous for both the *Rf* gene and for the translocation. Each was testcrossed as a male parent onto (S) *rf rf wx wx* to determine the frequency of *Rf-wx* recombination, and was crossed as a female parent, as indicated here, to determine the frequency of male-sterile plants among the offspring.

male-sterile progeny from crosses of (S) *Rf(CE1)* N *Wx/rf* T *wx* plants as female parents with (N) *rf* T *wx/rf* T *wx* testers. Sixteen (column 9) of the 30 *Rf(CE1)* heterozygotes, when used as male parents in testcrosses, gave *Rf-wx* recombination from 16.0% to 45.4% (column 8, row 1); the distribution of male-fertile and male-sterile offspring from these 16 *Rf(CE1)* heterozygotes is given in columns 10-13, row 1. Because of their higher levels of *Rf-wx* recombination these 16 heterozygotes are regarded as *Rf* transpositions. Corresponding data for *Rfl* and *Rflll* heterozygotes are given in Table 1. In the case of *RfVI*, only higher-level recombination heterozygotes were available to score.

The data provide no evidence for higher levels of male-sterile plants among the progeny of *Rf* heterozygotes with high *Rf-wx* recombination rates. The frequencies of male-sterile plants among offspring of *Rf* heterozygotes in low and high recombination categories are remarkably similar in *Rf(CE1)*, *Rfl* and *Rflll* restorer strains and all are at control-rate levels. In the case of *RfVI*, for which data are available only for the high *Rf-wx* recombination category, the male-sterile rate (6.5%) is also at the control level. Overall, a total of 349 plants from *Rf* heterozygotes with low *Rf-wx* recombination rates, and 333 plants from *Rf* heterozygotes with high *Rf-wx* recombination rates were scored. The frequencies of male-sterile offspring in these two groups were 5.4% and 5.1%, respectively. The contingency chi-square for these data corresponds to a P value of 0.85.

It is evident that an overwhelming proportion of plants with enhanced *Rf-wx* recombination rates (transpositions) retain an *Rf* at or near the original Rf^0 site. This is not to say that, with the use of other crossing protocols, Rf^0 can not be removed through recombination; we are currently studying this possibility.

On the way to tagging the *Rf2* restorer of cms-T

--John R. Laughnan and Susan Gabay-Laughnan

There are two known nuclear restorers of cms-T, *Rf1* in chromosome 3L, and *Rf2* in chromosome 9S closely linked with the waxy-1 (*wx*) locus. *Rf1* and *Rf2* are complementary in their interaction, that is at least one dose of each is required for restoration of fertility to cms-T. Moreover, unlike the gametophytic mode of cms-S restoration, cms-T restoration is sporophytic in nature, meaning that it is the genotype of the pollen-producing plant rather than the genotype of the pollen grains themselves that governs pollen viability. Hence, a cms-T plant that is heterozygous for both restorers, *Rf1 rf1 Rf2 rf2*, produces all normal pollen even though only one-fourth of the pollen grains carry both restorers (*Rf1 Rf2*).

Like others, we are attempting to tag the *Rf2* gene because molecular studies on cms-T restoration have so far shed no light on its function. To this end we have searched for exceptional male-sterile offspring among the progeny of the cross: (T) R213: *Rf1 Rf1 Wx rf2/Wx rf2 Bz2 Bz2*; no *Ac* X (N) *rf1 rf1 Wx:Ac Rf2/wx Rf2 bz2-m/bz2-m*. The R213 *Rf1 Rf1 rf2 rf2* female parent is an inbred line derived from inbred lines WF9 and Ky21 by Jack Beckett and is male-sterile in cms-T plants. The male parent, kindly provided by Drew Schwartz, carries an insertion of *Ac* (Activator) in the starchy allele (*Wx:Ac*), and is homozygous for *bz2-m* (bronze-2 mutable), which derives from an insertion of *Ds* (Dissociation) into wildtype *Bz2*, in chromosome 1L. We have determined, by suitable testcrosses, that this male parent has the genotype *rf1 rf1 Rf2 Rf2*, which means that it carries the sought after cis arrangement of the two closely-linked genes *Wx:Ac* and *Rf2*. Since the male parent carries *Wx:Ac Rf2* in one chromosome 9, and *wx Rf2* in the other, two progeny types are expected with equal frequency. One has the genotype (T) *Rf1 rf1, Wx:Ac Rf2/Wx-rf2, Bz2/bz2-m*, and the other carries *wx* in place of *Wx:Ac*, but is otherwise the same.

Table 1 summarizes the results of our search for male-sterile plants among the offspring of this cross. Fourteen male-parent sources were involved in a total of 89 crosses that produced 8,876 offspring. Four of the pollen sources were tassels of main plants and 8 sources were tassels of tillers, here designated as, for example, "1833-9a" and "1833-9b". We were constrained to use tiller tassels because the main tassels of the male-parent strain were often "shed out" by the time the later-maturing (T) R213 plants were silking. Among the 8,876 offspring, 90 (1.0%) were male-sterile plants. Most of these exserted no anthers, but

Table 1. Male-sterile progeny from the cross: (T) R213: *Rf1 Rf1, Wx rf2/Wx rf2, Bz2 Bz2*; no *Ac* X (N) *rf1 rf1, Wx:Ac Rf2/wx Rf2, bz2-m/bz2-m.*

			Male-sterile plants	
Male parent	No. of crosses	Total plants	No.	%
930-1a	5	310	1	0.3
930-4a	4	332	6	1.8
930-5a	10	1452	4	0.3
931-1a	9	735	0	---
931-2	2	103	0	---
931-3	6	642	0	---
931-6a	8	675	13	1.9
1833-7	1	20	0	---
1833-7a	2	64	0	---
1833-8	2	46	0	---
1833-9	17	2219	66	3.0
1833-9a	11	998	0	---
1833-9b	9	1046	0	---
1835-4	3	234	0	---
TOTALS	89	8876	90	1.0

some exhibited islands of florets, whose anthers contained normal pollen, on tassels that were otherwise sterile.

The distribution of male-sterile offspring among the male-parent sources provides a clue to their origin. Only 5 of the 14 sources produced male-sterile offspring, but at least three of these, 930-4a, 931-6a and 1833-9, have far higher frequencies of male-sterile offspring than would be expected if these were distributed randomly among the total population. This is not likely due to genetic variability in the progeny since both male and female parents are essentially inbred lines whose offspring are expected to be genetically uniform. That the male-sterile plants are not randomly distributed is evident from the progeny of crosses involving 1833-9 and its two tillers, 1833-9a and 1833-9b. Pollen from 1833-9 produced 66 male-sterile offspring in a population of 2,219 plants. The tiller tassels of this plant produced no male-sterile offspring among totals of 998 and 1046 plants, respectively. Clearly, all or most of the 66 male-sterile plants in the progeny of 1833-9 are the product of a somatic (premeiotic) event in the tassel elements of this plant, presumably at about the 32-cell stage of tassel primordia. A similar event may well account for the high frequencies of male-sterile plants from male parent sources 930-4a and 931-6a, but in the cases of the two remaining sources, 930-1a and 930-5a, no such conclusion is warranted.

At this time we have no conclusive evidence that *Ac* insertion into *Rf2* is involved in the occurrence of the male-sterile plants. Alternatively they could be the result of some other kind of forward mutation of either *Rf1* or *Rf2*. For those cases that result from a premeiotic event in the male parent the mutation must involve *Rf2*, not *Rf1*, since the male parent genotype is known to be *rf1 rf1 Rf2 Rf2*.

Genetic studies underway now will determine whether or not *Rf2* mutation is involved in the production of the male-sterile exceptions, and whether they have occurred in both types of progeny, only one of which carries *Ac*. Laboratory investigation is being conducted to determine whether any of these mutations represent *Ac*-tagged *Rf2* alleles.

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Notes on identification and genetics of high methionine Zein-D (10kDa)

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D-zein is of interest because it contains 22% methionine, an essential amino acid. It has 129 amino acids (true molecular mass is 14kDa). The inbred BSSS-53 contains high levels of D-zein (and thus of methionine), with over-expression being regulated by a gene termed *Zpr10/(22)*. *Zpr10/(22)* is located on chromosome 4, while the structural gene *Zps10/(22)* is located on chromosome 9. See Kirihara et al. (MGG 211:477-484, 1988) and Benner et al. (TAG 78:761-767, 1989) for details. I prefer the name D-zein rather than 10kDa zein because SDS-PAGE does not provide the correct molecular mass for any zein. My map of zeins on agarose IEF gels places D-zein at position 55, thus giving the term D/55 to identify this zein by position on SDS-PAGE and IEF gels, respectively (Wilson, Plant Physiol. 82:196-202, 1986).

Although D-zein is apparently identified by its position on SDS-PAGE, errors are possible because its mobility varies with different gel conditions and position identification depends upon its mobility relative to molecular mass standard proteins, which also vary considerably in mobility. Other zeins may move to the same position on IEF, another cause of misidentification. IEF followed by SDS-PAGE may provide identification of D-zein, though I have noted an alcohol-soluble protein with an apparent mass of 12kDa which is probably not D-zein (unpublished work). Because D-zein is lacking in tyrosine, D-zein can be distinguished from other zeins after separation by HPLC by a very low absorbance at 280nm relative to 210nm (Wilson, Plant Physiol. 95:777-786, 1991). HPLC also provides a quantitative assay, and preliminary results suggested that inbreds other than BSSS-53 may have above average contents of D-zein.

Hartings et al. (Genet. Agrar. 38:447-464, 1984) found two classes of D-zein which differ slightly by both SDS-PAGE and IEF. I reported what appears to be a variant D-zein (by SDS-PAGE only) in *Z. diploperennis* (Plant Physiol. 82:196-202, 1986). Recently I found a variant D-zein in an experimental version of B73, provided by R. P. Bergquist (Pfister Hybrid Corn Co., El Paso, IL), which also had a lower mobility by SDS-PAGE. However, agarose IEF did not show a different position. Elution from an HPLC gradient was delayed by 2 min. Thus this variant can be easily detected and confirmed to be D-zein by HPLC. I made crosses to normal B73, to W64A, and to A619. Reciprocal hybrids revealed the expected two peaks, with relative peak heights suggesting that they are determined by co-dominant genes.

This program was terminated and I subsequently retired before I could complete a genetic analysis of these crosses. F1, F2, and backcross seeds (but not the experimental B73) are available for interested researchers. An HPLC analysis of A619 x ExB73 backcross seeds would confirm or eliminate, without using a translocation waxy marker inbred, the suggestion that the structural gene for D-zein is on chromosome 7 (Wilson et al., TAG 77:217-226, 1989) rather than on chromosome 9 (Benner et al., TAG 78:761-767, 1989).

URBANA, ILLINOIS
University of Illinois
LABEGE, FRANCE
Rustica Semences

Embryolike structure formation and subsequent callus induction from anther cultures of several genotypes

--Vincent Beaumont, Pierre Dieu and Jack Widholm

The anther culture ability of 12 genotypes of different origins was evaluated. The first group (6 genotypes) had previously been evaluated by Dieu and Beckert (Maydica 31:246-259, 1986) (RC, progeny from RC1XRC20 and NS) and FR16, Pa91XFR16, H99XFR16, H99XPa91 which had been cultured by Petolino and Jones (Crop Sci. 26:1072-1074, 1986). The second group came from crosses between RC20, RC1 and genotypes which are presently being used for breeding purposes by Rustica Semences, France (Table 1).

The material was derived from field-grown plants during the summer of 1990 in Urbana, Illinois. Tassels with uninucleate microspores were harvested in the morning, wrapped in moist paper towels and aluminium foil and cold-treated at 8 C. After 14 days, tassels were sterilized with a 0.5% sodium hypochlorite solution and 30 anthers were plated in 20x60mm dishes containing 10ml of medium. The medium was YP salts and vitamins with the addition

Table 1. Origin of the genotypes tested.

Code	Progenitors	Origin of the parents
S1	RC20 x 640[2] Sib1 Self1	640: group Mo17 and Oh43
S4	RC20 x 9000[2] Sib1 Self1	9000: group Iodent
S9	(RC20 x 3454)x M7	3454: group Oh43 and Mo17
S10	(RC20 x 9000)x 3546	3546: group B73
S11	[RC20 x(RC525 x Co158)] x 710	710: group B14 and Oh43
S13	(RCC1 x 3454)x 3461	3461: group Mo17

RC1, RC20, RC525 have already been described by Dieu and Beckert (1986).

of 500mg/l enzymatic casein hydrolysate, 0.1mg/l TIBA (2,3,5-triiodobenzoic acid), 60g/l sucrose and 5g/l activated charcoal which was filtered after autoclaving. The pH was set at 5.8 before autoclaving.

The dishes were kept in the dark at 28 C. After 3 weeks, the anthers started to produce some embryos. Embryos 2mm and larger were transferred onto D medium (Duncan et al., Planta 165:322-332, 1985) in order to produce haploid regenerable calli lines.

Despite the high number of anthers plated, the variability of the results is very high (Table 2). Thus, embryo induction appears to be very dependent upon the conditions of the donor-plant. The yield in embryos is around 1.4 per 100 anthers, which matches with the results from other authors (Dieu and Beckert, 1986; Petolino and Jones, 1986).

Table 2. Embryos and regenerable calli production from 12 genotypes.

Genotype	No. tassels tested	No. anthers plated	No. embryos produced	Embryos/10 0 anthers	Regenerable calli produced
S1	14	1170	21	1.00 ± 3.10	1
S4	10	810	0	--- ---	-
S9	32	2760	61	2.20 ± 2.00	4
S10	15	1260	0	--- ---	-
S11	28	2430	30	2.23 ± 2.00	6
S13	34	2940	36	1.23 ± 1.07	2
RC	15	1290	41	3.17 ± 2.23	0
NS	5	450	0	--- ---	-
PF	10	900	17	1.90 ± 1.63	8
HF	24	1980	35	1.73 ± 1.17	0
Fr16	17	1470	18	1.23 ± 0.70	0
HP	6	510	0	--- ---	-

Twenty-one callus lines which appeared to be regenerable were obtained from the 259 embryos produced. Three of the genotypes (RC, FR16 and H99xFR16) which produced a high number of embryos, did not produce any regenerable calli. Other results in this laboratory show that FR16 is very difficult to obtain regenerable callus from but H99xFR16 usually responds well.

Four of the genotypes in the second group gave some results, both in embryo induction and regenerable calli production. These results suggest that it should be possible to introduce anther culture ability into populations used for breeding purposes.

WALTHAM, MASSACHUSETTS
University of Massachusetts

A pristine background for studies on the origin and evolution of maize

--Walton C. Galinat

Use of a maize background, even though from primitive extant popcorns, for inheritance and evolution studies of the key-trait differences between teosinte and maize carries with it the assumption of a wild maize as the wild ancestor. Conversely, use of a teosinte background implies, and probably correctly so, that teosinte was the wild ancestor. But use of a pristine background, intermediate between teosinte and a reconstructed Tehuacan type of the oldest known maize, can be justified for studies on inheritance, rate of evolutionary emergence of maize and for opening new directions for maize evolution. The Tehuacan wild traits that have been recovered from extant maize for use in the pristine background are the string cob (*Sg1, Sg2*) from Confite Morocho, the interspace (*is*) from Coroico and the weak tunicate (*tu-w*) from Chapalote. This synthetic pristine background is easier to manipulate for controlled pollinations than that of pure teosinte because it has larger solitary ears compared with the tiny clustered spikes of teosinte but ears smaller than those of maize and it is easier to attach paper bags for pollination control in a cornfield situation. The pristine background also has the advantage of stabilizing key-trait expression (single vs. paired female spikelets and two vs. many ranks of spikelets). It will give more stable and repeatable results of genetic analysis at either the conventional or molecular levels.

A pristine background as a new direction for maize improvement

--Walton C. Galinat

The process of selection for an increased energy sink in the maize ear started during its origin with the maize-ear key traits taken from within teosinte. The concentration of energy into fewer larger ears, then as now, was from a redistribution of energy at the expense of the number of ears. The hundreds of tiny impoverished ears of teosinte borne in clusters (fascicles) at every leaf were reduced to solitary ears at most leaves in maize and then as the size of the kernels, ears and cobs continued to increase over the millenia, the number of ears per plant continued to be reduced. The most advanced breeds of modern maize would have only one giant ear were it not for the inadequate husk-coverage possible for such giant ears. The result has been the investment of energy in two or more smaller cobs with better husk protection. But even more important, the smaller, thinner cobs have an advantage in Canada, the northern United States and northern Europe, where there is a wet fall harvest season. The advantage is that because thin cobs dry down more rapidly, they are less inclined to become moldy. The preference for growing small grains instead of corn in these wet fall areas may be more than just eating customs and agricultural traditions. The small grain preference may be just the impractical nature of the thick moldy cobs of the corn types that we offer them. Yes, corn is good as silage in their area but as a feed grain plant, forget it. They know from sad experience.

A new dominant yellow silk (*Ys*) gene for sweet corn improvement

--Walton C. Galinat

Since Don Taylor came out with the idea in the early 1940's that dark inner silks in milk stage sweet corn looked like contamination with horse hairs, there has been a constant effort by sweet corn breeders to keep the inner silk color either white or yellow. The result is almost all of today's sweet corn has either yellow or white silks in the milk stage, and inner silk color is more important and can differ from outer silk color. In contrast, dent corn and flour corn usually have dark silk color so that sweet corn breeders who outcross to these non-sweet corns for yield, kernel and ear

factors, must in addition to selection to recover either *su* or *sh2*, select for white silks and other immature quality factors such as tenderness, sweetness, and flavor. The white silks are inherited as a recessive, usually due to *r* in *su* sweet corn and, due to linkage, *a* in *sh2* super sweets. This means that recessive white silks must be transferred to both parents for expression in the F1 hybrid.

This is not the case with the dominant *Ys* gene. It need be present in only one parent of the hybrid. It is a paradox that it is linked to *su* just as *a* white silk is linked to *sh2*. The *Ys* (yellow silk) gene came to me with some EMS mutants supplied by Jerry Neuffer.

Sexual transmutation consistent with the cupule reduction mode of maize's origin

--Walton C. Galinat

Most of the oldest Tehuacan and all of the oldest Bat Cave cobs are like most modern corn in having well-developed cupules and elongated female rachillae. The pronounced cupules are female secondary sex traits of teosinte and the elongate rachillae are female baggage from the tunicate allele carried by Chapalote maize. They are not part of the complex of male secondary sex traits from teosinte which includes reduced cupules, short rachillae, paired spikelets, and foliaceous glumes. Since the entire male secondary sex baggage is not transported into the female rachis of these particular archaeological cobs, the origin of maize from teosinte in these cases, representing the parviglumis-Northern Flint pathway by rachilla elongation, is not consistent with the expectations of the CSTT theory of Iltis (Science 22:886-894, 1983).

But cupule reduction and short rachillae do occur in a different grouping of maize races, the pathway that I have termed Chalco-Southern Dent (Galinat, Agronomy 18:1-31, 1988). Furthermore, *ts2* (tassel seed 2) derivatives of both teosinte and *Tripsacum* have paired female spikelets with foliaceous glumes, reduced cupules and short rachillae in their "tassels" that are similar to certain Tehuacan specimens. Thus, contrary to what I have stated previously, there is some evidence that Iltis might correctly interpret as consistent with his CSTT. I should add that the string-cob trait of Confite Morocho involves cupule reduction and short rachillae controlled by the *Sg1* and *Sg2* genes without any transmutation of the tassel. Confite Morocho appears to be an ancient isolate of the Tehuacan maize that drifted to Peru at an early time.

The use of U.V. radiation to help rescue fungal infected seed

--Walton C. Galinat

The installation of an ultra-violet radiation tube in my seed germinator has been helpful in obtaining plants from slightly moldy seed. The seed is placed on wet filter paper in an uncovered glass petri dish and exposed to U.V. radiation for two days or until there are signs of embryo growth. At that point the U.V. radiation is turned off, or the glass cover is put on the petri dish. The sprouted seed is potted up and eventually transplanted to the field. Thus, seed which normally would have died from fungal infection is rescued. Exposure of the sprouted seed to the U.V. radiation is lethal. The removal of a pathogen from an inbred line may improve it with a vigor jump.

WEST LAFAYETTE, INDIANA
Purdue University

Placement of teosinte branched on the linkage map

--Patricia S. Springer and Jeffrey L. Bennetzen

We have undertaken a mapping project using RFLP markers on chromosome 1L in order to more accurately place teosinte branched on the map. In an experiment to identify plants that had undergone recombination events between *tb* and *Adh1*, progeny of the cross *Tb Adh1-S/tb Adh1-F* X *tb Adh1-F/tb Adh1-F* were analyzed. Recombinant plants were teosinte branched and *Adh1-S/Adh1-F* or normal and *Adh1-F/Adh1-F*. In an analysis of 222 progeny, 10 recombinants (3 teosinte branched, *Adh1-F/Adh1-S* and 7 normal, *Adh1-F/Adh1-F*) were identified. This is an approximate distance of 4.5cM. The parents differed at linked RFLP markers *BNL8.10*, *Phy1*, and *Kn1*. This analysis allowed us to determine that *Tb* is proximal to *Adh1*, and that the gene order is *BNL8.10 - Tb - Phy1 - Kn1 - Adh1 - BNL7.25*. We are in the process of analyzing more RFLP probes in order to obtain a more detailed fine structure map of this region. The purpose of these experiments is to facilitate chromosome walking around *Adh1*.

YANGZHOU, CHINA
Jiangsu Agricultural College

Study on male sterility. VIII. Identification of a group of YII-1 type male-sterile cytoplasm

–Taichen Qin, Jianguo Chen, Mingliang, Xu, Dexiang Deng and Yunlong Bian

The relationship between maintainer and restorer, the number of restored genes, the resistance against *Helminthosporium maydis* race T and the banding pattern of mitochondrial DNA of T, C and S group male-sterile lines, as well as the YII-1 type male-sterile line we created by means of combining the cytoplasm of restored line with maintainer nuclei, were studied. The results show that there are many more restorer lines for the YII-1 type male-sterile line than for the T and C groups, but fewer than for the S group. The character of the restorer gene of the YII-1 type is different from that of the C group. Inoculating with *Helminthosporium maydis* race T and its pathotoxin (T-toxin), YII-1 type shows a much higher resistance than that of T, C and S groups (Fig. 1, Fig. 2). The modified method of Kemble and Pring was used for identification of male-sterile line YII-1. Figure 3 shows high molecular weight bands were present in all lines. The S group possesses two unique mtDNAs, S1 and S2, that may be distinguished from other male-sterile cytoplasms. There are no

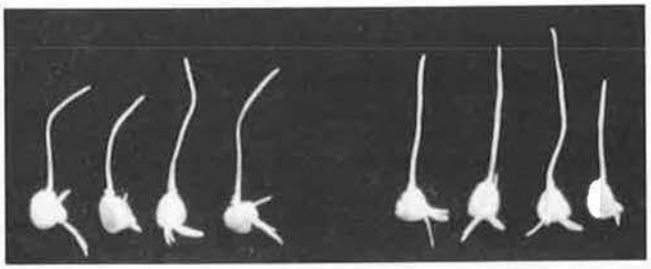


Figure 1. The root length of Y-cytoplasm hybrid YII-1 B37 x Jin03. Left, T-toxin treatment: T-toxin did not inhibit the root growth of Y-cytoplasm corn, and the root length is equal between T-toxin treatment and check. Right: without treatment with T-toxin (check).

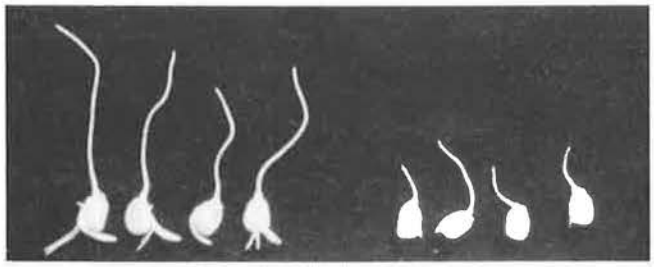


Figure 2. The root growth of T-cytoplasm hybrid cms-T B37 x Jin03. Left: without treatment with T-toxin (check). Right, T-toxin treatment: T-toxin highly inhibited the root growth of T-cytoplasm corn, and the root length in T-toxin treatment is about one third as long as that in check.

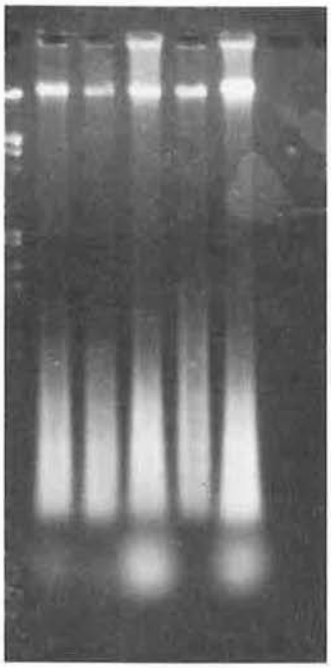


Figure 3. The tracks are respectively: Marker DNAs from bacteriophage digested with *Hin*dIII; Normal (N); T group; C group; S group; Y type.

obvious differences in mitochondrial DNA (mtDNA) banding pattern after agarose gel electrophoresis between T, C and YII-1 type male-sterile lines, but no S1 and S2 band within YII-1 type. YII-1 type may be a new type of male-sterile cytoplasm and different from the known groups.

ZEMUN-BELGRADE, YUGOSLAVIA
Maize Research Institute

Allozyme polymorphism of maize plants transformed by bacterial gene integration

--Kosana Konstantinov, Snezana Mladenovic, D.Kovacevic, J.Dumanovic and Bojana Tadic

Transformation of the inbred lines B73 and Mo17 was performed by pollen tube pathway and dry seed incubation in plasmid DNA solution. Plants, self-pollinated by pollen grains pasted both in plasmid pRT100neo (Topfer et al., Nuclei Res. 15(14):5990, 1987) and total DNA of *A. tumefaciens* strain B6S3, were labelled as T0 plants. Also, plants derived from the seeds germinated and grown for 5 days on medium containing plasmid pRT100neo DNA (designated as cocultivated seeds) were labelled as T0 plants. In these plants, obtained from cocultivated seeds, neomycinphosphotransferase activity was determined, and plants with the enzyme in active form propagated to obtain T1 and T2 plants (Mladenovic et al., Genetika 23(3), 1991). Dot-blot and Southern blot analysis, by plant DNA hybridization with a specific probe, showed both NPTII gene integration and transmission through plant generations (Konstantinov et al., Genetika 23(2), 1991).

Bacterial DNA integration induced several phenotypic changes, such as barren stalk, curled (rolled) leaves, dwarfs, anthocyanin synthesis, that were almost eliminated in the next generation. Plant height alteration and reduction of the period from seed sowing to flowering (days to flowering) were inheritable and no segregation was obtained in the T2 generation.

To test the level of genetic divergence of "early" flowering transformed plants from the original inbred lines, allozyme polymorphism was determined in typical T1 plants. Starch gel electrophoretic systems were employed to investigate several allozyme genotypes from coleoptile tissue (according to Stuber et al., N.C. State Exp. Res. Bull. No 286, 1988): acid phosphatase (*Acp1*), isocitrate dehydrogenase (*Idh1,Idh2*), malate dehydrogenase (*Mdh1, Mdh2, Mdh3, Mdh4, Mdh5*), phosphoglucomutase (*Pgm1, Pgm2*), 6-phosphogluconate dehydrogenase (*Pgd1, Pgd2*), phosphohexose isomerase (*Phi1*), betaglucosidase (*Glu1*) and arginine aminopeptidase (*Amp1, Amp3*). Distinct alterations were found in six of the 16 analysed loci. The results are presented in Table 1. The pattern of two enzyme systems, *Acp1* and *Mdh*, was changed in plants belonging to both B73 and Mo17 genotypes, transformed either by plasmid pRT100neo or *A. tumefaciens* B6S3 DNA. Characteristic electrophoregrams are presented in Figs. 1 and 2. Experiments, including bacterial gene localization in maize chromosomes and studies on allozyme polymorphism in T2 plants are in progress.

Table 1. Allozyme genotypes of the transformed plants. Numbers and letters refer to allelic variants present in 5-day old coleoptile tissue.

Inbred/genotype		Alleles with genotype alteration					
T1 plants	lane[1]	*Acp1*	*Mdh1*	*Mdh2*	*Mdh5*	*Idh2*	*Phi1*
B73-control		2/2	6/6	3.5/3.5	12/12	4/4	
B73-cocultivated plants							
plant 1	1	2/4	10.5/10.5	3.5/6	15/N	4/6	
plant 2	2	2/4	10.5/10.5	6/6	15/N	6/6	
B73 - pollen pasted in pRT100 DNA							
plant 1	3	2/4	*	3.5/6	*	4/6	
plant 2	4	*	*	3.5/6	*	4/6	
plant 3	7	*	*	3.5/6	*	4/6	
plant 4	8	*	*	3.5/6	*	4/6	
plant 5	9	4/4	*	6/6	*	6/6	
plant 6	10	4/4	*	6/6	*	6/6	
plant 7	11	*	*	6/6	*	6/6	
plant 8	12	*	*	3.5/6	*	4/6	
B73 - pollen pasted in B6S3 DNA							
plant 1	5	2/4	*	6/6	*	6/6	
plant 2	6	2/4	*	6/6	*	6/6	
Mo17 - control		2/2	6/6	6/6	12/12	4/4	4/4
Mo17 - pollen pasted in PRT100neo DNA							
plant 1	13	2/4	*	*	*	*	
plant 2	14	4/4	*	*	*	*	
Mo17-pollen pasted in B6S3 DNA							
plant 1	15	2/4	*	3.5/3.5	*	*	4/5
plant 2	16	2/4	*	3.5/6	*	*	5/5

*no changes
[1]number corresponds to the lanes on electrophoregrams (Fig. 1 and Fig. 2)

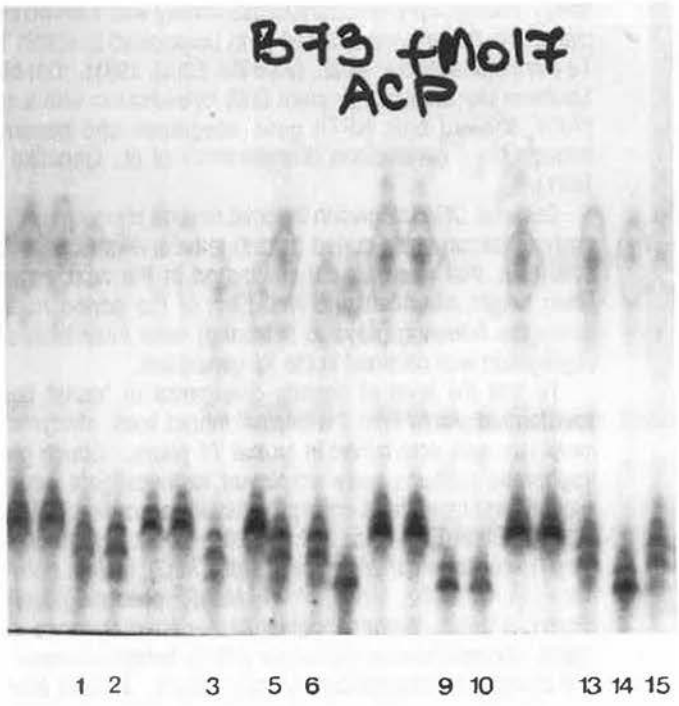


Figure 1.

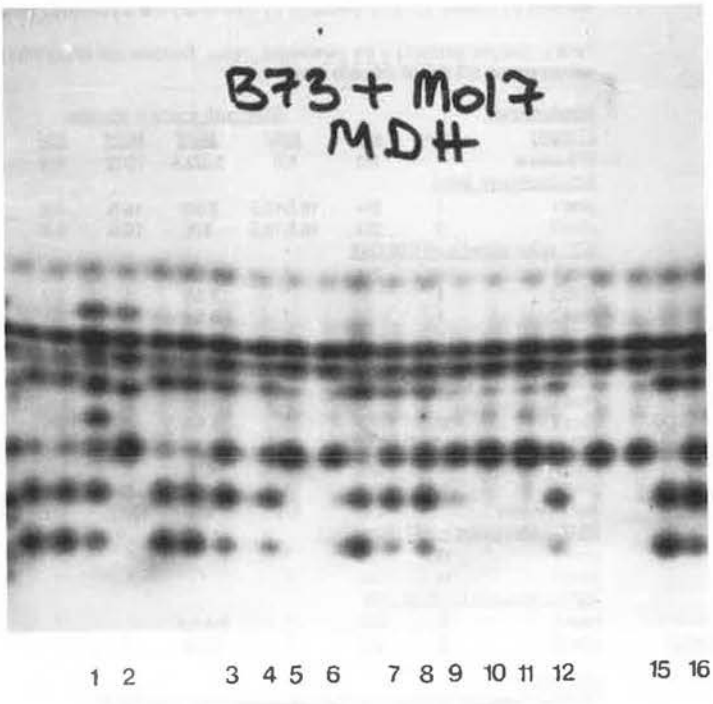


Figure 2.

III. ZEALAND 1992

This is a summary of selected genetic research information (e.g., new factors; mapping; cloning) reported in recent literature and in this News Letter ("r" refers to numbered references in the Recent Maize Publications section). The Symbol Index refers by number to all current published research involving genetic materials. Comments or suggestions on these research aids would be welcome.

It has not been feasible this year, because of the demands of the database effort, to summarize the information on "OTHER INHERITANCE; GERMPLASM", including resistances, quantitatively inherited traits, and germplasm characterizations. Systematic treatment of multifactorial information, or factors controlling characters that are subject to environmentally sensitive measurable variations, is yet elusive, either for the "Zealand" paradigm or for database representation. Prof. Ligate expresses regrets for this unavoidable slippage in coverage.

BS = Base Sequence; RM = Restriction Map.
* with symbols identifies loci needing allelism tests, documentation, or standardization of the symbol.

CHROMOSOME 1

an1-891339, genomic, *Mu*-induced --66:51

BNL7.25 - (rd1, NPI238) - BNL8.29A --66:93

BNL7.25 -6- *(ij2, BNL8.29A)* -11- *BNL6.32* --66:107

BNL8.05 - BNL5.62 - AGRr152 - AGRr22 - AGRr294A - AGRr77 - AGRc467 - UMC76 - AGRr92A - P1 - BNL12.06 - AGRr117 - AGRr153A - BNL7.21A - AGRr88A - AGRc634 - AGRx23 - AGRc516 - AGRr197 - BNL1.556 - Ocsbf-1 - UMC133 - BNL5.59 - AGRc584 - AGRr193 - AGRc587 - AGRc569 - AGRc512 - AGRr200 - AGRr175 - AGRr291 - AGRr250 - AGRc489 - AGRr185 - AGRp83B - AGRx1176 - AGRr110 - AGRr71 - AGRr299 - BNL8.10A - AGRr235A - AGRc362B - AGRr238A - AGRr246 - BNL15.18 - AGRr34A - AGRr278A - Adh1 - AGRr290 - BNL7.25 - Bm2 - BNL8.29A - AGRr103B - AGRc669B - AGRc707 - AGRc259A - BNL6.32 --66:66

UMC11 - AGRr92A - UMC13 --66:66

AGRc587 - UMC58 - AGRc512 --66:66

BNL7.25 - AGRr88b - AGRc707 --66:66

BNL8.10 - tb1 - Phy1 - Kn1 --66:116

bz2, homology to *Gmhsp26-A* of soybean --66:104

cpn60-1*, on 1L, cDNA; genomic, chaperonin; heat shock protein 60k --66:51

dek1-PI-AEO, floury endosperm, pigments absent, germless --66:6

dek32 (was *shop*-1322A*), on 1S, defective kernel: dull opaque dented normal size non-viable kernel; endosperm floury and soft, embryo tiny, degenerate and necrotic resting in a much larger embryo cavity; not allelic to *dek1* --66:39

emb-8502; emb*-8503; emb*-8517; emb*-8519; emb*-8520; emb*-8531; emb*-8545; emb*-8549*, on 1S --r105

emp1 (was *dek-Mu*2045*), on 1S, empty pericarp, germless --66:8

Glb1, pgGlb1S; pgGlb1-L, cDNA; genomic, RM, BS: X590984; X59083; X59085, unequal crossingover --r45

Kn1, cDNA, BS, homeobox --r634

Kn1, RM --r624

o10 -34- *zb7* -42- *bm2* --66:93

P1, cDNA, RM, BS: M62878; M62879 --r222

P1-ovov; P1-ww-d, RM, BS --r25

ptd1 (was *dek-Mu*1568*), pitted endosperm, lethal --66:8

py2 -17- *BNL6.32* --66:107

UMC94 - BNL5.62 - PIO200537 - PIO200603 - PIO200689 - PIO200640 - UMC76 - UMC137 - UMC11 - UMC13 - UMC1C12 - BNL7.21 - UMC133 - PIO200654 - BNL5.59 - PIO200575 - PIO200674 - PIO200644 - PIO200855 - UMC23 - UMC33 - PIO200668 - PIO200661 - UMC128 - UMC50 - BNL8.10 - PIO200518 - BNL7.25 - BNL8.29 - UMC84 - PIO200557 - BNL6.32 --66:52

wlu5 near br2 --66:93

CHROMOSOME 2

AGRr13 - AGRc805 - AGRc938 - Lg1 - AGRc539A - UMC6 - AGRp168A - UMC8B - AGRc321 - AGRr113A - AGRr167A - BNL10.42A - AGRp54a - AGRc593 - AGRp173 - AGRr216A - AGRr85A - UMC8C - AGRr267B - AGRp58 - AGRr85B - AGRr239 - AGRp62 - UMC139 - AGRr265B - AGRc333A - AGRc479 - AGRc939 - BNL8.21B - UMC122 - UMC150B - BNL6.20 - AGRx825 - C2B(whp) --66:66

UMC5 - AGRr111b - AGRc939 - BNL6.20 - rDNA5S - C2B(whp) - AGRx825 --66:66

UMC6 - UMC44B - UMC61 - UMC134 - UMC8B - UMC8C - UMC139 - UMC5 - AGRc333A --66:66

B1-Peru, cDNA, BS: X57276, myc homology --r499

BNL6.20 -12- *rDNA5S* -11- *C2B(whp)* --66:71

dek-PI-C3; dek*-PI-E3*, on 2L --66:6

emb-8518*, on 2L --r105

emp2 (was *dek*-1047*) -18- *w3*, empty pericarp, germless --66:6

Ht1 -1- *UMC150B* -5- *BNL6.20* --r47

Les11 (was *Les*-1438*), -14- T2-9c *wx1*, Like *Les1* but slightly later in expression and less responsive to temperature and to background genotype; EMS-induced --66:39

Les15 (was *Les*-2007*), -2- T2-9b *wx1*, tiny yellowish green plants with many small chlorotic and necrotic lesions on speckled yellow green leaf blade background that looks like iron deficiency symptoms; EMS-induced --66:39

NPI298 -19- *whp1* -6.4- *Rf3* -6.4- *BNL17.14* --66:45

Rf-CE1* -9- T2-9d *wx1* --66:111

Rf-III* -3 - T2-9d *wx1* --66:113

whp1, Whpc34, genomic, BS: X60204 --r188

CHROMOSOME 3

a1, intralocus recombination --r76

BNL8.15 - AGRr209A - UMC121 - AGRr116A - BNL8.35 - AGRc923 - AGRp50 - AGRc476A - AGRr206 - AGRc514A - AGRc332 - AGRp97 - AGRr19 - AGRp91 - AGRr179 - BNL6.06 - Aldolase - BNL5.37 - AGRr271 - AGRr184B - AGRc435 - BNL8.01 - AGRr274A - UMC60 - AGRp40 - BNL15.20 - BNL6.16 - AGRr144A - AGRr50A - AGRc461 - A1 - AGRc568A - AGRr43B - AGRc638 --66:66

BNL8.35 -10- *UMC10* -1.4- *Lg3* -9- *NPI219* --66:21

emb-8512; emb*-8514; emb*-8515; emb*-8521; emb*-8532; emb*-8551*, on 3L --r105

et1-43 (was *dek*-43*) --66:6

Les14 (was *Les*-2004*), -1- T3-9(8562) *wx1*, Many small round brown necrotic spots with light centers, some with anthocyanin halos on leaf blade beginning at the 6 leaf stage, no reduction in height or vigor; EMS-induced --66:39

Les17 (was *Les*-2345*), -3- T3-9c *wx1*, Profuse small to medium chlorotic and necrotic lesions expressed at 8-10 leaf stage causing plants to have a light green color; occasional normal green non-lesion sectors appear on leaves; spontaneous --66:39

NPI446 - Tpi4 - NPI220B -3.2- *BNL13.05B - NPI114B - BNL6.06* --r573

Sdw2 (was *D*-1991*), -6- T3-9c *wx1*; -8- *Lxm1*, Short plant, 1/3-1/2 normal height, with normal green erect leaves; does not respond to gibberellins; no anthers in ear; nitrosoguanidine-induced --66:39

vp1, pcvp23; pcvp11, cDNA, RM, BS: M60214 --r400

CHROMOSOME 4

AGRr115 - AGRc94 - Adh2 - UMC31A - BNL5.46 - AGRc39 - AGRr109 - AGRr301 - AGRp67 - AGRr321 - AGRc567 - AGRr190 - AGRr89 - Su1 - AGRr62 - AGRp83A - AGRr286 - BNL15.45 - AGRc4 - AGRc563B - AGRr27 - BNL10.05 - BNL7.65 - AGRc303a - C2a - AGRr324 - AGRp166 - AGRc300 - AGRr273 - BNL15.07 - AGRr248A - Ubi1 - AGRc445A --66:66

AGRc303A - AGRr248A - BNL8.23 --66:66

BNL5.46 -13- *su1* -1- *tga1* -1- *UMC42*, teosinte glume architecture: glumes indurated, erect, long, boat-shaped; factor transferred from teosinte --66:95

c2, C2Lc30, genomic, BS: X60205 --r188

dek-3252*, on 4S --66:6

dek7 -9- *su1* -18- *gl4* and *fl2* -7- *dek7* --66:6

dek25 -25- *fl2* -19- *su1* --66:6

dsc1-3252 (was *dek*-3252*), on 4S; crumpled, discolored, lethal; allelic *dek*-2058* --66:6

emb-8501; emb*-8509; emb*-8513; emb*-8534; emb*-8537; emb*-8538; emb*-8540; emb*-8547*, on 4L --r105

(Gpc3, Gpc1) - UMC42 --r534

Ms44 -1 - *c2* --66:49

su1 -9- *gl4* -23- *dek31* -18- *c2* --66:6

*Ys**, near *su1*, yellow silk --66:115

CHROMOSOME 5

a2 -19- *ae1* --66:3

AGRc259B - AGRc926 - AGRc66 - AGRc669A - AGRp53 - AGRr103A - BNL6.25 - AGRc23 - AGRx43 - Phy2 - AGRr278B - AGRc362A - AGRr238B - AGRc329 - AGRp52 - AGRr235B - AGRr199 - AGRr70 - AGRr142 - BNL6.10 - BNL6.22 - BNL10.06 - BNL7.71 - Ubi2 - AGRc637 - AGRc814 - AGRr248B - AGRx1128 - AGRr127 - AGRr106 - AGRr298 - AGRr215 - BNL5.40 - UMC51 - AGRx701 - AGRp90 - AGRr252 - UMC108 - AGRr288 - AGRc563A - AGRr45A - AGRr211 --66:67

AGRc926 - AGRr34b - AGRc329 --66:67

*cpn60-2**, on 5S, cDNA; genomic, chaperonin; heat shock protein 60k --66:51

Dap1-2 (was *Dap2*), mosaic aleurone color --66:6

dek-NS8070*, on 5S --66:6

dek33 (was *fldt*-1299*), *a2* -7- *bm1* -13- *dek33* -21- *pr1*, defective kernel: opaque floury dented wrinkled non-viable kernel of nearly normal size; endosperm floury with little or no corneous starch; embryo variably degenerate, arrested at Abbe and Stein stage 3; occasionally viviparous; cultured immature embryos germinate to produce amorphous masses of callus and distorted shoots and roots --66:39

emb-8504*, on 5S --r105

Hsf1 -0- *Hsf*-1603* --r56

ren1 (was *dek*-807*) ; *dek*-PI-OI*, on 5L, reduced endosperm, opaque, lethal --66:6

sh4-5133 (was *dek*-5133*), shrunken, collapsed, chalky endosperm --66:6

vp2 -2- T5-9(4817) --66:9

CHROMOSOME 6

AGRc67 - BNL10.42B - UMC85 - AGRp144 - AGRc3 - AGRc12 - AGRc611 - Zein15B - AGRr221 - Pgd1 - Enp1 - Y1 - AGRr47 - AGRr87 - AGRr189 - UMC59 - UMC65L - AGRr37 - AGRr118A - BNL15.37 - AGRr261 - BNL5.47A - UMC42 - UMC28 - AGRr213 --66:67

AGRr37 - AGRr118A - BNL3.03 --66:67

UMC85 - AGRr118A - BNL3.03 - BNL5.47 - UMC138 - UMC42 --66:67

Les13 (was *Les*-2003*), -11- T6-9b *wx1*, Frequent small to medium necrotic spots on leaf blade, sheath and culm, appearing at the 5 leaf stage; some enlarge and coalesce to form long necrotic strips along leaf veins, eventually spreading and causing senescence of the whole leaf; plants are lighter green and 1/3 to 2/3 normal height; EMS-induced --66:39

o14 (was *o*-924*), on 6L, distal to *Pl1*, opaque kernel: Large opaque kernel with mostly floury starch except for a small amount of corneous starch near the base on the abgerminal side; normal green seedling develops yellow striped appearance and is slow in growth --66:39

UMC85 - Mdm1 - BNL6.29 - y1 - UMC59 --r357

Wsm1, (Wsm1, po1, BNL6.29 ,UMC59) - UMC21, wheat streak mosaic virus R --r403

CHROMOSOME 7

AGRc261 - AGRc36 - AGRr128 - AGRr49 - AGRr267A - AGRc203 - AGRr241 - AGRr168 - BNL15.40 - Zein27G - AGRc914 - AGRr265A - AGRr111A - AGRr174 - BNL15.21 - AGRr73 - AGRc542 - AGRc701 - AGRr207 - BNL8.37 - BNL7.61 - BNL8.21A - AGRr131 - BNL14.07 - AGRr101 - AGRr132 - AGRr186 - AGRc6 - AGRr202 - BNL16.06 - AGRr55 - BNL8.44 - AGRc525 --66:67

AGRr241 - (AGRr111A, AGRc333B) - AGRr132 --66:67

AGRr73 - UMC57 - BNL14.07 - AGRr132 - AGRr44 - AGRr186 --66:67

BNL14.07 -3- *ren2-413* (was *dek*-NS413*) -5- *BNL8.39, ren2* reduced endosperm, lethal; allelic *dek*-NS326* --66:6

ij1, RM, BS --66:32

ptd2 (was *dek-Mu*3193*), pitted endosperm, lethal --66:8

ren2 (was *dek*-NS413, dek*-NS326, dek*-95*), reduced endosperm, on 7L --66:6

Rs1 -6- *PIO200581B* -19- *NPI400* -13- *o2* --66:23

sh6 (was *sh*-1295*), on 7S, shrunken opaque kernel like *sh1* but more extreme; normal size; pale green lethal seedling --66:39

sh6, left of TB-7Sc, not allelic to *cp2* --66:4

CHROMOSOME 8

BNL13.05A - AGRr169 - BNL9.11 - UMC103 - AGRr116B - BNL8.10B - AGRr209B - BNL10.39 - AGRc514b - AGRc20 - AGRx975 - AGRc747 - AGRr222 - AGRc478 - BNL8.26 - AGR6-1-1 - UMC89 - BNL12.30 - AGRc568B - AGRr50B - UMC48 - AGRr144B - AGRr51 - UMC150A - AGRr322 - AGRr184A - AGRr269 - J1 - AGRr274B - AGRr262 - UMC7 - AGRr21 --66:67

UMC124 - AGRr116b - UMC89 - UMC93 - UMC30 - UMC117 - AGRr322 --66:67

des17, right of TB-8Lc, defective seedling: reduced height, partial suppression of primary root growth, contorted leaves, lethal but responsive to yeast hydrolysate --66:80

hcf134, on 8L, pale seedling, mutable, lacks PSII core complex; *Mu*-induced --66:43

pro1 (= o6) -4- *b-32** --66:20

pro1-5132 (was *dek*-5132*), crumpled, opaque, lethal --66:6

Rf-I* -7 - T8-9(043-6) *wx1* --66:113

Rf4 -1.5- *NPI114A* -4.5- *NPI220A* --r573

UMC89 -9- *Ht2* -5 - *UMC48A* -1 - *UMC30* -9 - *AGRr51* -4 - *UMC150A* --66:70

CHROMOSOME 9

AGRr41 - Sh1 - AGRr147 - Bz1 - AGRc255B - AGRr58 - AGRc273 - AGRr205 - BNL3.06 - Wx1 - AGRr125 - AGRr64 - BNL5.04 - BNL7.13 - AGRr153B - Ss2(Css1) - AGRr90 - BNL8.17 - AGRr171 - AGRp1000 - AGRc595 - BNL5.09 - AGRr294B --66:68

AGRr118B - AGRr41 - Bz1 - AGRr205 - Ocsbf-2 - AGRr153B - AGRc595 - AGRr92B - AGRr294B --66:68

AGRr41 - UMC109 - AGRr118B - AGRr147 - Bz1 - (BNL3.06, AGRr205) - UMC81 - AGRr64 - AGRc445B - BNL7.13 - Css2 - AGRr153B - AGRr90 - AGRp1000 - AGRc595 - AGRr92B - BNL5.09 --66:68

bz1-m4D6843B, RM --r318
C1 - T3-9(6722) - *Bz1; Bz1*- T8-9(4463) - *Wx1*, distal duplication --r89
emb-8522*, on 9S --r105
gl15 -0- *(BNL5.04, BNL7.13)* --66:107
wx1-1240, genomic, RM, BS --r451

CHROMOSOME 10
BNL3.04 - *Rp1-G* - *(Rp1-I, Rp5)*- *NPI422* --66:71
BNL3.04 -1.5 - *Rp1-G* -1-*(Rp1-I, Rp1-F)*- *NPI422* - *NPI285* -10- *oy1* --r270
BNL10.17 - *AGRr43A* - *BNL3.04* - *AGRc561* - *AGRc714* - *AGRc528* - *AGRc255A* - *BNL7.49B* - *AGRc690* - *AGRr18* - *AGRr232* - *AGRr216B* - *AGRr57* - *AGRr104* - *AGRr255A* - *AGRr295* - *AGRr113B* - *AGRc459* - *G1* - *AGRp168B* - *UMC44A* - *BNL7.49A* --66:68
UMC44A - *BNL7.49A* - *AGRr167B* --66:68
cr4 -10- *oy1* -8- *y9* --66:3
Les12, Lesion (was *Les*-1453*), on 10, Many small to medium, chlorotic to necrotic lesions form in clusters on the leaf blade beginning at 5 leaf stage and rapidly coalesce to form large senescent areas that may spread over the whole leaf and cause early death; EMS-induced --66:39
Les16 (was *Les*-2016*), -10- T9-10b *wx1*, Pale green plant develops small chlorotic lesions on the leaf blade just before flowering; EMS-induced --66:39
PIO200075A -21- *oy1; oy1* -5- *PIO060005* --66:107
R1 (P) (S) (Q), pR-nj:1; p323I14, genomic, RM --r519
Sn1-bol3, genomic; cDNA, RM --r609
sr2 -6-*UMC44A; sr2* -10- *BNL10.13; sr2* -8- *R1; sr2* -8- *BNL17.02* --66:107

UNPLACED
bif-47330*, barren inflorescence: tassel branches and ear and tassel spikelets variably fail to develop --66:51
dib1, dichotomously branched: plants branch into two normal tops at nodes 4-8 variably, associated with aneuploidy --66:86
Gpc4, glyceraldehyde-3-phosphate dehydrogenase, EC1.2.1.12 --r534
hcf-124*, pale seedling, fails to accumulate multiple photosynthetic complexes/assemblies; *Mu*-induced --66:44
hcf-129*, pale seedling, few ribosomes; *Mu*-induced --66:44
hcf-131*, pale seedling, few ribosomes; *Mu*-induced --66:44
hcf-133*, pale seedling, few ribosomes; *Mu*-induced --66:44
hcf-136*, pale seedling, defective in endolytic cleavage of transcript for *pet*B and *pet*D; *Mu*-induced --66:44
hcf-137*, pale seedling, fails to accumulate multiple photosynthetic complexes/assemblies; *Mu*-induced --66:44
hcf-139*, pale seedling, fails to accumulate multiple photosynthetic complexes/assemblies; *Mu*-induced --66:44
hcf-140*, pale seedling, fails to accumulate multiple photosynthetic complexes/assemblies; *Mu*-induced --66:44
hcf-142*, pale seedling, fails to accumulate *pet*A transcript; *Mu*-induced --66:44
hcf-143*, pale seedling, aberrant transcripts of *rps12* and *atp*F; *Mu*-induced --66:44
hcf-146*, pale seedling, few ribosomes; *Mu*-induced --66:44
HSP-17*+; HSP-17-, presence-absence; segregation 90:45 --r457
NPI114C -15.6- *BNL13.05C* --r573
ns1, narrow sheath and lower blade, brachytic plant; not allelic to *br1, lg1, lg2* --66:49
pra1, prophase I arrest --r212
su3, not allelic to *sh1, su1, su2, ae1, du1, sh2, bt1, bt2* --66:4
tsh-50330*, tassel-sheath: leaves develop at base of each tassel branch, spikelets, and cupules --66:50
vp10 (was *vp*-86GN5*), viviparous: yellow endosperm, colored aleurone, green seedlings, adherent --66:34
vsi1, variable short internodes: clusters of 2-4 short internodes --66:38
zein27kD-SA; zein27kD-SB; zein27kD-Ra; zein27kD-Rb, RM, BS, zein-27kD, recombination --r124

B CHROMOSOME
(no entries this year)

CLONES, PROBES
3A6; 3C12; 4H3; 4H7, cDNA, BS: X57272; X57274; X57273; X57275, pollen-specific --r522
*Abp**, genomic, BS: X56737, auxin-binding protein --r688
anp27, cDNA, anaerobic protein; homology with *Mu1.7* and MrsA --66:21
*Ant**, MANT1; MANT2, cDNA, BS: X57556; X59086, adenine nucleotide translocator --r669
b-70*, pcb70.2, cDNA, BS: M59449, induction --r69
beta-1,3-glucanase, cDNA --66:109
Bg, rbg, RM, BS --r247
Bg, RM, BS: X56877 --r246
cab-m9*, cDNA, BS: X55892, chlorophyll a/b binding protein --r632
cdc2, cdc2ZmA; cdc2ZmB, cDNA, BS: M60526, p34cdc2 protein kinase --r108
cDNA, invertase --66:46
chitinase, cDNA --66:109
DHPS*, pZMDHPS5, cDNA, BS: X52850, dihydrodipicolinate synthase, EC4.2.1.52 --r193
EMB564*, cDNA, BS: X55388, ABA-inducible --r658
enolase, pZM245, cDNA, BS: X55981 --r336
Fdl; FdlII, pFD5; pFD1; pFD1'; pFD3, cDNA, RM, BS, ferredoxin --r248
Glb2, pcGlb2, cDNA, BS: X53715 --r639
histone H1*, cH1C21, cDNA --r503
HMG1, cDNA, RM, BS, high mobility group protein --r218
HSP-15-18, ZmEMPR9', genomic, BS, small heat shock protein; meiotic --r139
HSP18, cMHSP18-3; cMHSP18-9, cDNA, BS: X54075; X54076, heat shock protein-18kDa --r213
In2-1*, In2-2*, pIn2-1.12; pIn2-2-3, cDNA, BS, polypeptide induced by safener; pathogenesis-related protein? --r257
kinase, p90.7, cDNA, BS, serine/threonine kinase catalytic domain --r59
MG19; MG14, cDNA, BS: X15704, alpha tubulin --r423
MT-L, pCIB1325; pCIB1324, cDNA; genomic, RM, BS, metallothionein-like gene --r129
Mu-A2, RM --66:6
Mu1, BS: X53604 --r185
MuA, RM, cosegregation with *Mu* activity --r495
MuA2, RM, BS, cosegregation with *Mu* activity --r494
MuR1, RM, Mu-Regulator1 --r101
NiRase, pCIB808, cDNA, nitrite reductase, EC1.6.6.1, light/dark --r70
NRase, pCIB831, cDNA, nitrate reductase, EC1.6.6.4, light/dark --r70
oleosin-18kD, KD18, cDNA --r496
Pdc1, genomic, BS: X5946, pyruvate decarboxylase --r307
PDK*, C4PPDK; ppdk1; ppdk5; ppdk11, cDNA; genomic, RM, BS, pyruvate, orthophosphate dikinase --r561
pFDGGT11; pFDGGT53, cDNA, RM, BS: J05739, glutamate synthase (ferredoxin), EC1.4.7.1 --r543
PG*, PG1, cDNA, RM, BS: X57575; X57627; X57628; X57743, polygalacturonase family --r443
Pltp1, 9C2; 6B6, cDNA, RM, BS: M57249, phospholipid transfer protein --r19
PRms, B8A2, cDNA; genomic, RM, BS, pathogenesis-related protein,

maize seed --r92
SPS, cDNA, BS, sucrose phosphate synthase, EC2.3.1.14 --r675
Ssu; rbcS*, rbcSZm2; rbcSZm3; rbcSZm1, genomic, RM, BS --r553
telomere (TTTAGGG-primed sequence), middle-repetitive, dispersed interstitial --66:96
TFIID, ZM-1; ZM-2, cDNA, RM, transcription factor (TATA sequence binding) --66:44
tnpA; tnpD, excision proteins --r88
Tub4, Tub5, beta2; beta10, cDNA; genomic, RM, BS: X52878; X52879, beta tubulin family --r273
ZEAR270 ZEAR266 ZEAR260 ZEAR237 ZEAR231, genomic, BS, *Zea* elements; repetitive family from *Z. diploperennis* --r502
zein-19kD A20 & A30 subfamilies, ZG14;ZG35; A20, BS --r254
ZMH1; ZMH2, cDNA, BS, homeobox --r634

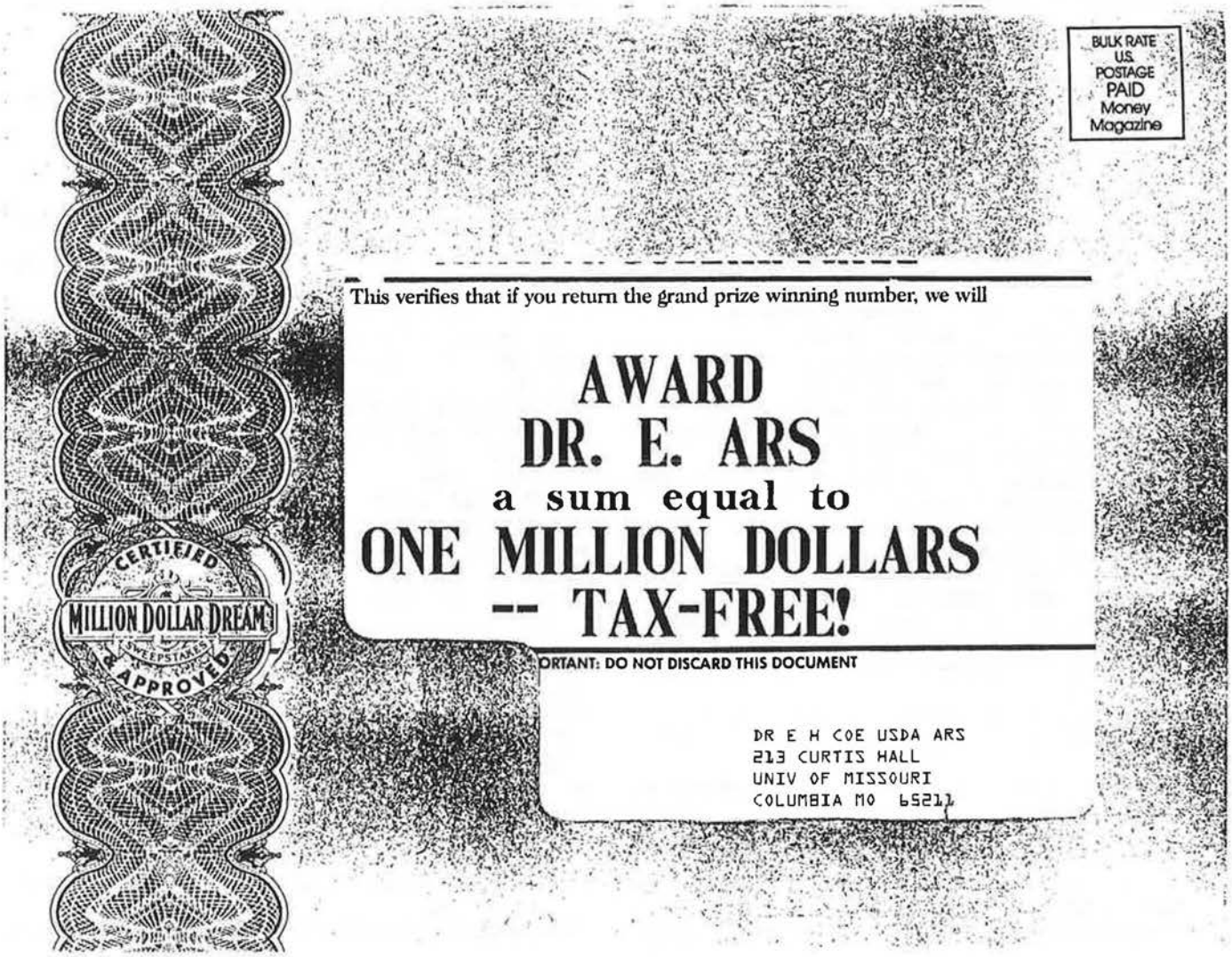
CHLOROPLAST
cp-ORF170, BS: X58080 --r304
cp-tRNAileGAU; cp-tRNAalaUGC; cp-tRNAglyUCC, RM, BS --r133
cp-rpl33; cp-rps18, RM, BS: X56673 --r652
MY503; MY504; MY505; MY518; MY526, YACs --r232
S11, BS: X55967, ribosomal protein 40S --r345

MITOCHONDRIA
120kb chromosome contains *mt-rrn26, mt-rrn18, mt-rrn5, mt-cox1, mt-cox3* --r351
mt-ORF2, DNA-dependent protein kinase? --66:47
*mt-orf221*cms-T, cDNA, BS: X60238; X60239 --r647
NCS3, RM, BS --r272
mt-repeat-2 ("1 kb repeat"), BS: X56904; X56905 --r285
TCM, teosinte-cytoplasm-associated-miniature; restored by *Rcm1* or *Rcm2* --r439

OTHER INHERITANCE (please see note at the beginning of this section)

--Assembled unrestricted by Prof. Ligate

Envelope of the Year:
(What is that period doing in there?)



IV. MAIZE GENETICS COOPERATION STOCK CENTER

During calendar 1991, 2985 seed samples were provided in response to 229 requests. As part of these totals, 535 samples were supplied in response to 44 requests from 21 foreign countries.

There were periods of severe drought during the summer that, in spite of irrigation, prevented adequate seed increase of numerous genetic stocks that lack vigor or ability to withstand stress conditions. Significant portions of all plantings will need to be repeated.

An important component of the summer's plantings consisted of selected stocks that are in low supply. In addition, there were plantings of newly submitted symbolized stocks and of newly acquired untested stocks. There were specific efforts to increase and improve stocks of chromosomes 2, 3, 7 and 8. There was a special increase of goldens, pale greens and yellow-stripes.

There were greenhouse sandbench plantings to determine or confirm genotypes relative to seedling traits. Observation field plantings were also grown to derive similar information relative to mature plant traits.

Catalog items change from year to year, so requests should be based on the latest listing. In making requests, you should indicate both the code number and the genotype of each stock. This information allows us to recognize typographical errors in some cases, or to seek verification of intent when these two types of information are in conflict.

It is sometimes necessary to discontinue supplying samples of particular listed items because of insufficient seed supply or because of detected pedigree errors. In these cases, we attempt to substitute stocks with closely similar genotypes.

We wish to re-emphasize that if you submit genetic stocks to our collection that involve traits that require special techniques, facilities or skills for classification (e.g., isozyme variants), the stocks should be homozygous for the designated alleles. In that way, the samples may be propagated by selfing, sibbing or intercrossing without the necessity for classifying segregating progenies. We hope that greater diagnostic capabilities may become available to us in the future.

We expect that during the next several months a Research Geneticist will be employed by USDA-ARS who will, as part of assigned responsibilities, serve as the future Director of the Maize Genetics Stock Center. This individual has not yet been selected. In the interim, it will be helpful in identifying and channeling inquiries relating to the Stock Center if inquiries are directed in the following way:

Director, Maize Genetics Stock Center
S-123 Turner Hall
Agronomy Department
University of Illinois
1102 S. Goodwin Avenue
Urbana, IL 61801
Phone: (217) 333-6631
FAX: 217-333-9817

E. B. Patterson

CATALOG OF STOCKS

CHROMOSOME 1

101A *sr1 zb4 P1-WW*
101B *sr1 P1-WR*
101C *sr1 P1-WW*
101D *sr1 P1-RR*
101F *sr1 ts2 P1-RR*
102B *sr1 P1-WR an1 bm2*
102C *sr1 P1-RW ad1 bm2*
102D *sr1 P1-RR ad1 bm2*
103C *sr1 P1-WR bm2*
103D *vp5*
103E *zb4 ms17 P1-WW*
103G *sr1 P1-RR bm2*
104B *zb4 ts2 P1-WW bm2*
105A *zb4 P1-WW*
105E *ms17 P1-WR*
105F *ms17 P1-WW*
106A *zb4 P1-WW bm2*
106B *ts2 P1-RR*
106C *ts2 P1-WW bm2*
107A *P1-CR*
107B *P1-RR*
107C *P1-RW*
107D *P1-CW*
107E *P1-MO*
107F *P1-VV*
107G *P1-OR*
107H *P1-WW*
108C *P1-RR br1 f1 an1 gs1 bm2*
109A *P1-RR an1 ad1 bm2*
109B *P1-RR an1 gs1 bm2*
109D *P1-RR ad1 bm2*
109E *P1-WR br1 f1*
110B *P1-WR an1 Kn1*
110C *P1-WR an1 ad1 bm2*
110D *P1-WR an1 bm2*
110E *P1-WR ad1 bm2*
110F *P1-WR br1 Vg1*
110G *P1-WR br1 f1 gs1 bm2*
110K *P1-WR br1*
111A *P1-WW rs2*
111D *P1-WW hm1 br1 f1*
112B *P1-WW br1 f1 bm2*
112E *as1*
112H *P1-WW br1*
113A *as1 br2*
113B *rd1*
113C *br1 f1*
113E *br1 f1 Kn1*
113K *hm1;hm2*
113L *Hm1;hm2*
114B *br1 f1 Kn1 bm2*
114D *Vg1*
114E *br1 Vg1 f1*
114F *hm1 br2*
115B *Vg1 br2 bm2*
115C *v22*
115D *bz2-m; m A1 A2 C1 R1 Pr1*
115E *br2 Vg1*
116A *bz2-m; M A1 A2 C1 R1 Pr1*
116C *an1 bm2*
116D *an1-bz2-6923* (Df)
116I *bz2 gs1 Ts6 bm2*
117A *br2*
117B *br2 bm2*
117D *tb1 (tb*-8963)*
117E *Kn1*
118A *Kn1 Ts6*
118B *Kn1 bm2*
118C *lw1*
119B *vp8*
119C *gs1*
119D *gs1 bm2*
119E *Ts6*
119F *bm2*
120A *id1*
120B *nec2*
120C *ms9*
120D *ms12*
120F *Mpl1*
121A *ms14*
121C *D8*
121D *Lls1*
121J *ms14 br2*
122A TB-1La
122B TB-1Sb
124A *v*-5688*
124B *j*-5828*
124C *w*-8345*
124D *v*-5588*
124E *w*-018-3*
124F *w*-4791*
124G *w*-6577*
124H *w*-8054*
124I *v*-032-3*
124J *v*-8943*
124K *yg*-8574*
125A *Les2*
127A *bz2 zb7 bm2*
127B *dek1*
127C *dek2*
127D *dek22*
127E *f1*
127F *Msc1*
127G *Tlr1*
128A *ij2*
128B *l16*
128C *l17*
128D *pg15*
128E *pg16*
128F *v25*
129A *w18*
129B *wlu5*
130A *o10*

CHROMOSOME 2

201F *ws3 lg1 gl2 b1*
203B *al1 = y3*
205B *lg1*
205C *lg1 gl2*
206A *lg1 gl2 B1*
206B *lg1 gl2 B1 gs2*
208B *lg1 gl2 B1 sk1*
208D *lg1 gl2 B1 v4*
208E *lg1 gl2 b1*
208F *lg1 gl2 b1 gs2*
208H *gl2*
209B *lg1 gl2 b1 gs2 v4*
209E *lg1 gl2 b1 sk1*
209F *lg1 gl2 b1 sk1 fl1*
210A *lg1 gl2 b1 sk1 v4*
211A *lg1 gl2 b1 fl1*
211D *gl2 b1 wt1*
212B *lg1 gl2 b1 fl1 v4*
212D *lg1 gl2 b1 v4*
212E *lg1 gl2 b1 v4 Ch1*
213A *lg1 gl2 mn1 v4*
213B *lg1 gl2 wt1*
213C *lg1 gl2 w3*
213D *lg1 gl2 w3 Ch1*
213E *lg1 gl2 b1 Ch1*
213F *lg1 B1-V Ch1*
213G *lg1 Ch1*
214C *d5*
214D *B1 gl11*

214E *B1 ts1*
214F *gl2 v4 Ch1*
214G *lg1 gs2 v4*
215B *gl11*
215C *wt1*
215E *fl1*
215G *fl1 v4*
216A *fl1 v4 Ch1*
216D *fl1 w3*
216E *fl1 v4 w3*
216F *fl1 w3 Ch1*
217A *ts1*
217B *v4*
217E *w3 Ht1 Ch1*
217H *ba2 v4*
218A *w3*
218C *w3 Ch1*
218D *Ht1* (source *A1* and *B1*)
218E *ba2*
218F *B1 ba2*
219B *b1("r2"); r1-g A1 A2 C1*
219C *Ch1*
220A *Les1*
220B 2 2T T2/*ws3 lg1 gl2*
(T=*Tripsacum*)
221B *B1 gs2*
222A TB-1Sb-2L4464
222B TB-3La-2S6270
223A Primary trisomic 2
224A *w*-4670*
224B *v*-5537*
224F *w*-062-3*
224G *yel*-8630*
224H *whp1; A1 A2 C1 c2 R1*
224J *ij-mos*-7335*
224K *gl-nec*-8495*
227A *dek3*
227B *dek4*
227C *dek16*
227D *dek23*
227E *Les4*
228A *l18*
228B *spt1*
228C *v26*
229A *rl3 Ch1*
229B *v24*

CHROMOSOME 3

301A *cr1*
302A *d1* = *d1-6016* (rosette)
302E *d1-tall*
303A *d1 rt1 Lg3*
303B *d1 Rf1 lg2*
303F *g2= v19= pg14 = g5*
303G *g2 d1*
304A *d1 ys3*
304B *d1 ys3 Rg1*
304G *Lg3 Rg1*
305A *d1 Lg3*
305D *d1 Rg1*
305K *d1 cl1; Clm4*
307C *pm1*
308A *d1 ts4 lg2 a1-m; A2 C1 R1 Dt1*
308B *d1 ts4*
308C *d1 lg2 a1-m; A2 C1 R1 Dt1*
308E *ra2*
308G *d1 ts4 a1-m; A2 C1 R1 Dt1*
309D *ra2 Rg1 lg2*
309E *ra2 pm1 lg2*
310A *ra2 ts4*
310C *ra2 lg2*
310D *Cg1*
310G *ra2 y10*
310I *Cg1 Lg3*
311A *cl1*
311C *cl1; Clm3*
311D *cl1-p; Clm4*
311E *rt1*
311F *ys3*
311G *Lg3 ys3*
312D *Lg3*
313A *gl6*
313C *gl6 Lg3 Rg1*
313E *gl6 Lg3*
314F *gl6 Rg1 lg2*
314G *gl6 lg2*
315B *Rg1 gl6*
315D *A1-b(P415)*
316A *ts4*
318A *ig1*
318B *ba1*
318C *w*-7748 = y10*
319C *lg2 a1-m et1; A2 C1 R1 dt1*
319D *lg2 a1-m et1; A2 C1 R1 Dt1*
320A *lg2*
320D *A1 sh2; A2 C1 R1 B1 Pl1 dt*
320F *A1 sh2; A2 C1 R1 b1 pl1*
320I *A1 sh2; A2 C1 R1*
321A *A1-d31; A2 C1 R1*
322A *A1-d31 sh2; A2 C1 R1 dt*
322B *A1-d31 sh2; A2 C1 R1 Dt1*
322D *a1; A2 C1 R1 B1 Pl1*
322E *a1-m; A2 C1 R1 B1 Pl1 dt1*
322F *a1-m; A2 C1 R1 b1 pl1 dt1*
322G *a1; A2 C1 C2 R1*
323A *a1-m; A2 C1 R1 Dt1*
323B *a1-m; A2 C1 R1 B1 Pl1 Dt1*
323C *a1-m sh2; A2 C1 R1 B1 Pl1 dt1*
324A *a1-st; A2 C1 R1 Dt1*
324E *a1-st et1; A2 C1 R1 Dt1*
324G *a1-st; A2 C1 R1 dt1*
325A *a1-p et1; A2 C1 R1 dt1*
325B *a1-p et1; A2 C1 R1 B1 Pl1 Dt1*
325C *a1-x1*
325D *a1-x3*
325G *a3*
325J *a1-p; A2 C1 R1 Pr1*
326A *sh2*
326B *vp1*
326C *Rp3*
327A TB-3La
327B TB-3Sb
327C TB-3Lc
327D TB-3Ld
328A Primary trisomic 3
329A *v*-9003*
329B *v*-8623*
329C *w*-022-15*
329D *yd2*
329E *w*-8336*
330A *h1*
331A TB-1La-3L5267
331B TB-1La-3L4759-3
331E TB-3Lf
331F TB-3Lg
331H TB-3Li
331I TB-3Lj
331J TB-3Lk
331K TB-3Ll
332B *dek5*
332C *dek24*
332D *Wrk1*
332E *gl9*
332F *gl19*
332G *dek6*
332H *dek17*
332I *Lxm1*
332J *ms23*

CHROMOSOME 4

401A *Rp4*
402A *st1*
402C *st1 fl2*
402D *Ts5*
403A *Ts5 fl2*
404A *Ts5 su1 zb6*
405B *la1*
405D *la1 su1 gl3*
405G *la1 su1 gl4*
406C *fl2*
406D *fl2 su1*
407B *fl2 su1 bm3*
407D *su1*
407E *su1-am*
408B *su1 bm3*
408E *bm3*
408K *su1 se1*
409A *su1 zb6 Tu1*
410D *su1 zb6 gl3*
412C *su1 gl3*
412E *su1 j2 gl3*
413B *su1 gl4*
414A *bt2*
414B *gl4*
414C *gl4 o1*
414E *de** (on Chr. 4 = *de16?*)
415A *j2*
415C *j2 C2; A1 A2 C1 R1*
416A *Tu1*
416B *Tu1-l* 1st
416C *Tu1-l* 2nd
416D *Tu1-d*
416E *Tu1-md*
417A *j2 gl3*
417B *v8*
417C *gl3*
417D *gl3 o1*
418A *gl3 dp1*
418B *c2; A1 A2 C1 R1*
418C *C2; A1 A2 C1 R1*
418D *C2-ldf1* (Active-1); *A1 A2 C1 R1*
418E *dp1*
418F *o1*
418G *v17*
419B *su1 gl3 ra3*
419F *Dt6 gl3; a1-m A2 C1 R1*
420A *Dt4 su1; a1-m A2 C1 R1*
420B TB-9Sb-4L6504
420C *nec*-rd*
420D *yel*-8457*
420I TB-9Sb-4L6222
421A TB-4Sa
421B TB-lLa-4L4692
421C TB-7Lb-4L4698
422A Primary trisomic 4
423A TB-4Lb
423B TB-4Lc
423C TB-4Ld
423D TB-4Le
423E TB-4Lf
427A *dek7*
427B *dek25*
427C *Ysk1*
427D *orp1; orp2*
427E *dek8*
427F *dek10*
427G *Ms41*
427H *dek31*
428A *gl5; gl20*
428B *lw4; lw3*
428C *nec5*
428D *spt2*
428E *wt2*

CHROMOSOME 5

501A *am1 a2; A1 C1 R1*
501B *lu1*
501C *lu1 sh4*
501D *ms13*
501E *gl17*
501H *gl17 a2 bt1; A1 C1 R1*
501I *am1*
502A *gl17 a2 bt1 v2; A1 C1 R1*
502B *A2 vp7 = ps1 pr1; A1 C1 R1*
502D *A2 bm1 pr1; A1 C1 R1*
503A *A2 bm1 pr1 ys1; A1 C1 R1*
503D *A2 bt1 v3 pr1; A1 C1 R1*
504A *A2 bt1 pr1; A1 C1 R1*
504B *A2 bm1 pr1 ys1 v2; A1 C1 R1*
504C *A2 bm1 pr1 zb3; A1 C1 R1*
505B *A2 pr1 ys1; A1 C1 R1*
505C *A2 bt1 ga2 pr1; A1 C1 R1*
505E *A2 v3 pr1 ys1; A1 C1 R1*
506A *A2 v3 pr1; A1 C1 R1*
506B *A2 pr1; A1 C1 R1*
506C *A2 pr1 v2; A1 C1 R1*
506D *A2 na2 pr1; A1 C1 R1*
506F *A2 pr1 v12; A1 C1 R1*
506L *A2 br3 pr1; A1 C1 R1*
507A *a2; A1 C1 R1*
508C *a2 bt1 bv1 pr1; A1 C1 R1*
508F *a2 bm1 pr1 ys1; A1 C1 R1*
510A *a2 bm1 pr1 v2; A1 C1 R1*
510B *A2 bm1 pr1 eg1; A1 C1 R1*
510G *a2 bm1 pr1 eg1; A1 C1 R1*
511A *a2 bt1 v3 pr1; A1 C1 R1*
511C *a2 bt1 pr1; A1 C1 R1*
512A *a2 bt1 v2; A1 C1 R1*
512B *a2 v3 pr1; A1 C1 R1*
512C *a2 bt1 ga2 pr1; A1 C1 R1*
513A *a2 pr1; A1 C1 R1*
513C *a2 pr1 v2; A1 C1 R1*
513E *a2 pr1 v12; A1 C1 R1*
513G *a2; A1 C1 R1*
515A *vp2*
515C *vp7= ps1*
515D *bm1*
516A *bm1 yg1; Ch1*
516B *bt1*
516C *ms5*
516D *td1 ae1*
516G *A2 bm1 pr1 yg1; A1 C1 R1*
516I *td1; Rp1*
517A *v3*
517B *ae1*
518A *sh4*
518B *gl8*
518C *na2*
518D *lw2*
518F *sh4 v2*
518H *gl8 v2*
519A *ys1*
519B *eg1*
519C *v2*
519D *yg1*
519E *A2 pr1 yg1; A1 C1 R1*
519F *A2 pr1 gl8; A1 C1 R1*
520B *v12*
520C *br3*
521A *nec3*
521C *nec*-8624*
521D *nec*-5-9(5614)*
521E *nec*-7476*
521F *nec*-6853*
521G *nec*-7281*
521H *nec*-8376*
521I *v*-6373*
521K *lw3; lw4*
521L *w*-021-7*
522A TB-5La
522B TB-5Lb
523A Primary trisomic 5
527A *dek18*
527B *dek9*
527C *dek26*
527D *dek27*
527E *grt1*
527F *nec7*
527G *pr1 sh5*

CHROMOSOME 6

601D *rgd1 Y1*
601E *po1 = ms6*
601F *po1 y1 pl1*
601G *po1 y1 Pl1*
602A *po1 y1 wi1*
602K *y1-gbl*
603A *y1 l10*
603C *y1 l12*
603D *y1 w15*
604A *y1 pb4 pl1*
604B *y1 pb4 Pl1*
604F *y1 ms1-si*
604H *y1 ms1*
604I *Y1 ms1*
605A *y1 wi1 Pl1*
605F *Y1 wi1 pl1*

606A *Y1 pg11; Wx1 pg12*
606B *y1 pg11; wx1 pg12*
606C *Y1 pg11; wx1 pg12*
606D *y1 pg11; Wx1 pg12*
606E *y1 pl1*
606F *y1 Pl1*
607A *y1 Pl1 Bh1; c1 sh1 wx1 A1 A2 R1*
607B *y1 pl1 Bh1; c1 sh1 wx1 A1 A2 R1*
607C *y1 su2*
607D *y1 pl1 su2*
607F *y1 Pl1 su2*
608G *Y1 l11*
609B *Y1 wi1 pl1*
609C *Y1 wi1 Pl1*
609D *Y1 su2*
610B *Pl1 Dt2; a1-m A2 C1 R1*
610C *pl1 sm1; P1-RR*
610H *Y1 Dt2 pl1; a1-m A2 C1 R1*
611A *Pl1 sm1; P1-RR*
611D *Pt1*
611E *w1*
611H *py1*
612A *w14*
612B *ms6*
612C *l*-4923*
612D *oro1*
613A 2NOR; *a2 bm1 pr1 v2; A1 C1 R1*
613F *whs*-8613*
613L *w*-8954*
613M *yel*-039-13*
613R *wh*-8889*
613T *pg*-6656 (= pg11 pg12)*
613U *wh*-8624*
614A TB-6Lb
614B TB-6Sa
614C TB-6Lc
615A Primary trisomic 6
627A *dek28*
627B *dek19*
627C *vp*-5111*

CHROMOSOME 7

701B *In1-D*
701D *o2*
703A *o2 v5 gl1*
705A *o2 gl1*
705B *o2 gl1 sl1*
705C *o2 lj1*
705D *o2 bd1*
707A *y8 v5 gl1*
707B *in1; A2 pr1 A1 C1 R1*
707D *v5*
707E *vp9*
707F *y8 gl1*
708A *ra1*
708G *y8*
709A *gl1*
710H *ms7 gl1 Tp1*
711B *lj1*
711G *ts*-br*
712A *ms7*
713A *Bn1*
713B *bd1*
714B *o5*
714D *va1*
715A *Dt3; a1-m A2 C1 R1*
715C *gl1 Dt3; a1-m A2 C1 R1*
716A *v*-8647*
716B *yel*-7748*
716F *Les9*
716G *y8*
717A TB-7Lb
718A Primary trisomic 7
727A *dek11*
727B *wlu2*

CHROMOSOME 8

801A *gl18*
801B *v16*
801C *v16 j1*
801D *v16 ms8 j1*
801G *v16 gl18*
803A *ms8*
803B *nec1*
803D *ms8 gl18*
804A *v21*
804D *wh*-053-4*
804E *w*-017-4*
804F *w*-034-16*
804G *w*-8635*
804H *w*-8963*
805A *fl3*
805C *gl18 v21*
805D *fl3 ms8 j1*
805E *el1*
805F *gl18 v16 ms8*
806A TB-8La
806B TB-8Lb
808 *ct1*
809A TB-8Lc
827A *dek20*
827B *dek29*
827C *Bif1*
827D *Sdw1*
827E *Clt1*

CHROMOSOME 9

901D *yg2 C1-I sh1 bz1 wx1; A1 A2 R1*
901E *yg2 C1 bz1 wx1; A1 A2 R1*
902A *yg2 c1 sh1 bz1 wx1; A1 A2 R1*
902B *yg2 c1 sh1 wx1; A1 A2 R1*
902C *yg2 c1 sh1 wx1 gl15; A1 A2 R1*
902D *K-S9 yg2 c1 sh1 wx1 gl15; A1 A2 R1*
902E *yg2 c1 bz1 wx1; A1 A2 R1*
903A *C1 sh1 bz1; A1 A2 R1*
903B *C1 sh1 bz1 wx1; A1 A2 R1*
903D *C1-I sh1 bz1 wx1; A1 A2 R1*
904B *C1 sh1; A1 A2 R1*
904C *C1 sh1 wx1; A1 A2 R1*
904D *C1 wx1 ar1; A1 A2 R1*
905A *K-L9 C1 sh1 wx1; A1 A2 R1*
905B *C1 sh1 ms2; A1 A2 R1*
905C *C1 bz1 Wx1; A1 A2 R1*
905D *K-L9 C1 sh1 wx1; K10 A1 A2 R1*
906A *C1 Ds wx1; A1 A2 R1 Pr1 y1*
906B *C1 Ds wx1; A1 A2 R1 pr1 Y1*
906C *C1-I Ds Wx1; A1 A2 R1*
906D *C1-I; A1 A2 R1*
906G *C1-I Ds Wx1; A1 A2 R1*
907A *C1 wx1; A1 A2 R1*
907D *C1 wx1; A1 A2 R1 B1 pl1*
907E *C1-I wx1; A1 A2 R1 y1*
907G *C1-I(p); A1 A2 R1 B1-b pl1*
907H *C1-I(m); A1 A2 R1 b1 pl1*
908B *C1 wx1 v1; A1 A2 R1*
908D *C1 wx1 gl15; A1 A2 R1*
908E *C1 wx1 gl15; A1 A2 R1 pr1*
908F *C1 wx1 da1; A1 A2 R1*
908H *C1 wx1; y1 A1 A2 R1*
909A *C1 wx1 Bf1; A1 A2 R1*
909B *c1 bz1 wx1; A1 A2 R1*
909C *c1 sh1 bz1 wx1; A1 A2 R1 y1*
909D *c1 sh1 wx1; A1 A2 R1*
909E *c1 sh1 wx1 v1; A1 A2 R1*
909F *c1 sh1 wx1 gl15; A1 A2 R1*
910B *c1 sh1 wx1 gl15 Bf1; A1 A2 R1*
910C *c1 sh1 wx1 bk2; A1 A2 R1*
910D *c1; A1 A2 R1*
910G *sh1-bz1-x2 Wx1; A1 A2 C1 R1*
911A *c1 wx1; A1 A2 R1 y1*
911B *c1 wx1 v1; A1 A2 R1*
911C *c1 wx1 gl15; A1 A2 R1*
911D *c1 wx1 Bf1; A1 A2 R1*
912A *sh1*
912B *sh1 wx1 v1*
913A *sh1 wx1*
913C *sh1 l7*
914A *wx1 d3*
914E *Wx1 pg12; y1 pg11*
914F *wx1 pg12; y1 pg11*
914G *Wx1 pg12; Y1 pg11*
914H *wx1 pg12; Y1 pg11*
915A *wx1* (Other alleles from O. Nelson avail.)
915B *wx1-a*
915C *w11*
916A *wx1 v1*
916C *wx1 bk2*
917A *wx1 Bf1*
917C *v1*
917D *ms2*
917E *gl15*
917F *d3*
918A *gl15 Bf1*
918D *Wc1*
918E *Wx1 bk2 bm4*
918G *Wc1-Wh Bf1 bm4*
918H *Wc1 bm4*
918I *Wx1 bk2*
919A *bm4*
919B *Bf1 bm4*
919C *l6*
919D *l7*
920A *yel*-034-16*
920B *w*-4889*
920C *w*-8889*
920E *w*-8950*
920F *w*-9000*
920G Tp9 N9 N3 Df3
920L *yg-zb*-5588*
920M *w-nl*-034-5*
921A TB-9La
921B TB-9Sb
921C TB-9Lc
921D TB-9Sd
922A Primary trisomic 9
924A *wd1* and Ring9 *C1-I; A1 A2 R1*
927A *dek12*
927B *dek13*
927C *dek30*
927D *Les8*
927E *Zb8*
927F *Dt7; a1-r; A2 C1 R1*
928A *v28*
928B *wlu4*
928C *C1 wx1 Bf1; A1 A2 r1*
930C *wx1 ms2 Bf1; A1 A2 r1*

CHROMOSOME 10

X01A *oy1*
X01B *oy1 R1; A1 A2 C1*
X01E *oy1 bf2 R1; A1 A2 C1*
X02G *oy1 zn1*
X02I *oy1 bf2 ms10*
X02K *oy1 zn1*
X03A *sr3*
X03B *Og1*
X04A *Og1 du1 R1; A1 A2 C1*
X04B *ms11*
X04C *ms11 bf2*
X04D *bf2*
X05A *bf2 zn1*
X05E *bf2 sr2*
X06C *nl1 g1 R1; A1 A2 C1*
X07C *y9*
X07D *nl1*
X09B *li1 g1 R1; A1 A2 C1*
X09F *ms10*
X10A *du1*
X10D *du1 g1 r1; A1 A2 C1*
X10F *zn1*
X10G *du1 v18*
X11A *zn1 g1*
X11F *g1 r1; A1 A2 C1*
X11H *zn1 R1-r; A1 A2 C1*
X12A *g1 r1 sr2*
X12E *g1 R1; A1 A2 C1*
X13D *g1 r1-r sr2; A1 A2 C1*
X13H *r1-g wx1; A1 A2 C1 y1*
X13I *r1-g Wx1; A1 A2 C1 y1*
X14A *lsr1 r1-r; A1 A2 C1*
X14F *v18 r1; A1 A2 C1*
X14G *v18 r1 sr2; A1 A2 C1*
X15C *R1-g; A1 A2 C1*
X15D *r1-ch; Pl1 A1 A2 C1*
X16B *r1 K10; A1 A2 C1*
X16C *R1-ch; A1 A2 C1 B1 pl1*
X16D *r1 sr2; A1 A2 C1*
X17A *r1-g; A1 A2 C1*
X17B *r1-r; A1 A2 C1*
X17C *R1-mb; A1 A2 C1*
X17D *R1-nj; A1 A2 C1*
X17E *R1-r; A1 A2 C1*
X17F *R1-nj* purple embryo Chase; *A1 A2 C1*
X18A *R1-lsk; A1 A2 C1*
X18C *R1-st; A1 A2 C1*
X18D *R1-sk; A1 A2 C1*
X18E *R1-st Mst1*
X18G *R1-scm2; bz2 A1 A2 C1 C2*
X18H *R1-nj; A1 A2 C1 bz2*
X19A *Lc1*
X19C *w2 l1*
X20B *l1*
X20C *v18*
X20F *yel*-8721*
X21A TB-10La
X21B TB-10L19
X21C TB-10Lb
X22A TB-10Sc
X23A Primary trisomic 10
X24A *cm1*
X24B *nec*-4889*
X24C *nec*-5876*
X24D *wh*-7165*
X24E *yel-gr*-8631*
X24F *wh*-8129*
X25A *R1-scm2; a1-st A2 C1 C2*
X25B *R1-scm2; c2 A1 A2 C1*
X25C *R1-scm122; pr1 A1 A2 C1 C2*
X25D *R1-scm2; a2 A1 C1 C2*
X25E *R1-scm2; c1 A1 A2 C2*
X26A *r1-x1; A1 A2 C1*
X27A *dek14*
X27B *dek15*
X27C *dek21*
X27D *Les6*
X27E *gl21*
X27F *Vsr1*
X27G *Oy1-700*
X27H *orp2; orp1*
X27I *l19*

UNPLACED GENES

U140C *l4*
U141A *ms22*
U141B *ms24*
U141C *o9*
U141D *o11*
U142A *o12*
U142B *o13*
U142C *rd3*
U142D *ub1*
U142E *y11*
U142F *y12*
U240A *Les7*

MULTIPLE GENE STOCKS

M141A *A1 A2 C1 C2 R1-g Pr1 B1 Pl1*
M141B *A1 A2 C1 C2 R1-g Pr1 B1 pl1*
M141C *A1 A2 C1 C2 R1-g b1 Pl1*
M141D *A1 A2 C1 C2 R1-g b1 pl1*
M241A *A1 A2 C1 C2 r1-g Pr1 B1 Pl1*
MX17A *A1 A2 C1 C2 r1-g Pr1 b1 pl1*
M241B *A1 A2 C1 C2 r1-g Pr1 B1 pl1*
M340A *A1 A2 c1 C2 R1-g Pr1 B1 pl1*
M241C *A1 A2 C1 C2 R1-r Pr1 B1 Pl1*
M341B *A1 A2 C1 C2 R1-r Pr1 B1 pl1*
M341C *A1 A2 C1 C2 R1-r Pr1 b1 Pl1*
M341F *A1 A2 C1 C2 R1-r Pr1 b1 pl1*
M441A *A1 A2 C1 C2 R1-r Pr1 B1 Pl1 wx1*
M441B *A1 A2 C1 C2 R1-r Pr1 B1 pl1 wx1*
M441F *A1 A2 C1 C2 R1-g Pr1 b1 pl1*

wx1
M541F *A1 A2 C1 C2 R1 Pr1*
M641B *A1 A2 C1 C2 R1 Pr1 wx1*
M641D *A1 A2 C1 C2 r1 Pr1 y1 wx1*
MX41A *A1 A2 C1 C2 R1 pr1 y1 wx1 gl1*
M941A *A1 A2 c1 C2 R1 Pr1 y1 wx1*
M341D *A1 A2 c1 C2 R1-r Pr1 B1 Pl1*
M341E *A1 A2 c1 C2 R1-g Pr1 b1 pl1*
M441D *A1 A2 C1 C2 r1-r Pr1 B1 Pl1*
M441E *A1 A2 c1 C2 r1-r Pr1 B1 Pl1*
MX41B *su1 pr1 y1 gl1 wx1; A1 A2 C1 C2 R1*
M841A *A1 su1 pr1; A2 C1 C2 R1*
MX41C *bz2 a1 c2 a2 pr1 Y1/y1 c1 bz1 wx1 r1*
M841B *a1 su1 A2 C1 C2 R1*
MX40A *bm2 lg1 a1 su1 pr1 y1 gl1 j1 wx1 g1*
M841C colored scutellum; *A1 A2 C1 C2 R1 Pr1*
M841E colored scutellum; *A1 A2 C1 C2 R1 pr1*
MX41D *a1 su1 pr1 y1 gl1 wx1 A2 C1 C2 R1*
M741C Stock 6 : Hi-haploid *R1-r B1 Pl1*
M741F Stock 6: Hi-haploid *A1 C1 R1-g* colored scutellum
M741G Stock 6: Hi-haploid *y1 C1-I wx1 A1 R1-g*

POPCORNS

P142A Amber Pearl
P142B Argentine
P142C Black Beauty
P242A Hulless
P242B Ladyfinger
P242C Ohio Yellow
P342A Red
P342B Strawberry
P342C Supergold
P342D South American
P442B White Rice

EXOTICS AND VARIETIES

E542A Black Mexican Sweet Corn (with B-chromosomes)
E542B Black Mexican Sweet Corn (without B-chromosomes)
E642B Gourdseed
E742A Maiz Chapalote
E742B Papago Flour Corn
E742C Parker's Flint
E842A Tama Flint
E842B Zapalote Chico
E942A Winnebago Flint
E942B Missouri Cob Corn

TETRAPLOID STOCKS
N103A *P1-RR*
N103D *P1-WR*
N104B *pr1; A1 A2 C1 R1*
N107C Synthetic B
N107B W23 conversion
N104C *su1 wx1*
N106D *sh1 Wx1; Y1*
N106E *sh1 wx1; y1*

CYTOPLASMIC TRAITS

C337A NCS2
C337B NCS3

CYTOPLASMIC STERILES AND RESTORERS

C836A WF9-(T) *rf1 rf2*
C836B WF9 *rf1 rf2*
C736A R213 *Rf1 rf2*
C736C B37 *rf1 Rf2*
C736D N6 *rf1 Rf2*
C736B Ky21 *Rf1 Rf2*

WAXY RECIPROCAL TRANSLOCATIONS*

wx01A *wx1* T1-9c (1S.48; 9L.22)
wx01B *wx1* T1-9(5622) (1L.10; 9L.12)
wx03A *wx1* T1-9(8389) (1L.74; 9L.13)
wx04A *wx1* T2-9c (2S.49; 9S.33)
wx05A *wx1* T2-9b (2S.18; 9L.22)
wx06A *wx1* T2-9d (2L.83; 9L.27)
wx07A *wx1* T3-9(8447) (3S.44; 9L.14)
wx08A *wx1* T3-9c (3L.09; 9L.12)
wx10A *wx1* T4-9e (4S.53; 9L.26)
wx11A *wx1* T4-9g (4S.27; 9L.27)
wx12A *wx1* T4-9(5657) (4L.33; 9S.25)
wx13A *wx1* T4-9b (4L.90; 9L.29)
wx15A *wx1* T5-9(4817) (5L.06; 9S.07)
wx16A *wx1* T5-9d (5L.14; 9L.10)
wx17A *wx1* T5-9a (5L.69; 9S.17)
wx18A *wx1* T6-9(4778) (6S.80; 9L.30)
wx20A *wx1 y1* T6-9b (6L.10; 9S.37)
wx21A *wx1* T6-9(4505) (6L.13; 9ctr.)
wx22A *wx1* T7-9(4363) (7ctr.; 9ctr.)
wx23A *wx1* T7-9a (7L.63; 9S.07)
wx24A *wx1* T8-9d (8L.09; 9S.16)
wx25A *wx1* T8-9(6673) (8L.35; 9S.31)
wx26A *wx1* T9-10(8630) (9S.28; 10L.37)
wx27A *wx1* T9-10b (9S.13; 10S.40)
wx28A *wx1* T5-9(8386) (5L.87; 9S.13)

NON-WAXY RECIPROCAL TRANSLOCATIONS*

Wx30A *Wx1* T1-9c (1S.48; 9L.22)
Wx30B *Wx1* T1-9(4995) (1L.19; 9S.20)
Wx30C *Wx1* T1-9(8389) (1L.74; 9L.13)
Wx31A *Wx1* T2-9c (2S.49; 9S.33)
Wx31B *Wx1* T2-9b (2S.18; 9L.22)
Wx32A *Wx1* T3-9(8447) (3S.44; 9L.14)
Wx32B *Wx1* T3-9(8562) (3L.65; 9L.22)
Wx32C *Wx1* T3-9c (3L.09; 9L.12)
Wx33A *Wx1* T4-9e (4S.53; 9L.26)
Wx33B *Wx1* T4-9(5657) (4L.33; 9S.25)
Wx33C *Wx1* T4-9g (4S.27; 9L.27)
Wx34A *Wx1* T5-9c (5S.07; 9L.10)
Wx34B *Wx1* T5-9(4817) (5L.06; 9S.07)
Wx34C *Wx1* T4-9b (4L.90; 9L.29)
Wx35A *Wx1* T5-9(8386) (5L.87; 9S.13)
Wx35B *Wx1* T5-9a (5L.69; 9S.17)
Wx35C *Wx1* T5-9d (5L.14; 9L.10)
Wx36A *Wx1* T6-9(4778) (6S.80; 9L.30)
Wx37A *Wx1* T6-9(8768) (6L.89; 9S.61)
Wx37B *Wx1* T7-9(4363) (7ctr.; 9ctr.)
Wx37C *Wx1* T6-9(4505) (6L.13; 9ctr.)
Wx38A *Wx1* T7-9a (7L.63; 9S.07)
Wx38B *Wx1* T8-9d (8L.09; 9S.16)
Wx38C *Wx1* T8-9(6673) (8L.35; 9S.31)
Wx39A *Wx1* T9-10(8630) (9S.28; 10L.37)
Wx39B *Wx1* T9-10b (9S.13; 10S.40)

* = Single cross of homozygotes between M14 and W23 versions will be supplied if available

INVERSIONS

I143B Inv1c (1S.35-L.01)
I143C Inv1d (1L.55-L.92)
I143D Inv1(5131-10) (1L.46-L.82)
I444A Inv2a (2S.70-L.80)
I243A Inv2(8865) (2S.06-L.05)
I243B Inv2(5392-4) (2L.13-L.51)
I343A Inv3a (3L.38-L.95)
I343B Inv3L (3L.19-L.72)
I343C Inv3(3716) (3L.09-L.81)
I443A Inv4b (4L.40-L.96)
I443B Inv4c (4S.86-L.62)
I543A Inv4e (4L.16-L.81)
I743A Inv5(8623) (5S.67-L.69)
I743B Inv6(8452) (6S.77-L.33)
I843A Inv6(8604) (6S.85-L.32)
I743C Inv6(3712) (6S.76-L.63)
I943A Inv7(5803) (7L.17-L.61)
I943B Inv7(8540) (7L.12-L.92)
I943C Inv7(3717) (7S.32-L.30)
IX43A Inv8a (8S.38-S.15)
I344A Inv9a (9S.70-L.90)
IX43B Inv9b (9S.05-L.87)

Cooperators (that means you) need the Stock Center.

The Stock Center needs Cooperators (this means you) to:

(1) Send stocks of new factors that you have reported in this News Letter or in publications, and stocks of new combinations, to the collection.

(2) Inform the Stock Center on your experience with materials received from the collection.

(3) Acknowledge the source of the stocks for research when you publish, and advice or help you have received in development of your research project.

FRANK AND ERNEST **By Bob Thaves**

V. GENE LIST AND WORKING MAPS

GENELIST: The accompanying table lists the defined and designated gene loci of maize. The table includes the symbol for the locus, the chromosome (L=long arm, S=short arm) and map location, name and a brief phenotypic description, availability from the Stock Center (S), photograph (P) in Mutants of Maize (Neuffer et al. 1968), and references to original descriptions. Stocks may be obtained from the Maize Genetics Stock Center (see the preceding section); other variants (e.g., isozymes and RFLPs) exist inherently among generally available strains.

For a table of mapped RFLP loci, see MNL 65:145-153.

Three enhancements of the resources for maize genetics research are currently under development: The Maize Handbook (Freeling and Walbot, eds., Springer-Verlag, fall 1992); Mutants of Maize (Neuffer, Coe, and Wessler, Cold Spring Harbor Lab., 1993); and the Maize Database, Maizedb (Plant Genome Initiative, 1993 projected on-line date; please see the following section).

NOMENCLATURE: New definitions of standards and criteria are in preparation and will be distributed as soon as they are completed.

MAPS: Our working maps follow the table. The traditional linkage map, based on recombinational analyses of Mendelizing variations in an expression or a gene product, is in the center. Each map represents the order and distances in centimorgans (1% recombination = 1 cM), for loci for which sufficient information is available to make a reasonable judgment of location. Each chromosome begins at the top with the most distal locus known in the short arm. The physical (cytological) map of each chromosome, immediately to the left of each linkage map, is drawn with the length of each arm in proportion. Locations of B-A translocations, which generate hemizygous segments, are shown as TB-..., and A-A translocations as T with chromosome numbers and identifiers (see MNL 55:140ff.); placement on the physical map is in accordance with observed breakpoints; placement on the linkage map is in relation to cytogenetic mapping data (see MNL 52:129ff., 59:159ff., 60:149ff.). Locations of the centromeres are indicated according to the best available data from cytogenetic studies. The vertical line associated with simple B-A translocations represents the segment within which the breakpoint is located (genes distal to the line on that arm should be uncovered). In the case of compound translocations, the associated vertical line on the linkage map for the first arm involved (e.g., 1L of TB-1La-5S8041) defines the segment within which the second breakpoint is located (genes distal to the line are not uncovered). On the map of the second arm involved (5S in the example), genes distal to the associated line are uncovered (as they are with simple B-A translocations). TB's shown spanning one or more genes may or may not uncover the indicated gene or genes. To the right of the linkage map are shown genes (alphabetically in groups) for which a "rough" placement has been defined, either near a gene already on the map or to a region of the map. Furthest to the right are shown genes placed only to chromosome (vertical line with arrows) or to one arm (vertical line from near the centromere to the end of the arm).

To the left of each physical map is a map showing RFLP and isozyme loci, representing information and data derived at Missouri and at Brookhaven supplemented with information provided by Pioneer, Agrigenetics, and others. This is a strictly approximate representation, designed to aid searching and comparisons; it is NOT a mutually interdigitated map. The several available RFLP maps, most of which are internally consistent, are largely but not completely consistent with each other. Construction of an integrated map requires systematic compilations of data, but more importantly the development of complex mapping engines that are not yet available in any form. Enhanced mapping engines are a target of some labs, including the Maizedb program, and may become available to apply to our data during the coming year.

Dashed lines tying the RFLP map to the linkage maps show approximate locations of gene loci. Positions shown are mutually interdependent (i.e., locations indicated are derived from the information from each source by circular logic).

The current Plastid Chromosome Genetic Map, prepared by Steve Rodermel, follows the nuclear working maps. For the Mitochondrial Map, see MNL 64:165.

MAP IT: The importance of placing loci defined by probes of known function cannot be overstressed. In a number of cases these give very accurate ties to the conventional map and, in the very least, provide functional significance to a particular region of the genome that will be important as further additional studies (particularly in the area of quantitative genetics) progress. Therefore, if you have a clone for a known function and know or believe that it hybridizes to a maize genomic sequence, please attempt to map the locus (or loci). This can be accomplished in a couple of ways (and we recommend doing both). First, the set of recombinant inbreds should be probed and the data sent to Ben Burr for analysis. Second, it would be appreciated if the probe could be sent to Missouri for mapping in the Immortal F2 population. We would also use the probe in correlation to physical and conventional markers. We have included in this Newsletter a sample form of the desired information for each clone you provide. If you have any questions regarding mapping of RFLP loci (both old and new), please call or write.

The quality of these resources is enhanced each year by corrections, clarifications, and suggestions provided by Cooperators; your input is welcome and needed.

Ed Coe

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
a1	3L-149.0	anthocyaninless: colorless aleurone, green or brown plant, brown pericarp with *P1-RR*; for alleles and interactions, see Coe et al., 1988; dihydroflavonol reductase; *BNL(A1)*(pAmu2), *NPI51(A1)*(), *NPI467(A1)*()	S	P	99
A1(2)	1L-near *as1*	see *NPI482(A1)*()			445
A1(3)	2L-near *w3*	see *NPI468(A1)*()			445
A1(4)	5L-near *Got2*	see *NPI469(A1)*()			445
A1(5)	7S- near *v5*	see *NPI470(A1)*()			445
A1(6)	8L-near *Idh1*	see *UMC189(A1)*()			129
a2	5S-35	anthocyaninless: like *a1*, but red pericarp with *P1-RR*; flavanone-3-hydroxylase or a dehydrase/oxidase	S	P	180
a3	3L-132	anthocyanin: recessive intensifier of expression of *R1* and *B1* in plant tissues	S	P	228
Abp1	3L-near *Rg1*	auxin receptor binding protein candidate (was *axr1*): see *BNL(ABP)*(cDNA probeG100)			422
Ac		activator: designator for autonomous transposable elements; regulates *Ds* transposition and dissociation; ex. *Ac9* designates element isolated from *wx1-m9*		P	253
Ac2		activator: similar to *Ac*			78

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
Acc1	?	acetyl-coenzyme A carboxylase (EC6.4.1.2): tissue-culture selected resistance to cyclohexanedione (e.g., sethoxydim) and aryloxy phenoxypropionate (e.g., haloxyfop) herbicides; ACCase altered			316
Aco1	4S-near *la1*	aconitase: electrophoretic mobility; monomeric			435
Aco2	?	aconitase: electrophoretic mobility			435
Aco3	?	aconitase: electrophoretic mobility			435
Aco4	?	aconitase: electrophoretic mobility; monomeric			435
Acp1	9L-near *wx1*	acid phosphatase (was *Ap1*, *Acph1*, *Phos*): electrophoretic mobility; cytosolic; dimeric			93 148
Acp2	?	acid phosphatase (was *Ap2*): electrophoretic mobility; dimeric			93 148 193
Acp4	1L-176	acid phosphatase: electrophoretic mobility; monomeric			193
Act1	8-near *Mdh4*	actin family: see *BNL(ACT)*(pMAcI), *NPI368(ACT)*()			44 375
ad1	1L-108	adherent: seedling leaves, tassel branches, and occasionally top leaves adhere	S	P	197
Adh1	1L-128	alcohol dehydrogenase: electrophoretic mobility; null allele is known; dimeric; intra/interlocus hybrid bands occur; *BNL(ADH1)*(pH2.3), *NPI21(ADH)*()			374
Adh2	4S-27	alcohol dehydrogenase: electrophoretic mobility; null allele is known; dimeric; intra/interlocus hybrid bands occur; *BNL(ADH2)*(pZML841), *NPI228(ADH2)*()			372
Adk1	6S-0	adenylate kinase: electrophoretic mobility; plastidial			436
Adp1	1L-near *f1*	ADP glucose pyrophosphorylase candidate: see *BNL17.15(BT2)*(), *NPI309(ADP)*()			445
Adp2		see *Agp2*			
Adp3		see *Bt2*			
Adp4	8L- near *pro1*	ADP glucose pyrophosphorylase candidate: see *BNL17.16(BT2)*			445
Adp5	10-near *oy1*	ADP glucose pyrophosphorylase candidate: see *NPI319(ADP*			445
Adr1	?	alcohol dehydrogenase regulator			210
ae1	5L-57	amylose extender: glassy, tarnished endosperm; high amylose content; endosperm-specific starch branching enzyme IIb	S	P	431
afd1	?	absence of first division: male and female sterility; anaphase I equatorial			144
Agp1	6L-near *w14*	ADP glucose pyrophosphorylase: embryo-specific; *UFG(AGP1)*(cDNA probe pBS.ABP)			45
Agp2	2L-near *v4*	ADP glucose pyrophosphorylase: embryo-specific; *UFG(AGP2)*(), *NPI310(ADP)*(cDNA probe pBS.ABP)			45
AGR		Agrigenetics: designator for loci defined by restriction fragment polymorphisms			
agt1	?	agravitropic: primary root unresponsive to gravity			88
al1	2S-4	albescent plant: variably cross-banded to white leaves; pale yellow endosperm, some alleles viviparous (see Coe et al., 1988)	S	P	325
Alb1	8-near *pro1*	albumin: abundant 32kD endosperm protein (b-32), cytosolic; ribosome-inactivating			241
Ald1	3-near *vp1*	aldolase: anaerobiosis-induced, cytoplasmic, fructose-1,6-diphosphate aldolase, EC4.1.2.13			152 195
alh1	1L-near *bm2*	histone la (was H1a): electrophoretic mobility			414
Alpha		*A1* locus component (see *Beta*): determines reduced aleurone and plant color, brown pericarp			216
Alr1	2L- near *Tpi2*	aleurain homolog: see *NPI348(ALR)*()			446
Alr2	7L	aleurain homolog: see *NPI349(ALR)*()			446
Als1	4L-near *orp1*	acetolactate pyruvate lyase candidate: see *BNL(ALS1)*()			45
Als2	5L-near *bv1*	acetolactate pyruvate lyase candidate: see *BNL(ALS2)*()			45
am1	5S-20	ameiotic: male and female sterility; anaphase I equatorial	S	P	314 341
Amp1	1L-near *Mdh4*	aminopeptidase: electrophoretic mobility; cytosolic; monomeric			312
Amp2	1-near *hm1*	aminopeptidase: electrophoretic mobility; monomeric			312
Amp3	5S-near *a2*	aminopeptidase: electrophoretic mobility; monomeric			312
Amp4	?	aminopeptidase: electrophoretic mobility; monomeric			312
Amy1	?	alpha amylase: electrophoretic mobility; monomeric			52
Amy2	5S-near *Mdh5*	beta amylase: electrophoretic mobility; monomeric			51
an1	1L-104	anther ear: andromonoecious dwarf, intermediate stature; few tassel branches; responds to gibberellins; *an1-6923* includes deletion of *Bz2*	S	P	96 106
anl1	5S-near *lu1*	anthocyaninless lethal: colorless aleurone; small kernels; embryo lethal			59
Ant1	5L	adenine nucleotide translocator candidate: see *UMC142(ANT)*(pZmc-ATP-01)			12
Ant2	?	adenine nucleotide translocator candidate (cDNA probe)			13
aph1	?	aphid resistance			50
ar1	9L-62	argentia: virescent seedling, greens rapidly; husk leaf tips striped	S	P	110
ARS		designator for autonomously replicating sequences			
as1	1-56	asynaptic: synaptic failure in male and female	S	P	20
Ask1	-near *o2*	aspartate kinase (was LT19, *Ltr*-1*, Lt1a): lysine-threonine resistance in cultures and seedlings, increased threonine in kernels			79
Ask2	?	aspartate kinase (was LT20): lysine-threonine resistance			79

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
Asr1	4S-0	absence of seminal roots			268
Atc1		see *Zb8*			
atn1	?	anaerobic tolerant null: enhances survival of ADH-null under anoxia			217
Atp2	?	ATPase candidate: F1 ATPase subunit 2 (cDNA probe)			92
ats1	8	atrazine susceptible: lacks glutathione S-transferase			151
B chr		B chromosome: supernumerary chromosome	S	P	335
b-32		see *Alb1*			
B1	2S-49	colored plant: anthocyanin in major plant tissues; some alleles affect aleurone and embryo color (for alleles, see Coe et al., 1988); regulates flavonoid enzymes; *NPI(B1)*()	S	P	102
ba1	3L-102	barren stalk: ear shoots and most tassel branches and spikelets absent	S	P	163
ba2	2-near *ts1*	barren stalk: like *ba1*, but tassel more normal	S		163
ba3	?	barren stalk			315
baf1	9S-near *w11*	barren stalk fastigiate (was *ba*-s*): ear shoots often absent; tassel branches erect			60
bd1	7L-109	branched silkless: ear silkless, branched at base; tassel proliferated, bushy	S	P	200
beta		*A1* locus component (see *alpha*): determines aleurone and plant color, red pericarp			216
Bf1	9L-137	blue fluorescent: homozygous seedlings (homozygous or heterozygous anthers) fluoresce blue under ultraviolet; anthranilic acid present	S	P	421
bf2	10L-33	blue fluorescent: similar to *Bf1* in expression; shows earlier, stronger seedling fluorescence	S		5
Bg		Bergamo: regulatory element mediating *o2-mr*			357
Bh1		blotched aleurone, colored patches on colorless (*c1*) background; allele at *Pl1*, which see	S	P	101
Bif1	8-near *Rf4*	barren inflorescence (was *Bif*-1440*): ear and tassel have many fewer spikelets, bare rachis appendages	S		300
bk2	9L-82	brittle stalk: brittle plant parts after 4-leaf stage	S	P	213
Blh1	1S	bleached (was Bleached-1593): pale green midveins and base in upper leaves			296
bm1	5S-41	brown midrib: brown pigment over vascular bundles of leaf sheath, midrib, and blade	S	P	109 190
bm2	1L-161	brown midrib: like *bm1*	S		43
bm3	4-near *bt2*	brown midrib: like *bm1* (C.R. Burnham, 1935, unpublished data)	S		107 207
bm4	9L-141	brown midrib: like *bm1*	S		40
Bn1	7L-71	brown aleurone: yellowish brown aleurone color	S		209
BNL		Brookhaven National Laboratory: designator for loci defined by restriction fragment polymorphisms			
br1	1L-81	brachytic: short internodes, short plant; no response to gibberellins	S	P	196 199
br2	1L-near *hm1*	brachytic: like *br1*	S		219
br3	5	brachytic: like *br1*	S		388
brn1	3S-19	brown aleurone: brown kernel, brown embryo; seedling lethal			353
Bs-1		barley stripe: transposable element, retrovirus-like; 1-5 copies in genome			185
bs1	?	barren sterile			259
bt1	5L-42	brittle endosperm: mature kernel collapsed, angular, often translucent and brittle; affects starch-granule-bound phospho-oligosaccharide synthase; *BNL(BT1)*(cDNA)	S	P	239 439
bt2	4S-48	brittle endosperm: like *bt1*; endosperm ADP glucose pyrophosphorylase subunit; (compare *sh2*) (G.F. Sprague, 1935, unpublished data); *BNL(BT2)*(cDNA), *NPI314(ADP)*	S		107 420
btn1	?	brittle node			194
bu1	?	leaf burn: leaves show burning, sometimes horizontal bands, accentuated by high temperature			128
bv1	5L-47	brevis plant: short internodes, short plant	S		221
bv2	?	brevis plant: plant height 30-50% of normal			326
bx1	4S	benzoxazinless: absence of cyclic hydroxamates (blue color in crushed root tip with FeCl3), which inhibit *Ostrinia nubilalis* and *Helminthosporium turcicum*			70 155
bz1	9S-31	bronze: modifies purple aleurone and plant color to pale or reddish brown; anthers yellow-fluorescent; UDPG-flavonol 3-O-glucosyl transferase; allele *bz1-m4* = *sh1-bz1-m4*; *AGR(BZ1)*(), *BNL(BZ1)*(pMBzPA), *NPI8(BZ1)*(), *UMC192(BZ1)*(pbz)	S	P	340
bz2	1L-106	bronze: like *bz1*; anthers not fluorescent; *an1-6923* mutation includes deletion for *Bz2*; potential function flavonoid acylation, glycosylation, transport, or deposition; *BNL(BZ2)*(pP300), *UMC181(BZ2)*(pP300)	S	P	310
C-bands		brightly stained regions (bands) on chromosomes with Giemsa stain; correspondence to knobs (see K)			1 433
C1	9S-26	colored aleurone: *c1* colorless; *C1-I* dominant colorless; *c1-p* pigment inducible by light (see Coe et al., 1988); regulates flavonoid enzymes; *BNL(C1)*(pEco1.0)	S	P	90
c2	4L-98	colorless: colorless aleurone, reduced plant color; chalcone synthase; *C2-Idf* dominant inhibitor (see Coe et al., 1988); duplicate factor with *whp1* for pollen color and for anthocyanins; *BNL(C2)*(pC2-c46)	S	P	30
Cab1	3L-near *et1*	chlorophyll a/b binding protein candidate: see *NPI477(CAB)*(); *UMC24(CAB)*()			445
Cab2	7L-near *st1*	chlorophyll a/b binding protein candidate: see *NPI478(CAB)*()			445
Cab3	7L-near *st1*	chlorophyll a/b binding protein candidate: see *NPI478(CAB)*()			445
Cab4	8L-near *ms8*	chlorophyll a/b binding protein candidate: see *NPI479(CAB)*(); *UMC174(CABM2)*(8-22)			445

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
Car1	1S	catalase regulator: enzyme activity level increased			363
Cat1	5S-near *Mdh5*	catalase: electrophoretic mobility; cytosolic/glyoxysomal; tetrameric; intra/interlocus hybrid bands occur			23
Cat2	1S	catalase: electrophoretic mobility; null allele is known; cytosolic/glyoxysomal; tetrameric; intra/interlocus hybrid bands occur			360
Cat3	4L-near *dp1*	catalase: electrophoretic mobility; null allele is known; mitochondrial; tetrameric; intralocus hybrid bands occur			362
Cdh1	?	cinnamyl alcohol dehydrogenase: electrophoretic mobility			119
Ce1	?	curled entangled: rolled leaves tend to be entangled; resembles *Rld1*			53
cfl2	?	complementary to *fl2*			313
cfr1	1S	coupling factor reduction: chloroplast ATP synthase affected, seedlings pale green and greatly reduced in vigor			91
Cg1	3S-35	corngrass: narrow leaves, extreme tillering	S	P	387
Cg2	?	corngrass: like *Cg1*; mutable			233
Cgl1	?	*Colletotrichum graminicola* resistance			11
Ch1	2L-155	chocolate pericarp: dark brown pericarp	S	P	7
Cin		Cinteotl corn insert: repetitive sequences dispersed in the genome			378
cl1	3S-60	chlorophyll: white to green seedlings, depending upon *Clm1*; pale yellow endosperm	S		108
clh1	?	histone lc: electrophoretic mobility			414
Clm1	8	modifier of *cl1*: greens *cl1* seedlings; does not restore endosperm carotenoids	S		108
Clt1	8	clumped tassel (was *Clt*-985*): variable dwarfing, developmental anomalies	S		132 298
cm1	10L-near *R1*	chloroplast modifier: white or yellow stripes on leaves (compare *ij1*); conditions chloroplast modifications that are maternally inherited	S		416
cms-C		cytoplasmic male sterility: female-transmitted male sterility, C type; restored by *Rf4*			21
cms-S		cytoplasmic male sterility: female-transmitted male sterility, S type; restored by *Rf3*			188 191
cms-T		cytoplasmic male sterility: female-transmitted male sterility, Texas type; restored by *Rf1 Rf2*			188 191
cp1	7S-near *vp9*	collapsed: endosperm collapsed and partially defective			231
cp2	7S-near *vp9*	collapsed: endosperm rough, collapsed, partially defective; seedling very light green with darker streaks; lethal			305
cpDNA		chloroplast DNA (= ctDNA): sequences or loci in chloroplast genome			
cr1	3S-26	crinkly leaves: plant short; leaves broad, crinkled, foreshortened	S	P	103
cr4	10-near *Rp5*	crinkly leaf (was *dap*-6143*): crinkly-leaved seedlings; plants short with rough, extremely crinkly leaves and club tassel; aleurone mosaic			411
CSH		Cold Spring Harbor: designator for loci defined by restriction fragment polymorphisms			
Css1	9L-near *gl15*	sucrose synthase (= *Sus2*, SS2): sucrose synthase-2 of embryo and other tissues; (compare *sh1*); *BNL(CSS)*(pshD13), *NPI121(CSS)*(), *UMC190(CSS)*()			250
ct1	8	compact plant: semi-dwarf plant, ear furcated	S		285
ct2	1S	compact plant: semi-dwarf plant with club tassel			135
ctDNA		chloroplast DNA (= cpDNA): sequences or loci in chloroplast genome			
cto1	?	cob turned out: ear inverted to a sheet or tube, kernels internally placed; variable expression			428
Cx1	10L-near bf2	catechol oxidase: electrophoretic mobility; null allele is known; monomeric; no hybrid bands			333
Cy	5L-near *pr1*	Cycler: regulatory element mediating *bz1-rcy*			366
d1	3S-44	dwarf plant: andromonoecious, short, compact plants; responds to gibberellins; *d1-t* intermediate in height	S	P	96
d2	3	dwarf plant: like *d1*	S		418
d3	9S-59	dwarf plant: like *d1*	S		75
d5	2S-34	dwarf plant: like *d1*	S		418
D8	1L-133	dwarf plant: dominant, resembles *d1*; not responsive to gibberellins; (compare *Mpl1*, possible allele)	S	P	323
D9	5S-6	dwarf plant (was *D*-2319*): dominant semidwarf with broad, dark green leaves; not andromonoecious, not responsive to gibberellins			291 292
da1	9	dilute aleurone: aleurone color diluted	S		114
Dap1	5L-near *Got2*	dappled aleurone: patches of normal and abnormal aleurone cells			413
db1	?	dichotomously branching plants (= dib): variable location of dichotomy, usually at 4-8th node (possible association with aneuploidy)			258 259
dek1	1S-27	defective kernel (was *clf, gay, clf*-792*): germless; floury endosperm; anthocyanins and carotenoids absent; cultured embryos not obtained	S		303 304
dek2	1L	defective kernel (was *dsc*-1315A*): discolored, scarred endosperm; lethal; cultured embryos green	S		303 304
dek3	2S	defective kernel (was *gm*-1289*): germless; cultured embryos white with green stripe	S		303 304
dek4	2L	defective kernel (was *clf*-1024A*): germless; floury endosperm; cultured embryos green, narrow leaved	S		303 304
dek5	3S	defective kernel (was *sh*-874A*): shrunken endosperm; white seedling with green stripes	S		303 304
dek6	3L	defective kernel (was *sh*-627D*): shrunken endosperm; lethal; cultured embryos normal	S		303 304

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
dek7	4S-near *Ts5*	defective kernel (was *su*-211C*): shrunken sugary endosperm; white seedling with green stripes	S		303 304
dek8	4L	defective kernel (was *sh*-1156A*): shrunken endosperm; lethal; cultured embryos green, small	S		303 304
dek9	5L	defective kernel (was *crp*-1365*): crumpled endosperm; lethal; anthocyanins and carotenoids reduced; cultured embryos not obtained	S		303 304
dek10	4L	defective kernel (was *cp*-1176A*): collapsed endosperm; lethal; cultured embryos green, curled, stubby	S		303 304
dek11	7L	defective kernel (was *et*-788*): etched endosperm; lethal; cultured embryos white with green stripes	S		303 304
dek12	9S	defective kernel (was *cp*-873*): collapsed endosperm; lethal; cultured embryos green, narrow-leaved, curled	S		303 304
dek13	9L	defective kernel (was *o*-744*): defective opaque endosperm; lethal; cultured embryos pale green with green stripes	S		303 304
dek14	10S	defective kernel (was *cp*-1435*): collapsed endosperm; lethal; cultured embryos yellow-green	S		303 304
dek15	10L	defective kernel (was *cp*-1427A*): collapsed floury endosperm; lethal; cultured embryos green	S		303 304
dek16	2L	defective kernel (was *fl*-1414*): floury endosperm; lethal; cultured embryos normal	S		380
dek17	3L	defective kernel (was *cp*-330D*): collapsed endosperm; lethal; cultured embryos not obtained	S		380
dek18	5S	defective kernel (was *cp*-931A*): collapsed endosperm; lethal; cultured embryos green, narrow-leaved	S		380
dek19	6L	defective kernel (was *o*-1296A*): collapsed opaque endosperm; lethal; cultured embryos green	S		380
dek20	8L	defective kernel (was *cp*-1392A*): collapsed endosperm; lethal; cultured embryos green	S		380
dek21		(= *w2*)	S		
dek22	1L	defective kernel (was *cp*-1113A*): collapsed endosperm; lethal; cultured embryos not obtained	S		55 381
dek23	2L	defective kernel (was *dcr*-1428*): defective crown; lethal; cultured embryos not obtained	S		55 381
dek24	3S	defective kernel (was *cp*-1283*): collapsed endosperm; lethal; cultured embryos normal	S		381
dek25	4S-near *Ga1*	defective kernel (was *sh*-1167A*): shrunken endosperm; lethal; cultured embryos normal	S		381
dek26	5L	defective kernel (was *cp*-1331*): collapsed endosperm; lethal; cultured embryos normal	S		381
dek27	5L	defective kernel (was *cp*-1380A*): collapsed endosperm; lethal; cultured embryos green	S		381
dek28	6S	defective kernel (was *o*-1307A*): opaque endosperm	S		381
dek29	8L	defective kernel (was *cp*-1387A*): collapsed endosperm; viable; cultured embryos green, narrow-leaved	S		381
dek30	9L	defective kernel (was *fl*-139*): floury endosperm; lethal; cultured embryos green, narrow-leaved	S		381
dek31	4L-near *Tu1*	defective kernel (was *ptd*-1130*): pitted endosperm; lethal	S		379
dek32	1S	defective kernel (was *shop*-1322A*): dull, opaque, dented kernel; endosperm floury and soft; embryo degenerate			290
dek33	5L-near *ga2*	defective kernel (was *fldt*-1299*): opaque, floury, dented, wrinkled kernel with floury endosperm; occasionally viviparous			290
dep1	6	defective pistils			260
des17	8L	defective seedling: reduced height, partial suppression of primary root growth, contorted leaves, lethal			122
Df		deficiency: general symbol for loss of segments of chromosome			
Dhn1		see *Rab17*			
Dia1	2-near *v4*	diaphorase: electrophoretic mobility; cytosolic; monomeric			435
Dia2	1L-near *bm2*	diaphorase: electrophoretic mobility; cytosolic; dimeric			435
dib1	?	dichotomously branched: plants branch into two normal tops at nodes 4-8, variably; associated with aneuploidy			261
dp1	4L-118	distal pale: seedling leaf tip virescent (E.G. Anderson, unpublished)	S		
Ds		dissociation: designator for transposable factors regulated by *Ac*; modifies gene function and/or chromosome breakage (termed " *Ds-2*"); ex. *Ds2* designates element isolated from *Adh1-2F11*	S	P	253
dsc1	4S	discolored kernel (was *dek*-3252*): crumpled, discolored, lethal			174 364
dSpm		defective suppressor-mutator: designator for transposable factors regulated by *Spm*			365
dsy1	?	desynaptic: male and female sterility; synaptic failure			140
dsy2	?	desynaptic: like *dsy1*			143
dsy3	?	desynaptic: like *dsy1*			137
dsy4	?	desynaptic: like *dsy1*			137
Dt1	9S-0	dotted: regulated controlling element at *A1*; responding *a1-m* alleles express colored dots on colorless kernels and purple sectors on brown plants	S	P	336
Dt2	6L-44	dotted: like *Dt1*	S		311
Dt3	7L	dotted: like *Dt1*, but expression variable	S		311

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
Dt4	4	dotted: like *Dt1*, but dots chiefly on crown of kernel	S		81
Dt5	9S-near *yg2*	dotted: like *Dt1*			81
Dt6	4-near *su1*	dotted: like *Dt1*	S		400
du1	10L-31	dull endosperm: glassy, tarnished endosperm; affects soluble starch synthase and branching enzyme IIa (P.C. Mangelsdorf, 1935, unpublished data)	S		107 240
dv1	?	divergent spindle: chromosomes unoriented at metaphase I; partial male and female sterility	S		54
dy1	?	desynaptic: chromosomes unpaired in microsporocytes; partial male and female sterility	S		287
E1	7L-near *Tpi1*	esterase: electrophoretic mobility; null allele is known; dimeric; intralocus hybrid bands occur			369
E2	?	esterase: presence-absence			371
E3	3S	esterase: electrophoretic mobility; dimeric; intralocus hybrid bands occur			370
E4	3S-near *cl1*	esterase (was *Est4*): electrophoretic mobility; null allele is known; monomeric			158
E5(I)	?	esterase (duplicate factor with *E5(II)*): electrophoretic mobility			235
E5(II)	?	esterase (duplicate factor with *E5-(I)*): electrophoretic mobility			235
E6	?	esterase: presence-absence			235
E7	?	esterase: presence-absence			235
E8	3S-14	esterase: electrophoretic mobility; null allele is known; dimeric; intralocus hybrid bands occur			235
E9	?	esterase: electrophoretic mobility; null allele is known			235
E10	?	esterase: electrophoretic mobility			235
eg1	5L	expanded glumes: glumes open at right angle	S		41
Ej1		(= *lsr1*)			
el1	8L	elongate: chromosomes uncoiled during meiotic metaphase and anaphase in male and female; frequent unreduced gametes	S	P	341
emp1	1S	empty pericarp (was *dek-Mu*2045*): germless, unfilled kernel			364
emp2	2-near *ts1*	empty pericarp (was *dek*-1047*): germless, unfilled kernel			174 364
Emu1	2	endogenous *Mu*: *NPI347(EMU)*()			446
En		enhancer: transposable element (equivalent to *Spm*); autonomous, regulates *I* transposition (e.g. at *g2-m* = *pg-m* = *pg14-m*)		P	321
Enp1	6L-near *y1*	endopeptidase: electrophoretic mobility; null allele is known; monomeric			257
Est		(see *E*)			
et1	3L-161	etched: pitted, scarred endosperm, virescent seedling; plastid membranes altered	S	P	405
f1	1L-86	fine stripe: virescent seedling, fine white stripes on base and margin of older leaves	S	P	224 225
fae1	?	fasciated ear: small, rounded ears branched at their tips			379
Fas1	?	fascicled ear: ears and tassels branch dichotomously, may fasciate			329
Fbr1	?	few-branched (was *Fbr*-1602*): tassel reduced to 0-3 branches; bract replaces next-to-bottom branch			296
Fcu		factor Cuna: controlling element of *r1-cu*			146
fl1	2S-68	floury endosperm (= *o4*): endosperm opaque, soft; dosage effect with *fl1-ref* (but *o4* is recessive)	S	P	160
fl2	4S-39	floury: endosperm opaque, soft; dosage effect (W.J. Mumm, 1935, unpublished data)	S		107 289
fl3	8L-0	floury: endosperm opaque, soft; dosage effect	S		282
Flt		flint: designator for factors determining flint endosperm type			272
g1	10L-50	golden plant: seedling and plant with distinct yellow cast	S	P	96 98
g2	3S-0	golden plant (= *g5* = *pg14* = *v19*): like *g*, but more extreme; sheaths whitish yellow-green	S		176
g5		(=*g2*)			
G6	9S-near *l7*	golden plant (was *G*-1585*): like *g1*; lighter yellowish sheaths			306
Ga1	4S-13	gametophyte factor (= *ga9*): *Ga1* pollen grains competitively superior to *ga1* on *Ga1* silks; *Ga1-S* super-gametophyte	S		189
ga2	5L-55	gametophyte factor: *Ga2* pollen grains competitively superior to *ga2*	S		39
ga7	3L-167	gametophyte factor: *ga7* pollen from heterozygotes 10-15% functional regardless of silk genotype			338
ga8	9S-near *lo2*	gametophyte factor: *Ga8* pollen grains competitively superior to *ga8* on *Ga8* silks			368
ga9		(= *ga1*)			
ga10	5	gametophyte factor			145
Gdh1	1L-near *vp8*	glutamic dehydrogenase: electrophoretic mobility; null allele is known (cold sensitivity); intra/interlocus hybrid bands occur			331
Gdh2	10	glutamic dehydrogenase: electrophoretic mobility; intralocus hybrid bands occur			147
Ger		glucoside earworm resistance: designator for earworm resistance factors from Cateto Palha Roxa			272
Gll		Genetics Inst. Inc.: designator for loci defined by restriction fragment polymorphisms			
gl1	7L-36	glossy: cuticle wax altered; leaf surface bright, water adheres	S	P	209
gl2	2S-30	glossy: like *gl1*	S	P	161
gl3	4L-93	glossy: like *gl1*	S		161
gl4	4L-62	glossy (= *gl16*): like *gl1* (G.F. Sprague, unpublished)	S		
gl5	4-near *su1*	glossy (was *gl5-1*, duplicate factor with *gl20*): like *gl1* (G.F. Sprague, 1935, unpublished data)	S		107 401
gl6	3L-69	glossy: like *gl 1* (G.F. Sprague, 1935, unpublished data)	S		107

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
gl7	?	glossy (= *gl12*): like *gl1* (G.F. Sprague, 1935, unpublished data)			107
gl8	5L-68	glossy (= *gl10*): like *gl1* (G.F. Sprague, 1935, unpublished data)	S		107
gl9	3L	glossy: expression poor (G.F. Sprague, 1935, unpublished data)	S		107
gl10		(= *gl8*)			
gl11	2S-near *B1*	glossy: like *gl* ; abnormal seedling morphology	S		398
gl12		(= *gl7*)			
gl14	2	glossy (duplicate factor with *gl24*): like *gl1*			6
gl15	9L-66	glossy: like *gl1*; expressed after 3rd leaf	S	P	6
gl16		(= *gl4*)			
gl17	5S-34	glossy: like *gl1*, but semi-dwarf with necrotic crossbands on leaves	S		342
gl18	8L-near *fl3*	glossy: like *gl1*; expression poor	S		6
gl19	3S	glossy (was *gl*-169*): like *gl1*; lethal	S		299
gl20	?	glossy (was *gl5-2*, duplicate factor with *gl5*): like *gl1*	S		401
gl21	10S	glossy (was *gl*-478B*, duplicate factor with *gl22*): like *gl1*	S		299
gl22	?	glossy (was *gl*-478C*, duplicate factor with *gl21*): like *gl1*			295
gl23	?	glossy (was *gl*-PI262490*): like *gl1*			403
gl24	?	glossy (duplicate factor with *gl14*): like *gl1*			403
Glb1	1L-121	globulin (was *Pro*, *Prot1*): Mr 63,000, electrophoretic mobility; null allele is known; embryo protein			206 373
Glb2	?	globulin: Mr45,000, presence-absence			206
Gln1	10L-near *sr2*	glutamine synthetase candidate: see *BNL(GLN)*(GS6.15)			45
Glu1	10L-near *orp2*	beta glucosidase: electrophoretic mobility; cytosolic; dimeric; intralocus hybrid bands occur			332
Got1	3L-120	glutamate-oxaloacetate transaminase (possibly = *Ta1*): electrophoretic mobility; null allele is known; glyoxysomal; dimeric; intralocus hybrid bands occur			361
Got2	5L-96	glutamate-oxaloacetate transaminase: electrophoretic mobility; null allele is known; plastidial; dimeric; intralocus hybrid bands occur			149
Got3	5S-near *a2*	glutamate-oxaloacetic transaminase: electrophoretic mobility; null allele is known; mitochondrial; dimeric; intralocus hybrid bands occur			149
Gpa1	10L-near *g1*	glyceraldehyde-3-phosphate dehydrogenase, chloroplastic, A subunit; *UMC188-GPA*(cDNA probe pZm57)			35
Gpc1	4-near *orp1*	glyceraldehyde-3-phosphate dehydrogenase: cytosolic, C subunit; *UMC191-GPC*(cDNA probe pZm9)			35 243 355
Gpc2	?	glyceraldehyde-3-phosphate dehydrogenase, cytosolic, C subunit (cDNA probe pGAPC2)			355
Gpc3	4S-near *su1*	glyceraldehyde-3-phosphate dehydrogenase, cytosolic, C subunit (cDNA probe pGAPC3)			355
Gpc4	?	glyceraldehyde-3-phosphate dehydrogenase, EC1.2.1.12: isozyme variant			354
grt1	5L	green tip (was *grt*-1308B*): pale yellow seedling with green first leaf tip; lethal	S		299
gs1	1L-135	green stripe: grayish green stripes between vascular bundles on leaves; tissue wilts	S	P	98 266
gs2	2S-54	green stripe: like *gs1*, but pale green stripes; no wilting (G.F. Sprague, 1935, unpublished data)	S	P	107
gs3	6L	green stripe (was *gs*-268*): like *gs2*			299
Gs4	10	green stripe (was *Gs*-1439*): like *gs1*			297
gt1	1	grassy tillers: numerous basal branches; vegetatively totipotent in combination with *id1* and factors for perennialism			377
h1	3	soft starch: endosperm soft, opaque	S		280
H3		histone H3: designator for histone H3 family			
H4		histone H4: designator for histone H4 family			
hcf1	2L	high chlorophyll fluorescence: affects NADP+ oxidoreductase; green seedling			263
hcf2	1L	high chlorophyll fluorescence: missing cytochrome f/b6 complex; yellow-green seedling			263
hcf3	1S-near *P1*	high chlorophyll fluorescence (= *hcf9*): missing PSII thylakoid membrane core complex; green seedling			263
hcf4	1L	high chlorophyll fluorescence: affects CO2 fixation; green seedling			264
hcf5	6S	high chlorophyll fluorescence: affects PSII reaction; green seedling			265
hcf6	1S	high chlorophyll fluorescence: missing cytochrome f/b6 complex; green seedling			220
hcf9		(= *hcf3*)			
hcf12	1L	high chlorophyll fluorescence			220
hcf13	1L	high chlorophyll fluorescence: affects CO2 fixation; green seedling			220 264
hcf15	2L	high chlorophyll fluorescence: affects photophosphorylation; yellow-green seedling, may survive			220
hcf18	5L-near *pr1*	high chlorophyll fluorescence (= *hcf43*): major loss of PSI; other thylakoid complexes reduced; yellow-green seedling			264
hcf19	3L	high chlorophyll fluorescence: affects PSII thylakoid membrane core complex; green/yellow-green seedling			220 264
hcf21	5L	high chlorophyll fluorescence: affects CO2 fixation, Rubisco; green seedling			264

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
hcf23	4S	high chlorophyll fluorescence: affects photophosphorylation; yellow-green seedling, may survive			220 264
hcf26	6S	high chlorophyll fluorescence: affects electron transport; yellow-green, viable seedling			220 264
hcf28	10L	high chlorophyll fluorescence: affects CO2 fixation; green seedling			265
hcf31	1S	high chlorophyll fluorescence: missing chlorophyll a/b binding protein; yellow-green seedling			265
hcf34	6L	high chlorophyll fluorescence: affects photophosphorylation; yellow-green seedling			220 264
hcf36	6L	high chlorophyll fluorescence: affects electron transport; green seedling			265
hcf38	5L	high chlorophyll fluorescence: affects cytochrome f/b6 complex, alpha and beta components of CF1; green seedling			220
hcf41	1L	high chlorophyll fluorescence: affects PSII thylakoid membrane core complex; green seedling			220 264
hcf42	9L	high chlorophyll fluorescence: affects Rubisco; green/yellow-green seedling			264
hcf43		(= *hcf18*)			
hcf44	1L	high chlorophyll fluorescence: affects PSI membrane core complex; pale-green seedling			264
hcf46	3L	high chlorophyll fluorescence			220
hcf47	10S	high chlorophyll fluorescence: affects cytochromes; yellow-green seedling			265
hcf48	6L	high chlorophyll fluorescence: affects electron transport; yellow-green seedling			265
hcf50	1L	high chlorophyll fluorescence: missing PSI thylakoid membrane core complex; green seedling			264
hcf101	7L	high chlorophyll fluorescence (was *Mu-5**): affects PSI thylakoid membrane core complex			265
hcf102	8L	high chlorophyll fluorescence: affects cytochrome f/b6 complex (D. Miles, unpublished)			
hcf103	7L	high chlorophyll fluorescence: affects PSII			67
hcf104	7L	high chlorophyll fluorescence: photosystem I-deficient			67
hcf106	2-near *ts1*	high chlorophyll fluorescence: affects PSI, PSII, cytochrome f/b6; *BNL(HCF106)*			242
hcf108	5	high chlorophyll fluorescence: ATPase-deficient			67
hcf111	7L	high chlorophyll fluorescence: cytochrome b/f-deficient			67
hcf113	9S	high chlorophyll fluorescence: multiple effects; yellow-green seedlings			66
hcf114		(= *hcf103*)			67
hcf134	8L	high chlorophyll fluorescence: lacks PSII core complex; pale seedling, mutable, *Mu*-induced			432
hcf316	10S	high chlorophyll fluorescence: affects chlorophyll a/b binding protein; yellow-green seedling			265
hcf323	6S	high chlorophyll fluorescence: affects photophosphorylation, coupling factor; green seedling			265
hcf408	6L	high chlorophyll fluorescence: affects chlorophyll a/b binding protein; yellow-green seedling			265
Hex1	3S-near *Cg1*	hexokinase: electrophoretic mobility; null allele is known; cytosolic; monomeric			437
Hex2	6L-near *Pt1*	hexokinase: electrophoretic mobility; null allele is known; cytosolic; monomeric			437
hm1	1L-64	*Helminthosporium carbonum* susceptibility: disease lesions vs. yellowish flecks (resistant) on leaves with race 1	S	P	427
hm2	9L-near *bk2*	*Helminthosporium carbonum* susceptibility: like *hm1*; masked by *Hm1*			286
Hrg1	2-near *wt1*	hydroxyproline-rich glycoprotein candidate (cDNA probe): see *UMC145(HRG)*(pMC56)			408
Hs1	7S-0	hairy sheath: abundant hairs on leaf sheath	S	P	419
Hsf1	5	hairy sheath frayed (was *Hsf*-1595*): pubescent sheaths and leaf margins; liguled enations at leaf margins			26
Hsp1	8L-near *Act1*	heat shock protein (70kD) candidate: see *NPI119(HSP70)*()			446
Ht1	2L-121	*Helminthosporium turcicum* resistance	S		167
Ht2	8L-near *j1*	*Helminthosporium turcicum* resistance			168
Ht3	?	*Helminthosporium turcicum* resistance: (from *Tripsacum floridanum*)			169
I		inhibitor (= *C1-I*, inhibitor allele at *C1* locus): also commonly used as a general symbol for inhibition and for the controlling elements responding to *En*; (see also Inv)			
id1	1L-near *an1*	indeterminate growth: requires extended growth and short days for flowering; vegetatively totipotent with *gt1* and factors for perennialism	S		386
Idh1	8L-near *Ht2*	isocitrate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			149
Idh2	6L-near *w14*	isocitrate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			149
ig1	3L-90	indeterminate gametophyte: low male fertility, polyembryony, heterofertilization, polyploidy, androgenesis (male and female affected)	S		202
ij1	7L-52	iojap striping: many variable white stripes and margin patterns on leaves (compare *cm1*); conditions chloroplast defects that are cytoplasmically inherited	S	P	175
ij2	1L-near *bm2*	iojap striping: like *ij1*; chloroplast inheritance unknown			299
in1	7S-20	intensifier: intensifies aleurone anthocyanin pigments; *In1-D* dominant dilute	S	P	117
Inv		Inversion: general symbol for inversion of a segment of chromosome	S	P	
is1	?	cupulate interspace			125
Isr1	10L-near *R1*	inhibitor of striate (was *Ej1*): reduces expression of *sr2* and other leaf-striping factors	S		203
j1	8L-42	japonica striping: white stripes on leaf and sheath; not expressed in seedling	S	P	98
j2	4L-87	japonica striping: extreme white striping of leaves, etc. (R.A. Emerson, 1935, unpublished data)	S	P	107
K		knob: general symbol for heterochromatic structures that are heritably polymorphic in size and are found at characteristic positions on the chromosomes; homology with 185bp probe			317

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
K3L	3L-115	knob: heterochromatic structure (see K)			77
K10	10L-near *sr2*	abnormal-10: heterochromatic appendage on long arm of chromosome 10; neocentric activity distorts segregation of linked genes	S	P	230
Kn1	1L-near *Adh1*	knotted: localized proliferation of tissue at vascular bundles on leaf	S	P	37
Kn2	?	knotted: finger-like projections of leaf at the ligule			120
Krn		kernel row number: designator for factors determining kernel row number			272
l1	10L-near *R1*	luteus: yellow pigment in white tissue of specific chlorophyll mutants *w1, w2, j1, ij1*, others	S	P	223 224
l3	?	luteus: lethal yellow seedling	S		183
l4	?	luteus: lethal yellow seedling	S	P	182
l6	9S-near *bz1*	luteus: like *l4* (W.H. Eyster, 1935, unpublished data)	S		107
l7	9S-42	luteus: yellow seedling and plant; lethal	S		114
l10	6L-19	luteus: like l4; fails to convert protochlorophyllide to chlorophyllide	S		348
l11	6S	luteus (was *l*-4120*): yellow seedling with green leaf tips; lethal	S		10
l12	6L-16	luteus (was *l*-4920*): like *l11*	S		71
l13	10L-94	luteus (was *l*-59A, l*-Neuffer2*): dark yellow, lethal seedling; fails to convert protoporphyrin IX to Mg-protoporphyrin			244 299
l15	6L-30	luteus (was *l*-Blandy3, l*-Brawn*): like *l4*			352
l16	1S	luteus (was *l*-515*): like *l4*; leaves bleach to paler yellow in patches			299
l17	1L	luteus (was *l*-544*): like *l4*; leaves with lighter yellow crossbands			299
l18	2L	luteus (was *l*-1940*): *like l4*			299
l19	10S	luteus (was *l*-425*): like *l4*			299
la1	4S-36	lazy plant: prostrate growth habit	S	P	181
lbl1	?	leaf bladeless: leaf blade reduced to absent; low temperature enhances expression			262
Lc1	10L-68	red leaf color: anthocyanin in coleoptile, nodes, auricle, leaf blade, etc.; (compare *Sn1*)	S		86
Lcs1	?	thylakoid membrane polypeptide: electrophoretic mobility			275
Lct1	?	thylakoid membrane polypeptide: electrophoretic mobility			275
lct2	?	thylakoid membrane polypeptide: presence-absence			275
Les1	2S-58	lesion (was *Les*-843*): large necrotic lesions resembling disease lesions formed by fungal infections on susceptible lines	S		302
Les2	1S-near *sr1*	lesion (was *Les*-845A*): small white lesions resembling disease lesions formed by fungal infections on resistant lines	S		302
Les3	10	lesion: like *Les1*			9
Les4	2L	lesion (was *Les*-1375*): late expression of large necrotic lesions	S		164
Les5	1S	lesion (was *Les*-1449*): like *Les2*			164
Les6	10S	lesion (was *Les*-1451*): like *Les4*	S		164
Les7	?	lesion (was *Les*-1461*): late expression of small chlorotic lesions	S		164
Les8	9S-near *lo2*	lesion (was *Les*-2005*): late expression of small, pale green lesions	S		164
Les9	7L-near *ra1*	lesion (was *Les*-2008*): late expression of small necrotic lesions	S		164
Les10	2-near *v4*	lesion (was *Les*-A607*): like *Les1*			165
Les11	2S	lesion (was *Les*-1438*): like *Les1* but slightly later in expression and less responsive to temperature and to background genotype; EMS-induced			290
Les12	10	lesion (was *Les*-1453*): Many small to medium, chlorotic to necrotic lesions form in clusters on the leaf blade beginning at 5 leaf stage and rapidly coalesce to form large senescent areas that may spread over the whole leaf and cause early death; EMS-induced			290
Les13	6L	lesion (was *Les*-2003*): frequent small to medium necrotic spots on leaf blade, sheath and culm, appearing at the 5 leaf stage; some enlarge and coalesce to form long necrotic strips along leaf veins, eventually spreading and causing senescence of the whole leaf; plants are lighter green and 1/3 to 2/3 normal height; EMS-induced			290
Les14	3L	lesion (was *Les*-2004*): many small round brown necrotic spots with light centers, some with anthocyanin halos on leaf blade beginning at the 6 leaf stage, no reduction in height or vigor; EMS-induced			290
Les15	2S	lesion (was *Les*-2007*): tiny yellowish green plants with many small chlorotic and necrotic lesions on speckled yellow green leaf blade background that looks like iron deficiency symptoms; EMS-induced			290
Les16	10	lesion (was *Les*-2016*): pale green plant develops small chlorotic lesions on the leaf blade just before flowering; EMS-induced			290
Les17	3L	lesion (was *Les*-2345*): profuse small to medium chlorotic and necrotic lesions expressed at 8-10 leaf stage causing plants to have a light green color; occasional normal green non-lesion sectors appear on leaves; spontaneous			290
Lfy1	?	leafy: increased number of leaves			376
lg1	2S-11	liguleless: ligule and auricle missing; leaves upright, enveloping	S	P	96 97
lg2	3L-101	liguleless: like *lg1*, less extreme	S	P	31
Lg3	3-65	liguleless: dominant, no ligule; leaves upright, broad, often concave and pleated	S	P	318
Lg4	8L	liguleless (was *Lg*-403*): dominant, no ligule or auricle but vestiges sporadically in blade			116
li1	10L-near *bf2*	lineate leaves: fine, white striations on basal half of mature leaves	S	P	65

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
lls1	1S	lethal leaf spot: chlorotic-necrotic lesions resembling *Helminthosporium carbonum* infection	S		426
ln1	6	linoleic acid: lower ratio of oleate to linoleate in kernel			72
lo2	9S-50	lethal ovule: ovules containing *lo2* gametophyte abort			287
loc1	?	low oil content in kernel: associated with albino seedlings			327
lp1	4	lethal pollen: *lp1* pollen fails in competition with *Lp1*			284
lte1	2	latente: drought, heat, aluminum tolerance; frost resistance; from Michoacan 21; dominance varies			269
Lte2	10L-near *g1*	latente: drought, heat, aluminum tolerance; from Cateto; epistatic to *lte1*			270
lty1	?	light yellow endosperm			83
lty2	?	light yellow endosperm			83
lu1	5S-29	lutescent: pale yellow green leaves	S		383
lw1	1L-near *Adh1*	lemon white: white seedling, pale yellow endosperm	S		424
lw2	5L-near *pr1*	lemon white: like *lw1*	S	P	424
lw3	5L-near *v2*	lemon white (duplicate factor with *lw4*): like *lw1*	S		424
lw4	4-near *zb6*	lemon white (duplicate factor with *lw3*): like *lw1*	S		424
Lxm1	3	lax midrib (was *Lxm*-1600*): leaves with wide, flat, flexible midrib	S		294
lyc1		(= *ps1-lyc*)			
mal1	9	multiple aleurone layering: recessive interacts with two complementary dominants *Mal2* and an unnamed factor, giving multiple cell layers			267
Mal2	4	multiple aleurone layering: (see *mal1*)			267
Mc1	?	mucronate: opaque endosperm; dosage effect			358
Mch1	?	maize CRY1 homolog: ribosomal protein gene family (cDNA probe)			214
Mch2	?	maize CRY1 homolog: ribosomal protein gene family (cDNA probe)			214
Mct1	?	modifier of *cox2* transcripts: changes transcripts of mitochondrial gene			68
Mdh1	8-near *fl3*	malate dehydrogenase: electrophoretic mobility; null allele is known; mitochondrial; dimeric; intra/interlocus hybrid bands occur			307
Mdh2	6L-near *w14*	malate dehydrogenase: electrophoretic mobility; null allele is known; mitochondrial; dimeric; intra/interlocus hybrid bands occur			307
Mdh3	3L-146	malate dehydrogenase: electrophoretic mobility; null allele is known; mitochondrial; dimeric; intra/interlocus hybrid bands occur			307
Mdh4	1L-near *an1*	malate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			307
Mdh5	5S-17	malate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			307
Mdm1	6-near *w15*	maize dwarf mosaic virus resistance			256
mdr1	4L	maternal derepression of *R*: maternally transmitted *R* with *Mdr1* expresses solid color, with *mdr1* mottled			201
Me1	3L-125	NADP malic enzyme: electrophoretic mobility; null allele is known; tetrameric; *NPI231(ME)*(), *YNH(ME)*() (may be ambiguous)			149
Me2	6L-near *py1*	NADP malic enzyme: cytosolic; *NPI330(ME)*()			446
Mei1	?	meiosis: chromosomes sticky in metaphase I; male sterile			139 142
mep1	5L	modifier of embryo protein: affects quantities of *Glb* (was *Pro*) protein forms			373
Mer		Maya earworm resistance: designator for earworm resistance factors from IAC Maya			271
mg1	?	miniature germ (replaces *mg* of Wentz): germ 1/4 to 1/3 of normal; viable			211
Mgs1	10-near *g1*	male-gametophyte specific: cDNA expressed in pollen; *BNL(MGS1)*(Zmc13)			154
Mgs2	4L-near *c2*	male gametophyte-specific: cDNA with pectin lyase homology; *BNL(MGS2)*(cDNA probe Zmc58)			45
mi1	1	midget plant: small plant (H.S. Perry, 1935, unpublished data)			107
mmm1	1L-near *an1*	modifier of mitochondrial malate dehydrogenases: mobilities			307
mn1	2-near *fl1*	miniature seed: small, somewhat defective kernel, fully viable; invertase reduced	S	P	232
mn2	7	miniature seed: small kernel, loose pericarp; extremely defective but will germinate (R.J. Lambert, unpublished)			
mn3	6-near *Mdm1*	miniature seed (was *de*-1184*): small kernel, etched/pitted endosperm; viable			412
Mod		modifier: inactive *Spm* element, enhances excisions elicited by active *Spm*			255
Mp		modulator of pericarp: transposable factor affecting *P1* locus; parallel to *Ac-Ds*			34
Mpi1		transposable element: 10-15 copies in the genome			441
Mpl1	1L-near *Adh1*	miniplant: dominant, andromonoecious, intermediate dwarf (compare *D8*, possible allele); not responsive to gibberellins	S		156
Mr	9S-near *l7*	mutator of *R1-m*: transposable factor, regulates *R1-m* mutation		P	49
Mrh	5	mutator: controlling element of *a1-m-rh*			343
ms1	6L-near *si1*	male sterile: anthers shriveled, not usually exserted; affected at microspore vacuolation	S		389
ms2	9L-64	male sterile: like *ms1*; affected between vacuolation and pore formation	S		112 114
ms3	3	male sterile: anthers shrivelled; not usually exserted			112 114
ms4		(= *po1*)			
ms5	5-near *v3*	male sterile: anthers not exserted; affected at microspore mitosis	S		18

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
ms6		(= *po1*)			
ms7	7L-near *ra1*	male sterile: like *ms2*	S		18
ms8	8L-28	male sterile: like *ms5*; affected in meiosis	S	P	18
ms9	1S-near *P1*	male sterile: like *ms5*; affected in meiosis	S		18
ms10	10L-near *g1*	male sterile: like *ms5*; affected at microspore vacuolation	S		18
ms11	10	male sterile: like *ms5*; affected at microspore mitosis	S		18
ms12	1	male sterile: like *ms1*; affected at microspore vacuolation	S		18
ms13	5S	male sterile: like *ms5*; affected at microspore vacuolation	S		18
ms14	1-near *as1*	male sterile: like *ms5*; affected at microspore mitosis	S		18
ms17	1S-23	male sterile: like *ms1*; affected variably in meiosis	S		104
ms20	?	male sterile			114
Ms21	6	male sterile: pollen grains developing in presence of *Ms21* are defective and nonfunctional if *sks1*, normal if *Sks1*			218 367
ms22	?	male sterile: affected in meiosis	S		440
ms23	3L	male sterile (allelic to *ms*-Bear7*): affected in meiosis	S		440
ms24	?	male sterile: like *ms*1; affected in microspore mitosis	S		440
ms28	?	male sterile: anaphase I disturbed, spindle persists			142
Ms41	4L	male sterile (was *Ms*-1995*)	S		306
ms43	8L	male sterile: anaphase I impaired			139 142
Ms44	4L-near *c2*	male sterile (was *Ms*-7255*)			2
Msc1	1L	mosaic (was *Msc*-791A*): aleurone mosaic for anthocyanin color	S		306
Msc2	5S	mosaic (was *Msc*-1124B*): aleurone mosaic for anthocyanin color			306
Mst1	10L-70	modifier of *R-st*: affects expression of *R1-st*	S		8
mtDNA		mitochondrial DNA: sequences or loci in the mitochondrial genome			
Mu		mutator: freely transposable element; *Mu1* designates element isolated from *Adh1-S3034*			351
Mut	2S-near *gl2*	mutator: controlling element for *bz1-m-rh*			343
Mv1	?	resistance to maize mosaic virus I ("corn stripe")			28
na1	3L-113	nana plant: short, erect dwarf; no response to gibberellins		P	173 222
na2	5S-near *bt1*	nana plant: like *na1* (H.S. Perry, unpublished)	S		
Nabp1	7S-near *vp9*	nucleic acid binding protein			45
NCR		North Carolina, Raleigh: designator for loci defined by restriction fragment polymorphisms			
NCS1		nonchromosomal stripe: maternally inherited light green leaf striping			384
NCS2		nonchromosomal stripe: maternally inherited pale green and depressed striping; mitochondrial	S		58
NCS3		nonchromosomal stripe: maternally inherited striations, distorted plants; mitochondrial	S		58
NCS5		nonchromosomal stripe: maternally inherited stunted growth, yellow stripes, aborted kernels; mitochondrial cytochrome oxidase subunit 2 (*cox2*) alteration			308
NCS6		nonchromosomal stripe: maternally inherited stunted growth, yellow stripes, aborted kernels; mitochondrial cytochrome oxidase subunit 2 (*cox2*) alteration			215
nec1	8L-near *fl3*	necrotic (was *nec*-669*, *sienna*-7748*): chlorotic seedling that stays rolled, wilts and dies	S		245
nec2	1S-34	necrotic (was *nec*-8147*, olive-necrotic-8147, ON-8147): green seedling develops necrotic lesions at 2-3 leaf stage; lethal (E.G. Anderson, 1952, unpublished data)	S		
nec3	5-near *bt1*	necrotic (was *nec*-409*): seedling emerge with tightly rolled leaves that turn brown and die without unrolling; manually unrolled leaves tan with dark brown crossbands	S		293
nec4	2S-near *d5*	necrotic (was *nec*-516B*): seedling yellow, leaf tips necrotic; lethal	S		166
nec5	4L	necrotic (was *nec*-642A*): pale green seedling becoming necrotic; dark brown exudate; lethal	S		299
nec6	5S-near *a2*	necrotic (was *nec*-493*): like *nec3*	S		299
nec7	5L	necrotic (was *nec*-756B*): seedling becoming necrotic in crossbands; lethal			299
NIU		Northern Illinois University: designator for loci defined by restriction fragment polymorphisms			
nl1	10L-near *bf2*	narrow leaf: leaf blade narrow, some white streaks (R.A. Emerson, 1935, unpublished data)	S	P	107
Nl2	5S-25	narrow leaf (was *Rgd2*, *Rgd*-1445*): leaves narrow and distorted; tillering			300
NOR	6S	nucleolus organizer: codes for ribosomal RNA; *BNL(NOR)*(pBF243)	S		251
NPI		Native Plants, Inc.: designator for loci defined by restriction fragment polymorphisms			
ns1	?	narrow sheath: sheath and lower blade tapered, brachytic plant			94
o1	4L-79	opaque endosperm: endosperm starch soft, opaque (W.R. Singleton and D.F. Jones, 1935, unpublished data)	S		107 289
o2	7S-16	opaque endosperm: like *o1* (W.R. Singleton and D.F. Jones, 1935, unpublished data); high lysine content; regulates b-32 protein (see *pro1*); reduced lysine degradation (lysine-ketoglutaric reductase); *BNL(O2)*(pXho0.9), *NPI480(O2)*()	S	P	107 289
o4		(= *fl1*)			
o5	7L-near *ra1*	opaque endosperm: like *o1*; virescent to yellow or white seedlings	S		347
o6		(= *pro1*)			
o7	10L-90	opaque: like *o* ; high lysine content	S		273
o9	?	opaque endosperm (was *ox*-74120*): crown opaque and light in color, frequently with a cavity; base or abgerminal side of kernel often corneous	S		283
o10	1L	opaque endosperm (was *o*-E1356*, *ox*-7747*): like *o1*	S		283

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
o11	?	opaque endosperm (was *ox*-7455*): thin, opaque, somewhat shrunken kernels with greyish cast	S		283
o12	?	opaque endosperm (was *ox*-7636*): thin, etched or scarred kernels, variable in size; plants chlorophyll deficient and small, with pollen but few ears	S		283
o13	1S	opaque endosperm (was *ox*-7729*): opaque, etched kernels with rim of corneous starch on abgerminal side	S		283
o14	6L	opaque kernel (was *o*-924*): large opaque kernel with mostly floury starch except for a small amount of corneous starch near the base on the abgerminal side; normal green seedling develops yellow striped appearance and is slow in growth			290
Obf1	1-near *as1*	octopine synthase binding factor: encodes protein with bZIP motif that binds to transcriptional enhancer sequences (ocs-elements)			385
Obf2	9-near *wx1*	octopine synthase binding factor: encodes protein with bZIP motif that binds to transcriptional enhancer sequences (ocs-elements)			385
Oec1	4	oxygen-evolving complex protein candidate: see *NPI472(OEC)*(), *UMC171A(OEC23)*(8-18)			445
Oec2	2S-near *B1*	oxygen-evolving complex protein candidate: see *NPI473(OEC)*(), *UMC171B(OEC23)*(8-18)			445
Oec3	5S-near *Pgm2*	oxygen-evolving complex protein candidate: see *NPI474(OEC)*()			445
Oec4	7L	oxygen-evolving complex protein candidate: see *NPI475(OEC)*()			445
Oec5	8	oxygen-evolving complex protein candidate: see *NPI476(OEC)*()			445
Og1	10S-19	old gold stripe: variable bright yellow stripes on leaf blade	S	P	228
ora2	?	orange endosperm			82
ora3	?	orange endosperm			83
oro1	6S	orobanche: yellow to tan necrotic with cross-banding when grown under light-dark cycle; some chlorophyll with *Orom1*; fails to convert Mg-protoporphyrin monomethyl ester to protochlorophyllide	S		244
oro2	?	orobanche: like *oro1*			244
Orom1	?	orobanche modifier: partially corrects chlorophyll loss in *oro1*			244
orp1	4S-near *su1*	orange pericarp (duplicate factor with *orp2*): pericarp orange over *orp1 orp2* kernels; lethal, tryptophan auxotroph; tryptophan synthase B; *BNL(ORP1)*(trpB), *UMC193A(ORP)*(trpB)	S		301
orp2	10S-near *y9*	orange pericarp (duplicate factor with *orp1*): *BNL(ORP2)*(trpB); *UMC193B(ORP)*(trpB)	S		301
os1	2S	opaque-endosperm, small germ: opaque crown; kernel larger, lighter color; viable; reduced oil content			394
oy1	10S-15	oil yellow: seedling oily greenish-yellow; viable; fails to convert protoporphyrin IX to Mg-protoporphyrin; *oy1-t* tinged green; *oy1-1039*, *oy1-1040* lethal; *Oy1-700* dominant yellow-green (see Coe et al., 1988)	S	P	113
P		plant color component at *R1*: anthocyanin pigmentation in seedling leaf tip, coleoptile, anthers			85 406 407
P1	1S-26	pericarp color: red pigment in cob and pericarp (for alleles, see Coe et al., 1988); *NPI370(P1)*(), *UMC185(P)*	S	P	95 229
pam1	?	plural abnormalities of meiosis: desynchronized meiotic divisions and premeiotic mitosis; male sterile, incompletely female sterile			141
pam2	?	plural abnormalities of meiosis: like *pam1*			143
pb1	6L-near *y1*	piebald leaves: very light, irregular green bands on leaf		P	76
pb4	6L-near *y1*	piebald leaves: like *pb1*	S		76
Pcr1	1L-near *br1*	protochlorophyllide reductase candidate: see *RNY(PCR1)*(OR1)			45
Pcr2	5S-near *Mdh5*	protochlorophyllide reductase candidate: see *RNY(PCR2)*(OR1)			45
Pcr3	2S-near *al1*	protochlorophyllide reductase candidate: see *BNL(PCR3)*(OR1)			45
Pcr4	7S-near *in1*	protochlorophyllide reductase candidate: see *BNL(PCR4)*(OR1)			45
pd1	3	paired rows: single vs. paired pistillate spikelets; quantitative, one of a family of loci differentiating maize vs. teosinte			212
Pdf1	?	thylakoid membrane polypeptide: dominant increase in electrophoretic mobility			276
Pdk1	6L	pyruvate, Pi dikinase candidate: see *UMC173(PDK)*(p1-9), *NPI229(PDK)*()			247
Pdk2	8L-near *pro1*	pyruvate, Pi dikinase candidate: see *NPI230(PDK2)*()			247
pe		perennialism: vegetatively totipotent in combinations with *gt1* and *id1*; multifactorial trait			
Pep1	9-near *pg12*	phosphoenol pyruvate carboxylase candidate: see *NPI332(PEP1)*()			446
Pep2	5-near *a2*	phosphoenolpyruvate carboxylase candidate: see *NPI(PEP)*()			434
Pep3	4L-near *c2*	phosphoenolpyruvate carboxylase candidate: see *NPI(PEP)*()			434
pg11	6L-38	pale green (duplicate factor with *pg12*): seedling light yellowish green; mature plant pale and vigorous	S	P	339
pg12	9-61	pale green (duplicate factor with *pg11*)	S		339
pg13	?	pale green: seedling light yellowish green; stunted growth			382
pg14		(= *g2*)	S	P	321
pg15	1S	pale green (was *ppg*-340B*): seedling light yellowish green; bleaches to near white in patches; lethal	S		299

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
pg16	1L	pale green (was *pg*-219*): seedling light yellowish green	S		299
Pgd1	6-near *rgd1*	6-phosphogluconate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			149
Pgd2	3L-near *Rg1*	6-phosphogluconate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			149
PGE		Plant Gene Expression Center: designator for loci defined by restriction fragment polymorphisms			
Pgm1	1L-near *Glb1*	phosphoglucomutase: electrophoretic mobility; null allele is known; cytosolic; monomeric			149
Pgm2	5S-0	phosphoglucomutase: electrophoretic mobility; null allele is known; cytosolic; monomeric			149
Ph		pith abscission: cob disarticulation; multifactorial family of loci differentiating maize vs. teosinte			
Phi1	1L-149	phosphohexose isomerase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intralocus hybrid bands occur			149
Php1	10S-near *orp2*	chloroplast phosphoprotein: isozyme			45
Phy1	1L-near *Adh1*	phytochrome phyA1: see *BNL(PHYA1)*(pcPhy101), *NPI251(PHY)*()			446
Phy2	5S-near *Pgm2*	phytochrome phyA2: see *BNL(PHYA2)*(pcPhy101), *NPI369(PHY)*()			446
pi1	?	pistillate florets (duplicate factor with *pi2*): secondary florets develop ("Country Gentlemen" or "Shoe Peg" expression) in *pi1 pi2* ears; quantitative character			171
pi2	?	pistillate florets (duplicate factor with *pi1*)			171
PIO		Pioneer Hi-Bred, International: designator for loci defined by restriction fragment polymorphisms			
Pl1	6L-49	purple plant: sunlight-independent purple pigment in plant; regulates flavonoid enzymes; *Bh1*, blotched aleurone, is allelic *BNL(PL1)*(pH3-Sal0.6)	S	P	102
pm1	3L-near *ts4*	pale midrib: midrib and adjacent tissue lighter green; reduced plant vigor	S	P	32
Pn1	7L-112	papyrescent glumes: long, thin papery glumes on ear and tassel		P	127
po1	6S-4	polymitotic (= *ms4*): repeats 2nd meiotic division in male and female	S	P	16
ppg1	5L	pale pale green (was *cb*-199A*): white seedling with faint green; white necrotic crossbands; lethal			299
pr1	5L-67	red aleurone: changes purple aleurone to red; flavonoid 3'-hydroxylase	S	P	90
pra1	?	prophase I arrest: meiosis arrested at prophase of first division			138
pro1	8L-near *fl3*	proline responding (= *o6*): crumpled opaque kernel; green-striped lethal seedling; proline supplementation improves growth			130
Prot1		(= *Glb1*)			
Prx		(see *Px*)			
ps1	5S-39	pink scutellum (= *vp7*): viviparous; endosperm and scutellum pink, seedling white with pink flush; *ps1-lyc* not viviparous	S	P	397
Pt1	6L-60	polytypic ear: proliferation produces irregular growth on ear and tassel	S	P	288
ptd1	1L-116	pitted endosperm: lethal			364
ptd2	7L	pitted endosperm: lethal			364
Px1	2L	peroxidase: electrophoretic mobility; null allele is known; monomeric			153
Px2	?	peroxidase: electrophoretic mobility; monomeric			234
Px3	7L-near *Pn1*	peroxidase: electrophoretic mobility; monomeric			234
Px4	?	peroxidase: electrophoretic mobility; null allele is known; monomeric			234
Px5	?	peroxidase: presence-absence			234
Px6	?	peroxidase: presence-absence			234
Px7	?	peroxidase: electrophoretic mobility; null allele is known; monomeric			234
Px8	?	peroxidase: electrophoretic mobility; monomeric			29
Px9	?	peroxidase: electrophoretic mobility; null allele is known; monomeric			29
py1	6L-69	pigmy plant: leaves short, pointed; fine white streaks	S	P	418
py2	1L-near *bm2*	pigmy: like *py1*			299
pyd1	9S-near *yg2*	pale yellow deficiency: pale yellow seedling; deficiency for short terminal segment of chromosome arm; lethal; (for alleles, see Coe et al., 1988)			252
R1	10L-64	colored: red or purple color in aleurone and/or anthers, leaf tip, brace roots, etc. (for alleles, see Coe et al., 1988); regulates flavonoid enzymes; *BNL(R10*(R5-4frag2), *NPI308(R1)*()	S	P	90
ra1	7L-32	ramosa: ear and tassel many-branched; tassel branches taper to tip	S	P	18 133
ra2	3S-49	ramosa: irregular kernel placement; tassel many-branched, upright (R.A. Brink, 1935, unpublished data)	S	P	107 309
ra3	4	ramosa: (H.S. Perry, 1954, unpublished data)	S		
Rab17	6L-near *py1*	responsive to abscisic acid (was *RNY1, Dhn1*): dehydration-induced mRNA & dehydrin protein; *BNL(RAB17)*(cDNA probe) *UMC170(DHN)*(pM3-4)			56 430
Rab30	1S-near *sr1*	responsive to abscisic acid: *BNL(RAB30)*(cDNA probe)			45
rbcS		see *Ssu1*			
rBg		receptor of *Bg*			357
Rcm1	7-near *vp9*	rectifier: restores miniature seed of teosinte cytoplasm to normal			3

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
Rcm2	?	rectifier: weakly restores miniature seed of teosinte cytoplasm to normal			3
Rcm3	?	rectifier: restores miniature seed of teosinte cytoplasm to normal; from *Z. diploperennis*			3
rcu		receptor of *Fcu*			146
rcy		receptor of *Cy*			366
rd1	1L-near *gs1*	reduced plant: semi-dwarf plant	S		285
rd2	6L	reduced plant: like *rd1*, but not as extreme			136
rd3	3L-near *vp1*	reduced plant: like *rd1*; anthocyanin interactions			249
rDNA		ribosomal DNA: rDNA5.8S, rDNA18S and rDNA25S located in NOR on 6S; rDNA5S on 2L near *Ht1*			
rDt		receptor of Dotted			395
ren1	5L-near *pr1*	reduced endosperm (was *dek*-807*, *dek*-PI-OI*): opaque, lethal			364
ren2	7L-near *Tpi1*	reduced endosperm (was *dek*-NS413*, *dek*-NS326*): lethal			174 364
Rf1	3S-near *Wrk1*	fertility restorer: restores fertility to cms-T; complementary to *Rf2*	S		187
Rf2	9-near *wx1*	fertility restorer: see *Rf1*	S		89
Rf3	2L-near *Ch1*	fertility restorer: restores fertility to cms-S	S		38
Rf4	8-near *Bif1*	fertility restorer (complementary with *Rf5* and *Rf6*): restores fertility to cms-C			150
Rf5	?	fertility restorer (complementary with *Rf4* and *Rf6*): restores fertility to cms-C			192 429
Rf6	?	fertility restorer (complementary with *Rf4* and *Rf5*): restores fertility to cms-C			192 429
Rf7	?	fertility restorer: partially restores fertility to cms-Y			334
Rg1	3-67	ragged leaves: defective tissue between veins of older leaves, causing holes and tearing	S	P	33
rgd1	6-8	ragged seedling: seedling leaves narrow, thread-like, have difficulty in emerging	S	P	205
Rgd2		(= *Nl2*)			
rgo1	?	reversed germ orientation: embryo faces base of ear; variable frequency, maternal trait			356
rhm1	6-near *rgd1*	resistance to *Helminthosporium maydis*: chlorotic-lesion reaction with race O			390
Rii	4S-8	rind abscission: cob disarticulation; quantitative, one of a family of loci differentiating maize vs. teosinte			126
Rld1	?	rolled leaf (was *Rld*-1441*, *Rld*-1990*): leaves tightly rolled and tend to be entangled; ligular flaps on abaxial surface of leaf; resembles *Ce1*			26
rMrh		receptor of *Mrh*			343
rMut		receptor of *Mut*			343
Rp1	10S-3	resistance to *Puccinia sorghi*	S	P	236 237
Rp3	3-near *Rg1*	resistance to *Puccinia sorghi*	S		442
Rp4	4S-5	resistance to *Puccinia sorghi*	S		442
Rp5	10S-0	resistance to *Puccinia sorghi*			359
Rp6	10S-near *Rp1*	resistance to *Puccinia sorghi*			442
RPA		Rhone-Poulenc Agrochimie: designator for loci defined by restriction fragment polymorphisms			
Rpp9	10S-near *Rp1*	resistance to *Puccinia polysora* and *P. sorghi*			425
Rs1	?	rough sheath: extreme ligule disorganization			204
rs2	1-near *as1*	rough sheath	S		204
Rs4	7- near *Hs1*	rough sheath (was *Rs*-1606*): leaf sheaths rough, vascular bundles enlarged			297
rt1	3S-near *cl1*	rootless: secondary roots few or absent	S	P	178
ruq		receptor of *Uq*			121
S		seed color component at *R1*: anthocyanin pigmentation in aleurone; (see also cms-S)			85 188 191 407
Sad1	10L-near *bf2*	shikimate dehydrogenase: electrophoretic mobility; plastidial; monomeric			435
sbd1	6L	sunburned (was *wxl*-2292*): sun-exposed leaves greyish-waxy			297
Sdw1	8	semi-dwarf plant (was *Sdw*-1592*): shortened internodes, erect leaves	S		25
Sdw2	3	semi-dwarf (was *D*-1991*): short plant, 1/3-1/2 normal height, with normal green erect leaves; does not respond to gibberellins; no anthers in ear; nitrosoguanidine-induced			290
se1	4L-near *dp1*	sugary-enhancer: high sugar content with *su1*; light yellow endosperm; freely wrinkled in Ill677a	S		115
sen1	3	soft endosperm (duplicate factor with *sen2*): endosperm soft, opaque			409
sen2	7	soft endosperm (duplicate factor with *sen*)			409
sen3	1	soft endosperm (duplicate factor with *sen1*): like *sen1*			409
sen4	?	soft endosperm (duplicate factor with *sen3*)			409
sen5	2	soft endosperm (duplicate factor with *sen6*): like *sen1*			409
sen6	5	soft endosperm (duplicate factor with *sen5*)			409
sft1	?	small flint type: ears on *sft1* plants produce only small flint endosperms; +/*sft1* ears are normal			84
Sg1	?	string cob: reduced pedicels		P	124
sh1	9S-29	shrunken: inflated endosperm collapses on drying, forming smoothly indented kernels; sucrose synthase-1 of endosperm (compare *Css1*); homotetramer; *BNL(SH1)*(Pst38 or Pvu55), *NPI15(SH1)*()	S	P	172

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
sh2	3L-149.2	shrunken: inflated, transparent, sweet kernels collapse on drying, becoming angular and brittle; endosperm ADPG pyrophosphorylase subunit (compare *bt2*); *BNL(SH2)*(1050)	S	P	238
sh4	5L	shrunken: collapsed, chalky endosperm	S		423
sh5	5-near *lu1*	shrunken: sides of kernel collapsed	S		403
si1	6L-20	silky (= *ms-si*): multiple silks in ear; sterile tassel with silks			118
sk1	2S-56	silkless ears: pistils abort, no silks	S		186
Sks1	2L-near *v4*	suppressor of sterility: pollen grains developing in presence of *Ms21* are defective and nonfunctional if *sks1*, normal if *Sks1*			218 367
sl1	7L-50	slashed leaves: leaves slit longitudinally by necrotic streaks	S		161
sm1	6L-59	salmon silks: silks salmon color with *P1-RR*, brown in *P1-WW*	S	P	4
Sn1	10L-near *R1*	scutellar node color: anthocyanin in coleoptile, nodes, auricle, leaf blade, etc. (compare *Lc1*)			131
Sod(2-2)	9	superoxide dismutase candidate: see *NPI463(SOD)*()			445
Sod1	?	superoxide dismutase: electrophoretic mobility; plastidial; Cu-Zn dimeric; intralocus hybrid bands occur			14
Sod2	7L-near *Bn1*	superoxide dismutase: electrophoretic mobility; cytosolic; Cu-Zn dimeric *NPI419(SOD)*()			47 48
Sod3	?	superoxide dismutase: electrophoretic mobility; mitochondrial; Mn tetrameric; intralocus hybrid bands occur			14
Sod4	1S-near *P1*	superoxide dismutase: electrophoretic mobility; cytosolic; Cu-Zn dimeric; intralocus hybrid bands occur; *NPI412(SOD)*()			14 446
Spc1	3L-near *ig1*	speckled (was *Spc*-1376*, *Les*-1376*): brown speckling on leaves and sheath at flowering; supporting tissues weak			300
spc2	1L	speckled (was *spc*-262A*): green seedling with light green speckles			299
spc3	3L	speckled (was *pg*-553C*): green seedling with dark and light green speckles			299
Spm		suppressor-mutator: autonomous transposable element (equivalent to *En*); regulates *dSpm* transposition and function at *a1-m1*, *a1-m2*, *bz1-m13*, etc.			254
spt1	2L	spotted (was *spt*-464*): pale green, weak seedlings with dark green spots	S		299
spt2	4S	spotted (was *pgspt*-1269A*): like *spt1*	S		299
sr1	1S-0	striate leaves: many white striations or stripes on leaves (A.M. Brunson, 1935, unpublished data)	S		107
sr2	10L-98	striate leaves: white stripes on blade and sheath of upper leaves	S	P	184
sr3	10S	striate leaves: virescent and striate to striped	S	P	135
sr4	6L	striate leaves (was *stp*-65A*): seedlings pale luteus, later leaves white-striped			295
Ss2		(see *Css1*)			
Ssu1	4L-near *c2*	ribulose bisphosphate carboxylase small subunit family: *BNL17.05(SSU)*(pC1), *RPA9B(SSU)*(pZmcRPA:SSU), *NPI331(SSU)*(), *UMC210B(SSU)*			44 446
Ssu2	2-near *ts1*	ribulose bisphosphate carboxylase small subunit family: *BNL(SSU)*(pC1), *NPI227(SSU)*(), *RPA9A(SSU)*(pZmcRPA:SSU), *UMC210A(SSU)*			446
st1	4S-43	sticky chromosome: small plant, striate leaves, pitted kernels resulting from sticky chromosomes; *st1-e* heightened by high temperature	S	P	19
stAc	10-near *bf2*	stabilized Activator (P. Chomet, unpublished): RFLP locus			
su1	4S-47	sugary: endosperm wrinkled and translucent when dry; sweet at milk stage; starch debranching enzyme I; *su1-am* sugary-amylaceous; *su1-st* recessive starchy (see Coe et al., 1988)	S	P	69
su2	6L-58	sugary: endosperm glassy, translucent, sometimes wrinkled	S		114
su3	?	sugary endosperm: endosperm glassy, smoother than *su1*			410
Sup1	?	suppressor: modifies *o2* kernels to semi-transparent			246
Sus1		(= *Css1*)			
sy1	?	yellow scutellum			396
T		reciprocal translocation: general symbol for exchange of parts between two nonhomologous chromosomes; (see also cms-T)	S	P	188 191
Ta1	?	transaminase (possibly = *Got1*): electrophoretic mobility; dimeric; intralocus hybrid bands occur			234
tb1	1L-near *Adh1*	teosinte branched: many tillers; ear branches tassel-like	S		42
td1	5-near *bt1*	thick tassel dwarf: (E.G. Anderson, unpublished)	S		
te1	3L	terminal ear: stalked ear appendages at tip; varying to infolded ears			248
tga1	4- near *su1*	teosinte glume architecture: glumes indurated, erect, long, boat-shaped; factor transferred from teosinte			80
Thc1	?	thiocarbamate sensitive: sensitive to Eradicane			322
tl1	?	tasselless			259
Tlr1	1L	tillered (was *Tlr*-1590*): extreme tillering	S		306
Tp1	7L-46	teopod: many tillers, narrow leaves, many small partially podded ears, tassel simple	S	P	227
Tp2	10L-48	teopod: like *Tp1*		P	320
tpe1	?	thin pericarp: reduced cell number in pericarp (from Coroica)			123
Tpi1	7L-59	triose phosphate isomerase: electrophoretic mobility; plastidial; dimeric; intra/interlocus hybrids occur with *Tpi2*			438

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
Tpi2	2L-100	triose phosphate isomerase: electrophoretic mobility; plastidial; dimeric; intra/interlocus hybrids occur with *Tpi1*			438
Tpi3	8L-near *Mdh1*	triose phosphate isomerase: electrophoretic mobility; cytosolic; dimeric; intra/interlocus hybrids occur with *Tpi4* & *Tpi5*; *NPI344(TPI)*()			438
Tpi4	3L-near *cl1*	triose phosphate isomerase: electrophoretic mobility; cytosolic; dimeric; intra/interlocus hybrids occur with *Tpi3* & *Tpi5*; *NPI345(TPI)*()			438
Tpi5	8L	triose phosphate isomerase: electrophoretic mobility; cytosolic; dimeric intra/interlocus hybrids occur with *Tpi3* & *Tpi4*			438
Tpi6	5L-near *Got2*	triose phosphate isomerase candidate: see *NPI346(TPI)*()			438 446
tpm1	?	thylakoid peptide modifier: dominant decrease in electrophoretic mobility			274
tr	2S	two-ranked ear: distichous vs. decussate phyllotaxy in ear axis; quantitative, one of a family of loci differentiating maize vs. teosinte			
trAc	1S-near *vp5*	transposed Activator sequence			45
tru1	?	tassels replace upper ears: upper ear branches tassel-like, tillers bear ears			379
ts1	2S-74	tassel seed: tassel pistillate and pendant; if removed, small ear with irregular kernel placement develops	S		100
ts2	1S-24	tassel seed: like *ts1*, but branches variably pistillate and staminate	S	P	100
ts4	3L-73	tassel seed: tassel compact silky mass, upright, with pistillate and staminate florets; ear silky and proliferated	S	P	324
Ts5	4S-34	tassel seed: tassel upright with scattered, short silks; branches mostly pistillate toward the base	S		105
Ts6	1L-158	tassel seed: tassel pistillate to mixed, compact; ear with irregular kernel placement	S	P	309
ts8		(= *si1*)			
Tu1	4L-82	tunicate: kernels enclosed in long glumes; tassel glumes large, coarse	S	P	63 64
Tub1	1L-near *Adh1*	alpha tubulin family: mRNA expressed primarily in roots; member of tandem repeat (see *Tub2*); *BNL17.04-TUB*(pUC9alpha-1)			44 277
Tub2	1L-near *Adh1*	alpha tubulin family: member of tandem repeat (see *Tub1*)			277
Tub3	?	alpha tubulin family: mRNA expressed in dividing cells			278
ub1	?	unbranched: tassel with one spike	S	P	305
ubi1	4L-near *dp1*	ubiquitin candidate: see *AGR1002A(UBI)*()			281
ubi2	5-near *bm1*	ubiquitin candidate: see *AGR1002B(UBI)*()			281
UCLA		Univ. Calif. Los Angeles: designator for loci defined by restriction fragment polymorphisms			
UCSD		Univ. Calif. San Diego: designator for loci defined by restriction fragment polymorphisms			
UFG		Univ. Florida Gainesville: designator for loci defined by restriction fragment polymorphisms			
Ufo1	10S-near *Rp1*	unstable factor for orange: anthers, silks, and most other plant parts orange with *P1-WR* or *P1-RR*; growth retarded			417
UMC		University of Missouri, Columbia: designator for loci defined by restriction fragment polymorphisms			
Uq		ubiquitous: controlling element mediating *a1-ruq*			121
v1	9L-63	virescent: yellowish white seedling, greens rapidly; low temperature accentuates	S	P	74
v2	5L-107	virescent: like *v1*, but greens slowly; low temperature accentuates	S	P	98
v3	5L-45	virescent: light yellow seedling, greens rapidly; low temperature accentuates	S	P	74
v4	2L-83	virescent: like *v2*	S	P	74
v5	7S-24	virescent: like *v1*, but older leaves have white stripes	S	P	74
v8	4L-near *Tu1*	virescent: like *v2*; lethal	S		75
v12	5L-near *ys1*	virescent: like *v3*	S		325
v13	?	virescent: first leaf with green tip; greens slowly			325
v16	8L-14	virescent: like *v2*	S		325
v17	4	virescent: like *v1*, but greening from base to tip	S		325
v18	10	virescent: like *v1*	S		325
v19		(= *g2*)			
v21	8L	virescent (was *v*-25*, *v*-A552*): grainy virescent, greening from tips and margins inward	S		22
v22	1L-near *an1*	virescent (was *v*-8983*): like *v1* (E.G. Anderson, unpublished)	S		
v23	4-near *su1*	virescent (was *v*-8914*): like *v1* (E.G. Anderson, unpublished)			
v24	2L	virescent (was *v*-424*): like *v1*	S		299
v25	1S	virescent (was *v*-17*): greenish white seedling; greens from base upward	S		299
v26	2S	virescent (was *v*-453*): yellowish white seedling with green leaf tip and midrib	S		299
v27	7L	virescent (was *v*-590A*): like *v1*			299
v28	9S-near *yg2*	virescent (was *v*-27*, *v*-585*): like *v1*	S		299
v29	10L	virescent (was *v*-418*): grainy virescent			299
v30	9L-87	virescent (was *v*-8587*): like *v1*			59
v31	9S-near *yg2*	virescent (was *v*-828*): grainy, light green seedling; small green plant with longitudinal white stripes			157
va1	7L-near *ij1*	variable sterile: variable male and female fertility; cytokinesis fails in anaphase I	S		17

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
Vg1	1L-85	vestigial glume: glumes very small, cob and anthers exposed; liguleless leaves	S	P	399
vp1	3L-near *ts4*	viviparous: embryo fails to become dormant, viable if transplanted; some alleles dormant; chlorophyll and carotenoids unaffected; anthocyanins in aleurone suppressed; *BNL-VP1*(pVPM1B)	S		111
vp2	5S-38	viviparous: embryo fails to become dormant; white endosperm, white seedling; anthocyanins unaffected	S	P	111
vp5	1S-1	viviparous: like *vp2*	S	P	344
vp7		(= *ps1*)			
vp8	1L-154	viviparous: embryo fails to become dormant; chlorophyll and carotenoids unaffected; small, pointed-leaf seedlings	S		345
vp9	7S-25	viviparous (also known as *y7*): like *vp2*; *vp9-4889* dormant, pale endosperm, pale green seedling	S	P	345
vp10	?	viviparous(was *vp*-86GN5, vp*-2048*): yellow endosperm, colored aleurone, green seedlings, adherent			391
vsi1	?	variable short internodes: clusters of 2-4 short internodes, predominantly at base of plant but varies in location; temperature sensitive			87
Vsr1	10L	virescent striped (was *Vsr*-1446*): virescent seedling; greens to white and yellow striped plant	S		306
w1	6L-near *w14*	white: white seedling (yellow with *l1*); plastid transcripts variously aberrant	S		96 97 224
w2	10L-80	white: white seedling (yellow with *l1*); endosperm pitted and spotted (allele *dek21*); plastid DNA content decreased	S		226
w3	2L-111	white: like *vp2*; *w3-8686* pale endosperm, pale green seedling in dim light	S	P	226
w11	9S-54	white: like *w1*	S		75
w14	6L-78	white (was *w*-8657*): like *w1*	S		71
w15	6L-13	white (was *w*-8896*): like *w1*; fails to convert protochlorophyllide to chlorophyllide	S		71
w16	7S-near *vp9*	white: like *w1*			279
w17	7S-near *Hs1*	white: like *w1*			279
w18	1L	white seedling (was *w*-495A*, allelic to *w*-571C*): like *w1*	S		295
Wc1	9L-107	white cap: kernel with pale yellow endosperm (pearly white with *y1*), emphasized in soft-starch crowns	S		208
wd1	9S-near *yg2*	white deficiency: white seedling, deficiency for distal half of first chromomere of short arm (for alleles, see Coe et al., 1988)	S	P	252
wgs1	5L	white green sectors (was *sct*-206B*): white seedling with green sectors			299
whp1	2L-near *Ch1*	white pollen: duplicate factor with *c2* for pollen color and for anthocyanins; chalcone synthase; *BNL17.03-WHP*(pC2-c46)	S		61
wi1	6L-near *y1*	wilted: chronic wilting, leaves not as cool as normal; delayed differentiation of metaxylem vessels	S		330
Wi2	3	wilted: top leaves wilt under moisture/temperature stress			296
Wi3	?	wilted: like *Wi2*			297
wlu1	3L	white luteus (was *wl*-28*): pale yellow seedling; lethal			299
wlu2	7L	white luteus (was *wl*-543A*): like *wlu1*	S		299
wlu3	8L	white luteus (was *wl*-203A*): like *wlu1*			299
wlu4	9L	white luteus (was *wl*-41A*): like *wlu1*	S		299
wlu5	1L-near *br1*	white luteus (was *wl*-266A*): like *wlu1*	S		295
Wrk1	3S-62	wrinkled kernel (was *Wr*-1020*): kernels small and wrinkled	S		306
Wrp1	?	wrinkled plant: dominant dwarf, leaves and culm longitudinally corrugated; dosage effect			27 296
ws1	?	white sheath: light yellow leaf sheaths; duplicate factor with *ws2*			198
ws2	?	white sheath: see *ws1*			198
ws3	2S-0	white sheath: white leaf sheath, culm, husks	S	P	337
Ws4	1S	white sheath (was Pale green-1589): seedlings and plants lighter green in sheaths			296
wsp		weak streaked plant: maternally inherited reduced plants			36
wt1	2S-60	white tip: tip of first leaf white and blunt	S		404
wt2	4S	white tip (was *cb*-10*): seedling with white leaf tip and crossbands on first 2 leaves	S		299
wx1	9S-56	waxy: amylopectin (stained red by iodine) replaces amylose (blue staining) in endosperm and pollen; starch-granule-bound NDP-starch glucosyl transferase (for alleles, see Coe et al., 1988); *BNL-WX1*(pBF225), *UMC25-WX1*(pBF225), *NPI16-WX1*()	S	P	62
wyg1	7L-near *ra1*	white yellow green seedling			279
y1	6L-17	white: reduced carotenoid pigments in endosperm; some alleles affect chlorophyll in seedlings (e.g. *y1-8549*; see Coe et al., 1988)	S	P	69
y3	2S-near *al1*	pale yellow (compare *al1*)	S		319
y7		(= *vp9-y7*)			
y8	7S-18	pale yellow: pale endosperm	S		179
y9	10S-27	pale yellow: pale endosperm, slightly viviparous; green to pale green seedlings and plants	S		350
y10	3L	pale yellow (was *w*-7748*): pale endosperm; white seedling, lethal	S		346
y11	?	pale yellow: pale endosperm; green seedling	S		402

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
y12	?	pale yellow: like *y11*	S		402
yd2	3L-near *lg2*	yellow dwarf	S		349
yg1	5L-near *v2*	yellow-green: yellow-green seedling and plant	S		109
yg2	9S-7	yellow-green: like *yg1* (for alleles, see Coe et al., 1988)	S	P	177
YNH		Yale New Haven: designator for loci defined by restriction fragment polymorphisms			
ys1	5L-75	yellow stripe: yellow tissue between leaf veins, reflects iron deficiency symptoms	S	P	15
ys2	1S	yellow stripe: yellow tissue between leaf veins			328
ys3	3L-near *gl6*	yellow stripe: like *ys1*	S		444
Ysk1	4-near *su1*	yellow streaked (was *Ysk*-844*): longitudinal yellow streaks in top 3rd of mature leaves	S		300
z1		(= *vp9-z* = *y7-z*)			
zb1	?	zebra crossbands: yellowish crossbands on older leaves			73
zb2	?	zebra crossbands: crossbands on seedling leaves			415
zb3	5L-near *v2*	zebra crossbands: yellowish crossbands on older leaves (M. Demerec, 1935, unpublished data)	S		107
zb4	1S-19	zebra crossbands: regularly spaced crossbands on earlier leaves; enhanced by cool temperatures	S	P	159
zb6	4-60	zebra crossbands: regularly spaced crossbands on earlier leaves; enhanced by cool temperatures	S		162
zb7	1L-near *Adh1*	zebra crossbands (was *zb*-101*): lighter green crossbands on seedlings; glossy	S		299
Zb8	9-near *l7*	zebra crossbands (was *Atc1, Cb*-1443*): yellow-green crossbands on older leaves; strong anthocyanin expression in leaf tip and blade	S		300 306
zein		(see Zp, *ZpB36, ZpL, Zpr, Zps*)			
Zer		Zapalote Chico earworm resistance: designator for earworm resistance factors from Zapalote Chico			271
zn1	10L-29	zebra necrotic: necrotic tissue appears between veins in transverse leaf bands on half-grown or older plants	S	P	170
zn2	?	zebra necrotic: like *zn1*			134
Zp		zein polypeptide: designator for loci determining zein polypeptides			392 393
ZpB36	7-near *ra1*	zein family: *BNL(B36)*(B36)			46
zpg1	?	zebra-stripe pale green			83
ZpL1	4S-near *Ga1*	zein polypeptides: *Zp1La - Zp1Lf* complex			443
ZpL2a	4L-near *orp1*	zein polypeptides			443
ZpL2b	7S-near *o2*	zein polypeptides			443
ZpL3a	4L-near *orp1*	zein polypeptides			443
Zpr10/(22)	4S-20	zein-protein regulator: elevation of 10kD zein			24
Zps10/(22)	9-near *wx1*	zein-protein structural gene: 10kD zein; RFLP (probe 10kZ-1)			24

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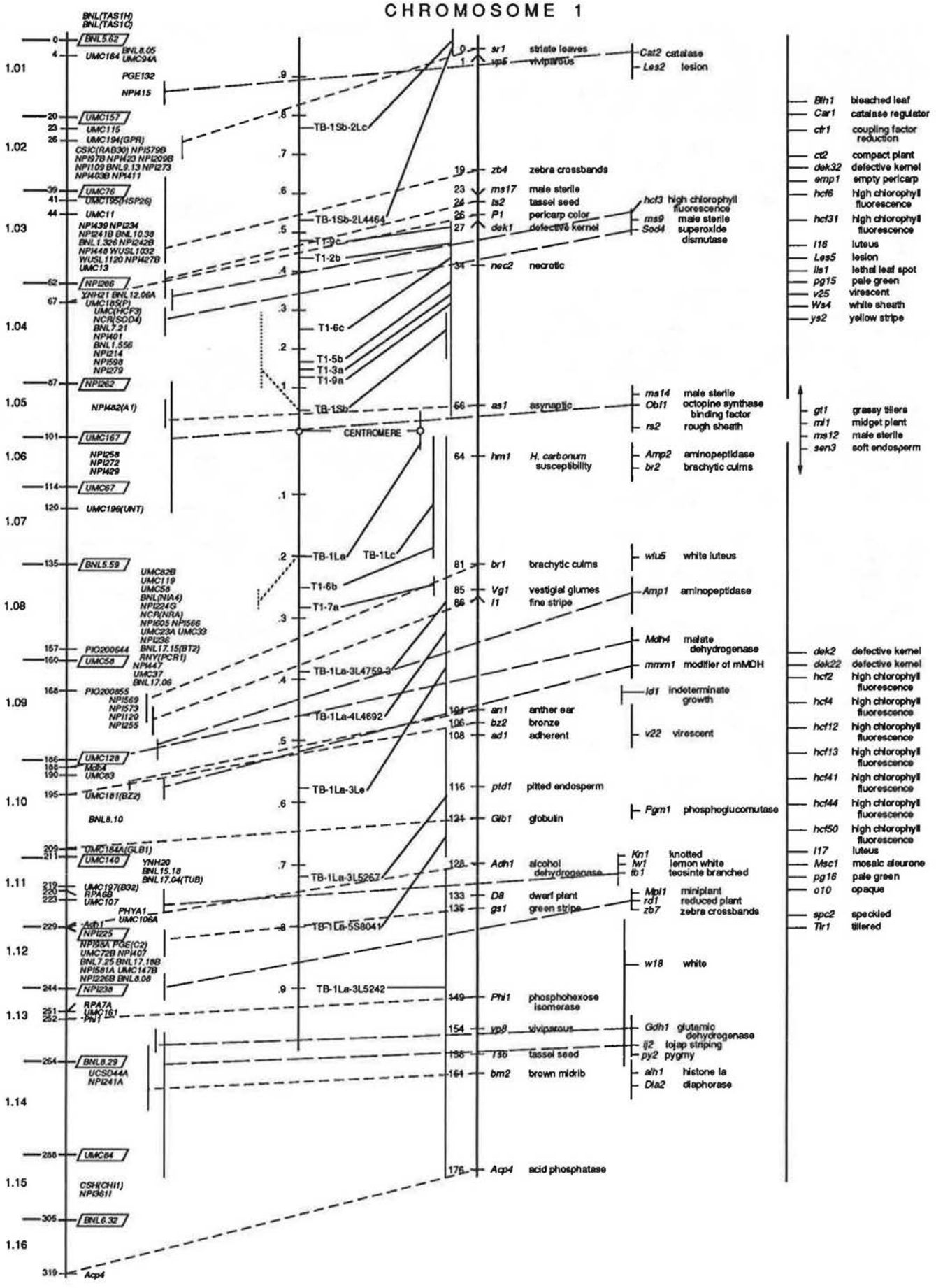
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UMC115
UMC194(GPR)
CSIC(RAB30) NPI579B
NPI97B NPI423 NPI209B
NPI109 BNL9.13 NPI273
NPI403B NPI411
UMC76
UMC195(HSP26)
UMC11
NPI439 NPI234
NPI241B BNL10.38
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NPI448 WUSL1032
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UMC13
NPI266
YNH21 BNL12.06A
UMC185(P)
UMC(HCF3)
NCR(SOD4)
BNL7.21
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BNL1.556
NPI214
NPI598
NPI279
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NPI482(A1)
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UMC67
UMC199(UNT)
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BNL(NIA4)
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UMC23A UMC39
NPI236
PIO200644
BNL17.15(BT2)
RNY(PCR1)
UMC58
NPI447
UMC37
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NPI573
NPI120
NPI255
UMC128
Mdh4
UMC83
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UMC140
YNH20
BNL15.18
BNL17.04(TUB)
UMC197(B32)
RPA6B
UMC107
PHYA1
UMC106A
Adh1
NPI225
NPI93A PGE(C2)
UMC72B NPI407
BNL7.25 BNL17.18B
NPI581A UMC147B
NPI226B BNL8.08
NPI238
RPA7A
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CSH(CHI1)
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Acp4
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T1-9c
T1-2b
T1-6c
T1-5b
T1-3a
T1-9a
TB-1Sb
CENTROMERE
TB-1La
TB-1Lc
T1-6b
T1-7a
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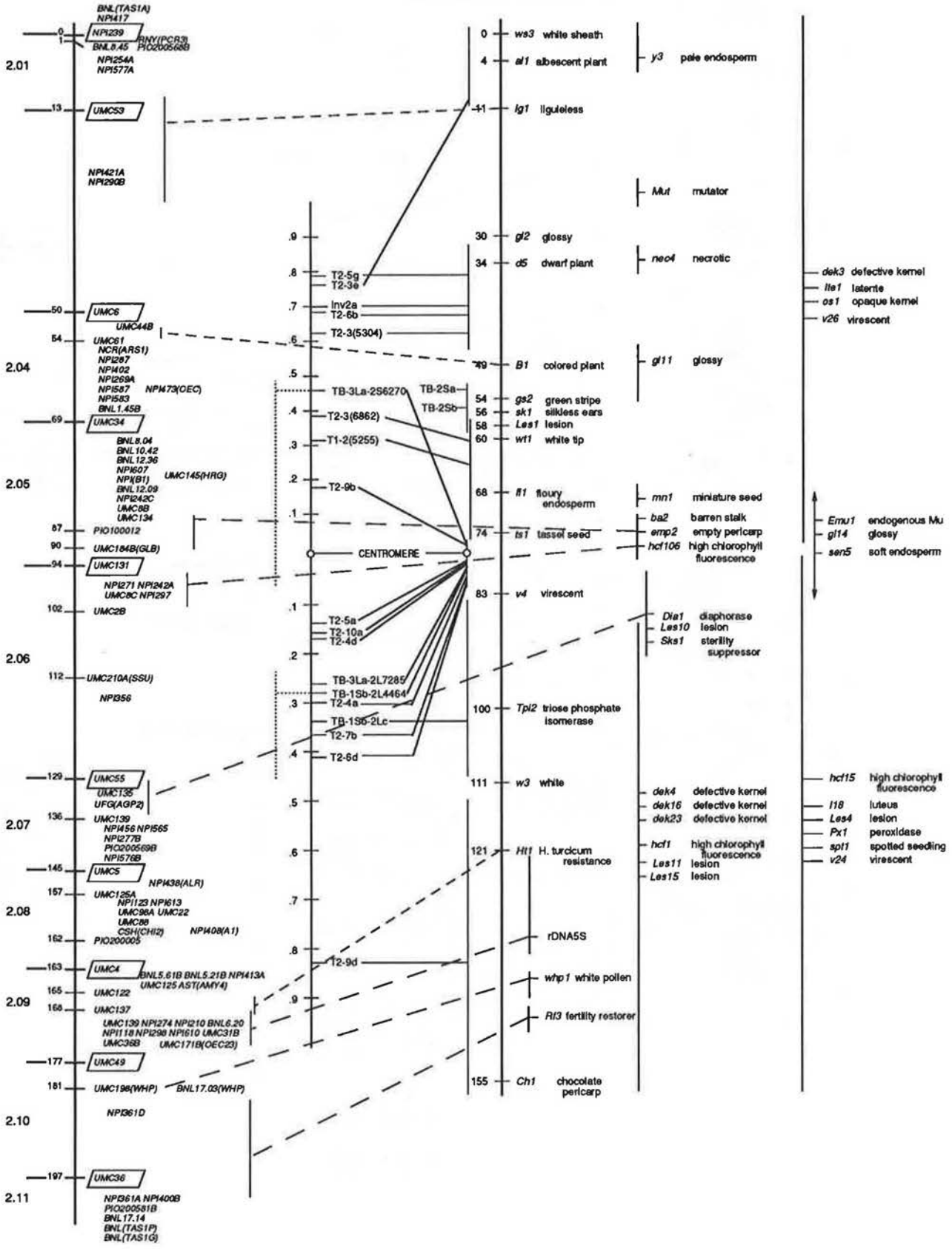
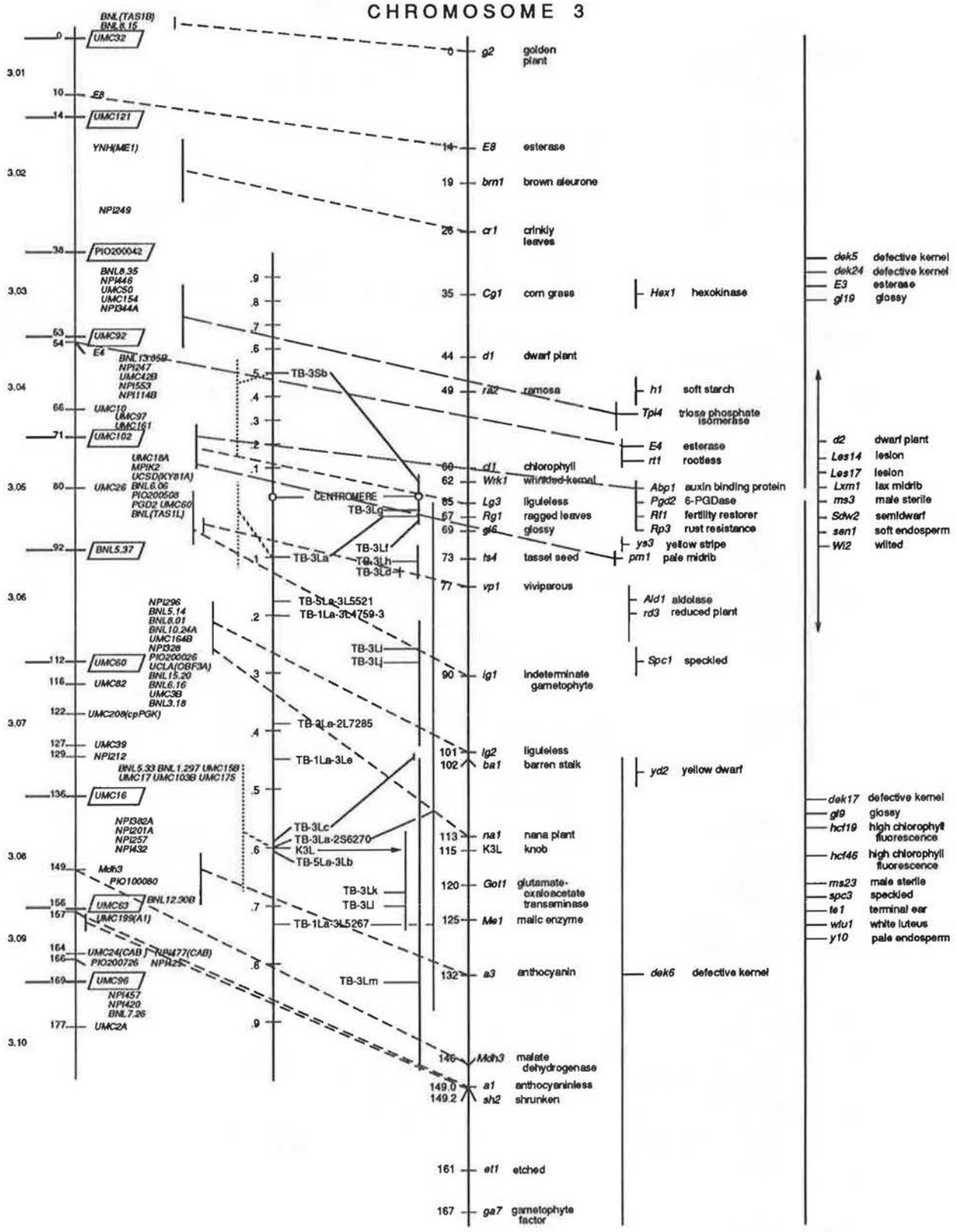
TB-1La-4L4692
TB-1La-3Le
TB-1La-3L5267
TB-1La-5S8041
TB-1La-3L5242
sr1 striate leaves
vp5 viviparous
zb4 zebra crossbands
ms17 male sterile
ts2 tassel seed
P1 pericarp color
dek1 defective kernel
nec2 necrotic
as1 asynaptic
hm1 H. carbonum susceptibility
br1 brachytic culms
Vg1 vestigial glumes
I1 fine stripe
an1 anther ear
bz2 bronze
ad1 adherent
ptd1 pitted endosperm
Glb1 globulin
Adh1 alcohol dehydrogenase
D8 dwarf plant
gs1 green stripe
Phi1 phosphohexose isomerase
vp8 viviparous
Tb1 tassel seed
bm2 brown midrib
Acp4 acid phosphatase
Cat2 catalase
Les2 lesion
hcf3 high chlorophyll fluorescence
ms9 male sterile
Sod4 superoxide dismutase
ms14 male sterile
Obf1 octopine synthase binding factor
rs2 rough sheath
Amp2 aminopeptidase
br2 brachytic culms
wlu5 white luteus
Amp1 aminopeptidase
Mdh4 malate dehydrogenase
mmm1 modifier of mMDH
id1 indeterminate growth
v22 virescent
Pgm1 phosphoglucomutase
Kn1 knotted
lw1 lemon white
tb1 teosinte branched
Mpl1 miniplant
rd1 reduced plant
zb7 zebra crossbands
w18 white
Gdh1 glutamic dehydrogenase
lj2 lojap striping
py2 pygmy
alh1 histone la
Dia2 diaphorase
Blh1 bleached leaf
Car1 catalase regulator
cfr1 coupling factor reduction
ct2 compact plant
dek32 defective kernel
emp1 empty pericarp
hcf6 high chlorophyll fluorescence
hcf31 high chlorophyll fluorescence
l16 luteus
Les5 lesion
lls1 lethal leaf spot
pg15 pale green
v25 virescent
Ws4 white sheath
ys2 yellow stripe
gt1 grassy tillers
mi1 midget plant
ms12 male sterile
sen3 soft endosperm
dek2 defective kernel
dek22 defective kernel
hcf2 high chlorophyll fluorescence
hcf4 high chlorophyll fluorescence
hcf12 high chlorophyll fluorescence
hcf13 high chlorophyll fluorescence
hcf41 high chlorophyll fluorescence
hcf44 high chlorophyll fluorescence
hcf50 high chlorophyll fluorescence
l17 luteus
Msc1 mosaic aleurone
pg16 pale green
o10 opaque
spc2 speckled
Tlr1 tillered

CHROMOSOME 2

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E8
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YNH(ME1)
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NPI446
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UMC154
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UMC92
E4
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NPI247
UMC42B
NPI553
NPI114B
UMC10
UMC97
UMC161
UMC102
UMC18A
MPIK2
UCSD(KY81A)
UMC26
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BNL10.24A
UMC164B
NPI328
UMC60
PIO200026
UCLA(OBF3A)
UMC82
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BNL6.16
UMC38
BNL3.18
UMC208(cpPGK)
UMC39
NPI212
BNL5.33 BNL1.297 UMC158
UMC17 UMC103B UMC175
UMC16
NPI382A
NPI201A
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NPI432
Mdh3
PIO100080
UMC63
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UMC199(A1)
UMC24(CAB)
RBM77(CAB)
PIO200726
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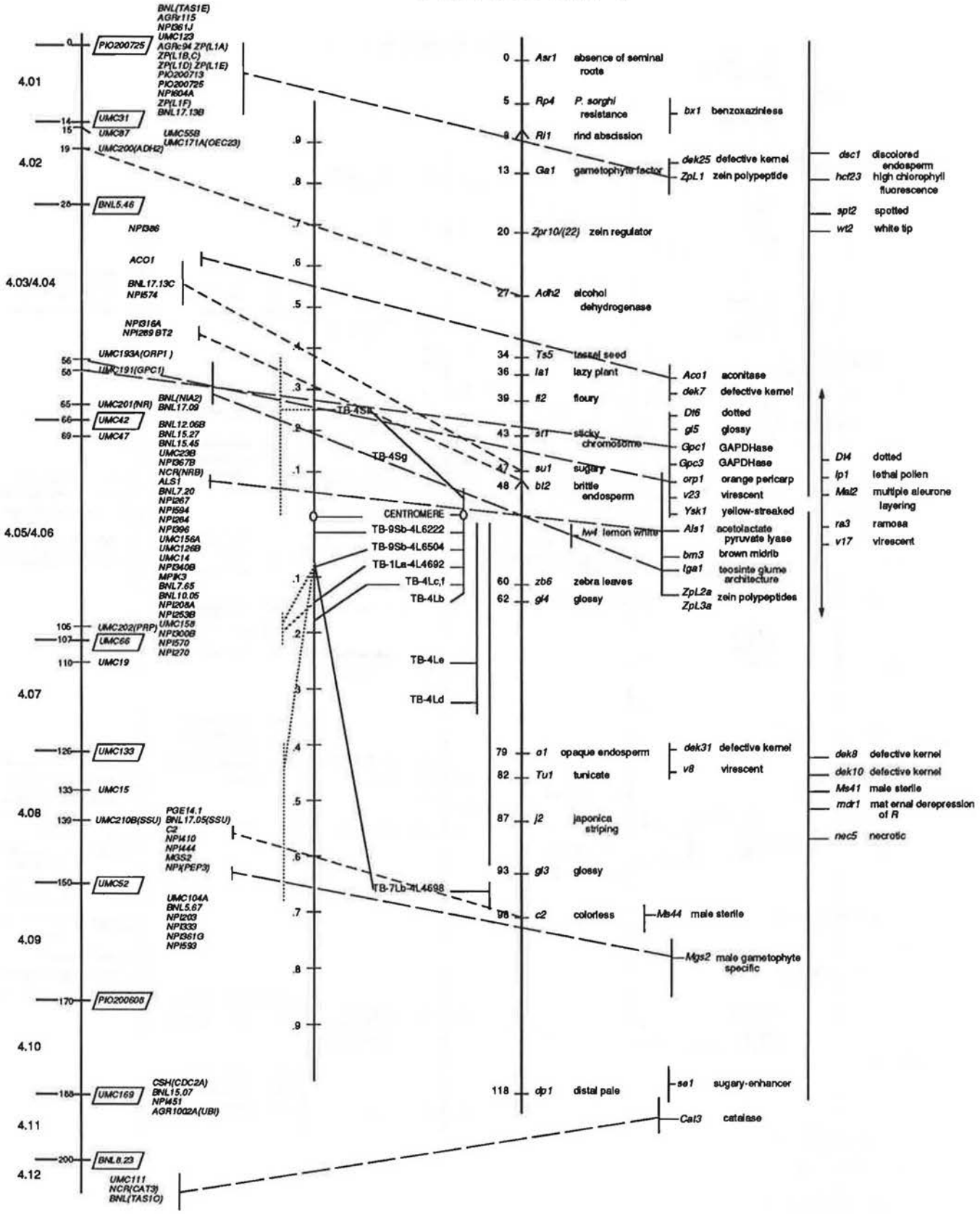
UMC96
NPI457
NPI420
BNL7.26
UMC2A
TB-3Sb
CENTROMERE
TB-3Lg
TB-3Lf
TB-3La
TB-3Lh
TB-3Ld
TB-3La-3L5521
TB-1La-3L4759-3
TB-3Li
TB-3Lj
TB-3La-2L7285
TB-1La-3Le
TB-3Lc
TB-3La-2S6270
K3L
TB-5La-3Lb
TB-3Lk
TB-3Ll
TB-1La-3L5267
TB-3Lm
g2 golden plant
E8 esterase
brn1 brown aleurone
cr1 crinkly leaves
Cg1 corn grass
d1 dwarf plant
ra2 ramosa
cl1 chlorophyll
Wrk1 wrinkled kernel
Lg3 liguleless
Rg1 ragged leaves
gl6 glossy
ts4 tassel seed
vp1 viviparous
ig1 indeterminate gametophyte
lg2 liguleless
ba1 barren stalk
na1 nana plant
K3L knob
Got1 glutamate-oxaloacetate transaminase
Me1 malic enzyme
a3 anthocyanin
Mdh3 malate dehydrogenase
a1 anthocyaninless
sh2 shrunken
et1 etched
ga7 gametophyte factor
Hex1 hexokinase
h1 soft starch
Tpi4 triose phosphate isomerase
E4 esterase
rt1 rootless
Abp1 auxin binding protein
Pgd2 6-PGDase
Rf1 fertility restorer
Rp3 rust resistance
ys3 yellow stripe
pm1 pale midrib
Ald1 aldolase
rd3 reduced plant
Spc1 speckled
yd2 yellow dwarf
dek6 defective kernel
dek5 defective kernel
dek24 defective kernel
E3 esterase
gl19 glossy
d2 dwarf plant
Les14 lesion
Les17 lesion
Lxm1 lax midrib
ms3 male sterile
Sdw2 semidwarf
sen1 soft endosperm
Wl2 wilted
dek17 defective kernel
gl9 glossy
hcf19 high chlorophyll fluorescence
hcf46 high chlorophyll fluorescence
ms23 male sterile
spc3 speckled
te1 terminal ear
wlu1 white luteus
y10 pale endosperm

CHROMOSOME 4



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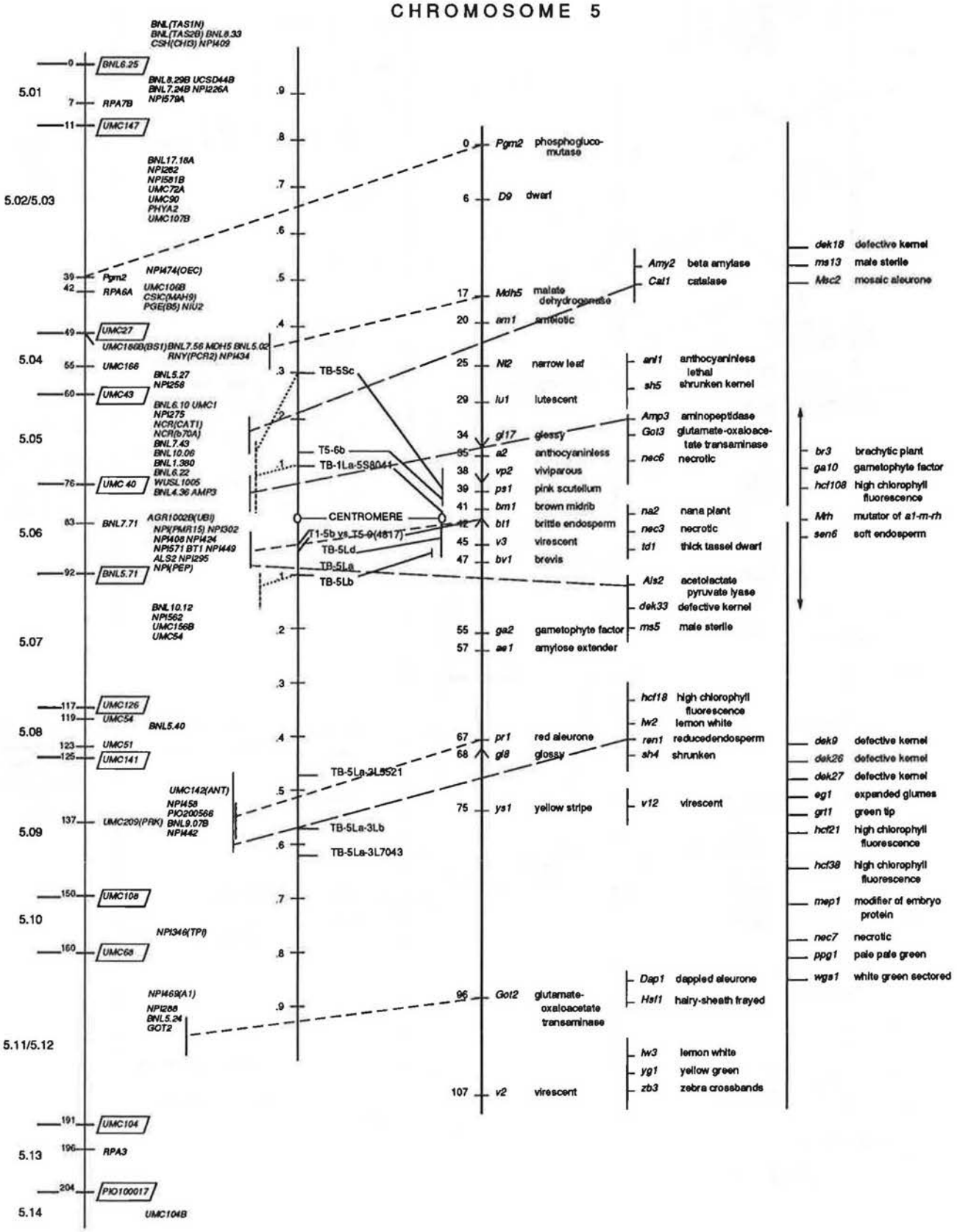

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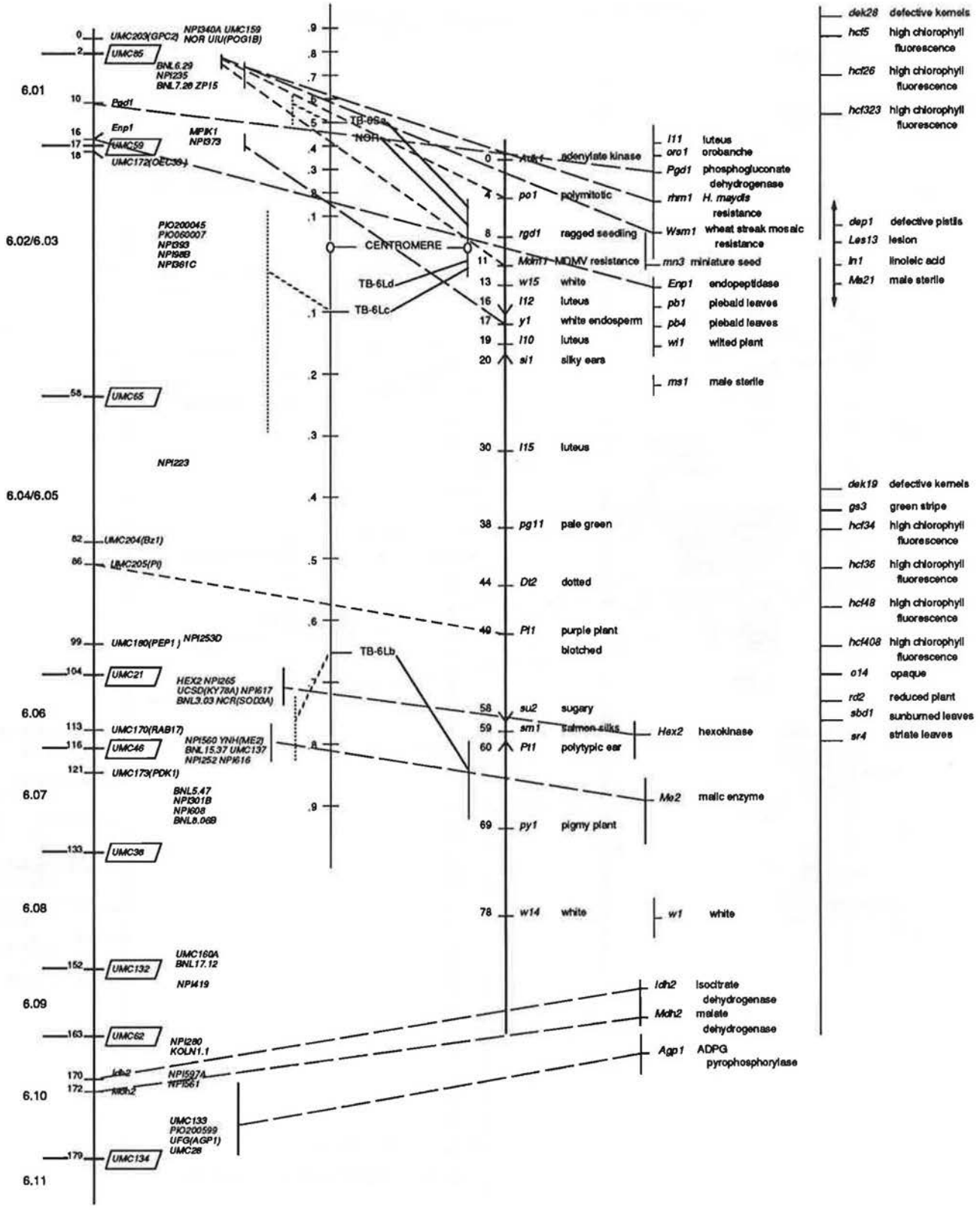

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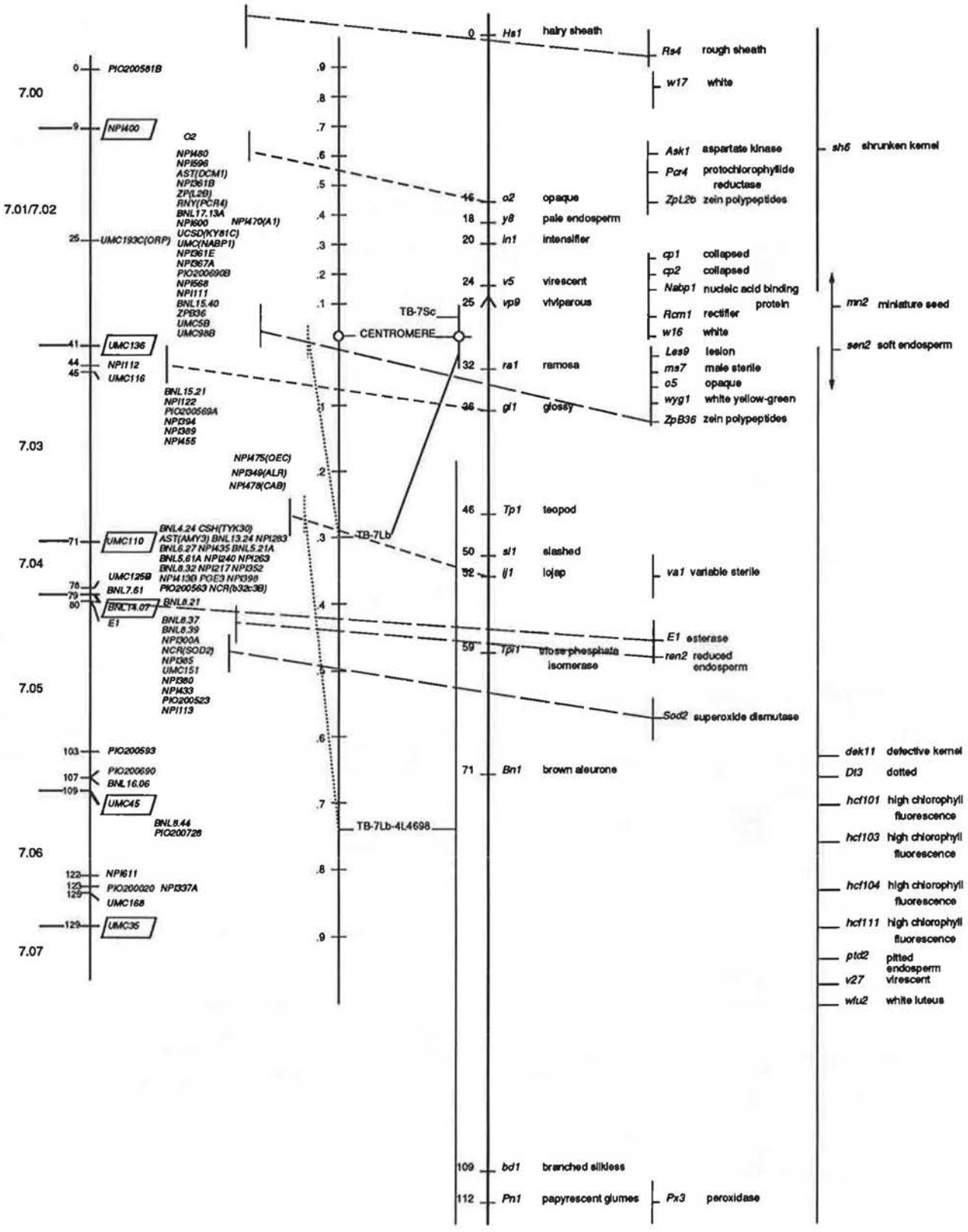

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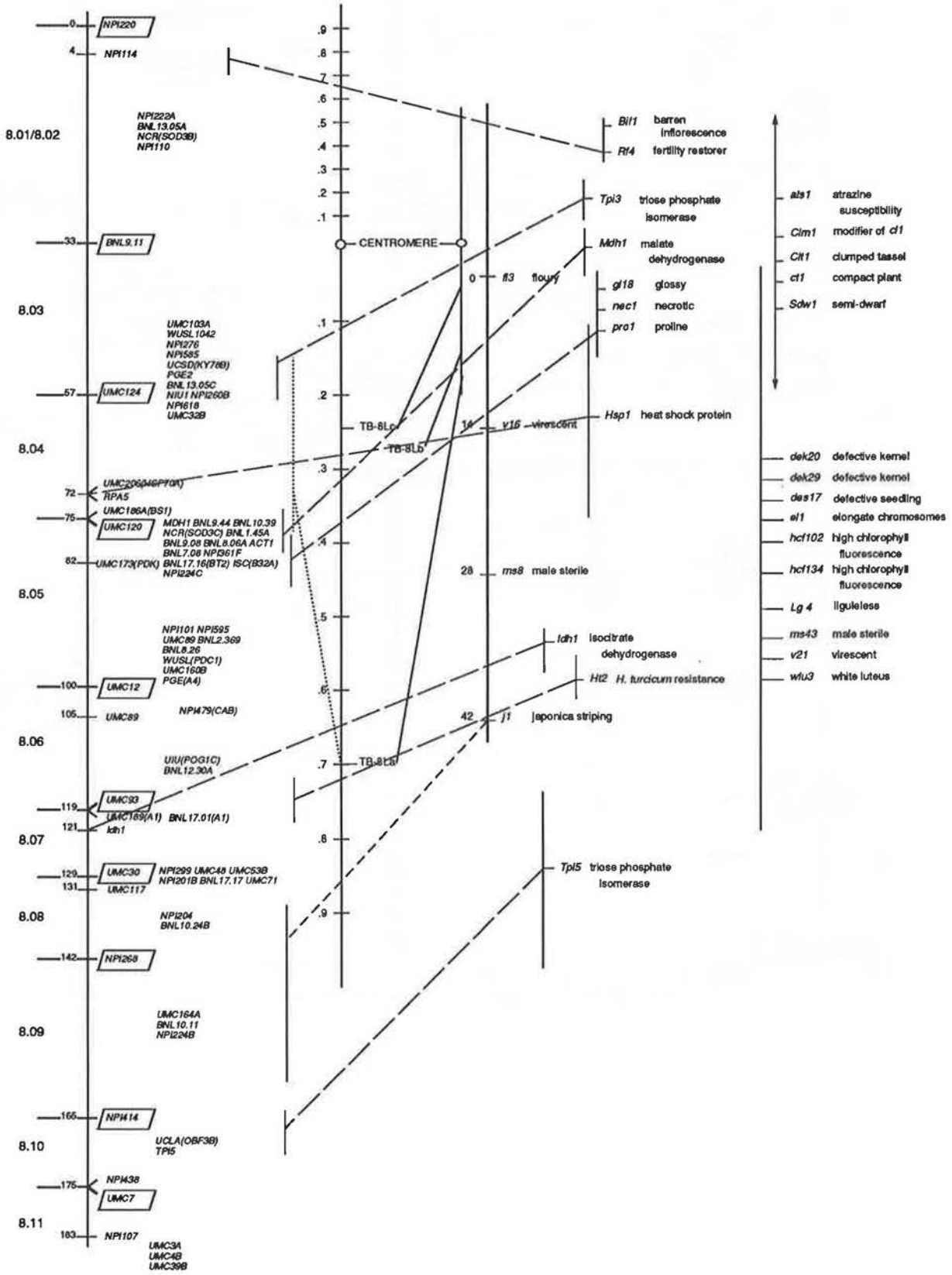

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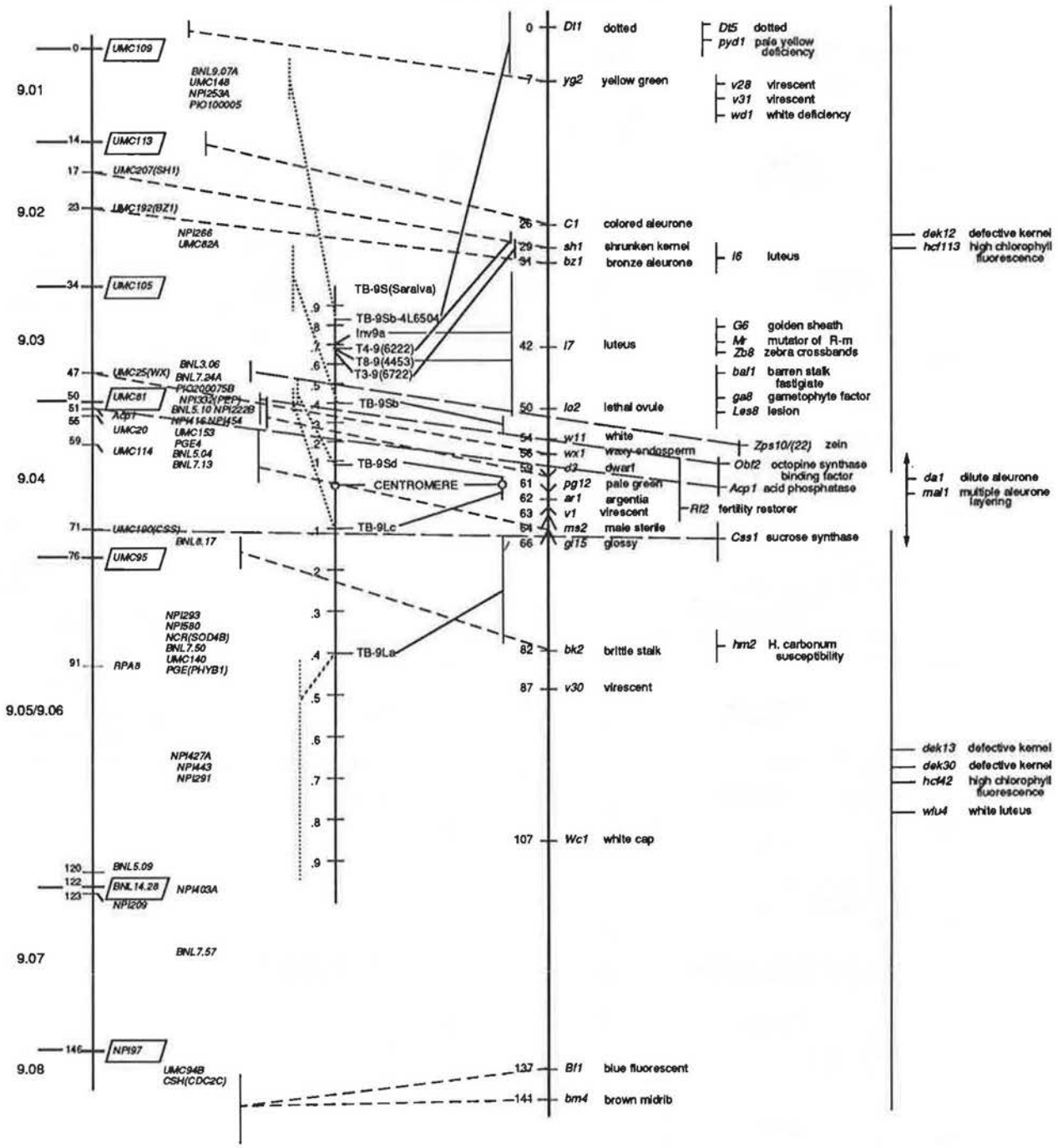

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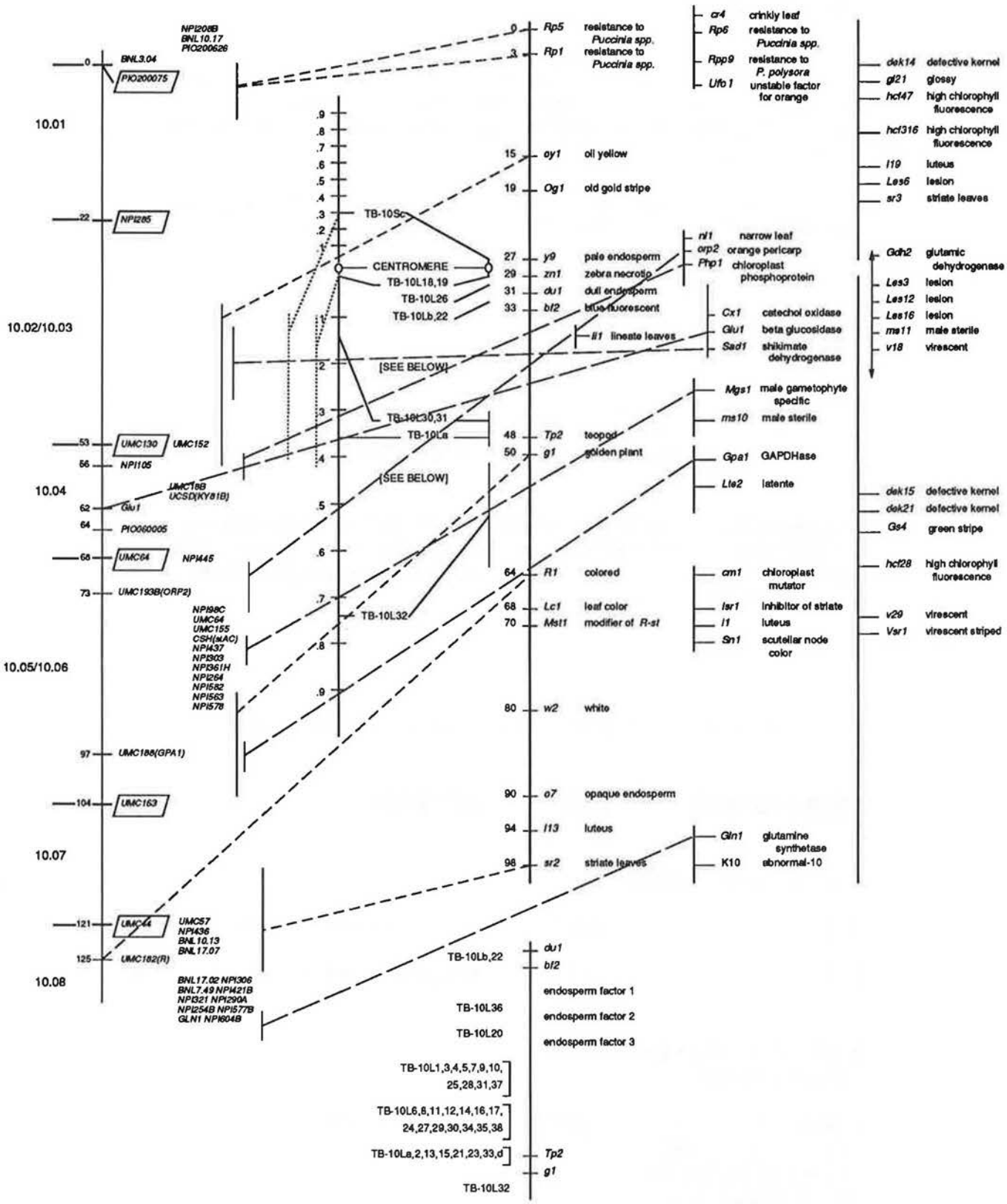

GENETIC MAP OF THE *ZEA MAYS* PLASTID CHROMOSOME

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Ames, IA 50011

The complete sequences of three maize plastid genes have been reported in the past year. Their locations are shown on the map below, and their gene products are briefly described in the following table. It is anticipated that within the year the sequence of the plastid chromosome will be completed by H. Kossel's lab (Universitat Frieberg).

See the 1987-1990 News Letters for descriptions of other sequenced genes: MNL 62:148; MNL 63:155; MNL 64 :164; MNL 65:164 .

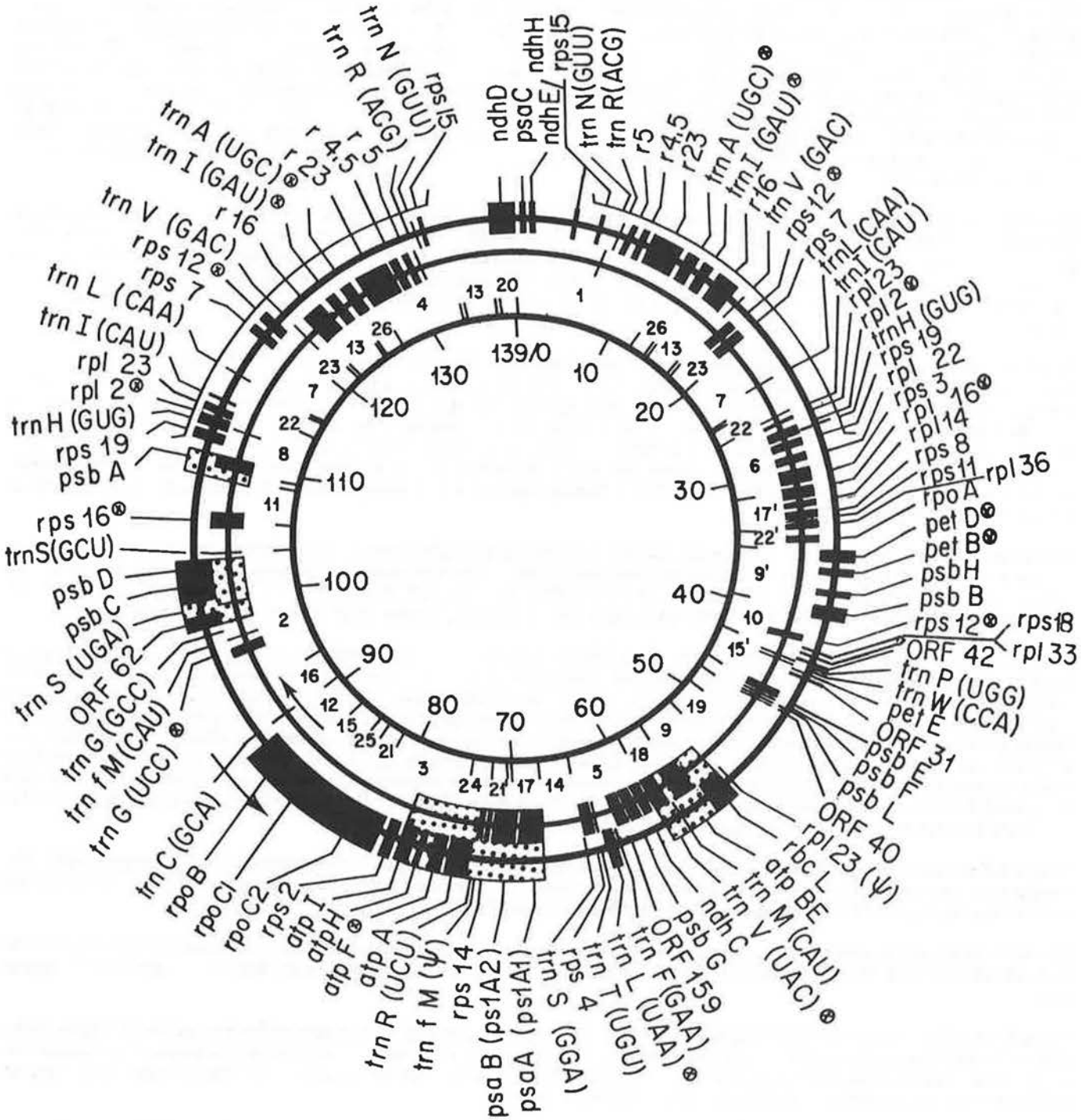
REFERENCES:

Maier, R.M. , I. Dory, G. Igloi and H. Kossel. 1990. The *ndh*H genes of gramminean plastomes are linked with the junctions between small single copy and inverted repeat regions. **Current Genet 18**: 245-250.

Weglohner, W., and A.R. Subramanian. 1991. A heptanucleotide repeat contributes to the unusual length of chloroplast ribosomal protein S18. Nucleotide sequence and map position of the *rpl*33-*rps*18 gene cluster in maize. **FEBS 279**: 193-197.

MAIZE PLASTID GENE SEQUENCES REPORTED IN 1991

GENE PRODUCT	GENE	REFERENCE
70S Ribosomal Proteins:		
S18	*rps*18	Weglohner and Subramanian,1991
L33	*rpl*33	Weglohner and Subramanian, 1991
NADH dehydrogenase subunit proteins:		
Homolog of the nuclear DNA-encoded 49-kD subunit of the bovine mitochondrial NADH dehydrogenase complex	*ndh*H	Maier et al., 1990


ndhD
psaC
ndhE
ndhH
rps15
trn N(GUU)
trn R(ACG)
r5
r4.5
r23
trn A (UGC) ®
trn I (GAU) ®
r16
trn V (GAC)
rps12 ®
rps 7
trnL (CAA)
trnI (CAU)
rpl23
rpl 2®
trnH (GUG)
rps 19
rpl 22
rps 3
rpl 16®
rpl 14
rps 8
rps11
rpl 36
rpo A
pet D®
pet B®
psbH
psb B
rps 12®
rps18
ORF 42
rpl 33
trn P (UGG)
trn W (CCA)
pet E
ORF 31
psb L
ORF 40
rpl 23 (ψ)
rbc L
atp BE
trn M (CAU)
trn V (UAC) ®
ndh C
psb G
ORF 159
trn F(GAA)
trn L (UAA) ®
trn T (UGU)
rps 4
trn S (GGA)
psa A (ps1A1)
psa B (ps1A2)
rps 14
trn f M (ψ)
trn R (UCU)
atp A
atpF ®
atpH ®
atp I
rps 2
rpo C2
rpo Cl
rpo B
trn C (GCA)
trn G (UCC) ®
trn f M (CAU)
trn G (GCC)
ORF 62
trn S (UGA)
psb C
psb D
trnS(GCU)
rps 16®
psb A
rps 19
trn H (GUG)
rpl 2®
rpl 23
trn I (CAU)
trn L (CAA)
rps 7
rps 12®
trn V (GAC)
r16
trn I (GAU) ®
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r4.5
r5
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trn N (GUU)
rps15
1
10
20
30
40
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100
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120
130
139/0

VI. Maizedb: MAIZE GENOME DATABASE

One of the key developments in the Plant Genome Initiative is the design and implementation of a database and network system for genetic data, analysis of data, and linked access to sequences, clones, biosynthetic pathways, and the like, across species boundaries. In addition to its grants through the Competitive Grants Program of Cooperative States Research Service, the Initiative supports database development through the Agricultural Research Service; both are branches of the U.S. Dept. of Agriculture. A Plant Genome Database is being derived by "Prototype Developers" working in concert, for maize, soybean, wheat, forest trees, and *Arabidopsis*. The structure will be inclusive of higher plant data and is to be focussed at the National Agricultural Library (NAL). The Newsletter "Probe", available from Plant Genome Data and Information Center, USDA - National Agricultural Library, 10301 Baltimore Blvd. Room 1402, Beltsville, MD 20705-2351, offers coverage of current issues, descriptions of developing programs, and updates on plant genome activities. Expectation is that on-line access to plant genome databases will be available by 1993 in their first implementations. The Maize Genome Database is a developing prototype in this network. This is a report on the development and progress for Maizedb.

In January of 1991 Cooperators (maize geneticists, breeders and molecular biologists) were surveyed for help in defining essential components and structures for the database, eliciting many suggestions, creative ideas, offers of data, and interest in contributing to and participating in the effort. Substantial further input on how to proceed was obtained as a result of presentations of descriptions and potential at the annual Maize Genetics Conference in March, 1991, and in this Newsletter (MNL 65:54). These responses made clear that interest was strong among respondents, that hopes were for the database to be as comprehensive as possible, and that integration with other genome databases was a much needed resource.

In April,1991, an Advisory Group of 18 scientists derived a conceptual framework and a beginning plan for the prototype effort. Consensus has been particularly strong to develop a useful and effective prototype as promptly as might be feasible. The Advisory also defined subcommittees and responsibilities for Nomenclature and Standards, User Needs and Quality Control, Clone Banks, Quantitative Characters and Descriptors, Germplasm Characterization, Prototype Development, and other high-priority data compilation and collection. Evaluations of database projects, through visits by one to several members of the group to Yale University (*E. coli*), Livermore National Laboratory (human), Welch Library at Johns Hopkins University (human GDB), DuPont (soybean, *Arabidopsis*, maize), University of Missouri and Washington University (nematodes), Agrigenetics (maize and other crops), and Lawrence Berkeley Laboratory (human), especially guided conceptualization of canonical structures and of planning for User Needs and Quality Control.

The Advisory, which provides continuing guidance and evaluations, includes geneticists from universities, government, and industry, each of whom enthusiastically agreed to help. The membership includes some scientists with extensive experience in developing large relational databases. The members of the Advisory Group are Bill Beavis, Mary Berlyn, Peter Bretting, Ben Burr, Vicki Chandler, Jim Coors, Neil Cowen, Larry Darrah, Tim Helentjaris, Dave Hoisington, Kendall Lamkey, Oliver Nelson, Jean Romero-Severson, Margaret Smith, Charles Stuber, and Scott Tingey.

A Working Group (Ed Coe, Mary Berlyn, Stan Letovsky, Mary Polacco, Marty Sachs, Denis Hancock) began in June, 1991, to build a prototype based on the *E. coli* design developed by Berlyn with Letovsky. Because the power and the specificity requirements of a relational database require highly structured planning for an interconnecting design, intensive communication between the Yale developers and the Columbia group ensued, which defined and developed conceptualization and refinements for the structure. The initial implementation of the Maizedb prototype was installed in December. Stock data were then imported after a combination of parsing and of manual revision of the stocklist to systematize the information. Site (gene list) data were imported, and in-depth entries of alleles, phenotypes, and products were made in selected components toward evaluations of the implications of the design. The list of reciprocal translocations with breakpoints, and a one-year block of references, were incorporated in February. Refinements of the details and the extent of the data are continuing. The implementation was immediately effective, but most importantly was instructive toward debugging and toward further conceptualization on the design.

Designs and structures among the Prototype Developers are shared and planned in quarterly meetings organized by D. Bigwood of NAL. Rationales of the implementation designs of each (Yale & Missouri for maize; LBL & Ames & Albany for soybean, wheat, and pines; Harvard & Ohio State for *Arabidopsis*; NAL for the combined prototype) are developing interactively.

The Nomenclature and Standards Committee, chaired by Oliver Nelson, developed recommended revisions for nomenclature and standards that have been presented and discussed at the 1992 Maize Genetics Conference. The new standards will be distributed to Cooperators in the near future.

A compilation of the data and a combined map of RFLP markers in recombinant inbred lines is being done by Ben Burr and Tim Helentjaris; similar data are in preparation for the Immortal F2 at Missouri, and other data from public and company sources are being assembled. An expanded set of recombinant inbreds is being typed by Charles Stuber. When completed, these materials will substantially increase the mapping-data resource and the ability of research workers to evaluate and to use markers and maps.

To provide a data baseline for Germplasm Characterization of selected production materials, priority characterization of 100 elite inbred lines for isozymes and DNA molecular (RFLP) probes is being carried out by Biogenetic Services; reproduction of seed of most of the 'typed' pedigrees has been completed at Missouri, toward placing these characterized strains in repository collections. Data from previous research in ARS and at NC State, with 22 isozymes on over 400 inbred lines, has been obtained and will soon be in the database. Pedigree data for inbred lines and other elite germplasm are expected to be available for incorporation in the near future.

Projects have been initiated to assemble, verify, and compile cytogenetic data and mutant data, to incorporate these data into systematic formats, and to carry out priority research on mapping of cytogenetic sites and mutants. This work is being carried out by Dave Weber at Illinois State University, and by Gerry Neuffer and colleagues at the University of Missouri.

Plans are under development, with the help of Al Kriz at Urbana, to develop a record of the Maize Genetics Cooperation Stock Center materials and to initiate computerized records for this operation.

A demonstration of the Maizedb Prototype was given at the Maize Genetics Conference in Asilomar, in March, 1992. Following up on comments and responses from the demonstration, and on a period of experience with the implementation before and after the demo, requirements have been defined for restructuring and will be carried through as part of the next phase of the implementation. Systematization of information (which will increase its accessibility to a much wider and more diverse research and development community), for genome analysis and manipulation and for resource and utilization requirements, is a conspicuous and desirable result (the fruit of less than a year of effort). The contrast with static files of decayable information, and with the comprehensive but casual organization of data of the past, stands out increasingly as the prototype is refined.

THE DESCENDANTS OF STAR DUST*

Here we are at Asilomar
Not far from Mt. Palomar
Where a mighty telescope
Looks toward the big bang
Where everything began
Earth and life sprang.
In trust we must
Descend from star dust
Along a DNA pathway.
If what they say is okay
We may uncork the time warp
On our biological evolution.
As an interdependent descendant,
We plead our need to be freed
From our fate to hate
So as not to terminate, eliminate, aggravate, or humiliate
But to investigate, communicate, adjudicate, and liberate
As well as cultivate, irrigate, pollinate, and domesticate.
Then our mortal succession
Will become immortal progression,
And democratic revolution
Will become cultural evolution.
You are me but from my past.
I am you but from your future.
Together we uncork the time warp
On our cultural evolution.
Together we see our future evolving from the past,
With the power of a peace that will forever last.
Our differences will become only raw material
For the cultural evolution of our human serial
And the biological evolution of our maize cereal,
As bright and just descendants of star dust,
We must adjust our thrust
Ablaze with praise that obeys
Our craze to raise maize.

Walton C. Galinat
12/6/91

*This is dedicated to everyone attending the Maize Genetics Conference at Asilomar, but especially to our Soviet colleagues as a vanguard of geneticists from the future.

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VIII. RECENT MAIZE PUBLICATIONS

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IX. SYMBOL INDEX

("r" refers to numbered references in the Recent Maize Publications section)

X. AUTHOR AND NAME INDEX

("r" refers to numbered references in the Recent Maize Publications section)
(* identifies articles authored in this Newsletter)

Hidden Glory in The Hole

Mike May

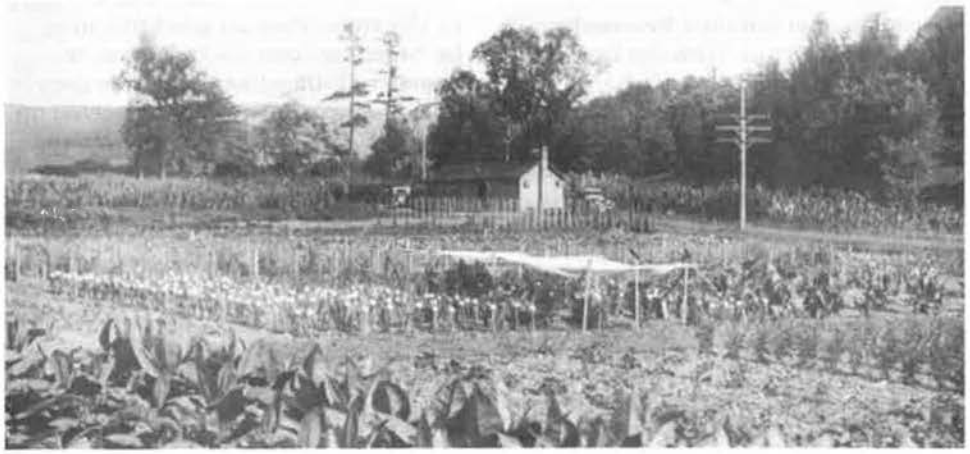
Some historical monuments flaunt their glory. Surrounded by signs and symbols, both Thomas Jefferson's Monticello in Virginia and Sigmund Freud's office in Vienna broadcast a clamor of significance. However, most historical sites remain obscure, largely forgotten by the passing years. Historical landmarks, some more alluring than others, cover the campus of Cornell University. One such site, "The Hole," should never be forgotten.

Plantations' headquarters building is at the northern edge of The Hole, a few acres of lowland surrounded by rising banks. Beyond a simple beauty, nothing significant roars from this plot of land. A small, flat field covers the center of The Hole. During spring and summer, Cornell's virologists plant botanical contestants in this field for battles against disease. By winter, only grassy paths break the bare earth.

A small shed guards the southern edge of The Hole, just before the ground rises to Tower Road. Stucco covers much of the shed, except for a small screened portion to the west. If you peek inside through the dusty windows, you see piles of stakes and labels used during the flowering months by the Plantations gardeners.

Most of the time The Hole seems peaceful, excepting an occasional car along Plantations Road. However, during the early 1900s excitement filled that little field and surrounded the small shed. The glory of discovery and the perspiration of success rang from The Hole—the home of Cornell's early corn geneticists.

Rollins Adams Emerson launched Cornell into an era of modern genetics through the use of corn (*Zea mays*). He came to the campus in 1914 as the chairman of the Department of Plant Breeding. Although trained in horticulture, he moved into genetics, where he first worked on garden beans. In one course Emerson used Indian corn to reveal the principles of Mendelian segregation. Corn soon became Emerson's preferred plant and he the



authority on its breeding.

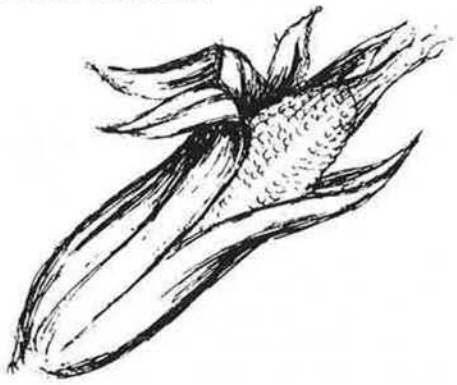
During the 1920s Cornell bred fields of corn and a band of celebrated corn geneticists. In those days Emerson's department excluded women graduate students, so it was from the Department of Botany that Barbara McClintock received her doctorate in 1927, though she worked specifically with corn. She remained at Cornell as an instructor, and her work sped toward the intersection of breeding and the structure of chromosomes.

Today we all know that chromosomes carry the genes that guide the development and maintenance of organisms. However, in the late 1920s this work had only begun, even in one of genetics' oldest standbys—the fruit fly, *Drosophila*. But McClintock looked beyond the present. She envisioned the possibility of correlating chromosomal morphology with specific traits in adult corn. This became known as the study of cytogenetics. When she found an unusually colored plant or seed, McClintock looked to the chromosomes for a novel bump or bend. Corn lent itself perfectly to this task. Years of breeding provided extensive information on corn. Furthermore, her cytological techniques revolutionized the visualization of the chromosomes. She saw them, the building blocks of genetics, in exacting detail.

By the end of the 1920s, Cornell housed a team of corn cytogeneticists. Marcus Rhoades and George Beadle came as graduate students to work on corn. Charles Burnham came from Wisconsin on a National Research Council Fellowship. With the breeding experience of Emerson and the cytological brilliance of McClintock, this group rode a wave of success.

With Emerson, Rhoades, Beadle, and Burnham on the team, this may appear as a predominantly masculine wave. It wasn't. Without doubt, McClintock's brilliance drove, if not dominated, the group. Marcus Rhoades described her role when he said, "I've known a lot of famous scientists. But the only one I thought really was a genius was McClintock."[1]

Moreover, another woman worked on this team. In the fall of 1929, Harriet Creighton came to the botany department as a graduate student. With no time wasted, Creighton joined this group of researchers. "By the end of the first day, Harriet Creighton had her entire graduate program organized—all according to McClintock's recommendations."[2]



The years from 1928 to 1935 became the Golden Age of corn cytogenetics.[3] And while these researchers at Cornell played key roles in this period, The Hole was their home.

Both Marcus Rhoades and Charles Burnham spoke to me about "The Garden"—the small field centered in The Hole. They all used this area for breeding corn. As Burnham remembered, they used The Hole for breeding because it was "protected on all sides by a high bank, an area not subject to the early fall frosts common in outlying fields."[4] Moreover, The

Hole offered some unexpected benefits. Burnham recalled that one summer when a water main broke in The Hole, the corn researchers built a dam to flood The Garden with water for future irrigation.

The small shed tucked into the southern end of The Hole became the local corn community center. It housed supplies, including the ear bags that were slipped over young shoots for protection from unknown pollen. The shed also served as a drying area. McClintock recalled that a coal stove with a hot-air blower dried the plants and seeds. That stove's chimney still peeks over the roof of the shed. Yet it was more than a simple supply shed. Burnham told me that on rainy days they ate their lunches in that small building. Those lunches, however, resembled seminars more than casual meals. Beadle once wrote that "lunches in the garden house during these periods of intense activity were events of great influence on students. It was on such occasions that the unprinted lore of corn genetics was transmitted."[5]

During those lunch meetings the small shed protected a group intellect rarely matched. They achieved success through hard work and persistence, not through luck. "It was accepted practice," Beadle wrote, "to work from dawn to dark seven days a week during the pollinating season."[6]

They reaped respect and knowledge from those long hours. In 1932 Cornell hosted the Sixth International Congress of Genetics, at which time a special "corn meeting" occurred. The participants decided to make Cornell a repository of breeding information for corn as well as a storehouse of seed stocks. Members of the corn meeting initiated the *Corn News Letter*. Printed on a mimeograph machine and edited by Rhoades, the newsletter summarized the latest findings and techniques. Seemingly suddenly, Cornell's corn research group served as the leader in corn cytogenetics.

A burst of compelling research papers certified Cornell's status as the home of corn cytogenetics. From 1931 to 1935 Rhoades published seven papers on the subject. In just three years, 1929 through 1931, McClintock published nine papers, and according to Evelyn Fox Keller "each was a major contribution to the field."[7]

By 1935 the Golden Age was over. That productive group of corn geneticists left The Hole. However, more years of prosperous research awaited those scientists.

Barbara McClintock found a new home at Cold Spring Harbor, Long Island, New York. She continued working with the cytogenetics of corn, as she does today. Her work with corn revealed transposable elements, the so-called jumping genes in chromosomes. In 1983 she received the Nobel Prize in physiology and medicine.

The late George Beadle's work progressed to molecular biology. He and Edward Tatum proposed the "one gene–one enzyme" hypothesis of molecular genetics. In 1958 he shared the Nobel Prize in physiology and medicine with Tatum and Joshua Lederberg. However, during his retirement Beadle returned to the biology of corn to study its evolutionary origins.

Both Marcus Rhoades and Charles Burnham remain active in science. Although they are retired, neither lost interest in corn over the years. Recently Burnham was drawn into an additional project: saving the American chestnut (*Castanea dentata*).

As a group, these researchers contributed as much to science as any other team during this century. One could easily argue that they contributed the most. Although disbanded by 1935, none of Cornell's early corn geneticists forgot those days in The Hole. Burnham and Rhoades still relate with excitement stories from those days. And years after leaving Cornell, McClintock still felt that it was home. Perhaps the days in The Hole changed the lives of those researchers—Beadle, Burnham, McClintock, Creighton, Rhoades, and Emerson—as much as they changed science.

When I stand in The Hole, next to the small shed, and look across The Garden, I can't see the plants presently fighting disease. The passing cars go unnoticed. When I look across that field, I see corn. The air still carries a hint of burning coal as I watch history in my mind. Glory remains in The Hole, if only we care to remember.

Mike May received his doctorate from Cornell University's Department of Neurobiology and Behavior in 1990. He is a free-lance writer, primarily on topics related to animals and medicine.

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The corn pioneers: In front, George Beadle; in the rear, L to R, Charles Burnham, Marcus Rhoades, Rollins Emerson, and Barbara McClintock.

Seeds Of Change exhibitions and programs have been made possible through the generous support of Xerox Corporation
and
the Smithsonian Institution Special Exhibition Fund, the National Endowment for the Humanities, The Potato Board, the National Corn Growers Association, Beneficial Management Corporation, AmeriFlora '92, ARCO Foundation, and the Potato Museum.

Four copies of a panel version of *Seeds Of Change*, cosponsored by the American Library Association and the Smithsonian Traveling Exhibition Service (SITES), will travel to public libraries in all fifty states and three territories of the United States. SITES is also circulating a copy to museums throughout the country.

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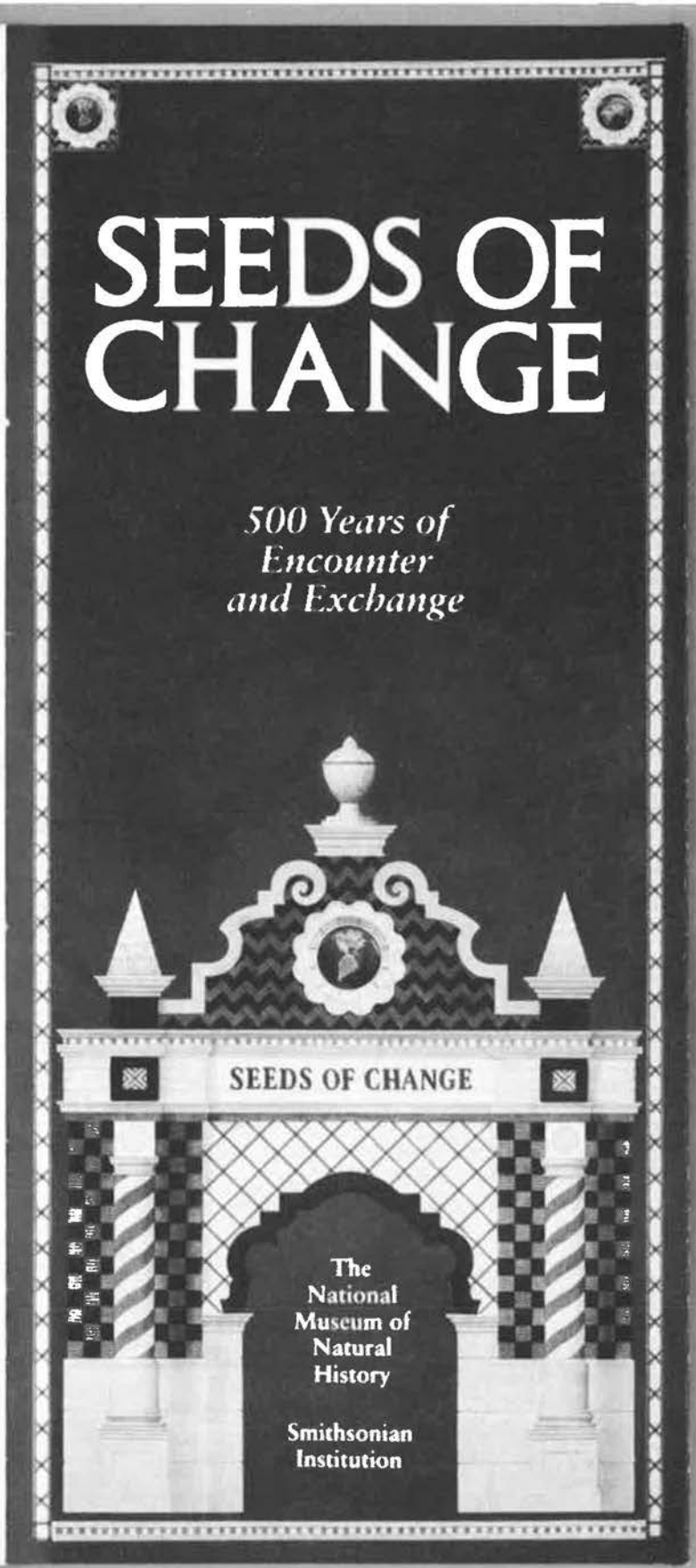

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This is an informal newsletter by which working research information on the genetics and cytogenetics of maize is shared. The information and data are shared by Cooperators with the understanding that they will not be used in publications without their specific consent.

Notes for the 1993 Maize Genetics Cooperation Newsletter need to be in my hands by January 1. Be concise, not formal, but include specific data, observations and methods. A double-spaced, letter-quality copy of your text is needed. Please follow the simple style used in this issue (title; authors; minimal citations). Whenever possible send an electronic version on 3-1/2 or 5-1/4 floppy disk, identifying the operating system (e.g., MS-DOS) and the word processor (e.g., Microsoft Word). Figures, charts and tables should be compact and camera-ready, and provided in electronic form if possible.

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Chas. R. Burnham sends word that reprintings of "Discussions in Cytogenetics" can be obtained from Burgess Internat. Group Inc., 110 Ohms Lane, Edina, MN 55435.