MAIZE GENETICS COOPERATION

NEWS LETTER

60

March 31, 1986

The data presented here are not to be used in publications without the consent of the authors.

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TABLE OF CONTENTS

I. FOREWORD 1

II. REPORTS FROM COOPERATORS 2

AMES, IOWA

c2-m1 - The frequency of revertant and colorless derivatives —P. A. Peterson 2

c-846432, a mutation of C-I to c —P. A. Peterson 2

En types: The weak effect of En-803920-3 —P. A. Peterson 2

c2-m and c2-s alleles confirmed in 1985 —P. A. Peterson 2

C-I, Sh and Wx mutants in a population containing En --P. A. Peterson 2

Inexpensive, high-quality kernel photography --B. E. Scheffler, P. A. Peterson 3

System relationships of vp-m451, a Mutator-induced, two-element system (vp-rcy and Cy) --B. E. Scheffler, P. A. Peterson 4

Crosses between a-mum2 and bz-rcy have demonstrated a relationship between Cy and Mu --P. S. Schnable, P. A. Peterson 4

Cy lines trigger mutability at two out of three Mutator-induced bz-mus alleles —P. S. Schnable, P. A. Peterson 4

Simultaneous loss of C, Sh, and Bz functions —P. S. Schnable, P. A. Peterson 4

Ac-flow, an Ac element that conditions a flow pattern —P. S. Schnable, P. A. Peterson 5

Activation of Ac and the coincident origin of a new P-RR allele —P. S. Schnable, P. A. Peterson 5

Frequency of Ug element activity —Y-B. Pan, P. A. Peterson 5

Brown aleurone (brn) update —P. Stinard 6

A putative Mu-induced viviparous mutant --P. Stinard 7

Studies of pollen and somatic instability in Mu-induced waxy mutants --S. Sackitey, D. S. Robertson 7

Evidence bearing on the relationship between germinal instability (mutator activity) and somatic instability (excision) in Mu-induced mutants —D. S. Robertson 8

Loss of Mu mutator activity when active Mu systems are transferred to inbred lines —D. S. Robertson 10

Mu-induced terminal deletions of the short arm of chromosome 9 --D. S. Robertson, P. S. Stinard 10

Putative Mu-induced deletions involving the end of the long arm of chromosome 9 —D. S. Robertson 11

New information on the timing of Mu's mutator activity —D. S. Robertson 12

Tests for Mu activity in the triple fusion nucleus or the early stages of development —D. S. Robertson 14

AMES, IOWA and COLUMBIA, MISSOURI

Update on a putative Mutator-induced chloroplast mutant --R. V. Masterson, D. W. Morris, D. S. Robertson, M. J. Skogen-Hagenson, J. G. Wheeler, M. L. Polacco 15

ATHENS, GEORGIA

Cloning of maize snap-back DNA —K. C. McElfresh, J. Strommer 17

Insertion of Robertson's Mutator in an exon affects transcript stability —J. Rowland, J. Strommer 17

Effect of Robertson's Mutator on chromatin structure at Adh1 —D. Ortiz, J. Strommer 17

AUSTIN, TEXAS

Adapting the Giemsa C-banding staining procedure for paraffin-sectioned material —M. Maguire 18

BALTIMORE, MARYLAND

Activation of Spm and modifier elements —N. Fedoroff 18

The structure of four bz-m5 alleles --R. Johns, N. Fedoroff, J. Banks 20

BARCELONA, SPAIN

Pollination and cytoplasmic effects on total dry weight --P. Fontanet, A. Alvarez, M. Blanco 21

Interaction between genetic and environmental effects on total dry weight —P. Fontanet, A. Alvarez 21

BATON ROUGE, LOUISIANA

Heterogeneity of ribosomal gene methylation in inbred lines --E. R. Jupe, E. A. Zimmer 22

BERKELEY, CALIFORNIA

Is the tassel a developmental compartment in the young meristem? —S. Hake, M. Freeling 23

Molecular characterization and genetical instability of an organ-specific mutant allele, Adh1-3F1124 --C-H. Chen, K. Oishi, M. Freeling 24

BERLIN and GATERSLEBEN, EAST GERMANY

Low content of nitrate reductase in chloroplast-ribosome deficient tissue of the 'iojap' mutant —T. Borner, J. Schiemann, R. R. Mendel 25

BLOOMINGTON, ILLINOIS

Effects of sodium azide seed treatments —R. W. Briggs, A. R. Bettendorf 26

BLOOMINGTON, INDIANA
Evidence that the K10 knob is not responsible for preferential segregation and neocentric activity --M. M. Rhoades, E. Dempsey 26
The effect of knob heterozygosity and competitive pairing on crossing over in the terminal region of chromosome 10 --M. M. Rhoades, E. Dempsey 27
BOLOGNA, ITALY
Response to recurrent gametophytic selection —P. Landi, E. Frascaroli 28
BOSTON, MASSACHUSETTS
Teosinte in Oaxaca, Mexico —G. Wilkes 29
BROOKINGS, SOUTH DAKOTA
Golden-2 linkage data --R. H. Whalen 30
Further mapping of esterase-4 --R. H. Whalen, A. L. Kahler 30
Genotypes at enzyme loci in 363 U.S. inbred lines of maize --A. L. Kahler, J. Matthees, R. Telkamp 31
CAMBRIDGE, ENGLAND
Nuclear DNA content varies between lines of maize and between maize and teosinte --D. A. Laurie, M. D. Bennett 31
Replicon size and rate of DNA replication in root meristem cells of Seneca 60 --D. Francis, A. D. Kidd, M. D. Bennett 32
CAMPINAS, SAO PAULO, BRAZIL
Genetics of how corn ticks --L. T. de Miranda, L. E. C. de Miranda 33
Genetics of horizontal resistance--measurement of the effects of B, P-WR, Plp and Krn on corn earworm resistance and yield --L. T. de Miranda, L. E. C. de Miranda, N. C. Schmidt 34
CANBERRA, AUSTRALIA
Segregation of red vs. white color in cob and tassel glumes —T. A. Peterson 36
CANBERRA, AUSTRALIA and BLOOMINGTON, INDIANA
Isolation of a candidate clone of the maize P locus --T. Peterson, D. Schwartz 36
CHAPINGO, MEXICO
Photosynthetic pigments in maize and teosinte —T. A. Kato Y., M. A. Vargas O. 37
CHESTNUT HILL, MASSACHUSETTS
A continued study on the stability of anther-culture derived maize callus lines —Y. C. Ting, M. G. Gu 37
Unstable progeny of a dihaploid maize line —Y. C. Ting 37
COLLEGE STATION, TEXAS
Dual sources of abscisic acid in maize kernels --J. D. Smith, F. Fong, C. W. Magill, B. G. Cobb, D. J. Hole 38
COLOGNE, WEST GERMANY - Institut fur Genetik
Studies of Ac-derived RNA —R. Kunze, P. Starlinger 39
Studies on the expression of the transposable element Activator at the protein level --M. Muller-Neumann, H. Fusswinkel, P. Starlinger 39
The aberrant Ds element in the Adh1-2F11::Ds2 allele --A. Merckelbach, P. Starlinger 39
Molecular cloning of bz2-m --K. Theres, P. Starlinger 40
Cloning of DNA from the P locus --C. Lechelt, A. Laird, P. Starlinger 40
The promoter of the shrunken gene promotes transient expression in a Triticum monococcum cell line --W. Werr, C. Maas, H. Lorz, P. Starlinger 40
COLOGNE, WEST GERMANY - Max-Planck-Institut fur Zuchtungsforschung
In vitro plant regeneration and somaclonal variation in the inbred A188 --E. Gobel, P. T. H. Brown, H. Lorz 41
Molecular cloning of the A2 locus --B. Martin, A. Gierl, P. A. Peterson, H. Saedler 41
Molecular cloning of the c locus —J. Paz-Ares, U. Wienand, P. A. Peterson, H. Saedler 42
Molecular analysis of the c2 locus --U. Wienand, U. Weydemann, U. Niesbach-Klosgen, P. A. Peterson, H. Saedler 42
Transposition of maize controlling element activator in a heterologous plant --B. J. Baker, J. Schell, H. Lorz, N. Fedoroff 42
COLUMBIA, MISSOURI
Lethal ovule (lo2) is distal to TB-9Sb —J. B. Beckett, B. Kindiger 43
Use of B-A translocations to locate duplicate genes —B. Kindiger, J. B. Beckett 43
Homozygous B-A translocation stocks —B. Kindiger, J. B. Beckett 43
A TB-9Lc stock with Wc on the B-A chromosome —B. Kindiger, J. B. Beckett 44
Large satellite on the short arm of chromosome 6 —J. B. Beckett 44
Analysis of small plants from nitrosoguanidine treated pollen —R. Vierling 44
The v*-576 mutation probably unmasks a developmental timing locus, Mof* —M. Polacco 44
v*-424: Allelism tests with v4, w3; linkage to w3 —M. Polacco 45
Endosperm protein analysis of thirty-five selected defective kernel mutants —M. T. Chang, M. G. Neuffer 46

Hypoploid test to locate defective kernel mutants on 8S or 8L proximal to TB-8Lc breakpoint —M. T. Chang, M. G. Neuffer 46
Breakpoint position of TB-8Lc —M. T. Chang, J. B. Beckett 46
A Mutator-induced PSII photosynthesis mutant —B. Cook, D. Miles 47
UFGT as a measure of Tripsacum dactyloides introgression into maize —J. B. Bussard, B. Kindiger, R. Larson 48
Pr:flavonoid 3'-hydroxylase in maize seedlings, leaf sheaths and aleurone —R. Larson 48
A mutation affecting the chloroplast coupling factor is located on 1S —C. Echt 49
Photo-induction of leaf lesions in Les1 plants —C. Echt 49
Preliminary summary of B-A translocation breakpoints —B. Kindiger, B.-Y. Lin, M. T. Chang, J. B. Beckett 50
An update on lesion mutants —D. Hoisington 50
Further localization of nec4 on chromosome 2S —D. Hoisington 51
Les9 is linked to ra1 on chromosome 7 —D. Hoisington 52
Zygomere mapping —G. G. Doyle 52
Breakpoints of TB-10L19, TB-10L30 and TB-7Lb —B.-Y. Lin 54
Correction on the B breakpoint of TB-3La —B.-Y. Lin 54
Mutations of aleurone color factors induced by EMS —E. H. Coe, M. G. Neuffer 54
Atrazine susceptibility factor —E. H. Coe, J. B. Beckett 55
Silk browning and cob color: P locus control —E. H. Coe, C-D. Han 55
Duplicate factors for orange pericarp (orp) —M. G. Neuffer, M. T. Chang 55
Transposition of chromosome-breaking Ds to marked chromosome arms —M. G. Neuffer 55
Expression of dek1 (clf) in leaf sectors —M. G. Neuffer 56
Checking for possible contaminants among new mutants —R. McK. Bird, S. Smith, M. G. Neuffer 56
Les1 lesion expression in non-green leaf tissue —D. Hoisington 57

FREIBURG, WEST GERMANY

On the use of the distal promoter in the zein genetic system —J. Brown, C. Wandelt, G. Feix 57
Organisation of the external spacer of nuclear rRNA genes —C. Toloczyki, G. Feix 58

GAINESVILLE, FLORIDA

Cloning of the sucrose synthetase-2 gene —D. R. McCarty, J. R. Shaw, L. C. Hannah 58
The sucrose synthetase-2 gene is genetically linked to Sh1 —D. R. McCarty, S. Wright, T. Helentjaris, J. Shaw, L. C. Hannah 60
A simple method for extraction of RNA from maize tissues —D. R. McCarty 61
The maize Adh1 promoter: chromatin structure —A.-L. Paul, R. J. Ferl 61
The maize Adh1 promoter: structures in supercoiled DNA —R. J. Ferl, H. S. Nick, B. J. Laughner 62

GAINESVILLE, FLORIDA and CANBERRA, AUSTRALIA

Biological activity of a putative maize Adh-1S promoter determined by transient expression in corn protoplasts —N. R. Isola, P. S. Chourey, D. Z. Sharpe, E. S. Dennis 62

GAINESVILLE, FLORIDA and ST. PAUL, MINNESOTA

A five-base-pair insertion is associated with mutation to male fertility and toxin insensitivity in T-cytoplasm maize —R. P. Wise, D. R. Pring, B. G. Gengenbach 63

GRAND FORKS, NORTH DAKOTA and COLUMBIA, MISSOURI

The dek mutants - new mutants defective in kernel development —W. F. Sheridan, J. K. Clark, M. T. Chang, M. G. Neuffer 64

HYDERABAD, INDIA

Somatic embryogenesis in glume callus cultures —K. V. Rao, P. Suprasanna, G. M. Reddy 64
Isozyme studies during differentiation in callus cultures —K. V. Rao, P. Suprasanna, G. M. Reddy 65
Anthocyanin accumulation and phenylalanine ammonia lyase (PAL) activity in callus cultures —P. Suprasanna, K. V. Rao, G. M. Reddy 66
Isolation of protoplasts from seedlings —P. Suprasanna, K. V. Rao, G. M. Reddy 66
Characterization of accumulated compounds in double recessive c2 pr —V. Sathyanarayana, P. Suprasanna, K. V. Rao, G. M. Reddy 67

IOWA CITY, IOWA

A meiotic function for the B chromosome nondisjunctional system —W. R. Carlson, C. Curtis 67
A further test of homology between the B chromosome and Abnormal 10 —W. R. Carlson 68

ITHACA, NEW YORK

Reversion from cms-S to fertility in vitro —E. D. Earle, V. E. Gracen 69

JOHNSTON, IOWA

Continued study of a defective WF9 cytoplasm, "wsp" —D. N. Duvick 69

KRASNODAR, USSR

Induced mutation process as source of new mutations —A. S. Mashnenkov 70

LA JOLLA, CALIFORNIA
A cloned maize genomic sequence shows homology to meiosis-specific cDNA clones from Lilium and to a small heat-shock protein gene of Glycine max --R. A. Bouchard 71
"Specious positive" maize genomic clones recovered due to annealing with oligo-dG/oligo-dC tails of cDNA-clone inserts--a cautionary tale --R. A. Bouchard, M. McKeown 72
LINCOLN, NEBRASKA
A restriction site polymorphism in ribosomal cistrons in a maize population selected for yield --T. R. Rocheford, J. C. Osterman, C. O. Gardner 73
Chromosomal location of additional genes for resistance to Corynebacterium (Clavibacter) michiganense ssp. nebraskense --T. R. Rocheford, C. O. Gardner, A. K. Vidaver 73
Developing maize breeding populations with resistance to Corynebacterium (Clavibacter) michiganense ssp. nebraskense --M. A. Thomas-Compton, T. R. Rocheford, C. O. Gardner 74
LLAVALLOL, ARGENTINA
Biochemical analysis of the somaclonal variation in maize regenerated plants --M. A. Rapela 74
Regeneration of maize plants from long-term embryogenic cultures selected in lysine-threonine medium --M. A. Rapela 75
Selection of stables or consolidated spontaneous mutants of the c locus --L. B. Mazoti 75
Relationships between photosynthesis, canopy traits and yield in flint type maize --J. R. Jatimliansky, M. I. Urrutia, M. J. Arturi 77
Vivipary in a breeding population --F. J. Babinec 77
LLAVALLOL, ARGENTINA and CASTELAR, ARGENTINA
Cytological evidences for a basic number x=5 in Zea polyploid complex --M. C. Molina, C. A. Naranjo 77
LOMAS DE ZAMORA, ARGENTINA and LLAVALLOL, ARGENTINA
Variation within Zea: numerical analysis of fruit traits --J. L. Magoja, L. M. Bertoia, I. G. Palacios, M. E. Streitenberger 79
More about teosinte characterization --M. B. Aulicino, J. L. Magoja 81
Introgression of teosinte germplasm in maize: a method to improve heterosis and variability --J. L. Magoja, G. Pischedda 82
Puna maize: results after three generations of selection for high protein quality and hard endosperm --A. M. Broccoli, M. E. Streitenberger, A. A. Nivio, J. L. Magoja 83
Expression of de*-7601 under different genetic backgrounds --I. G. Palacios, M. E. Streitenberger, J. L. Magoja 84
Rescue of de*-7601 homozygous plants --I. G. Palacios, J. L. Magoja 85
Perennial teosinte-Gaspe hybrids: inheritance of pericarp thickness --L. M. Bertoia, J. L. Magoja 86
Perennial teosinte-Gaspe hybrids: more about multilayer aleurone --L. M. Bertoia, J. L. Magoja 87
Perennial teosinte-Gaspe hybrids: storage proteins of different phenotypes --L. Ferrari, J. L. Magoja 88
LONDON, ONTARIO, CANADA
Fertilization and seed production with pollen from cultured tassels --D. R. Pareddy, R. I. Greyson, D. B. Walden 89
Generation of embryoids from primary and secondary anther cultures --R. I. Greyson, J. D. Dunlop 89
A change in the humidity level alters the response of seedlings to prolonged temperature stress --C. A. B. Rees, D. B. Walden, B. H. Rockwell, C. M. Nebiolo 90
Oncogene-related sequences in maize --R. Zabulionis, D. B. Walden, B. G. Atkinson 91
Confirmation of the isolation of a mRNA coding for a small heat shock polypeptide --C. A. B. Rees, D. B. Walden 91
Heavy metals induce changes in polypeptide patterns of plumules and radicles --A. Gullons, A. Mackenzie, D. B. Walden 92
Use of leaf discs to monitor protein synthesis under field conditions --D. B. Walden, T. G. Crowe 92
Seasonal variability in developing embryos --J. G. Boothe, D. B. Walden 93
Stamen development and microsporogenesis in cultured ear shoots --V. R. Bommineni, R. I. Greyson 94
LUDHIANA, INDIA
Resistance to charcoal rot in some elite maize populations --S. K. Dey, V. K. Saxena 95
MADISON, WISCONSIN
Condensed plant type (ct*) is actually reduced (rd) --W. F. Tracy 96
Pollen competition and heterosis --W. F. Tracy 96
The effect of GA_3 on corngrass (Cg) --W. F. Tracy, B. W. Ritchings 97
MEADVILLE, PENNSYLVANIA and LONDON, ONTARIO
The role of mitochondria in the generation and manifestation of hybrid vigor --C. M. Nebiolo, D. B. Walden, K. D. Nowicki 97
Response of mitochondria to chemical stress --C. M. Nebiolo, D. B. Walden, R. Rockar 98

MILAN, ITALY
Male gametophytic selection --E. Ottaviano, M. Villa, A. Legrenzi 98
HSP synthesis in the male gametophyte --C. Frova, G. Binelli, E. Ottaviano 99
Nucleolar organizer activity analyzed by silver-staining procedure --S. F. Dolfini 99
In vitro characterization of 54-1 and 56-1 mutants --M. L. Racchi, F. Sparvoli, G. Gavazzi 100
MILAN, ITALY and BERGAMO, ITALY
Opaque 6 allelic to pro1 mutant --C. Tonelli, G. Gavazzi, L. Manzocchi, N. Di Fonzo, C. Soave 100
MISSISSAUGA, ONTARIO
Variability in nuclear S1-homologous sequences among lines of maize --C. L. Baszczynski, J. E. Carlson, R. J. Kemble 101
Heat shock protein synthesis in sterile and fertile mitochondria --C. L. Baszczynsi, R. J. Kemble 101
MORGANTOWN, WEST VIRGINIA
Leaf protoplast RNA synthesis as influenced by IAA and ethanol as an estimate of heterosis --V. Ulrich, P. Smith 102
NASHVILLE, TENNESSEE
Knob expression in maize-Zea diploperennis hybrids --M. Eubanks 103
NEW DELHI, INDIA
Endosperm chromosome number in relation to kernel development --D. S. Mathur, K. R. Sarkar 103
Knob-heterochromatin distribution in Sikkim Primitive strains and Nal-Tel --S. Dash, J. K. S. Sachan, K. R. Sarkar 104
Meiotic abnormalities in North Eastern Himalayan (NEH) maize --S. Dash, J. K. S. Sachan, K. R. Sarkar 104
Discovery of Sikkim Primitive precursor in the Americas --J. K. S. Sachan, K. R. Sarkar 104
NOVOSIBIRSK, USSR
Locating of the mei-gene ms43 by TB-A stocks --I. N. Golubovskaya, E. E. Distanova 106
OAKLAND, CALIFORNIA
Mu1 as a transposon tag --E. Ralston, H. Dooner 107
PHILADELPHIA, PENNSYLVANIA
The location of Rf1 --R. S. Poethig 109
The location of ra2 --R. S. Poethig 109
RALEIGH, NORTH CAROLINA
Additional mapping of isozyme loci: Localization of Acp4, Dia2, Adk1, Tpi3, and Sad1 --J. F. Wendel, M. M. Goodman, C. W. Stuber 109
Origin of the S-1 plasmid-like DNA molecule of maize mitochondria --S. Elmore-Stamper, C. S. Levings, III 110
DNA sequence homology between the maize mitochondrial and chloroplast genomes --C. J. Braun, C. S. Levings, III 111
Cms-LBN differs from cms-L, its progenitor cytoplasm --P. H. Sisco 111
No linkage found between Rf4 and v16 or j on 8L --P. H. Sisco 112
RALEIGH, NORTH CAROLINA and COLUMBIA, MISSOURI
Tpi4 is located near the centromere on the long arm of chromosome 3 --J. F. Wendel, J. B. Beckett 112
Cms-ME (38-11) could be a Wf9-type revertant cytoplasm --P. H. Sisco, J. B. Beckett 113
ST. LOUIS, MISSOURI
A genetic map for a segment of the long arm of chromosome 6 --P. N. Mascia, D. F. Loussaert 113
ST. PAUL, MINNESOTA
Chromosomal location of a gene controlling high-methionine zein expression --M. S. Benner, R. L. Phillips 114
A tissue culture-induced mtDNA mutation reverts during a second tissue culture period --B. G. Gengenbach, H. Jessen, K. Storey 114
Allelism test for two threonine over-producing mutants --D. A. Frisch, B. G. Gengenbach 115
A suppressor-mutator transposable element system of independent origin --R. L. Phillips, L. G. Block, V. M. Peschke, C. R. Burnham 115
Tests for a cytoplasmic fertility restorer for ms1 --C. Burnham 117
pr gl8 stock for chromosome 5 --C. Burnham 117
su gl4 la --C. Burnham 117
SALINAS, CALIFORNIA
Perennialism attributes from Cuzco flour corn --D. L. Shaver 117
The fill period in Cuzco flour corn --D. L. Shaver 118
SALT LAKE CITY, UTAH and NORMAL, ILLINOIS
Construction of a genetic linkage map in maize using restriction fragment polymorphisms --T. Helentjaris, S. Wright, D. Weber 118
STANFORD, CALIFORNIA

Proposal for the nomenclature for the locations of cloned Mu elements --V. Walbot 120
Inheritance of somatic instability in Mutator lines --V. Walbot 120
Phenotypic changes in inactive Mutator stocks --V. Walbot 124
Reactivation of somatic instability in an inactive Mutator stock by treatment of seed with gamma rays --V. Walbot 124

STORRS, CONNECTICUT
Seed storage death --I. Greenblatt 124
Hydrogen peroxide treatment of seed --I. Greenblatt 125

TOCHIGI, JAPAN
Anther culture of different origins of varieties --K. Koinuma, N. Mochizuki, Y. Inoue 125

TSUKUBA, JAPAN and ST. PAUL, MINNEAPOLIS
Mitochondrial genomes of fertile revertants from cms-S --T. Ishige, F. Takaiwa, B. G. Gengenbach 126

URBANA, ILLINOIS
Changes in nuclear genomic background bring about reorganization of mitochondrial DNA --L. J. Escote, S. Gabay-Laughnan, J. R. Laughnan 127
A possible basis for the lag phenomenon observed in M825 conversion of cms-S strains with equimolar S1:S2 ratios --K. K. Kidwell, J. R. Laughnan, S. Gabay-Laughnan, L. J. Escote 128
Tagging the cms-S restorer genes --J. R. Laughnan, S. Gabay-Laughnan, P. Athma 129

WALTHAM, MASSACHUSETTS
Female fertility conferred upon the normally female sterile combination of papyrescent (Pn) with branched-silkless (bd) --W. C. Galinat 129
Progress in breeding silkless baby corn for whole ear consumption --W. C. Galinat 130
Dormancy and photosensitivity as premaize traits --W. C. Galinat 130
Maiz de Ocho, the frontier maize in the northward spread and adaptation of maize --W. C. Galinat 130
Duplicated parts of chromosomes in relation to the origin of maize --W. C. Galinat 131
The penetrance of the teosinte key traits, two-ranking and solitary female spikelets, in maize --A. E. Kennedy, W. C. Galinat 131
Noncupulate cob associated with soft outer female glumes --W. C. Galinat 132
Chromosome morphology of two inbreds, Mo17 and B73 --C. V. Pasupuleti 132
The cytology of the trigenomic hybrid --C. V. Pasupuleti, W. C. Galinat 133

WOOSTER, OHIO
Pleiotropies of recessive alleles and grain yields in maize hybrids --E. J. Dollinger 133

III. ZEALAND 1986 134

IV. MAIZE GENETICS COOPERATION STOCK CENTER 140

V. MAPPING 146

VI. MAILING LIST CHANGES 173

VII. RECENT MAIZE PUBLICATIONS 176

VIII. SYMBOL INDEX 195

IX. AUTHOR AND NAME INDEX 203

I. FOREWORD

This page can only be written after the rest of the Newsletter is completed, and at this point my appreciation crystallizes. The Cooperators are the key participants in our sharing of our field books, our observations and our note books in these pages, and the reason for this cooperative openness is in the traditions among maize geneticists. Data, techniques and tidbits are here shared with each of the rest of us, with the specific understanding that the information here is not to be used in publications without the consent of the authors.

Occasional contributors transmit an item to me and use the term "published" in asking that it be included. Information presented in these pages is not "publication", and neither the content nor the writing form should be in scientific journal style or formality. Items are received and assembled with minimum editing. Text should be double-spaced so that it can be readied and retyped with greater ease. References should be minimized. Tables, figures and charts must be compact, single-spaced, and ready for direct copying by the camera. The deadline for the next issue (number 61, 1987) is January 1, 1987.

Back issues of Nos. 30 (1956) to date will be sent on request; a microfilm of Nos. 1-29 and 33 is available for $9.50 (simply send a check to Coe, made out to Maize Genetics).

Shirley Kowalewski's skillful editing and refining of the copy and the proofs have aided me greatly during preparation of this issue, along with her care in the literature compilation and in keeping the year-round office tasks in order. Mary Brazil carefully produced and refined the literature citations, and Kathy Chappell and Christopher Browne aided with assembling and other vital tasks. Yvonne Ball and Dale Kennedy's efficient and skillful help in the University's Printing Services is also appreciated.

Dave Hoisington has especially provided me consultations, advice, and ideas through the year. This is in addition to compiling data, producing the superb working maps, and generally being a resource for computerable activities (e.g., how to distinguish plasmid from plasmic from plastic from plastid). I also appreciate help with proofing from Ming-tang Chang, Greg Doyle, Craig Echt, Chang-deok Han, and Bryan Kindiger.

This is the year to do the following:

Forward stocks of factors and combinations that are not already in the collection to the Stock Center.

Write Gil Fletcher about your experience with stocks requested; e.g., what you expected, what you received, how to improve the functioning.

Name that gene you have been keeping with an asterisk on it, and send mapping data for it and any others to next year's Newsletter.

Ed Coe

II. REPORTS FROM COOPERATORS

AMES, IOWA
Iowa State University

c2-m1 - the frequency of revertant and colorless derivatives

The *c2-m1* allele, an *Spm* allele (McClintock, Carnegie Yearbook 62:486, 1963), is an autonomous-acting allele that has a pattern of mutability that can be classified as medium-early.

In a search for revertants (Rev) among two families of plants that were homozygous *c2-m1/c2-m1* and were test crossed by a marked tester, the following was found:

	Rev (%)	Spotted	Clls	(%)	Other	Total
1	65 (6.2)	980	1	(0.9)	2	1048
2	40 (4.2)	902	2	(.2)	5	949

In previous studies with *En*-controlled alleles, as *a-m(papu)* (Peterson, TAG 40:367, 1970), it is the colorless non-spotting types that appear in high frequency. This high frequency of Rev relative to the low frequency of colorless types may possibly be indicative of its exon vs. intron placement of the element within the *c2* locus.

Peter A. Peterson

c-846432, a mutation of *C-I* to *c*

From an *En*-laden population with *C-I C-I* crossed by a *C* allele, a colored exception was isolated. In subsequent tests with an *En* responsive *c-m(r)* tester, colorless kernels appeared among the progeny. This would indicate that *En* is not present and the original *C-I* mutated to *c*. From these tests, an insert of an *I* element cannot be determined since this would give the same phenotype.

Peter A. Peterson

En types: the weak effect of *En-803920-3*

Many *En* types, readily differentiated in tests with *a-m(r)* or *a-m1-5719A-1*, can be rescued as excision products from the *a-m(Au)* allele (Nowick and Peterson, 183:440, 1981). One particular *En (En-803920-3)* has a very weak effect in its suppressive capacity against the *a-m1-5719A-1* allele. Used as a female, the coloration of the *a-m1-5719A-1* allele is motley and non-uniform. As a male, this *En* fails to suppress *a-m1-5719A-1* coloration. It would appear that the active component of this allele has a weakened capacity to suppress, and if the protein-DNA binding concept (Gierl et al., EMBO J. 4:579, 1985) is applied, it would imply a weakened binding capacity for this *En* product.

Peter A. Peterson

c2-m and *c2-s* alleles confirmed in 1985

In a continuing search for new independently originated *c2-m* alleles, several have been confirmed during the 1985 season. Their identity and minimal characterization follow:

c2-m	Muta-bility	Background Coloration	Notes
c2-m857210	+	palish	
c2-m857212	+	palish	
c2-m857213	+	palish	
c2-m857214	(+)	clear	
c2-m857229	+	clear	
c2-m857244	+	clear	male vs. female differential
c2-m857246	+	clear	male vs. female differential
c2-m857263	+ +	clear	
c2-m857271	+ +	clear	likely indep. control
c2-m857272	+ +	palish	
c2-m857273	+	clear	appears autonomous control
c2-m857274	+	clear	
c2-m857328	+ +	clear	
c2-m857353	+ +	clear	v. early and extremely late *spotting*
c2-m857364	+ +	clear	
c2-m857375	+	motley	

(+) = very low freq. of spotting; + = medium low freq. of spotting; + + = higher frequency of spotting

The stables include:

c2-s857230; c2-s857231; c2-s857234; c2-s857236; c2-s857241

These mutants appeared in two populations. The two populations differed in the rate of occurrence of each of the mutants as well as the numbered type of mobile elements included. Population 2 has been noted for a high incidence of mutant types, with rates per 10^6 as follows:

Population	*c2-m*	*c2-s*
1	7.5	0
2	25.0	20

Peter A. Peterson

C-I, Sh and *Wx* mutants in a population containing *En*

In a search for *wx-m* mutants, a (*C-I Sh Bz Wx/C-I Sh Bz Wx*) isolation plot included the *C-I*, *Sh*, *Bz* and *Wx* alleles that would mutate from the cross x *C sh bz wx* (although *Bz* would not be uncovered in a single-event mutation). Mutants include:

Deficiencies - Based on mutant type and differential male vs. female transmission: *Df-c-sh-bz-857003, Df-c-sh-bz-857164, Df-c-sh-bz-857182, Df-c-sh-bz-857183, Df-c-857177,* and a deficiency distal to *C, Df 9S-857078.*

C-I-weak - A weakened capacity to suppress the *C* allele: *C-I-weak857054, C-I-weak857061.*

C-I unstables: *C-I-m857056* (colored to colorless), *C-I-m857070* (colorless to colored spots), *C-I-m857101* (colored to colorless).

sh mutants - stable: *sh-857011, sh-857015.*

wx mutants: *wx-857027, wx-85-1.*

These mutants originated from four populations, each differing in type and number of mobile elements. The incidence of the mutant types from each of the populations also differed, with rates per 10^6 as follows:

Pop.	*Df-c-sh-bz*	*Df-C*	*Df-9S**	*C-I-weak*	*c*	*C-I-m*	*wx*	*sh*
1	2.12	0	—	—	—	—	—	0
2	0	7.1	—	—	—	—	—	0
3	.9	0	.9	.9	.9	1.36	1.8	1.8
4	4.7	0	—	—	—	—	—	—

- = no opportunity to detect

* via transmission tests, appears to be a deficiency 3 units distal to *C*

Peter A. Peterson

Inexpensive, high-quality, kernel photography

Often a readily accessible photographic record is desirable in the case of a new kernel phenotype or for the precise phenotype of a kernel that gives rise to a plant destined for molecular analysis. Due to the large number of mutants that we handle and the increasing use of our material for molecular analysis we found it necessary to develop a photographic system in our lab (versus use of campus facilities). In doing this we have developed a system that is easy to use, that results in high-quality slides, and for which the investment in equipment is very low. We believe the system developed in our lab is significant enough to be explained in detail in the following text.

The equipment required is dependent on what you already have in the lab. A zoom-stereo microscope is desirable because it allows for easy framing of the kernel. A trinocular microscope is not necessary if the eye tube diameter is less than 29mm, in some instances it may be necessary to remove the focusing ring for the left objective to achieve this maximum tube size (minimum tube size is 23mm). The Minolta X570 camera system is the only system available that can be used on eye tubes larger than 25mm and most eye tubes on stereo microscopes are larger than 25mm. The light source needs to have the capacity to produce an intense narrow beam. A fiber optic illuminator or an old external light source for a compound microscope are possible light sources with the fiber optic being the best choice. Polarizing filters are needed for the light source and microscope to decrease the amount of glare produced from the kernel. If a filter is not available for your microscope the best alternative is a camera filter; this will preserve the resolution. Cheaper plastic polarizing film can be used on the light source. The 35mm camera is attached to the microscope via the photo tube and the round microscope stage plate is replaced by three unattached microscope slides in an H formation.

To take a photograph the kernel is placed on a small piece of clay, the clay placed so as not to be visible in the photograph. The use of the slides allows for easy movement of the kernel and will help prevent shadows appearing in the picture. A black velvet cloth or other desirable background color is placed under the microscope stage. The cloth must be at least one inch from the slide, allowing the kernel's shadows to fall out of the focusing plane. The light is then concentrated on the kernel. If the light source is from one direction, a small piece of paper is needed on the opposite side of the kernel to reflect light onto the dark side. A bidirectional light source does not need the paper, but it poses a new problem in that the kernel's reflection will appear on one side of the picture. To overcome this the horizontal leg of the H formation on the opposite side of the reflection must be raised enough to eliminate the reflection as seen in the viewing frame. The magnification of the microscope is adjusted so that the desired amount of frame or specific section of the kernel is showing. Looking through the camera, the polarizing filter is adjusted to remove the glare, and the picture is taken using a cable release while at the same time covering the camera eye piece. Using the Minolta system the camera can be set on automatic, thus preventing the need to take a series of bracket shots to determine the shutter speed. If the ASA of the film is greater than 50 it may be desirable to set the camera at half the normal ASA for a greater depth of field. Other suggestions are: (1) Use an imprinting device to put an identificaion number on the picture for easy record keeping; (2) Do the photography in the dark; (3) The slower the ASA the greater depth of field. We use Kodak Professional 50 ASA Tungsten Ektachrome (EPY 135-36) for colored slides. Our equipment is as follows:

Camera body	Minolta X570	$149.95
Imprinter	Minolta Quartz Data back I	89.95
Photo tube	Minolta Microscope Adapter II	29.95
Polarizing filters	Promaster	20.00
	TOTAL COST	**289.85**

Brian E. Scheffler and Peter A. Peterson

System relationships of *vp-m451*, a Mutator-induced, two-element system (*vp-rcy* and *Cy*)

The independent regulatory element responsible for the control of mutability at *vp-m451* (derived from a Mutator source) was tested against receptor elements of known systems (Scheffler and Peterson, MGN 59:4). The test demonstrated that the unknown regulatory element activated *bz-rcy* (a tester for the regulatory element *Cy*) and the unknown element did not activate receptor testers for *Ac*, *Bg*, *Dt*, *En*, *Mrh*, *Mut* or *Uq*. Due to the relationship tests, the receptor element of *vp-m451* was designated as the *Cy* receptor element *vp-rcy*, derived from a Mutator source. The design of the test also allowed for the detection of any known regulatory elements in a Mutator-derived line that carried *vp-m451*. Active regulatory elements *Ac*, Bg, *Dt*, *En*, *Mrh*, *Mut*, and *Uq* were not found in the line, only active *Cy* was present.

In order to confirm the relationship of *vp-m451* to the *Cy* system, tests have been conducted to see if *vp-rcy* can be activated by any of the known regulatory elements. The relationship of *vp-m451* to the *Cy* system was confirmed, in that *vp-rcy* was only activated in the presence of *Cy*. The relationship of *Cy* to Robertson's Mutator system (Schnable and Peterson, MGN 59:4-5 and 60) and the confirmation that the receptor element *vp-rcy* responds to *Cy* demonstrates that genetically defined receptor elements (*rcy*) can be derived from Mutator lines. These genetic data indicate that Robertson's Mutator is no longer unique, in that a mutant derived from it can be defined in the terms of a classical two-element system.

Brian E. Scheffler and Peter A. Peterson

Crosses between *a-mum2* and *bz-rcy* have demonstrated a relationship between *Cy* and *Mu*

Crosses between *a-mum2* (= *A* + *Mu1*) and *bz-rcy* have shown that *a-mum2* stocks contain *Cy*, as do most Mutator-related stocks (Schnable and Peterson, MGN 59:5). In order to determine whether the *Mu1* element triggers mutability at *bz-rcy*, spotted and non-spotted selections were made from ears from cross 1 that exhibited 1:1:2 ratios of spotted:non-spotted-bronze:colored, suggestive of the segregation of a single *Cy*. These selections were testcrossed by *a*.

Cross 1: *a-mum2*/A, *Sh Bz*/*Sh bz-rcy* x *A*/*A*, *Sh bz-rcy*/*Sh bz-rcy*

If the 1:1:2 ratios from cross 1 were due to the segregation of a single *Cy*, and this *Cy* is at or closely linked to *a-mum2* (which would be true if the *Mu1* at *a-mum2* has *Cy* activity), the genotype of bronze spotted selections should be *a-mum2*/*A*, and the genotype of the non-spotted selections would be expected to be *A*/*A*. This was not observed. Selection for *Cy* activity did not select for *a-mum2* versus *A*. However, while all bronze spotted selections with the genotype *a-mum2*/*A* exhibited *a-mum2* mutability when testcrossed by *a*, none of the bronze non-spotted selections did so, i.e., this latter group of ears exhibited a 1:1 ratio of colorless non-spotted:colored. Mutability at *bz-rcy* cosegregated with mutability at *a-mum2*. Hence, again assuming the 1:1:2 ratios from cross 1 were due to the segregation of a single *Cy* element, the 1.4 kb *Mu1* element at *a-mum2* is not capable of triggering mutability at *bz-rcy*, and *a-mum2* is itself a *Cy* responsive allele.

Another interpretation is possible. The 1:1:2 ratios from cross 1 may have been due to a general deactivation of all *Cy* (*Mu1*) elements in the genomes of half the kernels. Such an interpretation would not require that *a-mum2* be a responsive allele, but would still demand that mutability at *a-mum2* and *bz-rcy* be controlled by the same family of elements (*Mu1*). Under this interpretation *a-mum2* would be an autonomously controlled allele, while *bz-rcy* would be independently controlled by *Mu1*.

Patrick S. Schnable and Peter A. Peterson

Cy lines trigger mutability at two out of three Mutator-induced *bz-mus* alleles

Cross 1: *Sh bz-mus*/*Sh bz-mus* x *sh bz*/*sh bz Cy*

In cross 1, *bz-mus* alleles responded to *Cy* lines as follows:

	Response	No. ears with spots/total
bz-mus4	+	8/8
bz-mus7	+	11/11
bz-mus8	-	0/11

Although these data do not conclusively demonstrate that *Cy* triggers mutability at *bz-mus4* and *bz-mus7*, they do strongly support this view. If this is true, *bz-mus4* and *bz-mus7* are *Cy* responsive alleles like *bz-rcy*. The alternative is that, concurrent with the origin of these *bz-mus*, all *Mu1* elements were deactivated. Under this latter model, *Cy*, or some other element in the *Cy* lines, would have to be capable of reactivating these deactivated elements. Either of these interpretations would lend further support to the relation between *Cy* and Mutator (see previous report).

Patrick S. Schnable and Peter A. Peterson

Simultaneous loss of *C*, *Sh*, and *Bz* functions

Five additional cases of simultaneous loss of *Sh* and *Bz* function have been recovered. These are in addition to the two cases previously reported (Schnable and Peterson, MGN 59:6). Five independent exceptional non-spotted bronze shrunken kernels (lines 1, 4-7 in the Table) were isolated from cross 1 (population size = 32,318 kernels).

Cross 1: *C Sh bz-rcy/C Sh Bz Cy* x *C sh bz/C sh bz*

		C Constitution	Female Transmission
1.	*sh-bz-82g760*	*C*	( )
2.	*c-sh-bz-825211w*	*c*	reduced
3.	*sh-bz-825211x*	*C*	( )
4.	*sh-bz-825211y-1*	( )	( )
5.	*c-sh-bz-825211y-2*	*c*	normal
6.	*sh-bz-825212x-1*	( )	( )
7.	*sh-bz-825215z*	*C*	( )

( ) = no test

In each case the new *bz* allele was shown to be a non-responder to *Cy*. Each exceptional type arose by a *Sh bz-rcy* change to *sh bz-n(rcy)* or a *Sh Bz* change to *sh bz*. In each case the *sh* phenotype is stable in the presence of *Cy*, arguing against the possibility of an imprecise excision of the *rcy* insert at *bz-rcy* (resulting in a non-responder), followed by its transposition into the *Sh* locus (resulting in a new *sh-m* allele).

In two of the seven total cases it has been shown that *C* function was also lost (Table). Of these two cases, female transmission is reduced in one case and normal in the other. In three cases *C* function remains intact. The *C* status has not been determined in the remaining two cases.

With the relation of *Cy* to Mutator, and the likelihood that these exceptional types represent deletions, it will be of interest to determine for each case whether the *rcy* insert is still present and whether it defines a deletion endpoint in a manner similar to that shown by Taylor and Walbot (EMBO J.4:869-876) for *Mu1* at *Adh1-S3034a*.

Patrick S. Schnable and Peter A. Peterson

Ac-flow, an *Ac* element that conditions a flow pattern

A novel *Ac*, which like *Ac2* (Rhoades and Dempsey, MGN 57:14), exhibits direct dosage, has been recovered from *bz-rcy* lines. One dose of *Ac-flow* is incapable of triggering Type II events at *bz-m4*, while two and three doses result in a flow pattern (Peterson, Genetics 54:249) of spots which are smaller in size (timing) and of greater density (frequency) than those produced by *bz-m4* in the presence of four standard *Ac* elements. Both *Ac2* and *Ac-flow* exhibit tissue specific expression.

Doses of *bz-m4*	Doses of *Ac-flow* 1	2	3
1	-	+	( )
2	-	+	( )
3	-	+	( )

- = no spots; + = spots; ( ) = not tested

Patrick S. Schnable and Peter A. Peterson

Activation of *Ac* and the coincident origin of a new *P-RR* allele

From cross 1 performed under controlled/isolated open pollination, an exceptional bronze shrunken kernel exhibiting *bz-m4* mutability was isolated. Since neither parent had *Ac*, this mutability demonstrates *Ac* activation. Testcrosses confirmed the following: the mutability of *bz-m4* is heritable, the new *Ac* is capable of triggering Type I events in *C-I Ds*, and the exceptional kernel was *P-RR*/–, which was surprising since both parents had carried *P-WR* or *P-WW* alleles. Contamination can be ruled out; a contaminant pollen grain would necessarily have to have been *C sh bz*, *Ac*, and *P-RR*, a combination not present in our nursery (which was in any event quite distant), and not present in commercial stocks or ornamental corn in home gardens.

Cross 1: *C sh bz-rcy/C- bz-m4, Cy* x *C sh bz/C sh bz*

The hypothesis: a *P-WR* or a *P-WW* allele in one of the parents consisted of *P-RR* with an insert of an inactive *Ac*. Transposition of the *Ac* resulted in its activation and release of the *P* locus, resulting in a change of *P-WR* or *P-WW* to *P-RR*. A linkage of the new *Ac* to *P-RR* would further support this interpretation.

Patrick S. Schnable and Peter A. Peterson

Frequency of *Uq* element activity

In a survey on the distribution of the *Uq* element, four inbred lines (B70, C123, 187-2, C103) were crossed by *a-ruq* testers and the resulting F_1 hybrids were then backcrossed by *a-ruq* testers. The backcrossed progeny kernels derived from each of the inbred lines fell into 3 distinct phenotypic classes: *colored* (.25), *mottled* (.25) and *colorless* (.50). No fully spotted kernels were found. This indicated that none of the 4 inbred lines had an active *Uq* element in its genome.

On the other hand, sectors of spotting were found on individual spotted kernels. This spotting occurred in 29 (.0008) out of the 37,434 colorless kernels as a single sector of color spots in the aleurone layer (Figure 1). These 29 single sectors showed two features. First, their sizes were different, varying from .05 mm^2 to 32.82 mm^2. This simply indicated that various numbers of aleurone cells (from 82 to 54,257) were involved in the sector area (timing of activation). Second, the sectors displayed a spotting pattern that was very similar to the one triggered by a standard *Uq* element. Therefore, we proposed that these single sectors of color spots resulted from the interaction between the *a-ruq* allele and a *Uq* regulatory element which had been activated.

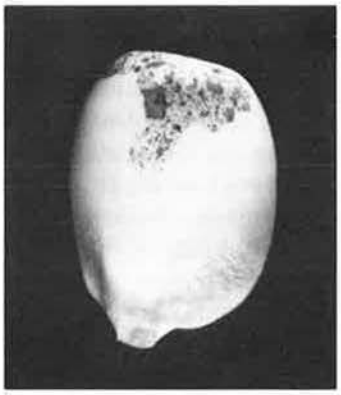


Figure 1. When an active *Uq* is not present in the genome, a colorless kernel of *a-ruq/a-ruq* shows a single sector of color spots upon activation of the *Uq* element during endosperm development.

To confirm that this was *Uq*-activated mutability and not *R*-mottling, we planted these 29 single-sectored kernels, their few-spot (from 1 to 5 single spots per colorless kernel) sib kernels as well as the colorless sib kernels. Twenty-five plants were grown from the 29 sectored kernels. These plants were first crossed by *r* testers and then reciprocally backcrossed to *a-ruq* testers. In addition, 4 tillers were selfed. The resulting progeny kernels were analyzed with the following results:

1. All were homozygous for *a-ruq* (Fourteen plants were *R/R*, the other 11 plants were *R/r*). The homozygosity of the *a-ruq* allele precludes *R*-mottling as a basis of sectoring.
2. None had an active *Uq* element in its genome, indicating that the *Uq* activation was not a germinal event.
3. Colorless kernels with a single sector of color spots were regenerated when the 25 plants were backcrossed to *a-ruq* testers reciprocally (Table 1). When they were used as maternal parents, 16 (.0047) out of the 3,398 progeny kernels had single sectors. On the other hand, 30 (.0018) out of the 16,235 progeny kernels of the reciprocal cross were sectored. Selfing of the additional 4 tillers also produced 4 single-sectored kernels with a frequency of .0052.

Similar results were obtained for the few-spot and the colorless sibs. They were all homozygous for *a-ruq* (the composition of the *R* locus was not tested) and generated single-sectored kernels when backcrossing to *a-ruq* testers (Table 1). However, the frequency of single-sectored kernels was lower for the few-spot sibs (.0017 as maternal and .0020 as paternal), and was the lowest for the colorless sibs (.0009 as maternal and .0008 as paternal). Finally, it was noted that no kernels with single sectors appeared in selfs or sibs of the *a-ruq* testers (Table 1).

Based on these results, we concluded that the presence of the sectors of color spots (sector of mutability) in an otherwise colorless aleurone layer

Table 1. Number and frequency of the single-sectored colorless progeny kernels generated in the reciprocal backcrosses to a-ruq testers of plants derived from different types of sib kernels

Kernel Type	Number of Plants Tested	Parental Type[1)]	Number of Progeny Ears	Number of Sectored Kernel(%)	Total Number of Colorless Kernels
Sectored	25	M	15	16(.47)	3,398
		P	67	30(.18)	16,235
Few-spot	64	M	71	46(.17)	26,431
		P	100	62(.20)	30,264
Colorless	147	M	155	53(.09)	59,758
		P	102	23(.08)	27,107
a-ruq (Control)	8	Self	6	0(0)	1,356
		Sib	2	0(0)	454

[1)] M: maternal; P: paternal

is due to the activation of a *Uq* element that triggers the mutability at the *a-ruq* locus at different times during endosperm development. Further, the genomes of the four inbred lines provide certain conditions that stimulate *Uq* activation. However, we are not able to determine whether the activated *Uq* is from the *ruq* receptor element at the *A* locus or if it is an inactive *Uq* becoming active in the genome, unless a germinal event can be rescued.

Yong-Bao Pan and Peter A. Peterson

Brown aleurone (*brn*) update

Last year crosses of TB-3Sb were made to plants heterozygous for brown aleurone (*brn*), a brown kerneled, seedling lethal mutant (MNL 59:7, 1985). The ears segregated for small, brown, almost defective kernels with brown embryos, and plump yellow kernels with shriveled, brown germs. It was suggested that the brown "defective" kernels had hypoploid endosperms and hyperploid embryos, while the yellow, near-germless kernels had hypoploid embryos and hyperploid endosperms. The defectiveness of the endosperm and embryo in these two seed types, respectively, were suggested to be due to hemizygosity at the *brn* locus.

In order to test these ideas, seeds of both types were planted this past summer. The yellow near-germless seeds did not germinate, but the brown defective kernels did, and the putative hyperploid plants were selfed and outcrossed onto *cl1cl1Cl3Cl3* silks to test for the presence of TB-3Sb. The selfed ears segregated for *brn*, but in a reduced ratio expected of hyperploid plants. The outcross ears segregated for the pale yellow kernels expected of cl_1. These results confirm the hyperploid status of the male parent, and the location of *brn* on the short arm of chromosome 3.

Since the brown defective kernels were shown to have hyperploid embryos, the brown pigmentation of these embryos must be due to diffusion of pigment from the endosperm. That the presence of the brown pigment in itself does not result in seedling lethality

is shown by the viability of these kernels. The seedling lethality seen in homozygous *brn* plants must be due to other factors, possibly the absence of an essential gene product, resulting in blockage of a metabolic pathway. The brown pigment may represent a precursor that accumulates behind such a block, or perhaps a degradation product of such a precursor.

Several attempts to extract the brown pigment from finely ground kernels were made, using a broad range of solvents. Standard procedures using hexane and acetone to extract carotenoids (F.P. Zscheile and J.W. Porter, Analytical Chemistry 19:47-51, 1947) removed carotenoid pigments, but left the brown pigment intact. Likewise, solutions of 100% and 90% methanol, and 70% ethanol, removed remaining carotenoids, and presumably any flavonoids that were present, but left the brown pigment. Solvents that partially extracted the pigment include .1N aqueous HCl and .5M NaCl. A buffer containing 5% 2-mercaptoethanol and 2.3% SDS seemed to remove most of the remaining pigment. These observations suggest that the brown pigment is not a carotenoid or an anthocyanin. Its extraction by solvents used in the extraction of proteins (.5M NaCl and SDS buffer) suggests that it could be a protein, a protein degradation product, or an unrelated compound that is tightly bound by or associated with protein. These possibilities, and others, will be further investigated.

Philip Stinard

A putative *Mu*-induced viviparous mutant

A viviparous, mutable aleurone mutant segregated on the selfed ear of a plant derived from our 1981 *a1 sh2*/purple Mutator isolation plot. The selfed ear segregated for purple, yellow-mutable-viviparous, and yellow shrunken2 kernels. The mutable viviparous kernels had a heavy pattern of purple spots (40 to 70 spots/mm^2), and each spot consisted of a single, heavily colored cell with little or no diffusion of anthocyanin into neighboring cells. This past summer, allele tests were made which confirmed that the mutant is an allele of *vp1*. We have chosen the allele designation of *vp1-Mum1* for this mutant. Ears from crosses of TB-3La onto heterozygous *vp1-Mum1* plants segregated for yellow-dormant, and purple-viviparous kernels, which is also characteristic of *vp1*.

Most viviparous kernels on selfed ears of plants from purple kernels of the original segregating ear, as well as those from selfs of outcrosses of such plants to a purple aleurone stock, have lost mutability (i.e., show no spotting). The few kernels which showed mutability had fewer than 0.2 spots/mm^2. We are currently trying to restore mutability by crossing heterozygous *vp1-Mum1* stable plants to purple Mutator stocks.

Philip Stinard

Studies of pollen and somatic instability in *Mu*-induced waxy mutants

In the 1984 Newsletter, we reported on studies of several *Mu*-induced waxy (*wx*) mutants. One mutable *wx* allele (*wx-Mum1*) had a high reversion rate as measured by the frequency of pollen grains that stained purple with KI/I_2 staining. In the 1984 tests and those reported here, procedures modified from O.E. Nelson (Genetics 60:507-524, 1968) were used for pollen staining.

In 1984 we grew 5 new *Mu*-induced waxy mutants, three of which were mutable. One of these, *wx-Mum2*, occurred on a selfed ear of a Mu^6 per se plant in 1983. Among the waxy seeds on this ear were both mutable and stable seeds. In 1984, shedding central spikes were collected and preserved in 70% alcohol. Eleven plants from mutable seeds and three plants from stable waxy seeds from this original ear were sampled. The pollen was stained and the frequency of phenotypically revertant pollen grains was determined (Table 1).

Table 1. The frequency of phenotypic reversions to Wx in pollen from homozygous wx-Mum2.

Plant No.	Total grain scored	Total Wx grains	Freq. of Wx grains	Phenotype of seeds planted	Phenotype on (X) ear of plant sampled
1280-1	204,159.90	17,275.95 13,035.75	846.20 X 10^{-4}* 638.51 X 10^{-4}*	Mutable	Mutable
1280-2	208,199.55	544.35 315.15	261.46 X 10^{-5} 151.37 X 10^{-5}	Mutable	Mutable
1280-3	242,522.25	687.60 573.00	283.52 X 10^{-5} 236.27 X 10^{-5}	Mutable	Mutable
1280-4	208,371.45	3720.50	178.55 X 10^{-4}	Mutable	Mutable
1280-6	262,319.40	802.20 630.30	305.81 X 10^{-5} 240.28 X 10^{-5}	Mutable	Mutable
1281-1	233,153.70	8050.65	345.29 X 10^{-4}	Mutable	Mutable
1281-2	268,536.45	257.85 85.95	96.02 X 10^{-5} 32.00 X 10^{-5}	Mutable	Mutable
1281-3	270,828.45	0.00	0.00	Mutable	Stable
1281-4	239,542.65	4985.10	208.11 X 10-4	Mutable	Mutable
1281-5	202,211.70	5987.85	296.13 X 10^{-4}	Mutable	Mutable
1281-6	238,883.70	7850.10	328.62 X 10^{-4}	Mutable	Mutable
1282-1	198,888.30	0.00	0.00	Stable	Stable
1282-2	232,294.20	0.00	0.00	Stable	Stable
1282-3	245,845.65	0.00	0.00	Stable	Stable

* Plants that have two entries had some pollen grains with an intermediate staining reaction. The first frequency is with these intermediate grains counted as starchy grains. The second frequency is that obtained if the intermediate grains are counted as waxy.

As with *wx-Mum1*, the reversion sectors in the endosperm of *wx-Mum2* are quite late and consist predominantly of single cells. This mutant has an extremely high frequency of phenotypically *Wx* pollen grains. It is of interest to note that stable derivatives of this mutant (Family 1282) do not show any reversion in the pollen. Plant 1281-3 is interesting. It came from a seed with a mutable endosperm but has lost instability in the pollen and also in the female germ line, since only stable seeds are observed on the selfed ear of this plant. In the production of the seed that gave rise to plant 1281-3,

perhaps the allele in the sperm fertilizing the egg carried a stable derivative *wx* allele while the other sperm still had an unstable allele. Heterofertilization could account for this seed, but it would require the simultaneous occurrence of rare events (i.e., mutation and heterofertilization).

Another mutable waxy mutant (*wx-Mum3*) occurred as a five seed sector on the ear of a *Wx Wx Mu* female plant pollinated by a *y1 wx gl1* stock. In 1984, these seeds, which were heterozygous for the *wx-Mum3* allele and the standard *wx* allele, were planted and the central spikes of the resulting plants sampled. All plants were self-pollinated. The results of these tests are given in Table 2. Selfs of all these

Table 2. The frequency of phenotypic reversions to Wx in pollen grains from wx-Mum3/wx plants

Plant No.	Total grains scored	Total Wx grains	Freq. of Wx grains	Phenotype of seeds on ⊗ ear
1143-2	195,851.40	0.00	0.00	Seg. mutable
1143-3	267,046.65	85.95	32.19×10^{-5}	Seg. mutable
1143-4	211,408.35	0.00	0.00	Seg. mutable
1143-5	204,430.80	0.00	0.00	Seg. mutable

plants segregated for mutable seeds, yet only one of them showed instability in the pollen. The reversion frequency in the pollen of the latter plant was high, considering that half of the pollen grains carry the standard *wx* allele. Plant numbers 2, 4 and 5 of this family seem to have lost instability in the cell lineage giving rise to the tassel, but not in the ear cell lineage. If this is true, the outcross plants resulting from the cross of these plants as males to standard should segregate for only stable waxy seeds on their selfed ears.

In 1983, some putative *Mu*-induced waxy mutants were obtained in a cross of *Wx Wx Mu* as a male with a *y1 wx* stock. Results of selfs of three of the plants from these seeds revealed that two segregated for stable seeds while one had only mutable seeds. These results and the results of the pollen examination of these plants are given in Table 3.

Table 3. The frequency of phenotypic reversions to Wx in pollen grains from three new Mu-induced waxy mutants which are heterozygous for the standard wx allele.

Plant No.	Total grains scored	Total Wx grains	Freq. of Wx grains	Phenotype of seeds on ⊗ ear
1145-1	241,118.40	28.65	11.88×10^{-5}	Stable
1145-2	279,022.35	0.00	0.00	Stable
1145-3	228,856.20	57.30	25.04×10^{-5}	Seg. mutable

These three mutants are all independent in origin since they come from pollen of different plants. One (1145-3) shows instability in both tassel and ear. Again the plants are heterozygous for the standard *wx* allele, and thus actual frequencies of *wx-Mum* reversions would be expected to be twice the observed values. The 1145-3 mutant has been assigned the symbol *wx-Mum4*. Plant 1145-1 shows an unexpected discordance between tassel and ear. The pollen has a high level of instability yet no mutable seeds are seen on the selfed ear. This allele may only be unstable in the germ line and not in the somatic tissue. The mutant carried by plant 1145-2 seems to be a stable *wx* mutant.

All of these *wx-Mum* alleles are being put in a multiply marked stock so that germinal reversion rates, as measured by revertant seeds, can be determined.

Solomon Sackitey and Donald S. Robertson

Evidence bearing on the relationship between germinal instability (mutator activity) and somatic instability (excision) in *Mu*-induced mutants

In the literature, *Mu* activity is measured by two criteria. The standard measure of this activity is in the germ line as indicated by the induction of mutants. More recently, with the advent of unstable (mutable) *Mu*-induced aleurone mutants, activity is often measured by the frequency of spots. These two phenomena may or may not be measuring the same thing. There is good evidence that events associated with germinal activity involve replicative transposition, while somatic instability is probably the result of excision events. The possibility that excision events are sometimes involved in germinal activity has not been ruled out. The following experiments bear on the relationship between germinal and somatic activity of *Mu*.

Last year (MGCNL 59:11-13, 1985) we reported on the possibility of restoring somatic mutability to stable mutant derivatives of *Mu*-induced *a1* mutants. This was done by crossing stable *a1* derivatives with active purple-aleurone-*Mu* (Pl *Mu*) stocks and then selfing the F_1. In many of these selfed progeny, the *a1* seeds had regained mutability. In several of the crosses, the Pl *Mu* parent had lost *Mu* activity for the first time (i.e., had become a first time *Mu*-loss stock). In some crosses to a stable *a1* derivative, where such Pl *Mu*-loss stocks were used, the selfed F_1 ears segregated for mutable seed. The *a1* stable lines were also outcrossed as males to non-*Mu* purple aleurone stocks. The selfed ears from these outcrosses segregated for predominantly stable seeds. This demonstrated that the stable condition was inherited. These latter self progeny were seedling tested to determine if new mutants were being induced in the stable parents. In none of these tests were new mutants observed. Thus the loss of somatic instability seems to be associated with the loss of germinal activity. Is the reverse true? The cross mentioned above, in which somatic mutability was restored to a stable *a1* derivative by crossing to a Pl *Mu* stock that had lost germinal mutator activity, will provide information bearing on this question. In this situation, both the *a1* stable derivative line and the Pl *Mu* line have been shown to lack germinal activity. Thus, neither parent had a functional germinal *Mu* system.

However, the F_1 has recovered somatic instability. If the two phenomena (i.e., somatic and germinal instability) are related or linked in some functional way, the F_1 plants might be expected to recover germinal *Mu* activity as well as somatic mutability. This was tested by outcrossing the *a1-Mum3 stable* /Pl *Mu*-loss F_1 (in which somatic instability is recovered) to a non-*Mu* purple line and selfing the outcross ears, scoring the segregating *a1* seeds for somatic mutability again and seedling testing the self progeny to determine if new mutants, expected in the progeny of a plant with a germinally active *Mu* system, are occurring. The results of such tests, along with the earlier results of the tests of the F_1 and parents, are given in Table 1. These admittedly limited data suggest that not only was somatic instability restored to the stable allele, but the mutable condition is now inherited. Only an occasional ear is seen in the outcrosses that does not segregate for mutable seeds. More importantly, the restoration of *Mu* activity in the aleurone does not appear to be accompanied by the restoration of germinal instability.

Table 1. Germinal and somatic activity in a reactivated stable derivative of the Mu-induced a1-Mum3 allele.

	Previous tests		1985 Tests			
a1 stable Germinal activity	Somatic instability in the self of a1 stable x P1 Mu-loss F_1	Germinal activity in the P1 Mu-loss parent	Somatic instability in the selfs of F_1 X non-Mu P1 aleur.	Total plants scored	Seedling mutants observed from the selfed ears	% of ears with only stable seeds
Negative	3.68*	Negative	2.4*	47	0	14%
			3.20*	44	0	4%

*These mutability values are the average values for all seeds from all ears in a family. The scale used is a 5 class scale with 5 being full color and 1 no spots. The values from the previous tests are actual counts. The values for 1985 are estimates because we have not yet had time to make the seed counts.

A second approach for answering the question about the relationship between somatic and germinal activity was to determine if there is a correlation between the extent of somatic instability, as measured by the frequency of spotting in aleurone mutants, and germinal activity, as measured by the induction of mutations. Selfed ears of outcross progenies of *a1-Mum1*, *a1-Mum2* and *a1-Mum3* with low, medium and high levels of somatic activity were scored for the level of somatic mutability and germinal mutator activity as measured by the frequency of the selfed ears segregating for new mutants. The outcrosses were to *a1 sh2*. Results are shown in Table 2. Some, but not all, of the parents of the plants that gave high average somatic mutability also had germinal *Mu* activity. None of the parents giving rise to low or medium outcross ears had germinal *Mu* activity. Thus, the mere presence of a system capable of causing somatic mutability is not sufficient for germinal activity of the *Mu* system. The two activities of the *Mu* system do not seem to be completely unrelated, however, since the greater the somatic activity the greater the likelihood of having germinal activity. The observation that stocks of stable derivatives of mutable *Mu*-induced mutants do not have germinal activity also suggests a relationship with respect to the loss of germinal mutations from stable derivative lines. It should be noted, however, that this is not the case with stable *Mu*-induced mutants that are stable when first observed. They usually have germinal *Mu* activity. Thus the loss of germinal activity in stable derivatives of mutable alleles is not the result of the stable phenotype per se.

Table 2. The results of tests for a correlation of somatic and germinal Mu activity.

Allele tested	Level* of somatic mutability of o.c. ears	Average* level of somatic mutability in selfed o.c. ears	No. of o.c. ears scored	% of ears segregating for germinal mutations
a1-Mum1	Low	1.6	46	0
a1-Mum1	Low	1.8	43	0
a1-Mum1	Low	1.8	44	0
a1-Mum1	Low	1.6	44	0
a1-Mum1	Low	1.5	45	0
	Avg.	1.7	Total 222	0
a1-Mum2	Low	1.3	49	0
a1-Mum3	Low	1.4	37	0
	Avg.	1.6	Total 308	0
a1-Mum3	Medium	1.8	45	0
a1-Mum3	Medium	2.3	47	0
a1-Mum3	Medium	2.7	46	0
a1-Mum3	Medium	1.9	50	0
	Avg.	2.17	Total 188	0
a1-Mum2	High	2.4	47	2.13
a1-Mum2	High	3.1	50	0
a1-Mum2	High	3.3	45	0
a1-Mum2	High	3.4	43	4.65
a1-Mum2	High	3.7	47	6.38
	Avg.	3.18	Total 230	2.61

*Somatic mutability is measured on a scale of from 1 to 5 with 1 being stable mutant (no spots) and 5 being full color. Class 4 is the highest mutable class. For the outcross ears the average value of mutability for all mutant seeds were about 3.5 for high 2.5 for medium and 1.5 for low.

When one considers what is known about factors affecting germinal *Mu* activity and somatic activity there are some obvious differences. Bennetzen (In: Plant Genetics vol. 35, in press) has shown that there is little correlation between *Mu1* copy number and the somatic reversion frequency (level of somatic mutability). In somatic mutability the level of activity ultimately depends upon what happens to the *Mu* component at the locus (i.e., does it excise or not and how often, etc.). However, in germline mutations the level of activity depends heavily on how many loci receive a transposed element. Thus, the more elements that can move, the greater the number of gametes with mutants. Therefore in somatic events we are looking at the factors influencing the movement of only one element, while in the classical germline events many transpositions are involved. The observations that somatic mutability can be turned on again while leaving germline mutability off (Table 1) and that the level of somatic mutability is not closely correlated with germinal activity (Table 2) suggest there are some differences in the two phenomena. The observation that the loss of somatic mutability is linked with the loss of germinal activity, on the other hand, suggests some similarities.

Donald S. Robertson

Loss of *Mu* mutator activity when active *Mu* systems are transferred to inbred lines

Early in our research program we desired to transfer the *Mu* system into the inbred lines B73, Mo17, A632 and B76. Each generation of backcrossing was tested for the retention of an active *Mu* system. However, only very rarely was it possible to retain mutator activity in these backcrossing programs after a few backcross generations, in spite of selecting each generation for crosses with the most active *Mu* parents. Table 1 lists the inbreds tested and the

Table 1

Mu activity in a backcrossing program designed to transfer Mu into selected inbreds.

Inbreds	Mutator Activity Generations					
	F_1*	1 st B.C.	2nd B.C.	3rd B.C.	4th B.C.	5th B.C.
B73	+ + +	+ + +	+ + +	0 + 0	- 0 -	- - -
MO17	+ + + +	+ + + +	+ 0 0 +	+ - - 0	+ - - -	0 - - -
A632	+ + +	+ + +	+ + 0	+ + -	+ + -	+ + -
B76	+ +	+ +	+ +	+ 0	0 -	- -

* + = Mu activity in the indicated generation; 0 = no Mu activity in that generation; - = backcrossing discontinued. Two or more conversion series were made for each inbred. The results (i.e., +, 0, or -) for each series are presented in the same order each generation.

number of backcross generations before *Mu* activity was lost. The generation of loss varied somewhat from inbred to inbred, but in all inbreds, except for two series of crosses with A632, *Mu* activity was gone by the fifth backcross generation.

In the early days of my work with the *y9* stock, which was the original source of *Mu*, I had transferred the *y9* gene, by a series of backcrosses, into the inbreds W22 and M14. When I began my first systematic investigation of the mutator system, I tested most of my *y9* stocks from different backgrounds for this activity, including the W22 and M14 *y9* stocks. These latter two stocks did not have mutator activity, while most other stocks of *y9*, which had been maintained in more or less heterogeneous backgrounds, showed the mutator phenotype. Thus, the M14 and W22 conversions probably represent two additional instances where mutator activity cannot be sustained in an inbred background.

These results may have significance in light of recent observations in laboratories at Stanford, Purdue and Iowa State University that some inbred plants have modified *Mu1*-like sequences and, hence, presumably possess the system for modifying the *Mu1*-like elements. Such active *Mu*-modifying systems might be expected to inactivate the *Mu1*-like elements contributed by the *Mu* parent in my attempts to transfer this system to inbred lines.

In 1983, my working hypothesis was that the loss of *Mu* activity was due to the inability of the "inbred condition" to support the transposition of *Mu* elements. To test this hypothesis, plants from the first *Mu*-loss backcross generation were crossed with a different inbred to produce a vigorous F_1 (e.g., B73 *Mu*-loss x Mo17 etc.). Selfed ears of plants from these F_1 progenies were scored for new mutants that would be observed if either parent had *Mu* activity, but no new mutants were found. These results are as expected since neither parent has an active *Mu* system. In 1984, these F_1's were outcrossed to an unrelated standard line. If restoring vigor to these inbred-*Mu*-loss backcross stocks activates the *Mu* system, mutants should be observed in the progenies of the selfs. Other phenomena might also activate the *Mu* system, such as a hybrid dysgenesis-like phenomenon in these F_1's, or possibly deactivation of the modification systems for either or both parents. These selfs were made last summer, but the ears are yet to be classified and the seedling tests made.

The seeds from these outcross plants were sown this summer and the plants selfed. If the *Mu* system has been reactivated, some of these self progeny should segregate for new mutants.

Donald S. Robertson

Mu-induced terminal deletions of the short arm of chromosome 9

Last year we presented the results of reciprocal crosses of plants heterozygous for putative *Mu*-induced deletions involving the *yg2* locus near the end of the short arm of chromosome 9 (MGNL 59:17-18, 1985). When putative heterozygous deletion plants (del./+) were reciprocally crossed to homozygous *yg2* plants, four kinds of heterozygous plants were found, as determined by the phenotypic ratios in their reciprocal outcross progeny:

1. Those that segregated 1:1 for green and yellow green seedlings when crossed as both female and male.
2. Those that segregated 1:1 for normal seedlings and yellow green when the del./+ plants served as females but fewer than half of the outcross progeny were yellow green when they were crossed as males.
3. Those that segregated 1:1 for normal seedlings and yellow green when the del./+ plants served as females but no yellow green seedlings were observed in the outcross progeny when they were crossed as males.
4. Those that segregated for less than 50 percent yellow green seedlings when the del./+ plants were used as females but produced only green progeny when they were crossed as males.

This past summer, seeds from the latter three kinds of crosses, in which the del./+ plants were used as females, were sown and the yellow green plants (putative genotype = del./*yg2*) were pollinated by heterozygous *wd* plants and heterozygous TB-9Sb plants. The *wd* chromosome has a short deficiency that includes the *yg2* locus. This deficiency is male transmissible. If the del./*yg2* plants do

indeed have deletions of the *yg2* locus, the break point could extend proximal to the *wd* break point. If such is the case, crosses with *wd* should segregate for white (albino) seedlings. If the deletion is not too extensive, pollination of del./*yg2* plants with TB-9Sb pollen might also result in segregation for albino seedlings when the hypoploid sperm fertilized a deletion bearing egg. The albino seedling would be homozygous for the deficient region that extends at least through the *wd* locus. Of course, both of these types of crosses will also be segregating for *yg2* seedlings as well, since both the B-A translocation and *wd* uncover the *yg2* locus carried on the nondeletion homolog. If the deletion is too extensive, it may be lethal in the embryos that are hypoploid for the B^A chromosome. The results of these crosses are given in Table 1. Based on the results of the reciprocal crosses of the del./+ plants with *yg2* and the crosses of the del./*yg2* plants with heterozygous *wd* and TB-9Sb plants, four classes of deletion plants now can be recognized as indicated in Table 1.

Table 1. Pollinations of putative heterozygous deletion plants with heterozygous wd and TB-9Sb plants.

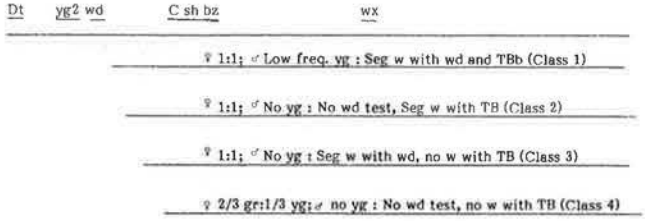
Deletion Number	Reciprocal tests of del./+ with yg2 as ♀	♂	wd Test	TB Test	Deletion Class
108-8	1:1*	2:1*	Seg w	Seg w	
110-8	1:1	2:1	Seg w	Seg w	
110-9	1:1	2:1	No test	Seg w	Class 1
114-1	1:1	3:1	No test	Seg w	
117-8	1:1	10:1	No test	Seg w	
116-10	1:1	No yg	No test	Seg w	Class 2
104-7	1:1	No yg	Seg w	No w	
107-2	1:1	No yg	Seg w	No w	
108-3	1:1	No yg	Seg w	No w	Class 3
110-6	1:1	No yg	No test	No w	
117-5	1:1	No yg	No test	No w	
107-1	2/3 gr: No yg 1/3 yg		No test	No w	Class 4

*In 1:1, 2:1, 3:1, 10:1 ratio the first number represents green seedling the second yellow-green.

Class 1 probably represents the shortest deficiencies because all yield some yellow green seedlings when del./+ plants are crossed as males. Since they segregate for albinos in the *wd* crosses, the deletion extends at least through that locus. The fact that all del./*yg2* plants segregate for albino seedlings when pollinated with TB-9Sb pollen suggests that a short deletion is involved, since the "homozygous" deletion condition is not lethal. The Class 2 deletions are probably a little longer than those of class one because no yellow green seedlings are observed in the male outcross progeny of del./+ plants to *yg2*; otherwise they behave like Class 1 deletions. The class 3 deletions are longer yet because no albino seedlings are observed when del./*yg2* plants are pollinated by TB-9Sb plants. This would be expected if the "homozygous" deletion condition is lethal. McClintock suggested that homozygous terminal deficiencies greater than that for *wd* (i.e., missing more than the first chromomere) would produce inviable embryos (Genetics 29:478-502, 1944). Class 4 deletions are probably the longest since they have reduced female transmission of the deficient chromosome when the del./+ plant is pollinated by *yg2*; otherwise they behave like Class 3 deletions. Only the relative lengths of these deletions are known, as indicated in Figure 1. Their exact extent and whether they are terminal or internal will have to wait on the results of cytological studies.

It is known that one of the Class 3 deletions (107-2) does not include the *sh* locus. In this case, the del./*yg2* plant carried *sh* on its normal chromosome linked to *yg2* and the heterozygous *wd* parent had *sh* linked to *wd* and *Sh* on the homologous chromosome. The F_1 segregated for shrunken seeds, and when the seedling test was made nonshrunken and shrunken seeds were planted separately. All the seedlings from the shrunken seeds were yellow green except for one green crossover seedling. The nonshrunken seeds gave both green and white seedlings but no yellow green seedling. If the deletion had extended through the *sh* locus, the seed giving rise to the del./*yg2* plant would have been shrunken (which it was not), and white seedlings would have been produced by some of the shrunken seeds in the *wd* cross.

Figure 1. Relative lengths of the four classes of putative Mu-induced deficiencies of the short arm of chromosome 9.



Donald S. Robertson and Philip S. Stinard

Putative *Mu*-induced deletions involving the end of the long arm of chromosome 9

Since there was evidence that *Mu* could induce deletion of the end of the short arm of chromosome 9 (MGCNL 59:16-18, 1985), I thought I would try for deletion of the end of the long arm of chromosome 9. It is known that deletions including the *Bf1* locus are transmissible through the female. In 1984 we had an isolation plot in which *Mu* plants were used as female parents and homozygous *Bf1* plants were used as male parents. In control crosses, standard lines were used as females. In the greenhouse this spring and early summer, 442,093 seedlings from the *Mu* cross were screened and 214 seedlings with blue fluorescence (referred to hereafter as *Bf-Mu*) were found, for a frequency of 4.8×10^{-4}. Five blue

Table 1. Pollen sterility in putative Mu-induced Bf1 mutants.

Normal pollen	75% Abortive pollen	50% Abortive pollen (empty grains)	50% Abortive pollen (Abortive grains 1/2 partly filled 1/2 empty)	50% Abortive pollen (All abortive grains partly filled)	50% large pollen grains 50% small pollen grains	50% Abortive pollen (Abortive grains partly filled & small)	25% Abortive pollen (All abortive grains empty)	25% Abortive pollen (Abortive grains 1/2 partly filled 1/2 empty)	15-20% Abortive pollen (Abortive grains partly filled & small)
134	1	4	5	10	5	1	1	4	9

fluorescing seedlings were found in a control population of 561,014, for a frequency of 8.9×10^{-6}.

The *Bf-Mu* seedlings were transplanted to the field and the plants were scored for pollen sterility and reciprocally crossed to standard. One hundred eighty-four good *Bf-Mu* plants were pollinated, as well as three of the control blue fluorescent mutants. All *Bf-Mu* plants were also pollinated onto homozygous *bf2* plants. Some of the original *Mu* parental stocks had *bf2* segregating in them, and thus it was possible in the isolation block for some of the blue fluorescent plants to be *bf2* instead of *Bf1* if the tassel of a *Mu* plant carrying *bf2* shed pollen before the tassel was pulled. All crosses to *bf2* proved negative.

The results of the pollen examination are given in Table 1. A total of 174 plants were scored for pollen sterility. Contrary to what was found for putative deficiencies in the short arm of chromosome 9 (in which very little pollen abortion was found), when *Bf1* is the target locus considerable pollen abortion of various types is present. This abortion ranged from 75 percent abortive pollen grains to as low as about 15 percent abortive pollen grains. The type of aborted pollen grains also varied from those that were completely empty to partly filled grains to grains that were completely filled with starch but smaller than normal. It has been observed by others that deficiencies with breakpoints in the neighborhood of *Bf1* will produce partly filled pollen grains. Larger deficiencies will result in 50 percent abortive pollen grains, either empty or partly filled or a mixture of both. Low frequencies of abortion might result from very small deficiencies where the deficient pollen grains frequently can make sufficient starch so that all deficient grains do not appear abortive. McClintock reported small pollen grains associated with deficiencies of the short arm of chromosome 9 (Genetics 29:478-502, 1944). All of these abortive classes would be expected if other types of aberrations were present (e.g., semi-sterility with reciprocal translocation, low sterility level with inversion). Since all sterility here is associated with the expression of the *Bf1* phenotype, it seems that most are probably deletions. It is known, however, that plants heterozygous for translocations with breakpoints in the vicinity of *Bf1* will have blue fluorescent anthers. Thus, translocation breakpoints in this region can affect *Bf1* expression. It is possible, therefore, that some of the observed pollen abortion is due to the induction of such an aberration.

To further substantiate the possible induction of deletions by *Mu*, the crosses by standard onto the silks of the *Bf-Mu* plants will have their pollen examined for evidence of sterility and be reciprocally crossed with homozygous *Bf1* plants. If a deletion has been induced, there is expected to be a deficiency of seedlings with the *Bf1* phenotype in the progeny of the male crosses of such heterozygotes, while the ears of the heterozygous plants will frequently give a one to one ratio. In a large deletion, female transmission of the deleted chromosome may also be reduced but not eliminated completely since, if the affected chromosome is completely female lethal, the blue fluorescent plant would not have been found in the first place. Both plants that showed pollen sterility and those with apparently normal pollen will be analyzed in this fashion.

At the same time, some of the plants from the female cross of the original induced *Bf1* plants will also be pollinated by TB-9La plants and by plants carrying genes that have been mapped to the long arm of chromosome 9 (e.g., *bm4*, *ms2*, *v1*). I would very much like seeds of any other mutants known to be on the long arm of nine that might be useful to delineate the extent of these putative deletions.

Donald S. Robertson

New information on the timing of *Mu*'s mutator activity

Previous studies (Genetics 9:969-978, 1980; Sci. 213:1515-1517, 1981) have indicated that *Mu* acts in the germ line tissue very late in development, meiotically or post-meiotically. In the past few growing seasons, we have learned much more about when *Mu* acts to produce new mutations. Since mutational events are presumably the result of the transposition of *Mu1*-like elements, these studies provide information as to when transposition is occurring.

New evidence on the timing of *Mu* activity has been obtained from studies of 1) mutants that occur in unexpected frequencies, 2) discordant endosperm mutants, and 3) gamma and ultraviolet ray treatment.

Mutants occurring in unexpected frequencies: Frequently, when new *Mu*-induced mutants are found segregating on selfed ears in *Mu* outcross families, they are observed in ratios much lower than the 3:1 ratio expected if the *Mu*-induced mutant had been carried by the gamete of the *Mu* parent. Such "low ratio mutants" are possible if the mutational event did not occur in the *Mu* parent but occurred very early in the development of the embryo of the seed producing the plant that was selfed. If such a mutation occurred before the cell lineages giving rise to the tassel and ear separated, both reproductive structures could have some germ line cells heterozygous for the new mutant. Depending upon the extent of the male and female germ tissue carrying this new mutant allele, various frequencies could be generated, varying from 25 percent mutant individuals to less than one percent. Frequencies as low as one mutant per ear have been observed. To test this explanation for "low ratio" ears, normal seeds were planted from ears segregating endosperm mutants in a low frequency. If the above explanation for these ears is correct, then one-half of the normal seeds from the ear will be heterozygous for the mutant allele, if for example, all tissue of the ear received the new mutant allele while only a very small portion of the tassel did. If only a small portion of the tassel and ear carries the mutant allele, very few of the plants from the normal seeds will be heterozygous for the allele. However, when a segregating selfed ear is found it should segregate in a 3:1 ratio. Results from the tests of two "low ratio" mutants support the suggestions that *Mu* can induce mutants early in development (Table 1). Another prediction can be made if *Mu* is inducing mutants at this time in development. In our standard *Mu* tests, the procedure is to self pollinate the *Mu* parent and the second ear of the outcross parent and screen the self progenies for new mutants. The selfing of the *Mu* and the outcross parents is essential because, if either parent is segregating for a new mutant, it would confuse our test results. However, occasionally, in spite of finding no mutants in the selfs, about half of the plants of the outcross progeny will segregate for a new mutant. Such a situation can be explained on the basis of a bookkeeping error or an environmentally sensitive mutant (e.g., a mutant that requires high temperature for expression, but the self progenies were tested at low temperature and the selfs of the outcross tested at high temperature). However, these exceptions could also be the result of a mutation in the cell lineage giving rise to the tassel, and *not* in the cell lineage giving rise to the ear of the *Mu* plant or vice versa. If this plant is selfed and outcrossed as a male, the self progeny will not segregate for the mutant, but the selfs of the outcrosses will. If the above explanation is correct, half of the plants from the seeds of the selfed *Mu* parent should be heterozygous for the mutant found segregating in the selfs of the outcross plants. These tests are yet to be made.

Discordant mutant seeds: For the past few years we have been studying the induction by *Mu* of mutants at the *y1* locus. We have induced mutants by using *Mu* plants as females and also as males. These mutants were induced by using *Y1 Y1 Mu* plants crossed onto or by *y1 y1 wx wx gl8 gl8* (See Mol. Gen. Genet. 200:9-13, 1985). When the mutant seeds (white or pale yellow endosperm) from these tests were planted and the resulting plants selfed, it was found that in some instances the selfed ears segregated for yellow seeds. In these discordant seeds the endosperm had the putative mutant allele, while the embryo was heterozygous for the nonmutant *Y1* allele. These discordant seeds are found in greater frequency in crosses in which the *Mu Y1 Y1* parent is used as a male than when the *Mu* parent is used as a female (Table 2). Such results are expected if *Mu* is inducing mutations in the gametophytes, because of the differences in the development of the male and female gametophyte. If mutations that result in the observed discordant seeds (i.e., white endosperm, *Y1 y1* embryo) are occurring in the gametophytes, the two sperm of the male gameto-

TABLE 1
TESTS OF PROGENY FROM
OFF RATIO EARS

su - 4582

1. Original ⊗ ear: 208 starchy seeds : 11 sugary seeds
   F_2 ⊗'s: segregating ears - 3 Su : 1 su
2. Frequency of segregating ears:
   50% IN = 10. Stand poor (10%) and weak plants)
3. Outcrosses of plants from mutant seeds to standard followed by selfing of the F_1's or crossing them to su su plants gave 3:1 and 1:1 respectively.

su - 4059

1. Original ⊗ ear: 227 starchy seeds : 1 sugary seed
   F_2⊗'s: segregating ears - 3 Su : 1 su ?
2. Frequency of segregating ears:
   61 Su Su : 14 Su su (Seeds planted - 120. Resulting plants were weak and most ears were nubbins and many were moldy.)

TABLE 2
TESTS FOR DISCONCORDANT MU-
INDUCED y1 MUTANTS

Mutant seeds from test in which MU plants were females:

Plants selfed - 283
Ears segregating yellow seeds - 25 (8.8%)

Mutant seeds from test in which MU plants were males:

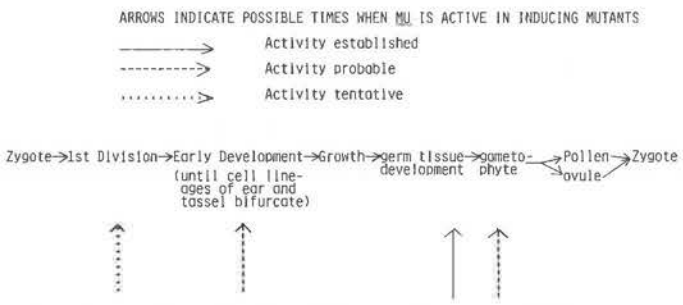
Plants selfed - 24
Ears segregating yellow seeds - 9 (37.5%)

phyte would be expected to differ with respect to mutant and nonmutant alleles much more frequently than would the polar fusion nucleus and the egg nucleus of the female gametophyte. In the male gametophyte only one mutational event is necessary to generate such a seed, while in the female gametophyte two mutational events must occur, one in each of the two cell lineages giving rise to the two nuclei of the polar fusion nucleus.

Pollen irradiation: Irradiation of *Mu* pollen with ultraviolet light and gamma rays has demonstrated that they have an effect on the mutation inducing ability of *Mu*. Ultraviolet light seems to enhance the effect of *Mu* (MGNL 56:2-4, 1982; 58:19-20, 1984), while gamma rays seem to diminish its effect (MGNL 58:15-16, 1985). Because DNA replication does not occur in mature pollen, it is unlikely that *Mu* transposition leading to mutations takes place at

FIGURE 1

PERIODS OF MU ACTIVITY IN THE LIFE CYCLE OF THE CORN PLANT



the time of irradiation. These effects on *Mu*'s mutational activity are most likely restricted to zygote or early developmental time periods, suggesting *Mu* might normally be active at these stages.

Figure 1 summarizes the evidence to date bearing on the time of *Mu* mutator activity.

Donald S. Robertson

Tests for *Mu* activity in the triple fusion nucleus or the early stages of development

In the previous report we presented evidence that *Mu* may be active in the zygote or early stages of development. To test this hypothesis, we set up an isolation plot this summer in which the female rows were *y1 y1 wx wx Mu* plants and the pollen parents were our standard line (*Y1 Y1*). If, in the triple fusion product giving rise to the endosperm, the *Mu* system contributed by the female is causing mutations at the *Y1* or *Wx* loci contributed by the sperms from the standard parents, completely white (or pale yellow) or waxy seeds should be observed. If *Mu* was active in an early stage of endosperm development, white/yellow or starch/waxy sectored seeds should result. The size of the sectors would depend upon how early the mutation occurred and how much of the endosperm arose from the two different cell lineages generated by the mutational event. Mutations in the first division might be expected to give rise to sectors in which half or more of the seed is white (or waxy), while mutations occurring in the second division might give seeds in which about a quarter of the seed is white (or waxy). Mutations occurring later in development would be expected to give sectors involving less than a quarter of the endosperm. The results are given in Table 1.

Table 1. The results of tests for Mu-induced mutation in the triple fusion nucleus or the early stages of development of the endosperm.

Total Seeds	Whole Seed y1 Mutants	Frequency	y1 Sectored Seeds	Frequency	Whole Seed wx Mutants	Frequency	wx Sectored Seeds	Frequency
	Mutator Test							
428,310	34	7.9×10^{-5}	248	5.7×10^{-4}	2	4.7×10^{-6}	3	7.0×10^{-6}
	Controls							
355,841	17	4.8×10^{-5}	133	3.7×10^{-4}	4	1.1×10^{-5}	0	-----

Table 2. Distribution seeds with various size y1 sectors

	< 25		25		50		75		>75		
Mutator Tests											
	99	(39.9%)	80	(32.3%)	46	(18.5%)	18	(7.3)	5	(2.0%)	248
Controls	42	(31.6%)	56	(42.1%)	26	(19.5%)	4	(3.0%)	5	(3.8%)	133

$X^2 = 7.7360$ $p = <.01$

The waxy mutants do not occur in a high enough frequency for a good test. A fair number of white (or pale yellow) seeds were observed. The frequency of this class was higher in the Mutator population than in the controls, but not significantly higher. The sectored seeds were frequent in both the Mutator tests and the controls, but the Mutator population had a significantly higher number.

The distribution of seeds with various sizes of white (pale yellow) sectors in the white/yellow sectored seeds is given in Table 2. Both Mutator and control populations had the same types of sectored seeds, but the distributions among classes varied.

The results of this test are inconclusive. The numbers are in the direction expected if *Mu* is inducing mutation in the triple fusion nucleus or early development, but more data are needed.

We did not plant a control for the test in 1985 because this was only a preliminary experiment to determine if there was any indication that *Mu* was functioning in these stages of seed development. The control used for the data reported here was a control population for a test made in 1984. The *y1 y1 wx wx* parent in the control was *y1 y1 wx wx gl8 gl8*, and therefore the female parent was not as well matched to the *Mu* parent (i.e., *y1 y1 wx wx Mu*) as it might be. Because these preliminary results are suggestive of *Mu* activity, we will repeat this work using as the *y1 y1 wx wx* control the recurrent parent stock used to make the *y1 y1 wx wx Mu* line.

If these early observations are substantiated, we will look for mutations in the zygote of the embryo. The white seeds obtained in the tests reported here

are most likely all discordant seeds, because it is very unlikely that a mutation at the *y1* locus would occur simultaneously in the triple fusion nucleus and the zygote of the embryo. To look for mutations that have occurred in the zygote, mature plants from the yellow seeds will have to be grown in an isolation plot and used as females with *y1 y1 wx wx* plants serving as male parents. Homozygous white ears are expected if a mutation has occurred in the zygote. Such a test will require a very large number of mature plants and is not worth undertaking until there is a very good likelihood of success.

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Update on a putative Mutator-induced chloroplast mutant

Some time ago, we described a chloroplast mutant that seemed to have been induced in one of our Mutator lines. This mutant (which we call 3366) was first found as a single striped plant in the outcross progeny of a male parent Mutator plant and a female standard Q60 line. This plant was crossed reciprocally to a B70 standard line, and 50 seeds from each of the outcrosses planted. In the female outcross progeny, we found that 17 plants were dead or dying by the fourth or fifth leaf stage, one plant was severely striped, while the remainder appeared normally green. The viable plant tissue that could be seen in the dying plants suggested that this class consisted of wholly yellow-green or heavily striped plants. The progeny from the reciprocal cross, in which the striped plant was used as a male parent, consisted entirely of green plants. Curiously enough, we have found recently that in crosses involving a modified B73 line with the WSP type of cytoplasm (kindly provided by Don Duvick of Pioneer Hi-Bred International), which has a moderate mutator activity and contains *Mu1*-like elements (unpublished observations), a striped plant was also recovered. The striping pattern we saw in this case, we must emphasize, was not similar to that seen in plants with the WSP type of cytoplasm, but was very much like that found in the apparently cytoplasmically-inherited striping pattern found in the Mutator stock.

In both of these mutants, further reciprocal crosses have confirmed that the yellow-green striping condition is maternally inherited. The first mutant line, 3366, has been crossed reciprocally for four generations, and has always given the same pattern of inheritance (i.e., the striped condition was transmitted only through the female). Normal green sibling plants, when reciprocally crossed, have never produced striped seedlings. Furthermore, no striped seedlings have been seen in the progeny of selfs of the male outcross progeny or the self progeny of outcrosses of green sibling plants. In progenies of striped plants which were pollinated by standard lines, various numbers of striped plants were found with considerable variation in regard to the degree of striping exhibited by the plants. In such crosses, at one extreme there were some cases that gave no striped seedlings (i.e., they were totally green); at the other, there were ears that produced seedlings consisting entirely of mutant tissue (i.e., they showed no stripes). We also noted that the ears giving striped seedlings formed a continuous spectrum of types between these two extremes. There was some correlation, however, between the degree of plant striping of the female parent and the frequency of striped, entirely green or entirely yellow-green seedlings present in the outcross progeny. Generally, the more intense the striping in the female parent, the more striped, and entirely yellow-green, were the seedlings obtained. The Pioneer striped mutator plant has only been crossed reciprocally for two generations, and so far is behaving like the original striped line.

We have carried out a comparative morphological examination by electron microscopy of the plastids in normal and in yellow-green plantlets of line 3366. Transverse sections were taken from about the first third of young leaves, and prepared and stained using conventional techniques. We found that bundle-sheath chloroplasts were quite normal in appearance in the mutant line; mesophyll chloroplasts, on the other hand, appeared abnormal. They were similar in abundance and size to those seen in the tissues of a normal plant; however, gross abnormalities were apparent in the grana stacks. These were few in number compared to normal mesophyll chloroplasts, and seemed to be either very large or very small in size, the intrathylakoidal membranes appearing compressed and often convoluted and distorted. In some cases partial disintegration of the membranes had occurred, no doubt increasing as development proceeded and leading, ultimately, to plant death. We suspect that these mesophyll chloroplasts are also largely unfunctional. We found that when the leaves were removed from wholly yellow-green plantlets, new leaf growth occurred from the cut bases. If the young leaves were not harvested, however, the plants died. This cycle could be repeated until the endosperm was depleted.

We have examined the leaf chlorophyll fluorescence induction kinetics of mutant 3366. The yellow-green mutant leaf regions of both the completely yellow-green leaves and the leaves with yellow-green and green sectors displayed a two- to three-fold higher initial yield of fluorescence and kinetics, indicative of a lesion in electron transport

on the reducing side of photosystem II. Blocks in photosystem I, plastocyanin, cytochrome f/b563 or plastoquinone mediated electron transport would generate this fluorescence data.

We have examined the polypeptide composition of thylakoids isolated from fully yellow-green 3366 plantlets and green siblings. On performing lithium dodecylsulfate polyacrylamide electrophoresis of gently solubilized membranes, we found no cytochrome f and no cytochrome b563 when gels were stained for heme. Heme-staining works well for cytochrome f, but poorly for cytochromes such as cytochrome b563, where the heme is not covalently bound. We also observed an approximate 50 percent reduction of the CP1-protein, believed to be the reaction center of photosystem I. Two nonallelic nuclear maize mutants are known that block cytochrome f/b563 assembly, *hcf*-2* and *hcf*-6*. Neither of these reduces the CP1-protein (Metz et al., Plant Physiol. 73:452-459, 1983). The cytochrome f/b563 complex can be isolated, and is known to comprise nuclear-encoded Rieske Fe-S proteins and three plastome-encoded polypeptides: apo-cytochrome f, apo-cytochrome b563 and subunit 4. We compared Western blots of mutants and normal thylakoids (antisera were kindly provided by W. Taylor, Berkeley) and found no cytochrome f or subunit 4 cross-reacting material and a reduced level of the Rieske Fe-S polypeptides. In the mutant we observed two polypeptides that bound anti-cytochrome b563, which were apparently 2-4 kD greater in molecular weight than the normal cytochrome b563. There was cross-reacting material of this apparent size class in normal thylakoids. We found no reaction in the mutant with a polypeptide corresponding to the normal cytochrome b563. This anti-cytochrome b563 serum also cross-reacts strongly with the Rieske Fe-S polypeptides. However, because we find no interaction of any of the other three antisera with additional polypeptides of either mutant or normal thylakoids, we believe the new polypeptides in the mutant are related to cytochrome b563 and not the Rieske Fe-S polypeptides. They may be unprocessed and partially processed apo-cytochrome b563.

We have compared chloroplast DNAs of mutant 3366 and wild type lines. Chloroplast DNAs were isolated by the method of Kolander and Tewari (Biochim. Biophys. Acta 402:372, 1975). Restriction endonucleases *Hin*dIII, *Pst*I and *Bam*HI were used to digest the DNAs. The fragments were fractionated by electrophoresis on agarose gels which were used subsequently in Southern blot analyses. Visual examination of the gels revealed no apparent differences between the restriction fragment patterns of wild type and mutant 3366 lines. Several Southern hybridizations, using an internal fragment of the cloned *Mu1* element as a probe to blots of these gels, failed to detect any homology with chloroplast DNA restriction fragments of both wild type and mutant lines. However, we did occasionally observe a faintly hybridizing band at a location between chloroplast DNA fragments in the mutant line that was about 4 kb in size in the *Hin*dIII and *Bam*HI profiles. We cannot, of course, rule out the possibility that this may be due to contamination of chloroplast DNA by genomic DNA. However, we have demonstrated that the mutant line has about 20 to 30 copies of *Mu1* -like elements in the genome, and these are randomly distributed in *Bam*HI and *Hin*dIII hybridization profiles. This is not consistent with the appearance of a single band in chloroplast DNA hybridization profiles should genomic DNA contamination have occurred. We have no explanation for this intriguing, and somewhat perplexing, result at the present. Our observation that the mutant line is deficient in cytochrome f led us to examine, again by Southern blot hybridization, these gene sequences in the chloroplast DNA of the mutant line. We used a spinach cytochrome f gene (kindly provided by R. Herrmann) as a probe to wild type and mutant chloroplast DNAs digested with *Hin*fI and *Pst*I. We found no discernible differences in the hybridization patterns of both lines.

We cannot rule out the possibility that Mutator and *Mu1* elements are involved in the induction of this mutant. The deficiency in the chloroplast-encoded polypeptides of the cytochrome complex raises the possibility of a *Mu1* insertion that occurred at one or another structural genes, or alternatively, at a locus specifying a protein essential for their incorporation into the membrane system of the chloroplasts. Genetic exchanges between nuclear and organellar DNAs are, by no means, improbable (see, for example, J.N. Timmis and N. Steele Scott, Nature 305:65, 1983). Perhaps the strong conservation of chloroplast DNA size led to the rapid excision of a *Mu1* element from one of these sites which, nevertheless, left a "footprint" resulting in a mutation. A detailed analysis, possibly involving DNA sequencing, would characterize the putative mutations at these loci.

In conclusion, we contend that the appearance of striped leaf mutants, similar in phenotype, in two lines (i.e., 3366 and B73 with the WSP type of cytoplasm), obtained from entirely different sources but having in common mutator activity and *Mu1* -like elements, strongly suggests that this unique system may be playing a role in the generation of chloroplast mutations.

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Cloning of maize snap-back DNA

We are cloning and characterizing maize snap-back DNA, that portion of the DNA reassociating at a Cot of 10^{-4} -10^{-5} M sec l^{-1}. We expect to find several classes of snap-back sequences, including those associated with the inverted repeats of transposable elements.

Maize DNA was partially digested with *Hae*III (300 ng DNA, 0.2 u *Hae*III μg^{-1} for 30 min), which yielded DNA fragments with an average length of 20 kb. These fragments were denatured in 0.15 M NaOH at 65 C for 15 min, then renatured by adding an equal volume of 0.15 M HCl, 1 mM Tris-Cl pH 7.0 at 65 C, and quenched in ice at a Cot<10^{-4} M sec l^{-1}. Ice cold 2X S_1 nuclease buffer (as specified by BRL) was added along with 43 u S_1 nuclease per ug DNA and the mix incubated for 4 hrs at 37 C. This treatment generated approximately 300 ng of S_1 - resistant DNA <400 bp long. S_1 nuclease should generate blunt-ended duplexes; however, for cloning it was necessary to fill in remaining single-stranded ends using T4 polymerase (4 u, 15 min, 37 C). This DNA was ligated into the *Sma*I site of the plasmid pUC18, and used to transform *E.coli* strain JM83. The transformation efficiency was 5.6 x 10^5 ampicillin-resistant, β-gal$^-$ clones per microgram snap-back DNA.

Ten clones, with insert sizes ranging from 42 to 200 bp, are currently being analyzed. The 200 bp insert from one of the clones, pCTE1409, has been used to probe a Southern blot of maize, teosinte, rice and petunia DNA. The banding pattern indicates that this clone represents sequences of variable copy number in different maize lines and present in at least one copy in all the genomes tested.

Kevin C. McElfresh and Judy Strommer

Insertion of Robertson's Mutator in an exon affects transcript stability

Recently we reported that insertion of the 1.4 kb *Mu1* element in the first intron of *Adh1* affects transcription but not processing (Rowland and Strommer, Proc. Natl. Acad. Sci, USA 82:2875-2879, 1985). To determine if *Mu* has a similar effect at other loci, we have analyzed a mutator-induced mutant of sucrose synthetase (*Sh*) recovered by Don Robertson.

Using anoxic roots as a source of RNA, we have compared the *Sh*-RNA level in this mutant line to that of a related non-mutant line. The mutant produces less than 5% of the *Sh*-RNA produced by the nonmutant, and the mutant allele's gene product is approximately 1.4 kb longer than normal *Sh*-RNA. By Southern mapping techniques, we have localized a *Mu1*-like insertion within the first 40 bp of the *Sh* gene. Using an in vitro transcription assay, we have found transcription of the mutant *Sh* allele is less than transcription of the nonmutant, but this decrease could account at most for a 50% decrease in the *Sh*-RNA level. Therefore, in contrast to our findings with *Adh1-Mu* mutants, where *Mu1* resides in an intron, insertion of *Mu* in an exon of *Sh* reduces message levels beyond the decrease associated with transcription. We conclude that this insertion of *Mu* within the first exon of *Sh* results in synthesis of an unstable transcript. Because this effect has not been seen in mutants containing *Mu* within introns, we hypothesize that processing of *Mu*-containing transcripts occurs normally but that aberrant mature transcripts are unstable.

Jeannie Rowland and Judy Strommer

Effect of Robertson's Mutator on chromatin structure at *Adh1*

The maize *Adh1-S* low expression mutant *S3034* contains a 1.4 kb insertion, designated *Mu1*, in the first intron. The reduction in ADH1 enzymatic activity has been correlated with reduced mRNA levels (Strommer et al, Nature 300:542-544, 1983); and the decreased mRNA level has been shown to be due to decreased levels of transcription (Rowland and Strommer, Proc. Natl. Acad. Sci. USA 82:2875-2879, 1985).

To determine whether the decrease in transcriptional activity is correlated with altered packaging of the DNA, we have been analyzing *Adh1* chromatin structure by means of DNAse I digestion of nuclei extracted from seedling roots. To control for trans-acting variability, *Adh1-S/S3034* heterozygotes have been used as a source of nuclei. The restriction fragment polymorphism conveyed by the *Mu1* insert provides us with a direct quantitative comparison of the general DNAse I sensitivity of the two alleles.

Our results indicate that there is a three- to five-fold reduction in the DNAse I sensitivity of the *S3034* allele compared to the *Adh1-S* allele. It appears, therefore, that insertion of *Mu1* into the first intron has changed the chromatin structure of the region flanking it, rendering the DNA less accessible to degradative enzymes. The alteration inhibiting DNAse I susceptibility may lead to impaired binding or transcription by the RNA polymerase complex and thereby account at least in part for decreased transcription of the mutant allele.

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Adapting the Giemsa C-banding staining procedure for paraffin-sectioned material

For three-dimensional viewing of chromosome positions with respect to each other, squash procedures are unsuitable. The following procedure, which has been developed for paraffin sections, provides consistent staining with differentiation of knob regions for at least premeiotic mitotic chromosomes at prophase (and interphase) stages.

Material is fixed in ethanol-chloroform 3:1 and stored indefinitely in a freezer before use, when it is transferred through three changes of 70% ethanol before paraffin infiltration through a standard graded tertiary butanol series (The laboratory is maintained at 26 C to keep the tertiary butanol from freezing). This is followed by standard paraffin embedding, sectioning and drying of slide-mounted ribbons. Slides are then treated as follows: xylene 3 minutes, a second xylene 2 minutes, 100% tertiary butanol 2 changes 2 minutes each, 95% ethanol 2 minutes, 70% ethanol 2 minutes, 45% acetic acid 5 minutes, a quick wash in boiled distilled water, saturated barium hydroxide at room temperature 10 minutes (saturated barium hydroxide is prepared by adding boiled distilled water at 85 C to barium hydroxide crystals in an Erlenmeyer flask, shaking, filling to the top, tight stoppering and allowing the crystals to settle out overnight; fresh barium hydroxide solution is prepared each day), five washes in boiled distilled water 2 minutes each, 2X SSC at room temperature for one hour, 3 washes in boiled distilled water 2 minutes each, Giemsa stain solution 25 minutes, two washes in Sorensen's buffer at pH 6.8 2 minutes each, one wash boiled distilled water, graded tertiary butanol series (10%, 20%, 40%, 80%) 2 minutes each, 95% ethanol 2 minutes (for destaining—can be varied for suitable degree of destaining), 100% tertiary butanol 2 changes 2 minutes each, 50% tertiary butanol 50% xylene 2 minutes, xylene several changes for a total of at least an hour, mount in permount. This method purposely avoids all air drying because air drying flattens the cells and nuclei. It may be that more consistent staining results are produced in the absence of air drying, so that this type of procedure might also be useful for initially squashed material. Initial fixation of material in ethanol-acetic 3:1 (instead of ethanol-chloroform 3:1) has so far given only poor results. Gibco Giemsa solution at pH 6.8 for use with Gibco Diagnostics chromosome kit #120-6706 has been used.

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Activation of *Spm* and modifier elements

Transposable controlling elements appear to be regular residents of the maize genome, but exist in an inactive form. There is substantial evidence that conditions or agents that induce chromosome breakage promote the activation of transposable elements. These include the genetic induction of chromosome breakage (B. McClintock, CIW Ybk. 49:157-167, 1950; ibid., 50:174-181, 1951; E.B. Doerschug, Theor. Appl. Genet. 43:182-189), X-ray and UV irradiation (M.G. Neuffer, Genetics 53:541-549, 1966; A. Bianchi, F. Salamini, and F. Restaino, MNL 43:91, 1969) and tissue culture (V.M. Peschke, R. Phillips and B.G. Gengenbach, MNL 59:91, 1985). The growing evidence that genetically active elements can be distinguished from inactive ones by their DNA modification patterns (N. Fedoroff, S. Wessler, and M. Shure, Cell 35:235-242, 1983; V. Chandler and V. Walbot, PNAS, in press) raises the possibility that elements are maintained in an inactive or cryptic form by methylation or other modifications of element sequences.

In the present communication, I report some evidence suggesting that *Spm* elements can themselves promote the genetic activation of previously cryptic elements. McClintock (CIW Ybk. 58:452-456, 1959; ibid., 70:5-17, 1971) showed that an active element could transiently activate an inactive element when present in the same plant, but that the inactive element subsequently segregated from the active one unchanged. The present study was undertaken to investigate the genetic basis of the apparent ability of an altered *Spm* element, designated a *weak Spm* (*Spm-w*) element, to revert to a *standard Spm* (*Spm-s*) element. McClintock (CIW Ybk. 56:393-401, 1957; ibid., 62:486-493, 1963) described the isolation of *Spm-w* elements, which transpose and trans-activate transposition less frequently and later in plant development than is characteristic of the *Spm-s* element, and reported that such elements can revert to give an *Spm-s* phenotype (McClintock, Am. Nat. 95:265-277, 1961). I have investigated the genetic basis of this apparent reversion, using an *Spm-w* derivative of the original *a-m5* allele (B. McClintock, CIW Ybk. 60:469-476, 1961).

The *a-m5* allele used has an *Spm-w* element inserted at the *a* locus. The phenotype of the *a-m5* allele is colorless with a sparse pattern of small, deeply pigmented *A* sectors. The *Spm-w* element at the *a* locus rarely excises in the germline (<.05%) to give stable null or revertant alleles. However, kernels on plants carrying the *a-m5* allele frequently show sectors within which the somatic reversion frequency is much higher than in the remainder of the aleurone layer. Moreover, individual kernels

TABLE 1

Isolation of New Spm-s and Modifier Elements from a-m5 Plants

	a-m5 kernels examined	Hft kernels	%	Hft plants analyzed	Hft plants with Spm-s	Hft plants with Modifier	Linkage of Spm-s or Modifier with a-m5
Year 1	3295	25	.76	6	2	1	none
Year 2	6106	51	.84	7	3	0	none

In year 1, 10 ears from plants that were homozygous for the a-m5 of the a locus and the closely linked Sh2 allele were examined for kernels that showed a high frequency of somatic excision of the Spm-w element at the a locus throughout the kernel (Hft). Hft kernels were grown and analyzed for the genetic transmissibility of the Hft phenotype. Each plant was crossed by and to plants that were homozygous for a sh2 and the Spm-responsive wx-m8 allele of the Wx locus. Three of the 6 plants analyzed showed the Hft phenotype in half of the kernels on backcrossed ears. Hft kernels from these ears were further backcrossed to a sh2, wx-m8 plants to determine whether the Hft trait cosegregated with the a-m5 allele and whether the Hft phenotype were attributable to an Spm-s element or a Modifier element, as judged by its ability to promote excision of the dSpm element of the wx-m8 allele in kernels not receiving the Spm-w element of the a-m5 allele. An element was assigned the designation Spm-s if it promoted frequent reversion of the wx-m8 allele in both the presence and absence of the Spm-w element and the designation Modifier if it promoted reversion of the wx-m8 allele only in kernels receiving the Spm-w element of the a-m5 allele. A similar selection was carried out in year 2 on ears obtained from plants grown in year 1 from kernels that had sectors of the high frequency phenotype in the aleurone layer, but did not show a high frequency of somatic reversion throughout the aleurone layer. Similar analyses were carried out on the Hft plants to determine the heritability of the Hft phenotype, as well as the nature and location of the element causing it.

that show a high frequency of somatic reversion (*Hft*) appear on ears of homozygous *a-m5* plants at a relatively high frequency (about 0.8% in the cultures whose analysis is described in Table 1). Plants were grown from a number of *Hft* kernels and tested for the genetic transmissibility of the *Hft* trait. Each plant was crossed by and to *a sh2, wx-m8* homozygotes. The *wx-m8* allele contains a defective *Spm* element (*dSpm*) whose excision can be promoted by either an *Spm-w* or an *Spm-s* element elsewhere in the genome, but which cannot itself excise from the waxy locus. Almost half of the *Hft* kernels gave plants in which the *Hft* trait was genetically transmissible (Table 1). Upon further backcrossing, it was determined that the *Hft* trait was not linked to the *a-m5* allele in any of the plants tested. In 5 of the 6 plants, the *Hft* trait appeared to be attributable to the presence of an independently segregating *Spm-s* element, as assessed by its ability to promote somatic reversion of the *wx-m8* allele in *a sh2* kernels of backcrossed ears. In 1 of the 6 plants the *Hft* trait showed the genetic behavior characteristic of a *Modifier* element (McClintock, op. cit., 1957, 1958). Half the kernels receiving the *a-m5* allele showed a high frequency of somatic reversion of both the *a-m5* and the *wx-m8* alleles. None of the *a sh2* kernels showed somatic reversion of the *wx-m8* allele, indicating that expression of the *Hft* phenotype was dependent on the simultaneous presence of the *Spm-w* element of the *a-m5* allele.

Thus in all of the plants analysed, the *Hft* phenotype appears to be attributable to the genetic activation of a previously cryptic element, either an *Spm-s* or a *Modifier*. In no case was the *Hft* phenotype found to be due to the reversion of the *Spm-w* element at the *a* locus to an *Spm-s* element. This is not altogether surprising, since another *Spm-w* element isolated from the *a* locus has been found to have an internal deletion within the *Spm* element (J. Banks, J. Kingsbury, V. Raboy, J. W. Schiefelbein, O. Nelson, and N. Fedoroff, Cold Spring Harbor Symp. Quant. Biol., in press). The overall frequency of activation of cryptic elements in this experiment is extremely high, approaching 0.4%. Although this type of study has not been carried out yet on other *Spm-w* alleles, they behave similarly. Several other *Spm-w* elements in different genetic backgrounds have been examined and show the same tendency to give *Hft* sectors, as well as *Hft* kernels at a frequency between 0.1 and 1%.

The spontaneous frequency of activation of cryptic elements can be assessed using an allele such as *wx-m8*, which has a *dSpm* element insertion. In a separate study of this type, using a *dSpm* allele of the *a* locus, only 2 new genetically transmissible elements were recovered from among more than 100,000 kernels examined. Similar or lower frequencies of spontaneous activation of *Dt* and *Spm* elements have been reported by McClintock (op. cit., 1951) and Neuffer (op. cit., 1966). Although controls of this type have not yet been conducted in precisely the genetic background of the present experiments, the frequency with which *Hft* kernels are detected has been followed in a number of outcrosses and appears to be characteristic of the *a-m5* allele, not the genetic background (Table 1). This raises the possibility that the *Spm-w* element itself promotes the genetic activation of cryptic elements. If this is the

case, it appears unlikely that the activation is mediated by an *Spm*-promoted DNA rearrangement, since the *Spm-w* element promotes its own excision and that of defective elements at other locations much less frequently (<.05%) than it promotes the activation of cryptic elements (~.4%). A different possibility is that an *Spm*-encoded gene product can interact with a cryptic element in such a way that it interferes with the maintenance of the cryptic state. A hypothesis that makes use of the recent observations on element methylation is that the cryptic state is maintained by DNA modification and the *Spm*-encoded gene product can occasionally interfere with modification of newly replicated element DNA, thereby promoting the conversion of a cryptic element to an unmodified (or less modified), genetically active form.

Nina Fedoroff

The structure of four *bz-m5* alleles

Salamini (MGNCL 41:100; 42:91; 43:91) first described a new bronze (*bz*) mutable allele in maize, now referred to as *AcBz*. McClintock determined that the *bz* mutable phenotype of *AcBz* was due to the presence of *Ds* at the *bz2* locus, and that there was an *Ac* element between the *sh* and *bz* loci (McClintock, pers. comm.). McClintock also observed that new *bz* mutable kernels frequently arose in the *AcBz* strain. The *AcBz*-derived *bz* mutable alleles are collectively called "*bz-m5*". Four independently derived *bz-m5* alleles (*A* through *D*; obtained from McClintock) were analyzed. They differ from one another in the frequency at which *Ac* excises from the *bz* locus, restoring the *bz* phenotype. The *bz-m5-A* allele shows the highest frequency of excision, followed by *B, C* and *D*. Of interest in the *bz-m5* alleles is the possible correlation between *Ac* excision patterns and the site of insertion of *Ac*. To date, differences in excision pattern in other mutable alleles have been correlated with alterations within the element itself (Fedoroff and Kingsbury, in prep.; J. W. Schiefelbien, et al., PNAS 82:4783, 1985; Zs. Schwarz-Sommer, et al., EMBO J. 4:2439, 1985). The present alleles, therefore, provide a unique opportunity for determining what effect sequences surrounding the *Ac* element have on its ability to excise.

Experiments were conducted to determine (1) if the *Ac* element of each allele is inserted at different positions within the *bz* gene, and (2) if the *Ac* element is the same in all four mutable alleles. To determine the site of insertion of *Ac*, DNA from plants of each strain (*A* through *D*) was digested with several restriction enzymes and analyzed by Southern blot hybridization. A 2.2 kb Pst fragment containing the entire *bz* coding sequences (D. Furtek, pers. comm.) was used as a probe. The sites of

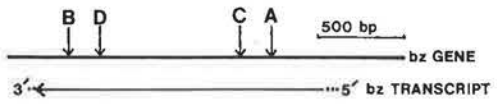

Figure 1. Arrows designate the position of Ac of bz-m5 alleles A through D within the bz gene.

insertion of *Ac*, determined from such analysis, are given in Fig. 1. There appears to be a complete 4.5 kb *Ac* insertion in all alleles, and the insertions are at different sites within the *bz* coding region. All four are within 2.0 kb of each other. For all four alleles, the orientation of *Ac* relative to the *bz* gene is the same. There were no insertions within the *bz* gene in DNA isolated from *AcBz* plants.

To investigate the similarity of the *Ac* elements in the four alleles, we have undertaken the cloning of the *bz* locus of the *bz-m5* alleles. Analysis of one clone with restriction enzymes indicates that the *Ac* element is indistinguishable from *Ac* elements isolated from two other mutable alleles, *bz-m2* and *wx-m9*. *Ac* elements from other *bz-m5* alleles are currently being isolated in a similar manner for further analysis and comparison.

From these results, we conclude that each *bz-m5* arises from the independent insertion of *Ac* as a result of short-range transposition of the *Ac* element into the *bz* locus. Furthermore, the differences in sites of insertion of *Ac* within the *bz* gene correlate with differences in excision pattern of *Ac* from the gene. There are several possible explanations for the latter observation. First, because the *bz* gene contains introns (J. Schiefelbein, pers. comm.), differences in the excision frequency of *Ac* could correspond to the presence of *Ac* in an exon or an intron sequence. A revertant allele of *wx-m9* contains a 6 bp duplication where *Ac* was originally inserted (Pohlman et al., Cell 37:635, 1984). This small insertion would maintain the correct reading frame of *wx*, allowing a wild-type *wx* phenotype. Excision of *Ac* from an intron, for example, may not have to be as precise as excision from an exon for *bz* activity to be restored. Second, if the sequences immediately flanking the *Ac* element are part of the substrate recognized by the putative *Ac* "transposase" (the transacting transposition function, Pohlman et al., Cell 37:636, 1984), altering these sequences could alter the rate at which *Ac* excises to restore gene activity. To further investigate these possibilities, we are analyzing the *bz* sequences flanking the *Ac* element in all four *bz-m5* alleles, as well as characterizing sites of *Ac* insertion in seven new, independently derived *bz-m5* alleles.

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Pollination and cytoplasmic effects on total dry weight

In 1983 (MNL 57:10-11), pollination and cytoplasmic effects for a single cross, including the reciprocal, were described. These hybrids were opaque, semident and middle cycle. The present work has two purposes: 1) To confirm reciprocal effects in long cycle hybrids; 2) To establish if there is a similar performance between the F_1 and the F_2 of three-way crosses, which are different in their cytoplasms.

The material was: a) the F_1 single cross, (AxB) direct cross and the reciprocal (BxA); these hybrids were opaque, flint and long cycle; b) two sets of three-way crosses obtained from these single crosses and three opaque dent inbred lines, 1, 2 and 3:

(AxB)x1 and (BxA)x1
(AxB)x2 and (BxA)x2
(AxB)x3 and (BxA)x3

Both classes of single cross plants were: 1) not pollinated; 2) self-pollinated; 3) pollinated with three inbred lines, 1, 2, and 3; and 4) pollinated with each parent line, A and B. Both sets of three-way crosses were: 1) not pollinated and 2) self-pollinated. The experimental design was "randomized plots" of 15 replications (one plot for all classes of material and types of pollination, 30 plants per plot). For each plant 5 parameters were measured, but in this paper we analysed one parameter only: total dry weight/plant.

Results were as follows (Table 1):

Table 1. Means of total dry weight/plant for all materials and treatments (g).

♀ \ ♂	♀(1)	■	x1	x2	x3	xA	xB
(AxB)x1	442,5	715,0					
(AxB)x2	506,0	690,0					
(AxB)x3	534,3	686,7					
(BxA)x1	519,0	710,8					
(BxA)x2	540,0	650,0					
(BxA)x3	531,4	682,7					
(AxB)	421,0	525,5	605,0	561,0	537,0	458,0	559,1
(BxA)	445,0	595,0	631,7	585,0	603,6	490,0	658,9

(1) : no pollinated.

1) Systematically, the reciprocal cross was better than the direct cross for all types of pollination:

(AxB) < (BxA), **P<0.01

2) There is a positive correlation (* P<0.05) between the F_1 and the F_2 in both sets of three-way crosses:

(AxB)x1 > (AxB)x2 > (AxB)x3
$[(AxB)x1]F_2 > [(AxB)x2]F_2 > [(AxB)x3]F_2$

r = 0.969 * P<0.05

(BxA)x1 > (BxA)x3 > (BxA)x2
$[(BxA)x1]F_2 > [(BxA)x3]F_2 > [(BxA)x2]F_2$

r = 0.987 * P<0.05

Thus we conclude: 1) There are reciprocal effects on total dry weight/plant in this single cross; 2) For both sets of three-way crosses, we notice that the F_1 (progeny of crossing with the inbred lines) and the F_2 (progeny of self-pollination) have the same sequence of yield.

P. Fontanet, A. Alvarez and M. Blanco

Interaction between genetic and environmental effects on total dry weight

Continuing the work reported in MNL 57: 10-11, we attempted to verify the interaction of environment with manifest events such as reciprocal and xenia effects, and the presence or absence of a similar performance between F_1 and F_2 progenies in two sets of three-way crosses. The same trial was repeated during two years and for two localities. We used a flint single cross, long cycle opaque-2 (AxB), and the reciprocal (BxA), and two sets of three-way crosses obtained from these single crosses and three dent opaque inbred lines, 1, 2 and 3. The treatments in each single cross were as follows: no pollination, selfing, crossing with 1, 2 and 3 inbreds, and backcrossing with their parental lines, A and B. In both sets of three-way crosses, the treatments were: no pollination and selfing. Experimental design was "randomized plots" of 15 replications. The parameter studied was total dry weight per plant.

Table 1. Means of total dry weight/plant for all materials and treatments (g).

♀ \ ♂	♀(1)	■	1983 x1	x2	x3	xA	xB
(AxB)x1	442,5	715,0					
(AxB)x2	506,0	690,0					
(AxB)x3	534,3	686,7					
(BxA)x1	519,0	710,8					
(BxA)x2	540,0	650,0					
(BxA)x3	531,4	682,7					
(AxB)	421,0	525,5	605,0	561,0	537,0	458,0	559,1
(BxA)	445,0	595,0	631,7	585,0	603,6	490,0	658,9

(1) : no pollinated

♀ \ ♂	♀(1)	■	1984 x1	x2	x3	xA	xB
(AxB)x1	510,4	533,3					
(AxB)x2	476,2	555,7					
(AxB)x3	460,0	591,2					
(BxA)x1	511,1	526,2					
(BxA)x2	543,1	530,0					
(BxA)x3	470,0	591,7					
(AxB)	397,7	447,8	493,6	501,1	489,5	457,5	494,5
(BxA)	421,8	461,7	503,3	526,7	510,0	481,4	554,6

(1) : no pollinated

In joint analyses measured during 1983 and 1984, the results (Table 1) were: 1) The reciprocal cross (BxA) is better than the direct cross (AxB). This result was the same during both summers and localities. Thus, to analyse all pollination treatments:

	1983	*1984*
(AxB) < (BxA)	**P<0.01; Δ = 9.5%	**P<0.01; Δ = 5.4%

2) Only in the three-way cross [(BxA)x3], the total dry weight per plant increased over no pollination with selfing treatment:

1983: no pollination<selfing ***P<0.001; Δ = 28.5%
1984: no pollination<selfing *P<0.05; Δ = 26.0%

3) In both forms of single cross, the no pollination treatment was always lower than crossing with 1, 2 and 3 inbred lines:

1983: (AxB) no pollination < (AxB)x1,2,3 *P<0.05
(BxA) no pollination < (BxA)x1,2,3 **P<0.01
1984: (AxB) no pollination < (AxB)x1,2,3 *P<0.05
(BxA) no pollination < (BxA)x1,2,3 **P<0.01

4) When the reciprocal single cross (BxA) is pollinated with the parental line B, the total dry weight is better than when the pollination is made with the parental line A. This result is always the same:

(*B*xA)x*B*>(BxA)x*A* *P<0.05
1983 Δ = 34.5%; *1984* Δ = 15.2%

5) When we compare the F_1 and F_2 progenies in both sets of three-way crosses, we notice that these hybrids do not have the same performance as far as yield is concerned during two years:

1983: (AxB)x1 > (AxB)x2 > (AxB)x3
[(AxB)x1]F_2>[(AxB)x2]F_2>[(AxB)x3]F_2
r = 0.969 * P<0.05
(BxA)x1 > (BxA)x3 > (BxA)x2
[(BxA)x1]F_2>[(BxA)x3]F_2>[(BxA)x2]F_2
r = 0.987 * P<0.05

1984: (AxB)x2 > (AxB)x1 > (AxB)x3
[(AxB)x3]F_2>[(AxB)x2]F_2[(AxB)x1]F_2
(BxA)x2 > (BxA)x3 > (BxA)x1
[(BxA)x3]F_2>[(BxA)x2]F_2>[(BxA)x1]F_2

In summary, we can conclude: 1) The reciprocal effects are invariable, there is not dependence with the environment for these hybrids; 2) The environmental interaction is very important in the xenia effect expression; 3) However, the environmental interaction changes, intensely, the sequence of production in the F_1 and F_2 progenies.

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Heterogeneity of ribosomal gene methylation in inbred lines

We have surveyed the ribosomal DNA (rDNA) of a number of inbred lines of maize with the methylation-sensitive restriction endonuclease HpaII. Although a large portion of the rDNA is not accessible to HpaII cleavage, MspI (which has the same recognition sequence as HpaII, but a different methylation specificity) produces an even distribution of rDNA fragments, indicating that the uncut fraction is due to methylation and not lack of the recognition site. A fraction of maize rDNA is always accessible to HpaII digestion. The characteristics of the HpaII-sensitive fraction varied among inbred lines of maize (see Fig. 1 and Table 1). HpaII digestion of maize rDNA

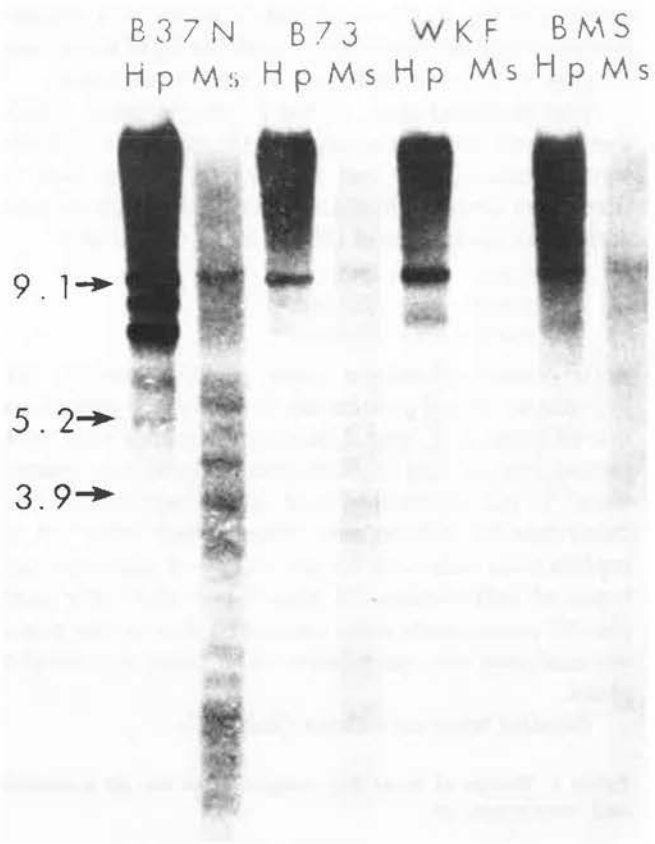

Figure 1. Maize DNA isolated from leaves (Zimmer et al. Plant Mol. Biol. Newsl. 2:93, 1981) was digested with either HpaII or MspI, and fragments were separated by electrophoresis on 0.8% agarose gels. Fragments were transferred to nitrocellulose by Southern blotting and probed with a soybean rDNA probe containing the entire repeat unit (pGmr1). The three bands produced by HpaII digestion of B37N were 9.1, 8.0, and 6.9 kbp in size, respectively.

Table 1. Inbred lines of maize surveyed with HpaII

Line	Number of Bands Present	Size of Bands (kbp)
B37N	3	9.1, 8.0, 6.9
BMS	2	9.1, 6.9
K10	2	9.1, 6.9
Ohio Pop	2	9.1, 6.9
Tx303	2	9.1, 6.9
WKF	2	9.1, 6.9
B73	1	9.1
GF	1	9.1
Mo17	1	9.1
Ny302	1	9.1
Tx601	1	9.1

produced either one repeat unit (RU) band, two bands, or three bands. In cases where two or three bands were observed, one fragment was of RU length and additional fragments were lower in molecular weight by 1.1 kbp and 2.3 kbp. B37N was the only inbred line in which all three bands were observed. All other inbred lines exhibited either a one- or two-band pattern. In lines with two bands, only the RU length band and the smallest band were present. Lines with a single band had only the RU length band present. The results from this survey of inbred lines show that not all rDNA repeat units in a maize genome are structurally identical with respect to methylation at HpaII sites. In addition, the number of hypomethylated HpaII sites present in the rDNA RU varies among inbred lines of maize. Experiments are being conducted to determine the functional significance of the observed structural heterogeneity in ribosomal gene methylation.

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Is the tassel a developmental compartment in the young meristem?

In a fate-map experiment designed to explore the autonomy and expression of the *Kn1* gene (Genetics 111:617, 1985; and to be published elsewhere), we noticed a number of leaf sectors that continued into the tassel (Figure 1). This result was unexpected because Johri and Coe (Dev. Biol. 97:154, 1983) found that tassel sectors excluded the rest of the plant body and vice versa.

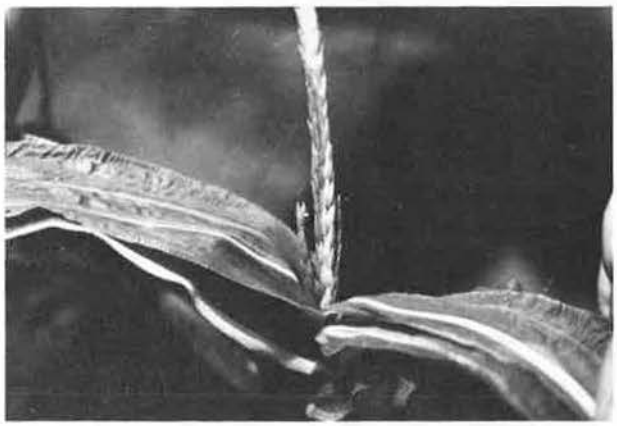
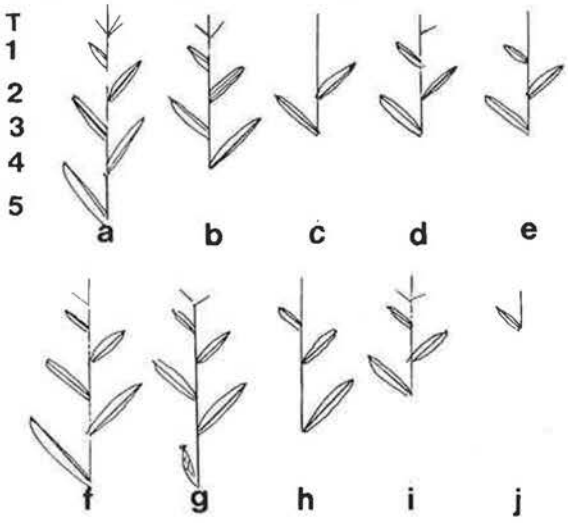


Figure 1. A leaf sector formed from the loss of *Lw* that extends into the tassel. The plant in the photograph is (f) of Table 1.

Seeds of the genotype *Lw Kn1-N2 Adh1-S/lw kn Adh1-FkFgamma25* were germinated 48 hours on 30 C benches, and then irradiated with 1000 rads at 50-80 rads/min. Approximately 750 plants were examined. Cell lineages that lost the dominant *Lw* gene were white. Of 15 sectors found in the flag leaf (or, in one case, the second highest leaf), 10 continued into the tassel (Table 1); 5 plants had sectors in the flag leaf or second highest leaf that did not continue into the tassel (Table 2). Tassel-only sectors were not seen, but it is possible that our "searching image" was for leaf sectors only. The fraction of plants with *lw* sectors that included the tassel, 10/750, is close to that seen by Johri and Coe, 44/3000.

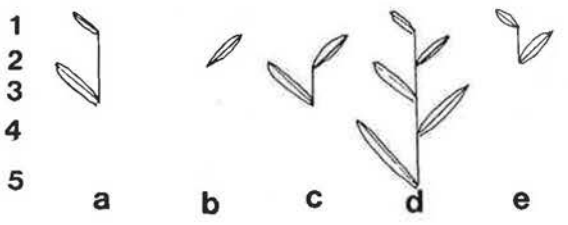
Table 1. Position of sectors found in leaves and tassel for 10 plants. The leaves are numbered from the tassel to the base: leaf 1 is the flag leaf. The leaves and tassels that are included in the sector are drawn. For example, the sector of (d) included leaves 3, 2 and 1, one lateral spike and the main spike of the tassel. All the tassel sectors included the main spike of the tassel with the exception of (g) which had only two sectored lateral spikes. The sector of (g) included the ear as diagrammed. The tassel sector of (j) was small, just entering the main spike and ending.



The size of the tassel sectors varied. In two cases the sectors were large, including one-half to one-third of the main spike and some of the lateral spikes (Table 1,a-b). In most cases, the sectors included only 1 row of spikelets of the main spike, plus or minus a sectored spikelet (Table 1,c-j). The number of cells extant at the time of irradiation that contribute to making the tassel can be estimated from the percentage of tassel that is occupied by the sector. From our limited sample, the apparent cell number

Table 2. Position of sectors in the flag leaf or second highest leaf that did not enter the tassel for 5 plants. Other plants with sectors on lower nodes are not included.

TABLE 2



conforms to previous findings: 20% of the tassels developed from 2-3 cells, 30% developed from 5 cells, 50% developed from 8-9 cells.

There are several possible explanations that might explain how our data differ from previous results, but none detract from the conclusion: there is no obligatory compartment boundary delineating leaf segments from tassel segments in the 2-day post-germination meristem. The plant shown in Table 1 (g) is particularly interesting: here a sector comprising 1/6 - 1/30 of the leaf area is represented as all of the ear and 1/9 of the tassel as well. A developmental compartment boundary, such as the anterior-posterior line bisecting each segment in *Drosophila melanogaster* must present an obligatory barrier to cell division (see D. Brower, Cell 41:361, 1985). Lineage restrictions resulting from programmed barriers to cell division may exist, but perhaps only in older "determined" apical meristems.

Sarah Hake and Michael Freeling

Molecular characterization and genetical instability of an organ-specific mutant allele *Adh1-3F1124*

A new *Adh1* mutant—*Adh1-3F1124*—was isolated in Berkeley in 1983, by screening F_2 families of maize lines that contain Robertson's Mutator (Robertson, Mutat. Res. 51:21, 1978) for anaerobic respiration defects. One family showed segregation of 1/4 of seeds that failed to germinate after 96 hr treatment of anaerobic stress. Seedlings were, however, rescuable by the removal of anaerobic stress immediately following the initial treatment (for detailed screening procedure ref. to C.-H. Chen et al., Maydica, McClintock volume; in press). The inability to grow under anaerobic conditions was attributed to lower expression of ADH enzyme activity in scutellum and root (<5% of its progenitor allele). A homozygous *Adh1-3F1124* line was later established by self-pollination of the rescued plants. The most dramatic mutant phenotype of *Adh1-3F1124* lies in the observation that ADH expression doesn't seem to be affected in the male gametophyte (pollen grain), although embryonic and somatic tissues continue to show less than 5% of ADH activity. The effect of mutation is therefore organ-specific.

Using DNA probes from the standard *Adh1-1S* allele, we have constructed a genomic restriction map of the mutant allele. Comparison between the progenitor *Adh1-3F* allele and the mutant *Adh1-3F1124* allele showed that the mutant was characterized by an insertion of about 2 Kb at 5′ end of the gene within a PstI and HindIII restriction fragment. This region represents 137 bp 5′ to the transcriptional unit, and 204 bp into the *Adh1* structural gene. Both the progenitor and the mutant allele have now been cloned into EMBL 3. Preliminary results indicate that the insertional DNA sequence is homologous but not identical to the *Mu1* transposable element, which has been previously characterized in other *Adh1* mutants isolated from Robertson's Mutator background. We have designated this new element as *Mu3*. We are now in the process of DNA sequencing and will soon know more about the precise location of the insertion point and the nature of the insertional element. At this point we do not know whether the mutant phenotype is reflected at the transcriptional level, nor do we have any data to speculate on the mechanism of the aberrant organ-specific expression.

Support of the idea that *Adh1-3F1124* is indeed caused by a transposable element insertion is provided by the fact that the allele is genetically unstable both somatically and germinally. Enzymatic staining for ADH activity in the aleurone layer clearly revealed that dark blue single cells or sectors of reverting cells was frequent (on the order of 10^{-3}). Several putative revertant kernels have been isolated (C. Fenczik and B. Kloeckener, unpublished data). Since the original mutant was ADH positive in

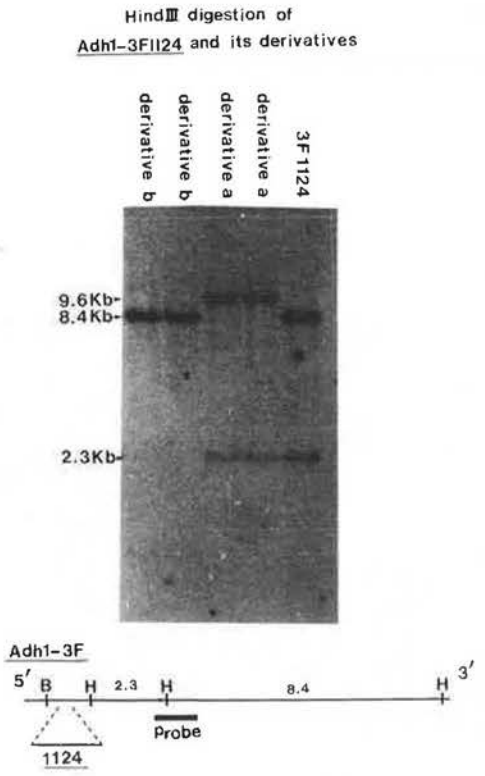


Figure 1. Autoradiograph of genomic Southern blot. Each lane contains 7 ug of HindIII digested DNA isolated from individual homozygous plants. The radioactive probe is an *Adh1* cDNA clone from plasmid PZML84 (Gerlach et al., PNAS 79:2981, 1982). It covers two HindIII fragments of *Adh1-3F1124* as represented by the thick bar below the restriction map. The broken lines denote the position of insertion in the progenitor *Adh1-3F* allele.

pollen, ADH null derivatives can be isolated by allyl alcohol treatment, which selects against ADH activity in pollen grains (Freeling and Cheng, Genet. Res. 31:107, 1978). Eleven ADH null derivatives have now been obtained by allyl alcohol selection using a single homozygous *Adh1-3F1124* tassel as the male parent. All of them showed stable null phenotype in all the organs and tissues that are known to express ADH. We believe that the derivative null phenotype is due to DNA rearrangement mediated by the *Mu3*, because all the derivatives show changes in restriction sites within the *Adh1* region. Three patterns of rearrangement were detected among 11 derivatives. Figure 1 shows two of them. In derivative-a, rearrangement occurs at the 3′ end of the gene where a HindIII site is displaced. This results in an increase of a HindIII fragment from 8.4 Kb to 9.6 Kb. In derivative-b, the 2.3 kb HindIII fragment that comprises ⅔ of the structural gene (intron 1 to exon 8) is missing. This is most likely due to a deletion, since a genomic probe representing from intron 1 to exon 6 of *Adh1* also failed to pick up the HindIII-HindIII signal when the same blot was re-hybridized (data not shown). A third kind of rearrangement is now being analyzed.

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Low content of nitrate reductase in chloroplast-ribosome deficient tissue of the 'iojap' mutant

The nuclear allele 'iojap', in homozygous condition, was previously shown to interfere with chloroplast biogenesis by causing a chloroplast ribosome deficiency (V. Walbot and E.H. Coe, Jr., PNAS 76:2760-2764, 1979; A. Siemenroth et al., Plant Physiol. 65:1108-1110, 1980, MNL 54:53-54, 1980). We analyzed the offspring of a cross, *ij ij* x ++, consisting of 3 types of seedlings: pure green, pure white, and pure yellowish. We measured the activities of nitrate reductase (NR) and nitrite reductase (NiR) according to published procedures (R.R. Mendel and A. Müller, Mol. Gen. Genet. 177: 145-153, 1979) and determined the content of the NR apoprotein by immunological methods (J. Schiemann and A. Müller, Biochem. Physiol. Pflanzen 180:63-74, 1985) using antiserum against NR purified from squash cotyledons. The results clearly demonstrate that chloroplast-ribosome deficient white and yellowish leaves exhibit only low levels of NR activity, whereas NiR activities are about 70% of normal wild-type activity:

	Leaf type		
	green	yellowish	white
Specific activity			
NR (nmoles NO_2^-,gfw^{-1}, h^{-1})	2240	425	220
NiR(nmoles NO_2^-, gfw^{-1},min^{-1})	180	133	126
Relative activity			
NR	100	19	10
NiR	100	74	70

The low NR activity is caused by a comparable low quantity of the NR apoprotein as tested by a 'protection of inhibition assay' using antiserum against NR. The molybdenum cofactor necessary for NR activity is present in all leaf types in equal quantities (Mendel unpubl.). NiR is a chloroplast enzyme (cf. E. Kessler, Progr. Bot. 43:74-82, 1981). Thus, it may not be surprising that it is affected in its activity by a mutation leading to the appearance of entirely undifferentiated plastids (although NiR is encoded in nuclear DNA and synthesized on cytoplasmic ribosomes, otherwise it would not be found in a chloroplast-ribosome deficient mutant). But it is striking that the NR shows low activity and even low protein content in the chloroplast-ribosome deficient leaves, because NR is located outside the plastids in the cytosol (Kessler, 1981). We conclude that the 'iojap' mutation, by inhibiting normal chloroplast development, leads to a reduced accumulation of the NR apoprotein. The reduced rate of accumulation might be caused by a repression (or missing derepression) of the synthesis or, less probably, by a specific and fast degradation of this protein.

It is known that the inactivation of photosynthesis may lead to a decrease in NR activity (cf. S.H. Duke and S.O. Duke, Physiol. Plant. 62:485-493, 1984). Neither the white nor the yellowish leaves of 'iojap' maize show any trace of photosynthetic activity as determined by measurements of delayed light emission (A. Siemenroth et al., Plant Physiol. 65:1108-1110, 1980; Börner and Matorin unpubl.). Therefore, the 'iojap' mutation could act via its effect on photosynthesis on NR *activity*. A direct and specific effect of the products of photosynthesis on the synthesis (or degradation) of the NR *protein*, however, is highly improbable. We postulate, therefore, that there exists in maize a dependence of NR accumulation on normal chloroplast development. There are a few examples for a control on the transcription or translation of the genetic information for certain *chloroplast* proteins in the nucleocytoplasmic compartment by the developmental stage of the chloroplast (J.W. Bradbeer et al., Nature 279:816-817, 1979; M.H. Harpster et al., Plant Mol. Biol. 3:59-71, 1984; L. Herrera-Estrella et al., Nature 310:115-120, 1984; S.P. Mayfield and W.C. Taylor, Eur. J. Biochem. 144:79-84, 1984; P. Eckes et al., Mol. Gen. Genet. 199:216-224, 1985). Our results suggest that the developmental stage of the chloro-

plast may also be important for the synthesis of at least one *non-chloroplastic* protein, the nitrate reductase. (We thankfully acknowledge generous gifts of seeds of 'iojap' maize by E.H. Coe, Jr., and of antiserum against NR by W.H. Campbell.)

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Funk Seeds Inernational

Effects of sodium azide seed treatments

In a continuing effort to induce changes in maize, kernels were treated with sodium azide (NaN_3). Maize kernels were pre-soaked in distilled water for 10 hrs. at room temperature. Sodium azide concentrations of 0.001M and 0.01M and a control were used in a phosphate buffer that was adjusted to a pH of 3.0. The treatments were carried out in a hood to vent the fumes. Ninety kernels were treated in 100 ml of the sodium azide solution for 2 hrs. Kernels were then rinsed twice and let stand in distilled water for approximately 1 hr. They were planted in trenches in the field, watered and then covered with moist soil (Briggs, MNL 51:5-6, 1977).

A common measure of effectiveness of a chemical mutagen and apparent genetic effect is to record the amount of physiological damage to the plant. Approximately 30 days after planting the material was rated for plant damage. A rating of 1 indicates no damage, a rating of 9 indicates extreme damage. A. Kleinhofs et al. (Genetic Engineering in Eukaryotes, Plenum, 1983) summarized some of their work on sodium azide in obtaining nitrate reductase mutants in barley. Also see Kleinhofs et al. (Mut. Res. 51:29-35, 1978) for seed treatment procedures and precautions in handling this mutagenic agent. In their paper they used a NaN_3 concentration of 10^{-3}M.

Five inbreds were used both in 1984 and 1985 (Table 1); four of these inbreds agree rather well in the plant damage ratings, i.e. within two ratings from year to year. However, there was considerable variability of the mutagen effect from inbred to inbred. In an attempt to explain this variability, the material was classified into germplasm source as Stiff Stalk and Lancaster lines (since all of the lines are proprietary except B73) and also classified by maturity. However, this did not explain the sensitivity of the different lines. Therefore, it appears that there are other factors that determine the sensitivity of each inbred to sodium azide.

If a rating of 6 is considered to show significant damage or effectiveness the 0.01M concentration of NaN_3 gave 58% of the inbreds rated 6 or above in 1984 and 60% in 1985. In both years the 0.001M concentration gave 33% of the lines rated 6 or above. Therefore, it is concluded that these doses of NaN_3 are in the range of acceptability as based on physiological damage and should be a good starting point for someone wanting to use NaN_3 seed treatments.

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TABLE 1A Inbred, dose of NaN3 and Rating for Physiological Damage Associated with the Treatment (1984 Data)

Inbred	Maturity	Germplasm Type	NaN3 Concentration .001M	.01M
51	M	LA	1	4
21	E	*	4	9
61	L	LA	1	4
41	M	LA	1	2
42	M	LA	1	3
22	E	LA	2	3
52	M	SS	8	9
53	M	LA	2	6
B73	M	SS	8	9
81	L	*	8	9
31	E	LA	8	9
82	L	*	5	8

TABLE 1B (1985 Data)

53	M	LA	4	6
51	M	LA	4	8
41	M	LA	2	3
32	E	SS	3	6
43	M	LA	2	4
42	M	LA	2	3
62	L	LA	7	8
43	M	SS	7	8
61	L	LA	3	5
33	M	SS	2	7
63	L	LA	7	9
34	M	SS	7	9
64	L	SS	6	8
54	M	SS	2	5
55	M	SS	4	5

E,M,L - Early, Medium, or Late maturity respectively
SS, LA - Stiff Stalk, Lancaster germplasm respectively
*Neither Stiff Stalk or Lancaster germplasm

Evidence that the K10 knob is not responsible for preferential segregation and neocentric activity

The K10-I and K10-II chromosomes possess large heterochromatic knobs, and both chromosomes are responsible for the induction of neocentromeres and preferential segregation. We no longer are convinced that the K10 knobs differ in any way from the genetically inert knobs in other locations in the genome. Previously, we believed that the knobs in the K10-I and K10-II chromosomes not only possessed the highly repetitive 185 bp sequence characteristic of all knobs, but carried a specific DNA sequence, not present elsewhere in the chromosomal complement, which effected preferential segregation and neocentromere formation. We now predict that both phenomena are controlled by chromatin situated in the more proximal "differential segment" of both K10 chromosomes.

There is convincing evidence suggesting a causal relationship between neocentromere formation in the meiotic divisions at all knob locations and preferential segregation of knobbed chromosomes to the basal megaspore. We have no reason to question the validity of this conclusion, but the observation made this past summer of neocentric activity in Df(C)/Df(H) sporocytes has forced us to reconsider our position that the K10 knobs were the responsible agents. The K10 knobs in common with all knobs of the comple-

ment are involved in preferential segregation but in a passive manner. Neither the Df(C) or Df(H) chromosome has the large K10 knob formerly held responsible for neocentric activity and preferential segregation, but neocentromeres were present in the sporocytes of these plants. Clearly, the knobs possessed by the two types of abnormal 10 are not the inducers of neocentromeres. What region(s) of the K10 chromosome could be involved? The answer is not yet at hand but we consider it highly likely that the differential segment of K10, comprised of chromatin not found in normal 10 or elsewhere in the normal chromosomal complement, controls both phenomena. We have not at this writing demonstrated that the chromatin of the differential segment controls or effects preferential segregation of knobbed heterologues. To determine whether or not it has this ability we have initiated experiments where both neocentric activity and preferential segregation will be studied in plants with Df chromosomes 10, lacking the large heterochromatic knob, and having knobbed and knobless chromosomes 9 marked by *Yg2* alleles. The data obtained from plants of Df(C)/N10, Df(H)/N10, and Df(C)/Df(H) constitution should clarify the role of the chromatin of the differential segment in neocentromere formation and preferential segregation.

M. M. Rhoades and Ellen Dempsey

The effect of knob heterozygosity and competitive pairing on crossing over in the terminal region of chromosome 10

Our studies with abnormal chromosome 10-Type I have shown that the terminal region of the long arm differs from normal 10L in possessing a differential segment of unknown origin marked cytologically by three prominent chromomeres, followed by a euchromatic segment containing the luteus-13, opaque-7, white-2 and striate-2 loci of normal 10L but with the order of *W2-O7-L13* inverted, and finally a large heterochromatic knob capped by a small euchromatic tip. A number of compounds involving K10-I, N10, and various Df K10-I chromosomes have been constructed in the course of our experiments. Listed below are some of these compounds and the values for *R-sr2* recombination detected in testcrosses of each. All of the Df K10-I chromosomes lack a terminal segment of the parent K10-I chromosome including the *Sr2* locus. It should be noted that *R-sr2* crossing over in K10-I/N10 and all Df K10-I/N10 compounds is restricted to a small homologous region between the *R* locus and the left end of the differential segment. In compounds involving K10-I/Df K10-I or Df K10-I/Df K10-I, there is a longer homologous segment, and *R-sr2* crossovers may occur anywhere between the *R* locus and the tip of the shorter of the two homologues.

Compounds 1-4 all show a drastic reduction in *R-sr2* recombination compared to the value of 35% found in N10/N10 bivalents because of the lack of homology in the distal regions of 10L. The lower crossing over observed in K10-I/N10 heterozygotes as compared with Df(C)/N10 (compounds 1 vs. 4) might be attributed to the presence of the knob of K10-I in the former compound, since the adverse effect of knob heterozygosity on crossing over in neighboring regions has been well documented in maize. However, a comparison of compounds 2, 3, and 4 shows that a second factor also plays a role in reducing crossing over. All crossovers classified as *R-sr2* recombinants occur in the short interval between *R* and the left end of the differential segment. Recombination in this short interval is highest (4.3%) in Df(C)/N10 compounds and progressively lower (2.7% and 2.2%) in Df(F)/N10 and Df(H)/N10 compounds, owing to the competitive pairing of the inverted *W2-O7-L13* region with the homologous uninverted region of N10 immediately adjacent to the *R* locus. Attempted synapsis between the two homologous segments, one of which is both transposed and inverted, will interfere with pairing of the

Testcrossed heterozygote	Diagram of bivalent	Sex of Heterozygous parent	% R-sr2	Population Total
(1) K10-I/N10		F	0.8%	3284
(2) Df(H)/N10		F	2.2%	686
(3) Df(F)/N10		F	2.8%	1548
(4) Df(C)/N10		M	4.3%	5058
(5) Df(C)/K10		M	7.5%	4158
(6) Df(C)/Df(H)		M	8.9%	1606

short region distal to *R* where crossing over occurs. No such interference is found in Df(C)/N10 compounds since Df(C) lacks the critical inverted *W2-O7-L13* segment. Even in those instances where pairing was accomplished between the standard and inverted *W2-O7-L13* regions, subsequent crossing over would produce dicentric chromosomes with a low rate of transmission. Thus, Df(F) and Df(H) compounds have a reduced rate of crossing over compared with Df(C), not because of knob heterozygosity since they lack the terminal knob, but because of the presence of the inverted *W2-O7-L13* segment (or a portion thereof). In K10-I/N10 compounds, both factors are operating to suppress crossing over and an even greater reduction occurs. The effect of the knob alone is apparent in the comparison of K10-I/N10 vs Df(H)/N10 (compounds 1 vs. 2) since K10-I differs from Df(H) mainly by the absence of the terminal knob.

Another example where the knob on K10 appears to play a subordinate role is the comparison of Df(C)/K10 vs Df(C)/Df(H) (compounds 5 vs. 6). In these bivalents the region from *R* to the tip of Df(C) is homologous and there is no opportunity for competitive pairing since the *W2-O7-L13* region is present only in the longer homologue and is completely absent from Df(C). In both testcrosses, large populations were scored and in both cases the heterozygous male parent was analyzed. They differ only by the presence of the knob in compound 5. There is 7.5% recombination distal to *R* in Df(C)/K10-I and 8.9% for the same interval in Df(C)/Df(H) compounds. Little if any reduction due to knob heterozygosity was observed. It is true that the K10 knob is not immediately adjacent or included in the synapsed region where crossing over occurs (it is separated from the synapsed region by the intervening *W2-O7-L13* segment) and this may prove to be a significant factor.

That crossing over is low per unit length of chromatin in the homozygous differential segment is indicated by our finding of only 8.9% recombination between *R* and the right terminus of the Df(C) chromosome in Df(C)/Df(H) compounds. Approximately one half of the 8.9% can be ascribed to exchanges between *R* and the left end of the differential segment, leaving only 4.6% derived from exchanges in the homozygous differential segment. More than 30% of recombination occurs in a region of comparable length in the terminal region of N10/N10 bivalents. Neither the Df(C) nor the Df(H) chromosome has the large knob in the long arm of the K10 chromosome, so it cannot be held responsible for the low incidence of crossing over.

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Response to recurrent gametophytic selection

In higher plants it has been demonstrated that gametophytic traits are to some extent under the control of genes which are also expressed in the sporophyte (genetic overlap). Therefore gametophytic selection can lead to correlated sporophytic responses which can involve agronomic traits, too (D.L. Mulcahy, Science 171:1155, 1971; Ottaviano et al., TAG 6:249, 1982).

As part of current research efforts aimed at assessing the real value of this selection in applied breeding programs, a recurrent gametophytic selection was implemented in maize. The preliminary evaluation of both direct and correlated responses is presented herein.

Gametophytic selection in maize is based on the silk length increase from the ear apex downward, which allows the pollen tubes with a higher growth rate to fertilize more frequently at the ear base than at the apex. As a breeding population we used the F_2 of the cross between the inbred lines Mu195 and A632, which show substantial differences for both gametophytic and sporophytic traits (Table 1). One

Table 1. Gametophytic and sporophytic traits of parental lines and selection cycles.

Genotypes	Slope (1) and standard error	Silking date (2)	Leaves per plant (n)	Plant height (cm)	Ear height (cm)
Parental lines					
Mu195	-1.05±0.29	27.7	15.7	154	61
A632	-2.52±0.34	37.5	20.0	163	80
LSD (0.05)		0.5	0.3	4	3
Selection cycles					
Base C2	-0.78±0.33	28.8	17.0	204	86
Base C4	-0.70±0.34	28.5	17.0	203	87
Mean	-0.74±0.24	28.6	17.0	204	87
Apex C2	-1.66±0.43	29.6	17.2	211	93
Apex C4	-1.69±0.38	29.3	17.6	212	94
Mean	-1.68±0.29	29.5	17.4	212	94
LSD (0.05) (3)		0.9	0.4	7	5
LSD (0.05) (4)		0.7	0.3	5	4

(1) Estimate of the relative speed of the pollen tube growth

(2) 1 = July 1st

(3) and (4) Comparison between cycle and means, respectively

hundred full-sib crosses were made between random F_2 plants. Their ears were shelled by taking 25 kernels from the base and as many from the apex, which were bulked according to the ear segment they were taken from, thus obtaining the Base C1 and the Apex C1. In subsequent years, while 100 full-sib crosses were made within each cycle as previously indicated, their ears were shelled by taking 25 kernels at the base only if derived from Base cycles, or at the apex only if derived from Apex cycles.

To evaluate the responses to selection, the 2nd and 4th Base and Apex cycles were compared together with the parental lines in a field trial with 4 replications. The gametophytic trait, i.e. the pollen tube growth rate, was measured in vivo by means of the pollen mixture technique (E. Ottaviano et al., TAG 63:249, 1982). Pollen was collected from at least 100 competitive plants per population, or about 20 plants per parental line, and then mixed with an equal quantity of pollen produced by the standard line W22, characterized by coloured aleurone (dominant marker).

This mixture was then used to pollinate plants of the related F_1 (Mu195 x A632). Fifteen and ten well-fertilized ears were considered for each population and line, respectively, and the pollen tube growth rate, relative to that of the standard line, was estimated as the slope of the regression line of the uncoloured kernel frequency on the ear segment number (from 1 [apex] to 5 [base]). Thus, the higher the slope, the faster the pollen tube growth.

The base C2 and C4 showed negative slopes, whose mean was, however, significantly higher than that of Apex cycles (Table 1), indicating a greater growth speed of Base pollen tubes. It should be noted that the slopes of the two Base cycles were slightly higher than that of the better parent (Mu195), whereas the two Apex cycles were intermediate between the two parents.

As regards the sporophytic traits, which were measured on 20 competitive plants per plot, the Base cycles were slightly earlier, with fewer leaves and with shorter plants then Apex cycles. These results indicate that gametophytic selection led to an earliness increase and to a plant size reduction. It is worth noting that the line with faster pollen tube growth (Mu195) was earlier, and with smaller plants, than the other line (A632).

Since an F_2 was used as the breeding population, the above correlated changes can be ascribed to pleiotropy (genetic overlap) and/or linkage disequilibrium. To have a clearer picture about the source(s) at work, additional cycles of gametophytic selection are needed.

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Teosinte in Oaxaca, Mexico

Teosinte has been relocated in Oaxaca, Mexico after a lapse of 140 years. The Danish botanist Fredrick Liebmann collected 10,000 plants from 1840-43 while living in Pochutla in coastal Oaxaca. This town also happened to speak an archaic form of Danish because its founders two centuries before had been Danish sailors from an English privateer. In his diary for October 1842, Liebmann writes of "over-looking the Pacific", and the locality of San Augustin on the herbarium label narrowed the search to the region of Coatlan or Loxicha in the Sierra of Miahuatlan. After more than 10 years of personal searching, teosinte has been recollected in two localities in Oaxaca.

The reason for the interest in relocating teosinte is that at Mitla, in the Oaxaca Valley, cave sites (2000 B.P.) have yielded beautifully preserved specimens of teosinte and maize x teosinte hybrids. This teosinte introgression profile is only 150 km away from the site of recovery of the prehistoric wild maize (7000 B.P.) at Tehuacan. Teosinte is not known from the Tehuacan remains nor is it found in that region. Therefore Oaxaca is the closest source for teosinte introgression observed in the Tehuacan specimens (3500-2300 B.P.)

Professors E. Hernandez-X. and T.A. Kato-Y. of the National School of Agriculture, Chapingo, Mexico have this last year discovered teosinte in San Francisco Honduras, 1150 m, on a recently opened logging road. This village is 5 km from San Pedro Juchatengo and the teosinte population covers approximately 15 sq km. Reportedly there are other populations up the Rio Miahuatlan, at least this is what the local inhabitants told me. The San Francisco Honduras population flowers in September and mature seed was ready in November. At San Francisco Honduras (settled by people from Honduras in the early part of the century) the slope is to the interior of Oaxaca. The vegetation was pine forest, now opened to seasonally dry cultivated hillsides with scattered pines/oaks.

The population at Loxicha, which used to be called San Augustin Loxicha, is a very small population of less than 2 sq km at an elevation of 1200 m and indeed overlooking, from SW facing slopes, the blue Pacific Ocean 60 km away. This population differs seasonally from San Francisco Honduras and flowers two weeks later, in late September. Both habitats relate to those of the teosinte race Balsas in the neighboring State of Guerrero. Further study will be necessary to characterize the collection, but it is definitely not a Guatemalan type of teosinte, which had been previously thought based on the single Liebmann herbarium specimen. Professor T. Kato is currently studying the chromosome knob positions, which are a diagnostic feature separating the two types of teosinte.

Teosinte is known locally as cocxle, which is interesting because of its similarity to the Nahuatl name for teosinte, cocopi. Cocxle is the Zapotec word for the road runner, a local bird. In the mountains of the Rio Balsas in Guerrero, teosinte is called huiscatote. Huiscatote is the local name for the same bird, the road runner. In other areas of Mexico teosinte is called maiz de pajaro, the generic "bird corn".

Because of the small size of these populations CIMMYT will grow out a large regeneration under cultivation to obtain seed for distribution, therefore seed will not be available until November 1986. The bulk of the wild collected seed is being set aside for future evolutionary studies in long term cold storage where it ought to remain viable for 40 to 60 years. Both wild populations are of limited distribution and are currently subject to grazing and habitat destruction by more intensive land use.

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Golden-2 linkage data

In MNL 57:20, 1983, I reported that golden-2 (*g2*) was located on the short arm of chromosome 3, distal to the breakpoint of TB-3Sb. The present note reports F2 and backcross data involving *g2* and the chromosome 3 genes *d1*, *ra2*, *Rg*, and *gl6*.

The *d1* allele used was *d-6016*, which never gives segregates of intermediate stature. B. O. Phinney and M. Ritzel (MNL 29:44, 1955) reported *d-6016* to be allelic to *d1*, and this was confirmed by us. Good greenhouse classification of *g2* seedlings was possible by their pale green leaves and whitish stems when the plants were grown under conditions of good sunlight. Confirmation of *g2* classification among the dwarf segregates was made by elongating the culms with a single spray application of aqueous gibberellic acid (137.5 mg/l).

The *g2-Rg* data were obtained from *Rg/g2 ra2* x *g2 ra2* testcrosses. The *g2-ra2* coupling data from these crosses are not reported here because of difficulty in scoring *ra2* on *g2* plants which, because of shading, often attain a height of only one or two feet. This problem was not encountered with the *g2-ra2* repulsion progenies.

Tester Mutant	Phase	+ +	+ t	*g2* +	*g2* t	Total	Recomb. Percent
d1	CB	259	111	122	239	731	31.9
	RB	71	143	142	58	414	31.2
						1145	31.6±1.4
ra2	RB	208	355	284	122	969	34.1±1.5
Rg	RB	380	379	340	314	1413	49.1±1.3
gl6	RS	266	98	68	30	462	52.5±3.4

These data place *g2* about 32 map units distal to *d1*, with *g2* a little closer to *d1* than to *ra2*. As the current map (MNL 59:172, 1985) shows *Rg* and *gl6* to be 25 and 27 units to the right of *d1* respectively, if *g2* were proximal rather than distal to *d1* it would have shown linkage with *Rg* and *gl6*; that was not the case. The current map indicates that *cr1* is 18 units distal to *d1*, so *g2* must be distal to *cr1*. The following map is suggested for distal 3S:

g2	*cr1*	*d1*	*ra2*
0	14	32	34

This extends the known genetic length of chromosome 3 by 14 map units.

R. H. Whalen

Further mapping of esterase-4

Initially, *E4* [referred to as *Est4* by A. L. Kahler (Jour. Hered. 74:239-246, 1983)] was placed near the centromere of chromosome 3 because of its linkage with the breakpoint of *wx* T3-9c (3L.09; 9L.12) (J. W. Harris, MNL 42:72-74, 1968). R. A. Kleese and R. L. Phillips (Genetics 72:537-540, 1972) further localized it to the middle of the short arm of chromosome 3 by virtue of its very close linkage to the breakpoint of T3-4(5156) (3S.47; 4L.67).

Our objective is to report linkage data on *E4* with the chromosome 3 genes *g2*, *d1*, and *gl6*. A. L. Kahler has given details of the electrophoretic procedures (Crop Sci. 23:572-576, 1983) and allele (allozyme) nomenclature (Jour. Hered. 74:239-246, 1983) used for this report.

Table 1. Observed numbers (N) of individuals in an F2 population segregating for the three loci *g2*, *d1*, and *E4*.

		E4		N
+	+	2/5	–	83
+	*d*	5/5	–	26
g	+	2/5	–	27
g	*d*	2/5	–	14
g	+	5/5	–	1
g	+	2/2	–	10
+	+	2/2	–	44
+	+	5/5	–	18
+	*d*	2/5	–	7
g	*d*	5/5	–	16
g	*d*	2/2	–	2
			Total	248

Table 1 gives the observed numbers of individuals among the selfed progeny of a *g2 d1* coupling heterozygote that also segregated for the two alleles *E4-2* and *E4-5*.

Contingency Chi-square tests for independence and recombination values calculated using the maximum likelihood LINKAGE-1 program of K. A. Suiter, J. F. Wendel and J. S. Case (Jour. Hered. 74:203-204, 1983) are as follows: For *g2-E4*, $\chi 2$ indep. = 1.89, df = 2, P = 0.388; *g2 - E4* = independent.
For *d-E4*, $\chi 2$ indep. = 75.62, df = 2, P = < 0.001; *d - E4* = 18.76 ± 2.71. For *g2-d*, $\chi 2$ indep. = 20.19, df = 1, P = < 0.001; *g2 - d* = 32.3 ± 5.6. The map is:

g2 32 *d1* 19 *E4*

To verify the above map, we analyzed progeny of the testcross + + *E4-2/g2 d1 E4-5* × *g2 d1 E4-5*, with the results as given in Table 2.

Table 2. Observed numbers (N) of individuals in a testcross population segregating for the three loci *g2, d1,* and *E4*.

		E4		*N*	*Crossover region*
+	+	2/5	–	52	0
g	*d*	5/5	–	54	0
+	*d*	5/5	–	35	1
g	+	2/5	–	30	1
+	+	5/5	–	17	2
g	*d*	2/5	–	13	2
+	*d*	2/5	–	1	1,2
g	+	5/5	–	0	1,2
			Total	202	

The backcross data place *g2* 32.7 ± 3.3 map units to the left of *d1*, confirming earlier results (Whalen, MNL 60, 1986). These data also place *E4* 15.3 ± 2.5 units to the right of *d1* which, according to the current map (MNL 59:172, 1985), indicates that *E4* should be about 14 units to the left of *gl6*. This point was confirmed by analyzing the selfed progeny of a + *E4-3/gl6 E4-2* heterozygote, which gave the following results:

E4	*Gl-*	*gl*
2/2	9	34
2/3	87	7
3/3	54	2

$\chi 2$ indep. = 103.35
df = 2
P = < 0.001
E4 – gl6 = 11.3 ± 2.4 map units

The map of these four genes is: *g2* – 32.7 – *d1* – 15.3 – *E4* – 11.3 – *gl6*.

We thank Joanne Matthees for technical assistance in conducting electrophoretic assays.

R. H. Whalen and A. L. Kahler

Genotypes at enzyme loci in 363 U.S. inbred lines of maize

[Ed. note: Table 1 of the article by A. Kahler in MNL 58:33, 1984, was inadvertently truncated during paste-up of the copy. The missing lines and footnote are reproduced below.]

Table 1 Continued.

Enzyme locus	Isozyme position (cm)	Allozyme numbers[1] lab 1	lab 2
Pgd2	5.0	1	5
	5.9	2	2,8
Enp1	5.4	1	10
	6.0	2	8
	6.3	3	6
	6.4	4	4

[1]Allozyme numbers assigned by authors (lab 1) and C. W. Stuber (lab 2).

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Nuclear DNA content varies between lines of maize and beween maize and teosinte

We have studied variation in 4C nuclear DNA content in the genus *Zea* by microdensitometry of Feulgen-stained, root-tip prometaphase cells. Our results, summarized in Table 1, showed that:

1) There was highly significant variation in 4C DNA content in maize, ranging from 9.84 pg for the single cross hybrid sweetcorn Seneca 60, to 13.49 pg for a Zapalote Chico accession from southern Oaxaca in Mexico (a difference of 37%).

2) The 4C DNA contents of annual teosintes from Mexico and Northern Guatemala were all within the range recorded for maize. However, the DNA contents of different races of teosinte were significantly different ($p < 0.025$). The DNA values for these teosintes were, for the most part, similar to those for three "primitive" races of maize from México (i.e. Palomero Toluqueno, Chapalote and Nal-Tel).

3) *Z. luxurians* (or Guatemala teosinte), a 2n = 20 annual from southern Guatemala, differed from all other 2n = 20 taxa having a much higher 4C DNA content (18.38 pg). This is 87% higher than the value for Seneca 60 (9.84 pg) and 36% higher than the value for Zapalote Chico (13.49 pg).

4) The 4C DNA content for perennial *Z. diploperennis* (10.57 pg) was slightly lower than for other "wild" *Zea*, but within the range recorded for maize. The tetraploid *Z. perennis* had a DNA content close to twice that of diploid *Z. diploperennis*.

Table 1. 4C nuclear DNA content in the genus *Zea* (N.B. All taxa had 2n = 20 chromosomes except *Zea perennis*, which had 2n = 40).

Taxon	Mean 4C DNA content (pg)
Zea mays	
a) maize	
Seneca 60	9.84
Knobless Tama Flint	10.28
Va35	10.31
Oh43	10.58
W64A	10.93
KYS	11.04
Palomero Toluqueno	11.26
Chapalote	11.65
Nal-Tel	11.92
Zapalote Chico	13.49
b) teosinte	
Central Plateau	10.88
Nobogame	11.01
Balsas	11.76
Hueheutenango	12.18
Chalco	12.36
Zea luxurians	18.38
Zea diploperennis	10.57
Zea perennis	21.13

Differences in DNA content between maize lines have also been found in an independent study by H.J. Price et al. (Texas A & M University). They also showed that variation in DNA content is positively correlated with knob number and percent C-band heterochromatin in the haploid component. Our observations are consistent with theirs since, for example, Seneca 60 has only 6 small heterochromatic bands on C-banded root-tip cells while Zapalote Chico has up to 24, including that of the abnormal K10 chromosome. It also seems likely that variation in DNA content is correlated with differences in the amount of heterochromatin in annual teosintes from Mexico and Northern Guatemala, since higher DNA contents were found in the races with the most heterochromatic knobs.

However, variation in the amount of heterochromatin is probably not the only cause of variation in DNA content in *Zea*, since: (i) Knobless Tama Flint did not have the lowest DNA content (Table 1), and, (ii) *Z. luxurians* does not appear to have enough heterochromatin to account for its markedly larger genome.

An interesting feature of the variation in DNA content in maize is its correlation with geographical features. Thus, there appears to be a decrease in both mean knob number and DNA content with increasing latitude of cultivation for maize stocks from the USA. Similarly there is a decrease in knob number (and hence presumably in DNA content) with increasing altitude for maize races grown in Mexico. The fact that such correlations exist suggests that these characters are of adaptive significance. This possibility deserves further study.

One way in which we hope to exploit the variation in DNA content in *Zea* is to construct hybrids where the chromosomes of both parental sets can be identified on the basis of chromosome size and arm ratio. In such hybrids, the spatial distribution of chromosomes from each known parental set could be investigated in a single cell using the serial thin-section, 3-D reconstruction technique.

D.A. Laurie and M.B. Bennett

Replicon size and rate of DNA replication in root meristem cells of Seneca 60

The experiments described here used the single cross hybrid Seneca 60, kindly provided by D.B. Walden, University of Western Ontario.

Seeds were surface-sterilized with 2% sodium hypochlorite and germinated aseptically in petri dishes at 20 C in darkness. When the seedlings were four days old the apical 1 mm of the tips of 3 cm-long roots were processed either as fibre autoradiographs for replicon measurements, or as squash preparations for cell cycle and DNA synthetic (S)-phase determinations.

For the fibre work the root tips were exposed to high specific activity (70-90 Ci $mmol^{-1}$) tritiated-[methyl-^{3}H]-thymidine (^{3}H-TdR) at a concentration of 1m Ci ml^{-1} for 30, 45, 60, 90, 120, 150 or 180 min at 20 C. Root meristem nuclei were then isolated and digested with trypsin on subbed microscope slides at 37 C. Sarkosyl and EDTA were then added and the resultant slurry, containing naked DNA, was spread across the slide. Slides were dried overnight in formalin fumes, treated with ice-cold trichloracetic acid, dehydrated with alcohol, air dried and prepared as permanent autoradiographs using Ilford K_2 photographic emulsion. The exposure time was two to four months. Labelled DNA spread in this way is represented by tandem arrays of silver grains running across the slide. Replicon size is estimated by measuring the distance from the gap between a pair of these arrays to the corresponding gap in an adjacent pair. The relationship between mean length of labelled DNA segment and exposure time to ^{3}H-TdR gives an estimate of the rate of single replicon fork movement, and dividing average replicon size by twice the rate of fork movement gives the time required for a replicon to replicate its allotted DNA (Rs).

For root meristem cells of Seneca 60 at 20 C, modal replicon size was 15 to 20 μm (about 45 to 60 kb of DNA), the rate of single fork replication was 3.0 μm h^{-1} (approximately 9 kb h^{-1}), and Rs was 2.7h.

For the cell cycle work, roots of four-day-old seedlings were exposed to: ^{3}H-TdR (S.A. 5Ci $mmol^{-1}$; concentration 1μCi ml^{-1}) for 0.5h, then unlabelled TdR (10^{-5}M) for 0.5h. Next they were grown on moistened petri dishes at 20 C in darkness. Roots were fixed in 3:1 (v/v) absolute ethanol: glacial acetic acid every 2h for 24h after the start of ^{3}H-TdR exposure. The apical 1 mm of the root tips were processed as Feulgen-stained squash preparations. These were made into permanent autoradiographs. The exposure time was 14 days. In each preparation 100 mitoses were scored for the presence or absence of silver grains, and hence for incorporation of ^{3}H-TdR, in a series of random transects across the slide. These data were plotted against time after the start of labelling to give a percentage labelled mitoses curve (PLM). The duration of S-phase (about 4.7h) was estimated as the interval between the 50% intercepts of the ascending and descending limbs of the first peak in the PLM curve minus the 0.5h labelling period. The duration of the cell cycle (about 12 hours) was estimated from the mid-point of the first peak in the PLM curve to the corresponding point on the second peak.

The mean time for a replicon to replicate its DNA (Rs), and the duration of S-phase (Ds), were used to calculate an Rs:Ds ratio, a parameter which is positively correlated with synchrony of replication

activation within a genome. The Rs:Ds ratio of 0.60 for Seneca 60 is within the range of values known for angiosperms (i.e. 0.11 - 0.71), and close to the values estimated by us for some other cereals (e.g. barley - 0.55; and hexaploid triticale - 0.53).

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Genetics of how corn ticks

In MNL 58:38-46 we reported a generally consistent association in nearly all chromosomes among *Krn* (kernel row number), *Flt* (flint or dent endosperm type), and *Ger* (glucoside earworm resistance), including *Mer* and *Zer* (Maya and Zapalote Chico earworm resistance). Chromosome # 4 was not covered and in # 10 only the flint factor was detected.

M.M. Goodman, C.W. Stuber, K.J. Newton & H.H. Weissinger (MNL 54:101-102) reported two parallel cases of linkage in isozymes, one involving MDH (malate dehydrogenases), and PGM (phosphoglucomutases), and the other between IDH (isocitrate dehydrogenases) and MDH pairs. They concluded that the duplication of chromosome segments involving isozyme loci may not be as uncommon as the cytological evidence would imply. Going one step further the logical sequences in Table 1 can be posed, using K for Krebs cycle, EMP for Embden-Meyerhoff-Parnass cycle, and PS for Pentose Shunt. Data are from working maps (MNL 59:168-186), from M.M. Goodman et al. (Maize for Biological Research, 1982), articles of J.F. Wendel et al. (MNL 59:87-90), and Genetic Maps Vol. 2 (Cold Spring Harbor Laboratory). In the Table are shown 5 sequences of the respiration cycle in which there are isozyme duplications and/or overlapping involving chromosomes 1L, 3, 4S, 6 and 8.

Table 1. Isozymes of respiration cycles, in biochemical order, which overlap along different chromosomes. It seems so far as if each chromosome has the complete set.

		Chromosomes				
		1L	3	4S	6	8
EMP	Phosphoglucomutase	Pgm1				
	Hexokinase		Hex1		Hex2	
	Phosphoglucoisomerase	Phi1				
	Phosphotrioseisomerase		Tpi4			
PS	Glucose-6-phosphate dehydrogenase		Pgd2		Pgd1	
K	Aconitase			Aco1		
	Isocitric dehydrogenase				Idh2	Idh1
	Malic dehydrogenase	Mdh4	Mdh3		Mdh2	Mdh1
	Malic enzyme		Me1			
A	Alcohol dehydrogenase	Adh1		Adh2		

Once it is seen that the respiration cycle is repeated among all chromosomes, the next step is to see whether other biochemical cycles are present and if they are in order within a chromosome. Rough sequences can be identified more or less in the following order: glucosides, anthocyanin and endosperm factors, the Krebs cycle, Embden-Meyerhoff-Parnass and Pentose Shunt, and the block *Krn Flt Ger*. They are more developed (not necessarily in this order) in 1S *p1 dek1 Car Cat2*, in 1L *bz2 Cat3 Mdh4 mmm Pgm1 Adh1 Phi1 Dia (Ger5 Flt11 Krn11)*, in 3L *E Hex1 Tpi4 Pgd2 Me a3 Mdh3 a1 sh2*, in 5 *Pgm2 Amy2 Mdh5 Cat1 a2 ps1 bt1 ae pr1*, in 6L *Pgd1 Dt2 Pl Bh1 su2 sm Hex2 Idh2 Mdh2* and in 8 *Mdh1, Idh1 Tpi3* all with *Krn Flt Ger* somewhere near.

Besides the sequences shown, using data from J.M. Chandlee and J.G. Scandalios (MNL 58:172-177) we can pose in chromosome 9 the sequence *Dt1 Dt5 C1 sh1 bz1 Atc1 Acp1 wx1*; *Acp1* is acid phosphatase; *Sh1* is sucrose synthetase; probably *C1* and *C2* have control of flavanone synthase (FS); *Bz1* is UDP-glucose: flavonoid 3-0 glucosyltransferase (UFGT); *Wx1*, starch-granule-bound nucleoside diphospho-glucose-starch glucosyl transferase; and somewhere *Krn9 Flt9 Ger4*. In 4S we have *Asr Bx=Mer?, su1 Krn4 Adh2 Dt6 Aco1* (*su1* is deficient in α-1,6 glucosidase debranching enzyme), and in 4L *Zer1 c2*. In 2, *B fl1 Dia1 Zer3 Flt2 Krn2, Dia* is diaphorase. In 10, *Sad1 bf2 Cx Glu1 R1*, we have a trend for aromatic amino acid biosynthesis, the shikimic acid pathway with *Sad1* (shikimate acid dehydrogenase) and *bf2* (blue fluorescent) if similar to *bf1* yielding anthranilic acid, and also *Cx1* (catechol oxidase), which turns phenols to quinones, toxic to pests in wounds, and *Glu1* (beta glucosidase) which splits saccharose or maltose yielding glucose, and the cluster distributing anthocyanin pigments around *R1*. The shikimic acid pathway through phenylalanine leads to the C-15 flavone skeleton.

In corn, the hint is usually for the Krebs cycle isozymes being nearer the glucosides, anthocyanin and endosperm factors, farther away usually on the same side are those for Embden-Meyerhoff-Parnass cycle, Pentose Shunt still farther away not necessarily on the same side, and the *Krn Flt Ger* block somewhere along the line.

According to the Report of the Tomato Genetics Cooperative, number 30, 1980, in chromosome 4 we find *Tpi-2* (triosephosphateisomerase) 6 units from *Pgm2* (phosphoglucomutase), both from the EMP cycle. In the anthocyanin series we find in chromosome 1 *y* colorless fruit epidermis, 2 units from *Prx-1*, (peroxidase-1): in 2 the sequence is *Prx-2* or *Prx-3* with *aa* (anthocyanin absent), *are* (anthocyanin reduced), and *aw* (without anthocyanin); in chromosome 3 is *pdc* (pudica), plant retarded dark pigmented (*Prx-7*) and *r* (fruit flesh yellow); in 9 *ah* Hoffman's completely anthocyaninless with *Est-2*.

So *Px* and correlated corn isozymes should also be searched near the above associations with the standard anthocyanin marker series, including also *dek1* and *vp1* and not forgetting the anthocyanin

block in 6L for completeness. Possibly there is still an unknown anthocyanin gene yet to be found in 4S, and perhaps in 8L.

In human genetics, V.A. McKusick and F.H. Ruddle (Science 196:390-405) comment: "Clustering of genes with similar functions suggests a biologic significance of the association. The genes for three enzymes (98) involved in the Embden-Meyerhoff glycolitic pathway, GAPG (glyceroaldehyde-3-phosphate dehydrogenase), *TPI* (triosephosphate isomerase), and *LDH-B* (lactate dehydrogenase B) are on chromosome 12. The tight linkage of *TK* (thymidine kinase) and *Galk* (galactosidase) is found not only in man and Chimpanzee (171) but also in Chinese hamster and in *Mus musculus*. The genes for *GOT-1* (glutamate oxaloacetic transaminase-1) and *GSS* (glutamate-y-semialdehyde synthetase) enzymes involved in proline metabolism are on chromosome 10", (sic) etc. . . We should further point out IDH and MDH in the short arm of chromosome 2, again paired as in corn.

It seems so far, pooling corn and tomato data, that each chromosome is a complete ordered assembly line, comprised of glucoside, anthocyanin or endosperm factors, Krebs cycle, and Embden-Meyerhoff-Parnass cycle and Pentose Shunt usually in that order, and with the *Ger Krn Flt* block probably near the linking point. The biochemical link between both these groups, respiration and synthesis, is probably the Pasteur effect. Long ago Pasteur noted that the path of yeast metabolism could be affected by oxygen: low oxygen favored fermentation, whereas high oxygen inhibited fermentation and stimulated oxidative respiration, as well as promoting the use of carbons from sugars for synthetic reactions. *Px* is probably involved in this assembly judging from tomato data, because it oxidizes NADH produced in respiration processes to NAD, which is necessary for the operation of the respiration cycles. There is a distinct possibility that these systems can be at least partially duplicated within a chromosome: in chromosome 1 around genes *p dek1*, and *bz2*, and in chromosome 4 (see also MNL 59:23-24) around *Asr1* and *c2*, in both cases about 100 units apart. How can this apparent independence be reconciled with the gene complementary action among blocks marked by anthocyanin genes? Are they some kind of regulators or pacemakers to the same processes?

Compare the distribution of the blocks described with the cytological map in G.F. Sprague, ed., Corn and Corn Improvement, 1977. Taking aside the structures beside the centromeres and nucleolus organizer and satellite in chromosome 6, we verify that there is a rough correspondence with the knobs and prominent chromomeres with the blocks in chromosomes 1, 4, 5, 6, 8, and 10. Chromosomes 2L, 3S, 7S and 9L are discrepant, which could maintain other linked processes. If no more tandem duplications are found, 1 should be homologous to 4 and 2 homologous to 10 by parallelism in the latente super-genes, which are very much elaborated. Finally, note that our conclusions agree and explain much of P.C. Mangelsdorf's results in 4 on the genetic nature of teosinte, pp. 37-52 in Corn, its Origin, Evolution and Improvement, 1974, especially in the linkage relations of teosinte characters with each other and with marker genes of maize. There are several independent blocks (super-genes) producing the same effect with reinforcing action. By our data there seem to be about 11 or 12 such blocks, instead of the 4 or 5 or 6 currently accepted, affecting floral and fruit characteristics, including kernel row number. In the next article, quantitative estimates of earworm resistance and kernel row number are presented for the groups around *P-WW, B* and *Plp*, showing their complementary dominant mode of action.

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Genetics of horizontal resistance: measurement of the effects of *B, P-WR, Plp* and *Krn* on corn earworm resistance and yield

In MNL 58:38-45, linkages of resistance to corn earworm (*Heliothis zea*) with *B, P-WR, Plp* and *Krn* were shown using the standard *wx* set of translocations in IAC Maya crossed to Purple Husk Cateto (PHC), and back-crossed to Maya *wx*. To preserve heterotic patterns we transferred these factors to the IAC-1 maize cultivar which is about 50% Cateto and Colombian flint and 50% Tuxpeño. IAC Maya is nearly all Tuxpeño and to it we tried to transfer Zapalote Chico resistance factors. In a modified ear-to-row scheme with reps of IAC-1, in the detasseling plot 25 female ear to row half-sib families were planted with the original cross and two backcrosses to IAC-1. PHC is *B, P-WR, Plp* and IAC-1 *B-W, P-WW, pl.* In the generation PHC x IAC - $1^{(3)}$ a reading was done for unholed husks, husks with the earworm exit hole, for the anthocyanin factors above, and for kernel row number. Our *B* gives purple color to the outer husks, usually shaded not a solid color, and our *B-W* gives no husk color. *P-WR*, corn cob color is an inadequate name, really it gives a reddish color to the inner soft bracts (lemma and palea), and *Plp* gives purple color to the outer hard bracts, the glumes. The data are tabulated in Table 1. For the total of the 25 families, a χ^2 was applied contrasting unholed minus holed for all genetic combinations, including 16 and more kernel row numbers, 14 rowed, and 12 are less rowed number, forming a 2x2x2x3 factorial design. The χ^2 are positively significant for + + + -, and + - + 0, and negatively for the - + - - combinations at P = 0.05. Quite unexpectedly *P-WW* from the recurrent male was better than *P-WR* in combinations with *B*, and *Plp* was better than the original combination from the

Table 1. Fenotipic classification of ears studied, unholed U, with exit hole H, of all 25 families, and of selected 8 with the treble linkage with B, P-WW and Plp, and weight in grams of ears.

B / B-W	P-WR / P-WW	Plp / plp	Krn 16 14 12	All 25 families U	All 25 families H	All 25 families U-H	All 25 families χ^2	Selected eight U	Selected eight H	Selected eight U-H	Selected eight χ^2	All 25 families ear weight grams U	All 25 families ear weight grams H	Selected 8 families weight in grams U	Selected 8 families weight in grams H
+	+	+	+	7	11	-04	0.88	4	4	0	--	179	166	196	173
+	+	+	0	35	39	-04	0.21	11	7	+04	0.89				
+	+	+	-	41	24	+17	4.44*	8	6	+02	0.29				
+	+	-	+	12	7	+05	1.30	5	1	+04	--	177	161	180	185
+	+	-	0	24	17	+07	1.19	7	8	-01	0.07				
+	+	-	-	27	16	+11	2.81	7	8	-01	0.07				
+	-	+	+	13	9	+04	0.72	9	2	+07	4.45*	188	175	207	188
+	-	+	0	52	31	+21	5.31*	24	10	+14	5.76*	13,12**			
+	-	+	-	39	35	+04	0.21	15	7	+08	2.91				
+	-	-	+	9	5	+04	1.14	3	1	+02	--	167	167	162	162
+	-	-	0	21	18	+03	0.23	5	2	+03	--				
+	-	-	-	23	20	+03	0.21	4	6	-02	0.40				
-	+	+	+	0	0	0	--	0	0	0	--	155	107	--	--
-	+	+	0	3	2	+01	--	0	0	0	--				
-	+	+	-	0	2	-02	--	0	0	0	--				
-	+	-	+	14	19	-05	0.76	3	7	-04	1.60	172	170	199	186
-	+	-	0	35	47	-12	3.46	13	17	-04	0.53				
-	+	-	-	29	51	-22	6.05*	12	14	-02	0.15				
-	-	+	+	2	1	+01	--	0	1	-01	--	182	124	185	130
-	-	+	0	1	6	-05	--	1	2	-01	--				
-	-	+	-	2	3	-01	--	1	1	0	--				
-	-	-	+	14	21	-07	1.40	5	8	-03	0.69	167	162	199	179
-	-	-	0	36	48	-12	1.71	19	17	+02	0.11				
-	-	-	-	44	46	-02	0.04	14	14	0	--				
Total				483	478	+05	32,15*	170	143	+27	17,92	1387	1232	1328	1203
Index				101	100			119	100			113	100	110	100

Table 2. Coeficients of the factorial analysis 2x2x2, B, P-WR, Plp with their standard errors calculated with data from Table 1, U=unholed, H=holed with data for the total 25 progenies transformed from 939 to 100 sample size. Variance base (0.5x0.5)÷n. The -++ and --+ combinations were not included in the analysis since Plp depended on the presence of B. Analysis of mean weight in grams of ears, with reps taken as 22,7 and mean variance within treatment of 15,7 grams. U=unholed, H=holed by earworm. Second line of line pairs refers to the analysis of eight families with simultaneous linkage of unholed with B, P-WW and Plp

	$\bar{x}$	B	P-WR	Plp	B P-WR	B Plp	P-WR Plp	B P-WR Plp	U-H
(25) %U-H	= 50,6	+2.5 ±6.0	-0.4 ±2.3	+1.3 ±6.0	+0.2 ±2.3	-1.2 ±6.0	-0.3 ±2.3	-0.9 ±2.3	
(8) %U-H	= 54,7	+2.5 ±10.5	-1.7 ±4.1	+3.1 ±10.5	-0.8 ±4.1	+0.6 ±10.5	-0.7 ±4.1	-1.7 ±4.1	
(25) Weight	=170.9	+2.4*±1.2	-0.1 ±0.5	+4.6**±1.2	-2.2**±0.5	+2.2 ±1.2	-2.2 ±0.5	-0.7 ±0.5	+4.1**±0.5
(8) Weight	=184.6	-4.6*±2.2	+1.8*±0.8	+4.7* ±2.2	+0.7 ±0.8	+9.3**±2.2	-6.2**±0.8	-5.0**±0.8	+5.8**±0.8

"resistant" non-recurrent source. To clarify the interactions, a further analysis was done for the 8 families in which there was the treble simultaneous linkage of resistance with *B, P-WW, Plp*. So filtered the results get crystal clear. The best combinations concentrated all in the + - + cell with all row numbers, with two significant deviations. Furthermore, correcting within it the χ^2 for family size n, $(\bar{n} \div n) \times \chi^2$ they turn to 9.03 for 16 rows, 3.78 for 14 rows and 2.95 for 12 rows. To quantify the effects a standard analysis was done as a 2x2x2 fractional factorial eliminating from the analysis -+ + and --+ cells and disregarding *Krn*, Table 2. The coefficients of the variables were adjusted to a sample size of 100 so the effects are presented in percentage. Note that for the group of 8 selected for earworm resistance if the treatment is *P-WW* (-), all effects including the third order interaction turn to positive, summing up to +11.1 over the mean. The numbers within a *Krn* class are insufficient to get a meaningful analysis, they should reach 22 or 23 family size to get good results, getting a binomial distribution. Pooling kernel row numbers in each of the 2x2x2 cells the ears were also weighted in grams, and a mean ear weight was obtained. With the mean weight, again a fractional factorial 2x2x2, *B* x *P-WR* x *Plp* was gotten and analysed. This fractioning increases enormously the error of estimation of *B, Plp* and *B Plp* coefficients. The best combinations for earworm resistance tended also to be the best yielders, 201 g for the unholed and 188 g for the holed for the + - + combination, confirming that greater earworm resistance also brought greater yield. As this is also a breeding program we are continuing the work with the eight families, and eliminated all *P-WR* individuals. Next breeding season we expect to do new readings so the available coefficients can be more accurately measured.

As our recurrent theme is horizontal resistance to pests, remember that as far as is known, *Plp* only appears if *B* is present. *B* spreads color (and resistance?) also to the coleoptile, roots, and the husk, the critical beginning and end of the plant cycle. The standard *Pl* allele includes the glumes and permits expression of all *B* effects in the dark. Yes indeed, as for an environmental resistance system *lte1*, which doesn't include pests, corn has evolved an horizontal resistance to pests which completes the first. The preceding article explains much of why these effects are quite measurable and parallel.

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Segregation of red vs. white color in cob and tassel glumes

E.H. Coe has reported a correspondence between brown tassel glumes and red cob color (MNL 57:33, 58:75, 59:40). Pigmentation in both tissues is apparently controlled by the *P* locus, which also controls pericarp color. Coe indicates that in most families which are segregating for red vs. white cob color (*P-WR* vs. *P-WW*), plants with brown tassel glumes produce red ears, and plants with white tassel glumes produce white ears.

Plants carrying the *P-VV* allele produce ears with variegated pericarp and cobs, and white tassel glumes. *P-VV* has been shown by Brink and others to result from the presence of the transposable element *Mp* (equivalent to *Ac*) at the *P* locus. Mutations of *P-VV* to *P-RR* arise by excision of *Mp* to restore *P-RR*, resulting in red pericarp and cob. It was of interest to examine the segregation of cob and tassel color in the progeny of a plant carrying a *P-RR* allele derived as a mutation from *P-VV*. A plant carrying the revertant *P-RR* allele heterozygous with *P-WW* was selfed and progeny were scored for colors of tassel glumes and cob. Fourteen plants with brown tassel glumes produced red pericarp, red cob ears; 7 plants with white tassel glumes produced white pericarp, white cob ears. In this case, reversion restored *P* expression in the tassel glumes as well as pericarp and cob.

The correlation was also tested with another *P* allele known as *P-Quebec36*. This allele conditions a faint red pericarp, red ear phenotype. A plant of genotype *P-Quebec36 ts2/P-WW* + was selfed (*ts2*, situated 2 map units distal to *P*, determines a tassel seed phenotype.) Progeny grown from this self comprised 16 with normal tassels, and 5 with the tassel seed character. Of the plants with normal tassels, 10 had brown tassel glumes and produced red cobs, while 2 had white tassel glumes and produced white cobs. No attempt was made to score tassel glume color on the tassel seed plants, but these produced red ears. Ears from the remaining plants were lost.

Thus, in these 2 small F2 families, pigmentation of tassel glumes and cob was always correlated. (This material is based upon work supported by the National Science Foundation under a grant awarded in 1984).

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Isolation of a candidate clone of the maize *P* locus

The *P* gene controls pigmentation of the pericarp and glumes of the cob and tassel. Many variants in *P* expression are known, including types displaying altered tissue specificity, and variegated types. Schwartz has presented a model of *P* expression (D. Schwartz, Proc. Natl. Acad. Sci. 79:5991-5992, 1982) involving a hypothetical regulatory mechanism termed presetting by McClintock (B. McClintock, Carnegie Inst. Washington Yearb. 63:592-601, 1964).

The *P-VV* allele, which conditions variegated pericarp, has been shown by Brink and others to result from the effects of the transposable element *Mp* (functionally equivalent to *Ac*) situated at the *P* locus. Since the *Ac* element has been cloned previously (Fedoroff et al., Cell 35:235-242, 1983), *Ac*-specific sequences can be used to isolate the *P-VV* allele.

DNA prepared from maize seedlings carrying a *P-VV* allele was digested with restriction endonuclease *Sal*I. The *Sal*I fragments were ligated into *Sal*I-cut phage vector EMBL3. The resulting library was screened with a probe prepared from an internal fragment of the *Ac* element. A hybridizing clone was detected which carried an 8 kilobase pair *Sal*I fragment containing a full-length *Ac* element. Sequences flanking the *Ac* element were used as probes for Southern blot analysis of DNA obtained from stocks carrying *P-VV* (variegated pericarp) and a *P-RR* (red pericarp) allele derived as a revertant of the unstable *P-VV* allele. When these DNAs were restricted with *Sal*I and probed with the presumptive *P*-specific probe, the *P-VV* DNA contained an 8 kilobase pair-hybridizing band, while the *P-RR* DNA contained a 3.5 kb band. This result is consistent with the presence of the 4.5 kb *Ac* element in the *P-VV* allele, and loss of the *Ac* element upon reversion to a *P-RR* allele.

The DNA flanking the cloned *Ac* element was sequenced. Surprisingly, the *Ac* element was not associated with a short (8 bp) direct repeat found flanking other *Ac* elements. The generation of short direct repeats upon insertion is a feature of nearly all transposable elements studied to date, and the significance of their absence in this particular case is unknown.

Starlinger and coworkers are also studying the *P* locus (see report in this issue). They report the isolation of an 8 kb *Sal*I fragment derived from *P-VV*. The independently isolated fragments are apparently identical, and sequence comparisons indicate that in both cases the *Ac* element is situated at the same nucleotide position, lacking an 8 bp repeat.

The pedigree relationship of the stocks used by each group is unclear, but we assume that these *P-VV* alleles are identical. (This material is based upon work supported by the National Science Foundation under a grant awarded in 1984).

Thomas Peterson and Drew Schwartz

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Centro de Genetica

Photosynthetic pigments in maize and teosinte

In a preliminary search for photosynthetic pigment differences in leaves of maize and Chalco teosinte by using the technique of thin-layer chromatography as described by Hirayama (J. Biochem. 61(2):179-185, 1967), two pigments were found in maize that were not present in teosinte. With this information it was thought to be worth analysing a larger sample of both maize and teosinte from various regions in Mexico where they grow in sympatry, in order to see to what extent this difference between these plant species is common. During September to October of 1984 and 1985 several trips to the states of Guanajuato, Guerrero, Jalisco, Mexico, Michoacan, Nayarit and Oaxaca were made to collect green leaves from sympatric maize and teosinte plants. A total of 29 paired samples were obtained.

Preliminary tests showed that leaves of 4-5-week old seedlings were as good as those of mature plants for the chromatograms. This allowed us to run pigment analyses on seedling leaves from seed originating both from previous collections from several localities not visited this time and from seed received from other investigators. Among these latter localities were those of the states of Chihuahua and Durango for Mexico, and Huehuetenango and Jutiapa for Guatemala.

The results obtained are as follows: in all maize collections the chromatograms showed the presence of 9 pigments expressed as 9 distinct bands; three pigments differentiated teosinte collections from maize collections. Two of them (pheophytin *a* and lutein) found in all samples of maize leaves analyzed (29 samples, 232 plants) were absent in those of teosinte (47 samples, 408 plants). A third pigment, not yet well characterized, was found only in the chromatograms of 21 of the 47 teosinte samples, and absent from all maize samples.

In order to learn about the inheritance of two of the differential pigments (pheophytin *a* and lutein) between maize and teosinte, leaves of F_1 and F_2 of the cross between an S_5 line of the race Cónico and a Chalco teosinte, and of its BC_1 to maize were chromatographically analyzed. It was found that both pigments present in maize were absent in the F_1's chromatograms, indicating absence of pigment as dominant. Furthermore, lutein segregated 3:1 in the F_2 and 1:1 in the BC_1 progenies, indicating this pigment follows a simple Mendelian inheritance. In the case of pheophytin *a*, no one F_2 or BC_1 plant showed the pigment, indicating that its inheritance is more complex and needs further study. Since the third pigment is absent in Chalco teosinte, it was not possible to get any information about its inheritance. However, in two of the teosinte collections from a southwest state of Mexico it was found that the pigment was segregating among different plants, a fact that at least suggests that it is genetically controlled.

All this information has been interpreted in the sense that it constitutes a further evidence in support of the hypothesis postulated, based on chromosome knob data, that between sympatric maize-teosinte populations introgression has not occurred for a long time (probably several millenia) and it is not actually occurring, in spite of the hybridization that constantly produces fertile hybrids. (This investigation has been partially financed by the Consejo Nacional de Ciencia y Tecnología of Mexico under the grant PCAFBNA-005293).

T. Angel Kato Y. and Mario A. Vargas O.

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A continued study on the stability of anther-culture derived maize callus lines

In last year's MNL (p. 29), we reported the results of a study on the cytological stability of three maize callus lines derived from anther culture in vitro. It was found that among the three lines, one (G7) gradually changed from haploid to diploid chromosome number ($2n = 2x = 20$), and two (N1 and G9) tended to maintain a haploid level ($2n = x = 10$) with over 90 percent of the cells having 10 chromosomes, after subculturing for three or more years. As a continued study on two more callus lines was carried out, it was observed, with great interest, that one of these lines evolved toward a polyploid constitution while the other manifested a stable dihaploid condition. In the first year of culturing of these callus lines, their chromosome number was consistently identified to be 10. The increase of chromosome number occurred following the first year of growth. This can be accounted for either by endomitosis or by fusion of adjacent cells. However, why the process stopped for one callus line (Dan-Sun 91) after doubling chromosome number once and continued for another (81-B5) is unclear. For the latter (81-B5), varying chromosome numbers from 30 to more than 100 were identified in different cells.

Y.C. Ting and M.G. Gu

Unstable progeny of a dihaploid maize line

In the last summer, about 70 kernels of a self-fertilized plant which originated from a maize muta-

ble plant height strain were sown. This maize strain, a dihaploid with $2n = 2x = 20$, descended from a microspore-plant by natural chromosome doubling. Two weeks after sowing, 47 healthy seedlings were obtained, indicating a more than 60 percent seed viability. All of them looked alike. In other words, uniformity was persistent in the early stage of growth. However, about six weeks after planting, some plants began to show slow growth, leaf-chlorophyll deficiency and gradual weakening. Within a short period these plants would die.

When the living plants reached tasselling stage, only 21 individuals survived. Among them, variability in number of ears and plant height was striking. For plant height, it ranged from 27.5 to 183 cm, and for number of ears per plant, it ranged from 1 to 3. In addition, two of the 21 plants bore tassel seed. Self-pollinations for a selected number of plants were made. Further studies will be carried out on the progeny instability of the plants.

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Dual sources of abscisic acid in maize kernels

Abscisic acid (ABA) is the plant hormone most commonly implicated in dormancy induction events in plants, and the premature sprouting associated with the viviparous mutants of maize has long been suspected to result from an ABA deficiency in the embryos. That this is true was first reported by Brenner, et al. (Plant Physiol. 59:S76, 1977), who reported that 24 days after pollination (DAP) ABA concentrations in mutant embryos were only 10-50% as high as in normal embryos. In our experiments with 20 DAP embryos of *vp2, vp5, vp7, vp9* and *w3*, ABA levels in homozygous mutant embryos were 20-37% as high as we determined for normal homozygous and heterozygous embryos segregating on the same ears, although older embryos had levels as high as 60% of wild type levels.

At least three alternative explanations would appear to exist for the reduced, but significant, levels of ABA found in viviparous embryos. The first of these relates to the biosynthetic pathway for ABA. Two pathways have been proposed, but neither has been critically established. The direct pathway suggests that ABA is derived from a 15-carbon precursor, farnesyl pyrophosphate. The indirect pathway suggests that ABA is derived from a 40-carbon carotenoid, probably violaxanthin. Since the viviparous mutants mentioned previously are all carotenoid-deficient, the observations concerning reduced ABA content of viviparous embryos could be explained if both pathways were operative in maize. This hypothesis presumes that ABA synthesis via the carotenoid pathway is blocked in the mutants, and that the ABA observed in the mutant embryos is synthesized via the farnesyl pyrophosphate pathway.

A second possible explanation is that ABA is actually synthesized via the carotenoid pathway in maize embryos, but the mutations are leaky. However, *vp5* blocks the 11-12 C desaturation and accumulates phytoene, *w3* blocks 11′-12′C desaturation and accumulates phytofluene while *vp7* blocks the second cyclization reaction and accumulates γ-carotene and δ-carotene. Since we have not identified α-carotene, β-carotene, lutein or zeaxanthin, which are distal in the pathway to the intermediates that accumulate in the mutants and are normally present as relatively large pools, these mutants do not appear to be leaky with respect to carotenogenesis. Under these circumstances, the mutants could not be leaky for ABA synthesis if ABA is actually a terminal carotenoid derivative.

A third possible explanation is that part of the ABA in the embryo, "maternal ABA", is synthesized in the plant and moves into the developing caryopsis through the phloem transport system, and the rest of the ABA, "in situ ABA", is synthesized within the kernel. The large day to day and plant to plant variation in embryo ABA content which we have observed has caused us to suspect a maternal contribution for some time. We determined that ^{3}H-ABA (18 Ci/mM) injected into the shank of the ear could be recovered from the kernels. While this showed that ABA could be translocated into the kernel, it doesn't show that such transport actually operates in the plant. However, when Karssen, et al. (Planta 157:158-165, 1983) reported dual sources of ABA in seed of *Arabidopsis thaliana*, we were convinced that a similar system exists for maize.

It finally occurred to us that a remarkably simple experiment, based upon the fact that ABA is rapidly metabolized, could be used to demonstrate dual origins for ABA in maize kernels. If viviparous mutant kernels or embryos are rescued, the albino seedlings can be cultured for a period sufficiently long to allow maternal ABA that was initially present in the embryos to be metabolized. Thus, such seedlings should not contain ABA if in situ ABA synthesis is completely blocked and all of the ABA in the mutant embryos is of maternal origin.

Table 1 summarizes the results of several experiments. Although the failure to detect ABA in homozygous mutant seedlings does not prove that no ABA is present, it argues very strongly that the ABA found in the mutant embryos actually is of maternal origin. The corollary of this argument is that the difference between ABA contents of wild type and mutant embryos from the same ear should provide a rough estimate of in situ ABA in wild type embryos.

In addition to establishing that the ABA in maize embryos has dual origins, our failure to detect

Table 1. Summary of average ABA contents determined for embryos and seedlings of homozygous mutants and wild type segregates from the same ears.

	Embryo 20 DAP (% of wild type)	Seedling 14-16 d (ng ABA g^{-1}FW±SD)
Wild type	100	337±78
vp5	25	ND[1]
vp7	20	ND
vp3	33	ND

[1]ND = None detected with an analysis sensitivity of about 4 ng ABA g^{-1}FW

ABA in homozygous, carotenoid-deficient seedlings supports the hypothesis that ABA is synthesized via the carotenoid pathway in maize.

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Studies of *Ac*-derived RNA

We want to study the transcription of transposable element *Ac*. We have probed mRNA from seedlings and from endosperm with probes derived from *Ac*. We detect a 3.5 kb band that we have seen in *Ac*-containing tissue only, and two smaller bands that are also found in RNA from *Ac*-free plants.

cDNA clones have been prepared. Those clones derived from the small bands are most probably not derived from *Ac*, but seem to be readthrough products of *Ds* insertions in active genes. cDNA clones derived from the 3.5 kb band are identical to *Ac* in those segments where the sequence has already been determined definitely. They differ from the *Ac* sequence by the absence of four introns together comprising 650 bp. The three cDNA clones span a length of about 3 kb. Approximately 500 bp from the 5'-end of the RNA have not yet been found in the form of the cDNA clone. We are presently trying to establish a full length cDNA clone.

Our experiments up to now show a single transcript which tentatively starts about 150 bp inside of *Ac* and ends at a position 264 bp from the other end of *Ac* with a polyadenylation signal. The present data indicate the presence of one long uninterrupted coding frame of 807 amino acids length and the presence of a smaller open reading frame of 102 amino acids length that is completely contained in the first one near its end terminus.

Reinhard Kunze and Peter Starlinger

Studies on the expression of the transposable element Activator at the protein level

In the last issue of MNL (59, p. 33, 1985) the sequence of the transposable element Activator was reported. Of the open reading frames, at least ORF1 is likely to encode a gene product, because a 194 bp deletion leads to loss of *Ac* activity and creation of the passive element *Ds9* (Pohlman et al., Cell 37:635, 1984).

DNA segments of ORF1 therefore seemed most suitable for the construction of expression vectors with the aim of expression of an *Ac*-specific protein or a part of it in *E. coli* cells. The fragments were appropriately inserted into the vector pEx31 (kindly given to us by H. Schaller, Heidelberg); this allowed the production of a fusion polypeptide. These proteins have been purified from bacterial cultures, and antisera against the fusions were raised in rabbits.

Initial Western blotting experiments with total and nuclear extracts from endosperm or root tissue show several bands, of which only an approximately 90 - 100 kD band is consistently seen in extracts from *Ac*-containing plants; this band is absent in extracts from genetically Ac- strains. Another band corresponding to a protein of about 40 kD size lights up in all tissues. These bands are not detected with pre-immune serum.

Meanwhile RNA and cDNA data are available (see accomp. report by R. Kunze and P. Starlinger). The size of the large protein is in agreement with the open reading frame of the *Ac* cDNA (2421 bp = 807 aa). Whether the protein detected by these tests is indeed an *Ac*-encoded protein remains to be established.

M. Müller-Neumann, H. Fusswinkel and P. Starlinger

The aberrant *Ds* element in the *Adh1-2F11::Ds2* allele

The *Ds* element in the *Adh1-2F11::Ds2* allele of *Zea mays* has been cloned and sequenced. The element, designated *Ds2*, belongs to a family of 10-12 similar or identical elements in the maize genome. It is identical to the *Ds* insertion in the *bz2-m* allele (see report by K. Theres and P. Starlinger, this volume). *Ds2* contains two segments at its termini that are nearly identical to *Ac*. These are approximately 550 and 300 bp, respectively. Adjacent to the left homologous part, there is a segment formed by duplications and triplications of *Ac* sequence. It is followed by a segment of approximately 200 bp length that is unrelated to *Ac*. Another sequence segment found both in *Ac* and *Ds2* seems to be derived from a common progenitor sequence. The first step in the evolution of this progenitor sequence was the duplication of 43 bp. Each of these sequences was further altered by the addition of small sequence duplications, which could be transposon footprints, and a few other insertions and deletions. By comparing the duplicated sequences both in *Ac* and in *Ds2*, it is seen that the original sequence is altered to a larger extent in *Ac* than in *Ds2*. These complicated events show that both *Ac* and its derivative *Ds* elements are in a process of rapid evolution.

Cleaving genomic DNA with restriction enzymes that cut inside the termini of *Ds2*, most of the *Ds2*-related sequences are found in one band of the size expected from cleaving cloned *Ds2*. Slightly deviating bands are found, however, indicating that *Ds2* itself is still evolving. The copy number of *Ds2*-related sequences varies between 6 and 12 copies in different maize lines investigated.

Armin Merckelbach and Peter Starlinger

Molecular cloning of *bz2-m*

The *bz2-m* allele carries a *Ds*-induced mutation in the *Bz2* gene (M. G. Nuffer, MNL 29:59). We have used the *Ds* element as a tag for the molecular cloning of a DNA fragment from this locus.

Genomic DNA preparations from *bz2-m*, *Bz2* and a stable recessive derivative of *bz2-m* were compared in Southern experiments, using probes derived from regions of *Ac* that are represented in many *Ds* elements. As these probes detect many bands, it was necessary to identify a band segregating with the *bz2-m* allele. Such a band was found in a BglII-digest, and the corresponding 4.3 kb fragment was cloned into the lambda vector NM 1151 (B. Klein and K. Murray, J. Mol. Biol. 133:289, 1979).

A proof that the cloned DNA fragment indeed came from the *bz2-m* locus was obtained using a DNA fragment flanking *Ds* as a probe in Southern experiments. DNA derived from the wildtype or from revertants of *bz2-m* to *Bz2* showed a band 1.3 kb smaller than the band detected in DNA from the mutant. A band of wildtype size was also seen in two stable recessive derivatives of *bz2-m* (which may have arisen by imprecise excision of the *Ds* element). In DNA from the deletion strain *an-bz2-6923*, no band hybridizing to the probe was detected.

The size difference between the wildtype and the mutant fragment indicated a length of 1.3 kb for the *Ds* element. A *Ds* element of this size is the *Ds2* insert in the *Adh1-2F11* allele. Restriction mapping and sequence analysis confirmed that the *Ds* insert in *bz2-m* is identical in sequence to the *Ds* insert in *Adh1-2F11*.

Klaus Theres and Peter Starlinger

Cloning of DNA from the *P* locus

A wildtype of the *P* gene, *P-rr*, is necessary for the pigmentation of the pericarp and the cob. In some alleles, either pigmentation of the pericarp or of the cob is lost independently (*P-wr* and *P-rw*). In the *P-vv* allele, however, the insertion of *Mp* (equal to transposable element *Ac*), leads to the loss of both pericarp and cob pigmentation. Excision of *Mp* from the locus restores pigmentation of both tissues simultaneously. Thus, in this allele *P* behaves as a single gene (Brink and coworkers). As a prerequisite for studies of this interesting gene, we have cloned DNA of this locus from both *P-vv* and *P-rr*.

For the cloning of *P-vv*, we used a central segment of *Ac* as a probe. We identified an 8 kb SalI fragment absent from *P-ww*. This fragment was cloned and shown to contain a 4.5 kb insertion identical to *Ac* by restriction analysis and partial DNA sequence.

A 2.6 kb BglII-SalI fragment flanking the *Ac* element was used as a probe for the identification of *P* sequences in the *P-rr* allele. *P-rr* contains a 3.5 kb SalI band absent from *P-vv*. This band is also absent from the null mutation *P-ww*, which may thus be a deletion. The 3.5 kb band is cleaved by BglII yielding a 2.6 kb fragment. This is expected from the DNA prior to the insertion of *Ac*. In addition to the 3.5 kb band, several other bands also hybridize to the flanking probe. The relation of these to the *P* gene is not known at present.

Partial sequencing of the termini of *Ac* inserted in the *P* sequences shows two interesting features:
1. The outermost nucleotides of *Ac* are not complementary to each other and resemble in this respect the two cloned copies of *Ac* from *wx-m9* (R. Pohlman et al., Cell 37:635, 1984) and from *wx-m7* (M. Müller-Neumann et al., MGG 198:19, 1984). As the *P-vv* allele was isolated by Emerson as early as 1917, while the two *Ac* elements in the waxy gene derive from an *Ac* activated from a silent state by McClintock, this similarity either shows that this difference of *Ac* to most *Ds* elements has a functional meaning or else that all the *Ac* elements cloned so far are evolutionarily closely related.
2. The absence of an 8 bp flanking duplication that is found accompanying all other *Ac* or *Ds* insertions possibly indicates the formation of an adjacent deletion after the insertion. The reversion of *P-vv* to *P-rr* must then tolerate this deletion event, possibly because the insertion has occurred in an intron. Peterson and Schwartz describe a clone of *P-vv* DNA identical to our clone (see report in this issue).

From *P-rr* DNA, lambda clones hybridizing to the above mentioned flanking probes have been isolated and are presently under investigation. The cloning of the *P* locus has also been performed by J. Chen and S. Dellaporta, Cold Spring Harbor (personal communication).

Christa Lechelt, Alan Laird and Peter Starlinger

The promoter of the shrunken gene promotes transient expression in a *Triticum monococcum* cell line

We have constructed a chimeric gene, consisting of a 2 kb DNA fragment from the shrunken promoter, the bacterial neomycin phosphotransferase II gene from Tn5 and the polyadenylation site of the octopin synthase gene derived from the t-DNA of *Agrobacterium tumefaciens*. The promoter fragment from *Zea mays* contains approximately 2 kb upstream sequences, the transcription start and 40 bp of exon

1 (W. Werr et al., EMBO J. 4:1373, 1985). This exon contains only untranslated sequences. The first ATG in the chimeric transcript is provided by the bacterial sequences and starts the open reading frame encoding the neomycin phosphotransferase II enzyme.

We have used this construction to transform *Triticum monococcum* protoplasts by polyethylene glycol treatment (H. Lörz et al., MGG 199:178, 1985). We found expression of this chimeric gene, that means NPTII activity, over a time period of at least 10 days. The maximum enzyme activity was determined 4 days after transformation. The enzyme activity was dependent on the presence of the maize promoter fragment within the construction. This result suggests that the maize promoter acts in the *Triticum monococcum* cells (W. Werr and H. Lörz, MGG, 1986, in press). For a detailed analysis of this maize promoter we have constructed a series of deletion mutants within the promoter fragment of the chimeric gene which will be tested for activity within this transient expression system.

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In vitro plant regeneration and somaclonal variation in the inbred A188

Immature embryos of the inbred line A188 were excised from milk stage kernels and cultured on 2,4-D (2 mg/l) containing medium for the induction of embryogenic callus. Approximately 3 months after culture initiation embryogenic callus parts were subsequently transferred to media with lower 2,4-D concentrations (1 mg/l, 0.5 mg/l) and without 2,4-D for plant regeneration. Our main interest with regards to the direct regenerants (R_1-generation) and the following generations is to determine whether any form of variation is transmitted to the selfed progeny. The discovery of variants amongst the offspring is being combined with molecular analyses of these altered plants in order to examine whether changes at the molecular level can be correlated to the occurrence of somaclonal variants.

Among 117 plants regenerated from embryogenic callus cultures and grown to maturity in the glasshouse (R_1-generation), the following phenotypic variations were observed:

all plants	:	reduced height
1 plant	:	longitudinal yellow leaf stripes that persisted to maturity
3 plants	:	no ear development
9 plants	:	no tassel development
6 plants	:	feminization of the male flower

Of the 117 regenerated plants, 99 could be self-pollinated; 91 set seed. The number of seeds per cob ranged from 2 to over 200.

To test the R_2-seeds, samples of 20 seeds from, up to now, 40 R_2-lines were potted and their development was followed. With different frequencies the following alterations were observed:

- lack of germination
- 'early death' of seedlings on the coleoptile stage or on a later seedling stage
- 'late death'; 5-6 week old plants suddenly turned brown and died
- albinos
- leaf stripes
- curling of leaves
- dwarfs
- sterile plants, either lacking tassels or ears or both

Phenotypically 'normal' looking plants were self-pollinated for the production of the R_3-generation. In a few lines this R_3-generation has already been tested as well, and similar distortions as described above were found. Further characterization of the R_2- and R_3-generations is in progress.

To find possible molecular origins of the described variation it is being determined whether gene methylation is altered as a consequence of the tissue culture pathway and whether somaclonal variation can be correlated with any changes in the methylation state. Total genomic DNA from over 100 R_2-plants has been extracted and digested with the isoschizomer restriction endonucleases MspI and HpaII. These digestions showed that the majority of the DNA's (85%) restricted with MspI, and the remainder showed restriction differences indicative of gross changes in the degree of DNA methylation. Further experiments include the attempt to determine whether particular gene sequences are also changed in their methylation state by probing MspI digested DNA's from phenotypically normal and disturbed plants with ^{32}P-labelled known gene sequences. Work is also being undertaken to map the region of known gene sequences for different somaclones by the process of restriction fragment length polymorphism. In conjunction with the methylation studies, this will allow us to determine whether tissue culture induced variation is a result of methylation changes alone, or occurs as a consequence of mutations in the genome.

Elke Göbel, Peter T.H. Brown, Horst Lörz

Molecular cloning of the *A2* locus

The *A2* locus is involved in anthocyanin synthesis and harbors a number of mutable alleles (Peterson, P. A., in Maize Breeding and Genetics, Wiley Interscience, Walden, D.B., ed., 1975) controlled by the *En* (*Spm*) system of transposable elements.

To enable study of molecular events at the *A2* locus, a transposon tagging experiment was undertaken using *En*-specific probes to screen a genomic library constructed with a line containing the *a2-m1(1511)* allele, in which an autonomous *En* element is present at the *A2* locus (Peterson, P. A., Genetics 59:391-398, 1968). One clone was isolated, lm305, which contained a complete 8.4 kb *En* element (Pereira, A., et al., EMBO J. 4:17-23, 1985) as shown by restriction mapping and heteroduplex analysis. Since it has been shown that the autonomous *En* elements from the *A1* (O'Reilly, C. et al., EMBO J. 4:877-882, 1985), *C2* (Wienand, U., et al., this volume), and *C* (Paz-Ares, J., et al., this volume) loci of maize are identical in structure by these criteria, the data suggested that sequences flanking the element in lm305 represent at least a portion of the *A2* locus. An lm305-derived fragment was used to probe Southern blots of genomic DNA from lines carrying various alleles of the *A2* gene. The results obtained are in good agreement with those expected for an *A2*-specific probe.

In order to directly analyze *En*-mediated gene suppression without interference from excision events, efforts are currently underway to isolate genomic clones for the class II state of *a2-m1* (McClintock, B., Carnegie Inst. Wash. Yearbook 57:415-429, 1958), the structure and properties of which will hopefully further develop concepts concerning the mechanism (Schwarz-Sommer, Zs., et al., EMBO J. 4:2439-2443, 1985) of *En* (*Spm*) action in the maize genome.

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Molecular cloning of the *c* locus

The *c* locus is involved in the regulation of anthocyanin biosynthesis and has been cloned using transposable elements as gene tags. The *En1* element (Pereira, A., et al., EMBO J. 4:17-23, 1985) as well as the *Ac* element (Fedoroff, N., et al., Cell 35:235-242, 1983; Müller-Neumann, M., et al., Mol. Gen. Genet. 198:19-24, 1984) were used as probes to screen genomic libraries made from the *En* induced mutable *c* alleles *c-m668655* and *c-m668613* (Reddy, A., and Peterson, P.A., Mol. Gen. Genet. 192:21-31, 1983) and the *Ds*-induced mutant *c-m2* (McClintock, B., Carnegie Inst. Yearbook 47:155-169). Three homologous clones could be isolated from these libraries. From the *c-m668655* and *c-m668613* libraries, one clone each was isolated which contained a complete 8.4kb *En1* element. The screening of the *c-m2* library revealed one clone containing a 2kb *Ds* element. The elements are integrated into a fragment about 1kb in size.

From a wild type maize line (LC) a homologous clone could be isolated lacking transposable element specific sequences. A 1kb EcoRI fragment of this wild type clone has been used to probe poly A+ RNA from developing kernels (30 days after pollination) and lights up 3 transcripts of different sizes (1.6kb, 1.4kb, 0.3kb). A clone representing the *C-I* allele was also isolated. The analysis of this clone revealed long regions of DNA rearrangement (>11kb) compared to the wild type clone.

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Molecular analysis of the *c2* locus

The *c2* locus is one of the structural genes involved in anthocyanin biosynthesis. This locus was cloned from the *Spm* (*En*) induced mutants *c2-m1* (unstable) and *c2-m2* (stable; McClintock, B., Carnegie Inst. Yearbook 65:568-578) as well as from the wild type allele (*C2*). The *En1* element (Pereira, A., et al., EMBO J. 4:17-23, 1985) was originally used to probe a genomic library constructed from the mutable *c2-m1* allele (this mutant contains the autonomous *Spm* at *c2*). A clone containing a major part of an *En1* element was isolated. Sequences of this clone flanking the element were shown to be *c2*-specific by using them as probes for cloning of the *c2-m2* and *C2* allele. From the *c2-m2* allele a clone could be isolated containing an *Spm* deletion derivative (*Spm-I2*), 3kb in size. The clone isolated from *C2* did not contain *Spm*-element-specific sequences. Northern experiments using part of this clone as probe light up a mRNA of about 1.5kb. A cDNA clone homologous to this mRNA was isolated and partially sequenced. On the amino acid level this sequence showed strong homology to the amino acid sequence of parsley chalcone synthase. These data positively confirm those of Hugo Dooner (Mol. Gen. Genet. 189:136-141, 1983), who suggested on the basis of enzyme activity measurements that *C2* encodes chalcone synthase.

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Transposition of maize controlling element Activator in a heterologous plant

The activity of a maize autonomous controlling element, Activator (*Ac*), and a non-autonomous derivative, Dissociation (*Ds*) were investigated in tobacco cells transformed with Ti plasmid vectors that contained *Ac9* or *Ds9* flanked by short maize *wx* gene sequences within their T-DNA (Fedoroff, N., Wessler, S., and Shure, M., Cell 35:235-242, 1983; Schell, J., Kaulen, H., Kreuzaler, F., Eckes, P., Rosahl, S., Willmitzer, L., Spena, A., Baker, B. and Fedoroff, N. in Molecular Biology of Development, C. S. H.

Symp. Quant. Biol., 50, 1985 in press). The structures of the elements and surrounding *wx* and T-DNA sequences were determined in nine *Ac* and five *Ds* tobacco transformants by digestion with restriction enzymes, Southern blotting and hybridization using specific probes. The results of our analysis indicated that in four out of nine *Ac*-transformed lines *Ac* had excised from its original position within the T-DNA and was inserted at several new sites within the tobacco genome. *Ds* did not excise from its original T-DNA position in any of the transformants examined. Two newly located *Ac* fragments along with cellular flanking sequences were cloned from a line of tobacco in which *Ac* had transposed. Fragments comprised of sequences flanking the newly integrated *Ac* elements were used as hybridization probes to normal tobacco DNA and to the tobacco DNA from which they were isolated. The two newly located *Ac* copies were integrated into repetitive tobacco DNA sequences. Two tobacco *Wx* revertant fragments situated within different T-DNA copies from which *Ac* had excised were cloned and sequenced. Excision of *Ac* from the one T-DNA examined was accompanied by loss of two base pairs of the original 8 bp repeated *Wx* target sequence. Our results indicate that the maize controlling element, *Ac*, is capable of transposition in a heterologous plant cell.

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COLUMBIA, MISSOURI
University of Missouri

Lethal ovule *lo2* is distal to TB-9Sb

When plants carrying *c lo2* and TB-9Sb were pollinated by a normal stock, most of the offspring carried, as expected, the B-A translocation, since *lo2* ovules abort. A confirmed tertiary trisomic plant (9 9 B-9Sb) derived from this material shed normal pollen but gave less than 50% seed set when self-pollinated. Except for occasional recombinants, all kernels were colored.

Since TB-9Sb and *lo2* are 0.7 and 6.1 map units, respectively, distal to *wx* (D. S. Robertson, MNL 52:134, 1978; O. Nelson, MNL 46:203-205, 1972), the breakpoint of TB-9Sb must be close to *lo2* and likely between *wx* and *lo2*. Since colored kernels and normal ovules were transmitted together to the progeny of the self-pollinated tertiary trisomic plant, and *c* is already known to be beyond the breakpoint, both *C* and *Lo2* must be on the B-9S chromosome. The trisomic plant was therefore of the constitution (*c lo2*)/(*c lo2*)/B-9S(*C Lo2*). Although the B-9Sb chromosome is transmitted through the pollen 7.8% of the time (D. S. Robertson, Egypt. Genet. Cytol. 4:189-200, 1975), the fact that the tertiary trisomic plant gave less than 50% seed set may indicate that the B-9Sb element is lost during megasporogenesis more than 7.8% of the time.

The tertiary trisomic stock can be maintained by self-pollination. By selecting semisterile plants that continue to segregate occasional colorless kernels, it should seldom be necessary to confirm the trisomic condition by root tip chromosome counts. Pollen from this stock should give progenies consisting almost entirely of *+/lo2* plants, because crossing over between the breakpoint and *lo2* should be rare. However, some of the plants will also carry the B-9Sb chromosome, which sometimes may be advantageous.

J. B. Beckett and Bryan Kindiger

Use of B-A translocations to locate duplicate genes

In general, the placement of duplicate genes to chromosomes has not been accomplished. This note outlines a procedure for locating such genes to chromosome arms by using the B-A translocation series. For example, the irregular kernel arrangement in Country Gentleman sweet corn results from the development of both the upper and lower florets of most pistillate spikelets (W. A. Huelsen and M. C. Gillis, Ill. Agric. Exp. Stn. Bull., Vol. 320, 1929 [ed. note: authorship attributed to W. A. Hudson or W. A. Huelson in past publications is inaccurate]). The symbols given to these duplicate genes are *pi1* and *pi2*. The initial step in the procedure involves pollinating Country Gentleman by the basic series of B-A translocations. During the next season, hypoploids for each arm are selected and pollinated by or crossed onto Country Gentleman. In the third season, seed of each backcross progeny is planted out and open pollinated ears are harvested at maturity. Only in those families where the appropriate B-A translocation has uncovered one of the two genes will there be a 1:1 segregation of normal vs. irregular row arrangement. Families involving translocations that fail to uncover either of the two genes will segregate the trait in the normal 3:1 ratio.

Some alterations in this technique may be required for other duplicate gene systems, such as those controlling lethal traits. Results from the Country Gentleman crosses by the B-A translocation series will be forthcoming.

Bryan Kindiger and J. B. Beckett

Homozygous B-A translocation stocks

Over the last few years, we have generated 23 B-A translocation stocks in the homozygous condition. These stocks, many of which are in undefined backgrounds, are listed below:

TB-1Sb	TB-4Sa	TB-5Lb	TB-8La	TB-10L9
TB-1Lc	TB-4Lc	TB-6Sa	TB-8Lc	TB-10L19
TB-3Sb	TB-4Ld	TB-6Lb	TB-9Sb	TB-10L20
TB-3La	TB-4Le	TB-6Lc	TB-9Sd	
TB-3Ld	TB-4Lf	TB-7Lb	TB-10Sc	

We expect to have sufficient materials for transmittal to the Stock Center by the fall of 1986. Other B-A translocation stocks that should soon be homozygous are TB-5Sc, -7Sc, -9Lc (*Wc*), and several more -3L's, -4L's and -10L's.

Bryan Kindiger and J. B. Beckett

A TB-9Lc stock with *Wc* on the B-A chromosome

A TB-9Lc stock that carries *Wc* on the B-A chromosome has been identified. Along with a seed size effect associated with the translocation, the placement of *Wc* on the B-A chromosome greatly simplifies identification of hyperploid and hypoploid kernels on a testcross ear. Limited quantities of seed of this translocation are available. Efforts to make the stock homozygous for the translocation should be completed by next summer.

Bryan Kindiger and J. B. Beckett

Large satellite on the short arm of chromosome 6

In the course of testing the Bolivian variety Pororo (my Acc 1698) for crossability with *Tripsacum dactyloides*, observations of root tip metaphase chromosomes showed that some plants were segregating for a conspicuously large satellite on the short arm of chromosome 6. The large satellite has been backcrossed twice to L289 to provide suitable material for pachytene studies. Small quantities of seed are available upon request.

The Pororo stock (Goodman 71-72-668) was obtained, via Robert McK. Bird, from M. M. Goodman. 71-72-668 was apparently an increase of BOV806 (WLB E-18). The large satellite reported here may be the same as that described by B. McClintock (personal communication, R. L. Phillips).

J. B. Beckett

Analysis of small plants from nitrosoguanidine treated pollen

N-methyl-N'-nitro-N-nitrosoquanidine (MNG) is one of the most powerful mutagens known for higher plants. Unlike ethylmethanesulfonate, MNG treatment of mature pollen grains does not produce a uniform, vigorous M1 generation. Throughout the M1 population are small plants, and this small plant effect does not segregate in Mendelian ratios in later generations. These small plants fall into four separate phenotypic categories. Type 1: This plant is half the normal height and normal in color. The leaves are one-half to two-thirds the normal width and fewer in number than normal plants. The nodes are not compressed. Type 2: This plant is also one-half the normal height and normal in color. The leaves are one-half to normal in width. The differences from Type 1 plants are that the number of leaves is normal and the nodes are compressed. Type 3: These plants are under 20 inches in height. They are pale green and the leaves are grainy and erect. The leaves are also less than a quarter the width of normal leaves and the tips are necrotic. Most plants have only four leaves and are female sterile. Type 4: The plants are less than one-half normal height and grow non-uniformly. The leaves are creased with fine white striping and corrugations, and are thinner than normal. Data analyzed from selfed small plants show the effect is not due to a normally segregating single dominant or recessive mutation. Differential transmission of gametes is not responsible for the aberrant ratios found in data from selfed small plants. The germination of pollen from normal and small plants was the same. The length of pollen tubes was graphed against the number of pollen tubes, and the graphs showed only one type of pollen was present. Data from outcrosses show the effect is not attributable to multiple dominant or recessive mutations. If the small plant effect was due to multiple mutations, outcrossing to inbreds would show plants of intermediate height. Several generations of outcrossing show no plants of intermediate height. Cytogenetic data prove that the small plant effect is not a result of aneuploidy. Restriction analysis of small plant, mitochondrial DNA shows no band differences from normal inbreds.

Richard Vierling

The *v*-576* mutation probably unmasks a developmental timing locus, *Mof**

The nuclear mutation *v*-576* was found by M.G. Neuffer in a M2 progeny derived from pollination with ethylmethylsulfonate-treated pollen. When crosses were made between inbred Mo17 and M2 material segregating *v*-576*, I found that most F2 progenies segregated two virescent phenotypes. A few F2 progenies segregated only one *v*-trait. I could distinguish the two *v*-phenotypes by noninvasive measurement of leaf chlorophyll fluorescence induction kinetics: one exhibited a high initial yield of fluorescence (hcf), the second, a lower than normal yield (lcf). Additionally, the ratio of the variable yield fluorescence component (f_v) to the initial yield (f_o) is greatly reduced for hcf-v but not lcf-v (see Figure 1). In both cases, abnormal fluorescence was developmentally conditional and observed only in partially, as opposed to completely, greened leaves. We have shown that hcf *v*-576* is associated with early assembly of the major chlorophyll light harvesting complex (LHCII) and lcf *v*-576* with late assembly of LHCII (Polacco et al., Plant Physiol. 77:795-800,1985). Normally, LHCII-assembly is a late event of thylakoid differentiation. Both *v*-576* traits are allelic to *v*-424* (MGNL 1985), a mutation similarly derived by M.G. Neuffer, which has continued to segregate only the hcf-v phenotype after four backcrosses to Mo17.

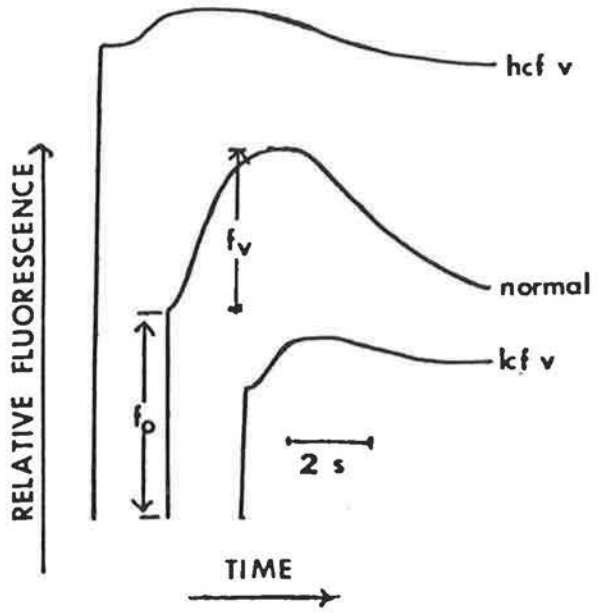


Figure 1. Leaf chlorophyll fluorescence kinetics of two v*-576 phenotypes and a normal sibling. The variable component, f_v, and initial yield, f_o, are indicated for the normal case.

Either modifying loci or mutability at *v*-576* are possible explanations for the two *v*-576* phenotypes. My recent data indicate a modifying locus, *Mof** (modifier of fluorescence), has been unmasked by *v*-576*. I have crossed Mo17 with individuals of progenies segregating only one *v*-576* phenotype where at least eight, self-fertilized siblings, heterozygous for *v*-576*, were found to segregate only the same *v*-trait. I have only analyzed crosses involving the lcf-v trait. Both lcf-v and hcf-v individuals appear in F2 progenies derived from two F1 generations. A large class of *v*-individuals with an intermediate fluorescence pattern predominates. This class has normal or somewhat elevated initial fluorescence yield and a significant f_v component. A histogram of the initial fluorescence yield data for pale-green *v*-leaf tissue shows a continuum of values that fit three superimposed, bell-shaped curves. The areas bounded by the curves approximate a 1:2:1 ratio for lcf:intermediate:hcf fluorescence yields (see Figure 2). Genetic analysis of the intermediate fluorescence yield class is required to confirm the 1:2:1 segregation. Typically, there is some overlap of analogous fluorescence distribution curves for normal and *v* siblings taken from progenies segregating only lcf *v*-576* or hcf *v*-576*. The overlap probably reflects the conditional character of the phenotypes. I tentatively conclude that two variant, codominant alleles at an unlinked locus (*Mof**) are involved, that Mo17 carries the allele responsible for the hcf-v trait (*Mof*-1*) and that the M2 stock carried both alleles (*Mof*-1, Mof*-2*). I am currently

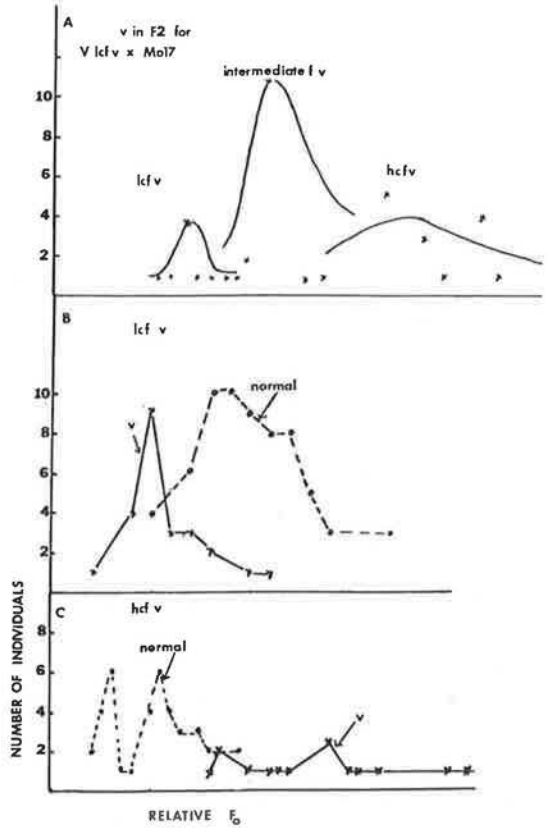


Figure 2. Distribution of initial fluorescence yield. Panel A depicts only v individuals segregating in the F2 progeny for the backcross of true-breeding lcfv*-576 with Mo17. Panel B depicts normal and v siblings of a progeny segregating only lcfv*-576. Panel C depicts normal and v siblings where only hcfv*-576 segregated.

testing whether the putative *Mof*-2* allele responsible for lcf-v can modify the hcf-v trait of other mutations allelic to *v*-576*

Mary Polacco

v-424:* Allelism tests with *v4*, *w3*; linkage to *w3*

All three loci map to the long arm of chromosome 2 (for *v*-424*, see Chang, MGNL 56:44, 1982). Allelism tests between *v*-424* and *w3* or *v4* were negative. I employed growth conditions which permitted expression of all three phenotypes and confirmed the parental genotypes for each pair tested. An F2 progeny of the cross: *V*-424 v*-424 W3 W3* x *V*-424 V*-424 W3 w3* segregated green, albino (*w*) and virescent (*v*) seedlings:

	Total	Green	*v*	*w*
Number of seedlings	91	39	38	4

Germination was 100 percent. I did not observe any vivipary as is sometimes found for *w3*. The combined number of *v* and *w* individuals is somewhat high, but consistent with the segregation of two independent, recessive loci, if the *v*-class includes *v*-424 v*-424 w3 w3*. I will be testing this possibility by backcrosses to material containing only *v*-424* or *w3*. Similar analysis of linkage between *v*-424* and

v4 is in progress. These two virescent loci can be distinguished by phenotype: *v4* requires cold temperatures for expression while *v*-424* is expressed at all temperatures tested (up to 95 F).

Mary Polacco

Endosperm protein analysis of thirty-five selected defective kernel mutants

Thirty-five EMS (ethyl methane sulfonate) induced defective kernel mutants were selected for study of their endosperm protein differences. These mutants were crossed by a large embryo stock Aho (Alexander high oil) and backcrossed for several generations. Progenies had fairly uniform phenotypic expression. Fifteen normal kernels from a good F_2 segregating ear were planted. Among these kernels ⅔ are heterozygous (+/*dek*) and ⅓ are homozygous (+/+) for the mutant gene. Plants were selfed or double pollinated by selfing half the silks and outcrossing the rest by the arm-locating B-A translocation. Phenotypic difference between normal and mutant kernels can be identified 16 to 20 days after pollination. Good segregating ears were harvested 18 days post-pollination and were stored at -20 C. From each segregating ear, 10 normal and 10 mutant kernels were selected, and the pericarps and embryos were removed. The weight of 10 normal or 10 mutant endosperms was measured, 5 ml sample buffer was added and tissues were ground. Extractions were made by centrifugation and soluble proteins in the supernatant were denatured 5 minutes at 80 C and stored in a freezer. Samples were loaded in equal amount and proteins were separated by SDS-PAGE method. The results are: three mutants did not have protein differences, five mutants showed single or few protein differences and the rest (27 mutants) had many differences with low storage proteins (zeins) as listed in Table 1.

Table 1: SDS-PAGE study of mutant endosperm proteins of thirty-four defective kernel mutants.

Mutant	Location	Lethality	Seedling Phenotype
A. No Protein Difference:			
orp-1186	?	viable	seedling lethal
o-1241	7L	vialbe	luteus
de-1296A	6L	lethal	-
msc-1330	10L	viable	white
smk-1373A	?	lethal	-
B. Single or Few Proteins Missing:			
clf-792	1S	lethal	-
sh-912	4S	viable	green stripe
sh-1053B	?	viable	streaky
C. Many Differences with Low Storage Proteins:			
sh-627D	3L	lethal	-
o-744	9L	lethal	-
cp-863A	5L	viable	normal
crp-888A	?	lethal	-
cp-931A	5S	lethal	-
o-945A	?	lethal	-
cp-991	1S	lethal	-
cp-1054	9S	viable	normal
cp-1078B	1S	viable	normal
crp-1121(prol)	8L	viable	green stripe
ptd-1130	4L	lethal	-
de-1142	1S	lethal	-
fl-1145A	?	lethal	-
fl-1163	?	lethal	-
de-1177A	?	lethal	-
de-1196	5L	lethal	-
cp-1275A	5L	lethal	-
cp-1294	?	lethal	-
o-1298	?	lethal	-
fl-1299	5L	lethal	-
cp-1308A	1S	lethal	-
dsc-1315A	1L	lethal	-
sh-1322A	?	lethal	-
sh-1324A	?	viable	pale pale green
sh-1339A	3S	lethal	-
smk-1437	?	lethal	-
crp-1528	8L	lethal	-

Ming T. Chang and M. Gerald Neuffer

Hypoploid test to locate defective kernel mutants on 8S or 8L proximal to TB-8Lc breakpoint

The defective kernel mutant collection has been tested for arm location by a set of B-A translocations which cover nineteen chromosome arms (all except the short arm of chromosome eight). Those receiving a full test but still not located were thought to be located either proximal to the breakpoint of one of the 19 arms or on the short arm of chromosome eight. Mutants that are actually located on the short arm of chromosome eight or on the long arm proximal to the centromere can be identified by selfing the hypoploids grown from the TB-8Lc translocation cross. Since the deficient A^B chromosome will not transmit, only the normal chromosome eight, carrying the mutant allele, forms viable gametes. Among 35 defective kernel mutants studied in this manner, six showed a higher mutant proportion than the 25% expected from a normal self. The transmission data in Table 1 showed that the mutant frequencies ranged from 62% to 100%.

Table 1: TB-8Lc hypoploid test. q = proportion of mutant gametes. ** = significant at 99% confidence level.

mutant	Normal Seed	Mutant Seed	Total	$\chi^2_{(3:1)}$	q
rgh*-974A	0	408	408	1220.00**	1.00
o*-1096A	35	218	253	501.57**	0.93
fl*-1145A	563	557	1120	364.06**	0.71
cp*-1255B	116	74	190	18.98**	0.62
gm*-1319C	89	819	908	2055.05**	0.95
cp*-1406	104	71	175	21.81**	0.64

Ming T. Chang and M. Gerald Neuffer

Breakpoint position of TB-8Lc

The breakpoint of TB-8Lc was determined by examining the pachytene stage of an L289 x TB-8Lc hybrid. Materials were kindly supplied by B.-Y. Lin. A modification of the smear technique (C. R. Burnham, Maize for Biol. Res., p.107, 1982) was used. Good pachytene figures were photographed and enlarged. The length of the short arm and the length from centromere to the breakpoint of the long arm were traced. The unpaired long arm region was not measurable, so the physical length of the long arm could not be directly determined. Since the arm ratio is 1 to 3.2 for the short and long arms of

chromosome eight, respectively (M. M. Rhoades, J. Hered. 41:58-67, 1950), the physical length of the long arm can be calculated by measuring the short arm. Distance between the centromere and the breakpoint, taken from ten measurable figures, was divided by the calculated length of the long arm, giving a breakpoint position on the long arm at 0.24 ± 0.05.

Ming T. Chang and J. B. Beckett

A Mutator-induced PSII photosynthesis mutant

We have produced several photosynthesis mutants by crossing stock bearing Robertson's Mutator to standard lines W23 and Mo17. Families with lesions in the cytochrome b6/f complex of the chloroplast thylakoid membrane (Taylor, 1986, in press), PSI (Hunt & Miles, MNL 58:68, 1984), and coupling factor (Hunt, personal communication) have been identified previously. This communication is to report the characterization of a Mutator-induced PSII mutant.

The mutant was identified as segregating for a marked increase in chlorophyll fluorescence compared with wild type plants of the same family. Figure 1 shows a typical fluorescence induction trace of an excised leaf from a mutant plant. The fluorescence yield among individual mutant plants ranges from 2.8-5.5 times the wild type. In Figure 1 the fluorescence trace is not square as would be expected from a complete block of electron transport, but retains about 10% variable fluorescence. Traces from other mutant individuals ranged from just over 0% to 22% variable fluorescence.

LDS polyacrylamide gel electrophoresis of thylakoid polypeptides from the mutant shows that all of the major polypeptides associated with PSII (49, 45, 32, 23, 16 & 9 kDa) are decreased or missing. An unidentified band with an apparent molecular mass of 20.1 kDa is present among the polypeptides from the mutants but not among those from normal plants (see Figure 2). Of the polypeptides associated

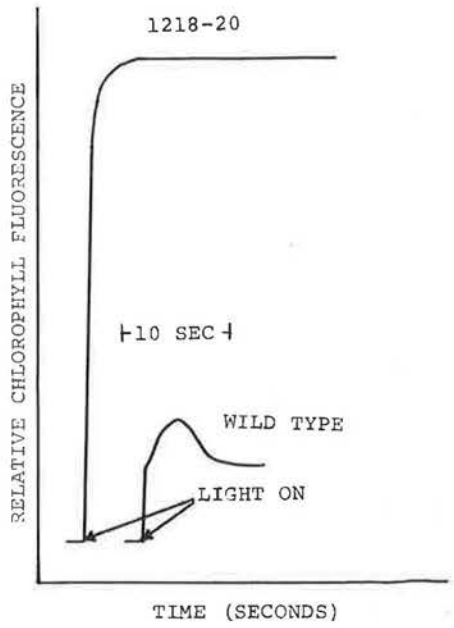


Figure 1.

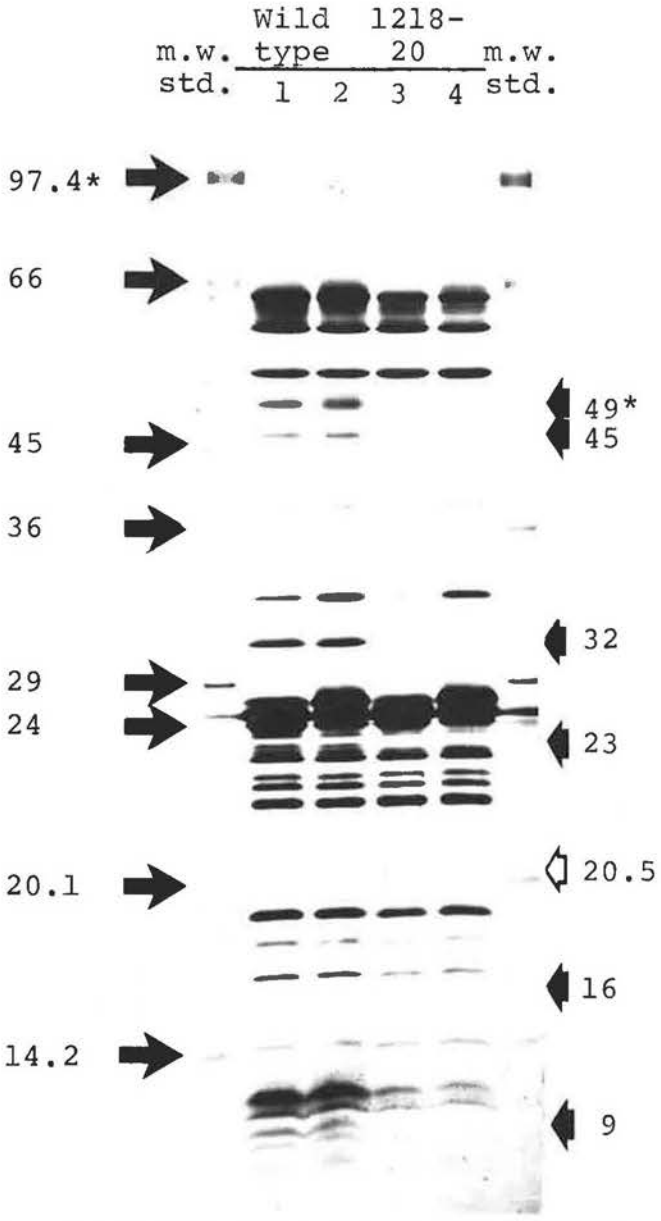


Figure 2. LDS-PAGE of thylakoid membrane polypeptides from mutant 1218-20 and wild type plants from the same family. Samples in lanes 1 and 3 were heated before being loaded onto the gel. ◖ PSII-associated polypeptides missing from 1218-20. ◊ Unidentified band not present in wild type.

with PSII only the peripheral water-splitting components are encoded by the nuclear genome. Nevertheless, all of the chloroplast encoded core proteins of PSII are missing as well. At this point we have no evidence to suggest whether the gene which is disrupted by the Mutator element codes for a regulatory, structural or enzymatic polypeptide.

Chloroplasts isolated from the mutant showed greater oxygen consumption than wild type when incubated in the light with methyl viologen, ascorbate, DCMU (diuron) and diaminodurene (DAD). This requires normal electron flow through PSI. When incubated with p-phenylenediamine and ferricyanide the mutant chloroplasts produced only 10% as much oxygen as those from normal siblings. This is consistent with a block of electron transport through PSII (Table 1).

Table 1. Oxygen consumption and evolution by chloroplasts isolated from 1218-20 hcf and normal plants of the same family.

	wild type	1218-20 hcf	%wt
	----------umol/hr/mg chlorophyll----------		
PSI oxygen consumed*	899	1223	136
PSII oxygen evolved	216	25	11.8

*Oxygen consumption and evolution were measured with an oxygen electrode. Isolated chloroplasts were incubated in the light in the presence of appropriate electron donors and acceptors. Electron flow through PSI was determined by measuring oxygen consumption by chloroplasts in the presence of 0.1 mM methyl viologen, 2.0 mM ascorbate, 8.0 um DCMU (diuron) and 0.5 mM diaminodurene (DAD). Electron flow through PSII was determined by measuring oxygen evolution in the presence of 0.4 mM p-phenylene diamine (PD) and 2.0 mM ferricyanide.

Leaves from the mutant plants were excised above the sheath at the three-leaf stage for the preceding tests. Following excision the plants generally died without producing any further growth. One individual plant continued to grow following the second defoliation, producing several leaves. Subsequent checks of the fluorescence induction kinetics and polypeptide composition of the plant showed that the characteristics described above did not change over time. The plant continued to grow for 90 days before it died. Among the fluorescence induction traces of the mutant individuals the surviving plant had the highest percentage of variable fluorescence. It appears that in this plant electron transport through PSII, though impaired, was sufficient to support growth for several weeks.

The oxygen evolution experiment was carried out using a sample of chloroplasts isolated from several plants. While the sample evolved only 11.8% as much oxygen as the wild type sample this value represents an average. The plant that survived must have had a rate of oxygen evolution (and so electron transport) significantly higher than the average.

Other *hcf* mutants identified by this lab are able to grow to as much as 25 cm before they die, in spite of a partial block in photosynthetic electron transport. Apparently, the surviving mutant was able to carry out electron transport at a level adequate to support growth but was not able to mature fully.

We conclude from the variability that we have observed among these mutant individuals and from the extended growth of a single plant that the putative Mutator mutation is variable in its effect. It may be that other Mutator-induced effects cause the variability we see among the *hcf* mutants. Or it may be that the actual mutation which causes the *hcf* phenotype is variable in its effect.

The identification of a Mutator-induced PSII mutant adds to the accumulating evidence that Mutator (*Mu1*) causes all of the types of photosynthetic mutations previously induced by chemical mutagens.

Bill Cook and Don Miles

UFGT as a measure of *Tripsacum dactyloides* introgression into maize

Simple techniques for measuring and quantitating introgression offer an opportunity to survey large populations rapidly. Uridine-diphosphoglucose: flavonoid-3-0-glucosyltransferase (UFGT), involved in anthocyanin biosynthesis, encoded by the *Bz1* locus, fulfills this need. This soluble enzyme can be rapidly extracted and quantitated using HPLC for the analysis.

Seedling tissues extracted from maize, *Tripsacum* and their F1 hybrids were surveyed for UFGT activity. Quercetin was used as the flavonoid substrate and UDPG was the glucose donor. *Tripsacum* from several different locations was found to have the active enzyme. The F1 tissue activity appears to have an additive effect when compared to either pure maize or *Tripsacum*. The following table summarizes the data for maize, Huey, and the maize X Huey hybrid.

	Specific Activity*
Maize	
ACR-rBP1	42.6
W23	22.9
Tripsacum	
Huey	17.2
F1 Hybrid	
ACR-rBP1 X Huey	48.4
W23 X Huey	27.3

*μmoles product produced min^{-1} mg protein $^{-1}$

J. B. Bussard, B. Kindiger and R. Larson

Pr:flavonoid 3′-hydroxylase in maize seedlings, leaf sheaths and aleurone

A microsomal preparation has been obtained from seedlings and leaf sheaths that catalyzes the addition of a hydroxyl group at the 3′-position of flavonoid compounds. This activity has also been assayed in aleurone tissue that had been scraped from the seed and used as the source of the hydroxylase. Thus far it has not been possible to obtain the hydroxylating, microsomal particle free of the

aleurone tissue. Utilization of genetic materials specifically defined as to the dosage of *Pr* or *pr* results in the confirmation of a gene:enzyme (*Pr*:hydroxylase) relationship that has long been thought to be true. The specific activity ratio for the hydroxylase in seedlings, considering *Pr Pr, Pr pr* and *pr pr* stocks is 3:1:0.15, respectively. The specific activity ratio for aleurone tissue of *Pr Pr Pr, Pr Pr pr, Pr pr pr,* and *pr pr pr* is 3.4:1.4:1:0. *Pr Pr* and *pr pr* stocks were assayed for leaf sheath activity over a six week period during the growing season. Anthesis occurred during the fifth week at or near the peak of hydroxylase activity, at which time the *pr pr* tissue had 18% of the hydroxylase activity found in the *Pr Pr* tissue.

When the flavonoid compounds in aleurone tissue were extracted and identified, cyanidin and pelargonidin were both found in all the stocks. For *Pr Pr Pr, Pr Pr pr, Pr pr pr* and *pr pr pr*, quantitative data for cyanidin and pelargonidin and cyanidin/pelargonidin ratios were 9.0:5.6:2.7:0.1, respectively. Reducing this to its simplest form gives a ratio of 3.3:2.1:1:0.03, which is very close to the gene dosage ratio of 3:2:1:0. The data obtained for the individual pigments did not follow this fixed ratio in the same manner. Thus, it would seem that the pigment ratio is more closely controlled by the gene dosage than is the individual pigment concentration.

R. Larson

A mutation affecting the chloroplast coupling factor is located on 1S

A mutation designated *cfr*-2018* (a lab symbol, not yet an official gene symbol) has been found that, in homozygous individuals, results in a severe reduction of the chloroplast H^+-ATPase, also known as the coupling factor (CF) complex. Heterozygous individuals appear completely normal. The defect first appeared as a severe reduction in a number of leaf proteins in an SDS-PAGE analysis of seedlings from an F2 population. The relative sizes and quantities of the affected proteins and their location on thylakoid particles indicated that the CF complex was involved.

Rocket immunoelectrophoresis using antibody raised against the F1 sector of pea CF complex (supplied by Doug Randall) demonstrated that the cross-reactive F1 sector of the maize CF complex in the mutant was indeed reduced to 1-2% of normal levels. Evidence that the Fo sector is similarly reduced comes from the reduction of a prominent protein with an M*r* of 5.5 Kd (on SDS-urea-gradient gels) that I think is subunit III of the Fo sector (The CF complex is composed of 2 sectors, F1 and Fo; F1 is composed of five different proteins and is located externally on the thylakoid membrane, and is attached to the Fo sector which is composed of three different proteins that are located within the membrane). No other thylakoid protein complexes appear to be affected by *cfr*-2018* and thus it is unique among those mutations that are known to affect the CF complex, i.e. *hcf*-38* and *hcf*-43*.

The mutant plant has slightly pale green leaves that become elongated and droopy, stops growing after the fourth leaf has expanded; and is usually dead by four to five weeks after germination. Occasionally a mutant individual will resume slow growth and produce a terminal infertile ear on a very odd-looking 30 cm tall plant.

The mutant phenotype of stunted plants and reduced CF proteins was uncovered by the B-A translocation TB-1Sb but not by TB-1Sb-2Lc. From crosses made with the TB-1Sb stock onto two different +/*cfr* plants the progeny segregated 22 mutants among the 133 progeny tested. The mutation was not uncovered among the progeny of crosses of the TB-1Sb-2Lc stock onto four individuals in the F2 family segregating for the mutation. The chances of missing a +/*cfr* plant among the four pollinated are 1 in 81. These data indicate that the *cfr* locus is on the short arm of chromosome 1 and possibly distal to *vp5*, which is located at map position 1 and is uncovered by TB-1Sb-2Lc. I'm grateful for the help of Dan England who set up and harvested the TB crosses.

Craig Echt

Photo-induction of leaf lesions in *Les1* plants

Factors previously shown to promote leaf lesion development in *Les1* plants are genetic background (i.e. W23), temperatures below 30 C, wounding (Hoisington et al. 1982 Devel. Biol. 93:381) and 5% propanol (Ray & Walbot 1984 MNL 58:190). I've now found that light is a strong promoter of lesion formation. In *Les1/Les1* seedlings the absence or great reduction of light inhibits lesions from forming at temperatures below 30 C, and at wound sites. A 5% propanol solution appears not to be an effective inducer of lesions in young seedlings at the light intensities used in these experiments.

Les1 wt1 homozygotes from an F2 population were scored by the *wt1* phenotype before lesion expression began. Because *Les1* is 1 m.u. from *wt1* (Hoisington, this issue) very few of the *wt1* seedlings will fail to be homozygous for *Les1*. Lesions began to appear on leaf #1 2 to 4 days after seedling emergence and, spreading down the leaf from tip to base, covered the entire leaf by 7 to 9 days under the growth conditions used (25 C, 14h days with 310 or 600 microEinsteins/m^2/sec; 20 C nights). When a section of the first leaf was covered with a reflective opaque mask before or at the beginning of lesion formation, lesions seldom developed in the covered area. In most cases covering the leaf also prevented preexisting lesions from expanding and prevented lesions from forming around wound sites. This inhibition of lesion formation continued as long as the

masks were left in place, even when the non-covered portions of the leaf had become completely necrotic due to lesion development. The masks were 1.5 x 4 cm, provided a 2-3 mm air space around the covered section, were coated with aluminum foil to reflect most of the radiant heat, and had a matte black finish on their leaf-side surfaces to reduce any reflected light entering through the open ends. They were held in place with a paper clip or with rubber bands.

Once lesions had formed proximally to the covered sections removal of the masks resulted in the exposed areas becoming densely covered with lesions or, more often the case, completely necrotic within 24 h. Re-covering of the exposed areas at various intervals after exposure demonstrated that lesions can be inhibited by about 50% when recovered within 4 h. Shorter intervals have not yet been studied. Re-covering after 6 h had no effect on subsequent lesion development. The same results were found when the entire plant was placed in the dark after 4 or 6 h exposure. This indicates that a certain amount of light is required for lesion initiation to occur but the subsequent necrosis occurs in the dark. I do not yet know if darkness is an absolute requirement for extensive necrosis to occur. I have observed lesions developing during the course of a day but they were always few and small.

In the instances where lesions did develop under the opaque masks they were small and few in number or, if large, were limited to the midrib region or the edge of the covered area. Whether these lesions are due to light leaks or to certain physiological or genetic conditions is not yet known.

The absence of a lesion-gradient and the rapid appearance of lesions in the uncovered areas indicates that the developmental or physiological signal(s) that determine tip-to-base lesion development can occur in the dark. The severity of lesion formation in the uncovered areas was greater than that in the surrounding leaf and was usually marked by total necrosis, not descrete lesions. Perhaps the factors that normally limit lesion initiation or expansion, or both, in the leaf require exposure to light for a certain length of time to become functional. Another possibility is that the factors that limit lesion initiation or expansion, or both, are induced by lesion necrosis. Without any light to trigger necrosis all of the cells become set (preinitiated?) to form lesions since there are no signals to prevent them from doing otherwise. If this latter possibility is what is actually occurring then lesion limitation appears to be a type of hypersensitive response.

Preliminary experiments using colored filters in place of opaque masks seemed to indicate that lesion development is related to general photosynthetic activity. Yet this relationship is not a straightforward one since lesions form quite well in the white sectors of *wd1*, ring-9 (*Wd, C-I*) or *j1* plants (D. Hoisington, this issue).

The tentative conclusions of these observations are that lesion-forming potential and lesion necrosis are (usually) light independent while lesion initiation is (usually) light dependent. Is, then, *Les1* a light regulated gene? The next set of experiments will try to answer this question.

Craig Echt

Preliminary summary of B-A translocation breakpoints

Listed below are the approximate breakpoints in the A chromosome of a number of simple B-A translocations. The data are compiled from several sources. All data except those published by Roman and Ullstrup (1951), and by Lin (1979), are considered to be preliminary and should not be cited in publications without obtaining permission from the individual investigators concerned.

Translocation	Approximate breakpoint	Investigator
T8-1Sb	1S .05	H. Roman and A. J. Ullstrup[1]
T8-1La	1L .2	H. Roman and A. J. Ullstrup[1]
T8-3Sb	3S .5	R. J. Lambert[2]
T8-3Lc	3L .55-.65[3]	B. Kindiger and J. B. Beckett, 1985 data
T8-3La	3L .1	H. Roman and A. J. Ullstrup[1]
T8-4Sa	4S .25	H. Roman and A. J. Ullstrup[1]
T8-4Lc	4L .15-.20	B. Kindiger and J. B. Beckett, 1985 data
T8-4Lf	4L .15-.20	B. Kindiger and J. B. Beckett, 1985 data
T8-5Sc	5S .3	B. Kindiger and J. B. Beckett, 1984 data
T8-6Sa	6S .5 (NOR)	H. Roman and A. J. Ullstrup[1]
T8-6Lb	6L .65	B. Kindiger and J. B. Beckett, 1984 data
T8-6Lc	6L .11	R. V. Kowles and J. B. Beckett[4]
T8-7Lb	7L .3	H. Roman and A. J. Ullstrup[1]
T8-7Lb	7L .34	B.-Y. Lin, this newsletter
T8-8La	8L .7	H. Roman and A. J. Ullstrup[1]
T8-8Lc	8L .25±.05	M. T. Chang and J. B. Beckett, this newsletter
T8-9Sb	9S .4	H. Roman and A. J. Ullstrup[1, 4]
T8-9Sd	9S .08±.24	R. V. Kowles and J. B. Beckett
T8-9Sd	9S centric heterochromatin	B. Kindiger and J. B. Beckett, 1985 data
T8-9La	9L .4	H. Roman and A. J. Ullstrup[1]
T8-9La	9L .47	C. Prywer, A. E. Longley and E. G. Anderson[6]
T8-9Lc	9L .1	B. Kindiger and J. B. Beckett, 1985 data
T8-10Sc	10S .3 or closer	B.-Y. Lin, unpublished data, 1986
T8-10L18	10L centromere	B.-Y. Lin[5]
T8-10L19	10L centromere	B.-Y. Lin[5] and this newsletter
T8-10La	10L .35	H. Roman and A. J. Ullstrup[1, 4]
T8-10Lb	10L .34±.17	R. V. Kowles and J. B. Beckett[4]
T8-10L30	10L .13	B.-Y. Lin, this newsletter
T8-10L32	10L .74	M. Maguire, personal communication

[1] Agron. J. 43:450-454, 1951
[2] MGCNL 46:208-222, 1972
[3] conspicuous lack of pairing between the A and A-B chromosomes, so more than a simple B-A translocation may be involved
[4] MGCNL 56:61, 1982
[5] Genetics 92:931-945, 1979
[6] MGCNL 28:17, 1954

B. Kindiger, B.-Y. Lin, M. T. Chang, & J. B. Beckett

An update on lesion mutants

For the past several years I have been identifying and mapping mutations whose phenotypes resemble those of the lesion mutants reported by Neuffer and Calvert (J. Hered. 66:265-270, 1975). The table lists the 16 dominant and 8 recessive mutations that have proven to be due to single dominant or recessive factors. Included in the table are the lab symbol and (where appropriate) the proposed gene symbol; whether the mutant arose in M. G. Neuffer's various EMS plots; a description of the lesion type; and any available chromosome linkage information. Three of the dominants and five of the recessives were

CURRENT LIST OF LESION MUTANTS UNDER STUDY

LAB SYMBOL[1]	GENE SYMBOL	MUTANT TYPE[2]	LESION TYPE: TIMING	SIZE	COLOR	LOCATION: CHR	LINKAGE[3]
			- DOMINANTS -				
Les-843*	*Les1*	EMS	Early	Medium	Necrotic	2S	1 mu from *wt1*
Les-845*	*Les2*	EMS	Late	Small	White	1S	1 mu from *sr1*
Les-1375*	*Les4*	EMS	Late	Large	Necrotic	2L	
Les-1378*		EMS	Late	Large	Necrotic	2L	
Les-1438*		EMS	Early	Medium	Necrotic		
Les-1442*		EMS	Early	Medium	Necrotic		
Les-1449*	*Les5*	EMS	Late	Small	White	1S	20 mu from *sr1*
Les-1451*	*Les6*	EMS	Late	Medium	Necrotic	10S	
Les-1453*		EMS	Early	Medium	Necrotic	10S	
Les-1461*	*Les7*	EMS	Late	Small	Pale green	1L	unlinked to *br1*, *bz2*, *gl1*, *bm2*
Les-A607*		SPON	Early	Medium	Necrotic	2L	
Les-2003*		EMS	Early	Medium	Necrotic		
Les-2004*		SPON	Early	Medium	Necrotic	2S	
Les-2005*	*Les8*	EMS	Late	Small	Pale green	9S	
Les-2007*		EMS	Late	Medium	Necrotic		
Les-2008*	*Les9*	EMS	Late	Small	Necrotic	7	3 mu from *ra1*
Les-2016*		EMS	Late	Small	Pale green	10S	
Les-A762*		SPON	Early	Medium	Necrotic		
			- RECESSIVES -				
lls	*lls1*	SPON	Early	Large	Necrotic	1S	20 mu from *sr1*
les-A467*		SPON	Late	Large	Necrotic		
les-1395*		EMS	Late	Small	Pale green		
les-501B*	*lls1-501B*	EMS	Early	Large	Necrotic	1S	allele of *lls1*
les-1521C*		EMS	Late	Small	Pale green		
les-2012*		SPON	Late	Medium	Necrotic		
les-2013*		SPON	Late	Small	Pale green		
les-A721*		SPON	Late	Large	Necrotic		allele of *les*-A467*

[1] All mutants recovered by Neuffer and co-workers except for: *Les*-A607*, J. Kermicle; *lls1*, F. Troyer; *les*-A467*, M. Blanco; *les*-A721*, C. Davis.

[2] All mutants seen in either the M1 or M2 generation following EMS treatment of pollen are considered EMS mutants; all others are considered of SPONTANEOUS origin.

[3] Linkage data presented is to nearest known gene.

spontaneous mutations, arising in non-EMS treated families at Missouri or supplied to us by fellow maize researchers. The remaining mutants arose following the treatment of maize pollen with EMS. Of these 24 mutants, 15 have been located to chromosome or chromosome arm and 7 have further linkage information available. None of the dominant mutants have proven to be allelic, while two pairs of alleles have been found in the recessive cases. It should be noted that several of the dominants are located on the same chromosome and allelism tests are still in progress for most of these. Until these tests are complete, only one of the mutants located on a chromosome has been given a gene symbol. Stocks segregating for all mutants given gene symbols will be sent to the Coop. Additional stocks and mutants are available from myself.

Dave Hoisington

Further localization of *nec4* on chromosome 2S

In MNL 57:157, I reported linkage data placing *nec4* approximately 4 map units from *gl2* on chromosome 2S. At that time it was impossible to determine whether *nec4* was proximal or distal to *gl2*. Additional linkage data analyzed last summer, involving *nec4*, *d5* and *B1*, indicate very tight linkage of *nec4* with *d5* (0/142 crossovers). Therefore, *nec4* appears to be proximal to *gl2* and very near *d5* on chromosome 2S. Linkage data for both crosses are presented here, since the actual linkage data for the cross involving *nec4* and *gl2* were not presented in MNL 57. All paired recombination values were determined by the maximum likelihood method.

```
          +    +   nec4  bl
F1  =  ------------------
       lgl  gl2   +    Bl
```

TYPE = Repulsion, Backcross

INDIVIDUAL TOTALS

lgl	gl2	nec4	Bl	TOTAL
46 (48.9%)	51 (54.3%)	39 (41.5%)	48 (51.1%)	94

RECOMBINATION CLASSES

Rgn	Genotype	#	Totals	Expected
P	+ + nec4 +	24		
	lgl gl2 + Bl	32	56	54.3
1	lgl + nec4 +	8		
	+ gl2 + Bl	8	16	15.6
2	lgl gl2 nec4 +	0		
	+ + + Bl	1	1	2.4
3	lgl gl2 + +	6		
	+ + nec4 Bl	7	13	15.6
1,2	+ gl2 nec4 +	0		
	lgl + + Bl	0	0	0.7
1,3	+ gl2 + +	5		
	lgl + nec4 Bl	0	5	4.5
2,3	+ + + +	3		
	lgl gl2 nec4 Bl	0	3	0.7
1,2,3	lgl + + +	0		
	+ gl2 nec4 Bl	0	0	0.2

PAIRED RECOMBINATION VALUES

```
  lgl --- gl2    0.2234 +/- 0.0430
  lgl --- nec4   0.2660 +/- 0.0456
  lgl --- Bl     0.3191 +/- 0.0481
  gl2 --- nec4   0.0426 +/- 0.0208
  gl2 --- Bl     0.2021 +/- 0.0414
 nec4 --- Bl     0.2234 +/- 0.0430
```

```
          nec4  +   bl
F1  =  ------------
          +    d5   Bl
```

TYPE = Repulsion, Backcross

INDIVIDUAL TOTALS

nec4	d5	bl	TOTAL
100 (56.2%)	78 (43.8%)	97 (54.5%)	178

RECOMBINATION CLASSES

Rgn	Genotype	#	Totals	Expected
P	nec4 + bl	87		
	+ d5 +	68	155	155.0
1	+ + bl	0		
	nec4 d5 +	0	0	0.0
2	+ d5 bl	10		
	nec4 + +	13	23	23.0
1,2	nec4 d5 bl	0		
	+ + +	0	0	0.0

PAIRED RECOMBINATION VALUES

```
 nec4 --- d5    0.0000 +/- 0.0000
 nec4 --- bl    0.1292 +/- 0.0251
   d5 --- bl    0.1292 +/- 0.0251
```

Dave Hoisington

Les9 is linked to *ra1* on chromosome 7

Les9, which was reported to be on chromosome 7 by linkage with *wx*T7c-9(4363) (MNL 58:83) was testcrossed with an *o2 v5 ra1 gl1* stock. *Les9* was originally designated *Les*-F331035142*. It was subsequently given the lab symbol *Les*-2008*, and the gene symbol proposed in this issue. Scoring of the progeny last summer indicates that *Les9* is linked to *ra1*. With the low number of progeny scored, it is not possible to determine the exact placement of *Les9* with respect to *ra1*. Additional progeny will be scored next summer. All paired recombination values were calculated by the maximum likelihood method.

Dave Hoisington

F1 = + + + Les9 + / o2 v5 ra1 + gl1

TYPE = Coupling, Backcross

INDIVIDUAL TOTALS

o2	v5	ra	Les9	gl	TOTAL
46 (48.4%)	49 (51.6%)	46 (48.4%)	46 (48.4%)	42 (44.2%)	95

RECOMBINATION CLASSES

Rgn	Genotype	#	Totals	Expected
0	+ + + Les9 +	38		
	o2 v5 ra + gl	32	70	68.1
1	o2 + + Les9 +	5		
	+ v5 ra + gl	7	12	10.8
2	o2 v5 + Les9 +	1		
	+ + ra + gl	0	1	3.8
3	o2 v5 ra Les9 +	0		
	+ + + + gl	0	0	2.2
4	o2 v5 ra + +	6		
	+ + + Les9 gl	1	7	7.1
1,2	+ v5 + Les9 +	1		
	o2 + ra + gl	0	1	0.6
1,3	+ v5 ra Les9 +	0		
	o2 + + + gl	0	0	0.4
1,4	+ v5 ra + +	0		
	o2 + + Les9 gl	0	0	1.1
2,3	+ + ra Les9 +	0		
	o2 v5 + + gl	2	2	0.1
2,4	+ + ra + +	1		
	o2 v5 + Les9 gl	0	1	0.4
3,4	+ + + + +	1		
	o2 v5 ra Les9 gl	0	1	0.2
1,2,3	o2 + ra Les9 +	0		
	+ v5 + + gl	0	0	0.0
1,3,4	o2 + + + +	0		
	+ v5 ra Les9 gl	0	0	0.0
2,3,4	o2 v5 + + +	0		
	+ + ra Les9 gl	0	0	0.0
1,2,3,4	+ v5 + + +	0		
	o2 + ra Les9 gl	0	0	0.0

PAIRED RECOMBINATION VALUES

o2 --- v5	0.1368	+/- 0.0353
o2 --- ra	0.1684	+/- 0.0384
o2 --- Les9	0.1579	+/- 0.0374
o2 --- gl	0.2105	+/- 0.0418
v5 --- ra	0.0526	+/- 0.0229
v5 --- Les9	0.0421	+/- 0.0206
v5 --- gl	0.0947	+/- 0.0300
ra --- Les9	0.0316	+/- 0.0179
ra --- gl	0.1053	+/- 0.0315
Les9 --- gl	0.0947	+/- 0.0300

Zygomere mapping

Zygomeres are units that are responsible for chromosome pairing. Their existence is, as yet, hypothetical. The length of the synaptonemal complex (SC) is very much shorter than the length of the DNA. In corn, for example, the SC:DNA ratio is 1.5:10,000 (Gillies). This implies that there is a special class of DNA responsible for chromosome pairing, because a random sample of DNA from each chromosome would have a very low probability of being homologous. These specialized DNA segments will be called zygomeres.

One way to establish the existence of zygomeres is to study preferential pairing in trisomes that have two homologous chromosomes and one odd homoeologous chromosome. Preferential pairing can be determined by its effects on genetic ratios as shown in Table 1. When the trisomes are used as the pollen parent only the monosomic gametes need to be considered because disomic gametes function in fertilization only very rarely. If only homologous pairing occurs, then only homogenetic bivalents (BU-A) will be formed, and all the gametes will be *g*. If the preferential pairing factor (p) has a value, then the random ratio of 1 *G*:2 *g* will change. The "frying pan" trivalents (T-3A and T-3B) behave like the bivalent and univalent types (BU-A and BU-B) because the chromosomes forming the "pan" will disjoin and go to opposite poles. This assumes that the chromosomes are reductional (*GG* or *gg*) instead

Table 1
Effect of preferential pairing on genetic ratios

Pairing configuration		freq.	Monosomic gametes expected G	g	
Bivalent and univalent					
Homogenetic	BU-A	1/3 + p	0	all	
Heterogenetic	BU-B	2/3 - p	1/2	1/2	
Trivalents					
Semihomogenetic	T-1A	2/3 + p	0	all	(alt. disj.)
			1/2	1/2	(adj. disj.)
Semiheterogenetic	T-1B	1/3 - p	all	0	(alt. disj.)
			0	all	(adj. disj.)
Semihomogenetic	T-3A	1/3 + p	0	all	
Semiheterogenetic	T-3B	2/3 - p	1/2	1/2	
Neutral	T-2		1/3	2/3	
	T-4		1/3	2/3	

of equational (*Gg*) as the result of crossing over. The chain trivalents (T-1A and T-1B) will not give a 1 *G*:2 *g* ratio if the frequency of alternate segregation is greater than the random value of ⅓. The frequency and placement of chiasmata are probably affected by the environment, and different relative frequencies of pairing configurations will result with concomitant changes in genetic ratios. Therefore, the expression of preferential pairing is somewhat variable.

Trisome 3 plants (*a Sh2/a Sh2/a sh2* or *a Sh2/a sh2/a sh2*) with all standard chromosome 3's have been crossed with many sources of possible homoeologous chromosomes that are marked with *A Sh2*. The progeny of these crosses (*A Sh2/a Sh2/a Sh2, A Sh2/a Sh2/a sh2*, or *A Sh2/a sh2/a sh2*) are crossed as the male with *a sh2/a sh2* testers. Four inbred lines (B41, Hy, 38-11, and Tr) whose chromosome 3's produced a strong preferential pairing response have been selected for an intensive study. Hybrids between these lines and hybrids between them and the standard have been used in crosses with the standard trisome 3 line. Recombinant chromosome 3's with different patterns of zygomeres were anticipated. The results are shown in Table 2, statistically evaluated in Table 3.

Table 2

Preferential pairing in trisomic 3 heterozygotes

Source of odd chrom.	No. of plants tested	No. of gametes tested	% odd (A or Sh2)	Number of plants with transmission rates of: 6*	9	12	15	18	21	24	27	30	33	36	39	42
Std.	51	59,007	33.25	-	-	-	-	-	-	-	-	10	26	14	1	-
B41	68	69,325	21.33	1	1	4	13	6	11	16	8	6	2	-	-	-
Hy	60	63,138	24.01	-	-	-	2	10	13	10	13	9	3	-	-	-
38-11	64	72,959	23.63	-	-	3	1	8	9	19	17	4	3	-	-	-
Tr	16	20,007	37.43	-	-	-	-	-	-	-	1	-	2	5	3	5
B41/Hy	17	15,652	24.36	-	-	1	2	1	2	2	2	6	1	-	-	-
B41/38-11	15	16,717	22.98	-	-	1	1	-	4	4	2	2	1	-	-	-
Hy/38-11	18	21,192	21.84	-	-	-	4	2	4	2	4	-	1	-	1	-
B41/Tr	12	17,388	33.24	-	-	-	-	-	-	1	-	3	4	1	3	-
B41/Std (A)	26	28,691	24.77	-	-	-	2	6	1	5	5	3	2	-	2	-
" " (Sh2)	14	17,978	28.04	-	-	-	-	-	3	1	1	5	3	1	-	-
Hy/Std (A)	28	28,836	26.59	-	-	1	-	-	1	2	3	5	5	7	-	-
" " (Sh2)	35	38,768	31.98	-	-	-	1	-	1	3	5	7	6	8	2	2
38-11/Std (A)	20	25,596	28.62	-	-	-	-	-	1	3	5	2	2	2	-	-
" "(Sh2)	22	26,238	27.41	-	-	1	1	1	2	2	4	4	3	3	1	-

* The numbers in this row indicate the midpoints of classes that cover 3 percentage points (thus 6 covers the frequencies from 4.51 to 7.50).

Table 3

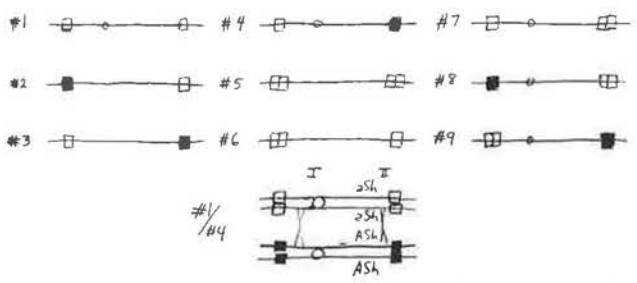
Statistical analysis of data from Table 2 with numbers and percentages of plants with transmission rates of the odd chromosome <1/3 (-), >1/3 (+), or 1/3 (N) as indicated by chi-square tests.

Source of odd chromo.	No. of plants	(-) Number .01	.05	N	(+) .05	.01	(-) Percent .01	.05	N	(+) .05	.01
Standard	51	1	2	46	1	1	1.96	3.92	90.19	1.96	1.96
B41	68	65	-	3	-	-	95.59	0.00	4.41	0.00	0.00
Hy	60	55	-	5	-	-	91.67	0.00	8.33	0.00	0.00
38-11	64	60	1	3	-	-	93.75	1.56	4.69	0.00	0.00
Tr	16	1	-	4	2	9	6.25	0.00	25.00	12.50	56.25
B41/Hy	17	11	4	2	-	-	64.70	25.53	11.76	0.00	0.00
B41/38-11	15	14	-	1	-	-	93.33	0.00	6.67	0.00	0.00
Hy/38-11	18	16	-	1	-	-	88.89	0.00	5.56	5.56	0.00
B41/Tr	12	1	1	6	-	4	8.33	8.33	50.00	0.00	33.33
B41/Std. (A)	26	20	1	3	-	2	72.92	3.85	11.54	0.00	7.69
" " (Sh)	14	6	3	5	-	-	42.86	21.43	35.71	0.00	0.00
Hy/Std (A)	28	13	1	14	-	-	46.43	3.57	50.00	0.00	0.00
" " (Sh)	35	11	3	13	3	5	31.42	8.57	37.14	8.57	14.28
38-11/St.(A)	20	11	3	5	1	-	55.00	15.00	25.00	5.00	0.00
" " (Sh)	22	11	3	7	-	1	50.00	13.64	31.82	0.00	4.54

If the data in Table 2 are plotted graphically, in most cases a normal curve is approximated. In some cases, such as B41, a bimodal distribution is found. In all cases the variability of preferential pairing allows for the upper tail of these normal curves to extend over the random pairing point (33.3%) and gives false readings for normality. The means of transmission rates do not fluctuate significantly from year to year.

The data in Table 2 can be explained by a simple model. It is assumed that chromosome pairing is initiated by two zygomeres, one each at or near the ends of the chromosome. There are nine possible patterns, as shown in Figure 1. The standard chromo-

Fig 1



some 3 is #1; the B41, Hy, and 38-11 chromosome 3's are #2, #3, or #4, and the Tr chromosome 3 is #5. The zygomeres indicated by the black squares are non-homologous to the standard white square zygomeres. The Tr zygomeres are thought to be homologous to the standard zygomeres, but are stronger. A Tr zygomere pairs more readily with a standard zygomere than do two standard zygomeres with each other. It is hypothesized that zygomeres consist of many tandemly repeated units and the Tr chromosomes have more units than the standard. Thus it gives *p* a negative value and the ratio is >1 *G*:<2 *g* (See Table 1).

The results from using hybrids B41/Hy, B41/38-11, and Hy/38-11 as the source of the odd chromosome indicate that the zygomere patterns of the hybrids are #2/#4, #3/#4, or #4/#4. They are not #2/#3 because crossing over would produce about ½ #1 chromosomes, assuming the zygomeres would show 50% recombination if they are at the ends of the chromosomes. The highest numbers of apparent #1 chromosomes, showing normal (33%) transmission, are from B41/Hy, where there are 11.76%. One or both of these chromosome pairs must be #4. Where the hybrid B41/Tr was used it may be seen that data, while limited, indicate segregation for <⅓ and >⅓ transmission of A, along with ⅓ transmission cases. Crossing over should produce #8 and #9 zygomere patterns which would be equivalent to #1 if these zygomeres acted independently to cancel out each other's effects.

The results from using the inbred line/standard hybrids as the source of the odd chromosome are quite instructive. If the Hy/Std is #2/#1 we would expect an equal frequency of *A Sh2* #1, *A Sh2* #2, *a Sh2* #1, and *a Sh2* #2 chromosomes following crossing over. The data show that this appears to be true, although there is an unexplainable enhancement of pairing affinity in 22.85% of the *a Sh2* chromosomes.

Table 4
Gametes expected from a #1 a Sh2/#4 A Sh2 plant

	A Sh2				a Sh2			
	#1	#2	#3	#4	#1	#2	#3	#4
No exchanges	0	0	0	2	2	0	0	0
One exchange in I	0	0	1	1	1	1	0	0
One exchange in II	0	1	0	1	1	0	1	0
Two exchanges								
I and II								
2 strand	1	0	0	1	1	0	0	1
3 strand	0	1	1	0	1	0	0	1
3 strand	1	0	0	1	0	1	1	0
4 strand	0	1	1	0	0	1	1	0

The other two inbred line/standard hybrids (B41/Std and 38-11/Std) are probably #4/#1. The consequences of crossing over in this case are shown in Table 4. If there are two exchanges the frequencies of *A Sh2* and *a Sh2* chromosomes with patterns of #1, #2, #3, and #4 are all ¼. The chromosomes #2, #3, and #4 will give preferential pairing. The data are in fairly close agreement.

The mapping of zygomeres, like any gene, should be possible although very difficult.

G. G. Doyle

Breakpoints of TB-10L19, TB-10L30 and TB-7Lb

TB-10L19 has a break in the proximal part of the distal heterochromatic region of the B chromosome. Its break on chromosome 10 is very close to the centromere on the long arm (Lin, Genetics 92:931-945, 1979). Since the 10-B chromosome carried the major portion of the distal heterochromatic region of the B chromosome, the exact break position could not be determined due to presence of the heterochromatic mass at its end. Additional observations to be reported here indicate the break position being right at the 10 centromere. A plant carrying the 10 10-B B-10 B-10 chromosome constitution was studied at pachytene stage. Two B-10 chromosomes were seen to pair most of the time. One of the two B-10s paired occasionally with 10 and 10-B chromosomes to become a trivalent with complete pairing in two arms but an incomplete pairing in the other arm as would be expected. There were several exceptional cells where 10, 10-B and B-10 were associated in a T-configuration. Pairing in three arms was perfect. Two of the three arms were from chromosome 10, and the third arm is the distal heterochromatic region of the B chromosome. The centromere of chromosome 10 was located at a place where the three arms met. What is unusual about these cells is that the B-10 chromosome did not carry the expected B structures, i.e., the centromeric knob, the proximal euchromatic region and the proximal portion of the distal heterochromatic region. The B-10 chromosome of these cells, however, carried the same B part as that borne on the 10-B chromosome, i.e., the distal heterochromatic region. In other words, this B-10 chromosome did not have the centromere of either chromosome 10 or the B chromosome. The fact that it is acentric and present at the pachytene stage, suggests a premeiotic origin.

The breakpoint of TB-10L30 was also identified by virtue of its abnormal structure (Lin, MGNL 52:114-116, 1978). This translocation has a break close to the junction between the proximal euchromatic region and the distal heterochromatic region on the B long arm. The 10-B chromosome did not carry the expected B structure, i.e., the distal heterochromatic region. Instead, it carried the proximal B parts, i.e., the same B parts carried by the B-10 chromosome. The pachytene picture of a heterozygous plant had a T-configuration with complete pairing in three arms, two of which are from chromosome 10 and the other from the proximal portions of the B chromosome. The break position on the long arm of chromosome 10 can thus be measured directly. The average of three separate measurements (0.14, 0.12 and 0.13) is 0.13.

TB-7Lb has a break on the euchromatic region of the long arm of the B chromosome (Lin, MGNL 59:44-45, 1985). Its breakpoint on the long arm of chromosome 7 was measured only once with tassels of a 7 7-B B-7 B-7 plant. Chromosome 7 in this particular cell paired with the 7-B chromosome. B parts of the 7-B chromosome (including part of the proximal euchromatic region followed by the entire distal heterochromatic region) did not fold back and thus allowed an accurate measurement of the breakpoint, which is at the 0.34 position.

Bor-yaw Lin

Correction on the B breakpoint of TB-3La

TB-3Lc in MGNL 59:44-45, 1985, is incorrect. It should be TB-3La.

Bor-yaw Lin

Mutations of aleurone color factors induced by EMS

M2 ears from EMS pollen treatments of colored-aleurone stocks were screened for segregating expressions affecting aleurone color. A selection of 62 potential cases has been examined by allelism tests and B-A translocation tests. We have identified the following:

	No.	Notes
a1 allele	1	pale
bz1 allele	1	
c1 allele	2	
c2 allele	6	3 pale, 3 colorless
pr allele	9	
r allele	5	
vp1 allele	1	
vp1 allele?	1	On 3L, not a1
dek1 allele	1	
w2 (assumed)	2	On 10L, phenotype like w2
New factor(s)	5	On 5S, not a2; 1 pale, 3 colorless, 1 colorless-small kernel; homoz. inviable
Pending	2	Tests incomplete
Not recovered	13	3 colorless, 5 brown kernel, 1 floury, 2 mottled, 1 defective, 1 red
Dropped	13	1 colorless - poor expression, 6 pale - poor expression, 4 pale floury - poor expression, 2 defective - poor expression

E. H. Coe and M. G. Neuffer

Atrazine susceptibility factor

A recessive, nuclear factor for susceptibility to atrazine was defined by C. O. Grogan et al. (Crop Sci. 3:451, 1963) in inbred line GT112, which was supplied to us in tolerant and susceptible versions by G. E. Scott. Linkage to chromosome 8 (Scott and Grogan, Crop Sci. 9:669, 1969) was defined by *wx* translocations, using field applications of the herbicide and classifying F2s for healthy vs. dead plants. The recombination percentages were, for *wx* T8-9d (8L.09), 23 ± 6%; for *wx* T8-9(6673)(8L.35), less than 25 ± 10% (there were 0 plants in the *wx* dead class). The factor may be in 8L or 8S. A small test of crosses with TB-8La did not show dead seedlings in a treated field, but the germination was poor. Susceptible material can be grown in pots and can be transplanted to the field when the plants are well established. Susceptible plants have been shown to lack the usual enzyme activity for conjugation of glutathione with atrazine (R. H. Shimabukuro et al., Plant Physiol. 47:10, 1971). The atrazine susceptibility factor has not been assigned a symbol; *ats* (atrazine susceptible) would seem to be suitable.

E. H. Coe and J. B. Beckett

Silk browning and cob color: *P* locus control

In MNL 59:40, observations were reported on browning of silks at the tips, 5-10 minutes after they are cut. Red-cob, *P-WR* lines show silk browning, while most white-cob, *P-WW* lines do not—i.e., the cut silks remain green. We have conducted tests to determine the relationship of determinants for browning silks in white-cob lines to the *P* locus. White-cob inbred lines were crossed to a hybrid between W23 and K55, *P-WR/P-WW*. W23 is red-cob browning (WRB), while K55 is white-cob green (WWW). The F1 plants were selfed, and F2s from red-cob and white-cob F1 plants were grown from each line for classification of silk browning and cob color.

White-cob green (WWW) inbreds segregated, from F1s with red cobs, approximately 3 red-cob browning to 1 white-cob green in F2 (3 WRB:1 WWW). From the F1s with white cobs, all F2s were white-cob green (WWW). The WWW inbreds we have tested are A619, CI66, FR29, FR35, FR802W, FR805W, FR806W, FR807W, FR808W, FR809W, FR810W, K41, K61, K63, K150, K155, K302, K306, K814, K816, Mo15W and Mo16W.

White-cob browning (WWB) inbreds segregated, from F1s with red cobs, about 3 red-cob browning to 1 white-cob browning (3 WRB:1 WWB). From the F1s with white cobs, segregation was around 3 white-cob browning to 1 white-cob green (3 WWB: 1 WWW). The WWB inbreds we have tested are K44, K166, Ky228, Mo1W and Mo14W. Results with one additional inbred were ambiguous and need to be tested further.

Evidently browning of silks in white-cob inbred lines is due to particular alleles, *P-WWB*, in contrast to the red-cob browning (*P-WRB*) and white-cob green (*P-WWW*). The evidence does not favor a silk-browning factor separate from *P* in WWB lines, nor a green-silk factor separate from *P* in WWW lines.

E. H. Coe and Chang-deok Han

Duplicate factors for orange pericarp (*orp*)

Among the selfed M1 ears from pollen treated with EMS, there appeared one ear which segregated for kernels with orange pigment in the pericarp. The ratio was 15 normal to 1 orange. The ear also segregated 9:7 for purple vs colorless aleurone due to *C* and *R* in the treated population. The orange pigment appeared in the pericarp of both the purple and non-purple kernels. Since pericarp is maternal, tissue differences in pigmentation should not segregate among the kernels. Apparently a product from a certain genotype in the underlying endosperm tissue is diffusing into the pericarp, where it causes pigment formation. The ratio of 15:1 suggests duplicate unlinked factors, the double recessive combination being the right genotype for pigment formation. Outcross to a large embryo stock (Aho) with subsequent selfs have repeated the 15:1 segregation. Selfing 17 plants from the normal kernels of a 15:1 ear produced 9 normal ears, 6 ears segregating 3 normal:1 orange, and 2 ears segregating 15 normal:1 orange. We have tentatively designated these factors *orp1* and *orp2*, although which is which will have to await location of one to chromosome.

When planted, the orange kernels grow into small, narrow leaf, pale green seedlings which grow slowly and die before maturity. SDS PAGE analysis of 18-day endosperm and embryo tissue failed to show protein differences from normal.

M.G. Neuffer and M.T. Chang

Transposition of chromosome-breaking *Ds* to marked chromosome arms

We plan to study the expression of lethal mutant tissue in chimeras produced by loss of the normal allele in a heterozygote (+/m). Losses may be produced in a number of ways, including x-radiation, marked ring chromosomes, and chromosome-breaking *Ds* elements. We have selected the latter for extensive testing. To do this we have produced a heterozygous *Ac*, homozygous chromosome-breaking *Ds* stock (actually *P-vv/P-wr, Ds-2 R-sc* from materials generously supplied by Dr. Jerry Kermicle). We used this stock as a pollen parent in an isolated detasseled plot where the ear stocks were marked for each of the 16 chromosome arms which carry a usable marker. Marked stocks with chromosome-breaking *Ds* on 9S (McClintock) and 10L (Kermicle) are

already available. The ears produced were examined for two types of events: (1) Transposition of *Ds* between the marker and the centromere to produce mosaics of recessive mutant tissue on a non-mutant background caused by chromosome breakage and, (2) transposition of *Ds* to the marker site to give a recessive mutant case with dominant revertant sectors (or without revertant sectors in the absence of *Ac*).

For ear stocks with endosperm markers the examination was for mosaic kernels and for recessive mutable or non-mutable kernels. For stocks with seedling markers all the kernels were planted in sand benches and examined for seedlings with multiple recessive sectors and for whole seedling mutant cases with and without sectors (*Ac* and no *Ac*).

The ear stocks used are listed below according to chromosome arms:

1S +/*dek* F2	6S no good marker
1L *bz2-s*, no *Ac*	6L +/*w*-278A*, F2
2S *b, r* (used *B:Peru Ds-2* male)	7S *vp9*
2L +/*w3*, F2	7L +/*o*-874B*, F2
3S +/*cl1*, F2	8S no marker
3L *a-s sh2, dt*	8L +/*pro*, F2
4S *bt2*	9S McClintock's *C Ds*
4L *c2*	9L *dek*-744/v*, F2
5S *a2 bt pr*	10S +/*oy*, F1
5L *a2 bt pr*	10L Kermicle's *Ds-2 R-sc*

To demonstrate the success of this approach preliminary results for two arms are listed below. For the kernel mutant *dek1* (*clf* on chromosome 1S) the results are described in the next MNL item. For *oy*, a seedling mutant on chromosome 10S, the results were as follows: a total of 92,000 kernels from the cross +/*oy* x pollen stock was planted in sand benches and produced 25 *oy* seedlings, of which 23 were non-sectored and 2 were clearly *oy-m*, and 9 green seedlings with frequent tiny *oy* sectors. These potentially good *oy*, *oy-m*, and *Oy Ds* cases, respectively, will need confirmation. The frequencies cannot be used because the original events that produced them took place in the male sometime before gamete formation. They may represent many single events or many copies of a single event.

M.G. Neuffer

Expression of *dek1 (clf)* in leaf sectors

The phenotype of *clf*, located on 1S, is a colorless (blocks anthocyanin and carotenoid pigments) floury kernel with a lethal embryo. Lethality may be forestalled when immature embryos (12-16 days) are excised before lethality and placed on a standard culture medium. If cultured this way they often produce roots, but no differentiation or growth of leaf tissue. In the experiments described in the News Letter item above, *p+/p clf, RR* ears were pollinated by *P-VV* +, *Ds-2 R-sc* pollen and the ears were examined for colorless floury mosaics, which would indicate transposition of *Ds* to a position between *clf* and the centromere. Several good cases were found and planted. They produced green seedlings with tiny sectors of almost white tissue indicating a failure of chlorophyll. Some of these sectors were several cells wide and several cm long. In these large sectors the tissue was distorted in growth, being raised on the top surface and indented on the underneath surface of the leaf. The larger sectors also showed some necrosis, the innermost cells apparently dying.

M.G. Neuffer

Checking for possible contaminants among new mutants

We have been surveying mutants resulting from EMS mutagenesis, with special attention being paid to those of possible interest to maize breeders. Some have stood out as exceptional (e.g., *Wlf*-1726* has a short plant and short, wide leaves which are dark green; *lgp*-2086* is larger in several dimensions, a little earlier and stays green longer). Some families have segregated for three or more out of a considerable list of divergent traits (tillering, earliness, husk flags, many kernel rows, many tassel branches, pale kernels often with larger dents). Some of these families look like they might be contaminations because of the many segregants, some of them possibly linked, so an initial test for purity of pedigree has been run using isozyme electrophoresis.

Four families with possible contamination (set C), four thought not to be contaminated (set B), and five checks (set A) were sent to Stephen Smith at Pioneer Hi-Bred International, Inc., to be analyzed by electrophoresis. The test materials resulted from fertilization of inbred line A632 by EMS-treated pollen of inbred line Mo17, selfing of the M1, and, in most cases, outcrossing to Mo17. Set C contained families segregating (1) *erl*-2077* (early by 6 days, also tillered, thick cob); (2) *lgp*-2087* (large plant, also many kernel rows—usually 17); (3) *Wlf*-1726* (above); and (4) *mbr*-2088* (many tassel branches, also tillering, earliness, husk flags, many kernel rows, pale kernels). Set B had (1) *Erl*-2102* (early by 3-5 days, also small plant); (2) *erl*-1729* (early, also short plant); (3) *shp*-1749* (short plant); (4) *lgp*-2086* (above).

There were 21 isozyme loci included in the assay representing most of those normally studied at Pioneer and elsewhere. Twelve plants were examined for each test family, and eleven for each check. Set B proved to have only the allozymes of the parents. Set C was mixed; the family with *mbr*-2088*, the most likely to be contaminated, showed deviation from parental norms for seven isozyme loci involving all plants. The *erl*-2077* family had one plant with a deviant allozyme band. The *lgp*-2087* family had deviant allozymes or gave obscure bands for five

isozyme loci involving all plants, and there was difficulty in reading two of the same isozymes for *Wlf*-1726*. The Mo17 backcrosses in sets A and B showed the expected predominance of Mo17 allozymes. However, there was a problem with one check, the bulk of ten untreated F2 families. All plants had three or more of the deviant allozymes or difficulties were seen in the scoring of *lgp*-2087* and *Wlf*-1726* families. Why, we do not know. None of the deviations in the test families matched the allozymes which separate inbred line Mo20 from inbred lines A632 and Mo17, eliminating that stock as a source of contamination.

Until we know more we can only state that *mbr*-2088* can be considered a contaminant and that all of set B seems uncontaminated. Remember that occasional allozyme variants might be due to mutagenesis.

Robert McK. Bird, Stephen Smith[1] and M.G. Neuffer

[1]Pioneer HiBred International, Inc.

Les1 lesion expression in non-green leaf tissue

Lesion expression in various types of non-green leaves of *+/Les1* plants has been examined (see table). Lesion expression in leaves with a low chlorophyll content due to *Oy1-1459* is delayed over normal green sibs by approximately 2-3 weeks. When lesions do form, they are larger and generally more banded in appearance. Since it is unclear whether photosynthesis is required for lesion formation (see article by C. Echt), the delayed lesion formation may be due to a slower accumulation of some metabolite which is required to trigger lesion formation or is involved in the production of visible necrosis.

LESION FORMATION IN NON-GREEN LEAVES OF *Les1* PLANTS

TISSUE PHENOTYPE	CAUSATIVE AGENT	LESION FORMATION
oil-yellow	+/*Oy1-1459*	delayed
white	*wt1*	-
green	"	+
white	Sandoz 9789	-
green	"	+
white	*wd1* - Ring-9	+
green	*wd1* + Ring-9	+
white	*j1*	+
green	*j1*	+

Lesion formation was also examined in various white sectors in leaves. Lesions were never seen to initiate or enlarge into the white tissue of *wt1* seedlings or white leaves following treatment of seedlings with the herbicide Sandoz 9789. Lesions forming in the green tissue near the white border never enlarged into the white tissue. These findings seem to indicate that lesion formation and enlargement requires photosynthesizing tissue; however, lesions were found to form in the white stripes of *j1* (japonica) and *wd1* + Ring-9 (*Wd1 C-I*) plants. There was some indication that lesion formation in the white and perhaps layered sectors of the *wd1* plants was more pronounced than in adjacent green tissue. Lesion formation in non-sectoring green sibs of *wd1* plants was also lower, perhaps indicating that sectored and/or layered leaf tissue is more primed to initiate lesions in the presence of *Les1*. Pure white seedlings resulting from the complete loss of the ring chromosome never formed lesions. These seedlings survived for only 2-3 weeks and lesion formation may require a longer period since the *Oy1-1459* plants were delayed in their expression. It is unclear why lesions formed in one type of white tissue and not the other. Perhaps longitudinal sectors such as those formed by *j1* and *wd1* are cross-fed by neighboring green tissue differently than lateral divisions of white and green tissue. This feeding may provide a necessary metabolite for lesion formation. It is also possible that the layering, which is absent in *wt1* and Sandoz tissue, is responsible for lesion formation.

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On the use of the distal promoter in the zein genetic system

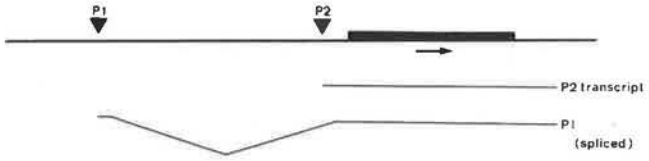
Zein is abundantly synthesized in the developing endosperm. The exact mechanism leading to the intense synthesis is unknown although the multigenic character of the system, the promoter set up and efficient translation of zein mRNAs may all contribute to this feature.

In this context our recent finding of multiple promoters before zein genes (see MNL 58:88-89, 1984) may be of relevance, in particular since the observed differences in the various promoter structures may allow a regulatory influence on efficiency of gene expression. However, for this to occur the RNAs synthesized from the P1 promoter regions lying 1 kb upstream from the start of protein synthesis must finally lead to protein synthesis.

We now have evidence which shows that this is indeed the case. Transient transformation experiments performed with cloned zein genes in the alga *Acetabularia* (system described in MNL 59:48, 1985) show that zein protein is synthesized in this system from RNA derived from the P1 region. Mutants with an impaired P1 structure and otherwise intact 5′ flanking regions do not lead to zein synthesis, indicating that the P1 structure does generate RNAs with translational capacity and that P2 is not active in this system.

The expression of the 700-800 bp long coding region from a 1.6-1.8 kb long RNA is apparently preceded by a splicing step which transfers the 5′ end of the RNA (presumably containing a cap structure) in front of the gene:

Evidence in support of such a mechanism was obtained by a combination of S1 mapping and primer extension experiments in the region of P2, and by DNA sequence comparison. The sequence of the relevant regions does show intron border sequences, branchpoint sequences and partial homology to snRNA at the expected positions.



The proposed mechanism is appealing since it accommodates the use of the cap structure of the primary transcript and also avoids the interference of the more than 15 AUG codons present in the 1 kb flanking sequences.

J. Brown, Chr. Wandelt and G. Feix

Organisation of the external spacer of nuclear rRNA genes

In maize, rRNA genes are present in thousands of copies leading to a steady supply of rRNAs. The rRNA gene units comprise the genes for the 18S, 5.8S and 25S rRNA in that order, with internal and external spacer regions. The external spacer region is of particular interest since it contains the transcription start for the primary transcript and, presumably, regulatory elements.

To gain insight into the structure of the external spacer we isolated an rRNA gene unit from a gene library of variety A619 and sequenced the spacer region.

The 3020 bp long external spacer contains 9 tandemly arranged 200 bp long repeat units with high homology and only minor insertions or deletions in some of the repeats. Interestingly, the basic repeat structure reveals some homology to the 130 bp repeat unit of the wheat rRNA gene unit isolated by Appels and Dvorak (Theor. Appl. Genet. 63:337-348, 1982). Potential start positions of RNA synthesis as well as the 3′ and 5′ ends, respectively, of the bordering 18S and 25S rRNA genes were determined by S1 mapping experiments (indicated in the scheme by the arrows).

It will now be interesting to study the influence of the repeat units on the expression of the rRNA gene unit. A comparison of this sequence from variety A619 with that from other varieties, such as the sequence obtained in the lab of J. Rubenstein (University of Minnesota), will furthermore allow the recognition of essential indispensable sequences of the external spacer region.

Ch. Toloczyki and G. Feix

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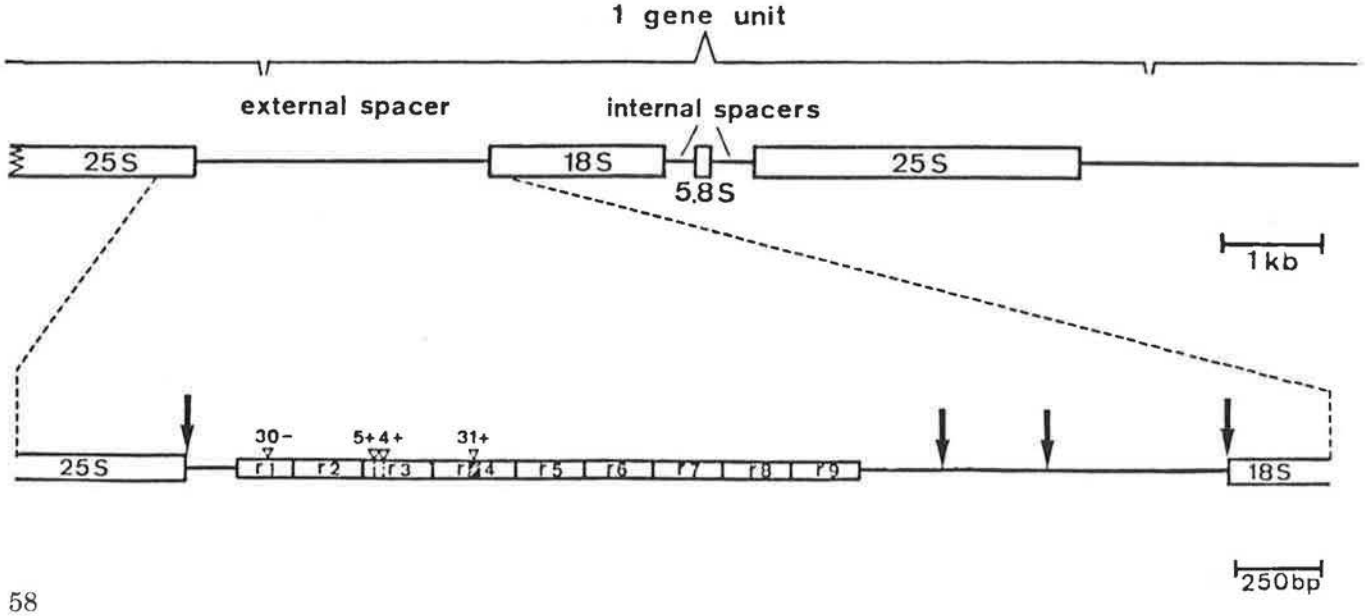
Cloning of the sucrose synthetase-2 gene

We have recently isolated and positively identified a genomic clone of the gene for sucrose synthetase-2 (*Ss2*) in maize. The clone was isolated by exploiting the partial homology between Shrunken-1 (*Sh1*) and *Ss2*. A BamHI genomic library was constructed in EMBL 3 lambda phage using DNA from a single Black Mexican sweet corn plant kindly provided by Paul Still and Prem Chourey. The library, consisting of approximately 300,000 plaques, was screened with a 3400 bp BglII fragment from the *Sh1* clone p17.6 (Sheldon et al., Mol. Gen. Genet. 190:421-426, 1983; Zack et al., in press). The probe begins in intron 2 of *Sh1* and ends in exon 14.



Nine strongly hybridizing plaques were isolated and were subjected to analysis. Of these clones, eight contained inserts that corresponded to either of two wild type *Sh1* alleles as judged by restriction mapping. The original plant from which these clones were derived was apparently heterozygous at *Sh1*. One of these alleles appeared to be identical to our original *Sh* clone, p17.6 (except that the new clones are BamHI fragments and p17.6 is an EcoR1 fragment), while the second allele exhibited small but interesting differences in its restriction patterns.

The ninth clone (designated lambda 21.2) differed markedly from the others. BamHI digestion of lambda 21.2 revealed three inserted fragments of DNA. Only one of these, a 6.6 kb BamHI fragment, hybridized to the 3400 bp BglII fragment of p17.6. The origin of the other two fragments is unclear. They may be random fragments of maize DNA which ligated to the 6.6 kb fragment during cloning, or the original BamHI digestion used to construct the library may have generated incomplete cleavage products. The 6.6 kb fragment was subcloned and is designated p21.2. Restriction maps of p21.2 and p17.6 are shown in Figure 1. The restriction maps of p21.2 and p17.6 bear only a modest resemblance. However, the two clones share substantial regions of homology as indicated by plasmid to plasmid Southern blot hybridization (solid bar). Thus, we had isolated a genomic clone with partial homology to *Sh1* from a library in which we had already identified both of the *Sh1* alleles.

We considered several possible explanations for the origin of p21.2: (1) p21.2 arose as a cloning artifact via an in vitro rearrangement of *Sh1* sequences or rearrangement of *Sh* sequences during plant growth. (2) p21.2 represents an inactive copy of the *Sh1* locus (i.e. a pseudogene). (3) p21.2 is a clone containing all or part of the sucrose synthetase-2 gene.

To test the first possibility we performed Southern blot analysis of DNA isolated from wild type and *sh1 bz-m-4* plants (*sh1 bz-m-4* lacks *Sh1* sequences). p21.2 sequences were clearly present in both wild type and *sh1 bz-m-4* stocks. Thus, p21.2 did not arise from an in vitro or in vivo rearrangement of *Sh1* sequences. Interestingly, however, the restriction fragment banding pattern observed in the two genotypes differed, indicating that a polymorphism exists between these genotypes. In principle, these stocks should be isogenic except in the region of *Sh1* on chromosome 9. This suggested to us that the p21.2 sequence may also be located on chromosome 9. Further mapping experiments have corroborated this (McCarty et al., this issue below).

In order to determine whether p21.2 represented an active gene, we examined polyA-RNA isolated from developing seeds of wild type and *sh1 bz-m-4* deletion stocks by Northern blot analysis. As expected p17.6, the *Sh* clone, hybridized strongly to an abundant 2.75 kb mRNA which was present in the wild type but absent in the *sh1* deletion stock. A second weakly hybridizing mRNA of slightly greater molecular weight could be identified in the deletion stock. p21.2 hybridized strongly to mRNA that was present in equal abundance in both wild type and deletion stocks. This mRNA is identical in size to the transcript that showed weak hybridization to *Sh1*. Weak hybridization of p21.2 to the *Sh1* transcript in the wild type was also evident. Therefore p21.2 represents an active gene distinct from *Sh1*, and this gene is most probably sucrose synthetase-2. The slightly higher molecular weight of the putative *Ss2*

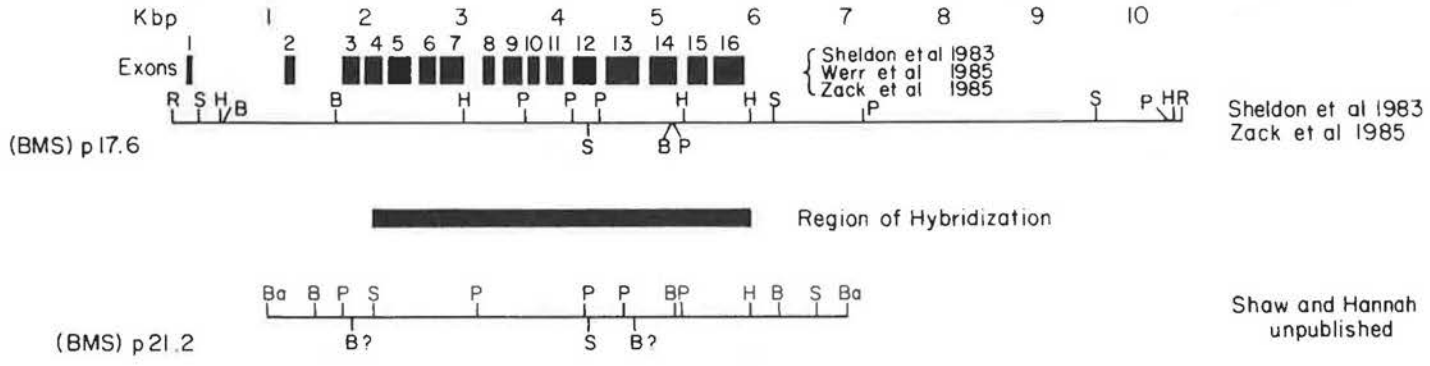


Figure 1. Restriction maps of the Shrunken-1 clone, p17.6 and the sucrose synthetase-2 clone, p 21.2. Exon-intron positions were taken from Werr et al. (1985, The EMBO J. 4:1373) as modified for allelic differences Zack et al.; (1986, Maydica, in press). Regions of hybridization are based on plasmid to plasmid Southern blots. Ba = Bam H1, S = SstI, H = HindIII, B = BglII, R = EcoR1 and P = PstI.

transcript is consistent with the observation of S. McCormick et al. (Mol. Gen. Genet. 187:494, 1982).

To positively identify the clone, we purified the p21.2 transcript from polyA-RNA extracted from *sh1 bz-m-4* by hybrid-selection with p21.2 DNA covalently linked to a solid matrix. When translated in vitro in a rabbit reticulocyte lysate system, the hybrid-select mRNA produced a single translation product with a molecular weight of 88,000 daltons, the size of the sucrose synthetase-2 subunit. Furthermore, using *Sh1* anti-serum (*Sh1* anti-serum generously provided by Paul Still and Prem Chourey will cross-react with sucrose synthetase-2) we can specifically immunoprecipitate the hybrid-select translation product. Thus, a p21.2 is a genomic clone containing the gene for sucrose synthetase-2.

From sequencing experiments we have found homology between p21.2 and p17.6 beginning at the SstI site in exon 12 of *Sh1* and extending 3′ into intron 12. Some 20 bp into intron 12 homology breaks down but resumes several base pairs before the intron 12-exon 13 border. It would appear that sequence organization of *Sh1* and *Ss2* is similar, at least in the vicinity of intron 12.

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

The sucrose synthetase-2 gene is genetically linked to *Sh1*

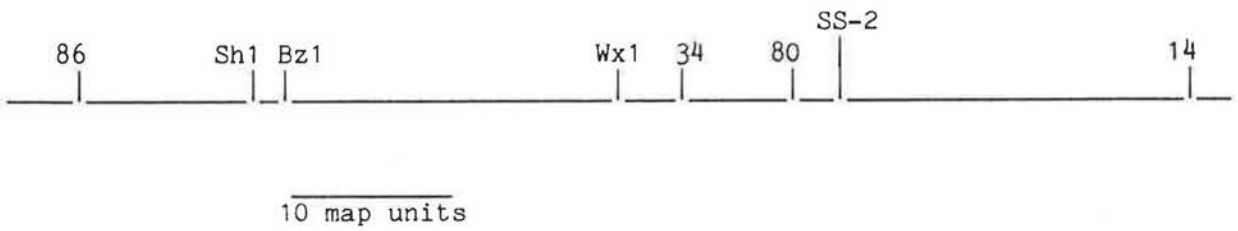
We have used a recently isolated clone of the gene for sucrose synthetase-2 (*Ss2*) to place the locus on the maize genetic map. *Ss2* is located in a region near the centromere on chromosome 9 approximately 32 map units from *Sh1*, the gene encoding the major endosperm form of sucrose synthetase.

Ss2 was assigned to chromosome 9 by Southern blot analysis of DNA isolated from a series of monosomic plants developed at NPI (Salt Lake City, Utah) using a cross involving the *r-x1* deficiency. The *r-x1* deficiency (D.F. Weber; Maize for Biological Research, University of North Dakota Press, 1982) conditions a high frequency of chromosome loss when transmitted through the female parent. From a cross of a Mangelsdorf tester as male (*R R*) with a *R-g r-x1* (in a W22 background) female, monosomic F1 plants generously supplied by Dave Weber have been obtained for all chromosomes except 1 and 5. Southern blot analysis of DNA from these plants and the parents provides a very direct means of determining the chromosomal location of molecular markers.

Based on the restriction map of p21.2 (the *Ss2* clone), digestion of maize DNA with SstI is expected to yield four fragments: two internal 2.3 kb fragments which migrate as a single intense band, and two border fragments of indeterminant size. When p21.2 was used to probe DNA digested with SstI from the parents, a normal diploid F1 and the monosomics, we observed a pattern that was consistent with this. All plants exhibited an intense band at 2.3 kb and a fainter band at 6.8 kb representing one of the border fragments. The second border fragment exhibited a size polymorphism between the parents. In the male parent (*R R*) a 12.0 kb fragment is observed, whereas in the female (*R-g r-x1*) the 12.0 kb fragment is replaced by a 4.7 kb fragment. Both the 12.0 kb and 4.7 kb fragments were present in DNA from the normal F1 (as expected) and all of the monosomics except those plants that were monosomic for chromosome 9. The chromosome 9 monosomic plants lacked the 4.7 kb fragment characteristic of the *Ss2* allele contributed by the female. This clearly demonstrated the loss of the female *Ss2* allele concomitant with the loss of chromosome 9 induced in the female by *r-x1*.

The assigment of *Ss2* to chromosome 9 is further supported by the observation that the intensity of the hybridizing bands (on equal DNA basis) of the chromosome 9 monosomic material was reduced relative to that of the parents and other monosomics. This was also evident when HindIII digests were probed. The reduced band intensity is consistent with the loss of one copy of *Ss2* with the loss of chromosome 9.

Figure 1. Position of SS-2 on Chromosome 9.



This map of the short arm region of chromosome 9 was adapted from a linkage map developed by S. Wright and T. Helentjaris at NPI, Salt Lake City, Utah. The numbers refer to loci defined by random clones.

The map position of *Ss2* was further resolved by screening 50 plants from an F2 population segregating for the SstI restriction fragment length polymorphism and other markers. As expected *Ss2* showed linkage to chromosome 9 markers. With these data *Ss2* was placed relative to two randomly cloned molecular markers (locus 80 and locus 14) that had been previously mapped at NPI (see Figure 1). *Ss2* maps 2 ± 2 map units from locus 80 and 21 ± 6 map units from locus 14. This places *Ss2* some 32 map units from *Sh1*, in the vicinity of the centromere on chromosome 9.

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A simple method for extraction of RNA from maize tissues

In the course of our studies on expression of the sucrose synthetase genes, a simple and efficient procedure was developed for the isolation of high-integrity, translatable mRNA from several maize tissues. The protocol for large scale preparation of RNA from developing kernels is as follows:

1. Ears (20-22 days post-pollination) are harvested and frozen immediately in liquid N_2 (this is done in the field). The frozen ears are stored at -90°C until needed.

2. 20 g of frozen kernels are thoroughly pulverized under liquid N_2 with a mortar and pestel. Care must be taken to avoid thawing.

3. The frozen powder is added to a 250 ml flask containing 60 ml of the following solution:

   100 mM Tris-HCl (pH 9.0)
   200 mM NaCl
   5 mM DTT
   1% (w/v) sarcosyl
   20 mM EDTA

   The frozen tissue is then dispersed and thawed by homogenization with a Polytron for 1 min. (Extensive foaming will occur, but this is not harmful.)

4. The suspension is immediately transferred to 50 ml Oak Ridge centrifuge tubes and centrifuged at 10,000 xg for 5 min.

5. The supernatant is transferred to clean Oak Ridge tubes and immediately extracted with an equal volume of 50:50:1 phenol:chloroform:-isoamyl alcohol and then centrifuged at 10,000 xg for 5 min.

6. The lower phase is removed and discarded leaving the interface and upper layer. An equal volume of chloroform/isoamyl (50:1) is added, mixed, and centrifuged as above.

7. The aqueous layer (upper) is transferred to a fresh tube and extracted a second time with chloroform/isoamyl (50:1). The aqueous upper phase is saved.

8. The volume of the extract is determined and the solution is adjusted to 2 M LiCl with a 12 M LiCl stock solution, mixed and transferred to clean, sterile Corex centrifuge tubes. These are allowed to stand overnight at 4°C.

9. The RNA is recovered by centrifugation at 10,000 xg for 10 min. The RNA pellets are thoroughly resuspended in 3 ml of 2 M LiCl and centrifuged saving the pellet. This is done twice.

10. The RNA is dissolved in 2 ml of 10 mM Tris-HCl (pH 7.5), 2 mM EDTA and centrifuged at 10,000 xg for 10 min to remove any insoluble material.

11. A 1/10 volume of 3 M Na Acetate (pH 5.2) is added followed by 2.5 volumes of ethanol. The mixture is incubated overnight at -20°C or for 1-2 h at -90°C then centrifuged at 10,000 xg for 10 min. The pellet is rinsed with 70% ethanol then resuspended in 10 mM Tris-HCl (pH 7.5), 2 mM EDTA.

Notes: Several precautions should be taken to avoid RNase. All solutions should be autoclaved and kept sterile. Glassware should be thoroughly washed and baked at 220°C overnight before each use. Gloves should be worn at all times.

The resulting total cellular RNA is highly intact as judged by Northern blots probed with *Sh1* and *Ss2* clones. PolyA-RNA prepared from this material by oligo-dT cellulose chromatography produced abundant high molecular weight translation products (>90 kD) in vitro in a rabbit reticulocyte lysate system. The yield from 20 g of kernel tissue ranged from 10-12 mg of RNA. The A_{260}/A_{280} of the RNA ranged from 2.0-2.2.

A modification of this procedure has been adapted to small sample sizes (less than 1 g) and used with other maize tissues. Frozen powdered tissue is added directly to a centrifuge tube and suspended in approximately 3 volumes of extraction buffer by vortexing. This suspension is immediately extracted with phenol/chloroform without prior centrifugation. The remainder of the procedure is as described above. Multiple samples are readily processed simultaneously with this protocol. We have obtained good results using this method with root and shoot tissues from seedlings.

We have not rigorously compared this procedure to others that are in use, however, the RNA we obtain compares very favorably in quality with that described in the literature. We have found this protocol to be straight-forward and effective with a wide range of sample sizes.

D.R. McCarty

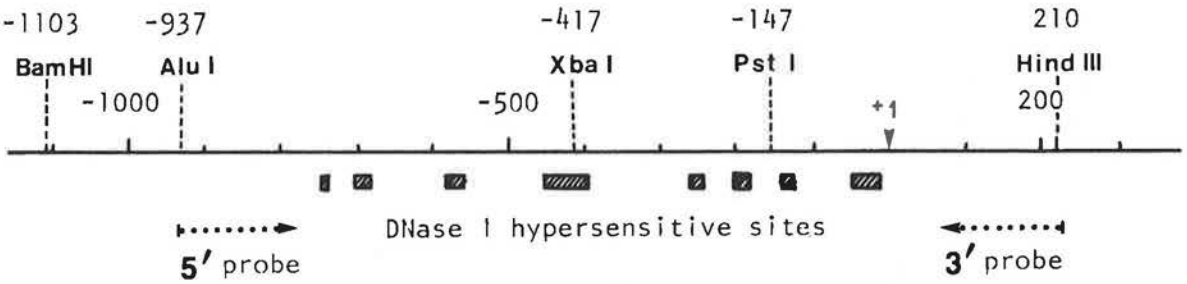
The maize *Adh1* promoter: chromatin structure

We are interested in the chromatin structure of the 5′ region of the *Adh1* gene in corn. Areas of chromatin which are less condensed in the nucleosome array have been associated with gene regulation. These "open" regions have been characterized by their hypersensitivity to DNase I and are especially prevalent in promoter regions of the animal systems studied thus far (Wu, Nature 309:229, 1984).

We examined the 5′ region of *Adh1* defined by the BamHI site at -1103 and the HindIII site at +210 by in situ digestion of Dekalb XL80 nuclei. Within this region we found seven sites of DNase I hypersensitivity, ranging from a site at -40 to one at -740. These are illustrated in the figure as cross-hatched bars under their respective sites. The width of the bar reflects the actual width of the band generated on the autoradiograph. The width of the band, in turn, reflects both the intensity and distribution of cuts introduced by DNase I. The data are the pooled results of several experiments.

In all cases, these sites were mapped using two distinct probes, one 5′ and one 3′ to the hypersensitive region, in order to confirm the presence and position of the hypersensitive sites (Wu, Nature 286:854, 1980).

These sites were found in all tissues examined (roots, shoots and leaves) regardless of the transcriptional state of the gene. While the *Adh1* promoters



in different tissues showed differences in their general sensitivity to DNase I, a suitable concentration of DNase I was found for each tissue to generate the hypersensitive sites.

Anna-Lisa Paul and Robert J. Ferl

The maize *Adh1* promoter: structures in supercoiled DNA

We have examined the alcohol dehydrogenase-1 promoter for supercoil-induced structural anomalies that would indicate regions of possible interaction with regulatory molecules. We have found and mapped with nucleotide level precision S1 nuclease hypersensitive sites that correspond to similar sites in many animal genes. The first site maps to position -65, the heart of a region of DNA characterized by drastic purine/pyrimidine asymmetry. The top strand of the region from -52 to -72 is almost exclusively pyrimidines (20 of 21 bases). Similar regions of homopurine/homopyrimidine asymmetry have been noted and shown to be sensitive to S1 while in the supercoiled state in many animal promoters, from chicken collagen to rabbit globin to some viral promoters, but have not yet been noted in plant promoters. This type of S1 hypersensitive structure has been implicated in the recognition and binding of regulatory and/or transcriptional proteins. For example, recently purified transcription factors involved in the histone gene system of *Drosophila* footprint to an area of DNA that is S1 hypersensitive in vitro (Parker and Topol, Cell 36:357, 1984).

We have determined that a second S1 hypersensitive "site" maps to the borders of a region of alternating pyrimidines and purines, a region theoretically capable of assuming the Z-DNA conformation. In fact, nucleotide level analysis showed that the region from -316 to -331 has the S1 nuclease "signature" of Z-DNA. We have very recently used chemical probes to further support the fact that this region exists as Z-DNA while in the supercoiled state.

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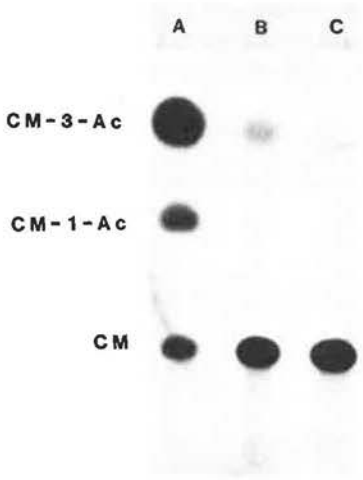
Biological activity of a putative maize *Adh-1S* promoter determined by transient expression in corn protoplasts

Maize alcohol dehydrogenase (*Adh*) genes constitute one of the most well characterized gene systems in plants. The *Adh-1S* allele has been cloned, sequenced and a 1.4 kb (BamHI-HindIII) fragment was identified as the promoter region (Dennis et al., Nucl. Acid. Res. 12:3983, 1984). We demonstrate biological activity of this fragment by a transient transformation assay described below.

A recombinant plasmid pSH 17 was constructed. This was a derivative of pSVcat plasmids used in transient expression studies in mammalian cells (Gorman et al., Molec. Cell. Biol. 2:1044, 1982) and *Drosophila* cell lines (DiNocera and Dawid, P.N.A.S. 80:7095, 1983). The promoter fragment of the *Adh-1S* allele (described above) was placed at the 5′ end of the CAT (Chloramphenicol Acetyl Transferase) coding region. This construct has the globin gene splice signals and polyadenylation sequences from the original pSV cat plasmid at the 3′ end of the CAT coding region. The control plasmid pCM 7 has CAT coding region at the 3′ end of the Tetracycline (*Tc*) resistance gene promoter. Purified plasmid DNA was delivered as a calcium phosphate co-precipitate, as described by Graham and VanderEb (Virology 52:456, 1973), to corn protoplasts and three days later the protoplasts were homogenized and assayed for CAT activity using ^{14}C-chloramphenicol, as described by Shaw and Brodsky (J. Bact. 95:28, 1968). The reaction products of CAT activity (chloramphenicol-1-acetate, 3-acetate and 1,3-diacetate) were resolved by thin layer chromatography and visualised by autoradiography (Figure 1).

The pSH 17 treated corn protoplasts show CAT activity, while protoplasts treated with pCM 7 did not. Interestingly, pSH 17 produced very little CAT in *E. coli* (basal level), while pCM 7 produced a significant amount of CAT (data not shown). These results indicate that the 1.4 kb fragment contains a biologically active promoter sequence, however it is not known if the total sequence is required for expression.



A. pSH 17 TREATED.
B. pCM 7 TREATED.
C. PROTOPLASTS WITHOUT DNA TREATMENT.

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A five-base-pair insertion is associated with mutation to male fertility and toxin insensitivity in T-cytoplasm maize

An important approach in identifying the molecular basis of cytoplasmic male sterility and disease toxin sensitivity in T-cytoplasm maize was obtained by the regeneration of plants from tissue culture. These plants displayed mutation to male fertility and insensitivity to two pathotoxins, *Helminthosporium maydis* race T toxin and *Phyllosticta maydis* toxin. These traits were shown to be maternally inherited; analysis of chloroplast and mitochondrial DNA restriction patterns revealed variation in mitochondrial DNA with no evidence of change in chloroplast DNA (Gengenbach et al., Theor. Appl. Genet. 59:161, 1981; Brettell et al., MNL 56:13, 1982; Gengenbach and Umbeck, MNL 56:140, 1982; Gengenbach, Pring, and Conde, unpublished results). Most of these lines (e.g., T-7 or R-5) carry an altered 6.7 kb *Xho*I restriction fragment. Homology to the 6.7 kb *Xho*I fragment appears at 6.5 kb in these lines. One mutant, T-4, retains the 6.7 kb *Xho*I fragment, but is distinguishable from parental T mtDNA (A188). In last year's newsletter, we reported that there was a ca. 180 bp *Alu*I fragment in T internal to the 6.7 kb *Xho*I fragment that was altered in the T-4 mutant. When these fragments were cloned and sequenced, it was found that the alteration was due to a G to A transition followed by a 5 bp insertion. The insertion generates a 5bp-tandem duplication. At present there do not appear to be any other changes.

We have probed *Bam*HI, *Xho*I, and *Hin*dIII digested total mitochondrial DNA from A188(T), T-4, T-7, R-5, and A188(N) (normal, male-fertile cytoplasm) with 16 *Alu*I clones derived from the 6.7 kb *Xho*I fragment, in addition to clones flanking this region. These analyses showed that T and T-4 are identical in their major and minor hybridization patterns, whereas T-7 and R-5 have undergone a 3 kb deletion generating an N-like genome structure in this region. This deletion extends into a 4.3 kb repeat, through which recombination occurs in T but not N cytoplasm mitochondrial DNA (Lonsdale et al., NAR 12:9249, 1984; Wise et al., MNL 59:50; Wise and Pring, unpublished results). Wf9(T) *Rf1 Rf2* mtDNA appears identical to T and T-4 in this region.

The insertion in T-4 occurs in sequences that are deleted in T-7 and R-5 and are absent in N mtDNA. In addition, the insertion appears to be centrally located in a 345 bp open reading frame (Dewey and Levings, personal communication). The sequences flanking the insertion carry 86 bp perfect homology to the 3′ flanking region of 26S ribosomal DNA. Farther downstream of the insertion is an exact 47 bp homology with the 26S ribosomal DNA coding region. This is not the 26S rDNA region, however. The insertion in T-4 results in sequences which more closely match the 3′ flank of the 26S rDNA (Figure 1).

3′ flank, 26S rDNA:
GCATGAGCTATCCTTCTCATCTCATGGTTGAGGGGGGTT
T-4:
GCATGAGCTATCCTTCTCATCTCATGGTTGAGGGGGGTT
T:
GCATGAGCTATCCTTCTCG---------TGGTTGAGGGGGGTT

Figure 1. The nucleotide sequence of a transcribed region of mitochondrial DNA from T-cytoplasm maize. The T-4 mutant, male fertile and disease resistant, has a G to A transition and a 5 bp insertion, resulting in a sequence which matches a portion of the 3′ flank of 266 ribosomal DNA. Underlined sequence is a 5 bp tandem duplication.

To check for differences in transcription of RNA's, [32P] 5′ end labeled mtRNA from T and T-4 was used to probe the 16 *Alu*I clones derived from the 6.7 kb *Xho*I fragment and flanking regions. No differences were seen between T and T-4. In addition, no apparent differences were seen when [32P] mtRNA's from T and T-4 were used to probe *Bam*HI, *Xho*I, and *Hin*dIII restriction digests of T and T-4 total mitochondrial DNA, suggesting that there are no other obvious changes in expression or rearrangement of coding sequences between T and T-4. However, when [32P] mtRNA from T-7 or N is used to probe the 16 *Alu*I clones, there seems to be a suppression of RNA's that hybridize to the putative open reading frame and 3 kb on either side. This includes a 663 bp open reading frame 3′ to the 345 bp ORF (Dewey and

Levings, personal communication). Sequences 3′ to the 345 bp ORF are not deleted in T-7 or R-5 and are also present in N.

This region displays a complex transcriptional pattern. The T *Alu*I clone which carries the insertion in T-4 hybridizes to six transcripts of ca. 3.5, 2, 1.7, 1.5, 1.0, and 0.8 kb in northern analysis of T and T-4 mtRNA. However, the clone only hybridizes to three transcripts in T-7 or N; the 1.7, weakly to the 1.5, and to a 3.1 kb transcript. The suppression of mtRNA's in T-7 and N which hybridize to sequences in this region can be explained by the deletion of some of the sequences which code for the large transcripts encompassing this region.

There are four basic conclusions we can derive from these data:

1. There are two kinds of mitochondrial DNA rearrangements in the tissue culture derived mutants to male fertility and toxin insensitivity. The rearrangement in the T-4 mutant is due to a G to A transition followed by a 5 bp insertion, while T-7 and R-5 have a 3 kb deletion encompassing a large portion of the 4.3 kb repeat.
2. These sequences are in a region that is heavily transcribed.
3. Transcription in this region is greatly reduced in T-7 or N.
4. Transcription is unaltered in T-4, suggesting that an aberrant gene product or loss of normal translational activity is associated with this mutant.

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The *dek* mutants - new mutants defective in kernel development

We are continuing the characterization of a large collection of EMS induced defective kernel (*dek*) mutants. All mutants of this type are defective in both their embryo and endosperm development. Most of these mutants are lethal because of inviability when tested as mature kernels, but a few have a mutant seedling phenotype. The pleiotropic nature of the defective kernel mutants is also expressed, in some cases, by an alteration of the carotenoid and anthocyanin synthesis in the kernels. The culturing of immature mutant embryos has revealed a wide range of mutant seedlings in some cases. Our previous note (W.F. Sheridan, M.T. Chang and M.G. Neuffer, MNL 58:98-99) reported on characteristics of 21 *dek* mutants and assigned *dek* numbers to each of them. This list is expanded below to include 30 mutants representing all of the chromosome arms except 7S and 8S. Since our published reports referred to these mutants by their E number, a laboratory designation, this number is included in the Table.

Symbol	E No.	Chrom. arm	Mature kernel pheno.	Lethality or Seedling Phenotype*	Culture Phenotype*	Carot. synth.**	Antho. synth.
dek1	792	1S	clf	L	(not germ)	B	B
dek2	1315A	1L	dsc	L	gr	N	N
dek3	1289	2S	gm	L	w-gs	N	N
dek4	1024A	2L	cp f1	L	gr-nl	N	N
dek5	874A	3S	sh	w-gs	w-gs	N	N
dek6	627D	3L	o sh	L	n	N	N
dek7	211C	4S	sh su	gs	w-gs	N	N
dek8	1156A	4L	sh	?	gr-sml	N	N
dek9	1365	5L	crp	L	(not germ)	dil	dil
dek10	1176A	4L	cp	L	gr-crl-stb	N	N
dek11	788	7L	et	L	w,gr	N	N
dek12	873	9S	cp	L	gr-nl-crl	N	N
dek13	744	9L	de o	L	pg-gs	N	N
dek14	1435	10S	cp	L	yg	N	N
dek15	1427A	10L	cp f1	L	gr	N	N
dek16	1414	2L	f1	L	n	N	N
dek17	330D	3L	cp	L	(not germ)	N	N
dek18	931A	5S	cp	L	gr-nl	N	N
dek19	1296A	6L	cp o	L	gr	N	N
dek20	1392A	8L	cp	L	gr	N	?
dek21	1330	10L	msc	L	w	dil	mosaic
dek22	1113A	1L	cp	L	(not germ)	N	N
dek23	1428	2L	dcr	L	(not germ)	N	N
dek24	1283	3S	cp	L	n	N	N
dek25	1167A	4S	sh	L	n	N	N
dek26	1331	5L	cp	L	n	N	N
dek27	1380A	5L	cp	L	gr	N	?
dek28	1307A	6S	o	?	nc	N	?
dek29	1387A	8L	cp	+	gr-nl	N	N
dek30	1391	9L	f1	L	gr-nl	N	?

* w, white; gs, green striped; gr, green; nl, narrow leaf; n, normal; sml, small; crl, curled; stb, stubby; pg, pale green; yg, yellow green; nc, not cultured.

** B, blocked; N, normal; dil, dilute; ?, undetermined

Efforts are underway for mapping these mutants. The mutants have been transferred from the original genetic stocks in which they were isolated to a large-embryo strain (Alexander's high oil), and some of them have been transferred to Black Mexican sweet corn, as well as to an early maturing genetic stock. We will be pleased to share these mutants with other investigators.

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Somatic embryogenesis in glume callus cultures

The phenomenon of somatic embryogenesis in maize has been studied by Green (Proc. Fifth Int. Cong. Plant Tissue Cell Culture, Japan, pp. 107-108, 1982) and Lu et al. (Theor. Appl. Genet. 62:109-112, 1982) using immature embryos as the explant. We have reported callus induction and plantlet regeneration from immature glume explants (MGNL 57:51-52, 1983). These friable cultures, upon closer examination, revealed the presence of somatic embryos. The present study deals with the characterization of glume cultures exhibiting somatic embryogenesis. Glumes of sweet corn and seed corn were cultured on MS medium with 2 mg/l 2,4-D. After callus induction, callus was transferred and maintained on MS medium with 1.0 mg/l 2,4-D for subsequent studies.

Factors affecting somatic embryogenesis (such as sucrose, auxins, 2,4-D analogues and nitrates in MS medium) were tested. In order to find the optimal sucrose concentration for callus and embryoid production, five concentrations of sucrose (2, 3, 6, 9 and 12%) were used; 2-3% sucrose was found to be optimal, whereas 6 and 9% decreased the embryogenic potential and higher concentrations (9 and 12%) inhibited callus growth. Among the auxins, 2,4-D was tested in concentrations ranging from 0.5-4.0 mg/l, of which 2 mg/l was effective. Of the two analogues of 2,4-D used, 2,4,5 trichlorophenoxy propionic acid and 2,4,5 trichloro phenoxy acetic acid, 2,4-D was superior both in callus induction and embryogenesis. Effect of nitrates was also tested; increased levels of NH_4NO_3 (25-75%), compared to the normal levels in MS medium, yielded a high frequency of embryogenic calli and embryoids.

Anatomical observations of isolated embryoids revealed typical scutellum, shoot meristem and coleoptile structures, similar to zygotic embryos. Different developmental stages, from the early globular stage to clearly differentiated somatic embryos, were observed.

Preliminary studies on certain isozymes, peroxidase, esterase, malate dehydrogenase (MDH), polyphenol oxidase (PPO) and IAA oxidase, indicate clear differences in their activity and/or pattern between embryogenic and non-embryogenic calli. Quantitative studies of peroxidase, PPO and IAA oxidase exhibited more activity in embryogenic calli than in non-embryogenic calli under similar nutrient and hormonal conditions. Qualitative studies of peroxidase, esterase and MDH isozymes exhibited specific banding patterns. Embryogenic calli showed two additional slow migrating peroxidase isozymes at Rf 0.11 and 0.14, which were absent in non-embryogenic calli. Esterase isozymes showed two specific isozymes at Rf 0.51 and 0.96 in embryogenic calli and at Rf 0.34 and 0.43 in non-embryogenic calli, while MDH isozymes at Rf 0.68 were present only in non-embyogenic calli.

K.V. Rao, P. Suprasanna and G.M. Reddy

Isozyme studies during differentiation in callus cultures

Plant cell and tissue culture techniques are being increasingly exploited in biochemical studies since they are easy to culture and can be studied under defined conditions. Isozyme analysis at different stages of cultures might throw light on the physiological, biochemical and genetical changes during differentiation.

An attempt was made in the present study to establish a correlation between morphogenic response and isozyme expression, which could be used as a marker system to differentiate the organogenetic potential of callus cultures. Peroxidase, esterase and malate dehydrogenese (MDH) isozyme pattern were studied at four stages, viz. explant, callus, root formation, and shoot formation of cultures, derived from seedling root, immature embryo and immature glume of a local variety of sweet corn (obtained from Maize Research Station, Amberpet, Hyderabad).

In general, during shoot formation all three explant cultures exhibited more peroxidases (PER), esterases (EST) and MDH isozymes than in the callus and root formation stages. Similarity index (SI) was made based on the analysis of isozymes present or absent in the four stages of the three explant cultures (Table 1).

The SI differences for the three enzymes in callus cultures varied from 0.222 to 0.636. The lowest SI was observed in MDH isozyme patterns for seedling root callus, during the shoot formation stage (greening), whereas the highest (0.636) was observed for the glumes during the shoot regeneration stage. There was a high similarity index for peroxidase and esterase for shoot formation in the shoot forming stage of the seedling root, whereas low levels were observed in the stages of shoot formation of embryo and glume. This variation in similarity index clearly indicates strong differences of isozyme patterns of peroxidase, esterase and MDH during differentiation.

Table 1: Similarity indices of the three enzymes studied at four stages of callus cultures

Isozyme		Seedling root			Embryo			Glume		
		Callus	Root	Shoot	Callus	Root	Shoot	Callus	Root	Shoot
PER	Explant	0.546	0.455	0.546	0.455	0.455	0.455	0.417	0.250	0.333
	Callus		0.273	0.546		0.364	0.273		0.333	0.333
	Root			0.546			0.455			0.500
EST	Explant	0.333	0.333	0.533	0.231	0.615	0.385	0.500	0.571	0.428
	Callus		0.267	0.533		0.461	0.307		0.50	0.357
	Root			0.400			0.539			0.500
MDH	Explant	0.333	0.555	0.333	0.455	0.273	0.546	0.273	0.273	0.273
	Callus		0.333	0.222		0.364	0.364		0.364	0.364
	Root			0.333			0.364			0.636

K.V. Rao, P. Suprasanna and G.M. Reddy

Anthocyanin accumulation and phenylalanine ammonia lyase (PAL) activity in callus cultures

In continuation of our earlier studies (MGNL, 1984) with anthocyanin pigment synthesis in tissue cultures of homozygous *Pr* and *pr* genotypes, the present study was undertaken to see the effect of different hormones on anthocyanin synthesis both in callus cultures and in endosperm grown in vitro, and phenylalanine ammonia lyase (PAL) activity in callus and suspension cultures.

Fresh 10 day old immature endosperms were collected from field grown plants and inoculated onto medium. Callus cultures of seedling root and node were also established from one week old germinated kernels. All the cultures were maintained on MS medium containing different concentrations (0.5-4 mg/l) and combinations of various hormones such as IAA, NAA, 2,4-D and BAP and kinetin. Anthocyanins from the tissues were extracted in methanolic HCl (0.1%), and the content is expressed in OD units at A530. Results are presented in the Table. In the case of endosperms 1 mg/l IAA + 1 mg/l KN containing MS media enhanced the pigment production about five times (0.661) over the control (0.126), whereas callus cultures from seedling root accumulated more pigment (0.074) on 2 mg/l 2,4-D MS medium. Of the different auxins and cytokinins tested, for fresh endosperms low levels of IAA (0.5 mg/l) and 1.0 mg/l KN resulted in accumulation of more pigment (i.e., 1.585 and 2.584, respectively) than in callus cultures of the same. Earlier studies in *Daucus carota* (Alfermann and Rienhard, Experentia, 1971) have indicated that the auxin may participate in the induction of necessary enzymes, and in turn in the synthesis of anthocyanins; however, the involvement of phytochrome was not ruled out.

Effect of hormones on synthesis of cyanidin 3-glucoside (cy-3-gluc)

Hormones and Concentration (mg/l)	(Cy-3-gluc) (A 530)	Hormones and concentration (mg/l)	Cy-3-gluc (A 530)
MS Basal	0.525	MS Basal	0.126
MS + 0.25 2,4-D	0.681	MS + 0.25 2,4-D + 0.25 KN	0.079
MS + 0.50 2,4-D	0.759	MS + 0.25 2,4-D + 0.50 KN	0.146
MS + 1.0 2,4-D	1.205	MS + 1.0 2,4-D + 1.0 KN	0.448
MS + 2.0 2,4-D	0.636	MS + 2.0 2,4-D + 2.0 KN	0.125
MS + 0.25 NAA	0.540	MS + 0.25 2,4- D + 0.25 BAP	0.115
MS + 0.50 NAA	1.205	MS + 0.50 2,4-D + 0.50 BAP	0.527
MS + 1.0 NAA	1.152	MS + 1.0 2,4-D + 1.0 BAP	0.125
MS + 2.0 NAA	0.622	MS + 2.0 2,4-D + 2.0 BAP	0.293
MS + 0.25 IAA	0.780	MS + 0.25 NAA + 0.25 KN	0.172
MS + 0.50 IAA	1.585	MS + 0.50 NAA + 0.50 KN	0.186
MS + 1.0 IAA	1.252	MS + 1.0 NAA + 1.0 KN	0.182
MS + 2.0 IAA	0.738	MS + 2.0 NAA + 2.0 KN	0.143
MS + 0.25 KN	1.030	MS + 0.25 NAA + 0.25 BAP	0.214
MS + 0.50 KN	1.057	MS + 0.50 KN + 0.50 BAP	0.223
MS + 1.0 KN	2.534	MS + 1.0 NAA + 1.0 BAP	0.651
MS + 2.0 KN	0.728	MS + 2.0 KN + 2.0 BAP	0.510
MS + 0.25 BAP	1.351	MS + 0.25 IAA + 0.25 KN	0.421
MS + 0.50 BAP	0.647	MS + 0.50 IAA + 0.50 KN	0.165
MS + 1.0 BAP	0.626	MS + 1.0 IAA + 1.0 KN	0.667
MS + 2.0 BAP	1.180	MS + 2.0 IAA + 2.0 KN	0.256
		MS + 0.25 IAA + 0.25 BAP	0.527
		MS + 0.50 IAA + 0.50 BAP	0.150
		MS + 1.0 IAA + 1.0 BAP	0.241
		MS + 2.0 IAA + 2.0 BAP	0.231

PAL activity was studied in callus and suspension cultures, and also at the root forming stage. During callus formation, activity was greater (4.04 units) than during cell suspension (0.07) and the root forming stage (0.4) of calli. There was no appreciable synthesis of anthocyanins in the callus stage where the PAL activity was high. During pigment synthesis, calli cultured on MS + 2 mg/l 2,4-D exhibited 0.47 units of enzyme activity. Further studies on PAL activity in different mutants of anthocyanin may reveal the role of PAL in biosynthesis of anthocyanin.

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Isolation of protoplasts from seedlings

For efficient genetic manipulation, the isolation and culture of protoplasts and plant regeneration are important. Our objective in the protoplast study was to use complementation experiments with mutants blocked at specific steps in anthocyanin biosynthesis. Initial experiments were conducted to see the efficiency of protoplast isolation from different explants (leaves, stem sections, root sections, suspensions and callus cultures). Leaves and stems collected from 7-8 day germinated seedlings of A188 under normal conditions of light at 25 ± 1 C were sliced into thin sections prior to enzyme treatment (1 gm tissue/10 ml mixture) with cellulase (0.2%), macerozyme (0.1%), pectinase (0.1%) and mannitol (0.3M). The incubation time varied for leaf and stem sections. Within 2-3 hrs of incubation, stem protoplasts of various sizes were observed, whereas leaf mesophyll protoplasts were observed after 5-6 hrs. Best yields of protoplasts ($1x10^6$/gm tissue) were obtained from stem sections compared to leaf sections. After the incubation, the suspension was filtered through cheese cloth to separate larger pieces of leaf and debris, followed by centrifugation at 200 g for 5-10 min to sediment protoplasts. The sediment was later rinsed in washing solution (medium containing 0.3M mannitol). The protoplast sediments were suspended in 25% sucrose solution and centrifuged at 100g for 5-10 min. Intact protoplasts, which float on the upper layers, were collected and plated at required density onto P2 culture medium (Potrykus et al., TAG, 347-350, 1977). The optimization of culture conditions for callus initiation is in progress.

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Characterization of accumulated compounds in double recessive *c2 pr*

Many genes controlling anthocyanin synthesis have been described in maize. Some of the genes control the synthesis and modifications of the C15 skeleton, whereas other genes act early in the pathway for anthocyanin biosynthesis (Reddy & Coe, 1962). The characterization of accumulated gene products in single and double recessives reveals the nature of gene controlled mechanisms. Earlier we reported (MGNL, 1985) the characterization of accumulated compounds in the aleurone tissue of *C-I* and *bz1 pr*, and the present report deals with chemical characterization studies of compounds in the double recessive, *c2 pr*. Based on chromatographic and spectroscopic data and chemical tests, the aleurone extract of *c2 pr* has shown the presence of four phenolic compounds. One of them, a major compound, was isolated on column chromatography silicagel-G ACME's particle size max. 75 μ and was characterized as p-coumaric acid by Co-TLC, Co-paper chromatography, physical and chemical methods. The solvent systems used in the study were (1) n-butanol (27%):aqueous acetic acid (1:1 v/v); (2) m-cresol:acetic acid:water (50:2:48 v/v); (3) phenol:water (73:27 w/w); (4) distilled water; and (5) acetic acid:conc. HCl:water (30:3:10). Average Rf values and UV absorption maxima are given below.

The Rf values and absorption maxima of double recessive c2 pr

Compound	n-BuoH HOAc H_2O	m-cresol HOAc H_2O	Phenol H_2O	H_2O	Forestal	λ max/nm
p-coumaric acid (Authentic)	0.93	0.82	0.68	0.88	0.84	222,290-310
Isolated compound	0.93	0.82	0.67	0.88	0.84	222,285-310

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A meiotic function for the B chromosome nondisjunctional system

The B^9 chromosome of TB-9Sb undergoes a significant amount of meiotic loss which can be detected genetically (Robertson, Genetics, 1967). Recently, the rate of meiotic loss for the standard B^9 was shown to be in the range of 12-16% of divisions. A much higher rate of loss (45-50%) was found with a modified B^9 chromosome (Carlson, *Critical Reviews in Plant Science*, in press). The modified B^9 lacks most of the centromeric heterochromatin normally found on B-type chromosomes. Consequently, it lacks the B nondisjunctional system which functions at the second pollen mitosis. Cytological data reported here suggest that the nondisjunctional system also operates at meiosis to prevent meiotic loss.

A comparison was made between the meiotic behavior of the standard B^9 and the modified B^9. Populations of siblings were constructed which contained two types of hemizygous plants: 9^B 9^B standard B^9 and 9^B 9^B modified B^9 (See section on experimental design). In these plants the B^9 is always unpaired, and its ability to migrate as a univalent to one pole can be determined. Sporocyte samples were taken from the plants and anaphase I surveyed. Slides were prepared and classified until a minimum of 50 cells had been examined. Results are given below:

Plant Number	No Lagging	B^9 Lagging	B^9 at pole
Standard B^9			
6577-12	18/54 (33%)	22/54 (41%)	14/54 (26%)
6577-16	15/61 (25%)	25/61 (41%)	21/61 (34%)
6577-31	17/53 (32%)	9/53 (17%)	27/53 (51%)
6578-12	7/61 (12%)	30/61 (49%)	24/61 (39%)
6579- 3	27/68 (40%)	17/68 (25%)	24/66 (35%)
6579- 9	13/60 (22%)	27/60 (45%)	20/60 (33%)
	97/357 (27.2%)	130/357 (36.4%)	130/357 (36.4%)
Modified B^9			
6577- 1	12/59 (20%)	45/59 (77%)	2/59 (3%)
6577-19	18/63 (29%)	41/63 (65%)	4/63 (6%)
6577-22	18/63 (29%)	42/63 (67%)	3/63 (5%)
6577-35	5/51 (10%)	42/51 (82%)	4/51 (8%)
6578- 3	19/55 (35%)	35/55 (64%)	1/55 (2%)
6578-16	11/51 (22%)	29/51 (57%)	11/51 (22%)
6578-21	13/53 (25%)	36/53 (68%)	4/53 (8%)
6578-23	15/58 (26%)	38/58 (65%)	5/58 (9%)
6579- 2	18/68 (27%)	35/68 (51%)	15/68 (22%)
	129/521 (24.8%)	343/521 (65.8%)	49/521 (9.4%)

The data show a high rate of lagging for the modified B^9 and a lower rate for the standard B^9. In addition, the standard B^9 gives a high frequency of early migration to one pole, whereas the modified B^9 does not. There is no overlap in the data and the average differences are sizeable.

The results can be explained in terms of the B-nondisjunctional system. The situation in meiosis for an unpaired B^9 is similar to the condition of a B^9 chromosome undergoing nondisjunction at the second pollen mitosis. In both cases, the centromere is fixed and resistant to division. The chromosome must migrate to one pole without the assistance of disjunction from a pairing partner. It may be that the nondisjunctional system operates in meiosis as well as the second pollen mitosis. It may control the ability of a standard B^9 to migrate early to one pole in anaphase I. The modified B^9, lacking nondisjunction, frequently lags in anaphase due to its inability to migrate to one pole. If the explanation is correct, a new function can be assigned to the B-nondisjunctional system. It may operate to rescue unpaired B^9's in meiosis from lagging and exclusion from daughter nuclei. Considering the variable numbers of B chromosomes present in a population of plants, the rescue of unpaired B's from meiotic loss may be a very important function of the system. (The findings also suggest that the process of nondisjunction may be unusual; it may involve precocious migration of B-type chromosomes rather than lagging and late migration).

The interpretation given above must be qualified, since the results are not entirely straightforward.

One problem with the data is that cells which show no lagging of the B^9 are difficult to interpret. In these cells, the B^9 may be migrating to one pole at the same speed as the other chromosomes. Or the univalent may have split and the chromatids are migrating to opposite poles. Or, the B^9 may be lagging, but not in the center of the cell. A second and more important problem is the difficulty of associating the chromosomal behavior in anaphase with meiotic loss. The two groups of hemizygous plants described earlier were classified for B^9 lagging at telophase I. By this time, lagging was considerably reduced from that found at anaphase I, and the distinction between standard B^9 and modified B^9 was less clear. Lagging by the standard B^9 class averaged 18.2% (80/439). The modified B^9 group averaged 23.5% (131/558). Actual B^9 loss through lagging and exclusion from daughter nuclei was probably even lower than the data suggest, due to some delayed migration of laggards.

The problem of relating meiotic behavior to loss of the B^9 may depend on male-female differences in meiosis. The phenomenon of meiotic loss was detected and measured in crosses involving female transmission of the B^9. Studies reported here are on microsporogenesis. Precise measurements of meiotic loss during male transmission have not been made, due to the problem of pollen competition causing reduced survival of one meiotic product (9 B^9 microspore). However, male transmission of the balanced translocation (9^B B^9 microspore) occurs at similar rates for the standard B^9 and modified B^9 (Carlson, in Maize Breeding and Genetics, 1978), suggesting little difference in meiotic loss. The cytological and genetic data, therefore, indicate low rates of meiotic loss for the modified B^9 during male transmission. Nevertheless, the cytological data may be useful in understanding meiotic loss during female transmission. Perhaps lagging in anaphase I occurs at similar rates during mega- and microsporogenesis. However, laggards are usually excluded from daughter nuclei in megasporogenesis, unlike microsporogenesis. If true, the B-nondisjunctional system has its primary effect in blocking meiotic loss during female transmission of B chromosomes.

Experimental Design: Methods used for constructing the hemizygous plants are given here. The standard B^9 and modified B^9 were combined in crosses of $9(wx)$ $9^B(Wx)$ $B^9(C)$ $B^9(C)$ female X 9^B (Wx) $9^B(Wx)$ modified $B^9(C)$ modified $B^9(C)$ male. Progeny with the *Wx Wx* genotype were selected by pollen classification. These contain $9^B(Wx)$ $9^B(Wx)$ $B^9(C)$ modified $B^9(C)$. (Since the modified B^9 does not undergo nondisjunction, there is no variability of B^9 number in the plants). The selected plants were crossed as female to a homozygous stock of TB-9Sb carrying the *C-I* marker. Among the progeny, kernels with the white endosperm (*C-I*) phenotype were selected and grown in the field. Sporocyte samples were taken and the plants were later classified for pollen viability. Plants with 50% aborted pollen were selected. These should be hemizygotes lacking $B^9(C\text{-}I)$ from the male parent. They fall into two classes with either the constitution $9^B(Wx)$ $9^B(Wx)$ $B^9(C)$ or $9^B(Wx)$ $9^B(Wx)$ modified $B^9(C)$. Plants were assigned to one of the classes by crossing the hemizygotes as male parents to a tester of nondisjunction (*bz bz*). Nondisjunction is found only in crosses involving the standard B^9. (The cross also confirmed absence of the *C-I*-containing B^9.) The experiment was designed to segregate the standard and modified B^9's in a single cross for a controlled comparison. However, it was necessary to select plants from three different ears to produce a reasonable sample of individuals (families 6577, 6578, 6579).

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A further test of homology between the B chromosome and Abnormal 10

A common origin for the B chromosome and the extra chromatin of Abnormal 10 was once proposed. Subsequent studies, however, generally did not support the hypothesis (Reviewed in Ann. Rev. Gen., p. 15, 1978). In one experiment, the ability of Abnormal 10 to carry out a B chromosome function was tested. The B^9 chromosome of TB-9Sb was isolated as a supernumerary in 9 9 B^9 plants. In this situation, the B^9 cannot normally undergo nondisjunction due to absence of a required B chromosome region carried on 9^B. The missing chromatin is the distal tip of the B and is referred to as region 1 (Carlson and Chou, Genetics, 1981). Plants containing 9 9 B^9 plus Abnormal 10 were tested for B^9 nondisjunction. Results were negative. It was concluded that Abnormal 10 lacks the nondisjunctional function found in region 1 of the B and, to that extent, lacks homology with the B chromosome.

Subsequently, several modified (deletion) derivatives of TB-9Sb have been recovered, allowing further tests of homology between the B and Abnormal 10 (Carlson, Maize Breeding and Genetics, 1978). For example, one modified TB-9Sb (#1866) has a B^9 which is missing most of region 2 (proximal euchromatin) and consequently lacks a function that is essential for nondisjunction. An experiment was designed to test nondisjunction of the deletion B^9 in the presence and absence of Abnormal 10. The *Wx* locus was used to mark TB-9Sb-1866. The *R* locus was used to mark the extra chromatin on Abnormal 10. Fortunately, both linkages of gene to chromosome type are very strong (Robertson, Genetics, 1967; Rhoades, Genetics, 1942). As a result, cytological studies to correct for crossing over were unnecessary.

Plants were constructed with the following constitution: 9(*wx*) 9^{B}(*Wx*) 1866-B^{9}(*Sh1*); N10(*R*) Abn10(*r*). Six plants of this type were crossed as male parents to an *r sh wx* tester. Among the progeny, *Wx* kernels were selected. The *Wx* phenotype results from transmission of pollen which derived from one microspore type: 9^{B}(*Wx*) 1866-B^{9}(*Sh1*). The *Wx* kernels were classified for nondisjunction using the *Sh1* marker. Kernels with the recessive (shrunken) phenotype result from one class of nondisjunction (hypoploid endosperm). The effect of Abnormal 10 on nondisjunction was determined by separating white (*r* - Abnormal 10) and colored (*R* -normal 10) seeds. Results are given below (Two ears per male parent):

Male Parent	R Sh Wx	R sh Wx	r Sh Wx	r sh Wx
6554 - 1	213	0	202	0
6554 - 3	167	1	182	0
6554 - 4	201	0	182	0
6554 - 9	201	0	177	1
6554 - 12	234	1	176	1
6554 - 19	220	2	168	1
	1236	4	1087	3

The number of shrunken kernels is very low in both the white and colored seed classes. The frequency of shrunken kernels is 0.3% for both groups. Obviously, nondisjunction of the 1866 B^{9} did not increase in the presence of Abnormal 10. The rate is negligible for both *R* and *r* kernels. Abnormal 10 does not, therefore, contain the nondisjunctional function present in region 2 of the B chromosome.

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Reversion from *cms-S* to fertility in vitro

Callus cultures were established from immature embryos of inbred W182BN with the S (USDA) type of cytoplasmic male sterility (*cms*) in 1981, 1983, and 1984; plants were regenerated from these cultures (MNL 57:53). At least some plants from 16 of 20 different cultures had fertile tassels. Male-fertility of other plants from these cultures could not be scored accurately because of abnormal plant morphology; however, seeds were obtained from many regenerates after pollination with standard W182BN. Fertile progeny were obtained from 13 of 14 cultures, including ones from different subgroups of *cms-S* (CA, S, Me, D, and L).

Fertile regenerated plants always gave fertile progeny, others produced either all fertile or all sterile ones. Some callus cultures produced only fertile progeny, others initially produced sterile progeny and later fertile ones, and still others produced both fertiles and steriles at the same time. Some fertile plants or progeny were recovered from cultures that were only 3 months old, indicating that at least some types of somaclonal variation in corn can occur rather rapidly.

Several lines of evidence indicate that the observed reversions to fertility are maternally inherited. These include the absence of segregation for fertility even after pollination with standard W182BN, results from reciprocal crosses, results from studies of pollen fertility, and mitochondrial DNA data (see below). Revertant plants did not restore standard *cms-S* lines to fertility, further evidence that they have not acquired nuclear restorer genes. Inheritance studies of the more recently isolated revertants are still in progress, but the results to date are consistent with those from earlier revertants.

Agarose gel electrophoresis of unrestricted mitochondrial DNA (mt DNA) of 37 regenerated plants and/or progeny from the *cms-S* cultures was carried out. All fertile lines lacked the S-1 and S-2 plasmid-like molecules characteristic of sterile *cms-S* lines. Fertility of mature regenerates (or their progeny) could be predicted with 100% accuracy from analysis of leaf samples from small regenerates. These results are consistent with those from studies of the first cytoplasmic *cms-S* revertants found in the field by Laughnan and Laughnan-Gabay; these also lacked free S-1 and S-2, portions of which were integrated into the high MW mtDNA. More recently, Escote et al. have reported that cytoplasmic revertants from inbred Wf9 *retain* free S-1 and S-2 (MGL 59:100). We have therefore initiated cultures from Wf9 with *cms-S* (subgroup D) and have begun regenerating plants. These plants will be checked for fertility and the presence of the free plasmids.

No revertants to fertility were seen among plants regenerated from cultures of W182BN with 2 other types of *cms* (C and T) or among their progeny. These results suggest that the mitochondrial DNA of *cms-S* maize is particularly unstable in vitro. The cytoplasmic revertants recovered from culture may aid in the molecular analysis of *cms-S* by providing large numbers of independent revertants for comparative studies.

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Continued study of a defective WF9 cytoplasm, *"wsp"*

A cytoplasmically inherited syndrome, called *"wsp"*, traces back to a single open pollinated plant of WF9 (Duvick, 1958 and 1961 Coop Newsletters). The stock has been maintained by selfing; via backcrossing its nuclear genotype also has been placed in "normal" WF9 cytoplasm.

Starting with the *wsp* cytoplasm line as original female, the inbred SK2 was crossed and backcrossed repeatedly (through 12 backcrosses) as pollen parent, giving a line - SK2*wsp* - with SK2 nuclear genotype and *wsp* cytoplasm. The phenotype of SK2*wsp* is identical to that of SK2; it shows no *wsp* traits. In fact, during all backcross generations SK2*wsp* plants have never shown any *wsp* traits. (Typical *"wsp"* plants have reduced vigor and a distinctive pale green streaking of the leaves; hence the name *"wsp"*, signifying "weak streaked plant".)

SK2*wsp* and SK2 were each crossed as female to a normal appearing strain of WF9 and the two F1s were (1) selfed, (2) backcrossed as female to WF9, and (3) backcrossed as female to SK2. Both F1s were of normal (non-*wsp*) phenotype. The resulting F2 and backcross progenies were grown out at Johnstoh, Iowa in 1985 and plants were classified for presence or absence of the *wsp* phenotype.

Results are summarized in Table 1. They show that the *wsp* phenotype appeared in WF9 backcrosses and F2 progenies when they were in *wsp* cytoplasm, but not when they were in SK2 cytoplasm (A possible exception is the appearance of 3 putative *wsp* plants in one F2 progeny with SK2 cytoplasm.) The *wsp* phenotype was not seen in any SK2 backcross progenies, in either *wsp* or SK2 cytoplasm.

Table 1. Classification of progenies for presence or absence of wsp phenotype.

	Cyto-	Plants			Ear-Rows		
Pedigree	plasm	Total	wsp1	% wsp	Total	wsp2	% wsp
(SK2wsp x WF9)⊗	wsp	579	36	6	30	17	57
(SK2 x WF9)⊗	SK2	649	3	1	30	3	10
(SK2wsp x WF9)WF9	wsp	172	33	19	9	8	89
(SK2 x WF9)WF9	SK2	162	0	0	9	0	0
(SK2wsp x WF9)SK2	wsp	156	0	0	8	0	0
(SK2 x WF9)SK2	SK2	148	0	0	8	0	0
SK2	SK2	169	0	0	8	0	0
WF9	WF9	118	18	39	10	3	30

1Number of plants with wsp phenotype

2Number of ear-rows with one or more plants of wsp phenotype

These data indicate that nuclear genotypes modify the expression of *wsp*, more or less as nuclear genes modify the expression of cytoplasmic male sterility. The nuclear genotype of SK2 dominantly suppresses expression of the *wsp* phenotype; it acts as a "restorer" line. The nuclear genotype of WF9 facilitates expression of the *wsp* phenotype but is recessive to SK2's nuclear suppressor genotypes; it acts as a "maintainer" line. However, precise genetic mechanisms are not clear in these data. Ear-rows varied widely in expression of *wsp*, for example, even though they should have had essentially identical nuclear gene segregation patterns.

Also, as in cytoplasmic male sterility, the data indicate that *wsp* cytoplasm seems to maintain its genetic identity during many generations of nuclear suppression of expression of the *wsp* phenotype. Its presence can be revealed by substitution of a "maintainer" genotype such as that of WF9.

The WF9 and SK2 plants used for backcrossing were selfed and their progenies were grown out alongside the backcross and F2 progenies. One WF9 family unexpectedly produced a large number of plants with the characteristic *wsp* phenotype. All but one of these *wsp* plants were in 2 of 5 ear-rows that arose from a single ear-row (of normal appearance) in the previous generation.

Backcross progenies made with pollen of the 2 "*wsp*-progeny" WF9 plants had *wsp* phenotypes only when in *wsp* cytoplasm, never in SK2 cytoplasm. They did not differ in this respect from backcross progenies made with pollen of "normal-progeny" WF9 plants; these backcrosses also had *wsp* phenotypes only when in *wsp* cytoplasm, never in SK2 cytoplasm. It appears, therefore, that the *wsp* phenotype of the aberrant ear-rows is not transmitted through the nucleus, at least not as a genetic dominant. Perhaps WF9 has again given rise to a *wsp* cytoplasm.

Open pollinated ears have been saved from all *wsp* plants vigorous enough to make an ear (many plants were barren). Seed from these ears will be planted out in 1986 for observation and controlled pollination. Tests will be set up to determine whether or not their *wsp* syndrome is inherited, if it is cytoplasmic, and if so whether or not it differs from the original *wsp* cytoplasm.

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Induced mutation process as source of new mutations

The efficiency of induced mutagenesis is much higher in self-pollinated crops than in cross-fertilized ones (A. Micke, Genetika, USSR, 2 (1):166-167, 1976). The latter accumulates spontaneous mutations. Therefore, a particular interest of corn breeders to this method could be explained by identical spectra of spontaneous and induced mutations. In addition, because of a too low rate of adaptive valuable induced mutations the great expenditures needed to detect them were not compensated.

H. Stubbe (Genetika, USSR, 11:9-30, 1966) reported the evidence of a lack of new factors in mutation process in self-pollinated crops. We tested it in corn. In 9 inbreds, 1106 visible mutations induced with NMU were found. The average frequency of mutation occurrence in all variants of the test

was more than 100 times higher than the control (μ = 0.389 ± 0.009) and (μ = 0.003 ± 0.016). The mutation frequency was evaluated in 100 oligogenes. All loci varying in their mutability were divided into 5 groups:

1. Highly mutable genotype-independent loci (μ = 0.005 - 0.010): *Su1, Sh2, Wc*;
2. Highly mutable genotype-dependent loci: *Ae* (μ = 0.011) in W64A, *Ys1* (μ = 0.010), *Sh1* (μ = 0.008), *Ts2* (μ = 0.005) in A344;
3. Mutable loci (μ = 0.002 - 0.004): *Su2, Wx, Bt1, Br1, Du, D1, Na1, Sh4, Bt2, O1, O5, Mn1, G1, Cr, Pl1, Ffr1,* F*fr4, Ba1;*
4. Low mutable loci (μ = 0.001):
   a. Well-known loci: *Et, Ts1, Bm3, Ba2, Ra1, Po, ts5, Pi, Ra2, Id, An, Si, Mn2, Ds, Py, Sk, La, Zn, Nl, Fl2, Y, Sl, Ad1, Ffr2, Ffr3, Fsh, Fmd, Fww. Sm;*
   b. New loci: *Sup-W70o2, Meg-Gb334, Tts-Gb834, Air-W23, Sin-A344, Dul-W64A, Tea-W64A, Prf-A344, Prf2, Afd-W23, Pam-A344, Pam2, Dsy-A344, Dsy2, Ms43, Ms28, mei-025-W64A;*
5. Stable loci (μ = 0): *02, 02+, Bm1, Bm2, Bm4, Lg1, Lg2, lg3, cg, tp1, tp2, tu, Br2, Gt, Gs, Pn, Td, Tb, ts6, ts3, Ts4, vg, D2, D3, G2, Rd, Br3, Bv, pt.*

New mutations in corn were induced with NAU at a frequency of 0.005. To identify new mutations, we used only mutants of kernels and plants with a high level of viability. Seven high-lysine corn mutants with floury endosperms were not included in the group (A.S. Mashnenkov, MGCNL, 53:111-112, 1973). They were tested for allelism with a limited number of identified genes. The brief characteristics of all new mutants are given in the Transactions of our Institute (A.S. Mashnenkov Sb. trudow KNIISH, USSR, Krasnodar, 27:127-139, 1984). The information on the meiotic mutations was reported elsewhere (A.S. Mashnenkov, I.N. Golubovskaya, Genetika, USSR, 16 (9):1632-1640, 1980).

Five mutations: *Sup-W70o2, Sin-A344* (short internodes), *tts-Gb834* (a tillering plant with tassel silk development), *meg-Gb834* (miniature endosperm and germ), *prf-A344* (prolific) could be used in corn breeding programs for a qualitative change of a reaction norm. [Ed. note: the symbol for prolific, *prf*, is suggested and used here in place of *pro*, because of prior use of *pro* for proline].

In diallel crosses, some indirect evidence of great importance, of new favourable induced mutations - polygenes controlling a high level of heterosis for kernel yield - was obtained (A.S. Mashnenkov Sb. trudow KNIISH, USSR, Krasnodar, 27:127-139, 1984).

A tendency to an increase in a number of new inbreds with high combining ability resulting from hybrid corn seed treatment with the mutagens was observed. However, we did not manage to evaluate true efficiency of induced mutagenesis as a method of improving corn productivity. The true efficiency of the method could only be evaluated from the number of commercial cultivars released. It could be realized in conjunction with breeders in comparative tests.

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A cloned maize genomic sequence shows homology to meiosis-specific cDNA clones from *Lilium* and to a small heat-shock protein gene of *Glycine max*

Despite the central role of meiosis in the genetics of all higher organisms, relatively little is known about the gene activities which accompany this basic process. For several years I have been working with a cDNA library prepared from *Lilium* microsporocytes, which at present provide the only source of plant material biochemically amenable to this type of recombinant DNA technology (R. Appels, R.A. Bouchard, and H. Stern, Chromosoma 85:591-602, 1982). These studies have shown that a particular set of cDNA clones representing a group of transcripts selected because they were expressed abundantly and specifically during meiotic prophase and showed homology to genomic DNA of wheat, rye, and maize, originate from a complex set of repeated sequences termed the EMPR (Expressed Meiotic Prophase Repeat) sequences. Studies of this set of sequences have now been extended directly to maize. Screening of a maize phage-lambda genomic DNA library, after initial difficulties (see accompanying report), has yielded a number of clones containing genuinely homologous sequences for the EMPR cDNA clones. A plasmid subclone for one of these regions, pZm9′-8, has been extensively characterized, and shows a number of characteristics which suggest that functionally significant aspects of the EMPR sequences are conserved in maize. The portion of its insert showing readily detectable cross homology to lily EMPR clone probes has been found to correspond to the region of these cDNA's which shows the strongest sequence conservation among different repeat subfamilies within the *Lilium* genome itself. Hybridization of probe for this conserved segment of pZm9′-8 to poly(A) RNA from successive stages of lily meiotic tissue shows that it is homologous to transcripts abundant only during meiotic prophase, just as is seen for the lily-derived EMPR cDNA clones. While preparation in quantity of pure meiotic prophase cells is not feasible with maize, it can be shown that transcripts homologous to pZm9′-8 are specifically present in RNA prepared from spikelets containing meiotic prophase microsporocytes, and absent from somatic tissue under standard growth conditions.

Recently, the inferred amino acid sequence of the conserved portions of the lily EMPR subfamilies has been found to be clearly homologous to the sequence predicted for a small heat-shock protein from a cloned soybean gene (Czarnecka et al., PNAS 82:3726, 1985). In light of this observation, I have compared the inferred sequence of a section containing the most conserved portion of one of the lily EMPR clones and the corresponding segment of the published soybean clone sequence with the sequence obtained for an M13 subclone, from pZm9′-8 containing the most strongly cross-reassociating portion. As shown in the accompanying figure, the inferred amino acid sequence for at least this section of the maize clone is clearly homologous both to the lily EMPR and the soybean hsp-gene products. The fact

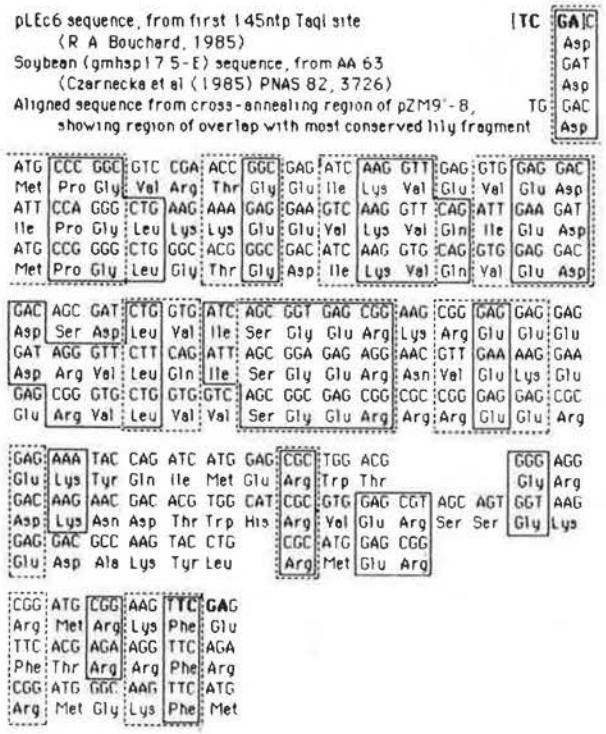

Figure 1. Alignment of inferred amino acid sequences for conserved TaqI fragment of *Lilium* EMPR cDNA clone pLEc6, *Glycine* genomic clone gmhsp 17.5-E, and *Zea* genomic subclone pZm9′-8. Dotted lines indicate amino acid residues homologous between the lily and maize sequences, while solid lines indicate residues common to all three, lily and soybean (upper four of six lines in each row), or soybean and maize (lower four of six lines).

that such substantial homology with both the conserved portion of the EMPR and the soybean clone lies here is particularly significant in light of the fact that this region corresponds to the portion of the small hsp heat shock genes where the strongest homology among different genes both between genomes and within the same genome has been found to lie. It therefore appears at the very least that an important functional domain is shared by small hsp heat shock genes, a large repetitive family naturally expressed during meiosis in lily, and a set of maize genes identified through their homology to the latter. Additional sequencing studies to examine further the nature and extent of this homology over the rest of the pZm9′-8 and other maize EMPR cognates, and other experiments examining whether transcripts complementary to maize EMPR genes are induced in somatic tissue during thermal stress, are underway.

Robert A. Bouchard

"Specious positive" maize genomic clones recovered due to annealing with oligo-dG/oligo-dC tails of cDNA-clone inserts—a cautionary tale

In the course of screening the maize genomic library for the work described above, a troublesome phenomenon was encountered which may be of interest to others attempting to isolate maize genomic cognates using heterologous cDNA clones. Initial screening of the library was performed at low stringency with nick-translated fragments containing entire *Lilium* EMPR cDNA inserts, including the oligo-dG/oligo-dC tails created during the cloning procedure, or nick-translated holo-plasmid DNA. Both approaches resulted in the isolation of a large number of positively-hybridizing phage which after plaque purification showed approximately the same signal strengths under either low (50% Formamide, 5X SSPE, 0.2% SDS-30 C), intermediate or high (same salts, 37 C or 42 C) stringency probe hybridization conditions. Southern blot analysis of restriction enzyme digested phage DNAs showed that this homology could be mapped to specific DNA fragments from the cloned maize DNA inserts of these phage. Upon further examination, however, it was found that all homology to the lily cDNAs vanished when these clones were probed with internal segments from the cDNA inserts, even though these internal segments contained the most conserved portions of the cDNA clones. Screening with these internal segments identified instead a much smaller subset of maize clones showing plaque cross-reaction *only* at lower stringencies, though the signal seen under the 30 C condition was somewhat stronger than that seen for the clones described above. When a Southern blot comparison was performed with equivalent amounts of purified, restricted DNA for such a positive and some of the clones described above at the lower stringency, the homologous maize segment from the second type of positive gave a much stronger signal even with whole insert or holo-plasmid probe, while it was the only fragment which showed a signal when the blot was probed with a fragment from within a lily EMPR insert. These observations suggested that the first type of positive clone was due to annealing of the oligo-dG/oligo-dC tracks of whole cloned cDNA inserts or holo-plasmids to oligonucleotide tracks in the maize genomic clones. This has since been confirmed by probing Southern blots

with end-labelled synthetic oligo-dG, which anneals specifically to the same specious positive bands as the plasmid probe.

These observations indicate that care should be exercised when screening maize genomic libraries with heterologous cDNA clones, due to the existence of genomic sequences with homology to the oligonucleotide tails created during the widely-used procedure for inserting cDNAs in the PstI site of pBR322 and other vectors. The length of oligo-dG/dC necessary to obtain such specious signals is not great: sequencing has shown that my usual lily probe has a tail of only 16 Gs, while the synthetic oligo-dG probe contained oligo-dG pieces from 12 to 18 nucleotides in length. Fidelity of the specious hybridization is quite good; in the present case signals were obtained at stringencies where true positives would not cross-react. It should be noted that the relative intensities of Benton plaque signals can be quite deceptive, particularly at intermediate stringencies where the cross-reaction of real but diverged homologous regions is marginal. For all these reasons, it is advisable to use fragments derived from the internal portion of cloned heterologous cDNA inserts if these are to be used to screen maize genomic clones.

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A restriction site polymorphism in ribosomal cistrons in a maize population selected for yield

The Hays Golden population has undergone mass selection strictly for yield for twenty-nine generations. DNA samples were isolated from seedlings of this selected population and the control population. DNA was digested with restriction enzyme SstI and Southern analyses were performed with the pGMR3 soybean ribosomal DNA clone (obtained from Elizabeth Zimmer), which contains the 3′ end of the 26S gene, the nontranscribed spacer region and the 5′ end of the 18S gene. This probe detects bands of 3.6kb, 1.8kb and a 1.6kb doublet; a size polymorphism is observed for the 3.6kb band, which increases to 3.9kb due to spacer length variation. We have detected a 5.3 kb band. The size of this fragment is consistent with the loss of the SstI restriction site in the external transcribed spacer between the 3.6kb and 1.6kb fragment.

This restriction site polymorphism generating the 5.3kb band was detected in all seedlings randomly sampled from the Hays Golden population that has undergone selection for yield. This specific polymorphism was not detected in seedlings randomly sampled from the control Hays Golden nonselected population. Both the selected and nonselected populations were polymorphic for the 3.6kb and 3.9kb fragments. These results are preliminary since only 11 plants from each of the populations have been assayed at this time. Experiments are in progress to evaluate more plants from generation 29, earlier generations and the control population to identify the origin of this variation and frequency of occurrence.

Torbert R. Rocheford, John C. Osterman
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Chromosomal location of additional genes for resistance to *Corynebacterium (Clavibacter) michiganense* ssp. *nebraskense*

Goss's wilt (Leaf Freckles and Wilt) is a vascular and foliar disease incited by the bacterial pathogen *Corynebacterium (Clavibacter) michiganense* ssp. *nebraskense*. We previously reported use of crosses between M14 (resistant) translocation stocks and inbred A632 (susceptible) to locate a gene for resistance on the long arm of chromosome 7 (MGCNL 59:57, 1985). Similar field tests conducted in 1985 indicate that genes for resistance are also located on the short arm of chromosome 4 and the long arm of chromosome 8 (Table 1). These findings are consistent with a quantitative mode of inheritance for resistance to this disease.

Table 1. 1985 FIELD RESULTS OF M14 TRANSLOCATION STOCKS USED TO LOCATE GENES FOR RESISTANCE TO GOSS' WILT

Translocation/Break Points		Mean Rating Differences
T8-9_{6673}	8L.35-9S.31	1.02*
T4-9e	4S.53-9L.26	1.22*
T7-9a	7L.63-9S.07	0.94*
T5-9_{4817}	5L.06-9S.07	0.14
T2-9_{5257}	2L.28-9L.20	0.34
A632 - M14 Checks		1.69*

* t-tests significant at .10 probability level

Environment appears to influence expression of this disease. The genetic stocks involving translocation T4-9e demonstrated a significant difference in 1985 but did not in 1984. T8-9(6673) was not in 1984 tests. The effect of environment was also demonstrated in greenhouse tests. Genetic stocks involving translocations T4-9e, T7-9a and T8-9(6673), all of which demonstrated differences in the field, were greenhouse tested in fall 1985. Only stocks involving translocation T4-9e demonstrated a significant difference.

Preliminary results from greenhouse studies conducted with A632 (susceptible) translocation stocks crossed with Mo20W (resistant) are supportive of a quantitative mode of inheritance for susceptibility to this disease. These stocks will be field tested before chromosomal locations of genes effecting susceptibility are reported. Of interest is the potential

location of a gene on the short arm of chromosome 4 that causes a susceptible reaction. Many sweet corn and popcorn cultivars have demonstrated susceptibility to this disease. The sugary-1 locus and the gametophyte factor locus, *Ga*, are both found on the short arm of chromosome 4. It may be worthwhile to evaluate for a possible relationship between selection for sugary-1 in sweet corn or selection for gametophyte factor in popcorn and the level of susceptibility to Goss's wilt.

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Developing maize breeding populations with resistance to *Corynebacterium (Clavibacter) michiganense* ssp. *nebraskense*

Increased expression of this disease has been observed with inbreeding in pop and dent maize populations. Reaction to this disease also appears to be quantitatively inherited in maize. A recurrent selection scheme that takes advantage of the enhanced expression of the disease, via rating genotypes more accurately at the inbred family level (S1 or S2), is recommended as a rapid and efficient means of developing maize breeding stocks resistant to *Corynebacterium (Clavibacter) michiganense* ssp. *nebraskense*. This approach is being utilized in the popcorn breeding program and, more recently, combined with full-sib selection in a long term selection study in dent corn.

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Biochemical analysis of the somaclonal variation in maize regenerated plants

In a previous communication (Rapela, MNL 59:59-60, 1985) we reported a partial biochemical (SDS-electrophoresis) somaclonal variation analysis of 3 floury-a and 5 normal red flint (NRF) maize plants regenerated through somatic embryogenesis. Cultures were initiated from immature maize embryos between 1 mm and 2 mm long, placed scutellum up on Yu-Pei (Ku et al., 1978) medium containing 1 mg/l 2,4-D, 120 g/l sucrose and 400 mg/l proline. Embryogenic callus pieces were transferred to modified Murashige-Skoog medium (Green, Hort. Sci. 12:131-134, 1977) in light. Plantlets were transferred to vermiculite and then to soil.

Here we present a more detailed analysis of the biochemical somaclonal variation of these plants in relation to control plants. We have determined the soluble leaf protein pattern and the soluble pollen protein pattern by SDS-electrophoresis in polyacrylamide gels; the endosperm protein pattern according to the procedures of Landry-Moureaux; and the zein polypeptide pattern of the grains derived from selfed regenerated plants.

An analysis based on presence or absence of polypeptides for the electrophoresis and Σ(minor fraction/major fraction)/5 for the protein pattern was used to obtain the Similarity Index (SI) between regenerated plants of each genotype and the control, and among regenerated plants. The SI analysis of floury-a and NRF plants is shown in Figures 1 and 2, respectively.

Floury-a

	LP	PP	EP	ZP	$\bar{X}$
0-1	0,88	1,00	0,90	1,00	0,95
0-2	0,88	0,89	0,78	0,79	0,84
0-3	0,94	1,00	0,90	0,94	0,95
1-2	0,88	0,89	0,80	0,71	0,82
1-3	0,92	1,00	0,88	0,94	0,94
2-3	0,83	0,89	0,84	0,68	0,80
$\bar{X}$	0,87	0,95	0,85	0,84	0,88

Fig.1. Similarity Index of floury-a plants.

LP= Soluble leaf protein Pattern; PP= soluble Pollen protein Pattern; EP= Endosperm protein Pattern; ZP= Zein protein Pattern.

0= control floury-a plant and grain; 1-2 and 3, floury-a regenerated plants.

NRF

	LP	PP	EP	ZP	$\bar{X}$
0-4	0,69	1,00	0,90	1,00	0,90
0-5	0,41	0,86	0,71	0,81	0,70
0-6	0,57	0,95	0,87	0,95	0,84
0-7	0,67	1,00	0,86	0,95	0,87
0-8	0,63	1,00	0,87	0,95	0,86
4-5	0,59	0,86	0,76	0,81	0,76
4-6	0,48	0,95	0,84	0,95	0,81
4-7	0,48	1,00	0,83	0,95	0,82
4-8	0,52	1,00	0,81	0,95	0,82
5-6	0,48	0,82	0,75	0,86	0,73
5-7	0,42	0,86	0,74	0,77	0,70
5-8	0,36	0,86	0,73	0,77	0,68
6-7	0,75	0,95	0,96	0,90	0,89
6-8	0,55	0,95	0,94	0,90	0,84
7-8	0,65	1,00	0,92	0,90	0,87
$\bar{X}$	0,55	0,94	0,83	0,89	0,80

Fig.2. Similarity Index of NRF plants.

0= control NRF plant and grain; 4,5,6,7 and 8, NRF regenerated plants.

It is difficult to make useful morphological evaluations of regenerated maize plants. However, certain biochemical evaluations among plants can be realistically measured by SIs. The occurrence of biochemical somaclonal variation among regenerated plants of each genotype is strongly indicative of considerable changes at the genetic level. The general homo-

geneity of the SIs for each pair of plants and determination support the relevance of such analysis for breeding purposes. For example, the low SI determined for floury-a 2 and NRF 5 plants in relation to the controls and the rest of regenerated plants of each genotype is of particular interest.

Our results to date suggest that biochemical analysis of electrophoresis and protein pattern of regenerated plants and seeds is worthwhile and that somaclonal variants can be quickly isolated by such techniques.

Miguel Angel Rapela

Regeneration of maize plants from long-term embryogenic cultures selected in lysine-threonine medium

In previous works (Rapela, Rev. Fac. Agr. UNLP 56:17-26, 1980; ibid. 56:27-37, 1980; Plant Cell Physiol. 23:285-291, 1982; MNL 59:60-61, 1985), tests were performed on embryo and callus cultures in lysine-threonine medium in order to isolate amino acid overproducer forms of maize. Also, different explant sources, culture media and environmental conditions were tested in order to obtain embryogenic tissues with the ability to regenerate plants after several subcultures. Immature maize embryos 1.5 mm long provide an excellent material to start embryogenic cultures in media with high levels of sucrose and proline (Rapela, J. Plant Physiol. 121:119-122, 1985).

Therefore, in order to use a long-term lysine plus threonine (LT) in vitro selection system with embryogenic cultures, we developed a selection scheme with 10 steps:

1. Embryogenic callus was induced from sterilized immature maize embryos on Yu-Pei (Ku et al., 1978) medium, supplemented with 1.0 mg/l 2,4-D;120g/l sucrose and 400 mg/l proline. Thirty days in darkness at 28 C.
2. First selection cycle: the proliferating embryogenic callus was divided into small pieces (about 50 mg each) and the pieces were transferred to Petri dishes containing the modified Yu-Pei medium (step 1) along with 1 mM LT. Thirty days in darkness at 28 C.
3. Second selection cycle: medium as in step 2 but with 2.5 mM LT. Thirty days in darkness at 28 C.
4. Third selection cycle: medium as in step 3. Thirty days in darkness at 28 C.
5. Live callus showing embryogenic aspect was transferred to the initial medium (step 1) without LT. Forty five days in darkness at 28 C.
6. Fourth selection cycle: medium as in step 3. Thirty days in darkness at 28 C.
7. First regeneration cycle: embryogenic calli were transferred to a regeneration medium for plant regeneration. The culture medium was modified Murashige and Skoog (1962) (Green, Hort. Sci. 12:131-134, 1977) supplemented with 20 g/l sucrose. Sixteen hours light and 8 hours dark cycle. Light intensity was 1000 lux for thirty days at 30/26 C.
8. Second regeneration cycle: medium as in step 7. Light intensity was 3000 lux for fifteen days.
9. Regenerated plants were transferred from Erlenmeyer flasks to plastic pots containing sterile vermiculite watered with ¼ x MS salts, and maintained in a growth chamber with 16 hours light/day at 3000 lux for fifteen days.
10. Well-established plants were moved to soil.

Approximately 200 immature maize embryos were used to start embryogenic cultures. During the selection and non-selection cycles more than 1000 calli were screened for LT resistance and embryogenic aspect.

Seventeen plantlets were regenerated from LT-resistant embryogenic calli. Six plants did not survive transplantation to vermiculite in the growth chamber. Five plants did not survive transplantation to soil in the greenhouse. One plant did not produce either flowers or ear. The remaining five plants, two of floury-a inbred genotype and three of BP normal red flint inbred genotype, each developed a single normal ear, but without flowers. These plants were pollinated in order to recover F_1 progeny for further study. The F_1 seeds were planted in the field during September '85.

Miguel Angel Rapela

Selection of stables or consolidated spontaneous mutants of the *c* locus

In the system (Line X x Line Y) x Line Y (BC_1), all lines homozygous of the genotype *A1 A2 C R Pr* (Materials were indicated in MNL 55:58, 1981), 40 BC_1 progenies (13519 kernels) gave 256 mutants with aleurone colorless. These are only possible by complementary aleurone gene mutation or mutation of *C* to *C-Im* inhibitor of aleurone color. For that reason in selfing plants originating by selection of colorless kernels it is expected to obtain all aleurone colorless, or the ratio 3 colorless: 1 colored. However, 50 progenies did not segregate colorless and 80 progenies segregated different ratios. These unexpected ratios would indicate mutation and back mutation as the following tests show for *C-Im sh/C Sh* x *C Sh/C Sh:*

Phenotype	Obs.	Calc.	Interpretation
C-Im sh	125	135.8	20.7% Mutation *C* to *C-Im*
C-Im Sh	73	4.2	change *C Sh* to *C-Im Sh*
C sh	4	4.2	
C Sh	78	135.8	

For *C-Im Sh/C sh* x *Csh/Csh:*

Phenotype	Obs.	Calc.	Interpretation
C-Im Sh	20	182.3	43% Back mutation
C-Im sh	25	5.6	*C-Im* to *C*
C Sh	169	5.6	*C-Im Sh* to *C Sh*
C sh	162	182.3	5% Mutation *C* to *C-Im*

Crossing (the pollen) of the above BC_1 with *c*-tester, all kernels had aleurone color, indicating that all *C-Im* back mutated to normal *C* gene during ontogeny.

Meanwhile in the selection of colorless aleurone progenies was obtained a line that segregates in a mendelian ratio, 3 colorless: 1 colored, during 3 generations: S_1 195:75; S_2 133:42; S_3 104:35. This mutant is symbolized *C-Im1*. Backcrosses with Line Y (activators) gave a deficiency of colorless kernels in 8 progenies: 2:208; all color; 33:310; all color; 5:345; 25:280; 30:270; 20:260.

In another line selected, another dominant mutant inhibitor of aleurone was obtained, symbolized *C-Im2*, that segregates also in mendelian ratio when selfed *(C-Im Sh/C sh):*

Phenotype	Obs.	Calc.
C-Im2 Sh	124	125.72
C-Im2 sh	5	2.52
C Sh	2	2.52
C sh	40	40.22

In the back-cross with Line Y (activators) in 12 progenies there was not the deficient segregation for colorless as found with *C-Im1:*

Colorless	Colored	χ^2
237	197	3.65
84	94	0.56
165	216	6.83
200	165	3.36
162	169	0.15
100	96	0.08
132	153	1.55
62	73	0.90
110	82	4.08
156	145	0.4
109	108	0.004
254	249	0.05
		21.65
1,771	1,747	0.16
	Heterogeneity	21.49* (P = 0.032)

Is even the gene *C-Im2* unstable? Is *C-Im1* different from *C-Im2* or does the line that carries *C-Im1* also carry a *Ds*-like element, and is *C-Im2* free of *Ds?* The question is one of molecular genetics, but classical genetics will answer it if the gene is of mendelian inheritance and during how many generations, and in what genetic environment it is stable.

The stability of the *C-Im* gene was not instantaneous as a gene mutation by DNA deletion. Perhaps the new mutant *C-Im2*, by means of adequate insertion of nucleotides and protection by histones, becomes stable against the selective pressure of the internal cellular environment (Darwinism in intra-organic molecular level? or DNA and protein metabolism?).

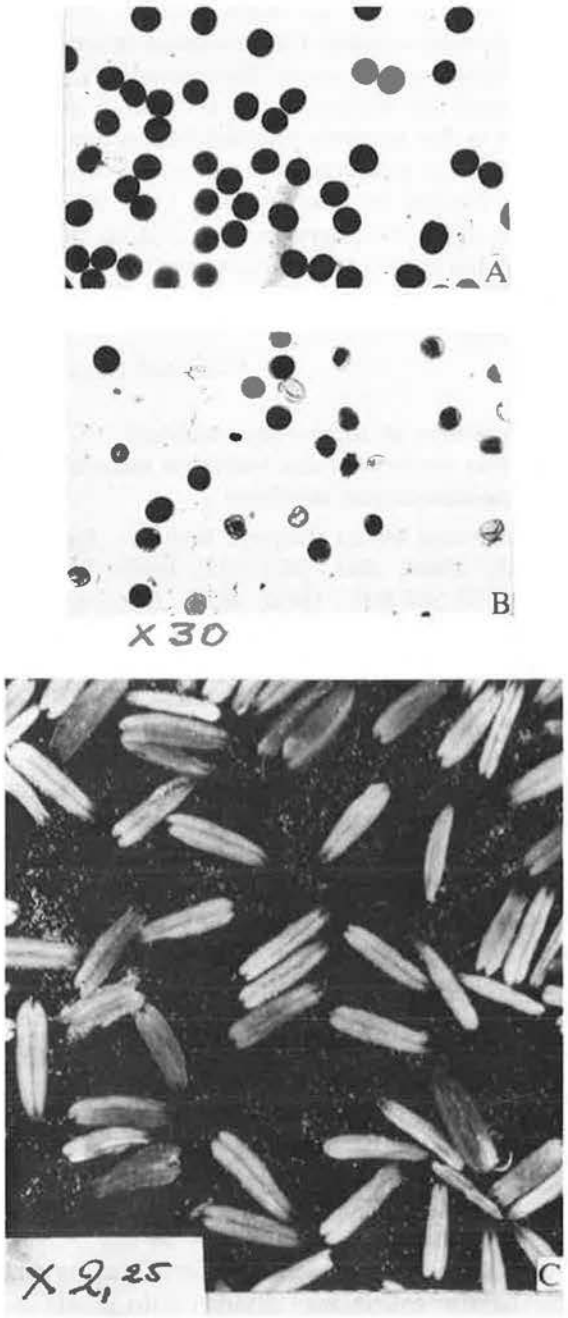

Figure 1. (A) Pollen of red anthers sector; (B) Pollen of yellow anthers sector; (C) Mosaicism in anthers.

In the system indicated above, (X x Y)x Y, the gene *C* also mutates to recessive *c-xm*. The classic *c* of *c*-tester is stable in the system, but *c-xm* originated by *C* mutation to *c-xm*. In mutagenic effect on its locus *c-xm Sh/c sh* x *C sh/C sh* gave 11 ears (2000 kernels) with 95% colorless kernels (bad expression of *sh*). The new line that carried *c-xm* crossed with line *C sh* (Activator Y) would be considered "a new system" of high mutagenic effects, starting from the mentioned unstable system (X x Y) x Y. Perhaps it is possible that by an evolutive process, many

communications of a "new system" of instability have the same origin. For that reason it would be useful to indicate with precision (when it is possible) the ancestral material that was studied, in order to establish that diverse evolutionary lines arise from the same system of instability.

In MNL 40:62-63, 1966, I indicated that "C^{IP} gen was originated in maize by teosinte". Such interpretation was originated because the cytoplasm of teosinte from Huixtla (south of México) is a mutational activator of the X line: "homozygous A_1A_2CRPrBCl, 1877; Dr. Randolph 1933".

The homozygous genotype of the X line in teosinte cytoplasm shows high mosaicism in anther color (Figure 1).

Luis B. Mazoti

Relationships between photosynthesis, canopy traits and yield in flint type maize

A path analysis was carried out on data obtained in 1984 from research aimed to determine the genetical basis of photosynthesis in maize and its relationships to yield and yield components (MNL 58:116-118, 1984). F_1 hybrids of crosses between flint type lines differing in photosynthetic activity were used. The influence of the traits considered on yield was analyzed classifying them as (a) first order factors (ear width, ear length, 100 grain weight, cob weight and plant height) and (b) second order factors (leaf area index and apparent photosynthesis measured during grain filling). Direct and indirect effect estimates according to the path-coefficient technique are shown in Tables 1 and 2. The results indicate that: (1) The first order factors explain 91.1% of yield variations. Among those traits the direct effect of ear width on yield is notorious. Indirect effects of 100 grain weight (+) and ear length (-) through ear width appear to be important; (2) The two second order factors show lower determination coefficients on first order factors and, as should be expected, low correlation coefficients with yield (about 0.15).

Table 1: Direct* and indirect effects of first order factors on yield

Via:	100 grains weight	ear width	ear length	cob weight	plant height	Total r_{ix}
Effect due to:						
100 grains weight	0.272*	0.587	-0.209	0.042	0.088	0.78++
ear width	0.220	0.725*	-0.352	0.040	-0.143	0.49
ear length	-0.120	-0.536	0.476*	0.023	0.217	0.06
cob weight	0.106	0.268	0.105	0.107*	0.074	0.66+
plant height	0.052	-0.225	0.224	0.017	0.462*	0.53

Residual path-coefficient = 0.298 Determ. coefficient $(1-0.298^2)$= 0.911

r_{ix}= phenotypic correlation coeff.; +, ++ significant at the 0.05 and 0.01 level.

Table 2. Effects of apparent photosynthesis on first order factors

	100 grains weight	ear width	ear length	cob weight	plant height
Direct	0.446	0.287	-0.323	-0,532	-0.014
Via LAI	-0.186	-0.257	0.243	0.032	0.314
Total	0.26	0.03	-0.08	-0.50	0.30

B. Effects of leaf area index on first order factors

	100 grains weight	ear width	ear length	cob weight	plant height
Direct	0.453	-0.628	0.592	0.078	0.766
Via AP	0.183	0.118	-0.132	-0.218	-0.006
Total	-0.27	-0.51	0.46	-0.14	0.76++

C. Residual effects

	100 grains weight	ear width	ear length	cob weight	plant height
Residual	0.873	0.819	0.838	0.863	0.649
1 - Res^2	0.238	0.329	0.298	0.255	0.579

Jaime R. Jatimliansky, María I. Urrutia and Miguel J. Arturi

Vivipary in a breeding population

BHDC is a breeding stock developed at this Institute around 1970 by intercrossing 37 commercial double crosses of Argentine maize, followed by several generations of free pollination in isolated blocks. In the 1982 S_1 selection plot, one ear showing typical viviparous grains was observed at harvest. This seems to be closely linked with white endosperm (χ^2 = 138.45***) and white seedling (not yet analyzed), like that described by Mazoti (Rev. Fac. Agr. UNLP 39:63, 1963, and previous papers). Selfed progenies showed low recombination percentages between vp_x and y_x (2 to 6%). Location tests and allelism tests are being carried out.

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Cytological evidences for a basic number x = 5 in *Zea* polyploid complex

In this report the main previous contributions that support the hypothesis concerning the tetraploid nature of *Zea mays* are reviewed. On the other hand, new cytological evidences supporting a basic number x = 5 for the polyploid complex genus *Zea* are discussed.

Darlington (Chromosome Botany, Allen & Unwin, London, 1956) considered the concept of basic number and pointed out that the inference of a basic number in a polyploid series is obviously an important step in fitting chromosome numbers to an evolutionary hypothesis. Sometimes the diploid members of the series have disappeared from the genus. At the same time the internal evidence of polyploidy may have disappeared also. According to Darlington "this is the situation in *Zea*. The ten chromosomes of the haploid set in *Zea mays* show no internal relations, no evidence of an earlier doubling. *Zea mays* is what we call and rightly call for all practical purposes, a diploid species". Nevertheless relatives, both in *Coix* and *Sorghum*, have the haploid number of 5. This

was a strong suggestion, therefore, that 5 is the ancestral basic number (Darlington, ibid.).

Cytological evidences of internal relations among the 10 chromosomes of the haploid set of maize were found by several authors. McClintock (Zeitschr. Zellf. u. mikr. Anat. 19:191-237, 1933) studied prophase pairing in a maize monoploid and reported the occurrence of a high degree of pachytene pairing which looked as intimate as homologous pairing. Chaganti (The Bussey Inst., Harvard Univ., 1965) made a detailed study of various types of chromosome associations in meta-anaphase I in haploid maize and 30% of the cells showed associations implying homology or homoeology. Similar associations have been found recently by Ting (Maydica 30:161-169, 1985) in the first meiotic prophase of haploid maize obtained from anther culture. Vijendra Das (Cytologia 35:259-261, 1970) found secondary association of bivalents in *Zea mays* (2n = 20) and a maximum of five groups of two bivalents were observed at diakinesis. Finally, the 3-D reconstructions study, using electron-micrographs of serial thin sections of somatic metaphase cells, made by Bennett (Kew Chromosome Conference II, Allen & Unwin, London, pp. 71-79, 1983), showed that the 20 chromosomes of *Zea mays* form four subsets of 5 chromosomes rather than two sets of 10. As Bennett (ibid.) pointed out, his model may have predicted intraspecific homoeology, suggesting that *Zea mays* is a tetraploid and indicating which chromosomes are homologues.

New cytological evidence supporting x = 5 has been obtained as a consequence of our analysis of the meiotic configuration of *Zea mays* subsp. *mays*, *Z. diploperennis*, *Z. perennis* and of four F_1 artificial interspecific hybrids. In Figure 1 the most frequent meiotic configuration in each taxon is presented. The majority of the results could be interpreted only assuming x = 5. Hypotheses about the genome constitution of each taxon are proposed.

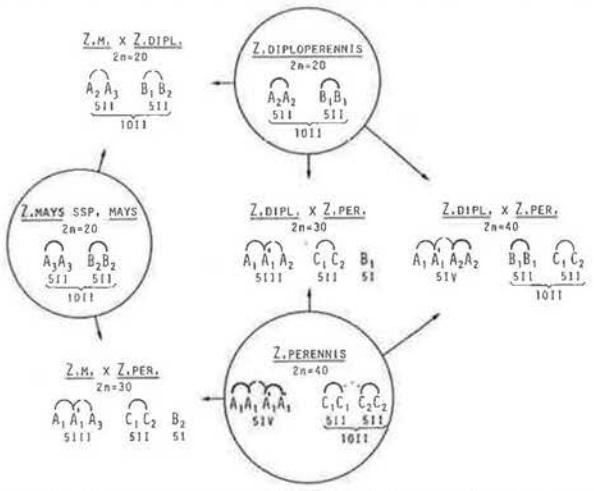

Figure 1. Chromosome numbers, most frequent meiotic configurations and hypothetical genomic constitution of species and hybrids of *Zea* assuming x = 5.

In *Zea mays* subsp. *mays* (2n = 20, Figure 1) 10 bivalents are regularly formed. Secondary associations were observed and a maximum of five groups of two bivalents was frequent at diakinesis-metaphase I. The meiotic configurations are those of a typical allotetraploid.

As was pointed out by Molina (The Nucleus 26:1-7, 1983; MNL 55:51-52, 1981) *Zea diploperennis* (2n = 20; Figure 1) presents a regular meiosis with the formation of 10 bivalents in 73% of the cells studied. The formation of 1 III + 8 II + 1 I in 4% of the cells would indicate the segmental allotetraploid nature of this taxon. Secondary associations were also found in this species and groups of four or five pairs of bivalents are frequent.

Zea perennis (2n = 40, Figure 1) had 10 II + 5 IV in 55% of the cells studied (Molina, MNL 56:96, 1982; Genética Ibérica, in press). These configurations suggest the existence of at least three different genomes (A_1, C_1, C_2). The formation of 5 IV in 55% of the cells would indicate the existence of four highly homologous genomes ($A_1A_1A_1A_1$). The cells with less than 5 IV (4, 3, 2, or 1 IV) probably result from lower chiasmata frequency and do not show formation of III and I. These results suggest high homology by pairs in A_1 genomes (A_1A_1 and $A_1'A_1'$). Sometimes 6 IV were observed and this could indicate some homologies between C_1 and C_2 genomes. On the basis of these results *Z. perennis* would be considered as an auto-allooctoploid with the genomic formula $A_1A_1A_1'A_1'$ $C_1C_1C_2C_2$.

The F_1 hybrid *Zea mays* subsp. *mays* × *Z. diploperennis* (2n = 20, Figure 1) presents the formation of 10 bivalents in ca. 70% of the 181 cells studied; the rest of the cells present 2 to 6 I (Rosales & Molina, MNL 57:63, 1983). These results suggest that there is exclusive allosyndetic pairing (A_2A_3 and B_1B_2). Secondary associations were observed and a maximum of three groups of two bivalents each at diakinesis-metaphase I was formed.

In the F_1 *Z. mays* subsp. *mays* × *Z. perennis* (2n = 20, Figure 1) 5 III + 5 II + 5 I formed in ca. 55% of the 80 cells studied (Molina, Rev. Agr. La Plata 56:513-519, 1978). The 5 III could be formed by autosyndetic pairing of A_1 and A_1' genomes from *Z. perennis* and by homoeologous pairing with the A_3 genome from *Z. mays*. The 5 II could be formed by autosyndetic pairing between the C_1 and C_2 homoeologous genomes from *Z. perennis*. Finally the 5 I would belong to the B_2 genome from *Z. mays*. The same considerations could be applied in the F_1 *Z. diploperennis* × *Z. perennis* (2n = 30, Figure 1) interpretations. Configurations of 5 III + 5 II + 5 I were present in ca. 40% of the 168 cells studied. In this hybrid the 5 I could be formed by the B_1 genome from *Z. diploperennis*.

The F_1 hybrid *Z. diploperennis* × *Z. perennis* (2n = 40, Figure 1) probably originated by the fertil-

ization of an unreduced egg cell from *Z. diploperennis* by a normal male gamete from *Z. perennis* (Molina, Cytologia 50:57-62, 1985). The formation of II + IV was observed in 94% of the 214 cells studied, and III + I were not detected. The most frequent configuration, 5 IV + 10 II, was observed in ca. 32% of the cells. These results suggest high homology between A_1 and A_1' genomes, which is also observed in *Z. perennis*.

The interpretation of the meiotic configurations of the three species and the four hybrids is more difficult as a whole, if a basic number x = 10 is considered (Figure 2). The present analysis constitutes strong evidence in favor of x = 5 for the genus *Zea*.

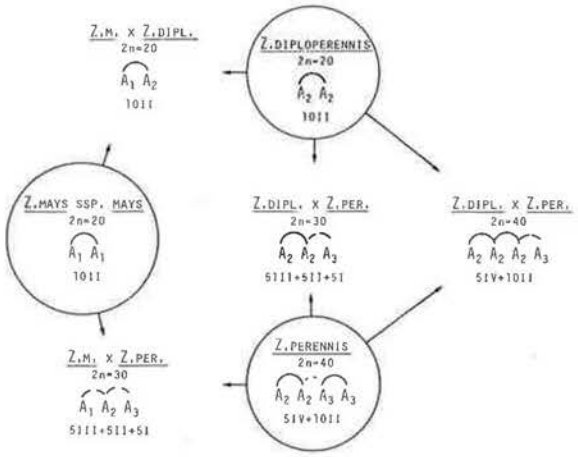

Figure 2. Chromosome numbers, most frequent meiotic configurations and hypothetic genomic constitution of the species and hybrids of Zea assuming x = 10.

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Variation within *Zea:* numerical analysis of fruit traits

When the evolutionary study of *Zea* began in the last Newsletter (MNL 59:61, 1985) we stated that clusters based only on fruit traits were not congruent with those based on other traits. We also stated that this might be due to the inclusion of some non-specific fruit traits. The aim of this article is to present a reexamination of the clusters based on fruit traits. So, we have used basically the same data previously employed but have included some other data. Our objective is to determine an appropriate selection of fruit traits, morphological and biochemical, that may lead to clustering congruent with clusters based on other plant characters. The same 7 operational taxonomic units (OTU's) were studied. Sixteen traits were scored for each OTU as follows: (1) Kernel (PG) enclosed (1), naked (0); (2) Kernel (TG) small (0), intermediate (1), large (2); (3) Cupulate fruit case (FCFC) trapezoidal (2), triangular (1), horizontally compressed (0); (4) Cupule orientation (OC) vertical (0), horizontal (1); (5) Kernels per cupule (GC) one (1), two (2); (6) Number of fruit cases per spike (NCF); (7) Endosperm protein content (%) (PE); (8) Landry-Moureaux saline soluble proteins (PSS); (9) Glutelin-1 (G_1); (10) Glutelin-3 (G_3); (11) Pericarp thickness (μm) (EP); (12) Aleurone layer thickness (μm) (ECA); (13) Starch granule size (μm) (TGA); (14) Zein body size of zone 1 (μm) (TGZ1); (15) Zein body size of zone 2 (μm) (TGZ2); (16) Zein body size of zone 3 (μm) (TGZ3). Given a basis data matrix (BDM) (Table 1) of 16

Table 1: Basic data matrix (BDM): Fruit traits

	Characters							
OTU's	1	2	3	4	5	6	7	8
Zp	1	0	2	0	1	5.1	21.0	1.8
Zd	1	0	2	0	1	7.5	27.0	3.1
Zl	1	0	2	0	1	6.4	23.6	3.3
Zmpp	1	0	1	0	1	8.8	26.5	2.8
Zmph	1	0	1	0	1	8.6	23.4	1.8
Zmmx	1	1	1	0	1	11.2	17.7	4.3
Zmm	0	2	0	1	2	NC	11.4	5.7
OTU's	9	10	11	12	13	14	15	16
Zp	20.7	9.2	17.0	42.3	17.3	1.7	1.6	1.4
Zd	9.2	10.0	21.0	39.7	12.5	1.5	1.2	1.0
Zl	7.9	9.9	13.6	31.8	17.6	1.7	1.5	1.1
Zmpp	7.1	9.3	12.8	28.3	17.1	1.7	1.2	0.9
Zmph	12.9	12.2	13.8	29.5	17.0	1.3	1.0	0.9
Zmmx	7.4	14.6	23.7	42.0	14.9	1.4	1.4	1.0
Zmm	12.3	13.0	95.8	45.9	16.0	1.8	1.5	1.4

characters by 7 OTU's the data were analyzed by cluster analysis. The BDM was standardized (BDMS) by characters to remove the unequal weights imposed on it by the use of different scales of measurement. Phenograms were derived by (1) the Pearson product-moment correlation coefficient, applying the unweighted pair group method using arithmetic averages (UPGMA) (Figure 1), (2) the "Mean Taxonomic Distance" between pairs of OTU's served as input in the calculation by UPGMA (Figure 2) and by the COMPLETE cluster analysis (Figure 3). A character x character correlation matrix was obtained from BDMS by calculating the Pearson product moment correlation coefficient between each pair of the 16 characters, and served as input in the

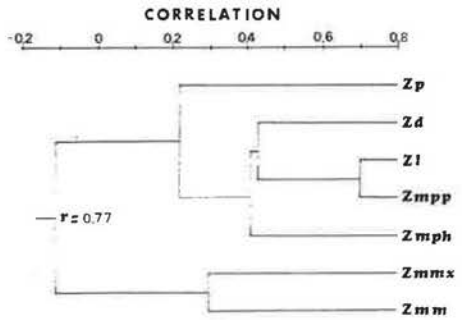

Figure 1: Phenogram of 7 OTU's resulting from the UPGMA cluster analysis of the OTU x OTU correlation matrix. r= cophenetic correlation coefficient.

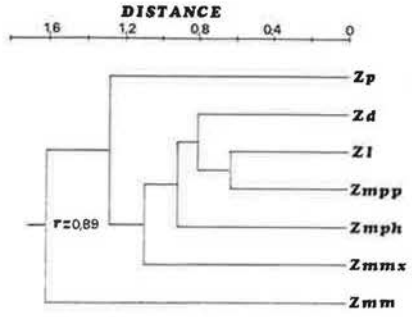

Figure 2: Phenogram of 7 OTU's resulting from the UPGMA cluster analysis of the OTU x OTU distance matrix. r= cophenetic correlation coefficient.

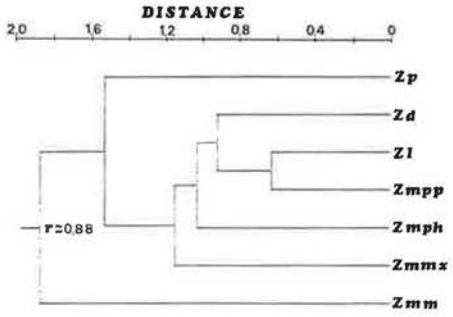

Figure 3: Phenogram of 7 OTU's resulting from the COMPLETE cluster analysis of the OTU x OTU distance matrix. r= cophenetic correlation coefficient.

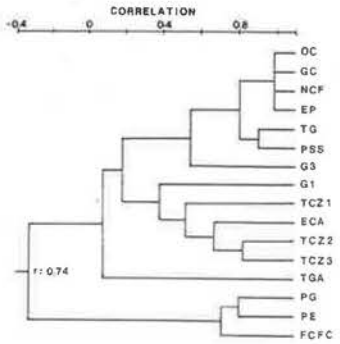

Figure 4: Phenogram of 16 characters resulting from the UPGMA cluster analysis of the character x character correlation matrix. r= cophenetic correlation coefficient.

calculation of a phenogram by UPGMA (Figure 4). The "Manhattan Distance" between pairs of OTU's served as input in the calculation of "Wagner Trees" (Figure 5).

The results obtained from *Zea* taxa cluster analysis based only on fruit traits appear to be highly congruent with clustering based on other characters (from plant, inflorescence, etc.) reported (MNL 59:61, 1985). Congruence measured by correlation coefficient was high and extremely significant (r = 0.83). This fact points out the high fruit trait taxonomic value, that allows the taxa to be grouped in an approximately similar way to those done on the basis of more complex studies. Phenograms of Figures 1, 2 and 3 based on fruit traits show that Zp and Zmm

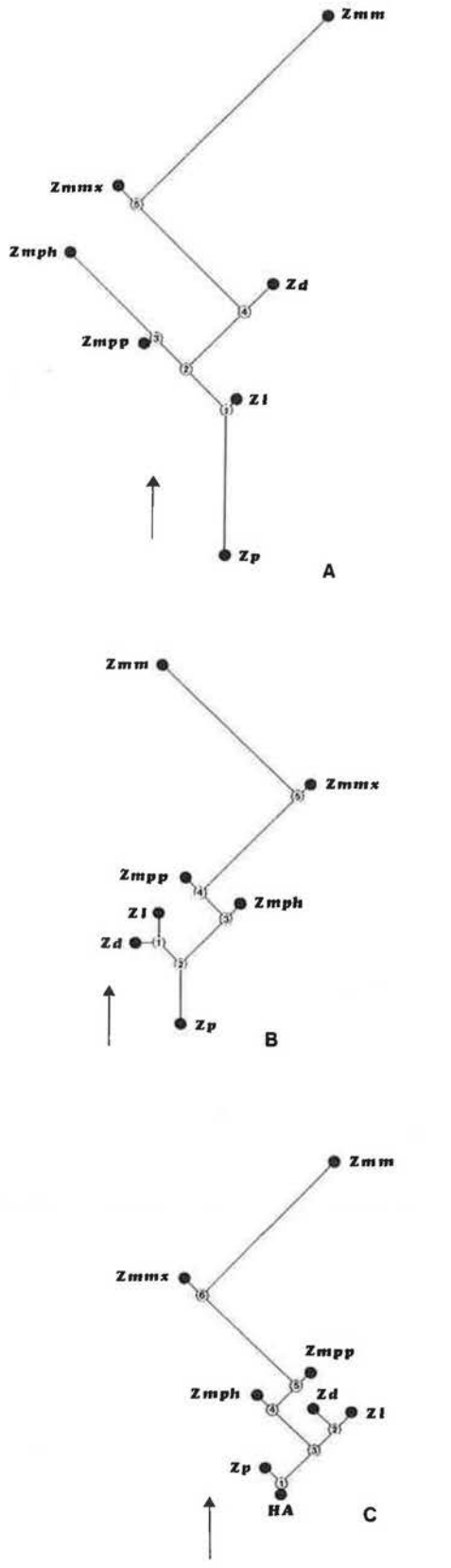

Figure 5: (A) Wagner Tree for the OTU's based on 16 characters, considering Zp as ancestor.
(B) The same based on 12 characters. (C) Wagner Tree based on 12 characters, considering a hypothetical OTU as ancestor (HA). Arrows indicate the evolutionary direction. Numbers corresponding to hypothetical taxonomic units represent the construction sequence of the tree.

are the most extremely differentiated taxa and that Zmpp and Zmph are more like Zd and Zl than Zmm.

Many traits are significantly associated and these traits are those which have most specific value. On the other hand, other traits are represented as isolated by the phenogram shown in Figure 2, and are weakly associated with the remainder, being perhaps, as was pointed out previously (MNL 59:61, 1985), of no specific importance and of little adaptive value. The evolutionary trees of Figure 5 are approximately similar between them. However a fundamental topic is found when choosing those traits in which "Wagner trees" construction is based. Using the 16 characters (Figure 5 A) a certain distortion exists if we consider only 1 to 12 traits (Figure 5 B and C). This happens because those traits which have suffered evolutionary changes and have adaptive value are to be previously stated clearly, removing those traits such as 13 to 16 that don't demonstrate specific value (see MNL 59:61, 1985).

Evolutionary trees built on the basis of 12 fruit traits with evolutionary significance keep a high similitude between them and a high congruence with those built on the basis of 28 traits (see MNL 59:61, 1985). Evolutionary trees show Zp as the most primitive teosinte (considering Zp as ancestral as well as a hypothetical ancestor) to which the other two taxa of *Luxuriantes* Section (*Z. diploperennis* and *Z. luxurians*) link at the lower part of the tree. Balsas teosinte and Huehuetenango are nearer to the most primitive teosintes than to *Z. mays* ssp. *mexicana* which is, in accordance with fruit traits, the teosinte more similar to maize.

Those demands, that Wagner's tree (monophyletic) has in its construction, make Zmm linked to Zmmx and placed at the top of the tree. This ought not to be interpreted as if maize had been originated from Zmmx, but as it is the most similar taxon because of being the teosinte with more maize introgression.

Based on our previously discussed hypothesis (MNL 59:61, 1985) it can be pointed out that fruit traits considered isolated may also support the idea that maize introgression, first into most primitive teosintes and then into their derived products, has been the principal factor in teosinte evolution, in the same way as it has played an important role in modern maize evolution.

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and M.E. Streitenberger

More about teosinte characterization

The taxonomy and evolution of *Zea* can be considered as a problem. When trying to solve a problem from different angles and using different techniques, you have the chance to obtain a global view of it. In relation to this, Bird (Maize for Biol. Res. p. 341, 1982) correctly states that: "a careful, integrated analysis of a large and varied body of data, including studies of plant, ear and tassel morphology, chromosome knobs, isozymes, and whatever other features can be surveyed, is needed." We are determined to obtain within our possibilities, the greatest amount of information for *Zea* taxa characterization. For this reason, several morphological and biochemical studies are being carried out. The aim of this report is to present some results obtained in the evaluation of some quantitative traits, especially fruit and tassel traits. These studies were performed on small populations of teosintes, which were cultivated during spring and summer of 1984/85. The great number of observations done on each trait considered are summarized.

Cultivated plants of *Z. perennis* (Zp), *Z. diploperennis* (Zd), *Z. luxurians* (Zl), *Z. mays* ssp. *parviglumis* var. *parviglumis* (Zmpp), *Z. mays* ssp. *parviglumis* var. *huehuetenangensis* (Zmph) and *Z. mays* ssp. *mexicana* (Zmmx) were employed for the measurements. The following 12 quantitative traits related principally to the fruit and tassel were recorded: fruit case weight (FCW), length (FCL), width (FCWD) and thickness (FCT); tassel branch number (TBN), tassel branching axis length (TBAL), central spike length (TCSL) and lateral tassel branch internode length (LTBIL); distance between the two primary lateral veins (DVL), number of veins between primary lateral veins (VBL) and total vein number (TV) of male spikelet outer glume; and number of tillers per plant (NT) (Table 1).

Table 1: Some quantitative traits of teosinte

Character	Taxa					
	Zp	Zd	Zl	Zmpp	Zmph	Zmmx
FCW (mg)	54.6 ± 12.0	59.7 ± 10.7	62.3 ± 19.2	36.4 ± 10.4	29.0 ± 9.0	104.6 ± 23.9
FCL (mm)	7.0 ± 0.3	7.5 ± 0.5	8.7 ± 0.6	6.1 ± 0.6	6.2 ± 0.5	8.4 ± 0.7
FCWD (mm)	4.1 ± 0.3	4.4 ± 0.3	4.1 ± 0.4	3.6 ± 0.5	3.6 ± 0.4	5.5 ± 0.6
FCT (mm)	3.3 ± 0.2	3.5 ± 0.3	3.6 ± 0.6	2.7 ± 0.3	3.0 ± 0.4	4.4 ± 0.4
TBN	2.8 ± 1.2	6.3 ± 2.7	14.0 ± 1.7	57.9 ± 18.7	29.4 ± 4.7	24.5 ± 5.0
TBAL (cm)	1.6 ± 0.4	1.8 ± 0.7	3.9 ± 0.8	9.1 ± 2.3	8.6 ± 2.1	14.1 ± 2.7
TCSL (cm)	9.4 ± 1.9	7.9 ± 2.0	6.3 ± 2.2	6.1 ± 1.5	8.3 ± 1.3	12.0 ± 6.5
LTBIL (mm)	3.7 ± 0.3	4.7 ± 0.7	4.7 ± 1.4	4.4 ± 0.4	5.2 ± 0.8	4.4 ± 1.6
DLV (mm)	2.4 ± 0.3	2.5 ± 0.3	2.4 ± 0.2	1.7 ± 0.1	1.9 ± 0.4	2.5 ± 0.4
VBL	12.0 ± 1.1	10.5 ± 1.3	15.3 ± 2.3	4.3 ± 0.5	6.8 ± 1.8	5.3 ± 1.9
TV	17.9 ± 1.7	16.6 ± 1.4	25.8 ± 3.1	8.8 ± 0.6	13.7 ± 1.7	10.2 ± 2.2
NT	—	—	11.8 ± 3.9	23.9 ± 7.1	20.6 ± 4.0	4.8 ± 1.5

Assuming Doebley and Iltis' (1980) proposal that teosintes can be clustered within two sections, we have found that some traits such as TBN, TBAL, VBL and TV fit in with this separation. The remainder, especially TCSL, DLV and LTBIL, doesn't give a clear discrimination within the groups. A phenogram resulting from cluster analysis based on these traits is shown in Figure 1. From the results obtained it can be inferred that Zp, Zd and Zl form a well defined group constituted by the most primitive teosintes. Zmpp and Zmph form another group that links at a lower distance from the first than from Zmmx. Zmpp and Zmph are more closely linked to the primitive teosinte group than to the most evolved one (Zmmx). Some traits such as the fruit traits considered and TBN, TBAL and DLV allow us to cluster Zmpp and Zmph separately from Zmmx, with which they appear to have lower affinity. Some

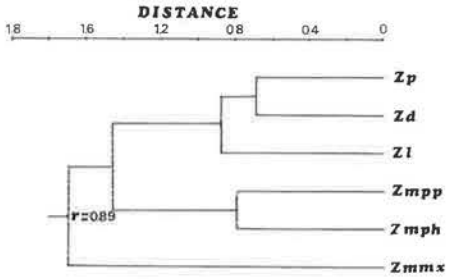


Figure 1: Phenogram of 6 OTU'S resulting from the UPGMA cluster analysis of the OTU x OTU distance matrix. r= cophenetic correlation coefficient.

differences are shown within those clusters reported (see MNL 59:61), based on characters that were not considered previously.

For this reason, and in accordance with what we have pointed out here, not only taxonomy but also phylogenetic relationships among the taxa of *Zea* must be reexamined upon the basis of a multidisciplinary study of the problem. Probably, the correct way of doing it is collecting a great amount of data with the aim of studying it by means of multivariant analysis methods.

Mónica B. Aulicino and Jorge L. Magoja

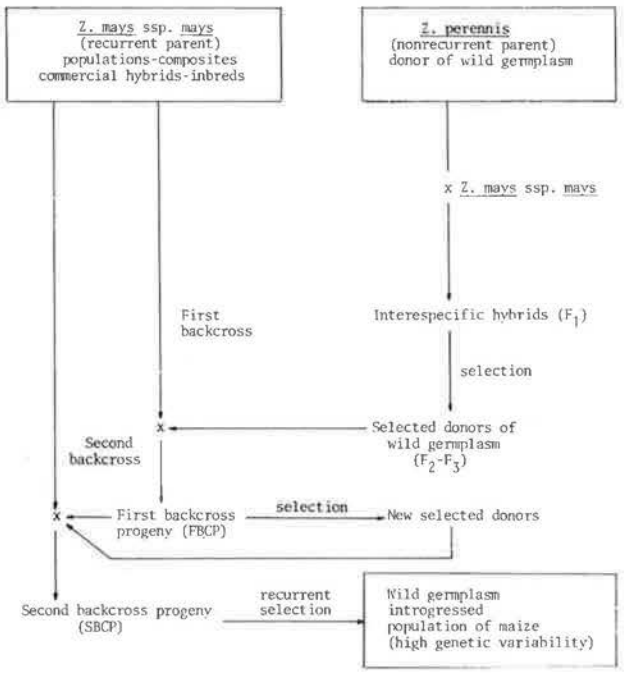
Introgression of teosinte germplasm in maize: a method to improve heterosis and variability

Most variation in maize has been produced or maintained by teosinte introgression. There is considerable documentation about the important role that teosinte has played in the evolution of modern maize. The idea of employing maize wild relatives to improve the cultivated species is not novel either in its theoretical conception or practical implementation. However, in view of the interesting results obtained from our perennial teosinte (*Z. perennis*) germplasm introgression into the maize project, it seems convenient to report the experience reaped since the beginning of the research in 1983.

Perennial teosinte (*Z. perennis*) and diploperennial teosinte (*Z. diploperennis*) have been our donors of wild germplasm. These species were chosen because they are the most primitive taxa of the genus and thus are the ones that produce more variability and heterosis in crosses with maize. Our main objective was to produce a controlled introgression of teosinte into maize, so as to transfer into the cultivated species those genes or alleles able to produce additional heterosis and increase genetic variability. Now we report results of perennial teosinte germplasm introgression. This is the project that raises more expectancy and where initially the main difficulties of practical implementation have been manifested.

As is shown in Figure 1, *Z. perennis* was the wild germplasm donor and maize was the recurrent parent.

Figure 1: Method of wild germplasm introgression in maize



Since hybrids between perennial teosinte and maize have low fertility and produce few viable kernels when crossed again with maize, the donating material was previously selected. So, individuals with maizoid phenotype, highly vigorous and fertile, were chosen among F_2 and F_3 progenies and used in the first backcross. Seed development was low (15% of available ovules) when maize ears were pollinated with pollen from perennial teosinte germplasm donating individuals, and only 10% of them or less were viable. However, the plants obtained were highly heterotic and showed a high genetic variability. The next year, a great number of maize ears were pollinated with a mixture of pollen collected from several plants of the first backcross progeny (FBCP). Maize pollen was also used to pollinate FBCP plants.

We thought that after the first backcross more seeds should be obtained, but surprisingly only 4 kernels for each 100 available maize ovules were obtained from the new pollinations (see Table 1). However, our surprise was even greater when, cultivating the second backcross progeny (SBCP), almost every seed was viable and produced very vigorous plants which presented a high variability within them. Since all maize traits were mainly recovered

Table 1: Results obtained in the second backcross using FBCP as male parent

Female parent				
Inbred hybrid or population	Total number of ears pollinated	Number of ovules per ear	Number of seeds developed per ear	Percent of seeds developed
84-BKP	92	579.5 ± 149.5	18.3 ± 26.6	3.2
84-700	49	372.6 ± 58.8	2.4 ± 3.4	0.6
84-7606	42	384.1 ± 94.8	31.5 ± 54.7	8.6
84-FE	22	401.9 ± 85.7	12.2 ± 9.9	3.0
84-717	7	264.3 ± 49.6	14.0 ± 15.8	5.3
84-718	9	247.5 ± 51.2	18.9 ± 18.5	7.6
84-7625	5	322.0 ± 82.3	2.2 ± 3.3	0.7
84-CF42	8	257.5 ± 76.3	3.8 ± 3.6	1.5
84-OU	4	174.0 ± 10.0	1.8 ± 1.5	1.0
84-7594	2	275.0 ± 21.1	32.5 ± 3.5	11.8

in the plants obtained after the second backcross, we have considered it appropriate to interrupt backcrosses and start a recurrent selection process with the aim of forming a maize population with wild germplasm introgression that may be used in the future to obtain inbred lines. During the application of this method we have evaluated 30 traits of agronomic and/or specific importance in the donating materials as in the progenies of the backcrosses obtained. These characters have been used to point out the progress in recovering the specific maize traits and to quantify the variability produced by wild germplasm contribution. We shall give only a few results obtained with the aim of showing that changes operated during the introgression process and variability occurred in many of the evaluated characters.

Table 2 shows the results obtained for some characters evaluated in FBCP. It is important to remark on their high variability. In general FBCP

Table 2: Relevant quantitative traits of the FBCP.

Character	Mean ± SD	Range
Days to tassel	74.4 ± 11.4	45-105
Days to silking	100.4 ± 13.8	74-128
Days to pollen	87.4 ± 10.8	66-118
Tassel branch number	23.6 ± 9.2	5-52
Tassel branching axis lenght (cm)	14.2 ± 3.4	5-24.1
Tassel central spike lenght (cm)	20.9 ± 4.4	14-30.4
Lateral tassel branch internode lenght (mm)	4.9 ± 0.9	3.1- 7.9
Pollen grain size (um)	99.5 ± 3.1	91.6-106.0
Number of leaves	12.5 ± 2.3	7.0- 19.0
Leaf lenght (cm)	87.4 ± 12.4	64.5-116.5
Leaf width (cm)	8.4 ± 1.2	5.9- 11.5
Stalk diameter (cm)	1.5 ± 0.3	0.9- 2.3
Number of tillers	7.0 ± 2.7	1-15
Number of productive nodes	3.4 ± 1.2	1-6
Number of ears in the uppermost node	6.9 ± 4.2	1-19
Number of ears per tiller	27.4 ± 18.6	1-91
Number of ears per plant	129.4 ±106.8	2-452
Number of kernel rows per ear	6.0 ± 2.0	4-12

plants have recovered enough maize specific traits, but they also keep undesirable traits such as numerous small ears per productive node and many stalks per plant. In general FBCP plants are very vigorous and extremely prolific, although ears produced are generally small and badly covered by husks. In spite of the fact that desired plant traits (that is to say plants like maize but with high vigour and prolificity) have not been reached yet, the progress towards our objective was very significant. This may be proved with the results shown in Table 3, where several FBCP traits are compared with perennial teosinte germplasm donating material (F_3SM).

At this moment SBCP is being cultivated in the field. All the same, we have some preliminary results available from this SBCP cultivated in the greenhouse (see Table 4). Although the evaluated traits are few and come from a small SBCP population, they are useful to demonstrate the extremely high engendered variability. The most important fact is that SBCP plants have recovered all maize traits, preserving in most cases an exceptional heterosis expressed through their prolificity. Although the number of ears per plant is lower than in FBCP, they are bigger and well covered by husks over vigorous

Table 3: Differences between the first backcross progeny (FBCP) and perennial teosinte germplasm donor (F_3SM)

	T	S	P	TBN	TBAL	TCSL
FBCP	74.4^a	100.4^a	87.4^a	23.6^a	14.2^a	20.9^a
F_3SM	86.0^b	113.4^b	97.2^b	17.9^b	10.9^b	19.5^a
	LTBIL	PGS	LL	LW	SD	NL
FBCP	4.9^a	99.5^a	87.4^a	8.4^a	1.5^a	12.5^a
F_3SM	4.7^b	91.3^b	69.1^b	7.1^b	1.2^b	12.8^b
	NT	PN	EUN	ET	KRN	
FBCP	7.0^a	3.4^a	6.9^a	27.4^a	6.0^a	
F_3SM	8.7^b	4.3^b	6.3^a	29.8^a	3.7^b	

Ref.: T: days to tassel; S: days to silking; P: days to pollen; TBN: tassel branch number; TBAL: tassel branching axis lenght (cm); TCSL: tassel central spike lenght (cm); LTBIL: lateral tassel branch internode lenght (mm); PGZ: pollen grain size (um) LL: leaf lenght (cm); LW: leaf widht (cm); SD: stalk diameter(cm); NL: number of leaves; NT: number of tillers; PN: number of productive nodes; EUN: number of ears in the uppermost node; ET: number of ears per tiller; KRN: kernel row number. Individual mean within a column followed by different letters are significantly at 5% level.

Table 4: Relevant quantitative traits of SBCP

Character	Mean ± SD	Range
Plant height (cm)	247.5 ± 44.3	153-320
Ear insertion height (cm)	118.0 ± 32.3	45-180
Leaf number	11.2 ± 2.0	8-15
Leaf number above the upper ear	4.0 ± 0.9	2-6
Tassel branch number	18.9 ± 14.0	5-71
Leaf lenght (cm)	87.8 ± 6.8	59-110
Leaf widht (cm)	6.8 ± 1.5	3.7-10.5
Stalk diameter (cm)	1.5 ± 0.2	0.9-1.9
Number of ear per plant	6.0 ± 3.1	1-12
Stalk number	1.2 ± 0.6	1-3
Days to 50% silking	64.4 ± 4.5	57-77
Number of kernel rows per ear	13.4 ± 2.2	8-18

and strong stalked plants. The variability engendered is enormous: from very poorly developed plants with thin stalks and few small ears to strong stalked plants with 2 to 5 ears, which are bigger and well formed and add as desirable traits simultaneity in pollen shed and silk emergence, and earliness given from Gaspé germplasm (parent of original crosses). SBCP results cannot be compared with FBCP ones, especially in plant size, stalk diameter, etc., because greenhouse grown plants are higher and have thinner stalks and smaller leaves. Nevertheless the results obtained show a real recovery of maize traits after the second backcross. Simultaneously, high diversity that will allow us to obtain in the future improved maizes from materials with perennial teosinte introgression have been produced. Most outstanding SBCP individuals have high yield power and therefore an appropriate selection of them may give a significant increase in kernel yield of current commercial maize.

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Puna maize: results after three generations of selection for high protein quality and hard endosperm

Obtaining and selection of maize inbreds with high protein quality and hard endosperm from a native population from Puna (Jujuy, Argentina) was

previously reported (MNL 57:73, 1982; MNL 59:67, 1985). The aim of this report is to communicate the progress obtained after three generations of selection. The selection was based on protein quality, evaluated by means of tryptophan content of endosperm protein. Other traits were also evaluated in order to get information that allowed selection of lines which combine high protein quality with hard endosperm. Kernel weight, endosperm hardness (measured on an arbitrary scale: 0= floury to 4= flinty) and kernel apparent density were measured. Forty-three S_3 lines were studied, 22 of them from the best S_2 (MNL 59:67, 1985). Results obtained on S_3 are shown in Table 1. S_3 lines have, on average, 75% more tryptophan than normal maize, 50% more than S_1 and 25% more than S_2 (Table 2).

During selection and inbreeding, some changes occurred such as kernel weight and protein content reduction. However, other traits do not change (endosperm hardness and kernel apparent density). Protein quality (tryptophan content) was not increased, due to the decrease of hard endosperm ratio. The best S_3 lines have a good combination of protein quality and endosperm hardness (Table 3).

Table 1: Characteristics of Puna S_3 lines

	Kernel weight (mg)	Hardness	Density (g/ml)	Protein (%)	Tryptophan (g/100g. prot.)
Mean	219.0	2.3	1.18	11.3	0.7
SD	40.4	1.1	0.23	1.5	0.1
Range	164-300	0-4	0.64-2.00	8.6-13.8	0.5-0.8

Table 2: Differences between S_1, S_2, S_3 lines

	Kernel weight (mg)	Hardness	Density (g/ml)	Defatted endosperm Protein (%)	Defatted endosperm Tryptophan (g/100g. prot.)
S_1	231.1^{ab}	—	—	13.3^{a}	0.5^{a}
S_2	256.8^{a}	2.5^{a}	1.21^{a}	12.0^{b}	0.6^{b}
S_3	210.0^{b}	2.3^{a}	1.18^{a}	11.3^{bc}	0.7^{c}

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

Table 3: Characteristics of the best Puna S_3 lines.

S_3 line	Kernel weight (mg)	Hardness	Density (g/ml)	Defatted endosperm Protein (%)	Defatted endosperm Tryptophan (g/100g. prot.)
H_6	195.0	3	0.90	9.3	0.7
H_7	194.0	4	0.64	10.6	0.8
H_8	176.0	4	1.27	10.6	0.8
H_9	189.0	3	1.24	9.6	0.7
H_{10}	202.0	3	1.28	9.6	0.8
H_{12}	190.0	3	1.20	12.6	0.7
H_{36}	189.0	4	1.22	10.3	0.8
Average	192.1	3.4	1.11	10.3	0.8

High protein quality of selected Puna lines may be attributed to a modification of normal endosperm protein pattern. From lines selected with high tryptophan content (H_8) the endosperm proteins were fractioned according to Landry-Moureaux and compared with normal inbreds (DY). Results obtained are given in Table 4. H_8 line with high tryptophan content has less zein and more saline soluble and glutelin-3 than normal DY. Results obtained indicated that: 1) protein quality selection has been rapid and positive and 2) Puna maize may be employed to improve protein quality of maize endosperm without affecting its normal corneous phenotype.

Table 4: Endosperm protein pattern of normal (DY) and hard endosperm-high quality protein Puna S_3 line (H_8)

Fraction (*)	Soluble nitrogen (% of total) DY	H_8
SS	6.8	8.3
Z	51.7	44.8
G_1	13.9	15.5
G_2	10.4	7.2
G_3	13.2	20.3
protein (%)	12.9	10.6
tryp.(g/100g prot.)	0.4	0.8

(*): SS: saline; Z: zein; G_1: glutelin-1, G_2: glutelin-2; G_3: glutelin-3.

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Expression of *de*-7601* under different genetic backgrounds

A spontaneous mutation that conditions defective kernels appeared in WK-01 inbred line. Its most evident effect is the dramatic reduction of kernel weight (MNL 56:108, 1982). This lethal character produces several effects in the original genetic background that may be quantified, such as modifications in storage proteins of mature kernels (MNL 56:108, 1982) or during their development (MNL 57:71, 1983). Perhaps the mutant is associated with other genes that increase the seed free amino acid content (MNL 58:120, 1984).

With the aim of studying the expression of the mutant under the influence of other genetic backgrounds, different or partly different from the original one, it was transferred into several genetic backgrounds in inbred conditions. An easy way to quickly detect the different expression is to quantify kernel weight and relate defective weight to the normal equivalent. A great variability in the expression is detected in accordance with genetic background as shown in Table 1. In the original genetic background defective kernels average 27.1% of their normal equivalent's weight, with a relatively narrow variation (25.2 to 32.1%). As Table 1 shows, the broad variation increases greatly, defective kernels weighing from only 13% of normal to more than half. The whole range of variation is represented with the 12 genetic backgrounds selected (Table 2). Within some of these genetic backgrounds, mutant expression is significantly different. When the frequency distribution of normal and defective kernel weight is represented under the influence of the

Table 1: Weight of normal and defective kernels: Means and ranges of 29 genetic backgrounds.

	Kernel weight (mg) Normal	Kernel weight (mg) Defective	Weight of defectives as percent of normal weight
Mean	214.7	59.1	28.3
SD	41.5	17.4	9.4
Range	134-279	29-106	13-55

Table 2: Weight of normal and defective kernels of 12 selected genetic background.

Genetic background	Kernel weight (mg) Normal	Defective	Weight of defectives as porcent of normal weight
A	212.6	28.5	13.4
B	277.5	44.6	16.1
C	195.7	37.5	19.2
D	254.5	57.7	22.7
E	232.4	58.8	25.3
F	251.4	71.5	28.4
G	257.5	83.8	32.5
H	144.5	51.1	35.4
I	165.6	62.3	37.6
J	249.5	105.7	42.4
K	176.2	85.1	48.3
L	161.3	87.9	54.5

diverse genetic backgrounds (Figure 1), it can be seen that at the same time as defective kernels represent higher weight (in relation to normals), the distributions are superimposed. While on one hand (background A) a clear gap appears within phenotypes, in another (background L) superimposition is considerable.

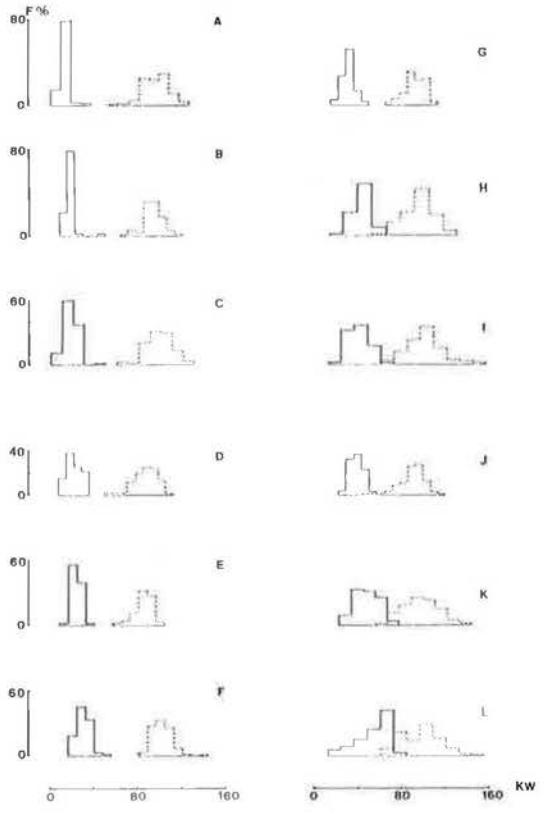

Figure 1: Frequency distributions of defective (solid lines) and normal (broken lines) kernel's weight (KW) in A-L genetic backgrounds. Kernel weight of normals and defectives are expressed for each background as % of the average kerenel weight of normal kernels.

The results obtained show that the different expression of *de*-7601* may be conferred by genetic modification. If this is possible an appropriate store of modifiers (feasible of being selected) may be able to condition, perhaps, a complete superimposition in the distribution of normal and defective kernel weight. This possibility is predictable starting from the given results. The increase of defective weight is associated with a modification in their viability: despite the fact that primitively in its original background it was a lethal trait, genetic backgrounds "J", "K" and "L" condition normal viability in mutant carrier kernels.

I.G. Palacios, M.E. Streitenberger
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Rescue of *de*-7601* homozygous plants

As was previously reported (MNL 56:108, 1982) the mutant *de*-7601*, which conditions defective kernels, is lethal in its original genetic background (WK-01). As seen in the previous report, other genetic backgrounds modify the expression of the mutant: defective kernels show better development, so they are able to germinate and to produce seedlings.

Ten defective kernels (those with better development) corresponding to "L", "K" and "J" genetic backgrounds (see previous report) were set to germinate in little pots. Generally, those selected grains produced a high percentage of plants, whose phenotypes are presented in Table 1. Germinative energy

Table 1: Mutant kernel germination, seedling and plant phenotype.

Genetic background	Number of seeds	Number of seedling(*) 5	6	7	8	9	10	Seedling phenotype	Mature Plant phenotype
L	10	5	7	7	7	7	7	normal	normal
K	10	0	2	5	8	8	8	mutant	dwarf ear-less
J	10	0	4	4	7	8	9	mutant	dwarf tassel-less

(*) Obtained at 5,6,7,8,9 and 10 days after planting.

Table 2: Characters of normal (+/+) and defective (de/de) plants with "L" genetic background

Character	Plant genotype +/+	de/de	Signif. (1)
Plant height (cm)	241.2	215.1	*
Ear insertion height (cm)	111.5	87.9	**
Leaf number	12.5	12.1	NS
Leaf number above upper ear	5.3	5.4	NS
Tassel branch number	12.6	12.2	NS
Leaf lenght (cm)	76.0	72.0	NS
Leaf widht (cm)	6.6	6.2	NS
Stalk diameter (cm)	1.2	1.2	NS

(1): *: Significant at 5% level; **: Significant at 1% level, NS: not significant.

was very different among genetic backgrounds considered, kernels from "L" genetic background germinated more quickly and produced normal and vigorous seedlings. Kernels from "K" and "J" genetic backgrounds germinated more slowly, though they produced a high percentage of seedlings with mutant phenotype. Plants from "J" genetic background were small, with short internodes and a mean height of 26.0 ± 3.2 cm (from 22 to 30 cm) with an average of 8 leaves (from 7 to 9). The stalk ended at the apex in a little distichous ear, which was fertile when pollinated with pollen from other corns. A little axillary ear developed too, and this one was silkless. Plants from "J" genetic background had a mutant phenotype and were completely female because they only produced female flowers. Plants from "K" genetic background were small, with short internodes and a mean height of 15.0 ± 1.8 cm (from 13 to 17 cm) and an average of 8 leaves (7 to 9). The stalk ended at apex in a small tassel without branching, formed by a single distichous axis with

sterile flowers. These plants were completely masculine. On the contrary, plants from "L" genetic background had normal appearance and fertility and had only less plant height and ear insertion height compared with normal equivalent plants (Table 2). From 7 plants with "L" genetic background, 6 ears were obtained by selfing and the remaining were crossed by normal pollen source. Self pollinated ears produced normal and defective kernels (3:1 ratio) and the crossed ears also produced normal and defective kernels, in a ratio of 9:1. Defective kernels were as frequent as those that originated the cultivated plants. Data obtained cannot be a mistake of kernel classification.

Though in "L" genetic background normal and defective kernels superpose their frequency distributions because of their weight, they have a very different phenotype: normal kernels are flinty and defective ones are floury. Heterofertilization phenomenon, instability of the mutant with reversion in the germ, or effects of genetic modifiers might be the cause for the obtained segregations on plants originating from defective kernels. Additional tests must be performed, cultivating new defective kernels, in order to throw light on the observed phenomenon. Results obtained indicate that viability of mutant kernels may be changed under favorable genetic backgrounds and a complete suppression of lethality may be expected.

I.G. Palacios and J.L. Magoja

Perennial teosinte-Gaspé hybrids: inheritance of pericarp thickness

Pericarp thickness is a fruit trait that allows differentiation not only between maize and teosinte but also within teosinte (MNL 59:61, 1985). The aim of this article is to amplify on the data we have presented (MNL 59:68, 1985) on pericarp thickness in hybrids between perennial teosinte (*Zea perennis*) and maize (Gaspé). As previously pointed out (MNL 59:68, 1985) pericarp thickness was studied in 20 plants of perennial teosinte, 20 plants of Gaspé, 50 plants F_1 (Gs x Zp), 50 plants F_1 (Zp x Gs) and 150 plants F_2 (Zp x Gs).

Although it seems unnecessary, it is important to remark that as the pericarp is maternal tissue, evaluation was performed on F_2 kernels (F_1 pericarp) and F_3 kernels (F_2 pericarp). The study was carried out considering individual plants from each of the 5 populations analyzed. As shown in Table 1, Gaspé has more than four times the pericarp thickness of perennial teosinte. Variability is low in both parents, probably pointing out lack of environmental effect over phenotypic expression. Reciprocal F_1 averages as well as F_2 are similar. The variation range and phenotypic variation coefficient are close.

The results presented in Table 2 show that the main variation is genetic and therefore the heritability of this character calculated from these data is high (0.80). The trait variability that can yet be observed in both reciprocal F_1s is similar to that from F_2. Consequently this led us to think that pericarp thickness "segregates" within F_1 plants. This particular phenomenon, that can be attributed to metaxenia, may be explained upon the basis of the marked influence exerted by the underlying genotype (F_2 seed) upon F_1 pericarp thickness. The germ or the endosperm or both (products of genetical recombination) present several F_2 genotypes, and probably these tissues may produce some metabolite which may influence pericarp thickness during kernel development. The fact that all F_2 kernels may have a F_1 pericarp with the same genotype led us to suppose "a priori" that this genotype ought to express a unique phenotype (thickness more or less constant). The only acceptable explanation, since there is low environmental influence on this character (as it is expressed in the parents) is metaxenia phenomenon. Metaxenia has been demonstrated on fruit traits in other vegetable species such as the genus *Solanum* (Andeew, 1969), apple (Kovacs, 1976) and palm (Ream, 1978). Such as in our case, Andeew (1969) pointed out that metaxenia is produced in hybrids between species genetically far apart.

Comparison among average values of pericarp thickness for the studied populations are shown in Table 3. No significant differences can be detected between F_1 in relation to F_2. This fact reinforces the hypothesis that the trait "segregates" in F_1 generation (F_1 pericarp and F_2 seed).

Figure 1 shows the distribution of frequencies of the studied trait. It can be observed that F_1 and F_2 distributions are similar, moving towards the left, that is to say towards perennial teosinte. This fact together with the results given in Tables 1 and 2 points out strongly that thin pericarp (perennial teosinte) partially dominates over thick pericarp (maize). It is important to remark that those F_2

Table 1: Number of plants (N), mean ($\bar{x}$), standard desviation (DS), range and phenotypic variation coefficient (PVC) in perennial teosinte (Zp), Gaspe (Gs) and its F_1 and F_2 populations.

		Pericarp thickness (um)		
	N	$\bar{x} \pm$ DS	range	PVC %
P_1(Zp)	20	17.0 ± 1.8	16.0-20.0	10.6
P_2(Gs)	20	76.0 ± 7.2	65.0-88.0	9.5
F_1(Gs x Zp)	50	30.1 ± 8.0	15.0-55.2	26.6
F_1(Zp x Gs)	50	33.8 ± 9.1	21.6-64.8	26.9
F_2(Zp x Gs)	150	31.2 ± 8.0	16.0-57.6	25.6

Table 2: Means, variances and genetic variation coefficients (gvc%) of perennial teosinte (Zp), Gaspé (Gs) and its F_1 and F_2 populations.

	Mean		Variance			
	obs.	calc.(1)	T(2)	G	E	gvc %
P_1(Zp)	17.0	—	3.3	—	3.3	
P_2(Gs)	76.0	—	52.4	—	52.4	
F_1(Gs x Zp)	30.1	35.9	64.0	50.9	13.1	23.7
F_1(Zp x Gs)	33.8	35.9	83.0	69.9	13.1	24.7
F_2(Zp x Gs)	31.2	35.9	64.0	50.9	13.1	22.9

(1): calc.--$(P_1 x P_2)1/2$; (2): T-- total; G-- Genetic, E-- enviromental.

Table 3: Differences between means for pericarp thickness of perennial teosinte (Zp), Gaspé (Gs) and its F_1 and F_2 populations.

Comparison	DF	Sig.(1)
P_1 vs. P_2	38	*
P_1 vs. F_1(Zp x Gs)	68	*
P_1 vs. F_1(Gs x Zp)	68	*
P_1 vs. F_2(Zp x Gs)	168	*
P_2 vs. F_1(Zp x Gs)	68	*
P_2 vs. F_1(Gs x Zp)	68	*
P_2 vs. F_2(Zp x Gs)	168	*
F_1(Zp x Gs) vs. F_1(Gs x Zp)	98	NS
F_1(Zp x Gs) vs. F_2(Zp x Gs)	198	NS
F_1(Gs x Zp) vs. F_2(Zp x Gs)	198	NS

(1): *--significant at 5% level, NS--no significant.

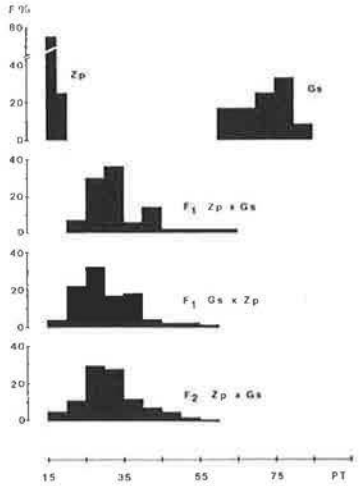

Figure 1: Frequency distributions for pericarp thickness (um) (PT) in perennial teosinte (Zp), Gaspe (Gs) and its F_1 and F_2 populations.

kernels (F_1 pericarp) developed on F_1 plants of this interspecific cross, are wholly enclosed in fruit cases such as perennial teosinte. Despite this fact there is thickness segregation, whereby, as was reported previously (MNL 59:68, 1985), pericarp thickness segregates independently from naked/enclosed kernels.

In short, the results obtained point out that: 1) pericarp thickness is quantitatively inherited, 2) thin pericarp is partially dominant over thick pericarp, 3) there is predominance of genes with dominant and also with additive effect, and 4) metaxenia effect conditions trait "segregation" in F_1 generation.

L.M. Bertoia and J.L. Magoja

Perennial teosinte-Gaspé hybrids: more about multilayer aleurone

As reported in the last issue (MNL 59:69, 1985), in F_2 progeny from hybrids between perennial teosinte (*Z. perennis*) and Gaspé (*Z. mays* ssp. *mays*) we find multilayer aleurone kernels. This trait may be attributed to a spontaneous mutation, as neither perennial teosinte nor Gaspé has multilayer aleurone. Kernels with multilayer (2 to 6 cell layers) have aleurone with different thickness (45.6 to 143.2 um), and this thickness is not associated with layer cell number. Multilayer aleurone shows peculiarities which were not reported in maize earlier: cells are rounded, with non uniform size and thin walls. Multilayer aleurone, in some cases, invaginates inside underlying tissue.

In order to determine whether different types of aleurone have some influence on kernel proteins, protein and tryptophan contents were determined in F_2 kernels from hybrids between perennial teosinte and Gaspé (Table 1). As we can see the number of aleurone cell layers has no influence on protein quality, but it does on protein content: more cell layers increases protein content of the whole kernel, significantly. In order to associate this trait with other kernel qualitative characters, 108 F_2 kernels were classified, using the following traits: number of aleurone layers (ALN) single (1), single and double (2), multiple (3); endosperm hardness (EH) floury (1), predominantly floury (2), intermediate (3), predominantly flint (4), flinty (5); pericarp color (PC) pale (1), pale brown (2), brown (3), dark brown (4); pericarp surface (PS) smooth (1), intermediate (2), wrinkled (3).

Data in Table 2 show that the number of aleurone layers is strongly associated with PC and PS: multilayers appear more frequently in kernels with dark pericarp and wrinkled surface. In addition ALN is inversely associated with EH: multilayer appears frequently in kernels with a higher ratio of floury endosperm.

Table 1: Protein and thryptophan content of F_2 kernels (Zp x Gs) with different aleurone layer cell number

Type of aleurone	Defatted whole kernel: Protein (%)	Defatted whole kernel: Thryptophan (g/100g prot.)
1- Single layer	22.6	0.4
2. Single and double layer	26.2	0.4
3. Multilayer	27.1	0.3

Table 2: Correlation coefficients between qualitative traits of F_2 kernels (Zp x Gs)

	ALN	EH	PC	PS
ALN	—	-0,32**	0.34**	0.31**
EH		—	-0.43**	-0.39**
PC			—	0.46**
PS				—

** Significant at 1% level.

Table 3: Segregation of aleurone layer number between 208 F_2 kernels (Zp x Gs).

Endosperm type	Aleurone layer number: Single	Aleurone layer number: multiple	X^2 for 3:1 ratio	P
Normal	140	51	0.294	0.50-0.70
Floury	12	5	0.053	0.70-0.90
Total	152	56	0.205	0.50-0.70

A new classification from a sample of 208 F_2 kernels according to their aleurone layer type (single or multiple) and their endosperm type (hard = normal or completely floury) gave the data in Table 3.

According to these results we can deduce that a single recessive gene would condition multilayer

aleurone trait and its spontaneous appearance may be due to mutagenic effect of hybridizing maize and teosinte as Mangelsdorf (1958) has postulated. Finally, it is important to remark that multilayer trait segregates independently from endosperm type (floury or flinty).

L.M. Bertoia and J.L. Magoja

Perennial teosinte-Gaspé hybrids: storage proteins of different phenotypes

Plant phenotype is closely associated with endosperm protein content in the progeny of hybrids between perennial teosinte and maize. Maizoid plants have lower protein content than those of intermediate phenotype, and the latter have a lower level than teosintoid phenotype (see MNL 59:69, 1985).

With the aim of establishing the existence of other ways of associations between plant morphological phenotype and kernel storage proteins, a more detailed study was done of F_2 and F_3 samples derived from perennial teosinte (*Z. perennis*) crossed with Gaspé. Twelve plants were chosen: 1 to 4 with teosintoid phenotype, 5 to 8 with intermediate phenotype and 9 to 12 with maizoid phenotype. Endosperm was separated from kernels of each one of these plants, and was used to fraction storage proteins in accordance with their solubility (Landry-Moureaux, 1970). Alcohol-soluble fraction (zein) was used to separate molecular components through polyacrylamide gel electrophoresis, following the Sastry and Virupasksha (1967) technique. Results obtained are shown in Table 1 and Figure 1. Protein pattern variability between plants is considerable. Not only are parental patterns partially recovered but also intermediate protein patterns and transgressive ones are produced (see Table 1). This fact clearly points out the enormous variability engendered as a product of germplasm recombination of distant species. At the storage protein level, this variability corresponds with that morphological variability that has been largely stated in previous articles published in this Newsletter. Zein electrophoretic patterns do not show such a high variability (see Figure 1). This fact may be due, perhaps, to the absence of a great differentiation between parental species for genes that code for zein (MNL 55:62) and the main differentiation may reflect the action of regulatory genes more than structural genes.

A basic data matrix (BDM) was built with the data present in Table 1 and Figure 1, and was used to calculate the distance matrix among the 12 individuals studied, which constitute the 12 OTU's. The phenogram shown in Figure 2 was derived by "Mean Taxonomic Distance" applying the UPGMA method.

Table 1: Endosperm protein pattern of Z. perennis (Zp), Gaspé (Gs) and F_2-F_3 selected plants derived from Zp x Gs. SS: saline; Z: zein; G_1: glutelin-1; G_2: glutelin-2; G_3: glutelin-3.

	Soluble Nitrogen % of total													
		F_2 - F_3 plants												
Fraction	Zp.	1	2	3	4	5	6	7	8	9	10	11	12	Gs.
SS	1.8	1.7	3.0	1.6	5.2	5.4	5.4	6.5	7.0	2.6	6.6	4.0	3.6	5.7
Z	57,1	47.4	51.0	50.7	49.4	37.1	50.8	44.8	45.7	55.0	37.2	49.8	66.5	51.9
G_1	20.7	23.0	24.4	17.5	11.6	22.2	13.9	16.8	18.1	11.9	16.7	13.2	14.1	12.3
G_2	3.3	3.3	2.2	3.2	3.0	4.1	3.8	3.7	6.5	2.7	3.0	3.6	3.2	9.6
G_3	9.2	13.8	15.8	12.5	12.0	15.6	12.2	12.6	15.2	14.4	21.3	12.0	11.7	13.0
Protein %	24.1	24.7	24.3	24.6	25.3	20.6	24.5	19.9	18.9	19.6	19.9	24.3	22.8	14.4

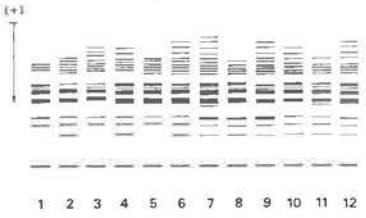

Figure 1: Polyacrylamide gel electrophoretic patterns of zein of F_2- F_3 selected plants derived from Zp x Gs.

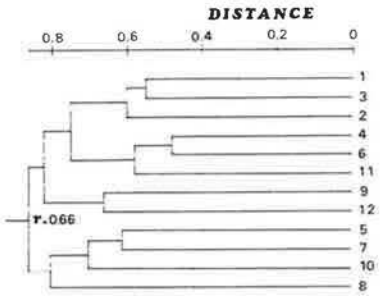

Figure 2: Phenogram of 12 OTU'S resulting from the UPGMA cluster analysis of the OTU x OTU distance matrix. r= cophenetic correlation coefficient.

In some cases storage proteins keep a tight relation with the plant morphological phenotype. This occurs in the cluster formed by 1, 2 and 3 teosintoid plants. Plants 4, 6 and 11 belong to different morphological groups, denoting that a partial character recombination has taken place. Plants 9 and 12 are maizoid and are linked between them by their relation to storage proteins. In the 5, 7, 10 and 8 cluster there are 3 plants with intermediate morphological phenotype and one (10) maizoid, pointing to recombination.

The results obtained show in some cases the existence of a height association between plant clustering by morphological traits and by storage proteins. Otherwise no congruence exists in clustering. Therefore it may be inferred that specific traits, both morphological and biochemical, which differentiate perennial teosinte from maize, are mainly inherited in an independent way. Bearing in mind that perennial teosinte and maize are the basic species of genus *Zea* (see MNL 59:61, 1985) results presented constitute another evidence that these species carry all the genetic information necessary to produce the main portion of possible variants in storage proteins, and that are in some instances the patterns of the remaining taxa.

Liliana Ferrari and Jorge L. Magoja

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Fertilization and seed production with pollen from cultured tassels

We previously reported (MGCNL 59:73, 1985) that 20% of normal spikelets of cultured maize tassels extrude anthers with elongated filaments. We report here the procedure for recovering the pollen from these extruded anthers, some characteristics of the pollen and its germination, and also fertilization and seed production with in vitro-derived pollen.

After 16-25 days of tassel culture (MGCNL 59:72, 1985), extruding anthers of both cv. Oh43 and Se60 tassels were excised separately, dried on Whatman #1 filter paper in a desiccator containing a mixture of dehydrated silica gel and $CaCl_2$ for ½ to 5 h. Fresh pollen, collected within ½ h after drying of the anthers, was used for measuring the characteristics listed in Table 1. Germination percentage and tube growth rate were calculated from pollen germinated on nutrient agar medium (Can. J. Bot. 43:779, 1965). As indicated, in vitro-produced pollen and greenhouse pollen were similar in the measured parameters.

Table 1. Characteristics of pollen from cultured tassels and greenhouse grown plants.

Genotype	Pollen source	Pollen diameter (μm) (X̄+S.E., N=1000)	% germination (X̄+S.E.)	tube growth rate (μm/min)
Se60	in vitro	88.29 + 0.45	68.76 + 2.18 (3244)	4.95
	in vivo	90.61 + 0.38	72.40 + 0.38 (3244)	5.05
Oh43	in vitro	96.49 + 0.50	50.02 + 1.74 (2920)	4.96
	in vivo	96.87 + 0.44	52.73 + 0.46 (2237)	5.00

The in vitro-produced pollen also germinated on receptive silks. For observations on in situ germination, ears with receptive silks were pollinated with fresh pollen and the general procedure of fixing, clearing, staining with aniline blue and fluorescence microscopy (Naturwissen. 44:1, 1957) was followed. No observable differences were detected between in vitro pollen and native pollen during germination and tube growth in or on silks.

The in vitro-derived pollen also fertilized ovules and produced viable seed. Female parent plants were grown in plastic pots in greenhouse, detasseled and bagged prior to the emergence of silks. The plants were moved to the laboratory and ears were pollinated with pollen collected after ½ to 5 h of drying of extruded anthers under monitored conditions in March and April, 1985.

All ears pollinated with pollen collected within 5 h after drying of anthers produced kernels. But the number of kernels produced per ear declined with increased drying time of anthers. Nevertheless, with generous application of fresh pollen, collected within 1 h after drying of anthers, up to 300 kernels per ear were produced. Kernels had 100% germinability and yielded normal, fertile, genetically true plants during summer, 1985.

The demonstration that the cultured tassels produce normal, germinable and viable pollen, and that the pollen is essentially similar to native pollen, suggests that the system has considerable potential for both basic and applied research.

D. R. Pareddy, R. I. Greyson and D. B. Walden

Generation of embryoids from primary and secondary anther cultures

Anther culture, via the production of haploids, is used to produce isogenic diploid lines. Because corn has responded poorly to attempts at anther culture, a comparison of two different methods was undertaken: the traditional method of anther culture (Primary Anther Culture, PAC) and a second method which produces anthers from tassels grown in liquid culture (Secondary Anther Culture, SAC).

Tassels for PAC were taken from Se60 (Seneca 60, a commercial sweet corn) plants at the 13th leaf stage. Segments of the tassel containing mainly anthers with uninucleate pollen (aceto-carmine stain) were sterilized with 20% Javex for 20 minutes and transferred to sterile dishes. The larger set of anthers from each spikelet was removed and transferred to 60 mm sterile plastic petri dishes containing test medium. After 24 anthers had been added, the dishes were sealed with two layers of Para-film and incubated in the dark at 8 C for 8 days. After this cold treatment the petri dishes were moved to 25 C in the dark for an additional 5 to 12 weeks.

For SAC, tassels of Se60 were first cultured on a modified liquid medium (0.1 M sucrose) as reported by Pareddy and Greyson (MGCNL 59:72-73, 1985). After 10 days of culture the anthers in the spikelets were carefully removed and placed, 24 to a dish, in sterile plastic petri dishes. Several anthers were removed for staging and care was taken to ensure that the anthers contained uninucleate pollen. The dishes were sealed and cold treated for 8 days at 10 C in the dark, then transferred to 25 C in the dark for 5 to 12 weeks.

The medium used for anther culture was a basic Yu-Pei (Y-P) medium (Genovesi and Collins, Crop Sci. 22:1137-1144, 1982) and the variations of that medium were as follows: Y-P (basic Y-P with TIBA, no agar, no charcoal); Y-P, C (Y-P with charcoal).

After 6 to 8 weeks of culture, embryoids or callus began to break out of the anthers. Usually, only one embryoid was produced per anther although a few anthers have produced as many as 3 embryoids. These embryoids were white, densely packed clusters of cells with an ovoid or spherical shape. They

ranged in size from 1 mm to 5 mm. The larger embryoids developed roots, shoots or continued to grow as callus when moved to transfer medium. The success of anther culture from Se60 anthers taken directly from the plant (PAC) and anthers taken from tassel culture (SAC) are compared in Table 1.

Table 1. Embryoid producing responses of cultured anthers to Primary Anther Culture (PAC) and Secondary Anther Culture (SAC).

	Media	# anthers	# embryoids	# tassels	# embryogenic tassels
PAC	YPC	1448	10 (0.7%)	7	4 (57%)
	YP	4729	30 (0.63%)	18	10 (55%)
SAC	YPC	404	0		0
	YP	2008	13 (0.65%)	33	12 (36%)

The average yield of embryoids is given as a percentage (# of embryoids/# of anthers). The individual yields, however, varied greatly, from .3% to 3.5% for different tassels, and many tassels yielded no embryoids at all. This is likely due to variability of some aspect of the mother plant at the time of culture as nutrition, lighting and stress all are reported to play a role in growth of the plant. Although the SAC anthers yielded approximately the same percentage of embryoids as the PAC anthers, the maximum yield for a given experiment was only 1.3% whereas maximum yield for PAC was 3.5%. Looking at the percentage of embryogenic tassels, it can also be seen that a higher percentage of tassels responded in the PAC system. The use of charcoal in the culture medium did not seem to influence the yield for PAC, however, in the SAC system the sample size for the charcoal medium may have been too small. Our yield of embryoids is comparable to some researchers (Genovesi and Collins, ibid.), but is lower than that of others (Ting, Yu and Wan-Zhen, Plant Science Letters 23:139-145, 1981). We conclude that despite the low yield of embryoids from SAC, it appears to be at least as good in yield as is PAC.

R.I. Greyson and J.D. Dunlop

A change in the humidity level alters the response of seedlings to prolonged temperature stress

Maize embryos, seedlings and mature plants respond rapidly and dramatically to the stress of a rapid increase to supraoptimal temperature (heat shock; hs) by (1) the new and/or enhanced synthesis of a group of polypeptides, the heat shock polypeptides (hsps) and (2) the reduced synthesis of polypeptides made at a control temperature (Baszczynski MGCNL 56:111, 1982).

When seedlings are maintained under temperature stress conditions for prolonged periods, synthesis of hsps gradually decreases and the rate of diminution is hsp specific (Rockwell, Lehman and Nebiolo MGCNL 59:78, 1985; Baszczynski, Ph.D. Thesis, 1985). Reports of the time required for the disappearance of hsp synthesis following prolonged temperature stress vary substantially. P. Cooper and T.-H.D. Ho (Plant Physiol. 71:215, 1983) observed the complete disappearance of hsp synthesis in excised roots after 10 hours of temperature stress while Rockwell et al. (1985), working with plumules of intact seedlings (Oh43), observed synthesis of some hsp classes following 18 hours of temperature stress. The disparity may reflect a tissue or genotype specific nature for the response or it may be due to the effects of environmental factors other than temperature. We attempted to determine if altering the humidity level would change the response to prolonged temperature stress.

Intact seedlings (Oh43) were maintained at 25 C (control) under conditions of low humidity (dry; seedlings were maintained on almost dry filter paper) or shifted to 42 C under conditions of high humidity (humid; seedlings were maintained on moist filter paper) or low humidity. At two hour intervals plumules of intact seedlings, incubated in ^{35}S-methionine (0.01 uCi/uL) for two hours, were excised and the proteins extracted in 200 mM Tris-HCl pH 7.4, 5% SDS, 7.5% 2-mercaptoethanol, 1 mM PMSF. Polypeptides were subjected to SDS-PAGE and following fluorography the patterns of newly synthesized polypeptides were compared. The identification of hsps was confirmed using antibodies prepared against either the low molecular weight (18kD) or high molecular weight (73 kD - 89 kD) group of hsps (Baszczynski MGCNL 58:134, 1984). Following labelling with ^{35}S-methionine as described above, proteins were extracted from plumules in 50 mM Tris, pH 7.2, 0.15 M NaCl, 1% NaDOC, 1% Triton X-100 and 0.1% SDS and immunoprecipitated with either antibody type. Immunoprecipitates were subjected to SDS-PAGE and fluorography and the presence of hsps among newly synthesized polypeptides was noted.

The level of hsp synthesis was not dramatically reduced during the first 8 hours of temperature stress in plumules maintained under humid or dry conditions. By 10 hours, no further synthesis was observed of most hsp classes in plumules incubated under humid conditions; under dry conditions, plumules continued to synthesize all hsp classes for at least twelve hours, although in reduced amounts. By 24 hours the pattern of polypeptide synthesis in plumules under humid conditions resembled that of control plumules but plumules maintained under dry conditions exhibited a new pattern of polypeptide synthesis which resembled neither that of control nor heat shock plumules.

The difference in the rate of disappearance of heat shock polypeptide synthesis under conditions of low and high humidity may reflect a difference in the physiological condition of the seedlings. We are examining the effect of the humidity level on the ability of seedlings to survive prolonged temperature stress.

Carol A. B. Rees, D. B. Walden,
Bruce H. Rockwell[1] and Christine M. Nebiolo[1]

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Oncogene-related sequences in maize

The acutely oncogenic retroviruses are capable of rapidly inducing a variety of leukemias and sarcomas in appropriate animal hosts. The retroviral genes responsible for these transformations arose from sequences of the respective host. These transforming genes are termed "oncogenes" whereas their apparently normal, cellular counterparts are referred to as "proto-oncogenes". Proto-oncogenes are transcribed and translated in young, growing tissues and are considered essential for growth and differentiation (Pardee, A. B. et al., Cellular oncogenes, growth factors, and cellular growth control, pp. 21-29, 1985).

Oncogene related sequences (proto-oncogenes) have been reported in many vertebrate species as well as in several invertebrate (fruit fly and nematode) and lower plant species (yeast and slime mold). This report extends the species to have oncogene related sequences to include *Zea mays*.

Nuclei were isolated from 5 day old Oh43 maize seedlings as described by Luthe and Quatrano (Plant Physiol. 65:305, 1980). Lysis of nuclei and isolation of DNA was carried out according to Zimmer and Newton (Maize for Biol. Res., pp. 165-168, 1982). The DNA was digested overnight with 4 times excess of ApaI, HindIII, or EcoRI using the buffer system recommended by the manufacturer (Boehringer-Mannheim). Digested maize DNA was size fractionated on 0.8% agarose gels. To serve as positive controls, avian and human EcoRI digested DNA was run on each gel. DNA from the gels was blotted to nitrocellulose membranes (BioRad, 0.45 um) using standard Southern transfer procedures. Viral Kirstein ras oncogene (v-Ki-ras) and viral src oncogene (v-src) were used to probe the blots. Prehybridization and hybridization procedures were adapted from Wahl et al. (PNAS 76:3683, 1979). To increase the likelihood of hybridization of the probes to the maize DNA, 30% formamide was used instead of 50% formamide and the hybridization temperature was 37 C.

DNA from human and avian tissue that was used as a positive control produced hybridization patterns similar to those previously reported (McGarth, J. P., et al., Nature 304:501, 1983; Gibbs, C. P., et al., J. Virol. 53:19, 1985). Both oncogene probes detected homologous sequences in maize DNA. The blots probed with v-Ki-ras produced a complex hybridization pattern (at least 4 bands in each lane of digested maize DNA - ApaI, HindIII, and EcoRI). This suggests that there is more than one gene in maize homologous to v-Ki-ras. (It is unlikely that a single gene would have multiple ApaI, HindIII, and EcoRI restriction sites.) ApaI digested DNA showed 4 bands at approximately 7.5, 6.2, 4.3 and 2.6 kb. HindIII and EcoRI digested maize DNA had 4 bands between 4.3 and 1.0 kb. These results are similar to those reported from animal studies: vertebrate genomes contain several ras proto-oncogenes. The v-src hybridized blot had 1 prominent band in each lane of maize digested DNA: ApaI, 6.6kb; HindIII, 1.9 kb; and EcoRI, 2.8 kb. This suggests that the nuclear genome of maize contains a single sequence homologous to v-src. From hybridization conditions, it was determined that these maize sequences have at least 70% homology to the viral oncogene probes (Howley, P. M., et al., J. Biol. Chem. 254:4876, 1979).

R. Zabulionis, D. B. Walden and B. G. Atkinson

Confirmation of the isolation of a mRNA coding for a small heat shock polypeptide

Last year (MGCNL 59:76, 1985), we reported on the isolation of a poly (A^+) RNA size fraction (630-850 nucleotides) from shoots of seedlings (Oh43) subjected to a 1 hour temperature shift (heat shock; hs), which upon translation in a cell-free system yielded a heat shock specific polypeptide of 18 kD. We have obtained further evidence that this polypeptide belongs to the 18 kD heat shock polypeptide class (hsp class) described previously (Baszczynski et al., Can. J. Biochem. 60:569, 1982; Baszczynski, MGCNL 56:111, 1982).

Polyclonal antibodies were raised against the 18 kD hsp class using the method outlined by Baszczynski (MGCNL 58:135, 1984). These antibodies were used to recognize 18 kD heat shock polypeptides among translation products of RNA from different sources.

Translation products (25 ul) were treated with 0.5% (1.25 ul) of ribonuclease A (1 ug/ul) and 0.5% (1.25 ul) of ribonuclease T1 (1 U/ul), for 15 mins. at 30 C. Antiserum (50 uL) and immunoprecipitation buffer (12.5 uL; 10 mM $NaPO_4$, pH 7.5, 150 mM NaCl, 10% NaDOC, 10% triton X-100) were added. After 20 mins. at room temperature 100 uL of a preparation of *Staphylococcus aureus* cells was added and the mixture was incubated for a further 20 mins. at room temperature. The samples were spun for 3 mins. in a microfuge and the pellets were washed 3 times in wash buffer (10 mM NaPO4, pH 7.5, 150 mM NaCl, 1% NaDOC, 1% triton X-100). After the final rinse, the pellets were suspended in protein extraction buffer (200 uM Tris pH 7.5, 5% SDS, 7.5% 2-mercaptoethanol, 1 mM PMSF) and

placed in a boiling water bath for 3 mins. The samples were spun in the microfuge for 3 mins. and the supernatants were subjected to SDS-PAGE and fluorography.

It was demonstrated first that these polyclonal antibodies recognize only heat shock specific polypeptides amongst translation products of total RNA from shoots of seedlings subjected to heat shock and that they do not recognize any polypeptides synthesized in the cell-free system by RNA isolated from shoots of control seedlings. The antibodies were then used in immunoprecipitation experiments with translation products of the poly (A^+) RNA size fraction (630-850 nucleotides) isolated from shoots of control and heat shock seedlings. The heat shock specific 18 kD translation product was immunoprecipitated using these antibodies. This confirms our suggestion that this polypeptide belongs to the heat shock polypeptide class previously described and it confirms the presence of a mRNA coding for a heat shock polypeptide among the RNA molecules of this size class (630-850 nucleotides). [*Note of correction:* In last year's newsletter (Rees, MGCNL 59:76, 1985) there is an error in one of the buffer recipes: Transfer buffer consisted of 10 mM Tris pH 7.6 500 mM NaCl, not 500 mM Tris pH 7.6.]

Carol A. B. Rees and David B. Walden

Heavy metals induce changes in polypeptide patterns of plumules and radicles

As reported last year (MGCNL 59:77, 1985), selected agrichemicals may elicit a variety of responses in five day old maize plumules and/or radicles, as measured by ^{35}S-methionine uptake and protein synthesis. We have extended this study to examine other possible stress inducing factors.

Maize 5 day old seedlings were treated with one of four heavy metals ($ZnSO_4$, $PbNO_3$, $CuSO_4$, $CdCl_2$) or salt (NaCl). Each heavy metal or salt treatment ranged in concentration from $2x10^{-8}$M to $5x10^{-1}$M. Control seedlings were placed in double-distilled water. Plumules or radicles were treated for three hrs. Proteins were extracted, precipitated with TCA, and incorporated radioactivity was measured using a liquid scintillation counter (C. L. Baszczynski et al., Can. J. Biochem. 60:569-579, 1982). A dose response was seen as both radicles and plumules showed increasing incorporation of ^{35}S-methionine into protein with decreasing concentrations of the factor. Copper sulphate and cadmium chloride showed the greatest inhibitory effect on ^{35}S-methionine incorporation. Least inhibition was seen with sodium chloride.

Two dimensional IEF-SDS PAGE and fluorography were carried out at the highest non-toxic concentration of treatment using standard techniques (C. L. Baszczynski et al., op. cit.) to examine the impact of the treatments on protein synthesis. Preliminary analysis of the fluorograms showed some differences between treated plumules or radicles and controls. Radicles and plumules treated with $2x10^{-3}$M cadmium chloride and radicles treated with $1x10^{-1}$M zinc sulphate showed synthesis of apparent heat shock or "stress" proteins (hsps) of molecular weight 18, 23, 73, 76, 84, and 89 kD. Radicles treated with $1x10^{-1}$M $PbNO_3$ showed an increase in synthesis of higher molecular weight hsps and possibly hsps of 18 and 23 kD. Treatment of plumules with $1x10^{-1}$M zinc sulphate or $2x10^{-3}$M copper sulphate did not induce a stress response. Treatment of radicles and plumules with varying concentrations of sodium chloride did not alter the protein pattern as compared to controls.

Since the pH of the heavy metal solutions used as treatments ranged from 3.4 to 6.1, it was possible that pH alone could be responsible for the change in the polypeptide patterns observed on fluorograms of 2D gels. To examine this possibility further, the plumule/radicle system was treated with an assortment of organic and inorganic buffers ranging in pH from 3.0 to 8.8. As compared to the dd H_2O controls, the incorporation of ^{35}S-methionine into protein was inhibited to some extent by all buffer solutions, although the inhibition was not as great as that seen with the heavy metals. No significant differences in incorporation were observed between buffers across the range of pH's for both plumules and radicles. Examination of fluorograms showed no major differences in polypeptide patterns among any buffer treatments and controls (dd H_2O), for radicles and plumules.

These results suggest that differences in the polypeptide pattern as visualized by 2D IEF-SDS PAGE and fluorography that were seen between treatments and controls were due to the treatments alone, and not due to changes in the pH of the solutions within the ranges tested. Thus high salt concentration or inorganic and organic buffers of varying pH's do not seem to significantly alter polypeptide patterns with respect to controls. However, from preliminary analysis it appears that treatment of radicles or plumules with some heavy metals will elicit varying degrees of a heat shock or "stress" response.

A. Gullons, A. Mackenzie and D. B. Walden

Use of leaf discs to monitor protein synthesis under field conditions

In recent Newsletters, we have reported a series of studies in which a physical, chemical or biological 'stress-inducing' factor has been examined, usually in a 5 day old plumule and/or primary radicle, grown under stringent laboratory conditions. Altered or enhanced protein synthesis as revealed by fluorography has been our main criterion for the assignment to a factor of the capacity to induce stress. We have

shown also that the laboratory techniques can be adapted to the study of certain other tissues of maize to address problems of development, etc.

This report outlines attempts to extend our studies to field grown material, i.e., to monitor the growth of a tissue or organ during the growing season. During the fall of 1984, in our nursery we created two plots (each approx. 15 x 20 m), designating one a minimum productivity plot and the other a maximum productivity plot. A third plot was selected as a control and left unaltered (normal nursery maintenance). In the minimum productivity plot the top soil was mixed with an equal amount of bank-run gravel; in the maximum plot, the top soil was enhanced with liberal applications of manure and premium top soil, to provide the best texture, etc. we could produce. Management practices (fertilization, irrigation, etc.) in 1985 were consistent with the intent of the plot, i.e. maximum or minimum (stressed) growth.

A selection of inbreds and some of their reciprocal hybrids were sown at two planting dates and thinned to a density of 60,000 plants per hectare in each plot. Measurements (height, number of leaves, etc.) were recorded several times through the growing season to provide a 'base-line' of growth. Fortunately we had a reasonable growing season such that the expected differences were obtained among plots, planting dates and cultivars.

Two cm (diameter) leaf discs, exposed to ^{35}S-methionine for 1.5 h in situ, provided leaf material to yield TCA precipitable protein in sufficient quantity to monitor uptake and to run two dimensional gels followed by appropriate fluorography. One to several leaves were sampled from representative plants of each cultivar in each planting every two weeks from mid-June until mid-September. The field techniques (MGCNL 59:75, 1985) and the laboratory techniques (Baszczynski et al., Can. J. Biochem. 60:569, 1982) required little modification.

This study will require at least the 1986 growing season before convincing data analysis can be offered. We are particularly interested in examining proteins that may be unique to cultivars, those that may have a role in heterosis, and those that are of developmental significance. We do not know yet whether this system (protein isolation - electrophoretic separation - fluorographic analysis) is sensitive enough to address any of the three questions. Our preliminary data from the 1985 fluorograms do demonstrate qualitative differences among cultivars in the three performance plots, including some inbred-hybrid comparisons. Interestingly, no qualitative differences have been found between planting dates of the same cultivar/plot when the leaf is of the same age, say 60 days. If such fidelity is the rule, differences among genotypes should be easier to detect.

D. B. Walden and T. G. Crowe

Seasonal variability in developing embryos

In previous reports (MGCNL 58:136, 1984 and 59:77, 1985) we have outlined our attempts to characterize molecular changes occurring during embryogenesis. Results obtained over the past 3 years permit a preliminary assessment of the amount of variation attributable to certain parameters inherent in this project. Ears were collected from field grown plants at 5 day intervals between 20 and 40 days post-pollination in 1983 and between 20 and 50 days post-pollination in 1984 and 1985. An additional planting in a controlled environment with minimum temperature of 60 F and maximum temperature of 85 F was made during the spring of 1985 with samples collected between 20 and 40 days post-pollination. Two inbreds, Oh43 and M14, and their reciprocal hybrids were included in this study. At each sampling date embryos were collected for determination of dry weights and individual kernels were labelled with ^{35}S-methionine as described by Kriz (MGCNL 56:14, 1982).

The profile of change in dry weight as a function of age appeared similar for all plantings and genotypes. A 7.5 fold average increase was observed between 20 and 40 days post-pollination which subsequently leveled off between 45 and 50 days post-pollination. Mean maximum dry weight values per embryo of all plantings for each genotype were Oh43 14.5 $\pm$ 4.0 mg, M14 15.2 $\pm$ 3.2 mg, Oh43/M14 16.2 $\pm$ 5.3 mg, M14/Oh43 14.8$\pm$5.2 mg. The largest range observed for all samples represented a 2.3 fold difference between values obtained for M14/Oh43 from field grown material in 1984 and greenhouse grown material in 1985. No two plantings, however, consistently yielded the greatest interseasonal range for all genotypes. Embryos produced on plants grown in the greenhouse in 1985 were invariably larger than those obtained from other plantings at all sampling dates. These plants also appeared to mature more rapidly, possibly indicating an accelerated rate of development under these near optimal conditions.

Incorporation of ^{35}S-methionine was calculated as a function of dry weight with values ranging between 16,200 cpm/mg and 483,900 cpm/mg. Variation on the order of 85% of the mean was observed among replicate samples, making difficult any determination of genotypic or developmental differences on this basis. This variation may be attributable in part to the method of label delivery (injection using a Hamilton syringe). For example, label may seep from the opening in the kernel following withdrawal of the syringe, possibly affecting the concentration to which individual embryos are exposed. Values for peak incorporation were found to vary across plantings by a factor of 4 and to a lesser extent between genotypes, by a factor of 2.5. Variation was also encountered with respect to the age at which maxi-

mum incorporation was observed. Samples collected from field grown material in 1983 and 1985 showed peak values between 35 to 40 days post-pollination and 20 to 30 days post-pollination respectively for all genotypes. Maximum incorporation for field grown material in 1984 occurred between 20 and 40 days post-pollination, varying among genotypes. This increased variation may have been a result of different degrees of damage suffered by individual plants following a severe hailstorm 17 days after the first pollinations were made. The most consistent results were obtained from the plants grown under controlled environmental conditions in 1985: all genotypes showed maximum incorporation at 25 days post-pollination. This variation between plants for the age at which peak incorporation is observed may indicate subtle differences, due to environmental conditions, in the metabolic state of the embryos at the time of collection.

Analysis of newly synthesized proteins completed for material collected in 1983 and 1984 also reveals some degree of seasonal variability in development. The order and types of changes which are observed on two-dimensional fluorograms during the course of embryogenesis appear to be consistent for both plantings. The specific age at which these changes are observed may vary by as much as 5 days. Completion of the analysis of the patterns of newly synthesized proteins for material for both plantings in 1985 should provide further insight into the extent of seasonal variability as reflected in protein synthesis and its relationship to dry weight and ^{35}S-methionine incorporation.

J. G. Boothe and D. B. Walden

Stamen development and microsporogenesis in cultured ear shoots

We reported previously (MGCNL 59:74-75, 1985) on the differential flower development in cultured ear shoot primordia. We noted that the optimum expression of this phenomenon depended on the concentration of kinetin (K) in the medium and the developmental stage of the initial explant. With K = 10^{-7}M, young (<10 mm long) ear shoots produced primarily male flowers whereas older shoots produced only female flowers. Based upon these observations, we have studied stamen development of cultured ear primordia more closely. Our observations are from experiments with Seneca 60 (Se60) ears under conditions which favor male flower development. These included the same medium as reported (MGCNL 59:74-75, 1985) with the following specific conditions: K = 10^{-7}M; sucrose = 6% w/v; flasks maintained without shaking on a white reflective surface in a lighted incubator.

After 24 hours of culture, 2-3 explants were sacrificed at 2-day intervals and fixed in Carnoy's fixative. Analyses of flower development and stamen growth were recorded and cytological observation made from anther squashes.

As documented in Figure 1, both upper and lower flowers at 16 days possess three stamens and lodicules and are morphologically similar to those that develop normally in the tassel. The dimensions of the stamens at different times during the experiment are summarized in Table 1.

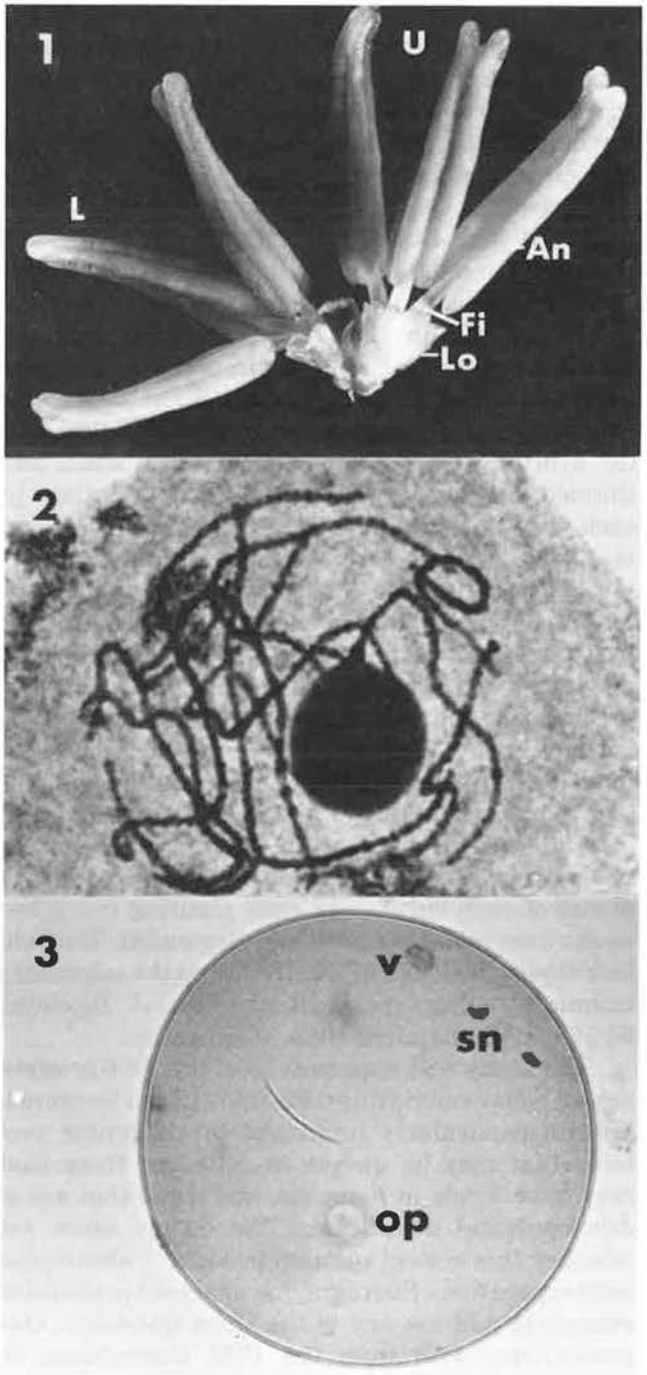

Figure 1. Differentiated anthers from the upper and lower flowers of an ear shoot spikelet after 16 days of culture. (An = anthers; Fi = filament; L = lower flower; Lo = lodicule; and u = upper flower). 12x

Figure 2. Pollen mother cell (pachytene) after 8 days of culture. 1600x

Figure 3. Mature pollen after 20 days of culture of ear shoot. (op = operculum; Sn = sperm nuclei; and v = vegetative nucleus). 880x

Table 1. Mean lengths (mm) of stamens (n = 100).

# of days in culture	Total length ± S.D.	Anther length ± S.D.	Filment length ± S.D.
8	2.65 ± 0.41	2.22 ± 0.31	0.43 ± 0.18
10	2.94 ± 0.38	2.45 ± 0.35	0.50 ± 0.11
12	3.42 ± 0.32	2.60 ± 0.24	0.82 ± 0.19
14	4.43 ± 0.36	3.10 ± 0.31	1.33 ± 0.33
16	4.58 ± 0.28	3.34 ± 0.39	1.24 ± 0.27
18	5.77 ± 0.58	3.56 ± 0.41	2.21 ± 0.40
20	5.98 ± 0.45	3.82 ± 0.44	2.16 ± 0.33

After 7 days in culture, anther sporogenous tissue (squashed and stained according to the procedure of Kindiger and Beckett, (Stain Tech. 60:265, 1985) was, for the most part, in early prophase of meiosis. The uninucleate microspore stage was reached after 10-12 days and binucleate pollen was found in cultures 13-18 days old. While we have not yet demonstrated that pollen from these cultured ear shoots will germinate and produce seed, we see no reason at present to expect failure. (*NOTE OF CORRECTION:* All references to sucrose concentration in our earlier note, MGCNL 59:74-75 (1985), should be expressed as % w/v and not M. Thus the values in Table 1 are 0%, 3%, 6%, 9% and 12%. Table 2 values are 2% and 6% and Table 3 value is 6%.)

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Resistance to charcoal rot in some elite maize populations

Charcoal rot (*Macrophomina phaseolina* Tassi G. Goid) of maize is a serious problem under light soil and post-flowering water stress conditions (A Compendium of Corn Diseases. 2nd ed., M.C. Shurtleff, ed.). Since the disease is soil-borne in nature and of senescence stage, chemical control measures show little promise and must be replaced by a strong resistance breeding programme. Genetic differences for resistance have been observed in maize for this disease (Payak and Sharma, Crop Improvement 6:75-85, 1979; Ivaschenko and Verenik, Nauchnoteckn. Byul. Vses. Sel.-genet. Inst. No. 2, 65-69, 1982, Plant Breeding Abstr. 53:6258, 1983). A judicious exploitation of such variabilities lies in identifying the source of resistance and its introgression into the breeding programme. In the present study 25 elite, both exotic (obtained from CIMMYT, Mexico) and indigenous germplasms, were evaluated for charcoal rot to detect a source of resistance to this disease.

The materials were evaluated in 2 years (1981 and 1982 monsoon) with 3 common entries in both years. The field used for the evaluation had sandy, loam soil with a past record of charcoal rot, and has been under maize cultivation for several years. The trial was laid using randomized complete-block design with four replications. Each plot consisted of 4 rows of 5m length, and the row-to-row and plant-to-plant distance was maintained at 75 and 22 cm, respectively. The data were recorded on the basis of percent of plants infected. The symptomatology and pure culture isolation of the pathogen confirmed the involvement of *Macrophomina phaseolina* in the disease. The statistical analysis was carried out using arcsine transformation.

Among the entries evaluated for both years, Across 7726, a CIMMYT population, showed the highest level of resistance (Table 1). The other promising materials were Poza Rica 7926, Across 7926, Tocumen (1) 7931 and Across 7835, four CIMMYT populations, and J684 and EH400175, two Indian populations. However, these were tested for only one year. The populations showing incidence below 5 percent were termed as resistant, and above 5 percent as susceptible.

Table 1. Reaction of the germplasm to charcoal rot.

Experimental variety	Name of population	Disease incidence (%) 1980	1981	Mean
1. Across-7726RE	Mezcla Amarilla	0.0	3.1	1.6
2. J54	Partap	3.7	3.2	3.5
3. Across-7635RE	Antigua Rep. Dominicana	5.5	7.6	6.6
4. Poza Rica 7926	Mezcla Amerilla	1.6	-	
5. Piura(1)7926	"	-	6.4	
6. Sanedja 7926	"	-	8.2	
7. Islamabad(1)7926	"	-	9.2	
8. Across 7926	"	3.2	-	
9. Jutiapa(1)7930	Blanco Cristallino-2	7.2	-	
10. Pirsabak(1)7930	"	7.2	-	
11. Sete Lagoas 7931	Amerillo Cristalino-2	-	8.1	
12. Tocumen(1)7931	"	2.0	-	
13. Poza Rica 7931	"	10.8	-	
14. Pichillingue 7931	"	-	14.5	
15. Across 7931	"	-	16.1	
16. Satipo(1)7931	"	-	18.2	
17. Satipo(2)7931	"	-	20.2	
18. Across 7835	Antigua Rep. Dominicana	3.2	-	
19. Suwan 8035	"	-	6.2	
20. Islamabad 8035	"	-	7.2	
21. Poza Rica 8035	"	-	7.2	
22. J 684	Navjot	4.6	-	
23. -	Sangam	5.7	-	
24. EH400175	Ganga-9	3.2	-	
25. EH 200174		-	7.9	
C.D. at 0.5		8.01	3.13	

The yield potential and maturity of the three materials which were evaluated for both years revealed about a 19% yield superiority of Partap, the indigenous composite, over Across 7726 RE, and 36% over Across 7635 RE. However, Across 7635 RE was earlier than Across 7726 and Partap by 3.5 and 4.5 days, respectively (Table 2).

Table 2. Agronomic performance of 3 elite populations.

Variety	Yield(kg/ha)	Days to flower (days)
1. Partap	7509.8	58.5
2. Across 7726 RE	6099.5	57.5
3. Across 7635 RE	5584.9	54.0

The above findings reveal that of all the entries, keeping in mind the resistance to charcoal rot, Across 7726 RE, Poza Rica 7926 and Partap seem to be of high promise. Partap, the indigenous composite, possesses a distinctly higher yield potential with a reasonable level of resistance. These materials should be further studied and be incorporated into a resistance breeding programme.

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Condensed plant type (*ct**) is actually reduced (*rd*)

Condensed plant type (*ct*) observed in crosses involving NY544 (Tracy and Everett MNL 54:57) has been identified as reduced (*rd*). NY544 is derived from the inbred CrS4HLA developed by the Crookham Company. No allelism tests have been done, however, Dr. Steve Marshall, Director of Research at Crookhams, has confirmed that *rd* was incorporated into this material.

W. F. Tracy

Pollen competition and heterosis

Preliminary studies of the relationship between pollen competition and heterotic patterns were carried out in 1985. White endosperm versions of A632 (stiff stalk) and A619 (Lancaster), designated K304 and H104 respectively, were pollinated with a mixture of pollen from a white version of Mo17 (Lancaster) designated K303 and a yellow B73 (stiff stalk). Tassels were bagged before shedding began in the morning and taken down at 11:00 a.m. - about 2 hours after shedding began. The mixture was made on a volume basis with 2 ml of each type. No attempt was made to examine pollen size or number of grains per volume. The same mixture was used to pollinate 5 ears of K304 and H104. Mature ears were measured and divided into 4 sectors based on the total length. The total number of kernels was counted in each sector as was the number of yellow kernels. Data are presented as percentage of yellow kernels. Assuming a one to one mixture of viable pollen χ^2 values were calculated to estimate deviation from expected for each sector and the total for each ear.

The percentage of yellow kernels in an entire ear was not significantly different from 50% for any of the H104 ears (Table 1). Only one sector of the 20 H104 sectors was significantly different from 50% yellow. The tip sectors of H104 were very close to 50% yellow while the butt sectors appeared to have fewer yellow kernels. However, the percentages of yellow kernels in the butt sectors were not significantly different from the expected. When H104 was the ear parent no clear competitive differences between the two types of pollen were observed despite the slightly higher frequency of white kernels at the butt end of the ears.

Table 1. Percentage of yellow kernels on ears of H104 and K304 pollinated by a mixture of B73 (yellow) and K303 (Mo17 white) pollen.

Ear Parent	Ear No.	Sector Butt I	Middle II	Middle III	Tip IV	Total	x^2 1:1 (based on total)	Total number of kernels	Ear length cm
H104	1	41.0	45.4	38.0*	61.3	44.3	2.62	404	18
(A619)	2	52.3	53.1	58.1	50.5	52.4	.51	476	15
White	3	38.1	61.5	61	47.6	53.0	.51	281	14
	4	41.1	47.0	47.4	47.3	45.6	1.44	379	17
	5	48.4	44.0	53.6	52.8	49.3	.01	162	13
K304	1	35.0*	18.7**	44.7	43.1	36.0	15.85**	410	17
(A632)	2	20.7**	42.5	49.0	49.0	40.0	8.43*	449	16
White	3	30.0	31.0	50.0	45.9	40.0	3.26	167	17
	4	17.6**	32.9*	40.6	50	34.7	16.47**	354	17
	5	22.6**	39.8	65.9*	64	49	.07	371	16

* significant x^2 deviation from a 1:1 ratio at the 5% level.
** significant x^2 deviation from a 1:1 ratio at the 1% level.

Pollen competition is indicated by the data from the crosses when K304 was used as the ear parent. Three of the five totals deviate significantly from the expected one to one ratio. Another has a high but nonsignificant χ^2 value. The fifth total is close to a one to one ratio due to unusually high numbers of yellow kernels in sectors III and IV. As we do not know the actual ratio of pollen grains from the white and yellow parents the totals alone do not indicate competition between the two types of pollen. If the mixture was a one to one ratio numerically and pollen competition was occurring, we would expect to see one to one ratios of yellow and white kernels at the tips of the ear with progressively more of the competitive type (in this case white) toward the butt. Such a pattern is obvious in the data from the K304 ears. The tip sectors while variable in ratio are not significantly different from a one to one. In contrast, all five of the butt sectors have an excess of white kernels. Four of the five butt sectors are significantly different from expected. The fifth χ^2 value was not significant due to a low number of kernels (33) in that sector. Sector II consistently has more white kernels than sector III and IV, while sector III is not very different from sector IV, thus indicating that competition by the white-endosperm parent is enhanced on K304 silk beyond the half way point of the ear. This observation was made on mature dry ears, and how it relates to silk length at pollination is not known.

When H104 (Lancaster) was the ear parent no competitive advantages were evident for pollen from either the stiff stalk (B73) or Lancaster (K303) pollen parent. However, the Lancaster pollen parent had a clear advantage when K304 (stiff stalk) was used as the ear parent. Based on the pedigrees it is expected that plants from the union of K304 and K303 will be more productive than those from the union of K304 and B73.

These studies were limited due to inadequate seed supplies. They will be expanded to include all possible combinations of pollen and ear parents including self pollen. Investigations have begun on factors that may be involved in these competitive differences.

White endosperm inbreds were supplied by Clyde Wassom of Kansas State and Paul Crane of Purdue.

W. F. Tracy

The Effect of GA_3 on Corngrass (*Cg*)

An attempt was made to normalize corngrass (*Cg*) plants using gibberellins as reported by N.H. Nickerson (Am. J. Bot. 47:809-15). The dosage and application schedule followed Nickerson's protocol. GA_3 was obtained from Sigma. Plants treated with GA_3 had reduced numbers and growth of tillers and their mainstalk was greatly elongated compared to the control. However, out of over 100 treated plants we observed no normal inflorescences. Nickerson reported normal inflorescences resulted from GA treatment. Perhaps the GA solution Nickerson used, in addition to GA_3, contained growth regulating substances that induced normal flower development.

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The role of mitochondria in the generation and manifestation of hybrid vigor

McDaniel and Sarkissian (Genetics 57:843, 1967) reported the manifestation of hybrid vigor in O_2 consumption and P/O ratios by isolated seedlings (4-5 d etiolated) from hybrids of maize. We are further investigating this phenomenon by monitoring O_2 consumption of highly purified mitochondria from pedigree stocks of 5 inbred and 8 reciprocal hybrid lines. We have also subjected purified seedling (4-5 d etiolated) mitochondria from the 13 lines to a system generating in organello protein synthesis to detect both quantitative and qualitative differences in protein synthesis by mitochondria.

Mitochondria were isolated from 4-5 d etiolated seedling tissue of 13 pedigreed lines (Oh43, W23, M14, Mo17, B73, Oh43 X W23, W23 X Oh43, Oh43 X M14, M14 X Oh43, W23 X M14, M14 X W23, Mo17 X B73, B73 X Mo17) and purified on discontinuous sucrose gradients by the method of Forde (PNAS 75:3841, 1978). For measurement of O_2 consumption under State 1 conditions (no exogenous Krebs cycle intermediates or ADP) depletion of O_2 was monitored on a Clarke electrode apparatus. Concentration of protein was determined (Bradford, Anal. Biochem. 72:248, 1976) and mitochondria concentration correlated with amount of protein. Mitochondria of the inbreds Oh43 and W23 and their reciprocal hybrids show positive heterosis in the slopes of the regression lines plotting $\%O_2$/ug protein versus time in minutes:

Line	Mean slope (B) x 10^{-4}
Oh43	3.12
W23	4.53
Oh43 X W23	9.14
W23 X Oh43	7.75

These differences are significant between the inbreds as a group and the hybrids as a group but not significant differences between inbreds or between hybrids ($p<0.05$). The remaining 9 lines are currently under investigation.

Mitochondria were isolated for in organello protein synthesis studies by a similar procedure modified to accommodate small samples (0.5 g fresh weight tissue) (Boutry, Eur. J. Biochem. 127:129, 1982). Purified mitochondria were incubated on a shaker in the dark for 90 min in media including ^{35}S-methionine and succinate/ADP as an energy-generating system to measure incorporation of label into acid-insoluble material according to Forde (ibid.). Bacterial contamination was ruled out by plating aliquots on complete media and performing replicate experiments with acetate as the only carbon source (Forde and Leaver, PNAS 77:418, 1980). Three to 4 replicates were carried out for each line.

Mitochondria purified from 0.5 g seedling tissue incorporate a significant amount of ^{35}S-methionine into TCA-insoluble material. Incorporation is chloramphenicol-sensitive and cycloheximide-insensitive. Inbred mitochondria incorporate significantly more than hybrid mitochondria of the same tissue sample:

Line	cpm
Oh43	5,681± 2902[a]
W23	61,465±11700
Oh43 X W23	1,808± 1072
W23 X Oh43	1,203± 33
M14	14,630± 5422
M14 X Oh43	1,578± 374
Oh43 X M14	1,650± 254
W23 X M14	1,331± 113

[a]Mean±SE

We are further investigating this observation. Some hypotheses we are testing include: 1) Hybrid cells are larger than inbred cells in seedlings of 4-5 d incubation, thereby reducing the number of mitochondria in 0.5 g fresh weight. 2) The number of mitochondria per cell, regardless of cell size, is modified in hybrids thereby altering number of mitochondria in 0.5 g fresh weight. 3) Hybrid mitochondria differ in uptake of ^{35}S-methionine and/or succinate/ADP

from their inbred parents, thereby altering specific activity of newly synthesized proteins.

Mitochondria labelled with ^{35}S-methionine for 90 min as described above were subjected to SDS-PAGE according to Laemmli (Nature 227:680, 1970), impregnated with PPO/DMSO and fluorographed. Typical N cytoplasm protein profiles were observed with consistent presence of several larger MW proteins (> 92 kD). Genotypic differences were apparent; these patterns seem to be strictly passed on in hybrids in a maternal fashion. No apparent differences were found attributable to hybridity per se.

Mitochondria were also isolated and purified from 0.5 g of 20 d plants as described above. Purification was complete monitored by the lack of green color in the final mitochondrial pellet. Incorporation of ^{35}S-methionine was very low (200-500 cpm) and no significant differences were detectable among lines. Protein profiles after SDS/PAGE/fluorography were virtually identical for all lines regardless of genotype or inbred/hybrid status.

We continue to investigate the manifestation of hybrid vigor by seedling mitochondria in both respiratory activity and quantitative and qualitative protein synthesis differences.

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and Kevin D. Nowicki

Response of mitochondria to chemical stress

To further investigate the role and/or response of mitochondria to heat shock, we have looked at the effect of various chemicals on both corn seedling in vivo and isolated mitochondrial in organello protein synthesis. We recently reported that Oh43 seedling (4-5 d etiolated) mitochondria respond to a 10 C, 30 min temperature shift by a slight but significant increase in ^{35}S-methionine incorporation in part manifested in the large enhancement of a 52 kD protein we have termed a mitochondrial HSP (heat shock protein) (Nebiolo and White, Plant Phys., in press). Dinitrophenol (DNP), KCN/oligomycin and arsenite induce the heat shock response in *Drosophila* and various other animal systems (Ashburner, Cell 17:241, 1979) but of these only arsenite has been shown to significantly induce mRNAs for the HSPs in a plant system, specifically soybean (Czarnecka, PMB 3:45, 1983). In our study, 700 uM DNP significantly reduced ^{35}S-methionine incorporation in purified mitochondria, while 100 uM, 70 uM and 10 uM had no effect. Protein profiles generated by SDS/PAGE/fluorography showed no evidence of the 52 kD HSP. KCN/oligomycin effectively abolished ^{35}S-methionine incorporation at the concentrations used in our study (Czarnecka, ibid.). Arsenite induced both nuclear-encoded HSPs in seedlings and the 52 kD mitochondrial HSP in purified mitochondria at all concentrations monitored (100 uM - 10 uM), optimally at 50 uM arsenite. This finding strengthens the hypothesis that the mitochondrial 52 kD HSP is a "stress" rather than a "heat shock" protein as well as ruling out preferential enhanced synthesis due to slight increase in temperature. We are continuing our investigation by treating mitochondria with various heavy metals, specifically cadmium, which has been shown to induce HSP mRNA in soybean tissue, and various agrichemicals hypothesized to induce a "stress" response.

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Male gametophytic selection

In a broad sense gametophytic selection (GS) can result from pollination competition as well as from post-pollination competition. Post-pollination male gametophytic fitness depends mainly on pollen germination time, tube growth rate and fertilization ability.

Several data recently produced indicate that GS can produce significant evolutionary changes and can be used to develop efficient methods of plant breeding. In fact the phenomenon is assumed to rely on two main factors: i) genetic variability of gametophytic origin and ii) gametophytic-sporophytic gene expression. Besides the classical examples of *wx*, *Ac* and *Adh*, information supporting that these assumptions are valid for a large portion of the genome has been obtained by different authors. The data concern male gametophytic selection of several chromosomal deletions, maize pollen tube elongation in vitro, tomato and maize isoenzyme patterns in pollen and in sporophytic tissues and mRNA, from pollen and shoots of *Tradescantia* and maize.

Selection for pollen competitive ability in maize produced positive and correlated response for sporophytic traits (Ottaviano et al., T.A.G. 63:249-254). However, in view of the selection procedure adopted, the results furnished only an indication that selection response is due to the variability of genes expressed in the gametophytic phase. For this reason a more comprehensive experiment strictly based on within-plant gametophytic selection has been carried out. The selection criterion derives directly from the maize ear structure: the silk length varies according to the position of the flower on the ear, increasing from the top to the base. Within-plant selection is applied when the pollen from the same heterozygous plant is used to pollinate a single plant, either for selfing or for crossing. Differences in the progeny due to the position of the kernels on the ear (apex or base) reveal response to gametophytic selection due to genes expressed in the postmeiotic male gametophytic phase. The selection procedure adopted is a recurrent selection scheme, where the

sporophytes (plants) were chosen strictly at random. After two cycles of selection two populations were produced: Base population (high gametophytic selection intensity) and Apex population (low gametophytic selection intensity). Response to selection was evaluated in 60 S2 and 160 FS (full-sib) families derived from the two populations. The S2's (30 base and 30 apex) were used to study pollen competitive ability and the FS's (80 base and 80 apex) to evaluate the correlated response of sporophytic traits: 50 kernel weight (50-KW), kernel number per row (KNR) and number of kernel rows per ear (RN).

Gametophytic competitive ability of each S2 family was evaluated in comparison with a standard inbred line by means of a mixed pollination technique. The value is expressed as the coefficient of regression (b) describing the variation of uncoloured kernels (the standard produces coloured aleurone) from the apex to the base of the ear.

The mean value of gametophytic competitive ability in the progeny produced at higher selection intensity is higher than that of the progeny produced at low selection intensity ($b = 0.31$ and -1.24 for the base and apex population respectively; the difference is statistically significant, $P<0.05$), showing that the variability of the character is largely based on genes expressed in the gametophytic phase. Sporophytic effects could also play an important role; however, considering the selection procedure used, they should not contribute to the selection response obtained.

A positive correlated response is observed for mean kernel weight ($50KW = 15.67$ and 14.53; a significant difference, $P<0.05$), a character which reflects growth in the endosperm. KNR and RN, which are mainly related to developmental processes, do not reveal significant differences. Considering that the base population was intercrossed for several generations and therefore linkage should not play an important role in the correlated response, the results obtained indicate that there are genes controlling basic physiological processes of growth which show sporophytic-gametophytic genetic overlap.

E. Ottaviano, M. Villa and A. Legrenzi

HSP synthesis in the male gametophyte

Most plant tissues respond to heat shock by synthesizing a specific set of polypeptides (HSPs). Pollen seems to be the only exception. Analysis of germinating pollen showed no HSP synthesis in *Tradescantia* (J.P. Mascarenhas and M.V. Altschuler, 1983, in "Pollen: Biology and Implications for Plant Breeding" pp. 3-8, D.L. Mulcahy and E. Ottaviano eds.) and only two, non-typical polypeptides in maize (P. Cooper et al., 1984, Plant Physiol. 75:431-441). However, there are no data concerning HSPs during pollen maturation, a time during which corn plants are very likely to suffer high temperature stresses.

In this report we analyze heat-shock response of immature pollen. Whole anthers, taken 12 days after meiosis or at anthesis, were heat-shocked (37 C) for 3 hours. Pollen was then collected, extracted, subjected to 1D SDS PAGE and the gels exposed at -80 C.

At anthesis no incorporation of ^{35}S-methionine into proteins could be detected. Twelve days after meiosis both heat-shock and heat-stroke proteins were found, although they differed both in the size and in the number from the typical HSPs induced in sporophytic tissues. Also, unlike most of the sporophytic ones, they appeared to be genotype dependent. Inbred N172 showed 2 HSPs, approx. 81 and 72 kD, while inbred Mo17 showed 4 HSPs, approx. 72, 69, 63 and 53 kD. These data indicate that: i) HSPs are inducible in pollen, during its maturation within the anthers. ii) gametophytic genetic variability in response to high temperature stress exists also at the molecular level, although no correlations can be established between specific HSPs and the degree of tolerance to heat shock expressed by different genotypes in terms of germination and tube growth.

We also explored the existence of such variability in the sporophyte. In a preliminary screening of 22 inbred lines, using root tips as sporophytic tissue, several differences between genotypes were found.

Further analysis of gametophytic HSPs induced at different stages of pollen maturation is in progress.

C. Frova, G. Binelli and E. Ottaviano

Nucleolar organizer activity analyzed by silver-staining procedure

Silver-staining methods, developed for localizing nucleolar organizer regions (NORs) both in animal and plant chromosomes, reveal gene activity at the ribosomal DNA sites. The procedure described by M. Hizume et al. (Stain Technology 55:87-90, 1980) was applied to maize root tip cells with some modifications. Pretreatment, fixation and washing are performed as described in the cited paper. Differences are as follows:

- Each step is carried out on a single root tip placed in a small container
- After washing off the enzyme solution by several changes of distilled water, a few drops of 60% acetic acid are added for 30 minutes
- Dissociation into cells is achieved mechanically by needles
- Chromosome permanent preparations are obtained by pipetting a few drops of the cell suspension onto clean slides warmed on a hot plate (40-45 C)
- For Ag-staining, slides treated with 50% $AgNO_3$ and covered with a coverglass are kept in a moisture chamber for one hour at 60 C and illuminated by a 60W bulb.

Figure 1 shows root tip metaphase chromosomes prepared and stained as described: NORs are clearly

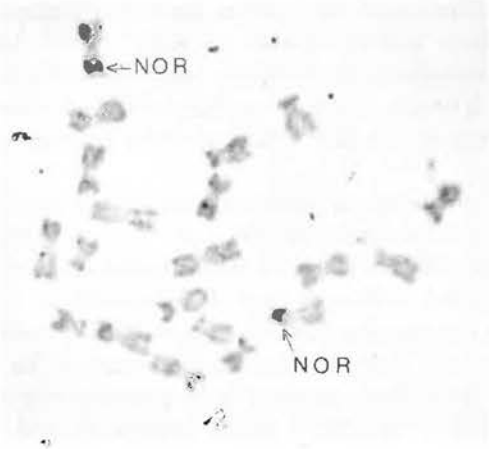

Figure 1 Silver stained metaphase chromosomes of *Zea mays*. Ag-NORs appear as black dots above the secondary constriction on short arm of chromosome 6. x 5625

visible on the short arm of chromosome 6. The possibility of visualizing ribosomal gene activity offers many opportunities in cytogenetics and genetics.

Silvana Faccio Dolfini

In vitro characterization of 54-1 and 56-1 mutants

The 54-1 and 56-1 mutants have been isolated in our lab following EMS pollen mutagenesis (56-1) and as a spontaneous event (54-1). They are recessive embryo-lethals with a small endosperm devoid of carotenoids and anthocyanins. Crosses with the TB-A set of translocations indicate that both mutants are uncovered by Tb-1Sb. An allelism test with *dek1*, uncovered by the same TB, revealed the allelic relationship between the two mutants and *dek1*.

Tests were then planned to find out whether the pigmenting capacity of the mutant could be recovered by feeding the mutant with pigment precursor. Accordingly immature mutant seeds, obtained on a segregating selfed ear, were cultured as soon as phenotypically recognizable, i.e., 10 days after pollination, on modified LS medium (Gengenbach and Green, Crop Sci. 15, 1975) supplemented with different precursors of flavonoid biosynthesis: phenylalanine, cinnamic acid, dihydroquercetin and malonyl-CoA (5×10^{-3}M for the former, 5×10^{-4}M for the latter) as well as on an unsupplemented medium.

None of the supplements led to positive response as far as pigment production is concerned.

Since the mutants are developmentally blocked at the proembryo stage we wanted to see if they maintain the capacity to grow as undifferentiated callus. Cultures were thus initiated from immature mutant and normal embryos obtained from the same selfed ear.

After two months of culture callus derived from normal seeds looks friable and embryogenic, mutant embryos on the other hand are generally arrested and appear necrotic. In a few cases small callus masses were observed. They were transferred to an enriched medium where they will be watched for growth and differentiation into somatic embryos.

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Opaque-6 allelic to *pro1* mutant

The *pro1* and *o6* mutants have been extensively studied since the first reports (Ma and Nelson, Cereal Chem. 52:412-419, 1975; Gavazzi, Racchi and Tonelli, Theor. Appl. Genet. 46:339-345, 1975). Both mutations affect storage protein synthesis in the endosperm and lead to seedling lethality. Both share a number of phenotypic traits like a dull and collapsed endosperm morphology, stunted seedling growth, striation of the first two leaves and seedling lethality.

Previous studies had shown that the *o6* mutation is associated with significant reduction in zein content (observed also in *pro1* mutants) as well as with suppression of an endosperm soluble protein, named b32 (Soave et al., Cell 27:403-410, 1981), while *pro1* mutant seedlings recover if supplemented with L or D proline and other amino acids are without effect (Racchi et al., Plant Sci. Letts. 13:357-364, 1978; Tonelli, Plant and Cell Physiol. 26:1205-1210, 1985).

The common features shared by the two mutants suggested to us to test the response of *o6* seedlings to proline supplementation. The positive result obtained prompted us to test the allelism between *pro1* and *o6*. Plants grown from non mutant seeds obtained upon selfing *+/o6* and *+/pro1* plants were crossed inter se.

Complementation was then ascertained on the basis of different criteria such as endosperm and leaf morphology, seedling growth and lethality. The *o6/pro1* heterozygous combination promoted collapsed endosperm morphology, striation of leaves and conditional seedling lethality, thus indicating functional allelism of the two mutants.

Out of 40 crosses, 24 gave ears with a 3 to 1 segregation for normal and collapsed endosperm seeds yielding mutant seedlings. These results are those expected if *pro1* and *o6* are functionally allelic.

Other monogenic recessive mutants resembling *pro1* in endosperm phenotype have been tested for allelism against *pro1*; however, none of them (ed-4-v, ed-47-1, 73-1, 118-1 and E-487-c) showed functional allelism with *pro1*.

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Variability in nuclear S1-homologous sequences among lines of maize

The presence of sequences homologous to the plasmid-like mitochondrial DNAs (mtDNAs) in the nuclear genome of maize (R.J. Kemble et al., Nature 304:744-747, 1983) has been examined further. In various cytoplasm and restorer combinations within hybrid background M825/Oh07, seven BamHI nuclear DNA (nDNA) restriction fragments (designated A-G) consistently exhibit homology at stringent hybridization conditions to pZmS21 (R.J. Kemble et al., 1983; and unpublished observations). PZmS21 is a clone containing a 4.0 kb S1 insert (R.D. Thompson et al., Nucl. Acids Res. 8:1999-2008, 1980), including part of the region of homology with S2 and psbA chloroplast gene homology (P. Bedinger et al., personal communication).

While all the S1 homologous BamHI nDNA fragments hybridize with pZmS21, pZmS42 hybridizes to all fragments except fragment D. PZmS42 is a clone containing a 1.5 kb insert of "unique" S1 DNA, i.e. neither containing homology with S2 nor with the psbA gene. BamHI restriction digests of chloroplast DNA (ctDNA) exhibit homology to clone pZmS21 in only one fragment, which is identical in size (4.7 kb) to the M825/Oh07 BamHI fragment D. Thus fragment D most likely represents contaminating ctDNA (i.e. the psbA gene; observation initially presented by P. Bedinger, 1984 Maize Genetics Conference). Alternatively, fragment D in BamHI nDNA digests may be composed both of psbA gene sequences residing in the nuclear genome and ctDNA contamination. Clones of fragment D are presently being screened. Examination of a number of additional maize lines indicates that there is variability in distribution of S1 homologous nDNA sequences between lines. Some lines that contain all of the fragments A-G identified in M825/Oh07 differ in their relative amounts of the bands. For example, compared to M825/Oh07, inbred WF9 contains lower levels of band C while band B is diminished in B37. Other lines contain S1 homologous fragments which have slightly different mobilities than fragments A, B and C. This variability could have resulted from differential sequence rearrangements among lines descended from an original S1 insertion event into nDNA. Furthermore, other lines, such as Tr, do not possess A, B or C fragments.

When two such lines (e.g. WF9 and M825) that differed in S1 nDNA sequence arrangement were crossed, the resulting hybrids contained S1 nDNA fragments from both parents. This demonstrates a stability in inheritance of the S1 nuclear sequences. Examination of S1 nDNAs following several generations of reciprocal crosses will be necessary to fully ascertain stability. In collaboration with D.B. Walden and C.A. Rees at the University of Western Ontario in London, we are using *r-x1* generated monosomics for each of the ten maize chromosomes to physically map the distribution of the S1 homologous sequences within the nuclear genome of selected lines.

C.L. Baszczynski, J.E. Carlson and R.J. Kemble

Heat shock protein synthesis in sterile and fertile mitochondria

In organello labelling and SDS-PAGE have been utilized to examine polypeptide synthesis in isolated mitochondria from normal and S-sterile maize (B37) seedlings under various temperature conditions. This was partially based on a concern that the temperature shifts involved in transferring the mitochondria from the 4 C isolation conditions to the 30-37 C labelling conditions used in in organello studies might be generating a "heat shock-like" condition and giving rise to synthesis of HSPs, which might then be interpreted as normal mitochondrial protein synthetic products.

Mitochondria were isolated from five day old etiolated coleoptiles (R.J. Kemble et al., 1980; Genetics 95:451-458), treated with DNAase and/or RNAase to eliminate cytoplasmic nucleic acids and purified over sucrose, all at 4 C. The mitochondria were gently resuspended in 150 ul of a buffer containing 50 mM Tris-HCl (pH 7.2), 20 mM $MgCl_2$, 40 mM KCl, 2 mM dithiothreitol, 2 mM ATP, 100 mg/ml BSA, 25 umol of 19 amino acids (minus methionine), 50 uCi of ^{35}S-methionine and sterile water to volume. The reaction mixtures were incubated for 60 minutes at 30, 37 or 42 C and then put on ice. After centrifugation, pelleted mitochondria were solubilized and 25,000 cpm of acid-precipitable lysates were subjected to SDS-PAGE as described elsewhere (C.L. Baszczynski et al., 1982; Can. J. Biochem. 60:569-579).

Incorporation of labelled precursor into polypeptides was 30-35% less at 42 C than at 30 C. While a broad spectrum of newly synthesized polypeptides was observed at 30 C in mitochondria from both N and S cytoplasms, incubation at 37 or 42 C resulted in a depression of synthesis of many of these polypeptides and a marked enhancement in synthesis of a prominent polypeptide of approximately 57 kD. Sterile mitochondrial isolations and nuclease pretreatments of intact mitochondria preclude the possibility of this polypeptide being a product of translation of non-organellar mRNA. In addition, it does not correspond in size to any of the previously described maize cellular HSPs (C.L. Baszczynski et al., 1982).

Since the 57 kD polypeptide is not present in samples labelled at 30 C, it would appear that the transfer from 4 C to 30 C does not lead to an HS response. However, it is clear that a similar shift to 37 C does give rise to a heat shock response, such

that the experimental temperature conditions must be taken into consideration when evaluating protein products. Earlier work on maize cellular HSPs revealed that the precise temperature regime of an experiment influences the spectrum of newly synthesized polypeptides (Baszczynski et al., 1984; Ph.D. Thesis, U.W.O., London, Canada).

The present results substantiate an earlier MNL report of HSP synthesis in normal maize mitochondria (C.M. Nebiolo and E.M. White, 1984; 58:144-145) as well as reports in a recent abstract (R.M. Sinibaldi and T.H. Turpen, 1985, 1st Int. Congr. Pl. Mol. Biol., PO-2-229); both suggest that a 50-60 kD polypeptide synthesized following a heat shock is encoded within the mitochondria. In addition the present report indicates that *cms-S* mitochondria do not differ from the normal cytoplasm in response to heat shock conditions.

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Leaf protoplast RNA synthesis as influenced by IAA and ethanol as an estimate of heterosis

Leaf protoplasts isolated by enzymatic digestion [3 percent Cellulysin in osmoticum (OS-0.6M sorbitol, 5 mM $CaSO_4$ and 5 m $MgCl_2$)] from seven-day old seedlings of a maize hybrid and its inbred parents (N28 and Mo17) were utilized for a study of RNA synthesis as a measure of heterosis. The influence of the growth hormone IAA and of ethanol (EtOH) at a concentration of 10^{-7} molar upon tritiated uridine (^{3}H-U) incorporation were measured over a seven hour period.

Incorporation of ^{3}H-leucine into protein over a 6 hour period was used as a measure of cell viability. It was found that the kinetics of ^{3}H- leucine incorporation paralleled those of ^{3}H-U (Figure 1) and that cells were retaining their viability over the time of the experiment.

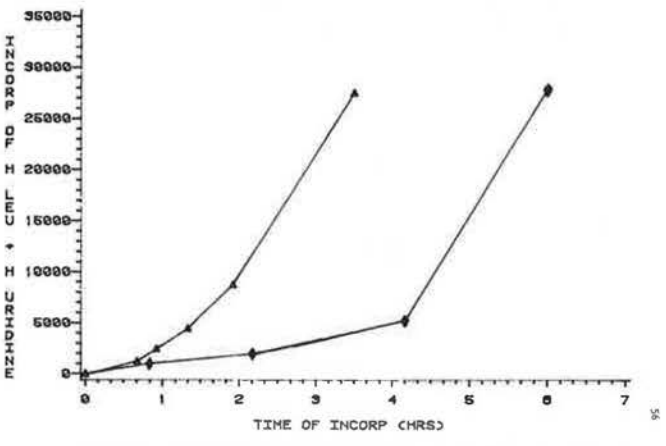

Figure 1. Incorporation of ^{3}H-uridine (▲---) and ^{3}H-leucine (●---) into the acid insoluble material of MO17 mesophyll protoplast.

No statistically significant differences were found in the quantity of RNA isolated from N28, Mo17 and their hybrid when their protoplasts were incubated in OS (Table 1). Treatment of N28, Mo17 and of the hybrid with EtOH produced an increase of 63.4, 23.5 and 79.8 percent respectively in the content of total RNA. Treatment of the same strains by 10^{-7}M IAA produced increases of 82.8, 42.1 and 109.7 percent respectively for N28, Mo17 and the hybrid. The IAA effect was, however, confounded because small quantities of EtOH were utilized to dissolve this hydrophobic compound into OS. Experiments are now in progress to measure the effect of IAA alone.

Table 1. RNA extracted from 10^{-7}M IAA treated and untreated corn mesophyll protoplasts.

Strain	Treatment	ug of RNA/ 10^6 mesophyll protoplasts
N28	OS	125.1
	EtOH Control	204.4
	10^{-7}M IAA	228.7
Mo17	OS	183.7
	EtOH Control	224.5
	10^{-7}M IAA	261.1
N28xMo17	OS	139.8
	EtOH Control	251.3
	10^{-7}M IAA	293.2

Oligo (dt)- columns were used to separate poly(A)+mRNA from the non-poly(A) species (Table 2). The quantity of poly(A)+RNA did not differ significantly between the strains when protoplasts were incubated in OS. Treatment with 10^{-7}M IAA and EtOH increased the synthesis of poly(A)+mRNA species with the exception of Mo17, which showed a decrease of 12 percent when exposed to EtOH only. N28 and the hybrid showed a 9 percent increase each. The effect of EtOH may be related to alterations in membrane permeability which permitted greater entry of ^{3}H-U into the protoplasts. This "leaky" response may not have been a characteristic of the Mo17 plasmalemma.

Table 2. RNA and poly(A)+mRNA extracted from 10^{-7}M IAA treated and untreated corn mesophyll protoplasts.

Strain	Treatment	CPM 10^6 cells (Total RNA)	CPM 10^6 cells (Poly(A)+mRNA)	%
N28	OS	99684	947	0.95%
	EtOH Control	109129	1017	1.04%
	10^{-7}M IAA	130114	1237	1.24%
MO17	OS	147959	1450	0.98%
	EtOH Control	50124	575	0.02%
	10^{-7}M IAA	70087	806	1.15%
N28xMO17	OS	94085	946	1.01%
	EtOH Control	102642	1034	1.10%
	10^{-7}M IAA	122430	1231	1.31%

The hybrid manifested the greatest increase in poly(A)+RNA with IAA. Although this result is in general agreement with Nebiolo et al. (Plant Sci. Lett. 28:195-206, 1982/83) it was less than that observed with application of 10^{-7}M gibberellic acid (GA_3) to maize coleoptile cells. This variation may be related in part to differences in the $260_{OD}/280_{OD}$ ratio between the two studies and to the fact that auxins are known to specifically increase (100-250%) rRNA content but induce a smaller increase (25-50%) of poly(A)+RNA (Ann. Rev. Plant Physiol. 28:537-564, 1977). GA_3 is also a specific inducer of poly (A)+RNA in immature plant tissue. The difference may therefore result from the specific action of each hormone. A significant difference also exists between the EtOH and the IAA treatment. The IAA treated protoplasts for N28, Mo17 and the hybrid have 19.2, 40.2 and 19.1 percent more poly (A)+RNA respectively than do the EtOH treated protoplasts. The increased synthesis may have resulted from enhanced activity and/or quantity of DNA-dependent RNA polymerase II produced by the IAA treatment or increased stability of the poly(A)+RNA. Part of the increase may also result from the availability of increased ^{3}H-U pools. However, regardless of treatment the hybrid manifested the greatest potential for ^{3}H-U incorporation.

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Knob expression in maize-*Zea diploperennis* hybrids

In order to trace the inheritance of chromosome knobs and how their expression may or may not change through successive generations, the race Chapalote with internal knobs was crossed with *Zea diploperennis* with terminal knobs. Cytological material from specific parent plants and their F_1 progeny was studied for precise comparison of chromosomes.

Theoretically, if the two parental strains were homozygous for knobs, the number found in the F_1 generation should equal half the sum of the total number of knobs in both parents. However, if the knobs are heterozygous in the parents, there will be segregation and variation in the knob number of the F_1 plants. Pure inbred lines were not used and the material is heterozygous.

Table 1 shows the number of knobs observed and counted in examinations of cells from specific plants of *Z. diploperennis*, Chapalote, the F_1 progeny, and the expected range in knob number for heterozygous F_1's. Analysis of the data reveals two points. First, there is a higher number in the range of internal knobs than the expected maximum number contributed by both parents. Secondly, there is a lower range in the number of terminal knobs in the F_1 hybrids than would be the expected minimum. This suggests that terminal knobs of *diploperennis* may shift to internal positions when hybridized with maize.

TABLE 1. Chromosome knob number in diploperennis, maize and F_1 hybrids.

Plant	Collection	Knob number: Observed Internal	Observed Terminal	Expected range Internal	Expected range Terminal
Chapalote (9-6)	SIN 2, INIA, R-19, 70-71	4-7	0		
Diploperennis (1-2)	Iltis #1190, Manatlan	1	6		
Diploperennis (2-4)	Guzman #777, La Ventana	1	4-5		
F_1 hybrid 1-2 X 9-6	I.U. 85-26	4-9	1-3	4-8	6
F_1 hybrid 2-4 X 9-6	I.U. 85-28	6-9	2-4	4-8	4-5

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Endosperm chromosome number in relation to kernel development

The chromosome numbers of the endosperm and the embryo have been considered to play a vital role in proper endosperm development. However, various hypotheses suggested from time to time could not fully explain seed failure in terms of genome balance. The hypothesis that triploidy per se is an essential condition for normal endosperm development and that imbalances in chromosome number lead to anomalous development was tested in a study involving interploidy crosses in maize and the results confirm the validity of this hypothesis.

The 3n x 2n crosses were made to manipulate the chromosome number of the endosperm. The kernels obtained from these crosses were classified into eight categories on the basis of extent of kernel development. The plump kernels were classed in category I and completely shrivelled kernels in category VIII.

The chromosome counts in the root tip of the seedlings from kernels in various categories were used for extrapolation of the chromosome number of the endosperm. It was observed (Table 1) that the endosperm chromosome number in different categories ranged from 30 to 46. However, no kernel with 42 chromosomes in the endosperm was observed.

The highest mean number of chromosomes in the endosperm was 37.33 in category VIII (shrivelled kernels). The lowest mean number (31.29) was observed in category I (plumps). The progressive increase in kernel shrivelling from category I to VIII was observed to be correlated with the increase in mean chromosome number in the endosperm. No significant differences in mean chromosome number were observed among categories I, II, and III, and among categories IV, V, and VI. Accordingly, four groups differing significantly in the endosperm chro-

Table 1. Endosperm chromosome numbers in different kernel categories from 3n x 2n cross

Kernel Category	Percentage of kernels with endosperm chromosome number								Total No. of kernels from which root tips were smeared	Mean chromosome number of endosperm
	30	32	34	36	38	40	44	46		
I	69.05	9.52	9.52	11.90	-	-	-	-	42	31.29
II	51.92	26.92	13.46	7.69	-	-	-	-	52	31.54
III	44.44	26.67	13.33	15.56	-	-	-	-	45	32.00
IV	16.22	35.14	18.92	16.22	10.81	2.70	-	-	37	33.57
V	15.38	23.08	30.77	23.08	-	7.69	-	-	26	33.85
VI	4.65	25.58	34.88	23.26	9.30	2.33	-	-	43	34.28
VII	1.67	15.00	26.67	18.33	18.33	15.00	1.67	3.33	60	36.20
VIII	-	4.44	8.89	26.67	35.56	24.44	-	-	45	37.33

mosome number could be identified. These four groups differed among themselves in the extent of endosperm development. These results suggest a positive correlation between the increasing endosperm chromosome number above 30 and the extent of kernel shrivelling.

D. S. Mathur and K. R. Sarkar

Knob-heterochromatin distribution in Sikkim Primitive strains and Nal-Tel

A striking similarity in knob constellation between Nal-Tel and two Sikkim Primitive strains, viz., Tripura collection No. 2 (T-2) and Muralia, a collection from Pithoragarh of Uttar Pradesh hills bordering Nepal, has been observed.

In strain T-2, there are 11 knob positions at 2L, 4L, 5L, 6S, 6Lb, 6Lc, 7L, 8La, 8Lb, 9S and 9L. Knobs at 2L, 4L, 6S, 7L, 8La, 9S and 9L are larger in size. The knob at 7L was homozygous and very large. Knobs at 5L, 6Lb, 6Lc are smaller in size.

Muralia showed 12 knobs. It has prominently large knobs at 2L, 4L, 5L, 6S, 7L, 8La, 9S and 9L while 6La, 6Lb and 6Lc positions showed smaller knobs. There is also a large chromomere in the long arm of chromosome 6 prior to knob 6La.

T-2 and Muralia share 10 knob positions in common, viz., 2L, 4L, 5L, 6S, 6Lb, 6Lc, 7L, 8La, 9S and 9L. 1sb and 8Lb are found only in Muralia, and 8Lb was present in T-2 alone.

The Nal-Tel knob number ranged from 8.6-12.2 with a mean of 10. There are 10 common knob forming positions in Sikkim Primitives and Nal-Tel, viz., 2L, 4L, 5L, 6Lb, 6Lc, 7L, 8La, 8Lb, 9S and 9L. It is possible that Nal-Tel, or rather maize belonging to a precursor or derivative of Nal-Tel/Chapalote, was one of the basic materials that was introduced in the North-Eastern Himalayan region.

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Meiotic abnormalities in North-Eastern Himalayan (NEH) maize

Asynapsis: In two collections of Sikkim Primitive maize from Nagaland, viz., N-3 and N-4, meiotic abnormalities were observed. Chromosomes at pachytene stage in these two collections do not spread well and remain as a knot-like structure of thin chromatin surrounding nucleolus. At metaphase I, there is a complete lack of chiasma formation and all the 20 chromosomes are scattered as univalents. It was also observed that homologues always remained attached end-to-end, or a remnant of prior attachment was at least visible. In N-3 all plants studied showed this abnormality, whereas in N-4 not all plants but a few showed abnormal meiosis. Anaphase showed abnormal segregation of chromosomes. Besides laggards, micronuclei and hexads were consistently observed. Surprisingly, no seed sterility was observed in the ears of these two collections, indicating that there was normal megasporogenesis in the pistillate spikelets and that fertilization was effected only by normal pollen grains formed by 10:10 chromosome segregation during Anaphase I in PMCs. The pollen grains with abnormal chromosome number were nonfunctional, as none of the plants studied had any deviation from the normal chromosome number of 20.

Centromeric fusion: The phenomenon of centromeric fusion was consistently observed in almost all collections of maize from NEH. Fusion persisted up to diakinesis stage. In the same plant or even in the same anther different cells showed different chromosomes involved in centromeric fusion. In some cases it was also observed that a particular chromosome was more frequently involved than others. As a whole, fusion of centromeres of non-homologous chromosomes seemed to be a random event.

The fusion of centromeres of non-homologous chromosomes has no effect on the normal segregation of chromosomes belonging to the two bivalents involved, as subsequent meiotic events were completely normal.

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Discovery of Sikkim Primitive precursor in the Americas

Till now, it has been thought that the legendary landrace Sikkim Primitive (SP) discovered by N. L. Dhawan (MNL 38:69-70, 1964) is distributed only in the remote pockets of North-Eastern Himalayas (NEH). Because of its ear morphology, which has

remarkable resemblance to pre-historic wild maize, it has aroused enormous interest among the crop plant evolutionists. Many speculations have been made about its origin and interrelationships with the established races of maize. P. C. Mangelsdorf (In Corn, its Origin, Evolution and Improvement, 1974) assigned it to the lineage of Palomero Toluqueno, an ancient indigenous race of Mexico. H. G. Wilkes (MNL 55:13-15, 1981; MNL 56:27-28, 1982) considered it a derivative of ladyfinger popcorn. Some others, however, did not attach any significance to the potentialities of SP maize.

The problem faced by scientists in identifying SP type of maize in the Western hemisphere is due to three reasons: (1) That the descriptions of the plant types of Sikkim Primitive given by N. L. Dhawan (MNL 38:69-70, 1964), D. Gupta and H. K. Jain (MNL 45:37-39, 1971) and Bhag Singh (In Races of Maize in India, I. C. A. R. Publ., 1977) do not provide full and accurate information. The studies made by these authors suffer serious drawbacks. The climate to which SP maize is adapted is somewhat akin to temperate zones. The SP maize grows in high hills of NEH in a humid and cooler environment. These authors have collected samples of SP maize from the altitude of 4000-6000 feet and directly grown them under the severe stress conditions in the extreme tropical summers at Delhi, where the mercury touches 45 C during the growth period of maize. As a result, the architecture of Sikkim Primitive plant was drastically altered, and the data based on such studies have led to erroneous conclusions and confusing speculations. (2) The second factor has been the strict U.S. quarantine laws, banning the entry of outside maize into the U.S.A., and (3) frustration of scientists not to have access to the natural habitat of SPs and expressing their views based on inadequate information, without accepting the truth that SP plant type still maintains its distinctive features of pre-historic wild maize (see J. K. S. Sachan and K. R. Sarkar, MNL 56:122-124, 1982).

A pragmatic and convincing view on the origin of Sikkim Primitives was conceived by Nobel Laureate, George W. Beadle, who in his personal communication to one of us, in 1978, emphasized that Sikkim Primitive plant type can be synthesized from the existing variability in *Zea*. We had been pondering over the question of antiquity of maize in India, especially of SP maize. After studying the knob heterochromatin distribution of SP maize, which is comparable to present day Nal-Tel, we were convinced that there must be a precursor or derivative of SP type of maize in the Western hemisphere. Now we feel that SP maize has its precursor in the Americas (see Figure). W. L. Brown's illustration of a prolific maize variety from South America (Proc. 20th Annual Hybrid Corn Ind. Res. Conf., 1965, p. 8) bears a striking resemblance to the Sikkim Primitive strains and in our view SP maize is closely related to this variety, probably both sharing a common ancestry. The illustrated plant encompasses all the characteristic features of Sikkim Primitive maize, namely, (1) prolificacy, (2) high placement of ear on the stalk, (3) upper ears terminating in a male spike, (4) popcorn type, (5) small ears, (6) uniform size of ears, (7) central spike of tassel bent, (8) gynoecious stalks and (9) tillering (see J. K. S. Sachan and K. R. Sarkar, MNL 56:122-124, 1982).

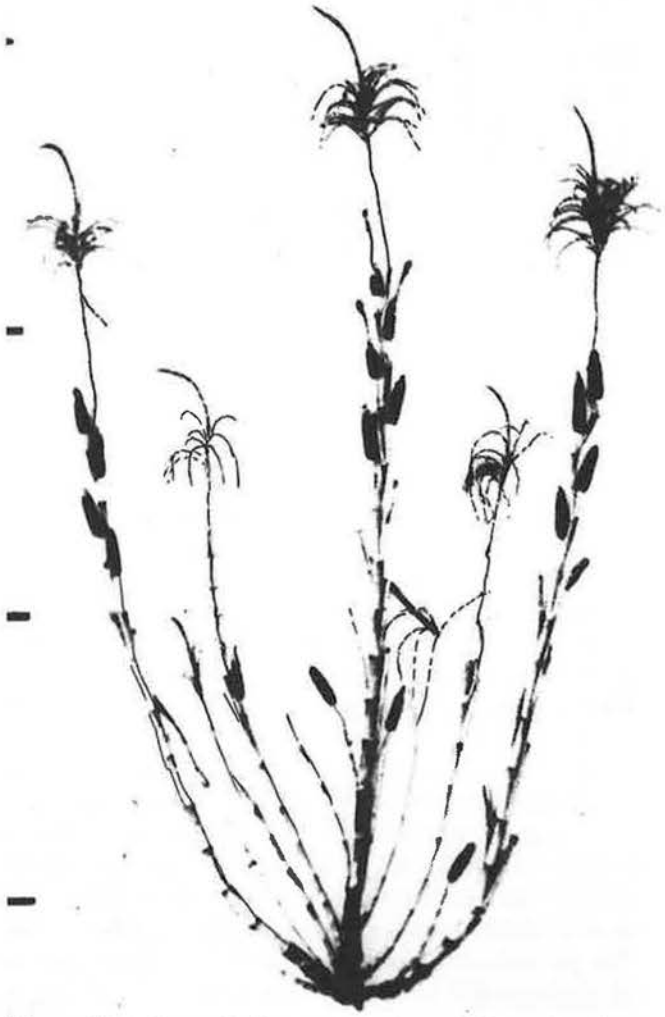

Figure: W. L. Brown's illustration of a prolific variety from South America, presumably precursor of Sikkim Primitive maize (see text).

The evidences gathered from the archaeological studies of the specimens and vegetal remains from Mexican Caves, namely, Bat Cave, Romero's Cave, Swallow Cave, Coxcatlan Cave, El Riego Cave, and San Marcos Cave, dating back to 2000-5000 B.C., amply suggest that the living fossil of pre-historic wild maize, which is considered as progenitor of both Nal-Tel and Chapalote races, is well preserved in the form of 'Sikkim Primitive' in the isolated pockets of NEH. The botanical characteristics of pre-historic maize (see P. C. Mangelsdorf, in Corn, its Origin, Evolution and Improvement, 1974) are exactly the same as that of Sikkim Primitive maize, viz., (1)

prolific nature, (2) ear placement high upon stalk, (3) uniformity in the size of ears, (4) upper ears terminating into male spike, (5) popcorn type, (6) small and slender ear with 8-10 irregular rows, (7) cylindrical ears, (8) soft rachis, (9) soft papery glumes, and (10) tillering potential.

Therefore, it seems that SP maize and Nal-Tel/Chapalote have evolved from the same common ancestor, the pre-historic wild maize.

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Locating of the mei-gene *ms43* by TB-A stocks

The B-A translocations of maize provide the most efficient method for locating the recessive genes (mei-genes especially) to the proper chromosome arm (Roman, Genetics 32:391, 1947; Roman, Ullstrup, Agr. J. 43:450, 1952; Beckett, J. Hered. 69:27, 1978). The cytogenetical data for mapping the mei-gene *ms43*, which induced abnormal chromosome segregation at the anaphase I of meiosis and male sterility, have been presented here.

For this aim the female plants homozygous for *ms43* were crossed by 18 stocks with B-A translocations kindly provided by G. B. Fletcher (Coop. Stock Center, USA). The F1 progenies from 15 cross combinations were studied cytologically. Thirty seeds from each cross combination were planted in a greenhouse. The pattern of meiosis and number of chromosomes at diakinesis-metaphase I have been analysed in each F1 plant.

Based upon cytological examination of F1 plants, the conclusion was made that *ms43* gene is uncovered by TB-8La. Among the progeny from the *ms43/ms43* x TB-8La cross, a total of 27 plants were examined. Twenty-two plants with normal meiosis composed the hyperploid class: 9 plants had 21 chromosomes and 13 had 22 chromosomes. Four plants were hypoploids, one of them appeared to be *ms43* phenotype (Figure 1, Table 1).

The cytological data from cross combination with TB-10Sc were excluded from the total data, because all of the 14 F1 plants studied had 10 bivalents at diakinesis-metaphase I of meiosis. This could mean that the individual male plant used in the cross with the *ms43* homozygote had no B^A chromosomes.

A total of 238 F1 plants from the 13 remaining cross combinations were analysed. No mutant plant with *ms43* spindle phenotype was found. Forty-four plants had 10 bivalents at diakinesis-metaphase I of meiosis and composed the hypoploid class. The hyperploid class contained 65 plants with 11 bivalents at diakinesis-metaphase I, and 27 plants with 10 bivalents and 1 univalent.

Based upon cytological data the *ms43* gene is placed on the long arm of chromosome 8.

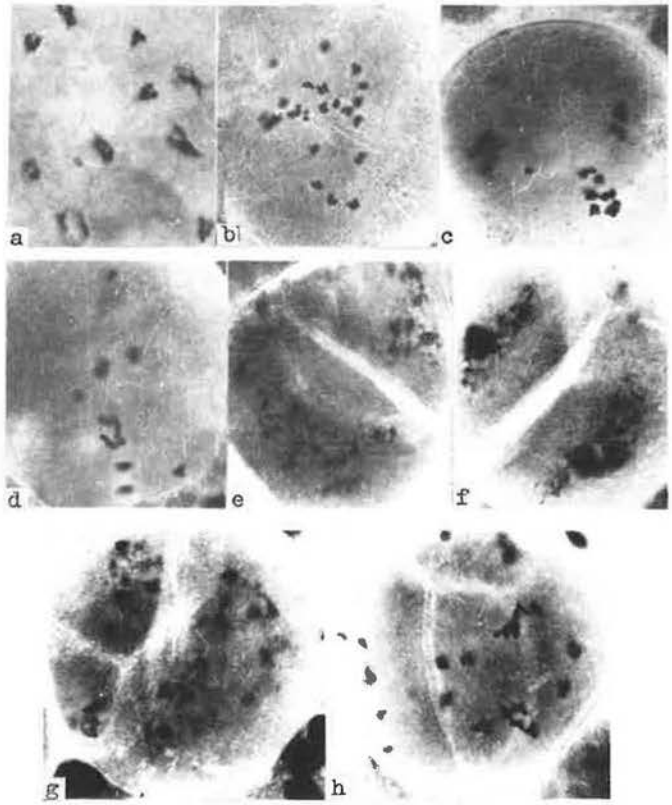

Figure. The meiotic pattern of the ms43 segregant appeared in F1 progeny from cross ♀ ms43/ms43 x ♂ TB-8La
a. diakinesis, 10 bivalents,
b-d. the cells with abnormal chromosome disjunction at anaphase I,
e-h. types of polyads as a result of cytokinesis in the cells with abnormal chromosome segregation.

LOCATING OF THE GENE ms43 UTILIZING TA-B TESTER STOCKS

(♀ ms43/ms43 x ♂ TAB)

Tester stock	Phenotype of meiosis in F1 progeny		Chromosome number in F1 progeny		
	normal	ms43	10II	10II+1I	11II
TB-8La	26	1	4	9	13
TB-1Sb	23	0	2	2	2
TB-1La	21	0	1	-	1
TB-2S:3L	13	0	-	-	-
TB-2L:1S	12	0	1	-	2
TB-3La	14	0	4	5	6
TB-3Sb	19	0	5	2	9
TB-4Sa	27	0	4	-	3
TB-4L:1L	24	0	-	-	4
TB-6Sa	12	0	9	3	2
TB-6Lc	9	0	3	3	3
TB-9S	29	0	4	7	18
TB-9La	19	0	8	2	8
TB-10Sc	20	0	14	0	0
TB-10La	16	0	3	3	7
Total without TB-8La & TB-10Sc	238	0	44	27	65

The *ms43* gene is the 15th gene which has been localized in chromosome 8 (Coe et al., 1983, MNL 57:175; Curtis, 1983, MNL 57:32). It is very interesting that the genes *ms43, el* and *ms8* are placed on the same chromosome.

The preferential or "directed" fertilization of the eggs by hyperploid sperms is clear from the cytological data (Table, right part). However, the TB-A stocks used in the experiment differed significantly from one another for this characteristic. The directed fertilization is strongly marked in the TB-8La and TB-9S, and is obvious in the TB-3La, 3Sb, 10La. The TB-6Sa has an opposite tendency. The chance to fertilize the eggs with hypoploid and hyperploid sperms is nearly equal in the TB-9La.

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Mu1 as a transposon tag

We have investigated the use of the transposable element *Mu1* as a molecular tag for cloning. The major advantage of the Mutator system is its high rate of mutation. This could make Mutator the system of choice for tagging previously unmarked genes. However, the high mutation rate is probably due in part to the high copy number of *Mu1* elements (about 30) in Mutator stocks and their ability to maintain their high copy number upon crossing to non-Mutator lines (Allemand and Freeling, pers. comm.). This high copy number of *Mu1* elements militates against the use of *Mu1* as a transposon tag.

There has been considerable interest expressed in the maize genetics community about cloning genes with a *Mu1* probe. However, to our knowledge, no one has succeeded in using *Mu1* homology alone to identify a target gene. We present below details of an unsuccessful attempt to clone the bronze gene using *Mu1* as a probe that was initiated before bronze was cloned using an *Ac* tag, and suggestions arising from this work on how to overcome the high *Mu1* copy number problem in the future.

I. A failed attempt to clone the bronze locus using Mu1 *as a probe*: The *bronze* locus was considered an ideal model system to test cloning with *Mu1*, since it had been well documented genetically, it was assumed to be present in single copy, and several mutable bronze alleles had been isolated by Don Robertson. We will designate mutable bronze alleles arisen in a Mutator stock as *bz-Mum*. We attempted to clone the *bz-Mum4* allele isolated by Robertson in a Mutator line that had about 30 copies of *Mu1*, using *Mu1* as a probe.

As a first step, we attempted to determine which *Mu1*-hybridizing band in a genomic Southern segregated with the *bz-Mum4* allele. The high number of bands makes the analysis difficult if individual plants are assayed, so we developed a pooled seedling assay, described below, that averages out the newly arisen *Mu1* bands that occur at each generation.

In a Mutator line with 30 copies of *Mu1*, approximately 15 of them will be passed to any one offspring in a mendelian fashion. Each individual in the progeny will receive a different subset of these parental copies. In addition, since *Mu1* tends to maintain its copy number at approximately 30, each offspring will also have about 15 new copies of *Mu1*. Analysis of individual offspring would show about 30 copies of *Mu1*, making it very difficult to follow segregation of a particular band because of the high background. However, if one pools the DNA from many plants which are selected for the mutable allele of interest and compares the pooled DNA with that of pooled sib plants not carrying the mutation in question, then the Southern analysis becomes slightly easier. The newly arisen copies of *Mu1* will not be the same in each plant. Therefore, in the pooled DNA these new bands will be diluted by the number of plants used, i.e., if 30 plants are used each new band will be 1/30th as dark as a regular band. Each parental band segregating independently of the selected mutable allele will be present in half of the progeny, and therefore will be half as intense as the band belonging to the selected gene. In addition, sib seeds not carrying the mutable allele will have all of the parental bands except those segregating with the targeted locus. Thus, the Southern of pooled DNA from plants selected for mutable vs. stable expression should show an intensity band difference.

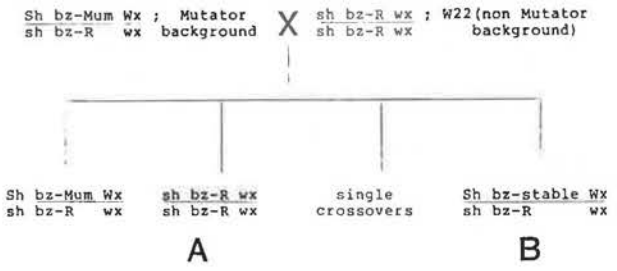
Using this approach it was still not possible to positively identify one band in genomic Southerns of *bz-Mum4* plants as corresponding to the bronze locus. However, a tentative assignment was made. A lambda library of 800,000 phage was constructed from a partial Sau3AI digest of genomic DNA and screened with a *Mu1* probe kindly provided by Mike Freeling. Twenty-seven independent phage were isolated and characterized by Southern blots. Several different phage had restriction fragments similar in size to the putative *bz-Mum4* Southern band. At about this time, we isolated a *bz*-specific probe from *bz-m2*, an *Ac*-mutable allele, so we used this probe in Southern blot hybridizations and to screen the *Mu1*-hybridizing phage. However, though the Southern analysis of *bz-Mum4* digests confirmed that a *Mu1*-hybridizing band also hybridized to the *bz*-specific probe, none of the isolated phage were found to contain *bz* homologous sequences.

We concluded that even if one could identify by Southern analysis which copy of *Mu1* was cosegregating with the target locus, cloning that gene would be very difficult because of the high copy number of *Mu1*. Similarly, in the cloning of the *a1*

locus, O'Reilly et al. (EMBO J. 4:877, 1985) found 1 correct clone out of 35 isolated and characterized. It is clear that if the *Mu1* copy number can be lowered, the job of cloning will be greatly reduced. The following report outlines how this might be achieved.

II. Reduction of copy number of Mu1: Observations made during the course of our genetic analysis of spotted *bz-Mum* alleles suggest that it may be possible to reduce the *Mu1* copy number. In our standard backcrossing scheme of *Sh bz-Mum Wx* to our W22 tester *sh bz-R wx*, we noticed in most ears a variable number of stable bronze seeds with the outside markers of the *bz-Mum* allele at too high a frequency (1 to 5%) to be double crossovers (Figure 1). Analysis of these stable bronze seeds showed that upon further backcrossing to our W22 tester or in selfs the phenotype remained bronze-stable; but upon crossing to *sh bz-R wx* sibs (i.e., extracted from a "Mutator background"), spotted seeds were again produced, although at a low frequency. This suggests that the change from a bronze-mutable to a bronze-stable phenotype was caused by the inactivation of the *Mu1* element's ability to transpose, and that subsequent exposure to a Mutator background reactivated this ability.

Figure 1



The inability of the *Mu1* element to transpose somatically apparently also reflects an inability to transpose germinally and thus an inability to maintain a high copy number. Test-cross progeny of the stabilized bronze plants described in the previous paragraph have a reduced copy number of *Mu1* elements, as low as 8, and Southern analysis of the individual offspring clearly shows that only one *Mu1*-hybridizing band is common to all (Figure 2). Hybridization with a bronze-specific probe confirms that this band corresponds to the bronze gene.

Modification of the *Mu1* element has been reported to be correlated with the stabilization of mutable *bz2* phenotypes (Chandler and Walbot, MNL 57:96, 1985) and with the loss of Mutator activity (Bennetzen, unpub.). We have verified that the *Mu1* element becomes modified when *bz-Mum* alleles change from a mutable to a stable null expression. At the bronze locus, the modification does not seem to extend beyond the *Mu1* element itself, because the HinfI sites in bronze are still cleaved in bronze-stable plants while the HinfI sites inside *Mu1* are not. In addition, we have found that, upon restoration of the mutable phenotype, most if not all copies of *Mu1* are cleaved again by HinfI.

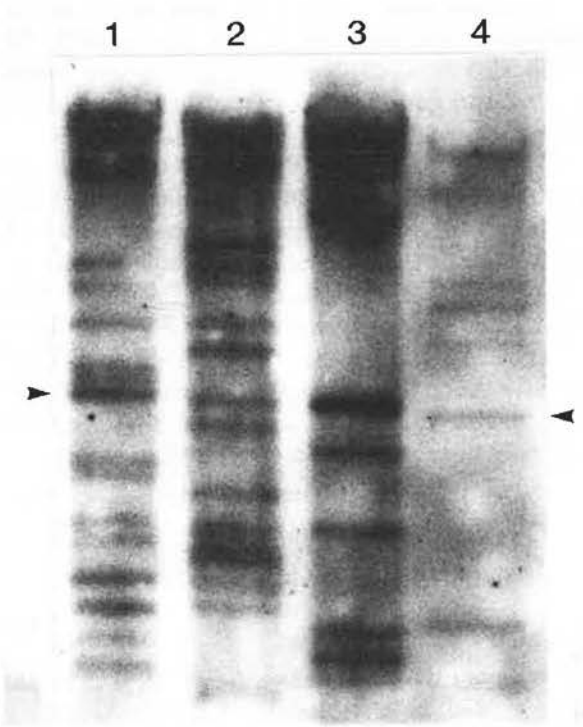


Figure 2. Southern blot of Pst-cut "miniprep" genomic DNA of progeny of a stabilized *bz-Mum* plant probed with *Mu1*. Lanes 1, 3, and 4: testcross progeny of a stabilized *bz* plant. Lane 2: plant with restored bronze-mutable phenotype obtained from a cross between plants A and B in Figure 1. The arrow marks the position of the common bronze hybridizing band.

The change from a mutable bronze to a stable bronze phenotype is very easy to detect even though the transposition events in *bz-Mum4* occur very late in seed development and, thus, the spotting pattern is very fine. The change from mutable to stable in a less easily scorable phenotype, (e.g., knotted, dwarf, nitrate reductase minus, etc.) would be hard, if not impossible, to detect. Thus, we propose to use the bronze-mutable to bronze-stable change as an indication of *Mu1* element activity, and envision the following scheme for identifying and isolating genes harboring a *Mu1* insertion:

1. Isolation of a mutant allele of the desired locus in an active Mutator line.

2. Crossing of the new mutant to a bronze-stable (ex *bz-Mum*) tester line where the *Mu1* element has become modified and does not transpose.

3. Backcrossing to the tester and selection of seeds in which *Mu1* has become modified as indicated by the stable bronze phenotype. One half of the plants derived from bronze seeds should be heterozygous for the mutation of interest and can be identified by selfing.

4. Repetition of the backcrossing scheme, determining at each generation the *Mu1* copy number of the individual offspring by Southern analysis. The

Mu1 copy number should be halved every generation, thus allowing the cloning of the desired locus with a *Mu1* probe 2 to 3 generations following the isolation of the mutant allele.

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The location of *Rf1*

Rf1, the dominant nuclear restorer of type T cytoplasmic male sterility, was mapped relative to *Lg3*, *Rg*, and *d*. The results shown below (Table 1) indicate that *Rf1* is about 3 cM distal to *Lg3*. This is probably an underestimate of the actual distance between *Lg3* and *Rf1* because one class of recombinants in this region was absent from test cross progeny. It is still unclear whether *Rf1* is on the short or long arm of chromosome 3 because the location of the centromere relative to *Lg3* and *Rg* is unknown.

Table 1: Progeny from the cross + + +, CMS-T X + + *Rf1* / *Rg Lg3* +

Parentals		Region 1		Region 2		Regions 1 & 2		Total
+ + *Rf1*	*Rg Lg3* +	+ *Lg3* +	*Rg* + *Rf1*	+ + +	*Rg Lg3 Rf1*	+ *Lg3 Rf1*	*Rg* + +	
183	166	1	4	10	-	-	-	
Totals 349		5		10				364
% recomb.		1.4		2.7				

R.S. Poethig

The location of *ra2*

Three-point test cross data involving *ra2*, *d*, and either *Rg* or *Lg3*, indicate that *ra2* is located about 6 cM proximal to *d* on the short arm of chromosome 3 (Table 1; Table 2).

Table 1: Progeny from the cross *Rg ra2 d*/ + + + X + *ra2 d*

Parentals		Region 1		Region 2		Regions 1 & 2		Total
Rg ra2 d	+ + +	*Rg* + +	+ *ra2 d*	*Rg ra2* +	+ + *d*	*Rg* + *d*	+ *ra2* +	
111	99	14	15	7	5	-	-	
Totals 210		29		12				251
% recomb.		11.5		4.7				

Table 2: Progeny from the cross *Lg3* + /+ *ra2 d* X + *ra2 d*

Parentals		Region 1		Region 2		Regions 1 & 2		Total
Lg3 + +	+ *ra2 d*	*Lg3 ra2 d* + + +		*Lg3* + *d*	+ *ra2* +	*Lg3 ra2* +	+ + *d*	
70	61	15	12	4	7	-	-	
Totals 131		27		12				170
% recomb.		15.9		7.0				

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Additional mapping of isozyme loci: Localization of *Acp4, Dia2, Adk1, Tpi3,* and *Sad1*

We recently listed the isozyme loci being studied by starch gel electrophoresis in our laboratory and summarized the available mapping data in a series of reports (Wendel et al., MGCNL 59:87-90). Subsequent work has resulted in further clarification of the chromosomal locations of markers on chromosomes 1 (*Acp4, Dia2*), 6 (*Adk1*), 8 (*Tpi3*), and 10 (*Sad1*). Previously unreported information on these loci and their chromosomal locations follows:

Chromosome 1 - Seven isozyme loci have been mapped on the long arm of chromosome 1: *Amp1, Mdh4, mmm, Pgm1, Adh1, Phi1*, and *Gdh1*. Joint segregation of several of these loci with two new isozyme markers, *Dia2* (Wendel et al., MGCNL 59:87-88) and *Acp4* (Kahler, J. Hered. 74:239-246), suggested that these loci also reside on chromosome 1. Their placement on 1L was confirmed by the testcross data of Table 1. These data indicate that *Acp4* and *Dia2* are

Table 1. Localization of Acp4 and Dia2 on the long arm of chromosome 1.

Cross A: Acp4-2 Dia2-6 Phi1-4 Adh1-4 / Acp4-3 Dia2-4 Phi1-8 Adh1-2 X Acp4-2/2 Dia2-4/4 Phi1-4/4 Adh1-4/4

Cross B: Dia2-6 Phi1-4 Adh1-4 / Dia2-4 Phi1-8 Adh1-2 X Dia2-4/4 Phi1-4/4 Adh1-4/4

	Parental Types	SCO region[1] 1	2	3	DCO regions[1] 1,2	1,3	2,3
Cross A	102	14	14	15	2	0	3
n=302	92	19	20	18	3	0	0
TOTALS	194	33	34	33	5	0	3
Recombination %		12.6	13.9	11.9			
(SE)		(1.9)	(2.0)	(1.9)			
Cross B	74	-	17	18	-	-	0
n=232	84	-	18	20	-	-	1
TOTALS	158	-	35	38	-	-	1
Recombination %			15.5	16.8			
(SE)			(2.4)	(2.5)			

[1]Regions 1,2, and 3 correspond to the segments Acp4-Dia2, Dia2-Phi1, and Phi1-Adh1, respectively. Each entry in the upper row begins with the upper left allele in the cross, viz. Acp4-2 for cross A and Dia2-6 for cross B. Each entry in the lower row begins with the lower left allele, viz. Acp4-3 for cross A and Dia2-4 for cross B. No triple crossovers were observed.

distal to all previously mapped isozyme loci on 1L, and collectively lead to the following map for the four most distal isozyme markers:

Acp4 - 12.6 - *Dia2* - 14.6 - *Phi1* - 14.0 - *Adh1*

Phi1 is currently thought to reside approximately at map position 140, and consequently, *Dia2* and *Acp4* should map to positions 155 and 167, respectively. *Acp4* may thus be the most distal marker known on 1L. Crossing experiments between *Acp4, Ts6* (tassel seed, 1-158), and *bm2* (brown midrib, 1-161) are underway in order to clarify the gene order among these terminal markers.

Chromosome 6 - Some preliminary data suggested that *Adk1*, the gene encoding adenylate kinase isozymes, segregated non-randomly with other isozyme markers on chromosome 6. Testcrosses of plants heterozygous for various combinations of the loci *Adk1*, *Pgd1* (phosphogluconate dehydrogenase), *Enp1* (endopeptidase), and *Hex2* (hexokinase) confirm that *Adk1* is on chromosome 6 (Table 2). Averaged across families (Kramer and Burnham, 1947; Genetics 32:379-390), there is 14.8% ± 1.9% recombination

Table 2. Localization of Adk1 near the centromere of chromosome 6.

Cross A: Adk1-5 Pgd1-2 Enp1-10 / Adk1-4 Pgd1-3.8 Enp1-6 X Adk1-4/4 Pgd1-3.8/3.8 Enp1-6/6

Cross B: Adk1-5 Pgd1-2 Enp1-10 / Adk1-4 Pgd1-3.8 Enp1-6 X Adk1-4/4 Pgd1-2.8/2.8 Enp1-2/2

Cross C: Adk1-5 Pgd1-3.8 Hex2-2 / Adk1-4 Pgd1-2 Hex2-1 X Adk1-4/4 Pgd1-3.8/3.8 Hex2-2/2

	Parental Types	Single crossovers[1] region1	region2	region3	Double crossovers
Cross A:	19	7	1		1
n=54	20	6	0		0
TOTALS	39	13	1		1
Recombination % (SE)		25.9(6.0)	3.7(2.7)		
Cross B:	92	13	2		2
n=224	100	10	4		1
TOTALS	192	23	6		3
Recombination % (SE)		11.6(2.1)	4.0(1.3)		
Cross C:	16	4		1	1
n=56	15	9		8	2
TOTALS	31	13		9	3
Recombination % (SE)		28.6(6.0)		21.4(5.5)	

[1]Regions 1, 2, and 3 refer to the Adk1-Pgd1, Pgd1-Enp1, and Pgd1-Hex2 segments, respectively. Each entry in the upper row begins with the upper left allele in the cross, viz. Adk1-5. Each entry in the lower row begins with the lower left allele, viz. Adk1-4.

between *Adk1* and *Pgd1*. These and previous data suggest the following gene order and map distances for isozyme markers on 6:

Adk1 - 14.8 - *Pgd1* - 3 - *Enp1* - 42 - *Hex2* - 43 - *Idh2* - 2 - *Mdh2*

The gene order among *Adk1*, *Pgd1*, and *Enp1* is consistent with the data of Table 2, and is additionally supported by previous work (Wendel et al., MGCNL 59:89-90). Because the B-A translocation stock TB-6Lc uncovers all markers distal to *Pgd1*, and because the TB-6Lc breakpoint is somewhere in the vicinity of the centromere (perhaps between 6-4 and 6-9) the above data suggest that *Adk1* is near the centromere, and perhaps even on 6S. It should be noted, however, that the recombination estimates between *Adk1* and *Pgd1* are heterogeneous across families, and consequently the distance between these two genes and the location on 6S for *Adk1* must be considered tentative. We are currently attempting to map all isozyme markers on chromosome 6 with respect to the morphological markers *rgd1* (ragged seedling, 6-4), *Y* (yellow endosperm, 6-13), *pg11* (pale green, 6-34), *su2* (sugary, 6-54), and *py1* (pygmy plant, 6-65).

Chromosome 8: Previous work has demonstrated that there is approximately 23% recombination between *Idh1* (isocitrate dehydrogenase) and *Mdh1* (malate dehydrogenase) on chromosome 8 (Goodman et al., Genetics 96:697-710). We crossed the B-A translocation stock TB-8Lc as male onto an *Idh1 -Mdh1* tester and uncovered *Idh1* (hypoploids recovered in 4 of 50 plants examined) but not *Mdh1*, suggesting that *Idh1* is on 8L, and *Mdh1* is either on 8L near the centromere or on 8S. Analysis of an F_2 segregating for *Idh1*, *Mdh1*, and *Tpi3* (triose phosphate isomerase) confirms the previous estimate of recombination between *Idh1* and *Mdh1* (30.0% ± 2.6%), and additionally suggests that *Tpi3* is on 8S approximately 22 map units from *Mdh1* (Table 3).

Table 3. Joint F_2 segregation data and maximum likelihood estimates of the recombination fractions (r) for three isozyme loci on chromosome 8 and for two isozyme loci on chromosome 10.

Locus pair	n	Genotypes[1] X_1Y_1	X_1Y_2	X_1Y_3	X_2Y_1	X_2Y_2	X_2Y_3	X_3Y_1	X_3Y_2	X_3Y_3	X^2 (DF)	r (SE)
Chromosome 8												
Tpi3-Mdh1	252	38	22	4	21	83	17	4	22	41	108.8(4)	.22(.02)
Tpi3-Idh1	252	3	35	26	32	63	26	25	24	18	23.9(4)	.39(.03)
Mdh1-Idh1	252	3	25	35	28	78	21	29	19	14	56.9(4)	.30(.03)
Chromosome 10												
Glu1-Sad1	208	53	1	0	0	104	1	0	2	47	392.0(4)	.01(.005)

[1]X and Y refer to the first and second locus listed for each locus pair. Genotypes are as follows: Tpi3: X_1=2/2; X_2=2/4; X_3=4/4. Mdh1: Y_1=6/6; Y_2=6/.65; Y_3=.65/.65, except substitute X for Y in the Mdh1-Idh1 line. Idh1: Y_1=2/2; Y_2=2/4; Y_3=4/4; Glu1: X_1=1/1; X_2=1/2.5; X_3=2.5/2.5. Sad1: Y_1=4/4; Y_2=4/6; Y_3=6/6.

Tpi3 thus becomes only the second factor (along with *Bif1*, barren inflorescence) mapped on the short arm of the most poorly marked chromosome of maize. With the available data it is not possible to determine whether *Mdh1* is on 8S or 8L. Testcrosses of the three isozyme loci with the morphological markers *fl3* (floury, 8-0) and *j1* (japonica striping, 8-42) will be analyzed in 1986.

Chromosome 10: An additional isozyme marker on chromosome 10 was confirmed through the analysis of the selfed progeny of an F_1 that was heterozygous for both *Glu1* (β-glucosidase) and *Sad1* (shikimate dehydrogenase). These data (Table 3) demonstrate tight linkage between *Glu1* and *Sad1*, with 1.0% ± 0.5% recombination. Efforts are being made to map these markers relative to various morphological markers on chromosome 10.

Jonathan F. Wendel, Major M. Goodman
and C.W. Stuber

Origin of the S-1 plasmid-like DNA molecule of maize mitochondria

The mitochondria of certain maize cytoplasms contain linear plasmid-like DNAs in addition to the main mitochondrial genome. The male-sterile cytoplasm, *cms-S*, has two plasmid-like DNA molecules. These are designated S-1 and S-2 and are 6.4 kilobases (kb) and 5.4 kb long, respectively. A few male-fertile South American maize races, such as Racimo de Uva, also contain two plasmid-like DNA molecules, R-1 and R-2, which are 7.5 kb and 5.4 kb long, respectively. Restriction mapping and heteroduplexing studies reveal that R-2 and S-2 are nearly identical. In contrast, S-1 contains regions of homology with R-1 and R-2. It has been suggested that S-1 may have arisen from a recombinational event between R-1 and R-2 (Figure 1) (see review in Plant Gene

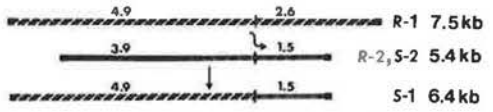

Figure 1. Homologies between the various plasmid-like DNAs. Vertical lines indicate probable recombination point. Curved arrow illustrates recombination event and straight arrow illustrates the resulting S-1 hybrid.

Research, Ch. 6, B. Hohn and E. S. Dennis, Ed., Springer-Verlag, New York).

To investigate this possibility, a 458 base pair (bp) HindIII-BamHI fragment, believed to contain the site of recombination, was isolated from R-1 (Racimo de Uva) and sequenced using the dideoxynucleotide chain termination protocol. This sequence was compared with the sequences of S-1 (EMBO J., 4:1125, 1985) and S-2 (Proc. Natl. Acad. Sci. USA, 80:4055, 1983). The comparisons show well defined regions of sequence homology and nonhomology that indicate the probable point of recombination (Figure 2).

```
R-1   341 GTTATAGAGA GACCGCAACC CCTTATATTC ACTGAAATAT  380
          ********** ********** ********** **********
S-1  4864 GTTATAGAGA GACCGCAACC CCTTATATTC ACTGAAATCT 4903
            ***  **  **   *   * ****
S-2  3920 TCTATTTAGT GATGTATAAA GCGTATAGCT TAGATTTAGC 3959

                                          ↓
R-1   381 GCACGATAAA TCTTTCTTTT CAATTTTAAA CGGAGCAACG  420
          ********** ********** ********** ** *  * *
S-1  4904 GCACGATAAA TCTTTCTTTT CAATTTTAAA CGCATGATCA 4943
          *  *** *  *   **   *  *  *        ********
S-2  3960 GTTTGATTAT TTCAAGTTGA AATGATATTT GTCATGATCA 3999
                                          ↑

R-1   421 AGCGCATATA AAGATAGTGC ATACGCATGA TAGGATCC    458
          *       *  *  *     *  *  * *
S-1  4944 ATACATATCA CTAAAGATTT CGTTTTTATT AACCTTTC   4981
          ********** *********   ********* ********
S-2  4000 ATACATATCA CTAAAGATTG AGTTTTTATT AACCTTTC   4037
```

Figure 2. Sequence comparison of R-1, S-1, and S-2 presented in a 5′ to 3′ direction. Arrows indicate point of recombination. The R-1 sequence from nucleotide 1 to 412 has perfect homology with the S-1 sequence from nucleotide 4864 to 4935 (not all data shown); beyond that point R-1 and S-1 have no significant homology. S-1 has nearly perfect homology with S-2 beginning at nucleotide 4936 in S-1 and 3992 in S-2, continuing to their 3′ termini. Prior to that point, S-1 and S-2 do not contain significant homology (not all data shown). S-1 and S-2 vary from each other at positions 4963,4964 and 4019,4020, respectively, in the homologous region.

The probable point of recombination occurs between bases 4935 and 4936 in S-1, bases 3991 and 3992 in S-2, and bases 412 and 413 in R-1 (Figure 2). Because S-2 and R-2 are virtually identical, S-2 was used for comparison.

There are no large open reading frames (ORFs) in this segment of S-2, but the HindIII-BamHI R-1 and corresponding S-1 strand contain a large unidentified reading frame (URF) that continues through the recombination point and beyond the HindIII site. This URF is homologous with the 768 bp URF-4 in S-1 (EMBO J., 4:1125, 1985). A search of BIONET protein libraries has revealed no significant homologies. Finally, we believe our results support the view that S-1 arose by a recombination between R-1 and R-2.

Susan Elmore-Stamper and C. S. Levings III

DNA sequence homology between the maize mitochondrial and chloroplast genomes

From a BamHI library of *cms-T* mitochondrial DNA, a 5.5 kbp cloned fragment, designated as T7, was isolated and shown to contain sequence homology to chloroplast DNA. Chloroplast homology was shown to be within a 1.95 kbp EcoRI to BamHI fragment of the T7 clone, and the nucleotide sequence of this fragment was determined. Subclones for DNA sequencing were generated by unidirectional exonuclease III digestion of the 1.95 kbp fragment (Henikoff, 1984, Gene 28:351). Homology to the 3′-end of the 23S rRNA, all of the 4.5S and 5S rRNAs, and to a nearly complete copy of the $tRNA_{Arg}$ gene was found on a 1270 bp contiguous stretch of DNA. Bionet computer programs were used to identify and compare regions of homology. Where the maize chloroplast sequence has been determined, the DNA sequence homology was greater than 90%; comparisons with other chloroplast sequences from tobacco and *Spirodela* showed approximately 85% homology.

In the chloroplast genome the 23S-4.5S-5S-$tRNA_{Arg}$ cluster of genes is located with the inverted repeats. In the T7 clone, the gene order is the same as in the chloroplast DNA. Moreover, the spacing and degree of sequence homology of the small intergenic regions is similar between the chloroplast DNA and the inserted DNA in clone T7. The organizational conservation of the chloroplast genes and their intergenic spaces suggested that the transfer of this DNA occurred as a single event. Interestingly, portions of the T7 clone are duplicated elsewhere in the mtDNA of T cytoplasm. Partial homology to the T7 clone is found in a 3.1 kbp BamHI fragment. Additionally, the partial $tRNA_{Arg}$ sequence has been found within a 9.0 kbp BamHI fragment (Dewey et al., 1986, Cell, in press).

Previous reports of homologous sequences between the chloroplast and mitochondrial genomes suggest that several interorganelle exchanges of DNA may have occurred (Stern and Lonsdale, 1982, Nature 299:698; Lonsdale et al., 1983, Cell 34:1007; Stern and Palmer, 1984, PNAS 81:1946). DNA sequence comparisons at the junctions of chloroplast DNA insertions may help elucidate the mechanism(s) by which these organelle exchanges occur.

C. J. Braun and C. S. Levings III

Cms-LBN differs from *cms-L*, its progenitor cytoplasm

In a survey of 25 S-group maize cytoplasms in 36 inbreds, one inbred/cytoplasm combination, *cms-L* in the inbred strain W182BN, was found to contain two highly abundant double-stranded RNA (dsRNA) molecules. *Cms-L* in W182BN was given a new designation, "*cms-LBN*", because *cms-L* in other inbred backgrounds did not contain these abundant

dsRNAs (Sisco et al., MNL 56:82, 1982). Three hypotheses to explain the presence of these dsRNAs in W182BN *cms-LBN* were:

1) These dsRNAs resulted from a mutation in *cms-L* during the backcrossing process to inbred W182BN. This would mean that *cms-LBN* was genetically different from *cms-L*.

2) These dsRNAs were simply an effect of the W182BN nuclear genome. This would mean that any *cms-L* cytoplasm backcrossed to W182BN would eventually gain these dsRNAs and that the designation *"cms-LBN"* should be discarded.

3) These dsRNAs were the genome of a virus that had infected W182BN *cms-L* plants. This would mean that the dsRNAs might be unrelated either to the cytoplasmic or nuclear genotype of the infected plant.

Evidence against hypothesis (3) was presented by Schuster et al. (pp. 437-444 in R. Goldberg, ed., *Plant Molecular Biology*, Alan R. Liss, New York, 1983), who showed that single-stranded RNAs homologous to the *cms-LBN* dsRNAs were present in all S-group cytoplasms and in RU, a type of male-fertile cytoplasm. Recently Finnegan and Brown (Abstract OR-08-06, First Int'l. Cong. of Plant Molec. Biol., Savannah, GA, 1985) have reported data suggesting that the dsRNAs are components of a mitochondrial plasmid found in S-group cytoplasms.

Hypothesis (2) was still likely, however, because Sisco et al. (Plant Sci. Lett. 34:127, 1984) showed that there was a strong effect of nuclear background on the abundance of the dsRNAs. Of 10 inbred backgrounds into which *cms-LBN* was crossed, eight reduced the amount of the dsRNAs and only two, W182BN itself and a sweet corn inbred strain 2132, maintained the high level of the dsRNAs. To further test hypothesis (2), V. E. Gracen at Cornell crossed five sources of *cms-L* as female to the inbred strain W182BN. After four crosses to W182BN, our laboratory analyzed the mitochondrial nucleic acids. The abundant dsRNAs characteristic of *cms-LBN* were not found.

It thus appears that hypothesis (1) is the most likely explanation, and that *cms-LBN* is different from its progenitor cytoplasm *cms-L*.

Paul H. Sisco

No linkage found between *Rf4* and *v16* or *j* on *8L*

In the 1984 Newsletter (MGNL 58:101), Johnson reported data indicating that *Rf4*, which restores fertility to *cms-C* cytoplasm, was on 8L linked to the *wx* T8-9(6673) breakpoint. Crosses were made by Johnson and V. E. Gracen at Cornell to determine whether *Rf4* is linked to *v16* or *j*, two markers on 8L. F_2 progeny were scored at Raleigh, North Carolina, but no linkage of *Rf4* with either marker was apparent (see data below). Note, however, that the

$$\left(\frac{Rf4\ +\ +}{rf4\ v16\ j} \times cms\text{-}C\right)\otimes$$

Class	#
Rf4 + +	147
rf4 + +	54

Class	#
Rf4 v16 +	45
rf4 v16 +	14

Class	#
Rf4 + j	56
rf4 + j	12

Class	#
Rf4 v16 j	12
rf4 v16 j	2

Two-point analyses: χ^2

Rf4 vs. v16	Rf4 + 203	Rf4 v16 57	rf4 + 66	rf4 v16 16	2.36	P>.10
Rf4 vs. j	Rf4 + 192	Rf4 j 68	rf4 + 68	rf4 j 14	2.58	P>.10
v16 vs. j	+ + 201	v16 + 59	+ j 68	v16 j 14	3.07	P<.10
Expected 9:3:3:1	192.4	64.1	64.1	21.4		

weak linkage between *v16* and *j* (28 m.u.) was not detected by this F_2 analysis. It is possible that *Rf4* is on 8L but proximal to *v16*, since the reported breakpoint of *wx* T8-9(6673) is 8L.35, while TB8-La at 8L.70 uncovers *v16*. Alternatively, the *Rf4* locus may not be on Chromosome 8, because remnant seed of the *wx* T8-9(6673) cross grown out at Raleigh produced very weak plants that could have been male-sterile for many reasons. Laughnan and Gabay-Laughnan (Ann. Rev. Genet. 17:27, 1983) said that data from their studies indicated at least two *cms-C* restorers, one of which was on chromosome 2. The data presented here are consistent with Kheyr-Pour et al.'s findings (Genetics 98:379, 1981) that the inbred A619 has a single major restorer gene for *cms-C* restoration. This gene was designated *Rf4* by Kheyr-Pour.

A *v16 j* source from the Maize Genetics Cooperation (Coop 78-630-8 self) was crossed as male to A619 *cms-C (Rf4/Rf4)* and to NyD410 *cms-C (rf4/rf4)*. The NyD410 control was male-sterile in the F_1, showing that the *v16 j* source did not carry restorer genes for *cms-C*. The F_1 of the A619 cross was selfed, and the F_2 progeny were scored.

P. H. Sisco

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Tpi4 is located near the centromere on the long arm of chromosome 3

Tpi4, one of the three genes encoding cytosolic triose phosphate isomerase isozymes, was earlier shown to be on 3L between *Pgd2* (phosphogluconate dehydrogenase) and the centromere (Wendel et al., MNL 59:88). In an effort to better localize *Tpi4*, a series of crosses was made between *Tpi4* testers and stocks carrying three B-A translocations believed to be near the centromere on 3L. The data are as follows:

Cross no.	Translocation	No. tested
1	TB-3La	14
2	TB-3La	14
3	TB-3La	14
4	TB-3Lf	14
5	TB-3Lf	14
6	TB-3Lg	14
7	TB-3Lg	14

No plants hypoploid for *Tpi4* were recovered in any of the crosses; consequently, *Tpi4* must lie proximal to translocation breakpoints and be near the centromere. This suggests a location close to *Lg3* (liguleless) and *Rg1* (ragged leaves) and proximal to *gl6* (glossy), which is uncovered by TB-3La, - 3Lf and -3Lg. F2's and testcrosses involving *Hex1* (on 3S), *Lg3*, and *Tpi4* will be evaluated next year.

J. F. Wendel and J. B. Beckett

Cms-ME (38-11) could be a Wf9-type revertant cytoplasm

An analysis of 25 accessions of S-group male-sterile cytoplasms maintained at Cornell showed heterogeneity within the *cms-ME* cytoplasms (Sisco et al., Theor. Appl. Genet. 71:5, 1985). Those *cms-ME* cytoplasms descended from *cms-ME* in inbred N6 were standard in mitochondrial DNA and fertility restoration, whereas those descended from *cms-ME* in inbred 38-11 were almost fully fertile and had a distinctive mitochondrial DNA restriction pattern. The 38-11(ME) types had not lost the S-1 or S-2 plasmid-like DNAs, however, as is characteristic of many S-group revertants to fertility (Levings et al., Science 209:1021, 1980).

To survey other sources of *cms-ME*, seed from Beckett's cold storage was grown out. *Cms-ME* in three other inbred backgrounds—Tr, Wf9, and W23—had the mitochondrial DNA rearrangements and male-fertile phenotype of 38-11 *cms-ME* and its descendants. Beckett's records show that 38-11(ME), Tr(ME) and W23(ME) all trace back to a single plant that was pollinated by Wf9 (Beckett row 61-304), whereas N6(ME), which has standard S-group mitochondrial DNA and male-sterility, was not pollinated by Wf9. Wf9 is known to cause reversions to fertility of S-group cytoplasms (D. F. Jones, MGCNL 28:19, 1954; 29:14, 1955) and recently Escote, Laughnan, and Gabay-Laughnan have found that these reversions do not involve the loss of the S-1 and S-2 plasmids (Plasmid, in press).

A reasonable hypothesis is that the unusual *cms-ME* phenotype found in the descendants of Beckett row 61-304 is due to a Wf9-type reversion to fertility.

P. H. Sisco and J. B. Beckett

ST. LOUIS, MISSOURI
Monsanto Co.

A genetic map for a segment of the long arm of chromosome 6

Test cross data presented in Tables 1, 2, and 3 provide insight into the arrangement of genes in the region adjacent to the centromere in the long arm of chromosome 6. While these data do not provide precise information on the distances between these mutants they do support the following order for the mutants in this region:

Linkage map of the long arm of chromosome 6

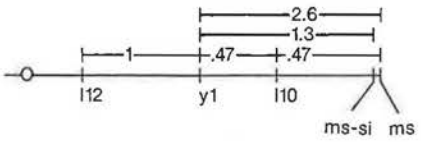

Table 1: Relationships among I12, y, and ms

F1 genotype:
I12 y +
\+ + ms

GENOTYPE OF PROGENY	FREQUENCY
I12 y +	274
+ + ms	209
I12 + ms	1
+ y +	4
I12 y ms	8
+ + +	5
TOTAL	501

I12 y ms
_|___|____|
|---1--|--2.6--|

Table 2: Relationships among I10, y, and ms

F1 genotype:
\+ + ms
y I10 +

GENOTYPE OF PROGENY	FREQUENCY
+ + ms	232
y I10 +	191
+ I10 +	1
y + ms	1
+ + +	1
y I10 ms	1
TOTAL	427

y I10 ms
__|___|____|
|-.47-|---.47--|

Table 3: Relationships among y, ms-sl, and ms

F1 genotype:
y ms-sl +
+ + ms

GENOTYPE OF PROGENY	FREQUENCY
y ms-sl +	40
+ + ms	34
y + ms	1
+ ms-sl +	0
y ms-sl ms	0
+ + +	0
TOTAL	75

y ms-sl ms
_|_____|_____|_
|--1.3-|--?--|

Peter N. Mascia and Dale F. Loussaert

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University of Minnesota

Chromosomal location of a gene controlling high-methionine zein expression

The elevated protein-bound methionine in maize line BSSS-53 is accounted for by an increase in both the proportion and methionine content of sulfur-rich, low molecular weight proteins (10kD) in the zein-2 fraction (R.L. Phillips and B.A. McClure, Cereal Chem. 62:213-218, 1985). Expression of the major 10kD zein segregates in crosses between inbreds differing in methionine content. We were able to map a gene responsible for overexpression of this high-methionine protein to the short arm of chromosome 4.

Segregation for protein expression was visually detected by isoelectric focusing. Zein-2 was extracted from BC and F_2 progeny from crosses between BSSS-53 and W23 waxy translocation and marker stocks. Recombination values of 35.5% and 33.9% were calculated by maximum likelihood from crosses involving *wx* T4-9g and floury-2, respectively (Tables 1 and 2). An IEF band corresponding in position to *Zp22/6* (formerly *Zp6*) showed complete linkage with the major 10kD band. This would place the gene near *Ga1*. Preliminary data involving translocations of the long arm of chromosome 4, *wx* T4-9B, *wx* T4-9 (5657), further support a gene location on the short arm. Independent segregation was observed in crosses involving opaque-2.

We propose the gene symbol *Zpr10/(22)* to represent this regulator of the 10kD zein polypeptide with tentative IEF position 22 (H. Hastings, S. Bonanomi, C. Soave, N. Di Fonzo, and F. Salamini, Genet. Agr. 38:447-464, 1984). The overexpression factor may be a unique regulatory gene or a modification of the structural gene.

M.S. Benner and R.L. Phillips

Table 1: Segregation of 10kD zein overexpression [Zpr10/(22)] and waxy in BC progeny from the cross [BSSS-53 X W23 wx[T4-9g]] X W23 wx

	Zpr10/(22) / +	+ / +	total
+ / wx	42	19	61
wx / wx	25	38	63
total	67	57	124

Table 2: Segregation of 10kD zein overexpression [Zpr10/(22)] and floury-2 in F_2 progeny from (BSSS-53 X 937 f12) ⊗

	Zpr10/(22) / Zpr10/(22)	Zpr10/(22) / +	+ / +	total
+ / +	36	10	0	46
+ / f12	17	70	23	110
f12 / f12	2	22	29	53
total	55	102	52	209

A tissue culture-induced mtDNA mutation reverts during a second tissue culture period

Rearrangements of mitochondrial DNA (mtDNA) occur during culture and regeneration of plants from corn tissue cultures. One specific rearrangement recovered in plants from T cytoplasm cultures results in the loss of the largest BamHI restriction fragment and in the appearance of two new fragments (R.J. Kemble and D.R. Pring, 1982, Plant Infection, Springer-Verlag. p. 187-195; B.G. Gengenbach and D.R. Pring, 1982, Maize Biol. Res. p. 257-262).

We were interested in determining whether this rearrangement was stable during a second tissue culture period or whether reversions to the original T genome arrangement or to new arrangements would occur. The seventh seed generation of mutant line R2 (B.G. Gengenbach, et al., 1981, TAG 59:161-167) was the source of embryos for initiation of new tissue cultures. This R2 mutant line lacked the largest Bam fragment and had two new fragments compared with the standard T arrangement (Figure 1A and B). Plants were regenerated from R2 cultures and progeny lines were analyzed for mtDNA changes. Thirty-nine lines retained the same pattern as the R2 culture line (Figure 1C), but one line had a Bam restriction digest pattern similar to that of standard T (Figure 1D).

These results suggest either that the mtDNA organization of R2 could revert back to the nonmutant T pattern or that a heterogeneous mitochondrial population was maintained for seven sexual generations, from which assortment during culture resulted in an apparent homogeneous nonmutant T mtDNA population. Reversion of the mutant, perhaps via recombination within duplicate mtDNA regions, to the nonmutant arrangement seems more likely than the alternative of heterogeneity. More detailed molecular analyses in progress should resolve this issue.

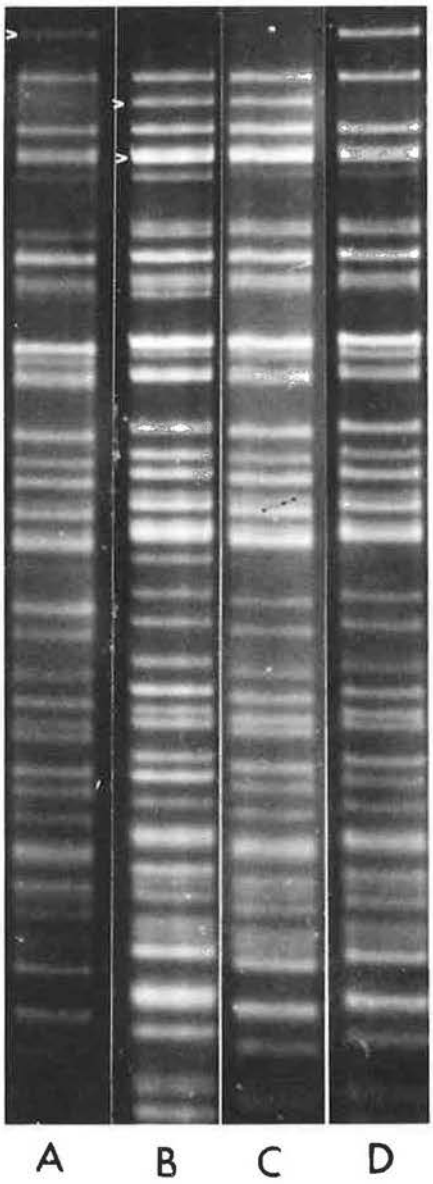

Figure 1. Mitochondrial DNA from A188 inbred line versions of T cytoplasm (A); mutant R2 (B); a nonrevertant line representative of 39 lines regenerated from tissue cultures of mutant R2 (C); and the one revertant regenerated sister line (D). The mtDNA was digested with Bam HI and electrophoresed on 0.7% agarose gels for 30 hr.

Burle Gengenbach, Holly Jessen and Kathleen Storey

Allelism test for two threonine over-producing mutants

Green and Phillips (Crop Sci. 14:827-830, 1974) first suggested a method for selecting amino acid over-producing mutants of the aspartate pathway. The addition of lysine plus threonine to corn tissue culture media inhibits callus growth by feedback inhibiting enzymes in the aspartate pathway. The inhibitory effect of lysine plus threonine can be overcome by the addition of methionine.

Two mutants resistant to increased levels of lysine plus threonine were isolated from tissue culture. The first mutant was designated LT19 (K.A Hibberd and C.E. Green, Proc. Nat. Acad. Sci. 79:559-563, 1982). A second mutant, LT20, was isolated by Diedrick (Ph.D. thesis UM-St. Paul, 1984). Both mutants essentially behave as though controlled by single dominant genes and have elevated free threonine levels (20- to 100-fold) in mature kernels.

The objective of this study was to determine whether LT19 and LT20 are allelic forms of the same gene. F1 plants obtained from crossing homozygous LT19 and LT20 lines were selfed and testcrossed to A619 wildtype plants. F2 and testcross kernels were classified for free threonine concentration by TLC separation of 5% TCA extracts of whole kernels. Segregation of mutant and wildtype kernels was determined by scoring for the intensity of the threonine spot.

Wildtype F2 segregants were found in a frequency of 30 wildtype to 620 mutants. This ratio deviates slightly ($p = .10$) from the 15:1 ratio expected for independent duplicate dominant genes. The expected number of wildtype kernels was 41; their deficiency could be from misclassification due to threonine contributed by the maternal F1 plant.

The testcross kernels segregated 127 mutants to 73 wildtype. This ratio is significantly different ($p < .005$) from the 3:1 ratio expected for independent duplicate dominant genes. This ratio is not significantly different from a 2:1 ratio, suggesting that pollen transmission of one or more mutant gametes might be affected. The reciprocal cross has not been analysed yet.

This study shows that the LT19 and LT20 mutants have two non-allelic genes conditioning the same phenotype. It remains to be seen if the two genes affect different enzymes or code for alternate isozymes of the aspartate pathway.

David A. Frisch and Burle G. Gengenbach

A suppressor-mutator transposable element system of independent origin

An ear segregating for a blistered-appearing kernel phenotype was given to C.R. Burnham by the Northrup King Company about 1960. Dr. Burnham discovered that the mutant when self-pollinated could produce ears also segregating for brittle-1. This material was provided to R.L. Phillips in about 1963 for further tests. After considerable effort, the hypothesis was developed that the blistered phenotype was a result of the kernel being mosaic for *bt1*. Tester stocks kindly provided by B. McClintock and P.A. Peterson allowed the discovery that a two-element transposable element system comparable to *Spm* and *En* was operating, with a non-autonomous element being present at the *bt1* locus. We have referred to this system as *Spm-P* because its independent origin may mean that it is unique, although the

Table 1. Linkage tests with Spm^P (Spm-P a2/wx T X a2-m-1 wx).

Cross	Colorless (a2) Wx,Spm	Wx,+	wx,Spm	wx,+	Colorless (a2)	Colored (A2)	Percent Recombination
2-9b							
25966-1 x 992-27	26	39	22	32	119	97	
25966-2 x 993-60	39	7	33	9	88	67	18.2
25966-3 x 992-32	3	2	2	4	11	12	
25966-4 x 992-27	48	12	59	11	130	113	17.7
25966-6 x 994-48	35	11	25	10	81	85	25.9
25966-15 x 994-15	25	8	18	4	55	59	21.8
2-9b							
25982-4 x 996-66	34	3	30	5	72	61	11.1
25982-12 x 996-71	14	17	17	16	64	71	
25982-14 x 993-53	25	10	20	20	75	93	
25982-15 x 996-47	34	4	31	6	75	67	13.3
3-9c							
25984-2 x 996-54	61	3	56	3	123	99	4.9
25984-9 x 993-77	14	6	18	9	47	48	
25984-12 x 994-52	37	4	27	7	75	84	13.1
25984-13 x 994-59	15	2	7	4	28	17	
4-9b							
25968-2 x 994-18	63	1	58	1	123	122	1.6
25968-3 x 994-8	84	8	48	5	145	123	9.0
25968-12 x 993-22	29	13	17	10	69	69	33.0
25968-13 x 993-22	73	0	63	2	138	131	1.4
4-9g							
25969-5 x 995-8	60	2	45	5	112	113	6.3
25969-8 x 994-7	48	5	54	2	109	95	6.4
25969-9 x 993-2	58	3	36	2	99	79	5.1
25969-16 x 995-17	71	9	48	8	136	109	12.5
5-9a							
25971-1 x 994-32	51	14	49	3	117	108	14.5
25971-5 x 993-4	51	26	38	19	134	127	33.6
25971-8 x 993-14	64	19	56	26	165	164	27.3
25971-11 x 995-15	65	3	57	6	131	109	6.9
25971-13 x 993-9	49	6	52	3	110	82	8.2
5-9a							
25986-2 x 996-93	38	39	29	20	126	95	
25986-6 x 996-92	63	35	62	36	196	199	36.2
25986-7 x 994-60	45	50	39	49	183	159	

Cross	Colorless (a2) Wx,Spm	Wx,+	wx,Spm	wx,+	Colorless (a2)	Colored (A2)	Percent Recombination
5-9c							
25987-1 x 993-9	57	4	0	1	62	76(74=wx)	8.1
25987-3 x 993-40	85	0	5	1	91	112(110-wx)	1.1
25987-5 x 993-70	124	0	4	0	128	120(117=wx)	0
25987-2 x 993-40	87	6	4	0	97	101(98=wx)	6.2
6-9a							
25988-10 x 994-39	68	18	69	16	171	162	19.9
25988-11 x 996-90	57	20	61	10	148	144	20.3
25988-13 x 996-76	27	35	32	38	132	131	
7-9a							
25975-1 x 995-14	24	11	26	5	66	68	24.2
25975-2 x 996-3	41	11	23	11	86	108	25.6
25975-3 x 996-11	41	16	35	16	108	104	29.6
25975-4 x 996-15	39	20	53	20	132	109	30.3
7-9g							
25989-1 x 995-6	49	38	42	45	174	177	
25989-2 x 993-71	72	1	53	3	129	109	3.1
25989-10 x 995-13	44	38	41	33	156	154	
25989-11 x 995-15	62	4	53	8	127	162	9.4
8-9d							
25977-11 x 993-33	32	8	21	3	64	63	17.2
25977-12 x 993-62	39	37	12	27	115	116	
25977-16 x 993-21	19	9	13	6	47	60	
8-9(6673)							
25978-11 x 996-27	52	3	37	3	95	119	6.3
25978-13 x 996-5	45	29	39	26	139	132	
25978-14 x 995-19	75	7	54	5	141	116	8.5
25978-16 x 995-24	40	9	48	7	104	88	15.4
8-9(6673)							
25990-1 x 993-84	71	15	54	15	155	156	19.4
25990-2 x 996-83			No Spm Present				
25990-3 x 993-82	50	0	49	1	100	76	1.0
25990-8 x 996-86	47	1	38	3	89	104	4.5
9-10b							
25979-6 x 993-74	45	17	49	12	123	134	23.6
25979-9 x 993-54	17	1	9	1	28	26	7.1
25979-13 x 993-75			No Spm Present				
25979-14 x 994-2	34	2	33	2	71	61	5.6
9-10b							
25991-1 x 994-11	24	15	35	9	83	94	28.9
25991-2 x 994-11	58	7	59	2	126	106	7.1
25991-3 x 994-24	21	20	22	19	82	75	
25991-8 x 994-9	41	40	45	44	170	139	

genetic tests have not revealed any differences via tests with McClintock's or Peterson's stocks.

Recently we have performed mapping tests in an attempt to locate *Spm-P*. We crossed the *Spm-P* stock (which is *a2 a2*) with members of the waxy translocation series and testcrossed to *a2-m-1 wx*.

We analyzed 111 crosses involving the waxy translocation series. Independence of a single *Spm-P* element with *a2* and *wx* was evident in all crosses involving T1-9c, 1-9(8389), 1-9(4995), 2-9d, 4-9(5657), 6-9b, and 7-9(4363). Linkage of *Spm-P* and *a2* was indicated in 43 crosses; these data are presented in

Table 1. We are discounting those cases where the apparent linkage value is around 25 map units, because this could simply reflect the independent segregation of two *Spm-P* elements. This result was obtained in crosses involving T2-9b (5 cases), T4-9b (1 case), T5-9a (1 case), T6-9a (2 cases), T7-9a (4 cases), T8-9d (1 case), T8-9(6673) (2 cases), and T9-10^{b} (2 cases). A linkage value of less than 10 map units between *Spm-P* and *a2* was found with crosses involving T3-9c (1 case), T4-9b (3 cases), T4-9g (3 cases), T5-9a (2 cases), T5-9c (4 cases), T7-9g (2 cases), T8-9(6673) (4 cases), T9-10b (3 cases). Linkage values of 10-15 map units were obtained with crosses involving T2-9b (2 cases—but 1 case fits the 2 *Spm-P* model), T3-9c (1 case), T4-9g (1 case), and T5-9a (1 case). Linkage values of 30-40 map units were obtained in two cases with T5-9a. Crosses involving culture 25987 (T5-9c) indicated linkage of *Spm-P* and *wx*, but this probably is because *a2* and *wx* are linked due to the T5-9c translocation.

These results indicate that *Spm-P* is often linked to *a2* on chromosome 5. The frequency of independence is similar to that reported by Nowick and Peterson (1981, Mol. Gen. Genet. 183:440-448) for the movement of *En* from its initial site.

R.L. Phillips, L.G. Block, V.M. Peschke and C.R. Burnham

Tests for a cytoplasmic fertility restorer for *ms1*

Tests to identify plants that are *ms1 ms1* but fertile because of an [R] cytoplasm that restores fertility were continued with the line derived from T6-9b. Plants shown to be *Ms1 ms1* by test-crosses on *ms1 ms1* produced self progenies that had no male sterile progeny. Plants in those progenies were selfed and test-crossed on *ms1 ms1* plants, of the 61 plants tested 42 were shown to be *Ms Ms* and 19 were *Ms ms*. None were *ms1 ms1* but fertile. Progeny from selfs of the *Ms ms* plants were grown only in small numbers. Larger numbers will be grown in 1986. These should have no male sterile plants if the line being tested has the restorer cytoplasm.

For testing additional stocks for a cytoplasm that restores fertility to a genetic male sterile, an interchange stock (T) with one breakpoint closely linked to the male sterile may be used. The stock to be tested is then crossed with pollen from T/*ms*. Except for crossovers between *ms* and the T breakpoint, the fertile progeny will be *Ms ms*. If these fertile plants are again crossed as female with T/*ms*, half the fertile progeny should be *ms ms*. This will provide a test of the ability of the stock to restore fertility. It will also increase the efficiency of identifying plants that are [R] *ms ms*. The first step was used to a limited extent in this study, but the usefulness of the second step was not realized until late this summer.

Chas. Burnham

pr gl8 stock for chromosome 5

These genes are very closely linked in chromosome 5L. In 1984, ears including selfs and crosses, segregating for this combination were tested in the greenhouse. The ears appeared to have no disease, but none germinated in two trials. In a repeat trial, a few seedlings were obtained. One survived transplanting in a small 4" pot. After being transferred to a larger pot when the tassel appeared, a small ear segregating for *Pr*, *pr* and colorless aleurone was obtained from open pollination (no other corn was flowering at the time). Some of the seed was shared with Dick Whalen, and the remaining *Pr* and colorless classes were grown here at Minnesota. Greenhouse tests of ears from selfs and also crosses show that the *pr* seeds from segregating ears are also *gl*. Stocks are being sent to the Coop.

One reason for this account is to call attention to the fact that corn plants can be grown in very small pots, and yet will produce ears with a usable number of seeds if transferred to larger pots when they are about to tassel. A little fertilizer at the same time helps.

Chas. Burnham

su gl4 la

Ears with this combination are now available, and also F_1 ears that can be used to produce material for demonstrating three-point linkage. This can be done in pots in the greenhouse. When the pots are laid on their sides, the normal, *La* plants will turn upward, the lazy plants will not. This provides a three-point test for *la su gl4* at 55, 66, and 81 respectively in the chromosome 4 linkage map. The previously available stock was *la su gl3*, *gl3* being at 112.

Chas. Burnham

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Cornnuts, Inc.

Perennialism attributes from Cuzco flour corn

In 1972 (MNL 46:20), we reported that Cuzco flour corn apparently carried perennialism factors that could be substituted for *pe* to produce a perennial phenotype in maize. Subsequent attempts have failed to reproduce this result. A check back through old records shows that the *gt/gt* stock used in that experiment could have also carried *pe/pe*, and we presume that this was the case. This leaves the status of my work on perennialism in *Zea* as found in the two papers, Genetics 50:393-406, and Heredity 58:270-273. In that work, it was found that perennial diploids in *Zea* are based upon a triply recessive genotype: *pe/pe*, isolated from "clone A", *id/id*, and *gt/gt*, the latter two being classical maize mutants. While the status of *pe* has always been weak, the failure to find factors in Cuzco which replace it is in

some degree supportive of the goodness of *pe*'s status, since it remains true that diploid perennials are not obtained without its use or incorporation.

D. L. Shaver

The fill period in Cuzco flour corn

It has been an item of discussion, because of the singular kernel size of this unique land race, as to whether this variety ever forms black layer, if good cultural conditions are maintained. It is said that in its native habitat in Peru's Urubamba Valley the ear-shanks are broken down by hand to terminate filling. Until this past season, moreover, we have never seen BL formation except by the intervention of an outside condition to end the growing season. However, in 1985, we were able to plant on March 15. First silks emerged by June 6. Black layer could generally be found in the population on Nov. 20, on plants that were untouched by stalk rot, and were still green and more or less succulent. Temperatures were above average for the Salinas Valley. Under these conditions, then, the filling period for Cuzco, as indicated by BL formation, was about 150 days, or about 2½ times the normal 60 day period for cornbelt hybrids.

D. L. Shaver

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Construction of a genetic linkage map in maize using restriction fragment polymorphisms

As reported at the meeting last year, we are in the process of constructing a genetic linkage map for maize using restriction fragment polymorphisms (RFPs) as our source of markers. The potential uses for such a tool have been well documented by others and ourselves and include applications to problems in basic genetics and plant improvement programs. For instance, we have already utilized them to map large duplicated areas of the maize genome to different chromosomal locations and to dissect quantitative traits into their individual genetic components. The ability of RFPs to detect variability in a wide range of lines, and hence be informative in many different crosses, may serve as a unifying mechanism to bring together genetic information of many different types such as morphological markers, isozymes, cytological data, quantitative traits, etc.

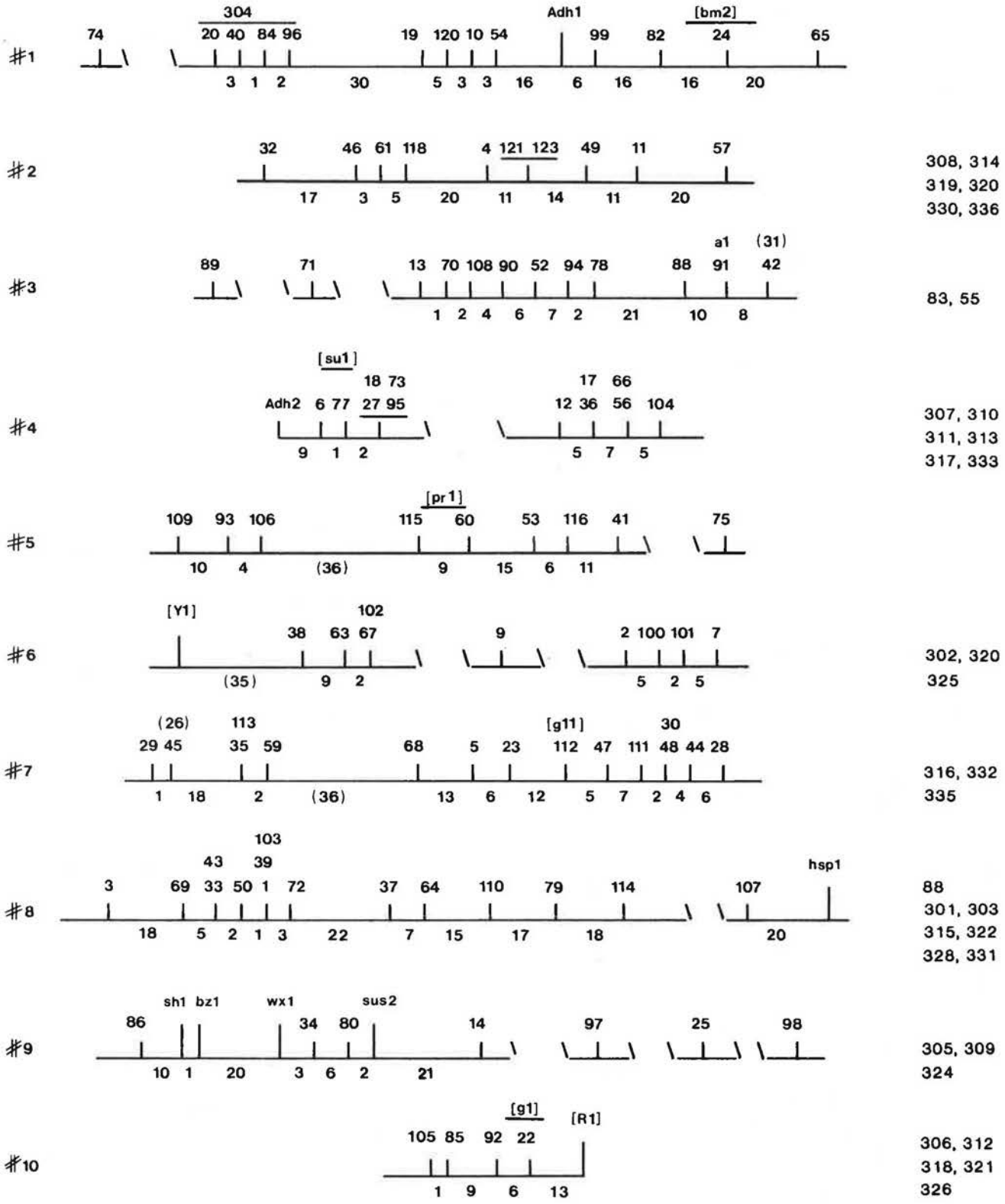
As our source of markers, we have utilized both cDNAs prepared from total leaf mRNA and unique sequence genomic clones less than 2kb in length. These were both initially screened for hybridization signal intensity, complexity of signal, and informativeness against two parents, H427 and 761, supplied by Tim Murphy of Northrup King. Those that satisfied our criteria, i.e. they consistently yielded a strong hybridization signal, were not too complex in their hybridization pattern, and differentiated the two lines used, were then analyzed for their inheritance in 50 F2 plants derived from these two lines. A number of clones for identified genes, such as shrunken-1 and waxy, were also obtained from other researchers whenever possible and similarly tested. Loci detected by these clones were examined for cosegregation by maximum likelihood analysis and placed into linkage groups. To date we have identified 117 loci detected by RFPs and have arranged them into just over 20 distinct linkage groups.

To assign these linkage groups to defined chromosomes and begin to correlate the map we are producing with that derived by other methods, we are using several approaches. Three linkage groups were assigned to chromosomes on the basis that they contained clones of genes with known genomic locations: *Adh1* on chromosome 1, *a1* on 3, *Adh2* on 4, and *sh1*, *bz1*, and *wx1* on 9. The remaining groups were assigned by analyzing individual clones by hybridization to a set of monosomics. Using the *r-x1* deficiency system, we obtained monosomic plants corresponding to eight of the ten maize chromosomes, excluding only 1 and 5, and produced genomic DNA from them. By hybridizing our probes against Southern blots containing DNA from these monosomics and testing for loss of signal contributed by the female parent, we have been able to assign these loci and linkage groups to their chromosomal origins. Seventy-five of our loci were tested by this method to insure the accuracy of these assignments. An additional 38 loci, that were uninformative in our original cross, were also assigned to chromosomes by this method and are awaiting inheritance analysis in other populations. We have also tested for cosegregation of our RFP loci with known morphological markers in a Mangelsdorf backcross population. Linkage of some of our loci to *bm2* on chromosome 1, *su1* on 4, *pr1* on 5, *Y1* on 6, *gl1* on 7, and *g1* and *R1* on 10 have yielded further information on the orientation of our map with respect to the conventional map.

The current version of our map, so derived, is shown in the accompanying figure. Chromosome designations are along the left side, loci designations along the top of the horizontal lines with map distances below the lines. Map distances in parentheses are tentative as they are probably beyond the resolution of our original analysis and need to be verified in a larger population. Loci assigned to chromosomes by monosomic analysis without linkage information are set along the right side of the figure. Symbols for known markers determined morphologically are shown in brackets, while those that were determined through the use of RFPs are shown without.

Future work will center on several areas to improve on these results as this version of the map

MAIZE GENETIC LINKAGE MAP



323, 327, 334 are either on chromosome 1 or 5
Unassigned loci: 201, 202
Loci in right hand column have been assign to chromosome, however linkage data not available

must be viewed as work in progress. First, more markers will be accumulated and mapped to improve resolution of the maize genome and insure that most areas are covered by informative marker sets. Secondly we will utilize a larger F2 population to obtain better estimates of map distances and arrangements of closely linked loci. We are currently working with C. Stuber and M. Edwards at NCSU, to evaluate these marker sets in a different population of approximately 200 F2s which have also been characterized by isozyme analysis. This will yield better resolution as well as improve our correlation with known genomic locations by testing for cosegregation with isozyme loci. Finally we plan to improve our correlation with the conventional map by testing the inheritance of our loci with B-A translocation sets and other morphological markers to establish the presence of centromeres. Our goals then are to produce a detailed set of RFP loci that cover most of the maize genome, to further correlate these loci with the conventional map, and to produce a tester set of RFPs that can be made available to other researchers for various applications.

Tim Helentjaris, Scott Wright and Dave Weber

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Proposal for the nomenclature for the locations of cloned *Mu* elements

After some discussion with various people working on cloned *Mu* elements, I suggest the following system of nomenclature for discussion. This nomenclature is directed at giving specific names to individual cloned *Mu* elements cloned from various sites in the genome. Assigning each new *Mu* clone a specific name should increase the accessibility of the *Mu* literature to non-experts, in contrast to the "Tower of Babel" when each lab assigns its own names.

The *Mu* family of transposable elements is to include all DNA sequences with homology to a terminal inverted repeat of the cloned and sequenced *Mu1* element from the *Adh1-S3034* allele (Bennetzen et al., 1984, PNAS 81:4125; Barker et al., 1984, Nucl. Acids Res. 12:5955). This particular *Mu* element would retain the colloquial name *Mu1*, but all other elements would have descriptors added to indicate the length of the *Mu* element in kb, the laboratory of origin, and either the allele from which it was isolated or a numerical designation (order of recovery) in a particular lab. For example, *Mu1* would have the specific name *Mu1.4MF-Adh1-S3034*. Individual derivative alleles of *S3034* which retained the 1.4kb *Mu* element would be named as they are now by appending a letter designation: *Mu1.4MF-Adh1-S3034a*. Revertants in which the *Mu* element is now gone would be designated by an R placed at the end of the name.

If an element is cloned from a region of DNA that does not contain a known gene, the cloned element would be named for its size, the laboratory of origin, and a number, beginning with one, reflecting the order in which individual *Mu* elements at unknown locations were cloned by that laboratory. For example, *Mu1.7VW-1* is the name of the first 1.7 kb *Mu* element cloned in our lab; it is from an unknown location.

If DNA hybridization studies indicate that a particular mutable allele contains a particular type of *Mu* element, but the *Mu* element has not yet been cloned, the mutable allele should be assigned a name based on current conventions, but the *Mu* element should be referred to simply as an element resident in the allele, for example, the 1.4kb element in *bz1-mu1*.

Based on what is currently known about the *Mu* family of transposable elements, size class designations of *Mu1.4*, *Mu1.7*, and *Mu1.0* would be required. More classes could be conveniently added to take into account additional isolates that differ in their length. A special designation for the presence of just one *Mu* terminal inverted repeat end (with essentially no middle sequence) would be *MuE-*. *Mu*-homologous sequences cloned from non-Mutator standard lines of maize (lines lacking a high mutation rate and a high copy number of *Mu* elements) would be named for the line from which they were isolated, i.e. *Mu1.4VW-B37*.

This nomenclature does not imply that all elements of 1.4kb in length are identical. It requires that an element be cloned before it is named; this is done to avoid ambiguities that might result from examination of the restriction profiles of modified elements resulting in an erroneous assignment of size (see Chandler and Walbot, 1986, PNAS, in press). One potential difficulty is that two labs could independently isolate precisely the same *Mu* insert from related biological material. To aid in detecting such duplications, descriptions of the initial isolation of individual *Mu* elements should include a pedigree of the stock and a restriction map of the element and some flanking sequences.

This nomenclature does not indicate which elements are autonomous and which are defective, because the nature of the presumed autonomous element regulating *Mu* element activity is as yet unknown. Upon discovery the nomenclature might be revised to indicate autonomous and non-autonomous elements.

Virginia Walbot

Inheritance of somatic instability in Mutator lines

We have previously reported that somatic instability in the mutable *bz2-mu1* allele recovered in a Mutator background can be lost (MNL 58:188; MNL

59:98). This loss can occur in both self and outcross progeny. The loss of somatic instability is correlated with de novo modification of *Mu* elements in these lineages detected by a lack of restriction by *Hinf*I (V. Walbot, et al., 1985, Plant Genetics, ARCO-UCLA Symposium, M. Freeling, ed.; V. L. Chandler & V. Walbot, 1986, PNAS, in press). Robertson (Mol. Gen. Genetics 191:86, 1983) previously reported loss of the high forward mutation rate characteristic of Mutator stocks, and molecular analysis of such stocks indicates an increased level of *Mu* element modification as well (J. L. Bennetzen, 1985, Plant Genetics, ARCO-UCLA Symposium, M. Freeling, ed.). Thus, two indicators of transposable element activity—high mutation rate and somatic instability of alleles—are both affected by modification of *Mu* elements. For the purpose of this discussion Mutator lines which are losing or have lost somatic instability at *bz2-mu1* will be termed OFF lines.

Initial experiments in 1983-1984 demonstrated that OFF lines remain off through subsequent outcrossing to non-Mutator lines and upon selfing. Attempts to "reactivate" the mutable phenotype by crossing an OFF line as female by an active Mutator line gave a low fraction of somatically unstable kernels in the progeny; more prominent was inactivation of a heretofore active Mutator line on crossing with an OFF stock. A few reciprocal crosses in which the OFF line was the male parent showed substantial reactivation of the *bz2* mutable phenotype suggesting that there could be a maternal effect.

During summer 1985 larger tests for the stability of the OFF and active states of Mutator lines were conducted as well as tests for maternal effects on inactivation of an active Mutator line by an OFF line and on reactivation of an OFF line by an active one.

The original *bz2-mu1* kernel was derived in 1982 from the cross of *bz2* tester (W23/K55) by an active Mutator line (B139-7). The resulting kernel was planted in the greenhouse, and it transmitted the spotted kernel phenotype normally in a self (3:1 segregation sp:cl K) and when used as the pollen parent to the standard *bz2* downtester or to the *an bz2* deletion stock (1:1 segregation sp:cl K). In 1983 progeny from the self and outcross ears were planted and it was in this generation that loss of the spotted kernel phenotype was noted and correlated with the presence of modified *Mu* elements. The first question addressed here is what fraction of progeny from each type of cross showed aberrant segregation.

As shown in Tables 1-3, loss of the spotted kernel phenotype occurred in the second generation self and outcross progeny of the original mutable plant. The most severe loss occurred in the self lineage (Table 1), in which about three-fourths of the second generation progeny failed to transmit the expected

Table 1. Progeny (C231) of the Selfed Ear of the Original Mutable Plant

Spotted Kernels Planted: genotype one-third *bz2-mu1* and two-thirds *bz2/bz2mu-1*

Type of Cross (Expected Phenotype)	Number of Ears	# Ears with Abnormal Seg. ($p < 0.005$)	sp:cl K Ratios on Abnormal Ears	Average % sp K
C231 (X) (3:1 or 1:0)	4	3	0:258, 51:310, 0:78	5
C231 x *bz2* tester (1:1 or 1:0)	6	4	1:127, 0:178, 19:57 0:19	9
bz2 tester x C231	3	3	49:207, 0:168, 4:231	7

Ears with Abnormal Segregation = 10/13 = 77%

Table 2. Progeny (C230, MF11) of the Outcross of the Original Mutable Plant

Spotted Kernels Planted: genotype *bz2mu-1/bz2*

C230 (X) (3:1)	12	4	0:168, 252:118, 188:85	46
C230 or MF11 x *bz2* (1:1)	7	2	1:275, 3:316	1
bz2 x C230 or MF11 (1:1)	11	7	63:2842[A], 108:179, 22:78 128:263, 73:167, 171:253 0:100	22

Ears with Abnormal Segregation = 13/32 = 40%

[A]sum of 11 ears of tester x C230-1

Table 3. Progeny (C233, C234) of the Cross of the Original Mutable Plant to *an bz2*

Spotted Kernels Planted: genotype *bz2mu-1/an bz2 deletion*

Mutable x Tester (1:1)	10	1	39:61#	39
Tester x Mutable (1:1)	1	1	30:70	30

Ears with Abnormal Segregation = 2/11 = 18%

#significant at $p < 0.01$

ratio of sp:cl kernels. Five of 10 OFF ears were completely colorless, and the average level of spotted kernels was less than 10%. In the outcross progeny of the original mutable plant (Tables 2 & 3) fewer switches to OFF occcurred (15/43 ears) and the average percent of spotted kernels on affected ears was considerably higher than in the self lineage. Considering all of the data together it is clear that an active Mutator state is labile in these lineages and that it can be lost in a substantial percentage of the progeny from an ear with a normal segregation pattern in the previous generation.

The state of *Mu* elements has been reported for eight plants and their progeny in the C230 lineage; there is a highly significant correlation between the loss of the spotted kernel phenotype and the presence of modified *Mu* elements (V. Chandler & V. Walbot, in press, PNAS). Most lines with normal segregation had only *Mu* that could be completely restricted by *Hinf*I; lines with abnormal segregation often had all modified elements, although spotted K from such ears always had at least a few unmodified *Mu* elements. Although molecular analysis is still in progress on the subsequent generations described below, I would like to report some of the genetic data at this time.

First, how stable is OFF? To test this colorless kernels from an ear of *bz2* tester x C230-1 in which there were few spotted kernels (63:2842) were planted (E302) and progeny tested (Table 4). Of the colorless K, one-half should be *bz2/bz2-mu1*, but loss of somatic instability masks the mutable phenotype. At the molecular level 10/10 colorless K tested had modified *Mu* elements. In tests of this and other lineages (C230-3(X) and C231 plants) in three subsequent generations there have been no instances of

Table 4. Test for spontaneous reactivation of somatic instability in the C230-1 lineage.

Type of Cross (Expected Phenotype)	Number of Ears	# Ears with Abnormal Seg. (p <0.005)	sp:cl K Ratios on Abnormal Ears	Average % sp K
Self (3:1 or 0:1)	12	12	all cl K	0
bz2 tester X E302 (1:1 or 0:1)	10	10	all cl K	0
E302 x *bz2* tester (1:1 or 0:1)	46	46	45/46 all cl K 27:107	<1

Spontaneous Reactivation = 1/68 x 2 (1/2 of ears could contain *bz2-mu1*) = 1/34 = 3%

Table 5. Transmission of the spotted kernel phenotype in progeny of *bz2* tester x C230-8.

Type of Cross (Expected Phenotype)	Number of Ears	# Ears with Abnormal Seg. (p <0.005)	sp:cl K Ratios on Abnormal Ears	Average % sp K
Plant colorless kernels: genotype should be *bz2*				
bz2 tester x E301 (0:1)	23	23	all cl K	0
Ears with expected segregation 23/23 = 100%				
Plant spotted kernels: genotype should be *bz2/bz2-mu1*				
(X) CF49 (3:1)	22	1	165:35	66
Ears with abnormal segregation = 1/22 = 5%				
bz2 tester x E306 (1:1)	34	7	35:110, 72:128, 38:190 59:102, 80:184, 22:78, 0:100	24
Ears with abnormal segregation = 7/34 = 21%				
E301 x *bz2* tester	25	1	26:74	26
Ears with abnormal segregation = 1/25 = 5%				

spontaneous reactivation. Consequently, the OFF condition appears to be very stable.

Second, how stable is an active Mutator line? To test this progeny of *bz2* tester x C230-7 and tester x C230-8 have been examined. These two plants were each crossed to >20 tester plants and each gave the expected 1:1 ratios. At the molecular level 5/5 C230-7 sp K tested showed complete *Mu* element restriction with *Hinf*I; for C230-8 18/18 sp K and 16/16 cl K seedling DNA preps showed complete restriction. These two lines were our best-characterized active Mutator lines with no evidence for loss of the somatic mutability character or of *Mu* element modification. The data for part of the C230-8 lineage are reported in Table 5 (from *bz2* x C230-8 = CF49 E301 E306).

Based on the 1:1 segregation of sp:cl kernels on the *bz2* tester x C230-8 plant, colorless kernels were picked as likely to be simply *bz2*; on subsequent outcrossing no evidence of "spontaneous reactivation" was found, however, the test is very small compared to a similar test for reactivation in an OFF lineage (Table 4). Spotted K from the C230-8 active Mutator line transmitted the spotted kernel phenotype in the expected fashion in most self (95%) and outcross progeny (79% as male, 95% as female). Similarly, a second active line, C230-7, showed 66% normal segregation upon selfing (2/3), 78% on crossing to tester (29/37), and 100% normal ears when crossed by tester (10/10). The switch to OFF upon selfing was only 5% for C230-8 selfed compared to an average of 40% for the C230 family as a whole in the previous generation (Table 2). Thus, lines picked because of their normal segregation pattern and absence of modified *Mu* elements are more likely to remain active. However, the characteristics of the previous generation do not guarantee that a line will remain active, because both C230-7 and -8 produced some OFF progeny in the next generation.

Further evidence that active lines are labile comes from tests with subsequent generations. For example, the selfed progeny in Table 5 (CF49) showed 95% expected segregation. When 10 sp K from each of these ears (and four similar ears from another family) were tested by selfing again, 17/25 or 68% showed the expected 1:0 and 3:1 ratios. The ears with a deficiency in spotted K progeny showed close to the expected percent of spotted K.

Combining all data in tests of the stability of the active state of Mutator, it is clear that lines can turn OFF at any time and once OFF tend to remain so. The turning OFF is progressive, noticed first as a deviation in the percentage of ears showing expected sp:cl K ratios; the abnormal ratio ears often have a substantial percentage of spotted K. In the next and subsequent generations both the percentage of normal ears and the percentage of spotted K/ear fall.

I also noticed a maternal effect. Active lines are more likely to switch to OFF when used as male onto tester plants than when crossed by tester pollen. For C230-7 & -8 used as female 34/35 ears (97%) showed normal segregation and the one abnormal ear retained 26% sp K. In reciprocal crosses where these active lines were used as male, 57/71 ears (80%) showed normal segregation; on the ears showing a deficiency of spotted K there were still about one-fourth sp K. One might speculate that transposase or other factors required to maintain the activity of the *Mu* elements is found in a higher concentration in the maternal nucleo- or cytoplasm than in the pollen. Alternatively, we could be seeing the effects of *Mu* dosage in these crosses. The *bz2* tester (W23/K55 hybrid) contains only a few highly modified copies of *Mu* elements and is unlikely to contribute any activities or dosage of *Mu*. Thus, the crosses of an active line as male may dilute the copy number of *Mu* below a threshold required for Mutator activities.

A maternal effect was also noted in the maintenance of the spotted K phenotype in the rare sp K (63:2482) from *bz2* tester x C230-1. All of the cl K from this cross had modified *Mu* elements; of the 5 sp K examined at the molecular level, one contained modified elements but four did not. In the next generation (E303) these sp K differentially transmitted the sp K trait depending on the direction of the cross (Table 6). Although nearly all ears showed abnormal transmission, the percentage of spotted K was much higher when the Mutator plant was used as male. This effect is the opposite of that seen in the C230-7 & 8 lineages. In the C230-1 lineage most progeny contained modified *Mu* elements in the previous generation; thus, it was expected that in the subsequent generation most progeny would be

Table 6. Transmission of the spotted K phenotype by the C230-1 lineage.

Planted rare spotted K from *bz2* tester x C230-1: genotype should be *bz2/bz2-mu1*

Type of Cross (Expected Phenotype)	Number of Ears	# Ears with Abnormal Seg. (p <0.005)	sp:cl K Ratios on Abnormal Ears	Average % sp K
bz2 tester x E303 (1:1)	11	10	28:81, 28:55, 18:90 5:300, 38:168, 36:138 4 ears were 0:>300	12
E303 x *bz2* tester (1:1)	15	15	4:>300, 2:202, 1:>250 1:>300 1:>300m 10 ears 0:>300	<1

Fraction of ears with abnormal segregation 25/26 = 96%

OFF. The two hypotheses given above about the nature of the maternal effect could apply here also. However, a third alternative is also suggested, namely the maternal effect in this case might be explained by dilution of the "modification system" by transmission through pollen.

Further evidence that the C230-1 lineage contains an active modification system, responsible for switching active lines to OFF at high frequency, comes from examination of the spotted K on the C230-1 selfed ear which showed 124:59 sp:cl K ratio ($p < 0.04$); at the molecular level 2/5 spotted K and 5/5 colorless kernels contained modified *Mu* elements. In subsequent generations almost no spotted K were recovered from crosses involving spotted K from C230-1 selfed (CC2 & 3, D90 & 91) or from the progeny of these ears (E305) (Table 7). Thus, in the C230-1 selfed lineage the rate of turning OFF is exceptionally high, even though a near normal sp:cl K ratio was found on the parent ear. The OFF condition is again very stable.

Table 7. Transmission of the spotted K phenotype from progeny of C230-1(X).

Spotted kernels planted; genotype should be 1/3 *bz2-mu1*, 2/3 *bz2/bz2-mu1*

Type of Cross (Expected Phenotype)	Number of Ears	# Ears with Abnormal Seg. (p <0.005)	sp:cl K Ratios on Abnormal Ears	Average % sp K
D90 & D91 (X) (1:0 or 3:1)	10	10	all cl	0
bz2 tester X CC or D	13	13	3:303, rest 0:>300	<1
CC or D x *bz2* tester	12	12	all cl	0

Fraction of abnormal ears = 35/35 = 100%

Subsequent generation taking 5 cl K from each (X) ear above

Self	47	47	all cl	0
bz2 tester x	39	39	all cl	0
stock x *bz2* tester	22	22	all cl	0

Fraction of abnormal ears = 108/108 = 100%

The last question is, what is the impact of crossing an active and an OFF line together? The first experiment that addresses this point involved crossing the C230-8 and C230-1 lineages. cl K of *bz2* tester x C230-8 (E301), which should be simply *bz2* in an active Mutator stock, were crossed as male and female by cl K taken from *bz2* tester x C230-1 (E302); 10/10 seedlings from this C230-1 stock contain some modified elements. In exact reciprocal crosses the ability of individual E301 plants to reactivate the suppressed sp K phenotype of E302 individuals was examined. As shown in Table 4 the "spontaneous reactivation" of this family is very low. In crosses with E301, however, 20/36 plants showed at least a few spotted K, close to the expected fraction (18/36) of these plants that should be *bz2/bz2 -mu1*. Averaging all ears (E302 as male or female) in which spots were found, the average percentage of spotted K was 27%, of 50% expected to be spotted. Twelve pairs of exact reciprocal crosses were completed between E301 and E302 plants in which spotted K were found in the progeny. Of these two instances were found in which only the E301 as female had any spotted K (22:278, 7:282 vs. 0:>300 for E302 x E301). Additional support for a positive maternal effect by active E301 plants on reactivation of the cryptic *bz2-mu1* somatic mutability comes from a comparison of the average percent of spotted K: with E301 as female, the average was 31%, with E302 as female the average was 25%.

The final evidence for a maternal effect comes from an "inactivation" experiment in which spotted K progeny of *bz2* tester x C230-8 (E306) were crossed by cl K from the C230-3 x *bz2* tester ear; this latter ear gave a 0:168 phenotypic ratio, 12/12 seedlings tested contained modified *Mu* elements, and in subsequent crosses to *bz2* tester 22/22 ears were completely OFF. Thus, the C230-3 lineage is by all criteria an OFF line. As in the reactivation experiment, each individual was crossed as male to tester, and then exact reciprocal crosses were completed between active and OFF individuals (Table 8).

Table 8. Effect of crossing active and OFF Mutator lines.

	E306 x E307 (active x OFF)	E307 x E306 (OFF x active)
# ears with normal segregation	13	3
# ears with abnormal segregation	0	10
% Normal segregation	100	23
Phenotypic segregation on abnormal ears		16:24, 5:36, 38:62, 12:25, 8:47, 12:29, 17:23, 22:41 4:66, 19:34

In the "inactivation" experiment there is again a strong maternal effect. All ears derived from an active line crossed by an OFF line showed the expected 1:1 segregation for sp:cl kernels. In the reciprocal crosses, however, only 3/13 ears showed normal segregation. Those showing abnormal segregation had an average of 28% spotted kernels suggesting that the switch to OFF is just beginning. It will be interesting to determine the transmission of the spotted K phenotype in subsequent generations and the correlation to modified *Mu* elements in this stock.

In summary, these data suggest that an active Mutator line can lose activity measured as somatic instability at a reporter mutable allele at a reasonably high frequency. The somatic instability of the previous generation is, in fact, a poor indicator of the behavior of the stock in subsequent generations: for example, essentially all spotted K of the C230-1 selfed ear failed to transmit sp K. Once OFF a Mutator line seems to remain off, and there are only rare instances of "spontaneous reactivation." Several kinds of maternal effects were noted in these

materials. In lines showing just a minor loss of Mutator function (C230-7 & 8) such losses occurred preferentially when these plants were crossed as male onto tester. This maternal effect suggests that a positive factor(s) required to maintain Mutator activity is being diluted. In contrast, lines such as C230-1 in which the switch to OFF is occurring at high frequency are more likely to retain activity when crossed as male onto tester; it is tempting to speculate that they are escaping an active modification system. These interpretations are supported by the reactivation and inactivation experiments. When active and OFF lines were reciprocally crossed, an active line (C230-8) shows completely normal sp:cl K segregation when used as the female parent but not as male (Table 8); I hypothesize that positive factors in the active female line promote Mutator activity, while negative factors in an OFF line suppress it. Similarly, an OFF line is more likely to be reactivated when crossed as male onto an active line, again suggesting that the state of the female parent is critical in determining the extent to which the active and OFF states are maintained.

Until the nature of the activities encoded by autonomous and non-autonomous *Mu* elements have been delineated, it is impossible to know precisely what the relationship between element modification and the OFF state really is and whether this relationship depends on the number of modified and unmodified copies in a stock. However, it does appear that there are two aspects to the regulation of Mutator activity: active lines contain factors which positively regulate activity and OFF lines contain factors which negatively regulate activity. These activities condition the state of the embryo sac producing maternal effects on Mutator activity. A simple explanation for this would be that active lines contain transposase and OFF lines contain the DNA modification system that renders *Mu* elements inactive and that both the transposase and modification system transmit poorly through the pollen.

Virginia Walbot

Phenotypic changes in inactive Mutator stocks

All of our Mutator stocks are drawn from a narrow genetic base and have been crossed to W23, K55 or W23/K55 materials for maintenance. Thus, we would not expect to find much variation in macro plant phenotypes, except as the result of mutation in individual plants. However, various Mutator stocks maintained in our lab that have switched from active to inactive show a delay in flowering time of up to 2 weeks in the field and greenhouse as well as growth chamber-grown material (J. Woodman, pers. communication). In addition to delayed flowering, OFF lines are also shorter and have fewer leaves than active sib lines. These phenotypic changes arise coincident with the loss of Mutator activity; they occur simultaneously in entire families whether the product of selfing or outcrossing, suggesting that the changes are related to the Mutator state, not to the segregation of a particular gene.

Virginia Walbot

Reactivation of somatic instability in an inactive Mutator stock by treatment of seed with gamma rays

Chromosome breakage, DNA damaging agents, and stress have been previously implicated as agents that may stimulate the activity of heretofore cryptic transposable elements. To test whether DNA damage—and the DNA repair following such treatments—could influence the activity of the Mutator system the following experiment was carried out. Colorless seed carrying a cryptic mutable *bz2* allele recovered from a Mutator parent was selected; the DNA of the *bz2-mu1* parent plant was shown to contain mainly modified copies of *Mu* elements as evidenced by poor *HinfI* digestion of the *Mu* element DNA. Seed were treated with gamma radiation from a ^{137}Cs source, planted and crossed by *bz2* tester. The radiation did not reduce seed germination. Control experiments of this lineage involving >30,000K have yielded no somatically unstable kernels when the OFF line was used as female parent. Although smut considerably reduced the yield in the gamma plot, 3 of 1,104K were spotted and one 5K purple ear sector was found. These results suggest that DNA damage can reactivate an OFF line. Molecular tests are underway to examine the state of the *Mu* elements in general and the *Mu* element presumed to be at *bz2-mu1* in the reactivated lines. The experiment will be repeated with a larger population this summer and with higher, calibrated dosages of gamma irradiation. Facilities for these experiments were provided by Dr. M. Christianson.

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Seed storage death

Twenty-year-old seeds stored at room temperature (70 ± 10 F) were place on moistened germination battens for thirty days in order to learn whether any were still alive. Six seeds in a population of 149 did germinate, but only their shoot grew through the pericarp; no root growth occurred. These six seeds occurred on 4 ears, each of which were somewhat related to W22 but genetically different from each other. It is of interest to note that the two growing points on a dormant seed do not apparently survive equally well after long storage times. The root loses its growth ability sooner than the shoot. I wonder why?

The shoot growth that was observed on the six plants noted above all showed coleoptile expansion and appeared normal, except in none of these cases did the coleoptile open. It seems instructive to view the dormant seed as a series of functioning systems each of which must be turned on for germination. These six seeds demonstrate that some systems can turn on after twenty years while others cannot. It would be of interest to determine if these plants could have been salvaged by removal of the embryo to artificial media. This test will be run during the coming year. In contrast to this experience, other seeds that have been stored under reduced temperature and humidity for 25 years showed greater than 95% germination, with both the root and shoot behaving normally.

Irwin Greenblatt

Hydrogen peroxide treatment of seed

In order to gain a higher percentage of seed germination than experienced in the population noted above, remnant seed from the same ears was first soaked in a 1.5% solution of commercial hydrogen peroxide (a 3% solution diluted 50 percent with tap water) for 48 h before being placed on germination battens moistened with tap water for 35 days. In a population of 129 seeds only one kernel germinated its shoot only. Thus, the increase of available oxygen made no improvement. In contrast to this experience, other seeds that were dormant due to day-length treatment were induced to germinate at twice the untreated rate with a H_2O_2 pretreatment. With fresh seeds which are not dormant, H_2O_2 for 48 h increases the speed of germination; protrusion of both the root and coleoptile through the pericarp occurs in less than 48 h.

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Anther culture of different origins of varieties

Since the first success in obtaining pollen-plants of maize in 1975 (Ku et al.), much effort has been made by many workers with maize anther culture. Considerable progress has been achieved in this field of research. However, the number of responding genotypes is limited, and almost all of them are Chinese or United States germplasm. In our experiment, we used Japanese and Chinese germplasm reserved in The National Institute of Agro-Biological Resources in Tsukuba, Japan, and some American dent hybrids and an introduced synthetic.

In 1983-1984, over five plantings, plant materials were grown in a greenhouse. Tassels with anthers containing uninucleate or early binucleate microspores were detached and incubated at 8 C for 14 days. After the cold treatment, tassels were sterilized with a saturated solution of calcium hypochlorite and anthers were plated in Yu-Pei medium containing casamino acid 500mg/l, sucrose 120g/l, activated charcoal 5g/l and TIBA 0.1mg/l. At the second planting in 1984, we used three different media, described in Table 2. Plated anthers were incubated at 25 C in the dark. After 30-60 days in culture, callus or embryoid formation was observed.

Table 1. Variation in callus and embryoid formation by anther culture in different origin of varieties in maize (1983 - 1984, 5 plantings)

Variety	No. of plants	No. of anthers plated(A)	No. of calli or embryoids formed (B)	Induction frequency (B/A)	Variation of induction frequency in individual plants
Japanese local variety				%	%
Kiwase	5	142	0	0	—
Sakashita	8	642	2	0.3	2.6 — 0
Sapporo hachigyou	8	561	3	0.5	4.5 — 0
Hakusyoku hachigyou	8	754	2	0.3	1.8 — 0
Hakusyoku hachigyou (Toya)	10	1247	8	0.6	3.0 — 0
Hirano	6	700	5	0.7	2.7 — 0
Kowase	18	1880	16	0.9	8.3 — 0
Kuma	6	363	2	0.6	1.8 — 0
Kamigane 1	6	500	0	0	—
Aso	10	1960	23	1.2	9.3 — 0
Shirotoukibi	10	1813	0	0	—
Kijiyama 33	10	1450	11	0.8	12.0 — 0
Hirano S_1	10	1448	3	0.2	0.8 — 0
JF2C2	11	1738	3	0.2	1.7 — 0
Total	126	15198	78	0.5	
Chinese variety					
Hakutousou	9	1193	5	0.4	1.5 — 0
Sankousyou zairaisyu	8	781	2	0.3	1.7 — 0
Moukosyu	10	1526	7	0.5	1.4 — 0
Souyaku 3	10	1798	9	0.5	3.3 — 0
Pekin sougyokumai	10	1153	0	0	—
Souken ougyokumai	9	911	7	0.8	3.3 — 0
Total	56	7362	30	0.4	
American dent hybrid and synthetic variety					
P 3732	7	670	0	0	—
P 3424	5	604	0	0	—
P 3358	5	563	0	0	—
H95rhm X N28Htrhm	6	537	0	0	—
Fla.FSHmR	7	601	0	0	—
Total	30	2975	0	0	

Table 2. Effect of culture medium on callus and embryoid formation*(1984 2nd planting)

Medium		No. of anthers plated (A)	No. of calli or embryoids formed (B)	Induction frequency (B/A)
				%
A: YuPei + TIBA	0.1mg/l	1811	14	0.8
B: YuPei + 2,4-D	2 mg/l	2595	22	0.8
kinetin	1 mg/l			
NAA	1 mg/l			
C: N_6 + TIBA	0.1mg/l	2201	12	0.5

Note: * Average of 13 varieties.

The response was different between genotype (Table 1): 5 American dent genotypes did not respond at all, but 11 of 14 Japanese genotypes and 5 of 6 Chinese genotypes responded. The frequency of responding anthers was low, and induction frequencies of most genotypes were under 1.0%. The highest response was 1.2% attained in the Japanese local variety 'Aso'. However, wide variation in induction frequency was observed among individual plants as well as among varieties, especially in 'Aso' and 'Kijiyama 33'. The values were 9.3 - 0% and 12.0-0%, respectively. The variation among individual plants was estimated to be caused by both genetic and environmental factors, and it may be possible to improve the response by selecting highly responsive plants and making hybrids among the selected plants. Though the differences of media were indistinct, Yu-Pei medium was somewhat more effective than N_6 medium (Table 2).

Calli or embryoids formed were transferred to regeneration medium (Yu-Pei medium containing

sucrose 30g/l, kinetin 1mg/l and other elements same as induction medium) and incubated at 25 C, 14h day length. In this condition, some calli and embryoids regenerated root or shoot: six formed roots and four formed shoots, but no plantlet was obtained. On the other hand, some of the embryoids on B medium (Table 2) were transferred to 16hr light condition without change of medium, and three embryoids grew into plantlets.

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Mitochondrial genomes of fertile revertants from *cms-S*

S1 and S2 DNAs are mitochondrial DNA (mtDNA) molecules found in male-sterile S cytoplasm (*cms-S*) of maize but not in fertile revertants with the nuclear background of inbred line M825 (Levings et al., Science 209:1021, 1983). Other revertants which were recovered from Wf9 lines of *cms-S* retained S1 and S2 DNAs (L.J. Escote et al., MNL 59:100, 1984; Ishige et al., Japan. J. Breed. 35:285, 1985). Three revertants designated J′, G′, R′ were obtained as spontaneous mutants from J, G, and R sources of S cytoplasm in the Wf9 background. The results from mtDNA restriction endonuclease analyses of these revertants showed that the different restriction sites were present only in chromosomal mtDNA and not in S1 and S2 DNA.

Direct DNA-DNA hybridization experiments for these revertants were done to check the insertion of S1 and S2 DNA into the chromosomal mtDNA. Uncut mtDNA preparations extracted from revertants J′, G′, R′ and sterile S cytoplasms were analyzed by agarose gel electrophoresis and Southern blots. S1 and S2 DNAs were extracted from another agarose gel by electroelution and purified by phenol-chloroform extraction. S2 DNA was nick-translated and used for the DNA-DNA hybridization probe. Autoradiographs of undigested mtDNAs showed that hybridization patterns of revertants and control *cms-S* were similar (Figure 1). The S2 DNA probe hybridized to S1 and S2 DNAs, and to S3, S4, and S5 DNAs as designated by Kemble and Mans (J. Mol. Appl. Genetics 2:161, 1983). Hybridization patterns of mtDNAs digested by XhoI were also similar to each other regardless of the different restriction fragment patterns of revertants (Figure 2).

These results indicate that the rearrangements of chromosomal mtDNA of revertants were not likely caused by the insertion of sequences homologous to S1 or S2 DNA.

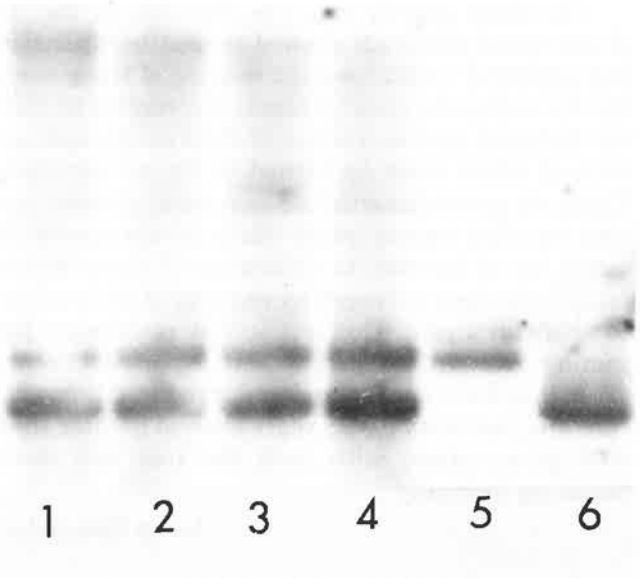

Figure 1. An autoradiograph of Southern blot of the undigested mtDNA after hybridization with a probe of S2 DNA. Undigested mtDNAs isolated from S(1), J′(2), G′(3), R′(4), S1(5) and S2(6) were resolved by electrophoresis on 0.8% agarose gel.

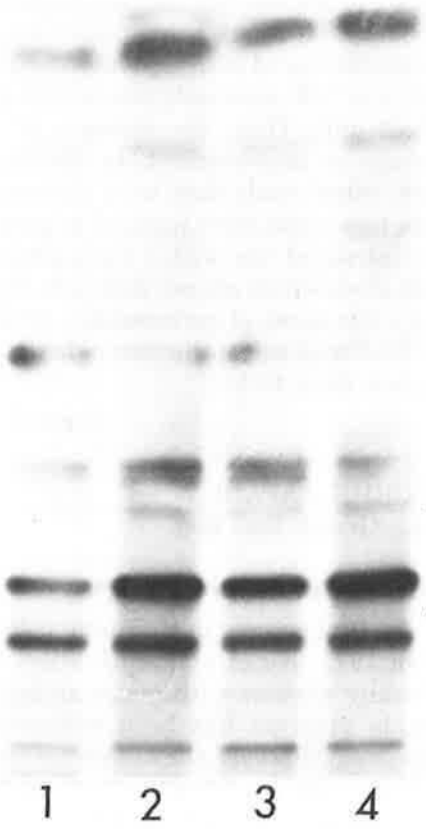

Figure 2. An autoradiograph of Southern blot of the XhoI digest of mtDNA after hybridization with a probe of S2 DNA. MtDNAs isolated from S(1), J′(2), G′(3) and R′(4) were digested with XhoI and resolved by electrophoresis on 0.7% agarose gel.

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Changes in nuclear genomic background bring about reorganization of mitochondrial DNA

S-type male-sterile cytoplasm in maize is characterized by the presence in the mitochondrial genome of two autonomously replicating linear plasmid-like DNA species called S1 and S2, 6.4 kb and 5.4 kb in length, respectively. In the S-male sterile M825 inbred line background, cytoplasmic reversion to fertility is accompanied by the loss of these plasmid-like DNAs and by rearrangements in the main mitochondrial genome that involve sequences homologous to S1 and S2 (Levings et al., Science 209:1021-1023, 1980; Laughnan et al., Stadler Genet. Symp. 13:93-114, 1981; Kemble and Mans, J. Molec. Appl. Genet. 2:161-171, 1983; Schardl et al., Cell 43:361-368, 1985). In the WF9 inbred line background cytoplasmic reversion to fertility does not involve the loss of the free S1 and S2 plasmid-like DNAs (Escote et al., MGCNL 59:100, 1985; Plasmid, in press; Ishige et al., MGCNL 59:98-99, 1985; Japan J. Breed. 35:285-291, 1985), and the pattern of rearrangement of sequences homologous to S1 and S2 in the main mitochondrial genome is distinct from that of M825 revertants. Revertants that arise following conversion of the RD- or ML- WF9 inbred-line background to the M825 nuclear background by recurrent backcrossing lose the free S1 and S2 episomes, and exhibit a rearrangement of the main mitochondrial genome that is characteristic of M825 revertants rather than of WF9 revertants (Escote et al., MGCNL 59: 100, 1985, and unpublished results). This indicates there is a major effect of the nuclear background on the organization of the mitochondrial genome of the fertile revertants. It was not clear, however, whether the nucleus exerts this influence on the mitochondrial genome organization upon reversion or prior to the reversion event. We therefore studied the organization of the mtDNA of the progeny in each generation of the conversion of the male-sterile RD-WF9 to the M825 nuclear background using as mtDNA probes the cloned S1 and S2 sequences, and the cloned mitochondrial genes for cytochrome oxidase subunits I (COI) and II (COII) and apocytochrome B (COB).

Figure 1 shows HindIII digests of mitochondrial DNA from the progeny of five generations of backcrosses of RD-WF9 by M825, hybridized to the probes COI (Panel A), COB (Panel B), and COII (Panel C). Lane O corresponds to the female parent RD-WF9, lanes 1 to 5 correspond to the progeny of the first to the fifth cross of RD-WF9 by M825, and lane N to the normal fertile maize mitochondrial genome. As shown in Figure 1, reorganization involving the COI gene is apparent as early as the F1 generation, with the loss of three COI fragments originally present in the RD-WF9 parent (refer to figure for approximate sizes of the fragments). In the progeny of the first backcross to M825, two more COI bands disappear. Accompanying the loss of these COI bands is the gradual development of a COI band of about 4.8 kb that is not detectable in the RD-WF9 female parent. The pattern of COI gene organization observed in the progeny of the first backcross by M825 persists through the progeny of the fourth backcross, except for the increasing level of the 4.8 kb band in later generations. A similar pattern of change is observed with S1 and S2 probes, although in this case the change is limited to early loss of bands without the accompanying appearance of a new band (data not

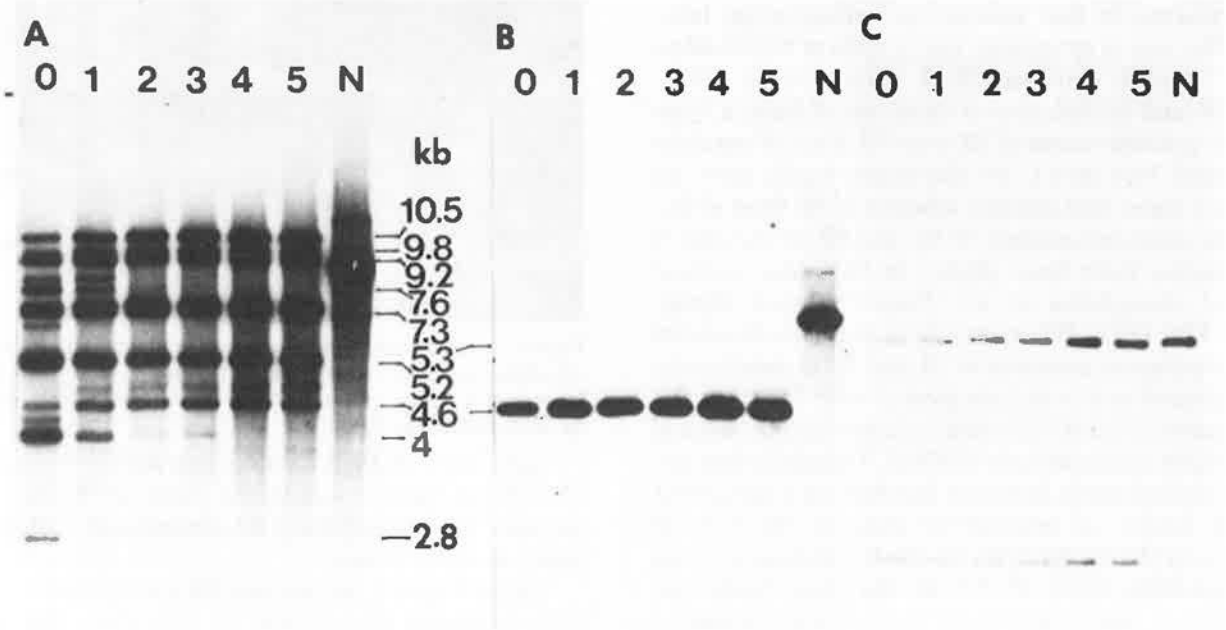

Figure 1. HindIII restriction digests of mitochondrial DNA from the progeny of each generation of crosses of RD-WF9 with M825, hybridized to the following probes: A, COI; B, COB; C, COII. See text for explanations.

shown). With the COB and COII probes (Panels B and C, respectively) there is no apparent alteration in organization accompanying the conversion process.

These results indicate that the influence of the M825 nuclear genome on mtDNA organization can occur in the absence of reversion. The changes appear to involve limited areas of the main mitochondrial genome, i.e. S1 and S2 regions of integration, and COI gene sequences but not COII and COB genes. The changes involve the loss of bands originally present in the RD-WF9 female parent, and the appearance of a new band that was not apparent in the original RD-WF9 parent. The pattern of changes observed is compatible with the model of a multipartite mitochondrial genome (Lonsdale et al., Nucl. Acids Res. 12:9249-9261, 1984) composed of different replicons with varying contents and sequences, e.g. those of S1, S2 and COI. Given that the WF9 nuclear genome is characterized by a specific distribution and content of such variant replicons, the M825 nuclear genotype appears to establish a different equilibrium that favors the replication of some of these elements, suppresses that of others and leaves still others unchanged.

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A possible basis for the lag phenomenon observed in M825 conversion of *cms-S* strains with equimolar S1:S2 ratios

The mitochondrial DNA (mtDNA) of the S-type cytoplasmically male-sterile maize (*cms-S*) is characterized by the presence of two linear DNA molecules, S1 and S2, 6.4 and 5.4 kb in length, respectively. In the *cms-S* versions of most inbred lines examined, these linear molecules are present in nearly equimolar amounts. In four inbred-line backgrounds, however, the *cms-S* cytoplasm has a ratio of S1:S2 other than 1:1 (J.E. Carlson, Ph.D. thesis, 1983). R825, M825K and M825L *cms-S* versions all have a four-fold or greater excess of S1 over S2. *Cms-S* versions of inbred line 38-11, on the other hand, have an average three-fold greater amount of S2 than of S1.

The relative amounts of S1 and S2 in the *cms-S* cytoplasms have been shown to be under nuclear control (Laughnan et al., Stadler Genet. Symp. 13:93-114, 1981). When an inbred-line *cms-S* version with equimolar amounts of S1 and S2 is recurrently backcrossed as the female parent with M825 as the male parent, the S1:S2 ratio is shifted to the reduced S2 pattern characteristic of M825. This shift does not occur immediately, however, but follows a lag period of one backcross generation; that is, there is no change in the S1:S2 ratio in the F1, a change to an intermediate level of S2 in the first backcross generation, and by the third backcross generation a change in level of S2 equivalent to that of *cms-S* M825 itself.

The lag phenomenon leads to questions about the number of nuclear genes controlling the relative replication rates of S1 and S2 and about their dominance relationships. Results from Carlson's experiments on genetic control were inconclusive, and new approaches have been taken in attempts to analyze the phenomenon. One of the approaches involves the use of the indeterminate gametophyte gene, *ig* (J.L. Kermicle, Science 166:1422-24, 1969; Amer. J. Bot. 58:1-7, 1971). The *ig* mutant increases the rate of occurrence of both maternal and paternal monoploids. The strain of *cms-S ig ig* used here has the nuclear background of W23 and A158 inbred lines, both of which maintain equimolar amounts of S1 and S2. This stock was crossed as female with M825 and 38-11, and the resulting diploids and monoploids were analyzed for S1:S2 ratios.

Mitochondrial DNA was isolated from young unpollinated ears using a rapid assay procedure developed by Kemble and Bedbrook (Maydica 24:175-180, 1979), and modified by Carlson (Ph.D. thesis, 1983), for use with etiolated shoots.

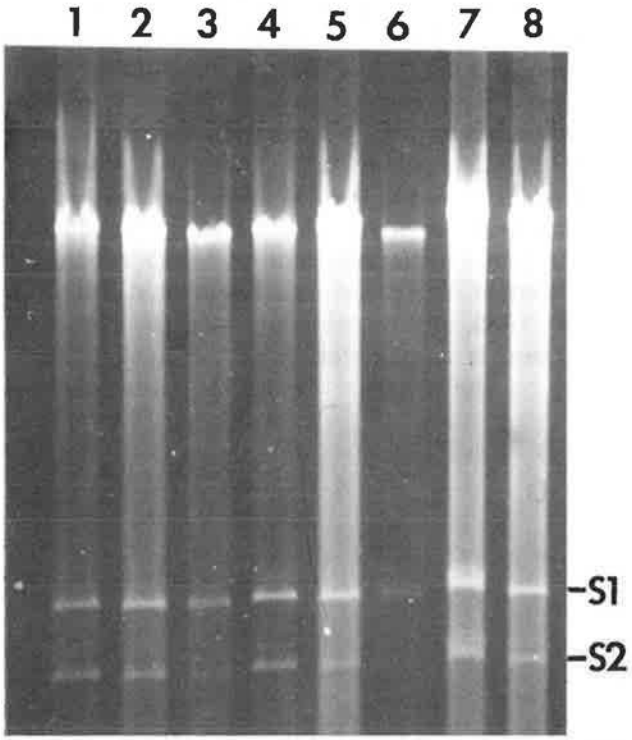

Figure 1. Agarose slab gel electrophoretogram of unrestricted mitochondrial DNA prepared by rapid assay procedure from unpollinated cob samples of the *cms-S ig* stock crossed by W23, M825 and 38-11.

Lane 1 is a mtDNA sample from an RD-WF9/38-11 F1 with a known equimolar ratio of S1:S2; it is included here as a control for comparison with S1:S2 ratios in other lanes.

Lanes 2 and 4 contain mtDNA samples from two W23 maternal monoploids; as expected, the S1:S2 ratio in this nuclear background is approximately 1:1.

Lanes 3 and 6 contain mtDNA samples from S-M825 paternal monoploids. The ratio of S1 to S2 in these monoploids is that characteristic of M825 inbred line, i.e. S1 greater than S2. This is a departure from the previously observed lag in the shift of S1:S2 ratios when *cms-S* lines with equimolar ratios of S1 to S2 are crossed as female parents with M825. In the case of the M825 paternal monoploids, the nuclear genes of the female parent are replaced by the M825 nuclear genes after only one cross, i.e. in the androgenetic embryo. Recurrent backcrossing of a *cms-S* equimolar strain by M825 as male parent requires numbers of backcrosses to even approach such a substitution. We discuss the further significance of this below.

Lanes 5 and 8 contain mtDNA samples from S-M825 spontaneously doubled monoploids. The ratios of S1:S2 observed are those characteristic of the M825 inbred line, and again no lag is observed.

Lane 7 contains mtDNA from an S-38-11 paternal monoploid. *Cms-S* versions of inbred line 38-11 exhibit higher levels of S2 than S1. Contrary to what was observed with the M825 monoploids and the androgenetic diploid M825 derivatives, however, the single 38-11 monoploid does not exhibit a ratio that is characteristic of the 38-11 inbred line; as shown in lane 7, the level of S1 is about the same as S2. Obviously we need to analyze more cases of 38-11 androgenetic derivatives from this cross and follow them through a longer time course.

We had earlier considered that the lag phenomenon might be attributed to a physiological delay associated with the time required for organellar components to adjust to a new equilibrium specified by a change in nuclear genotype, or alternatively, to "recessiveness" of the M825 gene allele(s) associated with the reduced level of S2 replication. The observations on M825 paternal monoploids and on the M825 androgenetic diploids provide no evidence that there is a delay in reaching a newly-established equilibrium; in fact, they strongly suggest that the lag phenomenon observed in the conversion experiments is due to dominance of the nuclear gene(s) controlling normal S2 replication.

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Tagging the *cms-S* restorer genes

The following controlling element (CE) stocks have been propagated and crossed onto *cms-S* male-sterile testers to determine their fertility restorer (*Rf*) status: *bz2-m* (*Ds-Ac*); *Adh1-FM335* (*Ds-Ac*); *a1-m4* (*Ds-Ac*); *a1-m1* (*I-En*); *a1-m1* and *wx-m8* (*Spm-S*); *bz1-m* (*Ds-Ac*); (*P-v R-sc*) (*Ds2-Mp*); and Mutator (*Mu*). In progeny of these crosses we have identified *rf rf* (non-restoring) plants in five of the eight CE pedigrees tested. The CE strains were also crossed as male parents onto *cms-S* carrying one of the spontaneously-arising restorers, *RfI* through *RfX*. These restorers have been located at a minimum of seven different sites in chromosomes 1, 2, 3 and 8 in maize. For the restorer tagging experiments we have emphasized those CE-*Rf* combinations in which one or more of the CE (autonomous or nonautonomous) tagging elements is linked with the *Rf* target. We are now in a position to search on a large scale among plants carrying a defined *Rf* element and a functional CE system for tassel phenotypes that indicate the insertion of CE elements into *Rf* genes.

We have shown that *RfI, RfIII, RfIV* and *RfVI* are themselves transposable and we hope to characterize them at the molecular level through an alternate route by identifying their insertions into maize genes such as *Adh1, Sh1, Bz1,* and *A1*, for which gene clones are already available. We plan for extensive plantings, mainly in isolation plots, to identify the rare events sought.

Crosses have also been made to develop stocks that will afford a search for CE and/or *Rf* into various maize genes for disease resistance, and into maize genes whose mutant forms are associated with profound developmental changes. Protocols for the use of CE and *Rf* strains in tagging such genes are being developed.

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Female fertility conferred upon the normally female sterile combination of Papyrescent (*Pn*) with branched-silkless (*bd*)

The homozygous recessive *bd bd* is tightly linked to the homozygous dominant *Pn Pn* near the end of the long arm of chromosome 7. In a typical background of present-day maize, the *bd* phenotype is highly branched like the ramosa genes but also female sterile by delayed style growth. Because of this female sterility, previously it has been impossible to propagate the double homozygous condition. The interaction of modifying genes on the *bd bd* phenotype is under study. The addition of the tassel seed-2 gene (*ts2*) on chromosome 1 not only leaves the ear female sterile but worsens the situation by also making the tassel male sterile and monstrously branched, but not hopelessly monstrous. The further addition of the silkless gene (*sk*) on chromosome 2 restores a sort of balance with a new previously unknown phenotype. Thus, the complex of the following four genes, *Pn Pn, bd bd, ts2 ts2, sk sk,* has a papyrescent ear that is highly branched and female fertile.

Other genetic systems to confer female fertility upon *Pn Pn, bd bd* are being explored. It is hoped

that an F1 hybrid between two papyrescent lines each with a different recessive system for female fertility will give a hybrid uniform for sterile "cauliflower ears". This would represent a new vegetable comparable to the cauliflower variety of *Brassica oleracea*.

Walton C. Galinat

Progress in breeding silkless baby corn for whole ear consumption

The recessive tassel seed genes (*ts1, ts2* and *ts4*), with selection for the desired phenotype, can act as silk-restorer genes for the silkless gene. The most successful combination so far has been the double recessive of *ts2 sk*. Some selected stocks of *ts2 sk* have essentially normal tassels and ears. The development of a few tassel silks and a few secondary ear florets may be discovered on close examination.

Hybrids between two silkless stocks, each with silks restored by a different recessive tassel seed gene, give a crop of tiny barren cobs that are completely silkless. Together with mechanization this new genetic system allows mass production of whole cob baby corn in a way that domestic agriculture may capture this expanding market now monopolized by importations. Because the silkless condition allows some delay in harvesting, presumably a machine could be designed to do the task, at least in part. The yield of baby ears is increased by the use of a multiple-eared background derived from certain popcorns. Baby ear production is further enhanced by the silkless condition because all of the energy becomes devoted to producing more cobs instead of going first into silk growth and then into kernel development. Yields of 25 or more silkless cobs per plant may be realized.

The present use of imported baby ears tends to be in the manner of a garnish in stir-fry preparations, in salads or at "salad bars" and as pickles. Their food value is chiefly as fiber. They add a crunchy texture, some corn flavor and eye appeal. The cob flavor is an independently inherited trait.

Walton C. Galinat

Dormancy and photosensitivity as premaize traits

One of the early effects of domesticating teosinte into maize must have been a genetic removal of dormancy from the mature seed so that the seed would germinate uniformly soon after planting. If the removal is carried too far in certain mutations we get a lethal condition known as uninterrupted embryo development in which the ear is a mass of seedlings at harvest time. In wild populations of teosinte, once its fruit cases reach the ground, dormancy substances in the seed inhibit germination until environmental conditions have removed them, which is usually adjusted to last until the most favorable time for growth. Variable soil conditions in nature result in corresponding differences in germination.

Synchronous flowering is usually important to cross-pollinated plants such as teosinte and maize in order to maintain heterozygosity and optimum size of an interbreeding gene pool. If variation in the time of germination resulted in isolation by flowering time, it could have harmful effects associated with inbreeding depression. Therefore, the dormancy trait became associated with another trait that insured uniform flowering. This trait, involving floral induction by short day-lengths continuing over a 2 to 3 week period, is now characteristic of all wild races of teosinte and to a lesser degree of certain tropical races of maize. Once induced, teosinte flowers regardless of plant size while short-day maize has a minimum length for the vegetative phase. Tropical maize tends to have large late-flowering plants that only tend to respond to short-day lengths.

Walton C. Galinat

Maiz de Ocho, the frontier maize in the northward spread and adaptation of maize

Ever since ca. A.D. 700, Maiz de Ocho has been the pioneer race at the northernmost fringes of maize cultivation in North America. At this date, some 3000 years after its spread southward into South America, this new race of maize broke through the previous barriers of short growing seasons and long day-lengths as it expanded northward. Maiz de Ocho has a special attribute that made this dramatic event possible. In response to selection directed toward early flowering it can progressively reduce the duration of its vegetative phase, as measured by the number of leaves from ground level to the lower ear shoot. The ultimate in earliness emerged when Maiz de Ocho finally reached the Gaspe Peninsula of Canada. In the earliest flowering selections of Gaspe Flint, the vegetative phase is reduced to zero with the ear shoot borne in the axil of the first leaf. The total leaf number is only five so that the preinduced plant comes up ready-formed like a crocus. Most Gaspe Flint plants have a total of seven leaves with a vegetative phase of only two leaves corresponding to two days of growth during germination.

Always somewhat behind in space and time, the more productive dent corns are still spreading northward into the former territory of the flints. By means of introgression into its more southernly neighbors, Maiz de Ocho has always prepared the genetic way for the dented immigrants to settle the north country. When we use the northernmost types of Maiz de Ocho to adapt southern types to the North in our breeding programs, we extend the successes of history. An understanding of the role of Maiz de Ocho in adapting tropical maize to temperate areas

will make it easier for corn breeders to better utilize the entire range of genetic variability in the total maize gene pool.

Walton C. Galinat

Duplicated parts of chromosomes in relation to the origin of maize

The replicated patterns being revealed by studies of comparative DNA sequencing have revived interest in a question that was first phrased by Rhoades (Amer. Nat. 75:105-110, 1951) as follows: "That the architecture of the germplasm of maize contains duplicated regions can hardly be doubted but whether or not they represent vestiges reflecting an ancient amphidiploid origin or represent later occurring duplications cannot be decided at this time." Now 35 years later we still do not have the answer but the new technologies may yet provide one.

It is generally agreed that the genomes of the various races of both corn and teosinte are more or less completely homologous despite their differences in chromosome knobs and allelic frequencies (see pg. 21 and 22 of my review: Chapter 1, The Origin of Corn. G. F. Sprague (Ed.) 1977. Corn and Corn Improvement.) It is, therefore, assumed that no traditional gross translocations have been involved in moving parts of chromosomes around that might result in duplications, at least in recent millennia. If maize (or teosinte) had an ancient amphidiploid origin, the base genomes would have only five pairs of chromosomes. This number does not occur in any of the genera of possible close relatives of maize (Chandravadana and Galinat, 1976) as they fit into a morphological sequence leading to formation of the cupulate fruit case of teosinte (Galinat, 1956). Millions of years back near the beginning of this sequence, the genus *Elyonurus* had a species *(E. argenteus)* with this base number of five pairs (R.P Celarier, 1957, Bull. Torrey Bot. Club 84:157-162). Celarier describes its chromosomes as being long, two of which have terminal knobs as well as knobby regions throughout the length of the chromosomes. In these respects, it has more similarities to maize-teosinte chromosomes than do the tiny knobless chromosomes of *Manisuris* (n = 9). Although native to South Africa and undoubted only remotely related to maize, *E. argenteus* deserves some experimental work including some comparative DNA sequencing with the American Maydeae.

Some of the duplications in the maize-teosinte genome may have been important during the separation of maize from teosinte by domestication and thereby be relatively recent (ca. 8000 years old). The key traits that separate maize and teosinte are each controlled at two loci although usually only a one-allele change is necessary for the switch in F2 segregations. If maize originated several times from independent domestications, sometimes one pair was the key, sometimes it was the other.

A duplication of loci was probably an important factor in evolution of the diverse races of maize. Alternative loci provide double pathways and, thereby, allow the accumulation of genetic variability in the redundant system. With genetic drift into isolated niches, then divergence during adaptation to these different niches followed still later by reconvergence, there is a sudden release of recessives due to recombination between races that depended upon different alternative systems. The double systems may also serve a developmental function in which one replicate has a regulatory action and/or an amplifying effect on the genetic signals from the other (see Galinat, 1982, in Maize for Biological Research, Sheridan, pg. 333, 334).

Walton C. Galinat

The penetrance of the teosinte key traits, two-ranking and solitary female spikelets, in maize

The key traits separating teosinte from maize, specifically two-ranked ears and solitary female spikelets, fail to penetrate a background of modern maize and imperfectly penetrate even a primitive maize background (W.C. Galinat, MNL 45:99, 1971; MNL 46:108, 1972). Transfer of key segments of teosinte germplasm into Texas 4R-3 by P. C. Mangelsdorf was accomplished by selecting not for key traits but for such indicators of teosinte germplasm as cob induration, reductions in ear and kernel sizes and reduced kernel row number (P.C. Mangelsdorf, MNL 21:19-22, 1947). Development of a stable two-ranked, single-female-spikelet maize, designated "airplane corn", was accomplished by reconstructing a congruous background for these key traits by W.C. Galinat (MNL 52:60, 1978; Maydica 30:137-160, 1985).

The cryptic nature of these key trait genes in maize, the apparent reversal of dominance in their evolutionary sequence, and the nature of penetrance in a series of backgrounds is the subject of an ongoing investigation. The 4R-3 teosinte derivatives, later transferred into A158 (P.C. Mangelsdorf, Cold Spring Harbor Symposia on Quant. Biol. 23:409-421, 1958), and A158 derivatives of Central and South American "Tripsacoid" races of maize (P.C. Mangelsdorf, Boletin de la Sociedad Argentina de Botanica 12:180-187, 1968) were crossed with a single plant of the inbred "airplane corn" in the summer of 1983. The F1 hybrids of these seven A158 teosinte derivatives x airplane and ten A158 tripsacoid derivatives x airplane plus an A158 control x airplane were grown out for observation and selfing in 1984. Ear-to-row F2 segregations were grown this past season and are being evaluated now. The essential complex on chromosome 4 is most important in stabilizing

the key traits of teosinte in a maize background (W.C. Galinat, Maydica 30:137-160, 1985). It is marked by the sugary (*su*) gene from the "airplane corn" parent. The F1 generation proved variable in penetrance of ranking and pairing both within and between lines. At one extreme, 73 percent of the F1 ears from the cross involving A158 containing segments of Florida teosinte chromosomes 3, 4, and 9 were two-ranked. Twelve lines, including the three whose parents contained one each of the above Florida teosinte segments, produced less than 20 percent two-ranked ears.

The distribution curve of the index for degree of pairing per ear was unimodal for all F1 lines except for the Honduras-Venezuela derivative hybrid whose pairing index distribution was bimodal. The means of the curves for the other hybrids were similar, clustering around 8.5 on a scale of 12, indicating a fairly strong degree of penetrance of this key trait.

The results suggest that a negative correlation exists between the penetrance of two-ranking and solitary female spikelets. Confirmation of this awaits evaluation of the F2 generation.

The superior penetrance of two-ranking in the A158 Fla 3, 4, 9 hybrid is compatible with the observation by W. C. Galinat (MNL:101-102, 1985) that these teosinte segments are responsible for the rind vascularization necessary for a congruous background for two-ranking.

Ann E. Kennedy and Walton C. Galinat

Noncupulate cob associated with soft outer female glumes

Because the trait for a non-cupulate cob selected by Galinat is deficient in the outer vascular system, the development of the outer glume is not supplemented by an additional source of energy (photosynthate). The result is that it remains soft and similar to the other floral bracts in texture. Its phenotype may appear similar to that of a weak expression of the papyrescent gene (*Pn* on 7L) as well as similar to some of the oldest known specimens of archaeological cobs.

Walton C. Galinat

Chromosome morphology of two inbreds, Mo17 and B73

The comparative cytology of two inbreds of maize, namely Mo17 and B73, has been studied in order to characterize these inbreds by chromosome morphology. Samples of these inbreds were collected at the Suburban Experiment Station at Waltham. Using the customary methods of staining, they were analysed at various stages of meiosis, with special emphasis on pachytene and diplotene stages in order to provide information such as presence or absence of knobs, the number, size and location of knobs, condition of knobs, whether homozygous or heterozygous for the particular knob in question. There is some controversy regarding the knob size, and this is considered to be purely subjective. But even as such it can be seen that in any particular location for a particular chromosome, the presence or absence and the size is always consistent. In these observations the knob size has been classified as small, medium and large.

Inbred Mo17: The sample from this inbred showed normal meiosis. The cytoplasm showed a tendency to stain faster than the chromosomes. At pachytene, synapsis was complete and ten bivalents are formed. A sporadic case of a pericentric inversion was observed (Figure 1). There was no sign of bridges or

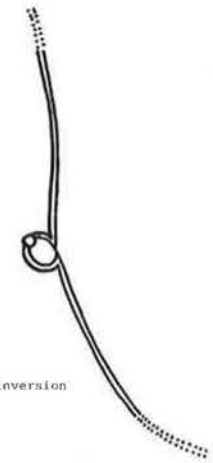

Fig. 1 - Camera lucida drawing of a pericentric inversion

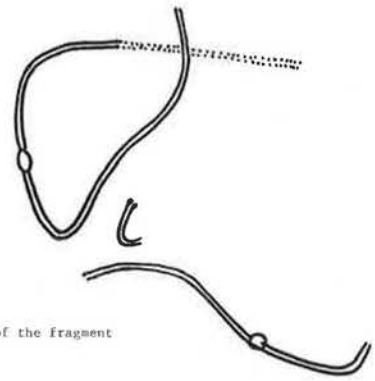

Fig. 2 - Camera lucida drawing of the fragment

fragments. The chromosome involved in this inversion has not been identified yet. Of the ten pairs of chromosomes, only one large knob was observed, and it is perfectly homozygous. The chromosome showed all the characteristics of chromosome 7 in its morphology in that it had an arm ratio of 2.6 and a conspicuous heterochromatic segment in the long arm adjacent to the centromere. The knob is located on the long arm of chromosome 7.

Inbred B73: Meiosis is normal except that it differed from Mo17 in that it showed two large knobs. Based on their total lengths and arm ratios the chromosomes have been identified as chromosome 7 and 8 of the complement. In both cases the knobs were located in the long arm. In this inbred an

interesting feature is the occurrence of a small fragment in almost all the cells analysed. It did not show any association with any one particular chromosome but was seen sticking to one or the other and in some cases free from the rest of the complement (Figure 2). It is not a "B" chromosome.

Significance of these studies is to emphasize the importance of using chromosome analysis especially pachytene morphology with special reference to knob number, location, size and such other features to identify the individual inbreds used in breeding programs. Inbreds have already been fingerprinted based on morphological characters and other traits, and one more criterion based on cytology would improve the sophistication of the existing classification. Variation of knob constitution in different inbreds could be used in identification and purity of the inbreds involved.

Chandra V. Pasupuleti

The cytology of the trigenomic hybrid

The trigenomic hybrid of *Zea mays, Zea diploperennis* and *Tripsacum dactyloides* has been studied in detail. The plant is weakly perennial and maintained by means of cuttings. Samples from this plant were analysed in detail at all stages of meiosis. Maize and *diploperennis* showed normal pairing at pachytene, as a result, 10″ of maize and *diploperennis*, and 18′ of *Tripsacum* were seen in the triploid in a great percentage of cells. At diakinesis the following associations of chromosomes were seen:

10″ (maize + *diploperennis*) + 18′ (*Tripsacum*) - 75% of cells.

9″ + 2′ (maize + *diploperennis*) + 18′ (*Tripsacum*) - 10% of cells.

7″ + 6′ (maize + *diploperennis*) + 18′ (*Tripsacum*) - 10% of cells.

In the remaining 5% of cells various combinations as 1) 5″ perhaps all maize, or all *diploperennis*, + 28′; 2) 5″ + 10′ + 18′; 3) 6″ + 1‴ + 23′. Occasionally the 18′ of *Tripsacum* showed non-homologous pairing within themselves; as a result there were about 12″ + 14′. There were cells that showed more than 38 chromosomes in this triploid material. This could be due to the presence of B chromosomes in the *Tripsacum* background.

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Pleiotropies of recessive alleles and grain yields in maize hybrids

The pleiotropies associated with recessive alleles have now been characterized for ten different recessive alleles in the Oh43 - recessive allele x Oh551 hybrid combination. Of the ten recessives, three increased grain yield in the hybrid. (Increases of 3%, 4%, and 11% respectively.)

The *lty1-16* recessive increased grain yield by 3% in the Oh551 hybrid combination. Preliminary studies were conducted to determine if the *lty1-16* allele would increase grain yield in other hybrid combinations. Grain yields were increased in two other hybrid combinations (increases of 5% and 7% respectively) and not in three combinations. The pleiotropy affecting grain yield appears to be turned on or off in specific hybrid backgrounds. (Incidentally, this type of gene action may explain how phenotypic traits skip generations in a family pedigree.)

Many inbred plants homozygous for a specific recessive allele are as vigorous as the normal inbred plants. Thus, the production and selection of recessive mutant alleles in an inbred line and their testing in various hybrid combinations provides an effective method for increasing grain yields in maize hybrids.

E. J. Dollinger

* in symbol identifies loci needing documentation, symbol standardization and/or allelism tests
BS = base sequence; RM = restriction map; RE = restriction endonuclease; R/S = resistance and susceptibility
r refers to numbers of references in the list of Recent Maize Publications

CHROMOSOME 1

K1S: C banding in Zapalote Chico --Aguiar-Perecin &, r4,r5
Adh1-S3034 RM, Mu1 insertion --Alleman &, r12
Adh1-n, -2, -4, -5, -6; Mdh4-n, -9, -12, -14.5, -15.5, -16; Mmm1-Mmm, -mmm, -m1, -m3; Pgm1-1, -7, -9, -9.5, -13, -16, -16.5, -17, -18, -19, -21; Phi1-1, -2, -3, -4, -5, -6: alleles in races & teosintes --Doebley &, r119; Smith &, r522
Adh1: 3 sites of S1-nuclease sensitivity; hypersensitivity sites for REs --Ferl, r149,r151
Kn1-O, -Z2, -N1, -N2, -2F11, -Z3, -Z4, Rs1-R allelic --Freeling &, Hake &, r161,r202,r203
hcf3, hcf6 uncovered by TB-1Sb; hcf50 by TB-1Lc --Miles &, r341
Adh1-Fm335 and revertants: RM, BS, Ds1 insertion --Peacock &, r404
Adh1-4, -6, -null; Mdh4-12, -14.5; Mmm-Mmm, -mmm; Pgm1-9, -16; Phi1-2, -3, -4, -5: alleles in lines & hybrids --Smith &, r519,r520,r521
bz2-mu1 Mu-elicited --Walbot &, r576
Adh1-S4477, -S4478, Mu insertions --Rowland &, r463
Adh1-S3034a null allele: Mu-1 and exon deletion 74bp, RM, BS --Taylor &, r545
Adh1-3F1124 Mu3 insert --Chen &, 60:24
P1-Quebec36 --Peterson, 60:36
P1-VV, Ac-hybridizing 8kb SalI clone; P1-RR revertant 3.5kb --Peterson &, 60:36
Ds2 from Adh1-2F11: BS --Merckelbach &, 60:39
bz2-m, Ac-hybridizing 4.3kb BglII clone; revertants and Bz2 1.3kb smaller; an1-bz2-6923 lacks hybridization --Theres &, 60:40
P1-vv, Ac-hybridizing 8kb SalI clone absent from P1-ww; P1-rr 3.5kb --Lechelt &, 60:40
clf1-792, cp*-991, cp*-1078B, de*-1142, cp*-1308A on 1S; dsc*-1315A on 1L --Chang &, 60:46
cfr*-2018, chloroplast coupling factor, uncovered by TB-1Sb and TB-1Sb-2Lc --Echt, 60:49
Les2 - 1 - sr1; Les5 (was Les*-1449) - 20 - sr1; lls1 - 20 - sr1 --Hoisington, 60:51
P1-WWB (silk browning) vs. P1-WWW (green) --Coe, 60:55
Adh1, RM upstream --Paul &, 60:61
dek1-54-1, dek1-56-1 --Racchi &, 60:100
Acp4 - 13 - Dia2 - 15 - Phi1 - 14 - Adh1 --Wendel &, 60:109
RFPs 74, 20, 40, 84, 96, 304, 30, 19, 120, 10, 54, 99, 82, 24, 65 mapped --Helentjaris &, 60:119
Mu1.4MF-Adh1-S3034, -S3034a --Walbot, 60:120

CHROMOSOME 2

K2S, K2L: C banding in Zapalote Chico --Aguiar-Perecin &, r4,r5
Dmr* (downy mildew resistance) --Ford &, r155
v*-424, -350, -588 allelic; not w3 or v4 --Polacco &, r429; 60:45
Les1 - 1 - wt1; Les*-2004 on 2S; Les4 (was Les*-1375), Les*-1378, Les7 (was Les*-1461) on 2L --Hoisington, 60:51
lg1 - 27 - nec4; gl2 - 4 - nec4; nec4 - 22 - B1; nec4 - 0 - d5 --Hoisington, 60:51
Rf4 independent of v16, j1 --Sisco, 60:112
RFPs 32, 46, 61, 118, 4, 121, 123, 49, 11, 57, 308, 314, 319, 320, 330, 336 mapped --Helentjaris &, 60:119

CHROMOSOME 3

K3S, K3L: C banding in Zapalote Chico --Aguiar-Perecin &, r4,r5
E8-n, -2.5, -3, -4, -4.5, -5, -5.8, -6, -7, -8; Got1-n, -1, -1.2, -4, -5.8, -6, -6.2, -7.5; Mdh3-n, -11.5, -15.8, -16.3, -16, -16.9, -17.2, -18; Me1-n, -S, -F, -R; Pgd2-n, -2.8, -5, -8, -10, -11: alleles in races & teosintes --Doebley &, r119; Smith &, r522
a1-m1-6078 I insert 2242bp and identical to Spm-I8; a1-m1-5719A-1 I insert 789bp, deletion product of Spm-I8 --Gierl &, r183; Schwarz-Sommer &, r494
a1-m61138-1, -pale-m(r), -m(Au), -m(papu), -m(dense) En-mediated --Peterson, r411
A1-Can progenitor of a1-m1:ACan, -m2:ACan, -m3:ACan, Dt-mediated --Pryor, r436
a1-Mum1, -Mum2, -Mum3 Mu-elicited --Robertson &, r458
Got1-4, -6; Mdh3-16, -18; Pgd2-2.8, -5: alleles in lines & hybrids --Smith &, r519,r520,r521
Mu-homologous clone c10Mu from a1-Mum2 stock and Spm-I8/En1-homologous clone c110En from a1-m(papu) stock cross hybridize; RMs --O'Reilly &, r380
a1-m2-8004 Spm- and En/I-mediated; a1-m2-7977B, -m2-7995, -m1-5719A-1, -m1-5720, -m1-5996-4, -m1-6078 pedigrees --Reddy &, r449

Pgd2-N, -2.8, -2.8*, -5, -6, -10, -11 alleles in races --Stuber &, r540
vp1-Mum1 Mu-elicited --Stinard, 60:7
g2 - 32 - d1; g2 - 34 - ra2; g2 - 49 - Rg1; g2 - 52 - g16; g2 - 50+ - E4; d1 - 15 - E4; E4 - 11 - g16 --Whalen &, 60:30,31
Pgd2-1, -2, -2.8, -5 --Kahler &, 60:31
sh*-627D on 3L, sh*-1339A on 3S --Chang &, 60:46
TB-3Lc breakpoint 0.55-0.65 --Kindiger &, 60:50
dek24 on 3S --Sheridan &, 60:64
Rg1 - 1 - Lg3 - 3 - Rf1; Rg1 - 12 - ra2 - 5 - d1; Lg3 - 16 - ra2 - 7 - d1 --Poethig, 60:109
Tpi4 not uncovered by TB-3La, TB-3Lf, TB-3Lg --Wendel &, 60:112
RFPs 89, 71, 13, 70, 108, 90, 52, 94, 78, 88, 91, 42, 31, 83, 55 mapped --Helentjaris &, 60:119

CHROMOSOME 4

K4S, K4L: C banding in Zapalote Chico --Aguiar-Perecin &, r4,r5
Rf*-var (restorer of cms-T) --Gontarovsky, r188
f12 - 0 - Zp12/29*; f12 - 1.25 - Zp23/16*; Zp12/19* - 1.25 - Zp23/16*; Zp22/6* was Zp6h*; Zp22/10* was Zp22*; Zp22/12* was Zp12*; Zp22/14* was Zp14*; Zp22/15* was Zp15*; Zp22/22* was Zp22h*; Zp20/27* was Zp27*; Zp20/28* was Zp28*; Zp20/29* was Zp29*; Zp20/30* was Zp30* --Hastings &, r215
Bx1 uncovered by mono-4, TB-4Sa; su1 - 41.2 - bx1 --Simcox &, r509
c2-m857210, -m857212, -m857213, -m857214, -m857229, -m857244, -m857246, -m857263, -m857271, -m857272, -m857273, -m857274, -m857328, -m857353, -m857364, -m857375, -s857230, -s857231, -s857234, -s857236, -s857241 --Peterson, 60:2
su1-4582, -4059 Mu-induced --Robertson, 60:13
c2-m1, Spm-hybridizing clone; c2-m2 hybridizes, contains Spm-I2, deletion derivative; C2 cDNA homology to parsley chalcone synthase --Wienand &, 60:42
sh*-912 on 4S, ptd*-1130 on 4L --Chang &, 60:46
TB-4Lc breakpoint 0.15-0.20; TB-4Lf 0.15-0.20 --Kindiger &, 60:50
dek25 on 4S --Sheridan &, 60:64
Zpr10/(22)* - 24 - f12; Zp10/(22)* - 0 - Zp22/6* --Benner &, 60:114
RFPs 6, 77, 18, 73, 27, 95, 12, 17, 36, 66, 56, 104, 307, 310, 311, 313, 317, 333 mapped --Helentjaris &, 60:119

CHROMOSOME 5

K5S, K5L: C banding in Zapalote Chico --Aguiar-Perecin &, r4,r5
Hsf1 (Hsf*-1595), hairy-sheath-frayed; Hsf*-1603 --Bird &, r49
Got2-n, -2, -4, -6, -7; Got3-n, -2, -3, -4, -6, -7; Mdh5-n, -5.5, -7, -8, -12, -14.4, -15, -16, -16.4; Pgm2-0.45, -0.5, -3, -4, -7.2, -7.3, -7.5, -8, -12: alleles in races & teosintes --Doebley &, r119; Smith &, r522
bt1-4 allele --Dollinger, r120
hcf38 uncovered by TB-5La --Miles &, r341
Got2-2, -4; Got3-4; Mdh5-12, -15, -null; Pgm2-3, -4, -8: alleles in lines & hybrids --Smith &, r519,r520,r521
a2-m1(1511), En-hybridizing clone --Martin &, 60:42
cp*-863A, de*-1196, cp*-1275A, fl*-1299 on 5L; cp*-931A on 5S --Chang &, 60:46
TB-5Sc breakpoint 0.3 --Kindiger &, 60:50
dek26, dek27 on 5L --Sheridan &, 60:64
Spm-P linkage with wx T5-9c --Phillips &, 60:115
RFPs 109, 93, 106, 115, 60, 53, 116, 41, 75 mapped --Helentjaris &, 60:119

CHROMOSOME 6

K6L2, K6L3: C banding in Zapalote Chico --Aguiar-Perecin &, r4,r5
Enp1-n, -2, -4, -5, -6, -6.2, -7, -8, -10, -14; Idh2-2, -3.8, -4, -4.1, -4.2, -5, -6, -7, -7.5, -7.8; Mdh2-0.2, -0.4, -3, -3.5, -3.8, -5, -5m, -5.6, -6, -7.7; Pgd1-n, -0.5, -1, -1.5, -1.8, -2, -3.8: alleles in races & teosintes --Doebley &, r119; Smith &, r522
ms4 allelic to pol --Golubovskaya &, r186
Enp1-4, -6, -null; Idh2-4, -4.2, -6; Mdh2-3, -3.5, -4.5, -6, -null; Pgd1-2, -3.8, -null: alleles in lines & hybrids --Smith &, r519,r520,r521
Pgd1-N, -0.5, -1, -2, -2.8, -3.8, -7, -12 alleles in races --Stuber &, r540
rDNA9.1, 8.0, 6.9: HpaII isomorphs --Jupe, 60:22
Enp1-1, -2, -3, -4, -6, -8, -10 --Kahler &, 60:31
sat-Pororo --Beckett, 60:44
de*-1296A on 6L --Chang &, 60:46
TB-6Lb breakpoint 0.65 --Kindiger &, 60:50
rDNAspacers and repeats --Toloczyki &, 60:58

dek28 on 6S --Sheridan &, 60:64
rDNA clone pGMR3 SstI polymorphisms 3.6 vs. 3.9kb, 5.3 vs. 3.6 & 1.6kb --Rocheford &, 60:73
Adk1 - 15 - Pgd1 - 3 - Enp1 - 42 - Hex2 - 43 - Idh2 - 2 - Mdh2 --Wendel &, 60:109
li2 - 1 - y1 - 0.5 - li0 - 0.5 - ms1; y1 - 1 - ms*-s1 —Mascia &, 60:113
RFPs 38, 63, 67, 102, 9, 2, 100, 101, 7, 302, 320, 325 mapped --Helentjaris &, 60:119

CHROMOSOME 7

K7S, K7L: C banding in Zapalote Chico --Aguiar-Perecin &, r4,r5
Px3-1, -2, -3, -4, -5, -6 alleles in 133 inbreds --Brewbaker &, r61
(Zp20/1*, Zp20/2*, Zp20/3*, which were Zp1*, Zp2*, Zp3*) - 4.5 - wx T7-9(4363); Zp10/20-16* - 1.9 - wx T; Zp20/1* - 3.8 - Zp10/20-16*; Zp20/1* - 15.0 - o2; Zp10/20-16* - 16.1 - o2; o2 - 22.7 - wx T; o2 - 9.0 - Zp18/15*; Zp20/1* - 7.7 - wx T; Zp20/1* - 2.5 - Zp10/20-16*; Zp20/1* - 23.9 - Zp18/15*; Zp10/20-16* - 2.5 - wx T; Zp18/18* - 20.6 - Zp20/20-16*; Zp18/15* - 32.9 - wx T; Zp20/6 was Zp6*; Zp20/16* was Zp16*; Zp20/22* was Zp20*; Zp20/29* was Zp29* --Hastings &, r215
O2-m1, o2-m2, O2-m3, O2-m4 alleles Ds-elicited --Motto &, r356
o2-wh, -261, -Columbian, -Agroceres Bg-mediated —Salamini &, r477
o*-1241 on 7L --Chang &, 60:46
TB-7Lb breakpoint 0.34 --Lin, 60:54
Les9 (was Les*-2008, Les*-F331035142) - 3 - ra1; o2 - 16 - Les9; v5 - 4 - Les9; Les9 - 9 - gl1 —Hoisington, 60:52
RFPs 29, 26, 45, 113, 35, 59, 68, 5, 23, 112, 47, 111, 30, 48, 44, 28 mapped --Helentjaris &, 60:119
K7L in Mo17 and B73 inbreds --Pasupuleti, 60:132

CHROMOSOME 8

K8L1, K8L2: C banding in Zapalote Chico --Aguiar-Perecin &, r4,r5
Ac2, activator of bz2-m --Dempsey, r106
Idh1-2, -3, -4, -6, -8; Mdh1-n, -0.05, -0.1, -1, -2, -2.8, -5, -6, -6.4, -8.5, -9.2, -10.5: alleles in races & teosintes --Doebley &, r119; Smith &, r522
o6 allelic to pro1 --Salamini &, r477; Tonelli &, 60:100
Idh1-4, -6, -8; Mdh1-1, -6: alleles in lines & hybrids --Smith &, r519,r520,r521
pro1-1121; crp*-1528 on 8L; rgh*-974A, o*-1096A, fl*-1145A, cp*-1255B, gm*-1319C, cp*-1406 linkage indicated with TB-8Lc --Chang &, 60:46
TB-8Lc breakpoint 0.24±0.05 --Chang &, 60:46
ats, atrazine susceptible, on 8 (Scott &, 1969) --Coe &, 60:55
dek29 on 8L --Sheridan &, 60:64
ms43 uncovered by TB-8La —Golubovskaya &, 60:107
Idh1 uncovered by TB-8La, Mdh1 not; Idh1 - 30 - Mdh1; Tpi3 - 22 - Mdh1; Tpi3 - 39 - Idh1 --Wendel &, 60:109
Rf4 independent of v16, j1 --Sisco, 60:112
RFPs 3, 69, 43, 33, 50, 103, 39, 1, 72, 37, 64, 110, 79, 114, 107, hsp1, 88, 301, 303, 315, 322, 328, 331 mapped --Helentjaris &, 60:119
K8L in B73 inbred -- Pasupuleti, 60:132

CHROMOSOME 9

K9S: C banding in Zapalote Chico --Aguiar-Perecin &, r4,r5
bz1-Mum4 allele, Mu-induced --Bennetzen, r41
Sh1 alleles: RMs --Burr &, r70
wx1-m7 Ac sequence; sh1-m5933 Ds insertions, RM, introns; Sh1-r5 Ds alteration --Courage &, r91
Sh1-A, -B; sh1-5582, -5586 with Tz86 insertion: RMs --Dellaporta &, r104
Dp9-McC, Dp9-MMR; R9 (rearranged 9) of McClintock --Dempsey, r106
Acp1-n, -1, -2, -2*, -3, -3.5, -3.8, -3*, -4, -5, -5.5, -6: alleles in races & teosintes --Doebley &, r119; Smith &, r522
bz1-s:2114(Ac) stable from bz1-m2(Ac): RM shows 1kb deletion from Bz; bz1-m2(DII): deletion mutation of Ac to Ds (3.9kb) --Dooner, r121
Bz1-McC, bz1-m1, bz1-m2, bz1-m2(DI): RMs --Dooner &, r123
sh1-5933, Ds insertion and structure; derivative Sh1-r5 revertant --Doring &, r125
bz1-m2 Ac, bz1-m11 Spm11, bz1-m13 Spm13, wx1-m8 Spm8: RMs —Fedoroff &, r147
Wx1-9r1, wx1-m9 Ac, wx1-m9 Ds, bz1-m2 Ac: RMs --Fedoroff &, r148
wx1-844 En1 insert in intron 8.4kb, BS; wx1-m8 Spm-18 insert in exon 2242bp, BS --Gierl &, r183,r184
hcf42 uncovered by TB-9Lc —Miles &, r341
C1-I-m836976 En-mediated --Peterson, r411

bz1-m2 and derivatives -m2(DII) and Bz1-wm Ac and Ds insertions; bz1-m13 derivatives -m13CS1, -m13CS3, -m13CS5, -m13CS6, -m13CS9, -m13CS12, Bz1-'3: dSpm13 insertion and derivatives - RMs --Schiefelbein &, r487,r488

Acp1-2, -3, -4, -5.5, -6: alleles in lines & hybrids --Smith &, r519,r520,r521

wx1-844 En-1 insertion: RS, En-1 and junction BS --Pereira &, r488

wx1-m8 Spm-I8 and revertants: RM, BS --Saedler &, r474; Schwarz-Sommer &, r495

sh1 genomic BS, upstream and 3'-untranslated end --Werr &, r584

wx1-90, -R, -B, -6, -I, -K, -M, -C1, -C2, -C3, -C4, -C31, -C34, -B1, -B2, -B5, -B6, -B7, -B8, -c, -Stoner, -BL2 alleles: RMs, insertions, deletions --Wessler &, r586

c1-846432 mutation from C1-I in En stock --Peterson, 60:2

Df-c1-sh1-bz1-857003, -857164, -857182, -857183; Df-c1-857177; Df9S-857078; C1-I-weak857054, -I-weak857061; -I-m857056, -I-m857070, -I-m857101; sh1-857011, -857015; wx1-857027, -85-1 --Peterson, 60:3

bz1-mus4, -mus7, -mus8 --Schnable &, 60:4

sh1-bz1-82q760, -825211x, -825211y-1, -825212x-1, -825215z; c1-sh1-bz1-825211w, -825211y-2 --Schnable &, 60:5

wx1-Mum1, -Mum2, -Mum3, -Mum4 --Sackitey &, 60:6

Df9S108-8, 110-8, 110-9, 114-1, 117-8, 116-10, 104-7, 107-2, 108-3, 110-6, 117-5, 107-1 --Robertson &, 60:11

bz1-m5-A, -m5-B, -m5-C, -m5-D Ac insertions --Johns &, 60:20

c1-m668655 and c1-m668613, En-hybridizable clones 9.4kb; c1-m2, Ds-containing clone 3kb; inserts into 1kb fragment; C1-I clone rearranged --Paz-Ares &, 60:42

lo2 distal to TB-9Sb --Beckett &, 60:43

o*-744 on 9L; cp*-1054 on 9S --Chang &, 60:46

TB-9Sd breakpoint 0.08±0.24, centric heterochromatin; TB-9Lc breakpoint 0.1 --Kindiger &, 60:50

Les8 (was Les*-2005) on 9S --Hoisington, 60:51

Ss2, sucrose synthetase-2, RM; RFP-80 - 2 - Ss2; Ss2 - 21 - RFP-14 --McCarty &, 60:58,60

dek30 on 9L --Sheridan &, 60:64

C1-Im1, C1-Im2, c1-xm --Mazoti, 60:75

RFPs 86, 34, 80, sus2, 14, 97, 25, 98, 305, 309, 324 mapped --Helentjaris &, 60:119

CHROMOSOME 10

Glu1-n, -1, -2, -2.5, -3, -3.2, -3.5, -4, -5, -6, -7, -7.8, -8, -10, -11, -12, -13, -16: alleles in races --Doebley &, r119

Sn1-s, Sn1-w, Sn-bol3 (scutellar node pigmentation) --Gavazzi &, r172,r173

Zp22/3* was Zp2* --Hastings &, r215

r1-r:n46, -r:n35, -r:n101, -r:n156; r-m1 (Ds insertion); R1-N1-571-1, -N1-571-2, -N1-575-1, -N1-576-3, -N1-571-3, -N1-575-3, -N1-575-4, -N1-576-5, -sc:124, -g:8pale: origins, components and recombination --Kermicle, r267

K10-I, K10-II; DfK10(C), (F), (H), (I), (K) vs. l13, w2, sr2 and recombination with R1 --Rhoades &, r455

Glu1-1, -2, -3, -6, -7, -9, -10, -null: alleles in lines & hybrids --Smith &, r519,r520,r521

K10-I, K10-II; in DfK10(C), DfK10(H), lacking terminal knob, neocentromeric and preferential segregation activity continues; R1-sr2 recombination with DfK10(H), (F), (C) heterozygotes --Rhoades &, 60:26,27

msc*-1330 on 10L --Chang &, 60:46

TB-10Sc breakpoint (0.3; TB-10L19 centromere; TB-10L30 0.13; TB-10L32 0.74 --Lin, 60:54

Les6 (was Les*-1451), Les*-1453, Les*-2016 on 10S --Hoisington, 60:51

Oy1-1459 --Hoisington, 60:57

Glu1 - 1 - Sad1 --Wendel &, 60:109

RFPs 105, 85, 92, 22, 306, 312, 318, 321, 326 mapped --Helentjaris &, 60:119

UNPLACED

Lxm*-1600, lax midrib; Rld*-1608 and Rld*-1441, rolled leaf; Rs*-1606, rough sheath --Bird &, r49

Px1-null, -1, -2, -3; Px2-1, -2; Px4-1, -2, -3 (& null lines occur); Px5-null, -1; Px6-null, -1; Px7-1, -2 (& null lines occur); Px8-1, -2; Px9-null, -1, -2: distributions in inbreds and varieties --Brewbaker &, r61

Cat3-n, -5, -7, -9, -9.5, -10.4, -10.6, -11.2, -12: alleles in races & teosintes --Doebley &, r119; Smith &, r522

zpg1 (zebra stripe and pale green); lty1, lty2 (light yellow endosperm); ora3 (orange endosperm) --Dollinger, r120

Sw*-1, Sw*-2 (Stewart's wilt resistance) --Ford &, r155

Kn2-Z4, knotted ligules; unlinked to Kn1 --Freeling &, r161

Dt-TL (transposed light dotted) --Pryor, r436

Cat3-7, -9, -12: alleles in lines & hybrids --Smith &, r519,r520,r521

Mu-L element RM: 300bp larger than Mu1, polymorphisms, small deletion --Walbot &, r576

de*-B70, de*-B40, De*-B30, de*-B3, -B22, -B50, -B76, -B1, -B116, -B18, -B37, -B246, -B112, -B21, -B69 --Trapani &, r560

Zein polypeptide IEF nomenclature --Wilson, r595,r596

Ac-flow --Schnable &, 60:5

pi1, pi2, secondary pistillate florets, duplicate factors (W. A. Huelsen &, 1929) --Kindiger &, 60:43
Mof*-1, Mof*-2, modifiers of v*-576 expression --Polacco, 60:45
orp*-1186, smk*-1373A, sh*-1053B, crp*-888A, o*-945A, fl*-1145A, fl*-1163, de*-1177A, cp*-1294, o*-1298, sh*-1322A, sh*-1324A, smk*-1437 --Chang &, 60:46
hcf*-1218-20, Mu-elicited --Cook &, 60:47
Les*-1348, Les*-1442, Les*-2003, Les*-2007, Les*-A762, les*-A467, les*-1395, les*-1521C, les*-2012, les*-2013, les*-A721 (allele of les*-A467) --Hoisington, 60:51
orp1, orp2, orange pericarp, duplicate factors --Neuffer &, 60:55
Wlf*-1726, wide leaf; lgp*-2086, large plant; erl*-2077, early; lgp*-2087, large plant; mbr*-2088, many-branched tassel; Erl*-2102, early; erl*-1729, early; shp*-1749, short plant --Bird &, 60:56
meg1 (miniature endosperm and germ), tts1, air1, sin1 (short internodes), dul1, tea1, prf1 (prolific), prf2 (prolific), ffr1, ffr2, ffr3, ffr4, fsh1, fmd1, fww1 --Mashnenkov, 60:71
Lt*-19, Lt*-20 --Frisch &, 60:115
RFPs 323, 327, 334 on chromosome 1 or 5; RFPs 201, 202 unassigned --Helentjaris &, 60:119
MuE-; Mu1.4VS-B37 --Walbot, 60:120

CHLOROPLAST

Polypeptides A1 & A2, genes psiA1 & psiA2, RM, BS --Fish &, r153
rbcL, atpB RM, BS upstream --Mullet &, r359
Photogene-containing regions, RMs and map locations --Rodermel &, r460
rRNA16S BS upstream; tRNAvalGAC BS upstream --Strittmatter &, r538

MITOCHONDRION

F1-ATPase alpha subunit, atpalpha RM, BS --Braun &, r60
Plasmid 1.94kb present in cms-I, -J, -RU, -S but absent in others of S group --Carlson &, r74
rDNA-18S BS --Dale &, r99
ATPase subunit 6, atp6 RM, BS --Dewey &, r111
F0-ATPase proteolipid, atp9: RM, BS --Dewey &, r112
tRNAhis BS; identical to ctDNA-tRNAhis --Iams &, r239
cox1 (cytochrome c oxidase subunit I) BS --Isaac &, r245
cms-G', -J', -R' revertants to cytoplasmic fertile in WF9 retain S1 and S2, have RE changes in main genomic sequences --Ishige &, r247
Sequences homologous to ctDNA-rbcL, ctDNA-rDNA16S, ctDNA-rDNA23S --Lonsdale, r304
Genome, RM for Wf9 normal cytoplasm --Lonsdale &, r306
tRNAphe BS, map location --Marechal &, r326
S1 BS; 3 URFs --Paillard &, r386
tRNAmet1, tRNAmet2, tRNAasp: BS, map location --Parks &, r395,r396
cms-R251, -R274, -R285, -R296, -R369, fertile revertants from -VG, and -R733 from -S: RM, BS; rearrangements and deletions at one end upon integration of S2 sequences --Schardl &, r484,r485
cms-S subgroups by fertility -B/D, -CA, -LBN, -ME, -S(USDA); RE correlations --Sisco &, r517
cms-T reversions, BS --Wise &, 60:63
R1 vs. S1 vs. S2, BS --Elmore-Stamper &, 60:110
Homologies to ctDNA-rRNA23S, -rRNA4.5S, -rRNA5S, -tRNAarg --Braun &, 60:111
cms-LBN different from cms-L in LBN-specific dsRNAs in the same nuclear background --Sisco, 60:111
cms-ME(38-11) possible revertant to fertility --Sisco &, 60:113
cms-T revertant R2: RE digests --Gengenbach &, 60:114
cms-J', cms-G', cms-R' revertants, RE digests --Ishige &, 60:126
COI changes RE patterns with nuclear change (backcrossing), COII and COB do not; S2 lags in changing --Escote &, 60:127; Kidwell &, 60:128

cDNA/GENOMIC CLONES/PROBES

Mu2 element, 1.7kbp, 0.4kb larger than Mu1; transpositions, segregation, copy nos. --Alleman &, r12
ATP/ADP translocator (nuclear coded mitochondrial) cDNA RM & BS --Baker &, r26
Two cDNA clones (1-407, 1-634) detect no polymorphisms in 3 inbreds; 16 cDNA clones [1-45, -67, -89, -123, -183, -297, -326, -343, -380, -501, -556, -779 (= -80); 2-226, -323, -369; B59] detect polymorphisms (2 to 8 isomorphs) in 2 to 6 inbreds --Burr &, r70
Z19 and Z21 zeins, conserved BS ca. -300bp upstream as in wheat & barley prolamins --Forde &, r156
cDNA clone pMx71 selects RNA for an anaerobiosis-induced aldolase; pMx59, pMx96 and pMx97 for other induced proteins --Hake &, r204
Cin1-001, -102, -103, -201: BS --Hehl &, r220

cDNA clones 9-72sa and 6-59s reveal polymorphisms in inbreds P4 vs. P7 with linkage; 7-31sb and 9-55s linked --Helentjaris &, r221
Bs1 transposable element: RM, distributions and variations in lines --Johns &, r253
Ds101, Ds103, Ds105, Ds132 elements: BS --Peacock &, r404
cDNA clone pME119, for 28kd glutelin-2: RM, BS --Prat &, r432
Heat shock protein 70 cDNA clone pMON9502: BS --Shah &, r501
Zein zA1 and cM1 cDNA clones: BS --Spena &, r527
Zein M6, M8, My2, E19 cDNA clones: BS --Viotti &, r572
Repetitive-sequence clones H2a, H2b, S1, T2, T3 and P(186bp): BS, in situ on A and B chromosomes --Viotti &, r573
Clone pCTE1409, 200bp, snap-back selected --McElfresh &, 60:17
Spm-s, -w; dSpm --Fedoroff, 60:18
cDNAs from Ac-hybridizing transcripts; translations from ORF1 --Kunze &, Muller-Neumann &, 60:39
Clone pZm9'-8 homologous to Expressed Meiotic Prophase Repeat of Lilium, BS --Bouchard, 60:71
Clones pZmS21, pZmS42 (mtDNA-S1): nuclear DNA homology, polymorphisms --Baszczynski &, 60:101

RESISTANCE/TOLERANCE/HERITABILITY

DNA concentration, soluble protein (SP), chlorophyll (Chl), Rubisco activity (RA) and quantity (RQ), pyruvate Pi dikinase activity (PDK) in leaves: GCA in 6x6 diallel highly significant, SCA less important. Heterosis over midparent highly significant for SP, Chl, RQ. High PDK in W37A dominant over low in Oh43, segregates in F2 and BCs. High DNA in Oh43 dominant over low in Mo17, segregates in F2 and BCs --Baer &, r22
DNA C values vary from 2.46pg in Seneca 60 (which has only 3 C-bands) to 3.37pg in Zapalote Chico (12-13 C-bands) --Bennett, r40
Alpha and gamma-tocopherol in embryos: heritability (h^2) 0.62 to 0.68 --Galliher &, r169
Tassel weight (TW), branch number (NB), length (TL) and number of ears (NE) highly heritable; yield (Y) correlated negatively with NB and positively with NE --Geraldi &, r180; Souza &, r525
European Corn Borer (Ostrinia nubilalis Hubner) vs. Northern Corn Leaf Blight (Helminthosporium turcicum Pass.) R/S not correlated among 7,537 screened genotypes --Guthrie &, r199
European Corn Borer R from inbred B86 partially dominant in a series of single crosses --Guthrie &, r200
Cold tolerance selection in BS13(SCT) and BSSS2(SCT) gains for percentage emergence, seedling dry wt. and seedling vigor --Hoard &, r227
Pollen tube growth affected by sporophyte; 8x8 diallel, dominance and additive effects; heterosis --Kumar &, r283
Pest R or yield, recurrent selection in 10 varieties, diallel evaluation shows joint selection required --Lamkey &, r289
Kernel breakage R/S highly heritable, correlated with flowering date --Leford &, r294,r295
Trypsin/activated Hageman Factor XIIa inhibitor protein heterogeneous at one aa position; 2 loci suggested --Mahoney &, r321
Stalk crushing strength increase 12-25% per cycle by recurrent selection; yield, lodging, kernel breakage and plant density correlations --Moentono &, r345
Yield correlates with plant dry wt., harvest index, rate of grain fill, & negatively with the silk-to-pollen interval among 4 O.P. varieties and 24 single crosses representing 4 per 10-year era; genetic gain for yield 78.8% of the total yield gain --Russell, r466,r467
Total leaf number (TLN), days to tassel initiation (DTI), days to pollen shed (DPS) in 6x6 diallel, 18 latitudes & short/long photoperiod phytotron: gca major contributor to each variable vs. sca; photoperiod primary for gca x environment for TLN and DTI --Russell &, r470
Embryogenic callus response largely additive in a partial diallel, maternal effects; heterosis --Tomes &, r557
Early flowering selection, 11 cycles, advanced 11 heat units (ca. 1 day) per cycle --Troyer &, r561
Flooding susceptibility in Mo17 vs. A632 not associated with ADH or pyruvate decarboxylase levels --VanToai &, r565
Fall armyworm (Spodoptera frugiperda) larval growth and preference for callus correlate with R/S of hybrids --Williams &, r591
Southwestern corn borer [Diatraea grandiosella (Dyar)] larval growth on callus in 6x6 R/S diallel correlates with leaf feeding; gca and sca important --Williams, r592
Earworm (Heliothis zea) larval growth and relative maysin content not correlated in silks of increasing age for R/S strains --Wiseman &, r599
Earworm (Heliothis zea) association with Bi, P1-WW, P1p1 in Purple Husk Cateto vs. IAC Maya --Miranda &, 60:34
Goss's wilt [Corynebacterium (Clavibacter) michiganense] R/S associated with T8-9(6673), T4-9e, T7-9a --Rocheford &, 60:73
Pericarp thickness, Z. perennis vs. Gaspe, partially dominant, multifactorial, shows metaxenia --Bertoia &, 60:86
Multilayer aleurone, Z. perennis vs. Gaspe, single recessive factor --Bertoia &, 60:88

--Assembled unrestricted by Prof. Ligate

Ligate's Corner:
Slacks are inactive genes

IV. MAIZE GENETICS COOPERATION STOCK CENTER

The Maize Genetic Stock Center would like to have some feedback concerning the needs of the cooperator community and our ability to satisfy them in the past few years. We have distributed over 2,300 packets this past year in response to over 150 different requests. We try to provide the materials as close to the requested material as we have on hand.

On 10 December 1985 we sent out approximately 180 letters to Cooperators asking them for specific information concerning the service that the Stock Center has provided in the past. Approximately 20 percent have already responded. For this we are grateful and many very useful suggestions have been made that we will attempt to incorporate in our future operations. The main concern mentioned is the furnishing of homozygous materials. This is a tough nut to crack.

One clarification is important. The items listed in the stocklist in the past have only indicated that we have stocks that contain the mutants indicated. It has never meant that all of those items were available in a homozygous condition nor in a variety of inbred backgrounds. Many of the mutants such as the viviparous group can not of course be provided in a homozygous condition. Others such as brittle are difficult to maintain in a homozygous state. The best results in general are obtained by crossing the stocks to a vigorous and suitable hybrid and then selfing this to get a segregating stock that provides reasonable quantities of more robust plants. Recently the needs of physiologists and genetic engineers have put a premium on providing known homozygous mutants in known inbred backgrounds. At present about the only stocks available in particular inbred backgrounds are endosperm traits, male steriles, virescents and glossies. We are underway with the project discussed at the March 1984 meeting of developing pairs of good markers in both arms of all 10 chromosomes. This summer was the second one in this program and progress has been good thus far.

In order to meet the needs of these specialized researchers we are attempting to expand the numbers of stocks that <u>are</u> available in the homozygous condition and in known backgrounds. This is an enormous project which makes our work more difficult as well since the known homozygotes often produce very poor ears. We will publish two stock lists this year, the major one listing, as in the past, all stocks that we can furnish which will contain the desired mutant and if it is in a segregating material we will insure that you should get at least 12 kernels that carry the desired trait or traits (in a 1:1 ratio we will send at least 24K, in a 3:1 at least 48). The other stocklist will list those stocks that we can furnish in a known homozygous condition. In future years we will attempt to expand the listing and provide information on inbred backgrounds where this can be done. This project will run parallel to the selected pairs of markers in selected inbreds mentioned earlier.

At the present time we are updating the entire computerized inventory to enable us to extract information on the homozygous condition and backcrossing information on all of the stocks. This should be a big project.

We have received some new stocks from Missouri this year and we are anxious to add any and all genetic materials that wuold be of use to the cooperators in general.

As Ed Coe has mentioned in his annual notice, we are trying to do the best job possible here at the Coop. If you have been dissatisfied with the materials please let us know. Both Jerry Chandler and I are anxious to improve the performance and the utility of the Coop stocks and to ameliorate the administrative procedures.

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Chromosome 1

101A sr zb4 P-WW
101B sr P-WR
101C sr P-WW
101F sr1 P-RR ts2
101G sr1 zb4 P-WW w4791
101H sr1 zb4 P-WW w8345
101I sr1 zb4 P-WW v032-3
101J sr1 zb4 P-WW w018-3
101K sr1 an A632 6BC
101L o13 sr1 zb4 P-WW
101M sr1 zb4 P-WW v8943
102A sr1 P-WR an gs bm2
102B sr1 P-WR an bm2
102D sr1 P-RR ad bm2
103A sr1 P-RR an bm2
103B sr1 P-RR gs bm2
103C sr1 P-WR bm2
103D vp5
103E zb4 ms17 P-WW
103F zb4 ms17 P-WW rs2
103H zb4 ms17 P-WR v032-3
103I zb4 ms17 P-WW w018-3
103J zb4 ms17 P-WR w8345
103K zb4 ms17 P-WW w4791
104A zb4 ts2 P-WW br f bm2
104B zb4 ts2 P-WW bm2
105A zb4 P-WW
105B zb4 P-WR
105C zb4 P-WW br
105D zb4 P-WW br f bm2
105E ms17
106A zb4 P-WW bm2
106B ts2 P-RR
106C ts2 P-WW bm2
106D ts2 P-WW br bm2
106E ts2 P-WW br f bm2
106H ts2 P-RR w4791
106J ts2 P-RR w018-3
106K ts2 P-RR w8345
106L ts2 P-RR v032-3
106M ts2 P-RR v8943
106N ts2
107A P-CR
107B P-RR
107C P-RW
107D P-CW
107E P-MO
107F P-VV
108A P-RR as br f an gs bm2
108B P-RR br f an gs bm2 rd
108C P-RR br f an gs bm2
108D P-RR br f an gs bm2 id
108E P-RR br f an gs bm2 v8983
108F P-RR br f an gs bm2 v8943
109A P-RR an ad bm2
109B P-RR an gs bm2
109C P-RR br f an ad gs bm2
110A P-WR an1 Kn bm2
110B P-WR an1 Kn
110C P-WR an1 ad1 bm2
110D P-WR an bm2
110E P-WR ad bm2
110F P-WR br Vg
110G P-WR br f gs bm2
110H P-WR br f bm2
110I P-WR br f bm2 id
110J P-WR an br2 bm2
110K P-WR br1
110L P-WR br1 f1 Kn Ts6 bm2
110M P-WR br1 an Kn bm2
110N P-WR br2 bm2
111A P-WW rs2
111B P-WW rs2 br
111C P-WW as br f bm2
111D P-WW hm br f
112A P-WW br f ad bm2
112B P-WW br f bm2
112C P-WW br f an gs bm2
112D P-WW br Vg
112E as
112F as rs2
112H P-WW br1
112I P-WW br1 gs1 bm2
113A as br2
113B rd Hy
113C br f
113D br f bm2 v5588
113E br f Kn
114A br f Kn Ts6
114B br f Kn bm2
114C br bm2
114D Vg
115A Vg an bm2
115B Vg br2 bm2
115C v22
115D bz2m: A A2 C Pr
115I Vg bm2 id

(continued)

116A bz2M: A A2 C R Pr
116B bz2 ad bm2: A C R
116C an bm2
116D an bz2 6923
117A br2
117B br2 bm2
117C br2 an bm2
117D tb-8963
117E Kn
118A Kn Ts6
118B Kn bm2
118C lw
118D Kn Ts6 bm2
119A Adh1-S
119B vp8
119C gs
119D gs bm2
119E Ts6
119F bm2
120A id
120B nec. 2
120C ms9
120D ms12
121A ms14
121B mi
121C D8
121D L1s
122A TB-1LA (1L.20)
122B TB-1Sb (1S.05)
125A Les2
126A bz2 gs1 bm2 ACR

Chromosome 2

201A ws3 lg gl2 B
201B ws3 lg gl2 B sk
201C ws3 lg gl2 B sk v4
201D ws3 lg gl2 B sk f1 v4
201E ws3 lg gl2 B ts
201F ws3 lg gl2 b
202A ws3 lg gl2 b sk v4
202B ws3 lg gl2 b gs2 v4
202C ws3 lg gl2 f1 v4
202D ws3 lg gl2 b sk f1 v4
202E ws3 lg gl2 b v4
203A ws3 lg gl2 B gs2 v4
203B al
203C ws3 lg gl2 b sk
203D al lg
203E al lg gl2 B sk v4
203F al lg gl2 B sk
204A al lg gl2 b sk v4
204B al lg gl2 b sk f1 v4
204C al lg gl2 B ba2
204D al lg gl2 b sk v4 ba2
204E al lg gl2 ba2
204F al lg gl2 B sk
205A al lg gl2 b
205B lg
205C lg gl2
206A lg gl2 B
206B lg gl2 B gs2
206C lg gl2 B gs2 v4
206D lg gl2 B gs2 Ch
206E lg gl2 B gs2 sk Ch
207A lg gl2 B gl11
208A lg gl2 B gs2 sk v4
208B lg gl2 B sk
208C lg gl2 B sk v4
208D lg gl2 B v4
208E lg gl2 b
208F lg gl2 b gs2
208G lg gl2 b gs2 Ch
208H gl2
209A lg gl2 b gs2 sk Ch
209B lg gl2 b gs2 v4
209C lg gl2 b gs2 v4 Ch
209D lg gl2 b gs sh v4 Ch
209E lg gl2 b sk
209F lg gl2 b sk f1
209G lg gl2 b sk f1 v4
210A lg gl2 b sk v4
210D lg gl2 b ts1
211A lg gl2 b f1
211B lg gl2 b gs2 wt
212A lg gl2 b wt v4
212B lg gl2 b f1 v4
212C lg gl2 b f1 v4 Ch
212D lg gl2 b v4
212E lg gl2 b v4 Ch
213A lg gl2 mn v4
213B lg gl2 wt
213C lg gl2 w3
213D lg gl2 w3 Ch
213E lg gl2 b Ch

(continued)

214A lg b gs2 v4
214B lg gl Ch
214C d5
214D B gl11
214E B ts
215A gl14
215B gl11
215C wt
215D mn
215E f1
216F f1 alleles from PI
216A f1 v4 Ch
216B f1 Ht v4
216C f1 Ht v4 Ch
216D f1 w3
216E f1 v4 w3
216F f1 w3 Ch
216G f1 v4 w3 Ch
217A ts
217B v4
217C v4 w3 Ht Ch
217D v4 Ht Ch
217E w3 Ht Ch
218C w3 Ch
218D Ht (soruce A and B)
218E ba2
219A R2: r A A2 C
219B r2: r-g A A2 C
219C Ch
219D Ht Ch
219E w3 ba2
220A Les.
220B 2 2TRIP TRIP2/ ws3 lg gl2
221A gs2
222A TB2L-1sb4464
222B TB2s-3La-2S6270
223A Primary Trisomic 2
224A w4670
224B v5537
224C v7752
224D v8945
224E v8949
224F v8891
224F w 062-3
224G yel 8630
224H gl nec. 8495
224H white pollen (Coe)

Chromosome 3

301A cr
301B cr d
301C cr d Lg3
301E cr ts4 na
301I cr d pm na
302A d=d*-6016 (short)
302B d rt
302C d rt Rg lg2
302D d rt Cg
303A d rt Lg3
303B d Rf lg2
303F g2=v19=pg14
304A d ys3
304B d ys3 Rg
304C d ys3 Rg lg2
304D d yg*-w23
305A d Lg3
305D d1 Rg
305J d Lg3 h
306A d Rg ts4 lg2
306D d Rg ts4
307A d pm
307B d ts4 lg2
308A d ts4 lg2 a-m: A2 CR Dt
308B d ts4
308C d lg2 a-m A2 C R Dt
308D d1 a-m A2 C R Dt
308E ra2
308F ra2 Rg
308G d ts4 a-m: A2 C R Dt
309A ra2 Rg ts4 lg2
309B ra2 ys3 Lg3 Rg
309C ra2 ys3 Rg
309D ra2 Rg lg2
309E ra2 pm lg2
309F ra2 Lg3 ys3
309G ra2 Rg gl6
310a ra2 ts4
310B ra2 ts4 lg2
310C ra2 lg2
310D Cg
311A cl
311B cl : Clm-2
311C cl: Clm-3
311D cl-p: Clm-4
311E rt
311F ys3
311G ys3 Lg3
312C ys3 ts4 lg2

(continued)

312D Lg3
315A pm lg2
315G gl6 a1 A2 C R
316A ts4
316B ts4 na
317A ts4 na pm
317B ts4 ba na
317C ts4 lg2 a-m A2 C R Dt
317D ts4 na a-m et: A2 C R Dt
318A ig
318B ba
318C w7748=y10
319A lg2 A-b et: A2 C R Dt
319B lg2 a-m sh2 et: A2 C R Dt
319C lg2 a-m et: A2 C R dt
319D lg2 a-m et: A2 C R Dt
319E lg2 a-st sh2 et: A2 C R Dt
319F lg2 a-st et: A2 C R Dt
320A lg2
320C na lg2
320D A sh2: A2 C R B Pl dt
321A A-d31: A2 C R
322A A-d31 sh2: A2 C R B PL dt
322B A-d31 sh2: A2 C R Dt
322E a-m: A2 C R B Pl dt
322F a-m: A2 C R b pl dt
323A a-m: A2 C R Dt
323B a-m: A2 C R B PL Dt
323C a-m sh2: A2 C R B PL dt
323D a-m sh2: A2 C R B PL Dt
323E a-m et: A2 CR Dt
324A a-st: A2 C R Dt
324B a-st sh2: A2 C R Dt
324C a-st sh2: A2 C R B PL Dt
324D a-st sh2 et: A2 C R Dt
324E a-st et: A2 C R Dt
324F a-p sh2 et: A2 C R B PL Dt
324G a-st: A2 C R dt
325A a-p et: A2 C R dt
325B a-p et: A2 C R B PL Dt
325C a-x1
325D a-x3
325E a Ga7: A2 C R
325G a3
326A sh2
326B vp
326C Rp on 3
326D te
326E yel 5787
327A TB-3La (31.10)
327B TB-3SB (3S.50)
327C TB-3Lc
327D TB-3Ld
328A tris. 3
330A h
330B d1 h
330C d Lg3 h
330D d1 ys3 h
330E d ts4 lg2 h
330F cr ts4 na h
330G et
331A TB-1La-3L5267
331B TB-1La-3L4759-3
331C TB-1La-3L5242
331D TB-1La-3Le
331E TB-3LF
331F TB-3LG
331G TB-3Lh
331H TB-3Li
331I TB-3Lj
331J TB-3Lk
331K TB-3L1
331L TB-3Lm

Chromosome 4

401A Rp4
401B Ga
401C Ga su
401D Ga-S
401E Ga-S: y
401f Ga-S: A A2 C R
402A st
402B st Ts5
402C st f12
402D Ts5
403A Ts5 f12
403B Ts5 su
403C Ts5 la su gl3
403D Ts5 la su gl3 o1
404A Ts5 su zb6
404B Ts5 su zb6 o
404C Ts5 su gl3 o
405A Ts5 Tu
405B la
405C la su Tu gl3
405D la su gl3
405E la su gl3 c2: A A2 C R
405F ba1 bm3
406A la su gl3 o
406B la su bt2 gl3
406C f12
406D f12 su
407A f12 bt
407B f12 su bm3
407C f12 su Tu gl4
407D su
407E su-am
407F su-am du
408A su bt gl4
408B su bm3
408C su zb6
408H la su bm3
409A su zb6 Tu
410A su zb6 gl3 dp
411A su gl4 j2
411B su gl4 o
414B gl4
414C gl4 o
412A su gl4 o Tu
412B su j2
412C su gl3
412D su gl3 o
413A su o
413B su gl4
414A bt2
414F bm3 gl4
408E bm3
415A j2
415B j2 c2: A A2 C R
415C j2 C2: A A2 C R
416A Tu
416B Tu-1 1st
416C Tu-1 2nd
416D Tu-d
416E Tu-md
416F Tu gl3
417a j2 gl3
417B v8
417C gl3
417D gl3 o
418A gl3 dp
418B c2: A A2 C R
418C C2: A A2 C R
418D C2-Idf (Active-1) : A A2 C R
418E dp
418F o
418G v17
419A v23
419B ra3
419F Dt6 Su gl3
420A Dt4 su: a-m A2 C R
420H Dt4: a-m A2 C R
421A Tb-4Sa (4S.20)
421B TB-1La-4L4692 (9S.40-.83:4L.09)
421C TB7Lb-4L4698 (7L.30-.74: 4L.08)
422A Primary Trisomic 4
423A TB-4Lb
423B TB-4Lc
423C TB-4Ld
423D TB-4Le
423E TB-4Lf

Chromosome 5

501A am a2: A A2 C R
501B lu
501C lu sh4
501D ms13
501E gl17
501F gl17 A2 pr: A C R
501G gl17 a2: A C R
501H gl17 a2 bt: A C R
501K gl17 v3
501L lu a2 bm1 pr A C R
502B A2 vp7 pr: A C R
502C A2 bm bt pr ys: A C R
502D A2 bm pr: A C R
503A A2 bm pr ys: A C R
503C A2 bm pr v2: A C R
503D A2 bt v3 pr: A C R
504A A2 bt pr: A C R
504D A2 bm pr v2: A C R
505A A2 bt pr ys in: A C R
506A A2 v3 pr: A C R
506B A2 pr: A C R
506C A2 pr v2: A C R
506C A2 pr na2: A C R
505B A2 pr ys: A C R
506E A2 pr zb3: A C R
506F A2 pr v12: A C R
507A a2: A C R
507B a2 bm bt bv pr: A C R
508A a2 bm bt pr: A C R
508B a2 bm bt pr ys: A C R
510A a2 bm pr v2: A C R
510B A2 bm pr eg: A C R
510F o9 a2 bm pr v2: A C R
511A a2 bt v3 pr: A C R
511B a2 bt v3 Pr: A C R
511C a2 bt pr: A C R

(continued)

511G o9 a2 bt1 v3 Pr: A C R
512A a2 bt v2: A C R
512B a2 v3 pr: A C R
513A a2 pr: A C R
513B ar2 pr: A C R B Pl
513C a2 pr v2: A C R
515A vp2
515B vp2 gl8
515C vp7
515D bm
516A bm yg Ch
516B bt
516C ms5
516F o9 pr yg1
516G bm1 pr yg1: A C R
517A v3
517B ae
516D td ae
518A sh4
518B gl8
518C na2
518D lw2
518G o9 gl8
519A ys
519B eg
519C v2
519D yg
519E pr yg: A C R
520B v12
520C br3
521A nec3
522A TB-5La
522B TB-5b
522C TB-5Sc
523A Primary Trisomic 5

Chromosome 6

601A rgd po y
601B rdg po Y
601C rdg y
601D rdg Y
601E po = ms6
601F po y pl
601G po y Pl
602A po y wi
602B po y pl
602C y = pb = w-n
602D y rhm
602E po y wi pl
602F po y wi pb4 pl
603A y 110
603B y 111
603C y 112
603D y w15
603E y pb4
604A y pb4 pl
604B y pb4 Pl
604C y si
604F y ms si
604G y wi pb4 Pl
604H y ms1
605A y wi Pl
605B Y Dt2 : a-m A2 C R
605C y pg11 : Wx pg12
605D y pg11 wi : wx pg12
606A Y pg11: Wx pg12
606B y pg11 : wx pg12
606C Y pg11 : wx pg12
606D y pg11 su2 : wx pg12
606E y pl
606F y Pl
607A y Pl Bh : c sh wx A A2 R
607B y pl Bh : c sh wx A A2 R
607C y su2
608A y 110
608B Y 112
609A Y pb4
609B Y wi pl
609C Y wi Pl
609D Y su2
610A wi
610B Pl Dt2 : a-m A2 C R
610C pl sm : P-RR
611A Pl sm : P-RR
611B Pl sm py : P-RR
611C Pl sm Pt py : P-RR
611D Pt
611E w
612A w14
612B ms6
612C 1*-4923
613A 2NOR : a2 bm pr v2
614A TB-6Lb
614B TB-6Sa
614C TB-6Lc
615A Primary Trisomic 6

Chromosome 7

701A Hs o2 v5 ra gl
701E Hs o2 gl
701B In-D
701C IN-D gl
701D o2
702A o2 v5
702B o2 v5 ra gl
702C o2 v5 ra gl sl
702D o2 v5 ra gl Tp
702E o2 v5 ra jl ij
703A o2 v5 gl
703B o2 v5 ra gl Pr
703C o2 v5 gl ms7
703D o2 ra gl
704A o2 ra gl ij
704B o2 ra gl sl
704C o2 v5 gl sl
705A o2 gl
705B o2 gl sl
705C o2 ij
705D o2 bd
706A o2 sl
706B o2 ij bd
707A y8 v5 gl
707B in : A2 pr A C R
707C in gl : A2 pr A C R
707D v5
707E vp9
708A ra
708B ra gl ij bd
708C ra gl ij bd g2
709A gl
709B gl-m
712B ms7 gl TP
710A gl Tp
710B gl mn2
710C gl g2 Tp
710D gl g2
710E gl o5 = pg
711A Tp
711B ij
711C gl sl val
712A ms7
713A Bn
713B bd
713C ms7 ral gl1 ij
714A Pn
714B o5
714C o5 mn2 gl
714D va
715A: Dt3 : a-m A2 C R
715B o2 ra gl Dt3 a-m A C R
716A v*-8647
716B yel*-7748
717A TB-7Lb (7L.30)

Chromosome 8

801A gl18
801B v16
801C v16 j
801D v16 ms8 j
801E v16 ms8 j nec
801F v16 j gl18
802A v16 ms8 j gl18
802B v16 ms8 j yel0245
803A ms8
803B nec
804A v21
805A f13
805B nec v21
806A TB-8La
806B TB-8Lb
807A Primary Trisomic 8

Chromosome 9

901A yg2 C bz Wx: A A2 R
901B yg2 C sh bz: A A2 R
901C yg2 C sh bz wx: A A2 R
901D yg2 C-I sh bz wx: A A2 R
901E yg2 C bz wx: A A2 R
901F yg2 C sh bz wx 12: A A2 R
902A yg2 c sh bx wx: A A2 R
902B yg2 c sh wx: A A2 R
902C yg2 c sh wx gl15: A A2 R
902D yg2 c sh wx gl15 K-S9: A A2 R
902E yg2 c bz wx: A A2 R
924A wd-Ring C-I: A A2 R
903A C sh bz: A A2 R
903B C sh bz wx: A A2 R
903D C-I sh bz wx: A A2 R
904A C sh bz wx gl15 bm4: A A2 R
904B C sh: A A2 R
904C C sh wx: A A2 R
904D C wx ar: A A2 R
904E C sh bz wx bm4: A A2 R
905A C sh wx K-L9: A A2 R
905B C sh ms2: A A2 R
905C C bz Wx: A A2 R
905D C sh wx K-L9: A A2 R K-10

(continued)

905E C sh wx v: A A2 R
906A C Ds wx: A A2 R Pr y
906B C Ds wx: A A2 R pr Y
906C C-I Ds Wx: A A2 R
906D C-I: A A2 R
906E C: A A2 R B Pl
906F C: A A2 R
907A C wx: A A2 R
907B C wx: A A2 R B Pl
907C C wx: A A2 R b Pl
907D C wx: A A2 R B pl
907E C-I wx: A A2 R y
907F C-I wx: A A2 R y B pl
908A C wx ar da: A A2 R
908B C wx v: A A2 R
908C C wx v: A A2 R Pl
908D C wx gl15: A A2 R
908E C wx gl15: A A2 R pr
909A C wx BF: A A2 R
909C c sh bz wx: A A2 R y
909D c sh wx: A A2 R
909E c sh wx v : A A2 R
909F c sh wx gl15: A A2 R
909G c sh wx mj2: A A2 R
910A c sh wx gl15 bk2: A A2 R
910B c sh wx gl15 Bf: A A2 R
910C c sh wx bk2: A A2 R
910D c: A A2 R
910E c sh wx gl 15 16
910F c sh wx gl15 bm4: A A2 R
911A c wx: A A2 R y
911B c wx v: A A2 R
911C c wx gl15: A A2 R
911D c wx Bf: A A2 R
912A sh
912B sh wx v
912C sh wx d3
912D sh wx pg12 gl15: y pg11
912E lo2
913A sh wx
913B sh wx v gl15: A A2 C R
913C sh 17
913D sh 16
914A wx d3
914B wx d3 w11
914C wx d3 v gl15
914D wx d3 gl15
914E Wx pg12: y pg11
914F wx pg12: y pg11
914G Wx pg12: Y pg11
914H wx pg12: Y pg11
915A wx*
915B wx-a
915C w11
915D wx pg12 bm4: y pg11
916A wx v
916B wx v gl15 bk2 Bf bm4
916C wx bk2
916D wx bk2 bm4
917A wx Bf
917B wx Bf bm4
917C v
917D ms2
917E gl15
918A gl15 Bf
918B gl15 m4
918C Wc bk2
918D Wc
918E Wx bk2 bm4
918F Wx Bf
919A bm4
919B bm4 Bf
919C 16
919D 17
919E wx 17
920A yel*-034-16
920B w*-4889
920C w*-8889
920E w*-8950
920F w*-9000
920G Tp9 N9 N3 Df3
921A TB-9La (9L.40)
921B TB-9Sb (9S.40)
921C TB-9Lc v
922A Primary Trisomic 9

* Additional waxy alleles available from collection of O. E. Nelson

Chromosome 10

X01A oy
X01B oy R: A A2 C
X01C oy bf2
X01D oy Og bf2
X01E oy bf2 R: A A2 C
X02A oy ms11
X02B oy bf2 ms11
X02D oy du R: A A2 C
X02E oy du v: A A2 C
X02F oy sr2

(continued)

X02G oy zn
X03A sr3
X03B Og
X03C Og B Pl
X03D Og: A C R
X04A Og du R: A C R
X04B ms11
X04C ms11 bf2
X04D bf2
X05A bf2 zn
X05B bf2 li g r: A A2 C
X05C bf2 g R sr2: A A2 C
X05D bf2 g r sr2: A A2 C
X05E bf2 sr2
X05F bf2 ms10
X06A bf2 r sr: A A2 C
X06B nl zn g R: A A2 C
X06C nl g R: A A2 C
X06D li bf2 g R-K10
X07A nl g r: A A2 C
X07B nl g R sr2: A A2 C
X07C Y9
X08A y9 v18
X08B li nl g R zn
X07D nl
X09A li zn g r: A A2 C
X09B li g R: A A2 C
X09C li g r d: A A2 C
X09D li g v18: A A2 C
X09F ms10
X10A du
X10B du sr2
X10C du o7
X10D du g r: A C R
X10E du sr2
X10F zn
X11A zn g
X11B zn g R sr2: A A2 C
X11C zn g r: A A2 C
X11D Tp2 g r: A A2 C
X11E g R sr2: A A2 C
X11F g r: A A2 C
X12A g r sr2:
X12B g r sr2 l: A A2 C
X12C g R-g sr2: A A2 C
X12D g R-g sr2 v18: A A2 C
X13A g R-g K10: A A2 C
X13B g R-g sr2: A A2 C
X13D g r-r sr2: A A2 C
X14A Ej r-r: A A2 C
X15A Ej r-r sr2: A A2 C
X15B r sr2 l
X15C R-g: A A2 C
X16A r-g sr2: A A2 C
X16B r K10: A A2 C
X17A r-g: A A2 C
X15D r-ch Pl: A a2 C
X17B r-r: A A2 C
X17C R-nb: A A2 C
X17D R-nj: A A2 C
X17E R-r: A A2 C
X16C R-ch B Pl: A A2 C
X18A R-lsk: A A2 C
X18B R-sk-mc.2: A A2 C
X18C R-st: A A2 C
X18D R-sk: A A2 C
X18E R-st M st
X18F R-st M st o7
X18G R-scm2: bz2 A A2 C C2
X25A R-scm2: a-st A2 C C2
X25B R-scm2: c2 A A2 C
X25C R-scm122: pr A A2 C C2
X25D R-scm2: a2 A C C2
X25E R-scm2: c A A2 C2
X19a Lc
X19B w2
X19C w2 l
X19D o7
X20A o7: o2
X20B l
X20C v18
X20D mst
X20E l yel*-5344
X20F yel*-8721
X20G yel*-8454
X20H yel*-8793
X24A cm
X21A TB-10La (10L.35)
X22A TB-10Sc
X21B TB-10L19
X23A Primary Trisomic 10

Unplaced Genes

U235A dv
U235B dy
U335A el
U435A l4
U635A Rs
U533A v13
U935A ws ws2
UX35A zb
UX35B zb2
U934B zn2
U734A nec*-8376
U933A o9
U933B o10
U933C o11
U933D o13

Multiple Gene Stocks

M141A A A2 C C2 R-g Pr B Pl
M141B A A2 C C2 R-g Pr B pl
M141C A A2 C C2 R-g b Pl
M241A A A2 C C2 r-g Pr B Pl
MX17A A A2 C C2 r-g Pr b pl
M241B A A2 C C2 r-g Pr B pl
M341A A A2 c C2 R-g Pr B pl
M241C A A2 C C2 R-r Pr B Pl
M341B A A2 C C2 R-r Pr B pl
M341C A A2 C C2 R-r Pr b Pl
M441A A A2 C C2 R-r Pr B Pl wx
M441B A A2 C C2 R-r Pr B pl wx
M441C A A2 C C2 R Pr
M641A A A2 C C2 R Pr wx
MX41A A A2 C C2 R pr y wx gl
M941A A A2 c C2 R Pr y wx
M741A A A2 C C2 r Pr Y wx
M341D A A2 c C2 R-r Pr B Pl
M441D A A2 C C2 r-r Pr B Pl
M441E A A2 c C2 r-r Pr B Pl
MX41B su pr y gl wx : A A2 C C2 R
M841A A su pr : A2 C C2 R
MX41C bz2 a c2 a2 pr Y/y c bz wx r
M841B a su A2 C C2 R
MX40A bm2 lg a su pr y gl j wx g
M841C colored scutellum
MX41D a su pr y gl wx A A2 C C2 R
MX40B ts2 : sk
MX40C lg gl2 wt : a-m A2 C C2 R Dt
M741B A A2 C C2 R-nj : purple embryo
S. Chase
M741C Stock 6 : Hi-haploid R-r B Pl

Popcorns

P142A Amber Pearl
P142B Argentine
P142C Black Beauty
P242A Hulless
P242B Ladyfinger
P242C Ohio Yellow
P342A Red South American
P342B Strawberry
P342C Supergold
P442A Tom Thumb
P442B White Rice

Exotics and Varieties

E542A Black Mexican Sweet Corn
(with B-chromosomes)
E542B Black Mexican Sweet Corn
(without B-chromosomes)
E642A Knobless Tama Flint
E442A Gaspe Flint
E642B Gourdseed
E742A Maiz Chapalote
E742B Papago Flour Corn
E742C Parker's Flint
E842A Tama Flint
E842B Zapalote Chico

Tetraploid Stocks

N103A P-RR
N103B P-VV
N103C a A2 C R Dt
N104A su
N104B pr : A A2 C R
N105A y
N106A gl
N106B Y sh wx
N106C wx
N107A g A A2 C R
N102A A A2 C R Pr B Pl

Cytoplasmic traits

C738A NCS2
C738B NCS3

Cytoplasmic steriles and Restorers

C836A WP9-(T) rf rf2
C836B WP9 rf rf2
C736A R213 Rf rf2
C736B Ky21 Rf Rf2

Waxy Reciprocal Translocations

WX01A wx 1-9c (1S.48: 9L.22) * Sx
WX02A wx 1-94995 (1L.19: 9S.20) * Sx
WX03A wx 1-98389 (1L.74: 9L.13) * Sx
WX05A wx 2-9b (2S.18: 9L.22) * Sx
WX08A wx 3-9c (3L.09: 9L.12) * Sx
WX13A wx 4-9b (4L.90: 9L.20) * Sx
WX12A wx 4-95657 (4L.33: 9S.25) * Sx
WX11A wx 4-9g (4S.27: 9L.27) * Sx
WX17A wx 5-9a (5L.69: 9S.17) * Sx
WX14A wx 5-9c (5S.07: 9L.10) * Sx
WX19A wx 6-9a (6S.79: 9L.40) * Sx
WX20A wx y 6-9b (6L.10: 9S.37) * Sx
WX23A wx 7-9a (7L.63: 9S.07) * Sx
WX22A wx 7-94363 (7 cent: 9 cent) * Sx
WX24A wx 8-9d (8L.09: 9L.16) * Sx
WX25A wx 8-96673 (8L.35: 9L.31) * Sx
WX27A wx 9-10b (9S.13: 10S.40) * Sx

Non-waxy Reciprocal Translocations

WX30A Wx 1-9c (1S.48: 9L.24) * Sx
WX30B Wx 1-94995 (1L.19: 9S.20) * Sx
WX30C Wx 1-98389 (1L.74: 9L.13) * Sx
WX31A Wx 2-9c (2L.49: 9S.33) W23 only
WX31B Wx 2-9b (2S.18: 9L.22) * Sx
WX32A Wx 3-98447 (3S.44: 9L.14) *
WX32B Wx 3-98562 (3L.65: 9L.22) * Sx
WX33A Wx 4-9e (4S.53: 9L.26) * Sx
WX33B Wx 4-95657 (4L.33: 9S.25) * Sx
WX34A Wx 5-9c (5S.07: 9L.10) * Sx
WX34B Wx 5-94817 (5L.69: 9S.17) M14 only
WX35A Wx 5-98386 (5L.87: 9S.13) * Sx
WX36A Wx 6-94778 (6S.80: 9L.30) * Sx
WX37A Wx 6-98768 (6L.89: 9S.61) * Sx
WX37B Wx 7-94363 (7 cent: 9 cent) *
WX38A Wx 7-9a (7L.63: 9S.07) * Sx
WX38B Wx 8-9d (8L.09: 9L.16) * Sx
WX38C Wx 8-96673 (8L.35: 9S.31) * Sx
WX39A Wx 9-108630 (9S.28: 10L.27) M14 only
WX39B Wx 9-10b (9S.13: 10S.40) * Sx

* = Homozygotes available in both
M14 & W23 backgrounds

Sx = Single cross of homozygotes between
M14 & W23 versions available

Inversions

I143A Inv.1a (1S.30-L.50)
I143B Inv.1c (1S.35-L.01)
I143C Inv.1d (1L.55-L.92)
I143D Inv.1L-5131-10 (1L.46-L.82)
I444A Inv.2a (2S.70-L.80)
I243A Inv.2S-L8865 (2S.06-L.05)
I243B Inv.2L-5392-4 (2L.13-L.51)
I343A Inv.3a (3L.38-L.95)
I343B Inv.3L (3L.19-L.72)
I343C Inv.3L-3716 (3L.09-L.81)
I443A Inv.4b (4L.40-L.96)
I443B Inv.4c (4S.86-L.62)
I543A Inv.4e (4L.16-L.81)
I743A Inv.5-8623 (5S.67-L.69)
I743B Inv.6-8452 (6S.77-L.33)
I843A Inv.6-8604 (6S.85-L.32)
I743C Inv.6-3712 (6S.76-L.63)
I943A Inv.7L-5803 (7L.17-L.61)
I943B Inv.7-8540 (7L.12-L.92)
I943C Inv.7-3717 (7S.32-L.30)
IX43A Inv.8a (8S.38-S.15)
I344A Inv.9a (9S.70-L.90)
IX43B Inv.9b (9S.05-L.87)
IX43C Inv.9c (9S.10-L.67)

STOCK NR	GENOTYPE
102A	srl P-WR an gs bm2
102D	srl P-RR ad bm2
105A	zb4 P-WW
105C	zb4 P-WW br
107A	P-CR
107B	P-RR
107C	P-RW
107E	P-MO
107F	P-VV
108C	P-RR br f an gs bm2
109A	P-RR an ad bm2
110C	P-WR an1 ad1 bm2
110E	P-WR ad bm2
111A	P-WW rs2
113B	rd Hy
114C	br bm2
115C	v22
117A	br2
117B	br2 bm2
202E	ws3 lg gl2 b v4
205B	lg
205C	lg gl2
208H	gl2
209B	lg gl2 b gs2 v4
211A	lg gl2 b f1
212A	lg gl2 b wt v4
212D	lg gl2 b v4
215D	mn
215E	f1
217B	v4
301B	cr d
302A	d=d*-6016 (short)
304A	d ys3
307B	d ts4 lg2
308B	d ts4
310B	ra2 ts4 lg2
310C	ra2 lg2
311E	rt
311F	ys3
316A	ts4
320A	lg2
326A	sh2
330A	h
401A	Rp4
402C	st fl2
402D	Ts5
405D	la su gl3
406C	fl2
407B	fl2 su bm3
407D	su
407E	su-am
408A	su bt gl4
408B	su bm3
411B	su gl4 o
414B	gl4
414C	gl4 o
412C	su gl3
413A	su o
414A	bt2
415A	j2
417B	v8
417C	gl3
418E	dp
418F	o
418G	v17
501B	lu
506B	A2 pr: A C R
507A	a2: A C R
510A	a2 bm pr v2: A C R
511B	a2 bt v3 Pr: A C R
511C	a2 bt pr: A C R
515D	bm
516B	bt
517A	v3
518B	gl8
519E	pr yg: A C R
520B	v12
520C	br3
602D	y rhm
604A	y pb4 pl
604B	y pb4 Pl
605C	y pg11 : Wx pg12
606B	y pg11 : wx pg12
606C	Y pg11 : wx pg12
606F	y Pl
607A	y Pl Bh : c sh wx A A2 R
607B	y pl Bh : c sh wx A A2 R
607C	y su2
609A	Y pb4
609B	Y wi pl
609D	Y su2
610C	pl sm : P-RR
613A	2NOR : a2 bm pr v2
701D	o2
702A	o2 v5
703A	o2 v5 gl
705B	o2 gl sl

STOCK NR	GENOTYPE
705C	o2 ij
707A	y8 v5 gl
707D	v5
709A	gl
710D	gl g2
711B	ij
714B	o5
801A	gl18
801B	v16
801F	v16 j gl18
804A	v21
805A	fl3
901C	yg2 C sh bz wx: A A2 R
901E	yg2 C bz wx: A A2 R
902A	yg2 c sh bx wx: A A2 R
902B	yg2 c sh wx: A A2 R
902C	yg2 c sh wx gl15: A A2 R
903A	C sh bz: A A2 R
903B	C sh bz wx: A A2 R
903D	C-I sh bz wx: A A2 R
904B	C sh: A A2 R
904D	C wx ar: A A2 R
904E	C sh bz wx bm4: A A2 R
905D	C sh wx K-L9: A A2 R K-10
905E	C sh wx v: A A2 R
907A	C wx: A A2 R
907D	C wx: A A2 R B pl
907E	C-I wx: A A2 R y
908B	C wx v: A A2 R
908D	C wx gl15: A A2 R
909A	C wx BF: A A2 R
909C	c sh bz wx: A A2 R y
909D	c sh wx: A A2 R
909E	c sh wx v : A A2 R
909F	c sh wx gl15: A A2 R
910C	c sh wx bk2: A A2 R
910D	c: A A2 R
911A	c wx: A A2 R y
911B	c wx v: A A2 R
911C	c wx gl15: A A2 R
911D	c wx Bf: A A2 R
912A	sh
912B	sh wx v
913A	sh wx
915A	wx*
915B	wx-a
915D	wx pg12 bm4: y pg11
916A	wx v
916C	wx bk2
917A	wx Bf
917C	v
918A	gl15 Bf
918B	gl15 bm4
918E	Wx bk2 bm4
918F	Wx Bf
919A	bm4
X01B	oy R: A A2 C
X01C	oy bf2
X01E	oy bf2 R: A A2 C
X04D	bf2
X05E	bf2 sr2
X06C	nl g R: A A2 C
X07A	nl g r: A A2 C
X07D	nl
X09B	li g R: A A2 C
X10A	du
X10F	zn
X11A	zn g
X11C	zn g r: A A2 C
X12C	g R-g sr2: A A2 C
X16A	r-g sr2: A A2 C
X17D	R-nj: A A2 C
X18A	R-lsk: A A2 C
X19D	o7
X20A	o7: o2
X20C	v18

* Additional waxy alleles available from collection of O. E. Nelson

V. MAPPING 1986

The current mapping coordinators for each arm are as follows (* indicates the lead coordinator):

1S *Fletcher, Patterson	4S Galinat	7S Steffensen	10S Albertsen
1L *Sisco, Goodman	4L *McCormick, Beckett	7L Whalen	10L *Kermicle, Patterson
2S Tracy	5S *Polacco, P. Crane	8S Neuffer	B chrom. W. Carlson
2L Patterson	5L *D. Weber, Shadley	8L Neuffer	Gene list Hoisington
3S Poethig	6S *Phillips, Mascia	9S O. Nelson	
3L *Sheridan, Beckett	6L *Phillips, Mascia	9L Coe	

CHROMOSOME 1, SHORT ARM

No report.

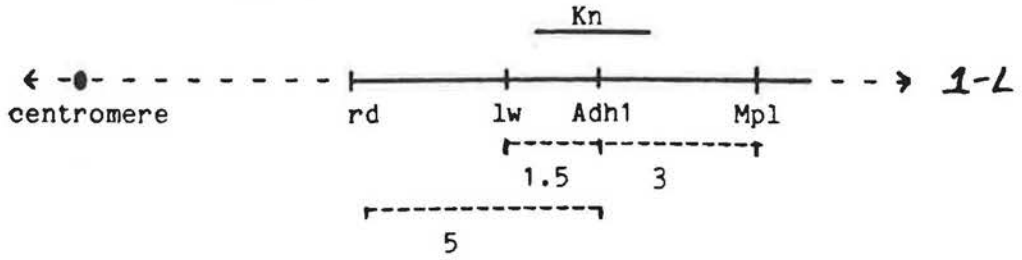
(Please see in this issue hcf3, hcf6, cfr*-2018, cp*-991, cp*-1078B, de*-1142, cp*-1308A, Les2, Les5, lls1).

CHROMOSOME 1, LONG ARM

This summer I should complete construction of a multiple marker stock for 1L: f bz2 gs bm2. This is being converted to A632 and Mo17 inbred backgrounds. I am not using br as a marker for the multiple tester, since I plan to add br2 to cover the most proximal part of 1L. Although br2 has not been mapped precisely (I am working on it), Lambert's data (MNL 37:41, 1963) placed it in the poorly marked proximal region of the arm.

Data of Wendel et al. (this issue) place Dia2 at 155 on 1L and add a new marker, Acp4, in the most distal region of the arm. A four-point test cross of gs, Phi1, bm2, and Acp4 will be scored by Wendel and me this summer. I am mapping Neuffer's EMS-induced mutants wl*-266A, wl*-709B, dek2, and dek22 relative to bz2, wx T1-9(4995), and wx T1-9(8389).

Hoisington continues to work on the location of Les*-1461, which he found was linked to wx T1-9(8389) (MNL 58:82, 1984). Freeling continues to work intensively on the markers close to Adh1. His results on two-point testcrosses are as follows (personal communication): lw - 1.5 - Adh1; lw - 4 - Mpl; Adh1 - 3 - Mpl; rd - 5 - Adh1; Kn - Adh1 is 0.1 to 1.0, depending on the allele of Kn. My tentative map based on these results:



(Please also see in this issue hcf50, dsc*-1315A, Phi1).

P. H. Sisco

CHROMOSOME 2, SHORT ARM

No information to report.

(Please also see in this issue nec4, Les1, Les*-2004).

W. F. Tracy

CHROMOSOME 2, LONG ARM

No report.

(Please see in this issue Les4, Les*-1378, Les7).

CHROMOSOME 3, SHORT ARM

Reports in this Newsletter indicate that ra2 is located 6cM proximal to d, while Rf1 is located 3cM distal to Lg3. Whalen (this issue) reports that g2 is 32 map units distal to d1, and ra2 is proximal to d1, thus extending the known genetic length of chromosome 3 by 14 map units. Whalen and Kahler (this issue) place Est4 15 units to the right of d1 and 11 units to the left of g16. The order is therefore g2 - cr1 - d1 - ra2 - Est4.

(Please also see in this issue sh*-1339A, dek24).

S. Poethig

CHROMOSOME 3, LONG ARM

No report.

(Please see in this issue Tpi4, sh*-627D, TB-3Lc breakpoint).

CHROMOSOME 4, SHORT ARM

No report.

(Please see in this issue Bx1, sh*-912, dek25).

CHROMOSOME 4, LONG ARM

No report.

(Please see in this issue ptd*-1130, TB-4Lc and TB-4Lf breakpoints).

CHROMOSOME 5, SHORT ARM

There has been progress in the search for new loci on this arm. In the summer of 1984 I self-fertilized several individuals with the genotype: a2-m5/a2 wx-m8/wx(?), Spm taken from an ear segregating Spm. Summer 1985 I grew 183 individuals representing seven progenies of the previous summer's planting. About one-half of the kernels planted showed developmentally early Spm activity with respect to a2-m5; one-half had very late or no observable activity. I did not observe any new traits segregating in this material, which I have self-fertilized and am now screening for new traits. I have noted one ear with several anthers scattered evenly throughout it. Summer 1986 I will use B-A translocations to ascertain whether any new phenotypes involve the 5S chromosome arm. I will also screen for segregation of new phenotypes in mature plants derived from the summer 1985 material.

(Please also see in this issue cp*-931A on TB-5Sc breakpoint).

Mary Polacco

CHROMOSOME 5, LONG ARM

No report.

(Please see in this issue hcf38, cp*-863A, de*-1196, cp*-1275A, fl*-1299, dek26, dek27).

CHROMOSOME 6, SHORT ARM

No report.

(Please see in this issue Adk1, dek28).

CHROMOSOME 6, LONG ARM

No report.

(Please see in this issue de*-1296A, Mdh2, 110, 112, TB-6Lb breakpoint).

CHROMOSOME 7, SHORT ARM

No report.

CHROMOSOME 7, LONG ARM

Hoisington reports in this Newsletter that Les9 (formerly Les*-2008) is very near ra1 on chromosome 7L but the order is uncertain. Lin reports (this issue) that the breakpoint of TB-7Lb is at 0.34 on the long arm of 7.

(Please also see in this issue o*-1241).

R. H. Whalen

CHROMOSOME 8, SHORT AND LONG ARMS

Please see in this issue o6 and pro1 (allelism), dek29, ats, ms43, Rf4, Tpi3, Mdh1, Idh1, Goss's wilt, and the TB-8Lc breakpoint. Data to be presented later: rgh*-1825 - 30.2±4.0 - 11, not linked to pro1 or v16; nec1 - 23.8±4.0 - Bif; cp*-1387A - 23.3±4.2 - Bif; crp*-1429A - 37.2±4.3 - Bif; pro1 - 36.1±6.2 - v16; pro1 - 20.5±4.4 - C1t.

M. G. Neuffer and D. England

CHROMOSOME 9, SHORT ARM

No report.

(Please see in this issue lo2, cp*-1054, Les8, Df9S, TB-9Sd breakpoint).

CHROMOSOME 9, LONG ARM

Please see in this issue hcf42, o*-744, dek30, Ss2, TB-9Lc breakpoint. Mapping crosses and testcrosses are in process for most of the factors tabulated in MNL 59:40.

E. H. Coe, Jr.

CHROMOSOME 10, SHORT ARM

No report.

(Please see in this issue Les6, Les*-1453, Les*-2016, TB-10Sc breakpoint).

CHROMOSOME 10, LONG ARM

No report.

(Please see in this issue msc*-1330, Glu1, Sad1, and TB-10L19, TB-10L30, TB-10L32 breakpoints).

B CHROMOSOMES

No new information to report.

W. Carlson

Ed. note: The RFP map of Helentjaris et al. in this issue, which includes a number of morphological markers and specific-gene clones as well, adds to the developing maps in progress.

(This space is saved for future expansion of linkage data, compilations, and cooperators' summaries)

MAIZE WORKING MAP

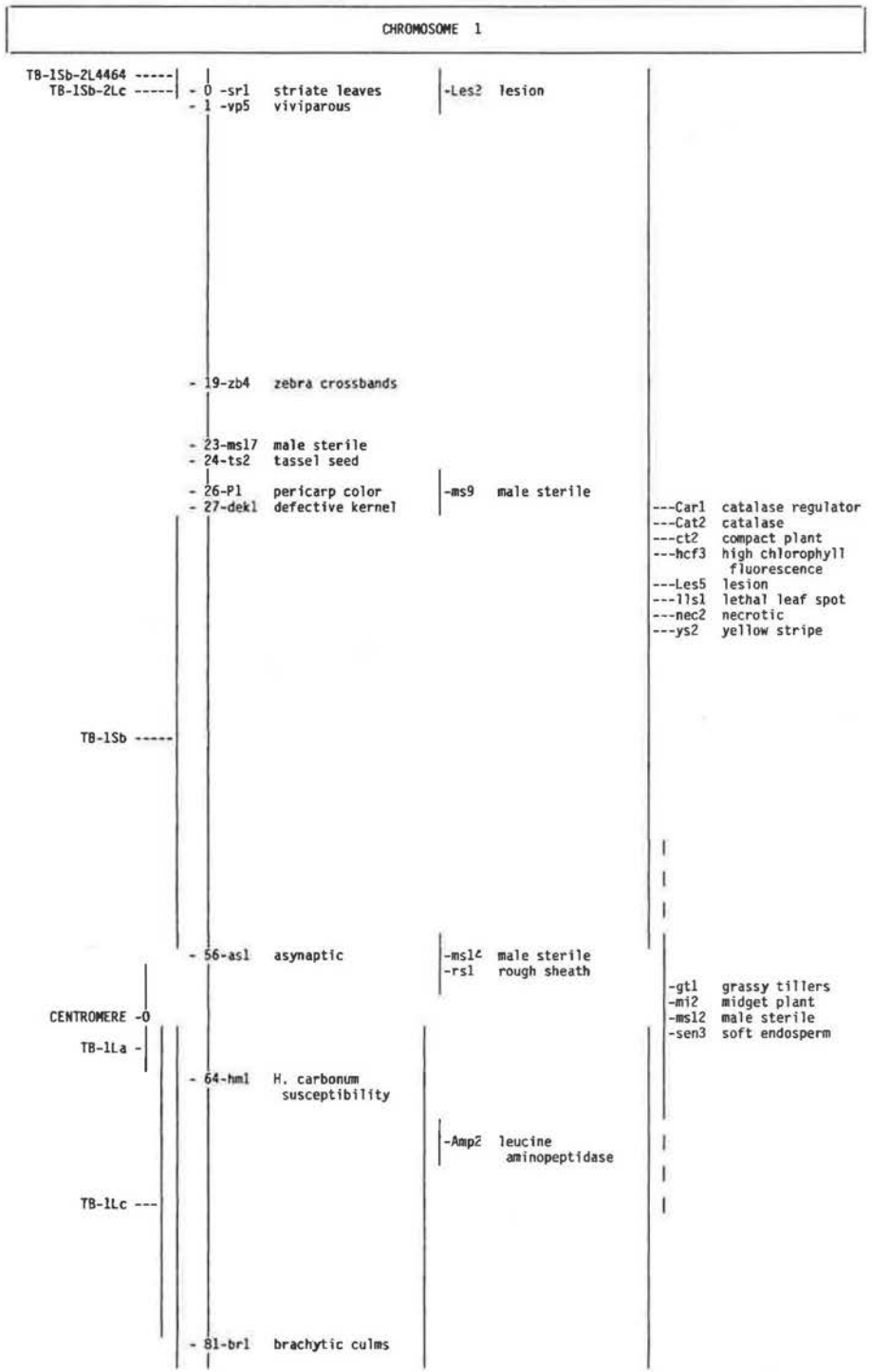
Immediately following is the newly revised and updated working linkage map. The same format as last year was followed and I refer you to last year's newsletter for an explanation of how the map is constructed (MNL59:110). Again, I appreciate and invite any and all comments and corrections regarding the map. Every chromosome had at least one change with chromosome 3 being the winner with almost a complete revision. This year, all genes from the linkage map are referenced in the symbol index at the end of the newsletter as an aid to finding particular genes in the map.

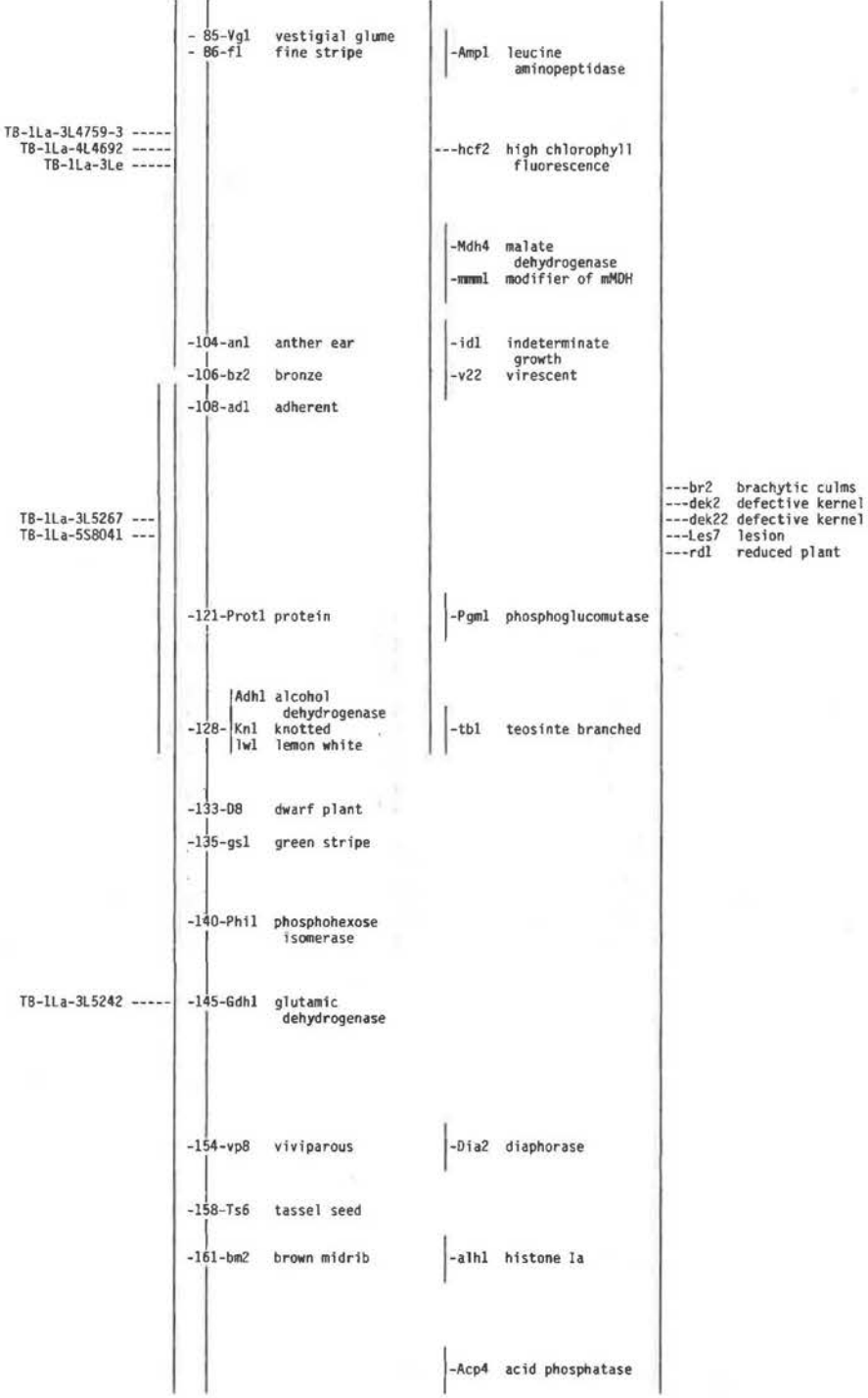
Only a few additions to the B-A translocation data tables (MNL59:159-167) were available this year, so I will just list them so that you may add the changes to last year's tables:

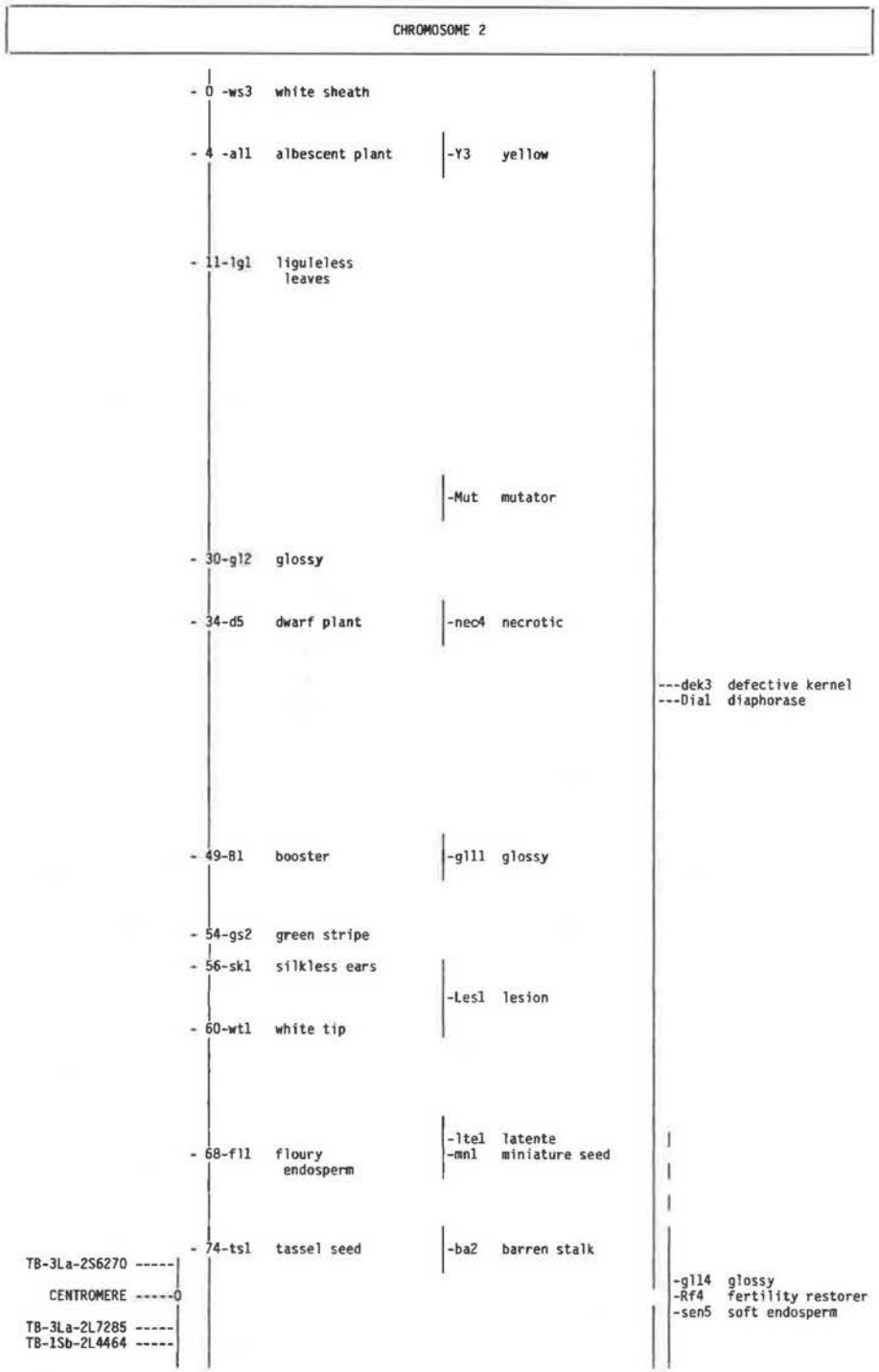
Chromosome 2S&L	TB-3La-2S6270 uncovers tsl	Beckett, 1985, personal communication
Chromosome 3L	TB-3La, 3Lf, 3Lg does not uncover Tpi4	Wendel & Beckett, MNL60:112, 1986
Chromosome 5S	TB-5Sc uncovers dek18	Chang, 1985, personal communication
Chromosome 8S&L	TB-8La uncovers ms43	Golubovskaya & Distanova, MNL60:106, 1986

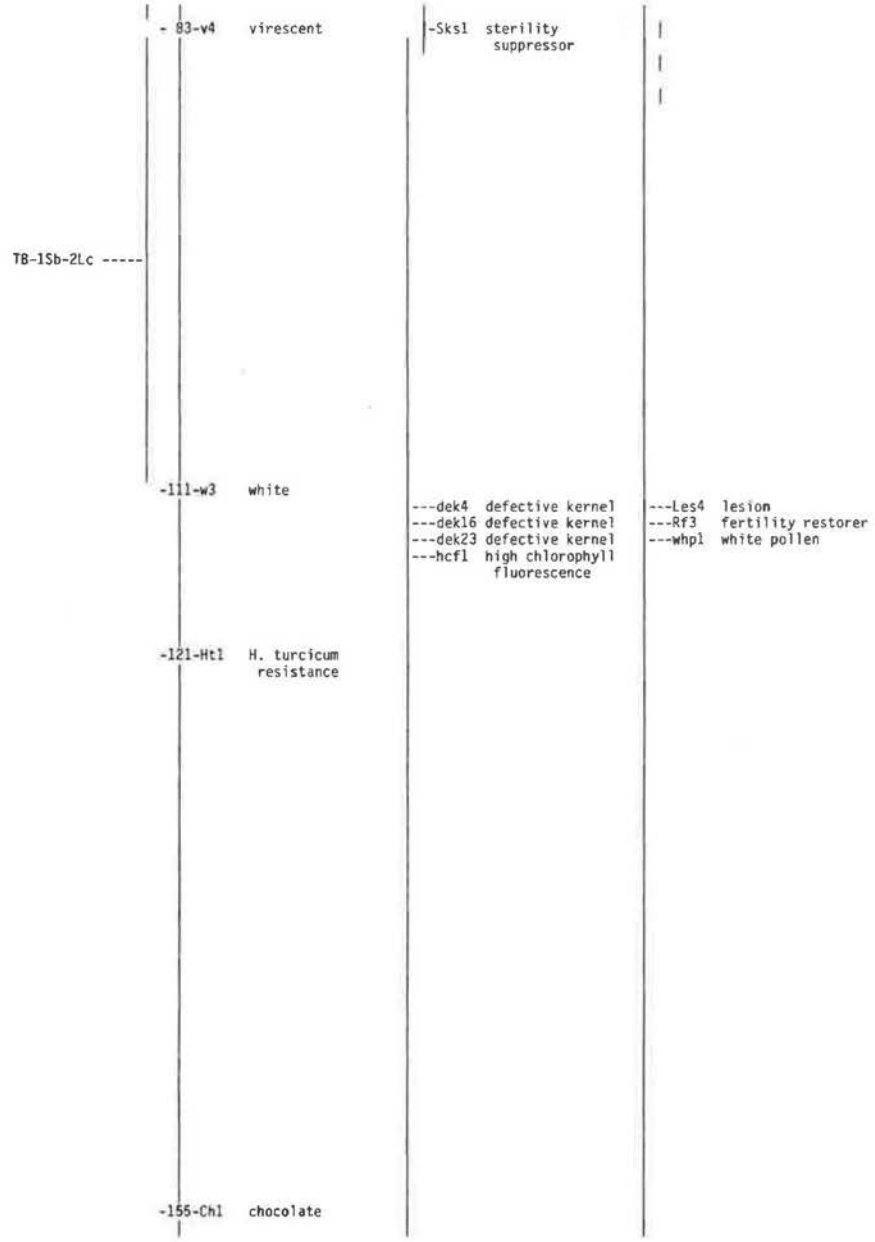
Dave Hoisington

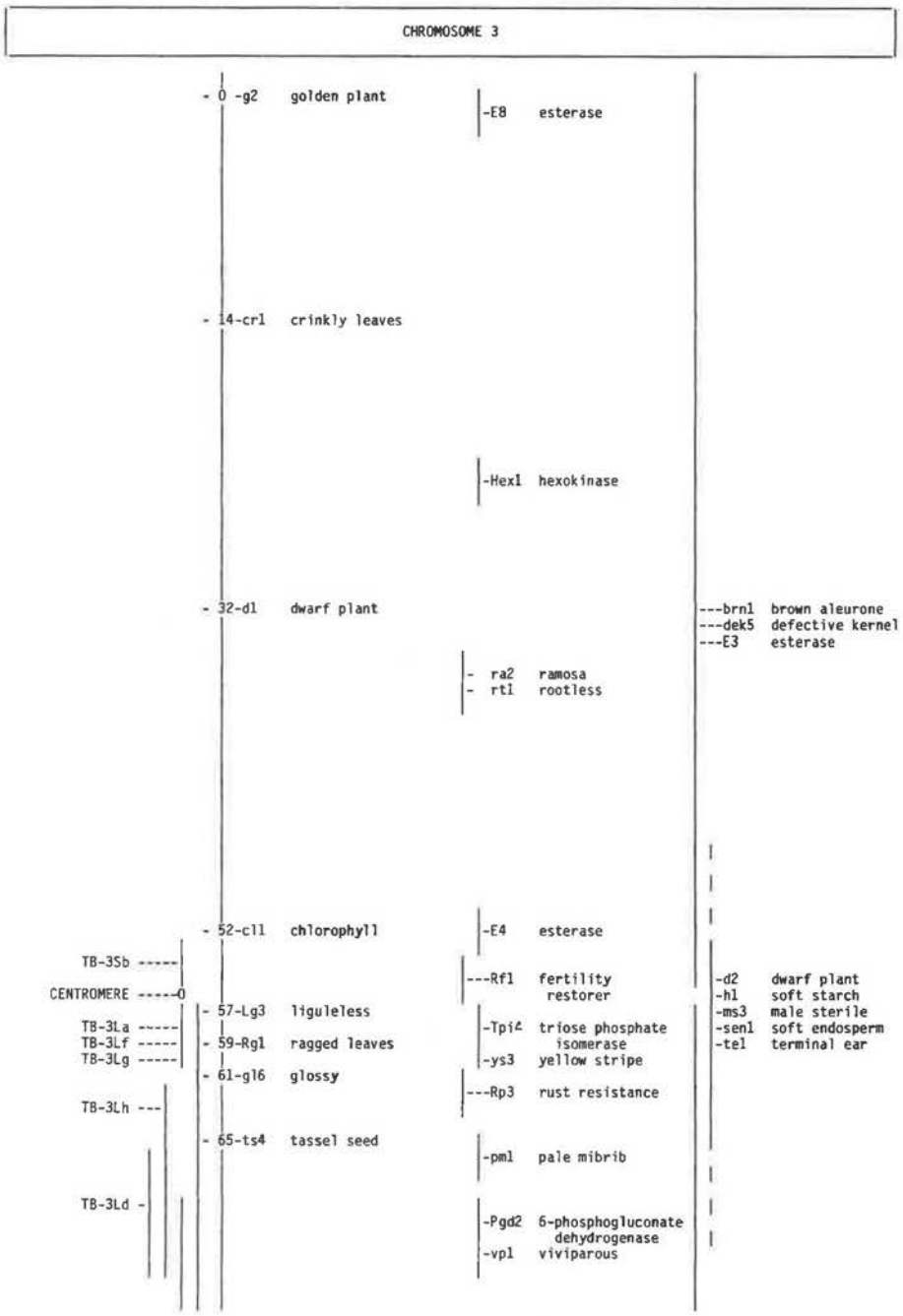
(This space is saved for future expansion of chromosome location data)

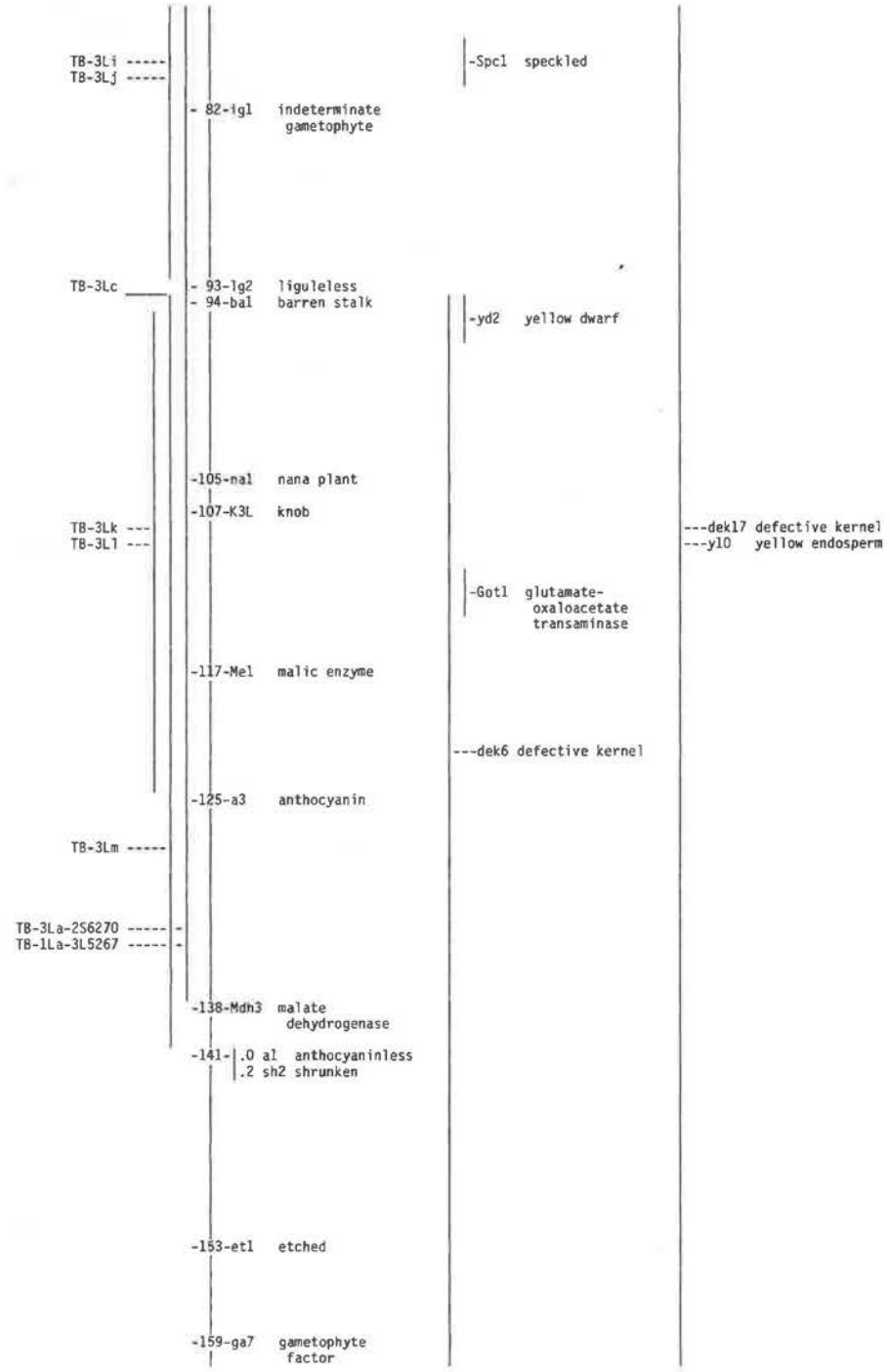

CHROMOSOME 1
TB-1Sb-2L4464 -----
TB-1Sb-2Lc -----
- 0 -srl striate leaves
- 1 -vp5 viviparous
-Les2 lesion
- 19-zb4 zebra crossbands
- 23-ms17 male sterile
- 24-ts2 tassel seed
- 26-P1 pericarp color
- 27-dek1 defective kernel
-ms9 male sterile
---Car1 catalase regulator
---Cat2 catalase
---ct2 compact plant
---hcf3 high chlorophyll fluorescence
---Les5 lesion
---lls1 lethal leaf spot
---nec2 necrotic
---ys2 yellow stripe
TB-1Sb -----
- 56-as1 asynaptic
-ms1c male sterile
-rs1 rough sheath
-gt1 grassy tillers
-mi2 midget plant
-ms12 male sterile
-sen3 soft endosperm
CENTROMERE -0
TB-1La -
- 64-hm1 H. carbonum susceptibility
-Amp2 leucine aminopeptidase
TB-1Lc ---
- 81-br1 brachytic culms

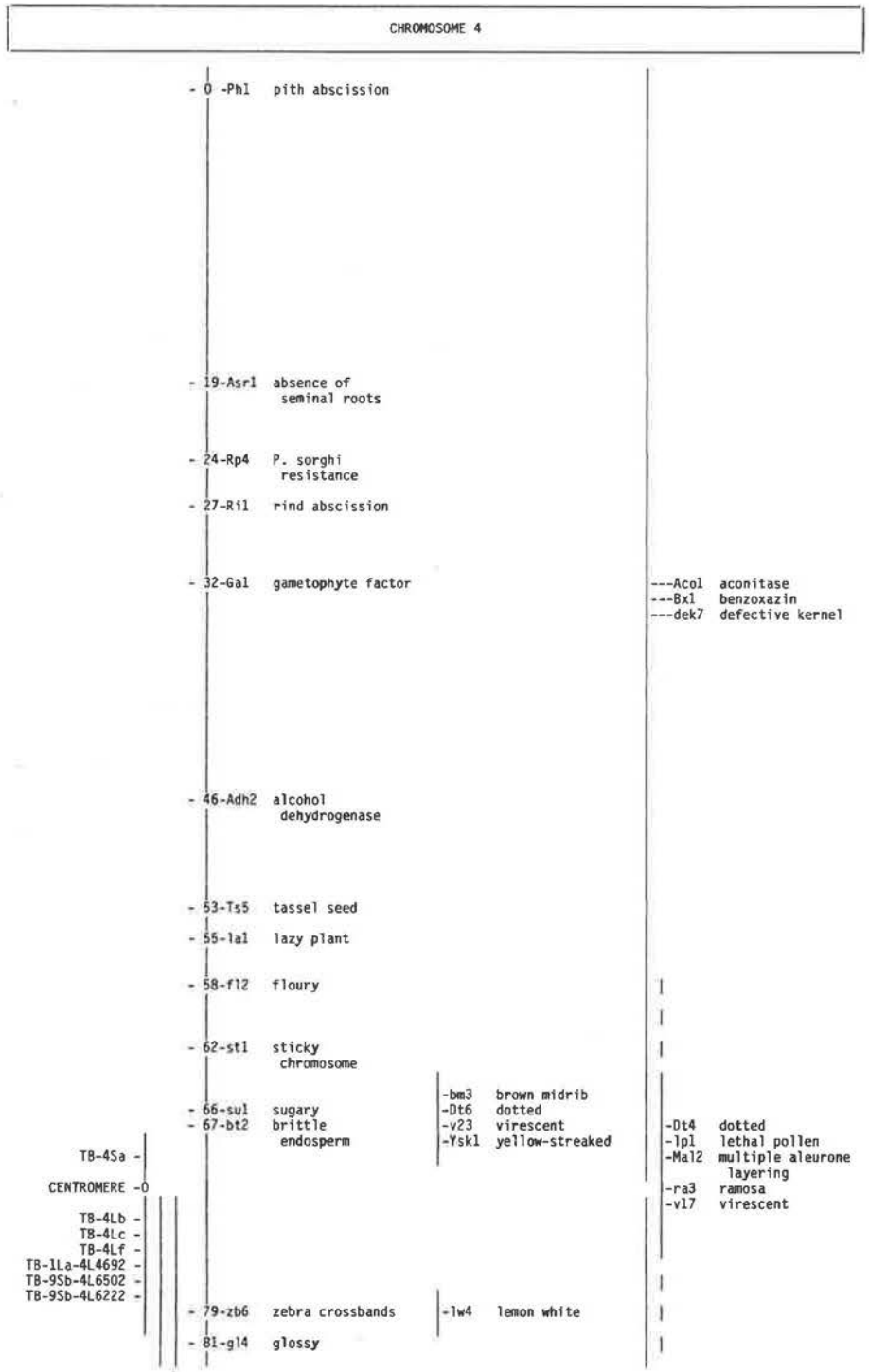

- 85-Vg1 vestigial glume
- 86-fl fine stripe
-Amp1 leucine aminopeptidase
T8-1La-3L4759-3
T8-1La-4L4692
T8-1La-3Le
---hcf2 high chlorophyll fluorescence
-Mdh4 malate dehydrogenase
-mmm1 modifier of mMDH
-104-an1 anther ear
-idl indeterminate growth
-106-bz2 bronze
-v22 virescent
-108-ad1 adherent
---br2 brachytic culms
---dek2 defective kernel
---dek22 defective kernel
---Les7 lesion
---rdl reduced plant
T8-1La-3L5267
T8-1La-5S8041
-121-Prot1 protein
-Pgm1 phosphoglucomutase
Adh1 alcohol dehydrogenase
-128- Kn1 knotted
lw1 lemon white
-tb1 teosinte branched
-133-D8 dwarf plant
-135-gs1 green stripe
-140-Phi1 phosphohexose isomerase
T8-1La-3L5242
-145-Gdh1 glutamic dehydrogenase
-154-vp8 viviparous
-Dia2 diaphorase
-158-Ts6 tassel seed
-161-bm2 brown midrib
-alh1 histone Ia
-Acp4 acid phosphatase

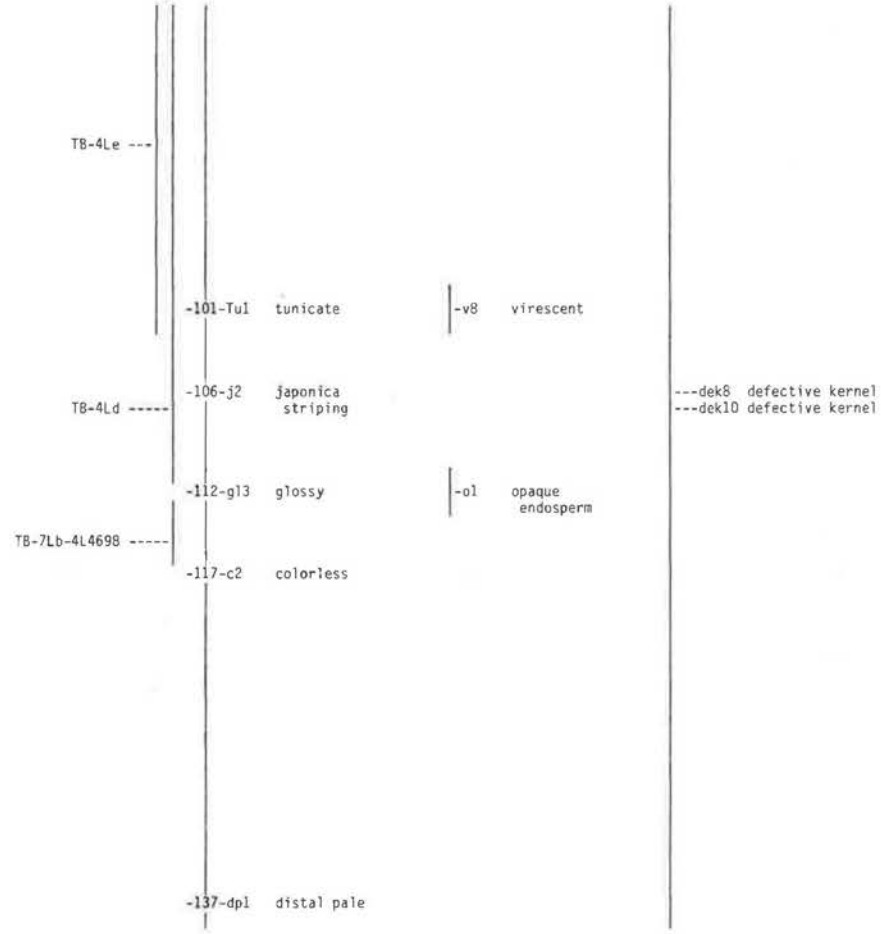

CHROMOSOME 2
- 0 -ws3 white sheath
- 4 -al1 albescent plant
-Y3 yellow
- 11-lgl liguleless leaves
-Mut mutator
- 30-gl2 glossy
- 34-d5 dwarf plant
-nec4 necrotic
---dek3 defective kernel
---Dial diaphorase
- 49-Bl booster
-gl11 glossy
- 54-gs2 green stripe
- 56-sk1 silkless ears
-Les1 lesion
- 60-wt1 white tip
-lte1 latente
- 68-fl1 floury endosperm
-mn1 miniature seed
- 74-ts1 tassel seed
-ba2 barren stalk
TB-3La-2S6270
CENTROMERE
TB-3La-2L7285
TB-1Sb-2L4464
-gl14 glossy
-Rf4 fertility restorer
-sen5 soft endosperm

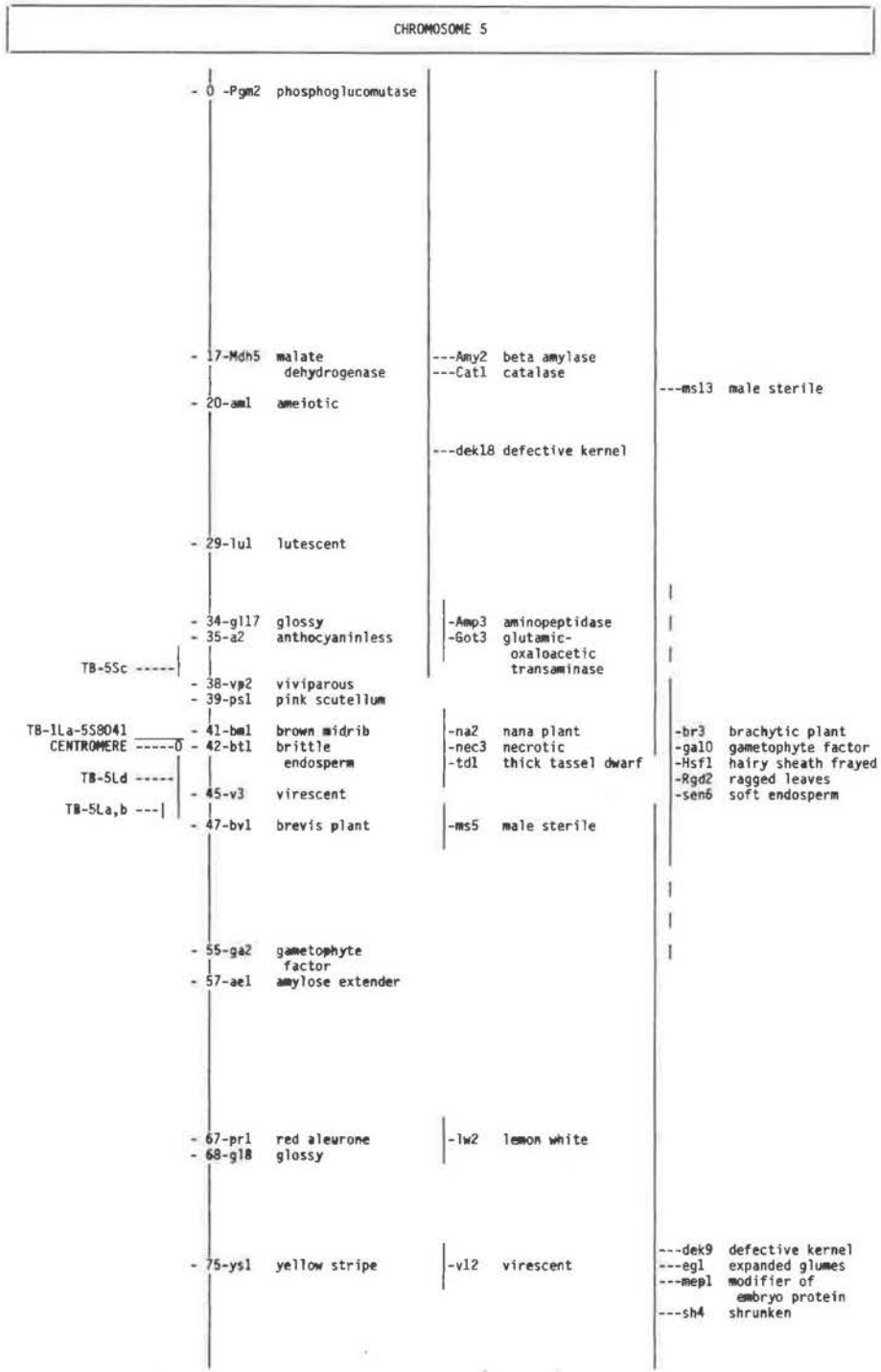

TB-1Sb-2Lc
-83-v4 virescent
-111-w3 white
-121-Ht1 H. turcicum resistance
-155-Ch1 chocolate
-Sks1 sterility suppressor
---dek4 defective kernel
---dek16 defective kernel
---dek23 defective kernel
---hcf1 high chlorophyll fluorescence
---Les4 lesion
---Rf3 fertility restorer
---whp1 white pollen

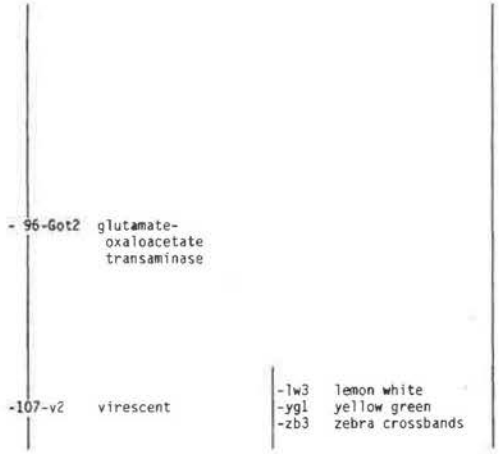

CHROMOSOME 3
- 0 -g2 golden plant
-E8 esterase
- 14-crl crinkly leaves
-Hex1 hexokinase
- 32-d1 dwarf plant
---brn1 brown aleurone
---dek5 defective kernel
---E3 esterase
- ra2 ramosa
- rt1 rootless
- 52-cl1 chlorophyll
-E4 esterase
TB-3Sb -----
---Rf1 fertility restorer
CENTROMERE -----0
-d2 dwarf plant
-h1 soft starch
- 57-Lg3 liguleless
-ms3 male sterile
TB-3La -----
-Tpi4 triose phosphate isomerase
-sen1 soft endosperm
TB-3Lf -----
- 59-Rg1 ragged leaves
-tel terminal ear
TB-3Lg -----
-ys3 yellow stripe
- 61-gl6 glossy
---Rp3 rust resistance
TB-3Lh ---
- 65-ts4 tassel seed
-pm1 pale mibrib
TB-3Ld -
-Pgd2 6-phosphogluconate dehydrogenase
-vp1 viviparous

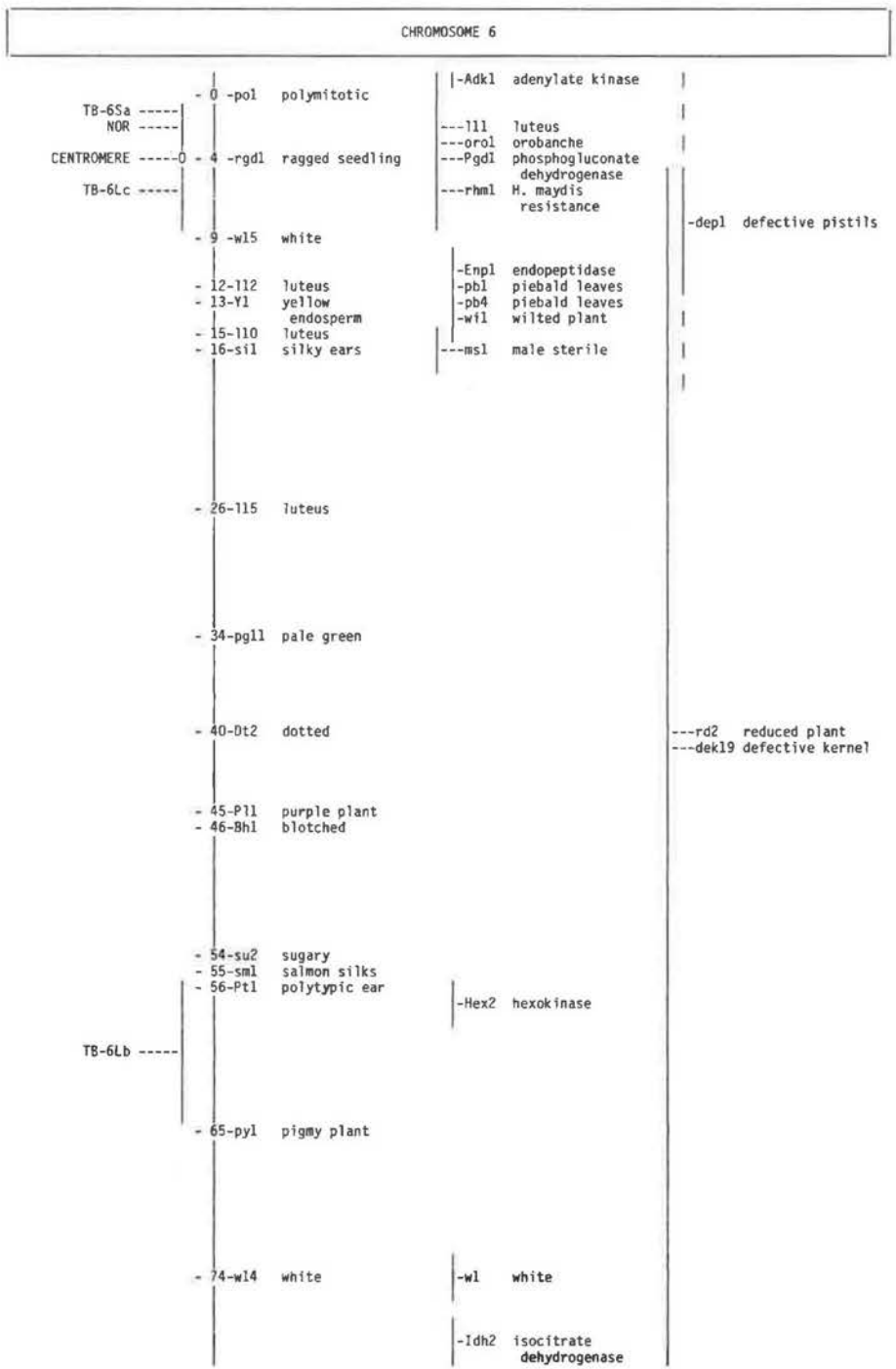

TB-3Li
TB-3Lj
-Spc1 speckled
- 82-ig1 indeterminate gametophyte
TB-3Lc
- 93-lg2 liguleless
- 94-ba1 barren stalk
-yd2 yellow dwarf
-105-na1 nana plant
-107-K3L knob
TB-3Lk
TB-3Ll
---dek17 defective kernel
---yl0 yellow endosperm
-Got1 glutamate-oxaloacetate transaminase
-117-Me1 malic enzyme
---dek6 defective kernel
-125-a3 anthocyanin
TB-3Lm
TB-3La-2S6270
TB-1La-3L5267
-138-Mdh3 malate dehydrogenase
-141-.0 al anthocyaninless
.2 sh2 shrunken
-153-et1 etched
-159-ga7 gametophyte factor

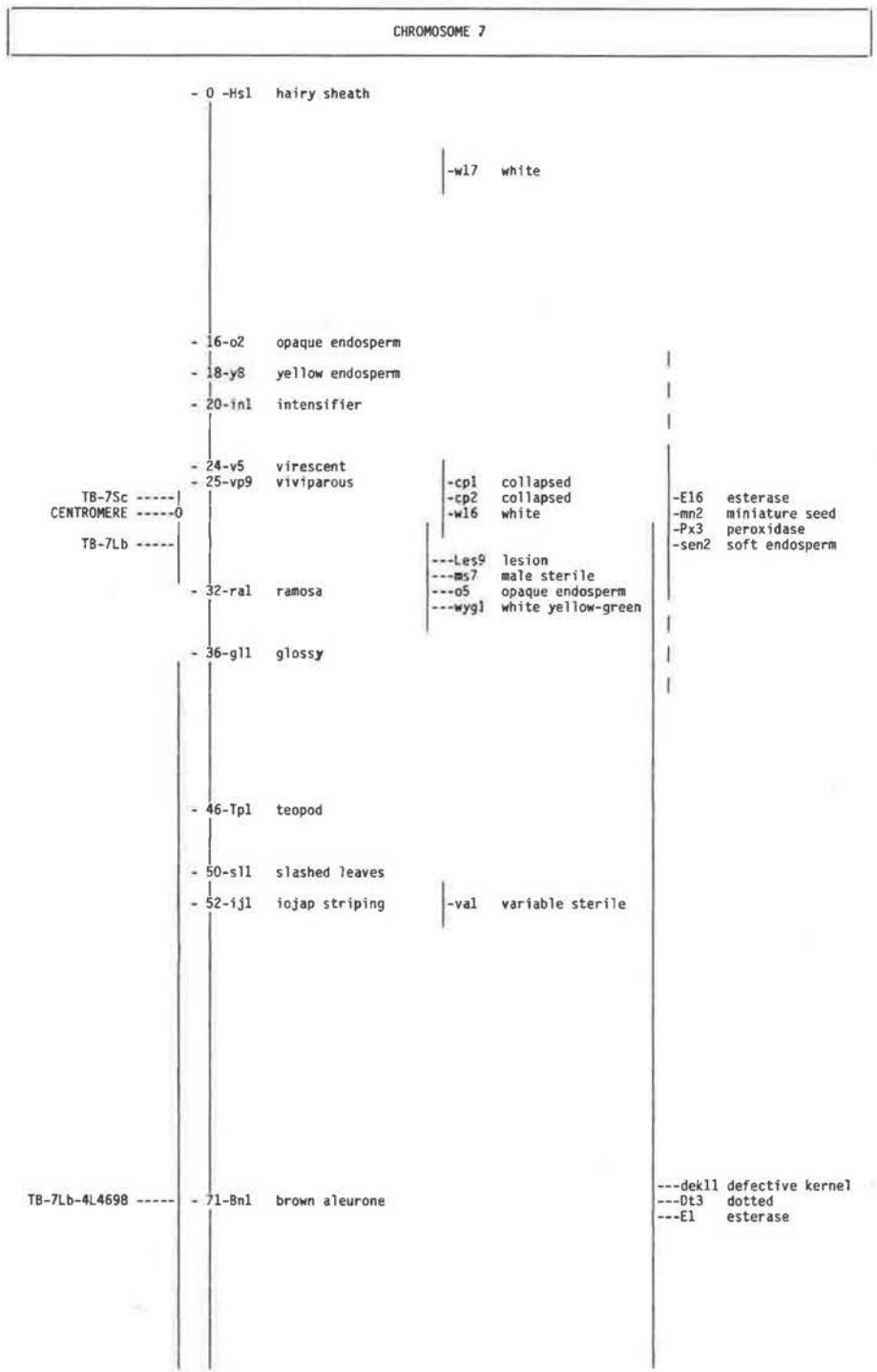

CHROMOSOME 4
- 0 -Phl pith abscission
- 19-Asr1 absence of seminal roots
- 24-Rp4 P. sorghi resistance
- 27-Ril rind abscission
- 32-Gal gametophyte factor
---Acol aconitase
---Bx1 benzoxazin
---dek7 defective kernel
- 46-Adh2 alcohol dehydrogenase
- 53-Ts5 tassel seed
- 55-la1 lazy plant
- 58-fl2 floury
- 62-st1 sticky chromosome
-bm3 brown midrib
- 66-su1 sugary
-Dt6 dotted
- 67-bt2 brittle endosperm
-v23 virescent
-Ysk1 yellow-streaked
-Dt4 dotted
-lpl lethal pollen
-Mal2 multiple aleurone layering
-ra3 ramosa
-v17 virescent
T8-4Sa -
CENTROMERE -0
T8-4Lb -
T8-4Lc -
T8-4Lf -
T8-1La-4L4692 -
T8-9Sb-4L6502 -
T8-9Sb-4L6222 -
- 79-zb6 zebra crossbands
-lw4 lemon white
- 81-gl4 glossy

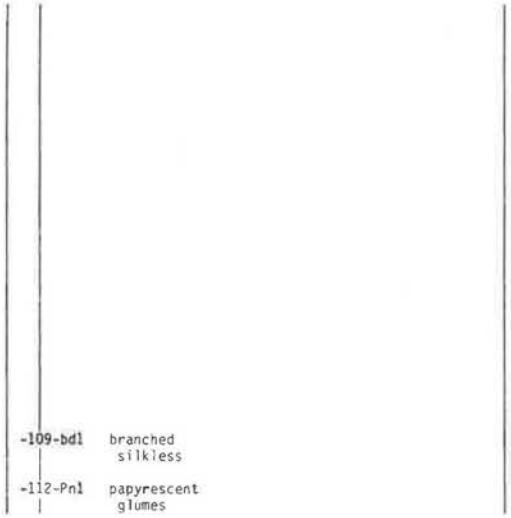

TB-4Le
-101-Tu1 tunicate
-v8 virescent
-106-j2 japonica striping
TB-4Ld
---dek8 defective kernel
---dek10 defective kernel
-112-gl3 glossy
-o1 opaque endosperm
TB-7Lb-4L4698
-117-c2 colorless
-137-dpl distal pale

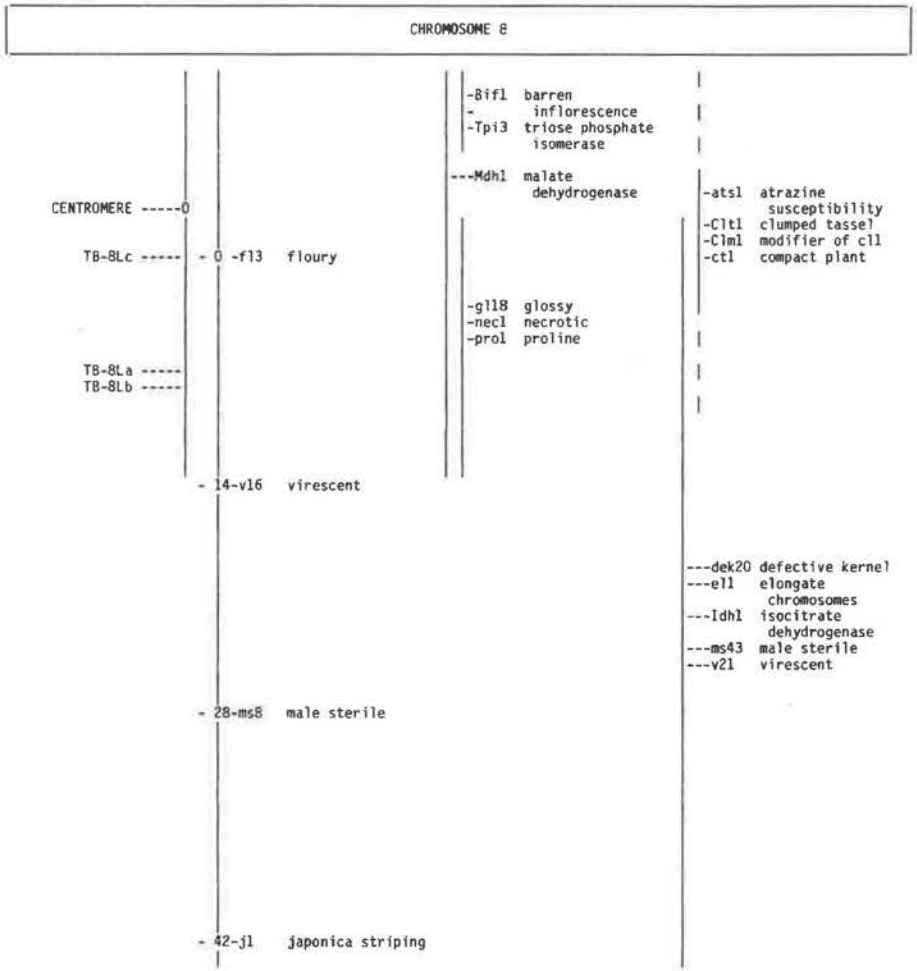

CHROMOSOME 5
0 -Pgm2 phosphoglucomutase
17-Mdh5 malate dehydrogenase
---Amy2 beta amylase
---Cat1 catalase
---ms13 male sterile
20-aml ameiotic
---dek18 defective kernel
29-lul lutescent
34-gl17 glossy
35-a2 anthocyaninless
-Amp3 aminopeptidase
-Got3 glutamic-oxaloacetic transaminase
TB-5Sc
38-vp2 viviparous
39-ps1 pink scutellum
TB-1La-5S8041
41-bml brown midrib
-na2 nana plant
-br3 brachytic plant
CENTROMERE
42-btl brittle endosperm
-nec3 necrotic
-tdl thick tassel dwarf
-ga10 gametophyte factor
-Hsf1 hairy sheath frayed
-Rgd2 ragged leaves
-sen6 soft endosperm
TB-5Ld
45-v3 virescent
TB-5La,b
47-bv1 brevis plant
-ms5 male sterile
55-ga2 gametophyte factor
57-ae1 amylose extender
67-pr1 red aleurone
-lw2 lemon white
68-gl8 glossy
75-ys1 yellow stripe
-v12 virescent
---dek9 defective kernel
---eg1 expanded glumes
---mep1 modifier of embryo protein
---sh4 shrunken

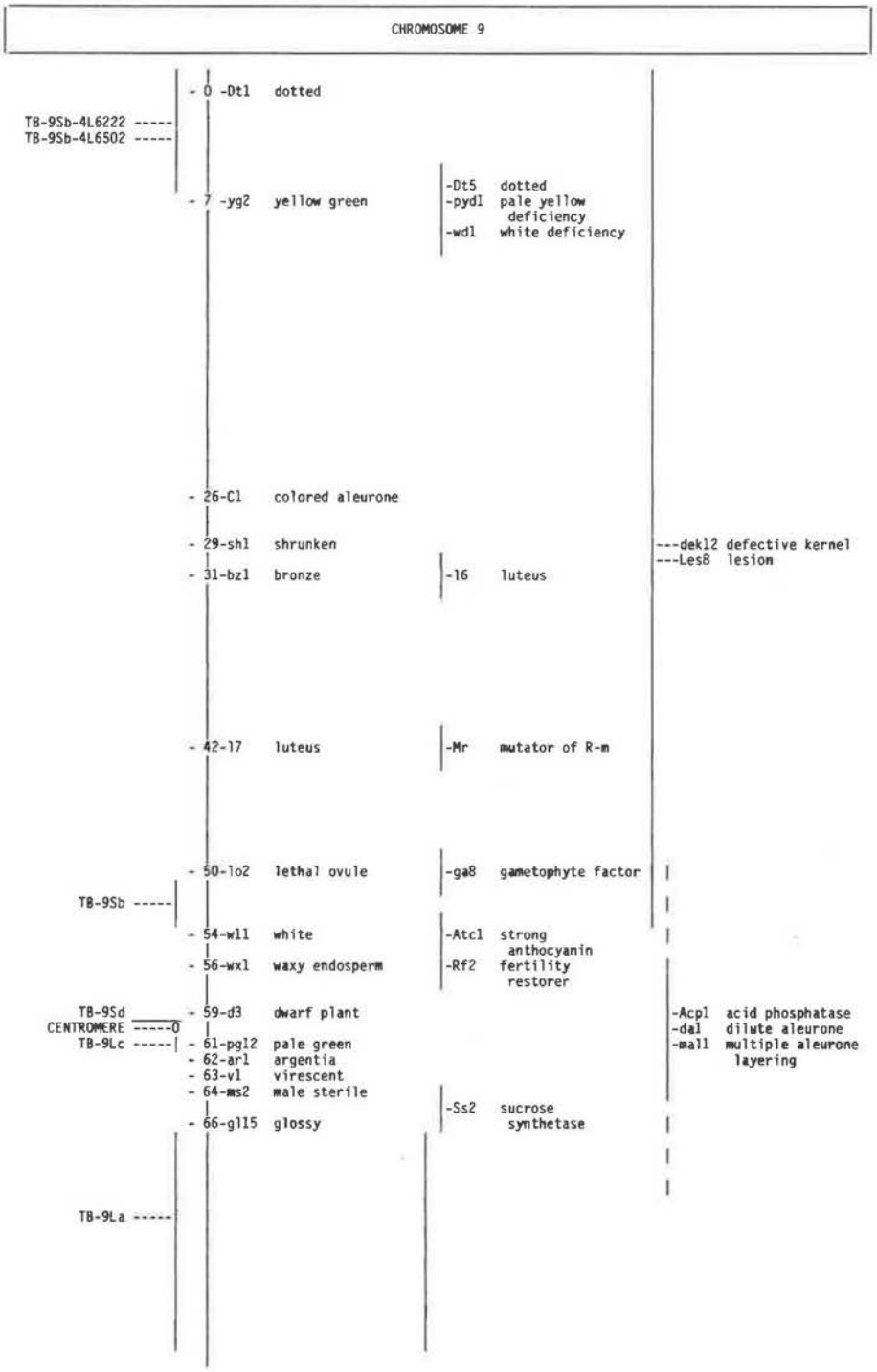

- 96-Got2 glutamate-
oxaloacetate
transaminase
-107-v2 virescent
-lw3 lemon white
-ygl yellow green
-zb3 zebra crossbands

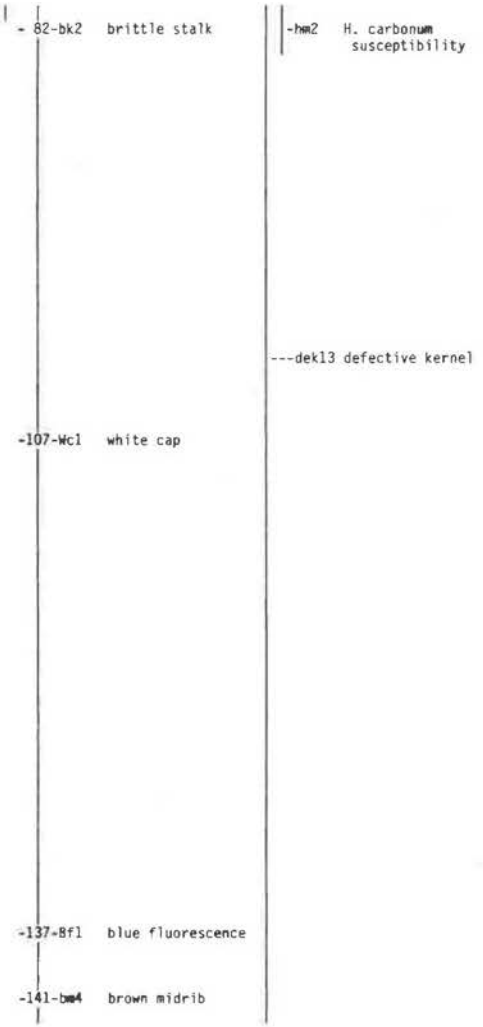

CHROMOSOME 6
TB-6Sa
NOR
CENTROMERE
TB-6Lc
TB-6Lb
0 -pol polymitotic
4 -rgd1 ragged seedling
9 -wl5 white
12-lt2 luteus
13-Y1 yellow endosperm
15-lt0 luteus
16-sil silky ears
26-lt5 luteus
34-pgl1 pale green
40-Dt2 dotted
45-Pl1 purple plant
46-Bh1 blotched
54-su2 sugary
55-sm1 salmon silks
56-Pt1 polytypic ear
65-py1 pigmy plant
74-wl4 white
-Adk1 adenylate kinase
---lt1 luteus
---oro1 orobanche
---Pgd1 phosphogluconate dehydrogenase
---rhm1 H. maydis resistance
-Enp1 endopeptidase
-pb1 piebald leaves
-pb4 piebald leaves
-wi1 wilted plant
---ms1 male sterile
-Hex2 hexokinase
-wl white
-Idh2 isocitrate dehydrogenase
-dep1 defective pistils
---rd2 reduced plant
---dek19 defective kernel

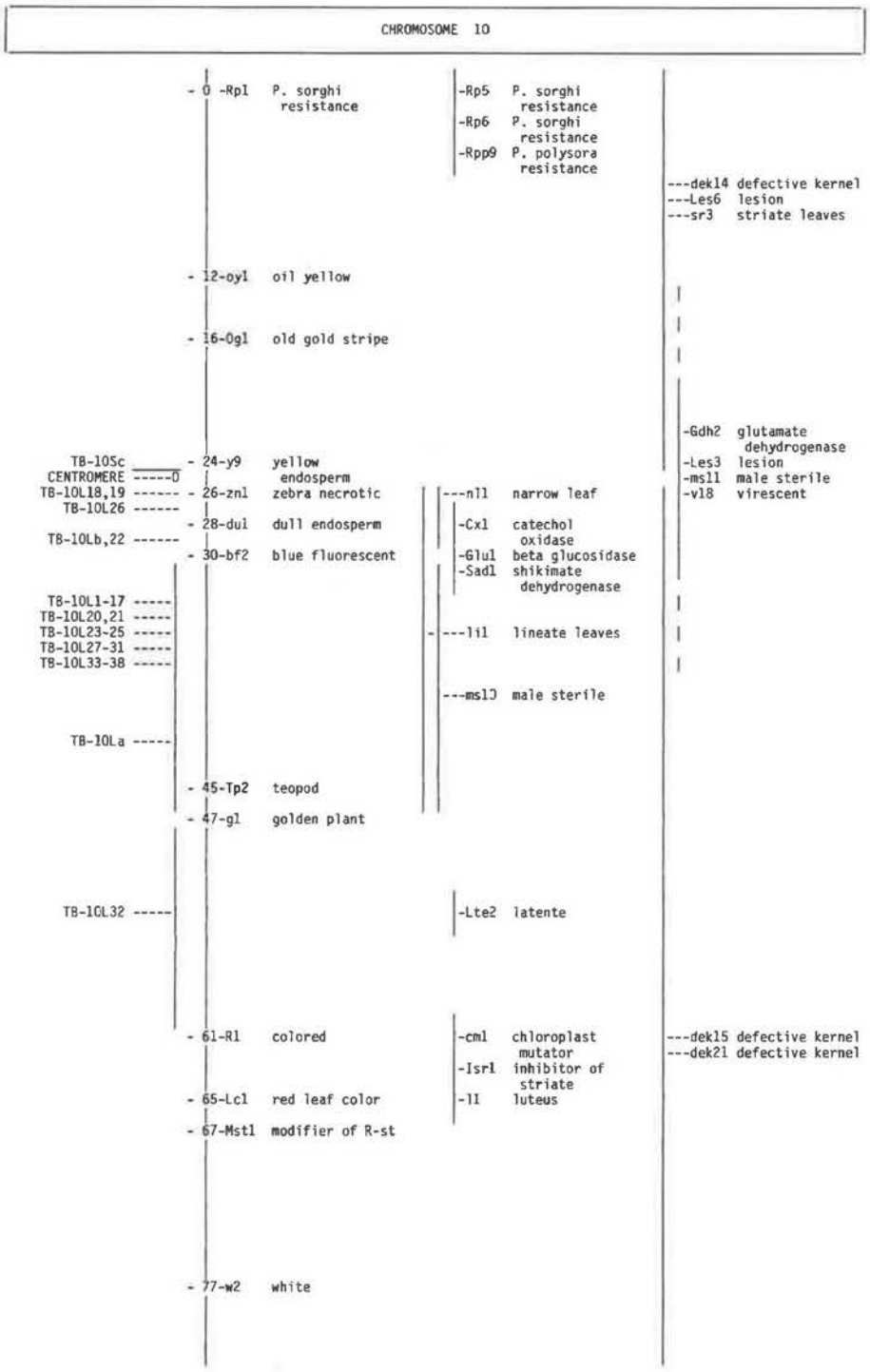

CHROMOSOME 7
- 0 -Hs1 hairy sheath
-w17 white
- 16-o2 opaque endosperm
- 18-y8 yellow endosperm
- 20-in1 intensifier
- 24-v5 virescent
- 25-vp9 viviparous
-cp1 collapsed
-cp2 collapsed
-w16 white
TB-7Sc
CENTROMERE
TB-7Lb
-E16 esterase
-mn2 miniature seed
-Px3 peroxidase
-sen2 soft endosperm
---Les9 lesion
---ms7 male sterile
---o5 opaque endosperm
---wyg1 white yellow-green
- 32-ra1 ramosa
- 36-gl1 glossy
- 46-Tp1 teopod
- 50-sl1 slashed leaves
- 52-ij1 iojap striping
-va1 variable sterile
TB-7Lb-4L4698
- 71-Bn1 brown aleurone
---dek11 defective kernel
---Dt3 dotted
---E1 esterase


-109-bd1
branched
silkless
-112-Pn1
papyrescent
glumes


CHROMOSOME 8
CENTROMERE
TB-8Lc
TB-8La
TB-8Lb
0
- 0 -f13 floury
- 14-v16 virescent
- 28-ms8 male sterile
- 42-j1 japonica striping
-8ifl barren inflorescence
-Tpi3 triose phosphate isomerase
---Mdh1 malate dehydrogenase
-gl18 glossy
-nec1 necrotic
-pro1 proline
-ats1 atrazine susceptibility
-Clt1 clumped tassel
-Clm1 modifier of clt
-ct1 compact plant
---dek20 defective kernel
---el1 elongate chromosomes
---Idh1 isocitrate dehydrogenase
---ms43 male sterile
---v21 virescent


CHROMOSOME 9
- 0 -Dt1 dotted
TB-9Sb-4L6222
TB-9Sb-4L6502
-Dt5 dotted
- 7 -yg2 yellow green
-pyd1 pale yellow deficiency
-wd1 white deficiency
- 26-C1 colored aleurone
- 29-sh1 shrunken
---dek12 defective kernel
---Les8 lesion
- 31-bz1 bronze
-16 luteus
- 42-17 luteus
-Mr mutator of R-m
- 50-lo2 lethal ovule
-ga8 gametophyte factor
TB-9Sb
- 54-w11 white
-Atc1 strong anthocyanin
- 56-wx1 waxy endosperm
-Rf2 fertility restorer
TB-9Sd
- 59-d3 dwarf plant
-Acp1 acid phosphatase
CENTROMERE
-da1 dilute aleurone
TB-9Lc
- 61-pg12 pale green
-ma11 multiple aleurone layering
- 62-ar1 argentia
- 63-v1 virescent
- 64-ms2 male sterile
-Ss2 sucrose synthetase
- 66-gl15 glossy
TB-9La


- 82-bk2 brittle stalk
-hm2 H. carbonum susceptibility
---dek13 defective kernel
-107-Wc1 white cap
-137-8f1 blue fluorescence
-141-bm4 brown midrib


CHROMOSOME 10
- 0 -Rp1 P. sorghi resistance
-Rp5 P. sorghi resistance
-Rp6 P. sorghi resistance
-Rpp9 P. polysora resistance
---dek14 defective kernel
---Les6 lesion
---sr3 striate leaves
- 12-oy1 oil yellow
- 16-0g1 old gold stripe
-Gdh2 glutamate dehydrogenase
TB-10Sc
- 24-y9 yellow endosperm
CENTROMERE -----0
-Les3 lesion
-msl1 male sterile
TB-10L18,19 ------ - 26-zn1 zebra necrotic
---nl1 narrow leaf
-v18 virescent
TB-10L26 ------
-Cx1 catechol oxidase
- 28-du1 dull endosperm
TB-10Lb,22 ------
- 30-bf2 blue fluorescent
-Glu1 beta glucosidase
-Sad1 shikimate dehydrogenase
TB-10L1-17 -----
TB-10L20,21 -----
TB-10L23-25 -----
TB-10L27-31 -----
TB-10L33-38 -----
---lil lineate leaves
---msl3 male sterile
TB-10La -----
- 45-Tp2 teopod
- 47-gl golden plant
TB-10L32 -----
-Lte2 latente
- 61-R1 colored
-cml chloroplast mutator
---dek15 defective kernel
---dek21 defective kernel
-Isr1 inhibitor of striate
- 65-Lc1 red leaf color
-ll luteus
- 67-Mst1 modifier of R-st
- 77-w2 white

- 87-o7	opaque endosperm		
- 91-113	luteus		
- 95-sr2	striate leaves	-K10	abnormal-10

COMPILED BY

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THE PHYSICAL MAP OF MITOCHONDRIAL DNA FROM THE MALE FERTILE CYTOPLASM OF MAIZE

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This paper presents the latest restriction endonuclease mapping data and gene locations for the mitochondrial genome of male fertile Zea mays (genotype Wf9). It is intended that updates be published in the Maize Genetics Cooperation News Letter and, to this end, a small data-base will be maintained containing relevant information. We would be pleased to receive and acknowledge any mapping data, either corrections to that given here or new data which is considered to be of interest to other researchers, for inclusion in this data-base and in future communications. The restriction mapping and gene location data will be made available on request.

As a service we will also be willing to position sequences of interest on the map. Such mapping data would not be included in the public data-base without permission or until after their publication.

The origin for all co-ordinate data is taken as the first base of the SstII recognition site in the '3Kb' repeat adjacent to the R1 homologous sequence. The positions are expressed in kilobase-pairs relative to this origin. The map is given in a single circular form which we have termed the master genome.

The data are shown in a form similar to that in the original publication of the map(6) to enable direct comparison of the two data sets. Table I gives the map co-ordinates of restriction endonuclease sites and the restriction fragments they yield in kilo-base pairs. The maps for SstII and SmaI are complete, whereas the map for XhoI is not. The regions not yet mapped are indicated by the word GAP and these gaps are shown in the graphical representation of the map (Figure I).

Table II shows the map co-ordinates of repeated sequences and genes as identified to date. The mapping data for all except the '3Kb' repeat are estimated, as sequence data are not yet available. References to published gene sequences or map data are given where available. The restriction map is shown graphically in Figure I.

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Table I. Restriction Endonuclease Data for Mitochondrial DNA from Male Fertile Zea mays

Circle Size:568.65Kb Number of Enzymes:3

XhoI (incomplete)		SstII		SmaI	
site	fragment	site	fragment	site	fragment
9.96		0.00		0.00	
10.63	0.67	3.57	3.57	26.40	26.40
12.36	1.73	15.27	11.70	38.30	11.90
14.15	1.79	38.77	23.50	39.26	0.96
16.90	2.75	44.45	5.68	46.96	7.70
18.12	1.22	47.22	2.77	49.33	2.37
30.57	12.45	48.06	0.84	58.99	9.66
35.42	4.85	59.36	11.30	68.19	9.20
42.82	7.40	73.66	14.30	75.34	7.15
47.62	4.80	74.11	0.45	76.67	1.33
105.38	GAP	94.93	20.82	78.41	1.74
109.28	3.90	106.68	11.75	88.07	9.66
116.28	7.00	115.81	9.13	92.04	3.97
121.08	4.80	116.65	0.84	92.73	0.69
182.28	GAP	119.42	2.77	103.05	10.32
185.77	3.49	125.10	5.68	105.39	2.34
187.60	1.83	126.68	1.58	108.59	3.20
193.10	5.50	135.95	9.27	108.89	0.30
195.85	2.75	162.63	26.68	114.56	5.67
209.13	13.28	185.13	22.50	116.93	2.37
212.86	3.73	205.19	20.06	124.72	7.79
217.30	4.44	231.25	26.06	125.68	0.96
230.32	13.02	248.25	17.00	134.43	8.75
231.20	0.88	252.05	3.80	137.25	2.82
233.81	2.61	267.35	15.30	142.95	5.70
234.26	0.45	268.82	1.47	153.95	11.00
245.06	10.80	271.82	3.00	159.20	5.25
253.50	8.44	281.02	9.20	160.35	1.15
254.88	1.38	283.92	2.90	161.83	1.48
262.68	7.80	303.92	20.00	171.03	9.20
402.05	GAP	327.42	23.50	181.63	10.60
403.39	1.34	328.95	1.53	186.48	4.85
408.85	5.46	330.70	1.75	188.13	1.65
419.47	10.62	336.60	5.90	224.33	36.20
421.39	1.92	342.20	5.60	229.03	4.70
425.59	4.20	364.10	21.90	232.96	3.93
427.89	2.30	388.40	24.30	240.81	7.85
437.39	9.50	389.53	1.13	241.66	0.85
442.19	4.80	401.83	12.30	243.43	1.77
442.57	0.38	411.63	9.80	243.80	0.37
450.69	8.12	412.26	0.63	248.25	4.45
454.34	3.65	416.57	4.31	251.90	3.65
468.44	14.10	418.27	1.70	258.40	6.50
472.54	4.10	419.25	0.98	266.50	8.10
476.19	3.65	424.65	5.40	267.66	1.16
479.09	2.90	472.89	48.24	270.06	2.40
479.79	0.70	474.94	2.05	277.36	7.30
486.68	6.89	496.04	21.10	283.46	6.10
498.98	12.30	543.92	47.88	285.04	1.58
508.58	9.60	544.42	0.50	298.84	13.80
510.68	2.10	549.27	4.85	301.49	2.65
515.36	4.68		19.38	308.89	7.40
521.24	5.88			320.75	11.86
534.51	13.27			327.75	7.00
537.42	2.91			344.65	16.90
542.69	5.27			345.81	1.16
553.99	11.30			346.94	1.13
556.06	2.07			363.70	16.76
556.74	0.68			368.50	4.80
561.04	4.30			369.38	0.88
563.30	2.26			370.12	0.74
564.60	1.30			375.82	5.70
	14.01			377.07	1.25
				385.37	8.30
				388.32	2.95
				390.95	2.63
				398.35	7.40
				403.15	4.80
				411.10	7.95
				416.51	5.41
				418.20	1.69
				453.32	35.12
				463.86	10.54
				469.24	5.38
				473.94	4.70
				475.97	2.03
				481.17	5.20
				482.37	1.20
				486.97	4.60
				489.85	2.88
				492.73	2.88
				493.27	0.54
				493.77	0.50
				494.22	0.45
				494.60	0.38
				498.04	3.44
				503.44	5.40
				508.79	5.35
				512.44	3.65
				515.19	2.75
				520.22	5.03
				530.76	10.54
				562.95	32.19
					5.70

Table II. Map Data for Repeated Sequences and Genes

Repeats			Genes			
Start	End	Size	Start	End	Gene	Reference
566.07	2.69	3KB	311.60	312.80	COB	2
245.67	250.94	3KB	353.60	355.20	COX I	4
6.96	7.96	1KB	398.42	401.97	26S	7,8
495.90	496.90	1KB	417.83	417.96	5S	7,10
15.90	17.14	2KB	418.07	420.04	18S	7,9
536.42	537.66	2KB	454.21	452.68	ATP A	1
37.52	51.52	14KB	521.11	519.58	ATP A	1
112.46	126.46	14KB	537.70	539.32	COX II	3
58.25	68.25	10KB	349.65	348.30	LS	5
161.09	171.09	10KB	329.50	328.01	ct 16S	8
452.03	464.20	12KB	2.50	8.85	R1	12,13
518.93	530.99	12KB	250.75	256.20	S2	12,13

NB. R1 and S2 refer to sequences in the N mitochondrial genome homologous to the linear episomes found in the RU and S-type cytoplasms respectively.

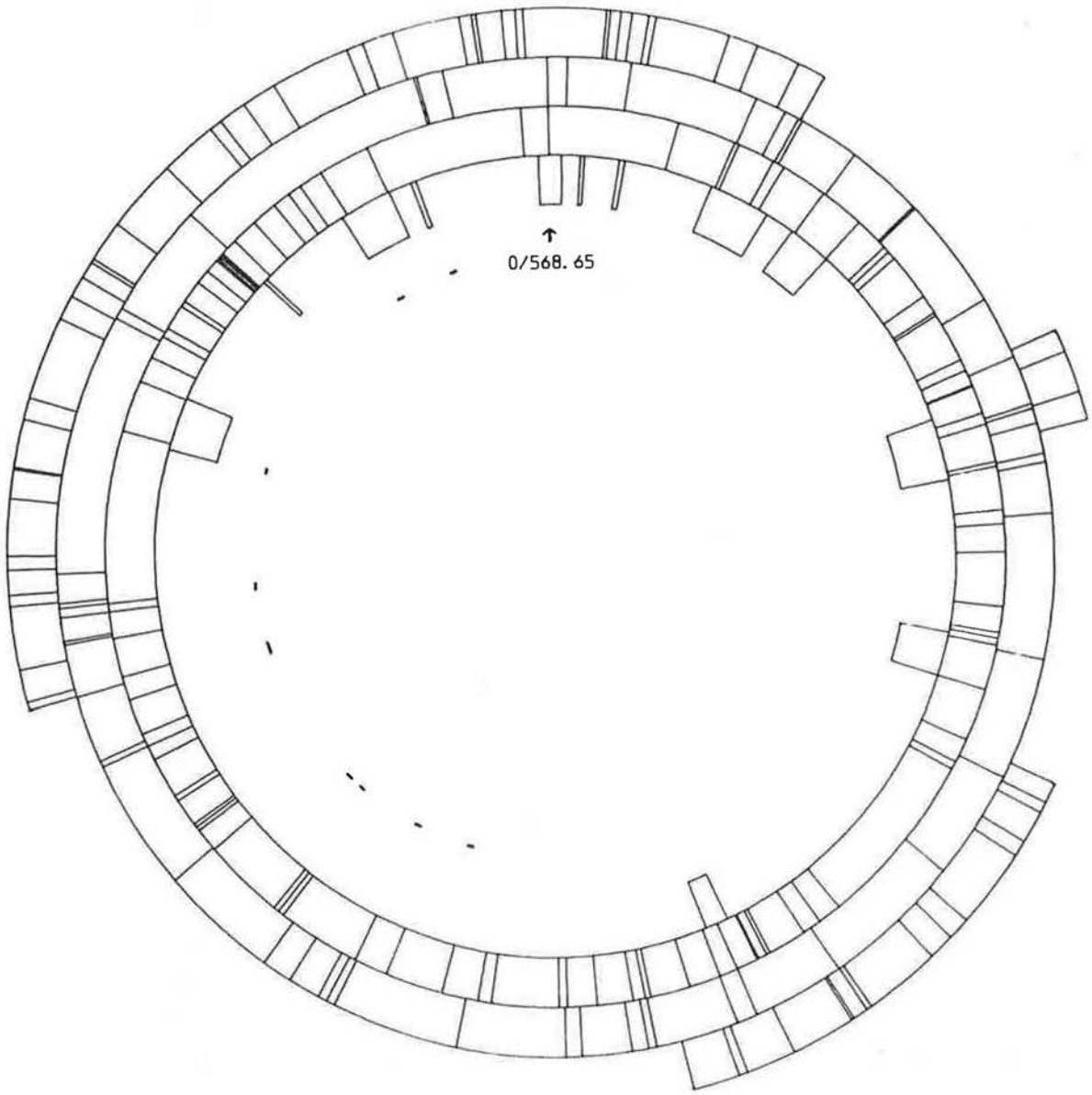

Figure I. Restriction endonuclease map of mitochondrial DNA from the male-fertile cytoplasm of Zea mays. The data shown in Table I are represented as a single circle with the enzymes XhoI, SstII and SmaI plotted in the outer, middle and inner circles respectively. The inner boxes show the approximate limits of the repeated sequences (Table II) and the inner set of short arcs are the map positions of the genes in Table II, excluding the sequences homologous to the R1 and S2 episomes.

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a1 7 8 33 42 53 54 107 118 155
A1-Can 134
a1-m1 129
a1-m1-5719A-1 2 134
a1-m1-5720 134
a1-m1-5996-4 134
a1-m1-6078 134
a1-m1:ACan 134
a1-m2-7977B 134
a1-m2-7995 134
a1-m2-8004 134
a1-m2:ACan 134
a1-m3:ACan 134
a1-m4 129
a1-m5 18
a1-m61138-1 134
a1-m(Au) 2 134
a1-m(dense) 134
a1-m(papu) 2 134
a1-m(r) 2
a1-Mum1 9 134
a1-mum2 4
a1-Mum2 9 134
a1-Mum3 9 134
a1-pale-m(r) 134
a1-ruq 5
a1-s 56
a2 33 56 116 147 158
a2-m1 42
a2-m1(1511) 42 135
a2-m5 147
a2-m-1 116
a3 33 155
Ac 4 5 20 36 39 40 42 55 98 107 129 134 136 136 137 139
Ac2 5 136
Ac9 42
Ac-flow 5 137
AcBz 20
Aco1 156
Acp1 33 166
Acp1-1 136
Acp1-2 136 137
Acp1-2* 136
Acp1-3 136 137
Acp1-3* 136
Acp1-3.5 136
Acp1-3.8 136
Acp1-4 136 137
Acp1-5 136
Acp1-5.5 136 137
Acp1-6 136 137
Acp1-n 136
Acp4 134 146 151
Acp4-2 109
Acp4-3 109
ad1 71 151
Adh1 33 61 62 98 118 129 134 134 146 151
Adh1-1S 24
Adh1-2 109 134
Adh1-2F11 40 134
Adh1-2F11::Ds2 39
Adh1-3F 24
Adh1-3F1124 24 134
Adh1-4 109 134
Adh1-5 134
Adh1-6 134
Adh1-FkFgamma25 23
Adh1-FM335 129
Adh1-Fm335 134
Adh1-n 134
Adh1-null 134
Adh1-S 17 23 62
Adh1-S3034 17 120 134
Adh1-S3034a 5 134 134
Adh1-S4477 134
Adh1-S4478 134
Adh2 33 118 156
Adk1 136 147 160
Adk1-4 110
Adk1-5 110
ae1 33 71 158
afd1 71
air1 71 138
al1 152
alh1 151
am1 158
Amp1 109 151
Amp2 150
Amp3 158
Amy2 33 158
an1 71 151
an1-bz2-6923 40 121 134
and 138
ar1 166
as1 150
Asr1 33 156
Atc1 33 166
ats 136
ats1 55 148 164
B1 33 34 51 134 139 152
B1-Peru 56
B1-W 34
B-chrom 133
ba1 71 155
ba2 71 152
bd1 129 163
Bf1 11 33 167
bf2 12 33 168
Bg 4 136
Bh1 33 160
Bif1 110 148 164
bk2 167
bm1 71 158
bm2 51 71 109 118 146 151
bm3 71 156
bm4 12 71 167
Bn1 162
br1 51 71 146 150
br2 71 146 151
br3 71 158
brn1 6 154
Bs1 139
bt1 33 56 71 115 158
bt1-4 135
bt2 56 71 156
bv1 71 158
bx1 33 135
Bx1 135 147 156
bz1 11 20 33 48 54 60 68 118 129 166
Bz1-'3 137
bz1-m 129
bz1-m1 136
bz1-m2 20 107 136 137
bz1-m2(Ac) 136
bz1-m2(DI) 136
bz1-m2(DII) 136 137
bz1-m4 5 59
bz1-m5 20
bz1-m5-A 20 137
bz1-m5-B 20 137
bz1-m5-C 20 137
bz1-m5-D 20 137
bz1-m11 136
bz1-m13 136 137
bz1-m13CS1 137
bz1-m13CS3 137
bz1-m13CS5 137
bz1-m13CS6 137
bz1-m13CS9 137
bz1-m13CS12 137
Bz1-McC 136
bz1-mu1 120
bz1-Mum4 107 136
bz1-mus4 4 137
bz1-mus7 4 137
bz1-mus8 4 137
bz1-n(rcy) 5
bz1-R 108
bz1-rcy 4 5
bz1-s:2114(Ac) 136
Bz1-wm 137
bz2 20 33 51
Bz2 134
bz2 146 151
bz2-6923 134
bz2-an1-6923 40 121
bz2-m 39 40 129 134 136
bz2-mu1 120 124 134
bz2-s 56
c1 11 33 42 43 54 55 56
C1 166
c1-846432 2 137
C1-I 2 5 42 50 57 137 137
C1-I-m836976 136
C1-I-m857056 3 137
C1-I-m857070 3 137
C1-I-m857101 3 137
C1-I-weak857054 3 137
C1-I-weak857061 3 137
C1-Im 75
C1-Im1 76 137
C1-Im2 76 137
c1-m2 42 137
c1-m(r) 2
c1-m668613 42 137
c1-m668655 42 137
c1-sh1-bz1-825211w 5 137
c1-sh1-bz1-825211y-2 5 137
c1-xm 76 137
c2 33 54 56 67 157
c2-m1 2 42 135
c2-m2 42 135
c2-m857210 2 135
c2-m857212 2 135
c2-m857213 2 135
c2-m857214 2 135
c2-m857229 2 135
c2-m857244 2 135
c2-m857246 2 135
c2-m857263 2 135
c2-m857271 2 135
c2-m857272 2 135
c2-m857273 2 135
c2-m857274 2 135
c2-m857328 2 135
c2-m857353 2 135
c2-m857364 2 135
c2-m857375 2 135
c2-s857230 2 135
c2-s857231 2 135
c2-s857234 2 135

c2-s857236 2 135
c2-s857241 2 135
Car1 33 150
Cat1 33 150
Cat2 33 150
Cat3 33
Cat3-5 137
Cat3-7 137 137
Cat3-9 137 137
Cat3-9.5 137
Cat3-10.4 137
Cat3-10.6 137
Cat3-11.2 137
Cat3-12 137 137
Cat3-n 137
cfr*-2018 49 134 146
Cg1 71 97
Ch1 153
Cin1-001 138
Cin1-102 138
Cin1-103 138
Cin1-201 138
cl1 6 56 154
Cl3 6
clf1 56
clf1-792 46 134
Clm1 164
Clone-1-407 138
Clone-1-634 138
Clone-c10Mu 134
Clone-c110En 134
Clone-cM1 139
Clone-E19 139
Clone-H2a 139
Clone-H2b 139
Clone-M6 139
Clone-M8 139
Clone-My2 139
Clone-P(186bp) 139
Clone-pCTE1409 139
Clone-pGMR3 73
Clone-pME119 139
Clone-pMON9502 139
Clone-pMx59 138
Clone-pMx71 138
Clone-pMx96 138
Clone-pMx97 138
Clone-pZm9'-8 71 139
Clone-pZmS21 101 139
Clone-pZmS42 101 139
Clone-S1 139
Clone-T2 139
Clone-T3 139
Clone-zA1 139
Clt1 148 164
cm1 168
cms-B/D 138
cms-C 69 112
cms-CA 69 138
cms-D 69
cms-G 126
cms-G' 126 138
cms-I 138
cms-J 126 138
cms-J' 126 138
cms-L 69 111 138
cms-LBM 111
cms-LBN 138
cms-Me 69
cms-ME 138
cms-ME(38-11) 113 138
cms-ML 127
cms-R 126
cms-R251 138
cms-R274 138
cms-R285 138
cms-R296 138
cms-R369 138
cms-R733 138
cms-R' 126 138
cms-RD 127
cms-RU 112 138
cms-S 69 101 110 112 113 126 127 128 129 138
cms-S(USDA) 138
cms-T 63 69 111 114 135 138
cms-T-R2 114
cms-VG 138
cmsR2 138
cp1 162
cp2 162
cp*-863A 46 135 147
cp*-931A 135 147
cp*-991 46 134 146
cp*-1054 46 137 148
cp*-1078B 46 134 146
cp*-1255B 46 136
cp*-1275A 46 135 147
cp*-1294 46 138
cp*-1308A 46 134 146
cp*-1387A 148
cp*-1406 46 136
cr1 30 71 146 154
crp*-888A 46 138
crp*-1429A 148
crp*-1528 46 136
ct1 164
ct2 150
ct* 96
ctDNA-3366 15
ctDNA-atpB 138
ctDNA-ps1A1 138
ctDNA-ps1A2 138
ctDNA-psbA 101
ctDNA-rbcL 138 138
ctDNA-rDNA4.5S 111
ctDNA-rDNA5S 111
ctDNA-rDNA16S 138
ctDNA-rDNA23S 111 138
ctDNA-rRNA4.5S 138
ctDNA-rRNA5S 138
ctDNA-rRNA16S 138
ctDNA-rRNA23S 138
ctDNA-tRNAarg 111 138
ctDNA-tRNAhis 138
ctDNA-tRNAvalGAC 138
Cx1 33 168
Cy 4 5
d1 30 71 109 135 146 154
d1-6016 30
d2 71 154
d3 71 166
d5 51 134 152
D8 151
da1 166
de*-1142 46 134 146
de*-1177A 46 138
de*-1196 46 135 147
de*-1296A 46 135 147
de*-7601 84 85
de*-B1 137
de*-B3 137
de*-B18 137
de*-B21 137
de*-B22 137
De*-B30 137
de*-B37 137
de*-B40 137
de*-B50 137
de*-B69 137
de*-B70 137
de*-B76 137
de*-B112 137
de*-B116 137
de*-B246 137
dek1 33 54 56 150
dek1-54-1 100 134
dek1-56-1 100 134
dek2 146 151
dek3 152
dek4 153
dek5 154
dek6 155
dek7 156
dek8 157
dek9 158
dek10 157
dek11 162
dek12 166
dek13 167
dek14 168
dek15 168
dek16 153
dek17 155
dek18 149 158
dek19 160
dek20 164
dek21 168
dek22 146 151
dek23 153
dek24 64 135 146
dek25 64 135 147
dek26 64 135 147
dek27 64 135 147
dek28 64 136 147
dek29 64 136 148
dek30 64 137 148
dek*-744 56
dep1 160
Df9S 148
Df9S104-7 11 137
Df9S107-1 11 137
Df9S107-2 11 137
Df9S108-3 11 137
Df9S108-8 11 137
Df9S110-6 11 137
Df9S110-8 11 137
Df9S110-9 11 137
Df9S114-1 11 137
Df9S116-10 11 137
Df9S117-5 11 137
Df9S117-8 11 137
Df9S-857078 3 137
Df-c1-857177 3 137
Df-c1-sh1-bz1-857003 3 137
Df-c1-sh1-bz1-857164 3 137
Df-c1-sh1-bz1-857182 3 137
Df-c1-sh1-bz1-857183 3 137
DfK10(C) 26 27 137
DfK10(F) 27 137
DfK10(H) 26 27 137
DfK10(I) 137
DfK10(K) 137

Dia1 33 152
Dia2 134 146 151
Dia2-4 109
Dia2-6 109
Dmr* 134
dp1 157
Dp9-McC 136
Dp9-MMR 136
Ds 5 20 39 40 42 55 56 71 129 136 136 136 137 137
Ds1 134
Ds2 39 40 129 134
Ds9 39 42
Ds101 139
Ds103 139
Ds105 139
Ds132 139
Ds-2 55 56
dsc*-1315A 46 134 146
dSpm 19 139
dSpm13 137
dsy1 71
dsy2 71
Dt 4 134
Dt1 11 33 166
dt1 56
Dt2 33 160
Dt3 162
Dt4 156
Dt5 33 166
Dt6 33 156
Dt* 19
Dt-TL 137
du1 71 168
du11 71 138
E1 33 162
E3 154
E4 135 154
E4-2 30
E4-3 31
E4-5 30
E8 154
E8-2.5 134
E8-3 134
E8-4 134
E8-4.5 134
E8-5 134
E8-5.8 134
E8-6 134
E8-7 134
E8-8 134
E8-n 134
E16 162
eg1 158
el1 107 164
En 2 4 41 42 115 129 134 135 136 137 137
En1 134 136
En-1 137
En-803920-3 2
Enp1 136 160
Enp1-1 31 135
Enp1-2 31 135 135
Enp1-3 31 135
Enp1-4 31 135 135
Enp1-5 135
Enp1-6 31 110 135 135
Enp1-6.2 135
Enp1-7 135
Enp1-8 31 135 135
Enp1-10 31 110 135 135
Enp1-14 135
Enp1-n 135
Enp1-null 135
erl*-1729 56 138
erl*-2077 56 138
Erl*-2102 56 138
Est4 30 146
et1 71 155
f1 146 151
ffr1 71 138
ffr2 71 138
ffr3 71 138
ffr4 71 138
fl1 33 152
fl1-a 75
fl2 71 114 135 135 156
fl3 110 164
fl*-1145A 46 136 138
fl*-1163 46 138
fl*-1299 46 135 147
Flt2 33
Flt9 33
Flt11 33
fmd1 71 138
fsh1 71 138
fww1 71 138
g1 71 118 168
g2 30 71 135 146 154
Ga1 74 114 156
ga2 158
ga7 155
ga8 166
ga10 158
Gdh1 109 151
Gdh2 168
Ger4 33
Ger5 33
gl1 8 52 118 136 162
gl2 51 134 152
gl3 117 157
gl4 117 156
gl6 30 31 113 135 146 154
gl8 13 14 117 158
gl11 152
gl14 152
gl15 166
gl17 158
gl18 164
Glu1 33 137 148 168
Glu1-1 110 137 137
Glu1-2 137 137
Glu1-2.5 110 137
Glu1-3 137 137
Glu1-3.2 137
Glu1-3.5 137
Glu1-4 137
Glu1-5 137
Glu1-6 137 137
Glu1-7 137 137
Glu1-7.8 137
Glu1-8 137
Glu1-9 137
Glu1-10 137 137
Glu1-11 137
Glu1-12 137
Glu1-13 137
Glu1-16 137
Glu1-n 137
Glu1-null 137
gm*-1319C 46 136
Got1 155
Got1-1 134
Got1-1.2 134
Got1-4 134 134
Got1-5.8 134
Got1-6 134 134
Got1-6.2 134
Got1-7.5 134
Got1-n 134
Got2 159
Got2-2 135
Got2-4 135
Got2-6 135
Got2-7 135
Got2-n 135
Got3 158
Got3-2 135
Got3-3 135
Got3-4 135 135
Got3-6 135
Got3-7 135
Got3-n 135
gs 146
gs1 51 71 146 151
gs2 152
gt1 71 117 150
h1 154
hcf1 153
hcf2 16 151
hcf3 134 146 150
hcf6 16 134 146
hcf38 135 147
hcf42 136 148
hcf50 134 146
hcf*-38 49
hcf*-43 49
hcf*-1218-20 48 138
Hex1 33 154
Hex2 33 136 160
Hex2-1 110
Hex2-2 110
Hft 19
hm1 150
hm2 167
Hs1 162
Hsf1 135 158
Hsf*-1595 135
Hsf*-1603 135
Ht1 153
I 2 129 134
id1 71 117 151
Idh1 33 148 164
Idh1-2 110 136
Idh1-3 136
Idh1-4 110 136
Idh1-6 136
Idh1-8 136
Idh2 33 110 136 160
Idh2-2 135
Idh2-3.8 135
Idh2-4 135
Idh2-4.1 135
Idh2-4.2 135
Idh2-5 135
Idh2-6 135
Idh2-7 135
Idh2-7.5 135
Idh2-7.8 135
ig1 128 155
ij1 25 162
in1 162
Isr1 168
j1 50 57 110 112 134 136 148 164
j2 157
K1S 134
K2L 104 134

K2S 134
K3L 134 155
K3S 134
K4L 104 135
K4S 135
K5L 104 135
K5S 135
K6L2 135
K6L3 135
K6La 104
K6Lb 104
K6Lc 104
K6S 104
K7L 104 132 136 136
K7S 136
K8L 132 136
K8L1 136
K8L2 136
K8La 104
K8Lb 104
K9L 104
K9S 104 136
K10 68 69 169
K10-I 26 27 137
K10-II 26 137
Kn1 137 146 151
Kn1-2F11 134
Kn1-N1 134
Kn1-N2 23 134
Kn1-O 134
Kn1-Z2 134
Kn1-Z3 134
Kn1-Z4 134
Kn2-Z4 137
Krn1 34
Krn2 33
Krn4 33
Krn9 33
Krn11 33
l1 168
l6 166
l7 166
l10 113 136 147 168
l11 168
l12 113 136 147 168
l13 27 137 169
l15 168
la1 71 117 156
Lc1 168
Les1 49 51 57 134 146 152
Les2 51 134 146 150
Les3 168
Les4 51 134 146 153
Les5 51 134 146 150
Les6 51 137 148 168
Les7 51 134 151
Les8 51 137 148 166
Les9 51 52 136 147 162
les*-501B 51
Les*-843 51
Les*-845 51
Les*-1375 51 134
Les*-1378 51 134 146
les*-1395 51 138
Les*-1438 51 138
Les*-1442 51 138
Les*-1449 51 134
Les*-1451 51 137
Les*-1453 51 137 148
Les*-1461 51 134 146
les*-1521C 51 138
Les*-2003 51 138
Les*-2004 51 134 146
Les*-2005 51 137
Les*-2007 51 138
Les*-2008 51 52 136 147
les*-2012 51 138
les*-2013 51 138
Les*-2016 51 137 148
les*-A467 51 138
Les*-A607 51
les*-A721 51 138
Les*-A762 51 138
Les*-F331035 52 136
lg1 71 134 152
lg2 71 155
Lg3 71 109 113 135 146 154
lgp*-2006 56 138
lgp*-2007 56 138
li1 168
lls1 51 134 146 150
lls1-501B 51
lo2 43 137 148 166
lp1 156
Lt*-19 115 138
Lt*-20 115 138
lte1 35 152
Lte2 168
lty1 137
lty1-16 133
lty2 137
lu1 158
lw1 23 146 151
lw2 158
lw3 159
lw4 156
Lxm*-1600 137
ma11 166
Ma12 156
mbr*-2008 56 138
Mdh1 33 148 164
Mdh1-0.1 136
Mdh1-0.05 136
Mdh1-1 136
Mdh1-2 136
Mdh1-2.8 136
Mdh1-5 136
Mdh1-6 110 136
Mdh1-6.4 136
Mdh1-6.5 110
Mdh1-8.5 136
Mdh1-9.2 136
Mdh1-10.5 136
Mdh1-n 136
Mdh2 33 110 136 147 160
Mdh2-0.2 135
Mdh2-0.4 135
Mdh2-3 135
Mdh2-3.5 135
Mdh2-3.8 135
Mdh2-4.5 135
Mdh2-5 135
Mdh2-5.6 135
Mdh2-5m 135
Mdh2-6 135
Mdh2-7.7 135
Mdh2-null 135
Mdh3 33 155
Mdh3-11.5 134
Mdh3-15.8 134
Mdh3-16 134
Mdh3-16.3 134
Mdh3-16.9 134
Mdh3-17.2 134
Mdh3-18 134
Mdh3-n 134
Mdh4 33 109 151
Mdh4-9 134
Mdh4-12 134
Mdh4-14.5 134
Mdh4-15.5 134
Mdh4-16 134
Mdh4-n 134
Mdh5 33 158
Mdh5-5.5 135
Mdh5-7 135
Mdh5-8 135
Mdh5-12 135 135
Mdh5-14.4 135
Mdh5-15 135 135
Mdh5-16 135
Mdh5-16.4 135
Mdh5-n 135
Mdh5-null 135
Me1 33 155
Me1-F 134
Me1-n 134
Me1-R 134
Me1-S 134
meg1 71 138
mei1 71
mep1 158
Mer1 33
mi2 150
mmm 33 109
mmm1 151
Mmm1-m1 134
Mmm1-m3 134
Mmm1-mmm 134
Mmm1-Mmm 134
Mmm-mmm 134
Mmm-Mmm 134
mn1 71 152
mn2 71 162
Modifier 19
Mof*-1 45 138
Mof*-2 45 138
mono-4 135
mono-9 60
Mp 36 40 129
Mp11 146
Mr 166
Mrh 4
ms1 113 117 136 160
ms2 12 166
ms3 154
ms4 135
ms5 158
ms7 162
ms8 107 164
ms9 150
ms10 168
ms11 168
ms12 150
ms13 158
ms14 150
ms17 150
ms28 71
ms43 71 106 136 148 149 164
ms*-si 113 136
msc*-1330 46 137 148
Mst1 168
mtDNA 170
mtDNA-1.94kb 138
mtDNA-atp6 138
mtDNA-atp9 138
mtDNA-atpalpha 138

mtDNA-COB 127 138
mtDNA-COI 127 138
mtDNA-COII 127 138
mtDNA-cox1 138
mtDNA-R1 138
mtDNA-R-1 110
mtDNA-R-2 110
mtDNA-R-5 63
mtDNA-rDNA-18S 138
mtDNA-S1 101 126 127 128 138
mtDNA-S2 101 126 127 128 138
mtDNA-S-1 69 110 113
mtDNA-S-2 69 110 113
mtDNA-T7 111
mtDNA-T-4 63
mtDNA-T-7 63
mtDNA-T-R2 114
mtDNA-tRNAasp 138
mtDNA-tRNAhis 138
mtDNA-tRNAmet1 138
mtDNA-tRNAmet2 138
mtDNA-tRNAphe 138
Mu 4 7 8 10 11 12 13 14 47 120 121 124 129 134 134 135 135 136 138
Mu1 4 5 10 12 15 16 17 24 48 107 120 134 137 138
Mu1.4MF-Adh1-S3034 120 134
Mu1.4Mf-Adh1-S3034a 120
Mu1.4VS-B37 138
Mu1.4VW-B37 120
Mu1.7VW-1 120
Mu2 138
Mu3 24 134
Mu-1 134
Mu-L 137
MuE- 120 138
Mut 4 152
na1 71 155
na2 158
nec1 148 164
nec2 150
nec3 158
nec4 51 134 146 152
nl1 71 160
NOR 99 160
o1 71 157
o2 52 71 136 162
o2-261 136
o2-Agroceres 136
o2-Columbian 136
O2-m1 136
o2-m2 136
O2-m3 136
O2-m4 136
o2-mh 136
o5 71 162
o6 100 136 148
o7 27 169
o*-744 46 137 148
o*-874B 56
o*-945A 46 138
o*-1096A 46 136
o*-1241 46 136 147
o*-1298 46 138
Og1 168
ora3 137
oro1 160
orp1 55 138
orp2 55 138
orp*-1186 46 138
oy1 56 168
Oy1-1459 57 137
P1 33 150
P1-Quebec36 36 134
P1-RR 5 36 40
P1-rr 134
P1-RR 134
P1-RW 40
P1-VV 36 40 55 56
P1-vv 134
P1-VV 134
P1-WR 5 34 36 40 55
P1-WRB 55
P1-WW 5 34 36
P1-ww 134
P1-WW 139
P1-WWB 55 134
P1-WWW 55 134
P-VV 129
pam1 71
pam2 71
pb1 160
pb4 160
pe1 117
pg11 110 160
pg12 166
Pgd1 33 136 160
Pgd1-0.5 135
Pgd1-1 135
Pgd1-1.5 135
Pgd1-1.8 135
Pgd1-2 110 135
Pgd1-2.8 135
Pgd1-3.8 110 135 135
Pgd1-7 135
Pgd1-12 135
Pgd1-n 135
Pgd1-N 135
Pgd1-null 135
Pgd2 33 112 154
Pgd2-1 31 135
Pgd2-2 31 135
Pgd2-2.8 31 134 135
Pgd2-2.8* 135
Pgd2-5 31 134 135
Pgd2-6 135
Pgd2-8 134
Pgd2-10 134 135
Pgd2-11 134 135
Pgd2-n 134
Pgd2-N 135
Pgm1 33 109 151
Pgm1-1 134
Pgm1-7 134
Pgm1-9 134
Pgm1-9.5 134
Pgm1-13 134
Pgm1-16 134
Pgm1-16.5 134
Pgm1-17 134
Pgm1-18 134
Pgm1-19 134
Pgm1-21 134
Pgm2 33 150
Pgm2-0.5 135
Pgm2-0.45 135
Pgm2-3 135 135
Pgm2-4 135 135
Pgm2-7.2 135
Pgm2-7.3 135
Pgm2-7.5 135
Pgm2-8 135 135
Pgm2-12 135
Phi 156
Phi1 33 134 146 151
Phi1-1 134
Phi1-2 134
Phi1-3 134
Phi1-4 109 134
Phi1-5 134
Phi1-6 134
Phi1-8 109
pi1 43 71 138
pi2 43 138
Pl1 33 160
pl1 34 71
Plp1 34 139
pm1 154
Pm1 71 129 163
po1 71 135 160
pr1 33 48 49 54 56 66 67 117 118 158
prf1 71 138
prf2 71 138
pro1 56 71 100 136 148 164
pro1-1121 46 136
Prot1 151
ps1 33 158
Pt1 71 160
ptd*-1130 46 135 147
Px1-1 137
Px1-2 137
Px1-3 137
Px1-null 137
Px2-1 137
Px2-2 137
Px3 162
Px3-1 136
Px3-2 136
Px3-3 136
Px3-4 136
Px3-5 136
Px3-6 136
Px4-1 137
Px4-2 137
Px4-3 137
Px5-1 137
Px5-null 137
Px6-1 137
Px6-null 137
Px7-1 137
Px7-2 137
Px8-1 137
Px8-2 137
Px9-1 137
Px9-2 137
Px9-null 137
py1 71 110 160
pyd1 166
R1 6 33 160
r1 27 54 55 69 118
R1-g 60
R1-g:8pale 137
r1-m1 137
R1-N1-571-1 137
R1-N1-571-2 137
R1-N1-571-3 137
R1-N1-575-1 137
R1-N1-575-3 137
R1-N1-575-4 137
R1-N1-576-3 137
R1-N1-576-5 137
r1-r:n35 137
r1-r:n46 137

r1-r:n101 137
r1-r:n156 137
R1-sc 55 56 129
R1-sc:124 137
r1-x1 60 101 118
R9 136
ra1 51 52 136 147 162
ra2 30 71 109 135 146 154
ra3 156
rd1 71 96 146 151
rd2 160
rDNA 22 73
rDNA5.8S 58
rDNA6.9 135
rDNA8.0 135
rDNA9.1 135
rDNA18S 58
rDNA25S 58
rDNA-HpaII-6.9 22
rDNA-HpaII-8.0 22
rDNA-HpaII-9.1 22
rDNAclone-pGMR3 136
rDNAspacer 58 135
rearr9 136
Rf1 109 135 146 154
Rf2 166
Rf3 153
Rf4 112 134 136 148 152
Rf*-var 135
RfI 129
RfII 129
RfIII 129
RfIV 129
RfIX 129
RFP-1 119 136
RFP-1-45 138
RFP-2 119 136
RFP-2-226 138
RFP-3 119 136
RFP-4 119 134
RFP-5 119 136
RFP-6 119 135
RFP-6-59s 139
RFP-7 119 136
RFP-7-31sb 139
RFP-9 119 136
RFP-9-55s 139
RFP-9-72sa 139
RFP-10 119 134
RFP-11 119 134
RFP-12 119 135
RFP-13 119 135
RFP-14 60 119 137
RFP-17 119 135
RFP-18 119 135
RFP-19 119 134
RFP-20 119 134
RFP-22 119 137
RFP-23 119 136
RFP-24 119 134
RFP-25 119 137
RFP-26 119 136
RFP-27 119 135
RFP-28 119 136
RFP-29 119 136
RFP-30 119 119 134 136
RFP-31 119 135
RFP-32 119 134
RFP-33 119 136
RFP-34 60 119 137
RFP-35 119 136
RFP-36 119 135
RFP-37 119 136
RFP-38 119 136
RFP-39 119 136
RFP-40 119 134
RFP-41 119 135
RFP-42 119 135
RFP-43 119 136
RFP-44 119 136
RFP-45 119 136
RFP-46 119 134
RFP-47 119 136
RFP-48 119 136
RFP-49 119 134
RFP-50 119 136
RFP-52 119 135
RFP-53 119 135
RFP-54 119 134
RFP-55 119 135
RFP-56 119 135
RFP-57 119 134
RFP-59 119 136
RFP-60 119 135
RFP-61 119 134
RFP-63 119 136
RFP-64 119 136
RFP-65 119 134
RFP-66 119 135
RFP-67 119 136 138
RFP-68 119 136
RFP-69 119 136
RFP-70 119 135
RFP-71 119 135
RFP-72 119 136
RFP-73 119 135
RFP-74 119 134
RFP-75 119 135
RFP-77 119 135
RFP-78 119 135
RFP-79 119 136
RFP-80 60 119 137 138
RFP-82 119 134
RFP-83 119 135
RFP-84 119 134
RFP-85 119 137
RFP-86 60 119 137
RFP-88 119 119 135 136
RFP-89 119 135 138
RFP-90 119 135
RFP-91 119 135
RFP-92 119 137
RFP-93 119 135
RFP-94 119 135
RFP-95 119 135
RFP-96 119 134
RFP-97 119 137
RFP-98 119 137
RFP-99 119 134
RFP-100 119 136
RFP-101 119 136
RFP-102 119 136
RFP-103 119 136
RFP-104 119 135
RFP-105 119 137
RFP-106 119 135
RFP-107 119 136
RFP-108 119 135
RFP-109 119 135
RFP-110 119 136
RFP-111 119 136
RFP-112 119 136
RFP-113 119 136
RFP-114 119 136
RFP-115 119 135
RFP-116 119 135
RFP-118 119 134
RFP-120 119 134
RFP-121 119 134
RFP-123 119 134 138
RFP-183 138
RFP-201 119 138
RFP-202 119 138
RFP-297 138
RFP-301 119 136
RFP-302 119 136
RFP-303 119 136
RFP-304 119 134
RFP-305 119 137
RFP-306 119 137
RFP-307 119 135
RFP-308 119 134
RFP-309 119 137
RFP-310 119 135
RFP-311 119 135
RFP-312 119 137
RFP-313 119 135
RFP-314 119 134
RFP-315 119 136
RFP-317 119 135
RFP-318 119 137
RFP-319 119 134
RFP-320 119 119 134 136
RFP-321 119 137
RFP-322 119 136
RFP-323 119 138 138
RFP-324 119 137
RFP-325 119 136
RFP-326 119 137 138
RFP-327 119 138
RFP-328 119 136
RFP-330 119 134
RFP-331 119 136
RFP-333 119 135
RFP-334 119 138
RFP-336 119 134
RFP-343 138
RFP-369 138
RFP-380 138
RFP-501 138
RFP-556 138
RFP-779 138
RFP-859 138
RFP-hsp1 119 136
RFP-sus2 119 137
RfV 129
RfVI 129
RfVII 129
RfVIII 129
RfX 129
Rg1 30 109 113 135 154
rgd1 110 160
Rgd2 158
rgh*-974A 46 136
rgh*-1825 148
rhm1 160
Ri1 156
ring-9 50 57
Rld*-1441 137
Rld*-1608 137
Rp1 168
Rp3 154
Rp4 156
Rp5 168
Rp6 168
Rpp9 168
rs1 150
Rs1-R 134
Rs*-1606 137

rt1 154
Sad1 33 137 148 168
Sad1-4 110
Sad1-6 110
sat-Pororo 44 135
sen1 154
sen2 162
sen3 150
sen5 152
sen6 158
Sh1 136
sh1 11 20 33 40 58 59 60 61 69 71 75 108 118 129 137 155 166
sh1-5582 136
sh1-5586 136
sh1-5933 136
sh1-857011 3 137
sh1-857015 3 137
Sh1-A 136
Sh1-B 136
sh1-bz1-82g760 5 137
sh1-bz1-825211x 5 137
sh1-bz1-825211y-1 5 137
sh1-bz1-825212x-1 5 137
sh1-bz1-825215z 5 137
sh1-m5933 136
sh1-Mu 17
Sh1-r5 136 136
sh2 7 9 19 33 53 56 71
sh4 71 158
sh*-627D 46 135 147
sh*-912 46 135 147
sh*-1053B 46 138
sh*-1322A 46 138
sh*-1324A 46 138
sh*-1339A 46 135 146
shp*-1749 56 138
si1 71 160
sin1 71 138
sk1 71 129 130 152
Sks1 153
sl1 71 162
sm1 33 71 160
smk*-1373A 46 138
smk*-1437 46 138
Sn1-bo13 137
Sn1-s 137
Sn1-w 137
Spc1 155
Spm 2 19 41 42 134 135 147
Spm8 136
Spm11 136
Spm13 136
Spm-I2 42 135
Spm-I8 134 136 137
Spm-P 115 135
Spm-s 18
Spm-S 129
Spm-s 139
Spm-w 18 139
sr1 51 134 150
sr2 27 137 169
sr3 168
Ss2 58 59 60 61 137 148 166
st1 156
su1 33 71 74 117 118 132 135 156
su1-4059 13 135
su1-4582 13 135
su2 33 71 110 160
Sup1 71
Sw*-1 137
Sw*-2 137
T1-2c 49 134
T1-9(4995) 116 146
T1-9(8389) 116 146
T1-9c 116
T2-9(5257) 73
T2-9b 116
T2-9d 116
T3-4(5156) 30
T3-9c 30 116
T4-9(5657) 114 116
T4-9b 114 116
T4-9e 73
T4-9g 114 116
T5-9(4817) 73
T5-9a 116
T5-9c 116 135
T6-9a 116
T6-9b 116 117
T7-9(4363) 116 136
T7-9a 73 116
T7-9c(4363) 52
T7-9g 116
T7c-9(4363) 52
T8-9(6673) 55 73 112 116
T8-9d 55 116
T9-10b 116
tb1 71 151
TB-1La 50 150
TB-1La-3L4759-3 151
TB-1La-3L5242 151
TB-1La-3L5267 151 155
TB-1La-3Le 151
TB-1La-4L4692 151 156
TB-1La-5S8041 151 158
TB-1Lc 43 134 150
TB-1Sb 43 49 50 100 134 134 150
TB-1Sb-2L4464 150 152
TB-1Sb-2Lc 49 134 150 153
TB-3La 7 43 50 54 107 113 135 149 154
TB-3La-2L7285 152
TB-3La-2S6270 149 152 155
TB-3Lc 50 54 135 147 155
TB-3Ld 43 154
TB-3Lf 113 135 149 154
TB-3Lg 113 135 149 154
TB-3Lh 154
TB-3Li 155
TB-3Lj 155
TB-3Lk 155
TB-3Ll 155
TB-3Lm 155
TB-3Sb 6 30 43 50 107 154
TB-4Lb 156
TB-4Lc 43 50 135 147 156
TB-4Ld 43 157
TB-4Le 43 157
TB-4Lf 43 50 135 147 156
TB-4Sa 43 50 135 156
TB-5La 135 158
TB-5Lb 43 158
TB-5Ld 158
TB-5Sc 44 50 135 147 149 158
TB-6Lb 43
Tb-6Lb 50
TB-6Lb 135 147 160
TB-6Lc 43 50 110 160
TB-6Sa 43 50 107 160
TB-7Lb 43 50 54 136 147 162
TB-7Lb-4L4698 157 162
TB-7Sc 44 162
TB-8La 43 50 106 107 112 136 149 164
TB-8Lb 164
TB-8Lc 43 46 50 110 136 148 164
TB-9La 12 50 107 166
TB-9Lc 44 50 136 137 148 166
TB-9S 107
TB-9Sb 10 11 43 50 67 68 137 166
TB-9Sb1866 68
TB-9Sb-4L6222 156 166
TB-9Sb-4L6502 156 166
TB-9Sd 43 50 137 148 166
TB-10L1 168
TB-10L2 168
TB-10L3 168
TB-10L4 168
TB-10L5 168
TB-10L6 168
TB-10L7 168
TB-10L8 168
TB-10L9 43 168
TB-10L10 168
TB-10L11 168
TB-10L12 168
TB-10L13 168
TB-10L14 168
TB-10L15 168
TB-10L16 168
TB-10L17 168
TB-10L18 50 168
TB-10L19 43 50 54 137 148 168
TB-10L20 43 168
TB-10L21 168
TB-10L22 168
TB-10L23 168
TB-10L24 168
TB-10L25 168
TB-10L26 168
TB-10L27 168
TB-10L28 168
TB-10L29 168
TB-10L30 50 54 137 148 168
TB-10L31 168
TB-10L32 50 137 148 168
TB-10L33 168
TB-10L34 168
TB-10L35 168
TB-10L36 168
TB-10L37 168
TB-10L38 168
TB-10La 50 107 168
TB-10Lb 50 168
TB-10Sc 43 50 137 148 168
td1 71 158
te1 154
tea1 71 138

Tp1 71 162
Tp2 71 168
Tpi3 33 136 148 164
Tpi3-2 110
Tpi3-4 110
Tpi4 33 112 135 147 149 154
ts1 71 130 149 152
ts2 36 71 129 130 150
Ts3 71
ts4 71 130 154
ts5 71
Ts5 156
Ts6 71 109 151
tts1 71 138
Tu1 71 157
Tz86 136
Uq 4 5
v1 12 56 166
v2 159
v3 158
v4 45 134 153
v5 52 136 162
v8 157
v12 158
v16 112 134 136 148 164
v17 156
v18 168
v21 164
v22 151
v23 156
v*-350 134
v*-424 45 134
v*-576 44 138
v*-588 134
va1 162
Vg1 71 151
vp1 54 154
vp1-m451 4
vp1-Mum1 7 135
vp2 158
vp5 150
vp8 151
vp9 56 162
w1 160
w2 27 54 137 168
w3 45 56 134 153
w11 166
w14 160
w15 160
w16 162
w17 162
w*-270A 56
Wc 44
Wc1 71 167
wd1 10 11 50 57 166
whp1 153
wi1 160
wl*-266A 146
wl*-709B 146
Wlf*-1726 56 138
ws3 152
wsp 15 69
wt1 49 51 57 134 152
wx1 2 8 11 13 14 30 33 34 43 52 55 60 69 71 90 100 112 114 116 118 135 136 146 147 166
Wx1-9r1 136
wx1-85-1 3 137
wx1-90 137
wx1-844 136 137
wx1-857027 3 137
wx1-B 137
wx1-B1 137
wx1-B2 137
wx1-B5 137
wx1-B6 137
wx1-B7 137
wx1-B8 137
wx1-BL2 137
wx1-c 137
wx1-C1 137
wx1-C2 137
wx1-C3 137
wx1-C4 137
wx1-C31 137
wx1-C34 137
wx1-G 137
wx1-I 137
wx1-K 137
wx1-M 137
wx1-m7 40 136
wx1-m8 19 129 136 137 147
wx1-m9 20 40 136
wx1-Mum1 7 137
wx1-Mum2 7 137
wx1-Mum3 8 137
wx1-Mum4 8 137
wx1-R 137
wx1-Stoner 137
wyg1 162
y1 8 13 14 71 110 113 118 136
Y1 160
Y3 152
y8 162
y9 10 168
y10 155
yd2 155
yg1 159
yg2 10 11 166
ys1 71 158
ys2 150
ys3 154
Ysk1 156
zb3 159
zb4 150
zb6 156
Zer1 33
Zer3 33
zn1 71 168
Zp1* 136
Zp2* 136 137
Zp3* 136
Zp6* 114 136
Zp6h* 135
Zp10/20-16* 136
Zp10/(22)* 135
Zp12* 135
Zp12/19* 135
Zp12/29* 135
Zp14* 135
Zp15* 135
Zp16* 136
Zp18/15* 136
Zp18/18* 136
Zp20* 136
Zp20/1* 136
Zp20/2* 136
Zp20/3* 136
Zp20/6 136
Zp20/16* 136
Zp20/20-16* 136
Zp20/22* 136
Zp20/27* 135
Zp20/28* 135
Zp20/29* 135 136
Zp20/30* 135
Zp22* 135
Zp22/3* 137
Zp22/6* 114 135 135
Zp22/10* 135
Zp22/12* 135
Zp22/14* 135
Zp22/15* 135
Zp22/22* 135
Zp22h* 135
Zp23/16* 135
Zp27* 135
Zp28* 135
Zp29* 135 136
Zp30* 135
zpg1 137
Zpr10/22* 114
Zpr10/(22)* 135

Abasi L r1
Abbott A G r2 r154 r238
Adams S r123
Adegov A V r189
Agarwal K N r437 r480
Aguiar-Perecin M L R de 134 135 136 r3 r4 r5 r6
Ahuja S C r7
Akhter S A r8
Akin D E r10
Albergoni F G r9
Albertsen M C 146 r582
Aldrich H C r10
Alexander D E 46 64 r169
Alexander W L r95
Alexandrescu V r11
Alfermann 66
Alleman M 107 134 138 r12
Alofe C O r1
Altman L K r13
Altschuler M V 99
Alvarez A 21 22
Andeew 86
Anderson E L r14
Anderson I C r456
Anderson L K r15
Andrade F H r456
Appels R 58 71
Apple A E r163
Arboleda-R F r257 r564
Argandona V H r16
Arihara J r17
Armengol M r313
Armstrong C L r18
Arturi M J 77
Ashburner 98
Ashworth D J r144
Asnani V L r450 r451
Athma P 129
Atkinson B G 91 r37
Aulicino M B 82
Avato P r19 r45
Ayers J E r20
Babinec F J 77
Baca F r407
Backer J S r21
Badaev N S r53
Badr E r499
Baer G R 139 r22 r23 r24 r25
Baker A 138 r26
Baker B J 42 43 r309
Balasubramanian K A r503
Ball Y 1
Bandyopadhyay S r401
Banks J A 19 20 r487
Barker R F 120
Barla-Szabo G r27
Barlow P W r28 r29
Barnabas B r30 r31
Barnes Taylor D r311
Barriere Y r32
Barrow M R r33
Barry B D r34 r199 r377
Bartkowiak E r35 r36
Bartkowiak S r281
Basso B r9
Baszczynski C L 90 91 92 93 101 102 139 r37
Batta R K r114
Baulcombe D r154
Beadle G W 105
Beckett J B 43 44 47 50 55 95 106 113 135 137 146 149 r273
Beckman J S r70
Bedbrook J R 128
Bedeer A H r137
Bedinger P 101
Beggs C J r38
Bell E r296
Belousov A A r39
Below F E r201 r506
Bendich A J r461
Benner M S 114 135
Bennett D C r160
Bennett M D 32 33 78 139 r40
Bennetzen J L 9 108 120 136 r41 r42
Bergquist R R r547 r548
Bernard S r43
Berndtgen R r494
Bertani A r559
Bertoia L M 81 87 88 139
Bertram I r380 r408
Berzonsky W A r219
Bettendorf A R 26
Bhole G R r44
Bianchi A 18 r45 r46
Bianchi G r19 r45
Bietz J A r47 r141
Binelli G 99
Birchler J A r48
Bird R M 44 57 81 135 137 138 r49 r202
Birky C W Jr r21
Blanco M 21 51
Block L G 117
Bochicchio A r50
Bockholt A J r555
Boer P H r54
Bogorad L r153 r460 r535 r607
Bohm H r51 r52
Bohnert H-J r96
Boiko E V r53
Bommineni V R 95
Bonanomi S 114 r215
Bonas U r474
Bonen L r54
Bonhomme R r108
Bonig I r537
Boothe J G 94
Borner T 26 r55
Boston R S r56
Botez C r394
Bouchard R A 72 73 139
Bouchet B r57
Boutry M 97 r58
Boyer C D r59 r135
Boyer J S r382
Bradbeer J W 25 r464
Bradford 97
Bradford J A r92
Branson T F r259
Braun C J 111 138 r60
Brazil M 1
Brembilla M r477
Bremenkamp M r477
Brenner M 38
Brennicke A 170
Brettell R I S 63
Brewbaker J L 136 137 r61
Briggs R W 26
Brink D E r110
Brink R A 36 40
Briquet M r58
Broccoli A M 84
Brodskii V Y r53
Brodsky 62
Brower D 24
Brown 112
Brown J 58
Brown P T H 41
Brown W L 105 r62 r63 r64 r65 r66
Browne C 1
Bruneau R r67
Buckley P M r591
Bullard R W r68
Burnham C R 46 109 117
Burr B 136 138 r69 r70
Burr F A r70
Buske M C r71
Bussard J B 48 r291 r181
Butler L G r117
Cabulea I r394
Cairo G r572
Calvert O S 50

Campbell W H 26
Cao Z r72
Carey E E r73
Carlson J E 101 128 138 r74
Carlson W R 68 69 146 148
Casadoro G r447
Case J S 30
Cavalieri A J r75
Celarier R P 131
Cesar C L r299
Chaganti R S K 78
Chalyk S T r76
Chandlee J M 33 r77
Chandler J 104 140
Chandler V 18 108 120 r576
Chandravadana P 131
Chang M T 1 45 46 47 50 55 64 134 135 136 137 138 149 r429
Chang T T r78 r79 r80 r81 r82
Channon P r338
Chao S 170
Chappell K 1
Charbonnier M r58
Chase C D r83
Chase M r290
Chen C r84 r85 r301
Chen C-H 25 134
Chen J 40
Chen Y-H r85
Cheng D S-K 25
Chiang M S r237
Chigvinadze T D r300
Chomet P S r104 r105
Chou T-S 68
Chourey P S 58 60 63 r136 r217 r594
Christianson M 124
Christie B R r229 r230
Chua N-H r213 r359
Chuanshan Z r15
Chung S H r86
Clark J K 64
Clark R L r87
Clarke A E r537
Clowes F A L r103
Cobb B G 39
Coe E H Jr 1 23 25 36 54 55 67 104 107 134 136 140 146 148 r88 r181
Cohen B N r89
Cohen C E r110
Coleman T A r89
Collins G B 89
Collins M D r312
Colvin T S r207
Combs G F Jr r90
Combs S B r90
Conde M F 63
Cook W 48 138
Cooper P 90 99
Corcuera L J r16
Corfman R S r321
Cornu A r109
Cortadas J r432
Cortezmendoza H r385
Courage U 136 r91 r533
Coyne P I r92
Crane P L 97 146
Crosbie T M r227 r355
Cross H Z r93 r94 r95
Crossland L D r535
Crouse E J r96
Crowe T G 93
Cruz C D r97
Cuevas R r98
Cullis C A r577
Curtis C A 68 107
Cuypers H r495
Czarnecka 72 98
Czuchajowska Z r430
Dale R M K 138 170 r99
Dana S r435
Dankov T r100
Danna K J r234
Darlington C D 77
Darrah L L r34 r345 r377
Das V 78
Dash S 104
daSilva W J r101 r299
Davis C 51
Davis D W r442
Davis F M r370 r592
Dawid 62
Dawson A J 170
Debnath S C r102
Delatorre C r103
Dellaporta S L 40 136 r104 r105
Deltour R r194
Dempsey E 5 27 28 136 r106 r455
Dennis E S 63 111 r107 r234 r235 r403 r404 r471
Derieux M r108 r109
Desouza I R P r308
Devine O J r258
deWet J M J r110
Dewey R E 63 111 138 r111 r112
Dey S K 96
Dhawan N L 104
Dhillon B S 96 r113 r114 r265 r268 r513 r514 r515
Dhillon M r265
Dhingra H R r115 r116
Dickerson D P r117
Dickey K r348
Dickinson D B r73
Diedrick T 115
DiFonzo N 100 114 r179 r215 r356 r452 r477
DiNocera 62
Distanova E E 107 149
Dobberstein B r527
Doebley J F 81 134 135 136 137 r118 r119
Doerschug E B 18
Dolfini S F 100
Dolgikh Yu R r424
Dolinka B r27
Dollinger E J 133 135 137 r120
Doncheva S r240 r241
Dooner H K 42 109 136 r121 r122 r123
Doring H-P 136 r91 r124 r125 r533
Dougherty W G r395 r396
Dowd P A r423
Doyle G G 1 54
Draganic M r407
Drobney V K r312
Drozdov S N r551
Duburcq J B r108
Dudley J W r126 r127 r585
Duke S H 25
Duke S O 25 r128 r129
Dunbar S 170
Duncan D R r130
Duncan W G r131
Dunlop J D 90
Dunn M E r132
Duvick D N 15 70 r133 r134
Duysen M r142
Dvorak J 58
Dybas L r142
Dybing C D r258
Dyer T A 170
Earle E D 69 r517 r578
Echeverria E r135
Echt C S 1 49 50 57 134 r136
Eckes P 25 42
Edwards J r375
Edwards M 120
Eisenberg A r593
Elhattab A H r137
Ellis J r471
Elmore-Stamper S 111 138
Elsahookie M M r138
England D 148

Engleman E M r566
English J r123
Enomoto S r413
Erion J L r139
Escote L J 69 113 126 128 129 138
Esechie H A r140
Esen A r141
Eskins K r142
Eubanks M 103 r143
Evans D A r523
Evans N A r537
Everett H L 96 r517
Everett N P r144
Evert R F r465
Evola S V r70
Ezra G r145
Faber A-M r58
Fakorede M A B r1
Faktor V M r53
Farina M P r338
Fauron C M-R 170 r306
Fausey N R r146 r565
Favreau M r123
Fedoroff N V 20 36 42 43 136 139 r147 r148 r487 r488
Feix G 58
Fenczik C 24
Ferl R J 62 134 r10 r149 r150 r151
Ferrari L 88
Figueira E r98
Filho J B M r180
Filichkin S A r424
Filipas T B r424
Findley W R r152
Finnegan 112
Finnegan E J r107 r471
Fish L E 138 r153
Fitzgerald P J r259
Flavell R B r2 r154 r238
Fletcher G B 1 104 106 140 146
Foley D C r87
Fong F 39 r352
Fontanet P 21 22
Ford R E 134 137 r155
Forde B G 97 138 r156 r280
Forde J r280
Fortnum B A r157
Fotos Kadar K r362
Fox M H r15
Fox T D 170
Fraley R T r501
Francis D 33
Frankovskaya M T r562
Frascaroli E 29
Freeling M 24 25 107 134 137 146 r12 r158 r159 r160 r161 r202 r203 r204 r253
Fridvalszky L r31
Friedman J r101
Frisch D A 115 138
Fromm M r162
Frommer W-B r91 r533 r584
Frova C 99
Fry W E r163
Funnemark W L r164
Furtek D 20
Furtek D B r487 r147 r148
Fussell C P r165
Fusswinkel H 39
Gabay-Laughnan S 69 112 113 128 129 r266
Galinat W C 130 131 132 133 146 r166 r167 r168
Gallant D J r57
Galliher H L 139 r169
Gama E E G e r170 r307 r308
Gao Y r236
Garber R r125
Gardner C O 73 74
Garfield E r171
Gaskin P r226
Gavazzi G A 100 137 r172 r173 r174
Gay J-P r175
Geadelmann J L r176
Geisler G r532
Gen G G r11
Gengenbach B G 18 64 100 115 126 138 r247
Genova I r177 r178
Genovesi A D 89
Gentinetta E r179 r452
Geraldi I O 139 r180 r525
Gerats A G M r173 r181
Gerlach W L 24 r107 r403 r404 r471
Gheith E M S r137
Ghosh P D r401 r402
Giannini A r182
Gibbs C P 91
Gierl A 2 42 134 136 r183 r184 r408 r474 r476 r494 r495
Gigot C r571
Gillies C B 52 r185
Gillis M C 43
Ginsburg H 170
Gobel E 41 r309
Gold J R r448
Goldberg R 112
Goldsbrough P B r56
Golubovskaya I N 71 107 135 136 149 r186 r187
Gontarovsky V A 135 r188 r189
Goodman M M 33 44 110 146 r119 r190 r520 r521 r522 r540
Gorchakova N S r310
Gorman 62
Gorya V S r310
Gould F r570
Gould S J r191
Gozdzicka-Jozefiak A r538
Gracen V E 69 112 r517
Graebe J E r226
Graham F L 62
Gray M W r54
Green B R r192
Green C E 64 74 75 100 115 115 r18
Greenblatt I M 125 r193
Greimers R r194
Greyson R I 89 90 95
Grieder A r578
Grienenberger J-M r326
Griffiths A J F r406
Grogan C O 55
Grontkowski C R r195
Grossmann K r373
Groth J V r442
Gu M G 37 r72 r196 r197 r236 r606
Gueldner R C r198
Guillemaut P r326
Gullons A 92
Gunn R E r154
Guo C r72
Gupta D 105
Guthrie W D 139 r199 r200
Gynkull T N r276
Hageman R H r201 r506
Hagerman A E r117
Hake S 24 134 138 r161 r202 r203 r204
Hall A J r472 r473
Hallauer A R r164 r205 r206 r207 r289 r385
Hamilton J L r208 r209
Hammond R W r210
Han C-d 1 55
Handa S r211 r212
Hanley-Bowdoin L r213
Hannah L C 60 61
Haque M N r8
Harlan J R r214
Harpster M H 25
Harris J W 30

Harris K F r555
Harrison B J r474
Hastings H 114 135 136 137 r215
Hawes C R r216
Hawes M C r217
Hawk J A r218 r219
Heckman J E r239
Hedden P r226
Hehl R 138 r220
Heidt A R r198
Helentjaris T 61 120 134 135 136 137 138 139 148 r221
Henikoff 111
Henry S r504
Herich R r222
Hermodson M A r321
Hernandez-X E 29 r223
Herrera-Estrella L 25
Herrmann R 16
Heslop-Harrison J r224 r225
Heslop-Harrison Y r224 r225
Heupel R C r226
Heyworth A r156
Hibberd K A 115
Hicks J B r105
Hightower R C r336
Hill R R Jr r20
Hils M H r10
Hinz P N r375
Hirayama 37
Hironaka C M r501
Hirota A r578
Hizume M 99
Ho T-H D 90
Hoagland R E r129
Hoard K G 139 r227
Hodge T P 170 r306
Hodgins R r228
Hoekstra G J r229 r230
Hohn B 111
Hoisington D A 1 49 50 51 52 57 134 136 137 138 146 147 149 r367
Hole D J 39
Hooker A L r231
Horne J C r216
Hoson T r232
Houchins J P r99
Hougas R W r233
Howard E A r234 r235
Howe C J 170
Howley P M 91
Hu L r72
Hu W W L 170
Hu X r344
Huang A H C r580
Huang D N r196
Huang D r236
Huber D M r211 r212
Hudon M r237
Hudson W A 43
Huelsen W A 43
Hung D r72
Hunt M 47
Hussain M r502
Hutchinson J r238
Iams K P 138 r239
Ignatov G r240 r241
Iken J E r242
Iltis H H 81
Innes R W r420
Inoue Y 126 r243
Introzzi F r244
Isaac P G 138 170 r245
Isenhour D J r246
Ishige T 126 127 138 r247
Isola N R 63
Ivanovic M r248
Ivaschenko 95
Jackson W A r390 r391 r392
Jain H K 105
Jarvis J L r199 r200
Jatimliansky J R 77
Jayos-Rios E r249
Jeannin G r326
Jelenic D r541
Jellum M D r250
Jenns A E r251 r252
Jessen H 115
Jewell D C r43
Johns M A 20 137 139 r253 r147
Johnson A 112
Johnson M I r254
Johnson M W Jr r20
Johri M M 23
Jones B r321
Jones D A r332
Jones D F 113
Jones M D r462
Jones R J r255 r511
Jones V P 170 r245
Joshi G S S r515
Jovicevic B r256
Jung J r373
Jupe E R 23 135
Jurado-Z O r257
K'Drev T r240 r241
Kahler A L 31 109 135 146 r258 r259
Kalinnikov D D r260
Kamprath E J r14 r392
Kannenberg L W r229 r230 r261
Kapoor W R r114
Karen R r262
Karssen 38
Karukstis K K r192
Kato-Y T A 29 37 r263 r566
Kaul M L H r264
Kaulen H 42
Kaur G P r265
Kazymova E M r409
Kelley P M r204
Kelly S r147
Kemble R J 101 102 114 126 127 128 r74 r154 r266 r542
Kennedy A E 132
Kennedy D 1
Kennedy G G r327
Kermicle J L 51 55 128 137 146 r267
Kern H r583
Kessler E 25
Khachidze O T r300
Khehra A S 96 r113 r114 r268 r513 r514
Kheyr-Pour A 112
Khotyleva L V r269
Khristolyubova N B r186 r187
Khristov K r270 r272
Khristova I r271
Khristova P r270 r272
Kidd A D 33
Kidwell K K 129 138
Kieft H r486
Kindiger B 1 43 44 48 50 95 135 137 138 r273
King G r221
Kingsbury J 19 20
Kleese R A 30
Klein B 40
Klein R R r274
Kleinhofs A 26 r275
Kloeckener B 24
Klosgen R B r474
Knievel D P r431
Knoke J K r152
Koeppe D E r274
Koinuma K 126
Kolodner R 16
Komarova G E r276 r277
Kommedahl T r334
Korobko O I r278
Kossel H r538
Kovacs 86
Kovacs G r342
Kowalewski S 1

Kowles R V r279 r413
Kramer H 109
Krause E r527
Krause G F r345
Krebbers E r474
Kreis M r156 r280
Kremer D A r601
Kreuzaler F 42
Kridl J C 170
Krivi G G r501
Kriz A L 93
Krolikowski Z r281
Ku 125
Ku M-K 74
Kuck U r153
Kumar D 139 r282 r283 r284
Kung S D r607
Kunze R 39 39 139 r91 r533
Kuo C-S r285
Laemmli U K 98
Lai F S r430
Laird A 40 r91 r533
Lal S r286
Lamb B C r287
Lambert R J 146 r288 r506 r585
Lamkey K R 139 r289
Lancaster H r290
Landi P 29
Landry J 88
Larkins B A r56
Larkins J R r561
Larson R L 48 49 r291 r181
Larter E N r339
Laughnan J R 69 112 113 128 129 r266
Laughner B J 62
Laurie D A 32
Lay M-M r292
Leaver C J 97 170 r26 r245
Lechelt C 40 134
Lee L r293
Leford D R 139 r294 r295
Legrenzi A 99
Lehman P 90
Leonard K J r251 r252
Leto K J r296
Levic J r407
Levings C S III 63 111 113 126 127 170 r60 r111 r112 r297 r386 r395 r396 r498 r517
Li J G r302
Lidansky T r271 r553
Liebenberg N V D W r544
Liebmann F 29
Lillis M r298
Lima C A S r299
Lima M B S r299
Lin B-Y 46 50 54 136 137 147
Lin Y-H r580
Liparteliani O G r300
Liu E H r61
Liu K-C r59 r135
Liu K-S r84 r301
Liu L r524
Liu Y N r302
Llewellyn D r403 r471
Lobov V P r303
Loerz H r403
Loi N r383
Lonsdale D M 63 111 128 138 170 r304 r305 r306 r433 r484 r485
Lopatina L M r388
Lopes M A r307 r308
Lorenzoni C r383
Lorz H 41 43 r309
Loseva Z I r310
Louie R r152
Loussaert D F 114
Lowe K r311
Lower W R r312
Lu C-Y 64 r567
Lu W-L r285
Ludevid M D r313
Luk'Yanova N L r278
Lukina L A r314
Luo Y r344
Luthe 91
Lysikov V N r76
Lyznik L r315
Ma 100
Maas C 41 r584
MacDonald F D r316
Mackenzie A 92
MacMillan J r226
MacNeish R S r317
Magill C W 39
Magnavaca R r170 r307
Magoja J L 81 82 83 84 85 86 87 88
Maguire M P 18 r318 r319 r320
Mahoney W C 139 r321
Major D J r322 r462
Malan C r323 r324 r325
Mandugano L r353
Mangelsdorf P C 34 88 105 118 131
Mans R J 126 127 r579
Manwiller A r157
Manzocchi L 100 r477
Marechal L 138 r326
Margolies D C r327
Markova M r328
Marotta R r356 r477
Marrero T R r312
Marshall S 96
Martin B 42 135
Martin C R r430
Martin W K r588
Martineau B r329 r492 r546
Martinez-Izquierdo J A r313
Martiniello P r330 r331
Marwaha K K r481 r508
Maryam B r332
Mascarenhas J P 99 r534 r593
Mascia P N 114 136 146
Mashingaidze K r333
Mashnenkov A S 71 138
Masterson R V 16
Mathur D S 104
Matorin 25
Matyac C A r334
Mayfield S P 25 r546
Mazoti L B 77 137
McCarthy S r142
McCarty D R 60 61 137
McClelen C E r349 r350
McClintock B 2 11 12 18 19 20 24 36 40 42 44 55 78 115
McClure B A 114 r99 r414
McCormick S M 60 146
McDaniel 97
McDonald M B Jr r146 r565
McElfresh K C 17 139
McGarth J P 91
McIntosh L r296
McKeown M 73
McKusick V A 34
McMillian W W r335 r598 r599
McMullen M D r413
McNay J W r517
McWilliams W W r587
Meagher R B r336
Medeiros C A B r360
Meinke L J r570
Meliya N S r337
Mendel R R 26
Mendes P r338
Mendoza M r353
Mendu N 170
Menkir A r339
Merckelbach A 40 134 r91 r533
Messing J r148 r374
Messmer M J r506
Metz J G 16 r341
Michael G r608 r609
Micke A 70

Miflin B J r280
Mikel M A r155
Mikerezi I r172 r173
Miku V E r277
Milas S r340
Miles C D 48 134 135 136
Miles D r341
Milinko I r342
Miller J A r343
Miranda L E C de 34 35
Miranda L T de 34 35 139
Mladenova I I r536
Mo H r344
Mochizuki N 126
Mock J J r355
Moentono M D 139 r345
Molina M del C 79
Moll R H r14 r390 r391 r392
Monotti M r483
Montanelli C r477
Montes G r464
Moore R r346 r347 r348 r349 r350 r351 r352 r371
Moreno M L r103
Moreno-Martinez E r353 r354
Morris D W 16 r458
Mosely P R r355
Mottinger J P r105 r253
Motto M 136 r244 r356 r477
Moureaux T 88
Moussa M r499
Mozer T J r336 r501
Muchena S C r333
Mukherjee B K r357
Mulcahy D L 28 99
Muller A 25
Muller E r358
Muller-Neumann M 39 40 42 139 r91 r533
Mullet J E 138 r359
Mundstock C M r360
Murashige T 75
Murphy T 118
Murray K 40
Murthy T G K r264
Nagai C r61
Nagy L r361
Nagy M r362
Nanda D K r363
Naranjo C A 79
Naspolini V r170
Nasyrova G F r276
Nebiolo C M 91 98 102 103
Nelsen B r125
Nelson O E Jr 7 19 43 100 146 r384 r487 r488 r364 r147 r148
Nemenyi M r365
Netzer W J r366
Neudachin V P r424
Neuffer M G 18 19 44 46 50 54 55 56 57 64 138 146 148 r49 r202 r367 r429
Nevers P r474 r475
Newton K J 33 91 r368 r369 r381
Ng S-S r370
Ng Y-K r371
Nicholson R L r117
Nick H S 62
Nickerson N H 97
Nielsen G r372
Niesbach-Klosgen U 42
Niland A M r292
Nitsche K r373
Nivio A A 84
Norrander J M r374
Norton D C r375
Notani N K r376
Nowick E M 2 117
Nowicki K D 98
Nuffer M G 40
Ochieng J A W r377
ODell M r2 r238
Ohad I r492
Oishi K 25
Olness A E r258
Onukogu F A r378
Openshaw S J r379 r363
O'Reilly C 42 108 134 r380
Oro A E r381
Orozco E M Jr r213 r359
Ort D R r382
Ortiz D 17
Osler R r383
Osterman J C 73
Ottaviano E 28 99
Ouattar S r255
Owen R D r384
Oyervidesgarcia M r385
Pagliarini M S r5
Paillard M 138 r386
Pajic Z r387
Pakudin V Z r388
Palacios I G 81 85 86
Palau J r313 r432
Palfi G r389
Palfi Z r389
Palii A F r276
Palladina T A r276
Palmer J D 111
Pan W L r390 r391 r392
Pan Y-B 6
Pandey K K r393
Panfil C r394
Papinutti P r172
Pardee A B 91
Pareddy D R 89
Parker 62
Parks T D 138 r395 r396
Parlov D r397
Pascholati S F r117
Pasupuleti C V 133 136
Pataky J K r398
Paterniani E r399
Paterson K E r311
Patil R C r44
Patterson E B 146 146
Pauk J r400
Paul A-L 62 134
Paul N K r401 r402
Paulis J W r141
Payak M M 95 r7
Paz-Ares J 42 137
Peacock W J 134 139 r107 r234 r403 r404 r471
Pedersen W L r405
Peeters J P r406
Pencic V r407
Penny L H r259
Pereira A 42 137 r183 r380 r408
Pereverzev D S r409
Perkins J M r405
Peruansky Yu V r410
Perucci P r483
Peschke V M 18 117
Peterson P A 2 3 4 5 6 40 42 115 134 135 136 137 r46 r183 r380 r408 r411 r449 r474 r495
Peterson T A 36 37
Pharis R P r462
Phillips R L 18 30 44 114 115 117 135 146 r279 r412 r413 r414
Phinney B O 30 r226 r415 r416 r417 r418 r419 r489
Pinter L r389
Pischedda G 83
Pizzolato T D r219
Plata M I r217
Plazinski J r420
Plewa M J r421 r422 r423 r497
Plotnikov V K r424
Pochmelnych G A r425
Poethig R S 109 135 146 r426 r427
Pogna N r573
Pohlman R F 20 39 40 r148

Polacco J C r428
Polacco M L 16 45 46 134 138 146 147 r429
Polonetskaya L M r269
Pomeranz Y r430
Ponziani G r179 r477
Porter G A r431
Porter J W 7
Potrykus I 66
Powers D D r321
Prasad S K r8
Prasad S S r8
Prat S 139 r432
Preiss J r316 r512
Price H J 32 r448
Pring D R 64 114 r83 r433 r484 r485 r517
Prioli L M r299 r434 r523
Privitera E r573
Prodhan H S r435
Pryor A J 134 137 r403 r436
Pueppke S G r217
Puigdomenech P r313 r432
Pywell J r156
Qadri M I r437 r480
Quang V D r438
Quatrano 91
Raboy V 19 r487 r488
Racca E r98
Racchi M L 100 134 r173 r439
Raczynska-Bojanowska K r315
Radic L r440
Radojevic L r441
Radosavljevic M r387
Rafalski A r315
Ralston E 109 r123
Ramirez J r354
Randall D 49
Randle W M r442
Randolph L F 77
Ranhotra G S r443
Rao K V 65 66 67
Rapela M A 75 r444 r445 r446
Rascio N r447
Ray L r57
Ray N 49
Rayburn A L r448
Ream 86
Reddy A R 42
Reddy G M 65 66 67
Reddy L V 134 r449
Reddy P R r450 r451
Reeck G R r321
Reed J E r363
Rees C A B 91 92 101
Refatti E r383
Reger B J r224 r225
Reggiani R r452
Renaudin S r454
Renfro B L r453
Restaino F 18
Rey L r454
Rhoades M M 5 27 28 47 68 131 137 r455
Rhodes A M r73
Riegel J r91
Rienhard 66
Ritzel M 30
Robbins J C r200
Roberts J K M r456
Robertson D S 4 8 9 10 11 12 14 15 16 17 24 43 47 67 68 107 134 135 137 r380 r457 r458
Robinson J B D r459
Rocheford T R 73 74 136 139
Rochester D E r501
Rockar R 98
Rockwell B H 90 91
Rodermel S R 138 r460
Roessler J r255
Rogers S O r461
Rolfe B G r420
Roman H L 50 106
Rood S B r462
Rosahl S 42
Rosales T P 78
Rosic K r248
Rossman E C r199
Rotar A I r277
Rottmann W L r518
Rowland L J 17 134 r463
Rubenstein I 58 r374 r413 r518
Ruddle F H 34
Ruffer-Turner M E r464
Russell S H r465
Russell W A 139 r200 r294 r295 r466 r467 r468 r469
Russell W K r470
Ryadchikov V G r424
Ryan F J r210
Ryan P r311
Sachan J K S 104 106
Sachs M M r107 r403 r404 r471
Sackitey S 8 137
Sadras V O r472 r473
Saedler H 42 137 r183 r184 r220 r380 r408 r474 r475 r476 r494 r495 r496
Safonova V T r187
Saha B C r357
Sala E r572 r573
Salamini F 18 20 114 136 r19 r45 r179 r215 r356 r452 r477 r560
Salvo R r478
Samuels G J r358
Sanghi A K r437 r479 r480
Sanguineti M C r174
Sarkar K R 104 106 r102 r282 r283 r284 r435
Sarkissian 97
Sarup P r481 r508
Sastry 88
Sathyanarayana V 67
Sauer K r192
Sauerbrey E r373
Savic N r482
Savin V N r410
Saxena V K 96 r113 r114
Scandalios J G 33 r77
Scarponi L r483
Schaalje G B r322
Schaller H 39
Schardl C L 127 138 r484 r485
Scheffler B E 3 4
Schel J H N r486
Schell J 42 43 r309
Schiefelbein J W 19 20 137 r147 r487 r488
Schiemann J 26
Schlichter T M r472 r473
Schmidt N C 35
Schmitt J J r89
Schmitt J M r96
Schnable P S 4 5 137
Schneider G r489
Schnell F W r490
Schrader L E r22 r23 r24 r25 r491
Schussler L r544
Schuster A M 112 r112
Schuster G r492
Schuster W r51 r52
Schwartz D 37 40 r403 r404 r493
Schwarz-Sommer Z 20 42 134 137 r183 r184 r380 r408 r474 r476 r494 r495 r496
Schy W E r497
Sclyar Yu V r189
Scott G E 55
Scott N S 16
Sederoff R R 170 r386 r498
Sediyama C S r97
Seehy M A r499
Sehgal S M r500
Seif R D r568
Sembdner G r489

Shaban S A r137
Shadley J D 146
Shah D M 139 r336 r501
Shakoor A r502
Shamina Z B r438
Shankerlingam S r503
Shannon J C r431 r135
Shapiro L r504
Sharma R C 95
Sharpe D Z 63 r217
Shaver D L 118 r588
Shaw 62
Shaw J R 60 61
Shcherbak V S r505 r603
Sheldon E 58
Shepherd N S r220 r380
Sheridan W F 64 131 135 136 137 146
Sherrard J H r506
Sherudilo E G r551
Shevardnadze G A r507
Shevtsova V V r260
Shewry P R r280
Shimabukuro R H 55
Shumny V K r425
Shure M 18 42 r147 r148
Shurtleff M C 95
Siddiqui K H r481 r508
Siedenstrang C r221
Siemenroth A 25
Silva J C r97
Silva W J r434 r523
Sim W S r86
Simcox K D 135 r509
Simeonov N r510
Simmons S R r511
Simpson R B r298
Sinclair J H r239
Singh B 105
Singh B K r512
Singh H G r516
Singh H r268 r513 r514
Singh I S r286
Singh J r515
Singh K N r516
Singh S N r516
Sinibaldi R M 102
Sisco P H 112 113 134 136 138 146 r517
Sivolap Y M r53
Skinner G r555
Skogen-Hagenson M-J 16
Skoog F 75
Skripka L V r303
Slocum M r221
Smith J A r518
Smith J D 39 r351 r352 r448
Smith J S C 134 135 136 137 r519 r520 r521 r522
Smith O S 57 r75 r557
Smith P 103
Snidero M r383
Soave C 100 114 r179 r215 r356 r452 r477
Soldatini G F r182
Solonenko T A r277
Sommer H r474
Sondahl M R r434 r523
Song Y r524
Souza C L Jr 139 r525 r526
Sparvoli F 100
Spena A 42 139 r527
Spielmann A r298
Sprague G F 34 131 r528
Spray C R r226 r418 r419
Stack S M r15
Stahelin H r578
Stamp P r529 r530 r531 r532
Starlinger P 36 39 39 40 41 r91 r533 r584
Steffensen D M 146
Stephenson G R r145
Stern D B 111 170
Stern H 71
Still P 58 60
Stinard P S 7 11 135 r458
Stinson J r534
Stirdivant S M r535
Stocking C R r464
Stoilov M r536
Stolarz A r309
Stone B A r537
Storey K K 115 r247
Streitenberger M E 81 84 85
Strittmatter G 138 r538
Strommer J N 17 r463
Struik P C r539
Stubbe H 70
Stuber C W 33 110 120 135 r119 r470 r520 r521 r522 r540
Sukalovic V H-T r541
Sultan M r256
Suprasanna P 65 66 67
Sutter G R r258
Sutton W D r403
Suzuki A r578
Swanson E B r542
Szel S r362
Szodfridt G r365
Tadic B r543
Takaiwa F 126
Tavares M J C M S r360
Taylor J R N r544
Taylor L P 5 134 r162 r545 r576
Taylor W C 16 25 47 r204 r329 r492 r546
Telkamp R E r259
Tewari K K 16
Thalouarn P r454
Theres K 39 40 134
Thiraporn R r532
Thompson D L r547 r548
Thompson R D 101 170
Thuriaux P r549
Tiemeier D C r501
Tillmann E r91 r125 r533
Timmis J N 16
Timothy D H 170 r111 r112 r395 r396
Ting Y C 37 38 78 90 r196 r197 r550
Titov A F r551
Todesco G r174
Todorova L r271 r552 r553
Toenniessen G H r554
Toler R W r555
Toloczyki C 58 135
Tomes D T 139 r556 r557
Tonelli C 100 136 r172 r558 r559
Topol 62
Torrent M r313
Tracy W F 97 146
Tragani N 137 r560
Treharne K J r459
Troyer A F 51 139 r561
Tsai C Y r211 r212 r293
Turpen T H 102
Ullstrup A J 50 106
Ulrich V 103
Umbeck P 63
Underbrink A G r312
Upadhyaya K r474
Urbakh V G r187
Urrutia M I 77
Vakhrusheva E I r562
Valencia G r353
VanderEb A J 62
VandeVenter H A r323 r324 r325 r563
Vanegas-A H r564
vanHuystee R B r228
VanLammeren A A M r486
VanToai T T 139 r565
Varagona M J r586
Vargas H r299

Vargas-O M A 37 r566
Vargas-S E r257
Vargas-S J E r564
Varghese T M r115 r116
Vasil I K r567
Vasil V r567
Vasilas B L r568
Vencovsky R r180
Verenik 95
Vianna R T r170 r308
Victoria-R M T r564
Vidal J r454
Vidaver A K 74
Vieira J r374
Viereck A r569
Vierling R 44
Villa M 99
Villamizar N R r5
Villani M G r570
Vinarova K r536
Vincentz M r571
Viotti A 139 r572 r573
Virupasksha 88
Visser J H r323 r324 r325
Vitale A r572
Voronova L P r574
Vosa C G r6
Vulchev V r575
Wach M J r144
Wagner E D r423
Wahl 91
Waiss A C Jr r599 r600
Walbot V 5 18 25 49 108 120 124 134 137 138 r162 r367 r368 r369 r381 r545 r576 r577
Walden D B 32 41 89 91 92 93 94 98 101 r37
Wall J S r141
Walton J D r578
Wan-Zhen W-Z 90
Wandelt C 58
Wang B r579
Wang S-M r580
Wara-Aswapati O r464
Warren H L r211 r212
Wassom C 97
Watanabe K r17
Watson M r581
Weber D F 60 120 146 r509
Weber E J r169
Weck E r91 r123
Wegman S r221
Weil J-H r326
Weissbach H r89
Weissinger A K r582
Weissinger H H 33
Wellmann E r38
Wendel J F 30 33 110 113 134 135 136 137 146 149
Werder J r583
Werr W 41 59 137 r91 r533 r584
Wesselbeaver L r585
Wessler S R 18 42 137 r586 r148
Whalen R H 30 31 117 135 146 147
Wheeler J G 16 r458
White D G r405
White E M 98 102
Widholm J M r130
Widstrom N W r246 r335 r587 r588 r597 r598 r599 r600
Wienand U 42 42 135 r474
Wilkes H G 30 105 r406 r589 r590
Williams M E r130
Williams W P 139 r370 r591 r592
Willing R P r593
Willmitzer L 42
Wilson A J r594
Wilson C M 137 r595 r596
Wilson D M r198 r335 r587
Wise R P 64 138
Wiseman B R 139 r246 r597 r598 r599 r600
Witkowski J F r71
Wojtas A r36
Wong R S C r542
Wood J A r105
Woodman J C 124 r601
Wright S 61 120
Wu 61
Wu Q r605
Wuiter K A 30
Wydemann U 42
Xu W r605
Yamada M r602
Yanders A F r312
Yang T X r604
Yoder J r91 r533
Yoder O C r163
York J O r68
Young J R r588
Yu M 90
Yu S-M r105
Yudin B F r314
Zabirova E R r603
Zabulionis R 91
Zack 58
Zehr B E r130
Zelenin A V r53
Zeng M Q r604
Zhang C r605
Zhang X A r196 r72 r197 r236
Zhao Y r605
Zhao Z r236 r606
Zhu X r605
Zhu Yu S r607
Zimmer E A 23 73 91
Zink F r608 r609
Zinsly J R r525 r526
Zryd J-P r549
Zscheile F P 7
Zsuzsanna Rakk V r342
Zuber M S r34 r345 r377

THE PURPLE MAIZE STORY

Once upon a time, there was 'Purple maize',
it asked God, about how it came in the aleurone.
He turned and there were geneticists;
One by one they spoke; Purple, stood astounded.,
for there were fourteen loci and several alleles.
"Oh. God. it's terrible", the Purple said.
"Thou shalt witness many more', god fled
and sent chemists, biochemists and geneticists.
"I am becoming more complex", Purple wept.
It said, "CI is making things colorless",
"R- is becoming more complex day by day",
"my enzymes are still undone", and
"Now the threat of transposable elements".
(Dr.Mc Clintock slightly blinked at the remark).
God again got up from sleep.
He cursed that he's disturbed
but was also moved by tears.
He cried,
"it shall continue, the CI and R,
enzymes will be done",
"but the elements", the Purple added,
"Yes, the elements shall continue their attacks", god laughed.
And sent molecular biologists this time.
Purple returned and sat under the plant.
Suddenly there were cries,
"Bz is done, C2 is done, A is done.,
and many more".
Purple kneeled before god and pleaded,
gracefully god spoke,
"my dear purple maize, this shall go on,
be happy, never cry,
Thou shall remain, '<u>Purple pigment System</u>'
remembered by one and all,
and thus your name 'll ring for ages and ages".
Purple remained and remained there, motionless.
It knew how it came.
And now feels great in giving.

P. Suprasanna
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This is an informal news letter by which working research information on the genetics and cytogenetics of maize is shared. The information and data are presented by Cooperators with the understanding that they will not be used in publications without the specific consent of the author.

Author and Name Indexes	
Nos. 3 through 43	Appendix to No. 44, 1970
Nos. 44 through 50	No. 50, pp. 157-180
Nos. 51 to date	Annual in each issue
Mailing List	No. 59; updated in this issue
Stock Catalogs	
Genetic Stocks	Annual in each issue
Translocations	No. 55
Symbols	
Nos. 12 through 35	Appendix to No. 36, 1962
Nos. 36 through 53	No. 53, pp. 153-163
Nos. 54 to date	Annual in each issue
Rules of Nomeclature	No. 49, pp. 3-4
Cytogenetic Working Maps	No. 52
Gene List	No. 57
Working Linkage Maps	This issue