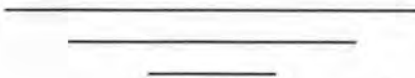
# MAIZE GENETICS COOPERATION

# NEWS LETTER

# 59



March 31, 1985

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

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

This issue is dedicated to

R. Alexander Brink

and

Albert E. Longley

Cooperators

TABLE OF CONTENTS


Page

I. FOREWORD . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 1

II. REPORTS FROM COOPERATORS . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 2

ALBANY, NEW YORK
Construction and use of a cloned cDNA library to messenger RNAs from pollen --A. Eisenberg, R. P. Willing, J. R. Stinson, M. E. Pe, J. P. Mascarenhas 2

AMES, IOWA
The movement of the En receptor I or En itself into C-I: the origin of the unstable allele C-I-m836976 --P. A. Peterson 2
Deficiency c-sh-836882 --P. A. Peterson 3
c-836957 --P. A. Peterson 3
The isolation of En-1 in the wx-84-4 allele --P. A. Peterson 3
A dominant color allele, C-m(r), responsive to a specific Uq --P. A. Peterson 3
vpl linkage with B-A translocations --B. E. Scheffler, P. A. Peterson 3
A new dormant allele of Vpl --B. E. Scheffler, P. A. Peterson 4
vpl-m451, a mutable allele of vpl that responds to Cy --B. E. Scheffler, P. A. Peterson 4
Uq controlled mutable allele at the c locus --E. E. Oberthur, P. A. Peterson 4
System relationships of the Cy transposable element system including tests against Robertson's Mutator system --P. S. Schnable, P. A. Peterson 4
Distribution of genetically active Cy elements --P. S. Schnable, P. A. Peterson 5
Simultaneous change at bz and Sh in bz-rcy, Cy: bz-n(rcy)-sh-835211w; bz-n(rcy)-sh-835211x-1 --P. S. Schnable, P. A. Peterson 6
Cycling states in the Cy transposable element system: Tissue specificity of two states of bz-rcy that cycle in the presence of Cy --P. S. Schnable, P. A. Peterson 6
Anther fluorescence of several non-responsive bronze derivatives of the autonomous En controlled bz-m826301 --P. S. Schnable, P. A. Peterson 7
Update on recessive brown seed mutant (brn) --P. S. Stinard 7
A possible test for putative quantitative alleles of qualitative mutants --D. S. Robertson 8
Some observations on the loss of Mutator activity in Mutator lines --D. S. Robertson 8
Can lines that have lost Mutator activity as a result of inbreeding regain it by outcrossing? --D. S. Robertson 10
The frequency of a1-Mum somatic reversions in reciprocal crosses with a1 testers --D. S. Robertson 10
Studies on the nature of the loss of somatic mutability for two Mu-induced mutable a1 mutants --D. S. Robertson 11
The relationship between somatic instability of Mu-induced mutants and Mutator activity --D. S. Robertson 13
Evidence for Mutator activity in the male gametophyte --D. S. Robertson 14
The effect of gamma ray irradiation on Mutator activity --D. S. Robertson 15
Putative Mu-induced deficiencies --D. S. Robertson 16

BARCELONA and VALENCIA, SPAIN
Influence of chromosome segments linked to pale green-11 and pale green-12 on quantitative traits --L. Bosch, F. Casanas, F. Nuez 18
pg11 pg11 pg12 pg12 and smut --L. Bosch, F. Casanas 20
Differences in days to flower between inbreds, and grain yield of single crosses among them --F. Casanas, L. Bosch, F. Nuez 20

BLOOMINGTON, INDIANA
Ac--an insertional derivative of Ds? --D. Schwartz 22

CAMPINAS, SAO PAULO, BRAZIL
Linkages in chromosome four, horizontal resistance and kernel row number --L. T. de Miranda, L. E. C. de Miranda 23
Linkage between Ga-S and ol --L. T. de Miranda, L. E. C. de Miranda 24

CANBERRA CITY, AUSTRALIA
A somaclonal mutant of maize alcohol dehydrogenase --R. Brettell, M. Jeppesen 24

CASTELAR, ARGENTINA
A potential alternative method in maize breeding --J. C. Salerno 24
Lines of maize with low gametic transmission --J. C. Salerno 25
The linkage of chlorophyll lethal genes with floury-2 --J. C. Salerno 25
The linkage of chlorophyll lethal genes with iojap --J. C. Salerno 26
Manipulation of a balanced lethal system --J. C. Salerno 26

CHAPEL HILL, NORTH CAROLINA
Teosinte introgression, a probable source of mobile genetic elements --P. C. Mangelsdorf 26

CHESTNUT HILL, MASSACHUSETTS
Meiosis and phenotypical expression of a dihaploid maize --Y. C. Ting 28
Ploidy stability of maize callus lines --M. G. Gu, Y. C. Ting 29
Anther culture of tetraploid maize --S. Raja, Y. C. Ting 30

COLLEGE STATION, TEXAS
Rapid isolation and purification of immature zygotic embryos --F. Fong, J. D. Smith 30
Gravitropism in abscisic-acid-deficient seedlings --J. D. Smith, R. Moore, F. Fong 31
Effects of a systemic insecticide and varietal resistance in reducing corn earworm damage --M. H. Yu, J. D. Smith 32

COLOGNE, WEST GERMANY
Studies of the shrunken gene on chromosome 9 --W. Werr, W.-B. Frommer, C. Maas, P. Starlinger 32
Regulation of the sucrose synthase genes --B. Springer, W. Werr, P. Starlinger 32
The sequence of transposable element Ac --M. Muller-Neumann, J. Yoder, P. Starlinger 33
The Adh1-2F11 allele is characterized by the insertion of an "aberrant" Ds element --A. Merckelbach, P. Starlinger 33
Molecular analysis of the En/Spm transposable element system --R. Berndtgen, H. Cuypers, A. Gierl, R. B. Klosgen, A. Pereira, P. A. Peterson, H. Saedler, Zs. Schwarz-Sommer 34
The use of Spm(En) and Mu transposable elements for molecular analysis of the A1 and C2 loci --C. O'Reilly, N. S. Shepherd, A. Pereira, Zs. Schwarz-Sommer, U. Niesbach, U. Weydemann, U. Wienand, D. S. Robertson, P. A. Peterson, H. Saedler 35
The Teo1 insert from Zea mexicana teosinte Guerrero is a composite of the Cin2 and Cin3 elements --J. Blumberg vel Spalve, N. S. Shepherd, H. Saedler 35
COLUMBIA, MISSOURI
Flavonoid 3' hydroxylase and Pr --R. L. Larson, J. B. Bussard 36
A computer-assisted model of aneuploidy in autotetraploid maize populations --G. G. Doyle 36
Chloroplast developmental timing: nuclear perturbation --M. Polacco 37
Isolation of a mutable hcf*-19 allele --M. Polacco 38
Mapping hcf mutants: progress report --M. Polacco 38
Position of loci on chromosome 3L with respect to B-A translocations --J. B. Beckett 39
TB-5Ld, a new B-A translocation on the long arm of chromosome 5 --J. B. Beckett 39
Establishment of homozygous B-A translocation stocks --J. B. Beckett 39
Blue-fluorescent plants are fragrant --E. H. Coe 39
Silk browning is related to cob color --E. H. Coe 40
Chromosome 9 factors --E. H. Coe 40
The modification of viability of some defective kernel mutants --M. T. Chang, M. G. Neuffer 41
Location of four new dominant mutants --R. M. Bird, M. G. Neuffer 42
Computer listing and summary of new EMS-induced mutants and variants --R. M. Bird 43
Ancient tiny cobs and the evolution of Zea --R. M. Bird 43
A Tale of Two Necrotics --D. Hoisington 44
B breakpoints of the most proximal translocations --B-Y. Lin 44
Chromosome basis of small seed effect associated with r-x1 --B-Y. Lin 45
Some information about nondisjunction incurred by the r-x1 deletion --B-Y. Lin 45
COLUMBIA, MISSOURI and AMES, IOWA
Significant reciprocal differences between F1 crosses of Revolution and TELE --L. L. Darrah, M. S. Zuber, L. M. Pollak 46
DEKALB, ILLINOIS
Producing single cross seed corn on hybrid plants --A. F. Troyer 48
FREIBURG, WEST GERMANY
Expression of zein genes and possible intron structure(s) --J. W. S. Brown, P. Langridge, G. Feix 48
GAINESVILLE, FLORIDA
Distribution of minilinear and minicircular mtDNA sequences within Zea --D. R. Pring, A. G. Smith 49
GAINESVILLE, FLORIDA AND ST. PAUL, MINNESOTA
Mitochondrial DNA rearrangements associated with reversion of T cytoplasm to male fertility and disease resistance --R. P. Wise, D. R. Pring, B. G. Gengenbach 50
HYDERABAD, INDIA
Effect of 2,4-D analogues on callus cultures --P. Suprasanna, K. V. Rao, G. M. Reddy 50
Induction of somatic embryos from root callus --K. V. Rao, P. Suprasanna, G. M. Reddy 51
Extraction and characterization of certain maize flavonoids --K. V. Rao, P. Suprasanna, G. M. Reddy 51
Phenylalanine ammonia lyase activity and anthocyanin accumulation in the aleurone tissue --C. Jayaram, A. R. Reddy 51
Chloroplast ultrastructure studies of the etched mutant --S. V. Ramesh, A. R. Reddy 52
Chloroplast pigment analysis in individual leaves of light-grown seedlings --S. Kumari, H. G. Sangeetha, A. R. Reddy 53
IOWA CITY, IOWA
Duplication and stable inheritance of chromosomal segments --W. Carlson 53
Initiation of synapsis by proximal chromosomal regions --W. Carlson 54
Calculation of meiotic loss for TB-9Sb --W. Carlson 54
ITHACA, NEW YORK
Somaclonal variation in plants regenerated from the inbred W182BN --E. D. Earle, V. E. Gracen 55
Mosaic tassels in iojap progeny --C. A. Lemke, V. E. Gracen, H. L. Everett 56
KINGSTON, RHODE ISLAND
Expression of a plump phenotype in the endosperm, independent of bronze --J. P. Mottinger 56
LINCOLN, NEBRASKA
Chromosomal location of a gene for resistance to Corynebacterium michiganense ssp. nebraskense --T. R. Rocheford, C. O. Gardner, A. K Vidaver 57
Growth of Corynebacterium michiganense ssp. nebraskense on maize callus --T. R. Rocheford, A. K. Vidaver, C. O. Gardner 57
Effect of wind-generated sand abrasion on infection by Corynebacterium michiganense ssp. nebraskense --T. R. Rocheford, A. K. Vidaver, C. O. Gardner, D. L. Armbrust 58
LLAVALLOL, BUENOS AIRES, ARGENTINA
Tissue cultures of Zea mays x Zea perennis x Zea diploperennis --M. A. Rapela 58
Maize organogenesis and somatic embryogenesis: stage of the donor tissue --M. A. Rapela 59
SDS-protein pattern of maize plants regenerated through somatic embryogenesis --M. A. Rapela 59
Lysine plus threonine inhibition in hybrid cell cultures: A possible case of allelic assortment --M. A. Rapela 60

LOMAS DE ZAMORA and LLAVALLOL, BUENOS AIRES, ARGENTINA
Evolution of Zea --J. L. Magoja, I. G. Palacios, L. M. Bertoia, M. E. Streitenberger 61
Puna maize: selection for high protein quality and hard endosperm --A. M. Broccoli, M. E. Streitenberger, J. L. Magoja 67
Differences between Gaspe x Z. perennis and Gaspe x Z. diploperennis F1 hybrids --J. L. Magoja 68
Perennial teosinte-Gaspe hybrids: preliminary results on inheritance of pericarp and aleurone layer thickness --L. M. Bertoia, J. L. Magoja 68
Perennial teosinte-Gaspe hybrids: multilayer aleurone --L. M. Bertoia, J. L. Magoja 69
Perennial teosinte-Gaspe hybrids: specific relationship of endosperm protein content --L. Ferrari, J. L. Magoja 69
Perennial teosinte-Gaspe hybrids: selection for maizoid characters --G. Pischedda, J. L. Magoja 70
Effect of diploperennial teosinte germplasm on maize endosperm proteins --A. A. Nivio, J. L. Magoja 71
Immediate effect of perennial teosinte introgression in maize: endosperm storage proteins --A. A. Nivio, J. L. Magoja 72
LONDON, ONTARIO, CANADA
In vitro liquid culture of corn tassels - an update! --D. R. Pareddy, R. I. Greyson 72
In vitro germination of pollen from cultured tassels --D. R. Pareddy, R. I. Greyson, D. B. Walden 73
In vitro culture of immature ear shoots --V. R. Bommineni, R. I. Greyson 74
Characterization of polypeptides synthesized by mRNA isolated from pollen --M. J. Dunlop, D. B. Walden 75
Incorporation of radioactively labeled amino acids into protein in leaves does not require prior surface abrasion --C. A. Rees, T. G. Crowe, D. B. Walden 75
Isolation of a mRNA for the 18,000 dalton maize heat shock polypeptide --C. A. Rees 76
Heat shock polypeptide synthesis in monosomic plants --C. A. Rees, D. B. Walden 76
Comparisons of newly synthesized polypeptides obtained from maize embryos of different genotypes and developmental ages --J. G. Boothe, D. B. Walden 77
Response to certain agrichemicals as measured by $^{35}$S-methionine uptake and protein synthesis --D. B. Walden, R. B. Zabulionis 77
MEADVILLE, PENNSYLVANIA
Long-term heat shock in maize seedlings --B. H. Rockwell, P. Lehman, C. M. Nebiolo 78
MILAN, ITALY
Growth of embryos, shoot and root tips of Zea mays L. in media devoid of nitrogen --C. Tonelli, I. Viani 78
A new cherry allele at the R locus: R-ch(Hopi) --G. Gavazzi, M. L. Racchi, A. Gorrini 79
The effect of light on pigment accumulation in germinating r-ch(Hopi) seeds --M. L. Racchi, G. Gavazzi, A. Gorrini 80
Extent of gene expression in pollen --M. Sari Gorla, C. Frova, G. Binelli, E. Ottaviano 81
MORGANTOWN, WEST VIRGINIA
A comparison of methods for isolation of maize seedling nuclei --S. E. Palmer, V. Ulrich 81
Distribution of nonhistones, histones, and putative HMG proteins between fractions of maize seedling chromatin --S. E. Palmer, V. Ulrich 82
Comparisons of chromatin conformation and compositions of heterotic hybrid and parental inbred maize --S. E. Palmer, V. Ulrich 83
NEW DELHI, INDIA
Maximizing grain yield --B. C. Saha, B. K. Mukherjee 83
NEW HAVEN, CONNECTICUT
Plant regeneration from tissue culture --P. N. Gordon 84
OAKLAND, CALIFORNIA
The genetic location of vp1 --H. K. Dooner 85
ONGOLE, ANDHRA PRADESH, INDIA
A new orcein technique for mitotic chromosome analysis --J. S. P. Sarma 85
PHILADELPHIA, PENNSYLVANIA
Dosage analysis of Tp1 and Tp2 --S. Poethig 86
The location of c1 --S. Poethig 86
The location of Esterase-8 --J. Sorrentino, S. Poethig 86
PRESCOTT, WISCONSIN and JOHNSTON, IOWA
Three new male-sterile genes --D. P. West and M. C. Albertsen 87
RALEIGH, NORTH CAROLINA
Twelve new isozyme loci in maize: Progress report on chromosomal locations, the subunit composition, and subcellular localization of their products --J. F. Wendel, C. W. Stuber, M. M. Goodman 87
Localization of two new isozyme loci, Hex1 and Tpi4, to chromosome 3 --J. F. Wendel, C. W. Stuber, M. M. Goodman 88
Linkage relationships between a new locus, Hex2, and previously assigned loci on chromosome 6 --J. F. Wendel, C. W. Stuber, M. M. Goodman 89
Mapping data for 34 isozyme loci currently being studied --J. F. Wendel, M. M. Goodman, C. W. Stuber 90
ST. LOUIS, MISSOURI
Linkage data for 2L and 4L mutants --S. McCormick, A. Barnason 91
ST. PAUL, MINNESOTA
Discovery of Ac activity among progeny of regenerated maize plants --V. M. Peschke, R. L. Phillips, B. G. Gengenbach 91
Genetic control of plant regeneration from maize tissue cultures --C. L. Armstrong, C. E. Green, R. L. Phillips, R. E. Stucker 92
rDNA restriction analysis of tissue culture regenerated A188 plants --G. Benzion, B. G. Hunter, I. Rubenstein, R. L. Phillips 93
Tests for a cytoplasm that restores genetic male sterile-1 --C. R. Burnham 94
The su gl4 la stock --C. R. Burnham 94
Discussions in Cytogenetics --C. R. Burnham 94

SRINAGAR, KASHMIR, INDIA and BOMBAY, INDIA
Genetic variability and character association in maize grown in Kashmir --P. N. Jotshi, K. A. Patel, M. K. Bhan 94
STANFORD, CALIFORNIA
Variability among mutator elements --L. P. Taylor, V. Walbot 96
Cloning and characterization of the linear 2.3 kbp mitochondrial plasmid --P. Bedinger, E. de Hostas, V. Walbot 96
Aberrant segregation of a mutable phenotype in a Robertson's Mutator line --V. Chandler, V. Walbot 96
TSUKUBA, JAPAN and ST. PAUL, MINNESOTA
Fertile revertants possessing S-1 and S-2 DNAs in cms-S --T. Ishige, B. Gengenbach 98
UNIVERSITY PARK, PENNSYLVANIA
Properties of starches from nonmutant and mutant endosperm cultures --C. D. Boyer, C. Haemmerle 99
URBANA, ILLINOIS
Mapping zein polypeptides using translocations --L. M. M. Ottoboni, D. M. Steffensen 99
Cytoplasmic reversion to fertility in cms-S without loss of linear mitochondrial plasmids --L. J. Escote, S. Gabay-Laughnan, J. R. Laughnan 100
VICTORIA, B. C., CANADA
Flavonoids in brown pericarps --O. Ceska, E. D. Styles 100
Expression of the R-r:Ecuador(1172) allele --E. D. Styles 101
WALTHAM, MASSACHUSETTS
Vascular anatomy of the female rachis in teosinte and maize --W. C. Galinat 101
Outer-inner vascular connections and glume phenotype --W. C. Galinat 101
Suitable support system for key-trait expression --W. C. Galinat 101
Necessity for a more comprehensive cytogenetic and phenotypic analysis of the maize-teosinte relationship --W. C. Galinat 102
Kernel interlocking (tesselation) resulting from cupule reduction --W. C. Galinat 102
Silkless baby corn, seed production genetics --W. C. Galinat 102
Morphology of maize --W. C. Galinat 103
WOOSTER, OHIO
Alleles at the ora2 locus --E. J. Dollinger 104

ADDENDUM
GRAND FORKS, NORTH DAKOTA
Developmental profiles for two new embryo lethal mutants, dek22 and dek23 --W. F. Sheridan, J. K. Clark 104
KRASNODAR, U.S.S.R.
Relationships between sensitivity of inbred lines to NMU and their mutability --A. S. Mashnenkov 104
MADISON, WISCONSIN
The deficiency of a starch granule-bound enzyme phospho-oligosaccharide synthase in developing bt1 bt1 bt1 endosperms --D. Pan, O. E. Nelson, Jr. 105
NORMAL, ILLINOIS
Printing strip tags using a data base --D. Weber 106
PRESCOTT, WISCONSIN
Polymitotic: supernumerary repetitions of meiosis II --D. P. West, R. L. Phillips 106
Polymitotic, canalization and probability --D. P. West 107

III. MAPPING 1985 . . . . . 108

IV. ZEALAND 1985 . . . . . 111

V. REPORT OF MAIZE GENETICS COOPERATION STOCK CENTER . . . . . 115

VI. MAILING LIST . . . . . 120

VII. RECENT MAIZE PUBLICATIONS . . . . . 134

VIII. SYMBOL INDEX . . . . . 149

IX. AUTHOR AND NAME INDEX . . . . . 153

X. COMPILATION OF B-A TRANSLOCATION DATA AND WORKING MAPS . . . . . 159

# I. FOREWORD

First, a "Thank You" and a silken ear to all the Cooperators who sent their Reports on time, with concise articles, compact figures and tables, honest-to-goodness data, well-spelled phrases, carefully considered symbols, double-spaced copy, mapping data, useful techniques and tidbits. No tongue-in-cheek, simply an appreciation of the helpfulness that was evident this year. The Cooperation is alive and well, as can be seen from the Reports within.

Second, be prepared for a surprise inside, and be careful not to draw the conclusion that this has become a book or a publication. To newcomers in these covers, whose experience has not included the joy of being told at an appropriate knee about the traditions in maize genetics (MUST READ: Rhoades, Ann. Rev. Genet. 18, 1984), be informed:

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

This means that, while the information here has been shared, it is still the property of the author or authors whose field books and note books are opened in these pages, and whose concurrence must be obtained in citing or using the information.

The typeset pages and two-column form were suggested by University Printing Services as a more efficient combination that would be attractive and less costly in plates, paper and mailing; the result is a considerable reduction in bulk. My experience in this kind of production was limited, and I ask your forebearance in any oddities that resulted; they are mine, not the printer's.

The Mailing List, updated, includes telephone numbers that were supplied in response to postcard mailings. A number of addressees were dropped following two rounds of postcard inquiries; if there are individuals who are not listed, and should have been, I would be pleased to hear from them, with a proper mailing address and telephone number for future listings.

The list of Recent Maize Publications is numbered this year, to aid in author-hunting (no doubt you have found it tedious, as have I, to search for junior authors from a page reference). These numbers are prefixed by "r" in the Author and Name Index, and in Zealand. Dave Hoisington performed the programming magic for linking of the author names and reference numbers, among many other useful and helpful aids for efficient searching, address-finding, etc. He is even more to be commended for the compilations and working maps offered for your use, your critiques, your suggestions, your ideas, your help, your modifications, your improvement and your upgrading. These maps are living and refinable things, to which all information, old and new, can and should be brought. We solicit your help in studying the working maps and the Mapping section, and we ask all who are doing mapping to let the coordinator know about the work in progress and to supply information and data toward the documenting, compiling and evaluating effort that is ahead.

Shirley Kowalewski and Mary Brazil gave their skillful attention to the year-round office work, including especially the literature compilation. Christopher Browne and Kathryn Chappell helped with numerous vital tasks, in addition to keeping field and lab work going. Proofing, suggestions and critiques by Robert Bird, Ming-tang Chang, Christine Curtis, Craig Echt, Chang-Deok Han, Bryan Kindiger and Rick Vierling are greatly appreciated.

If you have not yet seen the display cases of diverse ear types at the Ames Plant Introduction Station, mounted by 20th Century Fox for a scene and returned to Ames for use, they are well worth a visit -- almost as interesting as the facility itself. Dr. Raymond Clark, Director of the Station, showed the mounts to me in September.

With flowery wishes for an ideal pollinating season, and the pick of the crop.

Ed Coe

# II. REPORTS FROM COOPERATORS

ALBANY, NEW YORK
State University of New York at Albany

## Construction and use of a cloned cDNA library to messenger RNAs from pollen

Corn pollen at maturity contains a store of messenger RNAs (N. T. Mascarenhas et al. 1984, Theoret. Appl. Genet. 68:323-326). In order to study the regulation and function of these mRNAs, we have constructed a library of complementary DNA (cDNA) clones made to poly(A) RNAs isolated from mature pollen of maize, hybrid "Gold Cup" (Harris Seeds, Rochester, New York). The cDNAs were cloned in pBD1, a plasmid vector-primer system modified from Okayama, H. P. and Berg, P. (1982 Mol. Cell. Biol. 2:161-170; D. C. Alexander, B. G. Williams, D. McKnight, manuscript in preparation). The vector was kindly provided by Drs. Danny C. Alexander and Bill G. Williams. The plasmids containing the double stranded inserts were introduced into *E. coli* (HB 101) by transformation. Colonies containing sequences complementary to pollen messenger RNAs (mRNAs) were identified by colony hybridization to $^{32}$P-cDNA made to pollen poly(A) RNA.

We have selected 100 clones from the library for further study. Several of the clones are pollen-specific. This has been determined by Northern blot hybridizations using RNA isolated from pollen and several vegetative tissues. The majority of the clones, however, are expressed in both pollen and vegetative tissues. The cloned inserts range in size from about 50 nucleotide pairs to 1268. The mRNAs to which the clones are complementary range from about 600 to 2500 nucleotides, as determined by Northern hybridizations. Based on Southern hybridizations to restriction-enzyme-digested genomic DNA, the pollen-specific clones thus far tested seem to be represented by single genes or a very few genes.

RNA was isolated from pollen of different stages of development and analyzed by Northern hybridizations with one of the pollen-specific clones (pZmc30), which hybridizes to a mRNA, approximately 2000 nucleotides in size. The results indicate that transcription of the mRNA is initiated during the interphase following microspore mitosis but prior to generative cell division, and the mRNA continues to accumulate reaching a maximum in the mature pollen grain. Similar analyses are being carried out with several other pollen-specific clones and clones that are expressed both in pollen and vegetative tissues.

We are currently using several of the cDNA clones to isolate genomic clones of the inbred line W22, with the aim of characterizing the pollen-expressed genes in greater detail.

Arthur Eisenberg, R. Paul Willing,
Jeffery R. Stinson, M. Enrico Pé
and Joseph P. Mascarenhas

AMES, IOWA
Iowa State University

## The movement of the *En* receptor *I* or *En* itself into *C-I*: the origin of the unstable allele *C-I-m836976*

*C-I-m836976* arose by the insertion of a mobile element into the *C-I* allele in a population of *C-I/C-I En, En, (En?)*. This allele was one of several unstable *C-I* alleles (MGCNL 58:2) uncovered in this population. When in combination *C-I-m/C*, the phenotype is colored, mutating to colorless. The *C-I-m* allele has not been seen as a homozygote and thus it is not known whether it is *C-m* or *c-m*. What is clear is that it mutates to *C-I*, as is evident in the colorless sectors of the *C-I-m/C* heterozygote. In the cross of *c2-m2 wx-m8* x *C-I-m Sh Bz Wx/C sh bz wx; En, En* (Cross A), the following progeny were observed in a small sample from two ears:

| Full Colored - Not Sectored | | | | Sectored | | |
|---|---|---|---|---|---|---|
| Ear | *Wx* | *wx* | *wx* → *Wx* | *Wx* | *wx* | *wx* → *Wx* |
| 1 | 14 | 5 | 33 | 45 | 0 | 10 |
| 2 | 6 | 3 | 16 | 27 | 0 | 14 |

The following comments can be made on this small sample. The *wx* → *Wx* phenotype is from *En* acting on the *wx-m8* allele originating from the female parent in the heterozygote, *wx/wx-m8*. *En* came from the *C-I* parent. Thus the excess *wx* → *Wx* kernels among the full colored - not sectored class must be caused by many *En* rather than a linked *En*. The kernels that are sectored and *wx* →*Wx* are from cross-overs.

There is an excess of sectored in ear 2. This is not explainable in view of the observation of the expected *Wx* vs. *wx* ratios in ear 1 and ear 2.

The absence of kernels that are sectored and waxy and the presence of full colored and waxy is strong support that sectoring is strongly correlated with the *En* effect on the *wx-m8* allele. It is not possible to conclude on the state of *En* at the *C-I-m* allele since the additional *En* in the material does not allow a more definitive answer. It can, however, be concluded that the *C-I-m* allele sectoring is *En*-dependent.

There is a possible explanation for the *C-I* allele mutation event. It appears that the original *C-I* is

caused by a stable insert (MGCNL 58:2) that becomes part of a chimeric transcript and the resulting protein is consequently distorted (Peterson and Leleji, Canad. J. Genet. Cytol.). For *C-I-m836976, En* or *I* was inserted in such a manner to inhibit the locus such as typical *c-m(r)* or *a2-m(r)*. By excision of *I*, the original locus condition is restored and capable of inhibiting anthocyanin formation.

Peter A. Peterson

### Deficiency *c-sh-836882*

In a population derived from the cross of *C-I Sh Bz Wx; En, En, En* x *C sh bz wx* (Cross B), a colored shrunken kernel appeared. This is a simultaneous change in both the *C-I* and *Sh* genes. Crosses were made as follows, with the indicated results:

| Colored shrunken kernel | |
|---|---|
| x *C sh bz wx* | 74 sh Bz, 77 sh bz |
| From *sh Bz* class | |
| -1 selfed | 81 sh Bz, 109 sh bz |
| -1 on *C sh bz wx* | all sh bz |
| -7 selfed | 124 sh Bz, 116 sh bz |
| -7 on *C sh bz wx* | all sh bz |
| -4 x *C sh bz wx* | 156 sh Bz, 178 sh bz |
| -7 x *c-m(r)* | 172 Colored, 59 spotted, 82 colorless |

Comments on these preliminary data: lines 1 and 6 indicate adequate female transmission. Lines 2 and 4 show the absence of male transmission from this deficiency. This is also true of the tests of males on the *C sh bz wx* tester as in lines 3 and 5. Line 7 indicates that an *En* is present and the *C-I* allele changed to a null or deficiency that also includes parts of the *Sh* locus.

Peter A. Peterson

### *c-836957*

A *c* mutant arose from a *C-I* allele. From the same cross shown as cross B above a colored kernel appeared amidst an ear that was otherwise completely colorless. This kernel was designated *c-836957*. Crosses with *C sh bz wx* gave a progeny distribution shown below.

| | Round | | | | shrunken | |
|---|---|---|---|---|---|---|
| | Cl | sp | cl | mottled | Bz | bz |
| Confirmation ear | 187 | 0 | 6 | 60 | 5 | 124 |
| Mottled from line 1 x *c-m(r) Sh Bz Wx* | 129 | 99 | 106 | 45 | — | — |

The mottled in lines 1 and 2 are not easily explained, but their distribution excludes the segregation of *r*. The colorless in line 1 are more easily explained by a reversion of the mutant *c* to *C-I*. *En* is present as is evident from the spotted (sp): colorless (cl) distribution seen in line 2.

Peter A. Peterson

### The isolation of *En-1* in the *wx-84-4* allele

*En* was isolated from an unstable *wx* allele, identified as *wx-84-4*. This arose out of a population cross of *C-I Sh Bz Wx; En, En* x *C sh bz wx*. *wx-84-4* was one of 12 *wx* alleles isolated but two appeared to have instability. Because only two were isolated from two different populations, only those values will be presented.

*wx-84-4* - 1 in $1.9 \times 10^{-6}$ = *En-1*
*wx-84-11* - 1 in $3.9 \times 10^{-5}$

This is a conservative estimate because the other 11 have not been verified for instability or *wx* confirmation.

Peter A. Peterson

### A dominant color allele, *C-m(r)*, responsive to a specific *Uq*

In evaluating the Rhoades-Dempsey "Hi-loss" lines (MGCNL 56:21) for mobile elements, a *Uq* was found. This was evident in the initial cross *c-ruq* x *C Uq*, where the progeny showed colored kernels with spots. The spots resulted from the *Uq-Hi-loss* on *c-ruq* and appeared in the *c-ruq/c-ruq/C Uq* endosperm. At first, it appeared that this *C* was not able to express full dominance in these kernels though in crosses with another *C* allele, no spots were evident.

On testing these colored kernels with spots from the cross *c-ruq/c-ruq* x *C/c-ruq Uq/-*, the following progeny were observed.

| Colored(163) | | colorless(165) | |
|---|---|---|---|
| no spots | spots | no spots | spots |
| 89 | 74 | 78 | 87 |

The colored-no spots kernels are clearly distinguishable from the colored-spots kernels. Unless there is a closely linked color suppressor with *Uq*, it appears there is a coincident suppression of this *C* allele by *Uq*. It is similar in phenotype and effect with the *A-m(r)* alleles found in the Cuna series (MGCNL 50:59).

Peter A. Peterson

### *vp1* linkage with B-A translocations

In crosses of B-A translocations (provided by J.B. Beckett) with the recessive mutant *vp-m451*, the kernels will be dormant and spotted or viviparous and colored when *vp-m451* is distal to the translocation. Tests were conducted as follows:

| | |
|---|---|
| TB-3La | uncovered |
| TB-3Lc | not uncovered |

| | |
|---|---|
| TB-3Ld | uncovered |
| TB-3Lf | no test |
| TB-3Lg | uncovered |
| TB-3Lh | uncovered |
| TB-3Li | not uncovered |
| TB-3Lj | not uncovered |
| TB-3Lk | not uncovered |
| TB-3Ll | not uncovered |
| TB-3Lm | not uncovered |

TB-3La, d, g, and h were previously known to uncover *vp1* (Beckett MNL 58:73); TB-3Lc was previously known not to uncover *vp1* (Beckett MNL 55:27). The best estimate is that *vp1* is located between *lg2* and *ts4* on the long arm of chromosome 3.

Brian E. Scheffler and Peter A. Peterson

### A new dormant allele of *Vp1*

Spotted dormant kernels were isolated when *vp-m451* was crossed to a homozygous *c sh wx* tester stock. It was demonstrated that a colorless dormant allele of *vp1* was segregating in the *c sh wx* stock and the allele was designated *vp-c821708*. *vp-c821708* is similar to the colorless dormant allele discovered by Robertson (MNL 39:104) in that dormancy is not always complete when these alleles are homozygous or heterozygous with other recessive alleles of *vp1*.

Brian E. Scheffler and Peter A. Peterson

### *vp-m451*, a mutable allele of *vp1* that responds to *Cy*

Mutable viviparous kernels were uncovered in the progeny of a self-pollinated ear in a colored line that was derived from a population that had mutator activity. The mutant was identified as being an allele of *vp1* and was designated *vp-m451* (Scheffler and Peterson MNL 58:5). Mutability of this allele is under the control of the independent element *Cy* (Schnable and Peterson MNL 58:9) and thus designated *vp-rcy*.

The relation of *vp-m451* to *Cy* is illustrated below, where receptor alleles of known transposable element systems were tested with the regulatory element responsible for the viviparous mutability.

| Receptor element | System tested | Mutability |
|---|---|---|
| *bz-m4* and *C Ds* | *Ac* | – |
| *o2-m(r)* | *Bg* | – |
| *bz-rcy* | *Cy* | + |
| *a-mdt* | *Dt* | – |
| *a-m(r)* and *a-m1* | *En* | – |
| *a-mrh* | *Mrh* | – |
| *bz-mut* | *Mut* | – |
| *a-ruq* and *c-ruq* | *Uq* | – |

Brian E. Scheffler and Peter A. Peterson

### *Uq* controlled mutable allele at the *c* locus

*c-m804531* is a previously unreported *c* unstable mutant from the 1979 *c sh wx* isolation plot. This plot contained *Uq* and its receptor *a-ruq* (Peterson and Friedemann, Maydica 28:213-249,1983), and produced $3.1 \times 10^6$ kernels. This mutant is spotted with a colorless background and the kernel proved to contain one *Uq* in a cross with *a-ruq*: *(c-m Sh Wx/c sh wx; A; Uq/+* x *C a-ruq)* x *C a-ruq*.

The *Uq* relationship was established by backcrossing *a-ruq* spotted and colorless kernels to the *c sh wx* tester. If *c-m804531* is *Uq* controlled only the *a-ruq* spotted selections will express *c* mutability. This is confirmed in Table 1A. Table 1B shows that the colorless *a-ruq* selections do not express the spotted *c-m* phenotype; instead they express a colorless phenotype.

Table 1. *Uq* relationship test for *c-m804531*. Frequency of ears showing *c* mutability versus colorless from the cross

c-m Sh Wx a-ruq / C Sh Wx a-ruq (+/-)Uq X c sh wx A / c sh wx A

| Cross (1984-family #'s) | *c* locus mutable | colorless |
|---|---|---|
| A. *a-ruq* spotted selection | | |
| c-m Sh Wx a-ruq Uq / C Sh Wx a-ruq + | | |
| 1. 3228 | 4 | 0 |
| 2. 3236 | 5 | 0 |
| B. *a-ruq* colorless selection | | |
| c-m Sh Wx a-ruq + / C Sh Wx a-ruq + | | |
| 1. 3229 | 0 | 7 |
| 2. 3237 | 0 | 5 |

Independent segregation of *Uq* and the *c* locus is demonstrated by sib mating colorless round and colorless shrunken progeny of *c-m* by *c sh wx*:

```
c-m Sh Wx A Uq     c sh wx A
-------------- x   ---------
c   sh wx A +      c sh wx A
        |
        v
   1/4  spotted     c-m Sh Wx A Uq
                    c   sh wx A +

   1/4  colorless   c-m Sh Wx A +
        round       c   sh wx A +
                                        sib mate
   1/2  colorless   c sh wx A Uq/+
        shrunken    c sh wx A   +
```

Nine out of nineteen sib matings produced the *c-m804531* spotted phenotype. *c-m804531* is due to the interaction of *c-ruq* (*c* responsive to *Uq*) and *Uq*.

Elizabeth E. Oberthur and Peter A. Peterson

### System relationships of the *Cy* transposable element system including tests against Robertson's Mutator System

The *Cy* responsive *bz-rcy* receptor (Schnable and Peterson, MGN 58:9) is not triggered to produce spots by any of the following regulatory elements: *Ac, Bg, c2-m3, Dt, En, Fcu, Mrh, Mut, Uq*. Similarly, *Cy* does not trigger mutability at *Ac, Bg, Dt, En,*

*Fcu, Mrh, Mut,* or *Uq* responsive alleles. These data are summarized in Table 1.

Tests of *Cy* versus Robertson's Mutator place *Cy* as a newly described system subject to a negative relationship to Robertson's Mutator. In order to test this relationship five plants from Mutator stocks homozygous for *bz-o* were assayed for *Cy* content by crosses to *bz-rcy/bz-o* (cross 1). Simultaneously, these plants were tested for Mutator activity via Robertson's seedling test. These results are shown in Table 2. These data can not rule out the possibility that *bz-rcy* responds to the same transposable element responsible for Robertson's Mutator phenomena *(Mu1)*.

The appearance of *Cy* in Mutator stocks (Table 2) is in itself of interest as *Cy* had not previously been observed in stocks unrelated to *bz-rcy* (see, however, Scheffler and Peterson MGN 59 and the following note). The pedigree of the population (1963 1005/828) from which *bz-rcy* arose (Schnable and Peterson, MGN 58:9) has been traced back to 1952. It has never been crossed by Mutator stocks. Plants derived from Mutator stocks which displayed *Cy* activity in cross 1 contain many *Cy* elements based on the segregation ratios of spotted bronze:not spotted bronze.

Table 1. Tests of the interaction of the Cy system with the Ac, Bg, c2-m3, Dt, En, Fcu, Mrh, Mut and Uq transposable element systems.

Other regulatory elements against bz-rcy

| | Ac | Bg | c2-m3 | Dt | En | Fcu | Mrh | Mut | Uq |
|---|---|---|---|---|---|---|---|---|---|
| bz-rcy | (-) | (-) | (-) | (-) | (-) | (-) | (-) | (-) | (-) |

Other receptors against Cy

| | bz-m4 | o2mr | a-mdt | a2m(r) am-1 a-m(r) | rcu | a-mrh | bz-mut | a-ruq |
|---|---|---|---|---|---|---|---|---|
| Cy | (-) | (-) | (-) | (-) | (-) | (-) | (-) | (-) |

(-) refers to the absence of interaction

Table 2. Results of the simultaneous assay for Cy and Mutator activity in plants originating from Mutator stocks and unrelated to bz-rcy.

| Plant # | Cy content (Cross 1) | Mutator activity (seedling test) |
|---|---|---|
| 1 | + | + |
| 2 | + | + |
| 3 | - | - |
| 4 | - | - |
| 5 | + | + |

The presence of many genetically active *Cy* in Mutator stocks is consistent with the hypothesis that *bz-rcy* is responding to the element responsible for the Mutator phenomena, which must be present in high copy numbers. Tests are currently underway to assay *Cy* stocks for the presence of *Mu1* homologous sequences.

Patrick S. Schnable and Peter A. Peterson

**Distribution of genetically active *Cy* elements**

The *Cy* content of various lines was assayed by crosses with *bz-rcy*. If the line to be tested was *Bz/Bz*, as most are, the $F_1$ was backcrossed to *bz-rcy*. The appearance of colored spots on a bronze background indicates the presence of one or more genetically active *Cy* elements in the stock. The data are shown in Table 1. Due to the small sample size for many of the tests it is not possible to state that particular stocks lack genetically active *Cy* elements. However, *Cy* is clearly not widely distributed in stocks unrelated to the population from which *bz-rcy* arose. The exception is lines derived from Mutator stocks which in some cases have many independently segregating genetically active *Cy* elements present.

Patrick S. Schnable and Peter A. Peterson

Table 1. The results of the test for genetically active Cy in various tester stocks and plant introduction (PI) lines.

| Stock | Presence of Cy capable of triggering mutability at bz-rcy | # of gametes with Cy / Total tested |
|---|---|---|
| Unrelated to bz-rcy | | |
| Fcu, rcu/r-g | - | 0/2 |
| Mrh | - | 0/4 |
| Bg/Bg $o2^{mr}/o2^{mr}$ CI/CI | - | 0/2 |

| | | |
|---|---|---|
| c2-m3/c2 | - | 0/2 |
| Mut, Cshbz$^{o}$/Cshbz$^{o}$ | - | 0/6 |
| Ac, Cshbz$^{o}$/Cshbz$^{o}$ | - | 0/28 |
| line C (color converted w22) | - | 0/4 |
| Cshbz$^{o}$/Cshbz$^{o}$ (Coe origin) | - | 0/100s |
| a-ruq/a-ruq | - | 0/2 |
| bz-m4/bz-m4 | - | 0/16 |
| cShbz$^{o}$/cShbz$^{o}$ | - | 0/18 |
| Aet/Aet | - | 0/4 |
| bz-mut/bz-mut | - | 0/2 |
| PI 213737 | - | 0/2 |
| PI 217678 | - | 0/2 |
| PI 213787 | - | 0/2 |
| PI 213750 | - | 0/2 |
| PI 303881 | - | 0/2 |
| a-m(r)/a-m-1 | (-) | 0/2 |
| Mutator related source 1 | (+) | 64/64 |
| source 2 | (+) | 32/32 |
| source 3 | (-) | 0/12 |
| source 4 | (-) | 0/22 |
| source 5 | (+) | 18/18 |
| Related to bz-rcy | | |
| 63 1005/828[1] related | (+) | 22/24 |

[1] The progenitor population of bz-rcy

## Simultaneous change at *bz* and *Sh* in *bz-rcy, Cy*: *bz-n(rcy)-sh-835211w; bz-n(rcy)-sh-835211x-1*

Two cases of simultaneous *bz-rcy* to *bz-n(rcy)* and *Sh* to *sh* have been observed. Both cases arose from the cycling state *bz-rcy812215* (see following note) out of a population of 1733 *bz-rcy, Cy* gametes.

*bz-n(rcy)-sh-835211w:* In the first case three bronze, non-spotted shrunken kernels *(835211w-1, w-2, w-3)* were isolated as an ear sector from Cross 1: *C Sh bz-rcy; Cy* x *C sh bz;* no *Cy* and as such most likely arose as a single *Sh* to *sh* event. The heritability of the shrunken phenotype was confirmed by crossing these exceptional kernels by *bz-m4* (Cross 2). The absence of round kernels in the resulting ears confirms that the *Sh* to *sh* event caused a heritable change.

The absence of spots on the original bronze shrunken isolates was unexpected since Cross 1 contained many *Cy* elements. This is suggestive that coincident with the *Sh* to *sh* event a *bz-rcy* to *bz-n(rcy)* (non-responsive to *Cy)* event occurred. In order to test this possibility bronze shrunken kernels from Cross 2 were crossed by *bz-rcy.* Colored spots on the bronze background of round kernels confirmed that *Cy* was present in the original shrunken isolates *835211w-1* and *835211w-3* and that *bz-rcy* changed to *bz-n(rcy)*.

*bz-n(rcy)-sh-835211x-1:* In the second case a single bronze shrunken, non-spotted kernel was found on an ear resulting from Cross 1. This new *sh (bz-n(rcy)-sh-835211x-1)* was handled in the same fashion as described above. The results were the same; *bz-n(rcy)-sh-835211x-1* is heritable and coincident with the *Sh* to *sh* event and *bz-rcy* to *bz-n(rcy)*.

The likely sequence of events that caused these two cases of simultaneous loss is as follows:

1. Trans-active signals from an independently segregating *Cy* element induced the *rcy* receptor element at the *bz-rcy* allele to transpose.
2. Excision was imprecise, resulting in the production of a *bz* allele that no longer responds to *Cy*. Simultaneously with this loss of receptivity, two map units distal to the *Bz* locus, *Sh* changed to *sh*.

Patrick S. Schnable and Peter A. Peterson

## Cycling states in the *Cy* transposable element system: Tissue specificity of two states of *bz-rcy* that cycle in the presence of *Cy*

The original isolate of *bz-rcy* displays a fine-high spotting pattern in the presence of the *Cy* regulatory element (Schnable and Peterson, MGN58:9). Numerous states of the receptor element have been rescued from this original isolate which differ in the timing, frequency and tissue specificity of their mutability.

Two cycling states of *bz-rcy* have been characterized that arose out of *bz-m805137,* the original isolate of *bz-rcy. bz-rcy824325w* has a low pattern of late mutations. In aleurone tissue events occur which signal *bz-rcy824325w* to cycle back to a high state resulting in clusters of frequent mutations surrounded by regions of low frequency spotting (Figure 1). No germinal reversions to a high state have been observed.

Table 1: Anther fluorescence of bz-m(nr) derivatives of bz-m 826301 of independent origin.

| Allele | # of plants sampled | genotype of sampled plants | anther fluorescence[1] |
|---|---|---|---|
| bz-m(nr) 83g123-21 | 2 | bz-m(nr)/$bz^{O}$ | + |
| bz-m(nr) 83g123-22 | 3 | bz-m(nr)/$bz^{O}$ | + |
| bz-m(nr) 83g123-24 | 4 | bz-m(nr)/$bz^{O}$ | mild + |
| bz-m(nr) 83g123-25 | 5 | bz-m(nr)/$bz^{O}$ | + |
| control | 15 | $bz^{O}$/$bz^{O}$ | + |
| control | 3 | Bz/Bz | - |
| control | 7 | bz-m4/bz-m4 | mild + |

[1] + refers to a level of fluorescence indistinguishable from the fluoescence observed from $bz^{O}$/$bz^{O}$ anthers. Mild + is clearly distinguishable from +. It resembles the mild fluorescence observed from bz-m4/bz-m4 anthers(Schnable and Peterson, MGN 58:9). - refers to the complete absence of fluorescence.

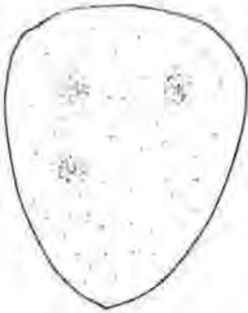


**Figure 1. The pattern conditioned by *bz-rcy824325w* in the presence of *Cy*.**

**Figure 2. The pattern of aleurone spotting conditioned by *bz-rcy812215* in the presence of *Cy*.**

*bz-rcy812215* shows a reduced frequency of spotting in aleurone tissue when compared to the original *bz-rcy* isolate. In contrast to *bz-rcy824325w* no reversions to a high state occur in aleurone tissue (Figure 2). However, germinal reversions to a high state occur among approximately 1% of the *Cy*-containing *bz-rcy812215* gametes. These high states are phenotypically indistinguishable and heritable. Ear sectors of reversion have been observed demonstrating that reversions of *bz-rcy812215* can occur early in ear development but not in aleurone tissue.

Patrick S. Schnable and Peter A. Peterson

## Anther fluorescence of several non-responsive bronze derivatives of the autonomous *En* controlled *bz-m826301*

Bronze non-responsive *(bz-m(nr))* derivatives have been isolated from the autonomous *En*-controlled *bz-m826301* (Peterson, MGN:58:3). These were characterized as to anther fluorescence under UV irradiation. The results are shown in Table 1.

Patrick S. Schnable and Peter A. Peterson

## Update on recessive brown seed mutant *(brn)*

In 1984 (MGNL58:18), a putative *Mu*-induced, seedling lethal, recessive brown aleurone mutant was described. The scutella of mutant kernels are also brown. This past summer, crosses were made to characterize this mutant further and to locate it to chromosome arm.

A series of B-A translocations was crossed onto outcross progeny of plants heterozygous for *brn*, and it was found that TB-3Sb uncovers the brown kernel locus. The kernels with hypoploid endosperm were quite small or defective. Since crosses of TB-3Sb onto sibling plants not segregating for *brn* did not produce small, defective kernels, and since the brown kernels of selfed *brn* heterozygotes are completely filled, the presence of defective brown kernels in TB-3Sb crosses may indicate a deleterious hemizygous effect of *brn* in the endosperm. The scutella of these defective kernels were also brown, suggesting that the pigmentation in the scutellum of hemizygous *brn*-endospermed kernels might be the result of diffusion of the brown pigment from the aleurone and/or endosperm into the scutellum. From the same TB-3Sb crosses, some yellow germless or nearly germless kernels were observed. The latter seeds had very defective brown embryos. These kernels may be those that are hemizygous for *brn* in the embryo, again suggesting a possible deleterious hemizygous effect. It would also appear that at least some synthesis of brown pigment in the embryo is possible. Very few germless or defective kernels were observed on the selfed second ears from these same plants.

The results from the TB-3Sb cross suggest that the brown pigment may be produced in all of the endosperm tissue. In *brn* seeds the aleurone is dark brown, however, the endosperm is also brown but not as darkly pigmented as the aleurone. It could be that the aleurone is the source of all pigment production, which diffuses through the endosperm to the embryo. It is also possible, however, that all endosperm cells produce the pigment, and that the different intensity of pigmentation in the aleurone and endosperm is due to the difference in cellular morphology (e.g., aleurone with small cells capable of being intensely pigmented vs. the large endosperm cells gorged with starch granules). If all the endosperm cells are capable of synthesizing this pigment, this mutant probably should be renamed brown endosperm.

Crosses to determine linkage to genes in the short arm of chromosome 3 will be made this winter and next summer.

Yellow kernels from a selfed ear segregating for *brn* were grown in 1983, and the plants were selfed and outcrossed to purple aleurone stocks. The progeny plants of crosses with known heterozygote *brn* plants were again selfed. These selfed ears segregated purple mottled, yellow, and brown kernels. Some of the purple mottled seeds were also brown. This result would seem to indicate that the *brn* gene is not involved in the anthocyanin biosynthetic pathway.

Crosses of *brn* to a *y* stock will be made this winter in order to explore the relationship of *brn* to the biosynthesis of carotenoid pigments.

We would be happy to supply seeds to anyone interested in studying the biochemistry of this mutant, whether it be with regard to the nature of the pigment involved or the possibility that it is an auxotrophic mutant.

Philip S. Stinard

**A possible test for putative quantitative alleles of qualitative mutants**

Last year I suggested that some quantitative genes might be alleles of qualitative mutants and that it might be possible to isolate genic DNA for individual quantitative genes (MGCNL 58:10-11, 1984). A more detailed description of this proposed relationship has been submitted for publication.

I would like to suggest here a possible way of testing this proposal. Seed size is probably the trait most amenable to study at the present time since it is an important quantitative trait and there are numerous qualitative mutants that affect seed size. These mutants range from small seeds to those with completely empty pericarps. Neuffer and Sheridan (Genetics 95:929-960, 1980) have reported that 27% of EMS-induced mutants were recessive kernel mutants, and a large proportion of these fell into the defective category. We have found in our *Mutator* stocks that kernel mutants are one of the most prevalent seed mutants. By using a transposable DNA system like *Mu* or *Ac*, etc. to induce qualitative seed-size mutants, it should be possible to locate the genic DNA for many of them. The DNA from these in turn can be used to locate the wildtype alleles (quantitative genes?). If the wildtype alleles are indeed responsible for quantitative variation, then corn varieties that differ markedly in seed size might be expected to have different wildtype alleles at some of these loci, which possibly could be distinguished at the molecular level. In early tests it might be wise to study strains that show extreme differences in seed size, such as Argentine, Strawberry or other small seeded popcorns and a present day dent inbred or maybe even a large-seeded variety, such as Cuzco.

Once the genic DNAs are isolated, a variety of molecular investigations can be initiated. Differences in the DNAs in these diverse lines can be studied by restriction endonuclease mapping and DNA sequencing. Differences in gene expression and regulation also can be studied through the analysis of DNA transcripts, and eventually even differences in the final gene products could be investigated. If indeed a qualitative defective mutant gene is the allele of a wildtype quantitative gene, isolation of the quantitative (wildtype) alleles at this locus from lines differing in seed size should yield genes or gene products, in some instances at least, that differ in measurable ways.

There must be many gene loci other than those more or less directly responsible for seed size (i.e., the *de* loci) that also are involved in the expression of this trait in less direct ways—for example, genes involved in such traits as time of maturity, number of rows of seeds per ear, number of ears per plant, etc. Thus, a study of the defective mutants will not provide the complete picture but has the potential for testing the original hypothesis and, if it proves valid, providing information and material that might be useful in a breeding program.

Donald S. Robertson

**Some observations on the loss of Mutator activity in Mutator lines**

The results of 563 crosses of Mutator stocks that had been made in the years 1973 through the winter crop of 1980-81 have been summarized. Of these crosses 85 (13.1%) of the Mutator parents had lost mutator activity. If one line that had an exceptionally high loss rate (24.4%) is excluded, the rate becomes 11.8%. It has been shown (MGCNL 51:36, 1977) that not all apparent losses are real. Occasionally, the sample of 50 outcross plants used to score for the presence of Mutator activity will not include a plant carrying a mutant, as a result of sampling error. If larger samples of outcrossed plants are tested such "sampling error" *Mu*-loss events can be

recognized. A limited test of 13 apparent *Mu*-losses revealed that 5 were due to sampling error. This is not a large enough sample to establish a frequency for such misclassifications but it does serve to demonstrate that they are not insignificant. Because of this uncertainty, it is difficult to estimate the true *Mu*-loss rate. The value of 13.1% determined for 563 crosses is surely an overestimate because most of these *Mu* losses were not tested further to determine whether or not they were true losses. The observation that one line, mentioned above, has a 24.4% loss rate suggests that different stocks may differ with respect to this phenomenon. In light of the uncertainties, we have suggested a value of 10% as the best estimate of the loss frequency.

To test whether *Mu*-loss plants had some system (genetic or otherwise) that in some manner inhibited transposition of the *Mu1* transposon, active Mutator plants were crossed as males to first generation *Mu*-loss stocks. The $F_1$'s were crossed to standards and the resulting progeny were scored for presence of new mutants (Table 1).

Table 1. Mutator activity in Mu-loss/Mu $F_1$ plants.

| O.C. Family* | Total | Total Mutants | % Total Mutants | Total Different Mutants | % Total Different Mutants |
|---|---|---|---|---|---|
| 80-81-1530 | 48 | 16 | 33.3 | 5 | 10.4 |
| 80-81-1531** | 40 | 13 | 32.5 | 2 | 5.0 |
| 80-81-1532 | 49 | 9 | 18.4 | 4 | 8.2 |
| 81-9031 | 47 | 4 | 8.5 | 3 | 6.4 |
| 81-9032 | 48 | 0 | - | 0 | - |
| 81-9033 | 48 | 2 | 4.2 | 1 | 2.1 |
| 81-9220 | 43 | 0 | - | 0 | - |
| 81-9221 | 41 | 5 | 12.2 | 3 | 7.3 |
| 81-9222 | 42 | 7 | 16.7 | 4 | 9.5 |
| 81-9251 | 45 | 7 | 15.6 | 4 | 8.9 |

*Each outcross family has different parents.

**Most of the mutants in this outcross were of a dubious nature 9 were 2nd leaf yellow green mutants and 2 were poor pale greens. These phenotypes could be the result of environmental factors (e.g. disease). In most instances only a few "mutant" seedlings were observed in families of about 50 seedlings. At most, only one probably was a true mutant since it was a mutable pale green.

It does not appear that the *Mu*-loss parent contributed anything that induced the inactivation of Mutator. Some of the above outcrossed plants (i.e. stand. x (*Mu*-loss/*Mu*)) were again outcrossed as males to determine if there was a delayed effect of the original *Mu*-loss parent on the activity of *Mu*, or if there was any evidence of Mendelian segregation for the *Mu* phenotype (Table 2).

Only three 1980-81 families were tested. Two of them (i.e. 80-81-1530 and 1532) appear to be low *Mu*-loss lines, although the numbers are not large enough for determining the frequency of *Mu*-loss with any accuracy. Most of the outcrosses from family 80-81-1531, however, did not segregate for new mutants, indicating that their 80-81-1531 parents had lost Mutator activity. The level of Mutator activity in the parent of the 80-81-1531 family can not be determined with certainty. At most, the parent appears to be a weak mutator, which may account for the high *Mu* loss frequency in the next generation.

It is obvious that Mutator activity frequently can be retained after crossing active Mutator plants with those that have lost *Mu* activity. The one instance (involving family 80-81-1531) where there might be an effect of the *Mu*-loss condition on *Mu* may have nothing to do with the *Mu*-loss parent but may just represent an ordinary *Mu* loss phenomenon. Thus there is no convincing evidence that *Mu*-loss stocks possess an inhibitor for *Mu1* transposition.

Table 2. Mutator activity in plants of the first outcross generation of Mu-loss/Mu plants i.e., stand/ (Mu-loss plants x Mu)

| O.C. Family Numbers* | Family No. of Stand/Mu-loss/ x Mu) parent | Total | Total Mutants | % Total Mutants | Total Different Mutants | % Total Different Mutants |
|---|---|---|---|---|---|---|
| 81-9156 | 80-81-1530 | 38 | 12 | 31.6 | 5 | 13.2 |
| 81-9159 | " | 41 | 7 | 17.1 | 4 | 9.8 |
| 81-9163 | " | 42 | 14 | 33.3 | 6 | 14.3 |
| 81-9154 | 80-81-1531 | 34 | 10 | 29.4 | 3 | 8.8 |
| 81-9157 | " | 38 | 3 | 7.9 | 2 | 5.3 |
| 81-9160 | " | 35 | 0 | - | 0 | - |
| 81-9161 | " | 34 | 0 | - | 0 | - |
| 81-9162 | " | 29 | 0 | - | 0 | - |
| 81-9164 | " | 31 | 0 | - | 0 | - |
| 81-9209 | " | 46 | 0 | - | 0 | - |
| 81-9155 | 80-81-1532 | 45 | 0 | - | 0 | - |
| 81-9158 | " | 38 | 13 | 34.2 | 6 | 15.8 |
| 81-9207 | " | 48 | 14 | 29.2 | 5 | 10.4 |
| 81-9208 | " | 46 | 7 | 15.2 | 5 | 10.9 |

*Each outcross family has different parents.

Donald S. Robertson

## Can lines that have lost Mutator activity as a result of inbreeding regain it by outcrossing?

In inbreeding experiments with Mutator *(Mu)* stocks (Mol. Gen. Genet. 191:86-90, 1983), by the $Mu^{16}$ per se generation plants had lost Mutator activity. Molecular studies (unpublished) suggest that at inbreeding levels above $Mu^{8}$ the copy number of *Mu1* levels off and transpositions cease. To test if the *Mu1* inserts are permanently altered when this happens or if the loss of transposition is a transitory phenomenon that is only observed when copy number is maintained at a high level, $Mu^{16}$ per se plants were outcrossed for three generations (1xo.c., 2xo.c., 3xo.c.). By the 3xo.c., if no transpositions are occurring, the level of *Mu1* copies on average should be equivalent to that of $Mu^{4}$ per se, and the *Mu* activity of the 3xo.c. should approximate that of the $Mu^{4}$o.c. (15.54%). Table 1 gives the results of such a series of crosses. The results are not clear cut. In the first series of tests (series a), there seems to be a slight increase in mutation frequency. Certainly it does not approach the frequency previously reported for $Mu^{4}$o.c. However, the single mutants that occurred in 2xo.c. and 3xo.c. are really very questionable. The mutant in the 2xo.c. consisted of one pale green seedling on one ear from which about 50 seedlings were grown. In the 3xo.c. generation, again, one ear had one yellow-green seedling out of about 50 seedlings. There is a definite possibility that both of these "mutant" seedlings are the result of environmental factors rather than *Mu* activity. Neither seedling was mutable, as is the case with many, but not all, *Mu*-induced mutants.

Table 1. Frequency of mutations in the first (1xo.c.), second (2xo.c.) and third (3xo.c.) generations of outcrossing of $Mu^{16}$ per se plants.

| Generation | Total No. of o.c. ears tested | No. of ears segregating mutants | % of ears segregating mutants |
|---|---|---|---|
| Series a | | | |
| 1x | 90 | 0 | 0.00 |
| 2x | 102 | 1 | 0.98 |
| 3x | 89 | 1 | 1.12 |
| Series b | | | |
| 1x | 228 | 0 | 0.00 |
| 2x | 222 | 19 | 8.56 |

In the second series of crosses (series b), the 2xo.c. generation appears to have a sizable frequency of mutation (but not near the 15.14% value observed for $Mu^{4}$o.c.). As with the "series a" test, the 19 putative mutant events that were observed were, for the most part, not clear cut. The frequencies of mutant seedlings were very low (frequently only one or two per ear) and were of such a type (pale green or slightly yellow-green etc.) that they could be due to environmental factors. There was one mutant, however, that was unambiguous. This was an albino that segregated in a reasonable frequency. None of these mutants in "series b" was mutable. As yet the third outcross generation (3xo.c.) has not been tested.

The results of these tests are not as definitive as one would like. It is uncertain as to whether or not, once *Mu1* transposition ceases as a result of high copy numbers, it can again be reactivated. I feel the weight of the evidence is more negative than positive, but this opinion depends upon an admittedly subjective evaluation of what is and is not a *Mu*-induced mutant. Even if one accepts all the "mutants" in question as truly *Mu*-induced, the lower-than-expected frequency suggests that the *Mu1* elements have not recovered their full transposition potential. Thus, I think it is safe to say that when *Mu1* transposition ceases in high copy number lines, the *Mu1* element is modified in some manner that has resulted in the loss of the ability to transpose. Once lost, this ability, at best, is regained only slowly or possibly not at all.

Donald S. Robertson

## The frequency of *a1-Mum* somatic reversions in reciprocal crosses with *a1* testers.

The three *Mu*-induced *a1* mutable mutants *(a1-Mum1, a1-Mum2, a1-Mum3)* have the same very late somatic reversion pattern. This pattern is independent of the doses of *a1-Mum* in the aleurone, being the same whether the mutable allele is present in one, two or three doses. The frequency of reversions, however, is quite variable for all alleles from cross to cross and frequently even among seeds upon the same ear.

Reversion is probably the result of transposition of the *Mu* insert away from the *a1* locus. Evidence is accumulating that suggests that transpositions are dependent upon the number of *Mu1* inserts in the genome. Both too high a number of inserts and too few (below 10) seem to result in the loss of transposition in the germ line (unpublished) and in the aleurone (Walbot, MGCNL 58:188-189, 1984). Whether or not variation in the number of *Mu1* inserts between these two extreme conditions influences the frequency of transpositions is not known. Since the number of *Mu1* elements varies by a factor of two on average in reciprocal crosses of *a1-Mum* to *a1* testers, it may be possible to get an estimate of the effect of copy number upon somatic transposition. The fact that the *a1-Mum* allele also varies by a factor of two

Table 1. Intensity of somatic Mutator activity in reciprocal crosses.

| | a1Mu allele | Mutability Score ♀ No. seed | Score | ♂ No. seed | Score | ♀/♂ |
|---|---|---|---|---|---|---|
| 82-2149-1 | $a_1$Mum1 | 187 | 3.3 | 262 | 1.8 | |
| 82-4129-7 | " | 227 | 3.3 | 171 | 2.0 | |
| 82-4129-10 | " | 224 | 2.6 | 277 | 1.4 | |
| 82-2150-4 | " | 119 | 2.6 | 147 | 2.3 | |
| 82-2150-5 | " | 188 | 1.5 | 237 | 1.0 | |
| 82-2150-6 | " | 288 | 2.4 | 121 | 1.4 | |
| 82-2150-7 | " | 244 | 2.1 | 165 | 1.1 | |
| 82-2151-2 | " | 169 | 2.4 | 152 | 1.8 | |
| 82-2151-3 | " | 173 | 3.0 | 202 | 1.6 | |
| 82-2151-4 | " | 161 | 2.9 | 160 | 2.2 | |
| 82-2151-8 | " | 248 | 2.3 | 151 | 1.2 | |
| Total | $a_1$Mum1 | 5,748 | 2.58 | 3,263 | 1.58 | 1.63 |
| 2152-4 | $a_1$Mum1 | 264 | 7.6 | 60 | 3.9 | |
| 2152-7 | " | 170 | 5.0 | 127 | 3.9 | |
| 2153-9 | " | 250 | 7.4 | 156 | 3.3 | |
| 2154-10 | " | 150 | 7.6 | 121 | 8.0 | |
| 2155-1 | " | 274 | 5.3 | 138 | 4.2 | |
| 2155-2 | " | 116 | 4.6 | 301 | 2.7 | |
| 2155-4 | " | 270 | 4.8 | 171 | 4.2 | |
| 2155-5 | " | 286 | 5.4 | 207 | 2.2 | |
| 2155-7 | " | 201 | 4.4 | 180 | 3.4 | |
| 2156-6 | " | 197 | 7.4 | 203 | 5.3 | |
| 2156-7 | " | 225 | 7.2 | 236 | 4.4 | |
| 2156-8 | " | 227 | 8.3 | 235 | 6.7 | |
| 2156-9 | " | 138 | 7.6 | 229 | 4.3 | |
| 2157-2 | " | 164 | 7.6 | 210 | 5.1 | |
| 2157-3 | " | 139 | 7.1 | 120 | 1.9 | |
| 2157-5 | " | 235 | 6.5 | 302 | 5.3 | |
| 2157-9 | " | 255 | 1.0 | 171 | 1.1 | |
| Total | $a_1$Mum1 | 21,268 | 6.07 | 13,153 | 4.15 | 1.46 |
| 2161-2 | $a_1$Mum1 | 255 | 4.4 | 222 | 3.9 | |
| 2161-3 | " | 204 | 4.2 | 215 | 4.1 | |
| 2161-4 | " | 226 | 6.7 | 232 | 4.7 | |
| 2161-5 | " | 139 | 7.4 | 46 | 3.5 | |
| 2161-7 | " | 260 | 4.8 | 211 | 4.8 | |
| 2162-3 | " | 173 | 6.2 | 153 | 5.5 | |
| 2162-4 | " | 223 | 6.0 | 136 | 5.3 | |
| 2162-5 | " | 188 | 5.6 | 125 | 4.8 | |
| Total | $a_1$Mum1 | 9,235 | 5.54 | 6,160 | 4.60 | 1.20 |
| Grand Total | $a_1$Mum1 | 36,611 | 4.91 | 22,576 | 3.45 | 1.42 |

* 10 point scale - 1 = stable mutant, 10 = full color.

complicates the interpretation of the results of such a test. Data from such reciprocal crosses are given in Table 1. The level of mutability (i.e., frequency of transposition) was measured on a 10 point scale with 1 being stable mutant and 10 being full color (purple).

Although there is considerable variation from cross to cross it is obvious that crosses of *a1-Mum* plants as males consistently result in a lower level of mutability than the reciprocal crosses. The female frequency is 1.42 times greater than the male on average, and does not seem to vary in a consistent manner relative to the level of mutability found in these crosses.

Although the female crosses have twice as many *a1-Mum* alleles as the male crosses and probably have on average twice as many *Mu1* sequences, the female reversion frequency is not twice that of the male. Thus, there does not seem to be an additive relationship between the number of copies of the mutated locus or of the *Mu1* inserts. In the studies of the mutator activity of $Mu^1$o.c. and $Mu^2$o.c. (Robertson, Mol. Gen. Genet., 191:86-90, 1983), where the number of copies of *Mu1* insert differs by a putative value of two, the $Mu^2$o.c. has only 1.5 fold higher mutation frequency than $Mu^1$o.c. Again there is not an additive relationship between the putative *Mu1* dosage and transpositions.

In the case of the *a1-Mum* alleles the nonadditive effect of *Mu1* copy number is seen even when the *a1-Mum* parent is crossed as a female, and twice as many *a1-Mum* alleles are available at which transpositions restoring function to this locus can occur. It may be that the depressive effect on the frequency of transpositions resulting from an increase in copy number of *Mu1* in the aleurone more than offsets the increased opportunity for transposition that results from doubling the number of *a1-Mum* alleles.

Donald S. Robertson

**Studies on the nature of the loss of somatic mutability for two *Mu*-induced mutable *a1* mutants**

Three mutable *a1* mutants have been induced by the *Mu* system *(a1-Mum1, a1-Mum2,* and *a1-Mum3).* The frequency of somatic mutability is quite variable for all of these mutants. The timing of the

somatic reversions is quite late for all of them and has not been seen to vary significantly for the several generations that they have been studied. No true germinal reversions have been found as yet but stable mutant phenotypes are quite common. Two stable mutant derivatives have been studied *(a1-Mum1-stable* and *a1-Mum3-stable)*. Stable seeds were crossed to a purple aleurone stock and 9 of the $F_1$ progeny were selfed or pollinated by *a1 sh2*. These $F_1$ plants were at the same time crossed to a purple aleurone *Mu* stock (Pl *Mu)* and a non-*Mu* purple aleurone line (Pl aleur). The Pl *Mu* parent was outcrossed to a non-*Mu* purple aleurone line. None of the selfs or outcrosses to *a1 sh2* of the Pl aleur/*al-Mum1-stable* crosses segregated for mutable seeds. Most of the selfs or outcrosses to *a1 sh2* of the Pl aleur/ *a1-Mum3-stable* gave nothing but stable *a1* seeds, but two ears had one medium mutable seed each. From each stable family, the outcrosses from two plants that had not segregated for any mutable seeds on the selfed or outcrossed ears were grown in the winter of 1983-84. Fifty seeds of each cross were planted and the selfed ears scored for mutability and the segregation of new mutants. The occurrence of new mutants would indicate *Mu* activity in one or both of the parents. A 5-class-scale measure of mutability was used in classifying somatic mutability: 1 stable, 2 low mutability, 3 medium mutability, 4 high mutability and 5 self color (purple). The results are found in Table 1. In column 4 we have a measure of the somatic mutability after the stable derivatives were again outcrossed to Pl aleur and selfed. The mutability value is the average for all *a1* seeds produced on all the ears of the outcross progeny. The numbers in parentheses indicate the total numbers of seeds scored. It is obvious that the stable phenotype is retained through these outcrosses. The results in column 5 reveal whether or not the stable *a1* parent had Mutator activity. Since in none of the selfed progeny of these crosses were any segregating mutants found, there is no evidence that these stable lines have retained Mutator activity. Column 6 measures the mutability observed in *a1* seeds of the Pl *Mu/a1-Mum-stable* crosses; in three of the crosses significant somatic mutability was restored. Thus, it appears that in these three instances something is still present at the *a1* locus that can respond to the *Mu1* elements contributed by the Pl *Mu* parent. In one instance (8197-4) there does not appear to be a response. Perhaps this stable represents a modified insertion at the *a1* locus that now only transposes infrequently. In column 7 the presence of new mutants segregating in the selfs of a cross indicates that one of the parents has Mutator activity. Since the stable *a1* parent has already been shown not to have Mutator activity (column 5), the Mutator activity must have been due to the Pl *Mu* parent of the first two crosses. The results from column 8 confirm that the Pl *Mu* parent indeed had Mutator in the case of the first cross (8186-7). Unfortunately, there was not a separate test of the Pl *Mu* parent for the second cross (8186-10), but since the *a1* stable parent did not have Mutator activity, the activity found in column 7 could only have come from the Pl *Mu* parent. The results from cross number 3 (8197-3) are quite interesting. The Pl *Mu* parent, when tested against Pl aleur for Mutator activity, is shown not to have any. That it had truly lost Mutator activity is indicated by the results in column 7, for there was no evidence of Mutator activity in this cross either. Such results are expected since neither the *a1* stable or Pl *Mu*

Table 1. Tests of alMum1 and alMum3 stable alleles.

| (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|
| | | | crosses | | | | |
| | | | Pl aleur/alMum stable | | Pl Mu/alMum stable | | Pl aleur/ Pl Mu |
| 83 alMum stable parent | Original alMum allele | Average class of stable parent* | Aleurone mutability* | Mutator activity | Aleurone mutability* | Mutator activity | Mutator activity |
| 8186-7 | alMum1 | 1.00 | 1.003 (1400)** | Neg. | 2.17 (960) | Pos. | Pos. |
| 8186-10 | alMum1 | 1.00 | 1.00 (1700) | Neg. | 2.04 (575) | Pos. | No test |
| 8197-3 | alMum3 | 1.00 | 1.05 (868) | Neg. | 3.72 (507) | Neg. | Neg. |
| 8197-4 | alMum3 | 1.00 | 1.05 (2079) | Neg. | 1.09 (586) | Neg. | Neg. |

*5 class scale used: class 1-stable, class 2-low mutability, class 3-medium mutability, class 4-high mutability and class 5-self color.

**Numbers in ( ) are total seeds scored.

tested positive for Mutator activity. However, somatic mutability is restored in the Pl *Mu/a1-Mum3 -stable* in spite of the fact that there is no evidence for *Mu1* transposition (germinal or somatic) in either parent.

These results are in agreement with the evidence that transposition of *Mu1* ceases if the copy number drops below a minimal number. Walbot (MGCNL 58:188-189, 1984) has evidence for this with regard to somatic transpositions at the *bz2* locus. Bennetzen, Morris and Hagenson (unpublished) have evidence that if the *Mu1* copy number falls below 10, germinal transpositions no longer occur. The stables in this study may have less than 10 copies of *Mu1*, thus eliminating somatic and germinal transpositions. When crossed to an active Pl *Mu* stock this parent could contribute sufficient copies of *Mu1* to restore transposition and, if a potentially active *Mu1* element is still at the locus, somatic mutability might be restored. The results of the third cross (8197-3) are of particular significance since, if the above interpretation is correct, both the *a1* stable and Pl *Mu* parent evidently had a *Mu1* copy number below the critical number required for transposition. However, by pooling their *Mu1* elements in the Pl *Mu/a1-Mum3 -stable* cross there resulted sufficient *Mu1* copies to initiate transpositions and hence somatic mutability was restored. Whether or not germinal mutability was restored will have to be seen in the results of the outcross of Pl *Mu/a1-Mum3-stable* plants. That outcross was made this summer and the progeny are in our winter nursery.

Medium mutable seeds (class 2) were selected from one of the selfed ears of the Pl *Mu/a1-Mum1 -stable* cross (8186-7) and planted. The resulting plants were self pollinated (Table 2). The eight ears are almost uniformly class 2 (low). At most, only one stable (class 1) seed was found per ear and that only for 3 ears. A few seeds higher than class 2 were found on two ears. These results give no indication that a dual factor controlling-element-like system is involved in the restoration of mutability to *a1-Mum* stables, similar to *Ac-Ds*. The results suggest that sufficient copies of the *Mu1* element are again present in this one stable line to maintain a discernible pattern of mutability.

Donald S. Robertson

Table 2. Classification for somatic a1Mum1 mutability in the $F_2$ progeny of the cross, Pl Mu/a1Mum1 stable. The $F_1$ seeds planted were medium mutables (class 3).

| Plant # | Mutable class | | | | | | Class Average |
|---|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | | |
| 6174-1 | | 209 | | | | | 2.0 |
| 6174-2 | | 229 | | | | | 2.07 |
| 6174-4 | 1 | 331 | 24 | 1 | | | 2.0 |
| 6174-5 | | 371 | | | | | 2.0 |
| 6174-6 | 1 | 333 | 1 | | | | 2.0 |
| 6174-7 | 1 | 206 | | | | | 2.0 |
| 6174-8 | | 28 | | | | | 2.0 |
| 6174-9 | | 94 | | | | | 2.0 |
| Total | 3 | 1801 | 25 | 1 | | 1830 | 2.01 |

## The relationship between somatic instability of *Mu*-induced mutants and Mutator activity

In order to be sure that a given plant has Mutator activity, the plant must be outcrossed, 50 or more of the outcross progeny selfed, and the selfs scored for the segregation of new mutants. This is a very burdensome procedure. With the advent of mutable aleurone mutants induced by *Mu*, somatic instability may be a possible indicator of *Mu* activity in the germ line. Certainly the mutable phenotype indicates *Mu1* transpositions are occurring in the somatic cells. But is the somatic behavior of *Mu1* correlated with its germinal behavior?

In the previous paper, evidence was presented that the loss of somatic mutability seems to be correlated with the loss of germinal transposition. In addition to the test for Mutator activity in stable derivatives of *a1-Mum* mutants reported in the previous paper, two additional stables also tested out to have lost Mutator activity. Thus it seems that loss of germinal mutability accompanies the loss of somatic mutability. But is the converse true? Does the presence of somatic mutability indicate the presence of germinal mutability? In Table 1 are the results of some tests made this summer. Seeds from outcrosses to a purple aleurone non-*Mu* stock of plants with ears having predominantly seeds with three different levels of mutability, low, medium and high, were sown and selfed. The parental mutability was scored either on selfed ears of the *a1-Mum* parent or on ears of the *a1-Mum* parent that had been pollinated by *a1 sh2*. The selfed-outcross progeny were scored for the presence of new mutants (Table 1).

Table 1. Comparison of Mutator activity with somatic mutability of a1 Mum mutants.

| Family | a1 allele | Predominant mutability class on ears of plants crossed to P1 aleur | Type of pollination on ear of plant crossed to P1 aleur | Estimated** mutability range in the ⊗ progeny of the family* | Total ears harvested | Mutator Activity: Total mutants | % Total mutants | Total different mutants | % Total different mutants |
|---|---|---|---|---|---|---|---|---|---|
| 5059 | a1Mum2 | 4 | ⊗ | 3-4 | 44 | 1 | 2.27 | 1 | 2.27 |
| 5066 | a1Mum2 | 4 | o.c. to a1sh$_2$ | 3-4 | 37 | 5 | 13.51 | 4 | 10.81 |
| 5067 | a1Mum2 | 4 | ⊗ | 1-4 | 49 | 0 | 0.00 | 0 | 0.00 |
| 5063 | a1Mum3 | 3 | ⊗ | 1-2 | 49 | 0 | 0.00 | 0 | 0.00 |
| 5064 | a1Mum2 | 3 | ⊗ | 1-2 | 44 | 0 | 0.00 | 0 | 0.00 |
| 5065 | a1Mum3 | 3 | o.c. to a1sh$_2$ | 1-4 | 37 | 5 | 13.51 | 5 | 13.51 |
| 5057 | a1Mum2 | 2 | ⊗ | 1-2 | 49 | 1 | 2.04 | 1 | 2.04 |
| 5058 | a1Mum2 | 2 | ⊗ | 1-4 | 47 | 0 | 0.00 | 0 | 0.00 |
| 5062 | a1Mum3 | 2 | ⊗ | 1-2 | 44 | 0 | 0.00 | 0 | 0.00 |

*5 class scale used: class 1-stable, class 2-low mutability, class 3-medium mutability, class 4-high mutability, class 5-self color.

**Estimates were made here since we have not yet had time to classify individual seeds on these ears for mutability.

It does not appear that there is any correlation between the level of somatic mutability per se and Mutator activity observed in the germ line. The highest Mutator activity came from the outcross of plants whose ears had been pollinated by *a1 sh2* and showed a high or medium level of mutability. High, medium or low mutability on selfed ears did not appear to be a good predictor of Mutator activity in a plant. Only two out of 8 crosses showed Mutator activity, and that was relatively weak. Why this difference between mutability scored on selfs versus scored on outcrosses? Since the number of plants that were scored after outcrossing rather than selfing is small, this may just be a chance event. However, there is a basic difference between the *Mu1* situation found in a self and that of an outcross. In a self both gametes carry the *a1-Mum* allele and other copies of *Mu1* that are present elsewhere in the genome. Thus the mutable phenotype one observes results from the sum of these two contributions. Any one (male or female) gamete may not have sufficient *Mu1* copies to cause transpositions, but combining the contributions from both the pollen and egg may be sufficient for somatic transpositions to occur. When the plant bearing the selfed ear is outcrossed for the Mutator tests reported here, the low copy number carried in only the male germ line may not be sufficient for transpositions to occur, and hence no mutation would be found in the outcross progeny scored in such a cross. In the case where mutability scoring at the *a1-Mum* locus was done on ears pollinated by *a1 sh2*, only the female gamete contributed the *a1-Mum* allele and any other *Mu1* sequences that might be present. If *a1-Mum* mutability is a function of the number of active *Mu1* segments present, then aleurone instability in this case may be a good predictor since it may more accurately represent the condition of the *Mu1* state in the male germ line. More tests will be required in which the *a1-Mum* parent's mutability is scored by outcrossing to a tester to confirm these results.

It should be noted, however, that even in Mutator tests that showed no Mutator activity there was some somatic mutability found in the outcross progeny; sometimes a significant amount. Thus the mere presence of seeds with somatic mutability in an outcross progeny does not insure that the *Mu* parent of the outcross had Mutator activity.

Donald S. Robertson

**Evidence for Mutator activity in the male gametophyte**

It has been demonstrated conclusively that Mutator does induce mutants premeiotically (Genetics 94:969-978, 1980; MGCNL 58:11-12, 1984). This article will consider some evidence for activity of the Mutator system in the gametophyte. Last year (MGCNL 58:11-12, 1984) we presented the results of a large experiment to determine the *Mu*-induced mutation frequency at the *wx* and *y1* loci. In this test, over 300 putative *y1* mutants were found. In 1983 and 1984, seeds of these putative mutants were sown and the resulting plants were self-pollinated. Out of 283 selfed ears, 25 (8.8%) segregated for yellow seeds. We have not finished classifying these 25, but some are known to have a lower frequency of yellow seeds than would be expected if only *Y1* was involved. The *Mu* parent used in this test was segregating for modifiers of *y1*. These modifiers, when present, produce a pale yellow (instead of white) endosperm. Thus, many mutant seeds were in reality pale yellow rather than white. Most of these proved to be *y1* upon selfing since no yellow seeds occurred on the selfed ears, but they did segregate for pale yellow and white seeds.

There are several possible explanations for the seeds with apparent *y1 y1 y1* endosperms that produce plants that segregate for *Y1:* 1) Environmental

factors resulted in a pale yellow endosperm in the original isolate. 2) The occurrence of a pollen grain with a dominant white or white cap allele. 3) The original *Mu*-induced mutant was unstable and reverted to *Y1* early in the development of the embryo. 4) Simultaneous mutation of *Y1* to *y1* in the two polar nuclei (but not the egg nucleus). Some of these events are more likely than others but, in view of the possibilities, it is not surprising that some ears segregating *Y1* are found. The important point, however, is that such exceptional seeds are rare.

In 1983 we tested for the induction of *y1* mutants in the reverse direction (i.e., using *Mu* plants as males). This test was on a much more limited scale. Twenty-four pale yellow and white seeds were obtained which produced plants that were selfed last summer. Of these, 15 were homozygous for *y1* while nine (37.5%) segregated for *Y1*.

Thus there is a marked difference between the results obtained when mutants are induced in a male *Mu* parent compared to those produced in a female *Mu* parent. In the latter instances, very few putative mutant seeds proved to have embryos carrying *Y1* (8.8%). On the contrary, when the *Mu* parent is used as a male, 37.5% had such discordant seeds. Such a high frequency of discordant seeds in this latter instance would be expected if *Mu* can mutate in the time span between the DNA replication prior to the formation of sperm and the first DNA replication of the triple fusion nucleus giving rise to the endosperm. Mutations may also occur in the female gametophyte but its more complex development, especially that involved in the production of the polar fusion nucleus, insures that such discordant seeds are rarely observed as a result of *Mu* activity in female *Mu* plants.

It may be too early to say with certainty that *Mu*-induced mutants are occurring in the gametophyte but the data are suggestive. We have a large number of additional putative *Mu*-induced *y1* mutants from male Mutator plants that were produced this summer. These will be selfed next summer and should give definitive results.

It would be possible to test for mutations in the female gametophyte that produce the reciprocal class of discordant seeds (i.e., homozygous *y1* embryo in yellow seeds) by growing large numbers of yellow seeds from these crosses in an isolation plot where the male rows would be homozygous for *y1*. If such mutations are occurring, some of the yellow seeds should have embryos with a *Mu*-induced *y1* mutant allele, and homozygous *y1 y1* ears would result.

The same discordant class should be found in the male crosses. In the test with *Mu* plants used as males, the two types of discordant seeds (i.e., *y1 y1 y1* endosperm, *Y1 y1* embryo and *Y1 y1 y1* endosperm, *y1 y1* embryo) should occur in equal frequency. When *Mu* plants, however, are used as females the discordant seeds (i.e., *y1 y1 y1* endosperm, *Y1 y1* embryo and *Y1 Y1 y1* endosperm, *y1 y1* embryo) would not be expected in equal frequency. Yellow seeds with homozygous *y1* embryos are expected in a much higher frequency and should be about equal to the equivalent class in the male test.

Unfortunately, all yellow seeds from both the male and female tests were discarded. These tests will be repeated using *Mu2* per se stocks as the *Mu* parent and *y1 wx gl8* or *y1 wx gl1* plants as the *y1* parent. The use of *Mu2* per se will result in a much higher mutation frequency, permitting the use of smaller populations. Hand pollinations, therefore, will be practical, and exact reciprocal crosses can be made so that comparisons between results of using *Mu* plants as males and females will not be complicated by differences in genetic background.

Donald S. Robertson

**The effect of gamma ray irradiation on Mutator activity**

For the last two summers I have been testing the effects of gamma irradiation on the Mutator system. Shedding central spikes were collected early in the morning just as pollen was beginning to shed. Only plants which were not shedding pollen the previous day were selected. The shedding central spikes were placed in glass test tubes stoppered with a cork. The tassel of each plant sampled was bagged and selfed and outcrossed to produce the controls. The central spikes were exposed to gamma irradiation from a cobalt 60 source sufficiently long to provide dosages of 600, 1,200 and 1,400 rads. The irradiated central spikes were removed from the test tubes (in the fieldhouse) and placed in an earshoot bag. A second earshoot bag was used to cap the first. These samples were placed in a protected sunny spot until pollen was shedding well, and were pollinated on standard silks that had been cut back the day before.

In 1981, a treatment of 600 rads resulted in very little observable effect (Table 1). But there are two slight trends that can be detected: 1) The irradiation slightly reduced the number of ears obtained. For the *Mu* test, 200 seeds from both controls and irradiated crosses were planted. For the standards, 150 seeds of each were sown. Neither the reduction for the *Mu* or the standard crosses is statistically significant. 2) For the *Mu* crosses, there is reduction in the mutation frequency in the irradiated material. This reduction was not statistically significant.

In 1982, plants were irradiated with 1,200 and 1,400 rads. In most of these crosses we obtained good seed set (normal looking ears). Thus I thought we still were not getting much of a biological effect. Therefore, only two *Mu* plants were tested at 1,200 rads and one standard at 1,400 rads (Table 2).

Table 1. The effects of 600 rads gamma irradiation of Mu and standard pollen.

| | Number of seeds Planted | Number of ears harvested | Total Mutants | % Total Mutants | Total Different Mutants | % Total Different Mutants |
|---|---|---|---|---|---|---|
| Mu Control | 200 | 179 | 15 | 8.4 | 12 | 6.7 |
| Mu + gamma | 200 | 142 | 7 | 4.9 | 6 | 4.2 |
| Standard control | 150 | 139 | 1 | 0.7 | 1 | 0.7 |
| Standard + gamma | 150 | 116 | 1 | 0.9 | 1 | 0.9 |

Table 2. Effects of 1,200 rads gamma irradiation on Mu pollen and 1,400 rads gamma irradiation of standard pollen

| | Number of seeds Planted | Number of ears harvested | Total Mutants | % Total Mutants | Total Different Mutants | % Total Different Mutants |
|---|---|---|---|---|---|---|
| Mu controls | 200 | 175 | 28 | 21.7 | 22 | 12.6 |
| Mu + gamma | 200 | 108 | 9 | 8.3 | 4 | 3.7 |
| Standard controls | 100 | 86 | 0 | - | 0 | - |
| Standard + gamma | 200 | 63 | 0 | - | 0 | - |

Again, as with the lower doses, there is a reduction in the number of ears produced. For the *Mu* crosses 200 seeds were sown, and for the standard crosses 100 control and 200 seeds from irradiated pollen were planted. The reduction in number of ears recovered is statistically significant at the 1% level in both sets of crosses. In the *Mu* crosses, the reduction in mutant frequency in the outcrosses from irradiated pollen is significant at the 1% level.

Some gamma-ray-induced recessive seedling mutants could be expected in the progeny from irradiated pollen of standard plants, but none were observed. However, a sample of only 63 plants is too few for a good test.

As far as Mutator activity is concerned, gamma irradiation markedly reduces the frequency of mutants obtained in the outcross progeny. Why is there this reduction? I believe it might be telling us something about the timing of Mutator activity. It has been shown that Mutator acts late in development (Robertson, Genetics 94:969-978, 1980; MGNL 58:11-12, 1984). Small sectors of allelic mutants have established this fact. However, many *Mu*-induced mutants are singular events. Some of these may be postmeiotic (gametophytic or zygotic). It may be that gamma irradiation inactivates the Mutator system in pollen so that no or a greatly reduced number of mutations are taking place between the time of pollen shedding and the first division of zygotic DNA. Thus, the mutations we observe in the irradiated *Mu* crosses could be predominantly those that occurred before pollen maturation. The synergistic response between *Mu* activity and U.V. (MGNL 56:2-4, 1982; 58:19-20, 1984) also involves pollen irradiation and hence is active in the same developmental stages as the gamma irradiation. Thus both types of irradiation act in the same time frame. It is improbable that *Mu* transpositions occur in the sperm since no DNA replication is occurring. However, the direct effects of irradiation impinge upon the sperm. It is likely that the irradiation damage is repaired in the zygote at the time of the first DNA replication. This is also the first opportunity for *Mu1* transpositions to occur. If, at this time, *Mu1* transposes to an unreplicated portion of a chromosome, a plant could result that is heterozygous for a new *Mu*-induced mutant. The results from U.V. and gamma irradiation suggest that such mutations do occur, and that changes induced in the male genome by both types of irradiation have a marked but different effect on the frequency of such mutants.

Perhaps gamma irradiation is completely inactivating *Mu* and hence eliminating the zygotic transposition. Tests will be made next year to determine if the gamma irradiation effect is a transitory or permanent change. If the latter, gamma irradiation may provide a method for eliminating *Mu* transposition and thus facilitate the location of genic DNA by *Mu* mutagenesis.

This last summer we performed similar experiments with gamma dosages of 1600 and 1800 rads. In these crosses, a marked reduction in seed set was observed and hence greater biological effects can be expected.

Donald S. Robertson

### Putative *Mu* induced deficiencies

When I was a guest in Dr. Mel Green's laboratory at Davis in the 1980-81 school year, we had a discussion about the possibility of Mutator inducing deficiencies, and he suggested that the short arm of chromosome nine (especially the *yg2* locus) might be used to look for deletions in a systematic way. This was just one of those "why didn't I think of that" ideas that was too good not to follow up. In the summer of 1981, we increased our stock of *yg2* and in the summer of 1982 we set up a large isolation plot in which *Mu* plants were used as females and homozygous *yg2* plants served as males. In 1983 at

planting time, we used all the seedling growing space we could obtain to grow seedlings of this cross. Out of 779,213 seedlings, 125 yellow-green seedlings were obtained (freq. $1.6 \times 10^{-4}$). These were transplanted to the field (we lost only one or two plants in transplanting). Most plants were strong enough to get reciprocal crosses to standard stocks. In the 1983-84 winter nursery, seeds from 10 crosses were sown in which the mutant plants (putative *yg2-Mu/yg2)* were used as females in crosses with standards and the resulting plants were reciprocally crossed to *yg2 yg2* plants. If the *yg2-Mu* mutant involves a deficiency, about half of the plants in a given family carrying the *Mu*-induced allele could give 1:1 *Yg2:yg2* ratios when used as a female, but no yellow-green seedlings or less than 50 percent yellow-green seedlings when used as a male. The other half of the plants should give 1:1 ratios in both directions because they would be carrying the standard *yg2* allele. If the original *Mu*-induced *yg2* allele does not involve the production of a deficiency, then all plants will segregate 1:1 in both directions of the reciprocal cross. Of the ten families with *Mu*-induced mutants tested in 1983-84, two had plants that gave off ratios when the plants heterozygous for the putative deficiency mutants were used as males but not as females (Table 1).

**Table 1. Reciprocal crosses of families in which about half of the plants are heterozygous for a putative *Mu*-induced *yg2* deficiency (i.e., *yg2-Mud/Yg2)***

| Family | Heterozygous parent used as ♀ | | ♂ | |
|---|---|---|---|---|
| | Yg | yg | Yg | yg |
| 8626-1 | 100 | 90 | 15 | 21 |
| -2 | 34 | 35 | all green | |
| -3 | all green | | all green | |
| -5 | 12 | 9 | 23 | 30 |
| -6 | no ear | | 77 | 59 |
| -8 | 17 | 25 | no ear | |
| -9 | 24 | 20 | no ear | |
| -10 | 78 | 105 | 76 | 64 |
| -11 | 21 | 23 | no ear | |
| -12 | all green | | all green | |
| 8628-1 | 121 | 102 | 110 | 31 |
| -3 | all green | | no ear | |
| -7 | 36 | 30 | 70 | 21 |
| -8 | 36 | 53 | 27 | 26 |
| -9 | 91 | 77 | no ear | |
| -10 | 109 | 85 | no ear | |
| -11 | 117 | 117 | 95 | 59 |

Plant number two of 8626 behaved as would be expected for a deficiency that is not male transmittable. In family 8628 plants 1, 7 and 11 gave crossing patterns expected if there is a deficiency that has reduced transmission through the male.

We must pause to comment on the crosses where the putative heterozygous *yg2-Mud* plants used as females gave only green progeny. In all cases where this occurred, the reciprocal (if made) cross also gave only green. Such a phenomenon was observed in 5 of the families tested. The number of such plants in these families were as follows:

| Total plants tested in the family | Number of instances of all green ♀ progeny |
|---|---|
| 8 | 1 |
| 10 | 3 |
| 7 | 2 |
| 7 | 1 |
| 10 | 2 |

All of these families came from crosses where the female was a mutant (i.e., *yg2-Mu/yg2)* that retained a strong yellow-green phenotype at maturity. Thus the "all green" class can not be explained by contamination. In all cases, the *yg2* tester parent of the "all green" crosses was used in other crosses that gave positive *yg2* tests. Thus the *yg2* tester parent was indeed *yg2*. It could be that the original yellow-green mutant seedling and plant was a pseudo-yellow-green due to some undefined environmental factor(s). If so, this phenotype persisted to maturity. These also could be the result of back mutation of the *Mu*-induced allele carried by the gametes of the original *yg2-Mu/yg2* plant. This explanation seems plausible for most families where this class occurred, but seems unreasonable for family 8626 where there is evidence that a deletion is involved. It is possible that in this family the deletion is outside of the mutated *yg2* genic DNA. Larger numbers of sibling plants will need to be tested from these families with "all green" individuals to determine the frequency with which these classes occur.

In 1984, we grew 33 more of the *(yg2-Mu/yg2)/* stand. crosses to be tested by reciprocally crossing with *yg2* (Table 2). There are nine *Mu*-induced

**Table 2. Results of the 1984 reciprocal test crosses of putative *Mu*-induced *yg2* in which there was evidence of the presence of a deficiency.**

| Family | No. of plants in the family | Reciprocal crosses of putative deficient plants used as ♂ | | ♀ | |
|---|---|---|---|---|---|
| | | Yg2 | yg2 | Yg2 | yg2 |
| 6044 | 8 | 46 | 11 | 31 | 24 |
| 6045 | 9 | All | 0 | 26 | 17 |
| | | All | 0 | 21 | 10 |
| | | All | 0 | 20 | 24 |
| | | All | 0 | 21 | 17 |
| | | All | 0 | 22 | 21 |
| | | All | 0 | 35 | 14 |
| 6046 | 9 | All | 0 | 34 | 16 |
| | | All | 0 | 32 | 18 |
| | | All | 0 | 29 | 10 |
| 6047 | 8 | All | 0 | 25 | 27 |
| | | All | 0 | 21 | 27 |
| | | All | 0 | 29 | 10 |
| 6049 | 10 | All | 0 | 26 | 10 |
| | | All | 0 | 21 | 21 |
| | | All | 0 | 24 | 18 |
| 6050 | 9 | 38 | 15 | 25 | 24 |
| | | 27 | 18 | 19 | 21 |
| 6053 | 10 | 29 | 19 | 21 | 19 |
| | | 28 | 18 | 23 | 16 |
| 6055 | 10 | All | 0 | 24 | 18 |

| | | | | | |
|---|---|---|---|---|---|
| 6057 | 10 | 14 | 9 | 22 | 18 |
| | | 44 | 10 | 23 | 26 |
| | | 39 | 9 | 24 | 17 |
| | | 34 | 13 | 24 | 18 |
| | | 35 | 18 | 23 | 20 |
| 6061 | 10 | All | 0 | 30 | 19 |
| 6063 | 8 | 36 | 11 | 25 | 24 |
| | | 41 | 11 | 25 | 17 |
| | | 56 | 1 | 29 | 17 |
| 6067 | | All | 0 | 26 | 23 |
| | | All | 0 | 20 | 19 |
| | | No test | | All | 0 |
| | | All | 0 | All | 0 |
| 6073 | 10 | All | 0 | 21 | 18 |
| | | All | 0 | 25 | 24 |
| | | All | 0 | 26 | 24 |
| 6074 | 10 | All | 0 | 32 | 24 |
| | | All | 0 | 15 | 10 |
| | | All | 0 | 25 | 20 |
| 6075 | 10 | 45 | 15 | 22 | 23 |
| | | 65 | 3 | 20 | 19 |
| | | 50 | 6 | 19 | 28 |
| | | 61 | 12 | 25 | 23 |

events involving the *yg2* locus that appear to involve large enough deficiencies to eliminate male transmission. Six of the *Mu*-induced events might be smaller deletions. In two families, 6046 and 6061, it appears that the deficiency may be of sufficient size to interfere with female transmission as well. In all other families there appears to be no consistent evidence that female transmission is affected.

There are of course other phenomena that might account for these transmission patterns, such as gametophyte factors or mutations that induce pollen lethals. This winter we are crossing these putative deficient plants from these families with TB-9b, *wd* and *yg2*. If the off ratios are due to deficiencies, then the plants giving off ratios might produce albino instead of yellow-green seedlings in crosses with *wd* and TB-9b.

Cytological samples were taken last summer from the two putative deficiencies found in the 1983-84 crop, but as yet we have not examined them. The 1984 deficiencies will be sampled cytologically next summer.

Donald S. Robertson

BARCELONA, SPAIN
Escola d'Enginyeria Tècnica Agrícola
VALENCIA, SPAIN
Escuela Superior de Ingenieros Agrónomos

## Influence of chromosome segments linked to pale green-11 and pale green-12 on quantitative traits

The loci pale green-11 and pale green-12 in the double recessive homozygote, *pg11 pg11 pg12 pg12*, determine a pale green plant phenotype. In dominant genotypes, which have a wild phenotype, we have studied, at the quantitative level, differences in the following traits: days from planting to silking and pollen shedding date, number of tassel branches, number and weight of ears, number of nodes below the ear, number of total nodes, ear height and plant height.

From an inbred line with an inbreeding coefficient of 0.996 with origin in ((UR x A545*o2*)UR$^2$)sel.[6], but still segregating at the loci pale green-11 and pale green-12, near isolines *Pg11 Pg11 Pg12 Pg12* (1111), *Pg11 Pg11 pg12 pg12* (1100), *pg11 pg11 Pg12 Pg12* (0011) and *pg11 pg11 pg12 pg12* (0000) were separated by means of a *wx* marker. By selfing and crossing among near isolines, the 8 possible wild genotypes were obtained in a common homozygous genetic background. The same near isolines were crossed with each one of the inbreds MC (0000), W64*o2* (0011), UR*o2* (1100) and B37*o2* (1111), and heterozygous $F_1$ progeny were obtained. From these materials and the traits mentioned above comparisons were made among: 1) different homozygous genotypes in a common background, and 2) heterozygous genotypes in different genetic backgrounds.

In the homozygous genetic background trial, the genotype *Pg11 pg11 Pg12 pg12* (1010) was represented twice, originating from crosses of different near isolines (Table 1). Between these two progenies, significant differences in three traits were found, which can be ascribed to the segregation of general heterozygosity (0.4%) or to genetic recombination in the differential chromosome segments linked to the markers. The recombination event should have occurred in the passage from the original plant to the

**Table 1. Comparison between two progenies with the same genotype and different origin. * The difference between progenies is significant (P<0.05).**

| Progeny genotype | Origin of progeny | Days to pollen shedding * | Days to silking * | No. of tassel branches | No. of ears | Weight of ears * | No. of nodes below the ear | No. of total nodes | Ear height | Plant height |
|---|---|---|---|---|---|---|---|---|---|---|
| 1010 | 0011 x 1100 | 71.86 | 72.13 | 15.46 | 1.969 | 164.5 | 8.247 | 13.18 | 47.46 | 100.6 |
| 1010 | 1111 x 0000 | 74.2 | 74.47 | 16.05 | 2.127 | 144.8 | 7.941 | 13.26 | 44.92 | 98.04 |

first generation of near isolines. Assuming that the differential segments linked to the markers arose mostly from UR, if linked to *Pg11* or *pg12*, and from A545*o2* if linked to *pg11* or *Pg12*, the recombinational hypothesis becomes the most likely. For example, in the trait weight of ears, the isolines show a very low value (Table 2), indicating inbreeding depression, ascribed to the homozygosity of the differential chromosome segments linked to the markers. If recombination is assumed in 0000 and 0011 isolines, the resulting genotypes could be those indicated in Table 3a. Recombination in the female parent (0011 → $001_0 1_0$) could only affect cell (5) of Table 3 because within an isoline there must be genetic variation for the recombination, and in each type of cross a different plant was used as female parent. Recombination in the male parent (0000 → $000_1 0_1$) must affect the whole column because within an isoline, only one plant was used as male parent. Accepting these two recombinations, the heterotic hypothesis is coherent in all progenies (See Table 3b). Thus, the pale green-12 locus would not be responsible for the differences among genotypes, which should be ascribed to differential loci linked to the marker.

**Table 2. Homozygous background trial. a) Origin and value of wild "pale green 11, 12" genotypes. b) Statistical significance (P<0.05). G= genotype.**

a)

| ♀ \ ♂ | 0000 | 0011 | 1100 | 1111 |
|---|---|---|---|---|
| 0000 | | | | |
| 0011 | 137.2<br>0010 (2) | 88.33<br>0011 (3) | 165.5<br>0110 (4) | 171.8<br>0111 (5) |
| 1100 | 165.5<br>1000 (6) | | 92.30<br>1100 (7) | |
| 1111 | 144.8<br>1010 (8) | | 150.3<br>1110 (9) | 113.7<br>1111 (10) |

b)

| G | x |
|---|---|
| 0011 (3) | 88.33 |
| 1100 (7) | 92.30 |
| 1111 (10) | 113.7 |
| 0010 (2) | 137.2 |
| 1010 (8) | 144.8 |
| 1110 (9) | 150.3 |
| 1010 (4) | 165.6 |
| 1000 (6) | 165.6 |
| 1011 (5) | 171.8 |

**Table 3. Transformation of Table 2 assuming that the isolines 0000 and 0011 are recombinants. $0_1$ means that the differential segment linked to *pg12* has recombinant genes proceeding from the differential segment linked to *Pg12*. Analogously in $1_0$.**

a)

| | 0000 ↓ $000_1 0_1$ | 0011 | 1100 | 1111 |
|---|---|---|---|---|
| 0000 | | | | |
| 0011 ↓ $001_0 1_0$ | 137.2<br>$0010_1$ (2) | 88.3<br>0011 (3) | 165.5<br>0110 (4) | 171.8<br>$011_0 1$ (5) |
| 1100 | 165.5<br>$1000_1$ (6) | | 92.3<br>1100 (7) | |
| 1111 | 144.8<br>$1010_1$ (8) | | 150.3<br>1110 (9) | 113.7<br>1111 (10) |

b)

| G | x |
|---|---|
| 0011 (3) | 88.33 |
| 1100 (7) | 92.30 |
| 1111 (10) | 113.7 |
| $0010_1$ (2) | 137.2 |
| $1010_1$ (8) | 144.8 |
| 1110 (9) | 150.3 |
| 1010 (4) | 165.6 |
| $1000_1$ (6) | 165.6 |
| $011_0 1$ (5) | 171.8 |

The results in the heterozygous genetic background trial for ear weight are presented in Table 4. In the W64A*o2* x isolines background the double heterozygous genotype 1010 is not significantly distinct from the other genotypes, even from the double homozygous 0011. In the MC x isolines genetic background the double heterozygous genotype is significantly inferior to the 1000 genotype. In the UR*o2* x isolines genetic background the double heterozygous genotype presents the highest value, easily explained by greater heterozygosity in the differential chromosome segments, due to the origin of isolines from UR x A545*o2*. We concluded that the distinct "pale green 11, 12" wild genotypes are not different in this character and thus, differences among genotypes within genetic backgrounds are due to loci linked to the markers.

**Table 4. Origin, mean and statistical significance ($P<0.05$) of progenies in the heterozygous background trial.**
**G = genotype.**

MC x Isolines genetic background

| Parents genotype | | Progeny | |
|---|---|---|---|
| MC | Isolines | G | $\bar{x}$ (grs.) |
| 0000 | 0011 | 0010 | 347.2 |
| " | 1111 | 1010 | 347.8 |
| " | 1100 | 1000 | 384.8 |

URo2 x Isolines genetic background

| Parents genotype | | Progeny | |
|---|---|---|---|
| URo2 | Isolines | G | $\bar{x}$ (grs.) |
| 1100 | 1100 | 1100 | 189.3 |
| " | 1111 | 1110 | 190.8 |
| " | 0000 | 1000 | 212.8 |
| " | 0011 | 1010 | 233.1 |

W64Ao2 x Isolines genetic background

| Parents genotype | | Progeny | |
|---|---|---|---|
| W64Ao2 | Isolines | G | $\bar{x}$ (grs.) |
| 0011 | 1111 | 1011 | 233 |
| " | 0000 | 0010 | 233.5 |
| " | 0011 | 0011 | 234.2 |
| " | 1100 | 1010 | 238.6 |

B37o2 x Isolines genetic background

| Parents genotype | | Progeny | |
|---|---|---|---|
| B37o2 | Isolines | G | $\bar{x}$ (grs.) |
| 1111 | 1111 | 1111 | 230.4 |
| " | 0011 | 1011 | 232.3 |
| " | 0000 | 1010 | 249.9 |
| " | 1100 | 1110 | 254.8 |

In the other traits, assuming different types of genetic action, analogous results have been obtained. Therefore, the wild genotypes "pale green 11, 12" would not be different in the studied traits.

Differences between genotypes of extreme value, within genetic background, are presented in Table 5. The influence of loci linked to the markers, in all the studied traits, was significant. According to the relative influence of such loci, three groups of traits can be separated: 1) number of tassel branches, number of ears, and weight of ears, 2) upper ear height and plant height, and 3) days to silking and pollen shedding, number of nodes below the ear and number of total nodes.

Ll. Bosch, F. Casañas and F. Nuez

**Table 5. Differences between genotypes of extreme value, expressed in % of highest.**
**+ Not statistically significant ($P<0.05$).**

| Trait | Genetic background | | | | |
|---|---|---|---|---|---|
| | MC x Isol. | W64Ao2 x Isol. | URo2 x Isol. | B37o2 x Isol. | Iso-lines |
| Days to pollen shedding | 2.48 | 3.14 | 4.61 | 3.90 | 3.96 |
| Days to silking | 3.57 | 3.68 | 5.10 | 3.59 | 4.67 |
| No. of tassel branches | 17.23 | 20 | 16.19 | 24.58 | 48.10 |
| No. of ears | $7.6^{+}$ | $11.5^{+}$ | 13.70 | 30.37 | 31.8 |
| Ears weight | $9.7^{+}$ | $2.1^{+}$ | 18.79 | 9.57 | 48.58 |
| No. of nodes below the ear | 7.06 | 6.18 | $2^{+}$ | 3.56 | 7.2 |
| No. of total nodes | 2.74 | 5.08 | 4.06 | $1^{+}$ | 8 |
| Ear height | 5.52 | 12.51 | 10.61 | $1^{+}$ | 25.8 |
| Plant height | 4.27 | 10.36 | 7.66 | 3.17 | 18 |

### *pg11 pg11 pg12 pg12* and smut

The near isoline *pg11 pg11 pg12 pg12* was more resistant to smut than its corresponding near isolines *Pg11 Pg11 Pg12 Pg12, Pg11 Pg11 pg12 pg12,* and *pg11 pg11 Pg12 Pg12,* in 1981 and 1984 (Table 1). In spite of this, we observed that some detasseled plants *pg11 pg11 pg12 pg12* showed smut in the damaged area. In 1982 and 1983 none of the near isolines were affected.

**Table 1. Number of plants with (+) and without (-) smut, in 1981 and 1984.**

| Genotype | 1981 | | 1984 | |
|---|---|---|---|---|
| | Smut | | Smut | |
| | + | - | + | - |
| pg11pg11 Pg12Pg12 | 7 | 36 | 3 | 11 |
| Pg11Pg11 pg12pg12 | 11 | 33 | 4 | 12 |
| Pg11Pg11 Pg12Pg12 | 12 | 33 | 3 | 10 |
| pg11pg11 pg12pg12 | 0 | 43 | 0 | 13 |

Ll. Bosch and F. Casañas

### Differences in days to flower between inbreds, and grain yield of single crosses among them

We have tried to establish if, in a large sample of public inbreds, there is any relationship between the distance among inbreds measured in the genetic system controlling days to pollen shedding, and grain yield of their hybrids. Important differences between days to flower would make genetic exchanges difficult and consequently greater heterosis for yield could be expected in such crosses.

A large number of crosses between short cycle x long cycle (S x L), and middle cycle x middle cycle (M x M) inbreds were made (Tables 1 and 2). In the

**Table 1. Results from crosses among short cycle x long cycle inbreds (S x L). The mean of days to pollen shedding of each genotype is shown in the upper part of the squares, and the mean of yield (grs./plant) in the lower one.**

| Long Cycle \ Short Cycle | | CG8 | F201 | F192 | F7 | F196 | F226 | F2 | F71 | F65 | CG14 | F227 | F212 | F131 | F225 | F215 | F1049 | F228 | E59 | E83 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 59,6 / 64,6 | 60 / 51,7 | 60,7 / 32 | 60,9 / 54,1 | 61,1 / 34,4 | 61,1 / 69,8 | 62,2 / 50,7 | 62,6 / 102 | 63,4 / 54,5 | 64,2 / 76,9 | 64,2 / 75,6 | 64,7 / 92,6 | 64,8 / 63,5 | 64,9 / 80,1 | 65 / 78,6 | 65 / 70,6 | 66,4 / 68,1 | 66,8 / 98,1 | 68,5 / 91,2 |
| Mo17 | 76 / 89 | | 65,1 / 311,1 | 62,1 / 234,2 | 64,3 / 249,8 | 65,1 / 259,7 | 64,1 / 250,7 | 65,2 / 283,7 | | 64,9 / 299,2 | | 66,8 / 376 | 66,2 / 292,5 | 65,7 / 319,3 | | | 67,1 / 247,9 | 66,4 / 761,8 | | 66,5 / 370 |
| Mo15 | 77,1 / 136 | | 66,4 / 287,2 | 62,5 / 263,5 | 63,6 / 273,6 | 63,5 / 266,6 | 64,4 / 311,6 | 65,7 / 279,1 | | 63,2 / 277,2 | | 67,7 / 294 | 66,4 / 317 | 66,7 / 367,6 | | | 67,5 / 283,9 | | | 67,2 / 405,1 |
| F570 | 77,2 / 147 | 64,8 / 244,6 | 64 / 278 | | 65,5 / 240,9 | | 64,3 / 247,2 | 65,1 / 266,7 | | | 67 / 293,2 | 66,1 / 254,4 | 66,4 / 265,7 | 67,2 / 285 | | | 67,2 / 270,7 | 67,6 / 294 | 68,6 / 277,2 | 66,8 / 346,7 |
| B37 | 77,8 / 117 | 63,9 / 270,1 | 66,5 / 296,7 | 63,2 / 252,3 | 65,9 / 258,2 | 66,3 / 263,2 | | 66,7 / 286,5 | 66,1 / 376,5 | 66,2 / 276,3 | | | | 66,5 / 286,5 | | | | | | 68,7 / 405,9 |
| B73 | 79,1 / 198,1 | 64,8 / 286,2 | 65 / 283,6 | | 65,9 / 264 | 65,7 / 315,2 | 65,3 / 337,3 | 65,9 / 303,6 | | 65,3 / 270,8 | | 66,1 / 290,2 | 67,1 / 322,4 | 67,2 / 303,1 | 65,2 / 281,4 | | 67,2 / 277,1 | | 69,4 / 269,8 | 66,8 / 383,7 |
| SHK89 | 79,6 / 153,1 | 64,6 / 359,4 | 65,1 / 286,7 | 62,1 / 221,5 | 64,4 / 219,1 | 63,9 / 313 | 65,3 / 325,2 | 64,6 / 262,2 | 66 / 395,7 | 63,8 / 279,4 | | 64,7 / 312,8 | 66 / 308,6 | 66,6 / 309 | | | 68,3 / 245,9 | | | |
| Z1 | 80 / 102,3 | | | 61,3 / 212,3 | 65,3 / 269,8 | 65 / 265 | 64,2 / 260,7 | 64,1 / 244 | | | | 65 / 363,2 | 66,4 / 346,6 | 66,4 / 334,6 | | | | | 67 / 269,8 | |
| B 65 | 80,1 / 128,1 | 64,2 / 248 | 63,8 / 272 | 63,4 / 252,8 | 65,8 / 242,3 | 63,7 / 261,5 | 65 / 260,8 | 66,6 / 262,2 | | 63,4 / 248,7 | | 66,5 / 294,7 | 66,9 / 289,5 | 66,3 / 262,1 | | | 65,8 / 261 | | | |
| B57 | 80,4 / 110 | | 66,4 / 320,7 | | 63 / 237,5 | | 64,3 / 252,3 | | | 64,7 / 274,2 | | 66,3 / 331,6 | 68 / 329 | 68 / 307,3 | | | 68,1 / 308,2 | 68,6 / 280,3 | | 68,8 / 298,5 |
| B84 | 81 / 120,6 | | 66,5 / 326,3 | 63,1 / 285,8 | 64,3 / 287,3 | 65,3 / 292,6 | 65 / 341,7 | 65,6 / 362,5 | | 63,8 / 299,7 | | 65,7 / 330 | 66,7 / 390,7 | 67,5 / 318,5 | 66,5 / 325,8 | | 67 / 321,1 | 69,6 / 380,6 | 69,1 / 316 | 67,1 / 493,7 |
| 27-Q | 81,5 / 163,6 | 65,1 / 281,5 | 67 / 313,3 | 65,9 / 278,4 | 68 / 240,5 | 66,5 / 285,1 | 66,9 / 242,2 | 68,2 / 294,8 | | 65,8 / 265,2 | 70,5 / 338,2 | 6',8 / 252,6 | 69 / 268,6 | 72,2 / 248,6 | | | 69,1 / 266,7 | | 69,5 / 281,4 | |
| BIND | 81,9 / 141,9 | | 67,5 / 316,1 | 65,8 / 287,2 | 66,5 / 355,5 | 67,5 / 396,7 | 67,2 / 372,6 | | 68,9 / 421,5 | 67,5 / 358,2 | | | 68,7 / 447,5 | 73 / 356,6 | | 69,1 / 415,5 | 68,2 / 335,5 | | 71,3 / 306,5 | |
| B59 | 82 / 198,4 | 64,5 / 346,2 | | 62,9 / 321,6 | 65,8 / 264 | 66 / 367,5 | 65,2 / 318,4 | 65,8 / 322,1 | 67 / 364,1 | 64,6 / 328,6 | | 66,5 / 342,9 | 65,2 / 358,5 | 67,1 / 329,4 | | 66,1 / 328,4 | 67,1 / 308,4 | | 69,1 / 309 | 68,6 / 500,7 |
| NC230 | 84,5 / 114 | | | | 67,4 / 372,2 | 69,5 / 417,1 | 67,2 / 382,1 | 68,7 / 384,2 | | 68,6 / 416,8 | | 68,8 / 403,5 | 69,1 / 489,2 | 69,7 / 405,4 | | | 69 / 382,8 | 72,5 / 415,6 | | 68,2 / 527 |
| B49 | 84,6 / 72,3 | | | 63,5 / 236,4 | | | | | | | | | | | | | | | 69,6 / 278,3 | |
| NC232 | 84,8 / 150,8 | | | 66 / 251,7 | 68,8 / 295,7 | | 67,3 / 353,2 | | | | | 69,5 / 389,2 | 70,3 / 421,3 | | | | 70,6 / 431 | 68,2 / 342,2 | | |
| C131A | 88 / 60,8 | | 68,2 / 309,2 | 64,8 / 217,3 | 66,8 / 248,7 | | 67,7 / 248,3 | | | | | | 67,8 / 300 | | | | | | 71,5 / 274,1 | |
| B52 | 90,5 / 105,5 | | | | 65,2 / 276,5 | | | 65,3 / 243 | | 64 / 221,7 | | | 67,8 / 293,4 | | | | 68,5 / 252,7 | | 70 / 264,2 | |

latest group, and in order to avoid crosses among related inbreds, pedigrees were considered and a pattern of crosses of flint x dent was established.

As measures of distance in the genetic system controlling days to flower we used: 1) the difference between days to flower of inbreds involved in each hybrid (d), and 2) the heterosis degree for days to pollen shedding of each hybrid (H). Distances among inbred lines in the plane determined by first and second axes of the Principal Components Analysis, "days to pollen shedding", "nodes to ear", "total nodes", "ear height", "plant height", and "grain yield", were also considered (D).

Inbreds involved in the experiment and the hybrids analyzed are shown in Tables 1 and 2, where yield and days to flower of each genotype are also indicated. Relationships among different measures of distance between inbred lines and hybrid yield were calculated considering the whole population of hybrids and grouping them into short intervals of days to flower, in order to diminish the influence that this factor has on yield.

The correlation values found are very low (Table 3), the highest being of negative sign. Therefore the distance among inbreds in the system controlling days to flower does not seem to be a good indicator of their specific hybrid yield. The use of a larger number of traits to discriminate inbreds does not increase, in our case, the capacity to predict the yield of the hybrid. High correlation between days to flower and the other traits considered in the Principal Components Analysis could explain why this last analysis does not show different results from the others mentioned above.

F. Casañas, Ll. Bosch and F. Nuez

**Table 2. Results from crosses among middle cycle x middle cycle inbreds (M x M). The mean of days to pollen shedding of each genotype is shown in the upper part of the squares, and the mean of yield (grs./plant) in the lower one.**

| Dent Middle Cycle \ Flint Middle Cycle | F64 | F431 | F208 | F481 | 00-91 | F576 | F120 | F502 | F522 | F485 | F564 | F483 | IUK-5 | F575 | 00-79 | IUK-22 | 3-RE | 0-10 | 4-RE | F534 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 66,2 / 42,2 | 67,7 / 68,4 | 68,6 / 81,4 | 69,5 / 134,9 | 70,1 / 111 | 70,1 / 130,6 | 70,2 / 79,3 | 70,7 / 106,1 | 71,1 / 120,7 | 71,9 / 120,1 | 72,3 / 146 | 73,2 / 116,9 | 73,8 / 83,7 | 74,8 / 86,1 | 75,8 / 91 | 76,5 / 177,9 | 77,7 / 81,4 | 78,3 / 74,6 | 78,8 / 105,8 | 79,5 / 87,2 |
| F113 69,5 / 81,3 | 63,3 / 277,5 | 63,9 / 307,5 | | | 66,3 / 375,4 | 65,2 / 430,9 | | 64,9 / 314,8 | 65,9 / 313,9 | | 65,6 / 425,4 | 66,5 / 350,1 | | 66,5 / 243,8 | | 67,2 / 392,7 | | 67,1 / 266,1 | | 65,7 / 286,1 |
| F230 69,8 / 57 | 65,1 / 226,2 | 65,5 / 328 | 65,1 / 196,2 | 66 / 302,7 | 66,5 / 308 | 66,9 / 374,2 | 65,2 / 293,3 | 66,9 / 250 | 66,5 / 302,8 | 68,1 / 350 | 66,8 / 355,3 | 67,1 / 366,1 | 68,7 / 318,5 | 69,5 / 278,3 | 68,2 / 341,9 | | | | | 66 / 256,8 |
| IUK-07 71,8 / 97,3 | 63 / 218,5 | 64,2 / 278,8 | 62,4 / 215,4 | | 67,5 / 292,1 | 67,1 / 317,5 | 63,5 / 283,6 | 66,9 / 290,3 | 67,5 / 333 | 68,8 / 315,2 | 65,8 / 266,4 | 67,3 / 347,2 | | 67,8 / 278,5 | | | | | | 67,2 / 217,7 |
| A619 72,4 / 85,5 | 62,6 / 248,6 | 66,5 / 278,7 | 62,5 / 240 | 66 / 286 | 67,1 / 284,8 | 67,2 / 322,9 | 63,1 / 268,7 | 65,6 / 279,1 | 66,8 / 333,2 | 67,2 / 338,3 | 66,7 / 282,5 | 68,1 / 317,8 | 68,1 / 353,6 | 68,6 / 263,6 | 67,2 / 313 | | | | | 67,9 / 228,4 |
| F546 72,5 / 157,7 | 65,3 / 341,2 | 67 / 398,2 | 64,7 / 300,3 | 67,1 / 469,4 | | 67,7 / 428 | | 68,5 / 304,6 | 68,7 / 283,4 | | 67,2 / 399,4 | | 68,3 / 437 | 68,6 / 349,2 | 68,7 / 389,4 | 71,4 / 355 | | 70,4 / 399,7 | | 68 / 344,4 |
| A639 72,6 / 149,5 | 63 / 255,6 | 65,8 / 311 | 65,1 / 331,7 | 67,2 / 388,1 | 66,4 / 363,1 | 68,4 / 430 | | 67,2 / 307,9 | 67,1 / 273,3 | 70,3 / 363,1 | 66,4 / 440,2 | 66,7 / 367,5 | 67,5 / 449,4 | 68,6 / 312,3 | 67,8 / 419,9 | | | | | 66,3 / 335,8 |
| A641 72,6 / 114,2 | 64,2 / 313,5 | 67,1 / 313,4 | 64,3 / 287,2 | 65,8 / 267,8 | 68,5 / 367,1 | 68,2 / 390,1 | | 67,3 / 293,4 | 68,8 / 266 | 68,1 / 243,5 | 67,5 / 387 | | | 68,5 / 371 | 68,3 / 437,4 | 71,3 / 271,8 | | 67,1 / 303,5 | | |
| W64A 73,1 / 79,3 | 65,8 / 289,8 | | 66,1 / 225,3 | | 69,7 / 270 | 69,1 / 372 | 66,4 / 339,1 | 68,6 / 269,3 | 67,3 / 304,1 | 68,1 / 309,7 | 67,4 / 345,1 | | | 72 / 255,3 | 69,8 / 338,2 | 70,1 / 332,2 | 68,1 / 292,8 | 68,4 / 269,4 | 72,2 / 302,5 | 67,1 / 271 |
| M14 74,2 / 140,4 | 65,5 / 342,9 | 67,5 / 342,2 | 64,6 / 230,2 | 68,5 / 315,4 | | 69,7 / 344,6 | 66,4 / 322,7 | | 69,7 / 263,8 | 69,7 / 377,5 | 69,2 / 345 | | 71 / 369 | 72,5 / 310,2 | 70,4 / 386,4 | 72,1 / 403,1 | 70,6 / 397,7 | | 73,2 / 357,6 | 70,8 / 253,3 |
| F542 74,2 / 169,3 | 66,4 / 390,4 | 67 / 396,1 | 65 / 309,5 | 67,1 / 418,9 | 67,1 / 418,3 | 67,5 / 461,5 | 65,9 / 397,4 | | 69,6 / 300,9 | 71,6 / 309,6 | 69,3 / 378 | | 68,6 / 459,7 | 71 / 306 | 70,4 / 502 | | 70,5 / 361 | 70,3 / 402,2 | | |
| A632 74,5 / 166,4 | 64,3 / 291,8 | 67,7 / 283,4 | 64,3 / 296,7 | 67,1 / 355 | | 68,1 / 413,8 | 63,7 / 335,3 | 68,6 / 310,1 | 73,2 / 362,8 | 69,3 / 299,1 | 66,1 / 379,6 | 69,4 / 368,1 | 68,2 / 386,1 | 68,9 / 295,5 | 67,1 / 329,3 | | | | | 67,9 / 262,4 |
| SD115 74,7 / 124,2 | 66,5 / 355,2 | | 65,4 / 274,9 | 66,5 / 442,1 | 69 / 412,7 | 68 / 410,5 | 65,1 / 320,5 | 68,8 / 400,7 | 69,2 / 430,2 | 67,9 / 418,2 | 67,8 / 416,8 | 68,4 / 376,4 | 70 / 377,8 | 68,7 / 310,1 | 68 / 406 | | | | | 68 / 282,3 |
| Oh43 74,8 / 99,5 | 64,8 / 246,9 | | 62,2 / 252,5 | 67,4 / 420,6 | 67,2 / 322,4 | 68,5 / 342 | 66,3 / 354,5 | 69,4 / 339,9 | 67 / 224,3 | | | 68,8 / 324,4 | 67,8 / 389,7 | 72,3 / 292,4 | 69,1 / 410,7 | 71,3 / 424,3 | 69,3 / 385,7 | | 72,2 / 368,3 | 68,5 / 215,7 |
| F497 75,4 / 139 | 67,2 / 322,4 | 66,1 / 311 | 64,8 / 286,4 | 68,2 / 386,4 | 69 / 392,9 | 69,2 / 375,7 | 66,5 / 379,8 | 70,7 / 312,1 | 69,6 / 301,1 | 70,6 / 384,8 | | | 70,9 / 406,2 | 73,1 / 275,2 | 70 / 383,6 | 70,2 / 359,3 | | | | 67,7 / 302,6 |
| F549 75,7 / 198,8 | 66 / 340,1 | | 64,1 / 307 | 68,2 / 434,4 | 68,7 / 378,7 | 68,5 / 453,5 | 65,3 / 392 | 69,5 / 254,8 | 71,5 / 280,1 | 72,1 / 278,4 | | 71,3 / 361,1 | | 70,1 / 329,5 | 71,7 / 459,8 | | | 70,5 / 358,3 | | 69,4 / 386,9 |
| W-9 76,8 / 122 | 68,2 / 282,5 | 69 / 351,8 | 65,4 / 279,1 | 68,7 / 375,3 | 69 / 358,7 | 68,4 / 356,9 | 66 / 366,1 | 69,8 / 294,7 | 69,4 / 336,2 | | 69,4 / 357,1 | | 71,8 / 298,5 | 72 / 283 | 73 / 326,9 | 72,5 / 301,6 | | | 73,1 / 307,3 | |
| C103 79,5 / 130 | 68,8 / 298,7 | | 68,3 / 266,8 | | 71,1 / 417,8 | 70,1 / 415,7 | | 71,1 / 352,8 | 71,6 / 414 | 72,3 / 481 | 68,7 / 410,3 | 71,5 / 443,8 | 72,5 / 333,1 | | | 72,2 / 477,1 | 72,1 / 459,2 | | 73,4 / 470 | 71,2 / 339,2 |
| B65 80 / 101,2 | 69,2 / 298,7 | | 66 / 267 | 68,8 / 354,2 | 70,6 / 335,2 | 68,6 / 374,6 | 67,5 / 394,2 | 70 / 314,7 | 70,5 / 357,7 | | | 71,1 / 419,2 | | 73,3 / 337,4 | 75,2 / 324,3 | 72,5 / 399 | 72 / 358,2 | 69,3 / 336,1 | | 68,9 / 306,8 |

**Table 3. Correlations among different measures of distance between inbreds and the corresponding yield of their hybrids (* P<0.01). (d= phenotypical distance in days to flower system; H= heterosis degree for days to flower; D= distance in the plane determined by the two first axes of the principal components analysis).**

| Days to pollen shedding | d | H | D | Number of hybrids included into each group and their origin | |
|---|---|---|---|---|---|
| 63-64 | -0.25 | -0.16 | 0 | 55 | S x L =37<br>M x M =18 |
| 65-66-67 | -0.27* | -0.06 | -0.21* | 208 | S x L =106<br>M x M =102 |
| 68-69 | 0.02 | 0.08 | 0.04 | 117 | S x L =36<br>M x M =81 |

BLOOMINGTON, INDIANA
Indiana University

### *Ac*—an insertional derivative of *Ds*?

*Ac* sequences act autonomously in that they can control their own transpositions. They work *in trans* and probably produce a transposase, although the *Ac* product has not yet been isolated nor identified. *Ds* is a transposable element which behaves as though it lacks transposase activity and moves only in the presence of an *Ac* element. Thus, an alteration in the transposase portion of the *Ac* element should be sufficient to convert *Ac* to *Ds*, and such conversions have been described by McClintock. Molecular analysis of *wx-m9Ds*, a *Ds* derivative of *Ac* in the waxy locus, by Fedoroff et al. (Cell 35:235, 1983), has shown that these *Ac* and *Ds* elements differ by the deletion of about 200 bases from the central region

of the *Ac* element. Restriction mapping of the *wx-m6* and *sh-m5933 Ds* elements have shown them to be similar to the *Ac* element, but lacking about 2 kb of DNA from the central region of *Ac* (Fedoroff et al., 1983; Courage-Tebbe et al. Cell 34:383, 1983). These observations seem to support the proposition that these *Ds* elements are derivatives of *Ac* that arose from internal deletions that abolish the transposase function. However, examination of the data presented in the Fedoroff et al. paper on the molecular analyses of *wx-m9Ac* and *wx-m9Ds* led me to suggest an alternative scheme for the relationship between *Ac* and *Ds*. I propose that *Ac* is an insertional derivative of *Ds*, resulting from an insertion of an element equivalent to the *Ac* central portion into a *Ds* element.

The maize genome contains multiple sequences that are homologous to the *wx-m6, sh-m5933 Ds* elements. At least 20 discrete bands are detected that hybridize to *Ds* probes. Similarly, Fedoroff et al. (1983) have shown that multiple copies of the central element of *Ac* are also present, even in material that does not exhibit *Ac* function. When they digested DNA from various maize stocks with restriction enzymes that yielded internal fragments of *Ac* and probed with an internal fragment, up to 8-10 copies of a fragment were detected which co-migrated with the fragment of a cloned *Ac* element. However, when the DNA was digested with restriction enzymes that yielded fragments containing a portion of the terminal *(Ds)* sequence in addition to a segment of the internal sequence that overlapped the internal probes, only a single copy of a fragment that co-migrated with the fragment from a similar digest of a cloned *Ac* element was detected in plants which carried a single *wx-m9Ac* allele. The *wx-m9Ds* plants did not yield this fragment, but instead gave a slightly smaller fragment that co-migrated with the fragment of a cloned *wx-m9Ds* allele.

Thus, the two components of *Ac* may exist as separable elements in multiple copies, and transposase functions in effecting transposition limited to the unique composite structure where the central element exists as an insertion in a *Ds* element.

The alternative schemes for the relationship between *Ac* and *Ds* elements lead to strikingly different predictions. If the multiple *Ds* elements are defective *Ac*'s they should have sequence homology to both the *Ds* probe and the central portion of *Ac*, except where the entire central portion had been deleted. Thus, in Southern Blot analyses, most bands that are lit up by the *Ds* probe should also show hybridization with the *Ac* internal probe. If, on the other hand, *Ac* is an insertional derivative of *Ds*, restriction fragments which contain the *Ds* elements should not also contain sequences homologous to the *Ac* central element. Fedoroff et al. digested DNA from a number of maize lines with BstE II, and probed with both an Ava I-Eco RI fragment of the central element and the Ava I fragment of the *pDs6* plasmid, which contains most of the *Ds* element. The results presented in Figure 7 of their paper support the proposition that *Ac* is an insertional derivative of *Ds*.

*Ac* elements may play a role in evolution by causing gross changes in genetic make-up via chromosomal rearrangements, and it is exciting to speculate that the two "stable" basic components of this potentially highly unstable system are present in all maize strains and conjoin under conditions of environmental stress.

Drew Schwartz

CAMPINAS, SÃO PAULO, BRASIL
Instituto Agronômico

**Linkages in chromosome four, horizontal resistance and kernel row number**

Continuing the work reported in MNL 56:32-33 regarding linkages of *Ga-S* with absence of seminal roots *(Asr1)*, we attempted to obtain homozygous families for both factors together with long mesocotyl. Only after five generations of selection, alternating selfings and open pollinations and testing hundreds of progenies, did we succeed. Whenever we had a pure *Asr Asr* progeny it was killed by a reddish mold, a *Penicillium* sp. probably *P. citrinum*. This happened although we treated the seeds in the paper dolls with Vitavax. The fungicide was enough to control all other fungi except this one. These results confirm our previous report in MNL 56:30-32 postulating a Zapalote Chico earworm resistant factor *(Zer1)* in the short arm of chromosome four, now shown to be very near the *Asr1* locus. It is still more of a confirmation if we accept that the mechanism of horizontal resistance to pests by glucosides (MNL 58:38-46) is always linked to the kernel row number *(Krn)*, as will be shown in the following. The pure healthy progeny *Ga-S Asr1* with long mesocotyl was crossed and backcrossed to IAC Maya latente. It was planted at 20 cm depth with a stick. From the backcross 464 seeds were planted at 15 cm of depth. 207 seedlings emerged. This is significantly different from 50% (Chi-square 5.39 with $P = 0.05$), and much more than 25%. It is a result which would be expected if the long mesocotyl character were due to the action of two dominant pairs of linked alleles. The value of p is $(-AB + Ab + aB + ab)/n$ with standard error the square root of $(3 + p)/n$: $p = 12.3 \pm 8.2$ between the two presumed long mesocotyl genes, *Lmc1* and *Lmc2*. 200 seeds harvested were planted again at 17 cm depth in four lines of 50 ears each with 16, 14, 12, and 10 kernel rows, separately. From these emerged, respectively, 37, 33, 38, and 36 in each class, giving no linkage between row number and long mesocotyl. The total of 144 seedlings gives

$p = 6.2 \pm 7.5$ between the two long mesocotyl pairs of alleles. From this planting 59 ears were harvested without selection. From each ear, 11 seeds were tested in a germinator, and those with 5 or more *Asr1* were considered maternal *Asr1*. The line planted with 16 rows gave 12 *Asr1* and 10 *asr1*. The remaining 14 to 10 rows gave 11 *Asr1* and 26 *asr1*. The Chi-square for interaction gives 3.57 approaching the $P = 0.05$ significance limit. Thus, it seems that there is a kernel row number factor linked to *Asr1* (for which we propose the *Krn4* symbol). This, together with the *Zer1* factor, reinforces the report in MNL 58:38-46 that the genes for horizontal resistance to pests by glucosides are linked to those responsible for kernel row number.

In the presence of *br2 br2* the long mesocotyl factors are not expressed. The mesocotyl remains short, in some progenies shorter than the coleoptile, which is not affected.

Luiz Torres de Miranda and
Luiz Eugênio Coelho de Miranda

### Linkage between *Ga-S* and *o1*

*o1* is reported (W.R. Singleton and D.F. Jones, unpublished) as being near *gl3*. We received from the MGSC a sample segregating *o1* and *gl3*. A double recessive *o1 gl3* plant was crossed to IAC Maya *br2* and selfed. The resulting opaque glossies were lethal. Opaque seeds were pollinated by our *Ga-S O1* adapted material and selfed. In one ear we counted 51 *o1* seeds in a total of 267 seeds. This gives $p = 38.2 \pm 4.8$ and would put *o1* around position 80 in chromosome four.

Luiz Torres de Miranda and
Luiz Eugênio Coelho de Miranda

CANBERRA CITY, AUSTRALIA
CSIRO, Division of Plant Industry

### A somaclonal mutant of maize alcohol dehydrogenase

In many plant species, including maize, stable variants have appeared after a cycle of tissue culture. However, few of these somaclonal variants have been obtained at defined loci which are amenable to molecular analysis that would shed some light on the mechanisms responsible for somaclonal variation. The maize alcohol dehydrogenase genes have been studied at a molecular level, and this report describes the isolation and preliminary characterisation of a tissue-culture-derived *Adh1* mutant.

Plants were regenerated from cultures that were initiated from immature embryos carrying both the Fast allele (from A188) and the Slow allele (from Berkeley Slow) of *Adh1*. Shoots were generated from cultures that were maintained on a modified MS-medium (C. E. Green and R. L. Phillips, Crop Sci. 15:417-421, 1975) containing between 1.0 mg/l and 2.0 mg/l of 2,4-D. Roots developed from the shoots upon transfer to medium without growth regulators. The roots had a good activity of both ADH1 and ADH2 without a specific induction treatment, and extracts from the roots were run on lithium borate starch gels which were stained for ADH activity (A. D. Hanson and A. H. D. Brown, Biochemical Genetics 22:495-515, 1984). A total of 385 individual regenerant ($SC_1$) plants deriving from 122 embryos have so far been tested in this way. The screening has revealed one ADH1 electrophoretic variant which was first detected by the absence of a band corresponding to the ADH1 Fast-Slow heterodimer. The regenerated plant was self-pollinated. The progeny segregate for the normal ADH1-Fast and an unexpected ADH1 isozyme which runs slightly slower than the homodimer of the *Adh1-U725* mutant obtained by R. J. Ferl, S. R. Dlouhy and D. Schwartz (MGG 169:7-12, 1979). This new variant is called *Adh1-Usv*. The heterodimer *(F/Usv)* has the same electrophoretic mobility as the ADH1-Slow homodimer. The *Usv* mutant has full ADH1 activity as judged by the enzyme reaction on starch gel, and the segregation among the seed progeny conforms to Mendelian expectations:

| Number of seeds tested | F/F | F/Usv | Usv/Usv |
|---|---|---|---|
| 71 | 17 | 39 | 15 |

$\chi^2_{(2)}$ (1:2:1) = 0.8

No further variants have been detected as yet; however, the screening programme is being continued to find additional *Adh* mutants, both for enzyme activity and for electrophoretic mobility.

Richard Brettell and Maria Jeppesen

CASTELAR, ARGENTINA
Departamento de Genética, INTA-Castelar

### A potential alternative method in maize breeding

The possible use of maize populations selected for presence of lethal genes was reported earlier by Salerno (MNL 58:52-53). Additional data involving this subject were later gathered from field evaluations.

One of the populations previously reported (Salerno, MNL 58:52-53) was subjected to yield evaluation and compared with the best two commercial hybrids from Argentina as a check. The results are presented in Table 1, showing grain yield (Kg/Ha), percent moisture content and percent stalk lodging. Assuming the grain yield of the best hybrid (hybrid 1) as 100%, the grain yield of the population that was selected for the presence of the lethal genes was

Table 1: Yield trial.

| | Kg/Ha | % Moisture | % Stalk lodging | % of best hybrid | % of base population |
|---|---|---|---|---|---|
| Population selected for presence of lethals | 11453 | 17.5 | 5.0 | 107 | 142 |
| Base population | 8080** | 18.7 | 13.0 | 75 | 100 |
| Population selected for absence of lethals | 9800** | 18.6 | 23.0 | 91 | 121 |
| Hybrid 1 | 10734 | 17.2 | 6.0 | 100 | |
| Hybrid 2 | 10570 | 17.5 | 7.0 | | |

**: Significant at the 0.05 level.

7% more than the best hybrid, while the population selected for the absence of the lethal genes yielded 9% less than the best hybrid. Considering the populations themselves and assuming the grain yield of the base population as 100%, the grain yield of the population selected for the presence of the lethal genes was 42% more than the base population. At the same time, the population selected for the absence of the lethal genes yielded 21% more than the base population. The population selected for the presence of the lethal genes also had good characteristics with respect to moisture content and stalk lodging as compared with the other two populations.

Results of this study confirm the usefulness of this alternative method in maize breeding.

Juan C. Salerno

### Lines of maize with low gametic transmission

A study of 154 $S_1$ lines of maize was made for the frequency of chlorophyll lethal genes. These lines showed a frequency of chlorophyll lethals between 3-25%. They were crossed with a line without chlorophyll lethals. The $F_1$ plants did not show any chlorophyll lethals. The frequency of chlorophyll lethals in $S_1$ and $F_2$ of these crosses is shown in Figure 1. Forty-seven percent of the studied lines showed a tendency to increase frequency of chlorophyll lethals, 2% of them had the same frequencies for chlorophyll lethals, 20% showed a tendency to decrease the frequency of chlorophyll lethals, and 31% of the lines showed no chlorophyll lethals in $F_2$. Study of these lines as females and males revealed low gametic transmission in both sexes.

This may indicate an effect on differential gametic viability, which would explain the deficit of the lethals in the heterozygous segregation (gametic drift). This may also suggest the presence of zygotic lethals, gametic lethals, and combinations thereof.

Juan C. Salerno

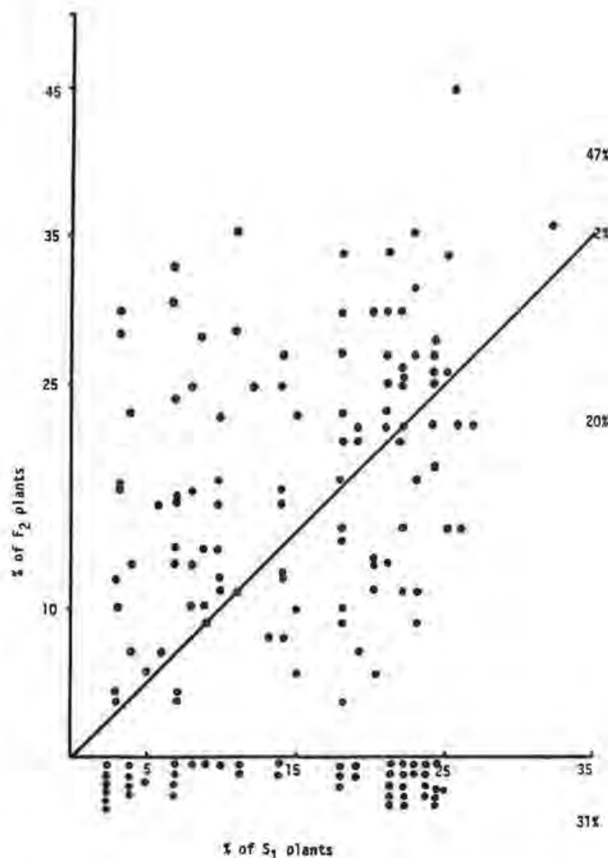



Figure 1: Frequency distributions of chlorophyll lethals, shown in percent of plants.

### The linkage of chlorophyll lethal genes with floury-2

An operation of a balanced lethal system in line 394 was reported previously by Salerno (MNL 58:52). This line was later crossed to a series of genetic markers to determine the chromosomal location of chlorophyll lethal genes involved in this line. The results from the $F_2$ and $F_3$ families of these crosses revealed that the chlorophyll lethal genes involved in this line were linked with the floury-2 gene on chromosome 4. The chromosomal location of this linkage group will be important for the future finding of heterotic regions in the maize genome.

Juan C. Salerno

## The linkage of chlorophyll lethal genes with iojap

An operation of a balanced lethal system in line 74 was reported previously by Salerno (MNL 58:52). This line was later crossed to a series of genetic markers to determine the chromosomal location of chlorophyll lethal genes involved in this line. The results from the $F_2$ and $F_3$ families of these crosses revealed that the chlorophyll lethal genes involved in this line were linked with the iojap gene on chromosome 7. The chromosomal location of this linkage group will be important for the future finding of heterotic regions in the maize genome.

Juan C. Salerno

## Manipulation of a balanced lethal system

A chromosomal disjoining and rejoining technique was developed to permit a balanced lethal system (Figure 1). Using this technique, line 394,

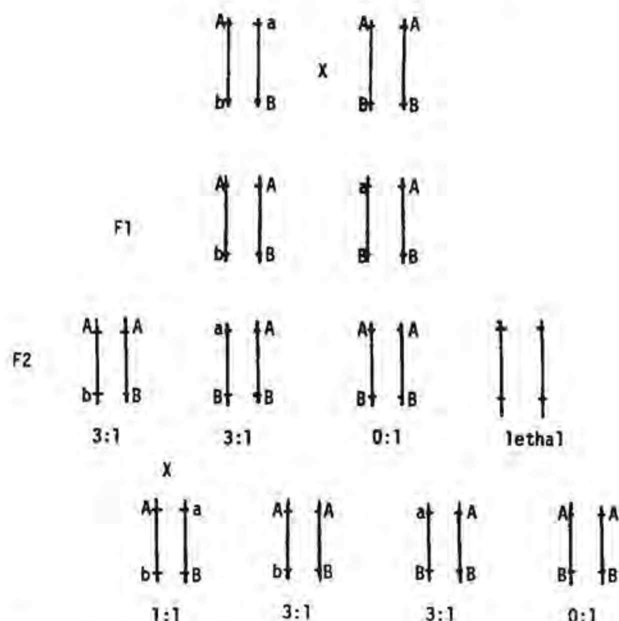



**Figure 1. Disjoining and rejoining of a balanced lethal system.**

which is regulated by a balanced lethal system (Salerno, MNL 58:52), was crossed with a normal line (without lethals). This resulted in disjoining of the lethal chromosome pair. Then, the $F_1$ plants were self-pollinated. The $F_2$ generation grown in the greenhouse segregated two 3:1 heterozygotes to one normal, to one lethal.

The two heterozygous progenies of the $F_2$ generation were planted in the field. At maturity, they were intercrossed. The progenies of this intercrossing, grown in the greenhouse, segregated 1:1 lethal to normal, in a balanced lethal system again. These progenies also segregated two 3:1 heterozygotes to one normal.

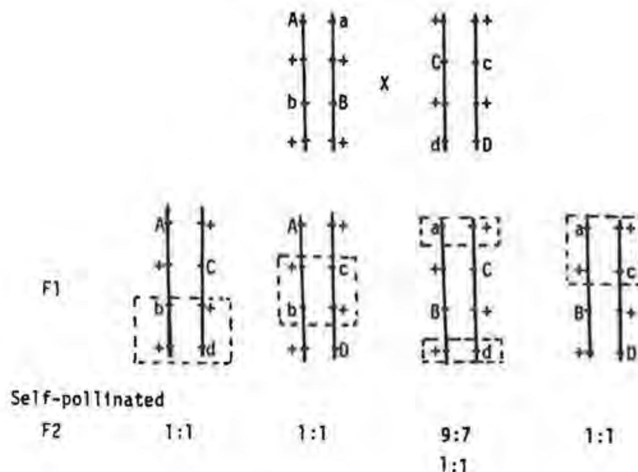



**Figure 2: Cross between balanced lethal systems.**

Figure 2 shows what happens when a cross is made between two balanced lethal systems. This technique would permit increasing the variability of these lines through the use of such a balanced lethal system.

Juan C. Salerno

### CHAPEL HILL, NORTH CAROLINA
### University of North Carolina

## Teosinte introgression, a probable source of mobile genetic elements

This report is concerned mainly with a new look at data previously reported in MNL or elsewhere. In two publications (1958, 1974), I suggested that a mutation system that had originated in my hybrids of corn and teosinte had features in common with those of McClintock (1950), Brink (1951), and others. An article by Nina Fedoroff in the June number of *Scientific American* has revived my long-dormant interest in mutation systems and prompted me to reexamine the results of my earlier experiments, especially to see if they could explain the source of the mobile genetic elements now known as transposable elements.

In the cytogenetic research on corn and its relatives in which Robert Reeves and I were engaged from 1927-1940, we found that annual teosinte, corn's closest relative, differs from corn, not by a small number of genes controlling specific morphological traits but by blocks of genes affecting many characteristics. Later in crosses with a nine-gene linkage tester, I found four of these blocks to be located on chromosomes 1, 3, 4, and 9. The tester had no marker-gene for chromosome 5 (MNL, 1947, p.20, 1974, p.41).

To study these blocks in a uniform genetic background, I crossed them with a well adapted Texas inbred 4R-3 and by repeated backcrossing

produced modified strains differing primarily in the blocks of genes introduced from annual teosinte. When I moved to Cambridge in 1940, I found these modified Texas strains to be poorly adapted to Massachusetts and by another cycle of backcrossing, introduced them into a second inbred, Minn. 158, an early-maturing strain. After four backcrosses to A158, the strains were selfed.

In several of the $S_1$ progenies, mutations occurred at rates higher than those produced by massive doses of x-rays. The mutations were of two kinds, stable and unstable. The latter are of special interest here, one in particular, a defective-seed mutant that inhibits the development of the endosperm in varying degrees from almost none to almost complete, the latter resulting in caryopses with little more than collapsed pericarps.

On a doubly-pollinated ear, homozygous for the mutant defective, the weights of the outcrossed seeds ranged from 140 to 250 mg and the frequency curve depicting these was the well-known normal frequency curve with a single conspicuous peak (Figure 1). In sharp contrast was the frequency distribution depicting the weights of the defective seeds. These varied from 25 to 200 mg and their polygon had 5 distinct peaks.

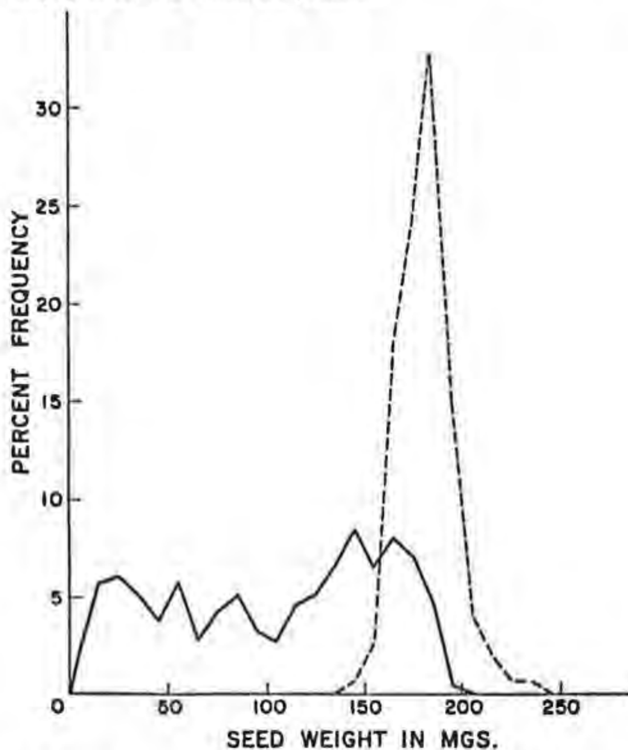



**Figure 1. Curves showing the frequency distributions of weights of selfed homozygous defective seeds (solid line) and normal outcrossed seeds (broken line) borne on the same ear. This ear is the ancestor of the forty populations shown in Fig. 2.**

In the next several generations, the peaks depicting the weights of the defective caryopses increased from 5 to 6, 7, 8, 9 and 10. Also, there were "repeats" in the patterns of the polygons. Some pairs were so similar that their peaks coincided almost perfectly.

Finally, in a population of forty $S_5$ ears, there were ten more or less distinct patterns each, with one or two exceptions having ten peaks *but no more* (Figure 2). Ten happens also to be corn's chromosome number; whether this is more than coincidence is a moot question. What is clear, however, is that the $S_2$ population with 5 distinct peaks has been the ancestral source of a family of populations with numerous patterns of frequency polygons each with numerous peaks. This may suggest that mobile elements of some sort were not only becoming more numerous but may have been moving from chromosome to chromosome, until they became, in Fedoroff's words, "ubiquitous".

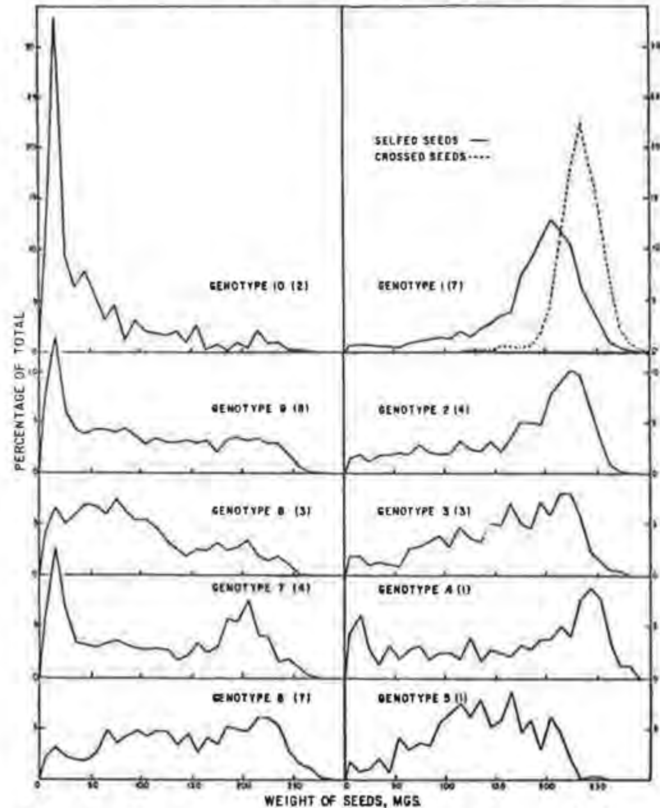



**Figure 2. Frequency polygons of forty $S_5$ populations derived from the population illustrated in Fig. 1. Note that there are ten more or less distinct patterns of polygons, most of which have ten peaks. What this means is not yet clear, but it suggests that something may be moving from locus to locus on the same chromosome and also from chromosome to chromosome.**

In both of my publications (1958, 1974) I called attention to similarities between this mutation system and that of McClintock and others, but offered no convincing explanation of their origin. In the more recent one (1974), I did suggest that chromosomes introduced into corn from teosinte, although appearing by linkage tests and conventional cytological studies to be homologous to those of maize, might not be in perfect register, so that crossing over at

various points could produce small deficiencies and duplications. The former might be lethal or inhibiting in their effects; the latter might create useful changes that were preserved under domestication. The possibility that unequal crossing over has been a factor in corn's evolution under domestication should not be ruled out unless studies at the molecular level do so.

Having reexamined my own data, I turned to the more recent results of McClintock and others. Of these the most interesting and perhaps most significant are those set forth in Table 1 in a 1965 paper (McClintock, 1965). This shows that the chromosomes identified as being involved in the *Ac*, *Spm*, and *Dt* mutation systems are 1, 3, 4, 5, and 9. This rings a bell! I am reminded that my 1946 linkage studies of blocks of genes derived from annual teosinte showed these to be located on four of these same chromosomes: 1, 3, 4, and 9. The tester, it may be recalled, had no marker gene on chromosome 5. The odds against even four of the same chromosomes being involved in the two mutation systems by chance alone are considerable. Calculated in one way they are significant; with other valid assumptions, substantially higher, and they are augmented by other circumstances.

All four of the blocks of genes that I had introduced into an adapted inbred strain of corn were associated with "tripsacoid" cobs. These are identified by stiff indurated outer glumes, often curved, that give the cobs the impression and feeling of a wood rasp. In fact it was this characteristic, more than any other, that enabled me to identify the blocks through two cycles of backcrossing.

In Plate I of a 1967 paper (McClintock, 1967) 13 cobs illustrating several types of variegation are depicted. All appear to be tripsacoid. Of almost equal interest is an ear illustrated in Plate 2D of the same paper showing an ear segregating for defective seeds (abortive caryopses).

In view of all of these additional similarities between the two mutation systems, McClintock's and mine, can there still be much doubt that they are in some way related? Fortunately the question can now be answered by studies at the molecular level. I am told that these are now in progress at several laboratories in this country and abroad. I await the results with more than casual interest!

In the meantime it may be useful for me to suggest to those who are engaged in research in this area some of the abundant materials that are available. Especially useful should be the modified inbred strains of A158 that have given rise to mutation systems. Walton Galinat may know what is available in this group. Also useful may be modified strains of A158 that have been introgressed by chromosome 4 of *Zea diploperennis*, which our experiments have shown to be probably ancestral to the annual teosintes. A small $S_1$ population of A158 in which the gene for perennialism has been introduced by repeated backcrossing segregated in 1983 for defective seeds, dwarfs and male sterility. This is an apparent mutation system tracing directly to the ancestral source rather than to the annual teosintes that represent an intermediate stage.

Fedoroff suggests that some of the variegation in races of maize of Central and South America may represent mutation systems and that it would be of interest to determine if these are similar or different from those already recognized. There is such a system in the Peruvian race Huayleño in which virtually all the pericarp and cob colors and patterns known anywhere in the world occur in one narrow valley in the Department of Ancash (Grobman *et al.*, 1961). Since variegation is not found in the prehistoric ears of corn in Peru, where they are abundant in archaeological sites, it may be assumed that there has been hybridization either with an introduced race from Mexico carrying teosinte germplasm or with a species of *Tripsacum* of which several occur in Peru. Other Peruvian races of special interest are Cuzco with variegated pericarp and Piscorunto for its stippled aleurone.

All of the authors of the ten publications on the races of maize of the countries of this hemisphere, beginning with Wellhausen's classic, *Races of Maize of Mexico*, may enjoy a degree of satisfaction if some of the races that they collected, described, and preserved for utilitarian purposes turn out to be also useful in solving the mystery of the mobile genetic elements popularly known as "jumping genes".

Paul C. Mangelsdorf

CHESTNUT HILL, MASSACHUSETTS
Boston College

### Meiosis and phenotypical expression of a dihaploid maize

A special feature of the meiotic divisions in the microsporocytes of this dihaploid was the configuration of bivalents at diakinesis. A salient characteristic of the bivalent was the intimate pairing of the homologues for each bivalent. In other words, the distance between any two chromosomes of a given pair was clearly shorter than that between any two homologues of the same division at diakinesis of an inbred maize. Even though this is just an estimate, lacking support by statistical data, the evidence was truly convincing. It is conceivable that this diploid plant was a homozygote originated by endomitosis during growth and differentiation. This plant was healthy and vigorous during the whole growth season. It was self-fertilized and the seed sets were close to 100 percent. However, its progeny plants were distinctly variable in height. The other characters such

as time of flowering, leaf chlorophyll-content, ear-height and number were impressively uniform. These are probably tangible evidence indicating the character of "mutable plant height" originated from mutation during culturing, since this character was not detected in the ancestral varieties. This strain of maize was subsequently named mph-1 (mutable plant height No. 1). Whether a transposable element is involved in this mutant is under investigation.

Y. C. Ting

**Ploidy stability of maize callus lines**

During the last few years a study on the ploidy stability of three maize callus lines was carried out. These callus lines were obtained through standard anther culture *in vitro* of different maize varieties and hybrids. It was found that if the majority of cells examined of a callus line were haploid in the beginning, the number of haploid cells became gradually increased during a long-term subculturing, such as callus line $N_1$. After one year of culturing, 90.2 percent of the cells were haploid. Five years later, 98 percent of them reached the same ploidy. On the contrary, for the same callus line, the number of diploid cells changed from 2.1 per cent in the first year to 0.4 per cent five years later. The number of aneuploid cells also decreased from 7.9 percent to 1.6 percent in the same period (Table 1).

For callus line $G_7$, it was found that the majority of cells were diploid in the first five months of growth. A little over three years later, the number of diploid cells in this line rose from 91 percent to 99.2 percent. However, the number of either haploid cells or aneuploids was consistently decreasing during that time. It was further observed after two and a half years of subculturing that about two percent of a total of 275 cells studied belonged to a higher ploidy level, either triploid or tetraploid. One year later cells with larger than diploid chromosome number were no longer present in the same callus line.

Samples of callus line $G_9$ were taken only twice for a term of 42 months of culturing. In the first 15 months, over 50 percent of the cells were identified as haploid with $2n=x=10$. Over two years later, a second sample of the same line was examined. In a total of 307 cells, 97.8 percent were definitely haploid. In contrast, both diploid and aneuploid cells were notably decreasing in number. The details of the change in ploidy level for both lines $G_7$ and $G_9$ are also clearly shown in Table 1.

In view of the above, it appears tenable to conclude that selection was for euploid cells, both haploid and diploid, of those maize callus lines during a long-term of subculturing. Among euploids, a haploid callus line (the majority of the cells are haploid in the beginning) tends to eliminate all the other ploidy cells and eventually becomes an absolute haploid clone. This is also true for a diploid callus line.

M. G. Gu and Y. C. Ting

**Table 1. Ploidy stability during long term subculture of maize callus lines derived from anther culture *in vitro*.**

| Call line | Length of subculture (month) | No. of cells studie | Variations in no. of ploidy () indicates % | | | |
|---|---|---|---|---|---|---|
| | | | Haploid | Diploid | Aneuploid | TridTetra |
| $N_1$ | 12 | 1539 | 1388 (90.2) | 32 (2.1) | 119 (7.7) | 0 |
| | 21 | 288 | 260 (90.3) | 7 (2.4) | 21 (7.3) | 0 |
| | 25 | 172 | 163 (94.8) | 5 (2.9) | 4 (2.3) | 0 |
| | 27 | 1125 | 1079 (95.9) | 15 (1.3) | 31 (2.8) | 0 |
| | 60 | 227 | 214 (94.3) | 3 (1.3) | 10 (4.4) | 0 |
| | 72 | 307 | 301 (98.0) | 1 (0.4) | 5 (1.6) | 0 |
| $G_7$ | 5 | 69 | 1 (1.8) | 63 (91.0) | 5 (7.2) | 0 |
| | 7 | 111 | 5 (4.5) | 98 (88.3) | 8 (7.2) | 0 |
| | 15 | 167 | 17 (10.2) | 132 (79.0) | 18 (10.8) | 0 |
| | 30 | 275 | 35 (12.7) | 220 (80.0) | 16 (5.8) | 4 (1.5) |
| | 42 | 258 | 2 (0.8) | 256 (99.2) | 0 | 0 |
| $G_9$ | 15 | 285 | 149 (52.3) | 126 (44.2) | 10 (3.5) | C |
| | 42 | 314 | 307 (97.8) | 7 (2.2) | 0 | 0 |

## Anther culture of tetraploid maize

In the last year over 1000 anthers of tetraploid maize were inoculated on Zheng-14 medium. The other procedures followed were the same as reported by Ting *et al.* (1981). It was observed that over two percent of the anthers responded positively by growing into callus or embryoid. Some of the embryoids regenerated into plantlets. Hence, it is possible to dissect tetraploid maize into diploids by anther culture *in vitro*. Whether the regenerants are mostly diploid is currently under investigation.

Sandha Raja and Y. C. Ting

COLLEGE STATION, TEXAS
Texas A&M University

## Rapid isolation and purification of immature zygotic embryos

The following procedure for isolating embryos exploits differences in both size and density between developing embryos and other tissues of the caryopsis. It requires about 30 minutes to recover immature (coleoptilar and older) embryos from 3 to 7 ears. Embryos prepared in this fashion have been used in our lab to study uptake of abscisic acid, and as a source of tissue for isolation of mRNA, enzymes, and pigments.

Husk leaves, tassels, and insect- or disease-damaged sections are cleaned from each ear of corn. These ears are rinsed in cold tap water and kept on crushed ice until shelled. With a serrated paring knife, each row of kernels is cut carefully at the base and lifted from the cob. The kernels may be collected in a beaker or released directly into the shallow pan which contains 800 ml of collection medium (20%, w/v, sucrose in 0.05M potassium phosphate buffer, pH 6.5 to 7.0). About 1500 kernels may be collected and processed at one time. While the shallow pan can accommodate twice as many kernels, the capacity of sieves (8" dia.) is only about 1500 kernels. Additional sieves could be used if additional capacity is required.

The kernels should be one layer thick in the pan. The shallow pan is 2 cm deep, with enough width to permit the rolling pin to easily rest along the edges and still leave a 2 to 3 mm clearance between the *bottom* of the pan and the nearest surface of the rolling pin. This clearance is adequate for embryos up to 2 mm in their largest dimension. For larger embryos, greater clearance can be achieved by adding suitable strips of wood to raise the edges of the pan appropriately.

The seeds are firmly crushed with a single pass of the rolling pin over the kernels. The entire solution is poured into the stacked sieves (stainless steel, USA standard testing #8 [2.36 mm], #18 [18 mm], #30 [0.60 mm], #80 [0.18 mm]) with largest pore size on top, decreasing in size to the smallest pore size at the bottom. The kernels are rapidly rinsed with cold tap water to wash through the embryos which were released from the kernels. The kernels are then returned to the shallow pan, mixed with 800 ml of fresh collection medium, and crushed again with the rolling pin. Several cycles are needed to open all kernels. It is important to remove the embryos from the broken kernels before crushing the kernels again as the rolling pin will damage the freed embryos. Rinsing the kernels in the stack sieves is best accomplished in a sink where excess solutions rapidly drain away.

After the last crushing, the kernels are thoroughly rinsed with cold tap water, with gentle stirring to ensure the passage of embryos into the lower sieves. Broken kernels retained on the largest pore sieve are discarded. Each subsequent sieve is rinsed until reaching the sieve which retains the majority of the embryos. With a gentle stream of water, trapped embryos and other tissues are moved to one side of the sieve and transferred to a large beaker (500 ml). The majority of the very light chaff is removed by filling the beaker with water and pouring off the chaff after the embryos sink.

The remaining tissue is primarily embryos and endosperm contaminated with a small amount of chaff and silks. This suspension is increased in sucrose concentration until density separation is achieved. Variations among inbred lines and age of embryo affect the exact density of embryos and endosperm tissue.

Once the sucrose density is reached for maximum separation of embryos and endosperm, the test tube is capped and placed horizontally for about 60 seconds to permit rapid separation of the tissues (Figure 1). Then the tube is very gently raised to a 45-degree position, cap end up. The tube is gently agitated to facilitate the migration of the tissues to their new positions. Finally, the tube is raised to a fully upright position. By starting in the horizontal position, there is minimal interference between the tissues as they move to their final positions. Embryos will float to the top of the tube and can be readily recovered by pouring that fraction into a petri dish.

For embryos to be used in physiological or ultrastructural studies which require intact embryos, a single flotation separation is adequate to remove the majority of nonembryonic tissues. This preparation then can be transferred to a petri dish and the intact embryos manually sorted from debris with a flat spatula or a pasteur pipette. Generally, about 70% of the embryos are intact with a typical preparation of stage one embryos (12 to 15 days after pollination). The efficiency of recovery of undamaged embryos increases as they develop beyond coleoptilar stages (3).

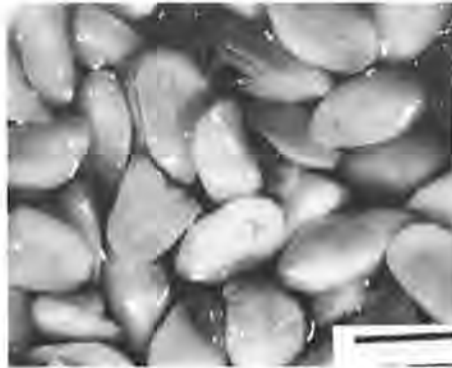


**Figure 1. Top: Embryos floating away from nonembryo tissues in sucrose solution. Bottom: Embryos isolated from commercial sweet corn; bar = 5 mm.**

With very young embryos 5 to 9 days after pollination, it is necessary to use a dissecting microscope to pick out the embryos with a pasteur pipette. Since the differences in density between the embryos and endosperm are not as great in these younger kernels, the primary enrichment for these embryos occurs at the sieving steps. With embryos from mature and nearly mature caryopses with desiccated endosperm, it is very difficult to break the pericarp open without damaging the embryo with the rolling pin. This procedure is most effective for immature embryos when endosperm tissues are still soft and pliable.

Franklin Fong and J. D. Smith

### Gravitropism in abscisic-acid-deficient seedlings

Many investigators have suggested that abscisic acid (ABA) is the causative agent for root gravitropism. Differential ABA concentrations in the upper and lower halves of the root presumably result in the more rapid growth of the upper half, which leads to the positive gravitropism characteristic of primary roots. Most experimental evidence for this mechanism was derived from applications of exogenous ABA, which may not correctly depict the functions of ABA at physiological concentrations.

We used two carotenoid-deficient mutants, *vp5* and *w3*, and the carotenogenesis inhibitor Fluridone for studies of primary root gravicurvature, as previous studies have shown ABA levels are reduced in carotenoid-deficient embryos. The mutants were in a Tx5855 background while Iochief is an unrelated sweet corn cultivar. Data related to ABA concentrations and gravicurvatures are presented in Tables 1 and 2, respectively.

**Table 1. Abscisic acid content (ng ABA $g^{-1}$ fresh wt.) ± s.d. of normal, Fluridone-treated and carotenoid-deficient mutant seedlings.**

| Cultivar | Leaves | Roots |
|---|---|---|
| Iochief | | |
| Untreated | 678 ± 74 | 398 ± 64 |
| Fluridone-treated | 0 | 0 |
| *w3* (Tx5855) | | |
| Normal segregates | 594 ± 122 | 379 ± 71 |
| Normal + Fluridone | 0 | 0 |
| *w3/w3* segregates | 0 | 0 |
| *vp5* (Tx5855) | | |
| Normal segregates | 402 ± 77 | 309 ± 32 |
| Normal + Fluridone | 0 | 0 |
| *vp5/vp5* segregates | 0 | 0 |

**Table 2. Gravicurvatures (degrees) ± s.d. of primary roots of normal, Fluridone-treated and carotenoid-deficient seedlings.**

| | Time h | |
|---|---|---|
| Cultivar | 3 | 6 |
| Iochief | | |
| Untreated | 66 ± 15 | 80 ± 16 |
| Fluridone-treated | 68 ± 12 | 84 ± 18 |
| *w3* (Tx5855) | | |
| Normal segregates | 41 ± 9 | 74 ± 13 |
| Normal + Fluridone | 38 ± 7 | 72 ± 11 |
| *w3/w3* segregates | 36 ± 10 | 66 ± 9 |
| *vp5* (Tx5855) | | |
| Normal segregates | 29 ± 7 | 54 ± 8 |
| Normal + Fluridone | 33 ± 6 | 60 ± 12 |
| *vp5/vp5* segregates | 27 ± 9 | 57 ± 10 |

The obvious conclusion to be drawn from these data is that ABA is not necessary for positive gravitropism of primary roots, since the Fluridone-treated and mutant seedlings were strongly graviresponsive in the absence of ABA.

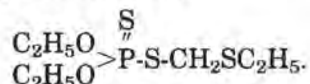
Our second conclusion is that ABA is a carotenoid derivative, as we were not able to detect ABA in roots or leaves of carotenoid-deficient seedlings with an analysis sensitivity of 2 ng ABA $g^{-1}$ fresh weight. This was not unexpected, as we had suspected for some time that the low levels of ABA found in carotenoid-deficient viviparous embryos were translocated from the maternal parent, and such ABA should be degraded in two-week-old seedlings.

J. D. Smith, Randy Moore* and Franklin Fong

*Baylor University, Waco, Texas

**Effects of a systemic insecticide and varietal resistance in reducing corn earworm damage**

This study was conducted to evaluate the effectiveness of a systemic insecticide, phorate, and varietal resistance in reducing earworm, *Heliothis zea* (Boddie), damage to maize. Phorate, also designated as Thimet®, contained O,O-diethyl S-(ethyl-thiomethyl) phosphorodithioate,




A split-plot design was employed with hybrids on whole plots and insecticide treatments on subplots. Significant differences among the four single cross hybrids were found for all the six characteristics studied, viz., ear and grain weights, larval penetration, damaged seed percentage, husk extension, and husk tightness. The effect of phorate was significant in reducing the larval penetration depth and in increasing the undamaged grain weight. There was a tendency for the differences among hybrids to be accentuated following the insecticide treatments. Husk tightness was an important morphological character in determining corn earworm resistance, but excess husk extension did not contribute additional resistance.

M. H. Yu* and J. D. Smith

*USDA-ARS, Agric. Res. Sta., Salinas, California

COLOGNE, WEST GERMANY
University of Cologne

**Studies of the shrunken gene on chromosome 9**

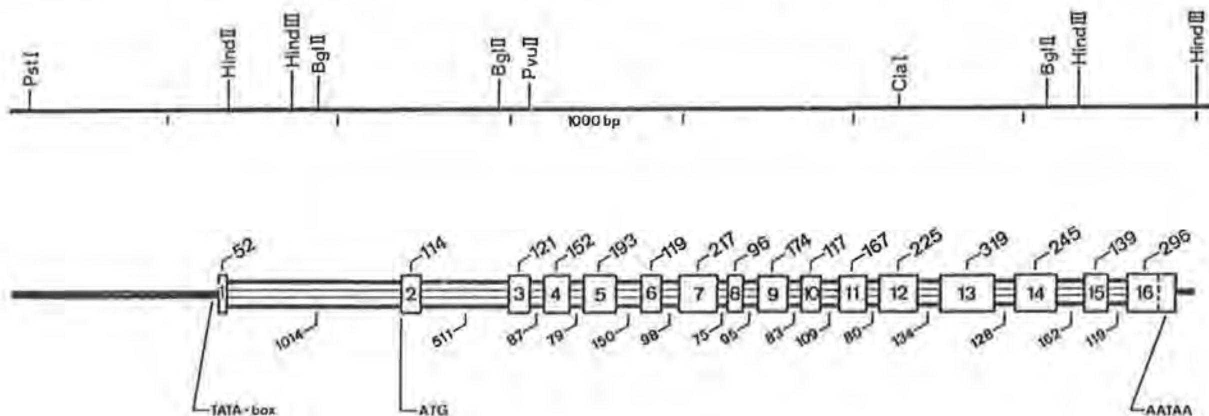
The exon structure shown in the figure is deduced from comparison of genomic sequence to a 2.5 kb long cDNA clone, which has been isolated from a cDNA library kindly provided by Zs. Schwarz-Sommer and A. Gierl (MPI, Köln-Vogelsang). This cDNA clone extends into exon 3. Exons 1 and 2 have been identified and positioned by S1-mapping, primer extension and RNA sequencing experiments. An open reading frame encoding for a polypeptide of 802 amino acids starts in exon 2 and stops in exon 16. The introns begin with GT and end with AG. 31 bp in front of the polyA tail a polyadenylation signal AATAAA is found. Upstream (-29) of the transcription start a sequence TATTTATT has homology to the TATA-box.

From the evolutionary point of view, it is interesting to note that we find 17 bp exchanges at 3rd codon positions between the cDNA clone pWW 11071 (line C) and our genomic clone (material from McClintock, *Ds* at standard position). None of these base substitutions causes an amino acid exchange. Calculations using base substitution rates derived from animal genes indicate that these two alleles would be more than 3 million years apart. Since present day maize is much younger, our observation could be explained either by an exceptionally high mutation rate in maize or by the assumption that the allelic diversity of maize ancestors has been introduced into *Zea mays*, as discussed by Galinat (1977, Corn and Corn Improvement). At the 3′-end we find two small duplications, which could be the consequence of the insertion and subsequent excision of transposable elements, as has been suggested by Schwarz-Sommer et al. (EMBO J., in press).

W. Werr, W.-B. Frommer, C. Maas, and P. Starlinger

**Regulation of the sucrose synthase genes**

The shrunken gene on chromosome 9 encodes the enzyme sucrose synthase (A), which is found at a high level in the endosperm of the developing maize kernel but not in the embryo. A second sucrose synthase (B) enzyme is found in both tissues (Chourey and Nelson, Genetics 14:1041, 1976).




The scheme below the restriction map shows the sequenced gene region. Exon and intron sizes are given in base pairs.

DNA fragments of the shrunken gene cross-hybridize to a slightly larger mRNA species in polyA⁺-RNA from *sh* mutants. This transcript was interpreted to be a mRNA of the sucrose synthase B gene (McCormick et al., Mol.Gen.Genet. 187:494, 1982). We have analyzed the mRNA levels in different tissues of maize plants including kernels, shoots, roots and leaves. The genotype of the plant material used for polyA⁺-RNA isolation was *Sh*, or homozygous for the *sh bz-m4* allele, in which the shrunken gene is deleted. Under the assumption that the slightly larger mRNA encodes the enzyme sucrose synthase B, we find identical low amounts in all tissues examined from *sh bz-m4* plants. In polyA⁺-RNA from *Sh* plants, we find hybridizing mRNA not only in endosperm but also in roots and shoots of germinating maize kernels at high levels. In roots and shoots we find 10 times less hybridizing polyA⁺-RNA than in endosperm 20 days after pollination.

Interestingly, the level of mRNA in roots and shoots increases ca. 20 times upon anaerobic stress for 19 hours. In leaves the mRNA for sucrose synthase A is 1/100 of the amount found in endosperm at 20 days. The level is comparable to the expression of the sucrose synthase B gene. We conclude that the gene is regulated during development of the maize plant at the transcriptional level and can respond to environmental signals. In all tissues examined we find a protein which can be precipitated by sucrose synthase antiserum and has similar migration properties in acrylamide gels as those of sucrose synthase.

B. Springer, W. Werr, and P. Starlinger

**The sequence of transposable element *Ac***

Two independent insertions of an *Ac* element into the waxy locus (McClintock, Carnegie Inst. Wash. Yearb. 62:486, 1963 and 63:592, 1964) have recently been cloned, *Ac9* and *Ac7* (Fedoroff et al., Cell 35:235, 1983; Behrens et al., MGG 194:346, 1984). The pattern of somatic reversions from the waxy phenotype to normal is different in the two alleles, since in homozygous plants reversion events occur earlier in endosperm development with the *wx-m7* than with the *wx-m9* allele (O. Nelson, pers. comm.). Furthermore, *wx-m7* is not a null mutation. The *wx-m7* allele carries *Ac* near the 5′ end of the gene, while the *Ac* element in the *wx-m9* allele is inserted in an exon 2.5 kb downstream from this site (Fedoroff et al., Cell 35:235, 1983; Behrens et al., MGG 194:346, 1984; A. Gierl and Zs. Schwarz-Sommer, pers. comm.). The DNA sequence of *Ac7* has been determined (Mueller-Neumann et al., MGG 198:19, 1984) and compared to that of *Ac9*, which has been sequenced by Pohlman et al. (Cell 37:635, 1984).

In the sequence, three open reading frames (ORF) are detected, two of which read to the left (ORF1 and ORF2), the other to the right (ORF3). Two of the ORFs qualify as potential protein-encoding genes. We have no information about splicing, and RNA and protein studies are needed.

All *Ds* elements sequenced so far (Döring et al., Nature 307:127, 1984; Pohlman et al., Cell 37:635, 1984; Sutton et al., Science 223:1265, 1984; Merckelbach, pers. comm.) contain an 11 bp perfect inverted repeat at their ends: TAGGGATGAAA. *Ac*, however, shows a replacement of the 5′ terminal T by a C residue at one end. This base substitution does not abolish transposition capability. Variability of the outermost nucleotides has not yet been observed in other transposons.

*Ac* contains other inverted and even more direct repeats, the latter mostly clustered in the terminal 1 kb sequences. The inverted sequences can partially be used to build up secondary structures. The base composition of *Ac* is inhomogeneous. ORF3 contains more CpG dinucleotides than expected on a random basis, which is interesting, because these sites are sensitive to methylation.

The DNA sequences of the *Ac* elements of both the *wx-m7* and the *wx-m9* alleles are identical. The phenotypic differences between the two alleles must therefore have another cause. It will be interesting to see whether the different insertion sites are responsible for the respective phenotypes, as has been suggested for enhancer elements by Peterson (In: Bukhari, ed., CSH Lab., NY, p. 429, 1977).

M. Müller-Neumann, J. Yoder, P. Starlinger

**The *Adh1-2F11* allele is characterized by the insertion of an "aberrant" *Ds* element**

*Adh1-2F11*, an unstable allele of the *Adh1* gene on chromosome 1, is due to a *Ds* insertion into the fourth exon of the gene. This mutation reverts only in the presence of *Ac*. Thus, the mutation is physiologically caused by a *Ds* element, and we have reported last year that indeed an insertion is found in the *Adh* gene.

This insertion has now been sequenced. It is 1319 bp long. 600 bp on the left and 300 bp on the right are about 95% homologous to *Ac* at its related *Ds* elements on chromosome 9. The remaining sequence of 400 bp in the center distinguishes this element from other *Ds* elements. This central sequence can be divided into two segments. One segment of 150 bp at one terminus is clearly related to *Ac*. In this region, several short DNA sequences of between 10 and 17 bp can be found that are also present in a limited region of 250 bp extension in *Ac*. The order of these fragments, however, is not colinear with *Ac* and they are interrupted with sequences not identifiable in *Ac*. This DNA segment may have arisen by a yet unknown sequence-shuffling mechanism. The remaining 250 bp of the central segment of *Ds-2F11* has no detectable homology to *Ac*. Neither do they

have a detectable similarity to *Ds1*, the "aberrant" *Ds* element described at the *Adh1* locus by Sachs et al. (1984).

The structure of *Ds-2F11* is thus intermediate between the *Ds* elements that are internal deletions of *Ac*, and *Ds1*, which shares with *Ac* only 13 and 19 bp respectively at either terminus and is otherwise unrelated to *Ac*.

A. Merckelbach, P. Starlinger

COLOGNE, WEST GERMANY
Max-Planck-Institut für Züchtungsforschung

**Molecular analysis of the *En/Spm* transposable element system**

*Comparison of the autonomous element and its receptor component:* Both the autonomous element *En1* (Peterson, 1953, Genetics 38:682-683) and its receptor homologue *Spm-I8* (McClintock, 1961, Carnegie Inst. Wash. Yearbook 60:469-476) were molecularly cloned from the *wx-844* (Pereira et al., EMBO Journal, in press) and *wx-m8* (Schwarz-Sommer et al., 1984, EMBO J. 3:1021-1028) alleles, respectively. The autonomous component *En1* is 8.4 kb in size and is inserted in an intron of the *wx* gene, while the receptor or Inhibitor element *Spm-I8* is 2.2 kb in size and is inserted in an exon of the *wx* gene. Heteroduplex analysis revealed that *Spm-I8* is a deletion derivative of the autonomous element. *Spm-I8* has retained 1kb and 1.2kb, respectively, from each end of the *En* element. Sequence analysis of their termini and of their flanking regions at their integration site revealed strong structural homology between the autonomous element *En1* with its receptor component *Spm-I8* (e.g. 3 bp target site duplication, 13 bp perfect terminal inverted repeat, extended stem and loop structure at the ends, etc.). This proves molecularly the genetic homology (Peterson, 1965, Am. Naturalist 99:391-398) of both the *En* and *Spm* system.

*Transcription products of En:* Two *En*-specific RNAs, 2.5 and 7 kb in size, were detected in Northern blot analysis of polyA$^+$ RNA from a line that contains an active *En* element. A cDNA copy of the smaller transcript containing 1.2 kb of its 3′-end was cloned and sequenced. Comparison to the *Spm-I8* DNA sequence revealed that the 3′-half of the cDNA is homologous to *Spm-I8* and that the region of homology is interrupted by intervening sequences. We intend to clone the cDNA into expression vectors to raise antibodies against the putative *En* protein.

*An assay system for the suppressor function of En:* In the absence of the autonomous element two chimeric transcripts, 2 kb and 3.2 kb in size, are observed in the *wx-m8* line which are probably initiated at the *wx* promoter and terminate in the *Spm-I8* insertion. In the presence of *En* these transcripts are suppressed, possibly by a trans-acting function of *En*, inhibiting transcriptional readthrough into *Spm-I8*. We intend to use this system to characterize the suppressor function of the *En/Spm* system (Gierl et al., manuscript submitted).

*Plant transposable elements generate the DNA sequence diversity needed in evolution:* Two germinal and sixteen somatic *En*-induced reversion events of the *Spm-I8* receptor element from the *wx-m8* allele were cloned and studied by sequence analysis (Schwarz-Sommer et al., manuscript submitted). Excision of *Spm-I8* from the *wx* gene results in various mutant DNA sequences. This leads to altered gene products, some of which are still capable of restoring the wild type phenotype. Thus the excision of a transposable element generates sequence alterations which can change the amino acid sequence of a protein and hence visitation of genes by transposable elements can be useful in the evolution of new gene functions.

All revertant sequences studied are compatible with a model of the transposition mechanism recently proposed (Saedler and Nevers, EMBO J., in press). Possible "footprint" sequences that may have arisen by the excision of transposable elements were observed when intron sequences of the wild type *(wx$^+$)* and the mutant *(wx-m8)* alleles of the *wx* gene were compared. These findings show that in evolution transposing elements are also involved in more subtle changes of the genetic material besides the well known gross chromosomal rearrangements such as duplications, deletions, inversions, translocations etc.

*Two states of the* a-m1 *allele differ in the size of the Inhibitor element inserted at the* A1 *gene:* Two states of Inhibitor at the *a-m1* allele (McClintock, 1965, Brookhaven Symp. Biol. 18:162-184; McClintock, 1968, Carnegie Inst. Wash. Yearbook 67:20-28) have been cloned and analysed by restriction mapping and Southern hybridization. Within the limits of resolution of the methods applied, the site of the *I*-homologous insertions is identical in both alleles. However, the sizes of the insertions differ considerably: state 5719A contains a 400 bp insert while that of state 6078 is 2.2 kb. This can be correlated with the residual gene activity present in the mutants in the absence of an autonomous *En/Spm*. Thus the basic color of state 5719A is medium dark, because the relatively small insert of 400 bp might allow some *A1* gene expression. The large 2.2 kb insert of state 6078 allows virtually no gene expression and, accordingly, this state is colorless in the absence of *Spm*.

Sequence analysis of these insertions will allow identification of sequences involved during *En/Spm*-directed excision events because the two states also differ in the frequency and timing of spotting. Molecular cloning and sequence analysis of derivatives of these two states as well as others differing in back-

ground color will shed light on structures necessarily present for the suppression of residual gene activity when the receptor elements are supplied with the S function of *En/Spm*.

Rita Berndtgen, Heinrich Cuypers, Alfons Gierl, Ralf Bernd Klösgen, Andy Pereira, Peter A. Peterson[1], Heinz Saedler and Zsuzsanna Schwarz-Sommer

[1]Dept. of Agronomy, Iowa State University, Ames, Iowa

## The use of *Spm(En)* and *Mu* transposable elements for molecular analysis of the *A1* and *C2* loci

The use of transposable elements as tags allows the isolation of genes whose products are unknown. Since *En/Spm* and *Mu-1*-induced *a1* mutants are available we took advantage of the *Mu-1* DNA and the recently cloned *En*-element (see accompanying report by Berndtgen et al.) to clone the *A1* gene.

Genomic libraries were prepared from both maize stocks, containing either the *En*-induced *a1-m(papu)* mutation (Peterson, P.A. Theor. Appl. Genet. 40:367-377, 1970) or Robertson's *a1-Mum2* mutant (Robertson, D.S. Mutat. Res. 51:21-28, 1978. These libraries were then screened with *En1* and *Mu1*-specific probes, respectively. However, since *En1* and *Mu1* are present in many copies in the maize genome, cross screening of the positives from each of the two libraries facilitated the identification of the clones containing the *a1* gene. Using the *a1* part of the two mutant clones the gene could be isolated from a wildtype line (the clone is available and has already been distributed).

A similar strategy to that described above was used in the isolation of the *C2* locus. One of the mutant lines used is the *Spm(En)*-induced *c2* mutant *c2-m1* (the autonomous element is present at the *C2* locus (McClintock, B. Carnegie Inst. Year Book 65:568-578, 1967). Since the screening of the *En*-induced *a1* mutant library resulted in the isolation of only one clone identical to the autonomous *En* element present in *wx-844*, we assumed that the same would be true for the *c2-m1* line. The analysis of this library, however, showed the presence of several copies of an element very similar to the autonomous *En* element in size and sequence. This 8kb-long element is identical to the autonomous *En* element over a 6kb internal region but differs partially in the terminal sequences.

The presence of this element, which is structurally very similar to an autonomous *En* element, causes problems in quickly screening the library, because the analysis of each positive clone has to be done very carefully. This element is also present in a *Spm*-induced *c2* mutant line *(c2-m2)* containing the receptor *(I)* part of the element at the locus. The screening for a clone containing the *C2* gene is still going on.

Catherine O'Reilly, Nancy S. Shepherd, Andy Pereira, Zsuzsanna Schwarz-Sommer, Ulla Niesbach, Ulrike Weydemann, Udo Wienand, Donald S. Robertson[1], Peter A. Peterson[2] and Heinz Saedler

[1]Dept. of Genetics, [2]Dept. of Agronomy, Iowa State University, Ames, Iowa

## The *Teo1* insert from *Zea mexicana* teosinte Guerrero is a composite of the *Cin2* and *Cin3* elements

The 3 kb *Teo1* DNA insert has been described recently in CGNL 57:159. Further analysis revealed that it is a composite structure consisting of a *Cin2* and a *Cin3* element. The *Cin2* element is 1.2 kb long, contains about 140 bp direct repeats at its ends and is flanked by a 3 bp duplication probably generated during the integration process, as usually observed with transposable elements. Into this element the *Cin3* element has integrated. This second element is 1.8 kb in length and has 634 bp inverted repeats flanked by a 9 bp duplication. This composite structure, previously called *Teo1*, was found in teosinte Guerrero by its homology to a unique 5 kb fragment, *LC1* of *Zea mays* Line C. DNA sequence comparison between *LC1* and the *Teo1*-containing clone *TG2* revealed that *LC1* still contains one of the direct repeats of *Cin2* flanked by the same 3 bp duplication found to border the composite *Teo1* structure. Hence we think that *LC1* or an equivalent clone isolated from teosinte is a recombinational derivative of *Teo1*.

Using *Cin2* and *Cin3* as specific probes in Southern hybridization experiments with genomic DNAs of various maize and teosinte lines, both elements were found to be repetitive in these lines. *Cin2* seems to be less repetitive than *Cin3*. Cloning experiments revealed that *Cin2* and *Cin3* usually occur as individual elements in the maize and teosinte genome. For example, a 6.2 kb DNA clone from the *Zea mays* line Hickory King contains only the *Cin2* element in an *LC1*-homologous segment.

The *Cin2* structure resembles copia-elements of Drosophila and also Ty elements of yeast. As in these systems solo copies of the LTR-like direct repeats are found. *Cin3* has a similar structure to that of Robertson's Mutator. Both are flanked by a 9bp duplication, but the *Mu-1* element has shorter inverted repeats (215 bp). The integration of one element into a different element has been described already in other organisms including Drosophila and some prokaryotes. Such composite structures have not been described in plants until now.

Jutta Blumberg vel Spalve, Nancy S. Shepherd and Heinz Saedler

COLUMBIA, MISSOURI
University of Missouri-Columbia

## Flavonoid 3′-hydroxylase and *Pr*

A microsomal preparation has been obtained from newly germinated maize seedlings that catalyzes hydroxylation at the 3′-position of the flavonoid structure. The gene *Pr*, which has long been associated with this activity, showed average specific activities of 100, 36 and 6.3% for the homozygous dominant, heterozygous and homozygous recessive stocks. The level of enzyme observed in seedlings recessive for *a1* or *c2* or *bz1* was noticeably in excess of that observed in seedlings having all the anthocyanin factors in the dominant state. The hydroxylase preparation was obtained by bicine buffer extraction of the seedlings followed by high speed centrifugation to yield a microsomal preparation. Seedlings were germinated on filter paper saturated with 1 mM $CaSO_4$ at 30°C in the dark (4 da) followed by exposure to light (1 da). Qualitative evidence has been obtained for the presence of the enzyme in immature aleurone tissue.

Russell L. Larson and James B. Bussard

## A computer-assisted model of aneuploidy in autotetraploid maize populations

A population of autotetraploid maize may consist of a bare majority of euploids and many different aneuploids. Aneuploidy is the result of the numerical non-disjunction (a 3 to 1 division) of the chromosomes of a quadrivalent. The frequency of numerical non-disjunction has been estimated (Doyle, 1973 Theor. Appl. Genet. 43:139-146) to be about 3% per quadrivalent.

A theoretical study of aneuploidy in autotetraploid populations even using the simplest assumptions requires a great amount of calculations. If we limit our consideration to genotypes that have four or less chromosomes in excess or in deficiency or a combination thereof, we must deal with 15 different gametic types and 30 zygotic types (these genotypes are shown along the left margins of the first two tables).

If we assume that sets of homologous chromosomes disjoin independently of each other and that they all have the same frequency of numerical nondisjunction, then the gametic output of a 4n plant may be calculated by expanding the trinomial, $(m + t + d)^n$, where m, t, and d are the frequencies of functioning gametes with 1, 3, or 2 chromosomes, respectively, from a set of four homologous chromosomes, and n is the number of chromosomes in the genome. This is shown in Table 1.

The gametic output of aneuploid plants may be calculated by separating sets with different chromosome numbers and then combining them (Table 2).

**Table 1. Formulae for estimating the gamete production of a euploid (4n) autotetraploid.**

| | Gamete type | General formulae* | if n=10 | if d = .970 and m and t = .015 |
|---|---|---|---|---|
| a | 2n | $d^n$ | $d^{10}$ | .737424 |
| b | 2n-1 | $nmd^{n-1}$ | $10md^9$ | .114035 |
| c | 2n+1 | $ntd^{n-1}$ | $10td^9$ | .114035 |
| d | 2n-1-1 | $nC2m^2d^{n-2}$ | $45m^2d^8$ | .007935 |
| e | 2n+1+1 | $nC2t^2d^{n-2}$ | $45t^2d^8$ | .007935 |
| f | 2n-1+1 | $nC1,1mtd^{n-2}$ | $90mtd^8$ | .015871 |
| g | 2n-1-1-1 | $nC3m^3d^{n-3}$ | $120m^3d^7$ | .000327 |
| h | 2n+1+1+1 | $nC3t^3d^{n-3}$ | $120t^3d^7$ | .000327 |
| i | 2n-1-1+1 | $nC2,1m^2td^{n-3}$ | $360m^2td^7$ | .000982 |
| j | 2n-1+1+1 | $nC2,1mt^2d^{n-3}$ | $360mt^2d^7$ | .000982 |
| k | 2n-1-1-1-1 | $nC4m^4d^{n-4}$ | $210m^4d^6$ | .000009 |
| l | 2n+1+1+1+1 | $nC4t^4d^{n-4}$ | $210t^4d^6$ | .000009 |
| m | 2n-1-1-1+1 | $nC3,1m^3td^{n-4}$ | $840m^3td^6$ | .000035 |
| n | 2n-1+1+1+1 | $nC3,1mt^3d^{n-4}$ | $840mt^3d^6$ | .000035 |
| o | 2n-1-1+1+1 | $nC2,2m^2t^2d^{n-4}$ | $1260m^2m^2d^6$ | .000053 |
| | residual | | | .000006 |

*nC2 = number of combinations 2 out of n.

**Table 2. Formulae for gamete production for 30 possible chromosome constitutions.**

| | | |
|---|---|---|
| A | 4n | $(m + t + d)^n$ |
| B | 4n-1 | $(m + t + d)^{n-1}(M + D_1)$ |
| C | 4n+1 | $(m + t + d)^{n-1}(T + D_2)$ |
| D | 4n-1-1 | $(m + t + d)^{n-2}(M + D_1)^2$ |
| E | 4n+1+1 | $(m + t + d)^{n-2}(T + D_2)^2$ |
| F | 4n-1+1 | $(m + t + d)^{n-2}(M + D_1)(T + D_2)$ |
| G | 4n-1-1-1 | $(m + t + d)^{n-3}(M + D_1)^3$ |
| H | 4n+1+1+1 | $(m + t + d)^{n-3}(T + D_2)^3$ |
| I | 4n-1-1+1 | $(m + t + d)^{n-3}(M + D_1)^2(T + D_2)$ |
| J | 4n-1+1+1 | $(m + t + d)^{n-3}(M + D_1)(T + D_2)^2$ |
| K | 4n-1-1-1-1 | $(m + t + d)^{n-4}(M + D_1)^4$ |
| L | 4n+1+1+1+1 | $(m + t + d)^{n-4}(T + D_2)^4$ |
| M | 4n-1-1-1+1 | $(m + t + d)^{n-4}(M + D_1)^3(T + D_2)$ |
| N | 4n-1+1+1+1 | $(m + t + d)^{n-4}(M + D_1)(T + D_2)^3$ |
| O | 4n-1-1+1+1 | $(m + t + d)^{n-4}(M + D_1)^2(T + D_2)^2$ |
| P | 4n-2 | $(m + t + d)^{n-1} + (-1)$ |
| Q | 4n+2 | $(m + t + d)^{n-1} + (+1)$ |
| R | 4n-2-1 | $(m + t + d)^{n-2}(M + D_1) + (-1)$ |
| S | 4n+2+1 | $(m + t + d)^{n-2}(T + D_2) + (+1)$ |
| T | 4n-2+1 | $(m + t + d)^{n-2}(T + D_2) + (-1)$ |
| U | 4n+2-1 | $(m + t + d)^{n-2}(M + D_1) + (+1)$ |
| V | 4n-2-1-1 | $(m + t + d)^{n-3}(M + D_1)^2 + (-1)$ |
| W | 4n+2+1+1 | $(m + t + d)^{n-3}(T + D_2)^2 + (+1)$ |
| X | 4n-2+1+1 | $(m + t + d)^{n-3}(T + D_2)^2 + (-1)$ |
| Y | 4n+2-1-1 | $(m + t + d)^{n-3}(M + D_1)^2 + (+1)$ |
| Z | 4n-2-1+1 | $(m + t + d)^{n-3}(M + D_1)(T + D_2) + (-1)$ |
| A' | 4n+2-1+1 | $(m + t + d)^{n-3}(M + D_1)(T + D_2) + (+1)$ |
| B' | 4n-2-2 | $(m + t + d)^{n-2} + (-1-1)$ |
| C' | 4n+2+2 | $(m + t + d)^{n-2} + (+1+1)$ |
| D' | 4n-2+2 | $(m + t + d)^{n-2} + (-1+1)$ |

Thus the genotype I (4n-1-1 + 1) has the formula $(m + t + d)^7(M + D_1)^2(T + D_2)$. The seven 4-chromosome sets are represented by $(m + t + d)^7$, the two 3-chromosome sets are expressed $(M + D_1)^2$, and the 5-chromosome set is $(T + D_2)$. A 3-chromosome set will give gametes with 1 and 2 chromosomes from the set with frequencies symbolized by M and $D_1$ respectively. Likewise a 5-chromosome set will give gametes with 3 (T) and 2 ($D_2$) chromosomes. In this case the expected frequency of 2n (a) gametes would be $d^7{D_1}^2D_2$, for example. Space does not permit an expansion of these formulae here. Genotypes that have 2-chromosome sets as in P (4n-2) would form a bivalent for that set and only one chromosome would go to all gametes. Genotypes with 6-chromosome sets Q (4n + 2) are assumed to always contribute 3 chromosomes from the set to all gametes. This is probably a poor assumption, but is necessary for the simplicity of the model.

**Table 3. Zygotes formed from combining gametes (a - o).**

| | a | b | c | d | e | f | g | h | i | j | k | l | m | n | o |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| a | A | B | C | D | E | F | G | H | I | J | K | L | M | N | O |
| b | | DP | AF | GR | CJ | BIT | KV | EN | DMZ | FOX | # | H# | G# | J# | I# |
| c | | | EQ | BI | HS | CJU | DM | LW | FOY | ENA' | G# | # | I# | H# | J# |
| d | | | | KVB' | AFO | DM<br>PZ | # | CJ# | GR# | BI<br>T# | # | EN# | KV# | FO<br>X# | DM<br>Z# |
| e | | | | | LWC' | EN<br>QA' | BI# | # | CJ<br>U# | HS# | DM# | # | FO<br>Y# | LW# | EN<br>A'# |
| f | | | | | | AFO<br>XYD' | GR# | HS# | BI<br>T# | CJ<br>U# | KV# | LW# | DM<br>Z# | EN<br>A'# | FOX<br>Y# |
| g | | | | | | | # | AF<br>O# | KV<br>B'# | DMP<br>Z# | # | CJ# | # | BI<br>T# | GR# |
| h | | | | | | | | # | ENQ<br>A'# | LW<br>C'# | BI# | # | CJ<br>U# | # | HS# |
| i | | | | | | | | | DMP<br>Z# | AFOX<br>YD'# | # | HS# | GR# | CJ<br>U# | BI<br>T# |
| j | | | | | | | | | | ENQ<br>A'# | GR# | # | BI<br>T# | HS# | CJ<br>U# |
| k | | | | | | | | | | | # | AF<br>O# | # | DMP<br>Z# | KV<br>B'# |
| l | | | | | | | | | | | | # | ENQ<br>A'# | # | LW<br>C'# |
| m | | | | | | | | | | | | | KV<br>B'# | AFOX<br>YD'# | DMP<br>Z# |
| n | | | | | | | | | | | | | | LW<br>C'# | ENQ<br>A'# |
| o | | | | | | | | | | | | | | | AFOX<br>YD'# |

# designates genotypes with more than four chromosomes plus or minus.

After computing the expected frequencies of these 15 kinds of gametes, they must be united in 120 different combinations (See Table 3). Depending on whether the excess and deficient chromosomes are homologous or not, different zygotic genotypes are possible in most cases. A union of b (2n-1) and c (2n+1) gametes will yield A (4n) and F (4n-1+1) zygotes with frequencies of 1/n and n-1/n, respectively. This situation is quite complex and the relative frequencies of different zygotes that result from the union of gametic types require elaborate formulas. Along with the zygotes A to D′, there are a great number of aneuploids with more than 4 plus or minus chromosomes, symbolized in Table 3 by #.

To run through one generation of random mating or self-fertilization of an autotetraploid population using a desk calculator required two days. Consequently, my son, Ted Doyle, devised computer programs that can do a generation in 20 seconds. The program has nine inputs, m, t, d, $D_1$, $D_2$, M, T, n, and g (the number of generations desired—essentially unlimited). The genome chromosome number may vary from 8 to 32. Under 8 chromosomes the aneuploidy component is too limited and over 32 chromosomes overloads the combination-figuring component of the program.

A number of insights into the behavior of aneuploidy in autotetraploid populations have been made. Some of these insights are obvious after they have been demonstrated in the model. For example, the greater the value of n the more aneuploids there will be in the population, but the relative frequency of genotypes P through D′ will be less. Random mating populations achieve an equilibrium state after which there is no change in the relative frequencies of the various genotypes. Self-fertilizing populations continue to become more and more aneuploid until they all become B′ (4n-2-2), C′ (4n+2+2), or D′ (4n-2+2). The approach to this state is very slow.

Copies of these programs will be sent on request.

G. G. Doyle

### Chloroplast developmental timing: nuclear perturbation

Normally assembly of the major chlorophyll (a/b) light harvesting complex (LHCII) is a late event of thylakoid differentiation, both for angiosperms and algae. Mutation at an unmapped locus on the 2L chromosome arm delays greening and at the same

time permits early assembly of LHCII (Polacco, Chang and Neuffer, 1985, Plant Physiol., in press). A visible phenotype is observed: virescence, with a developmentally conditional high yield of leaf chlorophyll fluorescence (dc-hcf). High chlorophyll fluorescence only occurs when virescent individuals are partially greened and not for fully greened leaves.

Four independently isolated mutant alleles have been found for this locus: *v*-350*, *v*-424*, *v*-576A*, *v*-588A* (see also MGNL 56:44, 1982). All four result in virescent individuals with the dc-hcf phenotype. Curiously, a second virescent phenotype, without the dc-hcf trait, also segregated in M3 progenies segregating *v*-576A* and *v*-588A* individuals that were dc-hcf. Over 25 virescent individuals have been examined for each of several *v*-350* and *v*-424* progenies and these were always dc-hcf. It was not clear whether both virescent phenotypes arise from mutation at the same locus. The original allelism tests were performed with material segregating both virescent phenotypes. Crosses performed in 1984 between +/*v* individuals of low-fluorescence progenies segregating only one virescent phenotype show that the virescent variants of *v*-576A* and *v*-588A* without the dc-hcf trait are allelic to *v*-424*, and in the case of *v*-588A*, to the dc-hcf variant of *v*-588A*. Ears segregating only dc-hcf virescent individuals are not yet available for *v*-576A*. The various combinations of virescent (not dc-hcf) with virescent (dc-hcf) yield varying degrees of fluorescence for the 4-10 individuals checked for each of several crosses. It should be noted that all of these mutations are in stocks that retain 12.5% of the original EMS-treated genome.

One explanation for the two virescent phenotypes is that *v*-576A* and *v*-588A* unmask loci that regulate the timing of LHCII assembly during thylakoid morphogenesis. Crosses have been made to test this hypothesis. Characterization of leaf chlorophyll fluorescence is being done for the early greening stages of siblings to dc-hcf *v*-576A* variants and non-dc-hcf, *v*-576A* variants.

Allelism tests of *v*-424* with *v4* and *w3* were made and are being analyzed. Mapping of *v*-424* relative to *Ch w3* is in progress.

M. Polacco

### Isolation of a mutable *hcf*-19* allele

Two nuclear loci have been identified that block photosystem II (PSII) assembly: *hcf3* (chromosome arm 1S) and *hcf19* (tentatively assigned to chromosome 3L; it is uncovered by a confirmed TB-3La stock but linkage to known markers remains to be established). The molecular basis of the mutant phenotype is unknown. A second phenotype is associated with mutation at the *hcf19* locus: low PSII activity with no apparent block in the insertion of PSII-polypeptides into thylakoids.

A line which had only segregated *hcf* individuals with the second *hcf19* phenotype described above (based on electrophoretic analysis of thylakoid polypeptides for separate individuals) generated some progenies that segregated *hcf* seedlings with the same *hcf19* phenotype as the parental material and others with green and yellow green sectors (see Table 1). The leaf chlorophyll fluorescence induction kinetics for yellow green sectors indicated a PSII lesion. While I have tentatively called the mutation mutable *hcf19*, allelism tests need to be made to confirm this assignment. The F2 progenies of crosses to mutable *wx* testers for *Spm* and *Ac* were generated in the summer of 1984 and are being analyzed.

Table 1. Segregation of *hcf19* and mutable *hcf19* in three $F_3$ progenies of a cross between +/*hcf19* and inbred Mo17. This material is 12.5% EMS-treated genome, the remainder being 12.5% N28, 50% Mo17 and 12.5% some other line.

| Total | *hcf* (no sectors) | *hcf-m* | Percent *hcf* + *hcf-m* |
|---|---|---|---|
| 72 | 3 | 13 | 22.2 |
| 24 | 5 | 1 | 25.0 |
| 17 | 0 | 5 | 29.4 |

In any given progeny segregating mutable *hcf19*, sectors were both large and small, and in some instances were clonal. The yellow green sectors were not noticeable on first expansion of leaves and became apparent as plants matured. These sectored plants could be grown to maturity in the field, all producing pollen and some producing a small ear.

The segregation of mutable and non-mutable *hcf19* in some progenies suggests a two element system is involved. If true, the suitability of using this particular mutable allele to clone *hcf19* is dubious. Defective mobile elements have a good chance of being homologous to multiple regions on the genome, as has been established for *Ds*. However, identification of the inserted element would indicate that the locus is susceptible to insertion of a particular transposon class. This knowledge would be useful for future direction to this site of a transposon amenable to cloning the *hcf19* locus.

M. Polacco

### Mapping *hcf* mutants: progress report

The F2 progenies have been generated (summer 1984) for crosses performed the previous summer to test linkage of all known *hcf* loci (seven) on the 1L chromosome arm with *f*, *an*, *gs*, and *bm2*. The loci used are listed in MGNL 58:65 (1984). Both *f* and *an* have unambiguous seedling phenotypes in the material I am using, and data for the linkage of *hcf* loci with *f* and *an* should be available for the next Newsletter. The markers *bm2* and *gs* are not expressed at an early seedling stage and to determine

linkage to these loci, outcrosses will be made to a standard inbred line using material segregating these markers and an *hcf* allele. The F2 progenies will be analyzed and data used to establish a gene order and preliminary assessment of linkage. Analogous crosses were performed in the summer of 1984 for material segregating *hcf19, ys3, lg2* and *et* and for material segregating *hcf18, bm, pr* and *ys*. The F2 progenies for this material will be available in the fall of 1985.

M. Polacco

**Position of loci on chromosome 3L with respect to B-A translocations**

1984 field tests of *+/pm1*, *+/ba1* and *+/na1*, crossed by several B-A translocations on the long arm of chromosome 3, yielded the following information:

| | *pm1* | *ba1* | *na1* |
|---|---|---|---|
| TB-3La | uncovered | (uncovered) | (uncovered) |
| TB-3Lf | uncovered | uncovered | uncovered |
| TB-3Lg | uncovered | uncovered | |
| TB-3Ld | not uncovered | (uncovered) | (uncovered) |
| TB-3Li | not uncovered | uncovered | uncovered |
| TB-3Lc | not uncovered | (uncovered) | (uncovered) |
| TB-3Lh | | | not uncovered |
| TB-3Lk | not uncovered | | |
| TB-3Ll | not uncovered | not uncovered | not uncovered |
| TB-3Lm | not uncovered | | |

Results of crossing *ba1* and *na1* by TB-3La, -3Lc, and -3Ld, reported in NL 49:130, are included parenthetically for completeness.

The translocations are listed in the table above in the approximate order of their breakpoints on 3L, but further testing is required to establish order of breakpoints and position with respect to marker genes.

J. B. Beckett

**TB-5Ld, a new B-A translocation on the long arm of chromosome 5**

A new B-A translocation on 5L, designated TB-5Ld, has been produced by crossing a *pr* stock with pollen X-rayed before the second pollen division (Lin, B.-Y., NL 46:193). The translocation uncovers (i.e., is proximal to) *bv1, ae1, pr1, lw2,* and *gl8*. The genes *bt1* and *td1* are not uncovered, and the position of *v3* with respect to the translocation is uncertain because the phenotype is poorly expressed.

Although further testing with *na2* and *v3* is needed, the breakpoints of TB-5La, -5Lb, and -5Ld appear to be nearly the same, as all uncover or fail to uncover the same genes. Because *bt1* and *bv1* are mapped only 5 units apart, the region may be particularly susceptible to breakage by X-rays or perhaps may be quite long with little crossing over.

J. B. Beckett

**Establishment of homozygous B-A translocation stocks**

Eleven B-A translocations have been obtained in homozygous form. Most were produced by self-pollinating F1's involving marker genes on the same chromosome as the translocation. These translocation stocks are as follows:

| | |
|---|---|
| TB-3Sb | TB-7Lb |
| TB-3La | TB-8Lc |
| TB-4Sa | TB-9Sb |
| TB-4Lf | TB-10Sc |
| TB-6Sa | TB-10L19 |
| TB-6Lb | |

All of these stocks have been crossed onto the vigorous F1 hybrid Mo17 *Ht* x B68 and seed is available for distribution. Please note, however, that the hypoploid progeny of these crosses will not carry the full B-A translocation and should not be used for uncovering mutant genes, etc.

In addition to the above stocks, the following homozygous translocation stocks have been obtained from other research workers (some are still in the process of being confirmed):

| | |
|---|---|
| TB-1Sb | W. C. Galinat |
| TB-1Sb-2L4464 | D. S. Robertson |
| TB-4Sa | W. C. Galinat |
| TB-4Sa | W. C. Galinat (2nd stock) |
| TB-7Lb | A. Ghidoni via R. H. Whalen |
| TB-9Sb | A. Ghidoni via R. H. Whalen |
| TB-10La | W. C. Galinat |
| TB-10La | J. Kermicle |

Most homozygous stocks are weak and shed little pollen, so they are unsatisfactory for many uses. However, hybrids of translocations in different backgrounds should be more vigorous. It is planned to increase all homozygous translocation stocks, intercross for vigor if possible, and pollinate inbred lines and F1's by the homozygous translocations. Whenever vigorous B-A stocks or hybrids can be produced, they will be pollinated by inbred lines to produce seed that will give vigorous plants that are certain to carry the desired translocation.

When sufficient seed of these homozygous stocks is available, they will be sent to the Maize Genetics Cooperation Stock Center for further increase and distribution. Homozygous stocks received from others will not be distributed unless the requester has obtained permission from the deriver.

We would appreciate receiving additional homozygous B-A translocation stocks from anyone who has them.

J. B. Beckett and Bryan Kindiger

**Blue-fluorescent plants are fragrant**

On warm, still, sunny days, in the vicinity of plants of *Bf Bf* constitution, a mild, pleasingly

aromatic fragrance can be recognized. Some persons detect it easily; some do not. I have been able to identify, under good conditions (bright sunlight, still air), individual plants homozygous for *Bf Bf* in segregating families, by the fragrance from a warm leaf surface in the sun, and am satisfied that the judgment of full-grown plants by a sensitive nose can be as reliable as ultraviolet fluorescence readings in the seedling stage. From the sandbench, a bouquet of *Bf Bf* seedlings displays a faint fragrance (again, if exposed to bright sunlight before pulling). For classification purposes, homozygous and heterozygous constitutions can be recognized as follows:

| | *+/+* | *+/Bf* | *Bf/Bf* |
|---|---|---|---|
| Seedlings | normal | normal | fluorescent |
| Plants (leaves) | normal | normal | fragrant |
| Anthers | normal | fluorescent | fluorescent |

The combination of fluorescence and fragrance could be used in a laboratory seedling exercise (e.g., by separation for fluorescence, then comparing the fragrance between the separated seedlings - promptly). The fragrance is like that of bottle-on-the-shelf anthranilic acid, derivatives of which accumulate and cause the fluorescence in anthers of *Bf/Bf* and *Bf/+* plants (H. J. Teas and E. G. Anderson, PNAS 37:645, 1951) due to a combination of changes in anthranilate synthetase activity levels, feedback response, and inhibition of one of the anthranilate-utilizing enzymes (M. Singh and J. M. Widholm, Biochem. Genet. 13:357, 1975). While sunlight might contribute to stimulation of accumulation, enhancement of volatilization may be more likely.

E. H. Coe

**Silk browning is related to cob color**

Presence vs. absence of the polyphenol:polyphenol oxidase browning reaction of cut silks was found by C. S. Levings and C. W. Stuber (Genetics 49:491, 1971) to involve a dominant vs. recessive factor pair, which they designated *Fv* (ortho-dihydroxy flavonols present; browning) vs. *fv* (ortho-dihydroxy flavonols absent; non-browning). In families segregating 1:1 for cob color due to *P-WR* vs. *P-WW* in 1984 (grown for study of segregation of the expression of brown vs. white tassel glumes—see MNL 57:33, 58:75), segregation was noted for browning vs. non-browning silks. The browning-silk plants each showed brown color in the tassel glumes, and the non-browning plants none; cob colors in the harvested ears confirm that all plants with browning silks were *P-WR* (red cob, brown tassel glumes) and that all plants with non-browning silks were *P-WW* (white cob, white tassel glumes). The red:white ratios were, in families from five separate sources of *P-WR* backcrossed to A619 *(P-WW)*, 6:10, 8:8, 11:5, 9:7 and 18:23. The inbred lines studied by Levings and Stuber and classified as *Fv Fv* (browning silks) were CI21, Hy2, L317, NC232, T61, T204 and WF9; all are *P-WR*, red-cob lines. Those classified as *fv fv* were Kys, NC34, NC45 and NC236; the first and fourth are *P-WW*, white-cob lines; the other two may or may not be white, information being unavailable (the help of C. Stuber in tracking down some of the cob colors for which I had no information is appreciated).

The association of *P-WR* with silk browning due to ortho-dihydroxy flavones is consistent with the involvement of *P-WR*, as proposed by E. D. Styles and O. Ceska (Phytochem. 14:413, 1975), in determining a key, early step in the flavone branch of the flavonoid pathway. All is not settled on the association, however, since it is clear that some white-cob lines have browning silks. Among 40 white-cob inbreds that were checked in 1984, 10 showed browning (yet had white tassel glumes, consistently with their cob color). Among these inbreds is Mo20W, for which a small backcross *(P-WR/P-WW* x *P-WW)* segregated red:white cobs (17:27), but all had browning silks. Thus, red cob appears to determine browning silks, while white cob determines non-browning silks only in most sources, not all.

Should browning in white-cob inbreds turn out to be related to the undesirable "smoky" colors in white field corn, browning of silks would be a simple and convenient means by which this tendency could be detected; in any event, if silks do not show browning when cut the indication at this time is that the cob will be white.

E. H. Coe

**Chromosome 9 factors**

The accompanying list summarizes the results of allelism tests and TB tests from several seasons, for a series of factors on chromosome 9; the observations are indicated as + (i.e., normal) if the test was negative, or as recessive if the test was positive.

| | Dt | yg2 | wd | c | sh | bz | 17 | TB-9Sb | lo2 | wl1 |
|---|---|---|---|---|---|---|---|---|---|---|
| ba*-s | | | | | | | | ba | | |
| l6 | | + | + | | | | | l | | |
| yg*-5588 | | + | + | | | | + | yg | | |
| w*-s9000 | | + | | | | | | | | |
| w*-6-10a | | | | | | | | + | | |
| w*-034-5 | | | | | | | | + | | |

| | TB-9Lc | pg12 | ar | v | ms2 | gl15 | TB-9La |
|---|---|---|---|---|---|---|---|
| w*-9000 | | + | + | | | | |
| v*-8587 | v | | | | | | v |
| w*-6-10a | + | | | | | | |
| w*-8889 | w | | | | | | w |
| yel*-034-16 | yel | | | | | | |
| wlv*-EM53 | wlv | + | + | + | | | wlv |
| wl*-EM41 | wl | | | | | | |
| v*-MS61 | v | | | | | | |
| w*-034-5 | + | | | | | | |
| w*-8950 | | | | + | | | w |
| rghd* | rghd | | | | | | |
| dek13 | dek | | | | | | |
| fdt* | fdt | | | | | | |
| cp*-1381 | cp | | | | | | |
| de*-1409 | de | | | | | | |

E. H. Coe

## The modification of viability of some defective kernel mutants

The defective kernel mutants we have been working with generally show embryo lethality, but under favorable genetic backgrounds some mutant embryos are viable. Forty-one defective kernel mutants, most of which initially had lethal embryos, but which showed a few good mutant embryos on a segregating ear, were chosen for this study. For each case fifty mutant kernels were removed from the ear and ten with the best looking embryos were selected. Kernels were sterilized and germinated in petri dishes for a germination test. Those that grew were transplanted into flats for seedling observation. At the 2-3 leaf stage, surviving seedlings were transplanted to the field for plant trait observation. Those that matured were selfed and the ears recorded for mutant segregation. At least one kernel germinated in each case. The results (Table 1) were as follows: one mutant, *cp*-1430*, was embryo lethal (class L), twenty-one mutants had clear seedling phenotypes but were seedling lethal (failed before maturity, class I), three mutants gave mature plants that were sterile (class S), eight mutants were viable and were proven by at least one homozygous mutant ear (class V), and the remaining eight mutants did not get a good test (class X). Among the twenty-one mutants, five had white seedlings, six had pale green seedlings, three had very pale green seedlings, three had small weak seedlings, three had tiny seedlings and one had virescent seedlings. In all these cases the mutant seedlings died early. The four sterile mutants were as follows: Mutant *cp*-935* had normal weak plant phenotype, few florets in the tassel, with shriveled anthers; mutant *de*-1153* had small weak plants with barren tassels; and mutant *fl*-1390B* had small plants with non-shedding anthers. Sixteen mutants gave normal mature plants which were selfed. Eight of the sixteen, *crp*-888A*, *smk*-890*, *cp*-936A*, *o*-948B*, *o*-1310A*, *smk*-1373A*, *o*-1388* and *smk*-1484*, had one or more homozygous mutant ears, but four of these mutants, *crp*-888A*, *o*-948B*, *o*-1388* and *smk*-1484* also had a high proportion of heterozygous ears. Mutant *cp*-936A* had one ear which showed an ear sector of all mutant kernels (homozygous) and the remaining part had a 1 to 1 segregation for normal and mutant

Table 1: Summary of mutant kernel germination, plant number and phenotype[1], ears harvested and segregation ratio of 41 defective kernel mutants.

| mutant | number seeds | number seedlings | seedling phenotype | mature plants | selfed ears | segregation ratio: all N | segregation ratio: 3N:1m | segregation ratio: all m | class[2] |
|---|---|---|---|---|---|---|---|---|---|
| dek5 | 10 | 10 | white | 0 | - | | | | I |
| crp*-888A | 10 | 8 | N | 8 | 8 | - | 5 | 3 | V |
| dnt*-889 | 10 | 10 | N | 9 | 7 | - | 7 | - | X |
| smk*-890 | 10 | 10 | N | 10 | 9 | - | 1 | 8 | V |
| o*-933 | 10 | 7 | weak | 0 | - | | | | I |
| cp*-935 | 10 | 10 | weak | 7 | - | | | | S |
| cp*-936A | 10 | 7 | pg | 6 | 4 | - | 1 | 3 | V |
| o*-948B | 10 | 10 | N | 8 | 4 | - | 2 | 2 | V |
| de*-1007 | 10 | 5 | weak | 0 | - | | | | I |
| de*-1057B | 10 | 7 | roll leaf | 1 | - | | | | I |
| de*-1104B | 10 | 10 | small pg | 0 | - | | | | I |
| o*-1119A | 10 | 9 | white | 0 | - | | | | I |
| de*-1153 | 10 | 10 | small | 9 | - | | | | S |
| dcr*-1156B | 10 | 8 | white | 0 | - | | | | I |
| smk*-1167B | 10 | 5 | N | 3 | 1 | - | 1 | - | X |
| smk*-1168A | 10 | 8 | virescent | 0 | - | | | | I |
| orp*-1186A | 13 | 12 | pg | 0 | - | | | | I |
| o*-1189A | 20 | 18 | N | 13 | 8 | 3 | 5 | - | X |
| cp*-1286A | 15 | 12 | N | 3 | 2 | 1 | 1 | | X |
| cp*-1294 | 10 | 1 | pg | 0 | - | | | | I |
| sh*-1295 | 10 | 9 | ppg | 0 | - | | | | I |
| o*-1310A | 15 | 14 | N | 3 | 2 | - | - | 2 | V |
| o*-1313 | 12 | 9 | ppg | 0 | - | | | | I |
| sh*-1324A | 10 | 9 | pg | 0 | - | | | | I |
| fl*-1333B | 10 | 5 | ppg | 1 | - | | | | I |
| sh*-1341 | 11 | 1 | tiny white | 0 | - | | | | I |
| smk*-1373A | 10 | 10 | N | 8 | 5 | - | - | 5 | V |
| o*-1383 | 10 | 7 | N | 5 | 2 | - | 2 | - | X |
| o*-1384A | 10 | 5 | pg | 0 | - | | | | I |
| o*-1388 | 10 | 10 | N | 8 | 5 | - | 3 | 2 | V |
| de*-1390A | 10 | 10 | tiny | 0 | - | | | | I |
| fl*-1390B | 10 | 6 | small | 4 | - | | | | S |
| cp*-1393A | 10 | 10 | N | 6 | 6 | - | 6 | - | X |
| de*-1396A | 10 | 9 | ppg | 0 | - | | | | I |
| smk*-1423 | 10 | 10 | tiny | 0 | - | | | | I |
| smk*-1424 | 10 | 10 | tiny | 0 | - | | | | I |
| fl*-1426 | 10 | 7 | N | 2 | 2 | - | 2 | - | X |
| cp*-1430 | 12 | 1 | N | 1 | 1 | - | 1 | - | L |
| smk*-1484 | 10 | 10 | N | 9 | 6 | 1 | 4 | 1 | V |
| de*-1520B | 15 | 12 | pg | 0 | - | | | | I |
| sh*-1530 | 10 | 2 | N | 1 | 1 | - | 1 | - | X |
| | | | | | Total | 73 | 5 | 42 | 26 |

[1] Symbols: N = normal, pg = pale green, ppg = pale pale green

[2] Class: L = embryo lethal, I = seedling lethal, V = viable, S = sterile and X = unknown

kernels, indicating a possible reverse mutation in early ear development. Eight of the sixteen mutants had no homozygotes among the selfs. The reasons for getting heterozygous and homozygous normal plants from mutant kernels appear to be several: (1) misclassification in the selection of mutant kernels; (2) heterofertilization which would be enriched by selecting the best kernels; (3) gene dosage, perhaps the mutant was semidominant; (4) mutant instability giving reversion to normal in the embryo; and (5) genetic modification where a combination of other genes gave a phenotype.

Mutant *crp*-888A*, for example, had eight selfed ears harvested, of which five ears segregated 3 normal to 1 mutant kernel and three ears had all mutant kernels. The parent ear from which the best mutant seeds were taken showed a wide range of mutant kernel expressions, kernels from almost normal to severely collapsed. Kernel separation showed 432 normal kernels vs. 108 combined mutant kernels (64 crumpled and 44 defective or collapsed) or a 3 normal to 1 mutant ratio. The best mutant kernels selected for the experiment were the better developed crumpled kernels. The homozygous ears from surviving mutant plants had fewer crumpled kernels and more collapsed. One selfed homozygous ear had 123 crumpled, 174 defective or collapsed kernels and a scattering of nearly normal kernels. The expression of variability could be caused by a combination of gene dosage of the mutant and genetic modification involving both dominant and recessive modifiers. From these tests, it is clear that genetic modification of mutant expression is common and very effective within certain limits (ex. lethality due to albinism cannot be overcome by modifiers). It is also clear that in certain genetic backgrounds an otherwise recessive mutant may be expressed in a heterozygous m/m/+ endosperm.

M-T Chang and M. G. Neuffer

**Location of four new dominant mutants**

Part of the study of a new set of EMS-induced mutations (MNL 57:30, 58:71) in 6418 M1 plants (about 5000 good ears) has been to describe dominants and locate them to chromosome. So far 55 have been assigned mutant numbers after proving heritable in outcrosses. Most are partial dominants. Many more await testing. Four have been located to chromosome using the waxy reciprocal translocation series (T *wx* linkage; Table 1).

*Hsf*-1595* (hairy-sheath-frayed) first expresses as long hairs on leaf 4 or 5, especially along the margin. Sheaths and margins of upper leaves are densely pubescent, the leaves are narrower, and the sheaths bulge somewhat, in the manner of pubescent, high-altitude Mexican races. Plants are smaller and later. A very unusual feature is "fraying" of the leaf

Table 1. T wx linkage data for dominant mutants (only the significant linkages). 1:1 ratios are expected if unlinked to translocation.

| T wx stock | Break point | # pl | Wx Mut | Wx Norm | wx Mut | wx Norm | % CO | Chi-square |
|---|---|---|---|---|---|---|---|---|
| Hsf*-1595 (Hsf1) | | | | | | | | |
| 5S-9c | .07 L.10 | 62 | 17 | 15 | 9 | 21 | 39 | 4.93 |
| 5L-9a | .69 S.17 | 140 | 59 | 15 | 6 | 60 | 15 | 70.34 |
| Hsf*-1603 | | | | | | | | |
| 5L-9a | .69 S.17 | 118 | 60 | 3 | 2 | 53 | 4 | 98.86 |
| Sdw*-1592 (Sdw1) | | | | | | | | |
| 4S-9g | .27 L.27 | 52 | 6 | 22 | 4 | 20 | 50 | 19.81 |
| 5S-9c | .07 L.10 | 78 | 14 | 35 | 7 | 22 | 54 | 16.76 |
| 6S-9a | .79 L.40 | 48 | 4 | 20 | 4 | 20 | 50 | 21.33 |
| 8L-9d | .09 S.16 | 37 | 11 | 5 | 3 | 18 | 22 | 12.96 |
| 8L-9(6673) | .35 S.31 | 79 | 31 | 2 | 4 | 42 | 8 | 56.88 |
| Les*-2005 | | | | | | | | |
| 1L-9(4995) | .19 S.20 | 38 | 19 | 0 | 0 | 19 | 0 | 38.00 |
| 1L-9(8389) | .74 L.13 | 28 | 13 | 1 | 2 | 12 | 11 | 17.43 |
| 4S-9g | .27 L.27 | 110 | 46 | 5 | 3 | 56 | 7 | 80.57 |
| 4L-9b | .90 L.29 | 27 | 10 | 0 | 0 | 17 | 0 | 27.00 |
| 6S-9a | .79 L.40 | 32 | 15 | 1 | 0 | 16 | 3 | 28.25 |
| 7L-9a | .63 S.07 | 24 | 12 | 1 | 1 | 10 | 8 | 16.67 |
| 8L-9(6673) | .35 S.31 | 31 | 15 | 0 | 0 | 16 | 0 | 31.00 |

d.f. = 3; significant at $P<0.05$ if over 7.82, at $P<0.01$ if over 11.35

margin on some of the hairy-sheath plants. Enations of the margin can be up to 3 cm long and have the morphology of the sheath in miniature—curled lengthwise, shiny inside the curl, and a "ligule" between enation and blade, plus a cluster of hairs at the tip. "Fraying" is subject to modifiers. Ears are small with especially short husks, silking deep in the sheath. It is probably located on the long arm of chromosome 5. Its phenotype is quite different from *Rgd2*, also on chromosome 5, so we propose designating it *Hsf1*.

*Hsf*-1603* (hairy-sheath-frayed) is very like *Hsf1* but the incidence of "fraying" can be higher, and an odd expression distinguishes it. Two "coleoptile tubes" are often found across from the coleoptile. They may be very narrow and threadlike, have a small blade, or grow into tillers often nearly as large as the main stem. It also is located on chromosome 5, probably the long arm, and may be allelic to *Hsf1*.

*Sdw*-1592* (semi-dwarf) plants are definitely not typical dwarfs. Height in the heterozygote is ⅔ of normal—internodes are shortened. Leaves are erect and short as are tassel branches, which can have barren tips. Ears are small. Wilting is frequent. It is probably on the long arm of chromosome 8; the data in Table 1 are skewed because intermediate forms, resulting from crowding, are not counted. Its proposed designation is *Sdw1;* its morphology is significantly different from *Clt*-985*.

*Les*-2005* (lesion) is late-expressing starting as small yellowish spots and changing to small necroses 1-2 mm across. The homozygote has many more lesions than the heterozygote. It is probably on chromosome 9, possibly between *wx1* and *bk2*.

Robert McK. Bird and M. G. Neuffer

## Computer listing and summary of new EMS-induced mutants and variants

The maize genetics group here has been rapidly computerizing its word-processing and data management using a Helix 6809 computer and many purchased and in-house programs. A large data base listing all previous mutants maintained by M. G. Neuffer and associates is on the system. Two data sets are being compiled to summarize three years of characterization of a new set of EMS-induced variants (see above article). One data set lists the more unusual kernel variants, their segregations and other information. Collapsed, empty, defective-crown and many other variants which are very common on M1 ears are listed only in the original data book. Kernel variants are numerous—over 5000 ears have been examined and 7 *su1* alleles have been found. Nine more variants like *su1* are being tested for allelism. There are over 100 viviparous variants (9 loci?), 28 like *ae* or *su2* (2-3 loci), and 53 like *bt* or *sh* (4-5 loci), all needing allelism tests. Thus it seems we have gotten about 1 mutant per locus per 500 M1 plants!

The other data set lists family, segregation and other information from the last three seasons for most of the plant, pigment, and inflorescence variants being studied. Subsets of this information, sorted by any column, are easy to obtain.

Robert McK. Bird

## Ancient tiny cobs and the evolution of *Zea*

If one hypothesizes that teosinte was domesticated to give us modern maize, it seems unexpected that very early cobs from many parts of the New World share less with teosinte than do later cobs. Many of the archaeobotanical specimens from Mexico, Belize, Costa Rica, Ecuador, Peru and Brazil studied over the past nine years have been extremely small with very reduced cupules and glumes very unlike those of teosinte (Fig. 1). The earliest available maize, from near Tehuacan, Mexico, have cupules 20-25 (34) mm long and lower glumes only 17-31 mm long. Cupule depths of 2-3000 year old maize from Costa Rica, Panama, Ecuador and Peru range only 4-12 mm. The oldest lowland maize, from Belize (1950-1000 B.C.), has similar ranges. Glumes of the earliest Peruvian maize are membranous and relaxed toward the ear base. No lower glumes of maize have the apical thickening (0.4-0.5 mm) of *Zea mexicana*, *Z. luxurians* and *Z. diploperennis*. No teosinte has the pith which is always found in maize cobs. The differences between teosinte and early maize are many, more than have generally been given.

Robert McK. Bird

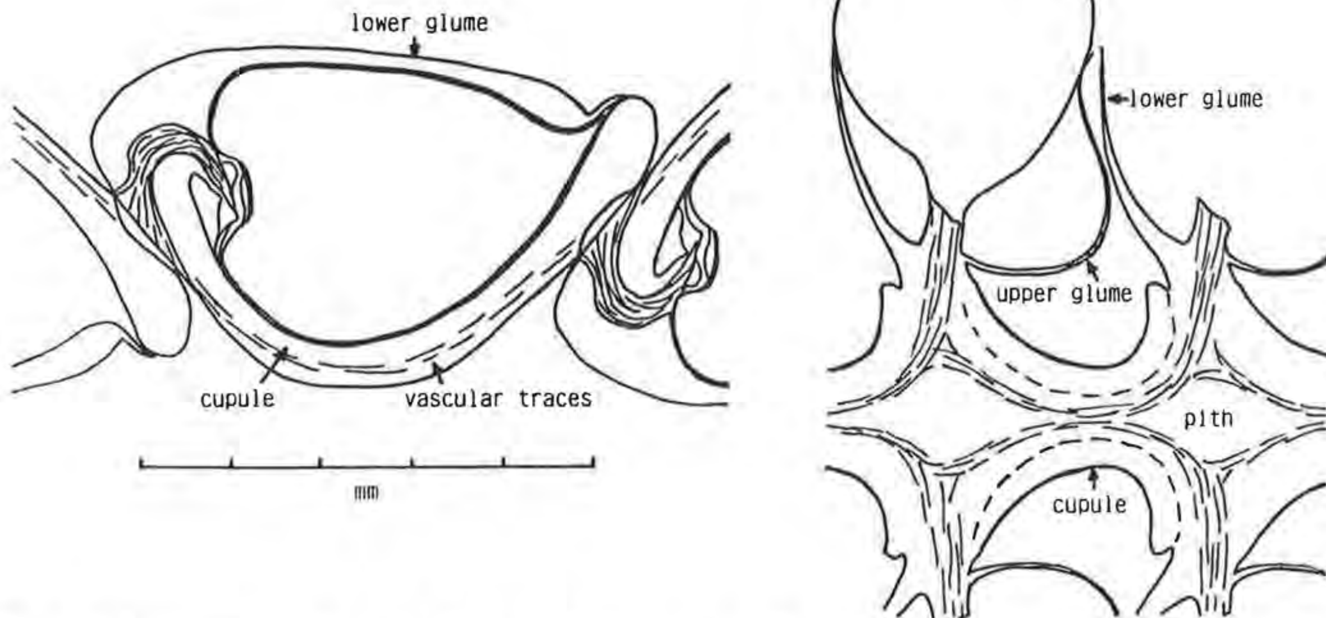



**Figure 1. Long sections of female alicoles of Balsas teosinte (*Zea mexicana* - El Salado population) and of early domesticated maize from Tehuacan, Mexico. The maize cob is a reconstruction using many measurements of Coxcatlan phase cobs, but the cupule dimensions fit many of the tiny cobs found elsewhere.**

## A Tale of Two Necrotics

I reported in the last Newsletter (MNL 58:84) that *nec3* and *nec*-493* were non-allelic. However, in the same issue, Shadley and Weber (MNL 58:160) reported that *nec*-493* is allelic to *nec*-409 (nec*-409* is the original lab designation of *nec3)*. In order to verify my allelism test, I planted 30 seeds from the ear of the F1 between two plants, each heterozygous for one of the necrotic mutants. The resulting 27 plants were selfed and 24 ears were recovered. Approximately 100 seeds from each ear were planted in the sandbench and the necrotic seedlings were scored. Out of the 24 ears, 5 segregated all normal seedlings, 12 segregated 3 normal:1 necrotic seedlings, and 7 segregated 1 normal:1 necrotic seedling. The 1:2:1 ratio of the above types of segregating ears and the 1:1 necrotic-seedling segregating ears verify that the original ear had a genotype of (+ *nec3*)/(*nec*-493* +) and that *nec3* and *nec*-493* are non-allelic. As reported, both mutants are linked to *a2* and *bt1* on 5S. The order and exact map distances are not known and may prove difficult to ascertain with two identical lethal mutants.

The story could end here; however, a second plant phenotype was seen in this study which is intriguing at best. Eleven of the above 24 ears also segregated for necrotic-banded seedlings. These seedlings have a single necrotic band or patch (approx. 1-2 cm wide) on the first three leaves. The band appears to have formed at the same time on all leaves in that it was located in the middle of the first leaf, close to the tip on the second and at the tip on the third. All ears segregating for necrotic bands also segregated for necrotic seedlings. The exact numbers are shown below.

| Segregation | # Ears |
|---|---|
| All normal | 5 |
| All normal + nec-banded | 0 |
| 3:1 nec | 5 |
| 3:1 nec + nec-banded | 7 |
| 1:1 nec | 3 |
| 1:1 nec + nec-banded | 4 |
| | 24 |

From previous families, I had an indication that the necrotic-banding was linked to *nec3* and these data support the idea that only one of the necrotic mutants is involved. I thought it might be possible that *nec3* was a semi-dominant or there was a dominant modifier affecting *nec3*, such that +/*nec3* resulted in the necrotic-banded phenotype. Unfortunately, the segregation data presented below do not really support either case.

Segregation of 3 normal : 1 necrotic ears

| Normal | Banded | Necrotic |
|---|---|---|
| 25 | 54 | 27 |
| 28 | 55 | 31 |
| 30 | 64 | 27 |
| 25 | 42 | 30 |
| 34 | 53 | 21 |
| 16 | 35 | 26 |
| 47 | 45 | 34 |

Segregation of 1 normal : 1 necrotic ears

| Normal | Banded | Necrotic |
|---|---|---|
| 15 | 50 | 75 |
| 7 | 50 | 41 |
| 12 | 32 | 48 |
| 18 | 44 | 56 |

All but the last ear of the 3:1 ears fits a 1:2:1 ratio, which is in line with *nec3* being a semi-dominant (but why are only $4/7$ 1:1 ears segregating necrotic-banded and the presence of normal seedlings in the 1:1 ears?) or a linked heterozygous dominant modifier of *nec3* or unlinked homozygous dominant modifier (but again why the segregation in the 1:1 ears?). The 1:1 ears fit a 1:3:4 ratio as expected for a double heterozygote of linked necrotic genes and an unlinked heterozygous dominant modifier. However, this is in conflict with the 3:1 ear data. I have tried several additional schemes, but none appear to satisfy all the data. My plan is simply to outcross a necrotic-banded plant in an attempt to study the linkage of the "modifier" and *nec3*. According to Coe (pers. comm.), 5S has often given unusual segregation ratios and this certainly fits that picture.

Dave Hoisington

## B breakpoints of the most proximal translocations

As the first step in an attempt to identify the breakpoints of B-A translocations, a set of the most proximal simple translocations was chosen for studying the break positions on the B chromosome. The set includes translocations between the B chromosome and eight A chromosomes which cover 13 chromosome arms. It does not include translocations on chromosome 2, since no simple translocation is available. Also not included are translocations involving chromosome 8 and the short arm of chromosome 5, because no favorable cytological material was available this summer. The most proximal translocations on the long arm of chromosome 10 (TB-10L18 and TB-10L19) are not in this set, since they have been published elsewhere (Lin, Genetics 92: 931-945, 1979).

The maize B chromosome at pachytene has a very short arm, a centromeric knob and a long arm. The long arm consists of, from the proximal to the distal end, a proximal euchromatic region, four segments of heterochromatic region and a tiny distal euchromatic tip as documented by Ward (Chromosoma 43:177-186, 1973). The length of the euchromatic region is about half that of the heterochromatic region. The first and the second segments of the heterochromatic region have an equal length, and are closely associated most of the time to become a single long segment. The third segment is the largest of the four segments, about one and one-half times the length of the first segment. The fourth segment is as long as the first one.

No translocation of this group has a break on either the short arm or the distal euchromatic tip of the long arm. There is a translocation (TB-10Sc) that has a break at the centromeric knob. This conclusion is based on two facts: (1) the B-A chromosome of this translocation has a centromeric knob smaller than that of the B or a regular B-A chromosome. (2) The same chromosome does not carry any heterochromatic region of the B chromosome, and the length of its euchromatic region is slightly shorter than that of the short arm of chromosome 10.

Four translocations (TB-1La, TB-4Sa, TB-7Lb and TB-9Lc) have a break in the proximal euchromatic region of the long arm. Their relative position in the region can not be determined at this point.

There are nine translocations with breaks in the heterochromatic region of the B long arm. TB-1Sb, TB-4Lb, TB-6Lc and TB-6Sa have breaks in the second segment, with the breakpoint of TB-6Sa close to the junction between the second and the third segment and the breakpoints of TB-1Sb, TB-4Lb and TB-6Lc in the middle of the segment. TB-3Lc, TB-5La and TB-9Sd have breaks in the third segment, with the breakpoint of TB-9Sd at the medial position of the segment and that of TB-3Lc proximal but close to it. TB-5La has a break position slightly proximal to the junction between the third and the fourth segment. The translocation having the most distal breakpoint of the set is TB-3Sb, at the proximal one-third position of the fourth segment.

The break positions of TB-4Sa and TB-6Sa in this study are consistent with those documented by Ward (1973).

Bor-yaw Lin

### Chromosome basis of small seed effect associated with *r-x1*

Some kernels (about 3 to 8%, depending upon the genetic background) on *r-x1* ears are smaller than the normal ones. They are variable in size (ranging from about one-tenth to three-fourths of the normal kernels), well filled and viable. Since it is well documented that seed size reduction in many cases results from an aneuploid chromosome constitution in the endosperm, it is of interest to see whether the same rule applies to these cases.

A selection of 25 small kernels, from one-third to nearly one-half the size of the normals, were germinated along with 26 large ones, and chromosome counts were obtained as follows:

| Seed type | Chromosome number | Frequency |
|---|---|---|
| large | 20 | 25 |
| | 21 | 0 |
| | 19 | 1 |
| | | 26 |
| small | 20 | 13 |
| | 21 | 4 |
| | 19 | 5 |
| | | 22 |

25 of the large kernels had 20 chromosomes, and one had 19. The rate of monosomy was 4.76%. The monosomy rate reported by Weber (1983) is 9.91%. Under the assumption that the aneuploid state of half of the 10 maize chromosomes can cause seed size reduction, the present rate is reasonably consistent with the previous one. No trisome was found in this group.

The chromosome constitution is different in the small kernel group. 4 out of 22 small kernels (18%) were trisomic and 5 others (22%) were monosomic. The frequency of aneuploidy for this group is 41%, which is nearly ten times higher than that of the large kernel group. Although the chromosome constitution of the associated endosperms can not be determined for the time being, it is at least very probable that they are also aneuploid. Results of this study suggest that seed size reduction in these cases is similar to those reported by Beckett (Can. J. Genet. Cytol. 25:346-353, 1983) and Lin (Genetics 100:475-486, 1982), where an aneuploid chromosome constitution in the endosperm results in a reduction of seed size.

Bor-yaw Lin

### Some information about nondisjunction incurred by the *r-x1* deletion

Data of the preceding article provide some instructive information about the time of the occurrence of chromosome nondisjunction under the following assumptions (cf. Shadley et al., MGNL 58:158): a) seed size reduction is due to an aneuploid chromosome constitution in the endosperm, b) only a single nondisjunction event occurs at either the micropylar or the chalazal pole during the embryo sac development, c) after the first post-meiotic division, the two nuclei move to the opposite pole, d) each of the four nuclei at either pole has an equal chance to become

polar nuclei before cellularization, e) any of the four micropylar nuclei can randomly develop into the egg and f) all of the nondisjunction events take place at the same cell division.

Nondisjunction does not occur at the first division. If it did, nuclei at one pole would have a chromosome constitution complementary to those of the other pole, i.e. nuclei at one pole being hypoploid and those at the other pole being hyperploid. The endosperm would be euploid, since the hyperploid polar nucleus compensates the hypoploid one. No seed size reduction would be expected. The consistent occurrence of small seeds contradicts this hypothesis.

Nondisjunction may take place at the second division. If a chromosome fails to disjoin at the micropylar pole during this division, half of the kernels would carry a hyperploid embryo and the other half, a hypoploid embryo. Among the former, $2/3$ of them would be in contact with a hypoploid endosperm, and $1/3$ of them with a hyperploid endosperm. Among the latter, $2/3$ of the embryos would be associated with a hyperploid endosperm and $1/3$ of them with a hypoploid endosperm. On the other hand, nondisjunction at the chalazal pole would give kernels carrying only an euploid embryo. Half of these would have a hyperploid endosperm, and the other half a hypoploid endosperm. Altogether, for the small kernels with an aneuploid endosperm, the rate of euploid, hyperploid and hypoploid embryos is $2/4$, $1/4$ and $1/4$ respectively. The present data (13:4:5) fit this expectation (2:1:1) very well ($P>0.5$).

The agreement of the present data with the supposition of nondisjunction at the second cell division can only be accepted with reservation. This is due to the fact that the assumption of an aneuploid endosperm as the basis of seed size reduction may not be valid. As mentioned in the previous article, the size of the small kernels selected for chromosome counting is half the size of the normal kernels or smaller. To this writer's knowledge, reduction in seed size caused by a hyperploid endosperm has never been reported to reach 50%. Therefore, it is more realistic to assume that the endosperm of the small kernels is hypoploid. Under this assumption, the expected ratio of euploid, hyperploid and hypoploid embryos of the smaller kernels would be 3:2:1 ($1/4$:$2/12$:$1/12$), which is statistically consistent with the data ($P>0.25$).

Nondisjunction does not occur at the last division for the following reasons: If it occurs at the micropylar pole, $2/4$ of embryos would be euploid, $1/4$ of embryos hyperploid and $1/4$ of embryos hypoploid. $2/3$ of the euploid embryos would be associated with an aneuploid endosperm and $1/3$ of them with an euploid one. On the other hand, $2/3$ of the hyperploid and hypoploid embryos would be associated with an euploid endosperm and the remaining $1/3$ with an aneuploid endosperm. When nondisjunction takes place at the chalazal pole, all embryos produced are euploid, but half of the endosperms are euploid and the other half, aneuploid. Taking all into consideration, the frequency of aneuploid endosperms for euploid, hyperploid and hypoploid embryos is $10/24$, $1/24$ and $1/24$ respectively. The ratio is 10:1:1, which is greatly different from that of the observed data.

Taking into consideration that the endosperm of the small kernel is hypoploid, nondisjunction is more unlikely to occur at the third division. Under this assumption, a hypoploid endosperm would never be associated with a hypoploid embryo, since, by being so, the embryo sac would have to carry two hypoploid nuclei at the micropylar pole. When a chromosome fails to disjoin at this division, such a situation would never occur. Yet, 5 (22%) embryos were found to be hypoploid in this study.

The results likewise rule out the possibility that the egg nucleus is a sister nucleus of one of the two polar nuclei (with the complementary constitution), since no small kernels would be expected to have an euploid embryo. The fact that 13 out of 22 embryos are of this type rejects this supposition.

In conclusion, the data of this study fit satisfactorily the supposition of nondisjunction at the second post-meiotic division. The aneuploid nature (hyperploid or hypoploid) of the endosperm of the small kernels does not affect this conclusion.

Bor-yaw Lin

COLUMBIA, MISSOURI
USDA-ARS and University of Missouri
AMES, IOWA
USDA-ARS and Iowa State University

**Significant reciprocal differences between $F_1$ crosses of Revolution and TELE**

"Revolution" was introduced into the U.S. because of its tolerance to the African maize streak virus. The original collection was made on the island of Réunion by Dr. K. Bock, who was a virologist for the Overseas Development Ministry of the U.K., then assigned to the East African Agriculture and Forestry Research Organization in Muguga, Kenya. Some tolerance to MDMV was noted during the quarantine greenhouse growout required for importation. Revolution flowered after all other nursery material when grown in Missouri; selection for earliness was done as part of the seed increase procedure.

"Tennessee early low ear" (TELE) was developed by Dr. L. M. Josephson at the University of Tennessee. When grown in Missouri, TELE is relatively early flowering and has a very desirable ear height.

Crosses were initially made between Revolution and Mo940, BS13, and a mixture of white lines with the gametophyte factor. Selection currently continues in material advanced from the first two crosses. Significant $F_1$ plant heterosis was noted with plant

Table 1. Revolution-TELE maternal effect evaluation, combined data from three sites.

| Entry | Cross type | Stand (%) | Plant height (cm) | Ear height (cm) | Yield (q/ha) | Moist (%) | Days to flower (no.) |
|---|---|---|---|---|---|---|---|
| TELE | 1 | 88.2 | 221.0 | 78.0 | 31.4 | 21.6 | 74.9 |
| TELE(TELE x Revolution) | 1(1x2) | 92.8 | 221.0 | 89.9 | 48.2 | 21.7 | 76.3 |
| (TELE x Revolution)TELE | (1x2)1 | 93.9 | 243.3 | 95.5 | 45.2 | 22.3 | 74.4 |
| (TELE x Revolution)Revolution | (1x2)2 | 88.3 | 270.0 | 141.0 | 54.0 | 24.4 | 80.9 |
| Revolution(TELE x Revolution) | 2(1x2) | 84.2 | 263.0 | 140.1 | 54.4 | 26.1 | 82.0 |
| TELE x Revolution | 1x2 | 89.2 | 231.5 | 101.7 | 61.3 | 23.6 | 74.9 |
| (TELE x Revolution) F2 | (1x2)F2 | 89.5 | 207.8 | 92.1 | 39.6 | 24.2 | 76.1 |
| Revolution x TELE | 2x1 | 80.2 | 259.3 | 133.8 | 46.6 | 27.2 | 80.6 |
| (Revolution x TELE) F2 | (2x1)F2 | 72.7 | 231.8 | 109.3 | 18.2 | 24.2 | 82.3 |
| TELE(Revolution x TELE) | 1(2x1) | 94.5 | 241.3 | 105.1 | 48.7 | 25.5 | 77.3 |
| (Revolution x TELE)TELE | (2x1)1 | 93.2 | 245.5 | 101.7 | 52.2 | 22.6 | 76.4 |
| (Revolution x TELE)Revolution | (2x1)2 | 81.4 | 252.5 | 129.6 | 41.2 | 23.3 | 78.8 |
| Revolution(Revolution x TELE) | 2(2x1) | 70.9 | 250.0 | 145.0 | 27.1 | 28.6 | 90.9 |
| Revolution | 2 | 47.7 | 218.5 | 134.8 | 5.8 | 40.0 | 92.5 |
| B73 x Mo17 | Check | 77.0 | 241.3 | 112.8 | 64.8 | 21.7 | 77.5 |
| Mean | | 85.4 | 239.8 | 114.0 | 44.6 | 24.5 | 79.4 |
| LSD 0.05 | | 8.7 | 17.1 | 15.1 | 12.3 | 3.7 | 4.3 |
| CV% (based on site x entry MS) | | 12.2 | 5.0 | 15.8 | 33.1 | 18.2 | 4.4 |
| Site means: Columbia, MO | | 81.1 | . | 106.5 | 51.7 | 19.9 | 77.9 |
| Columbia (ARC), MO | | 90.1 | . | 111.4 | 47.4 | 21.8 | . |
| Ames, IA | | 85.4 | 239.8 | 114.0 | 44.6 | 24.5 | 79.4 |

heights near 300 cm. In an attempt to "tame down" the $F_1$ response, crosses were made to TELE and, for contrast, to "Tennessee late low ear" (TLLE). Crosses were made both ways to increase seed production and to ensure retention of any cytoplasmic benefit from Revolution in subsequent virus selection. On growout, the $F_1$'s of Revolution and TLLE appeared similar, but a noticeable difference in flowering date and ear placement was observed between the $F_1$ crosses of Revolution and TELE.

The $F_1$ crosses were selfed and reciprocal backcrosses to both parents produced for evaluation to clarify the unusual behavior. Revolution, TELE, the $F_1$'s and $F_2$'s, the backcrosses to both parents, and B73 x Mo17 were grown at Rollins' Bottom, Columbia, MO; the Agronomy Research Center (ARC), east of Columbia, MO; and Ames, IA, in 1984.

Significant differences between TELE x Revolution and Revolution x TELE for stand percent (89.2 vs. 80.2%), plant height (231.5 vs. 259.3 cm), ear height (101.7 vs. 133.8 cm), yield (61.3 vs. 46.6 $qha^{-1}$), and days-to-flower (74.9 vs. 80.6) were observed (Table 1). Similar significant differences occurred in the $F_2$ generation for the same characters with plant and ear height being reduced and days-to-flower being increased. No differences among entries were found for root and stalk lodging. The greatest expression of differences was at Ames, IA, where the test material was most unadapted.

Differences among the backcross progenies of the form 1(1x2) vs. (1x2)1 and 2(1x2) vs. (1x2)2, as well as backcrosses of the (2x1) $F_1$ cross, were inconsistently significant. The ear height response is depicted graphically in Fig. 1. Ear height was measured at

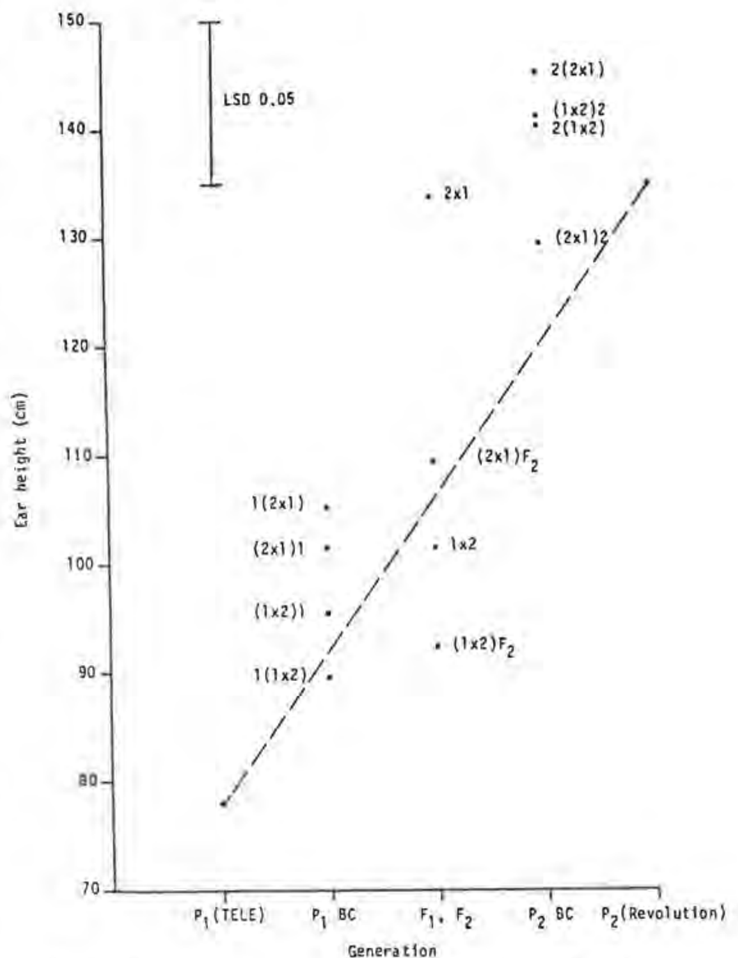



Fig. 1. Ear height responses of TELE, Revolution, their $F_1$, $F_2$, and backcross generations.

all sites and usually is thought of as being highly repeatable. Stands of Revolution per se were very poor. Yield measurements were not done on the full number of plots for Revolution because some plots were judged missing.

Seed of the crosses and parents is available in limited quantity from the University of Missouri.

L. L. Darrah, M. S. Zuber, and L. M. Pollak

DEKALB, ILLINOIS
DeKalb-Pfizer Genetics

**Producing single cross seed corn on hybrid plants**

Objective - To increase the quantity and quality of hybrid seed corn production by use of cytogenetic phenomena. Donor inbreds are developed whose chromosomes function in hybrid plant development (sporophyte) but do *not* take part in seed development (gametophyte). This results in single cross hybrid seed production on a vigorous hybrid plant instead of on a weak inbred plant.

Background - Mitosis, the process of nuclear division that accompanies plant growth, differs from meiosis, where the chromosome complement is reduced from diploid (2n) to haploid (n) to form gametes. The intent is to take advantage of natural differences between these two processes or to take advantage of an unusual genetic or cytological control of chromosomes in these two processes to cause chromosomes from a donor inbred to take part in mitosis but *not* in meiosis.

Definitions - Female inbred is a normal inbred that is used as the female in conventional production fields of a commercial hybrid. Male inbred is a normal inbred that is used as the male in production fields of a commercial hybrid. Donor inbred is a corn inbred that contains an unusual genetic or cytological entity that controls its own genome function when in hybrid combination with a normal inbred so that its chromosome complement will be included in mitosis but not be included in meiosis.

Procedure - First generation, the foundation seed is produced. The donor inbred is crossed with the female inbred; the donor inbred is used as a female to incorporate cytoplasmic male sterility. A series of donor inbreds of different flowering date will allow the particular female cross chosen to silk at the proper time to receive the pollen of the male inbred in the production field. Second generation, the production field is grown. The donor inbred times female inbred seedstock is grown as the female. The male inbred seedstock is grown as the male. Both seedstocks are planted at the same time and detasseling is not necessary in the sterile portion of the field. The single cross hybrid seed that is to be sold to the farmer is harvested from the hybrid female in the usual way.

Advantages - Seed yields are increased. Seed quality is improved. Same-time planting of male and female in production fields is achieved. Fewer seedstocks for sterile conversion are necessary in foundation seed.

A. Forrest Troyer

FREIBURG, WEST GERMANY
University of Freiburg

**Expression of zein genes and possible intron structure(s)**

The expression of zein genomic clones has been studied in our lab in *in vitro* transcription systems (Hela cell and *Xenopus* oocyte germinal vesicle extract systems: P. Langridge and G. Feix, Cell 34:1015-1022, 1983) and *in vivo* in yeast (P. Langridge et al., EMBO J. 3:2467-2471, 1984) and *Acetabularia.* In particular, the *Acetabularia* system has proved to be extremely valuable as zein genes are expressed to the protein level. Essentially, supercoiled plasmid DNA of zein genomic clones was injected into isolated *Acetabularia* nuclei. These nuclei were reimplanted into anucleated *Acetabularia* cells. After incubation, cytoplasmic smears were reacted with zein antibodies which were then reacted with an anti-IgG-fluorescein conjugate. Positive results, as detected by immunofluorescence, were observed for a number of genomic clones. *Acetabularia* is, therefore, a very valuable *in vivo* test system for the functionality of isolated zein genes.

In one case an isolated gene which gave a strong positive result in *Acetabularia* does not have the full reading frame. According to DNA sequence analysis, an insertion of an adenosine residue at +140 has occurred resulting in a termination codon after 48 amino acids. Such a clone would normally be looked upon as a "pseudogene"; however, due to the result in *Acetabularia,* this conclusion must be re-examined. Either a truncated protein which is still recognised by zein antibodies is produced, or some zein genes contain intron structures to overcome premature termination of translation. Until now the zein gene family has been thought to contain no intron structures. S1-mapping in the region of the single base insertion has shown S1 signals which may represent the borders of an intron. If this is the case, the intron(s) which may occur in some zein genes are postulated to be small ($\leqslant$ 50 bases) as they have not been detected by electron microscopic R-loop analysis.

J.W.S. Brown, P. Langridge and G. Feix

GAINESVILLE, FLORIDA
USDA-ARS and University of Florida

**Distribution of minilinear and minicircular mtDNA sequences within *Zea***

Maize and its relatives share a number of small minilinear or minicircular mtDNAs, ranging from ca. 7.4 to 1.4 kb. An inspection of recent literature suggests at least 20 such small molecules; common among these are the D1/D2, R1/R2, S1/S2, and 2.3/2.1 kb minilinear DNAs and the 1.9 and 1.4 kb minicircular DNAs. As part of investigations of mtDNAs in maize and teosinte, we have assembled a limited compilation of occurrence of these extra-principal-genome molecules. Occurrence of the DNAs was determined by probing with cloned S1/S2, 1.9, and 1.4 kb molecules, and by visual inspection of DNAs for the 2.3/2.1 kb minilinear DNAs. The data suggest an element of randomness of distribution within *Zea*.

Our survey (Table 1) is limited in extent, but nevertheless certain trends are indicated. We have examined at least 15 normal cytoplasms, two entries of T cytoplasm, five members of the C group, and nine members of the S group. The teosintes utilized were the same as previously described (Timothy et al., Proc. Natl. Acad. Sci. 76:4220, 1979). One seed lot of *Zea diploperennis* (designated 540), one seed lot of RU (ECU 398), and the Latin American race Guirua, were provided by D. H. Timothy, NC State, Raleigh.

**Table 1. Distribution of minilinear and minicircular mtDNAs among *Zea***

| | 1.4 | 1.9 | 2.3/2.1 | D1/D2,R1/R2,S1/S2 |
|---|---|---|---|---|
| MAIZE | | | | |
| N's to date | +/- | + | 2.3/2.1 | - |
| T's to date | - | + | 2.1 | - |
| C's to date | +/- | + | 2.3 | - |
| S's to date | +/- | +/- | 2.3 | S1/S2 |
| Racimo de Uva (ECU 398) | + | + | 2.3 | R1/R2 |
| Guirua | + | + | 2.1 | - |
| TEOSINTES | | | | |
| Central Plateau | - | + | 2.3 | - |
| Chalco | - | + | 2.3 | - |
| Balsas/Guerrero | - | + | 2.3 | - |
| Huehuetenango | - | - | 2.3 | - |
| Guatemala | + | + | - | - (integrated S2) |
| *Zea perennis* (ZP) | - | + | - | - (substantial integrated S1) |
| *Zea diploperennis* (ZD) | + | + | - | D1/D2 |

The 1.4 kb minicircular DNA is limited in distribution, present in some N's, C's, and S's, and in only Guatemala and *Zea diploperennis*. The 1.9 kb minicircular DNA is more widely distributed, but absent in some S's and in Huehuetenango. However, sequences of this molecule appear to vary in size in the teosintes, RU, and Guirua. This variation is reflected in apparent kb differences of undigested and restriction endonuclease-digested samples, with most bearing slightly larger molecules than the 1.9 kb molecule.

The 2.3/2.1 kb minilinear DNA is absent in only Guatemala, ZD, and ZP in entries examined to date. The 2.1 replaces the 2.3 kb DNA in T's, some N's, and in the Latin American race Guirua. An accession designated Puya Grande also carries the 2.1.

A number of these mtDNAs were probed with the three *Pst*I clones which comprise most of the S2 DNA, an internal 1.55 kb *Hin*dIII fragment of S1, and a 6.2 kb *Pst*I clone of S1, to survey the frequency of homologies to S1/S2 sequences in the principal genome. All normal cytoplasms examined to date and the teosintes Central Plateau, Chalco, Balsas/Guerrero, Huehuetenango, and ZP are similar and characterized by an apparently small deletion at the end of the S2 sequence which shares ca. 1500 bp homology with S1. Guirua has only trace homology to S2, which is unique among apparently normal, male fertile cytoplasms, and is a characteristic of the T and C cytoplasms. Guatemala apparently carries a total integrated copy of S2, as evidenced by intactness of the three *Pst*I fragments which comprise most of the molecule; the only other maize which is similar to date is S cytoplasm. Even though ZP is like normal cytoplasm maize in the S2 region, ZP may carry a large integrated part of the S1 molecule. One major *Bam*H1 fragment and at least two large *Pst*I fragments carry homology to the S1-specific *Hin*dIII 1.55 kb clone and to the *Pst*I 1.6 kb clone which includes most of the ca. 1500 sequence shared by S1/S2. A complete copy of S1 was not detected by virtue of absence of a 6.2 kb *Pst*I fragment which hybridizes to these two clones. Nevertheless, no other *Zea* cytoplasm examined to date except S yields evidence of integration of S1 to this extent.

Guatemala, ZP, and ZD, at least our seed lots, display unique and diagnostic endonuclease restriction patterns, but do share some pattern affinity. These patterns are distinct from other teosintes (Timothy et al., Proc. Natl. Acad. Sci. 76:4220, 1979) or other normal cytoplasms (Sederoff et al., Proc. Natl. Acad. Sci. 78:5953, 1981). ZD carries the free D1/D2 DNAs, which are similar to the R1/R2 DNAs, which may be progenitors of S1/S2. Although the significance of these observations is unclear at present, these teosintes may be intimately associated with the evolution of S1/S2 sequences. The three teosintes are unique in that they are the only *Zea* entries examined to date that lack the 2.3/2.1 minilinear DNAs. It is also interesting to note that defective endosperm is associated with the ZP cytoplasm in certain lines, and that Guatemala cytoplasm is also associated with a small seed trait.

These observations suggest randomness of distribution and independent evolution of extra-principal-genome mtDNA sequences in *Zea*. Sequences of these molecules appear to be dispensable to the genus, in that one entry or another may lack all of these sequences. However, an intimate role of S2 sequences, or more likely S2/R2/D2 sequences, in mtDNA rearrangements associated with reversion to fertility in the S cytoplasm is suggestive of involvement in the evolution of *Zea* mtDNA. That Guirua, T, and C cytoplasms share very little homology with S1/S2 indicates that these sequences, too, are not universally present in *Zea*.

D. R. Pring and A. G. Smith

GAINESVILLE, FLORIDA
University of Florida, USDA-ARS
ST. PAUL, MINNESOTA
University of Minnesota

**Mitochondrial DNA rearrangements associated with reversion of T cytoplasm to male fertility and disease resistance**

Male-fertile plants resistant to *Helminthosporium maydis* race T have been regenerated from tissue culture of susceptible, male-sterile T cytoplasm maize (A188). Most lines derived from these plants carry altered mitochondrial DNA, as evidenced by restriction patterns. Common among these alterations is the rearrangement of a 6.6 kb *Xho*I fragment (Gengenbach, et al., Theor. Appl. Genet 59:161, 1981; Brettell, et al., MNL 56:13, 1982; Gengenbach and Umbeck, MNL 56:140, 1982). Eighteen of 19 lines examined to date carry the rearranged fragment. One interesting mutant, T-4 (Gengenbach and Umbeck, MNL 56:140, 1982), retains the intact 6.6 kb *Xho*I fragment and is not detectably different from parental T cytoplasm mtDNA, at least as detected by visual examination after restriction endonuclease digestion with several enzymes. This mutant was used for more extensive study. 1900-colony cosmid libraries of parental T and T-4 mutant mtDNAs were constructed in pHC79 and Grunstein-Hogness hybridizations were performed with a 6.5 kb *Bam*H1 fragment of Wf9(N) mtDNA, which shares homology with the 6.6 kb *Xho*I fragment of T mtDNA (D. M. Lonsdale, personal communication).

Selected cosmids were isolated, hybridized, and mapped. Sequences of the 6.5 kb *Bam*H1 fragment from N cytoplasm hybridized to the intact 6.5 kb fragment and to a *Bam*H1 fragment of 9.0 kb in T cytoplasm. An *Xho*I fragment of 4.5 kb is internal to the 6.5 kb *Bam*H1 fragment, while the 6.6 kb *Xho*I fragment is internal to the 9.0 *Bam*H1 fragment. The 9.0 kb *Bam*H1 and 6.6 kb *Xho*I fragment sequences share homology with the 6.5 kb *Bam*H1 and 4.5 kb *Xho*I fragments. Mapping of these regions indicates that these fragments carry a recombination site; the two regions are co-linear to the recombination site, and diverge within the 6.5 and 9.0 kb *Bam*H1 fragments. No apparent differences were observed between T and T-4 with *Bam*H1 and *Xho*I. However, when a series of tetradeoxynucleotide-requiring restriction endonucleases were used on the isolated fragments, cosmids, or mtDNAs, *Alu*I identified a rearrangement which distinguishes T-4 from T. The 6.6 kb *Xho*I fragment from T cytoplasm mtDNA hybridized to an *Alu*I fragment of ca. 180 bp in T but not in T-4. Three *Alu*I fragments of ca. 200, 650, and 1000 bp are shared by the 4.5, 6.5, 6.6, and 9.0 kb fragments.

Lack of visual differences between restriction patterns of parental and mutant DNAs does not indicate that the DNAs are identical, as evidenced by the observations with *Alu*I. We do not know if other regions of the genomes are rearranged, or if this alteration is causal to the male-fertile or disease-resistant phenotypes. We have probed mitochondrial DNA from T and T-4 with a series of cosmids from N and S mtDNA, and detect no other differences with *Bam*H1 in about 250 kb of probed sequences. Based on these observations, therefore, we can only suggest that we have identified a small deviation which characterizes the T-4 mutant, which may or may not be associated with the phenotypes.

R. P. Wise, D. R. Pring, and B. G. Gengenbach

HYDERABAD, INDIA
Osmania University

**Effect of 2,4-D analogues on callus cultures**

One of the basic prerequisites for any tissue culture system is the establishment of efficient callus initiation and subsequent plantlet regeneration. Callus cultures were established from seedling roots and immature glumes on Linsmaier and Skoog (LS) and Murashige and Skoog (MS) media. In the present study, 2,4-dichlorophenoxy acetic acid (2,4-D) and its analogues such as 2,4-5 trichlorophenoxy acetic acid (2,4-5T) and 2(2,4-5 trichlorophenoxy) propionic acid (2,4-5P) were used in concentrations ranging from 0.5 - 4 mg/l for callus induction and maintenance of cultures. Callus growth was assessed by fresh and dry weight analysis after four weeks (See Table). The results indicate that low levels of 2,4-5T and 2,4-5P were superior over 2,4-D for callus initiation from root cultures whereas 2,4-D is the auxin requirement for glume explants. Multiple roots were observed in medium with low levels of 2,4-D, 2,4-5T and 2,4-5P. The differential optimal requirement of auxins for callus induction in roots and glumes may be due to physiological status of the explants and that auxin sites may have a higher affinity for analogues than 2,4-D (Sanchez et al., Ann. Appl. Biol. 98:347-353, 1981). Suspension cultures were also obtained from the above callus cultures in

Comparative effect of 2,4-D Analogues in induction of Callus growth from Maize Roots

| Auxin | | Concentrations tested (mg/l) | | | |
|---|---|---|---|---|---|
| | | 0.5 | 1.0 | 2.0 | 4.0 |
| 2,4-D | FW | 597.6 | 648.3 | 356.3 | 402.0 |
| | DW | 51 .6 | 51.9 | 35.3 | 38.3 |
| 2,4-5T | FW | 1009.3 | 599.3 | 416.3 | 164.6 |
| | DW | 73.6 | 52.6 | 43.0 | 20.6 |
| 2,4-5P | FW | 853.0 | 691.6 | 368.0 | 489.3 |
| | DW | 62.6 | 54.0 | 34.6 | 49.6 |

FW : Fresh Weight in mgs.
DW : Dry weight in mgs.

liquid medium. Further studies of plating of cells or cell aggregates and regeneration of roots and shoots are in progress.

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

## Induction of somatic embryos from root callus

Cultures were initiated from seedling roots of different genotypes of maize on LS medium containing 2.0 mg/l 2,4-D. After one month these cultures were transferred to 1.0 mg/l 2,4-D and 0.5 mg/l NAA and maintained on the same medium for about 5-6 subcultures. The friable callus disaggregated when suspended in a 250 ml culture flask containing 30 ml liquid medium on a shaker for about a week. We observed somatic embryos both in hormone-free and hormone-containing media. However, in hormone-free medium more somatic embryos were identified. These embryos were transferred to different levels of hormones containing media. Some of them germinated and exhibited only root initiation without any shoot formation. These results suggest that somatic embryos can be induced from root cultures, similar to immature embryo cultures as reported earlier (Lu, et al., Theor. Appl. Genet. 66:285-289, 1983). Studies are in progress with regard to standardization of media, concentration of various levels of sucrose and hormones involved in embryogenesis of root calli.

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

## Extraction and characterization of certain maize flavonoids

The flavonoid pigment system is governed by a number of genes that have been described in terms of their regulation in the synthesis of various classes of flavonoid compounds. Isolation and characterization of compounds accumulated in various tissues is needed to know about the factors controlling their synthesis. The *C* locus is one of the loci in the biosynthetic pathway for anthocyanin biosynthesis in maize (Reddy & Coe, Science 158:115, 1962) with several allelic forms, including *C-I*, the dominant inhibitor, which suppresses anthocyanin formation. The present report deals with the study of the chemical nature of accumulated substance(s) of *C-I* with chromatographic, spectrophotometric and chemical tests. *C-I* aleurone tissue showed the presence of more than one compound. One of the compounds was isolated by preparative TLC (ethyl acetate and methanol, 2:1) and characterized as caffeic acid by co-paper chromatography, UV, IR, Mass and 'H' NMR. Average Rf values and Abs. Max. in UV are given below.

| | Rf values | | | |
|---|---|---|---|---|
| Compound | B A W | Phenol-$H_2O$ | Forestal | Abs. max. |
| Caffeic Acid (Authentic) | 0.86 | 0.46 | 0.78 | 325,296 234,215 |
| Isolated Compound | 0.86 | 0.45 | 0.78 | 325,296 234,215 |

Study of the gene-product relationship in anthocyanin biosynthesis has been possible with the isolation and characterization of accumulated intermediates in single and double recessive mutant aleurone tissues. The double recessive of *bz pr* aleurone showed the presence of apigenin and was characterized by chromatography, UV, IR, Mass and 'H' NMR spectral studies. Average Rf and Abs. max. values are given below.

| | Rf values | | | | |
|---|---|---|---|---|---|
| Compound | B A W | m-cresol-HOAc-$H_2O$ | Phenol -$H_2O$ | Forestal | Abs. max. |
| Apigenin (Authentic) | 0.92 | 0.88 | 0.96 | 0.83 | 335,268 |
| Isolated | 0.91 | 0.88 | 0.97 | 0.83 | 335,268 |

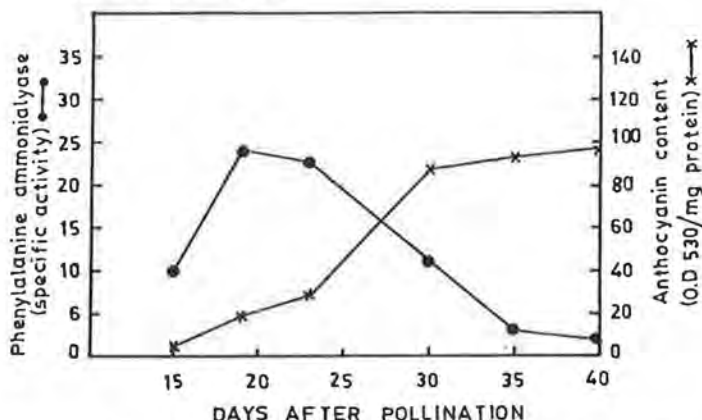
K. V. Rao, P. Suprasanna and G. M. Reddy

HYDERABAD, INDIA
University of Hyderabad

## Phenylalanine ammonia lyase activity and anthocyanin accumulation in the aleurone tissue

The phenylalanine ammonia lyase activity in the developing aleurone tissue of *C C* kernels was investigated in order to see whether there is any correlation between anthocyanin accumulation and phenylalanine ammonia lyase levels. Phenylalanine ammonia lyase was assayed (Zuker, 1965 and Saunders et al., 1975 with certain modifications) in acetone powder extracts of fresh aleurone tissue (25mM sodium borate buffer pH 8.8 with polyvinylpolypyrrolidone, mg/mg). The 3 ml reaction mixture consists of 1 ml of enzyme extract, 1 ml of sodium borate buffer and 1 ml of 10 mM L-phenylalanine. The reaction mixture was incubated at 40 C and O.D. at

290 nm was recorded at 30 and 60 minutes. One unit of enzyme activity is defined as that quantity of enzyme which catalyzes the formation of 1 µg of cinnamic acid per hour at 40 C. Protein content was estimated as per Lowry's method. The results are given in the figure.




These preliminary studies indicate that the phenylalanine ammonia lyase activity is at a maximum in 19 DAP aleurone and gradually decreases thereafter. Aleurones from 30 DAP kernels show significantly reduced levels of phenylalanine ammonia lyase activity. Anthocyanin accumulation reaches a peak around 30 DAP and continues to remain the same or increase slightly thereafter. Further, it was found that *C-I C-I* aleurone extracts also show phenylalanine ammonia lyase activity, though in greatly reduced amounts during development (data not shown). It was also observed that phenylalanine ammonia lyase activity was localized primarily in aleurone (84%) compared to the endosperm (5%) and the embryo (11%) tissue. Further studies using both radioactive and non-radioactive procedures including high performance liquid chromatography are in progress (We thank Prof. E.H. Coe Jr., University of Missouri for providing us the *C C* and *C-I C-I* stocks).

Ch. Jarayam and A. R. Reddy

## Chloroplast ultrastructure studies of the etched mutant

The virescent seedlings of *et et* (virescent) and normal genotypes were grown under light (1 w/$m^2$) at 25 C and leaves were harvested six days after germination and processed for transmission electron microscopy. The mutant leaves show a poorly developed chloroplast ultrastructure compared to normal (Fig. 1). The internal membrane structure of the mutant chloroplast is significantly affected and the grana are less numerous than the normal chloroplast. Further, it was found that there were fewer discs per granum in the mutant chloroplast. On the contrary, chloroplasts from the normal leaf preparations show no such defects and exhibit normal ultrastructural development and differentiation (Fig. 1). These observations are in agreement with our earlier data (Ramesh, Kumari and Reddy, Biochem. International 1984) showing pigment deficiencies in the 6-day old mutant seedlings grown under light.

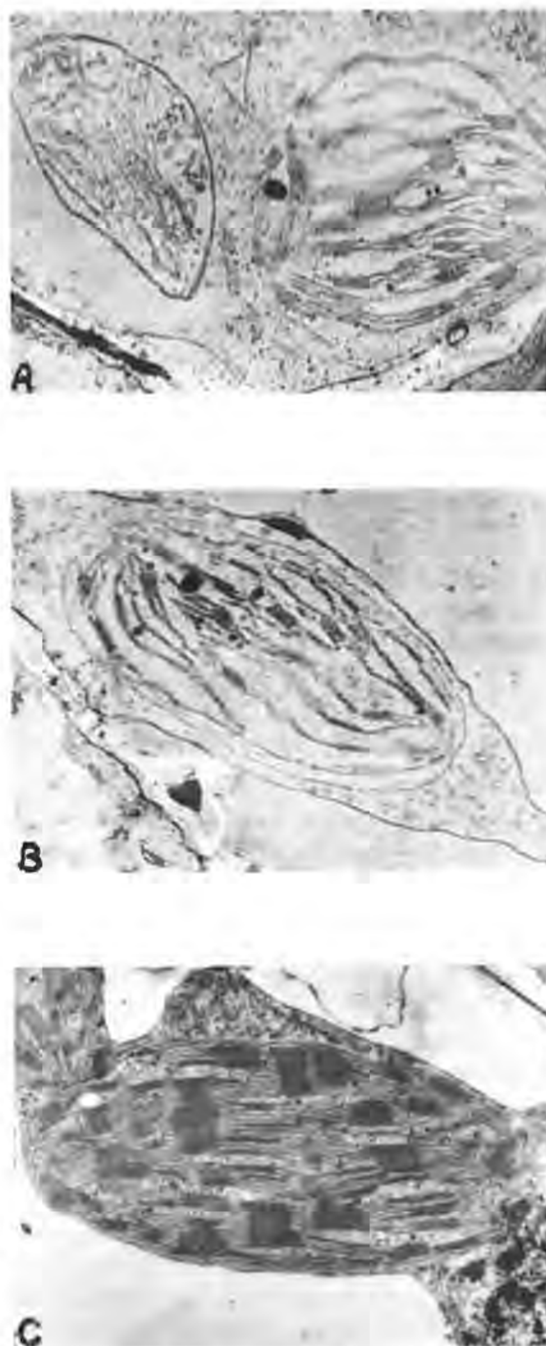



**Figure 1. Chloroplasts prepared from leaf samples of six day old seedlings grown under light (1 w/$m^2$) at 25 C. A and B, mutant *(et et)* chloroplasts from virescent seedlings showing poorly developed grana, membranes and abnormal amoeboid shape. C, normal chloroplasts from normal seedlings showing well-developed internal structures and membranes (X 20,000).**

S. V. Ramesh[1] and A. R. Reddy

[1]Dept. of Biology, Univ. of Utah, Salt Lake City, Utah

### Chloroplast pigment analysis in individual leaves of light-grown seedlings

Chlorophyll and carotenoid pigments of individual leaves of both *et et* and normal seedlings (light-grown 1 $w/m^2$ at 25 C) have been investigated in order to see whether these values agree with the whole seedling data. The 6-day old seedlings, under our experimental conditions, develop only two leaves whereas 10-day old seedlings show three leaves. These are designated as first, second and third leaf from the base, i.e., the top leaf is the third leaf and the bottom one (the oldest) is the first leaf. chl a/chl b and chl/carotenoid ratios have also been computed. Although the overall pattern of these ratios remains more or less the same among the corresponding leaves of mutant and normal, the absolute values of these pigments differ within and also between seedlings. Comparatively the mutant shows reduced levels of chlorophyll and carotenoids. A significantly greater variability and error was observed in individual leaf experiments compared to that of whole seedlings. The factors that contribute to this error include variation in the leaf size, overall physiological condition of the seedlings, etc. On the other hand, leaves of 10-day-old seedlings show no significant differences in any of the parameters studied. Further studies on the thylakoid membrane proteins, including chlorophyll-protein complexes, using SDS-PAGE and HPLC procedures are in progress.

S. Kumari, H. G. Sangeetha and A. R. Reddy

## IOWA CITY, IOWA
University of Iowa

### Duplication and stable inheritance of chromosomal segments

It is not difficult to duplicate various regions of the maize genome. The spontaneous origin of primary trisomics, the production of tertiary trisomics by translocations and the nondisjunction of $B^A$ chromosomes provide opportunities for duplication. However, it is difficult to produce a duplication that is inherited in a stable manner. Stable inheritance depends on a) formation of the homozygous (tetrasomic) x condition and b) production of only the duplication class of gamete by the homozygote. The latter problem is discussed here. Duplication homozygotes often produce normal gametes as well as the duplication class, due to their synaptic properties. B-A translocations may be useful in producing homozygous duplications with stable inheritance. Two methods are proposed, one involving proximal chromosomal regions and the other distal regions.

a) *Proximal regions.* It was recently shown that proximal chromosomal regions can be duplicated using B-A translocations (Maydica 28:317, 1983). In the procedure, two opposite arms B-A translocations were combined by crossing over to form a double translocation. TB-9Sb and TB-9La were used. The double translocation consists of a $B^9$ from each translocation plus a composite $9^B$. The $9^{B(La+Sb)}$ contains proximal regions of 9S and 9L. It was separated from the other translocation chromosomes and used to duplicate the central region of chromosome 9. Plants containing 9 9 $9^{B(La+Sb)}$ and 9 9 $9^{B(La+Sb)}$ $9^{B(La+Sb)}$ were constructed.

In the duplications described above, $9^{B(La+Sb)}$ is supernumerary and easily lost during gamete formation. However, proximal 9S and 9L regions can be duplicated in a different manner that is potentially stable. The procedure involves substituting two intact $9^B$ chromosomes ($9^{BLa}$ and $9^{BSb}$) for chromosome 9. The homozygous duplication constitution is $9^{BLa}$ $9^{BLa}$ $9^{BSb}$ $9^{BSb}$. Regular transmission of the duplication chromosomes by the homozygote should occur because neither $9^{BLa}$ nor $9^{BSb}$ is dispensable. They both carry unique vital genes in addition to the duplication. However, the duplication could break down through crossing over between $9^{BLa}$ and $9^{BSb}$, with production of a normal 9. The rate of crossing over depends on the pattern of synapsis in the duplication homozygote. The work of Burnham et al. (Genetics 71:111, 1972) indicates that pairing initiates in distal chromosomal regions. Since $9^{BLa}$ and $9^{BSb}$ do not have distal regions in common, bivalent pairing may prevail in the homozygote ($9^{BLa}/9^{BLa}$ and $9^{BSb}/9^{BSb}$). If so, crossing over between $9^{BLa}$ and $9^{BSb}$ will be prevented.

b) *Distal regions.* B-A translocations can also be used to duplicate distal chromosomal regions. In this case the $B^A$ chromosome is separated from the $A^B$. The $B^A$ is combined with chromosomes from a standard (A-A) translocation to produce the duplication. However, it is not the $B^A$ segment that is duplicated. Instead, the $B^A$ is used to allow survival of gametes produced by adjacent segregation from the translocation. For example, a T1-2 translocation heterozygote may produce the $1^2$ 2 chromosome combination by adjacent-1 segregation. It contains a duplication of a chromosome 2 segment and a deficiency of a chromosome 1 segment. If a $B^1$ chromosome is present with the correct exchange point to cover the deficiency, the gamete will be viable. The homozygous duplication contains $1^2\,1^2\,2\,2\,B^1\,B^1$. The duplication has the potential for stable inheritance because a) none of the chromosomes involved is dispensable; b) crossing over between the duplicated regions on $1^2$ and 2 does not cause reversion to normal chromosomes; c) the $B^1$ is not subject to nondisjunction in the absence of $1^B$.

The techniques involved in constructing the duplications will be described elsewhere *(Critical Reviews in Plant Science,* in press). The methods are not considered difficult and production of the duplications should be straightforward.

W. R. Carlson

### Initiation of synapsis by proximal chromosomal regions

Studies by Burnham et al. (Genetics 71:111, 1972) showed that synapsis in maize usually or always begins in distal chromosomal regions. The proximal parts of chromosome arms don't participate in synaptic initiation. The finding raises the question of whether distal chromosomal sites possess a unique capacity for synapsis that is absent in proximal regions. The idea was tested using a double translocation which combines TB-9Sb and TB-9La, as described in the accompanying article. Crosses were made of *bz bz* x heterozygous TB-9(La + Sb). Progeny were selected with the *Bz* kernel phenotype and *bz* plant phenotype (*B* and *Pl* were present in the tester for plant classification of *bz*). The nondisjunctional phenotype demonstrated transmission of the double translocation. It also indicated the absence of $B^{9Sb}$ in selected plants. Tassel samples were taken from the bronze plants and the meiotic stages examined. Several cases were found in which $B^{9La}$ was absent as well as $B^{9Sb}$. These are double hypoploids with the chromosome constitution 9 $9^{B(La+Sb)}$. In these plants, pairing between 9 and 9 $^{B(La+Sb)}$ can be easily studied in diakinesis. If pairing and crossing over occurs, ten bivalents are formed. If pairing does not occur, 9 bivalents plus two univalents result. Data are given in Table 1.

**Table 1: Pairing in diakinesis between 9, $9^{B(La+Sb)}$ chromosomes. Observations were made by Lekkala Reddy (LR), and Wayne Carlson (WC). Occasional cells that showed 9 pairs plus one univalent were assumed to have a second univalent associated nonhomologously with a bivalent.**

| Plant Number | Observer | 10 pairs | 9 pairs + 2 univalents |
|---|---|---|---|
| 3352-8 | LR | 27 | 14 |
| | WC | 14 | 16 |
| 3352-13 | WC | 26 | 18 |
| 5611-1 | LR | 78 | 70 |
| | WC | 27 | 12 |
| | | 172 (57%) | 130 (43%) |

Pairing between 9 and $9^{B(La+Sb)}$ must initiate in proximal regions of chromosome 9. The $9^{B(La+Sb)}$ chromosome carries the proximal 0.4 of 9S and 0.4 of 9L, but no distal regions. Since pairing was found in 57% of cells examined, it is obvious that proximal initiation of synapsis can occur when distal initiation is not possible. Whether proximal initiation is less efficient than distal initiation is not certain. Pairing of 9 and $9^{B(La+Sb)}$ is well below 100%. However, the map distance covered by the regions on $9^{B(La+Sb)}$ is short and may, by itself, account for the lack of pairing in 43% of cells.

A general explanation for pairing between 9 and $9^{B(La+Sb)}$ will be proposed. Under normal circumstances, distal alignment of chromosomes precedes pairing and causes distal synaptic initiation. When distal alignment is absent, a slower method of recognition causes proximal synapsis. The slower method of recognition could involve a tendency for unpaired chromosomes to associate, regardless of homology, after the initial period of synapsis. The association of unpaired chromosomes would only be stable when homology was present between them.

W. R. Carlson

### Calculation of meiotic loss for TB-9Sb

Robertson (Genetics 55:433, 1967) determined the meiotic properties of TB-9Sb heterozygotes. He crossed genetically marked heterozygotes as female to a chromosome 9 tester: 9*(c wx)* $9^B$*(Wx)* $B^9$*(C)* X *c c wx wx*. Three major classes were found in the progeny, corresponding to transmission of 9*(c wx)*; 9 $B^9$ *(C wx)* and $9^B$ $B^9$ *(C Wx)*. The three classes were found in approximately equal frequency, although the chromosome 9 class was somewhat larger than the other two. Robertson concluded that $9^B$ always disjoins from 9 in meiosis but the $B^9$ goes randomly to one pole or the other. The result is production of 4 gametic classes, with one class ($9^B$) being lethal. The excess of the chromosome 9 class was attributed to occasional meiotic loss of the $B^9$.

Recently, very high rates of meiotic loss have been detected in certain derivatives of TB-9Sb which lack nondisjunction (unpublished observations). The finding necessitates development of a method for calculating rates of meiotic loss. Three classes of meiotic disjunction must be considered in the calculation:

a. Proper disjunction of 9 and $B^9$ (with 9-$9^B$ disjunction).
   Meiotic products: 9 and $9^B$ $B^9$
b. Meiotic nondisjunction of 9 and $B^9$ (with 9-$9^B$ disjunction).
   Meiotic products: 9 $B^9$ and $9^B$ (lethal)
c. Meiotic loss of $B^9$ (with 9-$9^B$ disjunction).
   Meiotic products: 9 and $9^B$ (lethal)

A fourth category, meiotic nondisjunction of 9 and $9^B$, is uncommon and will be considered later.

There are two problems in calculating rates of meiotic loss. First, not all meiotic products are viable. Therefore, testcross data are not representative of meiotic events. The solution is to select testcross progeny that received chromosome 9*(wx)*. The selected kernels give a representative measure of each meiotic class, and the method eliminates the problem of inviability. Linkage of *Wx* to $9^B$ is so strong that genetic classification of 9*(wx)* vs. $9^B$*(Wx)* is virtually error free (Robertson, 1967).

The second problem in analyzing testcross data comes from crossing over between 9*(c)* and $B^9$*(C)*. Calculation of meiotic loss will first be described on the assumption that crossing over between 9 and $B^9$

is absent. A correction will later be added to the basic formula. Meiotic loss in the absence of crossing over is: *(c wx - Wx)/wx*. The denominator is *"wx"* because only chromosome 9*(wx)*-containing gametes are being analyzed. The numerator is a measure of *(wx)* gametes that originate by meiotic loss. Since two types of disjunction (categories a and c above) produce the chromosome 9 class, total *c wx* is not a measure of meiotic loss. Subtraction of *Wx* from *c wx* removes an amount equal to the chromosome 9 class from category a. As a result, the numerator contains only kernels produced by meiotic loss.

The formula must be modified to account for crossing over that produces 9*(c wx)* $B^9$*(c)* and 9*(C wx)* gametes. A 9*(c wx)* $B^9$*(c)* gamete gives the same phenotype as 9*(c wx)* and could incorrectly contribute to the numerator of the formula. A 9*(C wx)* gamete could be mistaken for 9*(c wx)* $B^9$*(C)* and incorrectly be left out. Unfortunately, the two misclassifications of chromosome type do not cancel each other out and must be separately accounted-for. A third crossover class, $9^B$*(Wx)* $B^9$*(c)*, can be used to make the corrections. All members of the *c Wx* class result from crossing over. As a result, they can be used as a measure of 9-$B^9$ crossing over. The *c Wx* individuals result from only one class of disjunction: category a. They are equivalent in number to the crossover chromosome 9*(C wx)* class that originates from category a disjunction. Evidence is given in *Critical Reviews in Plant Science* (in press) that crossing over tends to prevent meiotic loss, so that 9*(C wx)* should seldom originate from category c disjunction. Consequently, the *c Wx* class can be used to correct for 9*(C wx)* crossovers by adding it to the numerator: Meiotic loss = *[(c wx + c Wx) - Wx]/wx*. This ensures that all chromosome 9 gametes are accounted for, prior to subtraction of *Wx*.

The other crossover class, 9*(c wx)* $B^9$*(c)*, must be removed from the *c wx* phenotypic class since it is a 9 $B^9$ gametic class. This crossover results from category b disjunction. If disjunctional categories a and b occur with equal frequency, as suggested by Robertson, the 9*(c wx)* $B^9$*(c)* class should equal one-half of the $9^B$*(Wx)* $B^9$*(c)* class. Subtraction of ½ *c Wx* from the numerator makes the correction (the 50% rate depends on the equal chance of forming 9*(C wx)* $B^9$*(c)* and 9*(c wx)* $B^9$*(c)* classes by category b disjunction). However, Carlson (MNL 52:38, 1978) found a tendency for category a disjunction to occur more frequently following crossing over than category b. If the extreme assumption is made that category b disjunction never occurs following crossing over (which is untrue), the correction for 9*(c)* $B^9$*(c)* approaches zero. A range of corrections to the numerator can, therefore, be made. Subtraction of zero to one-half of the *c Wx* class makes the correction. Meiotic loss then becomes: *[(c wx + c Wx* - 0 to 0.5 *c Wx) - Wx]/wx*. Simplified, the formula is: *[(c wx* + 0.5 to 1.0 *c Wx) - Wx]/wx*.

A recent finding by Kindiger, Beckett and Curtis (MNL 58:66, 1984) can also be incorporated in the calculation. They found evidence for the production of A $A^B$ gametes by B-A translocation heterozygotes. In TB-9Sb heterozygotes, this could account for some of the *c Wx* class. As a result, the previous corrections for crossing over would be incorrect. If the extreme assumption is made that all *c Wx* kernels result from 9*(c wx)* $9^B$*(Wx)* gametes, it follows that the crossover $B^9$*(c)* class does not exist. In this case, no correction for crossing over is needed and the formula for meiotic loss reverts to the original one presented.

Taking all possibilities into account, the formula becomes: meiotic loss = *[c wx* + (0 to 1.0 *c Wx) - Wx]/wx*. The formula can be applied to Robertson's data in his Table 6. The range of values obtained is 10.7 to 15.1% meiotic loss. (It is also possible to calculate the rate of category a disjunction as *Wx/wx*. Category b disjunction can be determined by subtraction. It is assumed, in the latter case, that meiotic nondisjunction of 9 and $9^B$ makes up a small proportion of meiotic events).

W. R. Carlson

ITHACA, NEW YORK
Cornell University

**Somaclonal variation in plants regenerated from the inbred W182BN**

Progeny of plants regenerated from callus cultures of W182BN demonstrated interesting and potentially useful somaclonal variation. The variants can be assigned to several different categories:

1) Mutant phenotypes such as brachytics, dwarfs, virescents, seedling lethals, leaf splotch mutants and others that resemble traditional single gene mutants. Most of these appear to involve single recessive mutations. Some appear to be single dominant mutants.
2) Variations in plant size and vigor, plant height, ear height, number of ears, number of tillers and plant color were also observed. Some of these variant lines have been selfed to a homozygous state and are promising as good, vigorous, healthy inbred lines. Several of these have been tested in hybrid combinations. Their performance was outstanding. Additional testing will be conducted this year.
3) Variation in cytoplasmic male sterility has also been observed. This included reversions from cms-S sterility to fertility, apparent nuclear mutations to fertility restoration, and the origin of cytoplasmic male sterility from non-male-sterile callus cultures.

Further characterization of these somaclonal variants is in progress, and publications describing the techniques and results are in press.

E. D. Earle and V. E. Gracen

**Mosaic tassels in iojap progeny**

Of more than 500 plants examined in the winter corn nursery at Homestead, Florida, 3 individual plants were found that appeared to have mosaic tassels with distinct fertile and sterile sectors. The individuals were among progeny of $F_2$ plants expressing the iojap phenotype crossed with the inbred W182BN. The $F_2$ plants originated from crosses of iojap males onto non-restoring inbreds. Sectors of these tassels appeared fully fertile, producing viable pollen. Anthers were either not exserted on sterile sectors or exserted anthers were small and flat and did not produce viable pollen. These individuals were crossed by the inbred W182BN which maintains most known cytoplasmic steriles. Fertility levels of the resulting progeny will be studied. Any cytoplasmic male sterile individuals found will be further characterized by genetic studies. We feel that these plants may give rise to additional cytosteriles similar to those reported earlier from iojap sources (MNL 58:102-103).

C. A. Lemke, V. E. Gracen and H. L. Everett

KINGSTON, RHODE ISLAND
University of Rhode Island

**Expression of a plump phenotype in the endosperm, independent of bronze**

In the summer crop of 1984, testcrosses were made to screen for revertants of shrunken mutations that were recovered from "Aberrant Ratio" stocks. Among the offspring of plants that were heterozygous for several shrunken mutations, a plump phenotype segregated. Crosses of *sh bz* individuals as male parents to both *sh-5586 Bz/sh-5588 Bz* and *sh-5586 Bz/sh-bz-x2* heterozygotes generated plump offspring (*sh-bz-x2* is a deletion of the shrunken and bronze loci). The pedigrees of the plump individuals are shown in Figure 1. Several observations can be made from the segregation patterns. First, from the cross of *sh-5586 Bz/sh-5586 Bz* x *sh-5588 Bz/sh-bz-x2*,

```
Cross:  sh-5586 Bz/sh-5586 Bz x sh-5588 Bz/sh-bz-x2

   (1) F1 sh-5586 Bz/sh-5588 Bz  x  sh bz

     3/4 of ears all shrunken
     1/4 of ears segregate 1 plump:1 shrunken

   (2) F1 sh-5586 Bz /sh-bz-x2  x  sh bz

     3/4 of ears all shrunken
     1/8 of ears segregate
        1 plump, purple:1 shrunken, purple
     1/8 of ears segregate
        1 plump, purple:1 shrunken, purple
        :1 plump, bronze:1 shrunken, bronze
```

Figure 1. Pedigrees of the ears on which shrunken and bronze segregate independently.

plants were produced in a 3:1 ratio, 3 which bore no plump kernels in the following generation and 1 that segregated for plump and shrunken. The number of plants tested was 83, 63 of the first class and 20 of the second. On those ears which contained plump kernels, the ratio was 1 plump:1 shrunken; moreover, the phenotypes of shrunken and bronze assorted independently, indicating that the gene responsible for the plump phenotype is not linked to bronze. A chromosomal aberration with one break between the shrunken and bronze loci could have occurred; however, such an event would generate substantial ovule abortion and none was seen at levels above background.

An explanation which can account for the data is that the minor sucrose synthetase gene (or another gene which codes for the enzyme) has been derepressed. According to the proposed model, a dominant inhibitor was heterozygous in both parents of the first cross (see Figure 2) and plants in the following generation segregated 3:1 for the presence of this suppressor. Plants with the inhibitor bore

```
Cross:  sh Bz/sh Bz, Inh/inh  x  sh Bz/sh-bz-x2, Inh/inh

   3/8 sh Bz/sh Bz, Inh/-
   1/8 sh Bz/sh Bz, inh/inh
   3/8 sh Bz/sh-bz-x2, Inh/-  x  sh bz
        all shrunken, 1 purple:1 bronze
   1/8 sh Bz/sh bz-x2, inh/inh*  x  sh bz*
        *To account for the 1:1 ratio of plump and shrunken
         kernels, a second factor affecting the phenotype
         must be heterozygous in one parent and homozygous
         recessive in the other
        1 plump, purple:1 plump, bronze
        :1 shrunken, purple:1 shrunken, bronze
```

Figure 2. Proposed inheritance pattern of minor sucrose synthetase inhibitor (Inh) and second factor.

ears with all shrunken kernels while progeny on ears from those plants lacking the suppressor segregated 1:1 for plump and shrunken. Since the 3:1 segregation for plump-bearing ears occurred in the generation prior to the one in which the plump phenotype was expressed, the genotype of the maternal parent apparently plays a determining role in the phenotype of the offspring. This could be due, for example, to a repressor that is produced in the megaspore mother cell and then carried over to the embryo sac. Or, a regulatory factor could be involved in a presetting or imprinting whereby repression of genes expressed in the endosperm occurs in the previous generation.

The action of a second factor affecting expression must be invoked to explain the 1:1 ratio on ears containing plump and shrunken kernels. Either the mutant heterozygotes (second generation in Figure 1) or the *sh bz* parent was heterozygous for this gene and the other parent homozygous recessive. This factor could be a second regulatory component or the structural gene.

J. P. Mottinger

LINCOLN, NEBRASKA
University of Nebraska

### Chromosomal location of a gene for resistance to *Corynebacterium michiganense* ssp. *nebraskense*

Leaf Freckles and Wilt (LFW) (Goss's Wilt), caused by *Corynebacterium michiganense* ssp. *nebraskense,* is a foliar maize disease occurring throughout Nebraska and spreading in surrounding states. A set of 27 reciprocal translocation stocks in the M14 background (resistant to LFW) were crossed to susceptible inbred A632. The F1's were selfed, and plants grown from F2 seed were classified for pollen and selfed. F3 seed from the normal pollen plants were separated by the presence or absence of the translocation-linked waxy gene marker and were planted in paired rows. Some translocation stocks had insufficient segregation for waxy and normal F3 seed from different normal pollen F2 plants. For these stocks, a waxy-marked row was paired with two rows still segregating for the heterozygous translocation/homozygous normal conditions. Inoculation of *C. m.* ssp. *nebraskense* was performed at the five-leaf stage, and individual plants were scored from 0 to 5 for disease reaction (0 = no symptoms to 5 = dead) 7, 17, and 42 days later. Mean disease readings were compared between paired rows and t-tests were performed. The 42-day readings were determined unreliable due to secondary pathogens and environmental stress. The only significant differences detected between paired rows involved the T7-9a and T7-9b translocations. Each stock has a break on the different arms of chromosome 7 and a break on the short arm of chromosome 9. Stocks marking similar breakpoints in chromosome 9 but having translocations with chromosomes other than 7 did not show significant differences. This indicates that a gene or genes for resistance may be located on chromosome 7. The present data are preliminary and confirmation of these findings is needed, particularly for translocation T7-9b, which expressed significance for only one reading. No significant differences between paired rows for 25 of 27 translocation stocks indicates resistance may be quantitative, which is consistent with earlier research. This year planned experiments are designed to evaluate whether resistance genes can be detected that have effects only in certain environments or only at certain time intervals after inoculation.

| Translocation/Break Points | | Difference Between Means | |
|---|---|---|---|
| | | Reading #1 | Reading #2 |
| T7-9a | 7L.63-9S.07 | 1.19* | 1.64* |
| T7-9b | 7S.76-9S.19 | 1.03* | 0.30 |
| T5-9(4817) | 5L.06-9S.07 | 0.29 | 0.08 |
| T1-9(4995) | 1L.19-9S.20 | 0.46 | 0.57 |
| T9-10b | 9S.13-10S.40 | 0.09 | 0.19 |
| A632 - M14 Checks | | 1.63* | 1.37* |

* t-test significant at .05 probability level.

Torbert R. Rocheford, Charles O. Gardner
and Anne K. Vidaver

### Growth of *Corynebacterium michiganense* ssp. *nebraskense* on maize callus

Maize callus tissue derived from hybrids Mo17 x B73 and A632 x A619 and inbred A632 were inoculated with *Corynebacterium michiganense* ssp. *nebraskense* colony forming units (CFU) and monitored for population growth at time intervals. Mo17 x B73 is field-resistant to Goss's wilt whereas A632 x A619 and A632 are susceptible. Seed from each genotype was germinated for five days in the dark at 30 C. Slices from the scutellar node were placed on modified Murashige and Skoog medium and routinely transferred. Callus pieces (approx. 0.1 gram) were abraded with microforceps prior to application of 2 or 5 μl of inoculum. At sampling times, callus pieces were homogenized in 5 ml of phosphate buffer. This mixture was serially diluted and plated onto nutrient medium. Results (Tables 1 and 2) indicate that bacterial populations at most sampling points were lower in callus initiated from the resistant genotype than in callus initiated from the susceptible genotype.

**Table 1. Estimates of *Corynebacterium nebraskense* cell concentration in 1 ml of phosphate buffer used to homogenize callus.**

| | Hours after inoculation | Bacterial Population (Colony-forming units/ml) | |
|---|---|---|---|
| | | Mo17 x B73 | A632 x A619 |
| Exp. #1 | | | |
| | 18 | $2.5 \times 10^1$ | $2.5 \times 10^1$ |
| | 72 | $5.4 \times 10^5$ | $1.2 \times 10^6$ |
| | 192 | $9.1 \times 10^6$ | $5.7 \times 10^7$ |
| | 240 | $5.0 \times 10^7$ | $4.6 \times 10^7$ |
| Exp. #2 | | | |
| | 24 | $2.2 \times 10^2$ | $3.3 \times 10^2$ |
| | 48 | $6.5 \times 10^1$ | $8.5 \times 10^1$ |
| | 96 | $5.6 \times 10^5$ | $1.8 \times 10^6$ |
| | 720 | $5.1 \times 10^8$ | $2.1 \times 10^9$ |

Inoculum: 1 - 5 colony-forming units

**Table 2. Estimates of *Corynebacterium nebraskense* cell concentration per gram of callus tissue.**

| Hours after inoculation | Bacterial Population Colony-forming units/gram callus | | |
|---|---|---|---|
| | Mo17 x B73 | A632 x A619 | A632 |
| 48 | $6.3 \times 10^8$ | $1.5 \times 10^9$ | --- |
| 96 | $1.7 \times 10^9$ | $3.2 \times 10^9$ | $3.1 \times 10^{10}$ |
| 144 | $2.6 \times 10^{10}$ | $1.2 \times 10^{10}$ | $3.3 \times 10^{10}$ |

Inoculum: 110 - 130 colony forming units

Another experiment tested callus initiated from immature embryos of inbreds W64A and WF9T, both resistant to Goss's wilt. Differences between

these two genotypes were not detected. Bacterial growth in these cultures was much lower than anticipated. Callus initiated from the scutellar node of A632 was also inoculated and assayed at one time interval as a comparative check. At 88 hours A632 callus contained 1 x $10^9$ CFU/gram tissue, whereas W64A and WF9T callus contained no more than 3 x $10^6$ CFU/gram tissue. The different method of callus initiation may have affected bacterial growth in W64A and WF9T. Alternatively, these genotypes may inhibit bacterial growth in callus to a greater extent than Mo17 x B73. Bacterial growth in callus initiated from immature embryos of both resistant and susceptible genotypes will be examined. The results indicate some promise for use of callus cultures in screening for resistance to this bacterial pathogen. The time interval of 48-96 hours after inoculation currently appears most suitable for detection of bacterial population differences.

T. R. Rocheford, A. K. Vidaver and C. O. Gardner

**Effect of wind-generated sand abrasion on infection by *Corynebacterium michiganense* ssp. *nebraskense***

Sand and wind damage may facilitate entry of *Corynebacterium michiganense* ssp. *nebraskense* cells into maize seedlings. To test this hypothesis, wind tunnel experiments were conducted courtesy of the U.S.D.A. Wind Erosion Research Unit at Kansas State University. Fourteen day old seedlings of Mo17 x B73 (resistant) and A632 x A619 (susceptible) were given three treatments: 1) wind only, followed by spray inoculation; 2) sand abrasion followed by spray inoculation; and 3) abrasion with sand containing inoculum. Seedlings were exposed to sand abrasion for 10 minutes at a rate (31 grams/cm width/minute) that simulates erosion before thunderstorms. Spray inoculation consisted of applying 5 ml of inoculum (2 x $10^8$ colony forming units/ml) per pot of three seedlings. The sand/inoculum mixture contained one ml of inoculum per 115 grams of sand. Both wind and sand treatments followed by spray inoculation resulted in infection and expression of disease symptoms in susceptible A632 x A619 seedlings. *C. m.* ssp. *nebraskense* colony forming units (CFU) were isolated from plants exposed to these treatments. The wind treatment caused damage to leaf tips, which may have allowed bacterial cell entry. Sand treatment damaged both leaf tips and leaf surfaces, which caused inoculum droplets to adhere to the leaf surface. The sand/inoculum treatment did not produce disease symptoms in the susceptible A632 x A619 seedlings. The resistant Mo17 x B73 did not express symptoms after all treatments.

This experiment was repeated with the following changes: all plants were staked before treatment to assure contact of the sand with the plants, genotypes used were A632 x A619 and A632, bacterial inoculum concentration was increased to 2 x $10^9$ CFU/ml, and 1 ml per 125 grams of sand were mixed together for the sand/inoculum treatment. Fourteen days after the treatments, there were no distinct disease symptoms on any of the plants. *C. m.* ssp. *nebraskense* CFU were isolated from plants that received the wind followed by spray inoculation treatment and from plants that received the sand abrasion followed by spray inoculation treatment. Bacterial CFU were not isolated from plants that received the sand abrasion/inoculum mixture treatment. This experiment was still in progress at the time this report was submitted. The results of these experiments indicate that both wind and sand damage may facilitate entry of *C. m.* ssp. *nebraskense* cells into maize seedlings. Planned experiments are designed to evaluate the effect of higher levels of inoculum for the sand/inoculum treatment.

T. R. Rocheford, A. K. Vidaver, C. O. Gardner and D. L. Armbrust

LLAVALLOL, BUENOS AIRES, ARGENTINA
Universidad Nacional de La Plata

**Tissue cultures of *Zea mays* x *Zea perennis* x *Zea diploperennis***

The experiments described here were conducted with a greenhouse $F_1$ generation of the interspecific tri-hybrid *Zea mays* x *Zea perennis* x *Zea diploperennis*, obtained by María del Carmen Molina in our Institute (MNL 58:114-115, 1984).

Immature seeds were removed and were sterilized with 2% sodium hypochlorite and washed in sterile distilled water. Immature embryos that varied in size from 1.0 to 2.0 mm long were excised and placed scutellar-side-up on a solid agar medium. Calli were initiated on the same three media (A-B-C) described earlier (Rapela, MNL 58:106-108, 1984). The cultures were incubated in the dark at 27 C.

The responses of the hybrid embryos were similar to the responses of *Zea mays* embryos. In Medium A, the scutellum of cultured immature embryos produced an opaque, white to pale yellow, soft and friable callus. Structures resembling the organized scutellum of the original explant were not observed in these calli. In Medium B and Medium C, the scutellum produced an opaque, white to pale yellow and compact callus. Organized structures resembling the early stages of zygotic embryos were observed after 20 days in culture. Calli formation were obtained at more or less similar rates in the three media used (Table 1).

Root regeneration, but no shoots, was obtained after the transfer of calli from Medium A to asparagine-minus Medium A without 2,4-D in light. Root and shoot regeneration were obtained after the

transfer of calli from Medium B and Medium C to 2,4-D free MS medium with 2% sucrose in light.

**Table 1. Frequency (in %) of callus formation. 1 = % of immature embryos forming soft and friable callus. 2 = % of immature embryos forming compact callus. 3 = % of compact callus forming somatic embryos.**

| Genotype | Medium A | Medium B | | Medium C | |
|---|---|---|---|---|---|
| | 1 | 2 | 3 | 2 | 3 |
| Tri-hybrid | 40 | 40 | 50 | 60 | 33 |

It is important to point out that, as with maize immature embryos, embryogenic callus was obtained both in Medium B and in Medium C. In spite of the strong differences between media, only the osmotic components (sucrose and proline) seem to have importance for somatic embryo formation. These facts lead us to suppose that it is possible to develop a very simple synthetic medium to obtain high rates of somatic embryogenesis in maize, as in related species.

Miguel Angel Rapela

## Maize organogenesis and somatic embryogenesis: stage of the donor tissue

There are many factors that influence maize callus growth in vitro, including genotype, source and stage of donor tissue and medium components. In previous investigations (Rapela, MNL 58:110-112, 1984) we have detected differences in the percentage of formation of compact callus tissue and of such callus forming somatic embryos due to the genotype and the effect of the medium. New results in our laboratory reveal that genotype (inbred or hybrid nature of the explant) is not an important factor in determining the response for somatic embryogenesis in vitro. Also, differences between media basal components did not have as much influence on somatic embryogenesis as did the amount of sucrose and proline.

The stage of donor tissue, however, seems to be a very important factor in obtaining somatic embryogenesis in vitro. We investigated the relationship between developmental stage of the embryo at the time of excision and the percentage of formation of organogenic or embryogenic tissue (Table 1).

Younger embryos, less than 0.9 mm in length, showed poor or no response in Medium B and Medium C. In Medium A, the percentage of such embryos forming organogenic callus was reduced. Older embryos, larger than 3.5 mm in length, showed no response and germinated in Medium B and Medium C. Attempts to induce formation of embryogenic tissue from mature excised embryos were done with several genotypes using different 2,4-D concentrations and with or without agar, but we have not obtained any positive results.

These results with floury-a, flint and *fl-a* x flint hybrid genotypes are consistent with the results

Table 1. For references see above. Between 10 and 15 embryos were cultured in each medium.

| Genotype | Embryo long mm | Medium A | Medium B | | Medium C | |
|---|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 2 | 3 |
| A. 84-3306 floury-a | 0.7 | 10 | 40 | 80 | 40 | 40 |
| | 1.0 | 20 | 60 | 100 | 40 | 100 |
| | 1.6 | 20 | 50 | 100 | 20 | 100 |
| | 2.0 | 15 | 30 | 50 | 0 | 0 |
| | 2.5 | 25 | 0 | 0 | 0 | 0 |
| | 3.0 | 20 | 0 | 0 | 0 | 0 |
| | 4.0 | 20 | 0 | 0 | 0 | 0 |
| | Mature | 10 | 0 | 0 | 0 | 0 |
| B, 84-3349 | 0.6 | 10 | 20 | 0 | 10 | 0 |
| | 0.9 | 30 | 100 | 100 | 80 | 100 |
| | 1.5 | 25 | 100 | 100 | 66 | 100 |
| | 2.1 | 30 | 90 | 100 | 60 | 100 |
| | 2.5 | 20 | 60 | 50 | 40 | 25 |
| | 3.0 | 20 | 10 | 0 | 15 | 0 |
| | 4.1 | 10 | 0 | 0 | 0 | 0 |
| | Mature | 10 | 0 | 0 | 0 | 0 |
| A x B | 0.5 | 40 | 20 | 0 | 10 | 0 |
| | 1.1 | 80 | 75 | 60 | 100 | 100 |
| | 1.7 | 75 | 75 | 50 | 90 | 80 |
| | 2.0 | 70 | 60 | 70 | 70 | 80 |
| | 2.6 | 60 | 50 | 40 | 40 | 30 |
| | 3.2 | 75 | 20 | 0 | 20 | 10 |
| | 4.2 | 40 | 0 | 0 | 0 | 0 |
| | Mature | 40 | 0 | 0 | 0 | 0 |

obtained by Lu et al. (TAG 66:285-289, 1983) working with dent genotypes. On the other hand, the induction of soft and friable (organogenic) callus in Medium A appears, to a great extent, independent of the developmental stage of the donor tissue.

Miguel Angel Rapela

## SDS-protein pattern of maize plants regenerated through somatic embryogenesis

In vitro plant regeneration in maize can be obtained either through organogenesis or by somatic (nonzygotic) embryogenesis. The phenotypic alterations (somaclonal variation) found in regenerated plants have a strong dependence on such morphogenetic events. Quite recently, Rapela (MNL 58:106-108, 1984) proposed a model experimental system in maize to obtain organogenesis or somatic embryogenesis working with local materials. The true nature of the morphogenetic events was tested using SEM examination (Rapela and Herkovits, Rev. Fac. Agr. UBA 5:95-105, 1984; MNL 58:108-109, 1984; Rapela, Bol. Asoc. Cien. Nat. Lit. 4:8-13, 1984).

We have obtained several plants regenerated through organogenesis or by somatic embryogenesis. Plants regenerated through organogenesis were aberrant, with several phenotypic alterations, and were very difficult to transfer to soil. Only one of 22 organogenic-regenerated plants reached maturation. On the other hand, plants regenerated through somatic embryogenesis were quite normal and relatively more easy to transfer to soil. The molecular analysis of the association of a particular phenotype with a particular polypeptide configuration was not carried out in organogenic-regenerated plants due to the fact that these plants were always in different

development stages. However, among our regenerated plants, 3 floury-a and 5 normal red flint (NRF) plants regenerated through somatic embryogenesis from one single callus each (originated from an immature embryo) and reached maturation almost at the same time (5 days of difference among plants). These plants are adequate for such analysis.

The terminal 10 cm of the upper leaf of such regenerated plants were removed and cut into small pieces. Polypeptides were extracted in two volumes of extraction buffer (200 mM Tris-HCl, pH 8, 5% SDS, 7.5% 2-mercaptoethanol and 20% glycerol), with homogenization in porcelain mortars at 0-5 C. The mixture was allowed to sit on ice for 10 minutes. The homogenization step was repeated and the homogenates were centrifuged at 12,360 g for 10 minutes. The supernatants were transferred to glass tubes, boiled for 5 minutes and stored frozen. Electrophoresis was carried out on 8.3% polyacrylamide gels prepared according to the method of Laemmli (1970). Gels were fixed for 24 hours in 3:1:1, methanol-acetic acid-water, stained in 0.2% Coomassie Brilliant Blue R-250 and destained in 7% acetic acid. Two independent preparations and two gels were run for each plant.

As shown in Figure 1, the SDS-protein pattern of floury-a and NRF maize plants regenerated through

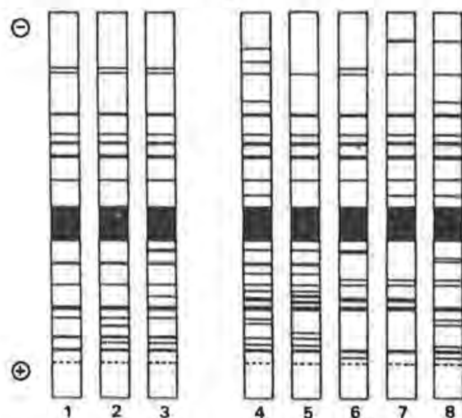



Fig. 1. One dimensional representation of SDS gel electrophoresis from leafs of regenerated plants. Channels 1, 2, and 3 from floury-a regenerated plants. Channels 4, 5, 6, 7, and 8 from Normal Red Flint regenerated plants.

somatic embryogenesis from one single-embryo has a particular configuration for each plant. There is a detectable variation between the SDS-polypeptides present in each plant extract and any counterparts.

An analysis based on presence or absence of polypeptides (without quantifying different staining intensities) shows a similarity index (SI = No. of pairs of similar bands / No. of different bands + No. of pairs of similar bands) of 1-2 = 0.88; 1-3 = 0.82; 2-3 = 0.83, for floury-a plants and, 4-5 = 0.59; 4-6 = 0.48; 4-7 = 0.48; 4-8 = 0.52; 5-6 = 0.48; 5-7 = 0.42; 5-8 = 0.36; 6-7 = 0.75; 6-8 = 0.55; 7-8 = 0.65, for NRF plants.

Cultures were initiated and the plants were regenerated and transplanted to vermiculite and then to soil at the same time. Therefore, although differences in the SDS-protein pattern due to environmental conditions and/or different developmental stages among plants prior to extraction cannot be ruled out, they are minimized. Eight of the 10 SI determined for NRF plants were lower than 0.6, indicating a strong qualitative difference between patterns. The percentage of SI variation due to somaclonal variation (genetic + epigenetic), although unknown under our experimental conditions, is possibly high. Moreover, the SI differences between floury-a (SI average = 0.84) and NRF (SI average = 0.53) plants seem to have a genotypic dependence, and support this last point of view.

Miguel Angel Rapela

**Lysine plus threonine inhibition in hybrid cell cultures: A possible case of allelic assortment**

We have reported that floury-a inbred embryos were insensitive to lysine plus threonine (LT) growth inhibition in vitro (Rapela, Rev. Fac. Agr. UNLP 56:17-26, 1980; Plant Cell Physiol. 23:285-291, 1982). Also, embryos of a particular normal red flint inbred line (BP) were partially insensitive to such inhibition (Rapela, Rev. Fac. Agr. UNLP 56:27-37, 1980; Phyton 41:63-66, 1981). Normal flint inbred embryos were sensitive to LT inhibition. We report here the behavior of cell cultures of these inbred lines and their hybrids to LT inhibition.

Cultures were initiated with immature (1-2 mm long) embryos in MS medium plus 2% sucrose and 1 ppm 2,4-D. After two subcultures in the dark the proliferated callus was divided into small pieces (about 50 mg each), and pieces were transferred separately to the MS medium with LT, 1 mM. Calli were screened for LT resistance after 45 days of culture in LT medium. Embryo cultures were initiated with mature embryos in MS medium with LT, 1 mM, and were screened after 14 days in culture (Table 1).

The behavior of the inbred genotypes was similar both in embryos and in cell cultures. If the embryos were insensitive, partially insensitive or sensitive to LT inhibition the cell cultures were also insensitive, partially insensitive or sensitive, respectively. However, the behavior of two hybrid genotypes was different between embryo and cell cultures. A x C and A x D cell cultures were partially insensitive to LT inhibition while embryo cultures were sensitive. Surprisingly, the genotype A x B was sensitive to the LT inhibition either in embryo or in cell cultures.

Up to now none of the hybrid, partially insensitive cell cultures regenerated plants. So, our observations are very difficult to explain. However P.S. Carlson (Cell Tiss. Cult. Tech. for Cereal Crop Imp. p. 407, 1983), has suggested that in the culture of

Table 1. Behavior of embryo and cell cultures in LT 1 mM. I= Insensitive (root length 60 % of control for embryo cultures, or 60 % healthy calli after 45 days for cell cultures). PI= Partially Insensitive (root length between 20-59 % of control for embryo cultures or 20-59 % healthy calli after 45 days for cell cultures). S= Sensitive (No roots or necrotic calli).

| Genotype | Embryo Cultures | Cell Cultures |
|---|---|---|
| A. 83-0322 (fl-a) | I | I |
| B. 83-0334 (BP) | PI | PI |
| C. 83-0344 (NH) | S | S |
| D. 83-052 (YF) | S | S |
| E. 83-0355 (DY) | S | S |
| A x B | S | S |
| A x C | S | PI |
| A x D | S | PI |
| A x E | S | S |
| B x C | S | S |
| B x D | S | S |
| B x E | S | S |
| C x D | S | S |
| C x E | S | S |
| D x E | S | S |

heterozygous clones (like our A x C and A x D genotypes), a number of distinct and stable subcultures appear with the expression of only one of the two alleles (in our case the floury-a). Such a process, termed allelic assortment, could explain our results. We are now investigating the molecular phenotypes of the hybrid LT resistant cell cultures via SDS-gel electrophoresis.

Miguel Angel Rapela

LOMAS DE ZAMORA, ARGENTINA
Lomas de Zamora University
LLAVALLOL, ARGENTINA
Instituto Fitotecnico de Santa Catalina

## Evolution of *Zea*

Phylogenetic relationships between maize and its close wild relatives (the teosintes) are a problem closely linked to the origin of maize, which has a century of controversy in its study. In spite of the quantity of hypotheses that have been suggested during this time, basically two schools defending opposite ideas in regard to maize origin were formed: 1) George Beadle's, that maize derives from teosinte, and 2) Paul Mangelsdorf's, that a wild maize is the ancestor of the cultivated one. The aim of this article is to present new proofs or new ways of setting in order the existing data.

*Morphological traits:* As laid down in the last taxonomic treatment (Doebley and Iltis, Amer. J. Bot. 67:1980), *Zea* has the following taxa: *Z. perennis* (perennial teosinte) (Zp), *Z. diploperennis* (diploperennial teosinte) (Zd), *Z. luxurians* (Guatemala teosinte) (Zl), *Z. mays* ssp. *parviglumis* var. *parviglumis* (Balsas teosinte) (Zmpp), *Z. mays* ssp. *parviglumis* var. *huehuetenangensis* (Huehuetenango teosinte) (Zmph), *Z. mays* ssp. *mexicana* (Nobogame-Chalco-Central Plateau teosinte) (Zmmx), and *Z. mays* ssp. *mays* (maize) (Zmm). These constitute the 7 operational taxonomic units (OTU's).

Ten morphological traits were scored for each OTU as follows: (1) tassel central spike distichous (0), polystichous (2); (2) Cupulate fruit case trapezoidal (2), triangular (1), horizontally compressed (0); (3) Kernel size small (0), intermediate (1), large (2); (4) Number of fruit cases per spike; (5) Female spike distichous (0), polystichous (1); (6) Female spikelet arrangement single (1), paired (2); (7) Growth habit perennial (1), annual (0); (8) Rhizomes present (1), absent (0); (9) Chromosome number 2n=40 (2), 2n=20 (1); (10) Chromosome knob position terminal (1), terminal and internal (2). Given a basic data matrix (BDM) (Table 1) of 10 characters by 7 OTU's,

Table 1: Basic data matrix (BDM). Morphological traits.

| | Characters | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| OTU's | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
| Zp | 0 | 2 | 0 | 5.1 | 0 | 1 | 1 | 1 | 2 | 1 |
| Zd | 0 | 2 | 0 | 7.5 | 0 | 1 | 1 | 1 | 1 | 1 |
| Zl | 0 | 2 | 0 | 6.4 | 0 | 1 | 0 | 0 | 1 | 1 |
| Zmpp | 0 | 1 | 0 | 8.8 | 0 | 1 | 0 | 0 | 1 | 2 |
| Zmph | 0 | 1 | 0 | 8.6 | 0 | 1 | 0 | 0 | 1 | 2 |
| Zmmx | 0 | 1 | 1 | 11.2 | 0 | 1 | 0 | 0 | 1 | 2 |
| Zmm | 1 | 0 | 2 | NC | 1 | 2 | 0 | 0 | 1 | 2 |

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 and arithmetic averages (UPGMA) (Figure 1), (2) the "Mean Taxonomic

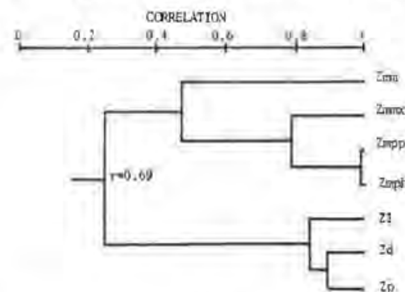



**Figure 1: Phenogram of 7 OTU's resulting from the UPGMA cluster analysis of the OTU x OTU correlation matrix. r= cophenetic correlation coefficient.**

Distance" between pairs of OTU's (Figure 2), and (3) the "Manhattan Distance" between pairs of OTU's to calculate a "Wagner Tree" (Figure 3). Methods (1) and (2) gave similar phenograms, coinciding with Doebley and Iltis' new taxonomy separating section LUXURIANTES (Zp, Zd, Zl) from ZEA (Zmm, Zmmx, Zmpp and Zmph); the cophenetic correlation coefficient of -0.82 indicates a lower distortion in method (2), where Zmm segregates further from the others. The tree derived by method (3) was constructed by selecting Zp as ancestor because, in our opinion, it has the largest quantity of morphological characters

**Figure 2: Phenogram of 7 OTU's resulting from the UPGMA cluster analysis of the OTU x OTU distance matrix. r= cophenetic correlation coefficient.**

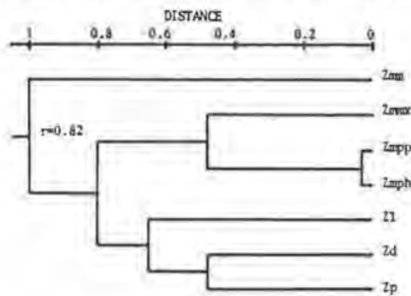
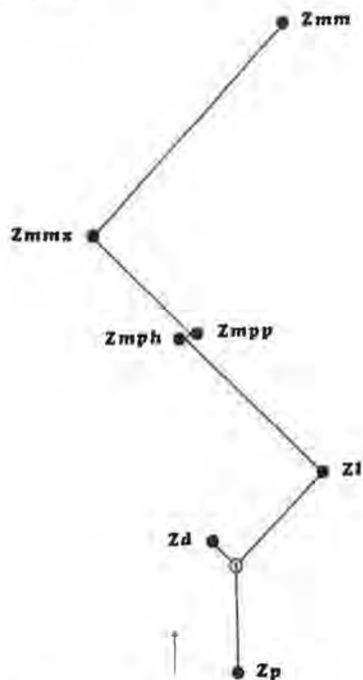



**Figure 3: Wagner Tree for the 7 OTU's based on 10 characters, considering *Z. perennis* as ancestor. Number corresponding to HTU's. Arrow indicates evolutionary direction.**




in a primitive state: it is perennial, has rhizomes, trapezoidal fruit cases and the lowest number of fruit cases per spike. It does not present, unlike *Z. diploperennis*, an exuberant development, perhaps because it is the only teosinte which has not been contaminated with maize germplasm. Zd and Zl are derived based on a hypothetical taxonomic unit ($HTU_1$) which, together with Zp, constitute the most primitive teosinte group placed at the bottom of the tree. Zmph, Zmpp and Zmmx, of which the first two are very close together, are clustered in the medium level of the tree. At the top of the tree, and very far from the other OTU's, is placed Zmm. Wagner's tree index of consistency is high (C = 0.91) and points out few extra evolutionary processes. The cluster of the taxa presented, based on few morphological characters and using different methods (cluster analysis and Wagner's tree), comes out as highly concordant.

In our opinion the key to mechanisms that have conditioned *Zea* evolution lies in maize (Zmm) and perennial teosinte, two basic species (and the most distant) that gather all necessary genetic information to explain the other taxa differentiation. The greatest part of individuals in hybrid progeny between perennial teosinte and maize are highly heterotic. In accordance with their morphology, they present phenotypes that move away not only from parental ones but also from those that might be expected as a consequence of their combination. These types of individuals may have distichous spikes with single spikelets, tenacious rachis and enclosed kernels. They may also have distichous spikes with badly enclosed kernels and fragile rachis, as well as any other possible combinations of these characteristics. There is no doubt that hybrids between Zp and Zmm individuals are almost completely inadequate to perpetuate in a natural environment. This failure to adjust prevents them from perpetuation without human aid. This is a consequence of the loss of wild characters, which has taken place with maize germplasm introgression. The maladjustment originates in the incapacity to spread their kernels freely, and because their female inflorescence structure (half-naked kernels not protected by husks) makes them vulnerable to insect attack, diseases, and birds and other animal injuries. Two principal facts demonstrate that maize introgression into primitive teosintes led to evolved teosinte differentiation: a) triangular-fruit-case individuals appear in hybrid progeny, resembling annual teosintes; b) maize germplasm introgression into teosinte with a low number of fruit cases per spike conditions an increase in their number (more-evolved annual teosintes also have this characteristic). Based on tassel characters (neutral characters), it can be stated that maize introgression into more primitive teosintes conditions the phenotype formation with characteristics of the most evolved group.

All things considered, it can be stated that there are enough morphological proofs to hold up the hypothesis that the different teosintes are products of maize germplasm introgression (followed by natural selection), first in perennial teosinte and secondly in the products derived from this first introgression. Consequently, if *Z. mays* ssp. *mexicana* is a lot like maize as a result of the maize germplasm accumulation, the cultured species could have never been derived from it. Maize should derive from other species (perhaps a primitive maize in accordance with Mangelsdorf) and, as can be demonstrated experimentally, maize has performed a fundamental role in teosinte evolution.

*Endosperm storage protein traits:* In previous reports (MNL 55:62, 1981; MNL 56:106, 1982; Magoja et al. Rev. Fac. Agron. 3:255, 1982) we stated that endosperm protein pattern obtained according to

Landry-Moureaux's technique could be considered as a specific characteristic that may be used to distinguish maize from its wild relatives. We inferred that maize and perennial teosinte protein patterns were basic and highly different, and intermediate for *Z. diploperennis* and *Z. mays* ssp. *mexicana* (according to the results of other authors), although up to 1982 protein patterns only were studied for *Z. perennis, Z. diploperennis* and several maizes. The hypothesis that intermediate protein patterns appearing in the other taxa of *Zea* might derive from that of *Z. perennis* (primitive protein pattern) by maize germplasm introgression was formulated. Now we present evidence that ratifies this hypothesis. A sufficient amount of annual teosinte seed was generously sent by Hugh H. Iltis in the beginning of 1984, allowing us to start biochemical analysis and to complete protein pattern study in each taxon of *Zea*. Five Landry-Moureaux endosperm storage protein traits were scored for each OTU as follows: (11) saline soluble proteins; (12) zein; (13) glutelin-1; (14) glutelin-2; (15) glutelin-3. Given a basic data matrix (Table 2), "Mean Taxonomic Distance" served as input in the calculation of a phenogram by the UPGMA (Figure 4), and "Manhattan Distance" in the calculation of a "Wagner Tree" (Figure 5).

Reasonable congruity exists between evolutionary trees based on morphological characteristics and on protein patterns: Zp is situated at the bottom of the tree, Zmm on the top, Zmmx looks like maize. The other taxa are located in medium positions between Zp and Zmmx.

**Table 2: Basic data matrix (BDM). Endosperm storage protein traits.**

| | Characters | | | | |
|---|---|---|---|---|---|
| OTU's | 11 | 12 | 13 | 14 | 15 |
| Zp | 1.8 | 57.1 | 20.7 | 3.3 | 9.2 |
| Zd | 3.1 | 67.1 | 9.2 | 7.5 | 10.0 |
| Zl | 3.3 | 70.5 | 7.9 | 2.3 | 9.9 |
| Zmpp | 2.8 | 71.5 | 7.1 | 1.9 | 9.3 |
| Zmph | 1.8 | 67.9 | 12.9 | 2.4 | 12.2 |
| Zmmx | 4.3 | 58.9 | 7.4 | 2.4 | 14.6 |
| Zmm | 5.7 | 51.9 | 12.3 | 9.6 | 13.0 |

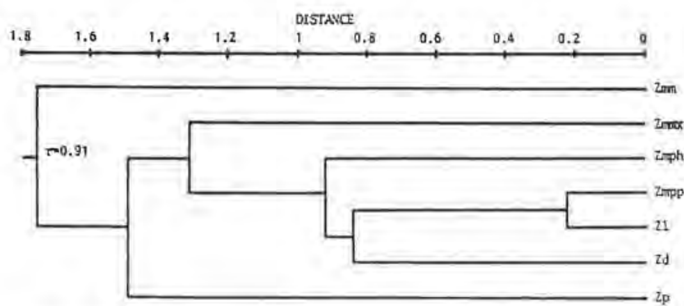



**Figure 4: Phenogram of 7 OTU's resulting from UPGMA cluster analysis of the OTU x OTU distance matrix. r = cophenetic correlation coefficient.**

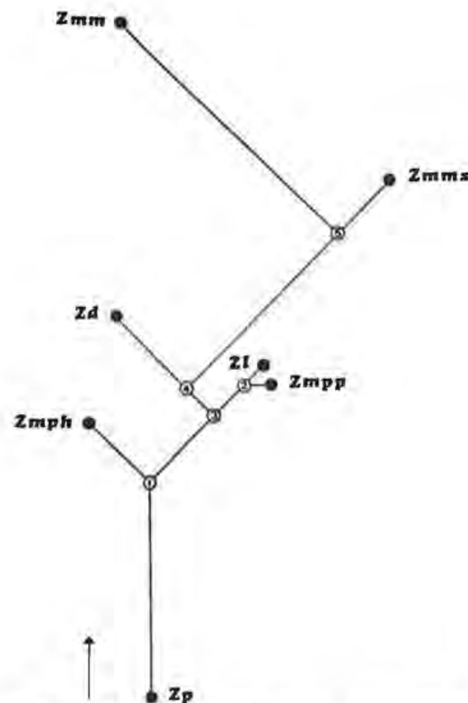



**Figure 5: Wagner Tree for 7 OTU's based on 5 characters, considering *Z. perennis* as ancestor. Number corresponding to HTU's. Arrow indicates the evolutionary direction.**

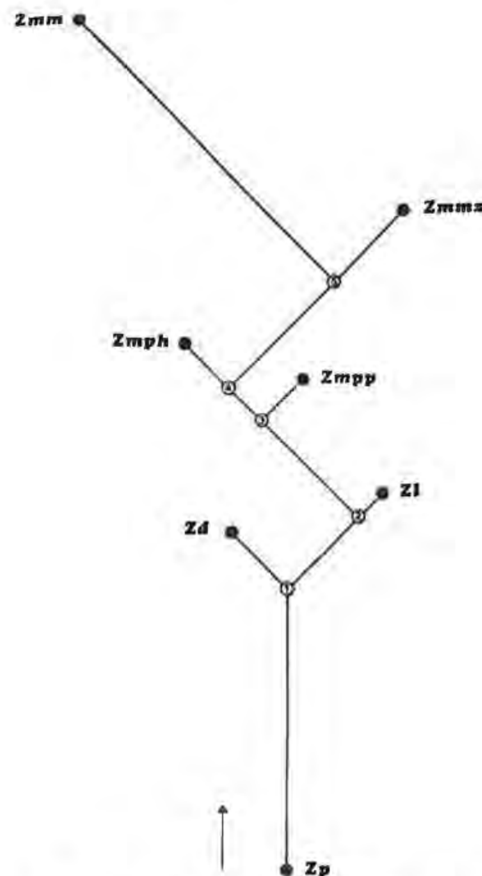



**Figure 6: Wagner Tree for 7 OTU's based on 15 characters, considering *Z. perennis* as ancestor. Number corresponding to HTU's. Arrow indicates the evolutionary direction.**

Finally, an evolutionary tree, such as represented in Fig. 6, may be built considering morphological and protein characteristics jointly. This tree has at the bottom Zd and Zl in addition to Zp (the ancestral species), forming the most primitive group of taxa (sect. LUXURIANTES, Doebley and Iltis, 1980). In the medium level appear Zmph, Zmpp and Zmmx (sect. ZEA), the first two taxa nearer to Zl than to Zmmx. Maize (Zmm) is placed far away from the rest of the taxa, at the top of the tree. The index of consistency of the tree is high (C = 0.77).

Based on the hypothesis that maize introgression into *Z. perennis* was the predominant factor in *Zea* evolution, it can be stated, in accordance with morphological and protein traits, that the evolutionary tree shown in Fig. 6 represents what might have occurred in nature: a former maize introgression into Zp led first to Zd (a species that retains perennialism) and then to Zl (annual) differentiation. Consecutive introgressions into Zl led to Zmpp and Zmph formation, which are (except Zl) the annual teosintes closest to the primitive group (Zl-Zd-Zp). Zmmx was produced by the latter maize introgression, and is the most evolved teosinte, because it resembles maize, both in morphological and in protein characters. In short, Zmmx is a maize that retains from teosinte only the essential genes for wild life, turning out to be, consequently, the teosinte with the largest quantity of maize germplasm.

*Fruit traits:* Four quantitative characteristics of grain were evaluated: pericarp thickness (PT), aleurone layer thickness (AT), starch granule size (SGS) and zein or protein body size (PBS) in three endosperm zones (See MNL 58:129). PT and AT were measured in the abgerminal side of the kernel, SGS measurement was made in the 10th cell layer counting from the aleurone layer downwards inside the grain. All the measurements were made upon three cuts of each kernel, using ten grains taken at random from each species.

The results corresponding to maize are the average of measurements obtained over several types whose PT and AT are different (Table 3). PT is lower in teosinte than in maize. Zmmx has the thickest pericarp of all teosintes. AT is lower in teosinte than in maize, although in maize the aleurone layer thickness is quite variable. SGS seems to have no specific relationship, and no significant differences were found among the diverse taxa. The same can be pointed out for protein body size, which is quite similar in the different taxa of *Zea*.

The quantitative characteristics were assessed for correlations, as well as other characteristics of these fruits, such as kernel presentation (KP), cupulate fruit case shape (CFCS), grain size (GS), endosperm protein content (EP), saline soluble protein content (SS) and zein content (Z) (Table 4). Some fruit characteristics are significantly associated: grains enclosed in fruit cases are small or intermediate, have a low rate of SS, and their pericarps are thin. Trapezoidal-fruit-case teosintes have the smallest grains. They have higher endosperm protein content, low SS content, high Z content and thinner pericarp. Those taxa with higher endosperm protein content have more zein and thinner pericarp. Those taxa with higher SS content have thicker pericarp. Zein content is higher in those taxa with a thinner aleurone layer and zein bodies are smaller in zone 3. Zein body sizes in zones 2 and 3 are positively associated. From all the characteristics

**Table 3: Quantitative fruit traits.**

| | | PT (um) | AT (um) | SGS (um) | PBS (um) 1Z | 2Z | 3Z |
|---|---|---|---|---|---|---|---|
| Z.perennis | $\bar{X}$ ± SD | 17.0 ± 1.8 | 42.3 ± 4.0 | 17.3 ± 2.9 | 1.7 ± 0.3 | 1.6 ± 0.4 | 1.4 ± 0.4 |
| | range | 16.0 -20.0 | 36.0 -48.0 | 14.4 -24.0 | 0.8 - 2.5 | 0.7 - 2.9 | 0.7 - 2.0 |
| Z.diploperennis | $\bar{X}$ + SD | 21.0 ± 3.9 | 39.7 ± 2.7 | 12.5 ± 1.0 | 1.5 ± 0.4 | 1.2 ± 0.4 | 1.0 ± 0.3 |
| | range | 16.0 -28.0 | 36.0 -44.0 | 11.2 -14.4 | 0.8 - 2.6 | 0.4 - 2.7 | 0.4 - 1.9 |
| Z. luxurians | $\bar{X}$ + SD | 13.6 ± 1.7 | 31.8 ± 3.4 | 17.6 ± 1.3 | 1.7 ± 0.4 | 1.5 ± 0.3 | 1.1 ± 0.2 |
| | range | 12.0 -16.0 | 26.0 -36.0 | 16.0 -19.2 | 1.0 - 2.7 | 0.8 - 2.3 | 0.6 - 1.7 |
| Z. mayss ssp. parviglumis var. parviglumis. | $\bar{X}$ + SD | 12.8 ± 2.2 | 28.3 ± 3.2 | 17.1 ± 1.9 | 1.7 ± 0.4 | 1.2 ± 0.3 | 0.9 ± 0.2 |
| | range | 10.0 -16.0 | 24.0 -32.0 | 14.4 -19.2 | 1.0 - 2.5 | 0.6 - 1.9 | 0.5 - 1.7 |
| Z. mays ssp. parviglumis var. huehuetenangensis | $\bar{X}$ + SD | 13.8 ± 2.1 | 29.5 ± 5.7 | 17.0 ± 1.5 | 1.3 ± 0.3 | 1.0 ± 0.2 | 0.9 ± 0.2 |
| | range | 12.0 -16.0 | 24.0 -40.0 | 14.4 -19.2 | 0.8 - 2.5 | 0.6 - 1.9 | 0.5 - 1.4 |
| Z. mays ssp. mexicana | $\bar{X}$ + SD | 23.7 ± 3.6 | 42.0 ± 4.3 | 14.9 ± 1.3 | 1.4 ± 0.3 | 1.4 ± 0.4 | 1.0 ± 0.3 |
| | range | 16.0 -28.0 | 36.0 -48.0 | 12.8 -16.0 | 0.8 - 2.0 | 0.6 - 3.2 | 0.5 - 1.9 |
| Z. mays ssp. mays | $\bar{X}$ + SD | 95.8 ±44.0 | 45.9 ±10.4 | 16.0 ± 2.2 | 1.8 ± 0.3 | 1.5 ± 0.2 | 1.4 ± 0.2 |
| | range | 60.0-168.0 | 28.0 -60.0 | 9.6 -19.2 | 0.6 - 3.9 | 0.5 - 2.5 | 0.3 - 3.0 |

**Table 4: Correlation between fruit traits.**

| | KP | CFCS | GS | EP | SS | Z | PT | AT | SGS | ZBS-1 | ZBS-2 | ZBS-3 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| KP | — | 0.75 | -0.88** | 0.81 | -0.78* | 0.68NS | -0.99** | -0.55NS | 0.01NS | -0.51NS | -0.32NS | -0.61NS |
| CFCS | | — | -0.80* | -0.69NS | -0.64NS | 0.46NS | -0.74NS | -0.20NS | -0.08NS | -0.08NS | 0.12NS | -0.10NS |
| GS | | | — | -0.93** | 0.91** | -0.78* | 0.92** | 0.68NS | -0.14NS | 0.28NS | 0.37NS | 0.49NS |
| EP | | | | — | 0.74NS | 0.89** | 0.84* | -0.71NS | -0.08NS | -0.28NS | -0.53NS | -0.67NS |
| SS | | | | | — | -0.54NS | 0.82* | 0.57NS | -0.29NS | 0.35NS | 0.37NS | 0.33NS |
| Z | | | | | | — | -0.74NS | -0.91** | 0.10NS | -0.26NS | -0.60NS | -0.80* |
| PT | | | | | | | — | 0.64NS | -0.12NS | 0.46NS | 0.34NS | 0.62NS |
| AT | | | | | | | | — | -0.43NS | 0.24NS | 0.62NS | 0.74NS |
| SGS | | | | | | | | | — | 0.33NS | 0.22NS | 0.18NS |
| ZBS-1 | | | | | | | | | | — | 0.68NS | 0.66NS |
| ZBS-2 | | | | | | | | | | | — | 0.83* |
| ZBS-3 | | | | | | | | | | | | — |

evaluated only pericarp thickness and aleurone layer may be considered specific.

In general, specific characteristics are significantly associated between themselves and with other specific characteristics considered before, such as cupulate fruit case shape, kernel size, saline soluble protein content and zein content. Only endosperm protein content, pericarp thickness and aleurone layer may be considered specific among all the new characters presented in this work. The other ones are very variable in each taxon, and no significant differences are manifested.

Cluster analyses were performed on fruit data from 10 characters (3 qualitative and 7 quantitative) of taxa of *Zea*, as follows: (2) Cupulate fruit case shape trapezoidal (2), triangular (1), horizontally compressed (0); (3) Kernel size small (0), intermediate (1), large (2); (16) Kernels enclosed (1), naked (0); (17) Kernel protein content (%); (18) Pericarp thickness (μm); (19) Aleurone layer thickness (μm); (20) Starch granule size (μm); (21) Zein body size of zone 1 (μm); (22) Zein body size of zone 2 (μm); (23) Zein body size of zone 3 (μm). Given a basic data matrix (BDM) (Table 5), the Pearson product-moment correlation coefficient served as input in the calculation of a phenogram by UPGMA (Figure 7), and "Mean Taxonomic Distance" between pairs of OTU's was calculated (Figure 8). In both phenograms Zmm links at the farthest distance, which means it is the most differentiated species in regard to fruit characteristics. There is no high congruency in clustering based only on fruit characteristics, as observed in those based on other characteristics. This may occur because of having included some non-specific fruit characteristics (which have been pointed out before) with the same weight as the specific ones according to the exigencies of the numerical method chosen.

**Table 5: Basic data matrix (BDM) Fruit traits.**

| | Characters | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| OTU's | 2 | 3 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 |
| Zp | 2 | 0 | 1 | 21.0 | 17.0 | 42.3 | 17.3 | 1.7 | 1.6 | 1.4 |
| Zd | 2 | 0 | 1 | 27.0 | 21.0 | 39.7 | 12.5 | 1.5 | 1.2 | 1.0 |
| Zl | 2 | 0 | 1 | 23.6 | 13.6 | 31.8 | 17.6 | 1.7 | 1.5 | 1.1 |
| Zmpp | 1 | 0 | 1 | 26.5 | 12.8 | 28.3 | 17.1 | 1.7 | 1.2 | 0.9 |
| Zmph | 1 | 0 | 1 | 23.4 | 13.8 | 29.5 | 17.0 | 1.3 | 1.0 | 0.9 |
| Zmmx | 1 | 1 | 1 | 17.7 | 23.7 | 42.0 | 14.9 | 1.4 | 1.4 | 1.0 |
| Zmm | 0 | 2 | 0 | 11.4 | 95.8 | 45.9 | 16.0 | 1.8 | 1.5 | 1.4 |

In accordance with the results obtained, it can be determined that fruit characteristics can not be used separately to assemble the taxa of *Zea*, and that from the characteristics studied, protein content, pericarp thickness and aleurone layer are the most specific ones with evolutionary significance.

*Analysis of dispensable traits:* In addition to the characteristics employed in previous articles, we also used "neutral characters" or tassel traits according to Doebley and Iltis (1980). Ten neutral morphological traits were scored for each OTU as follows: (24) Tassel branch number low (1), high (2); (25) Tassel branching axis short (1), long (2); (26) Lateral tassel branch internode short (1), long (2); (27) Male spikelet outer glume wing absent (0), present (1); (28) Male spikelet outer glume primary lateral vein narrow and scarcely (0), wide and prominent (1); (29) Male spikelet outer glume total veins few (0), numerous (1); (30) Tassel branching abscission layer absent (0), strongly to weakly developed (1), strongly

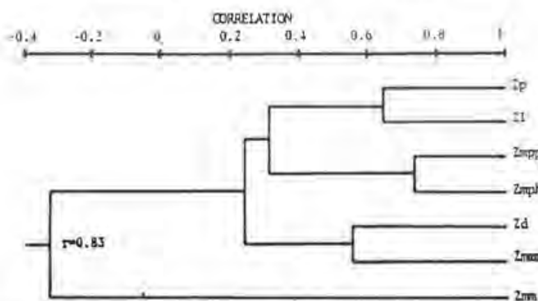



**Figure 7: Phenogram of 7 OTU's resulting from the UPGMA cluster analysis of the OTU x OTU correlation matrix. r = cophenetic correlation coefficient.**

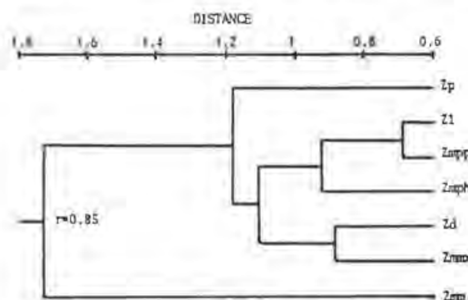



**Figure 8: Phenogram of 7 OTU's resulting from the UPGMA cluster analysis of the OTU x OTU distance matrix. r = cophenetic correlation coefficient.**

**Table 6: Basic data matrix (BDM)**

| OTU's | Characters 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|---|
| Zp | 0 | 2 | 0 | 5.1 | 0 | 1 | 1 | 1 | 2 | 1 |
| Zd | 0 | 2 | 0 | 7.5 | 0 | 1 | 1 | 1 | 1 | 1 |
| Zl | 0 | 2 | 0 | 6.4 | 0 | 1 | 0 | 0 | 1 | 1 |
| Zmpp | 0 | 1 | 0 | 8.8 | 0 | 1 | 0 | 0 | 1 | 2 |
| Zmph | 0 | 1 | 0 | 8.6 | 0 | 1 | 0 | 0 | 1 | 2 |
| Zmmx | 0 | 1 | 1 | 11.2 | 0 | 1 | 0 | 0 | 1 | 2 |
| Zmm | 1 | 0 | 2 | NC | 1 | 2 | 0 | 0 | 1 | 2 |
| **OTU's** | **11** | **12** | **13** | **14** | **15** | **17** | **18** | **19** | **24** | **25** |
| Zp | 1.8 | 57.1 | 20.7 | 3.3 | 9.2 | 21.0 | 17.0 | 42.3 | 1 | 1 |
| Zd | 3.1 | 67.1 | 9.2 | 7.5 | 10.0 | 27.0 | 21.0 | 39.7 | 1 | 1 |
| Zl | 3.3 | 70.5 | 7.9 | 2.3 | 9.9 | 23.6 | 13.6 | 31.8 | 1 | 1 |
| Zmpp | 2.8 | 71.5 | 7.1 | 1.9 | 9.3 | 26.5 | 12.8 | 28.3 | 2 | 2 |
| Zmph | 1.8 | 67.9 | 12.9 | 2.4 | 12.2 | 23.4 | 13.8 | 29.5 | 2 | 2 |
| Zmmx | 4.3 | 58.9 | 7.4 | 2.4 | 14.6 | 17.7 | 23.7 | 42.0 | 2 | 2 |
| Zmm | 5.7 | 51.9 | 12.3 | 9.6 | 13.0 | 11.4 | 95.8 | 45.9 | 2 | 2 |
| **OTU's** | **26** | **27** | **28** | **29** | **30** | **31** | **32** | **33** | | |
| Zp | 1 | 1 | 1 | 1 | 2 | 0 | 1 | 0 | | |
| Zd | 1 | 1 | 1 | 1 | 2 | 0 | 1 | 0 | | |
| Zl | 1 | 1 | 1 | 1 | 2 | 0 | 1 | 0 | | |
| Zmpp | 2 | 0 | 0 | 0 | 1 | 1 | 2 | 1 | | |
| Zmph | 2 | 0 | 0 | 0 | 1 | 1 | 2 | 1 | | |
| Zmmx | 2 | 0 | 0 | 0 | 1 | 1 | 2 | 1 | | |
| Zmm | 2 | 0 | 0 | 0 | 0 | 1 | 2 | 1 | | |

developed (2); (31) Tassel branching rachis flattened (0), rounded (1); (32) Male spikelet outer glume stiff (1), papery (2); (33) Male spikelet outer glume flattened (0), rounded (1). Given a basic data matrix (BDM) (Table 6) of 28 characters by 7 OTU's, the data were analyzed by cluster analysis. The BDM was standardized (BDMS) and data were analyzed by "Mean Taxonomic Distance" (Figure 9), by "Manhattan Distance" (Figure 10) and a "Prim Network" (Figure 10A), and by "Manhattan Distance" and "Wagner Trees" (Figure 10B, C).

Figure 9 shows a phenogram with 2 definite clusters: one of them constituted by the most primitive teosintes (Zp-Zd-Zl) and the other by the most evolved ones (Zmpp-Zmph-Zmmx). Maize (Zmm) segregates separately from the rest of the taxa, and is located as distant from one group as from the other. The Prim Network given in Figure 10A shows that Zp and Zmm are the two taxa situated at the farthest distance from each other, whereas the most primitive teosintes link nearer to Zp than the most

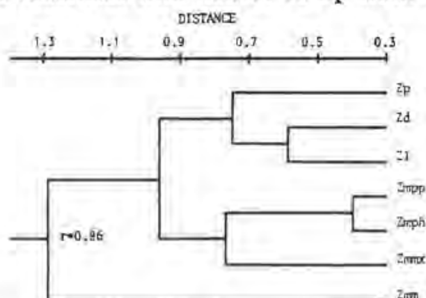



**Figure 9: Phenogram of 7 OTU's resulting from the UPGMA cluster analysis of the OTU x OTU distance matrix. r = cophenetic correlation coefficient.**

evolved ones. The latter are located between Zl and Zmm. Wagner Trees shown in Figure 10B and C are similar. High congruity exists whether 18 traits (1 to 10 orthodox morphological traits + 11 to 15 endosperm protein traits +17-18-19 fruit traits; see previous reports), or 28 traits including "neutral morphological characters", are considered. The taxa ordering is similar in both trees and coincidental with the Prim Network. Results obtained point out that evolutionary trees constructed with either 18 or 28 characters have a high index of consistency (C=0.78 and C=0.82, respectively), denoting a few extra evolutionary processes. Both phenograms (Fig. 9) and evolutionary trees (Fig. 10) clearly demonstrate an extremely particular and well defined clustering: teosinte grouped in 2 levels (one primitive and the other evolved), and maize separated from them.

Results obtained in cluster analysis strongly uphold that taxa of *Zea* are the product of maize introgression, first in the most primitive teosinte (Zp) and secondly into the derived taxa. Introgression degree was increased at the same time as the tree is climbed, until it reaches Zmmx, the teosinte with the maximum maize introgression. If Zmmx is the teosinte with the highest maize introgression, as can be supported by morphological, biochemical and cytological evidence, it could never have been the maize ancestor.

When this series of reports about "Evolution of Zea" was initiated in this MNL, it was stated that our purpose was that new data or new ways of ordering the existing data would be provided, in order to clarify phylogenetic relationships among

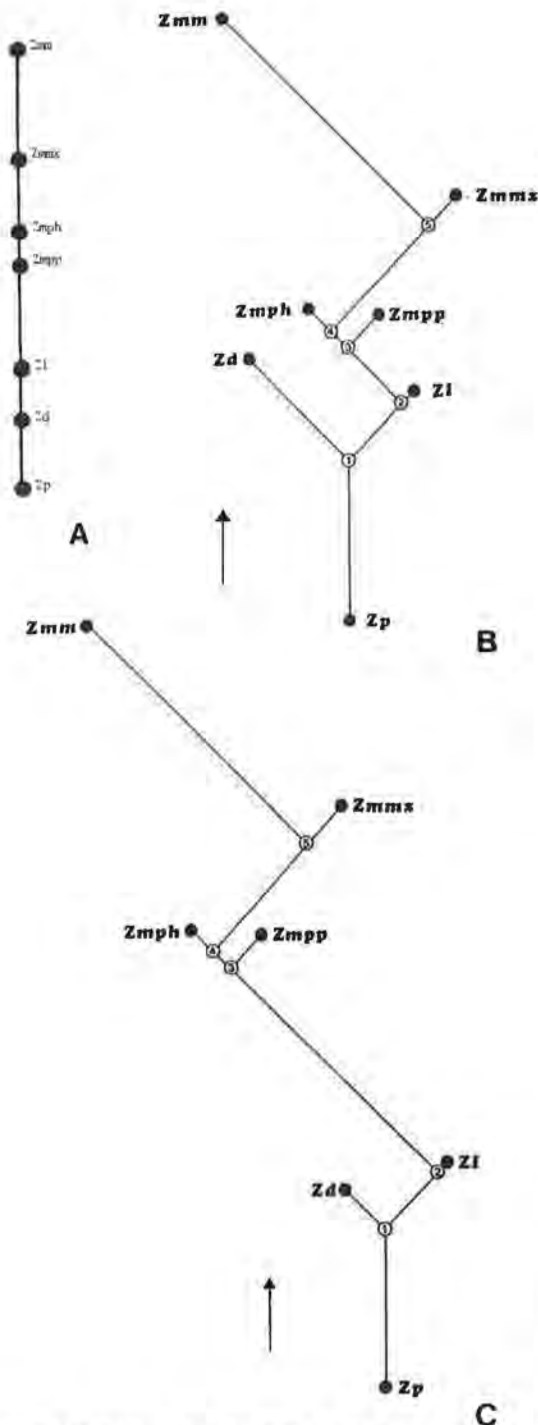



**Figure 10: Prim Network and evolutionary trees for the genus *Zea*. A—Prim Network for the 7 OTU's based on 28 characters. B—Wagner Tree for the 7 OTU's based on 18 characters, considering *Z. perennis* as ancestor. C—The same based on 28 characters. Number corresponding to HTU's represents the construction sequence of the tree. Arrows indicate the evolutionary direction.**

the taxa of *Zea*, reinforcing an old hypothesis. Now, we want to explain that these new data and the new way of ordering them come from our own research, and this old hypothesis to which we refer is Paul Mangelsdorf's.

J. L. Magoja, I. G. Palacios,
L. M. Bertoia and M. E. Streitenberger

## Puna maize: selection for high protein quality and hard endosperm

In previous reports (MNL 57:73, 1983) it was communicated that a Puna maize germplasm has been the starting point of selection for high protein quality not associated to soft endosperm. The aim of this report is to communicate the results through two generations of selection for high protein quality and hard endosperm. The objective of this selection is to obtain maize inbred lines in which these characters are combined.

Characteristics of 31 $S_2$ lines were studied.The results obtained are shown in Table 1. The selection was based on the tryptophan content of endosperm protein. Hardness (measured on an arbitrary scale: 0 = floury to 4 = flint) and kernel apparent density were measured in $S_2$ lines. Protein content and protein quality are inversely associated, whereas kernel hardness and kernel density show a negative correlation with tryptophan content (Table 2). There are lines that combine a good ratio of hard endosperm with high protein quality (Table 3). $S_2$ lines have, on average, 50% more tryptophan than normal maizes, and 20% more than $S_1$ lines (Table 4).

Table 1: Characteristics of Puna $S_2$ lines.

| | Kernel Weight (mg) | Hardness | Density (g/ml) | defatted endosperm Protein (%) | defatted endosperm Tryptophan (g/100 g prot.) |
|---|---|---|---|---|---|
| Mean | 256.8 | 2.5 | 1.21 | 12.0 | 0.6 |
| SD | 60.4 | 1.3 | 0.09 | 1.4 | 0.1 |
| Range | 161.5-376.1 | 0-4 | 1.07-1.45 | 8.4-13.9 | 0.4-0.8 |

Table 2: Correlation coefficients between kernel weight (KW), hardness (H) density (D), endosperm protein content (EP) and endosperm tryptophan content (ET) in Puna $S_2$ lines. *-- significant at 5 % level; **-- significant at 1 % level.

| | KW | H | D | EP | ET |
|---|---|---|---|---|---|
| KW | — | | | | |
| H | -0.55** | — | | | |
| D | -0.19 | 0.49* | — | | |
| EP | 0.22 | 0.20 | 0.14 | — | |
| ET | 0.15 | -0.37* | -0.39* | -0.52** | — |

Table 3: Characteristics of the best Puna $S_2$ lines

| $S_2$ line | Kernel Weight (mg) | Hardness | Density (g/ml) | Defatted endosperm protein (%) | Defatted endosperm tryptophan (g/100 g.prot.) |
|---|---|---|---|---|---|
| $D_4$ | 316.4 | 3 | 1.26 | 12.3 | 0.7 |
| $D_9$ | 189.3 | 3 | 1.16 | 8.4 | 0.7 |
| $D_{27}$ | 261.2 | 4 | 1.10 | 11.1 | 0.6 |
| $D_{32}$ | 265.6 | 3 | 1.12 | 11.1 | 0.7 |
| Average | 258.1 | 3.3 | 1.16 | 10.7 | 0.7 |

Table 4: Differences between $S_1$ and $S_2$ lines.
**-- significant at 1 % level.

| | $S_1$ | $S_2$ | $(S_2-S_1)$ |
|---|---|---|---|
| Kernel weight (mg) | 231.1 | 256.8 | +25.7 |
| Protein (%) | 13.3 | 12.0 | -1.3** |
| Tryptophan (g/100 g prot.) | 0.5 | 0.6 | +0.1** |

Protein quality selection has been positive, and the quick progress obtained in tryptophan content improvement may foretell the obtaining, in a few generations of selection, of lines whose protein quality is similar to that conditioned by known mutants. The best $S_2$ lines obtained have 75% more tryptophan than normal maizes, and present good endosperm hardness. According to the results obtained, it can be inferred that Puna maizes constitute a germplasm source which may be used to improve endosperm protein quality without modifying its normal phenotype (hard).

Ana María Bróccoli, Mirta E. Streitenberger and Jorge Luis Magoja

## Differences between Gaspé x *Z. perennis* and Gaspé x *Z. diploperennis* $F_1$ hybrids

Perennial teosinte *(Z. perennis)* and diploperennial teosinte *(Z. diploperennis)* are the most primitive taxa of *Zea*. Both species have primitive characters because they are perennial, have rhizomes, trapezoidal fruit cases and low number of fruit cases per spike. There is widespread confusion about which of the two species is the ancestral one, although according to different points of view, both species may be considered the most primitive.

$F_1$ hybrids between Gaspé and each teosinte species were comparatively analyzed in order to clarify this topic. Data resulting from the observation of some characteristics of Gs x Zp and Gs x Zd hybrids are given in Table 1. Due to the fact that the

**Table 1: Comparison of some characters between Gaspé x *Z. perennis* and Gaspé x *Z. diploperennis* $F_1$ hybrids.**

| Character | Gs x Zp | Gs x Zd |
|---|---|---|
| Days to tassel | 61.6 ± 7.2 | 38.7 ± 3.5 |
| Type of dichogamy | protoginous | protoginous |
| Fertility | low | high |
| Tillers | numerous | few |
| Tassel central spike | distichous | distichous |
| Female spike | distichous | distichous |
| Female spikelets arrangement | single | paired |
| Kernels | enclosed | ± naked |
| Growth habit | perennial | annual |
| Rhizomes | present | absent |
| Heterosis | high | low |
| Prolificity | high | intermediate |
| $F_1$ kernel viability | very low | high |

same maize was used as female parent, results are highly comparable. Thus, differences in character expression may be attributed to the diverse action of the wild species' germplasm. $F_1$ grain viability is very different: Gs x Zp are not very viable, whereas Gs x Zd develop normally. Germplasm behaviour exhibited by both species is similar in some specific characters, such as tassel central spike and female spike, whereas it is quite different in others, such as female spikelet arrangement, growth habits and rhizomes. Another group of characters, fertility, number of tillers, heterosis and prolificacy, may be considered as an index of the distance between maize and the two wild species. Shorter distance is displayed by high fertility, low number of tillers, low heterosis and less prolificacy of hybrids. In accordance with this, it may be stated that a shorter distance exists between *Z. diploperennis* and maize than between *Z. perennis* and maize.

One of the most significant aspects to consider lies in the hybrids' different heterosis linked to an evident difference in prolificacy. Consequently, it may be said that *Z. perennis* is the most primitive taxon of *Zea* and, thus, *Z. diploperennis*' ancestral species. *Z. diploperennis* is almost identical to *Z. perennis* in specific characteristics. *Z. diploperennis* stands out because of its more exuberant development and greater vigour. In our opinion, this happens on account of being derived from maize introgression into *Z. perennis*. Morphological and biochemical proofs as well as comparable hybrids' different behaviour suggest that *Z. diploperennis* could never have been the ancestral species of *Z. perennis* but maize introgression into *Z. perennis* was the predominant factor in the differentiation of these species.

Jorge Luis Magoja

## Perennial teosinte-Gaspé hybrids: preliminary results on inheritance of pericarp and aleurone layer thickness

Teosintes have pericarps and aleurone layers that are thinner than maize. The pericarp especially is thinner, since maize has a considerable variation in aleurone layer thickness. In order to study the inheritance of these characters in hybrids between perennial teosinte and maize (Gaspé), we analyzed the results obtained which, up to now, come from measurements made on Gaspé, *Zea perennis* and $F_2$ kernels from reciprocal crosses between both parents.

Kernels were prepared for measurement according to Tracy et al. (MNL 52:60, 1978). Pericarp and aleurone layer thickness were evaluated on 20 Gaspé and perennial teosinte kernels, making 5 measurements on each. These traits were also evaluated on 100 $F_2$ reciprocal grains (50 of Gs x Zp and 50 of Zp x Gs). Results obtained are shown in Table 1. Perennial teosinte has less than one fourth the pericarp thickness of Gaspé. The $F_1$ reciprocal pericarp thickness is significantly different, probably pointing out

Table 1: Pericarp thickness (PT) and aleurone layer thickness (AT) in perennial teosinte (Zp), Gaspe (Gs) and their reciprocal $F_1$ and $F_2$ (thickness in microns)

| | PT | AT |
|---|---|---|
| Zp | 17.0 ± 1.8 | 42.3 ± 4.0 |
| Gs | 76.0 ± 7.2 | 34.3 ± 4.7 |
| Zp x Gs | 33.8 ± 9.1 | — |
| Gs x Zp | 30.0 ± 8.0 | — |
| Zp x Gs $F_2$ | — | 54.1 ±16.2 |
| Gs x Zp $F_2$ | — | 52.4 ±10.0 |

some cytoplasmic effect on character expression. Thin pericarp (perennial teosinte) is partly dominant over thick pericarp (Gaspé). $F_1$ pericarp thickness is nearer to the parental than to perennial teosinte, thus it may be inferred that the wild character (thin pericarp) does not behave as a dominant.

It is important to remark that in both reciprocal $F_1$'s the pericarp thickness is a highly variable character (range: 15 to 70 microns). This points out that: 1) gametes with different information for pericarp thickness are brought in by perennial teosinte, and 2) pericarp thickness does not have simple inheritance but the action of genes with additive effect prevails. It is also appropriate to point out that thin pericarp is not associated with enclosed kernels, as it can be observed that every $F_2$ grain is enclosed in a fruit case like *Z. perennis*, but has pericarp thickness similar to maize. In short, the enclosed-naked kernel character segregates independently from thick-thin pericarp character.

From the analysis of aleurone layer data, it must be especially remarked that in this particular case, perennial teosinte's aleurone layer is thicker than maize's (Gaspé). No significant difference was found on means in reciprocal $F_2$, according to crossing direction. That is to say, no cytoplasmic effect is detected. The mean thickness of aleurone in $F_2$ kernels is superior not only to that of the parent but also to the thicker parent *(Z. perennis)*. This may occur because in hybrids between perennial teosinte and Gaspé a particular phenomenon takes place: high frequency production of kernels with multilayer aleurone. The frequency distribution of aleurone layer thickness in $F_2$ kernels is transgressive in the positive direction and varies from 36 to 143 microns. Preliminary results allow distinguishing not only between maize and its wild relatives, but also between different teosintes, and seem to point out that they are the consequence of quantitative inheritance and not associated to the enclosed-naked kernel characteristic.

Luis M. Bertoia and Jorge L. Magoja

**Perennial teosinte-Gaspé hybrids: multilayer aleurone**

An unexpected fact was observed during the study of pericarp and aleurone layer thickness in *Z. perennis* x Gaspé hybrids: $F_2$ grains with multilayer aleurone. This result is completely unusual, mainly because none of the parents present this characteristic. In the same way, our attention was attracted to the high frequency in which multilayer aleurone kernels come out. Aleurone with a 2 to 6-cell layer appears with the same frequency in $F_2$ kernels in both directions of crossing between Gaspé and perennial teosinte (see Fig. 1). Thirty two of 100 kernels studied presented multilayer aleurone; that is to say, a third of $F_2$ grains have this character.

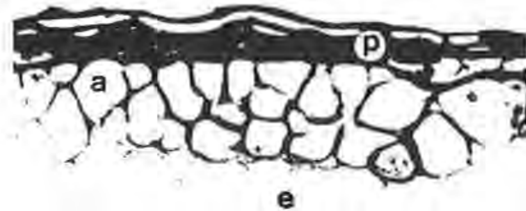



**Figure 1: Photomicrograph of free hand section from the abgerminal side of the kernel showing multilayer aleurone. 290x. a— aleurone; e— endosperm; p— pericarp.**

Aleurone layer thickness is not strictly associated with the number of cells in the layer, but multilayer kernels have significantly thicker aleurone (average 75.7 microns) than those with a single layer (average 42.6 microns). Aleurone layer thickness in those multilayer kernels is not uniform, since there are zones with different numbers of layers. Even within the same kernel, single-aleurone zones are adjoined to multilayer ones. Nevertheless, exceptional kernels can be observed with a homogeneously distributed multilayer in all the grain periphery. Sometimes its thickness reaches 140 microns.

At first, we thought that the multilayer aleurone character was associated with floury endosperm, as occurs in Coroico maize. However, after checking the kernels once more, we could notice that this trait appears indifferently not only in floury but in hard endosperm kernels. Up to now it could not be determined if there is any association to other kernel traits, nor the effect produced on endosperm protein quality and quantity. The reason for its spontaneous appearance is still unknown, but in view of the high frequency in which this character comes out in hybrids between perennial teosinte and Gaspé progeny, it is probable for the multilayer aleurone trait to be the consequence of a single mutation.

Luis M. Bertoia and Jorge L. Magoja

**Perennial teosinte-Gaspé hybrids: specific relationship of endosperm protein content**

Kernel protein content in wild relatives of maize is higher than in the cultivated species. In most cases it is twice the normal content of maize. Endosperm protein content (EPC) is a specific character of *Zea* taxa (see "Fruit traits" in a previous report in this MNL), apparently associated with other fruit morphological characteristics. The aim of this research is to investigate if EPC is associated with other morphological traits in plants derived from interspecific crossing. Hybrids between *Z. perennis* and Gaspé are a suitable material for this study: EPC is 24.1% in *Z. perennis* whereas in Gaspé it is 14.4%.

In general, individuals of $F_2$ and $F_3$ progeny resulting from crosses between *Z. perennis* and Gaspé have a high EPC, approximating that of *Z. perennis*.

Thirty four plants that stood out for their definite phenotype: 1) teosintoid, 2) intermediate and 3) maizoid were taken from $F_2$ and $F_3$ populations to study EPC association to other characters. Teosintoid phenotype belongs to those plants that combine mainly perennial teosinte traits: perennial, distichous spike, single spikelets, distichous tassel central spike, high number of tillers, enclosed kernels and narrow leaves. Maizoid phenotype belongs to those plants that combine mainly maize characteristics: annuals, polystichous ears, paired spikelets, polystichous tassel central spike, few tillers, naked kernels and wide leaves. Intermediate phenotype belongs to those plants in which the furthest phenotypic traits are combined. Based on each plant, EPC was analyzed. The average results discriminated by phenotypes are given in Table 1. Teosintoid plants

Table 1: Endosperm protein content of different phenotypes of *Z. perennis* x Gaspe hybrids.

| Phenotype | Endosperm protein content % |
|---|---|
| Teosintoid | 23.9[a] (*) |
| Intermediate | 22.7[ab] |
| Maizoid | 21.0[b] |

(*) Individual means followed by different letters are significantly different at 5 % level

have the same EPC as perennial teosinte, whereas maizoid plants are significantly lower than teosintoid, but superior to Gaspé. EPC of intermediate phenotypes is also intermediate, but no statistical difference was detected when compared with the extreme phenotypes.

These results point out a significant association, although it is not the expected one, between EPC and plant phenotype. Maizoid plants retain a high protein content. When association between EPC and plant individual characteristics is analyzed it may be inferred, as shown in Table 2, that: 1) those plants with later pollen production have high EPC; 2) those plants with a high number of tillers have high EPC; 3) those plants with distichous spikes have higher protein content than those with polystichous ones; 4) those plants with single female spikelets have higher EPC than those with paired ones; 5) plants whose spikes have a low number of kernel rows have high protein content. Despite the fact that the variation amplitude of EPC is not that which might be expected, a clear association between EPC and some specific characters exists within the restricted variability. Since this is a significant association, it can be stated that EPC is a specific trait, linked to other morphological characteristics that distinguish maize from perennial teosinte.

Table 2: Correlation coefficients (r) between endosperm protein content (EPC) and several morphological traits. *-- significant at 5 % level, **-- significant at 1 % level.

| Comparison | r |
|---|---|
| EPC vs. days to tassel. | 0.29 |
| vs. days to silking. | 0.32 |
| vs. days to pollen. | 0.36* |
| vs. number of tillers. | 0.46** |
| vs. number of leaves. | 0.13 |
| vs. number of productive nodes. | 0.05 |
| vs. number of ears in the uppermost node. | 0.17 |
| vs. number of ears per stalk. | 0.11 |
| vs. growth habit. | -0.02 |
| vs. tassel central spike type. | -0.18 |
| vs. female spike type. | -0.48** |
| vs. female spikelets arrangement. | -0.52** |
| vs. female rachis type. | -0.30 |
| vs. number of row of kernels. | -0.45** |

It is interesting to mark the fact that, even in those plants whose phenotype is clearly maizoid, the protein content is maintained considerably high. This suggests that high protein content transference into maize might take place without great inconvenience, although this high protein content may not be completely retained in maize. The fact that EPC is not wholly associated with specific traits suggests that a good part of the genes of the polygenic complex that conditions protein content segregate independently, and most of them may be dominant instead of having additive effects.

Liliana Ferrari and Jorge Luis Magoja

**Perennial teosinte-Gaspé hybrids: selection for maizoid characters**

As part of the plan whose aim is to select maizoid plants from hybrids between perennial teosinte and maize, selection was started in 1982. Maizoid plants of an $F_2$ population originating from the crossing between *Zea perennis* and Gaspé were selected, harvesting grains produced by free pollination. They initiated in 1983 the $F_3$ select maizoid ($F_3$SM) progeny, made up of approximately 70 plants. A number of specific or agronomically important characters were evaluated in the $F_3$SM. The studied traits were: days to tassel (T), days to silking (S), days to pollen (P), pollen grain size (PGS), pollen fertility (PF), tassel branch number (TBN), tassel branching axis length (TBAL), tassel central spike length (TCSL), lateral tassel branching internode length (LTBIL), lowermost tassel branch length (LTBL), stalk diameter (SD), number of tillers (NT), number of leaves (NL), leaf width (LW), leaf length (LL), number of productive nodes (PN), number of ears in the uppermost node (EUN), and number of ears per tiller (ET). In the same way some ear traits, such as ear type, spikelet type, kernel type and number of kernel rows, were evaluated. The studied traits were used to compare $F_3$SM with $F_2$ and $F_3$ populations, the latter coming from *Z. perennis* and Gaspé crosses in which no selection was made.

Tables 1-4 show the results obtained. Selection for maizoid characters had a positive result, because the $F_3$SM population stands out significantly for most of the characters over the non-selected $F_2$ and

Table 1: Evolutive cycle. T-- days to tassel; S-- days to silking; P-- days to pollen.

| | T | S | P |
|---|---|---|---|
| $F_3$SM (68) | $86.0^{ab}$(*) | $113.4^{ab}$ | $97.2^{a}$ |
| $F_2$ (580) | $83.3^{a}$ | $114.8^{a}$ | $104.1^{b}$ |
| $F_3$ (377) | $86.5^{b}$ | $108.8^{b}$ | $102.1^{ab}$ |

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

Table 2: Tassel branching and pollen traits. TBN-- tassel branch number. TBAL-- tassel branching axis length; LTBIL-- lateral tassel branching internode length; PF-- pollen fertility; PGS - pollen grain size.

| | TBN | TBAL (cm) | LTBIL (mm) | PF (%) | PGZ (um) |
|---|---|---|---|---|---|
| $F_3$SM (63) | $17.9^{a}$(*) | $10.9^{a}$ | $4.7^{a}$ | $86.7^{a}$ | $91.3^{a}$ |
| $F_2$ (427) | $7.8^{b}$ | $5.1^{b}$ | $4.5^{a}$ | $53.4^{b}$ | $88.1^{b}$ |

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

Table 3: Number of leaves (NL), number of productive nodes (PN) number of ears in the uppermost node (EUN) and ears per tiller (ET).

| | NL | PN | EUN | ET |
|---|---|---|---|---|
| $F_3$SM (58) | $12.8^{a}$(*) | $4.3^{a}$ | $6.2^{a}$ | $29.8^{a}$ |
| $F_2$ (129) | $10.8^{b}$ | $5.0^{b}$ | $5.0^{b}$ | $25.9^{a}$ |
| $F_3$ (347) | $10.2^{b}$ | $4.9^{b}$ | $4.6^{b}$ | $26.1^{a}$ |

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

Table 4: Ear traits

| | % of plants | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | spikelets | | kernel | | row number | | | |
| | single | paired | enclosed | naked | 2 | 4 | 6 | 8 |
| $F_3$SM (62) | 22.6 | 77.4 | 22.6 | 77.4 | 22.6 | 71.0 | 6.4 | - |
| $F_2$ (195) | 52.8 | 47.2 | 89.4 | 10.6 | 56.0 | 38.3 | 4.1 | 1.6 |
| $F_3$ (289) | 43.5 | 56.5 | 69.1 | 30.9 | 45.4 | 52.9 | 1.4 | 0.3 |

$F_3$ populations. Maizoid plants of the $F_3$SM population stand out for their high vigour and they present most of the maize-specific characteristics: high number of tassel branches, long tassel branching internodes, big pollen grains, paired female spikelets and naked kernels. Likewise, maizoid plants have thick stalks, low number of tillers, wide and long leaves, and they are highly productive.

Table 5 shows the significant correlations between characters. Most of the specific traits are associated. It means that selection based on a few maizoid characteristics implicitly leads to an indirect selection of those strongly associated traits. Maizoid plants stand out for their high prolificity. Selection on these types of plants in hybrids between perennial teosinte and maize progeny may have great practical importance to be used directly or indirectly in the improvement of maize production.

Table 5: Phenotypic correlation coefficients between characters of $F_3$SM plants. (*)-- Significant at 5 % level; (**)-- Significant at 1 % level.

| | | | r |
|---|---|---|---|
| T | vs. | S | 0.65** |
| | | P | 0.96** |
| | | TBN | 0.34** |
| | | TBAL | 0.25** |
| | | NT | -0.32** |
| | | NL | 0.43** |
| | | SD | 0.33* |
| | | LW | 0.34* |
| | | LL | 0.35* |
| S | vs. | P | 0.73** |
| | | TPN | 0.49** |
| | | TBAL | 0.30* |
| | | NL | 0.47** |
| | | SD | 0.34* |
| | | LW | 0.30* |
| | | LL | 0.29* |
| P | vs. | PGS | 0.25* |
| | | TBN | 0.37** |
| | | TBAL | 0.25* |
| | | NT | -0.32** |
| | | NL | 0.38** |
| | | SD | 0.30* |
| | | LW | 0.31* |
| | | LL | 0.33* |
| PGS | vs. | TBAL | 0.25* |

| | | | r |
|---|---|---|---|
| PGS | vs. | NT | -0.30* |
| PF | vs. | ET | -0.37** |
| TBN | vs. | TBAL | 0.64** |
| | | TCSL | -0.34** |
| | | NL | 0.33** |
| | | SD | 0.42** |
| | | LL | 0.28* |
| TBAL | vs. | ET | 0.27* |
| | | SD | 0.33* |
| | | LW | 0.42** |
| | | LL | 0.27* |
| LTBIL | vs. | PN | -0.25* |
| TCSL | vs. | NL | -0.38** |
| | | SD | -0.35* |
| | | LW | -0.39** |
| LTBL | vs. | EUN | 0.28* |
| NL | vs. | PN | 0.29* |
| NL | vs. | SD | 0.43** |
| | | LW | 0.49** |
| PN | vs. | ET | 0.51** |
| EUN | vs. | ET | 0.61** |
| SD | vs. | LW | 0.69** |
| | | LL | 0.67** |
| LW | vs. | LL | 0.46** |

The results obtained point out a positive answer to selection. This encourages great expectancy to obtaining high productivity maizes by means of wild germplasm utilization.

Gabriela Pischedda and Jorge Luis Magoja

## Effect of diploperennial teosinte germplasm on maize endosperm proteins

With the purpose to study the effect that diploperennial teosinte *(Zea diploperennis)* germplasm produces on storage proteins of hybrids with maize, endosperm protein patterns (Landry Moureaux protein pattern) were studied in maize inbred line OU, *Z. diploperennis* and $F_1$ hybrids (OU x Zd). Table 1 shows the results obtained. Parents differ significantly in their protein patterns, whereas the $F_1$ presents an intermediate ratio of saline soluble, zein and glutelin-3 proteins. High glutelin-1 character, which is brought forward by the tripsacoid line OU, behaves as dominant. Results obtained point out that *Z. diploperennis* effects on storage proteins in hybrid combinations with maize differ significantly from the effects produced by *Z. perennis* (see MNL 55:60). $F_1$

**Table 1: Endosperm protein pattern of OU, *Z. diploperennis* (Zd) and its $F_1$ hybrid. SS— saline soluble proteins; Z— zein; $G_1$— glutelin-1; $G_2$— glutelin-2; $G_3$— glutelin-3.**

| | soluble nitrogen (percent of total) | | |
|---|---|---|---|
| Fraction | OU | OU x Zd | Zd |
| SS | 4.8 | 4.0 | 3.1 |
| Z | 45.2 | 59.6 | 67.1 |
| $G_1$ | 16.2 | 16.4 | 9.2 |
| $G_2$ | 8.9 | 5.0 | 7.5 |
| $G_3$ | 15.8 | 14.1 | 10.0 |
| protein % | 13.4 | 13.8 | 27.0 |

hybrid kernels from crosses between maize and diploperennial teosinte are highly viable, in contrast to hybrids with perennial teosinte. This happens because diploperennial teosinte does not cause any dramatic alteration in maize protein pattern.

In accordance with maize genetical position, the different effect produced by *Z. perennis* and *Z. diploperennis* on storage proteins demonstrates that the first species is situated further than the second from maize. Production of balanced protein patterns in hybrids between maize and *Z. diploperennis* shows their high affinity. In our opinion, this similarity takes place because of the important maize germplasm introgression assimilated by *Z. diploperennis*.

Angel Alberto Nivio and Jorge Luis Magoja

### Immediate effect of perennial teosinte introgression in maize: endosperm storage proteins

Perennial teosinte germplasm introgression into maize conditions a considerable increase of vigour, because they are the two taxa of *Zea* genetically farther apart. This fact has begun to be used with practical purposes. In spite of that, immediate introgression effects do not seem to be advantageous, because kernels produced by crosses between maize and perennial teosinte germplasm have low viability in most cases. The aim of our work was to research the association between low kernel viability and endosperm storage protein patterns. Three maize inbred lines, OU, SBP and SAP, were pollinated with a donating material from perennial teosinte germplasm, which was a selected $F_3$ progeny ($F_3S$) from hybrids between perennial teosinte and Gaspé. This $F_3S$ was selected for its high fertility and predominance of maizoid characteristics. Endosperm proteins were fractionated according to Landry-Moureaux (1970) in lines, donating material and kernels produced by the cross between them (see Table 1).

The lines used have a tripsacoid protein pattern (see MNL 56:106), characterized by its relatively low ratio of saline-soluble and high ratio of glutelin-1 proteins. These lines with tripsacoid protein pattern (associated with other tripsacoid characters such as glume induration) were chosen with the purpose of

Table 1. Endosperm protein pattern of OU, SBP, F3S and its hybrids. SS--saline soluble proteins; Z--zein; G1--glutelin; G2-- glutelin-2; G3--glutelin-3.

| | soluble nitrogen (percent of total | | | | | | |
|---|---|---|---|---|---|---|---|
| Fraction | OU | OU x $F_3S$ | SBP | SBP x $F_3S$ | SAP | SAP x $F_3S$ | $F_3S$ |
| SS | 4.8 | 9.1 | 3.9 | 11.1 | 3.4 | 6.6 | 4.7 |
| Z | 45.2 | 43.0 | 42.7 | 33.1 | 58.4 | 49.3 | 63.4 |
| $G_1$ | 16.2 | 15.0 | 25.6 | 23.4 | 15.4 | 15.7 | 12.9 |
| $G_2$ | 8.9 | 6.3 | 7.0 | 8.9 | 5.0 | 6.7 | 5.5 |
| $G_3$ | 15.8 | 20.4 | 17.2 | 18.9 | 15.6 | 16.2 | 12.6 |
| protein % | 13.4 | 15.1 | 13.3 | 15.0 | 15.2 | 16.0 | 15.8 |

helping (or favouring) perennial teosinte germplasm combinations. The donating material ($F_3S$) has a normal protein pattern that does not differ significantly from the one of maize.

A deep protein pattern modification may be observed in hybrid kernels, making them significantly different from maize maternal lines. This modification is approximately similar in the 3 analyzed crossings; the protein patterns that result are characterized by: 1) an increase in saline soluble proteins (approx. twice) 2) a diminution in zein and 3) an increase in glutelin-3. The phenomenon presented here is similar to that reported previously (MNL 55:60) in crosses between Gaspé and perennial teosinte.

Without any doubt, the most remarkable fact is the high increase of saline soluble proteins, which have been associated to kernel inviability (see MNL 55:60). Hybrid protein patterns are a lot like those from maize defective kernels. They are also very similar to those protein patterns affected on a higher or lesser scale by several mutants. The results obtained point to a strong association between endosperm protein pattern and low kernel viability. Consequently, it can be stated that the immediate effect of perennial teosinte introgression into maize becomes deleterious, because when endosperm proteins are unbalanced they alter kernel viability. The immediate effect observed differs deeply from introgression mediate effect. In accordance with introgression progress, kernel viability is quickly recovered. Simultaneously, the individuals obtained acquire a tripsacoid protein pattern (correctly balanced) and characterized by its low ratio of saline-soluble and high ratio of glutelin-1 proteins.

Angel Alberto Nivio and Jorge Luis Magoja

## LONDON, CANADA
University of Western Ontario

### *In vitro* liquid culture of corn tassels - an update!

The procedures reported for the successful culture of Seneca-60 tassels (MGCNL 55:116, 1981) do not support spikelet development in field corn cultivars such as Oh43. Considerable normal tassel development in Oh43 is achieved, however, with the modifications which we now report.

Tassels 1.0 to 1.5 cm long (approximately 35-45 days from seed imbibition) are explanted into 40 ml of liquid medium in 125 ml Erlenmeyer flasks. Flasks are maintained for 21 to 23 days in a growth cabinet at 28 C and an 18 hr day (3-400 ft. c.) and 6 hr night regime. The flasks are placed flat on a white reflective surface and are not shaken during the culture period. The composition of the liquid medium and other conditions were arrived at from

1

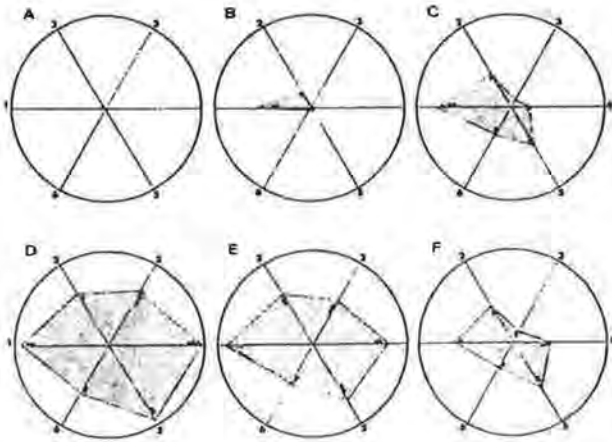



Fig. 1. Effect of sucrose on tassel growth and development: kinetin at $10^{-7}$ M.

A) control B) 0.1 M C) 0.2 M D) 0.3 M
E) 0.4 M F) 0.5 M*

2

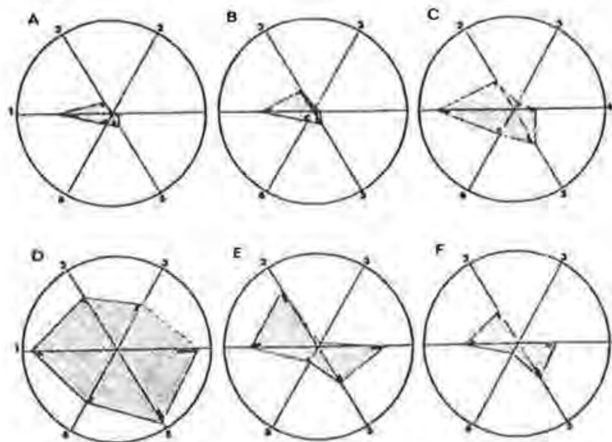



Fig. 2. Effect of kinetin on tassel growth and development: sucrose at .3 M.

A) control B) $10^{-9}$ M C) $10^{-8}$ M
D) $10^{-7}$ E) $10^{-6}$ M F) $10^{-5}$ M*

* Identification of axes: Units and/or range of values per axis are bracketed.

1. Final length: (0-6 cm)
2. Final fresh weight: (0-6 g)
3. Number of normal spikelets per tassel: (0-200)
4. Total number of spikelets per tassel: (0-200)
5. Glume length: (0-6 mm)
6. Number of anthers per spikelet: (0-6)

the following observations on the effect of different levels of sucrose, plant growth regulators, casein hydrolysate and physical conditions, such as the size of explant and shaking vs. non-shaking conditions.

To characterize growth and development, the tassels were assessed after 21 days of culture by six parameters: (1) final length; (2) final fresh weight; (3) total number of spikelets per tassel; (4) number of normal spikelets per tassel; (5) glume length and (6) number of anthers per spikelet. The response to different sucrose levels is summarized in the polygon figures (Fig. 1; a to f), where the extent of the development is represented by the relative amount of shading. For all parameters measured, the tassel explants responded to the greatest extent at a sucrose level of 0.3 M. Concentrations both below and above this value supported less development. These results with sucrose were obtained with the cytokinin, kinetin at $10^{-7}$ M and casein hydrolysate at 30 mg/l.

The importance of cytokinin for development of spikelets in culture is indicated by the data summarized in Fig. 2, a-f. At kinetin levels of $10^{-7}$ M, the maximum development of all parameters measured was achieved. At concentrations both above and below this value, significantly less development took place. At the higher concentrations ($10^{-6}$ and $10^{-5}$ M), spikelet abnormalities and other inhibitory effects were common. The addition of indole acetic acid and gibberellic acid was not beneficial to development, and in fact were inhibitory at low concentrations. They were therefore not included in the medium. From some preliminary data we conclude that casein hydrolysate (30 mg/l) enhances spikelet development, though the total requirements or effects of individual amino acid supplementation have yet to be studied.

In addition to these nutritional features, the success of normal growth of spikelets is related to the initial explant size. Tassels 1.0 to 1.5 cm long underwent normal differentiation producing 100-200 normal two-flowered spikelets. Younger tassel explants (0.5 cm or less) grew abnormally and frequently produced vegetative plantlets. Older tassel explants (2-3 cm) developed poorly. Shaking is no longer considered to be necessary for significant normal development of the cultured tassels.

In summary, tassels of Oh43 grow well on a liquid medium containing M&S major and minor minerals, White's vitamins and glycine, i-inositol 100 mg/l, sucrose 0.3 M, kinetin $10^{-7}$ M, casein hydrolysate (30 mg/l).

D. R. Pareddy and R. I. Greyson

### *In vitro* germination of pollen from cultured tassels

Previous workers have championed the potential of pollen as a "window" through which fresh insights on the life cycle will be gleamed and hereditary transformation might be induced (K. Raman et al., J. Hered. 71:311, 1980; R. Flavell and R. Mathias, Nature 307:108, 1984; J. H. Krieger, Chem. Eng. News, Oct. 1984, p. 16). What might be true for normal pollen should be equally true for pollen derived from the tassel culture system, if in fact the pollen is normal, germinable and viable. Polowick

Table I: Summary of data from a number of experiments on germination on CWBM of pollen derived from cultured tassels.

| Cultivar | Anther type | % ghosts ± S.E.* | % pollen burst ± S.E.* | % germ. pollen ± S.E.* | % un-germ. pollen ± S.E.* | Total (N) |
|---|---|---|---|---|---|---|
| Oh43 | a) un-extruded | 26.51 ± 5.26 | 14.87 ± 0.59 | 7.49 ± 1.27 | 51.10 ± 5.01 | 3261 |
| | b) extruded | 7.58 ± 0.43 | 16.44 ± 1.10 | 43.27 ± 0.17 | 31.19 ± 3.43 | 3647 |
| Se60 | a) un-extruded | 27.00 ± 3.69 | 10.15 ± 1.92 | 10.54 ± 0.17 | 52.28 ± 2.00 | 2300 |
| | b) extruded | 4.21 ± 1.29 | 2.79 ± 1.97 | 51.33 ± 2.15 | 41.89 ± 1.62 | 2241 |

* Standard errors are based on arcsin-transformed data.

(M.S. thesis, U.W.O., 1981; Can. J. Bot., in press) documented the apparently normal cytogenetic features of pollen development in Seneca 60. The present report extends these observations to Oh43 and documents the *in vitro* germinability of this pollen.

Cultured tassels grow *in vitro* and produce spikelets after 16-20 days for cv. Seneca 60 and 20-25 days for cv. Oh43. Frequently, of the 100-200 normal spikelets per tassel, 5-20% yield extruded anthers with elongated filaments. Pollen from these extruded anthers, when spread on the surface of Cook and Walden Basal Medium (CWBM) (Can. J. Bot. 43:779, 1965) with an agar content of 1.5%, germinates well. For both Seneca 60 and Oh43, 40-50% of the pollen grains (Table 1) produce pollen tubes, while 30-40% of the grains possessing apparently normal cytoplasm remain ungerminated. Germination from unextruded anthers is also observed (8-11%), but at a reduced rate for both cultivars. In these, a much higher frequency (~25%) of "ghosts" is observed.

The *in situ* germinability of this *in vitro* derived pollen has yet to be demonstrated convincingly. Other than minor technical complications, we foresee, however, no serious reason why pollen from the tassel cultures cannot be used to produce viable embryos—whether *in vivo* or *in vitro*. When this is achieved, tests which attempt to introduce foreign hereditary factors into the pollen can be attempted.

D. R. Pareddy, R. I. Greyson
and D. B. Walden

## *In vitro* culture of immature ear shoots

Polowick's (M.S. thesis, U.W.O., 1981) *in vitro* inflorescence techniques provide an opportunity to explore the regulation of sexuality more closely than heretofore. It allows for (1) more precise application of exogenous factors, (2) control of environmental factors like photoperiod, temperature, etc., (3) testing of only one growth regulator at a time, (4) more uniformity in supplying the nutrients with a defined media, and (5) considerable ease of observation.

In order to provide a basis for future studies of cultured immature ear shoots, we have explored the importance of sucrose and kinetin. Our basal medium consisted of Murashige and Skoog (1962) major and minor minerals, White's vitamins and glycine, and inositol (100 mg/l). Ears were explanted into 125 ml Erlenmeyer flasks containing 40 ml of media. They were first exposed to 3 days on an orbital shaker and another 20+ days unshaken, on a white reflective shelf in an illuminated incubator. A day/night cycle of 18/6 hours of light with 400-500 f.c. and 28 ± 2 C temperature was maintained throughout the growth period.

Table 1 summarizes the effect of the sucrose concentration on explanted ear shoots. In terms of

**Table 1. Mean final fresh weight (gm) values of cultured Seneca-60 ear shoots (5.0-10.0 mm) after 25 days, under different sucrose conc. levels.**

| Sucrose conc./ (with $10^{-8}$ M) kinetin | Fresh Wt. (gm) ± S.E. (N) |
|---|---|
| 0 M | 0.016 ± 0.00 (3) |
| 0.3 M | 8.81 ± 1.76 (3) |
| 0.6 M | 14.97 ± 1.51 (4) |
| 0.9 M | 8.19 ± 2.38 (4) |
| 1.2 M | 4.71 ± 1.01 (4) |

fresh weight of the whole inflorescences, an optimum concentration is found at 0.6 M. This concentration is also apparently optimal for flower development. Representative experiments with kinetin are summarized in Table 2. Depending upon the concentration of sucrose, the level of kinetin for optimum growth (fresh weight) of the ear is $10^{-7}$ or $10^{-6}$ M. The proportion of male to female flowers developing on explanted ears under optimal kinetin and sucrose concentrations varies depending upon the size of the initial explants (Table 3). Short (young) ears produce more male flowers whereas longer (older) ears produce more female flowers. While the significance of this observation has yet to be explored, it should

Table 2. Mean fresh wt. of cultured ear shoots after 20 days, under different kinetin and sucrose levels.

| Sucrose conc. (M) | Kinetin conc. (M) | 0 | $10^{-10}$ | $10^{-9}$ | $10^{-8}$ | $10^{-7}$ | $10^{-6}$ | $10^{-5}$ |
|---|---|---|---|---|---|---|---|---|
| | | Fresh wt. (gm) ± S.E. (N) | | | | | | |
| 0.2 | | 0.52± 0.1 (7) | 0.94± 0.08 (6) | 1.31± 1.0 (6) | 3.52± 1.0 (6) | 4.7± 1.1 (7) | 0.08± 0.2 (6) | 0.02± 0.0 (6) |
| 0.6 | | 1.08± 0.35 (7) | 3.55± 0.83 (8) | 2.93± 0.61 (8) | 8.0± 1.19 (8) | 11.31± 0.68 (8) | 14.39)± 1.63 (6) | 0.08± 0.02 (5) |

**Table 3. Mean percentage of differentiated flowers of cultured Seneca-60 ear shoots after 20 days in the presence of kinetin ($10^{-7}$ M); sucrose (0.6 M).**

| Size of explant (mm) (N) | Male | Bisexual | Female | Abnormal |
|---|---|---|---|---|
| 5.0-10.0 (5) | 87.8 | 1.83 | 3.05 | 7.32 |
| 10.1-15.0 (5) | 51.84 | 7.36 | 31.02 | 9.78 |
| 15.1-20.0 (3) | — | — | 100 | — |

be noted that this differential result is achieved without the application of either IAA or $GA_3$.

V. R. Bommineni and R. I. Greyson

## Characterization of polypeptides synthesized by mRNA isolated from pollen

Total RNA and polysomes extracted from maize pollen were translated *in vitro* in the rabbit reticulocyte lysate system. Protein products were separated on 1 and 2 dimensional SDS-PAGE gels, and protein profiles compared. Pollen from 35 inbred cultivars and 8 hybrids was collected and stored at -70 C. As a control for environmental variability, samples of Oh43 were collected at various times during the growing season (1983 and 1984). A series of pollen storage experiments was also undertaken. Pollen was collected and stored at 4 C in the dark for up to 8 days.

Translation products from total RNA separated on SDS-PAGE gels show 20 distinct bands for one-dimensional and 80-100 spots for two-dimensional separations. The size classes range from 94Kd to 14Kd with two major groups of proteins at 43 and 32 Kd. Unlabeled pollen proteins, stained with Coomassie blue, show a similar spread of size classes with major bands at 32, 38, 43 and 50 Kd. The polypeptides for translation products of free and bound polysomes show similar banding patterns with some qualitative and quantitative differences. A comparison of translation product patterns from both total mRNA and polysomes shows at least 24 spots in common, 17 of which are major spots. Total mRNA and free polysome patterns show at least 17 spots in common, 8 of which are most prominent.

A comparison of polypeptide patterns from samples of Oh43 monitored at weekly intervals over the growing season show no quantitative or qualitative differences on 1D or 2D gels. Preliminary comparison of polypeptide patterns from inbreds and hybrids shows no significant differences at the 1D level. Cold storage of pollen at 4 C shows deterioration of mRNA in Oh43 after 8 days of storage but very little deterioration of mRNA in SC after 6 days.

M. J. Dunlop and D. B. Walden

## Incorporation of radioactively labeled amino acids into protein in leaves does not require prior surface abrasion

Research interests in our laboratory require examination of the pattern of polypeptide synthesis in leaf tissue throughout the life of the plant. We find it desirable to label the leaf tissue of the intact plant since previous work in our laboratory (J. G. Boothe et al., MNL 56:113, 1982) suggested that injury to the leaf can induce a change in the pattern of polypeptide synthesis. Other workers have described a method for incorporating labeled amino acids into leaves of intact plants following abrasion of the upper leaf surface (P. Cooper et al., Plant Physiol. 75:431, 1984).

Various protocols for incorporation of $^{35}$S-Methionine into proteins of leaf tissue were conducted last summer. We compared the incorporation of $^{35}$S-Methionine into TCA-precipitable material of leaf tissue with and without abrasion immediately before delivery of the label. Abrasion was achieved by lightly rubbing a 4 cm x 2 cm area of the upper leaf surface with fine sandpaper (No. 400). $^{35}$S-Methionine (10 $\mu$Ci) was applied to this area. Labeling time was 1 h; during the period of labeling, temperature was monitored using a thermo-couple and recorded every 10 min using a digital data logger (DL-2020, ECD). The average temperature over this time period was 20 C. Proteins were extracted, TCA precipitated and liquid scintillation counted as described previously (C. L. Baszczynski et al., Can. J. Biochem. 60:569, 1982). Representative data are:

| Leaf Sample | Treatment | cpm/μl/2 cm² sample |
|---|---|---|
| 1 | Abrasion | 15,873 |
| 2 | Abrasion | 13,918 |
| 3 | None | 13,987 |
| 4 | None | 9,925 |

We conclude that abrasion is not required to achieve incorporation of $^{35}$S-Methionine into proteins of maize leaf tissue. We have omitted the use of an abrasive from our procedure since the injury response may alter the pattern of peptides synthesized.

C. A. Rees, T. G. Crowe and D. B. Walden

**Isolation of a mRNA for the 18,000 dalton maize heat shock polypeptide**

Plants respond to heat shock by synthesizing a set of heat shock polypeptides (HSPs) (C. L. Baszczynski et al., Can. J. Bioch. 60:569, 1982, P. Cooper et al., Plant Phys. 75:431, 1984). Poly A+ RNA isolated from 5-6 day old seedlings following heat shock directs the synthesis of the HSPs, in both a rabbit reticuloycte lysate system and a wheat germ system (Baszczynski et al., Can. J. Bioch. 61:395, 1983). RNA was isolated as previously described from plumules of 5-6 day old seedlings (Oh43) following temperature shift (25 C-42 C, 1 h). Oligo-dT-chromatography of the RNA was carried out and the poly A+ fraction was collected. Approximately 2 mg/g fresh tissue of RNA were recovered, of which 20% was the poly A+ fraction. This fraction was contaminated with rRNA (seen as 28S and 18S bands on agarose gels) even after repeated passage through the column.

Agarose gel electrophoresis (under denaturing conditions) was used to separate RNA molecules. Use was made of mRNA affinity paper, which reversibly binds poly A+ RNA (D. H. Wreschner and M. Hersberg, Nucl. Acid. Res. 12:1351, 1984). Poly A+ RNA was separated in urea/citrate denaturing, 1.75% agarose gels (R. E. Smith and Y. Furuichi, Virology 103:279, 1980). This "non-invasive" denaturing system was recommended by the developers of mAP paper. RNA samples (50 μg/3.5 cm well) were prepared by diluting with an equal volume of 9 M urea, boiling for 1 minute and cooling on ice. Gel electrophoresis was carried out for 22 h (2 V/cm, RT). Standard and sample lanes for staining were cut from the gel and stained with ethidium bromide (2 μg/ml). Sample lanes were equilibrated in 500 mM Tris HCl pH 7.6 for 2 h. The gel was blotted to mAP paper using the same buffer (20 h). The mAP paper was washed in transfer buffer and sections containing mRNA (determined from the stained sample lanes) were cut from each lane. The sections were cut sequentially into 0.5 cm pieces which were rinsed in ethanol (70%), dried and placed in 0.6 ml Eppendorf tubes. Sterile $ddH_2O$ (100 μl) was added and the tubes were placed at 70 C for 3 minutes. The tubes were punctured at the bottom and placed in 1.5 ml Eppendorf tubes which were centrifuged 3 min. The RNA was ethanol precipitated, dried briefly and translated in a rabbit reticulocyte lysate system using $^{35}$S methionine as labeled precursor. Translation products were separated by SDS-PAGE and fluorography was conducted. Examination of the fluorograms showed a prominent translation product of molecular mass 18,000 daltons. The message which translates into this polypeptide was eluted from an agarose gel slice determined to contain RNA molecules 630-850 nucleotides in length. Work is underway to prepare complementary DNA to this RNA using reverse transcriptase.

C. A. Rees

**Heat shock polypeptide synthesis in monosomic plants**

A study is underway in our laboratory to determine the effect of loss of a particular chromosome on the maize heat shock response at the level of protein synthesis. In the summer of 1984, ten groups of plants were produced, each monosomic for one of the ten chromosomes of *Zea mays* L. The *r-X1* deficiency was used to generate monosomics. We are grateful to Jerry Kermicle and John and Susan Laughnan for the provision of *r-X1* stocks several years ago. The seed used in this study was produced in our nurseries during the period 1975-1980. Seed produced by crossing females carrying the *r-X1* deficiency with males homozygous recessive for a visible seedling marker was germinated and grown to the 2-4 leaf stage (14 days). Plants expressing the recessive phenotype, suggesting loss of the chromosome carrying the marker gene, were selected. Disomic plants from the same cob were retained as controls. Table 1

Table 1.

| Chromosome | Total Seed No. | Phenotype | Frequency | Total Freq. |
|---|---|---|---|---|
| 1 | 1481 | sr1 | 0.0034 | |
| | | white | 0.0027 | 0.0068 |
| 2 | 881 | v4 | 0.0035 | |
| | | striped | 0.0045 | 0.0125 |
| 3 | 869 | gl6 | 0.0012 | 0.0012 |
| 4 | 815 | gl3 | 0.0074 | 0.0074 |
| 5 | 736 | v3 | 0.0027 | 0.0027 |
| 6 | 1006 | v*-8520 | 0 | |
| | | striped | 0.0050 | 0.0050 |
| 7 | 326 | gl1 | 0.0123 | 0.0123 |
| 8 | 1597 | v16 | 0.0263 | 0.0263 |
| 9 | 1684 | yg2 | 0.0053 | 0.0053 |
| 10 | 1553 | oy | 0.0039 | 0.0039 |

Total seeds examined = 10948; Total monosomics = 96

lists the markers used for each chromosome and indicates the frequency of plants expressing the recessive phenotype. Cytogenetic confirmation of chromosome constitution was obtained from primary root tips.

Presently, the patterns of polypeptide synthesis (generated by SDS-PAGE and fluorography) in secondary roots of plants at the 6 leaf stage (50 days) are being compared before and following heat shock of intact plants. Preliminary results suggest that loss of chromosomes 1, 2, 3, 4, 7, 8, 9 and 10 does not affect the response to heat shock at the level examined. Plants monosomic for chromosomes 5 and 6 have not been examined. The change in the pattern of polypeptide synthesis following heat shock appears the same for specific monosomic plants and their disomic controls. The group of polypeptides (HSPs) characteristic of the heat shock response is evident in both disomic and monosomic plants. The level of synthesis of these polypeptides also appears similar. Fluorograms are being scanned spectrophotometrically and the distribution of $^{35}$S-Methionine among polypeptides is being compared in monosomic and disomic plants. Antiserum raised to the 18,000 dalton polypeptide will be used to immunoprecipitate and quantify this polypeptide.

C. A. Rees and D. B. Walden

**Comparisons of newly synthesized polypeptides obtained from maize embryos of different genotypes and developmental ages**

As reported last year (MGCNL 58:136, 1984), 1D SDS PAGE separations of protein extracted from maize embryos enabled the detection of qualitative and quantitative differences in newly synthesized polypeptides occurring as a function of both development and genotype. In order to better resolve some of these differences, 2D IEF-SDS PAGE was performed.

Included in this study were two inbred cultivars, Oh43 and M14 and their reciprocal hybrids. Embryos were selected at 17 and 20 days post-pollination and subsequently at 5 day intervals to 40 days post-pollination. Labelling of embryos *in situ* with $^{35}$S-methionine was performed as described by Kriz (MGCNL 56:14-15, 1982).

Between 150 and 300 polypeptides were resolved routinely following fluorographic analysis of 2D gels. Initial attempts to quantitate some of these data have revealed an apparent increase of 20-30% in the number of polypeptides synthesized at 30 days over that at 20 days post-pollination. Conversely, the number of polypeptides synthesized at 40 days decreased by 15% from that observed at 30 days post-pollination. A shift in the distribution of newly synthesized polypeptides was also evident, in that a larger proportion of low molecular weight polypeptides (below ~30 Kd) were observed during the latter stages of development, between 30 and 40 days post-pollination.

Comparisons of 2D patterns obtained from different inbreds have suggested that at least 5% of the polypeptides synthesized may be genotypically unique. A prominent example of this was observed in Oh43 where a pair of spots of approximate pI 5 and molecular weight 69 Kd could be detected throughout the period of study but at no time were evident in M14. Also in M14 a group of spots migrating at approximately pI 4 and molecular weight 76 Kd were observed at all stages after 25 days, but could not be detected in Oh43 prior to 35 days post-pollination. In the latter case this may indicate that regulation of the synthesis of these polypeptides is timed differently in the two cultivars. All of the polypeptides synthesized by the inbred parents were detected in both reciprocal hybrids.

A number of polypeptides appeared to vary quantitatively with respect to relative amounts synthesized across both development and genotype. In those cases where the inbreds showed qualitative differences, these were reflected quantitatively in the hybrids, such that the quantity synthesized appeared to be most influenced by the maternal parent. These findings suggest a possible allele dosage effect in the case of the specific peptides studied.

The utilization of computerized image processing and statistical analysis are presently being developed to facilitate the collection, manipulation and interpretation of data obtained from 2D electrophoretic separations.

J. G. Boothe and D. B. Walden

**Response to certain agrichemicals as measured by $^{35}$S-methionine uptake and protein synthesis**

An assortment of agrichemicals (herbicides, pesticides and fungicides) recommended for use on maize was examined as additives to our 5 day old plumule/radicle system designed earlier for stress studies. Each agrichemical was examined in five temperature regimes (20, 30, 40, 20-30 and 30-40), at three 'doses' (0, 0.01, 1.0 and 10.0 'F.D.', where 'F.D.' was the manufacturer's recommended field dose) for both plumules and radicles.

Standard procedures (Baszczynski, C. L., D. B. Walden and B. G. Atkinson, Canad. J. Biochem. 60:569-579, 1982) were followed to produce fluorograms from 1 and 2D IEF-SDS PAGE electrophorograms. Visual examination of these fluorograms permitted the response to be classified in one or more of four categories:

| Class | Effect | Examples |
|---|---|---|
| I | Equal to control | Zineb (F)<br>Sevin (I)<br>Malathion (I) |
| II | Dose response | Atrazine (H)<br>Roundup (H)<br>Banvel (H)<br>Bladex (H) |

| | | |
|---|---|---|
| | | Diazinon (I) |
| | | Lannate (I) |
| | | Thiram (F) |
| III | Tissue specificity | Atrazine (H) |
| | | Roundup (H) |
| | | Thiram (F) |
| IV | General inhibition | 2-4 D (H) |
| | | Lasso (H) |

Equally revealing and undoubtedly more accurate were the data from the uptake/incorporation of $^{35}$S-methionine into TCA precipitable material. In controls, on a dry weight basis, there is approximately 3x protein in plumules as in radicles. However, the incorporation of $^{35}$S is 10x greater per unit protein in radicles than in plumules. Agrichemicals in classes II, III and IV above provoke a similar response in incorporation of $^{35}$S-methionine as in protein synthesis.

These studies are continuing and being extended to other possible stress inducing factors.

D. B. Walden and R. B. Zabulionis

MEADVILLE, PENNSYLVANIA
Allegheny College

**Long-term heat shock in maize seedlings**

A rapid upward shift in temperature, or heat shock, inhibits normal protein synthesis and induces the new synthesis of proteins called heat shock proteins (HSP). Corn seedlings synthesize a set of HSP in response to short exposure to high temperature (Baszczynski et al., Can. J. Biochem. 60:569, 1982). Most studies on heat shock in corn as well as other higher plants have utilized short (1-4 hr) heat shock incubations and detection of HSP by fluorography of newly synthesized proteins. HSPs accumulate to 10% of total protein detectable in Drosophila embryos after 10 hr heat shock incubation (Moran et al., Phil. Trans. Roy. Soc. Lond. B. 283:391, 1978). We have investigated the effect of long term heat shock on synthesis of HSP in corn. Oh43 seeds were germinated for 4-5 days and subjected to heat shock of 41C in an incubator under high moisture conditions for varying lengths of time (2, 4, 6, 8, 10, 12, 18, 24 hr). Proteins were extracted according to Baszczynski et al. and separated by SDS-PAGE. After 10-12 hr of heat shock treatment (27C to 41C) we found significant accumulation of higher molecular weight HSP (85K to 70K proteins). By 24 hours the HSPs corresponded to 10-15% of total protein detectable by Coomassie blue staining. Our results directly contradict the published results of Cooper and Ho (Plant Physiol. 71:215, 1983). These investigators report that HSPs do not accumulate in excised corn roots in response to various intervals of heat shock. There may be an important difference between excised roots and intact seedlings in terms of accumulation of proteins. Newly synthesized proteins were also prepared by subjecting seedlings to various intervals of heat shock and subsequently labelling for 2 hr with $^{35}$S-methionine. Proteins were extracted and separated as described above, and fluorographs compared for detection of newly synthesized proteins. New HSP synthesis occurs up to 18 hr, with no detectable protein synthesis at 24 hr. This indicates that proteins accumulated at 24 hr are previously synthesized and stable.

Thermotolerance is the proposed function of HSPs. Pretreatment of soybean seedlings at a high incubation temperature (40C) for a short period of time (10 to 20 min) provides protection from an otherwise lethal exposure to 45C (Lin et al., Plant Physiol. 74:152, 1984). We have investigated the role of HSP in the induction of thermotolerance in corn by subjecting seedlings to various incubation time and temperature regimes. A 2 hr exposure of 45C is lethal to 5 day Oh43 seedlings. However, if seedlings are incubated at 41C for periods of not less than 2 hr and not more than 6 hr (heat shock) and then exposed to the lethal temperature for 2 hr, the seedlings survive to grow. Growth is severely inhibited but seedlings will eventually grow to maturity. Longer heat shock times (greater than 6 hr) do not protect the seedlings from lethal temperature. Seedlings incubated at the original temperature after long heat shock incubations (6 to 24 hr) survive to maturity, but growth is inhibited. Measures of growth (seedling length in mm, fresh weight in g, dry weight in g) are all decreased by 40-60% at long heat shock incubations compared to controls. Our conclusion is that accumulation of HSPs in corn seedlings after long term heat shock (10-24 hr) does not correlate with the induction of thermotolerance. This does not preclude a causative role for HSPs in the phenomena but points to a more complex model for induction of proteins, accumulation of HSPs and thermotolerance, at least in corn.

Bruce H. Rockwell, Patricia Lehman
and Christine M. Nebiolo

MILAN, ITALY
University of Milan

**Growth of embryos, shoot and root tips of *Zea mays* L. in media devoid of nitrogen**

This note is for those who use or intend to use maize organ cultures for physiological or genetical work.

Nitrogen and carbon are the two major macronutrients required by living cells. In the majority of media formulations commonly used for plant tissue culture, the main nitrogen sources are $NO_3^-$ and $NH_4^+$ and also a wide range of amino acids and

GROWTH OF EXCISED EMBRYOS, SHOOTS AND PRIMARY ROOT TIPS CULTURED FOR DIFFERENT DAYS IN PRESENCE (+N) OR ABSENCE (-N) OF NITROGEN SOURCE. THE VALUES ARE MEAN OF AT LEAST 20 DETERMINATIONS. FIGURES IN BRACKETS REFER TO STANDARD ERROR.

| | | 0 | 7 DAYS | | 14 DAYS | |
|---|---|---|---|---|---|---|
| | | | $-N_a$ | $+N_a$ | $-N_a$ | $+N_a$ |
| ROOTS | LA | 1.3 (0.6) | 6.8 (0.3) | 5.8 (0.4) | 7.6 (0.3) | 7.4 (0.2) |
| | FW | 15.9 (0.8) | 37.3 (2.3) | 34.7 (2.3) | 41.1 (1.3) | 36.3 (0.7) |
| | DW | 1.1 (0.1) | 3.6 (0.2) | 2.5 (0.2) | 5.2 (0.4) | 3.1 (0.3) |
| | DW/FW | 7.1% | 10.4% | 7.2% | 12.7% | 8.6% |
| SHOOTS | LA | 1.6 (0.1) | 6.6 (0.2) | 9.0 (0.2) | 6.1 (0.2) | 12.8 (0.5) |
| | FW | 36.1 (2.9) | 133.9 (5.1) | 212.4 (11.6) | 188.0 (5.7) | 892.0 (39.1) |
| | N leaves | - | 1 | 2 | 1 | 3 |
| | Secondary roots | no | no | yes | no | yes |
| | | | 12 DAYS | | 28 DAYS | |
| | | | $-N_b$ | $+N_b$ | $-N_b$ | $+N_b$ |
| SEEDLINGS | LA | | 12.3 (0.5) | 15.5 (0.5) | 12.9 (0.9) | 30.9 (1.2) |
| | FW | | 283.6 (16.2) | 432.6 (11.4) | 574.8 (25.2) | 1476.6 (117.8) |
| | N leaves | | 2 | 3 | 2 | 5 |

$N_a$ : $Ca(NO_3)_2 \cdot 4H_2O$ (288 mg/l) , $KNO_3$ (80 mg/l)
$N_b$ : $NH_4NO_3$ (1650 mg/l) , $KNO_3$ (1900 mg/l).
Abbreviations: LA = mean length in mm , FW = mean fresh weight in mg , DW = mean dry weight in mg.

casein amino acids. During the course of experiments on amino acid metabolism we observed that primary root tips were able to grow in a medium devoid of nitrogen. This unexpected result induced us to find out if the presence of nitrogen in the medium is an absolute nutritional requirement for short term cultures.

For this purpose we cultured embryos, shoot and primary root tips in the presence or absence of nitrogen. Seeds of W22 inbred line were surface sterilized and germinated as previously described (Gavazzi, G. et al., Theor. Appl. Genet. 46:339-345, 1975). Embryos dissected from the endosperms were cultured on a medium consisting of the major and minor salts of Murashige and Skoog (Physiol. Plant. 15:473-497, 1962) supplemented with thiamine (0.4mg/l), sucrose (30g/l) and solidified with agar. For shoot and root cultures, tips were excised shortly after their emergence (10-15 mm long), transferred aseptically in a flask and cultured in a modified White (Growth 7:53-56, 1943) and F (Racchi et al., Plant Sci. Lett. 13:357-364, 1978) media, respectively. The flasks were kept on a rotary shaker (100 rpm) at 25± 2 C under 14 hours of daily light.

The results obtained (See table) indicate that embryos and shoots show an absolute requirement for a nitrogen source; in fact, after a few days of culture (7 for shoots and 12 for embryos) in the absence of nitrogen, they show a visible growth inhibition with necrosis of the leaves. In contrast, root tips have normal growth, at least for the time period tested, in media devoid of the commonly used nitrogen sources. Root tips seem to grow in these conditions even better than in the presence of nitrate, and the ratio dry weight/fresh weight is higher than that of the control.

This result could be very useful in view of the fact that the lack of a nitrogen source in a culture medium reduces bacteria and fungal growth, thus preventing culture contaminations. Further analyses are under way to evaluate $NO_3^-$ pool, and protein and amino acid content of root tips grown in these conditions.

Chiara Tonelli and Isabella Viani

## A new cherry allele at the *R* locus: *r-ch(Hopi)*

The symbol *r-ch(Hopi)* refers to an *r* allele isolated from a stock kindly supplied to us by the late Dr. Brink and tracing back to a Hopi Indian strain. Like other "cherry" accessions it leads to red pigmentation in seedling and plant tissues, while its presence together with the *Pl* factor in the plant genotype results in a deep purple pigmentation. In addition, following 24 hours of illumination, the colorless scutellum of *r-ch(Hopi)* seeds turns red and patches of pigment appear in the aleurone. Anthocyanin production in these seed tissues is a unique feature of this allele.

Plants of *R-st o7/r-ch O7* genotype crossed by *r-g o7* males yielded ears showing the following segregation:

| | Stippled | | Colorless | |
|---|---|---|---|---|
| No. kernels | + | *o7* | + | *o7* |
| 4489 | 81 | 1676 | 2709 | 23 |

These results differ from expectation in two respects: (1) the *R-O7* recombination amounts to 2.3%, a value much lower than the expected 26%; and (2) colorless kernels are found in the progeny of the testcross in excess of the expected value (61 vs. 50%), indicating a preferential transmission of the *r-ch* marked chromosome. Decrease of recombination in the *R-O7* region and preferential segregation are observations consistent with an abnormal structure of the *r-ch* chromosome.

The "cherry" phenotype conditioned by *r-ch(Hopi)* could be accounted for by a genetic determiner found in some *r* accessions extracted from Bolivian populations (Gavazzi et al., 1985, in press) lying 1-2 units *R* distal and symbolized *Sn* (red scutellar node). The female parents of the previous cross would then be designated *R-st sn o7/r-g Sn O7*, and evidence in favour of the component structure of *r-ch* would be searched for among the stippled kernels in the progeny of these females crossed with *r-g sn o7* males. Upon germination in the presence of light, parental stippled seeds would remain colorless in the scutellum tissues, while recombinants with an *R-st Sn* chromosome would quickly turn red. Accordingly, stippled kernels were germinated 48 hours in darkness, then transferred to light (20,000 lux) and scored 24 hours later for scutellum pigmentation.

The following results were obtained:

| Stippled kernels | + | | *o7* | |
|---|---|---|---|---|
| | r[1] | nr | r | nr |
| 1752 | 29 | 47 | 3 | 1673 |

[1]r and nr refer to red and nonred scutellum as determined after 72 hours of germination

None of the 23 presumed *R-st sn* recombinants so far tested could be confirmed, all of them being *R-st/r-ch* contaminants.

Such negative results cannot be taken as evidence against the compound structure of *r-ch(Hopi)* considering that *Sn* lies 1-2 units *R* distal and that recombination in the *R-O7* region appears dramatically reduced in the *r-ch* bearing chromosome.

Further testing is required to prove that the "cherry" phenotype of this *r* allele is due to *Sn*.

G. Gavazzi, M. L. Racchi and A. Gorrini

**The effect of light on pigment accumulation in germinating *r-ch(Hopi)* seeds**

As outlined in the previous note, a characteristic feature of *r-ch(Hopi)* is its light dependent potential to accumulate anthocyanin in the scutellum and aleurone tissues of germinating seeds. A 24-hour period of illumination is in fact sufficient to allow complete pigmentation of the scutellum and the appearance of patches of pigment in the aleurone, similar to those exhibited by the light-inducible *c-p* allele of *c1*. To ascertain whether germination plays any role in this light mediated process, immature seeds (35 days after pollination) were placed in agar, embryos up, in plastic petri dishes as whole seed or as embryo together with its scutellum. Following 48 hours darkness they were exposed to light for another 48 hours and then scored for scutellum pigmentation. Under these conditions only embryos devoid of their endosperm germinate and accumulate anthocyanin in their scutellar tissues, while whole seeds neither germinate nor develop pigment. These results indicate that both light and germination are effective in triggering biochemical processes leading to pigment accumulation.

To see whether light quality affects the *r-ch(Hopi)* expression, homozygous *r-ch* seeds plated on wet filter paper in petri dishes were irradiated with continuous light of different qualities for 36 hours, and anthocyanins extracted from their scutella in a 1% HCl ethanol solution at the end of the irradiation time. Lamps Power stars HQ1-T400 W/DV OSRAM were the light source. The different light qualities obtained with gelatin or dielectric layer filters had fluence rates of 68, 19, 34 and 4.3 W $m^{-2}$ for white, blue, red and far red light, respectively. The pigment content (A 530/scutellum) following irradiation with continuous light of different qualities is given below:

| Dark | White | Blue | Red | Far Red |
|---|---|---|---|---|
| 0.11 | 0.64 | 0.49 | 0.21 | 0.22 |

Each value is the average of the determination of two samples of five scutella each. These results suggest the involvement of at least two photoreceptors in mediating the light effects, one active in the red, far red and another one in the blue portion of the spectrum.

A similar interaction with light is observed in the presence of *Sn* (unpublished results), thus suggesting that the seed pigmentation conditioned by *r-ch(Hopi)* might represent the response of a modified *Sn* differing from standard *Sn* in its capacity to be expressed in the mature seed tissues following germination.

M. L. Racchi, G. Gavazzi and A. Gorrini

### Extent of gene expression in pollen

The evaluation of the extent of gametophytic gene expression and of the type of transcription (haploid or haplo-diploid) of the genes, has been continued. The study was performed using isozymes as genetic markers; the determination of the type of expression of genes coding for multimeric enzymes was based on comparison of electrophoretic pattern of pollen and of sporophytic tissues from plants heterozygous for electrophoretic mobility (M. Sari Gorla et al., MGCNL 58:145-146, 1984).

During 1984, ACP-2, CAT-1, CAT-2, CAT-3, ENP, GOT-2, GOT-3, IDH-1, IDH-2, PGM-1, PGM-2, 6-PGD-1, 6-PGD-2, SOD and INVERTASE were included in the analysis. Some enzymes present in pollen could not be tested for haploid expression because they were monomeric forms (ENP, PGM-1, PGM-2), or proved to be invariant (GOT-3, SOD). The others gave the following results.

For ACP-2, one band was observed in parental sporophytic genotypes and two in the hybrid, thus suggesting a monomeric structure of the enzyme; the pollen produced two and four bands, respectively, which reflected the mobility of the sporophytic bands. This concordance was confirmed in about twenty different genotypes analyzed and indicates that the same gene is active in both phases; the different processing of the enzyme in pollen and sporophyte points to haploid expression.

CAT-1, CAT-2 and CAT-3 are expressed in different tissues and developmental stages. Only one form of catalase is present in pollen, probably CAT-1; in fact, a mobility concordance between pollen and one-day-old scutellum variants was observed; moreover, plants heterozygous for CAT-1 and homozygous for CAT-2 and CAT-3 showed two bands in pollen, while pollen from plants heterozygous for CAT-2 or CAT-3 and homozygous for CAT-1 revealed but one activity band.

GOT-2 formed three bands in the extracts from F/S scutellum and only two in pollen from the same plants: it is a dimer with haplo-diploid genetic control.

IDH-1 and IDH-2 gave the same type of zymogram as GOT-2.

Two enzymatic forms of 6-PGD are present in the sporophyte and only one (6-PGD-2) in the gametophyte; pollen from heterozygous 6-PGD-2 F/S plants produced only two parental bands, thus indicating haploid expression of the gene.

Finally, invertase showed a completely different electrophoretic pattern from gametophytic and sporophytic extracts, suggesting that different genes are active in the two phases.

M. Sari Gorla, C. Frova, G. Binelli
and E. Ottaviano

MORGANTOWN, WEST VIRGINIA
West Virginia University

### A comparison of methods for isolation of maize seedling nuclei

During our work with maize chromatin, several existing methods for the isolation of plant nuclei have been employed. These methods have been optimized for other plant species and/or tissues. We have found most to be unsuitable for etiolated maize shoots, for nuclear fractions did not meet the criteria for purity and yields obtained with the original tissue or plant. An early method which isolates a nuclear-chromatin fraction (Bonner, J., *et al.*, Methods Enzymol. 12B:3-65, 1968) yielded few intact nuclei despite high yields of nucleic acids and chromatin proteins, and thus allowed the possibility of cytoplasmic contamination. Grinding of maize shoots with a mortar and pestle in a solution containing glycerol, sucrose, KCl, and tris buffer, pH 7.0 (Stern, H., Methods Enzymol. 12B:100-112, 1968), gave a clean preparation of intact nuclei, but the yields were low and the time required was excessive. The popular method of Y. M. Chen *et al.* (Plant Physiol. 56:78-82, 1975) employs homogenization of tissue in a solution including glycerol, sucrose, KCl, $MgCl_2$ in MES buffer (pH 6.0), followed by centrifugation through dense buffered sucrose. This technique resulted in extremely low yields of maize shoot nuclei. The method of R. H. Hamilton *et al.* (Anal. Biochem. 49:48-57, 1972) involves rinsing the tissue in ether for 30 sec., followed by homogenization in a Waring blendor in sucrose, $MgCl_2$, and tris, pH 7.6. High yields of a nuclear fraction were obtained from maize, but the nuclei were severely disrupted.

Various homogenization and filtration techniques were tried with each of the above methods. In general, certain trends were consistently observed with maize seedlings, regardless of the media and method used. The Waring blendor caused severe fragmentation of nuclei in all situations. Homogenization via the Sorvall Omni-Mixer increased the number of intact nuclei in the preparation. Best results were consistently obtained by the use of the VirTis "45" homogenizer. Filtration of the resulting slurry was best accomplished by the use of cheesecloth and miracloth (Chen *et al.*, 1975) rather than mesh or screens; this gave high yields with less contamination.

Methods employing gum arabic in the isolation media gave improved results with maize shoots. A recent method (Stout, J. T. and C. K. Hurley, Methods Cell Biol. 16:87-96, 1977) gave higher yields of chromatin material, but fragmented nuclei and contaminating debris were abundant. The method of K. J. Tautvydas (Plant Physiol. 47:499-503, 1971) gave superior results when reduced-volume gum arabic gradients were used with the original 14-hour incu-

bation period; high nuclear yields were obtained and low cytoplasmic contamination was observed. Activities of endogenous RNA polymerases remained intact (Nebiolo, C. M. and V. Ulrich, Fed. Proc. 41:1294, 1982).

To avoid the proteolysis of nuclear proteins, however, a shorter incubation period was desired. We have developed a modification of the Tautvydas method which is optimized for etiolated maize shoots (to be published). Briefly, 72-hr. shoots are harvested and placed on ice; all operations are performed at 0-4 C. Proteolysis of histones and nonhistone proteins is prevented by the inclusion of 1 mM phenylmethylsulfonyl fluoride and 1 μg/ml soybean trypsin inhibitor (TI) in all solutions. 5.5 gm of tissue are minced and rinsed with diethyl ether for only 1-3 sec. (prolonged exposure causes severe nuclear damage). The tissue is vacuum infiltrated (4 min., twice) in 20 ml of Buffer A, or 4% gum arabic (w/v) in Buffer B: 0.15 M sucrose, 4 mM magnesium acetate, 5 mM 2-mercaptoethanol, 0.1% octanol (v/v), and 5 mM MES (2-[N-morpholino] ethanesulfonic acid), pH 6.05. Following storage in the dark for 3 hr., the suspension is sheared for 8 sec. at medium speed with a VirTis "45" homogenizer and filtered through 4 layers of cheesecloth and one of miracloth.

The filtrate is layered over 4 step-gradients, each constructed of 12 ml layers of 12%, 10%, and 8% gum arabic in buffer B. These are centrifuged for 12 min. at 900 g in a swinging bucket rotor. The cellular debris trapped in the upper layers of the gradient is aspirated, and the nuclear pellets are homogenized in 24 ml of Buffer A by one stroke of a glass-teflon tissue grinder (.13-.18 mm clearance). The suspension is layered over 3 additional gradients and centrifuged as before; the final pellets are resuspended in 30 ml of Buffer B without TI. If desired, 3 lots of tissue (5.5 gm each) can be processed simultaneously, each staggered by only 10 min. to prevent autolysis. Aliquots can be routinely stained with aceto-orcein to monitor purity and quantified on a hemacytometer. This procedure yields 5-7 X $10^7$ nuclei from 17.5 gm of the FRMO17 X FRN28 strain of maize. Examination by EM showed intact nuclei with little cytoplasmic contamination. Subsequent chromatin isolation and analysis of DNA, RNA, histone, and nonhistone content, as well as digestion kinetics with DNase II, show these nuclei to be quite suitable for a variety of biochemical studies.

S. E. Palmer and V. Ulrich

### Distribution of nonhistones, histones, and putative HMG proteins between fractions of maize seedling chromatin

The DNase II, $Mg^{2+}$ procedure (Gottesfeld, J. M., Phil. Trans. R. Soc. Lond. B. 283:343-357, 1978) was used to cleave and separate fractions from maize seedling chromatin. Under gentle conditions of digestion with this nuclease, a fraction with a less compact structure can be isolated from chromatin; the fraction is also enriched in RNA polymerase activity and nascent mRNA (Mathis, D., *et al.*, Prog. Nucleic Acid Res. Mol. Biol. 24:1-55, 1980). This fraction meets all criteria for transcriptionally-active chromatin, and probably corresponds to the euchromatin portions of the genome. Although much is known about fractionated animal chromatin, little has been reported on corresponding fractions from plants.

When 72-hr. maize seedling chromatin (FRMO17 X FRN28 hybrid strain) was digested with DNase II, a longer incubation period (90 min. vs. 15 min.) and a lower DNA concentration (from 370 μg/ml to 75 μg/ml) than required for animal chromatin was necessary to achieve proper digestion kinetics. The amount of $Mg^{2+}$-soluble "euchromatin" released, at 14%, is comparable to animal chromatin fractions.

The distribution of chromatin proteins in fractionated animal chromatin has been well characterized, but data on plants are lacking. In the present study, total chromatin proteins in each fraction were dissociated with SDS, then reduced with 2-mercaptoethanol prior to electrophoresis on 12% polyacrylamide SDS slab gels. Molecular weights (MW) were determined from histones and other standard proteins. Positions of maize histones were confirmed by prior extraction with 0.4 N $H_2SO_4$. The results are shown in Table 1. Histones H1, H2A,

Table 1. Summary of proteins found in fractionated maize chromatin

| Basic Proteins (found in both fractions) | | MW |
|---|---|---|
| Histones | H1 subfractions | 28,000-35,000 |
| | H2A | 18,000 |
| | H2B variants | 18,000<br>14,000 |
| | H3 | 13,500 |
| | H4 | 11,000 |
| Putative HMG proteins | | 19,500<br>15,000 |
| **Acidic Proteins** | | **MW range** |
| NHCP of $Mg^{2+}$-soluble euchromatin fraction | | 43,000-68,000<br>38,000<br>27,000-28,000<br>25,000 |
| NHCP of $Mg^{2+}$-insoluble and digestion-resistant heterochromatin fraction | | 35,000-62,000<br>27,000<br>18,000-25,000 |

H2B, H3, and H4 were present in both euchromatin and heterochromatin fractions, but each had common and unique H1 subfractions. Also found were low MW nonhistone proteins (NHCP) in the MW range of 15,000-20,000. To determine the existence of HMG proteins in this range, chromatin was extracted by standard protocol (Spiker S. *et al.*, PNAS 80:815-819, 1983). Two such proteins were found at 19,000 and

15,000 MW, respectively, in each fraction; on the basis of MW, extraction in 0.35 M NaCl, and solubility in 2% trichloroacetic acid (w/v), it is possible that these nonhistones correspond to HMG proteins.

Each fraction contained distinct electrophoretic NHCP patterns. Table 1 indicates the various MW range positions of NHCP in euchromatin and heterochromatin fractions; individual proteins are not listed. Relatively few proteins were common to both fractions. Heterochromatin contained over 15 distinct NHCP bands and various fainter ones, while the other fraction contained fewer. However, the NHCP are the predominant proteins in the $Mg^{2+}$-soluble fraction.

It appears that NHCP and histones are distributed among maize chromatin fractions in a manner similar to those of animal chromatin, as reflected by the variety of unique NHCP as well as unique H1 subfractions in each chromatin fraction. However, HMG proteins are enriched in animal $Mg^{2+}$-soluble chromatin, but this was not observed with maize chromatin.

S. E. Palmer and V. Ulrich

## Comparisons of chromatin conformation and compositions of heterotic hybrid and parental inbred maize

The DNase II, $Mg^{2+}$ procedure was used to fractionate chromatins of a heterotic maize hybrid and parental inbred seedlings. DNase II has been reported to digest DNA at open regions where transcription is occurring, *i.e.*, "euchromatin". Therefore the $Mg^{2+}$-soluble fraction released by this procedure is thought to be a suitable model for the euchromatin portion of the genome, both in quantity released (a measure of chromatin condensation via nuclease accessibility) and in the quantitative and qualitative analysis of chromatin components. The small $Mg^{2+}$-insoluble fraction released can be pooled with the considerable digestion-resistant portion of the genome to serve as a model for heterochromatin.

Table 1 shows the results for hybrid and inbred chromatins after 90 min. of digestion. In terms of heterosis, the results are difficult to interpret due to the absence of strong correlations with the quantity of DNA in the hybrid fractions. The amount of $Mg^{2+}$-soluble "euchromatin" released from hybrid chromatin was intermediate to those of the inbreds, and was similar to that of FRN28, the more vigorous inbred. This does not strongly support a model of heterosis by a mechanism of chromatin conformation, but may do so on the basis of greater transcriptional and regulatory efficiency of the hybrid. This may be supported by data in Table 1, which also shows that the hybrid putative "euchromatin" fraction contains 1) relatively more protein and RNA than DNA than the inbreds; 2) higher proportions of protein and RNA than unfractionated hybrid chromatin; and 3) relatively larger proportions of protein and RNA than the inbred "euchromatin" fractions. Therefore, this procedure isolated maize chromatin fractions that are distinct in composition. Furthermore, the hybrid $Mg^{2+}$-soluble fraction has unique features which distinguish it from chromatins of the parental inbreds.

Table I. DNA:Protein:RNA ratios of chromatin fractions from parental maize inbred and F1 heterotic hybrid

| Strain | Chromatin fraction | % DNA as euchromatin | Ratios to DNA DNA:Protein:RNA |
|---|---|---|---|
| FRMO17 X FRN28 | Unfractionated | | 1.0 : 4.7 : 1.7 |
| | $Mg^{2+}$-soluble | 14 ± 1.5 | 1.0 : 6.1 : 3.1 |
| | Digestion-resistant | | 1.0 : 4.7 : 1.6 |
| FRMO17 | Unfractionated | | 1.0 : 6.2 : 3.3 |
| | $Mg^{2+}$-soluble | 30 ± 3.8 | 1.0 : 0.6 : 1.0 |
| | Digestion-resistant | | 1.0 : 9.0 : 4.3 |
| FRN28 | Unfractionated | | 1.0 : 6.7 : 1.6 |
| | $Mg^{2+}$-soluble | 11 ± 0.5 | 1.0 : 1.7 : 1.6 |
| | Digestion-resistant | | 1.0 : 6.5 : 1.4 |

S. E. Palmer and V. Ulrich

NEW DELHI, INDIA
Indian Agricultural Research Institute

## Maximizing grain yield

Maize, in spite of being one of the first cereal crops that attained the advantage of the phenomenon of heterosis, is not known to give higher grain yield than crops like wheat and rice. Considerable work has been carried out with varied success in the development of promising inter-varietal or inter-racial hybrids, yet very few hybrids are known in the tropical and sub-tropical regions which give higher *per se* performance. To maximize the mean performance, it is imperative that the base germplasm complexes be developed from genetically diverse sources so that intra-population heterosis may aid the inter-population heterosis and boost the magnitude further.

With this in mind, we selected three newly developed, genetically broad-based populations (JML 305, JML 24 and Early Yellow Composite) and three exotic varieties (Caribbean Flint, St. Croix 4D and Antigua Gr.1) from different geographical regions, and made 15 $F_1$ (without reciprocal) crosses. Data were recorded for ear length, number of grains per ear, number of grains per row, 100 grain weight and grain yield per plant.

It was interesting to note that the populations gave a better performance over the varieties with regard to grain yield and other characters. The population x population type of cross gave the highest grain yield, followed by the other two types, namely, population x variety and variety x variety, in that order (Table 1). For other characters also it was found that, with the exception of ear length, the

Table 1. Mean performance of parents and their crosses.

| Character | Parents | | Crosses ($F_1$'s) | | | Critical Difference | |
|---|---|---|---|---|---|---|---|
| | P | V | P x P | P x V | V x V | at 5% | at 1% |
| Ear length (cm) | 14.91 | 14.27 | 15.82 (6.10**) | 14.87 (1.92) | 15.88 (11.28*) | 1.37 | 1.62 |
| No.of grains/ear | 445.83 | 399.00 | 522.00 (17.08**) | 437.00 (3.45) | 429.40 (7.61**) | 64.42 | 76.14 |
| No.of grains/row | 31.00 | 30.31 | 33.98 (9.61) | 31.00 (1.13) | 31.66 (4.46) | 3.51 | 4.17 |
| 100 grain wt (g) | 23.38 | 17.83 | 24.61 (5.26) | 24.19 (17.39) | 22.04 (23.61) | 6.35 | 7.72 |
| Grain yield/plant (g) | 97.58 | 76.55 | 122.83 (25.88**) | 107.22 (23.15**) | 95.50 (24.75**) | 15.61 | 18.76 |

P = Population, V = Variety, Figures in parenthesis indicate mean heterosis per cent

* Significant at 5%, ** Significant at 1%

Table 2. Best combiners in different types of crosses

| Type of cross | Best combiner | Ear length (cm) | No. of grains/ear | No. of grains/row | 100 grain wt (g) | Grain yield (kg/ha) |
|---|---|---|---|---|---|---|
| P x P | JML24 x Early Yellow Composite | 16.50 | 497.00 | 34.00 | 26.64 | 3457 (130.50) |
| P x V | JML305 x St.Croix 4D | 13.92 | 445.80 | 31.50 | 25.42 | 3247 (122.60) |
| V x V | Caribbean Flint x Antigua Gr.1 | 15.02 | 428.60 | 31.25 | 23.30 | 2929 (110.60) |
| Best Check (E. H. 2310) | | 15.40 | 442.00 | 32.00 | 21.62 | 2649 (100.00) |

P = Population, V = Variety

population x population type of cross was superior to the other two types. Regarding the performance of individual crosses it was found that JML 24 x Early Yellow Composite (a population x population cross) gave the highest grain yield, and out-yielded the best check by 30.50 percent (Table 2).

We therefore argue that higher *per se* performance and heterotic expression in the population x population crosses may have resulted from intra-population heterosis already present (due to diverse gene combinations) in the populations. This also indicates that the low heterosis and *per se* performance in the inter-varietal crosses might have been due to restricted recombination of variability in the $F_1$ generation.

B. C. Saha and B. K. Mukherjee

NEW HAVEN, CONNECTICUT
Yale University

**Plant regeneration from tissue culture**

Methodology for regeneration of complete plants from maize tissue cultures of immature embryos was established by Green & Phillips (Crop Sci. 15:417-421, 1975). A report by Harms, Lorz & Potrykus (Z. Pflanzenzucht. 77:237-259, 1976) describes regeneration from tissue culture using 4-day seedlings. We wish to report regeneration of plants from maize tissue cultures obtained from 14-day seedlings.

Cultures were initiated from 14-day seedlings of the A188 inbred line. Seed was germinated and plants were grown in test tubes (2.5 x 10 cm) on White's medium for 4 days in the dark at 27 C, then in daylight for 10 days more. About 2-3 mm cross sections of stem, between and including the mesocotyl and the apical meristem, were placed basal side down on an agar-solidified culture medium in plastic petri dishes (100 x 25 mm) prepared as described by Green & Phillips (1975) and containing 2 mg/l of 2,4-dichlorophenoxyacetic acid (2,4-D). The explants were incubated in the dark at 27 C for one month, during which time callus formed. The colorless callus was subcultured on the same medium in the dark for 5 days and then in the light as previously described (Gordon, Robarts & Rice, MGCNL 51:79-80) for regeneration of maize tissue cultures. In a few

days, areas of the callus greened, and within 6 days leaf primordia could be observed. Subcultures were made onto the same medium for 14 days, then onto a medium with 0.5 mg/l 2,4-D. Shoots began to appear after 9 days. The shoots were transferred to plates without 2,4-D until roots were observed (after approximately 14 days). Plantlets (1 cm high) were placed onto the same medium in storage jars (100 x 80 mm) for 3 weeks or until the plantlets were 8-10 cm high. They were then transferred to Jiffy peat pellets and subsequently to soil in the greenhouse.

The tissue culture "clone" is similar morphologically to the "clones" obtained from immature embryo culture and is maintained on the basic medium with 1 mg/l 2,4-D. Twenty plantlets and 5 plants have been obtained from this particular "clone" (Work done at Pfizer Central Research, Eastern Point Road, Groton, CT 06340).

Phillip N. Gordon

OAKLAND, CALIFORNIA
Advanced Genetic Sciences

### The genetic location of *vp1*

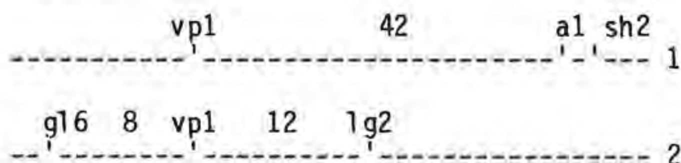
*vp1* is an interesting pleiotropic mutation which, besides causing premature germination of the embryo, fails to synthesize anthocyanin pigments in the aleurone. Anthocyaninless *vp1* aleurones have been found to be deficient in at least 3 enzymes of flavonoid biosynthesis (PAL, chalcone synthase and UFGT), as well as in several other metabolically unrelated enzymes that show pronounced increases in late stages of aleurone development, such as ADH and catalase (Dooner, Plant Physiol., in press). Though *vp1* has been the subject of rather extensive physiological studies, we only know its location to chromosome arm. Its precise genetic location has not been established, mostly because of the difficulty of maintaining homozygous *vp1* stocks, which have to be grown continuously due to the absence of dormancy in the mutant stock. However, Robertson has described a *vp1* allele extracted from a stock of Ken McWhirter that shows considerable dormancy, making it possible to maintain a homozygous *vp1* stock (MNL 35:104-105, 1965). We have taken advantage of the existence of this allele to map *vp1*.

Two sets of crosses were performed in order to map *vp1*. The first set of crosses was aimed at obtaining the approximate location of *vp1* in 3L. The heterozygote *(Vp1) a1 sh2/ (vp1) A1 Sh2* was backcrossed to *(Vp1) a1 sh2*, and from that cross the infrequent crossovers *A1 sh2* and *a1 Sh2* were selected and selfed in order to score *vp1*. Both *A1 sh2* individuals segregated *vp1;* of the two *a1 Sh2* individuals, one was *Vp1/Vp1* and the other one segregated *vp1*. On this basis, *vp1* was placed proximal to the *a1 sh2* pair in 3L. A random sample of parental *a1 sh2* and *A1 Sh2* types from the above cross were also planted and selfed to classify their *vp1* constitution. The following classes were obtained: 37 *Vp1 a1 sh2;* 28 *vp1 A1 Sh2;* 22 *vp1 a1 sh2* and 25 *Vp1 A1 Sh2*. Thus, there is very loose linkage between *vp1* and the *a sh2* pair. From the above data, *vp1* would map 42 map units proximal to the *a1 sh2* pair.

Based on the above results, a second three-point cross was set up as follows. *gl6 (Vp1) lg2/ Gl6 (vp1) Lg2* heterozygotes were backcrossed to *gl6 (Vp1) lg2*. Progeny from the backcross were scored for *gl6* and *lg2* and selfed to classify *vp1*. They fell into the following classes:

| | |
|---|---|
| *gl6 Vp1 lg2* | 31 |
| *Gl6 vp1 Lg2* | 36 |
| *Gl6 Vp1 lg2* | 3 |
| *gl6 vp1 Lg2* | 4 |
| *Gl6 vp1 lg2* | 3 |
| *gl6 Vp1 Lg2* | 7 |

From the above results the following maps can be drawn:




A comparison of distances in the distal (right-hand) side of *vp1* reveals reasonable agreement with the current linkage map for the *lg2-a1* interval, and places *vp1* between *gl6* and *lg2*.

Hugo K. Dooner

ONGOLE, ANDHRA PRADESH, INDIA
C. S. R. Sarma College

### A new orcein technique for mitotic chromosome analysis

In our laboratory several chemicals were tested for analysing the somatic chromosome morphology of maize. The following pretreatment and staining schedule was found to yield satisfactory preparations.

The seeds were allowed to germinate on moist filter paper in petri plates. The primary and secondary roots were collected when they were about 1.0 - 2.0 cm in length. Both types of roots gave equally good preparations by slightly varying the maceration timings. The optimum time of collection was between 12:00 P.M. and 1:00 P.M. in summer months. The root tips were pretreated with an aqueous solution (0.2 percent) of calcium chloride for 2 hrs. at 10-12 C. This included one minute chilling time in the freezer chest. The concentration of $CaCl_2$ solution and the duration of pretreatment were very critical. After pretreatment, the thoroughly washed

root tips were fixed in 1:3 acetic ethanol for overnight and preserved in 70 percent ethanol. They were macerated in a 5 percent aqueous solution of pectinase for 2 hrs. at 37 C and then cleared in 45 percent acetic acid for 10 minutes. They were stained in 2 percent aceto-orcein overnight and squashed gently. The resultant preparation was well scattered with proper condensation of chromosomes, followed by spindle dissolution, centromere exaggeration and metaphase arrest.

Calcium chloride exists in the form of $Ca^{++}$ and $Cl^-$ ions in solution. The treatment with an aqueous solution of $CaCl_2$ causes a change in the ionic environment and upsets the ionic balance, resulting in viscosity change and spindle dissolution. $CaCl_2$ is a well known dehydrating agent. It is possible that DNA-histone binding is enhanced by differential (unilateral) dehydration, resulting in chromosome condensation and clarification of chromosome morphology (This work was carried out under the supervision of Prof. A. K. Sharma, at the Department of Botany, University of Calcutta, Calcutta, India).

J. S. P. Sarma

PHILADELPHIA, PENNSYLVANIA
University of Pennsylvania

### Dosage analysis of *Tp1* and *Tp2*

The nature of the dominant mutations *Tp1* and *Tp2* was studied by comparing the expression of these mutations in the presence of different numbers of copies of their wildtype alleles. Plants of the genotype *Tp2 g r-/Tp2 g r-* and *Tp1 gl1/+ +* were crossed, respectively, by plants carrying the B-A translocations TB-10L19 and TB-7Lb. Hyperploid, hypoploid and diploid progeny were identified on the basis of kernel *(R-scm)* or seedling markers *(g* and *gl1)*, or by chromosome number. Hyperploid progeny from both crosses were clearly mutant in phenotype. This demonstrates that these mutations involve a gain-of-function and confirms an observation made earlier in the case of *Tp2* (Poethig, MNL 57:35, 1983). In the case of *Tp2*, all 3 dose classes (i.e. *Tp2/+/+*, *Tp2/+*, *Tp2/-)* had an identical phenotype. Thus *Tp2* is either a constitutive mutation, or a mutation that causes $Tp2^+$ to be expressed at greater than twice its normal level. In contrast, *Tp1/-* individuals were more severely mutant than *Tp1/+/+* individuals, suggesting that this mutation in some way antagonizes normal gene activity.

S. Poethig

### The location of *cl*

*cl* was originally mapped relative to *a*, *lg2* and *ts4* by Robertson (MNL 33:63, 1959). These data place *cl* between *Lg3* and *d* on the short arm of chromosome 3. Recent 2-point crosses between *cl* and *d*, and *cl* and *Lg3*, appear to confirm this location. The results shown in Table 1 indicate that *cl* is about 17 map units from *d*, while the data in Table 2 indicate that *cl* is about 5 map units from *Lg3*. Given that *Lg3* and *d* are 25 map units apart (MNL 58:255, 1984), these data confirm the current location of *cl*. The results of a 3-point test cross will be scored this summer.

Table 1: Progeny from the cross + +/cl d; Clm4/clm4 X cl d; Clm4

| | Parentals | | Recombinants | | Total |
|---|---|---|---|---|---|
| | + + | cl d | + d | cl + | |
| + +/cl d | 208 | 197 | 37 | 48 | |
| Subtotals | 405 | | 85 | | 490 |
| % Recombination | | | 17.3 ± 1.7 | | |

Table 2: Progeny from the cross + cl; Clm3 X Lg3 +/+ cl; Clm3/clm3

| | Parentals | | Recombinants | | Total |
|---|---|---|---|---|---|
| | Lg3 + | + cl | Lg3 cl | + cl | |
| Lg3 +/+ cl | 341 | 288 | 17 | 19 | |
| Subtotals | 629 | | 36 | | 665 |
| % Recombination | | | 5.4 ± 0.9 | | |

S. Poethig

### The location of Esterase-8

Esterase 8 *(E8)* was mapped relative to *d* and *cl* using a slow allele obtained from M. Goodman (KN *E8-S)*. The *d cl; Clm4* stock used for this analysis carried a faster allele *(E8-F)*, whose exact identity is unknown. Preliminary results indicate that *E8* is located at the distal end of 3S, about 30 map units from *d* (Table 1). This puts *E8* in the vicinity of *g2* (R. Whalen, pers. communication), so the relative position of these 2 markers will now have to be determined.

Table 1: Progeny from the cross + + E8-S/cl d E8-F; Clm4/clm4 X cl d E8-F; Clm4

| | Parentals | | Region 1 | | Region2 | | Regions 1 & 2 | | Total |
|---|---|---|---|---|---|---|---|---|---|
| | + + S | cl d F | + d F | cl + S | + + F | cl d S | + d S | cl + F | |
| + + E8-S/cl d E8-F | 26 | 20 | 3 | 5 | 9 | 12 | 1 | 1 | |
| Subtotals | 46 | | 8 | | 21 | | 2 | | 77 |
| % Recombination | | | 12.9 ± 3.8 | | 29.9 ± 5.2 | | | | |

J. Sorrentino and S. Poethig

PRESCOTT, WISCONSIN
Jacques Seed Company
JOHNSTON, IOWA
Pioneer Hi-Bred International, Inc.

**Three new male-sterile genes**

While working to develop a nuclear male-sterility system for hybrid seed corn production (Phillips, MNL 49:118 and 52:67; West, M.S., Univ. Minnesota, 1984), three nonallelic, recessive, nuclear male-sterile, female-fertile mutants were recovered. Seed samples have been deposited with the Maize Genetics Stock Center. These mutants have received the designations *ms22-A632A*, *ms23-A619B*, and *ms24-LT*.

These new male-sterile mutants were compared cytologically with the known male-steriles as described by Beadle (Genetics 17:413, 1932) and Albertsen and Phillips (Can. J. Genet. Cytol. 23:195, 1981). Microspore mother cells (MMC's) of *ms22-A632A* degenerate prior to pachynema. MMC cell walls appear extremely fragile before degeneration and may be the site of the mutant's primary effect. Allelism tests were conducted with *ms8*, *ms9*, and *ms17* because these male steriles had been described previously as exhibiting early MMC breakdown. There were no sterile plants in any of the $F_1$ progenies of the crosses with *ms8* (72 plants), *ms9* (62 plants), or *ms17* (17 plants).

Degeneration of *ms23-A619B* is also during meiosis I, although intact prophase II configurations have been found. We sometimes have observed that the tapetal cells lose staining intensity before the MMC's exhibit cellular disorganization. Whether this is a cause or effect has not yet been determined. As with *ms22-A632A*, MMC cell walls appear fragile. The nuclear events of meiosis continue, however, until meiosis II. Because this pattern of microsporogenesis breakdown is similar to that described for *ms8*, we made the appropriate testcross, but found no sterile plants among 38 $F_1$ progeny. We did find, however, *ms23-A619B* to be allelic with a previously undescribed male sterile *(ms*-Bear7)* from Earl Patterson (Univ. Illinois) by testcrossing a homozygous recessive *ms*-Bear7* plant with a known *ms23-A619B* heterozygote. The $F_1$ progeny segregated 8 fertiles:11 steriles ($X^2=0.47$; $.90 \leq P \leq .75$), indicating the same locus but from independent mutations. We previously had testcrossed *ms*-Bear7* plants with *ms1* (20 plants), *ms2* (18 plants), *ms5* (13 plants), *ms7* (21 plants), *ms8* (21 plants), *ms9* (21 plants), *ms10* (17 plants), *ms11* (20 plants), *ms12* (17 plants), *ms13* (19 plants), *ms17* (22 plants), *ms20* (21 plants) and *po* (39 plants) and observed that all progeny were fertile. Relationship of *ms22-A632A* with *ms23-A619B* was tested by observing $F_1$ and $F_2$ progenies. Forty-three $F_1$ plants were completely fertile. The selfed progeny segregated 414 fertile plants:315 sterile plants, a good 9:7 fit ($X^2=0.02$; $.90 \leq P \leq .75$) for two different loci.

Homozygous recessive mutant *ms24-LT* extrudes anthers and exhibits occasional engorged pollen grains among degenerated pollen grains when the mature anther locule is viewed under low power. No effective pollinations were achieved, however, when homozygous recessive *ms24-LT* plants were used as males. $I_2$-KI staining also revealed irregular starch accumulation in pollen ranging from undetectable to fully engorged. This suggests that *ms24-LT* may be involved in the inhibition of pollen starch synthesis or deposition. Germinability of these engorged pollen grains has not been examined *in vitro*. Migration of vegetative and generative nuclei also appears irregular. The generative nucleus eventually degenerates. This male sterile appears to degenerate later than *ms5*, *ms11*, or *ms14*, which had been previously described as being late-degenerating mutants. We found it to be nonallelic to *ms14* (25 $F_1$ plants examined). It has not yet been testcrossed with *ms5* and *ms11*.

New male-sterile mutants *ms22-A632A* and *ms24-LT* constitute the earliest and latest, respectively, degenerating male-sterile mutants thus far described. Male-sterile mutant *ms23-A619B* is cytologically, but not genetically, similar to *ms8*. An independent mutation for the same locus has occurred in another previously unknown male sterile, *ms*-Bear7*. Mutant *ms24-LT*, because it appears to proceed nearly to the completion of pollen development, might be induced to complete microsporogenesis and succeed to fertility by an appropriate chemical agent or environmental condition, as has been suggested previously for *ms14* (Albertsen, Ph.D., Univ. Minnesota, 1980).

David P. West and Marc C. Albertsen

RALEIGH, NORTH CAROLINA
North Carolina State University

**Twelve new isozyme loci in maize: Progress report on chromosomal locations, the subunit composition, and subcellular localization of their products**

To improve the degree to which the corn genome is biochemically marked, we have expanded the number of enzyme systems analyzed by starch gel electrophoresis. Seven new enzyme systems encoded by a minimum of 16 loci have been added to the 23 loci used for previous work in our laboratory. A wide range of domestic and exotic maize has been screened for variation at these new loci and genetic analysis of variant electromorphs is well under way. Crosses with B-A translocation stocks (kindly provided by Jack Beckett), previously mapped allozyme loci, and in a few cases morphological markers, are being studied in order to elucidate the chromosomal locations of these new loci. Organelle extractions have been helpful in determining the subcellular location

Table 1. New isozyme loci in maize, subunit composition and subcellular localization.

| Enzyme | Quaternary Structure | Locus | Subcellular Compartment | Chrom |
|---|---|---|---|---|
| Aconitase | monomer | Aco1 | cytosol? | 4S |
| | ? | Aco2 | mitochondria? | ? |
| | ? | Aco3 | mitochondria? | ? |
| | monomer | Aco4 | mitochondria? | ? |
| Adenylate kinase | monomer | Adk1 | plastids | ? |
| Aminopeptidase* | monomer | Amp1 | cytosol | 1L |
| | monomer | Amp3 | cytosol | 5S |
| Diaphorase | monomer | Dia1 | cytosol | 2S |
| | dimer | Dia2 | cytosol | 1L |
| Hexokinase | ? | Hex1 | cytosol | 3S |
| | monomer | Hex2 | cytosol | 6L |
| Shikimate dehydrogenase | monomer | Sad1 | plastids | 10 |
| Triose phosphate isomerase | dimer | Tpi1 | plastids | ? |
| | dimer | Tpi2 | plastids | ? |
| | dimer | Tpi3 | cytosol | 8? |
| | dimer | Tpi4 | cytosol | 3L |

*Reported by Ott and Scandalios (Genetics 89: 137-146).

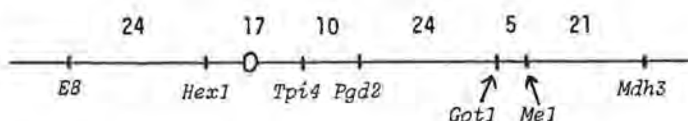
of the encoded products. A summary description of the new loci and their products is presented in Table 1.

J. F. Wendel, C. W. Stuber, and M. M. Goodman

## Localization of two new isozyme loci, *Hex1* and *Tpi4*, to Chromosome 3

Recent work with starch gel electrophoresis of coleoptile extracts has indicated that *Hex1*, the structural locus for the most anodal set of hexokinase isozymes, and *Tpi4*, which encodes the slowest migrating set of triose phosphate isomerase bands, are located on chromosome 3. Their placement relative to previously mapped isozyme loci on this chromosome *(E8, Pgd2, Got1, Me1,* and *Mdh3* - see Goodman and Stuber, 1983, In: Isozymes in plant genetics and breeding, Part B. S. D. Tanksley and T. J. Orton, Eds., Elsevier) was revealed through the analysis of an $F_2$ between the two inbreds CM37 *(E8-4.5, Hex1-null, Tpi4-4, Pgd2-5, Mdh3-16)* and T232 *(E8-4, Hex1-4, Tpi4-null, Pgd2-2.8, Mdh3-18)*. Joint segregation data and maximum likelihood estimates of recombination fractions are presented in Table 1.

These data are consistent with the previously reported relationships among the loci *E8, Pgd2*, and *Mdh3*, and further indicate that *Hex1* and *Tpi4* lie between *E8* and *Pgd2*. The placement of *Hex1* on the short arm of chromosome 3 was confirmed with a cross of the B-A translocation stock TB-3Sb *(Hex1-4)* onto our *Hex1-null* tester (Ky201). Null phenotypes (i.e., hypoploids) were recovered in 5 of 13 plants examined. These data collectively lead to the following isozyme map for chromosome 3:




The discovery of the markers between *E8* and *Pgd2* forces the genetic map to grow by about 12 units *(E8-Pgd2* = 24 + 17 + 10 = 51 vs. older reports of 39, see ref. above).

J. F. Wendel, C. W. Stuber, and M. M. Goodman

Table 1. Two-locus segregation data and maximum likelihood estimates of the recombination fractions (r) between 5 loci on chromosome 3.

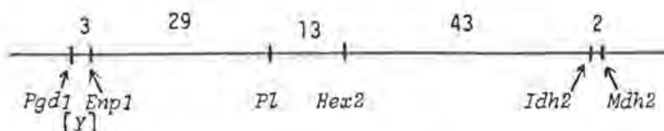
| | | Genotypes* | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Locus pair | n | $X_1Y_1$ | $X_1Y_2$ | $X_1Y_3$ | $X_2Y_1$ | $X_2Y_2$ | $X_2Y_3$ | $X_3Y_1$ | $X_3Y_2$ | $X_3Y_3$ | $X^2$ (DF) | r (se) |
| E8-Hex1 | 1925 | 259 | 234 | - | 160 | 811 | - | 18 | 443 | - | 364.3(2) | .24(.01) |
| E8-Tpi4 | 1928 | - | 409 | 85 | - | 724 | 247 | - | 239 | 224 | 124.2(2) | .36(.01) |
| E8-Pgd2 | 1928 | 168 | 227 | 99 | 212 | 483 | 276 | 66 | 198 | 199 | 88.6(4) | .40(.01) |
| E8-Mdh3 | 1928 | 125 | 241 | 128 | 228 | 488 | 255 | 89 | 242 | 132 | 5.4(4) | n.s. |
| Hex1-Tpi4 | 1925 | - | 422 | 15 | - | 948 | 540 | - | - | - | 177.7(1) | .17(.02) |
| Hex1-Pgd2 | 1925 | 279 | 136 | 22 | 166 | 771 | 551 | - | - | - | 552.5(2) | .20(.01) |
| Hex1-Mdh3 | 1925 | 112 | 225 | 100 | 330 | 744 | 414 | - | - | - | 5.0(2) | n.s. |
| Tpi4-Pgd2 | 1928 | - | - | - | 442 | 819 | 111 | 4 | 89 | 463 | 1078.6(2) | .10(.01) |
| Tpi4-Mdh3 | 1928 | - | - | - | 345 | 680 | 347 | 97 | 291 | 168 | 14.4(2) | .45(.01) |
| Pgd2-Mdh3 | 1928 | 145 | 211 | 90 | 215 | 474 | 219 | 82 | 286 | 206 | 65.8(4) | .41(.01) |

*X and Y refer to the genotypes at the first and second locus listed for each locus pair. The subscripts 1,2, and 3, indicate homozygosity for the CM37 allele, heterozygosity for the CM37 and T232 alleles, and homozygosity for the T232 allele, respectively. Parental genotypes are listed in the text. Two classes of progeny exist for Hex1 and Tpi4, and the subscript 2 indicates the pooled class of heterozygotes with the active variant homozygotes. The 1 or 3 indicates the null variant class.

## Linkage relationships between a new locus, *Hex2*, and previously assigned loci on chromosome 6

Starch gel electrophoresis of maize coleoptile extracts in our laboratory has shown two zones of staining for isozymes of hexokinase. Genetic analyses indicate that the slower migrating set of bands is encoded by the structural gene *Hex2* and that the enzyme is monomeric. Recent linkage analyses and work with B-A translocation stocks (generously supplied by Jack Beckett) have allowed for the placement of *Hex2* on the long arm of chromosome 6. Its location relative to other markers on 6L was determined from the analysis of two very large $F_2$ populations generated for other reasons. One of these (the $F_2$ of inbreds Tx303 and CO159) segregated for 6 loci on 6L (isozyme loci *Pgd1*, *Enp1*, *Hex2*, *Idh2*, *Mdh2*, and color factor *Pl*). The other $F_2$ (from inbreds CM37 and T232) was polymorphic only for *Hex2*, *Idh2* and *Mdh2*. Joint segregation data and maximum likelihood estimates of recombination are presented in Table 1.

The data of Table 1 allow for the following map to be constructed:




This map was generated with the assumptions that *Pl* is located at position 45. The linkage between *Enp1* and *Y* (yellow) remains to be determined. As illustrated, the map has two particularly noteworthy features:

1) *Idh2* and *Mdh2*, which were previously shown to be on 6L (Goodman et al., Genetics 96:697-710) perhaps 12 units from *py* (footnote in McMillin et al., Genetics 92:1241-1250) at approximately position 77, must be moved a minimum of 23 units distal to their previously assigned locations. As a consequence, the genetic map for 6L must be expanded by a third over its prior size.

Table 1. Two-locus segregation data and maximum likelihood estimates of recombination between loci on the long arm of chromosome 6.

| Locus pair | n | Genotypes* $X_1Y_1$ | $X_1Y_2$ | $X_1Y_3$ | $X_2Y_1$ | $X_2Y_2$ | $X_2Y_3$ | $X_3Y_1$ | $X_3Y_2$ | $X_3Y_3$ | $X^2$ (DF) | r (se) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A. $F_2$ from Tx303 and CO159: | | | | | | | | | | | | |
| Pgd1-Enp1 | 1842 | 446 | 17 | 1 | 31 | 880 | 22 | 0 | 28 | 417 | 3117.2(4) | .03(.003) |
| Pgd1-Pl | 1835 | 408 | - | 54 | 738 | - | 190 | 231 | - | 214 | 180.5(2) | .32(.01) |
| Pgd1-Hex2 | 1677 | 157 | 206 | 68 | 204 | 455 | 186 | 52 | 184 | 165 | 110.3(4) | .38(.01) |
| Pgd1-Idh2 | | | | | | independent | | | | | | |
| Pgd1-Mdh2 | | | | | | independent | | | | | | |
| Enp1-Pl | 1835 | 427 | - | 49 | 739 | - | 181 | 211 | - | 228 | 239.0(2) | .29(.01) |
| Enp1-Hex2 | 1677 | 173 | 204 | 61 | 195 | 461 | 184 | 45 | 180 | 174 | 155.7(4) | .35(.01) |
| Enp1-Idh2 | | | | | | independent | | | | | | |
| Enp1-Mdh2 | | | | | | independent | | | | | | |
| Pl-Hex2 | 1674 | 404 | 754 | 93 | - | - | - | 10 | 90 | 323 | 815.6(2) | .13(.01) |
| Pl-Idh2 | | | | | | independent | | | | | | |
| Pl-Mdh2 | | | | | | independent | | | | | | |
| Hex2-Idh2 | 1677 | 125 | 190 | 98 | 211 | 442 | 192 | 65 | 210 | 144 | 37.6(4) | .43(.01) |
| Hex2-Mdh2 | 1677 | 118 | 197 | 98 | 214 | 439 | 192 | 72 | 209 | 138 | 25.4(4) | .45(.01) |
| Idh2-Mdh2 | 1842 | 427 | 18 | 0 | 19 | 888 | 13 | 0 | 22 | 455 | 3266.1(4) | .02(.002) |
| B. $F_2$ of CM37 and T232: | | | | | | | | | | | | |
| Hex2-Idh2 | 1924 | 129 | 210 | 76 | 233 | 512 | 276 | 88 | 229 | 171 | 40.6(4) | .43(.01) |
| Hex2-Mdh2 | 1925 | 119 | 215 | 82 | 233 | 508 | 280 | 95 | 231 | 162 | 24.1(4) | .44(.01) |
| Idh2-Mdh2 | 1927 | 427 | 19 | 5 | 15 | 906 | 32 | 5 | 30 | 488 | 3051.4(4) | .03(.003) |

*Genotypes of the parents are as follows:
Tx303 - Pgd1-2,Enp1-6,Pl,Hex2-2,Idh2-6,Mdh2-3; CO159 - Pgd1-3.8,Enp1-10,pl,Hex2-1,Idh2-4,Mdh2-6; CM37 - Hex2-1,Idh2-6,Mdh2-3.5; T232 - Hex2-2,Idh2-4,Mdh2-6.
X and Y refer to the genotypes at the first and second locus listed for each locus pair. The subscripts 1,2, and 3, indicate homozygosity for the Tx303 (or CM37) allele, heterozygosity for the Tx303 and CO159 (or CM37 and T232) alleles, and homozygosity for the CO159 (or T232) allele, respectively. In the case of Pl, 1 indicates pooled Pl/Pl and Pl/pl genotypes and 3 indicates pl/pl genotypes.

2) The B-A translocation stock TB-6Lc, which uncovers *Y*, also uncovers all isozyme markers on 6L except *Pgd1* (Table 2). Consequently, *Pgd1* must be proximal to the TB-6Lc breakpoint.

Table 2. TB-6Lc uncovers all isozyme markers on 6L with the exception of Pgd1.

| Tester | Locus | Number hypoploid for the tester allele | Number heterozygous for the TB stock and tester alleles |
|---|---|---|---|
| Ky201 | Enp1 | 4 | 8 |
| | Hex2 | 4 | 8 |
| | Idh2 | 4 | 8 |
| | Mdh2 | 4 | 8 |
| T226 | Pgd1 | 0 | 18 |
| | Enp1 | 11 | 7 |

The proper gene order of the two closely linked loci *Idh2* and *Mdh2* cannot unequivocally be decided from the $F_2$ data at hand. The order illustrated must be considered tentative until testcross progenies are evaluated. Stocks have been obtained for the re-examination of the relationship of *Hex2-py-Idh2 -Mdh2* and for the relationships among *Pgd1, Enp1* and *Y*.

J. F. Wendel, C. W. Stuber, and M. M. Goodman

## Mapping data for 34 isozyme loci currently being studied

With the recent localization of several new loci, a composite listing of these loci and chromosomal locations has been compiled and is shown in Table 1. Although locations of some of the loci are tentative, many are very precisely located from studies involving 1600 to more than 1900 $F_2$ plants.

J. F. Wendel, M. M. Goodman, C. W. Stuber

Table 1. Mapping data for 34 isozyme loci currently being studied.

| Symbol | Name | Location |
|---|---|---|
| Acp1 | acid phosphatase-1 | 9 |
| Aco1 | aconotase-1 | Probably 4S |
| Adh1 | alcohol dehydrogenase-1 | 1L - 127 |
| Amp1 | aminopeptidase-1 | 1L - 89 |
| Amp3 | aminopeptidase-3 | 5S - 32 |
| Dia1 | diaphorase-1 | 2S |
| Dia2 | diaphorase-2 | 1L |
| E8 | esterase-8 | 3S - 20 |
| Enp1 | endopeptidase-1 | 6L - 13 |
| Glu1 | β-glucosidase-1 | 10L - approx. 30 |
| Gdh1 | glutamate dehydrogenase-1 | 1L - 144 |
| Gdh2 | glutamate dehydrogenase-2 | 10 |
| Got1 | glutamic-oxaloacetic transaminase-1 | 3L - 95 |
| Got2 | glutamic-oxaloacetic transaminase-2 | 5L - 96 |
| Got3 | glutamic-oxaloacetic transaminase-3 | 5S - 34 |
| Hex1 | hexokinase-1 | 3S - 44 |
| Hex2 | hexokinase-2 | 6L - 58 |
| Idh1 | isocitrate dehydrogenase-1 | 8L |
| Idh2 | isocitrate dehydrogenase-2 | 6L - 101 |
| Mdh1 | malate dehydrogenase-1 | 8 |
| Mdh2 | malate dehydrogenase-2 | 6L - 103 |
| Mdh3 | malate dehydrogenase-3 | 3L - 121 |
| Mdh4 | malate dehydrogenase-4 | 1L - 97 |
| Mdh5 | malate dehydrogenase-5 | 5S - 16 |
| Me1 | malic enzyme | 3L - 100 |
| mmm | modifier of mitochondrial MDH's | 1L - 101 |
| Pgd1 | 6-phosphogluconate dehydrogenase-1 | 6L - 10 |
| Pgd2 | 6-phosphogluconate dehydrogenase-2 | 3L - 71 |
| Pgm1 | phosphoglucomutase-1 | 1L - 121 |
| Pgm2 | phosphoglucomutase-2 | 5S - 0 |
| Phi1 | phosphohexose isomerase-1 | 1L - 139 |
| Sad1 | shikimate dehydrogenase-1 | 10 |
| Tpi3 | triose phosphate isomerase-3 | Probably 8L |
| Tpi4 | triose phosphate isomerase-4 | 3L - 61 |

ST. LOUIS, MISSOURI
Monsanto Company

## Linkage data for 2L and 4L mutants

The following 2-point linkage data were obtained from F2 repulsion crosses. The % recombination was calculated using the product method.

Chromosome 4L: ol to dcr*-1005A

| progeny | + + | ol + | + dcr* | ol dcr* | % Recomb. |
|---|---|---|---|---|---|
| 1 | 236 | 101 | 62 | 15 | 42±2.06 |
| 2 | 222 | 77 | 47 | 13 | 47±2.46 |

Chromosome 2L: w3 to o*-1195A

| progeny | + + | o* + | + w3 | o* w3 | % Recomb. |
|---|---|---|---|---|---|
| 1 | 176 | 58 | 66 | 9 | 37±2.17 |
| 2 | 266 | 71 | 79 | 15 | 45±2.17 |
| 3 | 221 | 57 | 78 | 15 | 46±2.37 |

v4 to fl*-1316A

| progeny | + + | v4 + | + fl* | v4 fl* | % Recomb. |
|---|---|---|---|---|---|
| 1 | 278 | 102 | 96 | 10 | 33±1.47 |
| 2 | 220 | 70 | 64 | 8 | 37±1.93 |
| 3 | 258 | 99 | 92 | 18 | 40±1.87 |

v4 to pgspt*-579B

| progeny | + + | v4 + | + pgspt* | v4 pgspt* | % Recomb. |
|---|---|---|---|---|---|
| 1 | 201 | 79 | 70 | 9 | 35±1.82 |
| 2 | 319 | 70 | 68 | 6 | 37±1.72 |

w3 to ogm*-1488B

| progeny | + + | + ogm* | w3 + | w3 ogm* | % Recomb. |
|---|---|---|---|---|---|
| 1 | 145 | 65 | 69 | 24 | 46±2.65 |

In addition to the above mutants, good expression and 3:1 segregations can be obtained with the 2L mutants *pgspt*-278B, pgspt*-464, mn*-1120A, wlvpg*-424* and *cpfl*-1024A*. However, the 2L mutants *pg*-330A, rgh*-786A,* and *o*-999A* and the 4L mutants *dek8, ptd*-1130A* and *et*-516C* show poor expression and/or penetrance. Our inclination is to follow Marcus Rhoades' advice for such bad genes. "Don't map them!"

Sheila McCormick and Arlene Barnason

ST. PAUL, MINNESOTA
University of Minnesota

## Discovery of *Ac* activity among progeny of regenerated maize plants

The occurrence of a high frequency of chromosome breakage in regenerated maize plants led us to speculate that transposable elements might be present. In crosses between regenerated plants and a *Ds*-containing tester stock, three cases of *Ac* activity have been found among 66 immature-embryo-derived cell lines (5%).

All cell lines tested in this study were derived from A188 or progeny of crosses of A188 and Oh43 or B73. Regenerated plants or their progeny (all *c c)* were used to pollinate stocks homozygous for *C* and *Ds* at its standard location in chromosome 9. The resulting ears were scored for the presence of colored kernels with colorless sectors, indicating breakage at *Ds* and subsequent loss of *C*. Potential cases of *Ac* were then retested onto *c-m-2* to confirm the *Ac* activity. These tests have identified three cell lines which contain *Ac,* as summarized in Table 1.

It is important to test whether these embryos contained *Ac* before culturing. For embryos 4-41 and 1-42, a few seeds were available from the same ears used to initiate the cultures; also available was remnant seed from the parent rows. Twenty-three tests of these controls onto the *Ds* stock produced no variegated kernels. Our A188 sources were tested since they were involved in the parentage of the $R_o$ plants; no *Ac* was detected in over 25 crosses. In addition, the fact that all three cell lines are heterogeneous for *Ac* indicates that the element was not present in the original embryos cultured. Further tests with regenerated plants and controls are in progress.

V. M. Peschke, R. L. Phillips
and B. G. Gengenbach

Table 1. Summary of cell lines producing regenerated plants with Ac activity.

| Embryo* | No. $R_0$ plants tested onto Ds | No. plants showing positive test | No. showing positive test on c-m-2 | Ac in control crosses |
|---|---|---|---|---|
| 4-41 | 27 | 11 | 2/2; more in progress | 0/12 |
| 1-42 | 7 | 1 | ** | 0/11 |
| 8-27 | 3 | 1 | ** | ** |

* Embryos cultured by M. Lee (M.S. thesis, University of Minnesota, 1984), from sib-pollinated ears of (Oh43 x A188) $F_2$ plants.
** In progress.

## Genetic control of plant regeneration from maize tissue cultures

Although maize tissue culture techniques have been improving steadily in recent years, a major limitation still remaining is the poor *in vitro* response of many agronomically elite genotypes. We conducted a study in 1982 to obtain information on the inheritance of plant regeneration ability of tissue cultures initiated from immature maize embryos. The two populations used in this study were derived from crosses of A188 (excellent tissue culture response) with the inbred lines B73 and A619 (both agronomically elite, but poor in terms of tissue culture response). Each population was composed of six generations (Tables 1 and 2; the reciprocal $F_1$'s constitute one generation). Note that $F_1$ embryos are produced on the parental inbreds, and $F_2$ and BC embryos are produced on $F_1$ plants. Immature embryos ranged from 1.0 to 1.5 mm in length. Cultures were scored twice from 14 to 37 days following immature embryo isolation for regenerable structure formation (somatic embryoids, shoot meristems, or scutellar-like bodies).

The variation among generations was partitioned using generation means analysis (B. I. Hayman, Heredity 12:371-390, 1958; B. I. Hayman, Genetica 31:133-146, 1960). Least squares regression techniques were used to fit increasingly complex models to generation means of each population (K. Mather and J. L. Jinks, pp. 65-81 in Biometrical Genetics: The Study of Continuous Variation, third edition, Chapman and Hall, N.Y., 1982). The results of the regression analysis were evaluated in terms of the proportion of the uncorrected sums of squares among generation means accounted for by fitting the population mean (m), additive (d), and dominance (h) effects (Table 3).

Mean squares for the pooled additive genetic effects were highly significant for both sets of crosses (Table 3). Selection for regenerable structure formation from maize tissue cultures should therefore be effective within both populations. Mean squares for the additional pooled dominance effects after fitting the pooled additive effects were significant for the A188/B73 crosses, but not for the A188/A619 crosses. The estimate of the parameter h for the pooled dominance effects in the A188/B73

Table 1. Regenerable structure formation from immature embryos of maize inbreds A188 and B73, and the specified crosses. All embryos were cultured on N6 medium with 1 mg/l 2,4-D, 100 mg/l casamino acids, and 25 mM L-proline.

| Cross | Ear 1* | Ear 2* | Ear 3* | Mean** | Vigor*** |
|---|---|---|---|---|---|
| A188 ⊗ | 22/30 | 9/30 | 17/30 | 53.3 | 3.1 |
| B73 ⊗ | 0/30 | 0/30 | 0/30 | 0.0 | --- |
| A188 X B73 | 2/30 | 3/30 | 6/30 | 12.2 | 1.7 |
| B73 X A188 | 1/30 | 1/29 | 7/30 | 10.1 | 1.7 |
| (A188 X B73) ⊗ | 16/200 | 19/198 | 18/190 | 9.0 | 2.5 |
| (A188 X B73) X A188 | 60/197 | 71/200 | 30/156 | 29.1 | 2.8 |
| (A188 X B73) X B73 | 0/200 | 0/191 | 1/190 | 0.2 | 2.0 |

* Number of embryos forming regenerable structures/total number of embryos.
** Percentage of embryos forming regenerable structures.
*** Mean of vigor scores assigned to all embryos forming regenerable structures (1 - 5 scale, 5 = best).

Table 2. Regenerable structure formation from immature embryos of maize inbreds A188 and A619, and the specified crosses. All embryos were cultured on N6 medium with 1 mg/l 2,4-D, 100 mg/l casamino acids, and 25 mM L-proline.

| Cross | Ear 1* | Ear 2* | Ear 3* | Mean** | Vigor*** |
|---|---|---|---|---|---|
| A188 ⊗ | 22/30 | 9/30 | 17/30 | 53.3 | 3.1 |
| A619 ⊗ | 0/30 | 0/30 | 0/29 | 0.0 | --- |
| A188 X A619 | 28/30 | 13/30 | 3/30 | 48.9 | 2.8 |
| A619 X A188 | 14/20 | 10/30 | 7/29 | 39.2 | 2.7 |
| (A188 X A619) ⊗ | 31/198 | 59/200 | 57/180 | 25.4 | 2.3 |
| (A188 X A619) X A188 | 111/200 | 55/174 | ------ | 44.4 | 2.6 |
| (A188 X A619) X A619 | 28/180 | 26/199 | ------ | 14.2 | 2.0 |

* Number of embryos forming regenerable structures/total number of embryos.
** Percentage of embryos forming regenerable structures.
*** Mean of vigor scores assigned to all embryos forming regenerable structures (1 - 5 scale, 5 = best).

Table 3. R-squared values indicating the proportion of variability among generation means accounted for by fitting m, and the additional effects of adding d subsequent to m, d and h together subsequent to m, and h subsequent to d and m.

| Population | $R^2$ (m) | $R^2$ (d/m) | $R^2$ (d,h/m) | $R^2$ (h/d,m) |
|---|---|---|---|---|
| A188/B73 | 45.02** | 47.18** | 52.99** | 5.81* |
| A188/A619 | 72.18** | 24.02** | 26.41** | 2.39 |

*, ** Significant at the 5% and 1% probability levels, respectively.

population was negative, indicating that B73 possesses a partially to completely dominant gene or genes which inhibit(s) the formation of regenerable structures. Deviations from the additive and dominance effects were not significant for either population; thus, epistatic effects did not contribute significantly to the variation among generation means. A genetic model based only on the mean and additive genetic effects accounted for 96.2% of the variation among generation means for the A188/A619 population. A model based on the mean and both additive and dominance effects accounted for 98.0% of the variation among the A188/B73 generation means.

In summary, additive genetic effects were highly significant for both the A188/A619 and A188/B73 sets of crosses. Neither dominance nor epistatic effects contributed significantly to the variation among generation means for the A188/A619 crosses; however, dominance effects were significant for the A188/B73 crosses. The results of this analysis do not rule out the possibility that regenerable structure formation is qualitatively inherited, but we have not yet developed a qualitative model that fits all the data.

C. L. Armstrong, C. E. Green,
R. L. Phillips and R. E. Stucker

**rDNA restriction analysis of tissue culture regenerated A188 plants**

DNA restriction endonuclease cleavage and Southern hybridization analysis of maize ribosomal DNA were undertaken to determine if large scale restriction site polymorphism or repeat length heterogeneity can arise in regenerated plants due to the tissue culture cycle. Nuclear DNA from 25 regenerated plants and one seed-grown plant (control) of the maize inbred A188 were subjected to restriction analysis by cleavage with EcoR1, BamH1, and Sst1, and hybridized with a nick-translated probe of the maize 9kb ribosomal repeat. The plants used in this analysis were regenerated from a single tissue culture line produced over a period of 22 months. In addition, the cell culture pedigree, that is the cell lineage relationship of each regenerated plant to each other plant, was maintained.

The maize inbred A188 contains approximately 7,000 copies of the ribosomal repeat per 2C nucleus. Based on a series of reconstruction experiments, a large scale change such as variation in the length of the external spacer could be detected if approximately 100 copies (1.43% of the total number of repeats) had the occurrence of a common change. A new site dividing the 9kb fragment into two 4.5kb fragments would be detected if 200 copies of the 4.5kb ribosomal fragment were present in the new band. This would stoichiometrically correspond to a total of 100 copies of the rDNA repeat. The limits of other detectable variation would be dependent on the number of site changes that occurred in common and the DNA fragment size variation that those changes produced. Overall, no large scale variation in the rDNA genes isolated from the 25 regenerated plants was detected by this analysis. This implies that these genes appear to be stable in the regenerated plants over a 22 month period of growth in tissue culture.

Southern hybridization of the BamH1 digest, however, showed variation in the amount of DNA present in the three different restriction size fragments produced by this endonuclease. There are two BamH1 sites located in the maize ribosomal repeat. Cleavage of the ribosomal repeat at the restriction site present in the 18S gene results in a 9kb fragment. Site heterogeneity, believed due to methylation of the BamH1 site located in the 26S rDNA gene, is responsible for the presence of the 5 and 4kb bands. Approximately 50% of the 26S BamH1 sites are believed to be methylated, resulting in an even distribution of the ribosomal DNA between the 9kb and the combined 5 and 4kb fragments. A decrease in methylation would be detected by a decrease in intensity of the 9kb band and a corresponding increase in the intensity of the 5 and 4kb bands in each sample. Such variation was detected in densitometer scans of the BamH1 digest. The extent of this variation observed in the 26 samples showed a distribution around a 9kb fragment mean of 55% with a standard deviation of 8%. The range about

the mean is from 41 to 68% for the 9kb fragment. The level of variation observed in this analysis does not show a correlation of culture age to restriction site heterogeneity. In addition, there is no correlation of tissue culture pedigree to DNA fragment distribution. Further, the variation fits a normal distribution about the mean (based on a Wilk-Shapiro W statistic) and therefore appears to be random. If the variation observed was due to a directed genetic change, such as a reduction in site-specific methylation, the data would not be expected to be normally distributed about the mean. Because these measurements do not show discrete differences in the density of the different DNA fragment size classes, and the data approximate a normal distribution, the observed differences may be due to physiological variation or experimental error and not genetic variation. These data do, however, indicate the possibility of random changes in the cleavage heterogeneity of this BamH1 site due to the tissue culture cycle.

Gary Benzion*, Brenda G. Hunter, Irwin Rubenstein and Ronald L. Phillips

*Department of Agronomy, University of Kentucky, Lexington, KY

### Tests for a cytoplasm that restores genetic male sterile-1

Inbred A188 does not have a cytoplasm that restores *ms1*. Selfs of plants shown by test crosses to be A188/*ms* segregated for male sterility.

Similar tests of the "standard normal" line derived from a T6-9b stock did not segregate for male sterility. Selfs and test crosses of those progeny are being grown in 1985. Test crosses on male-sterile plants that have only male steriles will identify plants that are *ms* but fertile because of a restorer cytoplasm. I will believe it only when those results are obtained.

Charles R. Burnham

### The *su gl4 la* stock

Ears supposed to be 3-point backcrosses segregated only for *su* and *la* (see 1984 Maize News Letter). The *su la* plant used for the backcrosses was neither *gl* nor heterozygous *gl*. From the increases obtained in 1984, I can now furnish seed that should provide *su gl4 la* plants. My face will be *red* if it does not.

Charles R. Burnham

### Discussions in Cytogenetics

My book "Discussions in Cytogenetics" was reprinted in Sept., 1984. They again are available from me, but at a higher price, $17.00 (postage included). Order from:

Charles R. Burnham
1539 Branston St.
St. Paul, MN 55108

SRINAGAR, KASHMIR, INDIA
Regional Research Laboratory, C.S.I.R.
BOMBAY, INDIA
Bhavan's College, Andheri

### Genetic variability and character association in maize grown in Kashmir

The present investigation was undertaken to assess the genetic parameters and correlation coefficients of 15 local maize varieties (MNL 57:134-137, 1983) grown in sub-mountainous areas of Kashmir valley.

In a randomized block design replicated 4 times at Srinagar, 15 varieties of maize were grown in rows 12 m long, spaced 75 cm apart. The distance between plants was 25 cm. When plants attained a height of 12-15 cm, they were thinned to maintain 1 plant to a hill. Each row represented a plot, and observations were recorded on 10 plants in each plot on 12 characters (Table 1) which were found signifi-

Table 1. Mean, range, phenotypic genetopyic coefficient of variability, heritability and variance for different traits in maize

| Characters | Mean | Range | Pcv | gcv | Heritability % | Variance | Error variance |
|---|---|---|---|---|---|---|---|
| Number of leaves/plant | 12.03 | 9.15 - 14.88 | 14.21 | 13.30 | 87.38 | 10.62** | 0.37 |
| Plant height (cm) | 150.85 | 112.83 -188.91 | 16.99 | 14.25 | 70.28 | 2044.12** | 195.39 |
| Leaf-Index (cm)% | 49.92 | 29.07 - 64.69 | 36.52 | 23.10 | 40.01 | 715.68* | 195.07 |
| Number of tassel branches/plant | 19.91 | 11.91 - 28.87 | 26.82 | 23.45 | 76.91 | 94.57** | 6.60 |
| Shelling (%) | 70.33 | 55.75 - 76.62 | 11.65 | 6.42 | 43.71 | 155.37* | 37.83 |
| Ear height (cm) | 69.35 | 49.51 - 95.67 | 23.64 | 20.98 | 78.84 | 903.94** | 66.82 |
| Ear diameter (cm) | 3.70 | 2.70 - 4.00 | 11.89 | 8.64 | 53.24 | 0.50** | 0.09 |
| Ear length (cm) | 13.38 | 9.10 - 16.20 | 18.31 | 13.15 | 55.11 | 15.96** | 2.70 |
| Number of Ears/plant | 1.60 | 1.25 - 2.02 | 33.12 | 32.50 | 97.86 | 0.17* | 0.06 |
| Grain moisture % at harvest | 31.12 | 15.25 - 38.50 | 22.49 | 18.95 | 70.55 | 153.67** | 14.52 |
| Days to 75% silking | 84.11 | 73.75 - 92.00 | 6.42 | 4.87 | 57.15 | 79.70** | 12.58 |
| Tassel length from flag leaf (cm). | 41.25 | 34.67 - 46.01 | 10.76 | 6.59 | 37.37 | 42.07* | 12.42 |

* Significant at 1%
** Significant at 5%

Table 2. -Correlation coefficient among different traits in maize

| Characters | Leaf-index (cm)$^2$ | Number of tassel branches/plant | Shelling (%) | Ear height (cm) | Ear diameter (cm) |
|---|---|---|---|---|---|
| Plant height (cm) | 0.4615 (0.7453)** | 0.6267 (0.6861)** | 0.5736 (0.8123)** | 0.8731** (0.9721)** | 0.7191** (0.7647)** |
| Leaf-index (cm)$^2$ | | 0.6203 (0.8007)** | 0.0650 (0.2607) | 0.4860 (0.8081)** | 0.3600 (0.6103) |
| No. of tassel branches/plant | | | 0.2171 (0.3830) | 0.6137 (0.6543)** | 0.5712 (0.6808)** |
| Shelling (%) | | | | 0.5040 (0.7892)** | 0.4128 (0.8666)** |
| Ear height (cm) | | | | | 0.6225 (0.6713)** |
| Ear diameter (cm) | | | | | |
| Ear length (cm) | | | | | |
| No. of ears/plant | | | | | |
| Grain moisture (%) at harvest | | | | | |
| Days to 75% silking | | | | | |
| Tassel length from flag leaf (cm) | | | | | |

(Table 2 (continued)

| Character | Ear length (cm) | Number of ears/plant | Grain moisture % at harvest | Days to 75% silking | Tassel length from flag leaf (cm) | Number of leaves/plant |
|---|---|---|---|---|---|---|
| Plant height (cm) | 0.7752** (0.8757)** | 0.0712 (0.2164) | 0.5481 (0.7750)** | 0.3886 (0.7069)** | 0.6036 (0.6965)** | 0.7289** (0.9049)** |
| Leaf-index (cm)$^2$ | 0.4531 (0.7049)** | 0.1054 (0.1261) | 0.5434 (0.7038)** | 0.4872 (0.9600)** | 0.2512 (0.2986) | 0.6089 (0.9228)** |
| No. of tassel branches/plant | 0.5964 (0.7026)** | 0.5207 (0.0740) | 0.5615 (0.6586)** | 0.4817 (0.7919)** | 0.2669 (0.2626) | 0.6757** (0.7593)** |
| Shelling (%) | 0.5492 (0.8877)** | 0.0384 (0.0179) | 0.3974 (0.8098)** | 0.2990 (0.4900) | 0.5071 (0.7290)** | 0.3705 (0.7660)** |
| Ear height (cm) | 0.7496 ** (0.8938)** | 0.3135 (0.2577) | 0.5021 (0.6394) | 0.4296 (0.6919)** | 0.4245 (0.4653) | 0.8015 ** (0.9264)** |
| Ear diameter (cm) | 0.7221 ** (0.8965)** | 0.0396 (0.0668) | 0.6709 ** (0.7821)** | 0.4932 (0.8751)** | 0.4833 (0.6676)** | 0.5965 (0.9348)** |
| Ear length (cm) | | 0.2049 (0.1951) | 0.6578 ** (0.9758)** | 0.3560 (0.8780)** | 0.5033 (0.5997) | 0.7151 ** (0.8234)** |
| Number of ears/plant | | | 0.0255 (0.0866) | 0.0439 (0.0864) | 0.0951 (0.0923) | 0.2102 (0.1913) |
| Grain moisture(%)at harvest | | | | 0.6458 ** (0.9367)** | 0.3906 (0.7485)** | 0.7191 ** (0.9296)** |
| Days to 75% silking | | | | | 0.1704 (0.4084) | 0.6392 (0.9005)** |
| Tassel length from flag leaf (cm) | | | | | | 0.2860 (0.5633) |

**Values in parentheses are the genotypic correlations.**
**** Significant at 1% and 5%**

cant. The analysis of variance showed that there was adequate variation for all the characters. Little difference was observed between the phenotypic and genotypic coefficient of variability in all the characters studied excepting leaf-index, indicating that these characters respond less to environmental factors. The genotypic coefficient of variability for most of the characters was very low, i.e. for number of ears per plant (32.50), number of tassel branches per plant (23.45), leaf-index (23.10), ear height (20.98) and shelling percentage (6.42), indicating that these characters are more influenced by environment.

The relative amount of the heritable portion of variation can be assessed through heritable percentage. In our study high heritable values in the broad sense were observed for number of ears per plant, number of leaves per plant, ear height, number of tassel branches per plant and plant height, indicating the importance of these characters in varietal improvement through selection. A high heritable value may be attributed to additive and non-additive gene action. High heritability values, 97.86, 87.30 and 78.84, were observed in number of ears per plant, number of leaves per plant and ear length, respectively. Data on the coefficient of correlation (Table 2) show that ear length x grain moisture percent at harvest had a maximum positive significant genotypic association, followed by plant height x ear height, and 75 percent silking x leaf index, grain moisture percent at harvest and number of leaves per plant, respectively. All the characters except number of ears per plant, shelling percentage and tassel length from flag leaf showed a positive significant correlation at the genotypic level, and this was higher than that of phenotypic correlation.

P. N. Jotshi, K. A. Patel and M. K. Bhan

STANFORD, CALIFORNIA
Stanford University

**Variability among Mutator elements**

The transposable element *Mu-1* has been isolated from maize lines exhibiting Robertson's Mutator activity, characterized by a mutation rate 20-50 fold higher than normal (Bennetzen et al., P.N.A.S. 81:4125, 1984). *Mu-1* is 1367 bp in length and contains terminal inverted repeats of 213 and 215 bp and four open reading frames (Barker et al., N.A.R. 12:5955, 1984).

We have isolated 30 *Mu-1* homologous clones from a Mutator library. Twenty-six contain elements of the same size class as *Mu-1*, but four contain Mutator elements which differ from *Mu-1* in both size and organization. *Mu-L*, a 1.7 kbp element, contains 300 bp which are not present in *Mu-1*. Electron microscopy and hybridization experiments show that the additional 300 bp are not a duplication of sequences found in other Mutator elements, nor are they highly represented in the DNA of non-Mutator plants. The other three novel elements are smaller than *Mu-1*. *Mu-S1* (1.0 kbp) has intact terminal repeats and internal segments which are homologous to *Mu-1*. Both *Mu-L* and *Mu-S1* contain small deletions and/or restriction site polymorphisms which indicate they could not have arisen from *Mu-1* by a single insertion/deletion event. *Mu-S2* (800 bp) and *Mu-S3* (650 bp) do not have intact terminal repeats and show less homology to *Mu-1*. We are sequencing the elements to determine their coding capacity and their relationship to *Mu-1*. The 300 additional base pairs present in *Mu-L* may have the capacity to code for a protein. Approximately 250 bp of this additional DNA has been subcloned for use as a specific probe with which to follow the segregation and transposition of *Mu-L* independently of the other elements in the Mutator system.

Loverine P. Taylor and Virginia Walbot

**Cloning and characterization of the linear 2.3 Kbp mitochondrial plasmid of maize**

The mitochondria of maize, as well as many other higher plants, contain small circular and linear DNA molecules known as mitochondrial plasmids. The mitochondria of all maize lines examined to date contain a linear 2.3 kilobase pair (or related 2.1 Kbp) linear plasmid. Since this is the only mitochondrial plasmid found consistently in all maize lines, it is the best candidate for encoding an essential mitochondrial function. It has been shown that this plasmid has protein(s) tightly associated with its 5′ termini that would interfere with standard molecular cloning techniques. In order to clone this mitochondrial plasmid into a bacterial vector, we first separated the 2.3 Kbp plasmid from the high molecular weight (HMW) main mitochondrial genome on CsCl gradients. Next, complementary homopolymer "tails" were added to the 2.3 Kbp plasmid and the linearized bacterial vector pUC8, and the annealed DNAs were transformed into bacterial cells. By using this technique we were able to bypass the terminally bound 5′ protein and obtain full length DNA clones of the 2.3 Kbp plasmid, which we have named pZm2.3. Nucleotide sequence analysis of one end of this clone reveals that 15 out of 17 base pairs are homologous with the termini of the linear S plasmids, which are found in plasmid form only in *cms-S* type mitochondria. The S plasmids also have protein(s) tightly associated with their 5′ termini. Further analysis using pZm2.3 as a hybridization probe has shown that there is an integrated form of the plasmid in the HMW DNA of N(fertile), *cms-T*, *cms-C* and *cms-S* mitochondria. It has been previously shown that S plasmid related sequences exist in an integrated form in N and *cms-S* HMW mitochondrial DNA. Small amounts of dimers of the 2.3 Kbp plasmid can be detected, and may represent replicative forms. The 2.1 Kbp linear plasmid found in *cms-T* and some fertile inbred lines is very homologous to the 2.3 Kbp plasmid, and apparently is deleted near one terminus. We are currently sequencing the cloned plasmid and are especially interested in investigating the possibility that the 2.3 Kbp plasmid may encode the protein that is associated with the 5′ termini of this plasmid.

P. Bedinger, E. de Hostas and V. Walbot

**Aberrant segregation of a mutable phenotype in a Robertson's Mutator Line**

In last year's Newsletter we described an unstable *bz2* mutable allele *(bz2-Mu1)* that was isolated from a Mutator line. The original spotted kernel *(bz2-Mu1/bz2)*, when testcrossed and selfed, showed normal segregation, 1:1 and 3:1, respectively. The spotted progeny were again selfed and testcrossed. Although many of the ears yielded the expected segregation ratios, a few plants did not. In the eight plants examined last year, the two plants with a low copy number of Mutator elements and the two plants with approximately five fold more Mutator elements yielded unexpected segregation ratios, while the four plants with intermediate numbers of *Mu* elements showed normal segregation. This led V. Walbot to postulate that either low or high copy numbers of Mutator elements might inhibit somatic reversion at *bz2-Mu1*, resulting in fewer spotted kernels than expected. A model was proposed in which lines carrying an intermediate number of Mutator copies would have an active Mutator system.

During the past year, we have continued to analyze whether there is a relationship between Mutator activity and the total copy number of Mutator elements in the genome. We have examined the segregation of Mutator activity in the *bz2-Mu1*

lineages for two additional generations by following forward mutation at selected loci (presumably measuring transposition) and somatic reversion at *bz2-Mu1* (presumably measuring excision at one locus). We have evidence for at least two mechanisms that may control Mutator activity.

We wanted to know whether the somatic reversion at *bz2-Mu1* is correlated with *Mu* activity in general. To do this we have looked at the frequency of seedling mutations (albinos, lethals) in four lineages that vary in *Mu* copy number and segregation ratios of spotted and bronze kernels. We find that lines with fewer spotted kernels than expected also tend to have a decreased seedling mutation rate. We are therefore using the somatic reversion at *bz2-Mu1* as a phenotypic marker for Mutator activity. However, this correlation must be made for each lineage examined.

Examination of a much larger sample size than presented last year revealed two plants with equivalent *Mu* copy number, but one appears to have an active Mutator system, while the other does not. This suggests that the simple model that total copy number controls Mutator activity does not hold for all plants.

We were then interested in testing whether any detectable alteration in the *Mu-1* element (as first described by J. Bennetzen) correlated with Mutator activity. DNA from the maize lines described below was digested with the restriction enzyme HinfI and analyzed on Southern blots, probing with the cloned *Mu-1* element. HinfI digests within the terminal inverted repeats, resulting in a characteristic 1.3 kbp fragment for *Mu-1*. Surprisingly, in some of our *bz2-Mu1* lines the Mutator elements no longer show the typical restriction pattern of *Mu-1*, instead many larger fragments are observed. Controls were done to test for completeness of digestion, and these larger fragments are not due to incomplete digestion of total DNA. When the DNA was digested with other enzymes that restrict within the internal portion of the element, fragments larger than those predicted from the restriction map of *Mu-1* were also obtained. We are currently investigating whether this altered restriction pattern is due to DNA modification, amplification, or rearrangement of sequences within the element. Results from two lineages are summarized below.

The selfed ear of plant 230-3 *(bz2-Mu1/bz2)* had no spotted kernels, but behaved normally when testcrossed. Single seedling DNA was prepared from eight kernels off the selfed ear and examined as described above. In all cases no 1.3 kbp HinfI fragments, characteristic of *Mu-1*, were detectable. Instead, we observed larger *Mu-1* homologous fragments ranging in size from approximately 1.4 kbp to 8.0 kbp. We next analyzed both bronze and spotted kernels from the testcrossed ear. Of eight bronze *(bz2/bz2)* kernels, four contained the characteristic *Mu* pattern, two contained the 1.3 kbp size class as well as larger fragments, and two contained only larger fragments. Of eight spotted *(bz2-Mu1/bz2)* kernels, six contained the characteristic *Mu* pattern, but two contained larger fragments as well as the 1.3 kbp size class. Leaf DNA from the 230-3 plant was analyzed. It contained approximately 30 copies of *Mu* elements in the characteristic pattern; no larger fragments were observed. This suggests that both altered and unaltered *Mu* elements can exist together, and that the change can occur within one generation. Two other lineages with aberrant segregation of the mutable phenotype have been analyzed with similar results.

Our working hypothesis is that the altered elements are not functional, and when the element at *bz2-Mu1* is altered it is no longer capable of somatic excision. However, not all of our lines that demonstrate aberrant segregation of the mutable phenotype contain this altered pattern. Plant 230-1 *(bz2-Mu1/bz2)*, containing approximately 12 *Mu* elements, when selfed showed normal segregation, but only 1% spotted kernels were observed in the testcross. Single seedling DNA was prepared from four kernels off each ear. All samples contained only the characteristic *Mu* HinfI restriction fragments. No larger fragments were observed. The only difference we could detect between the selfed and testcrossed progeny on Southern blots was that the total copy number of Mutator elements tended to be approximately 2-3 fold lower in the backcrossed progeny. Therefore, this plant may be losing Mutator activity due to dilution of *Mu* copy number or some other alteration not yet detected.

These data suggest there may be at least two mechanisms for decreasing somatic reversion at *bz2-Mu1*; one, an alteration of the elements as observed in plant 230-3, and two, a dilution of elements as observed for plant 230-1. If the number of functional Mutator elements is too low, somatic excision of the element at *bz2-Mu1* may be too infrequent or too late in development to be observed in the aleurone tissue.

This hypothesis predicts that somatic excision at *bz2-Mu1* might be restored by introducing additional elements from an active Mutator line. We have tested whether crossing *bz2-Mu1* 230-1 progeny to a *bz2/bz2* Mutator line would restore somatic reversion at *bz2-Mu1*. For this experiment kernels from the testcrossed ear were planted and testcrossed to determine which had the *bz2-Mu1* allele. (Spotted kernels are observed at low frequency which allows us to determine which plants have the *bz2-Mu1* allele.) These plants were also crossed to an active Mutator line *(bz2/bz2)* to observe somatic reversion at *bz2-Mu1*. Results are presented in the accompanying table. When the plants were testcrossed, the number of spotted progeny deviated dramatically from that expected for Mendelian segregation, $P<.0005$. How-

Table 1. Segregation ratios of spotted to bronze kernels

TESTCROSS

| Plant # | spt:bz | % spt k Actual | Expected | $\chi^2$ | P value |
|---|---|---|---|---|---|
| 1 | 5:269 | 2 | 50 | 254 | <.0005 |
| 2 | 13:240 | 5 | 50 | 204 | <.0005 |
| 3 | 3:337 | .09 | 50 | 328 | <.0005 |
| 4 | 51:162 | 24 | 50 | 58 | <.0005 |

CROSS TO MUTATOR (bz2/bz2)

| spt:bz | % spt k Actual | Expected | $\chi^2$ | P value |
|---|---|---|---|---|
| 93:127 | 42 | 50 | 5.2 | .025 |
| 136:139 | 50 | 50 | .03 | .88 |
| 125:132 | 49 | 50 | .19 | .63 |
| 105:128 | 45 | 50 | 2.3 | .14 |
| 67:82 | 45 | 50 | 1.5 | .23 |

ever, when crossed to an active Mutator line, there was an increased number of spotted kernels in all cases, and in 4/5 cases the expected number of spotted progeny were observed. These results suggest that some component in an active Mutator line can stimulate somatic reversion at *bz2-Mu1*, resulting in normal segregation of the mutable phenotype. Whether this is due to simply increasing the copy number of active Mutator elements, or to the introduction of some other factor, is an open question. Currently, we are investigating whether the seedling mutation rate is also increased in these lines. Similar experiments to determine if the altered elements can be returned to normal by crossing with an active Mutator line are now in progress with progeny from 230-3.

Vicki Chandler and Virginia Walbot

TSUKUBA, JAPAN
National Institute of Agrobiological Resources
ST. PAUL, MINNESOTA
University of Minnesota

## Fertile revertants possessing S-1 and S-2 DNAs in *cms-S*

*Cms-S* can be distinguished from other types of cms on the basis of the two plasmid-like mtDNA's, termed S-1 and S-2 DNAs (Pring et al., P.N.A.S. 74:2904, 1977). Several cytoplasmically reverted strains from male sterility to male fertility have been reported (Singh and Laughnan, Genetics 71:607, 1972), and all of these revertants show the lack of the S-1 and S-2 DNA's (Levings et al., Science 209:1021, 1980).

In this study, we used *S*-type cytoplasmic lines *G, H, J, L, ME, My, R, S,* and *Vg,* which were in WF9 nuclear background. Three lines, *J'*, *G'*, and *R'* developed from *J, G,* and *R* cytoplasms, respectively, produced exceptional male fertile progenies. These revertant lines were advanced crosses with WF9 lacking the nuclear gene *Rf3* for fertility restoration of *cms-S*. In the following generations of crosses with Wf9 *(rf3 rf3)* males, all of the progenies from the three revertants were male fertile. This suggested that the male fertility was not caused by nuclear mutation of restorer genes, but by cytoplasmically inherited changes.

Mitochondrial DNA (mtDNA) was isolated as described previously (Umbeck and Gengenbach, Crop Sci. 23:584, 1983) from *Vg* and revertant *J'*, *G'*, and *R'* cytoplasms possessing WF9 nuclear background. Terminally attached proteins of S-1 and S-2 DNAs were examined by proteinase K treatment prior to phenol-chloroform extraction (Kemble et al., Nucl. Acids Res. 10:8181, 1982). To check S-1 and S-2 DNAs, undigested mtDNAs were analyzed by agarose gel electrophoresis. The restriction sites of chromosomal mtDNA, S-1 and S-2 DNAs were examined by digestion with several restriction enzymes.

Electrophoresis of undigested mtDNA (Fig. 1) shows that revertants *J'* and *G'* have S-1 and S-2 DNAs, and *J'* has terminally attached proteins like other standard *cms-S* lines. The restriction fragment patterns in the revertants were different from each other and also differed from that of *Vg* cytoplasm (Fig. 2). Moreover, these patterns were not identical with those of other revertants reported by Levings et al. The restriction sites of S-1 and S-2 DNA in *J'*, *G'*, *R'* and *Vg* cytoplasms were checked by restriction with BamHI, EcoRI, HindIII, PstI, and XhoI (data

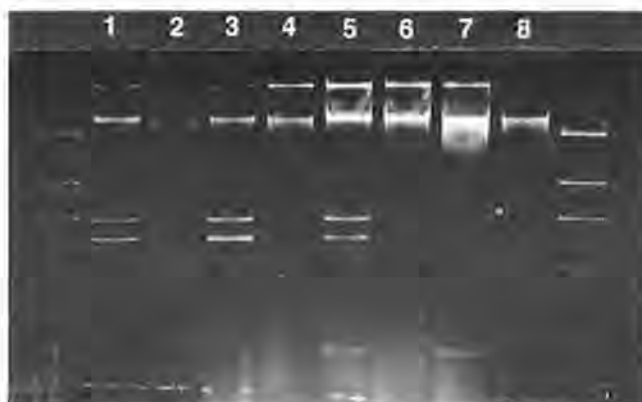



**Figure 1. Agarose gel electrophoresis of undigested mt-DNA isolated from *G'* (1), *J'* (2,3), *S*(4,5), *T*(6), *C*(7), and *N*(8) cytoplasm maize. 2 and 4 were not treated with proteinase K during isolation. *J'* and *G'* are cytoplasmic revertants. Markers are 23.7, 9.5, 6.7, and 4.3 kb.**

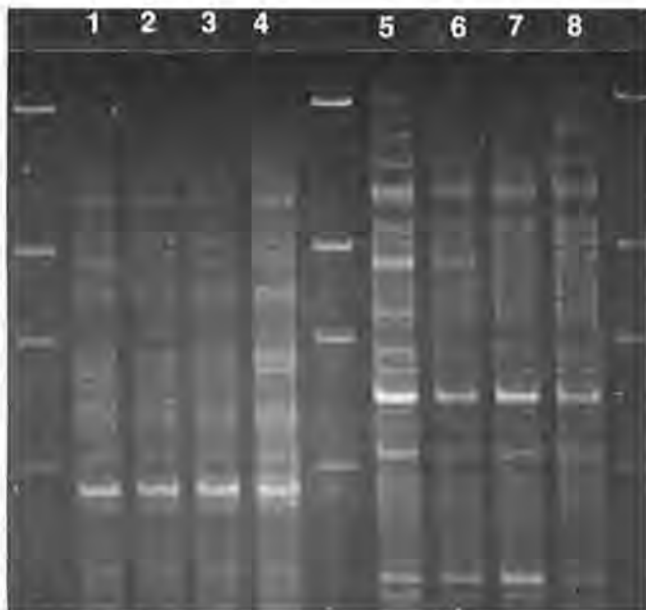



**Figure 2. Agarose gel electrophoretic patterns of mtDNA digested by EcoRI (1-4) and XhoI (5-8) restriction endonucleases. 1 and 5 were isolated from *R′* cytoplasm, 2 and 6 from *G′*, 3 and 7 from *J′*, 4 and 8 from *Vg*. *R′*, *G′* and *J′* are cytoplasmic revertants. *Vg* is a member of the *cms-S* group. DNA markers are 23.7, 9.5, 6.7 and 4.3 kb.**

not given), and there were no restriction site differences detected among these S-1 and S-2 DNAs.

These results indicated that the cytoplasmic changes of *J′*, *G′* and *R′* cytoplasm from male sterility to male fertility were associated with rearrangements of mitochondrial chromosomal mtDNA and not with disappearance of plasmid-like S-1 and S-2 DNAs.

Teruo Ishige and Burle Gengenbach

UNIVERSITY PARK, PENNSYLVANIA
The Pennsylvania State University

### Properties of starches from nonmutant and mutant endosperm cultures

In order to evaluate the potential mutant expression of endosperm genes in cell cultures, we initiated cultures of normal, *ae* and *wx* endosperm in 1983. All lines were in the A636 inbred background. We reasoned that starches of these genotypes could be compared at several levels, including starch granule morphology, starch granule iodine staining and distribution of the starch polymers, amylose and amylopectin. Starch formation was studied in nine-month-old suspension cultures of each genotype. Isolated starches were compared with starches isolated from mature endosperm of the same genotype. Starches from normal cultures and endosperm stained blue-black with iodine, and were round to polygonal in shape. Amylose accounted for 30.4% and 31.6% of the starch from cultures and mature endosperm, respectively. Starch granules from *ae* endosperm and cultures stained blue-black with iodine, and contained abnormal (budded, oblong) starch granules. However, the frequency of abnormal granules was greater in the mature endosperm. Amylose comprised 67.7% and 39.9% of mature endosperm and culture starches, respectively. Starch granules from *wx* endosperm and cultures stained brown-orange with iodine, contained no amylose and contained a few large "ragged-edged" granules. Therefore, we concluded that endosperm specific genes are phenotypically expressed in cultures up to nine months of age.

C. D. Boyer and Carole Haemmerle

URBANA, ILLINOIS
University of Illinois

### Mapping zein polypeptides using translocations

This is the first report of an extensive plan to locate and map all of the zein proteins to chromosomes using reciprocal translocations as chromosome arm markers, duplicate-deficient types and segmental aneuploids. The greatly improved method for the separation of zein polypeptides by isoelectric focusing (IEF) in agarose gels (Wilson, Cereal Chem. 61:198-200, 1984) makes our goal possible. At least 36 different zeins have been identified in various stocks and inbred lines. Isopropanol-mercaptoethanol soluble endosperm proteins from four homozygous inbred lines (W23, L317, Oh43 and W64A) were separated by charge in IEF agarose. From each of the four gel patterns, individually stained bands were cut out from the dry agarose gel, boiled in SDS buffer, loaded and separated in 12% acrylamide SDS-6M urea gels. Our numbering system is like that of Wilson (Biochem. Genetics, in press, 1985) in IEF gels, expanded by mol. wt. designations from SDS gels. For example, zein band 10 in IEF agarose from Oh43 and Oh45 has a molecular wt. of 19.6kd in SDS-urea. Our notation is 10-19.6, combining an Rf equivalent (distance from the cathode) with the observed mol. wt.

As observed by others, each inbred line and homozygous translocation stock has a specific zein profile or "fingerprint". IEF band 22 (22-22.5, 22-19.6) is a double band in SDS gels and it is present only in the Oh43 group (Oh43, Oh45, Oh545 and W153R), while band 21.5 (21.5-22.5, 21.5-19.6) is in the SSS group (RSSC, B73, A632, A634) and in L317. These two 22.5 and 19.6kd zein genes appear to be linked and to segregate with 4L.

The maize stocks used in the experiments were fourteen homozygous inbred lines and several homozygous translocation stocks with breakpoints on chromosome 4 and on other chromosomes. The $F_1$s

were obtained by crossing homozygous inbred lines to chromosome 4 translocations. The $F_1$s then were backcrossed to several inbred lines to obtain the $F_2$. Homozygosity in the parents was important to obtain a known dosage of genes without extraneous segregating factors. To test for homozygosity, zein extractions from ten seeds from each of the parents were run side by side in IEF agarose gels. No segregation was observed in any of the parents.

Distally located translocation break points often survive on the female side and give duplicate-deficient endosperm. The $F_1$ seed from T4-10f (4L .94; 10L .14) provide such duplicate-deficient progeny. We assume 10L is duplicate and 4L is deficient, since 10L deficient and 4L duplicate seeds are lethal as observed on the $F_2$ ear. With those assumptions, we have tentatively assigned 41-14.3 and 49-19.6 to map at 10L and 22-22.5, 22-19.6 are located distal to 4L between .94-1.0.

Dosage effects were observed in the IEF pattern of hybrids as is expected with the triploid endosperm, since hybrids receive two genes from the female parent and one gene from the male parent. The cross W153R x T4-9g (4S .27; 9L .27) backcrossed to L317 gave two zein bands, 54-19.6 and 53-19.6, that are not visible in any of the parents. These zeins seem to be under the control of a regulatory gene.

The results from the other crosses between the inbred lines and the translocation stocks are summarized as follows:

W23 x T4-9(5974) (4L .80; 9L .87) was backcrossed to N28. Bands 22-22.5, 22-19.6 and 53-19.6 are in coupling, thus linked. Both are located on the long arm of chromosome 4.

B73 x T3-4(5156) (3S. 47; 4L. 67) was backcrossed to Oh43. 28-22.5 and 33.5 - 22.5 are in repulsion with one putative crossover observed between these two loci. Both are located on chromosome 4.

N28 x T3-4(5156) (3S. 47; 4L. 67) was backcrossed to W23; a. 21.5 - 22.5, 21.5 - 19.6 and 53 - 19.6 are in coupling with their location on 4L; b. 28 - 22.5 and 54 - 19.6 are in repulsion and also on 4.

W153R x T4-9g (4S. 27; 9L. 27) was backcrossed to W64A, 10 - 19.6 and 22 - 22.5, 22-19.6 showed a perfect correlation, like the band pattern of the T4-9g parent.

Laura M. M. Ottoboni and Dale M. Steffensen

**Cytoplasmic reversion to fertility in *cms-S* without loss of linear mitochondrial plasmids**

The *S* type of sterile cytoplasm in maize is characterized by the presence in the mitochondria of autonomously replicating linear plasmid-like DNAs, S1 and S2. These plasmids have not been found in any normal fertile maize mitochondria, nor previously in male-fertile cytoplasmic revertants. Recent findings in our laboratory, however, indicate that the retention or loss of S1 and S2 in association with cytoplasmic reversion to fertility may be primarily a function of the nuclear genetic background, rather than a characteristic of the *S*-type cytoplasm itself.

The cytoplasmic revertants studied previously (Levings et al., Science 209:1021-1023; Laughnan et al., Stadler Genet. Symp. 13:93-114) have the nuclear background of inbred line M825, which shows the highest frequency of spontaneous reversion to fertility (Gabay-Laughnan and Laughnan, Maydica 28: 251-263). So far we have examined 27 cytoplasmic revertant strains with the M825 nuclear background, and in all cases the reversion event was correlated with disappearance of both S1 and S2.

Five spontaneous cytoplasmic revertants recovered from WF9, four from RD-WF9 and one from ML-WF9, all retained free S1 and S2 plasmids in the mitochondria. The amount of the free plasmids remained undiminished relative to that of the sterile parent line, even after four generations of crossing to the maintainer line, thus discounting the possibility that the free plasmids are merely carry-over from the sterile parent.

Cytoplasmic revertants, recovered after RD-WF9 and ML-WF9 were crossed seven times with M825 (recurrent male parent), did not show the free plasmids S1 and S2. Since these revertants presumably differed from the above WF9 revertants only in nuclear genotype, this indicates that the nuclear genome has the major influence on retention or loss of free plasmids in the mitochondria during cytoplasmic reversion to fertility. Alternatively, changes in nuclear genotype may impose changes in organization of the mitochondrial genome prior to cytoplasmic reversion, so that the same reversion event in two strains with different nuclear genotypes might well produce revertants with different characteristics. In either case, these studies indicate that the *cms-S* sterility-fertility phenomenon is not directly dependent upon the status of the S1 and S2 plasmids.

Loida J. Escote, Susan Gabay-Laughnan
and John R. Laughnan

VICTORIA, BRITISH COLUMBIA
University of Victoria

**Flavonoids in brown pericarps**

Both *a P-RR* and *a r-ch Pl* have brown pericarps when mature (as compared to the red and cherry pericarps of the *A P-RR* and *A r-ch Pl* genotypes, respectively). Methanol extracts of mature *a P-RR* pericarps contain flavones, and extracts of *a r-ch Pl* contain flavonols. Just prior to the formation of the brown pigments, extracts of *a P-RR R-r* pericarps have flavones, flavanones, chalcones and aurones, but no flavonols. Extracts of *a P-WR r-ch* pericarp at

the same stage have large quantities of flavonols, but none of the flavones, flavanones, chalcones and aurones found with *a P-RR R-r*.

O. Ceska and E. D. Styles

**Expression of the *R-r:Ecuador(1172)* allele**

*R-r:Ecuador(1172)* is a complex allele of the *R* locus that has been the subject of many recombination studies. Most of these studies have been done against a genetic background of the W22 inbred line. We have found that when our W22 *R-r:Ecuador* stocks are outcrossed, the specific features associated with the Ecuador allele (leaf, silk, pericarp color, etc.) are not always expressed; in fact some of our non-W22 lines of *R-r:Ecuador* cannot be distinguished from lines carrying other *R-r* alleles. Possible explanations include the loss by segregation of specific modifiers carried in W22, or the loss of structural components of the *R* locus required for tissue specific pigment production. Introduction of the Ecuador allele from these non-W22 stocks into plants homozygous for *a3*, a recessive intensifier of plant pigment, confirms that it is still fundamentally different from other *R-r* alleles. The enhancing effect of *a3* on most other *R-r* alleles is minimal, and can be shown clearly only in the absence of *B* locus action (i.e., with *b*). The young leaves of *a3 R-r:Ecuador* plants are deep purple as they emerge from the whorl, but become progressively greener as they mature, so that at any one point in time, the plant appears continuously graded in anthocyanin pigmentation.

E. Derek Styles

WALTHAM, MASSACHUSETTS
University of Massachusetts

**Vascular anatomy of the female rachis in teosinte and maize**

More effort is necessary to understand the type and distribution of the vascular system in *Zea* in relation to the origin of the maize cob, to the expression of genes affecting the cob and to the development of energy sinks. A double vascular system was described in the cob as two independent systems (Laubengayer, 1948, 1949; Reeves, 1950). A small type of bundle occurs in the cupule wings and rind, while a larger type is found near the margins of the pith, rarely in the center. Connections between the two systems were discovered later in the glume-cushion (Galinat, 1959). In describing the vascular anatomy of a four-rowed ear of maize that was apparently a mutation out of eight-rowed maize, Laubengayer (1948) observed that because the barren sides without spikelets had the same inner and outer systems corresponding to eight-rowed maize, a reduction in ranking had occurred in this case. Apparently, genes that alter ranking operate independently from those that change vascular development, although selection tends to produce genetic combinations that are balanced either for teosinte in the wild or maize under domestication. Such changes in one or more genes that are coadaptive with changes in other genes are part of the evolutionary process. Another example within maize is genetic increases in husk length that became necessary for bird and insect protection following genetic increases in ear length.

In the teosinte fruitcase, vascular development is primarily in the outer bundles of the rind, while in maize the shift is to the inner bundles of the pith in proportion to increases in kernel size and row number. This reflects the importance of channeling photosynthate into induration of the teosinte fruit case, in contrast to supplying photosynthate for kernel enlargement and increased endosperm storage in maize. In maize with large hard kernels, there is an adaptive need for an induration of the cupules in order to prevent cob shrinkage during drying and, thereby, continue to provide adequate kernel space.

Walton C. Galinat

**Outer-inner vascular connections and glume phenotype**

The connection between the two vascular systems in the glume cushion provides a double supply source to the lower female glumes. The tunicate inhibitor gene, which appears to be on chromosome 6 (Mangelsdorf, 1958), acts by greatly reducing development of the outer system and lacks outer-inner connections. The consequence is that in the presence of the *Ti* (tunicate inhibitor) gene, the usual elongate growth of tunicate glumes (*Tu* on chromosome 4) as well as papyrescent glumes (*Pn* on chromosome 7) is stunted by starvation into about the length of normal glumes. In their modified expressions, the tunicate glumes still have their usual foliaceous texture but of normal (non-tunicate) length, while likewise the papyrescent glumes have their usual papery texture but of normal (non-papyrescent) length. In normal maize (*tu, pn*), the *Ti* gene inhibits cupule development and in some backgrounds may result in a non-cupulate female rachis in either maize or teosinte. Non-cupulate teosinte has no fruit cases to protect its kernels.

Walton C. Galinat

**Suitable support system for key-trait expression**

The key traits of teosinte and maize are unstable in phenotype in each other's background. Stability depends upon a vascular system that can deliver the photosynthate necessary for initiation of the key traits without being either excessive or limiting.

Once a congruous combination is achieved, stability is assured. In reconstructing such a background for maize to have stable expression of two-ranking from teosinte, it was necessary to reduce vascular development in the pith with the string cob trait of primitive corn and increase vascular development in the rind with the combined effects of teosinte segments on chromosomes 3, 4, and 9. Interactions with teosinte segments on chromosomes 1 and 7 may also be involved, all of which increase the proportion of vascular development in the rind.

Walton C. Galinat

**Necessity for a more comprehensive cytogenetic and phenotypic analysis of the maize-teosinte relationship**

The teosinte segments transferred to A158 derivatives developed by Mangelsdorf were selected initially solely on the basis of their dominant effects on induration of the lower glume and reductions in the ear and kernel size and reduced kernel-row-number (MNL 22:19-20, 1948). No selective attention was given to the key traits of teosinte which immediately disappeared. It wasn't until after about five generations of backcrossing of small families to the same maize inbred (first 4R3 and then A158), followed by selfing to make the segments homozygous, that a linkage analysis, using his nine gene marker stock (WMT), was made to identify the segments in terms of number and location. Regardless of how tight the integrity of these segments due to close linkage and cross-over suppressors, their size must have been significantly reduced.

It is clearly necessary that new teosinte derivatives be developed using multiple marker gene stocks for various individual maize chromosomes (at least chromosomes 1, 3, 4, 7 and 9, following those pinpointed by Mangelsdorf) both as the outcross and backcross parent. The relatively intact teosinte chromosome to be captured would be identified from the start by virtue of its dominant alleles masking the recessives on a particular pre-selected maize chromosome. Since only plants showing the dominants for a certain teosinte chromosome are selected for backcrossing to that multi-marker stock and any individuals showing independent teosintoid traits are discarded, probably the extraneous teosinte material would be reduced to much less than 6 percent after three backcrosses. Ideally, both cytologically and morphologically (e.g., for the penetrance of two-ranking), the maize background for the new derivatives should be the knobless Wilbur's Flint. But, expediently, the backgrounds of the available marker stocks may serve, even though the derivatives would not be strictly isogenic. Some attempt has been made by the curators of the Maize Coop collection of tester stocks to give them a common background. The development of Wilbur's Flint tester gene stocks should be started for such comparative studies, but their completion followed by application would probably be beyond the tenure of at least this investigator. More immediate results may be obtained on the now available tester gene stock backgrounds. Without an isogenic background or otherwise known inbred background, the original marker gene stock has to serve as the control. This would prohibit the effective use of certain markers affecting cob morphology such as ramosa on chromosome 7. It is at this point that a more comprehensive cytogenetic and phenotypic analysis of the origin and evolution of maize may commence.

Walton C. Galinat

**Kernel interlocking (tesselation) resulting from cupule reduction**

Because the dry kernels of sweet corn wrinkle at maturity, they are coadaptive with a soft cob with reduced cupules. By introducing genes for the non-cupulate condition into high row sweet corn, the kernel row number was reduced by half as the adjoining members of different pairs interlocked. A 24-rowed ear became 12-rowed and a 20-rowed ear 10-rowed. Apparently, some minimal degree of cupule development is necessary in sweet corn in order to maintain the desired high kernel row numbers. For the purpose of quality seed production, it may in cases of extremely high kernel row numbers (24 to 30) be advantageous to use interlocking to allow better seed development. Cupule development together with expression of the high kernel row number would be restored to the hybrid from the other parent.

Walton C. Galinat

**Silkless baby corn, seed production genetics**

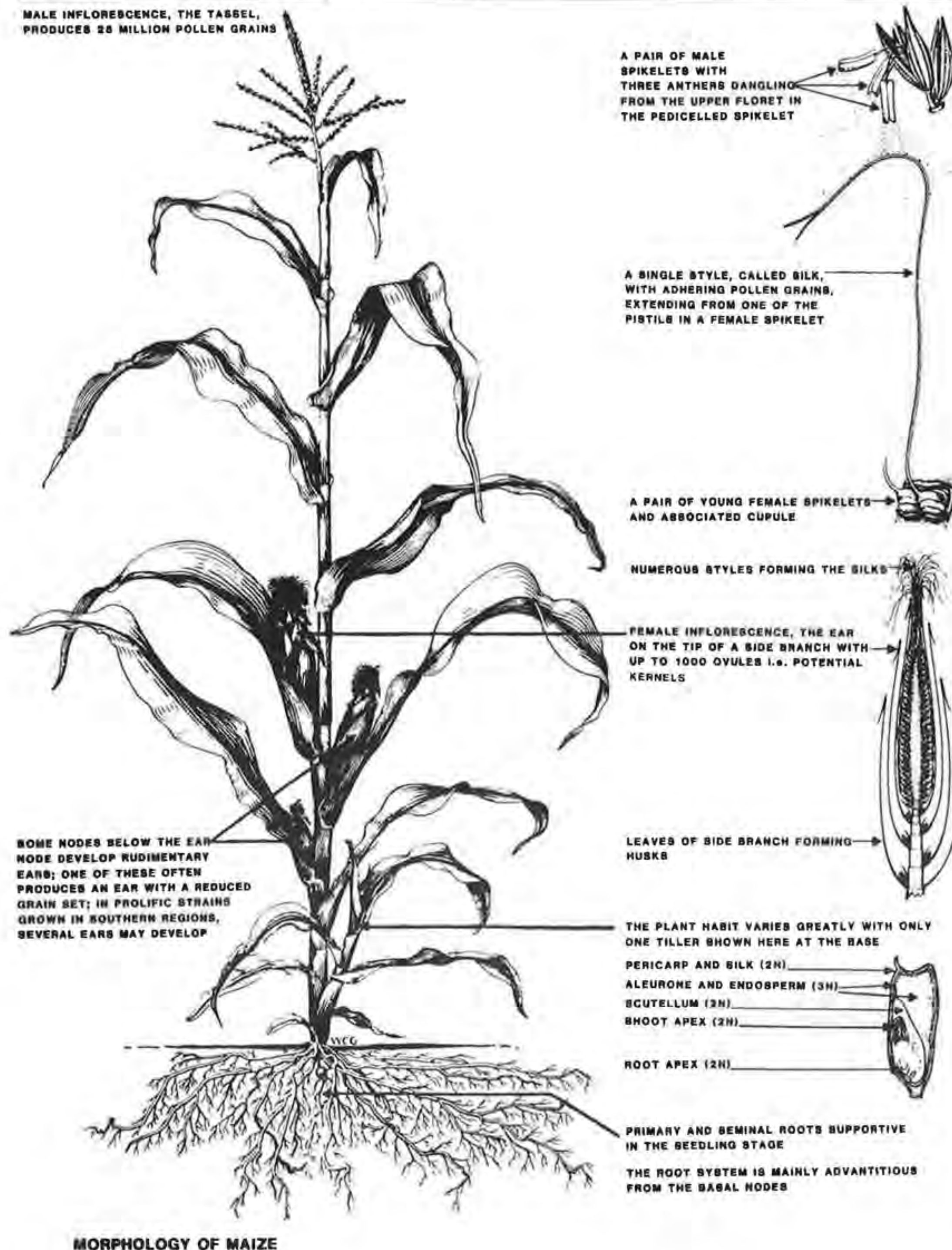
A market has been established through Chinese-American restaurants for the consumption of immature ears of corn harvested at the time the silks just start to emerge above the husk leaves. Multiple-eared hybrids are grown in Taiwan and the immature ears picked by hand, husked, desilked and shipped by air in cold salt brine to the U.S. and Canada. The gourmet specialty departments may sell such corn for about $2.30 for a 14-ounce tin or even at higher prices if pickled and sold in small jars. The prohibitively high labor costs, and the lack of a genetic type suitable to allow mechanization, have excluded the development of this potentially important industry in the United States.

The mechanization problem is partly the critical timing necessary for the harvest. If the silks are pollinated, within hours kernels start to develop and the cob becomes hard and inedible. The answer is to breed a silkless corn that can be mechanically harvested and husked.

The genetic system to do this is based on the use of two different recessive genes for the tassel-seed trait *(ts2* and *ts1)* on chromosomes 1 and 2 respectively as silk restorers for the silkless gene *(sk)*, also on chromosome 2. The double mutants *(sk sk, ts2 ts2)* and *(sk sk, ts1 ts1)*, with selection for a normal sexual balance, function as normal corn. The double hybrid *ts2 Ts2, ts1 Ts1, sk sk* between these two double mutants is 100% silkless because each parent carries the normal dominant allele that masks the recessive tassel-seed gene in the other parent.

Walton C. Galinat

Ed. note: Dr. Galinat has provided a copy of the accompanying figure, titled Morphology of Maize, suitable for reproduction.




**MORPHOLOGY OF MAIZE**

WOOSTER, OHIO
OSU-Ohio Agr. Res. & Dev. Ctr.

### Alleles at the *ora2* locus

Two mutant recessive alleles were produced at the *ora2* locus (orange endosperm) in inbred line Oh43, namely *ora2-13* and *ora2-15*. Pleiotropic vigor effects in populations of +/- and -/- genotypes were compared with +/+ genotypes in tests in the inbred for each allele (the plants in populations of a given genotype are quite uniform).

There are some similarities and some differences in the arrays of pleiotropic effects involving the two alleles. Flowering time behaves in much the same manner in both cases, i.e. the homozygotes flower later and the heterozygotes earlier than the +/+ homozygotes. Perhaps the most striking difference is in plant size as measured by plant height. In the case of the *ora2-13* allele, the homozygotes are shorter and the heterozygotes the same height as the +/+ homozygotes, in contrast to the case of the *ora2-15* allele where both the homozygotes and heterozygotes are taller than the +/+ homozygotes. In the case of the *ora2-13* allele, there was a decrease in grain yield for the -/- homozygotes and an increase in grain yield for the +/- heterozygotes when compared with the +/+ homozygotes. In the case of the *ora2-15* allele, there were no significant differences in grain yield for the -/- or +/- genotypes compared with the +/+ genotypes. The genetic reasons for the differences are not clear at this time.

Perhaps the most likely possibilities as to the genetic nature of the pleiotropic effects would be (1) a block of closely linked genes produced by the mutational event which also produced the recessive allele, or (2) mutations spreading along the chromosome produced by the same mutational event which produced the recessive allele.

E. J. Dollinger

## ADDENDUM

GRAND FORKS, NORTH DAKOTA
University of North Dakota

### Developmental profiles for two new embryo lethal mutants, *dek22* and *dek23*

We have been studying the embryo development of two defective kernel mutants *(cp*-E1113A* and *dcr*-E1428)* previously reported (W.F. Sheridan and M.G. Neuffer, 1982) to lack leaf primordia at the mature kernel stage. These two mutants have been tested for allelism with the named *dek* mutants on 1L and 2L respectively, since the first mutant is on 1L (Sheridan and Neuffer 1981) and the second mutant is on 2L (Sheridan and Neuffer, in press). By use of a double pollination technique (MNL 58:92-95) we have demonstrated non-allelism of *cp*-E1113A* with *dek2* on 1L, and non-allelism of *dcr*-E1428* with both *dek4* and *dek16* on 2L. Consequently we now designate *cp*-E1113A* as *dek22* and *dcr*-E1428* as *dek23*.

Developmental profiles were obtained by examining mutant embryos at intervals from early in kernel development until maturity using paraffin sectioning, fresh dissection, and scanning electron microscopy techniques. Both *dek22* and *dek23* express their mutant phenotypes early in development. Mutant *dek22* embryos reach the transition stage during early kernel development. They remain healthy and morphologically normal, but they are blocked in further growth and development. Mutant embryo expression is markedly uniform both with regard to size and stage of development. Mutant embryos of *dek23* are variable in size but otherwise uniform in mutant phenotype. They reach an abnormal coleoptilar stage with a characteristic arrowhead shape during early kernel development and remain thereafter blocked at that stage, although they continue to enlarge. The mutant embryos fail to differentiate a shoot apex; instead, localized necrosis appears where a shoot apex would normally form, and subsequently spreads throughout the embryo. These two lethal mutants, blocked at different stages of embryo development, appear to define unique steps in the sequence of gene activities essential for normal embryogenesis. A detailed report on these two mutants will soon be submitted for publication in Developmental Biology.

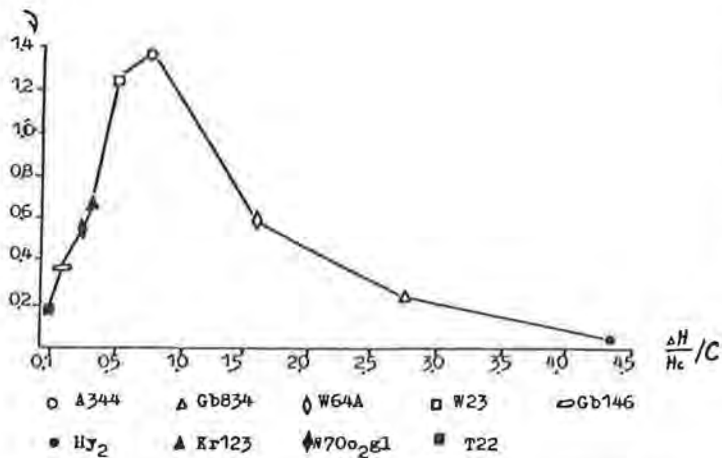
William F. Sheridan and Janice K. Clark

KRASNODAR, U.S.S.R.
Krasnodar Research Institute of Agriculture

### Relationships between sensitivity of inbred lines to NMU and their mutability

Nine inbreds of maize significantly differing in their physiological response to NMU in M1 were taken for study. Two hundred seeds of each inbred were treated for 24 hours with water solutions of the mutagen at 9 concentrations, over a range from 0.005 mM to 3.2 mM. M3 mutations were selected visually. Mutation frequency was estimated on the basis of the number of M1 selfed plants.

It was found that the inbreds displaying minimum and maximum sensitivity to a depressive action of the mutagen proved more stable against the inbreds of moderate resistance. Curvilinear relationships between unit depression per mM, $(\Delta H/Hc)/C$, in plant height and mutability ($\nu$) was most evident in the variants with optimal frequency of mutation (Fig.).



Similar regularities were shown by E.M. Witkin (1969) in classical experiments with *E. coli Str*$^s$. In our experiment the most sensitive inbreds, Hy2 and Gb834, and the most resistant inbreds, T22 and Gb146, were quite like the supersensitive *Uvr*$^-$ *Rec*$^-$ and wild *Uvr*$^+$ *Rec*$^+$ strains, and A344 and W23 looked similar to the highly mutable *Uvr*$^-$ *Rec*$^+$ strain. The similarity in reaction to the mutagen suggests a common universal mechanism of protection and mutability.

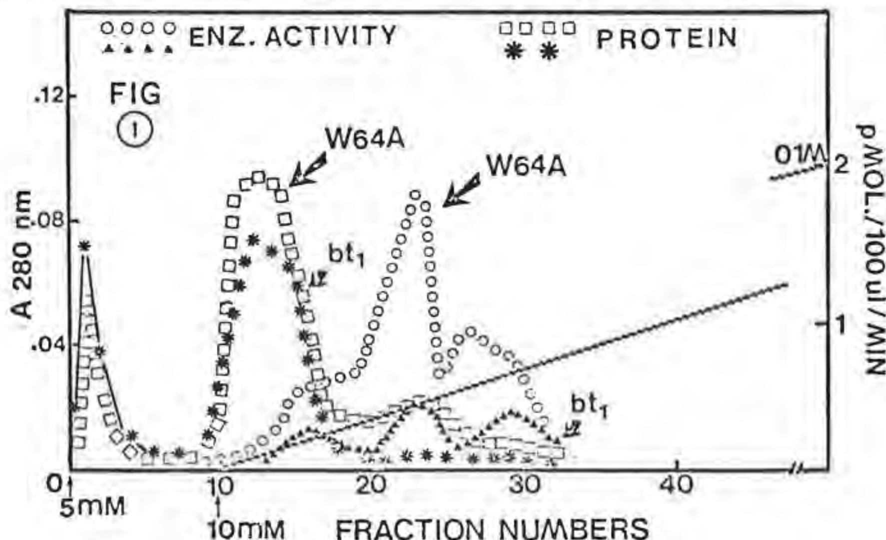
A. S. Mashnenkov

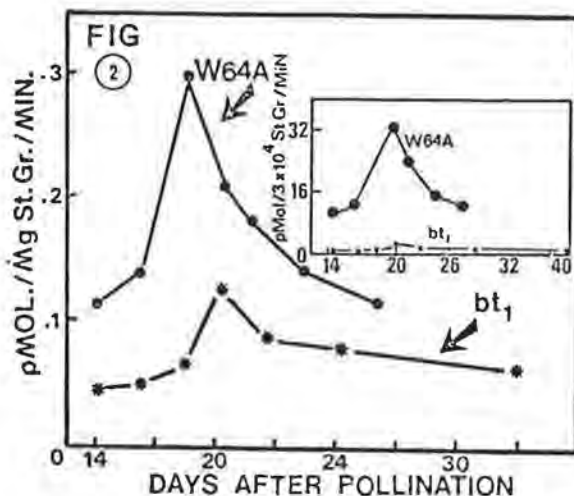
MADISON, WISCONSIN
University of Wisconsin

### The deficiency of a starch granule-bound enzyme phospho-oligosaccharide synthase in developing *bt1 bt1 bt1* endosperms

The brittle-1 *(bt1)* mutant was first described by Mangelsdorf in 1926. The gross phenotypic effect is very similar to *bt2* and *sh2* in producing a shrunken endosperm of brittle texture, and intermediate in translucency between normal and *su1*. The starch content was found to be markedly lower in *bt1* seed than in normal at all stages of development. Large differences between *bt1* and normal were also found for sucrose and reducing sugars during development. The specific biochemical lesion of the *bt1* locus has not been known. We report here that *bt1* endosperms are deficient in a starch granule-bound enzyme, phospho-oligosaccharide synthase, relative to non-mutant endosperms. Phospho-oligosaccharide synthase is an enzyme which converts Glc-1-P into a series of short chain length ($G_2$ to $G_{10}$) phospho-oligosaccharides and oligosaccharides (Pan and Nelson, ms. in preparation). Starch granules of developing kernels (22 days after self-pollination) of a normal inbred line (W64A) and mutant *bt1* were isolated by washing 4 times with 50mM glycylglycine buffer, pH 8, and finally with cold acetone. The solubilization of phospho-oligosaccharide synthase from starch granules was based on the method of F.D. MacDonald and J. Preiss (Plant Physiol. 73: 175-178). The proteins solubilized from starch granules were then precipitated with ammonium sulfate (60%), dialyzed, and finally fractionated on a hydroxyapatite column with Tris-HCl buffer, pH 7, as shown in Fig. 1.

The assay system for the starch granule-bound enzyme contained 0.1 μmol. of ($^{14}C$) Glc-1-P (500 cpm/nmol.), 0.4 μmol. of $Mg^{2+}$, 5 mg of starch granules in a final volume of 60 μl of 0.125 M MES buffer, pH 6.0. After incubation, either the reaction mixture was centrifuged to remove the starch granules, and an aliquot of the supernatant fraction was placed in 0.5 ml (5mg) of sweet corn phytoglycogen solution, then precipitated with 1 ml of 95% ethanol; or to the reaction mixture, 0.5 ml (5mg) of sweet corn phytoglycogen solution was added (without removing the starch granules), then precipitated with 1 ml of 95% ethanol. The precipitate was collected and washed four times with a total of 4 ml of 95% ethanol before suspending in 0.5 ml of water for counting. The assay system for the solubilized enzyme contained 1 μmol. of ($^{14}C$) Glc-1-P (30 μl. 500 cpm/nmol.), and 100 μl of the hydroxyapatite column-purified enzyme in a final volume of 130 μl






of 0.125 M MES buffer, pH 6.0 containing 1 mM DTT. After incubation, the reaction was stopped by the addition of 0.5 ml (5mg) of sweet corn phytoglycogen and precipitation with ethanol as described above for the starch granule-bound enzyme.

The present study shows that the reference mutation at the *bt1* locus results in a deficiency of a starch granule-bound phospho-oligosaccharide synthase. The enzyme activity of hydroxyapatite column-purified enzymes from nonmutant and the *bt1* mutant is shown in Fig. 1. The data show the phospho-oligosaccharide synthase activity in the developing endosperms of *bt1* is remarkably low at this stage of development. Fig. 2 shows the enzyme activity of starch granule-bound phospho-oligosaccharide synthase measured at various post-pollination times in developing endosperms of nonmutant versus *bt1* endosperms. The main point illustrated in this figure is that a similar developmental profile for both nonmutant and *bt1* was observed; however, the enzyme activity of *bt1* is significantly lower than the normal line, particularly when the data are expressed in terms of the number of starch granules used for assay (see inset of Fig. 2), rather than on equal weights of starch granules as is done in Figures 1 and 2. Moreover, we also found that there is no significant difference in the level of the soluble phospho-oligosaccharide synthase in the developing endosperms of either normal or *bt1* (data not shown).

Thus, the results reported here show a marked decrease in the activity of a starch granule-bound phospho-oligosaccharide synthase as a consequence of the *bt1* mutation although it has not yet been demonstrated that the deficiency is the primary biochemical lesion in this mutant.

David Pan and Oliver Nelson, Jr.

NORMAL, ILLINOIS
Illinois State University

**Printing strip tags using a data base**

We have been using a 128K Apple IIe with Appleworks as a data base this past year and find that the program is excellent for working with one season's records (including the seed inventory from crosses). All information was entered into this data base in place of a field notebook. A field notebook was then printed from this data base.

The information encoded in this and most other data bases can be used in numerous ways. This past summer I printed out strip tags on 100 pound tag custom continuous paper which was purchased from Data Documents, a Pitney Bowes Company. I simply printed the contents of the data base regarding each family (family number, pedigree, genotype, number planted, number that grew, and several fields for comments) with an Apple Imagewriter (alias C-ITOH prowriter among others) on this paper, using a standard Imagewriter ribbon with a fixed record length of 11 lines, and cut each page into 6 parts, each containing information about a single family. These were stapled onto the first plant in each family in the research nursery. This ink and paper did not deteriorate appreciably during the growing season, and the tags were in good condition and easily read at the end of the growing season. After harvest, information about specific crosses made with each family was also entered into the data base.

David Weber

PRESCOTT, WISCONSIN
Jacques Seed Co.

**Polymitotic: supernumerary repetitions of meiosis II**

V. Ambros and H. R. Horvitz (Science 226:409, 1984), in their analysis of cell lineages in the nematode *Caenorhabditis elegans,* have found mutations that cause supernumerary moltings or repetitions of other developmental programs. The maize mutant polymitotic causes supernumerary divisions of meiocytes. We suggest (West, Ph.D. thesis, Univ. of Minn., 1985) that polymitotic *(po)* and the *lin* mutants of *C. elegans* share the common characteristic of affecting genetic switches which control developmental subroutines. Mutation results in recycling of the program.

In the case of polymitotic, the events of meiosis II repeat several times. Immediately upon telophase II, chromosome condensation recommences, followed by spindle formation and cytokinesis. Each cycle is very rapid. Lacking chromosome duplication (as meiosis II does), these polymitoses produce nonviable cell remnants, varying in size, and containing variable

amounts of chromatin, with resulting male and female sterility. The term "megacyte" was suggested for megaspore remnants observed replacing embryo sacs (West, M.S. thesis, Univ. of Minn., 1984), following Beadle's suggestion of the term "microcyte" for microspore remnants which replace pollen (G.W. Beadle, Cornell Ag. Exp. Stat. Mem. #135, 1932).

D.P. West and R.L. Phillips*

*Univ. of Minn.

**Polymitotic, canalization and probability**

The maize mutant polymitotic (G.W. Beadle, Science 70:406, 1929, Cornell Ag. Exp. Stat. Mem. #135, 1932; West, M.S. thesis, 1984, Ph.D. thesis, Univ. of Minn., 1985) reveals a locus involved in the orchestration of the developmental steps of meiosis. Specifically, this locus is critical to the cessation of this developmental subroutine. Mutation at this locus results in the failure of meiosis II to conclude. Male sterility is complete, but female sterility only partial.

Histological analysis of polymitotic megasporogenous cells reveals telophase II chalazal cells either degenerating from polymitoses or proceeding to a normal conclusion of meiosis. These two options are reflected in the interspersion of ovules containing megacytes and ovules containing normal embryo sacs on ears of polymitotic homozygotes, the former causing female sterility and the latter giving rise to randomly scattered, normal, viable seed (West, 1985).

Mutation at polymitotic reveals a genetic switch operative in the control or canalization (C.H. Waddington, The Strategy of the Genes, 1957; J.M. Rendel, Canalization and Gene Control, 1967) of development. This is an example of a class of mutants that decanalize development and in so doing reveal an "underlying genotype" (A.S. Fraser, Genetics 57:919, 1967; see also B.T.O. Lee and P.A. Parsons, Biol. Rev. 43:139, 1968, and J.N. Thompson, Stadler Symp. 9:63, 1977). The canalized mode of expression is characterized by dominance (with some exceptions), regulation and very low rate of failure in the orchestration of developmental events. Canalized development gives rise to traits that distinguish species. The decanalized mode is probabilistic, exhibits genetic variance and selection response.

In the case of polymitotic, at the end of meiosis II, each ovule on any given ear faces the same probability of polymitoses. Those not so afflicted are lucky, not fitter. The probability of normal embryo sac formation on mutant plants is a heritable trait of the ear-bearing sporophyte. The number of lucky embryo sacs (and eventual seed) borne on an ear of a polymitotic homozygote is a direct reflection of the genotypic probability value (or potential) of the individual sporophyte. These values vary widely among different genotypes and can be analyzed in the standard quantitative genetic manner, using seed-set as the metric. Seed-set levels are easily modified by selection (West, 1985). The genotypic value altered by selection is a probability. This suggests that the non-mutant allele canalizes development by raising the probability at this genetical switch to 1 (or nearly 1) for the proper conclusion of meiosis.

For polymitotic, canalization, dominance and regulation appear as different aspects of a single mode of genetic function. The canalized mode contrasts an alternative mode which is probabilistic and tractable to selection. This view is consistent with observations of several mutants in *Drosophila* that affect the canalized development of body hairs and wing veins (cf. Waddington, Lee and Parsons, Rendel, Thompson, op.cit.). However, the probabilistic nature of the decanalized mode would not be recognized with mutations which decanalize traits manifest in unreplicated organs or tissues, except as variance in penetrance or expressivity of the decanalizing mutation, or non-correspondence between genotypic and phenotypic segregations (opaque-7? cf. K.S. McWhirter and R.A. Brink, in Walden, ed., Maize Breeding and Genetics, p. 373, 1978). The high level of replication of a single structure and event (the ovule and megasporogenesis) on the maize inflorescence provides a unique opportunity for observing this phenomenon in polymitotic homozygotes.

A mechanism by which natural and artificial selection modify the level, extent or timing of expression of a trait is suggested by the alterability by selection of the probability of alternative states of a genetic switch. Whether such changes in probabilities are a consequence of alleles at multiple loci (D.S. Falconer, Intro. to Quant. Genetics, 1981), or to potentiation of a single functional unit at different levels (W.E. Castle and J.C. Phillips, Carnegie Inst. Wash. Pub. #114, 1914) has not been determined.

David P. West

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 | McCormick | 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 | | |

COOPERATOR POLL: What segments or factors are you mapping? The particular coordinator would like to know about factors you are studying, or about mapping you have in progress, AND TO HAVE SPECIFIC DATA, to aid in the compiling, documenting and evaluating that each coordinator is doing. If you're shy, communicate through Coe (if the information has a long half-life).

CHROMOSOME 1, SHORT ARM

Additional crosses were made using all of the available mutants that have been considered to be possibly located on 1S. We should have some definitive information for our report for next year.

Gil Fletcher

CHROMOSOME 1, LONG ARM

All 1L stocks from the Maize Coop., plus additional stocks from E. H. Coe, M. G. Neuffer, and R. L. Phillips, were grown out at Raleigh, NC, in the 1984 summer nursery for observation and seed increase. All mutants were crossed to A632 and the progeny selfed in the Florida winter nursery. Mapping studies will begin in 1985. Stuber, Wendel and Goodman have placed a new enzyme locus, Dia2, on 1L.

Paul Sisco

CHROMOSOME 2, SHORT ARM

The following mutants (supplied by M. G. Neuffer) were crossed to appropriate marker stocks: v*-453, wt*-472A, dsc-ptd*-901A, de*-1122A, gm*-1289, cp*-1436A. The F2 generations will be grown next summer.

(Wendel et al. report in this News Letter that Dia1 is on 2S).

W. F. Tracy

CHROMOSOME 2, LONG ARM

Please see the item by McCormick in this News Letter.

Sheila McCormick

CHROMOSOME 3, SHORT ARM

Reports in this News Letter confirm the position of cl1 and suggest that E8 is located at the distal end of 3S, about 30 m.u. from d1. Linkage data from test crosses involving the following will be available next year: Lg3 - cl1 - d1, Rg1 - d1 - ra2, and Rg1 - Lg3 - Rf1. Please note that--contrary to last year's report--Neuffer's dwarf mutations d*-E339 and d*-E446 are allelic to d1.

(Stinard reports in this News Letter that brn1, brown endosperm, is uncovered by TB-3Sb; Wendel et al. report recombination E8 - 24 - Hex1 - 17 - Tpi4)

S. Poethig

CHROMOSOME 3, LONG ARM

In this issue of the News Letter are four reports regarding the location of mutants on this arm. On the basis of two sets of crosses, Dooner places vp1 between gl6 and lg2. Based on his linkage data it is most likely that vp1 lies between ts4 and lg2. Scheffler and Peterson report, on the the basis of B-A translocations, that their best estimate is that vp1 is located between lg2 and ts4. Beckett reports that pm1 is not uncovered by TB-3Ld and that, since this translocation was previously reported by him to uncover ba1 and na1, it appears that pm1 is proximal to ba1 and na1. Wendel, Stuber and Goodman report the localization of a new isozyme locus, Tpi4, on 3L near the centromere and 10 map units to the left of Pgd2. In addition, a new isozyme locus, Hex1, was reported on 3S 17 map units from Tpi4 and 24 map units to the right of E8. They note that the mapping of these two new loci between E8 on 3S and Pgd2 on 3L results in the genetic map growing by about 12 units. By utilizing their data together with the working map (MNL 58:255), the following working map can be constructed:

cr1(0), d1(18), E8(20), cl1(38), Lg3(43), Hex1(44), Rg1(45), gl6(47), ts4(52), Tpi4(61), ig1(68), Pgd2(71), lg2(79), ba1(80), na1(91), K3L(93), Got1(95), Me1(100), a3(111), Mdh3(121), a1(127), sh2(127+), et1(139), ga7(145).

William F. Sheridan

CHROMOSOME 4, SHORT ARM

I would like to make a few observations. 4S is of major importance in separating the teosinte spike from the maize ear. It has several loci affecting the kernel, including endosperm composition and kernel shape. It has genes affecting the cob, including spikelet initiation, glume and cupule induration, and abscission layer development. 4S-teosinte has a mutagenic effect on 4S-maize, apparently due to cryptic inversions that evolved as protection for the integrity of the block of teosinte genes. Certain teosintes such as Guatemala teosinte carry a gametophyte allele on 4S for protective purposes, apparently in lieu of cryptic inversions.

(Robertson, 1984, reports recombination in hypoploids for TB-4Sa to gl3, 32.6 to 40.1, and to gl4, 9.0 to 19.3; Wendel et al. report in this News Letter that Acol is probably on 4S).

Walton C. Galinat

CHROMOSOME 4, LONG ARM

Please see the item by McCormick in this News Letter.

(Miranda and Miranda report o1 - 38 - Ga1 in this News Letter; Robertson, 1984, reports recombination in hypoploids for TB-4Sa to gl3, of 32.6 to 40.1, and to gl4, 9.0 to 19.3).

Sheila McCormick

CHROMOSOME 5, SHORT ARM

Two defective kernel (dek) mutations were located in summer of 1984 by M. G. Neuffer and his group to the 5S chromosome arm using a B-A translocation for the region from a2 (inclusive) to points distal. Even though this arm is a short one, there is plenty of room for coding loci. Presumably many of them have visible mutant phenotypes. An effort is in progress to generate new mutants on this arm using a mutable a2 (a2-m5) obtained from S. Dellaporta, and known to have a defective Spm at the a2 site. The stock also contains functional Spm and a mutable wx-m8. Several plants were grown in the summer of 1984 and self-fertilized. They will be used in the summer of 1985 to generate more of this material while screening for visible mutations in mature plants. Screening for mutations that are evident in young seedlings is in progress. Any new variants that arise on transposition of Spm from a2 have good probability of being nearby, viz., on the 5S arm. New variants could also arise from the mutable wx allele and/or transposition of the functional Spm. A compound B-A translocation that involves all known loci for the entire 5S arm has been obtained from D. Robertson and will be employed in addition to the B-A translocation referred to above to ascertain location of new mutants. These mutants may prove accessible to ready cloning depending on the homology the defective Spm shares with other regions of the genome.

(Robertson, 1984, reports recombination in hypoploids for TB-5La to a2, 4.4 to 18.9, and to bt1, 0.2 to 3.6; Bird reports in this News Letter linkage of Hsf1 and Hsf*-1603 with wx T5-9a and T5-9c).

Mary Polacco

CHROMOSOME 5, LONG ARM

(Please see the items by Beckett and by Bird in this News Letter).

CHROMOSOME 6, SHORT AND LONG ARMS

(Please see the item by Wendel, Stuber and Goodman in this News Letter).

CHROMOSOME 7, SHORT ARM

No report.

CHROMOSOME 7, LONG ARM

Linkage tests were begun with yel*-7748, a lethal mutant that the Coop. previously found to be uncovered by TB-7Lb. My preliminary F2 data place it about 30 map units to the right of gl1.

R. H. Whalen

CHROMOSOME 8, SHORT AND LONG ARMS

A number of mutants induced by EMS, by controlling elements and of spontaneous origin have been placed on chromosome 8 using TB-8Lc and wx T8-9d. They are listed below with their approximate locations. de*-1897 resulted from a bz2-m transposition.

Uncovered by TB-8Lc (on 8L distal to the breakpoint):

| | |
|---|---|
| cp*-1387A, 1392A, 1405A, 1528 | collapsed kernel |
| crp*-1058, 1121, 1533 | crumpled kernel |
| de*-1386A, 1897 (see above) | defective kernel |
| o*-1214A, 1383 | opaque kernel |
| ppg*-199B | pale-pale-green seedling |
| rgh*-1154A, 1285 | rough kernel |
| sh*-1530 | shrunken kernel |
| v*-7B, 25, 29B, 358C, 779A, 826 | virescent seedling |
| wl*-203A, 1982, 1985 | white luteus seedling |

Linked to wx T8-9d: Sdw1 (was Sdw*-1592) (Bird and Neuffer, MNL 59) (semi-dwarf).
Proximal to TB-8La by hypoploid test: ub*-76C (unbranched tassel).
Allelic to prol: crp*-1058, crp*-1121, rgh*-1154A, cp*-1528.

Similar mutants will be tested for allelism. Representatives from each type are being crossed with a Bif Clt vl6 j linkage tester, which presumably covers a part of the short arm and most of the long arm of chromosome 8.

Reports from other cooperators have been minimal. Charles W. Stuber, Raleigh, reports three isozyme loci on chromosome 8, Idh1, Mdh1 and Tpi3, with no positions given (see MNL 59).

M. G. Neuffer

CHROMOSOME 9, SHORT ARM

d*-660B is allelic to d3.

(Please see also the item by Coe in this News Letter).

O. E. Nelson, Jr.

CHROMOSOME 9, LONG ARM

R. Bird reports in this News Letter that Les*-2005 is on chromosome 9, showing low to 0 recombination with wx in translocations involving chromosome 9. Please see the item by Coe in this News Letter; allelism tests and mapping for those factors on 9L, and for factors on 9 but unplaced, are in progress. Wendel et al. report in this News Letter that Acp1 is on 9.

Ed Coe

CHROMOSOME 10, SHORT ARM

(Marc Albertsen has volunteered to coordinate mapping information for this arm).

(Robertson, 1984, reports recombination in hypoploids for TB-10Sc to bf2, 0.0 to 2.0, to r, 27.2 to 43.1; for TB-10La to oy, 21.7 to 29.1, to bf2, 6.8 to 14.6, to y9, 11.5 to 20.9; Wendel et al. report in this News Letter that Sad1 is on chromosome 10).

CHROMOSOME 10, LONG ARM

In a recent paper Binelli, Soave and Ottaviano (Plant Sci. Lett. 33:259-265, 1984) report close linkage between a gene specifying a zein protein (Zp 22/2) and opaque-7. No recombinants were observed in 122 testcross kernels. Band 22/2 evidently is not specified by O7 since its presence vs. absence in various stocks is not associated with allelic state of O7.

The position of luteus-1, shown between R and Lc on recent maps, is in question because crosses of it with an R to Lc deletion stock give white rather than luteus stripes in sr2 sr2 progeny. Either luteus-1 is not represented in the R to Lc region or its hemizygous expression is not luteus. The same luteus stock has been crossed to an R to Mst deletion to determine whether the gene lies distal to Lc, in the Lc to Mst interval.

(Robertson, 1984, reports recombination in hypoploids for TB-10Sc to bf2, 0.0 to 2.0, to r, 27.2 to 43.1; for TB-10La to oy, 21.7 to 29.1, to bf2, 6.8 to 14.6, to y9, 11.5 to 20.9; Wendel et al. report in this News Letter that Sad1 is on chromosome 10).

Jerry Kermicle

B CHROMOSOMES

(Please see the item by Bor-yaw Lin in this News Letter, on B breakpoints of B-A translocations).

Wayne Carlson

MAIZE WORKING MAPS

As was done last year, a "working map" for each chromosome is included at the end of this newsletter. I greatly appreciate all of the corrections and comments I received concerning last year's maps. I only hope that I was able to correct all of last year's mistakes, and not make too many more this year. Several changes should be apparent in this year's maps and I'll briefly describe the major ones.

First off, I hope they are a bit more readable. By using two pages per map, the amount of reduction was kept to a minimum. As more and more genes are placed on the map, this problem increases. Any ideas on how to present the maps better? A second change is the inclusion of the gene name just to the right of the gene symbol. As with last year's maps, the "core map" is the published linkage map of maize (in Genetics Maps, 1984). I have made only one significant change, anticipating the availability of documented compilations and evaluations from coordinators. Immediately to the right are those genes that have some information leading to a "rough" placement on the map, either near a gene already on the map or to a region of the map. Further to the right are those genes which have been placed only to the chromosome (represented by the vertical line with dashes at both ends) or to one arm (represented by a vertical line running from near the centromere to the end of the arm).

The last difference is the inclusion of all known B-A translocation breakpoints. I was impressed with the recent presentations of Jack Beckett concerning genes found to be or not to be uncovered by various TB's. I have found in the past that this information often helps in resolving certain gene placements and set out to gather that data together. The result is presented in the tables just prior to the working maps, and I refer you to the explanation of the tables for further information regarding them. The vertical line associated with each TB represents the area on the linkage map where the breakpoint might be located (i.e. all genes distal to a simple TB would be uncovered). In the case of compound B-A translocations, the line indicates the second breakpoint in that arm and would represent the point at which all genes distal would not be uncovered.

Concerning next year's working maps, one suggestion I received was whether all mutants (including "starred" ones) could be included in the map. I think this is a good idea, since even an unnamed mutant may be helpful to someone working in a particular region. This information could be easily incorporated if each coordinator could supply me a list of all mutants known to be on the chromosome arm they are involved with, along with any linkage or TB information. What about it?

As we expect to publish the next update of the gene list and linkage map in the next edition of Genetic Maps, around February, 1986, I would like to encourage all maize mappers to make an extra effort to (1) get their information into next year's newsletter early and (2) to send it to the appropriate coordinator, so that it can be included in the updated map. This would also be a good time to "name that gene" if sufficient information is available (you might consider consulting the coordinator about updated information for factors on that arm).

Dave Hoisington

**Mapping is often worth more than other endeavors -- You've heard of 3-point mapping?**

(r refers to numbered references in the list of Recent Maize Publications)

CHROMOSOME 1

Adh1-S3034, Mu1 sequence 1367bp, 9bp direct insertion repeats; inverted terminal repeats 215, 213bp; two internal direct repeats, 104bp, and 4 ORFs, 2 in each strand, symmetrically distributed in halves --Barker &, r24

Adh1-S3034 vs. progenitor, Adh1-S: Mu1 insertion in intron-1 73bp down from its 5' junction, associated with a 9bp direct duplication of intron sequence --Bennetzen &, r32

Adh1 allele -CroF (fast) cDNA clone sequence, 1592bp, vs. -S genomic sequence having 9 introns, promoters; vs. alleles -F, -FkF, -FkF, -gamma25, -PrF, -78F, -33F, -Ct, -FCm, and -S5657, -F63, -F207 (3 CRM- nulls, EMS-induced) --Dennis &, r97

Adh1-5 allele; Mdh4-8.2, -8.7, -9, -10, -10.5, -12, -12-, -14, -14.7, -15.5 alleles; Mmm-m, -m1 alleles; Pgm1-12 allele; Phi1-1, -1.7, -3.5, -4.5, -8 alleles --Doebley &, r104; Smith &, r439

Adh1 restriction map with insertions in alleles -Fm335 (Ds), -S5446 (Bs1), -S3034 (Mu1), -S4477 (Mu1), -S4478 (Mu1), -2F11 (Ds) --Freeling, r132

Mp transpositions proximal to P1 occur only beyond 4 units from P1; distal mostly closer, decreasing with distance --Greenblatt, r150

Adh1 stable mutants -S5657, -S664, -S1015, -S719, -S1108, -S96, -S3020, -S3034a, -F460, -F207, -FkFgamma25 compared in mRNA length, CRM, genomic restriction map --Hake &, r159

K1L1, K1S2 location, distribution --Kato, r204

Adh1-S5446, -S5453 alleles arose in BSMV-infected plants, both unstable; -S5446 contains 3.3kb insertion --Mottinger &, r312

Adh1-Fm335, Ds-induced, restriction maps vs. Adh1-F progenitor, two revertants, and Adh1-S --Sutton &, r468

Oleic-linoleic ratio association, wx T1-9c vs. inbred X-187 --Widstrom &, r520

Monosome-1 plants delayed in flowering --Weber, r510

Adh1-Usv allele from regenerated tissue culture, from -S allele in -F/-S --Brettell &, 59:24

Adh1-2F11: Ds-2F11 insert in 4th exon sequenced; 1319bp, of which 600 on left and 300 on right show high homology to Ac --Merckelbach &, 59:33

Dia2 (diaphorase, cytosolic, dimeric) on 1L --Wendel &, 59:87

dek22 (was cp*-E1113A) designated; on 1L --Sheridan &, 59:104

CHROMOSOME 2

K2L1, K2S1 location, distribution --Kato, r204

Monosome-2 plants delayed flowering, indehiscent; 16.8% plump pollen --Weber, r510

v*-350, v*-424, v*-576A, v*-588A (virescents with developmentally conditional hcf) are allelic --Polacco, 59:38

Dia1 (diaphorase, cytosolic, monomeric) on 2S --Wendel &, 59:87

w3 - 43 - o*-1195A; v4 - 37 - fl*-1316A; v4 - 36 - pgspt*-579B; w3 - 46 - ogm*-1488B --McCormick &, 59:91

dek23 (was dcr*-E1428) designated; on 2L --Sheridan &, 59:104

CHROMOSOME 3

E8-1, -2, -2.5, -7, -8, -9 alleles; Got1-3.8, -4.5, -4.7, -5.8, -6.2, -7, -7*, -8 alleles; Mdh3-7.3, -11.5, -17.2 alleles; Pgd2-8, -10 alleles --Doebley &, r104; Smith &, r439

K3L1, K3S1 location, distribution --Kato, r204

Monosome-3 plants delayed flowering --Weber, r510

vp1 uncovered by TB-3La, d, g, h; not by TB-3Lc, i, j, k, l, m; vp1-c821708 and vp1-m451 (Cy-responsive) colorless dormant alleles --Scheffler &, 59:3,4

brn1 uncovered by TB-3Sb --Stinard, 59:7

a1-Mum1, -Mum2, -Mum3 alleles Mu-generated --Robertson, 59:10

a1-m1 5719A state (medium dark) contains 400bp I insert; a1-m1-6078 (colorless), 2.2kb insert --Berndtgen &, 59:34

hcf19 uncovered by TB-3La --Polacco, 59:38

| Tests with TB-3L | a | f | g | d | i | c | h | k | l | m |
|---|---|---|---|---|---|---|---|---|---|---|
| | pm1 | pm1 | pm1 | + | + | + | | + | + | + |
| | ba1 | ba1 | ba1 | ba1 | ba1 | ba1 | | | + | |
| | na1 | na1 | | na1 | na1 | na1 | + | | + | |

--Beckett, 59:39

vp1 - 42 - a1; gl6 - 8 - vp1 --Dooner, 59:85

d1 - 17 - cl1; cl1 - 5 - Lg3 --Poethig, 59:86

E8 - 30 - d1 - 13 - cl1 --Sorrentino &, 59:86

Hex1 (hexokinase, cytosolic) uncovered by TB-3Sb; Tpi4 (triose phosphate isomerase, cytosolic, dimeric); E8 - 24 - Hex1 - 17 - Tpi4 - 10 - Pgd2 - 41 - Mdh3 --Wendel &, 59:88

d*-E339 and d*-E446 are allelic to d1 --Poethig, 59:108

CHROMOSOME 4

K4L1, K4S2 location, distribution --Kato, r204

TB-4Sa (B breakpoint at junction of prox. euchr. and 1st heterochr. segment), recombination in hypoploids: - 32.6 - gl3 female, - 40.1 - gl3 male; - 9.0 - gl4 female, - 19.3 - gl4 male --Robertson, r388

Monosome-4 plants typically retain tassel within leaf whorl, delayed flowering; 40.2% plump pollen --Weber, r510

Oleic-linoleic ratio association, wx T4-9b vs. inbred X-187 --Widstrom &, r520

Krn4 (kernel row number) association with Asr1 --Miranda &, 59:23

o1 - 38.2+4.8 - Ga1-S --Miranda &, 59:24

Aco1 (aconitase, cytosolic?, monomeric) probably on 4S --Wendel &, 59:90

o1 - 45 - dcr*-1005A --McCormick &, 59:91

Zein IEF band 22, 22.5kd (22-22.5), and 22-19.6 are lost with Df in Dp-Df of T4-10f (4L.94, 10L.14), and 53-19.6 and 10-19.6 are linked with them; 28-22.5 and 33.5-22.5 are linked and on 4; 21.5-22.5, 21.5-19.6 and 53-19.6 are on 4L; 10-19.6 is linked to 22-22.5 and 22-19.6 --Ottoboni &, 59:99

CHROMOSOME 5
Got2-3, -7, -10 alleles; Got3-n, -2, -6 alleles; Mdh5-7.7, -8, -13, -14, -14.4, -16.7 alleles; Pgm2-n, -0.3 alleles --Doebley &, r104; Smith &, r439
K5L1, K5S1 location, distribution --Kato, r204
TB-5La hypoploids: a2 - 4.4 - female, a2 - 18.9 - male; btl - 0.2 - female, btl - 3.6 - male --Robertson, r388
Oleic-linoleic acid ratio association, wx T5-9a vs. inbred GE82 --Widstrom &, r520
TB-5Ld uncovers bv1, ael, prl, lw2, gl8, but not btl, tdl --Beckett, 59:39
Hsfl (was Hsf*-1595; hairy-sheath frayed) - 39 - T5-9cwx; - 15 - T5-9a wx; Hsf*-1603 - 4 - T5-9a wx--Bird &, 59:42

CHROMOSOME 6
rDNA gene number in 16 genotypes, ranging from 5000 to 23,000 per 2C; no obvious relation with rRNA content --Buescher &, r57
Enpl-1, -4, -6, -7, -8, -9, -10, -12, -14 alleles; Idh2-2.5, -3.8, -5, -5.5, -8 alleles; Mdh2-.02, -.25, -.31, -.35, -4, -4m, -5m, -5.8, -7.5, -7.7, -8.5, -9.3 alleles; Pgdl-0.5, -1.8, -9, -12, -14 alleles --Doebley &, r104; Smith &, r439
K6L1, K6L2, K6L3 location, distribution --Kato, r204
rDNA restriction pattern alterations in tripsacoid maize (one instance a new SphI site in spacer between 26S and 17S) --Lin &, r254
rDNA17S base sequence --Messing &, r291
Monosome-6 plants have shortened internodes, upright leaves, delayed flowering; 46.9% plump pollen --Weber, r510
Hex2 (hexokinase, cytosolic, monomeric); Pgdl - 3 - Enpl - 29 - Pl1 - 13 - Hex2 - 43 - Idh2 - 2 - Mdh2; TB-6Lc uncovers Enpl, Hex2, Idh2, Mdh2 but not Pgdl --Wendel &, 59:89

CHROMOSOME 7
K7L1, K7S location, distribution --Kato, r204
Monosome-7 plants have soft, wrinkled, thin leaves, delayed flowering; 37.0% plump pollen --Weber, r510
Leaf Freckles and Wilt (LFW) (Goss's Wilt) R/S association with wx T7-9a, T7-9b in inbred M14 (R) vs. A632 (S) --Rocheford &, 59:57
yel*-7748 is about 30 units to the right of gl1 --Whalen, 59:109

CHROMOSOME 8
Mdh1-0.3, -0.9, -2.8, -3.2, -4, -8.5 alleles --Doebley &, r104; Smith &, r439
K8L1, K8L2 location, distribution --Kato, r204
Monosome-8 plants have narrow leaves, slender stalks, delayed flowering; 25.8% plump pollen --Weber, r510
Sdwl (was Sdw*-1592; semi-dwarf) - 22 - T8-9d wx; - 8 - T8-9(6673) wx --Bird &, 59:42
Tpi3 (triose phosphate isomerase, cytosolic, monomeric) on 8? --Wendel &, 59:90
Uncovered by TB-8Lc: cp*-1387A, cp*-1392A, cp*-1405A, crp*-1533, de*-1386A, de*-1897, o*-1214A, o*-1383, ppg*-199B, rgh*-1285, sh*-1530, v*-7B, v*-25, v*-29B, v*-358C, v*-779A, v*-826, wl*-203A, wl*-1982, wl*-1985, but not ub*-76C; allelic to prol: crp*-1058, crp*-1121, rgh*-1154A, cp*-1528 --Neuffer, 59:109

CHROMOSOME 9
wx-m7, Ac: 4.3kb, 2.5kb upstream from wx-m9 and indistinguishable from it by restriction analysis --Behrens &, r30
Acpl-0.05, -0.1, -0.2, -2.5, -5 alleles --Doebley &, r104; Smith &, r439
shl-m5933 contains 30kb Ds insertion; 4.2kb sequenced at its 3' end contains 2 identical 2040bp segments, reversed, with one inserted in the other (double Ds); 11bp inverted terminal repeats, an 8bp direct insertion repeat, other internal repeats; one sizeable ORF --Doring &, r107
Shl progenitor, shl-m6233, shl-m5933 (and 6 Sh revertants from it), shl-m6258, shl-m6795 compared in restriction maps --Fedoroff &, r124
bzl-m2 Ac-containing fragment isolated by homology to Ac9; adjacent segment isolated from genomic Bz-r DNA, as a probe on a Bz revertant from bzl-m2, yields insertion-free fragment, and on shl-bzl-x2 and shl-bzl-x3 no fragment --Fedoroff &, r120
K9L2, K9S location, distribution --Kato, r204
shl-5582 (unst.), -5584 (unst.), -5586 (unst.), -5588, -5592, -5594 (unst.), -5596 (unst.) alleles and bzl-5598 arose in AR strains; alterations in restriction maps --Mottinger &, r311
wx-m9: Ac9 base sequence 4563bp with imperfect 11bp terminal repeat; two ORFs (839, 210 aa's), the larger being altered in a derivative, Ds9; comparisons to several other Ds isolates and revertants; Ac9 base sequence corrections reveal 9 ORFs, of which one (D3) is altered in Ds9 --Pohlman &, r364, r365
TB-9Sb (B breakpoint prob. in 1st heterochr. segment), recombination in hypoploids: - 0.2 - wxl female, - 0.7 - wxl male; - 11.7 - vl female, - 29.5 - vl male; TB-9La (B breakpoint prob. in 3d heterochr. segment) hypoploids: shl - 32.3 - female, shl - 46.0 - male --Robertson, r388
wxl-m8 Spm-I8 insertion 2kb in transcribed region, 3bp target site duplication, 13bp inverted terminal repeats, excised in presence of En; other multiple insertions; restriction map --Schwarz-Sommer &, r414
Monosome-9 plants have narrow, stiff leaves, anthesis not notably delayed; 3.4% plump pollen --Weber, r510
shl-m6233 Ds insertion, 4kb, consists of 2 identical 2kb elements, one inserted in the other, identical to double Ds in shl-m5933; 8bp insertion duplication; restriction map and partial sequence; revertant retains a 6bp duplication --Weck &, r511
Cl-I-m836976 En-mediated, from Cl-I; cl-shl-836882 En-mediated, from Cl-I Shl; cl-836957 from Cl-I; wxl-84-4 (contains En-1) and wxl-84-11 En-mediated; Cl-m(r) allele, Uq-responsive; cl-m804531 allele, Uq-controlled --Peterson, 59:2,3
bzl-n(rcy)-shl-835211w, -835211x-1 from bzl-rcy Shl; bzl-rcy824325w, 812215 from bzl-m805137 (Cy-mediated) --Schnable &, 59:6
Shl genomic sequence vs. cDNA: restriction map and 16-exon structure, TATTTATT box to AATAAA --Werr &, 59:32
Ac7 from wx-m7 and Ac9 from wx-m9, base sequences identical; 3 ORFs (2 coding candidates)--Muller-Neumann &, 59:33
wxl-844 En insert is 8.4kb, in an intron; wxl-m8 Spm-I8 insert is 2.2kb in an exon and has 1kb and 1.2kb of ends of Enl --Berndtgen &, 59:34

TB-9Sb uncovers ba*-s, 16, yg*-5588, but not w*-6-10b or w*-034-5; 16 and yg*-5588 and w*-s9000 not allelic to wd; yg*-5588 not allelic to 17; TB-9Lc uncovers v*-8587, w*-8889, yel*-034-16, wlv*-EM53, wl*-EM41, v*-MS61, rghd*, dek13, fdt*, cp*-1381, de*-1409, but not w*-6-10a or w*-034-5; TB-9La uncovers w*-8950; w*-s9000 not allelic to pgl2 or arl; wlv*-EM53 not allelic to pgl2, arl, or vl; w*-8950 not allelic to vl --Coe, 59:40
Les*-2005 (lesion) - 0 - T8-9(6673) wx; - 0 - T1-9(4995) wx; - 8 - T7-9a wx; - 11 - T1-9(8389) wx; - 7 - T4-9g wx; - 0 - T4-9b wx; - 3 - T6-9a wx --Bird &, 59:42
sh1-5586, -5588 alleles arose in AR stocks; plump seed segregation suggests sh1 with Inh1 (inhibitor of minor sucrose synthetase) has shrunken endosperm, sh1 inh1 plump --Mottinger, 59:56
Acp1 (acid phosphatase; supplants Ap1, Ap2, Ap3) on 9 --Wendel &, 59:90
d*-6608 is allelic to d3 --Nelson, 59:109

CHROMOSOME 10
Zp22/2 (zein IEF band 2, 22kd, presence/absence): dosage-dependent in trisome-10 tests, 0 recombinants with o7 in 122 BC progeny --Binelli &, r38
Glu1-1.2, -1.5, -1.8, -3.5, -4.5, -7.2, -7.5, -7.8, -8, -11, -12 alleles --Doebley &, r104; Smith &, r439
K10L1, K10L2 location, distribution --Kato, r204
R1-st element I-R transposes frequently to near sites, modifies R1-st expression; deletions arise by apparent unequal crossing over between I-R and Mst; alleles r-g:nc3-5, r-g:Sd, r-g:e; r-r:n19, r-r:n35, r-r:n101, r-r:W22, R-g:1, R-g:8pale; deletions r-g:de4-128, r-g:de4-137, r-g:de4-139, r-g:de4-142, r-x1 --Williams &, r524; Kermicle, r208
TB-10Sc hypoploids: - 27.2 - r1 female, - 43.1 - r1 male; - 0.0 - bf2 female, - 2.0 - bf2 male; TB-10La (B breakpoint at junction of prox. euchr. and 1st heterochr. segment) hypoploids: oy1 - 21.7 - female, oy1 - 29.1 - male; bf2 - 6.8 - female, bf2 - 14.6 - male; y9 - 11.5 - female, y9 - 20.9 - male --Robertson, r388
Monosome-10 plants have delayed flowering; 43.9% plump pollen --Weber, r510
r1-ch(Hopi) allele, - 2.3 - o7; Sn1 (red scutellar node) 1-2 units distal to r1 --Gavazzi &, 59:79
Sad1 (shikimate dehydrogenase, plastidial, monomeric) on 10 --Wendel &, 59:88
Zein IEF band 41, 14.3kd (41-14.3), and 49-19.6 are included in duplication of 10L in Dp-Df from T4-10f (4L.94, 10L.14) --Ottoboni &, 59:99
l1 is not expressed in sr2 plants hemizygous for the R1 to Lc interval --Kermicle, 59:110

UNPLACED
Cat3-5.5, -6, -9+, -9.5, -10, -10.5 alleles --Doebley &, r104; Smith &, r439
Cg2: independent of ra2, and of bm2, lg1, su1, y1, gl1, j1, wx1, gl; unstable, mutates to normals that revert --Lysikov &, r261
Lfy1 (leafy): Dominant, increased number of leaves --Shaver, r425
Lmc1, Lmc2 (long mesocotyl) linked, 6-12% recombination --Miranda &, 59:23
ms22-A632A, ms23-A619B (allelic to ms*-Bear7), ms24-LT designated & characterized --West &, 59:87
Aco2 (aconitase, mitochondrial?), Aco3 (mitochondrial), Aco4 (mitochondrial?, monomeric); Adk1 (adenylate kinase, plastidial, monomeric); Tpi1 (triose phosphate isomerase, plastidial, dimeric); Tpi2 (plastidial, dimeric) --Wendel &, 59:87
Mu-L element (1.7kbp) contains 300bp not present in Mu-1; Mu-S1 (1.0kbp); Mu-S2 (800bp) and Mu-S3 (650bp) lack the terminal repeats --Taylor &, 59:96
ora2-15, allelic to ora2-13 --Dollinger, 59:104

CHLOROPLAST
LS 5' end, only the 2d of 2 met-ser coding sites (18bp apart) initiates translation, with tRNA1-ser --Bloom &, r43
Putative 16SrRNA binding site sequences for rp-S4 (ribosomal protein S4), rcL (LS), cf1B (coupling factor beta), cf1E (epsilon), 1.6kb transcript, and 4 unidentified ORFs; map including previous localizations and ORFs and PGs (photogenes) --Bogorad &, r44, r45
tRNA2ile base sequence with intron --Burkard &, r58
LS mRNAs, 1.6 and 1.8 kb, 238 nucleotides different at 5' terminus; ratio condition-dependent --Crossland &, r87
tRNA tV-UAC (val) base sequence with intron; tS-UGA (ser) base sequence --Krebbers &, r223
rDNA homology to rDNA probe from E. coli --Stern &, r453
Homologies between ctDNA and mtDNA sequences --Stern &, r455
tRNA arg1, ile1, leu3, lys, met1, ser1, ser2, asn2, asp, thr1, tyr, pro, trp map locations --Weil &, r512, r513

MITOCHONDRION
rDNA18S: nucleotide sequence, comparison to ct-rRNA16S & nuclear rRNA17S --Chao &, r71
rDNA26S base sequence; 3546 bases; comparison with maize ct 23S rDNA, human mt 16S rDNA, yeast mt 21S rDNA, yeast nuclear 25S rDNA --Dale &, r89
COB (apocytochrome b gene) base sequence: 1164bp, no introns; preceded by sequence 5'-AGTTGTCA (potential ribosome binding site) --Dawson &, r93
COII (cytochrome oxidase subunit II; was mox1), 780bp, one intron; no introns in COI (1581bp) --Leaver &, r241
mtDNA from normal main genome: One S1-hybridizing region lacks the S1 end regions, and an S2-hybridizing region lacks one S2 end; homologies to R1 and R2 are greater --Levings &, r249
Sequences in main genome homologous to S1 and S2 are flanked by common sequences, sigma and psi; recombinations yield linear and internal variants of genome --Lonsdale &, r255; Schardl &, r408
Only minor, stoichiometric alterations in restriction patterns for Black Mexican kept in cell suspension cultures 4 years --McNay &, r286
Correlated changes of 8kb and 20.5kb XhoI bands in NCS2, of 20kb and 16kb XhoI bands in NCS3 --Newton, r328
Two single-stranded RNAs (2900 bases, 750 bases)of cms-S and RU cytoplasms do not hybridize to the DNA of S or R plasmids or other mtDNA, but do hybridize to dsRNA of LBN cytoplasm --Schuster &, r411
LBN (L cytoplasm in inbred W182BN) contains two double stranded RNAs, MW 1.9 and 0.5 x$10^6$) --Sisco &, r434
rDNA probe from E. coli hybridizes to ct rRNA16S sequence and mt rRNA18S, weakly with mt rRNA26S --Stern &, r453
Homologies between ctDNA and mtDNA sequences --Stern &, r455

Fertile revertants of cms-T, induced by gamma or EMS, have N restriction patterns but no 2.35kb plasmids --Vuillaume &, r502

mtRNA screening of genomic library indicates about 50kb (approx. 40 transcripts) of genome is transcribed; restriction map and location of a 1kb transcript --Walbot &, r506

Distributions, by probes (for 1.4 & 1.9 minicircular and S1/S2 minilinear DNAs) and visually (for 2.3 & 2.1 minilinear) show diversity in maizes and cytoplasms, and in teosintes, Z. perennis (ZP), Z. diploperennis (ZD); 2.1 or 2.3 in all maizes, not found in Guatemala teosinte (GT) or ZP or ZD; GT & ZP contain integrated S sequences; D1/D2 only in ZD; R1/R2 only in Racimo de Uva (RU) --Pring &, 59:49

Fertile revertant from cms-T, T4, studied more closely, shows a small deviation in the 6.6kb XHoI fragment in which other revertants show substantial rearrangements --Wise &, 59:50

2.3kbp linear plasmid termini are similar to S plasmids; homologous form in main genome of N, cms-T, C, and S --Bedinger &, 59:96

Fertile revertants from WF9 strains of cms-S types J, G and R, maternally inherited (J', G', R'), retain unchanged S1 and S2 plasmids and show diverse alterations in restriction patterns of main DNA --Ishige &, 59:98

Fertile revertants of cms-S in M825 lose S1 and S2; revertants in WF9 (5 cases), RD WF9 (4 cases) and ML WF9 (1 case) retain S1 and S2, and lose them when backcrossed to M825 --Escote &, 59:100

CDNA/GENOMIC CLONES/PROBES

Knob-specific repeat probe, 180bp, hybridizes in situ to knobs in teosintes, Z. diploperennis, Tripsacum, not in Coix spp.; variants affecting single bases are common; 202bp variant in P100 and teosintes --Dennis &, r98

Cin1-homologous sequences: locations heterogeneous, disperse; restriction maps, homologies inter se, heteroduplex data for 14 clones --Gupta &, r152, r153

Zein genomic clone Z7 from W22, base sequence 1587bp including noncoding sequences; comparisons to genomic clone zA1 & cDNA clones pZ22.1 and B49 --Kridl &, r224

Zein cDNA clones cZ19A-1, cZ19B-1, cZ19B-2, gZ19AB-1, cZ19A-2, cZ19C-1, cZ19C-2, cZ19D-1, cZ22A-1, cZ22A-2, cZ22B-1, cZ22C-1, cZ15A-2, cZ15A-3; genomic clones ZG19.7, ZG19.27, ZG19.12, ZG19.30, ZG19.31, ZG19.32: restriction maps, cross-homologies --Larkins &, r236; Marks &, r272

Actin genomic clone MAc1, 2420bp sequence, 3 introns; multigene family --Shah &, r421

Cin1 base sequence, 6bp inverted terminal repeats (terminal 5 identical to copia) --Shepherd &, r427

Zein probes for MW 19 & 22kd estimate 70-80 sequences in W64A; MW 15kd probe estimates 2-3 --Wilson &, r528

Teo1, 3kb, consists of element Cin2, 1.2kb with 140bp direct repeats at the ends flanked by 3bp duplication, containing Cin3 element insertion, 1.8kb with 634bp inverted repeats flanked by 9bp duplication --Blumberg vel Spalve &, 59:35

RESISTANCE/TOLERANCE/HERITABILITY

In vitro culture aptitude (callus, roots, shoots), in diallel trial among 8 strains, shows significant heritability and high SCA, and reciprocal effects --Beckert &, r29

Blight (Bipolaris maydis (Nisik.) Shoemaker) R/S in RbU-W and DIC vs. UVE and ZPSc-58c varieties monogenic recessive and allelic --Faluyi &, r118

Ear rot (Gibberella zeae) R/S in diallel shows significant GCA --Hart &, r166

MDMV R/S in inbreds, 1 to 5 genes --Mikel &, r299; Rosenkranz &, r391

Callus growth, in diallel of 6 inbreds, showed significant GCA and SCA, and cytoplasmic effects --Nesticky &, r324

Ear fasciation in Portuguese varieties not allelic to ra1, ra2 or ra3; highly heritable, correlated with ear diameter and kernel-row numbers --Pego &, r343

Isozyme associations for 8 and 9 loci in two populations, with 20 morphological characters --Pollak &, r369

Southern Corn Rust (Puccinia polysora Underw.) resistance, homozygous in 4 populations, one or two gene models with complete, partial or no dominance; each includes factor allelic to Rpp9 or closely linked --Scott &, r417

Ear rot (Fusarium moniliforme Sheld.) R/S conditioned by pericarp genotype --Scott &, r416

Southern Corn Leaf Blight (B. maydis) race 0 reaction in mature plants influenced by rhm1 (seedling resistance); NC250 resistance independent of seedling resistance, additive --Thompson &, r474

European Corn Borer (Ostrinia nubilalis Hubner) leaf feeding and DIMBOA levels responsive to recurrent selection for either leaf feeding or increased DIMBOA levels in BS1 synthetic --Tseng &, r489

Oleic/linoleic levels in GE82 (high/low), X-187 (low/high), associations with wx translocations --Widstrom &, r520

Long mesocotyl, two linked factors --Miranda &, 59:23

Leaf Freckles and Wilt (LFW) (Goss's Wilt) R/S association with wx T7-9a, T7-9b in inbred M14 (R) vs. A632 (S) --Rocheford &, 59:57

Kernel hardness and kernel density negatively correlated with tryp content, but separated by selection in Puna variety --Broccoli &, 59:67

Thin pericarp of Z. perennis partly dominant over thick Gaspe, segregates from enclosed/naked --Bertoia &, 59:68

Multilayer aleurone appears in F2 from Z. perennis x Gaspe --Bertoia &, 59:69

Associations in Z. perennis x Gaspe F2 and F3 of endosperm protein content with late flowering, high tillering, distichous spikes, single female spikelets, low kernel number --Ferrari &, 59:69

Regenerability from tissue culture, for A188 x A619, significant additive effects; for A188 x B73, significant additive and dominance effects --Armstrong &, 59:92

Ligate's Corner:
If Cin is cornucopia,
to thin is cornutopia

## V. REPORT OF MAIZE GENETICS COOPERATION STOCK CENTER

During 1984 there were 172 seed requests and 2104 seed packets were sent. This was once again the highest figure ever. Domestic requests amounted to 142 for 1618 packets while there were 30 foreign requests for 486 packets.

The estimated uses of the seed were distributed among the following categories:

| | |
|---|---|
| Geneticists | 57% |
| Physiologists | 13% |
| Breeders | 8% |
| Educators | 7% |
| Genetic Engineers | 15% |

In 1984 we were able to complete the transfer of the entire inventory onto the computer. We have upgraded the hard disk to 44 megabytes that enables us to keep the records on line at all times.

We were able to get the first crosses completed this summer in the program to convert selected pairs of markers into the inbreds which were discussed in the March 1984 meeting.

Dr. Nina Fedoroff was kind enough to supply the Coop with some of her materials which we are in the process of increasing so that they can be added to the stock list as soon as possible. Similarly, Dr. Jack Beckett has provided several new B-A translocations that we are increasing.

We have added a series of stock numbers which will make the administrative handling of requests a bit easier. When you make a request please use this format:

702B o2 v5 ra gl Prefer v5 and ra homozyg.

The following listing shows the stocks that are available. Translocation stocks remain as listed in the 1981 News Letter.

Requests for seed and information should be sent to:


Dr. Gilbert B. Fletcher
Department of Agronomy
University of Illinois
S-123 Turner Hall
1102 S. Goodwin Ave
Urbana, IL 61801

Phone (217) 333-9644
Lab (217) 333-6631

Chromosome 1

101A sr zb4 P-WW
101B sr P-WR
101C sr P-WW
102A sr P-WR an gs bm2
102B sr P-WR an bm2
103A sr P-RR an bm2
103B sr P-RR gs bm2
103C sr P-WR bm2
103D vp5
103E zb4 ms17 P-WW
103F zb4 ms17 P-WW rs2
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
106A zb4 P-WW bm2
105E ms17
106B ts2 P-RR
106C ts2 P-WW bm2
106D ts2 P-WW br bm2
106E ts2 br f bm2
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
108C P-RR br f an gs bm2 rd
108D P-RR br f an gs bm2 id
108E P-RR br f an gs bm2 v*-8983
108F P-RR br f an gs bm2 v*-8943
109A P-RR an ad bm2
109B P-RR an gs bm2
109C P-RR ad bm2
110A P-WR an Kn bm2
110B P-WR an ad bm2
110C P-WR an bm2
110D P-WR an br bm2
110E P-WT=WR an bm2
110F P-WR br Vg
110G P-WR br f gs bm2
110H P-WR br f an lw gs bm2
110I P-WR br f bm2 id
111A P-WW rs2
111B P-WW rs2 br f
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
113A as br2
112F as rs2
113B rd Hy
113C br f
113D br f bm2 v*-5588
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 bz2 m : A A2 C Pr
116A bz2 M : A A2 C R Pr
116B bz2 ad bm2 A C R
116C an bm2
116D an-bz2-6923 (apparent def. incl.an & bz2)
117A br2
117B br2 bm2
117C br2 an bm2
117D tb-8963
117E Kn
118A Kn Ts6
118B Kn bm2
118C lw
119A Adh1-S
119B vp8
119C gs
119D gs bm2
119E Ts6
119F bm2
120A id
120B nec2
120C ms9
120D ms12
121A ms14
121B mi
121C D8
121D Lls
125A Les2
122A TB-1La (1L.20)
122B TB-1Sb (1S.05)

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 fl v4
203A ws3 lg gl2 B gs2 v4
201E ws3 lg gl2 B ts
201F ws3 lg gl2 b
203C ws3 lg gl2 b sk
202A ws3 lg gl2 b sk v4
202B ws3 lg gl2 b gs2 v4
202C ws3 lg gl2 b fl v4
202D ws3 lg gl2 b sk fl v4
202E ws3 lg gl2 b v4
203B al
203D al lg
203E al lg gl2 B sk v4
205A al lg gl12 b
204A al lg gl2 b sk v4
204B al lg gl2 b sk fl v4
205B lg
205C lg gl2
206A lg gl2 B
207A lg gl2 B gl11
206B lg gl2 B gs2
206C lg gl2 B gs2 v4
206D lg gl2 B gs2 Ch
206E lg gl2 B gs2 sk Ch
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
209A lg gl2 b gs2 sk Ch
209B lg gl2 b gs2 v4
209C lg gl2 b gs2 v4 Ch
209D lg gl2 b gs2 sk v4 Ch
209E lg gl2 b sk
209F lg gl2 b sk fl
209G lg gl2 b sk fl v4
210A lg gl2 b sk v4
212A lg gl2 b wt v4
211A lg gl2 b fl
212B lg gl2 b fl v4
212C lg gl2 b fl v4 Ch
212D lg gl2 b v4
212E lg gl2 b v4 Ch
213A lg gl2 mn v4
213B lg gl2 wt
211B lg gl2 b gs2 wt
213C lg gl2 w3
213D lg gl2 w3 Ch
213E lg gl2 Ch
214A lg b gs2 v4
214B lg Ch
208H gl2
214C d5 = d*-037-9
214D B gl11
214E B ts
215A gl14
215B gl11
215C wt
215D mn
215E fl
216A fl v4 Ch
216B fl Ht v4
216C fl Ht v4 Ch
216D fl w3
216E fl v4 w3
216F fl w3 Ch
216G fl v4 w3 Ch
217A ts
217B v4
217C v4 w3 Ht Ch
217D V4 Ht Ch
218A w3
218B w3 Ht
217E w3 Ht Ch
218C w3 Ch
218D Ht (A & B source)
218E ba2
219A R2: r A A2 C
219B r2: r-g A A2 C
219C Ch
221A gs2
220A Les
220B 2 2Trip Trip2/ ws3 lg gl2
222A TB-1Sb-2L4464
222B TB-3La-2S6270
223A Primary Trisomic 2

Chromosome 3

301A cr
301B cr d
301C cr d Lg3
301D cr pm ts4 lg2
301E cr ts4 na
302A d-tall = d*-6016 (short)
303A d rt Lg3
303B d Rf lg2
304A d ys3
304B d ys3 Rg
304C d ys3 Rg lg2
305A d Lg3
305B d Lg3 gl6
305C d Lg3 ts4 lg2
305D d Rg
306A d Rg ts4 lg2
307A d pm
304D d yg*-(W23)
307B d ts4 lg2
308A d ts4 lg2 a-m : A2 C R Dt
308B d ts4
306B d gl6
308C d lg2 a-m A2 C R Dt
308D d a-m A2 C R Dt
308E ra2
308F ra2 Rg
309A ra2 Rg ts4 lg2
309B ra2 ys3 Lg3 Rg
309C ra2 ys3 Rg
309D ra2 Rg lg2
309E ra2 pm lg2
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
312A ys3 gl6 lg2 a-m et : A2 C R Dt
312B ys3 ts4
312C ys3 ts4 lg2
312D Lg3
312E Lg3 Rg pm
313A gl6
314A gl6 lg2 A : A2 C R
314B gl6 lg2 A-b et : A2 C R Dt
314C gl6 lg2 a-m et : A2 C R Dt
315A pm lg2
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 : A2 C R Dt
318A ig
318B ba
318C y10
320A lg2
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
320B na
320C na lg2
320D A sh2 : A2 C R B Pl dt
321A A-d31 : A2 C R
321B A-d31 : A2 C R pr dt
321C A-d31 : A2 C R B Pl dt
321D A-d31 : A2 C R Dt
321E A-d31: A2 C R pr Dt
322A A-d31 sh2 : A2 C R B Pl dt
322B A-d31 sh2 : A2 C R Dt
322C A-d31 sh2 : A2 C R B Pl Dt
322D A-d31 et : A2 C R Dt
322E 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 C R 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
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

Chromosome 3 (continued)

326A sh2
326B vp
326C Rp3
325F pgI4
325G a3
326D te
330A h
326E yel*-5787
327A TB-3La (3L.10)
327B TB-3Sb (3S.50)
327C TB-3Lc (distal to 3 La (3L.10)
328A Primary Trisomic 3

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
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
406A la su gl3 o
406B la su bt2 gl3
406C f12
406D f12 su
407A f12 bt2
407B f12 su bm3
407C f12 su gl4 Tu
407D su
407E su-am
408A su bt2 gl4
408B su bm3
408C su zb6
408D su zb6 bt2
409A su zb6 Tu
410A su zb6 gl3 dp
411A su gl4 j2
411B su 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
408E bm3
414B gl4
414C gl4 o
416A Tu
416B Tu-1 1st
416C Tu-1 2nd
416D Tu-d
416E Tu-md
416F Tu gl3
415A j2
415B j2 c2 : A A2 C R
415C j2 C2 : A A2 C R
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
420A Dt4 su : a-m A2 C R
421A TB-4Sa (4S.20)
421B TB-1La-4L4692
420B TB9Sb-4L6504 (9S.40-.83: 4L.09)
421C TB7Lb-4L4698 (7L.30-.74: 4L.08)
422A Primary Trisomic 4

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
502A gl17 a2 bt v2 : 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
503B A2 bm pr ys eg : 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
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
506D 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 v3 pr : 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
512A a2 bt v2 : A C R
512B a2 v3 pr : A C R
513A a2 pr : A C R
513B a2 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
516B bt
516C ms5
517A v3
516D td ae
517B ae
518A sh4
518B gl8
518C na2
518D lw2
519A ys
519B eg
519C v2
519D yg
520A ms13
520B v12
520C br3
521A nec3
522A TB-5La
522B TB-5Lb
523A Primary Trisomic 5

Chromosome 6

601A rgd po y
601B rgd po Y
601C rgd y
601D rgd Y
601E po = ms6
601F po y pl
601G po y Pl
602A po y wi
602B po Y pl
602C y = pb = w-m
602D y rhm
603A y l10
603B y l11
603C y l12
603D y w15
603E y pb4
604A y pb4 pl
604B y pb4 Pl
604C y si
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

Chromosome 6 (continued)

607B y pl Bh : c sh wx A A2 R
607C y su2
608A Y l10
608B Y l12
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 l*-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
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 gl ij
703A o2 v5 gl
704A o2 ra gl ij
704B o2 ra gl sl
705A o2 gl
705B o2 gl sl
705C o2 ij
705D o2 bd
707A y8 v5 gl
707B in : A2 pr A C R
707C in gl : A2 pr A C R
707D v5
707E vp9
707F vp9 gl
708A ra
708B ra gl ij bd
709A gl
709B gl-m
710A gl Tp
710B gl mn2
711A Tp
711B ij
712A ms7
712B ms7 gl Tp
713A Bn
713B bd
714A Pn
714B o5
714C o5 mn2 gl
714D va
715A Dt3 : a-m A2 C R
716A V*-8647
716B yel*-7748
717A TB-7Lb (7L.30)
718A Primary Trisomic 7

Chromosome 8

801A gl18
801B v16
801C v16 j
801D v16 ms8 j
801E v16 ms8 j nec
802A v16 ms8 j gl18
803A ms8
803B nec
804A v21
805A f13
805B f13 j
806A TB-8La (8L.70)
807A Primary Trisomic 8

## Chromosome 9

| Stock | Genotype |
|---|---|
| 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 |
| 902A | yg2 c sh bz 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-g |
| 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 |
| 903C | C sh bz wx bm4 : 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 |
| 905A | C sh wx K-S9 : A A2 R |
| 905B | C sh ms2 : A A2 R |
| 905C | C bz Wx : 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 |
| 906F | C : A A2 R B Pl |
| 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 |
| 909B | c bz wx : 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 |
| 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 |
| 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 |
| 915A | wx* |
| 915B | wx-a |
| 915C | wl1 |
| 914A | wx d3 |
| 914B | wx d3 wl1 |
| 914C | wx d3 v gl15 |
| 914D | wx d3 gl15 |
| 914E | Wx pg11 : y pg11 |
| 914F | wx pg12 : y pg11 |
| 914G | Wx pg12 : Y pg11 |
| 914H | wx pg12 : Y pg11 |
| 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 bm4 |
| 918C | bk2 Wx |
| 918D | Wc |
| 919A | bm4 |
| 919B | bm4 Bf |
| 919C | l6 |
| 919D | l7 |
| 920A | yel*-034-16 |
| 920B | w*-4889 |
| 920C | w*-8889 |
| 920D | w*-8951 |
| 920E | w*-8950 |
| 920F | w*-9000 |
| 920G | Tp9 N9 N3 Df3 |
| 921A | TB-9La (9L.40) |
| 921B | TB-9Sb (9S.40) |
| 921C | TB-9Lc |
| 922A | Primary Trisomic 9 |

* Additional waxy alleles available from collection of O. E. Nelson

## Chromosome 10

| Stock | Genotype |
|---|---|
| X01A | oy |
| X01B | oy R : A A2 C |
| X01C | oy bf2 |
| X02A | oy ms11 |
| X01D | oy bf2 R : A A2 C |
| X02B | oy bf2 ms10 |
| X02C | oy zn R : A A2 C |
| X02D | oy du R : A A2 C |
| X02E | oy du r : A A2 C |
| X02F | oy sr2 |
| X02G | oy zn |
| X03A | sr3 |
| X03B | Og |
| X03C | Og B Pl |
| X04A | Og du R : A A2 C |
| 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 |
| X06A | bf2 r sr2 : A A2 C |
| X06B | nl zn g R : A A2 C |
| X06C | nl g R : A A2 C |
| X07A | nl g r : A A2 C |
| X07B | nl g R sr2 : A A2 C |
| X07C | y9 |
| X08A | y9 v18 |
| 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 r v18 : A A2 C |
| X09E | li g r v18 : A A2 C |
| X09F | ms10 |
| X10A | du |
| X10B | du v18 |
| X10C | du o7 |
| X10D | du g r : A A2 C |
| 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 : A A2 C |
| 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 |
| X13C | g R-r K10 : 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 : A A2 C |
| 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 |
| X17B | r-r: A A2 C |
| X15D | r-ch Pl : A A2 C |
| X17C | R-mb : 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-sk : A A2 C |
| X18D | R-st : A A2 C |
| X18E | R-st Mst |
| X18F | R-st Mst 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

| Stock | Genotype |
|---|---|
| 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

| Stock | Genotype |
|---|---|
| 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

| Stock | Variety |
|---|---|
| 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

| Stock | Variety |
|---|---|
| E542A | Black Mexican Sweet Corn (with B-chromosomes) |
| E542B | Black Mexican Sweet Corn (without B-chromsomes) |
| E642A | Knobless Tama Flint |
| E442A | Gaspe Flint |
| E642B | Gourdseed |
| E742A | Maiz Chapolote |
| E742B | Papago Flour Corn |
| E742C | Parker's Flint |
| E842A | Tama Flint |
| E842B | Zapaluta Chica |

## Tetraploid Stocks

| Stock | Genotype |
|---|---|
| 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 WF9-(T) rf rf2
C836B WF9 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)

## VI. MAILING LIST

Adeniyi, Gideon I., Dep Ag & H Ec, Coll Ed, Kwara Tech, Ilorin, Nigeria
Agrawal, B. D., G. B. Pant Univ Agr Tech, Pantnagar 263145, India
Agrema, Hesham A., 42 Mumphis Street, Camp Caesar, Alexandria, Egypt
Ainsworth, Thomas C., Ainsworth Seed Company, Mason City, IL, 62664, 217-482-3281
Albertsen, M. C., Pioneer Hi-Bred Internatl, P. O. Box 38, Johnston, IA, 50131, 515-270-3648
Alexander, D. E., Dept Agron, S-110 Turner Hall, Univ Illinois, 1102 S. Goodwin, Urbana, IL, 61801, 217-333-4254
Ali, S. Mahboob, Genetics & Plant Breed, College of Agriculture, Rajendranagar, Hyderabad-500 030, India
Alleman, M., Dept Genetics, 345 Mulford, Univ of California, Berkeley, CA, 94720, 415-642-7085
Altschuler, Marsha I., Dept. of Biology, Washington University, St. Louis, MO, 63130
Alvey, David, FFR Cooperative, 4112 East State Road 225, West Lafayette, IN, 47906, 317-567-2115
Amano, Etsuo, Inst of Radiation Breeding, PO Box 3, Ohmiya-machi, Naka-gun, Ibaraki-Ken 319-22, Japan
Anderson, E. L., Bldg 007, Rm 251, BARC-West, Beltsville, MD, 20705, 301-344-3148
Andrew, R. H., Department of Agronomy, University of Wisconsin, Madison, WI, 53706, 608-262-2587
Arikoglu, Osman, Sakarya Zirai Arastirma Inst, P.K. 25, Adapazari, Turkey
Armstrong, Charles L., Dept Agron & Plant Genet, University of Minnesota, St. Paul, MN, 55108, 612-646-8716
Arntzen, C. J., MSU-DOE Plant Res Lab, Michigan State Univ, East Lansing, MI, 48824, 517-353-2270
Ashman, R. B., Dept of Botany, Purdue University, Lafayette, IN, 47907, 317-494-4634
Avila L., Gonzalo, Centro Eco-Genetico De, Pairumani, Casilla 128, Cochabamba, Bolivia
Axtell, John D., Department of Agronomy, Purdue University, West Lafayette, IN, 47907, 317-494-8056
Ayala-Osuna, Juan, Univ Ext Paul Julio Mesq F, Fac Med Vet Agron, 14870 Jaboticabal SP, Brazil, 0163-220260
Aycock, Harold S., Dept of Agronomy, Va Polytech Inst & State Univ, Blacksburg, VA, 24061, 703-961-6484
Ayers, J. E., Dept Plant Pathology, 211 Buckhout Lab, Pennsylvania State Univ, University Park, PA, 16802, 814-865-7776
Baenziger, P. Stephen, Monsanto, 800 N. Lindbergh Blvd, St. Louis, MO, 63167, 314-694-3924
Bahadur, Khan, NWFP Maize & Millets Res, Nowshera Cantt, Pakistan
Bailey, Zeno E., Botany Dept, Eastern Illinois Univ, Charleston, IL, 61920, 267-581-3525
Baker, Raymond, Pioneer Hybrid Seed Co, Johnston, IA, 50131, 515-270-2108
Baldi, G., Centro di Ricerche Sul Riso, C. P. 99, 27036 Mortara (PV), Italy
Balint, Andor, Agrar Egyetem, Novenynemesitestani, Tanszek, Godollo H-2103, Hungary
Ballenger, Steve, 4431 NW 86th St, Urbandale, IA, 50322
Banchero, Carlos, Santa Ursula, SA, Paraguay 577.P.1, 1057 Buenos Aires, Argentina, 311-0446-0510-4803-4846
Banerjee, U. C., Dept Biol, Box 19692, North Carolina Central Univ, Durham, NC, 27707, 919-683-6170
Bansal, R. K., Crop Research Substation, D.S.I.R., Cronin Road/R.D.1, Pukekohe, New Zealand
Banuett, F., Dept Biochem Biophys, Univ of California, San Francisco, CA, 94143, 415-666-4985
Bar-Tsur, Avri, Dept Maize Breed/Genet, Newe Yaar Exper Sta 31999, PO Haifa, Israel, 04-933-186
Baracco, Nestor P., Compania Continental S.A., C.C. 294, 2600 Venado Tuerto, Santa Fe, Argentina, 2815
Barbosa, Helio M., Dept Biologia Geral, Univ Vicosa, 36570-Vicosa, Minas Gerais, Brazil, 891-1790 R. 157
Barghoorn, E. S., Biological Laboratories, Harvard University, 16 Divinity Avenue, Cambridge, MA, 02138, 617-495-5224
Barry, Edward G., Department of Biology, Univ of North Carolina, Chapel Hill, NC, 27514, 919-962-6933
Baszczynski, C. L., Dept Plant Sciences, Univ of Western Ontario, London, Ontario, Canada, 519-679-2908
Bates, Lynn S., LSB Products, 731 McCall Road, Manhattan, KS, 66502, 913-537-9773
Bauman, L. F., Department of Agronomy, Purdue University, Lafayette, IN, 47907, 317-463-9455
Bayne, Peter D., PO Box 11596, Shorewood, WI, 53211, 414-263-4111
Bdliya, Paul Mari, Department of Agronomy, Iowa State University, Ames, IA, 50011
Beadle, G. W., 900 E Harrison Ave, Apt D-33, Pomona, CA, 91767, 714-621-0621
Bear, Robert P., Bear Hybrid Corn Company, PO Box 950, Decatur, IL, 62525, 217-422-5621
Beckett, Jack, Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-8214
Bedinger, Patricia, Dept of Biol Sci, Stanford University, Stanford, CA, 94305, 415-497-2609
Benner, Michael, Dept. Agron. & Plant Genetics, Univ. of Minnesota, St. Paul, MN, 55108
Bennett, Clark, Genetics Department, University of California, Berkeley, CA, 94720, 415-642-7085
Bennett, J. H., Dept of Genetics, Univ of Adelaide, Box 498, Adelaide 5001, S. Australia
Bennett, M. D., Plant Breeding Institute, Maris Lane, Trumpington, Cambridge CB2 2LQ, U K, 0223 840411
Bennetzen, Jeff, Department Biological Science, Purdue University, Lafayette, IN, 47907, 317-494-4763
Benson, Dirk L., CIMMYT Maize Program, Londres 40 Apdo Postal 6-641, 06600, Mexico D. F., Mexico
Benton, W. D., ARCO Plant Cell Research Inst, 6560 Trinity Court, Dublin, CA, 94568, 415-833-3455

Benzion, G., Department of Agronomy, N-107A Agr Sci Ctr North, Lexington, KY, 40546-0091, 606-257-7310
Bergquist, Richard R., Plant Pathologist, 401 East Sixth Street, El Paso, IL, 61738, 309-527-6000
Berndt, Ken, RBA Seeds Inc, Rainbow Lane, Olivia, MN, 56277
Berville, A., Lab d'Amelior Plantes, Univ Paris-Sud, Batiment 360, Orsay 91405, France
Bhat, D. K., RRL BR Srinagar, PIN-190005 J + K, India
Bianchi, Angelo, Ist Sper Cerealicoltura, Via Cassia, 176, 00191 Rome, Italy, 06-3285705
Birchler, J., Genetics Department, 345 Mulford Hall, Univ of California, Berkeley, CA, 94720, 415-642-9025
Bird, Robert McK., Dept of Agronomy, 202 Curtis Hall, Columbia, MO, 65211, 314-882-2033
Bjarnason, Magni, IITA, P.M.B. 5320, Oyo Road, Ibadan, Nigeria
Blackwood, Margaret, School of Botany, Univ of Melbourne, Parkville, Victoria 3052, Australia, 03-341-7157
Blake, Tom, Department of Plant & Soil Sci, Montana State University, Bozeman, MT, 59717-0002, 406-994-4601
Blanco, J. L., Inst Biol Barcelona (Genet), C/Jorge Girona Salgado S/NO, Barcelona 34, Spain, 217-35-73
Blanco, M., Inst Biol Barcelona (Genet), C/Jorge Girona Salgado S/NO, Barcelona 34, Spain
Blanco, M. H., McCurdy Seed Company, Fremont, IA, 52561, 515-933-4291
Bogorad, L., Biol Labs, Harvard Univ, Cambridge, MA, 02138, 617-495-4292
Bojanowska, Constance, Inst Plant Breeding, Biochemical Lab Box 1019, 00-950 Warsaw, Poland
Bokde, S., UNDP-FAO, C. P. 179, Bissau, Guine Bissau, West Africa
Bolen, P. L., NRRL-USDA, 1815 N. Univ St, Peoria, IL, 61614, 309-685-4011 EXT 272
Boothe, Joe G., Dept Plant Sci, Univ Western Ont, London Ont, Canada, 519-679-2908
Borner, T., Sekt Biol, Humboldt Univ, Invalidenstr 43 DDR-1040, Berlin, E Germany
Bosch, L., Casablancas 108 2-2A, Sabadell, Barcelona, Spain, 93-7255689
Boston, Rebecca S., Dept Botany & Plant Pathol, Lilly Hall, Purdue University, W Lafayette, IN, 47907, 317-494-5878
Boyer, C., Dept Hort Penn State Univ, 103 Tyson Building, University Park, PA, 16802, 814-863-3371
Brady, Kevin P., Department of Biology, Indiana University, Bloomington, IN, 47405, 812-335-4234
Brakke, M., Dept of Plant Path, Univ of Nebraska-Lincoln, Lincoln, NE, 68583, 402-472-3166
Brar, G. S., Cetus Madison Corp, 2208 Parview Road, Middleton, WI, 53562, 608-836-7300
Brawn, R. I., CIBA-GEIGY AG, Rosental, AG 5.1, CH-4002, Basel, Switzerland
Brettell, R., CSIRO Div Plant Industry, P.O. Box 1600, Canberra City ACT 2601, Australia, 062-46-5581
Brewbaker, James, Horticulture, Univ of Hawaii, 3190 Maile Way, Honolulu, HI, 96822, 808-948-7985
Briggs, Robert W., Funk Bros Seed Co, 1300 W Washington Street, Bloomington, IL, 61701, 309-829-9461
Brown, D., Dept of Biology, Bishops University, Lennoxville Quebec, Canada, 819-569-9551 EXT 289
Brown, R. P., Ohio Foundation Seeds, Croton, OH, 43013, 614-893-2501
Brown, William L., Pioneer Hibred Internatl, 6800 Pioneer Parkway, Johnston, IA, 50131, 515-270-3155
Browne, C., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-2674
Burmood, T., Research, Jacques Seed Company, Prescott, WI, 54022, 715-262-3223
Burnham, C. R., Agronomy & Plant Genetics, University of Minnesota, St. Paul, MN, 55108, 612-644-7797
Burr, Benjamin, Biology Dept, Brookhaven National Lab, Upton, NY, 11973, 516-282-3396
Bussard, James B., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-3410
Cabulea, I., Plant Breeding Laboratory, Str Agriculturii Nr 27 Jud, Cluj, Turda, Romania
Callis, Judith, Dept Biol, Stanford Univ, Stanford, CA, 94305, 415-497-2609
Camara-Hernandez, J., Altolaguirre 1295, Buenos Aires 1427, Argentina, 51-6464
Cameron, James, Dept of Botany & Plant Sci, University of California, Riverside, CA, 92521, 714-686-0801
Cantrell, Ronald P., CIMMYT, Maize Program, Apartado Postal 6-641, 06600 Mexico, D.F., Mexico
Carlson, John E., Allelix, 6850 Goreway Drive, Mississauga L4V 1P1, Ont., Canada, 416-677-0831
Carlson, Lawrence A., 7 N. Winthrop Street, St. Paul, MN, 55119, 612-735-3423
Carlson, Wayne, Botany Department, University of Iowa, Iowa City, IA, 52240, 319-353-5245
Cassals, Gladys, Department of Biology, Washington University, St. Louis, MO, 63130, 314-889-6813
Catcheside, D. G., Waite Agr Res Inst, Glen Osmond 5064 S, Australia, 08-797901 EXT 389
Chandlee, Joel M., USDA-PGGI, Seed Research Laboratory, Building 006, Beltsville, MD, 20708
Chandler, Vicki, Dept Biol Sci, Stanford University, Stanford, CA, 94305, 415-497-2609
Chandravadana, P., Sub Exp Sta, Univ Mass, 240 Beaver Street, Waltham, MA, 02154, 617-891-0650 EXT 67
Chang, Ming-Tang, Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-7818
Chang, S. C., Known-You Seed Co. Ltd., 55 Min-Yi Rd., Yen-Pu Hsiang, Pingtung Hsieng, 904, Taiwan
Chao, Chuan-Ying, Tunghai University, Box 917, Taichung, Taiwan
Chao, Eddie, Center For Laboratories & Rsch, NYS Health Dept, Empire St Plaza, Albany, NY, 12201, 518-473-7333
Chase, Christine D., Plant Pathol Dept, Univ of Florida, Gainesville, FL, 32611, 904-392-3631

Chase, Sherret S., Box 193, Shokan, NY, 12481, 609-829-0110
Chaudhry, A. R., Punjab Maize + Millets Res, Yousafwala Sahiwal, Pakistan
Chen, Che-Hong, 345 Mulford, Univ of Calif, Berkeley, CA, 94720, 415-642-8058
Chen, Dexing, Dept of Agronomy, Shanghai Agric College, Shanghai, China
Chen, Shu-Mei, 7841 Arcadia, Morton Grove, IL, 60053
Chen, Weicheng, Dept of Agronomy, Henan Agric College, Zhengzhou, China
Cheng, P. C., Anatomy Dept, Univ Illinois, 808 S. Wood St., Chicago, IL, 60612, 312-996-8062
Choe, Bong-Ho, Agronomy Dept, College of Agriculture, Chungnam National University, Dae-Jon 300-31, Korea
Chourey, Prem, USDA ARS, Plant Pathology Department, University of Florida, Gainesville, FL, 32611, 904-392-7237
Christian, J. A., Dept Bot & Bact, Box 3158, Tech Station, Ruston, LA, 71272, 318-257-4772
Chung, J. H., (C) Yaounde, Agency for Internatl Develop, Washington, DC, 20523
Churchill, Gerald A., Asgrow Seed Company, P.O. Box 1235, Twin Falls, ID, 83301
Clark, E. M., Dept Botany & Microbiol, Auburn University, Auburn, AL, 36830, 205-826-4830
Clark, Jan, Dept of Biology, Box 8238, University Station, University of North Dakota, Grand Forks, ND, 58202, 701-777-4479
Clark, Raymond L., USDA Plant Introduction Sta, Iowa State University, Ames, IA, 50010, 515-294-3255
Coe, E. H., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-2768
Cohen, Joel, Dekalb-Pfizer Genetics Inc, RR #2, Box 275L, Palmyra, PA, 17078, 717-964-3642
Colbert, Terry, Funk Seeds International, P.O. Box 921, Union City, TN, 38261, 901-885-7538
Colless, J. M., Agric Research Sta, Grafton, New S. Wales, Australia, 066-447210
Conde, Mary F., 9602-18-2, Experimental Agr Sci, The Upjohn Company, Kalamazoo, MI, 49001, 616-385-6781
Coors, James G., Department of Agronomy, University of Wisconsin, 1575 Linden Drive, Madison, WI, 53706, 608-262-1390
Cormack, Jean, 27 Agronomy, Iowa State University, Ames, IA, 50011, 515-294-5076
Cornu, A., Sta d'Amelior des Plantes, B V 1540, 21034 Dijon, France, 80-237221
Costa-Rodrigues, L., Estacao Agron. Nacional, 2780 Oeiras, Portugal
Crane, Paul L., Dept of Agronomy, Purdue University, West Lafayette, IN, 47907, 317-494-8088
Crawford, C. G., Northern Regional Res Lab, USDA-ARS, 1815 N. University Street, Peoria, IL, 61604, FTS 360-4011
Creech, Roy G., Agronomy, PO Box 5248, Mississippi State University, Mississippi State, MS, 39762, 601-325-2459 EXT 2311
Cress, Dean, USDA-ARS, Room 116, Bldg 011A, Beltsville Agr Res Ctr, Beltsville, MD, 20705, 301-344-4109
Crosbie, T. M., Garst Seed Co., 2515 Elwood Dr., Ames, IA, 50010, 515-296-2550
Cross, Harold, Dept of Agronomy, North Dakota State Univ, Fargo, ND, 58102, 701-237-8138
Cuany, Robin L., Department of Agronomy, Colorado State University, Fort Collins, CO, 80523, 303-491-6832
Cuellar, Richard, Dept Bot & Plant Pathol, Lilly Hall of Life Sciences, Purdue University, W Lafayette, IN, 47907, 317-494-5878
Cullis, Christopher A., John Innes Institute, Colney Lane, Norwich NR4 7UH, U K, 011-44-603-52571
Cummings, D. P., Dekalb-Pfizer Genetics, Box 367, Windfall, IN, 46076, 317-945-7121
Curtis, Christine A., Curtis Hall, Univ of Missouri, Columbia, MO, 65211, 314-882-8215
Cutter, Gary L., Cargill, Inc, 324 Troy Street, Covington, OH, 45318, 513-473-5265
Da Silva, W. J., Genet Evol, Univ Campinas, Cidad Univ Barao Geraldo, Campinas Sp, Brazil
Dahmen, Wayne J., Merck, Sharp & Dohme, Bldg 33-Ag Chem, Rahway, NJ, 07065, 201-574-4176
Dallmier, Donald J., Sturdy Grow Hybrids Inc, Box 94, Arcola, IL, 61910, 217-268-3838
Dankov, Toma, Ivan Assen Str.-93, Sofia 1504, Bulgaria, 43-82-73
Darrah, L., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-7707
Davidson, D., McMaster University, Biology Department, Hamilton, Ontario L8S 4K1, Canada, 416-525-9140 EXT 4588
Day, Peter R., Plant Breeding Institute, Maris Lane, Trumpington, Cambridge CB2 2LQ, England, 0223-840411
De Aguiar-Perecin, Margarida L R, Esc Sup Agr Luiz de Queiroz, Inst de Genetica, C.P. 83, 13400 Piracicaba SP, Brazil
De Araujo, S. M. C., Fac Med Vet Agron, Univ Est Paul Julio Mesq F, 14870 Jaboticabal SP, Brazil, 0163-221798
De Jong, A. W., Zelder B. V., Postbus 26, 6590 AA GENNEP, Nederland
De Leon, Carlos, Apartado Postal 6-641, Mexico 6 D F, Mexico
De Leon, J. L., Vilela 103, 2600 Venado Tuerto, Santa Fe, Argentina
Dellaporta, Steve, Cold Spring Harbor Lab, Cold Spring Harbor, NY, 11724, 516-367-8469
Demopulos-Rodriguez, J., 218 Genetics, Univ of Wisconsin, Madison, WI, 53706, 608-262-3286
Dempsey, Ellen, Biology Dept, Indiana University, Bloomington, IN, 47401, 812-335-4061
Dhawan, N. L., Ford Foundation, 1, Sharia, Osiris, PO Box 2344, Garden City, Cairo, Egypt
Dhillon, B. S., Dept of Plant Breeding, Punjab Agric University, Ludhiana 141004, India
Di Fonzo, Natale, Ist Sper Cereal, Via Stezzano 24, P.O. 164, 24100 Bergamo, Italy, 035-248823
Dicke, F. F., 1430 Harding Avenue, Ames, IA, 50010, 515-232-3140
Dickinson, D. B., Horticulture Department, University of Illinois, 1103 West Dorner Drive, Urbana, IL, 61801, 217-367-2756

Diedrick, T., Agron Plant Genetics, Univ of Minnesota, St. Paul, MN, 55108, 612-373-1346
Dierks-Ventling, C., Friedrich Miescher-Inst, R W60 5.26, PO Box 273, 4002 Basel, Switzerland
Dodd, J. L., P-A-G Seeds, Box 470, Aurora, IL, 60507, 312-892-4331
Doebley, John F., Biology Department, Texas A&M University, College Station, TX, 77843, 919-737-2292
Dollinger, E. J., Ohio Agric Res & Devel Center, Wooster, OH, 44691, 216-263-3893
Dooner, Hugo K., Advanced Genetic Sciences Inc, 6701 San Pablo Avenue, Oakland, CA, 94608, 415-547-2395
Doring, H.-P., Inst Genetik, Univ Koln, D-5000 Koln 41, Weyertal 121, W Germany, 0221-4702465
Dowling, Edward J., C/O Jacques Seed Co, P.O. Box 370, Lincoln, IL, 62656, 217-735-2770
Doyle, G. G., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-8215
Drolsom, Paul, Department of Agronomy, University of Wisconsin, Madison, WI, 53706, 608-262-0876
Duclos, Zachary M., Rt 3, Box 132, Leesburg, GA, 31763, 912-759-2161
Duesing, John H., MSU-DOE Plant Res Lab, Mich State Univ, East Lansing, MI, 48824, 517-353-9216
Duke, Stephen O., S W S L, P.O. Box 225, Stoneville, MS, 38776
Dumanovic, Janko, Maize Res Inst, P.O. Box 89, 11081 Zemun, Yugoslavia
Dunlop, Joan, Plant Sciences Dept, Univ of Western Ontario, London, Ontario N6A 5E7, Canada
Dunn, G. M., Plant Sci Nesmith Hall, Univ of New Hampshire, Durham, NH, 03824, 603-868-2104
Dunn, Mary Eubanks, Department of Anthropology, University of Cincinnati, Cincinnati, OH, 45221, 513-321-1244
Duvick, Donald N., Pioneer Hi-Bred Internat, Johnston, IA, 50131, 515-270-3310
Earle, Elizabeth D., Dept Plant Breed & Biom, Cornell University, 516 Bradfield, Ithaca, NY, 14853, 607-256-3102
Eastin, John A., Box 389, Grant, NE, 69140
Echt, Craig, Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-2033
Eichholtz, David A., Monsanto Co, U4G, 800 N. Lindbergh Blvd, St. Louis, MO, 63167, 314-694-2146
El-Ard, Raghad Shiekh, Malki-Abdul Monem, Regad St Build 77 #7, Damascus, Syria
Empig, L. T., San Miguel Corp Box 271, 6766 Ayala Avenue, Makati, Rizal, Manila, Philippines
Endo, Toru, National Inst of Genetics, Misima 411, Japan, 0559-75-0771 EXT 319
England, Dan, Curtis Hall, Univ of Missouri, Columbia, MO, 65211, 314-882-7818
Esen, Asim, Dept Biology, Va Polytech Inst & State Univ, Blacksburg, VA, 24061, 703-961-5894
Everett, Herbert L., Cornell University, 252 Emerson Hall, Ithaca, NY, 14853, 607-256-3103
Evola, S. V., CIBA-GEIGY Corporation, P.O. Box 12257, Research Triangle Park, NC, 27709-2257, 919-549-8164
Faberge, A. C., Department of Zoology, University of Texas, Austin, TX, 78711, 512-471-7442
Fedoroff, Nina, Carnegie Inst Wash, 115 West Univ Parkway, Baltimore, MD, 21210, 301-467-1414
Feix, G., Institut Fur Biologie III, Universitat Freiburg, Freiburg, W Germany, 0761-2032724
Ferguson, V., Custom Farm Seed, 2761 N Main, Decatur, IL, 62526, 217-875-2826
Ferl, R. J., Dept Bot, Univ Florida, Gainesville, FL, 32611, 904-392-2370
Ficsor, G., Dept of Biology, Western Mich Univ, Kalamazoo, MI, 49008, 616-383-1820
Figoni, Richard, IPRI, 830 Bransten Road, San Carlos, CA, 94070, 415-595-5335
Filion, W. G., Erindale College, University of Toronto, 3359 Mississauga Road, Mississauga, Ontario, Canada, 416-828-5326
Fincher, Robert R., Pioneer Hi-Bred Internatl, Department of Corn Breeding, P.O. Box 270, Union City, TN, 38261, 901-885-7710
Findley, William R., Agronomy Department, Ohio Agri Res & Dev Center, Wooster, OH, 44691, 216-263-3894
Fitzgerald, P. J., USDA ARS, 2000 W Pioneer Parkway, Peoria, IL, 61614, 309-671-7176
Fleming, A. A., Miller Bldg, University of Georgia, Athens, GA, 30602, 404-542-2461
Fletcher, G., Agronomy Dept, S-122 Turner, University of Illinois, 1102 S. Goodwin, Urbana, IL, 61801, 217-333-4254
Floren, Catalina, CIMMYT, Londres 40, Apdo Postal 6-641, 06600 Mexico, D.F., Mexico
Foard, Donald, UT-CARL, Oak Ridge, TN, 37830
Foley, Terry, Holden's Foundation Seeds Inc, P.O. Box 839, Williamsburg, IA, 52361, 319-668-1100
Foster, David J., Garst Seed Co/Research Dept, 2515 Elwood, Box 2, Ames, IA, 50010, 515-296-2550
Fourie, A. P., Dep Landb Vis, Highveld, Privaatsak X1251, Potchefstroom 2520, S Africa, 01481-25504
Fowler, Robert G., Dept Biological Sciences, San Jose State University, San Jose, CA, 95192, 408-277-2355
Fraley, Robb, Monsanto Co, U4G, 800 N. Lindbergh Blvd, St. Louis, MO, 63167
Francis, T. R., Northrup King Co., R. R. 1, Arva, N0M 1C0, Canada, 519-452-3019
Freeling, Michael, Dept of Genetics, Univ of California, Berkeley, CA, 94720, 415-642-7085
Funk, Don E., Edward Funk and Sons, P.O. Box 67, Kentland, IN, 47951
Furtek, Douglas, Lab of Genetics, Univ of Wisconsin, Madison, WI, 53706, 608-262-3287
Gabay-Laughnan, S., Dpt Genetics & Dvl/515 Morrill, Univ of Illinois, 505 S. Goodwin Avenue, Urbana, IL, 61801, 217-333-2919
Galinat, Walton C., Sub Exp Sta, Univ Mass, 240 Beaver Street, Waltham, MA, 02154, 617-891-0650
Gallais, A., G.I.S. du Moulon, Ferme du Moulon, 91190 Gif sur Yvette, France

Gamble, Edwin E., Dept of Crop Science, University of Guelph, Guelph, Ontario N1G 2W1, Canada, 519-824-4120 X3574
Gamborg, Oluf, United Energy Corporation, 420 Lincoln Centre Drive, Foster City, CA, 94404, 415-952-1000 EXT 6313
Garber, E. D., Barnes Lab, 5630 S Ingleside Avenue, Chicago, IL, 60637, 312-753-2671
Garber, Robert C., Institute of Genetics, University of Cologne, 5000 Cologne 41, W Germany, 0221-470-2465
Garcia-Olmedo, F., Dept Biochemistry, E T S Ingenieros Agronomos, Madrid-3, Spain, 1-24444807
Gardner, C. A. C., Pioneer Hybrid Internatl, Highway 65 South, PO Box 218, Carrollton, MO, 64633, 816-542-3780
Gardner, C. O., Department of Agronomy, University of Nebraska, Lincoln, NE, 68583-0915
Garwood, D. L., Garwood Seed Company, RR 1, Box 20, Stonington, IL, 62567, 217-325-3715
Garzonio, Dennis, PAG Seeds, PO Box 467, Monticello, IL, 61856, 217-762-2711
Gavazzi, Guiseppe, Universita de Milano, Dip. Biologia/Sec. Genetica, Via Celoria 26, Milano, Italy, 02-230823
Geadelmann, J. L., Dept Agron & Pl Gen, Univ of Minnesota, St Paul, MN, 55108, 612-373-0872
Gebauer, Juan E., Corn Breeding, Casilla 190, Buin, Chile, 552 Buin
Geerthsen, J. M. P., Dept of Genetics, University of Pretoria, Pretoria 0181, S Africa, 012-436381
Geiger, Hartwig H., Univ Hohenheim, Inst Pflanzenzucht, Postfach 700562(350), D-7000 Stuttgart 70, W Germany
Geng, Qing-Han, Dept of Agronomy, Inner Mongolia Agric College, Inner Mongolia Aut Reg, China
Gengenbach, B.G., Agron & Plant Genetics, Univ of Minnesota, St Paul, MN, 55108, 612-373-1371
Georgiev, Trifon, Technical University, "A. Kanchev", Rousse 7004, Bulgaria, 082-23469
Gerats, Tom, Genetisch Institute, Universiteit Van Amsterdam, Kruislaan 318, 1098 SM Amsterdam, Nederland
Gerrish, Everett E., Box 774, Grinnell, IA, 50112, 515-236-4911
Ghidoni, Achille, Istituto di Genetica, Universita di Milano, Via Celoria 26, Milan 20133, ITALY, 02-230823
Ghobrial, Helmy K., 4100 Valentine Crest Rd, St. Paul, MN, 55112
Ghosh, P., Dept of Botany, Kalyani Univ, Kalyani, Nadia, W Bengal, India, 220/ext 9
Giesbrecht, John, Res Sta Canada Agr, P.O. Box 3001, Morden, Manitoba, Canada, 822-4471
Gillies, Christopher, Botany Bldg A12, Univ of Sydney, Sydney N S W 2006, Australia, 02-692-2688
Giusto, John P., Department of Genetics, 345 Mulford Hall, University of California, Berkeley, CA, 94720, 415-642-8058
Glover, David V., Dept of Agronomy, Purdue University, Lafayette, IN, 47907, 317-494-8067
Goertzen, Kenneth L., Goertzen Seed Res Inc, R R 2 Box 48, Scott City, KS, 67871, 316-872-2807
Goldman, Stephen, Dept of Biology, University of Toledo, Toledo, OH, 43606, 419-537-4197
Golubovskaya, I. N., Inst Genet & Cytol, Siberian Branch Akad Sci, Novosibirsk, USSR
Gonella, Jaime, CIBA-GEIGY, 26020 Casalmorano (CR), Italy, 0374-74144
Good, Ronald L., Funk Seeds International, PO Box 417, Bluffton, IN, 47614, 219-824-5056
Goodman, Major M., Department of Crop Sciences, North Carolina State Univ, P.O. Box 7620, Raleigh, NC, 27695, 919-737-2704
Gordon, P. N., Department of Biology, 918 Kline Biology Tower, Yale Univ, P.O. Box 6666, New Haven, CT, 06511-8112
Gottlieb, Leslie D., Dept of Genetics, Univ of Calif-Davis, Davis, CA, 95616, 916-752-0268
Gracen, V. E., Dept Plant Breeding, Cornell Univ, Ithaca, NY, 14853, 607-256-3103
Graham, K. M., Genetics Department, Faculty of Life Sciences, Natl Univ of Malaysia, Bangi, Malaysia
Gray, Dennis J., University of Florida, Agricultural Research Center, P.O. Box 388, Leesburg, FL, 32749-0388, 904-787-3423
Greder, Rod, 2106 Orchard St #302, Urbana, IL, 61801, 217-333-7585
Green, C. E., Molecular Genetics Inc., 10320 Bren Road East, Minnetonka, MN, 55343, 612-935-7335
Green, M. M., Dept of Genetics, University of California, Davis, CA, 95616, 916-752-1109 EXT 6295
Greenblatt, I. M., Genetics & Cell Biology, U-131, Univ of Connecticut, Storrs, CT, 06268, 203-486-2915
Greyson, R. I., Dept of Pl Sci-Faculty of Sci, Univ of Western Ontario, London, Ontario N6A 5B7, Canada, 519-679-3107
Grier, Stephen L., Agrigenetics Corp, Vegetable Products Group, 1120-220th St West, Farmington, MN, 55024, 612-463-4646
Grobman, Alexander, Grobman Genotecnica SA, Apartado 5161, Lima 1, Peru, 234464
Gross, D. F., Dekalb-Pfizer Genetics, PO Box 9096, Madison, WI, 53715
Grumbles, Robert M., Department of Genetics, University of Alberta, Edmonton T6G 2E9, Canada, 432-5556
Hagan, William, Del Monte Corp, 850 Thornton St, PO Box 36, San Leandro, CA, 94577, 415-351-2661
Hagemann, R., Dept Genetics, Martin Luther University, Domplatz 1, 4020 Halle/S., E Germany
Hague, Donald R., Department of Biology, University of Oregon, Eugene, OR, 97403, 503-686-4545
Hake, S., Genetics Dept, Univ of California, Berkeley, CA, 94720, 415-642-7948
Hallauer, Arnel R., Agronomy Building, Iowa State University, Ames, IA, 50010, 515-294-3052
Hamilton, R. H., Buckhout Laboratory, Pennsylvania State Univ, University Park, PA, 16802, 814-865-8800
Han, Changdeok, Curtis Hall, University of Missouri, Columbia, MO, 65211
Hannah, L. C., Veg Crops Dept, Univ of Florida, IFAS, Gainesville, FL, 32611, 904-392-1794
Hansel, W. C., Hansel Cons & Mgmt, Box 283, Carrollton, MO, 64633, 816-542-1616
Hansen, Dale J., Agrigenetics Res Corp, 3375 Mitchell Lane, Boulder, CO, 80301, 303-443-5900 EXT 230

Hanson, Maureen R., Dept Biol, Gilmer Hall, Univ Virginia, Charlottesville, VA, 22901, 804-924-0706
Hanson, W. D., Dept of Genetics, North Carolina State Univ, Raleigh, NC, 27607, 919-737-2289
Harlan, Jack, Agronomy Department, University of Illinois, 1102 S. Goodwin Avenue, Urbana, IL, 61801, 217-333-4373
Harris, John W., Dept of Biology, Tennessee Tech Univ, Cookeville, TN, 38505, 615-528-3143
Harris, Robert M., Committee on Genetics, University of Arizona, Tucson, AZ, 85721, 602-621-3584
Hartman, Carl J., Hunt Wesson Foods, P.O. Box 468, Valparaiso, IN, 46383, 219-465-0540
Harvey, P. H., Dept Crop Sci, NC State Univ, Raleigh, NC, 27650, 919-787-6054
Haskins, F. A., Dept of Agronomy, Univ of Nebraska, Lincoln, NE, 68583, 402-472-1567
Hauptli, Holly, Calgene Inc, 1910 Fifth Street, Davis, CA, 95616, 916-753-6313
Hawk, James A., Dept Plant Science, University of Delaware, Newark, DE, 19711, 302-738-2531
Hayden, D. B., Dept of Botany, Univ of Western Ontario, London, Ontario, Canada, 519-679-6804
Hedman, K., 805 Stratford Drive, Apt 8, State College, PA, 16801, 814-234-3277
Heidrich-Sobrinho, E., Dept Genetica Ib Ufgrs, Caixa Postal, 1953, Porto Alegre Rs, Brazil
Helgason, S. B., Dept of Plant Science, Univ of Manitoba, Winnipeg, Man. R3T 1N1, Canada, 474-8221
Helm, James L., Asgrow Seed Company, Building 190, Kalamazoo, MI, 49001
Helsel, D., Agronomy Curtis Hall, Univ of Missouri, Columbia, MO, 65211, 314-882-6606
Henderson, Clarion B., Illinois Foundation Seeds, PO Box 722, Champaign, IL, 61820, 217-485-6260
Henke, Randolph R., Department of Botany, University of Tennessee, Knoxville, TN, 37996, 615-974-6227
Herskowitz, I., Dept Biochem & Biophys, Univ of California, San Francisco, CA, 94143, 415-666-4977
Hibberd, K. A., Molecular Genetics Inc, 10320 Bren Road East, Minnetonka, MN, 55343, 612-935-7335
Ho, David, Department of Biology, Washington University, St. Louis, MO, 63130, 314-889-4632
Hoagland, R. E., USDA ARS SWSL, PO Box 225, Stoneville, MS, 38776, 601-686-2311
Hochmuth, George J., Dept of Plant Soil Sci, University of Massachusetts, 205 Bowditch Hall, Amherst, MA, 01003, 413-545-2250
Hodges, Thomas K., Dept Botany Plant Pathol, Purdue University, Lafayette, IN, 47907, 317-494-4657
Hoegemeyer, Thomas C., Hoegemeyer Hybrids Inc, Rt 2, Hooper, NE, 68031, 402-654-3399
Hoisington, David, Curtis Hall, Univ of Missouri, Columbia, MO, 65211, 314-882-2033
Holden, A., Corn States Hybrid Serv, PO Box 2706, Des Moines, IA, 50315, 515-285-3091
Hole, David, Soil & Crop Sci. Dept., Plant Sci. Bldg., Texas A&M University, College Station, TX, 77843, 409-845-1126
Hooker, A. L., Dekalb-Pfizer Genetics, Corn Research Center, 3100 Sycamore Road, Dekalb, IL, 60115, 815-756-3671
Hopkins, W. G., Dept of Plant Sciences, Univ of Western Ontario, London, Ontario, Canada
Hourcade, Dennis, Monsanto, 700 Chesterfield Parkway, GG4H, Chesterfield, MO, 63017, 314-537-6783
Hsieh, S. C., Taichung District, Agricultural Improvement Sta, Tienyang Village, Tatsuen Hsiang, Changhua, Taiwan, 048-358-130
Hsu, Lilian M., Carr Lab, Mount Holyoke College, South Hadley, MA, 01075, 413-538-2609
Hu, Wilma Wei-Lin, Dept of Crop Science, North Carolina State Univ, Raleigh, NC, 27607
Huang, Yi Xiang, Crop Breed & Cult Res Inst, Sichuan Academy Agr Sci, Chengdu, Sichuan Province, China
Hunt, Marjorie, Dept Biol, Univ of Missouri, 108 Tucker Hall, Columbia, MO, 65211, 314-882-3240
Hunter, Brenda, Dept Genet Cell B, Univ Minnesota, St. Paul, MN, 55108
Hunter, R. B., Dept of Crop Science, University of Guelph, Guelph, Ontario N1G 2W1, Canada, 519-836-1596
Iltis, Hugh H., Dept of Botany, Univ of Wisconsin, Madison, WI, 53706, 608-256-7247
Inglett, George, USDA-NRRC, 1815 N University Ave, Peoria, IL, 61604, 309-685-4011
Ingram, David S., Botany Department, University of Cambridge, Downing Street, Cambridge CB2 3EA, U K, 0223-61414
Inoue, Yasuaki, National Grassland Res Inst, 768 Nishinasuno, Tochigi 329-27, Japan
Irish, Erin, Box 46 Jordan Hall, Indiana Univ, Bloomington, IN, 47401, 812-335-6060
Jackson, R. C., Biology Department, Texas Tech University, Lubbock, TX, 79409, 806-742-2717
Jacobs, M., Plant Genetics Univ Brussels, 65 Paardenstraat, B-1640st Genesius-Rode, Belgium, 2-358-65-22
Jakob, Karl M., Department of Plant Genetics, Weizmann Inst Sci, Rehovot, Israel, 054-83301
Jalani, B. S., Nat Univ of Malaysia, Bangi-Selangor, Malaysia
Jan-Orn, Jinda, Division of Field Crops, Department of Agriculture, Bangkok 10903, Thailand
Jauhar, Prem P., U.S. Agric. Labs, 12155 Magnolia Ave, Bldg 4C, Riverside, CA, 92503, 714-688-6160
Jaynes, Jesse, Department of Biology, 322 Choppin Hall, Louisiana State University, Baton Rouge, LA, 70803
Jellum, Milton D., Georgia Agr Exp Station, Experiment, GA, 30212, 404-228-7278
Jenkins, M. T., 1200 Mira Mar Avenue, Apt 826, Medford, OR, 97504, 503-776-5255
Jennings, Paul H., Plant & Soil Sciences, Univ of Massachusetts, Amherst, MA, 01003
Jewell, David C., CIMMYT, Londres 40, Apdo Postal 6-641, Mexico D.F. 06600, 585-4355 EXT 1110
Jimenez, J., Biology Dept, Purdue Univ, Fort Wayne, IN, 46805, 219-482-5253
Johnson, Alice, 412 Bradfield Hall, Cornell Univ, Ithaca, NY, 14853, 607-256-3104

Johnson, E. C., IPRI, Internatl Plant Res, 830 Bransten Road, San Carlos, CA, 94070, 415-595-5335
Johnson, Elizabeth, Box 371, Kaunakakai, HI, 96748, 808-553-5468
Johnson, Ken, Sigco Research, Box 289, Breckenridge, MN, 56520, 218-643-2155
Johnson, M. W., Dept of Agronomy, Pennsylvania State Univ, University Park, PA, 16802, 814-865-0324
Johri, M. M., Molecular Biol Unit, Tata Inst Fundamental Res, Bombay 400 005, India, 219111/ext 338
Jones, J. K., Dept of Agricultural Bot, University of Reading, Whiteknights, Reading RG6 2AS, England, 0734-875123 EXT 7905
Jones, John Edward, Department Biol Sci, Rm B-217, Box 84 Lilly Hall, Purdue University, W Lafayette, IN, 47907, 317-494-4931
Jones, Q., Natl Program Staff, USDA-ARS, Beltsville, MD, 20705, 301-344-3311
Josephson, L. M., 350 Plant Sci Bldg, Univ of Tennessee, Knoxville, TN, 37916, 615-974-7391
Jotshi, P. N., Regional Research Lab (Branch), Council Sci & Indus Res, Sanat Nagar 190005, Srinagar, India
Julstrom, P., Dekalb-Pfizer Genetics, P.O. Box 719, 2391 Likens Road, Marion, OH, 43302
Juvik, J. A., Dept of Horticulture, University of Illinois, Urbana, IL, 61801, 217-333-1966
Kaan, F., INRA ENSA, Place Viala 34060, Montpellier Cedex, France, 67-63-43-20
Kahler, A. L., N Grain Insects Res Lab, RR 3, Brookings, SD, 57006, 605-693-5207
Kaltsikes, P. J., Dept Plant Breed & Biometry, Athens School of Agr Sci, Iera Odos 75, Athens T.T.301, Greece, 347-1504
Kaneko, Koji, National Agr Res Center, Yatabe-Cho, Tsukuba-Gun, Ibaraki-Ken 305, Japan
Kang, M. S., U Fla Agr Res Educ Ctr, P.O. Drawer A, Belle Glade, FL, 33430, 305-924-5227
Kannenberg, L. M., Dept of Crop Science, University of Guelph, Guelph, Ontario N1G 2W1, Canada, 519-824-4120 EXT 2506
Karpoff, A. J., Dept of Biology, Univ of Louisville, Louisville, KY, 40208, 502-588-6771
Kasha, K. J., Dept of Crop Science, Univ of Guelph, Guelph, Ontario N1G 2W1, Canada, 824-4120 EXT 2507
Kato Y., T. A., Centro de Genetica, Colegio de Postgraduados, Chapingo 56230, Mexico, (5)5-85-07-20
Keil, R. L., Yale U/Biology Dpt/PO Box 6666, Kline Biol Tower Rm 812, New Haven, CT, 06511, 203-436-2266
Keim, Kent R., Plant & Soil Sci Dept, Texas Tech University, Lubbock, TX, 79409
Keller, Patrick, Biochemistry Dept, Univ of Rhode Island, Kingston, RI, 02881, 401-295-0298
Kemble, R. J., Allelix Inc, 6850 Goreway Drive, Mississauga, Ont. L0N 1K0, Canada, 416-677-0831
Kermicle, Jerry, Dept of Genetics, University of Wisconsin, Madison, WI, 53706, 608-262-1253
Kerr, E. A., Stokes Seeds Limited, 39 James Street, P.O. Box 10, St. Catharines, Ontario L2R 6R6, Canada, 416-688-4300
Khavkin, Emil E., All-Russian Res Inst Chem Agr, Nemchinovka, Moscow, USSR
Khehra, A. S., Punjab Agr University, Department of Plant Breeding, Ludhiana, India
Kikudome, Gary Y., Tucker Hall, University of Missouri, Columbia, MO, 65211, 314-882-2634
Kim, Byung-Dong, Department of Plant Science, University of Rhode Island, Kingston, RI, 02881, 401-792-2529
Kim, Soon Kwon, Internatl Inst Tropical Agr, PMB 5320, Ibadan, Nigeria
Kind, K., Curtis Hall, Univ of Missouri, Columbia, MO, 65211, 314-882-2674
Kindiger, Bryan, Curtis Hall, Univ of Missouri, Columbia, MO, 65211, 314-882-8214
King, Patrick J., Friedrich Miescher-Inst, Postfach 2543, CH-4002 Basel, Switzerland, 61-375583
Kinugawa, Kenjiro, 4-23-3, Takakuradai, Univ of Osaka Prefecture, Sakai, Osaka 590-01, Japan, 0722-91-5497
Kiss, Charles, Northrup King Semences, 18 Avenue Gollieni, 49130 Les Ponts de C, France, (41) 663831
Kleese, Roger, Molecular Genetics, 10320 Bren Road East, Minnetonka, MN, 55343, 612-935-7335
Klein, Anita S., Department of Biochemistry, Spaulding Life Science Bldg, University of New Hampshire, Durham, NH, 03824
Kohashi, Josue, Dept of Botany, Colegio de Postgrad, ENA, Chapingo, Edo de Mex, Mexico
Konzak, Calvin F., Dept of Agronomy & Soils, Washington State Univ, Pullman, WA, 99164-6420, 509-335-3635
Kovacs, Istvan, Agriculture Research Instit, Hungarian Acad Sci, 2462 Martonvasar, Hungary, 26-45774
Kowles, R. V., Biology Department, St Marys College, Winona, MN, 55987, 507-452-4430 EXT 1554
Krapovickas, Antonio, Dept Bot y Ecol, Casilla Correo 209, 3400 Corrientes, Argentina
Kratochvil, D. E., Sokota Hybrid Producers, Brookings, SD, 57006, 605-692-6258
Krill-Shadley, Gwen, Dept Biology, Ill State Univ, Normal, IL, 61761, 309-438-2685
Kriz, A. L., Dept of Botany & Plant Pathol, Lilly Hall, Purdue University, W. Lafayette, IN, 47907
Kumar, D., Div Plant Br & Genet JARI, PO Barrackpore D 24 Pargana, West Bengal, India, 24-4006
Kumar, Santosh, Professors' Colony, Aghoriachowk, PO Ramna, Muzaffarpur-842002 BIHAR, India
Laible, Charles A., Research Department, Funk Seeds Internat, Bloomington, IL, 61701, 309-829-9461
Lambert, A., Semences Nickerson, 49160 Longue, Jumelles, France
Lambert, R. J., Agronomy Dpt/S-118 Turner Hall, University of Illinois, 1102 S. Goodwin Avenue, Urbana, IL, 61801, 217-333-4254
Lampoh, E., Crops Res Inst, PO Box 20, KPEVE, Volta Reg, Ghana, W Africa, 2 ext 5
Larkins, B., Dept Bot & Plant Path, Purdue Univ, W Lafayette, IN, 47907, 317-494-4624
Larson, Russell L., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-3410
Laughnan, John R., Genetics & Devl/515 Morrill Hl, U of Illinois/505 S Goodwin Av, Urbana, IL, 61801, 217-333-2919

Laughner, Bill, Dept Biology, Hiram College, Hiram, OH, 44234, 216-569-3211 EXT 260
Law, Colin, Plant Breed Inst, Maris Lane, Trumpington, Cambridge CB2 2LQ, England
Leaver, C. J., Dept Botany, Kings Bldgs, U. of Edinburgh/Mayfield Road, Edinburgh EH9 3JH, Scotland, 031-667-1081 EXT 3304
Lee, Chong, Oil Palm Res Station, PO Box 207, Banting, Selangor, Malaysia
Lee, Michael, Dept Agron Plant Genet, Agronomy Bldg/1509 Gortner Ave, St. Paul, MN, 55108, 612-373-1372
Lee, Wayne, 108 S. Grant, Milford, IL, 60953, 815-889-4426
LeFord, Duane, Pfister Hybrid Corn Co, El Paso, IL, 61738, 309-527-6000
Lemke, Carol A., 405 Bradfield Hall, Cornell Univ, Ithaca, NY, 14853, 607-256-3104
Leonard, Kurt, USDA-ARS, Box 5397 State Coll Sta, Raleigh, NC, 27607, 919-737-2751
Leto, K. J., Central Research Dept, E. I. Du Pont Exp Sta, Bldg 328, Rm 259, Wilmington, DE, 19898, 302-772-1525
Levings, C. S., Department of Genetics, North Carolina State Univ, Raleigh, NC, 27607, 919-737-2289
Levites, E. V., Inst Cytol Genetics, Novosibirsk, USSR
Lewis, Herman, Div of Phys, Cell & Mol Biol, National Science Foundation, Washington, DC, 20550, 202-357-7647
Li, Ching Hsiung, Chinese Acad Agric Sci, Beijing, China
Li, H. W., Institute of Botany, Academia Sinica, Taipei, Taiwan
Li, Jigeng, Institute of Genetics, Academia Sinica, Beijing, China
Liang, George H., Dept of Agronomy, Kansas State University, Manhattan, KS, 66502, 913-532-7255
Lin, Bor-yaw, 310 Curtis Hall, Univ of Missouri, Columbia, MO, 65211, 314-882-3856
Lin, Chi C., Lilly Research Lab, Box 708, Greenfield, IN, 46140
Lin, Ji-Zhou, Institute of Crops, Shanxi Acad Agric Sci, Shanxi Province, China
Lindsey, M. F., Dekalb-Pfizer Genetics, Box 408, Dayton, IA, 50530, 515-547-2550
Liu, Edwin H., Savannah River Ecology Lab, P.O. Drawer E, Aiken, SC, 29801, 803-725-2472
Liu, Enxun, Dept of Agronomy, Laiyang Agric College, Laiyang, Shandong, China
Liu, Jilin, Dept of Agronomy, Huazhung Agric College, Wuhan, Hubei Province, China
Loeffel, F. A., NAPB Res. Center, Rural Route #2, Brookston, IN, 47923, 317-563-3111
Lorenzoni, C., Istituto di Genetica Vegetale, Universita Cattolica, 29100 Piacenza, Italy
Lorz, Horst, Max-Planck-Institut Fur, Zuchtungsforschung, D5000 Koln 30, W Germany
Lower, W., 235 Environ Trace Ctr, Univ of Missouri, Columbia, MO, 65211, 314-445-8595
Ma, Te-Hsiu, Dept Biol Sciences, Western Illinois University, Macomb, IL, 61455, 309-298-1457
Maggini, Fabio, Ist di Biologia Agraria, Cattedra di Botanica, Via Riello, 01100 Viterbo, Italy
Magoja, J. L., Inst Fitotec Santa Catalina, Llavallol, Argentina, 243-0233
Maguire, Marjorie, Genetics Foundation, University of Texas, Austin, TX, 78712, 512-471-7451
Majeste, A, Coop Agric Cereales, Ave Gaston Phoebus, 64230 Lescar, France, 59-328446
Maletsky, S. I., Tereshkova St, House 6, Flat 49, Novosibirsk 630090, USSR
Malone, Carl, 205 McKinley Avenue, Milford, IL, 60953, 815-889-4079
Maloney, Alan, Biol Sci Dept, Stanford Univ, Stanford, CA, 94305, 415-497-2609
Mangelsdorf, P. C., Apt. 1200, Carol Woods, Chapel Hill, NC, 27514, 919-942-4396
Manrique, Antonio, Univ Agraria Progr, Coop, Inv Maiz, La Molina, Apt 456 Lima, Peru, 312746
Mans, Rusty J., Dept of Biochem/J245 JHMHC, Univ of Florida, Gainesville, FL, 32610, 904-392-2332
Marchand, J. L., IRAT, Ligne Paradis, 97610 St. Pierre, Reunion
Mariani, Giuseppe, Ist Sper Cerealicoltura, Via Cassia 176, Roma 00191, Italy
Marks, Michael D., Dept Botany & Plant Pathol, Lilly Hall of Life Sciences, Purdue University, W Lafayette, IN, 47907
Martin, I. F., Research Station, PO Box 27, Kairi, Q 4872, Australia
Martin Sanchez, J. A., Esc T Sup Ingenieros Agron, Carretera de Huesca Km 3, Lerida, Spain, 973-233841
Marton, Csaba Lajos, 2462 Martonvasar, P.O.B. 19, Hungary, 26-45774
Mascarenhas, Joseph P., Department of Biol Sci, State University of New York, Albany, NY, 12222, 518-457-3300
Mascia, P., Monsanto, U1F, 800 N. Lindbergh Blvd., St. Louis, MO, 63166, 314-694-5011
Mashnenkov, A. S., Krasnodar Res Inst Agric, Krasnodar, USSR
Mathur, D. S., 126, Indian Agr Res Inst, New Delhi, India
Matthews, David L., Agway Inc, Box 4933, Syracuse, NY, 13221, 315-477-6391
Maunder, A. Bruce, Dekalb-Pfizer Genetics, Route 2, Lubbock, TX, 79415, 806-763-3336
Mazoti, Luis B., C. Croce 145, 1832 Lomas de Zamora, Argentina
McBride, A. C., Phytotech, 624 South 775 E, Lafayette, IN, 47905, 317-296-3497
McClintock, Barbara, Cold Spring Harbor Laboratory, Cold Spring Harbor, NY, 11724, 516-367-8385
McCormick, Sheila, Monsanto Co, U4B, 800 Lindbergh Blvd, St. Louis, MO, 63167, 314-694-4576
McCoy, T., USDA ARS, Univ of Nevada, Reno, NV, 89557, 702-784-5336

McDaniel, R. G., Dept of Plant Sciences, University of Arizona, Tucson, AZ, 85721, 602-261-3357
McMillin, David E., Department of Biology, Georgia State University, Atlanta, GA, 30303
McMullen, Michael, Dept Genet & Cell Biol, University of Minnesota, 1445 Gortner Avenue, St Paul, MN, 55108, 612-373-1734
McNay, James W., Dept Plant Pathol, Univ of Florida, Gainesville, FL, 32601
McWhirter, K. S., Dept of Agric Genet & Biometry, University of Sydney, Sydney NSW 2006, Australia, 02-692-2938
Meinke, D., Dept Botany & Microbiology, Oklahoma State University, Stillwater, OK, 74078, 405-624-5559
Mello-Sampayo, Tristao, Centro di Biologia, Inst Gulbenkian de Ciancia, Oeiras, Portugal
Mertens, Thomas R., Dept of Biology, Ball State University, Muncie, IN, 47306, 317-285-1639
Mettin, Dieter, Akad der Wissenschaften DDR, Zentralinstitut fur Genetik, Corrensstrasse 3, 4235 Gatersleben, E Germany
Metzger, Robert, 255 Crops Science, Oregon State College, Corvallis, OR, 97331, 503-757-4449
Michel, Kenneth, Department of Biology, Slippery Rock University, Slippery Rock, PA, 16057, 412-794-7326
Micu, V. E., Moldav Inst Maize & Sorghum, Kriulianskii Raion Krcv-Nova, 278335 Moldavian SSR, USSR
Miernyk, Jan, Northern Regional Res Lab, USDA-ARS, 1815 N. University Street, Peoria, IL, 61604, FTS 360-4011
Mikula, Bernard C., Defiance College, Defiance, OH, 43512, 419-784-4010 EXT 426
Miles, Donald, Tucker Hall, University of Missouri, Columbia, MO, 65211, 314-882-6675
Miranda, L. T. De, Instituto Agronomico, Caixa Postal, 28, 13.100 Campinas SP, Brazil, 0192-420235
Mock, James J., Northrup King Company, 1500 Jackson St NE, PO Box 959, Minneapolis, MN, 55440
Modena, Stephen, Dept. of Crop Science, North Carolina State Univ., Box 7620, Raleigh, NC, 27695, 919-737-2704
Mogford, D. J., Asgrow Research Sta, P.O. Box 152, Bronkhorstpruit 1020, S Africa, (012) 982400
Mohamed, A. H., Department of Biology, University of Missouri, Kansas City, MO, 64110, 816-276-1385
Mohl, Leo, PO Box 825, Colorado Springs, CO, 80901, 303-633-0683
Moll, Robert H., 2623 Gardner Hall, Genetics, North Carolina State Univ, Raleigh, NC, 27607, 919-737-2289
Molnar, S. J., Agriculture Canada, Gen Eng Ottawa Res Sta, Ottawa, Ontario K1A 0C6, Canada, 613-995-3700 EXT 313
Moore, Carol W., Dept Rad Biol & Biophysics, School of Medicine, University of Rochester, Rochester, NY, 14642, 716-275-5220
Mora, G., GIE Pioneer-France, Epuiseau, 41290 Ducques, France
Morris, Rosalind, Department of Agronomy, University of Nebraska, Lincoln, NE, 68583-0915, 402-472-1534
Mottinger, John, Department of Botany, University of Rhode Island, Kingston, RI, 02881, 401-792-2625
Muchena, S. C., Ministry of Agriculture, Private Bag 7701, Causeway Harare, Zimbabwe, 706081
Mukai, Yasushi, 1-11-10, Tachibana-Higashi, 880 Miyazaki, Japan, 0985-27-1838
Mukherjee, B. K., Cummings Lab, Indian Agr Res Inst, New Delhi 110012, India, 586198
Mulcahy, David, Botany Department, Univ of Massachusetts, Amherst, MA, 01003, 413-545-2238
Mumm, W. J., Box 306, Milford, IL, 60953, 815-889-4550
Murphy, C. F., USDA NPS, Bldg 005, Rm. 239, BARC-W, Beltsville, MD, 20705, 301-344-1560
Murphy, T. C., Northrup King Co, Stanton, IN, 55081, 507-645-5621
Myers, Jr, Oval, Dept of Plant & Soil Science, Southern Illinois Univ, Carbondale, IL, 62901, 618-453-2496
Nagle, B. J., Dekalb-Pfizer Genetics, Box 33, Mason City, IL, 62664, 217-482-3213
Nanda, Dave K., O's Gold Seed Co, PO Box 460, Parkersburg, IA, 50665, 319-347-6633
Narayan, Kishen, Maize Res Station, Amberpet, Hyderabad 500013 A.P., India
Natti, Thomas A., Robson Seed Farms Corp, PO Box 270, Hall, NY, 14463, 716-526-5879
Nault, Lowell R, Dept Entomology, Ohio Agr R&D Ctr, Wooster, OH, 44691
Ndegwa, C., P.O. Box 553, Kitale, Kenya, 20941
Nebiolo, Christine M., Dept of Biology, Allegheny College, Meadville, PA, 16335, 814-724-5360
Neely, J. Winston, Cokers Pedigreed Seed Co, Hartsville, SC, 29550
Nel, P. M., Dept of Genetics, Univ of The Witwatersrand, Johannesburg 2001, S Africa, 011-716-2134
Nelson, O. E., Jr., Department of Genetics, University of Wisconsin, Madison, WI, 53706, 608-262-3344
Nelson, S. W., CIBA-GEIGY Ltd, PO Box 92, Isando, 1600, S Africa, 011-706-7257
Nemeth, J., Gabonater Kutatointezet, Szeged, Also Kikoto sor 9, Hungary, 62-17-025
Neuffer, M. G., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-7735
Newhouse, Keith, GG4I, c/o Monsanto Ag Products, 800 N. Lindbergh Blvd, St. Louis, MO, 63167, 314-537-6685
Newton, K., Dept Biol Sci, University of Missouri, Columbia, MO, 65211, 314-882-4049
Nickerson, N. H., Department of Biology, Tufts University, Medford, MA, 02155, 617-381-3544
Nider, Fabio, Dekalb Argentina Saciai, C.C. 25, 2741 Salto, Buenos Aires, Argentina, 0474-22350
Nieto-Sotelo, Jorge, Department of Biology, Campus Box 1137, Washington University, St. Louis, MO, 63130, 314-889-4632
Nitsch, C., CNRS, Genet Phys Devel Plantes, 91190 Gif-Sur-Yvette, France, 6-907-7828 EXT 387
Noble, Jr., Stephen W., Dept of Corn Breeding, Pioneer Hi-Bred Internat Inc, Johnston, IA, 50131, 515-270-3318
Notani, N. K., Bhabha Atomic Res Centre, Biology Division, Mod Lab, Trombay, Bombay 400085, India, 5510323

Novak, F. J., Czech Acad Sci, Inst Exp Bot, Sokolovska 6 772 00, Olomouc, Czechoslovakia, 068-29521
Oaks, Ann, Biology Department, McMaster Universtiy, Hamilton, Ontario L8S 4K1, Canada, 416-525-9140 EXT 4377
Oberthur, Elizabeth E., 27, Cytogenetics, Agronomy Department, Iowa State University, Ames, IA, 50010, 515-294-1360
Ochieng, Joseph A. W., NARS-Kitale, P.O. Box 450, Kitale, Kenya
Ogle, Charles W., RR 1, Box 162, Sugar Grove, IL, 60554, 312-466-4742
Ohta, Y., Inst Agr & Forestry, Univ of Tsukuba, Sakura-Mura, Ibaraki-Ken 305, Japan, 0298-53-6691
Okabe, Takashi, Hokkaido Nat Agr Exper Sta, Hitsujigaoka, Sapporo, Japan
Okuno, K., Div Genetics, NIAS, Yatabe, Tsukuba 305, Japan
Openshaw, Steve, Dekalb Pfizer Genetics, 3100 Sycamore Road, Dekalb, IL, 60115, 815-756-3671
Osler, R. D., CIMMYT, Apartado Postal 6-641, 06600 Mexico D.F., Mexico, (905) 5-85-43-55
Osterman, John, School of Life Sciences, University of Nebraska-Lincoln, Lincoln, NE, 68588, 402-472-5129
Ottaviano, E., Dip de Biol Sez di Genet, e Microbiol, Via Celoria 26, 20133 Milano, Italy, 02-230823
Paez, Alix V., PO Box 316, Pioneer Hybrid Intern Inc, 6800 Pioneer Pkwy, Johnston, IA, 50131, 515-270-3145
Palmer, Reid, Agronomy Department, Iowa State University, Ames, IA, 50010, 515-294-7378
Palmer, Susan E., Box 54, Medical Center, School of Medicine, West Virginia University, Morgantown, WV, 26506
Paterniani, Ernesto, Univ Sao Paulo Esc Sup Agr, Luiz de Queiroz, Piracicaba, Brazil
Patterson, Earl, Agron Dept, S-116 Turner Hall, Univeristy of Illinois, 1102 S. Goodwin Avenue, Urbana, IL, 61801, 217-333-4254
Pawar, S. E., Biol/Agric Div, Bhabha Atomic Research Center, Trombay Bombay 400085, India, 5514910 ext 2340
Peacock, W. J., Division of Plant Industry, CSIRO, Canberra ACT, Australia, 062-465250
Pelham, John, Inst of Terrestrial Ecol, Bush Estate, Penicuik, Midlothian EH26 0QB, Scotland
Penny, L., Asgrow Seed Co, 634 E. Lincoln Way, Ames, IA, 50010, 515-232-7170
Pereira, Andy, 27, Cytogenetics, Agronomy Department, Iowa State University, Ames, IA, 50010
Perkins, K. W., Carolina Bio Supply Co, Burlington, NC, 27215, 919-584-0381
Perry, H. S., 517 Westwood Drive North, Tallahassee, FL, 32304, 904-575-7970
Peschke, Virginia M., Dept. Agron. & Plant Genetics, Univ. of Minnesota, St. Paul, MN, 55108
Peters, D. W., O's Gold Seed Co, PO Drawer D, Farmer City, IL, 61842, 309-928-9445
Peterson, Karl, Box 187, Marshalltown, IA, 50158, 515-752-2170
Peterson, Peter A., Dept of Agronomy, Iowa State University, Ames, IA, 50011, 515-294-9652
Peterson, Thomas A., CSIRO, Division of Plant Industry, P.O. Box 1600, Canberra, A.C.T. 2601, Australia
Pfahler, P. L., Agronomy Dept Univ Fl, 304 Newell Hall, Gainesville, FL, 32611, 904-392-6186
Pfister, Walter, Pfister Hybrid Corn Co, El Paso, IL, 61738
Phillips, Ronald, Agronomy & Pl Genetics, University of Minnesota, St. Paul, MN, 55108, 612-373-1366
Phinney, Bernard O., Division of Botany, University of Calif, Los Angeles, CA, 90024, 213-825-3177
Pinter, Lajos, Research Inst for Forage Crops, Szentlorinc P.O. 005, H-7940, Hungary
Piovarci, A., Maize Research Institute, 917 52 Trnava, Czechoslovakia
Plewa, Michael, 302 Environ Res-1005 W Western, University of Illinois, Urbana, IL, 61801, 217-333-3614
Poethig, R. Scott, Biology Department, Leidy Labs/G7, University of Pennsylvania, Philadelphia, PA, 19104, 215-898-8915
Poff, Kenneth L., MSU-DOE Plant Res Lab, Michigan State University, East Lansing, MI, 48824-1312
Pogna, N. E., Exp Inst Cer Res, 20079 S. Angelo, Lodigiano, Italy
Polacco, Mary, Biochemistry 121 Med Sci, Univ of Missouri, Columbia, MO, 65211, 314-882-6675
Pollacsek, M., Station D Amelior Des Pl-INRA, 63039 Clermont Ferrand, France, 73-92-45-30
Pollak, Linda, Dept of Agronomy, Iowa State Univ, Ames, IA, 50011, 515-294-3052
Pollmer, W. G., Universitat Hohenheim Lh, Postf 700562-304, Garbenstrasse 9, 7000 Stuttgart 70, W Germany
Polowick, Patricia L., Department of Biology, Univ of Saskatchewan, Saskatoon, Sask. S7N 0W0, Canada, 343-5280
Poneleit, Charles G., Agronomy, University of Kentucky, Lexington, KY, 40546-0091, 606-257-4934
Popow, G., Dubendorfstrasse 30, CH-8602 Wangen (ZH), Switzerland, 01-833-2456
Potts, Yvonne, 1826 12th Avenue N., Grand Forks, ND, 58201, 701-746-7896
Pradhan, K., West Bengal Haringhata Farm, PO Mohanpur, Dist Nadia, West Bengal, India
Prensky, Wolf, Braton Biotechnical Inc, Box 234, Hawthorne, NY, 10532, 914-747-1522
Price, S. C., Standard Oil Co, 3092 Broadway, Cleveland, OH, 44115, 216-271-8192
Pring, D. R., Dept of Plant Pathology, University of Florida, Gainesville, FL, 32611, 904-392-3638
Pryor, A. J., Div Plant Ind CSIRO, PO Box 1600, Canberra Act 2601, Australia, 062-46-5896
Purdy, J. L., Green Giant Company, Le Sueur, MN, 56058, 612-665-6730
Putnam, P., USDA ARS, P.O. Box 70, Ames, IA, 50010
Raab, Quinton J., Asgrow Seed Company, 205 N. Michigan, Rt. 1 Box 22, Oxford, IN, 47971, 317-385-2201
Racchi, M. L., Univ di Milano, Dip di Biol, Sez di Genetica, Via Celoria 26, 20133 Milan, Italy

Rakha, Farouk A., Dep Genetics Fac Agric, Alexandria University, Alexandria, Egypt, 966386
Raman, Rajhi, 114, NRL, Indian Agr Res Inst, New Delhi 110012, India
Rao, Arelli P., 16-11-417/6, Mosarambagh, Hyderabad-500036 AP, India
Rao, P. N., Dept of Botany, Andhra University, Visakhapatnam 530003, India, 64871 ext 390
Reddy, A. R., School of Life Sci, Univ of Hyderabad, Hyderabad-500001, India
Reddy, G. M., Department of Genetics, Osmania University, Hyderabad-500007, India
Reddy, Lekkala V., Biochemistry Department, University of Iowa, Iowa City, IA, 52242, 319-353-7333
Redei, G. P., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-6434
Reed, J. E., Pfizer Genetics, Box 367, Windfall, IN, 46076, 317-945-7121
Rees, Carol, Plant Sciences Dept, Univ of Western Ontario, London, Ontario, Canada, 519-679-2900
Rees, Hubert, Agricultural Botany Dept, Univ College of Wales, Aberystwyth, Wales, U K, 0790-3111
Reid, W. R., O's Gold Seed Company, P.O. Box 460, Parkersburg, IA, 50665
Reksodihardjo, S., Pioneer Agricultura Ltda, Caixa Postal 131, 86100 Londrina PR, Brazil, 0432 271512
Remley, Frank M., Cargill Inc, Seed Div, PO Box 5645, Minneapolis, MN, 55440, 612-890-1852
Rhoades, M. M., Biology Dept, Indiana University, Bloomington, IN, 47401, 812-335-9319
Rhodes, C., Calgene Inc, 1920 Fifth St, Suite F, Davis, CA, 95616, 916-753-6313
Riccelli, Mauricio, Apartado Postal 236, Maracay 2101A, Venezuela, 43-23793
Rice, Thomas B., Pfizer Inc, Eastern Point Road, Groton, CT, 06340, 203-441-4576
Rieffel, Larry D., 598 East 2nd Street, El Paso, IL, 61738, 309-527-2676
Rinke, E. H., 9617 N. Glen Oaks C.R.N., Sun City, AZ, 85351, 602-933-9646
Rivin, Carol, Dept. of Botany, Oregon State University, Corvallis, OR, 97331, 503-754-4433
Robbelen, G., Inst fur Pflanzenbau, 34 Gottingen, Vonsiebold Strasse 8, W Germany
Roberts, Jean L., 203 N. Hugo Street, Indianapolis, IN, 46229, 317-894-1490
Robertson, Dominique, Dept Plant Breeding, Cornell University, Ithaca, NY, 14853
Robertson, Donald S., Department of Genetics, Iowa State University, Ames, IA, 50010, 515-294-3900
Robichaud, C., Dept of Biology, Yale Univ, New Haven, CT, 06511, 203-432-4636
Rodrigues, Delmo Diogo, Sem Cargill Ltda, Sitio Sao Jao Barao Geraldo, CP 6553, Campinas S.P. 13.100, Brazil, 0192-39-13-81
Roper, J. A., Dept of Genetics, The University, Sheffield 10, U K, 0742-78555
Rossman, E. C., Dept of Crop & Soil Sci, Michigan State University, East Lansing, MI, 48823, 517-355-2289
Rubenstein, Irwin, Dept Genetics & Cell Biol, University of Minnesota, St Paul, MN, 55108, 612-373-1733
Russell, W. A., Dept of Agronomy, Iowa State University, Ames, IA, 50010
Ryan, G. S., E.E.A. INTA, 5988 Manfredi, (Cordoba), Argentina
Sachan, J. K. S., 114, NRL Div Genet, IARI, New Delhi-12 110012, India, 588783
Sachs, M. M., Department of Biology, Campus Box 1137, Washington University, St. Louis, MO, 63130, 314-889-4631
Saedler, H., Max-Planck Inst Zuchtungsforsc, Egelspfad, D 5000 Koln 30 Vogelsang, W Germany, 221-5062200
Sager, Ruth, DANA Cancer Ctr, 44 Binney Street, Boston, MA, 02115, 617-732-3300
Saha, B. C., Agriculture College, PO Sabour Dist Bhagalpur, Bihar, India 813210
Salama, Fawzy A., Nubaria Research Station, El-Naser North Tahrir Sector, El-Ameria, Egypt
Salamini, Francesco, Ist Sper Cerealicoltura, Via Stezzano 24 (PO Box 164), 24100 Bergamo, Italy, 035-248823
Salerno, Juan C., Dept Genetica, INTA, Castelar, C.C. 25, 1712-, Castelar- Buenos Aires, Argentina
Sanchez, F. M., Colegio de Postgraduados, Chapingo, Mexico, 585-0728
Sano, Y., Nat Inst Genetics, Misima, Japan
Saraiva, L. S., Genetica Dept Biol Geral, Univ Fed Vicosa, 36570 Minas Gerais, Brazil
Sarkar, K. R., Room 114, Nuclear Research Lab, Indian Agric Res Inst, New Delhi-12, India
Sarma, J. S. P., Dept of Botany, C.S.R. Sarma College, Ongole-523 001, Andhra Pradesh, India, 2684
Sastry, G. R. K., Dept of Genetics, University of Leeds, Leeds-2, U K, 0532-431751 EXT 598
Sauvaire, D., IRAT, B.P. 32, Bobo-Dioulasso, Upper Volta
Saxena, V. K., Dept Plant Breed, Punjab Agric Univ, Ludhiana 141004, India
Sayed, Sayed Galal, PO Box 953, Cairo, Egypt, 847718
Scandalios, J. G., Dept of Genetics, N C State Univ, Raleigh, NC, 27650, 919-237-2291
Schaeffer, Jurgen R., Dept of Plant + Soil Sci, Montana State University, Bozeman, MT, 59715, 406-994-5066
Schertz, K. F., Dept Crop & Soil Sci, Texas A & M Univ, College Station, TX, 77843, 409-260-9252
Schnable, Patrick, Dept of Agronomy, Iowa State Univ, Ames, IA, 50011, 515-292-3909
Schnell, F. W., Windhalmweg 19, 7000 Stuttgart 70, W Germany, 0711-453367
Schwartz, Drew, Biology Dept, Indiana University, Bloomington, IN, 47401, 812-335-6060
Schwer, Joseph F., Lilly Greenfield Labs, P.O. Box 708, Greenfield, IN, 46140

Schy, W. E., 112 Environ Res-1005 W Western, Univ of Illinois, Urbana, IL, 61801, 217-333-0010
Scoles, Graham J., Crop Sci Dept, Univ of Sakatchewan, Saskatoon S7N 0W0, Canada, 306-343-5700
Scott, Gene E., Agronomy Dept, Box 5248, Mississippi State University, Mississippi State, MS, 39762, 601-325-2311
Sehgal, S. M., Pioneer Hi-Bred Intnl Inc, 6800 Pioneer Pkwy, Johnston, IA, 50131, 515-270-3103
Sevilla P., Ricardo, Programa de Maiz, Univ Nacional Agraria, Aptdo 456, La Molina, Lima, Peru
Shadley, Jeff D., Illinois State University, Dept Biol Sciences, Normal, IL, 61761, 309-438-2685
Shafer, John, 3031 N Roselawn Drive, Logansport, IN, 46947, 219-753-5547
Sharma, N. D., 114 N R L, Indian Agric Res Inst, New Delhi-12, India
Shaver, D. L., Plant Br Dept, Cornnuts, 24 Winham St, Salinas, CA, 93901, 408-424-1023
Shcherbak, V. S., Krasnodar Res Inst Agric, Krasnodar, USSR
Shepherd, Hurley, Dept Botany, Univ of Kansas, Lawrence, KS, 66045, 913-864-3939
Sheridan, W. F., Biology Department, Univ of North Dakota, Grand Forks, ND, 58202, 701-777-4479 or 777-4206
Shortess, David K., Department of Biology, New Mexico Inst M + T, Socorro, NM, 87801, 505-835-5608
Shulman, Alan H., Dept Biology, Yale Univ Box 6666, New Haven, CT, 06511, 203-432-4636
Shure, M., Carnegie Inst Wash, 115 W Univ Parkway, Baltimore, MD, 21210, 301-467-1414
Sidhu, G., Dept Plant Path, Univ of Nebraska, Lincoln, NE, 68583, 402-472-6024
Simchen, Giora, Department of Genetics, The Hebrew University, Jerusalem 91904, Israel 91904
Simcox, K., Dept Plant Path, Univ of Illinois, N-519 Turner Hall, Urbana, IL, 61801
Simon, R. D., Dept of Biology, State Univ Coll A + S, Geneseo, NY, 14454, 716-245-5301
Sinclair, John, Biology Department, Indiana University, Bloomington, IN, 47405, 812-335-1201
Singh, I. S., Dept of Plant Breeding, G. B. Pant Univ, Agr & Tech, Pantnagar, Nainital, India
Singh, Joginder, Cereal Research Lab, Indian Agricultural Res Inst, New Delhi 110012, India, 582372
Sisco, P. H., Dept Crop Science, North Carolina State Univ, P.O. Box 7620, Raleigh, NC, 27695-7620, 919-737-2704
Skogen-Hagenson, Mary Jane, Department of Genetics, Rm. 10 Curtiss Hall, Iowa State University, Ames, IA, 50011, 515-294-4618
Smeltzer, Dale, The Rockefeller Found, GPO Box 2453, Bangkok, Thailand
Smith, James D., PO Box 2132, Department Soil & Crop Sci, Texas A & M University, College Station, TX, 77843, 409-845-8276
Smith, Oscar S., Dept of Agronomy, Iowa State University, Ames, IA, 50011, 515-769-2212
Smith, Steve, Pioneer Hi-Bred Internatl, Plant Breeding Division, 7301 N.W. 62nd Avenue, Johnston, IA, 50131, 515-270-3353
Snope, Andrew, Div of Science + Math, Essex Community College, Essex, MD, 21237, 301-522-1280
Snyder, John R., United Agriseeds, 104 W University Ave, Urbana, IL, 61801, 217-344-0900
Soave, C., Inst Biosintesi Veg Cnr, Via Bassini 15, Milano, Italy
Soledad, S. V., Tagum, Northdavao 9401, Philippines
Song, Maoshan, Shanxi Acad Agric Sci, Wugong, Shanxi, China
Soong, Tom, Monsanto, 800 N. Lindbergh, GG4G, St. Louis, MO, 63167, 314-537-6601
Sorenson, John C., Experimental Ag Sci, The Upjohn Co, Kalamazoo, MI, 49001, 616-385-4582
Sprague, G. F., Dept Agron, S-120 Turner Hall, University of Illinois, 1102 S. Goodwin Avenue, Urbana, IL, 61801, 217-333-4254
Stark, Frank, Illinois Foundation Seeds Inc, 6959 River Road, Deforest, WI, 53532, 608-846-3066
Starlinger, P., Inst Genetik, Univ Koln, D-5000 Koln 41, Weyertal 121, W Germany, 221-470-2465
Staub, Rick W., Dept of Biology, Carleton College, Northfield, MN, 55057, 507-663-4394
Steffensen, D. M., 515 Morrill Hall, Univ of Illinois, Urbana, IL, 61801, 217-333-3087
Stein, Otto L., Dept of Botany, Univ of Massachusetts, Amherst, MA, 01003, 413-545-0861
Stern, Herbert, Dept of Biology, Univ of Calif-San Diego, La Jolla, CA, 92037, 619-452-3746
Stiles, J. I., Dept. of Botany, Univ. Hawaii at Manoa, Honolulu, HI, 96822
Stout, John T., Upjohn Company, 9602-18-2, Kalamazoo, MI, 49001, 616-385-4583
Strommer, Judith, Dept of Genetics, Univ of Georgia, Athens, GA, 30602, 404-542-7980
Stroup, D., 4740 Conn. Ave. NW #613, Washington, DC, 20008
Stuber, Charles W., Dept of Genetics, North Carolina State Univ, Raleigh, NC, 27650, 919-737-2289 FTS672-4564
Stucker, Robert, Dept of Agron + Pl Gen, University of Minnesota, St Paul, MN, 55108
Styles, E. D., Biology, Univ of Victoria, Victoria, British Columbia V8W 2Y2, Canada, 604-721-7101
Sung, Tong Ming, Dept of Agronomy, Beijing Agric Univ, Beijing, China, 28-5831
Sussex, I. M., Dept Biol, PO Box 6666, Yale University, New Haven, CT, 06511, 203-432-4636
Swaminathan, M. S., Indian Agricultural Res Inst, 1, Motilal Nehru Marej, New Delhi 1100011, India
Tantravahi, R. V., Sub Exp Sta, Univ Mass, 240 Beaver Street, Waltham, MA, 02154
Taylor, Brian H., CSIRO, Division Plant Industry, Clunies Ross St, Acton, A.C.T., Australia
Taylor, Loverine P., Dept Biol Sci, Stanford Univ, Stanford, CA, 94305, 415-497-2609
Thompson, D., 191 Summer Blvd, Collinsville, IL, 62234, 618-345-0426

Thompson, Jack C., FFR Coop, P.O. Box 10, Providence Forge, VA, 23140, 804-966-2888
Thompson, M. E., Medical Department, Brookhaven National Lab, Upton, NY, 11973
Thornbury, David W., S-305 Ag Science Center N, Dept Plant Path, University of Kentucky, Lexington, KY, 40546, 606-257-2681
Timothy, David H., Dept of Crop Science, North Carolina State Univ, Raleigh, NC, 27607, 919-834-5161
Ting, Chu-Po, Shandong Agricultural College, Taian, Shandong, China
Ting, Yu-Chen, Biology, Boston College, Chestnut Hill 67, Boston, MA, 02167, 617-969-0100 EXT 3551
Tintorri, John A., 53 Hampshire Drive, Mendham, NJ, 07945
Tomov, Nicola, Institute for Maize, Kneja, Bulgaria
Tonelli, Chiara, Dip Biol, Sez di Gen & Micro, Univ Milano, Via Celoria 26, 20133 Milano, Italy, 02-230823
Tracy, W. F., Department of Agronomy, University of Wisconsin, Madison, WI, 53706, 608-262-1390
Troyer, A. F., Dekalb-Pfizer Genetics, 3100 Sycamore Road, Dekalb, IL, 60115, 815-758-3461
Tsai, C. Y., Dept Bot Plant Pathol, Purdue University, Lafayette, IN, 47907, 317-494-4640
Tsotsis, Basil, Dekalb-Pfizer Genetics, Sycamore Road, Dekalb, IL, 60115, 815-756-3671 EXT 265
Tsuchiya, T., Dept of Agronomy, Colorado State University, Fort Collins, CO, 80523, 303-491-6648 EXT 6237
Uchimiya, H., Inst Biol Sciences, Univ Tsukuba, Sakura-Mura, Ibaraki-Ken, Japan, 0298-51-1384
Ukai, Yasuo, Inst Radiation Breed, Nias Maff, Ohmiya-Machi, Naka-Gun, Ibaraki 319-22, Japan, 02955-3-0807
Ulmer, R. L., Anheuser-Busch Tech Cent, One Busch Place, St. Louis, MO, 63118, 314-577-2824
Ulrich, V., Div Plant & Soil Sci, West Virginia Univ, Morgantown, WV, 26506, 304-293-6258
Umbeck, P. F., Cetus Madison Corporation, 2208 Parview Road, Middleton, WI, 53562, 608-836-7300
Van Der Walt, W. J., C/O Asgrow South Africa, Box 2054, Pretoria, S Africa
Van Schaik, N., Dept Genetics/U Witwatersrand, 1 Jan Smuts Avenue, Johannesburg 2001, S Africa, (011) 716-2125
Varner, Joseph E., Department of Biology, Washington University, St. Louis, MO, 63130, 314-889-6803
Vasal, S. K., Londres 40 1 Piso, Apartado Postal 6-641, Mexico 6, D.F., Mexico, 5-85-43-55 EXT 115
Vasil, Indra K., Dept Botany, 220 Bartram Hall, Univ Florida, Gainesville, FL, 32611, 904-392-1193
Vega, Pedro C., Instituto de Genetica, Univ Central Venezuela, Maracay, Venezuela
Verma, S. C., Botany Department, Panjab University, Chandigarh-160014, India
Vetturini, Mario, Via Marco Polo, 48, 40138 Bologna, Italy, 051 364181
Vineyard, Marvin L., 695 Dogwood Drive, Arcola, IL, 61910, 217-268-3426
Violic, Alejandro D., CIMMYT, Apartado Postal 6-641, Mexico 6, D.F., Mexico, 5854355
Vodkin, Lila Ott, Seed Res Lab PGGI 006, Beltsville Agr Res Ctr-W, Beltsville, MD, 20705, 301-344-2491
Vojnikov, V., Siber Inst Pl Phys & Biochem, USSR Academy of Sciences, P.O. Box 1243, Irkutsk-33, 664033, USSR
Volk, B., Agronomy Dept, 135 Mumford, Univ of Missouri, Columbia, MO, 65211, 314-882-2801
Von Bulow, J. F. W., Univ Brasilia, Cx Postal 15-2958, 70.910 Brasilia D.F., Brazil, 274-0022 r.2429
Von Wettstein, D., Carlsberg Lab, Gamle Carlsbergvej 10 DK-2500, Copenhagen Valby, Denmark, 01-221022
Voris, Merle, Box 457, Windfall, IN, 46076, 317-945-7777
Vosa, Canio G., Botany School, Oxford University, Oxford OS1 3RA, U K, 53391 EXT 233
Waines, J. Giles, Dept of Botany/Plant Sciences, University of California, Riverside, CA, 92502, 714-787-3706
Walbot, V., Dept Biol Sci, Stanford Univ, Palo Alto, CA, 94305, 415-497-2007
Walden, D. B., Dept of Plant Sciences, Univ of Western Ontario, London N6A 5B7, Canada, 519-679-6757
Waller, Jerome H., Berea College, C P O Box 2325, Berea, KY, 40404
Wallin, J. R., Curtis Hall, Univ of Missouri, Columbia, MO, 65211, 314-882-6320
Walton, Jonathan, Dept Plant Breed & Biometry, Cornell University, Ithaca, NY, 14853, 607-256-3102
Wang, Andrew S., Sungene Technologies Corp. , 3330 Hillview Ave., Palo Alto, CA, 94304
Wang, Lai-Weng, Institute of Maize, Shandong Acad Agric Sci, Jinan, Shandong Province, China
Wang, Mao-Hua, Dept Agronomy, Henan Agric College, Zhengshou, Henan Province, China
Warren, Herman L., Dept Bot Plant Pathol, Purdue University, Lafayette, IN, 47907, 317-494-4630
Wassom, Clyde E., Agronomy Dept, Kansas State Univ, Manhattan, KS, 66506, 913-532-7253
Watson, John T., Pioneer Hibred Int, 1206 Mulberry Street, Des Moines, IA, 50308, 515-270-3152
Weber, David, Dept of Biological Sci, Illinois State Univ, Normal, IL, 61761, 309-438-2685
Weck, Edward A., Stauffer Chemical Co, 1200 S 47th St, Richmond, CA, 94804, 415-231-1000
Wei, Jia-Kun, Hebei Agric Reclaim Inst, Tangshan, Hebei Province, China
Weiser, Glen C., Agronomy Dept, Walster Hall, North Dakota State Univ, Fargo, ND, 58102, 701-327-8164
Wessells, Catherine P. B., 106 Peter Coutts Circle, Stanford, CA, 94305, 415-497-2609
Wessler, Sue, Dept of Botany, Univ of Georgia, Athens, GA, 30602, 404-542-3732
West, D. P., Research Dept, Jacques Seed Company, Prescott, WI, 54021, 715-262-3162
West, Dennis R., Dept Plant and Soil Sci, Univ of Tennessee, Knoxville, TN, 37901, 615-974-7391

Whalen, R. H., Dept of Biology, South Dakota State Univ, Brookings, SD, 57007, 605-688-6141
Whitwood, W., Musser Seed Co., P.O. Box 787, Caldwell, ID, 83605, 208-459-0243
Wicks, Z. W., Dept Plant Sci, South Dakota State Univ, Brookings, SD, 57007, 605-688-5156
Widholm, J. M., Dept of Agronomy, U Illinois/1102 S. Goodwin, Urbana, IL, 61801, 217-333-1279
Widstrom, Neil W., Coastal Plain Exp Sta, PO Box 748, Tifton, GA, 31793, 912-382-6904
Wiesmeyer, Herbert, Dept Molecular Biol, Vanderbilt University, Nashville, TN, 37235, 615-322-4737
Wilcox, Wesley C., Funk Seeds Internat, 1300 West Washington St, Bloomington, IL, 61701, 309-829-9461
Wilkes, H. Garrison, Biology-College II, Univ of Mass/Boston, Boston, MA, 02125, 617-929-8448
Williams, A. F., Rt 4, Box 172, Mitchell, IN, 47446, 812-388-6385
Williams, Robert E., Columbiana Seed Company, PO Box 294, Pittsfield, IL, 62363, 217-285-2530
Williams, Terrill E., Pioneer Hi-Bred Internatl, P.O. Box 426, Windfall, IN, 46076, 317-945-8217
Williams, W. P., Crop Sci Res Lab, PO Box 5248, Miss. State, MS, 39762, 601-325-2311
Wilmarth, C. E., Cargill Research, 515 River Ave North, Belmond, IA, 50421, 515-444-4488
Wilson, Curtis M., Dept Agron, Turner Hall, University of Illinois, 1102 S. Goodwin, Urbana, IL, 61801, 217-333-1117
Woodman, James C., Molec Genetics Inc, 10320 Bren Road East, Minnetonka, MN, 55343, 612-935-7335
Wright, Steven, Dairyland Res Internat, R.R. #1, Box 51, Clinton, WI, 53525
Wurtzel, Eleanore, Biology Dept - Bldg 463, Brookhaven National Lab, Upton, NY, 11973, 516-282-3396
Xie, Daohong, Jilin Acad Agric Sci, Gongzhuling, Jilin, China
Xie, You-Ju, Dept of Agronomy, Beijing Agricultural Univ, Beijing, China
Xu, Qifeng, Dept of Agronomy, Beijing Agric Univ, Beijing, China
Yabuno, T., Yamanouchi Moto Machi 13-2, Sumiyoshi-Ku, Osaka 558, Japan
Yaklich, Robert W., Seed Research Lab, PGGI, B-006, Rm 103, BARC-W, Beltsville, MD, 20705, 301-344-2491
Yamada, Minoru, Div Genet NIAS, Kan-Nondai 3-1-1 Yatabe, Tsukuba Ibaraki 305, Japan, 02975-6-8372
Yan, Tang Ching, CIMMYT, Londres 40, Apdo Postal 6-641, 06600 Mexico, D.F., Mexico
Yi, Bu-young, Inst Biological Chemistry, Washington State University, Pullman, WA, 99164-6340, 509-335-3956
Yoder, O. C., Dept Plant Path, Cornell Univ, Ithaca, NY, 14853, 607-256-3243
Yong, H. S., Dept Genetics, University of Malaya, Kuala Lumpur, Malaysia
Yu, Ming H., Agr Res Service, PO Box 5098, Salinas, CA, 93915, 408-443-2253
Zabala, Gracia, Dept Biol Sci, Stanford University, Stanford, CA, 94305, 415-497-2609
Zainal-Abidin, M. I., Dept Agron/Hort, Univ Agr Malaysia, Serdang, Selangor, Malaysia, 356101-326
Zeng, M. Q., Institute of Genetics, Academia Sinica, Beijing, China, 446551-556
Zhao, Ji-ping, Department of Agronomy, Shandong Agric University, Taian, Shandong Province, China
Zhao, Zuo-Yu, Department of Biological Sci, Illinois State University, Normal, IL, 61761, 309-438-3669
Zimmer, Elizabeth, Dept of Biochemistry, 322 Choppin Hall, Louisiana State University, Baton Rouge, LA, 70803, 504-388-1556
Zimmering, Stanley, Dept of Biology, Brown University, Providence, RI, 02912
Zuber, M. S., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-7707

## VII. RECENT MAIZE PUBLICATIONS


1. Abbott, A. G., Ainsworth, C. C., Flavell, R. B., 1984. Characterization of anther differentiation in cytoplasmic male sterile maize using a specific isozyme system (esterase). Theor. Appl. Genet. 67:469-474.
2. Abdelrahman, A. A., Hoseney, R. C., 1984. Basis for hardness in pearl millet, grain sorghum, and corn. Cereal Chem. 61:232-235.
3. Adams, N. J., Scott, G. E., King, S. B., 1984. Quantification of Aspergillus flavus growth on inoculated excised kernels of corn genotypes. Agron. J. 76:98-102.
4. Afuakwa, J. J., Crookston, R. K., 1984. Using the kernel milk line to visually monitor grain maturity in maize. Crop Sci. 24:687-691.
5. Afuakwa, J. J., Crookston, R. K., Jones, R. J., 1984. Effect of temperature and sucrose availability on kernel black layer development in maize. Crop Sci. 24:285-288.
6. Albergoni, F., Basso, B., Pe, E., Ottaviano, E., 1983. Photosynthetic rate in maize. Inheritance and correlation with morphological traits. Maydica 28:439-448.
7. Alexander, W. L., Cross, H. Z., 1983. Grain fill characteristics of early maize (Zea mays) strains selected for variable R-nj expression. Euphytica 32:839-844.
8. Altman, A., Cohen, B. N., Weissbach, H., Brot, N., 1984. Transcriptional activity of isolated maize chloroplasts. Arch. Biochem. Biophys. 235:26-33.
9. Altschuler, M. I., Schwartz, D., 1983. Minor electrophoretic forms of maize alcohol dehydrogenase: their in vitro production by beta-mercaptoethanol. Maydica 28:401-410.
10. Altschuler, M. I., Schwartz, D., 1984. Effects of phytate on maize alcohol dehydrogenase isozymes. Maydica 29:77-87.
11. Anderson, E. L., Kamprath, E. J., Moll, R. H., 1984. Nitrogen fertility effects on accumulation, remobilization, and partitioning of N and dry matter in corn genotypes differing in prolificacy. Agron. J. 76:397-403.
12. Anderson, E. L., Kamprath, E. J., Moll, R. H., Jackson, W. A., 1984. Effect of N-fertilization on silk synchrony, ear number, and growth of semiprolific maize genotypes. Crop Sci. 24:663-666.
13. Aranda, G., Gauvrit, C., Cesario, J., Guilhem, C., Pascard, C., Tran Huu Dau, M. E., 1983. Biological activity of the two geometrical isomers of Methomyl on maize mitochondria. Phytochemistry 22:2431-2436.
14. Arntzen, C., 1983. Introduction of herbicide resistance into crop plants via molecular techniques. Proc. Annu. Corn and Sorghum Res. Conf. 38:144-152.
15. Atanassov, A., Todorova, L., 1984. In vitro embryo cultures of intergeneric Zea mays x Tripsacum dactyloides hybrids. Genet. Sel. 17:250-257.
16. Atlin, G. N., Enerson, P. M., McGirr, L. G., Hunter, R. B., 1983. Gibberella ear rot development and zearalenone and vomitoxin production as affected by maize genotype and Gibberella zeae strain. Can. J. Plant Sci. 63:847-854.
17. Atlin, G. W., Hunter, R. B., 1984. Comparison of growth, forage yield and nutritional quality of diploid and autotetraploid maize synthetics. Can. J. Plant Sci. 64:593-598.
18. Avato, P., Bianchi, G., Gentinetta, E., Salamini, F., 1984. Effect of trichloracetic acid on wax composition of normal and mutant maize (Zea mays L.). J. Exp. Bot. 35:245-252.
19. Ayala-Osuna, J., Araujo, S. M. C. D., Lara, F. M., Caetano, M. D. F., 1983. Analysis and selection of characters associated to corn earworm (Heliothis zea Lepidoptera Noctuidae) in the dent composite Jaboticabal XI cycle. An. Soc. Entomol. Bras. 12:175-186.
20. Bagnara, D., Daynard, T. B., 1983. Reciprocal differences in kernel growth among four maize inbreds. Maydica 28:357-364.
21. Bagrationi, N. N., 1984. Dynamics of activity of auxins, gibberellins, and natural growth inhibitors during ontogenesis of tall and short corn. Sov. Plant Physiol. 31:19-22.
22. Baktash, F. Y., Younis, M. A., Al-Younis, A. H., Al-Ithawi, B. A., 1983. Relative effectiveness of 2 systems of selection for protein and oil percentage of the corn grains. Mesopotamia J. Agric. 17:31-36.
23. Barker, R. F., Thompson, D. V., Talbot, D. R., Swanson, J., Bennetzen, J. L., 1984. Nucleotide sequence of the maize transposable element Mu1. Nucl. Acid Res. 12:5955-5967.
24. Barlow, P. W., Pilet, P. E., 1984. The effect of abscisic acid on cell growth, cell division and DNA synthesis in the maize root meristem. Physiol. Plant. 62:125-132.
25. Baron, V. S., Daynard, T. B., 1984. Factors affecting grain dry-down in early-maturing European and Canadian corn hybrids. Can. J. Plant Sci. 64:465-474.
26. Barriere, Y., Montalant, Y., Boyat, A., 1984. Agronomic value and protein content of maize x teosinte progenies. Agronomie 4:417-422.
27. Barriere, Y., Vincourt, P., Montalant, Y., 1984. Study of prolificacy in the 1st selection cycle of 2 early maize populations. Agronomie 4:355-360.
28. Bates, G. W., Gaynor, J. J., Shekhawat, N. S., 1983. Fusion of plant protoplasts by electric fields. Plant Physiol. 72:1110-1113.
29. Beckert, M., Cao, M. Q., 1984. Results of a diallel trial and a breeding experiment for in vitro aptitude in maize. Theor. Appl. Genet. 68:247-251.
30. Behrens, U., Fedoroff, N., Laird, A., Mullerneumann, M., Starlinger, P., Yoder, J., 1984. Cloning of the Zea mays controlling element Ac from the wx-m7 allele. Mol. Gen. Genet. 194:346-347.
31. Bennett, M. D., Heslop-Harrison, J. S., Smith, J. B., Ward, J. P., 1983. DNA density in mitotic and meiotic metaphase chromosomes of plants and animals. J. Cell Sci. 63:173-180.
32. Bennetzen, J. L., Swanson, J., Taylor, W. C., Freeling, M., 1984. DNA insertion in the first intron of maize Adh1 affects message levels: Cloning of progenitor and mutant Adh1 alleles. Proc. Nat. Acad. Sci. USA 81:4125-4128.
33. Bergquist, R. R., Pryor, A. J., 1984. Virulence and isozyme differences for establishing racial identity in rusts of maize. Plant Dis. 68:281-283.
34. Berville, A., Ghazi, A., Charbonnier, M., Bonavent, J.-F., 1984. Effects of methomyl and Helminthosporium maydis toxin on matrix volume, proton motive force, and NAD accumulation in maize (Zea mays L.) mitochondria. Plant Physiol. 76:508-517.
35. Bettencourt, E., Gusmao, L., 1982. Collecting maize germ-plasm in the Azores Islands, Portugal. I. San-Miguel and Terceira. Agron Lusit. 41:241-258.
36. Bhargava, S. C., 1984. Effect of altitude on maize (Zea mays) protein in equatorial environment. Indian J. Plant Physiol. 26:250-257.

37. Bianchi, G., Avato, P., Salamini, F., 1984. Surface waxes from grain, leaves, and husks of maize (Zea mays L.). Cereal Chem. 61:45-47.
38. Binelli, G., Soave, C., Ottaviano, E., 1984. Location and differential expression of a zein gene in maize. Plant Sci. Lett. 33:259-265.
39. Birchler, J. A., 1984. Allozymes in gene dosage studies. Pp. 85-108 in Developments in Plant Genetics and Breeding, Vol. 1, Pt. A, S. D. Tanksley and T. J. Orton, Eds., Elsevier, Amsterdam.
40. Bird, R. McK., 1984. South American maize in Central America? Pp. 43-65 in Pre-Columbian Plant Migration, D. Stone, ed., Harvard University Press, Cambridge, Mass.
41. Blasing, T. J., Duvick, D., 1984. Reconstruction of precipitation history in North American corn belt using tree rings. Nature 307:143-144.
42. Bloc, D., Gay, J. P., Gouet, J. P., 1983. Effect of temperature on the development of maize. Bull. Organ. Eur. Mediterr. Prot. Plant 13:163-170.
43. Bloom, M., Brot, N., Cohen, B. N., Weissbach, H., 1984. Determination of the translation start site of the large subunit of ribulose-1,5-bisphosphate carboxylase from maize. Plant Mol. Biol. 3:403-406.
44. Bogorad, L., Crossland, L. D., Fish, L. E., Krebbers, E. T., Kuck, U., Larrinua, I. M., Muskavitch, K. M. T., Orr, E. A., Rodermel, S. R., Schantz, R., Steinmetz, A. A., Stirdivant, S. M., Zhu, Y. S., 1984. The organization of the maize plastid chromosome--Properties and expression of its genes. Pp. 257-272 in Biosynthesis of the Photosynthetic Apparatus: Molecular Biology, Development and Regulation, J. P. Thornber et al., eds., Alan R. Liss, New York.
45. Bogorad, L., Krebbers, E. T., Larrinua, I. M., Muskavitch, K. M. T., Rodermel, S. R., Steinmetz, A. A., Subramanian, A., 1983. The structure of maize plastid genes and their transcription in vitro. Pp. 63-80 in Advances in Gene Technology: Molecular Genetics of Plants and Animals, K. Downey et al., eds., Academic Press, Orlando.
46. Bonhomme, R., Derieux, M., Duburcq, J. B., Ruget, F., 1984. Variations in ovule number at silking in various corn genotypes. Maydica 29:101-108.
47. Bonhomme, R., Derieux, M., Duburcq, J.-B., Ruget, F., 1984. Variation in leaf number induced by soil temperature in various maize genotypes. Photosynthetica 18:255-258.
48. Boone, L. V., Vasilas, B. L., Welch, L. F., 1984. The nitrogen content of corn (Zea mays) grain as affected by hybrid, population and location. Commun. Soil. Sci. Plant Anal. 15:639-650.
49. Boyer, C. D., Fisher, M. B., 1984. Comparison of soluble starch synthases and branching enzymes from developing maize and teosinte seeds. Phytochemistry 23:733-737.
50. Brakke, M. K., 1984. Mutations, the Aberrant Ratio phenomenon, and virus infection of maize. Ann. Rev. Phytopathol. 22:77-94.
51. Branca, C., Ricci, D., 1984. Studies on elongation of corn stem and root segments by a new auxanometer. Maydica 29:185-191.
52. Briat, J. F., Dron, M., Mache, R., 1983. Is transcription of higher plant chloroplast ribosomal operons regulated by premature termination? FEBS Lett. 163:1-5.
53. Brink, R. A., 1984. Maize endosperm mutants affecting soluble carbohydrate content as potential additives in preparing silage from high protein forages. Maydica 29:265-286.
54. Broglie, R., Coruzzi, G., Keith, B., Chua, N.-H., 1984. Molecular biology of C4 photosynthesis in Zea mays: Differential localization of proteins and mRNAs in the two leaf cell types. Plant Mol. Biol. 3:431-444.
55. Brown, W. L., Zuber, M. S., Darrah, L. L., Glover, D. V., 1984. Origin, adaptation, and types of corn. National Corn Handbook, Purdue University Coop. Ext. Serv. NCH-10. 6 pp.
56. Bruehl, G. W., 1983. Nonspecific genetic resistance to soil-borne fungi. Phytopathology 73:948-951.
57. Buescher, P. J., Phillips, R. L., Brambl, R., 1984. Ribosomal RNA contents of maize genotypes with different ribosomal RNA gene numbers. Biochem. Genet. 22:923-930.
58. Burkard, G., Canaday, J., Crouse, E. J., Gloeckler, R., Gordon, K., Guillemaut, P., Keith, G., Kuntz, M., Mubumbila, M., Osorio, M. L., Steinmetz, A., Weil, J. H., 1982. Protein synthesis in chloroplasts: Sequence studies and gene mapping studies on chloroplast transfer RNA. Cienc. Biol. 7:27-38.
59. Burr, B., Burr, F. A., 1983. McClintock, Barbara--Nobel Prize in Physiology Medicine. Trends Biochem. 8:429-431.
60. Burr, F. A., Burr, B., 1983. Slab gel system for the resolution of oligopeptides below molecular weight of 10,000. Meth. Enzymol. 96, pt. J:239-243.
61. Callow, R. S., Gladwell, I., 1984. A general treatment of chromosome synapsis in even-numbered polyploids. J. Theor. Biol. 106:455-494.
62. Camussi, A., Spagnoletti Zeuli, P. L., Melchiorre, P., 1983. Numerical taxonomy of Italian maize populations: genetic distances on the basis of heterotic effects. Maydica 28:411-424.
63. Capitanio, R., Gentinetta, E., Motto, M., 1983. Grain weight and its components in maize. Maydica 28:365-380.
64. Caporali, L., 1983. Cytological study of cultured cells from maize root cap. Plant Sci. Lett. 31:231-236.
65. Carvajal M., A. T., Kato Y., T. A., Velazquez, A. G., 1983. Variacion en el numero cromosomico del teocintle perenne tetraploide, Zea perennis (Hitchcock), Reeves y Mangelsdorf. Sobr. Agrociencia (Chapingo, Mexico) 54:75-89.
66. Castleberry, R. M., Crum, C. W., Krull, C. F., 1984. Genetic yield improvement of U. S. maize cultivars under varying fertility and climatic environments. Crop Sci. 24:33-36.
67. Caviedes C., M., Quiros, A. C., Kato Y., T. A., Villegas M., E., 1983. Correlaciones fenotipicas entre rendimiento y contenidos de proteina, triptofano y zeina, en familias de medio hermanos de maiz (Zea mays L.) opaco-2 modificado. Sobr. Agrociencia (Chapingo, Mexico) 54:101-110.
68. Ceska, O., Styles, E. D., 1984. Flavonoids from Zea mays pollen. Phytochemistry 23:1822-1823.
69. Chandlee, J. M., Scandalios, J. G., 1983. Gene expression during early kernel development in Zea mays. Develop. Genetics 4:99-116.
70. Chandlee, J. M., Scandalios, J. G., 1984. Regulation of Cat1 gene expression in the scutellum of maize during early sporophytic development. Proc. Nat. Acad. Sci. USA 81:4903-4907.
71. Chao, S., Sederoff, R., Levings, III, C. S., 1984. Nucleotide sequence and evolution of the 18S ribosomal RNA gene in maize mitochondria. Nucl. Acid Res. 12:6629-6644.
72. Chevallier, M.-H., Dattee, Y., 1984. Variabilite enzymatique chez le mais. Can. J. Genet. Cytol. 26:214-228.
73. Chivkunova, O. B., Aksenova, V. A., Kuznetsov, L. V., Timoschenko, A. S., 1982. Analysis of cytoplasmic proteins and amino-acids in strains of the causal agent of common smut in maize differing in ploidy. Biol. Nauki (8):79-83.

74. Chow, K. H., 1984. Alcohol dehydrogenase synthesis and waterlogging tolerance in maize. Trop. Agr. 61:302-304.
75. Christeller, J. T., 1984. Seedling growth of Zea mays at 13 C: Comparison of a corn belt dent hybrid and a hybrid selected for rapid plumule emergence at cool temperatures. J. Exp. Bot. 35:955-964.
76. Churchill, G. A., Andrew, R. H., 1984. Effects of two maize endosperm mutants on kernel maturity, carbohydrates, and germination. Crop Sci. 24:76-81.
77. Chyi, Y. S., Sanford, J. C., Reisch, B. I., 1984. Further attempts to induce "egg transformation" using irradiated pollen. Theor. Appl. Genet. 68:277-283.
78. Cocking, E. C., Green, C. E., Chourey, P., Riedel, G., Gordon, M. P., Chaleff, R., 1983. Somatic cell genetic systems in corn--Discussion. Pp. 156-158 in Advances in Gene Technology: Molecular Genetics of Plants and Animals, K. Downey et al., eds., Academic Press, Orlando.
79. Coe, E. H., Jr., Hoisington, D. A., Neuffer, M. G., 1984. Linkage map of corn (maize) (Zea mays L.). In S. J. O'Brien, ed., Genetic Maps, Vol. 3, Cold Spring Harbor Laboratory, N.Y.
80. Coe, E. H., Jr., Thompson, D. L., Walbot, V., 1982. Nuclear genes and chloroplast modifications in maize. Stadler Genet. Symp. 14:29-46.
81. Cohen, J. I., Galinat, W. C., 1984. Potential use of alien germplasm for maize improvement. Crop Sci. 24:1011-1015.
82. Colbert, T. R., Darrah, L. L., Zuber, M. S., 1984. Effect of recurrent selection for stalk crushing strength on agronomic characteristics and soluble stalk solids in maize. Crop Sci. 24:473-478.
83. Conroy, J. M., Esen, A., 1984. An enzyme-linked immunosorbent assay for zein and other proteins using unconventional solvents for antigen adsorption. Anal. Biochem. 137:182-187.
84. Cooper, P., Ho, T.-H. D., Hauptmann, R. M., 1984. Tissue specificity of the heat-shock response in maize. Plant Physiol. 75:431-441.
85. Crafts-Brandner, S. J., Below, F. E., Harper, J. E., Hageman, R. H., 1984. Differential senescence of maize hybrids following ear removal. I. Whole plant. Plant Physiol. 74:360-367.
86. Crafts-Brandner, S. J., Below, F. E., Wittenbach, V. A., Harper, J. E., Hageman, R. H., 1984. Differential senescence of maize hybrids following ear removal. II. Selected leaf. Plant Physiol. 74:368-373.
87. Crossland, L. D., Rodermel, S. R., Bogorad, L., 1984. Single gene for the large subunit of ribulosebisphosphate carboxylase in maize yields two differentially regulated mRNAs. Proc. Nat. Acad. Sci. USA 81:4060-4064.
88. Cully, D. E., Gengenbach, B. G., Smith, J. A., Rubenstein, I., Connelly, J. A., Park, W. D., 1984. Endosperm protein synthesis and L-[$^{35}$S]methionine incorporation in maize kernels cultured in vitro. Plant Physiol. 74:389-394.
89. Dale, R. M. K., Mendu, N., Ginsburg, H., Kridl, J. C., 1984. Sequence analysis of the maize mitochondrial 26 S rRNA gene and flanking regions. Plasmid 11:141-150.
90. Darmstadt, G. L., Balke, N. E., Price, T. P., 1984. Triazine absorption by excised corn root tissue and isolated corn root protoplasts. Pestic. Biochem. Physiol. 21:10-21.
91. Davidson, D., Pertens, E., Zhao, J.-P., 1983. Chromosome distribution between 2 restitution nuclei in a cell following colchicine treatment. Can. J. Genet. Cytol. 25:437-445.
92. Davidson, D., Pertens, E., Zhao, J.-P., 1983. Chromosome distribution between two restitution nuclei in a cell following colchicine treatment. Can. J. Genet. Cytol. 25:437-445.
93. Dawson, A. J., Jones, V. P., Leaver, C. J., 1984. The apocytochrome b gene in maize mitochondria does not contain introns and is preceded by a potential ribosome binding site. EMBO J. 3:2107-2115.
94. De Jimenez, E. S., Aguilar, R., 1984. Protein synthesis patterns: Relevance of old and new messenger RNA in germinating maize (Zea mays) embryos. Plant Physiol. 75:231-234.
95. Deleens, E., Brulfert, J., 1983. Phosphoenolpyruvate carboxylase capacity and establishment of autotrophy in maize seedlings. Physiol. Veg. 21:827-834.
96. Dellaporta, S. L., 1983. A plant minipreparation: Version II. Plant Mol. Biol. Reporter 1:19-21.
97. Dennis, E. S., Gerlach, W. L., Pryor, A. J., Bennetzen, J. L., Inglis, A., Llewellyn, D., Sachs, M. M., Ferl, R. J., Peacock, W. J., 1984. Molecular analysis of the alcohol dehydrogenase (Adhl) gene of maize. Nucl. Acid. Res. 12:3983-4000.
98. Dennis, E. S., Peacock, W. J., 1984. Knob heterochromatin homology in maize and its relatives. J. Mol. Evol. 20:341-350.
99. DeWet, J. M. J., Newell, C. A., Brink, D. E., 1984. Counterfeit hybrids between Tripsacum and Zea (Gramineae). Amer. J. Bot. 71:245-251.
100. DeWet, J. M. J., Fletcher, G. B., Hilu, K. W., Harlan, J. R., 1983. Origin of Tripsacum andersonii Gramineae. Am. J. Bot 70:706-711.
101. Dhillon, B. S., Khehra, A. S., 1984. Suggestions to overcome the limitation of inbreeding in modified full-sib selection. Cereal Res. Commun. 12:105-106.
102. Dodd, J., 1983. Corn stalk rot: Accounting for annual changes. Proc. Annu. Corn and Sorghum Res. Conf. 38:71-79.
103. Doebley, J. F., 1983. The maize and teosinte male inflorescence--a numerical taxonomic study. Ann. Mo. Bot. Gard. 70:32-70.
104. Doebley, J. F., Goodman, M. M., Stuber, C. W., 1984. Isoenzymatic variation in Zea (Gramineae). Syst. Bot. 9:203-218.
105. Dolbeer, R. A., Woronecki, P. P., Stehn, R. A., 1984. Blackbird (Agelaius phoeniceus) damage to maize: Crop phenology and hybrid resistance. Prot. Ecol. 7:43-64.
106. Donald, C. M., Hamblin, J., 1984. The convergent evolution of annual seed crops in agriculture. Adv. Agron. 36:97-143.
107. Doring, H. P., Geiser, M., Weck, E., Werr, W., Courage-Tebbe, U., Tillmann, E., Starlinger, P., 1983. Comparison of genomic clones derived from the Sh gene in Zea mays L. and of two mutants of this gene which are caused by the insertion of the controlling element Ds. Pp. 203-209 in T. Kosuge et al., eds., Genetic Engineering of Plants, NY/London, Plenum Press.
108. Doring, H. P., Starlinger, P., 1984. Barbara McClintock's controlling elements: Now at the DNA level. Cell 39:253-259.
109. Doring, H. P., Tillmann, E., Starlinger, P., 1984. DNA sequence of the maize transposable element Dissociation. Nature 307:127-130.
110. Dow, E. W., Daynard, T. B., Muldoon, J. F., Major, D. J., Thurtell, G. W., 1984. Resistance to drought and density stress in Canadian and European maize (Zea mays L.) hybrids. Can. J. Plant Sci. 64:575-586.

111. Duburcq, J. B., Bonhomme, R., Derieux, M., 1983. Duration of the vegetative and reproductive periods in maize--Genotypic and environmental effects. Agronomie 3:941-946.
112. Dudley, J. W., 1984. A method for identifying populations containing favorable alleles not present in elite germplasm. Crop Sci. 24:1053-1054.
113. Dudley, J. W., 1984. A method of identifying lines for use in improving parents of a single cross. Crop Sci. 24:355-357.
114. Durbey, S. L., Sarup, P., 1984. Physical characters (plant whorl indices) of different maize germplasms and their possible association with oviposition by the stalk borer, Chilo partellus. J. Entomol. Res. (New Delhi) 7:6-9.
115. Duvick, D. N., 1984. Genetic contributions to yield gains of U.S.A. hybrid maize, 1930-1980. Pp. 15-48 in W. R. Fehr, ed., Genetic Contributions to Yield Gains of Five Major Crop Plants, Amer. Soc. Agron., Madison, WI.
116. El-Nagouly, O. O., El-Kady, M. A., El-Sherbieny, H. Y., 1983. Genetic variance in an opaque-2 maize (Zea mays L.). Egypt. J. Genet. Cytol. 12:277-290.
117. Fakorede, M. A. B., Agbana, S. B., 1983. Heterotic effects and association of seedling vigor with mature plant characteristics and grain yield in some tropical maize cultivars. Maydica 28:327-338.
118. Faluyi, J. O., Olorode, O., 1984. Inheritance of resistance to Helminthosporium maydis blight in maize (Zea mays L.). Theor. Appl. Genet. 67:341-344.
119. Fedoroff, N. V., 1984. Transposable genetic elements in maize. Sci. Amer. 250:84-99.
120. Fedoroff, N. V., Furtek, D. B., Nelson, O. E., Jr., 1984. Cloning of the bronze locus in maize by a simple and generalizable procedure using the transposable controlling element Activator (Ac). Proc. Nat. Acad. Sci. USA 81:3825-3829.
121. Fedoroff, N., 1983. Barbara McClintock (book review). Cell 35:593.
122. Fedoroff, N., 1983. Comparison of host strains for cloning maize DNA in bacteriophage lambda. Plant Mol. Biol. Reporter 1:27-29.
123. Fedoroff, N., 1983. Workshop summary--Genome rearrangements. UCLA Symp. Molec. Cell. Biol. 12:485-488.
124. Fedoroff, N., Chaleff, D., Courage-Tebbe, U., Doring, H. P., Geiser, M., Starlinger, P., Tillmann, E., Weck, E., Werr, W., 1983. Mutations at the Shrunken locus in maize caused by the controlling element Ds. In Structure and Function of Plant Genomes, O. Ciferri and L. Dure, eds. Life Sci. 63:61-72.
125. Fischer, K. S., Johnson, E. C., Edmeades, G. O., 1982. Breeding and selection for drought resistance in tropical maize. Pp. 377-400 in Drought Resistance in Crops with Emphasis on Rice, International Rice Research Institute, Laguna, Philippines.
126. Flavell, R. B., 1984. DNA Transposition--A major contributor to plant chromosome structure. BioEssays 1:21-22.
127. Flavell, R. B., 1984. Transposable elements. Oxford Surv. Plant Mol. Cell. Biol. 1:207-210.
128. Flottum, P. K., Robacker, D. C., Erickson, E. H., Jr., 1984. A quantitative sampling method for airborne sweet corn pollen under field conditions. Crop Sci. 24:375-376.
129. Foley, D. C., Clark, R. L., 1984. Mechanical properties of maize stalks from the plant introduction collection. Crop Sci. 24:1116-1118.
130. Fong, F., Smith, J. D., Koehler, D. E., 1983. Early events in maize seed development. 1-methyl-(3-phenyl-5-(3-[trifluoromethyl]phenyl)-4-(1H)-pyridinone induction of vivipary. Plant Physiol. 73:899-901.
131. Freeling, M., 1983. Isozyme systems to study gene regulation during development--a lecture. Pp. 61-84 in Developments in Plant Genetics and Breeding, Vol. 1, Pt. A, S. D. Tanksley and T. J. Orton, eds., Elsevier, Amsterdam.
132. Freeling, M., 1984. Plant transposable elements and insertion sequences. Annu. Rev. Plant Physiol. 35:277-298.
133. Freier, G., Vilella, F., Hall, A. J., 1984. Within-ear pollination synchrony and kernel set in maize. Maydica 29:317-324.
134. Frohlich, W. G., Dinklaker, J., Klein, D., Pollmer, W. G., 1983. Increase of weight and percentage of dry matter, black layer formation, and expression of pericarp characters in kernels of European maize. Cereal. Res. Commun. 11:237-244.
135. Galante, E., Vitale, A., Manzocchi, L., Soave, C., Salamini, F., 1983. Genetic control of a membrane component and zein deposition in maize endosperm. Mol. Gen. Genet. 192:316-321.
136. Galinat, W. C., 1984. The origin of maize (letter). Science 225:1093-1094.
137. Gamez, R., Leon, P., 1983. Maize Rayado fino virus--Evolution with plant host and insect vector. Pp. 149-168 in Current Topics in Vector Research, Vol. 1, K. F. Harris, ed., Praeger, New York.
138. Gatenby, A. A., 1984. The properties of the large subunit of maize ribulose bisphosphate carboxylase/oxygenase synthesised in Escherichia coli. Eur. J. Biochem. 144:361-366.
139. Geczki, I., Balint, A., Hajos, L., 1984. Investigations into S1 and half-sib progenies of maize. Novenytermeles 33:105-110.
140. Gengenbach, B. G., 1984. Invitro pollination, fertilization, and development of maize kernels. Pp. 276-282 in Cell Culture and Somatic Cell Genetics of Plants, Vol. 1, I. K. Vasil, ed., Academic Press, Orlando.
141. Georgiev, T. M., 1984. Improvement of harvest yield in maize. Proc. 10th Cong. EUCARPIA, Wageningen, the Netherlands, 19-24 June 1983, p. 314.
142. Georgiev, T. M., 1984. Breeding for Stalk and Grain Quality in Maize. Publishing House of the Bulgarian Academy of Sciences, Sofia. 152 pages. (In Bulgarian; tables, a 5-page summary, and Contents in English).
143. Gerrish, E. E., 1983. Indications from a diallel study for interracial maize hybridization in the Corn Belt. Crop Sci. 23:1082-1084.
144. Giles, K. L., Shehata, A. I., 1984. Some observations on the relationship between cell division and cell determination in the epidermis of the developing leaf of corn (Zea mays). Bot. Gaz. 145:60-65.
145. Glass, B., 1984. A feeling for the organism--the life and work of McClintock, Barbara--E. F. Keller. ISIS 75:600.
146. Goldsbrough, P. B., Karcher, S. J., Gelvin, S. B., Larkins, B. A., 1983. Introduction of a zein gene into the Ti plasmid of Agrobacterium tumefaciens. UCLA Symp. Molec. Cell. Biol. 12:35-44.
147. Gomez, L. A., Rodriguez, J. G., Poneleit, C. G., Blake, D. F., Smith, C. R., Jr., 1983. Chemosensory responses of the rice weevil (Coleoptera: Curculionidae) to a susceptible and a resistant corn genotype. J. Econ. Entomol. 76:1044-1048.
148. Gould, S. J., 1984. A short way to corn. Natur. Hist. 93:12-21.

149. Green, C. E., Armstrong, C. L., Anderson, P. C., 1983. Somatic cell genetic systems in corn. Pp. 147-155 in Advances in Gene Technology: Molecular Genetics of Plants and Animals, K. Downey et al., eds., Academic Press, Orlando.
150. Greenblatt, I. M., 1984. A chromosome replication pattern deduced from pericarp phenotypes resulting from movements of the transposable element, Modulator, in maize. Genetics 108:471-485.
151. Gu, M. G., Zhang, X. Q., 1982. Studies on the karyotype of pollen callus clone of maize in subcultures. Kexue Tongbao 27:551-553.
152. Gupta, M., Bertram, I., Shepherd, N. S., Saedler, H., 1983. Cinl, a family of dispersed repetitive elements in Zea mays. Mol. Gen. Genet. 192:373-377.
153. Gupta, M., Shepherd, N. S., Bertram, I., Saedler, H., 1984. Repetitive sequences and their organization on genomic clones of Zea mays. EMBO J. 3:133-139.
154. Guseva, A. I., 1982. Effect of low positive temperatures on maize plants differing in ploidy. Biol. Nauki (8):84-87.
155. Gutheil, R. A., Krause, G. F., Brooker, D. B., Anderson, M. E., 1984. Effect of corn cultivar and sample variance on the performance of three electronic moisture meters. Cereal Chem. 61:267-268.
156. Guthrie, W. D., Barry, B. D., 1983. Evaluation of a mutable system for inducing resistance to European Corn Borer (Lepidoptera: Pyralidae) in susceptible inbred lines of dent maize. J. Kans. Entomol. Soc. 56:552-554.
157. Guthrie, W. D., Jarvis, J. L., Reed, G. L., 1984. Development of a second backup system for European corn borer egg production. Maydica 29:21-26.
158. Guthrie, W. D., Jarvis, J. L., Reed, G. L., Lodholz, M. L., 1982. Plant damage and survival of European corn borer cultures reared for 16 generations on maize plants and for 120 generations on a meridic diet (one generation per year on resistant or susceptible maize plants, eight generations per year on the diet). J. Econ. Entomol. 75:134-136.
159. Hake, S., Taylor, W. C., Freeling, M., 1984. Molecular analyses of genetically stable mutants of the maize Adhl gene. Mol. Gen. Genet. 194:42-48.
160. Hall, A. J., Chimenti, C., Trapani, N., Vilella, F., Cohen de Hunau, R., 1984. Yield in water-stressed maize genotypes: Association with traits measured in seedlings and in flowering plants. Field Crops Res. 9:41-58.
161. Hallauer, A. R., 1984. Reciprocal full-sib selection in maize. Crop Sci. 24:755-759.
162. Hallick, R. B., Bottomley, W., 1983. Proposals for the naming of chloroplast genes. Plant Mol. Biol. Reporter 1:38-43.
163. Hanson, W. D., 1984. Intergradation among Latin American maize based on an analysis of chromosome knob frequencies. Theor. Appl. Genet. 68:347-354.
164. Harpster, M. H., Mayfield, S. P., Taylor, W. C., 1984. Effects of pigment-deficient mutants on the accumulation of photosynthetic proteins in maize. Plant Mol. Biol. 3:59-72.
165. Harris, J. B., Schaefer, V. G., Miksche, J. P., 1984. Influence of aging on nuclear DNA in corn leaves. Plant Cell Physiol. 25:225-232.
166. Hart, L. P., Gendloff, E., Rossman, E. C., 1984. Effect of corn genotypes on ear rot infection by Gibberella zeae. Plant Dis. 68:296-298.
167. Hawes, M. C., Wheeler, H., 1984. Detection of effects of nuclear genes on susceptibility to Helminthosporium maydis Race T by a root cap cell bioassay for HMT toxin. Physiol. Plant Pathol. 24:163-168.
168. Hedin, P. A., Davis, F. M., Williams, W. P., Salin, M. L., 1984. Possible factors of leaf-feeding resistance in corn to Southwestern Corn Borer. J. Agr. Food Chem. 32:262-267.
169. Hedman, K. D., Boyer, C. D., 1983. Allelic studies of the Amylose-Extender locus of Zea mays L.: Levels of the starch branching enzymes. Biochem. Genet. 21:1217-1222.
170. Heikkila, J. J., Papp, J. E. T., Schultz, G. A., Bewley, J. D., 1984. Induction of heat shock protein messenger RNA in maize mesocotyls by water stress, abscisic acid, and wounding. Plant Physiol. 76:270-274.
171. Helms, T. C., Compton, W. A., 1984. Ear height and weight as related to stalk lodging in maize. Crop Sci. 24:923-924.
172. Henry, V. C. R., 1982. Hybridization of maize and its possible application to a local maize improvement programme (reprinted). J. Ag. Soc. Trinidad Tobago 82:215-227.
173. Henson, A. R., Zuber, M. S., Darrah, L. L., Barry, D., Rabin, L. B., Waiss, A. C., 1984. Evaluation of an antibiotic factor in maize silks as a means of corn earworm (Lepidoptera: Noctuidae) suppression. J. Econ. Entomol. 77:487-490.
174. Heslop-Harrison, J. S., Bennett, M. D., 1983. The positions of centromeres on the somatic metaphase plate of grasses. J. Cell Sci. 64:163-178.
175. Heslop-Harrison, Y., Reger, B. J., Heslop-Harrison, J., 1984. The pollen-stigma interaction in the grasses. 5. Tissue organisation and cytochemistry of the stigma ('silk') of Zea mays L. Acta Bot. Neer. 33:81-99.
176. Heslop-Harrison, Y., Reger, B. J., Heslop-Harrison, J., 1984. The pollen-stigma interaction in the grasses. 6. The stigma ("silk") of Zea mays L. as host to the pollens of Sorghum bicolor (L.) and Pennisetum americanum (L.) Leeke. Acta Bot. Neer. 33:205-227.
177. Hibberd, K. A., 1984. Induction, selection, and characterization of mutants in maize cell cultures. Pp. 571-576 in Cell Culture and Somatic Cell Genetics of Plants, Vol. 1, I. K. Vasil, ed., Academic Press, Orlando.
178. Holden, M. J., Sze, H., 1984. Helminthosporium maydis T toxin increased membrane permeability to $Ca^{2+}$ in susceptible corn mitochondria. Plant Physiol. 75:235-237.
179. Hopkins, W. G., Elfman, B., 1984. Temperature-induced chloroplast ribosome deficiency in virescent maize. J. Hered. 75:207-211.
180. Hunter, B., 1983. Good ears on strong stalks--A challenge for corn breeders. Proc. Annu. Corn and Sorghum Res. Conf. 38:116-127.
181. Iltis, H. H., 1984. The origin of maize (letter). Science 225:1094-1096.
182. Inouchi, N., Glover, D. V., Sugimoto, Y., 1984. Development changes in starch properties of several endosperm mutants of maize. Starke 36:8-12.
183. Inoue, Y., 1984. Specific combining ability between six different types of maize (Zea mays L.) obtained from a diallel set of eleven open-pollinated varieties. Jpn. J. Breed. 34:17-28.
184. Inoue, Y., Okabe, T., 1982. Studies on the breeding method of inbred lines of corn (Zea mays L.). 1. The effect of visual selection in early inbred generations on combining ability. Hokkaido Nat. Agric. Exp. St. Res. Bull. 133:1-6.

185. Inoue, Y., Okabe, T., 1983. Studies on the breeding method of inbred lines of corn (Zea mays L.). 2. Estimating the combining abilities from grain yields and other characteristics of lines per se in early inbred generations. Hokkaido Nat. Agric. Exp. St. Res. Bull. 136:1-14.
186. Ishige, T., Yamada, M., Shiga, T., 1983. Screening for resistance to lodging based upon the discriminant function value in maize and biometrical analysis for genetical components. Bull. Nat. Inst. Agric. Sci. (Japan) Ser. D No. 35:125-152.
187. Ishii, S., Mogi, Y., 1983. Identification of enzymes that are effective for isolating protoplasts from grass leaves. Plant Physiol. 72:641-644.
188. Ivakhnenko, A. N., Dudka, E. L., Zhurba, G. M., Borisov, V. N., 1983. Breeding early maize hybrids for resistance to fusarium rots. Dokl. Vses. Akad. S.-Kh. Nauk 12:15-16.
189. Ivancic, A., 1982. Genetic parameter investigation of the maize leaf area and maize leaf vascular system. Poljopr. Znan. Smotra 59:179-200.
190. Ivanovic, M., Rosic, K., 1983. Partial and semipartial correlations between some traits of the maize stalk (Zea mays L.). Arhiv Poljopr. Nauke 44:173-184.
191. Ivantsov, A. I., Akhmetov, R. R., Shamsnakhmetova, F. Sh., 1983. The reciprocal influence of pure lines and hybrids of maize callus tissues when cultivated together. Fiziol. Rast. (Mosc.) 30:794-796.
192. Jarvis, J. L., Clark, R. L., Guthrie, W. D., Berry, E. C., Russell, W. A., 1984. The relationship between second-generation European corn borers and stalk rot fungi in maize hybrids. Maydica 29:247-263.
193. Jarvis, J. L., Guthrie, W. D., Berry, E. C., 1983. Time and level of infestation by second-generation European corn borers on a resistant and a susceptible maize hybrid in relation to yield losses. Maydica 28:391-400.
194. Jenkins, C. L. D., Russ, V. J., 1984. Large scale, rapid preparation of functional mesophyll chloroplasts from Zea mays and other C4 species. Plant Sci. Lett. 35:19-24.
195. Jensen, S., McConnell, R., Kuhn, W., 1983. Combining ability studies in elite U.S. maize germplasm. Proc. Annu. Corn and Sorghum Res. Conf. 38:87-96.
196. Jones, R. J., Ouattar, S., Crookston, R. K., 1984. Thermal environment during endosperm cell division and grain filling in maize: Effects on kernel growth and development in vitro. Crop Sci. 24:133-136.
197. Jordan, E. G., Perkins, J. M., Schall, R. A., Pedersen, W. L., 1983. Occurrence of race 2 of Exserohilum turcicum on corn (Zea mays) in the central and eastern USA. Plant Dis. 67:1163-1165.
198. Jotshi, P. N., Bhan, M. K., 1983. Metroglyph analysis of morphological variation in maize grown in Jammu. Indian J. Agr. Sci. 53:981-984.
199. Kahler, A. L., Gardner, C. O., Allard, R. W., 1984. Nonrandom mating in experimental populations of maize. Crop Sci. 24:350-354.
200. Kahn, E. J., Jr., 1984. The staffs of life. I. The golden thread. The New Yorker, June 18, pp. 46-88.
201. Kalinnikov, D. D., Kamyshova, T. V., 1982. Amount of zein amino acids and correlations between them. Fiziol. Biokhim. Kult. Rast. 16:27-30.
202. Kalinnikov, D. D., Shevtsova, V. V., 1983. Efficiency of corn selection for increased tryptophan content in zein. S-KH Biol. (3):66-68.
203. Kang, M. S., Zuber, M. S., Krause, G. F., 1983. Path coefficient analyses of grain yield and harvest grain moisture in maize (Zea mays). Trop. Agric. 60:253-256.
204. Kato, T. A., 1984. Chromosome morphology of maize and its races. Pp. 219-254 in Evolutionary Biology, Vol. 17, M. K. Hecht et al., eds., New York, Plenum.
205. Keim, K. R., Gardner, C. O., 1984. Genetic variation for cold tolerance in selected and unselected maize populations. Field Crop. Res. 8:143-152.
206. Kelley, P. M., Freeling, M., 1984. Anaerobic expression of maize fructose-1,6-diphosphate aldolase. J. Biol. Chem. 259:14180-14183.
207. Kelley, P. M., Freeling, M., 1984. Anaerobic expression of maize glucose phosphate isomerase I. J. Biol. Chem. 259:673-677.
208. Kermicle, J. L., 1984. Recombination between components of a mutable gene system in maize. Genetics 107:489-500.
209. Khehra, A. S., Dhillon, B. S., Saxena, V. K., Malhotra, V. V., Malhi, N. S., 1983. Variability in harvest index in winter maize. Natl. Acad. Sci. Lett. 6:177-178.
210. Khehra, A. S., Rao, A. V. B. S., Dey, S. K., Dhillon, B. S., Malhi, N. S., 1984. Inheritance of resistance to maydis leaf-blight in maize. Indian J. Agr. Sci. 54:881-883.
211. Khristov, K., Genov, M., Antonov, M., 1983. Cytogenetic effect of helium neon laser on maize seeds. Genet. Sel. 16:274-281.
212. Khristova, P., Khristov, K., 1984. Mutation breeding in maize. I. Genetic approach in the selection of objects for mutagen treatment and goals of mutation breeding. Genet. Sel. 17:237-244.
213. Kimber, A., Sze, H., 1984. Helminthosporium maydis T toxin decreased calcium transport into mitochondria of susceptible corn. Plant Physiol. 74:804-809.
214. King, P. J., 1984. From single cells to mutant plants. Oxford Surv. Plant Mol. Cell. Biol. 1:7-32.
215. Kirkham, M. B., Suksayretrup, K., Wassom, C. E., Kanemasu, E. T., 1984. Canopy temperature of drought-resistant and drought-sensitive genotypes of maize. Maydica 29:287-303.
216. Kleese, R. A., 1984. Prospects for genetic engineering in corn. Cereal Foods World 29:357-359.
217. Klein, A. S., Nelson, O. E., Jr., 1983. Biochemical consequences of the insertion of a suppressor-mutator (Spm) receptor at the bronze-1 locus in maize. Proc. Nat. Acad. Sci. US-Biol. Sci. 80:7591-7595.
218. Koleva, S., Marinova, E., Marekov, L. N., Beltchev, B., 1984. Isolation and characterization of high mobility group-like chromosomal proteins from meristematic root cells. Biochem. Physiol. Pflanz. 179:181-189.
219. Kondrat'ev, M. N., Kamalova, T. G., 1983. Leaf proteases in plant ontogenesis. Fiziol. Biokhim. Kul't. Rast. 15:107-115.
220. Kono, Y., Suzuki, Y., 1984. The chemistry of host specificity in corn leaf blight. J. Agric. Chem. Soc. Japan 58:1015-1024.
221. Kostyshin, S. S., Masikevich, Y. G., 1984. Pecularities of genome structure in relation to maize heterosis. Tsitol. Genet. 18:25-30. Cytol. Genet. 18:26-31.
222. Kovacevic, V., 1984. The ear-leaf percentage of calcium and magnesium in maize inbred lines and their diallel progeny. Theor. Appl. Genet. 68:521-524.
223. Krebbers, E., Steinmetz, A., Bogorad, L., 1984. DNA sequences for the Zea mays tRNA genes tV-UAC and tS-UGA: tV-UAC contains a large intron. Plant Mol. Biol. 3:13-20.

224. Kridl, J. C., Vieira, J., Rubenstein, I., Messing, J., 1984. Nucleotide sequence analysis of a zein genomic clone with a short open reading frame. Gene 28:113-118.
225. Kruger, G. H. J., 1984. The effect of photoperiod on the initiation of the staminate inflorescence in different hybrids of Zea mays. Ann. Sci. Natur. 5:81.
226. Kuang, V. D., Shamina, Z. B., 1983. Effect of macerating enzymes on corn cell culture. Sov. Plant Physiol. 30:472-478.
227. Kuang, V. D., Shamina, Z. B., Butenko, R. G., 1983. Use of nurse tissue culture to obtain clones from cultured cells and protoplasts of corn. Sov. Plant Physiol. 30:613-620.
228. Kudoyarova, G. R., Erkeev, M. I., 1983. Comparative study of activity of the translation apparatus in leaves of corn hybrids and their parent forms. Sov. Plant Physiol. 30:543-548.
229. Lamkey, K. R., Dudley, J. W., 1984. Mass selection and inbreeding depression in 3 autotetraploid maize synthetics. Crop Sci. 24:802-806.
230. Landry, J., Guyon, P., 1984. Zein of maize grain: I. Isolation by gel filtration and characterization of monomeric and dimeric species. Biochimie 66:451-460.
231. Landry, J., Guyon, P., 1984. Zein of maize grain: II. The charge heterogeneity of free subunits. Biochimie 66:461-470.
232. Landry, J., Moureaux, T., 1984. A quantitative analysis of amino acid accumulation in developing grain of normal and opaque-2 maizes. J. Sci. Food Agr. 35:1054-1062.
233. Langridge, P., Eibel, H., Brown, J. W. S., Feix, G., 1984. Transcription from maize storage protein gene promoters in yeast. EMBO J. 3:2467-2473.
234. Lapina, T. V., Grigorenko, N. V., Morgun, V. V., Logvinenko, V. F., Merezhinsky, Yu. G., 1984. A study of mutagenic effect of herbicides (Treflan, Ramrod and Banvell-D) on maize. Tsitol. Genet. 18:119-122.
235. Larkins, B. A., Pedersen, K., Marks, M. D., Wilson, D. R., 1984. The zein proteins of maize endosperm. Trends Biochem. Sci. 9:306-308.
236. Larkins, B. A., Pedersen, K., Marks, M. D., Wilson, D. R., Argos, P., 1983. Structure and expression of zein genes in maize endosperm. In Structure and Function of Plant Genomes, O. Ciferri and L. Dure, eds. Life Sci. 63:73-83.
237. Laughnan, J. R., 1984. A career in genetics (review of A Feeling for the Organism). Science 223:482-483.
238. Laughnan, J. R., Gabay-Laughnan, S., 1983. Cytoplasmic male sterility in maize. Annu. Rev. Genet. 17:27-48.
239. Leaver, C. J., Dixon, L. K., Hack, E., Fox, T. D., Dawson, A. J., 1983. Mitochondrial genes and their expression in higher plants. In Structure and Function of Plant Genomes, O. Ciferri and L. Dure, eds. Life Sci. 63:347-361.
240. Leaver, C. J., Hack, E., Dawson, A. J., Isaac, P. G., Jones, V. P., 1983. Mitochondrial genes and their expression in higher plants. Pp. 269-284 in Mitochondria 83--Nucleo-Mitochondrial Interactions, R. J. Schweyen et al., eds., Walter de Gruyter, Hawthorne.
241. Leaver, C. J., Hack, E., Dawson, A., Isaac, P., Jones, V. P., 1984. Mitochondrial genes: Structure, mutation and expression. Curr. Topics Plant Biochem. Physiol. 3:123-132.
242. LeDrew, H. D., Daynard, T. B., Muldoon, J. F., 1984. Relationships among hybrid maturity, environment, dry matter yield and moisture content of whole-plant corn. Can. J. Plant Sci. 64:565-574.
243. Lee, L., Tsai, C. Y., 1984. Effect of RNase on zein synthesis in endosperms of brittle-2; opaque-2 maize double mutant. Plant Physiol. 76:79-83.
244. Lee, L., Tsai, C. Y., 1984. Zein synthesis in the embryo and endosperm of maize mutants. Biochem. Genet. 108:729-738.
245. Lee, M. H., Brewbaker, J. L., 1984. Effects of brown midrib-3 on yields and yield components of maize. Crop Sci. 24:105-108.
246. Leto, K. J., 1984. Functional discrimination between Photosystem-II associated chlorophyll a proteins in Zea mays. Biochim. Biophys. Acta 766:98-108.
247. Levic, J., Radovic, G., Pencic, V., 1983. Local maize varieties as a source of resistance to H. turcicum Pass. Arhiv Poljopr. Nauke 44:145-150.
248. Levings, C. S., III, Braun, C. J., Sisco, P. H., 1984. Plasmid-like DNAs of the maize mitochondria. Curr. Topics Plant Biochem. Physiol. 3:119-122.
249. Levings, C. S., III, Sederoff, R. R., Hu, W. W. L., Timothy, D. H., 1983. Relationships among plasmid-like DNAs of the maize mitochondria. In Structure and Function of Plant Genomes, O. Ciferri and L. Dure, eds. Life Sci. 63:363-371.
250. Lillehoj, E. B., Mcmillian, W. W., Widstrom, N. W., Guthrie, W. D., Jarvis, J. L., Barry, D., Kwolek, W. F., 1984. Aflatoxin contamination of maize kernels before harvest. Mycopathologia 86:77-82.
251. Lillehoj, E. B., Zuber, M. S., Darrah, L. L., Kwolek, W. F., Findley, W. R., Horner, E. S., Scott, G. E., Manwiller, A., Sauer, D. B., Thompson, D., Warren, H., West, D. R., Widstrom, N. W., 1983. Aflatoxin occurrence and levels in preharvest corn kernels with varied endosperm characteristics grown at diverse locations. Crop Sci. 23:1181-1184.
252. Lima, M., Miranda Filho, J. B., Boller Gallo, P., 1984. Inbreeding depression in Brazilian populations of maize (Zea mays L.). Maydica 29:203-215
253. Lin, B-Y., 1984. Ploidy barrier to endosperm development in maize. Genetics 107:103-115.
254. Lin, L.-S., Ho, T.-H. D., Harlan, J. R., 1984. Rearrangement of ribosomal RNA genes in tripsacoid maize. Curr. Topics Plant Biochem. Physiol. 3:177.
255. Lonsdale, D. M., 1984. A review of the structure and organization of the mitochondrial genome of higher plants. Plant Mol. Biol. 3:201-206.
256. Lonsdale, D. M., Hodge, T. P., Fauron, C. M. R., Flavell, R. B., 1983. A predicted structure for the mitochondrial genome from the fertile cytoplasm of maize. UCLA Symp. Molec. Cell. Biol. 12:445-456.
257. Lonsdale, D. M., Shardl, C. L., Pring, D. R., 1984. The mitochondrial genome of the S-male sterile cytoplasm of maize: Organization and rearrangements associated with fertility reversion. Curr. Topics Plant Biochem. Physiol. 3:133-140.
258. Lopez-Brana, I., Delibes, A., 1984. Effects of glyphosate on isolated maize mitochondria. J. Exp. Bot. 35:905-911.
259. Loughman, B. C., Roberts, S. C., Goodwin-Bailey, C. I., 1983. Varietal differences in physiological and biochemical responses to changes in the ionic environment. Plant Soil 72:245-260.
260. Ludevid, M. D., Torrent, M., Martinez-Izquierdo, J. A., Puigdomenech, P., Palau, J., 1984. Subcellular localization of glutelin-2 in maize (Zea mays L.) endosperm. Plant Mol. Biol. 3:227-234.
261. Lysikov, V. N., Krivov, N. V., Golubovsky, M. D., 1984. Genetic analysis of the corn-grass macromutation in maize. Genetika SSR 20:90-99; Sov. Genet. 20:72-80.

262. Maguire, M. P., 1983. Homologue pairing and synaptic behavior at zygotene in maize. Cytologia 48:811-818.
263. Maguire, M. P., 1984. The mechanism of meiotic homologue pairing. J. Theor. Biol. 106:605-615.
264. Maguire, M. P., 1984. The pattern of pairing that is effective for crossing over in complex B-A chromosome rearrangements in maize. Chromosoma 89:18-23.
265. Mahey, R. K., Sawhney, J. S., Narang, R. S., 1982. Maturity indexing in maize (Zea mays L.). Indian J. Plant Physiol. 25:237-240.
266. Mandoli, D. F., Briggs, W. R., 1984. Fiber optic plant tissues: Spectral dependence in dark-grown and green tissues. Photochem. Photobiol. 39:419-424.
267. Mandoli, D. F., Tepperman, J., Huala, E., Briggs, W. R., 1984. Photobiology of diagravitropic maize roots. Plant Physiol. 75:359-363.
268. Mangelsdorf, P. C., 1984. The origin of maize (letter). Science 225:1094.
269. Markov, E. Y., Khavkin, E. E., 1982. Tissue specific differences in lectin erythrocyte binding and erythro-agglutinating capacities in the tissues of maize seedlings. Izv. Sib. Otd. Akad. Nauk SSR S. Biol. Nauk (3):35-39.
270. Markova, M. D., 1983. Isoenzyme composition of cytochromoxidase in cytoplasmic male-sterile lines of maize. Dokl. Bolg. Akad. Nauk 36:1101-1104.
271. Marks, M. D., Pedersen, K., Wilson, D. R., 1983. Molecular structure and expression of maize zein genes. Pp. 369-382 in Advances in Gene Technology: Molecular Genetics of Plants and Animals, K. Downey et al., eds., Academic Press, Orlando.
272. Marks, M. D., Pedersen, K., Wilson, D. R., DiFonzo, N., Larkins, B. A., 1984. Molecular biology of the maize seed storage proteins. Curr. Topics Plant Biochem. Physiol. 3:9-18.
273. Martin, M. J., Russell, W. A., 1984. Correlated responses of yield and other agronomic traits to recurrent selection for stalk quality in a maize synthetic. Crop Sci. 24:746-750.
274. Martin, M. J., Russell, W. A., 1984. Response of a maize synthetic to recurrent selection for stalk quality. Crop Sci. 24:331-337.
275. Martinez Izquierdo, J. A., Ludevid, M. D., Puigdomenech, P., Palau, J., 1984. Two-dimensional gel electrophoresis of zein proteins from normal and opaque-2 maize with non-ionic detergent acid urea-polyacrylamide gel electrophoresis in the first dimension. Plant Sci. Lett. 34:43-50.
276. Martiniello, P., 1984. Outline of the major components of maize breeding programs for semi-arid regions, Capitanata Plain Southern Italy. Genet. Agrar. 37:361-390.
277. Marwaha, K. K., Siddiqui, K. H., Panwar, V. P. S., Sarup, P., 1983. Evaluation of maize germplasms comprising different maturity groups for resistance to the stalk borer Chilo partellus under artificial infestation. J. Entomol. Res. (New Delhi) 7:99-106.
278. Maryam, B., Jones, D. A., 1983. The genetics of maize (Zea mays L.) growing at low temperatures. II. Germination of inbred lines, F-1 and further generations at fluctuating temperatures. Euphytica 32:791-798.
279. Mascarenhas, N. T., Bashe, D., Eisenberg, A., Willing, R. P., Xiao, C.-M., Mascarenhas, J. P., 1984. Messenger RNAs in corn pollen and protein synthesis during germination and pollen tube growth. Theor. Appl. Genet. 68:323-326.
280. Masnica, M., 1984. The importance of maize grain specific weight as a yield character. Rostl. Vyroba 30:359-368.
281. Mason, J. R., Dolbeer, R. A., Arzt, A. H., Reidinger, R. F., Woronecki, P. P., 1984. Taste preferences of male red-winged blackbirds among dried samples of ten corn hybrids. J. Wildlife Manage. 48:611-615.
282. Mateyka, C., Schnarrenberger, C., 1984. Starch phosphorylase isoenzymes in mesophyll and bundle sheath cells of corn leaves. Plant Sci. Lett. 36:119-123.
283. Matzke, M. A., Susani, M., Binns, A. N., Lewis, E. D., Rubenstein, I., Matzke, A. J. M., 1984. Transcription of a zein gene introduced into sunflower using a Ti plasmid vector. EMBO J. 3:1529-1532.
284. Mayfield, S. P., Taylor, W. C., 1984. Carotenoid-deficient maize seedlings fail to accumulate light-harvesting chlorophyll a/b binding protein (LHCP) mRna. Eur. J. Biochem. 144:79-84.
285. Mayfield, S. P., Taylor, W. C., 1984. The appearance of photosynthetic proteins in developing maize leaves. Planta 161:481-486.
286. McNay, J. W., Chourey, P. S., Pring, D. R., 1984. Molecular analysis of genomic stability of mitochondrial DNA in tissue cultured cells of maize. Theor. Appl. Genet. 67:433-437.
287. McRae, K. B., White, R. P., 1983. A multivariate approach for the selection of cultivars with antagonistic traits. Can. J. Plant Sci. 63:861-878.
288. Meghji, M. R., Dudley, J. W., Lambert, R. J., Sprague, G. F., 1984. Inbreeding depression, inbred and hybrid grain yields, and other traits of maize genotypes representing 3 eras. Crop Sci. 24:545-548.
289. Messing, J., 1983. The manipulation of zein genes to improve the nutritional value of corn. Trends Biotechnol. 1:54-59.
290. Messing, J., 1984. Data storage and handling of plant nucleotide sequences. Plant Mol. Biol. Reporter 2:32-35.
291. Messing, J., Carlson, J., Hagen, G., Rubenstein, I., Oleson, A., 1984. Cloning and sequencing of the ribosomal RNA genes in maize: The 17S region. DNA 3:31-40.
292. Messmer, M. J., Lambert, R. J., Hageman, R. H., 1984. Classification of certain N-traits as criteria for the identification of productive maize genotypes. Crop Sci. 24:605-610.
293. Mesterhazy, A., 1983. Relationship between resistance to stalk rot and ear rot of corn influenced by rind resistance, premature death and the rate of drying of the ear. Maydica 28:425-438.
294. Metz, J. G., Krueger, R. W., Miles, D., 1984. Chlorophyll-protein complexes of a photosystem II mutant of maize. Evidence that chlorophyll-protein a-2 and a chlorophyll-protein complex derived from a photosystem I antennae system comigrate on polyacrylamide gels. Plant Physiol. 75:238-241.
295. Metz, J. G., Ulmer, G., Bricker, T. M., Miles, D., 1983. Purification of cytochrome b-559 from oxygen-evolving photosystem II preparations of spinach and maize. Biochim. Biophys. Acta 725:203-209.
296. Micke, A., 1983. International research programs for the genetic improvement of grain proteins. Pp. 25-44 in Advances in Agricultural Biotechnology: Seed Proteins: Biochemistry, Genetics, Nutritive Value, W. Gottschalk and H. P. Mueller, eds., Martinus Nijhoff/Dr. W. Junk, The Hague.
297. Miflin, B. J., Forde, B. G., Shewry, P. R., Kreis, M., Forde, J., 1984. Repeated sequences in cereal storage proteins. Oxford Surv. Plant Mol. Cell Biol. 1:231-234.
298. Mikel, M. A., D'Arcy, C. J., Rhodes, A. M., Carey, E. E., Juvik, J. A., 1983. Sugary su sweet corn (Zea mays) germplasm with resistance to the maize dwarf mosaic virus. Hortscience 18:964-965.
299. Mikel, M. A., D'Arcy, C. J., Rhodes, A. M., Ford, R. E., 1984. Genetics of resistance of two dent corn inbreds to maize dwarf mosaic virus and transfer of resistance into sweet corn. Phytopathology 74:467-473.

300. Mikel, M. A., Darcy, C. J., Ford, R. E., 1984. Seed transmission of maize dwarf mosaic virus in sweet corn. Phytopathol. Z.-J. Phytopathol. 110:185-191.
301. Milinko, I., Peti, J., Jozsa, S., Kobza, S., 1984. Maize dwarf virus resistance ratings of corn hybrids. Novenytermeles 33:147-156.
302. Miranda Filho, J. B., Vencovski, R., 1984. Analysis of diallel crosses among open-pollinated varieties of maize (Zea mays L.). Maydica 29:217-234.
303. Misharin, S. I., Mladenova, I., Mozgova, E. A., Sukhorzhevskaya, T. B., Khavkin, E. E., 1983. Antigen and isoenzyme spectra of maize and teosinte. Dokl. Biol. Sci. 265:379-382.
304. Mitchell, J. G., 1984. Where corn is king. Smithsonian 15:34-50.
305. Molina, M. del C., 1983. Cytogenetic studies on Zea diploperennis. Nucleus 26:1-7.
306. Moll, R. H., Hanson, W. D., 1984. Comparisons of effects of intrapopulation vs. interpopulation selection in maize. Crop Sci. 24:1047-1052.
307. Moller, G., Geisler, G., Stamp, P., 1984. A serial assay procedure for simultaneous spectrophotometric tests of 4 photosynthetic enzymes--Adaptation of maize inbred lines and crosses to extreme temperatures for active growth. Z. Acker- Pflanzenbau 153:167-175.
308. Montanelli, C., Difonzo, N., Marotta, R., Motto, M., Soave, C., Salamini, F., 1984. Occurrence and behavior of the components of the O2-M(R)-Bg system of maize controlling elements. Mol. Gen. Genet. 197:209-218.
309. Moore, R., Smith, J. D., 1984. Growth, graviresponsiveness and abscisic-acid content of Zea mays seedlings treated with Fluridone. Planta 342-344.
310. Mosely, P. R., Crosbie, T. M., Mock, J. J., 1984. Mass selection for improved cold and density tolerance of two maize populations. Euphytica 33:263-269.
311. Mottinger, J. P., Dellaporta, S. L., Keller, P. B., 1984. Stable and unstable mutations in Aberrant Ratio stocks of maize. Genetics 106:751-767.
312. Mottinger, J. P., Johns, M. A., Freeling, M., 1984. Mutations of the Adh1 gene in maize following infection with barley stripe mosaic virus. Mol. Gen. Genet. 195:367-369.
313. Muchena, S. C., Contreras, M. R., Galt, D. L., Peairs, F. B., 1983. Progress from recurrent full-sib family selection for potential yield improvement in two tropical maize (Zea mays L.) populations. Field Crops Res. 7:283-298.
314. Muchena, S. C., Schweppenhauser, M. A., Mashingaidze, K., 1983. Gene complementation in seed coloration of maize (Zea mays L.) hybrids of inbreds extracted from Zimbabwean and exotic open-pollinated varieties. Zimbabwe J. Agr. Res. 21:167-169.
315. Muldoon, J. F., Daynard, T. B., Vanduinen, B., Tollenaar, M., 1984. Comparisons among rates of appearance of leaf tips, collars, and leaf area in maize (Zea mays L.). Maydica 29:109-120.
316. Muldoon, J. F., Leask, W. C., Daynard, T. B., Zuber, M. S., 1984. Potential use of stalk pH and stalk percent dry matter as estimators of lodging susceptibility in corn. Can. J. Plant Sci. 64:559-564.
317. Muller-Neumann, M., Yoder, J., Starlinger, P., 1984. The DNA sequence of the transposable element Ac of Zea mays L. Mol. Gen. Genet. 198:19-24.
318. Munoz-Orozco, A., Stevenson, K. R., Ortiz-Cereceres, J., Thurtell, G. W., Carballo-Carballo, A., 1983. Transpiration photosynthesis effectiveness in the use of water and hydric potential in maize strains resistant to dry soil and freezing. Agrociencia 51:115-154.
319. Nagy, J., 1984. Yield response to fertilization in maize hybrids on calcareous chernozem soil. Novenytermeles 33:253-264.
320. Nakagawa, H., Poulle, M., Oaks, A., 1984. Characterization of nitrate reductase from corn leaves (Zea mays cv W64A x W182E). Two molecular forms of the enzyme. Plant Physiol. 75:285-289.
321. Naumenko, A. I., Kirpa, N. Y., Zolotov, V. I., Ponomarenko, A. K., 1983. Moisture content and quality of seed in maize hybrids of various earliness. S-KH. Biol. 10:40-43.
322. Nelson, O. E., Klein, A. S., 1984. Characterization of an Spm-controlled bronze-mutable allele in maize. Genetics 106:769-779.
323. Nelson, T., Harpster, M. H., Mayfield, S. P., Taylor, W. C., 1984. Light-regulated gene expression during maize leaf development. J. Cell Biol. 98:558-564.
324. Nesticky, M., Novak, F. J., Piovarci, A., Dolezelova, M., 1983. Genetic analysis of callus growth of maize (Zea mays L.) invitro. Z. Pflanzenzucht. 91:322-328.
325. Nesticky, M., Piovarci, A., 1984. Influence of aleurone thickness on lysine content of maize kernels. Z. Pflanzenzucht. 92:173-176.
326. Neudachin, V. P., Lebedev, A. V., 1984. Fractional protein composition and isoelectric patterns of zein from endosperm mutants of maize. Prikl. Biokhim. Mikrobiol. 20:267-272.
327. Newton, K. J., 1983. Genetics of mitochondrial isozymes. Pp. 157-174 in Developments in Plant Genetics and Breeding, Vol. I, Pt. A., S. D. Tanksley and T. J. Orton, Eds., Elsevier, Amsterdam.
328. Newton, K. J., 1984. Approaches to studying the unique features of plant mitochondrial gene expression. Curr. Topics Plant Biochem Physiol. 3:141-147.
329. Nirmala, A., Rao, P. N., 1984. Chromosome instability in Coix gigantea Koen. (Maydeae). Can. J. Genet. Cytol. 26:334-338.
330. Novak, F. J., Dolezelova, M., Nesticky, M., Piovarci, A., 1983. Somatic embryogenesis and plant regeneration in Zea mays. Maydica 28:381-390.
331. Ohmasa, M., 1984. Relation between cytoplasmic male sterility and mitochondrial enzyme activities in maize. JARQ 18:12-19.
332. Oliveira, E. L., Muzilli, O., Gerage, A. C., Cataneo, A., 1983. Maize cultivars tolerance to soil acidity and efficiency to liming response. Pesq. Agrop. Brasil. 18:1045-1052.
333. Onukogu, F. A., 1984. Oviposition behavior, biology, and host plants resistance studies of the West African maize borer, Sesamia calamistis. Maydica 29:121-132.
334. Ottaviano, E., Landi, P., Villa, M., 1984. Use of reciprocal full-sib selection to develop maize (Zea mays L.) hybrids. Maydica 29:161-174.
335. Palmer, J. D., Osorio, B., Watson, J. C., Edwards, H., Dodd, J., Thompson, W. F., 1984. Evolutionary aspects of chloroplast genome expression and organization. Pp. 273-284 in Biosynthesis of the Photosynthetic Apparatus: Molecular Biology, Development and Regulation, J. P. Thornber et al., eds., Alan R. Liss, New York.
336. Paly, A. F., Rotar, A. I., 1982. Effect of gene modifiers on the physical and biochemical properties of grain of opaque maize. Izv. Akad. Nauk Mold. SSR Ser. Biol. Khim. Nauk (14):36-39.
337. Pan, D., Nelson, O. E., Jr., 1984. A debranching enzyme deficiency in endosperms of the sugary-1 mutants of maize. Plant Physiol. 74:324-328.

338. Pan, S., 1984. Studies of salt tolerance in corn, Zea mays L. 1. Screening for salt tolerance line and its acid phosphatase determination. J. Agr. Assn. China-Taiwan 127:58-67.
339. Panwar, V. P. S., Marwaha, K. K., Siddiqui, K. H., 1982. Response of introduction nursery comprising exotic and indigenous maize germplasms to the stalk borer Chilo partellus under artificial infestation. J. Entomol. Res. 6:202-205.
340. Pasztor, K., Pepo, P., Egri, K., 1984. Combining ability and growth analysis of maize hybrids. Novenytermeles 33:193-202.
341. Paulsen, M. R., Hill, L. D., White, D. G., Sprague, G. F., 1983. Breakage susceptibility of Corn-Belt genotypes. Trans. ASAE 26:1830-1836.
342. Peacock, W. J., Dennis, E. S., Gerlach, W. L., Llewellyn, D., Lorz, H., Pryor, A. J., Sachs, M. M., Schwartz, D., Sutton, W. D., 1983. Gene transfer in maize--Controlling elements and the alcohol dehydrogenase genes. Pp. 311-323 in Advances in Gene Technology: Molecular Genetics of Plants and Animals, K. Downey et al., eds., Academic Press, Orlando.
343. Pego, S. E., Hallauer, A. R., 1984. Portuguese maize germplasm with abnormal ear shape. Maydica 29:39-53.
344. Pernes, J., 1983. La genetique de la domestication des cereales. La Recherche 14:910-919.
345. Perrino, P., Hammer, K., 1984. Collection of land races of cultivated plants in south Italy, 1982. Kulturpflanze 31:219-226.
346. Phillips, R. L., Wang, A. S., 1984. Chromosome analysis. Pp. 712-727 in Cell Culture and Somatic Cell Genetics of Plants, Vol. I, I. K. Vasil, ed., Academic Press, Orlando.
347. Phinney, B. O., 1984. Gibberellin $A_1$, dwarfism and the control of shoot elongation in higher plants. Pp. 17-45 in The Biosynthesis & Metabolism of Plant Hormones (S.E.B. Seminar Series 23), Oxford Univ. Press.
348. Phinney, B. O., Spray, C., 1983. Gibberellin biosynthesis in Zea mays: The 3-hydroxylation step
349. Phipps, R. H., McAllan, A. B., 1984. Carbohydrate constituents and amino acid composition of maize silage grown on commercial farms in the UK. Maydica 29:27-38.
350. Piche, Y., Peterson, R. L., Ackerley, C. A., Rauser, W. E., 1984. RNase-gold labelling in primary roots of Zea mays L.: Evaluation of a particulate marker. Plant Sci. Lett. 36:155-160.
351. Pierson, E. E., Clark, R. B., Maranville, J. W., Coyne, D. P., 1984. Plant genotype differences to ferrous and total iron in emerging leaves. I. Sorghum and maize. J. Plant Nutr. 7:371-387.
352. Pietrzak, M., Wojtowicz, B., Cudny, H., 1983. A new method of purification of ribonuclease from maize seeds based on affinity chromatography. Pr. Nauk. Uniw. Slask. Katowicach 541:19-24.
353. Pilet, P.-E., Blaschek, W., Senn, A., Franz, G., 1984. Comparison between maize root cells and their respective regenerating protoplasts: Wall polysaccharides. Planta 161:465-469.
354. Pilinskaya, M. A., Shigaeva, M. Kh., Kasinova, G. V., Malashenko, A. M., Vyskubenko, I. F., 1984. Concerning the mutagenic activity of Captan (a fungicide). Tsitol. Genet. 18:123-128.
355. Pinter, L., 1983. Investigation of the standing ability of silage maize. Cereal Res. Commun. 11:245-251.
356. Pinter, L., 1984. Effect of genetic contamination on maize yield (Zea mays L.). Z. Pflanzenzucht. 93:101-105.
357. Pjon, C. J., 1984. Temperature-dependent inhibitive actions of alpha-alpha'-dipyridyl and cycloheximide on the senescence of maize leaves. Plant Cell Physiol. 25:1103-1106.
358. Plewa, M. J., 1984. Review of Plant Systems and Mutation Research. Environ. Mutagen. 6:117-118.
359. Plewa, M. J., Wagner, E. D., Gentile, G. J., Gentile, J. M., 1984. An evaluation of the genotoxic properties of herbicides following plant and animal activation. Mut. Res. 136:233-246.
360. Plotnikov, V. K., Filichkin, S. A., Ryadchikov, V. G., Neudachin, V. P., Dolgikh, Y. R., 1983. Distribution of polyadenylated RNA of membrane-bound polyribosomes in subcellular preparations of maturing maize endosperm. S-KH. Biol. 10:120-125.
361. Podleckis, E. V., Curtis, C. R., Heggestad, H. E., 1984. Peroxidase enzyme markers for ozone sensitivity in sweet corn. Phytopathology 74:572-577.
362. Podolskaya, A. P., 1984. Study of genetics of selective fertilization in annual teosinte. Genetika 20:808-816.
363. Poethig, R. S., 1984. Cellular parameters of leaf morphogenesis in maize and tobacco. Pp. 235-260 in Contemporary Problems in Plant Anatomy, R. A. White and W. C. Dickison, eds., Academic Press, Orlando.
364. Pohlman, P. F., Fedoroff, N. V., Messing, J., 1984. Correction: Nucleotide sequence of Ac. Cell 39:417.
365. Pohlman, R. F., Fedoroff, N. V., Messing, J., 1984. The nucleotide sequence of the maize controlling element Activator. Cell 37:635-643.
366. Pokhmelnykh, G. A., Shumny, V. K., 1984. On the nature of heterochromatic knob regions in maize chromosomes. II. The nature of knob changes during plant inbreeding. Genetika 20:1649-1662.
367. Polacco, M. L., 1984. Chl (A/B) light harvesting complex assembly in maize: Genetic evidence that it may compete with PSII for chl. Curr. Topics Plant Biochem. Physiol. 3:167.
368. Pollak, L. M., Gardner, C. O., Kahler, A. L., Thomascompton, M., 1984. Further analysis of the mating system in 2 mass selected populations of maize. Crop Sci. 24:793-796.
369. Pollak, L. M., Gardner, C. O., Parkhurst, A. M., 1984. Relationships between enzyme marker loci and morphological traits in two mass selected maize populations. Crop Sci. 24:1174-1179.
370. Polowick, P. L., Greyson, R. I., 1984. The relative efficiency of cytokinins in the development of normal spikelets on cultured tassels of Zea mays. Can. J. Bot. 62:830-834.
371. Pomeranz, Y., Martin, C. R., Traylor, D. D., Lai, F. S., 1984. Corn hardness determination. Cereal Chem. 61:147-150.
372. Poneleit, C., 1983. Selection for grain filling period in maize. Proc. Annu. Corn and Sorghum Res. Conf. 38:53-65.
373. Pueppke, S. G., Hymowitz, T., 1984. Genetically uniform seeds: Important starting materials for molecular studies. Plant Mol. Biol. Rep. 2:1-7.
374. Rak-Soltes, E., Tucic, B., 1983. Relationship between genetic distance of maize inbred lines and hybrid performance in their crosses. Arhiv Poljopr. Nauke 44:267-278.
375. Ramesh, S. V., Kumari, A. S., Reddy, A. R., 1984. Photoacoustic and physiological studies on a maize mutant: A delay in leaf pigment synthesis in virescent seedlings. Biochem. Int. 9:121-128.
376. Rapela, M. A., 1984. El cultivo in vitro de vegetales y su utilidad para el mejoramiento en plantas superiores. Bol. Asoc. Cienc. Nat. Litoral 4:8-13.
377. Rapela, M. A., Herkovits, J., 1984. Microscopia electronica de barrido en cultivos in vitro de embriones inmaduros y mesocotilos de plantulas de maiz. Rev. Fac. Agronomia U.B.A. 5:95-105.
378. Rascio, N., Mariani, P., Casadoro, G., 1984. Etioplast-chloroplast transformation in maize leaves: Effects of tissue age and light intensity. Protoplasma 119:110-120.

379. Reddy, L. V., Peterson, P. A., 1984. Enhancer transposable element induced changes at the A-locus in maize--The a-m-16078 allele. Mol. Gen. Genet. 194:124-137.
380. Reddy, V. M., Daynard, T. B., 1983. Endosperm characteristics associated with rate of grain filling and kernel size in corn. Maydica 28:339-356.
381. Rhoades, M. M., 1984. The early years of maize genetics. Ann. Rev. Genet. 18:1-29.
382. Rhodes, L. L., Eagles, H. A., 1984. Origins of maize in New Zealand. N. Z. J. Agr. Res. 27:151-156.
383. Riley, G. J. P., 1984. Effects of high temperature on RNA synthesis during germination of maize (Zea mays L.). Plant Sci. Lett. 201-205.
384. Robbins, W. A., Jr., Ashman, R. B., 1984. Parent-offspring popping expansion correlations in progeny of dent corn x popcorn and flint corn x popcorn crosses. Crop Sci. 24:119-121.
385. Roberts, J. K. M., Callis, J., Jardetzky, O., Walbot, V., Freeling, M., 1984. Cytoplasmic acidosis as a determinant of flooding intolerance in plants. Proc. Nat. Acad. Sci. U.S. 81:6029-6033.
386. Roberts, J. K. M., Callis, J., Wemmer, D., Walbot, V., Jardetzky, O., 1984. Mechanism of cytoplasmic pH regulation in hypoxic maize root tips and its role in survival under hypoxia. Proc. Nat. Acad. Sci. USA--Biol. Sci. 81:3379-3383.
387. Robertson, D. S., 1984. A study of heterofertilization in diverse lines of maize. J. Hered. 75:457-462.
388. Robertson, D. S., 1984. Different frequency in the recovery of crossover products from male and female gametes of plants hypoploid for B-A translocations in maize. Genetics 107:117-130.
389. Rojanaridpiched, C., Gracen, V. E., Everett, H. L., Coors, J. G., Pugh, B. F., Bouthyette, P., 1984. Multiple factor resistance in maize to European corn borer. Maydica 29:305-315.
390. Romheld, V., Marschner, H., 1984. Plant-induced pH changes in the rhizosphere of "Fe-efficient" and "Fe-inefficient" soybean and corn cultivars. J. Plant Nutr. 7:623-630.
391. Rosenkranz, E., Scott, G. E., 1984. Determination of the number of genes for resistance to maize dwarf mosaic virus strain A in five corn inbred lines. Phytopathology 74:71-76.
392. Russell, W. A., 1984. Dedication--Sprague, George F. Corn breeder and geneticist. Pp. 1-12 in Plant Breeding Reviews, Vol. 2, J. Janick, ed., AVI Publ. Co., Westport.
393. Russell, W. A., 1984. Further studies on the response of maize inbred lines to N-fertilizer. Maydica 29:141-150.
394. Russell, W. K., Stuber, C. W., 1984. Prediction of tassel initiation in corn. Crop Sci. 24:507-510.
395. Rymzhanova, R. S., Mynbaev, T. T., 1982. Features of interaction between mutant genes regulating maize low growth. Izv. Akad. Nauk Kaz. SSR Ser. Biol. (3):15-19.
396. Saccomani, M., Cacco, G., Ferrari, G., 1984. Changes in the kinetic parameters of sulfate uptake in maize hybrids during the selection period 1930 through 1975. Maydica 29:133-140.
397. Sack, F. D., Suyemoto, M. M., Leopold, A. C., 1984. Kinetics of amyloplast sedimentation in gravistimulated maize coleoptiles. Planta 161:459-464.
398. Sanchez de Jimenez, E., Aguilar, R, 1984. Protein synthesis patterns. Relevance of old and new messenger RNA in germinating maize embryos. Plant Physiol. 75:231-234.
399. Sanchez de Jimenez, E., Yanez, L., Vargas, M., 1983. Establishing a cellular line of maize (Zea mays) in cultivation in vitro: The capacity for redifferentiation. Agrociencia 52:101-113.
400. Sanford, J. C., Chyi, Y. S., Reisch, B. I., 1984. Attempted "egg transformation" in Zea mays L., using irradiated pollen. Theor. Appl. Genet. 68:269-275.
401. Santos, J. P., Foster, J. E., 1983. Weevil resistance mechanisms in corn kernels. Pesq. Agrop. Brasil. 18:1059-1064.
402. Santos, M. A., Torne, J. M., Blanco, J. L., 1984. Methods of obtaining maize totipotent tissues. I. Seedling segments culture. Plant Sci. Lett. 33:309-316.
403. Sapre, A. B., Barve, S. S., 1983. Somatic chromosomes from intercalary meristems of grasses. Cytologia 48:539-542.
404. Sarma, J. S. P., Sharma, A. K., 1984. Amount of DNA in different strains of maize and its importance in selection. Proc. Indian Natl. Sci. Acad. 50:107-112.
405. Sarup, P., Marwaha, K. K., Panwar, V. P. S., Siddiqui, K. H., 1983. Response of experimental maize germplasms evaluated under different stages of testing and maturity groups to factitious release of egg masses of Chilo partellus. J. Entomol. Res. (New Delhi) 7:43-51.
406. Sarup, P., Marwaha, K. K., Siddiqui, K. H., 1984. Response of maize germplasms to the stalk borer Chilo-partellus under rainfed conditions. J. Entomol. Res. (New Delhi) 7:196-198.
407. Scandalios, J. G., Tsaftaris, A. S., Chandlee, J. M., Skadsen, R. W., 1984. Expression of the developmentally regulated catalase (Cat) genes in maize. Develop. Genetics 4:281-293.
408. Schardl, C. L., Lonsdale, D. M., Pring, D. R., Rose, K. R., 1984. Linearization of maize mitochondrial chromosomes by recombination with linear episomes. Nature 310:292-296.
409. Schliesing, T. G., Dahl, B. E., 1983. Ecotypic variation in Tripsacum dactyloides evaluated in Texas. J. Range Manage. 36:665-667.
410. Schmidt, W., 1984. Red/far-red photoreversibility: not an appropriate phytochrome assay for red-light preirradiated corn coleoptiles. Photochem. Photobiol. 39:267-270.
411. Schuster, A. M., Sisco, P. H., Levings, C. S., 1983. Two unique RNAs in Cms-S and RU maize mitochondria. UCLA Symp. Molec. Cell. Biol. 12:437-444.
412. Schuster, M. L., Smith, C. C., Smith, D. J., 1983. Population trends of epiphytic Corynebacterium nebraskense on leaves of popcorn (Zea mays var. everta) genotypes. Fitopatol. Bras. 8:237-242.
413. Schwartz, D., 1984. Analysis of the Ac transposable element dosage effect in maize. Mol. Gen. Genet. 196:81-84.
414. Schwarz-Sommer, Zs., Gierl, A., Klosgen, R. B., Wienand, U., Peterson, P. A., Saedler, H., 1984. The Spm (En) transposable element controls the excision of a 2-kb DNA insert at the wx-m-8 allele of Zea mays. EMBO J. 3:1021-1028.
415. Scott, G. E., King, S. B., 1984. Sample size to detect genotypic differences in maize to kernel infection by Fusarium moniliforme. Maydica 29:151-160.
416. Scott, G. E., King, S. B., 1984. Site of action of factors for resistance to Fusarium moniliforme in maize. Plant Dis. 68:804-806.
417. Scott, G. E., King, S. B., Armour, J. W., 1984. Inheritance of resistance to Southern Corn Rust in maize populations. Crop Sci. 24:265-267.
418. Scowcroft, W. R., Larkin, P. J., Brettell, R. I. S., 1983. Genetic variation from tissue culture. Pp. 139-162 in Use of Tissue Culture and Protoplasts in Plant Pathology, J. P. Helgeson and B. J. Deverall, eds., Academic Press, Sydney, Australia.

419. Sebestyen, E., 1984. Investigations into the nutrient content of inbred maize lines and their hybrid. Novenytermeles 33:227-234.
420. Sederoff, R. R., 1984. Structural variation in mitochondrial DNA. Advan. Gen. 22:1-108.
421. Shah, D. M., Hightower, R. C., Meagher, R. B., 1983. Genes encoding actin in higher plants: Intron positions are highly conserved but the coding sequences are not. J. Mol. Appl. Genet. 2:111-126.
422. Shannon, J. C., Garwood, D. L., 1984. Genetics and physiology of starch development. Pp. 25-86 in Starch, 2nd Edition, R. L. Whistler et al., eds., Academic Press, Orlando.
423. Sharma, S. R., Khehra, A. S., Dhillon, B. S., Malhotra, V. V., 1982. Evaluation of S-1 lines of maize (Zea mays) crossed in a diallel system. Crop Improv. 9:42-47.
424. Sharma, T. R., Adamu, I. M., 1984. The effects of plant population on the yield and yield attributing characters in maize (Zea mays L.). Z. Acker- Pflanzenbau 153:315-
425. Shaver, D., 1983. Genetics and breeding of maize with extra leaves above the ear. Proc. Annu. Corn and Sorghum Res. Conf. 38:161-180.
426. Shaw, J. R., Dickinson, D. B., 1984. Studies of sugars and sorbitol in developing corn kernels. Plant Physiol. 75:207-211.
427. Shepherd, N. S., Schwarz-Sommer, Z., Blumberg vel Spalve, J., Gupta, M., Wienand, U., Saedler, H., 1984. Similarity of the Cin1 repetitive family of Zea mays to eukaryotic transposable elements. Nature 307:185-186.
428. Sherchan, K., Ellis, R., Jr., Whitney, D. A., Wassom, C. E., 1983. Response of several genetic sources of corn to acidic soil. Agron. J. 75:993-996.
429. Shields, C. R., Orton, T. J., Stuber, C. W., 1983. An outline of general resource needs and procedures for the electrophoretic separation of active enzymes from plant tissue. Pp. 443-468 in Developments in Plant Genetics and Breeding, Vol. 1, Pt. A., S. D. Tanksley and T. J. Orton, Eds., Elsevier, Amsterdam.
430. Shimamoto, K., Ackermann, M., Dierks-Ventling, C., 1983. Expression of zein in long term endosperm cultures of maize. Plant Physiol. 73:915-920.
431. Sidorov, A. N., 1983. Selection of maize cultivars. Izv. Sib. Otd. Akad. Nauk SSSR Ser. Biol. Nauk (2):47-50.
432. Silva, J., Hallauer, A. R., 1984. Factors affecting production of corn forage. Iowa Acad. Sci. 91:76-81.
433. Singh, T., Bains, G. S., 1984. Malting of corn: Effect of variety, germination, gibberellic acid, and alkali pretreatments. J. Agr. Food Chem. 32:346-348.
434. Sisco, P. H., Garcia-Arenal, F., Zaitlin, M., Earle, E. D., Gracen, V. E., 1984. LBN, a male-sterile cytoplasm of maize, contains two double-stranded RNAs. Plant Sci. Lett. 34:127-134.
435. Skretkowicz, A. L., Thurtell, G. W., 1983. Comparative water stress studies on drought resistant and susceptible corn grown in chamber and field environments. Can. J. Plant Sci. 63:775-788.
436. Slvolpa, Y. M., Toptikov, V. A., 1983. Effective in vivo labeling of plant DNA during inhibition of endogenous synthesis by thymidylate. Sov. Plant Physiol. 30:342-346.
437. Smith, J. A., Green, C. E., Gengenbach, B. G., 1984. Feeder layer support of low density populations of Zea mays L. suspension cells. Plant. Sci. Lett. 67-72.
438. Smith, J. S. C., 1984. Isozyme electromorphs in developing embryos of maize (Zea mays L.) Maydica 29:175-184.
439. Smith, J. S. C., Goodman, M. M., Stuber, C. W., 1984. Variation within teosinte. 3. Numerical analysis of allozyme data. Econ. Bot. 38:97-113.
440. Smith, O. S., 1984. Comparison of effects of reciprocal recurrent selection in the BSSS(R), BSCB1(R) and BS6 populations. Maydica 29:1-8.
441. Soave, C., Salamini, F., 1984. Organization and regulation of zein genes in maize endosperm. Phil. Trans. Roy. Soc. London B 304:341-347.
442. Soave, C., Salamini, F., 1984. The role of structural and regulatory genes in the development of maize endosperm. Develop. Genetics 5:1-26.
443. Song, Y.-C., Liu, L.-H., 1983. Comparison between C-banding patterns on root tip chromosomes of different cultivars in maize (Zea mays). Acta Bot. Sin. 25:40-45.
444. Sorenson, J. C., 1984. The structure and expression of nuclear genes in higher plants. Advan. Gen. 22:109-144.
445. Sprague, E. W., Paliwal, R. L., 1984. CIMMYT's maize improvement programme. Outlook Agr. 13:24-31.
446. Spray, C., Phinney, B. O., Gaskin, P., Gilmour, S. J., MacMillan, J., 1984. Internode length in Zea mays L. The dwarf-1 mutation controls the 3-beta-hydroxylation of gibberellin $A_{20}$ to gibberellin $A_1$. Planta 160:464-468.
447. Stamp, P., 1984. Emergence and seedling growth of tropical highland maize and its top crosses with Middle European hybrids. Z. Acker- Pflanzenbau 153:116-121.
448. Stamp, P., Kullman, A., 1984. Shoot growth, green leaf area development and NAR of maize. Comparison between cultivars grown in Germany 1875 and 1976. Maydica 29:235-246.
449. Stamp, P., Thiraporn, R., Geisler, G., 1984. Leaf anatomy of maize lines from different latitudes at sub- and supraoptimal temperatures. J. Exp. Bot. 35:384-388.
450. Starlinger, P., 1984. Transposable elements. Trends Biochem. Sci. 9:125-127.
451. Starlinger, P., Courage-Tebbe, U., Doring, H. P., Frommer, W. B., Theres, K., Tillmann, E., Weck, E., Werr, W., 1983. Isolation of transposable elements in maize. Pp. 67-75 in Genetic manipulation: Impact on Man and Society, W. Arber et al., eds., ICSU Press/Cambridge University Press.
452. Starlinger, P., Couragetebbe, U., Doring, H. P., Frommer, W. B., Tillmann, E., Weck, E., Werr, W., 1983. Transposable elements in plants. UCLA Symp. Molec. Cell. Biol. 12:423-434.
453. Stern, D. B., Hodge, T. P., Lonsdale, D. M., 1984. Homology between the ribosomal DNA of Escherichia coli and mitochondrial DNA preparations of maize is principally to sequences other than mitochondrial rRNA genes. Plant Mol. Biol. 3:355-362.
454. Stern, D. B., Newton, K. J., 1984. Isolation of intact plant mitochondrial RNA using aurintricarboxylic acid. Plant Mol. Biol. Rep. 2:8-15.
455. Stern, D. B., Palmer, J. D., 1984. Extensive and widespread homologies between mitochondrial DNA and chloroplast DNA in plants. Proc. Natl. Acad. Sci. USA 81:1946-1950.
456. Stiborova, M., Leblova, S., 1983. Isolation and partial characterization of 2 phosphoenol pyruvate carboxylases from maize (Zea mays) cultivar CE-2055. Photosynthetica 17:379-385.
457. Stiborova, M., Leblova, S., 1983. Regulation of maize (Zea mays) phosphoenol pyruvate carboxylase EC-4.1.1.31 by glycine, pyruvate and sulfate ions. Photosynthetica 17:386-390.

458. Stolte, D., 1983. 3.3 billion bushels by 1990: How do we do it? Proc. Annu. Corn and Sorghum Res. Conf. 38:80-86.
459. Straub, R. W., 1984. Maize dwarf mosaic virus: Symptomatology and yield reactions of susceptible and resistant sweet corns. Environ. Entomol. 13:318-323.
460. Strauss, A., Bucher, F., King, P. J., 1984. Application of the disc method to cultures. Plant Cells. 2. Exhibition zones. Plant Cell Tissue Organ Cult. 3:123-130.
461. Strittmatter, G., Kossel, H., 1984. Cotranscription and processing of 23S, 4.5S and 5S rRNA in chloroplasts from Zea mays. Nucl. Acid Res. 12:7633-7648.
462. Stromberg, E. L., Stienstra, W. C., Kommedahl, T., Matyac, C. A., Windels, C. E., Geadelmann, J. L., 1984. Smut expression and resistance of corn to Sphacelotheca reiliana in Minnesota. Plant Dis. 68:880-883.
463. Struik, P. C., 1984. An ideotype of forage maize for north-west Europe. Neth. J. Agr. Sci. 32:145-147.
464. Styer, C. H., Cutler, H. G., 1984. Effects of moniliformin on mitosis in maize (Zea mays L.). Plant Cell Physiol. 25:1077-1082.
465. Styer, R. C., Cantliffe, D. J., 1984. Dependence of seed vigor during germination on carbohydrate source in endosperm mutants of maize. Plant Physiol. 76:196-200.
466. Styer, R. C., Cantliffe, D. J., 1984. Infection of two endosperm mutants of sweet corn by Fusarium moniliforme and its effect on seedling vigor. Phytopathology 74:189-194.
467. Sumner, D. D., Cassidy, J. E., Szolics, I. M., Marco, G. J., Bakshi, K. S., Brusick, D. J., 1984. Evaluation of the mutagenic potential of corn (Zea mays L.) grown in untreated and atrazine (AAtrex ) treated soil in the field. Drug Chem. Toxicol. 7:243-258.
468. Sutton, W. D., Gerlach, W. L., Schwartz, D., Peacock, W. J., 1984. Molecular analysis of Ds controlling element mutations at the Adh1 locus of maize. Science 223:1265-1268.
469. Szaniel, I., Sagi, F., Palvolgyi, L., 1984. Hardness determination and quality prediction of maize kernels by a new instrument, the molograph. Maydica 29:9-20.
470. Taylor, W. C., Fragoso, L., 1983. Gene switching during maize leaf development. UCLA Symp. Molec. Cell. Biol. 12:381-389.
471. Tewary, B. K., Mookerjee, A., 1982. Effect of pre and post treatment of ascorbic acid on wheat and maize seeds exposed to gamma-radiation. Indian J. Plant Physiol. 25:258-265.
472. Thiraporn, R., Geisler, G., Stamp, P., 1983. Yield and relationships among yield components and N-related and P-related traits in maize genotypes under tropical conditions. Z. Acker- Pflanzenbau. 152:460-468.
473. Thirion, J. P., Peacock, W. J., Mans, R., Cross, J., Jacobs, M., Banuett, F., 1983. Gene transfer in maize--Controlling elements and the alcohol dehydrogenase genes--Discussion. Pp. 324-326 in Advances in Gene Technology: Molecular Genetics of Plants and Animals, K. Downey et al., eds., Academic Press, Orlando.
474. Thompson, D. L., Bergquist, R. R., 1984. Inheritance of mature plant resistance to Helminthosporium maydis Race 0 in maize. Crop Sci. 24:807-810.
475. Thompson, D. L., Rawlings, J. O., Zuber, M. S., Payne, G. A., Lillehoj, E. B., 1984. Aflatoxin accumulation in developing kernels of eight maize single crosses after inoculation with Aspergillus flavus. Plant Dis. 68:465-467.
476. Timmis, J. N., Domoney, C., Gavin, M., 1983. The relationship between ribosomal RNA gene dosage and the production of ribosomal RNA in diploid plants. J. Life Sci. R. Dublin Soc. 4:211-218.
477. Toldi, E. T., 1984. Relationship between DIMBOA content and Helminthosporium turcicum resistance in maize. Novenytermeles 33:213-218.
478. Tollenaar, M., Muldoon, J. F., Daynard, T. B., 1984. Differences in rates of leaf appearance among maize hybrids and phases of development. Can. J. Plant Sci. 64:759-764.
479. Torne, J. M., Santos, M. A., Blanco, J. L., 1984. Methods of obtaining maize totipotent tissues. II. Atrophic tissue culture. Plant Sci. Lett. 33:317-325.
480. Torti, G., Lombardi, L., Manzocchi, L. A., Salamini, F., 1984. Indole-3-acetic acid content in viable defective endosperm mutants of maize. Maydica 29:335-343.
481. Toth, R., Page, T., Castleberry, R., 1984. Differences in mycorrhizal colonization of maize selections for high and low ear leaf phosphorus. Crop Sci. 24:994-996.
482. Tozawa, H., 1984. A new indicator of earliness of maize varieties for whole crop silage, and its application to varietal combination in Hokkaido. JARQ 18:6-11.
483. Trapani, N., Gentinetta, E., 1984. Screening of maize genotypes using drought tolerance tests. Maydica 29:89-100.
484. Trapani, N., Motto, M., 1984. Combining ability for drought tolerance tests in maize populations. Maydica 29:325-334.
485. Troyer, A. F., 1984. Breeding corn for heat and drought tolerance. Proc. Annu. Corn Sorghum Res. Conf. 38:128-143.
486. Truongandre, I., Demarly, Y., 1984. Obtaining plants by in vitro culture of unfertilized maize ovaries (Zea mays L.) and preliminary studies on the progeny of a gynogenetic plant. Z. Pflanzenzucht.-J. Plant Breed. 92:309-320.
487. Tsai, C. Y., Huber, D. M., Glover, D. V., Warren, H. L., 1984. Relationship of N deposition to grain yield and N response of three maize hybrids. Crop Sci. 24:277-281.
488. Tsai, C.-L., Chung, H.-W., 1984. Development and yield of corn hybrids as affected by planting date. J. Agr. Assn. China-Taiwan 127:52-57.
489. Tseng, C. T., Guthrie, W. D., Russell, W. A., Robbins, J. C., Coats, J. R., Tollefson, J. J., 1984. Evaluation of two procedures to select for resistance to the European corn borer in a synthetic cultivar of maize. Crop Sci. 24:1129-1133.
490. Tseng, C. T., Tollefson, J. J., Guthrie, W. D., 1984. Evaluation of maize single-cross hybrids and inbred lines for resistance to 3rd-instar black cutworm larvae (Lepidoptera: Noctuidae). J. Econ. Entomol. 77:565-568.
491. Usuda, H., Ku, M. S. B., Edwards, G. E., 1984. Activation of NADP-malate dehydrogenase, pyruvate, Pi dikinase, and fructose 1,6-bisphosphatase in relation to photosynthetic rate in maize. Plant Physiol. 76:238-243.
492. Valenti, V., Stanghellini, M. A., Pupillo, P., 1984. Glucose 6-phosphate dehydrogenase isozymes of maize leaves. Some comparative properties. Plant Physiol. 521-526.
493. Valois, A. C. C., Filho, J. B. D., 1984. Comparison among selection methods in maize cv. Centralmex. Pesq. Agrop. Brasil. 19:169-178.
494. Valois, A. C. C., Filho, J. B. D., 1984. Estimation of variance components in the maize cultivar Centralmex. Pesquisa Agrop. Brasil. 19:479-488.

495. Valois, A. C. C., Vencovsky, R., 1983. Effects of stratified mass selection on 2 corn populations and on heterosis of their crosses. Pesq. Agrop. Brasil. 18:1099-1108.
496. Vasil, I. K., 1983. Isolation and culture of protoplasts of grasses. Int. Rev. Cytol. Suppl. 16:79-88.
497. Vasil, V., Vasil, I. K., 1984. Isolation and maintenance of embryogenic cell suspension cultures of Gramineae. Pp. 152-158 in Cell Culture and Somatic Cell Genetics of Plants, Vol. 1, I. K. Vasil, ed., Academic Press, Orlando.
498. Vasil, V., Vasil, I. K., 1984. Isolation and culture of embryogenic protoplasts of cereals and grasses. Pp. 398-404 in Cell Culture and Somatic Cell Genetics of Plants, Vol. 1, I. K. Vasil, ed., Academic Press, Orlando.
499. Vasil, V., Vasil, I. K., Lu, C. Y., 1984. Somatic embryogenesis in long-term callus cultures of Zea mays L. (Gramineae). Amer. J. Bot. 71:158-162.
500. Vidal, B. de C., da Silva, W. J., Strikis, P. C., 1984. Nuclear phenotypes and DNA content of root cells of Zea mays mays, Zea diploperennis and of a mazoid hybrid. Cell. Mol. Biol. 30:11-22.
501. Vincourt, P., 1984. Relation between rate of leaf emergence, total leaf number and earliness in maize. Agronomie 4:795-800.
502. Vuillaume, E., Vedel, F., Boutry, M., 1984. Analysis of native mitochondrial DNA in male-fertile maize mutants resistant to Helminthosporium maydis Race-T obtained by mutagenic treatments of seeds with Texas cytoplasm. Agronomie 4:291-294.
503. Walbot, V., 1983. Morphological and genomic variation in plants: Zea mays and its relatives. Pp. 257-278 in British Society for Developmental Biology Symposium, Vol. 6. Development and Evolution, B. C. Goodwin et al., eds., Cambridge University Press, New York.
504. Walbot, V., 1983. Suggestion for the efficient recovery of visible mutants. Plant Mol. Biol. Reporter 1:30-31.
505. Walbot, V., Cullis, C. A., 1983. The plasticity of the plant genome--Is it a requirement for success? Plant Mol. Biol. Reporter 1:3-11.
506. Walbot, V., Newton, K. J., Maloney, A., Sandie, T., Masters, B. S., McCarty, D., Fejes, E., Hauswirth, W. W., 1983. Mapping genes of the maize mitochondrial genome. UCLA Symp. Molec. Cell. Biol. 12:457-466.
507. Wall, J. S., Fey, D. A., Paulis, J. W., Landry, J., 1984. Improved two-dimensional electrophoretic separation of zein proteins: Application to study of zein inheritance in corn genotypes. Cereal Chem. 61:141-146.
508. Walton, J. D., Earle, E. D., 1984. Isolation and bioassay of fungal phytotoxins. Pp. 598-608 in Cell Culture and Somatic Cell Genetics of Plants, Vol. 1, I. K. Vasil, ed., Academic Press, Orlando.
509. Wang, A. S., Phillips, R. L., 1984. Synchronization of suspension culture cells. Pp. 175-181 in Cell Culture and Somatic Cell Genetics of Plants, Vol. 1, I. K. Vasil, ed., Academic Press, Orlando.
510. Weber, D. F., 1983. Monosomic analysis in diploid crop plants. Pp. 352-378 in Cytogenetics of Crop Plants, M. S. Swaminathan et al., eds., Macmillan India Ltd.
511. Weck, E., Courage, U., Doring, H.-P., Fedoroff, N., Starlinger, P., 1984. Analysis of sh-m6233, a mutation induced by the transposable element Ds in the sucrose synthase gene of Zea mays. EMBO J. 3:1713-1716.
512. Weil, J. H., Mubumbila, M., Kuntz, M., Keller, M., Crouse, E. J., Burkard, G., Guillemaut, P., Selden, R., McIntosh, L., Bogorad, L., Loffelhardt, W., Mucke, H., Bohnert, H. J., Dietrich, A., Souciet, G., Colas, B., Imbault, P., Sarantoglou, V., 1983. Comparative studies on tRNAs and aminoacyl-tRNA synthetases from various photosynthetic organisms. In Structure and Function of Plant Genomes, O. Ciferri and L. Dure, eds. Life Sci. 63:167-180.
513. Weil, J. H., Mubumbila, M., Kuntz, M., Keller, M., Steinmetz, A., Crouse, E. J., Burkard, G., Guillemaut, P., Selden, R., McIntosh, L., Bogorad, L., Loffelhardt, W., Mucke, H., Bohnert, H. J., 1983. Gene mapping studies and sequence determination on chloroplast transfer RNA from various photosynthetic organisms. Molek. Biol. 17:1147-1153; Mol. Biol. (Mosc.) 17:937-943.
514. Weller, R. F., Phipps, R. H., Griffith, E. S., 1984. The nutritive value of normal and brown midrib-3 maize. J. Agr. Sci. 103:223-228.
515. Wen, Z.-M., 1983. Study on the identification and classification of male-sterile cytoplasm in maize. Acta Genet. Sin. 10:477-482.
516. Wenzel, G., Foroughiwehr, B., 1984. Anther culture of cereals and grasses. Pp. 311-327 in Cell Culture and Somatic Cell Genetics of Plants, Vol. 1, I. K. Vasil, ed., Academic Press, Orlando.
517. Wessel-Beaver, L., Beck, R. H., Lambert, R. J., 1984. Rapid method for measuring kernel density. Agron. J. 76:307-309.
518. White, R. P., McRae, K. B., 1984. Assessment of yield improvement of short season corn (Zea mays) hybrids in Atlantic trials. Can. J. Plant Sci. 64:197-202.
519. Widstrom, N. W., Bagby, M. O., Palmer, D. M., Black, L. T., Carr, M. E., 1984. Relative stalk sugar yields among maize populations, cultivars, and hybrids. Crop Sci. 24:913-915.
520. Widstrom, N. W., Jellum, M. D., 1984. Chromosomal location of genes controlling oleic and linoleic acid composition in the germ oil of two maize inbreds. Crop Sci. 24:1113-1115.
521. Widstrom, N. W., McMillian, W. W., Wilson, D. M., Garwood, D. L., Glover, D. V., 1984. Growth characteristics of Aspergillus flavus on agar infused with maize kernel homogenates and aflatoxin contamination of whole kernel samples. Phytopathology 74:887-890.
522. Widstrom, N. W., Wilson, D. M., McMillian, W. W., 1984. Ear resistance of maize inbreds to field aflatoxin contamination. Crop Sci. 24:1155-1157.
523. Wienand, U., Sommer, H., Schwarz, Z., Shepherd, N., Saedler, H., Kreuzaler, F., Hahlbrock, K., Harrison, B., Peterson, P. A., 1983. Cloning of plant genes. Gene 22:293-294.
524. Williams, W. M., Satyanarayana, K. V., Kermicle, J. L., 1984. R-stippled maize as a transposable element system. Genetics 107:477-488.
525. Williams, W. P., Buckley, P. M., Taylor, V. N., 1983. Southwestern corn borer growth on callus initiated from corn genotypes with different levels of resistance to plant damage. Crop Sci. 23:1210-1211.
526. Williams, W. P., Davis, F. M., 1984. Reaction of a resistant and a susceptible corn hybrid to various southwestern corn borer infestation levels. Agron. J. 76:855-857.
527. Wilson, C. M., 1984. Note: Isoelectric focusing of zein in agarose. Cereal Chem. 61:198-200.
528. Wilson, D. R., Larkins, B. A., 1984. Zein gene organization in maize and related grasses. J. Mol. Evol. 20:330-340.
529. Wilson, R. L., Jarvis, J. L., Guthrie, W. D., 1983. Evaluation of maize for resistance to black cutworm larvae. Maydica 28:449-453.

530. Wiseman, B. R., Widstrom, N. W., McMillian, W. W., 1983. Influence of resistant and susceptible corn silks on selected developmental parameters of corn earworm (Lepidoptera: Noctuidae) larvae. J. Econ. Entomol. 76:1288-1290.

531. Yamada, M., 1983. Superiority of pollen from F1 plants of maize in selective fertilization. Jpn. Agr. Res. Quart. 17:166-172.

532. Yamada, M., 1984. Selective fertilization in maize, Zea mays L. III. Independence of gametophyte factors on superiority of pollen grains from F1 plants. Jpn. J. Breed. 34:9-16.

533. Yamada, M., Murakami, K.-I., 1983. Superiority in gamete competition of pollen derived from F1 plant in maize. Pp. 389-395 in Pollen: Biology and Implications for Plant Breeding, D. L. Mulcahy and E. Ottaviano, eds., Elsevier Science Publ. Co., New York.

534. Yurku, A. I., Balashova, N. N., Lazu, M. N., Bazelyuk, F. M., Prisyazhnaya, V. G., 1982. Maize resistance to Ustilago zeae populations in the Moldavian-SSR USSR. Izv. Akad. Nauk Mold. SSR Ser. Biol. Khim. Nauk (6):37-44.

535. Yurku, A. I., Palii, A. F., Lazu, M. N., Tsyganash, V. I., Balashova, N. N., 1983. Resistance to diseases and pests of analogs of a maize line with respect to separate genes of endosperm. Izv. Akad. Nauk Mold. SSR Ser. Biol. Khim. Nauk 3:29-32.

536. Zeleneva, I. V., 1983. Growth and activities of enzymes of oxidative metabolism in gamma-irradiated corn seedlings. Sov. Plant Physiol. 30:534-538.

537. Zenkteler, M., Nitzsche, W., 1984. Wide hybridization experiments in cereals. Theor. Appl. Genet. 68:311-315

538. Zhou, D., Guthrie, W. D., Chen, C., 1984. A bioassay technique for screening inbred lines of maize for resistance to leaf feeding by the European corn borer. Maydica 29:69-75.

539. Zurawski, G., Clegg, M. T., Brown, A. H. D., 1984. The nature of nucleotide sequence divergence between barley and maize chloroplast DNA. Genetics 106:735-749.

a1 36 85 86 100 108 162
a1-m1 4 5 6 34
a1-m1-5719A 111
a1-m1-6078 111
a1-m(papu) 35
A1-m(r) 3
a1-m(r) 4 5 6
a1-mdt 4 5
a1-mrh 4 5
a1-MumL 10 11 111
a1-Mum1-stable 12
a1-Mum2 10 11 13 35 111
a1-Mum3 10 11 13 111
a1-Mum3-stable 12
a1-ruq 4 5 6
a2 44 109 112 164
a2-m5 109
a2-m(r) 2 5
a3 101 108 162
Ac 4 6 8 13 22 28 33 38 91 111 112
Ac7 33 112
Ac9 33 112
Aco1 88 90 108 111 163
Aco2 88 113
Aco3 88 113
Aco4 88 113
Acp1 90 109 113
Acp1-0.1 112
Acp1-0.2 112
Acp1-0.05 112
Acp1-2.5 112
Acp1-5 112
actin-MAc1 114
ad1 160
Adh1 90 160
Adh1-2F11 33 111
Adh1-5 111
Adh1-33F 111
Adh1-78F 111
Adh1-CroF 111
Adh1-Ct 111
Adh1-F 24 111
Adh1-F63 111
Adh1-F207 111
Adh1-F460 111
Adh1-FCm 111
Adh1-FkF 111
Adh1-FkFgamma25 111
Adh1-Fm335 111
Adh1-gamma25 111
Adh1-PrF 111
Adh1-S 111
Adh1-S96 111
Adh1-S664 111
Adh1-S719 111
Adh1-S1015 111
Adh1-S1108 111
Adh1-S3020 111
Adh1-S3034 111
Adh1-S3034a 111
Adh1-S4477 111
Adh1-S4478 111
Adh1-S5446 111
Adh1-S5453 111
Adh1-S5657 111
Adh1-U725 24
Adh1-Usv 24 111
Adh2 163
Adk1 88 113
ae1 39 43 99 112 164
al1 161
alh1 160
am1 164
Amp1 88 90 160
Amp2 160
Amp3 88 90 164
Amy2 164
an1 38 160
Ap1 113
Ap2 113
Ap3 113
AR 56 112 113
ar1 40 113 166
as1 160
Asr1 23 111 163
Atc1 166
B1 54 101 161
B-chrom 110
ba1 108 111 162
ba2 161
ba*-s 40 113
bd1 165
Bf1 39 166
bf2 109 110 113 167
Bg 4 5
Bh1 165
Bif1 109 166
bk2 42 166
bm1 39 164
bm2 38 113 160
bm3 163
bm4 166
Bn1 165
br1 160
br2 24 160
brn1 7 108 111 162
Bs1 111
bt1 39 43 44 105 109 112 164
bt2 105 163
bv1 39 112 164
bx1 163
bz1 2 3 36 39 40 51 54 56 112 166
bz1-5598 112
bz1-m2 112
bz1-m4 4 5 6 33
bz1-m(nr)83g123-21 7
bz1-m(nr)83g123-22 7
bz1-m(nr)83g123-24 7
bz1-m(nr)83g123-25 7
bz1-m805137 6 112
bz1-m826301 7
bz1-mut 4 5
bz1-n(rcy)-sh1-83521 6 112
bz1-o 5 6
Bz1-r 112
bz1-rcy 4 5 6 112
bz1-rcy812215 6 112
bz1-rcy824325w 6 112
bz2 13 160
bz2-m 109
bz2-Mu1 96
c1 4 40 54 91 166
c1-836957 3 112
C1-I 2 3 5 51 52 112
C1-I-m836976 2 112
c1-m2 91
c1-m(r) 2 3
C1-m(r) 3 112
c1-m804531 4 112
c1-p 80
c1-ruq 3 4
c1-sh1-836882 2 112
c2 36 163
c2-m1 35
c2-m2 2 35
c2-m3 4 6
Car1 160
Cat1 81 164
Cat2 81 160
Cat3 81
Cat3-5.5 113
Cat3-6 113
Cat3-9+ 113
Cat3-9.5 113
Cat3-10 113
Cat3-10.5 113
Cg2 113
Ch1 38 161
Cin1 114
Cin2 35 114
Cin3 35 114
cl1 86 108 111 162
Clm4 86
Clt1 109
Clt*-985 42
cm1 167
cms-C 49 98 114
cms-G 98 114
cms-G' 98 114
cms-H 98
cms-J 98 114
cms-J' 98 114
cms-L 98
cms-LBN 113
cms-ME 98
cms-ML 114
cms-My 98
cms-R 98 114
cms-R' 98 114
cms-RD 114
cms-RU 49 113 114
cms-S 49 96 98 100 113 114
cms-T 49 50 96 98 114
cms-T4 50 114
cms-Vg 98
cp1 165
cp2 165
cp*-935 41
cp*-936A 41
cp*-1286A 41
cp*-1294 41
cp*-1381 113
cp*-1387A 109 112
cp*-1391 40
cp*-1392A 109 112
cp*-1393A 41
cp*-1405A 109 112
cp*-1430 41
cp*-1436A 108
cp*-1528 109 112
cp*-E1113A 104 111
cpf1*-1024A 91
cr1 108 162
crp*-888A 41
crp*-1058 109 112
crp*-1121 109 112
crp*-1533 109 112
ct2 160
ctDNA-cf1B 113
ctDNA-cf1E 113
ctDNA-LS 113
ctDNA-rcL 113
ctDNA-rDNA16S 113
ctDNA-rDNA23S 113
ctDNA-rp-S4 113
ctDNA-tRNA1ser 113
ctDNA-tRNA2ile 113
ctDNA-tRNAarg1 113
ctDNA-tRNAasn2 113
ctDNA-tRNAasp 113
ctDNA-tRNAile1 113
ctDNA-tRNAleu3 113
ctDNA-tRNAlys 113
ctDNA-tRNAmet1 113
ctDNA-tRNApro 113
ctDNA-tRNAser1 113
ctDNA-tRNAser2 113
ctDNA-tRNAthr1 113
ctDNA-tRNAtrp 113
ctDNA-tRNAtyr 113
ctDNA-tS-UGA 113
ctDNA-tV-UAC 113
Cx1 167
Cy 4 5 6 111 112
cytopl-G' 98 114
cytopl-J' 98 114
cytopl-R' 98 114
cytopl-Revolution 46
cytopl-T4 50 114
cytopl-TELE 46
cytopl-WF9 50
cytopl-ZD 49 114
cytopl-ZP 49 114
d1 86 108 111 162
d3 109 113 166
d5 161
D8 160
d*-660B 109 113
d*-E339 108 111
d*-E446 108 111
dcr*-1005A 91 111
dcr*-1156B 41
dcr*-E1428 104 111
de*-1007 41
de*-1057B 41
de*-1104B 41
de*-1122A 108
de*-1153 41
de*-1386A 109 112
de*-1390A 41
de*-1396A 41
de*-1409 40 113
de*-1520B 41
de*-1897 109 112
dek1 160
dek2 104 160
dek3 161
dek4 104 161
dek5 41 162
dek6 162
dek7 163
dek8 91 163
dek9 164
dek10 163
dek11 165
dek12 166
dek13 40 113 166
dek14 167
dek15 167
dek16 104 161
dek17 162
dek18 164
dek19 165
dek20 166
dek21 167
dek22 104 111 160
dek23 104 111 161
Dia1 88 90 108 111 161
Dia2 88 90 108 111 160
dnt*-889 41
dp1 163
Ds 4 13 22 33 91 111 112
Ds1 34
Ds9 112
Ds-2F11 33 111
dsc-ptd*-901A 108
Dt 28
Dt1 4 40 166
Dt2 165
Dt3 165
Dt5 166
Dt6 163
du1 167
E1 165
E3 162
E4 162
E8 90 108 162
E8-1 111
E8-2 111
E8-2.5 111
E8-4 88
E8-4.5 88
E8-7 111
E8-8 111

E8-9 111
E8-F 86
E8-S 86
egl 164
el1 166
En 2 4 7 35 112
En1 3 34
En-1 112
Enp1 81 90 112 165
Enp1-1 112
Enp1-4 112
Enp1-6 89 112
Enp1-7 112
Enp1-8 112
Enp1-9 112
Enp1-10 89 112
Enp1-12 112
Enp1-14 112
et1 6 39 52 53 108 162
et*-516C 91
fl 38 160
Fcu 4 5
fdt* 40 113
fl1 161
fl2 25 163
fl3 166
fl*-1316A 91 111
fl*-1333B 41
fl*-1390B 41
fl*-1426 41
fv1 40
g1 86 113 167
g2 86 162
Ga1 108 163
Ga1-S 23 24 111
ga2 164
ga7 108 162
ga8 166
Gdh1 90 160
Gdh2 90
gl1 15 76 86 109 112
113 165
gl2 161
gl3 24 76 108 111 163
gl4 94 108 111 163
gl6 76 85 108 111 162
gl8 15 39 112 164
gl11 161
gl15 40 166
gl17 164
gl18 166
Glu1 90 167
Glu1-1.2 113
Glu1-1.5 113
Glu1-1.8 113
Glu1-3.5 113
Glu1-4.5 113
Glu1-7.2 113
Glu1-7.5 113
Glu1-7.8 113
Glu1-8 113
Glu1-11 113
Glu1-12 113
gm*-1289 108
Got1 88 90 108 162
Got1-3.8 111
Got1-4.5 111
Got1-4.7 111
Got1-5.8 111
Got1-6.2 111
Got1-7 111
Got1-7* 111
Got1-8 111
Got2 90 164
Got2-3 112
Got2-7 112
Got2-10 112
Got2-F 81
Got2-S 81
Got3 81 90 164
Got3-2 112
Got3-6 112
Got3-n 112
gs1 38 160
gs2 161
hcf1 161
hcf2 160
hcf3 38 160
hcf18 39
hcf19 38 39 111
hcf*-19 38
Hex1 88 90 108 111 162
Hex1-4 88
Hex1-null 88
Hex2 88 90 112 165
Hex2-1 89
Hex2-2 89
hm1 160
hm2 166
Hs1 165
Hsf1 42 109 112
Hsf*-1595 42 112
Hsf*-1603 42 109 112
Ht1 161
I 2 34 35 111
I-R 113
id1 160
Idh1 81 90 109 166
Idh2 81 90 112 165
Idh2-2.5 112
Idh2-3.8 112
Idh2-4 89
Idh2-5 112
Idh2-5.5 112
Idh2-6 89
Idh2-8 112
ig1 108 162
ij1 26 56 165
in1 165
inh1 113
Isr1 167
j1 109 113 166
j2 163
K1L1 111
K1S2 111
K2L1 111
K2S1 111
K3L 108
K3L1 111
K3S1 111
K4L1 111
K4S2 111
K5L1 112
K5S1 112
K6L1 112
K6L2 112
K6L3 112
K7L1 112
K7S 112
K8L1 112
K8L2 112
K9L2 112
K9S 112
K10L1 113
K10L2 113
Kn1 160
Knob180bp 114
Knob202bp 114
Krn4 24 111
l1 110 113 167
l6 40 113 166
l7 40 113 166
l10 165
l11 165
l12 165
l13 165 167
l15 165
la1 94 163
Lc1 110 113 167
Les1 161
Les2 160
Les*-2005 42 109 113
Lfy1 113
lg1 113 161
lg2 4 39 85 86 108 162
Lg3 86 108 111 162
li1 167
lls1 160
Lmc1 23 113
Lmc2 23 113
lo2 40 166
lte1 161
Lte2 167
lu1 164
lw1 160
lw2 39 112 164
lw3 164
lw4 163
Mdh1 90 109 166
Mdh1-0.3 112
Mdh1-0.9 112
Mdh1-2.8 112
Mdh1-3.2 112
Mdh1-4 112
Mdh1-8.5 112
Mdh2 90 112 165
Mdh2-3 89
Mdh2-3.5 89
Mdh2-4 112
Mdh2-4m 112
Mdh2-5.8 112
Mdh2-5m 112
Mdh2-6 89
Mdh2-7.5 112
Mdh2-7.7 112
Mdh2-8.5 112
Mdh2-9.3 112
Mdh2-.02 112
Mdh2-.25 112
Mdh2-.31 112
Mdh2-.35 112
Mdh3 90 108 162
Mdh3-7.3 111
Mdh3-11.5 111
Mdh3-16 88
Mdh3-17.2 111
Mdh3-18 88
Mdh4 90 160
Mdh4-8.2 111
Mdh4-8.7 111
Mdh4-9 111
Mdh4-10 111
Mdh4-10.5 111
Mdh4-12 111
Mdh4-12- 111
Mdh4-14 111
Mdh4-14.7 111
Mdh4-15.5 111
Mdh5 90 164
Mdh5-7.7 112
Mdh5-8 112
Mdh5-13 112
Mdh5-14 112
Mdh5-14.4 112
Mdh5-16.7 112
Me1 88 90 108 162
mep1 164
mmm1 90 160
Mmm1-m 111
Mmm1-m1 111
mn1 161
mn*-1120A 91
monosome-1 111
monosome-2 111
monosome-3 111
monosome-4 111
monosome-6 112
monosome-7 112
monosome-8 112
monosome-9 112
monosome-10 113
Mp 111
Mr 166
Mrh 4 5
ms1 87 94 165
ms2 40 87 166
ms5 87 164
ms7 87 165
ms8 87 166
ms9 87 160
ms10 87 167
ms11 87
ms12 87
ms13 87 164
ms14 87 160
ms17 87 160
ms20 87
ms22-A632A 87 113
ms23-A619B 87
Ms23-A619B 113
ms24-LT 87 113
ms*-Bear7 87 113
Mst 110 113
Mst1 167
mtDNA 50
mtDNA-1.4 49 114
mtDNA-1.9 49 114
mtDNA-2.1 49 96 114
mtDNA-2.3 49 96 114
mtDNA-2.35 114
mtDNA-7.4 49
mtDNA-COB 113
mtDNA-COI 113
mtDNA-COII 113
mtDNA-D1 49 114
mtDNA-D2 49 114
mtDNA-mox1 113
mtDNA-psi 113
mtDNA-R1 49 113 114
mtDNA-R2 49 113 114
mtDNA-rDNA18S 113
mtDNA-rDNA26S 113
mtDNA-S1 49 96 98 100
113 114
mtDNA-S2 49 96 98 100
113 114
mtDNA-sigma 113
Mu 6 7 8 11 14 15 111
Mu1 5 10 13 16 35 96
111
Mu-I 113
Mu-L 96 113
Mu-S1 96 113
Mu-S2 96 113
Mu-S3 96 113
Mut 4 6 161
na1 39 108 111 162
na2 39 164
NCS2 113
NCS3 113
nec1 166
nec2 160
nec3 44 164
nec4 161
nec*-409 44
nec*-493 44
nl1 167
o1 24 91 108 111 163
o2 165
o2-m(r) 4 5
o2-mr 5
o5 165
o7 80 110 113 167
o*-933 41
o*-948B 41
o*-999A 91
o*-1119A 41
o*-1189A 41
o*-1195A 91 111
o*-1214A 109 112

o*-1310A 41
o*-1313 41
o*-1383 41 109 112
o*-1384A 41
o*-1388 41
Ogl 167
ogm*-1488B 91 111
ora2-13 104 113
ora2-15 104 113
orol 165
orp*-1186A 41
oyl 76 109 110 113 167
Pl 111
pl 160
Pl-RR 100
Pl-WR 40 100
Pl-WW 40
pbl 165
pb4 165
pgl1 18 20 165
pgl2 18 20 40 113 166
pg*-330A 91
Pgdl 81 90 112 165
Pgdl-0.5 112
Pgdl-1.8 112
Pgdl-2 89
Pgdl-3.8 89
Pgdl-9 112
Pgdl-12 112
Pgdl-14 112
Pgd2 90 108 162
Pgd2-2.8 88
Pgd2-5 88
Pgd2-8 111
Pgd2-10 111
Pgd2-F 81
Pgd2-S 81
Pgml 81 90 160
Pgml-12 111
Pgm2 81 90 164
Pgm2-0.3 112
Pgm2-n 112
pgspt*-278B 91
pgspt*-464 91
pgspt*-579B 91 111
Phl 163
Phil 90 160
Phil-1 111
Phil-1.7 111
Phil-3.5 111
Phil-4.5 111
Phil-8 111
Pl1 54 79 89 100 112 165
pml 39 108 111 162
Pnl 101 165
pol 87 106 107 165
ppg*-199B 109 112
prl 36 39 51 112 164
prol 109 112 166
Protl 160
psl 164
Ptl 165
ptd*-1130A 91
pyl 89 165
pydl 166
rl 3 86 109 110 167
rl-ch 100
rl-ch(Hopi) 79 80 113
rl-cu 5
rl-g 80
Rl-g:1 113
Rl-g:8pale 113
rl-g:de4-128 113
rl-g:de4-137 113
rl-g:de4-139 113
rl-g:de4-142 113
rl-g:e 113
rl-g:nc3-5 113
rl-g:Sd 113
Rl-r 101
Rl-r:Ecuador(1172) 101
rl-r:n19 113
rl-r:n35 113
rl-r:n101 113
rl-r:W22 113
Rl-scm 86
Rl-st 80 113
rl-xl 45 76 113
ral 114 165
ra2 108 113 114 162
ra3 114
rdl 160
rd2 165
rDNA 112
rDNA17S 112 113
rDNA26S 112
Rfl 108 162
Rf2 166
Rf3 98 161
Rgl 108 162
rgdl 165
Rgd2 42
rgh*-786A 91
rgh*-1154A 109 112
rgh*-1285 109 112
rghd* 40 113
rhml 114 165
Ril 163
Rpl 167
Rp3 162
Rp4 163
Rp5 167
Rp6 167
Rpp9 114 167
rsl 160
rtl 162
Sadl 88 90 109 110 113
Sdwl 42 109 112
Sdw*-1592 42 109 112
shl 2 3 4 6 32 40 43 56 112 113 166
shl-5582 112
shl-5584 112
shl-5586 56 112 113
shl-5588 56 112 113
shl-5592 112
shl-5594 112
shl-5596 112
shl-835211w 6
shl-835211x-1 6
shl-836882 2
shl-bzl-m4 33
shl-bzl-x2 56 112
shl-bzl-x3 112
shl-m5933 23 112
shl-m6233 112
shl-m6258 112
shl-m6795 112
sh2 12 13 85 105 108 162
sh*-1295 41
sh*-1324A 41
sh*-1341 41
sh*-1530 41 109 112
sil 165
skl 103 161
Sksl 161
sll 165
sml 165
smk*-890 41
smk*-1167B 41
smk*-1168A 41
smk*-1373A 41
smk*-1423 41
smk*-1424 41
smk*-1484 41
Snl 80 113
Spcl 162
Spm 28 35 38 109
Spm-I8 34 112
srl 76 160
sr2 110 113 167
sr3 167
stl 163
sul 94 105 113 163
su2 43 165
Tl-2(4464) 39 160 161
Tl-2c 160 161
Tl-3(4759-3) 160 162
Tl-3(5242) 160 162
Tl-3(5267) 160
Tl-3e 160
Tl-4(4692) 160 163
Tl-5(8041) 160 164
Tl-9(4995) 42 57 113
Tl-9(8389) 42 113
Tl-9c 111
T2-3(6270) 161
T2-3(7285) 161
T3-4(5156) 100
T3-5(5521) 162
T3-5(7043) 162 164
T3-5b 162 164
T4-7(4698) 163 165
T4-9(5974) 100
T4-9(6222) 163 166
T4-9(6504) 163 166
T4-9b 42 111 113
T4-9g 42 100 113
T4-10f 100 111 113
T5-9(4817) 57
T5-9a 42 109 112
T5-9c 42 109 112
T6-9a 42 113
T6-9b 94
T7-9a 42 57 112 113 114
T7-9b 57 112 114
T8-9(6673) 42 112 113
T8-9d 42 109 112
T9-10b 57
tbl 160
TB-1La 45 160 162 163 164
TB-1La-3L4759-3 160 162
TB-1La-3L5242 160 162
TB-1La-3L5267 160 162
TB-1La-3Le 160 162
TB-1La-4L4692 160 163
TB-1La-5S8041 160 164
TB-1Lc 160
TB-1Sb 39 45 160 161
TB-1Sb-2L4464 39 160 161
TB-1Sb-2Lc 160 161
TB-3La 3 38 39 111 161 162
TB-3La-2L7285 161 162
TB-3La-2S6270 161 162
TB-3Lc 3 39 45 111 162
TB-3Ld 4 39 108 111 162
TB-3Lf 4 39 111 162
TB-3Lg 4 39 111 162
TB-3Lh 4 39 111 162
TB-3Li 4 39 111 162
TB-3Lj 4 111 162
TB-3Lk 4 39 111 162
TB-3Ll 4 39 111 162
TB-3Lm 4 39 111 162
TB-3Sb 7 39 88 108 111 162
TB-4Lb 45 163
TB-4Lc 163
TB-4Ld 163
TB-4Le 163
TB-4Lf 39 163
TB-4Sa 39 45 108 111 163
TB-4Sg 163
TB-5La 39 45 109 112 162 164
TB-5La-3L5521 162 164
TB-5La-3L7043 162 164
TB-5La-3Lb 162 164
TB-5Lb 39 164
TB-5Ld 39 112 164
TB-5Sc 164
TB-6Lb 39 165
TB-6Lc 45 90 112 165
TB-6Ld 165
TB-6Sa 39 45 165
TB-7Lb 39 45 86 109 163 165
TB-7Lb-4L4698 163 165
TB-7Sc 165
TB-8La 109 166
TB-8Lb 166
TB-8Lc 39 109 112 166
TB-9(La+Sb) 53 54
TB-9La 40 53 54 112 113 166
TB-9Lc 40 45 113 166
TB-9Sb 18 39 40 53 54 112 113 163 166
TB-9Sb-4L6222 163 166
TB-9Sb-4L6504 163 166
TB-9Sd 45 166
TB-10L1 167
TB-10L2 167
TB-10L3 167
TB-10L4 167
TB-10L5 167
TB-10L6 167
TB-10L7 167
TB-10L8 167
TB-10L9 167
TB-10L10 167
TB-10L11 167
TB-10L12 167
TB-10L13 167
TB-10L14 167
TB-10L15 167
TB-10L16 167
TB-10L17 167
TB-10L18 44 167
TB-10L19 39 44 86 167
TB-10L20 167
TB-10L21 167
TB-10L22 167
TB-10L23 167
TB-10L24 167
TB-10L25 167
TB-10L26 167
TB-10L27 167
TB-10L28 167
TB-10L29 167
TB-10L30 167
TB-10L31 167
TB-10L32 167
TB-10L33 167
TB-10L34 167
TB-10L35 167
TB-10L36 167
TB-10L37 167
TB-10L38 167
TB-10La 39 109 110 113 167
TB-10Lb 167
TB-10Sc 39 45 109 110 113 167
tdl 39 112 164
Teol 35 114
Til 101
Tpl 86 165
Tp2 86 167
Tpil 88 113
Tpi2 88 113
Tpi3 88 90 109 112 166
Tpi4 88 90 108 111 162

Tpi4-4 88
Tpi4-null 88
trisome-10 113
ts1 103 161
ts2 103 160
ts4 4 86 108 162
Ts5 163
Ts6 160
Tu1 101 163
ub*-76C 109 112
Uq 3 4 112
Uq-Hi-loss 3
v1 40 112 113 166
v2 164
v3 39 76 164
v4 38 76 91 111 161
v5 165
v8 163
v12 164
v16 76 109 166
v21 166
v22 160
v23 163
v*-78 109 112
v*-25 109 112
v*-298 109 112
v*-350 38 111
v*-358C 109 112
v*-424 38 111
v*-453 108
v*-576A 38 111
v*-588A 38 111
v*-779A 109 112
v*-826 109 112
v*-8520 76
v*-8587 40 113
v*-MS61 40 113
va1 165
Vg1 160
vp1 3 85 108 111 162
vp1-c821708 4 111
vp1-m451 3 4 111
vp1-rcy 4
vp2 164
vp5 31 160
vp8 160
vp9 165
w1 165
w2 167
w3 31 38 91 111 161
w11 40 166
w14 165
w15 165
w16 165
w17 165
w*-6-10a 40 113
w*-034-5 40 113
w*-8889 40 113
w*-8950 40 113
w*-s9000 40 113
Wc1 166
wd1 18 40 113 166
whp1 161
wi1 165
wl*-203A 109 112
wl*-1982 109 112
wl*-1985 109 112
wl*-EM41 40 113
wlv*-EM53 40 113
wlvpg*-424 91
ws3 161
wt1 161
wt*-472A 108
wx1 2 3 4 14 15 38 42 54 99 109 111 111 112 113 113 114 166
wx1-84-4 3 112
wx1-84-11 3 112
wx1-844 34 35 112
wx1-m6 23
wx1-m7 33 112
wx1-m8 2 34 109 112
wx1-m9 33 112
wx1-m9.Ac 23
wx1-m9.Ds 22
wyg1 165
y1 8 14 15 89 113
Y1 165
Y3 161
y8 165
y9 109 110 113 167
y10 162
yd2 162
yel*-034-16 40 113
yel*-7748 109 112
yg1 164
yg2 16 40 76 166
yg2-Mud 17
yg*-5588 40 113
ys1 39 164
ys2 160
ys3 39 162
Ysk1 163
zb3 164
zb4 160
zb6 163
zein10-19.6 100 111
zein21.5-19.6 99 111
zein21.5-22.5 99 111
zein22-19.6 99 111
zein22-22.5 99 111
zein28-22.5 100 111
zein33.5-22.5 100 111
zein41-14.3 100 113
zein49-19.6 100 113
zein53-19.6 100 111
zein54-19.6 100
zein-ZG19.7 114
zein-ZG19.12 114
zein-ZG19.30 114
zein-ZG19.31 114
zein-ZG19.32 114
Zer1 23
zn1 167
Zp1 165
Zp2 165
Zp3 165
Zp6 163 165
Zp10 163
Zp12 163
Zp14 163
Zp15 163
Zp16 165
Zp21 165
Zp22 163
Zp22/2 110 113
Zp27 163
Zp28 163
Zp29 165
Zp30 163

(r refers to the numbered references in Recent Maize Publications)


Abbott A G r1
Abdelrahman A A r2
Ackerley C A r350
Ackermann M r430
Adams N J r3
Adamu I M r424
Afuakwa J J r4 r5
Agbana S B r117
Aguilar R r94 r398
Ainsworth C C r1
Akhmetov R R r191
Aksenova V A r73
Al-Ithawi B A r22
Al-Younis A H r22
Albergoni F r6
Albertsen M C 87 108 109
Alexander D C 2
Alexander W L r7
Allard R W r199
Altman A r8
Altschuler M I r9 r10
Ambros V 106
Anderson E G 40
Anderson E L r11 r12
Anderson M E r155
Anderson P C r149
Andrew R H r76
Antonov M r211
Aranda G r13
Araujo S M C D r19
Argos P r236
Armbrust D L 58
Armour J W r417
Armstrong C L 93 114 r149
Arntzen C r14
Arzt A H r281
Ashman R B r384
Atanassov A r15
Atkinson B G 77
Atlin G N r16
Atlin G W r17
Avato P r18 r37
Ayala-Osuna J r19
Bagby M O r519
Bagnara D r20
Bagrationi N N r21
Bains G S r433
Bakshi K S r467
Baktash F Y r22
Balashova N N r534 r535
Balint A r139
Balke N E r90
Banuett F r473
Barker R F 96 111 r23
Barlow P W r24
Barnason A 91
Baron V S r25
Barriere Y r26 r27
Barry B D r156 r173 r250
Barve S S r403
Bashe D r279
Basso B r6
Baszczynski C L 75 76 77 78
Bates G W r28
Bazelyuk F M r534
Beadle G W 61 87 107
Beck R H r517
Beckert M 114 r29
Beckett J B 3 39 45 55 87 89 108 109 110 111 112 115
Bedinger P 96 114
Behrens U 33 112 r30
Below F E r85 r86
Beltchev B r218
Bennett M D r31 r174
Bennetzen J L 13 96 97 111 r23 r32 r97
Benzion G 94
Berg P 2
Bergquist R R r33 r474
Berndtgen R 35 35 111 112
Berry E C r192 r193
Bertoia L M 67 69 114
Bertram I r152 r153
Berville A r34
Bettencourt E r35
Bewley J D r170
Bhan M K 95 r198
Bhargava S C r36
Bianchi G r18 r37
Binelli G 81 110 113 r38
Binns A N r283
Birchler J A r39
Bird R McK 1 42 43 109 112 113 r40
Black L T r519
Blake D F r147
Blanco J L r402 r479
Blaschek W r353
Blasing T J r41
Bloc D r42
Bloom M 113 r43
Blumberg vel Spalve J 35 114 r427
Bock K 46
Bogorad L 113 r44 r45 r87 r223 r512 r513
Bohnert H J r512 r513
Boller Gallo P r252
Bommineni V R 75
Bonavent J-F r34
Bonhomme R r46 r47 r111
Bonner J 81
Boone L V r48
Boothe J G 75 77
Borisov V N r188
Bosch L 20 22
Bottomley W r162
Bouthyette P r389
Boutry M r502
Boyat A r26
Boyer C D 99 r49 r169
Brakke M K r50
Brambl R r57
Branca C r51
Braun C J r248
Brazil M 1
Brettell R I S 24 50 111 r418
Brewbaker J L r245
Briat J F r52
Bricker T M r295
Briggs W R r266 r267
Brink D E r99
Brink R A 26 79 107 r53
Broccoli A M 68 114
Broglie R r54
Brooker D B r155
Brot N r8 r43
Brown A H D 24 r539
Brown J W S 48 r233
Brown W L r55
Browne C 1
Bruehl G W r56
Brulfert J r95
Brusick D J r467
Bucher F r460
Buckley P M r525
Buescher P J 112 r57
Bukhari A I 33
Burkard G 113 r58 r512 r513
Burnham C R 53 54 94
Burr B r59 r60
Burr F A r59 r60
Bussard J B 36
Butenko R G r227
Cacco G r396
Caetano M D F r19
Callis J r385 r386
Callow R S r61
Camussi A r62
Canaday J r58
Cantliffe D J r465 r466
Cao M Q r29
Capitanio R r63
Caporali L r64
Carballo-Carballo A r318
Carey E E r298
Carlson J r291
Carlson W R 53 54 55 108 110
Carr M E r519
Carvajal M, A T r65
Casadoro G r378
Casanas F 20 22
Cassidy J E r467
Castle W E 107
Castleberry R M r66 r481
Cataneo A r332
Caviedes C, M r67
Cesario J r13
Ceska O 40 101 r68
Chaleff D r124
Chaleff R r78
Chandlee J M r69 r70 r407
Chandler V 98
Chang M-T 1 38 42
Chao S 113 r71
Chappell K 1
Charbonnier M r34
Chen C r538
Chen Y M 81
Chevallier M-H r72
Chimenti C r160
Chivkunova O B r73
Chourey P S 32 r78 r286
Chow K H r74
Christeller J T r75
Chua N-H r54
Chung H-W r488
Churchill G A r76
Chyi Y S r77 r400
Clark J K 104
Clark R B r351
Clark R L 1 r129 r192
Clegg M T r539
Coats J R r489
Cocking E C r78
Coe E H Jr 1 5 40 44 51 52 108 109 113 159 r79 r80
Cohen B N r8 r43
Cohen de Hunau R r160
Cohen J I r81
Colas B r512
Colbert T R r82
Compton W A r171
Connelly J A r88
Conroy J M r83
Contreras M R r313
Cook F S 74
Cooper P 75 76 78 r84
Coors J G r389
Coruzzi G r54
Courage-Tebbe U 23 r107 r124 r451 r452 r511
Coyne D P r351
Crafts-Brandner S J r85 r86
Crane P L 108
Crookston R K r4 r5 r196
Crosbie T M r310
Cross H Z r7
Cross J r473
Crossland L D 113 r44 r87
Crouse E J r58 r512 r513
Crowe T G 76
Crum C W r66
Cudny H r352
Cullis C A r505
Cully D E r88
Curtis C A 1 55
Curtis C R r361
Cutler H G r464
Cuypers H 35

da Silva W J r500
Dahl B E r409
Dale R M K 113 r89
D'Arcy C J r298 r299 r300
Darmstadt G L r90
Darrah L L 48 r55 r82 r173 r251
Dattee Y r72
Davidson D r91 r92
Davis F M r168 r526
Dawson A J 113 r93 r239 r240 r241
Daynard T B r20 r25 r110 r242 r315 r316 r380 r478
De Jimenez E S r94
Deleens E r95
Delibes A r258
Dellaporta S L 109 r96 r311
Demarly Y r486
Dempsey E 3
Dennis E S 111 114 r97 r98 r342
Derieux M r46 r47 r111
DeWet J M J r99 r100
Dey S K r210
Dhillon B S r101 r209 r210 r423
Dickinson D B r426
Dierks-Ventling C r430
Dietrich A r512
DiFonzo N r272 r308
Dinklaker J r134
Dixon L K r239
Dlouhy S R 24
Dodd J r102 r335
Doebley J F 61 111 112 113 r103 r104
Dolbeer R A r105 r281
Dolezelova M r324 r330
Dolgikh Y R r360
Dollinger E J 104 113
Domoney C r476
Donald C M r106
Dooner H K 85 111
Doring H-P 33 112 r107 r108 r109 r124 r451 r452 r511
Dow E W r110
Doyle G G 37
Doyle T 37
Dron M r52
Duburcq J-B r46 r47 r111
Dudka E L r188
Dudley J W r112 r113 r229 r288
Dunlop M J 75
Durbey S L r114
Duvick D N r41 r115
Eagles H A r382
Earle E D 55 r434 r508
Echt C 1
Edmeades G O r125
Edwards G E r491
Edwards H r335
Egri K r340
Eibel H r233
Eisenberg A 2 r279
El-Kady M A r116
El-Nagouly O O r116
El-Sherbieny H Y r116
Elfman B r179
Ellis R Jr r428
Enerson P M r16
Erickson E H Jr r128
Erkeev M I r228
Escote L J 100 114
Esen A r83
Everett H L 56 r389
Fakorede M A B r117
Falconer D S 107
Faluyi J O 114 r118
Fauron C M R r256
Fedoroff N V 22 26 33 112 115 r30 r119 r120 r121 r122 r123 r124 r364 r365 r511
Feix G 48 r233
Fejes E r506
Ferl R J 24 r97
Ferrari G r396
Ferrari L 70 114
Fey D A r507
Filho J B D r493 r494
Filichkin S A r360
Findley W R r251
Fischer K S r125
Fish L E r44
Fisher M B r49
Flavell R B 73 r1 r126 r127 r256
Fletcher G B 108 115 r100
Flottum P K r128
Foley D C r129
Fong F 31 r130
Ford R E r299 r300
Forde B G r297
Forde J r297
Foroughiwehr B r516
Foster J E r401
Fox T D r239
Fragoso L r470
Franz G r353
Fraser A S 107
Freeling M 111 r32 r131 r132 r159 r206 r207 r312 r385
Freier G r133
Friedemann P O 4
Frohlich W G r134
Frommer W-B 32 r451 r452
Frova C 81
Furtek D B r120
Furuichi Y 76
Gabay-Laughnan S 76 100 r238
Galante E r135
Galinat W C 28 32 102 103 108 r81 r136
Galt D L r313
Gamez R r137
Garcia-Arenal F r434
Gardner C O 57 58 r199 r205 r368 r369
Garwood D L r422 r521
Gaskin P r446
Gatenby A A r138
Gauvrit C r13
Gavazzi G 79 80 113
Gavin M r476
Gay J P r42
Gaynor J J r28
Geadelmann J L r462
Geczki I r139
Geiser M r107 r124
Geisler G r307 r449 r472
Gelvin S B r146
Gendloff E r166
Gengenbach B G 50 91 99 r88 r140 r437
Genov M r211
Gentile G J r359
Gentile J M r359
Gentinetta E r18 r63 r483
Georgiev I M r141 r142
Gerage A C r332
Gerlach W L r97 r342 r468
Gerrish E E r143
Ghazi A r34
Gierl A 32 33 35 r414
Giles K L r144
Gilmour S J r446
Ginsburg H r89
Gladwell I r61
Glass B r145
Gloeckler R r58
Glover D V r55 r182 r487 r521
Goldsbrough P B r146
Golubovsky M D r261
Gomez L A r147
Goodman M M 86 88 90 108 109 r104 r439
Goodwin-Bailey C I r259
Gordon K r58
Gordon M P r78
Gordon P N 85
Gorrini A 80
Gottesfeld J M 82
Gouet J P r42
Gould S J r148
Gracen V E 55 56 r389 r434
Green C E 24 84 93 r78 r149 r437
Green M M 16
Greenblatt I M 111 r150
Greyson R I 73 74 75 r370
Griffith E S r514
Grigorenko N V r234
Grobman A 28
Gu M G 29 r151
Guilhem C r13
Guillemaut P r58 r512 r513
Gupta M 114 r152 r153 r427
Guseva A I r154
Gusmao L r35
Gutheil R A r155
Guthrie W D r156 r157 r158 r192 r193 r250 r489 r490 r529 r538
Guyon P r230 r231
Hack E r239 r240 r241
Haemmerle C 99
Hageman R H r85 r86 r292
Hagen G r291
Hagenson M J 13
Hahlbrock K r523
Hajos L r139
Hake S 111 r159
Hall A J r133 r160
Hallauer A R r161 r343 r432
Hallick R B r162
Hamblin J r106
Hamilton R H 81
Hammer K r345
Han C-D 1
Hanson A D 24
Hanson W D r163 r306
Harlan J R r100 r254
Harms C T 84
Harper J E r85 r86
Harpster M H r164 r323
Harris J B r165
Harrison B r523
Hart L P 114 r166
Hauptmann R M r84
Hauswirth W W r506
Hawes M C r167
Hayman B I 92
Hedin P A r168
Hedman K D r169
Heggestad H E r361
Heikkila J J r170
Helms T C r171
Henry V C R r172
Henson A R r173
Herkovits J 59 r377
Hersberg M 76
Heslop-Harrison J S r31 r174 r175 r176
Heslop-Harrison Y r175 r176
Hibberd K A r177
Hightower R C r421
Hill L D r341
Hilu K W r100
Ho T-H D 78 r84 r254
Hodge T P r256 r453
Hoisington D A 1 44 108 110 159 r79
Holden M J r178
Hopkins W G r179
Horner E S r251
Horvitz H R 106
Hoseney R C r2

Hostas E de 96
Hu W W L r249
Huala E r267
Huber D M r487
Hunter B r180
Hunter B G 94
Hunter R B r16 r17
Hurley C K 81
Hymowitz T r373
Iltis H H 61 r181
Imbault P r512
Inglis A r97
Inouchi N r182
Inoue Y r183 r184 r185
Isaac P G r240 r241
Ishige T 99 114 r186
Ishii S r187
Ivakhnenko A N r188
Ivancic A r189
Ivanovic M r190
Ivantsov A I r191
Jackson W A r12
Jacobs M r473
Jarayam C 52
Jardetzky O r385 r386
Jarvis J L r157 r158 r192 r193 r250 r529
Jellum M D r520
Jenkins C L D r194
Jensen S r195
Jeppesen M 24
Jinks J L 92
Johns M A r312
Johnson E C r125
Jones D A r278
Jones D F 24
Jones R J r5 r196
Jones V P r93 r240 r241
Jordan E G r197
Josephson L M 46
Jotshi P N 95 r198
Jozsa S r301
Juvik J A r298
Kahler A L r199 r368
Kahn E J Jr r200
Kalinnikov D D r201 r202
Kamalova T G r219
Kamprath E J r11 r12
Kamyshova T V r201
Kanemasu E T r215
Kang M S r203
Karcher S J r146
Kasinova G V r354
Kato Y, T A 111 112 113 r65 r67 r204
Keim K R r205
Keith B r54
Keith G r58
Keller M r512 r513
Keller P B r311
Kelley P M r206 r207
Kemble R J 98
Kermicle J L 76 108 110 113 r208 r524
Khavkin E E r269 r303
Khehra A S r101 r209 r210 r423
Khristov K r211 r212
Khristova P r212
Kimber A r213
Kindiger B 1 39 55
King P J r214 r460
King S B r3 r415 r416 r417
Kirkham M B r215
Kirpa N Y r321
Kleese R A r216
Klein A S r217 r322
Klein D r134
Klosgen R B 35 r414
Kobza S r301
Koehler D E r130
Koleva S r218
Kommedahl T r462
Kondrat'ev M N r219
Kono Y r220
Kossel H r461
Kostyshin S S r221
Kovacevic V r222
Kowalewski S 1
Krause G F r155 r203
Krebbers E T 113 r44 r45 r223
Kreis M r297
Kreuzaler F r523
Kridl J C 114 r89 r224
Krieger J H 73
Krivov N V r261
Kriz A L 77
Krueger R W r294
Kruger G H J r225
Krull C F r66
Ku M S B r491
Kuang V D r226 r227
Kuck U r44
Kudoyarova G R r228
Kuhn W r195
Kullman A r448
Kumari A S 52 53 r375
Kuntz M r58 r512 r513
Kuznetsov L V r73
Kwolek W F r250 r251
Lai F S r371
Laird A r30
Lambert R J r288 r292 r517
Lamkey K R r229
Landi P r334
Landry J 72 r230 r231 r232 r507
Langridge P 48 r233
Lapina T V r234
Lara F M r19
Larkin P J r418
Larkins B A 114 r146 r235 r236 r272 r528
Larrinua I M r44 r45
Larson R L 36
Laubengayer R A 101
Laughnan J R 76 98 100 r237 r238
Lazu M N r534 r535
Leask W C r316
Leaver C J 113 r93 r239 r240 r241
Lebedev A V r326
Leblova S r456 r457
LeDrew H D r242
Lee B T O 107
Lee L r243 r244
Lee M 91
Lee M H r245
Lehman P 78
Leleji O 3
Lemke C A 56
Leon P r137
Leopold A C r397
Leto K J r246
Levic J r247
Levings C S III 40 98 100 113 r71 r248 r249 r411
Lewis E D r283
Lillehoj E B r250 r251 r475
Lima M r252
Lin B-Y 39 45 46 110 r253
Lin C-Y 78
Lin L-S 112 r254
Liu L-H r443
Llewellyn D r97 r342
Lodholz M L r158
Loffelhardt W r512 r513
Logvinenko V F r234
Lombardi L r480
Lonsdale D M 50 113 r255 r256 r257 r408 r453
Lopez-Brana I r258
Lorz H 84 r342
Loughman B C r259
Lu C Y 51 59 r499
Ludevid M D r260 r275
Lysikov V N 113 r261
Maas C 32
MacDonald F D 105
Mache R r52
MacMillan J r446
Magoja J L 67 68 69 70 71 72
Maguire M P r262 r263 r264
Mahey R K r265
Major D J r110
Malashenko A M r354
Malhi N S r209 r210
Malhotra V V r209 r423
Maloney A r506
Mandoli D F r266 r267
Mangelsdorf P C 28 61 101 102 105 r268
Mans R r473
Manwiller A r251
Manzocchi L A r135 r480
Maranville J W r351
Marco G J r467
Marekov L N r218
Mariani P r378
Marinova E r218
Markov E Y r269
Markova M D r270
Marks M D 114 r235 r236 r271 r272
Marotta R r308
Marschner H r390
Martin C R r371
Martin M J r273 r274
Martinez-Izquierdo J A r260 r275
Martiniello P r276
Marwaha K K r277 r339 r405 r406
Maryam B r278
Mascarenhas J P 2 r279
Mascarenhas N T 2 r279
Mascia P 108
Mashingaidze K r314
Mashnenkov A S 105
Masikevich Y G r221
Masnica M r280
Mason J R r281
Masters B S r506
Mateyka C r282
Mather K 92
Mathias R 73
Mathis D 82
Matyac C A r462
Matzke A J M r283
Matzke M A r283
Mayfield S P r164 r284 r285 r323
McAllan A B r349
McCarty D r506
McClintock B 22 26 32 33 34 35
McConnell R r195
McCormick S M 33 91 108 111
McGirr L G r16
McIntosh L r512 r513
McKnight D 2
McMillian W W r250 r521 r522 r530
McMillin D E 89
McNay J W 113 r286
McRae K B r287 r518
McWhirter K S 85 107
Meagher R B r421
Meghji M R r288
Melchiorre P r62
Mendu N r89
Merckelbach A 33 34 111
Merezhinsky Yu G r234
Messing J 112 r224 r289 r290 r291 r364 r365
Messmer M J r292
Mesterhazy A r293
Metz J G r294 r295
Micke A r296

Miflin B J r297
Mikel M A 114 r298 r299 r300
Miksche J P r165
Miles D r294 r295
Milinko I r301
Miranda Filho J B r252 r302
Miranda L E C de 24 108
Miranda L T de 24 108 111 113 114
Misharin S I r303
Mitchell J G r304
Mladenova I r303
Mock J J r310
Mogi Y r187
Molina M del C 58 r305
Moll R H r11 r12 r306
Moller G r307
Montalant Y r26 r27
Montanelli C r308
Mookerjee A r471
Moore R 31 r309
Moran L 78
Morgun V V r234
Morris D W 13
Mosely P R r310
Mottinger J P 56 111 112 113 r311 r312
Motto M r63 r308 r484
Moureaux T 72 r232
Mozgova E A r303
Mubumbila M r58 r512 r513
Muchena S C r313 r314
Mucke H r512 r513
Mukherjee B K 84
Muldoon J F r110 r242 r315 r316 r478
Muller-Neumann M 33 112 r30 r317
Munoz-Orozco A r318
Murakami K-I r533
Murashige T 74
Muskavitch K M T r44 r45
Muzilli O r332
Mynbaev T T r395
Nagy J r319
Nakagawa H r320
Narang R S r265
Naumenko A I r321
Nebiolo C M 78 82
Nelson O E Jr 32 33 106 108 109 r120 r217 r322 r337
Nelson T r323
Nesticky M 114 r324 r325 r330
Neudachin V P r326 r360
Neuffer M G 8 38 42 43 104 108 109 r79
Nevers P 34
Newell C A r99
Newton K J 113 r327 r328 r454 r506
Niesbach U 35
Nirmala A r329
Nitzsche W r537
Nivio A A 72
Novak F J r324 r330
Nuez F 20 22
O'Reilly C 35
Oaks A r320
Oberthur E E 4
Ohmasa M r331
Okabe T r184 r185
Okayama H P 2
Oleson A r291
Oliveira E L r332
Olorode O r118
Onukogu F A r333
Orr E A r44
Ortiz-Cereceres J r318
Orton T J r429
Osorio B r335
Osorio M L r58
Ott L A 87
Ottaviano E 81 110 r6 r38 r334
Ottoboni L M M 100 111 113
Ouattar S r196
Page T r481
Palacios I G 67
Palau J r260 r275
Palii A F r535
Paliwal R L r445
Palmer D M r519
Palmer J D r335 r455
Palmer S E 82 83
Palvolgyi L r469
Paly A F r336
Pan D 106 r337
Pan S r338
Panwar V P S r277 r339 r405
Papp J E T r170
Pareddy D R 73 74
Park W D r88
Parkhurst A M r369
Parsons P A 107
Pascard C r13
Pasztor K r340
Patel K A 95
Patterson E B 87 108
Paulis J W r507
Paulsen M R r341
Payne G A r475
Pe E r6
Pe M E 2
Peacock W J r97 r98 r342 r468 r473
Peairs F B r313
Pedersen K r235 r236 r271 r272
Pedersen W L r197
Pego S E 114 r343
Pencic V r247
Pepo P r340
Pereira A 35
Perkins J M r197
Pernes J r344
Perrino P r345
Pertens E r91 r92
Peschke V M 91
Peterson P A 3 4 5 6 7 33 35 112 r379 r414 r523
Peterson R L r350
Peti J r301
Phillips J C 107
Phillips R L 24 84 87 91 93 94 107 108 r57 r346 r509
Phinney B O r347 r348 r446
Phipps R H r349 r514
Piche Y r350
Pierson E E r351
Pietrzak M r352
Pilet P-E r24 r353
Pilinskaya M A r354
Pinter L r355 r356
Piovarci A r324 r325 r330
Pischedda G 71
Pjon C J r357
Plewa M J r358 r359
Plotnikov V K r360
Podleckis E V r361
Podolskaya A P r362
Poethig R S 86 108 111 r363
Pohlman R F 33 112 r364 r365
Pokhmelnykh G A r366
Polacco M L 38 39 108 109 111 r367
Pollak L M 48 114 r368 r369
Pollmer W G r134
Polowick P L 73 74 r370
Pomeranz Y r371
Poneleit C G r147 r372
Ponomarenko A K r321
Potrykus I 84
Poulle M r320
Preiss J 105
Price T P r90
Pring D R 50 98 114 r257 r286 r408
Prisyazhnaya V G r534
Pryor A J r33 r97 r342
Pueppke S G r373
Pugh B F r389
Puigdomenech P r260 r275
Pupillo P r492
Quiros A C r67
Rabin L B r173
Racchi M L 79 80
Radovic G r247
Raja S 30
Rak-Soltes E r374
Raman K 73
Ramesh S V 52 r375
Rao A V B S r210
Rao K V 51
Rao P N r329
Rapela M A 59 60 61 r376 r377
Rascio N r378
Rauser W E r350
Rawlings J O r475
Reddy A R 52 53 r375
Reddy G M 51
Reddy L 54
Reddy L V r379
Reddy V M r380
Reed G L r157 r158
Rees C A 76 77
Reeves R G 26 101
Reger B J r175 r176
Reidinger R F r281
Reisch B I r77 r400
Rendel J M 107
Rhoades M M 3 91 r381
Rhodes A M r298 r299
Rhodes L L r382
Ricci D r51
Rice T B 84
Riedel G r78
Riley G J P r383
Robacker D C r128
Robarts M 84
Robbins J C r489
Robbins W A Jr r384
Roberts J K M r385 r386
Roberts S C r259
Robertson D S 4 5 8 9 10 11 13 14 15 16 18 35 54 85 86 96 108 109 110 111 112 113 r387 r388
Rocheford T R 57 58 112 114
Rockwell B H 78
Rodermel S R r44 r45 r87
Rodriguez J G r147
Rojanaridpiched C r389
Romheld V r390
Rose K R r408
Rosenkranz E 114 r391
Rosic K r190
Rossman E C r166
Rotar A I r336
Rubenstein I 94 r88 r224 r283 r291
Ruget F r46 r47
Russ V J r194
Russell W A r192 r273 r274 r392 r393 r489
Russell W K r394
Ryadchikov V G r360
Rymzhanova R S r395
Saccomani M r396
Sachs M M 34 r97 r342
Sack F D r397
Saedler H 35 r152 r153 r414 r427 r523
Sagi F r469
Saha B C 84

Salamini F r18 r37 r135 r308 r441 r442 r480
Salerno J C 25 26
Salin M L r168
Sanchez de Jimenez E 50 r398 r399
Sandie T r506
Sanford J C r77 r400
Sangeetha H G 53
Santos J P r401
Santos M A r402 r479
Sapre A B r403
Sarantoglou V r512
Sari Gorla M 81
Sarma J S P 86 r404
Sarup P r114 r277 r405 r406
Satyanarayana K V r524
Sauer D B r251
Saunders 51
Sawhney J S r265
Saxena V K r209
Scandalios J G 87 r69 r70 r407
Schaefer V G r165
Schall R A r197
Schantz R r44
Schardl C L 113 r408
Scheffler B E 4 5 108 111
Schliesing T G r409
Schmidt W r410
Schnable P S 4 5 6 7 112
Schnarrenberger C r282
Schultz G A r170
Schuster A M 113 r411
Schuster M L r412
Schwartz D 23 24 r9 r10 r342 r413 r468
Schwarz-Sommer Z 32 33 35 112 r414 r427
Schwarz-Sommer r52
Schweppenhauser M A r314
Scott G E 114 r3 r251 r391 r415 r416 r417
Scowcroft W R r418
Sebestyen E r419
Sederoff R R 49 r71 r249 r420
Selden R r512 r513
Senn A r353
Shadley J D 44 45 108
Shah D M 114 r421
Shamina Z B r226 r227
Shamsnakhmetova F Sh r191
Shannon J C r422
Shardl C L r257
Sharma A K 86 r404
Sharma S R r423
Sharma T R r424
Shaver D 113 r425
Shaw J R r426
Shehata A I r144
Shekhawat N S r28
Shepherd N S 35 114 r152 r153 r427 r523
Sherchan K r428
Sheridan W F 8 104 108 111
Shevtsova V V r202
Shewry P R r297
Shields C R r429
Shiga T r186
Shigaeva M Kh r354
Shimamoto K r430
Shumny V K r366
Siddiqui K H r277 r339 r405 r406
Sidorov A N r431
Silva J r432
Singh A 98
Singh M 40
Singh T r433
Singleton W R 24
Sisco P H 108 113 r248 r411 r434
Skadsen R W r407
Skoog F 74
Skretkowicz A L r435
Slvolpa Y M r436
Smith A G 50
Smith C C r412
Smith C R Jr r147
Smith D J r412
Smith J A r88 r437
Smith J B r31
Smith J D 31 32 r130 r309
Smith J S C 111 112 113 r438 r439
Smith O S r440
Smith R E 76
Soave C 110 r38 r135 r308 r441 r442
Sommer H r523
Song Y-C r443
Sorenson J C r444
Sorrentino J 86 111
Souciet G r512
Spagnoletti Zeuli P L r62
Spiker S 82
Sprague E W r445
Sprague G F r288 r341
Spray C r348 r446
Springer B 33
Stamp P r307 r447 r448 r449 r472
Stanghellini M A r492
Starlinger P 32 33 34 r30 r107 r108 r109 r124 r317 r450 r451 r452 r511
Steffensen D M 100 108
Stehn R A r105
Steinmetz A A r44 r45 r58 r223 r513
Stern D B 113 r453 r454 r455
Stern H 81
Stevenson K R r318
Stiborova M r456 r457
Stienstra W C r462
Stinard P S 8 108 111
Stinson J R 2
Stirdivant S M r44
Stolte D r458
Stout J T 81
Straub R W r459
Strauss A r460
Streitenberger M E 67 68
Strikis P C r500
Strittmatter G r461
Stromberg E L r462
Struik P C r463
Stuber C W 40 88 90 109 r104 r394 r429 r439
Stucker R E 93
Styer C H r464
Styer R C r465 r466
Styles E D 40 101 r68
Subramanian A r45
Sugimoto Y r182
Sukhorzhevskaya T B r303
Suksayretrup K r215
Sumner D D r467
Suprasanna P 51
Susani M r283
Sutton W D 33 111 r342 r468
Suyemoto M M r397
Suzuki Y r220
Swanson J r23 r32
Szaniel I r469
Sze H r178 r213
Szolics I M r467
Talbot D R r23
Tautvydas K J 81
Taylor L P 96 113
Taylor V N r525
Taylor W C r32 r159 r164 r284 r285 r323 r470
Teas H J 40
Tepperman J r267
Tewary B K r471
Theres K r451
Thiraporn R r449 r472
Thirion J P r473
Thomascompton M r368
Thompson D L 114 r80 r251 r474 r475
Thompson D V r23
Thompson J N 107
Thompson W F r335
Thurtell G W r110 r318 r435
Tillmann E r107 r109 r124 r451 r452
Timmis J N r476
Timoschenko A S r73
Timothy D H 49 r249
Ting Y C 29 30
Todorova L r15
Toldi E T r477
Tollefson J J r489 r490
Tollenaar M r315 r478
Tonelli C 79
Toptikov V A r436
Torne J M r402 r479
Torrent M r260
Torti G r480
Toth R r481
Tozawa H r482
Tracy W F 68 108
Tran Huu Dau M E r13
Trapani N r160 r483 r484
Traylor D D r371
Troyer A F 48 r485
Truongandre I r486
Tsaftaris A S r407
Tsai C Y r243 r244 r487
Tsai C-L r488
Tseng C T 114 r489 r490
Tsyganash V I r535
Tucic B r374
Ulmer G r295
Ulrich V 82 83
Umbeck P F 50 98
Usuda H r491
Valenti V r492
Valois A C C r493 r494 r495
Vanduinen B r315
Vargas M r399
Vasil I K r496 r497 r498 r499
Vasil V r497 r498 r499
Vasilas B L r48
Vedel F r502
Velazquez A G r65
Vencovsky R r302 r495
Viani I 79
Vidal B de C r500
Vidaver A K 57 58
Vieira J r224
Vierling R 1
Vilella F r133 r160
Villa M r334
Villegas M, E r67
Vincourt P r27 r501
Vitale A r135
Vuillaume E 114 r502
Vyskubenko I F r354
Waddington C H 107
Wagner E D r359
Waiss A C r173
Walbot V 10 13 96 98 114 r80 r385 r386 r503 r504 r505 r506
Walden D B 74 75 76 77 78
Wall J S r507
Walton J D r508
Wang A S r346 r509
Ward E J 45
Ward J P r31
Warren H L r251 r487
Wassom C E r215 r428
Watson J C r335

Weber D F 44 45 106 108 111 112 113 r510
Weck E 112 r107 r124 r451 r452 r511
Weil J H 113 r58 r512 r513
Weissbach H r8 r43
Welch L F r48
Weller R F r514
Wellhausen E J 28
Wemmer D r386
Wen Z-M r515
Wendel J F 88 90 108 109 110 111 112 113
Wenzel G r516
Werr W 32 33 112 r107 r124 r451 r452
Wessel-Beaver L r517
West D P 87 107 113
West D R r251
Weydemann U 35
Whalen R H 86 108 109
Wheeler H r167
White D G r341
White R P r287 r518
Whitney D A r428
Widholm J M 40
Widstrom N W 111 112 114 r250 r251 r519 r520 r521 r522 r530
Wienand U 35 r414 r427 r523
Williams B G 2
Williams W M 113 r524
Williams W P r168 r525 r526
Willing R P 2 r279
Wilson C M 99 r527
Wilson D M r521 r522
Wilson D R 114 r235 r236 r271 r272 r528
Wilson R L r529
Windels C E r462
Wise R P 50 114
Wiseman B R r530
Witkin E M 105
Wittenbach V A r86
Wojtowicz B r352
Woronecki P P r105 r281
Wreschner D H 76
Xiao C-M r279
Yamada M r186 r531 r532 r533
Yanez L r399
Yoder J 33 r30 r317
Younis M A r22
Yu M H 32
Yurku A I r534 r535
Zabulionis R B 78
Zaitlin M r434
Zeleneva I V r536
Zenkteler M r537
Zhang X Q r151
Zhao J-P r91 r92
Zhou D r538
Zhu Y S r44
Zhurba G M r188
Zolotov V I r321
Zuber M S 48 r55 r82 r173 r203 r251 r316 r475
Zuker 51
Zurawski G r539

COMPILATION OF B-A TRANSLOCATION DATA

The following tables, divided by chromosome or chromosome arm, present the current information available on the expression of various genes in the hypoploid condition (i.e., whether they are uncovered or not). Genes on the linkage map along with genes placed in regions of the map (included within brackets) are listed first, across the top of each table. Following the double slash are additional genes reportedly on that arm. If both arms are represented in the table, those genes known only to be on the short arm are listed first, followed by a "//", then the genes on the short arm, the genes on the long arm, a "//", and finally those genes known only to be on the long arm. The centromere is represented by the symbol "-O-". Documenting references are included. The sources of information were the compilation of Coe and fellow cooperators in 1978 (MNL52:129-145) followed by Zealand and the symbol index in each successive newsletter. I must thank Ed Coe for diligently including Zealand and symbol indexes in each issue. Without this information, compilations such as this one would be extremely time consuming, if not impossible. For each TB, if the gene has been stated to be uncovered by that TB, the gene symbol is listed under the gene. If the gene has been tested and shown not to be uncovered, a "+" is listed. A blank space means no information was found concerning that gene and TB.

I am sure there is a wealth of additional information concerning the B-A translocations which would help to fill in the various gaps in the tables. Toward the accumulation of such data, a short note either directed to me (as a personal communication) or sent for inclusion in the newsletter [such one or two sentence articles are more than welcome (Coe, pers. comm.)] would be adequate to substantiate the data. All of the tables are generated from raw data files by computer programs I wrote, so the addition of new information is very easy.

Dave Hoisington

ABBREVIATIONS USED IN THE TABLES:

MNL : Maize Genetics Newsletter
MBR : Maize for Biological Research
TAG : Theoretical and Applied Genetics
pc : personal communication

CHROMOSOME 1S

| | sr1 | [Les2] | vp5 | zb4 | ms17 | ts2 | [ms9] | p1 | dek1 | as1 | [ms14 | rs1] | -0- | // | Car1 | Cat2 | ct2 | hcf3 | lls1 | nec2 | ys2 | REFERENCES |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| TB-1Sb-2Lc (1S.05-.77,2L.33)Df | | | vp5 | | | | | | | | | | | // | | | | | | | | 11 |
| TB-1Sb (1S.05)Df | sr1 | | vp5 | | | | | | dek1 | + | | + | | // | | Cat2 | ct2 | hcf3 | lls1 | nec2 | ys2 | 1 2 3 4 5 6 7 8 9 10 |
| TB-1Sb-2L4464 (1S.05-.53,2L.28)Df | | | vp5 | | | | | | | | | | | // | | + | | hcf3 | | | | 11 5 7 |
| TB-1La (1L.20)Df | | | | | | | | | | | | | | // | | | + | | | + | | 2 |
| TB-1Lc | | | | | | | | | | | | | | // | | | + | | | + | | 12 |

REFERENCES


1. Robertson, Genetics 40:745, 1955
2. Beckett, MNL49:130-134, 1975
3. Leto, etal, MNL53:36, 1979
4. Dooner, MNL54:80, 1980
5. Roupakias, etal, TAG 58:211-218, 1980
6. Pogna, etal, MNL56:154, 1982
7. Leto, MBR 317-325, 1982
8. Hoisington, 1985(pc)
9. Curtis, 1985(pc)
10. Beckett, 1985(pc)
11. Rakha & Robertson, Genetics 65:223-240, 1969
12. Beckett, MNL49:131


CHROMOSOME 1L

| | -0- | hm1 | [Amp2] | br1 | Vg1 | f1 | [Amp1 | Mdh4 | mmm1 | id1 | v22] | an1 | bz2 | ad1 | Prot1 | [Pgm1 | Adh1 | Kn1 | lw1 | tb1] | D8 | gs1 | Phi1 | Gdh1 | vp8 | Ts6 | bm2 | [alh1] | // | br2 | dek2 | dek22 | Dia2 | hcf2 | rd1 | REFERENCES |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| TB-1La (1L.20)Df | | hm1 | | br1 | | | | Mdh4 | mmm1 | id1 | | an1 | bz2 | | | | | | lw1 | | | gs1 | | | | | bm2 | | // | br2 | dek2 | | | hcf2 | | 1 2 3 4 5 6 |
| TB-1Lc | | | | br1 | | f1 | | | | id1 | | an1 | bz2 | | | | | | lw1 | | | gs1 | | | | | bm2 | | // | br2 | | | | | | 11 |
| TB-1La-5S8041 (1L.20-.80,5S.10)Df | | | | | | | | Mdh4 | mmm1 | | | | bz2 | | | | + | | | | | | | | | | | | // | | | | | hcf2 | | 8 10 3 5 4 |
| TB-1La-3L5242 (1L.20-.90,3L.65)Df | | | | | | | | | mmm1 | | | | | | | | Adh1 | | | | | | | | | | | | // | | | | | | | 3 8 |
| TB-1La-3L5267 (1L.20-.72,3L.73)Df | | | | | | | | | mmm1 | | | | bz2 | | | | + | | | | | | | | | | | | // | | | | | | | 9 3 7 |
| TB-1Sb (1S.05)Df | | + | | | | | | | | | | | | | | | | | | | | | | | | | + | | // | | | | | | | 1 2 |
| TB-1La-3L4759-3 (1L.20-.39,3L.20)Df | | | | | | | | | + | | | | + | | | | | | | | | | | | | | | | // | | | | | | | 3 7 |
| TB-1La-3Le (1L.20-.58,3L.45)Df | | | | | | | | | + | | | | + | | | | | | | | | | | | | | | | // | | | | | | | 3 7 |
| TB-1La-4L4692 (1L.20-.46,4L.15)Df | | | | | | | | | + | | | | | | | | | | | | | | | | | | | | // | | | | | | | 3 |

REFERENCES


1. Roman & Ullstrup, Agron. J. 43:450-454, 1951
2. Beckett, MNL49:130-134, 1975
3. Newton, MNL53:19, 1979
4. Leto, MBR 317-325, 1982
5. Newton, MNL54:14, 1980
6. Neuffer & Sheridan, MNL55:29, 1981
7. Newton & Birchler, MNL54:14, 1980
8. Birchler, Genetics 94:687-700, 1980
9. Birchler, MNL52:29, 1978
10. Robertson, MNL49:80, 1975
11. Beckett, MNL49:131

CHROMOSOME 2S AND 2L

| | dek3 | Dia1 | // | ws3 | al1 | [Y3] | lg1 | gl2 | [Mut | nec4] | d5 | B1 | [gl11] | gs2 | sk1 | [Les1] | wt1 | [lte1 | wn1] | fl1 | ts1 | [ba2] | -O- | v4 | [Sks1] | w3 | Ht1 | Ch1 | // | dek4 | dek16 | dek23 | hcf1 | Rf3 | whp1 | REFERENCES |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| TB-3La-2S6270<br>(3L.10-.60,2S.46)Df | dek3 | | // | | al1 | | lg1 | gl2 | | nec4 | d5 | | gl11 | + | sk1 | | wt1 | | | | | | | | | | | | // | | | | | | | 5 1 2 6 |
| TB-1Sb-2L4464<br>(1S.05-.53,2L.28)Df | | | // | | | | + | + | | | | | | | + | | | | + | | | | | v4 | | w3 | | | // | dek4 | dek16 | | hcf1 | | | 1 2 3 4 |
| TB-3La-2L7285 | | | // | | | | + | + | | | | | | | + | | | | | | | | | v4 | | w3 | | | // | | | | | | whp1 | 5 1 7 |
| TB-1Sb-2Lc<br>(1S.05-.77,2L.33)Df | | | // | | | | | | | | | | | | | | | | | | | | | + | | w3 | | | // | | | | hcf1 | | | 1 3 |

REFERENCES


1. Beckett, MNL49:130-134, 1975
2. Neuffer & Sheridan, MNL55:29, 1981
3. Leto, MBR 317-325, 1982
4. Sheridan, etal, MNL58:90, 1984
5. Rakha & Robertson, Genetics 65:223-240, 1970
6. Hoisington, 1984(pc)
7. Modena, MNL57:39, 1983

CHROMOSOME 3S

| | cr1 | d1 | [ra2 | rt1] | cl1 | [Lg3 | Rg1 | Rf1 | ys3] | -0- | // | brn1 | dek5 | E3 | E4 | E8 | Hex1 | g2 | REFERENCES |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| TB-3La (3L.10)Df | | | + | | + | | | | ys3 | | // | | | | | | | | 1 7 |
| TB-3Sb | cr1 | d1 | ra2 | | cl1 | | | | + | | // | brn1 | dek5 | | | E8 | Hex1 | g2 | 1 2 3 4 5 6 |
| TB-3Ld | | | + | | | | | | | | // | | | | | | | | 1 |


REFERENCES

1. Beckett, MNL49:130-134, 1975
2. Neuffer & Sheridan, MNL55:29, 1981
3. Newton, etal, MNL56:154-155, 1982
4. Whalen, MNL57:20, 1983
5. Stinard, MNL59:7-8, 1985
6. Wendel, etal, MNL59:88, 1985
7. Beckett, MNL58:73-74, 1984


CHROMOSOME 3L

| | -0- | gl6 | [Rp3] | ts4 | [pm1 | vp1 | Spc1 | Tpi4] | ig1 | lg2 | ba1 | [Pgd2 | yd2] | na1 | Got1 | Me1 | a3 | Mdh3 | a1 | sh2 | et1 | ga7 | // | dek6 | dek17 | y10 | REFERENCES |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| TB-3La (3L.10)Df | | gl6 | | ts4 | pm1 | vp1 | | | | lg2 | ba1 | | yd2 | na1 | | | a3 | Mdh3 | a1 | sh2 | et1 | | // | dek6 | dek17 | y10 | 4 3 5 6 7 8 9 10 |
| TB-3Lf | | gl6 | | | pm1 | | | | | lg2 | ba1 | | | na1 | | | | | a1 | sh2 | et1 | | // | | | y10 | 7 10 |
| TB-3Lg | | gl6 | | | pm1 | vp1 | | | | lg2 | ba1 | | yd2 | | | | | | a1 | sh2 | et1 | | // | | | | 7 9 10 |
| TB-3Ld | | + | | + | + | vp1 | | | | lg2 | ba1 | | | na1 | | | | Mdh3 | a1 | sh2 | et1 | | // | dek6 | | y10 | 3 2 12 7 9 10 |
| TB-3Li | | + | | | + | + | | | | lg2 | ba1 | | yd2 | na1 | | | | | a1 | sh2 | et1 | | // | | | y10 | 7 9 10 |
| TB-3Lj | | + | | | | + | | | | lg2 | ba1 | | yd2 | na1 | | | | | a1 | sh2 | et1 | | // | | | | 7 9 |
| TB-3Lc | | + | | + | + | + | | | | + | ba1 | | yd2 | na1 | | | | Mdh3 | a1 | sh2 | et1 | | // | | | y10 | 3 2 12 7 9 10 |
| TB-3Lh | | + | | | | vp1 | | | | lg2 | | | yd2 | + | | | | | a1 | sh2 | et1 | | // | | | y10 | 7 9 10 |
| TB-3Lk | | + | | | + | + | | | | + | | | | | | | a3 | | a1 | sh2 | et1 | | // | | | | 7 9 10 |
| TB-3Ll | | + | | | + | + | | | | + | + | | | + | | | a3 | | a1 | sh2 | et! | | // | | | | 7 9 10 |
| TB-1La-3L5267 (1L.20-.72,3L.73)Df | | | | | | | | | | | | | | | | | | Mdh3 | a1 | | | | // | | | | 1 2 |
| TB-3Lm | | + | | | + | + | | | | + | | | | | | | | | a1 | sh2 | et1 | | // | dek6 | | | 7 9 10 |
| TB-3Sb | | + | | + | | | | | | + | | | + | | | | | | | | | | // | | | | 3 |
| TB-3La-2S6270 (3L.10-.60,2S.46)Df | | | | | | | | | | | | | | | | | | | + | + | | | // | | | | 11 |


REFERENCES

1. Birchler, MNL52:33, 1978
2. Newton, MNL53:21, 1979
3. Beckett, MNL49:130-134, 1975
4. Robertson, Genetics 40:745-760, 1955
5. Newton & Schwartz, Genetics 95:425-442, 1980
6. Neuffer & Sheridan, MNL55:29, 1981
7. Beckett, MNL58:73-74, 1984
8. Sheridan, etal, MNL58:98, 1984
9. Scheffler & Peterson, MNL59:4, 1985
10. Beckett, MNL59:39, 1985
11. Beckett & Coe, MNL52:131, 1978
12. Beckett, MNL55:27, 1981

CHROMOSOME 4S

| | Ph1 | Asr1 | Rp4 | Ri1 | Ga1 | [Zp6] | Adh2 | [Zp22 | Zp27 | Zp28 | Zp30] | Ts5 | la1 | fl2 | [Zp14] | st1 | su1 | bt2 | [bm3 | Dt6 | v23 | Ysk1 | Zp12] | -D- | // | Aco1 | bx1 | dek7 | REFERENCES |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| TB-4Sa (4S.25)Df | | | | | | | | | | | | | la1 | | | st1 | su1 | bt2 | + | | | | | | // | | bx1 | dek7 | 1 3 4 5 |
| TB-1La-4L4692 (1L.20-.46,4L.15)Df | | | | | | | | | | | | | | | | | + | | + | | | | | | // | | | | 1 2 |
| TB-4Lb | | | | | | | | | | | | | | | | | | | + | | | | | | // | | | | 5 |
| TB-4Ld | | | | | | | | | | | | | | | | | | | + | | | | | | // | | | | 5 |
| TB-4Le | | | | | | | | | | | | | | | | | | | + | | | | | | // | | | | 5 |
| TB-4Lf | | | | | | | | | | | | | | | | | | | + | | | | | | // | | | | 5 |
| TB-7Lb-4L4698 (7L.30-.74,4L.08)Df | | | | | | | | | | | | | | | | | | | + | | | | | | // | | | | 5 |
| TB-9Sb-4L6504 (9S.40-.68,4L.03)Df | | | | | | | | | | | | | | | | | + | | + | | | | | | // | | | | 1 5 |
| TB-9Sb-4L6222 (9S.40-.68,4L.03)Df | | | | | | | | | | | | | | | | | + | | | | | | | | // | | | | 1 5 |

REFERENCES


1. Beckett, MNL49:130-134, 1975
2. Beckett, MNL58:74, 1984
3. Neuffer & Sheridan, MNL55:29, 1981
4. Simcox & Weber, MNL57:107-108, 1983
5. Beckett, MNL58:73-74, 1984


CHROMOSOME 4L

| | -D- | zb6 | [lw4] | gl4 | [Zp10 | Zp15 | v8] | Tu1 | j2 | gl3 | [o1] | c2 | dp1 | // | dek8 | dek10 | REFERENCES |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| TB-1La-4L4692 (1L.20-.46,4L.15)Df | | | | gl4 | | | | | | gl3 | | c2 | dp1 | // | | | 1 2 3 |
| TB-4Lb | | | | gl4 | | | | | | gl3 | o1 | c2 | dp1 | // | | | 5 4 |
| TB-4Lc | | | | gl4 | | | v8 | | | gl3 | o1 | c2 | dp1 | // | | | 4 |
| TB-4Lf | | | | gl4 | | | | | | gl3 | o1 | c2 | dp1 | // | dek8 | dek10 | 6 4 7 |
| TB-9Sb-4L6504 (9S.40-.68,4L.03)Df | | | | gl4 | | | | | | gl3 | o1 | c2 | dp1 | // | | | 1 2 4 |
| TB-9Sb-4L6222 (9S.40-.68,4L.03)Df | | | | gl4 | | | | | | gl3 | o1 | c2 | dp1 | // | | | 1 2 4 |
| TB-4Le | | | | | | | v8 | | | gl3 | o1 | c2 | dp1 | // | | | 4 |
| TB-4Ld | | | | | | | | | | gl3 | o1 | c2 | dp1 | // | | | 4 |
| TB-7Lb-4L4698 (7L.30-.74,4L.08)Df | | | | + | | | | | | + | + | c2 | dp1 | // | | | 1 2 4 |
| TB-4Sa (4S.25)Df | | | | + | | | | | | + | | + | + | // | | | 2 4 |

REFERENCES


1. Rakha & Robertson, Genetics 65:223-240, 1970
2. Beckett, MNL49:130-134, 1975
3. Beckett, MNL58:74, 1984
4. Beckett, MNL58:73-74, 1984
5. Beckett, MNL56:47, 1982
6. Neuffer & Sheridan, MNL55:29, 1981
7. Sheridan, etal, MNL58:98, 1984

CHROMOSOME 5S

| | Pgm2 | Mdh5 | [Amy2 | Cat1] | am1 | lu1 | gl17 | a2 | [Amp3 | Got3] | vp2 | ps1 | bm1 | -0- | // | dek18 | ms13 | nec3 | REFERENCES |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| TB-5Sc | | | | | | | gl17 | a2 | | | | | | | // | | | | 6 |
| TB-1La-5S8041 (1L.20-.80,5S.10)Df | | Mdh5 | | | | | | a2 | | | vp2 | ps1 | bm1 | | // | dek18 | | nec3 | 1 2 3 4 5 |
| TB-5La (5L.10)Df | | | | | | | | | | | | | + | | // | | | + | 7 5 |
| TB-5Lb | | | | | | | | | | | | | + | | // | | | | 7 |

REFERENCES

1. Robertson, MNL49:80, 1975
2. Robertson, MNL50:71, 1976
3. Newton, MNL54:14, 1980
4. Sheridan, etal, MNL58:98, 1984
5. Shadley & Weber, MNL58:160-161, 1984
6. Beckett, MNL56:47, 1982
7. Beckett, MNL49:130-134, 1975

CHROMOSOME 5L

| | -0- | bt1 | [na2 | ms5] | v3 | bv1 | ga2 | ae1 | pr1 | gl8 | [lw2] | ys1 | [v12] | Got2 | v2 | [lw3 | yg1 | zb3] | // | dek9 | eg1 | mep1 | td1 | REFERENCES |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| TB-5La (5L.10)Df | | + | + | | + | bv1 | | ae1 | pr1 | gl8 | lw2 | ys1 | v12 | | v2 | | yg1 | | // | dek9 | eg1 | mep1 | + | 1 2 3 |
| TB-5Lb | | + | + | | + | bv1 | | ae1 | pr1 | | | | | | v2 | | yg1 | | // | | eg1 | | + | 1 |
| TB-5Ld | | + | | | | bv1 | | ae1 | pr1 | gl8 | lw2 | | | | | | | | // | | | | + | 4 |

REFERENCES

1. Beckett, MNL49:130-134, 1975
2. Schwartz, MGG 174:233-241, 1979
3. Neuffer & Sheridan, MNL55:29, 1981
4. Beckett, MNL59:39, 1985

CHROMOSOME 6S AND 6L

| | dek19 | rhm1 | // | po1 | rgd1 | [l11 | oro1] | -O- | [Pgd1] | w15 | l12 | Y1 | l10 | si1 | [Enp1 | ms1 | pb1 | pb4 | wi1] | l15 | pg11 | Dt2 | Pl1 | Bh1 | su2 | sm1 | Pt1 | [Hex2] | py1 | w14 | [w1 | Idh2 | Mdh2] | // | rd2 | REFERENCES |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| TB-6Sa (6S.50)Df | | | // | po1 | + | | | | | | | | | | | | | | | | | | | | | | | | | | | | | // | | 1 2 |
| TB-6Lc | dek19 | | // | + | + | | | | + | w15 | l12 | | | | Enp1 | | | | | | | | Pl1 | | su2 | | | Hex2 | py1 | | | Idh2 | Mdh2 | // | | 1 4 3 5 6 |
| TB-6Lb | | | // | | | + | + | | | + | + | | + | | | + | | | + | | | | + | | + | + | | | py1 | w14 | w1 | | Mdh2 | // | | 1 3 |

REFERENCES


1. Beckett, MNL49:130-134, 1975
2. Beckett, 1985(pc)
3. Newton, MNL54:13-14, 1980
4. Newton, MNL53:20, 1979
5. Sheridan, etal, MNL58:98, 1984
6. Wendel, etal, MNL59:89-90, 1985


CHROMOSOME 7S AND 7L

| | [l13 | Zp6] | Hs1 | [w17] | Zp21 | Zp29 | o2 | y8 | in1 | v5 | vp9 | [cp1 | cp2 | w16] | -O- | [ms7 | o5 | wyg1 | Zp1 | Zp2 | Zp3 | Zp16] | ra1 | gl1 | Tp1 | sl1 | ij1 | [va1] | Bn1 | bd1 | Pn1 | // | dek11 | Dt3 | E1 | REFERENCES |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| TB-7Sc | | | | | | | | | | | vp9 | | | | | | | | | | | | | | | | | | | | | // | | | | 1 |
| TB-7Lb (7L.30)Df | | | | | | | + | | | + | + | | | | | ms7 | o5 | | | | | | ra1 | gl1 | | | ij1 | va1 | | | | // | dek11 | | | 2 3 |
| TB-7Lb-4L4698 (7L.30-.74,4L.08)Df | | | | | | | | | | | | | | | | | | | | | | | | gl1 | | | | | | | | // | | | | 4 |

REFERENCES


1. Beckett, 1985(pc)
2. Beckett, MNL49:130-134, 1975
3. Neuffer & Sheridan, MNL55:29, 1981
4. Rakha & Robertson, Genetics 65:223-240, 1970

CHROMOSOME 8S AND 8L

| | [Bif1] | -O- | fl3 | [pro1] | v16 | ms8 | j1 | // | dek20 | el1 | gl18 | Idh1 | Mdh1 | nec1 | Tpi3 | v21 | REFERENCES |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| TB-8La (8L.70)Df | | | | + | v16 | ms8 | j1 | // | | | + | | | + | | v21 | 1 2 3 |
| TB-8Lb | | | | | v16 | ms8 | j1 | // | | | | | | | | v21 | 1 |
| TB-8Lc | | | | pro1 | | | | // | dek20 | el1 | | Idh1 | + | | | v21 | 3 4 5 6 |

REFERENCES


1. Beckett, MNL49:130-134, 1975
2. Gavazzi & Todesco, MNL53:63, 1979
3. Beckett, MNL56:47, 1982
4. Curtis, MNL57:31-32, 1983
5. Goodman, etal, MNL57:131, 1983
6. Sheridan, etal, MNL58:98, 1984


CHROMOSOME 9S AND 9L

| | dek12 | // | Dt1 | yg2 | [Dt5 | pyd1 | wd1] | c1 | sh1 | bz1 | [16] | 17 | [Mr] | lo2 | [ga8] | wl1 | wx1 | [Atc1 | Rf2] | d3 | -O- | pg12 | ar1 | v1 | ms2 | gl15 | bk2 | [hm2] | Wc1 | Bf1 | bm4 | // | dek13 | REFERENCES |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| TB-9Sb-4L6504 (9S.40-.68,4L.03)Df | | // | | yg2 | | | | | sh1 | | | | | | | | | | | | | | | | | | | | | | | // | | 6 |
| TB-9Sb-4L6222 (9S.40-.68,4L.03)Df | | // | | yg2 | | | | | sh1 | | | | | | | | | | | | | | | | | | | | | | | // | | 6 |
| TB-9Sb (9S.40)Df | dek12 | // | | yg2 | | pyd1 | wd1 | c1 | sh1 | bz1 | 16 | 17 | | lo2 | | | + | | | + | | | | | | | | | | | | // | | 1 2 3 4 5 |
| TB-9Sd | | // | | | | | | | | | | | | lo2 | | | | | | d3 | | | | | | | | | | | | // | | 7 4 |
| TB-9Lc | | // | | | | | | | | | | | | | | | + | | | + | | | ar1 | v1 | ms2 | gl15 | bk2 | | | Bf1 | bm4 | // | dek13 | 1 3 |
| TB-9La (9L.40)Df | | // | | | | | | | | | | | | | | | + | | | + | | + | + | + | + | + | bk2 | | | Bf1 | bm4 | // | | 1 |

REFERENCES


1. Beckett, MNL49:130-134, 1975
2. Robertson, MNL52:134, 1978
3. Neuffer & Sheridan, MNL55:29, 1981
4. Kindiger, 1985(pc)
5. Coe, MNL59:40, 1985
6. Rakha & Robertson, Genetics 65:223-240, 1970
7. Beckett, 1985(pc)

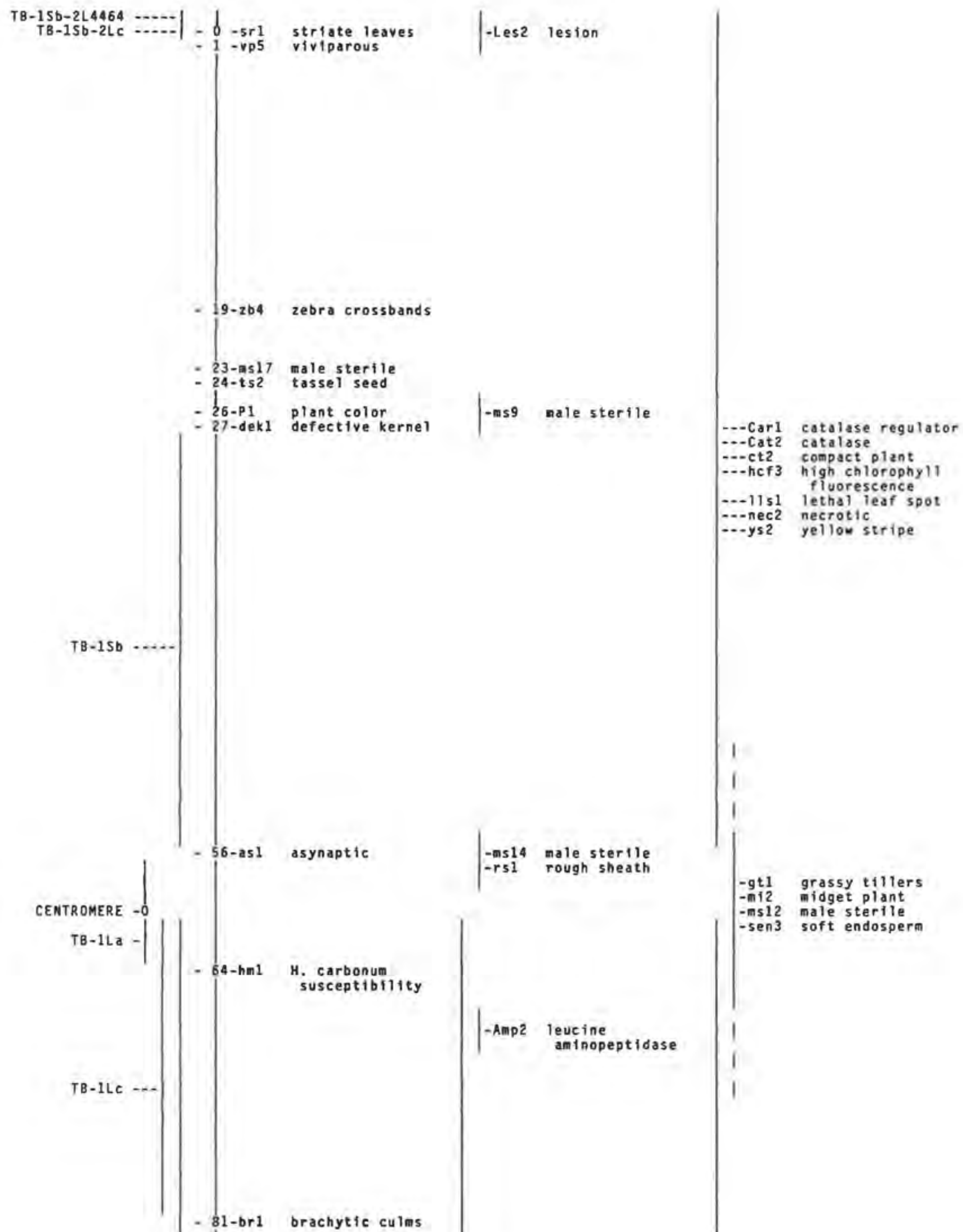
CHROMOSOME 10S AND 10L

| | dek14 | sr3 | // | [Rp5 | Rp6 | Rpp9] | Rp1 | oy1 | Og1 | y9 | -O- | zn1 | du1 | bf2 | [Cx1 | Glu1 | nl1 | ms10 | li1] | Tp2 | g1 | Lte2 | [cm1 | Isr1] | r1 | l1 | Lc1 | Mst1 | w2 | o7 | l13 | sr2 | // | dek15 | dek21 | REFERENCES |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| TB-10Sc | dek14 | sr3 | // | | | | | oy1 | | y9 | | + | + | + | | | | | + | | + | | | | + | | | | | | | | // | | | 1 2 |
| TB-10L18 | | | // | | | | | | | | | zn1 | du1 | bf2 | | | | ms10 | li1 | | g1 | | | | r1 | | | | | | | | // | | | 3 1 4 |
| TB-10L19 | | | // | | | | | | | | | zn1 | du1 | bf2 | | | | ms10 | li1 | | g1 | | | | r1 | | | | | | l13 | | // | dek15 | dek21 | 3 1 4 5 2 6 |
| TB-10L26 | | | // | | | | | | | | | + | du1 | bf2 | | | | | li1 | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L22 | | | // | | | | | | | | | + | + | bf2 | | | | | li1 | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10Lb (10L.34)Df | | | // | | | | | | | + | | + | + | bf2 | | | + | ms10 | li1 | | g1 | | | | r1 | | | | w2 | | | sr2 | // | | | 1 |
| TB-10L1 | | | // | | | | | | | | | + | + | + | | | | ms10 | + | | g1 | | | | r1 | | | | | | | | // | | | 3 4 |
| TB-10L3 | | | // | | | | | | | | | + | + | + | | | | ms10 | + | | g1 | | | | r1 | | | | | | | | // | | | 3 4 |
| TB-10L7 | | | // | | | | | | | | | + | + | + | | | | ms10 | + | | g1 | | | | r1 | | | | | | | | // | | | 3 4 |
| TB-10L8 | | | // | | | | | | | | | + | + | + | | | | ms10 | + | | g1 | | | | r1 | | | | | | | | // | | | 3 4 |
| TB-10L10 | | | // | | | | | | | | | + | + | + | | | | ms10 | + | | g1 | | | | r1 | | | | | | | | // | | | 3 4 |
| TB-10L20 | | | // | | | | | | | | | + | + | + | | | | ms10 | + | | g1 | | | | r1 | | | | | | | | // | | | 3 4 |
| TB-10L25 | | | // | | | | | | | | | + | + | + | | | | ms10 | + | | g1 | | | | r1 | | | | | | | | // | | | 3 4 |
| TB-10L28 | | | // | | | | | | | | | + | + | + | | | | ms10 | + | | g1 | | | | r1 | | | | | | | | // | | | 3 4 |
| TB-10L31 | | | // | | | | | | | | | + | + | + | | | | ms10 | + | | g1 | | | | r1 | | | | | | | | // | | | 3 4 |
| TB-10L36 | | | // | | | | | | | | | + | + | + | | | | ms10 | + | | g1 | | | | r1 | | | | | | | | // | | | 3 4 |
| TB-10L2 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L4 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L5 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L6 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L9 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L11 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L12 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L13 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L14 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L15 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L16 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L17 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L21 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L23 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L24 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L27 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L29 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L30 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L33 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L34 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L35 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L37 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10L38 | | | // | | | | | | | | | + | + | + | | | | | + | | g1 | | | | | | | | | | | | // | | | 3 |
| TB-10La (10L.35)Df | | | // | | | | | | | + | | + | + | + | | | + | + | + | | g1 | | | | r1 | | | | w2 | | | sr2 | // | | | 3 1 |
| TB-10L32 | | | // | | | | | | | | | + | + | + | | | | | + | | + | | | | | | | | | | | | // | | | 3 |

REFERENCES


1. Beckett, MNL49:130-134, 1975
2. Neuffer & Sheridan, MNL55:29, 1981
3. Lin, MNL48:182-184, 1974
4. Lin, MNL54:102, 1980
5. Mascia & Robertson, J Hered 71:19-24, 1980
6. Sheridan, etal, MNL58:98, 1984

CHROMOSOME 1



- 85-Vg1 vestigial glume
- 86-f1 fine stripe

-Amp1 leucine aminopeptidase

TB-1La-3L4759-3 -----
TB-1La-4L4692 -----
TB-1La-3Le -----

---hcf2 high chlorophyll fluorescence

-Mdh4 malate dehydrogenase
-mmm1 modifier of mMDH

-104-an1 anther ear
-106-bz2 bronze
-108-ad1 adherent

-id1 indeterminate growth
-v22 virescent

TB-1La-3L5267 ---
TB-1La-5S8041 ---

---br2 brachytic culms
---dek2 defective kernel
---dek22 defective kernel
---Dia2 diaphorase
---rd1 reduced plant

-121-Prot1 protein

-Pgm1 phosphoglucomutase

-128- Adh1 alcohol dehydrogenase
-128- Kn1 knotted
-128- lw1 lemon white

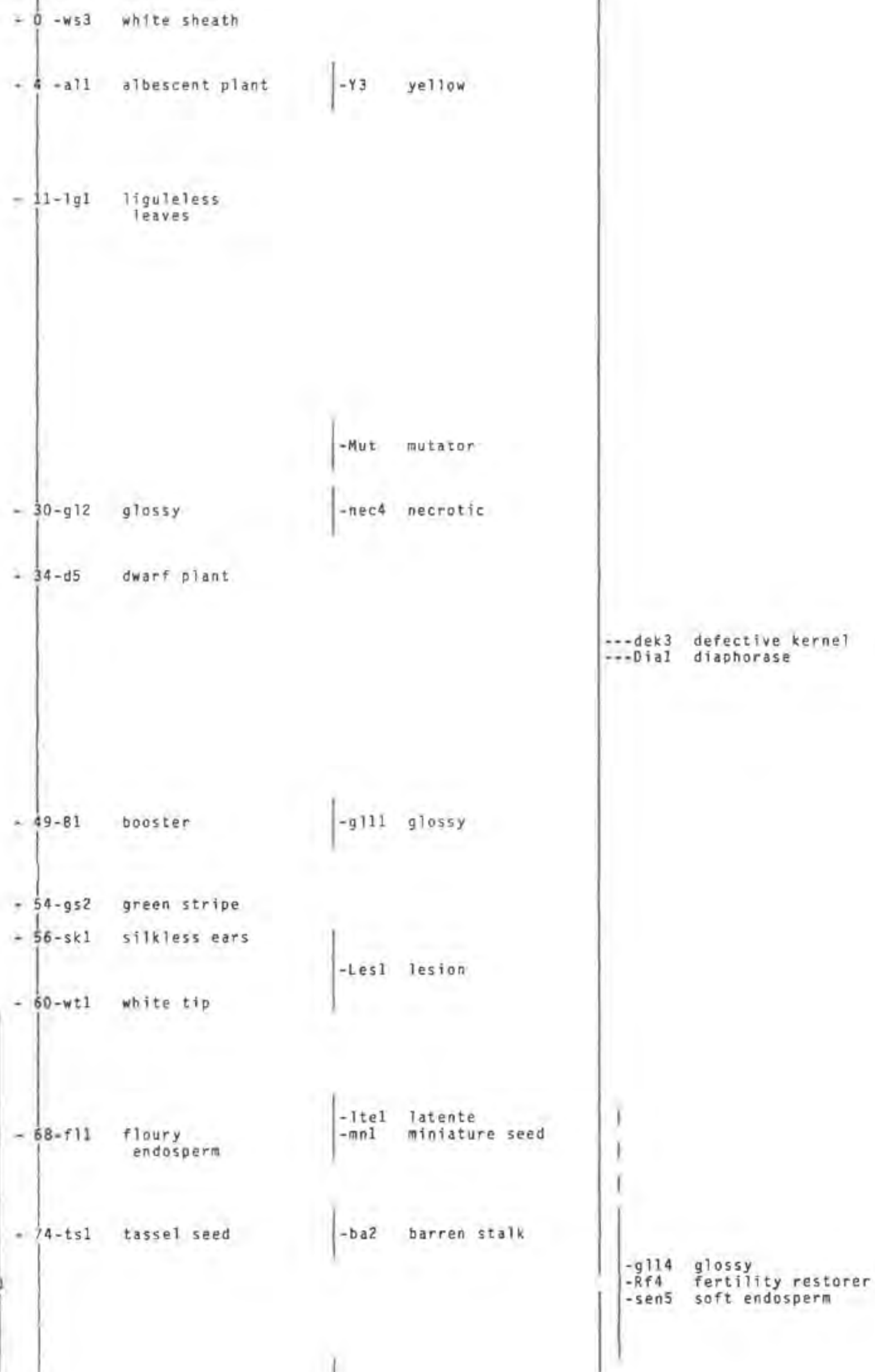
-tb1 teosinte branched

-133-D8 dwarf plant
-135-gs1 green stripe
-140-Phi1 phosphohexose isomerase

TB-1La-3L5242 ----- -145-Gdh1 glutamic dehydrogenase

-154-vp8 viviparous
-158-Ts6 tassel seed
-161-bm2 brown midrib

-alh1 histone Ia


CHROMOSOME 2
0 -ws3 white sheath
4 -al1 albescent plant
-Y3 yellow
11-lg1 liguleless leaves
-Mut mutator
30-gl2 glossy
-nec4 necrotic
34-d5 dwarf plant
---dek3 defective kernel
---Dia1 diaphorase
49-B1 booster
-gl11 glossy
54-gs2 green stripe
56-sk1 silkless ears
-Les1 lesion
60-wt1 white tip
-lte1 latente
-mn1 miniature seed
TB-3La-2S6270 -----
68-fl1 floury endosperm
74-ts1 tassel seed
-ba2 barren stalk
-gl14 glossy
-Rf4 fertility restorer
-sen5 soft endosperm
CENTROMERE -----0
TB-3La-2L7285 -----
TB-1Sb-2L4464 -----

- 83-v4 virescent

-Sks1 sterility suppressor

TB-1Sb-2Lc -----

-111-w3 white

---dek4 defective kernel
---dek16 defective kernel
---dek23 defective kernel
---hcf1 high chlorophyll fluorescence
---Rf3 fertility restorer
---whp1 white pollen

-121-Ht1 H. turcicum resistance

-155-Ch1 chocolate

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 | 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 |