MAIZE GENETICS COOPERATION

NEWS LETTER

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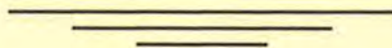

March 1, 1977

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

Department of Agronomy
University of Missouri
Columbia, Missouri

Some sources of general information on maize genetics and cytogenetics:

The Mutants of Maize. M. G. Neuffer, L. M. Jones and M. S. Zuber, Crop Sci. Soc. Am., Madison, Wisc., 1968.

Handbook of Genetics, vol. 2, pp. 3-30. R. C. King, ed., Plenum Press, New York, 1974.

Handbook of Biochemistry and Molecular Biology, 3d edition, vol. II, pp. 833-847. G. D. Fasman, ed., CRC Press, Cleveland, Ohio, 1976.

Maize Research and Breeders Manual No. VIII. C. B. Henderson, Illinois Foundation Seeds, Inc., Champaign, Illinois, 1976.

Chapters on genetics and cytogenetics will be included in Corn and Corn Improvement, 2d edition, G. F. Sprague, ed., Amer. Soc. Agron., 1977 (in press).

Dr. G. P. Redei, organizer of the Stadler Genetics Symposia, calls attention to the 9th Symposium, to be held April 15-16, 1977, at Columbia, Missouri:

J. W. Allen (Children's Hospital, Boston): New Approaches for Extending BrdU-Dye Analysis of DNA Replication and Sister Chromatid Exchange Formation to In Vivo Systems.

Winston J. Brill (University of Wisconsin, Madison): Genetics of Nitrogen Fixation.

Moshe Feldman (Weizmann Institute, Rehovot, Israel): Historical Aspects of the Discovery of Wild Tetraploid Wheat.

J. R. S. Fincham (University of Leeds): Gene-Enzyme Relationships in *Neurospora crassa*.

G. Gavazzi (Università di Milano): The Genetic Complexity of the *R* locus in Maize.

Ruth Kavenoff (University of California, La Jolla): On the Organization of Chromosomal DNA.

Don Riddle (University of Missouri, Columbia): A Genetic Pathway for Dauer Larva Formation in *Caenorhabditis elegans*.

Gary Stein (University of Florida, Gainesville): Chromatin as a Model System for Examining the Regulation of Gene Expression in Mammalian Cells.

J. N. Thompson (University of Oklahoma, Norman): Analysis of Gene Number and Development in Polygenic Systems.

S. G. Wildman (University of California, Los Angeles): Polypeptide Composition of Fraction I Protein as a Diagnostic Aid in the Study of Plant Evolution.

A detailed Program and Proceedings of this Symposium as well as of previous Symposia can be obtained from SGS, 117 Curtis Hall, University of Missouri, Columbia, MO 65201 USA. The price is $5.00 per volume plus 50¢ per order for postage and handling.

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URGENT APPEAL

Several cooperators, spurred by Marjorie Maguire, have expressed concern that systematic mapping of genes and chromosomal features has been advancing recently at about the same pace as speciation. Research (both basic and applied) is hampered by limitations in the markers and maps, and by the absence of consolidated cytological and genetic information in a cytogenetic map.

To get something started, please do the following:

1) Let me know if you will take on part or all of the compiling and synthesis, or if you have a better idea.

2) Include research in your own particular interests that adds to mapping; genetic variations become analyzable and manipulable with the special power and exquisite precision of genetics only after they are located.

3) For the next News Letter, assemble and send your previous or new data, published and unpublished, that unequivocally locate specific genes relative to specific cytological points (for example, tests for uncovering of recessives in hemizygotes, positive and negative results). The items will be tabulated next year in rough form, with citations:

Chromosome/arm	Information & conclusion (Aberration; cytological data; cross)	Cooperator & Reference

I. FOREWORD

Found on a tattered shred of wet-strength kraft paper:

"If phytochrome varies among strains, TB's and marker stocks should show electroph....."

A set of all available copies of the News Letter will be awarded to the first person who finds the rest of this manuscript, with data.

****

Over 680 copies of this volume will be sent to research workers, laboratories and libraries around the world. During the past year we have checked the mailing list by inquiring of some addressees whether they wished to continue to receive it; a few asked to be dropped . . . a number of new addressees have been added.

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Please note the appeal on the opposite page.

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Converged strains of a few laboratories are listed in Section III, in response to a request for listings by cooperators. The availability of such strains and multiple genetic combinations is increasingly important to research; we hope to enhance contacts among potential collaborators by expanding this list in the future as cooperators make known specialized strains in their laboratories.

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The costs of preparation, reproduction and mailing of this News Letter are borne by a grant from the National Science Foundation. We are grateful for this indispensable support.

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A microfilm of volumes 1-29 and 33 is available for $9.50 U.S.; checks should be made payable to E. H. Coe, Jr.

Airmail service to addresses outside the U.S. is expensive; we will send the next issue to you by air if we receive $3.00 from you by January 1, 1978.

Deadline for the next issue is January 1, 1978; please see the back cover.

Corrigenda for the 1961 symbol index are wanted.

Errata are listed on the last page.

****

I wish to thank M. G. Neuffer for help in planning and developing this volume; Karen A. Sheridan for exacting attention to editing, composing and proofing; Sheila McCormick and Marion D. Murray for help with literature; and Mary Nelson for thorough and precise work in producing the final copy.

E. H. Coe, Jr.

II. REPORTS FROM COOPERATORS

BOSTON COLLEGE
Department of Biology, Chestnut Hill, Massachusetts

Synaptonemal complex of the F_1 hybrids of maize x perennial teosinte

Anthers of the F_1 hybrids of maize x perennial teosinte were studied by following the techniques of electron microscopy previously employed by the author (Ting, Chromosomes Today 4, 1973). At pachytene stage it was consistently observed that synaptonemal complex was present. The characteristics of the complex resemble those of both haploid and diploid maize. For instance its diameter, on the average, measured 2200 Å. The three components (two lateral elements and one central element) were discernible. All of them appeared bipartite in organization. No cross-fibers between central and lateral elements were definitely identified. Furthermore it was impossible to distinguish the complexes formed by autosyndesis (teosinte chromosomes synapsed with teosinte chromosomes) from those formed by allosyndesis (teosinte chromosomes synapsed with maize chromosomes). However, contact between the complex and the nuclear envelope was not observed. On several occasions, isolated chromosome cores were found and they are apparently from unpaired chromosomes, either from maize or from teosinte. From diplotene stage to diakinesis, as in both haploid and diploid maize, the complexes gradually disorganized. It seems that this observation provides another evidence that maize and teosinte should be congeneric.

Y. C. Ting

Fine structure of maize anther callus

A few maize anther calli about four weeks old and less than three millimeters in diameter were isolated and fixed in 3% glutaraldehyde for two hours and rinsed and again fixed in 2% osmium tetroxide for one hour. The fixed calli were embedded in Epoxy resin and sectioned with MT-I ultramicrotome. Thin sections were double-stained with uranium and lead. All observations were made with EM Philips 200. The following characteristics of the calli were consistently found:

The nuclei were irregular in shape and varied a great deal in size. Chromatin was condensed and much darkly stained. With a limited amount of material studied, no dividing cells were identified. Hence fine structures of chromosomes were not available for study.

In the nucleoli, fibrous elements, frequently spirally arranged, were apparent. These elements were embedded in a homogeneous granular background which was also heavily stained. No vacuoles were present in the nucleoli.

In the cytoplasm well developed chloroplasts were not observed, but some proplastids. Thylakoids of the proplastids were limited in number, and generally only one or two were found in a single organelle. They were usually appressed to the inner membrane of the proplastids, and left the center devoid of any visible thylakoids. The number and size of proplastids were inconsistent from cell to cell. Mitochondria were abnormal and frequently extended into rod-shape. Some of them were clearly grown out into two or three branches. Cristae or membranes within the mitochondria were plentiful. Occasionally small lipid granules were observed in the matrix. Furthermore endoplasmic reticulum did not have the well-packed membranes. Instead the membranes were dilated and scattered in the cytoplasm. Only a few ribosome granules were attached to the surface of each membrane and most of the other ribosomes were in the hyaloplasm. Elements of microtubules close to the cell periphery were readily identifiable, and most of them were less than 2000 Å in length.

Y. C. Ting and R. McLellan

COMMONWEALTH SCIENTIFIC AND INDUSTRIAL RESEARCH ORGANIZATION
Division of Plant Industry, Canberra, Australia

Glutamic dehydrogenase (GDH) in maize

The role of GDH in nitrogen assimilation is not certain in plants, and this has stimulated our interest in this enzyme system. Previous work (starch gel electrophoresis) has shown that three isozymes are specified by alleles of the gene Glutamic dehydrogenase: Gdh-F, Gdh-N and Gdh-S. Field observations and growth chamber experiments suggested that plants carrying the Gdh-S allele were more sensitive to low night temperatures than were plants carrying the normal or common allele, Gdh-N. While most enzyme activity occurs in root or mesocotyl mitochondria significant levels can be recognized in pollen, and it seemed possible that a differential transmission of the alleles through the pollen after pollination at low temperatures might provide a sensitive measure of the effects of temperature. Plants from seeds of the F6 generation of Gdh-N/Gdh-S heterozygotes were used for the assay. Plants homozygous Gdh-N/Gdh-N were grown at 27 C day and 22 C night (27-22 C) until the silks emerged and were then placed in the test temperature for 24 hr prior to pollination. Five days after pollination the plants were returned to 27-22 C until the kernels matured. Seedlings were assayed for GDH isozymes as previously described (Heredity 32:397-401), and the results (Table 1) demonstrate that the pollen transmission of Gdh alleles was not influenced by temperature.

Table 1. Transmission of Gdh-S and Gdh-N alleles in pollinations at different temperatures.

Temperature		27-22 C	21-16 C	18-13 C	15-10 C
Ears		2	4	4	2
Progeny:	Gdh-N	59	187	161	91
	Gdh-S	42	215	172	94
Σ		101	402	333	185
$\chi^2[1:1]$		0.44	1.95	0.36	0.05
p		n.s.	n.s.	n.s.	n.s.

There are many obvious weaknesses in this sort of experiment; it is not known whether the GDH that is present in pollen grains is in any way active during the germination and growth of pollen. Our future work will concentrate more on the effects of the different Gdh alleles on root growth at different temperatures. Preliminary characterization of the GDH isozymes indicates that all three have the same pH optima (about pH 8.5); the apparent Michaelis constants are Km[NADH], 7.1×10^{-5}M; Km[α-ketoglutarate], 2.5×10^{-3}; and Km[NH_4^+], 5×10^{-2}M.

Tony Pryor

DEPARTAMENTO DE INVESTIGACIONES ANTROPOLÓGICAS Y GENETICAS
Barcelona, Spain

Relationship between a luteus character, not yet identified, and earliness

In S_n populations from the crosses of one inbred line (B) we have observed segregations of luteus plants. We noticed that, generally, luteus plants were more precocious than normal plants. For individual plants (normal and luteus) the periods from planting to pollen shedding were registered.

For comparison of corresponding periods, the data were classified in three main groups, as follows: 1. Normal plants versus the luteus ones, segregating by selfing from the same inbreds. 2. The groups of normal plants from segregating inbreds versus non-segregating inbreds of 100% luteus plants. 3. Inbreds 100% normal versus different inbreds 100% luteus. Relationship between compared groups is indicated by the respective pedigrees. From the six comparisons (Table 1), the results were: 1) Five of the luteus groups were more precocious than the corresponding normal ones; such differences (ranging between 5.3 days and 11.3 days) were significant at level of $p < 0.001$. 2) There was only one non-significant difference.

Table 1. Test of significance of M-M' for pollen shedding periods of normal versus luteus.

Pedigree	Class inbred	Class plants	No. pl.	Period (days)	M-M'	t
1. [(LxB)xB] -1-1-4 " -3 " -1	Segregating	normal luteus	17 37	75.05 68.88	6.17	17.97**
2. [(LxB)xB] -3-2-1 [(LxB)xB] -6-1-3	Segregating Luteus 100%	normal luteus	4 18	81.00 69.72	11.28	9.14**
[(LxB)xB] -1-1-2 [(LxB)xB] -6-1-3	Segregating Luteus 100%	normal luteus	35 18	75.05 69.72	5.35	13.65**
3. [(MxB)xB] -1-1-1 [(MxB)xB] -1-1-5	Normal 100% Luteus 100%	normal luteus	12 19	80.16 71.26	8.90	14.83**
M x [(MxB)xB] -3 M x [(MxB)xB] -1	Normal 100% Luteus 100%	normal luteus	19 7	68.89 68.85	0.04	0.096
[(LxB)xB] -1-2 [(LxB)xB] -1-3	Normal 100% Luteus 100%	normal luteus	7 5	76.57 67.20	9.37	18.22**

$^{**}p < 0.001$

From these results, it appears that this luteus character induces precocity. If, from researches going on, these conclusions were confirmed, this luteus could be used for practical purposes; for example, to reduce the flowering period of pure lines in order to obtain hybrid seed with inbreds originally of different periods.

Angel Alvarez, Luis Bosch, Mariano Blanco and José Luis Blanco

FARMERS FORAGE RESEARCH COOPERATIVE
West Lafayette, Indiana

Homozygous stocks of HtN available

FFR Cooperative released a source of resistance to northern corn leaf blight called HtN in 1975; the released stocks were segregating for both Ht and Ht2 genes. We now have available for distribution two lines, W22HtN and K64HtN, which are homozygous for the gene.

Ht2 is a single dominant gene which gives excellent resistance to *Helminthosporium turcicum*. This source of resistance was originally discovered by S. W. Nelson of Rhodesia and H. O. Gevers of South Africa and was brought to the U.S. through efforts of M. T. Jenkins and A. J. Ullstrup. Send seed requests to A. J. Ullstrup, FFR Cooperative, 4112 East State Road 225, West Lafayette, Indiana, 47906.

David Alvey

Diverse cytoplasms available for genetic studies

FFR Cooperative has developed stocks of B37 in three diverse cytoplasms: B37Ht in white (K6) cytoplasm, B37Ht in flint corn cytoplasm and B37Ht in popcorn cytoplasm. These stocks have been backcrossed eight times to B37Ht, and there are no readily apparent phenotypic differences between the cytoplasms.

FFR will supply seed of these stocks to all interested geneticists and breeders as long as the seed supply lasts. There will be no charge for the stocks. Address requests to David Alvey, FFR Cooperative, 4112 East State Road 225, West Lafayette, Indiana 47906.

David Alvey

FUNK SEEDS INTERNATIONAL
Bloomington, Illinois

Attempts to induce cytoplasmic male sterility with streptomycin and ethidium bromide

It has been proposed (MNL 47:35-37) that nuclear fertility restorer genes in plants treated with chemical mutagens may disguise an induced cytoplasmic male sterile (i.e., a sterile cytoplasm may be induced with a mutagen but still produce fertile plants due to the presence of nuclear restorer genes). Previously reported research (MNL 50:28-29) was with the inbred A632. According to Gracen and Grogan (MNL 48:20-23) and Gracen (personal communication), this inbred has restorer genes for many different sterile cytoplasms; therefore, it could also carry restorer genes for sterile cytoplasms that might be induced with chemical agents. To examine this idea, an inbred that does not restore many sterile cytoplasms should be chosen, and the inbred W59M was suggested by Gracen.

Seeds of inbred W59M were germinated for 30 hours on Kimpak at 27 C; at the end of this time some radicles had emerged. The germinated seeds were placed in petri dishes on Kimpak saturated with the mutagen: streptomycin in doses of .001%, .005%, .01%, .05%, .10%, .15% and control, and ethidium bromide in doses of .001 M, .005 M, .01 M, .05 M and control; all treatments were conducted for 24 hours at approximately 22 C. The treated material was planted in trenches in the field, watered and then covered. The M_1 plants were self-pollinated in 1975 and planted ear-to-row in 1976. Among 128 M_1 and approximately 3840 M_2 plants from the streptomycin treatment and 91 M_1 and 2730 M_2 plants from the ethidium bromide

treatment, no male steriles were detected. Since no male steriles were found, the value of using nonrestoring inbreds cannot be determined.

Robert W. Briggs

Success of a system to make self- and cross-pollinations on the same ear

Last year (MNL 50:29-30) a procedure was described by which it is possible to make self- and cross-pollinations on the same ear, using purple aleurone markers to separate the selfed and crossed portion of the ear. This system was used to carry out a form of full-sib reciprocal recurrent selection. In 1976 in replicated yield trials, among approximately 5000 plants from 86 crosses, only nine ears showed the presence of aleurone markers (i.e. only nine ears were not actually crossed genotypes); no more than two such ears in any one plot were noted. Such a small amount of contamination did not interfere with the success of this technique.

Robert W. Briggs

ILLINOIS STATE UNIVERSITY
Department of Biological Sciences, Normal, Illinois

The effects of trisomy on kernel weight

Trisomy is being used as a tool to screen the maize genome for genetic loci that influence kernel weights. If a gene(s) influencing kernel weight and exhibiting gene dosage effects is present on a given chromosome, sibling kernels disomic and trisomic for this chromosome will have different kernel weights. By comparing the weights of sibling trisomic and disomic kernels, we are comparing the effects of three versus two copies of all genes on a specific chromosome.

The maize stocks employed in this study were generously provided by Dr. Gregory Doyle of the University of Missouri. Each trisomic type has been backcrossed three to eight generations to the inbred KYS to increase the isogenicity of each type. Thirty sibling kernels from the final backcross of each trisomic type were selected for analysis. Progeny kernels from the backcross to KYS of a female plant trisomic for chromosome 1 were designated family 1; kernels from the backcross of a female parent trisomic for chromosome 2 to KYS were designated family 2, etc. The smallest kernels from each trisomic type were selected for these experiments.

Each kernel employed in this study was dried in a Thelco model 18 forced-air oven at 40 C for 10 days then at 60 C for one week. These conditions reduce kernel water content to less than 1.3% w/w (Plewa and Weber, 1973. Can. J. Genet. Cytol. 15:313). This drying was necessary for nuclear magnetic resonance (NMR) spectroscopy studies that were carried out subsequently on the same kernels. The NMR studies will be discussed elsewhere. All kernels used in this experiment were dried simultaneously in the same oven; thus, the relative weights of the kernels should not be affected by this procedure.

After the kernels were dried, their weights were determined with a digital balance. The weight determinations were made prior to the identification of individual trisomic kernels; thus, possible experimental bias was absent from this experiment. To identify the trisomic kernels in each family, each kernel was germinated to produce a source of meristematic tissue for use in somatic chromosome counts. Root-tips were harvested from individual plants and were analyzed cytologically by the standard Feulgen squash technique. Due to rodent predation, the entire family segregating for trisomy of chromosome 9 was lost before the identification of trisomic kernels. Thus, no data are available on trisomic 9 kernels.

Plants that were identified as trisomics were transplanted to the field where they were allowed to grow to maturity. Approximately two weeks prior to anthesis,

microsporocyte samples were removed from each plant and analyzed cytologically by the standard propiono-carmine smear technique. The identity of the trisomic chromosome was confirmed in aneuploid plants in each family.

The results are presented in Table 1. The mean weight in mg of disomic kernels was compared with that for their sibling trisomic kernels using Student's t-test.

Table 1. Comparison of mean kernel weights of sibling disomic and trisomic kernels.

Trisomic Type	Disomic kernels		Trisomic kernels	
	No. of Kernels	Mean weight mg ± S.E.	No. of Kernels	Mean weight mg ± S.E.
1	8	150.4 ± 2.5	7	148.6 ± 5.8
2	13	162.4 ± 3.1	9	139.8 ± 4.6**
3	16	142.8 ± 3.7	8	146.0 ± 3.0
4	12	136.0 ± 6.4	5	134.6 ± 4.4
5	16	166.4 ± 5.5	4	163.3 ± 6.5
6	16	137.1 ± 2.6	4	140.0 ± 4.0
7	9	161.6 ± 7.8	5	137.6 ± 8.7*
8	10	143.4 ± 2.8	5	151.0 ± 8.1
10	19	133.2 ± 1.6	4	134.3 ± 8.5

*significantly different at 0.05 confidence interval
**significantly different at 0.01 confidence interval

Because trisomy of specific chromosomes affects plant vigor in different ways, the progeny of distinct trisomic plant types will be affected differently by the specific aneuploid condition of the female parent. Variations in kernel weights between disomic kernels of different families reflect the distinct effects of trisomy of different chromosomes in their maternal parents. Hence, pooled data for all disomic kernels could not be compared accurately with pooled data for all trisomic kernels due to the considerable differences in kernel weights. However, the trisomic kernels in five of the families were lighter than their diploid sibling kernels, and in four families they were heavier. Thus, there was no general tendency for trisomic kernels to be either lighter or heavier than normal diploid kernels in this sample.

A comparison of kernel weights between sibling disomic and trisomic kernels within each family showed that significant differences existed in only two families. Trisomic 2 kernels were significantly lighter ($p < 0.01$) than their sibling disomic kernels. The mean weight for diploid kernels was 162.4 mg while the sibling trisomic 2 kernels had a mean weight of 139.8 mg. Trisomic 7 kernels were significantly ($p < 0.05$) lighter than their disomic sibling kernels. The mean kernel weights were 137.6 mg and 161.6 mg respectively. These data indicated that incompletely dominant genetic factors that influence kernel weight are present on chromosomes 2 and 7 of maize.

In family 8, the mean weights for sibling trisomic 8 and disomic kernels were 151.0 mg and 143.4 mg respectively. Although this difference is not significant at the 95% confidence interval, trisomic 8 kernels show a definite tendency to be heavier than their disomic siblings. Thus, incompletely dominant genetic factors that influence kernel weight may be present on chromosome 8 in addition to chromosomes 2 and 7 of maize.

Walter D. Fox and David F. Weber

The effects of trisomy on embryo volume

In the preceding paper we demonstrated that maize kernel weight is influenced by incompletely dominant genetic factors present on chromosomes 2 and 7. We also determined the effects of trisomy on embryo volume by comparing the embryo volumes of sibling trisomic and disomic kernels. In this way, most of the maize genome was screened for incompletely dominant genetic loci that influence embryo volume.

The kernels employed in this study were the same kernels analyzed in the previous investigation of kernel weight. In addition, the same designation of kernels into families was maintained throughout these experiments.

After the weight of the kernels was determined, they were scanned with a nuclear magnetic resonance (NMR) spectrometer. The results of the NMR spectroscopy analyses will be presented elsewhere. Following NMR spectroscopy, the kernels were placed at 100% humidity for 24 hours. This hydration was necessary to soften the kernels for slicing, which was part of the embryo measurement procedure. The measurements were made immediately after the kernels were removed from the humid environment.

The method used to estimate the embryo volumes was that developed by Plewa (Plewa and Weber, 1973, Can. J. Genet. Cytol. 15:313). A dissecting microscope equipped with an ocular micrometer was used at 10X magnification to determine the embryo dimensions. First, the length and width of the surface of the embryo of each uncut kernel was measured. Each kernel was then cut in half parallel and adjacent to the embryonic axis. The portion of the kernel containing the embryonic axis was turned on its side and the depth of the embryo was measured. Each measurement was made by determining the greatest linear distance within the boundaries of the scutellum.

The product of length times width times depth was calculated for each kernel; this product is an estimate of embryo volume. All determinations of embryo volumes were made prior to the identification of trisomic kernels and thus, experimental bias was absent from this study.

Kernel halves containing the embryonic axis were germinated and root-tips were collected from the resultant plants. These root-tips were used in somatic chromosome counts as described in the preceding report in this Newsletter. Unfortunately, the entire family segregating for trisomy of chromosome 9 was lost during germination due to rodent predation; thus no data are available from trisomic-9 kernels.

Plants cytologically identified as trisomic were transplanted to the field and allowed to grow to maturity. Microsporocyte samples were removed from trisomic plants in each family and analyzed cytologically to confirm the identity of the trisomic chromosome

In each family of sibling trisomic and disomic kernels, the embryo volume estimates (hereafter designated as embryo volume) of trisomic and sibling disomic kernels were compared. These comparisons were made using Student's t-test. The results of these comparisons are presented in Table 1.

The effects of trisomy of different chromosomes on embryo volumes were highly specific. Disomic kernels had larger embryo volumes than their trisomic siblings in six of the nine trisomic types studied. Thus, the volumes of embryos in the trisomic kernels are generally smaller than in their disomic sibling kernels.

When the mean embryo volumes for trisomic kernels were compared with those for their sibling disomic kernels, significant differences were found for three trisomic types analyzed. The embryo volume of trisomic 2 kernels was significantly smaller ($p < 0.01$) than their disomic siblings. Trisomic 2 kernels had a mean embryo volume of 3.16 mm^3 compared to 4.37 $mm^3 \times 10^{-3}$ for their disomic siblings. Trisomic 7 kernels also had embryo volumes that were significantly smaller ($p < 0.01$) than their disomic siblings; the respective mean embryo volumes were 2.51 $mm^3 \times 10^{-3}$ and 3.11 $mm^3 \times 10^{-3}$. In contrast, kernels trisomic for chromosome 4 had a mean embryo volume of 4.81 $mm^3 \times 10^{-3}$, while sibling disomic kernels' mean embryo volume was 3.40 $mm^3 \times 10^{-3}$. This difference was also significant ($p < 0.05$).

Because kernels that were trisomic for chromosomes 2, 4, or 7 had embryo volumes that were significantly different from those for their respective disomic siblings, incompletely dominant genetic factors that influence embryo volume reside on these chromosomes.

Table 1. Comparison of mean embryo volume of sibling disomic and trisomic kernels.

Trisomic Type	Disomic kernels: No. of Kernels	Disomic kernels: Mean Volume ($mm^3 \times 10^{-3}$) ± S.E.	Trisomic kernels: No. of Kernels	Trisomic kernels: Mean Volume ($mm^3 \times 10^{-3}$) ± S.E.
1	8	3.17 ± .22	7	3.26 ± .28
2	13	4.37 ± .23	9	3.16 ± .23**
3	13	3.41 ± .25	6	3.16 ± .16
4	12	3.40 ± .25	5	4.81 ± .63*
5	16	5.20 ± .25	4	4.78 ± .35
6	16	3.60 ± .19	4	3.44 ± .33
7	9	3.11 ± .22	5	2.51 ± .39**
8	10	3.61 ± .25	5	3.40 ± .57
10	19	2.45 ± .09	4	2.70 ± .49

*significantly different at 0.05 confidence interval
**significantly different at 0.01 confidence interval

It is interesting to note that an earlier investigation (Plewa and Weber, 1973, Can. J. Genet. Cytol. 15:313) employing maize kernels monosomic for chromosomes 2, 7, 8, or 10 showed that monosomic 10 embryos were significantly smaller than diploid control embryos. In the current study, trisomic 10 embryos were only slightly, but not significantly larger than disomic kernels. Plewa and Weber's study found that the volumes of monosomic 2 and monosomic 7 embryos were not significantly different from disomic control embryos. Trisomic 2 and trisomic 7 embryos were found to be significantly smaller than disomic sibling embryos in the present study. The reasons for the differences in these two studies are not known, but they might be attributed to strain differences in the maize stocks employed. In addition, monosomic embryos are accompanied by euploid (triploid) endosperm whereas trisomic embryos are surrounded by aneuploid (pentasomic) endosperm. The trisomic chromosome in the embryo is present in five copies in the endosperm. Perhaps the aneuploid endosperm of trisomic kernels exerts some influence over embryo volume.

A comparison can be made of the relative effects of trisomy of specific chromosomes on kernel weight (discussed in the preceding paper) and on embryo volume. To determine the effects of trisomy of a specific chromosome on kernel weight, the ratio of mean disomic kernel weight to mean sibling trisomic kernel weight was calculated for each family. Similarly, the ratio of mean disomic embryo volume to mean sibling trisomic embryo volume was calculated for each family. These ratios are presented in Table 2.

A comparison of the absolute values of the deviation from 1.00 of the kernel weight ratios and the embryo volume ratios for each trisomic type can be used as an index of the magnitude of the effect of trisomy on these kernel parameters. The deviation of the embryo volume ratio is greater than the deviation of the kernel weight ratio for all but one of the trisomic types analyzed. Hence, the effect of trisomy was greater on mean embryo volume than on mean kernel weight for all nine chromosomes tested.

Table 2. Relative effects of trisomy of specific chromosomes on mean kernel weight and mean embryo volume.

	Mean kernel weight ratio		Mean embryo volume ratio	
Trisomic type	Disomic/Trisomic	Absolute value of deviation from 1.00	Disomic/Trisomic	Absolute value of deviation from 1.00
1	1.012	0.012	0.972	0.028*
2	1.162	0.162	1.383	0.383*
3	0.978	0.022	1.079	0.079*
4	1.010	0.010	0.707	0.293*
5	1.019	0.019	1.088	0.088*
6	0.979	0.021	1.047	0.047*
7	1.174	0.174	1.239	0.239*
8	0.950	0.050	1.062	0.062*
10	0.992	0.008	0.907	0.093*

*denotes largest deviation

Walter D. Fox and David F. Weber

A study of the meiotic cytology of thirteen nuclear male sterile genes

Many different nuclear male sterile (ms*) genes have been isolated in maize. In a plant homozygous recessive for such a gene, the pollen fails to develop to maturity. We are analyzing a number of nuclear male sterile genes in an attempt to determine the mode of action of these genes. It is possible that certain of these genetic factors control some portion of the meiotic process. If this were the case, plants homozygous recessive for one of these genes would have an abnormal meiotic sequence. Thus, this material might be a source of new meiotic mutants. For this and other reasons, we are analyzing meiosis in these nuclear male steriles. To date, we have analyzed meiosis in thirteen different nuclear male steriles.

The lines under analysis were generously provided to us by Dr. Earl Patterson of the University of Illinois. In each of the lines provided to us, a plant homozygous recessive for a male-sterile locus (ms*/ms*) was crossed as a female parent with a male heterozygous for the same locus (Ms*/ms*). Half of the progeny of such a cross are fertile (Ms*/ms*) and the other half are male-sterile (ms*/ms*). To date, we have examined lines segregating for homozygosity at the following loci: ms, ms2, ms3, ms5, ms7, ms8, ms9, ms10, ms11, ms12, ms13, ms14, and ms17.

Each of the lines was planted and microsporocyte samples were removed from several plants in each line. These samples were placed immediately in a fixative solution (ethanol:propionic acid, 3:1 v/v) and stored under refrigeration.

When the plants reached anthesis, each was examined to determine if it was ms*/ms* (male sterile) or Ms*/ms* (male fertile). Microsporocyte samples from fertile and male-sterile plants were analyzed cytologically using the standard propiono-carmine smear technique.

Five diagnostic stages in meiosis were compared to determine if the meiotic process was altered by a given male-sterile gene. In all male-sterile types, pairing between homologous chromosomes appeared normal at pachytene. At diakinesis, 10 bivalents were regularly present. At metaphase I, 10 bivalents were consistently present on the metaphase plate in each cell, and the bivalents separated in a normal manner at anaphase I.

The quartet stage is a powerful diagnostic stage because a study of it can show if a loss or nondisjunction of chromosome 6 took place during either the first or second meiotic division. If the disjunction of chromosome 6 proceeds normally, a nucleolus will be present in each of the four haploid meiotic products because chromosome 6 bears the nucleolar organizing region of maize. If nondisjunction or loss of chromosome 6 took place, one or more members of the quartet would lack a chromosome 6, and consequently it would lack a nucleolus. If nondisjunction took place during either division, two nucleoli would sometimes be found in one or two members of the quartet if nucleolar fusion had not taken place. Also, if chromosome loss took place, micronuclei would be found in quartet cells.

In the male-sterile lines examined, one nucleolus was regularly found in each of the four meiotic cells at the quartet stage. Thus, nondisjunction or loss of chromosome 6 did not occur during meiosis in any of the male-sterile lines analyzed. Since the disjunction of chromosome 6 was normal in these types, the disjunction of other chromosomes also presumably was normal. The fact that micronucleoli were not found in these cells supports this last conclusion.

Another type of male-sterility is cytoplasmic male sterility (CMS). In certain cytoplasmic male-sterile plants, breakdown of the tapetum is believed to be responsible for the male sterility. Tapetal cells at the quartet stage were also examined in each of the male-sterile types, and all tapetal cells appeared normal. It is possible, however, that the tapetal layer might break down later in development in some types and be responsible for the male sterility.

We are continuing our investigation of additional male-sterile types. We will also be examining post-meiotic stages in an attempt to determine the stage and mode of breakdown in each type.

If additional male-sterile types are available from any source, we would appreciate it if you could send them to us for analysis. Dr. Patterson has supplied us with lines segregating for the following genes: ms, ms2, ms3, ms5, ms7, ms8, ms10, ms11, ms12, ms13, ms14, ms17, as, po, ms*-Bear#1, ms*-Bear#2, ms*-Bear#3, ms*-Bear#4, ms*-Bear#5, ms*-Bear#6, ms*-Bear#7, ms*-Bear#8, ms*-Bear#10, ms*-Holden 4439, ms*-M11, ms*-M70, ms*-B76, at-si.

Tau-San Chou and D. F. Weber

The effect of TB-4a and TB-8a on intergenic recombination of chromosome nine

I have been studying the effects of monosomy on intergenic recombination in the maize genome (Weber 1971, MGNL 45:32-35; 1976, Genetics 83:s81). I recently determined that intergenic recombination in the sh-wx region of chromosome 9 is consistently and significantly lower in monosomic 4 plants (11.2 ± 0.8%) and monosomic 8 plants (17.1 ± 0.9%) than in diploid control plants (23.0 ± 0.8%) (Weber, 1976). It appears to be unchanged in monosomio 7 plants (21.8 ± 1.8%); thus, monosomy per se does not alter intergenic recombination. In these experiments the monosomics and diploids were testcrossed as males. I also determined the effect of trisomy of chromosome 4 on intergenic recombination in this same region and found that trisomy of chromosome 4 increased recombination slightly in both the male and female parents, but the increases were not significant (Weber 1976, MGNL 50:35-36). Thus, monosomy alters genetic recombination in the region analyzed far more than trisomy. I am attempting to localize these sites of monosomic and trisomic-induced recombinational alterations utilizing TB translocations. Initial results of this work are reported in this communication.

As indicated above (Weber 1976, Genetics 83:s81), monosomy of chromosome 4 decreased recombination in the sh-wx region of chromosome 9 to only 49.7% of that found in diploid control plants. To further localize the site of the recombinational alteration, hyperploid TB-4a plants were crossed as male parents by a c sh wx tester inbred line. Sibling hyperploid and hypoploid progeny of this cross were test-crossed as male parents and the results of these crosses are presented in Table 1.

Table 1. Comparison of recombination in chromosome 9 in hypoploid and hyperploid TB-4a plants.*

Plant type		% Recombination in c-sh region	% Recombination in sh-wx region	Population
Hypoploid (5 plants)			26.3	156
			38.5	122
			19.5	113
		5.8	32.2	345
		3.3	25.8	302
	$\overline{X}$	4.6	28.5	
Hyperploid (3 plants)			28.3	311
		2.4	25.7	416
		4.5	18.4	397
	$\overline{X}$	3.5	24.1	

*All hypoploids and hyperploids were crossed as males by tester stocks

It can be seen that recombination in the sh-wx region in hypoploid TB-4a plants was not lower, and possibly higher than in hyperploid sibling plants. Therefore it can be concluded that the factor(s) altering recombination in monosomic 4 plants is not in the segment distal to the breakpoint in TB-4a. The factor(s) must therefore lie proximal to the breakpoint in the short arm or in the long arm of chromosome 4.

In monosomic 8 plants, the percent recombination between sh and wx on chromosome 9 is only 74.3% of the value found in diploid control plants. TB-8a hyperploid plants were also crossed as male parents by a yg sh bz wx tester inbred line, and the sibling hyperploid and hypoploid progeny of this cross were testcrossed as male parents. The results are presented in Table 2.

Table 2. Comparison of recombination in chromosome 9 in hypoploid and hyperploid TB-8a plants.*

Plant type		% recombination in yg-sh region	population	% recombination in sh-bz region	% recombination in bz-wx region	population
Hypoploid (3 plants)		30.4	214	2.3	26.7	476
		20.5	308	4.5	28.4	331
		25.2	405	2.7	18.7	411
	$\overline{X}$	25.4		3.2	24.6	
Hyperploid (2 plants)		24.8	282	2.2	21.9	465
		21.8	211	2.6	20.0	571
	$\overline{X}$	23.3		2.4	21.0	

*Hypoploid and hyperploid plants were crossed as males by tester stocks.

It is clear that recombination in the sh-wx region in hypoploid TB-8a plants is not lower than in hyperploid plants. There is a slight increase in recombination in the hyperploid plants. It is concluded that the region(s) on chromosome 8 that reduces recombination in monosomic 8 plants is not missing in the hypoploid TB-8a plants. The factor(s) affecting recombination on chromosome 8 must be located

between the breakpoint and the centromere in the long arm of chromosome 8 or in the short arm of chromosome 8.

It is well established that B chromosomes increase intergenic recombination in the maize genome. Hyperploid plants contain two additional copies of the segment of the B chromosome proximal to the breakpoint on the B chromosome. If these additional B chromosome segments increased recombination, recombination would be higher in the hyperploid plants than in hypoploid plants. Exactly the opposite result was found. If the factor(s) on chromosome 4 or 8 that alter recombination in monosomic 4 or 8 plants were distal to the TB breakpoints, recombination in hyperploid plants would also be higher in hyperploid TB-4a and TB-8a plants than in their hypoploid counterparts. However, recombination in the hyperploid plants was lower than in their sibling hypoploids in both cases. Thus, the influence of B chromosomes on recombination did not interfere with the results of this study, and it can clearly be concluded that the factors on chromosomes 4 and 8 are not distal to the breakpoints in TB-4a or TB-8a. (This work was supported in part by a contract from the USERDA, EY-76-S-02-2121; we would also like to express our appreciation to Funk's Seeds International for generously providing field space in a summer nursery and in a winter nursery in which this work was carried out).

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The expression of certain enzymes in a dosage series produced by TB-1La

In the process of extending a gene dosage study of the *Adh* locus, it was found that the expression of certain enzymes is affected by aneuploidy in a precise manner. There is a negative correlation between the enzyme level and the number of long arms of chromosome one present. A discussion of this phenomenon and its implications is the subject of this and the following reports.

Those enzymes discussed in this report are alcohol dehydrogenase, ADH; glucose-6-phosphate dehydrogenase, G6PDH; 6-phosphogluconate dehydrogenase, 6PGDH; isocitrate dehydrogenase, IDH; and phosphoglucomutase, PGM. The *Adh* locus is carried in the portion of chromosome one that has been translocated to the centromeric portion of the B in the B-A translocation 1La. The *Adh* gene has been mapped to within 2 map units of *lw* in the long arm of chromosome one (Schwartz, 1971, Genetics 67:411). The locations of the other enzyme loci are unknown. The comparisons of enzyme activities of different levels of aneuploidy were always performed on kernels from the same ear. The tissue studied was the mature scutellum, separated from the endosperm.

The aneuploids were generated in the following way. A euploid female homozygous for one ADH electrophoretic variant was crossed by pollen from a hyperploid plant (1 $B^1B^1 1^B$) that carried a different electrophoretic variant in the translocated arm. Such a cross will generate monosomics, disomics, and trisomics. The trisomics from this type of cross exhibit a small kernel phenotype and consequently were usually discarded. The remaining kernels are normal in size and consist of monosomic and disomic embryos. The monosomics can be distinguished from the disomics by excising a small portion of the scutellum and subjecting an extract of this to starch gel electrophoresis. The monosomic kernels have only a single electrophoretic variant, i.e. the female contribution, whereas the disomics have two electrophoretic variants present.

Trisomic vs. disomic comparisons were made on kernels produced by using a hyperploid female that had one electrophoretic ADH variant in the normal chromosome and a second variant in the two translocated arms, and crossing it by a diploid male, which carried a third electrophoretic variant of ADH. Kernels produced from this

cross which had three electrophoretic variants present were considered to be trisomics. Kernels with two electrophoretic variants present in a 1:2:1 homo:hetero: homodimer ratio were considered to be disomic. Trisomic production was performed in this manner to minimize the difference in kernel size in the two classes. In some cases, trisomics were produced by crossing a hyperploid male having a different variant. Trisomic vs. disomic scutella, in this case, can be distinguished by isozyme band ratios.

Finally, tetrasomics were produced by using, as females, hyperploid plants that had one variant on the normal chromosome and a second on the two translocated chromosomes, in crosses with hyperploid males that were homozygous for a third electrophoretic variant. In crosses of this type, the entire series of 1-4 doses of the long arm of chromosome one can be distinguished. The class of kernels with only one electrophoretic variant may be slightly ambiguous. Most of the kernels of this type should be monosomics, although some disomics may be of this type due to crossing over between the centromere and the Adh locus so that both the normal and translocated chromosome introduced into the egg carry the same allele. If this egg is fertilized by a sperm deficient for the long arm of chromosome one, the resulting scutellum would be indistinguishable from a monosomic scutellum, which has only one ADH variant. However, controls show that recombination between the normal A chromosome and the two B-A chromosomes is minimal in the hyperploids of the type used. An example of a cross to produce tetrasomics is as follows. If a hyperploid, ADH CFF plant with the F allele on the translocated chromosome were used as a female, three major classes of gametes are formed: C, F, CF. If this plant is pollinated by a hyperploid male of ADH SSS constitution, each class of gamete could unite with a sperm that has no S, one S, or two S's. The major ADH genotypes from such a cross are as follows: C, F, CF, CS, FS, CFS, CSS, FSS, CFSS. Each of these can be distinguished from the others on the basis of the ADH variants present and their ratios.

Representative trisomic/disomic enzyme ratios per dry weight of mealed scutellum are shown in Table 1. Monosomic/disomic enzyme ratios are shown in Table 2. The data in Tables 1 and 2 are from crosses, which are from diverse backgrounds. The variability among crosses was great and the values were not averaged. However, if one considers the modal values, certain trends become evident. From a strict dosage effect for Adh, one would expect the one dose/two dose ratio to be .50, but it is much greater than expected. Likewise, in three doses vs. two doses, the modal value is near 1.10 to 1.14. This value is much less than the 1.50 relationship expected from a dosage effect. The MDH and PGM values seem to be little affected by the aneuploidy. However, the G6PDH, 6PGDH and IDH values are altered. The G6PDH activity in trisomes is less than in disomes. In monosomes, the G6PDH activity is higher than in disomes. 6PGDH and IDH are not reduced to the same degree as G6PDH in trisomes nor are they increased as much in monosomics. Because of the values found for G6PDH, 6PGDH and IDH, it is doubtful that their structural gene loci reside in the long arm of chromosome one. This has not been rigorously proven, however.

Since some enzyme levels are affected but others apparently are not, the changes in enzyme expression are thought to be specific and not a general effect of aneuploidy on protein synthesis or metabolism. In order to test whether the change in enzyme levels associated with aneuploidy is the result of a change in the cell number per dry weight, DNA estimations were performed on scutella of trisomes, disomes, and monosomes. It was found that all three classes, after correction for the difference in chromosome number present per cell, had very similar amounts of DNA per dry weight. This shows that the aneuploidy in this case does not drastically change the number of cells per mass of tissue. The estimation of the hydrolysable DNA was by a modification of the method of Webb and Levy (J. Biol. Chem. 213:107-117). To test the sensitivity of the method, similar maize lines with 0 or about 8 B chromosomes were compared. The determined values were near

Table 1. Representative ratios of trisomic/disomic enzyme level per dry weight of scutella.

ADH genotype	Trisomic/Disomic Ratios						
	ADH	G6PDH	MDH	6PGDH	IDH	PGM	Protein
CFF/CF	1.16	.76	1.11				.92
SCF/CF	1.12	.88	1.09	.88	.95		.92
SCF/CF	1.26	.70	.97	.85	.90		.96
SCF/CF	1.01	.70	.91	.75	.83		.74
CFF/CF	1.13	.72	.99	.85	.92		.85
SCF/CF	1.08	.81	.98	.83	.89	.93	.91
SCF/CF	1.33	.75	1.01	.91	.83		.82
SCF/CF	1.40	.97	1.00	.96	.98	1.00	.98
FSS/FS	1.11	.76	.99	.81	.86	1.00	.94
SCF/CF	1.38	1.05					1.07
FFC/FC	1.10		1.09				
SFF/SF	1.07		1.01				
CFF/CF	1.19		1.27				
FFC/FC	.98		.96				
FFC/FC	1.11		1.22				
SCF/SF	1.29		1.01				
SCF/CF	1.25	.84	1.13	.89	.86		

Table 2. Ratios of monosomic/disomic enzyme level per dry weight of scutella.

ADH genotype	Monosomic/Disomic Ratios						
	ADH	G6PDH	MDH	6PGDH	IDH	PGM	Protein
C/CF	.51	1.35	.98				
S/SF	.62		1.03				1.06
C/CF	.73		.99				
S/SF	.63		.77				
C/CF	.78		1.25				
C/CF	.48		.81				
C/CF	.70		1.00				
S/SF	.80		.95				
C/CF	.74		1.05				
C/CF	.65		1.04				
S/FS	.65		.99				
S/FS	.77		.96				
S/FS	.67	1.52	.92	1.32	1.20	1.14	
C/CF	.67	1.84	1.08	1.53	1.41	.98	1.06
F/FS	.63	1.61	1.19			.97	.98
F/FC	.64	1.68	1.07	1.52		1.06	1.13

those expected from given knowledge of the DNA content of a B chromosome (Ayonoadu and Rees, 1971, Heredity 27:365).

In order to test whether any qualitative differences in these enzymes were produced by aneuploidy, electrophoresis of extracts of mature scutellum was conducted and the gels were stained for the enzymes mentioned here by the methods (with modifications) of Brewer (An Introduction to Isozyme Techniques, 1970). MDH patterns were similar to those found by Yang and Scandalios (1974, Arch. Biochem. Biophys. 161:335). Zymograms of G6PDH, 6PGDH, IDH and PGM showed only a single major zone of activity. No discernible differences in isozyme pattern were seen between the aneuploid and euploids.

To test if there is any effect of B chromatin on the expression of these enzymes, other B-A translocations were used to generate aneuploids in which 1, 2 and 3 doses of the translocated portion could be phenotypically distinguished. Since six other aneuploid series do not give the same spectrum of enzyme expression as reported here for the TB1La series, it is concluded that the effects found are specifically produced by the A chromatin.

The tetrasomic analysis was conducted in a different manner due to the unavoidable circumstance that tetrasomic kernels are very small. The small size of the endosperm reduces the growth of the embryo. Since MDH activity did not seem to be affected by aneuploidy produced by TB1La in aneuploids of similar kernel size, ADH and G6PDH values were standardized against MDH. In one ear the standardized ADH trisomic/disomic and tetrasomic/disomic ratios were 1.19 and 1.17, respectively. G6PDH in the same ear gave ratios of 3/2 = .68; 4/2 = .54. In a second ear, ADH ratios were 1/2 = .46; 3/2 = 1.19; 4/2 = 1.25. G6PDH values were 1/2 = 1.40; 3/2 = .83; 4/2 = .67.

Considering all of the above data, it seems that the expression of G6PDH, 6PGDH and IDH is affected by a region of chromatin (a gene?) involved in TB1La in such a manner that there is a negative correlation between the dose of the varied region and the expression of these enzymes. In the case of G6PDH, the expression closely approaches the inverse of the ratio of the number of long arms of chromosomes one in the aneuploid to the number in the euploid. That is, in monosomics the ratio of expression of G6PDH approaches 2/1 (the inverse of 1/2) times the expression in disomics. In trisomics, the expression approaches 2/3 (the inverse of 3/2) the expression in disomics. For this reason the phenomenon has been tentatively referred to as the "inverse effect," and the chromosomal region responsible as the "inverse effect region" (IE region).

The equalization of ADH in these aneuploids deserves discussion. In this case the structural locus has been varied in a 1, 2, 3, 4 relationship, but the expression is not found to be directly proportional. One explanation for this could come from the gene competition hypothesis (Genetics 67:411). According to this interpretation, the expression is nearly equal in the aneuploid series because there is a limiting factor, produced elsewhere in the genome, that is required for ADH expression. Thus the ADH level would not depend on the number of structural genes present.

An alternative explanation is that a dosage effect of varying the _Adh_ locus is cancelled by an inverse effect influencing ADH expression. Hence, when only one dose of _Adh_ is present, there is also only one dose of an inverse region. Consequently, a reduction of ADH expression due to a reduced structural gene number is compensated by an increased expression due to the reduction in the number of IE regions. Any increase expected in the trisomic due to an increased number of structural genes would be cancelled by a reduction in expression due to the _increased_ number of IE regions. Evidence that G6PDH, 6PGDH, IDH and ADH are all affected by the same IE region comes from exceptional ears in which ADH shows a dosage effect and G6PDH, 6PGDH, and IDH no longer show an inverse effect. This suggests, but does not prove, that the latter explanation for the compensation of ADH is correct. Experiments to distinguish these alternatives are in progress.

To understand the nature of the "inverse effect" more fully, these data from maize should not be considered alone. There is much evidence (although it has often gone unrecognized) for the presence of inverse regions in Drosophila and Datura (O'Brien and Gethman, 1973, Genetics 75:155; Rawls and Lucchesi, 1974, Genetical Research 24:59; Pipkin and Chakrabartty, 1975, Genetics 80:s64; Carlson, 1972, Molec. Gen. Genetics 114:273; Smith and Conklin, 1975, Isozymes, Vol. III). A discussion of their possible role in the regulation of gene expression, aneuploid depression, dosage compensation, sexual dimorphisms and sex determination is the subject of the other reports in this series. This report is only an abbreviated version of the data collected on this phenomenon.

James A. Birchler

On the biochemical basis of "aneuploid depression"

Geneticists have long noted that organisms that are either monosomic or trisomic for a particular chromosome are less vigorous than the euploid siblings (Khush, Cytogenetics of Aneuploids). The basis of this observation has been attributed to "genic unbalance." The studies of the biochemical consequences of aneuploidy reported above have possibly led to a greater understanding of this phenomenon on a biochemical level. In this report, the loss of vigor associated with aneuploidy will be referred to as "aneuploid depression."

On the biochemical level, there are two major types of effects on enzyme levels as a consequence of aneuploidy. First of all, there are gene dosage effects. That is, when segmental aneuploidy is produced for a chromosomal region that is known to carry the structural locus of a certain enzyme, the level of expression of that enzyme is directly proportional to the dosage of the chromosomal region in the dosage series. Secondly, there are inverse effects. The previous report has described the nature of this phenomenon. Basically, this effect involves a negative correlation between the dose of the chromosomal region varied and the level of an enzyme whose structural locus is not included in the aneuploid region. The data of O'Brien and Gethman (1973, Genetics 75:155) and Rawls and Lucchesi (1974, Genetical Research 24:59), for examples, illustrate these effects. When the structural loci of alpha-glycerophosphate, alcohol and isocitrate dehydrogenases were varied by segmental aneuploidy in a 1, 2, 3 relationship, the levels of these enzymes were in a respective 1, 2, 3 relationship. However, when some other segmental regions, which did not include the structural loci, were made trisomic, the enzyme level in the aneuploid was reduced relative to the euploid. The data presented in the previous report show that the inverse effect extends to segmental monosomy as well.

If a segmental aneuploid region contains both a structural gene locus and an inverse effect region that acts upon that gene, it is conceivable that the two effects would cancel each other. This cancellation would result in little net change in the level of the gene expression being monitored. The prevalence of such cancellations is unknown. The expression of ADH in the dosage series produced by TB1La (see previous report) and dosage compensation in Drosophila (see following report) may be examples.

The purpose of this note is to point out the possibility that reductions in enzyme levels may play a significant role in aneuploid depression. The above discussion has pointed out that both segmental monosomy and segmental trisomy produce reductions in the expression of sets of genes. The reductions in monosomics are due to a reduction in the number of structural genes present. The reductions in trisomics are due to an inverse effect of the aneuploid region upon the expression of a gene located elsewhere in the genome. Thus, since aneuploidy of either type causes reductions in a number of enzyme levels, a significant factor in aneuploid depression may be these reductions.

The concept that aneuploid depression is due mainly to enzyme level reductions is consistent with the fact that phenotypic mutants for which the biochemical bases are known overwhelmingly correlate a reduction of a gene product with a less vigorous phenotype (e.g. endosperm mutants; bobbed and enzyme loci in Drosophila). It is true that single gene reductions of the magnitude produced by aneuploidy are often not sufficient to reduce vigor by themselves. But we know that increasing the size of aneuploidy increases the loss of vigor (Khush, Cytogenetics of Aneuploids; Lindsley, et al., 1972, Genetics 71:157). This fact indicates that aneuploid depression is a cumulative effect of several minor components. Therefore, the loss of vigor due to aneuploidy may be the cumulative effect of the reduced expression of a number of enzymes.

The enzyme levels and vigor of the dosage series produced by TB-1La illustrate the possible correlation between aneuploid depression and enzyme reductions. The Adh gene, which is varied in this dosage series, shows a reduction in expression in the monosomic below the disomic, although it is usually not to the 50% level expected from a strict dosage effect. This intermediate value is thought to be due to a partial cancellation of a dosage effect by an inverse effect or to a limiting factor necessary for gene expression produced elsewhere in the genome. Although it is not currently known, it is conceivable that some genes included in TB-1La would show a reduction in the monosomic to 50% of the disomic level. The levels of G6PDH, 6PGDH and IDH, which are believed not to be included in the translocation, show reductions in the trisomes and tetrasomes produced by TB-1La. G6PDH is usually reduced in trisomes to about 2/3 of the disomic level and even further reduced in tetrasomes. If one considers only the reductions in enzyme levels, the vigor order of the dosage series can be explained. The vigor order is as follows from greatest to least: disomic, trisomic, tetrasomic, monosomic. This order is based on plant height and general appearance.

Although there is a correlation of enzyme reductions and vigor, one should not ignore the fact that each aneuploid also increases certain enzyme levels above the disomic value. Monosomic increases are due to a reduction of inverse effect regions; trisomic and tetrasomic increases are due to increased numbers of structural genes. Consequently, it could be argued that aneuploid depression is due to an imbalance of metabolic processes by increases OR a combination of increases and decreases. On the imbalance argument, aneuploid depression would also give a vigor order in which the aneuploids are also below the disomic.

Although the data on changes in enzyme levels in aneuploids do not allow an unequivocal discrimination of the above mentioned hypotheses, they do show that increases above euploidy usually cause decreases in the expression of sets of enzymes. The importance of the hypothesis of a correlation between aneuploid depression and reduced enzyme levels is that it allows an understanding of aneuploid depression consistent with our knowledge of the detrimental effects of single gene mutations. If this synthesis of observations is meaningful, aneuploid depression could be explained on large reductions of enzyme levels. It is reasonable, however, to propose that increases would produce stress in the cells or raise some metabolites to toxic levels. The challenge of future research is to discriminate among these alternatives, if indeed that is technically feasible or necessary.

James A. Birchler

Inverse effect regions in maize and Drosophila and their possible role in dosage compensation, sexual dimorphism of autosomal genes and sex determination in the latter

In studies involving segmental trisomy, designed to locate structural gene loci for enzymes, it was observed that trisomy for certain small regions in Drosophila and Datura caused reductions in the expression of some enzymes to a lower limit of

approximately 67% of the euploid control (References are in the first report). The phenomenon appears to be the same as that which has been described for the dosage series produced by TB-1La in maize. This literature does not, however, contain any data on the effect of segmental monosomy on enzyme expression. The fact that monosomy (in maize at least) increases the expression of some enzymes above the euploid level allows one to eliminate the possibility that the trisomy effect on enzyme level is a result of reduced vigor in these aneuploids, since monosomics are less vigorous than the euploids. In other words, the change in expression of these enzymes does not correlate with the vigor of the plant; it does, however, negatively correlate with the ploidy of particular chromosomal regions. The order of enzyme activity from greatest to least is monosomic, disomic, trisomic, tetrasomic; for vigor the relationship is disomic, trisomic, tetrasomic, monosomic.

Several trends become apparent when one takes an overview of these data: 1) Almost every enzyme (about 15 different ones) studied is affected by some trisomic regions, which cause reductions to about 67% of the euploid. The only exception is isocitrate dehydrogenase in *Drosophila*. In this case, several regions caused slight increases (Rawls and Lucchesi, 1974, Genetical Research 24:59); 2) A single enzyme can be affected by multiple regions; 3) These regions are scattered throughout the chromosomes and do not appear to be restricted in their location.

The precise role of these regions in the expression of enzymes is, of course, not known. The facts that they are found in higher eukaryotes as diverse as maize, *Datura* and *Drosophila*, that they affect a wide spectrum of enzyme types, and that there are many such regions, suggest that these effects are a manifestation of a central mechanism of gene expression. Some possibilities to be considered are as follows:

1) Inverse effect (IE) regions contain genes that encode enzymes of certain biochemical pathways that produce metabolites that affect the expression of other pathways at some level. If a metabolite affects the rate limiting process of gene expression inversely to the concentration of the metabolite, the observed values would be realized. A difficulty for this hypothesis is the fact that there are multiple regions affecting any one enzyme. This would require that each metabolite affecting a particular gene would be operating in a very similar manner.

2) IE regions contain genes that encode for enzymes that degrade specifically certain enzymes or messenger RNA's. This seems unlikely since trisomic vs. disomic values do not usually surpass the .67 lower level. Since different enzymes are undoubtedly expressed at different levels, a change in the level of every individual degradative enzyme would not necessarily produce an inverse effect. It should, however, be considered and rigorously tested.

3) IE regions contain genes which compete against the one which is being monitored.

4) IE regions contain genes whose products are repressors. This has been proposed by Smith and Conklin. There can be little argument that IE regions produce a negative effect on the enzyme monitored. But repressors as we know them from prokaryotes do not show different levels of repression dependent upon their dosage. If repressors of this type were present in the normal euploid, one would not expect to be able to detect any enzyme. Repressors that function in a different manner remain a possibility.

5) IE regions are genes whose products are negative modulators of enzyme expression such that the concentration of the modulator determines the level of enzyme expression. In order to produce such a close fit to an exact inverse effect in relation to their concentration, they must affect the rate limiting process of enzyme expression. If one believes that IE regions underlie the phenomenon of dosage compensation in *Drosophila*, they probably affect the process of transcription. Problems involved with the possibility that IE regions produce negative modulators are: a) The contrast of enzyme expression in an aneuploidy vs. ploidy series (to be discussed more fully later); b) In maize, multiple regions that

affect ADH expression have been found (Birchler, unpublished). Yet no cis dominant, constitutive mutant of Adh has been discovered in a large unselective screen (Schwartz, personal communication, and Birchler, unpublished). This may be due to the difficulty, for unknown reasons, of inducing such a mutant; c) Some data suggest that IE regions act in a positive manner under certain circumstances (to be discussed more fully later).

6) IE regions contain genes that encode positive effectors of enzyme expression, but act in a negative way under the conditions in which they have been detected. The reasons for suggesting this possibility will be discussed in the section on dosage compensation.

It is obvious that none of these is completely satisfactory or even that all possibilities have been considered. There is no precedent for this phenomenon and an understanding of the mechanism involved will require much more research.

Regardless of their mechanism of action, the existence of presumptive IE regions on all the chromosomes of Drosophila that affect sets of genes on all the chromosomes is sufficient to explain a huge volume of data and observations on dosage compensation, autosomal sexual dimorphic mutants and sex determination. Dosage compensation in Drosophila appears to fit into the total picture of inverse effects. Multiple regions which, when trisomic, reduce certain enzymes to about 67% of the euploid occur on the X and chromosomes 2 and 3. Thus if IE regions on the X affect structural genes on the X, males would have structural gene reductions to 50% of the female value, but since IE regions are only present once, the expression of the genes it controls would be approximately doubled. Hence, the affected genes would have a net expression in males nearly equal to females. In other words, an increase, due to the IE region, to approximately 200% of the female value is cancelled by a reduction of the number of structural genes to half that of females. Since suggestions of IE regions show up in data with such regularity, it is difficult to avoid such an explanation. This hypothesis will explain the observed compensation in metafemales [3/2 structural genes X 2/3 (the reciprocal of the dosage ratio of inverse regions) = 1.00] and in triploid intersexes vs. triploid females [2/3 structural genes in intersexes X 3/2 (the reciprocal of the dosage of inverse regions) = 1.00] (Muller, 1948, The Harvey Lectures; Lucchesi and Rawls, 1973, Genetics 73:459).

The IE regions hypothesis predicts that duplications of structural genes in males would produce more net gene expression than an extra dose (total 3) in females. Likewise, deficiencies of structural genes in females would have less net expression than in males. This is expected because the reduction of IE regions in males would allow each individual gene greater expression. This is the case (Muller, 1950, The Harvey Lectures).

The IE regions hypothesis does not readily account for the fact that triploid flies have approximately 150% enzyme expression per cell as diploids. That is, in a triploid, the IE regions would be increased 3/2 over the diploid and the structural genes also would be increased. This would give an hypothetical gene expression [3/2 structural genes X 2/3 (the reciprocal of the IE regions ratio)] equal to the diploid. This is not the case, as reported in the literature (Lucchesi and Rawls, 1973, Bioch. Genetics 9:41. The basis for this seeming paradox is unknown. One way to reconcile it would be to consider that when all the IE regions of a cell are increased they act in a positive manner. But when only one of several IE regions for a particular gene is increased, there is an inhibition of the total group. Other suggestions that IE regions may act positively under some conditions are: 1) The data of Rawls and Lucchesi show that IDH is exceptional in that it has multiple positive effecting regions instead of negative effecting regions; 2) If one examines the data of Lucchesi, Rawls and Maroni (1974, Nature 248:564), Abraham and Lucchesi (Genetics 78:1119), and Ananiev and Gvozdev (1974, Chromosoma 45:193-201), s/he will find that the dosage of the X chromosome

produces a slight positive "dosage effect" on autosomal genes in Drosophila larvae. The dosage of the X shows an inverse effect on some autosomal genes when adult flies are assayed.

Another way to reconcile the aneuploidy/ploidy paradox is to consider that the processes that control cell volume are independent of IE regions. Thus triploid cells would have correspondingly larger cells. Since the concentration of the IE region products (3/2 as much in 3/2 the volume) would be equal to each gene present in the diploid, so the increase in structural genes (3/2) is directly expressed. These are only two ways to reconcile this apparent paradox.

Another explanation of dosage compensation deserves discussion. It postulates that the activity of X-linked genes is limited by gene products produced by autosomal genes (Maroni and Plaut, 1973, Chromosoma 40:361). Since the autosomal level in males and females is equal, the females' two X's would be limited to the same level as the males' one X. This hypothesis suffers from the fact that screens for dosage dependent regions affecting X linked genes have failed to show any such regions (Rawls and Lucchesi, 1974, Genetical Research 24:59). In the data, however, are values consistent with the presence of IE regions. The limiting factor hypothesis does not account for IE regions and their roles.

Nor does this hypothesis account for sexual dimorphism of autosomal genes. In the vast majority of sexual dimorphisms of autosomal mutants, the males are more like wild type than are females even though the gene dosage is the same in the two sexes (Goldschmidt, 1955, Science; Smith and Lucchesi, 1969, Genetics 61:607). If positive factors on the X controlled these autosomal genes, males would be either less like or equal to wild type as females, depending upon whether the positive factor itself is not dosage compensated or dosage compensated, respectively.

If dosage compensation is to be explained as the natural consequence of a cancellation of structural gene dose and inverse effect, certain predictions would follow:

1) There should be IE regions on the X that affect autosomal genes. Since the inverse regions are reduced in males, the affected autosomal genes would be expressed at greater levels in males than in females. Phenotypic and quantitative studies have shown autosomal genes to be expressed more strongly in males (Smith and Lucchesi). Biochemical studies show some indication that this is so (Lucchesi, Rawls, Maroni, 1974, Nature 248:564). However, the degree of the phenomenon could have been obscured in these studies by correcting enzyme levels per mg protein, since the protein per cell in males and females may change in the same direction as the autosomal genes tested.

2) Regardless of the composition of the X in various species of Drosophila, there should be dosage compensation. Abraham and Lucchesi (Genetics 78:1119) present evidence that this is true, although their data may be obscured by correction against total protein in males and females.

The biochemical studies may not reveal very accurately the true nature of the phenomenon. Since the hypothesis predicts that many autosomal genes will have increased expression in males, the total protein/cell ratio may be different in males and females. Thus "correcting" enzyme levels by protein per extract may cancel out to some degree the level of increase. Also these studies are complicated by the fact that males and females possess different anatomies. Undoubtedly, many enzymes are expressed differently in these different tissues and organs. For these reasons, biochemical comparisons of male and female flies should be interpreted with great circumspection.

It may be that IE regions on the X would be indirectly responsible for sex determination in Drosophila. Since it appears that IE regions themselves show a dosage effect, they would increase the level of expression of many autosomal genes in adult males as compared to females. In triploids the increase of 2X3A over 3X3A would not be as great as 1X2A over 2X2A and hence an intersex would result. Genes such as transformer, intersex, and masculinizer that convert females to

malelike with little or no effect on males may be major inverse "genes" (or other genes that affect their action) that have mutated to a hypomorphic allele. One would predict from such an hypothesis that they would raise many enzyme levels in genetic females but not in males. Komma (1966, Genetics 54:497) claimed transformer raised the level of G6PDH in females but not in males. 6PGDH was not affected in either. Smith and Lucchesi showed that transformer increased the expression of autosomal glass mutants in females more than in males. Since genetic males already have presumably many inverse effect regions varied in relation to normal females, varying another, even though major, might not produce as radical a difference. In females, the transformer gene would be the first inverse "gene" varied and as a result a larger increase would be seen. The mutant doublesex, which converts both males and females to intersexes, is not readily understood by this hypothesis, although it also produces a greater gene expression in genetic females than in males.

Evidence that dosage compensation, sexual dimorphism of autosomal mutants and sex determination are affected by the same mechanism comes from studies of the effect of temperature on these processes. Lee (1968, Genetical Research 11:115) presented evidence that at high temperature, dosage compensation of an X linked gene was lost, whereas it occurred at low temperature. Smith and Lucchesi showed that sexual dimorphism of autosomal glass mutants was less evident at high temperature than at low. Finally, Dobzhansky (1930, Am. Naturalist 64:261) showed that low temperatures caused intersexes to become more male-like while high temperatures had a feminizing effect. If low temperatures enhance the inverse effect and high temperatures decrease it (there are no data available), and if the inverse effect is responsible directly or indirectly for all three phenomena, these results would be found.

In conclusion, it should be stated that much research is needed to clarify the nature of the inverse effect. Its apparent presence in such diverse higher organisms as maize and Drosophila suggests that it has a central importance in gene expression and possibly differentiation. This report hopefully has pointed out this possibility.

James A. Birchler

Chromosome elimination from a structurally modified chromosome 9

In the high-loss strain first studied by Rhoades, Dempsey, and Ghidoni in 1967, a knobbed chromosome 9 undergoes chromatin elimination in the second division of microspores with two or more B chromosomes. One of the two sperm has a normal chromosome and the other a deficient one. Fertilization of egg and polar nuclei by the dissimilar sperm appears to be random. Breaks at any position proximal to the Yg2 locus in the short arm of 9 produce a deficient chromosome which results in a yellow-green F_1 seedling in testcrosses when the deficient sperm unites with the egg. When the deficient sperm with a freshly broken end fuses with the polar nuclei, break positions can be localized to specific regions in the arm. A break between C and the knob (region 1) gives a broken chromosome with the C and Wx alleles, which undergoes the bridge-breakage-fusion cycle in the endosperm mitoses resulting in kernels with variegation for C and Wx. Breaks between C and Wx (region 2) yield wholly colorless kernels which are variegated for Wx or wholly Wx when the broken end is capped, while breaks proximal to Wx (region 3) give colorless and waxy endosperms (Table 1).

Following a cross of yg2 c wx with pollen from a K^L Yg2 C Wx high-loss plant, one of the seedlings from a colored kernel (no loss in the endosperm) exhibited the recessive yg2 phenotype--i.e., it had received a chromosome 9 deficient for the Yg2 locus from the pollen parent and a normal 9 with the yg2 allele from the egg parent. This particular plant (33166) had normal appearing pollen. When used in reciprocal testcrosses, 33166 gave approximate 1:1 ratios for C:c and for Wx:wx

Table 1. Distribution and frequency of breaks in the short arm of chromosome 9 in endosperms coming from the cross of c wx ♀ X K^L Yg2 C Wx ♂ of the high-loss line with four or more B chromosomes.

	Kernel Phenotypes					
	C, Wx (no breaks)	C-c, Wx-wx (breaks in region 1 distal to C)	c, Wx-wx (breaks in region 2)	c, Wx (capped breaks in region 2)	c, wx (breaks in region 3)	Σ
No. kernels	5342	72	52	20	1865	7351
% of total	72.6%	1.0%	0.7%	0.3%	25.4%	
% of loss kernels		3.6%	2.6%	1.0%	92.8%	2009

both as female and as male parent. Clearly, this chromosome 9 was deficient for Yg2 but had the C and Wx alleles, and the distal deficient segment carried no genes essential for male and female gametophyte development and functioning. Surprisingly, the C kernels from the male testcross included many which had a bridge-breakage-fusion type of variegation for the C and Wx loci. The loss phenomenon was not expected in this cross since the deficient 9 presumably had lost the terminal heterochromatic knob when the yg2 deficiency was produced. Cytological examination revealed that the 33166 chromosome was of the same length as a normal knobless 9 but possessed at the tip of its short arm a small heterochromatic knob--i.e., the 33166 chromosome, deficient for a short terminal piece including the Yg2 locus, had been capped by a small knob of uncertain origin. It may represent the small knob at the end of 7S since no chromosome of the high-loss line other than 9 and 7 had terminal knobs, but this surmise has not been verified. We consider it unlikely that the small knob on the 33166 chromosome is a remnant of the large knob on the parental chromosome 9 because such a constitution would require two closely spaced breaks at the tip of 9S accompanied by the deletion of the acentric fragment with most of the knob.

The possibility that the unexpected mosaic kernels were due to a modified type of chromatin elimination was investigated by comparing the behavior of a normal K^L9 and the 33166 chromosome in heterozygotes with 1B or 3B's. Table 1 shows the loss rate in K^L9/K^L9 homozygotes from previous studies while Tables 2 and 3 give the data from male parents heterozygous for K^L9 and 33166, having 3B's and 1B,

Table 2. Distribution and frequency of breaks in the short arm of chromosome 9 in endosperms coming from the cross of Yg2/yg2 c wx ♀ X K^L Yg2 C Wx/33166 C Wx ♂ with 3 B's and a modified high-loss background.

	Kernel Phenotypes					
	C, Wx	C-c, Wx-wx	c, Wx-wx	c, Wx	c, wx	Σ
No. kernels	4030	543	11	4	147	4735
% of total	85.1%	11.5%	0.2%	0.1%	3.1%	
% of loss kernels		77.0%	1.6%	0.6%	20.8%	705

respectively. In heterozygous plants, breaks at the second microspore mitosis may involve either the K^L9 or the 33166 chromosome. Previous studies have shown that loss of K^L9 occurs only in spores with two or more B's. Therefore, no loss of K^L9 is expected in the 1B plants of Table 3 and a reduced loss frequency should be

Table 3. Distribution and frequency of breaks in the short arm of chromosome 9 in endosperms from the cross of Yg2/yg2 c wx ♀ X K^L Yg2 C Wx/33166 C Wx ♂ with 1 B chromosome. These plants are sibs of those in Table 2.

	Kernel Phenotypes					
	C, Wx	C-c, Wx-wx	c, Wx	c, Wx-wx	c, wx	Σ
No. kernels	1565	63	0	0	3	1631
% of total		3.9%			0.2%	
% of loss kernels		95.4%			4.6%	66

detected in the 3B plants of Table 2. Since over 90% of the breaks in the K^L9 chromosome are proximal to the Wx locus in microspores of K^L9 homozygotes of the high-loss line, it appeared likely that the minority class of c wx kernels of Table 2 came solely or chiefly from chromatin loss in 2B spores with the K^L9 chromosome. This possibility was tested by determining the genotype of the embryos in C-c Wx-wx and in c wx kernels. If, for example, the c wx kernels all came from loss of the K^L9 chromosome with the Yg2 allele, the ensuing seedlings (no loss in embryo) would be green while involvement of the Yg2-deficient 33166 chromosomes would yield green and yellow-green seedlings in a 1:1 ratio, due to heterozygosity for yg2 in the tester female parent. The tests made to date reveal that in microspores from K^L9/33166 heterozygotes the vast majority of breaks occur in the 33166 chromosome and that the K^L9 chromosome has a low rate of chromatin elimination in 2B microspores. This can be ascribed to the changed background of genetic modifiers in the K^L9/33166 heterozygotes, relative to those in the original high-loss strain, which differentially affects the K^L9 and the 33166 chromosome. Moreover, as is clearly shown in Table 3, the 33166 chromosome is capable of loss in spores with only 1B, unlike the K^L9 chromosome. In high-loss strains, a normal chromosome 9 with a small terminal knob (K^S9) undergoes a low percentage of chromatin elimination at the second microspore division. However, in heterozygotes for K^L9 and 33166, the 33166 chromosome with a small knob comparable in size to K^S has a rate of loss much greater than that of the normal chromosome 9 with its large knob.

The K^L normal 9 and the derived 33166 chromosome differ in the distribution of breaks in 9S, as is shown by a comparison of the data in Tables 1 and 2. In the high-loss strain homozygous for the K^L9 chromosome, breaks between the C locus and the terminal knob at the second microspore mitosis are relatively infrequent (3.6% in Table 1). The vast majority of breaks occur in the proximal half of the arm to the right of the Wx locus. In contrast are the data on break position in Table 2 (where breaks are occurring primarily in the 33166 chromosome). 77.0% of the breaks are distal to C and only 20.8% take place in the proximal half of 9S. Clearly the position of the primary break at the second microspore division differs in the K^L9 and 33166 chromosome and the susceptibility of different regions of 9S to breakage is a heritable trait.

Expression of the differential breakage pattern by which the two chromosomes differ is restricted to the second microspore division. For example, if a chromosome with the C allele and a freshly broken end is produced at the second microspore mitosis, fusion of the two broken ends takes place following replication. The ensuing chromatid type of bridge-breakage-fusion cycle in the developing endosperm results in a kernel mosaic for colored (C) and colorless (loss of C) sectors. The pattern of mosaicism is determined by the position of bridge breakage and there is a wide range in the size and distribution of colored spots between different mosaic kernels. However, the mosaic patterns amongst kernels arising from breaks distal to C in either the original high-loss chromosome 9 or in the 33166 chromosome are not sensibly different. In other words, although there is a

striking difference between the two chromosomes in the position of breaks at the second spore mitosis, it is a difference which is not maintained during endosperm ontogeny.

No data are presently available for rate of chromatin loss in the microspores of plants homozygous for the 33166 chromosome since these homozygotes give lethal albino seedlings, as do wd/33166 heterozygotes. Viable (green-white striped) homozygotes are being synthesized by adding McClintock's Wd ring chromosome which possesses the Yg2 allele.

The data presented in this report suggest that a mechanism other than the postulated dicentric resulting from delayed knob replication in the second microspore division may be responsible for the inception of the bridge-breakage-fusion cycle. The nature of this mechanism can only be conjectured at this time, but it may be somewhat akin to the situation described by McClintock where breaks occur specifically at the Ds locus, which is transposable. There are significant differences between our high-loss phenomenon and Ac-Ds. In the former the position of the primary break, although occurring preferentially along the chromosome, is not restricted to one specific site as is true for Ds. Another difference is that the high-loss phenomenon involves the interaction of two known heterochromatic elements, B chromosomes and knobs, while the nature of Ac and Ds remains to be established. A third difference is the stage in development at which the primary breaks occur. In the high-loss phenomenon, the initial break takes place at one specific cell division while Ac-Ds breaks are not so restricted.

We have recently found a derivative of 33166 in which the majority of breaks take place between C and Wx rather than between Wx and the centromere for $K^{L}9$ and between the knob and C for 33166. The response of this new chromosome to dosage of the B chromosome and any change in chromosome structure are being investigated.

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Analysis of the activity and isozyme patterns of alcohol dehydrogenase in a polyploid series of maize

Alcohol dehydrogenase (ADH, E.C.1.1.1.1.) is an advantageous model for studying polyploidy effects at the molecular level. The maize tetraploid lines used in this analysis were obtained by the method of nonreduced gametes. The relative contributions of diploid lines to the genotypes of the tetraploids (RCD) were different (from 7/8 of the genotype to 15/16). The RCD depended on the number of backcrosses of the tetraploid to the initial diploid line. The following diploid-tetraploid pairs were analyzed: Gb627(2x)-Gb627(4x), Gb300(2x)-Gb300(4x).

We also used diploid line R109 and line R109(4x), which was identical to this diploid line. R109(4x) was obtained by heat shock (L. F. Randolph, 1937).

Haploids were obtained by the method of Chase (Chase, 1947; Nanda, Chase, 1966) modified in our laboratory. The homozygous Adh-S Adh-S diploid line W155 was crossed to the homozygous Adh-F Adh-F marker line with purple endosperm and embryo. Haploid plants were identified by the absence of coloring in embryos at the seed stage, with subsequent electrophoretic analysis of ADH. Alcohol dehydrogenase activity was assayed in the scutellum of dry maize seeds.

The results of the comparisons of ADH activity in diploids and tetraploids of maize lines are given in Table 1.

We introduced index α = T-D/D to estimate quantitatively differences in the activities of this enzyme in diploids and tetraploids of the compared pair (T and D are the specific activities of the tetraploid and diploid). This index varies

Table 1. ADH specific activity in diploids and tetraploids of maize lines (nmoles NAD ÷ min/mg of protein).

Lines	RCD	ADH specific activity	$\alpha = \frac{T - D}{D}$
R109(2x)		925 ± 66	
R109(4x)	1	570 ± 50	0.38
Gb627(2x)		975 ± 46	
Gb627(4x)	0.938	779 ± 42	0.20
Gb627(2x)		975 ± 46	
Gb627(4x)	0.875	816 ± 44	0.16
Gb300(2x)		380 ± 58	
Gb300(4x)	0.875	420 ± 56	0.10

P = 0.95

largely in different lines. The data obtained evidence that the degree of the differences in ADH activity between diploids and tetraploids is determined by genotypic environment.

Table 2 summarizes the results of the comparisons of ADH activity in haploid and diploid plants of line W155 and in plants of the marker line.

Table 2. ADH specific activity in haploid and diploid plants of line W155 and in plants of the marker line (nmoles NAD ÷ min/mg of protein).

Lines	ADH specific activity
W155 (2x)	848 ± 50
maternal haploids W155(x)	810 ± 45
paternal haploids W155(x)	870 ± 70
marker line (2x)	718 ± 45

P = 0.95

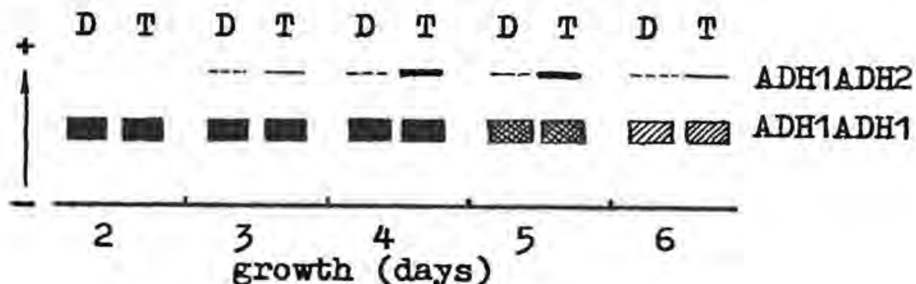
As Table 2 shows, ADH activity in maternal and paternal haploids is close to its activity in the diploid line W155 and significantly different from ADH activity in the marker line. This permits us to exclude, in this case, the maternal effect in ADH activity. Thus, there were no differences in ADH activity in line W155 diploids and haploids. This was attributed to either genotypic features of the line or to some specificities in the transition from the diploid to the haploid level.

Comparisons of ADH activity in diploids and tetraploids during growth demonstrated that differences in ADH activities between diploids and tetraploids are conditioned by the developmental stage and environmental conditions.

The relative expression of ADH at the structural loci was investigated, in view of the fact that ADH in maize is controlled at two non-linked loci, Adh and Adh2 (Schwartz, 1969; Freeling and Schwartz, 1973). The ADH isozyme patterns in diploids and tetraploids of line R109(2x) and R109(4x) during growth are represented schematically in Fig. 1.

The relative intensity of the heterodimeric zone in tetraploids is higher than in diploids at all growth stages. This indicates that changes in ploidy level are associated with changes in the relative expression of genes Adh and Adh2.

Fig.1. ADH isozyme patterns in diploids and tetraploids of maize line R109 during growth.



Thus, maize ADH is a good case illustrating that polyploidy changes ADH activity in maize tissues and modifies the phenotypic expression of genes controlling ADH. It may be suggested that polyploidy affects similarly the activity of many enzymes, which control biochemical processes underlying the formation of morphological and physiological characters in plants.

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Because of the strategic role of nitrate reductase in nitrogen metabolism, relationships between activity and productivity or quality of the kernel have been the subject of much research. Deckard, Lambert and Hageman (Crop Science 13:343, 1973) found in six genotypes of maize hybrids that the "nitrate reductase activity of the total leaf canopy showed a significant positive correlation with grain protein (kg N/ha), grain yield and total reduced N in the vegetative material and grain and stover at maturity."

In maize seedlings this enzymatic activity reaches a maximal level eight to ten days after sowing, and its measurement might be a successful means for an early selection of genotypes with high nitrate reductase activity. As a first step, we looked for differences between three groups of inbred lines and for variability within each group.

The first group is twelve "current lines" (five from our Station and seven released American lines), which are the check material. The second group is twelve "high protein lines" (eight from our Station, three from Eastern Europe and the origin: Illinois High Protein), which could possibly exhibit high nitrate reductase activity and the third group is twelve "ear prolific lines" (seven from our Station, two from Eastern Europe and three American lines), which were selected to obtain prolific hybrids with broad adaptability and strong productivity in a high plant population. In a high plant population with a reduction of light by mutual shadowing, current hybrids cannot reduce nitrates efficiently enough to insure a high productivity; under the same conditions prolific hybrids might possibly have either a higher nitrate reductase activity or enzymatic activity less sensitive to lowered light intensity.

We used a modified in vitro assay (Hageman and Hucklesby, Methods in Enzymology 23:491, 1970) on leaves of ten-day-old seedlings grown in a growth chamber. Two sets of experiments were conducted, and the results are presented in Table 1.

Table 1. Averages of early nitrate reductase activities in 36 inbred lines of maize.

Group	Inbred Lines	Nitrate Reductase Activity (micromoles NO_2^-/hr/g fresh wt.) Set I average of 3 assays	Set II average of 3 assays
current	A619	1.86	2.72
	C103	2.17	2.76
	FR64A	6.07	3.18
	Oh7	3.01	3.06
	Oh43	2.05	3.81
	WF9	1.42	2.20
	W117	1.88	2.33
	F485[a]	2.30	3.05
	F497[a]	2.86	3.19
	F502[a]	1.37	1.52
	F515[a]	3.60	3.66
	F546[a]	3.13	3.31
	group average	2.64	2.90
high protein	IHP	2.96	5.10
	BGR934[b]	2.50	4.21
	Ky303[c]	1.46	2.50
	YuT789[d]	3.71	3.49
	FP5[a]	2.83	3.68
	FP10[a]	1.27	3.11
	FP19[a]	2.28	4.73
	FP23[a]	1.37	3.33
	FP32[a]	2.48	4.32
	FP99[a]	4.13	5.12
	FP313[a]	4.89	4.47
	FP345[a]	2.63	3.48
	group average	2.71	3.96
ear prolific	A632	3.58	4.23
	B77	2.26	5.54
	Blue Prolific[e]	3.40	5.63
	F576[a]	3.20	4.37
	T115	1.79	2.09
	Yu1071[d]	2.94	3.67
	F576x1278-93[a]	3.17	7.15
	F576xBx55-85-53[a]	3.90	6.82
	F576xB59-6[a]	3.14	8.38
	F576xA632-52[a]	3.51	8.16
	F564xF576xF49-10[a]	2.93	6.35
	A632xB59-88[a]	4.78	9.50
	group average	3.22	5.99

[a]Inbred lines from the Plant Breeding Station, Montpellier (France)
[b]Inbred lines from the Plant Breeding Station, Roussé, (Bulgaria)
[c]Inbred lines from the Plant Breeding Station, Kouban (North Caucase)
[d]Inbred lines from the Plant Breeding Station, Zemun-Polje (Yugoslavia)
[e]Inbred line from the Genetic and Plant Breeding Institute, Sofia (Bulgaria)

There are differences in enzymatic activity between the two sets of assays that can be explained by slight differences in growing conditions and by conservation of the samples. However, the correlations between the averages of the two sets are +0.93, +0.95 and +0.97 for the current, high protein and prolific lines, respectively. For all lines, there is considerable variability of the nitrate reductase activity. A wide range of activity (up to six-fold) is observed in the two sets. In the second one, this range goes up to 2.5-fold in current and high protein lines and up to 4.6-fold in prolific lines. Statistical analyses have supported the following conclusions: the activities are significantly different between lines and between groups of lines; the prolific lines have a higher nitrate reductase activity than either the current or the high protein lines; the activities of the high protein lines do not seem different from the current lines.

The low nitrate reductase activity of high protein lines is surprising. However, we must remember that the reduction of nitrates is only one step leading to a high protein content in the kernel. The capacity to mobilize and carry the reduced nitrogen might also be of great importance and could therefore explain the degradation of nitrogen compounds during kernel filling by assaying the protease activity in leaves.

In the prolific lines the high nitrate reductase activity is of great interest. The adaptation to high plant population seems to be analogous in these and certain American lines (Zieserl, Rivenbark and Hageman, Crop Science 3:27, 1963); we assume that this material maintains a sufficient level of nitrate reductase activity for a high productivity even if it is affected by shading when sown densely. With this material we intend to study the relationship between early nitrate reductase activity and ear prolificacy. The variation of nitrate reductase activity with the quantity of reduced nitrogen in the plant during the next stages of growth will also be followed up.

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A method to estimate the degree of semisterility on ears from a heterozygous translocation

The degree of semisterility of a maize stock with a heterozygous translocation (N/T) is difficult to estimate when the kernels are fully mature. This is because each well-developed kernel may occupy the space of two embedded ovaries where one fertilized ovary has aborted. In the early stages of ear development, 2 weeks following pollination, the semisterility (%) of an N/T maize ear could be easily determined. This is because the functional fertilized ovary or the zygote only occupies one ovary position at this stage. However, this method of estimating has the disadvantage of sacrificing a maize ear before the ear reaches maturity.

In order to overcome this difficulty and avoid sacrificing the mature ear, we have adopted an alternative method. First, we have assumed that:

$$\text{Semisterility (\%)} = \frac{\text{total no. of developed kernels}}{\text{no. of ovaries per row x no. of rows}} \times 100$$

The total number of developed kernels and number of rows per examined ear is recorded. In order to determine the number of ovaries per row, the kernels of two or three rows from different areas of an examined ear are removed. Then, this kernel-less cob area is scraped with a scalpel to expose the cupules (Galinat, 1956, Botanical Museum Leaflets 17:221). This process reveals the site of each ovary attachment. The numbers and types (either fertile or aborted zygotes) of ovaries per row are obtained by averaging the number of ovaries in these two or three rows.

It was found that the functional fertilized ovary or zygote is embedded much deeper on the cob than those that are aborted.

Sun-Yuan Hsu and Peter A. Peterson

A method of investigating anthesis

This note is a report on an observation that is common to all maize geneticists but has rarely been quantified. A method has been adapted for investigating anthesis that appears useful for examining several features of pollen shedding that include: 1) sequence of anther dehiscence (the order of the onset of anther dehiscence among four spikelet positions of a tassel--see figure), 2) the time period of anthesis of a tassel (number of days when 95% of the spikelets of a tassel have completed anther dehiscence), and 3) the time interval (U:L) (number of days) that separates the upper (U) and lower (L) flowers of the same spikelet.

For anther dehiscence studies, spikelets within each of the four positions were randomly selected. In order to identify the date of the first shedding anther of each position, Dennison marking tags (size 3 x 2 cm^2) were tied in each position.

To identify the time interval (U:L) separating pollen shedding between upper (U) and lower (L) flowers, a color code was used to mark spot(s) (Sanford's Sharpie marking pen) on the surface of the inner glume of the examined spikelet. A blue spot marked the first day of pollen shedding of the upper flower of a spikelet; a red spot was used on the second day of the lower flower if the same spikelet did not shed pollen, and the color marks were continually applied until the time the lower flower began to shed pollen. The number of days separating the upper and lower flowers in pollen shedding was obtained by counting the number of color spots on a spikelet. Ten to 20 spikelets from each of the four positions of a tassel were sampled. The time interval (U:L) separating two florets of the same spikelet was estimated by averaging the time interval (U:L) for the examined spikelets at a given position within a tassel. This method provides a convenient way to handle a large number of samples (spikelets) in the field within a relatively short time.

Based on the investigation of a total of 4,941 spikelets among 25 genotypes, the average time interval separating pollen shedding between upper and lower flowers of the same spikelet is 2.06 days (Table 1). There is a longer time interval among tetraploids and Mexican varieties than among inbreds and hybrids. This time interval is longer in position 3 and 4 than in positions 1 and 2 (Table 2)

When genotypes are pooled and observations are averaged, the first pollen-shedding anther is found to be in position 2, followed by 1, then 3 and, finally, 4 (Table 3).

The time period of anthesis of a tassel was similar among inbreds, tetraploids, and hybrids (Table 4); there was a significantly longer time period in Mexican varieties.

Sun-Yuan Hsu and Peter A. Peterson

Table 1. Comparison of the mean time interval (U:L) that separates pollen shedding between upper and lower flowers among 4 genotype groups.

Genotype groups	No. spikelets investigated	Interval (U:L) (days)	LSD (.05)[1] = 0.15
Inbreds	3,055	1.95	b
Tetraploids	689	2.48	a
Mexican varieties	662	2.39	a
Hybrids	535	1.79	c
Total	4,941	2.06	

[1]LSD(.05) = least significant difference at 5% level. Values with the same letter are not significantly different ($p < .05$).

Table 2. Comparison in the mean time interval (U:L) (in days) that separates pollen shedding between upper and lower flowers among 4 different positions.

Positions	Interval (U:L)[1] (days)	LSD (.05) = 0.10
1	1.94	b
2	1.97	b
3	2.13	a
4	2.22	a

[1]Average time interval (U:L) of the sampled spikelets of 25 genotypes for each position.

Table 3. The mean sequence of the onset of anther dehiscence within a tassel.

Spikelet position	Anther dehiscence (i^{th} day)	LSD (.05) = 0.16
1	1.53	b
2	1.00	a
3	2.20	c
4	3.55	d

Table 4. Comparison of the mean time period of anthesis among 4 different genotype groups.

Groups	No. of tassels investigated	Period of anthesis (days)[1]	LSD (.05) = 0.61
Inbreds	45	6.68	b
Tetraploids	9	6.89	b
Mexican varieties	12	10.17	a
Hybrids	9	7.00	b

[1]Mean of the time period of anthesis of the three tassels for each of the genotypes of a group.

The relative duration of several cytological stages during microsporogenesis

Following their survey of cytoplasmic male sterility Laser and Lersten (1972, Bot. Rev. 38:425) proposed a diagrammatic scheme of normal microsporogenesis (MSG) which included eight cytological stages beginning with the onset of the sporogenous cell division and ending in the trinucleate pollen stage. In our study the determination of cumulative frequency--an indicator of microspore stage development--begins with the leptotene stage instead of the sporogenous cell stage and ends in the trinucleate pollen stage when the pollen is shed.

The relative frequencies of each of the eight cytological stages were estimated from a total of 3,404 upper flowers and are listed in Table 1, column 4. Approximately one-fifth (19.83%) of the MSG period was spent in the process of meiosis and within this meiotic period 92.3% (18.30/19.83) occupied meiosis I. Meiosis II, on the other hand, occupied only 1.53% (19.83-18.30) of the total MSG period or 7.7% (1.53/19.83) of the meiosis period.

Table 1. Duration of meiosis and pollen maturation stages during microsporogenesis.

	Relative duration	Stage	Relative frequency (%)	Cumulative frequency
Meiosis	19.83%	Meiosis I	18.30	18.30
		Meiosis II	1.53	19.83
Pollen maturation	80.17%	Tetrad	6.64	26.47
		Early non-vacuolate microspore	9.64	36.11
		Vacuolate microspore	25.18	61.29
		Vacuolate pollen	10.34	71.63
		Engorged binucleate pollen	14.98	86.61
		Tri-nucleate pollen	13.39	100.00

Approximately four-fifths (80.17%) of the whole MSG period is spent in the maturation of pollen grains. The tetrad stage consumes 6.64% of the MSG period where four newly formed young microspores are still held together by callose. The length of the other stages can be seen in Table 1.

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Comparison of mutation rate and mutations in selfed and outcross progeny of mutator stocks

In previous reports (MGCNL 45:81-87, 1971, 49:73-79, 1975) we have described the effects of a mutator system (*Mu*) in corn. New mutants were observed in self-pollinated progeny of plants derived from the outcross of plants carrying *Mu*. Outcrosses of *Mu* plants showed a mutation rate from 15-30 times higher than that observed in non-*Mu* lines. In making these outcross tests, the *Mu* parents were always self-pollinated and scored for the presence of any mutations that might complicate the outcross results. Outcrosses were routinely discarded if the *Mu* selfs segregated for mutants.

Since selfs and outcrosses of the *Mu* stocks were made at the same time with a given pollen sample, the same population of mutants might be observed in selfs of

the selfed progeny as in the selfs of the outcross progeny. This would only be the case if mitotic (premeiotic) mutations were occurring that might give rise to tassel sectors heterozygous for the mutation. Thus, the appearance of similar mutants in these two progenies would suggest that Mu might be acting during mitosis. This occurrence is not conclusive proof, since we have shown (MGCNL 50: 68-70, 1976) that mutants in a given outcross family with similar phenotypes are not necessarily allelic. Some instances of allelism have been observed, however, suggesting that mitotic mutations can occur.

A 50-seed sample from the selfed ears of Mu lines that had been tested in outcrosses was sown. The resulting plants were self-pollinated and the self progeny were scored for seedling mutants. The array of mutants found in the progeny of the selfed Mu plants was compared with that observed in the outcross progeny of the same Mu plants. The outcross progeny and the self progeny were grown in different years. The person scoring the self progeny was not aware of the mutants that had been found in the outcross progeny, which had been tested in a previous year.

In Table 1, the mutation rates and mutant phenotypes in outcross and self progeny of Mu plants are compared. Fifty-seed samples of both outcross and self progeny were planted. The self progeny had consistently fewer ears than the outcrosses due to poorer germination and weaker plants that failed to produce ears as a result of inbreeding depression and/or weak mature plant mutants (e.g., dwarfs, runts, male steriles, etc.). The total mutation rate of self progeny is slightly higher (but not significantly) than that of the outcross progeny. A priori, the mutation rate of the self progeny would be expected to be twice that of the outcrosses since mutation would be expected in both male and female gametes of selfed Mu plants. The results suggest that Mu induced changes may not occur in female tissue. Tests specifically for female transmission of mutations were made this past summer but the results have not been analyzed as yet.

In all but one stock (1040-1), mutants with similar phenotypes were found in both outcrosses and self progeny. As pointed out above, such results would be expected if Mu is inducing mitotic mutations. Positive allele tests of similar outcross and self-mutants will be needed before definite conclusions can be made in this regard.

Donald S. Robertson

The persistence of Mu activity in the absence of y9

Early analyses of Mu stocks had suggested that mutability was associated with the y9 locus (MGCNL 45:81-87, 1971). However, later tests (MGCNL 49:73-79, 1975) demonstrated that non-y9 plants could also transmit mutability. The non-y9 plants were siblings to heterozygous y9 plants in the mutator stocks. In such stocks, all plants, y9/+ or +/+, transmitted mutability. There are several explanations for the deviation from the Mendelian pattern of transmission expected if the y9 locus (or something closely linked to it) was responsible for the observed mutability. One of the possibilities is that a paramutagenic-like change had occurred in the heterozygous y9 plant of the previous generation that were used to produce the tested generation. If in the previous generation, Mu on the y9-bearing homologue had induced a change on the non-y9 homologue converting mu to Mu, both y9 and non-y9 bearing gametes would carry an Mu and all the plants of the tested family would exhibit Mu activity. This suggested change of mu to Mu could be a permanent change or a transitory one. In the latter instance, the induced Mu may weaken in its mutator ability with time resulting in lower mutation rates or even a complete loss of the ability to induce mutations in later generations.

To test for the persistence of the Mu activity, plants from the outcross non-y9 mutator plants were again outcrossed to test for mutability in the next generation (second generation). A comparison of first and second generation mutability is

Table I. Comparison of phenotypes of mutants in outcross and selfed progeny of Mu plants.

	Outcross Progeny			Selfed Progeny				
Mu Stock	Total Plants Selfed	Total Different Mutants[a]	% Different Mutants (Mutation Rate)	Total Plants Selfed	Total Different Mutants[a]	%[a] Different Mutants (Mutation Rate)	Number of Mutants with Similar Phenotypes in Common	Phenotypes of Common Mutants*
8266-9	45	4	8.9	28	2	7.1	1	pg
8266-12	37	2	5.4	30	1	3.3	(1)**	w vs. nlw**
8269-1	41	3	7.3	26	3	11.5	2	py, yg
8269-2	39	2	5.1	23	4	17.4	1	w
1040-5	41	5	12.2	23	1	4.3	1	l
1040-7	39	3	7.7	21	3	14.3	1	yg
8265-5	44	4	9.1	36	3	8.3	1 + (1)**	pg, w vs. nlw**
8265-11	37	4	10.8	11	4	36.4	2	w, py
8266-5	35	3	8.6	30	6	20.0	3	w, nlw, l
8269-9	38	4	10.5	30	4	13.3	3	w, l, pg
1040-1	40	3	7.5	20	0	0	0	------
1041-4	40	3	7.5	33	4	12.1	2	w, pg
Totals	476	40	8.4	311	35	11.3	16 (18)	

*pg = pale green, w = albino, nlw = narrow leaf albino, py = pale yellow, yg = yellow green, l = luteus, pg = pale green

**nlw is variable in expression. In the same family, expression can vary from a normal full size albino to a very narrow leaf albino seedlings. Ratios are frequently deficient for nlw seedlings. The numbers of extremely affected seedling will vary from family to family. In these two instances it is possible that w and nlw are variable expressions of the same phenotype.

[a]See MGCNL 45:81-87, 1971 for definition of "different mutants".

given in Table 1. In most instances second generation non-y9 stocks show a somewhat increased mutation rate over the first generation stocks. The exception to this was in the second generation lines derived from the 3123 first generation stock. Three of these second generation lines had no mutants. These three lines as well as some others that showed a substantially lowered mutation rate may possess a weakened induced Mu. However, these instances with zero or low mutation rates may just be due to sampling error. Instances have occurred of Mu outcrosses

Table 1. Mutability of the first generation outcross of non-y9 Mu lines compared with that observed in the second generation outcross.*

		First Generation			Second Generation		
First gen.	Second gen.	Plants selfed	Different mutants[a]	Mutation rate (%)	Plants selfed	Different mutants[a]	Mutation rate (%)
3120		35	3	8.6			
	1032-1				45	5	11.1
	1032-2				47	8	17.0
	1033-10				48	5	10.4
Total		35	3	8.6	140	18	12.9
3123		37	4	10.8			
	1034-1				32	3	9.4
	1035-2				45	1	2.2
	1035-9				44	2	4.5
	1035-4				44	5	11.4
	1035-6				48	0	0.0
	1035-5				48	0	0.0
	1035-3				48	0	0.0
Total		37	4	10.8	309	11	3.6
Total minus 0 mutants		37	4	10.8	165	11	6.7
3128		41	3	7.3			
	1036-3				29	3	10.3
	1037-2				46	5	10.9
	1037-1				43	6	14.0
	1037-4				43	1	2.3
	1037-6				39	3	7.7
Total		41	3	7.3	200	18	9.0
3134		38	4	10.5			
	1039-5				31	6	19.4
Total		38	4	10.5	31	6	19.4
Grand Total		151	14	9.3	680	53	7.8
Grand total minus 0 mutant stocks		151	14	9.3	536	53	9.9

*Second generation stocks between lines all are derived from the first generation stock indicated.
[a]See MGCNL 45:81-87, 1971 for definition of "different mutants."

that have one year given zero mutation rates but on retesting a second year revealed an elevated rate similar to other stocks. Since families are small (50 plants or less) and mutants, even in families with high rates, are few, it is not surprising that as a result of sampling error an occasional Mu family occurs in which no mutations are found (see following report).

In summary, there is no consistent diminution of the mutation rate in non-y9 lines in one generation. There is a suggestion that sporadic reduction may occur in some stocks but these need to be confirmed by further testing.

Donald S. Robertson

Studies on the apparent loss of mutability in Mu lines

In MGCNL 45:81-87, 1971, we indicated that most outcrosses of lines segregating for heterozygous y9 plants displayed mutability. However, we do have some long standing y9 stocks which seem to have lost the ability to induce mutation (MGCNL 49:73-79, 1975). Since the mutability in the mutator (Mu) y9 lines can be traced back to the original y9 material I received from Dr. Kermicle, and since the nonmutating y9 stocks came from the same source, it is obvious that y9 and mutability can be separated. But how and under what conditions is not known.

Occasionally in our mutator tests an outcross family (test family) will be found that gives no mutations. Do these represent instances where mutability has been lost? They could be. However, the test families are small (50 or less plants) and even at the most the numbers of mutants are few. Therefore, the principles of probability dictate that occasional test families with no mutations will be found even in Mu lines. Additional plants from four test families of Mu lines that gave zero mutation were grown. The results are summarized in Table 1. Two of the putative losses (viz., stocks 3111 and 3127) of Mu on further testing turned out to

Table 1. Additional tests of outcross Mu families that originally gave zero mutations.

	First Test			Second Test			Both Tests	
Mu Stock	Plants selfed	Diff. Muts.	Mut. Rate	Plants selfed	Diff. Muts.	Mut. Rate	Mut. Rate	Sibling Family Mut. Rate
3111	43	0	0	86	4	4.7	3.1	4.5
3119	33	0	0	90	1	1.1	0.8	4.5
3127	42	0	0	83	4	4.8	3.2	3.6
7501-02	85	0	0	67	0	0	0	3.5

actually have Mu activity. Therefore, the apparent lack of Mu activity in the original tests was probably due to sampling error. One stock (viz., 3119) gave ambiguous results. The 1.1% mutation rate of the second test is somewhat higher than previously observed control values (range from 0.1% to 0.5%). However, if the results from the first and second tests of this stock are totaled the mutation rate becomes 0.8%, which approximates control values. In the case of the 7501-02 stock there is apparently a bona fide loss of Mu. Both the 3119 and 7501-02 stocks will be outcrossed again, in the first stock to determine if there is a low level or a complete absence of Mu activity and in the second stock to test the permanency of the loss of Mu activity.

Donald S. Robertson

Tests for increased mutation rate in mutable controlling element stocks

A survey of seedling mutants isolated from the mutator line (Mu) indicates that 15% are mutable. Because mutability is a characteristic of mutants influenced by controlling elements, the relationship between Mu and other mutable systems such as Dt, En and Mp is of interest. This relationship can be tested in two ways: 1) by determining if controlled alleles of the above systems respond to Mu and 2) by using mutable controlling element stocks in a manner analogous to the Mu line to test for an increase in the mutation rate. Preliminary results of the latter test are presented here.

Plants were self-pollinated and crossed to a standard line, and a sample of the outcrossed seeds were self-pollinated. The following results were obtained:

Stock	Number of outcrosses tested	Total number of plants	Number of mutants	% mutations
Dt	8	342	0	0
En	8	343	0	0
Mp	8	318	0	0
Total	24	1003	0	0

It is clear that there is no dramatic increase in mutation rate in these stocks.

Peter Mascia and D. S. Robertson

Results of additional allele tests of mutants from the Mu line

The results of a number of allele tests of phenotypically similar mutants that arose in the same family from the mutator line were presented last year (MGCNL 50: 68-70, 1976). Several ambiguities were noted. In family 72-3120, 4 luteus mutants arose. All seemed to be allelic by process of elimination. Mutants 6 x 18 and 6 x 20 gave positive tests but crosses of 18 x 20 gave negative results. Also, tests in family 72-3125, in which 5 luteus mutants arose, were incomplete. Crosses of mutants 18 x 29 were not done, making uncertain the results of crosses 18 x 20, which gave one positive test, and 20 x 29, which gave three positive tests. The following table presents results of allele tests made this summer that clarify these inconsistencies.

Family number	Mutant numbers	Number of crosses	Number of positive tests	Probability of allelism
72-3120	18 x 20	15	8	1
72-3125	18 x 20	17	0	< .01
	18 x 29	9	0	< .01
	20 x 29	6	6	1

Thus in family 72-3120 the four mutants are allelic while in family 72-3125, of five mutants tested, only mutants 20 and 29 are allelic.

These results, and results of additional allele tests done this summer do not alter last year's conclusion that most mutations occur very late in development or during meiosis.

Peter Mascia and D. S. Robertson

Chlorophyll synthetic mutants in maize

The porphyrin molecule is ubiquitous in nature and its synthesis has been studied in a variety of organisms. The study of mutants has been instrumental in explicating porphyrin and specifically chlorophyll biosynthesis. In higher plants chlorophyll synthetic mutants have been studied extensively only in barley (see von Wettstein et al., 1974, Science 184:800 for information and references). The techniques used in studying barley mutants are directly applicable to corn. A preliminary report of some luteus mutants in corn is presented here. Figure 1 presents a summary of the chlorophyll pathway showing possible regulatory molecules

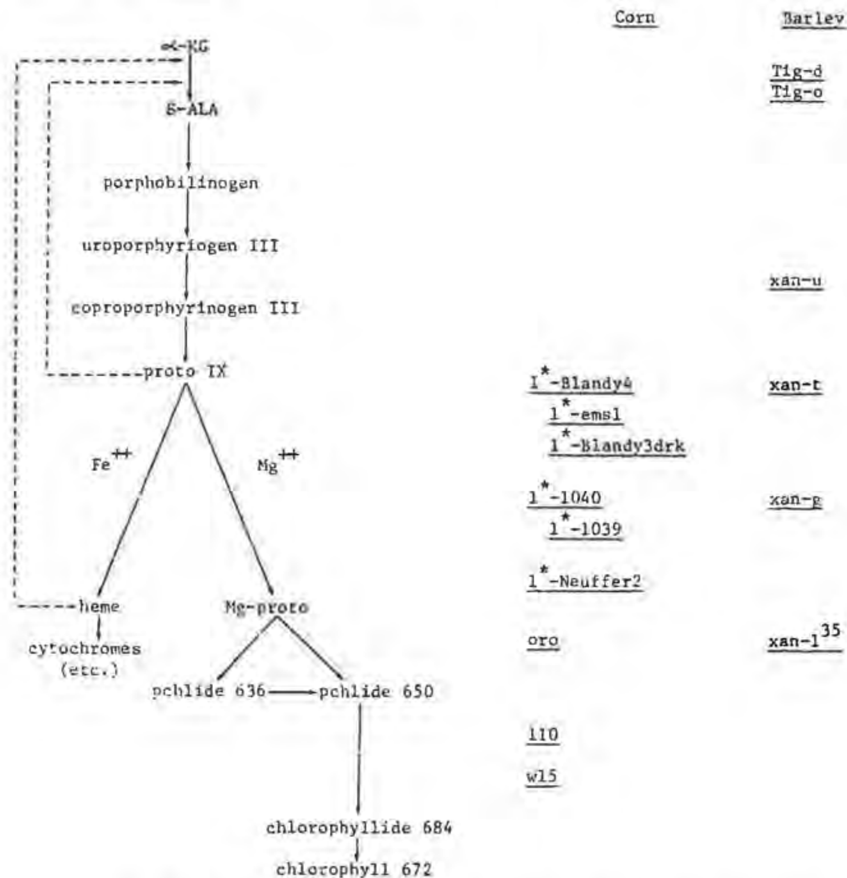

Figure 1. The biosynthetic pathway of chlorophyll and heme modified from several sources. See Wang, et al., Cell 6:75-85 (1975) for references. Mutants are positioned adjacent to proposed biochemical lesions. Dashed arrows indicate possible regulatory molecules.

Abbreviations:

α-KG	- α ketoglutarate
δ-ALA	- δ amino laevulinic acid
proto IX	- protoporphyrin IX
MG proto	- Magnesium protoporphyrin
pchlide	- protochlorophyllide holochrome

and mutants of corn and barley affecting various steps in the pathway. Table 1 presents the phenotypes of the mutants discussed. Only orobanche-modified (oro-mod) develops any visibly detectable chlorophyll at 2000 ft-c of light, albeit several do accumulate a small amount of chlorophyll not visible to the naked eye.

Dark grown material is particularly useful in determining the defective step in chlorophyll synthesis since higher plants are naturally blocked in the conversion of protochlorophyllide to chlorophyllide. The pathway is further inhibited by a feedback mechanism preventing δ-aminolevulinic acid synthesis. In normal plants

Table 1. Chlorophyll synthesis in light grown mutant and normal seedlings*.

Material	Phenotype	Average chlorophyll accumulated at 2000 ft-c, 10-14 days (mg/gm)**	% of normal
normal +/+; +/l	green	1.51 ± .06	100
l*-Blandy4	dark yellow	0	0
l*-5ms1	dark yellow	0	0
l*-Blandy3drk	dark yellow	0	0
l*-1040	dark yellow	0	0
l*-1039	dark yellow	.0079 ± .0004	.523
l*-Neuffer2	dark yellow	.0193 ± .0001	1.26
oro-mod	dark yellow, sometimes necrotic, some green detectable	.073 ± .004	4.83
l10	medium yellow	.0070 ± .0002	.464
w15	pale yellow	.0016 ± .0003	.105

*Mutants in groups are allelic.
**Determined using the procedure of Arnon, D.I. (Plant Physiol. 24:1-15, 1949).

this results in accumulation of a small amount of protochlorophyllide which is attached to holochrome protein (Rebeitz, C. and Castelfranco, P. Ann. Rev. Plant Physiol. 24:129-172, 1973). In vivo at least two types of protochlorophyllide holochrome are present. One, absorbing maximally at approximately 650 nm, is immediately convertible to chlorophyllide on exposure to light. The other is non-photo-transformable and has an absorption maximum of 636 nm. A variety of dark grown mutants has been examined by in vivo and in vitro spectroscopy (Table 2). The mutants discussed here can be divided into three physiological categories although genetically these classes overlap: 1) completely blocked mutants which accumulate no protochlorophyllide, 2) leaky mutants which accumulate protochlorophyllide 650, and 3) leaky mutants which accumulate reduced protochlorophyllide absorbing maximally at 636 nm.

The completely blocked mutants include l*-Blandy4 and alleles (l*-EMS1 and l*-Blandy3drk); l*-1040 ; and some recently found stocks of oro. The former two produce no detectable chlorophyll precursors while the latter produces a small amount of Mg-protoporphyrin probably corresponding to the amount of protochlorophyllide produced in normal leaves. We interpret this to indicate that protoporphyrin IX is involved in regulating the porphyrin pathway. The small amount of either Mg-protoporphyrin in oro or protochlorophyllide in normal seedlings probably results from competition between synthetic enzymes and regulatory molecules for the small amount of protoporphyrin IX produced. Presumably the heme pathway also competes for the same protoporphyrin IX, however, this has not been clearly established in higher plants.

The leaky mutants which accumulate protochlorophyllide 650 include oro-mod, l*-Neuffer2 and l*-1039, a leaky allele of l*-1040. Orobanche was originally found in a leaky background and was placed in this category. However, this leakiness may be due to the presence of a modifier gene. The recent observation of 3 non-leaky plants out of 12 mutant seedlings tested from a self pollinated ear approximates a 3:1 ratio among mutants, expected if the non-leaky oro plants are homozygous for

Table 2. Phenotypes of dark grown plants unfed and fed δ-aminolevulinic acid.

Material	Protochlorophyllide accumulated in dark grown plants (mg/cm)*	S.E.	% Normal	In vivo max dark grown leaves	In vivo max after exposure to lt for 1 min (25 ft.c.)	Predominant precursor(s) accumulated in dark grown leaves Fed δ-ALA**
1*-1040	0	0	0	none	none	proto IX (Figure 2d)
1*-1039	.0057	.0003	39.8	650	684	proto IX pchlide (Figure 2c)
+/+ +/1*-1039	.0142	.0007	100	650	684	pchlide (Figure 2a)
1*-1040/1*-1039	.0056	.0002	38.3	650	684	---
+/+ ; +/1	.0146	.0006	100	650	684	---
1*-Blandy3-drk	0	0	0	none	none	proto IX (Figure 2d)
+/+ ; +/1	.0161	.0035	100	650	684	pchlide (Figure 2a)
(1) oro	0	0	0	590	590	Mg proto (later proto IX)(Figure 2e)
(2) oro-mod	.0137	.001	124	650	684	Mg proto pchlide proto IX (not shown, like c plus e)
(3) +/+ ; +/oro	.0111	.001	100	650	684	pchlide (Figure 2a)
1*-Neuffer2	.0089	.0035	90.7	650	684	proto IX pchlide (Figure 2c)
+/+ ; +/1*-Neuffer2	.0098	.0007	100	650	684	pchlide (Figure 2a)
110	.0068	.0004	50.8	636	672	proto IX pchlide (Figure 2b)
110/+ ; +/+	.0133	.001	100	650	684	pchlide (Figure 2a)
w15	.0041	.0007	23.5	636	672	proto IX pchlide (Figure 2b)
w15 ; +/+	.0176	.0009	100	650	684	pchlide (Figure 2a)

*determined using mM extinction coefficient for protochlorophyllide given by S. Gough (Biochem. Biophys. Acta. 286: 36-54, 1972).

*determined by in vivo and in vitro spectroscopy and thin layer chromatography.

a recessive allele at an independent modifier locus. Further genetic tests are necessary to determine the nature of the modifier but it seems reasonable to believe that basically oro is a non-leaky mutant. Both 1*-Neuffer2 and 1*-1039 accumulate protochlorophyllide 650 which is photoconvertible to chlorophyllide 684 on exposure to light: the former accumulates an approximately normal amount while the latter accumulates about 40% of the normal level of protochlorophyllide. It is interesting to note that the heterozygote 1*-1040/1*-1039 has a leaky phenotype producing the same amount of protochlorophyllide as the homozygous leaky mutant. These mutants probably represent one of the following: 1) leaky enzymes of chlorophyll synthesis, 2) mutants in structural elements to which precursors and/or enzymes must bind or 3) mutants whose expression is partially suppressed by modifier genes. In any case a small amount of protochlorophyllide can be synthesized when the demand is low, as in darkness, but under normal lighting conditions they cannot meet the demand for chlorophyll.

The third class of mutants produces reduced protochlorophyllide which absorbs maximally at 636 nm and is thus not photoconvertible to chlorophyllide 684. There are many mutants with this phenotype, including 110 and w15, which may result from defects in any of the undefined processes necessary for normal chloroplast development.

Although protochlorophyllide synthesis is generally tightly regulated the regulatory step can be bypassed by feeding detached shoots δ-aminolevulinic acid (δ-ALA) (.01M in distilled H_2O, 24 hrs). After this treatment, normal plants accumulate a large amount of protochlorophyllide 636 as well as a small amount of protoporphyrin IX (Figure 2, A). Examination of mutants after feeding δ-ALA by in vivo spectroscopy and thin layer chromatography makes possible identification of precursors built up between δ-ALA and protochlorophyllide. The results are

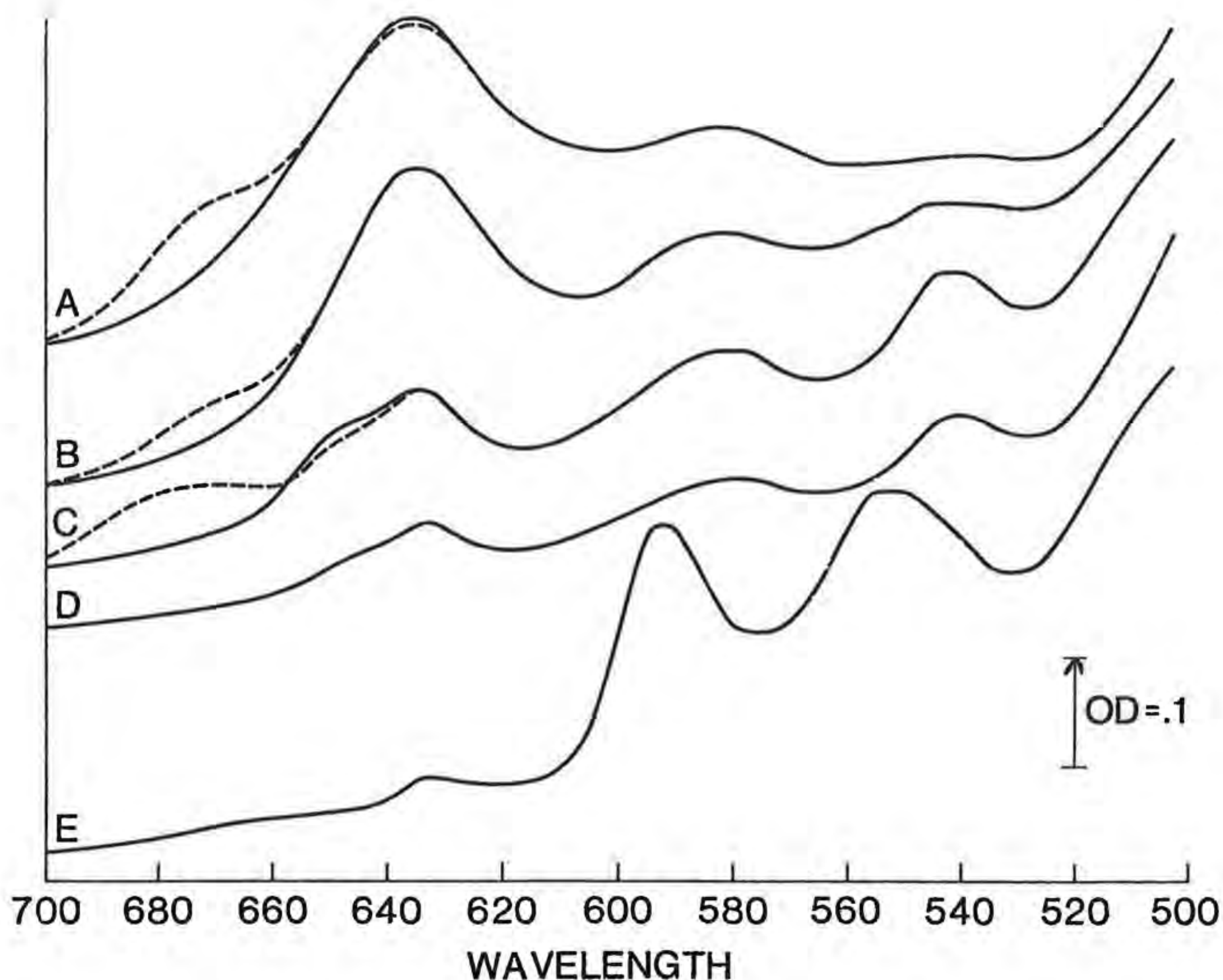

Figure 2. *In vivo* spectra of dark grown leaves fed δ-aminolevulinic acid showing accumulations of chlorophyll precursors. Spectra are characteristic of:

A) wild type and many pale yellow and albino seedling mutants; accumulate mainly protochlorophyllide.

B) *l10* or *w15*; accumulate mostly protochlorophyllide with an increase in protoporphyrin IX.

C) *l*-Neuffer2* or *l*-1039*; accumulate mostly protoporphyrin IX and a small amount of protochlorophyllide.

D) *l*-1040* and *l*-Blandy4* and alleles; accumulate mostly protoporphyrin IX.

E) *oro* accumulates mainly Mg-protoporphyrin and a small amount of protoporphyrin IX.

Dashed lines represent conversion of protochlorophyllide to chlorophyllide on exposure to light (25 ft-c)

Approximate absorption maxima (nm)

protochlorophyllide	636, 578, 540
protoporphyrin IX	636, 578, 540
Mg-protoporphyrin	590, 550

summarized in Figure 2 and Table 2. There are many pale yellow and albino mutants that accumulate a normal level of protochlorophyllide when fed δ-ALA (Figure 2, A). Several mutants, including l10 and w15, accumulate a small amount of protoporphyrin IX as well as protochlorophyllide (Figure 2, B). These lesions are not believed to be directly involved in chlorophyll synthesis but result in the inability of chloroplasts to stabilize chlorophyll. The leaky mutants l*-Neuffer2 and l*-1039 accumulate mostly protoporphyrin IX, but some protochlorophyllide is accumulated (Figure 2, C). Completely blocked mutants l*-Blandy4 and alleles, and l*-1040, accumulate predominantly protoporphyrin IX when fed δ-ALA (Figure 2, D). No protochlorophyllide has been detected in these seedlings even after feeding. Figure 2, E, which represents oro fed δ-ALA for 7 hours, demonstrates accumulation of predominantly Mg-protoporphyrin. After 24 hours a large amount of protoporphyrin IX was also accumulated. Leaky oro (oro-mod) seedlings accumulate Mg-protoporphyrin as well as protoporphyrin IX and protochlorophyllide.

In summary: Several mutants specifically involved in chlorophyll synthesis either directly as chlorophyll synthetic enzymes or indirectly as structural chloroplast components have been analyzed. Three loci are defective in conversion of protoporphyrin IX to Mg-protoporphyrin: 1) l*-Blandy4 and alleles; 2) l*-1040 and allele l*-1039; and 3) l*-Neuffer2. The former two can be completely blocked but no non-leaky allele of l*-Neuffer2 has been found. One mutant, oro, is blocked in the conversion of Mg protoporphyrin to protochlorophyllide. Several other mutants, including l10 and w15, are probably defective in chloroplast synthesis. No blocks prior to protoporphyrin IX such as xan-u in barley have been found. However, the number of mutants examined in corn is relatively small compared to the barley study. The discovery of a putative modifier gene in oro which allows synthesis of protochlorophyllide is novel and will be examined further. It is possible that other leaky mutants are the result of similar modification.

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The use of Tr5 from Tripsacum as a maintainer for polygenic sweet corn

In previous MNL items (1974, 1975) we have described the use of alien addition monosomes and disomes for Tr7 (*Tripsacum* chromosome 7) on a sugary-shrunken-2 background in order to allow seed production of the double recessive (su sh2) by the temporary masking of su with its Su allele on Tr7. The low transmission rate of the alien monosome for Tr7 results in the expression of the desirable double recessive in most of the progeny crop ears. Ordinarily, seed production of su sh2 is nearly impossible because of its low germination rate (about 10%).

A new example of this genetic technique involves seed production of the triple recessive su sh wx. Tr5 (*Tripsacum* chromosome 5) is the alien maintainer. As a homeolog to maize chromosome 9, it not only carries the dominant Sh Wx alleles, but also the dominant C allele controlling aleurone color. All three of these dominants are important to the Tr5 system. Although the alien addition disome for Tr5 is largely male sterile, the monosomic condition is fully fertile. The monosomic inbred su, c sh wx, Tr5(1), with the undesirable low germination masked by Tr5, may be crossed with a standard sweet corn of the genotype su, c Sh Wx. The purple kernels (representing the transmission of Tr5 with its dominant C allele) scattered on each ear in the progeny of the cross may be removed by a

Sortex* or other electric sorting machine. Thus, the triple recessive su sh wx is completely unmasked and segregates on the crop ear in the expected ratio when the remaining (non-purple) seed is planted.

Walton C. Galinat

The floury-1 and floury-2 genes for improving sweet maize

In expanding the volume of endosperm slightly during the milk stage, both floury genes stretch the pericarp cells and produce the effect of increased tenderness. In increasing water retention in the endosperm, the floury genes extend the period of harvestability and the holding capacity after harvesting; the mature sugary-floury ears are slow to dry.

The floury-1 gene on the short arm of chromosome 2 is incompletely dominant. It is the common floury gene in the varieties native to the Southwest and to most arid areas of Latin America; it occurs in some "Evergreen" sweet maize varieties, and it has been used in at least one parent of some commercial hybrids (Tendermost, Tendertreat and apparently certain other hybrids).

The floury-2 gene on the short arm of chromosome 4 has dominance relative to the background; in some crosses it is recessive. Its distribution in native races of corn is unknown. In combination with sugary, it produces a paler yellow, much sweeter (honey-sweet) and more deeply wrinkled kernel than either floury-1 sugary or sugary alone.

Walton C. Galinat

Pericarp thickness and the origin of maize

The wild relatives of maize, namely teosinte and *Tripsacum*, have a smaller, thinner pericarp (two cells at 35μ thickness) than that known in maize (4 to 18 cells totaling 70 to 250μ). In the wild relatives, the strength of the pericarp is supplemented by physical reinforcement from the enclosing cupulate fruit case; the kernels of these relatives will pop on heating, when within the fruit case, but not when they are naked by removal from their enclosure.

With the emergence of the kernel from the fruit case during the evolution of maize, apparently there was selection for a thicker pericarp that provided protection against disease, allowed popping and gave the evolving maize kernel the potential to expand into gigantic sizes.

When man carried primitive maize to South America, where it gained freedom from backcrossing to the thin pericarp of teosinte, it followed a pathway toward greater productivity by an increase in kernel size. The ultimate product, the giant-kerneled Cuzco flour maize, has a pericarp of about 18 cells at 200μ.

In Mexico, introgression by certain genetic components from teosinte into maize is now limited in part by teosinte's thin pericarp and the upward inclination of its outer glume, positioned to puncture the pericarp should an expanding maize-type kernel press against it. As a result the pathway to high productivity per ear in Mexico was usually one of high condensation and increases in kernel row number. Apparently the thick pericarp of the best popcorns became protected from teosinte introgression by certain gametophyte (Ga) genes.

The thin-pericarp barrier is especially important with sweet corn, which has kernels that balloon out in the milk stage at a time when they are often handled roughly during harvesting. Both machine picking and cutting from the cob require some kernel strength. The extra-thin pericarps tend to split in the milk stage, resulting in either the direct destruction of the kernels or their eventual loss through pathological infections.

Ordinarily maize and its relatives, as well as most other grasses (except barley), have only a single-celled aleurone. But the variation in maize from some South American races (Coroico) includes a multi-layered aleurone. Experiments are

now underway to determine if several aleurone layers can help contain the turgor pressure of the developing endosperm and thereby permit a maize pericarp only two cells thick. The substitution of a thick aleurone for a thick pericarp would be nutritively ideal; the pericarp is largely indigestible cellulose, while the aleurone layer is highly digestible and rich in proteins.

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Frequency of two tubes in "in vitro" germinated pollen grains

Pollen grains from two inbred lines, W22 and 824, and from a derivative of "Stock 6" (the high haploid line developed by E. H. Coe, Jr.) were cultured on a medium developed by Pfahler (Canad. J. Bot. 45:839, 1967) in petri dishes at 27 C. Three hours after inoculation all activity was stopped by flooding the surface of the medium with Farmer's solution; the pollen grains could then be observed under a dissecting microscope. The following data include the frequency of grains which germinated and the frequency of grains which developed more than one pollen tube:

	Total pollen grains (T)	Single tube		Two tubes		
		Germ. (G)	%	Germ.	% of T	% of G
Inbred line 824	11,831	5,859	50.0	22	0.18	0.38
Inbred line W22	7,156	5,077	71.0	64	0.89	1.26
Stock 6 derivative	9,550	6,593	69.0	277	2.90	4.20

These data show significant differences in the germination ability of pollen grains of the two inbred lines examined (824 and W22), which may reflect differences at the genotypic level. The derivative of "Stock 6" showed a percentage of pollen grain germination close to that of W22.

The differences in frequency of pollen grains showing two tubes may be of some significance. The highest value was found with the derivative of "Stock 6," the high haploid line. The heterofertilization frequency was also found to be high with Stock 6 (Sarkar, K. R., Diss. Abst. 27:1736, 1966; Coe, E. H. and K. R. Sarkar, J. Hered. 62:118, 1971). The present data, although preliminary, suggest that the abnormal germination of pollen grains with two tubes may have some correlation with the frequent induction of maternal haploids and with the high frequency of heterofertilization, perhaps as a consequence of an abnormal distribution of nuclei in double pollen tubes. Cytological examination of 96 grains with two pollen tubes from Stock 6 derivative material showed 76 cases with a 3:0 nuclei distribution (i.e., three nuclei in one pollen tube and none in the other) and 20 cases with a 2:1 distribution. One could suggest that the tubes having two nuclei might be capable of accomplishing an incomplete fertilization: the polar nuclei might receive a sperm nucleus, while the egg cell received none. The unfertilized egg could then either receive a sperm coming from another pollen tube (heterofertilization) or start development as a haploid.

N. Pogna and A. Marzetti

Numerical taxonomy of Italian populations of maize

A classification of maize populations extracted from the Italian germplasm collection has been undertaken. A good taxonomy of populations showing high adaptability to different environments is of great importance not only for a descriptive and phylogenetic purpose, but also because local populations are a very important reserve of genetical variability, useful in breeding programs.

105 populations were extracted from the germplasm collection (including more than 500 local populations), according to morphological criteria and climatic-geographical considerations.

The populations were grown during the 1975 season in a randomized block design with plots of eight plants. The following traits were considered: dimensional traits of the ear (ear weight, length, base and apex diameter, row number, kernel number per row, 50-kernel weight, thickness, length and width of kernels); morphological-vegetative traits of the plant (plant height and number of leaves--when growth is completed--, number of ears and total yield); physiological-adaptive traits (leaves emission rate--time from 4 to 8 leaves and from 8 to 12 leaves--, tassel flowering time and time from tassel flowering to silking).

All traits show significant differences between populations. The values measured on individual plants were analyzed by multivariate statistical methods (Canonical Analysis). The first three canonical variates (linear transformation of the original ones) explain 71% of the differences between populations. Particularly: the first canonical variate is connected with the vegetative "size" of the plant (growth behavior, cycle length and yield); the second variate is connected with the shape and weight of kernels; the third with the ear size. Mean values of populations were plotted according to the canonical axes (first and second variate) and the points with common 90% significance area in the three dimensions were clustered (see figure).

According to increasing values of the first variate the clusters change from early maize patterns (quarantini, cinquantini, etc.) to late patterns; according to the second variate, from types with small ears and numerous rows (multi-rowed) to types with few rows and large kernels (8-rowed, etc.). Many populations (about 30%) don't cluster: they were regarded as transition forms or as points of adaptation to peculiar environments.

On the basis of physiological, vegetative and adaptive traits it was possible to subdivide groups of populations with very similar morphological features: this shows a true variability that, according to morphological traits alone, can be underestimated.

The next phase will imply a design of crosses between highly differentiated populations to evaluate the genetical distance: it will be possible to obtain a taxonomy of phylogenetical significance.

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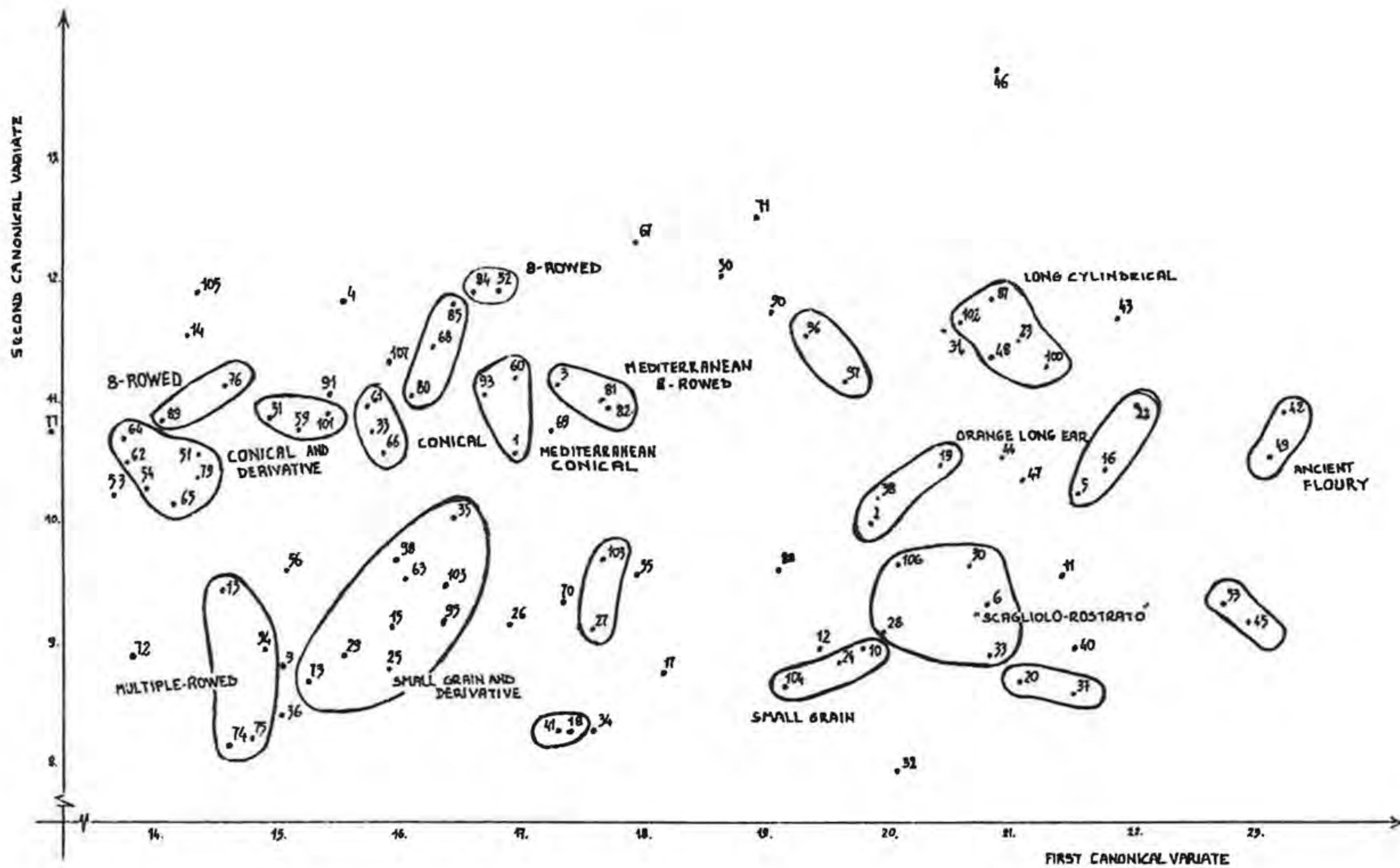
Response of an olive necrotic mutant to gibberellic acid

Conditional lethal mutants are an important genetic tool for investigating the links between the lethal phenotype and the primary event at the gene level. They might be recovered, in the flowering plants, among those mutants, generally defined as seedling lethals, that die at an early developmental stage when the endosperm nutrients have been exhausted. Their detection rests on the possibility


SECOND CANONICAL VARIATE
FIRST CANONICAL VARIATE
8-ROWED
8-ROWED
CONICAL AND DERIVATIVE
CONICAL
MEDITERRANEAN 8-ROWED
MEDITERRANEAN CONICAL
LONG CYLINDRICAL
ORANGE LONG EAR
ANCIENT FLOURY
MULTIPLE-ROWED
SMALL GRAIN AND DERIVATIVE
SMALL GRAIN
"SCAGLIOLO-ROSTRATO"

of inducing phenotypic repair by changing environmental factors like temperature, pH, or nutrients.

The data to be presented refer to the analysis of a series of seedling lethal mutants, isolated in the M2 generation following pollen mutagenesis (Neuffer, 1973). Only seven out of 50 mutants originally isolated were chosen for this test because of their property to express lethality at a very early stage of development. They were grown, as embryo cultures, on both a mineral (D) and an enriched (D+) medium (Gavazzi et al., 1975). Only one mutant, referred to as E283B, showed a slight improvement in its growth if cultured on D^+ rather than D medium. This improvement becomes more evident if the mutant is allowed to grow for 24 rather than 12 days. Mutant E283B is easily distinguishable from normal siblings because of the olive pigmentation, necrosis of the leaf tissues starting before the first leaf emergence, and stunted growth. The latter feature might reflect an alteration in hormone metabolism. Accordingly the effect of IAA, GA_3 and kinetin on mutant growth was tested by adding these hormones separately to a liquid mineral medium and allowing excised mutant shoots, 1 mm long, to grow on these media for 15 days. The results (Table 1) indicate that GA_3 has a slight growth promoting effect on the mutant shoots. The effect of increasing doses of GA_3 on root tips and shoots cultured on liquid media is given in Fig. 1. In both organs GA_3 elicits a positive growth response. The best response is observed at 1 and 10 γ/ml for roots and shoots respectively. At these concentrations the mutant appears to grow as much as the normal sibling. The effect of GA_3 was also tested by incubating root and shoot tips on a mineral medium and then transferring them to a fresh medium supplemented with GA_3 (10 γ/ml). The aim of the incubation is to deplete the organs of their endogenous gibberellins. The results (Table 2) confirm the capacity of GA_3 to induce growth of the mutant organs. The phenotypic repair applies to growth, but not to the "olive" phene. It looks as if a single mutational event has led to simultaneous loss of two metabolic functions, one resulting in growth impairment and the other affecting the photosynthetic pigments. Only the former function is restored by GA_3 administration.

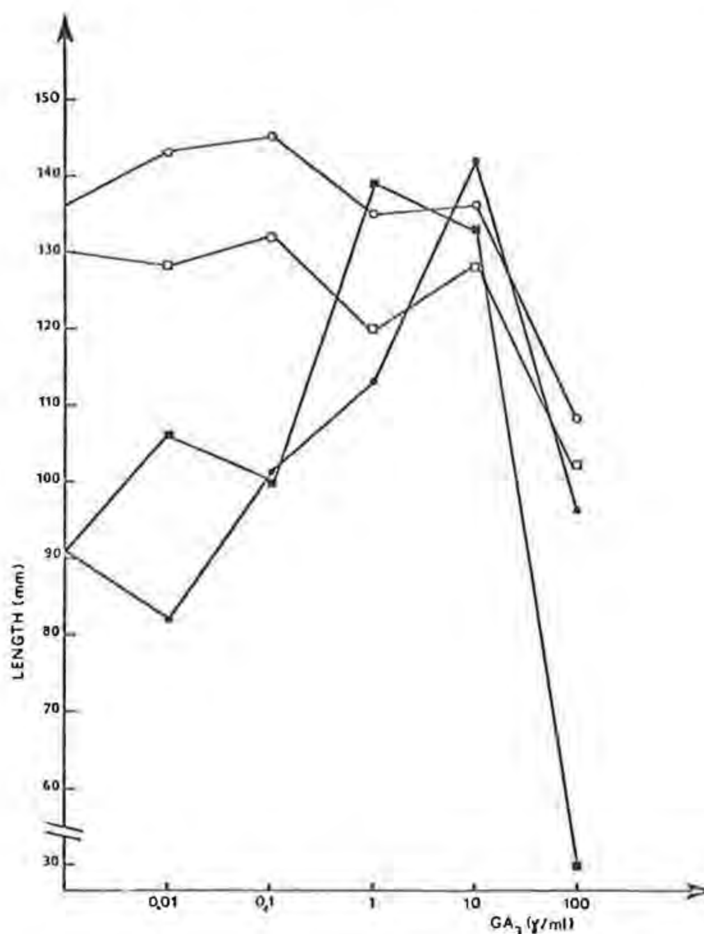


Fig. 1 : Changes in length of excised shoots (• mutant, o normal) and primary roots (■ mutant, □ normal) of mutant and normal seedlings with increasing concentrations of GA_3 in the medium.

To account for this observation one might hypothesize that the mutation E283B affected an early step of a branching

Table 1. Growth of excised shoots from mutant and normal seedlings in liquid media supplemented with hormones (LA = mean length in mm; FW - mean fresh weight in mg).

Growth medium	No. tested +	No. tested m.	LA +	LA m.	FW +	FW m.
F	10	6	130.60 ± 10.20	63.50 ± 7.03	1306.00 ± 15.41	254.33 ± 26.47
F+	11	8	118.38 ± 7.21	54.12 ± 5.37	1189.77 ± 21.36	352.37 ± 24.44
F+IAA(0.05 γ/ml	6	6	142.66 ± 6.89	56.06 ± 5.16	1350.12 ± 14.89	255.02 ± 24.71
F+GA_3(0.01 γ/ml	5	6	125.33 ± 8.76	80.00 ± 4.83	1250.00 ± 251.34	382.00 ± 88.70
F+Kin(0.10 γ/ml	5	5	129.00 ± 1.00	58.00 ± 2.17	1212.33 ± 25.66	318.20 ± 50.50

Table 2. Effect of GA_3 (10 γ/ml) on the growth of organs (shoots and roots) excised from normal and mutant seedlings and preincubated for 48 hr in a mineral medium (LA = mean length in mm.; FW = mean fresh weight in mg).

Growth medium	No. tested +	No. tested m.	LA +	LA m.	FW +	FW m.
			SHOOTS			
F	10	7	137.50 ± 8.71	35.00 ± 2.64	1526.30 ± 109.91	100.57 ± 7.27
F+GA_3	12	10	130.00 ± 5.05	89.30 ± 4.87	631.42 ± 55.01	218.00 ± 16.92
			ROOTS			
H	12	8	65.92 ± 3.18	18.88 ± 2.08	83.66 ± 7.87	16.88 ± 1.84
H+GA_3	12	9	65.25 ± 1.82	42.56 ± 3.08	79.58 ± 3.33	38.22 ± 2.74

metabolic chain lying before the branch point. In this case administration of GA_3, one of the two end products of this pathway, would lead to repair of only one of the two missing functions. A branching point in the biosynthesis of gibberellins is in fact known and is represented by the synthesis of geranylgeranyl pyrophosphate, a common precursor of carotenoids, diterpenes, phytol and gibberellins. Further work with the olive necrotic mutant is aimed at proving whether in fact the above hypothesis can be validated.

Media composition: The H medium for root tip growth is the Heller medium, while that for shoots (F medium) is composed of major salts as in White (1943), minor elements as in Nitsch (1951) and vitamins as in White (1963). The composition of the D mineral and enriched medium for embryos culture is that already employed in previous tests (Gavazzi et al. MNL 47). (Research financially supported by NATO Research Grant No. 950).

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Cytogenetic mapping of genes in the nucleolus organizer - satellite region of chromosome 6

The standard genetic map for maize places ragged (*rgd*) at the zero position of the chromosome 6 linkage map and polymitotic (*po*) at the 4 position. The genes are both shown as proximal to the nucleolus organizer region (NOR). The proximal location is suggested by the fact that Kramer and Duclos (MGCNL 36:66) mapped *po* proximal to *rgd* from three-point F_2 data, and *rgd* was found by Palmer and Dempsey (MGCNL 42:75) not to be uncovered by TB-6a (breakpoint midway in NOR-heterochromatin) or by T2-6(5419), T4-6(4341), T6-9a or T6-9(4778). All of these interchanges have a break in the NOR.

The data reported here place *po* either in the first chromomere of the satellite of chromosome 6 or in the distal half of the site giving rise to the NOR-secondary constriction. The *rgd* locus is placed either as proximal to the NOR or in the proximal 10% of the NOR-heterochromatin. The evidence comes from two approaches. First, 10 of the 13 available interchanges with a break in the satellite have been tested for their usefulness in the duplicate-deficient (Dp-Df) scheme of utilizing nuclear male-sterile genes (MGCNL 49:118). The useful interchanges are those which have a chromosome 6 breakpoint distal to *po*; these interchanges have been shown to be T3-6b, T4-6c, T4-6(5227), T5-6b, T5-6d and T5-6(8219). Limited data also suggest that T2-6(001-15), Tr-6(003-16) and T6-7(7036) are useful. Plants with Dp-Df complements and heterozygous for *po* have not been recovered from T6-10f.

The above results are consistent with the second approach of placing *po* by hemizygous tests. These tests consist of crossing the heterozygous interchange (or in a few cases the heterozygous Dp-Df/+) with a heterozygous +/*po* male parent and scoring male-sterile (-/*po*) plants. Table 1 gives the results of planting the smaller seeds from such crosses and noting the existence of male-sterile plants; experience suggests that the selection of smaller seeds enriches for duplicate-deficient heterozygotes.

Note that none of the satellite-interchanges listed in Table 1 uncovers *po* except T6-10f. This result suggests that all of the satellite-interchanges have a breakpoint distal to the *po* locus except for T6-10f. The cytological placement of the breaks in the three-chromomere satellites of these interchange stocks is reported by Phillips and Wang (this Newsletter). The genetic and cytological data suggest that *po* is not distal to the first satellite chromomere. An apparent inconsistency is the cytological location of the T6-10f breakpoint in the last

Table 1. Tests for -/po hemizygotes from T/+ x +/po or Dp-Df/+ x +/po crosses.

Interchange	Breakpoints 6	Breakpoints Other	Number of small kernels planted	Surviving plants	Male steriles (-/po)
Satellite-interchanges					
2-6(001-15)	sat.[1]	2S.72	10	3	0
6-7(7036)	sat.	7L.63	103	93	1[3]
3-6b	sat.	3S.73	40	16	0
4-6c	sat.	4S.33	47	37	0
6-10f	sat.	10S.28	183	124	23
4-6(5227)	sat.	4S.46	61	27	0
4-6(003-16)	sat.	4L.50	70	29	0
4-6(7328)	sat.	4S.53	58	31	0
6-9(017-14)	sat.	9L.50	20	14	1[3]
5-6d	sat.	5S.64	34	26	0
1-6b	sat.	1L.25	13	9	0
5-6(8219)	sat.	5L.69	28	10	0
NOR-interchanges					
3-6(032-3)	S.C.-midway	3S.34	65	11	4
5-6f	"	5S.23	32	14	2
5-6(8696)	"	5L.79	45	24	3
2-6(5419)	S.C.-.25	2L.82	68	32	4
3-6(030-8)	"	3S.05	78	39	18
6-7(035-3)	"	7L.59	35	29	4
1-6Li	S.C.-prox.	1L.81	12	10	0
1-6(4986)	"	1S.11	75	46	10
1-6(8415)	"	1L.31	50	30	5
2-6(8441)	"	2L.95	21	17	0
2-6(027-4)	"	2L.04	22	14	1
6-10(5519)	"	10L.10	136	102	17
6-9(4778)	Het.95	9L.30	194	141	21
4-6(7037)	Het. 90	4L.61	114	65	11
2-6(8786)	Het. 88	2S.97	79	53	8
6-7(5181)	Het.71	7L.85	83	47	15
6-9a	Het.67	9L.32	157	126	10
4-6(4341)	Het.50	4S.36	67	18	6
6-9d	Het.46	9L.84	27	22	4
6-7(4964)	Het.32	7L.67	42	30	10
6-10(5253)	Het.30	10L.41	162	131	18
1-6(6189)	Het.10	1S.50	7	6	1
1-6d	6S.74[2]	1L.13	62	39	8
1-6(5495)	6S.80	1S.25	53	26	7

[1]sat. = chromosome 6 satellite; S.C.-midway, S.C.-prox., and S.C.-.25 = breakpoint midway, proximal portion or between midway and proximal portions of the NOR-secondary constriction, respectively; Het.95 = breakpoint in NOR-heterochromatin 95% of the distance from proximal to distal ends of the heterochromatic segment.

[2]Breakpoints (determined by Longley) represent the distance of the breakpoint from the centromere to the end of 6S.

[3]Probably male sterile due haploidy.

satellite chromomere while po is uncovered in crosses of T6-10f heterozygotes x +/po heterozygotes. Pachytene analysis of T6-10f was repeated and clearly showed the break position as reported. Presumably, the T6-10f stock possesses a complicated aberration not fully understood.

The interchanges with a break in the NOR nearly all generate Dp-Df complements that allow the expression of po (Table 1). The only exceptions seem to be T1-6Li and T2-6(8441), which may simply reflect insufficient sample size. These data are consistent with the placement of po either in the distal half of the site giving rise to the NOR-secondary constriction or in the first satellite chromomere. The results also demonstrate that deficiencies for various portions of the NOR are ovule-transmissible. The deficiency can include the NOR-secondary constriction and as much as 90% of the NOR-heterochromatin [see data for T1-6(6189)].

In 1972, Perkins, Newmeyer and Turner (Genetics 71:s46) reported that Dp-Df chromosomes produced from Neurospora interchanges with one break near the chromosome tip could break, resulting in a modified chromosome. Pachytene analyses were performed on many of the putative hemizygous -/po plants in this study to determine (1) if a heteromorphic chromosome 6 bivalent was present as expected in a Dp-Df heterozygote, or (2) if a modified deficient chromosome was present as the result of chromosome breakage. The male steriles chosen for analysis included one from the T6-10f cross, the only satellite interchange that uncovered po, and several NOR-interchanges (Table 2). In many cases, the other chromosome involved in the parental interchange was identified at pachytene; a normal bivalent would be expected and was observed.

Table 2. Cytological confirmation of -/po hemizygotes.

Interchange	Chromosome 6 breakpoint[1]	Plants analyzed	Remarks	
			Chromosome 6	Other
6-10f	sat.	1	Heteromorphic	
3-6(032-3)	S.C.-midway	1	"	Normal 3
3-6(030-8)	S.C.-.25	3	"	
2-6(027-4)	S.C.-prox	1	"	Normal 2
6-9(4778)	Het.95	2	"	Normal 9
4-6(7037)	Het.90	1	"	Normal 4
6-10(5253)	Het.30	3	"	Normal 10

[1]See Table 1 footnotes

In all cases, the expected heteromorphic bivalent associated with the nucleolus was present, indicating that chromosome breakage had not occurred and that the male-sterile plants were hemizygotes for the po locus. They possessed heterozygous deficiencies for parts of the satellite or NOR, depending on the interchange.

With the indication that po is distal to the NOR-heterochromatin, the possibility seemed unlikely that rgd could be distal to po by 4 map units. Therefore, similar hemizygous tests were performed to determine whether interchanges used in this study would uncover rgd. Smaller seeds from crosses of interchange heterozygotes with heterozygous +/rgd plants were germinated in laboratory trays; remnant seed was germinated in a greenhouse sandbench. The lack of ragged plants (Table 3) suggests that rgd is not distal to po but is located proximally either in the short arm of chromosome 6 between the centromere and the NOR or in the proximal 10% of the NOR-heterochromatin. The two markers rgd and po may serve as flanking markers for most if not all of the NOR.

Table 3. Tests for -/rgd hemizygotes from T/+ x +/rgd crosses.

Interchange	Chromosome 6 breakpoint[1]	Smaller seeds (Rgd plants/total seeds)	Remnant seeds (Rgd plants/total seeds)
6-7(7036)	sat.	76/76	229/232
4-6(5227)	sat.	148/154	342/365
4-6(003-16)	sat.	39/41	77/80
4-6(7328)	sat.	92/110	177/246
1-6(4986)	S.C.-prox.	48/59	230/243
2-6(027-4)	"	41/45	-
6-10(5519)	"	66/67	133/143
4-6(7037)	Het.90	36/36	59/83
2-6(8786)	Het.88	65/67	159/159
6-7(5181)	Het.71	71/72	144/153
1-6(6189)	Het.10	29/29	100/103
1-6(5495)	6S.80	32/37	200/200

[1]See Table 1 footnotes

R. L. Phillips, E. B. Patterson and P. J. Buescher

Interchange breakpoints in the chromosome 6 satellite

Phillips and Wang (MGCNL 46:123) reported 13 interchanges with a breakpoint in the satellite of chromosome 6. Table 1 lists the position of the break within the satellite for 12 of the 13 interchanges. The satellite region of these stocks

Table 1. Cytological location of interchange breakpoints within the satellite region of chromosome 6.

Interchange	Satellite Breakpoint
2-6(001-15)	Distal end of the distal chromomere
6-7(7036)	"
3-6b	Proximal end of the distal chromomere
4-bc	"
6-10f	"
4-6(5227)	Between middle and distal chromomeres
5-6b	"
4-6(003-16)	" (tentative placement)
4-6(7328)	Between proximal and middle chromomeres
6-9(017-14)	"
5-6d	" (tentative placement)
1-6b	" (tentative placement)

consists of three prominent chromomeres. The cytology of the interchange homozygotes enabled the precise placement of the chromosome 6 breakpoint for nine interchanges. The cytological analysis of the other three was more difficult, allowing only a tentative placement of the satellite-interchange breakpoint.

R. L. Phillips and A. S. Wang

Nucleolar capacity of NOR segments

In 1972, Phillips and Wang (MGCNL 46:123) listed 20 interchanges with a break in the nucleolus organizer region (NOR). The nucleolar association of two bivalents at diakinesis in homozygous interchange stocks was the criterion employed to determine that the break was in the NOR. The three interchanges T4-6(7037), T6-9(4778), and T6-10(5253) were thought to have a break proximal to the NOR in 6S because only one bivalent associated with the nucleolus in the homozygote and the satellite was of normal size. In the process of cytogenetically mapping

polymitotic (po) by hemizygous F1 tests in which the deficiency derived from heterozygous interchanges (see article by Phillips, Patterson, and Buescher, this issue), the above three interchanges all transmitted a deficiency-duplication complement that uncovered po. If the breaks are in 6S proximal to the NOR, the expected deficiency would include the entire NOR. Believing this to be unlikely, pachytene analysis was performed on the three interchange homozygotes. All three were found to have a break in the NOR heterochromatin.

The NOR break in T4-6(7037) was determined to be about 90% of the distance from the proximal to distal end of the heterochromatin (Het.90). Only a small heterochromatic segment (10%) was associated with the nucleolus. The bivalent possessing the large proximal segment (90%) was nearly always not associated with the nucleolus and usually formed no nucleolus. This 6^4 bivalent formed a small nucleolus comprising approximately 0.2% of the total nucleolar volume in less than 1% of the pachytene cells. Thus this NOR segment, consisting of nearly the entire NOR heterochromatin, was largely inactive in nucleolar formation at pachytene in the homozygote.

The T6-10(5253) interchange was found to have identical properties to T4-6(7037) in terms of nucleolar capacity. However, the break was determined to be at .3 in the NOR heterochromatin (Het.3). Thus the NOR heterochromatin can be separated at these two points (.3 and .9) with essentially no nucleolar capacity associated with the different proximal portions. These observations are consistent with the functional map developed for the NOR and presented by Phillips at the International Symposium on Genetics and Breeding, September, 1975.

The break in T6-9(4778) was extremely close to the distal end of the NOR heterochromatin (Het.95). This interchange differed from the other two in that both bivalents with NOR segments associated with a single nucleolus in at least 53% of the cells. The earlier cytology at diakinesis indicating only one bivalent associated with the nucleolus apparently was in error, perhaps representing a sampling problem. In 19% of the 136 pachytene cells analyzed, the 6^9 bivalent possessing nearly the entire NOR heterochromatin had associated a small nucleolus representing 1.7% of the total nucleolar volume. Apparently the NOR breakpoint in this interchange is positioned such that some nucleolar capacity is associated with the proximal heterochromatic segment.

In summary, these homozygous interchanges separate the NOR-heterochromatin into two parts with the proximal segment possessing a very low nucleolar formation capacity. The finding that the breaks are in the NOR explains why these interchanges uncovered po in appropriate crosses. Deficiencies for various parts of the NOR have been shown to be ovule transmissible (see Phillips, Patterson and Buescher, this News Letter).

R. L. Phillips and P. J. Buescher

Cytological analysis of plants regenerated from maize tissue cultures

Plants may be regenerated from maize callus tissue growing on an appropriate medium (Green and Phillips, 1975, Crop Sci. 15:417). Plant tissue cultures often possess aneuploid and polyploid cells. The data reported here give a preliminary indication as to whether or not the chromosome constitution and behavior of plants regenerated from maize tissue cultures, derived according to the methods described in the above reference, are normal. The cultures used in this study were initiated from A188 x W22 A C R-nj b pl embryos 1.5-2.0 mm in length (12 days postpollination) on MS medium containing 1 mg 2,4-dichlorophenoxyacetic acid (2,4-D) per liter. Plants were regenerated during the fourth through sixth subcultures, 65-125 days after culture initiation, by transferring callus to asparagine-minus MS medium which contained 0 or 0.25 mg 2,4-D/l. One hundred eight plants collected during this period were transplanted to vermiculite, watered with a 1/4 strength MS salts

solution and allowed to grow for 10-14 days in an incubator at 70-75% relative humidity, 27-28 C, and a 16 hour photoperiod with a light intensity of 3500 lux. Eighty-seven plants (80%) survived and were transplanted to soil and grown to maturity in the greenhouse. At the appropriate stage, a portion of the immature tassel was collected from 43 plants and fixed in 3 parts 95% ethanol:1 part glacial acetic acid. The portion of the tassel not collected allowed for subsequent pollen sterility determinations on each plant.

Normal chromosome numbers, pairing, and pollen fertility levels were observed for 41 plants. The two cytologically aberrant plants were both mosaics. One had tetraploid tassel sectors while the other had aneuploid sectors which were observed to be monosomic for chromosome 5. Both plants possessed the chromosome pairing and behavior patterns expected with their respective changes in chromosome number. Pollen fertility was determined to be normal for both plants. Since the pollen analysis was based on only one sample per plant and performed prior to the cytological analysis, we assume that the expected pollen sterility was missed due to the sampling procedure. In these materials, therefore, 95% of the regenerated plants appeared to be cytologically normal. The aberrant types were mosaics. At what point in development the mosaics arose is unknown.

In addition to the above materials, eleven plants were regenerated from three-year-old maize callus tissue of the same genotype and by the same procedures as described above. These plants possessed oppositely arranged (decussate) leaves and were about three feet tall at maturity. Three were analyzed at pachynema and all possessed a heteromorphic chromosome 6 bivalent. One chromosome 6 in each plant was deficient for the distal one-third of the long arm. Since all three plants were cytologically identical, the callus tissue from which the plants were derived may have been homogeneous for the deficiency.

C. E. Green, R. L. Phillips and A. S. Wang

Progress report on tests to establish lines homozygous for "inactivated" Ms* alleles

Plants from selfs of plants whose progeny did not segregate male sterile but when crossed on male steriles produced some male sterile offspring should include some that are homozygous for the "inactivated" Ms* locus.

For male sterile-1, there are two lines which give male and female transmission. Plants from selfing the heterozygote were tested on ms ms. For one line, 27 had only fertile progeny, 31 had fertile and male-sterile progeny. For another line, there were 24 and 3, respectively. None of the plants had only male sterile progeny in the test on male sterile-1.

Two lines for "inactivated" Ms2 have been established which show both male and female transmission. For the tests of line 6 on ms2 ms2, there was one plant that produced only fertile progeny and six that had fertiles and male steriles. For line 7, the numbers were 16 and 29, respectively. These numbers suggest a 1:2 ratio, but there was no plant homozygous for the "inactivated" locus. The number of plants tested for line 7 are seemingly adequate for an expectation of one or more homozygotes.

For Ms8, lines were established from two male sterile plants that appeared in F_1 from x-rayed pollen applied on ms8 ms8 plants. The male steriles were crossed with a normal inbred and the progeny were selfed. All the selfs segregated for male-sterility, 20 from one line and 12 from the other.

For ms10, lines were established from four male sterile plants that appeared in F_1 from ms10 ms10 plants crossed with x-rayed pollen. Again, all the selfs segregated male-steriles, the numbers tested being 22, 3, 21 and 23, respectively, for the four lines.

For golden-1(g) there was one line from a golden plant crossed with x-rayed pollen. The 13 selfs of plants from the cross with normal all segregated golden.

For the two ms8, four ms10, and one g lines, the x-rayed chromosome which allowed expression of the recessive probably was not transmitted through the female. A possible alternative explanation is that the homozygote itself expresses the recessive character. Tests are continuing with the lines carrying the "inactivated" Ms and Ms2 loci.

Charles R. Burnham

Crossover frequency in the balanced lethal: white-8896/luteus-10 for chromosome 6

Self seed from two stocks, one that was Y+/y w15-8896 and the other Y+/y l10, was obtained from Dr. Robertson. Both recessives are closely linked with the y locus. Crosses between the two stocks were made, using green plants from the yellow seeds. The white seeds from crosses that segregated yellow and white seeds were heterozygous for albino and luteus in a balanced makeup which produced green plants. These were crossed with a yellow endosperm stock, and the progeny were selfed. All segregated yellow vs white seeds: 155 segregated for albino, 158 segregated for luteus seedlings. In addition, there were two classes that came from crossing over between the two loci. There were 9 in one crossover class that lost both recessives and therefore had only green seedlings. The complementary class has both recessives. Plants homozygous for both recessives have the albino phenotype. Plants heterozygous for this crossover are recognizable only when a new crossover separates the two factors. There were 3 plants of this type. The seedling counts on these are:

	yellow seeds			white seeds		
	green	white	luteus	green	white	luteus
39530-71	92	4			35	1
39533-187	102	1		4	31	1
39535-264	109	6		3	33	1

Larger samples from the ears segregating white seedlings need to be grown to identify all plants of this crossover type. Using the 9 plus 3 identified crossovers, the recombination value is 3.7%.

Lindstrom (Genetics 10:442, 1925) reported that a plant homozygous for luteus-1 is green, but if homozygous also for w, w2 or w3 is yellow. The homozygote for luteus-2 is yellow, but the homozygote for both luteus-2 and w is white. I tested many combinations of different yellow and white seedlings in barley. In all cases, the combination of white with yellow was a white seedling.

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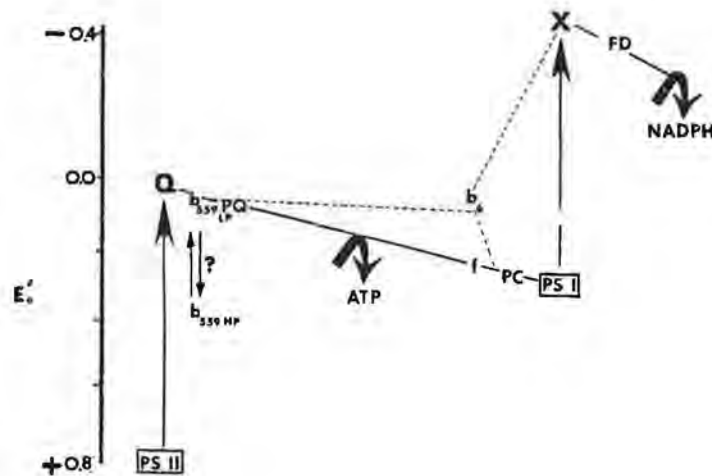
High fluorescent mutants in maize: biochemistry and physiology

We are currently using high fluorescent mutants in maize to study the mechanism of light reaction photosynthesis. Although photosynthetic mutants have been used to advantage in algae (Levine 1969, Ann. Rev. Plant Physiol. 20:523), very few large scale systematic studies of photosynthesis have been carried out using the mutant approach in higher plants. To date we have obtained about 70 isolates blocked in photosynthesis by selecting high fluorescent seedlings from M_2 material as described previously (MGNL 46:127).

The high level of chlorophyll fluorescence (hcf) seen in mutant seedlings is a result of the inability of these plants to carry out normal rates of electron

transport. The light-induced flow of electrons in chloroplasts is mediated by a chain of electron carriers whose arrangement with respect to a chemical potential gradient can be represented schematically by the familiar Z-scheme originally formulated by Hill and Bendall (1960):



Normally, light trapped by photosystem II (PS II) and photosystem I (PS I) reaction centers induces electron flow and the resulting chemical energy is efficiently used in the production of ATP and NADPH. Blockages in electron flow due to missing electron carriers or inefficient production of ATP and/or NADPH result in an inability to dissipate trapped light energy in useful ways and elevated levels of chlorophyll fluorescence result. This phenomenon is independent of the level of pigmentation.

During the past few years we have been able to identify by various means some of the electron carriers and biosynthetic processes affected by mutation in our stocks, as follows:

- hcf-1: (C. D. Miles and D. J. Daniel 1974, Plant Physiol. 53:589) reduction in NADPH diaphorase activity; slightly yellow-green (YG) semi-lethal (survives under good field conditions with poor or little seed set).
- hcf-2: (Miles and Daniel 1974) loss of cytochrome f and most of the plastoquinone pool (PQ); YG; seedling lethal.
- hcf-3: (Miles and Daniel 1974) loss of Q (physiological quencher of chlorophyll fluorescence, presumably a protein), loss of cyt b_{559} high potential (b_{559HP}); fully green; seedling lethal.
- hcf-4: reduced capacity for ATP synthesis, over half PQ missing; slightly YG; semi-lethal.
- hcf-6: loss of cyt. f; slightly YG; seedling lethal.
- hcf-9: loss of Q and both b_{559HP} and b_{559LP}; fully green; seedling lethal.
- hcf-19: two forms recently recognized, both seedling and lethal:
  - hcf-19 green: Q and b_{559HP} missing; no capacity for O_2 evolution.
  - hcf-19 YG: Q partially present or functional; b_{559HP} missing; retains capacity for some O_2 evolution.

hcf-38: loss of cyt. f; YG; seedling lethal.

hcf-47: missing cyt. b_6; YG; seedling lethal.

hcf-48: greatly reduced capacity for ATP synthesis; YG; seedling lethal.

In all cases except hcf-19, segregation is as a single gene nuclear mutation giving 3:1 ratios in selfs and F_2 populations (hcf-47 not yet carried to F_2). The green:yellow-green heterogeneity present in family hcf-19 has only recently been detected and is not yet genetically investigated. Selfed material segregates approximately 1:1:2 normal but independence has not been established.

It is apparent that several of the mutations are pleiotropic, notably hcf-3, 9 and 19. This observation has its counterpart in the mutational analysis of algal photosynthesis, where the loss of a 47K polypeptide has been correlated with the simultaneous loss of both Q and cyt. b_{559} in Chlamydomonas reinhardtii (Chua and Bennoun 1975, PNAS 72:2175). From this work and that of other investigators in the field, it appears probable that at least some of the proteins present in the thylakoid membrane are necessary for binding electron carriers.

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Kenneth Leto and Donald Miles

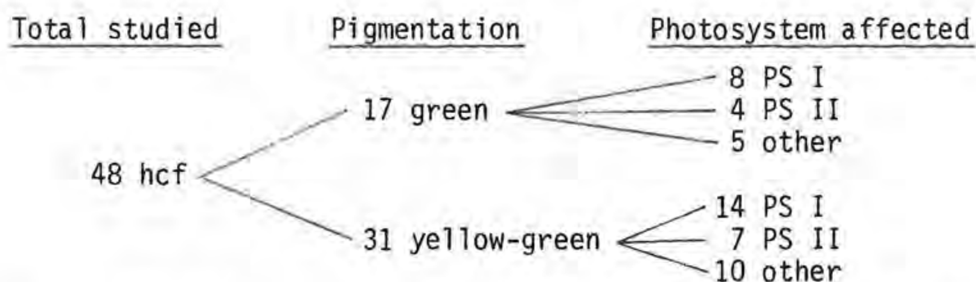
High fluorescent mutants in maize: selection and genetic investigations

The high chlorophyll fluorescence phenotype in maize has been used to select plants blocked in light reaction photosynthesis. High fluorescent plants were selected from M_2 material ultimately derived from chemically treated pollen and normal silks as described in MGNL 45:146. Our material was recovered by screening sandbench material at night using a hand-held UV lamp (UVL 56, Ultra-Violet Products, Inc.) to excite fluorescence and a pair of welder's goggles fitted with UV-protective filters to view the characteristic red chlorophyll fluorescence. Individuals marked at night were taken from the bench the next day and the hcf phenotype confirmed by measuring whole-leaf fluorescence induction kinetics in a fluorimeter. Seed was obtained from families segregating confirmed hcf individuals and the phenotype examined under growth chamber conditions. The material flowed through our hands in the following manner:

	No. families	% Total
M_2 families screened	3191	100
Number of families segregating hcf in the greenhouse	155	4.0
hcf kinetics confirmed in lab-sandbench material	105	2.7
hcf kinetics confirmed in growth chamber material	57	1.5

In preliminary tests, all segregated 3:1 in selfs.

Of these 57 families, 48 have been examined in some detail. On the basis of standard biochemical and biophysical tests the lesions occurring in these mutants can be classified as affecting either photosystem I or photosystem II preferentially. In some cases the site affected by the lesion is not at all clear. The data presently available allow us to draw the following profile:



It is clear that the hcf phenotype is independent of the amount of chlorophyll present in mutant leaves. During the initial selection phase many families segregated YG plants which did not appear to fluoresce highly.

As seed became available an attempt was made to locate the hcf markers to chromosome arm following the procedures outlined in MGNL 45:144; 46:130; and 47:148 using an improved set of B-A translocations. We have been able to locate tentatively nine hcf mutations to chromosome arm as listed below (terminology after Beckett, MGNL 50:89):

hcf	B-A tester	Arms partially uncovered	Probable hcf location	Pigmentation
1	1Sb-2L-4464	2L, 1S	2L	slightly YG
2	1La-5S-8041	5S, 1L	5S	YG
3	1Sb-2L-4464	2L, 1S	2L	Green
13	1Lb	1L	1L	slightly YG
15	1Sb-2L-4464	2L, 1S	2L	yellow or YG
19-YG	3Sb	3S	3S	slightly YG
23	4Sa	4S	4S	YG
26	6Sa	6S	6S	YG
34	6Lc	6L	6L	YG

In cases where the compound translocations of F. A. Rakha and D. S. Robertson (Genetics 65:223, 1970) or Robertson (MGNL 49:79) were used, the B-A translocations used to develop the compound tester were also used as male in the mapping test and failed to uncover fluorescing individuals. We are reasonably certain, therefore, that the markers lie on the designated arms although further confirmation is needed.

We now know the electron carriers or biosynthetic capacities affected by mutation in some of our lines (previous article). Coupling this information with the tentative map locations, it was possible to select from the larger collection individuals that might be related genetically. The following allelism tests were performed:

Same arm:		Missing cyt. b559:	
15 x 1	not allelic	9 x 19	not allelic
15 x 3	not allelic	9 x 3	allelic
1 x 3	test failed	19 x 3	test failed

The other obvious test, that involving hcf-2, 6, and 38 (all missing cyt. f) was carried out but gave ambiguous results in the first test.

The interesting finding is the allelism between hcf-3 and 9. We originally assumed that hcf-3 and hcf-9 might not be allelic since, while both are missing Q and cyt. b559HP, hcf-9 alone is missing both forms (HP and LP) of the cytochrome. The finding that hcf-3 and hcf-9 are allelic raises some interesting possibilities; for instance, we may hypothesize that a single protein necessary for photosystem II

function has been altered at two different sites by mutation such that 1) in both cases (hcf-3 and 9) the protein is non-functional as an electron carrier and hence seedlings are hcf; 2) it is unable to bind or allow the proper integration of either form of the cytochrome into the thylakoid membrane in the case of hcf-9; and 3) it can bind or create a favorable membrane environment for the proper integration of $b_{559}LP$ but not $b_{559}HP$ in the case of hcf-3. This is of course only one possible hypothesis, but it is consistent with experimental observations made on these two mutants.

Although the allelism tests for hcf-19 x hcf-3 failed due to poor field conditions it is improbable that at least the yellow-green form of hcf-19 is allelic to hcf-3 since hcf-19YG appears to map to the short arm of chromosome 3 while hcf-3 tentatively maps to the long arm of chromosome 2. Excluding confounding factors (suppressors, background effects) the following conclusions can be drawn at this time:

1) hcf expression is independent of the amount of chlorophyll present (although extremely YG or yellow plants are obviously deficient in the fluorescing species);
2) hcf-3, hcf-9 and hcf-19 are pleiotropic mutants affecting photosystem II function; hcf-3 and hcf-9 are allelic although their biochemical properties differ slightly; both are probably not allelic to hcf-19 YG and possibly not allelic to hcf 19 green;
3) A gene coding for NADPH diaphorase is located on the long arm of chromosome 2;
4) Subject to confirmation an hcf marker, (which is also conveniently yellow-green) is located on the short arm of chromosome 6 beyond the NOR.

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Dominant mutants from EMS-treated pollen

Selection in the M_1 from a cross of normal ears by pollen treated with EMS (ethyl methanesulfonate) has yielded a total of 45 dominant mutants. The frequency of these mutants is comparable to the 2 x 10^{-3} reported elsewhere (Neuffer, M. G., International Maize Symposium: Genetics and Breeding, D. B. Walden (ed.), 1977); and although all the M_1 ears in these experiments have not been tested, the frequency of recessive mutants seems to be within range of the 412 x 10^{-3} also reported.

The collection includes a wide array of phenotypes, some expressed in the kernel and others in the seedling or in the mature plant. The most frequent type is the disease lesion mimic (Neuffer, M. G., and O. H. Calvert, J. Heredity 66:265, 1975), of which there are eleven separate cases. Some of these may be duplicates, but preliminary data indicate at least four separate loci. Other interesting types include dominant dwarf (6) and yellow-green (3) plants, striped virescent seedlings (3), mosaic aleurone (2) and etched endosperm (2) mutants, a yellow-streaked plant, a thin-tassel mutant, a rolled leaf mutant (like Ce, reported by Mouli, MNL 50:5) and a defective morphology mutant with split leaves and a distorted growth pattern.

Eleven of these mutants have been located to chromosome, most of them by the waxy translocation method:

mutant	description	chromosome
Yg-700	Yellow-green plant	10 (allele of oy)
Yg-1459	Yellow-green plant	10 (allele of oy)
Les	Lesion-1	2 (near bd)
Ysk-844	Yellow-streaked plant	4
D-985	Dwarf	8
Spc-1376	Speckled plant	3
Tht-1440	Thin tassel	8
Atc-1443	Strong anthocyanin	9 (very near wx)
Rgd-1445	Ragged leaves	5
Gcb-1456	Grainy crossbanding	7
Mos-791	Mosaic kernel	10 (linked to R)

Three-point linkage data that place Lesion-1 (on chromosome 2) between gl and fl are presented in Table 1.

Table 1. Placement of Les on chromosome 2.

$$\frac{+\ +\ Les\ +}{lg\ gl\ +\ fl} \times lg\ gl\ +\ fl$$

Phenotype	Number
+ + Les + (parental)	231
lg gl + fl	193
+ gl + fl	43
lg + Les +	54
+ + + fl	92
lg gl Les +	105
+ + Les fl	37
lg gl + +	30
+ gl Les +	6
lg + + fl	7
+ gl + +	3
lg + Les fl	5
+ + + +	16
lg gl Les fl	15
+ gl Les fl	1
lg + + +	0
Total	838

lg—gl 119/838 = 0.1420

gl—Les 242/838 = 0.2887

Les—fl 107/838 = 0.1276

lg 14 gl 29 Les 13 fl

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Efficient testing for allelism among lethals

When a series of lethals is to be tested inter se for allelism, the ideal test is one in which two lethals (A and B) are both known to be heterozygous in the parents that are crossed. If two or more ears per plant can be pollinated, there is no difficulty in making a test self on A and B plus a cross of A with B, repeating enough times to assure that the ideal test has been made [for a 3:1 trait, each paired test has 4/9 chance of success and 5/9 of failure to be an ideal test; thus since $(5/9)^6 = .03$, 6 pairs consisting of 12 plants with 18 appropriate pollinations will have 97% certainty of including at least one ideal test, resolving the question of allelism].

Most often two ears per plant cannot be assumed and an optimal alternative is needed. In recent research here in which large numbers of such tests have been required, the procedure has been optimized as follows: Self a plant in source A and cross onto two plants in source B; test each of the two plants in B by crossing each onto one ear of a sib (set consists of 5 pollinations; failure of heterozygosity in source A is 1/3 and failure of showing heterozygosity in one or both plants of source B is 25/81; three such sets, totalling 15 plants, will have about 75% certainty of including an ideal test; 6 sets will have about 93% certainty).

E. H. Coe, Jr.

Expressions of alleles at the B locus

From a variety of sources, including genetic strains, inbred lines and varieties, 31 sources of plant color independent of R (i.e., color expressed with r-g) have been converged four times to a standard strain of K55 background with A C r-g b Pl. All sources have single-factor inheritance of the plant color expression; with Pl, all have glume bar color; all are allelic to B.

Mature plants carrying each allele heterozygous with b were graded (3 to 9 plants each) in randomly planted families. Intensity grades on a scale from 0 to 7 were tallied in three sheaths (basal; lower than ear; upper), leaf blades, auricles, nodal rings, tassel glumes, glume bars, brace roots, culm and husks. The average of these 11 grades for each tissue is arrayed in rank order in the left portion of Fig. 1. The strongest allele I know, B-I (replicated four times in the figure), is the standard allele used in studies of paramutation at the B locus; B'-I is the paramutant form of B-I (among the other alleles only B'-K and B'-V, from genetic strains, are paramutagenic); B-Bolivia and B-Peru are the alleles that confer aleurone color replacing R (the first irregular and faint, the second uniform and intense); B-V and B'-V are mutable; B-N6, B-W22 and B-38-11 were derived from the respective inbred lines; B-Bolita and so forth were derived from varieties, and the rest from various genetic stocks. The alleles seem to fall generally into about four discrete levels of expression, although they may actually represent a continuous series.

If the intensity of expression is considered for each tissue in the rank order established by the overall average grade, tissue-specific effects that are characteristic of each allele may be identified by variations in a profile line (right portion of Fig. 1). Generally, the grades for these three tissue examples follow the rank order, but some alleles evidently are not coordinate from tissue to tissue; discontinuities for auricle color are especially prominent.

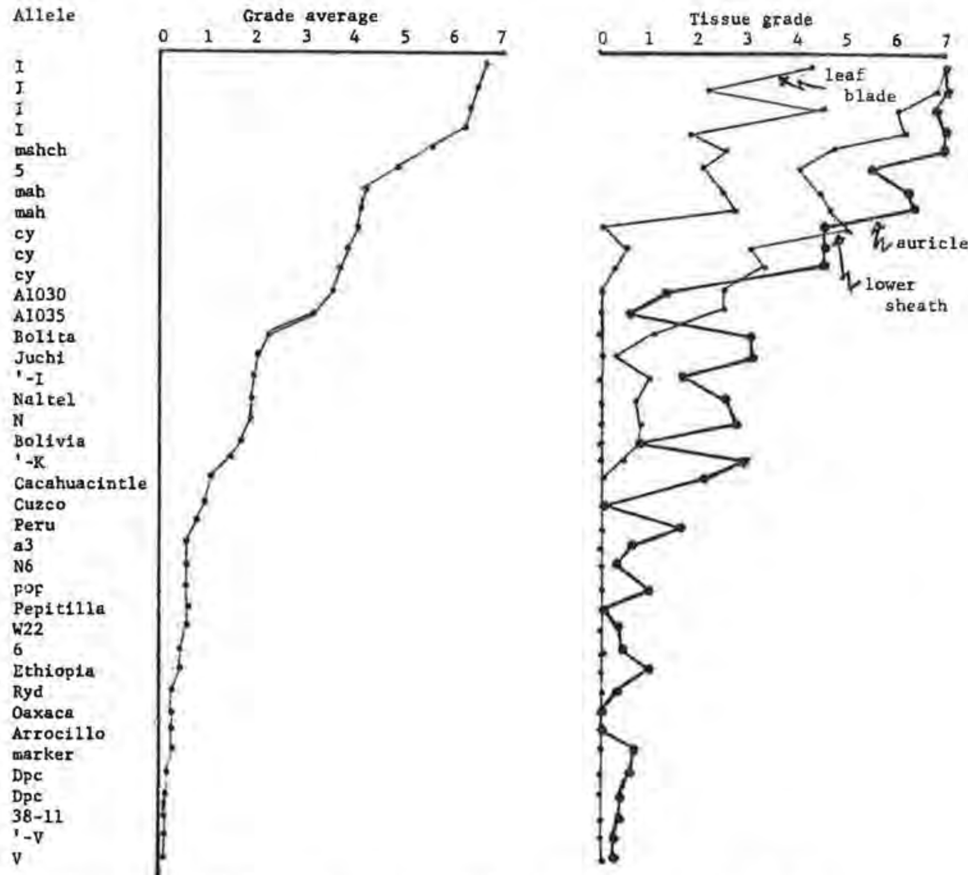

Fig. 1. B alleles arrayed according to rank order (left); grade profiles for the same alleles expressed in three specific tissues (right).

Assuming that these alleles are a reasonably representative sample of the possible range of expression at this locus, the data suggest that quantitative variation is parallel only roughly from tissue to tissue, and that the determinants for variation may vary non-coordinately with determinants for tissue specificity.

E. H. Coe, Jr.

Cells in the embryo and their destinies in tassel, ear and vegetative parts

Dry seeds heterozygous for four plant-color markers (A a B b Pl pl R-r r-g) were x-rayed (8000 r), grown, and scored for sectors in the plants at flowering and at maturity. For numerical data, we determined numbers of tassel branches affected, fractions of sheath and culm perimeters, and lengthwise extent in nodes. From these fractions and percentages, apparent cell numbers (ACN) have been calculated.

Among over 500 plants examined, 45 had tassel sectors, of which 10 had sectors in the body of the plant as well, 6 with coincidental (i.e., separate) sectors and 4 with possible continuity of the sector from the remainder of the plant into the tassel. Sectored main spikes were usually divided evenly in half, so main spikes were scored as equivalent to two branches; most single branches were undivided. The average affected portion of the tassel was 34.8% (modal class 40-50%); ACN for the tassel in this material is 2.88 cells (1/0.348). Most tassels (56%) were represented by about 2 cells, the rest by 3-4 cells (24%) or more.

In the body of the plant, sectors could be scored in the sheath and culm beginning with the 7th leaf; 94 sectors were found, on which perimeter measurements gave the following ACN values for each node:

Node	7	8	9	10	11	12	13	14	15	16	17	18	19
ACN	19	40	36	32	32	30	31	33	22	17	16	15	21

The average extent of sectors (number of contiguous nodes affected) ranged from 2 to 8.5:

Top node	7	8	9	10	11	12	13	14	15	16	17	18	19
Av. extent	2.0	2.0	2.0	3.0	2.3	2.0	2.3	2.8	3.4	5.0	5.5	8.5	7.0

From these numbers it is evident that the ACN in the embryo drops from about 32 cells spanning 2 to 3 nodes to about 16 cells spanning 4 to 8 nodes, at about the 15th node. Ears were distributed mostly at nodes 12 to 14--i.e., just below the change point.

At nodes bearing palpable ears 21% of sectors crossing that node entered the husks; all 23 husk sectors were continuous with sectors covering one or more nodes of the culm and sheaths. ACN for the husks is 4.52 cells, a subset of the 32 cells spanning the node at this level. Only one cob sector was found in the test population; this sector was continuous with a body sector covering 4 nodes, divided the shank and husks equally, and included the vast majority (97.5%) of the cob (all but 16 of about 640 floral units). ACN for the cob must be quite small (not much more than 2 cells) and is probably a subset of the husk cells.

From these data, the meristem in this material, for the plant body from about leaf 7 upward, can be represented diagrammatically as a stack, roughly as follows:

Destiny	Cells
Tassel	2-4
Top 4-5 nodes	16
2-3 nodes (ears)	32 (4-8)
2-3 nodes	32
2 nodes	32
2 nodes	32

These cell groups are determined as compartments in the embryo, though presumably with some degree of plasticity. Those cells destined to elaborate the ear clearly do not extend into tassels and vice versa; thus, non-concordance of mutational events (see Neuffer, MNL 49:126, 1975) is the situation for seed treatment of maize with mutagenic agents, and recessive mutations would not be expressed in the selfed progeny ("M_2") from treated seed.

E. H. Coe, Jr. and M. G. Neuffer

Location of genes conditioning stalk quality with translocations

Various techniques have been employed to identify and select genotypes with superior stalk quality. Zuber and Grogan (Crop Sci. 1:378) measured stalk strength mechanically, where they determined the pressure (in load lbs.) to crush a mature stalk section. They also determined weight of a stalk section and rind thickness. Studies of these traits by Loesch, Zuber, and Grogan (Crop Sci. 3:173) indicated they were quantitatively inherited. Recurrent selection for stalk crushing strength through five cycles has increased it by 57 load-kg per cycle.

We used reciprocal translocations for locating genes affecting crushing strength, stalk section weight, and rind thickness. Twenty translocation stocks in M14 background were obtained from W. A. Russell. M14 has low crushing strength, low stalk section weight and thin rind. Each translocation stock was crossed to

B37 and B14A; both of these lines have excellent stalk quality. Each semi-sterile F_1 was test-crossed to Oh43 and N31, each with low crushing strength, low stalk section weight, and thin rind. Mean values for the five inbred lines are given in Table 1.

Table 1. Mean values for five lines *per se*.

Inbred	Section Weight	Crushing Strength	Rind Thickness
	(gm)	(lbs.)	(mm)
Oh43	1.48	176.1	0.52
M14	2.11	130.7	0.60
N31	2.51	584.9	0.63
B14A	2.97	855.2	0.65
B37	4.88	1,282.0	0.70
LSD(5% level)	0.25	174.4	0.05
C.V. (%)	7.2	23.5	6.9

Table 2. Example: ANOVA of stalk section weight for (T 1-9c x B14A) Oh43.

Source	DF	Mean Square	F Value
Regression	6	2.84	7.31**
Loc	1	0.06	0.16
Rep : Loc	4	2.75	7.06**
Classes	1	6.02	15.48**

**Significant at the 1% level.

The testcrosses were grown in 1975 at Columbia, Missouri, and Belleville, Illinois. Plants were classified as fertile or semi-sterile by anther examinations and verified by seed-set at harvest. Stalk sections from the second internode above ground level were evaluated. Data were analyzed separately for each testcross as illustrated in Table 2 for stalk section weight involving (T1-9c x B14A) Oh43. Mean differences between fertile and semi-sterile plants (classes) for these characters are given in Tables 3, 4, 5, and 6. Significance implies that a gene or genes might be located in either or both arms involved in that specific translocation. When two translocations involving a common arm show a significant difference between the fertile and semi-sterile plants, it indicates that a gene for the trait under study is present on that arm. However, designation of a gene(s) in a specific chromosome arm does not rule out the possibility that the gene might actually be in the other arm of that chromosome but close enough to the break point to show linkage. Arms that could not be completely ruled out have been noted as 'possible'. A summary of the chromosome arms is given in Table 7 for stalk section weight, in Table 8 for crushing strength, and in Table 9 for rind thickness.

Additional translocations involving the following arms need to be studied further: 1L, 3L, 5S, 6S, 6L, 8S, 10S, and 10L. The testcrosses marked —— have been produced and will be reported later. This study indicates that the three stalk characters are conditioned by several genes. Chromosomes or chromosome segments favorable to stalk quality may be transferred into breeding populations through translocations as described by Burnham (In: Plant Breeding, A Symposium, ed. K. J. Frey:139-187). It may also be possible to transfer desirable genes through use of B-A translocations.

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Table 3. Mean differences in stalk section weight between fertile and semi-sterile plants for two sets of testcrosses.

Translocation	Break Position	Oh43					
		B14A			B37		
		Difference	F	SS	Difference	F	SS
T1-9c	1S.48 - 9L.22	0.33**	46	50	0.57**	55	49
T2-3c	2S.46 - 3S.52	0.00	56	39	-	-	-
T2-9(5257)	2L.28 - 9L.20	0.52**	43	57	0.59**	71	32
T2-9d	2L.83 - 9L.27	-	-	-	0.37**	62	41
T3-9g	3L.40 - 9L.14	0.21	46	51	0.00	45	61
T3-10(4383)	3S.23 - 10L.30	0.00	39	61	0.12	65	36
T4-5(6831)	4S.32 - 5S.59	0.22	83	16	0.37**	39	50
T4-6a	4L.37 - 6L.43	0.38**	54	46	0.45**	51	44
T4-9b	4L.90 - 9L.29	0.08	49	45	0.24*	57	45
T4-9e	4S.53 - 9L.26	0.18	60	36	0.61**	54	46
T5-9a	5L.69 - 9S.17	0.28*	53	53	0.43**	56	47
T5-10a	5L.14 - 10S.54	0.33*	51	51	0.01	53	48
T6-9a	6S.79 - 9L.40	0.19	53	42	0.44**	50	40
T7-9(4363)	7ct. - 9ct.	0.49**	45	53	0.32*	45	53
T7-9a	7L.63 - 9S.07	0.40**	40	55	0.74**	51	53
T7-9b	7S.76 - 9S.19	-	-	-	0.07	77	14
T8-9(4643)	8S.37 - 9L.11	0.32**	52	45	0.46**	48	53
T8-9(4713)	8L.70 - 9ct.	0.30**	50	49	0.33	50	54
T8-9(6673)	8L.35 - 9S.31	0.15	48	56	0.14	46	51

*,** Significant at the 5% and 1% level, respectively.
F = No. of fertile plants; SS = No. of semi-sterile plants.

Table 4. Mean differences in stalk section weight between fertile and semi-sterile plants for two sets of testcrosses.

Transloction	Break Position	N31					
		B14A			B37		
		Difference	F	SS	Difference	F	SS
T1-9c	1S.48 - 9L.22	0.18	52	48	0.42**	48	51
T2-3c	2S.46 - 3S.52	0.09	45	55	0.06	48	46
T2-9(5257)	2L.28 - 9L.20	0.14	62	44	0.67	101	3
T2-9d	2L.83 - 9L.27	0.16	63	38	0.79**	52	49
T3-9g	3L.40 - 9L.14	0.18	56	47	0.27*	40	62
T3-10(4383)	3S.23 - 10L.30	0.02	54	47	0.21*	42	56
T4-5(6831)	4S.32 - 5S.59	0.07	64	36	0.08	66	33
T4-6a	4L.37 - 6L.43	0.07	54	43	0.39*	59	45
T4-9b	4L.90 - 9L.29	0.09	54	50	0.22	63	37
T4-9e	4S.53 - 9L.26	0.41**	49	49	0.54**	56	44
T5-9a	5L.69 - 9S.17	0.29*	55	44	0.47**	56	46
T5-10a	5L.14 - 10S.54	0.02	54	46	0.25	53	46
T6-9a	6S.79 - 9L.40	0.35*	50	42	0.22*	60	38
T7-9(4363)	7ct. - 9ct.	0.32**	56	46	0.35**	71	35
T7-9a	7L.63 - 9S.07	0.12	44	55	0.37**	44	57
T7-9b	7S.76 - 9S.19	0.39*	74	26	0.26	81	19
T8-9(4643)	8S.37 - 9L.11	0.16	46	47	0.31*	55	43
T8-9(4713)	8L.70 - 9ct.	0.35**	46	56	0.59**	39	51
T8-9(6673)	8L.35 - 9S.31	0.32*	51	52	0.13	52	44

*, ** Significant at the 5% and 1% level, respectively.
F = No. of fertile plants; SS = No. of semi-sterile plants.

Table 5. Mean differences in crushing strength between fertile and semi-sterile plants for four sets of testcrosses.

Translocation	Break Position	Oh43		N31	
		B14A	B37	B14A	B37
T1-9c	1S.48 - 9L.22	110.2**	18.8	46.5	25.1
T2-3c	2S.46 - 3S.52	106.3	-	67.1	52.8
T2-9(5257)	2L.28 - 9L.20	42.1	84.4	59.7	130.1
T2-9d	2L.83 - 9L.27	-	89.9	- 74.0	183.1
T3-9g	3L.40 - 9L.14	32.4	- 76.0	186.4**	- 4.5
T3-10(4383)	3S.23 - 10L.30	- 31.7	38.6	171.2*	111.3
T4-5(6831)	4S.32 - 5S.59	105.8	133.0	27.0	33.3
T4-6a	4L.37 - 6L.43	19.4	194.5*	281.0**	71.5
T4-9b	4L.90 - 9L.29	- 66.2	- 93.1	45.5	- 76.0
T4-9e	4S.53 - 9L.26	65.6	103.7	136.7	221.8**
T5-9a	5L.69 - 9S.17	- 49.0	171.8**	141.4	70.9
T5-10a	5L.14 - 10S.54	- 31.4	205.2**	- 19.8	40.5
T6-9a	6S.79 - 9L.40	118.1	116.0	34.4	89.6
T7-9(4363)	7ct. - 9ct.	94.3	26.0	193.1**	112.0
T7-9a	7L.63 - 9S.07	126.6	154.8**	37.8	132.0
T7-9b	7S.76 - 9S.19	-	22.1	168.7	- 19.1
T8-9(4643)	8S.37 - 9L.11	194.2*	141.1	205.1**	96.2
T8-9(4713)	8L.70 - 9ct.	33.7	100.4	107.2	389.4**
T8-9(6673)	8L.35 - 9S.31	39.0	45.7	30.5	100.0

*, ** Significant at the 5% and 1% level, respectively.

Table 6. Mean differences in rind thickness between fertile and semi-sterile plantsfor four sets of testcrosses.

Translocation	Break Position	Ch43		N31	
		B14A	B37	B14A	B37
T1-9c	1S.48 - 9L.22	0.03	0.02	0.00	0.00
T2-3c	2S.46 - 3S.52	0.04*	-	0.02	0.03**
T2-9(5257)	2L.28 - 9L.20	0.04	0.02	0.03	0.05
T2-9d	2L.83 - 9L.27	-	0.02	0.02	0.00
T3-9g	3L.40 - 9L.14	0.02	0.00	0.01	0.00
T3-10(4383)	3S.23 - 10L.30	0.01	0.01	0.02	0.03
T4-5(6831)	4S.32 - 5S.59	0.01	0.00	0.01	0.00
T4-6a	4L.37 - 6L.43	0.01	0.00	0.02	0.04*
T4-9b	4L.90 - 9L.29	0.01	0.00	0.00	0.00
T4-93	4S.53 - 9L.26	0.00	0.02	0.03*	0.02
T5-9a	5L.69 - 9S.17	0.01	0.01	0.03	0.01
T5-10a	5L.14 - 10S.54	0.03	0.03	0.02	0.01
T6-9a	6S.79 - 9L.40	0.01	0.01	0.02	0.03
T7-9(4363)	7ct. - 9ct.	0.03	0.91	0.02	0.01
T7-9a	7L.63 - 9S.07	0.02	0.04**	0.00	0.00
T7-9b	7S.76 - 9S.19	-	0.07**	0.00	0.04
T8-9(4643)	8S.37 - 9L.11	0.00	0.01	0.01	0.02
T8-9(4723	8L.70 - 9ct.	0.01	0.03	0.02	0.05*
T8-9(6673)	8L.35 - 9S.31	0.01	0.00	0.00	0.01

*, ** Significant at the 5% and 1% level, respectively.

Table 7. Summary of chromosome arms[a/] carrying gene(s) for stalk section weight.

Chromosome number	Oh43		N31	
	B14A	B37	B14A	B37
1	Short	Short possible	-	Short possible
2	Long	Long possible	-	Long possible
3	-	-	-	Long possible
4	-	Long & short possible	Short	-
5	Long	Long & short possible	-	Long possible
6	Long	Long & short possible	Short	Long & short possible
7	Long	Long, close to centromere	-	Long possible
8	Short	Short possible	Long possible	-
9	Centromere	Short possible	Long & short close to centr.	Long & short
10	Short possible	-	-	Long
Total Arms	8	12	4	10

a/The location of a gene(s) in a specific chromosome arm because linkage with the break point in that arm has been found, does not rule out the possibility that the gene(s) might actually be in the other arm of that chromosome.

Table 8. Summary of chromosome arms carrying gene(s) for stalk crushing strength.

Chromosome number	Oh43		N31	
	B14A	B37	B14A	B37
1	Short	-	-	-
3	-	-	Long	-
4	-	-	-	Short possible
5	-	Long	-	-
6	-	Long	Long	-
7	-	Long	Centromere	-
8	Short	-	Short	Long possible
10	-	-	Long possible	-
Total arms	2	3	5	2

Table 9. Summary of chromosome arms carrying gene(s) for rind thickness.

Chromosome number	Oh43		N31	
	B14A	B37	B14A	B37
2	Short	-	-	Short
4	-	-	Short possible	-
6	-	-	-	Long
7	-	Long & short	-	-
8	-	-	-	Long possible
Total arms	1	2	1	3

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Effects of genotypes and cytoplasms on agronomic performance

Until recently only perfunctory attention has been given to effects of cytoplasms in maize on agronomic performance. Interest in cytoplasms increased substantially after *Helminthosporium maydis* race T devastated the 1970 corn crop in various areas of the United States. This experience emphasized the importance of cytoplasm diversity. However, very little is known about the effects of different cytoplasms on agronomic performance.

The objective of this study was to determine the effect of four cytoplasms in four different inbred line backgrounds on agronomic characters and disease reaction. Four inbreds (N28, Mo17, CI21E, B37) selected for this study had been backcrossed seven generations for conversion to the three cytoplasms, cms-T, cms-S and cms-C, including the normal cytoplasms; 16 combinations were studied.

A split plot randomized complete block design with six replications was used and the following characteristics were studied: grain yield, kernel weight, days to silk, leaf area, number of leaves per plant, Stewart's wilt, stalk section weight, rind thickness and stalk crushing strength. Most measurements were taken on an individual plant basis within a plot but averaged to give a plot mean. Statistical analyses and correlation coefficients were determined on a plot basis (Table 1).

Table 1. Average for several agronomic characters showing the effect in four inbreds and four cytoplasms.

Inbred	Yield g/p	Plant Height cm	Ear Height cm	N° leaves p/p.	100 Kernel W/g	Leaf area cm^2	Rind Punct. (p)	Probe Moist. %	N°-of nodes/p	Stewarts Wilt	Days Silk	Section Stalk W/g	Rind Thick. cm	Crush Stalk Sect.	Cob Weight (g)
N28	33.6 a	110 a	50 a	11.7 a	22.4a	477a	9.41 a	71.8 a	10.5	3.5 a	78 a	2.57 a	.57 a	335 a	16.4 a
Mo 17	52.2ab	139 b	59 b	10.1 b	29.4b	450a	8.50 b	59.8 b	10.7	2.0 c	70 b	1.97 b	.54 a	497 b	13.4 a
CI21E	42.3bc	110 a	59 b	12.6 c	21.5a	654b	10.70 c	72.5 a	10.3	2.7 b	79 a	3.96 c	.56 a	725 c	17.5 b
B 37	24.8c	132 c	57 b	10.4 b	21.8a	513c	10.57 c	75.0 a	10.9	3.4 a	69 c	3.83 c	.65 b	828 d	20.4 b
F Test for Inb.	*	**	**	**	**	**	**	**		**	**	**	**	**	**
LSD	13.8	5.4	4.9	0.53	2.5	50.4	0.79	5.2		0.22	3.6	0.27	0.04	83.0	4.1
N	27.4	125	55a	11.2	22.5a	533a	9.59	70.3	10.2	2.9a	73 a	3.15	.58	523 a	16.0
T	32.0	118	54a	11.0	23.9a	496b	9.55	68.7	10.5	3.1b	75 b	3.04	.58	566 a	15.6
C	37.7	123	58b	11.2	24.6a	517b	9.80	70.7	10.7	2.7c	75 b	2.94	.58	674 b	17.0
S	46.6	125	58b	11.4	24.0a	549c	10.37	69.3	11.1	3.1a	73 a	3.21	.59	623 b	19.0
F Test for Cytoplasms			**			*				**	*			**	
LSD			2.4			36.4				0.24	1.4			88.4	

*, ** Denotes statistical significance at the 0.05 and 0.01 levels, respectively.

Means followed by the same letter(s) are not significantly different at the 0.05 level.

Estimates of yield were made but the results may be meaningless due to severe drought. Two of the six replications were hand pollinated and the remaining four were allowed to open pollinate; grain was produced on the hand pollinated material but very little on the open pollinated. Differences in grain weight among inbred lines were significant but among cytoplasms were not, nor was the inbred line by cytoplasm interaction. A more refined experiment with more replications and with additional pollen sources would be essential to determine whether different cytoplasms affect yield. Significant differences among cytoplasms were found for leaf area, Stewart's wilt, ear height, days to mid silk and crushing strength. Cytoplasm cms-S had greater leaf area, higher Stewart's wilt rating and greater ear height than the other cytoplasms. The C cytoplasm had the highest crushing strength and the normal cytoplasm the lowest. Cytoplasms T and C had two days later mid silking date than the normal and S cytoplasms. A significant interaction of inbred lines by cytoplasms was observed for probe moisture, ear height, and crushing strength. Although different cytoplasms appeared to affect some of the traits studied, further studies should be conducted to confirm the results obtained in this study.

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Usefulness of local varieties for developing resistant varieties to Philippine downy mildew disease

Evaluation of local varieties as genetic resources having resistant gene(s) to Philippine downy mildew disease, *Sclerospora philippinensis* Weston, has been carried out. The materials used in the present investigation were as follows: (NE#1 x Ph 9 DMR)F_1, Aroman, MIT VAR 2, Mimies(MIT), Tiniguib(CMU), Cebu, Marianas, Maguindanao Red, Cotabato, White Glutinous DMR, Isabela(CVIT), La Granja Popcorn, and UPCA VAR 3. MIT VAR 2, White Glutinous DMR, and UPCA VAR 3 are improved varieties, (NE#1 x Ph 9 DMR)F_1 is a single cross, and the other nine are local varieties with provincial names of the Philippines.

Experimental design was randomized complete blocks with three replications in which each plot had 50 plants. Inoculation was conducted by the method of Yamada et al. (JARQ 10:168, 1976) with 30×10^3 conidia/ml at 1.5-2.0 leaf stage of seedlings. This test was carried out at an experimental field of University of Philippines at Los Banos, College of Agriculture, where no natural infection could take place since there were no infected plants around. Diallel analysis on the inheritance of susceptibility of all entries was done by the program DIALLA and DIALLB coded by Kumagai (Bul. Comput. Cen. Res. Agr. For. Fish. A 1:273, 1968).

Infection percentages, based upon number of seedlings with systemic symptoms obtained on the 26th day after the inoculation, were compiled and their statistical parameters were calculated after arc-sine transformation. In Table 1, a diallel table of means in every array and other parameters is laid out. Infection percentages of UPCA VAR 3 and La Granja Popcorn used here as the susceptibles were not so high as expected, 60.34(75.5%) and 72.75 (91.2%), respectively. Correlation coefficients among genetic parameters are tabulated in Table 2. These data indicated that there were extremely high correlations between Mid-parent and F_1 means or G.C.A., between F_1 means and G.C.A., and between Heterosis and S.C.A.

The Wr/Vr graph in Fig. 1 indicates that the degree of dominance was partial with average dominance 0.594 and with regression coefficient of almost unity

Table 1. Diallel table on infection percentage of Philippine downy mildew disease of F_1s from crossing among thirteen materials including local varieties and of parents *per se* on the main diagonal on 26th day after inoculation (Inoculated by suspension of 30 × 10^3 conidia/ml at 1.5-2.0 leaf stage on February in 1975).

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	Rowwise mean	G.C.A. effect	Cytoplasmic effect
1. Maguindanao Red	8.19	8.05	5.42	22.32	9.47	25.79	20.14	20.29	33.18	29.36	45.00	27.76	40.15	22.69	-6.91	0.78
2. MIT VAR 2	20.42	20.65	21.36	21.35	19.26	27.42	28.84	22.87	29.63	23.38	22.78	30.91	36.63	25.04	-5.39	1.62
3. Mimies (MIT)	11.54	16.49	24.19	25.48	22.21	15.38	16.47	28.47	13.97	31.89	37.28	29.90	41.01	24.18	-4.56	-0.08
4. Tiniguib (CMU)	15.81	18.65	17.47	22.04	21.79	21.78	22.53	18.34	32.68	17.71	29.35	30.39	36.62	23.47	-4.84	-0.50
5. Marianas	15.68	13.63	20.66	9.88	27.91	18.05	28.02	28.73	14.76	33.06	24.08	30.32	51.71	24.35	-4.51	0.04
6. White Glutinous DMR	25.78	14.73	21.81	22.00	17.47	13.87	24.68	25.72	17.16	24.22	25.09	30.26	40.09	23.30	-4.53	-0.99
7. Isabela (CVIT)	24.42	25.09	39.16	23.69	29.35	20.95	20.39	28.54	31.56	25.84	38.37	45.63	43.88	30.53	-0.14	1.85
8. Aroman	26.77	21.93	18.73	23.53	21.94	15.70	28.22	26.83	21.06	26.92	21.46	25.84	37.03	24.28	-2.94	-1.59
9. Cotabato	28.11	20.97	28.62	24.65	21.70	28.22	28.84	26.75	35.74	32.75	36.79	32.49	52.18	30.60	1.39	0.40
10. Cebu	19.87	23.58	33.65	30.81	19.50	33.35	29.20	28.12	37.25	31.39	39.58	39.96	39.36	31.38	2.86	-0.49
11. (NE #1 x Ph 9 DMR) F_1	25.42	27.20	18.90	22.69	22.08	33.62	27.44	31.68	37.58	28.79	35.84	45.98	46.95	31.14	3.78	-1.46
12. UPCA VAR 3	20.42	37.16	24.59	31.03	47.16	28.36	30.87	36.58	38.61	58.86	44.42	60.34	58.76	39.78	9.62	1.34
13. La Granja Pop-corn	32.13	35.34	41.74	38.56	34.89	46.15	42.93	34.33	44.21	54.03	42.78	53.20	72.75	44.08	16.19	-0.93
Columnwise mean	21.12	21.80	24.33	24.46	24.46	25.28	26.82	17.47	29.38	32.17	34.06	37.10	45.94	18.82		

N.B. All figures in the table are after arc-sine transformation.

Table 2. Correlation coefficients among some genetic parameters from diallel table (13 × 13) shown in Table 1.

	MP	F_1	G.C.A.	H.	S.C.A.	Reciprocal
Mid-parent	1,000	0.846**	0.949**	-0.462	-0.161	-0.099
F_1 mean		1.000	0.916**	0.028	0.299	-0.192
G. C. A.			1.000	-0.270	-0.109	-0.116
Heterosis				1.000	0.713**	-0.111
S. C. A.					1.000	-0.200

(b = 0.945 ± 0.136). Moreover, there was significant correlation (r = 0.697) between parental value and (Vr+Wr). The $\overline{Vr}$-$\overline{Wr}$ relationship suggests that the two susceptible materials, UPCA VAR 3 and La Granja Popcorn, were the recessive parents and the other nine varieties, except Marianas and Cebu, had dominant gene(s).

Mochizuki (Trop. Agr. Res. Ser. 8:179, 1975) pointed out that resistance at the open-pollinated variety level was controlled by polygenic systems accompanied by no dominance, based on results reported in several papers. The test reported here was followed by the complete diallel table on recommended varieties and local open-pollinated ones, particularly collected at heavily infected areas, and was conducted under artificial inoculation. Results obtained seem to be different from those already published data. Since degree of dominance for resistance to Philippine downy mildew disease is regarded as partial dominance, it is concluded that the susceptibility was governed by recessive gene(s). Owing to high correlation between MP or F_1 and G.C.A., it seems possible to introduce resistant gene(s) from local varieties for breeding. In this test, a limited number of local varieties was used as materials for diallel analysis, regardless of whether they were typical local varieties or not. So, based on the results here it is expected to collect further the local varieties of maize as genetic resources having resistant gene(s) to Philippine downy mildew.

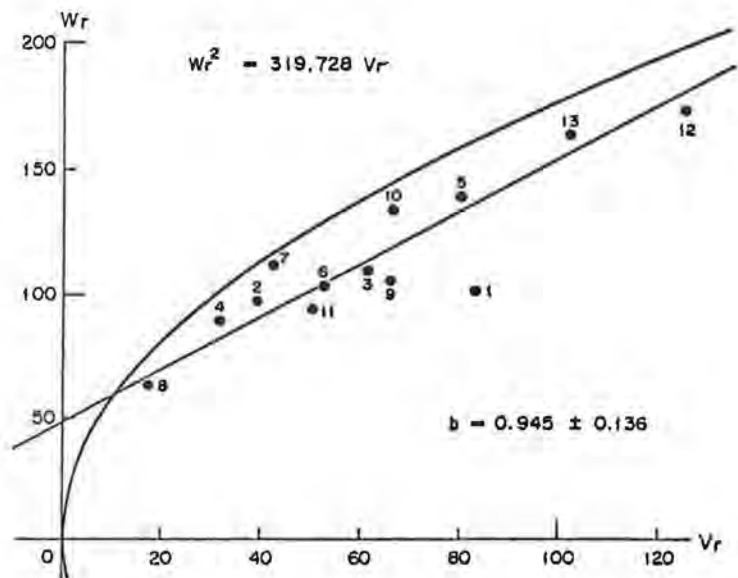

Fig. 1. Vr-Wr graph for infection percentages of Philippine downy mildew disease in thirteen materials (on arc-sine transformed data). Numerals in the figure represent materials shown in Table 1.

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<u>Two cycles of recurrent selection for hydroxamate concentration</u>

Cyclic hydroxamic acids have been implicated in the resistance of maize to several insects and fungi. The objective of this study was to perform two cycles of simple recurrent selection to increase hydroxamate concentration in maize.

Five inbred lines *Bx Bx*, B49, C131A, Oh45, and W22 were chosen for use from an original source population of 13 inbred lines varying widely in hydroxamate content. These five lines possessed the highest hydroxamate content according to an analysis by the rapid procedure (Long et al., Crop Sci. 14:601, 1974).

The selected lines were grown in the greenhouse for inter-pollination in the spring of 1974. Pollen was collected from each of these inbreds, mixed in approximately equal proportions and placed on silks of the same inbred lines. Seed was then harvested from the intercross population and bulked, and a random sample was planted in the field the following summer. Approximately 300 plants were self-pollinated and the bulked seed was saved.

A portion of this seed was planted in the greenhouse in the Fall of 1975. Two hundred and sixty-three plants 36 to 40 cm tall were analyzed non-destructively for hydroxamate concentration using the rapid procedure, and the fifteen plants having the highest concentration were selected to make up the next intercross population. At maturity these plants were intercrossed by hand in all combinations. Selfed seed was also obtained from a number of these high hydroxamate plants. The procedure thus far constituted one cycle of simple recurrent selection. An additional cycle was performed in the summer and fall of 1976.

Heritability estimates were obtained according to the equation $h^2 = (X_o - \overline{X})/(X_p - \overline{X})$, where X_o = the mean of the selected population and $\overline{X}$ = the mean of the original source population.

One cycle of recurrent selection increased the original population mean from 0.54 mg to 0.79 mg hydroxamates/g fresh weight, and an additional cycle of recurrent selection increased this mean to 0.93 mg hydroxamates/g fresh weight. Heritability estimates for the two populations were calculated as $h^2 = 0.66$ and $h^2 = 0.54$, respectively.

B. J. Long, G. M. Dunn and D. G. Routley

Relation of hydroxamate concentration to resistance to *Helminthosporium turcicum* in the field

In 1959 the cyclic hydroxamate 2,4-dihydroxy-7-methoxy-1,4-benzoxazin-3-one (DIMBOA) was first reported in maize and has since been directly implicated in resistance to several pathogens. DIMBOA occurs naturally in the glucosidic form and is converted to the fungitoxic aglucone through mycelial penetration or mechanical injury.

In our study, 16 inbred lines of maize commonly used as parents of many hybrids in the U. S. were utilized to test the relationship of concentration of hydroxamates and resistance to *Helminthosporium turcicum* in the field. One set of the 16 inbreds was analyzed for hydroxamates non-destructively by a rapid procedure using stem tissue from seedlings 36 to 40 cm high (Long et al., Crop Sci. 14:601, 1974). The same set of inbred lines was similarly analyzed at mid-silking stage by Hamilton's procedure using leaf tissue (J. Agric. Food Chem. 12:14, 1964). A third set of the 16 inbred lines was inoculated in the field with spore suspensions of *H. turcicum* and evaluated for resistance at mid-silking stage using a visual rating scale.

A highly significant correlation ($r = 0.76$) was obtained between concentrations of hydroxamates in stem tissue from seedlings 36 to 40 cm high and DIMBOA concentrations in leaf tissue at the mid-silking stage. A significant negative correlation ($r = -0.57$) was obtained between concentrations of hydroxamates in seedling stem tissue and susceptibility to *H. turcicum*. Similarly, a highly significant negative correlation ($r = -0.64$) was obtained between concentrations of DIMBOA in leaf tissue from inbreds at the mid-silking stage and susceptibility to *H. turcicum*. From these data we feel that the rapid procedure provides a reasonable measure of resistance to *H. turcicum* in the field.

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Sensitivities of purified alcohol dehydrogenase (ADH) to the endogenous ADH-specific inhibitor in maize

Commercially available purified yeast ADH, horse liver ADH (both from Sigma) and purified maize ADH (ADH-2F) from our laboratory were subjected to the ADH-specific inhibitor (Ho and Scandalios, 1975, Plant Physiol. 56:56) from different corn tissues. It was found that ADH from horse liver is almost totally insensitive to the inhibitor in all cases; however, yeast ADH exhibits a susceptibility similar to that of maize ADH (Table 1). In our previous report (IB 1976) we suggested that different isozymes may have different sensitivities to the endogenous inhibitor and that different variants of the same isozyme may have the same sensitivities. Although the specific mechanism(s) of the inhibitory effect observed in these studies remains ambiguous, our results indicate that the similarities of this enzyme, ADH (including isozymes), from different organisms may be examined on the basis of inhibitor sensitivity. Thus, the ADH-specific inhibitor presents a potential parameter for studying the possible structural divergence of enzyme molecules at the sub-unit level.

Table 1. Percent of inhibition of ADH from different organisms by the endogenous maize ADH-specific inhibitor(s) from different tissues.

ADH Source	Source of Inhibitor(s)[b]				
	Buffer[a]	Scutellum	Root	Shoot	Endosperm
Maize	1.05±0.70	41.56±7.28	60.56±2.22	56.38±2.66	3.91±1.13
Yeast	2.47±1.49	27.93±7.78	44.16±1.43	66.86±4.14	13.10±2.19
Horse liver	1.35±1.35	1.04±0.56	12.30±2.19	15.85±3.28	0

[a]Buffer -- Glyclglycine (pH 7.4, 25 mM).
[b]Tissue extract from 7-day-old seedlings (1 g fr. wt./10 ml buffer).

Yiu Kay Lai and John G. Scandalios

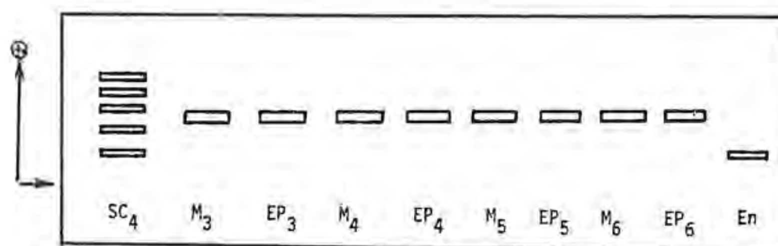
Catalase isozymes in etiolated epicotyls: a mitochondrial-specific form

In the highly inbred maize strain W64A, catalase isozyme Ct_1 is present in the developing kernels and in the dry seed stage; the Ct_2 isozyme dominates in the scutellum during germination (Scandalios, Isozymes III, p. 213-238, 1975). One catalase isozyme, observed both in meso- and epicotyls of etiolated seedlings, has a distinctive mobility on starch gel electrophoresis (Figure 1).

In our studies on the intracellular distribution of catalase isozymes in the scutellum, we found catalase to be associated with the glyoxysomes and the soluble fraction (Scandalios, J. Hered. 65:28, 1974). More recently we found, upon fractionating cells from the epicotyl, that there exists a fraction of catalase clearly associated with the mitochondria (using cytochrome oxidase as the marker enzyme)--see Figure 1. No detectable microbody marker enzymes (i.e., isocitratase) could be measured either in the crude homogenate or from the gradient. Catalase activity in the young etiolated epicotyl is only found in the mitochondrial and

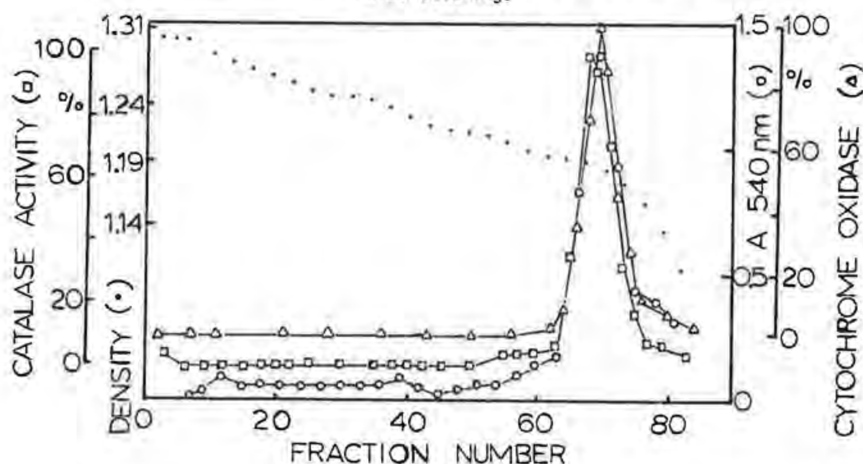
FIGURE I

Zymogram of catalase in developing etiolated tissues of maize (W64A)



SC, Scutellum; M, Mesocotyl; EP, Epicotyl; En, Liquid endosperm.
Number indicates the day after sowing.

Subcellular distribution of catalase in etiolated epicotyl of maize seedlings



soluble fractions. This mitochondrial-associated catalase cannot be washed out either by buffer or by high salts. These results indicate that the intracellular distribution of catalase is dependent on the cellular metabolic environment, which is obviously different in the etiolated epicotyl and the scutellum; the mitochondrial associated catalase may be involved in functioning in the alternative oxidase pathway of mitochondria.

W. F. Tong and J. G. Scandalios

Linkage relationship between aminopeptidase and alcohol dehydrogenase

Maize aminopeptidase exists in multiple forms. One aspect of our studies is to elucidate further the genetic background of the aminopeptidase isozymes. Previous studies have shown that the genes which code for Aminopeptidase-A and Aminopeptidase-D variants are linked but the chromosomal location is unknown (Beckman, et al., Genetics 50:899, 1964). In Table I, we present evidence of a linkage relationship between Lp (the gene that codes for Aminopeptidase-A) and a structural gene for alcohol dehydrogenase, Adh.

A backcross of the type $\frac{F}{S}\frac{F}{S}\frac{S}{F} \times \frac{F}{F}\frac{F}{F}\frac{S}{S}$ was examined, where the genotypes for Lp, Adh, and Cat (catalase) are listed in that order. F and S refer to fast or slow electrophoretic variants for the given enzyme. Kernels from three different ears (each ear represents an independent cross) were scored. The procedure was as follows. Electrophoresis of liquid endosperm from individual kernels was performed as described previously (Scandalios, Biochem. Genet. 3:37, 1969) with a starch-gel medium and a Tris-citrate buffer system. After electrophoresis the gel is sliced horizontally into three 2 mm slices. The gel slices are stained for aminopeptidase, alcohol dehydrogenase, and catalase. Thus, each kernel is genotyped with respect to these three enzymes.

Table 1 gives the number of each genotypic class found for aminopeptidase with respect to alcohol dehydrogenase, aminopeptidase to catalase, and alcohol dehydrogenase to catalase. In all cases the genotype of the first listed enzyme is

Table 1. Evidence of a linkage relationship between aminopeptidase and alcohol dehydrogenase genes in maize.

	Lp Adh						
	$\frac{F}{F}\frac{F}{F}$	$\frac{F}{F}\frac{S}{F}$	$\frac{S}{F}\frac{F}{F}$	$\frac{S}{F}\frac{S}{F}$	Total	Chi-square	Recombinants/Total
Ear 1	56	15	19	27	117	35.2**	0.29
Ear 2	52	23	27	51	153	18.7**	0.33
Ear 3	54	22	25	52	153	22.9**	0.31
Total	162	60	71	130	423	66.7**	0.31

	Lp Cat						
	$\frac{F}{F}\frac{S}{S}$	$\frac{F}{F}\frac{F}{S}$	$\frac{S}{F}\frac{S}{S}$	$\frac{S}{F}\frac{F}{S}$	Total	Chi-square	Recombinants/Total
Ear 1	33	34	23	21	111	4.86	0.51
Ear 2	36	38	45	33	152	2.05	0.55
Ear 3	11	31	20	20	82	8.83*	0.62
Total	80	103	88	74	345	5.48	0.55

	Adh Cat						
	$\frac{F}{F}\frac{S}{S}$	$\frac{F}{F}\frac{F}{S}$	$\frac{S}{F}\frac{S}{S}$	$\frac{S}{F}\frac{F}{S}$	Total	Chi-square	Recombinants/Total
Ear 1	33	36	22	19	110	7.45	0.53
Ear 2	39	39	42	32	152	1.42	0.53
Ear 3	14	30	17	21	82	7.07	0.57
Total	86	105	81	72	344	6.77	0.54

*Significant at the 0.05 level.
**Significant at the 0.001 level.

written first. If the genes coding for the two enzymes are unlinked or are very far apart on the same chromosome, then a 1:1:1:1 ratio is expected. As is seen, the outer two genotypes listed are parental types while the inner two genotypes are "new" types, i.e., possible recombinants.

In each cross examined Lp exhibits a loose linkage relationship with Adh and the chi-square value is significant at the 0.001 level. It can be seen from the data in Table 1 that the deviations from the expected allelic segregation ratios are not the cause of the large chi-square values. Only in Ear 1 is there significant deviation of aminopeptidase (71 fast:46 hybrid) and ADH (75 fast:42 hybrid) from expected 1:1 ratios.

On the other hand, the relationship of aminopeptidase to catalase and of ADH to catalase does not deviate significantly from the expected 1:1:1:1 ratio except in Ear 3 which reflects a distortion in catalase segregation ratios (31 slow:51 hybrid).

Linkage of Lp to Adh thus places both Lp and Lp2 (which, as mentioned, is known to be linked to Lp) on Chromosome 1 of maize since the ADH gene is located on chromosome 1. We are presently using other phenotypic markers on chromosome 1 in order to find the exact position of Lp and Lp2.

L. A. Ott and J. G. Scandalios

Small molecular weight DNAs associated with the "S" type of cytoplasmic male sterility

When maize mitochondrial DNA (mtDNA) was fractionated by agarose gel electrophoresis, it showed a single broad band of high molecular weight DNA at about 1.5 cm (Figure 1) for all cytoplasms studied (N, T, C, EP, S, H, EK, SD, and CA). Interestingly, members of the S group (S, H, EK, SD, and CA) manifested two additional bands which we have designated S-S (slow) and S-F (fast) DNAs. These unique bands and all bands described herein were abolished by deoxyribonuclease and unaffected by ribonuclease. The mtDNA was prepared by CsCl-ethidium bromide preparative ultracentrifugation as previously described (Science 193:158-160). The S-S and S-F DNAs have been obtained from upper and lower bands of CsCl-dye gradients which indicate that the DNA exists as open-circular or linear and supercoiled molecules. The two unique DNA bands associated with the S group of cytoplasmic male steriles (cms) are the subject of this report.

The S cms was studied in the nuclear backgrounds WF9, Mo17, FR37, WF9 x W64A, N6 x W64A, and FR37 x K21; EK, H, and SD cms were in Mo17; and CA cms was in W64A. All the above types yielded the two unique DNAs associated with the S group of cms. The cross FR37 cms S x K21 was restored for pollen fertility, but nonetheless contained the unique DNAs S-S and S-F. To further establish the association of the two unique DNAs with the S cytoplasm, we looked for the unique DNAs in 10 N (normal) cytoplasms, F6, F44, T204, W64A, WF9, 61M, A619 x A632, B37 x NC236, NC7 x T204, and B37 x NC236; in 5 sources of T cytoplasm, F44, T204, W64A, B37 x NC236, and NC 7 x T204; in 3 sources of C, Mo17, WF9, and B37 x NC236; and in 1 source of EP, A619 x A632. The

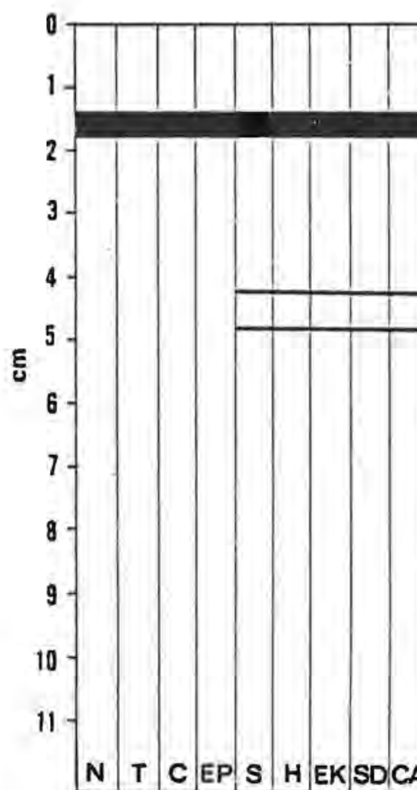


Figure 1. Agarose gel electrophoretic patterns of mitochondrial DNAs from N (normal), T, C, EP, S, H, EK, SD, and CA cytoplasms of maize. See text for details.

unique DNAs present in the S group of cms were not found in the N, T, or C cytoplasms.

Molecular weights of the S-S and S-F DNAs were estimated by two techniques, gel electrophoresis and electron microscopy (Table 1). Molecular weight determinations were made by electrophoresis in 1.5% agarose gels, with Eco RI-digested lambda and adenovirus type 2 DNA as molecular weight markers. Since the molecular weight markers were linear molecules, Hind III digests of S-S DNA were electrophoresed to better estimate the size of S-S DNA. Three fragments were obtained, with molecular weights of 1.47, 1.22, and 1.02 x 10^6 daltons, for a total molecular weight of 3.71 x 10^6 daltons. Hind III digestion of S-F DNA produced two fragments with molecular weights of 2.30 and 1.12 x 10^6 daltons, resulting in a total molecular weight estimate of 3.42 x 10^6 daltons. No apparent variation in molecular weight of the S-S and S-F DNAs was detected among the S, CA, EK, SD and H cytoplasms.

Table 1. Molecular weights of S-S and S-F DNAs as determined by gel electrophoresis and electron microscopy.

	Molecular Weight x 10^6 daltons	
DNA	Gel Electrophoresis	Electron Microscopy
S-S	3.71	3.84
S-F	3.42	3.41

The two unique DNAs associated with the mtDNA from the S cytoplasm were studied by electron microscopy. These DNAs were isolated by electrophoretic separation of the upper DNA band of a CsCl-ethidium bromide gradient, because our yield of lower band (supercoiled) DNA was insufficient for this study. Most of the DNA molecules isolated in this manner and examined by electron microscopy were linear except for a few circular molecules. Although the upper band from a dye-CsCl gradient contains circular as well as linear molecules, we have experienced consistent difficulty in obtaining high yields of circular molecules after gel electrophoresis.

The size of the linear molecules of S-S DNA was determined by measuring their length relative to the length of open-circular ΦX174 R II DNA which was used as an internal standard in the same spreading. The ratio of S-S DNA length to ΦX174 was 1.13. The molecular weight of S-S DNA is 3.84 x 10^6 when a molecular weight of 3.4 x 10^6 is used for ΦX174 DNA. Similarly, the size of S-F DNA was obtained by measuring length relative to the length of 4.67 x 10^6 molecular weight linear fragments from Eco RI-digestion of the bacteriophage lambda. The ratio of S-F DNA to the lambda DNA fragment was .73 so that the molecular weight of the S-F DNA is 3.41 x 10^6. These molecular weights of 3.41 and 3.84 x 10^6 for S-F and S-S respectively are comparable with those determined by gel electrophoresis of 3.42 and 3.71 x 10^6 for S-F and S-S, respectively.

The two sizes of low molecular weight, circular DNAs from the S cytoplasm seem to be distinct species, as demonstrated by electrophoresis of open circular and linear molecules, and by electron microscopy. Mitochondria from N and T cytoplasms also contain low molecular weight circular DNAs (Shah, et al. in preparation) but electrophoresis does not elaborate these as distinct bands. Apparently, in the N and T cytoplasms, small circular DNAs do not occur with sufficient frequency for electrophoretic visualization. The mtDNA of maize, whether from N, T, or S cytoplasm, is heterogeneous in contour length; it ranges from mini-circles (less than 3 μm) to circles as large as 33 μm. The reason for this intermolecular heterogeneity, and its relation to the unique S DNAs, is not clear. Perhaps the latter DNAs are a special form of intermolecular heterogeneity.

A most interesting characteristic of the S cytoplasms is their instability which has been described in detail by Laughnan and his associates (Theor. Appl. Genetics 43:109-116; Genetics 71:607-620). It is tempting to speculate that the circular DNAs described here are in some way related to the unstable nature of the S male-sterile cytoplasm, and in fact, may represent elements described by Laughnan and Gabay (In Birky, C. W., Jr., Perlman, P. S. and Byers, T. J., eds., Genetics and Biogenesis of Mitochondria and Chloroplasts, Ohio State University Press, pp. 330-349). They hypothesized an episomal element to explain the occurrence of fertility restoration by a nuclear gene, with the corresponding requirement that the cytoplasmic element be capable of transposition to, and stabilization in, a chromosomal site. Our data do not permit a distinction among an episomal, viral or other extraneous agent in our S cytoplasm stocks.

To date our evidence suggests that the unique DNAs associated with the S cytoplasm are confined to the mitochondria. We have interpreted our data as circumstantial evidence of a mitochondria-sterility association, but a causal relationship between the unique mtDNAs of the S cytoplasm described in this paper and the expression of male sterility in maize remains to be established.

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Evidence of chloroplast and mitochondrial DNA variation among male-sterile cytoplasms

Sources of cytoplasmic male sterility in maize have been classified into the T, C, and S groups, based on fertility restoration patterns in a variety of inbred lines. We previously (Science 193:158-160) reported that the T and N mitochondrial DNAs (mtDNAs) are distinct, using restriction endonuclease fragment analysis as an assay. These studies also showed that the mtDNA of the N recurrent parent is not inherited paternally. The present study was designed to examine the chloroplast and mitochondrial genomes of the three major sterility groups and of normal cytoplasms. MtDNAs from five lines in T cytoplasm, three lines in C cytoplasm, four lines in S cytoplasm, and seven lines in N cytoplasm were prepared. Cytoplasms EK, H, SD, and CA, members of the S group, were included. Chloroplast DNAs (ctDNAs) from most of the lines were also examined.

The DNAs were purified by cesium chloride-ethidium bromide preparative ultracentrifugation, and upper (linear and open circular DNA) and lower (supercoiled DNA) zones were collected. Routine analyses were conducted with the former since no apparent differences in fragment patterns were found between the two fractions. Restriction endonuclease Hind III and Bam I were used to digest the DNAs, and fragments were electrophoresed in 1.0% agarose gels in Tris-phosphate-Na2EDTA buffer. Restriction patterns of mtDNAs are quite complex, with 40-50 bands produced by each enzyme. A schematic diagram of the Hind III patterns is shown in Fig. 1. The fragment patterns clearly indicate that the mtDNAs of the N, T, C, and S cytoplasms are distinct. Bam I also clearly distinguished the four cytoplasms. Characteristic patterns of the T, C, and S cytoplasms were the same regardless of nuclear background. Cytoplasms H, EK, CA, and S gave Hind III patterns indistinguishable from each other and from S cytoplasm mtDNA, even though these cytoplasms have been reported to be different in terms of fertility restoration patterns. There was some variation among N cytoplasm mtDNAs, but the magnitude of their variation was much less than observed for the male-sterile cytoplasms.

The molecular weight of mtDNA was about 10^8, with some apparent variation among cytoplasms. This estimate is tentative in that the patterns are too complex with these enzymes for accurate molecular weight determinations. Interestingly, the C cytoplasm was more closely related to the N cytoplasm than were the S and T cytoplasms, using the molecular weights of common Hind III fragments as an index of relatedness. The T cytoplasm was most divergent of the three male-sterile

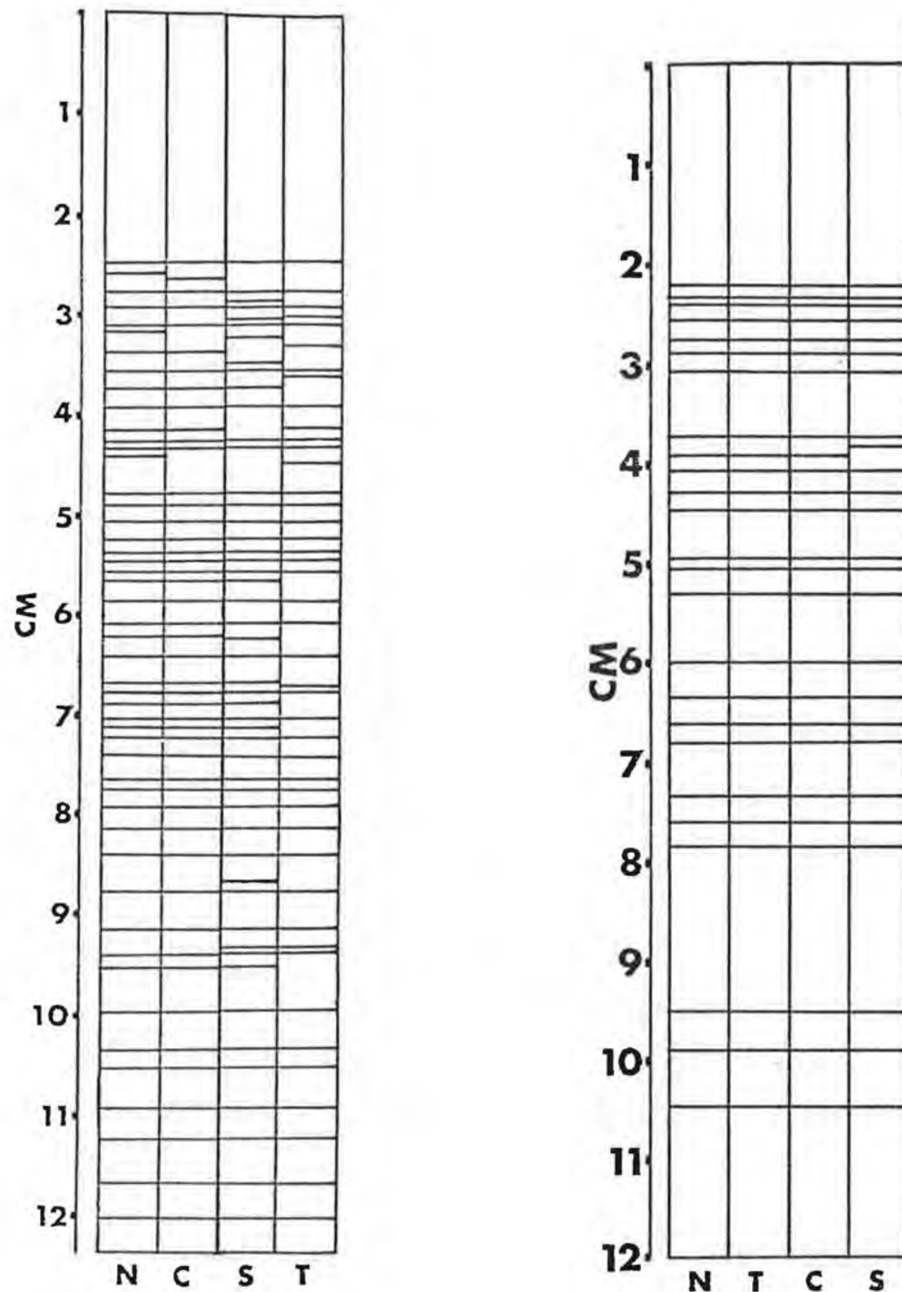

Figure 1 (left). Agarose gel electrophoretic patterns of Hin dIII-digested mitochondrial DNAs from N, C, T, and S cytoplasms. Electrophoresis was at 1.9 V/cm for 17 hr in 1% agarose.

Figure 2 (right). Agarose gel electrophoretic patterns of Hin dIII-digested chloroplast DNAs from N, T, C, and S cytoplasms. Electrophoresis was at 1.5 V/cm for 16 hr in 1% agarose.

cytoplasms, suggesting a possible basis for the susceptibility of T cytoplasm to B. maydis and P. maydis. Fragment patterns of mtDNA of all members of the S group are complicated by the presence of two unique circular DNAs (see Levings, et al., this issue). These DNAs were restricted by the endonuclease and contributed low molecular weight fragments to the S patterns.

In contrast to mtDNA, maize ctDNA restriction patterns with Hind III are quite simple (Fig. 2). Only 25 bands were observed among the N, T, C, and S cytoplasms, with several occurring in double frequency. No variation was found among the N, T, and C cytoplasms, while a slight shift of one band (3.6 - 3.8 cm in Fig. 2) was observed in the S cytoplasm. Cytoplasms H, EK, SD, and CA also were characterized by this single band shift. This slight displacement of one band in the S cytoplasm ctDNA suggests lack of paternal inheritance of the N ctDNA. The molecular weight of maize ctDNA was $7.5 - 8.0 \times 10^7$, which is similar to estimates made by other criteria.

These results imply that cytoplasmic genome variation, expecially for mtDNA, can be demonstrated among male-sterile cytoplasms. The cytoplasmic genomes appear to be not inherited through the pollen, although our assays probably would not detect 5% contamination. Restriction patterns of mtDNAs also indicate that the genome is conserved among nuclear backgrounds from diverse geographical areas; T cytoplasm, for example, was examined in lines W64A, B37 x NC236, T204, NC7 x T204, and F44. Variation of mtDNA among the T, C, and S cytoplasms is compelling evidence of mitochondria as probable carriers of factors conditioning cytoplasmic male sterility, but the evidence should be interpreted as circumstantial. Failure to distinguish ctDNAs among the N, T, and C cytoplasms with one restriction endonuclease does not prove that the genomes are identical, nor unrelated to the sterility-susceptibility traits. Other kinds of evidence will be required to effectively establish the nature of the cytoplasmic agent(s) involved.

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Plant regeneration from tissue cultures of maize

A method for regenerating complete plants from maize tissue cultures has been reported by Green and Phillips (Crop Sci. 15:417-421, 1975). Tissue cultures initiated from immature embryos from 4 of the 5 inbred lines tested subsequently formed plantlets. The proportion of embryos that formed differentiating cultures was as high as 20% in some cases, and approximately 200 complete plants were differentiated. Other efforts to regenerate plants from maize tissue cultures have been much less successful (Gresshoff and Doy, Aust. J. Biol. Sci. 26:505-508, 1973; Rafaill, this News Letter 50:84-86, 1976). Attempts to reproduce the findings described by Green and Phillips have apparently been unsuccessful according to both published (Rafaill, 1976) and unpublished accounts. In this brief note we report the regeneration of plants from maize tissue cultures using methods that reproduce as faithfully as possible the procedures and conditions described by Green and Phillips.

A188 seeds obtained from C. E. Green were grown in a greenhouse during the spring and summer of 1976. Immature embryos 1-2 mm in length were excised 14 to 20 days after self-pollination. The embryos were placed on an agar-solidified culture medium prepared as described by Green and Phillips and containing 2 mg/l 2,4-dichlorophenoxyacetic acid (2,4-D). The flat embryonic axis was placed in contact with the medium. Sixty excised embryos were incubated under conditions similar to those described by Green and Phillips with a 16/8 hr photoperiod from

fluorescent cool-white VHO tubes (Sylvania) with an intensity of 275-400 ft-c at 26-28 C. Tissue from under the scutellum of greater than 90% of the embryos proliferated and grew upwards eventually surrounding the scutellum. The tissue was pale white initially and was quite organized throughout the culture interval. After 4-5 weeks of incubation approximately 60% of the embryos gave rise to a number of "scutellar-like" structures, both colorless and green, that arched inward toward the scutellum of the excised embryo. As soon as shoot formation occurred, the nascent shoots (as many as 10 per embryo culture) were transferred to a medium containing 0.25 mg/l 2,4-D where they continued to develop. When the leaves were 1-4 cm in length, the shoots were transferred to a medium without 2,4-D, where roots formed within 3-4 days. When the plantlets were about 15 cm high and had 4-6 leaves and an extensive primary root system, they were transplanted to 6 cm peat pots containing a 1:1 peat-perlite mixture. Immediately after transplanting the plants were exposed to continuous light for 24 hr followed by the normal 16/8 hr photoperiod. The plants were maintained at 75% relative humidity at 27-28 C in light and watered with 25% MS salts (Murashige and Skoog, Physiol. Plant. 15:473-497, 1962). More than 85% of the plantlets survived the transfer to soil.

Although we have no data on the effect of subculture on the differentiating clones, this report substantiates many features of the method described by Green and Phillips. The results also emphasize the need for adherence to experimental details. Several modifications of this protocol resulted in tissue cultures that have not differentiated shoots.

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Some observations on inheritance of prolificacy

We've worked with a number of prolific sources over the years and wished to learn something about their inheritance. We grew two sets of six generations each at Mankato in 1970 that involved inbred lines where Ladyfinger pop (LP) and Southern dent (SD) were sources of prolificacy; the single eared parent was an early derivative of C103. In addition we have taken Duvick's data (1974 Crop. Sci. 14:69-71, Table 1) that involves Argentine pop (AP) and C103 grown at Johnston, Iowa, in 1965. The means were fitted by regression to Hayman's generation means analysis (Table 1).

Additive gene effects explain nearly all the genetic differences in the LP data. Additive and dominance effects explain most of the variation in the SD and AP data. A model for mean, additive, and additive x additive epistatic effects was also fitted to the SD and AP data, and it accounted for 99.5% of the variation. Addition of dominance to this model did not account for significant additional sums of squares.

Though additive and dominance effects account for 98.6 percent of the variance for the SD data, the P2 value observed at 1.62 appears too low compared with its genetic potency. If the P2 value for SD is omitted, the remaining five generations increase the fit to 99.8 percent, and indicate P2 should have had an observed value of near 2.8. P2 for SD is much later flowering (P1 - 1404 gdu, P2 - 1596 gdu) than the other materials grown at Mankato. The season during flowering and ear formation was hot and dry; later flowering of P2 was probably disadvantageous and reduced its phenotypic value.

Based on the present data inheritance of prolificacy for three sources appears to be mostly additive with some dominance effects.

Table 1. Mean prolificacy for the three sources crossed to C103.

	Ladyfinger pop		Southern dent		Argentine pop	
	Obs.	Pred.[1]	Obs.	Pred.[2]	Obs.	Pred.[2]
P1 (C103 or derivative)	1.04	1.11	1.04	1.06	.97	.91
P2 (Prolific parent)	2.80	2.83	1.62	1.84	4.45	4.18
F1	2.00	1.97	1.95	2.19	1.48	1.14
F2	1.93	1.97	1.85	1.82	1.55	1.84
BC1	1.64	1.54	1.65	1.62	1.04	1.02
BC2	2.41	2.40	2.44	2.01	2.26	2.66
SE of unweighted means	.03		.03		Not available	
R^2 for fit to model		.999		.986		.986

[1]Predicted from fitting mean and additive effects.
[2]Predicted from fitting mean, additive and dominance effects.

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Genetic polymorphism of mitochondrial enzymes: glutamate- and succinate dehydrogenases

Genetic studies of mitochondrial enzymes open up a new way to approach the possible nature of the so-called "mitochondrial complementation" that attracts particular interest in relation to the problem of heterosis in crop plants (cf. Hanson et al., Crop Sci. 15:62, 1975).

A collection comprising about 50 inbreds and 12 races of maize obtained from Krasnodar Agricultural Research Institute was used in our studies of two enzymes localized exclusively in mitochondria: matrix enzyme glutamate dehydrogenase (GDH) and succinate dehydrogenase (SDH) bound to the inner membrane. The GDH and SDH spectra were investigated primarily in the scutella of three-day-old seedlings, employing the routine procedures of disc-PAGE and tetrazolium staining (in the case of SDH, mitochondria-enriched 10,000xg sediment was used instead of total homogenate).

High resolution of GDH spectra (Fig. 1) was achieved using the alcohol dehydrogenase Adh-F band as an inner marker (R_m values were corrected in the case of Adh-S inbreds). A seven-band spectrum is characteristic of most of these genotypes, and two classes (A and B) differ only in the staining intensity of the faster bands. An inbred, C103, constitutes class C. Class A occurs with a frequency of about 2/3. Specific differences in staining of the isozymes are genetically determined and are not related to variability dependent on growth conditions. Additive inheritance of isoform staining intensity in reciprocal F_1 progenies suggests nuclear control of GDH synthesis (Fig. 2). Log R_m values of these isozymes depend in a linear mode on gel concentration, which means that the seven isozymes of the same molecular weight differ in their charge values. These data are consistent with a two-loci-control model of hexameric GDH (Pahlich, Planta 104:21, 1972; Pryor, Heredity 32:397, 1974). However, the following data do not fit into this model: (1) staining intensity distribution significantly different from binomial predicted by a two-loci model; (2) organ-specific features

in GDH spectra (Fig. 3); (3) the spectra of mixed extracts of strain C103 and any class A or B inbred or their hybrids (Fig. 4). These data suggest the presence

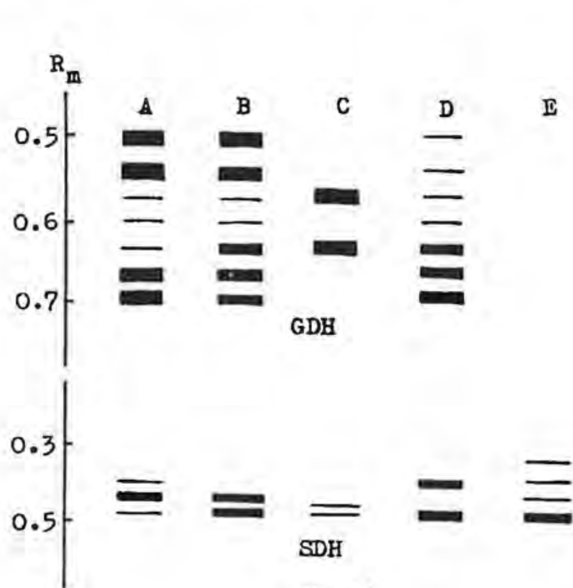


Fig.1

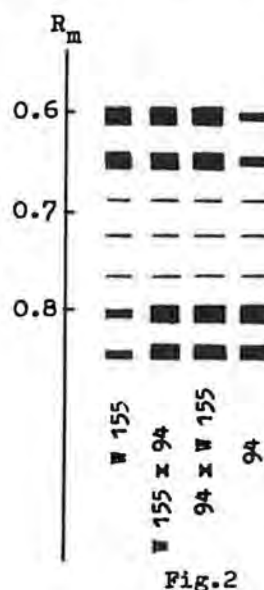


Fig.2

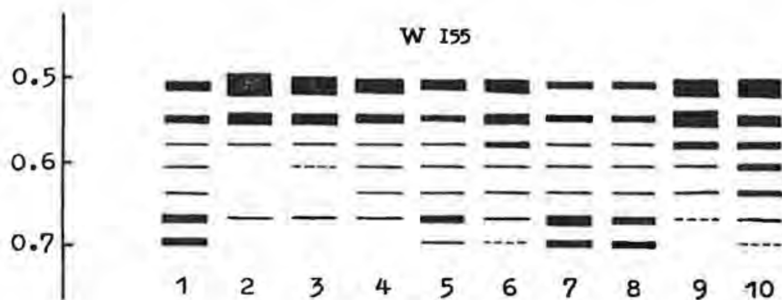


Fig.3

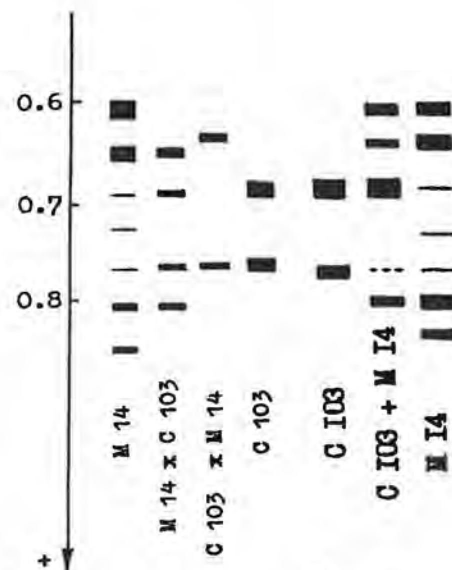


Fig.4

Figure 1. Phenotypic classes of GDH and SDH. For details cf. text.

Figure 2. Inheritance of GDH spectra.

Figure 3. Tissue specificity of GDH spectra in 3-day-old seedlings: scutellum (1), root (2), mesocotyl node (3) and coleoptile (4); in 7-day-old seedlings: leaf (5); in 1-month-old plants: stem parenchyma (6); in plants 12 days after pollination: embryo (7), endosperm (8), pericarp (9) and parenchyma of cob cortex (10).

Figure 4. GDH patterns in mixed extracts of strains C103 and M14 and their hybrids.

of some inheritable factor responsible for highly ordered association of GDH subunits and presume the existence of a third locus coding for this factor. Seven- and two-band patterns were maintained after removal of low-molecular-weight substances and partial GDH purification.

Five phenotypic classes of SDH are composed of two to four isozymes controlled by non-allelic genes (Fig. 1); class B occurs at a frequency of about 2/3. Isozymes are of the same molecular weight and differ in their charge values. No tissue- and organ-specific differences have been found in isozyme patterns. Additive spectra of reciprocal hybrids (Fig. 5) suggest nuclear control of SDH. The simultaneous appearance of two SDH classes, A and B, in the Jalisco group 33 race from Mexico and the Morocho race from Argentina is especially interesting as an apparent example of the development of genetic polymorphism in mitochondria.

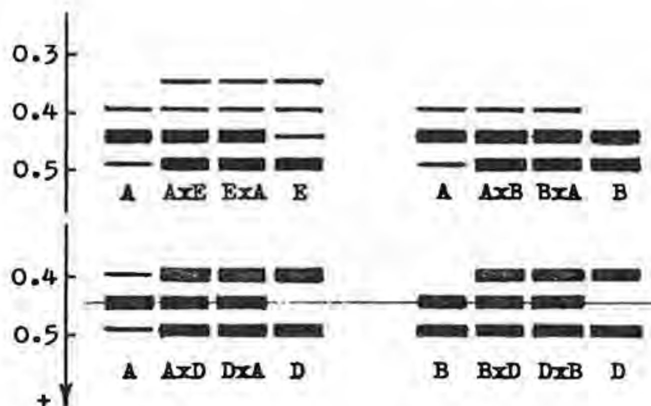


Figure 5. Inheritance of SDH spectra. A - E are phenotypic classes as in Fig. 1.

No new patterns of GDH and SDH were found in the collection of races representing most of the presumed centers of maize domestication, and the frequency distributions of phenotypic classes were similar in collections of races and inbreds.

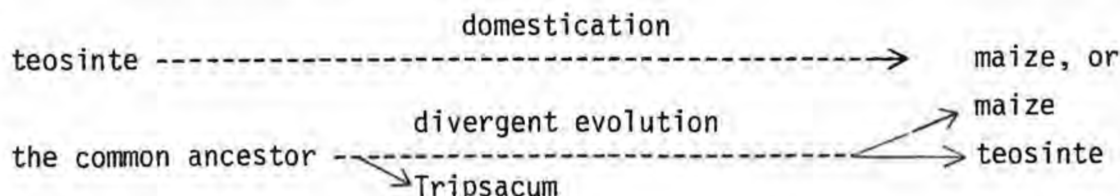
L. E. Monastyreva, T. B. Sukhorgevskaya and E. E. Khavkin

Isozyme spectra of maize, teosinte and Tripsacum

We compared isozyme patterns of glutamate (GDH), alcohol (ADH), and succinate (SDH) dehydrogenases and aspartate-aminotransferase (GOT) in scutella of seedlings of *Zea mays* L., *Zea mexicana* Mangelsdorfii (Balsas race from Guerrero, Mexico) and a tetraploid (2n = 72) form of *Tripsacum dactyloides* L; seeds were obtained from Krasnodar Agricultural Research Institute.

GDH, ADH, SDH and GOT spectra of teosinte corresponded to the phenotypic classes most frequent in maize. SDH and ADH spectra of *Tripsacum* were the same as in teosinte and most maize inbreds (62% and > 90% for SDH and ADH, respectively). A new class (D) of GDH spectra (cf. Fig. 1 of previous note), absent in maize and teosinte, was found in *Tripsacum*. The slow glyoxysomal GOT isozyme characteristic of *Tripsacum* was present in maize with a frequency of 1/6 and was absent in teosinte.

Our data are in line with observations by J. G. Waines (cf. Galinat, Annual Rev. Genet. 5:447, 1971) that reserve protein patterns are similar in maize and teosinte and distinct in *Tripsacum*. Both groups of data can be interpreted in two ways:



In our opinion, however, cytological, genetical and biochemical evidence on Maydeae taken as a whole seems to support the second assumption.

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Intracellular localization of malate dehydrogenase (MDH) and aspartate-aminotransferase (GOT) in seedling scutella

A study of mitochondrial and glyoxysomal isozyme mobilities is an important prerequisite for large-scale screening of enzyme genetic polymorphism by tissue homogenate analysis.

Scutella of three-day-old seedlings of Bukovinsky 3 hybrid (var. Gloria Yanetzki x inbred A344) were cut with a razor blade in the medium described by Dalling et al. (Biochim. Biophys. Acta 283:505, 1972). The slurry was filtered through three layers of cheesecloth, and the extract was layered upon a discontinuous sucrose gradient (20 - 60%), centrifuged for 4 h at 94,000xg and collected in 1 ml fractions. Cytochrome oxidase (CO) and catalase (C) served as marker enzymes of mitochondria and glyoxysomes (Fig. 1). Our zymographic data support the

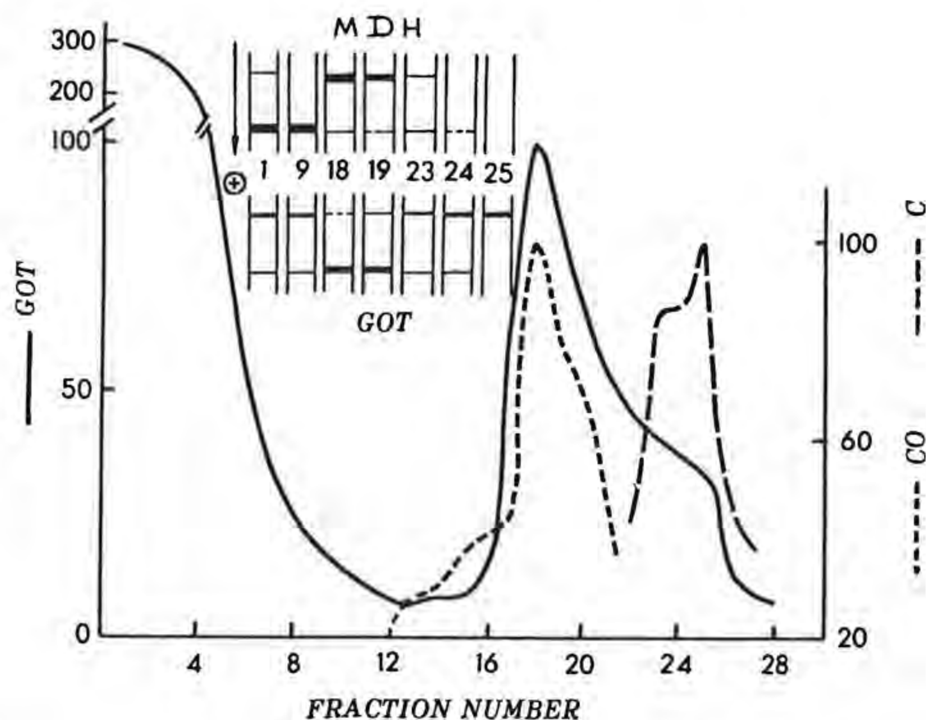

Figure 1. Intracellular localization of GOT and MDH isozymes in scutella. Enzyme activities are presented as % maximal values; for other details, cf. text.

evidence presented by other authors that the fast-moving GOT isozyme and the slow-moving MDH isozyme are of mitochondrial origin, while the opposite is true for glyoxysomes. In contrast to the results reported by Yang and Scandalios (Biochim. Biophys. Acta 384:293, 1975), we have not found multiple forms of mitochondrial MDH. So-called "cytoplasmic" isozymes of GOT and MDH seem to be related to enzyme release from glyoxysomes during tissue homogenization.

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Response of viviparous mutants to abscisic acid in embryo culture

Following its isolation from cotton and sycamore, abscisic acid (ABA) was shown to be present in a large number of plant species. Furthermore, ABA appears to be present in all tissues of the plant, the highest concentrations being localized in the seed and fruit pericarp. For this reason, and the fact that it inhibits germinating seedlings, ABA has been suggested to have a general function as a growth regulator which, by its interactions with other growth hormones, promotes seed dormancy. As such, it may also be involved in regulation of seed maturation processes determining seed size. A question arises concerning the failure of the "dormancy deficient" viviparous (vp) mutants of maize to respond to normal dormancy regulation. We have begun investigations to answer this question.

One line of evidence from gas chromatography indicates that no appreciable difference exists in ABA levels between wild-type and homozygous viviparous seeds (unpublished result, R. Newton).

Another line of investigation was begun using excised embryos and tissue culture techniques. Embryos were removed from surface-sterilized seeds and placed on an agar solidified medium (Murashige and Skoog, 1962) with various concentrations of ABA and no other growth regulators. Homozygous vp seeds were identified on an anthocyanin genetic background (purple aleurone) by their pleiotropic effect of pigment reduction (Robertson, 1955). Explanted embryos were then incubated at 34 C and 100 fc of illumination in a day:night regime of 12:12.

A preliminary run using only wild-type embryos incubated on log scale ABA concentrations indicated that a level of 1.0 mg% would significantly reduce germination frequency. Techniques were also perfected that allowed the removal of ABA-treated embryos to ABA-free environments (vermiculite tubes) and subsequent transfer to and maturation under greenhouse conditions. Using the 1.0 mg% ABA level, we tested homozygous and heterozygous vp embryos excised at various intervals post-pollination. Embryos of both genotypes and of the same age were placed on ABA-free medium as controls. Table 1 shows the results.

Table 1. Frequency of germination of variously aged vp and normal embryos on ABA^+ and ABA^- media.

Days post-pollination	No. of embryos explanted		Germination Percentage +ABA		Germination Percentage -ABA	
	vp/+	vp/vp	vp/+	vp/vp	vp/+	vp/vp
14	2	14	0.0	100.0	-	-
15	5	9	0.0	62.5	100.0	100.0
16	1	3	0.0	50.0	-	-
18	3	7	0.0	50.0	100.0	100.0
19	2	2	0.0	10.0	-	-
20	1	3	0.0	100.0	-	-
21	9	21	16.7	77.8	100.0	100.0
22	6	11	66.7	33.3	100.0	100.0
23	9	21	16.7	72.2	100.0	100.0
24	6	9	25.0	75.0	100.0	100.0
28	4	10	0.0	100.0	100.0	100.0
35	4	6	0.0	100.0	100.0	-

Around 77% of the vp/vp homozygous embryos plated on medium that inhibited wild-type growth grew at near normal rates. The vp/+ heterozygotes had about 10% germination on the same medium. Those normals that germinated never approached the growth rate of the homozygotes, indicating a real difference between the two genotypes.

These results coupled with the chromatographic analysis suggest that the viviparous phenotype is due not to a lack of ABA in the mutant seed but rather to a lack of response (dormancy) to the hormone. Current experimentation (concomitant with an expanded repetition of the above procedure) is being conducted with C-14 labeled ABA to detect the presence (or absence) of a protein receptor for ABA in these embryos.

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Comparison of univalent behavior in desynaptic, asynaptic and several trisomics

Evidence has been obtained that univalents in microsporocytes from plants homozygous for desynaptic represent true desynapsis, following (at least sometimes) events of crossing over. Pachytene in such material appears normal (or nearly so), as first reported by Nelson and Clary (J. Heredity 43:205-210, 1952). Recent observations in this laboratory show that, at diakinesis, matching and adjacent or aligned univalents (presumed homologues) may demonstrate equational separation of heterozygous knobs. This suggests that the desynaptic mutant may represent a lesion in the normal sister chromatid cohesiveness thought to be needed for maintenance of chiasmata until metaphase-anaphase I. The asynaptic mutant, on the other hand, has been reported to represent variable failure of synapsis, although regular synapsis was found in centromere regions and with lesser frequency in distal regions (Miller, O. J., Genetics 48:1445-1466).

Observations have been made in this laboratory of univalent behavior at metaphase I, anaphase I and prophase II in microsporocytes of plants which were trisomic for chromosome 5, 9, 10 or a maize-Tripsacum interchange chromosome, as well as of plants which were homozygous for asynaptic or desynaptic. In all cases, univalents may either move intact to a pole or lag and orient late on the metaphase I plate, with sister centromeres directed to opposite poles; in the latter instance, univalents of desynaptic cells tend to separate freely, sending a chromatid to each pole at anaphase I, while such univalents of asynaptic cells tend to remain in the plate region, at least until very late anaphase I or early telophase I and sometimes apparently indefinitely, while showing considerable decondensation. These laggards generally show apparent stickiness of sister centromere regions, but sometimes of distal or other regions. Univalents of all the trisomics studied show more variable behavior than the synaptic mutants, sometimes separating freely after lagging and sometimes apparently sticking together and remaining in the plate region until very late. Equational separation of the chromatids of one of the chromosomes of trivalent configurations at early anaphase I was also observed. At prophase II in the trisomics, some cells contain 10 normal dyads plus two monads (thought to have arisen from the premature separation of the chromatids of a univalent which had been distributed intact to a pole at the first division).

These observations are consistent with the suggestion that the normal development of meiotic sister chromatid cohesiveness (finally reversed normally for the last remaining regions, i.e. sister centromeres and adjacent zones, at early anaphase II) is dependent upon normal synapsis. A function of the fully elaborated

synaptonemal complex, then, might be somehow related to the development of sister chromatid cohesiveness.

Electron microscopy of the synaptonemal complex in normal, desynaptic and asynaptic cells is in progress. The quantitative relationship of synaptic failure frequency to premature separation of sister chromatids is also being explored.

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Flavonols in maize pollen

Thin layer chromatography of W22 A R-r Pr Bz pollen extracts shows the main flavonol glycosides to be different from those found in seedlings, husks and other plant tissues. Some of these 'pollen-specific' flavonol glycosides may be based on isorhamnetin (quercetin 3' methyl ether), which was reported to be present in maize pollen by Wierman (Ber. Dtsch. Bot. Ges. 81:3, 1968). 'Plant-tissue-type' flavonol glycosides are also present in pollen, but only in secondary amounts. In W22 A R-r Pr bz pollen there are no plant-tissue-type flavonol glycosides and only trace amounts of the pollen-specific type. The bz pollen has traces of flavonol aglycones and several fluorescent compounds which seem to be either 5- or 7-glycosides, or perhaps both, based on either quercetin or isorhamnetin. These fluorescent compounds are present in even higher concentrations in bz anthers and are almost certainly responsible for the fluorescence of bz anthers and pollen under UV light. Further characterization of these compounds is in progress.

E. Derek Styles and Oldriska Ceska

Pl effects on flavonoids in maize seedlings

In W22 strains capable of producing anthocyanins in seedlings and leaf sheaths, Pl at first represses, but then later enhances anthocyanin in these tissues (MNL 48:153, 1974; MNL 49:154, 1975). We are currently comparing extracts of W22 Pl and pl seedlings by thin layer chromatography in order to determine the effects of Pl on other flavonoids. Our initial findings indicate that, at the two-leaf stage (when Pl is repressing anthocyanin accumulation), Pl reduces the concentration of flavonols in approximately the same proportion as it does the anthocyanins. Flavone concentrations, on the other hand, appear to be increased by the presence of the Pl factor. This latter finding surprised us because flavone concentrations in the seedlings are usually a good indication of the level of P locus activity (MNL 50:107, 1976). In that W22 P-WR bz seedlings have traces of luteolinidin (a 3-deoxyanthocyanidin) glycosides, normally requiring P-RR if the seedlings are Bz, we thought it worthwhile to look at the effects of Pl on flavonoids in W22 bz seedlings also. So far we have found no 3-hydroxyanthocyanins in either Pl bz or pl bz seedlings, but flavonol glycosides are present in reduced amounts. Flavone concentrations again are higher in the Pl stocks, but the most noticeable effect of Pl is on the concentration of luteolinidin glycosides: whereas there are only trace amounts of these 3-deoxyanthocyanins in bz pl seedlings, there are measurable amounts in bz Pl seedlings. We should now be able to measure this effect of Pl quantitatively. We now intend to analyze P-WR bz stocks that have no anthocyanin-producing potential (e.g., r-g b) and also P-WW bz stocks that have no P locus activity. Hopefully we should then be able to tell if Pl is stimulating the production of luteolinidin via the 3-hydroxyanthocyanin pathway, or whether Pl can stimulate the production of flavonoids controlled by the P locus even when the normal anthocyanin pathway is blocked.

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Temperature sensitivity of virescent mutants

Several reports in the literature indicate a temperature component in the expression of virescent traits in maize. Phinney and Kay (Hilgardia 23:185, 1954) reported that the rate of greening for a virescent mutant, pale-yellow-1, was inversely related to temperature. In the most extensive study to date, Millerd and McWilliam (Plant Physiol. 43:1967, 1968) reported the inability of the mutant M11 to green at temperatures below 17 C; at 27 C, the M11 mutant greened as well as normal seedlings.

We have examined five virescent lines in order to define their temperature characteristics. The five lines are v, v3, v16 and v18, all in the Ohio 43 background, and v12 in an unknown inbred background. Greening responses were tested at six different temperatures ranging from 19 to 30 C. Greening was assayed by the in vivo technique previously described (Hopkins, Hayden and Walden, Can. J. Bot. 53:2720, 1975). In order to compensate for differences in the rate of seedling development at different temperatures, seedlings were assayed at the midpoint of the second leaf when that leaf was 5 to 7 cm in length.

With the exception of v, all mutants exhibited a threshold temperature below which greening did not occur (Table 1). Above the threshold, greening was a linear function of temperature. Greening of the wild-type was not influenced by temperature over the range tested. The extent of greening by v, v12 and v16 at 30 C was within 10 percent of the wild-type; greening of v18 at 30 C was only 25 percent of the wild-type and increased to only 50 percent at 35 C. The one mutant which did not appear to be markedly influenced by temperature was v. These seedlings exhibited considerable variability, greening to about 25 to 50 percent of the wild-type over the temperature range tested, but with no threshold.

Table 1. Threshold temperatures for greening of virescent mutants.

Genotype	Threshold Temperature (C)
v3/v3	23
v12/v12	20
v16/v16	25
v18/v18	22

Several temperature-shift experiments were conducted with v16. From these experiments we determined that at least three days at the lower temperature were required to produce a significant depression in chlorophyll content. When seedlings were transferred from 30 to 20 C, only those tissues which expanded at the lower temperature were bleached. There was no loss of chlorophyll in tissues which had already greened at the higher temperature. Following transfer from 20 C to 30 C, the newly expanded tissues greened but tissue which had been bleached at the lower temperature showed at best only a moderate tendency to green and then only in the region of the mid-vein.

The temperature characteristics described here correlate closely with our qualitative observations in the field. Those lines which show lower threshold temperatures generally green more rapidly in the field than do those with higher thresholds. The v18, which in laboratory trials never greened more than 50 percent of the wild-type, grows more slowly in the field and at maturity is of smaller stature than the wild-type or other virescent lines.

From these results we conclude that temperature sensitivity is a principal component in the expression of the virescent trait and that the response is allele specific.

W. G. Hopkins and D. B. Walden

III. CONVERGED STRAINS AND MULTIPLE COMBINATIONS

Cooperators have offered the following information regarding converged strains and multiple combinations:

Genetic materials	Background	Cooperator
TB's 4a, 7a, 9a, 9b, 10a	W22, M14, N25, Oh43	D. S. Robertson
White endosperm albinos	M14, W22	D. S. Robertson
Selected *wx* marked T9's	W22, M14, N25, Oh43	D. S. Robertson
Miscellaneous plant & endosperm factors	B8, NY511, NY821, Oh51A, Pa55	C. O. Grogan
Anthocyanin factors	K55, W23	E. H. Coe
Anthocyanin combinations	K55, W23 (*A C R*)	E. H. Coe
Dwarfs	K55, W23	E. H. Coe
9S markers	K55, W23	E. H. Coe
2S markers	K55 (*A C r-g Pl*)	E. H. Coe

IV. REPORT OF MAIZE COOPERATION

During 1976 the Maize Genetic Cooperation received 137 requests for maize genetic stocks. There were 100 (80%) domestic and 27 (20%) foreign requests. The number of seed requests in 1976 decreased 13% compared to 1975. Requests from geneticists were 46%, physiologists 19%, plant breeders 26% and educational 9% of the total requests received.

Certain chromosome tester stocks of chromosomes 4 and 5 were increased to supply future seed requests. In addition, the waxy alleles received from Dr. O. E. Nelson, Jr. were increased. Reciprocal translocation stocks of low viability were increased and attempts made to select cultures homozygous for the translocation where needed. Also, certain seedling lethals and all andromonoecious dwarfs were increased. Allele tests were made on a large amount of material to confirm what allele is present in the stock.

A list of reciprocal translocation stocks available from the Co-op is published in the Co-op News Letter report, Volume 43, 1969, or is available upon request.

Requests for seed or correspondence relative to the Stock Center should be addressed to:

Dr. R. J. Lambert
S-116 Turner Hall
Department of Agronomy
University of Illinois
Urbana, Illinois 61801

Catalogue of Stocks

Chromosome 1

sr zb4 P-WW
sr P-WR
sr P-WR an gs bm2
sr P-WR an bm2
sr P-RR gs bm2
sr P-WR bm2
vp5
zb4 ms17 P-WW
zb4 ms17 P-WW rs2
zb4 ts2 P-WW br f bm2
zb4 ts2 P-WW bm2
zb4 P-WW
zb4 P-WW br
zb4 P-WW br f bm2
zb4 P-WW bm2
ms17
ts2 P-RR
ts2 P-WW br bm2
ts2 P-WW bm2
ts2 br f bm2
P-CR
P-RR
P-RW
P-CW
P-MO
P-VV
P-RR as br f an gs bm2
P-RR br f an gs bm2
P-RR an ad bm2
P-RR an gs bm2
P-RR ad bm2
P-WR an Kn bm2
P-WR an ad bm2
P-WR an bm2
P-WR ad bm2
P-WR br Vg
P-WR br f gs bm2
P-WW rs2
P-WW rs2 br f
P-WW as br f bm2
P-WW hm br f
P-WW br f ad bm2
P-WW br f bm2
P-WW br f an gs bm2
P-WW br Vg
as
as rs2
rd-Hy
br f
br f Kn

Chromosome 1 (continued)

br f Kn Ts6
br f Kn bm2
br bm2
Vg
Vg an bm2
Vg br2 bm2
v22
bz2 m ; A A2 C Pr
bz2 M ; A A2 C R Pr
an bm2
an-bz2-6923 (apparent deficiency including _an_ and _bz2_)
br2
br2 bm2
tb-8963
Kn
Kn Ts6
Kn bm2
lw
vp8
gs bm2
Ts6
bm2
id
nec2
ms9
ms12
ms14
mi
D8
TB-1La (1L.20)
TB-1Sb (1S.05)

Chromosome 2

ws3 lg gl2 B
ws3 lg gl2 B sk
ws3 lg gl2 B sk v4
ws3 lg gl2 B sk fl v4
ws3 lg gl2 B ts
ws3 lg gl2 b
ws3 lg gl2 b fl v4
ws3 lg gl2 b sk fl v4
ws3 lg gl2 fl v4
ws3 lg gl2 b ts
ws3 lg gl2 b v4
al
al lg
al lg gl2 B sk v4
al lg gl2 b sk v4

Chromosome 2 (continued)

al lg gl2 b sk fl v4
lg
lg gl2 B
lg gl2 B gl11
lg gl2 B gs2
lg gl2 B gs2 v4
lg gl2 B gs2 Ch
lg gl2 B gs2 sk Ch
lg gl2 B sk v4
lg gl2 B v4
lg gl2 b
lg gl2 b gs2
lg gl2 b gs2 sk Ch
lg gl2 b gs2 v4
lg gl2 b gs2 v4 Ch
lg gl2 b sk
lg gl2 b sk fl v4
lg gl2 b sk v4
lg gl2 b wt v4
lg gl2 b fl v4
lg gl2 b fl v4 Ch
lg gl2 b v4
lg gl2 b v4 Ch
lg gl2 mn v4
lg gl2 wt
lg gl2 w3
lg gl2 w3 Ch
lg gl2 Ch
lg b gs2 v4
lg Ch
d5 = d*-037-9
B gl11
B ts
gl14
gl11
wt
mn
fl
fl v4 Ch
fl Ht v4
fl Ht v4 Ch
fl w3
fl w3 Ch
ts
v4
v4 w3 Ht
v4 Ht Ch
w3
w3 Ht
w3 Ch
Ht (A & B source)
ba2
R2 ; r A A2 C

Chromosome 2 (continued)

Ch
TB-3La-2S6270
Primary Trisomic 2

Chromosome 3

cr
cr d
cr d Lg3
cr pm ts4 lg2
cr ts4 na
d-Tall = d*-6016
d rt Lg3
d Rf lg2
d ys3
d ys3 Rg
d ys3 Rg lg2
d Lg3
d Lg3 ts4 lg2
d Rg ts4 lg2
d pm
d ts4 lg2
d ts4 lg2 a-m ; A2 C R Dt
ra2
ra2 Rg
ra2 ys3 Lg3 Rg
ra2 ys3 Rg
ra2 Rg lg2
ra2 pm lg2
ra2 lg2
Cg
cl
cl ; Clm-2
cl ; Clm-3
cl-p ; Clm-4
rt
ys3
ys3 Lg3
ys3 Lg3 gl6
ys3 gl6 lg2 a-m et ; A2 C R Dt
ys3 ts4
ys3 ts4 lg2
Lg3
Lg3 Rg
gl6 lg2 A ; A2 C R
gl6 lg2 A-b et ; A2 C R Dt
gl6 lg2 a-m et ; A2 C R dt
gl6 lg2 a-m et ; A2 C R Dt
ts4
ts4 na
ts4 ba na
ts4 lg2 a-m ; A2 C R Dt
ts4 lg2 gl7

Chromosome 3 (continued)

ts4 na a-m et ; A2 C R Dt
ts4 a-m ; A2 C R Dt
ba
y10
lg2 A-b et ; A2 C R Dt
lg2 a-m sh2 et ; A2 C R Dt
lg2 a-m et ; A2 C R dt
lg2 a-m et ; A2 C R Dt
lg2 a-st sh2 et ; A2 C R Dt
lg2 a-st et ; A2 C R Dt
na
A sh2 ; A2 C R B Pl dt
A-d31 ; A2 C R
A-d31 ; A2 C R pr dt
A-d31 ; A2 C R B Pl dt
A-d31 ; A2 C R Dt
A-d31 ; A2 C R pr Dt
A-d31 sh2 ; A2 C R B Pl dt
A-d31 sh2 ; A2 C R Dt
A-d31 sh2 ; A2 C R B Pl Dt
A-d31 et ; A2 C R Dt
a-m ; A2 C R B Pl dt
a-m ; A2 C R Dt
a-m ; A2 C R B Pl Dt
a-m sh2 ; A2 C R B Pl dt
a-m sh2 ; A2 C R B Pl Dt
a-m et ; A2 C R Dt
a-st ; A2 C R Dt
a-st sh2 ; A2 C R Dt
a-st sh2 et ; A2 C R Dt
a-st et ; A2 C R Dt
a-p sh2 et ; A2 C R B Pl Dt
a-p et ; A2 C R dt
a-p et ; A2 C R B Pl Dt
a-x1
a Ga7 ; A2 C R
sh2
vp
Rp3
pg14
a3
g5
yel*-5787
TB-3La (3L.10)
TB-3Sb (3S.50)
Primary Trisomic 3

Chromosome 4

Rp4
Ga
Ga su

Chromosome 4 (continued)

Ga-S
Ga-S bt2
st
st Ts5
st fl2
st Ts5 su
Ts5
Ts5 fl2
Ts5 su
Ts5 su zb6
Ts5 su zb6 o
Ts5 Tu
la
la su Tu gl3
la su gl3
la su gl3 c2 ; A A2 C R
la su gl3 o
fl2
fl2 su
fl2 bt2
fl2 su bm3
fl2 su gl4 Tu
su
su-am
su bt2 gl4
su bm3
su zb6
su zb6 Tu
su gl4
su gl4 Tu
su gl4 j2
su gl4 o
su gl4 o Tu
su j2
su gl3
su gl3 o
su o
bt2
bm3
gl4
gl4 o
Tu
Tu-l 1st
Tu-l 2nd
Tu-d
Tu-md
Tu gl3
j2
j2 c2 ; A A2 C R
j2 C2 ; A A2 C R
j2 gl3
v8

Chromosome 4 (continued)

gl3
gl3 o
gl3 dp
c2 ; A A2 C R
C2 ; A A2 C R
C2-Idf (Active-1) ; A A2 C R
o
v17
v23
gl7
ra3
Dt4 su ; a-m A2 C R
TB-4Sa (4S.20)
TB-1La-4L4692
Primary Trisomic 4

Chromosome 5

lu
lu sh4
ms13
gl17
gl17 A2 pr ; A C R
gl17 a2 ; A C R
gl17 a2 bt ; A C R
gl17 a2 bt v2 ; A C R
A2 vp7 pr ; A C R
A2 bm bt pr ys ; A C R
A2 bm pr ; A C R
A2 bm pr ys ; A C R
A2 bm pr ys eg ; A C R
A2 bm pr v2 ; A C R
A2 bt v3 pr ; A C R
A2 bt pr ; A C R
A2 bt pr ys ; in A C R
A2 v3 pr ; A C R
A2 pr ; A C R
A2 pr na2 ; A C R
A2 pr ys ; A C R
a2 ; A C R
a2 ; A C R B Pl
a2 bm bt bv pr ; A C R
a2 bm bt pr ; A C R
a2 bm bt pr ys ; A C R
a2 bm pr ys ; A C R
a2 bm pr v2 ; A C R
a2 bt v3 pr ; A C R
a2 bt pr ; A C R
a2 bt v2 ; A C R
a2 v3 pr ; A C R
a2 pr ; A C R
vp2

Chromosome 5 (continued)

vp2 pr
vp2 gl8
vp7
bm yg
bt
ms5
v3
td ae
ae
sh4
gl8
na2
lw2
ys
eg
v2
yg
ms13
v12
lw3 lw4
br3
TB-5La
TB-5Lb
Primary Trisomic 5

Chromosome 6

rgd po y
rgd po Y
rgd Y
po = ms6
po y pl
po y Pl
po Y pl
y = pb = w-m
y l10
y l11
y l12
y w15
y pb4
y pb4 pl
y pb4 Pl
y si
y wi Pl
y Dt2 ; a-m A2 C R
y pg11 ; Wx pg12
y pg11 wi ; wx pg12
Y pg11 ; Wx pg12
y pg11 ; wx pg12
Y pg11 ; wx pg12
y pl
y Pl

Chromosome 6 (continued)

y Pl Bh ; c sh wx A A2 R
y su2
Y 110
Y 112
Y pb4
Y wi pl
Y wi Pl
Y su2
wi
Pl Dt2 ; a-m A2 C R
pl sm ; P-RR
Pl sm ; P-RR
Pl sm py ; P-RR
Pt
w
w14
ms6
2NOR ; a2 bm pr v2
Primary Trisomic 6

Chromosome 7

Hs o2 v5 ra gl
In-D
In-D gl
o2
o2 v5
o2 v5 ra gl
o2 v5 ra gl sl
o2 v5 ra gl Tp
o2 v5 ra gl ij
o2 v5 gl
o2 v5 ms7
o2 ra gl ij
o2 ra gl sl
o2 gl
o2 gl sl
o2 bd
in ; A2 pr A C R
in gl ; A2 pr A C R
v5
vp9
vp9 gl
ra gl g2
ra gl ij bd
gl
gl-M
gl Tp
gl o5
gl g2
gl mn2
Tp

Chromosome 7 (continued)

ij
ij g2
ms7
ms7 gl Tp
Bn
bd
Pn
o5
g2
va
Dt3 ; a-m A2 C R
v*-8647
yel*-7748
TB-7Lb (7L.30)
Primary Trisomic 7

Chromosome 8

gl18
v16
v16 j
v16 ms8 j
v16 ms8 j nec
v16 ms8 j gl18
ms8
nec
v21
TB-8La (8L.70)
Primary Trisomic 8

Chromosome 9

yg2 C sh bz ; A A2 R
yg2 C sh bz wx ; A A2 R
yg2 C-I sh bz wx ; A A2 R
yg2 C sh bz wx K-L9 ; A A2 R
yg2 C bz wx ; A A2 R
yg2 c sh bz wx ; A A2 R
yg2 c sh wx ; A A2 R
yg2 c sh wx gl15 ; A A2 R
yg2 c sh wx gl15 K-L9 ; A A2 R-g
yg2 c bz wx ; A A2 R
wd-Ring C-I ; A A2 R
C sh bz ; A A2 R
C sh bz wx ; A A2 R
C-I sh bz wx ; A A2 R
C sh bz wx gl15 bm4 ; A A2 R
C sh ; A A2 R
C sh wx ; A A2 R
C wx ar ; A A2 R
C-I sh wx v ; A A2 R
C sh wx K-L9 ; A A2 R

Chromosome 9 (continued)

C sh ms2 ; A A2 R
C bz Wx ; A A2 R
C Ds Wx ; A A2 R y
C Ds wx ; A A2 R pr
C-I Ds wx ; A A2 R
C-I ; A A2 R
C ; A A2 R
C ; A A2 R B Pl
C wx ; A A2 R
C wx ; A A2 R B Pl
C wx ; A A2 R b Pl
C wx ; A A2 R B pl
C-I wx ; A A2 R y
C-I wx ; A A2 R y B pl
C wx ar da ; A A2 R
C wx v ; A A2 R
C wx v ; A A2 R Pl
C wx gl15 ; A A2 R
C wx gl15 ; A A2 R pr
C wx Bf ; A A2 R
c sh bz wx ; A A2 R y
c sh wx ; A A2 R
c sh wx v ; A A2 R
c sh wx gl15 ; A A2 R
c sh wx gl15 bk2 ; A A2 R
c sh wx gl15 Bf ; A A2 R
c sh wx bk2 ; A A2 R
c ; A A2 R
c wx ; A A2 R y
c wx v ; A A2 R
c wx gl15 ; A A2 R
c wx Bf ; A A2 R
c wx bk2 ; A A2 R
sh
sh bp wx ; P-RR
sh bp wx ; P-RW
sh wx v
sh wx d3
sh wx pgl2 gl15 ; y pgl1
bp wx ; P-RR
bp wx ; P-RW
bp wx ; P-WW
lo2
wx
wx-a
wl1
wx d3
wx d3 wl1
wx d3 v gl15
wx d3 gl15
Wx pgl2 ; y pgl1
wx pgl2 ; y pgl1

Chromosome 9 (continued)

Wx pgl2 ; Y pgl1
wx pgl2 ; Y pgl1
wx pgl2 bm4 ; y pgl1
wx v
wx bk2
wx bk2 bm4
wx Bf
wx Bf bm4
d3
v
gl15
gl15 Bf
gl15 bm4
bk2 Wc
Wc
bm4
bm4 Bf
l6
l7
yel*-034-16
w*-4889
w*-8889
w*-8951
w*-8950
w*-9000
TB-9La (9L.40)
TB-9Sb (9S.40)
Primary Trisomic 9

Chromosome 10

oy
oy R ; A A2 C
oy bf2
oy ms11
oy bf2 R ; A A2 C
oy bf2 ms10
oy zn R ; A A2 C
oy du R ; A A2 C R
oy du r ; A A2 C
oy sr2
oy zn
Og
Og du R ; A A2 C
ms11
ms11 bf2
bf2
bf2 zn
bf2 li g r ; A A2 C
bf2 g R sr2 ; A A2 C
bf2 g r sr2 ; A A2 C
bf2 r sr2 ; A A2 C

Chromosome 10 (continued)

nl zn g R ; A A2 C
nl g R ; A A2 C
nl g r ; A A2 C
nl g R sr2 ; A A2 C
y9
li zn g r ; A A2 C
li g R ; A A2 C
li g r ; A A2 C
li g r v18 ; A A2 C
li g R v18 ; A A2 C
ms10
du
du v18
du o7
du g r ; A A2 C
du sr2
zn
zn g
zn g R sr2 ; A A2 C
zn g r ; A A2 C
Tp2 g r ; A A2 C
g R sr2 ; A A2 C
g r ; A A2 C
g r sr2 ; A A2 C
g r sr2 l ; A A2 C
g R-g sr2 ; A A2 C
g R-g sr2 v18 ; A A2 C
g R-g K10 ; A A2 C
g R-g sr2 ; A A2 C
g R-r K10 ; A A2 C
g r-r sr2 ; A A2 C
Ej r-r ; A A2 C
Ej r-r sr2 ; A A2 C
r sr2 l ; A A2 C
R-g ; A A2 C
r-g sr2 ; A A2 C
r K10 ; A A2 C
r-g ; A A2 C
r-r ; A A2 C
R-mb ; A A2 C
R-nj ; A A2 C
R-r ; A A2 C
R-r(Boone) ; A A2 C
R-lsk ; A A2 C
R-sk-mc.2 ; A A2 C
R-sk ; A A2 C
R-st ; A A2 C
R-st Mst
R-st Mst o7
Lc
w2
w2 l

Chromosome 10 (continued)

o7
o7 ; o2
l
v18
Mst
l yel*-5344
yel*-8721
yel*-8454
yel*-8793
TB-10La (10L.35)
TB-10Sc
Primary Trisomic 10

Unplaced Genes

dv
dy
el
h
l4
Rs
v13
ws ws2
ub
zb
zb2
zb3
zn2
l*-4923
nec*-8376

Multiple Gene Stocks

A A2 C R-g Pr B Pl
A A2 C R-g Pr B pl
A A2 C r-g Pr B Pl
A A2 C r-g Pr B pl
A A2 c R-g Pr B pl
A A2 C R-r Pr B Pl
A A2 C R-r Pr B pl
A A2 C R-r Pr b Pl
A A2 c R-r Pr B Pl
A A2 C r-r Pr B Pl
A A2 c r-r Pr B Pl
A A2 C R Pr
A A2 C R Pr wx
A A2 C R Pr wx gl
A A2 C R Pr wx y
A A2 C R pr
A A2 C R pr y gl
A A2 C R pr y wx
A A2 C R pr y wx gl

Multiple Gene Stocks (continued)

A A2 c R Pr y wx
A A2 C r Pr y wx
a su A2 C R
bm2 lg a su pr y gl j wx g
colored scutellum
lg gl2 wt ; a Dt A2 C R
lg su bm2 y gl j
su y wx a A2 C R-g pr
y wx gl
hm hm2
ts2 ; sk

Popcorns

Amber Pearl
Argentine
Black Beauty
Hulless
Ladyfinger
Ohio Yellow
Red South American
Strawberry
Supergold
Tom Thumb
White Rice

Exotics and Varieties

Black Mexican Sweet Corn
(with B-chromosomes)
Black Mexican Sweet Corn
(without B-chromosomes)
Knobless Tama Flint
Knobless Wilbur's Flint
Gaspe Flint
Gourdseed
Maiz chapolote
Papago Flour Corn
Parker's Flint
Tama Flint
Zapaluta chica

Tetraploid Stocks

P-RR
P-VV
Ch
B Pl
a A2 C R Dt
su
pr ; A A2 C R
y

Tetraploid Stocks (continued)

gl
ij
Y sh wx
sh bz wx
wx
g A A2 C R
A A2 C R B Pl

Cytoplasmic Steriles and Restorers

WF9 - (T)	rf rf2
N6 (S)	
WF9	rf rf2
N6	rf Rf2
R213	Rf rf2
Ky21	Rf Rf2

Waxy Reciprocal Translocations

wx1-9c (1S.48;9L.22)
wx1-9-4995 (1L.19;9S.20)
wx1-9-8389 (1L.74;9L.13)
wx2-9b (2S.18;9L.22)
wx3-9c (3L.09;9L.12)
wx4-9b (4L.90;9L.29)
wx4-9-5657 (4L.33;9S.25)
wx4-9g (4S.27;9L.27)
wx5-9a (5L.69;9S.17)
wx5-9c (5S.07;9L.10)
wx6-9a (6S.79;9L.40)
wxy6-9b (6L.10;9S.37)
wx7-9a (7L.63;9S.07)
wx7-9-4363 (7 cent.;9 cent.)
wx8-9d (8L.09;9S.16)
wx8-9-6673 (8L.35;9S.31)
wx9-10b (9S.13;10S.40)

Inversions

gl2 Inv2a (2S.70; 2L.80)
wx Inv9a (9S.70; 9L.90)

V. RECENT MAIZE PUBLICATIONS

Al-Azzawi, M. J., and J. L. Hall, 1976. Cytochemical localization of adenylate cyclase activity in maize root tips. Plant Sci. Lett. 6:285-289.

Alekhina, N. D., and E. D. Shirshova, 1976. Isozyme composition of aspartate- and alanine aminotranspherases from roots of maize cultivated at lowered temperature. Dokl. Akad. Nauk SSSR 228:248-251.

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ERRATA

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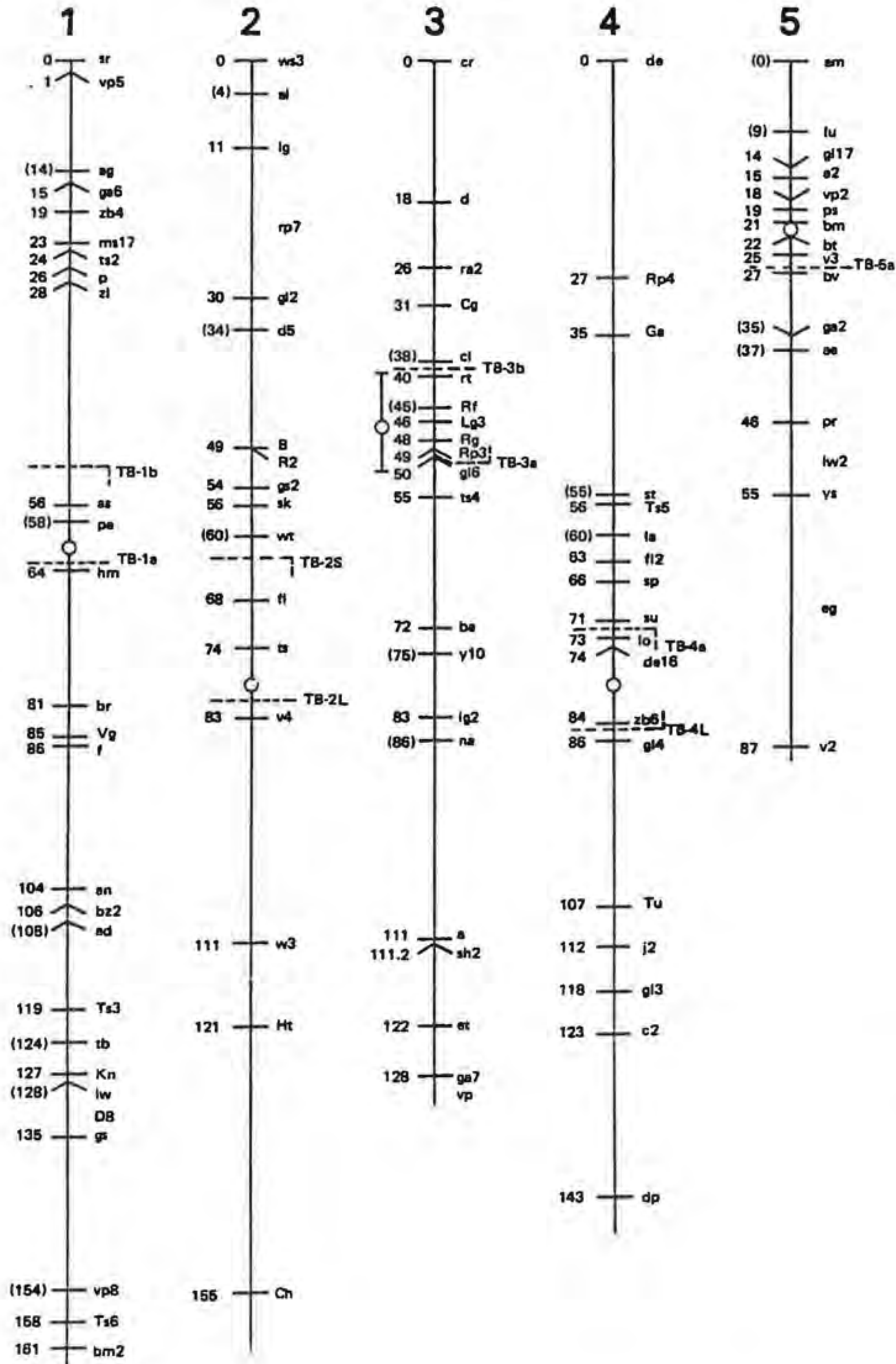
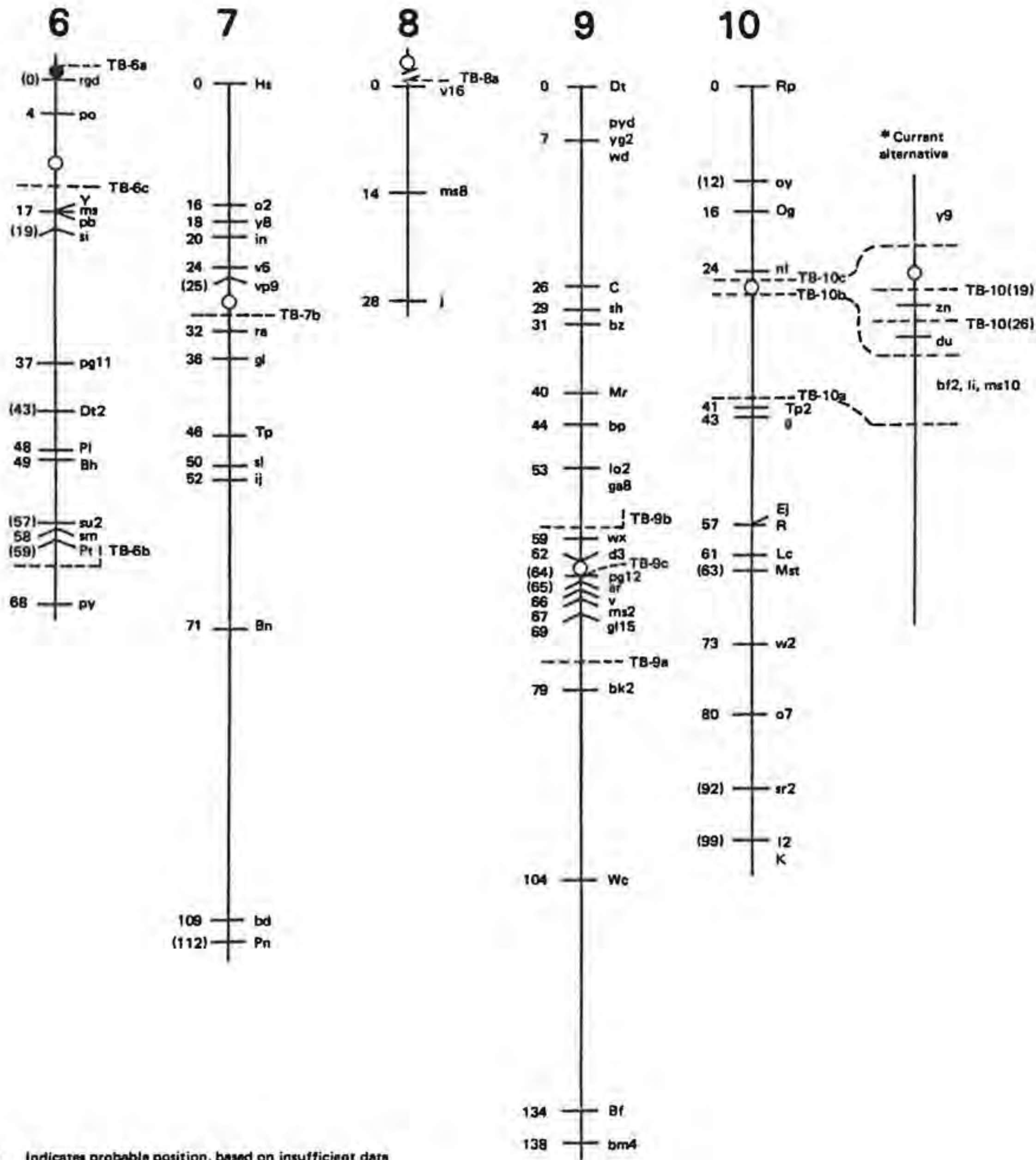
50:3	15	1 Apr 41	etc.		
	18	31 Jan 44	etc.		
	26	17 Mar 52	etc.		
	30	15 Mar 56	M. M. Rhoades, Illinois	164	Minutes of meeting regarding cooperation; reports; stocks; publications.

50:20 The correct pedigree for US13 is (WF9 x 38-11)(HY x L317).

LINKAGE MAP OF MAIZE

April 14, 1976





( ) Indicates probable position, based on insufficient data

O Indicates centromere position

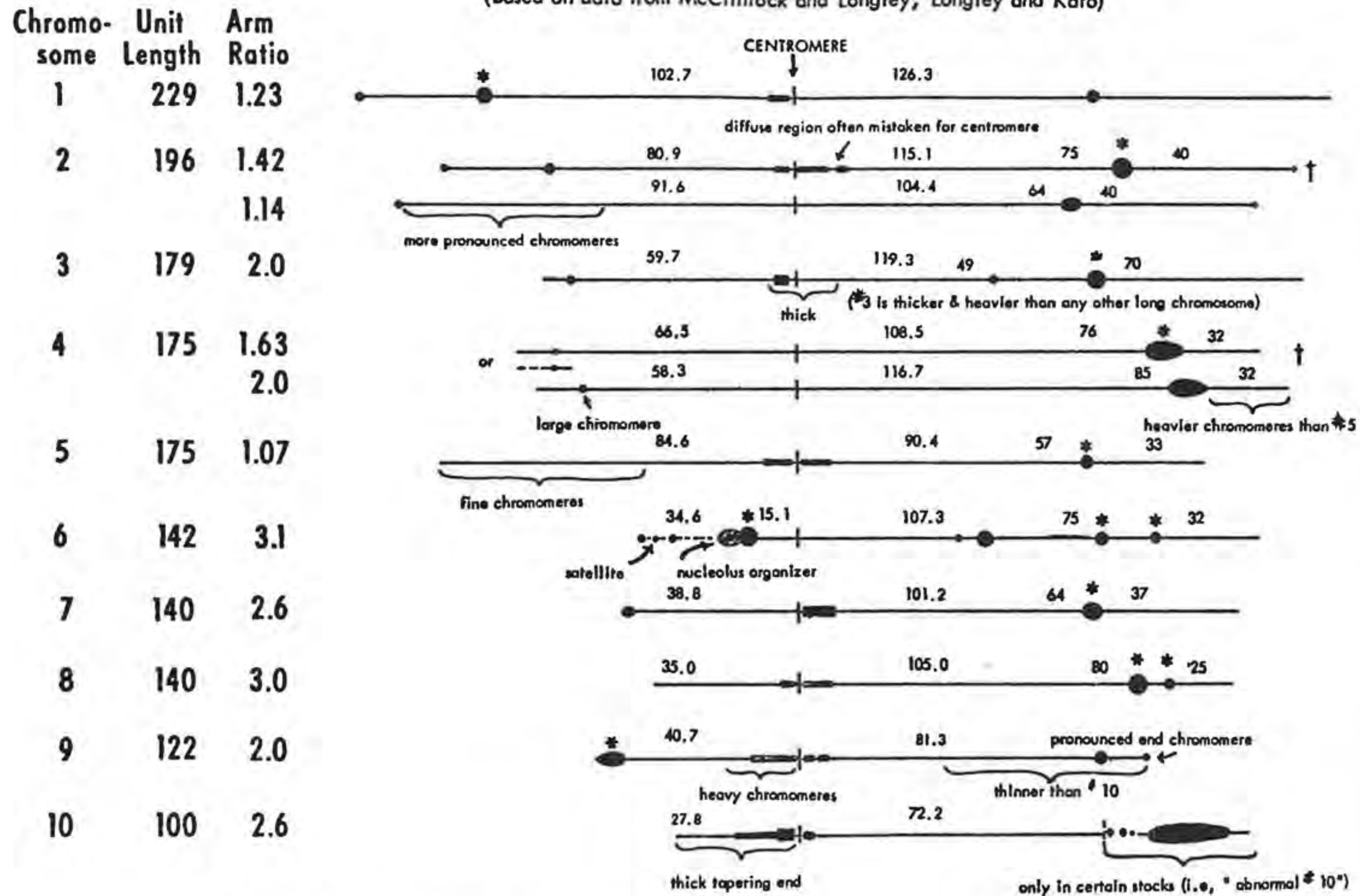
● Indicates organizer (NOR)

TB Indicates translocation involving A and B chromosomes, with A break point at broken line or in direction indicated. TB-2S, TB-2L and TB-4L are short designations for TB-2S,3L(6270), TB-2L,1S(4464) and TB-4L,9S(6222) from Rakha and Robertson (1970). TB-10(19) and TB-10(26) are as designated by Lin (1974).

* Lin (1974); Beckett (Personal communication)

Revised from Neuffer, Jones, and Zuber (1968) The Mutants of Maize Crop Science Society of America. Madison, Wisconsin.

Cytological Map of Maize Chromosomes
drawn to scale with major distinguishable characteristics
(based on data from McClintock and Longley, Longley and Kato)



Chromo-some	Unit Length	Arm Ratio
1	229	1.23
2	196	1.42
		1.14
3	179	2.0
4	175	1.63
		2.0
5	175	1.07
6	142	3.1
7	140	2.6
8	140	3.0
9	122	2.0
10	100	2.6

*Knobs found in more than 50% of races
† More common form of chromosomes 2 & 4
Smallest dots represent prominent chromomeres

This is not a journal of published research, but rather an informal news letter by which research workers exchange information about the genetics and cytogenetics of maize. The less formality and the more of techniques, data and observations, the greater the value. Communications are received and edited with that purpose; contributed items may be returned to the contributor for reconsideration of whether their content has impact in inheritance in maize and in genetics as a discipline, or of whether their degree of formality propels them to the journals.

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Figures can be reproduced from line drawings on plain paper; tables and figures may be reduced in size for reproduction.
Symbols must be consistent with current conventions (MNL 49:3-4).
References should be used sparingly; when needed, they should be identified in abbreviated form in the text, parenthetically.
Deadline for contributions for the next issue (volume 52, 1978) is January 1, 1978.

The editor will assume that contributed items require no editing; any that the editor is unable to repair with alacrity may be reproduced verbatim.