MAIZE GENETICS COOPERATION

NEWS LETTER

53

March 1, 1979

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

This is an informal news letter by which working research information on the genetics and cytogenetics of maize is shared. Communications are received and assembled with minimum editing. Specific data, methods and observations are appropriate.

Contributed items should be double-spaced in the text.
Tables and Figures will be reproduced directly by reduction whenever possible; they should be clear, compact, and ready for the camera.
References should be used sparingly; when needed, they should be identified in abbreviated form in the text (parenthetically), including authors' initials to facilitate indexing.
Deadline for contributions for the next issue (number 54, 1980) is January 1, 1980.

Some sources of general information on maize genetics and cytogenetics:

Emerson, R. A., G. W. Beadle and A. C. Fraser, 1935. A summary of linkage studies in maize. Cornell Univ. Agric. Exp. Sta. Memoir 180.

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.

Evolution of Crop Plants, Chap. 37, pp. 128-136. N. W. Simmonds, ed., Longman, N.Y., 1976.

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

Corn and Corn Improvement, 2d edition, G. F. Sprague, ed., Amer. Soc. Agron., 1977.

Maize Breeding and Genetics. D. B. Walden, ed., Wiley, N.Y., 1978.

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I. FOREWORD

The news items have been arranged alphabetically in this issue, in hopes this order will be helpful in your locating and reading; it is the arrangement used in early issues of the News Letter.

A much larger task than assembling the notes this year was the preparation of an index to the genetic symbols and cytogenetic features, for number 36 (1962) through the present issue. Hopefully the use of this aid will release more of our available information for development of refined cytogenetic maps, and will sharpen our endeavors generally. Included among items in this issue are new and considerable additions to this body of information; further data and further map clarifications are urgently needed.

For symbols, reinstatement of the numeral one for the first locus of a series appears to be widely desired. While it has not been followed consistently this year in editing notes, the numeral clarifies meanings and would be helpful to readers as future notes are written. The Symbol Index follows the practice.

Historical notes: 50 years ago (April 12, 1929) compilations of linkage data and "rainbow" maps were distributed from Cornell; the compilations are reproduced in this issue from the copy of E. G. Anderson. Archival deposits of others of E. G. Anderson's research papers, correspondence and record books (including attached-X data) have been made in the Western Historical Manuscripts Collection (Ellis Library, University of Missouri, Columbia, MO 65211). The papers of L. J. Stadler are also on deposit in this Collection.

A pleasing announcement of the Rosenstiel Award to Barbara McClintock, forwarded by H. O. Halvorson, is shared on the following page.

Publications of interest: Maize Breeding and Genetics, D. B. Walden, ed., has been published by John Wiley & Sons, N.Y. (1978); this is the collection of papers from the 1975 Symposium in Urbana, Illinois.

About 700 copies of this issue will be sent to research workers, laboratories and libraries around the world. The costs of preparation, reproduction and mailing of this News Letter are borne by a grant from the National Science Foundation, giving us indispensable support for which we are all grateful. The year-round office workload for the News Letter is provided for by the U. S. Department of Agriculture; facilities are provided by the University of Missouri.

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

Airmail service to addresses outside the U.S. will be provided for $3.00 if received by January 1st.

Deadline for the next issue is January 1, 1980; see inside front cover.

I appreciate the encouragement and support of M. G. Neuffer, J. B. Beckett and G. G. Doyle in planning and developing this volume; Charlene Bennett, Kim Berry, Pat Berry, Ken Bristol, Chris Browne, Marion Murray and Manh Nguyen aided in editing and proofing of copy and in bibliographic work; Mary Nelson once again applied her thorough, precise and diligent attention to composition and the production of final copy.

E. H. Coe, Jr., Geneticist, USDA; Professor of Agronomy
Curtis Hall, University of Missouri, Columbia, Missouri 65211

II. BRANDEIS SCIENCE AWARD

Waltham, Mass.---Dr. Barbara McClintock, a world renowned pioneer in genetics, has been named recipient of Brandeis University's seventh annual Rosentiel Award for excellence in scientific research. The selection was announced by an awards committee of distinguished Boston area scientists. The committee praised Dr. McClintock for the "imaginative and important contributions" she has made to the world of science. While she has long been recognized as an influential geneticist, the committee added, Dr. McClintock "has never received the formal recognition and honor due such a remarkable scientist." Presentation of the Rosenstiel Award medallion and its $5,000 prize were made to Dr. McClintock at a dinner April 13, 1978, at Brandeis.

Prof. H. O. Halvorson, director of the Rosenstiel Center, characterized her as "a forerunner in the field of genetics." And her work now is being interpreted at the molecular level--"trying to understand the DNA at the level of individual molecules." "She was one of the few biologists who took part in the early and truly exciting period of genetic studies," he said. "Using maize, she began to understand the mechanics of chromosome separation and segregation. She showed that you could get breaks in chromosomes and that chromosome pieces would re-assort themselves. If you were to separate a piece of chromosome number one, that piece might attach itself to, say, chromosome number three. The attachment might occur in a logical, correct direction, or it might occur in a backwards fashion--head to tail. And sometimes, the chromosome piece would be turned around and be put back on its original source. This is called inversion."

Dr. McClintock demonstrated that when pieces of chromosomes were moved, it affected the way in which genetic inheritance developed. She also realized that during such rearrangement, chromosomes acquired changing control properties and that they are regulated differently by virtue of their attachment to a new chromosome.

Now, much of her work is being interpreted at the molecular level. "Since we know that genes on chromosomes move around, and that there are rules that govern this movement, science is just beginning to understand how a gene in one place ends up on another chromosome, and how it acquires different kinds of properties of expression." Dr. McClintock first observed this movement and the associated control properties. Today, scientists who are "looking at very fine point genetics" are beginning to rediscover the ability of genes to move around. Science is only beginning to grasp the biochemistry of this, but Dr. McClintock's definition is "now being reinvestigated as the molecular biologist examines more complex problems."

Dr. McClintock, a native of Hartford, Conn., has been associated with the Genetics Research Unit at Cold Spring Harbor since 1941. In 1967, she was named a Distinguished Service Member of that organization. She studied at Cornell University for her bachelor's, master's and Ph.D. degrees, the last awarded in 1927. From 1965 to 1975, she was the Andrew White Professor at Cornell. She holds numerous awards and honorary degrees, and is widely published in many scientific journals. In 1970, she was awarded the National Medal of Science, and earlier won the Kimber Genetics Award of the National Academy of Sciences, among other honors. Besides Cornell, she has taught at the California Institute of Technology and the University of Missouri. She is also a member of several distinguished societies, including the American Academy of Arts and Sciences.

The Rosenstiel Award program was founded in 1971. It honors the late Lewis S. Rosenstiel of Miami Beach, who was responsible for creation of the Rosenstiel Center at Brandeis. The annual prizes are given to scientists as a means "to identify important work in basic medical research." Since its inception, the program has earned a wide reputation for acknowledging significant scientific developments.

III. REPORTS FROM COOPERATORS

AMES, IOWA
Department of Agronomy, Iowa State University

Enhancement of genetic exchange in maize: Intragenic recombination

Intragenic recombination is enhanced by two metal complexing agents, EDTA (Ethylene diaminetetra acetic acid) and kryptofix 222 (E. Merck Laboratory). The assay system included heteroallelic combinations of three wx alleles, wx-B, wx-C, and wx-90 on normal and homozygous interchanged chromosomes.

Spray treatment with solutions of these two chemicals resulted in a higher frequency (significant at the .05 level) of recombination (as judged from the occurrence of Wx pollen grains from wx-a/wx-b heteroalleles). Sample data comparing the effects of treatments (water, control) EDTA and kryptofix on Wx frequencies per 10^5 in various wx heteroallelic combinations:

Genotype	Control	EDTA	Kryp
N-B/C	42.34	55.62	48.52
N-C/90	106.88	115.17	114.11
T5C/90	66.11	72.45	74.44
Mean	71.78	81.08*	79.02*

*Significant at 0.05 level.

There were two chromosome arrangements for these heteroalleles: the normal and the chromosome interchange whereby the wx locus was moved a further distance away from the centromere. Greater distances away from the centromere resulted in higher frequencies of intragenic recombination but less than those of the standard chromosome even though in some of the interchange chromosomes, the centromere-wx distance was greater.

Kitsiri Sukhapinda and Peter A. Peterson

Activities associated with a controlling element allele

In the En controlling element system, two readily observable functions can be ascribed to the En transposon. These include both suppressor (S) and mutator (M) functions. The assorted activities and changes associated with En can be tested by the use of an unstable "A" state, a-m(Au), phenotypically indistinguishable from standard A except for large colorless areas (A to a) in the aleurone layer. Two observations indicate that a potentially active En is associated with a-m(Au): (1) fully active En elements containing S and M are coincident isolates with changes of a-m(Au) to a-m(nr) (colorless forms) and (2) a-m(Au) responds to an independently segregating En.

By combining a-m(Au) with various colored (pale to dark) responding alleles such as a-m-1 changes that occur in the change from a-m(Au) to a-m(nr) (the colorless areas of the "A" state) can be monitored. These observations show that the En at the a-m(Au) allele expresses sm, Sm, sM and SM phenotypes (s, m indicating lack of suppressor and mutator functions, respectively). Further, the changes are not permanent since sm to SM to sm and sm to sM to sm sequences are observed.

Peter A. Peterson

Controlling element components function in a tissue culture system

The S and M functions of En (as described in the previous note) have been demonstrated to be active in cultured endosperm tissue. Using controlled receptive alleles, wx-m-8, c-m(r) and a-m-2 in combination with the regulatory element, En, changes to Wx, to C and to A are triggered in cultured callus in a manner similar to the expression observed in kernels. With a-m-2, both functions of En, M and S are observed to function. The callus phenotypes are anthocyanin (for C and A) and amylose-staining starch (for Wx).

Mark B. Gorman and Peter A. Peterson

AMES, IOWA
Department of Genetics, Iowa State University

Further evidence on the timing of Mu activity

Evidence to date (see MGCNL 52:46-47, 1978) suggests that most, but not all, Mu induced mutations are probably meiotic in origin. The occurrence of occasional "clusters" of plants segregating for mutants of similar phenotype in outcross of Mu bearing plants as males suggested the existence of pre-meiotic mutants. Allelic tests of mutants from such putative "clusters" established that most were not allelic and hence were not derived from a premeiotic sector. However, 22 percent of the putative "clusters" did indeed possess allelic mutants and therefore probably were derived from a mutation producing a premeiotic sector.

If sectors are occurring, it should be possible to demonstrate them by planting ear maps of Mu plants outcrossed as females. Last year (MGCNL 52:47-49, 1978) I reported the results of three such ear maps. Two of them had putative "clusters" of mutants. This past summer mutants from seven of the eight possible clusters were tested to determine if the mutants in the putative sectors were allelic. Four pale yellow mutants found in one five plant sector of one ear all proved to be allelic to each other as did the mutants from a two pale-yellow and a white mutant sector of the second ear. Mutants from a putative two plant yellow-green sector proved to be non-allelic as did a yellow-green and luteus mutant in another two plant putative sector. Sufficient data were not obtained on a possible three plant luteus sector to give a clear cut answer as to whether or not the mutants were allelic. Test plants from two putative two-plant pale green sectors did not appear to segregate for pale green seedlings. These may be temperature sensitive mutants which were not expressed in the warm greenhouse conditions this fall. This material will be retested under cooler growing conditions.

The ear map results confirm those from the allele tests of male derived mutants reported last year. Mu can induce premeiotic mutations which result in "clusters" of allelic mutants. Although the sample is small, it would appear that the mutations responsible for these sectors are extremely late since the sectors only seem to involve a few ovules. The largest sector demonstrated, to date, consists of 5 seeds. This is consistent with the tests for somatic sectoring in Mu bearing plants and seeds heterozygous for yg2, c, sh, bz, wx, a sh2, a2, and bt. The somatic sectoring rate for all of these loci was no higher in Mu plants than the non-Mu controls. If Mu affects the normal alleles of these loci, it does not do it early enough in ontogeny to produce observable somatic sectors.

Since it appears that the premeiotically induced mutations occur quite late it could be that some single mutations which are normally classified as meiotic in origin could in reality be due to a very late premeiotic mutation in which only one

cell derived from the one in which the mutation occurred gave rise to an ovule. Thus it is impossible at this time to say what fraction of Mu induced mutations are meiotic or premeiotic.

Donald S. Robertson

The relationship of Mu to the Fcu controlling element system

Because approximately 30% of the Mu induced mutants have mutable phenotypes, it is possible that Mu is a very active and mobile controlling element system. If this is the case Mu may be related to one of the previously described controlling element systems. Crosses of Mu stocks with lines possessing only the receptor elements a, Ds and I proved that Mu did not have Dt, Ac or En activity. In 1977 we reported (MGCNL 51:37, 1977) that Ac Ds, En I and a Dt systems did not possess a mutation rate that differed from those of control populations studied in our Mu test.

Gonella and Peterson (Genetics 84:629-645, 1977) reported on a new controlling element system Fcu r-cu. Purple aleurone Mu lines were crossed to an r-cu (non-Fcu) stock and the F1 progeny self-pollinated and scored for Fcu activity. No Fcu activity was observed.

To test the mutator activity of the Fcu r-cu system, plants with both elements were self-pollinated and outcrossed to a standard line. The outcross seeds were planted and the resulting plants self-pollinated and the self-progeny were scored for the occurrence of new mutants (Table 1). Three mutations were observed out of a population of 330. This was more than those observed for the Dt, En and Mp systems combined (see MGCNL 51:37, 1977). The occurrence of a mutable yellow-green mutant is suggestive that these mutants could have been the result of the controlling element system. However, if the Fcu r-cu system is producing mutation it does so at a very low rate which does not differ significantly from the rate found in control populations. Certainly it does not approach the mutator activity of Mu.

Table 1. Tests for mutability in the Fcu r-cu controlling element system.

Outcross family	No. of plants ⊗	No. of ears in which mutants segregated (Mutant phenotype)	% Mutants
3024-9036	73	0	--
3025-9037	81	1 (luteus)	1.2
3026-9038	61	1 (yellow-green)	1.6
3027-9039	62	1 (yellow-green)*	1.6
3028-9040	53	0	--
Total	330	3	0.9
Typical control population	1,215	3	0.2
Previous Mu tests	1,541	98	6.4

Comparison of Fcu r-cu and control population reveal a contingency

$x^2 = 2.5381$ $p = .10\text{-}.20$ $p.05 = x^2 = 3.841$

Comparison of Fcu r-cu and previous Mu tests reveals a contingency

$x^2 = 22.2771$, $p.01 = x^2 = 6.635$

*mutable

In summary, to date, there is no evidence that Mu is related to any of the known controlling element systems. Also, to date, it does not appear that any of the controlling element systems approach Mu in their mutator activity.

Donald S. Robertson

ATHENS, GEORGIA
Department of Agronomy, University of Georgia

Effect of cytoplasm on various maize characters

In six paired comparisons in a double-cross maize hybrid, statistically significant differences between cytoplasms were found for the following characters: plant height, stalk diameter, number of leaves per plant, susceptibility to borers, Ostrinia nubilalis (Hubner) and Heliothis zea (Boddie), yield, stalk lodging, number of ears, grain quality, ear length, germination, and premature plant lodging. These characters represented over 73% of the characters studied.

J. Keith Contarino and A. A. Fleming

Methods of seed preparation for cytoplasmic studies

Two methods of seed preparation were compared in the production of a double-cross hybrid. One method consisted of preparing the seed by making individual-plant reciprocal crosses. The other method consisted of crossing reciprocally several plants from the female row to the respective single plant (of the male row) that was used in the individual-plant reciprocal cross.

The second method was designated as the bulked source since seeds from several female plants were bulked. Single ears from the individual-plant method were kept separately.

There were 72 pairs of plots in the experiment (equivalent to 72 replications). The statistical analyses were performed to maximize the chance of detecting differences between the two methods. However, only plant height and yield of the 15 characters studied exhibited significant differences for the two methods of seed preparation. These differences were small. Thus, there is evidence that differences attributed to cytoplasmic effects on agronomic characters in previous studies were not due to method of seed preparation.

J. Keith Contarino and A. A. Fleming

The cytoplasm and nutrient absorption

While working with maize seedlings grown in a modified Hoagland's solution for 20 days, we found significant cytoplasmic differences in the absorption of NO_3^-, NH_4^+, K, and dry-weight production during the 0-10 and 11-20 day growing periods. Significant differences in the absorption of P and Mg were observed only during the 0-10 day period.

No significant cytoplasmic effects were observed in the absorption of either Ca or micronutrients Fe, Zn, and Mn. Plants with GA 199 cytoplasm had 13% more root dry-weight and 18% more shoot dry-weight than plants with GT 112 cytoplasm. These results show that the cytoplasm can influence a plant's nutrition.

W. S. McElhannon and A. A. Fleming

Controlling weeds in maize nursery plots

Before 1978 we had to do a great amount of work in controlling annual weeds in our maize nurseries. Among the weeds at harvest would be sicklepod (Cassia obtusifolia), morning glory (Ipomoea purpurea), crabgrass (Digitaria sanguinalis L.), and fall panicum (Panicum dichotomiflorum) although the preemerge herbicides, AAtrex and Princep, were applied. Good weed control usually would occur until the soil was cultivated at time of applying ammonium nitrate as a sidedressing. After the cultivation a new crop of weeds would develop.

In 1978 we obtained excellent weed control by splitting the recommended rate per ha of AAtrex and Princep into two applications (one-half the recommended rate

per application). The first application was made at preemergence or soon thereafter. The second application was made immediately after the sidedressing cultivation.

A. A. Fleming

AUSTIN, TEXAS
Department of Zoology, University of Texas

A search for parthenogenetic maize

There is reason to predict that some kinds of crossover deficient meiotic mutants might lead to regular production of unreduced eggs. Mutations which block crossing over, in the presence of normal homologue pairing and synaptonemal complex formation, might have such an effect (see Chromosoma 65:173-183, 1978 and Exp. Cell Res. 112:297-308, 1978). Homozygotes for such mutations, in the presence of the additional features of capacity for diploid embryo and diploid endosperm development, would be expected to produce viable seeds without fertilization. The probability of natural occurrence of the fortuitous combination of events required is of course vanishingly small, but self-pollinated mutagen treated material is a somewhat more promising potential source. This leads to the suggestion that investigators who grow large mutagen treated progenies, and bag numerous earshoots, which are left bagged but unpollinated, may have a rare opportunity to find a parthenogenetic stock source, simply by checking for development of ears from unpollinated (covered) earshoots. This can probably be done effectively by feeling, without picking or husking. It is the purpose of this note, not only to suggest that such a search be made for a parthenogenetic stock source, but also that I would be very interested in studying its meiosis.

Marjorie Maguire

Direct cytological evidence for true terminalization of chiasmata

Recent reports in the literature which suggest that chiasmata do not terminalize appreciably between early diplotene and metaphase I in several organisms have raised the question whether the true positions of chiasmata actually change with stage advancement, or only appear to shift toward the distal ends of bivalent arms (because of strong prophase condensation) in maize. The positions of knob-bearing regions relative to chiasmata at diakinesis in material heterozygous for knobs provide relevant information.

Microsporocytes from a plant heterozygous for six or seven knobs, and homozygous for one were studied in systematically scanned acetocarmine squash preparations. Bivalents with equational separation of a heterozygous knob in the presence of a distal chiasma were frequently observed. This is taken as evidence that in such cases a crossover has occurred proximally to the knob, and the resulting chiasma has been terminalized through the knob. Equational separation of a heterozygous knob-bearing region was not found in the absence of a distal chiasma. Disjunctional separation of heterozygous knobs in diakinesis bivalents was also observed. This is interpreted in several ways, depending on the absence or presence (as well as position) of a chiasma: either no crossover has occurred in the arm in question (in the absence of a chiasma there), or crossing over occurred distally to the knob (in the presence of a distal chiasma), or crossing over occurred proximally to the knob and has not yet terminalized through it (in the presence of a proximal chiasma).

The plant studied was heterozygous for a reciprocal translocation. Pachytene translocation configurations indicated the presence of heterozygosity for a conspicuous knob, and diakinesis quadrivalent translocation configurations gave particularly clear demonstrations of equational separation of the knob-bearing regions in the presence of a distal chiasma.

Marjorie Maguire

Probable presynaptic alignment of homologues

When microsporocytes at synizesis in conventional acetocarmine squash preparations are observed with the high resolution light microscope optics available with the Zeiss 1.4 N.A. 63 x oil immersion bright field objective, it is possible to trace individual homologues and paired homologues with substantially greater clarity than is possible with the 1.3 N.A. bright field planapochromat. With the superior optical system, a sub-stage of synizesis is often seen at which homologues appear to be parallel throughout all or much of their length, but separated from each other at an apparently uniform distance which is obviously greater than the final synaptic distance. It seems reasonable to suggest that precursors for the synaptonemal complex central element may mediate parallel alignment which precedes true synapsis.

Marjorie Maguire

BARCELONA, SPAIN
Departmento de Investigaciones Antropologicas y Geneticas

Relationship between pg 11 pg 12, earliness and modification of some parts of the plants

In MNL 51:4, 1977, A. Alvarez et al. reported differences in earliness of chlorophyll-deficient plants in comparison with normal plants of similar genetic background. The origin of these chlorophyll-deficient plants was from the crosses of one inbred with three other different inbreds. The chlorophyll-deficient plants were earlier than normal plants. The difference of the mean, measured from planting to pollen shedding, between stocks, ranged from 5.3 to 11.3 days. The comparisons were made mainly between sister inbreds, originated from segregating plants in generations S1, S2, S3. We suggested that this chlorophyll-deficient character could be used for practical purposes in order to match flowering time of inbred lines originally of different periods. Later observations indicated that the character is determined by two independent recessive genes. Professor E. H. Coe suggested to us the hypothesis that the character could be the pale green, pg 11 pg 12. In fact, the test for allelism with pg 11 pg 12 fits such hypothesis.

To know behavior of the character in different stocks and in relation with the degree of homozygosis of the background, the study was done on eleven inbreds with inbreeding coefficient from 75 to 98% and on two F2 from single crosses of inbreds of quite different origin. The measured characters were: number of days from planting to silking; plant height; number of internodes; length of internodes below the main ear; length of internodes above the main ear; number of ears per plant; and number of kernels per plant. Comparisons were made between pale green and normal classes. Each pair of classes corresponds to: (1) sister plants of the same segregating inbreds (10 inbreds); (2) sister sublines originated for segregating selfed plants of the last generation (1 inbred); (3) sister plants of the same F2 (2 stocks). The inbreds were planted on two different dates: May 30, 1977 and June 8-10, 1977. For the earliness character, the comparisons have been subdivided according to these two planting dates. Means and differences for sister

plants of 10 segregating inbreds (1) and sister sublines of 1 inbred (2) were as follows:

Character	Planting	Normal (N)	Pale green (PG)	N-PG
Earliness (days from planting to silking)	first	72.58	62.07	10.51***
	second	68.61	61.14	7.47***
Plant height	both	150.68	118.43	32.25***
Number of internodes	both	15.25	12.42	2.83***
Length of internodes below ear	both	10	8.58	1.42
Length of internodes above ear	both	13.87	15.54	-1.67*
Number ears per plant	both	1.84	1.51	0.33**
Number kernels per plant	both	699	458	241***

*p < 0.05; **p < 0.01; ***p < 0.001

Means and differences for sister plants of the same F2 (3) of 2 stocks were:

Character	Normal (N)	Pale green (PG)	N-PG
Earliness	79.9	72.6	7.30***
Plant height	171.5	150.8	20.7**
Number of internodes	14.52	12.40	2.12***
Length of internodes below ear	10.66	10.33	0.33
Length of internodes above ear	13.40	14.20	-0.80*
Number of ears per plant	2.36	1.41	0.95**
Number of kernels per plant	913	476	437***

*p < 0.05; **p < 0.01; ***p < 0.001

The pg 11 pg 12 genotype (1) enlarges the length of the internodes above the ear ($p < 0.05$); (2) on the contrary, reduces the length of the internodes below the main ear, (significantly in the inbreds but not in the F2 groups); (3) reduces all other characters ($p < 0.01$ to $p < 0.001$); (4) had slimmer stems and a greater susceptibility to lodging than the corresponding normal plants (observations not quantified). In conclusion, it appears that pg 11 pg 12 can be useful in seed hybrid production, synchronizing the flowering time of parents originally of different flowering periods.

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

Inheritance of "decussate" character: Differences between the offspring from reciprocal crosses

In previous publications we described the phenotype of "decussate" plants (1967, M. Blanco et al., IV Jornadas de Genética Luso - Espanholas. Itto. Gulbenkian de Ciencias, Oeiras, Portugal, pgs. 41-44). Here we compare segregations in progenies resulting from reciprocal crosses of the same stocks. All the progenies studied resulted from crossing one inbred, A (homozygosis 87.5%), with another inbred, B (homozygosis 75%). Each progeny had only two parent plants (both plants

being "decussate"). In order to do comparisons, the progenies were classified into two groups (I and II). In group I, each "A" plant was crossed, in both directions, with another "B" plant. Therefore, from each pair of parent plants two progenies were obtained, $A_i \times B_i$ and $B_i \times A_i$. From six such pairs of plants, twelve progenies were obtained. In order to compare the progenies A x B with the reciprocal ones, all the six progenies of each class were put together. In group II, fifteen progenies of inbred A crossed (as female parent) with inbred B are compared with 10 of inbred B crossed (as female parent) with inbred A. In this group II, the two parent plants of each cross A x B are not the same plants of each cross B x A. Here too, all progeny of the same class were put together.

The inbred A was a S2 from the cross of the inbred "a" (as female parent) with the "decussate" stock ("De"): Inbred A = (a x De) selfed twice. The inbred B was originated crossing the "decussate" stock (as female parent) with a "b" inbred; crossing again the resulting F1 (as female parent) with the "decussate" stock, and selfing the resulting backcross: Inbred B = [(De x b) x D] selfed.

Due to their respective pedigrees, the cytoplasms of A and B could be different. Because of this, we call the cytoplasm of A, "normal cytoplasm" and the cytoplasm of B, "decussate cytoplasm." The data and the levels of significance of chi-square test, for both groups of comparison, were as follows:

Group	Cross	Progenies	Normal	Decussate	Chi-square
I	A x B	6	79	37	13.63*
I	B x A	6	28	43	
II	A x B	15	219	48	71.15*
II	B x A	10	51	77	

*$p < 0.001$

In group I and in group II, the progeny from female parents with "decussate" cytoplasm have a greater proportion of decussate plants than the corresponding reciprocals.

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

Relationship between "reversed germ" (r.g.), kernel weight, and number of rows of the ears

In MGCNL 30:84-85, Gertrud Joachim reported a study on mutants of maize which produced a variable proportion of kernels with reversed germ ("r.g.") disposition. In corn, two spikelet primordia develop into one upper and one lower floret each. Usually, the lower floret aborts in early primordia development and only the upper one develops into a mature kernel. The interpretation was that, if the abortion of the lower floret did not occur, its development originated a "reversed germ" kernel. This situation could have originated two kernels per spikelet, with a mirror image symmetrical disposition or only one kernel (which was "r.g.") per spikelet, if the upper floret aborted. The character "r.g." could be assigned to three recessive genes, each one of them, by itself, determined a variable proportion of kernels with "r.g.," but the espressivity never reached 100%. The inheritance of the character was found to be maternal.

In the backcrossing process of an opaque-2 flint inbred, plants with the character "r.g." were observed in the segregating offspring of two selfed plants. These two plants were sisters from selfing. From the study of the segregation,

Ear	Total Ears	Ears with "r.g." (%)	Row No. Ears with "r.g."	Row No. Normal ears	Row No. Diff.	Weight of kernels Ears with "r.g."	Weight of kernels Normal ears	Weight of kernels Diff.
1	20	5 (25)	13.6	16.61	3.01**			
2	21	5 (23.8)	13.2	16.12	2.92**			
Total	41	10 (24.4)	13.4	16.34	2.96***	0.31	0.27	0.04*

*p < 0.05; **p < 0.01; ***p < 0.001

concordance was found with Joachim's conclusions. In addition can be reported: a) In our stock the proportion of "r.g." kernels is greater. In fact, from 41 ears studied, 10 had "r.g." kernels, seven of them with 100% "r.g." kernels, and three with proportions of 70.3, 87.6 and 96.7%; b) the ears with "r.g." kernels had a number of rows significantly less than their normal sisters. The kernels of the ears with "r.g." were significantly greater and denser than the kernels of normal ears. However, no difference was found between normal and "r.g." kernels from the same ear. Tests for allelism with the materials studied by Joachim have not been done.

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

Heterosis between a spontaneous mutant, "floury-1," and normal inbred lines

Gardner (MGCNL 45:151-152, 1971) found heterosis in F1 crosses between the induced mutant "Necrotic leaf spot" N25 and normal N25 inbred lines. In this report is presented a similar case of heterosis observed in the F1 between a spontaneous mutant, floury-1 (PF fl1 inbred line) and the original PF inbred line.

PF inbred line has its origin in one variety. In 1971 this inbred segregated kernels of opaque phenotype. The test for allelism with fl1 was positive. When PF fl1 kernels were detected and separated, the inbred had an inbreeding coefficient of 99.96%. Possible contamination was discounted because: 1) the plants resulting from the mutant did not present heterosis; 2) we did not know about having fl1 in any of our stocks. A contamination in the origin of the F1 seed for trials is also discounted because the segregation of the seeds was the expected one. In 1978 were planted two trials in order to: 1) test the statistical significance of the differences of some traits among the mutant line, the normal parent and the F1 hybrid; 2) check the correlation between heterozygosis of floury-1 "locus" with vigor (measured by the height of the plants). In trial no. 1, the parent inbreds (PF fl1 and PF) and their F1 hybrid were planted in three replications, each one of 11 plants. Plant population was adjusted to 25,000 plants per ha. All measures were done on individual plants. Two characters, number of kernels per plant and plant height were measured. The PF fl1 inbred, in relation to normal PF, was: a) smaller in height ($p < 0.001$); b) with inferior number of seeds per plant (no significance); c) apparently with slimmer stems (no measures were done). The F1 hybrid, in relation to normal PF inbred, had greater height and number of seeds per plant ($p < 0.001$, in both cases). In trial No. 2, the F2 seeds were separated into two phenotype classes: normal (+++ and ++ fl1) and opaque (fl1 fl1 fl1 and fl1 fl1 +). Both phenotypes were planted separately and 53 plants of each were obtained. All plants were selfed. After grain maturity, plants were classified in the two classes: segregating and not segregating for the fl1 "locus." From the comparison of the height between the two classes, the segregating one was higher in both phenotype plantings, but not significantly. The second trial does not prove that the heterosis observed in the first one was the consequence of one mutation in one single "locus."

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

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Initiation of corn cell lines

Cell lines (dedifferentiated, rapidly dividing, friable and thus clonable cultures) arise rarely from cereal explants. In corn only three examples are documented (from Black Mexican Sweet, BMS, by W. Sheridan, see Green, 1977, Hortsci. 12:131; from B73, Potrykus et al., 1977, MGG 156:347; and from Orla 266, Funk Seeds Int., King et al., 1978, Physiol. Vegetale 16:381). We are examining several aspects of the biochemistry and physiology of cell line initiation. As in most areas of tissue culture, the genotype of the explant also governs the response. For example, BMS and other flint corns (as originally reported by W. Sheridan, North Dakota) are consistently responsive in our hands. We have studied characters of BMS which might relate to cell line initiation, e.g.: 1) In view of the correlation which clearly exists between cell line status and ploidy abnormalities (not necessarily causal), is the presence of B chromosomes in BMS important for the response? 2) In view of the importance of 2,4-D for culture initiation in dicots, is BMS more sensitive to 2,4-D than typical dent corns? Cultures were initiated from root or shoot explants at either 2 or 5 mg/l 2,4-D; A and B are two lines backcrossed to a common inbred. Cell lines were isolated from trials marked X below:

	With B chromosomes				No B chromosomes			
	Root		Shoot		Root		Shoot	
Plant	2	5	2	5	2	5	2	5
A 1								
2					X			
3					X			
4							X	X
5								
B 1								
2								
3			X					
4							X	
5					X			X

Whilst BMS cultures are atypical of our stock corn cultures in rapidly accumulating dividing polyploid cells, the results suggest that the presence of B chromosomes in the explanted tissues in fact reduces the incidence of cell line initiation.

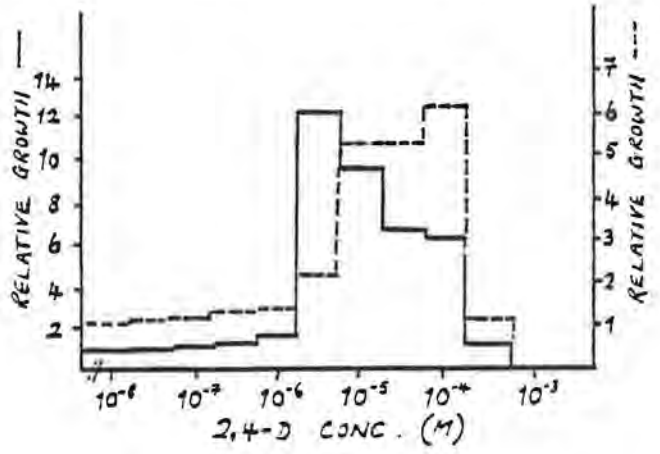

Figure 1. Relative growth (fresh weight) of cultures derived from Black Mexican Sweet (------) and a dent hybrid (———) at increasing concentrations of 2,4-D. Data taken from the end of the second passage at the concentrations indicated. Data given for BMS are for a line without B chromosomes; the results for the line with B chromosomes were essentially the same.

The data given in Fig. 1 also reverse the hypothesis being tested. Newly initiated cultures of BMS (plus or minus B chromosomes) appear less sensitive to 2,4-D than cultures obtained from a commercial hybrid. Higher levels of 2,4-D were required both for growth stimulation and inhibition of BMS compared to the dent corn. The idea that BMS is more responsive to 2,4-D perhaps through a lower rate of degradation is thus destroyed. It would be interesting to hear of data on BMS 2,4-D sensitivity in the field. A further characteristic of BMS is its high prolificacy; we are currently testing several possible biochemical relationships between high tillering and in vitro responses. (We gratefully acknowledge David Weber, Illinois State University for seeds and discussion).

P. J. King, H. Dhaliwal and A. Strauss

Haploid and aneuploid corn cultures

Haploid tissue cultures were initiated from mature embryos of Stock 6 (Coe, 1959, Am. Nat. 93:381) and young haploid yellow green seedlings produced from a cross with yg2 in the female. Cultures were induced on a slightly modified MS medium. Stock 6 cultures were very slow growing and were abandoned after one year; yg2 haploid cultures continue to thrive after more than 18 months. Ploidy analysis using chromocentre and chromosome counts indicated a persistent, high proportion of haploid cells in both cultures after 10 months. The yg2 cultures at present include sectors which are purely haploid as well as diploid and mixed sectors. No intact aneuploid cells were found.

Monosomic corn cultures were also established and their growth and ploidy stability studied. Progeny of a cross between heterozygous r-X1 stock and Mangelsdorf's Multiple Tester were screened for monosomics using recessive seedling markers and chromosome counting; each line was karyotyped. Monosomics (including double monosomics) involving all 10 chromosomes were identified as well as mono-telosomics, mono-trisomics, primary trisomics and double trisomics. Cultures were initiated from roots and stem tissue of seedlings at 2-3 leaf stage. Despite heavy infection losses cultures monosomic for chromosomes 2, 6, 7, 9 and 10 were established. After four months a very small proportion of cells were tetraploid; some purely monosomic and some mixed sectors were found. No significant differences in growth rate were observed between different monosomic cultures. In general the ploidy of the monosomics was more stable than the haploid cultures previously described.

The majority of cell lines in cereals and in dicots are highly polyploid and/or aneuploid, although it is not clear whether aneuploidy as such causes cell line induction by some gene imbalance or whether there is only an indirect relationship. To study this question further in corn, where cell lines rarely occur, we prepared cultures from seedlings which were primary trisomics for all chromosomes but 2 and 8. During two years of subculture none of the trisomic lines gave rise to cell lines. However, cultures trisomic for chromosomes 6 and 10 consistently grew much faster than the others. Extra ribosomal DNA sequences in trisomic 6 and perhaps in 10 (Phillips et al., 1974, Genetics 77:285) might be responsible for this increased vigour, but the differences might equally be the result of undefined differences in genetic background. After ten months most of the cultures remained purely trisomic. Trisomics 7 and 9 were abandoned because of their poor growth. Telocentric chromosomes were found in cells of cultures originally trisomic for chromosomes 1, 3 and 4 but it is not yet clear which chromosomes are involved in this change. Cells with 22 chromosomes were eventually observed in cultures trisomic for 5.

We have recently selected immature embryos which are haploid (using Stock 6), monosomic for chromosome 1 (after r-x1 x Adh^{-}) and monotelosomic for the long arm of chromosome 1 (using A/B interchanges). In all cases the embryos were

selected using the recessive Adh1$^-$ mutant (Schwartz and Osterman, 1976, Genetics 83:63; Cheng and Freeling, 1976, MGNL 50:11) in conjunction with allyl alcohol (details in a paper submitted to Genetics). Tissue cultures were induced on the scutella of the immature embryos and both monosomic and monotelosomic plants have been regenerated from the cultures. The embryos, cultures and plants will serve as a source of protoplasts for mutant selection experiments.

H. S. Dhaliwal and P. J. King

In vitro shoot cultures of teosinte and corn

Corn shoot cultures would be useful for micro-propagation of exceptional genotypes, haploids, etc., and for continuously supplying homogeneous, sterile leaf material for mesophyll protoplast isolation. There was no report up to now of in vitro shoot cultures of corn. The majority of corn lines in normal use possess little or no tillering capacity. Field grown plants of a teosinte collection, El Salado, gave numerous tillers and many axillary branches. Scutellum cultures from immature embryos of B73 x El Salado were induced. These cultures regenerated numerous plantlets during subsequent passages. The plantlets, when transferred to MS medium containing sucrose (30 mg/l), IAA (2 mg/l), kinetin (2 mg/l) and agar (0.8%) gave continuous shoot proliferation. The cultures were incubated at 27 C under 16 h photoperiod at 5000 lux. Shoots from mature seeds of B73, El Salado, and B73 x El Salado were also tested for in vitro shoot culture capacity using similar culture conditions. B73 showed absolutely no response while El Salado and hybrid shoots gave similar results as those of plantlets from immature hybrid embryo cultures. In corn the ability to give in vitro shoot cultures appears to correlate with tillering capacity at the plant level, and may be a further indication (see also the first article above) that breeding towards the highly specialized structure of a commercial dent corn has eliminated many characters essential for in vitro responses at the cell level.

H. S. Dhaliwal and H. Lörz

Selection of amino-acid analogue resistant corn cell lines

Selection of drug resistant variants was performed with a diploid cell line growing on agar medium, originally isolated from mature embryos of Orla 260 (Funk Seeds Int.). Several spontaneous and EMS-induced variant lines were isolated with increasing resistance to the proline analogue, azetidine-2-carboxylic acid (AZCA), the methionine analogue, ethionine (ETH), or the lysine analogue, S-2-aminoethyl-L-cysteine (AEC):

Drug	ID 50% (M)	Selection level (M)	Number of resistant lines found
AZCA	0.5×10^{-3}	3.0×10^{-3}	7
ETH	0.02×10^{-3}	0.2×10^{-3}	35
AEC	0.02×10^{-3}	0.3×10^{-3}	12

Some of the resistant lines show a stable phenotype after several passages on drug-free medium. For one AEC resistant line, AEC^R-12, on which the work has concentrated, the relative growth in fresh weight is inhibited at ca. 5 times the concentration inhibiting the wild type. On drug-free medium the growth rate of the variant is generally slightly less than the wild type. For AEC^R-12 and AZCA^R-3 the free amino acid content of cells grown in the absence of the analogue was measured. No increase in the corresponding amino acids (lysine and proline) was detected.

A. Strauss and P. J. King

Corn protoplasts

There are only two examples of isolated corn protoplasts undergoing sustained divisions to give cell cultures (callus or suspension): 1) protoplasts from corn stem tissue (Potrykus et al., 1977, MGG 156:347) which, however, has not proved repeatable, and 2) protoplasts isolated from established cell lines (those originating from corn stem protoplasts) (Potrykus et al., 1979, TAG in press). The cell lines can be routinely used for establishing protoplast-derived secondary cell cultures, and they have been distributed to various laboratories. In view of our aim--the genetic modification of corn plants through protoplast technology--both systems lack a very important feature: the capacity to regenerate plants.

We are now trying to establish a morphogenic corn protoplast system, but as cereals have proven to be extremely recalcitrant in vitro (King et al., 1978, Physiol. Veg. 16:381) we expect this to be a very long term project. For example there has been no report to date of cereal plant regeneration from cultured single cells or protoplasts.

We are isolating and culturing protoplasts from tissues or cells which have either expressed totipotency or which can safely be assumed to be totipotent: 1) very young embryos--2-3 days post-pollination, 2) immature embryos--12-18 days, 3) immature embryos undergoing multiple shoot formation (Green et al., 1975, Crop Sci. 15:417), 4) meristems (vegetative shoot tip, tassel and ear primordia, nodes, leaf base, 5) sporogeneous tissues, 6) pollen mother cells and 7) microspore tetrads. As there is no method available to detect or select for totipotent cells, even here we deal with mixed populations of protoplasts. In addition, we are continuing our trials with protoplast populations which may or may not include totipotent cells: internode, leaf sheath, leaf blade, crown roots, nucellus and endosperm. Our renewed interest in culture of protoplast from these organs stems from the observation that differentiating cells in some of these organs can be induced to embark upon additional divisions in vivo.

I. Potrykus, D. Hanold, C. Harms, P. Larkin and H. Lörz

Corn anther culture

Recently published work in China has demonstrated that it is possible to obtain haploid green maize plants from pollen by culturing anthers on agar medium containing high concentrations of sucrose (10-15%) (Anon., Acta Botanica Sinica 19:89-94, 1977). Yet, even under optimal conditions, the production of pollen callus or embryos is rarely observed in more than 1% of the cultured anthers. Although haploid maize plants are already available through the use of Coe Stock 6 and the ig mutation, the development of techniques permitting the growth of large numbers of microspores may be more useful for mutagenesis and the subsequent selection of desirable mutants in vitro.

During the summer months of 1978, over 100,000 anthers of maize and maize x teosinte were cultured in our laboratory from both greenhouse and field-grown material. Calluses that were distinct from the anther wall or filament were observed on a number of anthers after 6-8 weeks in culture, but these did not undergo morphogenesis. However, a single embryo was also produced on one anther of cultivar G-4507 (Funk Seeds International) cultured for 7 weeks on an N_6-medium containing 12% sucrose and 0.35 mg/l TIBA as recommended by the Chinese workers (G. Melchers, personal comm.). This embryo initially grew rapidly to produce a well-defined coleoptile, but after reaching a length of 5 mm could not be induced to develop further. No chromosome counts were obtained.

Cytological examination of the anthers showed that the normal processes of pollen development were generally inhibited in culture. Associated with this was a

rapid deterioration of the microspores. Under certain conditions, however, a proportion of the microspores were able to continue developing towards maturity in the cultured anthers. Thus, normal development resulting in starch accumulation appeared to occur in microspores cultured just prior to the first microspore mitosis. Multinucleate structures were observed rarely, but it was not possible to speculate as to whether they might represent the first steps of an androgenetic pathway.

The lack of significant success in our experiments might in part be attributed to a failure to optimize the many variables (such as genotype of the donor plant and its growth environment) which condition a favorable anther response. Further experiments are in progress to fractionate populations of isolated maize and sorghum microspores on percoll gradients (c.f. W. Wernicke et al., 1978, Naturwiss. 65:540). We hope that these experiments in conjunction with the anther culture work will help us to define conditions required for the induction of maize androgenesis in vitro.

E. Thomas, R. Brettel and W. Wernicke

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Genetic analyses of MDH: An operational model

Starch gel zymograms of maize malate dehydrogenase (MDH) often reveal complex isozyme patterns. Elucidation of the genetic basis of the observed gel phenotypes has been facilitated by obtaining isozyme variants and analyzing the banding patterns in various combinations. During the last three to four years, such genetic studies have advanced considerably our knowledge of the number and subunit interactions of the maize MDH genes. Based upon independent but concurring evidence from two laboratories (Goodman et al., MNL 52:99, and Newton, these articles), one can propose a model for the inheritance of the cytoplasmic (sMDH) and the mitochondrially-associated (mMDH) forms of MDH.

The essential features of this model have been previously suggested by Goodman et al. and are illustrated in Fig. 1-1. Yang's band designations (e.g. in Isozymes, vol. 3) are used in abbreviated form (s-MDH^1 is shortened to s-1, m-MDH^1 becomes m-1, etc.). They are shown at the left side of the depicted gel banding pattern. The presumed subunit composition of each of the bands is given to the right of the pattern in Goodman's informal terminology.

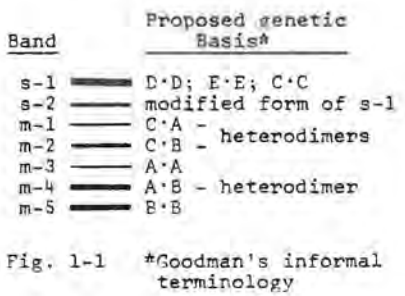


Fig. 1-1 *Goodman's informal terminology

In most of the commonly-encountered MDH isozyme profiles, the broad, heavily-staining s-1 band includes the homodimeric products of 3 independently segregating genes. Two of these (MdhD and E) encode forms active in the cytoplasm (sMDH's) and the third is a weakly staining mitochondrial isozyme gene (MdhC). The s-2 band appears to be a modified form of the s-1 band. Bands m-3 and m-5 correspond to homodimers of the products of two other independently segregating mitochondrial form genes (MdhA and B), while m-4 is the hybrid band (A·B). Bands m-1 and m-2 also represent heterodimers, between the C and A gene subunits and the C and B ones, respectively. Apart from these five structural loci, a recessive allele of a sixth locus modifies the normal migration of the mitochondrial MDH's. Additional structural loci probably also exist in isolated lines, but most of the patterns we have so far examined can be explained on the basis of the proposed 5 structural gene model.

In the subsequent articles of this series, we report some of the evidence which led us to formulate this type of model as well as results from some localization studies--which support it. In our analyses, slivers of soaked mature scutella were squashed onto filter paper, inserted into 12% starch gels made with a Tris-citrate buffer (pH 7.0) and subjected to electrophoresis for approximately 6 h at 220 volts.

Kathleen J. Newton

Evidence that the soluble MDH genes are duplicate in maize

In a line of Papago Flour corn originally obtained from the Maize Cooperation Stock Center, some selfed ears were found to be segregating two additional bands, as in the A and B patterns in Fig. 2-1. In pattern A the faster of these bands stained almost as intensely as the s-1 band, while the intermediate band was strongest. Pattern B was skewed toward the s-1 band. Pattern C contained the usual s-1 band. Upon selfing, patterns A and C were invariant, whereas pattern B segregated all three (A, B and C) patterns. These results are most simply explained by postulating the presence of an additional gene whose product can form a heterodimer with the s-1 band. If the faster (F) and the normally-migrating s-1 band (N) had represented alleles at a single locus, one would have expected segregation of F (alone), pattern A and pattern C in a 1:2:1 ratio in the progeny of a selfed pattern A plant.

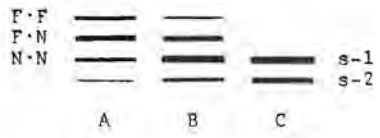
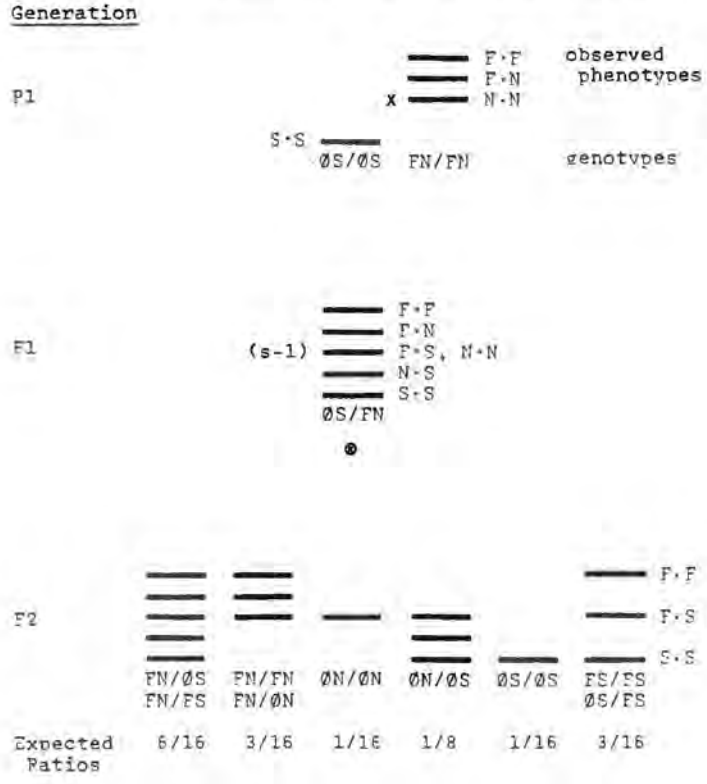


Fig. 2-1

Several lines were crossed reciprocally to plants exhibiting pattern A. In nearly all tested cases, only pattern B was observed in the F1 progeny kernels. The fact that the N·N homodimer band was stronger than the F·N heterodimer band in these hybrid plants is consistent with the interpretation that most of the maize lines we tested contain duplicate genes encoding the s-1 band. FN/NN heterozygotes would be expected to exhibit an approximate 9:6:1 relative ratio of the N·N, N·F and F·F bands, respectively, if the two gene products are approximately equal in activity. In all of these kernels there was a positive correlation between the intensity of the s-1 N band and the s-2 band.

In a screening of exotic lines available in our laboratory, a slow-migrating variant (which migrates to the approximate position of the m-1 band) was found to be segregating along with an apparent null for the other s-1 determining locus. The line was characterized by poor germination and general inviability; however, it was possible to recover double homozygous (slow, null) kernels in the F2 generation of an outcross. In order to answer questions about linkage and subunit interactions of the two sMDH genes, plants with this slow,



null phenotype were crossed by plants homozygous for the Papago phenotype. The F1 kernels exhibited a five-banded pattern (Fig. 2-2). Upon selfing, six different phenotypes (classified as to number and types of bands) were resolved in the progeny F2 kernels (Fig. 2-2). If the genes were closely linked, one would expect 3 major phenotypes; 25% of the F2 kernels would be "slow, null" and the homozygous FS/FS pattern would be a rare recombinant phenotype. If the genes were unlinked but the slow and fast subunits were alleles of the same locus, one would expect to see the F·F homodimer band segregate out alone 1/16 of the time. If, however, there are two independent genes and the fast and null variants derive from alleles of the same locus, then the ratios in Fig. 2-2 would be expected. Analysis from our initial data from these F2 kernels shows them to be in close agreement to the expected ratios (Fig. 2-2) and thus, the last situation holds. On the other hand, the s-2 band is NOT independent; it varies with the position of the s-1 band (trailing it) and is, therefore, probably due to a modification of one or both sMDH gene products. We see no hybrid bands forming between s-1 and s-2, suggesting that this modification occurs perhaps after dimerization.

Kathleen J. Newton

A gene affecting the electrophoretic mobilities of the mitochondrial MDH's is on chromosome 1

A modifier affecting the migration of the mitochondrial MDH bands was discovered independently by M. Goodman, et al. (MNL 52:99) and myself. The modifier is specific for the mitochondrial bands, effecting a slightly faster anodal migration. Simultaneously, the mitochondrial bands stain less intensely--indicating that modifier has a slight effect on the catalytic activity of these isozymes.

Several crosses with our homozygous modifier line have been analyzed. In one series of crosses, a normal pattern (A in Fig. 3-1) was crossed by the modified pattern (B in Fig. 3-1). All tested F1 kernels were "normal." When these kernels were selfed, a 3:1 ratio was observed (i.e. 58 normals: 20 modifieds). When they were backcrossed to the modifier line a 1:1 ratio (58:57) resulted; whereas, when backcross ears to the normal line were tested, no modified kernels were found. These data indicate that modifier is a recessive allele of a locus whose product is somehow involved in the processing of mMDH.

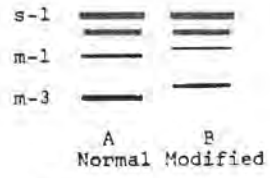


Fig. 3-1

Unlike most previously studied modifiers, this locus must be homozygous for the mutant allele in order for the abnormal phenotype to be observed. This suggests that when the normal functioning of the locus is impaired in this way, the mitochondrial MDH's migrate faster and are less active. F2 data from crosses between the modifier line and lines with mitochondrial MDH structural gene variants indicate that these structural loci are genetically independent from the modifier locus.

The standard B-A translocation method for locating recessive mutants (most recently reviewed by J. B. Beckett, J. Hered. 1978) greatly facilitated the localization of the modifier gene. Twenty-two B-A translocations covering 14 of the chromosome arms were used; only those containing a certain segment of the long arm of chromosome 1 "uncover" the modified MDH pattern.

	Breakpoints in 1L	Uncovers modifier?
TB-1La-3L4759-3	0.2-0.38	No
TB-1La-4L4692	0.2-0.46	No
TB-1La-3Le	0.2-0.58	No
TB-1La-3L5267	0.2-0.72	Yes
TB-1La-5S8041	0.2-0.80	Yes
TB-1La-3L5242	0.2-0.90	Yes
TB-1La	0.2-1.00	Yes

Provided that the reported breakpoints are accurate, it would appear that the MDH modifier locus lies between 0.58 and 0.72 on the long arm of chromosome one. The compound B-A 1L-3L translocation series was constructed by J. Birchler, who generously supplied these stocks to me.

Since the use of hyperploid plants (A A^B B^A B^A) as pollen parents facilitates recovery of hypoploid (hemizygous) progeny, the following protocol was instituted. Kernels bearing hyperploid scutella were identified on the basis of small size and/or mutant (hypoploid) endosperm phenotype. When possible, the anthocyanin marker systems (Birchler, MNL, 1979) were used to distinguish the hyperploid scutella. With TB-3La, and with the compound TB-1L-3L series, crosses to Birchler's a-m-1 R-scm tester line had previously been made, which facilitated the choice of the partially trisomic kernels. Plants grown from such kernels were crossed reciprocally with the modifier line. When testing modifier x TB-1La (1 1^B B^1 B^1), alcohol dehydrogenase (ADH) phenotypes could be used to distinguish the doses of 1L. Birchler had incorporated an Adh1-S allele onto the particular TB-1La used in these studies and crossed it to an Adh-C line. The modifier line is Adh1-F. Preliminary data from this cross are as follows: 1) Approximately 50% of the kernels on the ear were small and none of the fourteen which were tested exhibited the modified mMDH phenotype. 2) Twenty-five large kernels were tested--sixteen of them showed the modified pattern, the other nine kernels showed a normal migration for mitochondrial MDH's. 3) ADH phenotypes confirmed the fact that the kernels exhibiting the modified phenotype were hypoploid since they were all Adh-F/- and the presumptive euploids were F/S or F/C. In addition, kernels from the exact reciprocal cross (TB-1La x modifier) did not show the modified mMDH.

Although the numbers collected so far are small, the localization of modifier seems fairly certain. Contamination could not account for the "uncovering" of modifier: 1) The modifier line is su/su and none of the hypoploid embryos had sugary endosperm; 2) It should be noted that the uncovering of modifier occurs in 30-40% of the tested kernels, when hyperploid pollen parents carrying TB-1La or TB-1La-3L5267 were used.

Goodman et al. have determined that one of the sMDH(MdhD) genes is tightly linked to their modifier of mMDH mobility locus. If their modifier is allelic to the one described here, this would also place one of the duplicate soluble MDH genes on 1L. It is more difficult to map these duplicate genes since most of the B-A translocation-carrying stocks have both loci active and one must rely on distinguishing relative isozyme band ratios. Nonetheless, we are employing double variant sMDH tester lines and making exact reciprocal crosses with the TB-A's in our continuing efforts to localize the sMDH genes.

Kathleen J. Newton

The gene encoding the most active form of mMDH is uncovered by TB-6Lc

Yang et al. (PNAS, 1977) reported that the darkest staining mitochondrial band, m-3, in a common pattern consists of the product of two independent loci. In some lines, there is a variant, m-5, of the "stronger" of these genes (MdhB). Band m-4 is known to represent a heterodimer between the two different gene products.

In an attempt to locate the genes encoding mMDH's, we used a pattern (see A of Fig. 4-1) which was lacking a band at the m-3 position in reciprocal crosses with the B-A translocation lines. With TB-6Lc the F1 scutellar MDH patterns differed depending upon the direction of the cross. Our mMDH tester line was homozygous for the y allele, and, therefore, white endosperms in the F1 kernels were associated with hyperploid scutella. Because (1) we are dealing with duplicate genes and (2) the B^A chromosome is carrying the m-3 allele of the major locus for mMDH, phenotypes were only distinguishable on the basis of relative band activities. However, since three distinguishable patterns resulted from a cross between two "homozygous" lines, one of which was invariably associated with white endosperms and, due to the fact that pattern E (Fig. 4-1) is lacking in the exact reciprocal cross (where the same TB-6Lc plant is used as the silk parent), we conclude that the gene encoding the most active form of mMDH is located on the long arm of chromosome 6.

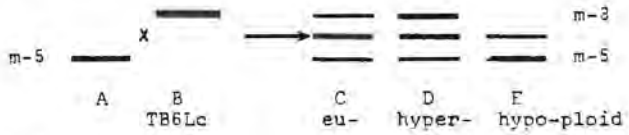


Fig. 4-1

This localization is supported by the independent work of Goodman et al. (MNL 52:100), since they demonstrated the simultaneous presence of three alleles of this gene in plants trisomic for chromosome six.

Kathleen J. Newton

A "null activity" allele of the mitochondrial MDH gene on chromosome six

In a Knobless Wilbur line obtained from the Maize Genetics Coop, we discovered an odd variant of the mMDH pattern. This line contained the m-3 homodimer and the m-4 heterodimer bands but lacked an m-5 homodimer. F2 data from outcrosses (Fig. 5-1) of this line showed that the Knobless Wilbur variant segregated as an allele of the gene we have localized to chromosome six. The fact that we were seeing a heterodimer (m-4) band suggested to us that the locus on 6L was producing a mutant protein which is capable of forming heterodimers with active subunits but which is itself catalytically inactive. Such null activity CRM+ alleles are known to exist for the Adh1 locus (D. Schwartz and T. Endo, Genetics, 1966).

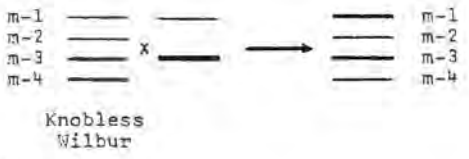


Fig. 5-1

We tested this hypothesis genetically with the cross depicted in Fig. 5-2. The variant pattern (Fig. 5-2A) in this cross is homozygous. Both alleles of the 6L locus encode products which homodimerize at the m-3 position. The second gene (MdhA, whose location is not yet confirmed) is homozygous for an allele encoding a slow migrating mitochondrial variant--the m-7 band. The A·B heterodimer in this case migrates to a position between m-4 and m-5. A cross

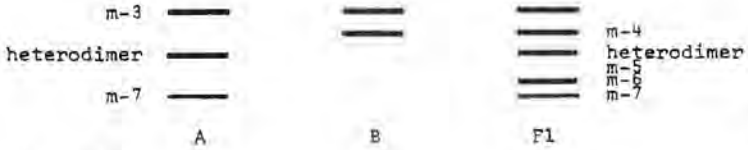


Fig. 5-2

between this line and Knobless Wilbur results in an F1 pattern which includes the hybrid band, m-6, that is normally formed when the m-5 and m-7 bands are present. These results support the interpretation that the Knobless Wilbur line contains a B gene activity null which is capable of forming heterodimers with two different A gene variants. Crosses of the Knobless Wilbur activity null by TB-6Lc demonstrated that this variant was also uncovered.

Kathleen J. Newton

A third independently segregating mitochondrial MDH structural gene has been localized to 3L

Goodman et al. (MNL 52:99) identified variants of a third mitochondrial MDH locus, MdhC, which is distinct from the other two mitochondrial genes, A and B (the 6L gene). Unfortunately, on a tris-citrate, pH 7.0 starch gel system, the homodimer of the most common allele of the C gene (C-16) migrates to a position obscured by the broad and intensely-staining s-1 band, composed largely of the soluble MDH's.

In our lab several methods have been used to demonstrate the fact that there is a residual activity at the s-1 position which is not due to the sMDH's: e.g. (1) Genetic removal of the sMDH's from the normal (N) s-1 position as in the homozygous "null slow" pattern of Fig. 2-2. Under these conditions, there is a faint band discernible at the N position. This band does not form a heterodimer with the sMDH slow variant. (2) Goodman, et al. have shown that use of an ascorbate-sucrose homogenizing medium selectively eliminates sMDH's from the starch gel zymograms. Although this technique causes a slight smearing and greater separation of the mitochondrial isozymes, they are still visible and scoreable. Both Goodman and L. Bauman kindly provided kernels of the H25 line--which is homozygous for a variant (C-18) migrating to a position well above the obscuring s-1 N band. In outcrosses of this line a heterodimer forms between the C-18 and s-1 "normal" position. Elimination of the sMDH's with sucrose-ascorbate reveals an approximate 1:2:1 staining intensity ratio of the C-18 homodimer:C-18·C-16 heterodimer:C-16 homodimer.

The H-25 line was crossed with the B-A translocation lines, per previously described protocol. The following TB's uncover the C-18 variant: TB-3Ld, TB-3Lc and TB-1La-3L5267. TB-1La does not uncover the C-18 variant. Dr. J. Beckett generously provided the TB-3Lc and 3Ld stocks which were then crossed to Birchler's a-m-1 R-scm tester. Therefore genetically defined hyperploid plants were used for the crosses with C-18. Approximately one-third of the kernels tested from an H25 x TB-3Ld cross uncovered C-18. (Although in a hemizygote, the homodimer band was difficult to see, such kernels were easy to identify by a lack of the appropriate heterodimers.) The fact that the compound TB-1La-3L5267, whose 3L breakpoint is listed at 0.73, uncovers the C-18 variant, indicates that the MdhC gene is located in the distal region of the long arm of chromosome 3. Genetic linkage studies are in progress.

Kathleen J. Newton

On the nature of the mMDH-1 and mMDH-2 bands

Had photographs of gels been included with these notes (instead of simplified diagrams), the reader would have had difficulty overlooking the coincident variation of the "intermediate" mMDH bands, such as m-1 and m-2 (in Fig. 1-1), with the positions of the "lower" (m-3, m-5, m-7) and "upper" (e.g. C-18) mMDH bands. For example, when the m-5 band is present, there is nearly always a band at the m-2 position. The notable exception to this rule occurs in the H25 line, where C-18 as well as m-5 is present. In this case, there is no mitochondrial band at the m-2

position; however, there is a mitochondrial MDH band migrating to approximately the s-2 position, which is intermediate between C-18 and m-5. One of the clearest illustrations of the formation of heterodimers between the C gene product and the products of the other mitochondrial loci is shown in Figure 7-1. The first pattern

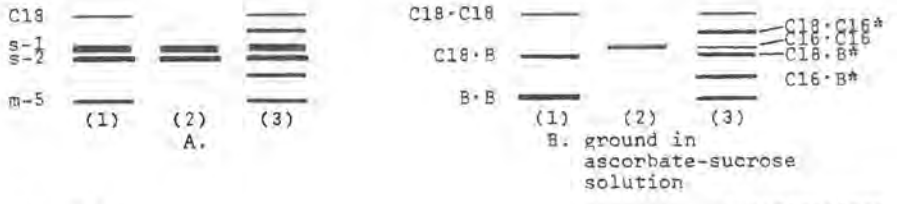
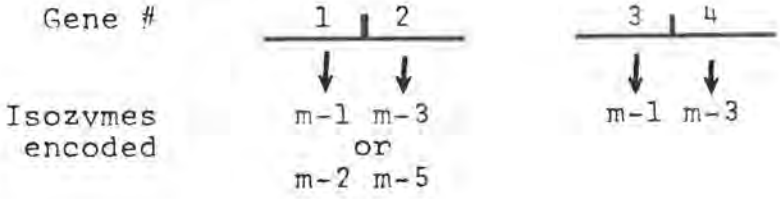


Fig. 7-1

is present in the H25 line and pattern two is present in a line kindly provided by Goodman. When these two lines were crossed together the F1 MDH profile in pattern three was observed. In Fig. 7-1B the kernels were ground in the ascorbate-sucrose solution in order to selectively eliminate the soluble bands. An obvious conclusion is that the C locus product can participate in the formation of both allelic and intergenic heterodimers. In short, there is classical genetic evidence supporting the idea that the products of the three identified mitochondrial MDH genes can freely heterodimerize. In addition, evidence from TB-A localization studies strongly support this interpretation.

In 1977, N. Yang, J. Sorenson, and J. Scandalios (PNAS 74:310) proposed an alternative genetic model for the mitochondrial MDH genes. They were the first to publish the fact that there is a duplication of the maize mMDH genes. However, in that paper, the authors attempted to explain their m-2 and m-1 bands as products of additional closely linked duplicate genes, as shown below:



The Model of Yang, Sorenson and Scandalios

This hypothesis is problematical from a mutational point of view since it requires correlated mutation in closely linked genes. When there is a mutation in their Mdh2 gene--leading to an altered electrophoretic mobility of its enzymatic product, a corresponding mutation (leading to a product changed in the same way) must occur in their Mdh1 locus, either subsequently or simultaneously.

The Yang, Sorenson and Scandalios model also requires restrictions on dimer formation. Despite the fact that Yang has demonstrated that the products of two independently assorting mMDH loci--their Mdh2 and Mdh4--form heterodimers (corresponding to band m-4), their similarly related Mdh1 and Mdh3 gene products would not form heterodimers. In addition, their Mdh1 and Mdh2, as well as Mdh3 and Mdh4 gene products, could not heterodimerize. In fact only the products of

Mdh2 and Mdh4 would interact on a subunit level. While such heterodimer formation restrictions are not conceptually impossible, our evidence suggests there is free heterodimerization among the products of three independently assorting mitochondrial MDH genes.

The evidence that Yang et al. cite in favor of their model is two-fold: recombinational and biochemical. In the discussion of the paper, the authors report seeing three recombinants (out of 977 tested F2 progeny) between Mdh1 and Mdh2 such that band m-1 was seen with m-5 instead of band m-2 with m-5. Bands m-3 and m-1 were also present from their loci Mdh3 and Mdh4. However, no attempt was made to establish these exceptional progeny as genetic stocks for further analysis. Without such confirmatory evidence, it is extremely difficult to eliminate the possibilities of electrophoretic artifact or, in the absence of contamination markers, fertilization by pollen from other lines.

Biochemical evidence presented by Yang includes the observations that m-1 and m-2 isolated isozymes are very similar in their kinetic properties, while m-3 and m-5 are also similar according to the same criteria. Such evidence is compatible with a heterodimer interpretation for bands m-1 and m-2. Assuming that the mitochondrial gene on chromosome 3 (MdhC) differs in its biochemical properties from the other two mitochondrial loci, then m-1 and m-2--which each have a C-gene subunit--would be expected to be similar to each other, but different from m-3 and m-5.

Finally, one can explain on a "heterodimer model" a type of pattern (found in their lines 81 and 6) that was difficult for Yang et al. to interpret on their model. The pattern with our interpretation is illustrated in Figure 7-2. In this case, bands were present at their m-3 and m-5 positions although the m-4 hybrid band, which they expected to see, was missing. On our model, the band at m-3 would not represent a homodimer--rather it would be a heterodimer between two different subunits: the product of the A gene encoding the m-7 homodimer and the C gene product, whose homodimer migrates at the s-1 position.

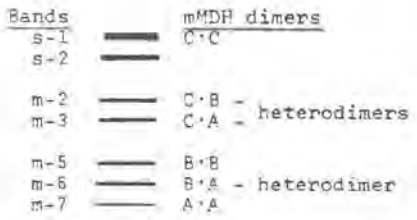


Fig. 7-2

Kathleen J. Newton

Proposed revisions in nomenclature for the maize MDH loci

In the above articles, we have presented our data in terms of previously published MDH isozyme and gene designations. However, since our evidence supports the idea that there are at least three mitochondrial and two soluble Mdh loci, we propose that the locus symbols now be revised to be consistent with the current conventions of maize genetics (MNL 49:3-4).

Listed below is our proposed nomenclature juxtaposed with the corresponding designations used by other investigators. We have attempted to make this

Proposed Conventional Symbols		Goodman et al. Laboratory Designations	Yang et al. Nomenclature
Mdh1		MdhA	mdh4
Mdh2	(on chr. 6)	MdhB	mdh2
Mdh3	(on chr. 3)	MdhC	-----
Mdh4	(sMDH)	MdhD	-----
Mdh5	(sMDH)	MdhE	-----

transition as easy as possible by merely changing the alphabetical designations of Goodman et al. to numerical ones. Since the MDH system involves so many loci contributing differentially to the isozyme pattern, we feel that it is important to always include the number 1 in the Mdh1 symbol. One could incorporate the laboratory symbols in the allele designations, e.g. Mdh3-C18. Additional loci, as discovered (whether soluble, mitochondrial or glyoxysomal) would simply be assigned the next number. Finally, the modifier locus has been named the "modifier of mitochondrial MDH" and given the symbol, mmm.

Kathleen J. Newton

Anaerobic stress and RNA production in seedling roots

The de novo production of specific proteins, most notably ADH, in seedlings in response to anaerobic stress is a well documented phenomenon (Sachs and Freeling 1978, MGG 161:111; Ferl et al. 1978, MGG in press). Our protein labeling studies have led us to an examination of RNA production under the same conditions.

Studies monitoring the incorporation of P32 or H3 RNA precursors into specific RNAs (as displayed in a variety of high resolution polyacrylamide gel systems and detected by autoradiography) have produced some very interesting results. The most notable among these concerns an RNA with an apx. mw of .8 x 10^6 dalton. Basically the facts on this particular RNA are as follows:

--It is an induced RNA species. That is, it does not appear in RNA preps from unstressed seedling roots labeled in air, but does appear in root preps from identical seedlings labeled early during anaerobic stress induced by a N_2 atmosphere.

--Pulse label time course experiments show that this induced RNA is made predominantly during the early stages of anaerobic stress, mostly in the first 8-10 hrs. Some slight production of this species has been noted at later times.

--This RNA is fairly stable. Pulse-chase experiments have shown that this RNA, after being labeled for a 2 hr period early during anaerobic stress treatment, maintained its label after 22 further hours of anaerobic chase in cold medium.

--This RNA is apparently poly A containing, as judged by its ability to reversibly bind to oligo (dT) cellulose. In fact, it appears to be by far the major poly-A containing species that takes up labeled precursor during anaerobic stress.

All the facts mentioned above are consistent with the possibility that this RNA is the messenger RNA for ADH. Although all the correlations fit, to date direct evidence to support this contention is lacking. Currently our efforts to test this possibility are two-fold: 1) We have available many Adh1 nulls, the Adh1-FCm duplication, an Adh1 molecular weight variant U725 and other Adh1 and Adh2 mutant types. These are all being tested in the hope that one or more of them may produce either no or a detectably altered form of this particular RNA. 2) In vitro translation is being used in an attempt to identify the protein product of this RNA and its similarity, if any, to ADH.

Studies concerning the effects of anaerobic stress on other RNA species are also in progress.

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Linkage tests of golden-2

Golden-2 (g2) has a more extreme phenotype than g. The culms and leaf sheaths are nearly white. The current linkage maps indicate that g2 is located on

chromosome 7, locus unknown. In scoring some F2 families segregating for this trait (source: Coop. 64-169) and various chromosome 7 markers, the ease with which multiple gene combinations were recovered suggested that g2 might not be on this chromosome. A definitive answer was sought from backcross tests with sizeable populations. The first four lines of the following table summarize the relevant two point tests from the testcross g2/o2 v5 ra gl x g2 o2 v5 ra gl (3 families, 1050 total plants). The last line is from the testcross g2/bd x g2 bd (4 families, 1180 total plants). There was good homogeneity among the families of each cross for single gene segregations as well as for independence. The single factor segregations of g2 and of each of the marker genes did not deviate significantly from the expected 1:1.

Tester Mutant	+ +	+ t	g2 +	g2 t	Total	Percent recombination
o2	285	248	270	247	1050	49.3
v5	275	258	265	252	1050	49.8
ra	271	262	268	249	1050	50.5
gl	270	263	265	252	1050	50.3
bd	307	296	295	282	1180	50.1

None of these deviate significantly from independence. The known map of chromosome 7 is presently 112 crossover units long. Based on chiasma frequency the theoretical length of this chromosome is 123 map units (Rhoades, 1955), therefore the above data adequately test the entire length of this chromosome. It is concluded that g2 does not lie on chromosome 7.

This conclusion is supported by a subsequent test with an A-B translocation. When F1 plants of the constitution g2/o2 v5 ra gl were pollinated by plants carrying TB-7Lb, none of the resulting hypoploid plants were g2.

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Division of Plant Industry, C.S.I.R.O.

Mapping of glutamic dehydrogenase (Gdh) on chromosome 1, 20.1 recombination units distal to Adh1

The waxy marked reciprocal translocation stocks were used to determine the location of Gdh in the distal region of the long arm of chromosome 1. Linkage with waxy was observed with wx T1-9(8389) (1L.74; 9L.13) but not with any other of the translocation stocks tested (Table 1), including wx T1-9(4995) (1L.19; 9S.20),

Table 1. Segregation of wx and Gdh in crosses with reciprocal translocations involving chromosome 9 (T wx Gdh-N/Wx Gdh-T x wx Gdh-N/wx Gdh-N).

Translocation	Wx Gdh-T	Wx Gdh-N	wx Gdh-T	wx Gdh-N	Total
1-9c	11	9	10	10	40
1-9(4995)	10	10	10	10	40
1-9(8389)*	16	4	4	16	40
2-9b	13	7	14	6	40
3-9c	7	13	10	10	40
4-9b	10	10	14	6	40

*Observed segregation deviates significantly from the expected 1:1:1:1.

Table 2. Segregation of waxy translocation 1-9(4995), Adh and Gdh (T wx Adh-S Gdh-N/Wx Adh-F Gdh-T x wx Adh-F Gdh-N/ wx Adh-F Gdh-N)

	Adh-F Gdh-T	Adh-F Gdh-N	Adh-S Gdh-T	Adh-S Gdh-N	Total
Wx	53	10	8	31	102
wx	41	8	17	46	112
Total	94	18	25	77	214

χ^2[1] L (Adh-Gdh) = 148.8; p <<0.001;
Recombination Wx Adh = 41 ± 3.3%, Adh Gdh = 20.1 ± 2.7%

which has the break point in the proximal region of 1L. This location was confirmed by the demonstration of 20.1% recombination between Gdh and the Adh1 locus (Table 2), which is known to be between 0.80 and 0.90 on 1L (Birchler MNL 52:27-31). This cross also involved the wx T1-9(4995) translocation which as mentioned previously showed no linkage with Gdh, but did show a low but significant linkage of waxy with Adh. This indicates that the Gdh locus is about 20.1 recombination units distal to the Adh1 locus on 1L.

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Different ploidy in perennial teosinte from Jalisco, Mexico

Populations of perennial teosinte were rediscovered in Jalisco by Rafael Guzmán Mejía of the Instituto de Botánica, Universidad de Guadalajara, at Guadalajara, Jalisco, in 1977. The author visited two of the localities last November. The first site was located at Rancho Los Cimientos in the Ejido de Piedra Ancha at about 30 km S.W. from Cd. Guzmán at an altitude of 2100 m; the second site was located in San Miguel at about 15 km east from Cuzalapa, Municipio de Cuautitlán, at an altitude of 1800 m. From rhizomes collected at each site, root tips were gathered in the greenhouse at Chapingo, and chromosome counts performed. It was found that plants from Cd. Guzmán were tetraploid (2n = 40) and those from San Miguel were diploid (2n = 20). Since both sites are separated about 70 km from each other on a straight line, it would be of interest to search the region with more detail, especially around the Nevado and Volcán de Colima, in order to determine how extensively the perennial teosinte is present, and what are the distributions of the populations at each ploidy level.

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Maize anther culture

Four basal media were tested. These were two media (N6 and Yu Pei) developed by Chinese workers, Linsmaier and Skoog's and Potrykus' media for maize protoplasts. Amino acids and hormones were added in various combinations resulting in the testing of 145 variations of media. At least 50 anthers were tested on each kind of medium. Several maize strains were tested. Positive results as evidenced by the formation of 4 identical nuclei in pollen grains were obtained on a modified Yu Pei medium. Limited callus growth was obtained but no plants have been regenerated.

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The recessive intensifier of plant color (a3)

The original description of a3 in 1934 by E. W. Lindstrom (MNL 8:10) identifies "a new recessive anthocyan gene" with data indicating 22% recombination with golden and 40% with R. Further description apparently is not in the literature, but the a3 stock supplied from the Stock Center is described as a recessive intense plant color. C. R. Burnham and R. V. Kowles (MNL 43:113) found no satisfactory evidence of linkage of a3 with R, sr2, or g. The recessive factor is uncovered by TB-3La (MNL 47:147). The data below indicate that a3 is a recessive gene whose expression requires an intermediate-level (barred) B allele. The barred allele in these experiments is B-a3, one of the low-intensity alleles at this locus (MNL 51:61); since this allele has been extracted from the a3 stocks, by intercrossing and selfing, as a homozygous strain that is only barred (i.e., not intense), the intense a3 expression is controlled by an additional gene. Presumably the additional requirement is the recessive factor on 3L which, when hemizygous in the presence of B-a3, intensifies the plant color.

F2 progenies from B-a3/b, a3/+ (F1 plants were barred):

R constitution, seed color	Barred	Intense*	Green
R-g/r-r, colored class	50	25	22
R-g/r-r, colorless	71	25	20
R-g/R-g	55	16	12
R-g/r-g, colored class	40	8	18
R-g/r-g, colorless	42	13	15
Total	258	87	87
Expected for 9:3:4	243	81	108

$\chi^2 = 5.45$ ns

F2 progenies from B-a3/B, a3/+ (F1 plants were purple):

R constitution, seed color	Purple	Intense*	Barred
r-r/r-r	71	8	4
R-g/R-g	61	4	18
R-g/r-g, colored class	27	2	7
R-g/r-g, colorless	28	2	7
Total	187	16	36
Expected for 12:1:3	179.2	14.9	44.8

$\chi^2 = 2.15$ ns

*Purple plants with green auricle, somewhat less darkly pigmented than B P1, with faintly pigmented cobs.

Intense individuals in progenies segregating for green and intense (as in the first of the above F2 progenies) have always given barred progeny from crosses with A3 b, demonstrating that intense plants must be a3 B-a3 (i.e., never a3 b). Similarly r-r, R-g and (in a few tests) R-r do not appear to duplicate the effect of B-a3, since intense individuals in segregating progenies have always given barred progeny in the same test cross (i.e., they were never a3 b r-r, for example).

The above observations demonstrate that a3 is a recessive intensifier of the plant color conferred by B-a3 (and presumably other B alleles). Chromosome 3 linkage tests are in preparation.

E. H. Coe, Jr.

MESS--Missouri Early Stressed Synthetic

A group of early lines (earliest Gaspé from R. Brawn; P.I.245134 and 245132; adapted genetic material) were combined in a population with purple aleurone and purple plant factors beginning in 1965, and the population has been subjected to diverse stresses intended to press toward early maturation in normal field planting, late field plantings, summer greenhouse, autumn greenhouse, winter greenhouse, extreme crowding, etc. Crosses with several other sources have been added to the population (Zapalote Chico; early sweet corns; Oaxaca 179, Latente) at times. Some of the plants in the population (which is very heterogeneous) flower very promptly. A few unsystematically collected examples:

Planted	First Flowers	Harvested
14 May 66	20 June (37 days)	15 Jul (62 days)
8 Aug 66	17 Sep (40)	1 Nov (85)
15 Nov 66	15 Jan (61)	20 Mar (125)
1 May 67	22 Jun (52)	---
1 May 68	22 Jun (52)	26 Jul (86)
31 Dec 68	24 Feb (55)	2 Apr (93)
9 Apr 69	20 May (41)	27 Jun (79)
27 May 70	28 Jun (32)	4 Aug (69)
14 Jan 72	15 Mar (61)	12 May (119)
11 May 72	22 Jun (42)	26 Jul (76)
25 May 72	25 Jun (31)	27 Jul (63)
19 Dec 72	8 Feb (51)	23 Mar (94)
26 Feb 76	---	6 May (70)
7 Dec 76	---	7 Mar (90)
12 May 77	---	30 Jul (79)

Harvesting has usually been conducted very early; slow-maturing plants and ears will have been excluded in the development of the population. The dates of harvest above are very inconsistent with respect to maturity, however.

Self-pollinated progenies tend to be quite uniform within the progeny, both in flowering and in plant habit; the Gaspé form (low-eared) is no longer evident in the populations or progenies. Purple aleurone and purple plant variations are present in the population. Samples of MESS are available upon request.

E. H. Coe, Jr.

White pollen

Plants with white pollen were segregating in a 1976 progeny from a self of +/c2 R-r/r-r (background K55 inbred; b pl P-WW); the colorless aleurone class had been planted. Two plants had white pollen and green anthers; the other plants had yellow pollen and green anthers or yellow pollen and red anthers. Pollinations with white pollen failed to yield seed, and only a few tests established the constitutions of some of the other plants with regard to c2 and r-r.

Subsequent genetic analysis has established that a recessive factor (designated whp), in combination with c2, determines white pollen. No effect of whp on aleurone or anther color is evident; other factors of flavonoid biosynthesis have not been implicated by any observations to date, although C2-Idf apparently is equivalent to c2. The genetic data and interpretations are presented in the following.

From the original source, +/c2 R-r/r-r +/whp selfed, in two plantings, the colorless class gave 17 yellow pollen-red anthered:17 yellow-green:4 white-green (expected for 3:3:1 is 16.3:16.3:5.4). A self of one of the 17 yellow-red plants gave 17 yellow-red:8 yellow-green:2 white-green (expected for 12:3:1 from +/c2 r-r/r-r +/whp is 21:5.2:1.7); a self of one of the yellow-green plants gave 19 yellow-green (from c2/c2 R-r/r-r +/+). An F2 progeny from a cross of a white pollen plant (c2 r-r whp) by inbred Mo17 (+ r-g +) gave 25 yellow:2 white expected for 15:1 is 25.3:1.7). In an F2 from a cross of a white-green plant (c2 R-r whp) by an unrelated male (+ R-g +), the colored class (3/4) gave 7 yellow-red:4 yellow-green and the colorless class (1/4) gave 7 yellow-red:5 yellow-green with no white pollen plants (1.7 would be expected). In two F2 progenies from crosses of white-green plants (c2 R-r whp) by unrelated males (+ R-r +), the colored aleurone class (3/4) gave 36 yellow-red and the colorless class (1/4) gave 28 yellow-green and 5 white-green (expect 24.8:8.2).

Certain of the progenies are consistent with whp/whp constitution. In an F2 from a cross of a white-green plant (c2 r-r whp) by a yellow-red sibling (+ R-r whp), the colored class (9/16) gave 5 yellow-red; the colorless class (7/16) gave 3 yellow-red and 11 white-green (expect 6:8). In a self from one of the preceding 5 colored-yellow-red, segregating 3 colored:1 colorless, colored seeds gave 22 yellow-red; colorless seeds gave 12 white-green. A self of one of the above 3 colorless-yellow-red gave 10 yellow-red:4 white-green (expect 10.5:3.5 from +/c2 r-r/r-r whp/whp). Thus it is possible, from +/c2 R/R whp/whp sources, to plant pure stands of white-pollen plants from colorless seeds, as in the next-to-last progeny described above.

White pollen is creamy white in bulk when fresh (more striking on a kraft surface than on a white one), turning buff as it dries. Under low power the grains are starch-filled but nearly white. Plants homozygous for whp and heterozygous for c2 have light yellow pollen, consistent with the dosage effects of c2 in other tissues. Neither +/c2 whp/whp nor c2/c2 +/whp plants show pollen segregation, however, so the trait is sporophytic. Numerous pollinations have been

attempted with white pollen, but few to no seeds have been obtained; progeny tests are in progress on these exceptions. No flavonols or flavones appear to be present in white pollen, according to preliminary studies by Susan McCormick (Dept. of Botany, University of Texas).

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The pattern of striping in iojap plants

Whereas seedling striping that is due to nuclear events (e.g., wd with unstable ring Wd) is uniform and random, that occurring in ij seedlings is conspicuously not uniform and not random. The first leaf tends to have a regular pattern in which the opposite margins are symmetrically white, with little or no striping elsewhere in the leaf. The second leaf often shows the inverse pattern, but also often repeats the first-leaf pattern; subsequent leaves may continue patterns leaf-after-leaf, or may change, but the impression to the eye is that margin patterns are most common (in some backgrounds the rule) throughout the life of the plant. We are seeking an explanation of the development of such patterns, which contrast with the well-defined sequence of cellular morphogenesis. No doubt they reflect aspects of chloroplast transmission, variation and maturation in concert with cell and tissue proliferation (studies described in a current manuscript by Walbot and Coe show that iojap conditions a programmed change in the ability of the plastid to differentiate, without notable changes in the chloroplast genome).

To examine the relationships of patterns and sectors in Wd ring and iojap seedlings, we have recorded white vs. green in successive millimeters across the width of the first and second leaves at their midpoint. The average percent of millimeters white across the width ("Area"), in the edge millimeter ("Margins"), adjacent to the midrib ("Midribs"), and in the center of the half-leaf ("Centers") gives numerical estimates for the visual impression:

	Obs.	Area		Margins		Midribs		Centers	
	No.	Leaf 1	Leaf 2	Leaf 1	Leaf 2	Leaf 1	Leaf 2	Leaf 1	Leaf 2
wd	121	12.8	14.8	18.6	14.9	10.7	16.5	14.0	18.2
ij	2780	28.5	20.4	67.9	33.6	14.1	29.4	15.8	20.6

As expected from visual impressions, ij seedlings have white margins on leaf 1 twice as often as on leaf 2, and the inverse is true for white midribs. The Wd ring events, however, show the same tendencies, though not to the same degree. A larger sample has not been grown because the backgrounds are not very similar between the Wd ring and ij sources; in fact, the above ij data are pooled from very variable progenies segregating in F2 or BC1 with inbred lines. Appropriate data will be possible to obtain when uniform comparable materials are derived.

The patterns of symmetry across leaves (margin = margin) and from leaf 1 to leaf 2 can be evaluated from the frequency of agreement vs. disagreement in phenotype of the segments in each seedling. For example, the opposite margins of leaf 1 may agree in phenotype; if leaf 2 is the inverse, then margins of leaf 1 and leaf 2 will disagree, while if leaf 2 is the same as leaf 1, they will agree. If the course of cellular morphogenesis is followed, in which the midrib is in the plane of the first vertical division in the formation of the embryo (D. M. Steffensen, Am. J. Bot. 55:354, 1968), then the margin of the first leaf should agree with the midrib of the second on the same side of that plane, and vice versa.

A few rough numbers have been collected to examine these agree/disagree alternatives, with the same reservations mentioned above regarding uniformity and comparability of the materials:

	Leaf 1 Margins		Leaf 2 Margins		Leaf 1 & 2 Margins		Margins & Midribs in the Same Plane	
	Agree	Disagree	Agree	Disagree	Agree	Disagree	Agree	Disagree
wd	61	62	75	49	274	237	274	217
ij	39	14	51	2	112	100	260	153

Iojap seedlings definitely show symmetry in both leaf 1 and leaf 2, as expected. However, it does not appear that the symmetry pattern of leaf 1 is reflected in leaf 2 of the same seedlings, since these margins neither strongly disagree nor strongly agree. Cellular morphogenetic lineages also may be reflected in iojap seedlings, since the agreement between margins and midribs in leaves 1 and 2 in the same plane is equal to or greater than that for Wd losses. These observations suggest that iojap striping in seedlings is not simply a sorting-out phenomenon, nor a random mutational event at the cell level.

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C-I and light

The C-I allele exhibits a dosage effect. McClintock (Carnegie Yearbook 50:174-181) showed that extra doses of C could overcome the inhibitory action of C-I. Kyle and Styles (MNL 47:181-83) showed that immature seeds competent genetically for pigment synthesis, removed from the ear and placed in germinating conditions, showed increased rates of pigment synthesis and developed more pigment if the seeds were germinated in the light rather than in the dark. Thus it seemed possible that the C allele could be given a competitive boost against C-I action if mature seeds were germinated in the light. The results are shown in Table 1; G86 has the other factors (besides C) required for scutellum pigmentation. Light is effective in boosting the C allele expression with respect to the C-I allele. This is seen most strikingly when the pigment levels in the scutellum and endosperm of G85 x G86 are compared. The endosperm of these seeds is only faintly colored (+), whereas the scutellum is darkly pigmented; this is probably due to the fact that the dosage ratio of C-I to C in the scutellum is 1:1, whereas in the endosperm the ratio is 2:1. These seeds develop no more pigment if germinated in the dark.

Table 1: Anthocyanin levels in seeds of various C-I and C allelic combinations, after germination under light.

	Aleurone			Scutellum	
	(+)	(+)	(-)	(+)	(-)
G159 @ (C Wx/C-I wx)					
wx seeds (probably C-I/C-I/C-I)			5		
Wx seeds with colored spots (probably C/C/C-I)	9				
Wx seeds with no spots (probably C-I/C-I/C)		5			
G85 @ C-I			15		15
G85 x G86 C-I x C		22	2	20	5
G86 x G85 C x C-I	19			18	1

(+) is fully pigmented, (+) is pale pigmentation, and (-) is colorless. The number of seeds tested is given for each genotype.

The c-p allele is apparently not equivalent to C in competitive ability against the action of C-I. When c-p/c-p/C-I seeds are germinated under light a few pigmented spots become visible (C-I somatic losses) but no additional pigment forms.

Sheila McCormick

c-m2 and light

The c-m2 allele is under the control of the Ac-Ds controlling element system. The c-m2 mutational events are most unusual (McClintock, Carnegie Yearbook 47:155-69, 1948). This allele produces mutations of at least two qualitative types (distinguished by their ability of cross-feeding the other type); within each type there is a series of mutants showing quantitative differences in pigment intensities. These quantitative differences (as observed in c/c/c-m2 seeds) range from very faint pigment (less than that produced with one dose of C) to very dark pigment (more than that produced with three doses of C). These quantitative differences were often seen as twin spots of darkly pigmented cells adjacent to pale cells (McClintock, PNAS 36:344-55, 1950). In the process of testing for c-p type expression among mutable c sources, I found that when c-n Ac x c-m2 ac or c-m2 Ac x c-n ac seeds were germinated under light sectors of light-inducible pigment became apparent. These areas of light-induced pigment were often seen as part of a twin spot with an already pigmented (c-m to C) sector.

Sheila McCormick

Pigment development on immature c-p ears

Chen and Coe reported (1977) that c-p seeds required both light and germination conditions to develop anthocyanin pigment. They also reported that immature c-p seeds did not develop pigment during maturation if exposed to light, but that the seed could "store" the light signal and use it later to develop anthocyanin when germinated in the dark. However, immature c-p seeds do not strictly require germination conditions to form pigment when removed from the ear. Strong anthocyanin pigment formed with seeds 30 days after pollination, removed from the ear and allowed to dry for 48 hours under light. Thus some condition present when the seeds are on the ear apparently prevents the light signal from being utilized by the c-p seeds.

In the 1978 greenhouse, several c-p (W22) self-pollinated ears were exposed to light, with the purpose of obtaining material which had "stored" a light signal. Surprisingly, these seeds developed pigment while still on the plant, and in distinct morphogenetic patterns. Three major types of events were observed: 1) crown pigmentation--pigment on the crown of the kernel, ranging in intensity from a pale blush of pigment up to fairly dark pigmentation; 2) pigment bands--a band of pigment on the lateral faces of the kernel, at the crown-base interface and 3) pigment clones--pigmented areas that resembled the clonal losses observed when the C-I allele is lost from C-I/C/C aleurone. These events occurred most often on the side of the ear most exposed to the light (i.e., the side opposite stalk insertion) and were more frequent and more darkly pigmented on ears that had been exposed early (18-25 DAP) than on ears that were exposed to light later in development (30-33 DAP). Some seeds showed all three types of events. If both bands and crown pigment were found on the same seed, there was usually a colorless zone between the crown pigment and the banded region. The clones are sometimes quite large with irregular shapes; the smaller clones are very similar in appearance to the C-I loss clones, being rectangular or square in outline. Clonal areas superimposed on pale crown pigment sometimes appeared to be twin spots composed of a colorless and more darkly pigmented region.

The areas of pigment that developed on the c-p(W22) ears in the greenhouse 1978 season were not observed until after 30 DAP, whereas C(W22) seeds are fully pigmented by 18 DAP. Thus if the physiological conditions are altered in certain regions of the seed so that these areas are no longer restricted (in whatever manner) from using the light signal, then the crown pigmentation and the banded pigment patterns can perhaps be explained. It is more difficult to explain the clonal regions of pigment, especially since some of these clones appeared to be twin spots. Twin spots are usually explained by somatic crossing-over or non-disjunction of chromosomal segments; thus the occurrence of these clonal regions may be related to the genetic organization of the C locus. For example, if the C locus is composed of a series of genetic units and a certain number of these units are required for c-p-like expression and a larger number of these units are required for C-like expression, then somatic crossing-over could yield sectors that have acquired enough units to develop pigment on the ear when given a light signal. It is also possible that the sectors are due to somatic loss of an inhibitor component at the c-p allele. For example, if the c-p allelic structure is c-p(I), with the (I) component preventing utilization of the light signal while the seed is still on the ear, then c-p(-)/c-p(I)/c-p(I) seeds might be able to develop pigment on the ear, due to the reduced dosage of the hypothetical (I) element.

Sheila McCormick

Aleurone pigment and plant color factors

Seeds that have all dominant factors for anthocyanin pigmentation in the aleurone sometimes do not form pigment if the plant also carries certain B and Pl alleles. The basal portion of these "shaded" ears does not develop pigment in the aleurone. The progeny in one family (E125) of E. H. Coe's material that segregated several ears with the "shaded" expression were tabulated. Of 5 B-w Pl (weak purple) plants, one showed a "shaded" phenotype; of 23 B Pl plants, 11 showed this expression; none of the 5 B' Pl plants or the 6 B pl plants show this phenotype. The tendency for plants with colored husks to show this phenotype is consistent with the hypothesis that some amount of light is required to induce pigmentation even in seeds with all dominant factors for aleurone pigment present. Possibly several layers of purple husks can screen the transmitted light so that the basal portion of the ear does not receive this required light. When colorless seeds from the basal portion of a B Pl "shaded" ear were germinated in the dark for 48 hours only a diffuse pale pigment developed; germination under light was required for development of full purple pigment in these seeds. The explanation for the "shaded" ears phenotype cannot be as simple as this, however, because many B Pl plants do not have "shaded" ears. Furthermore, plants with green husks that are wrapped in 2 layers of aluminum foil to exclude light can still develop purple pigment in the aleurone.

Sheila McCormick

Light inducibility of anthocyanins in vp aleurone

Homozygous vp suppresses anthocyanin pigment in the aleurone. Dooner and Nelson (Biochem. Genet. 15:509, 1977) suggested that the Vp locus is regulatory for the anthocyanin pathway because vp Bz tissue shows reduced levels of the Bz-coded glucosyl transferase activity.

Chen and Coe (Biochem. Genet. 15:333, 1977) reported that c-p vp seeds were able to synthesize pigment on the ear when exposed to light. This interaction is unusual in view of the observations that: 1) c-p Vp seeds are unable to form anthocyanins if supplied light while still on the plant, and 2) C vp seeds are

suppressed in anthocyanin synthesis. The first observation suggests that the Vp allele inhibits the utilization of the light signal by the c-p allele, and is thus antagonistic to pigment formation, while the second observation suggests that the Vp allele is required for pigment formation. A partial explanation of these interactions was suggested when it was noticed that a self-pollinated ear of C/C Vp/vp constitution had a few faintly pigmented vp seeds on a region of the ear that had accidentally been exposed to light during maturation. Further tests demonstrated that the vp suppression of anthocyanin synthesis in C seeds can be overcome with light exposure.

Dormant seeds from a C/C Vp/vp self-pollinated ear (some of which would again be heterozygous Vp/vp) were planted in the greenhouse in the 1977-1978 season and self-pollinated. At 14 days after pollination (DAP) some of the ears were stripped of husks and covered with plastic bags, to allow light exposure. From 15 DAP (when pigment first appears in this strain) until 40 DAP a record was kept of the time of pigmentation of the individual seeds of the ear and also of the viviparous seed segregation, which becomes apparent at approximately 30 DAP. Other ears in the family were left covered until harvest. Homozygous c-p vp and C vp seeds were stored in the cold after the greenhouse harvest and transplanted from the ear (where they were prematurely germinating) to the field in the summer, 1978. These plants were self-pollinated and the ears exposed to light on the plant, or left covered until harvest.

All of the seeds on light-exposed ears developed pigment in the aleurone layer by 30 DAP. The viviparous seeds on ears not exposed to light remained colorless. Most seeds on the segregating ears developed some pigment within the first few days after removing the husks. It might be expected that those seeds that will have (after 30 DAP) viviparous embryos would be late in developing pigment, since light is required for pigment formation. However, there was no obvious correlation between the time of pigment formation and the vivipary of the embryo; some seeds that pigmented on the first day were viviparous. All the seeds on the C vp ears were viviparous, whether they were exposed to light or not. Thus light has no "corrective" effect on the vivipary of the embryo, but only on the anthocyanin phenotype of the aleurone.

In order to explain the pleiotropic effect of vp, Dooner and Nelson suggested that Vp is a structural or regulatory gene directly involved in the anthocyanin pathway as well as in the synthesis of some dormancy factor other than abscisic acid. If this is so, then the light inducibility of vp could be explained by the light signal allowing a bypass of the requirement for Vp action. Possibly this light-induced shunt would open the pathway after the step where an intermediate is used for the synthesis of a dormancy factor, since the seeds are still viviparous. However, to explain the c-p vp interaction a requirement that the Vp product (or a derivative) is inhibitory to the use of the light signal by c-p aleurone would have to be invoked. The absence of this product (perhaps the putative dormancy factor) in vp tissue would allow c-p seeds to form pigment on the ear when light is supplied. Thus the light signal is coincidentally required to induce pigment synthesis and to open the shunt to allow pigment synthesis in c-p vp aleurone.

Dooner and Nelson (1977) suggested coumarin (derived from cinnamic acid) or naringenin (a flavanone) as possible dormancy factors. It is unlikely that naringenin is a dormancy factor in maize. Fresh or dormant c2 aleurone tissue is blocked in anthocyanin synthesis but can use exogenous naringenin to synthesize anthocyanin pigment (McCormick, 1978, Biochem. Genet. 16:777). If naringenin was a dormancy factor, c2 seeds might be expected to be viviparous, but they are not. The possibility of course remains that coumarins or other compounds related biosynthetically to the anthocyanins could be dormancy factors in maize.

Further tests will be necessary before an adequate explanation of the C and Vp interaction can be made. Whatever the molecular mechanism(s), it is clear that anthocyanin synthesis in vp aleurone is light-inducible, and it is plausible to suppose that this light-inducible step is at a point other than the c-p light-inducibility. Thus the vp light-inducibility supplies another "handle" for examination of the correlation between the genetic and physiological effects of pigment synthesis. Preliminary data suggest that certain pattern alleles at the R locus (e.g. R-nj) also show light inducibility.

It is of interest that the three purported regulatory genes (C, R, and Vp) all have alleles that are light-inducible for anthocyanin synthesis, whereas no alleles of the structural genes are light-inducible. If the C, R, and Vp loci do control the functions of the structural genes of the pathway then the light-inducible alleles of these loci will be useful in a practical sense for future molecular analyses with this system, because the turn-on of the structural genes can be precisely controlled with light.

Although it is premature to speculate on the molecular organization of the C locus, the following non-molecular model is proposed to account for the data. The data suggest that a quantitative variation distinguishes the C, c-p and c-n alleles from each other; the C-I allele does not fit in this quantitative series and is apparently an active antagonist of anthocyanin formation. If one assumes that the C locus is composed of two "cistrons," one with I function (the antagonistic effect) and one with C function (the activation of anthocyanin formation), then the following allelic compositions can explain the interactions at the C locus. The C-I allele has the antagonistic function but not the activation function, and thus is (Ic); the C allele has no antagonistic function, but has the activation function, and thus is (iC); the c-p allele has both the antagonistic function and the activation function, and thus is (IC); and the c-n allele has neither the antagonistic function nor the activation function and thus is (ic). Then in triploid aleurone tissue the constitution of c-p/c-p/c-p seeds is (IC)/(IC)/(IC); these seeds require light to give the activation component a competitive boost against the three doses of the antagonistic component. Colorless C-I/C/C seeds would have the constitution (Ic)/(iC)/(iC); if one further assumes that the antagonistic function is more efficient than the activation function the C-I/C/C seeds would require the action of light to out-compete the antagonistic function of the one (I) dose. Unique results of experiments designed to test these compositions at the recombinational and mutational level can be predicted. Further compoundness of the C component "cistron" would probably be required to explain the c-m2 data.

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Effect of chromosome arm dosage on the activity of isolated maize chloroplasts

Mutants in algae and flowering plants have proven to be valuable tools for the study of chloroplast structure, function, and assembly. The disadvantage of working with most higher plant photosynthetic mutants is that they are often pigment deficient, possess fragile plastids that are difficult to isolate, and are often seedling lethal. For these reasons we are currently studying the effect of segmental aneuploidy on the activity and structure of the chloroplast. This system provides the opportunity to study genetic alterations in photosynthesis in

green, non-lethal material. Additionally, one might expect that changes of arm dosage might be correlated with increases as well as decreases in chloroplast activity. Support for the general hypothesis that aneuploidy may influence enzymatic properties comes from the work of Birchler (MGCNL 51:13-17), who found that increases in the activities of certain soluble enzymes could be correlated with changes in the number of doses of the long arm of chromosome 1.

Hypoploid and hyperploid material was generated by crossing quarter sterile (simple translocations) or semisterile (compound translocations) plants from highly backcrossed B-A translocation bearing stocks onto arm testers. Euploid control material was generated by crossing fertile sibs or the recurrent inbred onto the same tester lines. In order to test whether the markers used to identify dosage had an independent effect on photosynthesis, some tester material was selfed to recover the marker. Suitable substitutes were made in cases of failure or when insufficient material was available. Dosage series (1 vs. 2 vs. 3 doses) for several different chromosome arms were generated in this way; the preliminary nature of this report precludes a complete listing.

We were initially interested in studying those arms carrying hcf loci (see reports of Leto and Miles) known to be essential for normal photosynthetic activity. Preliminary observations for chromosomes 1L (location of several hcf loci), chromosome 1S (location of hcf*-3) and chromosome 3S (location of hcf*-19YG) are given below:

Chromosome arm	Translocation	Seedling Genotype or Phenotype	Dose	PS-II (% euploid control)	Whole Chain (% euploid control)
1S	TB-1Sb	ct2/-	1	100	-
		+/+ or +/markers	2	100	100
		vp5/+/+	3	98	104
		ct2/ct2	2		
			marker control	105	-
3S	TB-3Sb	d-tn/-	1	104	100
		+/+ or +/markers	2	100	100
		cl/+/+	3	104	105
1L	TB-1La	narrow leaf	1	87	55
		bz/+	2	100	100
		bz/+/+	3	95	106

*Electron transport activities at saturating light intensities; PS-II=H_2O--DAD
Whole chain = H_2O--methyl viologen

Although chromosome arms 1S and 3S are known to carry loci important in the assembly and function of photosystem II, there appear to be no effects of arm dosage on photosystemII-dependent electron transport. Seedlings hypoploid for the long arm of chromosome 1 (carrying several hcf loci) show a small decrease in photosystem II activity but a significant decrease in whole chain electron transport relative to euploid control. This suggests that the major alteration in electron transport in these hypoploids probably involves either photosystem I itself or a portion of the electron transport chain connecting the two photosystems. Measurement of P-700, the reaction center pigment of photosystem I, indicates a 33%-50% decrease in P-700 on a per chlorophyll basis in hypoploid material relative to euploid control.

Preliminary electrophoretic data show an increased staining intensity of a lamellar polypeptide with an apparent molecular weight of 45kD in material hyperploid for the long arm of chromosome 1 relative to euploid control. The staining intensity of a diffuse band with an apparent molecular weight of 27.5kD is reduced relative to euploid control in material either hyperploid or hyperploid for the long arm of chromosome 9. The significance of these changes is under investigation.

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Arm location and allelism tests among several high fluorescent maize mutants

As reported previously (MGCNL 51:55-59) several photosynthesis mutants selected on the basis of high chlorophyll fluorescence have been located to chromosome arm by crossing with stocks bearing B-A translocations. Below is an amended and expanded list of markers located to date:

Isolate	Uncovering translocation	Arm
hcf*	TB-1Sb-2L4464	1S or 2L
hcf*-2	TB-1La	1L
hcf*-3	TB-1Sb	1S
hcf*-12	TB-1La	1L
hcf*-13	TB-1La	1L
hcf*-15	TB-1Sb-2L4464	1S or 2L
hcf*-19YG	TB-3Sb	3S
hcf*-23	TB-4Sa	4S
hcf*-26	TB-6Sa	6S
hcf*-34	TB-6Lc	6L
hcf*-41	TB-1La	1L
hcf*-46	TB-3La	3L

New additions include arm locations for hcf*-12, hcf*-41 and hcf*-46. Previously, hcf*-2 was found to be uncovered by TB-1La-5S8041 and hcf*-3 was uncovered by TB-1Sb-2L4464; further crossing with simple translocations gave uncovering by TB-1La and TB-1Sb respectively. Repeated crossing of families segregating hcf* and hcf*-15 by known TB-1Sb failed to uncover high fluorescent seedlings, strongly suggesting that these loci are located on chromosome 2L. Some ambiguity remains however, due to the failure to complete sib tests for the hcf marker stocks. Allelism tests among several hcf loci were reported in MGCNL 51:57-59. This list has also been expanded:

Cross	Common property	Allelism	No. Confirming Crosses
+/hcf-9 x +/hcf-3	PS-II mutant missing Q, cyt b-559, 31kD polypeptide	Allelic	3
+/hcf-19 G,YG x +/hcf-3	" " " "	Non-Allelic	3
+/hcf-9 x +/hcf-19 G,YG	" " " "	Non-Allelic	4
+/hcf-6 x +/hcf-2	" " " "	Non-Allelic	3
+/hcf-2 x +/hcf-38	" " " "	Non-Allelic	1
+/hcf-15 x +/hcf	TB-1Sb-2L4464 uncovers	Non-Allelic	1
+/hcf x +/hcf-3	" "	Non-Allelic	3
+/hcf-15 x +/hcf-3	" "	Non-Allelic	1

These findings suggest that mutations at genetically distinct nuclear loci can cause very similar changes at the level of the chloroplast. This is especially evident in the case of the photosystem II mutants listed above, all of which show similar blocks in the function and organization of photosystem II.

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Further characterization of photosystem II mutants

As reported previously (MGCNL 51:55-59) screening for families segregating high fluorescent seedlings led to the identification of mutants blocked specifically in photosystem II. Whole leaves and chloroplasts isolated from these photosystem II mutants have been extensively analyzed and the results are tabulated here:

Characteristic	Wild Type	Fully blocked		Partially blocked
		hcf*-3[1]	hcf*-19G	hcf*-19YG
Pigmentation[2]	Green	Green	Yellow-Green	Yellow-Green
Fluorescence	Normal	------high, no variable-----		High, reduced variable
PS-II Activity	100%	0	0	40%
C-550	+	-	-	reduced
F695	+	+	+	N.T.[3]
cyt. b-559HP	+	-	-	reduced
cyt. b-559LP	+	-	-	+
31kD polypeptide	+	------greatly reduced-------		reduced
Chsm. arm		1S	?	3S
Allelism		hcf*-3 ≠ hcf*-19G, hcf*-19YG		

[1]Characteristics of an allele, hcf*-9, are identical.

[2]At the three-leaf stage.

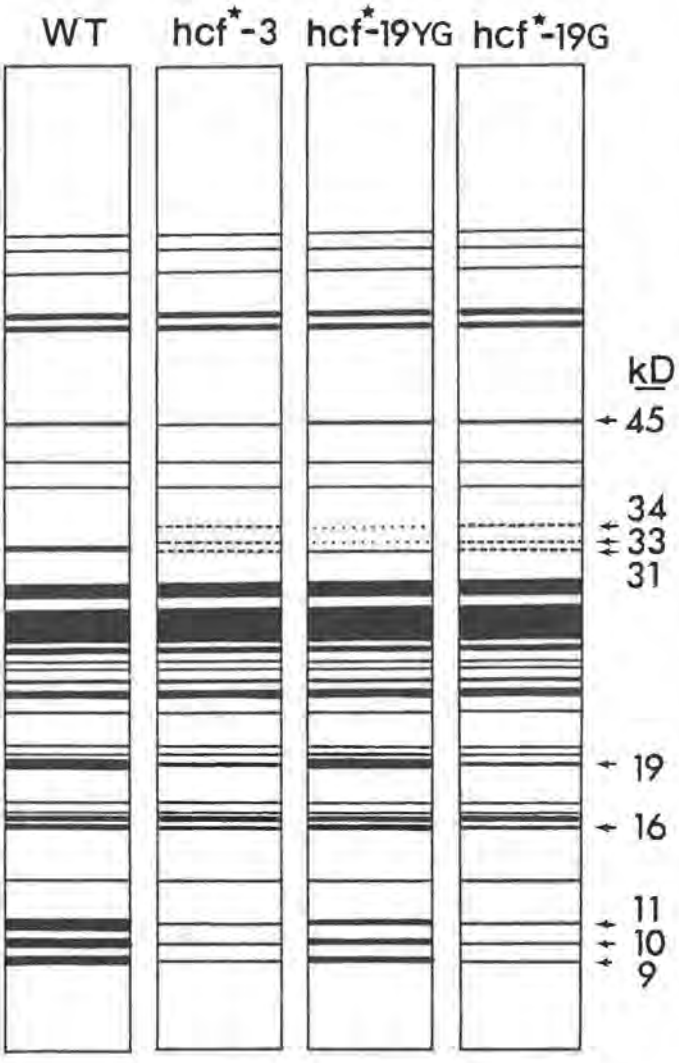
[3]Not tested; presence inferred because of presence of limited PS-II activity.

Seedlings were grown in locally constructed growth chambers to the two or early three leaf stage and mutants identified visually on the basis of high levels of chlorophyll fluorescence. Families segregating seedlings originally designated hcf*-19 were found to segregate two types of mutants. One of these mutants was fully blocked in photosystem II activity and emerged as a green seedling (hcf*-19G) while the second type was partially blocked in photosynthesis and emerged as a yellow green seedling (hcf-19YG). Both mutants were typically yellow green at the three leaf stage. The photosystem II mutants therefore fell into two classes, those fully blocked in photosystem II activity (hcf*-3, hcf*-19G) and one mutant which was partially blocked in photosystem II (hcf*-19YG). Chloroplasts isolated from the fully blocked mutants were unable to carry out photosystem II dependent electron transport while the partially blocked mutant hcf*-19YG retained a limited capacity for photosystem II dependent electron transport.

Photosystem II can be roughly divided into two "sides," an electron acceptor side (with unknown component Q as primary acceptor) and a donor side which terminates with a photoactive pigment, P680. In order to determine which side of photosystem II was affected by the mutational blocks signals from the primary acceptor (C-550 and variable fluorescence) and from the photoactive pigment (F695) were monitored. As shown above the acceptor signals were absent (or reduced) in these mutants while the signal from the photoactive reaction center pigments (F695) was present. This suggests that the lesions cause the loss (or inactivation) of the primary acceptor of photosystem II. This loss is only partial in the case of the "leaky" mutant hcf*-19YG.

In algae the mutational loss of photosystem II activity has been correlated with the loss of cytochrome b-559, a component which may play a role in the transfer of electrons between photosystem II and photosystem I. In isolated chloroplasts the cytochrome exists in at least two forms, a high potential form (b-559HP) and a low potential form (b-559LP). Both forms of the cytochrome are greatly reduced or absent in the fully blocked mutants hcf*-3 and hcf*-19G. In a preliminary report (MGCNL 51:57-59) hcf*-3 was described as having lost only the high potential form of the cytochrome; further tests revealed the loss of both forms. Chloroplasts isolated from the partially blocked mutant hcf*-19YG showed a partial loss of the high potential form of the cytochrome and a nearly normal level of the low potential form. These findings are consistent with the observations made earlier in algal photosystem II mutants where the loss of cytochrome b-559 is considered to be a secondary effect of the loss of photosystem II integrity.

Fig.1



Gradient slab polyacrylamide gel electrophoresis of SDS solubilized lamellar polypeptides suggests that the loss of photosystem II activity is correlated with the loss of a major lamellar polypeptide with an apparent molecular weight of 31kD (kilodaltons; Figure 1). This loss is nearly complete in the case of the fully blocked mutants and is intermediate in extent in the partially blocked mutant hcf*-19YG. This polypeptide is probably not cytochrome b-559 itself, since the molecular weight of the protein moiety of the cytochrome on SDS gels has been reported to be about 5600 daltons. The loss of the 31kD polypeptide is accompanied by the appearance of two minor polypeptides with apparent molecular weights of 33kD and 34kD. Additionally the staining intensity of polypeptides with apparent molecular weights of 45kD, 19kD, 16kD, 11kD, 10kD and 9kD is slightly reduced relative to wild type in the fully blocked mutants.

The major change however is the nearly complete loss of the 31kD polypeptide in the fully blocked mutants. The fact that the amount of this polypeptide present in lamellae correlates well with the extent of photosystem II competence suggests that this polypeptide is in some way necessary for the proper function of photosystem II.

Recently Bedbrook et al. (PNAS 75:3060-3064) suggested that the prominent 32kD polypeptide (most probably our 31kD polypeptide) is synthesized on chloroplast DNA Bam fragment 8 as a 34.5kD precursor which is subsequently cleaved by cytoplasmic products to yield the membrane bound 32kD polypeptide. Possibly then the nuclear mutants reported here are involved in regulating the post transcriptional modification of this protein. The observation that the loss of the 31kD polypeptide is accompanied by the appearance of minor species with slightly higher molecular weights is at least suggestive of a product-precursor relationship (this work was supported by NSF grant PCM 76-08831 to DM).

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Diallel analysis of the variable expression of R-nj phenotypes

In working with selections from the variety Cudu we observed that some selections produced different patterns of pigment formation. Some selections developed only small colored areas at the tip of the kernel while others produced pigment covering the entire crown and perhaps 50% of the surface area of the kernel.

A number of S2 plants selected from Cudu for homozygosity for R-nj and the other required color genes were seeded at staggered dates. A diallel among five early maturing inbreds also was seeded at three different dates in a replicated trial. Several ear shoots near silking were bagged in each row of the diallel set at each planting date. On a given date an ear from each plot was pollinated using pollen from a single R-nj/R-nj plant. Several ears were pollinated from different S2 plants within a 3 day period for each plot. A number of ears were obtained for each row at each planting date which exhibited the navajo color pattern. Colored kernels on each ear were visually rated for proportion of the kernel with anthocyanin pigmentation. Each ear was given a numerical rating. The rating scale ranged from 0 for no color to 9 for completely colored.

Table 1. Analysis of variance for 10 early hybrids tested for ability to develop R-nj aleurone pigmentation at three different dates of planting.

Source	df	Mean squares for color rating
Planting date	2	1.471*
Error A	4	0.408
Hybrids	9	27.053**
GCA	4	59.836**
SCA	5	0.586*
Hybrids x dates	18	0.827*
GCA x dates	8	0.727*
SCA x dates	10	0.892*
Error B	54	0.708

*Non-significant and **significant at the 0.01 probability level.

No apparent differences among pollen parents or pollination dates within plots were observed, so all observations within a plot were pooled before computing an analysis of variance (Table 1). The results indicate that differences in color expression among hybrids were due to GCA effects. There were no detectable planting date effects or interactions among planting dates. Color expression ranged from 2.3 for ND474 x CG10 to 6.9 for CO303 x ND363 (Table 2). ND363 had the largest positive GCA effects for color expression while ND474 and CG10 had negative GCA effects (Table 3).

Table 2. Mean values for anthocyanin color ratings for 10 hybrids pooled over three planting dates.

Hybrid	Pedigree	Color rating
NDB564	ND474 x ND363	5.7
NDB575	ND474 x CO303	4.4
NDB582	CO303 x ND363	6.9
NDB715	ND363 x ND302	6.0
NDB751	ND474 x ND302	3.2
NDC203	ND474 x CG10	2.3
NDC204	CO303 x ND302	5.0
NDC208	CO303 x CG10	4.7
NDC217	ND363 x CG10	5.6
NDC221	ND302 x CG10	3.0
	LSD (0.05)	0.8

Table 3. GCA estimates for five inbred parents for anthocyanin color ratings pooled over three planting dates.

Inbred	Color rating
ND474	-1.04
CO303	0.75
ND363	1.82
ND302	-0.48
CG10	-1.05
LSD (0.05)	0.53

These results seem to contradict those of Kumar and Sarkar (MGCNL 52:119, 1978), who concluded that the variations in the R-nj expression are not heritable and are due to developmental conditions. In this study the differences measured were among maternal parents. If the R-nj anthocyanins are synthesized by plastids as Trautman (MGCNL 49:32, 1975) has proposed for other flavonoids then perhaps interactions among nuclear genes and cytoplasmic factors might account for the differences between these results and those of Kumar and Sarkar. Also the genetic background of the R-nj source was different.

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Heterogeneity within the C group of male-sterile cytoplasms

The T, S, and C groups of male-sterile cytoplasms of maize have distinct mitochondrial DNAs, as revealed by restriction endonuclease fragment analyses. Examination within a group has only been attempted with the S group, and no evidence of variation has been found to date. We recently examined five members of the C group, and were able to differentiate three sub-groups, based on mitochondrial DNA differences. Mitochondrial DNA was prepared from cytoplasms C, RB, BB, E, and El Salvador. Restriction enzymes Xho I, Hind III, Bam H1, Eco RI, and Sma I were used. Xho I, Hind III, and Sma I each delineated three groups: Group CI, C; Group CII, RB, BB, and E; and Group CIII, El Salvador. Bam H1 and Eco RI did not distinguish C, RB, BB, or E from each other, but did differentiate El Salvador. The pattern changes were slight between Groups CI and CII, often revealing only one fragment shifts. El Salvador was clearly distinguished, suggesting a distant relationship to C, RB, BB, or E.

No differences have been observed among chloroplast DNAs of these cytoplasms to date.

It is apparent then, that cytoplasmic heterogeneity, as reflected by mitochondrial DNA variation, is present within the C male-sterile group. Since this source of male sterility is currently utilized in hybrid production, these data may identify sources of cytoplasmic diversity potentially useful in minimizing cytoplasmic uniformity.

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Adenylate content and the energy charge as related to seedling growth in inbred strains

ATP level in imbibed seeds of rape, annual ryegrass and crimson clover was shown to correlate with the rate of subsequent seedling growth and presumed to represent a convenient biochemical index of seedling vigour (T. M. Ching, Plant Physiol. 51:

Table 1

		Inbreds			
		Oh 56A	Oh 43	N 6	B 14
Root length, mm: 3-d-old seedlings		62	33	10	18
7-d-old seedlings		211	202	146	146
Air-dry weight of the scutellum of the non-germinated caryopsis, mg		23	17	24	16
Adenylate levels in the scutella, nmole per g air-dry weight	ATP	2640	3710	2930	3400
	ADP	1910	4220	1410	4650
	AMP	1820	1320	1210	1860
	TOTAL	6370	9250	5550	9910
Energy charge (ATP+0.5 ADP)/(ATP+ADP+AMP)		0.56	0.63	0.65	0.58

400, 1973). Therefore we have screened inbred strains for maximal and minimal growth rates of the seedling primary root and compared these values with adenylate levels in the scutella of air-dry kernels in four selected inbreds (Table 1). Rapidly and slowly-growing strains could not be distinguished by their ATP content and the energy charge values, while ADP content was 2-3-fold in the small-scutellum inbreds irrespective of the seedling vigour.

I. V. Zeleneva, E. V. Savostianova and G. G. Giulverdieva

Nitrate reductase activity and stability in inbreds and their crosses

The activity of nitrate reductase (NR) is generally presumed to be a sensitive efficiency parameter and a reliable criterion to select crop genotypes with the highest capacity to utilize fertilizer nitrogen. In our experiments 3-day-old dark-germinated seedlings were transferred to light (22,000 lux, 16 h) and grown on KNOP solution. NR activity was measured *in vitro* in 6 to 9-day-old seedlings. NR activity reached maximal values in 7-day-old plants and 4 hours after the onset of illumination. Genotype-related variability of NR activities was almost 3-fold in inbreds and less than 2-fold in 18 studied crosses and their parental inbred strains (Table 2). The enzyme activity of any cross under study never exceeded that of its parental inbred strain with the highest NR activity. Significant variability among the genotypes seems to suggest gene-controlled rates of NR accumulation.

Table 2

SV	df	Mean square	F
All 27 inbreds,			
range of NR activity 126–310 nmole per min per g fresh weight			
Between genotypes	26	13 850	
Residual	145	451	31*
Total	171		
18 hybrids,			
range of NR activity 132-248 nmole per min per g fresh weight			
Between genotypes	17	5 912	
Residual	58	96	6.1*
Total	75		
9 respective inbreds,			
range of NR activity 132-258 nmole per min per g fresh weight			
Between genotypes	8	19 925	
Residual	61	739	27*
Total	69		

* Significant at the 0.1% level

We investigated further to what extent maize genotypes differed in their rates of NR degradation. The rate of NR degradation (inactivation) was estimated *in vitro* at 0 C and 27 C. Two classes of inbreds were easily distinguished by their NR stability values ($t_{1/2}$ either 2 to 6 h or more than 18 h at 0 C). NR stability *in vitro* was not related to neutral (pH 7.6) protease activity: less than 20% variation of the latter was found in the inbred collection. The NR stability and activity values were also not interrelated, therefore these two parameters appeared to be inherited independently. The hybrids usually produced stable NR except in labile x labile crosses.

To estimate the NR activities *in situ* as the net nitrate reduction in intact tissues we measured either the accumulation of protein nitrogen or its enrichment with ^{15}N during three successive 24-h intervals in 6 to 10-day-old seedlings. Two estimates agree perfectly well (r 0.82). To increase variability of NR levels, the experiments were performed using several genotypes. In the first series of

experiments (inbreds M14, WF9, Oh43, Oh45 and crosses WF9 x Oh43, WF9 x Oh45, M14 x Oh45) protein accumulation values (^{15}N method) taken for the combined data were in highly significant correlation with the NR activity values (df 25, r 0.87). The extension of the sample size in the second series of experiments provided a means to analyze such correlation separately for individual genotypes (Table 3).

Table 3

Genotypes	Shoots		Roots	
	df	r	df	r
A 344	7	0.75	7	0.91
WF 9	21	0.60	20	0.67
Oh 56A	6	0.72	6	0.77
M 14	10	-0.17*	10	-0.64*
A 344 x WF 9	10	0.95	9	0.78
M 14 x WF 9	7	0.57*	6	0.62*
Total except M 14	59	0.69	56	0.72

* Nonsignificant at the 5% level

Both in the individual strains (except M14 and its hybrid) and in the whole block (the combined data excluding M14) the NR activity in vitro (the NR potential) was significantly in line with the protein nitrogen accumulation (net nitrogen reduction) in the shoots and the roots. The NR potential and net nitrogen reduction were linear functions of shoot and root growth of seedlings. Yet the in vitro/in situ ratio was 10-fold in the shoots and 2-3-fold in the roots. As nitrate levels in the seedlings were shown to be non-limiting, this excess of the NR potential was presumably due either to partial NR inactivation or NADH compartmentation in vivo.

Thus it appears that by measuring NR activities in vitro at the seedling stage we are able to select those genotypes that absorb and reduce more nitrate. Yet without field experiments we cannot relate these data to the final crop yield and protein production.

L. P. Voronova, A. A. Peshkova and E. E. Khavkin

Protein polymorphism in maize and its nearest relatives

Multiple electrophoretic forms of four enzymes were studied by the routine disc-PAGE-zymographical procedure and classified into several phenotypes (see also MNL 51:81, 1977) Fast-allelic forms of alcohol dehydrogenase (ADH) and aspartate aminotransferase (AAT) were predominant among the inbreds and also the most frequent in the collection of 33 races representing most of their groups (see M. Goodman and R. McK. Bird, Econ. Botany 31:204, 1977). The frequencies of glutamate dehydrogenase (GDH) phenotypes were similar in inbreds and races, while succinate dehydrogenase (SDH) patterns differed mostly

Table 4

Enzyme	Phenotype	Frequencies		
		inbreds	races	
			monomorphic	polymorphic
ADH	A	92	58	15
	B	8	27	
AAT	A	90	88	12
	B	10	-	
GDH	A	65	66	-
	B	34	34	-
	C	1	-	-
SDH	A	27	3	30
	B	51	67	
	C	8	-	-
	D	11	-	-
	E	2	-	-

in regard to class A, being more frequent in inbreds than in races. Some of the races--up to 30% in the case of SDH--were polymorphic (Table 4).

Segregation of SDH and GDH patterns was studied in F2 (selfs) and backcrosses. The data obtained in the case of SDH presume: (1) the existence of several non-allelic genes that conforms with the fact that animal SDH consists of two unequal subunits; (2) the allelic control of differences among the phenotypes.

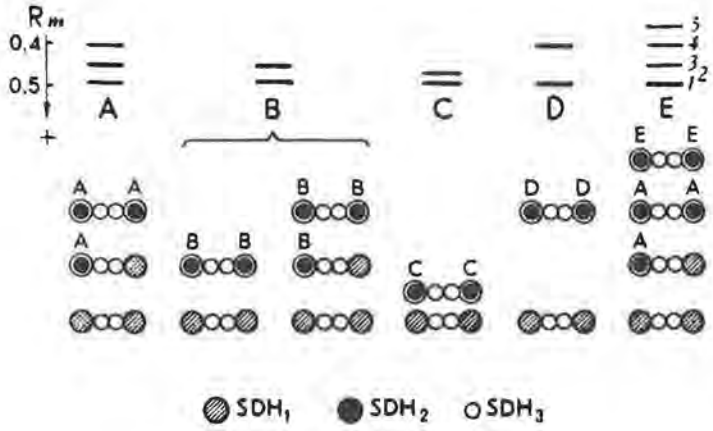

Fig. 1. First model of SDH genetic control. A-E--SDH phenotypes in the inbred collection. Molecular forms 4 and 5 in phenotype E are controlled by a complex locus Sdh2-AE. Two possible variants of genetic control are presented in the case of phenotype B. For other details see text.

Upon these presumptions two alternative models are suggested to describe the genetic control of SDH:

Model 1 (Fig. 1). Three structural genes control the synthesis of polypeptides SDH1, SDH2 and SDH3. Sdh1 and Sdh3 loci are monomorphic, while Sdh2 is represented by several alleles (A-D). The most mobile band 1 is formed as 2 (SDH1·SDH3) and present in all five phenotypes, while other multiple forms are formed as 2 (SDH2·SDH3) and/or SDH1·SDH2·2SDH3.

Model 2. The enzyme is formed by two heteromeric subunits SDH1·SDH3, and the diversity of phenotypes is attributed to SDH1 posttranslational modification under the control of a series of gene-modificator alleles.

In the case of GDH the analysis of the complicated phenotypes in seven-band x two-band crosses presented considerable difficulties, however, clear segregation of the parent phenotypes conformed to the expected frequencies based on the two-loci control presumption (Table 5). Posttranslational modifications of two polypeptides seem to produce the inverted binomial distribution of staining intensity in the seven-band GDH patterns (maximal staining of the 1st and 7th bands) in the scutella, as well as in other organ-specific GDH phenotypes lacking one or two anodal bands. However, the one-band GDH pattern found in Job's tears (Coix lacryma-jobi L.) does not agree with this two-loci model.

Table 5

	Phenotypes			Total
	Parents		Hybrids	
	C 103	WF 9		
C 103 x WF 9	5	9	88	102
WF 9 x C 103	6	8	85	99
(C 103 x WF 9) x C 103	20	-	58	78
(C 103 x WF 9) x WF 9	-	15	48	63

Antigen spectra of reserve globulins in the Andropogoneae were compared by double immunodiffusion and one-dimensional immuno-electrophoresis using antiserum raised against the purified globulin preparation from maize embryos (for details of the procedures see E. E. Khavkin et al., Planta 143:11, 1978).

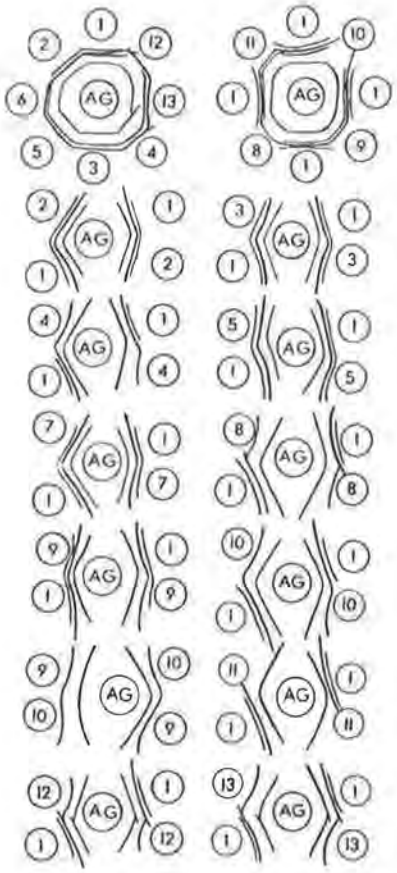

Fig. 2. Comparison of globulins in the Andropogoneae by double immunodiffusion test. AG--antiserum against maize globulin. (1-7) maize: 1--hybrid Bukovinsky (A344 x cv. Gloria Janetzki), (2-7) races Nal-Tel, Reventador, Cuzco, Canguil, Chapolote, Lenha; (8-9) teosinte, Euchlaena mexicana Schr., races Huixta and Chalco; (10,11) gama grass, Tripsacum dactyloides L., 36 and 72n; (12) Job's tears, Coix lacryma-jobi L.; (13) sorgho, Sorghum bicolor (L.) Moench.

Considerable quantitative variations were found when we compared globulin immunodiffusion patterns of six races confined to several geographical areas, however, all the antigens were identical (Fig. 2). Globulins of two teosinte races and two gama grass forms of different ploidy were identical to those of maize though even more polymorphic: teosinte Huixta was low and 36n gama grass almost lacking the slow-migrating globulin component. In Job's tears, sorgho and eulalia (Miscanthus) the same globulin component gave the reaction of partial identity with the respective maize protein. Another globulin was identical to that of maize in all the investigated representatives of the tribe Andropogoneae and partially identical in the Paniceae. Thus, no direct discrimination was found among maize, teosinte and gama grass globulins (see also data by J. W. Paulis and J. S. Wall, J. Agr. Food Chem. 25:265, 1977) to contribute to the current dispute on the evolutionary history of maize.

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Studies on the mechanism of Al response in maize

Work has progressed on our mineral stress studies using the maize composite, "Supermix." We presented data in the last newsletter indicating that aluminum response is primarily determined by a multi-allelic series at a single locus. Among our present objectives is to study the interaction between high aluminum, low calcium and low phosphorus stress response. This should help us better understand the mechanism of aluminum response.

Selections were made last Spring for aluminum, calcium and phosphorus response on 2-week old maize seedlings grown in different nutrient stress solutions. Selection was on the basis of primary root growth for aluminum response and visual deficiency symptoms for calcium and phosphorus response. The most stress-resistant and stress-susceptible seedlings were transplanted to the field and selfed. Eight to fourteen full-sib families were thus obtained for each group. Response of the full-sib family selections compared to response of full-sib randomly chosen controls estimates the selection effect.

Selection for calcium efficiency and inefficiency was not markedly successful. Aluminum and phosphorus selection was quite effective, as demonstrated in the tables below. The data compare seedlings grown for two weeks in stress and non-stress solutions.

Table 1. Effect of selection for Al response.*

	Al stress			No stress		
	Tolerant	Control	Sensitive	Tolerant	Control	Sensitive
Primary root lengths (cm)	22.2a	18.2b	17.7b	39.6	38.6	38.2
Plant height (cm)	36.1a	34.0b	33.5b	46.5a	45.5a	43.0b
Ca deficiency symptoms**	1.25	1.25	1.54	1.56	1.61	1.59
P deficiency symptoms***	2.05	2.21	2.27	1.07	1.11	1.08

*Significant differences between entries are denoted by different letters.
**1 = best, 7 = worst.
***1 = best, 5 = worst.

Table 2. Effect of selection for P response.*

	Low Phosphorus			No stress		
	Efficient	Control	Inefficient	Efficient	Control	Inefficient
P deficiency symptoms**	1.29a	1.82b	2.45c	1.02	1.36	1.64
% P in tops	.347ab	.358b	.327a	------not yet available------		
Plant height (cm)	43.3	41.7	42.0	47.1	47.3	49.0
Root length (cm)	46.0	42.0	44.4	45.5a	37.2b	45.4a

*Significant differences between entries are denoted by different letters.
**1 = best, 5 = worst.

Mineral analysis of both selections is still being completed. We will also be running the aluminum selections under phosphorus and calcium stress, and the phosphorus selections under aluminum and calcium stress.

Finally, other groups selected for differences in root size will be tested under all three stresses to see if root size affects stress response.

E. William Stockmeyer and Herbert L. Everett

Studies on multi-male hybrids

Replicated field tests of single cross and three-way multi-male hybrids and appropriate checks have been harvested. Inbreds CO150, Ayx157, B8, CO192, C153, and NY16 were utilized as males and W182BN, W182BN-PR, W182BN x W117, W182BN-PR x W117 as females in the early maturity group. In the later maturity group, inbreds W64A, Ayx157, NY821LERf, Oh43, NYD410, and Ay490-2A were entered as males and A632, A632-PR, A632 x Oh51A, A632-PR x Oh51A served as females. Additional replicated tests at several locations will be planted in 1979. Data for several years will be used to determine the advantages, if any, of using a multi-male pollen approach to hybrid corn production.

Herbert L. Everett and E. William Stockmeyer

Genetics of cms-C fertility restoration

The genetics of fertility restoration for the cms-C types of cytoplasms has not been extensively studied. Recently we have converted a series of 40 sources of cytoplasmic male sterility into approximately 30 inbred lines and have studied the degree of male sterility (Gracen and Grogan, Agron. J. 66:654-657). Sixteen of the inbreds formed relatively stable male sterile combinations with either the cms-T or cms-C cytoplasms (Table 1). In our initial studies, cytoplasms C and RB

Table 1. Summary of fertility restoration reactions and proposed genotypes of 16 inbred lines for restoration of T and C group male sterile cytoplasms.

Proposed genotype	$T\frac{Rf}{Rf}\ C\frac{Rf}{Rf}$	$T\frac{Rf}{Rf}\ C\frac{rf}{rf}$	$T\frac{rf}{rf}\ C\frac{Rf}{Rf}$	$T\frac{rf}{rf}\ C\frac{rf}{rf}$
T, HA, Q, P and RS	Fertile	Fertile	Sterile	Sterile
C, Bb, ES, PR and RB	Fertile	Sterile	Fertile	Sterile
Inbred lines	NY821LERf	Ay x 187-y-1	A619	Oh51A
			Oh43	W182BN
	Ay x 187-y-2	NYD410	W64A	CO107
			Pa405	SD10
			NY327	R181B
			Ay49W329	A636

comprised the C group. Subsequently we have collected several additional cytoplasms (El Salvador or ES, Bb, and Panama or PR) that seem to fall into the C group. Recently, we have attempted to determine:

(1) the number of restorer genes that interact with "C group" cytoplasms,
(2) whether or not the C, Bb, ES, PR and RB cytoplasms are identical with respect to their partial and full restoration patterns,

(3) the genotypes of various inbred lines for the cms-C restorer genes,
(4) and the nature of any interactions that occur between "C group" restorer genes and those of "T group."

Several crosses have been made and evaluated for degree of male-sterility. Additional progenies are now under study in a Florida winter nursery. Our preliminary results show that restoration of C, RB and PR cytoplasms follows a sporophytic pattern of restoration when the line NY821LERf is used as a source of RF genes (Table 2). The data also indicate that NY821LERF is homozygous dominant for CRf factors.

Also, the data indicate that there is apparently a single, major restorer gene for C, PR & RB cytoplasms segregating in crosses involving NY821LERf and/or Ay49W329 with recessive rf types (Tables 2 and 3). All of the BC_1 progenies involving these two lines give a good fit to 1:1 ratios and the selfed BC_1 progenies (with NY821LERf) give a good fit to a 3:1 ratio. In all of these crosses there are relatively few partially fertile plants and most of these are late breakers.

Table 2. Fertility restoration reactions of C, PR and RB cytoplasms in backcross and selfed backcrossed generations.

				Phenotype					Seg.	
			# Ears	1	2	3	4	5	ratio	X_1^2
1 - C cytoplasm										
W182BN	BC_1		bulk	44	-	7(LB)*	2	56	1:1	1.44
	BC_1	selfed	3	23	-	5(LB)	-	72	3:1	.03
R181B	BC_1		bulk	41	-	3(LB)	-	48	1:1	.55
	BC_1	selfed	2	14	-	2(LB)	-	50	3:1	.33
W182E	BC_1		bulk	36	-	10(LB)	-	49	1:1	1.99
	BC_1	selfed	3	25	-	9(LB)	4	55	3:1	1.67
2 - PR cytoplasm										
W182BN	BC_1		bulk	50	-	2(LB)	-	51	1:1	.01
	BC_1	selfed	3	33		3(LB)	-	88	3:1	.33
R181B	BC_1		bulk	38	-	7(LB)	-	54	1:1	2.78
	BC_1	selfed	3	22		2(LB)	-	76	3:1	.33
W182E	BC_1		bulk	27		13(LB)	3	38	1:1	1.85
	BC_1	selfed	3	11	-	4(LB)	-	50	3:1	1.57
3 - RB cytoplasm										
	BC_1			11	-	-	-	14	1:1	.35

All progenies were rated on the 1-5 scale (1 = sterile) in 1978 at Aurora, N.Y. The crosses studied all involved NY821LERf (CRfRf) x each of 3 inbreds of the Crfrf composition (W182BN, R181B, W182E), with the F_1 backcrossed to the recessive parent (BC_1) and subsequently selfed (BC_1 selfed).

*LB indicates plants that exhibit a "late breaking" of sterility.

The segregation patterns of C0150 (as a restorer line) and C0109 and C0220 (as sterile lines) in C and PR conversions give conflicting data for a one gene segregation pattern. There may be more than one nuclear gene interacting in these crosses to condition partial or full sterility. C0150 gave a good 1:1 ratio with Oh51A-C in 1975 (ear #1 gave 42 MS:47MF). In contrast in 1978 at Aurora nursery, three ears of the same BC_1 gave too many MS plants to fit a 1:1 ratio. The same difficulties are found in C0109 and C0220 crosses where progenies are characterized by too many sterile or partially fertile anthers. Since environment influences the degree of partial fertility expressed, additional studies of these apparent differences from a single gene ratio are needed. A selection program is underway to establish dependable and stable restorer C0109 and C0220 lines in a backcross program of Rf conversion.

In order to facilitate use of the "C group" cytoplasms in hybrid seed production, several widely used inbred lines have been converted to these cytoplasms. Some inbred lines, such as Minn. A632, typically exhibit a partial restoration

Table 3. Fertility restoration reactions of backcrosses involving several inbred lines in C and PR cytoplasms.

	# Ears	Phenotype 1	2	3	4	5	ratio	X_1^2
C cytoplasm:								
NYD10-C x [(Ay49W329xAyx157) x NYD410]	3	138	-	2	-	128	1:1	.38
Oh51A-C x [(" x ") x Oh51A]	8	183	-	35	-	204	1:1	1.14
Oh51A-C x (C0150 x Oh51A)	3	142	-	29	-	75	1:1	20.68***
Oh51A-C x (C0150 x Oh51A)*	1	42	-	-	-	47	1:1	.28
C0109-C x (NY821LERf x C0109)	bulk	31	-	3	-	55	1:1	7.68***
BC_1 selfed	4	43	3	12	8	43	3:1	.00
C0220-C x (NY821LERf x C0220)	bulk	21	3	3	4	69	1:1	25.60***
BC_1 selfed	3	12	0	4	5	69	3:1	6.72**
PR cytoplasm:								
C0220-C x (NY821LERf x C0220)	bulk	19	-	8	8	54	1:1	16.78***
BC_1 selfed	3	22	-	10	21	52	3:1	.88

Each cross involves a line that restores the C cytoplasms (Ay49W329, C0150, NY821LERf) times a line sterile in C cytoplasm (NYD410, Oh51A, C0109, or C0220).

*All evaluations were made using the 1-5 scale (1 = sterile) at Aurora, N.Y. in 1978 except for this entry which was evaluated in 1975.

pattern in these cytoplasmic sources. All A632-C, Bb, ES, PR and RB conversions are partially restored in certain environments. PR cytoplasm appears to be the most stable source of cyto-sterility. A selection program of A632-PR conversions to try to produce a stable, male sterile line is underway. Sixteen individual plants of A632-PR genotype were crossed with individual fertile (maintainer) A632-N plants. The male parents were also selfed. In 1978 in New York, 47 of these individual A632-PR x A632 pair-cross progenies were observed. Table 4 illustrates the performance of 6 of the most sterile and 6 of the most fertile pair-cross progenies. We must point out that there are two distinct classes: (1) plants that did not exsert any anthers during the whole observation period, and (2) plants that exserted from a few anthers to nearly fully fertile tassels. One generation of selection seems to have been efficient in the selection of sterile combinations. Additional crosses were

Table 4. Selection for stable, sterile versions of A632-PR from sterile and maintainer pair crosses (A632-PR x A632-N).

	Ear #	Phenotype 1	2	3	4&5
Most sterile pair crosses	1	7	-	6	5
	2	10	-	4	4
	3	9	-	8	-
	4	7	-	10	5
	5	12	-	5	4
	6	7	-	7	3
Most fertile pair crosses	7	-	-	4	15
	8	-	-	2	18
	9	-	-	5	19
	10	-	-	4	19
	11	-	-	0	23
	12	-	-	2	20

All data were made using the 1-5 scale (1 = sterile) at Aurora, N. Y. in 1978.

made between these sterile A632-PR plants and their sister A632-N rows last summer. Equivalent crosses were made between the most fertile A632-PR plants and their sister A632-N rows. These individual second generation progenies are under observation in a Florida nursery.

A study of environmental effects on partial fertility is also underway in greenhouse and environmentally controlled chambers. A comparative study of environmental effects of Florida and New York field environments, greenhouse and controlled growth chamber conditions will hopefully give us some concept of the role of environment vs. genetic differences for partial restorer genes in C cytoplasm restoration.

A. Kheyr-Pour, V. E. Gracen and H. L. Everett

Fusion of T cytoplasm corn protoplasts with soybean protoplasts confers resistance to Helminthosporium maydis race T toxin.

Studies of the interaction of the cytoplasmic determinants involved in sensitivity to Helminthosporium maydis race T are hampered by the fact that only maternal cytoplasm is transmitted after sexual crosses. Hybrid cytoplasms can, however, be produced by asexual fusion of protoplasts. We have therefore studied the effect of the toxin produced by H. maydis race T (HmT toxin) on fused and unfused protoplasts from plants with different cytoplasms.

HmT toxin has a clearcut cytoplasm-specific effect on cultured protoplasts. Protoplasts from corn plants with T cytoplasm collapse after 1-3 days of exposure to low levels of toxin while protoplasts with N, C, or S cytoplasm are unaffected by long-term treatment with high toxin levels (Plant Physiol. 61:420-424). Survival of protoplasts from oat leaf mesophyll and soybean callus is also unaffected by HmT toxin.

Toxin-sensitive T cytoplasm protoplasts can be distinguished from toxin-resistant protoplasts by visual markers. For example, green T cytoplasm leaf protoplasts look very different from non-green protoplasts isolated from callus or albino leaves. When mixtures of such protoplasts are treated with HmT toxin, the T cytoplasm protoplasts collapse while the others survive. Thus HmT toxin can be used to distinguish and select the toxin-resistant protoplasts in a mixed protoplast population.

Fusion of green T cytoplasm protoplasts with non-green soybean callus protoplasts can be induced by brief treatment with 30% polyethylene glycol (PEG) 6000, followed by rinsing in a high calcium, high pH solution. Fused corn-soybean protoplasts are recognized by the simultaneous presence of large corn chloroplasts and the dense cytoplasm characteristic of the soybean protoplasts. Such corn-soybean fusion products, which have a combination of T and non-T cytoplasm, survive at least 1-2 weeks in toxin levels which rapidly collapse all unfused T protoplasts. Survival of the corn-soybean fusion products in medium ± toxin is comparable. Similar results are seen when green T cytoplasm corn protoplasts are fused with non-green N cytoplasm corn leaf protoplasts from albino plants with the C1 gene. Many of the corn-soybean fusion products change shape (as soybean protoplasts typically do), and several have divided.

HmT toxin causes rapid ultrastructural damage to isolated T (but not N) mitochondria and to mitochondria within T cytoplasm roots and protoplasts (Tiss. & Cell 9:167). We are therefore examining the mitochondrial populations within toxin-treated corn-soybean fusion products. We are also exposing T cytoplasm protoplasts to isolated N mitochondria under conditions that facilitate organelle uptake in hopes of altering the response of treated protoplasts to HmT toxin. These studies may help pinpoint the location of the cytoplasmic genes for sensitivity to HmT toxin.

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Aleuroneless mosaic: A conditional developmental mutant

A conditional mutant of spontaneous origin which prevents differentiation of the aleurone has been recovered. In the affected kernels, the aleurone fails to develop in some regions resulting in a mosaic pattern of aleuroneless patches on the surface of the kernel. The degree of expression is quite variable ranging from kernels on which about 75% of the surface is defective to seeds having, at most, several minute sectors without aleurone cells, or no deficient regions at all.

The patterns of expression on the kernel surface vary considerably also. In some cases, there is only a small region in the center of the crown which is aleuroneless. In others, the abgerminal side may be completely devoid of aleurone with a gradient of increasing numbers of cells over the crown towards the germinal surface. In yet other cases, the opposite occurs with the germinal side deficient and the abgerminal surface normal.

On a given ear, the phenotypic patterns tend to be similar. Although there is latitude in expression, no case has been observed in which kernels having a deficient germinal surface occur on the same ear with those defective on the abgerminal side.

Scanning EM studies of aleuroneless and normal kernels have revealed size and shape differences between the aleurone cells of the two types. The cells on defective kernels are generally larger and more irregular in shape than those on normal ones. These differences may be due to abnormal differentiation of the aleurone cells or simply to the fact that there is less compaction on the defective kernels and hence, more room to expand.

The phenotype first appeared on ears of two unrelated stocks in which the pollen came from sibling plants of a third line; hence, the mutant was carried by the male parent. When it arose is uncertain since it could have been transmitted through a number of generations without the conditions necessary for expression of the phenotype (see below). Since the phenotype was expressed in the heterozygote, either the mutant allele is dominant or dosage effect is involved.

On two of the ears from these crosses, distribution of the phenotype was not random. Kernels with the most extreme expression occurred about an inch above the base and the degree of expression diminished in both directions from that point. Kernels on the distal portion of the ear showed either slight expression or no aleurone deficiencies at all. This distribution parallels the sequence of ovule development. Those about an inch above the base are most advanced with maturation proceeding in both directions.

When aleuroneless mosaic kernels were grown and used as both males and females in crosses with unrelated lines, the phenotype was expressed in the offspring but infrequently. In the progeny of both sexes there were cases in which kernels exhibiting the phenotype were clustered in a particular region of the ear but not necessarily at the oldest point.

In some instances, the phenotype has skipped a generation. When plants derived from normal sibs of aleuroneless kernels were outcrossed to unrelated lines, the phenotype was expressed in some kernels but infrequently.

The clustering of mutant expression at a point along the length of the ear suggests that either ovule age at the time of fertilization, or some condition present when the affected ovules developed but not when others matured, was responsible for appearance of the mutant phenotype. The fact that the aleuroneless

allele was introduced through the male in some of the crosses indicates that the ovule was preconditioned by some factor which allowed expression of the character in the resulting caryopsis. Tests are currently under way to identify the factor(s) responsible for phenotypic expression.

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The evolution of maize: a new model for the early stages

In searching for an explanation of the great morphological and ecological variability of maize, a wide range of hypothetical models have been propounded. Some propose that massive mutation was the major source of new traits, others suggest intergeneric hybridization, while other suggested evolutionary factors are multiple domestications of Zea populations in many places, and long, very effective, divergent breeding by Amerinds. In a new model of maize evolution, facets of each of these play parts, but none is wholly accepted.

It is proposed that the two principal events in maize evolution are its initial domestication and its hybridization with Zea luxurians (Durieu) Bird, the teosinte of Guatemala and Honduras (R. McK. Bird, 1978. Taxon 27:361-363). Perhaps in central, highland Mexico, a small maize-like plant was domesticated before 5000 B.C., principally by selection for firmer rachillae. Its form could have been much like that of the cultivar found at the lowest levels of Coxcatlán Cave and San Marcos Cave near Tehuacán, Mexico, with short glumes and a narrow, solid rachis (Table 1). Its ca. 64 kernels per ear approximated the number of seed on the fruiting branch of Z. luxurians. (One count of a branch at node 14 below the main tassel gives 65 seed--three to six seed per each of ca. 14 spikes, in cv. Florida grown in Columbia, Missouri by Dr. Jack B. Beckett.)

Table 1. Measurements of maize from Mexico and Peru -- six cobs from the Coxcatlán Cave and San Marcos Cave in the area of Tehuacán, Puebla, Mexico, and four cobs, representing four types, from Huaca Prieta excavation HP 5, Chicama Valley, La Libertad, Peru. Dimensions in millimeters.

Identification	Rachis Diam.	Row Number	Cupule Width	Cupule Length[2]	Cupule Wing Width	Rachis Segment Length	Cob Length[1]	Lower Glume Length	Cupule Depth	Lower Glume Thick.	Lower Glume Angle	Rachilla Angle	Type
Coxcatlán phase (3500-5000 B.C.) Zones XI to XIII of Coxcatlan Cave and Zones E and F of San Marcos Cave													
Tc50:J? (bottom)													
W3 13-61	3.4	12	2.4	3.2	0.5	4.3	(30.0)*	3.1	0.9	0.4	90	80	-
Tc254:2-41	3.4	10	2.3	2.3	0.3	3.3	(16.0)	2.8	0.9	0.4?	-	-	-
Tc254:2-41	1.8	4	2.8	2.1	0.6	2.5	(21.3)*	1.7	0.2	0.4	110	80	-
Tc254:3-51	3.5	6	3.7	2.4	0.6	3.1	(21.3)	2.6	1.2	0.5?	105	80	-
Abejas phase (2300-3500 B.C.) Zone VIII to X of Coxcatlan Cave and Zone D of San Marcos Cave													
Tc50:I9-42 S													
S2E12	2.7	4	3.6	1.9	0.4	2.8	(26.7)*	2.0	1.0	0.8?	80	-	-
Tc254:W1-level 1	4.2	10	2.8	2.3	0.5	3.5	41.9	3.0	1.3	0.5?	90	80	-
Cupisnique phase (850-300 B.C.) Layers A1 and A2 and House 2 of excavation HP 5 next to Huaca Prieta													
HP5A1S-3	9.8	20	3.6	1.2	0.5	3.1	(60)*	4.2	0.5	0.2	95	70	CU-1
HP5A2-3	7.5	20	2.5	1.7	0.4	2.5	40	3.4	0.4	0.3	120	80	CU-2
HP5H2V-2	7.2	16	2.8	1.8	0.5	2.9	49	4.1	0.8	0.2	90	80	CU-3
HP5A1-3	7.2	12	2.9	1.4	0.5	2.6	(33)	3.6	0.7	0.3	90	60	CU-4

* Cob length almost complete.
1 Parentheses indicate that cob length is incomplete.
2 Cupule length for Tehuacán specimens is average of two measurements.
3 Cupule wing width for Tehuacán specimens is estimated: (Cupule Width, Exterior - Cupule Width, Internal)/2

The cultivar was spread to Guatemala where it eventually hybridized with Z. luxurians. After considerable backcrossing with maize, a subset of the resulting hybrid swarm may have been taken to South America, where because of important new genes or alleles introgressed from teosinte and a new cultural environment, it evolved into a wide range of new races, especially those in the Central Andean Rotund Flour, Altiplano Small Flint and Amazonian Interlocked Flour complexes (R. McK. Bird and M. M. Goodman, 1978, Econ. Bot. 31:471-481). The earliest maize with good provenience on the coast of Peru--from many sites dating 850-300 B.C.--is very different from any Mexican maize yet described (Table 1). Meanwhile, the hybrid swarm in Guatemala, interacting with the original maize and teosinte, produced yet more types of maize through selection in the various cultural environments of Mesoamerica.

As Longley, Kempton and Popenoe suggested in 1937, annual teosinte of Mexico may be another derivative of this hybridization--the great cytogenetic similarity of Zea mexicana and of Mesoamerican-Caribbean Zea mays could be due to their sharing an ancestral stock. Maize containing a relatively high percentage of teosinte alleles could have been moved to Mexico because it had larger ears, kernels with new colors or some other feature. Because two very different species had contributed to this stock, it may have been difficult to stabilize the new maize races. Odd recombinant progeny could have been produced which contained the alleles necessary to the teosinte form, possibly resulting in weedy populations in old corn fields. These may have evolved to the present Zea mexicana, continually affected by introgression from maize (H. G. Wilkes, 1967, Teosinte: the Closest Relative of Maize).

This model does not account for Zea perennis of west Mexico (R. Guzmán M., 1978, Phytologia 38:177). Perhaps this also is a product of the hybridization and later movement described above, a segregate which became tetraploid, emphasizing the near-perennial character of Z. luxurians.

Archaeologists may provide evidence critical to testing the model by determining the forms and distributions of pre-5000 B.C. maize and teosinte, the time of first Z. mays-Z. luxurians hybridization, etc. Perhaps they will provide further evidence that people spread teosinte, as seems to be the case with teosinte found in the Tamaulipas caves (P. C. Mangelsdorf, R. S. MacNeish and W. C. Galinat, 1967, Bot. Mus. Leafl., Harvard Univ. 22(2):33-63).

Further morphological and chemical systematic studies are needed, while genetic and cytological studies should provide critical information, especially if they are structured to test the several evolutionary models.

Robert McK. Bird

Perennial teosinte: replanting in Jalisco, Mexico

In the summer of 1974 a plant of Zea perennis was placed in the yard of a house near Ciudad Guzmán, Jalisco, Mexico. It had been grown in the greenhouse at CIMMYT from a rooted stem cutting brought from Raleigh, North Carolina. The parent plant has the label number 67-281 and has been maintained by David H. Timothy for many years. It came from material vegetatively reproduced by R. A. Emerson and had been collected by G. N. Collins and J. H. Kempton at the type locality. I took the cutting to CIMMYT to extend their germplasm collection; it grew well and produced seed. One of the several clones cut off it was carried to Jalisco by Ing. Gil Olmos B. who planted it in the near vicinity of a house of a friend. I am obtaining the name and address of that friend from Ing. Olmos for a later note.

Robert McK. Bird

The USDA teosinte germplasm collection

The Department of Agriculture maintains 52 samples of teosinte at the Southern Regional Plant Introduction Station in Experiment (Griffin), Georgia (30202). These represent all the annual species of teosinte and most of the populations. Supplies are limited because the seed are not easily increased--isolation blocks in Puerto Rico are used. In order to help in the selection of samples for study, a listing by population is presented (Table 1), with information taken from the Catalogue of Seed Available at the Southern Regional Plant Introduction Station--Warm Season Grasses (January, 1977) and from the Plant Inventory of the USDA (1965 to 1973).

Table 1. Teosinte samples maintained by the USDA Southern Regional Plant Introduction Station. Revised nomenclature is used here (see H. G. Wilkes, 1977. Econ. Bot. 31:254-293; R. McK. Bird, 1978. Taxon 27:361-363).

Species and Race	Area	Plant Introduction Number
Zea luxurians		
Guatemala	SE Guatemala	306615, -6, -7, 343231
	S Honduras	(343230) only in Plant Inventory, not in the seed bank
Huehuetenango	NW Guatemala	343232, -3, 355921, -2, -3
?	Guatemala	311282, -3
Zea mexicana		
Balsas, SE	EC Guerrero	343237, -8, -9, 384061*, -2
Balsas, C	N Guerrero	331783, -4, 343240, -1, 343245*, 384064 to -074
Balsas, NW	S Michoacan	331785, 343242, -3
Central Plateau	W Mexico	331786, -7, 384063
"	E Michoacan	331788, -9
"	NE Michoacan	343244
"	W & S Guanajuato	343234, -6
Nobogame	S Chihuahua	343249
Chalco	SE Mexico	331779 to -782, 343246, -8
?	Oaxaca	384060

* 343245 is a very maizoid population sample; 384061 is available but is not in the 1977 catalogue.

There are other lists of samples available or once used (H. G. Wilkes, 1967, Teosinte: the Closest Relative of Maize, Tables 1 and 2, Appendix 1; Wilkes, 1972, Pl. Genet. Res. Newsl. 28:3-10; T. A. Kato Y., 1976, Mass. Agr. Exp. Sta. Bull. 635: Table 1). It can be seen that a number of populations or cultivars used in previous studies are missing from the collection (Table 2). It includes only seventeen of the 61 samples studied by Kato.

Table 2. Populations or cultivars lacking from the USDA Southern Regional Plant Introduction Station Catalogue of Seed (1977), by species, area and/or population.

Zea luxurians
- Honduras
- Southeast Guatemala: Lake Retana, Santa Rosa, Moyuta
- Northwest Guatemala: Nojoya
- Cultivars: Florida, El Valle

Zea mexicana
- Chapingo, Durango, Chilpancingo

Zea perennis
- All old samples and the populations newly found by Guzmán (1978). Although listed under PI 302671, *Z. p.* does not seem to be available now.

Anyone who can supply to the USDA the materials in Table 2 is urged to do so, as much seed as possible up to a quarter kilo. In some cases, return of samples in Table 1, if increased carefully, would be welcome (letter from James T. Strickland, Dec. 19, 1978). All accompanying information should be included.

Robert McK. Bird

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Cytoplasmic-genotypic effects in maize inbreds:

Additional studies were conducted with the CI 21 (Athens) inbred to evaluate the effects of nuclear-cytoplasmic interactions on agronomic characters within inbred and also to evaluate the best combination of cytoplasm and genotype on the basis of inbred per se.

Inbred CI 21 (Athens) was prepared with the following cytoplasms: GA 199 and GT 112. Each source of cytoplasm as the female parent was crossed to the CI 21 (A) as the pollen parent. The F1, as the female, was backcrossed successively to the CI (21) genotype until the CI 21 (A) nucleus was totally substituted into each of the cytoplasms.

The cytoplasms were tested in paired comparisons. Among the characters studied, significant differences were obtained for effect of herbicide, reaction to Fusarium sp., northern leaf blight of corn, silking on a given day, tasseling, plant height, ear height, lodging and number of ears.

These results revealed a favorable interaction of GA 199 cytoplasm with the CI 21 (A) genotype for desirable characters. Apparently plasmon-sensitive effects and plasmagenic-environmental effects caused the unfavorable results.

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More precise linkage data on f13

It was reported in the 1976 MNL (50) that f13 was located on chromosome 8 and that the gene order was f13 v16 ms8 j. From F2 data, the recombination percentages were estimated as f13 v16 14 percent and f13 ms8 28 percent.

Backcross data are available from the 1976 growing season. The backcross was + v16 ms8 j/+ v16 ms8 j x + v16 ms8 j/f13 + + +. The plants were scored for plant characters at appropriate times during the growing season and for f13 at maturity. The data are given below disregarding v16 which was difficult to score in 1976.

NCO	f13 + +	129
	+ ms8 j	129
Co I	f13 ms8 j	84
	+ + +	85
Co II	f13 + j	15
	+ ms8 +	9
Co I & II	f13 ms8 +	6
	+ + j	9
		466

These backcross data give a considerably higher estimate of recombination between f13 and ms8 (39 percent) than did the F2 data of 1975.

Oliver Nelson

The appearance of C-I in a stock carrying an Spm-controlled c-mutable

In 1977, an attempt was made to isolate Spm-controlled bz-mutable genes. The female parent was homozygous Spm c-m5 Sh Bz wx-m8 while the male parent was W22 (c sh bz Wx). The total number of F1 kernels was 61518. All variant kernels were saved for testing in 1978. No variegated kernel proved to be a heritable bz-mutable.

Among the variant kernels were five that resembled bz kernels with no hint of mutability. The plants from these kernels were tested by crossing reciprocally with the sh bz Wx (W22) tester. Four proved not to be changes at the bz locus since the backcrosses in both directions produced progeny that had ca. 0.5 bronze and 0.5 nonbronze kernels. The fifth plant on being crossed by the (sh bz Wx) tester produced ca. 0.5 colorless kernels and 0.5 bronze kernels. The counts were 190 colorless, nonshrunken; 183 bronze, shrunken; 3 colorless, shrunken; 9 purple, non-shrunken; and 6 purple, shrunken. The distribution is asymmetrical since one complementary crossover class (Region II, between Sh and Bz) was not distinguishable from other colorless, nonshrunken kernels.

The data suggest that C-I was generated in the c-m5 stock. The C-I allele could not have been brought in as a contaminant since it is on the chromosome contributed by the female parent, and the markers from the male parent sh bz are present in some of the testcross progeny.

The c-m5 stock came originally from Barbara McClintock in whose nursery it originated. She has kindly traced its origin as follows:

1951 (Plant 5919-2) $\frac{\text{Ac AcI Ds Sh Bz Wx}}{\text{Ac AcI Ds Sh Bz Wx}} \times \frac{\text{C sh bz wx}}{\text{C sh bz wx}}$; no Ac in ch. 9

1 odd kernel was I sh Wx; not variegated

1952 The plant from the odd kernel was used in many crosses, and its genotype was shown to be:

(6260A) Ac I Ds sh bz Wx/+ C + sh bz wx

(Unit of inheritance
No crossovers)

Among others, this plant was crossed to one which was

$\frac{\text{c sh Bz wx}}{\text{c sh Bz wx}}$; no Ac.

One exceptional kernel was c --- C, sh Bz Wx and this was the original kernel with c-m5. This mutable c allele was subsequently shown by McClintock to be Spm-controlled with Spm located at the C locus.

1953 (6617A) The constitution of this exceptional kernel was shown to be: c-m5 Ds sh bz Wx/c sh Bz wx; no Ac.

It is not possible to decide on genetic grounds whether the c-m5 arose from a locus that was originally C or one that was C-I. If C, then the departure of Spm is capable in some instances of generating C-I from a functional C. If C-I, the association of Spm with C-I is capable of producing a mutable c just as when a receptor associates with C.

Although I don't have precise data on the point, it appears that c-m5 produces both c and C derivatives far more often than C-I derivatives which may argue, although not strongly, for c-m5's origin from a functional C.

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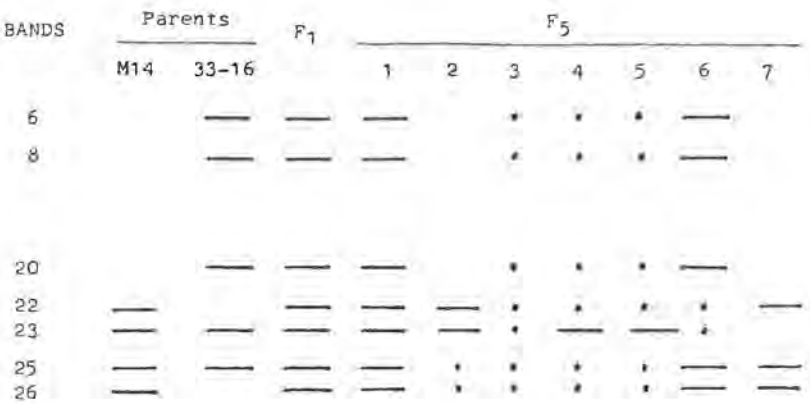
Inheritance of isoelectric-focusing (IEF) zein pattern

Isoelectric-focusing analysis reveals that zein is composed of several polypeptides with a very large variability between lines (Soave et al., 1976, Maydica 21:61-75). F1 kernels obtained by crossing inbred lines with different zein patterns show additivity and dosage effects in reciprocal crosses (Righetti et al., 1977, in Tech. Sep. Barley and Maize Prot.).

Analysis of advanced hybrid generations can furnish information on the number of factors controlling zein heterogeneity. Therefore 24 F5 ears from a single cross hybrid (33-16xM14) were analyzed by the IEF technique. Seven ears which clearly showed IEF hybrid patterns were further analyzed on single kernel basis in order to detect residual heterozygosis. A sample of 20 kernels for each ear was considered.



• indicates segregating bands.

In the figure IEF patterns of the parental lines, of F1 and of the seven F5 ears given refer only to those bands in which the two parents differ. Only two F5 lines revealed a fixed pattern, while the other showed segregation between kernels. On the basis of these results it can be concluded that there are at least three different groups of genes which control zein IEF heterogeneity. This conclusion agrees with other recent results indicating chromosomal localization of factors controlling the synthesis of zein bands (Soave et al., 1978, T.A.G. 52:263-267; Valentini et al., 1978, in press).

By means of TB-A translocations it was possible to locate a factor for zein band no. 13 on the short arm of chromosome 4. This segment analyzed with TB-4S in different genetic backgrounds (OH-43, N.25) was shown to contain factors controlling the expression of bands 4, 8, 12 and 14. Thus a group of zein genes is located in the short arm of chromosome 4.

E. Ottaviano and C. Soave

Amino acid effect on in vitro pollen germination and growth

Maize pollen can be cultured in vitro with great difficulty: it germinates, but with a very poor tube growth, on simple media. This report describes several attempts made to improve in vitro pollen tube growth.

Complex media, such as Murashige and Skoog (1962, Physiol. Plant, 15:473-497) or Gengenbach and Green media (1975, Crop Sci. 15:645-649) even modified in a number of ways, block its development.

Different types of stylar extracts were added to the standard medium. Extracts of not-pollinated silks proved to have an inhibitory effect on pollen growth, which become normal again (equal or larger than the standard medium control) when extracts of pollinated styles or not-pollinated silks plus pollen grains were employed. In any case a substantial growth increase was not obtained.

Another attempt was made by supplementing the growth medium with some amino acids, whose role may be particularly important in pollen metabolism. These are asparagine and glutamine, considered important for nitrogen uptake, and threonine, tyrosine, proline, which in maize are present in clearly different amounts in the pollen and silks (Linskens and Pfahler, 1977, Theor. Appl. Genetics, 50:173-177): tyrosine almost exclusively in the silks, proline only in the pollen grain, threonine in different but negatively correlated amounts in the pollen and silks of different genotypes. Six inbred lines and two mutants of one of them were used in the trial. The presence of asparagine and glutamine generally showed a negative effect on the growth. The findings relating to the other three amino acids are reported in Table 1. Threonine effects were in general favorable, but not for all

Table 1

LINES		Standard medium	tyr	threo	pro
C123	%G.	51.1	47.5	56.9*	58.9*
	T.L.	12.9	9.9*	13.4	14.*
M14	%G.	47.1	44.4	52.3	57.8*
	T.L.	6.8	7.1	8.3*	7.6
H3025	%G.	58.4	63.8	67.5*	71.5*
	T.L.	9.7	9.	10.8	11.2*
B14	%G.	47.9	55.6*	50.7	56.1*
	T.L.	7.8	8.	10.*	8.7
WF9	%G.	0.3	0.2	0.5	1.*
	T.L.	-	-	-	-
W22	%G.	41.9	40.2	43.9	38.8
	T.L.	5.8	4.5*	6.7	7.1*
W22 wx	%G.	55.3	55.1	58.3*	56.8
	T.L.	9.3	6.6*	10.8*	11.2*
W22 sh	%G.	50.1	49.6	46.3	62.3*
	T.L.	7.4	5.1*	7.7	8.3*

Standard medium: Sucrose 15%, agar 0.6%, H_3BO_3 0.01%, $Ca(NO_3)_2$ 0.03%
Tyr, threo, pro: standard medium supplemented with 0.04% of the aminoacids
%G. : germination percentage
T.L. : tube length in mm x 10
* indicates significant differences versus control

the genotypes; this is what could be expected in view of the different proportions of the content of this amino acid in the pollen and style of different genotypes. On the other hand, with regard to tyrosine and proline we obtained quite unexpected

results. Tyrosine, practically absent in maize pollen, was ineffective or even inhibiting for pollen growth, while proline, which represents the predominant component in the amino acid pool of pollen grain, was revealed to be effective in improving germination and tube growth.

On the basis of these results, we explored the effect of a wide range of doses of proline (from 0 to 0.64%). The results (which are not here reported) indicate a linear effect, up to a maximum of 0.16%. Thus the germination and growth improvement which can be obtained is in fact greater than those reported in Table 1. These findings might be explained by the central role of proline in pollen metabolism for energy delivery, protein synthesis and particularly--as hydroxyproline--for tube wall building.

M. Sari Gorla and E. Croci

Benzyladenine and anthocyanin biosynthesis

The role played by the R locus in anthocyanin biosynthesis is not known, even though studies on the activity of an enzyme involved in flavonoid biosynthesis (UDP glucose:flavonol 3-O-glucosyltransferase) point to a regulatory function (Dooner and Nelson, Biochem. Genet. 15:509-519, 1977). However, the information available is still too scant to attempt the formulation of a hypothesis on the gene action of R.

Pigment synthesis conditioned by a group of R accessions collectively known as R cherry is light dependent at least in some tissues like the cob or the mesocotyl, but the physiological basis of the photoinduction has not been explored. On the other hand in hybrid stocks of unknown R constitution it has been ascertained that at least two photoreceptor pigments are involved in photoinduction of anthocyanins in the mesocotyl (Duke, Fox and Naylor, Plant Physiol. 57:192-196, 1976). One of the two photoreceptors is phytochrome.

Table 1. Anthocyanin content (A_{530}) per mesocotyl and per unit length of mesocotyl following seed treatment with increasing concentration of BA.

Treatment	LA (cm)	A_{530}/msc	A_{530}/cm
		Dark(1)	
Control	8.16	0.196	0.024
BA (10^{-6})	3.91	0.495	0.126
BA (10^{-5})	2.25	0.777	0.345
BA (10^{-4})	1.41	0.718	0.509
		Dark + Light(2)	
Control	6.50	0.675	0.104
BA (10^{-6})	4.40	1.934	0.439
BA (10^{-5})	2.60	2.052	0.789
BA (10^{-4})	1.50	3.128	2.085

1) 9 days in darkness
2) 9 days in darkness followed by 48 hrs of light

Each determination is the average of at least 6 samples

LA : length of mesocotyl in cm.

In this note we report some preliminary results on the effects of a hormone, benzyladenine (BA), on anthocyanin production in the mesocotyl of homozygous cherry seedlings. This work is aimed to elucidate R gene action through an analysis of its interaction with a hormone in the process leading to anthocyanin synthesis. We have tested three cherry accessions, designated bol-1, bol-2 and bol-3, originally present in a Bolivian population and now introduced in the W22 background (5 backcrosses). Surface sterilized seeds were incubated in BA solutions of increasing concentrations (10^{-6} to 10^{-4} M) on a rotary shaker for 24 hours, transferred to large glass vessels layered with agar and grown for 9 days in darkness at 30 C. Anthocyanins of the mesocotyl were then extracted by grinding mesocotyl in a mortar with liquid nitrogen, adding a fixed volume of 0.1 percent HCL ethanolic (v/v) solution. The extract was then centrifuged and the supernatant used for spectrophotometric reading. The same procedure was followed on 9 days etiolated seedlings exposed to light for 48 hours.

The results obtained with bol-3 (Table 1) show that increasing concentrations of BA lead to a proportional increase in the pigment content of the mesocotyl as well as an inhibition in the seedling length, both effects being dose dependent. The other two alleles (bol-2 and bol-3) do not develop any pigment under these growth conditions. The response of bol-3 seems thus to suggest that anthocyanin enhancement reflects, in this case, a specific interaction between BA and the genetic material of bol-3. This cherry accession appears thus as a good candidate for analyzing the mechanisms of action of a hormone at the gene level.

C. Tonelli, G. Gavazzi and N. Avogadro

Determination of P5C reductase activity in the proline mutant

The biosynthesis of proline in all organisms so far studied proceeds from either glutamic acid or ornithine via glutamic-γ-semialdehyde which is in equilibrium with Δ'-pyrroline-5-carboxylic acid (P5C) (H. J. Vogel and B. D. Davis, J. Am. Chem. Soc., 74:109, 1952). P5C is metabolized to proline by P5C reductase, while proline degradation is accomplished by proline dehydrogenase (Mazelis M. and L. Fowden, J. Exp. Bot. 22:137, 1971) and proline oxidase (Boggess, Koeppe and Stewart, Plant Physiol. 63:22-25, 1978).

In our laboratory we isolated a proline requiring mutant, pro (Gavazzi, Racchi and Tonelli, Theoret. Appl. Genet. 46:339, 1975); that is seemingly the result of a genetic block between P5C and proline (Racchi, Gavazzi, Monti and Manitto, Plant Science Letters in press). Accordingly we analyzed the enzyme involved in this biosynthetic step.

Table 1. P5C reductase activity from normal and mutant embryos.

	pro_1		pro_2		pro_3	
	m	+	m	+	m	+
Soluble proteins mg/embryo	1.54	1.09	2.40	2.01	1.84	2.13
U / 100 mg FW	9.82	18.86	11.59	6.76	23.75	3.46
U / embryo	4.10	6.00	7.35	3.85	11.40	1.80
U / mg soluble proteins	2.66	5.50	3.06	1.91	6.19	0.84

U : enzyme unit
FW : Fresh Weight
Each value is the average of two determinations

Table 2. P5C reductase activity from normal and mutant (pro$_3$) callus grown on medium containing 2mM proline.

	m	+
Soluble proteins mg/100 mg FW	0.14	0.32
U / 100 mg FW	88.4	41.1
U / mg soluble proteins	631.4	128.4

U : enzyme unit
FW : Fresh Weight
Each value is the average of two determinations

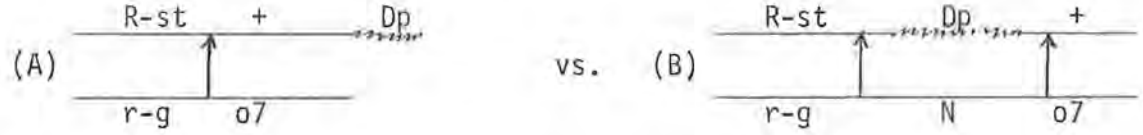
The enzyme was extracted from embryos of homozygous pro-1, pro-2 and pro-3 seeds (Racchi, Gavazzi this M.N.L.) previously soaked in water for 16 hours and from callus of homozygous pro-3 mutant grown on Murashige and Skoog medium supplemented with proline (2mM). The enzymatic assay was carried out following the procedure of Noguchi, Koiwai and Tamaki (Agr. Biol. Chem. 30: 452-456, 1966). An enzyme unit is defined as the enzyme amount which produces a decrease in optical density of 0.001 per minute at 340 nm. Soluble proteins were estimated by the method of J. Sedmak (Anal. Bioch. 79:544-552, 1977). Δ-pyrroline-5-carboxylate was prepared by the method of H. J. Strecker (J. Biol. Chem. 235:2045, 1960).

The results (Table 1) indicate that P5C reductase is present in both normal and mutant embryos; the mutant rates of pro-2 and pro-3 are higher than the normal. The enzymatic activity of pro-3/pro-3 and nonmutant callus parallels that found in the embryo tissues (Table 2). Presence of enzymatic activity in mutant tissues is in contrast with the hypothesis of a genetic block between P5C and proline. In view of these results the P5C reductase of normal and mutant tissues should be further characterized in terms of Km, pH optimum and so on. Such studies are under way.

Chiara Tonelli and Alcide Bertani

Locating duplications Dp 10-1, -3, -4 in relation to the o7 marker

The results reported in this note were obtained to establish whether the duplicated segments Dp 10-1, -3 and -4, lying in the long arm of chromosome 10 (Gavazzi, 1975, Heredity 35:389) are terminal or interstitial. Genetic markers used in testcrosses of the heterozygotes diagrammed here are R and o7 lying at positions 57 and 80 respectively on the long arm of chromosome 10:



As outlined in this scheme r-g + crossovers recovered in testcrosses of (A) parents should carry the duplicate segment while only a fraction of the crossover strands should carry it if the duplication is not terminal. Presence of the duplication was inferred from the development of anthocyanin in the sporophytic tissues since the Dp segment carries the P component of the R locus. The results are given here below:

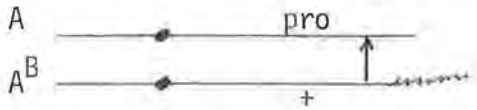
	N	Dp
Dp 10-1	2	49
Dp 10-3	0	112
Dp 10-4	0	7

These data, referring to the distribution of the Dp segment among verified r-g + crossover strands recovered in the progeny of heterozygous parents with an r-g o7 homozygous tester, are taken as evidence that all three duplications tested are terminal.

Giuseppe Gavazzi

Further data on the location of pro

The B-A translocations are currently used to locate factors to chromosome. Last year, in the 52 issue, we reported the results of a series of crosses of a recessive proline auxotroph (pro) not yet placed with a set of B-A translocations. The resulting hypoploids in the progeny of such crosses give normal 3:1 ratios, upon selfing, unless a hypoploid for the critical chromosome is involved. The only functional gametes yielded by the critical hypoploid in fact are those derived through crossing over between the locus and the break point as follows:



The pro mutant has been located, on this evidence, on chromosome 8. Hypoploids for chromosome 8 segregate 1545 mutant out of 2935 seeds, while normal siblings show a 3:1 ratio (799 mutants out of 3024 seeds).

A measure of the distance from the mutant to the breakpoint can be readily calculated from the relative frequencies of pro and Pro. The recombination fraction p, given by the formula:

$$p = \sqrt{1-(Pro/Pro+pro)}$$

(Beckett, 1978, J. Heredity 69:27) amounts to 27.2%. On the basis of this estimate we can predict that 72.8% of the gametes produced from a hypoploid for chromosome 8 are parentals and 27.2% recombinants. 84.4% (39.6/46.9) of the nonmutant plants in the progeny of a hypoploid are thus expected to be heterozygous for pro. 26 out of 31 (83.9%) of such plants are in fact segregating for pro nicely fitting the expected ratio and confirming the validity of our calculation.

G. Gavazzi and G. Todesco

Isolation of new alleles at the pro locus

The first proline auxotroph we isolated is easily recognized on the basis of its endosperm morphology and its lethality as seedling before the first leaf emergence (Gavazzi et al., 1975, Theor. Appl. Genetics 46:339). We relied on the collapsed endosperm phenotype as a criterion for recognizing other pro mutants. By screening populations of M2 ears, originally derived from seeds mutagenized with EMS or NG, we isolated six independent mutants resembling pro-1 in their endosperm morphology. A complementation test was then run by making each mutant heterozygous with pro-1 and scoring for production of collapsed endosperm phenotype as a proof of allelism between the mutant and pro-1. This test led to the identification of three additional alleles at the pro locus. They are tentatively symbolized pro-2, pro-3 and pro-4. They were originally isolated in an EMS (pro-2), control (pro-3) and NG (pro-4) treated population.

Nutritional tests have been performed on pro-2 and pro-3. Each mutant has been grown as excised shoot tips on liquid F media (Racchi et al., 1978, Plant Sci. Lett. in press) supplemented with different groups of amino acids at 1 mM concen-

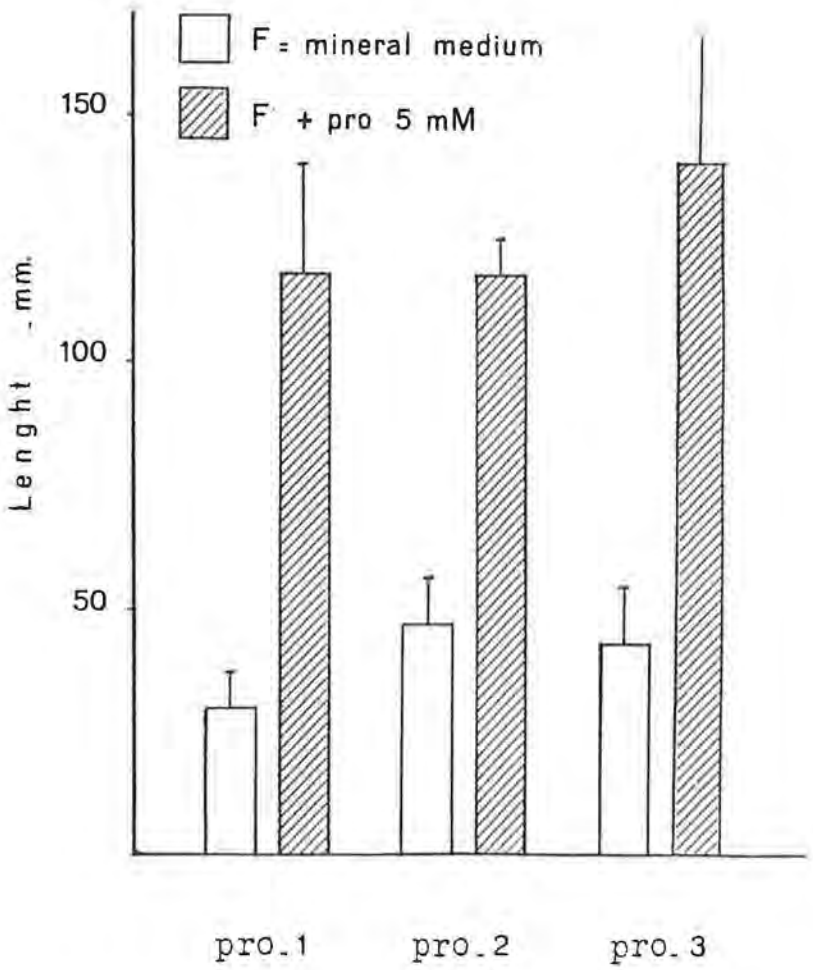


Fig. 1. Growth of excised shoot tips of pro-1, pro-2 and pro-3 homozygous mutants on either mineral medium (F) or F medium supplemented with proline (5 mM). Nonmutant sib values, not reported in the graph, are about 100, 130 and 100 mm for pro-1, pro-2 and pro-3 respectively and are the same on both minimal and supplemented media.

tration. The only group of amino acids growth promoting was the glutamate family (glu, arg, pro). Further tests on media with single additions of each of the three amino acids at 5 mM confirms that proline is the amino acid required by the mutants to resume normal growth (see Fig. 1).

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Resistance to common rust of maize in India

Puccinia sorghi, the common rust of maize in India, is one of the major disease problems, especially in rabi (= winter) crops in the state of Bihar. The two widely-grown hybrids, Hi-Starch and Ganga Safed 2, have shown a high degree of susceptibility. The known monogenic sources of resistance carrying gene Rp-d have been rated to be susceptible at Kalimpong, a location in the North-eastern Himalayas (Payak et al., 1974, Indian J. Gen. 34:31-35). It was, therefore, considered desirable to identify and utilize more broad-based or generalized type of resistance. In the All India Coordinated Maize Pathology Project, the inbred lines CM (= Coordinated Maize) 103, CM 104, CM 105, CM 106, CM 113, CM 500, GE 440 and Eto 25 have been rated to be resistant on the basis of their exposure to pathotypes prevalent at Bajaura (North Western Himalayas), Hyderabad (Andhra Pradesh) and Dholi (Bihar).

A study was initiated in 1972-73 to determine the genetic architecture of rust resistance employing the approaches of combining ability and di-allel analyses and estimation of gene effects. Heritability (narrow sense) as well as heterosis were also worked out. The trial was conducted at Dholi and Hyderabad in randomized block design under artificial disease epidemic. The parental materials consisted of six resistant (Eto 25, CM 103, CM 104, CM 105, CM 500 and GE 440) and two susceptible (CM 201 and PI 217407) inbred lines. Data obtained on the eight parental lines, 28 possible F1 single crosses (reciprocals mixed), 28 F2 generations and two sets (28 each) of back crosses were subjected to the above-mentioned biometrical analyses. Here we present in a summarized form the results and interpretations that have been arrived at.

Efforts to place the inheritance on a classical Mendelian basis were not successful. Significant deviation and heterogeneity components at Hyderabad and Dholi with the exception of 13:3 ratio of F2 and 3:1 ratio in test cross at the latter location indicated that on the whole the data did not fit in the expected segregation ratios. Thus the continuous nature of variation and failure to place the inheritance on a definite but small number of genes suggested that resistance to P. sorghi in the materials studied is controlled by polygenes. The plants were scored for disease according to 1-5 scale (1 = no disease and 5 = 76% or more leaf area covered). The disease scores in lines ranged from 1.5 to 3.0. However, the rust incidence was adequate enough at both the locations to draw valid conclusions. Original as well as transformed data were utilized for analyses.

The highly significant variation due to general combining ability (g.c.a.) at both locations suggested that it was playing a greater role in the expression of resistance. Lines CM 105, GE 440, CM 105, CM 103 and Eto 25 at Hyderabad and GE 440, CM 104, CM 105 and Eto 25 at Dholi gave negative values for g.c.a. thereby indicating that they were the best general combiners in the order of listing. Pooled analysis revealed that GE 440, CM 105 and CM 104 were the best general combiners. PI 217407 proved to be the poorest general combiner in location-wise as well as in the pooled analysis as it had the highest positive value for g.c.a.

In a similar type of study conducted at Hawaii in 1977 by Kim and Brewbaker (Crop Sci. 17:456-461) CM 105 was determined to be a good general combiner for resistance to this rust. However, CM 104 was rated by them as susceptible which in our study showed resistant reaction throughout at both locations. It probably suggests that the pathotype(s) of Puccinia sorghi prevalent in Hawaii may be different from those present in India.

The mean rating values and g.c.a. effects for the lines included in both these studies showed good agreement suggesting that the per se performance of the lines was a good indication of their ability to transmit resistance. The specific

combining ability (s.c.a.) effects contributed by combinations among resistant lines were non-significant. Susceptible x susceptible cross CM 201 x PI 217407 was not as susceptible as expected; this may be due to recessive alleles at different loci. The data revealed that while stability of performance was lacking for both the combining abilities, g.c.a. was more variable over locations. This observation highlights the importance of testing at more than one location.

The variance component analysis showed that both additive and non-additive gene systems are operative for resistance. Variances due to additive and non-additive gene effects were of the same magnitude at Hyderabad but at Dholi that due to the former was greater. Pooled analysis also showed that variance due to additive effects was greater than that arising out of non-additive ones or dominance. The inbred line, CM 105, contained a higher number of dominant genes for resistance. Graphic analysis showed the presence of over-dominance. Heritability estimates (narrow sense) were up to 40%, which is comparable with the value of 47% in the Hawaiian study.

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Meiotic mutation in maize

Studies with the identification and analysis of meiotic mutations in maize were started in 1973 as a joint work with Academician M.I. Khadzhinov and his collaborator A. S. Mashnenkov. Initial material was mutations of nuclear male sterility, induced by chemical mutagens in different maize lines (A344, W23, W64A). In all, 52 lines with nuclear male sterility (induced with N-nitroso-N-methyl urea by A. S. Mashnenkov, of P. P. Luk'yanenko Research Institute of Agriculture, Krasnodar) were investigated cytologically by me; in nine of these, meiotic mutations produced sterility. The collected mutations are listed in Table 1 and all mutations are inherited as monogenic recessive mutations (Table 2).

Table 1. The collection of meiotic mutations in maize induced by N-nitroso-N-methyl urea

Symbols	Short description
pam A A344, pam2 A344	Plural abnormalities of meiosis; two recessive monogenic nonallelic mutations responsible for the appearance of nonspecific meiotic abnormalities functioning both in premeiotic mitosis and in meiosis
afd W23	Absence of the first division of meiosis; recessive monogenic mutation causing the substitution of the first division of meiosis by mitosis
dsy A344, dys2 A344	Desynaptic; two recessive nonallelic monogenic desynaptic mutations
ms43 A344, ms28 A344, mei 025 A344	Three recessive monogenic mutations disturbing the regular chromosome segregation at AI
ms4 W64A	Recessive monogenic mutation inducing precocious postmeiotic mitosis

Table 2. Monogenic character of inheritance of meiotic mutations

Symbol	Crosses	Expected	Fertile, normal meiosis	Sterile, abnormal meiosis	χ^2
ms43 A344	F2(ms43/ms43 x A344)	3:1	102	21	2.76
ms28 A344	ms28/+, self	3:1	41	8	1.91
	ms28/ms28 x ms28/+	1:1	10	11	0.60
mei 025 W64A	mei 025/+, self	3:1	45	12	0.93
pam A A344	F2(pam A/pam A x A344)	3:1	75	22	0.27
pam2 W64A	pam2/+, self	3:1	43	10	1.05
dsy A344	dsy/+, self	3:1	44	15	0.08
dsy2 A344	dsy2/+, self	3:1	64	29	1.89
afd W23	afd/+, self	3:1	64	15	1.52
ms4 A344	ms4/+, self	3:1	22	8	0.13

A description of cytological effects of these meiotic mutations on meiosis is given briefly in this communication.

pam A A344, pam2 A344. The pam type of meiotic mutations affected both premeiotic mitoses and meioses. 25% of microsporocytes enter into meiosis as cenocytes. From prophase I, meiosis is desynchronized, so that all meiotic stages from prophase I to tetrads are observed in the same anther. Pycnosis and lysis of chromatin are observed in 18% of the microsporocytes. Various types of meiotic irregularities occur in the other sporocytes (desynapsis, impairment of the spindle apparatus, omission of cytokinesis). Mutant plants show complete male and incomplete female sterility. The occurrence of the pam type mei-mutations indicates that cells are being prepared for meiosis during the last 2-3 premeiotic mitoses (Golubovskaya and Mashnenkov, 1977, Genetika 13:1910-1921).

afd W23. In this mutant the typical steps of the first prophase of meiosis are omitted. At diakinesis, chromosome univalents are randomly scattered in the cells; they are morphologically distinct. At MI, univalents are orderly aligned forming a metaphase plate; at AI chromatids separate to each pole, i.e., centromeres separate during the first division in this mutant; as a result, the second division is much impaired. Gene afd substitutes the first division of meiosis by mitosis.

Studies of such types of meiotic mutations as am and afd open a new approach to the study of genetic mechanisms triggering all the programme from mitosis to meiosis. The existence of an ameiotic mutation blocking meiosis and mutation of afd type indicates that the underlying genetic mechanisms may be quite simple. Such highly specialized cells as meiocytes which have entered meiosis may, as a result of one or two mutation events, return to mitosis.

dsy A344, dsy2 A344. Desynaptic mutations are more frequently observed among meiotic mutations. The comparison of chromosome pairing at MI for two of these desynaptic mutations is given in Table 3. At pachytene, regular pairing is disturbed in some chromosome regions. At diakinesis, most chromosomes become univalents. There are, on the average, about 0.8 bivalents per MI cell. The other chromosomes do not pair. The second meiotic division proceeds synchronously in all the cells. The centromeres divide normally at AII. Polyads and tetrads with micronuclei are seen, instead of normal tetrads.

A preliminary electron microscopic study on the desynaptic mutant plants (dsy A344/dsy A344) was performed in the Laboratory of Ultrastructure of our Institute by Christoljubova. It was found that the s.c. in this mutant is abnormal.

Table 3. Character of chromosome pairing at MI for two desynaptic mutants of maize.

Chromosome configurations	dsy2 A344				dsy A344	
	diakinesis		metaphase I		metaphase I	
	number of cells	%	number of cells	%	number of cells	%
20'	34	49.3	39	46.40	92	56.7
18'	7	10.2	3	3.60	37	22.7
16'	17	24.6	16	19.04	19	11.7
14'	2	2.9	9	10.71	12	7.4
12'	6	8.7	12	14.30	2	1.5
10'	1	1.4	5	5.95		
8'	2	2.9				
Total	69		84		162	
Average number at MI			0.79" + 18.42'		0.74" + 18.52'	

To exclude an influence of inbreeding on the structure of the s.c., now we will make a comparison of s.c. in desynaptic plants, segregating from the poor line A344, with ones segregating in F2 from the cross +/dsy A344 (line A344) x +/+ (line W23).

ms43 A344, ms28 A344, mei 025 W64A. These are three meiotic mutations impaired in normal chromosome segregation at the first division of meiosis. The pairing pattern of mei mutant plants homozygous for ms43 is normal. Meiotic irregularities appear only at the first anaphase. The chromosomes are either randomly scattered in the cell (26%), or all the chromosomes remain in its center (33%). The chromosomes of 22% of cells move in groups of ten towards each pole. However, no condensed nuclei arise in this mutant at TI. This very irregular meiosis gives rise to polynuclear pollen grains. Only male sterility is characteristic of these mutant plants.

Meiotic behavior in the mutant ms43 was compared with that of the earlier described maize mutant dv (Clark, Amer. J. Bot. 27:547, 1940). These mutants were found to be similar in meiotic behavior at MI and AI. Their allelic relationships are now under study.

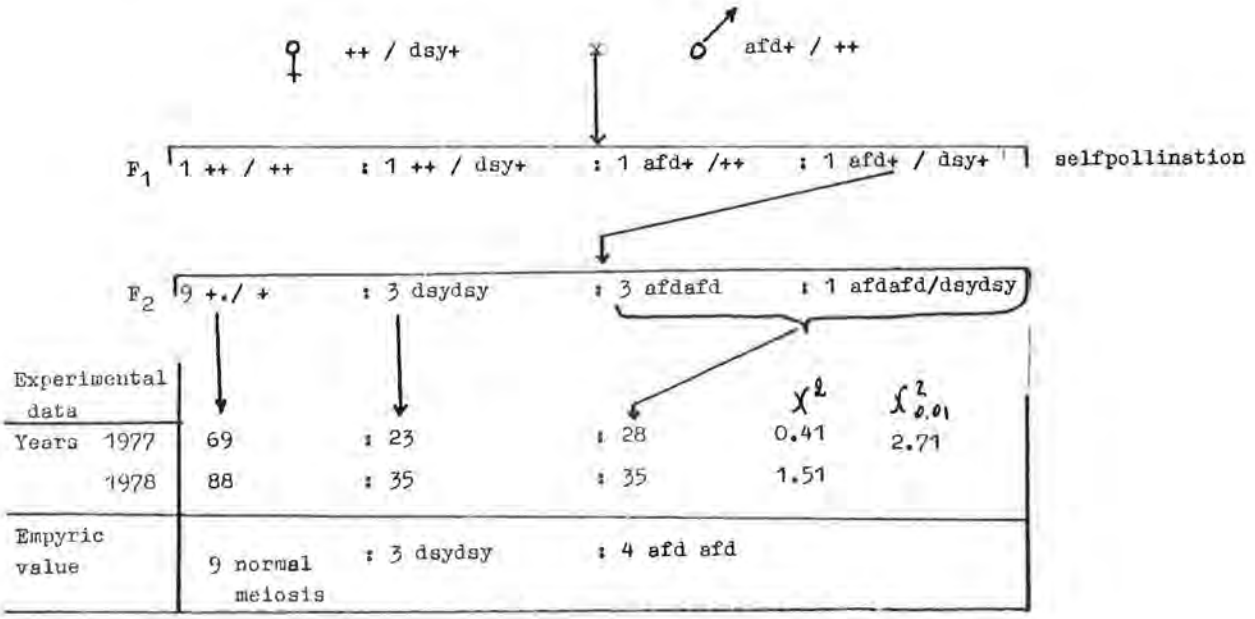
Mutation ms28 A344 impairs the chromosome segregation and moreover blocks cytokinesis partially or fully.

In meiotic mutants mei 025 W64A meiosis proceeds normally until MI. The chromosomes become sticky and cluster at MI and are immobile at AI. Typical TI figures are rarely seen. The chromosomes remain in pycnotic state from MI to interkinesis. Chromatin uncoils at interkinesis, but at MII the chromosomes become sticky again; at AII they are abnormally separated. Cytokinesis proceeds also after the first and second divisions.

Three mutations impairing the different functions of chromosome segregation at the first division of meiosis (ms43 A344, ms28 A344, mei 025 W64A) offer an approach to studies of the structure and function of the spindle in the meiotic cells.

ms4 W64A. Meiosis is regular until the tetrad stage in this mutation. The chromosomes of microsporads are subjected to precocious postmeiotic mitosis, chromosomes enter mitosis without replicating and this causes abnormal mitosis. Pollen grains have no pores in mutant plants. It would be needed to compare mei 025 with the mutation po studied by Beadle (Cytologia 5:118, 1933).

The set of meiotic mutations affecting or blocking different meiotic steps might help to clarify the genetic control of this complex process. The question was to determine whether loci affect the same or different pathways of meiosis. Double meiotic mutants may provide an answer to this question. Two monogenic recessive mutations were chosen as a model; desynaptic mutation dsy A344 and afd W23, substituting the first division meiosis with mitosis. It was possible to obtain a double homozygote for these recessive mutations, because they are completely sterile. A scheme experiment is as follows:



A double heterozygote dsy +/afd + was obtained. The segregation of meiotic pattern was analyzed in offspring obtained by self pollinating this heterozygote. The expected segregation was 9/16 plants with normal meiosis:3/16 plants with dsy type of meiosis:3/16 plants with afd type of meiosis:1/16 plants of genotype dsy dsy afd afd. What will be the meiotic pattern in the double homozygote? If afd and dsy genes control the same sequence of events in meiosis then meiosis in double homozygotes should be either as in the afd type (if afd is earlier involved in meiosis) or of the dsy type (when the desynaptic gene is involved earlier than gene afd). If genes afd and dsy control different alternative pathways of meiosis, then meiosis in a double homozygote should be different from those two mutations.

Analysis of the meiotic pattern in offspring of a double heterozygote demonstrated segregation of 9/16 plants with normal meiosis:3/16 dsy plants:4/16 afd plants (see the scheme above). This means that the genes afd and dsy control the same sequence of meiotic events and that for the accomplishment of events controlled by the gene dsy^+, events controlled by the gene afd^+ have to take place first.

Concluding I should say that a collection of meiotic mutants can be used:

1. To gain insight into meiosis and its triggering genetic mechanisms; meiosis may be considered as sequential events in a cell and these events are controlled by corresponding genes.
2. For detailed studies on meiotic mutants, which make it possible to establish relationships between cytologically visible structures (the s.c. and chiasmata) and cytogenetic processes (chromosome pairing, crossing over).

3. To establish similarities and differences in processes of meiotic recombination, DNA repair and mutability in eukaryotes.

4. To build models of meiosis occurring in nature. These models help to understand mechanisms blocking single steps of meiosis in apomictic plants and parthenogenetic animals.

I. N. Golubovskaya

Effects of translocations on the interallelic interactions in the alcohol dehydrogenase (ADH) system in maize

Isozymes as markers of gene activity provide an approach to some genetical problems such as interallelic and intergenic interactions. An advantageous enzyme is maize alcohol dehydrogenase (ADH) which is controlled by two nonlinked genes, Adh and Adh2 (Schwartz, 1966; Freeling and Schwartz, 1973). In heterozygotes Adh-F Adh-S the relative concentrations of FF, FS and SS isozymes are distributed binomially. This indicates that subunits of ADH molecules associate randomly with equal probability.

However it was also shown in our studies that the isozyme pattern controlled by nonallelic genes Adh and Adh2 is often not binomial (Fig. 1).

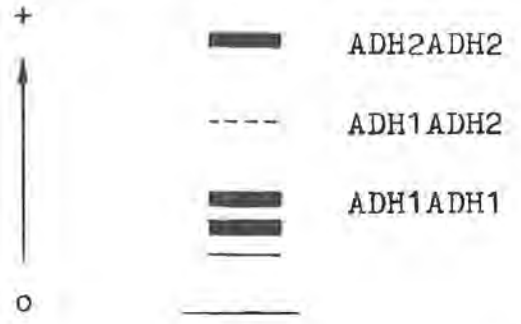


Fig.1. ADH isozyme pattern in maize leaves of 10-13-day-old plants.

The nonbinomial distribution may be the simple sum of patterns yielded by two types of cells, in which one of the genes is more active, either Adh or Adh2.

According to an hypothesis of Henderson (1968) the nonbinomial distribution pattern of isozymes controlled by nonallelic genes is the possible result of the spatial disconnection of the mRNA's of these genes during translation; and according to an hypothesis of Serov (1977) mRNA's of allelic genes have to be located in the same region of cytoplasm during translation to ensure interaction between alleles.

The question is raised whether the spatial disconnection of the translation of the mRNA's of the nonallelic genes is the result of the spatial disconnectedness of the genes themselves.

It is known the gene Adh is located in the 1 chromosome (Schwartz, 1971) and Adh2 is located in the 4 chromosome (M. Freeling, cited after Schwartz, 1976), and the spatial chromosome distribution is very much dependent on the structure of the interphase cell nucleus.

Here, a convenient model would be the interaction of the different alleles of a gene transferred to different chromosomes by translocations. Such a model was used in this study. Maize forms of American origin with breaks in the 1L (long) arm located proximally to the gene Adh were used: T1-9(4995)(1L.19-9S.20); T1-4d(1L.27-4L.30); T1-8a(1L.41-8S.52); T1-9b(1L.50-9L.60). The plants had to be simultaneously heterozygotes for translocations and heterozygotes for the Adh gene. It was first established that all the forms with translocations are homozygous for the F allele. Therefore the plants involved in crosses were a normal line W155 with genotype Adh-S Adh-S and initial forms heterozygous for translocations identified by the criterion of 50% sterile pollen. The semisterile progeny of this cross

were the experimental plants and their fertile sibs were controls. Plants of both groups had Adh-F Adh-S genotype.

ADH was analyzed by polyacrylamide gel electrophoresis. This analysis of forms with translocations and their normal sibs demonstrated similar ADH patterns of the scutella of dry seeds with a binomial distribution of staining intensity. There were no differences between the ADH patterns of leaves of control and experimental plants at the age of 10-13 days and at the flowering stage. Deviation from the binomial distribution pattern was observed only in one form, W155 x T1-9b in the flag leaves of plants 20 days after flowering: FF (58.2%):FS (27.6%):SS (14.2%); χ^2 = 18.1. In normal plants of this group the quantitative relationship of isozymes in the pattern was as follows: FF (57%):FS (31%):SS (12%); χ^2 = 4.9.

Thus, the distribution pattern in one of the maize forms studied deviates from binomial. The deviation was observed when the activity of the enzyme is very low.

There are two tentative explanations for these deviations: 1) It may be that translocation promotes cell heterogeneity slightly expressed even in normal plants judging by the insignificant deviation of the binomial distribution pattern. 2) As a result of translocation the Adh-F allele is further away from Adh-S than in the other forms studied. It cannot be ruled out that in this form the mRNA coded for by different alleles of the gene Adh passes to the cytoplasm from different nuclear regions.

Nonbinomiality of the interallelic ADH isozyme pattern is expressed very weakly, whereas nonbinomiality of the distribution of the intergenic isozyme pattern is quite clear-cut (Fig. 1).

Based on these observations it was inferred that even if the structure of the interphase nucleus does affect the interaction of nonlinked genes this effect is to a large extent mediated by some specific additional factors.

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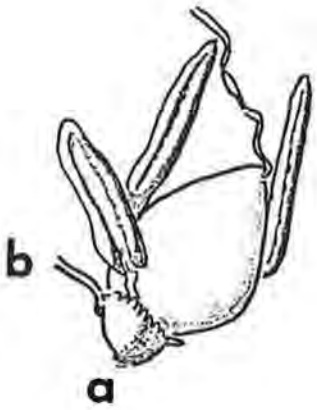
Hermaphrodite florets in a derivative of a corn x teosinte hybrid

Individual florets from corn, teosinte and Tripsacum inflorescences are typically unisexual. Although the tassel-seed mutants in corn produce part male, part female inflorescences, the individual florets are either male or female--not hermaphrodite. The material to be described arose from a complex parentage involving two marker stocks of corn (tassel-seed, Ts5, and unbranched, ub), a primitive popcorn from the Maize Collection identified as Argentine Altiplano 548, and a Mexican race of teosinte, "El Salado." Teosinte was used only as a male parent during the breeding procedure:

(Ts5 x Altiplano) x teosinte. A tassel-seed type extracted from this cross was selfed to provide Parent A.
(ub x teosinte). This hybrid was selfed and a homozygote, ub ub, extracted in F2 to provide Parent B.

Parent A was heterozygous for tassel-seed; Parent B was homozygous for unbranched. A small progeny from the F2, A x B, segregated for tassel-seed and what was, supposedly, a modified variant of unbranched (i.e., tassels with a main axis and only 2-3 laterals). Among the segregates was a new type, having the gross morphology and exterior spikelet morphology of a normal corn tassel, except that silks emerged from nearly all the spikelets. It was thought, at first, that all the spikelets had been converted completely from male to female, without the usual

condensation and rachis enlargement associated with tassel-seed. Accordingly, the "tassel" was outpollinated in an attempt to set seeds. Ten to twelve days after pollination the developing caryopses began to protrude through the sterile glumes and concomitantly anthers were extruded and began to shed a limited amount of pollen. Careful dissection of the spikelets showed that the basal floret in most spikelets was hermaphrodite. It included all the normal structures of a male floret (glumes, paleas, three anthers) and in addition a developing caryopsis with the withered base of the silk still attached (see text figure). The upper floret



Basal Floret, with both male and female organs, obtained from a tassel of a corn x teosinte hybrid. Glumes and lemma have been removed. The floret was attached to the tassel rachis at point A; the upper floret of the spikelet was removed at point B. (The upper floret had the normal male structure). The pattern of hermaphrodite basal floret and male upper floret persisted through most of the spikelets on the tassel. From ten to twelve days before the drawing was made, the tassel had been outcrossed with foreign pollen. The drawing shows the relatively advanced development of the fertilized ovule, though the anthers are still unexserted and indehisced. Subsequently the anthers dehisce, shedding a limited amount of apparently normal pollen. (Drawing traced from a photograph made by S. Flashman.)

in each spikelet was normal (i.e. unisexual, male). Spikelets located toward the extremities of the tassel branches were entirely male. Although the anthers shed a limited amount of pollen, and the pollen grains appear normal under microscopic examination, it is not yet known to what extent they are viable. In any event, the plant is completely self-sterile because of its strong protogyny. The development of axillary branches (ear shoots) is much delayed and no silks have emerged to date.

S. G. Stephens

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Further report on the genetics of MDH

We can now report that we have found linkage between the MdhD locus, which governs the inheritance of the darkest staining, soluble MDH bands, and the migration modifier (MdhM-m) present in such lines as Ky228, Mo24W, and W629A (see MNL 52:99-100). We observed only 3 crossovers among 120 chromosomes carrying MdhD-12 and MdhM-m among testcrosses of Mo24W x Tx325 to Ky228 and W629A. Tight linkage has also been observed between a rare MdhD allele (MdhD-8.5) found in a single Latin American collection and MdhM-m. These two observations have become

more significant since in this issue Kathleen Newton (University of Indiana) reports that she has determined the chromosomal location of a very similar, if not identical, migration modifier.

We also have limited data on a seventh MDH locus, also apparently linked to MdhD, discovered in a single collection of the race Pira from Venezuela. Each plant surveyed had the same rare variant, and most plants were homozygous for it, yet we had failed to detect such a variant in any of over 100 other Latin American collections, including several Venezuelan Piras.

Since last year's report we have determined that "null" alleles do exist at MdhC and that MdhE (MdhF in last year's report) is essentially independent of MdhA, MdhB, MdhC, and MdhD. We have also found three additional U.S. lines which may be helpful in studies of maize MDH. B8 from Iowa State and M49 from Pioneer International share the same rare MdhB allele (MdhB-4.5) which, when present in homozygous condition, results in no detectable MdhB locus homodimer on our gels, but which does result in heterodimers with the products of both MdhA and MdhC. When MdhB-4.5 is heterozygous with a more common allele, the homodimer corresponding to the more common allele is present, the expected MdhB locus heterodimer is also present, but the MdhB homodimer corresponding to the MdhB-4.5 allele is absent. For the MdhE locus, Mich77-7, an unreleased Michigan line produced by Dr. Elmer Rossman, may be useful. While the line is still segregating, it appears to have a fast migrating allele (MdhE-16.6) at a locus which is not highly variable among adapted U.S. materials.

Major M. Goodman and Charles W. Stuber

Genetics and linkage of PGM

In our studies of PGM isozymes, we have discovered that two loci are associated with the variation found in maize. Although resolution of many of the bands can be made using starch gels with a buffer system consisting of L-histidine and citric acid (pH 6.5), resolution of some overlapping bands can be accomplished only by lowering the pH of the buffer to 5.0. Therefore, we routinely use both buffer systems in our electrophoretic analyses of PGM isozymes.

We have identified 13 alleles at the Pgm1 locus and 6 alleles at the Pgm2 locus. Analyses of data from five different F2 populations suggest that the two loci segregate independently (Table 1).

Table 1. The *Pgm1* and *Pgm2* loci appear to segregate independently in F_2 populations.

	Pgm1-9/Pgm1-9	*Pgm1-9/Pgm1-3*	*Pgm1-3/Pgm1-3*	Total
Pgm2-4/Pgm2-4	(1.5)* 2 [1.5]†	(1.0) 2 [3.0]	(1.5) 0 [1.5]	4 [6.0]
Pgm2-4/Pgm2-8	(6.0) 6 [3.0]	(4.0) 3 [6.0]	(6.0) 7 [3.0]	16 [12.0]
Pgm2-8/Pgm2-8	(1.5) 1 [1.5]	(1.0) 1 [3.0]	(1.5) 2 [1.5]	4 [6.0]
Total	9 [6.0]	6 [12.0]	9 [6.0]	24

χ^2_4 (independence) = 3.42; $0.25 < P < 0.50$

χ^2_8 [4:2:2:2:2:1:1:1:1] = 13.50; $0.50 < P < 0.10$

Table 1. (Continued)

	Pgm1-1/Pgm1-1	*Pgm1-1/Pgm1-6*	*Pgm1-6/Pgm1-6*	Total
Pgm2-3/Pgm2-3	(9.4) 12 [9.6]	(24.0) 21 [19.3]	(11.7) 12 [9.6]	45 [38.5]
Pgm2-3/Pgm2-4	(15.2) 17 [19.3]	(38.9) 39 [38.5]	(19.0) 17 [19.3]	73 [77.0]
Pgm2-4/Pgm2-4	(7.5) 3 [9.6]	(19.2) 22 [19.3]	(9.4) 11 [9.6]	36 [38.5]
Total	32 [38.5]	82 [77.0]	40 [38.5]	154

x_4^2 (independence) = 4.94; 0.25 < P < 0.50

x_8^2 [4:2:2:2:2:1:1:1:1] = 7.01; 0.50 < P < 0.75

	Pgm1-5/Pgm1-5	*Pgm1-5/Pgm1-16*	*Pgm1-16/Pgm1-16*	Total
Pgm2-3/Pgm2-3	(9.0) 9 [8.2]	(25.5) 26 [16.4]	(12.6) 12 [8.2]	47 [32.8]
Pgm2-3/Pgm2-8	(15.1) 15 [16.4]	(42.8) 42 [32.8]	(21.1) 22 [16.4]	79 [65.5]
Pgm2-8/Pgm2-8	(1.0) 1 [8.2]	(2.7) 3 [16.4]	(1.3) 1 [8.2]	5 [32.8]
Total	25 [32.8]	71 [65.5]	35 [32.8]	131

x_4^2 (independence) = 0.21; 0.990 < P < .995

x_8^2 [4:2:2:2:2:1:1:1:1] = 35.72; P < .005

	Pgm1-5/Pgm1-5	*Pgm1-5/Pgm1-7*	*Pgm1-7/Pgm1-7*	Total
Pgm2-3/Pgm2-3	(1.9) 2 [4.4]	(3.5) 2 [8.9]	(2.6) 4 [4.4]	8 [17.8]
Pgm2-3/Pgm2-8	(10.1) 10 [8.9]	(18.3) 19 [17.8]	(13.6) 13 [8.9]	42 [35.5]
Pgm2-8/Pgm2-8	(5.0) 5 [4.4]	(9.2) 10 [8.9]	(6.8) 6 [4.4]	21 [17.8]
Total	17 [17.8]	31 [35.5]	23 [17.8]	71

x_4^2 (independence) = 1.63; 0.75 < P < 0.90

x_8^2 [4:2:2:2:2:1:1:1:1] = 9.26; 0.25 < P < 0.50

	Pgm1-3/Pgm1-9	*Pgm1-3/Pgm1-16*	*Pgm1-16/Pgm1-16*	Total
Pgm2-4/Pgm2-4	(14.5) 16 [12.3]	(21.3) 21 [24.5]	(13.3) 12 [12.3]	49 [49.0]
Pgm2-4/Pgm2-8	(30.2) 34 [24.5]	(44.2) 44 [49.0]	(27.6) 24 [24.5]	102 [98.0]
Pgm2-8/Pgm2-8	(13.3) 8 [12.3]	(19.5) 20 [24.5]	(12.2) 17 [12.3]	45 [49.0]
Total	58 [49.0]	85 [98.0]	53 [49.0]	196

χ^2_4 (independence) = 5.28; $0.25 < P < 0.50$

χ^2_8 [4:2:2:2:2:1:1:1:1] = 10.00; $0.25 < P < 0.50$

*Expected values, based upon independence, shown in parentheses.

+Expected values, based upon 4:2:2:2:2:1:1:1:1 segregation shown in brackets.

C. W. Stuber and M. M. Goodman

Genetics and linkage of GOT

Genetic control and intracellular localization of GOT isozymes in maize have been described (Scandalios, J. G., Sorenson, J. C., and Ott, L. A., 1975, Biochem. Genet. 13:759-769). They showed that one isozyme was associated with the mitochondrial fraction, one was associated with the soluble fraction, and one with the glyoxysomes. In their materials, only the latter was found to have more than one form. In the wide array of materials that we have studied in maize, we have found five alleles at the locus Got3 associated with the mitochondrial forms, five alleles at the locus Got2 associated with the soluble forms, and at least four alleles at the locus Got1 associated with the glyoxysome forms. In heterozygotes, hybrid bands are produced as a result of interaction between alleles at each of the three loci, suggesting that the maize GOT's are dimers. No hybrid bands are formed between pairs of loci, however.

Table 1. Results from F_2 and testcross populations involving three segregating *Got* loci.

	Got3-4/Got3-4	*Got3-4/Got3-6*	*Got3-6/Got3-6*	Total
Got1-4/Got1-4	(11.8)* 10 [12.4]+	(22.9) 20 [24.9]	(11.3) 16 [12.4]	46 [49.8]
Got1-4/Got1-6	(26.1) 30 [24.9]	(50.7) 51 [49.8]	(25.1) 21 [24.9]	102 [99.5]
Got1-6/Got1-6	(13.1) 11 [12.4]	(25.4) 28 [24.9]	(12.6) 12 [12.4]	51 [49.8]
Total	51 [49.8]	99 [99.5]	49 [49.8]	199

χ^2_4 (independence) = 4.43; $0.25 < P < 0.50$

χ^2_8 [4:2:2:2:2:1:1:1:1] = 4.72; $0.75 < P < 0.90$

	Got3-4/Got3-4	*Got3-4/Got3-6*	*Got3-6/Got3-6*	Total
Got2-4/Got2-4	(9.9) 4 [10.5]	(15.4) 15 [21.0]	(8.7) 15 [10.5]	34 [42.0]
Got2-4/Got2-7	(27.7) 32 [21.0]	(43.0) 40 [42.0]	(24.3) 23 [21.0]	95 [84.0]
Got2-7/Got2-7	(11.4 13 [10.5]	(17.6) 21 [21.0]	(10.0) 5 [10.5]	39 [42.0]
Total	49 [42.0]	76 [84.0]	43 [42.0]	168

χ^2_4 (independence) = 12.40; $0.01 < P < 0.025$

χ^2_8 [4:2:2:2:2:1:1:1:1] = 17.19; $0.025 < P < 0.05$

	Got2-4/Got2-4	*Got2-4/Got2-6*	Total
Got1-4/Got1-4	(40.8) 48 [42.0]	(53.2) 46 [42.0]	94 [84.0]
Got1-4/Got1-6	(32.2) 25 [42.0]	(41.8) 49 [42.0]	74 [84.0]
Total	73 [84.0]	95 [84.0]	168

χ^2_1 (independence) = 5.03; $0.01 < P < 0.025$

χ^2_3 [1:1:1:1] = 9.29; $0.025 < P < 0.05$

*Expected values, based upon independence, shown in parentheses.

†Expected values, based upon 4:2:2:2:2:1:1:1:1 or 1:1:1:1 segregation, as appropriate, shown in brackets.

The Got1 and Got3 loci appear to segregate independently as shown in Table 1. However, analyses of F2 and testcross data involving Got1 with Got2 and Got2 with Got3 indicate that the two pairs of loci are loosely linked ($r = 42.3$ and $r = 42.5$, respectively). Thus, the data suggest that all three loci are located on the same chromosome with Got2 between Got1 and Got3. We are attempting to determine the chromosome involved using primary trisomics.

C. W. Stuber and M. M. Goodman

Hormonal modulation of catalase expression in maize scutellum

Total catalase (H_2O_2:H_2O_2 oxidoreductase, EC 1.11.1.6) activity in maize scutellum increases dramatically during the first three or four days of germination and decreases gradually thereafter (Scandalios, J. Hered. 65:28-32, 1974). A similar pattern of scutellar catalase development has been observed for excised intact embryos (embryonic axis attached to scutellum) cultivated on solid nutrient medium in the dark. The influence of exogenously applied plant hormone--i.e., indoleacetic acid (IAA), gibberellic acid (GA_3), benzyladenine (BA) and abscisic acid (ABA)--on the developmental pattern of catalase were studied in an attempt to establish any relationship between the action of these hormones and those regulatory mechanisms already known to affect the expression of catalase in maize scutellum (Scandalios, Isozymes, Vol. III, 213-238, ed. C. L. Markert, 1975). Plant hormones were applied by incubating intact embryos of maize inbred line W64A, on solid nutrient medium containing different hormones at 10 μM. The developmental

profile of catalase activity in intact embryos following the application of ABA, BA, GA_3, or IAA is shown in Figure 1. Although treatment with GA_3 or BA does not cause a significant change in the developmental pattern of catalase activity, IAA promotes both the initial rise in and the retention of catalase activity. Application of ABA to intact embryos delays both the rise and subsequent fall in catalase activity in scutella during early germination.

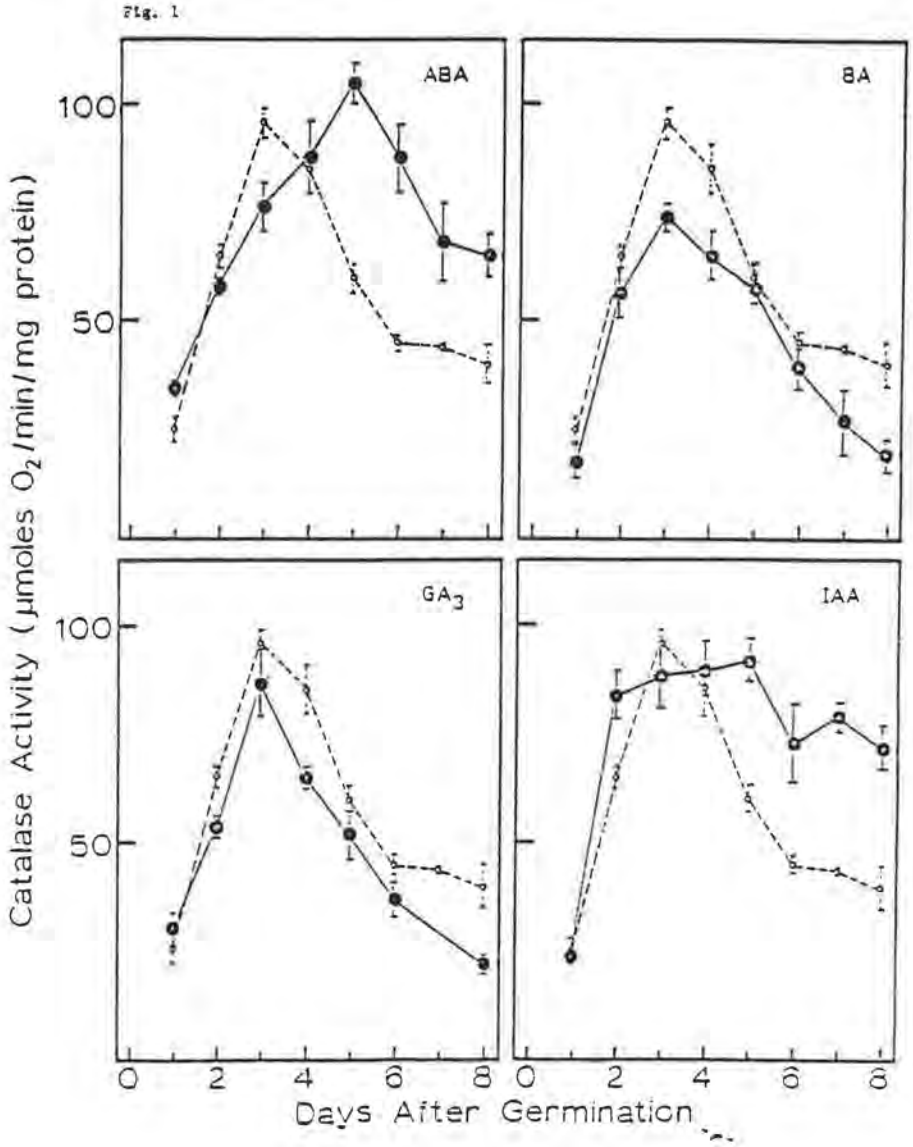

Fig. 1. Comparisons of the development of catalase activity (● ——— ●) in scutella of excised intact embryos incubated on nutrient medium containing ABA, BA, GA_3 or IAA (10 µM) with that of the control (○ -------- ○). Points and bars represent means and standard deviations of three replicates in three or more independent experiments.

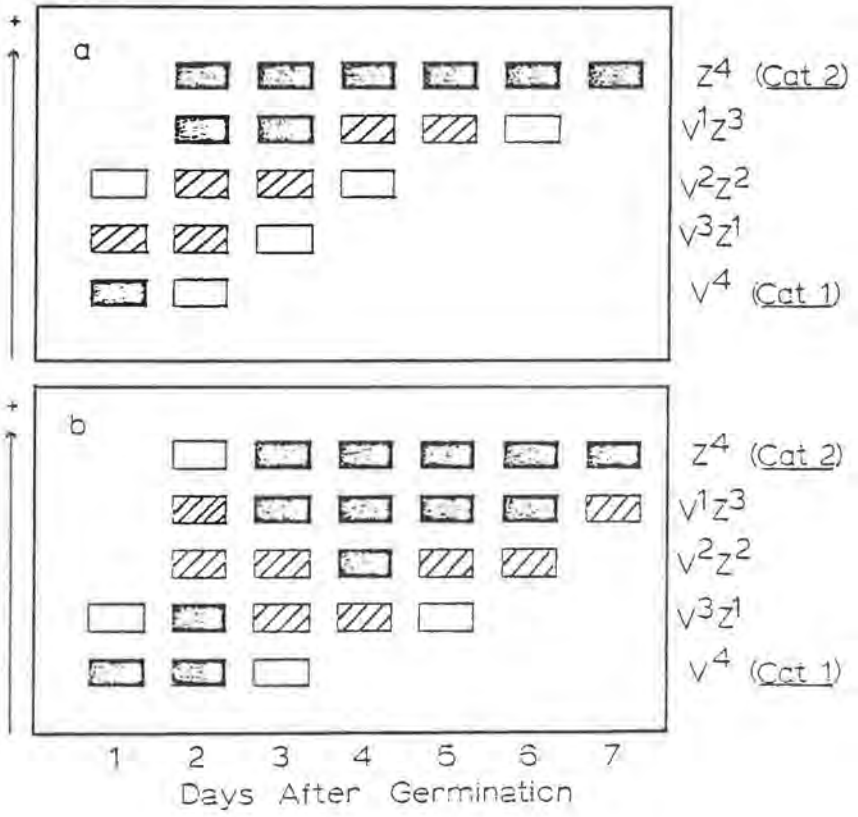

Fig. 2. Schematic diagram of the change of catalase isozymes in scutella of excised intact embryos grown on nutrient medium containing (a) water as control, or (b) ABA at 10 μM. The isozyme V^4 is composed of subunits from the product of the *Cat 1* gene. The isozyme Z^4 is composed of subunits of the product of the *Cat 2* gene. The intermediate isozymes are hybrids composed of subunits from both gene loci.

In addition, ABA alters the developmental pattern of catalase isozymes in scutella (Fig. 2). The expression of the Cat1 gene product (isozyme V) is prolonged after ABA treatment. On the other hand, the expression of the Cat2 gene product (isozyme Z) is delayed by ABA treatment. These data suggest that ABA may be involved in regulating the differential expression of catalase genes in maize scutellum.

It has been previously shown that catalase is turning over in the maize scutellum during germination (Quail and Scandalios, Proc. Nat. Acad. Sci. USA, 68:1402-1406, 1971). Data from density-labeling experiments indicate that catalase is synthesized de novo in the scutella of ABA-treated intact embryos. Comparing the density shift and the band width at the half-height peak of catalase distribution in CsCl gradients of ABA-treated scutella with those of the untreated control, it is suggested that ABA may be involved in regulating the rate of synthesis and the rate of degradation of catalase in maize scutella during early germination. Therefore, the turnover rates of a specific catalase gene product in response to the treatment of ABA are being investigated by density-labeling techniques and using the porphyrinogenic drug 2-allyl-2-isopropylacetamide (AIA) to inhibit catalase synthesis in an attempt to further evaluate mechanisms of differential gene expression.

Ray Whay Yen and John G. Scandalios

Catalase gene mapping on the maize genome

Maize catalase is coded by three independent genetic loci, Cat1 and Cat2 (Scandalios, J. Hered. 65:28-32, 1974), and Cat3 (Roupakias and Scandalios, unpublished). Artificial crosses of eighteen A-B translocation strains (as males) to W59, Oh51A and W10 inbred strains (as females) and screening, with starch gel electrophoresis, of the F1 progenies for Cat1, Cat2 and Cat3 showed that Cat1 is located on the short arm of chromosome 5 and approximately 10 map units away from the marker gene for brittle endosperm. Preliminary data indicate that Cat2 is located on the long arm of chromosome 10 and Cat3 on the short arm of chromosome 10. Furthermore, trisomic analysis also indicates that Cat2 and Cat3 are both located on chromosome 10 and approximately 40 map units apart from each other. Further investigation is underway for a conclusive location of these two genes on the maize genome.

D. G. Roupakias, D. E. McMillin and J. G. Scandalios

Chromosome location of two mitochondrial malate dehydrogenase structural genes

The model for the genetic control of mitochondrial malate dehydrogenase (mMDH) postulates two diallelic sets of duplicated loci (Yang, Sorenson and Scandalios, PNAS 74:310-314, 1977). The duplicated sets of loci and the isozyme products each codes for are:

Gene Set	Genotype	Isozymes Expressed
Mdh1 Mdh2	Mdh1-m1, Mdh2-m3	MDH1, MDH3
	Mdh1-m2, Mdh2-m5	MDH2, MDH5
Mdh3 Mdh4	Mdh3-m1, Mdh4-m3	MDH1, MDH3
	Mdh3-m3a, Mdh4-m7	MDH3a, MDH7

The gene Mdh1 codes for the isozymes MDH1 or MDH2 while Mdh2 codes for the isozymes MDH3 or MDH5. The gene Mdh3 codes for MDH1 or MDH3a, while Mdh4 codes for MDH3 or MDH7. The isozyme MDH4 is a hybrid product of MDH3 and MDH5. MDH6 is a hybrid between MDH5 and MDH7. The isozyme MDH5 can be formed as a hybrid between MDH3 and MDH7.

However, the isozyme MDH3a does not form detectable hybrids. This is exemplified by the discovery of an inbred line in our laboratory which expresses only mitochondrial MDH3a and MDH7. In this line no hybrid is seen between MDH3a and

MDH7. The model is supported by genetic, biochemical, and developmental data, and verified by other competent investigators elsewhere. We have undertaken a study to map the mMDH structural genes using trisomic analysis and B-A translocations. Using trisomic analysis, Mdh3 and Mdh4 were located on chromosome 6. B-A translocations have established that the structural genes Mdh3 and Mdh4 are on the long arm of chromosome 6. Additional experiments now being completed have allowed us to tentatively locate the Mdh1 and Mdh2 structural genes on another chromosome. The entire set of data will be shortly reported in a forthcoming detailed manuscript.

D. E. McMillin and J. G. Scandalios

Studies on the catalase inhibitor

In two previous reports, a catalase specific inhibitor which is differentially active in maize scutella during the first few days of germination, has been described. The molecule appears to be proteinaceous in nature, it is not a protease by several different criteria and its molecular weight, estimated by column chromatography is approximately 16,000 daltons. It inhibits catalase from different sources but not other enzymes and not even maize peroxidases, a group of catalytically related hemoproteins (Sorenson and Scandalios, MGCNL 49, 50). Recently evidence has been obtained that a protein in the glyoxysomal membrane inhibits catalase, that suggests that the inhibitor could be compartmentalized in the glyoxysomal membrane (Table 1).

Table 1. Inhibitor units in different scutellar cell fractions. Four days dark growing scutella extracts have been fractionated in a non-linear sucrose gradient and pure preparation of glyoxysomes and mitochondria fraction obtained. The organelles were disrupted by osmotic shock following centrifugation in high speed to separate the membrane (pellet) and the matrix (supernatant) material. The membrane pellet was then solubilized with detergents.

		Inhibitor Units
Crude Extracts		8
Glyoxysomes	Matrix	4
	Membrane	32
Mitochondria	Matrix	6
	Membrane	4
Top of the gradient	Supernatant	4
	Pellet	14

The inhibitor has been purified to homogeneity using conventional chromatographic techniques (DEAE-Sephacell, G-75 Sephadex) and affinity chromatography on immobilized beef liver catalase. This purified molecule appears to have a carbohydrate moiety on it since it positively stained by PAS staining and it is currently being characterized in terms of its MN in SDS denature gel electrophoresis, amino acid composition etc.

Specific antibodies have been raised by injecting rabbits with the purified molecule. Using the antibodies, we are currently trying to develop a double antibody precipitation radioimmunoassay for the inhibitor. The sensitivity of the assay and its independence from the presence of endogenous catalase will help us first to study more accurately its role in post-translation regulation, developmental expression, compartmentation and tissue specificity of catalase, during the

first days of seed germination. Secondly, the assay will help us to screen for inhibitor mutants in corn lines and corn seeds recently obtained from chemically mutagenized pollen. The recovery of any mutation will permit the genetic studies of gene(s) coding for the inhibitor protein. Towards that end we have labeled the inhibitor with ^{125}I for use in the radioimmunoassay.

Using different treatments, we have been able to perturb quantitatively catalase gene expression during the first days of germination. Culturing, for instance, isolated scutella in agar media containing 0.1% H_2O_2, we have been able to duplicate catalase activity. On the other hand, by adding the drug Allylisopropylacetamide (AIA), catalase levels decrease to almost zero values. These changes in catalase levels were always inversely associated with changes in the activity of the inhibitor. Blocking, for instance, catalase synthesis with AIA during the first days of germination, higher levels of inhibitor activity (assayed against purified catalase) has been obtained, contrary to the controls where on catalase peaks during the 3-4th day of germination the inhibitor drops to almost zero values (Sorenson and Scandalios, Plant Phys., 57:351, 1976).

Athanasios Tsaftaris and John Sorenson

Isolation of polyribosomes from maize

We have developed techniques for isolating free, membrane bound, and total polyribosomes from developing maize endosperm.

Field grown W64A was harvested at 18 days after pollination and frozen at -20 C. Freezing in liquid nitrogen did not substantially increase total yield or the proportion of higher order polyribosomes. Kernels were homogenized in the following grinding buffer: 0.25 M sucrose, 0.2 M Tris-HCl (pH 8.5), 0.06 M KCl and 0.03 M $MgCl_2$ plus 0.1 mg/ml heparin, 4 mg/ml bentonite and 1 mg/ml dithiothreitol. A 3 tissue:1 buffer volume ratio was found to work well while using a Waring blender for homogenization. For homogenization with a mortar and pestle a 9 tissue:4 buffer volume ratio was used. Homogenates were filtered through cheesecloth to remove cellular debris. The filtered homogenate was centrifuged at 16,000xg for 15 minutes to obtain a supernatant containing polysomes. Supernatants were then layered on a discontinuous gradient consisting of 10 ml 2.5 M sucrose in gradient buffer and 8 ml 1 M sucrose in gradient buffer. The gradient buffer was 0.04 M Tris-HCl (pH 8.5), 0.02 M KCl and 0.01 M $MgCl_2$ (Larkins and Hurkman, 1978). Centrifugation of samples at 90,000xg for 4 hours using an SW27 rotor at 26 Krpm resulted in the banding of polysomes at the sucrose interface. Polyribosomes were removed by aspiration.

Total, free cytoplasmic and membrane-bound polysomes were all isolated using this basic technique. For isolation of total polysomes 10 mg/ml sodium deoxycholate and 13% Triton X-100 were added to the grinding buffer in the above procedure. Separation of free and bound polysomes was accomplished by using the procedure as described without detergents in the grinding buffer to yield free polysomes in the 16,000xg supernatant. The pellet from this was then resuspended in the detergent buffer (that for total polysomes) and recentrifuged at 16,000xg for 15 minutes; the supernatant in this step contained bound polysomes. All other procedures were the same for isolating these three polysome populations.

Polysome profiles for total, free and bound polysomes were obtained by sedimenting polysomes suspended in gradient buffer on 12.5 ml 150-550 mg/ml sucrose gradients. Samples were centrifuged at 190,000xg for 135 minutes in a Beckman SW-41 rotor. Gradients were then monitored at 254 nm while being pumped through a flow cell using a dense chase solution of sucrose.

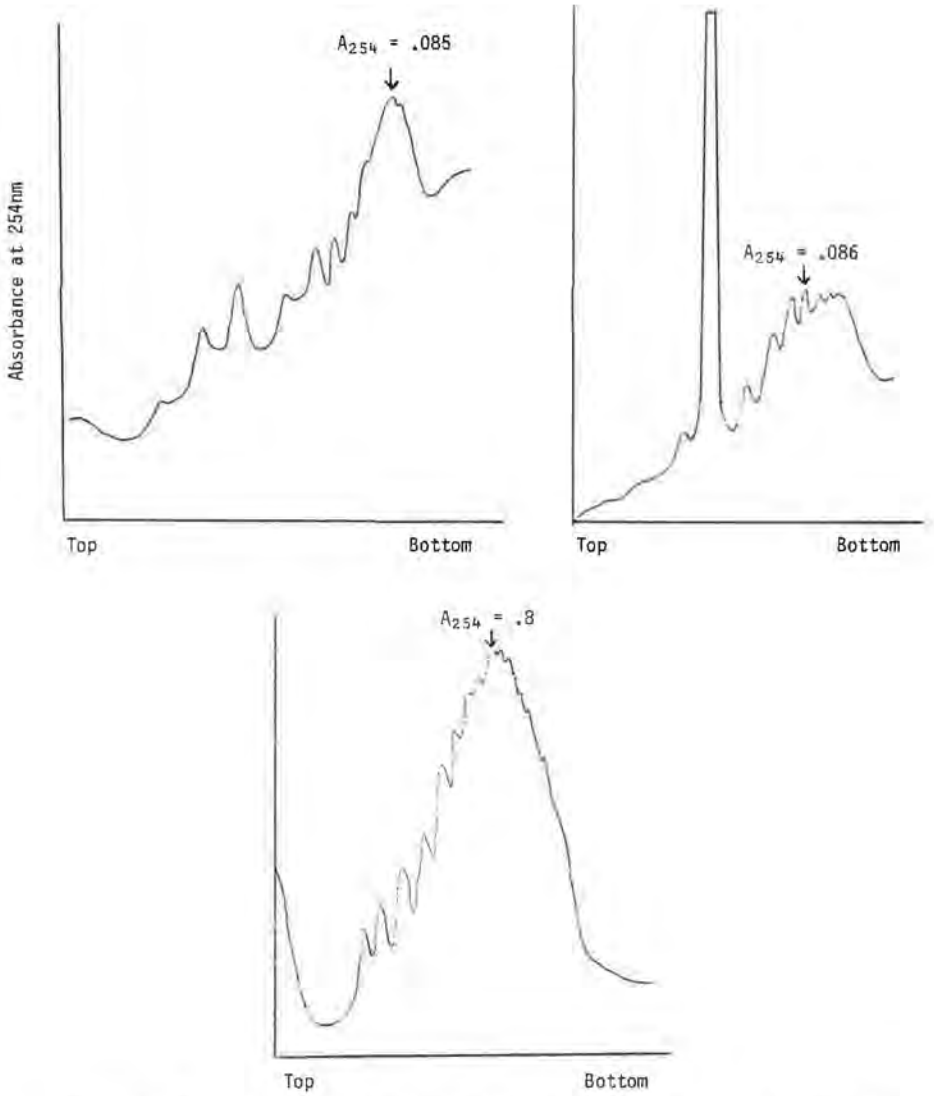

Figure 1. Sucrose density gradient sedimentation of polyribosomes from 18 day post-pollination W64A endosperm. Aopproximately 2 A_{260} units of polysomes were sedimented on 15-55% w/v linear sucrose gradients and monitored at 254 nm. (A) is a profile of bound polysomes, (B) is a profile of free polysomes, (C) is a profile of total polysomes.

Polysomes obtained in these experiments will be used for isolation of mRNA to be used in in vitro translation assays in an attempt to determine those polysome fraction(s) responsible for synthesis of the Cat1 isozyme found in maize immature endosperm.

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The basis for cytoplasmic instability in S cytoplasm

The S group is one of several sources of cytoplasmic male sterility (cms) in maize. It is distinguished from the other cms groups by its fertility restoration pattern. Furthermore, two plasmid-like DNAs are uniquely associated with mitochondrial preparations from cms-S maize (D. R. Pring, C. S. Levings III, W. W. L. Hu, and D. H. Timothy, PNAS 74:2904-2908, 1977). These DNAs have molecular weights of 4.1 and 3.5 x 10^6 daltons and occur in addition to the usual high molecular weight mitochondrial DNAs (mtDNA). So far these plasmid-like DNAs have been observed in every source of the S cytoplasm studied. Conversely, these small DNA species have not been detected in normal (fertile), T or C cytoplasms.

Unlike T and C, S cytoplasm has proven to be unstable (J. R. Laughnan and S. H. Gabay-Laughnan, 1978, Maize Breeding and Genetics, D. B. Walden, ed.). Hundreds of cases have been observed in which cms-S plants have reverted to male fertility. The majority of these reversions have been shown genetically to have arisen by cytoplasmic change rather than nuclear change, although this has recently been shown to be primarily determined by the nuclear genome (see contribution this News Letter, Laughnan and Gabay-Laughnan, University of Illinois). This report deals with biochemical studies of the cytoplasmic revertants.

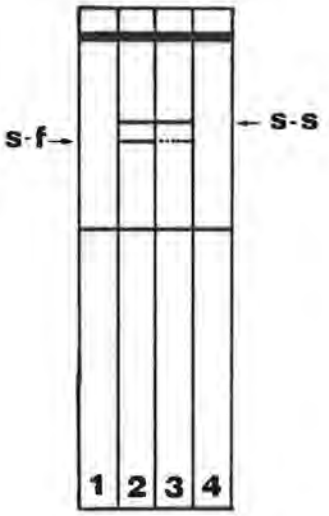

Fig. 1. Diagrammatic presentation of gel electrophoresis of mtDNA preparations from (1) Normal cytoplasm, (2) S cytoplasm, (3) Vg (M825 version) cytoplasm, (4) Vg (revertant) cytoplasm.

MtDNA preparations were made from the various cytoplasmic types and fractionated by gel electrophoresis. The results of these analyses are presented diagramatically in Fig. 1 where the mtDNA preparations are from the following types: lane 1, normal cytoplasm which is male-fertile; lane 2, S cytoplasm which is male-sterile; lane 3, Vg (M825 version) cytoplasm which is male sterile; lane 4, Vg (revertant) cytoplasm which has reverted from the male-sterile to the male-fertile condition. The Vg cytoplasm is a member of the S group of cytoplasms. In the M825 inbred, Vg cytoplasm is highly unstable; about 10% of plants exhibit reversion, and over 90% of such reversions occur at the cytoplasmic level.

All four cytoplasmic types contain a main band of high molecular weight mtDNAs as well as a fast migrating small (1.2×10^6) molecular weight DNA. The S cytoplasm, lane 2, contains the two plasmid-like DNAs, S-S and S-F, in equimolar amounts. The S-S and S-F DNAs are not found in normal cytoplasm, lane 1. In lane 3, the Vg cytoplasm (M825 source) has the two plasmid-like DNAs, but they do not occur in equimolar amounts; the S-F DNA species is present in reduced quantities. Lane 4 contains the mtDNA preparation from a cytoplasmic revertant which has changed from the male-sterile to the male-fertile condition. In this case, the two plasmid-like DNAs, S-S and S-F, are no longer observed.

Seemingly, the loss of the S-S and S-F DNAs is correlated with the cytoplasmic reversion from the male-sterile to the male-fertile condition. To date, five cytoplasmic revertants have been investigated, and in each case the plasmid-like DNAs were absent. This result constitutes compelling evidence that the genetic basis for cms-S resides in the mitochondrion, more specifically, that the S-type of male sterility is associated with the S-S and S-F DNAs.

Finally, it is clear that the unstable Vg (M825 version) cytoplasm is not a typical cms-S. It seems likely that the reduction in the amount of the S-F DNA species observed in mitochondrial preparations from this source may be related to the cytoplasmic reversions that occur so frequently in this strain.

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Reassociation kinetics of nuclear DNA

We have applied standard techniques of DNA reassociation analysis to determine the organization of the genome of Zea mays. DNA was prepared from seedlings, denatured, reassociated, chromatographed on hydroxylapatite, and the resulting reassociation data analyzed by a sum of squares curve-fitting computer program. The major finding is that the corn nuclear genome contains three kinetic classes of DNA: very rapidly reassociating (low Cot), fast reassociating (mid Cot) and slow reassociating (high Cot) DNA sequences; these three kinetic classes are general features of all eukaryotic DNAs so far examined.

Reassociation experiments were performed with DNA fragments sheared to an average of 1350, 1100, and 500 base pairs; only the data from the 1350 base pair material are presented here. At all fragment lengths, however, computer analysis demonstrates three major kinetic classes; changes in fragment length change the proportion of total DNA in each class due to the interspersion of the kinetic classes in the genome.

Low Cot, rapidly reassociating DNA: The low Cot DNA appears to be a large portion of the total genome (Figure 1) when whole genomic fragments of 1350 base pairs are analyzed; this is due to the interspersion of this component with mid and high Cot DNA. Minicot data (Figure 2) of this kinetic class, defined as DNA bound to HAP at Cot 0.1, show that this component is composed of several subclasses. About two-thirds of the minicot curve is a component of very low $Cot_{\frac{1}{2}}$ (0.0025) indicative of palindromic DNA and extremely repetitive DNA; the remaining one-third of the DNA is a portion of the mid-repetitive class with a $Cot_{\frac{1}{2}}$ of about 2.88 in this figure. The complexity of the very rapidly reassociating DNA calculated to be about 20% of the genome is 2×10^6 daltons.

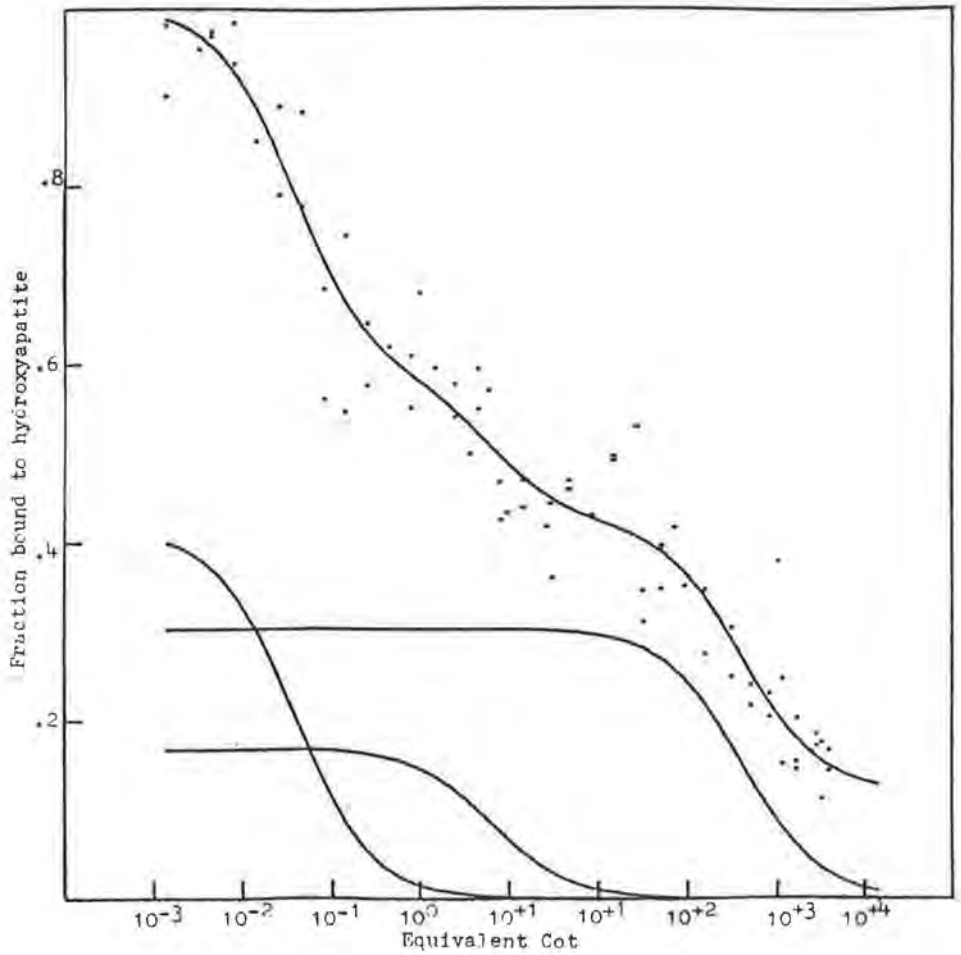

Fig. 1. Reassociation of unfractionated total DNA. Reassociation analysis was performed essentially according to Britten et al. (Methods in Enz. 29:363). The fraction of DNA that was bound or unbound was determined by measuring the A_{260nm}.

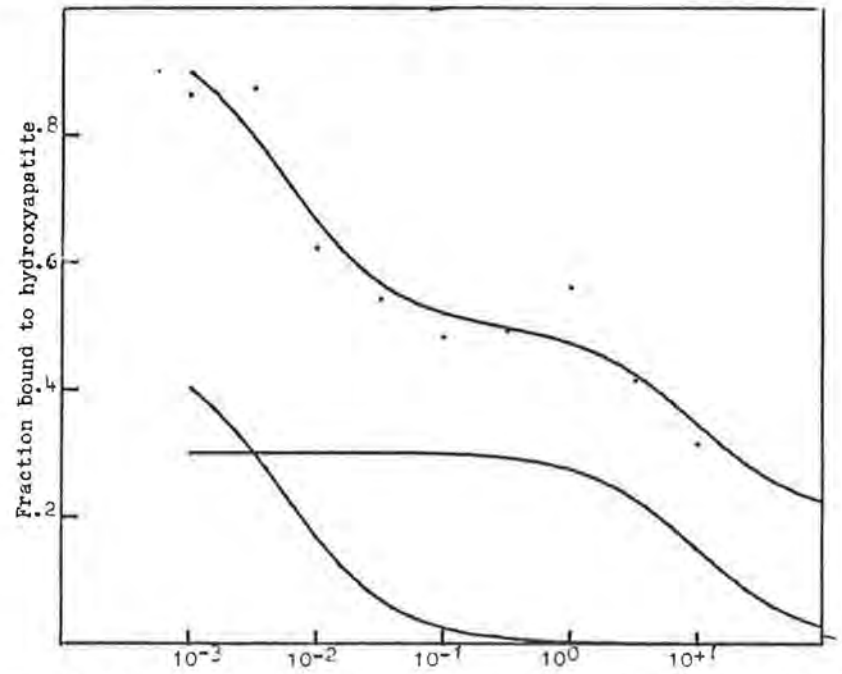

Fig. 2. Reassociation of highly repetitive DNA (bound at Cot 0.1).

Mid Cot, fast reassociating DNA: When whole genomic DNA is analyzed the middle repetitive component is about 20% of the genome with a $Cot_{\frac{1}{2}}$pure of 1.059 and a complexity of 7.06×10^8 daltons. A more direct analysis of the Cot(pure) value is obtained from minicot curves (Figure 3). When mid Cot DNA is purified as DNA reassociated at Cot 100, not reassociated at Cot 0.1, a single component approximately 40% of the genome is found with a $Cot_{\frac{1}{2}}$pure of 0.917. Spillover of mid Cot DNA into the very rapidly reassociating component occurs at 1350 base pair fragment length in the whole genome preparations (Figure 1).

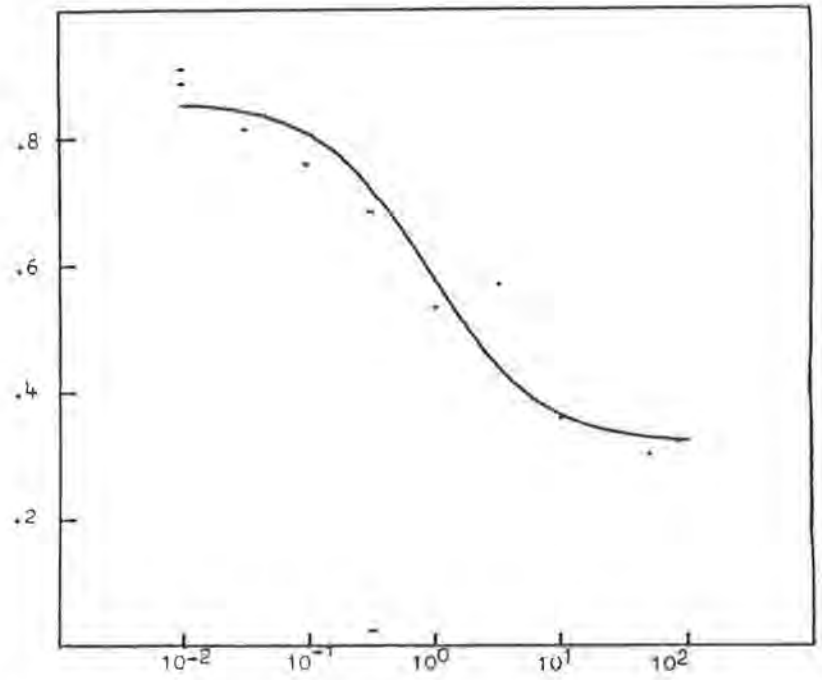

Fig. 3. Reassociation of mid-repetitive DNA (bound at Cot 100, unbound at Cot 0.1).

High Cot, slow reassociating DNA: The high Cot, presumably unique copy, DNA has a $Cot_{\frac{1}{2}}$pure of 1190 which gives a complexity of 7.93×10^{11} daltons; the unique copy DNA is about 30% of the genome. There would be approximately 8×10^5 different gene size sequences of unique copy DNA in the haploid maize genome.

Organization of the genome: As tabulated below, it is possible to calculate the reiteration frequency and number of individual sequences involved in the repetitive components. At 1350 base pair length there are approximately 80,000 copies of each very fast entity; if shorter DNA is analyzed, e.g. 400 base pair DNA, the apparent reiteration of this component increases to 470,000. Estimates of the reiteration of the fast, mid Cot component range from 400-1,300 for each family. The variability in these estimates is due to the relationship between the length of molecules used in the experiment compared to the actual average length of members of each kinetic class. Small changes in fragment length can produce profound changes in the apparent reassociation kinetics.

We have analyzed the average lengths of members of each kinetic class by electron microscopy. EM measurements of S1 nuclease-treated duplexes at mid Cot values give an average of 500 base pairs for the repetitive DNA sequences. The length of the average unique sequences was obtained by reassociating DNA of different fragment lengths to Cot 50. As fragment length increases, the percentage DNA in duplex increases due to single-stranded tails of unique DNA connected to the reassociated

Reassociation Curve	Component	Fraction of Fragments[a]	Cot½pure[b]	Complexity	Reiteration[c]
1350 base pr	very fast	0.41	0.015	1×10^7	79,333
	fast	0.17	1.059	7×10^8	1,123
	slow	0.30	1190.	7.9×10^{11}	1
400 base pr highly rep.	very fast	0.47	0.0025	1.7×10^6	476,000
	fast	0.30	2.88	2×10^9	400
400 base pr mid. rep.	fast	0.54	0.917	3.4×10^8	1,300

[a]The fraction of DNA fragments which contain a duplex region.

[b]Cot½ = 1/K where K equals the second order rate constant. Cot½pure = (Cot½) (Fraction of fragments)

[c]A value of 1 is assumed for single-copy components. The number of copies is Cot½ single-copy/Cot½ component.

repetitive DNA. An estimate of the length of the unique DNA is obtained from the breakpoint in % duplex vs. fragment length; the length of the interspersed unique sequences from this analysis is 2100 base pairs.

Using this information, it is possible to formulate a model for the general features of organization of the genome. At Cot 50 and long fragment length, all the repetitive DNA and two-thirds of the unique DNA are HAP bound. Given the calculated lengths and distribution of kinetic classes, our data indicate that two-thirds of the unique and about one-twentieth of the repetitive DNA are in a short term interspersion pattern. The remaining one-third unique DNA is probably in a long-term interspersion pattern with large blocks of repetitive DNA.

Preliminary data from melting profiles were also obtained for native and reassociated DNA samples. The native DNA has a Tm of 86.5 in 1x SSC, hyperchromicity of 30%, and a dispersion 2/3 of 13 (H.R. Mahler & Dutton, G. J.M.B. 10:157, 1964). Reassociated DNA to mid-Cot values (Cot 100) has a Tm of 79.5, hyperchromicity of 12%, and a dispersion 2/3 of 18. This indicates that the repetitive duplexes have 6% mismatch, that only 40% of the fragments are in duplex (at 1350 base pairs), and that the heterogeneity is much greater than native DNA. DNA reassociated to higher Cot values (Cot 3000) has a Tm of 85, hyperchromicity of 20% and dispersion 2/3 of 15.5. Thus there is less mismatch at the higher Cot values but still a small percent (1.5%), indicating some of the duplex molecules are not perfectly reassociated. The melts and further data will be published elsewhere.

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Tissue culture of monoploid maize

Genetic methods of producing monoploids in maize can be combined with current tissue culture techniques. The indeterminate gametophyte (ig) method of generating monoploids (Kermicle, 1966, Science 166:1422-1424) is particularly convenient because parental monoploids are produced in a useful frequency. This allows the inbred A188, or any genotype which readily produces tissue cultures capable of plant regeneration, to be used as the male parent in crosses with W23 ig. The resulting parental monoploids have the nuclear genome of the male parent and should readily culture.

The R-nj marker system (Nanda and Chase, 1966, Crop Science 6:213-215) of identifying monoploids by mature seed anthocyanin pigmentation has been used extensively. Although previous work has shown that it is possible to identify and recover monoploid cultures from immature embryos using R-nj as a marker, the frequency was lower than expected (Green and Donovan, 1978, Intl. Congress Plant Tissue and Cell Culture, Calgary, Canada, p. 157). This may have been due to variability in R-nj pigmentation in immature embryos isolated at a size suitable for producing cultures capable of plant regeneration. Experiments varying the growth media components, both organic and inorganic, have not improved the pigmentation response of immature embryos. Several R alleles, R-sc, R-scm:2, and R-scm:3, have been tested and produce scutellar pigmentation in immature embryos more reliably than R-nj. Crosses have been made to combine ig with each of these three alternate R alleles. The homozygous ig R (R-sc, R-scm:2, R-scm:3) plants will be crossed by A188 and embryos isolated and cultured to compare the pigmentation response of the R alleles, and to establish haploid cultures capable of plant regeneration.

A second method of obtaining monoploid maize cultures is being tested. Isolated shoot apices of fourteen-day-old seedlings can be used as explant tissue for initiating cultures capable of plant regeneration (Rice et al., 1978 Symp. Propagation of Higher Plants Through Tissue Culture, Knoxville, Tenn.). This allows a seedling marker, such as purple coleorhiza, glossy, or yellow-green, to be used in the identification of monoploids. For example, seed from crosses of W23 R-nj ig x A188 gl can be screened for scutellum color and the monoploids tentatively selected. After sterilization and germination, the remaining hybrid diploid seedlings can be discarded and the parental monoploids (glossy seedlings) sectioned for callus initiation. Root tips are easily obtained at this point for cytological confirmation of monoploidy.

The seedling method potentially offers several advantages over the immature embryo technique. First, it permits the use of both seed and seedling markers, for more efficient selection of monoploids. Secondly, no alteration in normal gene expression is necessary, contrary to the early pigmentation needed with the R alleles in immature embryos. Thirdly, only the putative monoploids are handled under sterile conditions. In addition, putative monoploid seeds can be accumulated and germinated as needed to initiate the desired monoploid cultures. Lastly, cytological confirmation of monoploidy coincides with callus initiation.

Carol A. Rhodes and C. E. Green

Tests for cytoplasmic restoration of nuclear genetic male sterility

F2 progenies from reciprocal crosses between inbred lines have been grown the past two summers to test for male sterility governed by an interaction between nuclear genes and cytoplasm. Such an interaction would be indicated by segregation for male sterility in F2's from all F1's of one cross, but no segregation in F2's from F1's of the reciprocal cross. Examples of this behavior have been reported in flax.

Tests in F2 of diallel crosses within one group of seven inbreds: A251, Oh43, N28, MS1334, K55, Mo17, and CI66; one group of six inbreds: A638, A495, A631, A73, A96, and NYN22; and another group of six inbreds: A660, A659, A344, A71-22, A648, and A632 are complete except A495 x A638, A96 x A73, A96 x NYN22, NYN22 x A96, and A659 x A660. Bulk seed of selfed F1 plants from many of the 7-line diallels and all but a few of the other F1's from those and the two 6-line diallels were available from the corn breeding project. A few new F1's were selfed in the Hawaii nursery. All F1's were tested by growing F2's from at least three different F1 plants. It will be evident from the results reported below that this latter

procedure is preferable for such tests. One of the three F2's from A344 x A659 segregated 3 normal:1 golden. The other two were all normal. It is being tested against g. One of the three F2's from N28 x Oh43 segregated 3 normal:1 intermediate dwarf. The three F2's from A344 x A660 segregated for pale green seedlings.

One of the three F2's from one cross, A660 x A648, did segregate 3 normal:1 male sterile. All the male sterile plants had short cobs, about two inches long, with silks very little longer than the cobs. The husks on several ears on male sterile plants were opened and pollen was applied. There were no seeds. In no case was there segregation for male sterility in F2's from all F1 plants of a cross. Such an occurrence in one cross but not in its reciprocal would indicate an interaction between cytoplasm and a nuclear gene. As shown in the next note, that result can be explained by either of two hypotheses.

The inbred lines in the above tests are being tested for cytoplasmic restoration of fertility by crossing them as the female parent with heterozygotes for ms; using y ms/Y + or Y ms/Y + stocks. These tests of many of the inbreds will be grown in 1979.

I wish to thank Dr. Jon Geadelmann and Robert Peterson for making seed of the inbreds, many F2's and F1's available for these tests.

Charles R. Burnham

Alternate explanations for male sterility governed by an interaction between nuclear genes and cytoplasm

During discussions of the usefulness of cytoplasmic restoration of fertility to nuclear genetic male sterility by Phillips and Albertsen, attention was directed to reports of male sterility in flax governed by an interaction between cytoplasm and nuclear genes. The first report was by Bateson and Gairdner in 1921. A procumbent strain, when used as the female parent with other varieties, segregated for male sterility in F2, but reciprocal crosses did not segregate. I found the same behavior for most crosses involving a variety from Crete, reported only briefly in my "Discussions in Cytogenetics." Although not recognized as possible examples of cytoplasmic restoration of fertility, the explanations used were essentially that. I thought it was a possible example until Dr. Phillips suggested that cytoplasmic male sterility interacting with restorer genes, the explanation used in corn, might fit the breeding results in flax also.

Assume: (c ms), (R), and (N) represent cytoplasmic male sterility, restorer cytoplasm, and normal (non-restorer) cytoplasm respectively. One possible explanation for the breeding behavior in flax is that a nuclear gene for male sterility (ms) is restored to fertility by a restorer (R) cytoplasm. On this hypothesis, procumbent and Crete are (N) Ms Ms and most other varieties are (R) ms ms.

The alternative explanation is: cytoplasmic male sterility (c ms) interacting with nuclear restorer vs. non-restorer genes, Rf vs. rf. On this hypothesis, the procumbent and Crete varieties of flax are (c ms) Rf Rf and most others are (N) rf rf. Either explanation will fit the results in flax. A paper is being prepared for Crop Science by myself, Albertsen and Phillips reporting data on the behavior of Crete and other varieties in flax and the kinds of tests needed to establish firmly a case of cytoplasmic restoration of fertility.

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Nuclear control over reversions to male fertility in cms-S maize strains

Spontaneous mutations (reversions) from male-sterile to male-fertile condition occur in plants with S male-sterile cytoplasm (cms-S). This event occurs more frequently in some inbred lines than in others, and may involve a change in the cytoplasm, corresponding to a change from cms-S to normal condition, or may involve a nuclear event, following which fertility is inherited in Mendelian fashion (J. R. Laughnan and S. J. Gabay-Laughnan, 1978, Maize Breeding and Genetics, D. B. Walden, ed.). We have also shown that nuclear integration of the fertility element may occur at different chromosomal sites, a property which led us to describe the element as an episome.

It is now known that some genetic backgrounds are highly favorable to this mutation and others are very stable. Moreover, among genetic backgrounds that exhibit rather high frequencies of the mutational event, there are striking differences in the proportions of mutations that are cytoplasmic versus nuclear in origin. In an attempt to determine the relative influence of cytoplasm and nucleus on both the frequency of the event and its origin, we undertook a backcrossing program involving a number of inbred lines carrying different subgroups of S cytoplasm as the nonrecurrent female parent, and the inbred line M825 as the recurrent male (maintainer) parent. Seven inbred lines that do not restore cms-S, that is WF9, 38-11, N6, K55, M14, I153 and IllA, and five subgroups of S cytoplasm, S, Vg, I, ML and Rd, were represented among the nonrecurrent female parents. In all, 14 different line-cytoplasm combinations were employed. Each of these, with the possible exception of WF9-Rd, can be characterized as highly stable, and for most, although they have been grown for a number of generations, no mutations from male-sterile to male-fertile have been observed. WF9-Rd plants have occasionally produced fertile tassel sectors but the frequency of this event is less than 1 percent. On the other hand, M825-Vg male-sterile plants, when crossed with the M825 maintainer that was used as the recurrent parent in this experiment, produce an abundance of mutations to male-fertile condition. Most of these occur as plants with fertile tassel sectors, many fewer as plants with entirely fertile tassels (entires), and the vast majority of these mutations are cytoplasmic in origin. Among 7,558 plants of this type that were analyzed for male-fertile mutations, 748 (9.9%) had fertile tassel sectors, and 74 (0.97%) had entirely fertile tassels.

The backcrossing program referred to above was undertaken without selection for mutational ability and is now in the ninth backcross step. In the summer of 1978 we replanted the second (3X) and sixth (7X) backcross generations of this experiment and looked for mutations from male-sterile to male-fertile condition. These exceptions were scored with respect to whether they were entires or sectors, and were pollen checked in the field to determine whether the pollen was essentially normal or half-aborted; the former is a preliminary indication of a cytoplasmic origin for the fertile revertant, and the latter signals a nuclear basis for the change.

The results indicate a strong influence of the genome of the M825 recurrent parent on both frequency and origin of the fertile event. The data in Table 1 indicate an increase in mutations after three generations of crosses by the M825 recurrent parent. Since the data from the 14 different line-cytoplasm nonrecurrent sources have been combined in Table 1, it is useful to know that the mean number of plants per source in the 3X group was 152, and that the numbers ranged from 95 to 312 per source. At least one fertile mutation occurred in 13 of the 15 sources; only the WF9-Vg and the 38-11-Vg sources, with 106 and 102 plants to score, respectively, had none. After an additional four generations of backcrossing

Table 1. Frequencies and pollen characteristics of male-fertile revertants in two different backcross generations involving 14 line-cytoplasm combinations as nonrecurrent female sources and the inbred line M825 as recurrent male parent.

	Frequency of event			Pollen analysis of revertants			
Generation	No. of plants	No. of revertants	%	No. of revertants	Normal	Semisterile	% normal
2nd backcross (= 3X)	2286	50	2.2	45	23	22	51.1
6th backcross (= 7X)	2044	169	8.3	156	149	7	95.5

by the M825 male parent (7X series) there is an overall fourfold increase in mutation rate, from 2.2 to 8.3 percent. All but one of the 14 sources showed an increase in mutation rate from the 3X to the 7X generation. The single exception was the Ill1A-S entry whose mutation rate in the 3X generation, 15/309 (4.9%) was highest among the fourteen sources. The rate for the Ill1A-S source in the 7X generation was 9/269 (3.3%), a reduction that is not significant. On the other hand, 5 of the sources exhibited striking increases in mutation rates in the 7X generation: WF9-Vg from 0/106 in the 3X generation, to 19/135 (14.1%) in 7X; WF9-Rd from 7/100 (7.0%) to 23/112 (20.5%); N6-Vg from 3/107 (2.8%) to 25/159 (15.7%); K55-Vg from 4/306 (1.3%) to 26/254 (10.2%); and M14-ML from 1/141 (0.7%) to 19/196 (9.7%). The overall mutation rate of 8.3% for the 7X generation approaches, but is still significantly lower than, the mutation rate of 10.9% for the M825-Vg strain whose maintainer is the recurrent parent in this experiment. Nevertheless, two of the 14 line-cytoplasm sources, WF9-Rd and N6-Vg (see above) had significantly higher rates of mutation than the M825 strain, and three of the sources had rates that were not significantly different from the M825 rate. To summarize, the near replacement of the inbred line nuclear genomes of 15 line-cytoplasm sources with the M825 nuclear genome results in an increase in spontaneous mutation rate from virtually zero to an average rate of 8.3%, which is close to the rate characteristic of the recurrent M825 strain itself.

As indicated above, an attempt was made to analyze the pollen character of each male-fertile mutation. We were able to score over 90% (201/219) of mutations for pollen type. Mutations that are cytoplasmic in origin produce mainly normal pollen which, when involved in test-crosses with cms-S male-sterile testers, produces all male-sterile progeny (except for newly occurring mutations). On the other hand, 50% of the pollen produced by a nuclear revertant is shriveled and aborted; since the normal grains in such a sample carry the equivalent of an Rf restorer allele, this pollen, when used on male-sterile testers produces all male-fertile progeny whose pollen characteristics are the same as those of the male parent. Among several strains of maize whose cms-S versions produce frequent mutations to male fertility, one designated wx bm4 has a high frequency of nuclear and a low frequency of cytoplasmic events. Another, M825/0h07, has about equal numbers of both. The M825-Vg strain whose maintainer counterpart was used as the recurrent parent in this experiment produces overwhelmingly cytoplasmic reversions to male fertility, the nuclear events being estimated at less than 5%. The summarized data on pollen character in Table 1 indicate that the nuclear genome exerts a strong influence over the type of mutational event. Though testcross data are not available yet, it can be inferred from pollen analysis that male-fertile

mutations in the 3X generation are cytoplasmic and nuclear in origin, in equal proportions. In the 7X generation, that is, after four additional backcrosses using M825 pollen, the mutations are mainly cytoplasmic, less than 5% being nuclear in origin. This trend is apparent in each of the 14 pedigrees. Moreover, this shift in proportion of cytoplasmic:nuclear cases is due to both an absolute increase in frequency of mutations with a cytoplasmic origin as well as an absolute decrease in frequency of revertants with a nuclear origin. Appropriate adjustments for the relatively few mutations for which pollen analyses are not available indicate that there is more than a 6-fold increase in the frequency of cytoplasmic mutations from the 3X to the 7X generation, from 1.1% to 7.3%. The corresponding values for the nuclear mutations are 1.0% and 0.36%.

Members of the S group of male-sterile cytoplasms carry two unique species of satellite mitochondrial DNA (D. R. Pring, C. S. Levings, W. W. L. Hu, and D. H. Timothy, PNAS 74:2904-2908, 1977). Recent studies (see North Carolina State contribution this News Letter) of five cytoplasmic male-fertile mutations by the agarose gel electrophoresis method indicate that the mutation in each case was associated with disappearance of these unique mitochondrial DNAs. It is apparent that the genetic basis for the S male-sterile trait is localized in the mitochondrion. Molecular hybridization studies carried out with labeled satellite mtDNA (B. D. Kim, C. S. Levings, D. R. Pring, M. F. Conde, J. R. Laughnan, S. J. Gabay-Laughnan and R. J. Mans, FASEB abstract, 1979) suggest that mutation of cms-S to male fertility is correlated with integration of at least some of the satellite DNA sequences into the mitochondrial circular genome. These studies are therefore consistent with the view that the fertility element in cms-S systems is an episome whose integration into the mitochondrial genome leads to a spontaneous cytoplasmic reversion to male fertility. Although there has as yet been no similar analysis of the spontaneous nuclear revertants to male fertility in cms-S strains, the above evidence supports the notion that these represent corresponding integrations of the F episome(s) into one or another chromosomal site. Of special interest in this connection is the finding (C. S. Levings III, W. W. L. Hu, D. H. Timothy and D. R. Pring, MNL 52:96-98) that both satellite DNAs from mitochondria of S male-sterile maize form lariat-like (lollipop) structures when they are denatured and allowed to renature under appropriate conditions. They are therefore similar to the numbers of insertional (IS) elements that have been described in numbers of prokaryotes, and, we like to think, admirably suited for the integration role they presumably play in the mutational events described here.

Whatever the details of the integration phenomena may be, the evidence from the study reported here indicates that the recurrent male parent, in this case M825, has the predominant influence over the frequency and localization of the initial event. In other words, the nucleus, not the cytoplasm, has primary control over both the frequency and the site of integration.

J. R. Laughnan and S. J. Gabay-Laughnan

Chromosomal location of 58 naturally-occurring cms-S restorers

Ten newly arisen restorers that were products of independent spontaneous mutations in S male-sterile cytoplasm (cms-S) have been under study. We believe they result from the integration of a cytoplasmic fertility episome at different chromosomal sites. Eight of these ten restorers have now been located to chromosome. Data indicate that restorers I and VIII are located in chromosome 8, restorers IV and VII in chromosome 3, restorers IX and X in chromosome I and, most recently, restorers III and VI in chromosome 2. Some of the inbred lines that ultimately derive from varieties through inbreeding, such as CE1, Tr, CI21E, Ky21 and C103, are homozygous for a naturally-occurring nuclear restorer that was

designated Rf3 by D. N. Duvick (Advan. Genet. 13:1-56, 1965). Rf3 is regarded as the standard restorer of cms-S. The Rf3 restorer, as represented in most, if not all, of the inbred lines studied is located in the long arm of chromosome 2 and these restorers appear to be allelic.

Only two of the eight newly arisen restorers located to chromosome are in chromosome 2. Since these new restorers occurred spontaneously, there is reason to believe that such changes should also occur in natural populations. In view of this it seems surprising that so many inbred lines with restoring capabilities for cms-S should carry a restorer located at the same chromosomal site. If, however, most such spontaneous events yield restorers similar to restorers I through X (J. R. Laughnan and S. J. Gabay-Laughnan, 1978, Maize Breeding and Genetics, D. B. Walden, ed.), they would, because of deleterious effects, be at a disadvantage from the standpoint of survival in maize populations. It is possible, therefore, that the Rf3 that is carried in established inbred lines does not carry these associated effects and has therefore survived in maize populations, and has become fixed in the homozygous state in some inbred lines developed from these populations. As a result of these considerations we searched among plantings of varieties of maize obtained from the North Central Regional Plant Introduction Station, Ames, Iowa, for naturally-occurring cms-S restorers. A total of 181 varieties were tested; 89 were nonrestorers and 92 were restorers of cms-S. The restorers identified in this way were tested to determine if they are located in chromosome 2. The procedure involved crossing the restorer strains as male parents with sterile cms-S rf In2a B/rf N b plants. Among the resulting progeny are plants that are male-fertile, have B plant color and carry the inversion 2a (breakpoints = 2S.75-2L.80); their genotype is Rf N b/rf In2a B. These plants are crossed to rf N b homozygous tester plants and the ratio of B to b is scored among the progeny. Since only Rf transmits through the pollen, deviation from a 1:1 ratio for B:b (excess of b) is an indication of linkage. Of the 92 restorer strains 26 had no test, 3 carried B in the tested strain, and 5 had too few progeny to test for linkage. Of a total of 58 that were successfully tested, 11 had ratios not significantly different from 1:1 and 47 had highly significant ratios, thus indicating chromosome 2 as the location of the restorer in 47 of 58 tested strains.

While the inversion testcross procedure used here identifies those restorers that are located in chromosome 2, it does not permit their localization within the chromosome. We have therefore made appropriate crosses of the chromosome-2-linked naturally occurring restorer strains with translocation strains rf T2-9b wx and rf T2-9d wx. Testcrosses of F1 offspring from this cross are expected to provide improved linkage information.

The inverted segment of In2a involves almost 80% of chromosome 2. Even so it is not possible to conclude that the 11 restorers that gave independent B:b ratios in this experiment are not in chromosome 2; if they are, however, they are not at the standard Rf3 position, and further studies are expected to resolve this question for at least some of these restorers.

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Isolation of chemical mutagen-induced oil content mutants of maize

Oil content in maize kernels is almost entirely under genetic control. Evidence for such control is the response of oil content to various selection methods (Sprague and Brimhall, Agron. J. 41:30-33, 1949; Sprague et al., Agron. J. 44:329-331, 1952; Jellum and Marion, Crop Sci. 6:41-42, 1966) and the inheritance of oil

content as an independent characteristic (Curtis et al., Agron. J. 48:551-555, 1956; Alexander and Lambert, Crop Sci. 8:272-274, 1968). It appears that from 20 to 40 genes are involved in the determination of oil content in the kernel and these genes exhibit incomplete dominance (Sprague and Brimhall, 1949; Miller, Agron. J. 43:229-234, 1951; Plewa and Weber, Can. J. Genet. Cytol. 15:313-320, 1973). Although many investigators have studied the genetics of oil production in corn, no point mutations that substantially influence oil content have been isolated. I proposed to induce oil content mutations with chemical mutagens. Mutant kernels would be detected and isolated by analysis of oil content with wide-line nuclear magnetic resonance spectroscopy (NMR) (Alexander et al., J. Am. Oil Chem. Soc. 44:555-558, 1967).

The maize lines used in this study were: R805, I-274, R84, IHO, ILO, R804, Alexho 197, Alexho 228, Alexho 724 and Alexho 274. The plants were exposed to the chemical mutagens ethylmethanesulfonate (EMS) and β-propriolactone (BPL). Each maize line was planted at the South Farms at the University of Illinois.

Within each line, pollen from each plant was pooled in a large test tube. The chemical mutagen was prepared in paraffin oil and applied to the pollen grains. The concentration of each mutagen in paraffin oil was 0.13% for EMS and 1% for BPL. The pollen grains were suspended in the mutagen for 20 min. The mutagen-pollen slurry was painted on the corn silks and sibling crosses were made within each line. Each M1 plant was self-crossed and 40 kernels from each M2 cob were individually analyzed by NMR. A representative sample of 10,564 kernels from 265 M2 families were tested for segregation with a Varian PAT-20 NMR.

Of the 265 M2 families analyzed for kernel oil content, five families exhibited an increase in variance. These exceptional families were MP631, MP663, MP707, MP784, and MP806. Families MP663, MP784 and MP806 were derived from IHO and families MP631 and MP707 were derived from R84. All exceptional families were from lines treated with EMS. If a recessive allele that affects the oil content of a kernel was induced by a mutagen, a 3:1 ratio of normal to mutant phenotype would appear in the samples from the M2 cobs. The data indicate a bimodal distribution and a 3:1 ratio was indicated for each family after analysis by the Chi-square test (Table 1).

TABLE 1

THE MEAN %, CHI-SQUARE, AND COEFFICIENT OF CORRELATION OF THE MAIZE FAMILIES SEGREGATING FOR KERNEL OIL CONTENT

Family Number	Mean %±SE High Oil	Mean %±SE Low Oil	χ^{2a}	r^b
MP631	6.10±0.23	2.08±0.23	0.01	0.47
MP663	13.40±0.31	4.54±0.36	0.83	0.13
MP707	4.84±0.11	2.60±0.22	0.17	0.50
MP784	11.71±0.20	3.34±0.59	0.03	0.52
MP806	9.88±0.34	2.30±0.34	3.77	0.19
MP4049-1	12.34±0.42	2.85±0.26	0.06	0.06

[a]The Chi-square values for every family indicated a 3:1 ratio (P>0.05).

[b]Coefficient of correlation was determined for kernel weight and oil content.

The lower kernel oil content in the presumed mutants could be due to a smaller germ, decreased concentration of oil in the germ, or both. If a mutation that lowered the oil content of a kernel did so by reducing the size of the germ, and

if endosperm weight was unaffected, there would be a correlation between kernel weight and oil content. The low oil kernels should weigh less than the high oil kernels of a segregating family. A coefficient of correlation (r) was calculated for the percent oil of the kernel and kernel weight for each exceptional family (Table 1). Families MP631, MP707, and MP784 have fairly high positive r values. There appears to be a relationship between kernel weight and oil content in these families, and the lower oil content in the segregants may be due entirely or partially to decreased germ size. Families MP663 and MP806 have very low r values and this indicates that there is no significant correlation between kernel weight and oil content. The low oil content segregants should have the same percent germ of the kernel as their high oil siblings. Thus the low oil kernels in these families are due to a decreased lipid concentration within the germ.

A low oil kernel (4.2%) and a high oil kernel (14.6%) were selected from family MP663 and the percent germ of each kernel was determined. The percent germ for the low and high oil kernels were 25.8% and 23.9%, respectively. I conducted a linear regression analysis (Figure 1) for percent germ and oil content from data compiled since 1898 (Dudley et al., 1974, Seventy generations of selection for oil and protein in maize, Crop Sci. Soc. Am., Inc., Madison, WI). For a kernel that

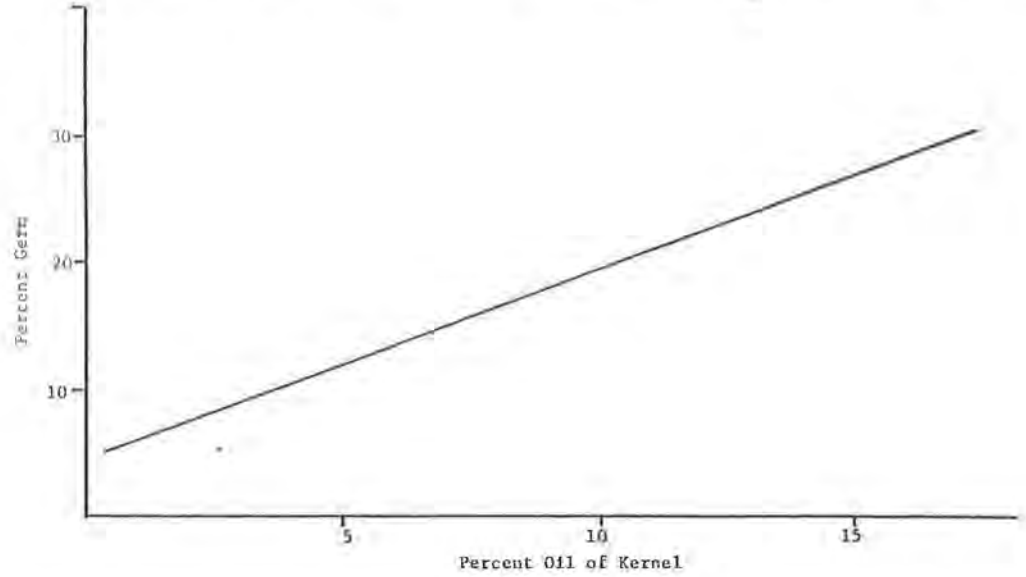

Figure 1. Linear regression analysis for data concerning kernel oil content and percent germ. Linear regression analysis of data collected from sources presented in table 20 of Dudley et al., 1974. Seventy Generations of Selection for Oil and Protein in Maize. Crop Sci. Soc. Am., Inc., Madison WI.

has 24% germ, the oil content should be approximately 14%. This agrees well with the 14.6% oil of the high oil kernel from family MP663. The low oil segregant has 4.2% oil and if this decrease in oil content was due entirely to a decrease in germ size, then one would expect a kernel with about 10% germ (Figure 1). This last series of relationships was verified by the lack of correlation between kernel weight and oil content. Thus, these data indicate that the low oil segregants of families MP663 and MP806 are the result of a mutation that affects oil concentration in the germ and not the amount of germ in the kernel.

To determine if the low oil content characteristic (loc) would act in a Mendelian fashion after a second generation, I planted kernels from family MP663. The kernels with low oil content ($\bar{X}$ = 4.54%), the presumptive homozygous recessives, produced albino seedlings. Albinism may be pleiotropic with loc or it may be a linked independently induced mutation. The presumptive heterozygote

(Loc/loc) and homozygous dominant (Loc/Loc) kernels with high oil content ($\bar{X}$ = 13.40%) produced normal seedlings. Each plant was self-crossed and 50 kernels from each cob were analyzed with NMR to detect segregation of loc. Four of the seven plants were heterozygous and loc segregated in a 3:1 ratio. Data for one heterozygote, family MP4049-1, is presented in Table 1. The r for kernel weight and oil content was only 0.06. There is no correlation between these two characteristics as was the case in the parental family MP663.

I speculate that loc is expressed in homozygous recessive embryos as a severe reduction in lipid content. In the sporophyte this lipid defect affects the chloroplasts resulting in an albino seedling. This speculation predicts that a single gene regulates embryo lipids (primarily triglycerides) and sporophyte lipids (primarily polar lipids). I further speculate that loc may be a regulatory gene rather than a structural gene. Studies are in progress to map the gene and to characterize the biochemical effects of loc in the embryo and sporophyte.

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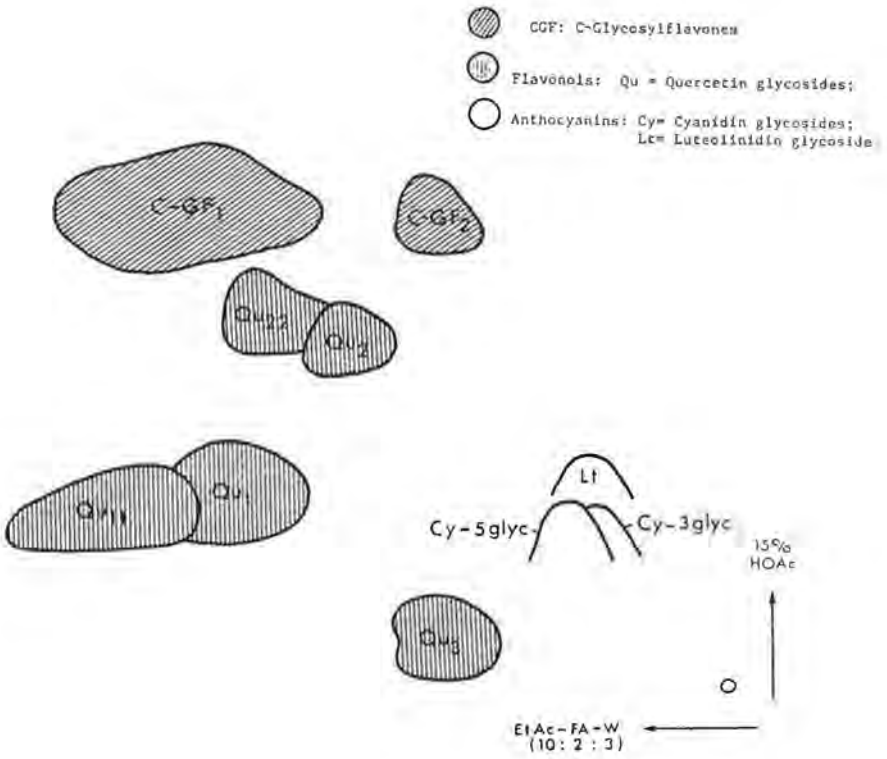
Flavonoids in coleoptiles of bronze seedlings

In the 1976 News Letter we presented a figure of a thin-layer chromatogram showing some of the more common flavonoids found in W22 seedlings. Quantitative comparisons with other lines are difficult in strains with strong anthocyanin concentrations because the anthocyanin spots tend to overlap those of other flavonoids. In bz seedlings, anthocyanins are rarely present, and then only in trace amounts. Quantitative measurements of other compounds are thus possible, in particular the flavones and flavonols. The bz allele affects flavonol concentrations as well as anthocyanins, of course, but the effect on flavonols seems to differ markedly in different backgrounds. We are presently investigating these background effects, and a summary of our findings so far is as follows (the figure shows those flavonoids specifically referred to in this article):

1. W22 P-WR A A2 R-r bz Pr B-b pl: Flavonols markedly reduced in comparison with Bz strains. Qu1 is the only flavonol present in measurable amounts. Flavone concentrations apparently unaffected by the bz allele.
2. K55 P-WW A A2 R-r bz Pr b pl (kindly supplied by E. H. Coe): Virtually no flavonoids in coleoptile. Other K55 Bz strains from Coe's stocks show only traces of flavones (as expected with the P-WW allele) and mostly Qu1 and Qu2 flavonols.
3. W23 P-WR A A2 R-r bz Pr b pl (again kindly supplied by E. H. Coe): Strong concentrations of flavones. Qu1, Qu11, Qu2, Qu22 and Qu3 all present, as well as several other minor flavonol spots. Some luteolinidin, and traces of cyanidin 3- and 5-glycosides. Other W23 Bz stocks from Coe show mostly Qu1 and Qu11, with Qu2 and Qu22 increasing with increasing anthocyanin concentrations. Some luteolinidin, but no Qu3 or cyanidin 5-glycosides.
4. F1 seedlings from crosses between the K55 and W23 bz stocks described above have approximately half the concentrations of all flavonoids found in the W23 bz parent. Some F2 seedlings have values in excess of those found in the W23 bz stocks. In general, the P-WR segregants have higher concentrations of all flavonoids, including flavonols. Most of the P-WW F2 seedlings, as well as P-WW seedlings from F1 x K55 bz backcrosses, have measurable amounts of all flavonoids, including flavones, in their coleoptiles.
5. P-WR A A2 R-r bz plants extracted from W22 P-WR A A2 R-r bz x P-WW a A2 R-g Bz (ex Co-op) by selfing show segregation for Qu3 concentrations; presence of Qu1 and Qu11 but traces only of Qu2 and Qu22; and conversely, presence of Qu2 and Qu22



but traces only of Qu1 and Qu11. Traces of cyanidin 5-glycosides are present in most plants. P-WW A A2 R-r bz sibs have reduced concentrations of all flavonoids.

6. C-GF1 is the flavone commonly found in most stocks that we have analyzed. A few stocks have C-GF2 as the predominant flavone. The difference appears to result from a single genetic factor, as far as we can tell not one presently recognized as being involved with any other phenotype.

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A hypothesis on the burn-blotch syndrome of Ia5125

The phenotype and environmental interaction of the bu (leaf burn) gene that occurs in the 5125 inbred of sweet corn suggest the following explanation. The bu gene may retard development or functioning of the plasmodesm connections passing through fine perforations in the bundle sheath wall in relationship to the transport demands of growth and photosynthesis. The supporting evidence is that when the growth rate is slowed down by environmental factors such as drought, low temperature or low light intensity, the burn-blotch phenotype is poorly expressed as if transport into the bundle sheath under these conditions is able to keep pace with slower growth. Under conditions favoring rapid growth, pale green blotches develop in which there is an accumulation of starch in the mesophyll with little or no starch in the bundle sheath. Necrosis sets into these paler areas, especially in younger rapidly growing tissue.

Although the thin-walled, loosely packed cells of the meosphyll may be specialized for photosynthetic activity, they are not adapted for starch storage. Thus, an accumulation of starch in such a C4 type of mesophyll could result in the death of the tissue. It seems that the bu gene represents a retrogressive step back toward the more primitive C3 system that can store starch in the mesophyll and lacks a bundle sheath specialized for such storage. In last year's Maize News Letter (52:58-59, 1978) we described the leaf-burning in 5125 as a monogenic trait bearing on C3-C4 photosynthesis.

Walton C. Galinat

On the low quality of certain sugary hybrids segregating shrunken-2

The new sugary hybrids that are segregating shrunken-2 are based on the development of viable sugary-shrunken inbreds that are used as one of the parents (usually the male). In selecting for increased germination approaching 100% in sugary-shrunken lines, we have found that selection is for a thicker tougher pericarp and a low wet percent of sucrose. The latter was measured with a hand refractometer on kernel juice pressed out in the milk stage. In all of the sugary-shrunken inbreds tested, the wet percent sucrose was only 15 percent which is at the low end of the scale for variation between standard sweet corn inbreds which range from 15 to 25 percent sucrose in their kernel juice. The low percent of starch in the sugary-shrunken combination gives it considerable taste preference despite its comparatively low wet percentage of sucrose.

But, what happens when we hybridize such sugary-shrunken inbreds with sugary inbreds of average quality? We obtain hybrid ears that carry 75% low quality kernels with 25% high quality sugary-shrunken kernels. The trade-off of so much bad for so little good leaves the balance in the red.

Walton C. Galinat and William F. Tracy

Dingy pericarp color as a complication to the breeding and use of the y ms system to eliminate detasseling

The photoelectric separation of white and yellow kernels in order to obtain a progeny of all male sterile plants from the white kernels depends on a reliable separation of the two kernel colors. Unfortunately, certain of the early sweet corn inbreds (C3 and C13) are like certain of the New England flints (Wilburs, Narraganset), in having a dingy pericarp color that obscures a clear-cut definition of mature white and yellow kernels. The obvious solution is to breed out the hindering pericarp color so that the phenotype of y is a snow white rather than a dingy or dirty white. The separation of white and yellow kernels may be further enhanced by the incorporation of orange aleurone and the elimination of any genes for pale or lemon yellow endosperm.

Walton C. Galinat

Small pollen in modern teosinte as a secondary adaptation for self-fertilization

Accepting the evidence that the fossil Zea pollen of 80,000 years ago was large, then its size would seem to be of independent origin from the large pollen now found in long-eared corn. The present-day evidence indicates that large pollen has a selective advantage over small pollen when the competition is within long styles. The selective pressure in wind pollinated plants that are widely dispersed is for a large shed of small pollen. In contrast with a localized dense stand, relaxed selection for small pollen would result in an increase in pollen size. On this basis, one might expect pollen that was larger than average in a perennial diploid

of teosinte. We shall soon have an answer on this aspect since such a perennial diploid has recently been discovered in Mexico (Iltis, in press). The perennial tetraploid teosinte does not have unusually large pollen.

If corn came from teosinte, we must then explain how most of the modern teosintes evolved pollen smaller than that of the ancient teosinte. It seems possible that small pollen has a competitive advantage over large pollen in short styles such as occur in teosinte while the reverse situation occurs in the long styles of corn. In short styles, the thinner tubes derived from small pollen may be able to penetrate more rapidly through the stylar tissue and, thereby, win in a short race. But in long styles, there is an accumulative lag in the descent of the organelles in the thin tubes resulting in a delayed fertilization. In contrast, the thick tubes from the large pollen of corn allow the organelles to successfully descend with the growth of the tube.

As corn and teosinte diverged during domestication, teosinte would become threatened with extinction by swamping with the domestic ear traits. There would be a selective advantage to small pollen that could win out in a short stylar race to the teosinte eggs. In certain isolated teosintes that did not either coevolve or coexist with corn as apparently with Jutiapa teosinte, the pollen remained moderately large or about the same size as that of primitive corn (Chapalote).

In testing these hypotheses, mixtures of corn and teosinte pollen are applied to short and long styles in which the fertilizing success of the larger corn pollen is marked by purple aleurone color. For style types it would be preferable to use teosinte where the competition is presumed to have taken place. From a practical point of view, the styles of corn may be shortened to about an inch in length, as is done in making corn-Tripsacum crosses, and the mixture of corn and teosinte pollen applied.

Walton C. Galinat

A miniature fruit-case type of teosinte as the wild ancestor of the first maize

A small rachis segment is expected in the connecting link between Andropogoneae and Maydeae in order to provide a smooth gradation in condensation level from Andropogoneae grasses such as Manisuris toward primitive maize such as the oldest Tehuacan cobs. In Manisuris the rachis segments are about 5 mm long and extremely slender with a cupule-like structure formed by a sterile pedicel positioned along one margin of the internode. Approaching the Tehuacán cob, a rachis segment about 3 mm long is expected, such as occurs in the smallest known fruit cases of teosinte. Any further condensation or reduction in the length of rachis segment results in a pinching off of the plastic primordia into additional ranks located in an area on the rachis with space available for further differentiation (as in Tehuacán cobs). Such 3 mm or miniature fruit cases are known in teosinte both archaeologically and at the low end of variation within present-day teosinte. C. Earle Smith, Jr. has found the carbonized remains of such tiny fruit cases from two sites (San Jose Magote and Tomaltepec) in the Oaxaca Valley that are dated about 3000 to 3400 B.P.

The apparent absence of teosinte in the premaize strata of ancient man's shelters and dump heaps may be explained by the difficulty in recovering and recognizing miniature sized fruit cases. A fine mesh screen such as is used to separate out Setaria seed is necessary for their recovery. The highly fragmented remains of carbonized teosinte fruit cases from the time frame of 8000 to 10,000 years ago could be easily overlooked in the deposited fill or lost in open camp

sites. Some of the intermediate products of incipient domestication may have been carried into isolated areas such as the Koster Site where these "missing links" wait to be discovered and, thereby, to document the transformation. Although present-day teosinte does tend to carry the early domestic trait for spike clustering, natural selection rapidly eliminates the other more harmful domestic traits.

Teosinte with tiny 3 mm long fruit cases was found during Beadle's "teosinte-hunt" near the village of Mazatlan in Guerrero in 1971 and then again near El Salada in 1972. In contrast, Chalco teosinte which coexists with maize in the Valley of Mexico has long (8.5 mm) thick fruit cases, partly because of its coevolution with the large-kerneled maize, partly to accommodate introgression from maize that carries factors for large kernels and, thirdly, to promote mimicry of corn during early vegetative growth. Jutiapa teosinte from Guatemala also has a long (8.0 mm) but more slender rachis segment as does Honduras teosinte, with a rachis segment 9.5 mm long.

Walton C. Galinat

On the usage of the terms pedicel and rachilla in description of the cob, the female spikelet and the grain in maize

The rachilla in the cob is commonly being called the pedicel while actually the pedicel is deeper in the cob and erroneously being called the spikelet trace. The difference is important because the onset of spikelet development is a regulatory switch point characteristic of the grass genotype. Glossaries define the pedicel as the foot stalk which elevates the pedicellate spikelet above the sessile one. The rachilla is the floral axis within the spikelet. In maize, the rachilla usually carries a pair of florets, each with a lemma and a palea. The lower floret on the ear does not usually develop. The relative lengths of the pedicel and the rachilla are inherited traits that are under separate genetic control.

On a larger scale of elaboration, the homologues of the pedicel and the rachilla are the uppermost internode of the shank and rachis or cob which terminates it. Like the spikelets, the ears may be paired with one sessile and the other pedicellate.

The correct usage of the terms pedicel and rachilla for the male spikelets in maize is clear as it is in most grasses. But when the terms are applied to the ear, there is confusion perhaps because of the extreme condensation, fusion and distortion of parts. The jargon "tip cap" of the kernel refers to a broken fragment of the rachilla together with its "chaff." The pedicel (spikelet trace) is fused into the floor of the cupule and sometimes buried further by a fusion of the roof of the cupule down onto the floor. The length of the rachilla together with the length of the pedicel and its associated cupule as well as the pith area constitute the thickness of the cob.

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A postulated origin of the B chromosome

Structural comparisons of K10 with the B chromosome suggest a direct origin of the B from a complete K10. I have traced Rhoades' photograph of a K10 bivalent from "Mutants of Maize," and darkened the more heavily stained regions of both long and short arms. Beside this tracing is my drawing of a B chromosome (Chromosoma 43:177, 1973). Regions of both the B and K10 have been numbered to

facilitate discussion. These chromosomes are remarkably similar from regions 3 to 7, and other differences are minor. Length and chromomere number distinguish region 2. The shorter length of this region in the B chromosome would follow from some condensation as this segment generally appears more heavily stained than euchromatin of the A set. The six chromomeres most usually seen in the B could have arisen by splitting of the three in K10. Perhaps the greatest difference between the drawings is in region 1. However, even here there is little difficulty. The distal euchromatic portion of K10 could heterochromatinize to become the minute short arm of the B. Degeneration of the original centromere and neocentromere formation in the knob would give rise to the new B chromosome centromere. An alternative for the progenitor of the B is K$_T$10 as described by Kikudome (Science 134:1006, 1961). Since no genes other than those dealing with B chromosome function have been found on this chromosome, loss of function of the k10 homologous region would have occurred by heterochromatinization. Because our attention has been focused on the large knob in the long arm of K10, we have been blinded to structural similarity of the B to K10.

Attempts to isolate B chromosome DNA have failed. Studies of DNA extracted from plants of an inbred line with and without B chromosomes could not distinguish B chromatin from the rest of the genome. Analyses of buoyant density and renaturation kinetics, as well as competition studies, led Chilton and McCarthy to conclude that B chromosome DNA was very similar to the remaining genome. These results are precisely those expected if the B arose from an intact K10.

Preliminary observations on C-banded chromosomes add credence to the possibility that B centric heterochromatin is similar to knob chromatin. Although a stock with K10 has not yet been analyzed, other knobs are stained very clearly, either in prometaphase or condensed metaphase root tip chromosomes. Only centric heterochromatin of the B stains heavily in this procedure. No other centric heterochromatin is similarly stained.

The greatest difference between activities of K10 and the B can be found in apparently very different accumulation mechanisms. If the B did arise from K10 we might expect a link between these apparently different systems. I suggest that neocentromere formation may be that link.

B chromosome nondisjunction is seen to be a consequence of delayed replication of the centric heterochromatin and neocentromere formation. Division of the centromere immediately adjacent to the neocentromere could begin pulling chromatids apart only after centric heterochromatin replication, by which time progress to one pole would be too well advanced to prevent nondisjunction.

Also important to consider is why the B chromosome does not undergo preferential segregation during meiosis. Miles has reported K10 derivatives with decreasing knob size that showed progressive reduction in ability to induce preferential

segregation. Loss of a sufficient amount of chromatin in the now centric heterochromatin of the B, could therefore render this chromosome incapable of inducing preferential segregation. It is also interesting that K_T10 has a reduced knob and lacks preferential segregation capacity.

Proposed mechanisms for B and K10 segregations are consistent with the interpretation of results obtained by Rhoades and Dempsey in their high loss line. In this line, knobbed segments of A chromosomes were eliminated at the second microspore division when two or more B chromosomes were present in the cell. These observations were interpreted as being a consequence of such late replication of knobs that bridges formed at anaphase. Deficient chromosomes and knobbed acentrics resulted from breakage of these bridges. Arguments presented above lead to a slight modification of the hypothesis advanced by Rhoades and Dempsey. Neocentromere formation in the unreplicated knob region would produce a morphologically different bridge; but completion of replication following a break would give the results reported.

A major implication of the suggested origin of B's is homology between these chromosomes and abnormal chromosome 10. Snope could find no supporting evidence in haploids and Ting's report of synaptic homology has been questioned by Rhoades and Dempsey. Loss of synaptic homology could have been an advantage if the B originated in a population segregating the progenitor K10 as a trisomic. Failure to pair with normal chromosome 10 would increase frequency of proper segregation of k10 chromosomes.

If I have stimulated anyone to think of B's and K10 in a different way, this letter will have been worth the effort.

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ADDENDUM

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The absence of fatty aldehydes in gl1 gl2 waxes

The modifications of chemical composition of surface waxes induced by the recessive alleles of nine independent loci have been recently summarized (Bianchi et al., Heredity, in press). As a general rule, in the mutations studied the reduction of the quantity of wax synthesized was accompanied by an enhancement of the percentage of long chain esters, while free alcohols and aldehydes decrease. Mutation gl5 was particularly interesting in accumulation up to 83.5% of free aldehydes compared to the normal where these long chain compounds reach 20.4% (Bianchi et al., Bioch. Gen. in press).

We report here the situation found in the double mutant gl1 gl2 where the aldehyde fraction disappears:

	Percent in the wax			
Component	Gl	gl1	gl1 gl2	gl2
Alkanes	1.4	6.3	3.0	12.1
Aldehydes	20.4	6.3	-	10.2
Alcohols	62.7	17.2	21.3	23.7
Esters	15.5	70.2	75.5	54.0

The absence of aldehydes in gl1 gl2 waxes is also accompanied by modification in the relative composition of free alcohol homologues.

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The construction of testers useful in the detection of dosage series at the kernel stage

For studying the effects of chromosomal dosage on enzyme levels, the B-A translocations have great potential. Roman (1947) suggested that TB-A's could be useful not only for investigating the inheritance of B chromosomes, but also for gene localizations and dosage studies. These translocations have since been extensively applied for localizing genes (Beckett, J. Hered. 69:27) but only minimally in dosage analyses. This has been the case because the appropriately marked genetic stocks have been lacking. In recent years we have begun developing special testers for several regions of the maize genome which, when combined with an appropriate TB-A, will allow the phenotypic distinction of the various doses (1-2-3) of the respective chromosome segment translocated to the B centromere. Some progress has been made, although considerable work remains. These testers involve either isozyme markers or the anthocyanin genes in combination with R-scm alleles, i.e. those alleles of the R locus which permit anthocyanin pigment in the

germ in the absence of the appropriate constellation of scutellum color factors (Sprague, U.S. Dept. Ag. Tech. Bull. 292). These systems involve modifications of testers previously suggested by Beckett and by Robertson (MNL 49:83) for identification of TB-A's. Robertson (J. Hered. 58:152) suggested the use of R-scm2 for this purpose and developed TB-10La with this allele and TB-9Sb with C-I for crosses to R-scm. The stocks described here extend its use to other anthocyanin loci. The complete set is not finished, but those that are may be useful to cooperators for dosage studies, induction of new TB-A's, construction of compound TB-A's, studies of nondisjunction or chromosome breakage at the second microspore division and the facilitation of the maintenance of TB-A stocks. The present status of these lines is due in part to the willing assistance of Michael Freeling and Kathleen Newton. The isozyme marker systems will be described elsewhere.

The testers and chromosomal regions are as follows:

1L--A tester of the constitution A A2 C C2 R-scm2 Bz bz2-m has been constructed. This stock is bronze in phenotype, but expresses full color in the presence of Bz2. The R-scm allele allows the expression of anthocyanin in the scutellum as well as in the aleurone. Thus when crossed by a TB-1La euploid heterozygote carrying Bz2 in 1L, the following phenotypes will correspond to the respective doses of 1L: bronze scutellum, purple aleurone, 1 dose; purple scutellum and aleurone, 2 doses; purple scutellum and bronze aleurone, 3 doses. It is known from systems using Adh (alcohol dehydrogenase-1) as a marker on TB-1La that hyperploid 1 B1 pollen grains cannot compete with the euploid grains under normal pollination conditions.

3L--The A locus, which is required for anthocyanin production, resides in 3L. Consequently three mutant alleles, a-st, a-m, and a-m-1 were used to construct testers for 3L that are homozygous for R-scm on chromosome ten. The constitutions of these testers are a-st A2 C C2 R-scm; a-m A2 C C2 R-scm; and a-m-1 A2 C C2 R-scm. If A is added to the embryo or aleurone genotype, anthocyanin will be expressed. When these testers are crossed by pollen from TB-3La euploid heterozygotes with A in 3L, the following phenotypes will correspond to the various doses of 3L: colorless scutellum and colored aleurone, 1 dose; colored scutellum and aleurone, 2 doses; and colored scutellum and colorless aleurone, 3 doses. E. Ward (Ph.D. dissertation, Indiana) found that hyperploid pollen for 3L could not successfully compete.

4L--An A A2 C c2 R-scm tester has been synthesized. This stock allows anthocyanin production in the scutellum and aleurone when C2 is added to the genotype. It is useful in distinguishing the various doses of 4L when crossed by compounds TB-7Lb-4L4698, TB-9Sb-4L6504, and TB-1Sb-4L4692 in a manner as described for 3L. In these cases it is necessary to use hyperploid heterozygotes since crossing over in euploid heterozygotes will regenerate the original TB-A used to construct the compound. The degree of crossing over between the A and B^{A-A} chromosomes in these hyperploid heterozygotes and the frequency of fertilizations by hyperploid pollen are unknown to the author.

5S--An A a2 C C2 R-scm tester should be completed after another season. It should prove useful for discerning the various doses of 5S using hyperploid heterozygotes of TB-1L-5S8041 with a2 in the normal chromosome and A2 in 5S. The class of hyperploid gametes which are duplicated for the 1L region distal to the 1L-5S(8041) breakpoint are not successful in competition (Birchler, MNL 52:29). The rare crossover derivatives that regenerate TB-1La can only form viable, competitive pollen grains when they segregate with the a2 marked normal chromosome five and will therefore be found in the colorless class of kernels. The complementary product of this recombination (1^5 with A2) can form a viable gamete in combination with the 5^1 chromosome. This class of pollen will produce completely colored kernels with two doses of all regions. Most of the kernels of this phenotype however will result from fertilizations by balanced 5^1 1^B B^{1-5} gametes in which disjunction of the B centromere occurred. These kernels will also have two doses

of all regions. Thus the colored scutellum, colored aleurone class will consist of kernels with two doses even though they may be of different chromosomal constitutions. Considering the genetic proximity of the A2 locus to the translocation breakpoint and the complex pairing in the hyperploid heterozygote, it is unlikely that the A2 allele would change its linkage relationship with the B centromere. This parameter has not been determined however.

5L--Dr. J. Kermicle has provided an A A2 C C2 R-scm122 pr stock. This line expresses anthocyanin in both the scutellum and the aleurone. The distinction in this case involves red (pr) versus purple (Pr) anthocyanin. In combination with a TB-5La euploid heterozygote with Pr in 5L, the various doses of 5L can be determined. The frequency of successful fertilizations by hyperploid pollen is not known.

7L--The Dt3 locus has been transferred to TB-7Lb. When this TBA is used in combination with the a-m-1 A2 C C2 R-scm tester described above, the Dotted gene can serve as a marker. At present the usefulness of this marker is limited for dosage studies, because we find that significant numbers of hyperploid pollen grains (7 B7) successfully compete with euploid grains in fertilization when the Esterase-1 isozyme marker is used. Consequently when the a-m-1 tester is crossed by a euploid heterozygote, the resulting kernels with dotted scutellum and aleurone are ambiguous as to whether they are disomic or partially trisomic. Perhaps by using a hyperploid heterozygote, 7 7B B7 B7 with dt3 in the normal 7 and Dt3 on B7, this problem could be circumvented. In this case the occasional transmission of only the chromosome 7 would produce colorless kernels that have two doses of 7L present. A complication in this case might arise from an occasional crossover between the translocation breakpoint and Dt3, which would again introduce ambiguity. We are currently attempting to introduce inversion 7e(S0.89-L0.93) into the a-m-1 R-scm stock to alleviate this problem.

9S--An A A2 c C2 R-scm tester has been synthesized and works in principle like the examples above when used in combination with TB-9Sb marked with C. Robertson (Genetics 55:433) has studied the mechanics of TB-9Sb.

10L--The TB-10L series induced by B-Y. Lin (cited in Beckett, J. of H. 69:27) have an R-scm allele present and are used with r testers.

We are currently attempting to transfer Dt2 onto TB-6Lc and also studying the feasibility of marking TB-1Sb with Mp in combination with the bz2-m tester. Anticipating the construction of a TB-A involving 7S, initial crosses for an in R-scm tester have been made.

For the maintenance of TB-A's, these tester lines have the advantage that they can be used repeatedly with the respective TB-A, obviating the requirement to alternate with other testers involving mutants on the same chromosome arm. That is, the use of hyperploid heterozygotes will generate the same classes of progeny with the same phenotypes generation after generation. Without the R-scm present, the use of an anthocyanin tester for TB-A's will result in kernels with colorless aleurones with hyperploid embryos. If these are used again onto the same tester, the normal chromosome with the recessive allele may be transmitted or an accidental self pollination of the tester would lead to an ambiguity in the classification of the colorless aleurone class, i.e. hyperploid heterozygotes or normal. The use of the R-scm tester series eliminates this ambiguity since the hyperploid heterozygotes will have colored scutella and the normals will be completely colorless. Although it is conceivable that heterofertilization might produce such a phenotype, the frequency of this phenomenon is very low in maize. Moreover, since the majority of successful male gametes are duplicated for the dominantly marked chromosome arm,

the vast majority of heterofertilizations with this phenotype would still be heterozygous hyperploids. Limited supplies of the following anthocyanin testers in combination with R-scm are available upon request: bz2, a-st, a-m, a-m-1 c2, pr, c, and TB-7Lb with Dt3.

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Meiotic studies of the P2 progeny of maize pollen-plants

Maize pollen-plants of the variety Eight-Row White were obtained with anther-culture technique. Those plants were then grown to maturity and intercrossed. Microsporocytes of 19 plants from the intercrossed progeny (P2) were collected and fixed with acetoalcohol (3:1) fixative for examinations. In the meantime, microsporocytes of the controls grown from the parental plants from which the anthers were originally derived were likewise prepared for comparison. The following characteristics of the P2 plants were found:

At pachytene stage, the chromosomes were generally well-spread. All of the cells were found to have 10 bivalents. No gross aberrations, such as inversions, translocations, deficiencies and duplications were observed. However, aberrant types of nucleolus were consistently present. These aberrant types varied from cell to cell and from plant to plant. The most commonly found types were rod-shaped, crescent-shaped and bell-shaped. In addition, more than 10 percent of the cells were found to have two nucleoli instead of one. Those two nucleoli were usually unequal in size and it was most likely to have the smaller one attached to the bivalent nucleolar chromosome, and the other remained free. These binucleolar sporocytes continued their binucleolar condition throughout the first meiotic prophase. The relationship between the nucleolus and the nucleolar chromosomes was very clear at diakinesis.

At anaphase I, chromatid bridges and fragments were also observed. In certain plants, more than 10 percent of the sporocytes had these kinds of irregularity. However, as stated in the foregoing, inversions were not definitely identified at pachytene stage. It is likely that these bridges and fragments are not the products of crossing-over within the inversion loop. They are probably caused by chromosome stickiness induced by certain mechanisms developed during in vitro growth. In view of the above findings it is reasonable to say that maize anther culture in vitro and regeneration through callus can bring about variations to the newly produced plants, since no such abnormalities were found in the controls.

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Maize tissue culture

Scutella of two diploid hybrids (77E-2, 77D-1) and one diploid inbred (sul/sul), and sections of apical meristems of one diploid inbred (77-01) were grown in vitro on modified MS (Murashige and Skoog, 1962) medium with 2 mg/l 2,4-D. After 10 days of culturing, more than 50% of the 252 explants grew into callus, irrespective of being illuminated or not. One month after initial growth of explants, callus pieces were excised and reinoculated into freshly prepared medium consisting of the same nutrients for continued culturing. One to two weeks after transfer, many calli developed into root-like structures. Particularly those which were originally kept

in light manifested profuse growth from primitive organ differentiations to teratomous expansions. These organ differentiations (root-like) soon sent out small branches with hairy appearance. It was also observed that some of the organ differentiations later cracked and a greening area exposed from inside. In about six weeks of subculturing, approximately 50 pieces of these developing calli were again cut off and reinoculated into regeneration medium prepared by substituting NAA for 2,4-D (2 mg/1). The rest of the calli were again subcultured on the same maintenance medium. Being on the regeneration medium for one week, these callus pieces began changing color from creamy-white to light-brown. In contrast to the other maintenance culture, their rate of growth was strikingly slow and their organ developments ceased. The original greening areas gradually became brown with compact instead of friable texture. Four to five weeks later, fine root-like organs appeared in some cultures and grew deep into the agar medium. But plantlets were not found. Even though regular transfers to newly prepared media for regeneration at four to six-week intervals were made for more than a half year, no regenerated plantlets were obtained.

Callus pieces from the maintenance cultures were excised and fixed for ordinary light microscopic examinations. It was found that cells in the friable and creamy-white calli were monstrous and extremely long, about 20 times longer than those in the normal tissues, while those in the hard and brown calli were round or nearly so, with enormous relative increase in size. However, due to limited amount of material available, the study in karyotype stability in various calli was still incomplete. Furthermore, controlled regeneration from maize calli into shoots and plantlets remains a problem to be solved. Maintenance of long-term tissue cultures of maize, nevertheless, can be carried out successfully.

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Chromosomes and genes of Chinese maize

Since last spring, F1 hybrids between 11 Chinese maize varieties and Wilbur's Flint (New England strain) were grown. Microsporocytes of these hybrids were collected and fixed in aceto-alcohol fixative. Meiotic chromosomes were examined by following standard aceto-carmine squash technique. Now studies of 51 plants from five crosses are complete, and those of the other hybrids are being carried on. Results of the latter will be reported in a late date, and those of the former are in the following:

The Chinese maize varieties employed in the foregoing five crosses were Tuo 229, Feng-1B, Tieh 84, Wu 3025 and Wu 105. The number of chromosome knobs of these five varieties varied from four to eight as shown in Table 1. It is also clear from this table that all of them had a knob on the long arm of chromosomes 4 and 7, either homozygous or heterozygous. In the long arm of chromosome 6, there was at least one knob for the five varieties. In addition, in three of the 10 F1 hybrid plants involving Tuo 229, chromatid bridges without fragments were observed at anaphase I of meiosis. However, no inversions or any other chromosome alterations were identified at pachytene stage. Fusions of knobs and centromeres were always present at pachynema in the 10 F1 hybrid plants involving Feng-1B. In the same hybrids, extrachromosome elements at pachytene stage, laggards, and chromatid bridges at anaphase I were also found. In the 11 F1 hybrid plants having Wu 105 as one parental variety, chromosome stickiness was consistently observed at diakinesis. Usually two, three or more bivalents were stuck together. The points of contact appeared at random. Material exchanges or chiasma-like configurations among the sticky chromosomes were not seen.

All of the above irregularities and knobs are from the Chinese maize, since the chromosomes of Wilbur's Flint were previously known to have no such irregularities and were also known to be practically knobless.

Table 1. Numbers and positions of chromosome knobs in Chinese Maize.
(L, S indicate long and short arms respectively of the chromosomes;
K, indicates homozygous knob, $\underline{K}$, indicates heterozygous knob,
KK two homozygous knobs.)

Varieties	CHROMOSOMES																			
	1		2		3		4		5		6		7		8		9		10	
	L	S	L	S	L	S	L	S	L	S	L	S	L	S	L	S	L	S	L	S
Tuo 229	$\underline{K}$		$\underline{K}$				K		K		KK		K							
Feng 1B	$\underline{K}$		$\underline{K}$				$\underline{K}$				K		K		$\underline{K}$ $\underline{K}$		K	K		
Tieh 84							K				K		$\underline{K}$				K			
Wu 3025	$\underline{K}$						$\underline{K}$				K				$\underline{K}$ $\underline{K}$					
Wu 105			$\underline{K}$		$\underline{K}$		K				K		K		$\underline{K}$			$\underline{K}$		

In the fall of 1978, from a selfed progeny of maize strain 7701, 51 seedlings were grown. Among them 32 plants had purple plumule, and the rest green. Even though the sample is small, only 51 individuals, it appears correct to say that the purple plumule character is governed by two pairs of dominant genes Pu1 and Pu2 which function complementarily. The ratio of 32 purple versus 19 green fits well the expected ratio of 9:7 for complementary genes.

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Dominant mutation Sup1-W70 o2/o2, improving opaque-2 endosperm pattern

After treatment of dry seeds of W70 o2/o2 gl/gl line with 0.582 mM solution of N-nitroso-N-methylurea kernels having a flint semi-transparent endosperm pattern were selected in M3. From normal corn the mutant thus obtained differed in a distinct pattern of a floury layer in the lower part of the kernel.

The genetic analysis showed that the character was controlled with a dominant gene which was not linked with an opaque-2 allele. At first sight the total data of the segregation account presented in Tables 1 and 3 contradict this conclusion and almost agree well with a semi-dominant type of the character inheritance. However, in F2 and testcrosses involving W64A line there was a perfect agreement between the actual observed segregation and theoretically expected one. All reciprocal A' x AM testcross combinations segregated modified kernels. Therefore, the numerous deviations from the expected ideal ratio of kernel classes may only be interpreted proceeding from the hypothesis of dominance.

Table 1. Segregation for an endosperm pattern in testcrosses

parent ♀	parent ♂	BC_1 kernel type: flint	modified	floury	χ^2	P
W 70 x W 70 o_2 mutant	W 70 o_2	1670	761	901	11.8*	< 0.01
W 70 o_2	W 70 x W 70 o_2 mutant	2590	57	2392	2167.9*	< 0.01
W 64A x W 70 o_2 mutant	W 64A o_2	602	285	299	0.6*	0.50-0.95
W 64A o_2	W 64A x W 70 o_2 mutant	288	139	148	0.3*	0.50-0.95
Cr25 x W 70 o_2 mutant	Cr 25 o_2	731	355	390	1.3*	0.50-0.95
Cr 25 o_2	Cr 25 x W 70 o_2 mutant	623	272	354	10.8*	< 0.01
A619 x W 70 o_2 mutant	A619 o_2	457	206	225	1.6*	0.20-0.50
A619 o_2	A619 x W 70 o_2 mutant	928	418	520	11.2*	< 0.01
Total		7889	2473	5229	976.6*	< 0.01
AM x A' crosses		3460	1587	1815	15.7*	< 0.01
A' x AM reciprocal crosses		4429	886	3414	1462.1*	< 0.01

* theoretically expected ratio 2:1:1

Careful analysis of the experimental data proves that the abnormalities in segregation are attributed to incomplete penetrance of a dominant allele (Table 2). The probability of gene penetrance is maximum at three doses and minimum at one dose. The good agreement between the observed segregation in F2 and the expected one calculated with the account of the degree of gene penetrance in testcrosses (χ^2 = 0.02, p = 0.99, Table 3) is convincing evidence of the adequacy of our interpretation.

Table 2. Degree of Sup1-W70 o2/o2 gene penetrance at various doses of a dominant allele (%).

Genotype	Gene dose ssS*	SSs*	SSS
W70	4.7	91.6	96.0
BC1(W64A x W70)	96.5	97.6	99.9
BC1(Cr25 x W70)	86.9	95.4	99.2
BC1(A619 x W70)	89.1	95.8	99.9
Mean	40.9	93.3	99.5

*Penetrance calculated considering the data of Table 1.

Table 3. Segregation for an endosperm pattern in F_2

Parent ♀	Parent ♂	F_2 kernel type: flint	modified	floury	X^2	P
W 70	W 70 O_2 mutant	9012	1445	1555	1150.9*	< 0.01
		(9009.0)**	(1443.8)	(1559.2)	(0.01)	(>0.99)
W 64A	W 70 O_2 mutant	1560	386	134	0.2*	0.50-0.95
		(1560.0)	(382.0)	(138.0)	(0.16)	(0.50-0.95)
Cr 25	W 70 O_2 mutant	2282	536	224	8.1*	0.01-0.02
		(2281.5)	(535.2)	(225.3)	(0.01)	(>0.99)
A 619	W 70 O_2 mutant	3719	872	360	11.7*	< 0.01
		(3713.2)	(881.6)	(356.2)	(0.16)	(0.50-0.95)
	Total	16573	3239	2273	773.4*	< 0.01
		(16563.7)	(3242.6)	(2278.7)	(0.02)	(0.99)

* expected ratio, ** the expected ratio calculated considering gene penetrance is in brackets
12:3:1

Instability of the gene manifestation is especially evident in W70 line. In F2 of this line there were some ears with a kernel ratio near to 12:2:2, 12:3:1 and 12:1:3. Some homozygous plants segregated kernels (on an average 4.0%) possessing a floury endosperm pattern. The test of the floury kernel progeny selected from such families indicated that the allele was not actually affected with irreversible changes and did not disappear. In the background of three other genotypes the phenotypical instability of the gene penetrance was less expressed against that of W70 line (Table 2). The reasons for this are not clear.

Table 4. Comparative characters of the corn with a modified endosperm pattern.

Index	Kernel endosperm type: Flint	Modified	Floury
100-kernel weight (g)	27.9	25.3	23.4
specific gravity (g/cm^3)	1.07	0.97	0.90
lysine content in kernel protein (%)	2.55	3.60	4.04
daily weight gain white rats (g)	1.56	2.83	3.22

The share of a floury part per endosperm depends on gene dose, genetic background and environments, and ranges from 5.0 to 90-95%. The partial restoration of a flint pattern in an opaque-2 endosperm results in a considerable improvement of some characters of high lysine corn (Table 4). On an average, 100-kernel weight

and specific gravity of the modified kernels are 8% higher. When infecting artificially and in natural conditions the resistance to Fusarium doubles. In lysine content and nutritional value the modified kernels are similar to the standard opaque-2 counterparts.

Thus, the dominant mutation partially suppresses a morphological and biochemical effect of an opaque-2 allele. The new gene is called a suppressor and is symbolized as Sup1-W70 o2/o2.

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The induced mutations increasing lysine level in corn kernels

Seven mutants with an increased lysine level in dry matter of an integrate kernel were found among 183 induced mutants with a modified endosperm (Table 1).

Table 1. Mutation effect on the accumulation of storage substances in corn kernels

Mutation	protein level (%)		lysine level			100 kernel weight (g)
	in kernel	in endosperm	in 100 g kernel a.d.m. (g)	in 100 g kernel protein (g)	in 100 g endosperm protein (g)	
A 344 +/+	12.7	11.6	0.292	2.3	1.8	22.5
A 344 o_2/o_2	13.2	12.1	0.554	4.2	3.2	20.0
fsh1 A 344	18.1	15.3	0.525	2.9	2.2	12.3
ffr1 A 344	13.5	12.2	0.473	3.5	2.6	12.5
ffr2 A 344	12.6	11.3	0.479	3.8	3.0	10.0
W 23 +/+	11.3	9.6	0.294	2.6	1.8	15.0
W 23 o_2/o_2	10.1	9.1	0.394	3.9	3.3	13.2
fww1 W 23	12.0	10.8	0.576	4.8	3.9	7.5
ffr3 W 23	11.6	10.7	0.406	3.5	3.0	10.0
W 64A +/+	14.3	12.6	0.300	2.4	1.6	19.5
W 64A o_2/o_2	14.2	12.8	0.568	4.0	3.0	16.0
fmd1 W 64A	15.3	12.8	0.566	3.7	2.8	7.5
ffr4 W 64A	15.4	12.4	0.508	3.3	2.4	14.0

a.d.m. - absulely dry matter

For all mutants a floury endosperm pattern was typical. The recessive mutants controlling this character were not allelic to the identified genes, o1, o2, fl2, o5. When symbolizing the mutants we tried to express the most typical characters of the phenotypical manifestation. The first two letters of the symbol reflect the changes in kernel pattern: f - floury endosperm, fr (or f) - friable endosperm, sh - shrunken endosperm, m - miniature kernel, w - white endosperm. The last letter as a rule shows a pleiotropic effect of the gene: d - dwarf, w - white seedling.

The mutation fsh1 A344 blocks the accumulation of storage substances in the endosperm. Therefore, protein content per kernel of the mutant primarily increases due to the contribution of the valuable embryo proteins. The four mutations designated as ffr loosen an endosperm to a greater extent than the identified opaque-2 allele. It results in less 100-kernel weight, in 1.4-2.3 times less, against the original alleles.

The mutation completely depressing kernel carotene and seedling chlorophyll synthesis is designated as fww1 W23. The mutation symbolized as fww1 W23 much surpasses an opaque-2 allele in its biochemical effect. When in a homozygous condition fmd1 W64A mutation proportionally decreases all kernel and plant parts.

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Reduced outcross seed set in 2NOR stock controlled by chromosome 4 gametophyte factor

The 2NOR strain, described by Phillips and co-workers, was used to show that the nucleolus organizer region is the chromosomal site of DNA complementary to rRNA (Chromosoma 36:79-88). When the initial crosses were made to establish W23 near-isogenic stocks of 2NOR and 1NOR, usually no seed was produced whenever the 2NOR stock was used as the female parent. From 55 exact reciprocal crosses, only 33 seeds were obtained when 2NOR was the female in crosses with W23 and 28 of these were from one cross. Normal seed set resulted when the 2NOR stock was used as the male parent in exact reciprocal crosses; from the same 55 exact reciprocal crosses, 20,186 seeds were obtained when W23 was the female parent and 2NOR the male parent. The 2NOR stock could be readily selfed or sibbed. A gametophyte factor present in the 2NOR stock was postulated.

A gametophyte locus (Ga) is located in the short arm of chromosome 4 approximately 35 map units distal to su. There are three alleles at this Ga locus; Ga, Ga-s and ga (J. Hered. 66:5-9). The Ga allele is cross neutral and will accept pollen of any genotype. However, Ga-s Ga-s plants can only be fertilized by Ga-s or Ga pollen; no seed set occurs with ga pollen. Heterozygous Ga-s plants can be fertilized by ga pollen in the absence of Ga-s pollen. If both Ga-s and ga pollen are present, the Ga-s pollen has a competitive advantage over the ga pollen.

Six reciprocal crosses were made between the 2NOR stock and a su su stock. The crosses with 2NOR as the male parent produced normal seed set, but the reciprocal with 2NOR as the female parent gave no seed set. If 2NOR were Ga-s Ga-s and the su stock were ga ga, the above result would be expected. If the proposed genotypes of the two stocks are correct, then (su su x 2NOR) F1 has the genotype su ga/Su Ga-s. If Ga-s pollen has a competitive advantage over ga pollen and su and GA are linked, then selfing the F1 would produce an F2 with less than 25% su kernels. The reduction in su kernels would be a function of the percent recombination between su and Ga-s. Only 17.5% su kernels will occur in the F2 if only Ga-s pollen effects fertilization and su and Ga-s are 35 map units apart. Since the su su F2 would be either su Ga-s/su ga or su Ga-s/su Ga-s, crossing su su F2's as the female with the F1 as the male would result in only 35% sugary kernels instead of 50%.

The results of self-pollinating the (su su x 2NOR) F1 are shown in Table 1, cross 1. Data for the 15 ears were combined because the heterogeneity chi-square was 12.17 with 14 d.f. (non-significant). The expected 3:1 ratio was not observed. The data fit the hypothesis of 35% recombination between su and Ga-s and only Ga-s pollen effecting fertilization. The su su parent, therefore, was su ga/su ga and

the 2NOR parent was Su Ga-s/Su Ga-s. The recombination frequency between Ga-s and su is 33.2%. Table 2 shows the results of crossing su su F2 plants by the F1.

Table 1. Gametophyte factor F_2 segregation tests.

Initial cross	No. of ears	Observed F_2			Expected F_2 (3:1)			Expected F_2 (35% recombination)		
		Su	su	Total	Su	su	χ^2	Su	su	χ^2
1. susu x 2NOR	15	5539	1103	6642	4981.5	1660.5	249.57**	5479.6	1162.4	3.67
2. susu x W23(2NOR)	5	2294	722	3016	2262	754	1.811	--	--	--
3. susu x W23(1NOR)	3	1540	496	2036	1527	509	.443	--	--	--

Table 2. Segregation tests of susu F_2 ♀ (su ga/su Ga-s or su Ga-s/su Ga-s) x F_1 ♂ (su ga/Su Ga-s).

No. of ears	Observed			Expected 1:1			Expected (33% Recombination)		
	Su	su	total	Su	su	χ^2	Su	su	χ^2
9	2582	1198	3780	1890	1890	506.7**	2532.6	1247.4	2.92

The hypothetical genotypes of the su su F2 would be either su ga/su Ga-s or su Ga-s/su Ga-s, and the genotype of the F1 is su ga/Su Ga-s. The data were combined because the heterogeneity chi-square was 10.48 with 8 d.f. (non-significant). The data do not fit the expected 1:1 ratio. However, the data fit the hypothesis of 33% recombination (being derived from F2 data) between Ga-s and su and only Ga-s pollen capable of fertilization. The recombination frequency between su and Ga-s obtained from the data in Table 2 is 31.7% (1198/3780 x 100), which is close to the value calculated from the F2 data. The results suggest that the 2NOR stock is most likely Ga-s Ga-s.

Tests were conducted on the W23 near-isogenic stocks of 2NOR and 1NOR to determine if the Ga-s allele was inadvertently selected in the development of these stocks. The results are presented in Table 1, crosses 2 and 3. The expected 3:1 segregation for su was obtained in the F2's of both su su x W23 (2NOR) and su su x W23 (1NOR). This indicates that the isogenics are probably ga ga at the chromosome 4 gametophyte locus.

Additional crosses will be made to determine if the Ga-s allele in the 2NOR stock is allelic to the previously described Ga-s allele in chromosome 4. The 2NOR Ga-s allele likely is allelic to the previously described Ga-s allele since the map distance from su is approximately the same.

T. J. McCoy, R. L. Phillips and P. J. Buescher

Introduction of genetic markers and interchanges into inbreds A188, A619, A632, and W23

The availability of genetic markers and chromosomal aberrations in common genetic backgrounds is a requirement for many genetic studies. Many of the mutants chosen for backcrossing mark the ends of the various chromosome arms. Most of the interchanges chosen for backcrossing involve the short arm of chromosome 6 mostly with breaks in the nucleolus organizer region or the satellite. The inbred A188

Table 1. Backcross of genetic markers into inbreds A188, A619, A632, and W23. Numbers in parentheses refer to number of backcrosses to A188, A619, A632, and W23, respectively. A hyphen indicates that no backcross seed is available.

Chromosome 1: sr (5, 2, 5, 6), br (3, 1, 5, 2), f (3, 1, 5, 2), an (-, 6, 1, 2), bm2 (3, 6, 5, 6)

Chromosome 2: lg (3, 5, 5, 6), gl2 (3, 5, 5, 6), B (4, 1, -, 6), fl (-, 5, 5, 2), v4 (-, 5, 5, 2), Ch (1, -, -, 2)

Chromosome 3: cr (2, 6, 4, 6), d (-, 3, 5, 6), lg2 (5, 3, 5, 6), a (4, 6, 4, 2), et (4, 6, 4, 6), pm (-, 4, 5, 6)

Chromosome 4: la (6, 6, 5, 2), su (6, 6, 5, 3), gl3 (6, 1, 5, 2), bt2 (4, -, -, 5)

Chromosome 5: a2 (6, 3, 5, 5), bm (6, 2, 5, -), pr (6, 2, 5, 5), v2 (1, 6, 5, -)

Chromosome 6: po (-, 3, 5, 6), rgd (3, 2, 5, 2), y (6, 3, -, 6), Pl (6, 6, 5, -), su2 (-, -, 3, 6), sm (-, 6, -, -), py (6, 6, 5, -) 2NOR (-,-,-,6)

Chromosome 7: o2 (4, 6, 5, 6), v 5 (6, 6, -, 6), ra (6, 6, -, 6), gl (6, 6, -, 6), ij (-, -, -, 3), bd (6, 6, 4, 6)

Chromosome 8: v16 (-, 5, 1, 3), j (6, -, 1, 6)

Chromosome 9: yg2 (-, 3, 5, 6), sh (-, 3, 5, 6), wx (3, 3, 5, 6), bm4 (2, 6, 5, 1)

Chromosome 10: oy (4, 6, 4, -), g (-, 6, -, 5), R-nj (1, 6, 4, 4), sr2 (-, 2, -, 2), bf2 (2, 3, -, 2). Abnormal chromosome 10 (6, 6, -, 6).

Table 2. Backcross of interchanges into inbreds A188, A619, A632, and W23. Numbers in brackets refer to number of backcrosses to A188, A619, A632, and W23, respectively. A hyphen indicates that no backcross seed is available.

Satellite - interchanges

1-6b [1, 1, 3, 4], 2-6 (001-15) [6, 3, 1, 2], 3-6b [6, 4, 2 4], 4-6 (7328) [6, 3, 2, 6], 4-6 (5227) [6, 5, 4, 6], 4-6 c [6, 6, 2, 5], 4-6 (003-16) [5, 4, 2, 6], 5-6b [6, 2, 1, 6], 5-6d [5, 4, 2, 6], 5-6 (8219) [6, 5, 2, 6], 6-7 (7036) [6, 5, 2, 5], 6-9 (017-14) [4, 5, 1, 3], 6-10f [4, 4, 2, 6].

NOR - interchanges

4-6 Li (actually 1-6) [5, 5, 2, 6], 1-6 (5495) [5, 1, 5, -], 1-6 (4986) [-, 1, 2, -], 1-6 (6189) [2, 5, 2, 3], 1-6 (8415) [4, 4, 4, 5], 2-6 (8786) [5, 1, 3, 6], 2-6 (027-4), [6, 3, 4, 6], 2-6 (5419) [6, 6, 5, 5], 2-6 (8441) [-, 2, 2, 1], 3-6 (030-8) [6, 4, 3, 6], 3-6 (032-3) [4, 3, 3, 6], 4-6 (4341) [6,5,4,6], 4-6 (7037) [5, 3, 5, 6],5-6f [6, 1, 5, 6], 5-6(8696) [2,5,4,6]6-7(035-3) [3, 6, 3, 6], 6-7 (5181) [5, 2, 1, 6], 6-7 (4964) [4, 4, 2, 6], 6-9a [2, 3, 4, 6], 6-9d [1, 3, 4, 6], 6-9 (4778) [6, 4 2, 6], 6-10 (5519) [3, 4, 4, 6], 6-10 (5253) [4, 6, 2, 6]

6S - interchange

1-6d [6, 1, 2, -]

Others

2-3e [1, 3, 3, 6], 2-9 (062-11) [5, 3, 1, 3], 2-10b [3, 4, 5, 2], 3-9 (6722) [3, 3, 4, 6], 6-7 (027-6) [1, 4, 4, 5], 7-9b [4, 4, -, 1], 8-9 (4453) [4, 4, -, 3], 8-9 (8525) [4, 2, 2, 2],

is excellent for tissue culture studies due to its propensity to regenerate plants from callus cultures. Inbreds A619 and A632 were chosen because of their superior performance in hybrid combinations. Inbred W23 is an extremely fertile line with excellent seed set and near zero background pollen sterility.

Seed is available on a limited basis for all the lines reported. We acknowledge the valuable assistance of Marc Albertsen, Tom McCoy, Steve Thompson, Pat Buescher, Warren Springer, Tim Murphy, Joe Ruegemer, and Clive Lake.

R. L. Phillips and A. S. Wang

Cytogenetic localization of a high chlorophyll fluorescence mutation (hcf-26) within the chromosome 6 satellite

A mutant (hcf-26) blocked in light reaction photosynthesis was shown to be controlled by a locus distal to the midpoint of the heterochromatic portion of the nucleolus organizer region (NOR) in chromosome 6 (Leto and Miles, MGCNL 51:57-59). The mutant hcf-26 possesses a yellow-green phenotype and was uncovered by TB-6a. In order to more precisely cytogenetically locate hcf-26, we crossed several heterozygous NOR--and satelite--interchanges as the female parent with heterozygous hcf-26 plants as male parents. Under our greenhouse sandbench conditions, seed from self-pollinations of the heterozygous hcf-26 plants resulted in green and nearly luteus seedlings. Several crosses between the heterozygous interchanges as female and heterozygous hcf-26 as male also yielded some near-luteus seedlings. The results suggest that hcf-26 is located in the distal chromomere of the satellite (Table 1).

Table 1. Positive tests to cytogenetically locate hcf-26. Only the smaller seed from crosses of heterozygous interchanges as female parent with heterozygous hcf-26 plants were planted.

Interchange	Breakpoints		Green	Chlorophyll deficient
NOR-interchages	6	other		
1-6(6189)	Het. 10[1]	1S.50	16	1
4-6(4341)	Het. 50	4S.36	13	1
2-6(8786)	Het. 88	2S.97	15	3
4-6(7037)	Het. 90	4L.61	19	2
6-9(4778)	Het. 95	9L.30	15	2
4-6(actually 1-6)Li	S.C. prox.	1L.81	19	1
2-6(5419)	S.C.-.25	2L.82	15	2
3-6(030-8)	S.C.-.25	3S.05	19	1
3-6(032-3)	S.C.-midway	3S.34	27	6
5-6f	S.C.-midway	5S.23	16	1
Satellite - interchanges				
4-6(7328)	Between prox. and middle chromomere	4S.53	16	2
4-6(5227)	Between middle and distal chromomere	4S.46	8	3

[1] Het. 10, for example, indicates the breakpoint is in the NOR-heterochromatin 10% of the distance from proximal to distal ends of the heterochromatic segment; S.C.-prox., S.C.-.25, S.C.-midway indicate the breakpoint is in the proximal portion, between proximal and mid-portions of the NOR-secondary constriction, or midway, respectively.

The lack of chlorophyll deficient plants in crosses with certain interchanges that should have uncovered hcf-26 may have been the result of incorrectly identifying a plant as an interchange heterozygote. These crosses will be repeated. Tests with interchanges that appear to have breaks in the most distal satellite chromomere (Phillips and Wang, MGCNL 51:52) all gave negative results but should be repeated.

Previous tests (Phillips et al. MGCNL 51:49-52) localized polymitotic (po) most likely to the first chromomere of the satellite by the same Dp-Df technique used above. Those results and those reported above for hcf-26 indicate that deficiencies for up to 90% of the NOR can occasionally be transmitted through the ovules. The hcf-26 marker located in the satellite and the rgd marker located most likely proximal to the NOR (Phillips et al. MGCNL 51:49-52) serve as seedling markers flanking the NOR.

The valuable assistance of Joe Ruegemer is gratefully acknowledged.

R. L. Phillips and S. A. Thompson

Stock request

Does anyone have seed of McClintock's "large satellite" strain?

R. L. Phillips

The following reference citations, received too late to incorporate in the main list, were helpfully supplied by Y. C. Ting:

Recent Maize Publications

1. Corn Institute, Kwangsi, Chuang Autonomous Regions; Experimental Station, Tung Pei Wang People's Commune; Hai Tian District, Peking; Institute of Botany, Academia Sinica, China. Induction of pollen plants in maize and observations on performance of their progenies. Proc. Symp. Ant. Cult. by Science Publisher, China, 11-17, 1978.

2. Ku Ming-kuang, Cheng Wan-chen, Kuo Li-chuan, Kuan Yueh-lan, An Hsi-pei, Huang Chiao-hsiang. Studies on induction of pollen plants in maize. ibid. 18-26, 1978.

3. Wang Yu-ying and Kuo Chung-chen. In vivo flowering and fruiting of pollen plants in maize. ibid. 27-28, 1978.

4. Li Pao-sen, Ma Chieh and Yang Kuei-shu. Screening of medium "762" for anther culture of Zea mays L. ibid. 211-213, 1978.

5. Wu Chia-lin, Chung Chiu-lan, Huang Shi-ling and Cheng Pi-lan. Transplantation, chromosome doubling and characteristics of pollen plants in Zea mays L. ibid. 237-238, 1978.

6. Li Shu-chin and Tsui Wen-fu. A preliminary report of anther culture of Zea mays L. ibid. 262, 1978. (Abst.)

7. Seed Amelioration Farm, Science and Technique Station, Red Star People's Commune, Peking. Experiments on induction of pollen plants in maize. ibid. 262, 1978. (Abst.)

8. Haploid Breeding Group, Pai Chuan College of Agronomy, Honan Province. Propagation and differentiation of callus tissue in corn. ibid. 263, 1978. (Abst.)

9. Hsia Hsiao-chang. Application of petroleum growth substances to increase induction and survival rates of pollen plants in wheat and maize. ibid. 262, 1978. (Abst.)

10. Ting, Y.C. Genetics in People's Republic of China (including maize genetics, breeding and tissue culture). BioScience 28(8): 506-511, 1978.

DEPARTMENT OF PLANT BREEDING

CORNELL UNIVERSITY

ITHACA, N. Y.

April 12, 1929

TO STUDENTS OF MAIZE GENETICS:-

You who attended the "corn club" in my hotel room at the time of the winter science meetings in New York will recall that I promised to prepare a summary of the published data involving linkage groups in maize, to add my own unpublished data, and to send those records to each of you for criticism and the addition of such unpublished records as you may care to furnish me. I am now enclosing the records promised, but can claim no credit for having assembled them. Professor Fraser had, before leaving for a year in Europe, abstracted the available published papers. Mr. Beadle has completed that work, has assembled my own unpublished records, and has arranged all the tables and charts.

I hope that each of you, whether or not you attended the New York meeting, will send me such relevant data as you have not yet published, showing either linkage or independent inheritance. In so far as you have data ready for publication, I prefer to receive a copy of your manuscript, but shall be glad to have also records which you are not ready to publish, if you care to send them. I agree not to publish any such data without your consent and in any case to give proper credit. Any records sent, however, should be with the understanding that I am at liberty to use them in an early revision of the mimeographed sheets for distribution to other workers, pending the publication of the general linkage paper which I have been threatening to bring out for some years now.

I indicated at New York that the records were too incomplete to warrant publication now, a fact made strikingly obvious by the "rainbows" on the maps. The distribution of the data in mimeographed form should serve temporarily the needs of those actively studying maize genetics; and others can wait. The coordination of effort agreed to in New York should go far toward straightening out many of the question marks in the next year or two.

In this connection, I add here, as a reminder, a list of those to whom linkage groups were parcelled out at New York.

C-Wx group - Eyster, Bucknell; Beadle, Cornell.

R-G group - Lindstrom, Jenkins, Wentz, Ames.

Su-Tu group - Emerson, Cornell.

B-Lg group - Stadler, Missouri; McClintock, Cornell.

Y-Pl group - Hill, Cornell.

P-Br group - Emerson, Cornell.

Ba-Gl_1 group - Brewbaker, Minnesota; Brunson, Manhattan; Fraser, Cornell.

Pr-V_2 group - Eyster, Bucknell; Jorgenson, Ohio; Li, Cornell.

D-Pg_2 group - Not assigned.

A-Ts_4 group - Brink, Wisconsin; Li, Cornell.

To those not at the New York meeting, it should be explained that this assignment of groups was, so far as possible, made in accordance with the expressed interests of those assuming the responsibilities entailed. It was far from our purpose to preempt groups for ourselves and thereby warn off other workers. Our purpose rather was to make sure that each known group would be given immediate and adequate attention to the end that the not very exciting job of chromosome mapping may go forward with some dispatch, thereby making possible an attack on certain important genetic problems now awaiting just such tools as accurate linkage maps afford. It should go without saying therefore that the help of those of you who were not at the New York conference will be welcomed.

I suggest that those who have made themselves responsible for any group, request needed material directly from the workers most likely to have it, as indicated by the names in the last column of the table for that group. We at Cornell shall be glad to furnish on request tester stocks in so far as our somewhat limited supply will permit. It would doubtless be helpful if those who have particularly desirable testers for any group would prefer them to the ones who are primarily responsible for that group.

Sincerely,

R. A. Emerson

GENERAL NOTES

Linkage data.-

In the last column of the tables giving the linkage data for the several linkage groups, papers from which the records have been summarized are indicated by author and year. Not all published data are included. For instance, F_2 data are omitted when abundant back-cross data are available. Records credited to an author without indicatio of the year are unpublished. In general, unpublished data received in personal correspondence are not included, except when no publishe records are available. Such data are doubtless incomplete. It is thought, therefore, that workers will prefer to add their complete data as of the spring of 1930.

X and Y in the column headings of the several tables indicate the dominant genes of the first column and x and y their respective recessive allelomorphs.

In the second column under the heading "Link. phase", C = coup ling and R = repulsion, Bc = back-crossed and S = selfed.

Data presented in the table of three-point tests are included in, not additional to, data in the several group tables. The first co of this table shows the genotype of one parent only, the other par having obviously the respective allelomorphs of the genes of parent no. 1. The genotypes involved in columns 2 - 5 will be clear from the following illustration:

Parent No. 1	Parental combinations No. 1	No. 2	Region 1	Region 2	Regions 1 and 2
C sh Wx	C sh Wx-c	Sh wx	C Sh wx-c	sh Wx	C sh wx-c
Sh Wx	C sh wx-c	Sh wx	C Sh wx-c	sh Wx	C sh wx-c
I Sh wx	I Sh wx-i	sh Wx	I sh Wx-i	Sh wx	I Sh Wx-i
sh wx	I Sh wx-i	Sh Wx	I sh Wx-i	Sh wx	I Sh Wx-i

lons.-

No attempt has been made to indicate map distance other than by observed cross-over percentages; 3 mm. = 1 per cent crossing over.

Starred genes (*) are those located with reasonable certainty; others probably belong in the general region indicated.

A gene tested with only one of the located genes is placed opposite that gene at a distance determined by the cross-over percentage, its locus being approximately at one end or other of the "rainbow".

Independence of linkage groups.-

This chart shows what tests have been made between genes of any one linkage group and those of other, presumably independent, groups. Thus, there are records involving approximately 9000 individuals in 5 selfed parents indicating independence between C or I and A and approximately 2000 individuals in back-cross progenies indicating independence of sh and A. It is obvious that the data are not adequate to establish the independence on all the groups, and it is hoped that other workers will have unpublished data to fill in some of the "holes". As an example of the necessity of obtaining more nearly adequate data, a manuscript by Hayes and Brubaker (received after the stencils for the linkage tables had been cut) indicates that gl_{-11} belong to the B-Ig group, while Beadle's unpublished records suggest that gl is in the C-wx group. The independence of these two groups is, therefore, questionable.

C-SH-WX GROUP

List of Genes

ar	Argentic - finely striped leaf	Eyster 1929
au_1	Aurea chlorophyll-yellow plant	Eyster 1929
au_2	Aurea chlorophyll-yellow seedling	Eyster 1929
bp	Brown pericarp with a	Meyers 1927
C	Colored aleurone with A and R	East and Hayes 1911
d_3	Dwarf plant	Suttle (Unpub.)
de_{15}	Defective endosperm	Brink 1927
~~fl~~	~~Floury endosperm~~	~~Hayes and East 1915~~
~~gl_2~~	~~Glossy seedling~~	~~Hayes and Brubaker 1925~~
gm_1	Germless	Eyster 1929
I	Inhibitor for aleurone color	East and Hayes 1911
pk	Polkadot leaf	Eyster 1929
v_1	Virescent seedling	Demerec 1924
v_{14}	Virescent seedling	Phipps (Unpub.)
v_{15}	Virescent seedling	Phipps (Unpub.)
w_{11}	White seedling	Demerec 1926
wx	Waxy endosperm	Collins 1909
yg	Yellow-green plant	Jenkins 1927

Notes

pk — The 1929 data of Eyster on pk are not consistent with his earlier data. He makes the statement in his 1929 paper that pk and ar show relatively close linkage--hence pk probably lies on the wx side of C.

d_3, w_{11} — In the material on which the d_3 and w_{11} counts were made, the C and R factors were segregating. Demerec states that a calculation of the recombination percentage with C would suggest that both d_3 and w_{11} were on the wx side of sh but that a calculation on such material could not be depended on.

au_1 — The location of au_1 to the right of sh is somewhat doubtful. Recombination values with C and sh are based on separate progenies. Neither au_1 or au_2 have been tested with yg for allelomorphism.

v_{14} — v_{14} is known to be located in the C-sh-wx linkage group but the data (Phipps unpub.) are of such a nature that a recombination value cannot be calculated.

Linkage Data

Genes X	Y	Link. phase	Number of individuals XY	Xy	xY	xy	Total	Recombinations No.	%	Authority
[illegible]	Wx	R Bc	115	340	298	92	845	207	24.5	Bregger '18
		C Bc	858	310	311	781	2260	621	27.5	Bregger '18
		C Bc	371	115	125	397	1008	240	23.8	Kempton '19
		C Bc	2542	717	759	2710	6708	1456	21.7	Hutchison '22
							42511	9588	22.1	Stadler '25
							249663	67402	27.0	Collins and
							302995	79314	26.2	(Kempton 27
C	Sh	C Bc	4032	149	152	4035	8368	301	3.6	Hutchison '22
		C Bc	10077	366	397	9865	20706	763	3.7	Eyster '29
		R Bc	638	21379	21096	672	43785	1310	3.0	Hutchison '22
							72649	2374	3.3	
Sh	Wx	R Bc	1531	5991	5885	1488	14895	3019	20.3	Hutchison '22
C	Sh	C Bc	9452	384	402	9377	19615	786	4.0	Hutchison '22
[illegible]	Wx	C Bc	1487	584	547	1520	4138	1131	27.3	Hutchison '22
		R Bc	790	2217	2283	792	6082	1582	26.0	Hutchison '22
							10220	2713	26.6	
C	V_1	R Bc	300	676	711	294	1981	594	30.0	Demerec '27
[illegible]	V_1	R	70	84	40	3	197	[illegible]	7±	Demerec '24
[illegible]	Pg	C S[1]	128	6	54	56	244	[illegible]	2	Eyster '24
		C S[2]	148	5	128	92	373		2	Eyster '24
Sh	Pg	R S[2]	140	61	60	2	263		10	Eyster '24
		R S[3]	382	173	173	11	739		24.5	Eyster '29
		R Bc[3]	73	363	366	70	872	143	16.4	Eyster '29
Sh	D_3	R S	329	162	138	8	637		22.8	Demerec '26
[illegible]	W_{11}	R S	487	193	161	16	857		31.2	Demerec '26
		C S	320	26	25	67	438		13.4	Demerec '26
[illegible]	Yg	Q S					30817		20.5	Jenkins '27
		R S					3885		23.0	Jenkins '27
		R Bc	10	57	52	7	126	17	13.5	Jenkins '27
Sh	Yg	R Bc	193	546	429	99	1267	292	23.0	Jenkins '27
		R S	2583	1212	1057	89	4941		28.6	Jenkins '27
Wx	Yg	C Bc	397	289	297	412	1395	586	42.0	Jenkins '27
		R Bc	78	120	136	80	414	158	38.2	Jenkins '27
							1809	744	41.1	Jenkins '27
[illegible]	Wx	C S	4075	461	1609		6145		13.4	Brink '27
D_{15}	Sh	R S	2449	1146	1237		4832		16.5	Brink '27
[illegible]	Bp	R Bc	9	56	49	9	123	18	14.6	Noyes '27
C	Ar	R Bc[4]	2178	4692	4166	1507	12543	3685	29.4	Eyster '29
Sh	Ar	R Bc[4]	1925	4763	4177	1221	12086	3146	26.0	Eyster '29
Sh	Au_1	C S	2108	311	310	492	3221		21.6	Eyster '29
[illegible]	Au_1	C S[1]	546	79	638	305	1568		26.5	Eyster '29
Sh	Au_2	R S[4]	340	133	146	10	629		28.0	Eyster '29
Sh	Gm_1	C S	2693	301	258	702	3954		15.3	Eyster '29
Wx	V_{15}	R S[3]	297	128	139	2	566		19	Phipps
Sh	V_{15}	R S[3]	366	172	172	5	714		20	Phipps

[1] C and R segregating - 9:7 ratio

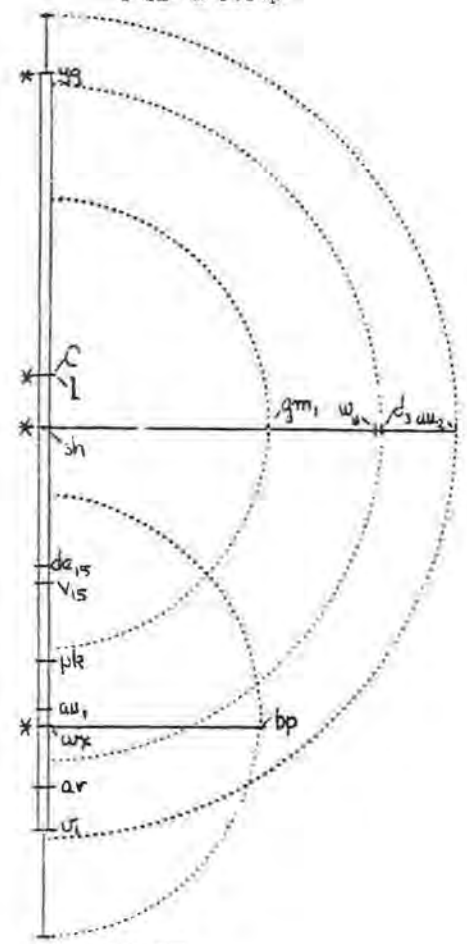
[2] A, C and R segregating - 27:37 ratio

[3] Ratio corrected for germination by author

[4] See Three-point test data

[5] Recombination value recalculated - author's calculation given as 39.7

C sh wx Group



R-G GROUP

List of Genes

df	Flint defective	Lindstrom 1925
g	Golden plant	Lindstrom 1918
gm_2	Germless	Demerec 1926
li_1	Linezte - striped leaves	Kempton 1920
l_1	Lutous seedlings	Lindstrom 1917
l_2	Lutous seedlings	Lindstrom 1925
nl	Narrow-leaf	Emerson (Unpub.)
pg_1	Pale-green seedling	Brunson 1924
R	Aleurone color	East and Hayes 191[illegible]
S	Spotted aleurone with Rrr	Kempton 1919
v_{12}	Virescent seedling	Phipps (Unpub.)
v_{20}	Virescent seedling	Phipps (Unpub.)
w_2	White seedling	Carver 1924

Linkage Data

Genes X	Genes Y	Link. phase	Number of individuals: X Y	X y	x Y	x y	Total	Recombinations: No.	%	Authority
R	G	C Bc	200	55	58	174	487	113	23.2	Lindstrom '17 & '18
		C Bc	227	36	33	195	491	69	14.1	Emerson
		R Bc	29	81	86	18	214	47	22.0	Lindstrom '18
		R Bc	18	117	156	28	319	46	14.4	Emerson
							1511	275	18.2	
R	L_1	C S	303	2	5	121	431		1.6[1]	Lindstrom '21
G	L_1	R Bc	8	35	21	5	69	13	18.8	Lindstrom '18
R	Pg_1	C S^2	1907	300	1053	686	3946		23.3	Brunson '24
		R S	1199	506	445	32	2182		27.2	Brunson '24
G	Pg_1	C S	628	59	57	146	890		14.6	Wentz
Li_1	Pg_1	R S	194	71			265		45	Brunson '24
R	w_2	C S	1329	171	202	402	2104		18.5	Carver '24
		C S	648	74	81	157	960		17.8	Lindstrom '24
		R S	43	16	22	2	83		30.8	Carver '24
w_2	L_1	R S	815		210	10	1035			Lindstrom '25
		R S^3	585		348	84	1017			Lindstrom '25
		R S^4	560		318	70	948			Lindstrom '25
		R S^5	380		402	115	897		22.0	Lindstrom '25
R	L_2	R S	986	405	473	69	1893		33.9	Lindstrom '24
		C S^2	837	197	582	277	1893		35.4	Lindstrom '24
R	Gm_2	R S	2239	784	976	84	4083		31	Demerec '26
		R S	6876	2947	1182	90	11095		27	Wentz
Pg_2	G	R S	2810	873			3683		50 ±	Wentz
Gm_2	Pg_1	R S	835	255			1090		50 ±	Wentz
R	V_{18}	C[6]	51	15	43	93	202		20	Phipps
R	V_{20}	C Bc[6]	77	10	80	152	319		12.5	Phipps
G	Li_1	R Bc	148	817	924	111	2000	259	13.0	Hutchison
R	Li_1	C Bc	208	74	86	138	506	160	31.6	Hutchison
		C Bc[6]	460	191	282	374	651[7]	191	29.3	Hutchison
							1157	351	30.3	
G	N1	R Bc	69	389	382	49	889	118	13.3	Emerson
P	N1	C Bc	219	93	116	191	619	209	33.8	Emerson

[1] 1918 data indicate complete linkage

[2] C and R segregating - 9:7 aleurone ratio

[3] W_1 and w_2 segregating

[4] W_2 and w_3 segregating

[5] W_1, w_2 and w_3 segregating

[6] C and R segregating

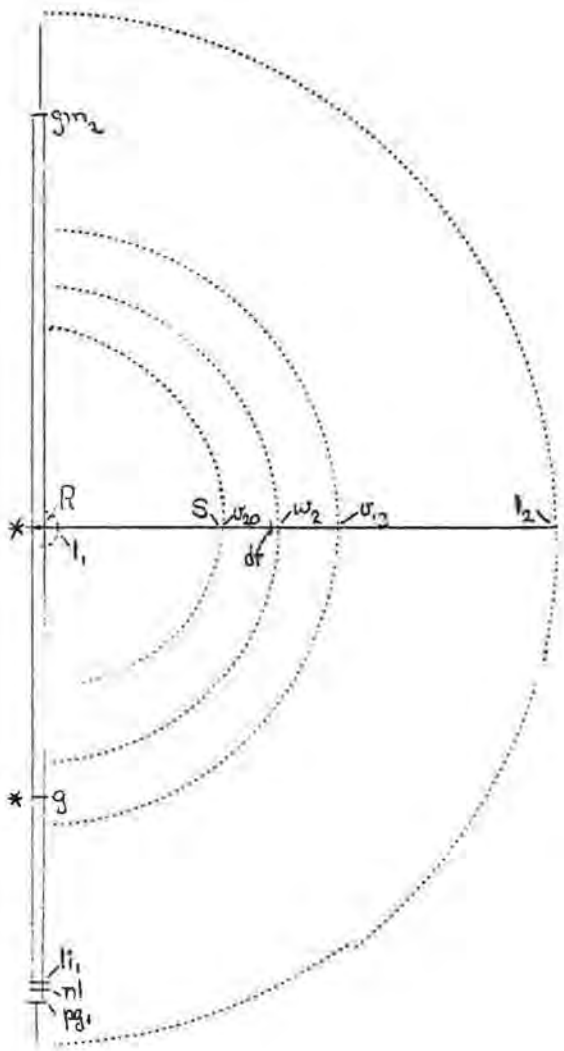
[7] First two classes only

Notes

df Lindstrom states that df and w_3 are very closely linked but presents no data.

S Hampton (1919) postulated this spotting factor, located so as to give about 12.5% recombinations with R. Emerson (Unpub.) has additional evidence in support of this assumption.

R g Group



List of Genes

de_1	Defective endosperm	Mangelsdorf/Jones 1925
de_6	Defective endosperm	Mangelsdorf 1926
de_{16}	Defective endosperm	Wentz 1925
Ga	Gamete - pollen tube growth	Mangelsdorf/Jones 1925
ge_1	Premature germination	Mangelsdorf 1926
su	Sugary endosperm	East and Hayes 1911
Ts_5	Tassel-seed	Emerson (Unpub.)
Tu	Tunicate ear	Collins 1917
wl	White-base leaf	Stroman 1925

Linkage Data

Genes X	Genes Y	Link. phase	Number of individuals: X Y	X y	x Y	x y	Total	Recombinations: No.	%	Authority
Su	Tu	C S	113	4	7	25	149	-	8.3	Jones & Gallastegui '21
		C Bc	430	175	169	406	1180	344	29.1	Eyster '21
		C Bc	612	290	208	562	1672	498	29.8	Emerson
		R Bc	1031	2498	2093	807	6429	1838	28.6	Eyster '22
		R Bc	63	215	164	57	499	120	24.0	Emerson
							9780	2800	28.6	
Su	Wl	R S	44	19	11	1	75		25.0	Stroman '24
		R S	4492	2018	1961	93	8564		22.0	Carver '27
De_{16}	Su	C S	20622	453	7201		28276	-	3.2	Wentz '25
Su	V_8	C S	940	214	179	148	1481	-	32.4	Somero '26
V_8	Tu	C S	450	1	Lethal		451		<1	Phipps
De_1	Su	R S	601	238	247	64	1150	-	39	Mangelsdorf & Jones '25
De_6	Su	R S	204	92			296		26	Mangelsdorf '26
Ge_1	Su	R S	1218	474			1692		40	Mangelsdorf '26
Su	Ts_5	C Bc	578	41	42	457	1118	83	7.4	Emerson
Ts_5	Tu	R Bc	49	166	115	48	378	97	25.7	Emerson

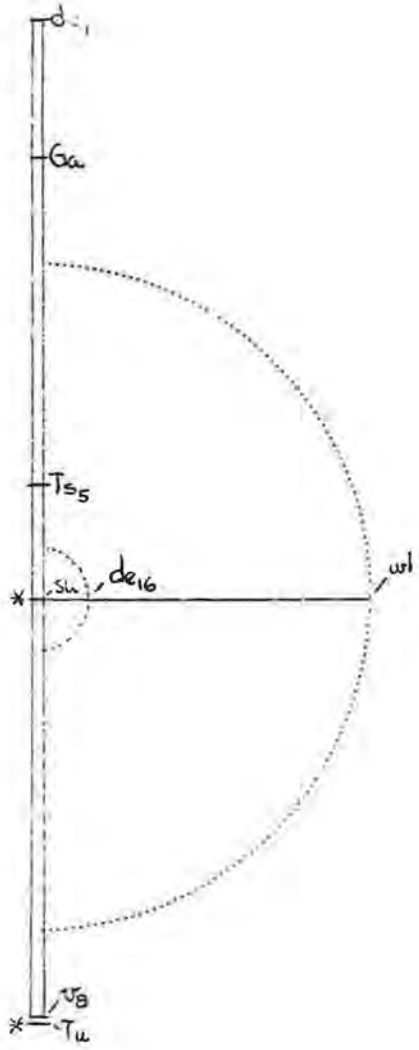
Notes

de_{16} is used instead of de_{su} for sugary defective of Wentz.

V_8 is very near Tu but whether to the left or right is unknown.

Ga is to the left of su because it disturbs the Tu-tu ratio very little if at all in pedigrees in which it disturbs the Su-su ratio materially (Emerson, Unpub.).

de_1 is presumably to the left of Ga, because Ga is between de and su (Mangelsdorf and Jones 1925).



B-LG GROUP

List of Genes

B	Intensifier of plant color	Emerson 1918
lg	Liguleless	Emerson 1918
sk	Silkless	Jones 1925
ts_1	Tassel-seed	Emerson 1920
v_4	Virescent seedling	Demerec 1924

Linkage Data

Genes X	Genes Y	Link. phase	Number of individuals X Y	X y	x Y	x y	Total	Recombinations No.	Recombinations %	Authority
B	lg	C Bc	240	134	102	243	719	236	32.8	Emerson '18
		C Bc	642	291	282	620	1835	573	31.2	Emerson
		C Bc	2487	1469	1557	2609	8122	3026	37.2	Emerson & Hutchison '[illegible]
		R Bc	498	1085	1037	504	3124	1002	32.1	Emerson
							13800	4837	35.0	
lg	ts_1	C Bc	117	52	72	74	315	124	39.4	Emerson
		R Bc	51	65	64	42	222	93	41.9	Emerson
							537	217	40.4	
B	v_4	C Bc	113	24	21	110	268	45	16.8	Demerec '24
v_4	lg	R Bc	412	501	521	366	1800	778	43.2	Demerec '24
B	Sk	C Bc	1332	97	106	1226	2761	203	7.4	Anderson
		R Bc	2	82	66	6	156	8	5.1	Anderson
							2917	211	7.2	
lg	Sk	R Bc	187	288	315	167	957	354	37.0	Anderson
			148	60	67	133	408	127	31.1	Anderson
							1365	481	35.2	

Y-Pl GROUP

List of Genes

Bh	Blotched aleurone with A c R	Emerson (Unpub.)
fi	Fine streaked leaves	Anderson 1922
Pl	Purple plant color	Emerson 1918
sm	Salmon silks	Anderson 1921
v_6	Virescent seedling	Carver 1927
v_7	Virescent seedling	Carver 1927
w_1	White seedling	Stroman 1924
w_5	White seedling with w_6	Demerec 1924
w_6	White seedling with w_5	Demerec 1924

Linkage Data

Genes Z	Genes Y	Link. phase	Number of individuals X Y	X y	x Y	x y	Total	Recombinations No.	Recombinations %	Authority
Y	Pl	C Bc	79	22	25	71	200	50	25.0	Emerson '18
			545	221	234	506	1506	455	30.2	Anderson '2[illegible]
			60	51	30	55	216	81	37.5	Anderson
			173	46	59	176	454	105	23.1	Hutchison
		R Bc	367	660	897	372	2516	739	29.4	Anderson 1921
			135	398	374	118	1025	253	24.7	Anderson
							5917	1683	28.5	
Pl	Sm	C Bc	1076	148	146	944	2361	291	12.3	Anderson '21
			84	1014	971	76	2145	160	7.5	Anderson '21
							4506	451	10.0	
[illegible]	Fi	[illegible] Bc	353	0		many				Anderson '22
Y	w_6	C S	250	37	35	54	376		24.3	Demerec '24
Y	w_5 / w_6	S'	349	12	60	33	454		24.3 / 24.5	Demerec '24
Y	w_1	C S	1020	257	259	191	1707		35	Lindstrom '24
		C S	1132	321	347	175	1975		42	Stroman '24
		R S	456	181	186	41	864		42	Stroman '24
Y	v_6	R S	467	225	209	12	913		28	Carver '27
Y	v_7	C S	592	149	178	79	998		42	Carver '27
		C S^2	445	277	106	116	944		36	Carver '27
v_6	v_7	R S	497	179	237		913		43	Carver '27
Bh	Y	C Bc	144	61	118	210[3]	523	159	32.3	Anderson
Bh	Pl	C Bc	86	1	26	47[3]	132		1.7[4]	Anderson

[1] w_5 and w_6 duplicate genes

[2] Segregating for another v - not linked

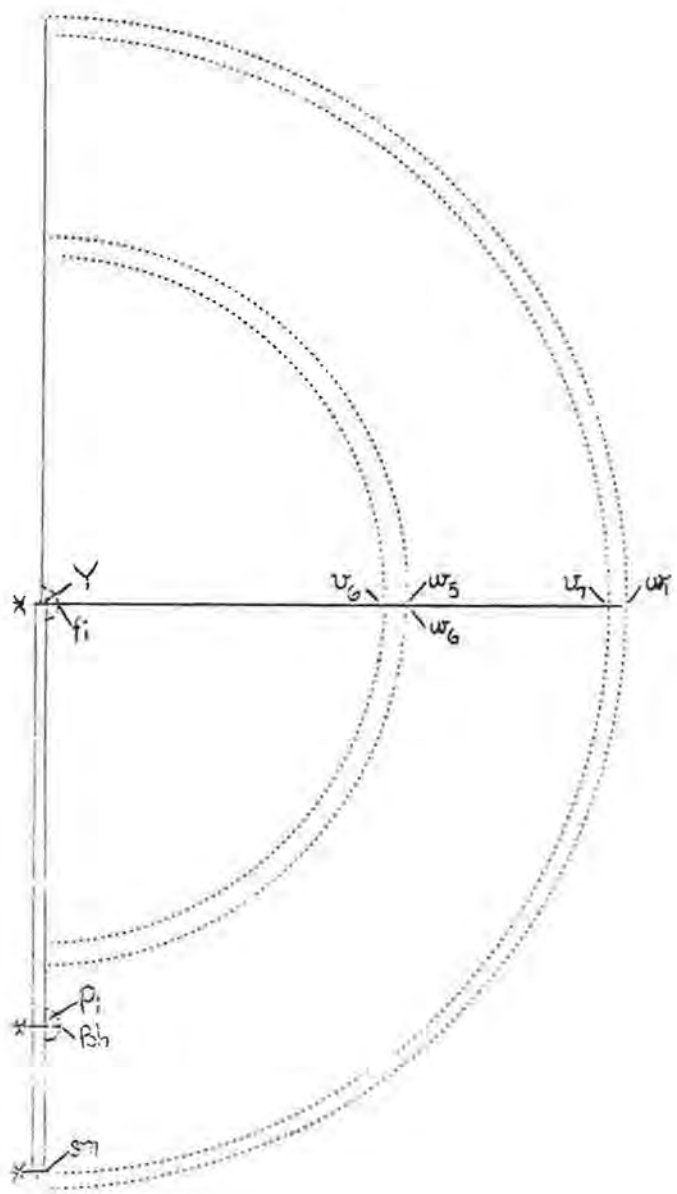
[3] Probably part of this class actually Bh

[4] From Bh class

Notes

m_1, m_2: Stroman presents data which he interprets as showing linkage between m_1 and m_2 and also between m_1 and Y. His data are sufficiently extensive only to suggest that these factors may belong to this linkage group.

Y Pl Group



RA-GL_1 GROUP

List of Genes

Bn	Brown chlorone	Kvakan 1924
gl_1	Glossy seedling	Kvakan 1924
in	Intensifier of chlorone	Fraser 1924
pg_1	Pale-green seedling	Demerec 1925
ra	Ramosa	Gernert 1912
sl	Slashed seedling	Hayes and Brewbaker 1928
sr_2	Stricte - striped leaf	Brunson (Unpub.)

Linkage Data

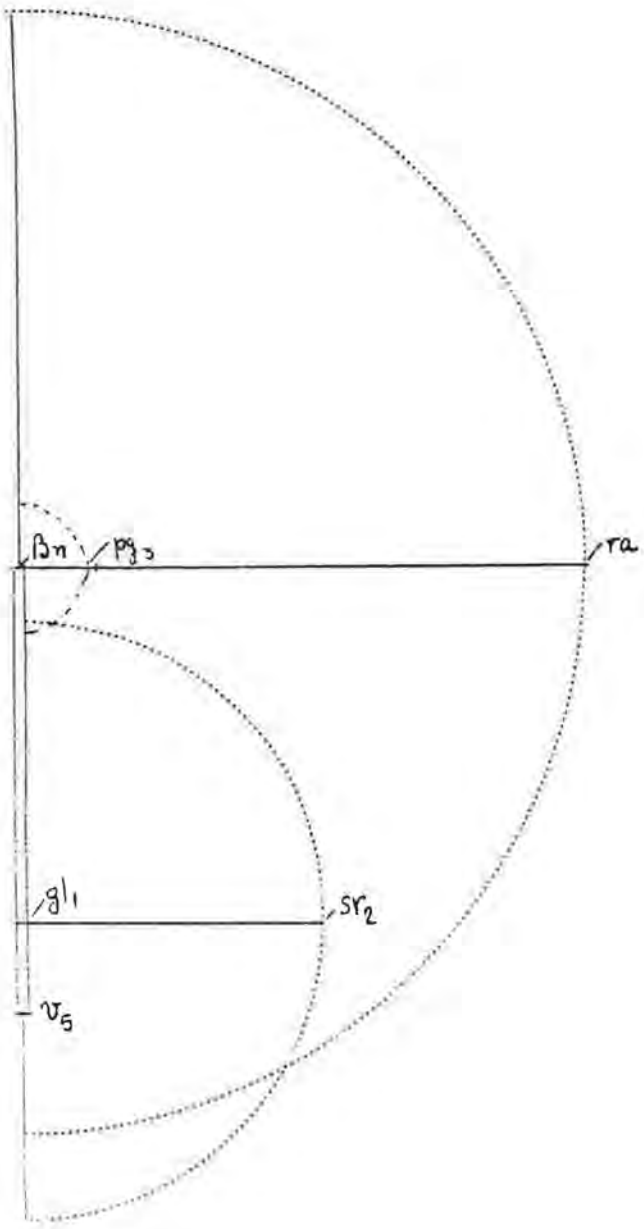
Genes X	Y	Link. phase	Number of individuals X Y	X y	x Y	x y	Total	Recombinations No.	%	Authority
Bn	Gl_1	C Bc	277	63	54	192	486	117	24.1	Kvakan '24
Gl_1	V_5	C Bc	106	9	6	120	241	15	6.2	Kvakan "
Bn	V_5	C Bc	83	31	29	98	241	60	24.9	Kvakan '24
Bn	Ra	C Bc	169	104	100	161	534	204	38.2	Kvakan '24
Bn	Pg_1	C S	203	8	5	65	281		4.5	Demerec '25
Gl_1	Sr_2	R Bc	97	289	342	63	791	160	20.2	Brunson

Notes

sl Hayes and Brewbaker state that sl belongs to this linkage group.

Y_2, Yp } Hayes and Brewbaker present data showing a linkage between two factors for yellow endosperm (Y_2 and Yp) and a glossy seedling factor. Since the relation of the glossy character to gl_1 is not evident, the placing of these two genes in this linkage group would appear uncertain.

ra-gl_1 Group



PR-V_2 GROUP

List of Genes

bm	Brown midrib	Eyster 1926
bv	Brevis - semi-dwarf plant	Suttle (Unpub.)
f_2	Fine striped leaves	Eyster 1926
Pr	Purple aleurone	East and Hayes 1911
sc_1	Scarred endosperm	Eyster 1926
tn	Tiny plant	Eyster 1926
v_2	Virescent seedling	Demerec 1924
v_3	Virescent seedling	Demerec 1924
v_{12}	Virescent seedling	Phipps (Unpub.)
yg	Yellow green	Eyster 1926
ys	Yellow-stripe	Beadle 1929

Linkage Data

Genes X	Genes Y	Link. phase	Number of individuals X Y	X y	x Y	x y	Total	Recombinations No.	Recombinations %	Authority
Pr	V_2	R Bc	377	532	499	366	1774	743	41.9	Phipps
		C Bc	67	46	41	51	205	87	42.4	
							1979	830	42.0	
Pr	V_3	R Bc	123	296	320	102	841	225	26.8	Phipps
Pr	V_{12}	C Bc	61	15	4	75	155	19	12.4	Phipps
		C S	492	37	39	137	705		11.4	
Pr	Ys	R	219	323	209	19	770		8.3	Beadle '29

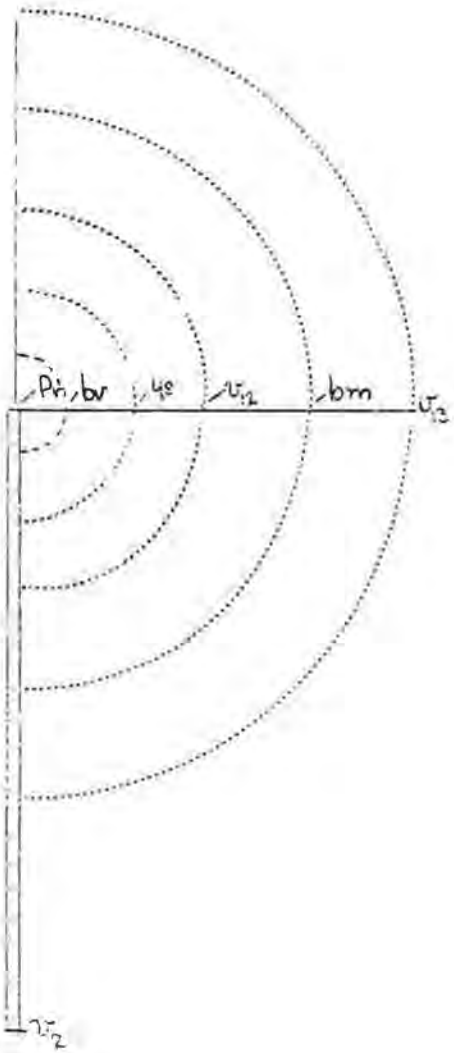
Notes

bm — Eyster states that bm shows about 20 per cent recombinations with Pr but presents no data.

f_2, sc_1, tn, yg — Eyster states that these genes belong to the Pr linkage group but presents no data.

bv — Li (Unpub.) has evidence that bv and Pr are relatively closely linked.

Pr v_2 Group



D_1 - PG_2 GROUP

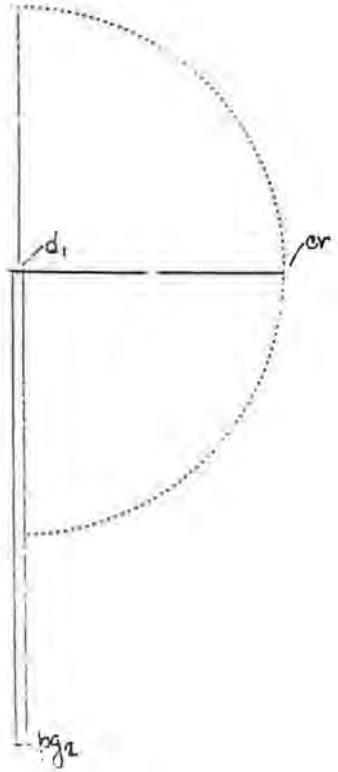
List of Genes

d_1	Dwarf plant	Emerson 1912
pg_2	Pale-green seedling	Demerec 1924
cr	Crinkly leaves	Emerson 1921

Linkage Data

Genes X	Genes Y	Link. phase	Number of individuals: X Y	X y	x Y	x y	Total	Recombinations: No.	%	Authority
D_1	Pg_2	R S	1364	584	580	65	2593		32	Demerec '24
D_1	Cr	R Bc	15	53	48	15	131	30	22.9	Emerson
		C Bc	518	102	107	482	1209	209	17.3	Emerson
							1340	239	17.8	

d pg_2 Group



A-Ts_4 GROUP

List of Genes

A	Anthocyanin pigment	Emerson 1918
na	Nana - dwarf plant	Li (Unpub.)
ts_4	Tassel-seed	Phipps 1926

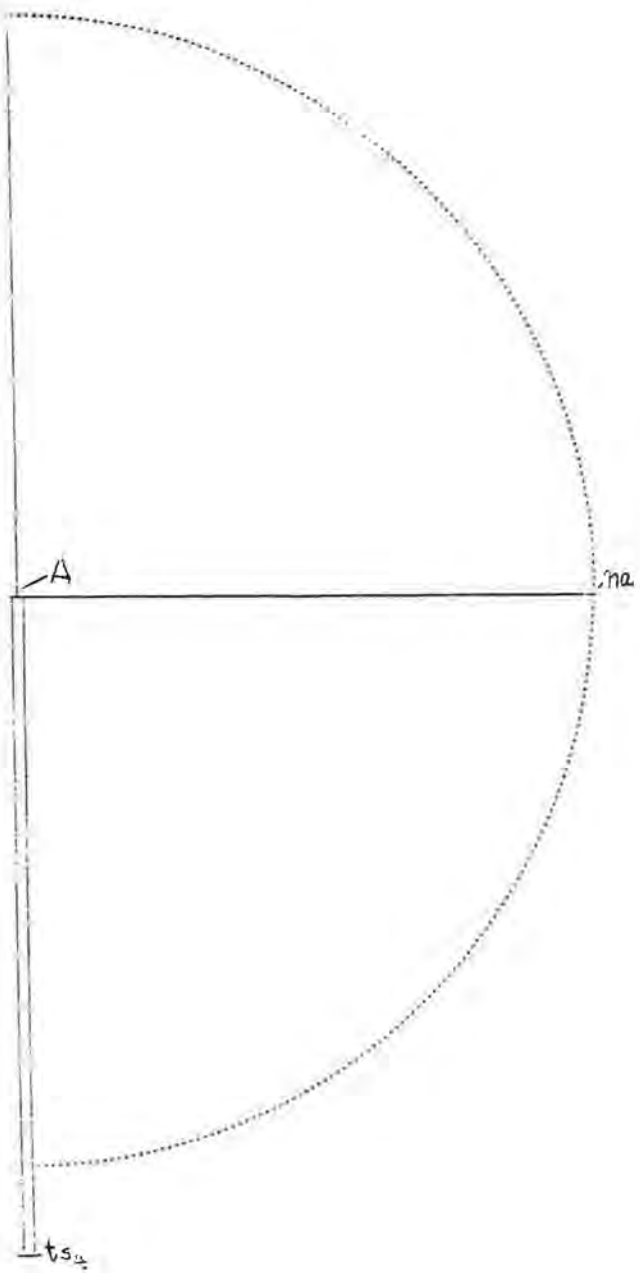
Linkage Data

Genes X	Genes Y	Link. phase	Number of individuals: X Y	X y	x Y	x y	Total	Recombinations: No.	%	Authority
A	Ts_4	C Bc	90	63	70	85	308	133	43.2	Phipps '28
		R Bc	262	351	372	333	1318	595	45.1	Phipps '28
							1626	728	44.8	

Notes

Li (Unpub.) has evidence that na is linked with A, showing about 40 per cent of recombinations. Jones (Unpub.) also has evidence of this linkage.

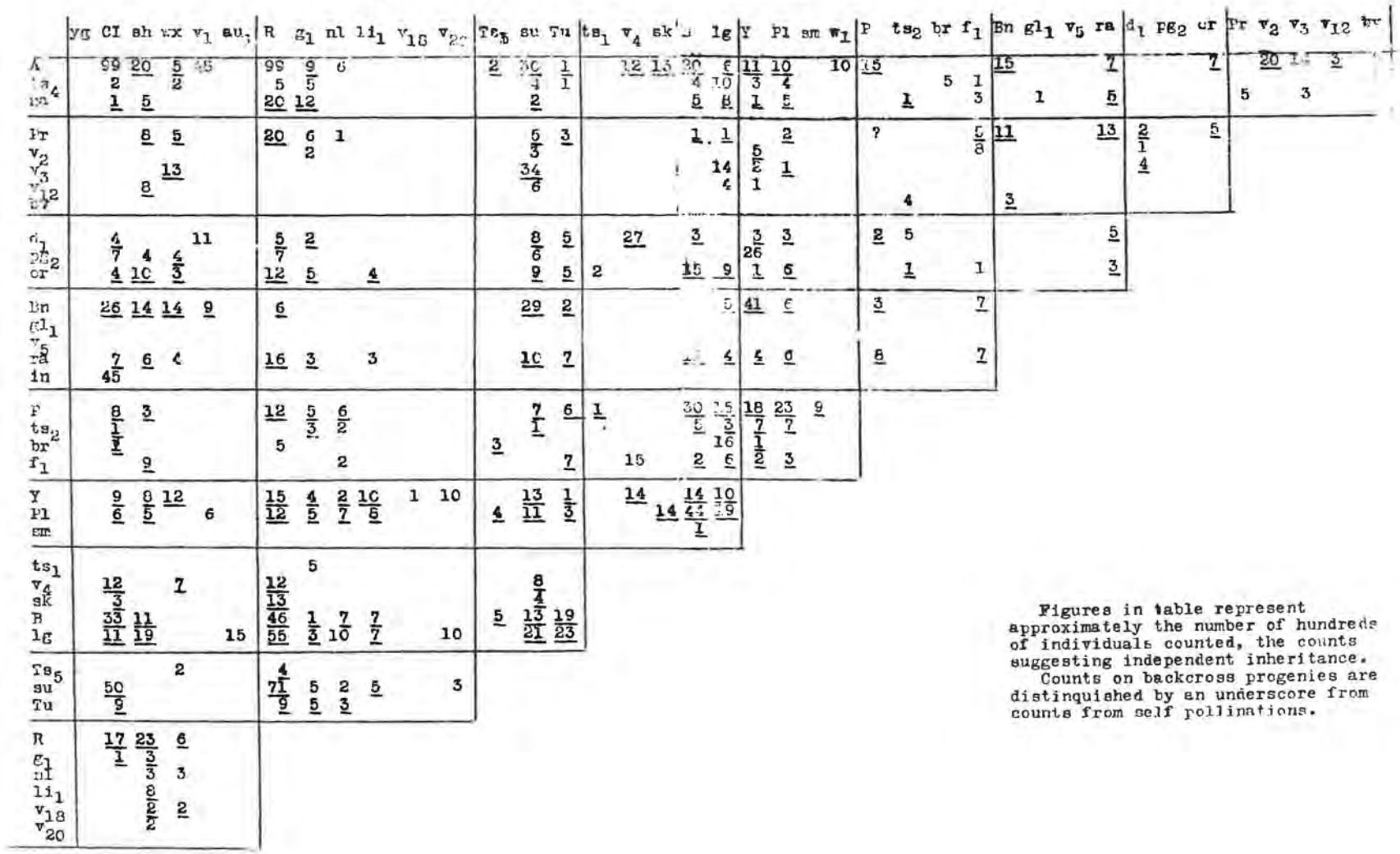
A ts_4 Group



SUMMARY OF THREE-POINT LINKAGE TESTS IN MAIZE

Parent No. 1	Parental combinations No.1 No.2	Recombinations Region 1	Region 2	Regions 1 & 2	Total	Coincidence	Authority
C sh Wx	2538 2708 5246	116 113 229 3.4%	601 626 1227 18.3%	4 2 6 0.12%	6708	0.14	Hutchir '22
C Sh wx	2215 2280 4495	121 139 260 4.3%	669 653 1322 21.7%	2 3 5 0.08%	6082	0.09	Hutchison '22
yg C Sh	54 51 105	7 9 16 12.7%	3 1 4 3.2%	1 0 1 0.8%	126		Jenkins '27
C Sh ar	4678 4138 8816	259 192 451 3.6%	1243 1986 3234 25.8%	14 28 42 0.34%	12543	0.33	Eyster '2?
Ts_5 Su tu	163 113 276	9 12 21 5.6%	37 39 76 20.1%	2 3 5 1.3%	378	0.88	Emerson
Ts_1 b Lg	111 71	24 17	48 35	6 3	315		Emerson
Ts_1 B lg	57 57 296	20 21 82 15.3%	31 21 135 25.1%	7 8 24 4.5%	222 537	0.77	
Sk B Lg	148 131 279	13 8 21 5.1%	56 52 108 26.3%	0 2 2 0.5%	410	0.36	Anderson
Y Pl Sm Y pl Sm Y Pl sm Y pl sm	191 180 436 377 305 265 333 411 2498	109 104 165 206 107 124 183 152 1150 28.6%	21 31 45 50 28 30 66 59 330 8.2%	5 5 5 1 0 1 16 12 45 1.12%	4023	0.40	Anderson '21
Ts_2 tr F_1	12 8 20	3 9 12 34.3%	0 3 3 8.6%	0 0 0 0.0%	35		Emerson
Sn Cl_1 V_5	83 98 181	22 23 45 18.7%	9 6 15 6.2%	0 0 0 0.0%	241		Kvalnm '24

Summary of Data on the Independence of the Linkage Groups in Maize



Figures in table represent approximately the number of hundreds of individuals counted, the counts suggesting independent inheritance.

Counts on backcross progenies are distinquished by an underscore from counts from self pollinations.

LITERATURE ON LINKAGE IN MAIZE

In general, only papers containing data on the linkage or independence of factors in known linkage groups are listed.

Anderson, E. G. - The inheritance of salmon silk color in maize. Cornell Univ. Agric. Exp. Sta. Mem. 48: 539-554. 1921.

_____ - Heritable characters of maize. XI. Fine streaked leaves. Jour. Heredity 13: 91-92. 1922.

_____ - Pericarp studies in maize. II. The allelomorphism of a series of factors for pericarp color. Genetics 9: 442-453. 1924.

_____ - Genetic factors for yellow endosperm color in maize. Papers Mich. Acad. Science, Arts & Letters 4: 51-54. 1924.

_____ and Emerson, R. A. - Pericarp studies in maize. I. The inheritance of pericarp colors. Genetics 8: 466-476. 1923.

Beadle, G. W. - Yellow stripe - a factor for chlorophyll deficiency in maize located in the Pr pr chromosome. Amer. Nat. 63: 189-191. 1929.

Bregger, T. - Linkage in maize: The C aleurone factor and waxy endosperm. Amer. Nat. 52: 57-61. 1918.

Brink, R. A. - A lethal mutation in maize affecting the seed. Amer. Nat. 61: 525-530. 1927.

Brunson, A. M. - The inheritance of a lethal pale green seedling character in maize. Cornell Univ. Agric. Exp. Sta. Mem. 72: 5-22. 1924.

Carver, W. A. - The genetic relation of endosperm and chlorophyll characters in maize. Proc. Iowa Acad. Sci. 31: 129. 1924. (A short note without data in which it is stated that brindled, a chlorophyll defect, shows linkage with sw with about 26 % of recombinations).

_____ - A genetic study of certain chlorophyll deficiencies in maize. Genetics 12: 415-440. 1927.

Collins, G. N. - Gametic coupling as a cause of correlations. Amer. Nat. 46: 589-590. 1912.

_____ - Mosaic coherence of characters in seeds of maize. U.S.D.A. B.P.I. Circ. 132: 19-21. 1913.

_____ - Hybrids of Zea tunicata and Zea ramosa. Proc. Nat. Acad. Sci. 3: 345-349. 1917.

_____ - Hybrids of Zea ramosa and Zea tunicata. Jour. Agric. Res. 9: 383-395. 1917.

_____ and Kempton, J. H. - Inheritance of waxy endosperm in hybrids of Chinese maize. IV. Conference internat de genetique (Paris). Compt. rend. 1911: 347-356. 1913.

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V. REPORT OF MAIZE GENETICS COOPERATION STOCK CENTER

During 1978, the Maize Genetics Cooperation received 128 requests for maize genetic stocks. The number of requests decreased by 11% in 1978 compared to 1977.

The following tables illustrate the nature of the seed requests and how seed packets used to fill the requests were distributed:

Table 1. Seed requests received by the Maize Stock Center-1978.

Category	Domestic	%	Foreign	%	Total	%
Genetics	61	47.6	19	14.8	80	62.4
Plant Physiology	17	13.3	2	1.6	19	14.9
Plant Breeding	8	6.3	6	4.7	14	11.0
Education	10	7.8	5	3.9	15	11.7
Total	96		32		128	

Table 2. Distribution of seed packets for 1978 seed requests.

Category	Domestic	%	Foreign	%	Total	%
Genetics	821	63.8	198	15.4	1019	79.2
Plant Physiology	96	7.5	18	1.4	114	8.9
Plant Breeding	30	2.3	28	2.2	58	4.5
Education	47	3.7	48	3.7	95	7.4
Total	994		292		1286	

The tables illustrate that the bulk of the seed requests and packets sent to fill requests are for domestic use (75% vs. 25% for requests and 77.3% vs. 22.7% for packets). In addition, the major requests and amount of seed sent are for genetic purposes.

Requests for stocks and correspondence relative to the stock center should be addressed to:

Dr. R. J. Lambert
S-118 Turner Hall
Department of Agronomy
University of Illinois
Urbana, Illinois 61801

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 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
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
Adh-1-S
vp6
gs bm2
Ts6
bm2
id
nec2
ms9
ms12
ms14
mi
D8
Lls
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
al lg gl2 b sk fl v4
lg
lg gl2 B
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
Ch
LES
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 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-xl
a Ga7 ; A2 C R
sh2

Chromosome 3 (continued)

vp
Rp3
pg14
a3
g5
ye!*-5787
TB-3La (3L.10)
TB-3Sb (3S.50)
Primary Trisomic 3

Chromosome 4

Rp4
Ga
Ga su
Ga-S
st
st Ts5
st fl2
Ts5
Ts5 fl2
Ts5 su
Ts5 su zb6
Ts5 su zb6 o
Ts5 Tu
Ia
Ia su Tu gl3
Ia su gl3
Ia su gl3 c2 ; A A2 C R
Ia 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 j2
su gl4 o
su gl4 o Tu
su j2
su gl3
su gl3 o
su o
bt2
bm3
gl4
gl4 o
Tu
Tu-1 1st
Tu-1 2nd
Tu-d
Tu-md
Tu gl3
j2
j2 c2 ; A A2 C R
j2 C2 ; A A2 C R
j2 gl3
v8
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
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
vp2 pr
vp2 gl8
vp7
bm yg
bt
ms5
v3
td ae
ae
sh4
gl8
na2
lw2
ys
eg
v2
yg
ms13
v12
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
y Pl Bh ; c sh wx A A2 R
y su2
Y l10
Y l12
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
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
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 wx v
sh wx d3
sh wx pg12 gl15 ; y pg11
lo2
wx*
wx-a
w11
wx d3
wx d3 w11
wx d3 v gl15
wx d3 gl15
Wx pg12 ; y pg11
wx pg12 ; y pg11
Wx pg12 ; Y pg11
wx pg12 ; Y pg11
wx pg12 bm4 ; y pg11
wx v
wx bk2
wx bk2 bm4
wx Bf
wx Bf bm4
d3
v
gl15
gl15 Bf

* Additional waxy alleles available from collection of O. E. Nelson.

Chromosome 9 (continued)

gl15 bm4
bk2 Wc
Wc
bm4
bm4 Bf
16
17
yel*-034-16
w*-4389
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
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 1 ; 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 1 ; 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

Chromosome 10 (continued)

R-r (Boone) ; A A2 C
R-sk ; 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 1
o7
o7 ; o2
1
v18
Mst
1 yel*-5344
yel*-8721
yel*-8454
yel*-8793
TB-10La (10L.35)
TB-10Sc
Primary Trisomic 10

Unplaced Genes

dy
dy
el
h
14
LES2
Rs
v13
ws ws2
ub
zb
zb2
zb3
zn2
1*-4923
nec*-8376

Multiple Gene Stocks

A A2 C R-g Pr B P1
A A2 C R-g Pr B pl
A A2 C r-g Pr B P1
A A2 C r-g Pr B pl
A A2 c R-g Pr B pl
A A2 C R-r Pr B P1
A A2 C R-r Pr B pl
A A2 C R-r Pr b P1
A A2 c R-r Pr B P1
A A2 C r-r Pr B P1
A A2 c r-r Pr B P1
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
A A2 c R Pr y wx
A A2 C r Pr y wx
bz2 a c2 a2 y c r
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
bm bm2
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 P1
a A2 C R Dt
su
pr ; A A2 C R
y
gl
ij
Y sh wx
sh bz wx
wx
g A A2 C R
A A2 C R B P1

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 (7cent.;9cent.)
wx8-9d (8L.09;9L.16)
wx8-9-6673 (8L.35;9S.31)
wx9-10b (9S.13;10S.40)

Inversions

Inv.1a (1S.30-L.50)
Inv.1c (1S.35-L.01)
Inv.1d (1L.55-L.92)
Inv.1L-5131-10 (1L.46-L.82)
Inv.2a (2S.70-L.80)
Inv.2-3713 (2S.93-L.65)
Inv.2-3778 (2S.44-L.84)
Inv.2S-L8865 (2S.06-L.05)
Inv.2L-5392-4 (2L.13-L.51)
Inv.3a (3L.38-L.95)
Inv.3L (3L.19-L.72)
Inv.3L-3716 (3L.09-L.81)
Inv.4b (4L.40-L.96)
Inv.4c (4S.86-L.62)
Inv.4e (4L.16-L.81)
Inv.5-8623 (5S.67-L.69)
Inv.6-8452 (6S.77-L.33)
Inv.6-8604 (6S.85-L.32)
Inv.6-3712 (6S.76-L.63)
Inv.7b (6S.32-L.30)
Inv.7L-5303 (7L.17-L.61)
Inv.7-8540 (7L.12-L.92)
Inv.7-3717 (7S.32-L.30)
Inv.8a (8S.38-S.15)
Inv.9a (9S.70-L.90)
Inv.9b (9S.05-L.87)
Inv.9c (9S.10-L.67)

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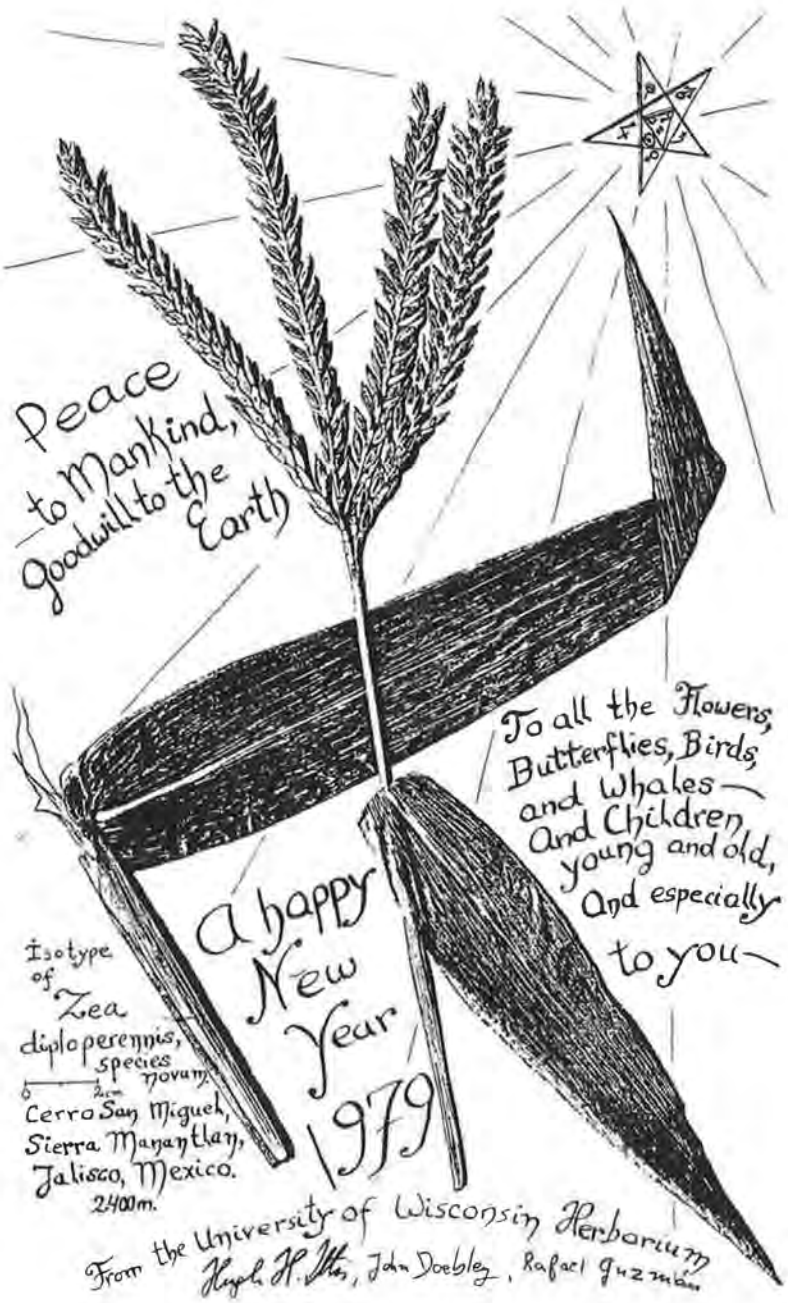

Dear Colleague:

[ ] The seed samples you requested are enclosed; I am pleased to be rid of this material and wish you luck with it.

[ ] The seed samples you requested are enclosed; I would rather not know what you intend to do with them.

[ ] I will be glad to look over your manuscript when the research is written up.

[ ] The seed samples you requested are enclosed; they are routine materials for which a simple acknowledgement would be appropriate in your Materials section.

[ ] The seed samples you requested are enclosed; they are materials for which a lavish acknowledgement would be appropriate.

[ ] The seed samples you requested are enclosed; they contain drops of both my perspiration and my blood, but you have invented a clever innovation that had not occurred to me and I humbly ask that you handle them tenderly.

[ ] The seed samples you requested are enclosed; if I understand correctly the study you plan to do with them, we both have had the same idea and now the materials are ready....can we get together and make a better study?

[ ] Phone me; urgent we join forces.

[ ] Where is your variant located on the map?

[ ] Take my breakpoints on faith; I am infallible.

[ ] How many bushels did you need?

[ ] Did you want these to grow in your cornfield?

[ ] Do you believe in collaboration between geneticists at different locations?

[ ] Do you believe in collaboration?

[ ] I have received your request and think you need my advice; the price of my advice is co-authorship.

[ ] In two more seasons I will have the materials you requested converged to uniform backgrounds and in sib comparisons so that we can both do the experiment.

[ ] The materials you requested will be available after I make the crosses, self the F1, select and test in F2, and identify the proper combination.

[ ] The materials you requested were an incidental byproduct of studies with greater purposes than those you have outlined.

[ ] Please increase and return with 10% interest.

[ ] I am sending you my complete collection of ( ) strains; future requests for these materials will be forwarded to you.

[ ] I have received your request; what do you plan to do with the whole collection?

[ ] I have not received your request.

Zealously,

Chrome O'Some

ERRATA (For previous Errata and Corrigenda see 50:180, 51:123, 52:178)

No.	Corrected Line
44A (44 Appendix, Author and Name Index to Nos. 3 through 43)	
5	(Bauman) 28:52,88,91 29:51,66
12	Cutter, V. 29:17
13	(Dempsey) 34:1,65,66,87 35:1,63,65,66
24	Kelley, D. 41:47
32	(Morris, R.) 24:14,60 25:41,52,60
48	(Wallace, H. A.) 25:58 31:172 32:154 33:2
50:158	(Banerjee) 48:219 49:19,51,174
50:173	(Reddy, A. R.) 47:165 48:138,139,233 50:65,67,132
50:173	(Rhoades) 46:1,2,51,53,58 47:1,59,62,65,67,
51:49	suggest that....T4-6(003-16)...
52:132	(Chrom. 6, T2-6d) Ref. 1 (not 17)
52:150	Alcohol dehydrogenase Adh1-S; Adh2-P Adh1-FunkFγ25; Adh2-N
52:175	(Adh) -FunkF 8 -FunkFγ25 150 -S 6,9,27,30,150
52:175	Adh2 9,94 -N 150 -P 150
52:175	add Df9S-33166 20
52:175	ig 11,150 ij 36,82,83,133
52:175	T8-9(043-6) 134
52:176	Ayers, J. E. 101,160

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