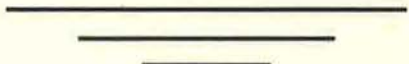


McMullen.

MAIZE GENETICS COOPERATION

NEWS LETTER

57



March 31, 1983

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

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

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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, Wisconsin, 1968.

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

Maize. E. Häfliger, ed., CIBA-GEIGY Monograph, Basle, Switzerland, 1979.

Maize Research and Breeders Manual No. IX. C. B. Henderson, Illinois Foundation Seeds, Inc., Box 722, Champaign, Illinois 61820, 1980.

Quantitative Genetics in Maize Breeding. A. Hallauer and J. B. Miranda, Iowa State Univ. Press, Ames, 1981.

Maize for Biological Research, W. F. Sheridan, ed., Plant Molec. Biol. Assoc., Box 5126, Charlottesville, VA 22905, 1982.

Linkage map of corn (maize) (*Zea mays* L.). Pp. 377-393 in Genetic Maps, vol. 2, S. J. O'Brien, ed., National Cancer Institute, Frederick, MD, 1982.

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I. FOREWORD

To Maize Geneticists :-

If you have any good tester combinations you wish to send in so that they may be made available for the whole group or if there is any combination of genes you would like to have, will you please notify us here at Cornell so that we may list your contributions and wants in the corn-letter which will come out in the near future. January 1st has been set as the dead line for receipt of material to be included in the letter. Will you please cooperate with us so that we can make this cooperative affair a real service to all concerned.

--M. M. Rhoades, December 12, 1932

Over 950 copies of this 1983 issue, MNL 57, will be sent to cooperators and colleagues around the world. Funds for preparation, reproduction and mailing are provided by the U. S. Department of Agriculture through a grant to the Maize Genetics Stock Center at the University of Illinois. Office support from the U. S. D. A. covers year-round needs, in facilities provided by the University of Missouri. The support and encouragement from these sources is indispensable, and we are all grateful for it.

I appreciate the valuable help of Ming-Tang Chang, Christine Curtis, Rodney Higgins and Bryan Kindiger with proofing of copy. Robert Bird, David Hoisington and Scott Poethig also helped with proofing and with planning of parts. Thanks especially to Stephen Modena for contributing essential ideas and aid in the enhancement of Zealand (as also to Larry Darrah), for developing our new, comprehensive indexing routines for symbols and names, and for help with proofing. Kathryn Chappell and Christopher Browne helped with library, mockup and other vital tasks. Shirley Kowalewski ardently handled the office load through the year, including especially the compilation and typing of the list of Recent Maize Publications, and applied exuberant skill to editing of the copy before typing; I accept responsibility, however, for whatever flaws may be present. The final copy, carefully and tastefully typed as always, was composed, refined and made accurate and attractive by Mary Nelson.

Some new publications of interest:

- Coe, E. H., D. A. Hoisington and M. G. Neuffer, 1982. Linkage map of corn (maize) (*Zea mays* L.). Pp. 377-393 in Genetic Maps, vol. 2, S. J. O'Brien, ed., National Cancer Institute, Frederick, Maryland.
- Sheridan, W. F., ed., 1982. Maize for Biological Research. Plant Molecular Biology Association, Charlottesville, Virginia.

Back issues of News Letter No. 30 (1956) to date will be sent upon request; a microfilm of volumes 1-29 and 33 is available for \$9.50 U.S.; orders should be sent to Coe at the University of Missouri and checks should be made out to Maize Genetics.

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

The deadline for the next issue (number 58, 1984) is January 1, 1984. Reports submitted normally should consist of information bearing on genetic understanding or genetic manipulation of maize. Brief items containing specific data, specific observations, and specific methods are of most value. Communications are received and assembled with minimum editing.

Please double space the text; text copy will be retyped.

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E. H. Coe, Geneticist, USDA-ARS, and
Professor of Agronomy, Curtis Hall, Univ. of
Missouri, Columbia, Missouri 65211

II. REPORTS FROM COOPERATORS

AMES, IOWA
Iowa State University

Newly originated mutable alleles of the c2 locus

Because of the establishment, by Dooner (Genetics 94:29, 1980), of the relation of c2 to chalcone synthase, impetus was given to rescue En-related c2-mutable alleles. Five c2-mutable alleles have been found and confirmed. They are as follows, with patterns according to time and frequency of c2 to C2 mutation events (late displays a fine, and early a coarse pattern):

- c2-m826019 -- a very fine type, similar to a2-m41629 (Peterson, Genetics, 1976);
- c2-m826021 -- very fine type, similar to a2-m41629;
- c2-m826040 -- fine, but low-rate type;
- c2-m826134 -- early sectors at a very low rate (coarse);
- c2-m826204 -- early sectors at a high rate.

In addition, a number of stable c2 alleles (826116, 826126, 826133, 816149, 816154, 816155) were uncovered. These mutants, both the c2-mutables and c2 stables, are currently being identified as to system relationship (En, Ac, Dt, Uq, Fcu and Bg). Though there is a strong possibility that these (or some of them) are En-related as in past searches (Peterson 1978), there are instances that, although the search was in an En-related plot, other systems may be involved.

The five c2-mutable alleles appeared at the rate of approximately one in 2.5×10^{-5} .

Peter A. Peterson

Newly originated mutable alleles at the bz locus

Again, with the relationship of bz to UDP-glucose 3-O-glucosyltransferase (Larson and Coe, 1968, Proc. XII Int. Congr. Genet. 1:131; Dooner and Nelson, 1977, Biochem. Genet. 15:509) a search was made for unstable bz alleles. bz-m805137 has previously been uncovered and is currently being genetically analyzed.

Two additional bz-m alleles have been found. These are: bz-m826301 (fine, very high rate and seemingly autonomous) and bz-m826302 (fine, high frequency).

In addition, four stable bz alleles have been confirmed: 826357, 826361, 826440.

The frequency of recovery of bz-m is considerably less than the case with c2. The rate here is approximately one in 6×10^{-5} . System relationship tests are currently underway.

Peter A. Peterson

New sh alleles

sh-826466 is the only sh uncovered in a search of 1.2×10^6 kernels. This rate is considerably below the rate in the 1975 c sh wx plot which yielded 18 new sh isolates at the rate of 1 in 6.8×10^{-4} . Of course, this differential can be assignable to observer and sh vs. dent complication among the assorted experimental deviations.

Peter A. Peterson

Latent Uq activity

Activation of latent Uq elements has been detected in certain outcrosses. These Uq elements appear and are expressed initially at a low frequency, but by strong selection, very active elements can be isolated and these behave similarly to Uq-standard (Friedemann and Peterson, 1980, MCNL).

Peter D. Friedemann and Peter A. Peterson

Chromosome 9S losses

In an effort to develop a C-I C-I En En stock, it was necessary to testcross putative C-I C-I plants to C C in order to verify this genotype. Though C-I to C changes are often observed by anyone working with the C-I allele, one particular plant (793450-5) showed an excess frequency of colored spots when tested on C C. Subsequent tests of the C-I allele (C-I Sh Bz Wx) with C sh bz wx as a tester show that the loss can include C-I alone or C-I and Bz. Each time Bz is lost, the tester wx phenotype is simultaneously uncovered. These appear as single loss rather than BBF type events.

Peter A. Peterson

En-61138-3, a case of simultaneous loss of En and Sh2

From an original a-m (dense) (dense-very heavy a-m to A1, Peterson, 1961), an exception arose showing a fine pattern that, in addition, displayed a loss of mutability. Both the parental dense and the exceptional fine derivative are autonomously heritable, i.e., En is located at the a locus. In an attempt to determine the extent of the mutability loss, plants containing this allele were crossed as males onto the receptive a-m-1 allele that is linked to sh.

The cross: a-m-1 sh2/a-m-1 Sh2 female x a-m En Sh2/a-m-1 Sh2 male.

If the loss of mutability is beyond the range of the a locus and included the Sh2 gene, colored shrunken sectors would appear (with En, the heterozygote a-m1 sh2/a-m En Sh2 is colorless-round with fine spots). From this cross, kernels with colored shrunken sectors were found among the progeny. The results of several crosses are presented in Table 1.

The frequency and size of colored shrunken sectors on fine-round kernels varied among the progeny. In order to evaluate the heritability of this frequency of colored shrunken sectors, the size of the colored shrunken sectors was measured with a scaled grid in the eyepiece at 10x magnification. Sector size was classified as small (less than or equal to .5 sq mm) or big (more than .5 sq mm).

- (1) All the round kernels were spotted, indicating that the En is located at the a-m locus (all of the shrunken kernels were colored).
- (2) All the colored shrunken sectors were found on the spotted-round kernels, indicating that the loss phenomenon is also autonomously controlled at the a locus.
- (3) The size and frequency of sectors varied on different kernels.
- (4) The percentage of the mutable kernels on individual ears varied among ears.

It is evident from these observations that the colored shrunken sector is due to loss of the a-m En Sh2 segment from chromosome 3. Tests of this hypothesis are in progress.

With reference to the heritability of kernel selection (presence or absence of colored shrunken sectors), a-m-1 sh2/a-m-1 sh2 tester plants were crossed by plants from the selected kernels. The results indicated that there was a significant

Table 1. Phenotypic frequencies from cross of $a_1m-1 sh2/a_1m-1 sh2$ or $a_1m Sh2/a_1m-l sh2$ by the colorless-round spotted with colored shrunken sector kernels $a_1m En Sh2/a_1m-l sh2$.

Crosses (1980)	Round**					% with sectors
	Non-spotted		Spotted			
	Colored	Colorless	No sector	Big*	Small*	
A. $a_1m-l sh2/a_1m-l sh2$ $\times a_1m En Sh/a_1m-l sh$						
1. 2534/1857-2			21	1	3	16.0
2. 2550/1857-2t			82	2	0	2.4
3. 0125/1857-3			190	0	5	2.6
4. 0125/1857-4			153	2	10	7.3
5. 0125/1857-5			89	1	9	10.1
6. 2535/1857-6			158	0	26	14.1
7. 2536/1857-7t	1		161	0	16	9.0
8. 0125/1857-9			127	5	45	28.2
9. 0125/1857-9			207	0	20	8.8
10. 0125/1857-9t			186	2	12	7.0
11. 2550-1/1857-10t			82	2	0	2.4
12. 0104/1858-1m			69	0	1	1.4
B. $a_1m Sh/a_1m-l sh2$ $\times a_1m En Sh2/a_1m-l sh2$						
13. 0719/1857-3	90	1	161	1	0	0.6
14. 0948/1857-4	120		233	2	2	2.1
15. 2131/1857-7	54	1	93	0	1	1.1
16. 4201/1857-7t	20		48	0	1	2.0
17. 2132/1857-11	95		201	0	2	0.1
18. 0942/1858A-1t	69		135	2	13	10.0
19. 0944/1858-1t	20		68	0	1	1.4

*The area of big sector is larger than 0.5 mm^2
The area of small sector is smaller than 0.5 mm^2

*The shrunken non-spotted colored are not listed but appear approximately 1/2 in the A and 1/4 in the B crosses.

positive correlation ($r = .9123^*$) between the percentage of sectors of parental kernels and that of their corresponding progeny. This shows that selection can be made for the frequency of loss and is thus heritable. Varied size and frequency of the sector on the kernels (see last column of Table 1) probably depends on the state of this En.

Yih Ching Huang and Peter A. Peterson

Mutator activity and leaf striping

For some years now there has been some interest in leaf striping and its relationship to mutator (Mu) activity (MGCNL 55:2-3, 1981; 56:2, 1982). As was mentioned in the 1981 report, considerable striping was observed in Mu stocks grown in Berkeley. These plants were scored on a six-step scale from 1 (no striping) to 6 (heavily striped), self-pollinated, and outcrossed to a standard line. The outcross progeny were selfed and scored for the segregation of new mutants and the mutation frequency calculated for each outcross. The seedling classifications were made without knowledge of the striping classifications. After the mutation frequencies were determined, the striping determination for each tested plant was obtained and the coefficient of correlation (CC) was calculated for the frequency of mutants and the degree of striping. Two sets of plants were tested. One set was derived from striped mutator stocks that Dr. Michael Freeling had developed and the second consisted of some of my Mu stocks which had been previously grown in Iowa and had shown no striping. At Berkeley, striping was observed in some of these I.S.U. stocks.

The correlation results are summarized below:

	No. of plants tested	CC (total mutants)	t value	Prob*	CC (different mutants)	t value	Prob*
Freeling's	41	.1053	0.661	>0.5	.0170	0.160	>0.5
Robertson's	23	.4292	2.178	0.02-0.05	.3740	1.848	0.05-0.10
Total	64	.1807	1.448	0.1-0.2	.1349	1.072	0.20-0.30

*High probability = low likelihood of correlation.
Low probability = high likelihood of correlation.

The distributions of striping classes were as follows:

	1	2	3	4	5	6	Total
Freeling	10	8	6	12	2	3	41
%	24.4	19.5	14.6	29.3	4.9	7.3	
Robertson	13	3	5	0	2	0	23
%	56.6	13.0	21.7		8.7		

In Robertson's material the percentage of nonstriped plants was twice that of Freeling's, and there was low correlation of striping with mutator activity. In Robertson's stocks, although fewer plants striped, there was a correlation of mutator activity with striping.

I believe the difference in these two stocks can be explained on the basis of what is known about their origin and environmental factors influencing striping. When Freeling grew the original Mu stock I sent him at Berkeley, he observed striping. Assuming the striping was the result of Mu activity, he selected these plants for crossing and continued to select for striped plants (= Mu plants?) in future generations. Thus he had selected strongly striped stocks. Striping was rare in Robertson's stocks grown in Iowa and Hawaii and no selection for the striping phenotype was made.

In last year's News Letter (MGCNL 56:2, 1982) it was reported that low temperature will cause striping in not only Mu stock but also normal lines. With these facts in mind, the following might explain the experimental results. The growing conditions (i.e., low temperatures) at Berkeley are such that in most years striping will be induced in many plants, normal or Mu. Perhaps Mu plants are more susceptible to striping at low temperature (this is yet to be established, but if true, it would not be inconsistent with a viral infection, since viral symptoms are sometimes more extreme at lower temperatures). Thus, Mu plants grown at Berkeley may have two types of stripes: Mu-induced and those induced by low temperature. If, over several generations, selection for striping was carried out at Berkeley for plants that were more prone to cold-temperature-induced striping, then there would be little correlation expected between striping and Mu. On the other hand, Robertson's stocks were never subject to selection for cold-temperature-induced striping. Thus, when grown at Berkeley, more of the striping might have been due to the Mu response to cold temperature relative to the normal cold-temperature-induced striping, and hence the stronger correlation with striping.

These results suggest that striping might be a phenotypic expression of Mu. However, one has to be aware of the low temperature effect when trying to select

Mu plants on the basis of striping (e.g., among Freeling's material there were two striped plants that showed no mutator activity). Before striping can be used as a diagnostic guide for Mu it will be necessary to better characterize cold temperature striping as opposed to mutator striping, if indeed they are different phenomena. The fact that most Mu progeny are Mu will complicate the analysis because the chance of picking a Mu plant each generation, regardless of phenotype, is high. In my early work with mutator, this transmission pattern led me to wrongly conclude that mutator was associated with y9, since selection and propagation were made on the basis of y9 being present.

There is one additional observation that bears on the striping phenomenon. This last spring was unusually cool in Iowa. My first planting (April 30) germinated well, but after the two to three leaf stage there were a couple of weeks of cool weather. During this time little growth took place and there was extensive yellowing of the leaves. A few weeks later, for the first time in my field, I saw considerable striping in my Mu lines. Similar striping was not observed in my second planting (June 2). In checking with the plant extension workers on our campus, they informed me that they were deluged with calls from farmers this spring about striped plants in their fields. They further informed me that striping is a commonly observed phenomenon in cool springs, and that there is considerable varietal variation with regard to susceptibility to striping.

I have talked to our corn breeders here and they report seeing such cold temperature striping, but they are not aware of any studies on this phenomenon. If anyone knows of reports and/or studies on this effect of low temperatures, I would appreciate being made aware of them.

Donald S. Robertson

Genetic instability in maize-teosinte hybrids

Mangelsdorf and Galinat have reported several instances of increased mutation rates in derivatives of maize-teosinte hybrids. I have also tested for instability in such hybrids using the outcross-selfing-seedling-testing technique utilized in the testing of mutator lines to pick up mutants. I have limited results on a Mexican teosinte line obtained from Plant Introduction (PI 384063) and a Guatemala teosinte furnished by Dr. Galinat. All crosses were made onto corn silks. In some instances, the F1's had to be backcrossed using maize as the female parent to get ears that would furnish sufficient seeds for seedling tests. The results of these tests are as follows:

<u>Teosinte source</u>	<u>Generation tested</u>	<u>Total</u>	<u>Total mutants</u>	<u>% Total mutants</u>	<u>Total diff. mutants</u>	<u>% Diff. mutants</u>
Mexican	F1	39	0	0	0	0
Mexican	1st B.C.	72	1	1.4	1	1.4
Total Mexican		111	1	0.9	1	0.9
Guatemala	F1	96	4	4.2	3	3.1
Guatemala	1st B.C.	101	3	3.0	1	1.0
Total Guatemala		197	7	3.6	4	2.0

These data are too limited for any sweeping conclusions at the present time. They do support the findings of Mangelsdorf and Galinat on the genetic instability of maize-teosinte derivatives. In light of the hybrid dysgenesis work in Drosophila, it would be of interest to perform test crosses in which the cytoplasm was derived from teosinte rather than maize. Such tests are currently being carried out.

Donald S. Robertson

A case of possible unequal sister chromatid exchange in Mu stocks

In a large isolation block set up to isolate Mu-induced a2 mutants, the male parent was a Mu A2 Bt stock that carried the self-color R-scm2 allele. This allele also results in colored scutellum. The female parent was a2 bt. In this test 4 seeds were found that were yellow and had purple scutellum. It was assumed that these seeds resulted from pollen grains in which one sperm nucleus had an A2 to a2 mutation, while there was no mutation in the other sperm. To confirm this, the seeds were planted and self-pollinated. If these seeds were indeed due to a mutation in only the sperm fertilizing the polar fusion nucleus, the selfed ears should segregate 3 A2 Bt:1 a2 bt (with a few rare crossover classes). This was not observed! In all cases, about half of the seeds were A2 Bt.

An unequal sister chromatid exchange during the division of the generative nucleus could result in a duplicate (A2 A2) chromosome that would be selected against when the F1 plants were selfed, and the a2 bt pollen only (or predominantly) would function through the male. No such selection would occur on the female side, and thus the selfed ears would be expected to have the observed 1:1 ratios. (See Figure 1.)

To test this hypothesis, plants from A2 Bt seeds from the selfed ears were reciprocally crossed with a2 bt. The ears on the a2 bt plants would be expected to have predominantly a2 bt seeds since the duplicate chromosome would not be transmitted through the pollen. The A2 Bt ears should have A2 Bt and a2 bt seeds in approximately a 1:1 ratio. Basically, these results were observed. Only an occasional Bt and/or A2 seed was observed on the a2 bt ears (= crossovers?).

If the a2 bt stock had a fifth chromosome Ga factor, and the A2 Bt stock ga, similar results might be observed. But if that were the case, how would the original seeds have been produced in the first place? This is possible if the Ga system allows the functioning of ga pollen on silks, and if no Ga pollen was available to compete with that carrying ga. However, such a possibility does not explain the original yellow endosperm-purple scutellum seeds.

The a2 bt stock was obtained many years ago from the Coop. When I first tried using it, I ran into all kinds of difficulty because it had a very strong Ga factor. Pollen with the ga allele would not function on the silks of homozygous Ga plants, even if ga was the only type of pollen available. Such crosses failed to set seeds. Before I could use this stock, I had to eliminate the Ga factor. There has been no evidence of Ga in this stock for years, during which time it has been used in a variety of different crosses.

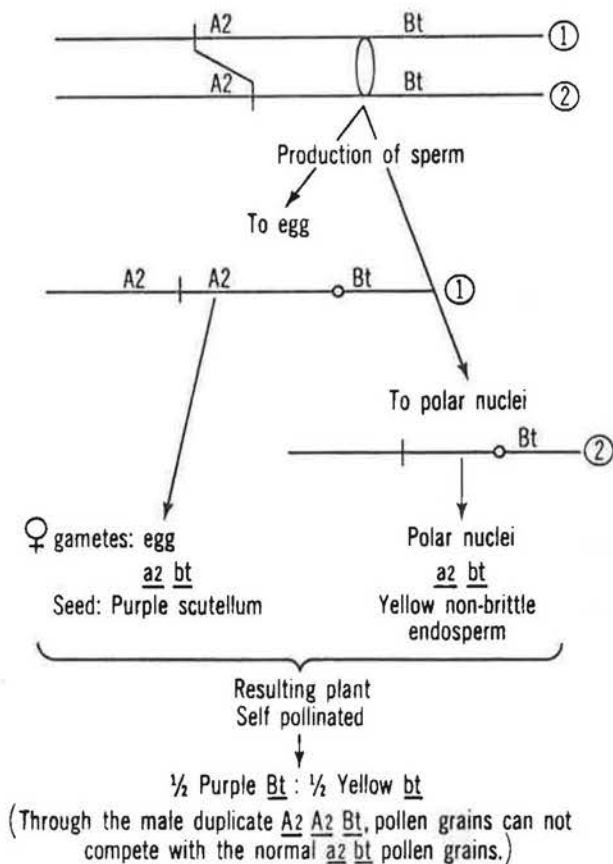


Figure 1

A few years ago I carried out an extensive test of crossing over in the a2-bt region using reciprocal tests. The purple stock used in this cross was the same

one used to produce the A2 Bt Mu line, and the a2 bt was the same as the one used in these tests. There was no evidence of a Ga factor being involved in these crossover tests!

It seems very likely that unequal sister chromatid exchange was responsible for producing the original seeds. If this is true, and if the duplications are long enough, it may be possible to demonstrate them cytologically.

Donald S. Robertson

Pollen sterility in inbred Mu stocks

In last year's News Letter (MGCNL 56:4-8, 1982), it was reported that Mu activity was lost as Mu stocks were inbred. One possible explanation of this loss could be the failure to transmit Mu as the dose of Mu increases. This might be accomplished by a Mu-induced abortion of ovules and pollen grains carrying high doses of Mu. That this does not take place in stocks that have been propagated by outbreeding, the usual method of propagation, is well established by the results of many such crosses. At least 87% of all outcross plants receive Mu. In last year's report we reported observations on ear sterility which might be indicative of ovule abortion in inbred populations. No consistent pattern of abortion was observed that would suggest high Mu doses result in such abortion.

This past summer we grew populations of Mu² (Mu¹ x Mu¹), Mu⁴ (Mu² x Mu²), Mu⁸ (Mu⁴ x Mu⁴), Mu¹⁶ (Mu⁸ x Mu⁸) and Mu³² (Mu¹⁶ x Mu¹⁶) and classified them for pollen abortion (Table 1).

Table 1. Pollen sterility in inbred Mu stocks.

Levels of <u>Mu</u> inbreeding 1982	Pollen sterility (%)									Total	
	n	n ⁻	n ⁵	n ¹⁰	n - n ¹⁰	n ¹⁵	n ²⁰	☞ ²⁵	☞ ⁴⁰		☞ ⁻
<u>Mu</u> ²	16(59.3)	2(7.4)	2(7.4)	3(11.1)	23(85.2)		1(3.7)	1(3.7)		1(3.7)	27
<u>Mu</u> ⁴	20(62.5)		4(12.5)	1(3.1)	25(78.1)	2(6.3)	3(9.4)	1(3.1)			32
<u>Mu</u> ⁸	18(66.7)		2(7.4)	4(14.8)	24(88.9)	2(7.4)	1(3.7)				27
<u>Mu</u> ¹⁶	29(96.7)				29(96.7)				1(3.3)		30
<u>Mu</u> ³²	26(86.7)			2(6.7)	28(93.4)	1(3.3)		1(3.3)			30
1979											
<u>Mu</u> ¹	379(87.3)		13(3.0)	19(4.4)	411(94.7)	18(4.1)	3(0.7)	1(0.2)	1(0.2)		434
Non-Mu	316(94.9)		7(2.1)	8(2.4)	331(99.4)	1(6.3)				1(0.3)	333

1982 Data - Heterogeneity $\chi^2 = 6.2635 = p .10-.20$

*n = no abortive pollen grains, n⁻ = few abortive grains, n⁵ = 5% abortive grains, etc. ---

☞²⁵ = 25% abortive pollen grains --- ☞⁻ = a little less than 50% abortive pollen grains, ☞ = 50% abortive pollen grains.

Again, there is no consistent pattern that would indicate Mu-induced pollen abortion is taking place. It is possible that pollen grains with high doses of Mu might not appear abortive and yet not be able to function or compete against normal pollen grains (with few or no Mu's). However, in producing these Mu stocks, each generation the female parent theoretically contributes as many Mu's to the next generation as the male parent. Since there is no evidence of

Mu-induced sterility on ears of such a cross, the female-contributed Mu's should be transmitted, and thus with inbreeding a certain minimal level of Mu, the female transmitted level, should be maintained. This has not been observed.

In sum, there is no good evidence that the loss in Mu activity when Mu stocks are inbred is due to selection against gametophytes with high Mu doses.

Donald S. Robertson

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Indirect evidence for alignment in advance of synapsis, with frequency dependent on extent of available homology

It is often proposed that meiotic homologue pairing depends primarily upon prior juxtaposition of homologous centromeres and/or telomeres, with synapsis initiated first near these structures and then zipping to completion. If this indeed is the case, there is nevertheless reason to believe that there is a crucial prealignment stage which seems to occur, in some cases at least, with a frequency directly related to the extent of homologous chromosome length.

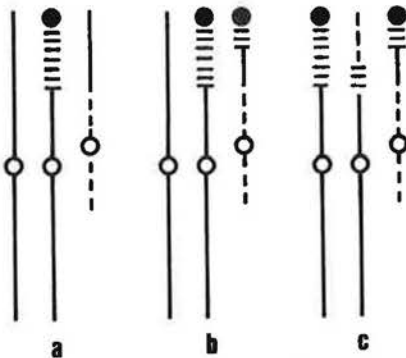


Figure 1

Figure 1 diagrams chromosome constitution with respect to maize chromosome 2 and a Tripsacum partial homologue in three interchange stocks (M.P. Maguire: *Genetics* 51:23-40; *Genet. Res., Camb.* 12: 21-27). Maize chromosome 2 is indicated by solid lines, the Tripsacum chromosome homoeologous portion by parallel horizontal bars, and the remainder of the Tripsacum chromosome by dotted lines; centromeres are designated by open circles, and there is a terminal knob on the Tripsacum homoeologous portion. The centromeres of maize chromosome 2 and the Tripsacum chromosome are not homologous.

Frequency of metaphase I trivalents is about 90% in types a and b, and about 54% in type c. Trivalent occurrence at metaphase I depends, in types a and c, upon homologous pairing and crossing over having occurred between the chromosome with the Tripsacum centromere and one of the two chromosomes carrying a maize centromere. In type a, with approximately twice the extent of homology available for these events (vs. type c), there is roughly twice the frequency of trivalents (vs. type c). In type b, the same total extent of homology is available for trivalent formation as in type a, but homology to the chromosome carrying the Tripsacum centromere is shared between the two maize centromere-carrying chromosomes, approximately equally. At pachytene in type b, in 24 cells with a trivalent configuration where analysis was possible, 19 contained homologous synapsis of the distal region only, 3 contained homologous synapsis of the intercalary region only, and 2 contained homologous synapsis of both distal and intercalary regions (with change of pairing partner). If this is a representative sample, most trivalent formation depended upon synapsis of the distal region in type b. Nevertheless, the frequency of both homologous synapsis and crossing over in type b corresponded to expectation from availability for these events of an extent equal to the combined potential extent within both distal and intercalary regions.

In the absence of some unrecognized artifact or sampling error, it is difficult to escape the conclusion that both synapsis and crossing over depend upon total extent of available homology, although most of the occurrences of these events seem to have been shunted into the distal region in type b. Is commitment to both synapsis and crossing over established at prealignment in a manner partially dependent on intercalary chromosome regions, although actual synaptic and crossover events tend to be distally located?

Marjorie Maguire

Sporadic, frequent bivalent interlocking at diakinesis in a variety of stocks

Clear interlocking of bivalents at diakinesis has now been found in sporocyte samples of plants from a number of widely differing stocks, although sibling plants of these stocks appear completely normal in this respect. Stocks involved include a Coop elongate stock, G. Y. Kikudome's 9K*Ks stock, Coop desynaptic stock, Coop Inv. 3a stock (which actually contained two B chromosomes), and KYS. In samples where the abnormality was found, some cells were normal, most contained one or two pairs of interlocked bivalents and some contained snarls involving all or most of the chromosomes. At pachytene, many cells contained untraceable, snarled knots involving many chromosomes, although very little synaptic failure was seen. Interlocking could be seen at metaphase I, but in most cases, resolution of the interlocks had yielded normal appearing cells by anaphase I, and later meiotic stages appeared normal.

All of these samples were from growth chamber grown plants, and it is suspected that the abnormality is a result of some aspect of the growth chamber condition which may have differentially affected these plants. I am interested in knowing whether others have found abnormalities of this sort in field grown or growth chamber material.

Marjorie Maguire

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Instituto de Biologia de Barcelona, C.S.I.C.

Pollination effects on grain yield and qualities of fertilized plants

Previous research suggests that the genetic information the pollen brings to the embryo and to the endosperm affects not only the vigour of the seed, but also the female parent plant photosynthesis capacity (Blanco, M., Doctoral thesis, Univ. of Zaragoza, May 1972; Bosch, L., and Blanco, M., et al. MNL 52:122-123, 1978). The present work is to test for differential interaction between the plant and the seed produced on it, in respect to: (1) The relationship between their respective genetical constitutions, (2) specific genes, and (3) cytoplasmic effects.

The material was (a) Two inbred lines of quite different origins, each in an o_2/o_2 version and a $+/+$ version: A o_2 , A +, B o_2 , B +; (b) the F1 single cross, A o_2 x B o_2 (direct cross), and the reciprocal, B o_2 x A o_2 ; and (c) the F2 of both direct and reciprocal single crosses.

As treatments, the inbreds were self-pollinated; both classes of F1 plants were (1) not pollinated, (2) self-pollinated, (3) pollinated with each version of both parent lines (A +, A o_2 , B +, B o_2); and both classes of F2 plants were self-pollinated.

The experimental design was "randomized plots" of 3 replications (one plot for each class of material and type of pollination; 25 plants per plot). The pollinated plants with ears not perfectly set were discarded. On each plant, 3

parameters were measured: (1) Refractometric reading of the stalk juice at maturity of the grain (34% moisture of the grain); (2) Dry weight of the ear/plant (15% moisture); and (3) Total dry weight/plant.

In each group of material (a, b and c) there were significant differences in reaction between corresponding treatments. This lets us advance that plants react specifically to the different classes of seeds, and vice versa. Such reactions depend on the relationship between their cytoplasm, genetic backgrounds and specific genes (o2 versus +). The results do not invalidate the hypothesis that in some cases the seed stimulates and increases the photosynthesis capacity of its mother plant.

Besides the working hypothesis, the homozygous o2 seeds in some of the cases were significantly superior in weight to the corresponding heterozygous seeds. Thus, "opaque 2" should not be considered a "defective" gene.

M. Blanco, P. Fontanet, A. Alvarez, J. Montserrat, and J. L. Blanco

BELTSVILLE, MARYLAND
Agricultural Research Service, USDA

Latin American maize collections

Negotiations are in final stages for cooperative projects in Colombia, Mexico, and Peru for U.S. maize scientists to work with them on the increase, evaluation, and preservation of maize collections.

For more details write: Dr. Quentin Jones, National Coordinator, National Plant Germplasm System, USDA/ARS, Bldg. 005, Beltsville, MD 20705.

Quentin Jones

BERKELEY, CALIFORNIA
University of California

A DNA insertion within Adh1 is transcribed

Last year H. P. Döring, M. Motto, F. Salamini and P. Starlinger (MNL 56:40, 1982) described an Adh1 mutant they recovered from plants homozygous for bz2-m, containing Ac. The mutant, 2F11, has no ADH1 activity and presumably contains a Ds element affecting the Adh1 gene. In collaboration with Starlinger's lab, we have been studying 2F11 to determine: (1) if it responds to Ac, (2) whether there is an insertion at or near the Adh1 gene, and (3) if there is transcription of the Adh1 gene. This note will describe the RNA data; the genetic and restriction mapping data will be described elsewhere.

I compared poly-adenylated ADH1-RNA from roots of 2F11 and a standard line, 1S, after seedlings were subjected to 12 hours of anaerobic stress. The RNA samples were electrophoresed on a denaturing formaldehyde gel, transferred to nitrocellulose and probed with the Adh1 cDNA clone, pZML84 (Figure 1). The ADH1-RNA of 2F11 is present to the same extent as in 1S, but the 2F11 RNA is 2 kb larger. The increase in size of the message is compatible with the results of our Southern data that demonstrate the presence of a 2 kb insert within the coding region (to be published elsewhere). In addition to the larger ADH1-RNA of 2F11, a minor amount of normal size message (1650 bp) is also present. The normal size message is probably not ADH2 because of the stringency of the hybridization and wash conditions (T_m -20 C and T_m -10 C, respectively). To determine if transcription starts and

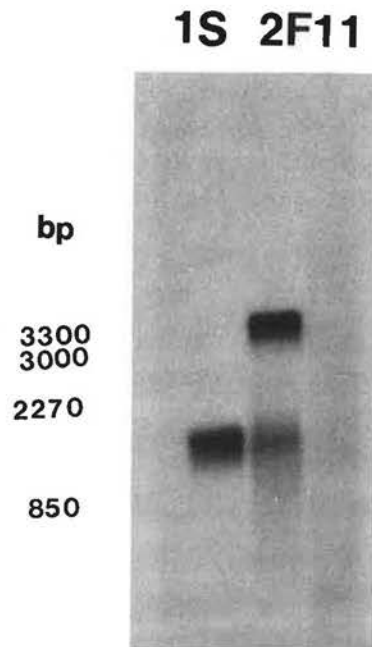


Figure 1. 2F11 and 1S seedlings were subjected to 12 hours of anaerobic stress. One ug of poly-adenylated RNA from 1S and 2F11 anaerobic roots were electrophoresed on a formaldehyde 1% agarose gel. The gel was transferred to nitrocellulose and probed with an Adh1 cDNA clone. BMV RNA was used as a size standard.

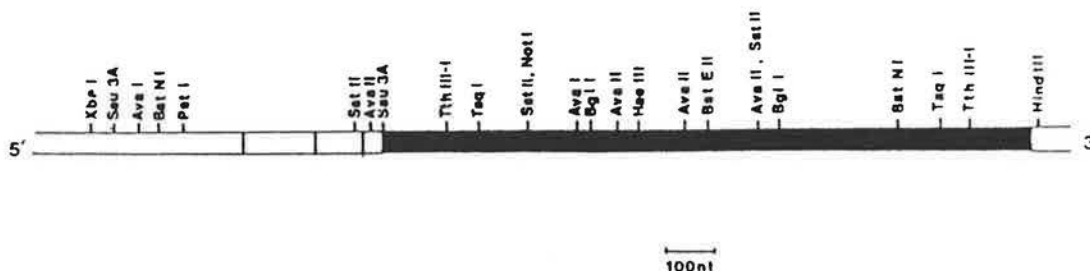
stops at the normal positions, 2F11 ADH1-RNA was probed with 3' and 5' fragments of the Adh1 genomic clone, pB428. The results of this experiment showed that the novel transcript has Adh1 coding sequence surrounding the insertion.

Our interest in 2F11 goes beyond the Adh1 gene. 2F11 displays a mild knotted phenotype that is linked to Adh1 (M. Freeling, pers. comm.). The knotted gene, Kn1, is on chromosome 1 and is the nearest known neighbor to Adh1 (< 0.1 mu). We intend to ask if the knotted phenotype is due to a position effect of the insertion at Adh1, or if Kn1 is at the insertional site within the Adh1 coding sequence (seems unlikely?) or, something more complicated.

Sarah Hake

Dear Maize Geneticists:

In the course of analyzing Adh1 mutants which had been induced in a Robertson's mutator genetic background, we have had the good fortune to capture a maize insertional sequence (Freeling, Cheng and Alleman, Develop. Genet., in press; Strommer, Hake, Bennetzen, Taylor and Freeling, 1982, Nature 300:542-544). Our mutator lines were a gift of Don Robertson. A 13 kb BamHI fragment containing the mutator-induced allele Adh1-S3034 was isolated from a lambda library. The cloning was done by Jeff Bennetzen at International Plant Research Institute, San Carlos, CA (Bennetzen, Swanson and Freeling, in preparation). Adh1-S3034 contains a 1.35 kb insert in an intervening sequence close to the 5' end of the gene. We call this insertional sequence Mu1 and believe it to be responsible for the induction of the



Restriction map of 5' end of Adh1 gene with Mu1 insert. The allele is Adh1-S3034

unstable mutants caused by Robertson's mutator genetic background. Typical maize cultivars contain no sequences with any detectable homology to Mu1, while lines presumed to carry Robertson's mutator genetic background contain very approximately 30 dispersed, intact Mu1 elements (Bennetzen and Swanson, in preparation).

Robertson (1978, Mut. Res. 51:21-28) has recovered several recessive mutants from plants carrying mutator genetic background, as have other investigators. We think that Mu1 insertion mutants may provide a handle with which to clone these dysfunctional genes. We will distribute a Mu1 specific clone to those interested in pursuing this strategy. Please write to Michael Freeling on or after March 1, 1983.

Being realists, we know that once Mu1 is released it is out of anyone's control. Since we have not yet published on Mu1 and related sequences, we hope that those who must compete with us directly will keep in touch. In particular, the use of Mu1 as an integration module for transformation studies interests us. We would like to hear about all results using Mu1, whether interesting or not.

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Identification and characterization of the embryo-specific proteins in maize

The major proteins specific to maize embryos have been identified as the products of the Pro locus on chromosome 1 (MGG 174:233-240, 1979) (this locus will be referred to as Prot in accordance with its current designation by the Maize Genetics Coop). This locus codes for two proteins, PROT' and PROT; PROT' is about 5,000 daltons larger than PROT (three size alleles, Prot-L, Prot-I and Prot-S, with molecular weights ranging from about 61,000 to 68,000 daltons, have been reported). These proteins fit the criteria of storage proteins as defined by Derbyshire et al. (Phytochemistry 15:3-24, 1976): they are first detected (by SDS-polyacrylamide gel electrophoresis) about 20 days post-pollination, they account for up to 30% of total protein in mature embryos, and they are rapidly degraded on germination. These proteins are classified as globulins by virtue of their solubility in 1M NaCl and insolubility in water, and by precipitation at pH 4.7. These characteristics also suggest that the products of the Prot locus comprise the "embryo antigen" P7 of Khavkin et al. (Planta 143:11-20, 1979).

Extensive homology between PROT' and PROT is observed by digestion with trypsin, chymotrypsin, *S. aureus* V8 protease, formic acid, and cyanogen bromide; the proteins specified by the three size alleles also show considerable homology in peptide digests. The cyanogen bromide digests have been found to be quite informative when comparing the size alleles. Cyanogen bromide cleaves the proteins into two major fragments: the size difference between PROT' and PROT is limited to the larger fragment, whereas the size differences observed between the proteins specified by the different size alleles are limited to the smaller fragment. Experiments are in progress to determine which fragment represents the amino-terminus of the protein.

PROT' is first detected in SDS gels about 20 days post-pollination, and PROT first appears two to three days later. The unlinked locus Mep (chromosome 5) controls the synthesis of PROT. In homozygous mep embryos, very little PROT is synthesized, and PROT' accumulates. The action of Mep does not appear to involve protein processing since preliminary in vivo experiments have failed to show a chase of radioactively-labeled amino acids from PROT' to PROT in immature embryos pulsed with label. It appears that the action of Mep is at the level of transcription or RNA processing.

A1 Kriz and Drew Schwartz

Further studies on two-unit mutable systems found in our high-loss studies and on the specificity of interaction of responding and controlling elements

In the 1982 Maize News Letter we presented a chart summarizing the interactions of the responding and controlling elements comprising previously studied two-unit mutable systems, as well as those of three new systems which arose in our high-loss studies. Several of the interactions had not been determined and were listed as "testing" or "to be confirmed." The missing diagnoses have now been ascertained and the complete set of interactions is given below where a plus signifies a positive and a minus sign a negative response. A positive interaction is evidenced either by breaks induced at the responding locus, followed by loss of the chromatin segment distal to the break, or by excision of the inhibiting Ds-like element from the affected locus, resulting in full or partial recovery of gene activity in a structurally intact chromosome. McClintock designated breakage as a Type I and excision of the regulatory element as a Type II event. Failure to find either breakage or excision would be scored as a negative response.

The Ds element used in our tests was proximal to Wx in the short arm of chromosome 9, and not between C-I and Wx as indicated in the pedigree provided by the Maize Stock Center. This Ds (called standard Ds) is not associated with any mutant phenotype and its interaction with Ac is demonstrated by Ac-induced breaks at Ds, resulting in the joint loss of the linked C-I and Wx markers. Phenotypically, there are colored, waxy (C wx) spots on a white, starchy (C-I Wx) background in endosperms of C wx/C wx/C-I Wx Ds; Ac constitution. Excision of Ds (Type II event) would not readily be detected with our Ds stock. It is only when transposition of Ds occurs and it is moved into or adjacent to a wild type structural gene, thereby inhibiting the activity of that locus and giving a recessive mutant phenotype, that excision of Ds from the inhibited locus is made evident by the recovery of full or partial gene activity in that cell and its descendants.

Controlling element	Responding loci					
	a-mrh	a-standard	a-m, a-mr	Ds+	bz2-m	bz-mut
<u>Mrh</u>	+	-	-	-	-	-
<u>Dt</u>	-	+	-	-	-	-
<u>En=Spm</u>	-	-	+	-	-	-
<u>Ac=Mp</u>	-	-	-	+	+	-
<u>Ac2</u>	-	-	-	-*	+	-
<u>Mut</u>	-	-	-	-	-	+

†The Ds tested is located proximal to Wx on chromosome 9.

*See following discussion.

kernels lacking their specific controlling element. Mutability of a-mrh is induced by Mrh and not by Dt or En, mutability of a-standard by Dt and not by Mrh or En, and mutability of both a-m and a-mr by En but not by Mrh or Dt. The chart could be enlarged to include other responsive loci into which Ds has been inserted, but nothing new in principle would be gained.

The behavior of Ac2 is anomalous. When McClintock's Ac is tested against Ds in the C-I Wx Ds chromosome, a frequent loss of both C-I and Wx occurs as the consequence of Ac-induced breaks in the C-I Wx Ds chromosome (event Type I). Her Ac tested against bz2-m, which has a Ds-like element in or adjacent to a wild type Bz2 allele, causes the excision of Ds from Ds Bz2. We have not yet determined whether or not Ac causes excision of Ds (Type II event) from the C-I Wx Ds chromosome or breaks (Type I) in the bz2-m chromosome. However, since Ac2 also induces mutability of bz2-m (although differing from Ac in dosage effects, time of mutation and its restriction to specific tissues), it might be anticipated that Ac2 would cause breaks in the C-I Wx Ds chromosome. This was not the case. The frequency of C wx spots in C wx/C wx/C-I Wx Ds endosperms having two Ac2 was not discernibly different from that in similar endosperms with no Ac2. We concluded that Ds in the C-I Wx Ds chromosome differed from the Ds-like component in bz2-m, since the former gave a Type I response to Ac but not to Ac2. Both Ac and Ac2 induced Type II events at bz2-m, but they differed in that Ac induced excision of the Ds element of the wx-m-1 and c-m-1 alleles, while Ac2 was completely ineffective in eliciting a Type II response at either locus. Our tests prior to 1982 established that unlike Ac, Ac2 had no effect on Ds in the C-I Wx Ds chromosome, on mutability of wx-m-1 (= Ds Wx), on c-m-1 (= Ds C) nor on wx-m-9 (= Ac Wx). It seemed, therefore that the response of bz2-m to Ac and Ac2 was due to some unknown modification in the molecular structure of the Ds component of the bz2-m allele.

Some recently obtained data cast doubt on the validity of this conclusion. The response of bz2-m to increased doses of Ac2 is dramatic. No (or rare) Bz2 spots of aleurone color are found in kernels homozygous for bz2-m and possessing one Ac2, a low number occurs with two Ac2 and many mutations of bz2 to Bz2 are found with three doses of Ac2. Inasmuch as we had found (see our 1982 News Letter report) instances where two Ac2 elements were situated in juxtaposition in the same chromosome, we thought it of interest to see what the response of Ds in the C-I Wx Ds chromosome would be to four doses of Ac2. We had observed no detectable response of this Ds to two Ac2, but since the mutation rate of bz2-m was so dramatically increased in going from two to three doses of Ac2, it seemed possible that Ds in

its standard position might exhibit a comparable response to higher numbers of Ac2. Crosses of bz2-m/bz2-m; C-w wx/C-w wx; Ac2 Ac2/ac2 ear parent x Bz2/Bz2; C-I Wx Ds/C-I Wx Ds; ac2 pollen parent should give equal numbers of F1 endosperms with two genotypes: (1) bz2-m/bz2-m/Bz2; Ac2 Ac2/Ac2 Ac2/ac2; C-w wx/C-w wx/C-I Wx Ds and (2) bz2-m/bz2-m/Bz2; ac2/ac2/ac2; C-w wx/C-w wx/C-I Wx Ds. The C-I allele was dominant to two doses of the weak C-w allele. Half of the kernels had four doses of Ac2 and one standard Ds, while the other half had no Ac2 and one Ds. A number of ears were obtained from this cross. The kernels on each ear fell into two distinct classes in a 1:1 ratio. One-half had a colorless aleurone with starch which stained blue with IKI. The majority of the kernels in this class had no colored-waxy (C wx) spots resulting from loss of the C-I Wx markers, but a low percent had a few colored-waxy spots which were ascribed to spontaneous loss. In sharp contrast to this group with no or low loss of the C-I Wx markers was the 50 percent of the kernels with a colorless-wx background on which there were many (literally hundreds) of colored-waxy spots. Clearly, frequent loss of C-I and Wx was occurring during endosperm development in this half of the F1 kernels, while the other half had no or a very low rate of loss. We knew from the pedigree of the parental plants that one-half of the kernels had four doses of Ac2 and the other half had none, but at this stage in our investigation we could not tell if the kernels with the high rate of C-I Wx loss also possessed four Ac2 elements with the other half having none. However, by testcrossing plants from kernels with a high rate of loss and plants from kernels with no loss of C-I Wx, we were able to conclusively demonstrate that all high rate kernels had four Ac2 while kernels with a low rate of loss had none. This demonstration was possible because the F1 plants were all heterozygous for bz2-m. If all bz2 kernels in a given testcross population had a stable bronze aleurone color, it was concluded that the two linked Ac2 were not carried by the tested plant. Conversely, if a testcrossed ear had equal numbers of bz2-mutable and bz2-stable kernels, the testcrossed parental plant was heterozygous for the two linked Ac2. Several score of plants coming from the two classes of endosperm were testcrossed, and without exception the kernels with a high rate of loss of the C-I Wx markers gave rise to plants which segregated for mutable and stable bronze-2 phenotypes in a 1:1 ratio. These plants were demonstrably heterozygous for Ac2. Plants derived from kernels with no or few losses of C-I and Wx had no Ac2 since no mutable bronze-2 kernels were found. These results challenge our earlier conclusion that Ds in its standard location does not respond to Ac2. It does respond, but only when four doses of Ac2 are present. We know that two or three doses of Ac2 induce Type II events (excision) at bz2-m but not in the wx-m-1 and c-m-1 mutable systems, and that increasing the dosage of Ac2 does not affect the mutation pattern of wx-m-9 as do extra copies of Ac.

In summary, breaks at Ds in its standard position are not induced by two doses of Ac2, while many breaks occur when the dosage of Ac2 is increased from two to four. This suggests that Ac2 is a weak variant of McClintock's Ac. Support for this tentative conclusion comes from the following observations. The wx-m-1 allele is a Wx allele with an inserted Ds which inhibits the activity of the Wx gene. Endosperms homozygous for wx-m-1, or having various combinations of wx-m-1 and recessive wx, have no Wx reversions if Ac is not present, but show many Wx spots upon the introduction of Ac. Since kernels with wx-m-1 and one to three doses of Ac2 have only wx starch in the endosperm, we concluded that Ac2 was unable to induce excision of the Ds element in wx-m-1. This observation strengthened our conclusion that Ac and Ac2 were too dissimilar for the one to be a mutant derivative of the other. However, in kernels with four doses of Ac2, standard Ds did undergo breakage, so a similar test was made of the effect of more Ac2 on the wx-m-1 system. Waxy kernels from crosses of bz2-m/bz2-m; Ac2 Ac2/ac2; wx/wx silks by Bz2/Bz2; ac2/ac2; wx-m-1/wx-m-1 pollen in the summer of 1982 gave endosperms of two different genotypes. They differed in that one class had four doses of Ac2 and

the other class had none. All kernels are Bz2 phenotypically and have one dose of wx-m-1. Upon closely examining the endosperms from this cross, we found a few with an occasional, usually small, blue or lavender stained spot with IKI. Tests are presently being conducted to determine if all of the relatively infrequent kernels with rare blue or lavender staining cells are heterozygous for the two linked Ac2. Our prediction is that they are, and that wx-m-1 shows some response to four doses of Ac2 although it showed no response to one, two, or three doses. If our predictions are confirmed, a reasonable interpretation of our data might be that Ac2 is an anemic version of Ac, but we are hesitant in accepting this conclusion because the two differ in so many attributes. Whatever the outcome, it would appear that there is heterogeneity among the different Ds elements since they have dissimilar responses to the same activator. This saga will be continued in next year's News Letter.

M. M. Rhoades and Ellen Dempsey

On the mapping of controlling elements and their effect on recombination

We reported in the 1982 Maize News Letter that Mut, the controlling element producing mutability of bz-mut, was probably in chromosome 2 since in heterozygotes for the wx T2-9b translocation there was 32% recombination between Mut and wx, but no Mut wx linkage was found in other wx translocation heterozygotes. The location of Mut in chromosome 2 has been confirmed by orthodox linkage tests. Heterozygotes of Ws3 Lg Mut G12/ws3 lg mut gl2; bz-mut/bz-mut constitution were crossed as the female parent with a ws3 lg mut gl2; bz-mut pollen parent. The ensuing kernels were scored for bronze mutable and bronze stable and planted in a sandbench where the seedlings were classified for ws3, lg, and gl2. The following 4-point test-cross data were obtained:

<u>Crossover Region</u>	<u>Phenotype</u>	<u>No. of Individuals</u>	
0	Ws Lg Mut G1	240	
0	ws lg mut gl	236	
1	Ws lg mut gl	32	
1	ws Lg Mut G1	24	
2	Ws Lg mut gl	46	Region 1 <u>Ws3 Lg</u> = 8.8% crossing over
2	ws lg Mut G1	46	
3	Ws Lg Mut gl	22	Region 2 <u>Lg Mut</u> = 15.8% " "
3	ws lg mut G1	16	
1-3	Ws lg mut G1	1	Region 3 <u>Mut G12</u> = 8.1% " "
1-3	ws Lg Mut gl	1	
2-3	Ws Lg mut G1	9	
2-3	ws lg Mut gl	4	
1-2-3	Ws lg Mut gl	1	
1-2-3	ws Lg mut G1	1	
		<u>679</u>	

The linear order and intervening crossover distances are Ws3 - 8.8 - Lg - 15.8 - Mut - 8.1 - G12, which place Mut in the short arm of chromosome 2. Since controlling elements are subject to transposition and move from chromosome to chromosome, no permanent map position can be assigned them. Nevertheless, the controlling element present in a particular stock can be located with considerable accuracy, as is shown by our positioning of Mrh in 9L and of Mut in 2S. The rate of transposition of controlling elements is not so frequent that map position cannot be determined, although not with the same precision as for conventional loci. Even though germinal transpositions may be relatively rare, they are not

without their effect on recombination data. The testcross data given above show an absence of chiasma interference for double exchanges in the 2-3 region (coincidence =1.59). Interference is normally high for short adjacent regions in this arm since we have found a coincidence of 0.13 for Ws3 Lg G12 doubles (MGCNL 33:54). At first glance it would appear that Mut, although heterozygous, eliminates chiasma interference in regions flanking it. However, if, in the above data, we ignore the Mut marker and consider 3-point data involving Ws3 Lg G12 loci, the coincidence value for doubles is very low. The apparent lack of interference in regions flanking Mut is caused, so we argue, by the excision of Mut from its parental chromosome and its insertion in a heterologous chromosome. Through meiotic segregation the recipient heterologous chromosome would fail in a predictable frequency to pass to the same pole as the donor chromosome. A functional gamete of this constitution would simulate a Ws3 Lg mut G12 double crossover and be scored as such. Obviously, more data are needed to substantiate this conclusion, but we are confident that heterozygosity for Mut does not eliminate chiasma interference. The validity of this scenario is being checked by determining the genetic constitution of all putative double crossovers involving flanking regions. Incidentally, the analysis of these apparent double crossovers may be an efficient method of screening for new transpositional events.

In the 1982 News Letter we presented evidence of an increase or amplification in the number of Ac2 elements present in an individual plant. The most frequent increase is from one to two. In approximately half the cases, the two Ac2 elements are in heterologous chromosomes, or else are so distantly situated in the same chromosome that they are independently segregated in meiosis, since in a testcross a 3:1 ratio of bronze-2 mutable to bronze-2 stable kernels is found. In the other half, the two Ac2 are in juxtaposition in the same chromosome. In order to demonstrate that transposition to heterologous chromosomes does occur, we are determining the chromosomal location of newly arisen Ac2, which no longer occupy the parental site in the long arm of chromosome 8. Several cases of transposed Ac2 are under study but in only one instance have we ascertained its new location. This is a transposed Ac2 (tr-Ac2) now in chromosome 3 as demonstrated by the following data, which are scant, because of vandalism in the experimental field, but convincing. Plants of bz2-m/bz2-m; G16 tr-Ac2 Lg2/g16 ac lg2 constitution were pollinated by bz2-m; g16 ac2 lg2 testers. A total of 314 bz2 mutable to 414 bz2 stable kernels was obtained. The excess of bronze stable is the result of a low number of Bz2 dots on the bz2 mutable kernels; consequently some kernels carrying the tr-Ac2 failed to have mutations of bz2 to Bz2. Linkage calculations were, therefore, restricted to the bz2-mutable kernels since they were known to possess the transposed Ac2. The following data were obtained:

0	<u>G16 Ac2 Lg2</u>	205	
1	<u>g16 Ac2 Lg2</u>	60	<u>G1 Ac2</u> = 22.8% crossing over
2	<u>G16 Ac2 lg2</u>	19	
1-2	<u>g16 Ac2 lg2</u>	6	<u>Ac2 Lg2</u> = 8.6% " "
		290	

The transposed Ac2 clearly lies between G16 and Lg2 in 3L. The data are far too meager to be conclusive, but it is of interest that the coincidence value is greater than 1.0. We believe that, as in the chromosome 2 linkage study, transposition of the controlling element to a heterologous chromosome is responsible for many of the apparent double crossovers.

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Spontaneous cell fusion during meiosis leads to unidirectional chromosome modification and B chromosome formation

In the last two years we have reported on our studies of maize from Sikkim, a popcorn of the Eastern Himalayas. During the study of the chromosome knob pattern a low frequency of abnormal meiosis was discovered in certain lines. Further investigation of pre-prophase to very early prophase stages discovered cell fusion of clonal pollen mother cells in the most precocious (early-to-flower) plants. It appears to us that these cells did not form a primary cell wall (possibly because of disruption induced by the very rapid photoinduction from long days of this short day tropical maize), and the naked cell membranes of cells in physical contact were subject to a process best described as fusion.

There is no question of the fusion and subsequent spontaneous increase in chromosome number (we have scored approximately 17,000 cells), but we are cautious about our observations because to our knowledge this is a first report. Following fusion, partially modified supernumerary chromosomes appear to stabilize their number and are known to be inherited through the third generation.

Plasmodiums of both meiotic cells and mitotic cells, plus somatic nurse cells, have been observed. Cell fusion composed of 3 or more cells always appears to abort, but the fusion of two cells resulted in a spectrum of products, from "stable" autotetraploids to the complete elimination of the invading complement and return to the 'normal' condition.

Most intermediate conditions involved one complement which appeared to differentially disintegrate while the other complement retained normal characteristics. Chromosome modification appeared to be unidirectional, with only the chromosomes from one complement undergoing partial to total modification and ultimately elimination. The conclusion that this modification-elimination was unidirectional is based on both direct microscopic observations and the recovery of normal karyotypes with supernumerary chromosomes in subsequent generations. We have stabilized lines through the F3 generation with $2n + 1B$, $2n + 2B$'s, and $2n + 3B$'s, the first being the most frequent, following fusion in the original parent. The early photoinduction is inherited, and these selfed stocks are 30 to 45 days earlier than the original Sikkim collection in days to flowering. The day length response genes we assume to be on the normal complement.

We have observed these stabilized heterochromatic chromosomes to be no different from B chromosomes. Despite the considerable work done on supernumerary or B chromosomes, nothing is definitely known about their origin. One hypothesis, that B chromosomes have an origin from A chromosomes, has never been confirmed by direct cytological observations. In our observations, we have followed what we take to be the origin of a B-like chromosome following stabilization of the invading chromosome set or complement created by fusion. The complement that remains "normal" following fusion is the one that was in a slightly more advanced stage of meiosis. We have called this the invaded cell.

Cells which had acquired additional modified chromosomes and which appeared to have stabilized were found to fall into two categories. In the first category, the additional chromosomes were meiotically active. These cells were stable throughout meiosis. The second category was broader and included acentric fragments and additional chromosomes which failed to contract (and were defined as not meiotically active). In the latter case, these chromosomes were usually found in a pachytene-like state of contraction in cells where the other chromosomes were at more advanced and essentially 'normal' stages of meiosis. These unusual cells suggested to us that fusion has occurred before or at pachytene, and not at advanced stages.

Although the causes of cell fusion and unidirectional chromosome modification are not understood, a sufficient number of observations have been made to realize that this is a real phenomenon and conclusions can be drawn. This report has shown that additional chromosomes, whether modified or not, can arise spontaneously in cells as a result of cell fusion and/or chromosome transfer. Meiotic cells which have acquired extra elements express various degrees of stability, but can result in fertile gametes and subsequent spontaneous karyotypic modification in the next generation. Further, rapid unidirectional modification of chromosomes from cells of clonal origin suggests that there must be a complex and finely tuned chromosome/cytoplasm equilibrium. Lastly, the fact that partial elimination appears to lead to inherited modified chromosomes shows that the elimination mechanism need not be an all or none reaction, and that additional chromosomes, whether modified or not, having passed a certain threshold, will acquire normal cellular stability. We expect to publish these findings in full this year.

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Location and allelism of golden-2

In MNL 53:24-25, 1979, I reported data showing that golden-2 (g2), which current maps indicated as being on chromosome 7, was not on that chromosome. Subsequently, plants of g2 (source: Coop. 64-169) were crossed by a set of B-A translocations. g2 was found to be uncovered by TB-3Sb, indicating that it lies in the distal portion of the short arm of chromosome 3. Since Beckett (J. Hered. 69:27-36, 1978) reported that TB-3Sb uncovers cl but not rt, and since these two genes are only two map units apart on 3S, it follows that g2 must lie distal to them. Two and three point linkage tests with other genes on this arm are in progress.

Golden-2 has proven to be allelic to mutants of similar phenotype received from others. A stock of pale green mutable (pg-m), later designated pg14, of P. A. Peterson's En system (received from him in 1973) had white culms as well as pale green leaves. Crosses of this stock with g2 indicated allelism. Beckett, Coe and Neuffer (MNL 47:147-148, 1973) found a new golden trait in an a3 Coop. stock. They designated it as g5 and found it to be uncovered by TB-3Sb. Beckett (MNL 52:79, 1978) reported that g5 was allelic to pg14. Crosses of g2 with g5 that segregated out of an a3 stock sent to me by Coe indicated allelism.

As golden-2 was first described in 1926 by M. T. Jenkins (Amer. Nat. 60:484-488), the symbol g2 has priority over pg14 and g5.

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Genetics of horizontal resistance to pests by glucosides and super-genes

Toxics in plants are usually alkaloids, glucosides, resins, and organic acids. Anthocyanins and flavones and related substances appear in glucoside form which is not toxic per se. Stimulated, the glucoside hydrolyzes, producing sugars and aglucones that are toxic. They are phytoalexins in the sense of Mueller. DIMBOA one aglucone confers resistance to Helminthosporium turcicum, Diplodia maydis, Gibberella zeae, Erwinia spp., Rhopalosiphum maydis, Ostrinia nubilalis, atrazine, etc.

Resistance in Zapalote Chico to *Heliothis zea* (corn earworm) is, at least in part, by antibiosis with maysin, a flavone glucoside. The authors (MNL 56:30-32) mapped, with the standard 9 *wx* translocations in IAC Maya latente, some effective factors for resistance to *H. zea* in Z. Chico and IAC Maya. (There is an error in Table 1, where the chi-square of *wx* T4-9b (4S.27; 9L.27) should read 11.46, with $P < 0.01$, instead of 1.116.) It was seen that resistance corresponded chiefly to super-genes and genes responsible for sunlight-independent synthesis of anthocyanins.

Table 1. Tests of cytogenetic mapping with *Ostrinia nubilalis* and *Heliothis zea*, from the literature. S=short arm, L=long arm; L, both arms tested, positive for L, not for S; S, vice-versa; S or L followed by n, not tested; --, both arms tested, results negative; (), resistance in the marker tester; ?, doubt between two arms or $0.10 > P > 0.05$.

Ref.*	Insect	Material	Chromosome										Totals	
			1	2	3	4	5	6	7	8	9	10	S	L
1	<i>O. nubilalis</i>	T x A411	n	--	L	SnL	L	n	--	--	--	--	00/07	02/08
2	"	T x C131A	S	S	--	SL	--	L	--	--	--	Sn	03/09	02/10
	"	T x B49	S	S	--	SL	L?	L	--	L	--	Sn	03/09	04/10
3	"	g17 v17 x M14 + Wf9	n	n	n	S7L7	n	n	n	n	n	n	01/01	01/01
4	"	T x B52	SL	L	S	L	S	--	--	L	--	--	03/10	04/10
5	<i>H. zea</i>	T wx 9 x 245	S	--	L	Ln	S	Sn	Sn	Sn	--	Sn	02/07	01/09
6	"	T wx 9 M14 x 20	S	Ln	SLn	(S)	--	--	--	Sn	--	Ln	03/09	00/07
	"	+ W23 x B1-1	L?	Ln	S7Ln	(L)	--	--	--	Sn	--	Ln	01/09	01/07
	"	+ L317 x 245	--	Ln	Ln	SL	L	--	--	Sn	--	Ln	01/09	02/07
	"	x 259	(S)	Ln	Ln	(L)	(S)	--	--	Sn	--	Ln	02/09	01/07
	"	x 322	(S)	Ln	Ln	--	(L)?	(S)L	--	Sn	--	Ln	02/09	02/07
	"	x L92w	(L)	Ln	Ln	--	(L)	--	--	Sn	--	Ln	00/09	02/07
7	"	Mite T wx 9 x Z.C.	S	S	(L)	S(L)	--	L	--	Sn	--	--	03/09	03/10
	TOTAL	S	07/11	03/12	03/12	06/12	03/12	02/10	00/11	00/04	00/12	00/10	24/106=23%	
	TOTAL	L	03/11	01/06	03/06	09/12	04/12	03/11	00/12	02/12	00/12	00/06	25/100=25%	

*(1) Ibrahim, M.A., 1954, *Agron. J.* 46:293-298; (2) Scott, G.E., Dicke, F.F. & Pesho, G.R., 1966, *Crop Sci.* 444-446; (3) Penny, L.H. & Dicke, F.F., 1957, *Agron. J.* 49:193-196; (4) Onugoku, F.A., Guthrie, W.D., Russell, W.A., Reed, G.L. & Robbins, J.C., 1978, *J. Econ. Entomol.* 71:1-4; (5) Robertson, D. & Walter, E.V., 1963, *J. Heredity* 54:267-272; (6) Widstrom, N.W. & Wiseman, B.R., 1973, *J. Heredity* 64: 83-86; (7) Miranda, L.T. de, Rossetto, C.J., Miranda, L.E.C. de, Sawazaki, E. & Schmidt, N.C., 1981, *MNL* 56:30-32.

We did bibliographic research and analyzed 21 references in three groups. The first (Table 1), for lepidopterans, has 4 references for *O. nubilalis* and 3 for *H. zea*. The second has 4 for "stunt" (spiroplasma) and 1 with atrazine (Table 2). The third, for fungi, has 3 for *H. turcicum*, 2 for *D. maydis*, 3 for *Ustilago zea* and 1 for *Puccinia sorghi* (Table 3). References are listed at the bottom of the tables.

Table 2. Tests of cytogenetic mapping with maize dwarf mosaic, "stunt" (spiroplasma), and atrazine.

Ref.*	Test	Material	Chromosome										Totals	
			1	2	3	4	5	6	7	8	9	10	S	L
8	MDM inf. nat.	T M14 x Oh07	L	L	S	--	--	SL	S	S	--	S7L?	05/10	04/10
	"	x Mo22	L	L	S	--	--	SL	SL	S	--	S7L?	05/10	05/10
9	"	T wx x Mp412	S	Sn	L	S	--	Ln	--	--	--	n	02/08	01/08
10	MDM spray	T M14 x GA209	SnL?	--	L?	LS?	L?	SL	--	--	S?	Ln	03/09	04/09
11	"	T wx x 61s	n	n	n	n	n	SL	n	n	n	n	01/01	01/01
12	Atrazine	T wx 9 x GT112	--	n	Sn	--	L	L?	--	L	--	--	00/08	03/09
	TOTAL	S	01/04	00/03	02/04	02/05	00/05	03/05	03/06	02/05	01/05	02/04	16/46=35%	
	TOTAL	L	02/05	02/04	02/05	01/05	02/05	04/04	02/06	01/05	00/05	02/03	18/47=38%	

*(8) Findley, W.R., Dollinger, E.J., Louie, R. & Knoke, J.K., 1973, *Crop Sci.* 13:608-611; (9) Scott, G.E. & Rosenkranz, E.E., 1977, *Crop Sci.* 17:923-925; (10) Scott, G.E. & Nelson, L.R., 1971, *Crop Sci.* 11:801; (11) Scott, G.E. & Rosenkranz, E.E., 1973, *Crop Sci.* 13:724-725; (12) Scott, G.E. & Grogan, C., 1969, *Crop Sci.* 9:669-670.

Table 3. Tests of cytogenetic mapping with *Helminthosporium turcicum*, *Diplodia maydis*, *Ustilago zea*, and *Puccinia sorghi* (0, data not available).

Ref.*	Fungus	Material	Chromosome										Totals	
			1	2	3	4	5	6	7	8	9	10	S	L
13	<i>H. turcicum</i>	R4 x Mo21A	S	(S)	L	SL	--	--	--	--	--	03/10	02/10	
	"	R4 x NC34	S	--	L	SL	--	--	--	--	--	02/10	02/10	
	"	A188 x Mo21	(S)	(S)	--	L	L	SL	S	--	--	04/10	03/10	
	"	A188 x NC34	L	--	--	--	L	--	S	--	--	02/10	02/10	
14	"	A188 x C190A	L	L	SL	L	L	--	S	--	--	02/10	05/10	
	"	A188 x Mo21A	(S)	(S)L	--	S	L	SL	S	--	--	05/10	03/10	
15	"	Marker genes	SL	Ln	SLn	SL	Sn	L	n	Lsn	SL	SLn	05/07	05/06
16	<i>D. maydis</i>	W22 x Co63	0	S?	0	0	0	(SL)?	L	(SL)?	0	(SL)?	04/05	04/05
17	"	B14 x Os420	S?	S?	SL	--	C	L	C	L	L	--	05/10	06/10
	"	B14 x Hy	L	--	S	--	C	L	C	L	L	--	03/10	06/10
	"	C103 x Os420	--	--	SL	--	C	L	C	L	SL	--	04/10	06/10
	"	C103 x Hy	L	--	--	--	C	L	--	L	L	--	01/10	05/10
18	<i>U. zea</i>		SL	SL	SL	L	Sn	SL	SL7	L	L	L	05/09	09/10
19	"	T x Minn13	--	Sn	--	--	--	S	Sn	Sn	SnL	Ln	01/06	02/09
	"	T x Rustler	--	Sn	L7	S	SL	L7	Sn	SnL	Sn	Ln	02/06	04/09
20	"	Marker genes	--	--	--	IV	--	V1	--	VIII	IX	n	04/09	04/09
21	<i>P. sorghi</i>	(T x Cuzco x susc)	Sn	Ln	--	--	--	L	--	Sn	--	Ln	00/08	01/08
	"	(Oh45 x W92) x T x B14917	Sn	Ln	Sn	SL	SL	Sn	n	--	--	Ln	02/06	02/07
	"	-901	Sn	Ln	Sn	SL	S	SnL	n	--	--	Ln	02/06	02/07
	TOTAL	S	07/15	06/17	06/16	09/18	07/16	06/17	08/14	02/15	03/16	02/08	56/162=35%	
	TOTAL	L	06/18	03/15	07/17	09/18	10/18	13/19	06/16	19/19	08/18	02/12	73/170=43%	

*(13) Jenkins, M.T., Robert, A.L. & Findley, W.R., Jr., 1957, *Agron. J.* 56:197-201; (14) Jenkins, M.T., Robert A.L. & Findley, W.R., Jr., 1961, *Crop Sci.* 1:450-455; (15) Findley, W.R., Jr. & Leffel, R.C., 1962, *Crop Sci.* 2:337-340; (16) Hoffbeck, L.L., 1962, *Microfilm Ph.D. Thesis*, U. of Wisconsin Library, Madison; (17) El-Rouby, M.M. & Russell, W.A., 1966, *Can. J. Genet. Cytol.* 8:233-240; (18) Burnham, C.R. & Cartledge, J.L., 1939, *Amer. Soc. Agron.* 31:924-933; (19) Saboe, L.C. & Hayes, H.K., 1941, *J. Amer. Soc. Agron.* 33:463-470; (20) Hoover, M.M., 1932, *Agric. Exp. Sta. Bul. W.Va.U.* 253:1-32; (21) Russell, W.A. & Hooker, A.L., 1962, *Crop Sci.* 2:477-479.

By the number of arms tested the expected frequency for random results was calculated and a X^2 test of independence was applied. This was done for short arm, long arm, and one or another, by chromosome.

Our interpretation of how to classify the data is subject to criticism. However, we point out that A. C. Waiss et al. (*Bull. Entomol. Soc. Am.* 27:217-221, 1981) reported that the maysin content varies tremendously (up to 20-fold) within a corn line, and the cross between corn varieties regardless of maysin content often contained higher levels of maysin in the F1 than either of the parents. These observations can explain many non-repetitive results, and reinterpretations. G. E. Scott and C. O. Grogan (12) reported that only 8L was associated with resistance to atrazine, but applying a contingency X^2 test to their data, which they did not apply, two more, 5L and 6L, were found. W. A. Russell and A. L. Hooker (21) did not consider 6L as associated, in part because it was not significant with 9c, but in combination with 4L, the X^2 are three times greater than with any other arm combinations in their results, being, probably, complementary dominants.

We have probably misclassified some entries, but expect that this should not impair general conclusions, although the accuracy could be improved. In Tables 4 and 5 the statistical analyses are presented.

The mean X^2 for lepidopterans was significantly greater than for fungi: X^2 divided by $X^2 = F = 4.67$ to 11.95 with $P < 0.05$ to $P < 0.01$. Four arms and 5 whole chromosomes were significant for insects, and only 1 arm and 1 chromosome for fungi. In total, for insects, 1S***, 1**, 4S**, 4L*** and 4*** were associated for resistance; 7L*, 7**, 9L*, 9** and 10* were not associated.

For "stunt" + atrazine (MDM+A), 6L** and 6** were associated with resistance and 9L and 9 tended not to be associated. For fungi, 6L* was associated and 10S* wasn't. For the total of the three groups there was association for 1S*, 4S*, 4L*, 4***, 6L** and 6*. There was no association for 9S*, 9**, 10S*, 10**. Only 3L and 2S, as expected, did not appear as significantly associated in any group or total.

Table 4. Statistical analysis of tables 1, 2 and 3. First column chromosome and arm or the entire chromosome (I) second to sixth columns. Chi-square for insects, MDM+A, fungi, and interaction (P=0.10*; P=0.05**; P=0.01***).

	Insects	MDM+A	Fungi	Total	Interaction
1S	+8.17***	-0.11	+0.63	+3.70*	5.21*
1L	+0.02	+0.01	-0.39	-0.17	0.25
1I	+4.35**	-0.03	+0.00	+0.00	3.44
2S	+0.03	-1.04	+0.00	-0.06	1.01
2L	-0.17	+0.14	-1.84	-1.08	1.07
2I	-0.02	-0.12	-0.95	-0.90	0.19
3S	+0.03	+0.27	+0.04	+0.15	0.19
3L	+1.50	+0.01	-0.01	+0.30	1.22
3I	+0.69	+0.15	+0.00	+0.40	0.44
4S	+3.69**	+0.04	+1.24	+3.71*	1.53
4L	+12.00***	-0.43	+0.21	+2.99*	9.65***
4I	+15.11***	-0.12	+1.15	+6.62***	9.76***
5S	+0.03	-1.74	+0.39	-0.00	2.16
5L	+0.33	+0.01	+0.67	+0.79	0.22
5I	+0.29	-0.75	+1.09	+0.43	1.70
6S	+0.03	+0.91	+0.00	+0.15	0.79
6L	+0.02	+3.99**	+2.87*	+4.59*	2.29
6I	0.00	+4.18**	+1.79	+3.52*	2.45
7S	-2.49	+0.40	+2.07	+0.24	4.72
7L	-3.00*	-0.04	-0.11	-1.58	1.57
7I	-5.47**	-0.08	+0.47	-0.37	5.65*
8S	-0.91	+0.04	-1.96	-1.52	1.39
8L	-0.33	-0.43	+0.09	+0.00	0.85
8I	-0.86	-0.12	+0.37	-0.86	0.49
9S	-2.72*	-0.31	-1.16	-3.68*	0.51
9L	-3.00*	-1.91	+0.01	-1.81	3.11
9I	-5.71**	-1.93	-0.37	-5.15**	2.86
10S	-2.26	+0.27	-2.86*	-3.42*	1.97
10L	-1.50	+0.63	-1.93	-1.76	2.31
10I	-3.81*	+0.81	-1.83	-5.40**	1.05
TOTAL S	20.63**	5.13	10.35	16.63*	19.48**
L	21.87**	7.60	8.13	15.06	22.53**
I	36.31***	8.29	8.02	24.59***	28.04***

Table 5. With the totals in the three last lines of table 4, mean χ^2 and test of F using mean χ^2 for fungi in the denominator. ($P=0.10^*$; 0.05^{**} ; 0.01^{***} . Degrees of freedom 10 and 10).

		Insects	MDM+A	Fungi	Total
No. significant items/total	S	24/106	16/46	56/162	96/314
	L	25/100	18/47	73/170	116/317
	I	49/206	34/93	129/332	212/631
Mean χ^2 for significant items	S	0.859	0.321	0.184	0.173
	L	0.875	0.422	0.111	0.130
	I	0.741	0.243	0.062	0.116
	F	4.67**	1.74	1.00	0.94
	F	7.88***	3.80**	1.00	1.17
	F	11.95***	3.92**	1.00	1.35
Mean χ^2 , number tested	S	0.195	0.112	0.064	0.099
	L	0.219	0.162	0.048	0.048
	I	0.176	0.089	0.024	0.020
	F	3.05**	1.75	1.00	1.54
	F	4.56**	3.37**	1.00	1.00
	F	7.33***	3.70**	1.00	0.67

The more probable cytogenetic locations of the resistance components to pests by glucosides are 6L, Dt2 P1 Bh sm (Dt2 is mutagenic of a to A, and Bh produces aleurone color even with c1 c1); 4L, Tu c2; 4S, Rp4; 3L, a3 a1; 2S, R2? rp7?; 1S, p. We reformulate our earlier suggestions in the sense that Zer1 should be in 4S around Rp4. There are clearly two effective factors with loose linkage in chromosome four. Other reformulation is that Zer3 could be nearer rp7 than to R2.

Resistance of plants to insects seems to depend much more on this system than resistance to diseases, that although present, appear blurred by other mechanisms.

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Giemsa banding of diploid perennial teosinte and its hybrids with maize

Since about 10 years ago, Giemsa banding procedure has been developed and successfully employed in karyotype analysis of various organisms. The advantage of this technique lies in its characterization and localization of heterochromatins in the somatic chromosomes and the interphase nuclei through DNA denaturation and reannealing procedures. These heterochromatins are usually not visible through the standard aceto-carmin staining technique.

Diploid perennial teosinte (*Zea diploperennis*) has aroused a great deal of interest among plant scientists. However, its mitotic chromosomes have not yet been examined with the Giemsa procedure. This report deals with the results of our most recent experiments on the recognition of mitotic chromosomes of diploid perennial teosinte and its hybrids with maize with the above technique.

At the premetaphase stage of root-tip cells, distinct terminal bands, varying in size, appeared in eight of the 10 chromosome pairs. The short arms of chromosomes 1 and 2 had a small band, while the long arms of chromosomes 3, 4, 6, 7, 8 and 9 had a medium-sized band. Most of the bands were homozygous. However, chromosomes 5 and 10 were without any visible bands. The nucleolar organizer regions (NOR) of chromosome 6 were consistently differentiated and were densely

stained. The satellites of these chromosomes were also darkly differentiated and frequently folded back on the NORs.

Mitotic chromosomes of the root-tip cells from F1 hybrids of the diploid perennial teosinte and an inbred maize, Zhi-35 from China, were stained using the same procedure as used for the teosinte parent. It was observed that recognizable terminal bands of the eight chromosomes of the teosinte parent reappeared. In addition, three intercalary bands on the long arms of chromosomes 4, 6 and 7 were identified. They came from the maize parental plant. The difference between the bands of the two parental species was very distinct.

The recognition of the parental chromosomes in the root-tip mitoses of the F1 hybrids between diploid perennial teosinte and maize was further investigated and confirmed by crossing the same teosinte with two other maize inbreds, Qi-330 and Huang-tzo-4, both from China. It was consistently found that the bands corresponded in shape, number and size with the knobs appearing on the pachytene chromosomes stained with conventional techniques. Furthermore, nucleolar organizer regions were persistently stained using the Giemsa banding procedure. This differs from the results reported by Ward on the Giemsa C-banding patterns of two varieties of maize (1980, *Can. J. Genet. Cytol.*). He could not stain the NORs of chromosome 6. This discrepancy could be accounted for by variations of technique and stage of mitosis.

In the past, knob counts have been used as reliable cytological markers to relate races of maize and teosinte. Because of the lack of a suitable technique to recognize knobs in the mitotic chromosomes and because of the small size of these chromosomes, the counts were mainly made on the basis of pachytene chromosomes. Nevertheless, due to the difficulty in distinguishing homozygous knobs from heterozygous ones, the results of the knob counts were often controversial and hard to confirm. From now on, it appears feasible that knob counts of maize and teosinte be done with Giemsa banding technique because the bands correspond exactly with the knobs. Thus it may better facilitate our understanding of the somatic chromosome characteristics of both maize and teosinte. It may also better reveal the relationships not only between these two species, but among the various races within each species as well. Therefore, it is conceivable that Giemsa banding has indeed great potential in both the study of variations of plant karyotypes and genetic research in general.

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Further studies on maize anther culture in vitro

Pollen-Plants: In the spring of 1981, more than 100 pollen plants of maize were obtained. Among them, 36 survived transplantation and grew vigorously in the summer field plot. As soon as chromosome numbers of the root-tip cells were checked, it was found that 21 of the plants were diploid ($2n = 20$), and the rest haploid ($n = 10$). Three of the diploid plants were self-fertilized and had approximately 100 percent seedset. The progenies of these plants were vigorous and uniform.

Anthers of some of the above H1 progenies were also cultured in vitro on zheng-14 medium. As expected, their response was highly favorable.

In the last summer, 304 maize pollen-plants were obtained. After transplantation to the greenhouse, 45 of them survived. They grew vigorously; 43 of them bore only pistillate inflorescence, while two of them bore both pistillate and staminate inflorescences. When the pollen fertility was examined it was observed to be totally sterile. It appeared that all of these 45 plants were haploid. Spontaneous doubling of chromosome numbers did not occur in them.

Evidence of Genetic Control: Anthers of maize Dan-San 91 had responded favorably to in vitro culturing in the past. In order to investigate the inheritance of this property, progenies from the self-fertilized as well as the cross-fertilized plants were grown in the summer of 1982. Anthers of these progenies were again cultured on the same medium. It was observed that 10.3 percent of them from both selfed and crossed progenies differentiated into either calli or embryoids. This again suggests that genotype plays an important role in maize anther culture response.

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Maize DNA minipreps

In order to exploit molecular cloning strategies in genetic studies it is often necessary to compare DNA samples obtained from a large number of individuals. Rapid screening methods have been applied to a variety of microorganisms to follow changes in DNA sequence organization and the insertion of foreign DNA. Isolation of plant DNA is complicated by the presence of a tough cell wall and large amounts of polysaccharides, phenolics, and tannins. Most existing large scale (e.g., Kislev and Rubenstein, *Plant Physiol.* 66:1140-1143, 1980; Murray and Thompson, *Nucl. Acids Res.* 8:4321-4325, 1980) and microscale (e.g., Zimmer and Newton, In "Maize for Biological Research," ed. W. F. Sheridan, pp. 165-168; Taylor and Powell, *Focus* 4(3):4-6) plant extraction procedures, therefore, rely on the isolation of nuclei and phenol extraction or preferential precipitation of DNA followed by equilibrium density centrifugation in CsCl. Such complex and cumbersome techniques are inappropriate for application to a large number of samples and small amounts of tissue.

We report here a plant DNA isolation procedure adapted from one commonly used on yeast (Davis et al., *Methods in Enzymology* 65:404-411, 1980), which requires 1.0 gm or less of plant leaf tissue and does not rely on isolation of nuclei, phenol extraction or CsCl gradient centrifugation. Partially purified DNA suitable for restriction endonuclease digestion can be obtained from twenty or more samples in only a few hours. Each prep (from 1.0 gram of maize tissue) yields approximately 50 micrograms of total cell DNA, enough for at least ten Southern blots.

An important application of this miniprep procedure in the genetic manipulation of maize is that leaf material may be harvested from seedlings at the 2-3 leaf stage without sacrificing the plant. Information obtained by Southern blotting or other genomic analysis is thus available long before the plants are sexually mature, and can be used in planning crosses involving those plants. For example, we have identified two normal Sh1 isoalleles, in addition to the sh1 tester allele, all of which can be distinguished from one another on the basis of BglII restriction site polymorphisms. By Southern blotting of DNA minipreps, genotypic identification can be made before pollinations are performed. Another instance where minipreps can be used to permit early genetic identification is in a cross such as Sh/Sh x sh/sh; minipreps can be used to screen Sh kernels in the F2 and immediately distinguish homozygous and heterozygous individuals without subsequent crosses. Using the same restriction site markers, we have also found the miniprep procedure useful to screen a number of greenhouse-grown seedlings for a particular genotype, thereby relieving the need to maintain the unwanted plants for more than a few weeks.

Figure 1 shows a case in which miniprep DNA blots were used to distinguish a standard sh1 tester allele (sh-t) from a newly arisen mutant Sh1 allele (sh*) (Mottinger et al., manuscript in preparation) in the progeny of a selfed sh-t/sh* plant. The DNA was digested with BglII restriction endonuclease, and the blot was

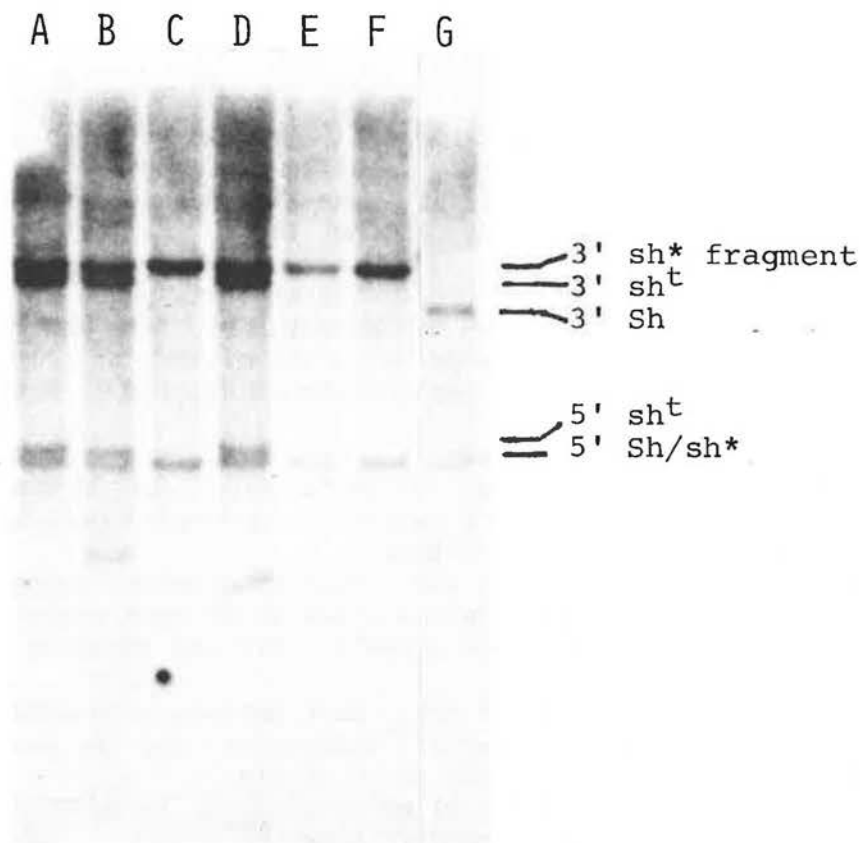


Figure 1. Southern blot hybridization of miniprep DNA from three-week seedlings digested with BglII and probed with ^{32}P -labelled Sh cDNA. BglII cuts within the structural gene, conveniently dividing each allele into characteristic 5' and 3' fragments which hybridize to the cDNA probe. Lanes a through f represent progeny obtained by selfing a plant heterozygous for the sh-t allele and the Sh1 insertion mutation (sh*). Lane g contains DNA from a plant homozygous for the normal Sh1 allele in which the sh* mutation occurred within the 3' region. Lane: (a) sh*/sh-t; (b) sh*/sh-t; (c) sh*/sh*; (d) sh*/sh-t; (e) sh*/sh*; (f) sh*/sh*; (g) Sh/Sh.

probed with the sucrose synthetase cDNA clone obtained from Nina Fedoroff. The sh* mutation is the result of an insertion of a DNA element within the coding sequence (Dellaporta et al., manuscript in preparation) rendering the gene defective. This causes the 3' BglII Sh1 fragment to increase in size by 1.1 kb (compare lane F with

lane G). All of these individuals will be shrunken and therefore phenotypically indistinguishable, but the blot clearly shows that the seedlings represented by tracks A, B, and D are heterozygous for the two alleles, while those in tracks C, E, and F are homozygous for, in this case, the insertion mutant.

The degree to which the procedures described above will be generally applicable depends, of course, on the availability of appropriate restriction fragment polymorphisms. Our experience with the *Sh1* region, however, indicates that such markers are common in the maize stocks now in use.

Miniprep Procedure

1. Weigh 1 gm of leaf tissue, quick freeze in liquid nitrogen and grind to a powder in a 3" mortar and pestle. Transfer powder with liquid nitrogen into a 30 ml Oak Ridge tube. It is imperative not to let the tissue thaw once frozen until buffer is added and not to cap the tubes while nitrogen is evaporating.
2. Add 15 ml of Extraction Buffer (EB): 100 mM Tris, pH 8; 50 mM EDTA, pH 8; 100 mM NaCl; 1% SDS; 10 mM mercaptoethanol. For maximum DNA yields, the cells are further broken by grinding the mixture at a low setting (about 3) with a Polytron (Brinkmann Instruments, Inc.), however, this step can be optional.
3. Incubate tubes at 65 C for 10 min.
4. Add 5.0 ml 5M potassium acetate. Incubate at 0 C for 20 min. Most proteins and polysaccharides are removed as a complex with the insoluble potassium dodecyl sulfate precipitate.
5. Spin tubes at 25,000 x g for 20 min. Pour supernatant through a Miracloth filter (Calbiochem) into a clean 30 ml tube containing 10 ml isopropanol and 1 ml 5M ammonium acetate. Mix and incubate tubes at -20 C for 20 min.
6. Pellet DNA at 20,000 x g for 15 min. Wash pellets with cold 70% ethanol and respin. Gently pour off supernatant and dry pellets by inverting tubes onto paper towels for 5-10 min.
7. Redissolve DNA pellets with 0.7 ml of 50 mM Tris, 10 mM EDTA, pH 8. Transfer the solution to an Eppendorf tube.
8. Add 75 ul 3M sodium acetate and 500 ul isopropanol. Mix well and pellet the clot of DNA for 30 sec in a microfuge. Wash pellet with 70% ethanol, dry, and redissolve in 100 ul 10 mM Tris, 1 mM EDTA, pH8. Precipitation from 0.3M sodium acetate using relatively small amounts of isopropanol (about 0.6 volumes) has been reported to separate high molecular DNA from polysaccharides (Marmur, J. Mol. Biol. 3:208-218, 1961). The sodium acetate also yields a tight, fibrous precipitate that is easily washed and dried. The DNA will dissolve readily if allowed to rehydrate at 4 C for one hour, followed by light vortexing.

Minipreps can be stored for several months without evidence of degradation and can be cut with a variety of restriction enzymes and ligated without further purification. We find that 7.0 ul of miniprep DNA is sufficient for a single 8 mm lane in an agarose gel which is to be used for filter hybridization with single-copy probes. Heat-treated RNAase must be added to the restriction reaction to digest contaminating RNA in each prep. Hence, a typical reaction would contain the following:

Miniprep DNA	7.0 ul
10X Restriction Buffer	2.5 ul
0.5 mg/ml RNAase	2.0 ul
Eco RI	8 units
dH ₂ O	to 25 ul

Digestion is usually complete after 3 hours at 37 C. Occasionally, minipreps are difficult to digest with certain enzymes. This problem can be overcome by adding 5.0 ul of 0.1M spermidine to the entire miniprep before digestion (see Focus 4(3):12, 1982). For lambda genomic library construction, we have found the packaging efficiencies are higher if the minipreps are further purified by CsCl-ethidium bromide gradient centrifugation. This can conveniently be done by pooling 2-5 identical minipreps per gradient.

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Ds at the shrunken locus

We have continued our study of clones obtained from genomic DNA of mutants sh-m5933 and sh-m6233. The following results have been obtained:

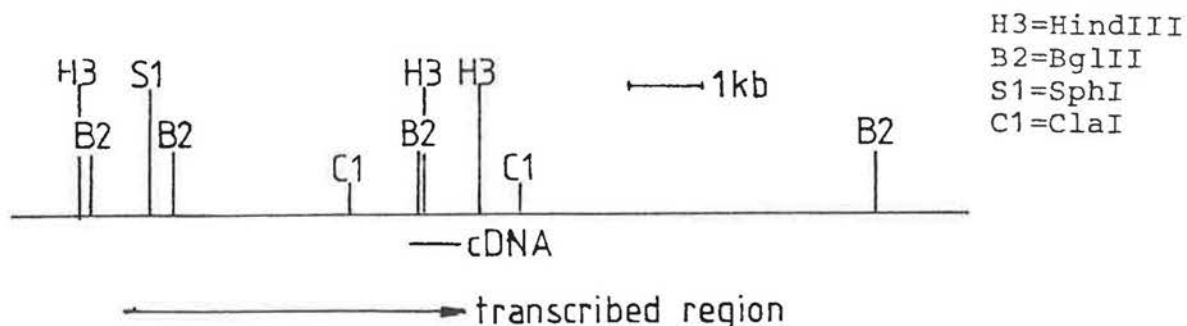
1. Both clones contain one segment homologous by hybridization and restriction analysis to a corresponding region in the clone derived from Sh DNA. This segment extends to a certain point (breakpoint). Beyond this point, the DNA extending to the end of the insert in the lambda phage does not show hybridization to the wild type-derived clone.
2. The breakpoints are located 2.5 kb apart. The breakpoint in sh-m5933 seems to be located in an intron. The breakpoint in sh-m6233 is located upstream and may be located outside the transcription unit.
3. The "foreign" DNAs adjacent to the breakpoints hybridize to each other, but differ in restriction patterns.
4. On sh-m5933 "foreign" DNA, two pairs of inverted repeats are present. These interdigitate with each other. One member of one pair terminates exactly at the breakpoint.
5. DNA sequence analysis of the repeat bordering at the junction in sh-m5933 detects a region which contains repeats of the hexanucleotide CCGTTT and derivatives thereof. These are oriented either directly or inverted to each other.
6. The DNA bordering directly at the junction has been subcloned and used as a probe for hybridization to genomic DNA. Up to 40 bands are revealed. The structure of these bands is heterogeneous.
7. If the DNA near the junction is Ds DNA, several non-identical copies of this element are present within genomic DNA of several maize lines. These show difference in restriction patterns.

Addendum: In the last issue of MNL, we reported the isolation of an unstable Adh1 mutant, which we assumed to be caused by a controlling element different from the Ds-Ac system. Further genetic analysis of the mutant, however, showed that the instability of the Adh1 gene expression is linked to the presence of an Ac element.

U. Courage-Tebbe, H.-P. Döring, P. Starlinger, E. Tillmann and E. Weck

Structure of the sucrose synthase gene of chromosome 9

We have further analyzed our genomic sucrose synthase clone by S1-mapping experiments and partial DNA sequencing. A partial restriction map is shown in the figure.



By using terminally labeled DNA subfragments, the direction of transcription was determined from the left to the right with respect to the above restriction map. The extension of the gene is at least 4.5 kb, exceeding the size of mature mRNA of 2.8 kb. The difference is due to the fact of many small introns. By S1-digestion of RNA protected DNA, electrophoretic separation and subsequent hybridization to radioactive subfragments, the number of introns was determined to be 14, if all the reproducibly found fragments correspond to exons and are not the product of incorrect splicing.

The position of two introns near the central BglIII site on the above map was verified by sequencing of genomic and cDNA. The leftmost S1-resistant DNA hybridizes to the DNA fragment between the leftmost BglIII and the SphI site. It may correspond to the transcription start. Otherwise, a very small leader sequence may be located further upstream and have escaped detection.

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

COLUMBIA, MISSOURI
University of Missouri

Chimeral dominants in the M1 from an EMS treatment

A large M1 from treatment of Mo17 pollen with EMS and crossing on A632 silks was planted and observed for dominant mutants. The method of treatment was that described in MNL 56:42. The purpose was to produce mutants for earliness, male sterility, short plants, and other agronomically useful traits. 7,997 kernels were planted, producing 6,425 seedlings. The M1 was notable for its high frequency of apparent genetic changes, including maternal and paternal haploids, monosomics, and dominant mutants. Among these last were 7 shredded leaves, 2 golden sheaths, 21 yellow-greens (including sectored forms, below), 4 with tillers, 3 male steriles, 5 with pubescent leaf sheaths, 35 dwarfs or very short plants, 21 with leaf lesions or leaf texture changes and a number of other miscellaneous traits.

A curious pattern was observed for 54 plants--one-half the plant (divided by a plane through the leaf midribs) was phenotypically different from the other half. Lesions, striping, inter-vein narrowing, shredding, rapid aging, and other traits could be restricted to 1/2, 3/4 or 1/4 of the leaf. Often the midrib limited the affected area and sometimes there was only a narrow stripe, but for all cases, both ranks of leaves were affected, in one rank the "left" half of the leaf, in the other the "right" half. Traits restricted to one rank were not observed. We

decided to use Edgar Anderson's terminology (*The Corn Plant of Today*, p. 17) to distinguish the two plant halves: front and back, with the front being that half of the plant with the outer sheath-overlaps (Fig. 1). About a third of the dominant mutations affecting leaves were expressed in such wide sectors (a careful count depends on observing the next generation).

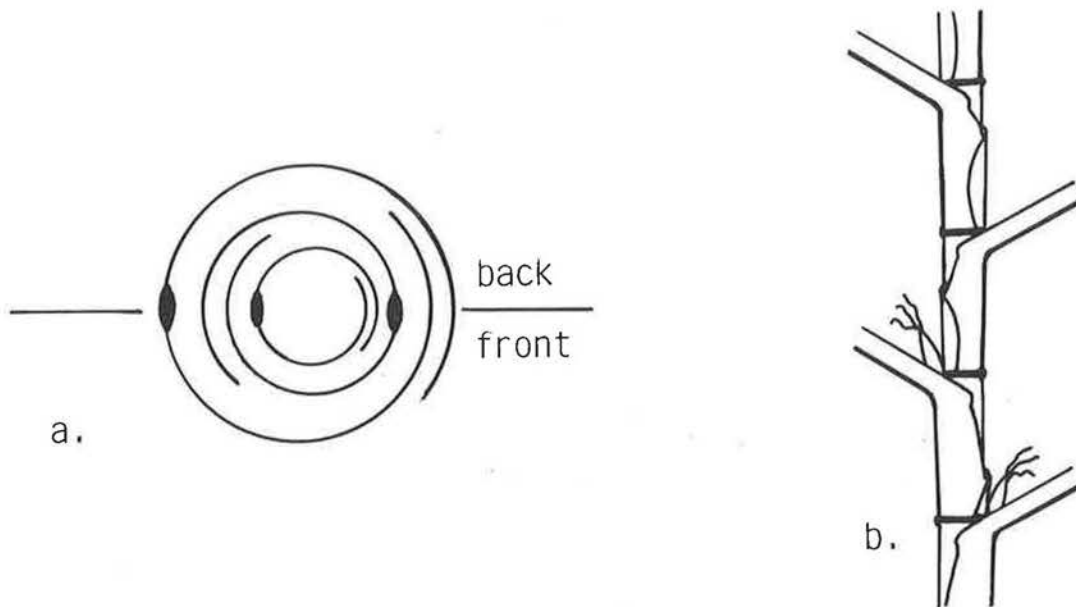


Fig. 1. Maize plant front/back differentiation: (a) cross-sections of three sheaths; (b) view of front of plant (note that the appearance of the overlapping changes at the upper ear node).

It is hard to explain this unless it reflects a mutational change of one-half of a DNA double-helix in the pollen grain gametic nucleus leading to genotypic difference between the first two embryonic cells. Often these two cells would contribute equally to the plant, but one cell could contribute to only a quarter or could become the entire plant. This, however, does not coincide with L. F. Randolph's description of maize embryogeny (1936, *J. Agr. Res.* 53:881).

All plants were self-pollinated, if possible, and those thought to be dominant mutations at the time of pollination were crossed onto Mo17, if available, or onto another stock. Ca. 4,000 relatively full ears and 1,200 semi-sterile or poorly pollinated ears are being scored for any observable differences from untreated material. Relatively few are fully or even nearly normal, perhaps 10%. A wide range of familiar and unfamiliar mutations or aberrations are being catalogued.

Robert McK. Bird and M. G. Neuffer

Mapping of *dv* and *e1*

dv/dv plants were crossed by a portion of the B-A translocation series. The various progeny families were grown and enriched for hypoploids and, subsequently, sporocyte samples were taken. The following chromosome arms can probably be excluded from further consideration since no sporocytes with a divergent spindle phenotype were seen:

arm 1S	TB-1Sb	9 samples taken
arm 5S	TB-1La-5S8041	37 samples taken
arm 5L	TB-5La	26 samples taken
arm 6S	TB-6Sa	70 samples taken
arm 7L	TB-7Lb	33 samples taken
arm 9S	TB-9Sd	19 samples taken

Work involving the remaining arms is continuing.

Families segregating e1/e1 plants were also crossed by some of the B-A translocations. The progeny from these crosses were grown and sporocyte samples were taken. Based upon cytological examination of sporocytes, tentative placement of e1 (elongate) on the long arm of chromosome 8 can be made. Two separate crosses of TB-8Lc onto plants in families segregating e1/e1 were obtained. Among the progeny of the first cross, a hypoploid (determined cytologically) appeared phenotypically e1. A total of twelve sporocytes were examined from this cross; those which were phenotypically E1 were not further analyzed. In the second cross, one hypoploid among the eight sporocyte samples taken also showed an e1 phenotype.

One must remember that the e1 phenotype is extremely variable in expression. e1/e1 segregants in families can be difficult to discern since they may show no more pollen abortion than their normal sibs. In other backgrounds or under different environmental conditions, e1/e1 plants may be nearly male-sterile. In the above two 8L hypoploids, early anaphases appeared normal and, as the segregation of chromosomes continued, the e1 phenotype became apparent.

The cross of e1/e1 plants by TB-8Lc will be made again to confirm these preliminary results. Meanwhile, an allelism test between e1 and ms8, which is located on 8L, will be made and a linkage study of e1 and several 8L markers will be started.

Chris Curtis

Modified root tip squash technique

A root tip squash technique for maize mitotic chromosome spreads was recently published (P. J. Sallee, in Sheridan, ed., *Maize for Biological Research*, p. 119, 1982). Our protocol, based on Sallee's method, gives a higher frequency of countable figures, primarily because cycloheximide greatly shortens prophase chromosomes. Cycloheximide also allows some visualization of the heterochromatic and knobbed regions of the chromosomes (J. Tlaskal, *Stain Tech.* 54:313-320, 1979).

Procedure:

1. Germinate, in a 30 C incubator, fungicide-treated kernels in a drainable, nearly covered container of moist, coarse sand until roots are 3-4 cm long. Germination of small or old seed is better in sand than in petri dishes (C. R. Burnham, *MNL* 49:122, 1975), and metaphases are more frequent in sand-grown root tips from all classes of seed. We use window screen to remove fine grains from river sand. After a few plantings, the sand should be sterilized or replaced.
2. Root tips (1 cm long) are collected in glass vials and prefixed in the following solution for 4½ hours at room temperature: Thoroughly dissolve 7 mg cycloheximide in 100 ml tap water. Warming the water will help dissolve the cycloheximide. Add 4 drops of monobromonaphthalene and mix vigorously by squirting back and forth with an eyedropper. Add one drop of dimethyl sulfoxide (DMSO) and mix again. It is important to add the chemicals in the order specified. It is best

to have two labeled eyedroppers, one for monobromonaphthalene and the other for DMSO. For consistent results, use these eyedroppers each time the mixture is prepared. All glass materials can be cleaned by rinsing in acetic acid and water.

3. Pour off the prefixative and replace with glacial acetic acid, cap the vial, and let stand overnight at room temperature. Fixation in acetic acid can be reduced to 1 hour, if necessary.
4. To hydrolyze the tips, pour off the acetic acid and replace with hot (60 C) 1N HCl and incubate at 60 C for 5-10 minutes.
5. Pour off all HCl, rinse in distilled water, and stain in basic fuchsin for 10-15 minutes or until tips become bright purple.
6. To prepare a squash, cut off about 1 mm of the tip of the stained root, place on a slide, and add a drop of propionic orcein. Place a plastic cover slip adjacent to the pool of stain, then cover the root tip with a second plastic cover slip so that one edge rests on the edge of the first slip. Tap the second slip many times with a pencil or dull needle to break up the tip and disperse the cells, then slide the first cover slip out from under the second cover slip and blot excess stain. Place slide between two pieces of folded filter paper and apply moderate thumb pressure to flatten the cells. This squash method, which eliminates the need to treat root tips with an enzyme, is commonly used by wheat cytogeneticists here and abroad.

If it is necessary to keep root tips for more than two days, complete Step 5, decant the basic fuchsin solution, add distilled water, cap and refrigerate. Both the distilled water and root will turn purple, but the root tip will remain dark purple. We have noted no deterioration after three weeks of storage, and R. O. Flagg reported (Stain Tech. 36:95-97, 1961) that root tips can be safely stored in the refrigerator for eight months. The usually recommended storage in 70% ethyl alcohol after Step 3 gives, in our experience, blurred, poorly stained chromosomes.

B. K. Kindiger and J. B. Beckett

Classification of red vs. white cob by tassel color

Red vs. white cob color (P-WR vs. P-WW) is expressed in other tissues besides the glumes of the cob. Brick-red color in dry husks is associated with cob color and can be used for classification without harvesting. Association of brown color in the tassel at flowering time has been checked during the past summer. The following F₂ progenies, segregating for P-WR/P-WW, were scored for color in the tassel at flowering time, and for cob color at harvest:

P-WR Source	P-WW Source	Tassel/Cob Classifications				
		Red /Red	Red /White	White? /Red	White? /White	White /White
W23 R-nj	Stock 6	16			3	1
W23 R-nj	Stock 6 C-I	2			1	3
W23 R-sc	Stock 6	7				2
W23 R-sc	Stock 6 C-I	9			2	1
W23 R-nj	A619	10		2*		3
W22	K55	18				4
W23	Mo20W	38	2	1	6	17

*pale cob

The separation is good but not perfect; perhaps a more careful examination of the tassels would be needed, or the expression is marginal. The brown color in the tassel is the faint, yellow brown that is familiarly seen in tassel glumes, especially in the hyaline margins of the glumes.

Undoubtedly others have noticed this correlation, but a few inquiries suggest that it is not widely recognized or used. It should be helpful to be able to distinguish red-cob from white-cob individuals at flowering time, either for genetic purposes or for breeding purposes.

E. H. Coe

Evidence bearing on the orientation of the first division of the zygote

Two experiments that we have conducted provide information about the first division of the zygote. The results of both are consistent with the morphological work of L. F. Randolph (J. Agr. Res. 53:881, 1936), and the conclusions of D. M. Steffensen (AJB 55:354, 1968) from x-ray induced events, that the first division is cross-planar (separating the embryo proper from the suspensor) rather than longitudinal. They make the observations of sectoring in plants from EMS-treated pollen (see article by Bird and Neuffer in this issue) difficult to explain.

In the first experiment, developing seeds from the cross b a pl R-r x B A Pl R-g were x-rayed 21-33 hours after pollination. Plants grown from these seeds were observed for losses of the markers (see table). Among 571 plants, 31 losses affected the whole plant and only 3 losses were fractional. The first division would be expected to be taking place at about the time of the irradiations in this

Hr	Seeds	Plants	Loss*								
			B	B?	A	A?	A sect.	Pl	Pl?	Pl sect.	
21	414	172	3		4						
23	225	100	1		1	1	1/4,1/64	1	1		
25	338	118	7	1	2			3			
29	132	45	1								1/2 varieg.
31	232	122			1			2	1		
33	48	14				1					
T	1389	571	12	1	8	2	1/4,1/64	6	2		1/2 varieg.

*B green, shortened plant; stubby ear, diminutive tassel
 B? faint purple color in near-normal plant
 A brown, diminutive plant; "thin" tassel
 A? brown, near-normal plant
 A sect. sectoried brown, in the fraction indicated
 Pl sun-red, diminutive plant; "pointed" tassel branches
 Pl? sun-red (?), near-normal plant
 Pl sect. sectoried (variegated purple & sun-red) in half the plant

experiment, according to Randolph's data, and divisions that were longitudinal would yield fractional individuals. Even if the 3 fractional events represent longitudinal divisions rather than delayed losses, they are infrequent. As an aside, the morphologies of the hemizygotes were variable, but each arm appeared to have a characteristic effect on plant form.

In the second experiment, pollen of B Pl Wd R-r was exposed to ultraviolet light and crossed onto b pl wd Ring-Wd R-g ear parents. Among 465 plants none were sectorial, while 9 were whole-plant exceptions (3 Wd losses, not validated for other markers before death; 2 B losses, validated; 1 pl loss, not validated; and 3 R-r losses, validated). L. J. Stadler (Proc. VII Int. Cong. Genetics, p. 269, 1939) has shown that UV induces fractional events in the endosperm, while x-rays do so only rarely. If the first division, separating strands with UV lesions from those without, were longitudinal, sectorial plants would have been expected.

E. H. Coe and R. S. Poethig

B and P1 are expressed in the internal tissue of the culm

Plants of the genotype A Bz1 Bz2 C2 R-r B/b P1/p1 have been used extensively for clonal analysis because x-ray-induced sectors resulting from the loss of B or P1 can be observed in every vegetative organ of the plant. Vegetative tissue carrying B and P1 is purple; tissue lacking B is green and tissue lacking P1 is sun-red. Until recently the pigmentation in B P1 plants was thought to be restricted to the epidermis (except in the sheath), implying that data from clonal analyses were relevant only to the behavior of this tissue layer. It now appears that this is not the case. Freehand sections of irradiated and unirradiated B/b P1/p1 plants demonstrate the presence of anthocyanin not only in the epidermis of the culm, but in internal tissue as well. All the cells in the peripheral millimeter of the culm are intensely pigmented; throughout the rest of the culm pigment is usually restricted to vascular bundles. The pigmentation of vascular bundles is most intense within about 1 cm of the node and becomes progressively weaker--and may disappear in some cases--toward the base of the internode.

Because of the intense pigmentation of subepidermal cells, b and p1 sectors are virtually invisible when they are restricted to the epidermis. Only sectors in the subepidermal tissue of the culm are distinct enough to be readily observable. Although sectors induced at a dry seed stage frequently encompass both the epidermis and subepidermal tissue, these tissues are not necessarily clonally related because sectors restricted to one or the other layer also occur. Thus, the pattern of cell division in the epidermis of the shoot meristem is somewhat variable. Sometimes epidermal cells divide anticlinally, and produce only a single layer of cells; however, they can also divide periclinally and contribute to internal tissue.

R. S. Poethig

Cg and Tp2 are gain-of-function mutations

The nature of a mutation can often be deduced by varying the dosage of the normal allele of the gene. A mutation that reduces the level of a gene product (i.e., a hypomorphic or null mutation) should, at least in theory, be phenotypically corrected by a duplication of the normal allele, whereas the phenotype of a gain-of-function mutation will either be accentuated or only partially corrected by a duplication.

In order to create plants carrying duplications of the normal alleles of Cg and Tp2, stocks homozygous for these mutations were crossed as female by B-A translocation stocks. The Tp2 stock used in this study was homozygous for g and r-g and was crossed by TB-10La and TB-10L19 G R-scm stocks. Hyperploids were identified as kernels having a colored embryo and colorless endosperm; as expected, all such kernels gave G seedlings. Because Cg was not linked to a recessive marker, hyperploids from the cross Cg x TB-3Sb were identified by chromosome number. In both instances hyperploids were clearly mutant in phenotype. In fact, none of the progeny from these crosses were completely normal. This result strongly suggests that Cg and Tp2 are not hypomorphic or null mutations, and therefore must involve a gain of function. Whether they are responsible for the overproduction of a normal gene product or the production of an antimorphic product will be examined by reversion studies, and by a more comprehensive dosage analysis.

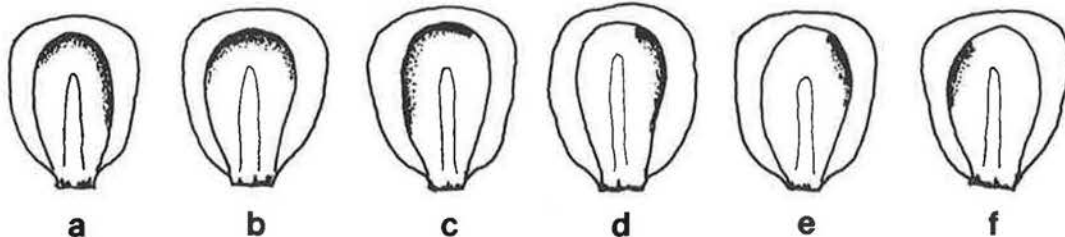
R. S. Poethig

The fate of embryonic cell lineages

Considerable confusion surrounds the fate of the cells produced by the first few divisions of the zygote in maize. It is generally agreed that the first division after fertilization is transverse (note, however, the perplexing results of Bird and Neuffer, in this issue), and that the two products of this division probably form the embryo and the suspensor. The next division of the embryo cell is in a longitudinal plane, but its exact orientation is unclear. Traditional interpretations hold that the products of this division are destined to form the embryo and scutellum. On the other hand, Steffensen has argued that the first longitudinal division defines the midrib line, dividing the plant into left and right halves. Recent results from a clonal analysis of embryogeny indicate that neither of these views is entirely correct.

In order to conduct a clonal analysis of the relationship between the embryo and scutellum, it is necessary to use cell marker mutations that are expressed in both of these structures. For this purpose, we used the stock Wd C-I/wd C; +/R-sc. Kernels of this genotype are colorless due to the dominant color inhibitor C-I, and yield purple sectors in both the aleurone and scutellum when this gene is lost. The loss of the chromosome arm carrying C-I coincidentally exposes wd--an albino mutation that is expressed in the seedling. Thus sectors present in the scutellum can also be observed in the embryo if these two structures share the same lineage.

Plants of the genotype Wd C-I; r-g were pollinated by wd C, Wd 9S ring; R-sc/() and ears were x-rayed (500R, 1 mm Al, 140 kVp) 56 hrs later (note that the Wd ring chromosome is poorly transmitted through pollen, so most of the progeny from this cross are of the appropriate genotype). Histological observations indicated that specimens had undergone 1-2 longitudinal divisions by this stage. At maturity, kernels were screened for sectors in the scutellum and selected kernels were then planted to determine whether sectors extended into the seedling. Six clearly sectorized seeds were observed out of a total of 815. Three of these seeds (a, b and c) gave white seedlings, and 3 gave green seedlings (d, e and f). In addition,



one of the seeds which appeared to lack a sector in the scutellum gave rise to a white seedling with a narrow strip of green tissue along the margin of the first leaf (the apparent absence of a sector in the scutellum of this kernel is probably due to the poor expression of R-sc in the scutellum; the experiment is being repeated using R-scm). White seedlings arose from kernels in which the sector was located at the apex of the scutellum directly in line with the embryonic axis; green seedlings arose from seeds in which the sector was located along one side of the scutellum.

These data demonstrate that the scutellum and the embryo can share the same lineage after the first or second longitudinal divisions. If the lineages of these two structures were separated at this stage, sectors would never encompass both of

them. It is also clear that the lineages resulting from these early divisions are not necessarily destined to form the left and right halves of the plant, otherwise sectors that encompassed 1/2 of the scutellum (Fig. 1d) would have produced half sectorized plants. We have observed half sectorized plants in material irradiated 6 days after pollination, when the embryo was at a globular stage. Such plants are rare, however, and it is more common to find sectors involving smaller or larger fractions of the plant body. Our observations confirm Randolph's conclusion that the early pattern of cell division during maize embryogeny is highly irregular, and provide no evidence of determinate cell lineages.

R. S. Poethig and E. H. Coe

Mixed pollinations with white pollen

Last year, exceptional kernels were reported from crosses that were made with mixtures of white (c2 whp) and yellow pollen. The results of progeny tests, and interpretations, are as follows:

Cross	Ear parent	PP1	PP2	Embryo received	No.	Interpretation
1	c2 whp y	c2 whp y	C2 sh bz wx	PP2	3	Heterofert. (Half-transm.)
				c2 PP2	3	Mut. of C2 to c2
2	c2 whp y	c2 whp Y	C2 sh bz wx	PP2	3	Heterofert. (Half-transm.)
				c2 PP2	1	Mut. of C2 to c2
3	c2 Whp y	c2 whp y	C2 sh bz wx	PP2	7	Heterofert. (Half-transm.)
				c2 Whp y	7	Self-contam.
4	C2 r-g y	c2 whp R-r Y	C2 r-g y	C2 sh bz wx R	1	Outcross
				PP1	5	Transmission
				PP2	5	Heterofert. (Half-transm.)
				c2 R-r y	1	Outcross
5	C2 r-g y	c2 whp R-r y	C2 r-g y	PP1	10	Transmission
				C2 r-r	1	Outcross heterof.
				PP2	4	Heterofert. (Half-transm.)
6	C2 sh bz wx	c2 whp Y	C2 sh bz wx	PP2	2	Heterofert. (Half-transm.)
				PP1	2	Transmission
7	C2 sh bz wx	c2 whp y	C2 sh bz wx	PP2	2	Heterofert. (Half-transm.)
				C2 Whp Sh Bz Wx	1	Outcross
				PP1	2	Transmission
Tests in which the yellow pollen was dried overnight before mixing:						
3	c2 Whp y	c2 whp y	C2 sh bz wx	c2 Whp y	1	Self-contam.
5	C2 r-g y	c2 whp R-r y	C2 r-g y	PP1	3	Transmission
7	C2 sh bz wx	c2 whp y	C2 sh bz wx	PP1	1	Transmission
				PP2	1	Heterofert. (Half-transm.)

Transmission of white pollen was found in 23 cases; all 23 occurred on C2 r-g y or C2 sh bz wx ear parents, and none on c2 ear parents. On the other hand, 27 cases of "Half-transmission" (in which the endosperm was like the white-pollen parent but the embryo received the sperm from the yellow-pollen parent) were found, of which 13 occurred on the c2 ear parents and 14 on the C2 ear parents. Consequently, transmission does occur at a low rate (0.3%) on silks of C2 ear parents, but not on white-pollen ear parents except by heterofertilization (0.2-0.3%). The reciprocal heterofertilization, in which the embryo would receive from the white-pollen source, has not been tested in this material; appropriate tests are planned. A few apparent mutations of C2 to c2 were found in the screening.

It is tempting to suggest, since white pollen appears to be "helped" occasionally by mixing with yellow pollen and by C2 silks, that cross-feeding or protection of the pollen tubes is occurring.

E. H. Coe

A new gene, Tpml: thylakoid polypeptide modifier

This is a preliminary report of a new gene, designated Tpml, an acronym for "thylakoid polypeptide modifier." The gene acts to modify the migrational mobility value of a specific peripheral protein of the chloroplast thylakoid on a density gradient SDS Laemmli gel. The dominant allele, Tpml, conditions a higher apparent molecular weight ("slow") than the recessive ("fast") allele, tpml. With caution I say that the relationship is dominant/recessive, since I have not yet definitively separated a mechanical mixture of the two forms. I have no information concerning chromosomal location.

The two alleles of this gene are prevalent throughout Zea: the domesticated maizes as well as the teosintes. The following typical data indicate Mendelian inheritance:

Cross Family	Phenotype		Ratio	Chi-square
	"slow"	"fast"		
K341-1 selfed	38	15	3:1	0.308
K318-6 x 341-1	39	39	1:1	0.000
K327 x 341-1	30	22	1:1	1.230
K328 x 341-1	12	0		***

***For a cross of heterozygotes, the probability of not encountering a "fast" phenotype is $(0.75)^{12} = 0.0317$; and for a testcross, $(0.50)^{12} = 0.0002$.

Pedigrees: K341-1, (Ky21[EP-cytopl.])xTr)xTr; K318-6, B73; K327, Hy; K328, Mo17.

As soon as I have assayed an increase of my pedigreed seed, I will deposit in the Co-op type-sources: B68 (Tpml/Tpml), B84 (tpml/tpml), and the F1 of those inbreds. After June 1, 1983 a detailed protocol of materials preparation and specific recipes for the gel conditions required to detect and differentiate Tpml vs. tpml will be available upon request to: Stephen A. Modena, c/o E. H. Coe, 210 Curtis Hall, University of Missouri, Columbia, MO 65211.

Stephen A. Modena

Notes on teosintes

Perhaps the "trick" to getting Huehuetenango to flower readily is to expose it to decreasing daylengths. In January 1982 I planted seed obtained from H. H. Iltis in the winter greenhouse here in Columbia. The plants grew well and became very vegetative, but never flowered. This contrasted with Zea diploperennis, Z. luxurians, Nobogambe, Chalco, and Balsas, all of which were profuse. This tall (18") Huehuetenango was maintained through the summer, in situ, until August when I chopped it down. From a tiller lying on the ground I potted a side-shoot (8") with extended, but not rooted, root primordia. It subsequently started to take off about October and flowered on December 1st. The main tassel shed abundant pollen, but all side-shoot tassels were aborted. As of January 1st it continues to flower, though the plant is clearly beginning to senesce. Good seed set was obtained from selfing and by crossing with maize.

Using seed obtained from H. H. Iltis, the following inter-teosinte crosses are readily made: Zea diploperennis x Z. luxurians, Z. luxurians x Z. diploperennis, and Z. diploperennis by Huehuetenango. Crossing Z. luxurians to several Corn Belt inbreds gave scattered seed, but to Mo20W gave full set.

Z. diploperennis is perennial from the fact that it forms rhizomes. Rhizome-like structures are also formed occasionally at above-ground internodes on potted tillers that are allowed to recline sharply (though not touch the soil), or that have been "topped." Typically, a side shoot is formed from a bud which is aligned with and located below the gap in the nodal ring of root primordia. A second type of structure can differentiate instead, characterized by a basal ring of root primordial buds, enclosing scales and a fleshy, rather than leafy apical structure. When this rhizome-like propagule is broken off and potted, it emerges with the same morphology as do the newly appearing shoots from the main rhizome. Perhaps Z. diploperennis would be a good candidate for regeneration experiments.

Stephen A. Modena

whp may be on 2L

Mapping effort for locating whp continued. The arm segments remaining (Modena, MNL 56:48) were tested. Candidate hypoploid plants were identified by distinctive morphology and at least 50% pollen abortion for 1S, 2S, and 10L. All shed yellow pollen. The test using TB-10Sc yielded all normal looking plants, but six out of sixty-two were semi-sterile and all shed yellow pollen. Therefore, the above arms are eliminated.

Randomization was indicated in linkage tests with the following markers: Vg, c2, pro, and wx. The 1-centromeric, 4L, 8-centromeric and 8S(?), and 9-centromeric regions are eliminated. A test with Lg3 confirms elimination of the 3-centromeric region.

Another test with a marked 2S chromosome yielded white pollen plants in coordination with multiple, distal homozygosities, confirming elimination of distal 2S. Unpublished data indicate that B and whp randomize, which eliminates the proximal 2S and 2-centromeric region.

Another test with a marked 2S chromosome yielded white pollen plants in coordination with multiple, distal homozygosities, confirming elimination of distal 2S. Unpublished data indicate that B and whp randomize, which eliminates the proximal 2S and 2-centromeric region.

A test with TB-3La-2L7285 appears to have uncovered whp, placing it on 2L. Among 54 plants observed, 16 plants were candidate hypoploids based on both morphology and pollen abortion percentage. Twelve shed white pollen and four shed yellow pollen. Additionally, two plants of normal stature with no pollen abortion, whatsoever, also shed white pollen. In the early stages of converting the TB stocks to c2/c2, they were all crossed to a c2 source that had partial K55 lineage. This was before K55 was recognized as the source of the whp allele. Effort will be directed to specific linkage tests on 2L.

But there is more! TB-9Sb-4L6222-generated hypoploids also shed white pollen. The putative hypoploids were semi-sterile and less than two feet tall. Thirteen yielded white pollen and five, yellow pollen. Four different TB pollen parents were involved: 3 yp:2 wp; 2 yp:4 wp; 0 yp:1 wp; 0 yp:6 wp. These have had contact with K55 in their derivation. All available TB arms have been tested and only these two arms have tested positively for white pollen phenotype. whp is not close to su or the TB-4Sa breakpoint (Modena, MNL 56:48). Linkage tests this past summer clearly demonstrated that c2 and whp are randomizing. If whp is distal to c2 on 4L, then it becomes impossible for hypoploid c2, whp plants to ever shed yellow pollen! A non-compound TB for 4L of different origin has been crossed to white pollen plants to clear this point up.

Stephen A. Modena

Centric fragments carrying anthocyanin markers

One of the most interesting chromosomal aberrations in plants is the centric ring--a chromosome whose arms are joined to one another so as to produce a continuous ring. Thorough analyses of ring behavior have been performed by McClintock (Genetics 23:315-376 and Cold Spr. Harb. Symp. 9:72-80) and Schwartz (Am. Nat. 87:19-28). They found that a ring chromosome may be lost or multiplied during mitosis and meiosis. Or, as a result of recombination, modified rings may arise which are either deficient or duplicated for the chromatin present on the original ring. When the ring chromosome carries a dominant allele of a mutant present on the homologous normal chromosome this behavior may be followed genetically. Loss or modification of the ring results in the expression of the recessive allele, producing a mosaic plant, i.e., one that shows both the dominant and recessive phenotypes.

Because recessive embryo lethal mutants die at an early stage of development they are difficult to study. The goal of this project was to produce a set of genetically marked ring chromosomes carrying the dominant alleles of these embryo lethal mutations. Such chromosomes could then be used to produce mosaic plants with both normal and lethal tissue. This would make it possible to determine whether or not an embryo lethal mutation is active in a mature plant and if so, provide a way of studying its effects. (Ring chromosomes could also be used to study whether recessive disease mutants show cell autonomy or whenever one needs a situation of producing a mosaic plant made up of sectors of dominant and recessive tissue for a particular gene.)

Plants recessive for eight plant color genes (a1, a2, c2, bz1, bz2, b1, p11, r-r) were fertilized by x-rayed pollen from plants homozygous for the dominant alleles of these genes. Nine thousand forty-eight seeds were planted and 21 mosaic plants were found. The mosaic plants were selfed and crossed onto a set of six tester stocks: a1, a2, c2, bz1, bz2, and r-g. All the tester stocks were also b1, p11. One could determine which of the mosaic-looking plants were caused by physiological conditions or because they contained a ring by examining the outcrossed ears. This is because ring chromosomes are not usually transmitted (or are greatly reduced) through the male. Therefore, if an ear showed the expected 1:1 ratio for a particular gene, one knew a ring carrying that gene was not involved. But if an ear showed only a recessive phenotype (or a greatly reduced number of dominant phenotypes) it was an indication that the mosaic plant carried a ring with that particular dominant gene on it. The data to date indicate we have produced five centric chromosome fragments: one involving chromosome 2 and the B1 locus; one involving chromosome 4 and the C2 locus; two involving chromosome 5 and the A2 locus; and one involving chromosome 6 and the P11 locus. These five plants and their progeny are now being studied cytologically to determine if they contain a ring chromosome. If they do, it should now be possible to undertake an analysis of the embryo lethal mutations or any other situation where a mosaic plant is needed involving these four chromosomes.

Rodney Higgins

Low crossover Y Dt segment not linked to P11

Last year (MNL 56:43), we reported on an x-ray induced abnormality involving reduced crossing over between Y1 and Dt2 on chromosome 6. Our data showed that the Y Dt2 linkage is very tight (less than 1%) as compared to the normal 26%. The low crossover segment's transmission is normal through the female but is reduced to only 32% through the male; there is no male or female gamete abortion in plants heterozygous for the low crossover segment. The Y linked to Dt2 is not allelic to the Y1 from Aho and suggests that Y in the low crossover segment has moved to a new position.

This year we have data suggesting that the low crossover (lco) Y Dt2 segment has moved from chromosome 6. The cross of al, lco Y Dt2 p11/A1, y1 dt2 P11 x al, y1 dt2 p11 was made and the colored seeds with yellow endosperm were planted. The plants were scored for P11. There were 45 P11 and 49 p11 plants, thus suggesting the lco Y Dt2 segment is not linked to p11 on chromosome 6.

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Recurrent selection for rind penetration values for stalk quality improvement

Several methods of evaluating stalk quality in maize have been developed and evaluated, but most breeders still rely on naturally occurring stalk lodging. We evaluated the effectiveness of a penetrometer device for stalk strength improvement by phenotypic recurrent selection.

B(K)RRS, from the Nebraska Agriculture Experiment Station, was planted and the 2nd elongated internodes of at least 300 plants were punched to obtain rind puncture values approximately one week pre-flowering. Ten percent, or approximately 30 plants, were intermated to complete a selection cycle.

Progress through three cycles of selection by the rind penetrometer technique was evaluated by growing the original population (C0) and the first (C1) through third (C3) cycles of selection. For comparison purposes, the original (C0), C1, C2, and C3 of MoSQA and MoSQB that had undergone cyclic selection for stalk strength improvement by the crushing strength method were included in the evaluation test.

Mature stalks from the 2nd elongated internode above the ground level were harvested. Crushing strength, weight of 5.1 cm stalk section and rind thickness were measured.

Gain in stalk crushing strength per cycle as the result of selection for high rind puncture values in synthetic B(K)RRS is shown in Table 1. Comparable values

Table 1. Least squares gains per cycle from selection measured by three stalk attributes as a result of selection for high rind puncture resistance in Synthetic B(K)RRS.

Population †	Stalk crushing strength	Stalk section weight	Rind thickness
	load-kg	g	mm
B(K)RRS (S-HRP)	25*	0.10*	0.0115*
MoSQA (S-H)	21	0.13*	0.0017
MoSQB (S-H)	58*	0.19*	0.0067

* Indicates significance at P = 0.05; tests were based on the appropriate AOV error term.

† S = S₀ plant selection; HRP = rind puncture; and H = High stalk crushing.

resulting from selection for crushing strength in MoSQA and MoSQB are also given. Three cycles of selection in B(K)RRS for high rind puncture values resulted in significant gains of 25 kg per cycle higher crushing strength, 0.10 g per cycle increase in stalk weight and 0.115 mm thicker rind per cycle. For comparison, MoSQA did not show a significant increase in stalk strength or rind thickness in

three cycles of selection for crushing strength, whereas MoSQB had significant increases for crushing strength (58 kg) and stalk weight (0.19 g) but no significant change in rind thickness. From this preliminary study, we suggest that recurrent selection for high rind puncture values in the pre-flowering stage may be an effective method of improving stalk strength, with the advantage that one cycle of selection could be completed each season.

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Light and temperature-related behavior of coleoptiles and epicotyls

In MGCNL 56 we reported data for five sweet corn lines. The present report concerns six field corn lines and two hybrids. Methods of seed germination and light treatments were detailed in the previous report.

As reported for the sweet corn lines, temperature sensitivity is best observed for seedlings germinated in the dark. Filtered and unfiltered light conditions

Table 1. Lengths (cm) of coleoptile and epicotyl tissues after seven days under different light conditions.

	Light Conditions							Temp. Tissue C
	Blue	Green	Yellow	Red	Far-red	White	Dark	
Coleoptiles								
H 95	2.4	1.8	2.7	2.8	2.4	2.0	1.8	21
	2.5	2.1	2.6	2.9	2.2	1.0	1.9	27
A 634	3.8	4.0	3.2	3.6	4.9	2.2	3.1	21
	3.4	3.6	3.1	3.8	4.9	1.8	2.7	27
B 73	3.5	3.5	3.1	3.5	4.5	1.9	3.4	21
	3.2	2.1	2.5	2.9	4.0	1.6	3.9	27
B 37	3.7	3.8	3.3	3.5	3.8	2.1	1.6	21
	3.6	3.6	2.9	3.5	4.7	1.8	4.0	27
Mo 17	2.1	1.6	2.2	2.8	3.2	1.4	0.6	21
	2.3	2.9	1.8	3.0	4.1	1.3	1.9	27
A 632	3.1	3.3	3.0	3.3	3.2	1.3	1.0	21
	2.4	2.6	2.4	2.8	2.6	1.5	1.5	27
B 73 x Mo 17	3.0	3.5	2.7	3.1	3.9	2.1	1.9	21
	3.2	2.2	2.5	3.0	3.9	1.4	2.9	27
A 634 x Mo 17	4.2	3.0	4.0	4.3	5.0	2.4	2.3	21
	3.9	3.9	3.0	3.9	5.2	2.1	3.5	27
Epicotyls								
H 95	0.9	0.7	0.7	0.8	2.1	0.6	10.7	21
	0.7	0.6	0.8	0.8	2.2	---	11.8	27
A 634	1.9	1.8	0.9	1.3	5.9	0.5	12.6	21
	1.0	1.3	0.8	1.3	4.9	0.5	8.9	27
B 73	1.6	1.4	1.1	1.6	4.8	0.4	8.3	21
	1.4	0.7	1.0	1.2	3.4	0.4	8.3	27
B 37	1.5	0.9	0.6	0.7	2.9	0.4	6.5	21
	1.1	1.1	0.6	0.8	2.3	0.5	9.1	27
Mo 17	1.1	0.6	0.7	1.1	2.4	0.5	2.0	21
	0.8	1.0	0.8	1.0	3.1	0.5	6.9	27
A 632	1.4	1.3	0.7	1.2	3.6	0.5	1.9	21
	0.9	0.9	0.7	0.9	2.0	0.6	3.4	27
B 73 x Mo 17	1.6	0.5	1.0	1.1	4.5	0.5	7.8	21
	1.2	1.0	0.8	1.2	3.0	0.5	7.7	27
A 634 x Mo 17	1.6	0.8	1.2	1.2	4.8	0.6	10.7	21
	1.3	1.3	0.7	1.3	4.9	0.5	13.3	27

tend to reduce performance differences at the two temperatures, 21 and 27 C. It is interesting to note for epicotyls and coleoptiles that less growth takes place in continuous white light, but under all light filters tested, coleoptiles, especially, grow longer when the light is continuous throughout the seven-day period. The longest coleoptiles are found under far-red light, even longer than those following continuous darkness. Though epicotyl growth is longest under dark conditions, a considerable range of variation is found among the six lines tested. Far-red light produces epicotyls which stand out as longer than those produced under blue, green, yellow and red. The two hybrids of Table 1 show epicotyls longer than those of the Mo17 parent under the conditions of darkness and far-red light. Though in the same direction as the epicotyls, less difference is noted for hybrid coleoptiles when compared back to the Mo17 parent coleoptiles.

An interesting observation from a comparison of sweet corns and field corns under the germination conditions above is that a sweet corn such as Sprite 142, with one fifth of the seed weight of certain hybrids, can produce similar volumes of germinating tissues with far less endosperm reserves. Where the number of seedlings produced per weight of dry seed is an economic consideration, sweet corns could deliver five times as many seedlings without serious sacrifice of tissue volume following seed germination (the help of Ohio Foundation Seed Inc. and Pioneer Hi-bred International in supplying seed is gratefully acknowledged).

Bernard C. Mikula and Amy Smith

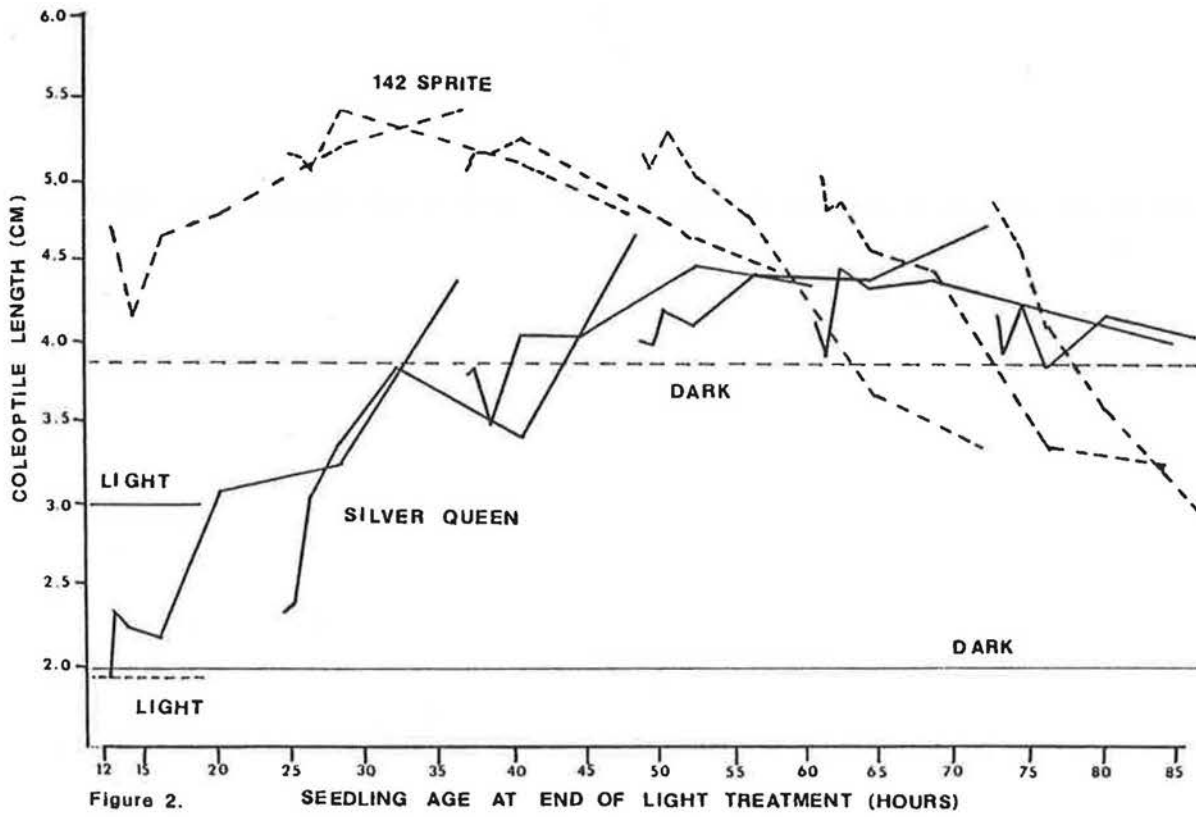
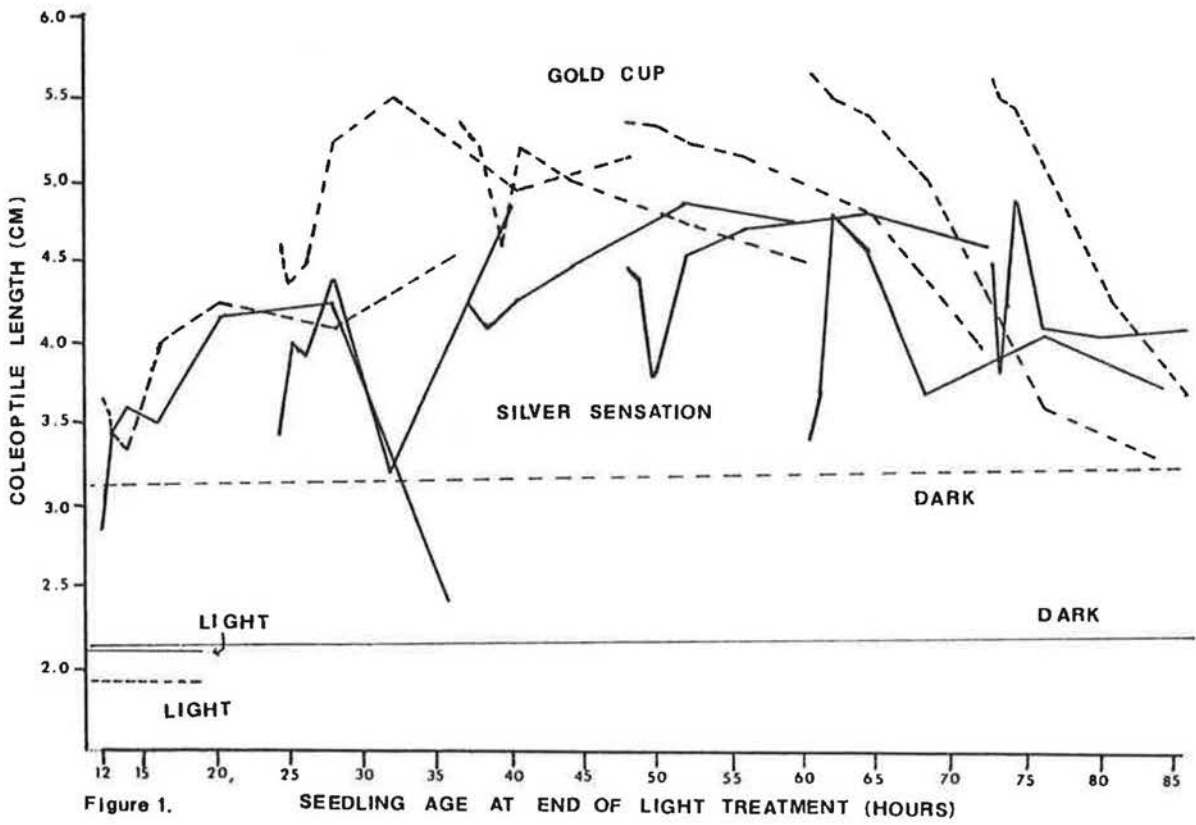
Light probing of developmental potentials of coleoptiles and epicotyls

In MGCNL 56 we reported that at 27 C coleoptiles of sweet corn variety Silver Queen were longer following seven days of continuous light treatment, compared with those grown for the same period in continuous darkness. At this temperature we have found most of the 15 different lines examined show quite the opposite: continuous light inhibits both coleoptile and epicotyl growth. This unusual behavior prompted a closer examination of the light dependence of coleoptile and epicotyl development.

Using the same methods reported in MGCNL 56, we have investigated the relation of short periods of white light on coleoptile and epicotyl elongation. Germinating seedlings were exposed to a single period of growth chamber light lasting for 1/2, 1, 2, 4, 8, 16, or 24 hours; seedlings remained in darkness for the remainder of the seven-day incubation period. Duplicate pans of germinating seeds of each of four different lines were subjected to each of the light treatments. Every twelve hours a new set of seedlings was given the above light treatments; the new seedlings were thus twelve hours older than the previous set. The last set of treatments was begun at 72 hours and completed at the 96th hour. Differences in coleoptile and epicotyl measurements represented the effect of a single light exposure in the seven-day period. Light treatments, therefore, represent a photo probe for the developmental potential of coleoptiles and epicotyls during these first 96 hours of seedling development.

Figures 1, 2, 3, and 4 show length of seven-day-old coleoptiles and epicotyls following exposure to single light periods of 1/2, 1, 2, 4, 8, 16, or 24 hours. Light periods are represented by points on lines at the termination of the light period. For each line, representing seven different light treatments, it is possible to associate the length of the light period with the age of the seedling and the resulting length of coleoptile or epicotyl. In each figure, the six solid lines and six dotted lines represent new sets of experiments begun at twelve hour intervals. The straight lines indicate the length of tissues grown under continuous light or darkness. Epicotyls fail to grow in continuous light.

In Fig. 1, seedlings of sweet corn variety Gold Cup, grown under conditions of continuous light, produce coleoptiles approximately 2 cm. In the dark these same



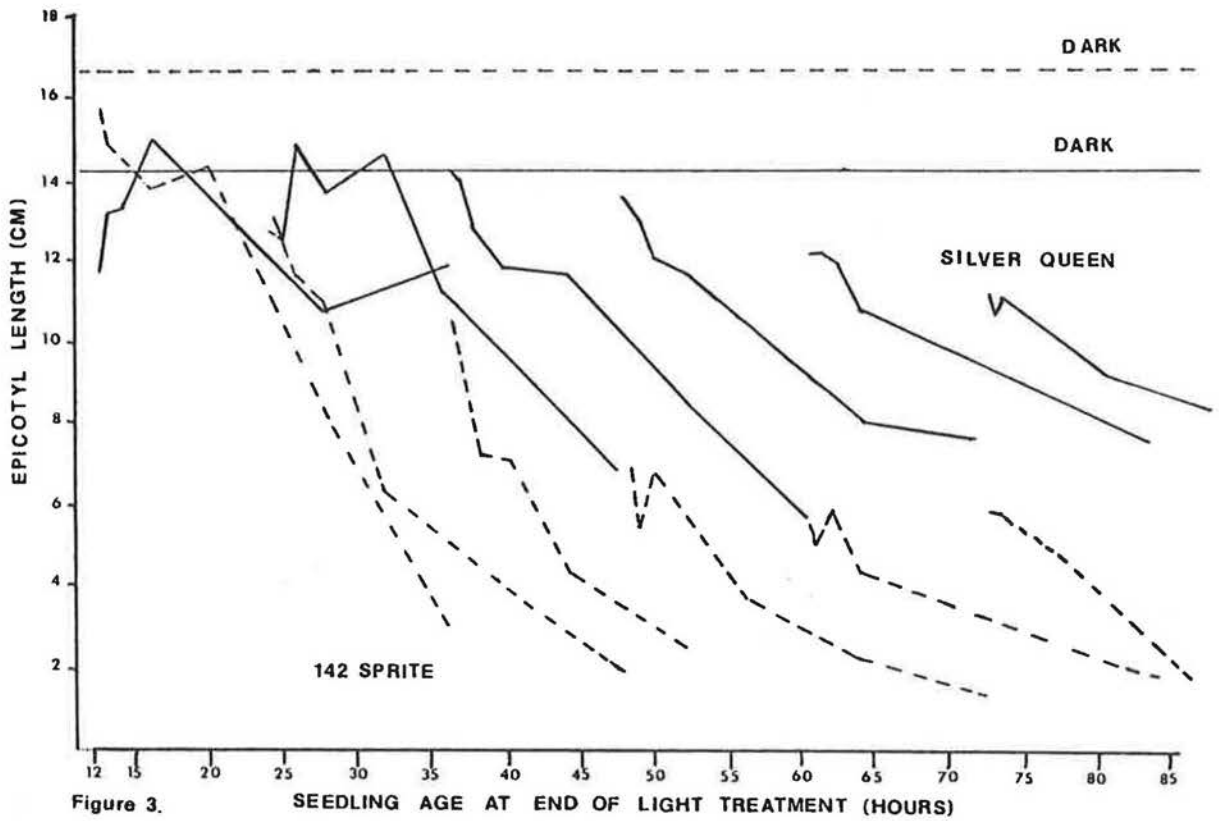


Figure 3.

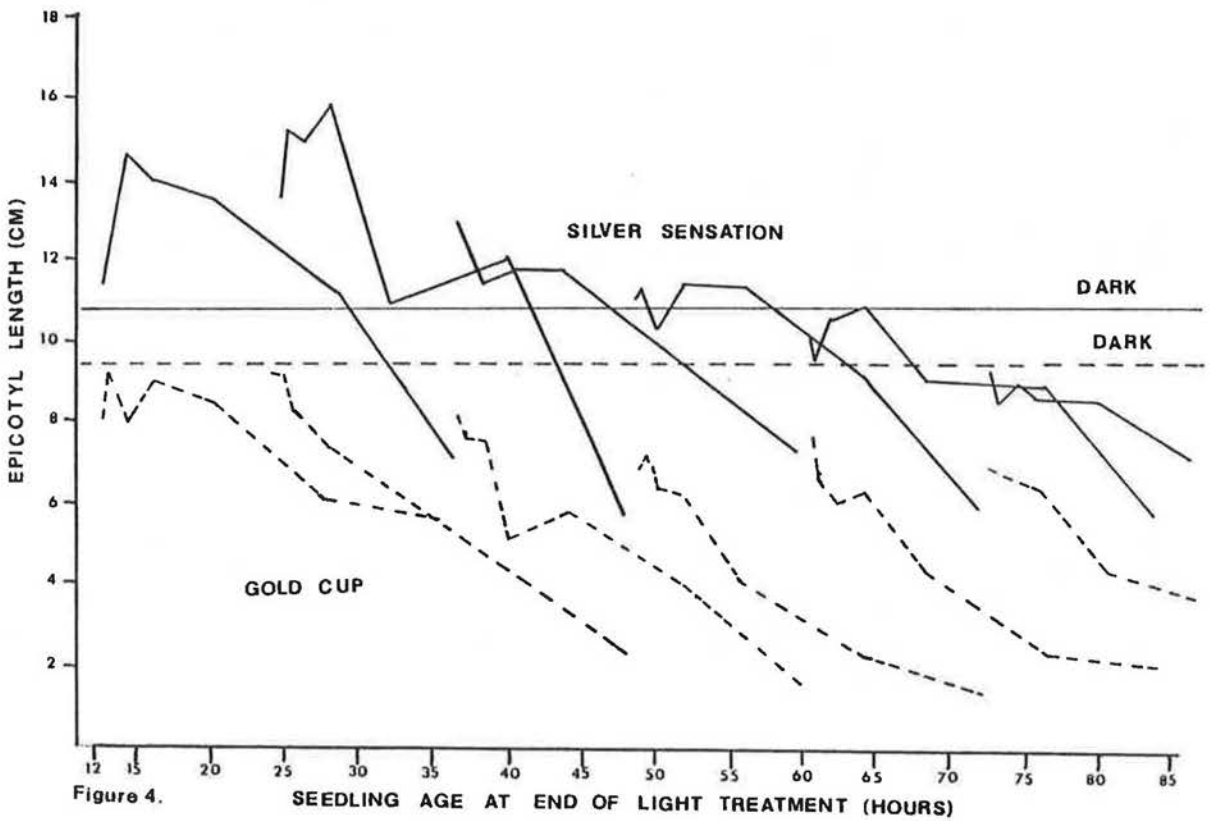


Figure 4.

coleoptiles reach 3.2 cm in length. One must conclude that continuous light inhibits coleoptile development. However, short light treatments can cause Gold Cup to reach 5.6 cm, nearly double the lengths found after continuous darkness. Figs. 1 and 2 show in four different lines that as the light treatments are lengthened from 1/2 hour to 24 hours, an increase in coleoptile length takes place. Depending on the line, age of seedling, and length of treatment, light can stimulate as well as inhibit coleoptile elongation. Coleoptiles of Silver Queen show a gradual increase in length in response to longer light periods, reaching a maximum at about 70 hours before increased inhibition becomes evident. In Sprite, a yellow sweet corn, coleoptiles reach a maximum after the first 24 hours, and after this period of development increases in light reduce coleoptile lengths as the seedling ages. It is remarkable, too, how soon seedlings are light receptive: Figures 1 and 2 show that 1/2 hour of light after 12 hours of imbibition produces a measurable increase in coleoptile length. Gold Cup, another yellow sweet corn variety, shows maximum coleoptile length following 1/2 hour light when seedlings are 60-72 hours old, resulting in coleoptiles twice the length of dark-grown and three times light-grown coleoptiles. Silver Sensation, a white-seeded sweet corn, shows erratic behavior, especially for light treatments of 1/2 to 8 hours for all 12 hour periods. To a limited extent this behavior is reflected in the first few light treatments of all lines. One may speculate that this same behavior is found and reported in phototropic behavior of coleoptiles as first positive and first negative curvature as a result of increasing light dosages.

Compared to coleoptiles, epicotyls show less pronounced increases in length in response to light over dark controls. Silver Queen epicotyls in Fig. 3 exceed dark controls for a few light treatments administered within the first 36 hours. Epicotyls longer than dark controls were recorded in Fig. 4 for Silver Sensation for treatments up to 60 hours. As seedlings age, Figs. 3 and 4 show that epicotyls tend to shorten with increased exposure to light. Epicotyls of the yellow sweet corns, Sprite and Gold Cup, remain shorter than those grown under continuous dark conditions.

From a practical standpoint, the behavior of coleoptiles and epicotyls of Silver Sensation and Silver Queen could represent a more desirable seedling quality for areas where seed emergence is a problem. Continued growth of both tissues under weak light conditions near the soil surface could result in higher percentages of emergent seedlings. Seedlings with greater light sensitivity could result in premature cessation of growth in both tissues before complete emergence. The methods used above provide a simple system for selecting the seedling behavior needed. Another interesting point from the four figures is that the greatest differences among the four lines can be seen during the early germination period. As one approaches the 70th hour a convergence of behavior is evident, especially among the coleoptiles. This might be expected, since under field conditions selection for seedling behavior is likely to be practiced at emergence, and most lines would be exposed to convergent selection at this time.

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Light dosage for 50% inhibition of epicotyl growth

In the previous section it was pointed out that epicotyl growth can be inhibited by light. Seedlings were germinated as outlined in MGCNL 56. White light from a 150W incandescent source (with internal reflector) was administered to seedlings 48 hours old. No light treatments exceeded 30 minutes; intensity was controlled by a rheostat. Table 1 shows the dosage-response relationship for Sprite, a yellow

Table 1. Effect of light dosage on epicotyl and coleoptile lengths of seven-day-old seedlings incubated in the dark except for the above light treatment. Light administered to 48-hour-old seedlings; no light treatments exceeded 30 min.

Dosage Meter Candle Seconds	Epicotyl Length (cm)	Coleoptile Length (cm)
15.5 x 10 ⁶	6.1	5.3
	6.1	5.4
7.75 x 10 ⁶	7.4	5.4
	7.0	5.4
3.88 x 10 ⁶	8.6	5.4
	8.8	5.5
1.55 x 10 ⁶	11.9	5.5
	10.4	5.4
1.03 x 10 ⁶	11.9	5.2
	11.4	5.6
5.17 x 10 ⁵	13.0	5.0
	13.5	4.7
2.58 x 10 ⁵	14.6	4.9
	12.4	5.0

sweet corn variety. As light dosage is increased to nearly 100x, the length of epicotyls is reduced to less than 1/2 those developed in continuous darkness. It is interesting to note that over this range little or no difference is produced in coleoptile length. It may be concluded that the energy for stimulating coleoptile elongation is of the order of 100x less than the energy required for 50% reduction in epicotyl lengths.

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Temporal synthesis of mitochondrial DNA in maize cell suspension culture

The maize mitochondrial genome is composed of small circular and linear DNAs, in addition to a large chromosomal DNA. Kemble and Bedbrook (Nature 284:565, 1980) described a 2.3 kb linear molecule and a 1.94 kb circular molecule in N cytoplasm maize. The circular molecule is detected as linear, open-circular, and covalently closed circular molecules in agarose gels. Cell suspension cultures of the Black Mexican Sweet line (N cytoplasm) contained the same family of low molecular weight DNAs as is characteristic of the intact plant (Chourey and Kemble, 5th International Cong. of Plant Tissue and Cell Culture, Tokyo, Japan, in press, 1982). The Black Mexican Sweet line also contains an additional small DNA detected as a covalently closed circle, open circle, and a linear molecule of

approximately 1.4 kb. The relative levels of the 2.3 kb and 1.94 kb molecules are higher than the 1.4 kb molecules. In the present report, we describe replicative properties of the low molecular weight DNAs using Black Mexican Sweet cell suspension cultures which readily incorporated ^{32}P -orthophosphate into mtDNA. The relative level of DNA synthesis in each class of DNA was monitored through UV fluorescence of ethidium bromide stained agarose gels and autoradiography intensities on X-ray film of the corresponding gels.

15 μCi ^{32}P per ml of culture medium was added to cultures in either logarithmic phase (7 days post subculture) or stationary phase (15 days post subculture), and allowed to incubate for 24 hours. The cells were harvested and the mitochondrial DNA was extracted and electrophoresed. The DNA extracted from logarithmic phase cultures showed ^{32}P incorporation into all classes of mitochondrial DNA. The 2.3 kb and 1.94 kb molecules had equal intensities in both ethidium bromide staining and autoradiography. The three forms of the 1.4 kb molecule were present, but in much lower intensity. However, DNA extracted from stationary phase cultures had the same relative intensities of DNA when stained with ethidium bromide, but no ^{32}P was incorporated into the DNA. At this sensitivity, no mitochondrial DNA synthesis could be detected, although the majority of cells were still viable, as evidenced by their fluorescence in the presence of fluorescein diacetate stain. Furthermore, if the stationary phase cells were removed from the exhausted medium and placed into new medium with ^{32}P for 24 hours, there was renewed incorporation of label into mitochondrial DNA. However, the stoichiometry of the low molecular weight DNAs had shifted so that the 1.94 kb molecule was in much higher concentration and had incorporated more ^{32}P , relative to the 2.3 kb and 1.4 kb molecules. When stationary cells are incubated for prolonged periods in fresh medium prior to the 24 hours labeling with ^{32}P , stoichiometry and ^{32}P incorporation characteristic of cells in logarithmic growth was observed.

These data indicate that mitochondrial DNA is being rapidly synthesized in cells from logarithmic growth phase cultures. The progression into stationary phase was correlated with a suspension of mitochondrial DNA synthesis, as seen by a failure of cultures to incorporate ^{32}P into mitochondrial DNA over a 24-hour period. The addition of new media to the stationary cells resulted in a preferential synthesis of the 1.94 kb molecule, relative to the other classes of low molecular weight DNA.

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S1 and S2 sequences are common among normal, fertile cytoplasm mitochondrial DNAs

Integrated sequences of the S1 and S2 episomal DNAs in Wf9(N) mitochondrial DNA (mtDNA) have been mapped through cosmid cloning (D. M. Lonsdale, R. D. Thompson and T. P. Hodge, Nucl. Acids Res. 9:3657, 1981). Since extensive deletion of these sequences is a characteristic of the T and C male-sterile cytoplasm, we surveyed 12 common North American normal cytoplasm lines to assess the frequency and conservation of these sequences in male-fertile cytoplasm. Lines examined were W64A, Mo17, A619, A632, F44, F6, B37, NC7, Wf9, W182BN, A188, and Black Mexican Sweet. MtDNAs were prepared from each line, restricted by BamH1, electrophoresed, and transferred to nitro-cellulose. Nick-translated S1 and S2, as well as cosmids 2c11 and 2c44, were used as probes. Cosmid 2c11 spans the S1 region, while cosmid 2c44 spans the S2 region; these cosmids were cloned from Wf9.

The mtDNA from each line displayed major homology to S1 and S2. Homology to S1 occurred in a BamHI fragment of 6.9 kb in 8 of 12 lines: Wf9, W64A, Mo17, NC7, A619, A632, F44, and B37, consistent with the Wf9 model structure. Homology in F6, W182BN, and A188 occurred in a BamHI fragment of 6.6 kb, while Black Mexican Sweet displayed major homology at 8.1 kb. Homology to S2 was conserved among all entries, with major homology at 4.4, 0.95, and 2.1 kb, consistent with the structure of the model Wf9 mtDNA.

Probing with cosmid 2c11 verified that mtDNA structure of the eight lines which carried homology to S1 at 6.9 kb was conserved through a 40 kb region spanning the S1 site. F6, W182BN, and A188 were altered in fragments adjacent to the S1 site, and by rearrangement of sequences ca. 16 kb from the S1 site. Black Mexican Sweet displayed singular rearrangements, unlike all other lines examined. Hybridization with cosmid 2c44 demonstrated that the S2 region was conserved among all entries.

As was demonstrated with Wf9, none of the normal cytoplasms carried complete integrated copies of the S1 DNA; the 1400 bp repeat, characteristic of isolated S1 and S2 DNAs, was absent in the integrated form of S1.

Associated with the rearrangements which characterize F6, W182BN, and A188, we detected an alteration in the size of a linear, free DNA, occurring at 2.3 kb in most lines. In these three lines, no 2.3 kb DNA was apparent, but a 2.1 kb DNA was detected, co-migrating with a similar DNA in T cytoplasm mtDNA.

Although this survey included only defined male-fertile cytoplasms, and not exotics nor teosintes, integrated copies of S1 and S2 sequences would appear to be a constituent of these normal cytoplasms. Whether or not integrated copies are an obligate component of a "normal" mtDNA cannot be determined by these data. MtDNA from S cytoplasm also carries extensive homology to the episomes, which clearly distinguishes S from C and T cytoplasms. The role(s) which sequences of these DNAs may play in differentiating normal cytoplasms from the S male-sterile cytoplasm is thus obscure. MtDNA structure of the S cytoplasm through the S1 and S2 regions is different from all normal cytoplasms examined to date, and our current efforts are directed toward constructing restriction maps of these regions in S cytoplasm. (Supported in part by NATO Research Grant 283.81 to DML and DRP).

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Chromosomal location of Px3 (peroxidase isozyme) locus

Peroxidase isozymes governed by the Px3 locus are among the most ubiquitous and easily studied in maize (Brewbaker and Johnson, MGCN 46:29-33, 1972). The chromosomal location of Px3 was studied using genetic markers and translocations on chromosome 7. Dr. David Weber (1975, unpublished) was able to localize Px3 to chromosome 7 using trisomics in crosses with our stocks. Four chromosome 7 mutants were used as genetic markers--opaque-2 (7-16), Teopod (7-46), slashed (7-50) and Papyrescent glumes (7-112). Waxy T7-9a, with breakpoint at 7L.63, was also used.

Testcrosses for the mutant linkage study were made between pertinent heterozygotes and homozygous testers of the U. Hawaii collection, based on the tropical flint inbred Hi27 (Brewbaker, MGCN 46:33-37, 1972). The unique allele Px3-5, derived from the Race Puya, was introduced to the crosses to rule out contamination during pollinations.

Highly significant linkage ($P < .01$) was obtained only between the Px3 locus and Pn (7-112), near the long arm end of chromosome 7 (Table 1). Our Pn stock shows

Table 1. Segregations of linkage testcrosses involving Px3 alleles (R and NR = Recombinant and Non-Recombinant genotypes)

Marker	Cross		Offspring	No. of Plants	Tot.	Chi Square
(7-16)	o2	o2Px3-2 X o2Px3-2	(NR) o2Px3-2/o2Px3-2	42	80	2.22
			(NR) + Px3-5/o2Px3-2	38	(NR)	P=
	o2Px3-2	+ Px3-5	(R) o2Px3-5/o2Px3-2	50	100	.5-.4
			(R) + Px3-2/o2Px3-2	50	(R)	
(7-46)	Tp	+ Px3-1 X TpPx3-2	(NR) TpPx3-2/+ Px3-1	44	86	2.92
			(NR) + Px3-5/+ Px3-1	42	(NR)	P=
	+ Px3-1	+ Px3-5	(R) TpPx3-5/+ Px3-1	38	65	.05-.1
			(R) + Px3-2/+ Px3-1	27	(R)	
(7-50)	s1	s1Px3-1 X s1Px3-2	(NR) s1Px3-2/s1Px3-1	28	74	1.69
			(NR) + Px3-5/s1Px3-1	46	(NR)	P=
	s1Px3-1	+ Px3-5	(R) s1Px3-5/s1Px3-1	26	59	.1-.2
			(R) + Px3-2/s1Px3-1	33	(R)	
(7-112)	Pn	+ Px3-1 X PnPx3-2	(NR) PnPx3-2/+ Px3-1	25	55	31.16
			(NR) + Px3-5/+ Px3-1	30	(NR)	P=
	+ Px3-1	+ Px3-5	(R) PnPx3-5/+ Px3-1	6	10	<.01
			(R) + Px3-2/+ Px3-1	4	(R)	

excellent penetrance in heterozygotes. The recombination percentage between Px3 and Pn was 15.38%, thus suggesting the location of Px3 to be at or near 7-96.62.

The translocation stock, wx T7-9a (breakpoints 7L.63 and 9S.07), was also used in crosses, with the heterozygotes testcrossed to a stock made homozygous for wx and Px3-1 through repeated backcrosses. Linkage between Px3 and the translocation breakpoint (7L.63) was highly significant (Table 2), with a recombination percentage of 41.84.

Table 2. Linkage testcrosses between Px3 and wx in translocation wx7-9a (R and NR = Recombinant and Non-Recombinant genotypes)

Marker	Cross		Offspring	Plants*
(7L.63)	wx	+ Px3-1 X wxPx3-1	(NR) wxTPx3-1/wxPx3-1	248
			(NR) wx Px3-1/+ Px3-2	240
	wxTPx3-2	wxPx3-1	(R) wxTPx3-1/wxPx3-2	177
			(R) wx Px3-1/+ Px3-1	174

*Chi-square testing the expected 1R:1NR ratio = 22.36, P<.01;
Mean recombination percentage = 41.84

The suggested genetic location of the 7L.63 breakpoint is 7-58.8 (Phillips, 1969, Genetics 61:107), based on linkage with 7L markers including s1 (7-50) and ij (7-52). Px3 showed no linkage with genes proximal to ij (Table 1), and must clearly be distal to the 7L.63 breakpoint, at or near 7-100.64 (58.8 + 41.84).

The locus Px3 may thus be localized between 96.62 and 100.64 on the long arm of chromosome 7. A working value of 7-99 is proposed, based simply on the average of these two values. This region of chromosome 7 is poorly marked:

s1 ij	Bn	Px3	Pn
50 52	71	99	112

We could not obtain a reliable stock of Bn, a gene which was together with slashed in the early studies of Hayes and H. E. Brewbaker (J. Amer. Soc. Agron. 18:761-767, 1926). The ubiquity of Px3 in maize tissues, its polymorphism and ease of identification (Brewbaker and Johnson, MGCN 46:29-33, 1972) thus make it a convenient marker for this region.

James L. Brewbaker

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Callus cultures of shoot base and node

The present study was undertaken mainly to test the efficiency of different anthocyanin genotypes and some local varieties of maize in callus initiation and plantlet regeneration. Different explants (seedling root, shoot base, node, endosperm, embryo and glume) were used. Regenerable cultures have been obtained from immature embryos, shoot bases, first nodes and glumes.

Shoot bases of germinated immature embryos and first nodes of 6- to 7-day-old seedlings were inoculated onto Linsmaeir and Skoog's media containing 2,4-dichlorophenoxy acetic acid (2,4-D), and the cultures were placed under diffused light at 26±1 C. Callus was initiated within two weeks of inoculation from shoot base and node. Subcultures were carried for every two weeks, the 6- to 7-week-old calli of the shoot bases were kept for regeneration on LS basal medium devoid of hormones, and nodal calli on LS medium supplemented with 1 mg/l indole 3-acetic acid (IAA) and 1 mg/l Kinetin (KN).

Germinated immature embryo shoot base not only callused more efficiently compared to mature seedling shoot base, but organogenesis resulting in distinct roots and shoots was observed with greater frequency, while only rhizogenesis was observed in the latter. First node callused efficiently on LS medium supplemented with 4 mg/l 2,4-D compared to medium with 2 mg/l 2,4-D. Regeneration into roots and shoots was observed on LS medium with 1 mg/l IAA and 1 mg/l KN. Callus initiation and plantlet regeneration was more efficient in the 150/2 Ludhiana variety of Dr. A. S. Khehra, for shoot base and heterozygote of bronze-1 and bronze-2 (Bz1 bz1 Bz2 bz2) for node, over other tested varieties.

K. V. Rao, P. S. Prasanna and G. M. Reddy

Plantlet regeneration from glume cultures

Successful plant regeneration was reported from the calli derived from immature embryos, immature tassels and nodal sections of maize (S. J. Molnar et al., MGNL 54:52, 1980; C. A. Rhodes et al., MGNL 56:148, 1982). The present study was mainly aimed at testing the totipotency of glumes of different developmental stages of four genotypes.

Fresh immature tassels of sweet corn, DHM-101, DHM-103 and Ganga-5 were collected from 55, 60 and 65-day-old field grown plants (Kharif season, 1982), and were sterilized with 0.1% mercuric chloride for three minutes. Antherless glumes were inoculated onto Murashige and Skoog's medium supplemented with 2 mg/l 2,4-D. Subcultures were made every three weeks on MS medium with 1 mg/l 2,4-D.

Glumes of sweet corn after two to three weeks of inoculation gave rise to callus, whereas other genotypes responded poorly. Rapidly growing light green friable callus was observed from the base of the glume irrespective of the position of the glume on the tassel. Of the different age groups tested, glumes of 55-day-old plants exhibited greater response of callus initiation (45-50%) compared to others. Rhizogenesis was observed on basal medium devoid of hormones, and complete plantlets regenerated on MS medium supplemented with 1 mg/l IAA and 1 mg/l KN.

Out of the different hormonal combinations and concentrations tested, 1 mg/l IAA and 1 mg/l KN gave high frequency of plantlet regeneration. The specific age of the glume seems to play an important role, both in callus initiation and plantlet regeneration. This finding may be useful in clonal propagation of male sterile lines.

P. S. Prasanna, K. V. Rao and G. M. Reddy

Esterase isozyme studies in callus cultures

The enzyme polymorphism exhibited by isozyme patterns serves as a model for analyzing the gene action at different developmental stages, and the physiological status of the tissue differentiated to perform specified functions. Multiple molecular forms of a number of enzymes which were tissue specific were analyzed in inbred lines of maize (J. G. Scandalios, Proc. XII Int. Cong. Genet. II:79-80, 1980). Tissue culture studies in our laboratory proved that the calli derived from immature embryos and glumes were capable of plantlet regeneration, whereas the seedling root calli failed to regenerate. The differences were mainly attributed to the physiological status and the extent of differentiation among the constituent cells of the explant cultures. Esterases exhibited polymorphism with different tissues at the whole plant level (Scandalios, J. Hered. 55:281, 1964). In the present study, regenerable calli derived from immature embryos and glumes and the nonregenerable calli of seedling root were compared for their esterase isozyme pattern.

Immature embryos (10-day-old) and glumes (60-day-old plants) of sweet corn were obtained from the field grown plants. Root explants were taken from one-week-old seedlings grown under aseptic conditions. Callus cultures were initiated on MS medium containing 2 mg/l 2,4-D. Four-week-old calli derived from the above explants were used for electrophoretic studies using standard PAGE technique. The gels were incubated for one hour in 100 ml of 0.6M phosphate buffer, pH 6.1, containing alpha-naphthyl acetate as substrate and fast blue RR as dye coupler to detect the esterases.

Significant differences were observed in esterase pattern in regenerable and non-regenerable calli. Calli of embryo, glume and root exhibited a total number of nine, eight and twelve bands, respectively, of which three common bands at Rf values 0.33, 0.46 and 0.70 were observed. The regenerable glume and embryo calli have shown one common band at Rf 0.22, which was absent in non-regenerable calli. The preliminary observations suggest that the presence of a specific band in regenerable, and its absence in nonregenerable calli, indicates a possible involvement of a specific esterase in differentiation.

P. S. Prasanna, K. V. Rao and G. M. Reddy

Isoperoxidases in root callus cultures

In this study, four-week-old calli derived from seedling root of sweet corn and r2 r2 stock were analyzed for their isoperoxidases by adopting standard polyacrylamide gel electrophoretic (PAGE) technique. The isozymes of 0.165, 0.47 and 0.87 were common for both the inbred lines. The isoperoxidases with Rf values of 0.09, 0.25, 0.43 and 0.81 were observed in the sweet corn line, whereas r2 r2 exhibited different isoperoxidases with Rf values 0.14, 0.52 and 0.74, suggesting that the genotypic differences in isoperoxidases also exist in callus cultures.

K. V. Rao, P. S. Prasanna and G. M. Reddy

Chromosomal studies in callus cultures

Preliminary cytological studies of root, endosperm and glume calli have shown significant variations in ploidy levels. Root calli have shown 96% of diploid cells; endosperm calli have shown 70% triploids with 9-20% diploids and 5-10% haploids. The glume calli exhibited differences in chromosome number from 11-28, with a greater frequency of hypoploids.

K. V. Rao, P. S. Prasanna and G. M. Reddy

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Cornell University

Plant regeneration from cultures of inbred W182BN in N, C and S cytoplasm

Regenerating cultures of the important New York State inbred W182BN in three different cytoplasm (N, C and S) have been established and maintained since March, 1981. Immature embryos (ca. 1 mm long) were cultured on Murashige-Skoog medium containing 5 mg/l 2,4-D. Scutellar callus from some of these embryos has been subcultured repeatedly on the same medium, either liquid or agar-solidified; the tissue forms small superficial organized areas. Reduction or elimination of the 2,4-D leads to development of compact white structures. Continued growth into plantlets is promoted by increase of the sucrose level to 10%. Morphogenesis appears to be via an embryogenic pathway. Further growth of leaves and roots proceeds on media containing decreased levels of salts and sucrose.

Several dozen plants from each cytoplasm have been transferred from culture to vermiculite and then to soil, and grown to maturity under greenhouse conditions. A wide range of morphology has been observed, from essentially normal phenotype to greatly reduced form with only a few leaves before development of a terminal ear shoot. Tassels that developed usually contained some silks as well. No shifts from male-sterile to male-fertile tassels (as have been seen in some plants regenerated from T cytoplasm scutellar cultures) were seen in the plants regenerated from W182BN cms-C and cms-S cultures, but the generally poor tassel development makes ratings difficult. Mitochondrial DNA from leaves of plants regenerated from S cytoplasm embryos is being analyzed to see whether the characteristic S-1 and S-2 plasmids are still present.

Many of the plants regenerated from N, C and S cultures have successfully been pollinated with pollen from seed-grown W182BN plants. Study of the progeny of regenerated material is in progress; it should help clarify whether morphological changes seen in the plants obtained directly from long-term cultures are ephemeral physiological ones induced by culture conditions, or true nuclear or cytoplasmic mutations.

Elizabeth D. Earle

Structure of the gene-specific toxin from *Helminthosporium carbonum* race 1

In the last year, several papers have appeared on the structure of HC-toxin, a phytotoxin produced by the maize pathogen *Helminthosporium* (perfect stage *Cochliobolus*) *carbonum* race 1. This toxin has the same host range as does the fungus and is thus "host-specific." Both *H. carbonum* race 1 and HC-toxin selectively affect maize that is homozygous recessive at the nuclear *Hm* locus, located on the long arm of the first chromosome.

The structure of HC-toxin, as established and confirmed by four different laboratories, is a cyclic tetrapeptide, cyclo(L-Aoe-D-prolyl-L-alanyl-D-alanyl) (Fig. 1), where Aoe stands for 2-amino-8-oxo-9,10-epoxidecanoic acid (J. M. Liesch et al., 1982, *Tetrahedron* 38:45-8; J. D. Walton et al., 1982, *Biochem. Biophys.*

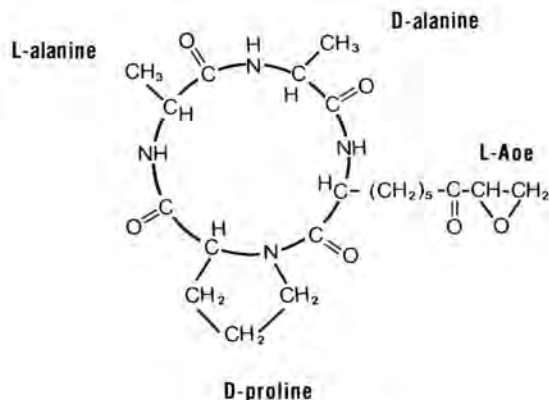


Fig. 1. Structure of HC-toxin

Res. Comm. 107:785-94; M. R. Pope et al., 1982, *Phytopathology* 72:941; M. Kawai, D. H. Rich, J. D. Walton, B. W. Gibson, in preparation). Walton et al. (1982) purified HC-toxin from culture filtrates of the fungus by solvent extraction, gel filtration, and HPLC. The MW and elemental composition were determined by high resolution fast atom bombardment (FAB) mass spectrometry. The amino acid composition was determined with an automatic amino acid analyzer and by ^1H and ^{13}C NMR, and their sequence by GC-MS of the derivatized products of a partial acid hydrolyzate. This last step was especially important because cyclic peptides are notoriously difficult to sequence by other techniques. Our original conclusion that HC-toxin contained two L-alanine residues has been re-examined in light of NMR studies on the conformation of HC-toxin by Daniel Rich at the University of Wisconsin, suggesting that the second alanine has the D configuration, and we have now established by two criteria (HPLC and automatic amino acid analysis of D- and L-amino acid oxidase digests of an acid hydrolyzate of HC-toxin) that this is, in fact, the case (M. Kawai et al., in preparation).

HC-toxin has elemental composition $\text{C}_{21}\text{H}_{32}\text{N}_4\text{O}_6$ and MW 436. It is unusual in being both chloroform and water soluble. It can be isolated from culture filtrates of *H. carbonum* race 1 in up to 10 mg/l quantities. It inhibits root growth half-maximally at between 0.2 and 1.0 $\mu\text{g}/\text{ml}$; resistant maize is affected at approximately 100 times higher concentration.

The unusual epoxide-containing alpha-amino acid Aoe has been found previously in two other fungal cyclic tetrapeptides, the phytotoxin Cyl-2, cyclo(L-Aoe-D-O-methyltyrosine-L-isoleucine-L-pipecolic acid), and the cytostatic agent chlamydocin, cyclo(L-Aoe-alpha-aminoisobutyryl-L-phenylalanyl-D-prolyl). Recent studies by Daniel Rich have shown that the peptide backbones of chlamydocin and HC-toxin have the same conformation in chloroform. Both chlamydocin and HC-toxin (J. D. Walton and E. D. Earle, 1983, *Physiol. Plant Path.*, in press) require an intact epoxide for activity. Based on several criteria, it seems likely to us that chlamydocin and HC-toxin have the same site of action, which is still unknown. Chlamydocin is much less host-specific than HC-toxin; although HC-toxin-sensitive roots (genotype *hm hm*) are equally sensitive to HC-toxin and to chlamydocin, HC-toxin-resistant roots (genotype *Hm Hm* or *Hm hm*) are much more sensitive to chlamydocin than to HC-toxin (unpublished observations). Mammalian cells in culture are sensitive to both chlamydocin and HC-toxin at less than 1.0 ng/ml (unpublished observations).

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University of Kalyani

Giemsa banding method in callus culture

With the development and refinement of plant tissue culture techniques, the prospects of chromosome manipulation at the cellular level are quite promising. Plant tissues and cells, like their animal counterparts, display more than the usual degree of genomic instability when they are removed from the stabilizing environment and plunged into the alien environment of a culture vessel. This ranges from increases in ploidy level to different degrees of aneuploidy. Given the widespread intentions to apply in vitro methods to somatic cell genetics, it is particularly important to develop a suitable banding methodology, which would aid not only in the identification of structural changes but also in the location of those points at which chromosomes break.

In the present communication, a suitable method for Giemsa banding in analysis of chromosomes under in vitro conditions is presented. It is interesting to note that all the numerical and structural changes begin very early in cultures of different explants of *Zea mays* cv. *compositae*. As a result, a number of abnormal karyotypes emerged. The revised method of Giemsa banding includes the following steps:

- Pre-treatment: Callus tissues from the growing regions were pre-treated with a saturated aqueous solution of alpha-bromonaphthalene at 14-18 C for 4 hours, after placing them in sunlight for 1-2 hours.
- Fixation: In 1:3 acetic-alcohol at 10 C for 4-12 hours and preserved in 70% alcohol.
- Maceration: In 9:1 orcein:HCl mixture at 60 C for 5-6 min. Callus tissues were squashed in a drop of 45% acetic acid. Cover glasses were detached from the slides by immersing them in ethanol, and both slide and cover glasses were air dried.
- Destaining: In 45% acetic acid for 10-15 min. Washed in distilled water for 5-10 min and air dried.
- HCl treatment: With 5 N HCl at room temperature for 5-7 min. Washed in running distilled water for 15 to 20 min.
- Staining: With 3% Giemsa solution diluted in 1/15 M sodium or potassium phosphate buffer at pH 6.8 for 30 seconds to 1 min. The staining process was monitored by microscopic observation of the slides at frequent intervals.

After adequate staining, the slides were rinsed in distilled water and air dried before mounting in Canada balsam.

The revised method of banding showed that fixation in acetic-alcohol plays a promoting role for the development of bands. This may be due to the disruptive lesion of chromosomal protein, which is primarily responsible for band development.

In addition, experimental conditions indicate that the ionic strength of the salt solution has some significant role in band development. Therefore, it can be inferred that the acetic alcohol and concentration of HCl might dissociate certain substantial amounts of protein from the chromosomes to which the Giemsa binds and ultimately develop a band. The mechanisms involved are yet to be clarified.



Fig. 1. Giemsa banded chromosomes from callus cultures of Zea mays showing terminal, intercalary and centromeric bands.

Most of the chromosomes showed terminal, interstitial, as well as centromeric bands (Fig. 1). Such a type of banding pattern not only helped to identify the chromosomes which have been eliminated preferentially during the culture regime, but also to detect structural changes in the chromosomes.

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Tissue cultures of Zea mays x Tripsacum dactyloides

All of the experiments described in this communication were conducted with a field F1 generation of the intergeneric hybrid between Zea mays and Tripsacum dactyloides and its reciprocal crosses, obtained by María del Carmen Molina in our Institute.

In the hybrids, when T. dactyloides was used as female parent, the seeds are completely enclosed in a fruit case made up of a horny rachis and a hardened glume like Tripsacum. To overcome this barrier, the fruit cases were nicked with a scalpel and all seeds with a recognizable endosperm and well-visible signs of embryo development and health were selected for culture. Mature seeds were sterilized following the procedure of Green et al. (Crop Sci. 14:54-58, 1974). Embryos were aseptically excised from the seeds, but the residual endosperm was difficult to remove and several embryos were cultured with minor parts of endosperm attached to the embryo.

Callus was initiated and maintained on modified Murashige-Skoog culture medium (Green and Phillips, Crop Sci. 15:417-421, 1975). The medium was adjusted to pH 6.0 and then autoclaved for 20 min at 15 psi. The embryos and subsequent callus cultures were grown at 30/20 C and exposed to 12/12 hours photoperiod.

The embryos of the hybrid Z. mays x T. dactyloides and its reciprocal crosses were cultured for 30 days on medium containing 2,4-D at 0, 2, 5, 10, and 15 ppm. In the absence of 2,4-D the mature embryos, cultured with the scutellum side

downward, germinated normally and after 21 days presented abundant roots and 2-3 green leaves. However, when these plantlets were transferred directly from the in vitro condition to the soil in the greenhouse they did not survive. An intermediate subculture in vermiculite is necessary to improve the survival rate.

Figure 1

		2,4-D ppm			
		2	5	10	15
Callus initiation phase	% of callus formation	30	80	20	0
Organ formation phase	% of surviving cultures	-	100	-	-
	% of organ formation	-	-	-	-

In the presence of 2,4-D at any level, all *T. dactyloides* x *Z. mays* embryos cultured with the scutellum side upward failed to grow as callus. However, *Z. mays* x *T. dactyloides* embryos showed callus initiation dependent on the 2,4-D concentration used in the media (Fig. 1).

The growth response observed in the cultures was similar to that described by Cure and Mott (Physiol. Plant. 42:91-96, 1978) as yellowish compact, callus-like Class 3. The optimal growth rate was

observed at 5 ppm 2,4-D. Each primary culture derived from 5 ppm 2,4-D medium produced 2 subcultures. After a 30 day cycle, each subculture produced 2 to 3 more subcultures. All subcultures were maintained in 2 ppm 2,4-D medium, but the 5 ppm 2,4-D derived cultures always showed the best growth. One of the cultures exhibited localized chlorophyll regions and one green 6 mm leaf from the callus.

When these tissues were excised and subcultured in media lacking 2,4-D to promote organ formation, none of them produced roots of any type, except the 5 ppm 2,4-D derived cultures surviving in medium without 2,4-D. Moreover, callus established and maintained with 5 ppm 2,4-D did not grow when transferred to a medium free of 2,4-D.

The lack of organ formation in such cultures could be related to Green's observation (Hortsci. 12:131-134, 1977) on the relation of embryo age-size and percent of callus capable of plant regeneration in maize.

In the next year we shall work with immature embryos of these hybrids.

Miguel Angel Rapela

Factors involved in callus formation and growth of mutant maize embryos

In order to study the genotype, age-size and hormonal dependence on callus initiation and maintenance, several tissue cultures of maize were carried out utilizing immature embryos of a *floury-a* x normal red flint hybrid, its parent inbred lines and other *floury-a* inbred lines. The culture protocols were the same as Green and Phillips (Crop Sci. 15:417-421, 1975), except that cultures were maintained in the dark at 28 C.

Figure 1 shows what apparently would be an optimal embryo age-size, in which the hormonal requirements would be the lowest for a good callus growth. Although the parameter used is not strictly the same, this result is in part different from Green's (Hortsci. 12:131-134, 1977), since he found a decrease of the percent of regenerating scutellar callus with embryo age.

Figure 2 shows the influence of genotype and 2,4-D concentration on the growth of callus. These results are in agreement with Green's, in the sense that immature embryos derived from hybrids produced callus at frequencies much higher than embryos from either parent individually.

Figure 3 shows the influence of genotype and orientation of maize embryos on the growth of callus. These results are also in agreement with the observations of Green and Phillips, but different from the ones of Harms et al. (Z. Pflanzenzücht. 77:347-351, 1976). When the orientation of the scutellar side was

Figure 1

Influence of embryo age-size and 2,4-D concentration on the growth of callus from the hybrid YF 82-0343 x floury-a 82-0312. Each value = % of 20 embryos from which callus cultures were obtained.

length(mm)	age(days)	2,4-D ppm	
		1	2
1.5 - 2	15	85**	85*
2.5 - 3	17	100*	85**
3.5 - 4	20	60***	70***

* good growth
** poor growth
*** very poor growth

Figure 2

Influence of genotype and 2,4-D concentration on the growth of callus from 2 mm long embryos. Each value = % 20 embryos.

Genotype	2,4-D ppm	
	1	2
YF 82-0343	50	15
f1-a 82-0312	15	15
YF x f1-a 0343 x 0312	85	85

Figure 3

Influence of genotype and orientation of maize embryos on the culture medium on the growth of callus from 2 mm long embryos at 2 ppm 2,4-D. Each value = % of 40 embryos.

Genotype	Orientation of the scutellar side	
	downwards	upwards
f1-a 82-0312	5	20
f1-a 82-0309	30	67
f1-a 82-0301	20	60
f1-a 82-0300	25	70

upwards, all of our primary cultures exhibited a short coleoptile emergent from the proliferating callus tissue. The subsequent embryo growth was quickly suppressed and the callus growth was fast. However, when the orientation of the scutellar side was downwards, the cultures exhibited long coleoptiles from the plumule-radicle axis, and very poor callus formation.

These preliminary results suggest that callus cultures (perhaps differentiating) can be obtained from immature floury-a and +floury-a embryos. However, there is variability between genotypes in capability to initiate callus cultures. Hence, the optimal embryo age-size for excision and the hormonal requirement should be tested for each particular genotype.

Miguel Angel Rapela

Tissue cultures of a red flint maize hybrid

Tissue cultures utilizing immature embryos of a field F1 red flint hybrid, 81-719 x 81-699, obtained by Francisco Babinec, were initiated in February 1982. The culture protocols were the same as mentioned above for the floury-a immature embryos.

Immature embryos of 15, 18 and 20 days after pollination were placed with their scutellar side upwards on MS medium containing 2,4-D at 2, 5 and 10 ppm. 2 ppm 2,4-D medium and 20-day embryo age was the most successful combination to promote

callus formation. After 4 subcultures, 30 days each, more than 20 test tubes with 0.5 - 1.5 cm diameter callus were developed from each primary culture. After the fourth subculture, attempts were made to subculture callus tissues in medium free of 2,4-D to promote organ formation. This organ formation phase was carried out at 30/20 C and 12/12 hours photoperiod.

After 5 months of culturing, a calliclinal variation synthesized in three kinds of responses was observed in the cultures. Some cultures (Fig. 1) did not grow

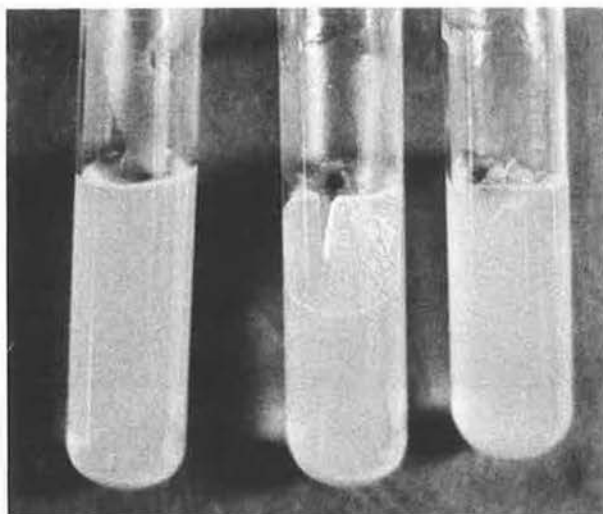


Figure 1

further during subsequent inoculation, and were brownish and usually necrotic after 60 days. No organized root vascular tissue was observed. A second kind of response (Fig. 2) was observed in the majority of the cultures, and was a kind of

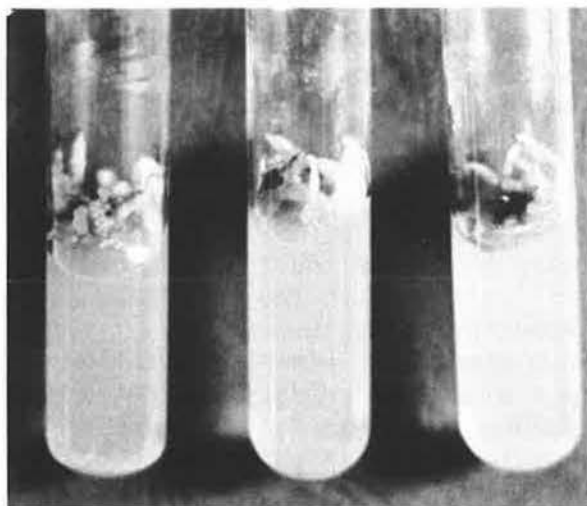


Figure 2

growth that appeared as a mixture of callus, agar roots and principally aerial roots. Both agar and aerial roots were hairless. A third kind of response (Fig. 3) was observed in 17% of the subcultures, and was characterized by a development of extensive roots with root hairs and few aerial roots. One of the cultures presented more than 40 adventitious roots with secondary and tertiary roots.

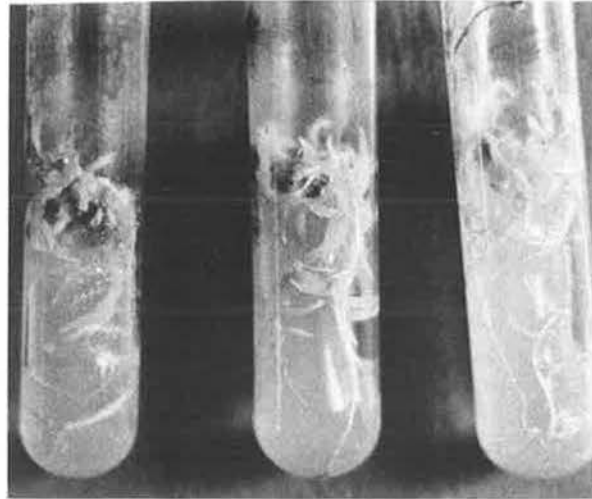


Figure 3

None of the cultures with extensive root development produced shoots at this time. Such observation is in agreement with Green and Phillips (Crop Sci. 15:417-21, 1975). Structures resembling organized scutellum were not observed in the cultures. Apparently, the root development appeared from the mass callus.

Attempts were made to characterize electrophoretically the three kinds of responses by conventional PAGE (Davis) and SDS-PAGE (Laemmli). However, the low protein content of the tissues was a difficult risk in our attempts. As much as the equivalent of 250 mg fresh weight of tissues was loaded in each acrylamide tube without obtaining successful visualization of the protein bands with Coomassie Blue R-250. However, it was evident that the SDS-PAGE patterns derived from each kind of culture were different and distinguishable. More than 25 bands were observed in the SDS-PAGE patterns of the cultures of Figure 3, while only 5 to 10 bands were observed in the SDS-PAGE patterns of the cultures of Figure 1.

Miguel Angel Rapela

The homoserine dehydrogenases of floury-a maize

Homoserine dehydrogenase (HSDH) is the third enzyme in the pathway leading to threonine, methionine and isoleucine in the aspartate family of amino acids. In maize, HSDH is feedback-controlled by threonine.

HSDH was extracted from shoot and internodes of floury-a and normal BP etiolated seedlings 3 days old. The extraction procedure of Bryan and Lochner (Plant Physiol. 68:1400-1405, 1981) was used but DTE was replaced by 5.0 mM of 2-mercaptoethanol. Extraction and all subsequent operations were carried out at 5 C. Weighed tissues were ground using a mortar and pestle. Homogenates were filtered through two layers of cheesecloth and centrifuged at 18,000 xg for 30 min. Crude supernatants were analyzed by discontinuous gel electrophoresis (Davies),

with the HSDH activity being located on the gels by the nitroblue tetrazolium dye precipitation procedure of Matthews et al. (Plant Physiol. 55:991-998, 1975).

Electrophoretic analysis of HSDH from extracts of 72-hour-old maize shoot and internodes indicated the presence of 3 to 4 enzyme forms (Fig. 1). Tissue-specific and genotype-specific differences in the proportion of the enzymes have

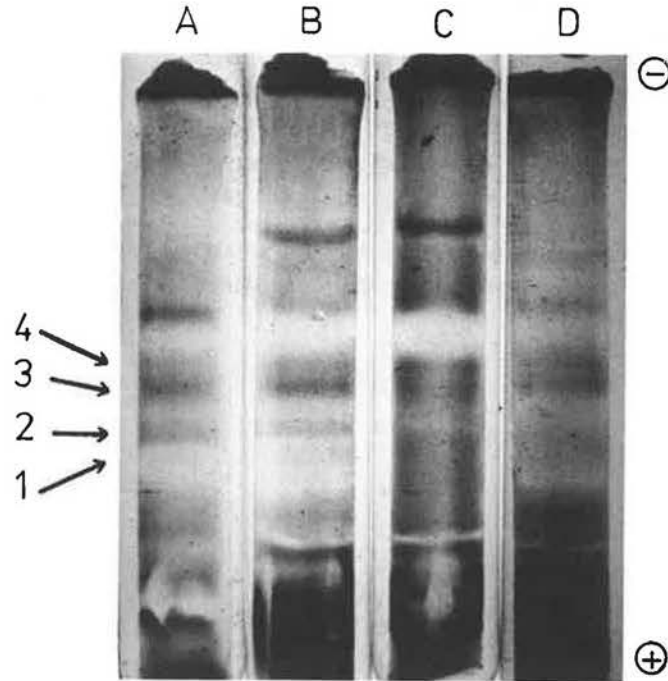


Figure 1. Gel electrophoretic analysis of HSDH. A: floury-a shoots; B: floury-a internodes; C: BP shoots; D: BP internodes. Bands have been numbered 1 to 4 in order of decreasing mobility. These bands were not detected when duplicate gels were incubated in the absence of homoserine after electrophoresis. A to D 5% acrylamide gels. Equal amounts of protein were applied to each gel.

been observed. Bands 3 and 4 were the major enzyme forms independent of the tissue and genotype. However, the ratio of Bands 3 and 4 has been changed from about 2:1 in the floury-a crude extracts (A-B) to 1:1 in the BP crude extracts (C-D). Band 1, the HSDH threonine resistant form, was difficult to localize in the BP crude extracts. However, in the floury-a extracts, Band 1 appeared as a faint band. Band 2 was clearly localized in all floury-a electrophoretic patterns independent of the tissue (root tissue, shoot tissue, whole kernel, internodes) and the age of seedlings. On the other hand, Band 2 was very difficult to localize and differentiate from Band 1 in the BP crude extracts.

These preliminary results suggest that changes in the HSDH are not only both tissue and species specific, as has been previously proposed, but also could be genotype-specific. These results could also explain in part some of the unusual responses of the floury-a material to lysine-threonine feedback regulation.

Miguel Angel Rapela

Cytogenetic study of a tetraploid hybrid between *Zea diploperennis* and *Zea perennis*

In April 1980, a hybrid between *Zea diploperennis* ($2n=20$) and *Zea perennis* ($2n=40$) was obtained. The hybrids are triploid, perennial and sterile. One plot was different from the others because of the morphological resemblance to *Zea diploperennis*. It had 40 chromosomes, so a possible explanation was suggested. The hybrid probably could have originated from the crossing of one unreduced gamete of *Zea diploperennis* with one normal gamete of *Zea perennis*.

Whatever mechanism could have originated this hybrid, it has a chromosome number ($2n=40$) with two sets of each of their parents so the F1 and F2 are perennial and fertile. The meiotic configuration of F1 plants was analyzed, and the results of examinations of 184 cells in diakinesis are summarized in Table 1. As the chromosomes had been paired totally or partially there were almost no monovalents and

Table 1: Meiotic configurations of a tetraploid hybrid between *Zea diploperennis* and *Zea perennis*.

Cells number	I	II	III	IV	%
62		10		5	33.69
38		12		4	20.65
29		14		3	15.76
20		8		6	10.86
7		16		2	3.80
4		18		1	2.18
2		6		7	1.08
5	2	11		4	2.71
4	2	13		3	2.18
4	1	10	1	4	2.18
4	2	9		5	2.18
3	1	12	1	3	1.63
2	1	14	1	2	1.08
184	0.18	10.74	0.046	4.03	

trivalents. That is one of the most remarkable differences between this hybrid and the triploid one (which has originated from the same parents), since it had a high percentage of trivalents and monovalents and a most frequent meiotic configuration of $5III + 5II + 5I$. The meiotic configurations and the average of bivalents and tetravalents were similar to those observed in a clone of *Zea perennis*. That is why, according to its chromosome-set, it was similar to *Zea perennis*, but considering the morphology it resembled *Zea diploperennis* (female parent).

At anaphase I, 20 chromosomes migrated towards each pole in 90% of the cells. In the remaining 10%, different numbers of chromosomes migrated towards each pole. Other abnormalities observed were lagging chromosomes, and exceptionally, chromatid bridges. The tetrads were normal. The percentage of fertile pollen was 56% and fertile seed was 80%.

From this study, it is important to remark that: (1) all the triploid hybrids of *Zea perennis* and *Zea diploperennis* are sterile due to the high number of monovalents and trivalents; (2) for the first time, a tetraploid fertile hybrid from crossing *Zea diploperennis* with *Zea perennis* was obtained; (3) apparently, the chromosome behavior in the hybrids varies if it is a triploid hybrid or a tetraploid one; (4) the chromosome studies of *Zea diploperennis*, *Zea perennis* and their hybrids provide enough evidence to demonstrate that *Zea perennis* is not an auto-tetraploid derivative of *Zea diploperennis*.

María del Carmen Molina

Cytogenetic study of the hybrid between *Zea mays* and *Zea diploperennis*

In 1980, a population of *Zea mays* "Colorado Klein" was successfully crossed with *Zea diploperennis*. Both species hybridize readily, and the F1 hybrid is not only fertile and vigorous but also preferably annual or biannual, except for 12% of the plants which are perennial. The cytological analysis of the hybrid indicated that the chromosomes of such species paired totally, while occasionally partial pairing and some deficiencies were observed.

In the counting of 121 cells in diakinesis, the meiotic configurations most frequently found were: 10II in 71.9% of the cells, 9II + 2I in 23.14%; 8II + 4I in 4.13% and 7II + 6I in 0.83%. The average of chiasmata was 15% per cell. At anaphase, 10 chromosomes migrated towards each pole, but occasionally 11 chromosomes to one pole and 9 of them to the other, and in rare cases, lagging chromosomes. The percentage of fertile pollen was 92%.

The size of the knobs exceeded considerably their parents, especially in the 4 chromosomes with terminal knobs provided by *Zea diploperennis*. In the hybrid, some of the chromosomes given by the parental species could be differentiated due to the position of knobs (subterminal in *Zea mays* and terminal in *Zea diploperennis*). However, it is not possible to determine the origin of the chromosomes without knobs.

It can be concluded that *Zea mays* markedly differs from *Zea diploperennis* in its morphology but not in its chromosome set, since their chromosomes pair totally, getting a fertile offspring. Thus, the chromosomes of such species are considered to be homologous or homeologous.

Teresa Pilar Rosales and María del Carmen Molina

Influence of annual teosinte cytoplasm on nuclear DNA content of maize inbreds

As previously communicated (MNL 55:51, 1981), the DNA contents in isogenic maize lines have been studied (*Zea mays* genotype). The test was practiced upon interphase nuclei of tapetum cells. Those lines possess the following constitution: Maize nucleus in its corresponding cytoplasm (NZZ), and maize nucleus in the cytoplasm of annual teosinte (NZE). A line was also analyzed with maize nucleus recuperated from annual teosinte cytoplasm to maize cytoplasm (NZRE) (Mazoti, Rev. Inv. Agr. 8:175, 1954). The three previously mentioned lines present highly significant differences for DNA contents, as can be observed in Table 1.

Table 1: Differences between mean of NZZ, NZE and NZRE lines for DNA content.

line	Mean DNA (A.U.)
NZZ (83)*	27.8 ^a **
NZE (67)	17.8 ^b
NZRE(77)	22.3 ^c

*-- number between parenthesis indicate number of nuclei studied.

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

Owing to the fact that an interphase nucleus population has been analyzed for each line, the DNA values would normally have to vary between 2C and 4C pre- and post-synthesis, respectively.

The distribution of frequencies for the DNA contents of the NZZ line adopts the classical bimodal distribution for interphase nuclei (Fig. 1). The first mode (with a higher 2C frequency) represents DNA pre-synthesis and the second mode (with

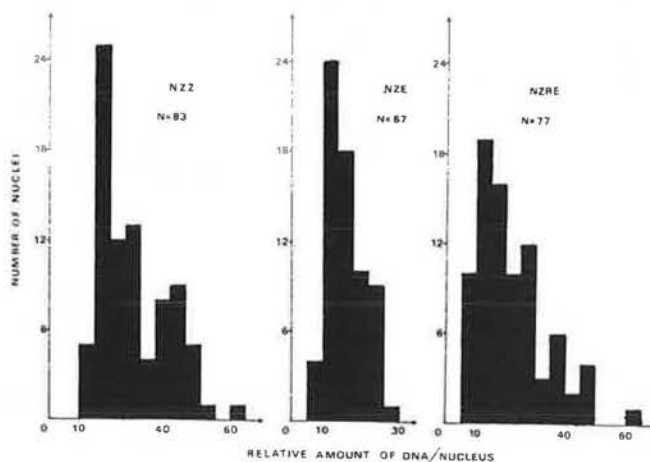


Fig. 1: Frequency distribution of the relative amount of DNA per nucleus in isogenic lines (*Zea mays* genotype) measured by Feulgen-stain microspectrophotometry. NZZ-- maize nucleus in its corresponding cytoplasm, NZE-- maize nucleus in the cytoplasm of annual teosinte and NZRE-- maize nucleus recuperated from annual teosinte cytoplasm to maize cytoplasm. N-- number of nuclei measured.

a lower 4C frequency) represents DNA post-synthesis. In the NZE line, the 2C nucleus proportion is maintained the same as in the NZZ line, and there are no 4C nuclei to be found. The frequency distribution for DNA contents in the NZRE line is characterized by the appearance of 4C nuclei.

In accordance with the results obtained, it is interesting to point out the imbalance manifested in the proportion of nuclei present in each period (pre- and post-synthesis periods), when the same maize nucleus is subject to different cytoplasm. The DNA distribution is normal for those nuclei that are found in their corresponding cytoplasm (NZZ), and varies when the nuclei are present in a foreign cytoplasm like the one of annual teosinte (NZE). Finally, it is interesting to point out how a normal tendency in the nucleus distribution is manifested, when transferred from the foreign cytoplasm to its original cytoplasm; that is to say, when rescued to their normal cytoplasm.

As the nucleus analysis was made on a specimen taken at random, the distribution according to the period must be the same in all three lines studied. Therefore, the differences in the average DNA content between lines must be attributed to the different nucleus frequency in each of the periods (G1 and G2), and probably there consequently exists a cytoplasmic control in the nucleus frequencies corresponding to each period.

Ida Graciela Palacios

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Perennial teosinte-Gaspé hybrids: Inheritance of the number of leaves

We have analyzed inheritance of the number of leaves in hybrids between perennial teosinte (Euchlaena perennis = Zea perennis) and Gaspé (Zea mays), and we have also tested the relationship between the number of leaves, growing habits and evolutive cycle. Gaspé is extremely precocious and has a low leaf number, whereas perennial teosinte has a very long cycle and has a high number of leaves (see Table 1). In the F₁, 88 plants, and in the F₂ and F₃, approximately 400

Table 1: Number of leaves (NL), days to tassel (T), days to silking (S) and days to pollen (P) in perennial teosinte (Zp), Gaspé (Gs) and its F₁, F₂ and F₃ populations.

	NL	T	S	P
Zp	15.3 ± 2.0	193	—	—
Gs	6.5 ± 0.7	29.9 ± 2.0	32.7 ± 5.5	36.5 ± 3.8
F ₁	10.8 ± 0.9	61.6 ± 7.2	—	—
F ₂	10.8 ± 2.4	89.9 ± 21.4	115.9 ± 21.3	—
F ₃	10.2 ± 2.8	86.1 ± 19.6	108.6 ± 22.3	101.9 ± 20.5

plants were studied. The number of leaves was taken from two stalks from plants selected at random, and the evolutive cycle was measured in days to tassel, days to silking and days to pollen.

Figure 1 shows that the frequency distribution of leaf number variation almost reached paternal extremes in the segregating generations. The average number of

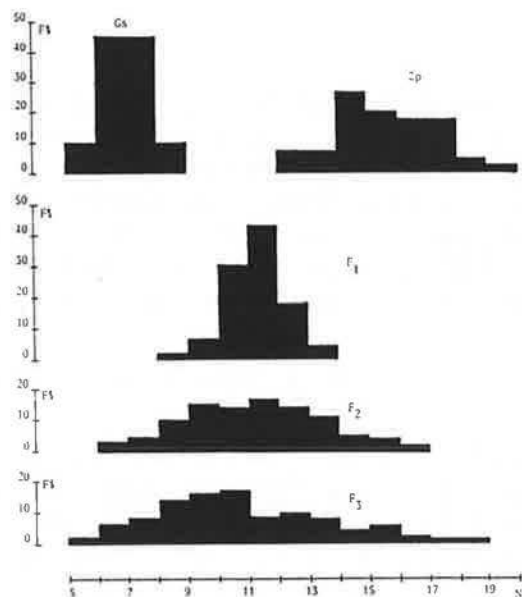


Figure 1. Frequency distributions for the number of leaves (NL) in perennial teosinte (ZP), Gaspé (Gs) and its F₁, F₂ and F₃ populations.

leaves found in F₁, F₂ and F₃ did not differ significantly from the theoretical mean (10.9) of its progenitors. The calculated inheritance is 0.71, and the number of leaves would apparently be controlled by very few genes of additive effect. In the F₂ and F₃ populations, the number of leaves is significantly correlated with the evolutive cycle (Table 2). As to growing habits in populations F₂ and F₃, the

Table 2: Correlation coefficients between number of leaves (NL) and days to tassel (T), days to silking (S) and days to pollen (P). **-- significant at 1% level.

Population	NL vs. T	NL vs. S	NL vs. P
F ₂	0.46**	0.49**	—
F ₃	0.54**	0.60**	0.58**

annual plants have a significantly lower number of leaves than the bi-annuals (Table 3).

Table 3: Number of leaves and growth habit in F₂ and F₃ populations.

Growth habit	Population	
	F ₂	F ₃
Annual	10.2 ^a (*)	9.8 ^a
Biannual (presumably perennial)	11.0 ^b	10.4 ^b

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

It can be deduced that the number of leaves associates as much with the evolutive cycle as with the growing habits. Perhaps the characteristics studied are conditioned by the same genes, or by different genes strongly linked in the same chromosomes. Nevertheless, we have found infrequently the recombination of characters in a segregating population, that is to say, precocious plants with high number of leaves and long cycle plants with low number of leaves. Therefore, it is probable that those characters are conditioned by linked genes more than by the same genes.

Jorge Luis Magoja and Gabriela Nora Benito

Perennial teosinte-Gaspé hybrids: Inheritance of prolificity

Hybrids between perennial teosinte (*Zea perennis*) and maize (Gaspé) are extremely heterotic, especially owing to the high prolificity manifested (MNL 56: 104, 1982). We have measured the prolificity of hybrids and their progenitors by counting the number of productive nodes (PN), number of ears in the uppermost node (EUN) and number of ears per tiller (E/T), as shown in Table 1. Figures 1, 2 and 3 show the frequency distribution for the characters studied.

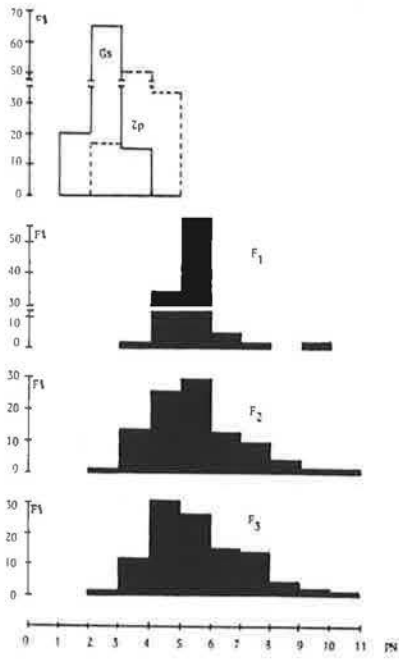


Fig. 1

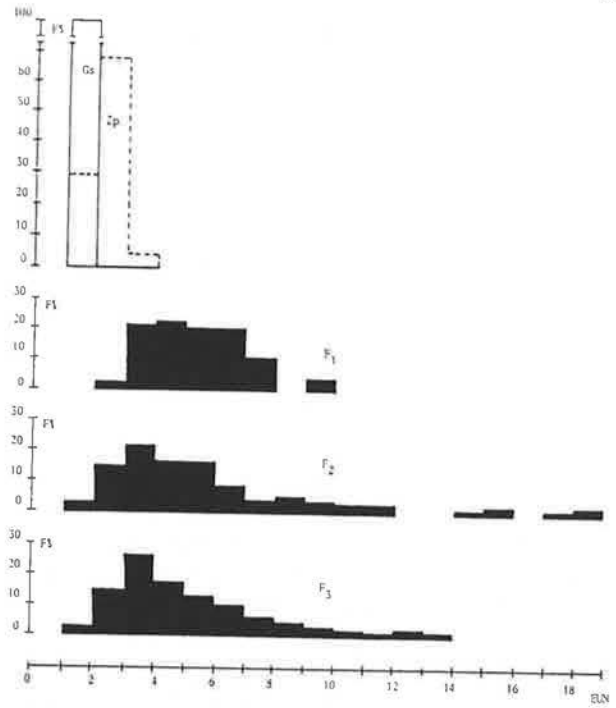


Fig. 2

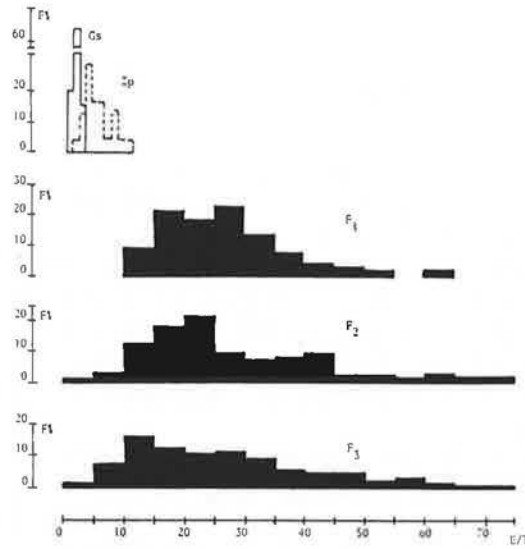


Fig. 3

Figs. 1-3. Frequency distributions in perennial teasinte (Zp), Gaspé (Gs) and its F1, F2 and F3 populations. Fig. 1: Number of productive nodes (PN). Fig. 2: Number of ears in the uppermost node (EUN). Fig. 3: Number of ears per tiller (E/T).

Table 1: Number of productive nodes (PN), number of ears in the uppermost node (EUN) and number of ears per tiller (E/T) in perennial teosinte (Zp), Gaspé (Gs) and its F₁, F₂ and F₃ populations.

	PN	EUN	E/T
Zp	3.2 ± 0.7	1.8 ± 0.5	5.3 ± 2.0
Gs	1.9 ± 0.6	1.0 ± 0.0	1.9 ± 0.6
F ₁	4.7 ± 0.8	4.8 ± 1.6	25.7 ± 9.5
F ₂	5.0 ± 1.4	5.0 ± 3.3	25.9 ± 13.1
F ₃	4.9 ± 1.4	4.6 ± 2.6	26.1 ± 15.6

All prolificacy values significantly exceeded the progenitors' theoretical mean, not only in F₁, but in F₂ and F₃. Prolificacy values stand out as they do not decrease in F₂ and F₃ with respect to those of F₁. This perhaps is owing to some type of preferential selection of prolific genotypes, since many seeds forming these hybrids are not viable.

There is a displacement of the frequency distribution of prolificacy characters towards the right of the progenitors. This type of distribution clearly represents a type of heterotic inheritance.

From the results obtained, inheritance values were exceeding 80% in each case. The hybrids' high prolificacy between perennial teosinte and maize, as a heterosis expression, contrasts with those of its progenitors, since normally maize is not prolific and teosinte is not highly prolific either. Consequently, the perennial teosinte germplasm may become valuable in introducing in maize additional heterosis, and so improving the productivity of cultivation.

Jorge Luis Magoja and Gabriela Nora Benito

Quantitative morphological differences between *Tripsacum dactyloides* and its F₁ hybrids with maize

Hybrid F₁'s between maize and *Tripsacum* are very like *Tripsacum*, although they have a series of characteristics (pointed out by different authors) which make it possible to distinguish them from *Tripsacum*. It is important to be able to distinguish true hybrids from false ones, as they are very frequently obtained when pollinating maize with pollen of *Tripsacum* (patroclinous plants). If the cross is made using *Tripsacum* as female parent, matroclinous plants may be obtained. In order to distinguish *Tripsacum* in a quantitative manner from its hybrids with maize, we have made a series of measurements in inflorescences (Table 1).

Determinations were made on *Tripsacum dactyloides* (2n=72) and on hybrids which we had obtained in 1979 crossing an inbred line of maize carrying a floury-2 gene with *Tripsacum*. There are highly significant differences for the inflorescence characters. The number of branches (NB) in the terminal inflorescences is higher in hybrids than in *Tripsacum*. Hybrids have terminal inflorescences with a shorter rachis and a lower number of female spikelets. The width of fruit cases in hybrids (FCW) is significantly higher, and there is a lower number of fruit cases (FCN). In the lateral inflorescences the hybrid has a lower number of fruit cases and has less male (ML) and female (FL) length than *Tripsacum*. Also, as in terminal inflorescences, the hybrid has wider fruit cases in lateral inflorescences.

Table 1: Differences between means of *Tripsacum dactyloides* and the F₁ (maize x *Tripsacum dactyloides*) NS-- no significant; *-- significant at 5% level; ***-- significant at 0.1% level.

	Td				Zm x Td				t	sig.
	Mean	range	S ²	N	Mean	range	S ²	N		
<u>Terminal inflorescences</u>										
NB(1)	2.9	2-4	0.37	22	4.7	3-8	0.83	14	-7.11	***
ML(cm)	15.4	10.5-20.7	5.79	64	14.6	9.0-20.3	7.58	66	1.76	NS
FL(cm)	4.2	1.7-7.4	0.98	64	1.6	0-4.9	1.45	66	15.85	***
FCN	5.2	2-8	1.43	64	2.1	0-6	2.72	66	12.21	***
FCL(mm)	8.1	6.2-11.7	0.72	64	7.9	5.6-11.5	1.46	53	1.05	NS
FCW(mm)	5.4	4.7-6.4	0.15	28	6.8	4.5-9	0.96	53	-7.24	***
<u>Lateral inflorescences</u>										
ML(cm)	13.2	9.2-18.2	4.04	23	11.7	6.8-15.3	6.59	26	2.26	*
FL(cm)	8.4	7.6-9.2	0.19	23	7.2	5.5-9.3	0.94	26	5.44	***
FCN	11.0	10-11	0.04	27	9.8	8-13	2.00	26	4.03	***
FCL(mm)	7.6	6.9-8.4	0.15	23	7.4	6.7-8.8	0.22	26	1.60	NS
FCW(mm)	4.8	4.3-5.4	0.08	23	9.2	7.5-11	0.56	26	-26.47	***

(1): NB-- number of branches; ML-- male length; FL-- female length,
FCN-- number of fruit cases; FCL-- fruit case length;FCW-- fruit case width.

From these results it can be deduced that the maize germplasm contribution shows up in hybrid terminal inflorescences with a high number of branches, predominating in male spikelets. In the lateral inflorescences the hybrid has a tendency to differ less in male inflorescences. We have also seen that approximately 13% of the female spikelets in hybrids are arranged in pairs, as in maize.

Likewise, hybrids have a longer period of vegetation and flowering than *Tripsacum*. In accordance with the results presented herein and those reported by other authors, the principal characteristics which distinguish the hybrid from *Tripsacum* are: (1) the hybrid has a higher number of branches in the terminal inflorescences; (2) a tendency in the hybrid to separate sexes in different inflorescences; (3) the absence or reduction in the number of female spikelets in terminal inflorescences in the hybrid; (4) terminal inflorescences in the hybrid with characteristics of the corn tassel or teosinte tassel; (5) in the hybrid, silks emerge before the inflorescence has emerged from the leaf sheath; (6) longer stigmas in the hybrid and the two branches of the style fused a greater distance than in *Tripsacum*; (7) fruit cases in the hybrid wider than in *Tripsacum*; (8) the hybrid has both single and paired female spikelets; (9) longer vegetative and flowering time in the hybrid; (10) a higher number of tillers in the hybrid; (11) larger size of leaves and stalks in the hybrid; and (12) full male sterility and high female sterility in the hybrid.

Jorge Luis Magoja and Ida Graciela Palacios

Cytological observations in F1 hybrids between maize and *Tripsacum dactyloides*

In the cross between *Zea mays* (2n=20) and *Tripsacum dactyloides* (2n=72), observations in diakinesis have determined that the 36 *Tripsacum* chromosomes pair forming 18 bivalents, while those of maize form 0 to 5 pairs. In hybrids, the greatest frequency possess 23 pairs of chromosomes (see Figure 1).



Fig. 1: Diagram showing the 23 pairs of chromosomes in diakinesis corresponding to hybrid between maize and *Tripsacum dactyloides*. The 5 bivalents (Z) presumably represent maize chromosomes.

Ten (10) pairs of chromosomes are formed during meiosis when maize conducts itself as a diploid. While in haploid cells there are only associations from 0 to 5, such associations are brought about between non-homologous parts of chromosomes (so-called freak "folding back," considered as pairing between the arms of the same chromosomes by Chase, Bot. Rev. 35:117, 1969). In pachytene observations, pairing of segments has not been found between maize chromosomes and those of *Tripsacum*. The forming of bivalents among maize chromosomes on one side and *Tripsacum* on the other agrees with the results obtained by other authors (Harlan et al., Science 167:1247, 1970), who manifest that such pairing frequency is influenced as much by the maize stock as by that of the *Tripsacum* used.

Ida Graciela Palacios and Jorge Luis Magoja

Endosperm structure of *Tripsacum dactyloides* (2n=72)

Optical microscopy shows that the storage protein of corn endosperm is deposited within the cells as two distinct components, globular bodies and an amorphous matrix in which the granules are embedded. Bates et al. (Cereal Chem. 58:138, 1981) reported that in *Tripsacum dactyloides* (2n=36), starch grains and protein bodies were only one-tenth as large as those of maize, and ranged from 1.3 to 1.8 μm and from 0.05 to 0.12 μm respectively. In accordance with the results of Bates et al., *Tripsacum dactyloides* (2n=36) has no visible protein bodies at the light microscope level. The purpose of the present paper is to make known the experimental results which we have obtained in the study of *Tripsacum dactyloides* (2n=72) endosperm structure. Mature kernels were thin sectioned, destarched with alpha-amylase, and endosperm tissue studied using optical microscopy at 700 x magnification. The procedure employed for observation of protein bodies was the same as the one described by Wolf and Khoo (Stain Tech. 45:277, 1970). Protein

body size and starch grain size were measured in the third or fourth cell layer below the aleurone layer, because the protein bodies decreased in size from the aleurone layer inward.

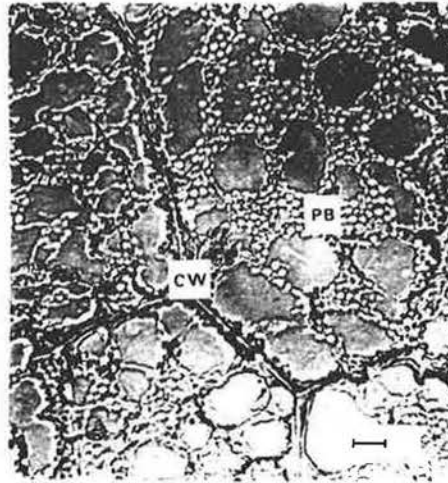


Figure 1: Destarched 3-4 μ m thick section of *Tripsacum dactyloides* endosperm showing protein bodies (PB). Dark lines are cell walls (CW)- (bar = 2 μ m).

Figure 1 shows that *Tripsacum* has visible protein bodies. We found that *Tripsacum* protein bodies ranged from 0.4 to 0.8 μ m in diameter (average 0.6 μ m) and that starch grains ranged from 3.4 to 4.5 μ m in diameter (average 4.0 μ m). The results presented here indicate that starch grains and protein bodies of *Tripsacum dactyloides* ($2n=72$) were only one-half as large as those of maize. Our results are significantly different from those reported by Bates et al., therefore it is probable that the ploidy level in *Tripsacum dactyloides* conditions different endosperm structures in the same species.

Jorge Luis Magoja and Luis Máximo Bertoia

Effect of de*-7601 on seed proteins during grain development

The genetic control of seed proteins produced by a spontaneous defective kernel mutant was previously reported (MNL 56:108, 1982). The variation during grain development of the patterns of soluble proteins and lipoproteins was studied in the red flint corn line WK-01 and its isogenic version of defective kernel, de*-7601. By electrophoresis in polyacrylamide gel, the soluble proteins of immature whole kernels were separated at 18, 23 and 28 days after pollination. The defective phenotype is perfectly distinguishable from normal after 14-16 days. As with the mature kernel (MNL 56), de*-7601 conditioned a blockage of certain soluble protein components, and for lipoproteins, new components (Figs. 1 and 2).

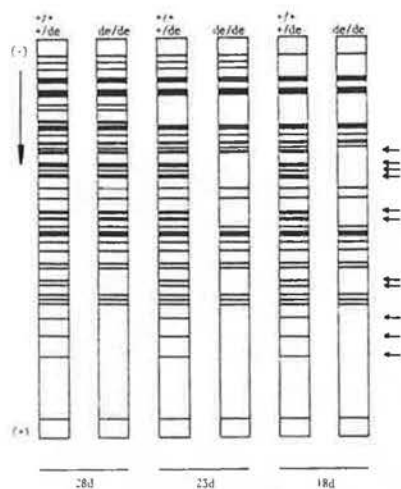


Figure 1: Polyacrylamide gel electrophoretic patterns of soluble whole kernel proteins during grain development. 28 d, 23 d, 18 d--days after pollination; +-- normal; de-- defective.

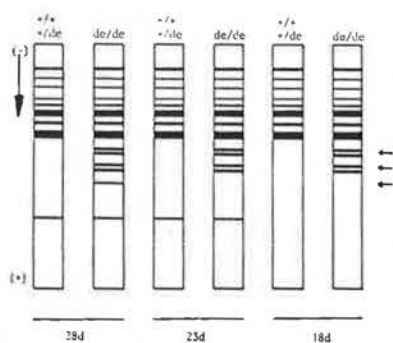


Figure 2: Polyacrylamide gel electrophoretic patterns of whole kernel lipoproteins during grain development. 28 d, 23 d, 18 d-- days after pollination; +-- normal; de-- defective.

From the results obtained it can be deduced that:

1. There are some polypeptides that, no matter the development stage, never appear in the soluble proteins of de*-7601 grains. This can be interpreted as a blockage of the structural genes which codify those components by one or more regulatory genes, or the expression of null alleles of the corresponding structural gene affected by mutation.
2. Some molecular components of soluble proteins have their expression blocked during the first developmental stages in defective kernels, but they appear in more advanced development stages. In this case, it is obvious that the structural gene/genes responsible for the codification of those components have an active allele, but one or more regulatory genes represses the expression with different intensity according to the developmental stage. The regulatory gene conditions a delay in the expression of certain polypeptides in defective grains.
3. The polypeptide patterns vary in normal kernels, as in defective ones, as time goes by during the development of the kernel. Generally, the pattern becomes more complex.
4. For soluble proteins the differences between normal patterns and defective ones decrease as maturity is reached, while for lipoproteins there is a greater pattern difference in the most advanced stages of the kernel development.

It can be said that in general the early blockage in the expression of certain genes which codify some components of the defective kernel proteins and retard expression of others, must possibly be the cause of tardiness in the development of the defective kernel, which brings about a lethal character. This may be interpreted as a phenomenon conditioned by a regulatory gene. Nevertheless, a particular anomaly occurs with lipoproteins of developing kernels similar to that previously reported (MNL 56:108, 1982). In fact, the lipoprotein patterns of defective kernels do not show fewer bands than normal, but have new fast bands. So as to interpret this phenomenon, electrophoretic runs for lipoproteins of normal kernels at a high sample concentration were carried out.

These experiments demonstrated that fast bands are also present in normal kernels, but in a low proportion, and therefore are not distinguishable in an electrophoresis run with normal protein concentration. These results suggest that the excess of those lipoprotein components in the defectives might also be the cause of the defective condition. This phenomenon might be interpreted as the uncontrolled synthesis of those components as a consequence of the mutation of the regulatory locus, which in normal kernels regulated the synthesized quantity. In the defectives, where such regulation is not exercised over the structural loci which codifies those lipoproteins, they are transcribed uncontrollably. All these results tend to strengthen the tests obtained to support the hypothesis that the locus de*-7601 is a regulatory gene that provokes dramatic alterations in the maize seed, becoming a lethal character.

Jorge Luis Magoja and Angel Alberto Nivio

Puna maize: Germplasm of high protein quality with hard endosperm

From a maize population native to the Puna (Jujuy, Argentina) and cultivated in Llavallol, several S1 lines were obtained by selfing. Whole kernels of thirty of them were analyzed for protein content (WKP), lysine content (WKL) and tryptophan content (WKT); the endosperm was also analyzed for protein content (EP), lysine content (EL) and tryptophan content (ET).

This maize population named Puna, as mentioned by Vorano (IDIA 32:13, 1976), consists of early plants cultivated at 3500 m above sea-level, and possesses the interesting characteristics of having low thermic and hydric requirements and very high capacity of mesocotyl elongation. From the lines already studied, the greater part (25) have hard endosperm, and a coat of variable thickness of hard endosperm is found in all the kernel periphery. Four lines are of the dent type and only one has totally floury endosperm.

Table 1 is a summary of the results of the chemical analysis, wherein variation can be observed in lysine as well as in tryptophan contents, the highest values of these amino acids being approximately similar to those conditioned by mutants of

Table 1: Protein, lysine, and tryptophan levels in Puna S₁ lines.

	defatted whole kernel			defatted endosperm		
	protein (%)	lysine (g/100g prot.)	tryptophan (g/100g prot.)	protein (%)	lysine (g/100g prot.)	tryptophan (g/100g prot.)
Mean	14.4	3.3	0.7	13.3	2.7	0.5
S D	1.5	0.3	0.1	1.7	0.3	0.1
range	10.8-17.7	2.8-4.0	0.6-0.9	9.8-17.1	2.1-3.3	0.3-0.8

high protein quality. What is really important is the fact that high levels of lysine and tryptophan are not associated with floury endosperm structure. Some S₁ lines with high lysine and tryptophan levels have been selected, with a hard endosperm, as shown in Figure 1.

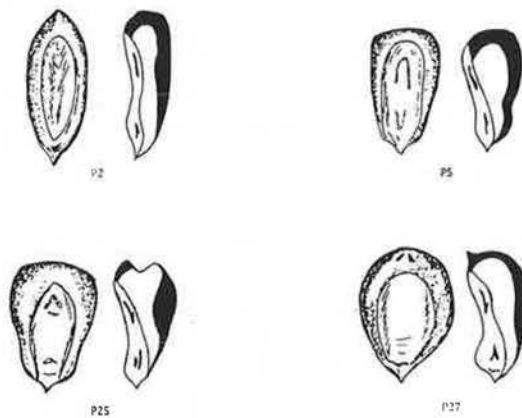


Figure 1: Kernels of Puna lines, black zones represent hard endosperm. These four lines has an average of 3.9 g lysine/100 g protein on whole kernel basis.

The biochemical characteristics already studied correlate with one another as stipulated in Table 2. From these results it can be deduced that the protein quality (lysine and tryptophan levels) is inversely associated with the protein content. There is a significant association between protein and lysine. We are actually working on the selection of high-quality protein and hard endosperm lines, and we have carried out crosses with normal lines in order to study the inheritance of that character.

Table 2: Correlation coefficients (r) between protein, lysine and tryptophan.
 **-- significant at 1% level.

	r		r
WKP vs. WKL	-0.838**	WKL vs. ET	0.471**
WKP vs. WKT	-0.511**	WKT vs. EP	-0.323
WKP vs. EP	0.848**	WKT vs. EL	0.452
WKP vs. EL	-0.774**	WKT vs. ET	0.525
WKP vs. ET	-0.480**	EP vs. EL	-0.812**
WKL vs. WKT	0.556**	EP vs. ET	-0.361
WKL vs. EP	-0.743**	EL vs. ET	0.493**
WKL vs. EL	0.782**		

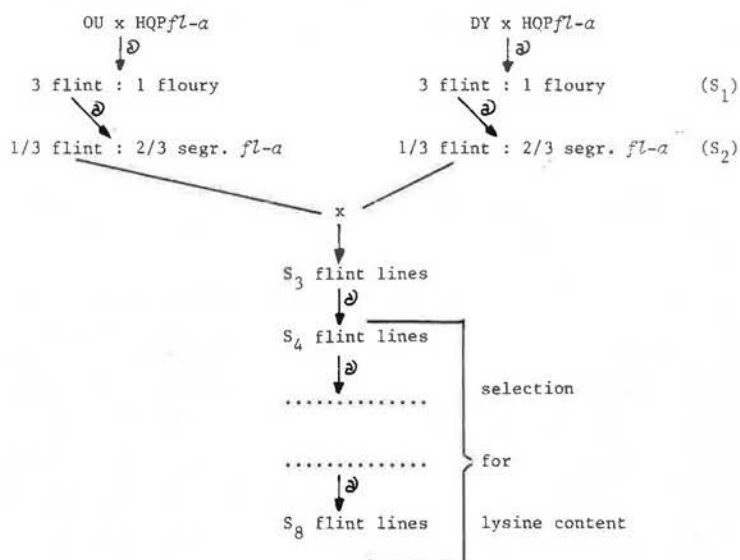
This is the third time we communicate the findings of maize of hard endosperm, which are not related to each other and bear high-quality protein. The first communication was by Magoja (MNL 52:37, 1978), the second by Magoja (Communic. Direc. Inv. U.N.L.Z. 1:2, 1978) and the third presented herein. The results obtained up to the present allow us to suppose that maize protein quality may be improved without modifying the endosperm's structure and without the use of floury mutants; and that the Puna maize studied constitutes a genetic reserve which may be employed to improve the lysine and tryptophan levels of maize endosperm, without affecting its normal corneous phenotype.

Angel Alberto Nivio and Jorge Luis Magoja

High-quality protein maize with normal genotype: Results after eight generations of selection

It has been possible to obtain maize lines of the red flint type with a high protein quality. These lines, as previously reported (MNL 52:37, 1978; MNL 55:66, 1981 and MNL 56:110, 1982), do not carry the floury mutation which modifies endosperm structure.

Figure 1: Genealogy of high-quality protein lines with normal genotype.



A corn line carrying the floury-a gene and with high protein quality (HQP fl-a) was crossed with two normal lines (red flint)OU and DY (Fig. 1). Some red flint

ears were discovered in S2 progeny, with a high level of endosperm tryptophan (MNL 52:37, 1978), which led us to think that the protein quality could be improved without floury-a. Subsequent tests (Magoja et al., Mendeliana 5 (2):71-80, 1982) show that these normal phenotype lines are also "normal genotype" because in their genetic background they do not carry any floury mutant. In order to accumulate genes conditioning high protein quality, S2 plants of different lineage were crossed: one of the progeny crossing with OU and another of the progeny crossing with DY. Up to the S3 generation, selection was based upon the amount of endosperm tryptophan. As of S4, selection was based on the amount of lysine (g lysine per 100 g of endosperm protein). The general methodology was to constantly inbreed the progeny of the initial crosses and analyze protein and lysine contents in the segregating generations. In each generation, between 50 and 100 ears were analyzed for protein and lysine contents. Those ears were always obtained by selfing, and the following generation always sprung from the best ears of the former generation, for the lysine content of the endosperm protein.

The progress achieved from the S4 and S8 generations is represented in Figure 2. In Table 1 a summary of the lysine and protein levels during the last selection generation is shown. As can be seen, from S6, the improvement progress with

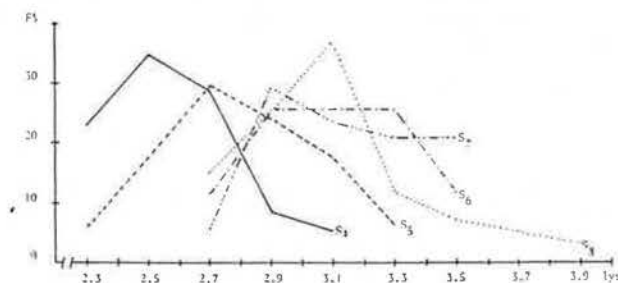


Figure 2: Frequency distributions for endosperm lysine content (g/100 g protein) in S₄-S₈ generations.

Table 1: Protein and lysine levels in S₄ to S₈ generations of high-quality protein lines with normal genotype.

Generation	defatted endosperm					
	protein (percent)			lysine (g/100 g protein)		
	Average	S D	range	Average	S D	range
S ₄	12.8 ^a (*)	1.2	10.3-14.8	2.6 ^a	0.2	2.2-3.1
S ₅	11.1 ^b	1.0	9.8-13.3	2.8 ^b	0.3	2.4-3.2
S ₆	10.5 ^c	1.0	8.5-13.1	3.0 ^c	0.3	2.6-3.6
S ₇	10.7 ^c	0.9	8.3-12.1	3.1 ^c	0.3	2.7-3.6
S ₈	10.8 ^c	1.1	8.3-13.1	3.1 ^c	0.3	2.7-4.0

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

respect to the content of lysine is not statistically important, although it should be pointed out that, nevertheless, the increase of frequency of lines with high lysine level continues. As a consequence of the selection for high lysine level in the endosperm protein, the endosperm protein level has diminished, owing to the fact that those features are inversely correlated, as shown in Table 2. Notwithstanding the protein content reduction, the average level is acceptable because it is connected to a better protein quality. Actually, some of the S8 lines obtained have a lysine content comparable to that conditioned by mutants such as opaque-2, and are stable.

Table 2: Correlation coefficients (r) between protein content (percent) and lysine content (g/100 g protein) for endosperm of S₄-S₈ generations.--**: significant at 1% level.

Generation	r
S ₄	-0.484**
S ₅	-0.547**
S ₆	-0.459**
S ₇	-0.639**
S ₈	-0.799**

It is important to point out that the selection for lysine content of the endosperm protein, and not for whole kernel, eliminates the possibility that the increase results from a greater germ proportion. In view of the fact that the germ has a good amino acid balance, the lysine levels on a whole kernel are superior to those of an endosperm. In these "normal genotype" lines the whole kernel has around 25% more lysine than the endosperm. It is also important to point out that the red flint lines have a 2.1 g average lysine per 100 g of protein in the endosperm, and the effective average increase of the lysine level has been approximately 48%. In the case of better lines obtained, which have an average of 3.6 g lysine per 100 g of endosperm protein, the increase over normal lines is about 70%. From the results obtained, it can be expected that high-quality protein lines and "normal genotype" may be used to improve the protein quality of flint corn without using floury mutants.

Jorge Luis Magoja and Angel Alberto Nivio

Influence of annual teosinte cytoplasm on SDS-protein subunits of maize endosperm

The cytoplasm of annual teosinte (*Zea mexicana*) influences not only the expression of some genes but also fertility, plant vigor, precocity and plant yield (Mazoti, Rev. Arg. Agr. 17:145, 1950; Mazoti, Rev. Inv. Agr. 8:175, 1954; Mazoti, Rev. Arg. Agr. 25:12, 1958). The purpose of this work is to investigate whether there is any influence of annual teosinte cytoplasm on the molecular pattern of endosperm soluble proteins. The study was performed in maize inbreds with an equivalent nucleus but with different cytoplasm, obtained by Mazoti. The genotype is a/a b/b C/C r/r pr/pr pl/pl ij/ij gl/gl, one of them with annual teosinte cytoplasm (E) and the other with maize cytoplasm (Z).

The soluble endosperm proteins were fractioned in both lines according to Laemmli (Nature, 227:680, 1970), by means of polyacrylamide gel electrophoresis in the presence of SDS (SDS-PAGE). The results obtained are shown in Figure 1.

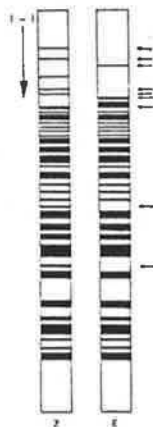


Figure 1: SDS-PAGE patterns of endosperm soluble proteins.
Z-- maize cytoplasm; E-- annual teosinte cytoplasm.

Since the nuclei of both lines are equivalent, the differences between them could be attributed to the influence of the cytoplasm. The protein patterns are different according to the cytoplasm. The line with teosinte cytoplasm shows either the lack of some protein subunits, or the presence of others which are absent in the line with maize cytoplasm. Based on these data, it is clear that teosinte cytoplasm also affects the expression of genes which codify endosperm proteins. A similar phenomenon conditioned by perennial teosinte cytoplasm was previously communicated (MNL 55:62, 1981). According to the results presented here, as well as those previously reported, it would be suggested that the main effect of the cytoplasm of the wild relatives of maize on storage proteins is revealed by a blockage of the expression of certain genes that codify storage proteins.

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A closer look at the gibberellin effect and sex expression in the reversible dwarf, anther-ear and

In the past, most studies dealing with the effects of gibberellic acid (GA) on genetic dwarfs of corn have dealt primarily with the vegetative characteristics of the plant and suppression of tassel development. The present investigations sought not only to repeat some of this work both in the field and under controlled environmental conditions, but also to re-examine some of the general observations with particular attention being paid to the cob, a structure which has been all but overlooked in this particular mutant.

Plants were treated with serial concentrations (10^{-6} M to 10^{-3} M) of GA on the average of every four days. Treatments began at the time of emergence of the first leaf through the coleoptile, and ceased at the time of emergence of the first silk

emergence. Each plant received a total of 100 ul of GA per visible leaf per treatment. The application was divided between the crown and at the ligule-leaf sheath junction of the expanded leaves.

Analysis of the data to be reported resulted from experiments initiated at the laboratories of Drs. Richard Greyson and Dave Walden at the University of Western Ontario in 1978 and continued at the University of Louisville in 1980-81.

In both controlled and field conditions, only the highest concentration, 10^{-3} M, of GA severely reduced the development of the tassels. Unlike some previous reports, the inhibition was not 100%. Inhibition ranged from completely sterile, poorly developed tassels to tassels that were 70-80% inhibited but still able to produce and shed pollen. Field grown plants exhibited a greater degree of inhibition than plants grown under controlled conditions. The inhibitory effects of GA were first seen as a reduction in the number of laterals on the tassel and a reduction in the number of florets reaching maturity on each lateral. Tassels produced on plants treated with 10^{-4} and 10^{-5} M GA had twice the number of laterals as the control plants. The main axis of the tassel was invariably the last portion of the tassel to show any reduction in development. The lower concentrations of GA produced no measurable reduction in tassel development, in fact, there was an increase in the number of tassel laterals at the lower GA concentrations.

Anthers produced on the 10^{-3} M treated plants were almost 20% smaller than those produced on control plants. When pollen grains from 10^{-3} M GA-treated plants were plated onto pollen germination medium, fewer grains germinated and a large number of these pollen grains spontaneously ruptured at a much higher frequency than grains from the control anthers. Studies are presently underway to examine the structure of these anthers and pollen grains to determine if structural variations can account for the apparent weakness of this pollen.

Previously reported experiments with GA reversible mutants rarely described effects on cob development. Anther ear is a mutant in which the cob contains, in addition to the female flowers, fully-formed, post-meiotic, non-dehiscent anthers. Apparently the normal genetic timing mechanism that causes the male parts of the flower to spontaneously abort in early development fails to act correctly. Coe and Neuffer have reported that GA-treated plants produce cobs that are "reverted," that is, they have gynoeocious flowers rather than the expected perfect (hermaphroditic) flowers peculiar, but not unique, to this mutant. Present experiments have confirmed this observation, but have made a more detailed analysis of the cobs.

Close observation of developing cobs reveals that the expression of sex within the flower appears as a gradient within the cob. The terminal portion of the cob is a sizable extension that contains only male flowers at maturity. These flowers initiate ovules in the early stages of development, and as in a tassel, abort early in development. These male flowers contained three or six mature anthers, but were never observed to shed pollen even though they were not enclosed in the cob and were exposed to the air. The major portion of the cob contains the normally expected, for an1, perfect flowers.

For the purpose of analysis, cobs were divided into three regions: Region 1, (R-1) terminal portion containing only male flowers (ovular stump may be present); Region 2, (R-2) containing male and female floral parts (hermaphroditic); and Region 3, (R-3) containing only female flowers (no evidence of stamens). The R-1 region of the cob normally dries up and is usually lost or discarded at harvest, and thus may be overlooked by someone examining only field-dried material.

As can be seen in Table 1, the application of gibberellin to plants of this mutant has a marked effect upon the sexual expression in the cobs.

Table 1. The effect of Gibberellic acid application upon sex expression in cobs of the anther-ear mutant.

Conc. of GA (M)	Cob length (cm.)	R-1 length (cm)	Male (%)	R-2 length (cm)	Herm. (%)	R-3 length (cm)	Female (%)
10^{-3}	5.8 \pm 2.1	0.3 \pm 0.8	(4)	0.4 \pm 1.3	(5)	5.2 \pm 2.2	(91)
10^{-4}	9.4 \pm 4.0	2.6 \pm 1.2	(32)	5.2 \pm 1.5	(51)	1.6 \pm 1.8	(17)
10^{-5}	9.4 \pm 5.0	3.8 \pm 1.9	(44)	5.5 \pm 1.5	(56)	0	0
10^{-6}	11.8 \pm 5.2	5.0 \pm 2.4	(46)	6.7 \pm 4.3	(53)	0	0
Control	15.7 \pm 3.9	6.7 \pm 2.5	(43)	8.8 \pm 2.6	(57)	0	0

Cobs from plants treated with 10^{-3} M GA were markedly smaller than those on the control. Much of this reduction was due to reversion of these cobs and the concomitant reduction of the R-1 region of the cob. Ninety-five percent of the 10^{-3} M GA-treated cobs were totally reverted. Dissection of these cobs under the microscope revealed no evidence of stamens. Five percent of these cobs were only partially reverted, and dissection revealed the presence of aborted stamens of almost normal size. The plants treated with 10^{-4} M produced cobs that were very much like the partially reverted cob produced at the higher concentration. A casual observation might indicate that these cobs were reverted; however, observation with a hand lens or microscope revealed numerous stamens that had aborted prior to meiosis and were now flaccid, semi-transparent structures similar to those reported in vitro (MNL 55:116). At 10^{-5} M GA most of the stamens appeared normal and were post-meiotic, but each cob still carried a large number of poorly developed or aborted stamens in the R-1/R-2 interface region. The cobs produced with 10^{-6} M GA were indistinguishable from the control. Thus the suppression of maleness as expressed in the cobs has the characteristics of a concentration related effect. As the concentration of GA increases, there is a concomitant decrease in the maleness and an increase in femaleness which is measurable in the sex expression gradient seen in these cobs.

Arnold J. Karpoff

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The close linkage between flourey-3 and pro

It has been reported previously that the location of flourey-3 (f13) is on chromosome 8 and that the gene order is f13 v16 ms8 j (MNL 53:56, 1979). We have also known that f13 is not allelic to pro-A342 (an allele of pro found by E. H. Coe, localized to chromosome 8), but that the two loci are closely linked. The pro-A342 mutant is homozygous lethal.

We now have a better estimate of recombination frequency derived from the following set of crosses. Plants that were + f13/+ + were crossed by pro +/+ +. The flourey seeds were planted, and those plants heterozygous for pro were

identified by test crosses. All plants in the F1 progeny were crossed by a + +/+ +; su/su male. The nonfloury seeds from a plant that was + f13/pro + were planted and the resulting plants selfed. Of the 64 selfed plants, 63 were segregating both for pro and for su. The remaining plant segregated for su but not pro. This leads to an estimate of 1.6 percent recombination between f13 and pro.

Oliver E. Nelson, Jr.

The catastrophic sexual transmutation theory (CSTT): From the teosinte tassel spike to the ear of corn

The uniquely monstrous, many-ranked ears of Zea mays have for too long remained an enigma. What species, what structure could possibly be ancestral? Most evidence has pointed to Zea mays subsp. mexicana, the annual teosinte. Yet its many, minute, 6- to 12-grained ears are fundamentally lateral on the primary branches, while those of maize are always solitary and terminal. Other differences abound. Maize ears could not have evolved from teosinte ears, structures which are not only rigidly canalized, but also edible only when immature and green.

Teosinte is, however, unquestionably ancestral; yet, since its primary lateral branches are always terminated by male inflorescences, the tassels, and since the central spike of the tassel is the homologue of the maize ear, as has long been recognized, the CSTT proposes that the maize ear is the transformed, feminized central spike of the tassels terminating the primary teosinte branches. Developmentally dominant because of its femininity in an apical position, and once past the sexual threshold, the proto-maize ear progressively suppressed all teosinte ears on the secondary and tertiary branches beneath it, drawing all nutrients of the whole branch system to itself. Feminization, and the resulting rapid catastrophic changes in resource allocation, thus transformed the slender terminal tassel spike into an increasingly effective nutrient sink. Because of their terminal position and their many non-cupulate axis segments, each with a pair of already fully fertile, soft-glumed, and free spikelets, primary branch tassel spikes were eminently preadapted to turn into free-grained, many-grained maize ears. These primitive tassel attributes, basic as they are throughout the Andropogoneae, are well-buffered genetically by what must be a large collection of polygenes accumulated long before the origin of maize. Once nutrient-overloaded, the distichous apical meristem would become polystichous by condensation-twisting as outlined by Collins, through slippage of rachid initials early in ontogeny. Femininity expressed on a male background per se, and not any of the postulated but elusive major genetic mutations, led to this epigenesis of the proto-ear, a probably easily induced abnormality with macro-evolutionary consequences. Canalization and amplification of these and additional cultivar attributes by human selection soon followed, saving this hopeful monster for posterity.

What exactly caused the sexual transmutation is unknown at present, but a shortening (condensation) of the primary branch internodes, which placed the tassel into the zone of female expression, was evidently involved (as either cause or effect?), triggered perhaps by abnormal environment (short days, cold nights), growth-substance-releasing pathogens (viruses, smuts), or mutations for branch condensation per se. Supported by much previous physiological work, and resolving nearly all archaeological, morphological and genetic paradoxes, the CSTT is amenable to experimental verification.

Hugh H. Iltis

[Ed. note: Accompanying material follows]

EVOLUTIONARY CRITERIA IN ZEA MAYS*

Assumptions of Iltis (1971), Galinat (1978, 1980) and Beadle (1972, 1978, 1980): a female structure of teosinte is ancestral to the female structure of maize.

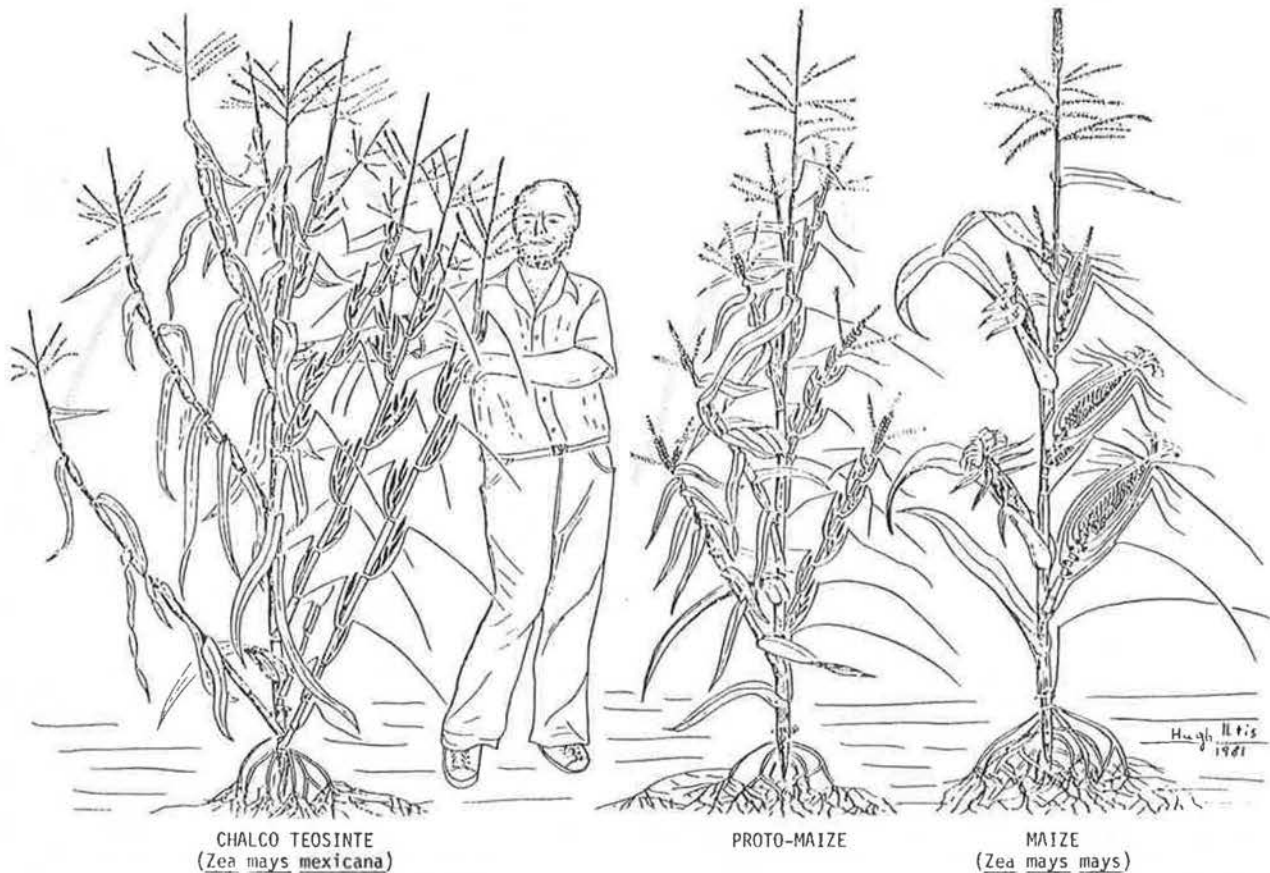
Alternative Assumptions (Iltis 1979, 1981; Allen and Iltis 1980; Doebley and Iltis 1980): a male structure of teosinte is ancestral to the female structure of maize.

- 1) The ear of maize evolved from a teosinte ear lateral to a primary branch or from an ear terminating a very short primary branch of an ear cluster lateral to the main stem, by the usual Darwinian process.
 - 2) The ear of maize became apically dominant gradually through human selection; no sudden reallocations of nutrients within the branch system are hypothesized. Sudden appearance of maize in the archaeological record, and the lack of teosinte fruitcases therein, are not explained.
 - 3) Domestication was initiated by harvesting teosinte grains for food. "Unbranched" teosinte plants with strongly clustered ears represent the crucial intermediary steps leading to maize, clustering of ears and lack of long tassel-bearing primary branches representing the direct consequences of selection for easier harvesting.
 - 4) Domestication of maize involved from the very beginning a step-by-step accumulation of single-gene mutations typical of Darwinian selection; it is therefore analogous to the domestication of the Old World *Hordeae* such as wheat or barley. All basic characteristics distinguishing maize from teosinte are due to human selection. Specifically, the reactivation of the suppressed pedicellate spikelet, that is, the doubling of the grain number per rachid, was due to a mutation favored by human selection.
 - 5) "The cupule [of the female teosinte spike] provides the connecting link between the maize cob and the fruitcase of teosinte" (Galinat 1975, p. 317); cupule homology represents the strongest morphological argument that the maize ear evolved from the teosinte ear.
 - 6) The so-called "freeing of the grain" from the hard teosinte fruitcase, a prerequisite to human use and subsequent grain expansion, was due to deliberate selection for various alleles of *Tu* ("tunicate"), genes which softened the glumes and flattened the fruitcases, allowing easy removal of grains. The very soft, papery glumes of the earliest archaeological maize and of primitive maize are due to "tunicate" genes.
 - 7) Variability in female teosinte spikes allowed selection for larger seeds and increase in spike units, this leading to the escape of the grain from the confines of the cupule and to the evolution of the maize ear. Multiplication of rachid units over the number found in teosinte ears (i.e., from 5-12 in teosinte to 18-40 or more in the most primitive archaeological maize) was due to a gradual stepwise accumulation of segment-increasing mutations analogous to the grain-increasing mutations in wheat, barley or rye.
 - 8) The genetic explanation for the evolution of the maize ear will be found in a relatively small number of simple mutations (ca. 5), each responsible for one distinguishing characteristic; admittedly, most are yet to be identified.
- The ear of maize evolved from the central spike of the terminal tassel of a well-developed, elongated, primary lateral branch, by way of a catastrophic sexual transmutation, a unique "macroevolutionary" event.
- By feedback loops, tassel feminization led exponentially to apical dominance; the tip of the primary branch, having developmental priority, preempting nutrients and thus suppressing development of ears terminating branches of lesser rank, changed from a nutrient-undemanding pollen-producing "governor" into a nutrient-requisitioning "dictator". The direct result of a sexual transmutation, this sudden reallocation of nutrients explains the sudden archaeological appearance of maize.
- Teosinte grains were not used for food. Openly branched teosinte plants were ancestral to maize, the primary branches stout, long, bearing ear-fascicles and terminating in tassels; "unbranched" plants represent normal ontogenetic responses of teosinte plants subjected to high competition resulting in shading.
- Only after a catastrophic sexual transmutation produced a free- and multi-grained proto-ear, thus allowing grain utilization, did step-wise selection under human domestication commence. Teosinte domestication is only in a minor way analogous to that of the Old World *Hordeae*. All basic characteristics distinguishing maize developed simultaneously as a consequence of tassel spike feminization. Specifically, doubling the grain number was an automatic consequence of the feminization of the teosinte tassel, each rachid of which already possesses two sexually functioning spikelets. Only non-fragmentation, husking, and increases in condensation, grain size and rachid number beyond those of the original feminized tassel spike are due to human selection.
- "In the oldest known archaeological maize cobs, the cupule is obsolete" (Galinat 1975, p. 317). The homology of the cupules is partial and indirect. The cupule of teosinte is phylogenetically a dead end, the cupule of the maize cob being derived from the flat, thin rind hypodermis of the tassel rachid facing each spikelet pair by an inward buckling and induration induced by feminization.
- The "freeing of the grain" from the teosinte fruitcase did not occur at all; free grains were a direct consequence of the expression of femininity on a male background; with each feminized tassel rachid folded back upon itself so as to exclude the grain and its soft-glumed spikelets, the grains were automatically "free" from the beginning. Tunicate genes induce atavistic abnormalities and have no bearing on the origin of maize. Archaeological maize ears are soft-glumed because they were derived from the soft-glumed male inflorescences.
- Female teosinte spikes (cupule, size of grain, number of units per spike) are under strictest genetic constraints and invariable within each taxon. Male teosinte spikes are morphologically indeterminate systems, greatly varying in unit number depending on plant size. "Multiplication of rachid units" above those found in the teosinte ear was initially simply due to a feminization of the many rachids (to 40 or more) of the central tassel spike, the comparison to Old World grains here once again based on false analogy. Zea is unique, the only monoecious major cereal.
- Most distinguishing characteristics of maize are based on fundamental Andropogonoid character syndromes, these still fully retained in teosinte and maize tassels and extremely well canalized by a multiplicity of polygenes unlikely to be individually identified.

RESULTS OF FOREGOING ASSUMPTIONS

The standard teosinte hypothesis creates paradoxes for which no solution can be found (soft-glumed and soft-cupuled primitive maize; inability to find monogenes differentiating maize and teosinte even for the simplest characters); does not permit creation of valid morphological criteria by which phylogenies for the races of maize can be established; no plausible morphological interpretation of maize ear morphology and anatomy are possible.

The Catastrophic Sexual Transmutation Theory resolves all paradoxes in maize evolution and archaeology; permits creation of consistent morphological criteria by which valid maize phylogenies may be established; allows a plausible interpretation of maize ear morphology and anatomy based on that of a teosinte tassel spike; promises experimental verification by environmental and genetic manipulation.



THE ORIGIN OF THE MAIZE EAR BY CATASTROPHIC SEXUAL TRANSMUTATION

The contraction of the branch internodes is coupled with a shift of their terminal male inflorescences (tassels) into the hormonal zone of female expression and the suppression of the lateral female inflorescences of teosinte. The shaded area indicates the threshold zone below which only female inflorescences form. Habit sketches are shown on the left side of each plant; diagrams of internode patterns, etc., on the right-hand side. Note increase in apical dominance associated with the feminization of the apical inflorescences of primary lateral branches.

MEMORABLE QUOTES IN THE ORIGIN OF MAIZE CONTROVERSY PERTINENT TO
THE CATASTROPHIC SEXUAL TRANSMUTATION THEORY

"Hybrids between maize and teosinté will always exhibit suggestive series; but, until we are more sure of the homologies between these two genera, it is futile to expect much information from the hybrids, for they will be speaking in a language that we cannot understand."

--Weatherwax, 1923

"To have changed into something like corn, [teosinte] would have had to do the unlikely thing of undergoing despecialization in two or three ways, and these changes would have had to occur so closely together that they immediately gave the plant an economic value which it did not previously have."

--Weatherwax, 1955

"The differences between teosinte and maize are complex both morphologically and genetically and it does not seem possible that maize could have been derived from teosinte during domestication by any genetic mechanism now known. If maize has originated from teosinte it represents the widest departure of a cultivated plant from its wild ancestor which still comes within man's purview. One must indeed allow a considerable period of time for its accomplishment or one must assume that cataclysmic changes, of a nature unknown, have been involved."

--Mangelsdorf, 1947

"The central stem of the 'tassel' borne by the primitive branch by virtue of its more favorable [terminal] position, drew into itself the main [nutrient] force of the branch, and became more highly developed at the expense of the surrounding tassel-branchlets, the latter becoming finally entirely aborted."

--Kellerman, 1895

"...in this evolution the central spike of the [teosinte] tassel developed into an ear [of maize].

...the terminal tassel-like structure...borne in a leaf axil, surrounded by a kind of husk as is an ear of maize, and [bearing] only pistillate flowers...is only a step in the production of an ear of maize, from teosinte, by a development of the central spike of the lateral [teosinte] tassel into [a maize] ear."

--Montgomery, 1913

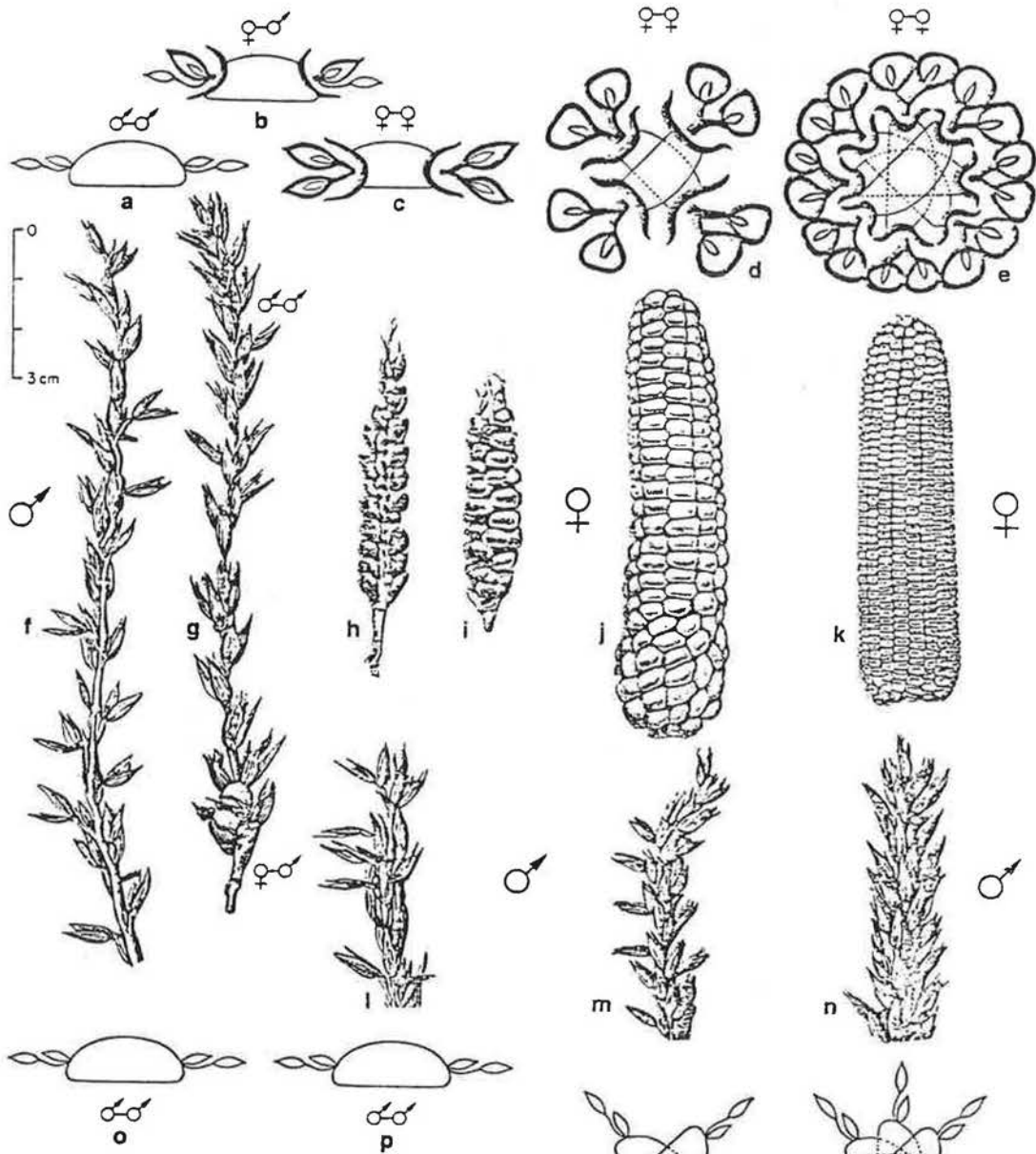
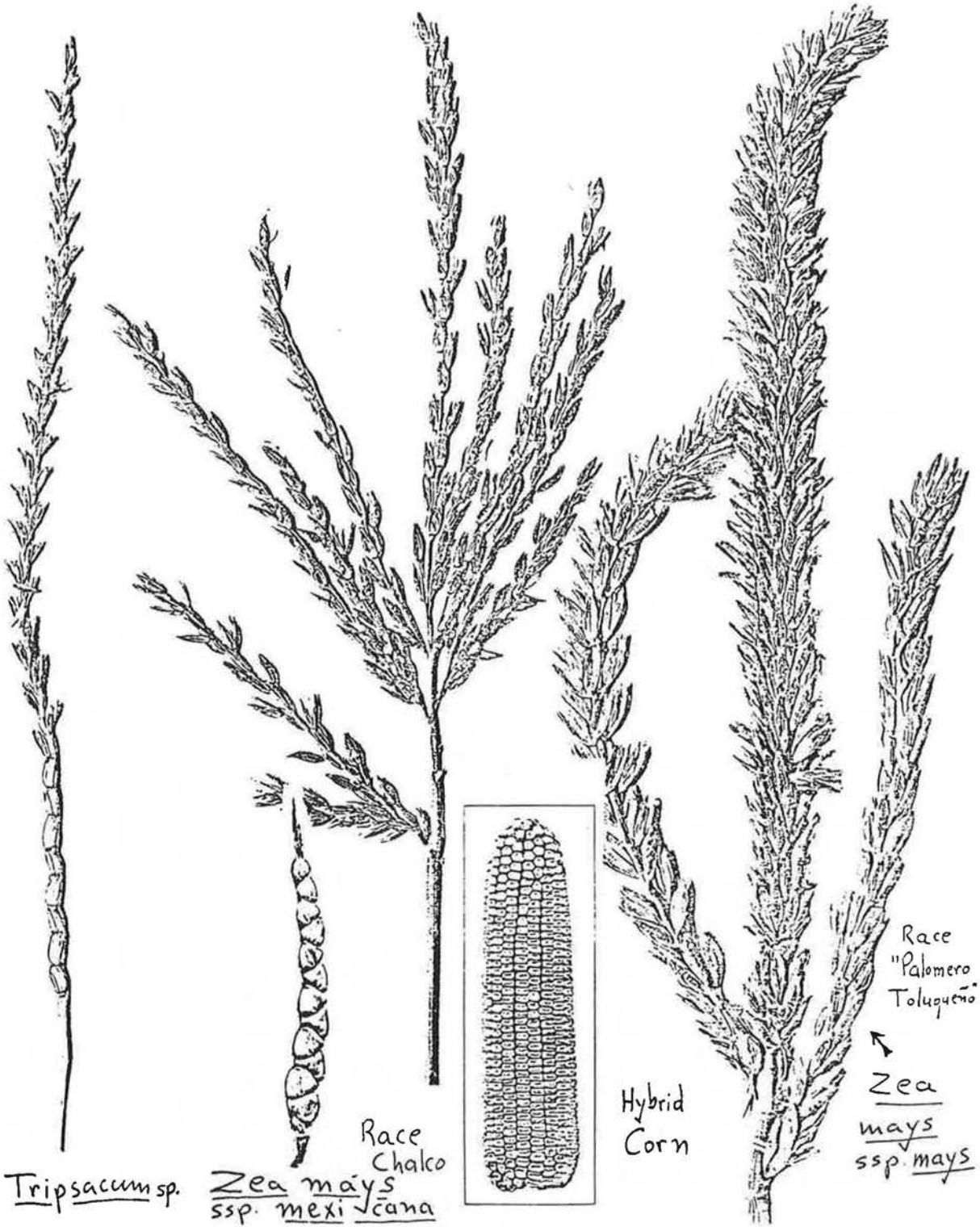


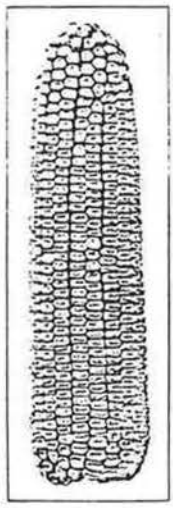
Fig. 2. The evolution of the maize ear by sexual transmutation [f-k; x-sect. a-e, using Collins' (1919) diagrams of the male sequence as base] leads from a distichous central tassel spike (f), becoming feminized at base (g), to a distichous (h), then polystichous 8- (i, j) to 16-rowed (k) ear, with condensation (contraction of internodes), twisting, compaction and (after i) multiplication the processes involved. The homologous male sequence [f, l-n; x-sect. o-r, from Collins (1919)] leads to the polystichous tassel spike of maize (n, r) consequent to human selection in the female sequence [f - Chalco teosinte (*Zea mays* subsp. *mexicana*); g - Chalco teosinte x maize; h, i - Chalco teosinte x Argentina popcorn hybrids, grown by G. Beadle in Mexico; j, k - maize; l - n to same scale; k - reduced by one-half].



Tripsacum sp.

Zea mays
ssp. *mexicana*

Race
Chalco



Hybrid
Corn

Race
"Palomero"
Toluqueno
↖
Zea
mays
ssp. *mays*

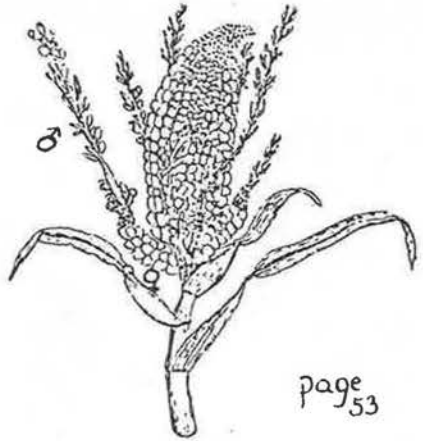
THE PRIMITIVE CORN.—Mrs. W. A. Kellerman offers the following interesting speculation on the origin of Indian corn:

"In MEEHANS' MONTHLY, January, 1892, there was a note concerning the primitive corn—with an illustration. There was also an article published last year relative to the development of the ear. In both articles we find practically the same thought, viz.: 'If we draw the branchlets of the 'tassel' upwardly with the hand we shall see exactly the resemblance to an eight-rowed ear of corn. No one can fail to see that the ear of corn is nothing more than the tassel which has had power to unite its branchlets and become succulent.'

To tell the whole story as concisely as possible—as the corn told me—I would say that the primitive Indian corn was a grass-like plant (a grass in reality) with a branch springing from the several nodes or joints. Each branch was crowned with both staminate and pistillate organs. The central stem of the 'tassel' borne by the primitive branch by virtue of its more favorable position, drew into itself the main force of the branch, and became more highly developed at the expense of the surrounding tassel-branchlets, the latter becoming finally entirely aborted.

Natural selection lifted the staminate flowers to the tassel of the main stalk and left the pistillate below on the side branches. These branches became shortened, and form the shank or footstalk of our present ear. The shortening of the branches brought the sheaths close together, thus forming the husk or general protective envelope of the ear.

Now instead of a cohering of the branchlets to form the ear, it seems quite clear that such reversions as the sketch on page 53, illustrates plainly how the ear was developed from the central stem of the primitive lateral tassel, while the branchlets became aborted. The woody substance became the cob, and the pistillate flowers, having here gained a monopoly, improved their opportunity, and made the most of themselves. Under the kind guardianship of Nature the Indian corn traveled along up through the centuries; but long continued cultivation has been an important factor in perfecting the splendid ear of the present.



THE PRIMITIVE CORN.—SEE PAGE 44. (Sexual abnormality in an ear of Indian Corn.)

Despite the great variety of explanations, Krafft's (1870) magnificent lithographs, Ascherson's (1880) diagram, and Kellerman's (1895) drawing, and countless others, from Boccone in 1674 to Montgomery (1906), Iltis (1911), East (1913), Weatherwax (1918, 1923, 1935) and Kempton (1935) on into more modern times, all show the common abnormality "Branched Corn" (Kempton, 1923), in which a polystichous ear (4- or more ranked) is subtended by 1-11 distichous (2-ranked) branches, showing a feminized tassel as well as that polystichy is a position effect related to apical dominance, the central spike blooming first and hence having hierarchical priority.

1880.

Herr Dammer (Gast) legte einen ästigen Maiskolben aus dem hiesigen Königl. botan. Garten vor. p.133

Herr P. Ascherson hat hierzu Folgendes zu bemerken:

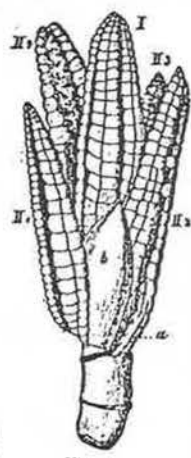


Fig. 1.

Der vorliegende, nach einer mit gewohnter Gefälligkeit angefertigten Zeichnung des Herrn F. Kurtz Fig. 1 in der Seitenansicht (1/2 der natürl. Grösse), Figur 2 im Diagramm dargestellte weibliche Blütenstand von *Zea Mays* L. stellt eine Bildungsabweichung dar, die allerdings bereits seit zwei Jahrhunderten bekannt, mehrfach beschrieben und abgebildet ist, dennoch aber in manchen Punkten noch eine eingehendere Besprechung und selbst eine genauere Abbildung verdient, als ihr bisher zu Theil geworden ist. Der erste Botaniker, welcher diese Missbildung beobachtete, war der Sicilianer Paolo Boccone, der sie in Calabrien „ad pagum et coenobium Sancti Dominici Soriani“ auffand und in den 1674 von Robert Mori-

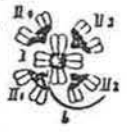
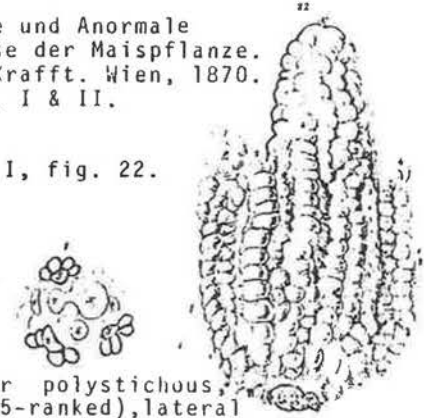


Fig. 2.

Die Normale und Anormale Metamorphose der Maispflanze. Dr. Guido Krafft. Wien, 1870. pp.71, Tab. I & II.

Tab. I, fig. 22.



Central ear polystichous, 10-rowed (5-ranked), lateral branches distichous, 4-rowed (2-ranked).

POPULAR SCIENCE MONTHLY 68:
55-62
WHAT IS AN EAR OF CORN?
E. G. MONTGOMERY
(1906)

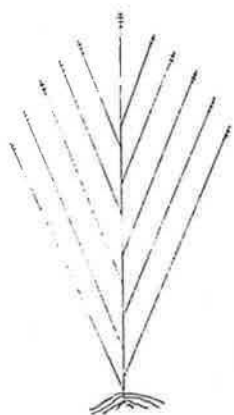


FIG. 13. DIAGRAM ILLUSTRATING PROBABLE STRUCTURE OF EARLY PROGENITOR OF CORN PLANT.

p. 61

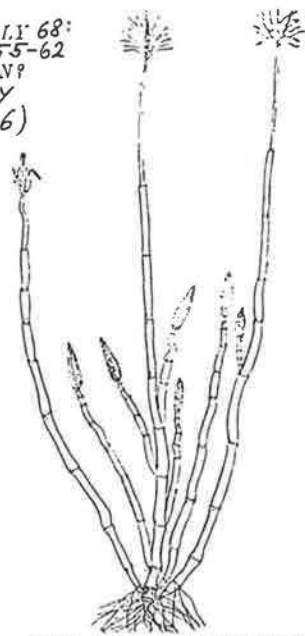


FIG. 14. DRAWING FROM PHOTOGRAPH OF A SWEET CORN PLANT TO COMPARE WITH DIAGRAM FIG. 13. Note that the number of nodes in the shortened ear-bearing branches corresponds exactly to the number of nodes in the main stem above point of attachment.

p. 61

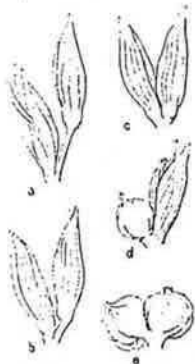


FIG. 5. MODIFICATION OF A PAIR OF STAMINATE SPIKELETS INTO A PAIR OF PISTILLATE SPIKELETS. a, b, c. The pedicellate spikelet shortens down until it becomes sessile. d. The sessile flowers become pistillate; e, both flowers become pistillate.

p. 58

may now be easily traced. First, the pedicellate spikelet in each pair of spikelets becomes sessile so that we have a pair of sessile spikelets as in Fig. 5, c. Then the upper flower in each spikelet becomes a perfect pistillate flower, while the lower flower in each spikelet becomes an abortive pistillate flower. The pairs of spikelets on the central spike are in four to eleven or more rows, so that by the mere development of the central spike of the tassel into

p. 61

18 THE CORN CROPS

Gama Grass ... looks

like maize. While it grows to a height of five to ten feet the stem is slender and the leaf about half the width of the maize leaf. The plant bears a tassel-like structure at the top and on the lateral branches, closely resembling the maize tassel, except that the seeds are borne on the lower part of each tassel and the pollen on the upper part.

Teosinte, which is sometimes cultivated but does not mature north of Mexico, is more like maize than is gama grass, the plant being larger and the terminal tassel bearing pollen only. The lateral branches of the plant are so shortened that the terminal tassel-like structure is borne in a leaf axil, surrounded by a kind of husk as is an ear of maize, and bears only pistillate flowers, or seed. It is only a step in the production of an ear of maize, from teosinte, by a development of the central spike of the lateral tassel into an ear.

It is probable that the early progenitor of maize was a grass-like plant having a tassel at the top and tassel-like structures on long, lateral branches, all tassels bearing perfect flowers. As evolution progressed, the terminal tassel came to produce only pollen, and the side branches only ovules, or seeds. Evolution often results in a greater "division of labor," as in this case. At the same time, the lateral branches were shortened or telescoped into the leaf sheaths, these sheaths forming a covering, or husk, for the ear. Also it is probable that in this evolution the central spike of the tassel developed into an ear.

The close relationship of maize and teosinte is proved by the crosses that have been made between the two. In this third or fourth generation after crossing, a peculiar type of corn is secured, identical with a type of maize that has been found growing wild in Mexico (*Zea canina*), and

THE CORN CROPS

E. G. MONTGOMERY

PROFESSOR OF FARM CROPS IN THE NEW YORK
STATE COLLEGE OF AGRICULTURE AT
CORNELL UNIVERSITY

New York

THE MACMILLAN COMPANY

1913

MONTGOMERY: ON THE RIGHT TRACK

Montgomery's 1906 drawings of the "early progenitor of corn plant" and its sweet-corn counterpart come indeed so close to the CSTT that one wonders why he did not make the connection. Apparently he had not seen branched teosinte plants, with male tassels at the end of the primary branches, and assumed that corn came from a plant with perfect-flowered panicles by differential sterilization. His 1913 paper (p. 18, shown above) gives a description of the CSTT, stressing the role of the central spike, but of the tassel-like female ear cluster of teosinte. However, his language is confusing, and one is at a loss to know what exactly he had in mind.

THE PHYLOGENY OF ZEA MAYS

29

Weatherwax
1935

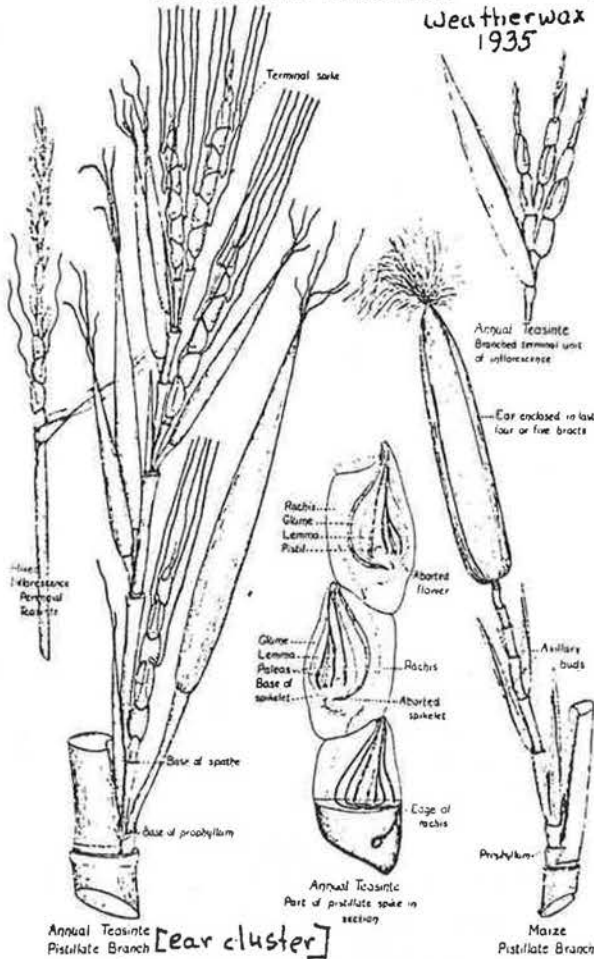


Fig. 9. Pistillate inflorescences of Zea and Euchlaena.

THE STORY OF THE MAIZE PLANT

PAUL WEATHERWAX
Associate Professor of Botany
Indiana University
1923

Weatherwax's (1923) prophetic statement ("...until we are more sure of the homologies between [teosinte and maize]... the hybrids... will be speaking in a language that we cannot understand.") applies to his own splendid drawing of 1935, which suggests visually that such an axillary ear cluster of teosinte as shown above is homologous to the maize ear and its shank and husks, a false conclusion which nevertheless forms the basis for the Standard Teosinte Hypothesis.

Anomalous inflorescences will doubtless contribute valuable information, but the investigator must avoid the common error of considering every anomaly a reversion.

Hybrids between maize and teosinte will always exhibit suggestive series; but, until we are more sure of the homologies between these two genera, it is futile to expect much information from the hybrids, for they will be speaking in a language that we cannot understand. When the true homologies of their inflorescences are clear, then these hybrids may afford checks upon our conceptions of morphology; but they will never alone constitute valid constructive evidence as to phylogenetic relationships or the course of evolution. Interaction between closely related entities is capable of giving rise to monstrosities that defy explanation in terms of the relationships of the parent-stocks; and only a sound working basis of morphology will save the investigator from the lure of suggestive analogy. *Weatherwax 1923:113*

BRANCHES OF THE SHOOT

57

Weatherwax
1918

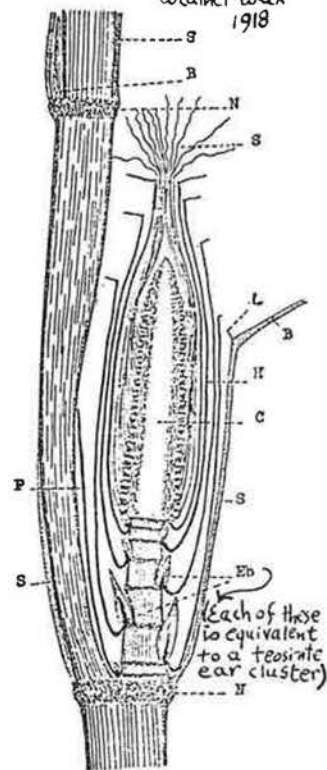


FIG. 45.—Diagram of longitudinal section of ear-bearing branch. S, leaf sheath; B, axillary bud, an undeveloped ear-bearing branch; N, node of the main axis; S, silks exposed beyond the ends of the husks; L, ligule; B, leaf blade; H, husk of the ear, a greatly enlarged leaf sheath; C, cob of the ear; Eb, secondary ear buds; P, prophyllum.

Über einige bei *Zea Mays* L. beobachtete
Atavismen, ihre Verursachung durch den
Maisbrand, *Ustilago Maydis* D. C. (Corda) und
über die Stellung der
Gattung *Zea* im System.

PP. 38-57, tab. II-III

Hugo III's

page 45. Es ist daher die Annahme einer Fasziation ganz und gar unnötig und die Goebelsche Anschauung viel natürlicher, daß der Maiskolben aus einem der ♂ Rispe gleichenden Blütenstand dadurch entstand, daß die Hauptachse, um die zur Bildung der großen Früchte nötigen Nährstoffe speichern zu können, sich korrelativ verdickte und daß die Ausbildung der seitlichen Rispenäste einfach unterblieb. Die Kolbenspindel, die sich freilich später auch ohne Befruchtung ausbildet¹⁾, ist also ursprünglich nichts anderes als der infolge der Fruchtbildung verdickte Hauptstrahl der Rispe. —

From: Banathy, B. H. (ed.) 1980, SYSTEMS SCIENCE AND SCIENCE. Proceedings of the Twenty-Fourth Annual North American Meeting of the Society for General Systems Research, With the American Association for the Advancement of Science. San Francisco, California, January 7-10, 1980, pp. 96-103. Society for General Systems Research, Systems Science Institute, Louisville, KY 40208.

OVERCONNECTED COLLAPSE TO HIGHER LEVELS:
URBAN AND AGRICULTURAL ORIGINS, A CASE STUDY

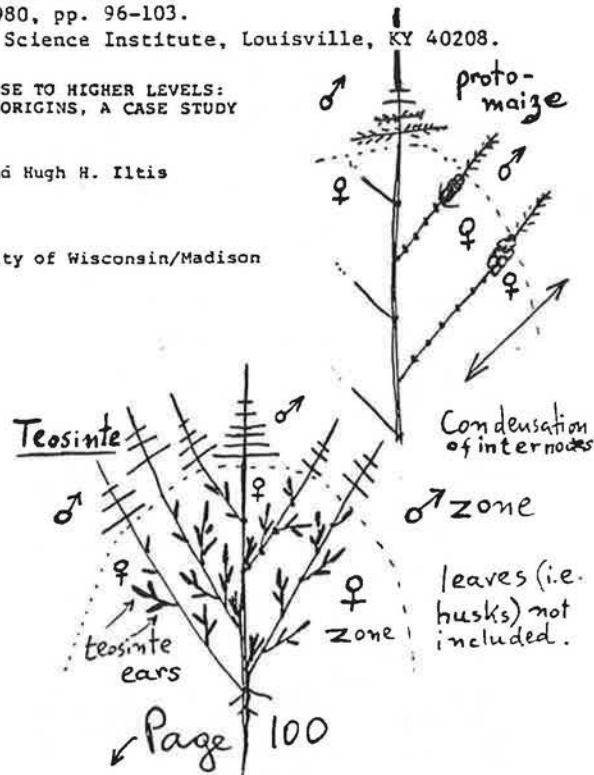
T.F.H. Allen and Hugh H. Iltis

Botany Dept., University of Wisconsin/Madison

ABSTRACT

Agricultural origins are seen as collapse of a hunter-gatherer system whose parts become too tightly connected. Overconnectedness becomes part of the stable functioning of the evolved higher level structure. Cities are a physical manifestation of the overconnectedness which collapsed the Paleolithic system. Competing historical theories appear to represent only local passages through a reticulum. The different relaxation times and rules on either side of the Neolithic Revolution require different, but complementary, time frames for observing the change. Narrative time frames appropriate before the event are inapplicable after the revolution. It is better seen as a catastrophe where system rules change faster than the system functioning. System constraints pass instantaneously from one state to another without existing in between.

THE CATASTROPHIC SEXUAL
TRANSMUTATION THEORY of
the origin of the maize
ear from the central spike
of a lateral branch
teosinte tassel.



Catastrophe in Plant Morphology:

There is a difference between Old and New World primitive agroecosystems. In the Old World, cities have continuously existed at various sites such that they feel as if they have always been, or at least were inevitable. Therefore, the question of scholars is: how did cities and agriculture arise? In the New World, however, there are a few cities of large dimensions, large enough to impress

Note : For Cob, read ear!

101

Invading Europeans, but there are no intermediate population aggregations. The demography of settlement size jumps from the city of Montezuma to clusters of people at crossroads with no moderate sized aggregation in between. Since cities appear anomalous in the New World and seem to be based upon only a marginally sufficient culture, then the question of the New World scholars is: how is it that cities occur at all? This difference in agroecosystem texture may be explained by considerations of catastrophic overconnection in the important New World cereal, corn.

In the Old World domesticated cereals, the differences between cultivated varieties and wild plants is clear but not distinct at an architectural level. Domesticated varieties have more grains which are larger, and they have a central axis to the ear which is solid and does not shatter at ripening. There are also differences in some chromosome numbers, but these are no more distinctive than occur in many wild interspecific relationships. By contrast, in New World corn, *Zea mays*, the domesticated plant has a completely different pattern of construction from its wild precursors. This is so much the case that despite a full knowledge of the complete interbreeding compatibility between corn and its parent species, scholars have only recently correctly identified the ancestor as such. Teosinte, wild corn, has many male tassels over the outer portion of the whole plant with many small female cobs distributed through the center of the plant's branching structure. Furthermore, the cobs are so small as to be unrecognizable as the antecedent of the familiar domesticated structure. The differences between wild and domesticated cobs are major: a) in the domesticated plant a second grain arises at loci where in the wild type there is only one; b) the central axis of the cob is not only rigid and non-shattering, but it becomes twisted in a complex fashion so as to give as many as 24 rows of grain where in the wild type there are two alternative rows; c) all the grains on the plant are found in one or two cobs instead of hundreds. The second author (Iltis, 1979) has synthesized the diverse evidence for structural homology and from this has worked out a very reasonable sequence of structural changes in the origins of the corn plant, explaining the sudden appearance of corn agriculture.

The ^{wild} corn plant ^(+teosinte) has two zones, an outer zone which is male and an inner zone which is female. Major side branches terminate in male tassels which are developmentally dominant over all other tassels and cobs on that branch. Once the terminal tassels have flowered then secondary tassels come into flower. These

dominate tertiary tassels and cobs, and so on. The dominance of the male is constrained by the limited resources which a tassel can use: some material for pollen and the rest for small scaly flowering parts. If the side branches become shortened, perhaps by genetic changes but more probably by crowding of plants, then the tip of the side branch finds itself in the female zone of the plant, and instead of producing male flowers, produces females. Unlike its male counterpart, this female exerts a vigorous and persistent dominance over all other tassels and cobs on that side branch. By changing the terminal structure of the side branch from male to female the whole balance in the dominance system is upset. There is a catastrophic connection between a larger, now female "tassel" and the major portion of the photosynthetic activity of the branch. Food is sucked into that side branch by the female with disastrous structural consequences. Unable to expand so as to accommodate the massive food supply delivered, the corn cob spontaneously twists. Thus, a single change from male to female in the terminal tassel of the lateral shoot, catastrophically changes the physiological balance within the plant and instantaneously produces a structure which would closely resemble a modern corn cob. The entire architecture of the plant is changed so that the old rule system of balanced, continuous reproduction is no longer applicable. For most plants this would be fatal. If, however, there is human intervention then a new set of rules may emerge which allows the regular production of domesticated corn cobs.

In the Old World the rule system of the plant does not change because the plants are already single-headed in their construction. Agricultural rules are the same as the rules for the wild type. In corn, however, the wild plant rules and the agricultural rules are entirely different. Presumably, the collapse of wild type rules had occurred millenium after millenium before agriculture. However, only when human population density and levels of technology were sufficiently high would the human creature have been in a position to incorporate the catastrophically changed corn plant into its social structure. The social structure had to be almost ready for a spontaneous collapse into agriculture before the catastrophe of the corn plant was signal enough to begin the inevitable collapse to intensive urban agriculture. In the Old World the plants are secondarily influenced by the primary collapse through overconnection of the human social structure. In the New World the capacity for change in the rules in the corn plant was the primary signal which, when it happened, dragged the human social structure into agriculture slightly, prematurely by Old World standards.

MILAN, ITALY
University of Milan

Developmental mutants and seed formation

Developmental mutants are a useful tool for dissecting development and understanding the genetic program underlying this process. We focused our attention on seed development by analyzing recessive mutants with defective seed morphology acting as lethals at the seed or seedling stage. The survey of these mutants is aimed at establishing their involvement in embryo development and, possibly, the embryogenic phase affected.

The preliminary data gathered so far are presented in the table below. The mutants analyzed, 13 in total, appear heterogeneous. They all show more or less

Mutant symbol	Origin (1)	Seed phenotype	Embryo age (days)	Length (mm)		Embryo morphology	Germination (%) (2)	Seedling growth (3)
				m	+			
ed-7 1	EMS	small seed with hooked pericarp	20	2.5	3.7	(a) reduced size, apparently normal development	52.4	35% necrotic 65% reduced growth
			31	4.8	6.2			
			42	6.0	7.8	(b) reduced size, normal morphology		
G7-2	EMS	flat, hooked seed	15	0.4	1.2	(a) red. size, apparently normal dvlpm	69.3	8% necrotic 92% red. growth
			45	5.2	7.9	(b) red. size, normal morphology		
G33	EMS	small seed	21	4.7	5.1	(a) slightly red. size, normal dvlpm.	93.2	all normal
			35	6.0	7.8	(b) slightly red. size, normal morph.		
			41	6.0	7.8			
ed-27 1	EMS	smaller size distorted growth	20	2.8	5.2	(a) reduced size, normal development (b) reduced size, normal morphology	not tested	not tested
ed-57 1	EMS	minute seeds	32	3.6	6.6	(a) delayed dvlpm., embryo axis recognizable only at 43 days (b) extr. red. size, normal morph.	not tested	not tested
			43	4.0	8.4			
ed-64 1	EMS	opaque, abnorm. morphology	20	gls	3.7			
ed-51 1	EMS	very small seeds	31	gls	7.3			
G22	EMS	aborted seeds	40	gls	8.4			
ed-55 1	MNNG	small flat s. shrunken like	31	3.8	7.6	(a) embryo axis not recognizable	24.6	all necrotic
			39	3.3	8.5	(b) red. size, abnormal morphology		
ed-54 1	control	smaller size no carotenoids	21	1.0	6.1	(a) dvlpm. blocked at proembryo stage	-	-
			34	1.0	8.4	(b) unchanged size, smooth surface and light colour		
			42	1.0	8.4			
ed-56 1	EMS	small seeds, opaque, no anthocyanin	27	1.0	6.0	(a) dvlpm. blocked at proembryo stage	-	-
			42	1.0	8.2	(b) unchanged size, rough surface and necrotic		
ed-48 1	EMS	small seeds with hooked pericarp	23	2.4	6.2	(a) 23 days: irregular morphology of scutellum. Embryo axis not recognizable. 31 days: embryo axis visible but coleoptilar primordium not recognizable or abnormal (b) red. size, abnormal morphology	40.7	70% necrotic 30% reduced growth
			31	4.2	7.3			
			42	4.5	8.8			
ed-61 1	control	defective endosperm, no anthocyanin	23	1.8	4.2	(a) 23 days: flat scutellum, embryo axis extremely reduced	-	-
			42	gls	5.2	(b) embryo not recognizable		

(a) during embryogenesis
(b) in the mature seed

(1) mutant isolated in M_2 populations following seed or pollen treatment with ethyl methane sulphonate (EMS) N-methyl-N-nitro-N'-nitrosoguanidine (MNNG) or in control populations

(2) n = 50 or more seeds

(3) as determined 20 days after germination

(4) gls = germless

drastic alterations of the endosperm, while the embryo is either normal but reduced in size, or arrested at some early developmental stage, or not recognizable at the time of observation. This heterogeneity is similar to that reported by Sheridan and Neuffer (J. Hered. 73:318-329, 1982) in other defective kernel mutants.

Some of these mutants have also been cultured as immature embryos, starting 20 days after pollination up to 50 days, on mineral and enriched RM media. A detailed presentation of the results will be given elsewhere. Here only mutants 7 1, 27 1 and 48 1 are considered. They all germinate and yield normal looking seedlings when cultured as immature embryos on both mineral and enriched media, while as mature embryos they exhibit a significantly lower germination capacity and reduced (7 1 and 48 1) or suppressed growth (27 1).

They can be further differentiated on the basis of their growth rate as excised shoot tips. In fact, after seven days of culture in liquid media, the length of seedlings is 80 mm for mutant 7 1, 21 mm for 27 1, and 8 mm for 48 1, respectively (control seedling length = 106 mm).

The recovery of normal seedlings by culturing immature embryos is the result expected from phase-specific lethal mutants, since lethality occurring in advanced stages of embryogenesis is bypassed by inducing precocious germination.

An unexpected result is germination on filter paper, even though incomplete, of mutants originally isolated as absolute lethals when planted in sand benches. Germination in these mutants varies from 24% for 55 1 to 93% for G33; seedlings are ostensibly retarded in their growth compared to normal siblings. At the time these observations were made (20 days after germination), part of the seedlings are necrotic, while the remaining are still alive but likely to die.

It might well be that this behavior is the result of hormone imbalance. It is hoped that further histological characterization of these mutants and an analysis of their metabolism requirements will allow an understanding of the basis of their lethality, as well as the role played by both embryo and endosperm in seed development.

S. Faccio Dolfini, G. Gavazzi and G. Todesco

Detection of haplo-diploid gene expression in maize

One of the most important problems concerning the role of the male gametophyte in the evolution of higher plants is the genetic relationship between the haploid and diploid phases: are the genes expressed in the gametophytic phase the same as those controlling sporophytic functions, or does a specialized set of genetic factors exist? As far as characters determined by single genes are concerned, observation of segregation in a gametophytic population deriving from a single heterozygous plant provides a means of revealing haplo-diploid expression. This can be made by means of specific staining of pollen grains, provided that +/- variants and suitable staining techniques exist. A more general method is provided by the analysis of the electrophoretic pattern of dimeric (or multimeric) enzymes. If the enzyme is of sporophytic origin, pollen extracts from plants heterozygous for electrophoretic mobility display the same banding pattern as sporophytic tissues (two homodimers and one heterodimer), whereas in the case of haploid transcription the pollen extracts reveal only the two parental homodimeric bands. However, it cannot be ruled out that causes other than haploid transcription may be responsible for preventing the activity of the heterodimeric enzyme, while this can be discarded if the heterodimeric form is found in pollen with heterozygous gene duplication.

This pollen type can be obtained for most of the maize genome using appropriate B-A translocations. Because of the B centromere nondisjunction at the second microspore division, a heterozygous B-A translocation, having an AA^{BBA} genotype,

produces pollen in which one of the two sperm cells has two B^A chromosomes, and the other none. In the progeny of a cross for which TB-A plants $A(F)ABBA(F)$ are used as male parent and normal $A(S)A(S)$ plants as female parent, seeds with hyperploid embryo, heterozygous for electrophoretic mobility, can be selected.

Hyperploid embryos are selected by means of genetic markers, chromosome dosage effects and/or root tip chromosome counting. At flowering, the resulting plants produce pollen that is in part partially diploid and heterozygous for electrophoretic mobility for enzymes coded by genes localized on the B-A translocated chromosome arm.

This method has been tested by analyzing electrophoretic variants of ADH-I, and has been used to study the gametophytic expression of GOT-1. For both the enzymes, sporophytic extracts of inbred lines revealed one band, while three bands were found in the hybrid; pollen extracts were identical with sporophytic extracts of the inbreds, while hybrid genotype gave rise to only two bands. Pollen from TB-A hyperploid plants revealed three bands, thus indicating that, when both alleles are present in the pollen grain the active heterodimeric enzyme is formed.

The method can also be used to map genes with gametophytic expression, specifying dimeric (or multimeric) enzymes and particularly to study genes with gametophytic expression.

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An indoor growth room for maize

One problem that concerns most maize researchers in northern climates is how to obtain adequate numbers of well-developed plants on a year-round basis. For some plant species that do not require intense light, standard greenhouses usually suffice. For others that require summer-like light intensities for normal development, supplemental lighting in a greenhouse is the usual manner of improving light quality and quantity. We have taken a different approach to solving this problem by constructing a large indoor growth room in which corn is capable of growing to maturity.

The dimensions of the growth room are 24 feet by 38 feet with a ceiling height of 19 feet. The floor is concrete with four floor drains. The two exterior walls are concrete block and the interior walls are covered with sheet-rock. The ceiling is corrugated steel. An epoxy paint was applied to waterproof the walls. There is a working area with sink and appropriately spaced water spigots for watering.

In order to provide adequate light intensity and quality, the growth room was outfitted with twenty-three 1,000 watt high pressure sodium (HPS) lamps and twenty-two 1,000 watt metal halide (MH) lamps. These lamps provided not only the intensity necessary, but when placed alternately, gave a very good spectral mix. The different lights are alternated within a row as well as between adjacent rows (i.e., one row starts with an HPS, the next with an MH). The spacing is four feet on center within a row and three feet on center between rows. This spacing was chosen to facilitate a blending of the light from the different lamps. The MH lamps are richer in the blue region of the visible spectrum (around 450 nm). The HPS lamps are rich in the red and far red region, especially around 550-600 nm. The plants are on a 14-hour light, 10-hour dark cycle. The ballasts for the lamps are mounted on a metal framework 12 feet from the floor. The reflectors are

attached to the ballasts with a power cord that allows them to be moved via a pulley system. The height available for plant growth is 10-11 feet.

Temperature control is accomplished with two units (one is an air conditioner and the other is a combination air conditioner/heater) that have a total cooling capacity of 181,000 BTU/hr and air movement of 6,000 CFM. The ventilation system for this room is independent from that for the rest of the building. Plastic convection tubing (usually used in greenhouses for good heat distribution) is being used to assure effective air distribution and temperature control. The air conditioners are controlled by a remote sensor that is mobile and can be placed where desired in the plant canopy. The temperatures maintained are 66 F at night and 80 F during the day.

The soil mix used is a 1:1:3 (soil:sand:peat) that is mixed 3:1 with perlite. After the plants reach the four leaf stage they are fertilized once a week with 20-20-20 fertilizer (30 g/liter) and a soluble trace element mixture (25 mg/liter). This is applied with a 1:15 proportioner, with each pot receiving approximately 1,200 ml of the solution. The plants are grown in three gallon pots.

We have had good success growing many public inbred lines, as well as some special stocks. One line, W22 R-nj, which can be difficult to self in a winter greenhouse environment, has been grown and selfed successfully in our growth room. Plants are routinely selfed, except for a few lines where silking and pollen shed do not allow proper nicking. A high degree of prolificacy has also been observed among plants grown. The plants mature properly in all respects, and resulting ears have good seed development. Typically, ears are harvested five to six weeks after pollination. This continuous supply of plants has allowed us to make considerable headway in our corn tissue culture program.

Additional information is available from us upon request.

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Knob distribution in Himalayan strains of maize

The results of the pachytene analysis of 29 maize collections from the North-Eastern Himalayas (N.E.H.) and two American races have been summarized in Table 1. Pachytene chromosomes have been well identified on the basis of their relative lengths and arm ratios. Hence, it was possible to assign exact positions of knobs on particular chromosomes. There was a preponderance of subterminal knobs in the N.E.H. collections, suggesting that Mexican teosinte has played a significant role in the evolution of Himalayan maize.

Except for the Sikkim Primitive types (S.P.), the knob number, in general, among the N.E.H. maize is low. In order to study relationships between knob number and altitude, material collected from different ethnic groups of Sikkim at various altitudes has been studied. Maize collections from Sikkim at altitudes ranging from 1,200 to 1,400 m have an average knob number of 4, while the collections from 1,440 to 1,700 m have an average knob number of 5, and the collections from the highest altitudes (1,740 to 2,440 m) have the mean knob number of 4.5. Thus, the strains collected from Sikkim do not differ in knob number with the change of altitude. These observations may also be interpreted to indicate that maize collections from different parts of Sikkim have a common origin.

However, the status of the Sikkim Primitive types is quite different. Of the seven strains of the S.P. type studied, four (S-18, S-23, T-1, and T-2) have a

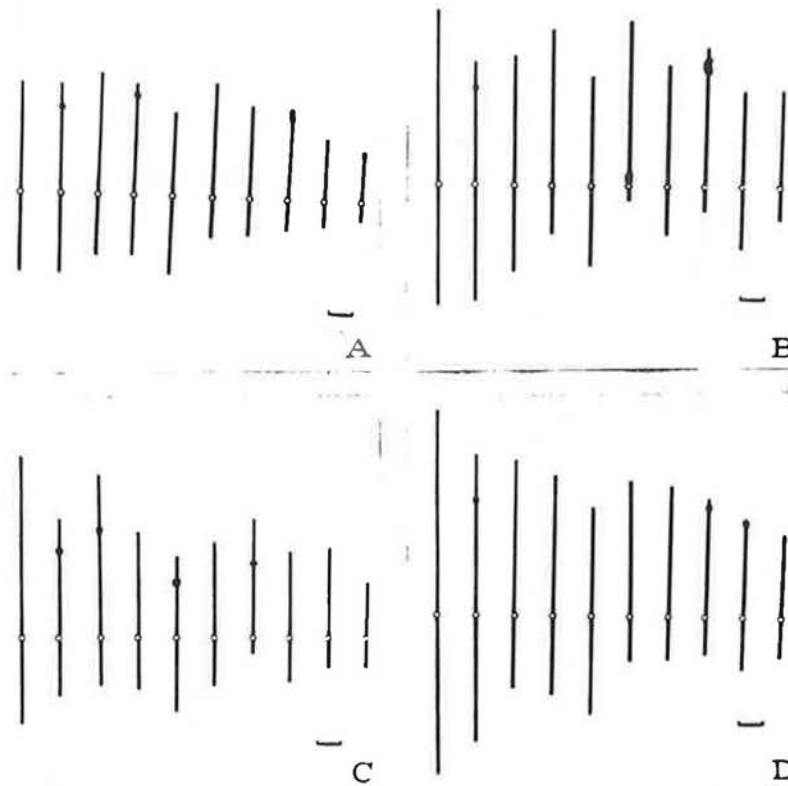
high mean knob number of 9, while three (M-1, M-15 and M-25), although classified as S.P. types on the basis of morphological similarities (MNL 56:122-123, 1982), appear to be different in lineage with a very low average knob number of 2.5.

Table 1. Distribution of knobs at pachytene.

S. No.	Strains	Altitude in meter	Mean knob number	Position of knob	
				Terminal	Subterminal
<u>N.E.H. strains</u>					
1.	S-18 (S.P.)	1500	6	1	5
2.	S-23 (S.P.)	1300	8	1	7
3.	M-1 (S.P.)	1200	3	1	2
4.	M-15 (S.P.)	1200	3	1	2
5.	M-25 (S.P.)	1350	2	1	1
6.	T-1 (S.P.)	1050	9		
7.	T-2 (S.P.)	1050	11	2	9
8.	S-58	1200	2	-	2
9.	S-16	1300	5	1	4
10.	S-29	1300	4	2	2
11.	S-57	1320	6	1	5
12.	S-35	1440	3	-	3
13.	S-20	1450	4	-	4
14.	S-31	1520	8	-	8
15.	S-45	1600	3	1	2
16.	S-25	1640	3	-	3
17.	S-55	1700	4	1	3
18.	S-28	1740	3	1	2
19.	S-59	2400	3	-	3
20.	S-21	2400	3	1	2
21.	S-30	2400	2	-	2
22.	S-56	2400	2	-	2
23.	S-38	2440	7	1	6
24.	S-39	2400	3	-	3
25.	M-14	1200	3	1	2
26.	M-26	1200	4	1	3
27.	M-27	1200	5	1	4
28.	M-9	1350	5	1	4
29.	T-26	1050	6	2	4
<u>American races of maize</u>					
30.	Confite Morocho		4	-	4
31.	Pira		13	-	13

S (Sikkim); M (Meghalaya); T (Tripura) and S.P. (Sikkim Primitive)

Of the two American races studied, Pira has a mean knob number of 13 and Confite Morocho, 4. Both these races have knobs at subterminal positions.



Pachytene chromosomes showing knob constellation: A, S-29; B, S-35; C, S-20; and D, S-45.

The variation in the knob position, number, shape and size in some of the collections from Sikkim is depicted in the ideograms.

Suchira Pande, J. K. S. Sachan and K. R. Sarkar

Comparison between knobs, C- and Q-bands in maize

Distribution of constitutive heterochromatin which is discernible by knobs, C- and Q-bands in maize was studied in the races of maize from the Americas and maize collections from the Northeastern Himalayas. Maize, being a highly cross-pollinated crop, shows a lot of heterozygosity in the population, and the differences are observable within different kernels of the same ear. Therefore, different plants of the same population also show variation in knob and/or band number.

Tables 1 and 2 show the close correspondence between knobs and C- and Q-bands. M-15, M-25, S-31 and M-14 exhibit an exact correspondence in both number and position of knobs and C-bands. M-1 shows a 1:2 correspondence between knob number and C-bands. The observation can be explained by the fact that, if homologous chromosomes are homozygous for the knob forming region, then double the number of bands will be observed in somatic metaphase, as compared to the pachytene knobs,

Table 1. Comparison between knob and C-band distribution

Strains	Total number of knobs	Knobs		Total number of bands	C-bands	
		Position of knobs Termi- nal	Subtermi- nal		Position of bands Termi- nal	Subtermi- nal
M-1 (S.P.)	3	1	2	6	2	4
M-15 (S.P.)	3	1	2	3	1	2
M-25 (S.P.)	2	1	1	2	1	1
S-35	3	-	3	6	4	2
S-20	4	-	4	8	6	2
S-31	8	-	8	8	-	8
M-14	3	1	2	3	1	2
P-26	6	2	4	6	6	-

M (Meghalaya); S (Sikkim); T (Tripura) and S.P. (SIKKIM Primitive)

where the homologous chromosomes are paired together. S-35 and S-20 also show a 1:2 correspondence between knobs and C-bands, but when the positions are compared, knobs which are subterminal in position were manifested as terminal C-bands because of the greater condensation of the somatic metaphase chromosomes.

Table 2. Comparison between C- and Q-band distribution

Strains	Total number of knobs	Knobs		Total number of bands	Q-bands	
		Position of knobs Termi- nal	Subtermi- nal		Position of bands Termi- nal	Subtermi- nal
M-1 (S.P.)	6	2	4	6	6	-
M-15 (S.P.)	3	1	2	4	2	2
M-25 (S.P.)	2	1	1	4	4	-
N-3 (S.P.)	13	9	4	11	9	2
N-4 (S.P.)	10	8	2	9	7	2
T-2 (S.P.)	12	8	4	12	8	4
S-45	8	4	4	8	4	4
Pira	15	6	9	14	10	4

M (Meghalaya); N (Nagaland); T (Tripura); S (Sikkim) and S.P. (Sikkim Primitive)

T-2 and S-35 have shown an exact 1:1 correspondence between number and position of C- and Q-bands. M-1 shows 1:1 correspondence between number of C- and Q-bands, but subterminal C-bands are observed as terminal Q-bands, as the tails are sometimes very difficult to discern. But in general, distribution and pattern of Q-bands correspond to those of C-bands.

Suchira Pande, J. K. S. Sachan and K. R. Sarkar

Karyotypic comparison between maize and its wild relatives

In order to compare the basic karyotype of maize with that of teosinte, Coix, Trilobachne, Chionachne and Manisuris, mitotic metaphase studies were carried out with conventional Feulgen procedure in growing root tips. The basic data on chromosomal types and form percent (Table 1), arm ratio (Table 2), and relative length (Table 3) are presented in the tables.

Table 1: Comparison of some of the karyotypic characters among the genera of Maydeae and Manisuris.

S.No.	Material	No. of chromosome pairs of				Total arm percentage	Total length of Genome (μm)
		Metacentric	Submeta-centrics	Telo-centrics	with satel-lite.		
1.	Palomero Toluqueno (2n=20)	5	4	1*	1	38.58	114.66
2.	Sikkim primitive (2n = 20)	4	5*	1	1	41.71	88.95
3.	Celaya (2n = 20)	1	9*	-	-	39.48	74.98
4.	<u>Zea luxurians</u> (2n=20)	1	5	4*	1	32.58	85.22
5.	<u>Z. mexicana</u> (2n = 20)	1	6	3*	1	34.32	76.92
6.	<u>Manisuris selloana</u> (2n=18)	4	1	4*	1	40.15	67.16
7.	<u>Coix aquatica</u> (2n = 10)	1*	3	1*	2	35.20	46.65
8.	<u>C. lacryma-jobi wild</u> (2n=20)	2	5	3*	3	37.00	77.28
9.	<u>C. lacryma-jobi Coix-31</u> (2n=20)	5	5*	-	1	42.80	62.75
10.	<u>Trilobachne cookei</u> (2n=20)	2*	2	6	1	30.76	121.21
11.	<u>Chionachne koenigii</u> (2n=20)	1	9*	-	1	38.90	97.31

*Arm ratio range from 1.00-1.25 for metacentric 1.26-1.75 for submetacentric and 1.7 and above for telocentric chromosomes.

*Category in which Sat. chromosome falls.

Table 1: Comparison of chromosome arm ratios among the different genera of Maydeae and Manisuris.

S.No.	Chromosome										
	I	II	III	IV	V	VI	VII	VIII	IX	X	
1.	Palomero Toluqueno	1.67	1.23	1.14	1.24	1.19	1.76	1.20	1.75	1.53	1.44
2.	Sikkim Primitive	1.07	1.10	1.45	1.03	2.72	1.62	1.39	1.39	1.19	1.31
3.	Celaya	1.29	1.52	1.26	1.40	1.43	1.53	1.35	1.44	1.55	1.20
4.	<u>Zea luxurians</u>	1.22	1.40	1.59	1.30	1.46	2.24	2.70	2.50	2.14	1.57
5.	<u>Zea mexicana</u>	1.27	1.20	1.51	1.61	1.73	2.24	2.74	1.75	2.53	1.39
6.	<u>Manisuris selloana</u>	1.37	1.11	2.01	1.86	1.85	1.80	1.12	1.14	1.23	-
7.	<u>Coix aquatica</u>	1.77	1.18	1.46	1.33	1.47	-	-	-	-	-
8.	<u>C. lacryma-jobi (wild)</u>	1.87	1.06	1.61	2.03	1.98	1.34	1.41	1.39	1.70	1.17
9.	<u>C. lacryma-jobi (cultivated)</u>	1.46	1.16	1.00	1.16	1.21	1.26	1.70	1.68	1.10	1.68
10.	<u>Trilobachne cookei</u>	1.09	1.74	1.72	1.89	1.21	6.87	4.67	7.51	4.36	7.00
11.	<u>Chionachne Koenigii</u>	1.37	1.63	1.45	1.40	1.29	1.23	1.48	1.32	1.31	1.63

Table 3: Comparison of relative length of individual chromosomes in the different genera of Maydeae and Manisuris.

S.No.	Material	Chromosome (length in μm)									
		I	II	III	IV	V	VI	VII*	VIII	IX	X
1.	Palomero Toluqueno	6.40	6.00	5.65	5.41	4.87	5.22*	4.30	3.70	3.67	3.38
2.	Sikkim primitive	6.14	5.72	5.57	5.53	5-50	4.85*	5.45	4.36	3.49	3.37
3.	Celaya	5.67	5.56	5.51	5.36	5-19	5.81*	4.95	4.15	4.01	3.56
4.	<u>Zea luxurians</u>	6.36	5.55	5.43	4.85	4.18	4.96*	3.89	3.63	3.52	3.19
5.	<u>Zea mexicana</u>	6.63	6.62	5.95	5.22	4.70	5.50*	4.57	4.01	3.86	2.96
6.	<u>Manisuris seloana</u>	7.43	6.89	6.21	5.58	5.22	5.11*	4.88	4.47	4.23	-
7.	<u>Coix aquatica</u>	15.77*	11.37*	9.87	6.97	6.27	-	-	-	-	-
8.	<u>C. lacryma-jobi</u> (wild)	8.31*	6.04	5.99	5.68*	5.15*	4.81	4.09	3.89	3.82	2.26
9.	<u>C. lacryma-jobi</u> (cultivated Coix 31)	5.83*	5.49	5.26	5.25	5.11	5.10	4.74	4.57	4.43	4.25
10.	<u>Trilobachne Cookei</u>	7.09	6.66	6.58	6.01	5.75*	3.90	3.62	3.50	3.45	3.36
11.	<u>Chionachne Koenigii</u>	6.65	6.53	5.93	5.37	5.25*	5.00	4.29	4.25	3.65	3.10

* Satellited pair

Both maize and teosinte have shown similar karyotypes. All maize races showed $2n=20$, with almost similar total form percent (T.F.%). Variation in arm ratio fell in the range of 6.40 to 3.37. Both teosinte species, Zea mexicana and Zea luxurians, showed similar karyotypes with arm ratio variability of 1.20 to 2.74 and similar T.F.%. Both maize and teosinte had chromosome 6 satellited.

Among the oriental genera of Maydeae, Coix species revealed a lot of intra- and interspecific variations. Coix aquatica had $2n=10$, whereas both the cultivated and wild forms of Coix lacryma-jobi had $2n=20$ counts. Coix aquatica showed two pairs of satellites, one each on chromosomes 1 and 2. Wild Coix had three pairs of satellites on chromosomes 1, 4 and 5. The short arm of chromosome 1 exhibited two tandem satellites. The cultivated Coix had only one pair of satellites, on chromosome 1. The relative length was largest in Coix aquatica followed by wild and cultivated Coix. The T.F.% of Coix aquatica came closer to that of the wild form and differed from the cultivated form.

Trilobachne cookei gave a bimodal karyotype with $2n=20$ having 12 telocentric, four metacentric and four submetacentric chromosomes. Arm ratio varied greatly, from 1.09 to 7.51. Chionachne koenigii too showed a $2n=20$ karyotype with 18 submetacentric and two metacentric chromosomes, indicating a highly symmetrical karyotype. Both Trilobachne and Chionachne showed chromosome 5 as satellited.

Thus, clearcut basic differences in karyotypes between the American and oriental genera of Maydeae are indicated.

V. V. Shenoy, J. K. S. Sachan and K. R. Sarkar

Comparative study of chromosome banding in maize and its wild relatives

Striking differences in the distribution and pattern of C- and Q-bands between the American and oriental genera of Maydeae have been noted. Both the C- and Q-bands depicted a similar pattern on the chromosomes.

Among the races of maize studied, Nal-Tel showed 16 C-bands followed by Celaya (14), Sikkim Primitive-18 (12) and Palomero Toluqueno (3), while Guatemalan teosinte showed 16 bands and Mexican teosinte 14 bands.

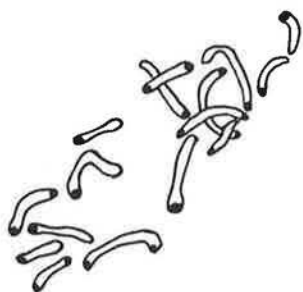


Fig-1

Fig. 1. C-banding pattern in cultivated Coix lacryma-jobi (incomplete complement)

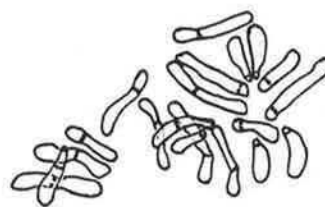


Fig-2

Fig. 2. C-banding pattern in Trilobachne cookei

In Coix, only the cultivated form of Coix lacryma-jobi showed 22 terminal bands (Fig. 1), while other Coix species did not yield any distinct band. Trilobachne was a typical genus which manifested minute centromeric bands in all chromosomes (Fig. 2). Chionachne did not reveal any distinct band, except for a few dark patches.

V. V. Shenoy, J. K. S. Sachan and K. R. Sarkar

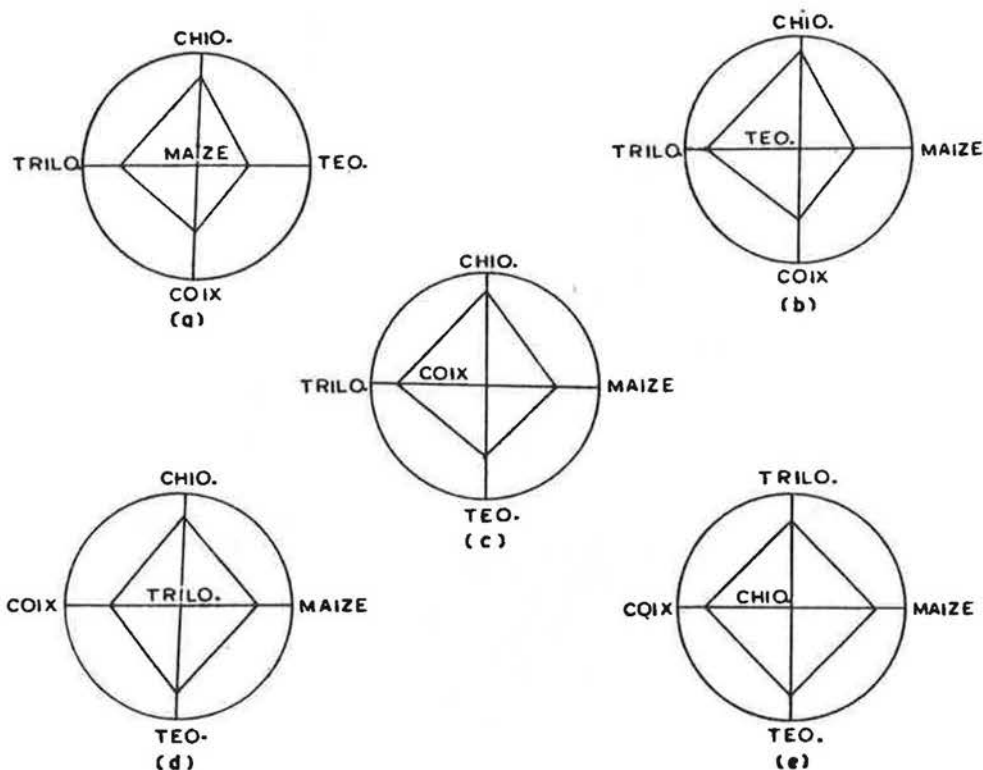
Genetic distance studies in maize and its wild relatives based on biochemical assays

Electrophoretic patterns produced by PAGE of soluble germ proteins and isoenzyme patterns of esterase in soaked germs of races of maize, species of teosinte, species of Coix, Trilobachne and Chionachne were used to calculate similarity indices (S.I.) in all possible combinations. Pooling the similarity indices of isoenzyme esterase and soluble germ proteins gave cumulative similarity indices (C.S.I.). The average similarity indices (A.S.I.) were worked out from C.S.I.'s of races, species and genera. This was found to give a concise picture of interspecific and intergeneric electrophoretic affinities. A.S.I.'s indicated the following similarity hierarchy:

Maize	:Teosinte > <u>Coix</u> > <u>Trilobachne</u> > <u>Chionachne</u>
Teosinte	:Maize > <u>Coix</u> > <u>Trilobachne</u> > <u>Chionachne</u>
<u>Coix</u>	:Maize = <u>Trilobachne</u> > Teosinte = <u>Chionachne</u>
<u>Trilobachne</u>	: <u>Coix</u> > Maize > <u>Chionachne</u> > Teosinte
<u>Chionachne</u>	: <u>Trilobachne</u> > maize > <u>Coix</u> > Teosinte

The comparison of A.S.I.'s revealed that Chionachne showed the least similarity with other genera, followed by Trilobachne and Coix. The similarity pattern of maize and teosinte was quite alike.

Fig. Polygon graphs of genetic distances. (a) maize, (b) teosinte, (c) Coix, (d) Trilobachne, (e) Chionachne. Distances from the center along the radii indicate the genetic distance



Values of genetic distances obtained were represented as polygon graphs. A comparative study of these graphs revealed that maize and teosinte were more or less equidistant from other genera. While the distance between maize and teosinte was less than 50 units, other genera were separated from them by at least 65 units. Maize was nearer to teosinte, followed by Coix, Trilobachne and Chionachne. A similar situation was observed with teosinte also. Coix polygon indicated equidistant placement of maize and teosinte, while Chionachne was the farthest and Trilobachne fell in between. Trilobachne exhibited nearly equidistant placement to Chionachne and teosinte along one diagonal, maize and Coix on the other. The squarish polygon of Chionachne indicated near equidistance of this genera with other genera of Maydeae.

V. V. Shenoy, K. N. Srivastava, J. K. S. Sachan and K. R. Sarkar

Genetic distance studies on maize and teosinte based on biochemical assays

Electrophoretic pattern of seed proteins, and isoenzymes, namely peroxidase and esterase at the young seedling stage have been studied among the races of maize and teosinte. The homology between all pairs of the sample was calculated, and the similarity indices were obtained for proteins, peroxidase and esterase isoenzymes. A cumulative similarity index was prepared by adding the similarity indices between the pairs of races/species studied. Genetic distances were

worked out from these cumulative similarities indices. Phylogenetic relationship is depicted in a two-dimensional diagram with the point of best fit.

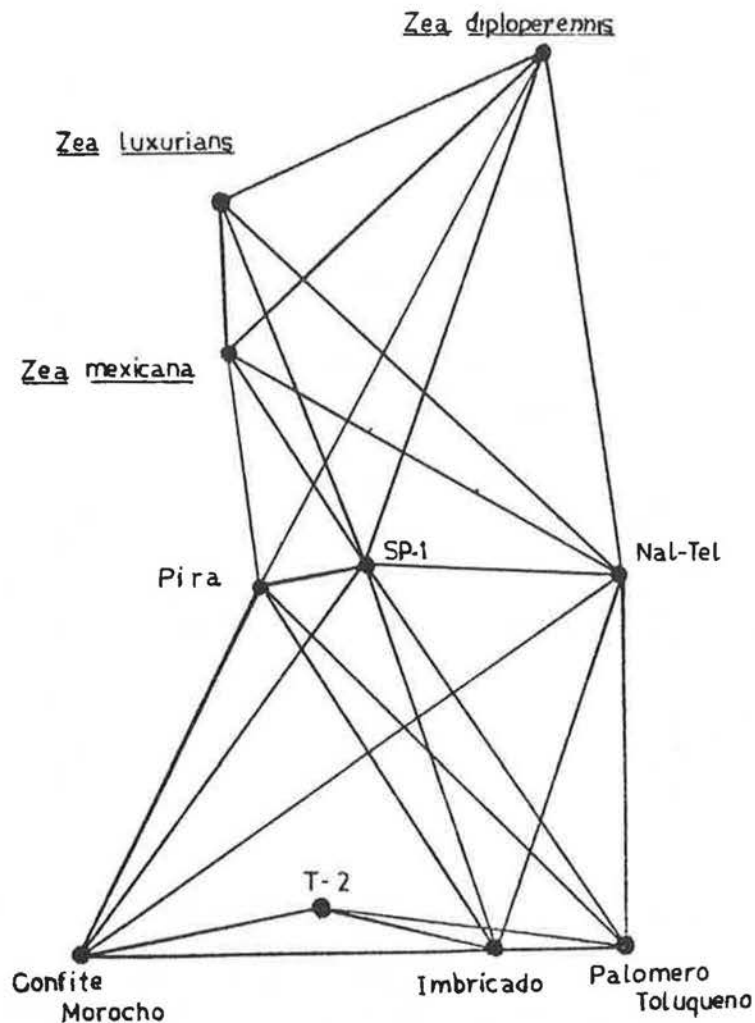


Fig: Genetic distance diagram showing phylogenetic relationship between maize and teosinte.

The material studied can be divided into three main groups with teosinte represented by *Zea mexicana*, *Zea luxurians* and *Zea diploperennis* at one end, and the maize races having low knob number (Confite Morocho, Imbricado and Palomero Toluqueno) at the other extreme. The races Nal-Tel, Pira and Arrocillo Amarillo and Sikkim Primitive (S.P.-1) lie in the middle. The races Nal-Tel and Pira have been recognized a long time as having been introgressed by teosinte. The grouping of S.P.-1 with Pira and Nal-Tel indicates that so-called Sikkim Primitive of the northeastern Himalayas is an advanced form of maize.

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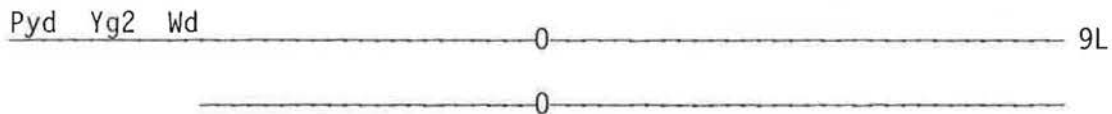
A new assay capable of distinguishing between gene mutations and deficiencies of the Yg2 locus in maize

One widely used maize test system for mutagenesis utilizes the yellow-green-2 (Yg2) locus. In several studies, dry or soaked kernels heterozygous for this locus have been treated with chemical or physical agents, and the resultant seedlings were classified for the frequency of sectors expressing the recessive (yellow-green) phenotype (Latterell and Steffensen, 1962, *Am. J. Bot.* 49:478; Smith and Rossi, 1966, *Rad. Res.* 28:302; Conger and Carabia, 1977, *Mut. Res.* 46:285; Fujii, 1980, *Jap. J. Genet.* 55:241; Plewa, 1982, pp. 411-419 in *Environmental Mutagens and Carcinogens*, ed. Sugimura, Kondo, and Takabe; and several other studies). For studies of the mutagenic activity of physical and chemical agents, the fourth and fifth leaves are typically classified. In these studies, it is usually acknowledged that the mutant sectors can originate in two distinct ways: (1) by mutation of the dominant allele at the yellow-green locus or (2) by a deficiency of the dominant allele of this locus. Because plants heterozygous for large terminal deficiencies of this chromosome, including much of the short arm, are known to be viable (McClintock, 1943, *Carnegie Inst. Wash. Yearbook* 42:148), and plants homozygous for smaller deficiencies including this locus are also viable (McClintock, 1944, *Genetics* 29:478), it is likely that a significant proportion of the sectors of yellow-green phenotype are due to deletions of the Yg2 locus. Furthermore, loss of an entire chromosome 9 in this test system would be tolerated because plants monosomic for chromosome 9 survive to maturity and are quite vigorous (Weber, 1973; *Theor. Appl. Genet.* 43:167).

Clearly, it is important to be able to distinguish between these two types of mutational events (terminal deficiencies including the Yg2 locus and gene mutations at the Yg2 locus) in mutagenesis studies. In this paper, I propose a modification of this test system which can be used to distinguish between these two types of events. It is also important to recognize that part of the sectors of yellow-green phenotype could also be due to events which do not involve the Yg2 locus. For this reason, an additional control series should be included in these tests. This neglected important control series will be discussed at the end of this report.

McClintock (1944) generated a large series of terminal deficiencies involving the short arm of chromosome 9 in maize, and she determined that plants homozygous for a deficiency including the knob and half of the most distal chromomere were albino. Thus, a factor which is necessary for chlorophyll synthesis lies distal to this breakpoint very close to the end of the short arm of chromosome 9. She named this factor the Wd factor (for white deficiency). A chromosome with this terminal deficiency is termed a wd chromosome. She also found that plants homozygous for an even smaller terminal deficiency of the short arm of chromosome 9 (deficient for the region distal to the first chromomere) were pale yellow. Thus, another factor, Pyd (for pale-yellow deficiency), is located distal to the wd locus, and the pale-yellow deficiency uncovered this locus. She also determined that the yg2 locus was uncovered by the wd deficiency but not by the pyd deficiency; thus, the yg2 locus lies between the wd and pyd breakpoints and all three factors are extremely close to each other or near the end of the short arm of chromosome nine distal to the terminal half of the most terminal chromomere.

The wd/Yg2 test system: Maize plants can be easily generated which carry one normal chromosome 9 containing the dominant alleles of the Yg2, Pyd, and Wd loci and a chromosome 9 carrying the wd deficiency (which deletes all three loci):



This plant is hemizygous for all three loci. Kernels of this genotype could then be exposed to chemical or physical agents. If a forward mutation occurred at the Pyd, Yg2, or Wd locus, a pale-yellow, yellow-green, or albino sector on an otherwise green leaf would be produced. However, if a break occurred proximal to the Wd locus in the normal chromosome, an albino phenotype would be produced because the Wd locus is deleted. Mutations at the Wd locus and deficiencies including the Wd locus would both be expressed as the same mutant phenotype. However, it is not unreasonable to assume that the forward mutation frequency determined at this locus would be similar to the mutation frequencies present at the other two test loci, and this estimated mutation frequency could then be subtracted from the total frequency of albino sectors. In this way, it would be possible to correct for forward mutation at the Wd locus. Albino sectors generated by loss or mutation of the Wd locus should be easily distinguishable from yellow-green and pale-yellow sectors generated by mutation at those loci; however, it is possible that pale-yellow and yellow-green sectors cannot be distinguished from each other.

It is important to note that the Pyd, Yg2 and Wd loci are extremely close to each other near the tip of chromosome 9, and it would be an extremely rare event where a break occurred distal to the Wd locus but proximal to the Yg2 or Pyd locus to generate a sector of yellow-green or pale-yellow. For this reason, expression of the Yg2 or Pyd recessive phenotypes would almost invariably be due to gene mutation at the respective loci. On the other hand, any breakpoint proximal to the Wd locus on the chromosome bearing the dominant alleles will generate an albino sector.

Seed of the above type would be treated and the frequency of albino, pale-yellow, and yellow-green sectors determined on the fourth or fifth leaves. The forward mutation frequencies at the Pyd and Yg2 loci are the number of yellow-green or pale-yellow sectors divided by the number of progenitor cells present in the kernel at the time of treatment (minus the frequency in the untreated control). The frequency of loss of the Wd locus is the frequency of albino sectors minus the mean frequency of yellow-green and pale-yellow sectors.

Generation of the test plants: The plants used in the above test system can easily be generated in very large numbers by making the cross described below. Plants homozygous for the Wd deficiency, homozygous for the C locus and all other dominant seed color factors, and also containing the Wd ring chromosome (which contains the dominant alleles of the Wd, Pyd, and Yg2 loci and also C-I which is dominant to C and inhibits anthocyanin synthesis) could be crossed by nearly any line of corn as either the male or female parent. The dominant alleles of the Wd, Pyd and Yg2 loci will be present in any true-breeding, non-chlorotic line of corn, including commercial varieties, and all factors necessary for kernel pigmentation are contributed by the ring-containing parent. Two types of progeny are produced, those containing the ring chromosome and those lacking the ring. The ring-containing progeny can be easily identified because the C-I locus is present on the ring, and kernels containing the ring have colorless endosperm (in tissue where the ring chromosome is present) with sectors of colored tissue (where the ring is lost). Kernels lacking the ring chromosome are colored because the C-I locus is not present, and these represent the vast majority of the progeny because small ring chromosomes are unstable, are frequently lost, and are present in only a small proportion of the cells of the organism (McClintock,

1938, *Genetics* 23:315). The latter kernel type (which lacks the Wd ring) would be used in mutagenesis studies described above. Kernels of this type are being generated and will be used in mutagenic studies in the near future. wd/wd ring-containing germplasm is available from several laboratories, including ours, and plants of this type can be increased simply by selfing or sibling. The ring-containing wd/wd kernels from such a self can be readily identified because they have endosperm which is colorless and also contain purple sectors.

A neglected important control: Another shortcoming of tests using plants heterozygous for the Yg2 locus is that it is not known if some of the mutant sectors are due to events other than mutation or loss of the Yg2 locus. It is not unlikely that dominant mutations at other loci can occur which produce a mutant phenotype indistinguishable from the Yg2 phenotype. Also, in my work with monosomics, I have found several plants in which half of each leaf is lighter green than the other half, and this altered phenotype is associated with monosomy. In these cases, the marker mutations utilized in selection of monosomic types (g and j) are not associated with the altered phenotype (Weber, 1973, *Theor. Appl. Genet.* 43:167; 1982, pp. 79-83 in *Maize for Biological Research*, W. F. Sheridan, ed.). Clearly, hemizygoty of certain unknown chromosomal segments can result in a lighter leaf phenotype, and this phenotype is not associated with the Yg2 locus. For this reason, an important control for this type of work would be to treat homozygous dominant (Yg2/Yg2) plants with the agent being tested to determine if the agent causes mutant sectors which are not associated with loss or mutation of the Yg2 locus. If such sectors are found, the frequency of such sectors minus the square of the mutant sector frequency in the experimental group (to correct for types where mutations took place at both Yg2 loci) would need to be subtracted from the frequency in the experimental group to correct for events which do not involve the Yg2 locus.

In fact, Neuffer (1966, *Genetics* 53:541) noted that this case was indeed found in plants grown from EMS-treated seed. Although no data were given, he mentioned that the frequency of mutant sectors on leaves of Yg2/Yg2 plants was higher than the square of the frequency found in Yg2/yg2 plants. It is important that these control plants be included in future tests. (Supported in part by DOE Contract 79EV02121.)

David Weber

Location of the bx locus in maize to the short arm of chromosome four by monosomic and B-A translocational analysis

The bx locus in maize is required for the production of cyclic hydroxamates. Root tips carrying a dominant allele of this locus form a deep blue-purple color complex when they are crushed in the presence of a 0.1N solution of ferric chloride while root tips with the recessive allele will not form a color complex.

F1 progeny of a cross between R/r-X1; Bx/Bx female parents and r/r; bx/bx male parents were germinated and screened for the presence of cyclic hydroxamates. Eighteen out of 8074 r-X1 deficiency-containing (colorless) kernels expressed the recessive bx phenotype, and root-tip chromosome counts indicated that they were monosomic individuals. Measurement of mitotic metaphase chromosomes indicated that the missing chromosome was a long metacentric chromosome, and this information was used to tentatively assign the bx locus to chromosome five (Weber, 1982, pp. 79-83 in *Maize for Biological Research*, W. F. Sheridan, ed.). However, when these monosomic plants were grown to maturity, the morphology of the monosomic plants was characteristic of plants monosomic for chromosome four, and pachytene analysis of these plants indicated that the univalent chromosome was chromosome four.

To confirm the cytological identification, B-A translocations for both arms of chromosomes 1, 2, 3, 4, and 5 were crossed by a Bx/bx heterozygote, and only TB-4S uncovered the recessive bx allele. Clearly, the bx locus is located distal to the breakpoint of TB-4S on the short arm of chromosome 4. (Supported in part by DOE Contract 79EV02121.)

Kevin Simcox and David Weber

Location of the $Px3$ locus to chromosome seven by monosomic analysis

Several different isozymic variants are known for the $Px3$ locus. $R/r-X1$ plants which contained a slow allele of the $Px3$ locus were crossed by Mangelsdorf's multiple chromosome tester which was homozygous recessive for marker alleles on all 10 maize chromosomes, was r/r , and also contained a fast allele of the $Px3$ locus. The corresponding dominant marker alleles were present in the female deficiency-bearing parent. Diploid F1 progeny and most monosomic types displayed two bands on acrylamide gels while plants monosomic for chromosome 7 (identified by the glossy marker mutation) contained only the rapidly-migrating band. Clearly, the $Px3$ locus is located on chromosome 7. Details of procedures used to generate and identify monosomics in maize are given in Weber, 1982, pp. 79-83 in *Maize for Biological Research*, W. F. Sheridan, ed. (Supported in part by DOE Contract 79EV02121.)

David Weber and J. L. Brewbaker

Separation of low molecular weight metabolites from maize leaves by high performance liquid chromatography

A relatively new technique in plant analysis is the use of chromatography to separate up to 100 or more components in a single sample, producing complex chromatograms, which have been termed "metabolic profiles." Metabolic profiling, because it measures so many different substances, may be useful in examining problems such as pathogen resistance, which have been very difficult to study by techniques which measure or separate only a few substances at one time. In our laboratory, we have therefore begun using metabolic profiling to examine the molecular basis of pathogen resistance in maize. We have developed simple methods for extruding water-soluble compounds from maize leaves and have used high performance liquid chromatography (HPLC) to measure the relative amounts of more than 50 components in these samples. We have investigated profiles from several varieties of maize, including lines isogenic for a specific pathogen resistance. This report describes our initial results from such studies.

One-half gram of maize leaves from 18-day-old seedlings raised in a greenhouse in sand were squeezed in a press designed for preparing KBr pellets for infrared spectrometry. The "juice" was filtered through a 0.22 micron filter and 20 microliters injected into a dual-pump, microprocessor-controlled HPLC. The components in the sample were separated on a reverse phase analytical column, and detected using two UV detectors and an electrochemical detector (Figure 1). The HPLC mobile phase consisted of a pH 2.1 phosphate buffer (0.2M)/acetonitrile gradient. We collected data on a minicomputer which has hard disks and the necessary peripherals for data acquisition, storage, and plotting.

The peak areas of each chromatogram were calculated by the computer system and compared. For the 40 to 50 peaks that could be accurately measured by the computer, we observed an overall precision of 15% (median relative standard deviation) when "corn squeezings" from subsamples of the same sample were each analyzed on the HPLC. The sample preparation time is less than 15 minutes; thus, sample degradation is minimized. The time required to analyze each sample with the HPLC is about one hour. Thus we can quantitate nearly one peak per minute.

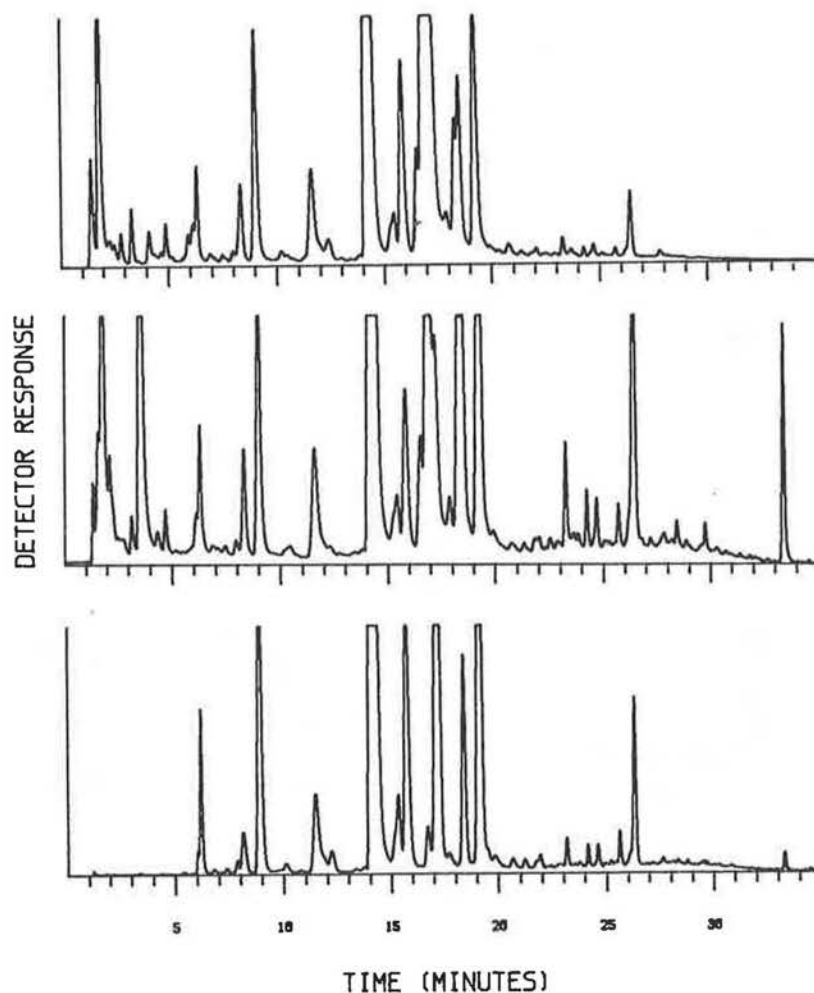


Fig. 1. Profiles of a single A632 plant simultaneously produced by three detectors. Top, electrochemical detector at 1.0 V; middle, UV detector at 280 nm; bottom, UV detector at 320 nm. Note that each detector gives a unique profile. Differences in the response of the three detectors can also be used to gauge the purity of individual peaks.

We have examined a number of maize inbreds and found that each inbred produces a unique profile (Figure 2). As expected, closely-related inbreds have highly similar but distinguishable profiles. Clearly, the genotype of the inbred is reflected in the profile (phenotype).

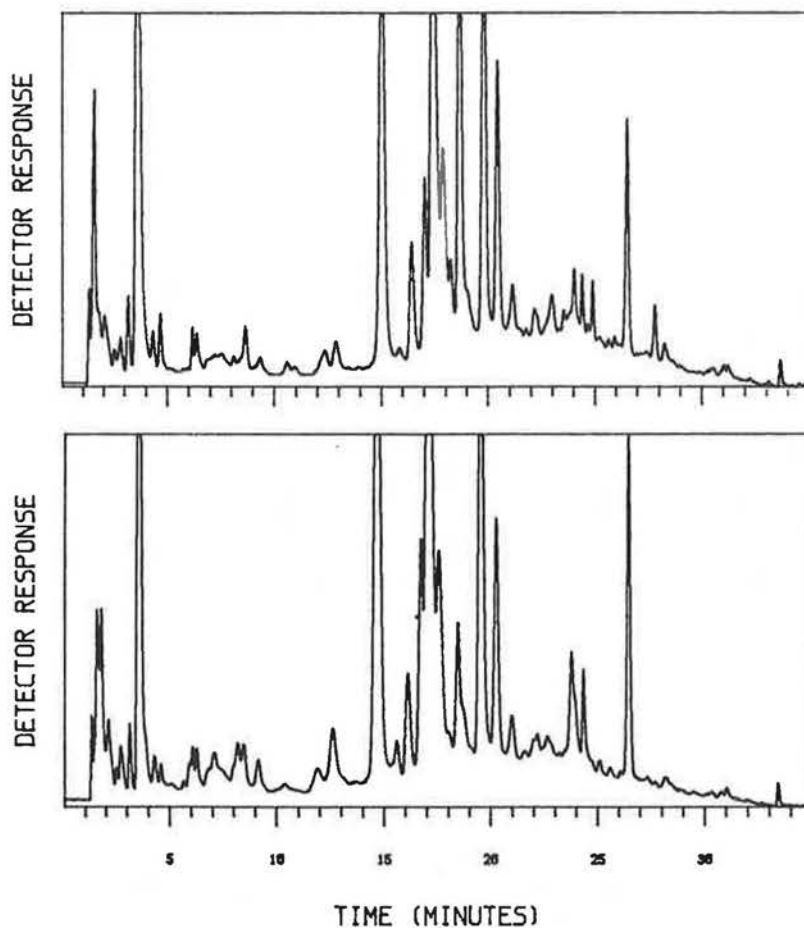


Fig. 2. Profiles of W64A (top) and A619 (bottom) at 280 nm. Note the differences between the profiles.

Plants which are known to be resistant to a specific disease, and "isogenic" plants which are not resistant to that disease but which are otherwise genetically as similar as possible, were also analyzed using this technique. Profiles showed clear differences between resistant and susceptible types (Figure 3).

Most of the compounds which we detect in these samples appear to be low molecular weight organic acids and amino acids; the technique is particularly sensitive to phenolic acids. Thus, the compounds detectable by the HPLC include many of the known primary and secondary metabolic intermediates in maize, and many of the substances already known to be involved in disease resistance. It will clearly be necessary: (1) to run much larger numbers of samples, (2) to chemically identify compounds putatively associated with the resistance or other trait, and (3) to perform similar tests on samples from segregating backcrosses before the results from such studies can be accepted as demonstrating that these compounds are in fact associated with that trait. Even if the compound(s) thus

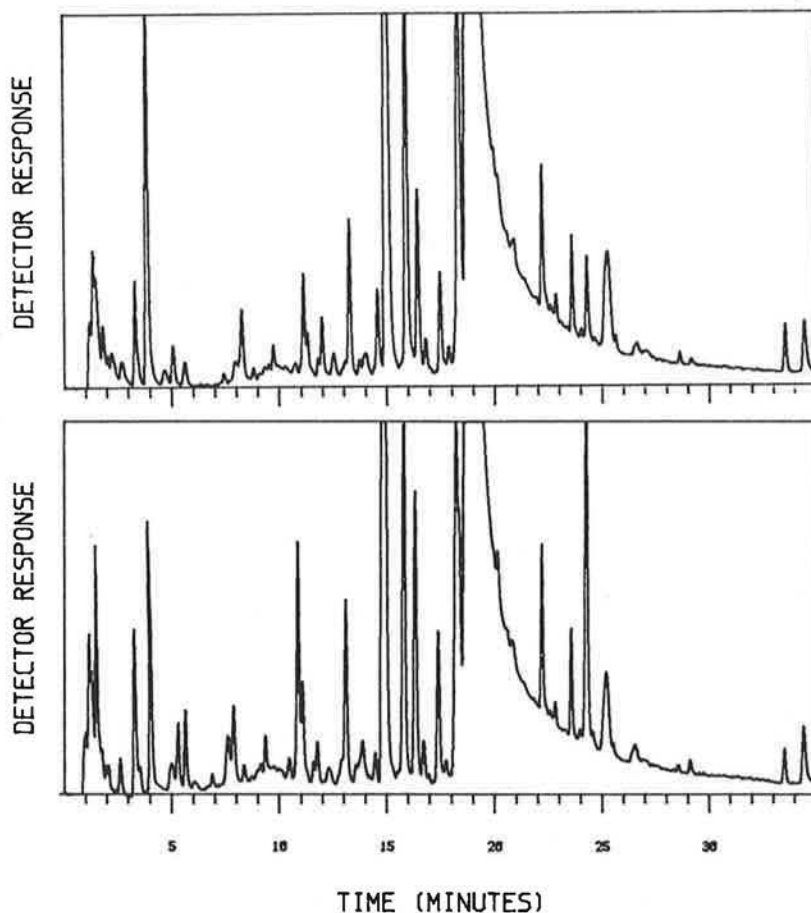


Fig. 3. HPLC profiles taken from maize leaves of two varieties which differ primarily in being susceptible (top, variety B37) and resistant (bottom, variety B37-Ht) to the pathogen *H. turcicum*. Note that when the peak heights are different in the two profiles, the peaks are usually larger in the bottom profile, indicating that the levels of these compounds are higher in the resistant type. The profiles were obtained at 254 nm.

identified are not the direct causal agents of the trait, they may prove useful in the eventual elucidation of the trait at the molecular level. However, we anticipate that this technique will eventually prove useful in studying not only disease resistance, but also other problems of agronomic significance, and that plant breeders may eventually be able to use specific compounds as markers in helping them to design crosses to achieve specific goals.

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Somatic embryogenesis and plant regeneration from callus cultures

Recently it has been demonstrated that cells of certain tissues of cereal and grass species can express totipotency via somatic embryogenesis (V. Vasil and I. K. Vasil, TAG 56:97, 1980). In 1982, C. Lu et al. (TAG 62:109, 1982) and C. E. Green (Proc. V Intern. Congr. Plant Cell Tissue Cult., Tokyo, Abstr., 1982) reported on somatic embryogenesis in *Zea mays*.

Immature embryos (1.1 - 1.5 mm in length) were taken from selfed plants of Chinese material Chi 31, a genotype appropriate to being used in maize tissue culture work (Y.-j. Xie and B. Gengenbach, MNL 55:95, 1981). Embryos were cultured with the scutellum facing upwards and the plumule and radicle sides in contact with agar. The MS and N-6 media (Chu et al., Sci. Sin. 18:659, 1975) were supplemented with 2.5 μ M 2,4-D and 120 g/l sucrose (Lu et al.). The cultures were maintained at 25 °C under 16-hour photoperiod. A compact, yellowish opaque callus was produced by proliferation of the scutellum one week after the initiation of the culture. The embryo axis was not involved in callus formation. Well-organized somatic embryos were formed on the surface of the scutellar callus within 2 to 3 weeks of the culture. The embryoids formed showed characteristic features (similar to zygotic maize embryos)--scutellum, coleoptile and coleorhiza. Numerous atypical embryoids were observed, however, especially those with two or more shoot apices. The scutellum of embryoids had a strong tendency toward secondary proliferation and embryogenesis. The germination of embryoids took place on the same basal media with 2.5 μ M 2,4-D. The embryoids transferred singly or in clumps onto MS medium without 2,4-D but, containing 1 ppm GA3 (Lu et al.), germinated irregularly and did not form plants transplantable into soil. Well-developed plants with roots were formed on 2,4-D media. The regenerants were placed into perlite saturated with Hoagland's solutions, and 2 weeks later into potting soil. At present there is a total of 322 plants growing in a greenhouse. Regenerants originated from 90 initial explants and they were formed during 4 months of the culture. The callus is subcultured every 20 days and its embryogenetic capacity is maintained. All regenerated plants were green without any sign of albinism, but abnormal phenotypes occurred in the seedlings: a prostrate growth habit, tillering, leaves with white or yellow stripes, virescent types. Regenerated plants show variability in growth. Chromosomal analysis, and scoring for the presence of mutation in the progenies derived from regenerated plants are in progress.

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Mitotic chromosome analysis of different strains of Maydeae

There have been only a few studies dealing with different aspects of mitotic analysis of maize, such as banding, somatic association, karyotype vs. translocations, endosperm cytology, etc. The comparatively insignificant work done with the mitotic material is attributable to several technical problems, viz. difficulty of securing uniform one-celled thick squashes in view of the very hard nature of the root tip, and very heavy cytoplasmic content. Filion (MNL 42:175, 1968) further pointed out that maize chromosome arms would not spread very

widely, and as such it would be difficult to locate accurately the centromere and to define the limits of arms. In view of the lacunae in our knowledge in understanding the mitotic chromosomes of maize, and also in view of the immense potential of improved methods of chromosome analysis, it was deemed worthwhile to make an attempt to unravel the variation patterns of somatic chromosomes of maize. The present report comprises a detailed investigation of somatic chromosomes of a representative cross-section of the cytological diversity existing in maize (mostly from Indian sources). A cultivar of teosinte and another of Coix lacryma-jobi were included for comparison.

The treatment schedule for mitotic analysis is as follows:

1. Pretreatment: 1:1 mixture of saturated bromonaphthalene and 0.05% colchicine, for 2 hr 45 min at 10-12 C (including three minutes chilling time)
2. Fixation: 1:3 acetic-ethanol overnight
3. Preservation: 70% ethanol
4. Maceration: 5% aqueous solution of pectinase at 37 C for 2 hr
5. Clearing: 45% acetic acid at room temperature for 10 minutes
6. Staining: 2% aceto-orcein overnight at room temperature

For Feulgen staining, the pretreated, fixed and macerated root tips were hydrolyzed in 1N HCl at 60 C for 15 min, and then stained in Feulgen solution for a period of 2 hr. Alternatively, the maceration might also be carried out after staining, but at room temperature and for a shorter duration.

Based on the nature of primary and secondary constrictions, the chromosomes of the cultivars studied were classified under three types:

A-type: Chromosomes with two constrictions; three subtypes were recognized.

A1-type: Chromosomes with two constrictions, with median to nearly median primary constriction and submedian to nearly submedian secondary constriction.

A2-type: Chromosomes with two constrictions, with median to nearly median primary constriction and subterminal to nearly subterminal secondary constriction.

A3-type: Chromosomes with two constrictions, with submedian to nearly submedian primary constriction and subterminal to nearly subterminal secondary constriction.

B-type: Chromosomes with median to nearly median primary constriction.

C-type: Chromosomes with submedian to nearly submedian primary constriction.

Even though B- and C-type chromosomes would actually form one type, they were classified based on the centromeric index.

The details of the comparative karyomorphology of the different cultivars studied are presented in Table 1. The karyotype is graded. The qualitative features of the ideograms are relatively uniform. The karyotypes of teosinte and Coix do not differ more than those of cultivars of maize.

Table 1: Comparative karyomorphology of different cultivars of maydeae.

Name of the cultivar	Karyotype formula	Range of chromosome length (μ)	T.C.L. (μ)	T.F. (%)	Variations, if any
1	2	3	4	5	6
<u>Cultivars of maize</u>					
1. Hal-Tel (Yucatan-7)	2A ₂ +8B+10C	4.18 - 2.64	31.90	37.24	3-5'B' Chromosomes present
2. Comfite Morocho	2A ₂ + 6B+12C	3.96 - 1.76	26.62	37.19	--
3. Tema Flint	2A ₂ +12B+ 6C	4.40 - 2.42	30.36	42.03	--
4. Sikkim Primitive	2A ₂ + 8B+10C	5.28 - 2.42	34.32	39.10	--
5. Kalimpong Local	2A ₂ +12B+ 6C	5.72 - 2.86	43.34	40.61	Haploid roots present
6. Sonada Local	2A ₂ +10B+ 8C	3.74 - 1.98	25.96	40.68	--
7. Warangal Local	2A ₂ +10B+ 8C	3.52 - 1.54	26.62	39.67	0-2'B' Chromosomes present
8. CH - 104	2A ₂ +12B+ 6C	6.82 - 3.96	50.82	38.96	--
9. CH - 105	2A ₂ + 8B+10C	7.92 - 4.40	56.76	38.76	--
10. CH - 114	2A ₂ +10B+ 8C	6.16 - 2.64	40.48	40.76	--
11. CH - 115	2A ₂ +12B+ 6C	3.96 - 2.20	28.82	41.22	--
12. CH - 201	2A ₂ + 8B+10C	5.94 - 2.86	41.36	37.77	--
13. CH - 206	2A ₂ +12B+ 6C	3.96 - 2.64	34.32	39.74	--
14. Ganga - 5	2A ₂ + 6B+12C	4.40 - 1.98	29.04	39.39	--
15. Ganga Sajed-2	2A ₂ +10B+ 8C	4.18 - 1.98	32.12	39.74	--
16. Globe Hybrid	2A ₂ +10B+ 8C	5.94 - 2.86	41.14	40.11	--
17. Golden Bantom	2A ₂ + 8B+10C	6.60 - 3.08	40.48	36.96	--
18. Stowell's Ever Green	2A ₂ + 8B+10C	3.96 - 1.98	29.04	39.39	1-2'B' Chromosome present

...2

Table 1: -::: 2 :::-

1	2	3	4	5	6
19. Vijay	2A ₂ +12B+ 6C	5.06 - 2.42	34.76	40.51	--
20. Diara	2A ₂ + 8B+10C	3.30 - 1.98	27.28	36.29	1-2'B' Chromosomes present; heteromorphic satellited chromosomes
21. Synthetic B19	2A ₂ +12B+ 6C	3.74 - 1.98	27.50	40.80	--
22. Synthetic B23	2A ₂ + 8B+10C	3.52 - 1.32	22.88	36.54	--
<u>Other members of Maydeae:</u>					
23. Annual teasinte	2A ₂ + 8B+10C	5.94 - 3.52	46.42	38.86	--
24. Coix lacryma-jobi	2A ₂ +14B+ 4C	4.62 - 2.42	36.74	43.71	--

T.C.L: Total Chromatin length of the haploid complement (in μ).

T.F. : Total centromeric index (per cent).

The normal somatic chromosome number of all the twenty-four cultivars studied is always twenty, except for occasional increments due to the presence of B-chromosomes in cultivars such as Nal-Tel (Yucatan-7), Warangal Local, Stowell's Evergreen and Diara, which was confirmed by Feulgen staining also. Their number ranges up to a maximum of five, although one or two is the most frequently encountered number. The longest chromosome measures 7.92 u in length, and the shortest chromosome 1.32 u. The total chromatin length of the haploid complements varies from 56.76 u in the case of CM-105, to 22.82 u in the case of Synthetic B23. Maximum TF value is 43.71 in Coix, and the minimum TF value is 36.29 in the cultivar Diara; however, the variation in TF values is relatively less compared to that of the total length of the haploid complements. Chromosomes with secondary constriction are always two in number and A2 is the most frequent type encountered, occurring in sixteen of the twenty-four cultivars studied, followed by A3 and A1 types in six and two cultivars, respectively. More frequently, it is the 6th chromosome in order of length, as is the case within the pachytene complements. The chromosome length within an individual complement ranges gradually from the longest to the shortest chromosome, and no abrupt change in the chromosome length was observed. The features of the ideograms analyzed are more or less uniform. The number of B-type pairs is either 4, 5 or 6, and that of C-type pairs 3, 4, or 5, except in the case of Coix, where there are 7 pairs of B-type and 2 pairs of C-type. Some other variations were also observed, such as the presence of heteromorphic satellited chromosomes in Diara, and the occurrence of haploid roots in Kalimpong Local.

The gross chromosome uniformity of maize is the result of judicious human selection. The presence of gross chromosomal aberrations as the possible cause of the wide range of length variation in the chromosome complements can be ruled out. Addition or deletion of nucleotypic DNA during the formation of the cultivars might possibly have played a role in causing the observed variation. Differential condensation patterns of the chromosomes have also been suggested to result in alterations of chromosome characteristics. In addition, several other factors such as minor aberrations, genetic differences, inherent length variability, nature of the origin of the cultivars and differential response to the treatment, may have acted individually and interacted with each other to produce the wide variation. Thus, mitotic chromosome analysis also brings out significant differences in maize.

Teosinte was regarded as a distinct genus or congeneric or even conspecific with maize. There was also controversy regarding the inclusion of the American and Oriental members in the subtribe Maydeae. The present report does not warrant the segregation of maize and teosinte into distinct genera. The observations further support the inclusion of maize, teosinte and Coix within the Maydeae. (The present investigation was carried out under the supervision of Prof. A. K. Sharma of the Department of Botany, University of Calcutta, Calcutta, India.)

J. S. P. Sarma

Pachytene ideograms, absolute lengths and interstrain differences in maize

A majority of the studies dealing with pachytene cytology of maize characterize the genome in terms of relative length. In the present report, an attempt was made to analyze the pachytene karyomorphology of ten cultivars of maize, mostly of Indian origin, in absolute terms, and the results were compared with those of Longley (J. Agr. Res., 59:475-490) and Maguire (Cytologia, 27:248-257). The patterns of length variability were examined by regression analysis of pooled data. In addition, observations were made regarding other quantitative features of meiosis.

The course of meiosis in all the cultivars is uniformly regular, except for minor quantitative variations in chiasma frequency and the nature of bivalents. The total lengths of the haploid complements range from 860.09 u (in Adecuba) to 512.81 u (in Pira Cundinamarca).

Of the ten cultivars analyzed in the present investigation, three (Stowells Evergreen, Kalimpong Local and Sonada Local) have knobless pachytene chromosomes. The knob number in other cultivars ranges up to a maximum of 11 in the case of Pira Cundinamarca. The knob constitution in some of the cultivars was found to be variable from plant to plant. This is especially true of composites such as Vijay and Diara, and synthetics such as Synthetic B19. The various methods of hybridity, selection and bulking may have contributed to the observed variation. Adecuba, Darjeeling Market, Diara and Synthetic B19 may be considered as low knob numbered, whereas the rest are medium knob numbered, excluding the highly numbered Pira Cundinamarca and the knobless ones. The IKL-1 is the most frequently encountered knob in the present investigation.

Absolute measurements are enormously variable. Arm ratios are more reliable than absolute lengths for chromosome identification. The pooled data of the absolute measurements of eight cultivars are presented in Table 1 along with other relevant statistical parameters.

Table 1: Quantitative characterization of pachytene karyomorphology of maize, based on pooled data

Chromosome number	No. of Observations	Total length			Long arm length			Short arm length			Arm ratio (L.A./S.A.)		
		Mean \pm S.D. in μ	Variance	Coefficient of variation	Mean \pm S.D. in μ	Variance	Coefficient of variation	Mean \pm S.D. in μ	Variance	Coefficient of variation	Mean \pm S.D. in μ	Variance	Coefficient of variation
1	56	88.70 \pm 24.09	580.33	27.16	47.94 \pm 14.71	216.30	30.68	39.69 \pm 10.89	120.78	27.69	1.202 \pm 0.142	0.020	11.814
2	62	75.42 \pm 26.33	693.27	34.91	42.59 \pm 15.31	234.40	35.95	31.40 \pm 10.84	117.51	34.52	1.356 \pm 0.123	0.015	9.071
3	65	74.07 \pm 20.79	432.22	28.07	49.74 \pm 14.63	214.04	29.41	23.40 \pm 6.84	46.79	29.13	2.104 \pm 0.203	0.041	9.648
4	70	67.21 \pm 23.29	542.42	34.65	40.02 \pm 13.91	193.49	34.76	25.34 \pm 8.69	75.52	34.29	1.585 \pm 0.141	0.020	8.896
5	90	62.74 \pm 21.68	470.02	34.56	32.25 \pm 11.10	123.21	34.42	29.15 \pm 10.75	115.56	36.80	1.117 \pm 0.115	0.013	10.295
6	27	54.51 \pm 14.52	210.83	26.64	40.61 \pm 11.06	122.32	27.23	12.54 \pm 3.59	12.89	28.63	3.274 \pm 0.357	0.127	10.904
7	50	57.24 \pm 17.68	312.58	30.89	40.48 \pm 12.77	163.07	31.55	15.37 \pm 4.94	24.40	32.14	2.603 \pm 0.237	0.056	9.105
8	42	59.14 \pm 16.38	260.30	27.70	43.95 \pm 12.59	158.51	28.65	13.88 \pm 3.86	14.90	27.81	3.157 \pm 0.200	0.040	6.335
9	53	49.79 \pm 16.40	268.96	32.94	31.77 \pm 10.52	110.67	33.11	16.69 \pm 5.59	31.25	33.49	1.941 \pm 0.184	0.034	9.480
10	30	47.27 \pm 13.76	189.34	29.11	33.34 \pm 10.44	108.99	31.31	12.75 \pm 3.65	13.32	28.63	2.586 \pm 0.206	0.042	7.966
B	12	15.27 \pm 4.86	23.62	31.83	-	-	-	-	-	-	-	-	-

Significance tests such as t- and F-tests were conducted to compare the means and variances of total length and arm ratios from the present report with those of Maguire's data (op. cit.), the results of which are given in Table 2, along with the latter report.

The mean total lengths of chromosomes 3, 4, 7, 8 and 10 from the present investigation are significantly different, whereas those for chromosomes 6 and 9 are significant at the 5% level but not at the 1% level. However, the means for chromosomes 1, 2 and 5 do not show significant differences. It is to be noted

Table 2: Maguire's (1962) report and results of tests of significance in comparison with the present report.

Chromosome	Maguire's report - Length			t-value	F-value	Maguire's report - Arm ratio			
	n	Mean ± S.D.				n	Mean ± S.D.		
1	271	83.50±19.40		1.750	7.576	52	1.330±0.200	3.779	1.867
2	271	69.20±14.90		2.517	13.089	56	1.420±0.251	1.768	4.072
3	271	64.50±14.90		4.285	8.264	48	2.160±0.558	0.742	9.168
4	271	59.80±12.60		3.698	11.980	73	1.590±0.259	0.093	3.276
5	271	58.40±12.90		2.298	8.569	56	1.160±0.145	1.944	2.364
6	271	48.30±12.00		3.090	15.204	20	3.100±0.841	0.943	5.539
7	271	49.60±10.80		4.104	14.767	57	2.830±0.587	2.542	4.601
8	271	46.30±10.10		6.966	17.321	52	3.060±0.584	1.024	5.665
9	271	44.50±10.70		2.987	12.198	63	1.860±0.375	1.421	3.008
10	271	37.00± 8.80		5.456	22.763	55	2.700±0.498	1.188	2.391

that chromosomes 1, 2 and 5 all have mean arm ratios less than 1.356 (nearly median chromosomes). However, the estimated variances of lengths show significant differences.

With respect to arm ratios, t-values for most of the chromosomes are insignificant. For chromosome 1, the t-value is highly significant and for chromosome 7, the same is significant at the 2.5% level, but not at the 1% level. Estimated variances of arm ratios are highly significant except for chromosome 1, the F-value of which is significant at the 5%, but not at the 1% level.

With respect to comparison of the present data with those of Longley, it is worth mentioning that Maguire obtained insignificant t- and F-values for all the chromosomes except for chromosome 7 (with a reciprocal translocation) during the comparative study of total length. Her mean arm ratios are quite close to those of Longley. However, it is not possible to carry out F-tests individually for each arm ratio, in view of the nature of the data presented by Longley. This indicates that the total lengths of chromosomes in the present data are considerably different from those of Longley, whereas most of the arm ratios are remarkably similar.

Correlation and regression analyses were conducted to analyze the relation of various parameters and the results are presented in Table 3.

Further extending the statistical analysis, it was observed that the longer the chromosome, the greater the variability in length, as was evident from a highly significant positive correlation between mean and variance of total length, and also from the fact that coefficient of variation of total length does not vary much from chromosome to chromosome. This corroborates the findings of Maguire that variability may tend to be distributed uniformly per unit length. This is also true individually for arm length and arm ratios, in view of the highly significant positive correlation coefficients.

Short arms are not consistently either more or less variable in length than the long arms, if the differences in their averages are taken into account. This is supported by the finding that coefficients of variation of arm length do not vary much between short arm and long arm. Thus the conclusion of Maguire (op.

Table:3. Correlation and regression analysis of various statistical parameters of pachytene chromosomes of maize

Sl. No.	Parameter - x	Parameter - y	Correlation: r(x-y)	Regression: b(y→x)	Regression: b(x→y)
1.	Mean - total length	Var.- total length	+ 0.855**	11.310	0.065
2.	Mean - total length	Coef. var. - total length	- 0.030	- 0.008	-0.115
3.	Mean - arm length	Var.-arm length	+ 0.940**	5.587	0.158
4.	Mean - arm length	Coef. var. - arm length	+ 0.065 ?	0.016	0.260
5.	Mean - arm ratio	Var. - arm ratio	+ 0.780**	0.033	18.446
6.	Mean - arm ratio	Coef. var. - arm ratio	- 0.447	- 0.864	- 0.231
7.	Coef. var.-total length	Coef. var. - long arm length	+ 0.247	0.054	1.120
8.	Coef. var.-total length	Coef. var. - short arm length	+ 0.248	0.064	0.959
9.	Coef. var.-total length	Coef. var. - arm ratio	- 0.201	- 0.091	- 0.440
10.	Coef. var. arm ratio	Coef. var. - long arm length	- 0.037	- 0.069	- 0.020
11.	Coef. var.-arm ratio	Coef. var. - short arm length	- 0.086	- 0.188	- 0.040

** Indicates significance

cit.) that the short arm is more variable than the long arm in a chromosome, does not coincide with that from the present data.

It appears that relative variability (coef. var.) of arm ratio has no significant influence on relative variability in total length. In fact, there is a slight negative correlation observed between these two characters. This is also true if the variabilities in long arm length and short arm length are separately examined.

The possible reason for the deviation of the results of the present report from those of Maguire and of Longley becomes quite intelligible if the nature of the materials and method of analysis are taken into account. Longley's data on length measurements represent pooled data of all the chromosomes in 28 cells from 14 Indian varieties. He also gave mean total length, arm lengths and arm ratios for 33 Indian varieties and 41 Mexican varieties. Maguire's data represent length measurements from all the chromosomes of 271 cells. For arm ratios, different numbers are measured for each chromosome. The materials included 6th and 7th backcross generations (to maize) of a maize-*Tripsacum* hybrid. The maize background included L289 and CC5, and a few had a reciprocal translocation. She reported a significantly different F-value for chromosome 7, which was the *Zea-Tripsacum* interchange chromosome. In these two reports, the same number of observations were not made for total lengths and arm ratios. The present analysis is based on pooled data from 8 cultivars (mostly Indian). The materials included are relatively more heterogeneous than those of Maguire, but less heterogeneous than those of Longley. Another factor of difference is that the same number of observations has been made, both for total lengths and arm ratios. These factors might have been responsible for the discrepancy in the present data on total lengths, but are more or less in complete agreement with respect to arm ratios, when compared with the previous records.

Based on the present data, the following conclusions can be made:

1. Variability of total length, arm length and arm ratio may be uniform per unit length.
2. Differences in the variabilities of long and short arms in a chromosome are not considerably apparent.
3. The relative variability of arm ratio has no significant influence on relative variability of total lengths.

The present investigation was carried out under the supervision of Prof. A. K. Sharma of the Department of Botany, University of Calcutta, Calcutta.

J. S. P. Sarma

Amount of DNA in different cultivars of maize and its importance in selection

The importance of DNA content in the origin and evolution of genotypes has been increasingly realized in recent years. It has, however, been recorded that both increase and decrease in DNA content may be associated with evolution. The increased DNA values have to a great extent been influenced by the large amount of amplified sequences. However, along with amplification there has been both individual duplication and polyploidy contributing towards the DNA increment in the origin of genotypes.

A comprehensive program has been undertaken on the study of somatic chromosomes of maize, with specific reference to intervarietal differences. The present report deals with in situ cytophotometric investigation of DNA content in the somatic chromosomes of 18 cultivars of maize, employing a two-wavelength technique. The two wavelengths selected were 550 nm and 562 nm and the aperture was 6.3. Extinction in the latter wavelength must be two times that in the former, and the relative absorbance values were noted. A minimum of 50 cells at metaphase (4C) were analyzed for each cultivar.

Table 1: Relative DNA estimation of different cultivars of maize (*Zea mays* L.)

Sl. No.	Cultivar	Mean L_1	Mean L_2	$\frac{\text{Mean } L_2}{\text{Mean } L_1}$	D Value	$m = KAL_1D$
1.	Stowell's Ever Green	0.1012	0.1836	1.8142	0.962	0.9660
2.	Sonada local	0.1010	0.2040	2.0198	0.867	0.8689
3.	Kalimpong local	0.1090	0.2006	0.8404	0.945	1.0221
4.	Warangal local	0.1050	0.1955	1.8619	0.934	0.9731
5.	Amber pop-corn	0.1123	0.2073	1.8459	0.940	1.0474
6.	KYS - strain	0.1100	0.2050	1.8636	0.934	1.0194
7.	CM - 104	0.1096	0.2008	1.8321	0.950	1.0331
8.	CM - 105	0.1110	0.2085	1.8784	0.923	1.0166
9.	CM - 114	0.1118	0.2216	1.8653	0.928	1.0295
10.	CM - 115	0.1112	0.2040	1.8345	0.950	1.0482
11.	CM - 201	0.1054	0.1950	1.8501	0.940	0.9831
12.	CM - 206	0.1120	0.2025	1.8080	0.962	1.0691
13.	Ganga - 5	0.1060	0.1960	1.8491	0.940	0.9887
14.	Dhara	0.1169	0.2141	1.8315	0.950	1.1019
15.	EV Composite BVI	0.1100	0.2025	1.8409	0.945	1.0314
16.	Synthetic B19	0.1084	0.2016	1.8598	0.934	1.0046
17.	Synthetic B21	0.1104	0.2012	1.8225	0.956	1.0472
18.	Synthetic B23	0.1108	0.2020	1.8231	0.934	1.0269

L_1 and L_2 values were calculated using appropriate conversion formulae. D values for respective proportions of mean L_1 and L_2 were referred to from Garcia (cited by Sharma and Sharma, 1980, Chromosome Techniques: Theory and Practice, Butterworths, London). The DNA values (m) were estimated by using the formula $m = KAL_1D$, where K is a constant and A is the area of the aperture (πr^2). Because K and π are constants, they were omitted for comparative purposes.

Table 1 gives the relevant details of the calculations. A scanning survey of Table 1 indicates that the relative DNA values of the different cultivars studied do not vary much. The minimum value is 0.8689 for Sonada Local, which is with knobless pachytene chromosome complement. The maximum value is 1.1019 for the cultivar Diara. It has knobs and B chromosomes as well. However, this correlation was not consistently observed in the case of other cultivars. In general, inbreds have relatively more DNA content than the knobless outbred races.

Table 2: Comparison of relative DNA values with total chromatin lengths and karyotype formulae

Sl. No.	Cultivar	Relative DNA value	Total chromatin length (in μ)	Karyotype formula
1.	Stowell's Ever Green	0.9660	29.14	$2A_2+8B+10C$
2.	Sonada local	0.8689	26.05	$2A_1+10B+8C$
3.	Kalimpong local	1.0221	43.49	$2A_2+12B+6C$
4.	Marangal local	0.9731	26.71	$2A_2+10B+8C$
5.	CM - 104	1.0331	50.99	$2A_3+12B+6C$
6.	CM - 105	1.0166	56.95	$2A_2+8B+10C$
7.	CM - 114	1.0295	40.62	$2A_2+12B+6C$
8.	CM - 115	1.0482	28.92	$2A_2+12B+6C$
9.	CM - 201	0.9831	41.50	$2A_3+8B+10C$
10.	CM - 206	1.0691	34.44	$2A_3+12B+6C$
11.	Ganga - 5	0.9887	29.14	$2A_2+6B+12C$
12.	Diara	1.1019	27.37	$2A_2+8B+10C$
13.	Synthetic B19	1.0046	27.60	$2A_2+12B+6C$
14.	Synthetic B25	1.0269	22.96	$2A_3+8B+10C$

Tables 2 and 3 furnish a comparison of the relative DNA values with relevant cytological features, analyzed in the preceding reports of this News Letter. Further observation of Tables 2 and 3 reveals that the relative DNA quantity in

Table 3: Comparison of relative DNA values with other relevant cytological features

Sl. No.	Cultivar	Relative value of DNA	T.C.L. of haploid mitotic complement	T.C.L. of pachytene complement	Knob range	Chiasma frequency	Variations, if any
1.	Stowell's Ever Green	0.9660	30.39	383.59	0	21.33 \pm 1.92	1-2 B present
2.	Sonada local	0.8689	25.96	646.35	0	23.50 \pm 2.21	-
3.	Kalimpong local	1.0221	43.34	794.41	0	21.00 \pm 2.83	Haploid roots
4.	Warangal local	0.9731	26.62	570.46	7-8	23.50 \pm 2.51	0-2 B present
5.	Diara	1.1019	27.28	516.38	0-5	21.67 \pm 2.42	1) 0-2 B present 2) Heteromorphy of Sat.pair
6.	Synthetic B19	1.0046	27.50	745.63	0-4	22.67 \pm 2.33	--

different cultivars remains more or less constant, notwithstanding differences in their other cytological features such as knobs, B chromosomes, chromatin length of the haploid complements, etc. (The present investigation was carried out under the supervision of Prof. A. K. Sharma, at the Department of Botany, University of Calcutta, Calcutta.)

J. S. P. Sarma

Nonrandom arrangement of somatic chromosomes of maize and its implication

Somatic association is a comparatively recent aspect of cytological analysis, and several reports regarding nonrandom arrangement of homologous chromosomes at mitotic stages have been forthcoming since the last decade. The present report deals with a pooled analysis of nonrandom organization of satellited chromosomes in twenty-two cultivars of maize.

The materials included six inbreds, CM-104, CM-105, CM-114, CM-115, CM-201 and CM-206; two synthetics, B19 and B23, two composites, Vijay and Diara, and three hybrids, G-5, GS-2 and Globe Hybrid. Also included were two varieties, Golden Bantam and Stowell's Evergreen, and seven races, Nal-Tel (Yucatan-7), Comfite Morocho, Tama Flint, Sikkim Primitive, Warangal Local, Kalimpong Local and Sonada Local. The treatment schedule for mitotic analysis was the same as was detailed in a previous report (see this News Letter).

Satellited chromosomes as mentioned above were used for analyzing the nonrandom organization. Altogether 93 plates were selected, adhering strictly to the following criteria:

- (1) All the chromosomes were contained within the perimeter of a figure, whose major-minor axis ratio would not exceed 2:1.
- (2) All the chromosome organelles (telomeres, centromeres and satellites) were present and clearly defined.

Distances between the centromeres of the two satellited chromosomes in a cell were measured. To minimize the differences due to the degree of squashing, each distance between the satellited chromosomes was divided by the distance between

location of all the chromosomes toward the nucleolar half of the pollen mother cells, even at late diakinesis. In 440 cells, this type of preferential location of chromosomes was observed in 76.5 percent. To test whether this abnormality was due to differential pressure of smearing, PMC with three different shapes--oval, circular and elliptical--were scored. 138, 58 and 38 cells were observed to exhibit this phenomenon out of 176, 96 and 50 PMC of the three shapes, respectively.

Various anaphase-I irregularities were also recorded. 13-10 segregation was the most frequent, being encountered in 54 percent of the cells. 12-11 segregation was less frequent, occurring in 18 percent of the meiocytes. Ten percent of the PMC had 14-9 segregation. Two percent of the cells exhibited 14-8, 15-7 and 16-7 segregations each. A few tripolar segregations (11-9-3; 11-2-10) were also recorded. Occasionally, laggards were seen at both the anaphases. Cell plate formation parallel to the spindle was noted in some microspore mother cells.

Several other irregularities in cell plate formation resulted in varied groupings of the pollen grains. Tetrads were observed in 639 cases. Triads, pentads, hexads, heptads and polyads were represented by 49, 52, 53, 4 and 1 groups, respectively. Pollen sterility was as high as 84 percent.

The cytogenetics of synaptic mutants has been recently reviewed (I. N. Golubovskaya, *Int. Rev. Cytol.* 58:247-290; W. Gottschalk and M. L. H. Koul, *Nucleus* 23:1-15 and 99-120; P. R. K. Koduru and M. K. Rao, *TAG* 59:197-214). It was not possible to determine whether the desynapsis was genic or environmental. Out of many plants, only one reached flowering stage. The desynapsis observed here may be rated as strong, as per Prakken's classification.

It is apparent that the difficulty in analyzing pachytenes, the formation of synezetic knot and the preferential location of the chromosomes toward the nucleolar side even at late diakinesis are interrelated. However, the nature of pachytene spread is known to be under genetic control. B. John and K. R. Lewis (*Protoplasmatologia*, Bd. 6) observed the tendency of univalents to remain scattered more toward the poles. Several other factors seem to influence the position of univalents: (a) the structure of meiotic chromosomes, (b) the stage of development of MI, and (c) interchromosomal effects (U. C. Lavania and A. K. Sharma, *Protoplasma*, in press). In addition, cytoplasmic factors may also influence the orientation of univalents. The other irregularities recorded are consequent upon the failure of maintenance of chiasmata and that of proper orientation of the chromosomes on the spindle. (The present investigation was carried out under the supervision of Prof. A. K. Sharma of Department of Botany, University of Calcutta, Calcutta, India.)

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Test for allelism of spontaneous mutants of maize

As a result of our previous studies which were presented in detail in the book, V. E. Micu, "The Genetical Studies of Maize," published in 1981, a large number of spontaneous mutants of maize were collected and partially studied. The genetic analysis of this material was continued. During the last 3 years, more than 350 mutants, including the following classes--kernel form and texture, upright leaves, brown midrib, dwarfism, ramosa ear and male sterility--were tested. For the endosperm, dwarf and ramosa mutants, a phenotypical classification was previously

conducted, and the nonidentified sources were crossed first with the similar genetic markers. This procedure helped us to reduce the number of crosses and to raise the effectiveness of tests.

Among 43 floury endosperm mutants, 4 new sources of o2 and 2 of f12 were identified. Besides, another 9 sources gave a positive test with f12 marker. However, taking into account the more complicated inheritance of f12 and availability of cf12 (A. Paliy, *Genetica* 11:5-7, 1975) the identification of these sources cannot be considered complete.

Two phenotypically different groups of dwarf mutants were studied: one including 30 sources of brachytic, and the other 16 sources of dwarf type. Most of the tested mutants turned out to be non-allelic to markers, but nevertheless many new sources of br1, br2 and d1 (Table 1) were identified. Among the ramosa ear mutants that we have in our collection, ra1 phenotypes predominate, and that

Table 1. The list of tested identified mutants.

Phenotypical classes	Markers	Number of sources	
		Tested	Identified
Floury endosperm	f11	21	2
	f12	20	-
	o2	31	4
Brittle endosperm	bt1	3	1
	bt2	14	5
	sh2	4	-
Sugary endosperm	su1	6	6
Brown midrib	bm1	3	1
	bm2	3	-
	bm3	7	-
	bm4	2	-
Upright leaves	lg1	20	3
	lg3	17	1
Brachytic	br1	28	7
	br2	30	6
Dwarf	d1	16	8
Ramosa ear	ra1	28	17
	ra2	11	3
	ra3	10	4
Male sterility	ms2	30	2
	ms9	5	1
	ms10	34	1
	ms13	10	2
Total		353	74

was confirmed by genetic analysis. New sources of male sterility genes *ms2*, *ms9*, *ms10* and *ms13* were identified. The sources of sterility were also tested with other markers, but the test turned out to be negative.

In all phenotypical groups which were studied, many mutants still remained unidentified. It is possible that this material contains unknown or rare genotypes.

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Increasing the protein content of maize by means of induced mutants

The deficiency of normal corn for the amino acids was recognized at the turn of the century by the studies of Osborne (1897, 1914), and Osborne and Mendel (1914). Improvement of the feeding qualities of normal corn was attempted by selection for higher crude protein in the corn kernel. In 1964, E. T. Mertz and co-workers reported that the opaque-2 gene changed the protein composition and increased the lysine content of corn endosperm. Protein mutants were isolated in the M3 generation after kernel treatment with combinations of radiation (gamma rays) and the chemical mutagens N-methyl-N-nitrosourea (MNUA) and sodium azide (SA). The mutant kernels analyzed were mature, air dried, and had been produced by self-pollination. Protein content (nitrogen x 6.25) was analyzed by the micro-Kjeldahl method. The amino acids in 100 mg of whole kernels were determined in an HD-1200E automatic amino acid analyzer.

The amino acid and protein contents of kernels are shown in Table 1. Our opaque mutant strains contained more protein than the normal strain (S-615) and

Table - 1. Protein and Amino-Acid Content in Kernels of Opaque - 2 /S-10/, Strain, Normal Inbred line /S-615/, and Mutant Strains.

Material	Protein %	Lysine	Histidine	Arginine	Aspartic acid	Threonine	Serine	Glutamic acid	Proline	Glycine	Alanine	Cysteine	Valine	Methionine	Isoleucine	Leucine	Tyrosine	Phenyl alanine
Normal line /S-615/	11,3	4,64	1,80	4,32	7,52	4,06	4,96	21,52	9,18	3,78	8,62	2,04	4,71	2,01	3,25	14,73	3,05	5,37
Opaque-2 /S-10/	11,5	5,87	1,82	4,22	8,68	4,38	5,36	22,14	8,86	3,45	8,12	2,22	4,50	2,00	4,06	14,48	2,88	4,42
M-204-1	13,7	3,86	1,71	3,96	7,03	4,17	4,84	23,29	10,10	3,32	8,97	2,80	4,17	1,83	3,70	15,86	3,23	5,27
M-222-1	14,4	5,02	2,05	4,60	7,27	3,86	4,98	21,09	9,05	3,69	7,91	2,30	4,08	1,25	3,62	14,48	3,26	4,76
M-218a-2	13,4	5,08	1,96	3,96	7,90	3,99	4,71	21,93	8,87	3,75	8,44	2,42	3,85	2,07	4,39	13,92	3,20	5,43
M-204-47	12,9	5,75	2,04	4,00	7,33	4,45	4,96	22,67	9,28	3,79	8,53	2,20	4,30	1,57	3,87	15,14	3,09	4,66
M-203-4	14,0	4,03	2,04	4,26	7,09	4,03	5,33	23,69	11,04	3,91	8,38	2,40	4,04	1,78	4,11	14,63	2,84	4,04
M-204-15	13,2	3,83	1,87	3,73	8,19	4,54	5,62	22,98	10,93	3,97	9,17	2,08	4,31	1,20	4,47	14,21	3,51	4,20
M-223-3-1	14,5	3,03	2,91	4,59	7,82	3,65	6,18	21,14	9,49	3,93	8,01	3,20	4,86	1,18	3,11	12,57	3,67	5,38

opaque-2 strain (S-10), but less lysine (except mutant M-204-47) than the opaque-2 strain. The content of total protein was found to be increased by 1.4-3% when compared to the opaque-2 line. Mutant strains contained more histidine, arginine, aspartic acid, threonine, serine, glutamic acid, proline, glycine,

alanine, cysteine, valine, isoleucine, leucine, tyrosine, and phenylalanine, but less methionine. It is apparent that a new source of opaque mutant gene, which has a different biochemical characteristic than the opaque-2 mutant, has been induced by mutagens.

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A brief note on the rediscovery of Durango teosinte

In 1896, Edward Palmer, an ethnobotanist with the Peabody Museum of Harvard University, collected teosinte near Durango, Mexico. Palmer collected both herbarium specimens, which were deposited at several major herbaria (Palmer 743, MO, US, F, BM and K), and seeds which were never successfully increased. Dr. H. V. Jackson of Durango also collected seed from this region, which he sent G. N. Collins of the U.S.D.A. Jackson's seed was successfully increased in southern California, but later the collection was apparently lost. Then in 1921, Collins visited the Durango region and found teosinte at the following locality:

" . . . along the bank of an old irrigating ditch at Alcalde Ranch, Anavacuyán, about 15 miles east of Durango." (Collins, 1921, Teosinte in Mexico, J. Hered. 12:339-350).

Collins collected both herbarium specimens, which were deposited at several U.S. herbaria (Collins 15, US, WIS), and seeds which were subsequently incorporated in numerous genetic studies from the 1920s until the 1950s. These studies include:

1. Beadle, 1932. Studies of Euchlaena and its hybrids with Zea. I. Chromosome behavior in E. mexicana and its hybrids with Zea mays. Zeitschr. Abst. Vererb. 62:291-304.
2. Arnason, 1936. Cytogenetics of hybrids between Zea mays and Euchlaena mexicana. Genetics 21:40-60.
3. Longley, 1937. Morphological characters of teosinte chromosomes. J. Agric. Res. 54:835-862.
4. O'Mara, 1939. Cytological observations on Zea-Euchlaena hybrids. Genetics 24:82-83.
5. Mangelsdorf, 1947. The origin and evolution of maize. Adv. Genet. pp. 161-207, New York.
6. Rogers, 1950. Fertility relationships in maize-teosinte hybrids. Texas Agric. Exp. Sta. Bull. 730.
7. Ting, 1958. Inversions and other characteristics of teosinte chromosomes. Cytologia 23:239-250.

Apparently, Collins' collection of Durango teosinte was not maintained and its use in genetic experiments ceased in the 1960s. In 1963, Wilkes (1967, "Teosinte: The Closest Relative of Maize") attempted to relocate Collins' station but was not successful.

On October 1, 1982, the author, Lowell R. Nault, and a group of entomologists from the Ohio Agricultural Research and Development Center were in the Durango

area collecting Tripsacum and leaf hoppers (Dalbulus spp.). Our attempt to relocate Collins' station was successful. The locality data are:

Ca. 2-3 km NE of Alcalde, a Pueblito 20 km ENE (by air) of Durango. 24°04'N, 104°31'W, 2050 m alt. Doebley
No. 625.

The population is reasonably large with teosinte plants occurring along at least a 1 km stretch of a small stream and irrigation ditch. Teosinte grew in thickets with other weedy species, including members of the genera Salix, Ipomoea, Bidens, Tithonia, Solanum, and Cosmos. Very few teosinte plants were found on the edges of maize, sorghum and Capsicum fields which bordered the stream and canal. Though maize was abundant in the region, only a single teosinte-maize hybrid was observed among about 100 plants checked. The plants were typical of Zea mays L. ssp. mexicana (Schrad.) Iltis Race Central Plateau or Nobogame.

Plants slender to moderately robust, 1-2 m tall, untillered but commonly with branches emerging from nodes along upper half of mainstem; leaf sheaths green or slightly red with a fringe of long, soft hairs along its edge and near the auricle; tassels with (0-) 5-15 (rarely more) branches; male spikelets 7-10 mm long (mean 8.1 mm); weight of 100 fruitcases 6.4 gr.

An examination of the isozyme constitution of Durango teosinte revealed it to be similar to Central Plateau, Chalco, and Nobogame teosintes (Doebley, Goodman and Stuber, Syst. Bot., in review). Both herbarium specimens and seeds were collected. Seeds are presently being increased and will be deposited at the U.S.D.A. Plant Introduction Station at Experiment, Georgia.

John F. Doebley

Inheritance and localization of PHI isozymes in maize

In most plant species investigated, phosphohexose isomerase (PHI, or PGI, or GPI) isozymes are dimers and are usually encoded by two loci, with the products of one locus active in the cytoplasm and the other in the plastids. Until recently, in our studies of PHI isozymes in maize, we have dealt only with those found in the cytoplasm, which are encoded by the Phi1 locus. This locus is localized on chromosome 1 between Adh1 (12% recombination) and Gdh1 (5% recombination). Enzymes encoded by this locus have been found in tissues throughout the plant, including roots, leaves, coleoptiles, scutella, and pollen. We have identified nine alleles at this locus.

Recently we have found a second set of PHI isozyme bands, presumably specified by a second locus, Phi2. Isozymes encoded by this locus are found in plastids (etioplasts) and may also be expressed in the cytoplasm. Genetic studies of this locus have not been completed.

C. W. Stuber and M. M. Goodman

Localization of Got2 isozymes in maize

In our studies of enzymes associated with plastids (etioplasts), we have found isozymes encoded by Got2 in this plastid fraction. An earlier report (Scandalios et al., 1975, Biochem. Genet. 13:759-769) indicated that isozymes encoded by Got2 were found exclusively in the cytosol, however the plastid fraction may have been

included with the cytosol in their analyses. In our studies, activity of Got2 isozymes in seedling leaves is considerably greater than that found in coleoptiles. This would be expected for isozymes localized in the plastids.

C. W. Stuber and M. M. Goodman

Evidence for additional 6-PGD loci in maize

In an earlier communication (MGCNL 54:99) we reported that two loci, Pgd1 and Pgd2, encoded the 6-phosphogluconate isozymes found in maize. From studies based primarily on coleoptiles and roots, these isozymes appear to reside in the cytoplasm. We have recently observed the existence of additional 6-PGD isozyme bands in extracts from leaf tissues. Although these new isozymes have not been studied extensively, the banding patterns suggest possibly two additional loci. The finding of 6-PGD isozymes localized both in the cytosol and, now, possibly with leaf plastids would be expected because 6-PGD isozymes have been found in both sites in several other plant species.

C. W. Stuber and M. M. Goodman

Cat3 is not on chromosome 1L

We reported linkage data in the News Letter last year which were not wholly consistent with the reported localization (D. G. Roupakias et al., 1980, TAG 58: 211-218) of Cat3 to 1L. Using a TB-1La stock (provided to us by Kathleen Newton) carrying Adh1-2 and Adh1-6 (Adh1-C and Adh1-S in the usual Schwartz notation), exact reciprocal crosses were made with a stock homozygous for Adh1-4 (Adh1-F of Schwartz) and for Cat3-14, the most extreme Cat3 variant.

We examined a total of 21 plants. Nine of these were from the cross using the TB-1La stock as female parent. All nine of those plants carried Adh1-4 and either Adh1-2 or Adh1-6 or both. Eight plants were Cat3-12/14. One was Cat3-14/null. An additional five plants were from small kernels from the cross using the TB-1La plant as the male parent. All five carried both Adh1-4 and Adh1-6. At least two of the five, and probably three, carried two doses of Adh1-6. Four were Cat3-12/14; one was Cat3-14/null. Seven plants were from large kernels from the cross with TB-1La as male parent. Each of these carried only the Adh1-4 of the female parent and lacked an Adh1 allele from the male parent. Yet of these seven plants, five were Cat3-12/14, while two were Cat3-14/null. All plants were heterozygous for Acpl, Glu1, and Pgm2, and results for 17 additional loci suggest that contamination was not a factor in these crosses. The seven plants lacking an Adh1 allele from the male parent also appeared to have only one Phl1 allele and one Mdh4 allele. These results demonstrate that Cat3 is not uncovered by TB-1La, but rather suggest that at least some TB-1La stocks carry null alleles or repressors for Cat3. Together with linkage data already available for loci known to be on 1L, these results suggest that Cat3 must reside elsewhere in the genome.

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Races of teosinte show differential crossability with maize when maize is used as the female parent

In surveying the isozyme variation within teosinte, we attempted to verify the inheritance of each newly encountered variant. For this purpose, many F1 hybrids were made between the various teosintes and maize. Usually, each teosinte plant

Table 1. Summary of teosinte (♂) x maize (♀) crosses for checking the inheritance of teosinte electromorphs.

Plant Numbers	Race	Collection	No. of Plants	No. of Crosses	No. of Seeds	Ave. No. of seeds per Cross
45-24	Balsas	K67-13	1	4	408	102
42-9		K67-14	1	2	260	130
43-9, 11		K67-15	2	4	505	126
49-1, 6		K67-5	2	4	231	58
47-2 to 12		Palo Blanca	4	5	415	83
44-4 to 21; 55-4, 5		El Salado	5	9	1023	114
89-8		TE 562561	1	2	134	67
89-15 to 26		TE 562562	3	8	995	124
92-1 to 16		TE 562564	4	16	1979	124
92-17 to 93-7		TE 562565	4	11	926	84
90-1 to 13		954541	2	4	506	126
43-15 to 23		W71-2	3	10	1203	120
D027-15		Iltia 1480	1	2	13	6
D026-3		Puga 11065-2	1	2	419	140
Summary for Balsas			36	84	9017	107
50-9	Central Plateau	K67-19	1	1	21	21
51-1 to 14		K67-21	5	6	770	128
45-14		K71-2	1	1	120	120
Summary for Central Plateau			7	8	911	114
46-1, 19	Chalco	K67-3	2	2	179	80
38-15, 16; 39-1 to 26	Huehucenango	2072	9	22	1498	68
41-25, 26		PI 349233	2	7	167	24
Summary for Huehucenango			11	29	1665	57
1849-1 to 4	Guatemala	40-202A	3	7	752	107
D024-18 to 23		Iltia G5	4	11	377	34
D024-2		Iltia G36	1	5	193	39
Summary for Guatemala			8	23	1322	57
D028-11	Diploperennis	Iltia 1155	1	2	56	28
D017-		1190	3	6	254	42
D016-		1275	3	5	26	5
Summary for Diploperennis			7	13	336	26
41-1 to 4, D027-3	Perennis	Collins	5	0	0	0
D020-		Iltia 1050	2	4	27	6
Summary for Perennis			7	4	27	5

carrying an isozyme of special interest was crossed with at least four maize plants representing at least two distinct genotypes. The female parents represented a wide range of commercial and exotic derivatives, including inbreds, single-crosses, and complex pedigrees of isozyme tester stocks. Few or no records were kept for crosses which totally failed, although there were many of these for diploperennis and even more for perennis, relative to the other teosintes. However, as the data in Table 1 suggest, there were substantial differences in seed-set among the successful crosses, both between and within races. Races Central Plateau and Balsas crossed most readily, while Guatemala, Huehuetenango, and Z. diploperennis resulted in much lower seed-set. Very limited data for Chalco also suggested a reduced crossing percentage.

The concepts that there are restrictions in crossing between teosinte and maize and that the various teosintes do differ in their ease of crossing are not new, but these are the most extensive experimental data on kernel set in maize-teosinte hybrids currently available.

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Further localization of Mdh1 and Idh1 on chromosome 8

Using a TB-8Lc stock supplied by Jack Beckett, reciprocal crosses were made with several Mdh1 and Idh1 tester stocks. For one such cross, both alleles at Mdh1 and both alleles at Idh1 could usually be scored unambiguously. The TB-8Lc parent carried Idh1-4 and Mdh1-6, while the tester stock carried Idh1-2 and Mdh1-1. Of 36 plants tested using the TB-8Lc plant as male, most were doubly heterozygous, Idh1-2/4, Mdh1-1/6. Not all plants could be scored with absolute certainty for Idh1, but several plants were Idh1-2, Mdh1-1/6, with no Idh1 allele contributed by the male parent. Since the latter plants were heterozygous for Acpl, Idh2, and Mdh2, as had been expected (the two putative parental plants were homozygous for different alleles for these three loci), contamination appears unlikely. (Sixteen other loci also revealed no contamination, but the latter were less informative.) Thus, it appears that Mdh1 lies outside the region spanned by TB-8Lc, while Idh1 lies within that region.

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Transmission of a deficiency for the entire nucleolus organizer region

Certain maize heterozygous interchanges with one breakpoint near the end of a chromosome will transmit through the ovules a deficiency plus duplication (Df + Dp) chromosome complement that is deficient for a short end segment. A genetic marker located in that end segment can be used to identify heterozygous Df + Dp plant types among the progeny. Previous crosses of heterozygous NOR-interchanges (interchanges with one breakpoint in the nucleolus organizer region) as female, with heterozygous polymitotic (+/po) males, produced male

steriles (-/po hemizygotes) among the progeny (MGCNL 51:49-52, 1977). Those data suggested that the transmitted deficiency can include the NOR-secondary constriction, and as much as 90% of the NOR-heterochromatin. This was a surprise, since the NOR is the site of the 18S/25S rRNA genes, and some of these transmitted deficiencies presumably were deficient for at least 90% of the rRNA genes. This report provides evidence for the transmission of gametes missing the entire NOR.

Two interchanges with breakpoints known to be proximal to the NOR were crossed as heterozygous females to +/po males. These interchanges were T5-6c (5L.89, 6S near centromere) and T6-9(067-6) (6S.39, 9L.47). Smaller seed were selected to increase the frequency of putative Df + Dp heterozygotes among the resulting plants. Partial microsporocyte samples were taken from all of the progeny, in order that the same plants could later be scored for male sterility. Tests with T5-6c yielded no male-sterile progeny among over 100 plants derived from smaller seed. This result was expected because the deficiency would be for the whole short arm of chromosome 6. However, tests with T6-9(067-6) indicated the successful transmission of the Df + Dp gamete deficient for the + allele of po. Planting 16 smaller seed from each of eight crosses yielded 26 male-sterile (-/po) plants from five of the crosses. At least six fertile plants were noticed to be phenotypically heterozygous for the Df + Dp chromosome complement; these presumably received the + allele from the +/po male parent in the cross. Cytological analysis of male-sterile putative Df + Dp heterozygotes clearly indicated the presence of a normal chromosome 6 and a Df + Dp chromosome. Synapsis was normal in all cases through 90% of 6S between the centromere and the NOR-heterochromatin. Occasionally, the normal and the Df + Dp chromosome would then show asynapsis just proximal to the NOR. No indication of the NOR-heterochromatin or the NOR-secondary constriction was observed in the Df + Dp chromosome. The original report of the 6S breakpoint being at .39 probably is incorrect. These results suggest the breakpoint is proximal but near the NOR.

The hemizygous tests reported here indicate that a megaspore missing the entire NOR region can function in the postmeiotic mitoses of the developing embryo sac, and in the subsequent fertilization and sporophytic developmental processes. Previous evidence in maize shows that the NOR is the site of the 18/25S rRNA genes. The evidence reported here showing transmission of a deficiency for the entire NOR indicates that megametogenesis can proceed on the basis of maternal ribosomes carried over during embryo sac development, or that rRNA genes exist in the genome at locations other than the NOR, or both. Such numbers of rRNA genes at any one non-NOR site must be fairly low since the rRNA/DNA in situ hybridization procedures we have employed in the past allow the detection of clusters of 50 rRNA genes or more.

R. L. Phillips, A. S. Wang and W. P. Bullock

Near-isogenic lines of various genetic markers and interchanges

Near-isogenic lines of genetic markers and interchanges are useful for a variety of genetic, biochemical, and physiological studies. This report summarizes the backcross program we have carried out for over 10 years (see tables), and is an updated version of the 1979 report (MGCNL 53:114-115).

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

Chromosome 1:	srl (6, 4, 6, 6), br (6, 6, 6, 6), f (6, 6, 6, 6), an (3, 6, 6, 6), bm2 (6, 6, 6, 6)
Chromosome 2:	lg1 (6, 6, 6, 6), gl2 (6, 6, 6, 6), B (6, 5, 3, 6), fl (4, 6, 6, 5), v4 (4, 6, 6, 5), Ch (6, -, -, 4)
Chromosome 3:	cr (6, 6, 6, 6), d (13, 6, -, 6), lg2 (6, 6, 6, 6), al (6, 6, 4, 6), et (6, 6, 6, 6), pm (-, 6, 6, 6)
Chromosome 4:	la (6, 6, 6, 6), sul (6, 6, 6, 6), gl3 (6, 6, 6, 6), bt2 (6, 6, 6, 6)
Chromosome 5:	a2 (6, 6, 6, 6), bml (6, 6, 6, 2), pr (6, 6, 6, 6), v2 (6, 6, 6, 6)
Chromosome 6:	po (1, 6, 6, 6), rgd (1, 3, 6, 2), y (6, 6, 6, 6), Pl (6, 6, 6, 6), su2 (4, -, 6, 6), py (5, 6, 5, -)
Chromosome 7:	o2 (7, 6, 6, 6), v5 (5, 6, 4, 6), ra (6, 6, 4, 6) gll (6, 6, 4, 6), ij (5, 3, -, 6)
Chromosome 8:	v16 (5, 6, 2, 6), j (6, 6, 2, 6)
Chromosome 9:	yg2 (5, 4, 6, 6), sh (-, 6, 6, 6), wx (6, 6, 6, 6) bm4 (5, 6, 6, 6)
Chromosome 10:	oy (6, 6, 6, 1), g (6, 6, 5, 6), Rnj (6, 6, 6, 5), sr2 (4, 6, 3, 6), bf2 (6, 6, -, 2), K10 (6, 6, 5, 6)

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

Satellite - interchanges	
1-6b	(6, 6, 1, 6)
2-6(001-15)	(6, 6, 3, 5)
3-6b	(6, 6, 6, 6)
4-6(7328)	(6, 6, 6, 6)
4-6(5227)	(6, 6, 6, 6)
4-6c	(6, 6, 4, 6)
4-6(003-16)	(6, 6, -, 6)
5-6b	(6, 6, 6, 6)
5-6d	(6, 6, 6, 6)
5-6(8219)	(6, 6, 6, 6)
6-7(7036)	(6, 6, 6, 6)
6-9(017-14)	(6, 6, 4, 5)
6-10f	(6, 6, 6, 6)
NOR - interchanges	
4-6li (actually 1-6)	(6, 6, 6, 6)
1-6(5495)	(6, 3, 6, 3)
1-6(4986)	(2, 2, -, 2)
1-6(6189)	(6, 6, 6, 6)
1-6(8415)	(4, 6, 6, 6)
2-6(8786)	(6, 6, 6, 6)
2-6(027-4)	(6, 2, 6, 6)
2-6(5419)	(6, 6, 6, 6)
2-6(8441)	(-, 5, 5, 6)
3-6(030-8)	(6, 6, 6, 6)
3-6(032-3)	(6, 6, 6, 6)
4-6(4341)	(6, 6, 6, 6)
4-6(7037)	(6, 6, 6, 6)
5-6f	(6, 6, 6, 6)
5-6(8696)	(6, 6, 6, 6)
6-7(035-3)	(6, 6, 6, 6)
6-7(5181)	(6, 6, 6, 6)
6-7(4964)	(6, 6, 5, 6)
6-9a	(6, 6, 6, 6)
6-9d	(6, 6, 6, 6)
6-9(4778)	(6, 6, 6, 6)
6-10(5519)	(6, 6, 6, 6)
6-10(5253)	(6, 6, 5, 6)
6S - interchanges	
1-6d	(6, 5, 6, 1)
Others	
2-3e	(6, 6, 5, 6)
2-9(062-11)	(6, 5, 5, 6)
2-10b	(6; 6, 6, 6)
3-9(6722)	(6, 6, 6, 6)
6-7(027-6)	(4, 6, 6, 6)
7-9b	(6, 6, -, -)
8-9(4453)	(6, 6, -, 6)
8-9(8525)	(6, 5, 6, 6)

Genetic markers are isolated in homozygous condition after six backcrosses. Certain backcross lines are now being combined to produce multiple marker stocks. Sixth backcross interchange lines are simply selfed; no attempt has been made to isolate homozygotes.

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Knobs in Kashmir maize

The source of the material for this study was a series of collections of local maize varieties from three districts (Anantnag, Pulwama and Baramulla) of Kashmir Valley bearing the same varietal names. In all, fifteen varieties were collected, out of which 4 were collected from Anantnag (Tripachi I, Tripachi III, Vozij IA and Vozij IB), 7 from Pulwama (Tripachi II, Badeh IIa, Badeh IIB, Badeh IV, Niver IVA, Mishri IIC, and Kani IVB) and 4 from Baramulla (Tripachi VI, Badeh V, Niver VA, and Ferozpur VB) districts. Out of these fifteen varieties, 4 are known as Tripachi, 4 as Badeh, 2 as Vozij, 2 as Niver, 1 as Mishri, 1 as Kani and 1 as Ferozpur.

The seed for cytological studies was sown during the year 1973 in the Regional Research Laboratory (Branch), Sanat Nagar, Srinagar. On an average, five plants from each variety were studied. However, in the case of Mishri only one plant could be studied. The material for cytological studies was fixed in a freshly prepared mixture of 1:3 acetic acid and ethyl alcohol (96%) and then stored at low temperature (4-7 C) until worked. Squash preparations were made in 2% acetocarmine. Chromosome morphology was studied at the pachytene stage of meiosis.

Knob number within the varieties of a group showed a lot of variation. Knob number in the Tripachi group varied from a low of 5 for Tripachi VI to a high of 23 for Tripachi II. In the Badeh varieties, there was also great variation. The highest number of knobs (31) of all the fifteen varieties was observed in Badeh V. Niver varieties did not show any difference between each other, but in Vozij varieties, significant differences were observed. The former had 12 and 14 knobs respectively, while the latter possessed 12 and 27. Mishri IIC, Kani IVB and Ferozpur VB possessed 1, 19 and 8 knobs respectively.

The location of the knobs on the two arms was not the same in all the varieties. This difference was of particular interest among the varieties of each group. The short arm of chromosome 1 terminated in a knob in three of the four Tripachi varieties, two of the four Badeh varieties and one of the two Niver varieties. In Tripachi II, Tripachi III and Badeh V the long arm of chromosome 3 had more than two knobs, while Tripachi VI and Badeh IV were knobless in this region. Another interesting feature noted from the knob location was that the short arm of chromosome 1, 4, 7 and 9 ended with a terminal knob in nine, ten, five and eleven varieties, respectively. Similar differences were noticed for other chromosomes of other varieties also.

Taking into consideration the district and group data (Tables 1 and 2), the following similarities/dissimilarities were observed. On the district level (Table 1), Pulwama, Anantnag and Baramulla carried 29, 30 and 31 knobs respectively while the distribution of knob number among five groups (Table 2) ranged from 18 to 32.

Chromosome 1: The long arm of this chromosome bore three intercalary knobs--a, b, and c--in all three districts with varying frequencies. Out of all the groups, the Niver group had only one knob at position a, 3.2 u away from the centromere. In others, three knobs were observed. Short arm knobs were observed in only two districts (Anantnag and Pulwama). These knobs were 3.7 and 5.6 u, respectively, away from the centromere. These knobs were missing from the Badeh and "others" groups. However, a terminal knob on the short arm was present in all the groups as well as in all three districts.

Chromosome 2: The knobs observed on the long arm from Pulwama were 3.5 and 5.0 u away from the centromere, while the other two districts had four knobs.

Table 1. Average knob number, frequency (in brackets) and relative position (u) in fifteen local maize Varieties grouped in three districts.

K n o b location	D I S T R I C T S		
	Anantnag	Pulwama	Baramulla
1La	2.4 (9.4)	3.4 (16.4)	2.0 (49.0)
1Lb	4.6 (11.7)	4.9 (24.2)	6.1 (7.1)
1Lc	7.0 (12.4)	6.7 (12.1)	7.1 (7.1)
1S	3.7 (8.3)	5.6 (14.5)	- (0)
1ST	7.0 (11.2)	7.3 (12.2)	6.9 (17.1)
2La	3.4 (11.2)	3.5 (15.9)	3.5 (23.5)
2Lb	3.8 (9.4)	- (0)	4.5 (7.1)
2Lc	4.3 (11.2)	5.0 (10.2)	5.8 (14.2)
2Ld	6.3 (8.3)	- (0)	6.8 (14.2)
2Sa	3.3 (18.4)	3.2 (28.1)	4.3 (7.1)
2Sb	- (0)	- (0)	5.6 (7.1)
3La	- (0)	3.0 (18.2)	4.1 (23.5)
3Lb	5.9 (10.2)	4.8 (20.8)	4.9 (7.1)
3Lc	6.1 (18.4)	5.7 (9.0)	7.0 (14.1)
3Sa	1.2 (14.2)	2.4 (8.1)	2.0 (7.1)
3Sb	- (0)	- (0)	3.4 (7.1)
4La	3.0 (11.2)	2.0 (20.0)	2.8 (17.0)
4Lb	4.9 (15.0)	3.8 (20.1)	5.4 (17.0)
4Lc	5.7 (14.2)	4.9 (10.0)	- (0)
4S	- (0)	- (0)	2.4 (7.1)
4ST	3.8 (22.5)	3.7 (14.8)	3.7 (60.7)
5La	2.5 (11.2)	2.9 (38.6)	2.9 (44.7)
5Lb	4.4 (14.2)	4.5 (24.7)	5.2 (21.4)
5Lc	- (0)	- (0)	6.7 (21.4)
6La	4.3 (11.2)	4.3 (28.5)	3.0 (13.5)
6Lb	4.4 (16.6)	4.5 (21.1)	3.4 (21.4)
6Lc	- (0)	- (0)	4.5 (7.1)
7La	3.5 (9.8)	3.3 (12.2)	3.5 (13.5)
7Lb	4.2 (8.3)	4.2 (8.4)	4.4 (7.1)
7ST	2.0 (19.7)	2.1 (14.2)	1.8 (50.0)
8La	2.1 (7.7)	3.4 (17.2)	- (0)
8Lb	4.2 (11.8)	4.7 (27.1)	4.5 (42.3)
9La	2.9 (15.4)	2.1 (13.3)	- (0)
9Lb	3.2 (16.6)	3.7 (10.0)	- (0)
9ST	2.3 (31.7)	2.3 (20.7)	2.2 (37.1)
10L	1.9 (25.0)	2.0 (28.9)	1.8 (53.5)
Total knob number	30	29	31
1,2,310	=	Chromosome numbers.	
a,b,qa	=	Different positions of knobs.	
T	=	Terminal knob.	
S	=	Short arm.	
L	=	Long arm	

Badeh and Vozij groups had four internal knobs, while in Niver, "others" and Tripachi groups ranged from one to three, respectively. No consistent trend was observed in frequencies. Short arm knobs (a & b) observed on this chromosome from Baramulla had the same frequencies (7.1). In the other two districts, one knob was present on this arm. This arm of the Niver group was knobless, while in Tripachi, Vozij and "others" groups, one knob was observed with varying distances from the centromere. However, the Badeh group had both these knobs.

Table 2. Varietal average knob number, frequency (in brackets) and relative position (u) in fifteen local maize varieties.

Knob location	GROUPS OF VARIETIES				
	Tripachi	Badeh	Vozi	Niver	Others
1La	2.5 (33.1)	3.3 (13.9)	1.6 (7.7)	3.2 (24.5)	3.2 (28.5)
1Lb	4.3 (18.9)	6.1 (7.1)	3.9 (8.3)	- (0)	6.2 (28.5)
1Lo	6.5 (9.1)	7.1 (7.1)	6.7 (16.6)	- (0)	7.6 (14.2)
1S	5.4 (20.0)	- (0)	3.7 (8.3)	5.7 (9.0)	- (0)
1ST	7.2 (10.8)	6.9 (13.3)	6.9 (11.2)	6.9 (20.0)	7.7 (14.2)
2La	3.5 (14.2)	2.9 (11.1)	3.4 (8.3)	- (0)	- (0)
2Lb	3.7 (15.2)	4.3 (6.6)	3.8 (7.7)	- (0)	- (0)
2Lc	4.4 (14.2)	5.8 (14.2)	4.2 (8.3)	4.2 (24.5)	4.0 (28.5)
2Ld	- (0)	6.8 (14.2)	6.3 (8.3)	- (0)	5.9 (14.2)
2Sa	- (0)	3.9 (6.6)	1.9 (8.3)	- (0)	2.9 (50.0)
2Sb	4.8 (28.5)	5.6 (7.1)	- (0)	- (0)	- (0)
3La	- (0)	3.5 (11.1)	2.6 (8.3)	4.2 (24.5)	1.8 (32.1)
3Lb	4.1 (10.8)	4.9 (7.1)	- (0)	4.8 (9.0)	4.6 (32.1)
3Lo	5.5 (19.2)	7.0 (14.1)	6.6 (8.3)	5.7 (9.0)	- (0)
3Sa	1.6 (12.1)	2.3 (6.6)	- (0)	- (0)	- (0)
3Sb	- (0)	3.4 (7.1)	- (0)	- (0)	- (0)
4La	3.1 (10.8)	2.8 (13.4)	2.2 (11.2)	1.1 (20.0)	2.4 (32.1)
4Lb	4.2 (12.1)	- (0)	4.8 (15.4)	- (0)	3.8 (32.1)
4Lc	4.9 (10.0)	5.1 (10.1)	5.7 (14.2)	5.2 (20.0)	- (0)
4S	- (0)	2.4 (7.1)	- (0)	- (0)	- (0)
4ST	3.7 (45.2)	3.8 (13.9)	4.0 (13.6)	3.8 (9.0)	- (0)
5La	2.7 (57.4)	3.1 (27.1)	3.3 (8.3)	2.7 (14.5)	2.8 (57.1)
5Lb	4.4 (17.1)	4.6 (30.7)	- (0)	4.0 (20.0)	4.2 (14.2)
5Lc	- (0)	5.2 (21.4)	- (0)	- (0)	- (0)
6La	- (0)	3.4 (21.4)	3.2 (8.3)	3.1 (19.0)	3.8 (32.1)
6Lb	- (0)	4.5 (7.1)	4.4 (16.6)	- (0)	4.3 (28.5)
6Lc	5.1 (17.1)	4.7 (11.9)	- (0)	- (0)	- (0)
7La	3.2 (12.1)	2.7 (11.9)	3.5 (7.7)	2.7 (14.5)	2.1 (14.2)
7Lb	3.6 (10.0)	4.3 (6.6)	4.2 (8.3)	4.4 (9.0)	- (0)
7ST	2.0 (28.5)	2.0 (32.1)	2.0 (15.4)	- (0)	- (0)
8La	3.5 (39.4)	- (0)	2.6 (7.7)	3.0 (14.5)	2.9 (32.1)
8Lb	5.4 (40.0)	4.3 (13.7)	4.2 (11.8)	- (0)	4.0 (14.2)
9La	3.4 (12.1)	2.1 (20.0)	1.9 (16.6)	1.9 (9.0)	1.8 (14.2)
9Lb	3.7 (10.0)	- (0)	3.2 (16.6)	- (0)	- (0)
9ST	2.2 (45.0)	2.3 (18.8)	2.3 (22.5)	2.4 (34.5)	2.0 (14.2)
10L	2.0 (60.0)	2.1 (20.4)	1.9 (25.0)	- (0)	1.2 (50.0)
Total knobs	28	32	28	18	21

1, 2, 3,10

= Chromosome numbers.

a, b, c, d

= Different positions of knobs.

T

= Terminal knob.

S

= Short arm

L

= Long arm

Chromosome 3: The long arm had three knobs each from Pulwama and Baramulla. In Anantnag, only two of these were observed. However, variations in their respective distances from the centromere and also in their frequencies were observed. On the other hand, two knobs were observed in three out of the five groups. The short arm of this chromosome carried two knobs (a and b) from Baramulla, but only one was observed from Anantnag and Pulwama. This arm was knobless in three of the five groups.

Chromosome 4: Anantnag and Pulwama had three knobs on the long arm, while Baramulla had only two. Tripachi and Vozij groups each had three intercalary knobs, whereas in other groups two knobs were present. The short arm knob was only seen in Baramulla and in the Bاده group. In addition to this, the short arm of this chromosome ended in a terminal knob in all three districts and in all the groups except in the "others" group.

Chromosome 5: On the long arm, Baramulla had three knobs, whereas in the other two districts, two knobs were present. Their respective distances from the centromere were somewhat similar. The Bاده group was the only group which had three knobs, and Vozij the only one which had one knob. All the remaining groups bore two knobs each.

Chromosome 6: The long arm knobs (a, b, and c) observed from Baramulla were 3.0, 3.4 and 4.5 u, respectively, away from the centromere; this arm of the remaining two districts had two knobs each at 4.3, 4.4 and 4.3, 4.5 u, respectively. One, two and three knobs were observed on this arm of Tripachi, Niver, Vozij, "others" and Bاده groups, respectively.

Chromosome 7: Two intercalary knobs were present on the long arm in all three districts, as well as in all groups, except the "others" group. Their relative distances from the centromere were approximately equal. Except for the last two groups, the short arm in all three districts, as well as in the remaining groups, ended in a terminal knob.

Chromosome 8: The long arm of this chromosome possessed two knobs in Anantnag and Pulwama at 2.1, 4.2 and 3.4, 4.7 u, respectively. On the other hand, this arm from Baramulla had only one knob at 4.5 u. Three out of five groups (Tripachi, Vozij and "others") bore three internal knobs, while the other two each had one knob on this arm.

Chromosome 9: Both Anantnag and Pulwama had two knobs on the long arm. Their respective locations were 2.9, 3.2 and 2.1, 3.7 u respectively. However, this arm from Baramulla was knobless. Two out of the five groups possessed two knobs. In others one knob was observed. A simple knob and a terminal knob were observed on the short arm of all three districts, as well as in all five groups. The relative positions of terminal knobs were approximately equal.

Chromosome 10: Except for the Niver group, the long arm of all three districts, as well as the remaining groups, possessed a knob. None of the varieties within a group had a consistent knob number and frequency. On the group level the same sort of variation existed. However, there was some consistency in the total knob numbers among the three districts.

The relative position of the knobs on the two arms was not the same in all varieties. This difference was observed among the five groups and also in the three districts. However a few similarities were also noticed, e.g., knobs present on the long arm of chromosome 7 were approximately at equal distances from the centromere. Likewise, the long arm knobs on chromosome 5 were also at more or less equal distances. The terminal knobs observed on the short arms of chromosomes 1, 4, 7 and 9 had approximately equal relative position within the five groups and also among the three districts for a particular chromosome.

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Leaf emergence in eleven inbred lines of maize

We have examined the expression of lesion mimic genes and nuclear genes affecting plastid greening (*ij*, *cm*, *j1*, *j2*) in several inbred lines of maize. These observations are complicated by two factors: (1) differences in leaf shape

and coloration in the inbred lines, and (2) inbred lines planted at the same time have a different number of leaves when measurements on leaf characters are made later in the season. This latter complication means that environmental influences are not comparable for the xth leaf's emergence in the various lines. This past summer we investigated why plants do not have the same number of leaves at specific times during the growth cycle. Ten-seed families of A188, A619, B37, K55, Ky21, Mo17, Mo20W, N6J, Oh51A, Tr, W23 and Wg were planted for three weeks beginning May 17. The field was irrigated the next morning. Leaf number was recorded three times per week for the second planting and once per week for the first and third planting. Emergence was scored when at least 1" of leaf was visible at the top of whorl; measurements continued until tassel emergence. Surprising to us was the observation that all 11 inbred lines had an average leaf emergence rate of 0.375 leaf per day beginning with the 5-6th leaf and continuing for the rest of the season. Inbred lines differed substantially in the amount of time required to reach the 5th leaf stage. In the California environment at least it should be possible to stagger planting dates to synchronize plants of different genetic backgrounds to produce the 5th leaf, and hence all subsequent leaves, on approximately the same day.

Carolyn Thum and Virginia Walbot

Copy number variation of repeated DNA in inbred lines of maize

We have been looking at the extent of copy number variation of repeated DNA sequences in inbred lines of maize. The sequences are highly repetitive, representing 0.2 to a few percent of the genome. We have examined two transcribed sequences (5S and rDNA), a sequence we believe to be a chromosome knob constituent, and various random sequences whose expression and chromosome position we do not yet know. The copy number of the sequences was measured by affixing equal quantities of DNA isolated from 10 inbred lines to nitrocellulose filters through a slotted template, and then hybridizing the filters with nick translated probes of cloned repeated DNA sequences. The intensity of hybridization, measured by scanning the autoradiogram with a densitometer, is a measure of the relative copy number.

Each repetitive probe we have tested shows some copy number variation within the set of inbred lines. The putative knob sequence is the most variable, varying at least sixfold. Other repeated sequences vary in copy number between two and fourfold. DNAs extracted from individual plants of a single inbred line do not show any variation in hybridization intensity.

We find no evidence for a generalized control over sequence copy number. Inbreds with very high numbers of some cloned sequences are on the low end of the scale for others, and vice versa. Pairwise comparisons of the clones did not reveal any more limited copy number coordination between them. Each inbred line was also unique, both for the pattern of hybridization intensity for the various probes and for the actual quantities of each repeat in the genome.

Carol Rivin and Christopher Cullis

Molecular correlates of cytoplasmic types

Cytoplasmic male sterile lines of maize are classified into three groups, T, C and S, based upon which nuclear genes restore fertility (Beckett, *Crop Sci.* 11: 724; Gracen and Grogan, *Agron. J.* 65:654). While field tests lead to conclusive classifications, they are time-consuming; therefore, efforts have been directed toward finding reliable molecular markers of cytoplasmic types. Recently a rapid classification method based upon the occurrence of low molecular weight mitochondrial DNA plasmids was proposed by Kemble and coworkers (*Nature* 284:565; *Genetics*

95:451). These plasmids may be seen after mitochondrial DNA isolated from a few seedling shoots is subjected to agarose gel electrophoresis without restriction endonuclease treatment. Pring et al. (PNAS 74:2904) had previously shown that mitochondrial DNA from S cytoplasms is readily distinguished by the prominent 6.2 and 5.2 kb linear S plasmids. Kemble et al. found that the mitochondrial DNAs from T cytoplasms lack a 2.35 kb linear plasmid present in C, S and normal (N) cytoplasms, and that C is distinguished by additional small plasmid bands of approximately 1.57 and 1.42 kb.

In examining N, T, S and C cytoplasms in B37 (obtained from Pioneer), as well as apparently normal cytoplasms from other inbred lines, we have found that misclassifications may result if the small plasmids are used to determine cytoplasmic type. With B37, the 1.57 and 1.42 kb "C" plasmids were not observed; hence, uncut B37C and B37N mitochondrial DNAs were indistinguishable. Following restriction enzyme digestion, these DNAs were characteristically different. In addition, the male sterility phenotype of B37C was confirmed by genetic tests in the field. On the other hand, the 2.35 kb plasmid is clearly missing in B37T plants and appears to be replaced by a smaller plasmid (approximately 2.2 kb).

When mitochondrial DNA from the fertile inbred Ky21 line was analyzed, it was also found to lack the 2.35 kb plasmid and to have the smaller plasmid reported to be characteristic of T mitochondria. Since Ky21 carries the nuclear restoration genes for all tested T, C, and S male steriles, the possibility was raised that Ky21 has T cytoplasm but is not sterile due to the presence of the dominant Rf1 and Rf2 fertility restoring genes. Genetic analyses will test this proposition: i.e., if Ky21 is cms-T and it is crossed by lines not carrying the nuclear restorers, male sterility should be observed as Rf1 and Rf2 segregate out. We are currently testing F2 and backcross progeny from crosses between Ky21 and B37N for sterile segregants. Other cooperators have not seen male steriles in F2 progeny of Ky21 crosses with maintainer lines (E. Coe, pers. comm.).

We have tested Ky21 for two other molecular correlations with cytoplasmic type: mitochondrial DNA restriction endonuclease digestion patterns (Pring and Levings, Genetics 89:121) and mitochondrially synthesized protein profiles (Forde et al., Genetics 95:443). The Ky21 mitochondrial DNA fragments resulting from digestion with BamHI and XhoI are much more similar to those of B37N than to B37T (or C or S). While several differences between B37N and Ky21 patterns exist, Ky21 lacks all the B37 T-specific bands, including the 6.6 kb XhoI band which has been strongly correlated with the cms-T trait (Gengenbach et al., TAG 59:161; MGCNL 56:140). Mitochondria isolated from cms-T plants synthesize large amounts of a 13,000 MW polypeptide, which is made only at very low levels, if at all, by mitochondria isolated from other cytoplasmic types (Forde and Leaver, PNAS 77:418). Tissue culture-induced fertiles originating from cms-T also synthesize very reduced levels of this 13 kD polypeptide, which strengthens the connection between its synthesis and the expression of the male sterility phenotype (Dixon et al. TAG 63:75). A preliminary study of protein synthesis in isolated Ky21 mitochondria indicates that they do not synthesize elevated levels of the T-associated polypeptide.

Thus, the cytoplasm associated with the Ky21 line would be classified as T on the basis of the rapid test of undigested mitochondrial DNA, due to the absence of a 2.35 plasmid. However, using restriction enzyme fragment and protein synthesis markers, Ky21 would not be considered cms-T. If the Ky21 cytoplasm is indeed normal, it suggests that, while the rapid test can give a useful preliminary diagnosis (especially for cms-S), certain mitochondrial DNA restriction enzyme fragments and mitochondrially synthesized polypeptides may be more reliable molecular tools with which to discriminate between N and cms-T.

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Developmental aspects of cytoplasmic reversion in cms-S

Cytoplasmic reversions to fertility in cms-S maize have been correlated with the loss of freely-replicating S1 and S2 plasmid-like mitochondrial DNAs (plmtDNAs) (Laughnan, Gabay-Laughnan, and Carlson, 1981, Stadler Gen. Symp. 13: 93) and rearrangements of S1 and S2 sequences in high molecular weight mtDNA (Levings et al., 1980, Science 209:1021). These results were obtained with lines established from individual cytoplasmic revertants and propagated for two or more generations.

We have examined several tissues and developmental stages for the presence of S1 and S2 plmtDNAs during the expression of cytoplasmic reversion. Plants were identified in which revertant tassel sectors were large and included the ears within the subtending plant sector. From such sectored revertants, crude mitochondrial lysates were prepared using tissue of both sterile and fertile tassel from the same plant. The mitochondrial lysates were electrophoresed into agarose and the gels stained with ethidium bromide. S1 and S2 plmtDNAs were detected by fluorescence in samples from both sterile and revertant (fertile) tassel sectors, after electrophoresis. That is, cytoplasmically reverted tassel sectors still carried free S1 and S2 at relatively high copy number.

S1 and S2 plmtDNA levels were also assayed in seed from ears included within large cytoplasmic revertant sectors. These ears had been fertilized with pollen from maintainer plants, and seeds throughout the ear had been subsequently confirmed to carry fertile cytoplasm. Dry seeds were powdered, mitochondria extracted and crude lysates electrophoresed into agarose. S1 and S2 were visible by fluorescent staining in only 2 of 11 revertant seed samples. Remarkably, however, after blotting the gels to nitrocellulose and hybridizing with nick-translation labelled S2 probe, S2 plasmid was detected in all of the seed samples, albeit at various levels.

Whereas S1 and S2 plasmids in first generation revertant seed are contributed maternally, they were not found in male gametes of revertants. When mtDNA samples were assayed by Southern hybridization using an S2 probe, pollen from fertile cytoplasmic-revertant sectors showed no S2 hybrid band, just as with "N" pollen, while the S2 hybrid band was detected in mtDNA from cms-S nuclear restored pollen. This indicates a block in transmission of the S1 and S2 plmtDNAs through microsporogenesis in revertant tassels.

In the first and second generations of lines propagated from cytoplasmic reversion mutants, seedlings were assayed for S1 and S2 DNAs both by fluorescent staining and Southern hybridization. S1 and S2 were not detected in mitochondria of several assayed revertant lines following propagation.

It appears that a strict causal relationship cannot be drawn between the presence of autonomous S1 and S2 plmtDNAs and the cms-S phenotype in maize. The vegetative tassel tissues of cytoplasmic revertants still carry the S1 and S2 plasmids at relatively high levels. Similarly, R1 and R2 plmtDNAs have been observed in fertile South American maize races (Weissinger et al., 1982, PNAS USA 79:1). The basis for cytoplasmic reversions to fertility in cms-S maize is not a mutation affecting an all or none control over S1 and S2 plmtDNA levels. The continued presence of S1 and S2 in seed from revertant sectors suggests that, concomitant with the reversion event, replication of S1 and S2 ceases, but a finite number of cell divisions is required to dilute the remnant S1 and S2 levels. The stability of S1 and S2 after replication stops may be the result of covalently bound terminal proteins on these plasmids (Kemble and Thompson, 1983, in press). In microsporogenesis, blockage of transmission of non-replicating DNAs

might explain the absence of S1 and S2 plasmids in pollen from revertant sectors. Further studies are to be attempted with isolated endosperm and embryo mtDNAs of cytoplasmic revertant seed to learn more about post reversion transmission of S1 and S2.

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The induction of micronuclei in root tip cells

Early-Early Synthetic (a rapidly maturing maize inbred) is being calibrated with physical and chemical mutagens to investigate the kinetics of mutation induction and as a monitor for environmental genotoxins. The genetic events under evaluation are forward mutation at the wx locus in pollen grains, forward mutation at the yg2 locus in leaves and chromosome aberrations resulting in micronuclei in root tip cells. Micronuclei are formed from acentric fragments, lagging chromosomes or multicentric chromosomes connected by bridges. A micronucleus test in root tip cells of Vicia faba has been developed by Degraffi and Rizzoni (1982, Mutation Res. 97:19-33).

Kernels heterozygous at the yg2 locus were surface sterilized by soaking in a 0.5% sodium hypochlorite solution for 5 min. Thirty-three kernels per group were soaked for 72 hr in aerated distilled water at 20 C. The kernels were treated for 8 hr at 20 C with aerated solutions of ethylmethanesulfonate (EMS). The concentrations tested were: 0 (control), 1 mM, 10 mM and 20 mM EMS. Following treatment, the kernels were rinsed for 30 min in running tap water and three kernels each were planted in soil in 10 cm diameter plastic pots. The pots were placed in

Table 1. Induction of Micronuclei in Root Tip Cells After Acute EMS Treatment (MCH/No. Cells Analyzed)

Sampled 5 Days After Planting				
Slide No.	Control	1 mM EMS	10 mM EMS	20 mM EMS
1	1/1042	3/1038	1/1033	4/1028
2	0/1074	1/1009	2/1054	4/1018
3	0/1062	1/1054	3/1056	2/1022
4	0/1043	0/1033	2/1039	1/1031
5	0/1061	1/1036	2/1045	3/1062
TOTAL	1/5282	6/5170	10/5227	14/5161
MCN Freq. ($\times 10^{-4}$)	1.89	11.61	19.13	27.13
Probability		P < 0.048	P < 0.004	P < 0.004
Sampled 9 Days After Planting				
Slide No.	Control	1 mM EMS	10 mM EMS	20 mM EMS
1	0/1015	0/1052	3/1080	0/1043
2	0/1035	0/1071	0/1119	1/1053
3	0/1087	1/1039	0/1126	2/1059
4	1/1040	0/1048	2/1047	0/1030
5	3/1047	0/1034	0/1082	0/1031
TOTAL	4/5224	1/5244	5/5454	3/5216
MCN Freq. ($\times 10^{-4}$)	7.66	1.91	9.17	5.75
Probability		NS	NS	NS

NS = Not Significant.

a plant growth chamber at 20 C with a 17 hr photoperiod. Root tips were cut from each group 5 and 9 days after planting and placed in a fixative of ethanol and acetic acid (3:1 v/v). The root tips were fixed overnight. For analysis, the root tips were rinsed in deionized water and placed in 1 M HCl at 60 C for 7.5 min. The root tips were rinsed again and placed in Feulgen stain for 1 hr followed by a 5% pectinase treatment for 1 hr. Slides were made by squashing the root tips in Feulgen stain. The number of interphase cells counted on each slide was approximately 1,000 and five slides were analyzed from each control or treatment group.

The results are presented in Table 1. After 5 days, the micronucleus frequencies for all treatment groups were significantly higher than the control. The concentration of EMS and the induction of micronuclei are highly correlated ($r = 0.94$) and the dose-response curve exhibited linear kinetics (Figure 1).

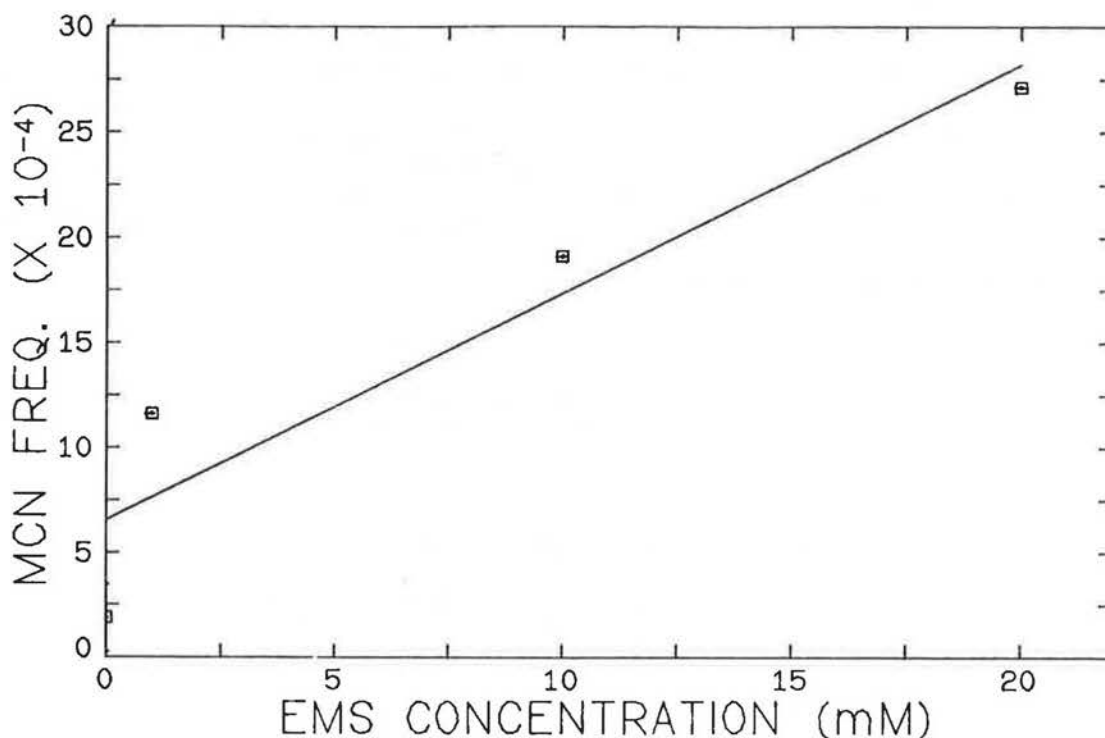


Figure 1

After nine days, the micronucleus frequencies of the treatment groups were not different from the control. The decrease in the frequencies in the treatment groups may be due to two causes, cell death due to gross chromosomal aberrations and the eventual disappearance of micronuclei by the action of cytoplasmic nucleases and proteases. It is interesting to note that the control frequency of 9 days is four times greater than the control frequency of 5 days. There may or may not be an age factor in the spontaneous frequency of micronuclei. However, this demonstrates the importance of conducting concurrent controls with all experiments.

The advantages of the micronucleus test in maize root tip cells are many. It is a relatively easy assay that can be conducted under acute or chronic exposure regimens. It is sensitive to acute treatments with a classic mutagen, EMS. Experiments are currently underway to simultaneously investigate the relationships between forward mutation at the *wx* and *yg2* loci and chromosome aberrations using identical plants. (This research was funded, in part, by NIEHS Grant No. ES01895 Gen.)

Elizabeth D. Wagner and Michael J. Plewa

Forward mutation at the *yg2* locus induced by N-ethyl-N-nitrosourea

Induction of forward mutation at the *yellow-green-2* (*yg2*) locus in *Zea mays* has been used as the genetic endpoint in mutation studies examining both physical and chemical mutagens. The *yg2* assay has proved to be a rapid, inexpensive, and sensitive assay in screening agents for mutagenic activity. We have used the *yg2* assay to study the kinetics of mutation induction following acute exposure to N-ethyl-N-nitrosourea (ENU). ENU is mutagenic in higher plants and animals. ENU is a monofunctional alkylating agent and was found to be the most potent chemical mutagen in the Mouse Specific Locus test (W. L. Russell et al., 1979, Proc. Natl. Acad. Sci. USA 76:5818).

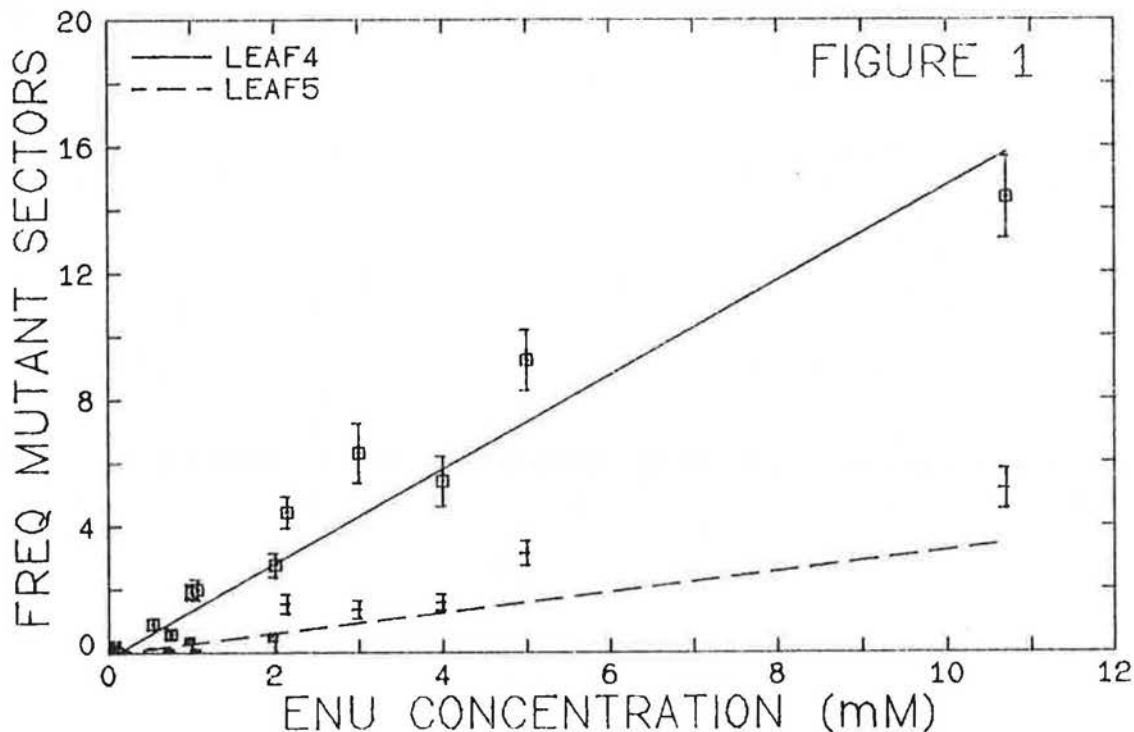
Kernels heterozygous for *yg2* were surface sterilized for 5 min in a 0.5% solution of sodium hypochlorite and rinsed for 10 min in running tap water. The kernels were soaked in aerated distilled water for 72 hr at 20 C. This allows the kernels to become metabolically active, completing one DNA replication during this period. Subsequently, the kernels were treated for 8 hr in various concentrations of aerated solutions of ENU at 20 C. A concurrent control of distilled water was also included. Following treatment the kernels were rinsed in running tap water for 30 min and were planted in soil in 10 cm plastic pots. The pots were placed in a plant growth chamber at 20 C with a 17 hr photoperiod (300 $\mu\text{E m}^{-2} \text{sec}^{-1}$ PRR). Between 20 and 25 days after planting, leaves four and

Table 1. Forward Mutation at the *yg-2* Locus Induced by ENU

Experiment Number	ENU Conc.	No. Leaf 4 Scored	$\bar{x} \pm \text{SE}$	No. Leaf 5 Scored	$\bar{x} \pm \text{SE}$
1002	0	38	0.08 \pm 0.06	36	0.06 \pm 0.04
1002	107 μM	20	0	18	0.28 \pm 0.11
1002	535 μM	26	1.12 \pm 0.25	23	0.09 \pm 0.06
1002	2.14 mM	24	4.29 \pm 0.58	10	2.00 \pm 0.42
1002	21.4 mM	-	Toxic	-	Toxic
1004	0	28	0.18 \pm 0.09	29	0.07 \pm 0.07
1004	535 μM	27	0.70 \pm 0.20	29	0.21 \pm 0.08
1004	1.07 mM	26	2.00 \pm 0.32	26	0.08 \pm 0.05
1004	2.14 mM	19	4.63 \pm 0.87	21	1.13 \pm 0.40
1004	10.7 mM	24	14.37 \pm 1.29	23	5.17 \pm 0.64
1005	0	24	0.29 \pm 0.13	24	0.04 \pm 0.04
1005	1 mM	23	2.22 \pm 0.37	23	0.35 \pm 0.12
1005	2 mM	21	3.19 \pm 0.45	21	0.62 \pm 0.13
1005	3 mM	31	6.32 \pm 0.95	30	1.37 \pm 0.29
1005	4 mM	17	6.82 \pm 1.30	17	2.12 \pm 0.40
1005	5 mM	19	11.68 \pm 1.33	19	3.95 \pm 0.52
1006	0	14	0	15	0
1006	750 μM	38	0.58 \pm 0.13	38	0.08 \pm 0.04
1006	1 mM	16	1.50 \pm 0.29	16	0.31 \pm 0.12
1006	2 mM	10	1.90 \pm 0.64	16	0.38 \pm 0.18
1006	4 mM	14	3.71 \pm 0.62	15	1.00 \pm 0.28
1006	5 mM	15	6.20 \pm 0.89	14	2.07 \pm 0.49

five were excised and scored for yellow-green sectors using a fluorescent light box to illuminate the leaves. Only sectors greater than 1 mm in length were counted. The mean frequency of mutant sectors per leaf was calculated for leaves four and five at each concentration of ENU. These data are presented in Table 1.

The data indicate that concentrations of 535 μ M ENU or greater induce a significant increase in the frequency of mutant sectors over control values. The data from four experiments were compiled and exhibit linear kinetics for mutation induction for leaves four and five (Figure 1). The slopes of the dose-response



curves for leaves four and five were 1.40 and 0.50, respectively. The induction of forward mutation at the *yg2* locus is highly correlated with the concentration of ENU for both leaves ($r = 0.97$). Clearly, ENU induces forward mutation in somatic cells of maize. (This research was funded, in part, by NIEHS Grant No. 5 R01 ES01895 GEN.)

William E. Schy and Michael J. Plewa

Induction of forward mutation at the *yg2* locus by gamma radiation

We conducted an investigation using the yellow-green-2 (*yg2*) locus as the genetic endpoint for the induction of mutation in somatic cells following exposure to ionizing radiation. The purpose of this research was to determine the sensitivity of the *yg2* locus to ionizing radiation and to develop a standard for use in comparing genetic damage induced by chemical mutagens.

Maize plants heterozygous at the *yg2* locus have a normal green phenotype. Loss of the dominant allele (*Yg2*) by point mutation or a chromosome break will allow the expression of the recessive *yg2* allele as a yellow-green sector in the leaf.

The fourth and fifth leaves are scored in this assay. Dormant maize embryos contain 1,500 primordial cells for leaf four and 250 primordial cells for leaf five (H. H. Smith and H. H. Rossi, Rad. Res. 28:302).

Kernels heterozygous at the *yg2* locus were used in these experiments. The kernels were surface sterilized by soaking for 5 min in a 0.5% sodium hypochlorite solution and rinsing for 10 min in running tap water. For each treatment group 33 kernels were soaked for 72 hr at 20 C in aerated distilled water. This procedure allows for one cell division to occur, thus the target population size is approximately 3,000 and 500 cells for leaf four and five, respectively (B. V. Conger and J. V. Carabia, 1977, Mut. Res. 46:285). The kernels were treated by exposure to $^{137}\text{Cesium}$ in a J. L. Shepard irradiator. The radiation doses were: 0 (control), 50, 100, 250, 500 and 1,000 rads of gamma radiation. Following treatment, the kernels were planted in soil in 10 cm diameter plastic pots, three kernels per pot. The pots were placed in a plant growth chamber at 20 C with a 17 hr photoperiod ($300 \mu\text{E m}^{-2} \text{sec}^{-1}$ PRR) for 20 to 25 days. The fourth and fifth leaves were scored for the presence of yellow-green sectors with a minimum length of 1 mm. A fluorescent light box and magnifying lens were used as aids in scoring.

The results of the experiments are presented in Table 1. The data are expressed as the mean number of yellow-green sectors per leaf four or leaf five.

Table 1. Forward Mutation at the *yg-2* Locus Induced by Gamma Radiation

Experiment Number	Gamma Rads	No. Leaf 4 Scored	$\bar{x} \pm \text{SE}$	No. Leaf 5 Scored	$\bar{x} \pm \text{SE}$
4625	0	30	0.13 ± 0.06	30	0
4625	100	32	0.65 ± 0.13	32	0.10 ± 0.05
4625	250	32	3.75 ± 0.29	32	0.50 ± 0.13
4625	500	32	9.19 ± 0.58	32	1.22 ± 0.16
4625	1000	Toxic	-----	Toxic	-----
4629	0	32	0.09 ± 0.05	32	0
4629	50	30	0.60 ± 0.13	30	0.07 ± 0.05
4629	100	33	1.48 ± 0.23	33	0.39 ± 0.11
4629	250	32	4.19 ± 0.54	32	1.06 ± 0.17

The variance within each group is expressed as the standard error (SE) of the mean. The data for leaves four and five are separately compiled and plotted in Figures 1 and 2. The primordial cells of both leaves are very sensitive to mutation induction by gamma rays.

For leaf four, the frequency of yellow-green sectors per leaf ranged from a mean of 0.11 ± 0.04 for the control to 9.19 ± 0.58 for kernels exposed to 500 rads. A radiation dose of 1,000 rads extensively damaged the leaves and they were unscorable. The dose-response curve for leaf four exhibited linear kinetics, especially within the range of 0 to 250 rads (Figure 1). The induction of yellow-green sectors and the dose of gamma were highly correlated ($r = 0.97$). The frequency of yellow-green sectors per leaf for leaf five ranged from 0 for the control to 1.22 ± 0.16 for kernels exposed to 500 rads. As in the case of leaf four, a dose of 1,000 rads severely damaged the fifth leaf of the plants. Although the mean frequency of sectors per leaf five was considerably lower than

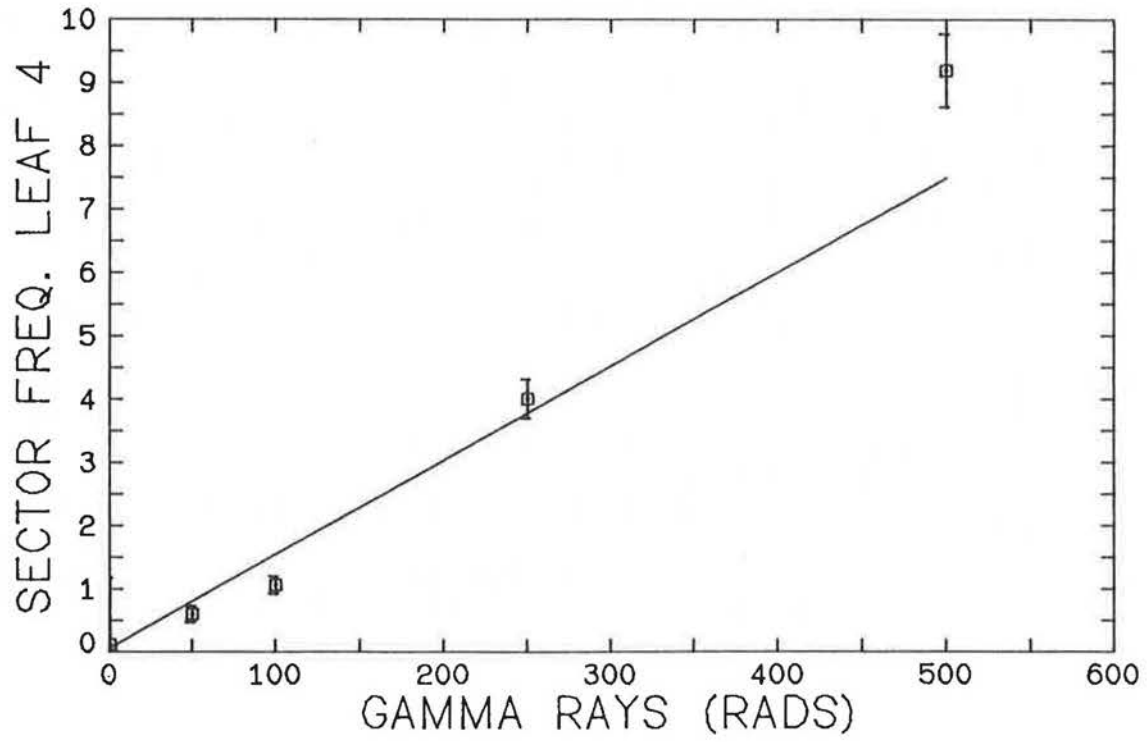


Figure 1

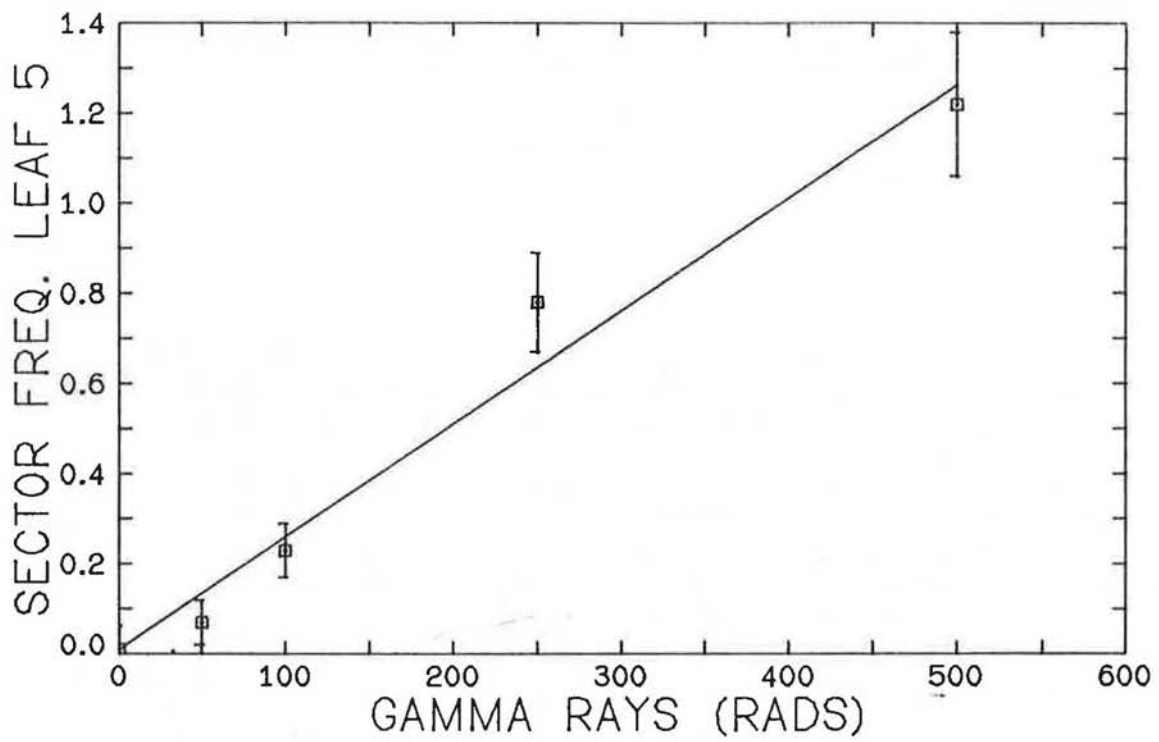


Figure 2

leaf four, the dose-response curve exhibited linear kinetics throughout the dose range analyzed (Figure 2). The induction of leaf sectors and the radiation dose were highly correlated ($r = 0.98$).

The yg2 locus is a sensitive and a relatively rapid assay for studies in environmental mutagenesis involving higher organisms. The data on gamma radiation presented here shall serve as a standard to compare the effective mutagenicity of chemical agents. (This research was funded, in part, by NIEHS Grant No. ES01895 GEN.)

Patrick A. Dowd and Michael J. Plewa

Induced forward mutation at the yg2 locus and a comparison with the ABCW relationship

The rates of mutation induced by ionizing radiation differ greatly among organisms. The induced specific locus mutations per locus per rad ranges from 1×10^{-9} in bacteria to 5×10^{-6} in angiosperms. In 1973 Abrahamson, Bender, Conger and Wolff (Nature 245:460) discovered a direct correlation between radiation-induced mutation and the DNA content per haploid genome. This correlation has been commonly referred to as the ABCW relationship. Their survey of the literature encompassed data from prokaryotes, lower eukaryotes and higher eukaryotes. The ABCW relationship suggests that it is the nucleus, not the gene locus, that determines the size of the target for mutation. J. A. Heddle and K. Athanasiou (Nature, 1975, 258:359) suggested three explanations for the ABCW relationship. These are: (1) the target for mutation is proportional to the haploid DNA content of the nucleus, (2) the DNA repair efficiency is inversely proportional to the genome size, and (3) the size of the mutational event is proportional to the size of the genome. The concept of the ABCW relationship has been the subject of controversy. Schalet and Sankaranarayanan doubt that the relation is valid (Mutation Res., 1976, 35:341; Mutation Res., 1978, 49:313).

We compared the rate of forward mutation in maize induced by ionizing radiation with the ABCW relationship. Mutation was induced at the yellow-green-2 (yg2) locus with gamma radiation. Kernels heterozygous at the yg2 locus were soaked for 72 hr in aerated distilled water at 20 C. The kernels were irradiated with various doses of gamma rays. The kernels were planted and yellow green sectors were scored in leaves four and five. The doses of gamma radiation used were 0 (control), 50, 100, 250, and 500 rads. The response in this dose range in terms of the mean frequency of yellow green sectors per leaf four was 0.11, 0.60, 1.06, 4.00, and 9.19, respectively. The mean frequency of sectors for leaf five was 0, 0.07, 0.23, 0.78, and 1.22, respectively. A complete discussion of the experiment is presented in the accompanying note by Plewa.

In these experiments and in the interpretation of the dose response data we make the following assumptions. (1) A yellow green sector is the result of the loss of the Yg2 allele. The term "forward mutation" is used to describe the loss of the phenotype of the dominant allele (Yg2). We realize that this may be due to a terminal deletion on chromosome 9 that includes the yg2 locus or to a true point mutation at the yg2 locus. (2) A single yellow green sector is due to a single mutational event. (3) The number of targets for mutation (loci) is equal to the number of cells in the leaf four primordium (3,000) and leaf five primordium (500) at the time of irradiation.

From the above data we calculated the induced forward yg2 mutations per locus per rad (m) according to equation (1).

$$m = S_j/N_0)(r) \quad (1)$$

where S_i is the frequency of induced mutant sectors, N_0 is the number of target or primordial cells for a specific leaf at the time of irradiation, and r is the dose of gamma rays in rads.

The frequency of induced mutant sectors was calculated by equation (2).

$$S_i = S_t - S_c \quad (2)$$

where S_t is the mean frequency of mutant sectors per specific leaf at a specific dose of radiation and S_c is the mean frequency of sectors per leaf from the relevant control (0 rads).

The induced mutant sectors per locus per rad for leaves four and five for each dose of radiation are presented in Table 1. The mean and standard error of the mean of yg2 mutations per locus per rad was calculated to be $4.5 \pm 0.5 \times 10^{-6}$. Note that the calculated mean m values for leaf four and leaf five were very similar (4.4×10^{-6} and 4.6×10^{-6} , respectively).

Table 1. Induced yg-2 Mutant Sectors Per Locus Per Rad for Leaf Four and Leaf Five at Various Doses of Gamma Radiation

Leaf No.	Rads	(m) Mutations/Locus/Rad ($\times 10^{-6}$)
4	50	3.3
4	100	3.2
4	250	5.2
4	500	6.0
5	50	2.8
5	100	4.6
5	250	6.2
5	500	4.9

Mean m value \pm SE for leaf four = $4.4 \pm 0.7 \times 10^{-6}$

Mean m value \pm SE for leaf five = $4.6 \pm 0.7 \times 10^{-6}$

Mean yg-2 mutations/locus/rad = $4.5 \pm 0.5 \times 10^{-6}$

We compiled data from the papers of Abrahamson et al. (1973) and Heddle and Athanasiou (1975) and replotted the haploid DNA content vs. forward mutation per locus per rad (Figure 1). The correlation expressed graphically in Figure 1 is the ABCW relationship. To determine if the ABCW relationship was supported with the data we obtained on induced mutation at the yg2 locus by gamma radiation, we plotted the position for maize. The DNA content per haploid genome of maize is 5×10^{12} daltons and the mean m value for the yg2 locus is 4.5×10^{-6} .

In Figure 1 two points are plotted for maize, one that includes the data for 50 to 500 rads and one that includes the data from 50 to 100 rads only. The best fit of a curve that represents the relationship between the mutation rate and the size of the haploid genome was demonstrated by the method of least squares on the logarithms of the data. The curve represents a fit to the equation $y = ax^b$ where y is the mutation rate and x is the size of the genome. The curve plotted in Figure 1 has the parameters of $a = 1.15 \times 10^{-18}$ mutations per locus per rad per

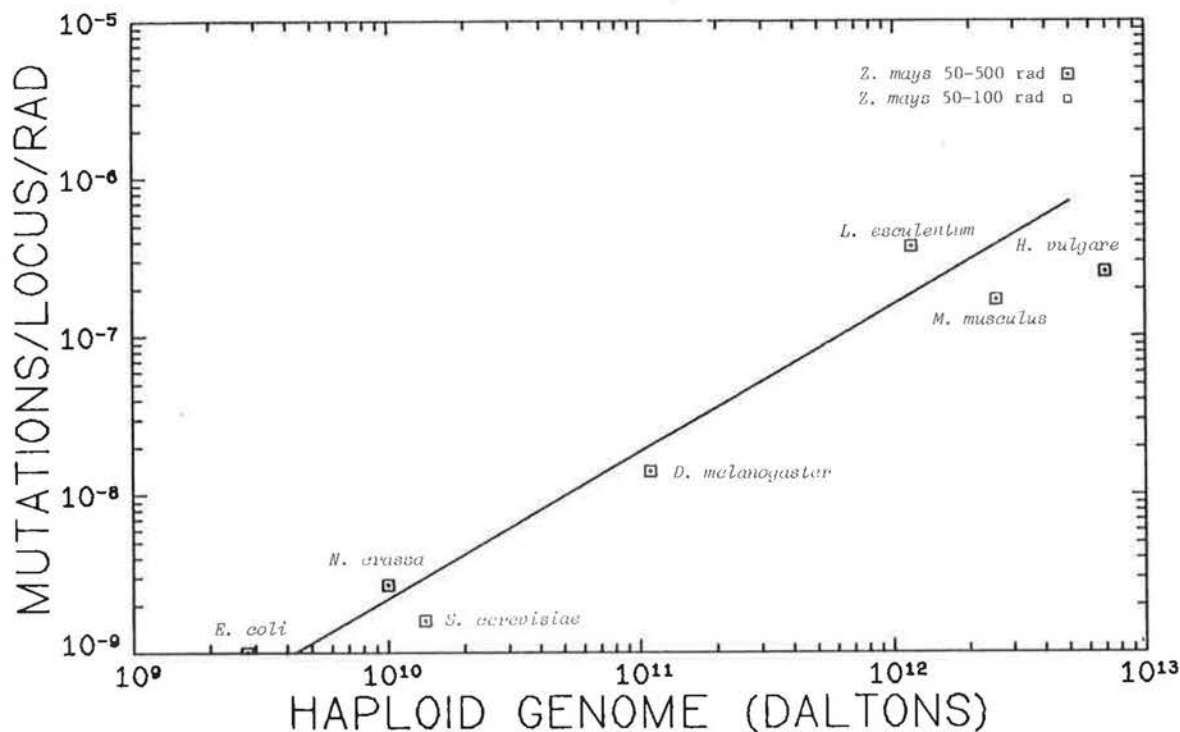


Figure 1

dalton and $b = 0.93$. The coefficient of determination of linearity (r^2) is 0.89. The maize data (50 to 500 rads) were included in this fit of the curve.

The deviation of the point for maize is obvious. This may be due to a number of factors. The data used by Abrahamson et al. were based on X-ray induced mutations, while our data were generated with gamma rays. Since the LET and RBE for X and gamma rays are similar we believe it reasonable to plot the maize data in this comparison. The loss of the dominant phenotype, green leaf, in a yellow green sector can be due to a point mutation or to a chromosome deletion. The data for maize involve both events. It would be interesting to speculate how much the point for maize would be lowered if it was possible to differentiate the sectors that were due to chromosome deletions that included *yg2* and point mutations at the *yg2* locus. Finally, it must be stressed that all of the points represented in Figure 1 were derived from the literature, while the maize data were generated as an experimental test of the ABCW relationship.

We conclude that the data generated at the *yg2* locus with ionizing radiation support the ABCW relationship. This indicates that induced mutation rates observed in angiosperms may not be significantly higher when compared to other organisms if the number of target loci and the haploid DNA content per cell are considered. (This research was funded, in part, by NIEHS Grant No. ES01895 GEN.)

Michael J. Plewa, Patrick A. Dowd,
William E. Schy and Elizabeth D. Wagner

Opaque 9 is tentatively located to the long arm of 5

The Maize Genetic Cooperation Stock Center has a continuing program to screen, allele test, locate to chromosome and map new potentially useful genetic markers.

A series of opaque candidate stocks was tested this past summer using the TB series. As a result of these tests opaque-9 has been tentatively located to the long arm of chromosome 5. Additional confirmatory tests will be run next summer.

Gilbert B. Fletcher

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a3 - shrunken linkage

A plant segregating a shrunken phenotype 'appeared' in our a3 stocks and gave the following segregation values on self-pollination and backcrossing to a3 (all plants A A):

A3 sh/a3 Sh selfed-- 3 A3/- sh sh : 9 A3/- Sh sh : 2 A3/- Sh Sh:

3 a3 a3 Sh sh : 3 a3 a3 Sh Sh

a3 a3 Sh Sh x A3 sh/a3 Sh-- 8 a3 Sh/a3 Sh: 1 a3 Sh/a3 sh: 6 A3 sh/a3 Sh :

4 A3 Sh/a3 Sh

The data indicate approximately 26% recombination between a3 and the shrunken locus. We assume that the shrunken is shrunken-2 and have made crosses with a for confirmation.

E. Derek Styles

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Suburban Experiment Station, University of Massachusetts

Pointed kernels, embryo-endosperm competition and oil content

The pointed shape of kernels is controlled by a single gene within a complex of genes on chromosome 4 that is important in separating the ears of teosinte and maize (P. C. Mangelsdorf and W. C. Galinat, 1963, MNL 37:30-31). Pointed kernels will fit snugly into either an elongate fruit case, as in Guatemalan teosinte, or a pod corn enclosure. Pointed kernels also have a higher embryo/endosperm ratio. Because corn oil is extracted chiefly from the embryo, the pointed kernel trait may be economically important in breeding for increased oil content. The longer embryos may have evolved in annuals (e.g., Palomero Toluqueño maize) as an adaptation to cool climates requiring a head start by rapid germination and early leaf display.

Walton C. Galinat

The efficiency of the Megasort 6 Machine from Geosource in separating white and yellow kernels within inbreds MA-400 and IL-677a

The physical basis for using the close linkage of the y (white endosperm) and ms (male sterile) genes to eliminate detasseling in the production of hybrid seed depends upon the ability of a seed sorter to separate white from yellow kernels.

About 4.6 lbs of hand-pollinated MA-400 segregating ca. 50% y ms, and about 2.4 lbs of IL-677a segregating ca. 50% y ms, were run by Mr. Al Rodriguez of Geosource in Houston, Texas through their Megasort 6, 7-ring light machine, at the rate of 250 lbs per hour. In each case, the kernels sorted as white (y ms) by the machine still carried 6 to 7% of the most pale yellow kernels. Apparently the machine can be adjusted to remove more of the yellow. It is significant that no white kernels were observed in the yellow and there was a slight deficiency in yellow kernels (46% instead of 50%). Also, if the sorted white kernels were sent through the machine a second time with the kernels in different positions, it would probably remove the balance of the yellow kernels. That is, if the electric eye just sees the white scutellum of a yellow kernel, it may respond as if the kernel were white.

The following data on the efficiency of the Megasort 6 operation were obtained by hand-picking the white for yellows accepted in error by the machine:

	<u>W400</u>	<u>677a</u>
Total machine-sorted as white	4390	3488
Yellows accepted as white	268	262
Percent efficiency of machine	94	93

During the backcrossing of the y ms segment into a given inbred, selection must be practiced to insure good color contrast. For some inbreds with dingy pericarp color (such as C-13) it is difficult to get acceptable contrast.

The y ms system seems ready to be put into use, at least as an alternative means when all sterile cytoplasms fail. In addition to various y ms sweet corn inbreds, B73 and MO-17 have been converted to y ms. They are in the hands of seed producers (Pioneer, Acco & Funks).

Walton C. Galinat

Pale blue aleurone as a tracer for multiple aleurone under two-celled pericarp

When aleurone color is intensified by a thicker and/or multiple-celled aleurone, it may be used to facilitate transfer of this trait by reducing the tedious task of sectioning hundreds of kernels. We selected a recessive pale blue aleurone color that will serve this purpose. This is important to us because we are attempting to recombine the two-celled pericarp, derived from teosinte, with a thick multiple-celled aleurone, from the race Coroico of South America, in an acceptable sweet corn background. The teosinte-type pericarp is lethal in a typical maize background with a single-celled aleurone. The pericarps split in the early milk stage and the kernels are soon destroyed by mold. But the teosinte pericarp is coadaptive with the thick aleurone, which substitutes for it in terms of containing turgor pressure from the endosperm.

Typical North American corn has a pericarp from 7 to 20 cells thick, although in certain extra tender sweet corn (Hayes White) it may be only 5 or 6 cells thick, or in high expansion popcorn 30 or 40 cells thick. But in the wild ancestor of corn the pericarp is only two cells thick because it has reinforcement from the superstructure of a fruit case. In Coroico of Bolivia and Peru, the thick aleurone apparently evolved when an early domesticate from teosinte, still with a thin pericarp, started down an independent pathway of a system allowing kernel expansion. Coroico has a pericarp often 3 or 4 cells thick with an aleurone of equal or greater thickness.

Walton C. Galinat, Josephine Starbuck and Chandra V. Pasupuleti

Is the heterosis of the Corn Belt Dent derived from the interspecific vigor of independent domestications of Mexican and Guatemalan teosinte?

Since the early studies of H. A. Wallace and W. L. Brown, of E. Anderson, of G. S. Johnston and others, the heterosis of Corn Belt Corn has been shown repeatedly to stem from combining Northern Flint-like inbreds with Southern Dent-like ones.

There are a number of floral and cytological traits that seem to associate the Northern Flints with Guatemalan teosinte on the one hand, and the Mexican teosintes with the Southern Dent on the other. The Northern Flints and Guatemalan teosinte have reduced floral condensation, flat staminate glumes and lack internal chromosome knobs. The Southern Dents and the Mexican teosintes have a higher level of floral condensation, round staminate glumes and internal chromosome knobs. There are several objections to this new hypothesis which I cannot at present explain, but this does not mean that the hypothesis is invalid. Rather, the objections may reflect inadequate information. The Guatemalan teosinte has terminal knobs unknown in corn--its Northern Flints included. Certain of these terminal knobs (or large chromomeres) are known to extend the length of the chromosome over its homologue in corn (C. V. Pasupuleti and W. C. Galinat, 1982). But why and how should such knobs be shed during an origin of the ancestor of the Northern Flints? Is a primitive form of Nal Tel such a domesticate of Guatemalan teosinte and the ancestor of the Northern Flint? Where is the archaeological record of such origin by domestication? The early archaeological record is incomplete, if not absent, but it may be discovered.

Walton C. Galinat

Coadaptation of cupule, kernel type and kernel row number

There are a series of multiple alleles for degree and induration of cupule development on the short arm of chromosome 4. As reported in MNL 56:163-164, the type of cupule has become coadaptive with kernel type in terms of spatial accommodation. Hard kernels, which do not shrink significantly on drying, will fly off from a soft cob that shrinks about 10% as it dries. Thus, large, hard kernels are coadaptive with hard cobs. Because of the transfer of cupule function involved, the cupule of teosinte has been retained in maize these last 8,000 years since domestication.

But the cupule is not now needed in sweet corn and other defective endosperm types with kernels that do shrink at least 10% on drying. In fact, a soft cob resulting from cupule reduction becomes coadaptive with defective kernels so that the cob will shrink along with the kernels, which thereby do not shatter by becoming too loose. Furthermore, the physical space required by the cupule at higher kernel row numbers promotes objectionable fasciation. By removing the cupule with the cupuleless allele on chromosome 4, we may expect to increase kernel row numbers in sweet corn to 40 or more without fasciation.

Walton C. Galinat and Ann E. Kennedy

The longevity of corn seed in cold storage

The oldest of the Mangelsdorf collection of Latin American corn seed, now about 40 years in cold storage in sealed glass bottles held at 40 F, appears to be dying off at a slightly higher rate than the 35-year-old seed stored in the same way. We had not expected this seed to live indefinitely and it is important to know how soon life begins to taper off.

Age	Germination Percent from 4 Good Kernels					Total
	0	25	50	75	100	
40 yrs	37	16	18	8	22	51
35 yrs	17	20	21	26	15	143

Because of the shortage of this precious seed, no further germination tests are planned. We have sent 1,792 samples of 5 kernels each to Dr. M. M. Goodman of North Carolina State University. He will attempt to increase the seed by hand pollination in Florida this winter.

Walton C. Galinat and Josephine Starbuck

Panicle vs. spike as secondary sex traits of tassel and ear

The normal structure of the tassel is that of a panicle, and that for the ear is a spike, because this is the most efficient arrangement for their sexual functions. But as most corn breeders are aware, certain variants have a single-spike tassel and a branched or panicle-type ear. Usually such variants retain the normal structure in one of the unisexual inflorescences and just extend this structure into that of the other sex. We have intercrossed the spike-tassel type with the panicle ear type to determine if through recombination we can obtain a reversal in sex programming for inflorescence structure.

Walton C. Galinat

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 USDA-ARS

Resistance to *Cercospora zae-maydis* in the A158 teosinte derivative carrying chromosomes 1, 7 and 9 from Durango teosinte

Results from inoculating the A158 teosinte derivatives developed many years ago by Mangelsdorf show that the A158 Dur 1, 7, 9 stock carries a high level of resistance to *Cercospora zae-maydis*, gray leaf spot disease. The A158 control and the various other teosinte derivatives were all highly susceptible. We assume that the resistance factor was introduced on one of the three chromosomes transferred from Durango teosinte. Although this teosinte is apparently now extinct, the maintenance of its A158 derivatives at Waltham has allowed its post-mortem contribution.

In order to identify which of the three chromosomes carries the resistance gene, we have crossed the Dur 1, 7, 9 derivative with the WMT marker stock. The F2 populations will be screened by Frances Latterell using the P p (red vs. white cob) marker for chromosome 1, the Gl gl (non-glossy vs. glossy seedling) for chromosome 7 and Wx wx (non-waxy vs. waxy endosperm) for chromosome 9.

Walton C. Galinat and Frances M. Latterell

ADDENDUM

BALTIMORE, MARYLAND
Carnegie Institution of Washington

Notes on cloning maize DNA

The lambda-1059 strain (J. Karn et al., PNAS 77:5172-5176, 1980) and its more recent EMBL derivatives (A.-M. Frischauf, H. Lehrach, A.-M. Poustka and N. Murray, in preparation) are in widespread use as cloning vectors. The *E. coli* Q358 strain (r^{-k} , m^{+k} , Su^{+II} , 80^R) is generally used as the permissive host for phage propagation, and the P2 lysogens Q359 (r^{-k} , m^{+k} , Su^{+II} , 80^R , P2) or Q364 (r^{-k} , m^{+k} , Su^{+II} , delta-lac-pro, P2) are used as the selective hosts for detection of recombinant phage (J. Karn et al., 1980). I have consistently found that phage constructed in vitro using purified Eco RI-cut arms of the EMBL4 vector, and either total Eco RI-cut maize DNA or maize EcoRI fragments in the 15-25 kb size range, plate much less efficiently on Q358 or Q364 than on other *E. coli* strains permissive for lambda replication. The data in Table 1 show the results of such

TABLE I
Relative plating efficiency, in p.f.u./ug phage-equivalent DNA,
of recombinant lambda phage

<u>DNA</u>	<u>E. coli strain</u>		
	Q358	Q364	K803
<u>Experiment 1</u>			
RI EMBL4 arms, 15-25 kb maize DNA from 5-day seedlings	7.2×10^4	1×10^5	1.1×10^6
RI EMBL4 arms	7×10^3	---	1.8×10^4
RI-cut, religated EMBL4 DNA	2.7×10^6	---	3.4×10^6
EMBL4 DNA	2.0×10^8	---	2.3×10^8
<u>Experiment 2</u>			
RI EMBL4 arms, total RI-cut <i>C. elegans</i> DNA	9.4×10^4	4.1×10^4	1.3×10^5
RI EMBL4 arms, total RI-cut maize DNA from 5-day seedlings	1.9×10^4	5×10^4	2.7×10^5
RI EMBL4 arms, total RI-cut maize DNA from 6-week plants (primarily immature tassels)	1.4×10^4	4.3×10^4	2.3×10^5
RI EMBL4 arms, total RI-cut maize DNA from 3-week plants	2×10^4	7×10^4	3.6×10^5
RI EMBL4 arms	7.3×10^3	1.9×10^3	8.5×10^3
EMBL4 DNA	3.1×10^8	$<10^6$	3.1×10^8

experiments, using the Q358, Q364 and K803 (strain LE392 [F^{-} , $hsdR514$ (r^{-k} , m^{-k}), $supE44$, $supF58$, $lacY1$, $galK2$, $galT22$, $metB1$, $trpR55$]; T. Maniatis, E. F. Fritsch and J. Sambrook, Molecular Cloning, Cold Spring Harbor Laboratory, Cold Spring Harbor, N.Y., 1982) strains as host bacteria. Although the number of p.f.u.'s is smaller when a total EcoRI digest of maize DNA is ligated into the EMBL4 vector than when 15-25 kb fragments of maize DNA are used, it is consistently observed

that the plating efficiency of the newly-packaged phage containing maize DNA is higher on the K803 strain by a factor of 13-18 than it is on the Q358 strain. This is not true of either the parental EMBL4 phage DNA, EMBL4 phage DNA that has been cut with EcoRI and religated, or recombinant phage constructed using *Caenorhabditis elegans* DNA (a gift of D. Burke). Phage containing these DNAs plate with approximately equal efficiency on both strains. The plating efficiency of maize DNA-containing phage is slightly higher on the selective host Q364 than on Q358, while the plating efficiency of phage containing *C. elegans* DNA is somewhat lower on the selective host (Table 1; D. Burke, personal communication).

To determine whether the inability to form plaques on Q358 is an inherent property of recombinant phage containing maize DNA, recombinant phage were propagated on K803 and tested for their ability to grow on the various strains. The results are shown in Table 2 and indicate that once the maize DNA has been

TABLE II

Titration of EMBL4 and recombinant phage on different host strains

Phage	Titer (p.f.u./ml)		
	Q358	Q364	K803
EMBL4	1.7×10^{10}	$<10^8$	1.7×10^{10}
Recombinant phage with maize DNA inserts	3.1×10^{10}	2.5×10^{10}	3.6×10^{10}

"laundered" through *E. coli*, the recombinant phage grow equally well on Q358, Q364 and K803. Since plant DNA's are known to be more extensively modified than other DNA's, it appears a reasonable conjecture that the difference in plating efficiency is attributable to differences among *E. coli* strains in the ability to replicate heavily modified DNAs.

Nina Fedoroff

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Univ. of California

Nonautonomy of the 10L small kernel effect

Crosses of chromosomally normal female stocks by certain of the B-A translocations result in a fraction of the progeny with hypoploid endosperms that lack the B^A paternal chromosome and that exhibit a small kernel phenotype. Lin (1982, Genetics 100:475) has shown that the small kernel effect does not merely result from the deficient state, since additional maternal doses will not restore the normal size.

Previous studies have shown that this phenotype did not function in a cell autonomous manner (Birchler, Genetical Research 36:111-116). The example studied involved the most extreme case of the small kernel phenotype, that is, TB-1La-5S8041 crossed to an a2 tester. Among the progeny were mosaic A2/a2 endosperms that had lost the paternal 1L-5S element as early as the first nuclear division. Even in these early events, there was no evidence of reduction in size in the a2 portion of the kernel. Determination of kernel mass did not reveal any

reduction in these mosaics despite the fact that the small kernels, which were deficient for a paternal contribution at fertilization, were only 45% as large as the normal siblings.

In addition to the 1L-5S compound, 1L-3L translocations were noted to show nonautonomy in crosses to an a R-scm2 tester. Subsequent studies of 1L-5S crosses to an a2 R-scm2 stock confirmed the earlier observations on 1L-3L, that the mosaics were almost exclusively observed in kernels that carried only a single copy of the translocation in the endosperm. This fact was concluded from the observation that the mosaics always had anthocyanin development in the scutellum, and thus this class must have arisen from fertilization by pollen in which nondisjunction of the B centromere did not occur. Similar studies using a bz2 R-scm2 tester and TB-1La also indicated that the 1L small kernel effect showed nonautonomous behavior as evidenced by normal sized Bz2/bz2 mosaic kernels. These also occurred in endosperms resulting from inheritance of single dose of 1L from the TB-A parent.

While the above observations were consistent, they basically tested only the autonomy of the 1L small kernel effect regions. In the course of further studies on the effects of chromosomal dosage on gene expression, TB-10L18 and TB-10L19, both of which show the extreme 10L small kernel effect (Lin, 1982), were crossed to an r-r tester obtained from Jack Beckett. This female line appears to promote a high rate of nondisjunction or loss of the B-10L elements during endosperm development. Both translocation crosses produced numerous R/r endosperms. Thirty-three mosaic kernels were observed in crosses with TB-10L18. All had anthocyanin in the scutellum, indicating they arose from fertilizations by pollen carrying one dose of 10L in both sperm. All mosaics were within the normal sized range. The kernel mass means follow.

Phenotype	n	Mean \pm s.e. (g)
anthocyanin in embryo; none in endosperm	36	0.154 \pm 0.004
anthocyanin in embryo and endosperm	38	0.220 \pm 0.002
anthocyanin in endosperm; none in embryo	23	0.216 \pm 0.003
anthocyanin in embryo; mosaic endosperm	33	0.210 \pm 0.002
colorless embryo and endosperm	19	0.206 \pm 0.008

These observations indicate that the 10L as well as 1L small kernel effects exhibit nonautonomy.

James A. Birchler

Preliminary characterization of a derivative allele of an unstable regulatory mutant at Adh1

A Robertson's Mutator-induced mutant at Adh1, Adh1-S3034, has been characterized as low in expression of enzyme activity, protein (CRM), and mRNA to approximately 40% of the wild type level. It is also genetically unstable: pollen stained for ADH activity shows grains with greater and lower levels of ADH. Two derivative alleles, S3034a and S3034b, having 0% and 13% levels of enzyme activity and mRNA relative to wild type Adh1-S, have been described previously (Freeling, Cheng and Alleman, 1982, *Devel. Genet.*, in press; Strommer, Hake, Bennetzen, Taylor and Freeling, 1982, *Nature* 300:542). A 1.35 kb insert (Mu1) in S3034 is unchanged in the derivatives at the level of resolution possible by restriction site mapping studies.

We have selected a derivative allele of S3034 which is altered in the protein produced by the Adh1 gene. This derivative, Adh1-S3034x1, produces a product which forms an active dimer with the electrophoretic variant allozyme subunit ADH1-F. The allozyme profile of Adh1-S3034x1/Adh1-F heterozygotes show that the S3034x1-F dimers migrate to the same position as S-F dimers. The allozyme ratio as well as CRM levels in the mutant homozygotes implies that 15-20% product is formed. S3034x1 homozygotes, however, have no detectable ADH enzyme activity.

Unpublished data of Hake, Taylor, Strommer and Bennetzen suggested that the inserts of Adh1-S3034 and the derivatives a and b are in the first intervening sequence of the gene; sequence information verified this (intron sequence communicated by W. J. Peacock; point of insertion is from unpublished sequence data from J. Bennetzen and J. Strommer). Since none of these alleles have been shown to alter the protein product, it was thought that the derivative S3034x1 could test for transposition of the Mu1 element. Gross transposition is not the case because of the following data: restriction site mapping shows that S3034x1 maps to the same location as S3034, a and b and has a BstEII site in the same place within the insert. There are no additional inserts elsewhere in the gene. The level of resolution of restriction site mapping using the size of DNA fragments generated in these experiments is not great (approximately 50 b.p.). Since the intron/exon junction nearest the Mu1 insertion is close, changes that could alter coding information but which could not be detected by genomic southern blots is possible. Although small scale rearrangements around the Mu1 insertion site have not been ruled out by these tests, simple transposition or excision of the element has. One of the many other derivatives that remain untested may provide the evidence for transposition.

It is easy to concoct hypotheses that could explain how insertions within an intron could lead to different mRNA levels. How Mu1 has come to affect coding sequence is more difficult to understand.

Mary Alleman and Michael Freeling

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Hybridization studies of Barley Stripe Mosaic Virus cDNA clones to virus induced maize mutants

Some multicomponent RNA virus strains of Barley Stripe Mosaic Virus (BSMV) or Wheat Streak Mosaic Virus (WSMV) are known to act as mutagenic agents in corn and induce mutations unspecifically (G. F. Sprague et al., Science 141:1052, 1963). The majority of these mutations are stable, although an unstable mutation has been identified recently (P. Friedemann and P. A. Peterson, Mol. Gen. Genet. 187:19, 1982).

Whether this mutagenic effect is correlated with the integration of viral sequences into the maize genome was tested by Southern type hybridization experiments. Therefore, cDNA clones from the BSMV four component strain Argentina mild (kindly provided by M. Brakke, Lincoln) were constructed and used to probe virus induced mutant maize lines. The two mutant lines tested were originally isolated as aberrant ratio lines 874-30 and 74, 287-4 by G. F. Sprague (G. F. Sprague and H. H. McKinney, Genetics 54:1287, 1967) and further characterized by M. K. Brakke, R. G. Samson, and W. A. Compton (Genetics 99:481, 1981) and P. Friedemann and P. A. Peterson (Mol. Gen. Genet. 187:19, 1982).

The cDNA clones homologous to BSMV RNA sequences were constructed by standard procedures (U. Wienand et al., Nucl. Acid Res. 6:2707, 1979) and characterized by hybrid released-translation and Northern hybridization. Two non cross-hybridizing cDNA clones homologous to BSMV RNA component I (720 and 520 base pairs in length) were identified, as well as clones homologous to RNA component II (1150 base pairs) and RNA component III (480 base pairs). No clone homologous to RNA component IV could be identified.

In a Southern experiment these clones were used to probe maize wild type DNA (a color converted W22 line) and the two mutant DNAs described above. The EcoRI digested DNAs were separated on 0.8% agarose gels, transferred to nitrocellulose and probed with each of the four nick translated cDNA clones. Although hybridization conditions were used to detect single gene sequences, no hybridization could be seen.

Since the cDNA clones represented only part of the viral genome (the size of the RNA component varies between 2200 and 2800 nucleotides), total four-component BSMV RNA was also used as a radioactive probe for hybridization. Three bands could be seen in all the maize lines tested after hybridization of the ³²P labeled RNA to the EcoRI digested DNA. Two of these could be competed with rRNA from maize endosperm. Using total barley RNA as competitor in the hybridization experiment, no bands could be detected any longer. Thus the hybridization signals seemed to be due to contamination of plant RNA in the virus preparation.

From both DNA and RNA hybridization experiments we conclude that there are no viral sequences present in the mutant maize stocks tested.

Udo Wienand, Peter Peterson and Heinz Saedler

The Cin repeat of *Zea mays* and *Zea mexicana*

Cin 1 is a dispersed repetitive DNA element in both *Zea mays* and *Zea mexicana* (teosinte). The element is approximately 700 bp in size and was first recognized due to its presence on a 5.7 kb EcoRI fragment of a Northern Flint maize line and its absence on the homologous 5 kb, unique fragment in the midwestern maize, Line C (Wienand et al., 1982, Mol. Gen. Genet. 187:195-201). Cloning of these maize fragments (Shepherd et al., 1982, Mol. Gen. Genet. 188:266-271) and subsequent DNA sequence analysis of the ends of the Cin 1 element show a short inverted repeat structure with the terminal 5 bases being identical to that of the Copia transposable element of *Drosophila* (5' TGTTG 3') (Levis et al., 1980, Cell 21:581). DNA sequence analysis of the "target" site in the 5 kb recombinant clone from Line C does not reveal a duplication of target site sequences immediately flanking the element.

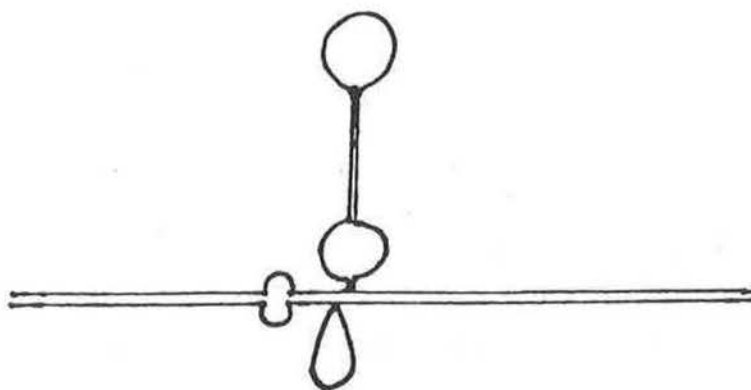
In order to analyze the conservation of the element and to confirm the lack of a target site duplication, several other Cin containing clones were isolated from the *Zea mays* Line C genome and from *Zea mexicana*, Teosinte Guerrero. The *Zea mays* recombinant clones contain maize fragments ranging from 2.5 - 10 kb.

All clones were found to contain Cin surrounded by repetitive DNA sequences. Thus, it has not yet been possible to screen other maize lines for the absence of the Cin element at these various loci in order to obtain target site information. However, the DNA sequence of one Cin containing clone, LC102, does show a 5 bp direct repeat in the sequence immediately flanking the element. Comparison of the Cin 102 and Cin1 sequences shows 87% conservation of the element. Although sequence analysis of the other clones is still in progress, it is clear that the Cin element has diverged significantly and represents more of a repetitive DNA family rather than a highly conserved element.

N. S. Shepherd, M. Gupta, Zs. Schwarz-Sommer, U. Wienand,
B. Deumling and H. Saedler

The Teo 1 DNA insert of Teosinte Guerrero

In a Southern hybridization experiment, the 5 kb maize EcoRI fragment, LC1, seems to be unique in the *Zea mays* Line C genome (Shepherd et al., 1982, Mol. Gen. Genet. 188:266-271). However, in a Northern Flint maize line the homologous fragment was found to contain the repetitive DNA insert Cin 1 (see previous article). Using LC1 as a radioactive probe in Southern hybridization experiments, the genomic fragment homologous to LC1 is observed to vary in size between various *Zea mays* lines and also in *Zea mexicana* (Teosinte). For example, both the 5 kb LC1 fragment and an 8 kb fragment are seen in Teosinte Guerrero. The 8 kb fragment, TG2, was cloned into lambda-gtWES (manuscript in preparation). A heteroduplex between TG2 and the maize LC1 or NF1 fragment clearly shows that the increase in molecular weight is due to a 3 kb insert having a very unique structure (see Figure). This insert is called Teo 1. It seems to occur



Schematic drawing of a TG2 and NF1 heteroduplex.

approximately 50 bp from the original site of the Cin 1 insert and does not contain sequences homologous to Cin1. DNA sequence analysis of Teo 1 is currently in progress.

N. S. Shepherd, B. Deumling, U. Wienand, J. Blumberg and H. Saedler

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nec4, a new necrotic mutant on 2S

In the search for recessive alleles at the *Les* loci, necrotic mutants which are in Neuffer's M2 collection and placed on chromosome arms containing *Les* loci are being mapped. One such mutant, *nec*-516B*, was located on the short arm of chromosome 2, which also contains *Les1*. This mutant germinates into a luteus seedling which rapidly becomes necrotic starting at the tip of the leaf. Seedlings never mature past the 2-3 leaf stage. Backcross linkage data from the cross of $++ \text{ nec}^*-516B \text{ b/lg g12} + \text{ b} \times \text{lg g12} + \text{ b}$ gave recombinational values of $\text{lg} - 22 - \text{g12} - 4 - \text{nec}^*-516B - 22 - \text{B}$ (94 plants). With the low number of progeny tested, it is impossible to rule out that *nec*-516B* could be 4 units distal to *g12* (between *lg* and *g12*). Further tests with *lg g12 d5 B* stocks are

underway. It is proposed that nec*-516B be designated nec4 in keeping with the accepted nomenclature.

David Hoisington and M. G. Neuffer

Sectorial loss of Les1

The role of the Les1 gene in the production of discrete necrotic lesions in leaves is being investigated. One question which is being asked is whether the Les1 gene is required in all stages of lesion formation. It is possible that the Les1 gene only triggers a cellular event which results in the production of cytotoxic compound(s). These compounds then diffuse to neighboring cells resulting in the observed lesion phenotype. Thus, Les1 could be required only for the initiation of lesions. Another possibility is that Les1 is also required for the enlargement of lesions. In this case, cells not containing the Les1 gene could not respond to a Les1 initiated lesion.

One method to study this question is the use of X-ray induced sectors of normal tissue in Les1 leaves. Les1 is located on the short arm of chromosome 2, approximately 5 map units proximal to B. It is therefore possible to use B to follow the loss of Les1 in leaf tissue. Seeds from the cross B Les1/b +, P1 x b +/b +, P1 were treated with 10,000 r of filtered X-rays in the dry seed state. Plants were observed for sectorial losses of B (green sectors in a purple leaf) and, hopefully, concomitant loss of Les1. None of the 173 plants grown in Missouri showed a loss of B or apparent sectoring for Les1. However, one out of 100 plants grown at Stanford University in California showed a single sectorial loss of B and, presumably, Les1. The sector occurred in the second leaf from the top and was approximately 1/10 of the width of the leaf. The sector occurred only in this one leaf and over the entire length of the leaf. No lesions were observed to initiate within the green tissue of the sector, nor did lesions that initiated just outside of the sector's border extend within the green tissue. The rest of the leaf expressed lesions in the normal manner. Thus, if this sector is the result of the loss of B and Les1, then the Les1 gene is a cell autonomous trait for all aspects of lesion expression--requiring its presence for initiation of lesions as well as enlargement to form mature lesions. This experiment is being repeated next summer in order to observe further sectors. Also, in order to maintain plants which could form sectorial losses of these genes as well as produce a larger number of sectors in one plant, the production of a ring chromosome which contains B and Les1 is being attempted.

David Hoisington, Virginia Walbot and M. G. Neuffer

Linkage calculation from hypoploids: transmission of the 10-B element

A few years ago (J. Hered. 69:27-36, 1978) the author published a formula (p. 31) for calculating the number of crossovers between a B-A translocation breakpoint and a proximal recessive gene, using F2 data from hypoploid plants. Although it was realized that movement of normal and A-B chromosomes to the same pole would give spurious evidence of a crossover event, such events were believed to be too rare to affect calculations. Occasional cases of nondisjunction of short chromosomes derived from B-A translocations recently prompted a reexamination of the normal kernels used to make the calculations of crossover frequency reported in MNL 47:145-147.

Plump kernels from wx wx du/+ hypoploids from TB-10La and TB-10Lb were planted in sand and root tip counts from all resulting seedlings made. The 10-year-old seed germinated poorly. All four seedlings from self-pollinated hypoploids of

TB-10La had 20 chromosomes, as expected, but two of the four seedlings from TB-10Lb hypoploids had 21 chromosomes. Although the 10-B chromosome was not actually observed, the 21-chromosome plants were probably partial trisomics (10 10 10-B) resulting from transmission of 10 10-B gametes.

Although the data are preliminary, it is likely that small chromosomal elements occasionally nondisjoin at meiosis. The breakpoint of TB-10Lb is probably less than one-third of the distance from the centromere to the end of 10L. The 10-B chromosome thus consists of the tiny short arm of 10 and less than one-third of the long arm, plus a portion of the long arm of the B chromosome. Presumably the short 10-B sometimes fails to pair with the normal 10 and the resulting univalents either move at random to the poles or remain on the equatorial plate and are lost. Consequently, when short chromosomal elements are involved, the formula in question will overestimate the crossover rate.

J. B. Beckett

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Temperature-dependence of the heat shock response

The heat shock phenomenon described in a variety of animal and plant systems, including corn (see Can. J. Biochem. 60:569-579, 1982), has generally involved rapid shifting of the tissue from the "normal" growing temperature to an elevated incubation temperature for brief periods of time. Seedlings of Oh43 were grown at several temperatures ranging from 27 to 37 C and were subjected to a series of shift regimes: (1) to ascertain the influence of pre-shift temperature (including some temperatures at which the heat shock response is observed, e.g. 37 C) on the types of polypeptides synthesized, and (b) to estimate the minimum temperature shift increments required to elicit new and/or enhanced synthesis of the heat shock peptides (HSPs). The following observations have emerged: (1) Seedlings grown at different temperatures exhibit very similar polypeptide patterns but also reveal some temperature-specific differences. (2) The HSPs are not produced as a result of exceeding a critical absolute temperature. While seedlings grown at 27 C first exhibit enhanced synthesis of the HSPs at 35 C, seedlings grown at 32 C and shifted to 35 C, or those grown directly at 35 C, do not synthesize these HSPs. (3) Rapid, upward temperature shifts from any of the growing temperatures result in new and/or enhanced synthesis of the HSPs described previously. The intensity of the response is dependent on the temperature range over which the seedlings are shifted, as well as the actual number of degrees. For example, a 6 C shift from 35 to 41 C yields a response similar to a 10 C shift from 27 to 37 C, and a 9 C shift from 35 to 44 C elicits the same response as a 15 C shift from 27 to 42 C. In all cases, enhanced synthesis of the same six M_r classes of polypeptides is observed.

These results suggest that the change in polypeptide synthetic patterns is qualitatively similar in response to a variety of temperature shifts, but that the intensity of the response is dependent on the actual temperature range and increment over which the seedlings are shifted.

C. L. Baszczynski

Polypeptide synthesis following upward and downward temperature shifts

The response of many animal and plant systems to heat shock has been examined almost exclusively at the upper temperature ranges. Maize grows normally over a broad temperature range (exceeding 15 C - 35 C) and offers the opportunity to

investigate the response not only to heat shock, but to a range of upward or downward temperature shifts which may still be within the "normal" growing range. We have examined a series of temperature shift regimes to ascertain whether heat shock polypeptides (HSPs) were specifically produced following a "heat shock" (40-45 C for Oh43 seedlings grown at 27 C) or whether they represented a general synthetic response to any upward shift in the culture temperature.

Seedlings of Oh43 were grown at 17 C and at 27 C until plumules were 1 to 2 cm long, at which time they were subjected to a 10 C increase in the incubation temperature for one hour. One- and two-dimensional PAGE separations and fluorographic analysis revealed that, while each temperature regime resulted in synthesis of some unique polypeptides, both 10 C shifts yielded enhanced synthesis of the same six Mr classes of HSPs. However, fewer isoelectric variants of these HSPs were noted in the 17 to 27 C shifts.

We subsequently investigated the effects of prolonged exposures to a shift temperature to determine if these conditions led to continued HSP synthesis, or to the establishment of a stable, temperature-specific polypeptide synthetic pattern (i.e., acclimatization). Seedlings grown as before were subjected to the same 10 C upward shift, but for 3, 12 or 24 hours (which included a two-hour labelling period). In both the 17 to 27 C and in the 27 to 37 C shifts, a 3-hour incubation resulted in new and/or enhanced synthesis of the six Mr classes of polypeptide described previously. By 12 hours at the shift temperature, very little synthesis of these polypeptides was evident, and by 24 hours, the synthetic patterns were similar, but not identical to the control patterns. These observations suggest that these HSPs may represent transition proteins synthesized during the rapid transfer from one incubation temperature to another. In experiments where the seedlings were shifted down from 27 to 17 C for 3, 12 or 24 hours, no changes were detected in one-dimensional electropherograms, suggesting that this enhanced synthesis is specific to upward temperature shifts.

To further investigate the possibility of an "acclimatization" in polypeptide synthetic pattern, seedlings grown at 27 C were shifted down to 21, 18 or 15 C for 6, 12, 24 and 48 hours, and then returned to 27 C, where plumules were labelled for two hours prior to extraction. Results indicate that return to the control temperature following a downward temperature shift leads to a time- and temperature-dependent enhancement of HSP synthesis. For example, 24 hours at 21 C are required before a response is observed, while synthesis of this same group of polypeptides is noted after only 6 hours at 18 C or 15 C. Thus, the greater the interval over which the seedlings are shifted, the shorter the time required at the shift temperature before response is noted when seedlings are returned to the control temperature, suggesting that the seedling is responding to cumulative 'heat units,' as distinct from changes in temperature.

It appears, therefore, that prolonged exposure to an increased or decreased incubation temperature leads to acclimatization as monitored by changes in polypeptide synthetic patterns. The observations in this report and in the previous one, that new and/or enhanced polypeptide synthesis occurs following a variety of temperature shifts and not only in response to high temperature shifts, suggests that it may be more appropriate to refer to these polypeptides as temperature shift polypeptides or proteins (TSPs) of which the HSPs would be a specific subclass induced during high temperature stress.

C. L. Baszczynski, J. G. Boothe, B. G. Atkinson and D. B. Walden

Analysis of the in vitro translation products from RNAs of heat-shocked seedlings

We have reported previously (MGCNL 56:111-113, 1982; Can. J. Biochem. 60:569-579, 1982) that various tissues exhibit a rapid and reversible response to a brief temperature shift (heat shock) with the new and/or enhanced synthesis of a

select set of heat shock polypeptides (HSPs), as detected by one- or two-dimensional PAGE separations and fluorographic analysis. Preliminary results from *in vitro* translations of total isolated RNA from 4- to 6-day-old heat-shocked seedlings indicated that the HSPs noted *in vivo* were also present among the *in vitro* translation products. Subsequent investigations have revealed that the majority of the products obtained following *in vitro* translation of total RNA from control or heat-shocked plumules are the same as those obtained from translation of poly(A)⁺ mRNAs purified by oligo(dT) cellulose chromatography. Similar results were obtained from translations in both the rabbit reticulocyte and the wheat germ extract *in vitro* translation systems, with the former being apparently more efficient in the extent of incorporation of labelled amino acid precursor into newly synthesized products. We have also noted that, while the high molecular weight HSPs from the *in vivo* lysates each resolve (by 2D-PAGE) into a number of polypeptides with different pIs, the HSPs with similar M_rs synthesized *in vitro* have fewer isoelectric variants. Equally striking is the observation that the 76,000 dalton HSP noted *in vivo* is absent among the *in vitro* translation products in either the heterologous rabbit reticulocyte, or in the more homologous wheat germ *in vitro* translation systems. This may be the result of a lack of stability of the mRNA for this polypeptide, or may indicate the requirement of "maize-specific" translational factors for its synthesis.

Our observations indicate that: (a) heat shock in maize induces new and/or enhanced translation of polypeptides (HSPs); (b) the heat shock polypeptides are synthesized from polyadenylated mRNAs which were not available for translation in control tissues, and (c) several levels of regulation (currently under investigation) appear to be operative in determining the final form(s) of these HSPs.

C. L. Baszczyński, D. B. Walden and B. G. Atkinson

Gene products in embryos and seedlings of certain inbreds and their hybrids

Fluorographic analysis of 2-D PAGE electropherograms coupled with the specific temperature-shift patterns (preceding contributions, this Newsletter) of known genotypes provides the technology for addressing a number of developmental and genetic problems. We currently are examining embryogenesis, seedling germination and flower development (see next contribution) employing inbreds and their reciprocal hybrids. We report the appearance of developmental shifts in the patterns of newly synthesized polypeptides obtained from maize embryos. The procedures adopted for this study involved the dissection under laboratory conditions of embryos resulting from controlled pollinations. The embryos were transferred to Murashige and Skoog medium and incubated at 27 C for 2 hrs in the presence of an isotopic probe (usually ³⁵S-methionine). The subsequent protein extraction and electrophoretic techniques were identical to those reported in the preceding contributions to this Newsletter.

Results obtained for the incorporation of the labelled probe indicated that material 23 days post-pollination or older provided sufficient quantities of labelled protein in a single embryo to achieve a concentration of at least 200 cpm/ul in a homogenization volume of 150 ul. Material 27 days of age or older enabled a concentration of 500 cpm/ul to be achieved in the same volume. These concentrations were suitable for 1D and 2D fluorographic analysis, respectively. In each case, embryos younger than these required bulking of material to achieve the desired concentrations. Of the material from which sufficient quantities were obtained (17-31 days post-pollination), 2D fluorographic analysis routinely resolved over 100 peptides. These were classified into one of three categories: (1) those where the relative level of synthesis appeared unaltered throughout the period of study; (2) those that showed variations in the level of synthesis; (3) those where synthesis appeared restricted

to particular time periods. Thus far we have been unsuccessful in obtaining sufficient material at such an early age as to recover only a few labelled spots in the 2-D fluorograms.

There appears to be a genetical component in addition to the developmental component, i.e., some inbreds differ from one another and some of the hybrids we have examined differ from their parental inbreds. We draw attention, however, to the inconclusiveness of these observations in the case of the embryos--until we can utilize an independent criterion, better than age and/or heat unit accumulation, we are reluctant to assign differences to the pedigree and ignore environmental variability.

In addition to these *in vivo* studies, total RNA derived from embryos of dried seeds was extracted and translated in a rabbit reticulocyte lysate system. The appearance of labelled bands on a 1D gel of the translation assay demonstrates the *in vitro* synthesis of proteins from this RNA, and thus the presence of stored mRNA in maize embryos. The time at which these messages were synthesized remains to be determined as does their ability to direct translation *in vivo* upon the onset of germination.

It is anticipated that these investigations will lead to a better understanding of the factors governing the regulation of gene expression in hybrids and their parental inbreds during early development.

J. Boothe, B. G. Atkinson and D. B. Walden

In vivo labelling of excised tassel florets from greenhouse-grown material and material grown in tissue culture

Individual florets or spikelets (S60) were excised from tassels grown in tissue culture (see Polowick MGCNL 55:116, 1981) or from greenhouse-grown material, and labelled with ³⁵S-methionine for 2, 3 or 4 hours. The florets from the greenhouse material had immature pollen in the larger anthers and quartets in the smaller anthers. Florets from the cultured material contained anthers with either pollen mother cells or quartets. Results show that incorporation of label increased by a factor of approximately 6, from the 2 to 4 hr labelling period, for both the greenhouse material and the cultured material. Total incorporation after 4 hrs of labelling was higher in the cultured material than in the greenhouse material. Average values for each were 12,000 ± 3,000 counts/ug protein for the greenhouse material and 32,000 ± 22,000 counts/ug protein for the cultured material. Comparison of the one dimensional SDS PAGE patterns for extracted proteins from greenhouse grown material indicates similar patterns for the same stage/tissue labelled over the three time periods. The cultured material showed a difference in banding pattern from the greenhouse material, and a comparison of banding patterns from different developmental stages of the greenhouse tissue also showed differences.

M. J. Dunlop, R. I. Greyson, D. B. Walden and B. G. Atkinson

Production of plants from tassels grown in tissue culture

Immature tassels (approximately 1 cm) were excised and grown in liquid media as described previously (Polowick, Raman and Greyson, MGCNL 55:116, 1981). Some tassels, if allowed to grow in culture for extended periods of time (longer than 3 weeks), produce plantlets which can be rescued and grown to sexual maturity. Plantlets have been produced in tissue culture from tassels of three cultivars: Oh43 heterozygous tunicate (Tu/+), Oh43 heterozygous waxy (Wx/wx) and S60. The success of plantlet initiation appears to be a function of the duration of culture, the size of the original explant, the genotype and other factors not yet identified. To date 60 plantlets have been isolated from the tassel culture system,

some of which have been transferred to a rooting medium (Raman, Walden and Greyson, *Ann. Bot.* 45:183-189, 1980); 16 others, which produced roots while in the original culture, have been potted directly and transferred to the greenhouse. Of the latter, two plantlets have been brought to flowering, producing both pollen and ear silks. The amount of pollen produced and the absence of aborted pollen grains suggests that these plants are diploid. The morphological origin of these plantlets appears to be vegetative proliferation of the floral tissue with initial leaf formation, followed by subsequent root development. These observations, although preliminary, suggest that the proliferation of plantlets from cultured tassels could eventually be a useful cloning technique.

M. J. Dunlop, R. I. Greyson and L. Olson

The position of the nucleolus in each microspore of the quartet

The distribution of specific chromosomes in the nucleus has been of interest to us for some time. Reports of our work with maize have appeared in this Newsletter and elsewhere over the last 15 years. Of several major problems attendant to this work, such as preparation of the material, choice and interpretation of statistical analyses, and reconstruction from serial sections, none has been more limiting than the availability of unambiguous cytological markers. One such marker we have utilized is the nucleolus. Since it is difficult to establish the position of one large nucleolus, or occasionally two smaller nucleoli in somatic interphase cells, we have attempted to analyze the position of nucleoli in recently formed microspore quartets. The choice of quartets has included only those that are intact and within which the limits of the microspore nucleus can be reasonably outlined. The nucleolus stains rapidly with carmine and other stains. Little or no squashing (other than the weight of the coverslip) was utilized.

Photomicrographs of quartets in squash preparations were taken and traced onto acetate sheets. The size of the quartets was standardized and the centroids were aligned. A composite tracing of twenty-five quartets was obtained. The maximum area covered by the nucleolus in the composite tracing was only one-eighth of the maximum area covered by the microspore. The position of the nucleoli was analyzed using four concentric circles drawn about the centroid of the quartet: circle 1 of equal radius to a circle circumscribed about the quartet tracing, circle 2 of radius three-quarters that of circle 1, circle 3 of radius one-half that of circle 1 and circle 4 of radius one-quarter that of circle 1. Eighty-nine percent of the nucleoli were found within the band encompassed by circles 2 and 3. The remaining eleven percent were found within the band encompassed by circles 4 and 3. No nucleoli were found external to circle 2. The location of the nucleolus was also examined using a circle of radius equal to that of circle 4 and divided into twelve sections, each making an angle of 30 degrees to the center. Ninety-three percent of the nucleoli were found in the sections 30 degrees to 60 degrees from the X axis.

Based on these analyses it would appear that the nucleolus does not have a random position inside the microspore in quartets in squash preparations. It is found in an area one-eighth the size of the microspore, in the central region of the cell. In the photomicrographs of quartets used, the extent of the nucleus in each microspore could be drawn as the area of the microspore in which chromatin was found. The area of the nucleus was estimated as one-quarter of the area of the microspore. In 82 percent of the microspores the nucleolus was seen at the perimeter of the nucleus.

C. A. Rees-Farrell and D. B. Walden

Dup/df male gametophytes

The maturation of pollen and its ability to function (germinate, produce a tube and penetrate the micropyle) are capable of being altered by environmental and genetical factors. For some purposes, in vitro germination studies may reveal aspects of this pollen plasticity. Studies proving, rather than just correlating, in vitro germination and the ability to complete fertilization are rare, although it has been demonstrated that an in vitro germinated male gametophyte may be recovered in an F1 zygote.

Earl Patterson and I have undertaken a study of the ability to function of adjacent segregants from a series of translocation heteromorphs. As part of that study, we have looked at the in vitro germination of certain classes of pollen grains.

Alternate segregants produce full, plump, functional pollen grains. Adjacent segregants are apparently less full, sometimes smaller, pollen grains. The adjacent segregants appear translocation-specific, indicating that their phenotype is a product of their genotype, in particular the duplication and/or deficient segment. Since many translocations in maize produce specific adjacent phenotypes in the pollen, there is the suggestion that loci for the 'biology' of the male gametophyte are distributed about the complement, at least in the interchange segments.

We have reported earlier that it is possible to utilize the size differences in the pollen from a translocation heteromorph by passing the pollen through a nest of sieves of decreasing pore size. Substantial populations of pollen may be obtained on some or all of the sieves.

Consistent features of pollen studied from field grown (1979, 1980, 1981, 1982) translocations (T5S.64-6S.89; T4-6 sat) include:

1. Pollen capable of germination was consistently recovered on sieves of pore size 125, 105, 90, 75 and 63 μ . Pollen collected in the AM was more likely to 'clump,' rendering separation difficult, than pollen collected after 1200 h.
2. The percent germination was consistently highest for pollen collected on the 90 μ sieve. The rank was 90, 105, 125, 75 and 63 with the differences usually significant (5%), except for the 90 and 105 μ pollen.
3. The percent germination for pollen from the 90 μ sieve averaged nearly 75% over the years; pollen from the 63 μ sieve averaged 8%.
4. Very little additional germination occurred among the larger sized samples after 60 minutes. Additional germination of the smaller, including the 63 μ pollen, continued for at least 150 minutes.
5. The rate of elongation of pollen tubes from the 90 μ sieve decreased from over 5 μ /min during the first hour to less than 1 μ /min after 90 minutes. This latter rate was maintained for several hours. In general, the smaller pollen grains had slower rates of growth.

Most of these data were recorded from films, permitting the measurement of the diameter of the pollen grain. None of the populations (each sieve type) was entirely homogenous. Larger than expected pollen grains apparently were somewhat shrivelled when sieved and expanded with uptake of water from the medium. Smaller than expected grains did not proceed through the nest of sieves in accordance with their size, for a variety of reasons. However, none of these 'off-type' grains were entered into the data.

Thus, it would appear from in vitro studies that adjacent segregants fail to produce progeny most of the time because either they are actually sterile and do not become a functional gametophyte, or as a functional gametophyte they cannot compete well (i.e., a lag in germination and/or a slower rate of elongation) with alternate segregants.

Patterson and I have some data on the progeny produced by samples of pollen from the same translocations, sieved and used in sparse pollinations. We have examined several hundred dup/df F1's, the frequency of which is inversely proportional to the size of the sieve pore on which the pollen was collected. Some samples of pollen, passed through the 74 u sieve and collected on the 63 u sieve, have produced as high as 75% dup/df (estimated by uncovering a known allele) plants.

The combined use of sieving pollen and sparse pollination may permit the male transmission of cytogenetic and genic variants heretofore considered as 'non-transmissible.'

D. B. Walden

ST. PAUL, MINNESOTA
University of Minnesota

Tests of Hooker-Russell exotic sources of cytoplasm converted to inbred H632 for a cytoplasmic fertility restorer

The six lines not previously tested (MNL 56) do not carry a cytoplasmic restorer of genetic male-sterile #1. This completes the test of 40 of those lines. One line (PI 174990 x B14¹⁶ x A632¹⁴) has not been tested. I have been unable to establish the stock.

If a cytoplasmic restorer is specific for a particular male-sterile gene, it will be necessary to test the other male-sterile genes.

Chas. R. Burnham

Another source of cytoplasmic male sterility

One of the Hooker-Russell lines (PI16164375 x B14¹⁶ x A632¹⁴), as mentioned in last year's newsletter, has only male sterile plants. This is another source of cytoplasmic male sterility. Crosses of male sterile plants with a line that restores fertility to the Texas cytoplasmic male sterile produce completely fertile plants.

Chas. R. Burnham

Tests of other lines for a cytoplasmic restorer of ms

Further tests of the derived A188 and the T6-9b sources show that neither is a cytoplasmic restorer of genetic ms1. Crosses made to test the cytoplasm of diploperennis will be grown in 1983.

I am now using the following method for testing. Step 1: The stock to be tested is crossed as the female parent with a known ms heterozygote. Step 2: The resulting F1 plants (5 to 10 plants) are crossed as female parents with the ms heterozygote and also crossed as the pollen parent on ms plants as females. If the stock being tested has the cytoplasmic restorer, none of the progeny in step 2 from the cross of the stock as female with ms heterozygotes will segregate male sterility. To be certain of the results, these progeny can be crossed again with ms heterozygotes. If there is no segregation for male sterility, many of the fertilized plants will be [R] ms ms. These are identified by the crosses on ms plants.

Chas. R. Burnham

A balanced lethal stock (repulsion) for chromosome 6, and a derived stock (coupling)

These stocks involve w15 (w*-8896) and l10, closely linked with each other and with Y vs. y. The original stocks obtained from Robertson were from selfs of Y +/y l10 and Y +/y w15. Crosses were made of the green progeny from one stock with those from the other. Ears from crosses that were between Y +/y l10 and Y +/y w15 were segregating for yellow and white endosperm. Most of the plants from the white seeds from that cross were heterozygous for w15 and for l10 in repulsion. These plants were selfed and also crossed on a Y Y normal stock. The progeny were grown in a somewhat isolated block and allowed to open pollinate, except for 54 which were self-pollinated. The results of the seedling tests for 299 ears are as follows:

	<u>All green</u>	<u>seg l10</u>	<u>seg w15</u>	<u>seg l10, w15</u>	<u>Total</u>
42201 selfed	0	19	34	1	54
42201 open poll.	2	74	69	2	147
42202 open poll.	<u>1</u>	<u>45</u>	<u>51</u>	<u>1</u>	<u>98</u>
TOTALS	3	138	154	4	299

All ears were segregating for yellow vs. white endosperm. There were 7 recombinants between l10 and w15, 2.34% recombination. In an earlier report (Maize Newsletter 51, 1977, p. 55), on a similar experiment using self-pollination, there were 158 that segregated l10, 155 that segregated w15, nine that had only green seedlings and three that segregated l10 and w15, a recombination value of 3.1%. Many more ears remain to be tested. There are four ears from coupling heterozygotes already identified and available for further use: one from this year's self-pollination and three from the open-pollination plot.

The gametic frequencies for a plant heterozygous for w15 and l10 are: $p/2 ++$, $(1-p)/2 + w15$, $(1-p)/2 l10 +$, and $p/2 l10 w15$, where p = recombination in repulsion. The frequencies of the three possible F2 phenotypic classes are: $(2+p^2)/4$ green, $(1-p^2)/4$ yellow, and $1/4$ white. Plants that are l10 l10 w15 w15 are white.

To calculate the expected frequencies in repulsion (R), substitute for p the recombination percentage for the numbers in coupling (C), substitute $1-p$ for p in the expressions.

The figures for a few p values are as follows:

<u>Recomb.</u>	<u>linkage phase</u>	<u>green</u>	<u>yellow</u>	<u>white</u>
0.0	R	.50	.25	.25
0.0	C	.75	--	.25
.01	R	.50025	.249975	.25
.01	C	.745025	.004975	.25
.03	R	.5002255	.249775	.25
.03	C	.735225	.014775	.25
.20	R	.51	.24	.25
.20	C	.66	.09	.25
.50	R	.5625	.1875	.25
.50	C	.5625	.1875	.25

Note that in F2 for repulsion 50% of the chlorophyll deficient plants are yellow at 0% recombination and 42.9% are yellow at 50% recombination. In F2 from

coupling when recombination is zero, there are no yellow seedlings. At the observed 3% recombination the ratio of yellow:white seedlings is about 1:17. To have only a .01 chance of failing to obtain at least one yellow seedling, 81 plants from the white seeds would have to be grown. If $\underline{Y} \underline{y}$ is not used as a marker, over 300 would need to be grown.

In this year's plot, comprised of plants from the repulsion heterozygote ($\underline{y} + \underline{w15}/\underline{y} \underline{l10} +$) crossed on $\underline{Y} \underline{Y}$; at 3% recombination, about 1.5% of the plants would be coupling heterozygotes, 1.5% would be heterozygous for neither $\underline{l10}$ or $\underline{w15}$, and the remainder would be either $\underline{l10}$ or $\underline{w15}$ heterozygotes in equal numbers. Of the green survivors from selfs of the coupling heterozygotes, about 64% of the green plants will be coupling heterozygotes, 32% homozygous green, 4% heterozygotes for $\underline{w15}$ or $\underline{l10}$. In the open pollination plot, the pollen mixture will be about $2 \underline{Y} + +:1 \underline{y} + \underline{w15}:1 \underline{y} \underline{l10} +$. For the white seeds from open-pollinated coupling heterozygotes the ratio of yellow:white seedlings will be about 1:1. For all seedlings about 1/8 will be yellow. Most of the green plants from white seeds will be repulsion heterozygotes; a few will be from crossovers between \underline{Y} vs. \underline{y} and $\underline{l10}$ or $\underline{w15}$. About 38% of the green plants from the yellow seeds will be coupling heterozygotes. Probably the best way to obtain and maintain demonstration ears that will give these higher numbers of yellow seedlings is to grow the progeny from those open pollinated ears, and pollinate the green survivors with a mixture of pollen from progeny of $\underline{Y} \underline{Y} \times \underline{y} + \underline{w15}/\underline{y} \underline{l10} +$.

These stocks should be useful for class use in genetics to illustrate:

1. Close linkage: $\underline{Y} \underline{y}$ vs. $\underline{l10}$ or vs. $\underline{w15}$.
2. Balanced lethal, a repulsion heterozygote: the surviving green plants are usually heterozygotes.
3. The coupling and the repulsion heterozygotes illustrate the explanation for the true breeding of heterozygotes in many wild races of *Oenothera*, also the rare occurrence of types that were believed at first to be mutations.
4. The repulsion heterozygotes are useful for making crosses for linkage studies, for mapping these lethal traits.
5. Open pollination plots using this material might be useful for population theory involving closely linked factors.

Chas. R. Burnham

A stock useful for demonstrating 3-point linkage

A lazy sugary glossy-4 stock with about 10% recombination in region 1 and 15% in region 2 has been established. Ears from backcrossing F1 heterozygotes with the triple recessive can be classified first for $\underline{Su} \underline{su}$ endosperms. If these are planted in pots, the seedlings can be classified first for glossy, then for lazy by laying the pots on their sides (after about 2 weeks). Normal plants will turn upward, lazy ones do not. Seeds of the multiple recessive stock and backcrosses are available and will also be sent to the Coop.

Chas. R. Burnham

Studies of colchicine- and colcemid-induced fertility in multiple interchange heterozygotes in corn and their F2 and F3

In Newsletter #56, pp. 151-153, one of the partially fertile plants obtained from colcemid-treated seedlings of F1 multiple interchange heterozygotes was reported to have 38 plump seeds. These were from cross A (T3-2-4-9-10 x T1-5-6-7-8). Five of these seeds were planted in the greenhouse in 1982. Three plants were fertile with pollen similar in size to that in normal diploid corn.

The other two were similar to their F1 parent, i.e., partially fertile with some 2n-size and some 1n-size pollen. The five plants were selfed and the three fertile ones were testcrossed on normal diploids. Self and testcross progenies were grown in the field in 1982, and sporocytes were collected. Test cross progenies of two of the fertile plants had a ring of ten chromosomes each at diakinesis, neither involving chromosome 6. These, probably, represent one of the original parental combinations. The testcross of the other fertile plant had a ring of four, also not involving chromosome 6. This must be the result of a crossover in one of the differential segments of one of the parental interchanges. The three fertile F2 plants were evidently diploids. Hence some of the pollen produced by the partially fertile F1 parent must have been 1n. Pollen classes b and c in last year's report are probably this type.

All but a few of the remaining F2 seeds and the F3 from selfing the two partially fertile greenhouse plants were planted in the field. Certain plants were fertile with normal-size pollen; others were partially fertile with some large-size pollen as well as normal-size pollen. The degree of fertility in the latter group varied somewhat from plant to plant.

The fertile plants were selfed and testcrossed on both diploid and tetraploid stocks. Crosses on the tetraploid produced only shriveled seeds (probably triploids). Excellent seed and seed set were obtained from the crosses on diploids.

Test crosses of the fertile plants will be grown for cytological examination. It is hoped that one or more of these will have two rings, each with ten chromosomes. This would indicate that the fertile parent was homozygous for the two original parental multiple translocations, T3-2-4-9-10 and T1-5-6-7-8.

Helmy Ghobrial

The use of colchicine and colcemid treatments to produce homozygotes for multiple interchanges involving all corn chromosomes

For this experiment F1 seeds from a cross between two interchange homozygous stocks, T5-7-1-9-10-8 and T6-3-2-4-8 were used. The seeds were placed on filter paper, in Petri dishes, and watered with 0.02% aqueous solution of colcemid or colchicine for 5 days (The colchicine concentration was erroneously reported as 0.2% instead of 0.02% in the 1981 Newsletter). The seedlings were placed in sand in the greenhouse for 10 days before transplanting them in the field. Most of the plants were highly sterile and did not shed pollen. However, there were 5 plants from the colcemid treatment that were partially fertile and shed pollen. No fertile plants were obtained in the colchicine treatment. A higher frequency of fertile plants was obtained in previous experiments when the seeds were presoaked in water 20-24 hours prior to treatment.

Microscope examination of pollen samples from the five plants that shed pollen indicated that all samples had starch-filled grains of large size as well as normal size. Four of these plants had tillers. Fertility of the tassels on the tillers was similar to that of their main stalks. Self seeds were obtained on the ear on the main stalk of the five plants and also on the ear on each of the four tillers. The events that gave rise to the fertility, probably chromosome doubling, must have occurred in the tassel, ear, and tiller primordia in the same plant. This is contrary to expectation, since in the mature seed the tassel and ear are each known to be represented by separate multiple-cell primordia. The probability of the same independent event occurring in the three primordia in the same seedling simultaneously is extremely small. Some other mechanism is more likely to account for these results.

Two possible explanations can be offered:

1. One chromosome doubling event in the meristematic tissue with the resulting cell subsequently taking over the other cell lines to form the vegetative portion of the plant.
2. One event, probably chromosome doubling, in a tiller primordium which is possibly represented by one cell in the germinating seed, with it subsequently forming the main stalk and possibly other tillers.

All five plants were testcrossed as male parents on diploid and tetraploid stocks. Crosses on the tetraploids resulted in only shriveled seeds, probably triploids. Crosses on the diploids resulted in normal ears with fully developed seed, and no shriveled kernels. These results are similar to those obtained for the partially fertile plants in the 1981 experiment (this Newsletter). These partially fertile plants appear to have viable diploid and haploid pollen in addition to the aborted class. The haploid pollen seems to have a competitive advantage over the diploid when applied on either diploid or tetraploid female parents.

The F2 progeny will be planted in the field this spring. Fertile plants with haploid-size pollen will be selfed and testcrossed with normal diploids as well as with the two parental translocations. F2 plants, whose testcross with the normal diploid shows an association of 20 chromosomes at diakinesis, must be homozygous for a multiple translocation involving all 20 chromosomes. This would be T5-7-1-9-10-8-4-2-3-6. Such an F2 plant would be produced by gametes resulting from a crossover in the differential segment of chromosome 8, the chromosome common to the two parental stocks. In T5-7-1-9-10-8 the breakpoint in 8 is in one chromosome arm, that in T6-3-2-4-8 in the opposite arm.

Fertile plants with diploid-size pollen would be either normal tetraploids or tetraploids homozygous for the multiple interchange involving all ten chromosomes. The latter combination would be expected to have similar fertility to that of normal tetraploid since it has ten tetrasomes (each tetrasome having four similar chromosomes). The two types can be separated by testcrossing to a normal tetraploid stock. Plants that give partially sterile testcross progeny would be the desired 4n multiple interchange homozygote.

Helmy Ghobrial

An alternative method of obtaining the multiple interchange stock to produce a ring of 10?

If the experiments by Ghobrial reported above prove to be a method of obtaining homozygotes for parental combinations in multiple interchange heterozygotes, there are several interesting applications. One is the following.

Test crosses have been made to establish a homozygous T6-3-2-4-8-10 stock, i.e., one with T8-10 added to the T6-3-2-4-8 that was used in the above experiment reported by Ghobrial. Since this T8-10 interchange is common to the 5-7-1-9-10-8 stock, F1's from the cross between the two will have two rings of 10. In these plants, barring crossing over, there are only three viable micro- and megaspore 1n combinations: one with one parental chromosome set, one with the other parental set, and the third one with all the interchange chromosomes, i.e., 5-7-1-9-10-8-4-2-3-6. This latter combination occurs without crossing over. The use of F1's from T5-7-1-9-10-8 x T6-3-2-4-8-10 for colchicine treatment should be worth a try.

A homozygote for all those interchanges might be used for gamete selection, but only if crossing over at meiosis is bypassed also.

Chas. R. Burnham

III. ZEALAND 1983

This is an extraction of new data reported in the current list of Recent Maize Publications (author, year) and in the Reports from Cooperators in this issue of the News Letter (author, 57:page). Genetic locations are given with recombination percentages when available; the source should be consulted for standard errors and other factors that affect the precision of the numbers. New alleles and new loci are also listed, as are, for the first time, a few genomic clones. Studies that contribute to the genetic structure of chloroplasts (ctDNA) and mitochondria (mtDNA) are cited without attempting to make a synthesis of the information; this is the first attempt to compile these reports. Finally, studies that contribute to definition of the inheritance of "resistance" and "tolerance" are cited, again for the first time. Comments, suggestions, corrections and ideas would be most welcome.

--Prof Ligate

Chromosome 1

Adh1 allele -2F11 contains a 2kb insert (Ds?) and displays Kn1 phenotype Hake, 57:11
Adh1 allele -S3034 contains a 1.35kb insert, designated Mu1; restriction sites for Freeling &, 57:13
 XbrI, Sau3A, AvaI, BstNI, PstI, SstII, AvaII, HindIII
Adh1 allele -S3034x1; locations of inserts relative to intron, in Mu-induced alleles Alleman &, 57:157
 -S3034, -S3034a, -S3034b
Adh1 cDNA, restriction map of Hinf, Sall, HindIII, Dde, Sau3A, TaqI sites; base sequence Gerlach &, 1982
 for 168 COOH-terminal aa's
Adh1 allele -Fm335, Ds-suppressed Osterman &, 1981
Adh1 alleles -S, -S3034, -S3034a, -S3034b, restriction maps for KpnI, BamHI, BstEII, SstI, Strommer &, 1982
XbaI, HindIII, BglII sites
Cat3 not on 1L; -null allele Goodman &, 57:129
hcf*-9 allelic to hcf*-3; hcf*-3 not allelic to hcf*-1 Leto, 1982
hcf*-2 uncovered by TB-1La "
hcf*-3 uncovered by TB-1Sb "
hcf*-12 uncovered by TB-1La "
hcf*-13 " "
hcf*-41 " "
Mu1 insert in Adh1; restriction sites for TthIII-1, TaqI, NotI, BglI, HaeIII, BstEII, Freeling &, 57:13
AvaI, AvaII, SstII, BstNI
Prot1 alleles -L, -I, -S Kriz &, 57:13

Chromosome 2

hcf*-1 uncovered by TB-1Sb-2L4464 but not by TB-1Sb Leto, 1982
hcf*-15 " " " ; not allelic to hcf*-1 or hcf*-3 "
lg1 - 22 - g12 - 4 - nec4 - 22 - B1; nec4 was nec*-5161B Hoisington &, 57:159
whp1 uncovered by TB-3La-2L7285 but not by TB-3La Modena, 57:39
ws3 - 8.8 - lg1 - 15.8 - Mut - 8.1 - G12 Rhoades &, 57:17
Ht1 alleles -A, -C, -D Raymundo &, 1982

Chromosome 3

al-ruq allele Friedemann &, 1982
a3 - 26 - sh* (sh2?) Styles, 57:150
Ac2 transposed from chromosome 8, (tr-Ac2): g16 - 22.8 - Ac2 - 8.6 - lg2 Rhoades &, 57:18
g2 alleles g5 and pg14 (pg*-m); uncovered by TB-3Sb Whalen, 57:20
Got1 alleles -2a, -2b Shumaker &, 1982
hcf*-19 uncovered by TB-3Sb Leto, 1982
hcf*-46 uncovered by TB-3La "

Chromosome 4

bx1 located by monosome-4 and TB-4Sa Simcox &, 57:107
c2 alleles -m826019, -m826021, -m826040, -m826134, -m826204, -826126, -826133, Peterson, 57:2
 -816149, -816154, -816155 from En-bearing sources
c2 putative genomic clone LC1 from W22 C1 R1 source, selected for homology to chalcone Wienand &, 1982
 synthase of Petroselinum, hybridizes to Spm-controlled c2-m-1 DNA EcoRI fragments
 and to revertants from this allele and to c2-m2 DNA and c2-m3 fragments, all of
 which are 800 bp larger than fragments from C2 source
cupule development and induction, teosinte vs. maize Galinat, 57:152
pointed kernels, teosinte vs. maize "
hcf*-23 uncovered by TB-4Sa Leto, 1982
zein, 22kd, map locations near f12, su1, g14 Soave &, 1982
zein, cDNA clone for 21-23kd chain hybridizes in situ to distal 1/3 of 4L, located Viotti &, 1982
 relative to T4-6(8764), T4-9g, T4-10b; two clones for 18-20kd chain hybridize
 to proximal 1/3 of 4L

Chromosome 5

Inv5P&G - paracentric inversion in 5L of Z. diploperennis x Z. mays Pasupuleti &, 1982
lyc1 (also called ly1) allelic to ps1, designated ps1-lyc Robertson &, 1982
o9 tentatively located in 5L by TB tests Fletcher, 57:150
zein mRNA, cDNA clone pcM4 hybridizes in situ to terminal 1/3 of 5L, located relative Viotti &, 1982
 to T5-9a, T5-9c

Chromosome 6

hcf*-26 uncovered by TB-6Sa
hcf*-34 uncovered by TB-6Lc
110 - 3 - w15
Ico1 (low crossover frequency in Y1 Dt2 interval); p11 assort independently
pol1 uncovered by Dp Df from T6-9(067-6)
ms4 allelic to pol1

Leto, 1982
 " "
 Burnham, 57:168
 Higgins, 57:41
 Phillips &, 57:132
 Golubovskaya &, 1981

Chromosome 7

o2 alleles -R, -m(r), -Charentes, -Italian, -Columbian, -Agroceres, -Crow
Px3 localized by trisome-7; Px3 - 15.4 - Pn1; s11 - 44.4 - Px3; 1p1 - 43.0 - Px3;
o2 - 56 - Px3; wx T7-9a - 41.8 - Px3
Px3 localized by monosome-7
y7 allelic to z1; vp9 redesignated y7; alleles y7-4889, y7-z, y7-Wisconsin#2
zein, 20kd and 14kd, map locations near o2 and de*-B30
zein, cDNA clones, two for 18-20kd chains, hybridize in situ to distal 1/3 of 7S,
 located relative to T7-9a and T7-9(4363)

Salamini, 1980
 Brewbaker, 57:49
 Weber &, 57:108
 Robertson &, 1982
 Soave &, 1982
 Viotti &, 1982

Chromosome 8

e11 uncovered by TB-8Lc
f13 - 1.6 - pro1
Idh1 distal to TB-8Lc breakpoint, Mdh1 not

Curtis, 57:32
 Nelson, 57:81
 Goodman &, 57:131

Chromosome 9

bz1 alleles -m826301, -m826302, -826357, -826361, -826440 from En-bearing sources
C1-w allele (weak)
Ds standard location C1 Wx1 Ds centromere
Inv9P&G terminal inversion in 9S of Z. diploperennis x Z. mays
sh1 allele -826466 from En-bearing sources
Sh1 isoallelic polymorphisms identified with BglII; allele sh1-t standard tester
 allele; mutant sh1-*
sh1 alleles -m5933, -m6233, genomic clones, relationships of breakpoints and inserted Ds
Sh1 genomic clone, restriction sites for HindIII, BglII, SphI, ClaI; 14 introns
Sh1 alleles -4864A, -5245, -6233A-2, -5652, -5919-1, -6795Rev; Ds-bearing alleles
sh1-m6795, -m5933, -m6233, -m6258, -m6598; restriction map of Sh1 and Ds with
AccI, BamHI, BcII, BglII, BstEII, EcoRI, HaeII, HincIII, HindIII, PstI, PvuI,
SstI, XbaI sites
sh1 alleles -W22, -7196, -7205, -7342, -7611, -7650, -7731, -m6233, -m5933, -bz1-m4;
Sh1-W22
Sh1 restriction map with EcoRI, XbaI, BcII, SstI, BglI, BglII sites

Peterson, 57:2
 Rhoades &, 57:16
 Rhoades &, 57:13
 Pasupuleti &, 1982
 Peterson, 57:2
 Dellaporta &, 57:26
 Courage-Tebbe &, 57:29
 Werr &, 57:30
 Burr &, 1982
 Chourey, 1981
 Döring &, 1981

Chromosome 10

TB-10L22 - Ef1 - TB-10L36 - Ef2 - TB-10L20 - Ef3 - TB-10L14 - (Ef4);
 TB's in 10L (18,19) - zn1 - (26) - du1 - (22) - (bf2, li1) - (36) - (20) -
 (1, 3, 4, 5, 7, 9, 10, 25, 28, 31, 37) - (6, 8, 11, 12, 14, 16, 17, 24, 27, 29,
 30, 34, 35, 38) - (2, 21, 23) - gl - (32) - r1; Ef3 - (13, 15, 33) - gl
zein cDNA clone for 18-20kd chains hybridizes in situ to distal 1/3 of 10L, with T9-10b

Lin, 1982
 Viotti &, 1982

Unplaced

Bg (Bergamo) regulatory element; *B2h regulatory element
Bg-in inactive regulatory element
d*-x - 1.35 - te* (terminal ear)
dys1 not allelic to dys2, afd1, asl; pam1 not allelic to pam2
dvl not uncovered by TB-1Sb, TB-1La-558041, TB-5La, TB-6Sa, TB-7Lb, TB-9Sd
ed*-7-1, -G7-2, -G33, -27-1, -57-1, -64-1, -51-1, -G22, -55-1, -54-1, -56-1, -48-1,
 -61-1 (developmental mutants in seed)
hcf*-6 not allelic to hcf*-2; hcf*-38 not allelic to hcf*-2
Ltr*-19 (lysine plus threonine resistance)
Phi2 (phosphohexoseisomerase) isozymes
Sd (South Dakota 15) regulatory element
Tpm1 (thylakoid polypeptide modifier); "slow" form dominant to "fast"
Uq (ubiquitous) controlling element
zein cDNA clones (two of 22kd, six of 19kd, six of 15kd chains), maps of BamHI, DdeI,
HaeIII, HincII, HinfI, PstI sites; base sequences of two of 22kd and one of 19kd
zein cDNA clones (four 21kd), maps of HaeIII, HpaII, PstI, BstNI, BamHI, PvuII, HincII,
PvuI, HinfI sites; homologies inter se and with a 19kd clone

Salamini, 1980
 Salamini &, 1982
 Mynbaev &, 1982
 Golubovskaya &, 1981
 Curtis, 57:31
 Dolfini &, 57:93
 Leto, 1982
 Hibberd &, 1982
 Stuber &, 57:128
 Salamini, 1980
 Modena, 57:38
 Friedemann &, 1982
 Marks &, 1982,
 & Pedersen &, 1982
 Pintor-Toro &, 1982

Genomic clones

Cin1, 700bp repetitive sequence (Cinteot1, young maize god; corn insert), present on
 5.7kb EcoRI fragment of McClintock Northern Flint line but not on W22 C1 R1, same
 fragment; map of SalI, BamHI, BglI, EcoRI, HindIII, KpnI, SstI sites

Shepherd &, 1982,
 & 57:158

Genomic clones, continued

Cin102 member of Cin1 family of dispersed, repetitive sequences, 87% homology to Cin1 Shepherd &, 57:158
 Teol insert, 3kb, in 8kb EcoRI fragment of Guerrero teosinte along with 5kb EcoRI fragment Shepherd &, 57:159

ctDNA

tRNAile2 base sequence, and intron Guillemaut &, 1982
 map of BamHI, BglII, HindIII sites, LS-RNA, wheat homologies Koller &, 1982
 base sequences of genes for beta and epsilon subunits of CF1 Krebbers &, 1982
 tRNAleuUAA base sequence, and intron Steinmetz &, 1982
 map of restriction sites, LS, tRNA's, rRNA's; includes tRNAhis, tRNAile, tRNAala, Stiles, 1982
 tRNAval, 16SrRNA, 23SrRNA, IVSI, IVSII, LS, and sites of AluI, AvaII, BamHI, BglII,
 EcoRI, HaeIII, HhaI, HincII, HindII, HinfI, HpaII, PstI, Sau96I, SmaI, TaqI

mtDNA

plasmids (2.35, 1.57, 1.42, 2.2kb) variable in cms-T, cms-S, cms-C, cytopl-B37, Newton, 57:139
 cytopl-Ky21
 plasmids (2.3kb linear, 1.94kb circular, 1.4kb linear) variable in stoichiometry Smith &, 57:47
 S1 and S2-homologous sequences in BamHI fragments McNay &, 57:48
 plasmids (1.5, 1.8kb), homologies to total mtDNA and mtRNA; HhaI restriction fragments Dale, 1981
mox1 (cytochrome oxidase subunit II) contains an intervening sequence; map of HinfI, Fox &, 1981
 TaqI, Sau3A, BamHI, EcoRI, HindIII, MspI, HaeIII sites; base sequence
 XhoI fragment differences in cms-T vs. fertile revertants Gengenbach &, 1982
 map of rRNA26S, rRNA18S, and BamHI, PstI, SalI, HindIII, SmaI sites Iams &, 1982
 S1 and S2 homologies inter se, maps of HindIII, XhoI, EcoRI, SacI, XbaI, SalI, BamHI, Kim &, 1982
 BstEII sites; inverted repeats
 BamHI fragments of cms-T vs. cytopl-NC7 (xT204); 15 of 35 sequences conserved Spruill &, 1981
 map of SstII and SmaI sites, rRNA15S, rRNA18S, rRNA26S; hybridizations and cosmid frags. Stern &, 1982
 map of BamHI, ClaI, SmaI, SstI, SstII sites, cosmid fragments; homology to ctDNA repeat Stern &, 1982
 region carrying 16SrRNA, tRNAval, tRNAile, tRNAala; alterations in cms-C, cms-S,
 cms-T restriction fragments
 R1 and R2 linear elements and BamHI sites; cytopl-RU Weissinger &, 1982

Resistance and Tolerance

anthracnose stalk rot (Colletotrichum graminicola) resistance/susceptibility; additive Carson &, 1981
 genetic effects accounted for 90% of total variation; heritability estimates
 anthracnose stalk rot R/S associated by translocation tests with chromosomes 1, 4, 6, 8 Carson &, 1982
 downy mildew (Peronosclerospora philippinensis) R/S dominance vs. additivity variable Kaneko &, 1981
 with age and intensity of infection; polygenic inheritance
 downy mildew (Peronosclerospora sorghi) R/S variation in diallel Lima &, 1982
 gray leaf spot (Cercospora zea-maydis) R/S association with chromosomes 1, 7, and/or 9 Galinat &, 57:53
 of Durango teosinte
 kernel rot (Fusarium moniliforme) R/S variation in diallel King &, 1981
 leaf spot (Cochliobolus carbonum = H. carbonum) R/S, additive gene effects Hamid &, 1982
 northern corn leaf blight (H. turcicum) R/S did not segregate in F2 from crosses Hooker &, 1980
 inter se or with Ht1 sources; 15:1 segregations occurred in crosses with Ht2 source
 northern corn leaf blight R/S polygenic, predominantly non-additive gene effects Moura &, 1981
 southern corn leaf blight (Helminthosporium maydis) T toxin exposure not lethal to Earle, 1982
 hybrid fusion protoplasts of cms-T with normal
 southern corn leaf blight R/S to race 0, 58.8% of variation due to additive effects Saxena &, 1981
 Maize Dwarf Mosaic Virus R/S to strain A, estimates of 1, 2, 3 genes Scott &, 1982
 Maize Streak Virus R/S simply inherited in some progenies Soto &, 1982
 Alachlor tolerance/susceptibility; non-additive genetic effects Francis &, 1980
 Eradicane R/S intermediate in hybrids Landi &, 1981
 Glyphosate T/S differences in 240 F2 hybrid tests Andersen &, 1982
 Diclofop T/S differences in F2 hybrid tests "

Ligate's Corner

Candidate Clone: Sequence not yet elect

Ligate's Corner

Gnomic Clone: Sequence from a dwarf,
 pygmy or brachytic locus

GENE LIST AND LINKAGE MAP OF CORN (MAIZE) (Zea mays L.)
March 1983

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The following list, arranged by gene symbol, identifies the unit factors for which stocks are available in the Maize Genetics Stock Center (Department of Agronomy, University of Illinois, Urbana, Illinois 61801), those for which variants exist in generally available strains (e.g., isozyme variants), and those upon which current or recent research studies have been published or reported in the Maize Genetics Cooperation News Letter. The information tabulated includes the chromosome (L=long arm, S=short arm) and map position or approximate location, the name and phenotype, availability from the Stock Center (S), a photograph (P) in The Mutants of Maize (reference number 199), and references to the original descriptions.

The linkage map represents the order and recombinational distances, in centimorgans, for those genes for which sufficient information is available to make a reasonable judgment of their location. Bracketing signifies that the exact order of the indicated genes is uncertain. Each chromosome is arranged beginning with the most distal gene in the short arm. The information on order and distances is not always unambiguous, and a number of positions are subject to correction with further data; this is particularly the case for parts of chromosomes 4, 5 and 10. Locations of the centromeres (Centr.) are indicated according to the best available data from cytogenetic studies. Locations of the B-A translocations, which generate hemizygous segments distal to the position indicated, are shown as TB-....; TB's shown as spanning one or more genes may or may not uncover the indicated gene or genes. The knob on the long arm of chromosome 3, K3L, has been placed by recombinational analysis. NOR is the nucleolus organizing region.

Note: This list and map is an updated version of the one recently published in Genetic Maps, Volume II, S.J. O'Brien, ed., National Cancer Institute NIH. The authors greatly appreciate the corrections supplied by fellow maize co-operators and encourage all those interested in maize genetics to make suggestions and/or corrections to this list.

Symbol	Location	Name, phenotype	S	P	Reference
a1	3L-127	anthocyaninless: colorless aleurone, green or brown plant, brown pericarp with P1-RR	S	P	65
a2	5S-35	anthocyaninless: like a1, but red pericarp with P1-RR	S	P	123
a3	3L-111	anthocyanin: red pigment in sheath, culm, and husks; resembles B1 but is recessive	S	P	159
Ac1		activator: transposable factor, regulates Ds activity		P	174
Ac2		activator: controlling element of bz2-m			236
ad1	1L-108	adherent: seedling leaves, tassel branches, and occasionally top leaves adhere	S	P	135
Adh1	1L-(128)	alcohol dehydrogenase (Adh2 of Scandalios): electrophoretic mobility; hybrid bands occur; null allele is known	S		260
Adh2	4S-46	alcohol dehydrogenase: electrophoretic mobility			258
Adr1	-	alcohol dehydrogenase regulator			145
afd1	-	absence of first division: first meiotic division replaced by mitosis			97
ae1	5L-57	amylose extender: glassy, tarnished endosperm; high amylose content	S	P	295
agt1	-	ageotropic: primary root unresponsive to gravity			57
al1	2S-4	albescent: erratic development of chlorophyll; pale yellow endosperm	S	P	219
alh1	1L-near bm2	histone Ia (symbol H1a, also used, is non-conforming): electrophoretic mobility			282
alpha	3L-111	alpha: component at A1 (see beta); pale aleurone, red-brown plant, dark brown pericarp with P1-RR		P	148
am1	5S-20	ameiotic: meiosis fails, sporogenous tissue degenerates	S	P	210 233
Amp1	1L-near f1	leucine aminopeptidase (was LapA, Lp1): electrophoretic mobility; no hybrid bands			208
Amp2	1	leucine aminopeptidase (was LapD, Lp2): electrophoretic mobility; no hybrid bands			208
Amp3	5 -near bt1	leucine aminopeptidase (was LapC): electrophoretic mobility			208

Symbol	Location	Name, phenotype	S P	Reference
Amp4	-	leucine aminopeptidase (was LapB): electrophoretic mobility		208
Amy1	-	alpha amylase: electrophoretic mobility; no hybrid bands		36
Amy2	5 -near Cat1	beta amylase: electrophoretic mobility; no hybrid bands		35
an1	1L-104	anther ear: dwarf with anthers in ear florets; few tassel branches; responds to gibberellins	S P	62 72
Ap1	-	acid phosphatase: electrophoretic mobility		60
Ap2	-	acid phosphatase: electrophoretic mobility		60
Ap3	-	acid phosphatase: electrophoretic mobility		60
aph1	-	aphid resistance		34
ar1	9L-62	argentina: virescent seedling, greens rapidly	S P	75
AR		aberrant ratio: distorted ratios following virus infection		276
as1	1 -56	asynaptic: synaptic failure of meiotic prophase chromosomes	S P	15
Asr1	4S-19	absence of seminal roots		182
Atc1	9 -near wx1	strong anthocyanin: on leaf blade after 4-6 leaf stage		200
B1	2S-49	colored plant: anthocyanin in major plant tissues: some alleles (see R2) affect seed color	S P	67
B chr		B chromosome: supernumerary chromosome		P 227
ba1	3L-80	barren stalk: ear shoot and most tassel florets missing	S P	113
ba2	2 -near ts1	barren stalk: like ba1, but tassel more normal	S	113
bd1	7L-109	branched silkless: branched ear and tassel; silks absent	S P	138
beta	3L-111	beta: component of A1 (see alpha); purple or red aleurone and plant color, red pericarp		148
Bf1	9L-137	blue fluorescent: homozygous seedlings (homozygous or heterozygous anthers) fluoresce blue under ultraviolet; anthranilic acid present	S P	288
bf2	10L-30	blue fluorescent: similar to Bf1 in expression; shows earlier, stronger seedling fluorescence than Bf1	S	2
Bh1	6L-46	blotched: colored patches on colorless (c1) aleurone	S P	68
Bif1	8 -near v16	barren inflorescence (=Tht): florets missing from ear and tassel		200
bk2	9L-82	brittle stalk: brittle plant parts after 4-leaf stage	S P	147
bm1	5S-41	brown midrib: brown pigment over vascular bundles of leaf sheath, midrib, and blade	S P	74
bm2	1L-161	brown midrib: like bm1	S	33
bm3	4 -near su1	brown midrib: like bm1 (C.R. Burnham, unpublished)	S	
bm4	9L-141	brown midrib: like bm1	S	30
Bn1	7L-71	brown aleurone: yellowish brown aleurone color	S	142
br1	1L-81	brachytic: short internodes, short plant; no response to gibberellins	S P	134 136
br2	1L	brachytic: like br1	S	150
br3	5	brachytic: like br1	S	266
bs1	-	barren sterile		178
bt1	5L-42	brittle: mature kernel collapsed, angular, often translucent and brittle	S P	169 296
bt2	4S-67	brittle: like bt1; ADP glucose pyrophosphorylase electrophoretic mobility (G.F. Sprague, unpublished)	S	
btn1	-	brittle node		133
bu1	-	leaf burn: leaves show burning, sometimes horizontal bands, accentuated by high temperature		89
bv1	5L-47	brevis: short internodes, short plant	S	152
bv2	-	brevis: plant height 30-50% of normal		220
Bx1	-	benzoxazin: blue color reaction of crushed root tip with FeCl3, indicating cyclic hydroxamates present		46
bz1	9S-31	bronze: modifies purple aleurone and plant color to pale or reddish brown; anthers yellow-fluorescent	S P	232
bz2	1L-106	bronze: like bz1; anthers not fluorescent	S P	205
c1	9S-26	colored aleurone: c1=colorless; C1-I=dominant colorless	S P	59
c2	4L-117	colorless: colorless aleurone, reduced plant color	S P	39
Car1	1S	catalase regulator: enzyme activity level increased		249
Cat1	5S-near Mdh5	catalase: electrophoretic mobility; hybrid bands occur		18
Cat2	1S	catalase: electrophoretic mobility; hybrid bands occur		250
Cat3	1L	catalase: electrophoretic mobility		252
Cdh1	-	cinnamyl alcohol dehydrogenase: electrophoretic mobility		84

Symbol	Location	Name, phenotype	S	P	Reference
Ce1	-	curled entangled: rolled leaves tend to be entangled			37 211
cfl2	-	complementary to fl2			209
Cg1	3S-23	corngrass: narrow leaves, extreme tillering	S	P	265
Ch1	2L-155	chocolate: dark brown pericarp	S	P	5
cl1	3S-39	chlorophyll: white to green seedlings, depending upon Clm1; pale yellow endosperm	S		73
clh1	-	histone Ic: electrophoretic mobility			282
Clm1	8	modifier of cl1: modifies seedling, not endosperm	S		73
Clt1	8	clumped tassel: variable dwarfing, developmental anomalies			92
cm1	10L-near R1	chloroplast mutator: like ij1	S		284
cms-C	-	cytoplasmic male sterility: female transmitted male sterility, C type; restored by Rf4			16
cms-S	-	cytoplasmic male sterility: female transmitted male sterility, S type; restored by Rf3			130 132
cms-T	-	cytoplasmic male sterility: female transmitted male sterility, Texas type; restored by Rf1 Rf2	S		130 132
cp1	7 -near vp9	collapsed: endosperm collapsed and partially defective			162
cp2	7 -near cp1	collapsed: endosperm rough, collapsed, partially defective; seedling very light green with darker streaks		P	199
cr1	3S-0	crinkly: plant short; leaves broad, crinkled	S	P	69
ct1	8	compact: semi-dwarf plant			194
ct2	1S	compact: semi-dwarf plant with club tassel			96
cto1	-	cob turned out: ear inverted to a sheet or tube, kernels internally placed; variable expression (compare te1)			294
Cx1	10 -near du1	catechol oxidase: electrophoretic mobility; no hybrid bands; null allele is known			226
d1	3S-18	dwarf: plant andromonoecious, short, compact; responds to gibberellins	S	P	62
d2	3	dwarf: like d1			286
d3	9S-59	dwarf: like d1	S		52
d5	2S-34	dwarf: like d1	S		286
D8	1L-133	dwarf: dominant, resembles d1; not responsive to gibberellins	S	P	217
da1	9	dilute aleurone: aleurone color diluted			79
db1	-	dichotomously branching plants (=dib): variable location of dichotomy, usually at 4-8th node (possible association with aneuploidy)			177 178
dek1	1S-27	defective kernel (was clf1, gay1): germless, floury; anthocyanins and carotenoids absent			201 202
dek2	1L	defective kernel: discolored, scarred endosperm; inviable			201 202
dek3	2S	defective kernel: germless			201 202
dek4	2L	defective kernel: collapsed, floury lethal			201 202
dek5	3S	defective kernel: shrunken endosperm; white seedling with green stripes			201 202
dek6	3L	defective kernel: shrunken, lethal			201 202
dek7	4S	defective kernel: shrunken sugary endosperm; white seedling with green stripes			201 202
dek8	4L	defective kernel: shrunken, lethal			201 202
dek9	5L	defective kernel: crumpled endosperm, lethal			201 202
dek10	6L	defective kernel: collapsed endosperm, lethal			201 202
dek11	7L	defective kernel: etched endosperm, lethal			201 202
dek12	9S	defective kernel: collapsed endosperm, lethal			201 202
dek13	9L	defective kernel: defective opaque endosperm, lethal			201 202
dek14	10S	defective kernel: collapsed endosperm, lethal			201 202
dek15	10L	defective kernel: collapsed floury endosperm, lethal			201 202
dep1	6	defective pistils			179
Df		deficiency: general symbol for loss of segments of chromosome			
dp1	4L-137	distal pale: seedling leaf tip virescent (E.G. Anderson, unpublished)	S		
Ds		dissociation: transposable factor, associated with chromosome breakage and/or control of expression of adjacent genes; regulated by Ac1	S	P	174
dsy1	-	desynaptic			98
dsy2	-	desynaptic			100
Dt1	9S-0	dotted: regulates controlling element at A1; responding a1-m alleles express colored dots on colorless kernels and purple sectors on brown plants	S	P	228
Dt2	6L-40	dotted: like Dt1	S		206
Dt3	7L	dotted: like Dt1, but expression variable	S		206

Symbol	Location	Name, phenotype	S P	Reference
Dt4	4	dotted: like Dt1, but dots chiefly on crown of kernel	S	54
Dt5	9 -near yg2	dotted: like Dt1		54
du1	10L-28	dull: glassy, tarnished endosperm (P.C. Mangelsdorf, unpublished)	S	
dv1	-	divergent: spindle nonconverging in meiosis in microsporocytes	S	38
dy1	-	desynaptic: chromosomes unpaired in microsporocytes	S	193
E1	7L	esterase: electrophoretic mobility; hybrid bands occur		255
E2	-	esterase: presence-absence		257
E3	3 -near E4	esterase: electrophoretic mobility; hybrid bands occur		256
E4	3S	esterase: electrophoretic mobility; no hybrid bands; null allele is known		108
E5-I	-	esterase: electrophoretic mobility; duplicate factor with E5-II		165
E5-II	-	esterase: see E5-I		165
E6	-	esterase: presence-absence		165
E7	-	esterase: presence-absence		165
E8	3S	esterase (=Est1): presence-absence		165
E9	-	esterase: electrophoretic mobility; null allele is known		165
E10	-	esterase: electrophoretic mobility		165
E12	-	esterase: electrophoretic mobility; no hybrid bands		26
E16	7	esterase: electrophoretic mobility; no hybrid bands		26
eg1	5L	expanded glumes: glumes open at right angle	S	31
Ej1	10	(see Isr1)		
el1	-	elongate: chromosomes uncoiled during meiotic metaphase and anaphase; frequent unreduced gametes	S P	233
En		enhancer: transposable factor, regulates pg14-m mutation; equivalent to Spm	P	215
Enp1	6 -near Y1	endopeptidase (was Ep1): electrophoretic mobility; no hybrid bands		176
et1	3L-139	etched: pitted, scarred endosperm; virescent seedling	S P	278
f1	1L-86	fine stripe: virescent seedling, fine white stripes on base and margin of older leaves	S P	156
Fcu		factor Cuna: controlling element of r1-cu		102
fl1	2S-68	floury: endosperm opaque, soft; dosage effect	S P	112
fl2	4S-58	floury: like fl1, but phenotypically dominant (W.J. Mumm, unpublished)	S	
fl3	8L-0	floury	S	190
fv1	-	flavones: polyphenols in silks absent		151
g1	10L-47	golden: seedling and plant with distinct yellow cast	S P	62 64
g2	3S	golden (allele pg14): like g1, but more extreme; sheaths whitish yellow-green		120
g5	3	(allele pg14)	S	
Ga1	4S-32	gametophyte factor: Ga1 pollen grains competitively superior to ga1 on Ga1 silks	S	131
ga2	5L-5	gametophyte factor: Ga2 pollen grains competitively superior to ga2		29
ga7	3L-145	gametophyte factor: ga7 pollen from heterozygotes 10-15% functional regardless of silk genotype		230
ga8	9S-near lo2	gametophyte factor: Ga8 pollen grains competitively superior to ga8 on Ga8 silks		254
ga10	5	gametophyte factor		101
Gdh1	1L-145	glutamic dehydrogenase: electrophoretic mobility		224
Gdh2	10	glutamic dehydrogenase: electrophoretic mobility		103
gl1	7L-36	glossy: cuticle wax altered; leaf surface bright, water adheres	S P	142
gl2	2S-30	glossy: like gl1	S P	110
gl3	4L-112	glossy: like gl1	S	110
gl4	4L-81	glossy: like gl1 (G.F. Sprague, unpublished)		
gl5	-	glossy: like gl1 (G.F. Sprague, unpublished)		
gl6	3L-47	glossy: like gl1 (G.F. Sprague, unpublished)	S	
gl7	-	glossy: like gl1 (G.F. Sprague, unpublished)		
gl8	5L-67	glossy: like gl1 (G.F. Sprague, unpublished)	S	
gl9	-	glossy: expression poor (G.F. Sprague, unpublished)		
gl11	2S-near B1	glossy: like gl1; abnormal seedling morphology	S	273
gl14	2	glossy: like gl1	S	275
gl15	9L-66	glossy: like gl1; expressed after 3rd leaf (G.F. Sprague, unpublished)	S P	3
gl17	5S-34	glossy: like gl1 but semi-dwarf with necrotic crossbands on leaves	S	234
gl18	8	glossy: like gl1; expression poor	S	4

Symbol	Location	Name, phenotype	S P	Reference
Glu1	10 -near du1	glucosidase: electrophoretic mobility		225
Got1	3L-near na1	glutamate-oxaloacetate transaminase (possibly = Ta1): electrophoretic mobility; glyoxysomal		251
Got2	5L-96	glutamate-oxaloacetate transaminase: electrophoretic mobility; cytosolic		104
Got3	5S-near a2	glutamate-oxaloacetate transaminase: electrophoretic mobility; mitochondrial		104
gs1	1L-135	green stripe: grayish green stripes between vascular bundles on leaves; tissue wilts	S P	68 181
gs2	2S-54	green stripe: like gs1, but pale green stripes; no wilting (G.F. Sprague, unpublished)	S P	
gt1	-	grassy tillers: numerous basal branches; vegetatively totipotent in combination with id1 and pe1		261
h1	3	soft starch: endosperm soft, opaque	S	188
hcf1	-	high chlorophyll fluorescence: strong red fluorescence under long-wave ultraviolet irradiation; very low CO2 fixation		180
hcf2	-	high chlorophyll fluorescence: like hcf1, but lethal		180
hcf3	-	high chlorophyll fluorescence: like hcf1, but lethal		180
hm1	1L-64	susceptibility to <i>Helminthosporium carbonum</i> : disease lesions on leaves, black masses of fruiting bodies on ears with race 1	S P	292
hm2	9L-near bk2	susceptibility to <i>Helminthosporium carbonum</i> : like hm1; masked by Hm1		196
Hs1	7S-0	hairy sheath: abundant hairs on leaf sheath	S P	287
Ht1	2L-121	resistance to <i>Helminthosporium turcicum</i>	S	114
Ht2	-	resistance to <i>Helminthosporium turcicum</i>		115
Ht3	-	resistance to <i>Helminthosporium turcicum</i> (from <i>Tripsacum floridanum</i>)		116
I		inhibitor (=C1-I, inhibitor allele at C1 locus): also commonly used as a general symbol for inhibition and for the controlling elements responding to En		56
id1	1L-near an1	indeterminate growth: requires extended growth and short days for flowering; vegetatively totipotent with gt1 and pe1	S	264
Idh1	8	isocitrate dehydrogenase: electrophoretic mobility		104
Idh2	6L-near py1	isocitrate dehydrogenase: electrophoretic mobility		104
ig1	3L-68	indeterminate gametophyte: polyembryony, heterofertilization, polyploidy, androgenesis	S	139
ij1	7L-52	iojap: many variable white stripes on leaves; conditions chloroplast defects that are cytoplasmically inherited	S P	119
in1	7S-20	intensifier: intensifies anthocyanin pigments	S P	82
Inv		inversion: general symbol for inversion of a segment of chromosome	S P	
is1	-	cupulate interspace		87
Isr1	10L-near R1	inhibitor of striate: reduces expression of sr2 and other leaf-stripping factors (replaces Ej1)		140
j1	8L-42	japonica: white stripes on leaf and sheath; not expressed in seedling	S P	64
j2	4L-106	japonica: extreme white striping of leaves, etc. (R.A. Emerson, unpublished)	S P	
K3L	3L-93	knob: constitutive heterochromatic element		53
K10	10L-near sr2	abnormal-10: heterochromatic appendage on long arm of chromosome 10; neocentric activity distorts segregation of linked genes	S P	161
Kn1	1L-(128)	knotted: scattered proliferation of tissue at vascular bundles on leaf	S P	27
L1	10L-63	luteus: yellow pigment in white tissue of chlorophyll mutants w1, j1, ij1, etc.	S P	154 155
L4	-	luteus: lethal yellow seedling	S P	125
L6	9S-near bz1	luteus: like L4 (W.H. Eyster, unpublished)	S	
L7	9S-42	luteus: yellow seedling and plant	S	79
L10	6L-15	luteus: like L4	S	241
L11	6	luteus: yellow seedling with green leaf tips	S	8
L12	6L-12	luteus: like L4	S	47
L13	10L-91	luteus (was L*-E59, L*-Neuffer2): like L4		170
L15	6L-26	luteus (was L*-Blandy3, L*-Brawn): like L4		245
la1	4S-55	lazy: prostrate growth habit	S P	126
Lc1	10L-65	red leaf stripe: red color in leaf surface	S	19
Les1	2S-near wt1	lesion: large necrotic lesions resembling disease lesions formed by fungal infections on susceptible lines	S	198

Symbol	Location	Name, phenotype	S P	Reference
Les2	1S	lesion: small white lesions resembling disease lesions formed by fungal infections on resistant lines	S	198
Les3	10	lesion: like Les1		7
Lg1	2S-11	liguleless: ligule and auricle missing; leaves upright, enveloping	S P	62 63
Lg2	3L-79	liguleless: like Lg1, less extreme	S P	22
Lg3	3 -43	liguleless: dominant, no ligule; leaves upright, broad, often concave and pleated	S P	212
Li1	10L-near bf2	lineate: fine, white striations on basal half of mature leaves	S P	44
Lls1	1	lethal leaf spot: chlorotic-necrotic lesions resembling Helminthosporium carbonum infection	S	293
Ln1	-	linoleic acid: lower ratio of oleate to linoleate in kernel		48
Lo2	9S-50	lethal ovule: ovules containing lo2 gametophyte abort	S	193
Loc1	-	low oil content in kernel: associated with albino seedlings		221
Lp1	4	lethal pollen: Lp1 pollen fails in competition with Lp1		192
Lte1	2	latente: heat tolerance		184
Lte2	10	latente: heat tolerance		185
Lu1	5S-29	lutescent: pale yellow-green leaves	S	262
Lw1	1L-(128)	lemon white: white seedling, pale yellow endosperm	S	290
Lw2	5L-near pr1	lemon white: like Lw1	S P	290
Lw3	5L-near v2	lemon white: like Lw1; duplicate factor with Lw4		290
Lw4	4 -near zb6	lemon white: see Lw3		290
Ly1	5	lycopenic: similar to ps1 (possible allelism untested) but not viviparous; accumulates lycopene		80
M		(commonly used as a general symbol for mutator or modifier)		
Mal1	9	multiple aleurone layering: recessive interacts with two complementary dominants Mal2 and an unnamed factor, giving multiple cell layers		183
Mal2	4	multiple aleurone layering (see Mal1)		183
Mdh1	8	malate dehydrogenase (mMdh4 of Scandalios and co-workers): electrophoretic mobility; mitochondrial		203
Mdh2	6L-near py1	malate dehydrogenase (mMdh2 of Scandalios and co-workers): electrophoretic mobility; mitochondrial		203
Mdh3	3L-124	malate dehydrogenase (mMdh1 of Scandalios and co-workers): electrophoretic mobility; mitochondrial		203
Mdh4	1L-near an1	malate dehydrogenase (sMdh1 of Scandalios and co-workers): electrophoretic mobility; cytosolic		203
Mdh5	5S-17	malate dehydrogenase (sMdh2 of Scandalios and co-workers): electrophoretic mobility; cytosolic		203
Me1	3L-103	malic enzyme: electrophoretic mobility		104
mep1	5L	modifier of endosperm protein: affects quantities of Prot1 protein forms		259
Mer1	4	IAC Maya earworm resistance		186
Mer2	3	IAC Maya earworm resistance		186
mi1	1	midget: small plant (H.S. Perry, unpublished)	S	
mmm1	1L-near an1	modifier of mitochondrial malate dehydrogenases (mobilites)		203
mn1	2 -near fl1	miniature seed: small, somewhat defective kernel; fully viable	S P	163
mn2	7	miniature seed: small kernel, loose pericarp; extremely defective but will germinate (R.J. Lambert, unpublished)	S	
Mp		modulator of pericarp: transposable factor affecting P1 locus; parallel to Ac-Ds		24
Mr	9S-near L7	mutator of R1-m: transposable factor, regulates R1-m mutation	P	207
Mrh		mutator: controlling element of a1-m-rh		235
ms1	6 -near Y1	male sterile: anthers shriveled, not usually exerted		267
ms2	9L-64	male sterile: like ms1	S	76 79
ms3	3	male sterile: like ms1		76 79
ms5	5 -near v3	male sterile: anthers not exerted	S	12
ms7	7L-near gl1	male sterile: like ms5	S	12

Symbol	Location	Name, phenotype	S	P	Reference
ms8	8L-28	male sterile: like ms5	S	P	12
ms9	1 -near P1	male sterile: like ms5	S		12
ms10	10L-near bf2	male sterile: like ms5	S		12
ms11	10	male sterile: like ms5	S		12
ms12	1	male sterile: like ms1	S		12
ms13	5S	male sterile: like ms5	S		12
ms14	1 -near as1	male sterile: like ms5	S		12
ms17	1S-23	male sterile: like ms1	S		70
Ms21	-	male sterile: dominant; pollen grains developing in presence of Ms21 are defective and nonfunctional if <i>sks1</i> , normal if <i>Sks1</i>			149 253
Mst1	10L-67	modifier of R1-st: affects expression of R1-st	S		6
Mu		mutator: non-Mendelian trait; increased mutation rate			244
Mut	2	mutator: controlling element for <i>bz1-m-rh</i>			236
Mv1	-	resistance to maize mosaic virus I ("corn stripe")			20
na1	3L-91	nana: short, erect dwarf; no response to gibberellins	S	P	118 153
na2	5L-near bt1	nana: like na1 (H.S. Perry, unpublished)	S		
NCS		nonchromosomal stripe: maternally inherited light green leaf striping	S		263
nec1	8	necrotic (was <i>nec*-6697</i> , <i>sienna*-7748</i>): chlorotic seedling that stays rolled, wilts and dies	S		170
nec2	1S	necrotic: green seedling develops necrotic lesions at 2-3 leaf stage; lethal (E.G. Anderson, unpublished)	S		
nec3	5 -near bt1	necrotic: seedlings emerge with tightly rolled leaves that turn brown and die without unrolling; manually unrolled leaves tan with dark brown crossbands	S		197
nl1	10L-near bf2	narrow leaf: leaf blade narrow, some white streaks (R.A. Emerson, unpublished)	S	P	
NOR	6S	nucleolus organizer: codes for ribosomal RNA	S		172
o1	4 -near gl3	opaque: endosperm starch soft, opaque (W.R. Singleton and D.F. Jones, unpublished)	S		
o2	7S-16	opaque: like o1; high lysine content (W.R. Singleton and D.F. Jones, unpublished)	S	P	
o5	7L-near gl1	opaque: like o1	S		240
o6	-	opaque: like o1; lethal seedling			189
o7	10L-87	opaque: like o1; high lysine content	S		187
o9	-	opaque endosperm: crown opaque and light in color, frequently with a cavity; base or abgerminal side of kernel often corneous			191
o10	-	opaque endosperm: like o1			191
o11	-	opaque endosperm: thin, opaque, somewhat shrunken kernels with greyish cast			191
o12	-	opaque endosperm: thin, etched or scarred kernels, variable in size; plants chlorophyll deficient and small, with pollen but few ears			191
o13	-	opaque endosperm: opaque, etched kernels with rim of corneous starch on abgerminal side			191
Og1	10S-16	old gold: variable bright yellow stripes on leaf blade	S	P	159
ora3	-	orange endosperm: heterozygous plants in hybrids taller, earlier, smaller-eared than homozygous normals			55
oro1	6	orobanche: yellow to tan necrotic with cross-banding when grown under light-dark cycle; some chlorophyll with <i>Orom1</i>			170
oro2	-	orobanche: like oro1			170
Orom1	-	orobanche modifier: partially corrects bleaching of oro1			170
oy1	10S-12	oil yellow: seedling oily greenish-yellow	S	P	78
P	10L-61	plant color component at R1: anthocyanin pigmentation in seedling leaf tip, coleoptile, anthers			279 280
P1	1S-26	pericarp color: red pigment in cob and pericarp	S	P	61 160
pam1	-	plural abnormalities of meiosis: desynchronized meiotic divisions and premeiotic mitosis; plants male sterile, incompletely female sterile			99
pam2	-	plural abnormalities of meiosis: like pam1			100
pb1	6L-near L10	piebald: very light, irregular green bands on leaf		P	51
pb4	6 -near Y1	piebald: like pb1	S		51

Symbol	Location	Name, phenotype	S P	Reference
pd1	-	paired rows: single vs. paired pistillate spikelets; pd1 is found in teosinte also		146
pe1	-	perennialism: vegetatively totipotent in combination with gt1 and id1		261
pg11	6L-34	pale green: seedling light yellowish green; mature plant pale and vigorous; duplicate factor with pg12	S P	231
pg12	9L-61	pale green: see pg11	S	231
pg14	3S	pale green (allele g2 and g5): pale green leaves with normal green sectors; mutable allele controlled by En (Spm)	S P	215
Pgd1	6L-near Y1	6-phosphogluconate dehydrogenase: electrophoretic mobility		104
Pgd2	3 -near lg2	6-phosphogluconate dehydrogenase: electrophoretic mobility		104
Pgm1	1L-near tb1	phosphoglucomutase: electrophoretic mobility		104
Pgm2	5S-0	phosphoglucomutase: electrophoretic mobility		104
Ph1	4S-0	pith abscission: cob disarticulation		88
Phi1	1L-140	phosphohexose isomerase: electrophoretic mobility		104
Pl1	6L-45	purple plant: sunlight-independent purple pigment in plant	S P	67
pm1	3L-near ts4	pale midrib: midrib and adjacent tissue lighter green	S P	23
Pn1	7L-112	papyrescent: long, thin, papery glumes on ear and tassel	S P	90
po1	6S-0	polymitotic: microspore division without chromosome division	S P	11
pr1	5L-66	red aleurone: changes purple aleurone to red	S P	59
pro1	8L	proline requiring: crumpled opaque kernel; green stripe lethal seedling		91
Prot1	1L-121	protein: embryo protein mobility variations		259
ps1	5S-39	pink scutellum (=vp7): viviparous; endosperm and scutellum pink, seedling white with pink flush	S P	272
Pt1	6L-56	polytypic ear: proliferation of pistillate tissue to produce irregular growth on ear and tassel	S P	195
Px1	-	peroxidase: electrophoretic mobility; no hybrid bands; null allele is known		106
Px2	-	peroxidase: electrophoretic mobility		164
Px3	-	peroxidase: electrophoretic mobility		164
Px4	-	peroxidase: electrophoretic mobility		164
Px5	-	peroxidase: presence-absence		164
Px6	-	peroxidase: presence-absence		164
Px7	-	peroxidase: electrophoretic mobility; null allele is known		164
Px8	-	peroxidase: electrophoretic mobility		21
Px9	-	peroxidase: electrophoretic mobility		21
py1	6L-65	pigmy: leaves short, pointed; fine white streaks	S P	286
pyd1	9S-near yg2	pale yellow deficiency: pale yellow seedling; deficiency for short terminal segment of chromosome arm		173
R1	10L-61	colored: red or purple color in aleurone and/or anthers, leaf tip, brace roots, etc.	S P	59
R2	2S-49	colored: duplicate factor with R1 for aleurone color (either R1 or R2 is required); allelic to B1, affects plant color	S P	285
ra1	7L-32	ramosa: ear branched, tassel conical	S P	12 93
ra2	3S-near d1	ramosa: irregular kernel placement; tassel many-branched, upright (R.A. Brink, unpublished)	S P	
ra3	4	ramosa: (R.A. Brink, unpublished)	S	
rd1	1L	reduced: semi-dwarf plant	S	194
rd2	6L	reduced: like rd1, but not as extreme		95
Rf1	3 -near Lg3	restorer: restores fertility to cms-T; complementary to Rf2	S	129
Rf2	9 -near wx1	restorer: see Rf1	S	58
Rf3	2L	restorer: restores fertility to cms-S		28
Rf4	-	restorer: restores fertility to cms-C		105
Rg1	3 -45	ragged: chlorotic tissue between veins of older leaves, causing holes and torn appearance	S P	25
rgd1	6 -4	ragged seedling: seedling leaves narrow, thread-like, have difficulty in emerging	S P	143
Rgd2	5	ragged leaves (was Rgd*-1445): distorted growth		200
rgo1	-	reversed germ orientation: embryo faces base of ear; variable frequency, maternal trait		246

Symbol	Location	Name, phenotype	S P	Reference
rhm1	6S	resistance to <i>Helminthosporium maydis</i> : chlorotic-lesion reaction with race 0	S	268
Ri1	4S-27	rind abscission: cob disarticulation		88
Rp1	10S-0	rust resistant: resistant to <i>Puccinia</i> spp	P	166 167
Rp3	3 -near gl6	rust resistant: resistant to <i>Puccinia sorghi</i>	S	297
Rp4	4S-24	rust resistant: resistant to <i>Puccinia sorghi</i>	S	297
Rp5	10S-near Rp1	rust resistant: resistant to <i>Puccinia sorghi</i>		248
Rp6	10S-near Rp1	rust resistant: resistant to <i>Puccinia sorghi</i>		297
Rpp9	10S-near Rp1	rust resistant: resistant to <i>Puccinia polysora</i>		291
Rs1	-	rough sheath	S	141
rs2	1 -near as1	rough sheath	S	141
rt1	3 -near Cg1	rootless: secondary roots few or absent	S P	122
Ruq1	-	receptor: responds to regulatory element of Uq1		85
S	10L-61	seed color component at R1: anthocyanin pigmentation in aleurone		279
se1	-	sugary-enhancer: high sugar content with su1		81
sen1	3	soft endosperm: endosperm soft, opaque; duplicate factor with sen2		281
sen2	7	soft endosperm: see sen1		281
sen3	1	soft endosperm: like sen1; duplicate factor with sen4		281
sen4	-	soft endosperm: see sen3		281
sen5	2	soft endosperm: like sen1; duplicate factor with sen6		281
sen6	5	soft endosperm: see sen5		281
Sg1	-	string cob: reduced pedicels	S P	86
sh1	9S-29	shrunken: inflated endosperm collapses on drying, forming smoothly indented kernels	S P	117
sh2	3L-127.2	shrunken: inflated, transparent, sweet kernels collapse on drying, becoming angular and brittle; ADPG pyrophosphorylase reduced	S P	168
sh4	5	shrunken: collapsed, chalky endosperm	S	289
si1	6L-16	silky (=ms-si): multiple silks in ear; sterile tassel with silks	S	83
sk1	2S-56	silkless: pistils abort, no silks	S	128
Sks1	2 -near v4	suppressor of sterility: pollen grains developing in presence of Ms21 are defective and nonfunctional if sks1, normal if Sks1		149 253
sl1	7L-50	slashed: leaves slit longitudinally by necrotic streaks	S	110
sm1	6L-55	salmon: silks salmon color with P1-RR, brown with P1-WW	S P	1
Sod1	-	superoxide dismutase: electrophoretic mobility		9
Sod4	-	superoxide dismutase: electrophoretic mobility		9
Spc1	3	speckled (was Spc*-1376, Les*-1376): brown speckling on leaves and sheath at flowering; supporting tissues weak		200
Spm		suppressor-mutator: transposable factor, regulates responsive element at a1-m1, c2-m, pg14-m, etc.		175
sr1	1S-0	striate: many white striations or stripes on leaves (A.M. Brunson, unpublished)	S	
sr2	10L-95	striate: white stripes on leaf and sheath	S P	127
sr3	10S	striate: virescent and striate to striped	S P	96
st1	4S-62	sticky chromosome: small plant, striate leaves, pitted kernels resulting from sticky chromosomes	S P	13
su1	4S-66	sugary: endosperm wrinkled and translucent when dry; sweet at milk stage	S P	45
su2	6L-54	sugary: endosperm glassy, translucent, sometimes wrinkled	S	79
Sup1	-	suppressor: modifies o2 kernels to semi-transparent		171
sy1	-	yellow scutellum		271
T		reciprocal translocation: general symbol for exchange of parts between two nonhomologous chromosomes	S P	
Ta1	-	transaminase (possibly = Got1): electrophoretic mobility; hybrid bands occur		164
tb1	1L-near Kn1	teosinte branched: many tillers; nodes with slender branches ending in unbranched tassel		32
td1	5L	thick tassel dwarf: (E.G. Anderson, unpublished)	S	
te1	3	terminal ear (was ie1): stalked ear appendages at tip; varying to infolded ears (compare cto1)	S	247
Thc1	-	thiocarbamate sensitive: sensitive to Eradicane		216

Symbol	Location	Name, phenotype	S P	Reference
tl1	-	tasselless		178
Tp1	7L-46	teopod: many tillers, narrow leaves, many small partially podded ears, tassel simple	S P	158
Tp2	10L-45	teopod: like Tp1	S P	214
tr1	-	two-ranked ear: distichous vs. decussate phyllotaxy in ear axis		146
ts1	2S-74	tassel seed: tassel pistillate and pendant; if removed, small ear with irregular kernel placement develops	S	66
ts2	1S-24	tassel seed: like ts1, but branches pendant rather than whole tassel	S P	66
ts4	3L-52	tassel seed: tassel compact, upright, with pistillate and staminate florets	S P	218
Ts5	4S-53	tassel seed: dominant; nearly normal tassel with scattered, short silks	S	71
Ts6	1L-158	tassel seed: tassel pistillate to mixed, compact; ear with irregular kernel placement	S P	204
Tu1	4L-101	tunicate: kernels enclosed in long glumes; tassel glumes large, coarse	S P	42 43
ub1	-	unbranched: tassel with one spike	S P	199
Uq1		ubiquitous: controlling element for Ruq1		85
v1	9L-63	virescent: yellowish white seedling, greens rapidly	S P	50
v2	5L-107	virescent: like v1, but greens slowly	S P	64
v3	5L-45	virescent: light yellow seedling, greens rapidly	S P	50
v4	2L-83	virescent: like v2	S P	50
v5	7S-24	virescent: like v1, but older leaves have white stripes	S P	50
v8	4L-near Tu1	virescent: like v2	S	52
v12	5L-near ys1	virescent: like v3	S	219
v13	-	virescent: first leaf white with green tip; greens slowly	S	219
v16	8L-14	virescent: like v2	S	219
v17	4	virescent: like v1, but greening from base to tip	S	219
v18	10	virescent: like v1	S	219
v21	8L	virescent: grainy virescent, greening from tips and margins inward	S	17
v22	1 -near an1	virescent: like v1 (E.G. Anderson, unpublished)	S	
v23	4 -near su1	virescent: like v1 (E.G. Anderson, unpublished)	S	
va1	7L-near ij1	variable sterile: male sterile with some fertile anthers	S	14
Vg1	1L-85	vestigial glumes: glumes very small, cob and anthers exposed	S P	274
vp1	3L	viviparous: embryo fails to become dormant; chlorophyll and carotenoids unaffected; anthocyanins in aleurone suppressed	S	77
vp2	5S-38	viviparous: embryo fails to become dormant; white endosperm, white seedling; anthocyanins unaffected	S P	77
vp5	1S-1	viviparous: like vp2	S P	237
vp7	5	(= ps1)		
vp8	1L-154	viviparous: embryo fails to become dormant; chlorophyll, carotenoids, and anthocyanins unaffected; small, pointed-leaf seedling	S	238
vp9	7 -25	viviparous: like vp2	S P	238
w1	6L-near w14	white: white seedling	S	62 63 155
w2	10L-77	white: white seedling	S	157
w3	2L-111	white: like vp2	S P	157
w11	9S-54	white: like w1	S	52
w14	6L-74	white: like w1	S	47
w15	6L-9	white: like w1	S	47
Wc1	9L-107	white cap: kernel with white crown and pale yellow endosperm	S	144
wd1	9S-near yg2	white deficiency: white seedling; deficiency for distal half of first chromomere of short arm	S P	173
whp1	-	white pollen: duplicate factor with c2 for yellow pollen and for anthocyanins		40
wi1	6-near Y1	wilted: chronic wilting, delayed differentiation of metaxylem vessels	S	223
ws1	-	white sheath: light yellow leaf sheaths; duplicate factor with ws2	S	137
ws2	-	white sheath: see ws1	S	137
ws3	2S-0	white sheath: white leaf sheath, culm, husks	S P	229
wt1	2S-60	white tip: tip of first leaf white and blunt	S	277
wx1	9S-56	waxy: amylopectin (stained red by iodine) replaces amylose (blue staining) in endosperm and pollen	S P	41

Symbol	Location	Name, phenotype	S	P	Reference
Y1	6L-13	yellow: carotenoid pigments in endosperm; some alleles affect green pigments in seedlings	S	P	45
Y3	2S-near al1	yellow: endosperm color			213
y8	7S-18	yellow: light yellow endosperm	S		124
y9	10S-24	yellow: pale yellow endosperm, slightly viviparous; green to pale green seedlings and plants	S		243
y10	3L	yellow: pale yellow endosperm; white seedling	S		239
yd2	3L-near lg2	yellow dwarf			242
yg1	5L-near v2	yellow-green: yellow-green seedling and plant	S		74
yg2	9S-7	yellow-green: like yg1	S	P	121
ys1	5L-75	yellow stripe: yellow tissue between leaf veins, reflects iron deficiency symptoms	S	P	10
ys2	1S	yellow stripe (previous use of ys2 has lapsed): yellow tissue between leaf veins			222
ys3	3L-near Lg3	yellow stripe: like ys1	S		298
Ysk1	4 -near su1	yellow-streaked: longitudinal yellow streaks top 3rd of mature leaves			200
z1	-	zeta-carotenic: carotenoids photosensitive; embryo viviparous; accumulates zeta-carotene			80
zb1	-	zebra: yellowish crossbands on older leaves	S		49
zb2	-	zebra: crossbands on seedling leaves	S		283
zb3	5 -near v2	zebra: crossbands on older leaves (M. Demerec, unpublished)	S		
zb4	1S-19	zebra: regularly spaced crossbands on earlier leaves; enhanced by cool temperatures	S	P	109
zb6	4 -79	zebra: regularly spaced crossbands on older leaves; enhanced by cool temperatures	S		111
Zer1	4	Zapalote Chico earworm resistance			186
Zer2	6	Zapalote Chico earworm resistance			186
Zer3	2	Zapalote Chico earworm resistance			186
Zer4	1	Zapalote Chico earworm resistance			186
zn1	10L-26	zebra necrotic: necrotic tissue appears between veins in transverse leaf bands on half-grown or older plants	S	P	107
zn2	-	zebra necrotic: like zn1	S		94
Zp1	7 -near ra1	zein polypeptide: variation in 20 kd zein proteins			269
Zp2	7 -near ra1	zein polypeptide: like Zp1			269
Zp3	7 -near ra1	zein polypeptide: like Zp1			269
Zp6	7S-near Hs1	zein polypeptide: like Zp1			269
Zp16	7S-near gl1	zein polypeptide: like Zp1			270
Zp21	7S-9	zein polypeptide: like Zp1			270
Zp29	7S-12	zein polypeptide: like Zp1			270

REFERENCES TO ORIGINAL DESCRIPTIONS AND DESIGNATIONS
(gene symbol(s) referenced included within brackets)
(MNL = Maize Genetics Cooperation News Letter)

1. Anderson, E.G. 1921. Cornell Univ. Agr. Exp. Sta. Memoir 48:533-554. (sm1)
2. Anderson, E.G. 1953. MNL 27:5. (bf2)
3. Anderson, E.G. 1955. MNL 29:5. (gl15)
4. Anderson, E.G. 1955. MNL 29:6. (gl18)
5. Anderson, E.G. and R.A. Emerson. 1931. Amer. Nat. 65:253-257. (Ch1)
6. Ashman, R.B. 1960. Genetics 45:19-34. (Mst1)
7. Ashman, R.B. and A.J. Ullstrup. 1976. J. Hered. 67:220-222. (Les3)

8. Bachmann, M.D., D.S. Robertson and C.C. Bowen. 1973. *J. Ultrastruct. Res.* 45:384-406. (L11)
9. Baum, J.A. and J.G. Scandalios. 1982. *MNL* 56:136-138. (Sod1 Sod4)
10. Beadle, G.W. 1929. *Amer. Nat.* 63:189-192. (ys1)
11. Beadle, G.W. 1931. *Cornell Univ. Agr. Exp. Sta. Memoir* 135:1-12. (po1)
12. Beadle, G.W. 1932. *Genetics* 17:413-431. (ms5 ms7 ms8 ms9 ms10 ms11 ms12 ms13 ms14 ra1)
13. Beadle, G.W. 1932. *Ztschr. induktive Abstam. U. Vererbungsl.* 63:195-217. (st1)
14. Beadle, G.W. 1932. *Cytologia* 3:142-155. (va1)
15. Beadle, G.W. and B. McClintock. 1928. *Science* 68:433. (as1)
16. Beckett, J.B. 1971. *Crop Science* 11:724-727. (cms-C)
17. Beckett, J.B. and M.G. Neuffer. 1973. *MNL* 47:147. (v21)
18. Beckman, L., J.G. Scandalios and J.L. Brewbaker. 1964. *Science* 146:1174-1175. (Cat1)
19. Bray, R.A. 1964. *MNL* 38:134. (Lc1)
20. Brewbaker, J.L. 1974. *Proc. 29th Ann. Corn and Sorghum Res. Conf.*, pp.118-133. (Mv1)
21. Brewbaker, J.L. and Y. Hasegawa. 1974. *MNL* 48:35-37. (Px8 Px9)
22. Brink, R.A. 1933. *J. Hered.* 24:325-326. (Lg2)
23. Brink, R.A. 1935. *J. Hered.* 26:249-251. (pm1)
24. Brink, R.A. and R.A. Nilan. 1952. *Genetics* 37:519-544. (Mp)
25. Brink, R.A. and P.H. Senn. 1931. *J. Hered.* 22:155-161. (Rg1)
26. Brown, A.H.D. and R.W. Allard. 1969. *Crop Science* 9:72-75. (E12 E16)
27. Bryan, A.A. and J.E. Sass. 1941. *J. Hered.* 32:342-346. (Kn1)
28. Buchert, J.G. 1961. *Proc. Nat. Acad. Sci.* 47:1436-1440. (Rf3)
29. Burnham, C.R. 1936. *J. Amer. Soc. Agron.* 28:968-975. (ga2)
30. Burnham, C.R. 1947. *MNL* 21:36. (bm4)
31. Burnham, C.R. 1958. *MNL* 32:93. (eg1)
32. Burnham, C.R. 1961. *MNL* 35:87. (tb1)
33. Burnham, C.R. and R.A. Brink. 1932. *J. Amer. Soc. Agron.* 24:960-963. (bm2)
34. Chang, S-H. and J.L. Brewbaker. 1976. *MNL* 50:31-32. (aph1)
35. Chao, S.E. and J.G. Scandalios. 1969. *Biochem. Genet.* 3:537-547. (Amy2)
36. Chao, S.E. and J.G. Scandalios. 1971. *Genetics* 69:47-61. (Amy1)
37. Chourey, P.S. and C. Mouli. 1975. *Genetics* 77:s11. (Ce1)
38. Clark, F.J. 1940. *Amer. J. Bot.* 27:547-559. (dv1)
39. Coe, E.H., Jr. 1958. *MNL* 32:102. (c2)
40. Coe, E.H., S.M. McCormick and S.A. Modena. 1981. *J. Hered.* 72:318-320. (whp1)
41. Collins, G.N. 1909. *U.S. Dept. Agr., Pl. Indus. Bur. Bull.* 161:1-30. (wx1)
42. Collins, G.N. 1917. *J. Agr. Res.* 9:383-395. (Tu1)
43. Collins, G.N. 1917. *Proc. Nat. Acad. Sci.* 3:345-349. (Tu1)
44. Collins, G.N. and J.H. Kempton. 1920. *J. Hered.* 11:3-6. (Li1)
45. Correns, C. 1901. *Bibliotheca Botanica* 53:1-161. (su1 Y1)
46. Couture, R.M., D.G. Routley and G.M. Dunn. 1971. *Phys. Pl. Path.* 1:515-521. (Bx1)
47. Cox, E.L. and D.B. Dickinson. 1971. *Biochem. Genet.* 5:15-25. (L12 w14 w15)
48. de la Roche, I.A., D.E. Alexander and E.J. Weber. 1971. *Crop Science* 11:856-859. (Ln1)
49. Demerec, M. 1921. *J. Hered.* 12:406-407. (zb1)
50. Demerec, M. 1924. *Cornell Univ. Agr. Exp. Sta. Memoir* 84. (v1 v3 v4 v5)
51. Demerec, M. 1926. *J. Hered.* 17:301-306. (pb1 pb4)
52. Demerec, M. 1926. *Amer. Nat.* 60:172-176. (d3 v8 w11)
53. Dempsey, E. 1971. *MNL* 45:58. (K3L)
54. Doerschug, E.B. 1973. *Theor. Appl. Genet.* 43:182-189. (Dt4 Dt5)
55. Dollinger, E.J. 1980. *MNL* 54:124. (ora3)
56. Dooner, H.K. and J.L. Kermicle. 1971. *Genetics* 67:427-436. (I)
57. Doyle, G.G. 1978. *MNL* 52:77. (agt1)
58. Duvick, D.N. 1965. *Adv. Genet.* 13:1-56. (Rf2)
59. East, E.M. and H.K. Hayes. 1911. *Conn. Agr. Exp. Sta. Bull.* 167. (C1 pr1 R1)
60. Efron, Y. 1970. *Genetics* 65:575-583. (Ap1 Ap2 Ap3)
61. Emerson, R.A. 1911. *Nebr. Agr. Exp. Sta. Ann. Rept.* 24:59-90. (P1)
62. Emerson, R.A. 1912. *Amer. Breeders Assoc. Ann. Rept.* 8:385-399. (an1 d1 g1 Lg1 w1)
63. Emerson, R.A. 1912. *Nebr. Agr. Exp. Sta. Ann. Rept.* 25:81-88. (Lg1 w1)
64. Emerson, R.A. 1912. *Nebr. Agr. Exp. Sta. Ann. Rept.* 25:89-105. (g1 j1 v2)
65. Emerson, R.A. 1918. *Cornell Univ. Agr. Exp. Sta. Memoir* 16. (a1)
66. Emerson, R.A. 1920. *J. Hered.* 11:65-76. (ts1 ts2)
67. Emerson, R.A. 1921. *Cornell Univ. Agr. Exp. Sta. Memoir* 39. (B1 PL1)
68. Emerson, R.A. 1921. *Amer. J. Bot.* 8:411-424. (Bh1 gs1)
69. Emerson, R.A. 1921. *J. Hered.* 12:267-270. (cr1)
70. Emerson, R.A. 1932. *Science* 75:566. (ms17)
71. Emerson, R.A. 1932. *Sixth Int. Congress Genet. Proc.* 1:141-152. (Ts5)
72. Emerson, R.A. and S.H. Emerson. 1922. *Genetics* 7:203-236. (an1)
73. Everett, H.L. 1949. *Proc. Nat. Acad. Sci.* 35:628-634. (cl1 Clm1)
74. Eyster, W.H. 1926. *Science* 64:22. (bm1 yg1)
75. Eyster, W.H. 1929. *Ztschr. induktive Abstam. U. Vererbungsl.* 49:105-130. (ar1)
76. Eyster, W.H. 1931. *J. Hered.* 22:99-102. (ms2 ms3)

77. Eyster, W.H. 1931. *Genetics* 16:574-590. (vp1 vp2)
78. Eyster, W.H. 1933. *Amer. Nat.* 67:75. (oy1)
79. Eyster, W.H. 1934. *Bibliographia Genetica* 11:187-392. (da1 l7 ms2 ms3 su2)
80. Faludi-Daniel, A., F. Lang, A. Nagy and B. Faludi. 1967. *Acta Agron. Acad. Sci. Hung.* 16:1-6. (ly1 z1)
81. Ferguson, J.E., A.M. Rhodes and D.B. Dickinson. 1978. *J. Hered.* 69:377-380. (se1)
82. Fraser, A.C. 1924. *J. Hered.* 15:119-123. (in1)
83. Fraser, A.C. 1933. *J. Hered.* 24:41-46. (si1)
84. Freeling, M. and J.C. Woodman. 1978. *MNL* 52:9-10. (Cdh1)
85. Friedemann, P.D. and P.A. Peterson. 1981. *MNL* 55:6-7. (Ruq1 Uq1)
86. Galinat, W.C. 1969. *Mass. Agr. Exp. Sta. Bull.* 577:1-19. (Sg1)
87. Galinat, W.C. 1971. *MNL* 45:98-99. (is1)
88. Galinat, W.C. 1975. *MNL* 49:100-102. (Ph1 Ri1)
89. Galinat, W.C., P. Chandravadvava and J. Starbuck. 1978. *MNL* 52:58. (bu1)
90. Galinat, W.C. and P.C. Mangelsdorf. 1957. *MNL* 31:67. (Pn1)
91. Gavazzi, G., M. Nava-Racchi and C. Tonelli. 1975. *Theor. Appl. Genet.* 46:339-346. (pro1)
92. Gelinat, D.A., S.N. Postlethwait and L.F. Bauman. 1966. *Amer. J. Bot.* 53:615. (Cl1)
93. Gernert, W.B. 1912. *Amer. Nat.* 46:616-622. (ra1)
94. Giesbrecht, J. 1965. *J. Hered.* 56:118,130. (zn2)
95. Glover, D.V. 1970. *Crop Science* 10:611-612. (rd2)
96. Glover, D.V. 1968. *MNL* 42:151. (ct2 sr3)
97. Golubovskaya, I.N. and A.S. Mashnenkov. 1975. *Genetika* 11:11-17. (afd1)
98. Golubovskaya, I.N. and A.S. Mashnenkov. 1976. *Genetika* 12:7-14. (dsy1)
99. Golubovskaya, I.N. and A.S. Mashnenkov. 1977. *Genetika* 13:1910-1921. (pam1)
100. Golubovskaya, I.N. and V.G. Urbach. 1981. *MNL* 55:80-81. (dsy2 pam2)
101. Gonella, J.A. and P.A. Peterson. 1975. *MNL* 49:71-73. (ga10)
102. Gonella, J.A. and P.A. Peterson. 1977. *Genetics* 85:629-645. (Fcu)
103. Goodman, M.M. and C.W. Stuber. 1982. *MNL* 56:125. (Gdh2)
104. Goodman, M.M., C.W. Stuber, K. Newton and H.H. Weissinger. 1980. *Genetics* 96:697-700. (Got2 Got3 Idh1 Idh2 Me1 Pgd1 Pgd2 Pgm1 Pgm2 Phi1)
105. Gracen, V.E., A. Kheyr-Pour, E.D. Earle and P. Gregory. 1979. *Proc. 34th Ann. Corn and Sorghum Res. Conf.*, pp.76-91. (Rf4)
106. Hamill, D.E. 1968. *MNL* 42:36-37. (Px1)
107. Harovitz, S. 1948. *MNL* 22:42. (zn1)
108. Harris, J.W. 1968. *Genetics* 60:186-187. (E4)
109. Hayes, H.K. 1932. *J. Hered.* 23:415-419. (zb4)
110. Hayes, H.K. and H.E. Brewbaker. 1928. *Amer. Nat.* 62:228-235. (gl2 gl3 sl1)
111. Hayes, H.K. and M.S. Chang. 1938. *MNL* 12:8. (zb6)
112. Hayes, H.K. and E.M. East. 1915. *Conn. Agr. Exp. Sta. Bull.* 188:1-31. (fl1)
113. Hofmeyr, J.D.J. 1930. Unpub. thesis, Cornell. (ba1 ba2)
114. Hooker, A.L. 1963. *Crop Science* 3:381-383. (Ht1)
115. Hooker, A.L. 1977. *Crop Science* 17:132-135. (Ht2)
116. Hooker, A.L. 1981. *MNL* 55:87-88. (Ht3)
117. Hutchison, C.B. 1921. *J. Hered.* 12:76-83. (sh1)
118. Hutchison, C.B. 1922. *Cornell Univ. Agr. Exp. Sta. Memoir* 60:1419-1473. (na1)
119. Jenkins, M.T. 1924. *J. Hered.* 15:467-472. (ij1)
120. Jenkins, M.T. 1926. *Amer. Nat.* 60:484-488. (g2)
121. Jenkins, M.T. 1927. *Genetics* 12:492-518. (yg2)
122. Jenkins, M.T. 1930. *J. Hered.* 21:79-80. (rt1)
123. Jenkins, M.T. 1932. *J. Agr. Res.* 44:495-502. (a2)
124. Jenkins, M.T. 1947. *MNL* 21:33. (y8)
125. Jenkins, M.T. and M.A. Bell. 1930. *Genetics* 15:253-282. (l4)
126. Jenkins, M.T. and F. Gerhardt. 1931. *Iowa Agr. Exp. Sta. Res. Bull.* 138:121-151. (la1)
127. Joachim, G. and C.R. Burnham. 1953. *MNL* 27:66. (sr2)
128. Jones, D.F. 1925. *J. Hered.* 16:339-341. (sk1)
129. Jones, D.F. 1951. *Proc. Nat. Acad. Sci.* 37:408-410. (Rf1)
130. Jones, D.F. 1954. *Proc. IX Int. Genet. Cong.* 1225-1237. (cms-S cms-T)
131. Jones, D.F. and P.C. Mangelsdorf. 1925. *Anat. Rec.* 31:351. (Ga1)
132. Josephson, L.M. 1955. *Empire J. Exp. Agr.* 23(89):1-10. (cms-S cms-T)
133. Kang, M.S. 1981. *MNL* 55:26. (btn1)
134. Kempton, J.H. 1920. *J. Hered.* 11:111-115. (br1)
135. Kempton, J.H. 1920. *J. Hered.* 11:317-322. (ad1)
136. Kempton, J.H. 1921. *U.S. Dept. Agr. Bull.* 925:1-28. (br1)
137. Kempton, J.H. 1921. *Amer. Nat.* 56:461-464. (ws1 ws2)
138. Kempton, J.H. 1934. *J. Hered.* 25:29-32. (bd1)
139. Kermicle, J.L. 1969. *Science* 166:1422-1424. (ig1)
140. Kermicle, J.L. and J.D. Axtell. 1981. *Maydica* 26:185-197. (Isr1)
141. Khadzhinov, M.I. 1937. *Bull. Appl. Bot. Gen. Pl. Breed. Ser. II* 7:247-258. (Rs1 rs2)
142. Kvakon, P. 1924. *Cornell Univ. Agr. Exp. Sta. Memoir* 83:1-22. (Bn1 gl1)
143. Kramer, H.H. 1957. *MNL* 31:120. (rgd1)

144. Kulkarni, C.G. 1927. Mich. Acad. Sci. Arts and Letters Papers 6:253-273. (Wc1)
145. Lai, Y-K. and J.G. Scandalios. 1980. Devel. Genet. 1:311-324. (Adr1)
146. Langham, D.G. 1940. Genetics 25:88-107. (pd1 tr1)
147. Langham, D.G. 1940. MNL 14:21. (bk2)
148. Laughnan, J.R. 1949. Proc. Nat. Acad. Sci. 35:167-178. (alpha beta)
149. Leng, E.R. and L.F. Bauman. 1955. Agron. J. 47:189-191. (Ms21 Sks1)
150. Leng, E.R. and M.L. Vineyard. 1951. MNL 25:31-32. (br2)
151. Levings, C.S. III and C.W. Stuber. 1971. Genetics 69:491-498. (fv1)
152. Li, H.W. 1931. J. Hered. 22:14-16. (bv1)
153. Li, H.W. 1937. J. Hered. 24:279-281. (na1)
154. Lindstrom, E.W. 1917. Amer. Nat. 51:225-237. (l1)
155. Lindstrom, E.W. 1918. Cornell Univ. Agr. Exp. Sta. Memoir 13:1-68. (l1 w1)
156. Lindstrom, E.W. 1921. Genetics 6:91-110. (f1)
157. Lindstrom, E.W. 1924. Genetics 9:305-326. (w2 w3)
158. Lindstrom, E.W. 1925. J. Hered. 16:135-140. (Tp1)
159. Lindstrom, E.W. 1935. Iowa St. Coll. J. Sci. 9:451-459. (a3 Og1)
160. Lock, R.H. 1906. Roy. Bot. Gard. Annals 3:95-184. (P1)
161. Longley, A.E. 1932. J. Agr. Res. 54:835-862. (K10)
162. Lorenzoni, C., M. Pozzi and F. Salamini. 1974. MNL 48:19-20. (cp1)
163. Lowe, J. and O.E. Nelson, Jr. 1946. Genetics 31:525-533. (mn1)
164. MacDonald, T. and J.L. Brewbaker. 1972. J. Hered. 63:11-14. (Px2 Px3 Px4 Px5 Px6 Px7 Ta1)
165. MacDonald, T. and J.L. Brewbaker. 1974. J. Hered. 65:37-42. (E5-I E5-II E6 E7 E8 E9 E10)
166. Mains, E.B. 1926. J. Hered. 17:313-325. (Rp1)
167. Mains, E.B. 1931. J. Agr. Res. 43:419-430. (Rp1)
168. Mains, E.B. 1949. J. Hered. 40:21-24. (sh2)
169. Mangelsdorf, P.C. 1926. Conn. Agr. Exp. Sta. Bull. 279:509-614. (bt1)
170. Mascia, P.N. and D.S. Robertson. 1980. J. Hered. 71:19-24. (l13 nec1 oro1 oro2 Orom1)
171. Mashnenkov, A.S. and M.I. Khadjinov. 1979. Proc. IX Eucarpia Corn and Sorghum Sect., pp.447-450. (Sup1)
172. McClintock, B. 1934. Z. Zellforsch. Mikrosk. Anat. 21:294-328. (NOR)
173. McClintock, B. 1944. Genetics 29:478-502. (pyd1 wd1)
174. McClintock, B. 1950. Proc. Nat. Acad. Sci. 36:344-355. (Ac1 Ds)
175. McClintock, B. 1956. Brookhaven Symp. Biol. 8:58-74. (Spm)
176. Melville, J.C. and J.G. Scandalios. 1972. Biochem. Genet. 7:15-31. (Enp1)
177. Micu, V. 1980. MNL 54:63-64. (db1)
178. Micu, V. 1981. Genetical Studies of Maize. Shtiintsa, Kishinev, Mold. SSR. (bs1 db1 tl1)
179. Micu, V. and S.I. Mustyatsa. 1978. Genetika 14:365-368. (dep1)
180. Miles, C.D. and D.J. Daniel. 1974. Pl. Phys. 53:589-595. (hcf1 hcf2 hcf3)
181. Miles, F.C. 1915. J. Genetics 4:193-214. (gs1)
182. Miranda, L.T. de. 1980. MNL 54:19. (Asr1)
183. Miranda, L.T. de. 1980. MNL 54:15-18. (mal1 Mal2)
184. Miranda, L.T. de. 1981. MNL 55:18-19. (lte1)
185. Miranda, L.T. de, L.E.C. deMiranda, E. Sawazaki and N.C. Schmidt. 1982. MNL 56:28-30. (Lte2)
186. Miranda, L.T. de, C.J. Rosseta, E. Sawazaki and N.C. Schmidt. 1982. MNL 56:30-32. (Mer1 Mer2 Zer1 Zer2 Zer3 Zer4)
187. Misra, P.S., R. Jambunathan, E.T. Mertz, D.V. Glover, H.M. Barbosa and K.S. McWhirter. 1972. Science 176:1425-1427. (o7)
188. Mumm, W.J. 1929. Anat. Rec. 44:279. (h1)
189. Nelson, O.E. 1972. MNL 46:203. (o6)
190. Nelson, O.E. 1976. MNL 50:114. (fl3)
191. Nelson, O.E. 1981. MNL 55:68. (o9 o10 o11 o12 o13)
192. Nelson, O.E. 1981. MNL 55:73. (lp1)
193. Nelson, O.E. and G.B. Clary. 1952. J. Hered. 43:205-210. (dy1 lo2)
194. Nelson, O.E. and A.J. Ohlrogge. 1957. Science 125:1200. (ct1 rd1)
195. Nelson, O.E. and S.N. Postlethwait. 1954. Amer. J. Bot. 41:739-748. (Pt1)
196. Nelson, O.E. and A.J. Ullstrup. 1964. J. Hered. 55:194-199. (hm2)
197. Neuffer, M.G. 1973. MNL 47:150. (nec3)
198. Neuffer, M.G. and O.H. Calvert. 1975. J. Hered. 66:265-270. (Les1 Les2)
199. Neuffer, M.G., L. Jones and M.S. Zuber. 1968. The mutants of maize. Crop Sci. Soc. Amer., Madison, Wis.. (cp2 ub1)
200. Neuffer, M.G. and K.A. Sheridan. 1977. MNL 51:60. (Atc1 Bif1 Rgd2 Spc1 Ysk1)
201. Neuffer, M.G. and W.F. Sheridan. 1980. Genetics 95:929-944. (dek1 dek2 dek3 dek4 dek5 dek6 dek7 dek8 dek9 dek10 dek11 dek12 dek13 dek14 dek15)
202. Neuffer, M.G. and W.F. Sheridan. 1980. MNL 55:29-30. (dek1 dek2 dek3 dek4 dek5 dek6 dek7 dek8 dek9 dek10 dek11 dek12 dek13 dek14 dek15)
203. Newton, K.J. and D. Schwartz. 1980. Genetics 95:425-442. (Mdh1 Mdh2 Mdh3 Mdh4 Mdh5 mmm1)
204. Nickerson, N.H. and E.E. Dale. 1955. Ann. Mo. Bot. Gard. 42:195-212. (Ts6)
205. Nuffer, M.G. 1954. MNL 28:63-64. (bz2)
206. Nuffer, M.G. 1955. Science 121:399-400. (Dt2 Dt3)
207. Nuffer, M.G. 1959. MNL 33:82. (Mr)

208. Ott, L. and J.G. Scandalios. 1978. *Genetics* 89:137-146. (Amp1 Amp2 Amp3 Amp4)
209. Paliy, A.F. and A.I. Rotar. 1979. *Genetika* 15:478-481. (cfl2)
210. Palmer, R.G. 1971. *Chromosoma* 35:233-246. (am1)
211. Pawar, S.E. and C. Mouli. 1973. *MNL* 47:17. (Ce1)
212. Perry, H.S. 1939. *MNL* 13:7. (Lg3)
213. Perry, H.S. and G.F. Sprague. 1936. *J. Amer. Soc. Agron.* 28:990-996. (Y3)
214. Peterson, H. 1959. *MNL* 33:41. (Tp2)
215. Peterson, P.A. 1960. *Genetics* 45:115-133. (En pg14)
216. Pfund, J.H. and C.W. Crum. 1977. *Agron. Abst.*, p.66. (Thc1)
217. Phinney, B.O. 1956. *Proc. Nat. Acad. Sci.* 42:185-189. (D8)
218. Phipps, I.F. 1928. *J. Hered.* 19:399-404. (ts4)
219. Phipps, I.F. 1929. *Cornell Univ. Agr. Exp. Sta. Memoir* 125:1-63. (al1 v12 v13 v16 v17 v18)
220. Piovarci, A. 1982. *MNL* 56:157. (bv2)
221. Plewa, M. 1979. *MNL* 53:93-96. (loc1)
222. Pogna, N.E., A. Ghidoni and P. Ranalli. 1982. *MNL* 56:153. (ys2)
223. Postlethwait, S.N. and O.E. Nelson, Jr. 1957. *Amer. J. Bot.* 44:628-633. (wi1)
224. Pryor, A.J. 1974. *Heredity* 32:397-401. (Gdh1)
225. Pryor, A.J. 1976. *MNL* 50:15-16. (Glu1)
226. Pryor, T. and D. Schwartz. 1973. *Genetics* 75:75-92. (Cx1)
227. Randolph, L.F. 1928. *Anat. Rec.* 41:102. (B chr)
228. Rhoades, M.M. 1935. *Amer. Nat.* 69:74-75. (Dt1)
229. Rhoades, M.M. 1939. *Genetics* 24:62. (ws3)
230. Rhoades, M.M. 1948. *MNL* 22:9. (ga7)
231. Rhoades, M.M. 1951. *Amer. Nat.* 85:105-10. (pg11 pg12)
232. Rhoades, M.M. 1952. *Amer. Nat.* 86:105-106. (bz1)
233. Rhoades, M.M. 1956. *MNL* 30:38-42. (am1 el1)
234. Rhoades, M.M. and E. Dempsey. 1954. *MNL* 28:58. (gl17)
235. Rhoades, M.M. and E. Dempsey. 1982. *MNL* 56:21. (Mrh)
236. Rhoades, M.M. and E. Dempsey. 1982. *MNL* 56:22. (Ac2 Mut)
237. Robertson, D.S. 1952. *Proc. Nat. Acad. Sci.* 38:580-583. (vp5)
238. Robertson, D.S. 1955. *Genetics* 40:745-760. (vp8 vp9)
239. Robertson, D.S. 1961. *Genetics* 46:649-662. (y10)
240. Robertson, D.S. 1967. *MNL* 41:94. (o5)
241. Robertson, D.S. 1973. *MNL* 47:82. (L10)
242. Robertson, D.S. 1974. *MNL* 48:70. (yd2)
243. Robertson, D.S. 1975. *J. Hered.* 66:127-130. (y9)
244. Robertson, D.S. 1978. *Mutat. Res.* 51:21-28. (Mu)
245. Robertson, D.S. 1981. *MNL* 55:115. (L15)
246. Sachan, J.K. and K.R. Sarkar. 1978. *MNL* 52:119-120. (rgo1)
247. Sarvella, P. and C.O. Grogan. 1966. *J. Hered.* 57:211-212. (te1)
248. Saxena, K.M.S. and A.L. Hooker. 1968. *Proc. Nat. Acad. Sci.* 61:1300-1305. (Rp5)
249. Scandalios, J.G., D.Y. Chang, D.E. McMillin, A. Tsiftaris and R.H. Moll. 1980. *Proc. Nat. Acad. Sci.* 77:5360-5364. (Car1)
250. Scandalios, J.G., E.H. Liu and M.A. Campeau. 1972. *Arch. Biochem. Biophys.* 153:695-705. (Cat2)
251. Scandalios, J.G., J.C. Sorenson and L.A. Ott. 1975. *Biochem. Genet.* 13:759-769. (Got1)
252. Scandalios, J.G., W.F. Tong and D.G. Roupakia. 1980. *Mol. Gen. Genet.* 179:33-41. (Cat3)
253. Schwartz, D. 1951. *Genetics* 36:676-696. (Ms21 Sks1)
254. Schwartz, D. 1951. *MNL* 25:30. (ga8)
255. Schwartz, D. 1960. *Proc. Nat. Acad. Sci.* 46:1210-1215. (E1)
256. Schwartz, D. 1964. *Proc. Nat. Acad. Sci.* 51:602-605. (E3)
257. Schwartz, D. 1965. *Proc. XI Int. Genet. Cong.* 2:131-135. (E2)
258. Schwartz, D. 1966. *Proc. Nat. Acad. Sci.* 56:1431-1436. (Adh2)
259. Schwartz, D. 1979. *Mol. Gen. Genet.* 174:232-241. (mep1 Prot1)
260. Schwartz, D. and T. Endo. 1966. *Genetics* 53:709-715. (Adh1)
261. Shaver, D.L. 1967. *J. Hered.* 58:270-273. (gt1 pe1)
262. Shortess, D.K., J.E. Wright and W.D. Bell. 1968. *Genetics* 58:227-235. (Lu1)
263. Shumway, L.K. and L.F. Bauman. 1967. *Genetics* 55:33-38. (NCS)
264. Singleton, W.R. 1946. *J. Hered.* 37:61-64. (id1)
265. Singleton, W.R. 1951. *Amer. Nat.* 85:81-96. (Cg1)
266. Singleton, W.R. 1959. *MNL* 33:3. (br3)
267. Singleton, W.R. and D.F. Jones. 1930. *J. Hered.* 21:266-268. (ms1)
268. Smith, D.R. and A.L. Hooker. 1973. *Crop Science* 13:330-331. (rh1)
269. Soave, C., N. Suman, A. Viotti and F. Salamini. 1978. *Theor. Appl. Genet.* 52:263-267. (Zp1 Zp2 Zp3 Zp6)
270. Soave, C., R. Reggiani, N. DiFonzo and F. Salamini. 1981. *Genetics* 97:363-377. (Zp16 Zp21 Zp29)
271. Sprague, G.F. 1932. *U.S. Tech. Bull.* 292:1-43. (sy1)
272. Sprague, G.F. 1936. *J. Amer. Soc. Agron.* 28:472-478. (ps1)
273. Sprague, G.F. 1938. *MNL* 12:2. (gl11)
274. Sprague, G.F. 1939. *J. Hered.* 30:143-145. (Vg1)
275. Sprague, G.F. 1955. *MNL* 29:6. (gl14)

276. Sprague, G.F. and H.H. McKinney. 1966. *Genetics* 54:1287-1296. (AR)
 277. Sprague, G.F., H.H. McKinney and L. Greeley. 1965. *MNL* 39:164. (wt1)
 278. Stadler, L.J. 1940. *MNL* 14:26. (et1)
 279. Stadler, L.J. and M.H. Emmerling. 1956. *Genetics* 41:124-137. (P S)
 280. Stadler, L.J. and M.G. Nuffer. 1953. *Science* 117:471-472. (P)
 281. Stierwalt, T.R. and P.L. Crane. 1974. *MNL* 48:139. (sen1 sen2 sen3 sen4 sen5 sen6)
 282. Stout, J.T. and R.L. Phillips. 1973. *Proc. Nat. Acad. Sci.* 70:3043-3047. (alh1 clh1)
 283. Stroman, G.N. 1924. *Genetics* 9:493-512. (zb2)
 284. Stroup, D. 1970. *J. Hered.* 61:139-141. (cm1)
 285. Styles, E.D., R.A. Brink and K.T. Seah. 1973. *Can. J. Genet. Cytol.* 15:59-72. (R2)
 286. Suttle, A.D. 1924. Unpub. thesis, Cornell. (d2 d5 py1)
 287. Tavcar, A. 1932. *Jugoslav. Akad. Znanosti i Umjetnosti Prestampo* 244:74-93. (Hs1)
 288. Teas, H.J. and E.G. Anderson. 1951. *Proc. Nat. Acad. Sci.* 37:645-649. (Bf1)
 289. Tsai, C. and O.E. Nelson. 1968. *Genetics* 61:813-821. (sh4)
 290. Tulpule, S.H. 1954. *Amer. J. Bot.* 41:294-301. (lw1 lw2 lw3 lw4)
 291. Ullstrup, A.J. 1965. *Phytopath.* 55:425-428. (Rpp9)
 292. Ullstrup, A.J. and A.M. Brunson. 1947. *J. Amer. Soc. Agron.* 39:606-609. (hm1)
 293. Ullstrup, A.J. and A.F. Troyer. 1968. *Phytopath.* 57:1252-1283. (Lls1)
 294. Vahrusheva, E.I. 1975. *MNL* 49:95-96. (cto1)
 295. Vineyard, M.L. and R.P. Bear. 1952. *MNL* 26:5. (ae1)
 296. Wentz, J.B. 1926. *J. Hered.* 17:327-329. (bt1)
 297. Wilkinson, D.R. and A.L. Hooker. 1968. *Phytopath.* 58:605-608. (Rp3 Rp4 Rp6)
 298. Wright, J.E. 1961. *MNL* 35:111. (ys3)

[The linkage map follows on page 191, after a brief piece of injeanuity
 by Paul Clark]

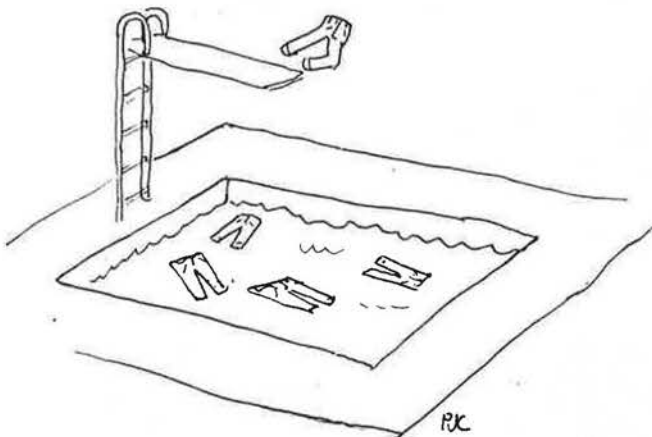


Figure 0
 A Jean Pool

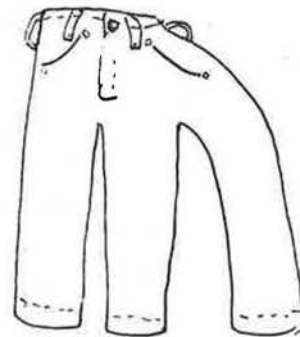


Figure 1
 Mutant. Jeans

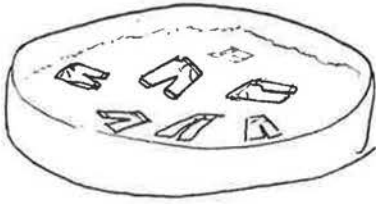


Figure 2.
Cloned Jeans

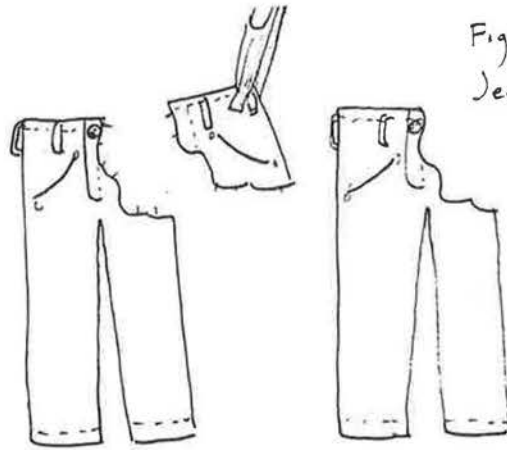


Figure 3
Jean Splicing

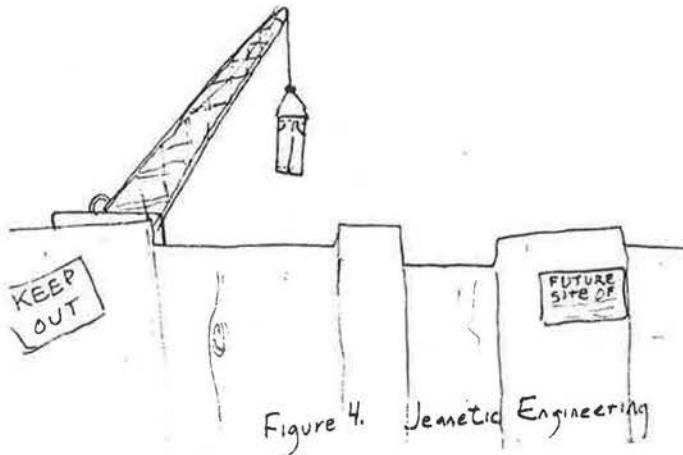


Figure 4. Genetic Engineering

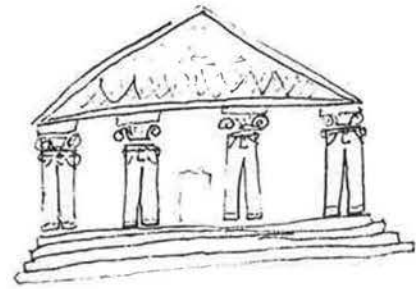


Figure 5
Structural Jeans

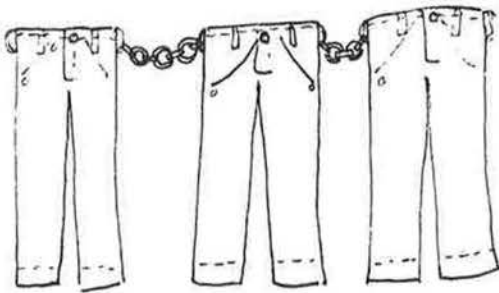


Figure 6
Linked Jeans

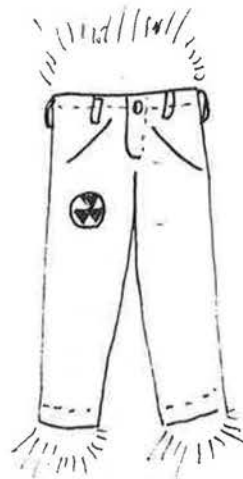


Figure 7
Nuclear Jeans

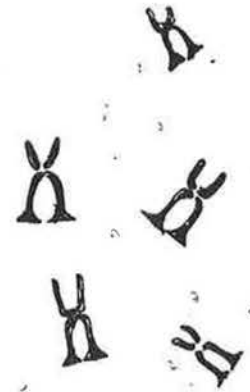
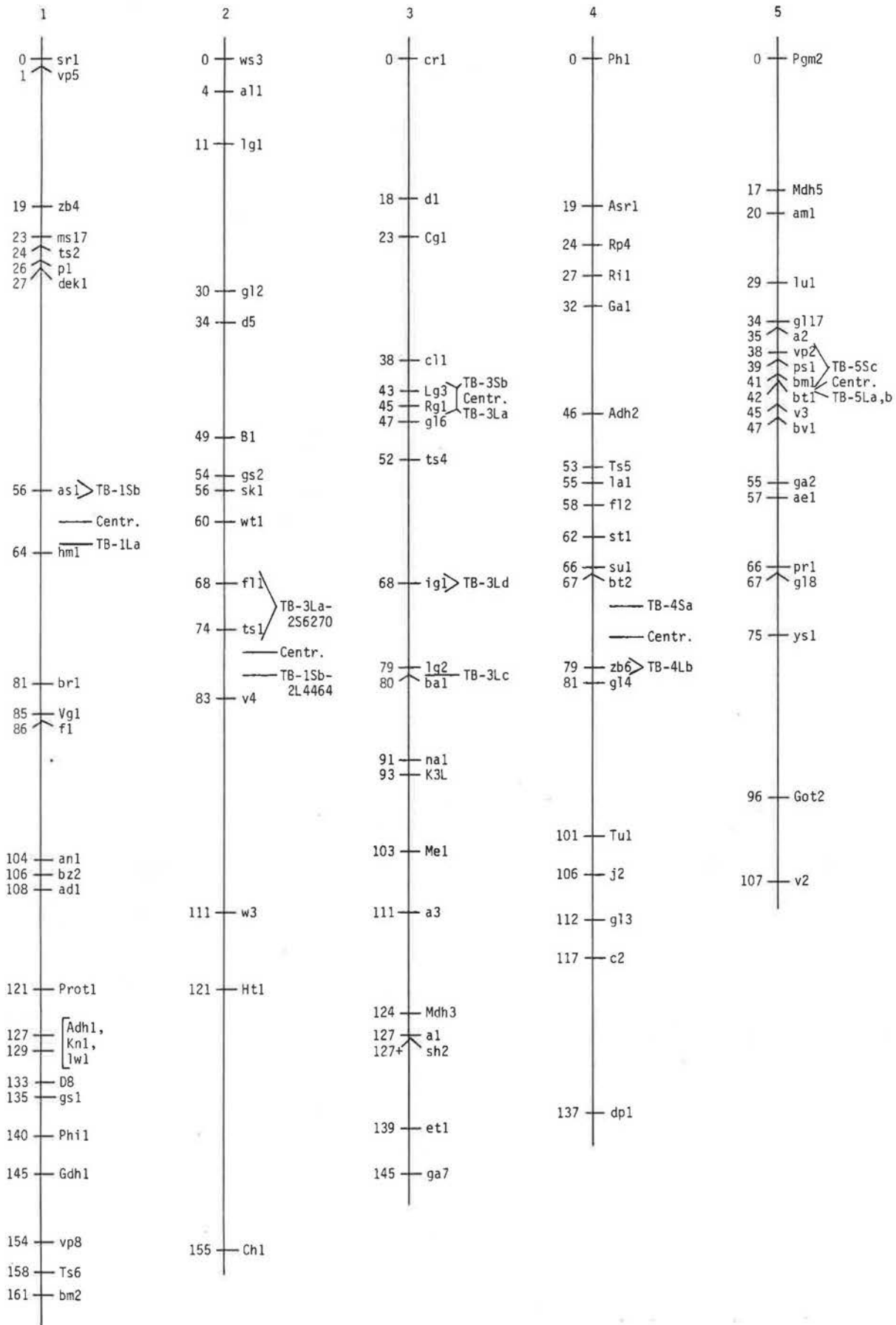


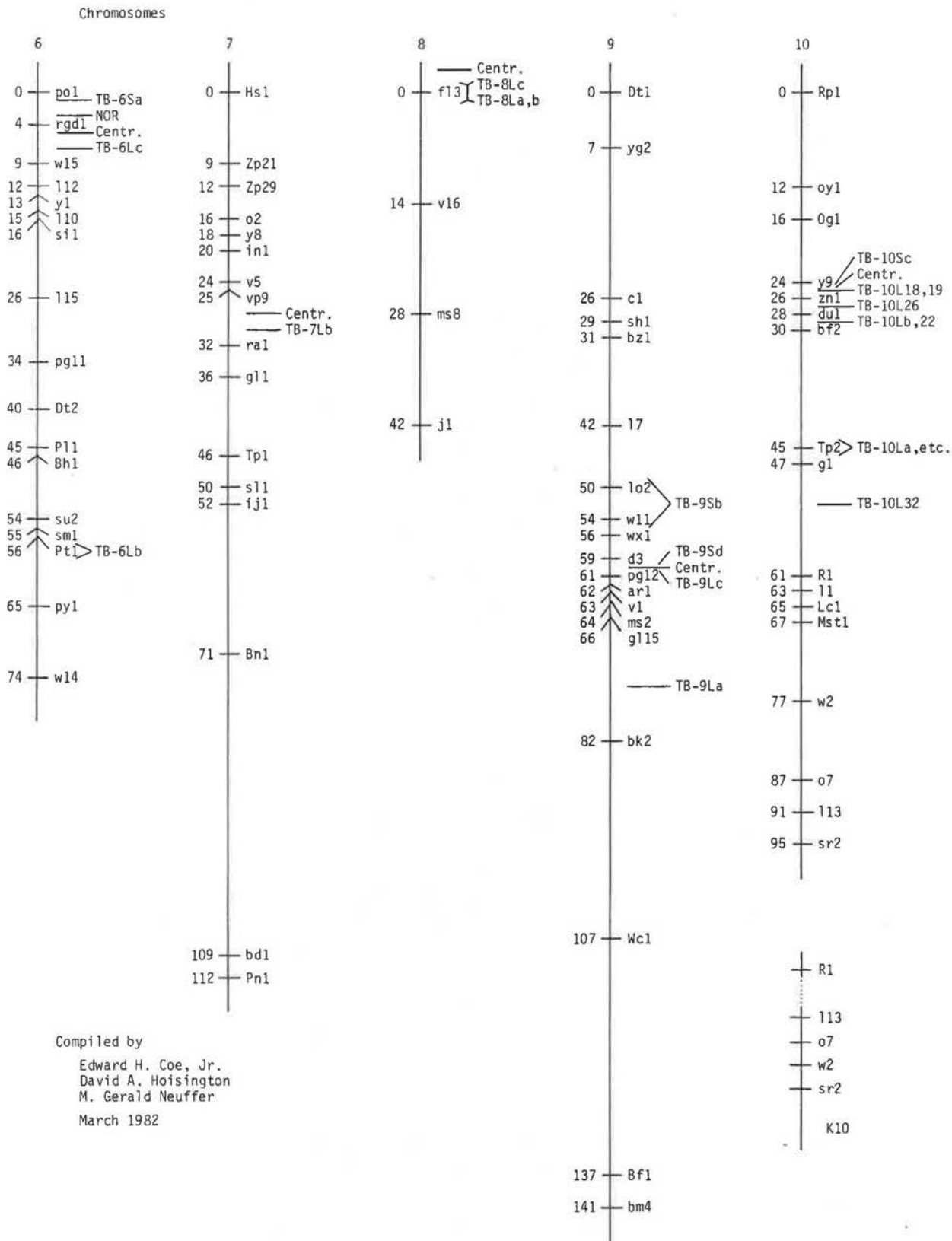
Figure 8
Bell Bottom Genes

LINKAGE MAP OF MAIZE

Chromosomes



LINKAGE MAP OF MAIZE (Continued)



V. FIFTY YEARS AGO IN OLD ZEALAND (MNL 3)

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NEW YORK STATE COLLEGE OF AGRICULTURE AT CORNELL UNIVERSITY
CORNELL UNIVERSITY AGRICULTURAL EXPERIMENT STATION
ITHACA, N. Y.

DEPARTMENT OF PLANT BREEDING

NEW YORK STATE COLLEGE OF AGRICULTURE AT CORNELL UNIVERSITY
CORNELL UNIVERSITY AGRICULTURAL EXPERIMENT STATION
ITHACA, N. Y.

DEPARTMENT OF PLANT BREEDING

EXHIBIT V

January 23, 1953

December 12, 1932

To Maize Geneticists :-

If you have any good tester combinations you wish to send in so that they may be made available for the whole group or if there is any combination of genes you would like to have, will you please notify us here at Cornell so that we may list your contributions and wants in the corn-letter which will come out in the near future. January 1st has been set as the dead line for receipt of material to be included in the letter. Will you please cooperate with us so that we can make this cooperative affair a real service to all concerned.

We plan to include in the letter a summary of the technic employed by the Russian physiologist, Lysenko, in his "Springefication" of corn.

If any of you have this year's linkage data which could be added to the linkage summary, we shall be glad to receive them at once. The summary is in preparation for publication.

Sincerely yours,

M. M. Rhoades

To maize geneticists :-

We are including in this report an inventory of all maize characters whose description has either been published or called to our attention. We are also including a summary of the technique employed by Lyssenko in his 'Jarovization' of corn. Denerec was kind enough to make the translation from the Russian.

The response of the maize geneticists to the two letters from this office asking for their cooperation in establishing a clearing house and central repository has been good. Either seed or the statement that certain stocks were available and would be sent later has been received from the following institutions: Wisconsin, Texas A. and M., Missouri, Carnegie Institution, U. S. Department of Agriculture, Connecticut Agricultural Experiment Station, California Institute of Technology, Minnesota, Ames, Bucknell and Cornell. A list of these stocks is included in this report.

The following wants have been received:

1. Related stocks homozygous for Ga and ga. Sprague.
2. A multiple recessive stock for each chromosome involving as great a map distance as possible with genes so situated as to reduce undetected double crossovers to a minimum. Sprague.
3. Variegated pericarp material from different sources. Whenever possible variegated/red cob white combination is preferable. Demerac.
4. Allelomorphs or suspected allelomorphs of k such as marbled, stippled, navajo, mottled, etc., and allelomorphs of R affecting plant characters. Stadler.
5. Multiple recessive combinations of genes in the pr-v₂ group. Rhoades.
6. Any recessive gene in the gl₁ v₅ group that is carrying dominant yellow endosperm. Hayes.
7. The combinations al-Y-Pl; ij-ra-gl₁; a₁-na₁ lg₁-gl₂-b; pr-bm₁ su-gl₃; Y-Pl pr-bm₁; P-f₁-an; p-f₁-an. Burnham.

8. Multiple seedling combinations for the same and different linkage groups; particularly new genes such as lg_2 , glossies, argostripe. Randolph.
9. The combination a_1 pr in with any glossy. Randolph.
10. Seedling genes in the Y-Pl group other than al and py . Randolph.

Recommendations concerning symbols for new characters:

Since approximately 290 different characters in maize have been described and assigned symbols it is becoming more and more difficult to find appropriate symbols, suggestive of the character, for new genes. Therefore, we recommend the following:

When a new character arises which is similar in its appearance to a previously described character it should be given the same symbol as that used for the old character except that the subscript, of course, shall be different. This has been done in the past, e.g. the different virescents, glossy seedlings, etc., but it has not been followed in all cases. As a concrete example of what we have in mind, we have different striped leaves described as fine streaked, fine striped, green striped, yellow striped, japonica, iojap, striate, etc. The number of genetically different striped characters will probably be great. Therefore, instead of trying to find a new symbol for a new stripe designate it as j_2 if it resembles japonica, or ys_2 if it resembles yellow stripe, etc. The same holds for the male steriles, dwarfs, etc. Unless we are willing to do this we shall be forced to use tri-literal symbols, or bi-literal symbols which in no way suggest the appearance of the character.

We strongly urge that you correspond with this office before assigning symbols to new characters. We shall keep the list of assigned symbols up to date so that we can be of assistance in assigning the proper symbols. The success of this project depends entirely upon your cooperation. There have been several instances in the past where the same symbol has been used for different genes. This is confusing not only to maize geneticists but to others.

Listed below are the best available multiple combinations of genes in each of the 10 chromosomes:

Some of these stocks have just been isolated and the supply of seed is limited. By next summer enough seed should be available for everybody having a legitimate use for the stocks. However an attempt will be made this spring to supply any of the listed stocks as long as the supply holds out.

Chromosome	Combination	Map distance covered by these factors	Total length of known genetic map
I	$p-br-f_1-bm_2$	125 ±	125 ±
II	$lg_1-gl_2-b-v_4$	80 ±	80 ±
III	$a_1-na-cr_1$	79 ±	79 ±
IV	$su-Tu-gl_3$	40 ±	70 ±
V	$ys-pr-bm_1$ $pr-bm_1-v_2$	30 ± 57 ±	87 ±
VI	$al-y-Pl-py$	69 ±	69 ±
VII	$Bn-gl_1-v_5$ $Bn-ra-v_5$	26 ± 26 ±	
VIII	$j-ms_8$	20 ±	27 ±
IX	$yg_2-c-sh-wx$	52 ±	96 ±
X	$r-g-nl$	33 ±	35 ±

Jarovization technique:

At the Sixth International Congress of Genetics, Professor Vavilov reported Lyssenko's discovery by which the growing period of plants can be appreciably shortened (jarovization). If the claims of the workers investigating this problem are justified, this discovery is of great importance to plant geneticists and to plant breeders.

Following is the description of the method worked out for maize and described in the Bulletin of Jarovization, 283: 105-108, 1932.

(1) Add water to increase the water content of the seed to 30 per cent of weight.

(2) Keep the seed in darkness for 10 to 15 days at a temperature of 20 to 30 centigrade and allow it to germinate. By regulating moisture the germination process should be controlled so that the germ does not develop excessively.

4.

The following stocks have been received:

- Brink - (1) $lg_1-ts_1-v_4$ x $lg_1-Ts_1-v_4$; (2) a_1-lg_2 ;
 (3) p-br-f-bm₂; (4) gl_2-fl-v_4 ;
 (5) $gl_2-ts_1-v_4$ x $gl_2-Ts_1-v_4$.
- Sprague - (1) r-g-nl; (2) $\frac{Ar^g}{a_1^g}$ B Pl su; (3) al-y-Pl;
 (4) Bm-gl₁-v₅; (5) $\frac{Pc^g}{a_1^g}$ Pc₁ Pc₂ Pc₃ Pc₄ - Pc = purple
 (6) bt₂ bt₂; (7) ACR so₁ so₂ - so = coleorrhiza;
 orange scutellum;
 (8) sy sy - sy = yellow scutellum;
 (9) Sx - scutellum color; (10) gl_1 ; (11) gl_2 ;
 (12) gl_3 ; (13) gl_4 ; (14) gl_5 ; (15) $gl_7 v_{17}$;
 (16) gl_8 ; (17) gl_9 .
- Beadle - (1) sr; (2) gs (early); (3) su-Tu- gl_3 .
- Demerec - (1) xn_2 ; (2) w_{11} ; (3) pg_1 ; (4) pg_4 ; (5) pg_3 ;
 (6) pb_1 ; (7) pb_2 and pb_3 (duplicate factors);
 (8) pb_4 ; (9) zebra₁; (10) zebra₂; (11) zebra₃.
- Stadler - (1) Y a R^g C pr in b pl; (2) a r C pr wx y;
 (3) p^{vv} A R^g c sh wx pr su;
 (4) A C r^g sh wx y pr Su su - r^g derived by
 mutation from R^g;
 (5) a C R^g pr in y wx Su su.
- Jenkins - (1) A₁ A₁ C C R R pr pr a₂ a₂ (Bt bt);
 (2) $gl_1 i_j YY$; (3) $gl_1 v_5$;
 (4) $gl_1 i_j YY$ seg. fr₁ and fr₂.
- Eyster - (1) g_3 ; (2) g_4 ; (3) pk; (4) l_6 ; (5) l_7 ; (6) l_5 ;
 (7) f_3 ; (8) su₂; (9) yt; (10) da; (11) ar; (12) sa₁;
 (13) au₁; (14) au₂; (15) cy; (16) ms₂; (17) ms₃;
 (18) vp₁; (19) ms₁₈; (20) cr₂; (21) ms₂₀; (22) bt₄;
 (23) pg₈.

5.

Mangelsdorf writes that he can furnish the following late stocks:

- (1) B b na na; (2) na g; (3) g; (4) Y y Pl pl;
 (5) lg gl_1 ra; (6) Pr Pr RR cc wx wx; (7) B b lg lg Sk sk;
 (8) pr pr RR CC su su; (9) Tu tu su su;
 (10) Tu tu Ts₅ ts₅ su su.

Kempton advises that he can furnish:

- (1) ra g li lg; (2) ra g lg br; (3) pr li lg f;
 (4) cr li gi - gi = gigas; (5) lg ad f; (6) wx lg gl.

Lindstrom can furnish:

- (1) r g li b pl; (2) R g li b pl; (3) r g nl b pl;
 (4) R g nl b pl.

Singleton and Jongs have the following multiple tester:

A c R lg g P Su y.

Anderson has seed of:

P-br-f-bm₂; various combinations of sm and sk.

We have not listed any stocks from Cornell. In the corn letter of October 5, 1932, we listed the multiple testers available here.

Appended herewith is the list of maize characters with their gene symbols. We have attempted to make this list as accurate and up to date as possible but mistakes and discrepancies are bound to occur. We will appreciate it if you will call any of these errors to our attention.

We are making an attempt to collect seed of all of the maize characters in the central repository at Cornell. In the list of genes we have noted the stocks of which we have seed. If any one has seed of a character listed as not on hand at Cornell, he should send us a small supply of such seed.

		6.			7.				
<u>Gene</u>	<u>Character affected</u>	<u>Chromosome</u>	<u>Seed at Cornell</u>	<u>Described by</u>					
a ₁ , etc.	plant, aleurone and pericarp color	III	"	Emerson '18, Emerson & Anderson '32	Bn ₁	brown aleurone	VII	"	Kvakan '24
a ₂	plant and aleurone color	V	"	Jenkins '32	bp	brown pericarp	IX	"	Meyers '27
ad ₁	adherent tassel	I	"	Kempton '20	br	brachytic	I	"	Kempton '20
ad ₂	" "		"	Eyster	bs	barren sterile			Woodworth '26
ad ₃	" "		"	Eyster	bt ₁	brittle endosperm	V	"	Mangelsdorf '26
al	albescence	VI	"	Phipps	bt ₂	" "		"	Sprague
an ₁	anther ear	I	"	Emerson '22	bt ₃	" "		-	Beadle
ar	argentea	IX	"	Eyster	bt ₄	" "		"	Eyster
?	argostripe	VII	"	Eyster	bv	brevis	V	"	Li
as	asynapsis	I	"	Beadle and McClintock '28	c	aleurone	IX	"	East & Hayes '11
au ₁	aurea	IX	"	Eyster '29	cb	chloroblotch	V		
au ₂	aurea		"	Eyster '29	Ch	chocolate pericarp		"	Emerson and Anderson '31
B	plant color booster	II	"	Emerson '24	cr ₁	crinkly	III	"	Emerson '21
ba ₁	barren stalk	III	"	Hofmeyr	cr ₂	"	IX		Eyster '32
ba ₂	" "	II	"	Hofmeyr	d ₁	dwarf	III	"	Emerson '12
bd	branched sterile		"	Collins and Kempton	d ₂	dwarf	-		Suttle
be	branched ear		"	Bryan	d ₃	dwarf	IX	"	Demerec '23
Bh	blotched aleurone	VI	"	Emerson	d ₄	dwarf			
?	branched silkless		"	Kempton	d ₅	dwarf	II	"	Perry
bk	brittle stalk		"	Wiggins	d ₆	dwarf	V		Eyster '32
bl ₁	blotched leaf		"	Emerson '27	da	dilute aleurone	IX	"	Eyster '32
bl ₂	" "		"	Wiggins	de ₁	defective endosperm	IV		Mangelsdorf '26
bu ₁	brown midrib	V	"	Eyster '26	de ₂	" "			Mangelsdorf '26
bu ₂	" "	I	"	Burnham	de ₃	" "			Mangelsdorf '26
bu ₃	" "		"	Burnham	de ₄	" "			Mangelsdorf '26
					de ₅	" "			Mangelsdorf '26
					de ₆	" "			Mangelsdorf '26
					de ₇	" "			Mangelsdorf '26

8.				9.			
de ₈	defective endosperm		Mangelsdorf '26	ge ₃	premature germination		Mangelsdorf '26
de ₉	" "		Mangelsdorf '26	ge ₄	" "		Mangelsdorf '26
de ₁₀	" "		Mangelsdorf '26	ge ₅	" "		Mangelsdorf '26
de ₁₁	" "		Mangelsdorf '26	gl	gigas		Kempton
de ₁₂	" "		Mangelsdorf '26	gl ₁	glossy	VII	" Kvakan '24
de ₁₅	" "		Mangelsdorf '26	gl ₂	glossy	II	" Hayes & Brew-baker '28
de ₁₄	" "		Mangelsdorf '26	gl ₃	glossy	IV	" Hayes & Brew-baker '28
de ₁₅	" "	IX	Brink '27				
de ₁₆	" "	IV	Wentz '25	gl ₄	glossy	IX	" Sprague
de _{pl}	" "		Mangelsdorf '26	gl ₅	glossy	-	" Sprague
df	flint defective	X		gl ₆	glossy	-	- Sprague
dt	dotted leaf		" Emerson	gl ₇	glossy	-	" Sprague
f ₁	fine striped	I	" Lindstrom '18	gl ₈	glossy	-	" Sprague
f ₂	" "	V	- Eyster '26	gl ₉	glossy	-	" Sprague
f ₃	" "	X	" Eyster	gm ₁	germless		Demerec '28
fi	fine streaked	VI	Anderson '22	gm ₂	germless	X	Demerec '28
fl	floury endosperm	II	" Hayes & East '15	gm ₃	germless		
fr ₁	frayed	VII	" Jenkins & Pope	gm ₄	germless	VI	
fr ₂	frayed	VII	" Jenkins & Pope	*gm _e	germless	IX	Eyster '29
fs	fasciated		- Collins & Kempton	gs	green striped	I	" Emerson '12
g ₁	golden	X	" Emerson '12	h	soft starch		Munn '29
g ₂	golden		" Jenkins '26	hs	hairy sheath		" Tavcar
g ₃	golden	I	" Eyster	I	inhibitor of aleurone color	IX	" East & Hayes '11
g ₄	golden	IX	" Eyster	ij	iojap	VII	" Jenkins '24
Ga	pollen tube growth factor	IV	" Mangelsdorf and Jones '26	in	intensifier of aleurone color	VII	" Fraser '24
gc	glucostactous		Eyster '24				
ge ₁	premature germination		Mangelsdorf '26				
ge ₂	" "		Mangelsdorf '26				

* reported as gm₁.

						10.			11.
j	japonica	VIII	"	Emerson '12	ms ₆	male sterile			Beadle "
kn	knotted leaf		"	Bryan	ms ₇	" "			Beadle "
l ₁	luteus	X	"	Lindstrom '17	ms ₈	" "	VIII		Beadle "
l ₂	luteus	X	"	Lindstrom '25	ms ₉	" "			Beadle "
l ₃	luteus	-	-	Jenkins & Bell	ms ₁₀	" "			Beadle "
l ₄	luteus	X	-	Jenkins & Bell	ms ₁₁	" "			Beadle "
l ₅	luteus	V	"	Eyster '32	ms ₁₂	" "			Beadle "
l ₆	luteus	IX	"	Eyster	ms ₁₃	" "			Beadle "
l ₇	luteus	IX	"	Eyster	ms ₁₄	" "			Beadle "
la	lazy		"	Jenkins	ms ₁₅	" "			Beadle "
lg ₁	liguleless	II	"	Emerson '12	ms ₁₆	" "			Beadle "
lg ₂	liguleless	III	"	Brink	ms ₁₇	" "	I	"	Emerson
li	lineate	X	"	Collins and Kenpton '20	ms ₁₈	" "	V	"	Eyster
lp	pollen lethal	V	"	Rhoades	ms ₁₉	" "	-		Eyster
m ₁	yellow white seedling			Stroman '24	ms ₂₀	" "	IX	"	Eyster
m ₂	" " "			Stroman '24	st	mottled aleurone	X	"	Kenpton '19
mc	micropyle color			Singleton and Jones	na ₁	nana	III	"	Hutchinson '22
					na ₂	nana			Perry
md	mid cob color			Demerec '27	nl	narrow leaf	X	"	Emerson
mg	miniature germ			Wentz '24	o ₁	opaque endosperm			Singleton and Jones
mi	midget plant			Perry	o ₂	" "			Singleton and Jones
mr	midrib			Kvakan	oy	oil yellow	V	"	Eyster '32
ms ₁	male sterile	VI	"	Singleton and Jones	P, etc.	pericarp color (many allelomorphs)	I	"	
ms ₂	" "	IX	"	Eyster	pb ₁	piebald		"	Demerec '36
ms ₃	" "	III	"	Eyster	pb ₂	piebald		"	Demerec "
ms ₄	" "			Beadle - '32	pb ₃	piebald		"	Demerec "
ms ₅	" "			Beadle "	pb ₄	piebald		"	Demerec "
					pb₅	piebald			Demerec

				12.					13.
pc ₁	coleorhiza color		"	Sprague	ro	rolled leaves			Carver '37
pc ₂	" "		"	Sprague	rs	rough sheath		"	
pc ₃	" "		"	Sprague	rt	rootless			Jenkins '26
pc ₄	" "		"	Sprague	S ₁	scutellum color	IV	"	Sprague
pg ₁	pale green	X	"	Brunson '24	S ₂	" "		"	Sprague
pg ₂	" "	III	"	Demerec '25	S ₃	" "		"	Sprague
pg ₃	" "	VII	"	Demerec '25	S ₄	" "		"	Sprague
pg ₄	" "		"	Demerec '25	S ₅	" " inhibitor			Sprague
pg ₅	" "			Demerec '25	sa ₁	Striped auricle	IX	"	Eyster
pg ₆	" "	IX		Eyster '32	sa ₂	" "	V	-	Eyster
pg ₇	" "	V		Eyster '32	sb	slit blade			Beadle
pg ₈	" "		"	Eyster	sc	scarred endosperm	V		Eyster '26
pg ₉	" "			Eyster	sh	shrunken endosperm	IX	"	Hutchinson '21
pg ₁₀	" "			Eyster	si	silky	VI	"	Fraser
pi ₁	development of secondary florets			Hudson and Gillis '29	sk	silkless	II	"	Jones '25
pi ₂	" " " " "			Hudson and Gillis '29	sl	slashed	VII	"	Brewbaker
pk	polkadot leaves	IX	"	Eyster '24	?	small kernel	IX	"	Eyster '32
po	polysitotic	VI		Beadle '31	so ₁	orange scutellum		"	Sprague
pr	red aleurone	V	"	East & Hayes '17	so ₂	" "		"	Sprague
pu ₁	purple plumule			Jenkins '26	sp	small pollen	IV		Mangelsdorf and Singleton
pu ₂	" "			Sprague	sr	striate	I	"	Brunson
py	pigmy	VI	"	Suttle	st	sticky chromosomes	IV	"	Beadle '32
R, etc.	allelomorphic series, aleurone, plant and pericarp color	X	"	many	su	sugary endosperm	IV	"	Correns '01
ra	ramosa	VII	"	Cornert '15	su ₂	" "		"	Eyster
Rg ₁	ragged	III	"	Brink & Senn	sy	yellow scutellum			Sprague
Rg ₂	ragged			Singleton and Jones	th	threaded			Singleton and Jones
					tn	tinged	V	"	Eyster '26

14.

TP	teopod	VII	"	Lindstrom	v ₁₈	virescent	X	"	Phipps '29
ts ₁	tassel seed	II	"	Emerson '20	v ₁₉	virescent			Phipps '29
ts ₂	" "	I	"	Emerson '20	v ₂₀	virescent	X		Phipps '29
Ts ₃	" "	-	"	Emerson	va ₁	variable sterile	VII		Beadle '32
ts ₄	" "	III	"	Phipps '28	va ₂	" "			Beadle '32
Ts ₅	" "	IV	"	Emerson	vp ₁	vivipary	X	"	Eyster
Ts ₆	" "				vp ₂	vivipary	V		Eyster
Tu	tunicate	IV	"	Collins '17	vp ₃	vivipary			Eyster
tw ₁	twisted seedlings			Kvakan '25	vp ₄	vivipary	IX		Eyster
tw ₂	" "			Kvakan '25	w ₁	white seedling	VI	"	Emerson '12
tw ₃	" "			Kvakan '25	w ₂	white seedling	X		Stroman '24
v ₁	virescent	IX	"	Demerec '24	w ₃	" "			Demerec '23
v ₂	virescent	V	"	Demerec '24	w ₄	" "			
v ₃	virescent	V	"	Demerec '24	w ₅	" "	VI		Demerec '23
v ₄	virescent	II	"	Demerec '24	w ₆	" "	VI		Demerec '23
v ₅	virescent	VII	"	Demerec '24	w ₇	" "			Demerec '23
v ₆	virescent	VI	"	Carver '27	w ₈	" "			Demerec '23
v ₇	virescent	VI	"	Carver '27	w ₉	" "			Demerec '23
v ₈	virescent	IV		Demerec '26	w ₁₀	" "			Demerec '23
v ₉	virescent			Phipps '29	w ₁₁	" "	IX	"	Demerec '26
v ₁₀	virescent			Phipps '29	wa	warty anthers			Beadle '32
v ₁₁	virescent			Phipps '29	wc	white cap endosperm		"	Kulkarni '24
v ₁₂	virescent	V	"	Phipps '29	wh	dominant white endosperm	VII	"	White '17
v ₁₃	virescent			Phipps '29	wl	white leaf base	IV	"	Stroman '24
v ₁₄	virescent (same as v ₁₂)	IX	"	Phipps '29	ws ₁	white sheath			Clark '32
v ₁₅	virescent	IX		Phipps '29	ws ₂	" "			Clark '32
v ₁₆	virescent			Phipps '29	wx	waxy endosperm	IX	"	Collins '09
v ₁₇	virescent		"	Phipps '29	xm ₁	xantha	X	"	Trajkovich '24
					xn ₂	xantha		"	Demerec '25

15.

Y	yellow endosperm	VI	"	Correns '01
yd	yellow dwarf	VI	"	Singleton and Jones
Yg ₁	yellow green	V	"	Eyster '26
Yg ₂	" "	IX	"	Jenkins '27
Yg ₃	" "		"	Burnham
Ys ₁	yellow stripe	V	"	Beadle '29
Ys ₂	" "	II	"	Brink
yt	yellow top	III	"	Eyster '31
z	zigzag stalk	-	-	Eyster '22
zg	" "	I	-	Eyster '22
zb ₁	zebra striped		"	Demerec '21
zb ₂	" "		"	Demerec
zb ₃	" "		"	Demerec
zb ₄	" " seedling		"	Hayes '32
zl	zygotic lethal	I	"	Emerson

It should be unnecessary to do so, but we urge everyone to go carefully over the list of "wants" and if he has the desired stock to send it to the chap who requested it. Failure to cooperate will defeat the purpose of this service.

If enough requests for material come in we shall send out another corn letter before spring planting.

M. M. Rhoades

VI. REPORT OF MAIZE GENETICS COOPERATION STOCK CENTER

During 1982 there were 136 seed requests and 1,333 seed packets were sent. This was the highest figure in the last five years. Domestic requests amounted to 112 for 908 packets while there were 24 foreign requests for 425 packets.

The estimated uses of seed are distributed among these categories:

Geneticists	60.3%
Physiologists	16.2%
Breeders	8.0%
Educators	6.0%
Genetic Engineers	9.5%

This is the first time that the Genetic Engineer category has been used, and interest in this area is expanding rapidly.

The Center has purchased a computer terminal, and efforts are underway to computerize inventories, request procedures, planting and testing.

The MGCSC has some P.I. stocks recently increased which contain untested and unlocated markers. These stocks contain glossies and dwarfs, and could be made available to workers interested in resolving these kinds of problems.

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Dispersal

When a young person begins planting for himself, by far the most common way to obtain seed is as a gift from his parents (referred to as father) or close relatives. As a result, the seed tends to remain in its original area; however, when a family member changes residential location he takes his own seed to plant at the new site. When a new variety, of whatever origin, is grown at a given village, the neighbors and friends are able to obtain the new seed. In the most remote areas this seed corn is a gift, but near the highly populated centers seed is supplied on a loan basis; the borrower is expected to return a comparable amount of corn if he has obtained a reasonable harvest from the seed. If the seed fails to produce, both parties just "forget it" and start over again the next year.

Feelings about the "necessity" of giving seed corn to any one who asks is not as strong in northcentral Guatemala as in the backwoods of Honduras or Costa Rica (Johannessen, 1970), where the Indians believe it is essential that all seeds be gifts. They are convinced that borrowed or purchased seed would displease the gods who "tend to these things" and who would thus cause poor production.

--Johannessen, 1982

Catalogue of Stocks

Chromosome 1

sr zb4 P-WW
 sr P-WR
 sr P-WW
 sr P-WR an gs bm2
 sr P-WR an bm2
 sr P-RR 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-WR
 zb4 P-WW br
 zb4 P-WW br f bm2
 zb4 P-WW bm2
 ms17
 ts2 P-RR
 ts2 P-WW bm2
 ts2 P-WW br 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 br f an gs bm2 rd
 P-RR br f an gs bm2 id
 P-RR br f an gs bm2 v*-8983
 P-RR br f an gs bm2 v*-8943
 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 an br bm2
 P-WT = WR an bm2
 P-WR br Vg
 P-WR br f gs bm2
 P-WR br f an lw gs bm2
 P-WR br f bm2 id
 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 br2
 as rs2
 rJ Hy
 br f
 br f bm2 v*-5568
 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
 bz2 ad bm2 ACR
 an bm2
 an-bz2-6923 (apparent deficiency
 including an and bz2)
 br2
 br2 bm2
 br2 an bm2
 tb-8963
 Kn
 Kn Ts6
 Kn bm2
 lw
 Adh1-S
 vp8

Chromosome 1 (continued)

gs
 gs bm2
 Ts6
 bm2
 id
 nec2
 ms9
 ms12
 ms14
 mi
 D8
 Lls
 Les2
 TB-1La (1L.20)
 TB-1Sb (1S.05)
Chromosome 2
 ws3 lg g12 B
 ws3 lg g12 B sk
 ws3 lg g12 B sk v4
 ws3 lg g12 B sk fl v4
 ws3 lg g12 B gs2 v4
 ws3 lg g12 B ts
 ws3 lg g12 b
 ws3 lg g12 b sk
 ws3 lg g12 b sk v4
 ws3 lg g12 b gs2 v4
 ws3 lg g12 b fl v4
 ws3 lg g12 b sk fl v4
 ws3 lg g12 b v4
 al
 al lg
 al lg g12 B sk v4
 al lg g12 b
 al lg g12 b sk v4
 al lg g12 b sk fl v4
 lg
 lg g12
 lg g12 B
 lg g12 B gl11
 lg g12 B gs
 lg g12 B gs2 v4
 lg g12 B gs2 Ch
 lg g12 B gs2 sk Ch
 lg g12 B gs2 sk v4
 lg g12 B sk
 lg g12 B sk v4
 lg g12 B v4
 lg g12 b
 lg g12 b gs2
 lg g12 b gs2 Ch
 lg g12 b gs2 sk Ch
 lg g12 b gs2 v4
 lg g12 b gs2 v4 Ch
 lg g12 b gs2 sk v4 Ch
 lg g12 b sk
 lg g12 b sk fl
 lg g12 b sk fl v4
 lg g12 b sk v4
 lg g12 b wt v4
 lg g12 b fl
 lg g12 b fl v4
 lg g12 b fl v4 Ch
 lg g12 b v4
 lg g12 b v4 Ch
 lg g12 mn v4
 lg g12 wt
 lg g12 b gs2 wt
 lg g12 w3
 lg g12 w3 Ch
 lg g12 Ch
 lg b gs2 v4
 lg Ch
 g12
 d5 = d*-037-9
 B gl11
 B ts
 gl14
 gl11
 wt
 mn
 fl
 fl v4 Ch

Chromosome 2 (continued)

fl Ht v4
 fl Ht v4 Ch
 fl w3
 fl v4 w3
 fl w3 Ch
 fl v4 w3 Ch
 ts
 v4
 v4 w3 Ht Ch
 v4 Ht Ch
 w3
 w3 Ht
 w3 Ht Ch
 w3 Ch
 Ht (A & B source)
 ba2
 R2 ; r A A2 C
 r2 ; r-g A A2 C
 Ch
 gs2
 Les
 2 2Trip Trip2 /ws3 lg g12
 TB-1Sb-2L4464
 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 (short)
 d rt Lg3
 d Rf lg2
 d ys3
 d ys3 Rg
 d ys3 Rg lg2
 d Lg3
 d Lg3 gl6
 d Lg3 ts4 lg2
 d Rg
 d Rg ts4 lg2
 d pm
 d yg*(W23)
 d ts4 lg2
 d ts4 lg2 a-m ; A2 C R Dt
 d ts4
 d gl6
 d lg2 a-m A2 C R Dt
 d a-m A2 C R Dt
 ra2
 ra2 Rg
 ra2 Rg ts4 lg2
 ra2 Rg gl6
 ra2 ys3 Lg3 Rg
 ra2 ys3 Rg
 ra2 Rg lg2
 ra2 pm lg2
 ra2 ts4
 ra2 ts4 lg2
 ra2 lg2
 Cg
 cl
 cl ; Clm-2
 cl ; Clm-3
 cl-p ; Clm-4
 rt
 ys3
 ys3 Lg3
 ys3 gl6 lg2 a-m et ; A2 C R Dt
 ys3 ts4
 ys3 ts4 lg2
 Lg3
 Lg3 Rg pm
 gl6
 gl6 lg2 A ; A2 C R
 gl6 lg2 A-b et ; A2 C R Dt
 gl6 lg2 a-m et ; A2 C R Dt

Chromosome 3 (continued)

pm lg2
 ts4
 ts4 na
 ts4 na pm
 ts4 ba na
 ts4 lg2 a-m ; A2 C R Dt
 ts4 na a-m ; A2 C R Dt
 ig
 ba
 y10
 lg2
 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
 na lg2
 A sh2 ; A2 C R B P1 dt
 A-d31 ; A2 C R
 A-d31 ; A2 C R pr dt
 A-d31 ; A2 C R B P1 dt
 A-d31 ; A2 C R Dt
 A-d31 ; A2 C R pr Dt
 A-d31 sh2 ; A2 C R B P1 dt
 A-d31 sh2 ; A2 C R Dt
 A-d31 sh2 ; A2 C R B P1 Dt
 A-d31 et ; A2 C R Dt
 a-m ; A2 C R B P1 dt
 a-m ; A2 C R Dt
 a-m ; A2 C R B P1 Dt
 a-m sh2 ; A2 C R B P1 dt
 a-m sh2 ; A2 C R B P1 Dt
 a-m et ; A2 C R Dt
 a-st ; A2 C R Dt
 a-st sh2 ; A2 C R Dt
 a-st sh2 A2 C R B P1 Dt
 a-st sh2 et ; A2 C R Dt
 a-st et ; A2 C R Dt
 a-p sh2 et ; A2 C R B P1 Dt
 a-p et ; A2 C R Dt
 a-p et ; A2 C R B P1 Dt
 a-x1
 a-x3
 a Ga7 ; A2 C R
 sh2
 vp
 Rp3
 pgl4
 a3
 g5
 te
 h
 yel*-5787
 TB-3La (3L.10)
 TB-3Sb (3S.50)
 TB-3Lc (distal to 3La*(3L.10))
 Primary Trisomic 3

Chromosome 4

Rp4
 Ga
 Ga su
 Ga-S
 Ga-S ; y
 Ga-S ; A A2 C R
 st
 st Ts5
 st f12
 Ts5
 Ts5 f12
 Ts5 su
 Ts5 la su g13
 Ts5 su zb6
 Ts5 su zb6 o
 Ts5 su g13 o
 Ts5 Tu
 la
 la su Tu g13
 la su g13

Chromosome 4 (continued)

la su g13 c2 ; A A2 C R
 la su g13 o
 la su bt2 g13
 f12
 f12 su
 f12 bt2
 f12 su bm3
 f12 su g14 Tu
 su
 su-am
 su bt2 g14
 su bm3
 su zb6
 su zb6 bt2
 su zb6 Tu
 su zb6 g13 dp
 su g14 j2
 su g14 o
 su g14 o Tu
 su j2
 su g13
 su g13 o
 su o
 su g14
 bt2
 bm3
 g14
 g14 o
 Tu
 Tu-1 1st
 Tu-1 2nd
 Tu-d
 Tu-md
 Tu g13
 j2
 j2 c2 ; A A2 C R
 j2 C2 ; A A2 C R
 j2 g13
 v8
 g13
 g13 o
 g13 dp
 c2 ; A A2 C R
 C2 ; A A2 C R
 C2-Idf (Active-1) ; A A2 C R
 dp
 o
 vl7
 v23
 ra3
 Dt4 su ; a-m A2 C R
 TB-4Sa (4S.20)
 TB-1La-4L4692
 TB9Sb-4L6504 (9S.40-.83; 4L.09)
 TB7Lb-4L4698 (7L.30-.74; 4L.08)
 Primary Trisomic 4

Chromosome 5

am a2 ; A A2 C R
 lu
 lu sh4
 ms13
 g117
 g117 A2 pr ; A C R
 g117 a2 ; A C R
 g117 a2 bt ; A C R
 g117 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 v2 ; A C R
 A2 pr na2 ; A C R
 A2 pr ys ; A C R
 A2 pr zb3 ; A C R

Chromosome 5 (continued)

A2 pr vl2 ; A C R
 a2 ; A C R
 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 v2 ; A C R
 A2 v3 pr ; A C R
 a2 bt v3 pr ; 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
 a2 pr ; A C R B P1
 a2 pr v2 ; A C R
 vp2
 vp2 pr
 vp2 g18
 vp7
 bm
 bm yg
 bt
 ms5
 v3
 td ae
 ae
 sh4
 gl8
 na2
 lw2
 ys
 eg
 v2
 YG
 ms13
 vl2
 br3
 nec3
 TB-5La
 TB-5Lb
 Primary Trisomic 5

Chromosome 6

rgd po y
 rgd po Y
 rgd y
 rgd Y
 po = ms6
 po y pl
 po y P1
 po y w1
 po Y pl
 y = pb = w-m
 y rhm
 y l10
 y l11
 y l12
 y W15
 y pb4
 y pb4 pl
 y pb4 P1
 y s1
 y w1 P1
 Y Dt2 ; a-m A2 C R
 y pgl1 ; Wx pgl2
 y pgl1 w1 ; wx pgl2
 Y pgl1 ; Wx pgl2
 y pgl1 ; wx pgl2
 Y pgl1 ; wx pgl2
 y pgl1 su2 ; wx pgl2
 y pl
 y P1
 y P1 Bh ; c sh wx A A2 R
 y pl Bh ; c sh wx A A2 R
 y su2
 Y l10
 Y l12
 Y pb4
 Y w1 pl
 Y w1 P1
 Y su2
 w1

Chromosome 6 (continued)

Pl Dt2 ; a-m A2 C R
 pl sm ; P-RR
 Pl sm ; P-RR
 Pl sm py ; P-RR
 Pl sm Pt py ; P-RR
 Pt
 w
 w14
 ms6
 2NOR ; a2 bm pr v2
 TB-6Lb
 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 ij
 o2 bd
 y8 v5 gl
 in ; A2 pr A C R
 in gl ; A2 pr A C R
 v5
 vp9
 vp9 gl
 ra
 ra gl ij bd
 gl
 gl-M
 gl Tp

 gl mn2
 Tp
 ij
 ms7
 ms7 gl Tp
 Bn
 bd
 Pn
 o5
 o5 mn2 gl
 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
 fl3
 fl3 j
 TB-8La (8L.70)
 Primary Trisomic 8

Chromosome 9

yg2 C Bz Wx ; A A2 R
 yg2 C sh bz ; A A2 R
 yg2 C sh bz wx ; A A2 R
 yg2 C-I sh bz wx ; A A2 R

Chromosome 9 (continued)

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-S9 ; A A2 R-g
 yg2 c bz wx ; A A2 R
 wd-Ring C-I ; A A2 R (temp. out of seed)
 C sh bz ; A A2 R
 C sh bz wx ; A A2 R
 C sh bz wx bm4 ; 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 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 Pr y
 C Ds wx ; A A2 R pr Y
 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-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 bz wx ; 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
 sh
 sh wx v
 sh wx d3
 sh wx pgl2 gl15 ; y pgl1
 lo2
 wx*
 wx-a
 wll
 wx d3
 wx d3 wll
 wx d3 v gl15
 wx d3 gl15
 Wx pgl2 ; y pgl1
 wx pgl2 ; y pgl1
 Wx pgl2 ; Y pgl1
 wx pgl2 ; Y pgl1
 wx pgl2 bm4 ; y pgl1
 wx v
 wx v gl15 bk2 Bf bm4
 wx bk2
 wx bk2 bm4
 wx Bf
 wx Bf bm4
 v
 ms2
 gl15
 gl15 Bf
 gl15 bm4
 bk2 Wx
 Wc
 bm4
 bm4 Bf
 16

Chromosome 9 (continued)

17
 yel*-034-16
 w*-4889
 w*-8889
 w*-8951
 w*-8950
 w*-9000
 Tp9 N9 N3 Df3
 TB-9La (9L.40)
 TB-9Sb (9S.40)
 TB-9Lc
 Primary trisomic 9
 *Additional waxy alleles available
 from collection of O. E. Nelson.

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
 oy du r ; A A2 C
 oy sr2
 oy zn
 sr3
 Og
 Og B Pl
 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
 y9 v18
 nl
 li zn g r ; A A2 C
 li g R ; A A2 C
 li g r ; A A2 C
 li g r v18 ; A A2 C
 li g r v18 ; A A2 C
 ms10
 du
 du v18
 du o7
 du g r ; A A2 C
 du sr2
 zn
 zn g
 zn g R sr2 ; A A2 C
 zn g r ; A A2 C
 Tp2 g r ; A A2 C
 g R sr2 ; A A2 C
 g r ; A A2 C
 g r sr2 ; A A2 C
 g r sr2 l ; A A2 C
 g R-g sr2 ; A A2 C
 g R-g sr2 v18 ; A A2 C
 g R-g K10 ; A A2 C
 g R-g sr2 ; A A2 C
 g R-r K10 ; A A2 C
 g r-r sr2 ; A A2 C
 Ej r-r ; A A2 C
 Ej r-r sr2 ; A A2 C
 r sr2 l ; A A2 C
 R-g ; A A2 C
 r-g sr2 ; A A2 C
 r K10 ; A A2 C
 r-g ; A A2 C
 r-r ; A A2 C
 r-ch Pl ; A A2 C
 R-mb ; A A2 C
 R-nj ; A A2 C

Chromosome 10 (continued)

R-r ; A A2 C
 R-ch B Pl ; A A2 C
 R-lsk ; A A2 C
 R-sk-mc.2 ; A A2 C
 R-sk ; A A2 C
 R-st ; A A2 C
 R-st Mst
 R-st Mst o7
 R-scm2 ; bz2 A A2 C C2
 R-scm2 ; a-st A2 C C2
 R-scm2 ; c2 A A2 C
 R-scm122 ; pr A A2 C C2
 R-scm2 ; a2 A C C2
 R-scm2 ; c A A2 C2
 Lc
 w2
 w2 1
 o7
 o7 ; o2
 1
 vi6
 mst
 1 yel#-5344
 yel#-8721
 yel#-8454
 yel#-8793
 cm
 TB-10La (10L.35)
 TB-10Sc
 TB-10L19
 Primary trisomic 10

Unplaced Genes

dv
 dy
 el
 14
 o9
 o10
 o11
 ci3
 Rs
 vi3
 ws ws2
 ub
 zb
 zb1
 zn2
 1#-4923
 nec#-6376

Multiple Gene Stocks

A A2 C C2 R-g Pr B Pl
 A A2 C C2 R-g Pr B pl
 A A2 C C2 R-g b Pl
 A A2 C C2 r-g Pr B Pl
 A A2 C C2 r-g Pr b pl
 A A2 C C2 R-r Pr B Pl
 A A2 C C2 R-r Pr B pl
 A A2 C C2 R-r Pr B Pl wx
 A A2 C C2 R-r Pr B pl wx
 A A2 C C2 R-r Pr B Pl
 A A2 C C2 r-r Pr B Pl
 A A2 C C2 r-r Pr B Pl
 A A2 C C2 R Pr
 A A2 C C2 R Pr wx
 A A2 C C2 R Pr wx gl
 A A2 C C2 R Pr
 A A2 C C2 R pr y wx gl
 A A2 C C2 R Pr y wx
 A A2 C C2 R Pr Y wx
 su pr y gl wx ; A A2 C C2 R
 A su pr ; A2 C C2 R
 bz2 a c2 a2 pr Y/y c bz wx r
 a su A2 C C2 R
 bm2 lg a su pr y gl j wx g
 colored scutellum
 lg gl2 wt ; a Dt A2 C C2 R
 a su pr y gl wx A A2 C C2 R
 hm hm2
 ts2 ; sk
 lg gl2 wt ; a-m A2 C C2 R Dt
 A A2 C C2 R-nj ; purple embryo S. Chase
 Stock 6 ; Hi-haploid R-r B Pl

Popcorns

Amber Pearl
 Argentine
 Black Beauty
 Hullless
 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
 Gaspé Flint
 Gourdseed
 Maiz Chapolote
 Papago Flour Corn
 Parker's Flint
 Tama Flint
 Zapaluta Chica

Tetraploid Stocks

P-RR
 P-VV
 B Pl
 a A2 C R Dt
 su
 pr ; A A2 C R
 y
 gl
 Y sh wx
 sh bz wx
 wx
 g A A2 C R
 A A2 C R B Pl

Cytoplasmic traits

NCS2
 NCS3

Cytoplasmic steriles and Restorers

WF9-(T) rf rf2 [Temp. out of seed]
 WF9 rf rf2
 R213 Rf rf2
 Ky21 Rf Rf2

Waxy Reciprocal Translocations

wx 1-9c (1S.48; 9L.22) * Sx
 wx 1-94995 (1L.19; 9S.20) * Sx
 wx 1-98389 (1L.74; 9L.13) W23 only
 wx 2-9b (2S.18; 9L.22) * Sx
 wx 3-9c (3L.09; 9L.12) * Sx
 wx 4-9b (4L.90; 9L.20) * Sx
 wx 4-95657 (4L.33; 9S.25) * Sx
 wx 4-9g (4S.27; 9L.27) W23 only
 wx 5-9a (5L.69; 9S.17) * Sx
 wx 5-9c (5S.07; 9L.10) W23 only
 wx 6-9a (6S.79; 9L.40) * Sx
 wx y 6-9b (6L.10; 9S.37) * Sx
 wx 7-9a (7L.63; 9S.07) * Sx
 wx 7-94363 (7 cent.; 9 cent.) * Sx
 wx 8-9d (8L.09; 9L.16) * Sx
 wx 8-96673 (8L.35; 9S.31) * Sx
 wx 9-10b (9S.13; 10S.40) * Sx

Non-waxy Reciprocal Translocations

Wx 1-9c (1S.48; 9L.24) * Sx
 Wx 1-94995 (1L.19; 9S.20) * Sx
 Wx 1-98389 (1L.74; 9L.13) * Sx
 Wx 2-9c (2L.49; 9S.33) W23 only
 Wx 2-9b (2S.18; 9L.22) * Sx
 Wx 3-98447 (3S.44; 9L.14) * Sx
 Wx 3-98562 (3L.65; 9L.22) * Sx
 Wx 4-9e (4S.53; 9L.26) * Sx
 Wx 4-95657 (4L.33; 9S.25) * Sx

Wx 5-9c (5S.07; 9L.10) * Sx
 Wx 5-94817 (5L.69; 9S.17) M14 only
 Wx 5-98386 (5L.87; 9S.13) * Sx
 Wx 6-94778 (6S.80; 9L.30) * Sx
 Wx 6-98768 (6L.89; 9S.61) * Sx
 Wx 7-94363 (7 cent.; 9 cent.) * Sx
 Wx 7-9a (7L.63; 9S.07) W23 only
 Wx 8-9d (8L.09; 9L.16) * Sx
 Wx 8-96673 (8L.35; 9S.31) * Sx
 Wx 9-108630 (9S.28; 10L.27) M14 only
 Wx 9-10b (9S.13; 10S.40) * Sx

* = Homozygotes available in both
 M14 & W23 backgrounds

Sx = Single cross of homozygotes between
 M14 & W23 versions available

Inversions

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.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-L69)
 Inv.6-8452 (6S.77-L.33)
 Inv.6-8604 (6S.85-L.32)
 Inv.6-3712 (6S.76-L.63)

Inv.7L-5803 (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)

VII. RECENT MAIZE PUBLICATIONS

The following 625 references have been compiled by screening the literature as described in MNL 55:145.

- Agafonov, A. V., and A. G. Maystrenko, 1979. Electron microscopic studies of the synaptonemal complex of a desynaptic mutant of maize. *Izv. Sib. Otd. Akad. Nauk SSSR Ser. Biol. Nauk*, 1979 (3):36-40.
- Albergoni, F. G., B. Basso, T. Brusa and L. M. Recalcati, 1982. On the measurement of photosynthetic potentiality. *Maydica* 27:97-105.
- Albrecht, S. L., 1981. Nitrogen fixation by corn (*Zea mays*-*Azospirillum brasilense*) associations in a temperate climate. *Crop Sci.* 21:301-306.
- Alexander, W. L., H. Z. Cross and H. A. Lamey, 1982. Early corn inbred reactions to head smut, common smut, and common rust. *N. Dak. Farm Res.* 40:29-31.
- Allen, D. J., and R. A. Skipp, 1982. Maize pollen alters the reaction of cowpea to pathogens. *Field Crop. Res.* 5:265-269.
- Aman, M. A., D. S. Mathur and K. R. Sarkar, 1981. Effect of pollen and silk age on maternal haploid frequencies in maize. *Indian J. Genet.* 41:362-365.
- Andersen, R. N., and J. L. Geadelmann, 1982. The effect of parentage on the control of volunteer corn in soybean. *Weed Sci.* 30:127-131.
- Anderson, L. E., A. R. Ashton, A. H. Mohamed and R. Scheibe, 1982. Light/dark modulation of enzyme activity in photosynthesis. *BioScience* 32:103-107.
- Andrew, R. H., 1982. Factors influencing early seedling vigor of shrunken-2 maize. *Crop Sci.* 22:263-266.
- Anguillesi, M. C., I. Grilli and C. Floris, 1982. Polyamines and protein metabolism in maize inbreds differing in seed protein content. *J. Exp. Bot.* 33:1014-1020.
- Anzola, D., C. P. Romaine, L. V. Gregory and J. E. Ayers, 1982. Disease response of sweet corn hybrids derived from dent corn resistant to maize dwarf mosaic virus. *Phytopathology* 72:601-604.
- Appels, R., R. A. Bouchard and H. Stern, 1982. cDNA clones from meiotic-specific poly(A)⁺ RNA in *Lilium*--homology with sequences in wheat, rye and maize. *Chromosoma* 85:591-602.
- Argos, P., K. Pedersen, M. D. Marks and B. A. Larkins, 1982. A structural model for maize zein proteins. *J. Biol. Chem.* 257:9984-9990.
- Arihara, J., and T. M. Crosbie, 1982. Relationships among seedling and mature root system traits of maize. *Crop Sci.* 22:1197-1202.
- Arruda, P., and W. J. Dasilva, 1982. Protein and amino acid metabolism in normal and high-lysine sugary opaque-2 maize endosperm during kernel development. *Rev. Brasil. Genet.* 5:313-328.
- Arruda, P., L. Sodek and W. J. Dasilva, 1982. Lysine-ketoglutarate reductase activity in developing maize endosperm. *Plant Physiol.* 69:988-990.
- Ayad, G., J. Toll and J. T. Esquinas-Alcazar, 1980. Directory of Germplasm Collections. III. Cereals. 2. Maize. *Internat. Board Plant Gen. Resources, FAO, Rome.* 23 pp.
- Ayuk-Takem, J. A., 1982. Response of SI maize varieties and varietal hybrids to altitude and environments in Cameroon. *J. Agr. Sci.* 98:615-622.
- Baba, T., Y. Arai, T. Ono, A. Munakata, H. Yamaguchi and T. Itoh, 1982. Branching enzyme from amylomaize endosperms. *Carbohydr. Res.* 107:215-230.
- Baba, T., T. Yamamoto, Y. Arai, M. Yokota and T. Itoh, 1981. Starch biosynthesis of amylomaize during endosperm development. *Phytochemistry* 20:1513-1518.
- Babitskii, A. F., 1981. Energy consumption in maize leaf mesophyll cells and the heterotic effect. *Izv. Akad. Nauk Mold. SSR Ser. Biol. Khim. Nauk* 74-75.
- Bagnara, D., and T. B. Daynard, 1982. Rate and duration of kernel growth in the determination of maize (*Zea mays* L.) kernel size. *Can. J. Plant Sci.* 62:579-588.
- Banks, P., E. J. Britten and D. E. Byth, 1982. Heritable para-fluorophenylalanine-induced aneuploidy in maize. *J. Hered.* 73:465-466.
- Barlow, P. W., 1981. Argyrophilic intranuclear bodies of plant cells. *Experientia* 37:1017-1018.
- Barnabas, B., and E. Rajki, 1981. Fertility of deep-frozen maize (*Zea mays* L.) pollen. *Ann. Bot.* 48:861-864.
- Baszczynski, C. L., D. B. Walden and B. G. Atkinson, 1982. Regulation of gene expression in corn (*Zea mays* L.) by heat shock. *Can. J. Biochem.* 60:569-579.
- Bates, L. S., 1982. Maize pollen as a uniform testing material for biochemical studies. Pp. 277-278 in W. F. Sheridan, ed., 1982 (which see).
- Bates, L. S., 1982. Maize breeding and future goals: Modified "hard-endosperm" opaque-2 maize. Pp. 357-359 in W. F. Sheridan, ed., 1982 (which see).
- Baum, J. A., and J. G. Scandalios, 1982. Expression of genetically distinct superoxide dismutases in the maize seedling during development. *Develop. Genet.* 3:7-23.
- Baum, J. A., and J. G. Scandalios, 1982. Multiple genes controlling superoxide dismutase expression in maize. *J. Hered.* 73:95-100.
- Bauman, L. F., 1981. Review of methods used by breeders to develop superior corn inbreds. *Proc. 36th Annu. Corn & Sorghum Res. Conf.* 36:199-208.
- Bausch, P., W. Schuster and E. Schlosser, 1982. Susceptibility of maize for stalk and root rots. *Angew. Bot.* 56:9-24.
- Bazzaz, M. B., and R. G. Brereton, 1982. 4 vinyl-4-desethyl chlorophyll--a new naturally occurring chlorophyll. *FEBS Lett.* 138:104-108.
- Beckert, M., 1982. Importance of the scutellum in obtaining in vitro regeneration of maize. *Agronomie* 2:611-616.
- Beckett, J. B., 1982. An additional mechanism by which B-chromosomes are maintained in maize. *J. Hered.* 73:29-34.
- Beckett, J. B., 1982. Locating genes to chromosomes: B-A translocations. Pp. 71-73 in W. F. Sheridan, ed., 1982 (which see).
- Belousov, A. A., 1981. Use of forms of maize differing in protein and lysine content for creation of hybrids with an increase in content and improved amino acid composition of protein. *Dokl. Vses. Ordena Lenina Ordena Trud Krasnogo Znameni Akad. S-KH. Nauk Im V. I. Lenina* 14-17.
- Below, F. E., L. E. Christensen, A. J. Reed and R. Hageman, 1981. Availability of reduced nitrogen and carbohydrates for ear development of maize. *Plant Physiol.* 68:1186-1190.

- Berville, A., and M. Charbonnier, 1982. Mitochondria and chloroplasts: Speculations and reflections on the molecular mechanism of heterosis. Pp. 267-274 in W. F. Sheridan, ed., 1982 (which see).
- Bianchi, G., P. Avato and F. Salamini, 1982. Epicuticular waxes of albino maize. *Phytochemistry* 21:129-131.
- Birchler, J. A., 1982. The mapping of genes by the use of simple and compound translocations. Pp. 75-78 in W. F. Sheridan, ed., 1982 (which see).
- Birchler, J. A., and K. J. Newton, 1981. Modulation of protein levels in chromosomal dosage series of maize: The biochemical basis of aneuploid syndromes. *Genetics* 99:247-266.
- Bird, R. McK., 1982. Maize and teosinte germplasm banks. Pp. 351-355 in W. F. Sheridan, ed., 1982 (which see).
- Bird, R. McK., 1982. Systematics of *Zea* and the selection of experimental material. Pp. 341-350 in W. F. Sheridan, ed., 1982 (which see).
- Blaich, R., 1981. Function of genetic material: Genetics of storage proteins and gene enzyme relationships in higher plants. *Prog. Bot.* 43:153-158.
- Bogorad, L., 1981. Chloroplasts. *J. Cell Biol.* 91:256s-270s.
- Bogorad, L., S. O. Jolly, G. Link, L. McIntosh, C. Poulsen, Z. Schwarz and A. Steinmetz, 1980. Studies of the maize chloroplast chromosome. *Coll. Ges. Biol. Chem.* 31:87-96.
- Bonhomme, R., F. Ruget, M. Derieux and P. Vincourt, 1982. Aerial dry matter production and intercepted solar radiation for various maize genotypes. *Comptes Rendus* 294:393-398.
- Borck, K. S., and V. Walbot, 1982. Comparison of the restriction endonuclease digestion patterns of mitochondrial DNA from normal and male sterile cytoplasms of *Zea mays* L. *Genetics* 102:109-128.
- Borowski, M. I., G. Karaiwanow, W. H. Schuster, H. T. Bohm, R. Marquard and X. H. Michailenko, 1982. Investigations about breeding of high protein and high lysine maize varieties. *Bodenkultur* 33:230-245.
- Borowski, M. I., G. Karaiwanow, W. Schuster, H. T. Bohm, R. Marquard and X. H. Michailenko, 1982. Investigations on improvement of protein content and protein quality of maize. *Angew. Bot.* 56:179-190.
- Boyer, C. D., P. A. Damewood and G. L. Matterns, 1980. Effect of gene dosage at high amylose loci on the properties of the amylopectin fractions of the starches. *Starke* 32:217-222.
- Boyer, C. D., E. K. G. Simpson and P. A. Damewood, 1982. The possible relationship of starch and phytyglycogen in sweet corn. II. The role of branching enzyme I. *Starke* 34:81-84.
- Boyer, J. S., 1982. Plant productivity and environment. *Science* 218:443-448.
- Brakke, M. K., R. G. Samson and W. A. Compton, 1981. Recessive alleles found at *R* and *C* loci in maize stocks showing aberrant ratio at the *A* locus. *Genetics* 99:481-486.
- Branson, T. F., G. R. Sutter and J. R. Fisher, 1982. Comparison of a tolerant and a susceptible maize inbred under artificial infestations of *Diabrotica virgifera virgifera*: yield and adult emergence. *Environ. Entomol.* 11:371-372.
- Braun, E. J., 1982. Ultrastructural investigation of resistant and susceptible maize inbreds infected with *Erwinia stewartii*. *Phytopathology* 72:159-166.
- Breeze, V. G., and G. M. Milbourn, 1981. Inter-plant variation in temperate crops of maize. *Ann. Appl. Biol.* 99:335-352.
- Brill, W. J., and S. W. Ela, 1981. How about a nitrogen fixing corn plant? *Amer. Soc. Microbiol. Conf. on Genetics and Molecular Biol. of Industrial Microorg. Microbiology* (Wash., DC), pp. 396-397.
- Brown, W. L., 1982. Exotic germplasm in cereal crop improvement. Pp. 29-42 in *Plant Improvement and Somatic Cell Genetics*, I. K. Vasil, et al., eds., Academic Press, New York.
- Bryan, J. K., and N. R. Lochner, 1981. Effects of plant age and extraction conditions on the properties of homoserine dehydrogenase isolated from maize seedlings. *Plant Physiol.* 1395-1399.
- Bryan, J. K., and N. R. Lochner, 1981. Quantitative estimates of the distribution of homoserine dehydrogenase isozymes in maize tissues. *Plant Physiol.* 68:1400-1405.
- Burnham, C. R., 1982. The locating of genes to chromosome by the use of chromosomal interchanges. Pp. 65-70 in W. F. Sheridan, ed., 1982 (which see).
- Burnham, C. R., 1982. Personal recollections of events leading to a correlation of linkage maps and chromosomes in maize and barley. Pp. 93-105 in W. F. Sheridan, ed., 1982 (which see).
- Burnham, C. R., 1982. Details of the smear technique for studying chromosomes in maize. Pp. 107-118 in W. F. Sheridan, ed., 1982 (which see).
- Burr, B., and F. Burr, 1981. Transposable elements and genetic instabilities in crop plants. *Stadler Genet. Symp.* 13:115-128.
- Burr, B., and F. A. Burr, 1982. *Ds* controlling elements of maize at the shrunken locus are large and dissimilar insertions. *Cell* 29:977-986.
- Burr, B., F. A. Burr, T. P. St. John, M. Thomas and R. W. Davis, 1982. Zein storage protein gene family of maize. An assessment of heterogeneity with cloned messenger RNA sequences. *J. Mol. Biol.* 154:33-49.
- Burr, F., and B. Burr, 1981. Heterogeneity and expression of the zein storage protein gene family. *Stadler Genet. Symp.* 13:79-92.
- Burr, F. A., and B. Burr, 1982. 3 mutations in *Zea mays* affecting zein accumulation: A comparison of zein polypeptides, in vitro synthesis and processing, messenger RNA levels, and genomic organization. *J. Cell Biol.* 94:201-206.
- Canard, S., and J.-F. Ledent, 1981. Effect of temperature, daylength and photoperiod on anthesis in maize. *Bull. Soc. Roy. Bot. Belg.* 114:189-192.
- Cao, Z., C. Guo and J. Hao, 1981. A study of embryogenesis in pollen callus in maize (*Zea mays*). *Acta Genet. Sin.* 8:269-274.
- Cardy, B. J., and L. W. Kannenberg, 1982. Allozymic variability among maize inbred lines and hybrids--applications for cultivar identification. *Crop Sci.* 22:1016-1020.
- Carey, E. E., D. B. Dickinson, L. Y. Wei and A. M. Rhodes, 1982. Occurrence of sorbitol in *Zea mays*. *Phytochemistry* 21:1909-1911.
- Carey, E. E., A. M. Rhodes and D. B. Dickinson, 1982. Postharvest levels of sugars and sorbitol in sugary enhancer (*su₁*, *se*) and sugary (*su₂*, *Se*) maize. *Hortscience* 17:241.
- Carlson, J. E., S. Gabay-Laughnan and J. R. Laughnan, 1982. Nucleo-cytoplasmic interactions in *cms-S* of maize. Pp. 243-245 in W. F. Sheridan, ed., 1982 (which see).
- Carson, M. L., and A. L. Hooker, 1981. Inheritance of resistance to stalk rot of corn caused by *Colletotrichum graminicola*. *Phytopathology* 71:1190-1196.
- Carson, M. L., and A. L. Hooker, 1982. Reciprocal translocation testcross analysis of genes for anthracnose stalk rot resistance in a corn inbred line. *Phytopathology* 72:175-177.

- Castaneda, G. C., and J. L. M. Arizmendi, 1981. Study of corn (*Zea mays* L.) pollen longevity during the selection process. *Turrialba* 31:305-308.
- Chaly, N., and J. V. Possingham, 1981. Structure of constricted proplastids in meristematic plant tissues. *Biol. Cell* 41:203-210.
- Chandra, S., J. F. Chabot, G. H. Morrison and A. C. Leopold, 1982. Localization of calcium in amyloplasts of root cap cells using ion microscopy. *Science* 216:1221-1223.
- Chebatar, A. A., 1981. Interrelations between embryo and endosperm in early developmental stages. *Izv. Akad. Nauk Mold. SSR Ser. Biol. Khim. Nauk* 34-39.
- Chebatar, A. A., 1981. Structural and physiological relationships between embryo and endosperm at the early stages of development. *Acta Soc. Bot. Pol.* 50:265-268.
- Chesneaux, M. T., and A. Kobilinsky, 1982. Identification of maize varieties using morphological characteristics of the seedlings. *Agronomie* 2:45-54.
- Chiang, H. C., and L. K. French, 1980. Host tolerance: A short-term pest management tool. Maize and corn rootworm (*Diabrotica virgifera*) as a model. *FAO Plant Prot. Bull.* 28:137-138.
- Chourey, P. S., 1981. Genetic control of sucrose synthetase in maize endosperm. *Mol. Gen. Genet.* 184:372-376.
- Chourey, P. S., 1982. Starch mutants and their protein products. Pp. 129-134 in W. F. Sheridan, ed., 1982 (which see).
- Christensen, L. E., F. E. Below and R. H. Hageman, 1981. The effects of ear removal on senescence and metabolism of maize. *Plant Physiol.* 68:1180-1185.
- Coe, E. H., 1982. Planning progeny sizes and estimating recombination percentages. Pp. 89-91 in W. F. Sheridan, ed., 1982 (which see).
- Coe, E. H., Jr., D. A. Hoisington and M. G. Neuffer, 1982. Linkage map of corn (maize) (*Zea mays* L.). Pp. 377-393 in *Genetic Maps*, vol. 2, S. J. O'Brien, ed., National Cancer Institute, Frederick, Maryland.
- Coe, E. H., and R. S. Poethig, 1982. Genetic factors affecting plant development. Pp. 295-300 in W. F. Sheridan, ed., 1982 (which see).
- Compton, W. A., and J. H. Lonquist, 1982. A multiplicative selection index applied to 4 cycles of full-sib recurrent selection in maize. *Crop Sci.* 22:981-983.
- Comstock, R. E., 1982. Commentary on Comstock, R. E., H. F. Robinson and P. H. Harvey, 1949. A breeding procedure designed to make maximum use of both general and specific combining ability. *Agronomy J.* 41:360-367. *Current Contents* 13:18.
- Conti, S., 1981. Book review of *Quantitative Genetics in Maize Breeding*, by A. R. Hallauer and J. B. Miranda F. *Maydica* 26:293-294.
- Corfman, R. S., and G. R. Reeck, 1982. Immunoabsorbent isolation of trypsin inhibitors from corn and teosinte seeds. *Biochim. Biophys. Acta* 715:170-174.
- Cornu, A., and S. Gabay-Laughnan, 1982. Reversion of T male sterile cytoplasm to male fertility. Pp. 247-250 in W. F. Sheridan, ed., 1982 (which see).
- Courter, J. W., and A. M. Rhodes, 1982. A classification of vegetable corns and new cultivars for 1982. *Univ. Ill. Coop. Exper. Sta. Dept. Hort. Ser.* 39:11-15.
- Craig, J., 1980. Sorghum downy mildew research at Texas A&M University. Pp. 195-199 in *Sorghum Diseases: A World Review*, G. D. Bengtson, ed., ICRISAT, Andhra Pradesh, India.
- Craig, J., 1982. Identification of sorghum downy mildew resistance in corn by leaf reaction to conidial inoculum. *Phytopathology* 72:351-352.
- Crawford, T. W., Jr., and V. V. Rendig, 1982. Accumulation of amino acid nitrogen and acid-hydrolyzable ammonium nitrogen in opaque-2 and normal maize grain. *Maydica* 27:11-26.
- Crevecoeur, M., R. Deltour and R. Bronchart, 1982. Quantitative freeze-fracture study of plasmalemma and nuclear envelope of *Zea mays* root cells during early germination. *J. Ultrastruct. Res.* 80:1-11.
- Crosbie, T. M., and R. B. Pearce, 1982. Effects of recurrent phenotypic selection for high and low photosynthesis on agronomic traits in 2 maize populations. *Crop Sci.* 22:809-813.
- Cross, H. Z., and J. J. Hammond, 1982. Plant density effects on combining abilities of early maize synthetics. *Crop Sci.* 22:814-817.
- Dale, R. M. K., 1981. Sequence homology among different size classes of plant mitochondrial DNA. *Proc. Natl. Acad. Sci.* 78:4453-4457.
- Danage, S. R. S., and R. J. Williams, 1980. The International Crops Research Institute for the semi-arid tropics: Sorghum downy mildew program. Pp. 209-212 in *Sorghum Diseases: A World Review*, G. D. Bengtson, ed., ICRISAT, Andhra Pradesh, India.
- Daskalyuk, A. P., 1981. Radioactive labeling of plant seedling DNA. *Fiziol. Biokhim. Kul't. Rast.* 13:321-323.
- Dell, B., 1981. Male sterility and anther wall structure in copper deficient plants. *Ann. Bot.* 48:599-608.
- Demarini, D. M., M. J. Plewa and H. E. Brockman, 1982. Use of 4 short-term tests to evaluate the mutagenicity of municipal water. *J. Toxicol. Environ. Health* 9:127-140.
- Derieux, M., and R. Bonhomme, 1982. Heat unit requirements for maize hybrids in Europe. Results of the European FAO sub-network. I. Sowing-silking period. *Maydica* 27:59-77.
- Derieux, M., and R. Bonhomme, 1982. Heat unit requirements for maize hybrids in Europe. Results of the European FAO sub-network. II. Period from silking to maturity. *Maydica* 27:79-96.
- Derissi, R., and E. Paterniani, 1981. Estimates of genetic parameters in 2 sub-populations of the variety of maize (*Zea mays* L.) Piranao. *Rev. Brasil. Genetica* 4:579-592.
- Dewet, J. M. J., J. R. Harlan and D. E. Brink, 1982. Systematics of *Tripsacum dactyloides* (Gramineae). *Amer. J. Bot.* 69:1251-1257.
- Diano, M., 1982. Electrophoretic comparison of mitochondrial polypeptides from maize lines susceptible and resistant to *Helminthosporium maydis* race T. *Plant Physiol.* 69:1217-1221.
- Diepenbrock, W., and P. Stamp, 1982. The fatty acid composition in leaves of maize (*Zea mays* L.) seedlings in relation to genotype and temperature changes. *Angew. Bot.* 56:25-34.
- Dierks-Ventling, C., 1982. Globulins of developing maize seeds: Preliminary characterization. P. 545 in *Embryonic Development: Cellular Aspects*, M. M. Burger and R. Weber, eds., Alan R. Liss, Inc., New York.
- Dierks-Ventling, C., 1982. Storage protein characteristics of proline-requiring mutants of *Zea mays* L. *Theor. Appl. Genet.* 61:145-150.
- Dierks-Ventling, C., and K. Cozens, 1982. Immunochemical cross reactivity between zein, hordein and gliadin. *FEBS Lett.* 142:147-150.

- Dierks-Ventling, C., and C. Tonelli, 1982. Metabolism of proline, glutamate, and ornithine in proline mutant root tips of *Zea mays* L. *Plant Physiol.* 69:130-134.
- Dierks-Ventling, C., and D. Ventling, 1982. Tissue-specific immunofluorescent localization of zein and globulin in *Zea mays* L. seeds. *FEBS Lett.* 144:167-172.
- Di Fonzo, N., M. Motto, T. Maggiore, R. Sabatino and F. Salamini, 1982. N-uptake, translocation and relationships among N-related traits in maize as affected by genotype. *Agronomie* 2:789-796.
- Divinagracia-Laysa, F., and O. R. Exconde, 1981. Virulence and aggressiveness of *Erwinia carotovora* var. *chrysanthemi* isolates on maize. *Philipp. Agric.* 64:307-322.
- Dixon, L. K., C. J. Leaver, R. I. S. Brettell and B. G. Gengenbach, 1982. Mitochondrial sensitivity to *Drechslera maydis* T-toxin and the synthesis of a variant mitochondrial polypeptide in plants derived from maize tissue cultures with Texas male-sterile cytoplasm. *Theor. Appl. Genet.* 63:75-80.
- Dolbeer, R. A., P. P. Woronecki and R. A. Stehn, 1982. Effect of husk and ear characteristics on resistance of maize to blackbird (*Agelaius phoeniceus*) damage in Ohio. *Prot. Ecol.* 4:127-140.
- Donovan, L. S., P. Jui, M. Kloek and C. F. Nicholls, 1982. An improved method of measuring root strength in corn *Zea mays* L. *Can. J. Plant Sci.* 62:223-228.
- Dooner, H. K., 1982. Gene-enzyme relationships in anthocyanin biosynthesis in maize. Pp. 123-128 in W. F. Sheridan, ed., 1982 (which see).
- Döring, H-P., M. Geiser and P. Starlinger, 1981. Transposable element *Ds* at the shrunken locus in *Zea mays*. *Mol. Gen. Genet.* 184:377-380.
- Döring, H-P., M. Geiser, E. Weck, U. Courage-Tebbe, E. Tillman and P. Starlinger, 1982. Controlling element *Ds* at the shrunken locus in *Zea mays*. Pp. 213-216 in W. F. Sheridan, ed., 1982 (which see).
- Doyle, G. G., 1982. The allotetraploidization of maize. Part 3: gene segregation in trisomic heterozygotes. *Theor. Appl. Genet.* 61:81-90.
- Draganic, M., 1982. Inheritance of resistance of maize to stalk rot (*Gibberella zeae* Schw. Petch) and screening for sources of resistance. *Arhiv Poljopr. Nauke* 43:223-249.
- Draganic, M., M. Kraljevic-Balalic and L. Kojic, 1982. Inheritance of grain yield in maize (*Zea mays* L.). *Arhiv Poljopr. Nauke* 43:63-70.
- Drenska, A. I., K. D. Ganchev and C. P. Ivanov, 1981. Ion exchange chromatography of aminoethylated zein and N-terminal sequence of its higher molecular weight fraction. *Dokl. Bolg. Akad. Nauk* 34:811-814.
- Dudley, J. W., 1982. Theory for transfer of alleles. *Crop Sci.* 22:631-637.
- Duvick, D. N., 1981. Genetic diversity in corn improvement. *Proc. 36th Annu. Corn Sorghum Res. Conf.* 36:48-60.
- Eagles, H. A., 1982. Inheritance of emergence time and seedling growth at low temperatures in four lines of maize. *Theor. Appl. Genet.* 62:81-88.
- Eagles, H. A., and I. R. Brooking, 1981. Populations of maize with more rapid and reliable seedling emergence than cornbelt dents at low temperatures. *Euphytica* 30:755-763.
- Earle, E. D., 1981. Application of genetic engineering to corn improvement. *Proc. 36th Annu. Corn & Sorghum Res. Conf.* 36:176-190.
- Earle, E. D., 1982. Cytoplasm-specific effects of *Helminthosporium maydis* race T toxin on corn protoplasts and mitochondria. Pp. 351-367 in *Variability in Plants Regenerated From Tissue Culture*, E. D. Earle and Y. Demarly, eds., Praeger Publ., New York.
- Earle, E. D., 1982. Effects of *Helminthosporium maydis* race T toxin on mitochondria and protoplasts from T cytoplasm maize. Pp. 251-255 in W. F. Sheridan, ed., 1982 (which see).
- Ebeid, M., J. Eder, M. Kutacek and A. Piovarci, 1981. Glutamic oxalacetic transaminase and glutamic pyruvic transaminase activity in extirpated sprouts of normal and opaque 2 maize (*Zea mays*) seedlings. *Biol. Plant.* 23:345-350.
- Echt, C. S., and D. Schwartz, 1981. Evidence for the inclusion of controlling elements within the structural gene at the waxy locus in maize. *Genetics* 99:275-284.
- El-Itriby, H. A., A. R. Selim and A. H. Shehata, 1981. Genotype X environment interaction from combining ability estimates in maize (*Zea mays* L.). *Egypt. J. Genet. Cytol.* 10:175-186.
- Elsahookie, M. M., 1982. A new technique for improving seed set in selfing maize. *Z. Pflanzzüchtg.* 89:55-59.
- Erb, N., H. D. Zinsmeister, G. Lehmann and D. Michely, 1981. Prussic-acid content of cereals in temperate climate regions. *Z. Lebensm. Unters. Forsch.* 173:176-179.
- Erkeev, M. I., and G. R. Kudoyarova, 1981. Activity of the protein synthesizing system in heterotic maize hybrids. *Fiziol. Rast.* 28:880-882.
- Erkeev, M. I., and G. R. Kudoyarova, 1981. Isolation of polysomes from corn leaves. *Sov. Plant Physiol.* 28:794-796.
- Esen, A., 1982. Chromatography of zein on phosphocellulose and sulfopropyl Sephadex. *Cereal Chem.* 59:272-275.
- Esen, A., J. A. Bietz, J. W. Paulis and J. S. Wall, 1982. Tandem repeats in the N-terminal sequence of a proline-rich protein from corn endosperm. *Nature* 296:678-679.
- Ezra, G., E. Krochmal and J. Gressel, 1982. Competition between a thiocarbamate herbicide and herbicide protectants at the level of uptake into maize cells in culture. *Pest. Biochem. Physiol.* 18:107-112.
- Fakorede, M. A. B., and J. J. Mock, 1982. Correlated responses to recurrent selection for grain yield in maize. *Iowa Agr. Home Ec. Ex. St. Res. Bu.* 596:177-208.
- Fauquet, C., and J-C. Thouvenel, 1982. A new viral maize disease in Ivory-Coast, the maize eye spot virus. *C. R. Acad. Sci. Ser. III-Vie* 295:293-298.
- Fedoroff, N., 1982. Introduction to transposable controlling elements in maize. Pp. 203-211 in W. F. Sheridan, ed., 1982 (which see).
- Fedoroff, N., S. McCormick and J. Mauvais, 1980. Molecular studies on the controlling elements of maize. *Carnegie Inst. Wash. Year Book* 1979:51-62.
- Feix, G., P. Langridge and U. Wienand, 1981. Cloning of DNA sequences coding for zein proteins of maize. Pp. 73-84 in *Genetic Engineering in the Plant Sciences*, N. J. Panopoulos, ed., Praeger Publ., New York.
- Filichkin, S. A., 1982. Changes in the cell structure during formation of maize opaque-2. *Fiziol. Biokhim. Kult. Rast.* 14:119-122.
- Findley, W. R., J. K. Knoke and R. Louie, 1982. 1980 virus tolerance ratings for corn strains grown in Ohio. *SEA Publ. ARR No. NC-9:5-*
- Flavell, R. B., 1981. The analysis of plant genes and chromosomes by using DNA cloned in bacteria. *Philos. Trans. R. Soc. Lond. B. Biol. Sci.* 292:579-588.
- Fonturbel, M. T., and A. Ordas, 1981. Mass selection for early silking time in 2 maize (*Zea mays*) populations. *Genet. Iber.* 33:225-235.

- Fornasari, E., N. Di Fonzo, F. Salamini, R. Reggiani and C. Soave, 1982. Floury-2 and opaque-7 interaction in the synthesis of zein polypeptides. *Maydica* 27:185-189.
- Foster, J. G., and J. L. Hess, 1982. Oxygen effects on maize leaf superoxide dismutase and glutathione reductase. *Phytochemistry* 21:1527-1532.
- Fox, T. D., and C. J. Leaver, 1981. The *Zea mays* mitochondrial gene coding cytochrome oxidase subunit II has an intervening sequence and does not contain TGA codons. *Cell* 26:315-324.
- Francis, T. R., and A. S. Hamill, 1980. Inheritance of maize seedling tolerance toalachlor. *Can. J. Plant Sci.* 60:1045-1047.
- Friedemann, P., and P. A. Peterson, 1982. The *Uq* controlling-element system in maize. *Mol. Gen. Genet.* 187:19-29.
- Frova, C., M. Sari and E. Ottaviano, 1981. Genic expression in the haploid phase in maize. *Atti Ass. Genet. Ital.* 27:181-186.
- Fuwa, H., D. V. Glover, Y. Sugimoto, Y. Ikawa and T. Takaya, 1982. Some properties of starches of opaque-2, sugary-2 opaque-2, and waxy opaque-2 mutants of two broad-based synthetic cultivars of maize. *J. Nutr. Sci. Vitaminol.* 28:127-138.
- Galatis, B., 1982. The organization of microtubules in guard cell mother cells of *Zea mays*. *Can. J. Bot.* 60:1148-1166.
- Galinat, W. C., 1982. Maize breeding and its raw material. Pp. 331-334 in W. F. Sheridan, ed., 1982 (which see).
- Ganchev, K. D., B. J. Stefanov and A. V. Dencheva, 1982. Isolation of pure glutelin components from maize seed endosperm and determination of their N-terminal groups. *Dokl. Bolg. Akad. Nauk* 35:641-644.
- Garwood, D. L., and S. F. Vanderslice, 1982. Carbohydrate composition of alleles at the sugary locus in maize. *Crop Sci.* 22:367-371.
- Gatenby, A. A., and J. A. Castleton, 1982. Amplification of maize ribulose biphosphate carboxylase large subunit synthesis in *E. coli* by transcriptional fusion with the lambda-N operon. *Mol. Gen. Genet.* 185:424-429.
- Gavazzi, G., M. L. Racchi and C. Tonelli, 1982. Somatic cell genetics of maize: In vivo and in vitro expressions of maize mutants. Pp. 401-404 in W. F. Sheridan, ed., 1982 (which see).
- Gengenbach, B., and D. Pring, 1982. Isolation of revertants from CMS-T by tissue culture techniques. Pp. 257-261 in W. F. Sheridan, ed., 1982 (which see).
- Genovesi, A. D., and G. B. Collins, 1982. In vitro production of haploid plants of corn via anther culture. *Crop Sci.* 22:1137-1144.
- Gentile, J. M., G. J. Gentile, J. Bultman, R. Sechriest, E. D. Wagner and M. J. Plewa, 1982. An evaluation of the genotoxic properties of insecticides following plant and animal activation. *Mutat. Res.* 101:19-29.
- Geric, I., M. Zlokolica, C. Geric and Z. Vidojevic, 1981. Phosphoenolpyruvate carboxylase and ribulose-diphosphate carboxylase activity in corn inbreds and hybrids. *Arhiv Polj. Nauke* 42:9-22.
- Gerlach, W. L., A. J. Pryor, E. S. Dennis, R. J. Ferl, M. M. Sachs and W. J. Peacock, 1982. cDNA cloning and induction of the alcohol dehydrogenase gene (*Adh1*) of maize. *Proc. Natl. Acad. Sci. USA* 79:2981-2985.
- Golbeck, J. H., I. F. Martin, B. R. Velthuys and R. Radmer, 1981. A critical reassessment of the photosystem-II content in bundle sheath chloroplasts of young leaves of *Zea mays*. Pp. 533-546 in *Photosynthesis, Vol. 5, Chloroplast Development*, G. Akoyunoglou, ed., Balban Internatl. Sci. Svcs., Philadelphia.
- Golubovskaya, I. N., and V. G. Urbach, 1981. Allelic relationships between maize meiotic mutations with similar disturbances of meiosis. *Genetika* 17:1975-1982.
- Gomez, L. A., J. G. Rodriguez, C. G. Poneleit and D. F. Blake, 1982. Preference and utilization of maize endosperm variants by the rice weevil. *J. Econ. Entomol.* 75:363-367.
- Gonzalez Diez, J. F., M. Rodriguez Pascual and A. Suarez Suarez, 1981. Comparative assays of maize varieties rich in essential amino acids using varieties of the common grain. 1. Yield of kernels and lysine content. *Zootecnia* 30:87-92.
- Goodman, M. M., C. W. Stuber and K. J. Newton, 1982. Isozyme loci in maize. Pp. 53-60 in W. F. Sheridan, ed., 1982 (which see).
- Gottlieb, L. D., 1982. Conservation and duplication of isozymes in plants. *Science* 216:373-380.
- Gracen, V. E., 1982. Role of genetics in etiological phytopathology. *Annu. Rev. Phytopathol.* 20:219-233.
- Gracen, V. E., 1982. Types and availability of male sterile cytoplasm. Pp. 221-224 in W. F. Sheridan, ed., 1982 (which see).
- Graf, L., E. Roux, E. Stutz and H. Kossel, 1982. Nucleotide sequence of a *Euglena gracilis* chloroplast gene coding for the 16S rRNA: Homologies to *E. coli* and *Zea mays* chloroplast 16S rRNA. *Nucl. Acid. Res.* 10:6369-6382.
- Grebenskikov, I., 1980. Possible estimation of F2 performances per plant for single cross hybrids in maize based on corresponding data for F1 and mean values for the parents. *Kulturpflanze* 28:239-256.
- Greber, R. S., 1982. Maize sterile stunt--a delphacid transmitted rhabdovirus disease affecting some maize genotypes in Australia. *Aust. J. Agr. Res.* 33:13-24.
- Green, C. E., 1981. Tissue culture in grasses and cereals. Pp. 107-122 in *Genetic Engineering for Crop Improvement*, K. O. Rachie and J. M. Lyman, eds., Rockefeller Foundation, USA.
- Green, C. E., and C. A. Rhodes, 1982. Plant regeneration in tissue cultures of maize. Pp. 367-372 in W. F. Sheridan, ed., 1982 (which see).
- Greyson, R. I., D. B. Walden and W. J. Smith, 1982. Leaf and stem heteroblasty in *Zea*. *Bot. Gaz.* 143:73-78.
- Grier, S. L., and D. W. Davis, 1982. Artificial versus natural infestation for evaluation of kernel and stalk damage in maize by the European corn borer. *J. Amer. Soc. Hort. Sci.* 107:519-521.
- Griesbach, R. J., R. L. Malmborg and P. S. Carlson, 1982. An improved technique for the isolation of higher plant chromosomes. *Plant Sci. Lett.* 24:55-60.
- Gu, M., and B. Yuan, 1980. Giemsa banding in meiotic chromosomes of the pollen mother cell of maize (*Zea mays*). *Acta Genetica Sinica* 7:36-39.
- Gu, M. G., and X. Q. Zhang, 1982. Studies on the karyotype of pollen callus clone of maize in subcultures. *Xexue Tongbao* 27:551-554. (In Chinese)
- Gu, M. G., X. Q. Zhang, Z. Y. Cao and C. Y. Guo, 1982. Differentiation potential and chromosome stability of pollen callus of maize in subcultures. *Acta Botanica Sinica* 24:319-325. (In Chinese)
- Gudkov, I. N., and D. M. Grodzinsky, 1982. Cell radiosensitivity variation in synchronously-dividing root meristems of *Pisum sativum* L. and *Zea mays* L. during the mitotic cycle. *Int. J. Radiat. Biol.* 41:401-409.
- Guikema, J. A., and L. A. Sherman, 1981. Electrophoretic profiles of cyanobacterial membrane polypeptides showing heme-dependent peroxidase activity. *Biochim. Biophys. Acta* 637:189-201.

- Guillemaut, P., and J. H. Weil, 1982. The nucleotide sequence of the maize and spinach chloroplast isoleucine transfer RNA encoded in the 16S to 23S rDNA spacer. *Nucl. Acid. Res.* 10:1653-1660.
- Guo, C. Y., and Z. Y. Cao, 1982. The effect of genotype. *Hereditas* (Beijing) 4:8-10. (In Chinese)
- Gupta, P. R. L., V. S. Rathore, S. V. Saradhi and G. K. Garg, 1982. Tryptophan synthase in maize (*Zea mays* L.). I. In vivo and in vitro demonstration of enzyme activity. *Biochem. Biophys. Res. Commun.* 109:121-129.
- Guthrie, W. D., E. B. Lillehoj, W. W. McMillian, D. Barry, W. F. Kwolek, A. O. Franz, E. A. Catalano, W. A. Russell and N. W. Widstrom, 1981. Effect of hybrids with different levels of susceptibility to second-generation European corn borers on aflatoxin contamination in corn. *J. Agr. Food Chem.* 29:1170-1172.
- Gutierrez, C., A. Guerrero, P. Castanera and J. V. Torres, 1982. A high-performance liquid chromatographic method for quantitation of DIMBOA and MBOA in maize plant extract. *J. Agr. Food Chem.* 30:1258-1260.
- Hadjinov (see Khadzhinov)
- Hagemann, R., and T. Borner, 1981. V. Extranuclear inheritance: Plastid genetics. *Fortschr. Bot.* 43:159-173.
- Hagerman, A. E., and R. L. Nicholson, 1982. High-performance liquid chromatographic determination of hydroxycinnamic acids in the maize mesocotyl. *J. Agr. Food Chem.* 30:1098-1101.
- Hallauer, A. R., and W. A. Russell, 1981. Use of index selection in recurrent selection programs in maize. *Euphytica* 30:611-618.
- Hamid, A. H., J. E. Ayers and R. R. Hill, Jr., 1982. Host x isolate interactions in corn inbreds inoculated with *Cochliobolus carbonum* race 3. *Phytopathology* 72:1169-1173.
- Hamid, A. H., J. E. Ayers and R. R. Hill, Jr., 1982. The inheritance of resistance in corn to *Cochliobolus carbonum* race 3. *Phytopathology* 72:1173-1177.
- Harlan, J. R., 1980. Crop monoculture and the future of American agriculture. Pp. 225-250 in *Future of American Agriculture as a Strategic Resource*, S. S. Batie and R. G. Healy, eds., Conservation Foundation, Washington, DC.
- Harms, C. T., 1982. Maize and cereal protoplasts--facts and perspectives. Pp. 373-384 in W. F. Sheridan, ed., 1982 (which see).
- Havel, L., and F. J. Novak, 1981. In vitro pollination of maize (*Zea mays* L.)--proof of double fertilization. *Plant Cell Reports* 1:
- Hedden, P., B. O. Phinney, R. Heupel, D. Fujii, H. Cohen, P. Gaskin, J. MacMillan and J. E. Graebe, 1982. Hormones of young tassels of *Zea mays*. *Phytochemistry* 21:391-393.
- Hedman, K. D., and C. D. Boyer, 1982. Gene dosage at the amylose-extender locus of maize: Effects on the levels of starch branching enzymes. *Biochem. Genet.* 20:483-492.
- Heidrichsobrinho, E., 1982. Isoenzymes as genetic markers to identify 9 corn lines. *Pesq. Agrop. Brasil.* 17: 281-286.
- Hibberd, C. A., D. G. Wagner, R. L. Schemm, E. D. Mitchell, Jr., R. L. Hintz and D. E. Weibel, 1982. Nutritive characteristics of different varieties of sorghum and corn grains. *J. Anim. Sci.* 55:665-672.
- Hibberd, K. A., and C. E. Green, 1982. Inheritance and expression of lysine plus threonine resistance selected in maize tissue culture. *Proc. Nat. Acad. Sci. USA* 79:559-563.
- Hicks, D. R., and R. H. Peterson, 1981. Effect of corn variety and soybean rotation on corn yield. *Proc. 36th Annu. Corn & Sorghum Res. Conf.* 36:89-94.
- Hinesly, T. D., D. E. Alexander, K. E. Redborg and E. L. Ziegler, 1982. Differential accumulations of cadmium and zinc by corn hybrids grown on soil amended with sewage sludge. *Agron. J.* 74:469-474.
- Hoisington, D. A., M. G. Neuffer and V. Walbot, 1982. Disease lesion mimics in maize. I. Effect of genetic background, temperature, developmental age, and wounding on necrotic spot formation with *Les1*. *Develop. Biol.* 93:381-388.
- Hooker, A. L., 1982. Genetic diversity of maize: Disease resistance. Pp. 361-366 in W. F. Sheridan, ed., 1982 (which see).
- Hooker, A. L., and Y.-K. Tsung, 1980. Relationship of dominant genes in corn for chlorotic lesion resistance to *Helminthosporium turcicum*. *Plant Dis.* 64:387-388.
- Hopke, P. K., M. J. Plewa, J. B. Johnston, D. Weaver, S. G. Wood, R. A. Larson and T. Hinesly, 1982. Multi-technique screening of Chicago, Illinois, U.S.A. municipal sewage sludge for mutagenic activity. *Environ. Sci. Technol.* 16:140-147.
- Hopkins, W. G., 1982. Formation of chloroplast pigments in a temperature-sensitive, virescent mutant of maize. *Can. J. Bot.* 60:737-740.
- Hristov (see Khristov)
- Hsu, L. Z., 1981. Studies on the genetical characteristics of H and H progenies of maize pollen-plants. *Bull. Bei-Zhuan Agriculture College* 2:1-6.
- Hsu, S.-Y., and P. A. Peterson, 1981. Relative stage duration of microsporogenesis in maize. *Iowa State J. Res.* 55:351-373.
- Huan, H., and S. Qiquan, 1981. Advances in plant cell and tissue culture in China. *Adv. Agron.* 34:1-13.
- Humphreys, T., 1982. Cytoplasmic pH of maize scutellum cells. *Phytochemistry* 21:2165-2171.
- Hussein, T. A., 1982. Seedling growth and cold tolerance of different maize genotypes. *Acta Agron. Acad. Sci. Hung.* 31:50-58.
- Hussein, T. A., and B. I. Pozsar, 1982. Maize inbred lines, single and double crosses as affected by cold wave and plant growth regulator. *Acta Agron. Acad. Sci. Hung.* 31:295-304.
- Iams, K. P., and J. H. Sinclair, 1982. Mapping the mitochondrial DNA of *Zea mays*-ribosomal gene localization. *Proc. Nat. Acad. Sci.* 79:5926-5929.
- Iahi, I., and K. Dorffling, 1982. Changes in abscisic acid and proline levels in maize varieties of different drought resistance. *Physiol. Plant.* 55:129-135.
- Iino, M., 1982. Action of red light on indole-3-acetic acid status and growth in coleoptiles of etiolated maize seedlings. *Planta* 156:21-32.
- Ikawa, Y., D. V. Glover, M. Iida, T. Takaya and H. Fuwa, 1982. Properties of residual starches of sugary-2 and sugary-2 opaque-2 maize following amylase hydrolysis. *Stärke* 34:44-49.
- Il'in, V. S., 1980. Creation of early ripening maize hybrids. *Sel. Semonovod.* 18-19.
- Ivanko, S., I. Strapac, E. Sulik and J. Smutny, 1982. Electrophoretic picture of native and reduced-alkylized prolamine proteins in maize grain. *Rostl. Vyroba* 28:979-984.
- Ivanov, V. B., P. A. Chel'tsov and R. N. Shchelokov, 1981. Two biological effects of platinum II complexes with different neutral ligands. *Izv. Akad. Nauk SSR Ser. Biol.* 551-557.

- Ivanova, I., and N. Khristov, 1981. Trends and methods in maize breeding for disease resistance. *Rasteniev'd Nauki* 18:119-129.
- Jain, S. K., 1982. Quantitative maize genetics. (Review of Quantitative Genetics in Maize Breeding, A. R. Hallauer and J. B. Miranda, Iowa State Univ. Press, Ames, 1981.) *J. Hered.* 73:315-316.
- Jarvis, J. L., R. L. Clark and W. D. Guthrie, 1982. Effect of second-generation European corn borers on resistance of maize to Diplodia maydis. *Phytopathology* 72:1149-1152.
- Jarvis, J. L., W. D. Guthrie and J. C. Robbins, 1981. Evaluation of maize plant introductions for resistance to black cutworm larvae. *Maydica* 26:219-225.
- Jelenic, D., D. Nikolic and V. H.-T. Sukalovic, 1982. Activity of some enzymes and isoenzyme structure in kernel of inbred lines and their maize hybrids. *Arhiv Poljopr. Nauke* 43:209-222.
- Jenkins, C. L. D., L. J. Rogers and M. W. Kerr, 1982. Glycollate oxidase inhibition and its effect on photosynthesis and pigment formation in Zea mays. *Phytochemistry* 21:1859-1864.
- Johannessen, C. L., 1982. Domestication process of maize continues in Guatemala. *Econ. Bot.* 36:84-99.
- Johnson, D. Q., and W. A. Russell, 1982. Genetic variability and relationships of physical grain-quality traits in the BSSS population of maize. *Crop Sci.* 22:805-809.
- Johnson, G. R., 1982. Two-locus theory in recurrent selection for general combining ability in maize. *Theor. Appl. Genet.* 61:279-283.
- Johnson, R. R., and M. P. Herrero, 1981. Corn pollination under moisture and high temperature stress. *Proc. 36th Annu. Corn & Sorghum Res. Conf.* 36:66-77.
- Johri, M. M., and E. H. Coe, 1982. Genetic approaches to meristem organization. Pp. 301-310 in W. F. Sheridan, ed., 1982 (which see).
- Jones, R. N., and H. Rees, 1982. B Chromosomes. Academic Press, New York.
- Jong, S. K., J. L. Brewbaker and C. H. Lee, 1982. Effects of solar radiation on the performance of maize in 41 successive monthly plantings in Hawaii. *Crop Sci.* 22:13-18.
- Jordan, D. B., and W. L. Ogren, 1981. Species variation in the specificity of ribulose biphosphate carboxylase oxygenase. *Nature* 291:513-515.
- Jovicevic, B., and M. Sultan, 1981. Methods of study of Fusarium infection in corn breeding programs. *Zast. Bilja* 31:341-346.
- Kaan, F., S. Rautou, A. Panouille, A. Boyat and M. Tersac, 1982. A study of European maize populations for certain agronomic traits. *Agronomie* 2:577-582.
- Kalir, A., and T. J. Flowers, 1982. The effect of salts on malate dehydrogenase from leaves of Zea mays. *Phytochemistry* 21:2189-2194.
- Kalman, L., A. Parducz and L. Pinter, 1982. Rb cytoplasmic male sterility in maize: Fertility restoration and histology of anther development. *Maydica* 27:1-10.
- Kaneko, K., and B. A. Aday, 1981. Inheritance of resistance to Philippine downy mildew Peronosclerospora philippinensis of maize. *Philipp. Agric.* 63:353-378.
- Karaiwanow, G., R. Marquard and E. W. Petrovskij, 1982. Fat and carotene contents in corn as well as fatty acid patterns and tocopherol contents in oil of Russian maize lines and maize hybrids with very different color of endosperm. *Fette Seifen Anstrichm.* 84:251-255.
- Karoly, C. W., J. C. Woodman, C.-H. Chen, M. L. Alleman, M. A. Johns and M. Freeling, 1982. An annotated bibliography of the Adh genes of maize, from 1966 through 1981, and prediction on the future of classical genetics. Pp. 145-153 in W. F. Sheridan, ed., 1982 (which see).
- Kealey, K. S., and F. V. Kosikowski, 1981. Corn smut as a food source--perspectives on biology, composition and nutrition. *CRC Crit. Rev. Food Sci. Nutr.* 15:321-352.
- Kertesz, Z., 1982. Changes in some composition features of maize hybrids (whole plant) in various phases of plant development. *Novenytermeles* 31:109-118.
- Khadzhinov, M. I., and V. S. Shcherbak, 1981. Current status of corn origin and evolution theory. *S-KH Biol.* 16:530-540.
- Khadzhinov, M. I., V. S. Shcherbak, N. I. Benko, V. P. Gusev, T. B. Sukhorzhevskaya and L. P. Voronova, 1982. Interrelationships between isozymic diversity and combining ability in maize lines. *Maydica* 27:135-150.
- Khare, B. P., S. Chandra and V. K. Sharma, 1982. Relative susceptibility of maize germplasms to red flour beetle, Tribolium castaneum Herbst. *Indian J. Agr. Sci.* 52:228-231.
- Khatab, A. A. S., G. B. El-Saadany and S. A. Mourad, 1977. Relative susceptibility of 5 maize cultivars to the infestation of the lesser cotton leafworm Spodoptera exigua in Egypt. *Bull. Soc. Entomol. Egypte* 75-78.
- Khavkin, E. E., and N. N. Varakina, 1981. Respiration of maize cells in batch suspension culture as compared to the intact root tip and coleoptile. *Z. Pflanzenphysiol.* 104:419-430.
- Khristov, K., and P. Khristova, 1980. Genetic analysis of maize mutant lines induced by accelerated seed aging. *Genet. Sel.* 13:420-428.
- Khristov, K., and P. Khristova, 1981. Genetic characterization of the mutant maize line XM-532. *Genet. Sel.* 14:346-353.
- Khristov, K., and P. Khristova, 1981. Intergeneric maize x teosinte hybrids and chemical mutagenesis in maize breeding. *Rasteniev'd Nauki* 18:3-14.
- Khristov, K., P. Khristova and I. Genova, 1982. Inheritance of quantitative characters in maize and variability of the genetic parameters. I. Plant height. *Genet. Sel.* 15:58-66.
- Khristov, K., P. Khristova and I. Genova, 1982. Inheritance of quantitative characters in maize and variability of the genetic parameters. II. Length of ear and grain and number of rows. *Genet. Sel.* 15:200-210.
- Kidd, G. H., and M. E. Davis, 1982. Maize RNA polymerases and in vitro transcription. Pp. 169-176 in W. F. Sheridan, ed., 1982 (which see).
- Kim, B. D., R. J. Mans, M. F. Conde, D. R. Pring and C. S. Levings, 1982. Physical mapping of homologous segments of mitochondrial episomes from S male-sterile maize. *Plasmid* 7:1-14.
- King, S. B., and G. E. Scott, 1981. Genotypic differences in maize to kernel infection by Fusarium moniliforme. *Phytopathology* 71:1245-1247.
- King, S. B., and G. E. Scott, 1982. Development of southern rust on maize at different stages of maturity. *Plant Dis.* 66:477-480.
- King, S. B., and G. E. Scott, 1982. Field inoculation techniques to evaluate maize for reaction to kernel infection by Aspergillus flavus. *Phytopathology* 72:782-785.
- King, S. B., and G. E. Scott, 1982. Southern rust and corn maturity. *Agric. Res.* 30:14.

- Kiniry, J. R., and M. E. Keener, 1982. An enzyme kinetic equation to estimate maize development rates. *Agron. J.* 74:115-118.
- Kitchen, L. M., W. W. Witt and C. E. Rieck, 1981. Inhibition of delta amino levulinic acid synthesis by glyphosate. *Weed Sci.* 29:571-577.
- Knoke, J. K., R. J. Anderson, W. R. Findley, R. Louie, J. J. Abt and D. T. Gordon, 1981. The reaction of sweet corn hybrids to maize dwarf mosaic virus strains and maize chlorotic dwarf virus. *Ohio Agr. Res. Develop. Ctr. Res. Bull.* 1135:3-22.
- Koch, K. E., C. L. Tsui, L. E. Schrader and O. E. Nelson, 1982. Source-sink relations in maize mutants with starch deficient endosperms. *Plant Physiol.* 70:322-325.
- Koller, B., H. Delius and T. A. Dyer, 1982. The organization of the chloroplast DNA in wheat and maize in the region containing the LS gene. *Eur. J. Biochem.* 122:17-23.
- Konarev, V. G., Sh. Ya. Gilyazetdinov and R. R. Akhmetov, 1981. Heterosis and its manifestation according to biochemical data and molecular genetics. *S-KH Biol.* 16:380-386.
- Konis, Y., E. Harel and S. Klein, 1981. The appearance of a new soluble polypeptide and the prevention of thylakoid assembly during inhibition of chlorophyll synthesis in maize leaves. Pp. 581-590 in *Photosynthesis, Vol. 5, Chloroplast Development*, G. Akoyunoglou, ed., Balban Internatl. Sci. Svcs., Philadelphia.
- Kostandi, S. F., Y. S. Koraiem, A. Kamara and M. A. Omar, 1981. Effect of phenols in host-pathogen-interaction of maize (*Zea mays* L.)--*Cephalosporium maydis* system. *Agrochimica* 25:367-376.
- Kostyshin, S. S., M. M. Baran and L. T. Oplachko, 1982. Different intensity of protein biosynthesis in heterosis maize hybrids. *Fiziol. Biokhim. Kult. Rast.* 14:123-126.
- Kovacevic, V., B. Bertic, V. Trogrlic and M. Seput, 1982. Genotype influence on the iron concentration in the grain of corn hybrids. *J. Plant Nutr.* 5:605-608.
- Krebbbers, E. T., I. M. Larrinua, L. McIntosh and L. Bogorad, 1982. The maize chloroplast genes for the β and ϵ subunits of the photosynthetic coupling factor CF1 are fused. *Nucl. Acid. Res.* 10:4985-5002.
- Kumar, V., and H. S. Shetty, 1982. A new ear and kernel rot of maize caused by *Trichoderma viride* Pers. ex Fries. *Curr. Sci.* 51:620-624.
- Kuzin, A. M., V. V. Medvedkova, M. E. Vagabova and Yu. A. Ivanovskii, 1981. Synergism of the joint effect of gamma radiation and radiotoxins. 2. Development DNA synthesis and chromosome aberrations. *Radiobiologiya* 21:348-351.
- Kuznetsova, G. A., M. G. Kuznetsova and G. M. Grineva, 1981. Some features of water exchange and the anatomical and morphological structure of maize plants under flooding conditions. *Fiziol. Rast.* 28:340-348.
- Lai, Y.-K., and J. G. Scandalios, 1982. Purification and characterization of an endogenous inhibitor of alcohol dehydrogenase from maize root. *Plant Sci. Lett.* 27:7-20.
- Lai, Y.-K., J. M. Chandlee and J. G. Scandalios, 1982. Purification and characterization of three non-allelic alcohol dehydrogenase isozymes in maize. *Biochim. Biophys. Acta* 706:9-18.
- Landi, P., P. Catizone and S. Conti, 1981. Differential response of maize inbreds and hybrids to Eradicane 6E ethyldipropylthiocarbamate plus R-25788. *Riv. Agron.* 15:85-94.
- Landi, P., and T. M. Crosbie, 1982. Response of maize to cold stress during vegetative growth. *Agron. J.* 74:765-767.
- Landry, J., and T. Moureaux, 1981. Physicochemical properties of maize glutelins as influenced by their isolation conditions. *J. Agr. Food Chem.* 29:1205-1211.
- Landry, J., and T. Moureaux, 1982. Distribution and amino acid composition of protein fractions in opaque-2 maize grains. *Phytochemistry* 21:1865-1870.
- Langridge, P., J. A. Pintor-Toro and G. Feix, 1982. Zein genomic clones from maize. Pp. 183-187 in W. F. Sheridan, ed., 1982 (which see).
- Langridge, P., J. A. Pintor-Toro and G. Feix, 1982. Repeated excision and analysis of developing kernels from a single maize ear. Pp. 311-312 in W. F. Sheridan, ed., 1982 (which see).
- Langridge, P., J. A. Pintor-Toro and G. Feix, 1982. Transcriptional effects of the opaque-2 mutation of *Zea mays* L. *Planta* 156:166-170.
- Langridge, P., J. A. Pintor-Toro and G. Feix, 1982. Direction of zein gene transcription in maize genomic clones. *Biochem. Biophys. Res. Commun.* 107:1236-1242.
- Langridge, P., J. A. Pintor-Toro and G. Feix, 1982. Zein precursor mRNAs from maize endosperm. *Mol. Gen. Genet.* 187:432-438.
- Larkins, B. A., A. C. Mason and W. J. Hurkman, 1982. Molecular mechanisms regulating the synthesis of storage proteins in maize endosperm. *CRC Crit. Rev. Food Sci. Nutr.* 16:199-215.
- Larkins, B. A., and K. Pedersen, 1982. Cloning of maize zein genes. Pp. 177-182 in W. F. Sheridan, ed., 1982 (which see).
- Larson, R. L., and R. P. Robles, 1981. P-coumaric acid hydroxylation by a *Zea mays* L. phenolase preparation. *Maydica* 26:199-207.
- Laughnan, J. R., and S. Gabay-Laughnan, 1982. Nuclear control over reversions to male fertility in S male sterile maize. Pp. 239-242 in W. F. Sheridan, ed., 1982 (which see).
- Laughnan, J. R., S. Gabay-Laughnan and J. E. Carlson, 1982. Cytoplasmic male sterile systems in maize and recent approaches to their molecular interpretation. Pp. 225-237 in W. F. Sheridan, ed., 1982 (which see).
- Lauritis, J. A., M. R. Sondahl, A. Y. Ciampi, L. M. Prioli and W. J. DaSilva, 1979. Ultrastructural cytology of callus tissues of normal and a luteus mutant of *Zea mays*. *Rev. Bras. Bot.* 2:117-124.
- Leaver, C. J., and M. W. Gray, 1982. Mitochondrial genome organization and expression in higher plants. *Annu. Rev. Plant Physiol.* 33:373-402.
- Leto, K., 1982. Photosynthetic mutants of maize. Pp. 317-325 in W. F. Sheridan, ed., 1982 (which see).
- Leto, K., and C. Arntzen, 1981. Cation mediated regulation of excitation energy distribution in chloroplasts lacking organized photosystem II complexes. *Biochim. Biophys. Acta* 637:107-117.
- Levi, Y., E. Noymeir and Y. Cohen, 1981. A simulation model of the Northern leaf blight of corn. Pp. 75-82 in *Developments in Arid Zone Ecology and Environmental Quality*, H. I. Shuval, ed., Balban Internatl. Sci. Svcs., Philadelphia.
- Levic, J., and V. Pencic, 1982. Effect of *Helminthosporium carbonum* Ullstrup on yield of maize. *Arhiv Poljopr. Nauke* 43:3-12.
- Li, J., 1980. Plant isozymes and their application to the crop genetic research. *Acta Agronomica Sinica* 6:245-252.

- Li, J., T. Yang and M. Zeng, 1980. Isozymic studies on the origin of cultivated corn. *Acta Genetica Sinica* 7: 223-231.
- Li, J., T. Yang and M. Zeng, 1980. Studies on the heterosis in relationship with isozymes in maize. II. Types of complementary isozymes and their distribution in organs. *Hereditas (Beijing)* 2:4-6.
- Li, Y. X., and J. G. Li, 1982. Studies on the chlorophyll-protein complexes of chloroplast mutants in maize. *Acta Genetica Sinica* 9:344-349.
- Lillehoj, E. B., W. F. Kwolek, W. D. Guthrie, D. Barry, W. W. McMillian and N. W. Widstrom, 1982. Aflatoxin accumulation in preharvest maize kernels--interaction of 3 fungal species, European corn borer and 2 hybrids. *Plant Soil* 65:95-102.
- Lillehoj, E. B., and M. S. Zuber, 1981. Variability in corn hybrid resistance to preharvest aflatoxin contamination. *J. Amer. Oil Chem. Soc.* 58:A970-A972.
- Lima, M., N. Gimenes-Fernandes, J. B. Miranda-Filho and J. C. V. A. Pereira, 1982. Introduction of maize (*Zea mays* L.) germplasm as sources for downy mildew (*Peronosclerospora sorghi*) resistance. *Maydica* 27:159-168.
- Lin, B.-Y., 1982. Association of endosperm reduction with parental imprinting in maize. *Genetics* 100:475-486.
- Lipps, P. E., and R. E. Hite, 1982. *Exserohilum turcicum* virulent on corn with the *Ht* resistance gene in Ohio. *Plant Dis.* 66:397-398.
- Lonquist, J. H., 1982. Maize breeding and future goals. Pp. 327-330 in W. F. Sheridan, ed., 1982 (which see).
- Lorz, H., J. Paszkowski, C. Dierks-Ventling and I. Potrykus, 1981. Isolation and characterization of cytoplasts and mini-protoplasts derived from protoplasts of cultured cells. *Physiol. Plant.* 53:385-391.
- Losyeva, Z. I., 1981. Effect of cytoplasm and nucleus Rf genes on the activity of some enzymes in maize anther mitochondria. *Vyestsi Akad. Navuk BSSR Syer. Biyal. Navuk* 42-44.
- Louie, R., D. T. Gordon, J. K. Knoke, R. E. Gingery, O. E. Bradfute and P. E. Lipps, 1982. Maize white line mosaic virus in Ohio. *Plant Dis.* 66:167-170.
- Lu, C., I. K. Vasil and P. Ozias-Akins, 1982. Somatic embryogenesis in *Zea mays* L. *Theor. Appl. Genet.* 62: 109-112.
- Lucas, E. O., 1981. Remobilization of stem assimilates in maize varieties grown under tropical conditions. *Maydica* 26:287-292.
- Lyubenov, A., 1982. Selective fertilization in maize. *Genet. Sel.* 15:244-249.
- Machado, V. S., 1982. Inheritance and breeding potential of triazine tolerance and resistance in plants. Pp. 257-273 in *Herbicide Resistance in Plants*, H. M. LeBaron and J. Gressel, eds., John Wiley & Sons, Inc., New York.
- Magoja, J. L., 1980. La primera generación (F1) de un híbrido entre teosinte perenne y una línea muy precoz de maíz. *Comunic. Direc. Inv. Univ. Nac. Lomas de Zamora, Año 3, No. 10.*
- Magoja, J. L., 1981. Efecto modificador de genes supresores de la expresión fenotípica del floury-a en maíz. *Rev. Fac. Agron. (UBA)* 2:65-77.
- Magoja, J. L., and G. N. Benito, 1982. Herencia del ciclo evolutivo en híbridos entre teosinte perenne y una línea muy precoz de maíz. *Rev. Univ. Nac. Lomas de Zamora* 1:57-64.
- Magoja, J. L., and G. N. Benito, 1982. Heterosis en híbridos entre teosinte perenne y maíz. *Rev. Fac. Agron. (UBA)* 3:237-245.
- Magoja, J. L., and A. A. Nivio, 1982. Relación específica de las proteínas de reserva en el maíz (*Zea mays*) y sus parientes silvestres. *Rev. Fac. Agron. (UBA)* 3:255-263.
- Magoja, J. L., A. A. Nivio and M. A. Rapela, 1982. Maíces de alta calidad proteica con genotipo normal. *Mendeliana* 5:71-80.
- Magoja, J. L., and I. G. Palacios, 1981. La primera generación (F1) de un híbrido entre maíz y *Tripsacum dactyloides*. *Comunic. Direc. Inv. Univ. Nac. Lomas de Zamora, Año 4, No. 13.*
- Maguire, M. P., 1982. Evidence for a role of the synaptonemal complex in provision for normal chromosome disjunction at meiosis II in maize. *Chromosoma* 84:675-686.
- Makonnen, D., 1982. The effect of pollination on the dry weight, length, and circumference of maize cobs (*Zea mays* L.). *Beitr. Trop. Landwirt. Vet.* 20:263-268.
- Mandoli, D. F., and W. R. Briggs, 1982. Optical properties of etiolated plant tissues. *Proc. Natl. Acad. Sci. USA* 79:2902-2906.
- Mann, C. E., and W. G. Pollmer, 1981. Reciprocal-cross differences between maize hybrids of inbred lines from different gene pools. *Maydica* 26:263-271.
- Mann, C. E., and W. G. Pollmer, 1981. Relationships between maize inbred lines and the reciprocal-cross differences of their hybrids. *Maydica* 26:253-261.
- Mann, C. E., W. G. Pollmer and D. Klein, 1981. Magnitude and stability over environments of reciprocal-cross differences in maize hybrids and their implications on maize breeding. *Maydica* 26:239-252.
- Mans, R. J., 1982. Episomal DNA as a molecular probe of cytoplasmic male sterility in *S Zea mays*. Pp. 263-266 in W. F. Sheridan, ed., 1982 (which see).
- Manuwoto, S., and J. M. Scriber, 1982. Consumption and utilization of 3 maize genotypes by the southern armyworm *Spodoptera eridania*. *J. Econ. Entomol.* 75:163-167.
- Markova, M. D., 1981. Isoenzyme composition on the NAD⁺-dependent glutamate dehydrogenase in cytoplasmic male-sterile lines of maize. *Dokl. Bolg. Akad. Nauk* 34:1177-1180.
- Marks, M. D., and B. A. Larkins, 1982. Analysis of sequence microheterogeneity among zein messenger RNAs. *J. Biol. Chem.* 257:9976-9983.
- Marquez-Sanchez, F., 1982. Modifications to cyclic hybridization in maize with single-eared plants. *Crop Sci.* 22:314-319.
- Marre, M. T., and G. Romani, 1982. Heterotic behavior of the transmembrane electric potential difference in some maize hybrid-parents combinations. *Plant Sci. Lett.* 27:265-273.
- Martin-Tanguy, J., E. Perdrizet, J. Prevost and C. Martin, 1982. Hydroxycinnamic acid amides in fertile and cytoplasmic male sterile lines of maize. *Phytochemistry* 21:1939-1945.
- Maslobrod, S. N., G. E. Komarova, T. N. Vrabii, V. V. Shkolenko, E. N. Krasnobaev and N. N. Lysikov, 1981. Effect of treating maize seeds with a magnetic field on the physiological biochemical state of seeds and shoots. *Izv. Akad. Nauk Mold. SSR Ser. Biol. Khim. Nauk* 5-14.
- Mason, C. E., and K. T. Tracewski, 1982. Diurnal foraging activity for corn pollen by honey bees. *Environ. Entomol.* 11:187-188.
- Mastenbroek, I., C. E. Cohen and J. M. deWet, 1981. Seed protein and seedling isozyme patterns of *Zea mays* and its closest relatives. *Biochem. Syst. Ecol.* 9:179-183.

- McCormick, S., J. Mauvais and N. Fedoroff, 1982. Evidence that the 2 sucrose synthetase genes in maize are related. *Mol. Gen. Genet.* 187:494-500.
- McMillian, W. W., N. W. Widstrom and D. M. Wilson, 1982. Aflatoxin production on various popcorn genotypes. *Agron. J.* 74:156-157.
- McMillian, W. W., N. W. Widstrom and B. R. Wiseman, 1982. Pink scavenger caterpillar (*Pyroderces rileyi*) resistance among selected dent corn hybrids (*Zea mays*). *J. Ga. Entomol. Soc.* 17:93-96.
- McMillin, D. E., and J. G. Scandalios, 1982. Genetic, immunological and gene dosage studies of mitochondrial and cytosolic MDH variants in maize. *J. Hered.* 73:177-182.
- Meeker, R., and K. K. Tewari, 1982. Divergence of transfer RNA genes in chloroplast DNA of higher plants. *Biochim. Biophys. Acta* 696:66-75.
- Menz, K. M., and C. F. Neumeyer, 1982. Evaluation of five emerging biotechnologies for maize. *Bioscience* 32: 675-676.
- Mesterhazy, A., 1982. Resistance of corn to *Fusarium* ear rot and its relation to seedling resistance. *Phytopathol. Z.* 103:218-231.
- Metivier, J., and A. M. Monteiro, 1981. Early seedling growth in normal and opaque-2 *Zea mays* cv. Maya. 1. Composition and mobilization of the endosperm protein reserve. *J. Exp. Bot.* 32:1321-1332.
- Metz, J. G., and D. Miles, 1982. Use of a nuclear mutant of maize to identify components of photosystem II. *Biochim. Biophys. Acta* 681:95-102.
- Miflin, B. J., and S. R. Burgess, 1982. Protein bodies from developing seeds of barley, maize, wheat and peas: The effect of protease treatment. *J. Exp. Bot.* 33:251-260.
- Migguang, G., 1981. Giemsa banding of maize (*Zea mays*) chromosomes. *Acta Genet. Sin.* 8:175-179.
- Migliori, A., and R. Lastra, 1980. Maize virus-like diseases transmitted by the planthopper, *Peregrinus maidis*, in French West Indies. *Ann. Phytopathol.* 12:277-294.
- Mikel, M. A., C. J. D'Arcy, A. M. Rhodes and R. E. Ford, 1982. Effect of maize dwarf mosaic virus infection of sweet corn pollen and silk. *Phytopathology* 72:428-431.
- Miku, V. E., G. E. Komarova and A. I. Rotar, 1980. Genetic variation of lignin content in corn. *Sov. Agric. Sci.* 11-13.
- Miles, J. W., J. W. Dudley, D. G. White and R. J. Lambert, 1981. Response to selection for resistance to 4 diseases in 2 corn populations. *Crop Sci.* 21:980-983.
- Milinko, I., O. Gyulavari, J. Tatrai and L. Farady, 1982. Some recent results of investigations on the dwarf mosaic virus resistance in maize. *Növénytermelés* 31:333-340.
- Milivojevic, D., 1981. Development of the structure of dimorphic chloroplasts of *Zea mays* in the presence of blue and red light. Pp. 895-904 in *Photosynthesis, Vol. 5, Chloroplast Development*, G. Akoyunoglou, ed., Balban Internatl. Sci. Svcs., Philadelphia.
- Miller, P. D., 1982. Maize pollen: Collection and enzymology. Pp. 279-293 in W. F. Sheridan, ed., 1982 (which see).
- Miranda, J. B., R. Vencovsky and A. R. Hallauer, 1982. Correlation between population means under selfing and full-sibbing. Two populations. *Brazilian J. Genet.* 5:299-311.
- Miranda, V., N. R. Baker and S. P. Long, 1981. Limitations of photosynthesis in different regions of the *Zea mays* leaf. *New Phytol.* 89:179-190.
- Mirzinski-Stefanovic, L., 1981. Effect of herbicides on the growth rate and yield of maize inbred lines. *Zast. Bilja* 32:71-78.
- Misevic, D., 1982. Genetic variability and selection indices for grain yield, oil and protein content, and kernel weight in synthetic population of maize. *Arhiv Poljopr. Nauke* 43:71-94.
- Misra, S., and A. Oaks, 1982. Enzymes of nitrogen assimilation during seed development in normal and high lysine mutants in maize (*Zea mays*, W64A). *Can. J. Bot.* 59:2735-2738.
- Misra, S., A. Oaks, K. W. Joy and M. McLimont, 1981. Enzymes of asparagine catabolism in the developing endosperm. *Can. J. Bot.* 59:1444-1448.
- Mitev, S., 1981. Inheritance of some characters following hybridization of early and mid early inbred maize lines. *Rasteniev'd Nauki* 18:22-29.
- Mladenova, Y. I., and M. V. Karadimova, 1981. Nitrogen-15 uptake by whole plants and root callus cultures of inbred maize lines and their F1 hybrids. *C. R. Acad. Bulg. Sci.* 34:419-422.
- Modena, S. A., E. H. Coe and L. L. Darrah, 1982. Inbreds vs. hybrids vs. random mating populations: Categories of maize (*Zea mays* L.) and their suitability for certain experiments. Pp. 31-36 in W. F. Sheridan, ed., 1982 (which see).
- Mogford, D. J., 1981. The frequency distribution of B chromosomes in a South African sweet corn cultivar Golden-Bantam. *J. S. Afr. Bot.* 47:765-768.
- Moll, R. H., M. Motto and J. F. de Toledo, 1981. Prediction and inheritance of prolific expression in maize hybrids. *Maydica* 26:273-285.
- Moreno-Gonzalez, J., and A. R. Hallauer, 1982. Combined S2 and crossbred family selection in full sib reciprocal recurrent selection. *Theor. Appl. Genet.* 61:353-358.
- Moro, J. R., V. N. Filho, R. T. Vianna and E. E. G. e Gama, 1981. New maize germplasm introductions in Brazil. *Pesq. Agrop. Bras.* 16:867-882.
- Morot-Gaudry, J. F., J. Farineau, J. P. Rocher and E. Jolivet, 1981. Influence of genotype on the 1st steps of the C-4 photosynthetic pathways in maize. *Agronomie* 1:739-744.
- Motto, M., and M. Perenzin, 1982. Index selection for grain yield and protein improvement in an opaque-2 synthetic maize population. *Z. Pflanzenzüchtg.* 89:47-54.
- Motto, M., and F. Salamini, 1981. Genetic basis of physiological traits related to maize productivity. *Riv. Agron.* 15:3-20.
- Moura, C. B., and W. J. Da Silva, 1981. Effects of *Helminthosporium turcicum* Pass. on agronomic traits of some maize families. *Pesq. Agrop. Bras.* 16:651-658.
- Moura, C. B., and W. J. Da Silva, 1981. Genetic effects of maize inbred lines resistance to *Helminthosporium turcicum* Pass. *Pesq. Agrop. Bras.* 16:319-324.
- Mueller, S. C., 1982. Cellulose-microfibril assembly and orientation in higher plant cells with particular reference to seedlings of *Zea mays*. Pp. 87-104 in *Cellulose and Other Natural Polymer Systems: Biogenesis, Structure, and Degradation*, R. M. Brown, ed., Plenum Publ. Corp., New York.

- Mullet, J. E., K. Leto and C. J. Arntzen, 1981. Structural organization and development of the light-harvesting pigment proteins for photosystem I and photosystem II. Pp. 557-568 in *Photosynthesis, Vol. 5, Chloroplast Development*, G. Akoyunoglou, ed., Balban Internat. Sci. Svcs., Philadelphia.
- Murthy, A. R., N. B. Kajjari and J. V. Goud, 1981. Diallel analysis of yield maturity components in maize. *Indian J. Genet. Plant Breed.* 41:30-33.
- Muschinek, G., G. I. Garab, L. A. Mustardy, A. Faludi-Daniel and K. Gorog, 1981. Biochemical characteristics of Linuron phytotoxicity in maize. Pp. 575-584 in *Photosynthesis*, vol. 6, G. Akoyunoglou, ed., Balban Internat. Sci. Serv., Philadelphia.
- Mustardy, L. A., T. T. Vu and A. Faludi-Daniel, 1982. Stomatal response and photosynthetic capacity of maize (*Zea mays*) leaves at low temperature: A study on varietal differences in chilling sensitivity. *Physiol. Plant.* 55:31-34.
- Mynbaev, T. T., G. I. Balan and H. N. Ishalina, 1982. The new dwarf radiomutant with the terminal location of ears. I. The nature of heritability of the mutant character. *Genetika* 18:1359-1362.
- Nagl, W., 1981. Genetics replication. *Progr. Bot.* 43:119-131.
- Naspolini, V., E. E. G. e Gama, R. T. Vianna and J. R. Moro, 1981. General and specific combining ability for yield in a diallel cross among 18 maize populations (*Zea mays* L.). *Rev. Brasil. Genetica* 4:571-578.
- Nault, L. R., D. T. Gordon, V. D. Damsteegt and H. H. Iltis, 1982. Response of annual and perennial teosintes (*Zea*) to 6 maize viruses. *Plant Dis.* 66:61-62.
- Nawar, A. A., A. A. Abul-Naas and M. E. Gomaa, 1981. Heterosis and general vs. specific combining ability among inbred lines of corn. *Egypt. J. Genet. Cytol.* 10:19-30.
- Nelson, O. E., 1982. Genetics and plant breeding in relation to stress tolerance. In *Crop Reactions to Water and Temperature Stress in Humid, Temperate Climates*, C. D. Raper, Jr. and P. J. Kramer, eds., Westview Press, Boulder, Colorado.
- Neuffer, M. G., 1982. Growing maize for genetic purposes. Pp. 19-30 in W. F. Sheridan, ed., 1982 (which see).
- Neuffer, M. G., 1982. Mutant induction in maize. Pp. 61-64 in W. F. Sheridan, ed., 1982 (which see).
- Niblett, C. L., J. H. Tsai and B. W. Falk, 1981. Virus and mycoplasma diseases of corn in Florida. *Proc. 36th Annu. Corn & Sorghum Res. Conf.* 36:78-88.
- Nitsch, C., S. Andersen, M. Godard, M. G. Neuffer and W. F. Sheridan, 1982. Production of haploid plants of *Zea mays* and *Pennisetum* through androgenesis. Pp. 69-91 in *Variability in Plants Regenerated from Tissue Culture*, E. D. Earle and Y. Demarly, eds., Praeger Publ., New York.
- Nivito, A. A., and J. L. Magoja, 1982. Herencia de las proteínas de reserva en híbridos recíprocos entre maíz y teosinte perenne. *Rev. Univ. Nac. Lomas de Zamora* 1:47-55.
- Nocera-Przybecka, D., and P.-E. Pilet, 1981. Activity gradients of transaminases in roots. *Z. Pflanzenphysiol.* 102:463-466.
- Nowakowski, J., and R. A. Morse, 1982. The behavior of honey bees in sweet corn fields in New York state. *Amer. Bee J.* 122:13-16.
- O'Leary, M. H., 1982. Phosphoenolpyruvate carboxylase: An enzymologist's view. *Annu. Rev. Plant Physiol.* 3: 297-316.
- Olejniczak, J., and H. Patyna, 1981. Mutagenic effect of N-methyl-N-nitrosourea (MNUA) and sodium azide (SA) in maize line S-75 (*Zea mays*). *Genet. Pol.* 22:289-294.
- Olsen, R. A., and J. C. Brown, 1980. Factors related to iron uptake by dicotyledonous and monocotyledonous plants. I. pH and reductant. *J. Plant Nutr.* 6:629-646.
- Ordas, A., 1981. Forage potential of some maize (*Zea mays*) populations and their possible utilization in a program of hybrid selection. *An. Edafol. Agrobiol.* 40:327-336.
- Osman, O., A. A. Ismail and M. A. Abul-Fadl, 1981. Genetic variability and correlation studies in maize (*Zea mays* L.). *Egypt. J. Genet. Cytol.* 10:69-76.
- Osterman, J. C., and D. Schwartz, 1981. Analysis of a controlling-element mutation at the *Adh* locus of maize. *Genetics* 99:267-273.
- Ottaviano, E., and A. Camussi, 1981. Phenotypic and genetic relationships between yield components in maize. *Euphytica* 30:601-609.
- Ottaviano, E., M. Sari Gorla and E. Pe, 1982. Male gametophytic selection in maize. *Theor. Appl. Genet.* 63: 249-254.
- Palacios, I. G., 1982. Contenido de ADN nuclear en híbridos F1 entre teosinte perenne y maíz. *Rev. Fac. Agron. (UBA)* 3:247-253.
- Palfi, G., and Z. Palfi, 1982. A rapid method for the determination of fertility of maize pollen with the proline-isatin reaction. *Maydica* 27:107-111.
- Palfi, G., and L. Pinter, 1980. Determination of the drought resistance of inbred maize lines with proline test. *Acta Univ. Szeged Acta Biol.* 26:109-116.
- Palfi, G., L. Pinter and Zs. Palfi, 1981. The proline content and fertility of the pollen of inbred maize lines. *Acta Botanica* 27:179-188.
- Palma, M. S., S. A. Rodrigues and A. Rossi, 1982. Kinetic properties of acid phosphatase from scutella of germinating maize seeds. *Phytochemistry* 21:1245-1248.
- Palmer, J. D., and W. F. Thompson, 1980. Higher plant chloroplast and mitochondrial DNA. *Carnegie Inst. Wash. Yearbook* 1979:120-123.
- Pan, D., and K. H. Tan, 1982. Regulation of thermostable phosphoenolpyruvate acid carboxylase from maize leaves by malate, citrate (activators) and fructose 1, 6-bisphosphate, acetyl-CoA (inhibitors). *Plant Sci. Lett.* 27: 69-75.
- Pasupuleti, C. V., and W. C. Galinat, 1982. *Zea diploperennis*. I. Its chromosomes and comparative cytology. *J. Hered.* 73:168-170.
- Patel, K. R., 1981. Cytochemical studies on the relationship between DNA/histone ratio and endogenous RNA level in differentiating metaxylem cells. *Nucleus* 23:67-69.
- Patterson, E. B., 1982. The mapping of genes by the use of chromosome aberrations and multiple gene marker stocks. Pp. 85-88 in W. F. Sheridan, ed., 1982 (which see).
- Paulis, J. W., 1982. Recent developments in corn protein research. *J. Agr. Food Chem.* 30:14-20.
- Payak, M. M., and R. C. Sharma, 1980. An inventory and bibliography of maize diseases in India. Division of Mycology & Plant Pathology, Indian Agricultural Research Institute, New Delhi.

- Payak, M. M., and R. C. Sharma, 1982. Jaundice stalk rot--a new disease problem of maize. *Curr. Sci. India* 51: 754.
- Pe, E., and A. Camussi, 1982. Yield components and plant traits in summer-sowing maize. *Maydica* 27:169-184.
- Pe, E., E. Ottaviano and A. Camussi, 1982. Structural analysis of relationships between ear and plant traits in maize. *Maydica* 27:41-53.
- Pedersen, K., J. Devereux, D. R. Wilson, E. Sheldon and B. A. Larkins, 1982. Cloning and sequence analysis reveal structural variation among related zein genes in maize. *Cell* 29:1015-1026.
- Pencic, V., and J. Levic, 1981. Contribution to the study of the effect of the opaque-2 gene on the resistance of maize to *Helminthosporium turcicum*, *Helminthosporium carbonum* and *Kabatiella zeae*. *Arhiv Poljopr. Nauke* 42:277-282.
- Pencic, V., and J. Levic, 1981. Contribution to the resistance of local maize varieties to northern leaf blight (*Helminthosporium turcicum* Pass.). *Arhiv Poljopr. Nauke* 42:381-388.
- Pereira, P. A. A., J. I. Baldani, J. Dobereiner and C. A. Neyra, 1981. Nitrate reduction and nitrogenase activity in excised corn roots. *Can. J. Bot.* 59:2445-2449.
- Peters, D. W., D. B. Shank and W. E. Nyquist, 1982. Root-pulling resistance and its relationship to grain yield in F1 hybrids of maize. *Crop Sci.* 22:1112-1114.
- Pfahler, P. L., M. Wilcox, D. L. Mulcahy and D. A. Knauff, 1982. In vitro pollination and pollen tube growth of maize (*Zea mays* L.) pollen. *Acta Bot. Neer.* 31:105.
- Phillips, R. L., 1982. Genetic engineering overview. *Sugar Azucar* 77:17.
- Phillips, R. L., and A. S. Wang, 1982. In situ hybridization with maize meiotic cells. Pp. 121-122 in W. F. Sheridan, ed., 1982 (which see).
- Phinney, B. O., and C. Spray, 1982. Chemical genetics and the gibberellin pathway in *Zea mays* L. Pp. 101-110 in *Plant Growth Substances*, P. F. Wareing, ed., Academic Press, New York.
- Phipps, R. H., F. F. Weller and A. Cooper, 1982. A comparison between the accumulation of dry matter, chemical composition, and nutritive value of isogenic sterile and fertile forage maize. *Maydica* 27:27-40.
- Pinter, L., 1982. Trends of above-ear and below-ear leaf areas and of grain yield per unit leaf area in maize (*Zea mays* L.) hybrids with different genotypes. *Acta Agron. Acad. Sci. Hung.* 31:201-206.
- Pinter, L., J. Nemeth and Z. Klein, 1981. Evaluation of maize (*Zea mays* L.) hybrids utilized as whole plant. *Növénytermelés* 30:481-492.
- Pinter, L., G. Palfi and L. Kalman, 1981. Individual analyses of maize (*Zea mays* L.) plants for increased drought resistance. *Z. Pflanzenzüchtg.* 87:260-263.
- Pintor-Toro, J. A., P. Langridge and G. Feix, 1982. Isolation and characterization of maize genes coding for zein proteins of the 21000 dalton size class. *Nucl. Acid. Res.* 10:3845-3850.
- Plewa, M. J., 1982. Specific-locus mutation assays in *Zea mays*: A report of the U. S. Environmental Protection Agency Gene-Tox Program. *Mutat. Res.* 99:317-337.
- Poethig, R. S., 1982. Maize--the plant and its parts. Pp. 9-18 in W. F. Sheridan, ed., 1982 (which see).
- Pohl, R. W., and M. C. Albertsen, 1981. Interspecific hybrids of *Zea mays* and *Z. diploperennis*. *Iowa State J. Res.* 55:257-260.
- Polisetty, R., and R. H. Hageman, 1982. Studies on nitrate uptake by solution grown corn (*Zea mays* L.) genotypes. *Biol. Plant.* 24:117-123.
- Pollacek, M., 1982. Effect of genetic background on recombination in maize. *Agronomie* 2:617-620.
- Polowick, P. L., and R. I. Greyson, 1982. Anther development, meiosis and pollen formation in *Zea* tassels cultured in defined liquid medium. *Plant Sci. Lett.* 26:139-145.
- Pomeranz, Y., 1981. Genetic factors affecting protein content and composition of cereal grains. Pp. 174-205 in *World Review of Nutrition and Dietetics*, Vol. 36, Human Nutrition and Diet, G. H. Bourne, ed., S. Karger, Basel, Switzerland.
- Pommer, C. V., and L. F. Razera, 1980. Effect of intrapopulation selection on the quality of seeds from 2 maize populations and their intervarietal hybrid. *Bragantia* 39:185-194.
- Popova, J., 1980. Possibilities for developing high protein maize hybrids. *Rasteniev'd Nauki* 17:11-17.
- Popova, J., and A. Pöpov, 1981. Developing 2 ear maize lines. *Rasteniev'd Nauki* 18:15-21.
- Poulsen, C., 1981. Comments on the structure and function of the large subunit of the enzyme ribulose biphosphate carboxylase oxygenase. *Carlsberg Res. Commun.* 46:259-278.
- Preiss, J., 1982. Regulation of the biosynthesis and degradation of starch. *Annu. Rev. Plant Physiol.* 33:431-454.
- Prodhan, H. S., S. Dana and K. R. Sarkar, 1981. The genetic variability of an experimental maize composite in relation to its future improvement. *Genetika (Yugosl.)* 13:41-47.
- Prodhan, H. S., S. Dana and K. R. Sarkar, 1981. Genetic variability in two heterozygous maize populations. *Indian J. Genet.* 41:349-353.
- Pryor, A., and J. L. Huppatz, 1982. Purification of maize alcohol dehydrogenase and competitive inhibition by pyrazoles. *Biochem. Int.* 4:431-438.
- Pylneva, P. N., and A. P. Levitsky, 1982. Studies in activity of inhibitor trypsin in grain of ordinary and high-lysine maize. *Fiz. Biokhim. Kult. Rast.* 14:378-382.
- Racchi, M. L., N. Lurani and G. Gavazzi, 1981. Root tip cultures as a means to detect nutritional mutants in maize. *Atti Ass. Genet. Ital.* 27:323-326.
- Rama Devi, G., and H. Polasa, 1982. Mycotoxins from fungi on maize. *Curr. Sci. India* 51:751.
- Randolph, L. F., 1982. Maize evolution--a manuscript. *Econ. Bot.* 36:193-194.
- Rapela, M. A., 1981. Possible intervention of homoserine dehydrogenase in changes of sensitivity to threonine in the roots of floury-a maize plantlets. *Phyton* 41:63-69.
- Rapela, M. A., 1982. Growth inhibition by lysine plus threonine--comparative inhibition profiles between floury-a and normal maize embryos. *Plant Cell Physiol.* 23:285-292.
- Ratkovic, S., M. Denic and G. Lahajnar, 1982. Kinetics of water imbibition by seed--why normal and opaque-2 maize kernels differ in their hydration properties. *Period. Biol.* 84:180-182.
- Rayburn, A. L., and J. R. Gold, 1982. A procedure for obtaining mitotic chromosomes from maize. *Maydica* 27: 113-121.
- Raymundo, A. D., and A. L. Hooker, 1982. Single and combined effects of monogenic and polygenic resistance on certain components of northern corn leaf blight development. *Phytopathology* 72:99-103.

- Reger, B. J., and J. James, 1982. Pollen germination and pollen tube growth of sorghum when crossed to maize and pearl millet. *Crop Sci.* 22:140-144.
- Reifschneider, F. J. B., and C. A. Lopes, 1982. Bacterial top and stalk rot of maize in Brazil. *Plant Dis.* 66: 519.
- Remison, S. U., 1982. Time of leaf blade removal on the performance of maize. *Maydica* 27:123-133.
- Remison, S. U., and J. M. Fajemisin, 1982. Comparative growth of maize cultivars with different leaf orientation. *J. Agr. Sci.* 99:61-66.
- Reyes, F. G. R., G. W. Varseveld and M. C. Kuhn, 1982. Sugar composition and flavor quality of high sugar (shrunken) and normal sweet corn. *J. Food Sci.* 47:753-755.
- Rhodes, A. M., E. E. Carey and D. B. Dickinson, 1982. Illinois sweet corn inbreds with the su-se genotype. *Hortscience* 17:411.
- Rhodes, P. R., and S. D. Kung, 1981. Chloroplast DNA isolation purity achieved without nuclease digestion. *Can. J. Biochem.* 59:911-915.
- Rivin, C. J., E. A. Zimmer and V. Walbot, 1982. Isolation of DNA and DNA recombinants from maize. Pp. 161-164 in W. F. Sheridan, ed., 1982 (which see).
- Robertson, D. S., 1982. Chlorophyll and carotenoid mutants. Pp. 313-315 in W. F. Sheridan, ed., 1982 (which see).
- Robertson, D. S., and A. Faludi-Daniel, 1982. Tests establishing the genetic relationship between carotenoid biosynthetic mutants of maize studied in Hungary and the United States. *J. Hered.* 73:473.
- Robertson, D. S., and P. N. Mascia, 1981. Tests of 4 controlling-element systems of maize for mutator activity and their interaction with Mu mutator. *Mutat. Res.* 8:283-289.
- Robinson, J. F., J. A. Klun, W. D. Guthrie and T. A. Brindley, 1982. European corn borer leaf feeding resistance: A simplified technique for determining relative differences in concentrations of 6-methoxybenzoxazolinone (Lepidoptera: Pyralidae). *J. Kans. Entomol. Soc.* 55:297-301.
- Robinson, J. F., J. A. Klun, W. D. Guthrie and T. A. Brindley, 1982. European corn borer (Lepidoptera: Pyralidae) leaf feeding resistance: Dimboa bioassays. *J. Kans. Entomol. Soc.* 55:357-364.
- Rood, S. B., and D. J. Major, 1981. Diallel analysis of leaf number, leaf development rate, and plant height of early maturing maize. *Crop Sci.* 21:867-872.
- Rood, S. B., and D. J. Major, 1981. Diallel analysis of the photoperiodic response of maize. *Crop Sci.* 21: 875-878.
- Rozenfeld, J., 1981. Resistance of maize to Helminthosporium carbonum. *Zast. Bilja* 31:347-356.
- Rubenstein, I., 1982. The zein multigene family. Pp. 189-195 in W. F. Sheridan, ed., 1982 (which see).
- Ryadchikova, E. A., 1980. Protein content in opaque-2 maize grain germ and endosperm during inbreeding. *S-KH Biol.* 15:712-715.
- Saccomani, M., G. Cacco and G. Ferrari, 1981. Efficiency of the 1st steps of sulfate utilization by maize hybrids in relation to their productivity. *Physiol. Plant.* 53:101-104.
- Sachs, M. M., H. Lorz, E. S. Dennis, A. Elizur, R. J. Ferl, W. L. Gerlach, A. J. Pryor and W. J. Peacock, 1982. Molecular genetic analysis of the maize anaerobic response. Pp. 139-144 in W. F. Sheridan, ed., 1982 (which see).
- Sadehdel-Moghaddam, M., P. J. Loesch, Jr., W. J. Wiser and A. R. Hallauer, 1982. Interrelationships and inheritance of protein quality and agronomic traits in an opaque-2 synthetic of maize (Zea mays L.). *Iowa State J. Res.* 57:85-96.
- Safeulla, K. M., and H. S. Shetty, 1980. Sorghum downy mildew in Asia: Assessment of present knowledge and future research needs. Pp. 173-183 in Sorghum Diseases: A World Review, G. D. Bengtson, ed., ICRISAT, Andhra Pradesh, India.
- Sagaral, E. G., and C. L. Foy, 1982. Responses of several corn (Zea mays) cultivars and weed species to EPTC with and without the antidote R-25788. *Weed Science* 30:64-69.
- Salamini, F., 1980. Controlling elements at the opaque-2 locus of maize: Their involvement in the origin of spontaneous mutation. *Cold Spring Harbor Symp.* 45:467-476.
- Salamini, F., M. Bremenkamp and R. Marotta, 1982. Origin of the inactive regulatory element Bg-in of the maize o2m(r)-Bg system of controlling elements. *Heredity* 49:111-115.
- Salamini, F., and C. Soave, 1982. Zein: Genetics and biochemistry. Pp. 155-160 in W. F. Sheridan, ed., 1982 (which see).
- Salem, A. M., A. A. El-Sayed and M. E. E. Gresi, 1981. Inheritance of resistance to the late wilt of maize caused by Cephalosporium maydis. *Egypt. J. Genet. Cytol.* 10:9-18.
- Sallee, P. J., 1982. Prefixation and staining of the somatic chromosomes of corn. P. 119 in W. F. Sheridan, ed., 1982 (which see).
- Sanderson, J. B., T. B. Daynard and M. Tollenaar, 1981. A mathematical model of the shape of corn leaves. *Can. J. Plant Sci.* 61:1009-1011.
- Santos, J. P., and J. E. Foster, 1981. Preference and reproductivity of maize weevil, as resistance factors in some corn populations and inbreds. *Pesq. Agrop. Bras.* 16:769-776.
- Satiat-Jeunemaitre, B., 1981. The cell walls of the 2 epidermis of maize coleoptile--texture and growth. *Ann. Sci. Natur.-Bot. Biol. Veg.* 2-3:163-176.
- Saxena, V. K., S. C. Sharma, N. S. Malhi, A. S. Khehra and W. R. Kapoor, 1981. Variability for resistance to leaf blight caused by Helminthosporium maydis race 0 in a composite variety of maize (Zea mays). *Cereal Res. Commun.* 9:253-258.
- Scandalios, J. G., 1982. Developmental genetics of maize. *Annu. Rev. Genetics* 16:85-112.
- Scandalios, J. G., and J. A. Baum, 1982. Regulatory gene variation in higher plants. *Adv. Genet.* 21:347-370.
- Schowitz, R., and H. Ziegler, 1982. Exudation of water-soluble vitamins and of some carbohydrates by intact roots of maize seedlings (Zea mays L.) into a mineral nutrient solution. *Z. Pflanzenphysiol.* 107:7-14.
- Schwartz, D., 1982. Amylose distribution in the starch granule of maize endosperm. *Maydica* 27:54-57.
- Schwartz, D., 1982. Tissue-specific regulation of gene function: Presetting and erasure. *Proc. Nat. Acad. Sci. USA* 79:5991-5992.
- Schwartz, D., and C. S. Echt, 1982. The effect of Ac dosage on the production of multiple forms of Wx protein by the wx-m9 controlling element mutation in maize. *Mol. Gen. Genet.* 187:410-413.
- Scott, G. E., and E. Rosenkranz, 1982. A new method to determine the number of genes for resistance to maize dwarf mosaic in maize. *Crop Sci.* 22:756-761.

- Sears, P. B., 1982. Fossil maize pollen in Mexico (letter). *Science* 216:932-934.
- Selim, A. R., H. A. El-Itriby and A. H. Shehata, 1981. The nature of a top-cross tester in relation to general combining ability evaluation of homozygous maize lines. *Egypt. J. Genet. Cytol.* 10:275-.
- Semuguruka, G. H., W. A. Compton, C. Y. Sullivan and M. A. Thomas, 1981. Some measures of temperature response in corn (*Zea mays* L.). *Maydica* 26:209-218.
- Shabanov, A. S., 1980. Heterosis in simple interlinear maize hybrids with multiple ears. *Izv. Akad. Nauk Az. SSR Ser. Biol. Nauk* 44-52.
- Shannon, J. C., 1982. A search for rate-limiting enzymes that control crop production. *Iowa State J. Res.* 56: 307-322.
- Shannon, J. C., 1982. Maize endosperm cultures. Pp. 397-400 in W. F. Sheridan, ed., 1982 (which see).
- Sharma, S. C., A. S. Khehra, V. K. Saxena, B. S. Dhillon and N. S. Malhi, 1982. Note on screening of germplasm of maize against *Drechslera maydis* (*Helminthosporium maydis*). *Indian J. Agr. Sci.* 52:341.
- Shaw, D. V., A. L. Kahler and R. W. Allard, 1981. A multilocus estimator of mating system parameters in plant populations. *Proc. Natl. Acad. Sci. USA* 78:1298-1302.
- Sheldon, E. L., 1982. The construction of maize DNA libraries. Pp. 197-201 in W. F. Sheridan, ed., 1982 (which see).
- Shepherd, N., Z. Schwarz, U. Wienand, H. Sommer, H. Saedler, K. Hahlbrock, F. Kreuzaler, H. Ragg and P. A. Peterson, 1982. Genomic DNA clones of *Zea mays*. Pp. 217-219 in W. F. Sheridan, ed., 1982 (which see).
- Shepherd, N. S., Z. Schwarz-Sommer, U. Wienand, H. Sommer, B. Deumling, P. A. Peterson and H. Saedler, 1982. Cloning of genomic fragment carrying the insertion element *Cin 1* of *Zea mays*. *Mol. Gen. Genet.* 188:266-271.
- Sheridan, W. F., ed., 1982. *Maize for Biological Research*. Plant Mol. Biol. Assn., Charlottesville, Virginia.
- Sheridan, W. F., 1982. Introduction to maize for biological research. Pp. 1-7 in W. F. Sheridan, ed., 1982 (which see).
- Sheridan, W. F., 1982. Maps, markers and stocks. Pp. 37-52 in W. F. Sheridan, ed., 1982 (which see).
- Sheridan, W. F., 1982. Black Mexican sweet corn: Its use for tissue cultures. Pp. 385-388 in W. F. Sheridan, ed., 1982 (which see).
- Sheridan, W. F., 1982. Anther culture of maize. Pp. 389-396 in W. F. Sheridan, ed., 1982 (which see).
- Sheridan, W. F., and M. G. Neuffer, 1982. Maize developmental mutants: Embryos unable to form leaf primordia. *J. Hered.* 73:318-329.
- Shieh, W. J., and M. B. McDonald, 1982. The influence of seed size, shape and treatment on inbred seed corn quality. *Seed Sci. Technol.* 10:307-314.
- Shimamoto, K., and O. E. Nelson, 1981. Isolation and characterization of aminopterin-resistant cell lines in maize. *Planta* 153:436-442.
- Shortess, D. K., 1982. A Fortran computer program for calculating linkage intensities from F2 data. *J. Hered.* 73:70.
- Shumaker, K. M., R. W. Allard and A. L. Kahler, 1982. Cryptic variability at enzyme loci in 3 plant species, *Avena barbata*, *Hordeum vulgare* and *Zea mays*. *J. Hered.* 73:86-90.
- Singh, I., A. F. Lusby and P. M. McGuire, 1982. Mutagenicity of HPLC fractions from extracts of AAtrex-treated corn. *Environ. Mutagen.* 4:45-54.
- Singh, T. P., S. A. Akhter, S. K. Prasad and M. D. Jha, 1982. Note on adaptability of some Yugoslav maize hybrids in Bihar. *Indian J. Agr. Sci.* 52:339-340.
- Slife, F. W., 1981. Chemical tolerance of inbred lines and an update on weed control. *Proc. 36th Annu. Corn & Sorghum Res. Conf.* 36:61-65.
- Slovin, J. P., and E. M. Tobin, 1981. Glyphosine, a plant growth regulator, affects chloroplast membrane proteins. *Biochim. Biophys. Acta* 637:177-184.
- Smith, C. S., J. J. Mock and T. M. Crosbie, 1982. Variability for morphological and physiological traits associated with barrenness and grain yield in the maize population, Iowa Upright Leaf Synthetic No. 1. *Crop Sci.* 22:828-832.
- Smith, D. R., and R. Toth, 1982. Histopathological changes in resistant (*rhm*) corn inoculated with *Helminthosporium maydis* race-0. *Mycopathologia* 77:83-88.
- Smith, J. S. C., M. M. Goodman and T. A. Kato, 1982. Variation within teosinte. 2. Numerical analysis of knob data. *Econ. Bot.* 36:100-112.
- Smith, O. S., A. R. Hallauer and W. A. Russell, 1981. Use of index selection in recurrent selection programs in maize (*Zea mays*). *Euphytica* 30:611-618.
- Smith, O. S., A. R. Hallauer, W. A. Russell and T. M. Crosbie, 1981. Use of selection indices in maize improvement and hybrid development programs. *Proc. 36th Annu. Corn & Sorghum Res. Conf.* 36:95-103.
- Soave, C., and F. Salamini, 1982. Zein proteins: A multigene family from maize endosperm. *Qual. Plant.* 31: 191-201.
- Soave, C., L. Tardani, N. DiFonzo and F. Salamini, 1981. Zein level in maize endosperm depends on a protein under control of the opaque-2 and opaque-6 loci. *Cell* 27:403-410.
- Sorenson, J. C., 1982. Catalase: A system for studying the molecular basis of developmental gene regulation. Pp. 135-138 in W. F. Sheridan, ed., 1982 (which see).
- Soto, P. E., I. W. Buddenhagen and V. L. Asnani, 1982. Development of streak virus-resistant maize populations through improved challenge and selection methods. *Ann. Appl. Biol.* 100:539-546.
- Spruill, W. M., Jr., C. S. Levings, III and R. R. Sederoff, 1981. Organization of mitochondrial DNA in normal and Texas male sterile cytoplasms of maize. *Develop. Genet.* 2:319-336.
- Sreenivasan, T. V., and N. C. Jalaja, 1982. Production of subclones from the callus culture of *Saccharum-Zea* hybrid. *Plant Sci. Lett.* 24:255-259.
- Srivastava, H. K., 1981. Intergenomic interaction, heterosis, and improvement of crop yield. *Adv. Agron.* 34: 117-195.
- Stamp, P., 1981. Activities of photosynthetic enzymes and pigment contents in leaves of maize seedlings at cool temperature as influenced by application of phytohormones. *Angew. Bot.* 55:409-418.
- Stamp, P., 1981. Activities of photosynthetic enzymes in leaves of maize seedlings (*Zea mays* L.) at changing temperature and light intensities. *Angew. Bot.* 55:419-428.
- Stamp, P., 1982. Relations of pigment contents and activities of photosynthetic enzymes with the fatty acid composition of membrane lipids in leaves of maize seedlings depending on genotypes and changing temperatures. *Angew. Bot.* 56:191-200.

- Stangland, G. R., and W. A. Russell, 1981. Variability within single crosses of S2 and S8 inbred lines of maize. *Maydica* 26:227-238.
- Starlinger, P., 1982. Transposable genetic elements in bacteria and in maize. Pp. 345-372 in *Molecular Biology of Plant Tumors*, G. Kahl and J. S. Schell, eds., Academic Press, New York.
- Steinmetz, A., E. J. Gubbins and L. Bogorad, 1982. The anticodon of the maize chloroplast gene for tRNA^{Leu}_{UAA} is split by a large intron. *Nucl. Acid. Res.* 10:3027-3038.
- Stern, D. B., T. A. Dyer and D. M. Lonsdale, 1982. Organization of the mitochondrial ribosomal RNA genes of maize. *Nucl. Acid. Res.* 10:3333-3340.
- Stern, D. B., and D. M. Lonsdale, 1982. Mitochondrial and chloroplast genomes of maize have a 12-kilobase DNA sequence in common. *Nature* 299:698-702.
- Stiles, J. I., 1982. Restriction endonuclease cleavage map of the maize chloroplast genome. Pp. 275-276 in W. F. Sheridan, ed., 1982 (which see).
- St. Martin, S. K., P. J. Loesch, J. T. Demopulos-Rodriguez and W. J. Wiser, 1982. Selection indices for the improvement of opaque-2 maize. *Crop Sci.* 22:478-485.
- Stoloff, L., and F. B. Lillehoj, 1981. Effect of genotype (open-pollinated vs. hybrid) and environment on preharvest aflatoxin contamination of maize grown in southeastern United States. *J. Amer. Oil Chem. Soc.* 58: A976-A980.
- Strommer, J. N., S. Hake, J. Bennetzen, W. C. Taylor and M. Freeling, 1982. Regulatory mutants of the maize *Adh1* gene caused by DNA insertions. *Nature* 300:542-544.
- Struik, P. C., 1982. Effect of a switch in photoperiod on the reproductive development of temperate hybrids of maize. *Neth. J. Agr. Sci.* 30:69-84.
- Stuber, C. W., M. M. Goodman and R. H. Moll, 1982. Improvement of yield and ear number resulting from selection at allozyme loci in a maize population. *Crop Sci.* 22:737-740.
- Styles, E. D., and O. Ceska, 1981. *P* and *R* control of flavonoids in bronze coleoptiles of maize. *Can. J. Genet. Cytol.* 23:691-704.
- Sukalovic, V. H.-T., 1982. Investigation of the activity of the more significant enzymes of the oxydoreductase and transaminase system in the leaf of the normal and opaque-2 genotype of maize during ontogenesis. *Arhiv Poljopr. Nauke* 43:127-144.
- Sukhorzhevskaya, T. B., and E. E. Khavkin, 1981. Isoenzymes of 6-phosphogluconate dehydrogenase in maize (*Zea mays*). *Dokl. Biol. Sci.* 257:167-171.
- Sundquist, W. B., K. M. Menz and C. F. Neumeyer, 1982. A technology assessment of commercial corn production in the United States. *Minn. Agr. Exp. Sta. Bull.* 546:1-80.
- Surico, G., and J. E. DeVay, 1982. Effect of syringomycin and syringotoxin produced by *Pseudomonas syringae* pv. *syringae* on structure and function of mitochondria isolated from holcus spot resistant and susceptible maize lines. *Physiol. Plant Pathol.* 21:39-54.
- Swank, J. C., F. E. Below, R. J. Lambert and R. H. Hageman, 1982. Interaction of carbon and nitrogen metabolism in the productivity of maize. *Plant Physiol.* 70:1185-1190.
- Tarr, J. B., and J. Arditti, 1982. Niacin biosynthesis in seedlings of *Zea mays*. *Plant Physiol.* 69:553-556.
- Thakur, P. S., and V. K. Rai, 1982. Dynamics of amino acid accumulation of 2 differentially drought resistant *Zea mays* cultivars in response to osmotic stress. *Environ. Exp. Bot.* 22:221-226.
- Thakur, P. S., and V. K. Rai, 1982. Effect of water stress on protein content in 2 maize cultivars differing in drought resistance. *Biol. Plant.* 24:96-100.
- Thompson, D. L., 1982. Grain yield of two synthetics of corn after seven cycles of selection for lodging resistance. *Crop Sci.* 22:1207-1210.
- Ting, Y. C., and M. G. Gu, 1982. Meiotic chromosome behavior of diploid perennial teosinte and its hybrid with maize. *Acta Genet. Sin.* 9:26-32.
- Tkemaladze, G. Sh., 1981. Effect of 2,4-D on activity of glutamate and malate dehydrogenases in pea and corn seedlings. *Sov. Plant Physiol.* 28:745-751.
- Todorov, G., 1981. Expression of heterosis in F1 maize crosses in respect to plant growth, photosynthesizing leaf area and dry matter accumulation. *Rasteniev'd Nauki* 18:30-36.
- Tomov, N., V. Vulichinkov, S. Mitev, P. Khristova, Y. Popova, K. Khristov and G. T. Getov, 1981. New maize hybrids. *Rasteniev'd Nauki* 18:13-19.
- Tonelli, C., C. Dierks-Ventling and G. Gavazzi, 1981. Metabolism of proline, glutamate and ornithine in proline mutant shoots of *Zea mays*. *Atti Ass. Genet. Ital.* 27:395-399.
- Tonelli, C., M. L. Racchi, G. Gavazzi and G. Beretta, 1982. In vitro expression of proline-requiring mutants in *Zea mays* L. *Z. Pflanzenphysiol.* 107:437-442.
- Torigoe, Y., and H. Kurihara, 1981. Developmental morphology and yield determining process of maize. *Jpn. Agr. Res. Quart.* 15:85-91.
- Toth, R., and D. R. Smith, 1982. Histopathological changes in susceptible corn inoculated with *Helminthosporium maydis* race-0. *Mycopathologia* 77:75-82.
- Truelove, B., and J. R. Hensley, 1982. Methods of testing for herbicide resistance. Pp. 117-131 in *Herbicide Resistance in Plants*, H. M. LeBaron and J. Gressel, eds., John Wiley & Sons, Inc., New York.
- Tuschall, D. M., and L. C. Hannah, 1982. Altered maize endosperm ADP-glucose pyrophosphorylases from revertants of a shrunken-2-dissociation allele. *Genetics* 100:105-111.
- Twumasi-Afriyie, S., and R. B. Hunter, 1982. Evaluation of quantitative methods for determining stalk quality in short-season corn genotypes. *Can. J. Plant Sci.* 62:55-60.
- Twumasi-Afriyie, S., and R. B. Hunter, 1982. Lodging-enhancing techniques for use on corn performance trials in short-season areas. *Can. J. Plant Sci.* 62:299-304.
- Ullstrup, A. J., 1978. Corn diseases in the United States and their control. *Agr. Handbook No.* 199.
- Van Lammeren, A. A. M., 1981. Early events during embryogenesis in *Zea mays* L. *Acta Soc. Bot. Pol.* 50:289-290.
- Van Staden, J., 1981. Cytokinins in germinating maize caryopses. 1. Transport and metabolism of 8(C-14)t-zeatin applied to the endosperm. *Physiol. Plant.* 53:269-274.
- Van Staden, J., 1981. Cytokinins in germinating maize caryopses. 2. Transport and metabolism of 8(C-14)t-zeatin applied to the embryonic axis. *Physiol. Plant.* 53:275-278.
- Vermeeer, J., and M. E. McCully, 1982. Nuclear and cytoplasmic anomalies in root tips of corn (*Zea mays* L.). *Can. J. Bot.* 60:463-467.

- Vermeer, J., and M. E. McCully, 1982. The rhizosphere in *Zea*: New insight into its structure and development. *Planta* 156:45-61.
- Vianna, R. T., E. E. Gomes e Gama, V. Naspolini Filho, J. R. Moro and R. Vencovskv, 1982. Inbreeding depression of several introduced populations of maize (*Zea mays* L.). *Maydica* 27:151-158.
- Vidal, J., and P. Gadal, 1981. Evidence for *de novo* synthesis of nicotinamide-adenine-dinucleotide phosphate malate dehydrogenase during greening of corn leaves. *Physiol. Veg.* 19:483-489.
- Viotti, A., D. Abildsten, N. Pogna, E. Sala and V. Pirrotta, 1982. Multiplicity and diversity of cloned zein cDNA sequences and their chromosomal localization. *EMBO J.* 1:53-58.
- Vitale, A., E. Smaniotto, R. Longhi and E. Galante, 1982. Reduced soluble proteins associated with maize endosperm protein bodies. *J. Exp. Bot.* 33:439-448.
- Vlakhova, M., and V. V'lev, 1980. The effect of irrigation, planting density and hybrid on growth development and yield of maize in southeastern Bulgaria. *Rasteniev'd Nauki* 17:87-102.
- Vodkin, L. O., and J. G. Scandalios, 1981. Genetic control, developmental expression, and biochemical properties of plant peptidases. Pp. 1-25 in *Isozymes: Current Topics in Biological and Medical Research*, vol. 5, M. C. Rattazzi, J. G. Scandalios and G. S. Whitt, eds., Alan R. Liss, Inc., New York.
- Walbot, V., D. Thompson and E. H. Coe, Jr., 1982. Analysis of development in *Zea mays* using somatic variability in gene expression. Pp. 148-159 in *Variability in Plants Regenerated from Tissue Culture*, E. D. Earle and Y. Demarly, eds., Praeger Publishers, New York.
- Wallin, J. R., D. V. Loonan, L. L. Darrah and C. A. C. Gardner, 1982. 1980 virus tolerance ratings for corn strains grown in Missouri. *SEA Publ. ARR No. NC-9:1-4*.
- Ward, B. L., R. S. Anderson and A. J. Bendich, 1981. The mitochondrial genome is large and variable in a family of plants (*Cucurbitaceae*). *Cell* 25:793-804.
- Warwick, D. R. N., and H. Warren, 1981. Sources of resistance to corn leaf blight. *Pesq. Agrop. Bras.* 16:659-664.
- Weber, D. F., 1982. Using maize monosomics to locate genes to specific chromosomes. Pp. 79-83 in W. F. Sheridan, ed., 1982 (which see).
- Weissinger, A. K., D. H. Timothy, C. S. Levings, W. W. L. Hu and M. M. Goodman, 1982. Unique plasmid like mitochondrial DNAs from indigenous maize races of Latin America. *Proc. Natl. Acad. Sci. USA* 79:1-5.
- Wessel-Beaver, L., and R. J. Lambert, 1982. Genetic control of modified endosperm texture in opaque-2 maize. *Crop Sci.* 22:1095-1098.
- Widstrom, N. W., A. C. Waiss, W. W. McMillian, B. R. Wiseman, C. A. Elliger, M. S. Zuber, R. W. Straub, J. L. Brewbaker, L. L. Darrah, A. R. Henson, J. M. Arnold and J. L. Overman, 1982. Maysin content of silks of 9 maize genotypes grown in diverse environments. *Crop Sci.* 22:953-955.
- Widstrom, N. W., D. M. Wilson and W. W. McMillian, 1981. Aflatoxin contamination of pre-harvest corn (*Zea mays*) as influenced by timing and method of inoculation. *Appl. Environ. Microbiol.* 42:249-251.
- Widstrom, N. W., D. M. Wilson and W. W. McMillian, 1982. Evaluation of sampling methods for detecting aflatoxin contamination in small test plots of maize inoculated with *Aspergillus flavus*. *J. Environ. Qual.* 11:655-657.
- Widstrom, N. W., B. R. Wiseman and W. W. McMillian, 1982. Responses to index selection in maize for resistance to ear damage by the corn earworm. *Crop Sci.* 22:843-846.
- Wielgat, B., and K. Kleczkowski, 1981. Nonhistone chromosomal proteins from gibberellic acid treated maize and pea plants, and their effect on transcription *in vitro*. *Int. J. Biochem.* 13:1201-1204.
- Wienand, U., H. Sommer, Z. Sh. Schwarz, N. Shepherd, H. Saedler, F. Kreuzaler, H. Ragg, E. Fautz, K. Hahlbrock, B. Harrison and P. A. Peterson, 1982. A general method to identify plant structural genes among genomic DNA clones using transposable element induced mutations. *Mol. Gen. Genet.* 187:195-201.
- Wilkes, G., 1982. Wild relatives of the maize gene pool. Pp. 335-339 in W. F. Sheridan, ed., 1982 (which see).
- Williams, J. T., 1980. Maize descriptors. *Internat. Board Plant Gen. Resources, FAO, Rome.* 9 pp.
- Wiseman, B. R., and W. W. McMillian, 1982. Alterations in corn earworm larval feeding behavior and corn ear penetration. *J. Ga. Entomol. Soc.* 17:321-326.
- Wiseman, B. R., N. W. Widstrom and W. W. McMillian, 1981. Effects of 'Antigua 2D-118' resistant corn on fall armyworm feeding and survival. *Fla. Entomol.* 64:515-518.
- Wrigley, C. W., J. C. Autran and W. Bushuk, 1982. Identification of cereal varieties by gel electrophoresis of the grain proteins. Pp. 211-260 in *Advances in Cereal Science and Technology*, vol. 5, Y. Pomeranz, ed., Amer. Assn. Cereal Chem. Inc., St. Paul.
- Wurtele, E. S., P. Hedden and B. O. Phinney, 1982. Metabolism of the gibberellin precursors ent-kaurene, ent-kaurenol, and ent-kaurenal in a cell-free system from seedling shoots of normal maize. *J. Plant Growth Reg.* 1:15-24.
- Wysong, D. S., B. Doupnik, Jr. and L. Lane, 1981. Goss's wilt and corn lethal necrosis--can they become a major problem. *Proc. 36th Annu. Corn & Sorghum Res. Conf.* 36:104-152.
- Yakoleff G., V., E. Hernandez X., C. Rojkind de C. and C. Larralde, 1982. Electrophoretic and immunological characterization of pollen protein of *Zea mays* races. *Econ. Bot.* 36:113-123.
- Yamada, M., 1982. Superiority of pollen from F1 plant in selective fertilization and its implication in maize breeding. *Natl. Inst. Agr. Sci. Bull.* 33:64-119.
- Yang, T. X., M. Q. Zeng and P. Wang, 1981. Analysis on peroxidase isozymes of waxy maize from south China. *Acta Botanica Sinica* 23:110-115. (In Chinese)
- Yudin, B. F., and L. A. Lukina, 1981. Occurrence of double non-reduced egg cells in maize homozygous for the elongate gene. *Dokl. Biol. Sci.* 259:378-380.
- Zaharieva, T., 1982. Differential response of corn genotypes to applied FeEDDHA. *J. Plant Nutr.* 5:897-904.
- Zaitseva, N. A., N. V. Volkova, N. P. Kanivets, L. I. Vasilenok, L. S. Mushketik, T. A. Reingard, L. K. Ostrovskaya and A. A. Yasnikov, 1980. Effect of proton transport inhibitor from maize chloroplasts on photophosphorylating and ATPase activities. *Fiziol. Biokhim. Kul't. Rast.* 12:563-566.
- Zeleneva, I. V., E. V. Savostyanova and E. E. Khavkin, 1982. Enzyme distribution of the cortex and the stele in cytodifferentiating maize seedlings. *Biochem. Physiol. Pflanz.* 177:97-106.
- Zeleneva, I. V., E. V. Savost'yanova and E. E. Khavkin, 1982. Enzyme profiles as a criterion of specialization of the cortex and conducting system in the corn seedling root and mesocotyl. *Sov. Plant Physiol.* 29:10-14.
- Zelitch, I., 1982. The close relationship between net photosynthesis and crop yield. *Bioscience* 32:796-802.
- Zeng, M., T. Yang and P. Wang, 1981. The relative analyses on maize cultivar Menghai-four-row-wax. *Acta Genet. Sin.* 8:91-96.

- Zhang, X. Q., M. G. Gu and Y. C. Ting, 1982. A preliminary study on diploid perennial teosinte. *Hereditas* (Beijing) 3:35-36. (In Chinese).
- Zima, K. I., 1981. Academician Mikhail Ivanovich Hadjinov (1899-1980). *Cereal Res. Commun.* 9:6-8.
- Zimmer, E. A., and K. J. Newton, 1982. A simple method for the isolation of high molecular weight DNA from individual maize seedlings and tissues. Pp. 165-168 in W. F. Sheridan, ed., 1982 (which see).
- Zimmerman, W., and A. Weissbach, 1982. Deoxyribonucleic acid synthesis in isolated chloroplasts and chloroplast extracts of maize. *Biochemistry* 21:3334-3342.
- Zorrilla, H. L., and P. L. Crane, 1982. Evaluation of 3 cycles of full sib family selection for yield in the maize (*Zea mays*) cultivar Colus-o2. *Crop Sci.* 22:10-12.

Cover ye corn ears while ye may,
The pollen's still a-flying;
And this same silk that's out today,
Tomorrow will be dying.

- With apologies to Robert Herrick (1591-1674),
and a citation to Sarah Blanton

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The computer routines for
sorting, manipulation and
verification of symbols
were derived by
Stephen A. Modena

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The computer routines for
 sorting, manipulation and
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Maize For Biological Research

Edited by

William F. Sheridan

Department of Biology
University of North Dakota
Grand Forks, North Dakota

ISBN 0-9608758-0-8

Library of Congress Catalog Card Number 82-60452

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