G. G. Doyle

MAIZE GENETICS COOPERATION

NEWS LETTER

52

March 1, 1978

Department of Agronomy
and
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University of Missouri
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Some sources of general information on maize genetics and cytogenetics:

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

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

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

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

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

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

Dr. G. P. Redei, organizer of the Stadler Genetics Symposia, calls attention to the 10th Symposium, to be held April 7-8, 1978, at Columbia, Missouri:

Arthur Chovnick (University of Connecticut, Storrs): Organization and Expression of the Eukaryotic Gene.

Robert Demars (University of Wisconsin, Madison): Mutations in Human Cells in Relation to Cellular Senescence and Transformation.

Norman H. Giles (University of Georgia, Athens): Expression in Escherichia coli of a Structural Gene in the qa Gene Cluster of Neurospora crassa.

M. M. Green (University of California, Davis): The Genetic Control of Mutation in Drosophila.

C. S. Levings, III (North Carolina State University, Raleigh): The Mitochondrial Genome of Higher Plants.

Barbara McClintock (Carnegie Institution of Washington, Cold Spring Harbor, New York): Mechanisms that Rapidly Reorganize the Maize Genome.

George P. Smith (University of Missouri, Columbia): Non-Darwinian Evolution and the Beard of Life.

Janine Zieg (University of California, La Jolla): Phase Variation in Salmonella: Analysis of the Controlling Element of H2 Gene Expression.

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

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New jeans?
Map them!

I. FOREWORD

Found: An empty manila folder marked "K linkage." A set of all available copies of the News Letter will be awarded to the first person who locates the recombination data.

Your "Secretary" this year edited less extensively the communications submitted by Cooperators, in keeping with the intent of informality (see inside the back cover of volume 51, 1977). The quantity of material submitted was nearly half again as much as in the preceding year; only because of reduction-photocopying of most of the tables and figures is the size within that of previous issues.

Over 680 copies of this volume are being sent to research workers, laboratories and libraries around the world. The costs of preparation, reproduction and mailing of this News Letter are borne by a grant from the National Science Foundation, indispensable support for which we are all grateful. The year-round office workload for the News Letter is lightened by the encouragement and support of the U.S. Department of Agriculture; facilities are provided by the University of Missouri.

Our symbolization system continues to have some difficult flaws, despite 55 years of attention (see letter of R. A. Emerson, March 7, 1923, reproduced on page 146 of this issue). We should consider reinstating numeral one for the first locus of a series (see MNL 49:3, Recommendation 2). At least as much confusion arises now from dropping the one as previously arose from ell/one confusion. Opinions?

Rough Cytogenetic Working Maps with reference data (pp. 129-145) have been derived from materials sent in by cooperators; additional data and suggestions are earnestly requested.

Some materials are still needed for our "archival" files; please see p. 146 and check your attic.

If the Symbol Index, new in this issue, is helpful, please let me know. Suggestions would be welcome.

Publications of interest: Corn and Corn Improvement, 2d edition, edited by G. F. Sprague, has been published by the American Society of Agronomy, Madison, Wisconsin. Discussions in Cytogenetics has been reprinted; see the note from C. R. Burnham in the Minnesota section.

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

Airmail service to addresses outside the U.S. is expensive; we will send the next issue to you by air if we receive $3.00 from you by January 1, 1979.

Deadline for the next issue is January 1, 1979; see inside the back cover for notes on the format for contributed items, and notes for the typist.

I wish to thank M. G. Neuffer for help in planning and developing this volume; Evelyn Bendbow, Sheila McCormick, Marion D. Murray, Manh Nguyen and Donna S. Reeves aided in editing and proofing of copy and in bibliographic work; Mary Nelson gave thorough, precise and diligent attention to composition and the production of final copy.

E. H. Coe, Jr., Geneticist, USDA; Professor of Agronomy
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II. REPORTS FROM COOPERATORS

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Observations on the leaves per plant in opaque-2 and analogous normal maize

It has been a general experience of the maize breeders that the trait, number of leaves per plant of a given genotype, remains either uninfluenced or influenced only to a minor extent by environmental factors. The late maturing maize types, however, have been observed to bear more leaves than those maturing early (S. S. Chase and D. K. Nanda, 1967, Crop Sci. 7:431-432; J. R. Allen et al., 1973, Agron. J. 65:233-235). It has been another experience of the breeders that the opaque-2 maize matures later than the analogous normal maize. An experiment was thus conducted to study if the o2 converted maize genotypes have more leaves on the main stalk than their normal analogues. The experiment comprised 6 o2 converted parental inbred lines, all possible 15 single cross hybrids, 60 three-way cross hybrids, 45 double cross hybrids and all the corresponding normal analogues. The trials were conducted at one location for two years in a split plot design. The results on total leaves per plant including those destroyed early in development (assured by marking the 10th leaf at 12-13 leaf stage), observed on five competitive plants in each row have been presented for each of the two years in Table 1.

It will be seen in the table that there were somewhat fewer leaves per plant in almost all the genotype groups in the wetter and cooler year 1974 than in the year 1973. This difference was, however, limited, on average, to only one leaf per plant.

Table 1. Mean observations on leaves per plant.

Genotype Group	o2	Normal	o2 as % of normal
	1973		
Inbreds	19.29	19.32	99.84
SCs	19.97	20.02	99.75
TCs	20.06	19.98	100.40
DCs	20.00	20.05	99.75
Overall	19.99	19.98	100.05
	1974		
Inbreds	19.27	18.81	102.45
SCs	19.49	19.26	101.19
TCs	19.05	19.24	99.01
DCs	19.01	19.13	99.37
Overall	19.10	19.18	99.58

It may be pointed out that the analyses of variance of these data showed a significant difference only between the various genotype groups, during the year 1973. The variance due to the opacity factors was not significant in either year. Hence it is concluded from the above data that the presence or absence of the o2 gene in the recessive homozygous condition has no influence on the number of leaves per plant, which is otherwise a relatively constant feature of a given genotype. This trait does not seem thus to be a reliable index of maturity in the case of o2 maize, since it has been observed that this genotype had 5.7% more relative grain

moisture at harvest in the o2 recessive homozygotes than in their normal analogues (unpublished data). This difference was statistically significant.

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Semigamy in Coix aquatica

In this abnormal form of fertilization the sperm nucleus penetrates the egg but does not fuse with the egg nucleus. The independent divisions of male and female nuclei will form paternal and maternal chimeral tissues in embryos, seedlings and plants. In Coix aquatica Roxb. (2n=10), in crosses involving stocks marked genetically (purple vs. green seedling base and purple vs. pearly white style) and cytologically (2n=10, 11, 12, etc.), the occasional observation of forking of the seedling stem with the constituents of forking, in some rare cases, differing genetically and cytologically, and resembling the male and female parents led to the identification of the occurrence of the phenomenon of semigamy. The male and female tissues so formed in most cases were 2n-2n, and in a few cases were presumed to be 2n-n since one of the constituents of forking grew slowly and produced fewer and weaker tillers all of which died before flowering. From the occurrence of apospory, apomixis and the functional nature of gametes with n=5 to 10 chromosomes, even on the male side, it might be expected that semigamy is of even wider occurrence in this species but escapes detection unless genetic markers are involved.

Panuganti N. Rao and D. S. Narayana

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The initiation of callus tissue from immature embryos of maize

As indicated in an earlier report (MNL 50:84-86, 1976), immature embryos can respond in either of two ways after excision: one, embryonic growth persists, causing the embryo to flip over, and callus proliferates directly from the developing embryo; two, embryonic growth aborts and, with the embryonic axis remaining in contact with the medium, callus proliferates directly from the scutellum. The origin of the callus is critical since the experiments of Green and Phillips (Crop Sci. 15:417-421, 1975) indicate that only scutellar callus is capable of regeneration.

Several conclusions may be drawn from the data presented in Table 1, which includes experiments conducted since the earlier report. First, if one is not concerned with the origin of the callus and simply observes whether or not callus is produced, then it appears that the auxin concentration has little influence on callus initiation. However, if one does consider the origin of the callus, then it appears that there is a definite relationship among four factors: the age, size, and genotype of the embryo, and the concentration of hormone. Scutellar callus was produced at all three concentrations of 2,4-D. In each case, the genotype was the "genetic stock" (see earlier report), the age was 12 days, and the size was 2-3 mm. All other samples from this line were either older and/or larger and produced only embryonic callus.

Table 1. Ability of immature embryos to form callus.

Genotype	Embryo age	Embryo size[1]	Number isolated	Embryonic callus[2]	Scutellar callus[3]
(Light intensity 15,000 lux; 2.0 mg/l 2,4-D)					
A188/"ABPHYLL"	18	6-7	20	20	0
A188/genetic stock	18	6-7	30	30	0
A188/genetic stock	16	5-6	20	20	0
genetic stock/"ABPHYLL"	16	4-5	10	10	0
A188	12	2-3	10	4	0
A188/genetic stock	12	2-3	20	8	0
genetic stock/"ABPHYLL"	12	3	1	0	0
genetic stock	12	1-3	5	2	0
(Light intensity 7,500 lux; 2.0 mg/l 2,4-D)					
genetic stock	18	5-6	20	20	0
A188	18	5-6	10	10	0
A188/"ABPHYLL"	18	5-6	20	20	0
genetic stock/"ABPHYLL"	18	5-6	10	10	0
"ABPHYLL"	18	3-4	5	5	0
(Light intensity 3,000-4,000 lux; 2.0 mg/l 2,4-D)					
A188	16	5	20	20	0
genetic stock	14	3-4	20	18	0
genetic stock	12	2-3	26	12	5
A188	12	1.5-2.5	20	4	0
(Light intensity 3,000-4,000 lux; 1.0 mg/l 2,4-D)					
A188	16	5	20	20	0
genetic stock	14	3-4	22	20	0
genetic stock	12	2-3	34	12	14
A188	12	1.4-2.5	4	2	0
(Light intensity 3,000-4,000 lux; 0.5 mg/l 2,4-D)					
A188	16	5	20	20	0
genetic stock	14	3-4	20	17	0
genetic stock	12	2-3	36	12	14
A188	12	1.5-2.5	4	1	0
(Light intensity 1,400-3,400; 2.0 mg/l 2,4-D)					
"ABPHYLL"	18	3-4	5	5	0
A188	16	4	2	2	0
(Light intensity 150 lux; 2.0 mg/l 2,4-D)					
A188/genetic stock	18	5-6	5	5	0
"ABPHYLL"	18	3-4	5	5	0
A188	16	4	2	2	0

[1]Length in millimeters.

[2]Scutellum flipped over due to embryonic growth, callus formed from the developing embryonic structures.

[3]Scutellum did not flip over, embryonic growth aborted, callus formed from the dome of the scutellum.

The effect of hormone concentration is even more evident after Tables 2 and 3 are examined. In Table 2, the percentages are calculated from the total number of embryos isolated while in Table 3 they are calculated only from the total number of embryos producing callus. Apparently, scutellar callus production is favored by lower hormone concentrations. As the concentration increases, the growth response is somehow shifted in favor of embryonic callus production.

Table 2. Influence of hormone concentration on the type of callus produced. Callus derived from the genetic stock and cultured under 3,000-4,000 lux. Hormone concentration expressed as mg/l.

Hormone concentration	Number isolated	Embryonic callus	Scutellar callus	% embryonic	% scutellar
0.5	36	12	14	33.3	38.9
1.0	34	12	14	35.3	41.2
2.0	26	12	5	46.2	19.2

Table 3. Influence of hormone concentration on the type of callus produced. Callus derived from the genetic stock and cultured under 3,000-4,000 lux. Hormone concentration expressed as mg/l.

Hormone concentration	Callus produced	Embryonic callus	Scutellar callus	% embryonic	% scutellar
0.5	26	12	14	46.2	53.8
1.0	26	12	14	46.2	53.8
2.0	17	12	5	70.6	29.4

Only embryonic callus has been initiated from the other genotypes tested. Clearly, the indication is that for every genotype there is a different combination of size, age, and hormone concentration necessary for the development of scutellar callus.

Unfortunately, attempts to regenerate plants from either type of callus have been in vain. Experiments are still being continued in this area.

William S. Rafaill

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Observations of maize from China

During the last two years 42 collections of maize from the People's Republic of China were obtained. Among them 14 collections were provided by the American Plant Studies Delegation to China. Last summer all of these collections were planted in the nursery here for cytological and genetical examination. Two weeks after seedling emergence, a study was made on the seedling characters. Three collections, 76-Honan-3, 76-Honan-7 and 76-Honan-8, showed a segregation of approximately three green seedlings to one virescent. The virescent seedlings appeared homozygous for virescent-1, because they gradually turned green after being six weeks old. When they were two months old, the plants were practically

normal in morphological characteristics, with green leaves and normal height. Pollen fertility and seedsets of these mutants were also about the same as the normal green sibs. From two to three virescent plants for each collection were crossed with a tester plant (v1/v1) for further studies.

In addition, 35 of the above 42 collections were successfully grown here for studies. Plants of the rest of the seven collections did not mature in time for experiments. For the well-grown plants three of each collection were employed for selfing and five for crossing with an inbred maize, Wilber's Flint. Many of the F_1 hybrids are now growing in the greenhouse for cytological examinations. The plants appear very healthy.

Y. C. Ting

Maize tissue culture

(a) Nodes of Hypocotyl. During last year 2479 sections of the nodes of hypocotyl of different diploid maize seedlings were cultured on the modified MS medium supplemented with a high content of sucrose. The cultures were equally divided into two groups, one was kept in the light (800 lux) with 12-hour daily illumination, and the other was kept continuously in the dark. The temperature was 72 F during the day and 60 F in the night. On the average, six weeks later, about 0.2% of the sections grew into callus. Two weeks later, the calli were transferred to new medium prepared by following the same formula as for the previous culture. These calli are now growing in the culture room. As soon as they have reached a certain size, they will be subjected to further experiments.

(b) Anthers. In the last year, about 3365 anthers having pollen at approximately mononucleate stage, of different strains of diploid maize were grown on modified MS medium with high sucrose content (15%) and 2,4-D (8 mg/l). About 50% of those anthers were kept in light having 800 lux intensity. The period of daily illumination was 12 hours and the average temperature was the same as for culturing the hypocotyl nodes. The other 50% were under continuous darkness. Six weeks after being cultured, about 0.5% of them had grown into callus varying in size; however, none of those calli regenerated into green plantlets, even though some of them were transferred to new media in which NAA was substituted for 2,4-D.

For continued maize anther culture, we have obtained two new strains of maize from the People's Republic of China. Those two maize strains were employed and selected there for anther culture. Both of them successfully grew *in vitro* into callus and later the calli regenerated into pollen plants.

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An organ-specific reciprocal effect demonstrated by Adh1 alleles

An Adh1-S/Adh1-F individual produces three alcohol dehydrogenase (ADH) Set I allozymes: the S·S and F·F homodimers and S·F heterodimers (Figure 1). In starch gels the relative intensities of these three allozyme bands are the reflections of (1) differential expression of the Adh1-S and Adh1-F alleles (D. Schwartz, Genetics 67:411, 1971), and (2) specific activities of the dimers themselves (e.g. Schwartz, TAG 43:117, 1973). A line expressing low levels of an Adh1-S isoallele relative to a standard Adh1-S allele has been described by Y. Efron (Science 170: 751, 1970).

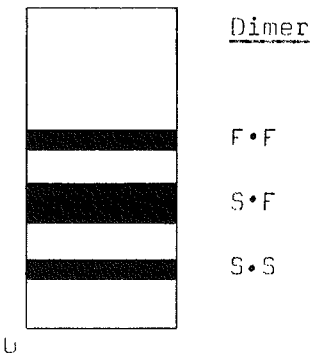

Figure 1. Starch gel of the Set I ADH allozymes of an Adh1-S/Adh1-F individual.

Various heterozygotes were constructed in hopes of detecting ADH regulatory variants on the basis of atypical allozyme ratios. Maize inbreds, exotics and standard Adh1-F/Adh1-S inbreds from lines originated by Schwartz and by Efron were used in these crosses. Two ADH allozyme patterns, one from dry scutellar slices and the other from the induced 5 cm root, were quantified for each heterozygous individual using a Transidyne General TG 2970 integrating densitometer. All the controls are to be published elsewhere. The percent contributions of each homodimer to the total Set I band intensities are reported in Table 1 as mean percent ($\bar{X}$) of each homodimer ± 95% confidence interval, as calculated from arcsine

TABLE 1

Mean percent of ADH Set I Homodimers in Maize Organs of Adh1 heterozygotes

	Adh1 genotype	Scutellum				Induced Root			
		%S·S	%F·F	F·F / S·S	n	%S·S	%F·F	FF/SS	n
GROUP I	F/S	38.2 ± 1.3	21.3 ± 0.8	.557	36	13.0 ± 1.1	39.5 ± 1.3	3.05	24
	24F/S	36.4 ± 0.7	23.8 ± 0.8	.654	27	12.5 ± 0.9	46.0 ± 1.3	3.67	18
	29F/S	35.1 ± 1.3	21.0 ± 0.6	.600	15	12.3 ± 1.6	43.9 ± 1.6	3.58	12
	FunkF/S	36.7 ± 0.7	24.3 ± 0.6	.662	38	14.8 ± 0.9	42.5 ± 0.9	2.87	27
	F/30S	36.7 ± 0.9	24.3 ± 0.9	.662	18	11.6 ± 1.6	49.6 ± 2.2	4.28	12
	F/35S	38.0 ± 0.7	24.9 ± 1.0	.657	27	11.8 ± 0.7	46.6 ± 0.8	3.94	17
	F/32S	40.2 ± 0.8	17.6 ± 0.7	.439	35	8.9 ± 0.4	46.1 ± 1.4	5.20	16
GROUP II	9F/S	31.3 ± 1.0	28.6 ± 1.0	.915	17	15.7 ± 1.5	37.7 ± 2.3	2.41	12
	15F/S	27.0 ± 1.2	31.6 ± 1.4	1.17	15	17.6 ± 1.4	33.3 ± 1.9	1.89	14
	33F/S	29.5 ± 0.8	27.4 ± 0.7	.927	27	16.4 ± 1.3	34.1 ± 1.4	2.03	12
	45F/S	29.3 ± 1.0	31.1 ± 1.1	1.06	30	16.8 ± 0.7	38.0 ± 0.9	2.27	18
	F/54S	27.9 ± 1.2	30.5 ± 1.3	1.10	36	14.8 ± 0.9	35.6 ± 1.3	2.41	16
	FunkF/S1951a*	31.2 ± 1.1	29.6 ± 1.0	.949	15	19.8 ± 2.0	36.6 ± 2.2	1.85	7
GROUP III	33F/54S	19.5 ± 0.9	36.3 ± 0.7	1.86	32	22.5 ± 1.7	34.6 ± 1.5	1.54	15

*This is the mutant allele induced by ionizing radiation.

transformed data. The sources of the Adh1 alleles used in Table 1 are as follows: F and S from Schwartz's inbred F and inbred S lines; 54S from Efron's AdhR-L, Adh1-S line; 9F from Maiz Chapolote; 15F from R2; 24F from Mo. CoB; 29F from Tama Flint; 30S from Parker Flint; 32S from Strawberry Pop; 33F from Super Gold Pop; 35S from Tama Flint Knobless; 45F from R177 and Funk F from Funk G4343 hybrid.

Three distinct scutellar allozyme patterns were found among the Adh1 heterozygotes; the Group II individuals have equal amounts of each homodimer band, whereas the Group I and Group III exhibit unequal amounts of each homodimer band (Table 1). Concurrently, individuals which have the lowest relative amount of a homodimer band in their scutella exhibit the greatest amount of this homodimer in their anaerobically induced primary root (24 hr anaerobic; 5 cm root; see Freeling, MGG 127:215, 1973). The relative amount of the homodimers is expressed as the ratio of the F·F band intensity to S·S band intensity. There is a reciprocal correlation between scutellar F·F:S·S ratio and the induced root F·F:S·S ratio. This reciprocal relationship will be termed "reciprocal effect," and should not be confused with Schwartz's "gene competition" (Genetics 67:411, 1971) or Birchler's "inverse effect" (MGCNL 51:13, 1977). This organ-specific reciprocal behavior is linked tightly to the Adh1 structural gene and is not affected by diverse genetic backgrounds (unpublished results). Since the reciprocal effect segregates with the Adh1 structural gene it may be the property of the structural gene itself or maybe some cis-acting regulatory component of the Adh1 gene. Additionally, since I have measured allozyme balances, some trans effects such as those anticipated on the basis of Schwartz's gene competition hypothesis might occur.

One of the radiation-induced Adh1-S mutants being studied in this laboratory (see following note by Freeling) has an altered reciprocal effect site. Normally, an Adh1-FunkF/Adh1-S heterozygote exhibits a Group I type allozyme pattern. The Adh1-S1951a mutant of this Adh1-S allele, when crossed to Adh1-FunkF, produces heterozygous individuals which exhibit the Group II allozyme pattern in the scutellum and induced root. In short, a mutant can simultaneously lower Adh1 expression in the scutellum while raising expression in the induced root. Ethane methylsulfonate-induced Adh1 mutants have not shown similar behavior. Since Adh1-S1951a is clearly break-point associated (see following note) and since its product is similar to that specified by its Adh1-S progenitor, it seems likely that the reciprocal effect is the property of a site outside of the Adh1 structural gene. Moreover, the variation in ADH Set I allozyme patterns detected among the lines could well be the result of regulatory gene variation. The test of these hypotheses will require careful protein and peptide comparisons, proof of genetic separability, or both.

Perhaps the most important conclusion to be drawn from the data as a whole involves the genetic basis of organ specific differential gene expression. Because a mutant affects Adh1 expression in two cell-types reciprocally, the DNA sequences used to regulate Adh1 in both the scutellum and root must be close-together or identical, and functionally related.

James C. Woodman

Comments on mutant Adh1-S1951a

In the previous note, Woodman used one of our new Adh1 mutants to better understand the organ-specific "reciprocal effect." Over the last three years, we have used chemical selection procedures to recover Adh1-deficient mutants via resistant pollen grains shed from mutagenized plants. Accelerated heavy neon (400 MeV/amu Ne^{10+}) has been most useful. Our first 69 radiation-induced mutants have undergone preliminary characterization, and our methods have been detailed (M. Freeling and D. S. K. Cheng, 1978, Genet. Res., in press). Adh1-S1951a is one of the mutants generated by this approach; the "a" denotes a derivative of the

original aberrant. This mutant is stable and linked closely with an electrophoretic site in the Adh1-S structural gene. Its phenotype includes underproduction of ADH1-S subunits in the scutellum, and--reciprocally--overproduction of ADH1-S subunits in the anaerobic primary root. The mutant has no trans effects. The original F_1 seed, of the genotype Adh1-F/Adh1-S1951 (see Woodman's figure), was recognized as an underproducer in the scutellum, but this characteristic was not stable upon backcrossing to the Adh1-F parent. Electrophoretic analyses of scutellar slivers from backcrossed progeny (BC_1) found that most Adh1-S alleles appeared to be wild-type, one (of 75 transmissions) showed underproduction, several displayed overproduction, and some hyperploids for Adh1 were transmitted through the F_1 male gametophytes and were detected in a test-cross. We call these backcrossed individuals "derivatives." I have looked at meiotic chromosomes in some of these Adh1-F/Adh1-S1951 derivatives. Extra centric fragments are usually present, and chromosome one appears to be involved in a complex rearrangement which leads to some bridges at both anaphases. I did not analyze the single Adh1-1951a derivative (underproducer) and much further chromosomal work will be necessary if we decide it is worth it to sort out these complex aberrations. The original F_1 gave 7% abortion. The underproducer derivative, Adh1-S1951a, was backcrossed to the Adh1-F parent once again (BC_2). Now the Adh1-S1951a mutant behaved as a stable allele of Adh1 with a phenotype as described.

The "reciprocal effect" regulatory behavior of Adh1-S1951 would not be expected of a structural gene mutant. All of the about 50 Adh1 mutants characterized by Schwartz, including those which I have tested for intragenic recombinational success, can be explained as lesions confined to the structural gene. The product of Adh1-S1951a appears nonmutant in electrophoretic mobility and thermostability; a comparison at the peptide level is in progress. Chances seem high that heavy-ions induced a complex aberration which, in turn, generated a stable mutant/aberrant in the cis-acting, programmable component of Adh1.

The origin of Adh1-S1951a parallels that of maize transposable element systems in several obvious ways (e.g. B. McClintock, PNAS 36:344, 1950 for the origin of Ac-Ds).

Michael Freeling

NADP-dependent cinnamyl alcohol dehydrogenases (CDH)

We have identified cinnamyl alcohol dehydrogenase enzymes in crude extracts from 2-8 cm primary roots. The spectrophotometric assay monitors the pseudo-zero order accumulation of NADPH in a reaction mixture composed as follows: enzyme extract, 0.1 mM NADP, 0.01 M cinnamyl alcohol from a 1.0 M stock in 100% MeOH, and 0.01 M $Na \cdot PO_4$ at pH 7.3; the cinnamyl alcohol (Ø-C=C-C-OH) tends to come out of solution as an oil. Root extracts were subjected to electrophoresis in standard borate starch gels, cut and stained (D. Schwartz and T. Endo, Genetics 53:709, 1966). The stain was as the spectrophotometric reaction mixture except that cinnamyl alcohol was added last to 0.1 M and both p-nitrobluetetrazolium chloride and phenazine methosulfonate were present at 0.1 mM. We found several different banding patterns among the inbred lines which we tested (Std. S and Std. F from Schwartz). The Figure diagrams the electrophoretograms from three of these lines and some of the hybrids.

By using the appropriate electrophoretic markers for Adh1 and Adh2, we found that ADH subunits are not involved in CDH activity.

We have performed the appropriate backcrosses and self-pollinations of the Std. S/Std. F and Std. F/B14 hybrids. An entire banding pattern segregates as a unit, although the F_1's may not be the exact sum of their parental patterns. The majority of ears from these crosses segregated patterns consistent with Mendelian expectations: the Std. S pattern is allelic to the Std. F pattern, and the Std. F pattern is allelic to the B14 pattern. We call the responsible gene Cdh1.

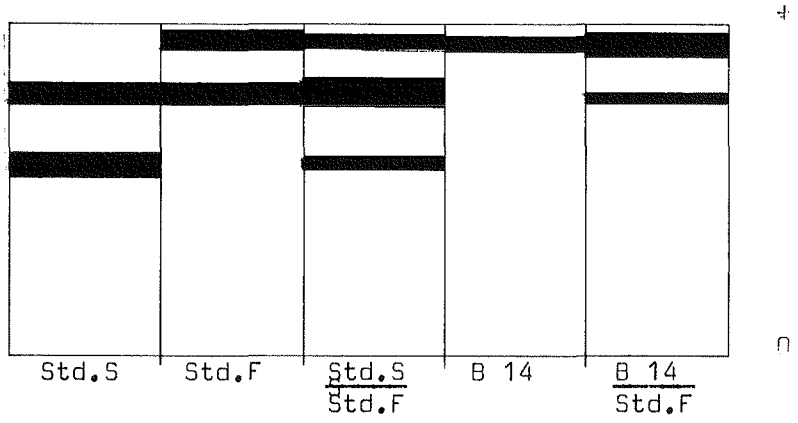

Figure. NADP-dependent CDH isozymes/allozymes according to genotype at the Cdh1 locus.

The three CDH's present in the Std. S/Std. F hybrid were excluded from a Sephadex G150 column, suggesting that their MW are above 150,000. However, a Biogel P300 column separated all three CDH bands; the fastest electrophoretically was the largest at or near the 400,000 MW exclusion limit, with the slowest electrophoretically being considerably smaller; the intermediate isozyme in electrophoretic mobility is also intermediate in size. Such large sizes suggested to us that maize CDH is multimeric. The allelic differences which we have found among maize inbreds may reflect degrees of polymerization, etc. Such problems lie outside of this laboratory's current interest; we will supply data and lines on request.

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Selection of maize callus resistant to Helminthosporium maydis T-toxin

Work is under way in three areas to determine the possibilities for producing novel resistance to H. maydis using tissue culture techniques.

1) Expression of T-toxin sensitivity in callus tissue: Our experiments confirm the results of Gengenbach and Green (Crop Sci. 15:645, 1975) and others that callus tissue derived from plants with T cytoplasm (T-callus) is markedly inhibited or even killed by the presence of quite small amounts of T-toxin in the nutrient medium. N-callus grows normally under these conditions. Malate oxidation as measured by DCPIP reduction (Peterson, Flavell and Barratt, Plant Disease Reporter 58:777, 1974) has been examined in mitochondrial preparations from both N and T callus. Despite difficulties in extracting a high mitochondrial malate dehydrogenase activity from callus, we find that oxidation is inhibited in a T-callus preparation and not in an N-callus preparation. That a differential 'cytoplasmic' sensitivity to T-toxin is expressed in callus means that it should be possible to use undifferentiated maize tissue (or a single-cell system) to select for genetic changes, whether nuclear or cytoplasmic, which affect this expression.

2) Selection of T-toxin resistance in T-callus: We are selecting T cytoplasm callus lines resistant to T-toxin without mutagenic treatment. Callus was initially obtained according to the method of Green and Phillips (Crop Sci. 15:417, 1975) in the presence of 2 mg/l 2,4-D. After 3 subcultures on medium containing 2 ml/l of a purified T-toxin extract, some of the T-calluses are showing a remarkable resistance to T-toxin, growing almost as well as N-callus. T-callus not previously exposed to T-toxin normally will not grow at all at this sublethal toxin concentration. These results substantially confirm the findings of Gengenbach and Green (1975, op. cit.). Experiments are under way to determine whether this toxin resistance reflects a permanent genetic change or is the result of a physiological adaptation by the callus to the presence of toxin in the medium.

We have obtained a similar pattern of resistance development in tobacco cultures by the selection of callus on medium containing sublethal levels of cycloheximide or 1-amino-2-nitrocyclopentane carboxylic acid (ANCPA). Further investigations have suggested that the high degree of resistance to these toxins developed in culture is not due to permanent genetic changes.

3) Development of a haploid T-callus cell line: We are working in this area to test the feasibility of selecting for nuclear genetic changes affecting the expression of T-toxin sensitivity in T-callus. Two possible approaches have been considered:

Anther culture. Success in generating haploids of cytoplasmically male sterile material by means of anther culture has been reported for wheat (E. Picard and J. de Buyser, CR Acad. Sci. Ser. D 277:1463, 1973) and tobacco (Tsikov et al., Dokl. Bolg. Akad. Nauk 27:1727, 1974). Systems for maize anther culture are not as well defined, although the Chinese workers are beginning to have some real success (Acta Botanica Sinica 19:89, 1977). We have plated out some 35,000 maize anthers from various maize hybrids and inbreds (including T cms material) on several types of medium. So far we have produced callus only from anthers of maize hybrid MV 201, plated on a modified MS medium containing 12-18% sucrose, 2.5 mg/l 2,4-D, 1.0 mg/l 6-BAP and 0.5 mg/l 1-NAA. One of the calluses obtained in this way has continued to proliferate away from the anther on a modified Green and Phillips medium for three months. The callus is rather reminiscent of callus obtained from small immature embryos and is producing shoots and roots. Experiments are in progress to confirm the pollen origin of this callus.

Spontaneous androgenesis/parthenogenesis. Means are not at our disposal to make use of the ig mutation or 'stock 6' to generate immature haploid embryos for culture. We are, however, trying to initiate callus from some mature haploid seed kindly supplied by Prof. Peter Peterson. Callus has been initiated from mature seed in this laboratory and has been maintained for over a year in culture, although it is generally slow growing and has so far differentiated to produce roots only.

With regard to the initiation of maize callus from immature embryos, our experience stresses the need to use embryos less than 2 mm long to generate callus successfully according to the method of Green and Phillips (Crop Sci. 15:417, 1975). Larger embryos tend to give results similar to those reported by Rafaill (MNL 50:84). We also find "the number of days post-pollination" as a misleading parameter for gauging embryo development. Owing to the vagaries of the British climate, our American seed took 30-35 days from pollination to produce embryos 1.0 mm long!

Dick Brettell and David Ingram

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Maize x Sorghum

To date all reported attempts to hybridize maize and sorghum have failed (E. E. Gerrish, 1967; J. J. Mock and W. H. Loescher, 1973). At CIMMYT 12,844 sorghum pollinations have been made onto shortened silks of dehusked, isolated, detasseled maize plants. Because of seed breakdown immature embryos were excised and cultured on artificial nutrient medium. Seven hybrids have been retrieved, five from diploid (2n = 20) and two from an artificially induced stable tetraploid (2n = 40) maize, pollinated with diploid (2n = 20) sorghum. Hybrids were identified mainly on their cytology at mitosis. All hybrids possessed 20 maize chromosomes and from 10 to 2 of sorghum, whether the maize parent was diploid or tetraploid. To produce viable hybrids therefore it may be necessary that 20 maize chromosomes are present so that only fertilizations of unreduced gametes from diploid maize survive.

Although sorghum chromosomes are much smaller than those of maize in their own cytoplasm, this difference was obvious in only very few hybrid cells, and was minimal in the majority. It appears that maize and sorghum chromosomes condense differentially so that the period during which the size difference may be observed is extremely short. The presence of knobs on maize chromosomes and their absence on sorghum also helped to distinguish the two genomes.

Chromosome elimination occurred in all hybrids. Most cells observed from seedlings contained more chromosomes than from older plants, and at flowering very few cells contained more than 20.

F_1 plants had a slow erratic growth and were morphologically more maize-like than sorghum. All produced a reduced tassel. In one no tassel was produced, five gave unbranched linear spikes with tassel seed at the base, and the seventh produced a tassel with two very short lateral branches. No hybrid gave viable pollen. All produced at least one ear but successful pollinations were made on only two. Thirty-three BC_1 plants have been obtained with maize and will be described in a later paper.

Three major phenomena important to the production of hybrids have been identified:

1) Seed stimulation was very low, and on average occurred in less than one percent of the pollinations made. This seemed to depend on the genotype and condition of the plant as well as environmental conditions.

2) Visible large scale endosperm breakdown occurred from seven to seventeen days after pollination, depending on the environment and the condition of the plant. In only very few seeds the endosperm appeared to develop normally after this critical stage, but these seeds were always smaller than control comparisons at all stages.

3) Embryo breakdown may occur at any stage throughout seed development. No living hybrid embryos have been observed at seed maturity. Embryo development proceeded at different rates in individual seeds, and was not necessarily related to the stage expected in normal development. Type of development was also different, and hybrid embryos were often so abnormal in shape that they were not recognizable as embryos, until further development in culture. Most were not able to survive because of endosperm breakdown. However, in seeds with apparently normal endosperm, embryo breakdown was independent of endosperm development.

In order to examine the potential value of sorghum germplasm in maize improvement, it is necessary to produce more hybrids of wide genetic diversity. Because of the low degree of seed stimulation large numbers of crosses must be made to give a sufficient number of hybrid embryos. Also, because of the abnormal development and complete breakdown of most hybrid embryos it is essential that embryo

culture is used routinely for hybrid production. The results obtained at CIMMYT have encouraged us to expand our program, and we hope others will be encouraged similarly. The techniques used and the results obtained will be published in more detail shortly.

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A genetic test demonstrating that β-glucosidase activity is not involved in the mechanism of hypersensitive resistance to maize rust

Many fungitoxic compounds in plants occur *in vivo* as inactive glycosides. The active aglycone appears only as a result of pathological or mechanical damage presumably due to the action of a host specified glycosidase activity. For the β-glucosidase specified by the gene *glu* (Pryor, MNL 50:15, 1976) this supposed role can be tested by determining if plant resistance is still expressed in an individual homozygous for the Null allele *glu-N*, and thus lacking β-glucoside activity. Lines carrying different genes for resistance to the rust *Puccinia sorghi* were derived from the International Monogenic lines developed by Hooker. The test individuals were produced among the progeny of the following back-cross:

$$\frac{Rp\ \ glu\text{-}A}{rp\ \ glu\text{-}N} \times \frac{rp\ \ glu\text{-}N}{rp\ \ glu\text{-}N}$$

and the results are presented in Table 1. Although the data are small the

Table 1. Backcross progeny from crosses involving genes for glucosidase (*glu*) and rust resistance (*Rp*).

Gene	Backcross	*Rp glu-A*	*Rp glu-N*	*rp glu-A*	*rp glu-N*	Σ	$^{a}\chi^2_{(1)}$	$^{b}\chi^2_{(1)}$	$^{c}\chi^2_{(3)}$
Rp-d(2)	G3 x 77.G3	17	4	5	5	31	3.9	13.0*	14.8*
Rp-f	70.G3 x G3	48	23	21	44	136	0.26	0.03	17.2*
Rp-g	G3 x 78.G3	10	9	12	10	41	0.22	0.22	0.46
Rp5	87.G3 x G3	47	25	15	46	133	0.91	0.61	22.6*
	Total	122	61	53	105	341	1.83	0.23	39.5*
Rp4-a	G3 x 85.G3	6	12	8	14	40	0.4	3.6	4.0
	85.G3 x G3	17	15	6	11	49	4.59*	0.18	5.77

Rp and *Rp5* are closely linked at or near position 0 on linkage group 10. *Rp4* is in linkage group 4.

Rust resistance lines - 70, 77, 78, 85 and 87 all carry the *glu-A* allele while the susceptible G3 is homozygous for *glu-N*.

a - test of significance for the segregation of *Rp/rp*.

b - test of significance for the segregation of *glu-A/glu-N*

c - test of significance for the independent segregation of *Rp* and *glu*

* - significant at the 5% level.

conclusion is clear. The expression of resistance was unaffected by the absence of glucosidase activity. The degree of resistance in the two resistance classes (Table 1) was indistinguishable. This conclusion applied to 3 alleles at the Rp locus and to the Rp4 and Rp5 loci.

The genetic data in two crosses deviate significantly from the expected 1:1 segregation of alleles at the Rp and glu loci. However the number of progeny is small.

On the other hand the segregation patterns of the resistance loci on chromosome 10 with the glu locus deviate very markedly in three of the four crosses, suggesting that Rp and glu may be linked. A pooled estimate (Table 1) from the relevant crosses indicates about 33% recombination between Rp and glu.

A. J. Pryor

Mapping of glucosidase on chromosome 10

Data from the crosses with rust resistance factors suggest that glu is located on chromosome 10 about 33 recombination units from Rp. The chromosomal location was confirmed using the waxy marked translocation stocks:

$$\text{Cross:} \quad \frac{\text{glu-A wx}}{\text{glu-C Wx}} \times \frac{\text{glu-A wx}}{\text{glu-A wx}}$$

Translocation	A wx	A Wx	C wx	C Wx
wxT9-10b	23	0	2	25
wxT1-9c	14	11	11	14
wxT6-9(4778)	13	10	12	15

Linkage of wx with glu was observed only with the wxT9-10b stock. Cx, the locus specifying catechol oxidase isozymes, is located at about position 33 on chromosome 10 and thus should show tight linkage to glu. Preliminary results indicate 5.8% recombination between these loci:

$$\text{Cross:} \quad \frac{\text{glu-C Cx-N}}{\text{glu-A Cx-F}} \times \frac{\text{glu-C Cx-N}}{\text{glu-C Cx-N}}$$

A/C F/N	A/C N/N	C/C F/N	C/C N/N
88	8	1	59

This is almost certainly an overestimate because the data presented involved segregation of the Cx-N allele and absence of the catechol oxidase isozyme would be misclassified as a recombinant. Further crosses using the Cx-S (slow isozyme) allele and dull endosperm as well as glu will help order these loci in the region 33 of chromosome 10.

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Aluminum tolerance in maize seedlings as measured by primary root length in nutrient solutions

We have been using nutrient solutions to study the genetics and mechanism of aluminum tolerance in maize. The screening technique has already been described (Agr. J. 69:755, 1977). A large number of inbreds have been classified by measuring relative primary root length (RRL) after two weeks growth.

The inbreds fall into four approximately equal classes when screened at 2.0, 1.0 and 0.5 mM Ca. Class I inbreds are the most tolerant, growing even at 0.5 mM Ca. Class IV inbreds are severely inhibited in the presence of Al, even when the Ca concentration is raised to 2.0 mM.

Tolerance was dominant in all crosses studied. Table I summarizes the F_2 and BC_1 data from crosses between inbred class levels. In every case, the progeny formed not a continuum but rather two discreet classes. A one-gene model was

Table I. Segregating ratios, chi squares, and probabilities for various F_2 and BC populations. The most Al tolerant inbreds are designated by (I), and the most sensitive by (IV). X^2 values are for a one-gene model, with tolerance dominant.

		F_2			BC		
P_1	P_2	Ratio	X^2	P	Ratio	X^2	P
W182BN (II)	A554 (IV)	159:54	.014	>.90	57:42	2.273	>.10
W182BN (II)	C0192 (III)	134:53	1.114	>.25	53:43	1.042	>.25
W64A (I)	B37 (III)	68:27	.593	>.25	49:61	1.309	>.25
Val7(I)	W182BN (II)	70:28	.667	>.25	-	-	-
W182BN (II)	C0113 (IV)	131:45	.031	>.75	18:17	.029	>.75
W64A (I)	B14 (III)	104:31	.299	>.75	42:52	1.064	>.25

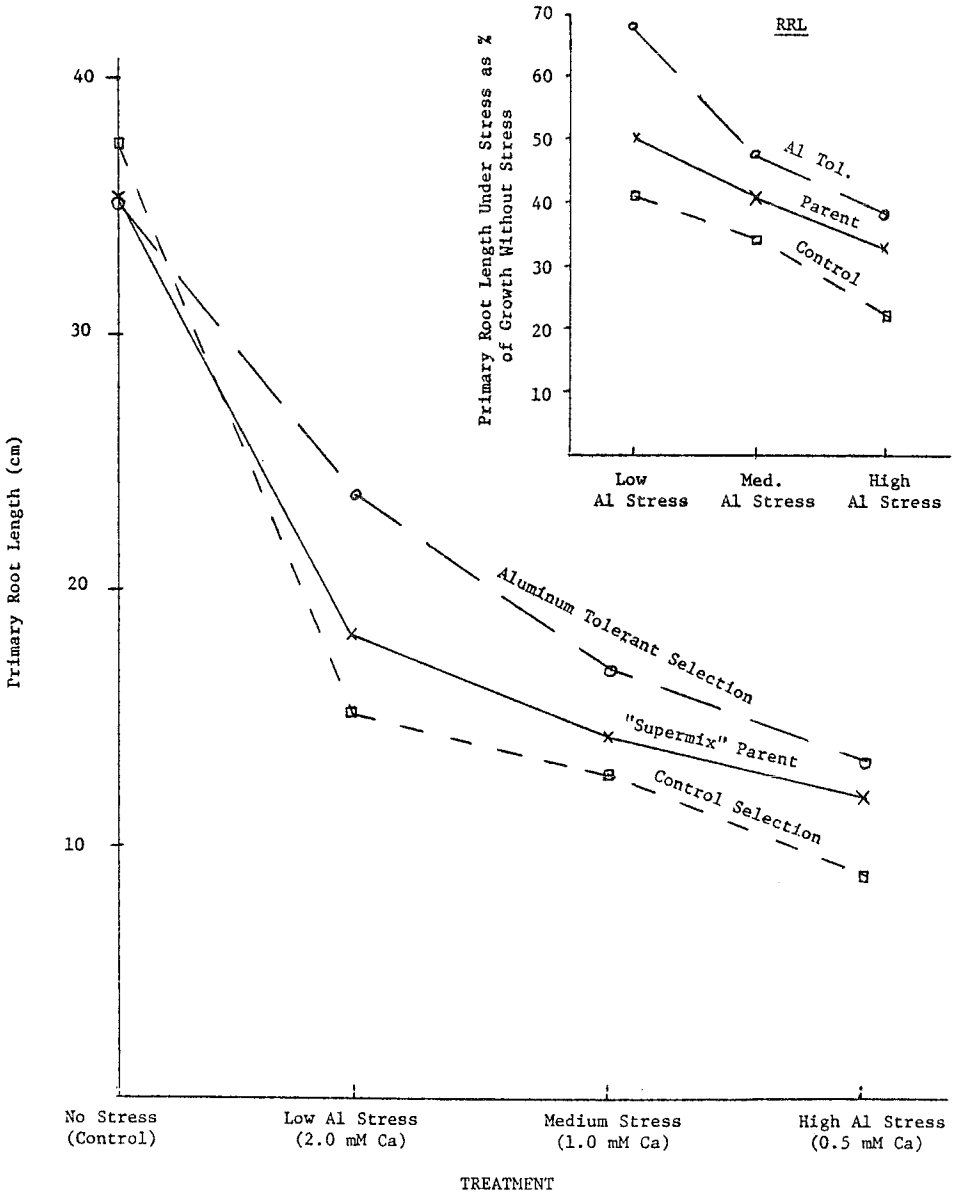
tested with the chi square statistic and found to be consistent with the data. We postulate that we are dealing with a multiallelic series at a single locus determining the root's ability to continue growth at a given Al stress level.

To study the mechanism of aluminum tolerance it is preferable to compare tolerant and sensitive plants which are otherwise genetically identical. Previous work in this area has compared cultivars of different genetic backgrounds. We have selected for aluminum tolerance within a temperate composite, "Supermix," as an alternative to developing isogenic lines. Selections were transplanted to the field and randomly intercrossed. Equal numbers of seeds from each ear were bulked together to minimize genetic drift.

Another population was similarly selected from "Supermix" on the basis of vigorous root growth under non-stress conditions. This was done to distinguish selection for aluminum tolerance from "natural" selection for adaptation to nutrient media.

Graph 1 shows that selection for aluminum tolerance, as measured by primary root length, has been effective. The aluminum tolerant selection shows no improvement over the parent population under non-stress conditions but is superior at every

GRAPH I



stress level. Only at 2.0 mM Ca is the difference significant (Duncan's Multiple Range). However, if the values at all the stress levels are pooled and analyzed together, the difference is highly significant.

The control selection is not significantly improved in primary root length under non-stress conditions. Its average performance at the three stress levels is poorer (α = .01) than the original parent.

We feel that these populations will be useful tools in studying the mechanism of stress tolerance, especially after another cycle of selection. Differences between tolerant and sensitive inbreds can be re-examined with materials of closely similar background genotype. The interactions between aluminum, calcium and phosphorus response are of particular interest since aluminum tolerance has been variously described as calcium or phosphorus efficiency. The role of pH control as an avoidance mechanism will also be investigated.

E. William Stockmeyer, Herbert L. Everett and Dean Rhue

Multiple male single cross

We have made up seed of appropriate crosses to test the advantages (if any) and disadvantages of the use of so-called multiple male ("multi-male") single cross and three-way hybrids. The combination of high yield and broader adaptability is sought in this approach to hybridization.

The following diagrammatic outline will clarify the approach taken:

♀ parents	♂ parents
Inbred A in normal and cytosterile (cms) versions.	Mix of six inbreds, B through G, which combine favorably with inbred A. These inbreds must have nearly identical maturity, but their hybrids may vary for cytosterile restoration and general adaptability (e.g. disease and pest resistance - environmental stress response).

Specific example of multi-male single cross hybrid:

♀ parents	♂ parents
W182 BN	C0150 (Rf)
W182 BN PR-cms	Ny16 (Rf)
	C0192 (Rf)
	C153 (Partial Rf)
	AyX157(rf)
	IoB8 (rf)

A multi-male 3-way hybrid has been made up for evaluation in similar fashion.

E. William Stockmeyer, Herbert L. Everett and Dean Rhue

Cytology of pollen abortion in cytoplasmic male sterile corn anthers

Recently, Warmke and Lee (J. Hered. 68:213-222) have reported that early mitochondrial degeneration occurs in tapetal cells of cms-T corn anthers shortly after meiosis. The degeneration of mitochondria is similar to that observed ultrastructurally in cms-T corn lines treated with H. maydis, race T toxin (H. C. Aldrich et al., Tissue and Cell 9:167-177). A study of pollen abortion in cms-C and cms-S cytoplasms of maize was initiated to determine whether similar mitochondrial degeneration occurs in these sources of cytoplasmic male sterility. Examinations of anthers in various stages of development from cms-T, cms-C, and cms-S versions of inbred W182BN, W64A, and NY821LERf were made at light and electron microscopic levels.

Anther development of cms-C and non-male sterile versions of inbred W182BN was similar through meiosis. Differentiation of male sterile from normal anthers was first seen in tapetal cells at the tetrad stage of development. Two types of tapetal differentiation occurred in cms-C. One type of tapetal variant had a dense cytoplasm, was binucleate, and had an increased number of small vacuoles when compared to male fertile anthers. In this type, tapetal inner and radial walls broke down as in fertile types but the deposition of Ubisch bodies was irregular and quantitatively less than in fertile anthers. The tapetum and the microspores disintegrated at the intermediate microspore stage.

The other type of tapetal variant exhibits a less dense cytoplasm and the presence of large vacuoles at the early tetrad stage. The tapetal cells increased in radial thickness by 2-2.5 times as compared to the meiosis stage. The cells usually contained only one nucleus and they developed rather thick cell walls which stained red with safranin. In this type of anther, microspores began to disintegrate a little earlier than in the other type of cms-C anther and the tapetal walls remained intact and thick when the tetrads aborted. The tapetal cells extended radially to fill most of the locule and the aborted tetrads were compressed into the center.

The tetrads of both types of tapetal variants generally appeared normal in their early development. Microspores of both types initiated exine development. However, none of the cms-C microspores was seen to form a single large vacuole and undergo mitosis as the fertile microspores did. Microspore development appeared to stop at an intermediate stage when the cytoplasm began to disintegrate.

At an electron microscopic level, differences in cytoplasmic density and cell wall thickness were observed between the two tapetal types, but changes in mitochondrial structure were not observed. The cytoplasm of the dense type appeared relatively normal except for the degree of vacuolation. Vacuoles tended to develop at the tetrad stage and enlarge during the early microspore stage. The cytoplasm became less dense and contained vacuoles, free ribosomes, and dilated endoplasmic reticulum just before degeneration at an intermediate microspore stage. The other type of tapetum was even more vacuolate, containing large vacuoles at even the early tetrad stage. The cytoplasm appeared to be less dense and endoplasmic reticulum occurred in a single elongate form or in aggregates. Multivesicular bodies and aggregates of ER occurred frequently near the radial cell wall.

The importance of the two patterns of tapetal breakdown is not known. Usually individual anthers exhibited one or the other type of tapetal breakdown but rarely a mixture of the two types was seen in different locules of the same anther. It is not known whether the two types of tapetal response represent two different mechanisms of pollen abortion. Although the latter would seem to be more compatible with current thoughts on cms-C sterility and restoration, more data on the nature of the cms-C type sterility are needed.

Preliminary studies on the cms-S type of cytoplasm revealed a completely different pattern of pollen abortion as might be expected since the cms-S cytoplasms follow a gametophytic pattern of pollen fertility restoration. The microsporogenesis of the cms-S system was identical to normal in all aspects of tapetal and microspore development. Deviation occurred late at the stage when pollen was nearly mature after the tapetum had disappeared. Pollen organelles appeared to be normal in structure during microsporogenesis. At a relatively late stage in pollen development, abortion occurred rapidly; however, distinct changes in organelles including mitochondria were not observed. Thus, changes in mitochondrial structure observed in cms-T pollen abortion and after treatment with H. maydis, T toxin were not seen in either cms-C or cms-S pollen abortions.

Sheu-Ling Janet Lee, Vernon Gracen and E. D. Earle

FUNK SEEDS INTERNATIONAL
Bloomington, Illinois

Treatment procedures in an attempt to induce cytoplasmic male sterility with ethidium bromide

Previous attempts by the author to induce cytoplasmic male sterility in maize have not been successful (MNL 51:5-6, 1977). During this past season, two treatment procedures and a single relatively high concentration of ethidium

bromide were used. Inbred W182BN was used in this current work and in previous MNL articles (51:5-6,1977; 50:28-29, 1976), rather than inbred W59M. Four-hundred kernels of the inbred W182BN were germinated for 30 hours on Kimpak at 27° C; at the end of this time, some radicles had emerged. These kernels were then completely submerged in a 0.05 M ethidium bromide solution for 24 hours. In the second treatment, 400 dry kernels were completely submerged in a 0.05 M ethidium bromide solution for 48 hours. The treated material was planted in trenches in the field, watered and then covered. The M_1 plants were self-pollinated.

The treatment using germinated kernels produced only 11 plants and 8 ears were harvested. In the second treatment, using dry kernels that were soaked in ethidium bromide for 48 hours, 100 plants were produced and 47 ears were harvested. In 1978, it is planned to plant the material ear-to-row and examine M_2 plants for male sterility.

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Cytoplasmic glossy - an unusual interaction system

A number of unknown glossies were tested for allelism in 1975. Among these were three which gave unexpected results. When used as female each gave glossy seedlings in F_1 when crossed with gl, gl2, gl3 and gl4. The three stocks have no ancestor in common for at least the last 10 generations. In subsequent tests, the three stocks, gl*-a, gl*-b and gl*-c, when used as female, gave glossy seedlings in F_1 with all standard glossies used. F_2 and F_3 seedlings are also glossy. When the three glossies are used as males in crosses with standard glossies, the F_1 seedlings are + and the F_2 segregations approximate 9 + : 7 gl.

The cytoplasmic element (cgl) when separated from the nuclear glossy appears to have no phenotypic effect and does not give glossy seedlings in F_1 with standard glossies. The nuclear gene when separated from cgl appears to behave like other simply inherited glossies. The unusual results therefore appear to be due to a cgl gl*-a (or b, c) interaction. Preliminary data suggest that the cgl gl*-a interaction leads to a "suppression-like" phenomenon. Evidence for this comes from three sources:

1. The absence of + type progeny in F_2 and F_3 crosses of cgl gl*-a/gl.
2. Crosses of gl2♀ x cgl gl*-a/gl2 produce no glossy progeny.
3. Crosses of +♀ x cgl gl*-a/gl2 give monohybrid +:glossy segregations in F_2.

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Maize monosomic mutational mapping

In maize, there is a non-random distribution of known mutants in the genome. Chromosome 8, for example, has only six known genes whereas the least number known on any other chromosome is 20. Furthermore, all known mutants in chromosome 8 are in the distal quarter of the long arm. Rhoades (Amer. Naturalist 85:105-110, 1951) suggested that the non-random distribution of known mutants in maize might be an indication of redundant chromatin in segments where relatively few genes are known.

I have begun a long-term study using monosomics to determine if there really are fewer genetic loci capable of being mutated on chromosome 8 than on other chromosomes. The procedure is outlined below.

R/r-X1 plants are crossed as female parents by pollen parents carrying a single recessive seedling mutation. The pollen of the male parent is treated with a mutagen such as ethylmethansulfonate (EMS) using standard procedures. All progeny expressing the seedling mutation are monosomic for the chromosome bearing the marker mutant. One simply grows plants expressing the mutant phenotype and examines these plants for the presence of a second mutant phenotype. The second mutant phenotype could be attributable to a dominant mutation induced anywhere in the genome (these are rare in maize) or a recessive mutation on the monosomic chromosome. Any mutation affecting the sporophyte morphology would be detected in this way. It is also possible that the mutagen would induce a new mutation which has a phenotype similar to the one used to identify the monosomic. If the F_1 plant was monosomic for the chromosome carrying this new mutation, it can be readily determined by testcrossing to plants recessive for the mutant in the original male parent.

Each plant expressing the marker mutant will also be selfed to determine if any mutation affecting kernel morphology was induced by the mutagen. If all kernels produced by the monosomic were mutant for a new phenotype, it would indicate that a mutation was induced on the monosomic chromosome that affects kernel morphology. Furthermore, these progeny would be homozygous recessive both for the marker mutant gene locus and the newly induced mutant gene locus.

Each monosomic carrying a new mutation will also be crossed by a standard inbred line. All F_1 progeny will be heterozygous both for the marker mutation used to identify the monosomic and the new mutation, and the genes would be in coupling. These F_1 progeny could then be crossed by plants produced by the selfed monosomic (which are homozygous both for the marker mutant and the newly induced mutant). This will greatly simplify mapping of the new mutants.

I am mutagenizing pollen from plants carrying recessive marker mutations on chromosomes 7, 8, or 10. I will determine the frequency of mutations on chromosomes 7, 8, and 10. I will determine if the frequency of mutants induced on chromosome 8 is similar to the frequency of mutation induction on the other chromosomes tested. Also, I will determine if most of chromosome 8 is actually refractory to mutation induction or if the lack of known mutants on chromosome 8 is a fortuitous mapping artifact. It is hoped that through this program it will finally be possible to map chromosome 8 in maize. Supported by ERDA Contract No. EY-76-S-OL-2121.

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Further observations on the nature of the B chromosome-Knob interaction resulting in chromatin loss

The Df 33166 chromosome was derived by breakage of a normal chromosome 9 with a large terminal knob on 9S at the second microspore division. Deficient for the Yg2 and Wd loci, its short arm was capped by a small heterochromatic knob of unknown origin. The size of the knob approximated that of the 9S knob present in inbred KYS. Previously reported data demonstrated that the high-loss phenomenon was restricted to knobbed chromosomes in microspores with two or more B chromosomes and that there was a positive correlation between knob size and rate of loss. Since the knob on 33166 was small, it was anticipated that its rate of chromatin loss would be much less than that of K-9. However, data reported by Rhoades and Dempsey in the 1977 Maize News Letter show that: (1) the 33166 chromosome underwent a much

higher rate of loss than did the K^{L}9 chromosome in microspores of heterozygous plants, (2) that breaks in the postulated dicentric bridge arising by delayed replication of the small knob of 33166 were predominantly distal to the C locus while the great majority of breaks in the dicentric coming from the K^{L}9 chromosome were proximal to Wx, and (3) that the 33166 chromosome underwent chromatin loss in microspores with 1B chromosome while two or more B's were needed to induce loss in the K^{L}9 chromosome. A summary of the data bearing on the frequency of loss of the C locus with varying numbers of B chromosomes is presented below, for crosses on c c female parents. Items 1, 2, and 3 are from previously reported data; items 2 and 3 are sibs, and items 4 and 5 are sibs.

Male parent	No. B's	C	C-c mosaics	c	% of breaks distal to C	% of breaks prox. to C
K^{L}9 C / K^{L}9 C	4 or more	5342	72	1937	3.6	96.4
" / 33166 C	3	4030	543	162	77.0	23.0
" / "	1	1565	63	3	95.4	4.6
					% C-c in C class	
K^{L}9 c / 33166 C	1	1239	83	1294	6.3	
" / "	0	1198	0	1123	0.0	

In line 2 of the above tabulation where the microspores had one or two B's the percentage of mosaic kernels amongst the C class was 543/4573 or 11.9%. All or nearly all of the mosaic kernels came from the bridge-breakage-fusion cycle involving the 33166 chromosome at the 2nd microspore mitosis. In the total of 1628 C kernels listed in line 3, approximately 30% or 488 came from a sperm cell produced by a microspore with one B chromosome (observations by Randolph show that only 30%, instead of the expected 50%, of the microspores from a 1B plant have a B chromosome, because of lagging of the univalent B during meiosis). Breaks distal to C occurred in 63 (12.9%) of the estimated 488 microspores with 1B chromosome. This percentage of chromosome breaks in 1B microspores is very close to the 11.9% C-c mosaicism coming from microspores with 1 and 2B's. In the 1977 Maize News Letter, we suggested that a mechanism other than our hypothesis of a delayed knob replication conceivably could be responsible for the inception of the bridge-breakage-fusion cycle. However, recent cytological observations by Luiz Saraiva (see his report in this News Letter) in this laboratory have demonstrated the validity of the delayed knob replication hypothesis. If we accept the correctness of this mechanism for induction of the bridge-breakage-fusion cycle producing mosaic endosperms, it is apparent that 1B is as effective in producing delayed replication of the small terminal piece of heterochromatin on the 33166 chromosome, which leads to dicentric formation followed by bridge breakage, as are 2B's--i.e., the threshold for inhibition of knob replication is reached by 1B chromosome and adding a second B has no effect. This is in contrast to the behavior of a structurally normal K^{L}9 chromosome where chromatin loss does not occur in 1B microspores and two or more B's are needed. However, it is true for both the K^{L}9 and 33166 chromosomes that once the threshold for delayed replication has been attained there is no enhancement in rate of dicentric formation (and loss) by increasing the number of B's.

The data in lines 4 and 5 demonstrate the stability of the 33166 chromosome in 0B microspores. Both the K^{L}9 and the 33166 chromosome are stable during the megaspore mitoses, irrespective of the number of B chromosomes.

The difference in the location of breakpoints in the dicentric bridges coming from the K^{L}9 and 33166 chromosomes may be ascribed to a weak union of the heterochromatic knob with the euchromatic tip of 9S in the origin of the 33166 chromosome. Under anaphase stress, breakage of the dicentric bridge would preferentially

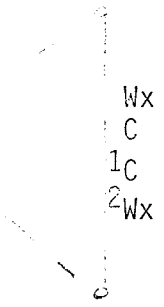
occur at this weak junction. If this be true, the great majority of deficient chromosomes recovered in the embryo should be identical in size.

As a consequence of the rupture of the dicentric bridge, formed by the 33166 chromosome, between the C locus and the knob at the 2nd microspore anaphase, one of the two sperm cells has a deficient chromosome 9 with a freshly broken end and the other has a 33166 chromosome. Following fertilization of the polar nuclei by the sperm with the broken 9, a typical bridge-breakage-fusion cycle is initiated in the first division of the primary endosperm nucleus. The mosaic pattern of colored and colorless aleurone involves all of the aleurone layer, indicating that the original break in the dicentric bridge consistently occurred at the second microspore mitosis.

This pattern was invariably found in the hundreds of mosaic kernels previously examined. However, a 33166 sub-line has arisen in which the mosaic pattern of C and c tissue suggests that the dicentric condition, caused by delayed replication of the terminal heterochromatic knob, may originate at either the second microspore mitosis or frequently in the first endosperm division. Bridge breakage at the first endosperm anaphase gives a cell with a 33166 chromosome carrying the C allele and a sister cell with a broken 9. That fraction of the aleurone layer descending from the cell with a broken 9 will be mosaic for C-c because of the ensuing bridge-breakage-fusion cycle when the break occurs between C and the knob. In such instances, approximately one-half of the kernel will have an aleurone layer uniformly self-colored (no mosaicism) while half will be mosaic for colored and colorless aleurone sectors. The size of the self-colored sector varies as expected from kernel to kernel but the observed pattern of mosaicism clearly indicates that breaks may be induced at the first endosperm mitosis.

Observations demonstrating dicentric formation at the first endosperm division came from the cross of a c wx tester by pollen from a 33166 C Wx/ N9 c wx plant. The constitution of the dicentric from the 33166 C Wx chromosome produced at either the second microspore or at the first endosperm division is diagrammatically shown below:

Meiotic crossing over between C and Wx gives a 33166 C wx chromosome and a resulting dicentric with the C and wx alleles. Crossovers between C and the knob result in a knob-c-wx chromosome whose involvement in a bridge-breakage-fusion cycle would not be detected genetically. Knobbed c Wx chromosomes capable of causing Wx-wx mosaicism arise from the infrequent double exchanges in the knob-C and C-Wx regions.



From the cross of c wx♀ X 33166 C Wx/N9 yg2 c wx♂ the following data were obtained:

Kernel phenotype	Number
C Wx	175
C wx	42
C-c Wx-wx, whole kernel mosaic for C-c and Wx-wx. Break at 2nd microspore division in region 1	70
Half C Wx, half C-c Wx-wx. Break at first endosperm division in region 1	80
Half C-c Wx-wx, half c wx. Break at 2nd microspore division in region 1 followed by break proximal to Wx at first endosperm division	1

Kernel phenotype	Number
C-c wx, whole kernel mosaic for C-c. Break at 2nd spore division in region 1	19
Half C wx, half C-c wx. Break at first endosperm division in region 1	11
c Wx-wx. Break at 2nd spore division in region 2	8
Half c Wx, half c Wx-wx. Break at first endosperm division in region 2	20
c Wx	69
c wx	324
	819

The position and time of bridge breakage in the mosaic kernels can be accurately deduced from their mosaic patterns with the exception of the c Wx-wx and c Wx, c Wx-wx classes. Here there is some difficulty in deciding whether the Wx-wx mosaicism includes the whole endosperm or is sectored for stable and mosaic portions. A further difficulty is that the Wx-wx phenotype could represent a break in region 2 between C and Wx in a 33166 chromosome or any break distal to Wx in regions 1 and 2 in a 33166 chromosome of double crossover origin. Even though we know that the great majority of breaks in a 33166 chromosome occur in region 1, we cannot with impunity assign all of the breaks in colorless kernels mosaic for Wx-wx to region 2. Most of them probably are, and are so designated in the tabulation. Considering only the kernels with C-c mosaicism, which come from breaks in region 1, there are 90 from breaks in bridges formed at the 2nd microspore division and 91 from breaks in bridges arising at the first endosperm mitosis. It should be emphasized that the initial breaks at either cell division stem from the delayed replication of the small heterochromatic knob terminating the short arm of the 33166 chromosome. Once the primary break has been induced, the pattern of C-c mosaicism is sensibly similar in endosperms coming from either second microspore or first endosperm breaks--i.e., once the bridge-breakage-fusion cycle has been initiated, the position of subsequent breaks in the developing endosperm is the same in dicentrics of different origin.

An alternative to the above explanation to account for the fractional kernels with half self-colored aleurone and half with the bridge-breakage-fusion cycle is the possibility of a healing in the first endosperm division of the broken end of one of the two chromatids while the other chromatid undergoes the customary bridge-breakage-fusion cycle during development. We are not favorably disposed towards this hypothesis because there is no extant evidence for cessation of the bridge-breakage-fusion cycle during endosperm ontogeny once it has begun.

It is of some interest that Carlson found that nondisjunction of the B^9 chromosome was not restricted to the second microspore mitosis and could occur in an early endosperm cell.

M. M. Rhoades and Ellen Dempsey

Proof that the hi-loss phenomenon is caused by late knob replication

Rhoades and Dempsey have published extensive data demonstrating that the B chromosomes of maize induce chromatin elimination from knobbed chromosomes of the regular complement (A chromosomes). The hi-loss phenomenon takes place at one specific mitosis--the second microspore division. This is the mitosis in which nondisjunction of B chromosomes occurs. Since both phenomena occur in the same cell division, they postulated that the B chromosomes induced a delayed replication of the heterochromatic knobs of the A chromosomes and of the proximal heterochromatic knob immediately adjacent to the centromere of the B. Late replication of the B's proximal knob caused B non-disjunction while delayed replication of the

distally situated knobs of the A chromosomes led to the formation and subsequent breakage of the dicentric bridges produced at anaphase.

Their genetic data involving the position and frequencies of chromatin loss for five different knobbed chromosomes are wholly consistent with this hypothesis. However, confirming evidence is needed before giving it unequivocal acceptance. The validity of the Rhoades-Dempsey hypothesis was tested by determining cytologically the kinds of structural changes found in the progeny of hi-loss crosses. To this end, crosses were made with pollen from a hi-loss strain onto silks of yg2 plants. Chromosome 9 of the hi-loss strain used in this experiment had the Yg2 allele, a large knob on 9S and a sufficient number of B's to insure induction of the hi-loss phenomenon. Several chromosomes of the complement were knobbed and these would also undergo breakage and chromatin loss at the second microspore division. The Yg2 locus is near the tip of 9S; all breaks in 9S between Yg2 and the centromere yield a chromosome 9 deficient for the Yg2 allele. Fertilization of the egg pronucleus by this deficient sperm produces a hemizygous yg2 seedling. All of the exceptional yg2 seedlings were grown to maturity and P.M.C.'s taken for pachytene examination. The exceptional yg2 plants which were amenable to cytological analysis all had a deficient 9 as expected. Most of them possessed a paternally-derived chromosome 9 with a simple terminal deficiency but in some plants structural rearrangements were found involving chromosome 9 and another knobbed chromosome. It is through the analysis of these structural rearrangements that critical evidence came bearing on the correctness of their hypothesis.

Let us consider the constitution of the sperm cells produced by breakage of the postulated dicentric bridges at the second microspore anaphase following concomitant breaks in the short arm of 9 and in a second knobbed chromosome. For illustrative purposes we have chosen a chromosome 3 with a knob in the long arm. The diagram is self-explanatory.

The Rhoades-Dempsey hypothesis assumes dicentric formation as the result of delayed knob replication. It is further assumed that, following breakage at anaphase, the knobbed acentric fragment passes to the same pole as does the intact knobbed chromatid because the two are conjoined by the still undivided knob. Replication of the knob finally occurs and at telophase or interkinesis the acentric fragment with a freshly-broken end is no longer attached. Fusion of broken ends to give a translocation can occur at this time in telophase nuclei with two broken ends. Presumably it is a matter of chance if breaks in a dicentric bridge occur above or below the equatorial plate.

Sperms 1 and 2 are produced when breaks in the two dicentric bridges are on the same side of the equatorial plate. Sperms 3 and 4 result from one break above and the other below the equatorial plate. Fertilization of a yg2 egg by sperm 1 gives a yg2 plant deficient for segments of chromosomes 3 and 9 and possessing a dicentric chromosome. This chromosome would be subject to strong selection against its survival in the sporophytic tissue because it would undergo the chromosome type of bridge-breakage-fusion and hence tend to be eliminated. It is unlikely that the population of exceptional yg2 plants surviving to maturity would include individuals of this constitution.

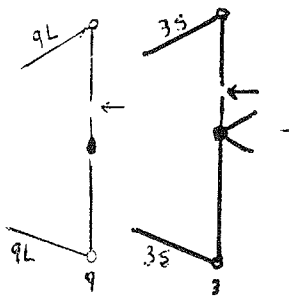
Sperm of class 2 would give a normal Yg2 zygote with structurally intact chromosomes 3 and 9.

Sperm of class 3 produce yg2 sporophytes with the $T9^3$ chromosome (deficient for the Yg2 allele) which is capped by a piece of 3L, a normal chromosome 9 and two normal chromosomes 3. These plants are trisomic for part of 3L and give chains of 4 at diakinesis.

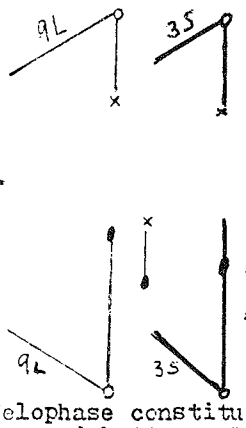
Sperm of class 4 yield Yg2 zygotes with two normal chromosomes 9, one normal 3 and the $T3^9$ translocated chromosome.

The zygotes from the functioning of sperms 3 and 4 are complementary. Those from sperm 3 are partial trisomics for chromosome 3 and those from class 4 are

I.

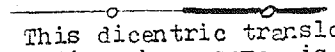


Anaphase of 2nd microspore mitosis . Short arrows indicate breaks in the two chromosomes in the same side of the equatorial plate.



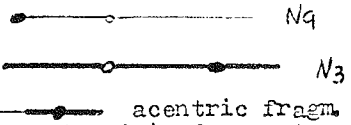
Telophase constitution at top and bottom poles following breakage and replication of knob. Since the knob was non-replicated at anaphase, both knobs pass to the same pole.

Sperm(1)



This dicentric translocation chromosome is deficient for segments of 9S and 3L.

Sperm(2)

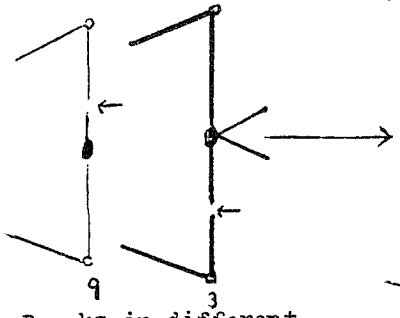


acentric fragm.

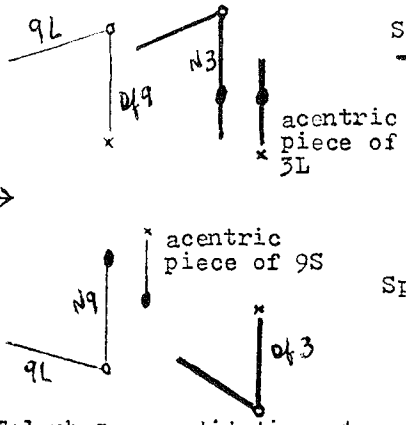
Acentric fragment will be lost in embryonic divisions.

Constitution of the two sperm cells following fusion of broken ends.

II.



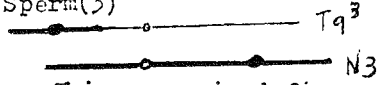
Breaks in different sides of equatorial plate



Telophase constitution at top and bottom poles following breakage and replication of knob.

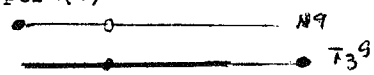
3.

Sperm(3)



This sperm is deficient for distal tip of 9S and redundant for distal segment of 3L

Sperm(4)



This sperm is deficient for piece of 3L and redundant for distal end of 9S.

Constitution of the two sperm cells following fusion of broken ends.

partial trisomics for chromosome 9. However, zygotes from class 4 sperm would not be detected in our experiments because they give rise to green plants and loss of the Yg2 allele was the marker used for selecting those plants for cytological studies. They should be found if P.M.C.'s were taken from green as well as yg2 plants.

In summary, when there are concomitant breaks at the second microspore anaphase in two different knobbed chromosomes, the only type of structural rearrangement expected in the exceptional yg2 plants is the class which is partially trisomic. Reciprocal translocations are not expected.

Given above are the theoretical expectations on the Rhoades-Dempsey hypothesis. Do the cytological observations support or negate this hypothesis? To date, I have examined meiosis in 97 exceptional yg2 plants. A simple terminal deficiency in chromosome 9 was found in 83. However, 14 of the yg2 plants possessed a translocation complex derived from breaks in chromosome 9 and in a second chromosome. These are as follows:

Plant #	Translocated chromosome	Partially trisomic for	Knob present in translocated chromosome
1. 166-1	T 9^3	3L	yes
2. 253-6	T 9^2 or T 9^5	2L or 5L	*
3. 253-10	T 9^3	3L	yes
4. 258-1	T 9^4	4L	yes
5. 259-2	T 9^3	3L	yes
6. 260-1	T 9^3	3L	yes
7. 264-3	T 9^6	6L	*
8. 281-4	T 9^6	6L	*
9. 304-3	T 9^5	5L	yes
10 335-2	T 9^5	5L	yes
11. 342-2	T 9^4	4L	yes
12. 400-5	T 9^2	2L	yes
13. 33878-21	T 9^2	2L	yes
14. 33893-1	T 9^2	2L	yes

*Due to the poor quality of the pachytene preparations, it was not possible to be sure if the translocated segment carried a knob.

Fourteen half-translocations have been generated which involve chromosome 9 and either chromosome 2, 3, 4, 5 or 6, all of which were known to be knobbed in the hi-loss stock. All pieces of a heterologous chromosome translocated to 9S were knobbed.

The reciprocal translocations normally occurring following the induction of breaks in two different chromosomes are non-existent or infrequent among the structural rearrangements found in the progeny of hi-loss plants. Only half-translocations (the recovery of one of the two expected translocated chromosomes) were found although breaks had clearly taken place in two chromosomes. These results are comprehensible on the Rhoades-Dempsey hypothesis, according to which reciprocal translocations should not occur. None were found. Sister sperm 3 and 4 each have one of the two expected translocated chromosomes. However, they were sequestered to opposite poles of the spindle as shown in the diagram and there was no opportunity for the fusion of broken ends needed to produce two reciprocally translocated chromosomes in one nucleus. The finding of 14 half-translocation complexes and of no reciprocal translocations constitute convincing evidence of the correctness of the Rhoades-Dempsey hypothesis.

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The location of Adh relative to certain translocation breakpoints

In order to manipulate the dosage of Adh and investigate the nature of ADH compensation (Birchler, 1977, MGNL 51:13), it was deemed necessary to localize the Adh locus relative to selected translocation breakpoints. It was previously known that the Adh locus was uncovered by TB1La and genetically localized to approximately 1.5 m.u. from lw (D. Schwartz, 1971, Genetics 67:411-425).

Experiments were designed to further localize Adh by producing interstitial chromosomal trisomics by the method outlined by D. M. Gopinath and C. R. Burnham (1956, Genetics 41:382) and by testing for the inclusion of Adh in compound B-A translocations (F. A. Rakha and D. S. Robertson, 1970, Genetics 65:223).

Basically, the experimental design was to cross together two different translocations, such that the breakpoints of both translocations are in the same two chromosome arms. Yet the breakpoint in one chromosome is proximal in one translocation and distal in the other and the breakpoint in the other chromosome must be distal in the first translocation and proximal in the second.

When a plant, heterozygous for interchanges of the types just described, proceeds with meiosis four types of gametophytes are formed, if homologous centromeres segregate during meiosis: 1) balanced translocation #1; 2) balanced translocation #2; 3) deficiencies for the regions between the breakpoints; 4) duplications for the regions between the breakpoints. Type three gametophytes usually abort. Thus if the heterozygote is used as a female, the resulting ear is about 25% sterile, and has 33% of the kernels with interchange #1, 33% of the kernels with interchange #2 and 33% segmentally trisomic for all the regions between the translocation breakpoints. Crossing over between the breakpoints will result in the production of normal chromosomes and insertion of the region between the breakpoints of one chromosome between a duplication for the region between the breakpoints on the other chromosome. Only when the complementary crossover strands segregate together would a viable gametophyte be produced. This gamete type would be duplicate for the regions between the breakpoints. Adjacent-2 segregations would produce severely deficient gametophytes which would be expected to abort.

In order to locate the Adh locus cytologically, heterozygotes of two appropriate interchanges were crossed as females by normal pollen from plants that were homozygous for an Adh-C (or W) electrophoretic variant. Since the C variant is not found in natural populations of maize, there will be a detectable heterozygosity for ADH mobility in the resulting scutella from such a cross. A majority of maize varieties have an Adh-F allele. If the Adh locus is located between the breakpoints of the two translocations, approximately a third of the kernels should show a 4:4:1 zymogram ratio for the FF homodimer, FC heterodimer, CC homodimer isozyme bands. These kernels are segmentally trisomic for the regions of the two chromosome arms between the breakpoints. The remaining two-thirds (euploids) of the kernels will show a zymogram ratio of approximately 1:2:1 FF:FC:CC isozyme bands.

If one of the translocations carries an Adh-F variant and the other carries an Adh-S variant, the resulting segmental trisomic scutella would show an Adh F/S/C constitution. The remaining scutella will be either C/S or C/F in genotype. If the Adh locus lies outside the region spanned by the breakpoints, none of the scutella would give zymograms that indicate trisomy for the Adh locus. That is, all kernels will show a 1FF:2FC:1CC isozyme band ratio if both translocation stocks have the Adh-F variant. If the heterozygote of two interchanges is also heterozygous for Adh-F and S variants, there will be only F/C and S/C scutella. Figure 1 lists the combinations of translocations tested for the inclusion of the Adh locus between the breakpoints. The reported breakpoints are also listed.

These combinations were crossed by homozygous Adh-C (or W) males, and the resulting genotypes from each combination are given in Table 1. Two of the combinations (#6,8) reported here produced segmental trisomics for Adh. Both of these had an Adh-F linked to one translocation and an Adh-S linked to the other. Consequently, both combinations produced approximately 33% F/C, 33% S/C and 33% F/S/C scutellar genotypes. Both of these combinations involve 1-3 translocations. The regions which are made trisomic, as based on the reported breakpoints only, are for combination #8:1L 0.72-0.90; 3L 0.65-0.73 and for combination #6: 1L 0.66-0.90; 3L 0.65-0.87. These data then place Adh distal to the breakpoint of T 1-3(5267) and T1-3(5476) and proximal to the breakpoint of T1-3(5242).

As a further check on this order, a compound TB-1L-3L (5267) translocation was synthesized. This was accomplished by crossing a hyperploid TB-1La (1 B1 B1 1B) female by the homozygous translocation 1-3(5267) and then crossing the F_1 plants, which include among their number balanced euploids heterozygous for both TB-1La and T1-3(5267), as males onto an a A2 C C2 R-scm-2 tester. This tester will allow the detection of newly arising compound TB-1L-3L's if they carry the A locus because the scutellum will be colored but not the

FIGURE 1. Combinations of overlapping translocations successfully tested and their reported breakpoints.

#	Translocations and Breakpoints
1	1-3e: 1L 0.58; 3L 0.45
	1-3(8405):1L 0.60; 3L 0.31
2	1-3(5476): 1L 0.66; 3L 0.87
	1-3(5267): 1L 0.72; 3L 0.73
3	1-6(4456): 1L 0.71; 6L 0.30
	1-6(5225-4): 1L 0.61; 6L 0.72
4	1-10(8375): 1L 0.69; 10L 0.64
	1-10 (d): 1L 0.50; 10L 0.68
5	1-9 (4997-6): 1L 0.37; 9S 0.28
	1-9 (4398): 1L 0.51; 9S 0.19
6	1-3(5476): 1L 0.66; 3L 0.87
	1-3(5242):1L 0.90; 3L 0.65
7	1-8(6766): 1L 0.54; 8L 0.77
	1-8(5821): 1L 0.65; 8L 0.31
8	1-3(5267): 1L 0.72; 3L 0.73
	1-3(5242): 1L 0.90; 3L 0.65
9	1-8(026-2): 1L 0.49; 8L 0.80
	1-8(6766): 1L 0.54; 8L 0.77

Table 1. ADH genotypes in the progeny of the translocation heterozygotes X Adh-C or Adh-W.

Combination	ADH Genotypes					
	CF	CS	CFS	WF	WS	WFS
1	33					
2	36					
3	76					
4	22	11				
5	54					
6	137	144	126			
7	7	10				
8	157	168	149	30	49	31
9				36		

aleurone if the sperm carrying the nondisjoined A locus fertilizes the egg instead of the polar nuclei. Such kernels were found. They were crossed again as males onto the a A2C C2 R-scm-2 tester to confirm their identity as a compound Tb-1L-3L (5267) translocation as opposed to a heterofertilization event. All such kernels so tested produced progeny indicative of the synthesis of a TB-1L-3L (5267) translocation, i.e., kernels with A scutellum and a endosperm; kernels with a scutellum and A endosperm; kernels with a scutellum and a endosperm; and kernels with A scutellum and A endosperm. The last two classes were less frequent than the first two classes.

When one of these confirmed cases of TB-1L-3L was crossed to an Adh-C tester, there were no scutella out of 72 analyzed which indicated nondisjunction of the Adh locus at the second microspore division. That is, there were no C/- or C/F/F, only C/F scutella.

These observations indicate the following: (1) The translocation T1-3(5267) has a breakpoint proximal to the A locus in 3L. (2) The breakpoint of this translocation is proximal to Adh in 1L. Therefore, in the combination T1-3(5267) and T1-3(5242), the former translocation has a breakpoint proximal to Adh and the latter must be distal to Adh. This conclusion is in agreement with the relative position of the reported breakpoints.

Further studies on the localization of Adh involved a test of whether the locus was included in TB-1La-5S(8041). This compound TB-A involves TB-1La and T1-5(8041)(1L 0.80; 5S 0.10). It was synthesized by Dr. D. S. Robertson and kindly supplied to me by him. The translocation stocks carried Adh-S and Adh-F. When hyperploid plants heterozygous for the translocation were crossed as males onto Adh-C or W silks, the results shown in Table 2 were found. As one can see, there is a low frequency of cases in which only the maternal electrophoretic

Table 2. ADH Genotypes in kernels from the cross of normal C/C or W/W by TB-1La-5S (8041).

	Large Kernels						Small Kernels			
Ear #	C	C/S	C/F	W	W/S	W/F	C/F	C/F/F	W/F	W/F/F
1	2	21	36				33			
2				5		43			22	
3		1	10				7			
4			11				6			

variant was present for two of the four ears analyzed. Otherwise, there are no ADH genotypes which would be expected if the locus were included in the portion of 1L remaining in the compound. If the Adh locus were included, scutella with only the maternal allele as well as scutella which show ratios typical of trisomics (i.e., C F F or C S S) in the defective endosperm (hypoploid) class of kernels would be found. Isozyme ratios in the hyperploid scutella were typical of diploid heterozygotes. They showed a ratio of CC:CF:FF of approximately 1:2:1 in the mature scutellum. The isozyme ratios of the normal kernels on the ear did not differ from those in the defective kernel class. When seedlings were grown from these two classes of kernels and ADH ratios compared from epicotyl and root tissues there was still no difference in isozyme band ratios. The exceptional scutella which had only the maternal allele are therefore believed to have arisen from self pollination or from an occasional crossing over event in the translocation parent to reconstitute the original TB-1La, which would then uncover the Adh locus. As stated above, the reported 1L breakpoint of T1-5(8041) is 0.80. Thus,

the Adh locus is approximately between 0.80 and 0.90 on 1L when the data from overlapping translocations and compound TB-A's are considered together. This determination is, of course, only as accurate as the reported cytological determinations.

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On the nature of the compensation of alcohol dehydrogenase in a dosage series of 1L

In a previous report (1977, MGCNL 51:13) it was noted that scutellar alcohol dehydrogenase (ADH) levels in a 1, 2, 3, 4 dosage series of 1L did not follow a direct proportionality to dosage even though the structural locus was varied. Among the possible explanations of this phenomenon, two were discussed. One interpretation is that the amount of enzyme produced is limited by an Adh specific limiting factor (see Genetics 67:411). This factor would be produced elsewhere in the genome and specifically limit the activity of the Adh genes. Thus the level of enzyme would not depend upon the number of structural genes present. A second explanation is that an inverse effect of varying 1L similar to that observed for glucose 6-phosphate, 6-phosphogluconate and isocitrate dehydrogenases would cancel a positive gene dosage effect of Adh. That is, in monosomics a reduction in ADH expression due to a reduced number of structural genes is compensated by an increased expression observed for specific enzymes. Likewise, in trisomic scutella any expected elevation in activity due to an increased number of structural genes would be cancelled by a reduction caused by a negative trisomic effect.

In order to further delimit the nature of ADH compensation, the following experiments were performed. It was reasoned that if ADH compensation were due to a cancellation of a gene dosage effect by a negative aneuploid effect, the following could be demonstrated: (1) smaller segmentally aneuploid regions that include the Adh locus should demonstrate a dosage effect for ADH; (2) a 1, 2, 3 dosage series of a portion of 1L that does not include the Adh locus should show an increase for ADH in the monosomic and a decrease in the trisomic. The magnitude of aneuploid changes must be such that when the results of 1 and 2 are multiplied together they would give the 1L observed values.

As a partial test of a dosage effect for ADH in smaller aneuploid regions, ADH, MDH (malate dehydrogenase), G6PDH (glucose-6-dehydrogenase), 6PGDH (6 phosphogluconate dehydrogenase), IDH (isocitrate dehydrogenase) and total protein were measured in trisomic scutella vs disomic scutella from kernels resulting from a cross of a female, heterozygous for T1-3(5267) and T1-3(5242), by a normal male. This cross is described in greater detail in the previous report. Scutella of Adh-S/C/F genotype (trisomic) were compared to Adh-C/F (disomic) for their ADH/MDH ratios. These data are given in Table 1. The mean ($\pm$ s.e.) 3 dose/2 dose ratio for ADH is 1.51 ± 0.059 and is clearly proportional to gene dosage in this segmental trisomic which is considerably smaller than the trisomics produced by TB-1La. The values observed here differ from the 1.16 mean trisomic/disomic ratio found in whole arm comparisons (Birchler, Ph.D. thesis, Indiana). The straightforward interpretation is that Adh shows a gene dosage effect in this trisomic-disomic comparison. Other possibilities include the following. The small partial 3L trisomic region has a positive effect on ADH levels. Since ADH levels (Table 2) in a 3L(TB-3La) dosage series do not show a trisomic effect, this seems unlikely. Secondly, a gene for an Adh limiting factor is encoded in the 1L or 3L region that is trisomic. The increase in ADH would then be due to an increase in its limiting factor. This possibility would require that an inverse effect would work on the limiting factor locus or its product in the aneuploids produced by TB-1La in order to produce the ADH compensation observed.

Table 1. Trisomic/disomic ratios corrected to MDH in segmental trisomics produced by T1-3(5267)/T1-3(5242). Trisomic kernels were Adh-S/F/C; disomic kernels were Adh-C/F.

Ear	ADH/MDH	G6PDH/MDH	6PGDH/MDH	IDH/MDH
1	1.47	0.97	0.98	0.97
2	1.38	0.91	0.92	1.07
3	1.74	1.10	1.10	1.16
4	1.45	0.87	0.98	0.90
5	1.43	0.92	0.92	0.90
6	1.59	0.94	0.94	1.02
Mean	1.51	0.94	0.97	1.00
s.e.	0.059	0.035	0.030	0.045

Table 2. Ratios of enzyme activities in a dosage series produced by TB-3La.

Comparison	ADH	G6PDH	6PGDH	MDH	IDH	Protein
monosomic:disomic	1.34	1.16	0.94	1.30	1.12	1.13
trisomic:disomic	1.02	1.16	1.08	1.01	1.11	1.09

Perhaps segmental trisomics involving other 1L breakpoints as well as other chromosome arms than 3L as the concomitant trisomic region will test these possibilities. Translocation combinations involving the region of 1L that will triplicate Adh as well as regions of 8L and 10L have been made and will be tested.

Another experiment designed to test whether a dosage effect for ADH is demonstrable in otherwise monosomic, disomic and trisomic scutella involved crossing the T1-3(5267)/T1-3(5242) heterozygote by TB-1La. This cross will add an extra dose of Adh to scutella monosomic, disomic and trisomic for the region of 1L involved in TB-1La.

In the interchange heterozygote mentioned above, Adh is also heterozygous for F and S alleles. Thus when it is used as female three types of viable gametes, with respect to Adh genotype, are formed: F, S and F/S. The F/S gametes are duplicate for the "between the breakpoints" regions of 1L and 3L. This 1L region includes the Adh locus. Thus if the interchange heterozygote is crossed by a plant hyperploid (1 B1 B1 1B) for TB-1La and homozygous for Adh-C, which will produce gametes with 0, 1 or 2 doses of 1L marked by this different Adh variant, the resulting zygotes are as follows: F/-, S/-, F/S/-, F/C, S/C, F/S/C, F/C/C, S/C/C and F/S/C/C. The first three classes of zygotes are monosomic for 1L; the second group of three have two doses of the region involved in TB-1La; the last three are trisomic for 1L. In each group the one with F and S have an extra dose of 1L 0.72-0.90. Thus one can compare monosomics to monosomics plus an extra Adh; disomics to disomics plus an extra Adh; and trisomics to trisomics with an extra Adh as well as the usual monosomic, disomic, trisomic comparison--all from the same ear. All classes of zygotes can be distinguished from the others on the basis of which Adh alleles are present and their isozyme band ratios. ADH was measured in the following classes of scutella: F/-, S/-, F/S/-, F/C, F/S/C, F/C/C and F/S/C/C. The ratios of ADH activities in various comparisons are given in Table 3.

Table 3. ADH/MDH activity ratios from the cross T1-3(5267)/T1-3(5242) by TB-1La C C C.

Adh Genotype	Dosage of Adh	Dosage of 1L	Observed ADH/MDH	Expected from dosage effect
F:F/C	1/2	1/2	0.66	0.50
S:F/C	1/2	1/2	0.67	0.50
F/C/C:F/C	3/2	3/2	1.10	1.50
F/S:F/C	2/2	1/2	1.22	1.00
F/S/C/C:F/C	4/2	3/2	1.40	2.00
F/S:F	2/1	1/1	1.84	2.00
F/S:S	2/1	1/1	1.76	2.00
F/S/C:F/C	3/2	2/2	1.39	1.50
F/S/C/C:F/C/C	4/3	3/3	1.27	1.33

The ADH expression in monosomics shows a typical level compared to the disomic (0.66). Also in the whole arm trisomic, ADH is compensated, showing 1.10 as the 3/2 dose ratio. The two doses of Adh in the monosomic class show close to twice (1.80x) as much ADH as found in the one dose monosomic. The three doses of Adh in the disomic show 1.39 times as much ADH activity as present in the disomic. Thus from kernels on the same ear the small trisomic region around Adh shows a significantly greater ADH expression than in the larger trisomic region (small/large = 1.39/1.10). Furthermore, if an extra Adh dose is added to the trisomic, a dosage effect is observed. One would expect the 4/3 dosage effect to be 1.33. The observed value was 1.27. If one compares these four doses in a whole arm trisomic to two doses in the disomic, one finds a ratio of 1.40. For a strict dosage effect, a ratio of 2.00 would be expected. Thus the trisomic condition must reduce the total output of the four doses to 70% of the expected value from a dosage effect.

From these data it appears that the expression of ADH is positively affected by the dosage of the Adh locus and negatively correlated with the dosage of 1L. At any level of aneuploidy, an increase in Adh dosage shows a dosage effect reasonably close to the expected effect. However, if the dosage of 1L is varied, there is a tendency for ADH compensation.

Further work on this problem will involve testing compound TB-A's which have replaced the terminal end of 1L with some other phenotypically marked region as well as placing the terminal end of 1L (using Adh as a genetic marker) onto other TB-A's. The compounds constructed will test several lengths of 1L proximal to Adh in a 1, 2, 3 dosage series as well as distal regions including the Adh locus. This will leave only the region distal to Adh to be tested independently of Adh dosage.

In conclusion, this progress report has noted evidence that suggests, but does not prove, that the compensation of ADH in the dosage series produced by TB-1La is due to a cancellation of a gene dosage effect by an inverse effect produced by 1L. Further studies as noted above will be conducted in order to discriminate between the other mentioned possibilities.

James A. Birchler

Protein profiles in selected dosage series

In a previous report (Birchler, 1977, MGCNL 51:13) it was noted that glucose-6-phosphate, 6-phosphogluconate and isocitrate dehydrogenase activities were negatively correlated with the number of 1L chromosome arms present in a dosage series produced by TB-1La. These enzymes were increased in monosomics and

decreased in trisomics relative to the disomic. Other enzyme activities were constant through the dosage series.

To test whether a similar phenomenon occurred on the protein level, aneuploids of several regions of the maize genome and their respective euploids were analyzed on SDS polyacrylamide gels to compare protein profiles. Electrophoresis and staining were by the method of Laemmli (Nature 227:680-685, 1970). Gels were 10% acrylamide. One scutellum was extracted per one ml of sample buffer. Fifteen microliters of extract were applied to each channel.

The regions studied were 1L, 3L, 4S, 5L, 7L and 9S. The dosage series for each region was generated as follows. For 1L, plants homozygous for an Adh-F (alcohol dehydrogenase) allele were crossed as females by males hyperploid for TB-1La (1 B1 B1 1B) and homozygous for an Adh-C allele. Monosomic kernels have only the Adh-F allele and originate from fertilization of the egg by the deficient sperm which is formed as a result of nondisjunction of the B1 chromosome at the second microspore division in B1 1B male gametophytes. The disomic scutella have Adh-F and Adh-C alleles present which form a three banded isozyme pattern in which the CC:CF:FF band ratio is approximately 1:2:1. These zygotes originate from fertilization of the egg by either a sperm with the normal chromosome one or a sperm from a 1B B1 pollen grain in which disjunction of the B centromere occurred. The trisomic scutella have one Adh-F and two Adh-C alleles present. The CC:CF:FF isozyme patterns of these scutella are in an approximate 4:4:1 ratio. These arise from fertilization of the egg by sperm carrying two B1 chromosomes as a result of nondisjunction at the second microspore division. Hyperploid (1 B1) pollen tubes are successful in competition at a frequency of less than 0.0002 (Birchler, unpublished) and would not contribute to the last class. Kernels were classified by subjecting an extract of a sliver of each scutellum to starch gel electrophoresis.

The dosage series for 3L was generated by crossing an a-m-1 A2 C C2 R-scm-2 female by a hyperploid male (3 B3 B3 3B) which carried the recessive a allele in the normal chromosome three. The B3 chromosomes carried the dominant A allele. This female line allows the expression of anthocyanin in scutellar tissue when crossed by another line possessing a dominant A allele. Since it is known that there is little recombination between the normal chromosome and the B3 translocated chromosomes and that hyperploid pollen duplicated for such long regions cannot successfully compete against haploid pollen, the following phenotypes are believed to correspond to the respective dosage of 3L: colorless embryo, purple aleurone = 1 dose embryo; purple embryo and scutellum = 2 dose embryo; purple embryo and colorless aleurone = 3 dose embryo; and colorless embryo and colorless aleurone = 2 dose embryo.

Aneuploids for 4S were generated by the following protocol. Females homozygous for sugary, su, were crossed by a hyperploid 4 B4 B4 4B plant homozygous for the starchy allele, Su. The sugary kernels in the progeny are segmental trisomics in the scutellum; the starchy kernels have either disomic or monosomic scutella. As a control on the effects of sugary endosperm on the scutellum, a backcross of Su/su X su/su was used as a source of segregating starchy vs. sugary kernels.

Aneuploids for 5L were generated in the following manner. Females of A A2 C C2 R-scm-122 pr were crossed by hyperploid 5 B5 B5 5B males which had pr on the normal chromosome 5 and Pr on the two B5 chromosomes. Since the pr locus is uncovered by TB-5La, kernels with pr scutellum and Pr aleurone are considered to be monosomic for 5L. Kernels with Pr aleurone and scutellum are considered to be disomics, resulting from union of a haploid egg with a balanced sperm from a pollen grain in which disjunction of the B centromere occurred. We note that if 5 B5 pollen tubes could compete with the euploid tubes, the resulting phenotype would be the same, but heavy pollinations were made to minimize this possibility. Kernels with Pr scutellum and pr endosperm are segmentally trisomic for 5L. This translocation was kindly supplied to me by Dr. J. B. Beckett.

Aneuploids for 7L were generated by crossing Esterase-1 N/N females by a hyperploid male 7 B7 B7 7B with E-F on the normal chromosome seven and E-S on the B7 chromosomes. The E locus is uncovered by TB-7Lb (D. Schwartz, personal communication). Monosomic kernels have only the E-N allele present. Disomic kernels are E-N/S and trisomic kernels are E-N/S/S. The latter two types of scutella can be distinguished from each other on the basis of isozyme band ratios. Moreover, the N/S/S class has an associated endosperm which is reduced in size.

Aneuploids for 9S were generated as follows. An A A2 C C2 R-scm-2 stock was used as a female for homozygous TB-9Sb also homozygous for C-I. This stock was kindly supplied to me by Dr. D. S. Robertson. In this case the colored scutellum, colorless aleurone class is considered monosomic; the completely colorless class disomic; and the colorless scutellum, colored aleurone class segmentally trisomic.

Scutella of the dosage series of these regions were examined on SDS polyacrylamide gels for the pattern of proteins in each aneuploid and the diploid. The number of discrete protein bands was > 60. Each dosage series produced a unique set of protein intensities. The most common effects were specific protein increases in monosomics, decreases in trisomics or a complete negative correlation with chromosome dosage. A positive correlation between protein intensity and chromosome dosage was observed for certain proteins in certain dosage series.

Of particular note from this study is the response of Protein-1, one of the major extractable scutellar proteins. Variants in molecular weight have been found for this protein by Drew Schwartz (personal communication). He has mapped the locus which encodes this protein to between bz2 and Adh on chromosome 1. In this study it was demonstrated that the gene for this protein was uncovered by TB-1La, by crossing a female with one molecular weight variant by a TB-1La stock homozygous for a second variant. The monosomics, as determined by the Adh genotype, have only the maternal variant. The intensity of this variant decreased through the 1, 2, 3 dosage series, with the monosomic intensity being the greatest. The paternally contributed variant increased slightly in the trisomic compared to the disomic. In the 1L dosage series in which Protein-1 did not differ in molecular weight between the maternal and TB-1La lines, there was little or no change in intensity through the dosage series. Thus, it is concluded that even though the locus for Protein-1 is uncovered by TB-1La and the number of structural genes is varied in a 1, 2, 3 relationship in the 1L series, there is a compensation such that the total amount of protein is similar in the monosomic, disomic and trisomic. The intensity of several other proteins is negatively correlated with 1L dosage but most of the protein species have similar intensities through the 1L series.

In the various other dosage series different protein species are affected. For some chromosome arms there are several proteins with altered intensities, while other dosage series only affect a few proteins. Two of the most striking cases are the aneuploids of 4S and 5L. The intensity of Protein-1 differs greatly between the trisomic scutella and the 1 or 2 dose scutella for 4S. The intensity of Protein-1 is much less in the trisomic scutella. Examination of starchy vs. sugary kernels from a segregating ear showed that this effect was not due to the sugary endosperm phenotype associated with the trisomic scutella.

Protein-1 is also negatively affected by the dosage of 5L. In this case the intensity of this protein was increased in the monosomic scutella and decreased in the trisomics relative to the euploid class. Dosage series of 3L, 7L and 9S had little or no effect upon the intensity of Protein-1.

Thus, there are at least two regions in the maize genome that produce a negative effect upon the level of Protein-1. In addition, the level of this protein is compensated in aneuploids of 1L. The basis of this compensation is not known but these results indicate that it occurs at least on the protein level. Whether the compensation of Protein-1 and ADH (see previous report) have a similar basis is not known. The compensation could be due to feedback control, a limiting factor

produced elsewhere in the genome, a cancellation of a gene dosage effect by an inverse effect or some other basis.

As pointed out previously (1977, MGNL 51:18), multiple inverse effect regions can affect a single locus in any one tissue. In this case 4S and 5L produce a negative correlation between the intensity of Protein-1 and chromosome dosage. Furthermore, this study shows that monosomic increases are usually associated with trisomic decreases. This is an important consideration. It has been previously noted that there exist in the literature data indicating specific enzyme level reductions in trisomics of Drosophila, Datura and Hordeum. With only disomic and trisomic data it is always necessary to consider that such effects are due to the reduced vigor of trisomics or to a slight developmental or anatomical asynchrony. However, when the corresponding monosomic shows an increase for the same enzyme or protein that the trisomic decreases, such an interpretation is no longer possible, since monosomics are also almost always less vigorous than the disomic. Since there is no correlation with vigor or developmental stage, an explanation of the phenomenon must take into account the fact that there is a negative correlation of enzyme/protein level with chromosomal dosage. The only segmental monosomy data available are from maize.

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Interaction between "jumping" gene C*-IE7002, activator genes and Euchlaena mexicana "prepotent" cytoplasm

In the chart below, constitutions under (1), (2), and (3) represent the following:

(1) "Jumping" gene, C*-IE7002 located in chromosome 9 in the original line, "dominant multiple" tester S^{40}, perhaps from Dr. Randolph's No. 1877 (1933).
(2) Ac* and li* activator genes of the "jumping" gene C*-IE7002.
(3) Exceptional Euchlaena mexicana "prepotent" cytoplasm.

(1)	(2)	(3)	
+	-	-	Some mosaicism for color in anthers and ears.
+	+	-	Abnormal mendelian-chromosomal segregation of aleurone color (MNL 49:66-70; Publ. 88, Inst. Fit. Sta. Catalina, 1975).
+	-	+	In relation to Zea cytoplasm: a) Mosaicism in anthers for purple and yellow color; mosaicism in ears for purple and colorless aleurone. b) Greater percentage of pollen sterility; stickiness of chromosomes (Rev. Fac. Agr. XXXIX (2):204-05, 1963). c) Greater volume of knobs; mutational phenomena (Proc. XI Int. Cong. Genetics (1):204, 1963). d) Minor vigor (Atti IX Cong. Int. Genetics, 1228-31, 1954).
+	+ sm pl	+	In relation to Zea cytoplasm: Inherited preferential somatic segregation (Proc. X Int. Cong. Genetics (2):83, 1958).

(1)	(2)	(3)	
+	+	+	In relation to Zea cytoplasm: Strong mosaicism in anthers and endosperm with abnormal development (see this communication, Part 2).
-	±	+	In relation to Zea cytoplasm: a) Greater vegetative cycle; non-expression of ij gene (Rev. Arg. Agr. 17 (3):145-62, 1950; Rev. Inv. Agr. VIII (2):175-83, 1954; Atti IX Cong. Int. Gen., 1228, 1954). b) Greater prolificity (Rev. Arg. Agr. 25:12-44, 1958).
-	±	-	Normal

Luis B. Mazoti

Activator cytoplasm of the C*-IE7002 gene

The interaction between C*-IE7002 gene, activator genes Ac* li* (MNL 49:66-70) and "prepotent" Euchlaena mexicana cytoplasm produced strong mosaicism in ears for purple and colorless aleurone with abnormal endosperm development. The genotypes of Zea used are: (1) multiple dominant tester (C*-IE7002) A C R Pr B Pl P, perhaps from Randolph No. 1877 (1933); (2) Tester chromosome 7 gl ij; (3) (c-tester) A B pl sh wx Ac* li*.

All testers are selfed more than 40 generations with occasional sibs and with BC^{14} to Euchlaena cytoplasm.

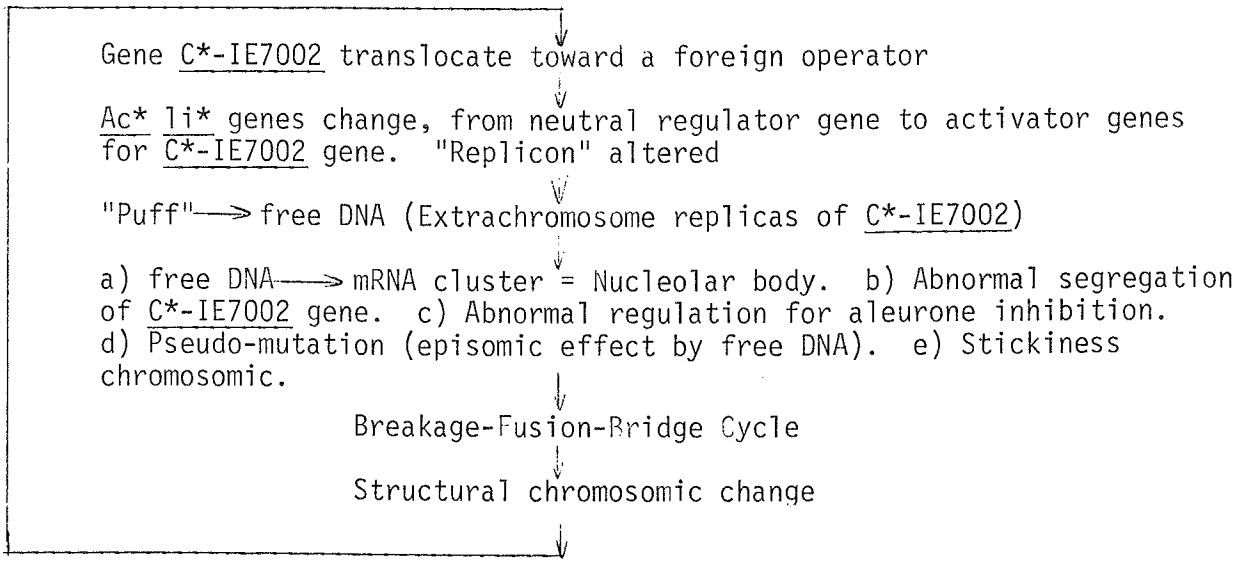
The original multiple dominant tester carries C*-IE7002 gene. The original c-tester carries activator Ac* li* genes for C*-IE7002 gene. In the present communication it was established that "prepotent" cytoplasm of Euchlaena mexicana is an activator for C*-IE7002 "jumping" gene.

Cytological analysis established: (1) normal meiosis (C.M.P.); (2) chromosome B is absent; (3) frequently nucleolar body of 1.5 to 4.5 and rarely we see puff-like in short arm of chromosome 9.

Luis B. Mazoti

Pseudo-mutational cyclic explosion hypothesis

(McClintock's work gives the base to the present hypothesis).



Luis B. Mazoti

Reciprocal crosses between Euchlaena perennis Hitch. and Zea mays L.

Reciprocal crosses between E. perennis and Z. mays were made. A very early maize line (35 days for flowering) was employed for these crosses. Highly heritable earliness was shown in F_1 plants, in the one as well as the other cross. F_1 plants are vigorous and with about 40 tillers per plant at 12 weeks of growth. The F_1 kernels resulting from E. perennis x Z. mays cross (E. perennis as mother) are highly viable, while Z. mays x E. perennis kernels (maize as mother) are little viable, since only three plants were obtained from fifty F_1 kernels.

Jorge Luis Magoja

Floury-a high lysine system

Different results were shown in reference to the action of fl-a on protein quality (MNL 45:70 and 47:166). These data led to a study in order to clear up those communications. Crosses between a high-quality floury-a line (lysine 3.5 g/16 g Nitrogen, tryptophan 0.8 g/16 g Nitrogen) and normal lines (hard endosperm) with normal level of lysine (1.6) and tryptophan (0.4) were made (these levels are expressed for defatted endosperm). The segregation for quality between F_2 kernels with floury phenotype permitted establishing a modification case on fl-a by two genes with accumulative effect and independent segregation without affecting endosperm texture (1/16 of F_2 floury kernels have the same high-quality protein as high lysine-tryptophan fl-a line). These two genes, tentatively designated lysine-1 (ly) and lysine-2 (ly2), regulate lysine and tryptophan levels in the endosperm. High lysine level is conditioned by fl-a/fl-a/fl-a ly/ly/ly ly2/ly2/ly2 genotype, and normal level by fl-a/fl-a/fl-a Ly/Ly/Ly Ly2/Ly2/Ly2 genotype. Intermediate lysine-tryptophan levels were found for all possible different genotypes. The hypothesis that two genes have interaction with the fl-a was confirmed by quality segregation between F_3 floury ears. Kernels and ears with high lysine-tryptophan levels have also approximately three times more water soluble free amino acids than their normal equivalents. Lysine-1 and lysine-2 genes in combination with fl-a have pleiotropic action for lysine, tryptophan and free amino acids of endosperm, and these features are highly positive correlated. The high-quality genetic system, floury-a lysine-1 lysine-2, modifies protein pattern: the effect was a repression of zein synthesis and a stimulation of albumin, globulin and glutelin synthesis. Floury-a high lysine has the following protein pattern: albumin, globulin 9%, zein 33% and glutelin 58% of total protein, respectively. Its normal equivalent has 4.5%, 50% and 45.5% for the same fractions, respectively. The high-quality genetic system formed by a floury gene (fl-a) and two non-floury genes, suggests the possibility of developing another system without floury genes, and a high-quality protein maize with normal genotype.

Jorge Luis Magoja

High-quality protein maize with normal genotype

High-quality protein maize with normal texture (flint endosperm) was discovered in the progenies from crosses of high-quality floury-a x normal lines. It is probable that high-quality was regulated by a genetic system without any floury gene in the background. Absence of fl-a was confirmed by crossing these strains with fl-a/fl-a lines as mothers. Expression of fl-a was not hidden by texture modifiers. These strains have normal phenotype (hard endosperm) and normal genotype (without floury genes) and it is possible, by preliminary tests, to find that high-quality protein may be regulated by a system involving very few genes, perhaps not more than two. These strains have high tryptophan level, 0.7-1.0 g/16 g Nitrogen compared with 0.45 g/16 g Nitrogen, in F_3 sibling ears.

Jorge Luis Magoja

Perennial character segregating in F_2 of Euchlaena perennis Hitchc. X Zea mays L. hybrid

In MNL 45:68 was communicated the obtaining of E. perennis X Zea mays hybrid, F_1 and F_2 study and forage potential of the sixth generation. A more detailed study of perennialism in F_2 shows that after four years of selection only 2.2% (11 plants) of the F_2 are similar to F_1 in perennialism. These F_2 plants during eight months of their vegetative cycle have produced between 20 to 120 tillers per plant and the fertility varied between 2% to 80%. Also, one progeny in the 13th generation of annual cycle has tillers ($\bar{x}$ = 11 per plant) and regular meiosis which gives fertility ($\bar{x}$ = 85%).

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Possible nonrandom disjunction of the B^9 chromosome during meiosis

In 1967, Robertson reported on meiotic segregation of the B-9b translocation heterozygote (Genetics 55:433). One conclusion was that the B^9 chromosome segregates randomly at first division with respect to its pairing partner, chromosome 9. That pairing occurred in Robertson's study was insured by the selection of c-sh crossovers between 9 and B^9 for analysis.

The experiment of Robertson with 9 9^B B^9 plants has essentially been repeated, using the longer yg2 bz interval. Testcrosses were performed as follows: 9 yg2 bz, 9^B Wx, B^9 Yg2 Bz x yg2 yg2 bz bz. In the offspring, three crossover classes are important: yg2 Bz Wx, yg2 Bz wx, Yg2 bz Wx. The yg2 Bz Wx class results from yg2-bz crossing over, followed by disjunction of the 9 and B^9. The yg2 Bz wx class can occur in two ways. Following crossing over between yg2 and bz, migration of the B^9 to the same pole as chromosome 9 can produce yg2 Bz wx. However, yg2 Bz wx may also be produced by a double crossover (cent.-bz and bz-yg2) followed by disjunction of the 9 and B^9. The third genetic class, Yg2 bz Wx, is also a double crossover product and is the reciprocal to the yg2 Bz wx double.

To compare types of segregation, the yg2 Bz Wx class is used as an indicator of 9-B^9 disjunction and the yg2 Bz wx kernels represent nondisjunction of the paired chromosomes. A correction must be made to remove double crossovers from the yg2 Bz wx class, since they result from 9-B^9 disjunction. The corrected term is yg2 Bz wx - Yg2 bz Wx. For random migration of the B^9, one might expect that yg2 Bz Wx = yg2 Bz wx - Yg2 bz Wx. However, the nondisjunction class is only expected at one-half the frequency of yg2 Bz Wx since it is derived from the anaphase I chromosome combination 9 yg2 bz wx/Yg2 bz wx + B^9 Yg2 Bz/yg2 Bz. Chromatid segregation at AII will allow recovery of the yg2 Bz wx phenotype in only one-half the cases.

For random migration of the B^9 in 9 9^B B^9 heterozygotes, one expects:

$$\frac{\text{yg2 Bz Wx}}{\text{yg2 Bz wx - Yg2 bz Wx}} = 2/1$$

The data found for the standard TB-9b were 150/(67-17) = 3.0. The results suggest that the 9 and B^9 have some tendency to disjoin from each other, although there is considerable migration to the same pole, as noted by Robertson. A collection of modified TB-9b's was also analyzed. The modified translocations lack the ability to undergo nondisjunction at the second pollen mitosis (Chromosoma 42:127). It was considered possible that they might also be altered in meiotic behavior, and the following data were gathered.

Modified TB-9b	Ratio
#1855 (telocentric B^9)	216/(81-20) = 3.54
#1866 (deletion on B^9)	69/(43-4) = 1.77
#10 (deletion on 9^B)	98/(35-8) = 3.6
#2150 (deletion on 9^B)	165/(75-13) = 2.66

Results are quite variable and may reflect differing genetic backgrounds rather than any substantive deviation from the pattern found with standard TB-9b. Tests with inbred lines are planned. In conclusion, it appears that the B^9 chromosome may not disjoin completely at random with respect to chromosome 9 in a translocation heterozygote.

Wayne R. Carlson

Telocentric B^9's produce incomplete nondisjunction and generate isochromosomes at the second pollen mitosis

Telocentric B^9's carry out nondisjunction at very low rates under most conditions (1-2%) and are considered blocked at some important step in nondisjunction (Chromosoma 42:127). However, in the presence of extra B chromosomes the rate of nondisjunction may rise appreciably. The "leakiness" of telocentric B^9's in nondisjunction is examined here.

Data reported earlier for telocentric 1852 (Proc. 1st Int. Maize Genetics and Breeding Conf. - 1975, in press) showed a rise in the rate of nondisjunction from 0.8% to 8% with the addition of 3-8 extra B chromosomes. The rate of B^9 instability also increased in the presence of added B chromosomes (Bz/bz sectoring), suggesting that the B^9 chromosome was destabilized at the second pollen mitosis by extra B's. The effect of extra B's was either nondisjunction or damage to the B^9 centromere, but not restoration of typical B chromosome behavior. It was concluded that telocentric 1852 lacks a cis genetic function essential to nondisjunction.

Findings reported here for telocentric 1854 show an increase in the rate of nondisjunction in the presence of extra B's from 1.3% to 19.6% (Tables 1 & 2). The frequency of Bz/bz sectors remains low in both the presence and absence of extra B's. It is difficult to find evidence in the data of Tables 1 and 2 for destabilization of the B^9, and one might conclude that extra B's restore to some extent the "normal" nondisjunctional properties of a B chromosome. However, nondisjunction was measured by the expression of the recessive bz phenotype in the endosperm, with the assumption that two telocentric B^9's were present in the embryo. To test the assumption, a number of bz kernels from crosses of Tables 1 and 2 were grown and the plants tested for the expected number of two B^9 chromosomes. Each plant was

Table 1. Percent bz and Bz/bz kernels in crosses of the type bz bz X 99^B telo B^9.

Male Parent	Percent bz	Percent Bz/bz*	Estimated rate nondisjunction (bz X 3). Preferential fertilization assumed.
Telo 1854 no B (99^B telo B^9 X B. mexican no B)			
1. 2380A	10/1385 = 0.7%	4/1385 = .3%	2.1
2. 2380B	10/1342 = 0.8%	5/1342 = .4%	2.4
3. 2380C	3/1510 = 0.2%	9/1510 = .6%	0.6
4. 2380D	6/1479 = 0.4%	1/1479 = .07%	1.2
5. 2380E	3/1651 = 0.2%	4/1651 = .2%	0.6
	32/7367 = 0.4%	23/7367 =0.3%	1.3%

crossed as male to a bz wx tester and some plants were also crossed as female. In 9 bz, 9^B Wx, B^9 Bz, B^9 Bz plants, pairing and disjunction of the two B^9's produces primarily 9^B Wx, B^9 Bz and 9 bz wx, B^9 Bz meiotic products (D. S. Robertson, Genetics 55:433). Due to the duplicate nature of 9 B^9 pollen, the 9 B B^9 (Wx) pollen succeeds in fertilization much more frequently than 9 B^9 (wx) pollen. The percent of Wx progeny is, therefore, expected to be significantly greater than 50% when two B^9's are present.

Table 2. Percent bz and Bz/bz kernels in crosses of the type bz bz X 99^B telo B^9 + B's.

Male Parent	Percent bz	Percent Bz/bz*	Estimated rate (bz X 4) nondisjunction. Assume no preferential fertilization.
Telo 1854 + B's (99^B telo B^9 X B. mexican + 7 B's)			
1. 2383A	49/1473 = 3.3%	3/1473 = 0.2%	13.3%
2. 2383D	51/1392 = 3.7%	3/1392 = 0.2%	14.7%
3. 2383E	101/1241 = 8 %	3/1241 = 0.2%	33 %
	201/4106 = 4.9%	9/4106 = 0.2%	19.6%

*Kernels with large sectors, covering one-fourth or more of the endosperm plus all multiple-sectored kernels are recorded as Bz/bz.

The data of Table 3 indicate that four plants (2516A, 2516B, 2571B, 2572B) from bronze kernels of Table 1 contain two B^9's. However, the remaining six plants tested gave rates of Wx transmission less than 50%. Since all six plants show some viability of Wx pollen, they were not missing the B^9 chromosome: a 9 wx, 9^B Wx plant gives all recessive progeny. It seems apparent that each plant contained one B^9. The conclusion was confirmed by reciprocal crosses carried out for some of the plants. For the chromosome type 9 bz wx, 9^B Wx, B^9 Bz, the frequency of bz wx progeny in a testcross is expected to be quite high in comparison to the low frequency expected with two B^9's. A control group, known to carry the telocentric as 9 bz wx, 9^B Wx, telo-B^9 Bz, gave a bz wx frequency of 56% (1324/2349). Rates of bz wx on the female crosses of 2516C, 2518A, 2571A and 2571C were similarly high. (No data are available for 2517A or 2572A, but their identification is unequivocal, as reported later).

Table 3. Testcross data for plants grown from bzWx kernels of Table 1. All plants were crossed as male parents to bzwx and some were also crossed as female parents.

Plant Number	Percent Wx when crossed as male	Percent bzwx when crossed as female	Classification
2516A	83% (163/196)		2-telos
2516B	84% (173/206)		2-telos
2516C	42% (70/165)	49% (88/180)	1-telo
2517A	5% (9/170)		1-iso
2518A	5% (11/218)	59% (87/148)	1-iso
2571A	41% (76/184)	61% (30/49)	1-telo
2571B	85% (164/193)		2-telos
2571C	36% (101/280)	44% (67/151)	1-telo
2572A	5% (12/240)		1-iso
2572B	79% (157/198)		2-telos

The results indicate that 0-1 disjunction frequently occurs among apparent cases of nondisjunction by telocentrics. However, three of the six plants considered to contain one B^9 gave anomalous results in the male cross. Control plants, known to carry 9 wx, 9^B Wx telo-B^9, gave 47% (1922/4071) Wx progeny when crossed as male

parent to a wx tester. Data for 2516C, 2571A and 2571C in Table 3 agree with the control. However, plants 2517A, 2518A and 2572A gave markedly lower frequencies of Wx. Subsequent cytological examination showed that the latter plants contained a B^9 isochromosome rather than a telocentric (see accompanying article). Three types of disjunction were, therefore, found: O-1, O-2, O-iso.

The bronze kernels of Table 2 were also tested for the presence of B^9's. Results from the male crosses (Table 4) indicate that 6 plants contained two telo-B^9's, 8 plants had one telo-B^9 and one plant had a B^9 isochromosome. (An additional three plants had two B^9's, but these B^9's showed very high rates of nondisjunction in testcrosses and probably were standard B^9's derived by crossing over between telocentric B^9's and standard B chromosomes). There are several difficulties with the data of Table 4. First, the isochromosome classification has not been confirmed cytologically. Second, male transmission of Wx for 2519E and 2574A seems rather low, although well above 50%. Third, the data from female crosses for bz wx frequency do not agree well with the expected in several cases. Among plants believed to have two B^9's, 2519A and 2573D have low frequencies of bz wx, as expected. However, 2519E has a very high bz wx frequency, near that expected for one B^9. Also, among plants classified as having one B^9 from the male crosses, the bz wx frequency on the female cross seems low for 2573C and 2574C. No explanation for the discrepancies can be proposed, but the effect of extra B's on pairing, crossing over and disjunction of B^9's may play a role.

Table 4. Testcross data for plants grown from bzWx kernels of table 2. All plants were crossed as male parents to bzwx and some were also crossed as female parents.

Plant Number	Percent Wx when crossed as male	Percent bzwx when crossed as female	Classification
2519A	86% (220)/(256)	9% (22)/(245)	2-telos
2519C	87% (156)/(179)		2-telos
2519D	52% (125)/(239)		1-telo
2519E	66% (130)/(196)	29% (62)/(217)	2-telos ?
2573A	88% (260)/(294)		2-telos
2573B	16% (47)/(289)		1-iso
2573C	52% (152)/(292)	36% (84)/(235)	1-telo
2573D	81% (205)/(253)	14% (38)/(277)	2-telos
2573E	50% (113)/(228)		1-telo
2573F	38% (114)/(299)	55% (112)/(205)	1-telo
2573H	49% (115)/(234)		1-telo
2574A	63% (173)/(273)		2-telos
2574B	42% (101)/(243)		1-telo
2574C	40% (139)/(350)	39% (45)/(114)	1-telo
2574D	46% (98)/(211)		1-telo

Despite the problems outlined, it appears the O-1 disjunction occurs for telocentric 1854 in the presence of B chromosomes as well as in their absence. Addition of B chromosomes does not eliminate this anomalous behavior at the second pollen mitosis. The finding suggests that B chromosomes contribute to the "stickiness" of the B^9 chromosome and make disjunction more difficult. The result may be a rise in nondisjunction, but the extra B's do not restore the missing element(s) of the telocentric which assures migration of the chromatids to one pole without difficulty.

Wayne R. Carlson

Isochromosomes from telocentric B^9's

A B^9 isochromosome was isolated in 1970 (Chrom. 30:356). It was shown to carry out nondisjunction at low, variable rates and to generate sectored kernels for B^9 markers at relatively high frequency (Chromosoma 42:127). Germination of sectored kernels produced plants that contained, in several instances, telocentric B^9's. Centromeric misdivision of the isochromosome at the second pollen mitosis apparently produced the telocentrics (E. Brannen, unpublished). Four telocentric B^9's were recovered from the B^9 isochromosome. Testcrosses showed negligible rates of nondisjunction for the telocentrics and little sector formation. Telocentric B^9's differ markedly from the isochromosome in these properties.

Telocentric B^9's occasionally give rise to isochromosomes by centromeric misdivision. Three potential isochromosomes, derived from telocentric 1854, were identified in Table 1 of an accompanying article. Cytological examination of root tip mitoses confirm the presence of an isochromosome in each case. Genetic tests indicate a low, but significant, rate of nondisjunction for each of the isochromosomes (Table 5). Also, they frequently produce sectored kernels and particularly multiple sectored kernels (Table 6). Properties of the new isochromosomes seem very

Table 5. Endosperm classification from crosses of the type bzbz X 9^B9^B iso B^9. The estimates of nondisjunction assume preferential fertilization.

Cross	Bz	bz	Estimated rate nondisjunction (bz x 1.5)	Large Bz/bz Sectors (All multiple sectors plus single sectors covering ¼ or more of endosperm)	Small Bz/bz Sectors (Single sectors covering <¼ endosperm)
A. Isochromosome 2820					
2763x2820A	204	25		40	4
2763x2820B	255	29		36	3
2763x2820C	161	30		57	1
2763x2820D	248	21		30	4
2763x2820E	216	43		93	8
	1084	148	15%	256 (17%)	20
B. Isochromosome 2821					
2845x2821A	269	33		22	4
2845x2821B	255	48		39	2
2845x2821C	230	10		8	2
2845x2821D	263	55		19	0
2845x2821E	264	50		39	2
	1281	196	18%	127 (8%)	10
C. Isochromosome 2822					
2845x2822A	307	71		27	2
2845x2822B	293	21		9	2
2845x2822C	263	65		26	5
2845x2822D	282	17		24	5
	1145	174	18%	86 (6%)	14

Table 6. Categories of sectoring among large Bz/bz sectors in Table 5.

Isochromosome	Multiple event (Two or more recessive sectors)	Single large recessive sector covering 1/4 to 3/4 of endosperm	Single large recessive sector covering >3/4 of endosperm
A. 2820	205 (13.6%)	44 (2.9%)	7 (0.5%)
B. 2821	88 (5.5%)	30 (1.9%)	9 (0.6%)
C. 2822	64 (4.5%)	21 (1.5%)	1 (0.1%)

similar to those of the original isochromosome. The results shown in Tables 5 and 6 may be compared to data for the original isochromosome in an accompanying article (Tables 7 and 8).

With the present information, it seems likely that the new isochromosomes are similar or identical in nondisjunctional properties to the original isochromosome and markedly different from telocentric 1854. If true, telocentric 1854 contains all the genetic factors needed to construct an isochromosome similar to the one from which it was derived. One may speculate, due to the work of Rhoades, Dempsey, and Ghidoni (PNAS 57:1626), that telocentrics fail to undergo nondisjunction because they lack heterochromatin on one side of the centromere. Stickiness of centric heterochromatin in both chromosome arms may play an important role in holding the centromere together for nondisjunction.

The instability of isochromosomes and their relatively low rates of nondisjunction suggest that they lack a factor, probably on the B short arm, which promotes nondisjunction. The B^9 isochromosome may stick together at sites flanking the centromere but then have difficulty in migrating to one pole.

Wayne R. Carlson

Genetics of the original B^9 isochromosome

The discovery of a B^9 isochromosome was reported in 1970 (Chromosoma 30:356). Nondisjunction of the chromosome occurred at variable rates, and an average value of 20% was found. Since the isochromosome carried out nondisjunction at significant levels, it was assumed that none of the genes required for nondisjunction was missing. Subsequently, instability of the B^9 isochromosome became apparent (Chromosoma 42:127). Twenty-two endosperm mosaics were found in a single ear of a $\underline{c}\ \underline{c}$ x $9^B\ 9^B$ iso-B^9 $\underline{C}\ \underline{C}$ cross. Cytological examination of plants from the twenty-two kernels showed that telocentric formation was associated with mosaicism. Elaine Brannen (unpublished) traced telocentric formation to the second pollen mitosis. The iso-chromosome is now considered to lack a factor, probably on the B short arm, required for efficient nondisjunction. Absence of the factor often leads to centromeric damage at the second pollen mitosis and telocentric production. However, the isochromosome also frequently undergoes nondisjunction, with no centromeric damage.

In this article, properties of the isochromosome in 9 9^B iso-B^9 and 9^B 9^B iso-B^9 plants are given. The plants were derived by self pollination of a 9 9^B iso-B^9 individual. Rates of nondisjunction and endosperm mosaicism were determined for 9^B iso-B^9 pollen. (In 9 $\underline{wx}$, 9^B $\underline{Wx}$, iso-B^9 plants, $\underline{Wx}$ progeny were selected, since the locus effectively marks 9^B iso-B^9 pollen). In addition, crosses of 9 9^B iso-B^9 plants were examined for competition between 9 $\underline{wx}$ and 9^B $\underline{Wx}$, iso-B^9 pollen types by classifying $\underline{Wx}$ vs $\underline{wx}$. Data from the crosses are given in Tables 7 and 8.

The four 9^B 9B iso-B^9 plants tested gave an average rate of 14% $\underline{bz}$ $\underline{Wx}$ kernels. Nondisjunction, assuming preferential fertilization, was, therefore, approximately 14% x 1.5 = 21%. The eight plants with the 9 9^B iso-B^9 chromosome combination showed the same average rate of nondisjunction. Endosperm instability in these crosses most frequently produced multiple recessive sectors, suggestive of bridge-breakage-fusion cycles. The average frequency of multiples for 9^B 9^B iso-B^9 plants was 2.3%. In 9 9^B iso-B^9 crosses, multiples were found in 4.3% of cases. In general, endosperm sectoring was more frequent for 9 9^B iso-B^9 than 9^B 9^B iso-B^9 plants. However, genetic background rather than 9^B chromosome content may be responsible for the difference. Transmission of the 9^B iso-B^9 chromosome combination (% $\underline{Wx}$) through pollen of the 9 9^B iso-B^9 plants ranged from 16% to 32% and averaged 24%. This is about one-half the rate found for standard 9^B B^9 pollen in a similar situation. Reduced transmission of isochromosome-containing pollen in competition with normal (9) pollen reflects the harmful effects of gene duplication.

Table 7. Testcross data from crosses of the type bz bz wx wx X 9^B Wx, 9^B Wx, iso-B^9 Bz Bz. Multiple sectored kernels have two or more recessive sectors of any size on the endosperm. Other Bz/bz kernels have a single recessive sector of the indicated size. Single events smaller than 1/16 of the endosperm were discarded.

Male Parent	Bz Wx	bz Wx	Bz/bz Wx Multiple sectors	Bz/bz Wx Single sector 1/4-3/4 of endosperm	Bz/bz Wx Single sector > 3/4 of endosperm	Bz/bz Wx Single sector 1/16 to 1/4 of endosperm
2011A	423	283	28	3	0	1
2011D	667	34	8	1	0	0
2011L	813	47	11	1	0	1
2011M	622	68	23	2	0	0
	2525	432	70	7	0	2

% bz Wx/Wx = 432/3036 = 14%; % Multiples = 70/3036 = 2.3%.

Table 8. Testcross data from crosses of the type bz bz wx wx X 9 bz wx, 9^B Wx, iso-B^9 Bz Bz. Multiple sectored kernels have two or more recessive sectors of any size on the endosperm. Other Bz/bz kernels have a single recessive sector of the indicated size. Single events smaller than 1/16 of the endosperm were discarded.

Male Parent	Bz Wx	bz Wx	Bz wx	bz wx	Bz/bz Wx Multiple sectors	Bz/bz Wx Single sector 1/4-3/4 of endosperm	Bz/bz Wx Single sector > 3/4 of endosperm	Bz/bz Wx Single sector 1/16 to 1/4 of endosperm
2011B	255	48	62	1595	7	4	0	1
2011C	285	140	85	1595	26	8	1	0
2011E	392	128	113	1883	47	14	1	2
2011F	556	135	127	1427	25	6	1	2
2011G	530	42	153	1599	6	0	1	1
2011H	382	54	86	1682	19	5	1	3
2011I	498	9	115	1932	23	7	1	3
2011J	599	56	87	1370	35	7	0	1
	3497	612	828	13,083	188	51	6	13

% bz Wx/Wx = 612/4367 = 14%; % Multiples/Wx = 188/4367 = 4.3%; % Wx/Wx+wx = 4367/18278 = 24%

Since the B^9 isochromosome shows frequent instability when crossed as male parent, cases of nondisjunction detected genetically as bz Wx kernels are suspect. The bz phenotype can result from nondisjunction with two isochromosomes in the embryo. However, 0-0 or 0-1 disjunction could also produce bz kernels. Consequently, twenty-four bz Wx kernels from crosses of plants 2011A and 2011M were germinated for examination of root tip mitoses. Among fourteen progeny of 2011A all had two isochromosome B^9's. Nine plants from 2011M had two isochromosomes, and one plant had a single isochromosome. Although a single case of 0-1 disjunction was found, the predominant event was conventional nondisjunction.

Wayne R. Carlson

Fractional formation is caused by instability of the B^9 isochromosome at the second pollen mitosis

The B^9 chromosome of TB-9b is relatively stable in mitotic divisions. However, an isochromosome derived from the B^9 frequently becomes unstable when transmitted through the male parent, producing mosaic kernels. Endosperm instability was correlated with the presence of B^9 telocentrics in plants derived from mosaic kernels, indicating misdivision of the isochromosome (Carlson, Chromosoma 42:127). Misdivision in the male parent could occur meiotically, due to orientation difficulties of a univalent isochromosome. Alternately, it could result from improper nondisjunction at the second pollen mitosis.

This problem was investigated by utilizing a C C-I B^9 isochromosome to produce endosperm mosaics. Crosses of the type C C bz bz X 9B 9B iso-B^9 C Bz C-I Bz, and C C bz bz X 9 9B C Bz C-I Bz were used to produce the mosaics. By selecting Bz/bz progeny, misdivision involving loss of the C-I arm of the isochromosome was examined. The Bz/bz kernels were germinated and the plants tested for the presence of C-I. If a plant contains C-I whereas the endosperm contained only C, the genotypes are said to be non-correspondent. A difference between endosperm and embryo indicates that the pollen grain which produced the kernel had genetically dissimilar sperm, suggesting misdivision at the second pollen mitosis. If, on the other hand, correspondence occurs between embryo and endosperm, misdivision may have preceded the second pollen mitosis, probably occurring at meiosis.

Sixty-one percent (71/117) of the kernels with the Bz/bz endosperm phenotype contained the C-I marker in the embryo and were classified as non-correspondent. (Chromosome constitution of the C-I containing plants has not been determined.) Correspondence between endosperm and plant for the C marker was found in 39% (46/117) of the cases, indicating that meiotic misdivision may also cause fractional formation. However, crossing over can occur between isochromosome arms, producing a C C isochromosome. This chromosome could undergo centromeric breakdown at the second pollen mitosis and, nevertheless, show correspondence between endosperm and embryo. Thus, 61% is a minimum estimate of the Bz/bz fractionals generated at the second pollen mitosis.

The isochromosome may be lacking a factor which aids nondisjunction. Absence of the factor makes migration of the isochromosome to one pole difficult, and misdivision frequently occurs (Carlson: Ann. Rev. Genet., 1978). In support of this idea, Carlson (MNL, 1978) found reduced rates of nondisjunction (20%) for the B^9 isochromosome in comparison to standard B^9's (50-98%).

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Evidence on when in ontogeny mutations are induced by Mu

A mutator system (Mu) has been previously described (MCGNL 45:81-87, 1971; 49:73-79, 1975; 50:68-70, 1976; 51:32-36, 1977). One question of interest to be answered about Mu has to do with its time of action. Are mutations induced during sporophytic cell divisions? If so, do they occur early in development or late? Or are new mutations induced only in meiosis? If mutations are meiotic (occurring any time after premeiotic S), one would expect only one occurrence of any particular mutant in the selfed outcross progeny of any Mu plant. However, if mutations occurred premeiotically (occurring any time prior to premeiotic S) there could be from two to half of the offspring that would carry any given mutation. Two mutants would be expected if the mutation occurred in the last premeiotic cell cycle. A mutation that occurred very early, in a cell destined to give rise to the whole tassel, would be transmitted to half of the offspring. Mutations occurring between these extremes would result in tassel sectors carrying the mutant and thus could give rise to a situation where varying numbers of outcross progeny may carry the mutation. If mitotic mutations occur, phenotypically similar mutants would be expected in the selfed outcross progeny of Mu plants. These should all prove to be allelic. However, two mutants are not necessarily allelic just because they have the same phenotype. Many instances of phenotypically similar mutants that are not allelic are known.

In our mutator tests we frequently found more than one mutant of a given phenotype segregating on ears of sibling outcrossed plants that had been selfed. Similar mutants from given outcross progeny were allele tested. The results are given in Table 1. Seventy-eight percent of the putative allelic situations turned out not to be.

Table 1. Allele tests of mutants with similar phenotypes occurring in a given outcross progeny of Mu plants.

Number of putative allelic mutants per outcross family tested					
Two		> two (No. of different combinations tested)			
Positive (allelic)	Negative* (non-allelic)	Positive* (allelic)	Negative* (non-allelic)	Mixed (allelic+non-allelic)	
5	16	1(3)+1(3) + (5)	1(3)+1(3)+1(3)	1(7-, 1+)	
Total 5	16	3	9	7-, 1+	

Note: Total positive allele tests = 9; total negative allele tests = 32; % allelic 9/41 = 21.95.
*Negative test = < .01 probability of allelism.

Among families that had only two mutants of similar phenotype tested, 23.81% (5/21) were allelic while in families with more than two similar mutants tested 50% of the families had allelic mutants.

These results indicate that both meiotic and premeiotic mutants occur. The bulk of the mutants seem to be meiotic (or perhaps very late mitotic). The probability of a given testcross family having premeiotic mutants increases if more than two similar mutants occur. Even in instances where premeiotic mutants occur they must be relatively late events giving rise to small tassel sectors, since none

of the outcrosses to date with mutants of similar phenotypes approach the 50% mutant frequency expected if a mutation occurred in the progenitor cell of the tassel. Other tests for early mitotic mutations have also proven negative. Mu lines heterozygous for selected recessive loci have failed to show more sectoring for the recessive phenotypes than the non-Mu controls. The following recessive loci have been tested: yg2, y, c sh bz wx, a sh2 and a2 bt.

If one goes back to the raw data analyzed in Table 1, a total of 69 mutants were included in the tests. Of these, 22 were involved in positive allele tests. Thus, 68.12% of the mutants involved in potentially allelic situations proved to be non-allelic. In all estimates of Mu activity to date, the occurrence of 2 or more mutants with similar phenotypes was counted as the result of a single mutation event (counted once) in our estimates of the total number of different mutations. However, in all of our tests the total numbers of mutants (irrespective of phenotype) were also recorded. The difference between these two totals represents the mutants of similar phenotype that were not counted as resulting from an independent mutation event. Data presented above would indicate that 68.12% of these would be the result of independent mutations. Table 2 summarizes the result of all Mu tests to date.

Table 2. Mutation rate from previous Mu tests.

	No. of Plants	Total Mutants	% Total Mutants	Total different mutants	% Total different mutants
Totals	1,541	171	11.1	98	6.4
Controls	1,265	3	0.2	3	0.2
	Mutators/Controls = .064/.002 = 32.0 X				

Comparison between the mutator and control populations reveals a contingency $X^2 = 97.9355$, = p .01 = $X^2 = 6.635$

In column 6 the mutation rate is estimated as 6.4%. However, this does not reflect the true mutation rate since two or more mutants with similar phenotypes in a given outcross were counted as being the result of only one mutational event. If we take 68.12% of the difference between column 3 and column 5 (all mutants including those with similar phenotype) we will get an estimate of the putative allelic mutants that are actually non-allelic (are the result of an independent mutation), 49.73. If these are now added to the total of column 5 we have a better estimate of the number of different mutations. This new total now gives an estimated mutation rate of 9.6%, which is 3.2% more than our previous estimate and is some 48-fold higher than our best estimate of the spontaneous rate.

Donald S. Robertson and Peter N. Mascia

Ear Maps of Mu induced events

If Mu induced mutations are occurring in mitotic divisions then there should be clusters of seeds carrying phenotypically similar mutants on the ear. In order to determine if such clusters were occurring, one ear of Mu plants with two ears was pollinated by a standard line while the second ear was self-pollinated (to make sure that the Mu parent was not segregating for a mutant). Six outcross ears with good seed set were selected and 150 seeds planted in an ear map pattern. Six rows of 25 seeds were taken off the ears in order and planted in order in the field.

Missing seeds on the ear were recorded and seeds that did not germinate were noted. The selfed progeny of the plants from these seeds were scored for mutations. Of the six tests made, 3 did not segregate for any mutants while three did. The ear maps are shown in Tables 1, 2, and 3.

There are indications that there might be mutant sectors in Tables 1 and 2. In Table 1 in row 5 there appears to be a sector of two plants (4 and 5) with both yellow-green and pale-yellow mutants. The white mutant in plant six is an off-white, which may be an extreme expression of pale-yellow. If so, this would extend the sector over 3 seeds. A two-seed pale-green sector appears to be present in row 4 (positions 18 and 19). The luteus mutants in row 3, position 25 and row 5, position 21 although separated by six seeds may represent two members of a sector. Since only half the seeds in a mutant mitotic sector would be expected to carry the mutant, + seeds are not unexpected in sectors. In Table 2 there are also indications of sectors (e.g., luteus mutants in row 1, position 13 and row 2, position 15; pale-green mutants in row 3, positions 6 and 10; pale-yellow mutants in row 4, position 18 and row 6, position 19; pale-yellow mutants in row 6, positions 3, 4, 5, and 7; and yellow-green mutants in row 6, positions 19 and 22). Table 3 did not have enough mutants to provide any useful information.

Tables 1, 2, and 3. Ear maps of mutants induced by Mu.

Table 1.

Row	Seed number from butt of ear*																								
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
1	py	+	+	+	0	+	+	+	+	+	+	+	+	+	0	+	+	+	+	+	+	+	+	+	+
2	+	+	0	0	+	0	+	+	+	+	+	+	+	+	+	+	+	+	+	+	0	yg	+	+	+
3	+	+	+	+	+	+	+	+	0	0	+	0	+	+	+	+	+	+	0	+	+	+	0	+	1
4	0	0	0	+	+	+	+	+	+	+	+	+	+	0	0	+	+	pg	pg	+	+	+	+	0	+
5	+	0	+	yg py	yg py	w	+	+	+	+	+	+	+	+	+	0	+	+	+	0	1	+	+	0	+
6	0	+	+	+	+	0	+	+	+	+	+	+	+	+	+	+	0	+	+	+	+	+	+	+	+

\+ = ear - no mutants segregating

0 = no ear (seed missing or seed failed to germinate or self did not take.)

letter(s) = mutant (l = luteus, w - albino, yg = yellow-green, py = pale-yellow, pg = pale-green, ws = white stripe)

Table 2.

Row	Seed number from butt of ear																								
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
1	+	+	0	+	+	+	+	+	0	0	+	+	l	+	+	+	0	+	+	+	+	0	+	+	+
2	0	+	+	+	+	+	+	+	+	+	0	+	0	+	l	0	0	0	+	+	0	+	l	+	0
3	+	+	+	0	+	pg	0	0	+	pg	+	+	0	+	+	+	+	0	0	+	0	0	0	0	0
4	l	0	+	+	+	0	+	+	0	+	+	0	+	+	0	0	+	py	0	+	+	+	+	0	pg
5	+	+	0	+	0	0	0	0	+	0	+	+	+	0	ws	0	+	0	+	0	0	+	+	0	+
6	+	0	py	pg py	py	0	l py	+	+	+	0	+	0	l	0	0	+	+	yg py	+	0	yg	+	0	0

Table 3.

Row	Seed number from butt of ear																								
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
1	+	+	+	0	+	+	0	+	+	+	0	+	+	+	+	+	+	+	0	+	+	+	+	+	+
2	+	+	+	+	+	+	0	+	+	+	+	+	+	+	+	+	+	+	+	0	+	+	+	1	+
3	+	+	+	+	+	+	+	+	0	+	+	+	+	+	0	0	+	+	0	0	+	+	0	+	+
4	+	0	+	+	+	+	+	+	0	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
5	0	+	+	+	+	0	+	+	+	+	+	+	+	+	0	0	+	+	+	+	+	+	pg	+	+
6	+	0	+	+	+	+	0	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	0

Certainly there are strong indications of sectors. If there are indeed sectors occurring, the mutants making up a sector should be allelic. This prediction will be tested next summer.

Donald S. Robertson

Female transmission of Mu

So far all reported tests for the presence of mutator have involved the crossing of Mu stocks as males. To test for the presence of Mu in a Mu line 50 outcross plants are grown and self-pollinated and the progeny of the selfed ears scored for the presence of seedling mutants. In tests made so far almost all outcross progeny of Mu plants carry Mu and transmit it to all of their outcross progeny. Only rare exceptions to this non-Mendelian transmission have been observed. Out of 130 outcross plants scored for the presence of Mu only 16 (12.3%) gave negative tests, suggesting the loss of Mu. Some of these have proven to be the result of false negative tests due to sampling error (see MGCNL 51:36, 1977). The true loss rate of Mu is probably less than 10%.

With the development of Mu lines that consistently produce two ears, it has been possible to test for the presence of induced mutation in outcross progeny when Mu plants are crossed as females. The results of such tests are shown in Table 1.

Thirty percent of the crosses (3 out of 10) did not segregate for any mutations (have lost Mu?) compared to about 10% of the crosses where Mu plants are used as males. However, the female sample is much too small for any firm estimates of loss rate at this time.

If the families which did not show any mutations are eliminated the resulting mutation rate is close to that observed for other Mu crosses. Thus it appears that Mu induced mutations occur in the female inflorescence as well as the male.

Table 1. Mutation rate observed in outcross progeny when Mu plants are crossed as females.

Outcross	Total	Total mutants	% mutants	Total different mutants	% different mutants
7011	44	4	9.09	3	6.82
7012	40	7	17.50	2	5.00
7013	37	1	2.70	1	2.70
7014	45	8	17.78	4	8.89
7064-66	96	19	19.79	9	9.38
7066-69	107	0		0	
7070-72	118	0		0	
7073-75	127	11	8.66	8	6.30
7076-78	128	0		0	
7079-81	129	2	1.55	2	1.55
Total (T)	871	52	5.97	29	3.33
T - 0-mutation families	518	52	10.04	29	5.60

Donald S. Robertson

Distribution of Mu induced mutants

One thing we would like to know about the action of the mutator system we have been studying is its specificity. Is it a general mutator affecting all loci equally or is it locus (loci) specific or specific to a certain chromosome or chromosome segment? In the latter case there may be a select group of loci that respond, confined to one or a few chromosome segments, while the great bulk of the genome would remain insensitive. In an attempt to answer these questions we have systematically tested our mutants with a set of A-B translocations that would permit us to locate many of them to chromosome arm. If we are dealing with a general mutator the mutants should be distributed among the chromosome segments tested. Mutator specificity would be expected if all the mutants occurred in one or a few of the tested segments. The A-B translocations listed below were used in these tests.

Translocation	Breakpoint	No. of mutants uncovered
1a	L.2	
1b	S.05	
3a, 2S (6270)	3L.1-.60, 2S.46	2
1b, 2L (4464)	1S.05-.53, 2L.28	
3a	L.1	1
3b	S ?	1
4a	S.25	1
9b, 4L (6222)	9S.40-.68, 4L.03	2
1a, 5S (8041)	1L.2-.8 5S.15	
5a	L?	3
6c	L?	3
7h	L.3	1
8a	L.7	
9b	S.4	
9c	L?	2
10c	S?	1
10-19	L?	2
	Total	19

The TB parents were known hyperploids except for TB-8a and TB-9c. Pollen samples from families of these two A-B translocations were examined and plants with 15-25% abortive pollen were selected for outcrossing. Each was also crossed to an appropriate tester (i.e., v*-A552 for TB-8a and bf for TB-9c). Most of the mutants were in families in which 2/3 of the plants would be expected to be heterozygous. An attempt was made to pollinate three plants from each mutant family with pollen from each A-B translocation. This was not always possible because of poor germination or weak plants.

Nineteen mutants have been located to date, as listed above. So far the distribution of mutants appears to be random. It is a little surprising that no genes have been located in chromosome one as yet since long chromosome segments are involved. However, the sample of mutants is not sufficiently large at this time to permit one to say that any particular chromosome is insensitive to Mu activity.

Four white-endosperm albino mutants that were found in Mu stocks were tested against all known white albinos for allelism. Two turned out to be allelic to lw (long arm of chromosome 1), one was allelic to w3 (long arm of chromosome 2) and the fourth was allelic to y7(vp9) (long arm of chromosome 7). One blue fluorescent mutant was allelic to bf2 (long arm of chromosome 10).

When the allele test results are considered along with those of the A-B crosses the long arm of chromosome one, the short arm of chromosome 10 and the long arm of chromosome 2 have been added to the list of chromosome arms in which mutations have been induced.

The pattern of distribution of induced mutants, at this time, supports a model of a general mutator as opposed to a more specific mutator.

Donald S. Robertson

Luteus alleles at the oil yellow (oy) locus

Two luteus alleles, oy-1040 and oy-1039 (formerly 1*-1040 and 1*-1039), derived from D. S. Robertson's mutator line, are lethal and severely defective in the conversion of protoporphyrin IX (proto) to Mg-protoporphyrin (Mg-proto) (Mascia & Robertson, MGCNL 51:38-42, 1977). This is in contrast with the observation that oy is a viable lesion. In an attempt to determine the relationships among these alleles the porphyrins accumulated by these mutants and various combinations have been analyzed.

Dark grown plants are normally blocked in the conversion of protochlorophyllide (pchlide) to chlorophyllide (chlide). The pathway is further inhibited by a feedback mechanism preventing δ-aminolevulinic acid (ALA) synthesis. Under these conditions normal plants accumulate a small amount of pchlide. Table 1 indicates the pchlide accumulated by oy alleles. Two observations can be made concerning these data. Firstly, oy-1040 is the most stringently blocked allele accumulating no detectable porphyrin pigments, while oy-1039 accumulates approximately 40% of the normal level of pchlide.

Table 1. Pigments accumulated by unfed oy alleles.

Allele	nmoles/gm* Pchlide	SE	% of normal
oy-1040	0	0	0
oy-1039	7	.57	40
control	17.7	1.2	100
oy-1040/oy-1039	6.9	.42	38
control	18.0	1.0	100
oy/oy	15.4	1.6	100
oy/oy-1040	18.0	1.2	100
control	17.3	1.8	100
oy/oy-1039	13.7	1.1	100
control	12.9	1.9	100

*Pigments were quantitated throughout this paper using the mM extinction coefficients of Kahn et al., 1976 in The Genetics and Biogenesis of Chloroplasts and Mitochondria ed. The Buchler and P. Slonimsky, Amsterdam: Elsevier/North Holland Biomedical Press. pp. 119-131.

Heterozygous oy-1040/oy-1039 seedlings accumulate the same concentration of pchlide as the leaky plants. Since these alleles were derived from closely related sources of the mutator line, the possibility that they arose from a single mutation event with a modifier gene in the leaky stock had to be examined. This can be tested as illustrated in Table 2. If the leakiness of the oy-1039 stock were due to an independent modifier gene, selfs of all of the above combinations would give rise to a 3:1 segregation of leaky to nonleaky seedlings among mutants. Results obtained were consistent with the conclusion that the oy-1039 allele itself is leaky. Secondly, oy and combinations of oy with oy-1040 and oy-1039 were at least equivalent to normal with respect to pchlide synthesis.

Table 2. Tests for an oy modifier.

Source	Results expected if leakiness is a characteristic of oy-1039
$\frac{+\text{-}1039}{+\text{-}1039} \times \frac{oy\text{-}1040}{+\text{-}1040}$	$F_1 \xrightarrow{\otimes}$ 1/2 green, 1/2 seg. nonleaky
$\frac{oy\text{-}1039}{+\text{-}1039} \times \frac{oy\text{-}1040}{+\text{-}1040}$	normal $F_1 \xrightarrow{\otimes}$ 1/3 green, 1/3 seg. leaky, 1/3 seg. nonleaky
$\frac{oy\text{-}1039}{+\text{-}1039} \times \frac{+\text{-}1040}{+\text{-}1040}$	$F_1 \xrightarrow{\otimes}$ 1/2 green, 1/2 seg. leaky

When dark grown plants are fed ALA the regulatory step in chlorophyll biosynthesis is bypassed and significant increases in precursors are accumulated. The actual amount of pigments accumulated by these plants is extremely variable due mainly to variation in uptake of ALA (Table 3). However the proportions of the pigments accumulated are fairly consistent (Figure 1). In these experiments, normal plants accumulated a mixture of approximately 40% proto and 60% pchlide (Figure 1, control values A-D). The most stringently blocked allele, oy-1040 (Figure 1A) accumulates almost exclusively protoporphyrin IX, however, a trace of pchlide may be present in these seedlings. Although slightly leaky, oy-1039 (Figure 1B) accumulates predominantly proto (90%) and only 10% pchlide. It is interesting to note that this mutant does not accumulate significantly more pchlide when fed ALA than when harvested unfed. Homozygous oy plants (Figure 1E) are similar to normal with respect to their precursor accumulation. Heterozygotes between oy and the lethal mutants (Fig. 1C & D) are intermediate in phenotype, accumulating approximately 65% proto and 35% pchlide. The oy allele thus appears to be semi-dominant to the lethal alleles.

Table 3. Pigments accumulated by recessive oy mutants fed ALA.

Mutant	nmoles/gm Proto 1X	SE	Pchlide	SE	Total	SE
oy-1040	116	34	(trace)		116	34
control	70	39	108	35	178	74
oy-1039	81	22	9.3	2.5	91	23
control	55	23	68	26	124	45
oy/oy	66	26	107	41	173	63
oy/oy-1040	177	35	94	6	271	40
+/oy	77	18	126	36	204	52
oy/oy-1039	137	73	71	21	208	92
+/oy	71	14	138	41	209	52

It is concluded from these data that the primary function of the oy^+ allele is in the conversion of proto to Mg-proto. The two lethal loci reported here, oy-1040

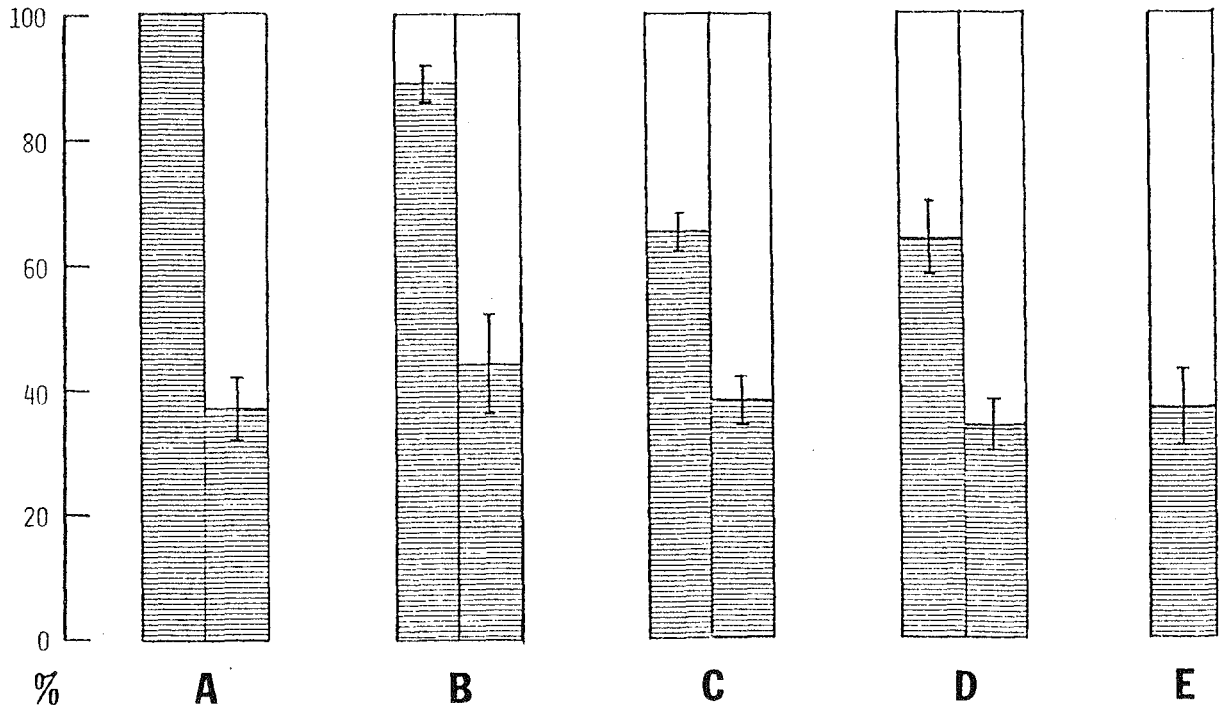

Figure 1. Relative proportions of proto and pchlide accumulated by dark grown oy alleles fed ALA for 24 hrs. A normal control is illustrated at the right of each mutant except oy which was analyzed in a homozygous stock.

A oy-1040
B oy-1039
C oy/oy-1040
D oy/oy-1039
E oy/oy

and oy-1039, are severely defective in conversion of proto to Mg proto while oy is sufficiently leaky not to appear defective in this conversion at all. The defect is, however, observed in the plants heterozygous for oy and either of the lethal alleles where one dose of the oy allele is not sufficient to allow normal synthesis of pchlide in dark grown plants fed ALA. Such seedlings, although appearing somewhat green in a seedling bench, are lethal. Several dominant visible, recessive seedling lethal oy alleles, Oy-yg-700 and Oy-yg-1459 have recently been obtained from Dr. Neuffer. Unfortunately, Oy-yg-1459 is in a heterozygous stock and homozygous Oy-yg-700 seeds have failed to germinate. Failure to germinate is also a characteristic of oy-1040 particularly in old seed. It is not unreasonable to speculate that these dominant alleles are also severely blocked in the conversion of proto to Mg-proto. Confirmation of this awaits further testing.

Peter Mascia

Mutants defective in conversion of Mg protoporphyrin monomethyl ester to protochlorophyllide

The orobanche locus (oro), phenotypically brownish and very necrotic, is defective in conversion of Mg-protoporphyrin monomethyl ester (Mg-proto me) to protochlorophyllide (pchlide). Last spring a number of additional oro mutants, apparently uncharacterized genetically, were obtained from Maize Genetics Cooperation. In order to clarify this situation allele tests of these mutants (excluding oro-1-6577) were carried out. On the basis of their superficial phenotypic resemblance, sienna and l*-4923 were also included in this test. The following results were obtained:

oro = oro-4889 = oro-8081 = oro-6474 = oro-64-4589-4 ≠ oro2 ≠ sienna ≠ l*-4923

Beyond these preliminary observations, more extensive work has been carried out only on oro and oro2. A dominant modifier of oro, Orom, has also been investigated.

Light grown, both oro and oro2 are very necrotic. Tissue that does develop appears to bleach in a manner corresponding to the light, dark cycle. No porphyrin pigments are detected in such plants:

Material	Chlorophyll Mg/gm*	% of normal
control	1.51 ±.06	100
oro	0	0
oro2	0	0
oro:Orom	.073 ±.004	4.83

*Formula of D. I. Arnon (1949) Plant Physiol. 24:1-5 was used.

It is likely that pigments produced by oro and oro2 are immediately photo-oxidized, resulting in cellular disruption and necrosis.

Dark grown, such plants develop perfectly normal leaf tissue and accumulate a small amount of Mg-proto me corresponding approximately to the amount of protochlorophyllide accumulated by normal plants:

Mutant	Pigment:nmoles/gm* Mg-proto-monomethyl ester	SE	Pchlide	SE	% of normal pchlide
oro	31.7	1.1	0	0	0
control	0	0	23.9	5.4	100
oro2	8.8	2.1	0	0	0
control	0	0	12	3	100
oro:Orom	0	0	16.9	3.6	124
control	0	0	13.8	2.1	100

*nM extinction coefficients of Kahn et al., (1976) in: Genetics and Biogenesis of Chloroplasts and Mitochondria, Amsterdam: Elsevier/North Holland Biomedical Press, pp. 119-131, were used throughout this report to quantitate precursors.

This is in contrast with the observation that completely blocked mutants defective in the conversion of protoporphyrin-IX (proto) to Mg-proto accumulate no detectable

proto. It would appear that Mg-proto me and pchlide are not as important in regulating the chlorophyll biosynthetic pathway as proto.

When these mutants, dark-grown, are fed δ-aminolevulinic acid (ALA) for 24 hr, a mixture of pigments, including proto, Mg-proto and their monomethyl esters, is accumulated. The protoporphyrins are spectrally indistinguishable from their monomethyl ester so for purposes of calculation they are treated as units. The total amount of pigments accumulated by these plants is extremely variable due mainly to variation in uptake of ALA:

Mutant	nmoles/gm proto's	SE	Mg proto's	SE	Pchlide	SE	Total	SE
oro	76	30	90	27	0	0	166	55
control	89	34	0	0	123	20	212	42
oro2	50	5	48	4	0	0	98	8
control	54	14	0	0	65	18	119	27
oro:Orom	312	140	159	81	41	20	513	240
control	55	18	0	0	103	31	158	45

However, the relative proportions of proto's and Mg-proto's are approximately equal (Figure 1A & B).

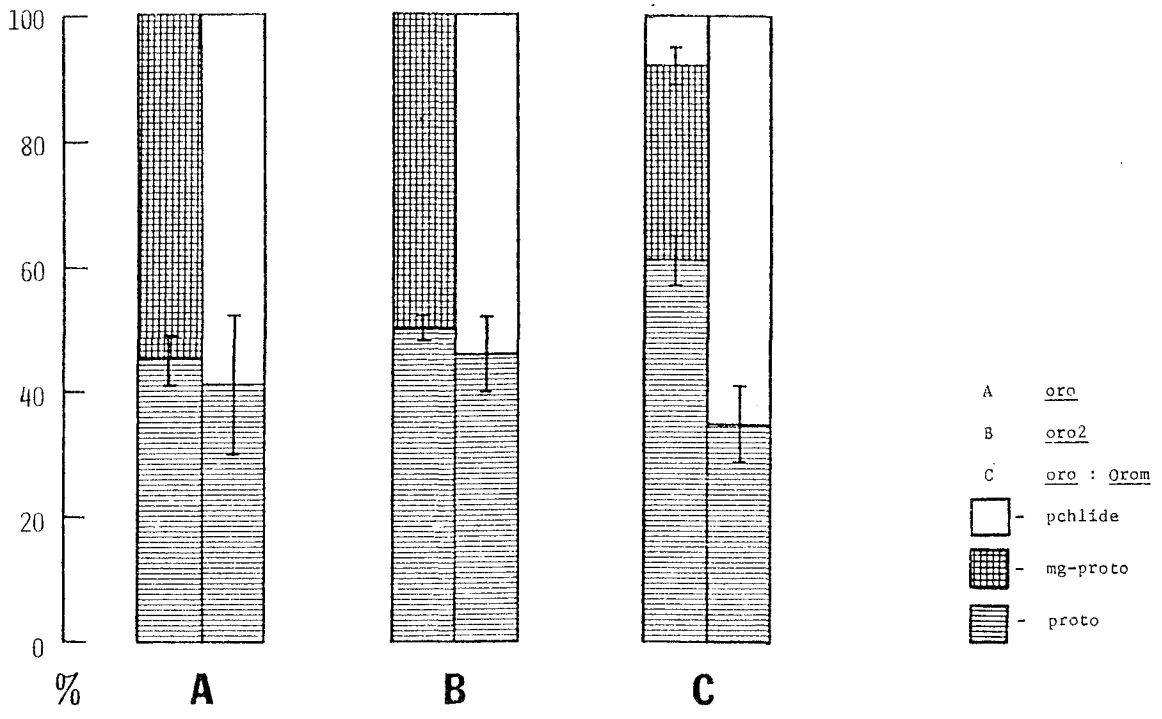

Figure 1. Relative proportions of precursors accumulated by orobanche plants. A control is illustrated at the right of each mutant.

Two possible explanations for the fact that more than one locus is involved in the same step are envisioned. Firstly, since there are actually several reactions involved in the conversion of Mg-proto me to pchlide these mutants may represent defects in subunits or components of a complex that normally carries out this conversion. Secondly, reactions after the formation of proto are carried out in the chloroplast membranes. Specific disruption of the membrane where enzymes or pigments normally bind might result in the same phenotypic expression.

All mutant alleles at the oro locus, involved in the allele tests reported here, are influenced in a similar manner by the modifier gene, Orom. This modifier acts in a dominant fashion and is genetically independent of oro:

Test:	seedlings counted green	modified	necrotic	χ^2	P
$\frac{oro}{+}:\frac{Orom}{orom} \xrightarrow{\otimes}$	534	120	48	1.56(12:3:1)	.5>P>.3
$\frac{oro}{+}:\frac{Orom}{orom} \times \frac{oro}{+}:\frac{orom}{orom}^{*} \longrightarrow$	218	26	37	2.73(6:1:1)	.3>P>.2

*The unmodified oro sources represent a total of seedlings counted from crosses of the various oro alleles to oro/+ : Orom/orom plants used in allele tests.

In the presence of Orom, oro plants bypass the lesion to some extent and produce some chlorophyll in light (first table). Nearly normal leaf development is observed in these plants and a luteus phenotype is typical in greenhouse material. Unfed dark grown material accumulates at least a normal level of pchlide (second table); however, when fed ALA modified oro seedlings accumulate a mixture of pigments including pchlide as well as the pigments accumulated by oro (third table, Figure 1C). The concentration of porphyrins in oro:Orom plants is extremely high compared to controls. The significance of this is unclear. These data suggest that Orom is not integrated with the primary chlorophyll biosynthetic system but may be involved in an alternative pathway. It does not appear that normal plants are affected by Orom. 75% of the normal plants in this experiment should have contained the modifier, however, these control values are similar to those of other experiments. The effect of the modifier on normal plants and the extent of its presence in normal stocks has not been systematically studied. It will be interesting to determine the effect of Orom on oro2 and normal plants. (Thanks to Dr. Lambert for providing adequate seed for the allele tests reported).

Peter Mascia

Genetic studies of luteus mutants

Mutants at three luteus loci are defective in the conversion of protoporphyrin IX to Mg-protoporphyrin. These mutants have been localized to chromosome arms by crosses with TB testers. The l*-Blandy4 locus is uncovered by TB-1La, 5S8041 but not by TB-1a in limited tests, therefore, it seems likely that this gene is located on the short arm of chromosome 5. The mutants l*-1040 and l*-1039 were located on the short arm of chromosome 10 with TB-10c. These are allelic to oy and have been renamed oy-1040 and oy-1039. The l*-Neuffer2 (E-59) locus is uncovered on 10L by TB-10(19). A four point testcross which localizes this gene between R and sr2 is presented in Table 1 (using the modified testcross system of D. S. Robertson, 1955, Genetics 40:746-760).

Table 1. Localization of 1*-neuffer2 (E-59) on chromosome 10.

F_1 genotype $\frac{g\ R\ +\ sr2}{+\ r\ 1\ +}$

Genotype	Region	Number
$\frac{g\ R\ +\ sr2}{+\ r\ 1\ +}$	parental	85 122
$\frac{g\ r\ 1\ +}{+\ R\ +\ sr2}$	1	17 33
$\frac{g\ R\ 1\ +}{+\ r\ +\ sr2}$	2	55 57
$\frac{g\ R\ +\ +}{+\ r\ 1\ sr2}$	3	10 3
$\frac{g\ r\ +\ sr2}{+\ R\ 1\ +}$	1-2	0 2
$\frac{g\ R\ 1\ sr2}{+\ r\ +\ +}$	2-3	3 2
$\frac{g\ r\ 1\ sr2}{+\ R\ +\ +}$	1-3	2 1
	Total	392

Region	Total recombination frequencies*	SE
1	.1397	.0174
2	.3016	.0231
3	.0454	.0105

*Phillips (1969) Gen. 61:107-116

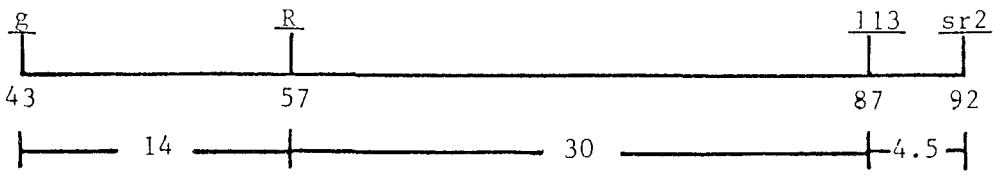

Figure 1: Genetic map of the long arm of chromosome 10

These data place the gene at 87 on the chromosome 10 linkage map (Figure 1). Since no stock of l2 is available and no other luteus mutants are located on chromosome 10L this mutant has been designated l13.

The advice and helpful discussions of D. S. Robertson in the preceding studies are gratefully acknowledged.

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Leaf-burning in Ia5125, a monogenic trait bearing on C3-C4 photosynthesis

Several days of high temperatures (ca. 100 F) in early July in Massachusetts accentuated the leaf-burning and stunting that tends to occur in the sweet corn inbred, Iowa5125. Three self-pollinated families of recovered derivatives of 5125 had F_2 ratios totaling 72 non-burn to 21 burn type plants and one sib-pollinated family had 19 non-burn to 18 burn plants. These good fits to the 3:1 and 1:1 Mendelian ratios clearly indicate that a single gene, hereby designated bu (burn), is involved. Seed from self-pollinations on a non-burn family is available as an improved form of 5125, hereafter to be designated as MA5125.

Sometimes leaf-burning in 5125 will occur in horizontal bands on the leaf, apparently corresponding to high day and low night temperatures. Evidence supporting temperature as a factor in leaf burning and area of starch deposition came from experiments in growth chambers, although no effect was found on "Krantz anatomy." Cross-sections in the green areas of burn type leaves that were stained with I-KI solution showed that, contrary to normal leaves, they failed to store starch in the bundle sheath plastids and, instead, starch was stored in the mesophyll plastids. There are a number of possible explanations, one of which is that the 5125 burn type plants have difficulty in the transfer of photosynthate from the mesophyll to the bundle sheath where the decarboxylation occurs. As the photosynthate

Table I: Corn and its Relatives having "Krantz-type" Leaf Anatomy.

Plant Examined	Number of Specimens
1. Gaspé Flint corn	8
2. Confite morocho corn	6
3. Confite puneño corn	14
4. Sweet corn inbred MA5125: Bu Bu	3
Ma5125 x 5125: Bu bu	1
Burn 5125: bu bu	5*
5. Coix lachryma - Jobi	4
6. Perennial teosinte	1
7. Jutiapa teosinte	1
8. San Antonio Huixta teosinte	1
9. Tripsacum 111.4n x Jutiapa teosinte	1
10. Tripsacum floridanum - Dade County	1
11. T. floridanum - Gate 8B, ENP	1
12. T. dactyloides (both 2n and 4n, 9 sources)	9
13. T. maizar	1
14. T. zopilotense	1
15. T. maizar x T. zopilotense	1
16. Seedling 111. 3N; 2 Tr; 1N genomes	1
17. Corn x T. dactyloides (Amphidiploid hybrid)	3
18. Elyonurus tripsacoides (3 sources)	7
19. Coelorachis ramosa	2
20. Manisuris cylindrica (2 sources)	8
21. M. rugosa	1
22. M. selloana	2
23. M. tesselata	1

*Although this burn type of 5125 had Krantz-type leaf anatomy, there was no starch stored in the bundle sheath.

backs up in the mesophyll of 5125, it seems to be converted and temporarily stored as starch in this area such as under the C3 system of photosynthesis at higher temperatures. Thus, the burn (bu) mutant gene may involve the loading and transfer system that is most efficient in plants with C4 anatomy.

While 5125 is less inclined to burn at lower temperatures, there is no evidence that it grows better at lower temperatures than other sweet corn inbreds.

The various races of corn and all of its known relatives that we have examined have only the "Krantz-type" of leaf anatomy that characterizes the C4 type of photosynthesis, as is summarized in Table I.

The derivatives of inbred 5125 showing the burn and non-burn phenotypes were examined cytologically. The meiotic behavior of the non-burn plants was normal. In contrast, about 10% of the cells from burn type plants showed meiotic irregularities such as asynapsis, fragmented chromosomes, mis-division of univalents, random distribution of univalents, etc. Examination of the pollen revealed about 4 to 6% abortion.

The C4 system of photosynthesis has evolved over the millenia in grasses adapted to high temperature conditions. A C3 photosynthesis type of corn might be better able to cope with the low temperatures that occur in the early part of the growing season or at high altitudes and latitudes.

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The evolution of string cob corn

The "string cob" trait that characterizes the popcorn race Confite morocho from the Ayacucho area of Peru has found practical utilization in the sweet corn hybrid, Candy Stick. Its natural selective values in Peru have not been explained.

It appears that the slender cob and long pointed kernels of Confite morocho are adaptations in this race for rapid ear-drying under humid conditions where thick cobs bearing soft compacted kernels are prone to mold. In maize that was grown in the northern Andes at a level where humid conditions develop through changes in elevation (3000 to 4000 m on eastern slopes), selection for viable storage capacity of the seed would favor both the string cob trait and long pointed kernels. The latter are loosely packed by virtue of their shape. Pointed kernels as well as thick pericarps may also occur at the opposite extreme such as with the thick compacted (fasciated) cobs of Polomero Toluqueno related races in order to facilitate drying and reduce pericarp splitting.

The archaeological evidence from the Ayacucho area of Peru with cobs collected by R. S. MacNeish is that the first corn to reach there (4000 B.C.) was basically similar to the Pollo-Nal Tel cobs tracing back to Mexico. They have a thicker, more condensed rachis than the type of Confite morocho that became adapted to the Andes.

There are those who maintain that Confite morocho is derived directly out of a hypothetical wild maize indigenous to Peru (Grobman, et al., 1961; Mangelsdorf, 1974). However, the string cob trait of Confite morocho may be a specialized condition, not a primitive one. That is, the string cob condition does not necessarily stem directly from primitiveness. It may have been reactivated or reintroduced as selective values changed when maize spread into the humid parts of the Andes. A similar type of slender cob corn became adapted to the Sikkim area of the Himalayas, possibly for the same reasons. This Sikkim corn was also once suggested as a relict primitive type (Stonor and Anderson, 1949). In a less extreme example, with the advent of the selection and drying of seed ears, as well as hybridization with the northern flints, the thick, many-rowed ears of dent corn from the Southeast moved into the Northeast and the Corn Belt.

Walton C. Galinat

The breeding of two-rowed (airplane) sweet corn

The occasional appearance of a phenotypically unstable type of four-rowed corn in normally eight-rowed corn is commonly observed by corn breeders. This condition results from two ranks of paired spikelets. The phenotype is hereby designated as two-squared corn. Its expression may be stabilized by the incorporation of a portion of Tripsacum chromosome 9 (Tr9) in an interchange with corn chromosome 2.

Four-rowed ears of corn may also be produced by having the gene for single female spikelets (pd) in a background of eight-rowed corn. In this case the four-rowed condition results from four ranks of single spikelets. This phenotype is hereby designated as four-squared corn.

The recombinant phenotype of two-ranks of single female spikelets provides a two-rowed ear of corn. Because the little flat ears of this phenotype will lie on an airline eating tray without rolling off into your lap, this combination is hereby designated as airplane corn. Work is underway to develop sugary-shrunken and sugary brittle hybrids of airplane corn.

Walton C. Galinat

More on pericarp and aleurone thickness in maize and its relatives

The pericarp of maize and its relatives was found to range from 2 to 20 cell layers and 25 to 200 microns. A number of crosses were made between thick and thin pericarp inbreds of maize. Both of these types were also crossed to teosinte, which has an extremely thin pericarp measuring 25 microns. The F_1 and F_2 progenies of these crosses were examined.

The method used to measure the pericarp is simple:

1) Select mature kernels from the middle of the ear.
2) Soak kernels for 24 hours.
3) Using a single-edged razor, remove the crown of the kernel.
4) Take free hand sections on the abgerminal side of the kernel.
5) Stain sections with dilute safranine.
6) Mount sections in glycerine.
7) Measure sections under high power (250X) using an ocular micrometer.
8) Take an average of 3 readings per kernel and 3 kernels per ear.

The relatives of maize have the thinnest pericarp (Table II). There was no selective pressure to evolve a tough protective pericarp in the presence of their hard fruit cases. A sample of sweet corn types shows a wide range, from the thin pericarp inbred 677a at 57 microns, to Spancross, a tough hybrid 140 microns thick. The thickest pericarp observed was found in Redenbachers Gourmet Popping Corn at 180 microns.

The inheritance of popcorn pericarp is believed to be controlled by one dominant gene for thinness and many modifying genes for thickness. The results of the sweet corn-teosinte crosses seem to agree with this observation (Table III). Here the F_1 pericarp is nearly the same thickness as that of the teosinte parent. The F_1 of the different crosses all have nearly the same pericarp thickness independent of the thickness in the sweet corn parents. Thin pericarp, a feature seen in teosinte, is dominant as expected for a wild type trait.

A number of popcorn varieties were examined for aleurone thickness as well as pericarp thickness. Six popcorn varieties were examined, two of which appear in Table II. All of the varieties examined were found to have a very thin aleurone layer about the same size as that of teosinte, and at most, one-half the size of sweet corn aleurone. The popcorns, with their very thick pericarp, have small

Table II. Pericarp and aleurone thickness.

Source	Pericarp		Aleurone	
	No. of Cell Layers	Thickness in Microns	No. of Cell Layers	Thickness in Microns
teosinte	2-3	25	1	28
Tripsacum	2-3	30	1	-
Coix	2-3	30	1	-
677a-867	4-6	57	1	60
Hayes white	5-6	61	1	52
Tender treat	7-8	55	1	74
Iosweet	6-7	60	1	63
C5-NT	10-11	120	1	-
C13-1	12-13	140	1	60
Spancross	12-13	140	1	70
Argentine Pop	9-10	96	1	31
Redenbachers Gourmet Pop	17-18	180	1	28

Table III. Thickness of F_1 pericarp and parental pericarp of various sweet corn-teosinte crosses.

Source	No. of Cell Layers	Thickness in Microns
Teosinte	2-3	25
(Hayes White x Teosinte) S1	3-5	30
Hayes White	5-6	61
(P51-TB x Teosinte) S1	3-5	33
P51-TB	9-10	80
(C13-1 x Teosinte) S1	7-8	35
C13-1	12-13	140

aleurone cells and teosinte, with its protective fruit case, also has small aleurone cells. But in the sweet corns, while man's selection has made the pericarp thinner, the aleurone cells have become larger. This would seem to indicate that thick aleurone compensates to some degree for thin pericarp in forming a protective layer.

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Further analysis of duplication strands with a duplicate segment in a displaced position on the long arm of chromosome 10

We have previously described the isolation and identification of abnormal 10's carrying a duplicate segment in a displaced position (Dp 10-1, -2, -3, -4). Presence of the duplication is inferred from the decrease of recombination values in regions adjacent to the duplication, the abnormal morphology of homozygous Dp10/Dp10 plants and the association of these effects with a gene marker carried on the duplicate segment (the gene component of the R locus, designated P, controlling anthocyanin synthesis in the plant tissues). Linkage studies had shown that duplications Dp10-1, -2, -3 and -4 lie on the long arm of chromosome 10 and in one case (Dp10-1) the cytological observations indicate that the duplicate segment is terminal. The data to be presented refer to additional linkage tests that were performed in order to get more precise information on the physical position of these duplications.

The genotype of Dp10/N10 heterozygotes used to locate Dp 10-1 in relation to R and o7 was R-st +/r-g o7, with the R-st strand carrying the duplication, while in the analysis of Dp10-3 and -4 R-sk +/r-g o7 heterozygotes were used.

The following distribution of seed phenotypes was observed in the progeny of these testcrosses:

	Colorless		Nonparentals	Variegated[a]		Nonparental
Duplication	o+	o7	%	o+	o7	%
Dp 10-1	124	2441	4.83	2056	8	0.39
Dp 10-3	302	2509	10.74	3387	157	4.43
Dp 10-3[b]	144	423	25.39	468	108	18.75
Dp 10-4	70	2919	2.34	3168	6	0.19

[a]Stippled or smoky

[b]These data refer to the testcrossed progeny of plants, originally in a Dp 10-3/N10 family, that lost the duplicate segment

Contrary to expectations, the recovery of the two classes of nonparentals is not equal, the frequency of colorless nonopaque kernels far exceeding that of the stippled opaque ones.

This difference is not likely to be accounted for by the difficulty of detecting the opaque phenotype among the stippled or smoky kernels as proved by the fact that seeds of the latter phenotype are recovered in high frequency in the progeny of heterozygous parents that lost the duplication (see third row in the above table).

Furthermore, progeny tests of both types of nonparentals, even though still incomplete, confirm the validity of the screening method as the data below show:

Nonparental endosperm phenotype	Duplication	Nonparentals Tested	Nonparentals Verified	Nonparentals %
colorless nonopaque	Dp 10-1	61	44	72.13
colorless nonopaque	Dp 10-3	99	99	100.00
colorless nonopaque	Dp 10-4	9	4	44.44
stippled opaque	Dp 10-3	12	12	100.00

In the presence of Dp 10-4 phenotypic classification proved to be unreliable. The basis for such a difference in recovery of the two classes of nonparentals remains at the moment unexplained.

Presence of the P marker on the duplicate segment allows the detection of whether the yield of nonparental strands from R-st Dp10 + / r-g N10 o7 parents is associated with loss of the Dp segment.

Data referring to this test, as obtained after progeny tests of presumed recombinants, have been obtained for one kind of nonparentals (colorless nonopaque aleurone) and for Dp 10-1 and -3 only. They are summarized here:

Duplication	n	r-g o^+ P	r-g o^+ p
Dp 10-1	44	42	2
Dp 10-3	56	45	11

These data together with the cytological evidence previously mentioned indicate that Dp 10-1 is most likely distal to o7. The results for Dp 10-3 are best interpreted by assuming the duplication to be proximal to o7; if it were distal about 20% (11/56) of the r-g o^+ recombinant strands should be double crossovers, an unlikely event in view of the positive interference known to occur in the long arm of chromosome 10.

G. Gavazzi and M. Sandri

Differential effect of benzyladenine (BA) on P_{Dp} and P gene expression

One dose of P carried on the displaced duplication (Dp 10-1) conditions a significantly lower pigment content in sporophytic tissues than a P gene component in its normal position. It has been suggested that position effect phenomena might account for this difference. The difference, however, is highly reduced or completely suppressed if the seedlings are grown on a medium supplemented with BA, as shown in Table 1. Seedlings were grown on a 2.5% sucrose solution solidified with 9% agar under continuous light and 21 C $\pm$ 2. BA concentration is 1×10^{-6} M. Pigment extraction in roots (75 mm sections) and first internodes (20 mm sections) was done on 11 and 15 day old seedlings respectively.

Table. 1 Anthocyanin content (A530) of seedling first internodes (a) and primary roots (b) in presence of one dose of P either in its normal position or on the displaced Dp 10-1 duplication.

Seedling genotype	Treatment	n	A530 (a)	A530 (b)
$\frac{p\ S\text{-}st}{P\ \ s}$	null	8	.493	.597
"	BA	8	.712	.868
$\frac{p\ S\text{-}st}{p\ \ s}$ P(Dp)	null	8	.267	.316
"	BA	8	.817	.665

Even though BA enhances the pigment content in the plant tissues of both genotypes, the response of P Dp is more pronounced and, at least in the internode, it leads to the same amount of pigment conditioned by P.

These results indicate that a displaced P is capable, under the proper conditions, of reaching the same level of gene expression conditioned by a normal P gene component. This observation attaches more credit to the view that position effect phenomena account for the low pigment level conditioned by P Dp.

G. Gavazzi and M. Sandri

Studies of callus tissue: growth behavior of three lines of maize

In this report I describe the growth behavior of callus of different origins. Sources of the callus were the inbred A 188 and two stocks, one homozygous for sh wx gl15 v and the other for yg, kindly provided by the Genetics Co-op. They will be referred to as S 1106 and S 1104 respectively. They have been chosen because of their tendency to form callus from the scutellum of mature seeds germinated on agar.

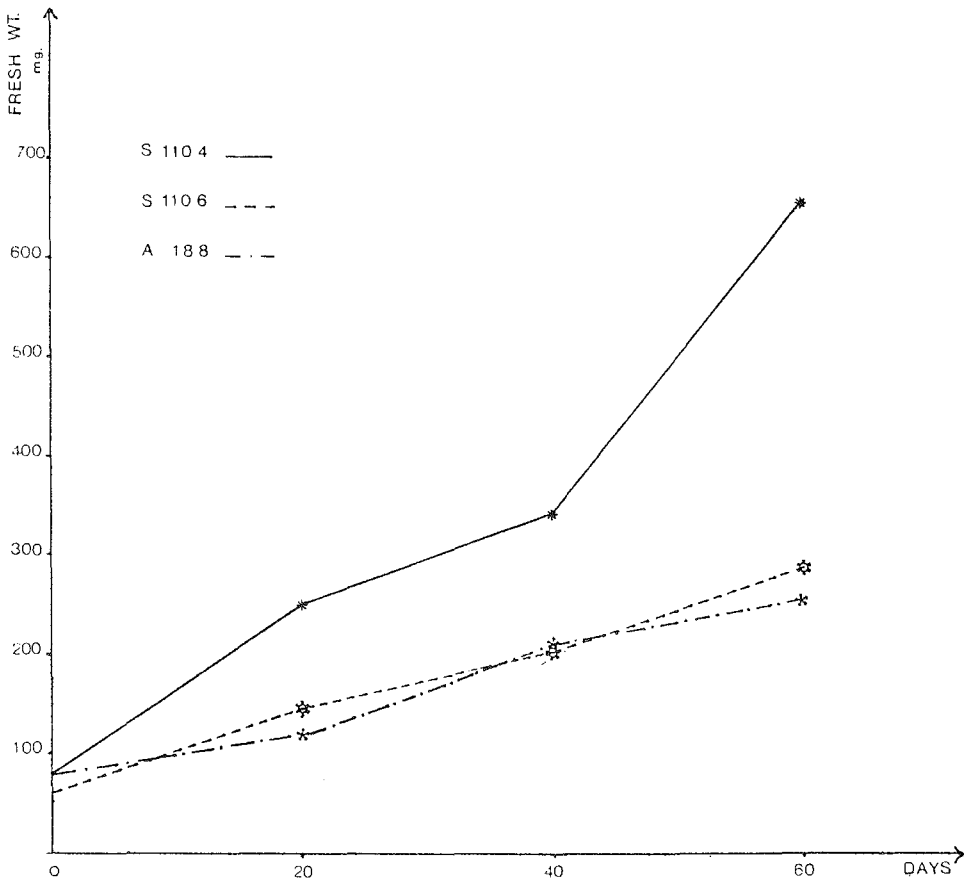
Callus induction from mature seeds has been obtained in detached embryos cultured on the Murashige and Skoog medium (1962) modified by Green (1975) with 4 mg/l 2-4D at 27 C $\pm$ 1, under continuous light. Under these conditions seedlings are produced with an extremely abnormal morphology, particularly in the mesocotyl and in the first node region where the callus develops. Callus is also obtained from roots, particularly those in contact with the medium. After 30 days callus is excised and transferred to a medium with 2 mg/l 2-4D. Subcultures are performed every four weeks.

The growth pattern of the three callus lines is shown in Figure 1, where each determination is the average of at least five replications. The lines differ in their growth, with S 1104 showing the fastest growth.

The amount of growth seems to depend on the initial weight of the explant as the data below indicate (S 1104 data only).

Fresh weight		Fold fresh weight increase at time (days) 20	40	60
81.7	(3.3)	3.1	4.1	8.1
131.9	(7.3)	4.0	3.9	10.1
294.7	(21.4)	2.0	3.0	5.3

(Each weight determination is the average of at least 10 samples).



The three lines show also morphological differences. Callus of S 1104 is friable, yellow with green spots, and shows areas with anthocyanin. No pigment is observed in the other two lines, which appear more compact. The three lines have been subcultured for 11 months.

Callus also has been obtained from immature embryos following the procedures outlined by C. E. Green and R. L. Phillips (1975). When 16-18 day-old embryos are excised and incubated on MS modified medium with 2 mg/l 2-4D, the scutellum gives rise to a yellow or pale green callus; subcultures have been propagated on the same medium. When callus is transferred to a medium with 0.25 mg/l 2-4D, roots start to differentiate. If these cultures are then transferred to a medium without hormone, roots are still formed, but in addition, shoot-like structures are also observed. These structures do not differentiate into plantlets. In the S 1106 line, however, three explants developed plantlets (3-4 from each piece) that were not further analyzed.

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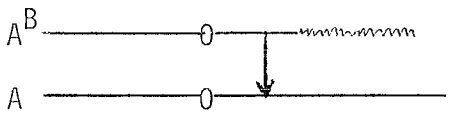
Location of the pro mutant to chromosome arm

The symbol pro refers to a recessive lethal gene mutant strictly auxotrophic for proline. Homozygous pro seeds are easily identified on the basis of the collapsed endosperm morphology. Upon germination the mutant dies before the first leaf emergence in the genetic background of W 22, while in other backgrounds it can reach a more advanced stage before dying. When necrosis ensues after the first leaf emergence, the leaf blade appears white with green stripes along the veins.

The data to be presented have been collected in order to locate the pro marker to chromosome arm following the procedures outlined in MGNL 45:144, using an improved set of B-A translocations. The procedure consisted of planting selfed seeds of a +/pro heterozygote, crossing at least three plants by each of the B-A translocation set and scoring the resulting ears for the appearance of kernels with collapsed endosperm expected in hypoploids with the pro marker uncovered. The set of B-A translocations used covered 16 chromosome arms to some degree. Since the results of these crosses were all negative, pro must lie in a portion of the genome not uncovered by the set of B-A translocations. One hundred kernels from each ear were then planted in the field, using at least three ears for each B-A translocation. Hypoploid plants were then identified on the basis of pollen sterility (50%), ear semisterility and, at least with some translocations, abnormal plant morphology. Hypoploids yield only one kind of viable gametes, i.e., those carrying the A chromosome or a crossover strand with an exchange between the centromere and the point of exchange, as outlined in the scheme below, left:



Accordingly only hypoploids for the chromosome marked with pro are expected to show, upon selfing, a segregation for the mutant significantly higher than the expected 1/4, and more so the closer the marker is to the exchange point.

The results of Table 1 indicate that pro is on chromosome 8. The frequency of the mutant in the selfed progeny of plants with TB-8 is in fact 26.4% (799/3024) and 52.6% (1545:2935) in normal and hypoploid sibs respectively.

Table 1. Selfed hypoploids tested for location of the pro mutant to chromosome arm.

B-A Translocation	Plants of B-A cross tested	Plants segreg. for pro	Hypoploids segregation 3:1	Hypoploids segregation >1/4 pro
1a	5	3/3	2	0
1b	5	1/3	2	0
2L,1S	2	2/2	15	0
3a	5	2/3	9	0
3b	3	2/3	7	0
4a	5	3/3	10	0
4L,1L	3	2/3	21	0
5S,1L	3	2/3	6	0
5a	3	2/3	9	0
6a	6	5/6	12	0
7b	3	3/3	7	0
8a	2	2/3	0	18
9b	3	1/3	1	0
9c	3	2/3	7	0
10c	3	2/3	3	0
10 (19)	3	1/2	2	0

These data are in accord with a location of pro either in the long arm in a region proximal to the centromere or in the short arm. A more precise location of the mutant will be attempted with the aid of markers in both arms of chromosome 8.

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Development of a nuclear male-sterility system for hybrid seed corn production

Nuclear male-sterile genes in hybrid seed corn production may provide useful alternatives to hand detasseling. Additional information on the Duplication-Deficiency (Dp-Df) 5-6b/polymitotic nuclear male-sterility system reported in the 1975 MGCNL (pp. 118-119) supports its utility. Important aspects of the system are stated below followed by the respective documentation.

a) Phenotype of heterozygous Dp-Df 5-6b plants is easily recognized: Plants heterozygous for Dp-Df 5-6b are chromosomally duplicated for 90% of the short arm of chromosome 5. Distinguishing features of the Dp-Df 5-6b heterozygote include shorter and wider leaves; top leaf short, usually wide, stiff, and not bending; tassel branches shorter, upright, and not bending; main tassel spike enlarged in diameter; and generally shorter plant stature. Dp-Df 5-6b heterozygotes shed abundant pollen. These phenotypic features are reminiscent of trisomic chromosome 5.

b) Smaller seed selected from the initial cross of heterozygous 5-6b translocation x heterozygous male-sterile (+/po) produces 56% Dp-Df 5-6b plants: Seed about three-fourths the size of the larger kernels was selected from the cross of the 5-6b translocation heterozygote (T5-6b/A632) as the female parent with the heterozygous male-sterile (+/po) stock. The results presented below indicate that 56% of the plants from the smaller seed were Dp-Df 5-6b heterozygotes. The frequency probably would be higher if smaller seed were selected before shelling the ear thus avoiding smaller kernels that occur naturally near the ends of the ear.

Number smaller seed planted per ear	Plant phenotype (no.)	
	Dp-Df 5-6b	Normal
8	4	2
17	8	5
21	10	6
8	3	3
21	10	2
8	5	2
6	5	1
13	2	10
7	3	4
12	3	6
121	53	41

Forty plants with the Dp-Df 5-6b phenotype and five with normal phenotype were testcrossed as the male parent onto polymitotic male-sterile plants. Since the male parent in the initial cross was heterozygous male-sterile (+/po) one-half of the resultant Dp-Df 5-6b heterozygotes should carry the po allele and give nearly all male-sterile progeny in the testcross. The half receiving the + allele should give all fertile progeny. Normal plants misclassified as Dp-Df 5-6b types should

result in a testcross ratio of 1 fertile:1 male-sterile if the po locus was heterozygous. The results given below indicate that identification of Dp-Df 5-6b heterozygotes was accurate on the basis of plant phenotype alone (no pollen classification). Only one case of misclassification was documented.

	Testcross progeny		
Phenotype	Nearly all male sterile	All fertile	Segregating 1 fertile:1 male sterile
Dp-Df 5-6b	18	21	1
Normal	0	2	3

In conclusion, selection of smaller seed and the identification of Dp-Df 5-6b heterozygotes on the basis of plant phenotype provides a simple method of extracting Dp-Df 5-6b heterozygotes.

c) Dp-Df 5-6b heterozygotes possess 39% normal, 53% smaller or partially filled with starch and 8% aborted pollen: The various categories and percentages of pollen phenotypes are reported below. Although pollen phenotype can be used to identify Dp-Df heterozygotes, the plant phenotype is sufficiently distinctive.

Plant number	Normal %(No.)	Partially filled-normal size	Well filled-smaller	Partially filled-smaller	Devoid of starch-smaller	Total
15314	43(216)	0(0)	44(221)	5(28)	8(39)	504
15315	43(426)	0(2)	43(425)	6(63)	8(84)	1000
15316-27	32(324)	4(44)	53(530)	4(42)	7(66)	1006
15316-23	47(469)	0(2)	43(435)	2(15)	8(82)	1003
15313	34(343)	3(32)	52(521)	4(45)	7(69)	1010
Total	39(1778)	2(80)	47(2132)	4(193)	8(340)	4523

d) Gametes carrying the Dp-Df 5-6b chromosome are not pollen transmitted: As reported under (e) below, 123 fertile plants have been recovered using the Dp-Df 5-6b/po heterozygote as the male parent. Using a pocket microscope, pollen from each of these plants was examined in the field. In every case the pollen was normal, suggesting that these fertile plants were the result of crossing over in the Dp-Df 5-6b/po male parent and not the result of transmission of a Dp-Df 5-6b chromosome. In addition, the fertile plants always possessed a normal plant phenotype.

Pollen of Dp-Df 5-6b heterozygotes was sieved through a 125μ/90μ/75μ/63μ/45μ U.S. Standard Sieve series. Pollen collected on the 90μ, 75μ, and 63μ sieves was plated on Cook-Walden solid germination medium. The separation of pollen was incomplete with many smaller grains remaining in the 90μ and 75μ sieves. Direct observation showed that the smaller grains, presumably carrying the Dp-Df 5-6b chromosome, did not germinate. The results reported below indicate that percent germination decreases as average pollen grain size decreases.

Plant number and constitution	Sieve dimension	Time (min.) 25	35	45	55	65	75
		% germination					
13301-15	90*	10	25	50	60	70	70
(Dp-Df 5-6b/+)	75*	5	10	15	25	40	50
	63	0	<1	1	1	1	1
13295-6	90*	10	20	30	30	50	50
(Dp-Df 5-6b/+)	75*	1	10	15	15	30	30
	63	0	0	0	0	<1	1
13301-4	90	10	20	40	40	60	60
(Normal sib)	75	10	20	40	50	50	60

*Contains many smaller grains that did not germinate after 3 hours.

The complete lack of male transmission of the Dp-Df 5-6b chromosome as indicated by the normal plant and pollen phenotype of fertile progeny in po po x Dp-Df 5-6b/po crosses probably can be explained by the effect of the Dp-Df 5-6b chromosome on pollen germination. This nuclear male-sterile system, therefore, is not based on pollen tube competition and should be effective even in environments or with genotypes where there is limited pollen production.

e) At least 98.5% male-sterile plants are produced in male-sterile (po/po) x Dp-Df 5-6b/po crosses: The 1975 MGCNL report indicated that the Dp-Df 5-6b/po had been extracted 8 times yielding 525 male-sterile plants among a total of 526. Additional data were obtained in the summers of 1975, 1976, and 1977. The total number of extractions per year and the number of male-sterile plants among the total tested per extraction are reported below.

1975: 19 extractions - 5/6, 25/25, 65/68, 50/50, 11/11, 14/14, 13/13, 10/12, 12/12, 13/13, 4/4, 61/62, 40/47, 109/109, 121/121, 76/76, 9/9, 125/127, 11/12.

1976: 44 extractions - 14/15, 16/16, 17/17, 15/16, 14/14, 14/15, 14/14, 14/14, 15/15, 11/12, 15/15, 13/15, 13/16, 15/15, 15/15, 16/16, 14/15, 103/107, 19/20, 13/14, 41/42, 86/87, 27/28, 13/14, 8/8, 36/36, 38/38, 82/82, 39/39, 67/67, 51/52, 80/83, 16/17, 13/13, 12/12, 64/65, 41/43, 32/35, 65/65, 25/25, 12/12, 9/9, 12/12, 13/13.

1977: 88 extractions - 193/202, 74/76, 85/86, 190/190, 116/117, 12/13, 232/237, 96/99, 16/16, 133/133, 90/90, 175/181, 3/3, 110/111, 94/95, 69/71, 17/18, 48/50, 37/38, 44/45, 109/109, 49/49, 77/77, 12/12, 109/109, 49/50, 13/13, 184/184, 16/16, 15/15, 11/13, 14/14, 11/11, 12/13, 14/14, 2/2, 15/15, 14/14, 15/15, 13/14, 47/47, 66/67, 88/90, 50/52, 95/95, 49/49, 69/69, 68/69, 57/57, 87/87, 43/44, 56/56, 38/40, 65/65, 68/68, 39/39, 61/62, 54/54, 105/106, 63/64 115/115, 51/51, 62/63, 118/118, 39/39, 80/81, 31/31, 56/56, 21/21, 36/36, 71/71, 100/101, 48/49, 119/124, 63/67, 21/21, 68/69, 87/87, 74/76, 95/95, 89/89, 71/71, 47/47, 32/34, 33/35, 80/80, 38/38, 48/48.

The grand total is 8210 male-sterile plants among a total of 8333, or 98.5% male-steriles. Over 58% of the extractions tested yielded all male-sterile plants, based on variable sample sizes.

f) Seed set on the male-sterile (po/po) plants can be genetically improved: A selection program aimed at improving seed set of po/po plants yielded ten lines (5 sister pairs) with good seed set after five alternating self and backcross generations to A632 (ten total generations). These lines traced back to a single selection in the fourth cycle of selection. The material was increased and tested again this season. Several ears on po/po plants were obtained with excellent seed set, suggesting that the improved seed set is heritable. Considerable variability existed among cultures and the number of male-sterile plants tested within a culture was too small to determine the stability of the improved seed set.

g) The Dp-Df 5-6b/po nuclear male-sterility system saves one generation in development: The fact that the male-sterile locus in this system is proximal to the breakpoint (see 1977 MGCNL) allows the extraction of the Dp-Df 5-6b/po heterozygote directly from a cross of heterozygous T5-6b as the female parent with heterozygous po (+/po). If the male-sterile locus is in the duplicated segment (distal to the breakpoint), the female parent must be heterozygous for the translocation and the male-sterile gene, requiring an additional generation. In both situations, only one-half of the resultant progeny will receive the male-sterile allele from the heterozygous po parent and must be identified by a testcross onto the homozygous male-sterile stock.

R. L. Phillips

Cytogenetic location of opaque-2

The data reported below cytogenetically locate opaque-2 (o2) relative to T7-9(8558), T7-9(8659), T7-9(071-1), and T7-9b. Only the opaque seeds were planted in the experiments reported here. The F1 was used as the female parent in all except one case (noted by an asterisk). The ratio of +:o2 seed is given in parentheses following the F1 genctype.

T7-9(8558) (7S.22, 9L.16)

$$\frac{+\ +\ \text{T7-9(8558)}\ +\ +}{o2\ v5\quad +\quad ra\ gl} \qquad (68\ +{:}54\ o2)$$

0	1	2	3	4	3,4	1,4	Total
33	2		1		3	1	40

T-o2 = 7.5%

T7-9(8659) (7S.55, 9S.35)

$$\frac{+\ +\ \text{T7-9(8659)}\ +\ +}{o2\ v5\quad +\quad ra\ gl} \qquad (639\ +{:}973\ o2)$$

0	1	2	3	4	1,3	2,3	2,4	3,4	1,2,3	1,2,3,4	Total
*41	1			3						1	46
63	1	1		1				3			69
62				4							66
55			3		1	1					60
43			4	1	1		1	2	1		53

T-o2 = 3.7%

T7-9(071-1)gl (7S.70, 9L.07)

+ T7-9(071-1) + + gl / o2 + v5 ra gl (561 +:481 o2)

0	1	2	3	Total
66			3	69
72			4	76
144			4	148

T-o2 = 0

T7-9b (7S.76, 9S.19)

T7-9b + + + + / + o2 v5 ra gl (215 +:105 o2, second and third cultures not counted)

0	1	2	3	4	1,2	2,3	2,4	3,4	1,2,4	Total
64	2	1		1		1	1	2	1	73
23					1		1			25
12	1		3							16

T-o2 = 4.4%

Six plants identified as duplicate-deficiency 7-9b heterozygotes (50% smaller pollen) were self-pollinated and produced the following +:o2 ratios;

178+:278o2, 29+:61o2, 110+:222o2, 101+:148o2, 151+:283o2, 217+:336o2

Grand total = 786+:1328o2

These data suggest that T7-9(071-1) has a breakpoint (7S.76) distal to the o2 locus and the Dp-Df 7-9b chromosome is transmitted through the megaspore about one-half the time. Crosses of heterozygous T7-9b♀ x o2 o2♂ yield no opaque kernels out of a total of 570.

In conclusion, the opaque-2 locus appears to be distal to 7S.55, not beyond 7S.76, and probably near 7S.70. Additional information was reported in Genetics 61:107-116, placing o2 approximately 2.5 map units distal to the 7S.62 breakpoint of T6-7(6885).

R. L. Phillips

Progress report on inactivated Ms* alleles

I had assumed that if the homozygote for the inactivated normal allele of a male sterile gene could be established, it could be used as an alternative method of producing "all male sterile progeny" for use in producing hybrid corn (MGNL 50: 82; MGNL 51:54). Although the progeny of male steriles crossed with homozygous inactivated (Ims/Ims) pollen should all be male sterile, the male steriles used for the crosses would occur in segregating cultures. Hence, as pointed out by Phillips and Albertsen (personal communication), this is not an efficient alternative method, since a system for the mass production of ms/Ims progeny (male sterile) would be difficult to develop.

Although the inactivated locus is transmitted through pollen and ovules for two lines for ms and two for ms2, tests in 1977 and earlier years, of the self progeny of heterozygotes (Ims/+), have not identified a single homozygote (Ims/Ims). Tests are in progress using linked genetic markers as an aid in selecting putative homozygotes. For ms, y and pb are being used, and for ms2, ar wx is being used. The crosses were selfed by A. S. Wang in Hawaii and the progeny were grown in 1977.

For ms, 13 cultures segregated male sterility and 15 did not. The latter should be from heterozygous Ims plants. From 6 of them which also had Y y as the marker, 72 plants were segregating Y y and one was Y Y. This Y Y plant is a putative homozygote for the inactivated locus and will be tested.

For ms2, 69 cultures segregated male sterility. The 29 which did not should be from heterozygous (Ims/+) plants. From the latter, 397 plants were segregating for waxy, 46 were Wx Wx (11.8% of the total). Although most of the latter are probably not homozygotes for Ims, since wx and ms2 are 8 units apart on the linkage map, the marker still greatly reduces the number of plants to be tested.

Hopefully if the homozygotes can be established, someone may find a use for them.

Charles R. Burnham

Cytoplasmic restoration of nuclear genetic male sterility

Hermsen (1968, Euphytica Sup. No. 1:63-67), discussed a system which would utilize a nuclear gene pair for fertility vs. male sterility (Ms* vs. ms*), together with a type of cytoplasm that would restore fertility to a genotype homozygous recessive at the ms* locus. No examples were cited. One of the advantages of such a system, as discussed by Washnok (1972, MGNL 46:25-27) is that the ultimate hybrid planted by the farmer would have normal cytoplasm. The purpose of this note is to call attention to the manner in which such a system was discovered in flax. Bateson and Gairdner (1921, J. Gen. 11:269) and Gairdner (1929, J. Gen. 21:117-124) reported that crosses using a procumbent strain as female with tall strains as male had fertile F_1's but segregated 3 fertile:1 male sterile in F_2. Progeny of the reciprocal crosses were all fertile in F_1 and F_2. The explanation offered was that the tall strains were homozygous for a recessive ms* gene but had a type of cytoplasm that made them fertile; the procumbent strain had the normal Ms* gene but a type of cytoplasm which, with an ms* ms* genotype, produced a male-sterile plant. We propose the usage of (R) for the restorer cytoplasm and (r) for the non-restorer cytoplasm. The procumbent strain would be (R) ms* ms* and the tall strain (r) Ms* Ms*.

During studies of the inheritance of wilt resistance in flax, a large-seeded Crete variety behaved in a manner similar to that of the procumbent strain (Burnham, Ph.D. thesis, Univ. of Wisc.). Only by the study of reciprocal crosses carried into F_2 was the system discovered in flax. A paper reporting results of crosses with the Crete variety of flax and outlining a system for using cytoplasmic restoration of nuclear genetic male sterility is in its final stages of preparation: Burnham, C. R., M. C. Albertsen, and R. L. Phillips, Cytoplasmic restoration of nuclear genetic male sterility.

For a similar test for such a system in corn, reciprocal dialleles within groups of inbreds in corn (7, 6, and 6 inbreds in each of three groups) are being checked in F_2 for segregation for male sterility. F_2's for all the crosses will be grown in 1978.

Charles R. Burnham

Discussions in Cytogenetics

A fifth reprinting was made in 1977. The price is $11.00 each, postage paid. Included are supplemental pages for several of the chapters. The supplement is also available separately for those who have earlier printings. Please pool orders for the latter or include the number needed with book orders.

Charles R. Burnham

Placement of genes in 5L using T5-9a data

T5-9a 5L.86-9S.4 is from a line of sweet corn breeding material, known to be segregating for partial sterility given to me by W. R. Singleton. Extensive cytological observations at pachytene and linkage tests using homozygotes and heterozygotes serve to place certain genes in chromosome 5 relative to the break point and the subterminal knob. The pachytene measurements place the subterminal knob at 5L.67 and the breakpoints at 5L.86 and 9S.4. Using T5-9a as the ♀ parent for 4-point backcross data (425 plants) the recombination values are: bm-26.3-pr-21.6-T-6.1-v2; and bm-23.8-pr-13.2-ys-14.3-T. In plants homozygous for T5-9a, the recombination values were: sh-19.3-wx-21.6-pr (301 plants). Also in the homozygote, v2 was no longer linked with pr. Hence v2 is in the distal .14 of 5L.

Charles R. Burnham

Azide as a mutagen in corn

Nilan, Kleinhofs, and coworkers (Mut. Res. 17:143, 1973) have demonstrated that azide is an effective mutagen in barley. In maize and other crops, azide treatment of seeds presents several potential advantages over other chemical mutagens. Azide is not classed as a serious mutagen, therefore it is safer than other commonly used chemicals like nitrosoguanidine and ethyl methanesulfonate. Another reported advantage is that seeds have been dried after azide treatment, stored, and planted at a later time without serious physiological damage. This report describes preliminary observations concerning the effectiveness of azide to create mutations in maize.

Inbred A619 seeds were presoaked in aerated water at 28 C in experiment 1 and at 25 C in experiment 2. The duration of the presoak time varied from 4 to 16 hours as shown in the tables. The seed from each presoak time was then treated at the same temperature for 2 hours with 10^{-3} M sodium azide in 0.1 M phosphate buffer at pH 3.0. Vigorous aeration was maintained during the presoak and treatment periods. Control seed was treated for 2 hours in phosphate buffer without azide. After the treatments were completed the seed was air dried at room temperature and planted within one month.

The M1 plants obtained from treated seed were self pollinated whenever possible and the remainder were crossed to control plants. From each M1 cob, 2 seeds were selected for the M2 generation and were planted in separate plots and reported as replications I and II in the tables. All M2 plants were self pollinated.

Physiological damage observed in M1 plants increased with the length of the presoak time. With 8 hours presoak-azide treatment, the ears on many M1 plants either did not form or ear emergence was delayed. These plants did tassel well. Most 12 and 16 hour presoak-azide treated plants were severely stunted. These plants formed tillers but generally did not form normal ears or tassels.

Kernel mutations were observed in seed from M2 plants including abortive, various defectives, brittle, pale yellow, and viviparous types. A confirmation of the numbers of mutants shown in the tables will be made by demonstrating their heritability in an additional generation. Combined frequencies in experiment I

Experiment 1. A619 seed presoaked for various times in aerated water at 28°C and then treated with sodium azide.

Presoak(hrs)	M1 population	M2 population / replication*	M2 recovered Rep I	M2 recovered Rep II	Kernel mutants segregating in M3 Rep I	Kernel mutants segregating in M3 Rep II	Seedling mutants segregating in M3 Rep I	Seedling mutants segregating in M3 Rep II
Control	600**	110	93	49	0	2	0	***
4	600	401	245	195	14	13	10	***
8	600	216	148	121	10	15	9	***
16	600	0	-	-	-	-	-	-

* Numbers listed for the presoak times represent 100% of M1 ears recovered and sampled to produce the M2 generation. Number listed for control represents samples from 90% of M1 control ears.

** M1 control represents a composite of 200 seed samples from 4,8, and 16 hour presoak times.

*** Not completed.

Experiment 2. A619 seed presoaked for various times in aerated water at 25°C and then treated with sodium azide.

Presoak(hrs)	M1 population	M2 population / replication*	M2 ears recovered Rep I	M2 ears recovered Rep II	Kernel mutants segregating in M3 Rep I	Kernel mutants segregating in M3 Rep II	Seedling mutants segregating in M3 Rep I	Seedling mutants segregating in M3 Rep II
Control	500**	120	54	60	1	2	3	***
4	500	417	279	239	13	7	9	***
8	500	172	100	79	5	6	8	***
12	500	46	35	23	4	6	6	***
16	500	7	3	0	0	-	0	-

* Numbers listed for presoak times represent 100% of M1 ears recovered and sampled to produce M2 generation. Number listed for control represents samples from 75% of M1 control ears.

** M1 control seed had 16 hour presoak treatment.

*** Not completed.

gave kernel mutations of 1% in controls, 6% for 4 hours, and 9% for 8 hours. In experiment II, the frequencies were 3% for controls, 4% for 4 hours, 6% for 8 hours and 15% for 12 hours.

M3 seedling mutations included albino, virescent, yellow, light or yellow green, dwarf, glossy, and photosynthetic types. The frequencies of seedling mutants were 0% for controls, 4% for 4 hours, and 6% for 8 hours in experiment I, and 6% for control, 3% for 4 hours, 8% for eight hours, and 17% for 12 hours in experiment II.

Our preliminary conclusions are that azide treatment of seed does increase the frequency of mutations recovered in maize, but the effectiveness is dependent on the length of the presoak period before azide treatment. Other factors such as the pH of the treatment buffer or the azide concentration may be varied to further improve mutation induction conditions for azide in maize.

Kenneth Hibberd and C. E. Green

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Hoyer's acetocarmine (HAC): a one-step staining and mounting technique for chromosome preparation

Hoyer's solution is a water soluble mounting medium; acetocarmine, a water soluble stain. Florence S. Wagner, at the University of Michigan, Ann Arbor, working with fern chromosomes, combined these 2 solutions in equal volumes, producing Hoyer's acetocarmine. She and her husband have used this with excellent results for several years.

In preparing a chromosome squash, the HAC is used as a conventional stain. Chromosomes stain lightly, but can be seen clearly using phase contrast. To make a preparation permanent the slide is allowed to sit for 2-3 days at room temperature and in this time the HAC slowly hardens. Slides are then sealed using clear nail polish. The great asset of this technique is that the slide becomes permanent without having to remove the coverslip.

Hoyer's solution

60 gms gum arabic	100 mls distilled water
400 gms chloral hydrate	40 mls glycerine

Dissolve gum arabic in water for 24 hours. Add chloral hydrate. Let stand 24 hours. Add glycerine. Do not use heat. Bubbles should disappear after several hours.

Acetocarmine

45 cc glacial acetic acid	55 cc distilled water

Add 0.5 gm of carmine, boil gently for 5 mins in a reflux condenser. Shake well and filter when cool. A drop or two of 45% acetic acid saturated with iron acetate can be added.

The Wagners at Michigan use Newcomers solution as the fixative of choice for fern material before HAC preparations. Newcomers was originally developed for bird chromosome research. An initial study using maize has been made to compare this fixative with the traditional Farmer's fluid (absolute alcohol 3 : acetic acid 1) and 2.5% glutaraldehyde in pH 6.8 buffer. The latter is used in electron microscopy for plant material and is recommended as a non-coagulative fixative. Newcomer's without dioxane was also tested but it was not satisfactory because the

sporocytes burst too easily and the chromosomes did not stain significantly. Material placed in Newcomer's is allowed to stand several hours at room temperature, before being stored in a freezer. Fern material fixed in Newcomer's has been stored for years without deterioration. Summary of initial results:

Character	Farmer's Fluid	Glutaraldehyde	Newcomer's Solution
Sporocyte plasmolemma	Often breaks in squash preparation	Rarely breaks	Often breaks in squash preparation
Appearance of chromosomes	Lightly stained	Stained very heavily	Acceptable staining. Chromosomes stained darker than material fixed in Farmer's.
Nucleolus	Large, irregular in outline; less dense than in glutaraldehyde fixations. Stains uniformly.	Small, dense, circular in outline with distracting halo effect between nucleolus and cytoplasm	Large, irregular in outline. Stains clearly but unevenly.
Nucleolus organiser region	Clear	Indistinct	Clear

With material fixed in Newcomer's it was easier to get more mother cells with good chromosome spreads than with material from any other fixative, otherwise the results with Newcomer's and Farmers were nearly equivalent.

When glutaraldehyde was used as a fixative the cytoplasm fixed well, but the nucleolus appeared to shrink in size, leaving a gap that appears as a distracting halo around the structure. Glutaraldehyde penetrates tissues very slowly and this may result in osmotic shrinking of the nucleolus before fixation.

James Leuck and Robert Dummer

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Light-induced pigmentation of c-n and c-2420 due to Bh interaction

While screening c isolates for ability to form anthocyanin pigment with light and germination conditions, I found that both c-2420, the recessive allele isolated from C-I (Coe, Genetics 47:779-83, 1962), and the reference c-n allele of Coe respond in some seeds and not others. For example, in c-2420/c-2420 seeds from a selfed progeny of c-2420/C Bh/bh the light response segregates approximately 3:1 in the blotched seeds (pigment developing in non-colored cells) and segregates approximately 1:1 in the non-blotched seeds. These four seed classes were planted and selfed. Some Bh seeds upon selfing gave only a few Bh progeny while other selfs gave nearly all Bh. Some bh seeds that pigmented with light may have been misclassified, since the selfs of a few such seeds segregated for Bh. These two

problems argue against bulking the data, but in general the results are as follows, where (+) = formation of pigment with light, and (-) = no change with light.

	Progeny Class			
	Blotched		Non-blotched	
Parent Class	(+)	(-)	(+)	(-)
Bh(+)	100%	0%	50%	50%
Bh(-)	Variable		Variable	
bh(+)	Variable		Variable	
bh(-)	No Bh progeny		0%	100%

The precise percentages listed above are probably not significant, because of the variability within families, but it is at least clear that the light response can only occur in bh seeds if they are from an ear segregating Bh. This suggests that the Bh effect may be maternal, which is being tested with the Florida crop.

The Bh effect is interesting because Bh action is known to elicit pigmentation to the C level in certain c cells. Perhaps Bh can elicit partial expression (to the light-inducible level) in all c cells.

Sheila McCormick

A method for obtaining homozygous duplications

One of the problems with working with duplications is getting them homozygous. Duplications such as tDp3La and dDp3L-3S (MNL 46:138-142) and those produced by crossing translocations or inversions with overlapping break points are difficult to transmit through the pollen because of competition with normal pollen. A solution to this problem is to cross the duplication/normal heterozygotes with the appropriate TB-A translocation and then to self-fertilize the hypoploid progeny. The chromosome with the duplication is freed from competition with normal chromosomes and homozygous progeny results. This was attempted last summer. Because of poor growing conditions few hypoploids survived. It will be repeated.

G. G. Doyle

An ageotropic primary root mutant

A new mutant, ageotropic (agt), was discovered in the selfed M_2 progeny of seeds which were treated with 0.2% EMS for 12 hours. The primary root is unresponsive to gravity and may grow horizontally or may find its way to the surface and grow several centimeters long before dying. The other roots, seminal and adventitious, have normal geotropic responses. Three families, all derived from a single treated kernel, have segregated agt. The segregation was 28 + : 2 agt, 21 + : 2 agt, and 44 + : 3 agt, for a total of 93 + : 7 agt, a ratio of 13.3 + to 1 agt. It is possibly a case of duplicate factors. More likely it is a recessive which is difficult to classify. The primary root may grow down just by chance in some cases.

G. G. Doyle

The genic balance of replicated elements and heterosis

It is known from reassociation experiments that much of the DNA of eukaryotes is highly replicated. If genes are in these replications they will fluctuate in number because of unequal crossing over and unequal sister chromatid exchange. The instability of inbred lines for quantitative characters, which was analyzed by Sprague et al., 1960 (Genetics 45:855-866), could be the result of changes in the

number of genes affecting these characters. A population will have chromosomes with a range in the number of units in a replication. An equilibrium is established between the formation of divergent numbers and stabilizing selection pressure, which removes replications with high and low numbers of units from the population. If this selection pressure is less effective in open-pollinating species than in self-pollinating species then we can construct an hypothesis to explain heterosis.

Because quantitative characters are controlled by many genes we must construct a model in which selection acts on a group of different genes which are replicated. This may be best explained by using a crude model. Let us assume that there are three genes (A, B, and C) on different chromosomes that control steps on a synthetic pathway.

Each of these genes is highly replicated at levels of 1, 2, 3, 4, or 5 (these numbers indicate relative numbers of units). The system is geared for the 3 level (6 in the diploid level). Because of unequal crossing-over or unequal sister chromatid exchange there is divergence from the 3 levels to other levels. The frequency of the different kinds probably approximates a normal curve. It is assumed that their frequencies are: 1(1/16), 2(4/16), 3(6/16), 4(4/16), and 5(1/16)--this is a binomial distribution. There are 5^3 (125) possible gametes and 9^3 (729) possible zygotes. Some genotypes found in a randomly mating population and inbred lines derived from it, with their relative fitness, are given below:

No. of Units			Frequency		
A	B	C	RM	IL	Fitness
2	2	2	.000000	.000244	1.0000
6	6	6	.020444	.052734	1.0000
10	10	10	.000000	.000244	1.0000
2	6	6	.000292	.008789	.7084
10	6	6	.000292	.008789	.9128
4	6	8	.003271	.023438	.8889
5	6	7	.013084	-------	.9722

The frequency of the various zygotes will be different in a randomly mating population (RM) and in inbred lines (IL) obtained from it. The frequency of 2-2-2 is $(1/256)^3$ in RM and $(1/16)^3$ in IL. Gametic frequencies of the RM give the zygotic frequencies in the IL. The RM population has less genic imbalance because of the possible combinations of low and high levels from two parents. Also some genotypes such as 5-6-7 (which has a high fitness) are not possible in the IL's.

The fitness, the relative amount of progeny produced by an individual, is suggested to depend on the genic balance of the system. This is given an arbitrary, but relative, numerical value by the use of the following formula where the replication levels of different genes in the same system are multiplied and then divided by their mean value raised to the n power, thus:

$$\frac{A \times B \times C}{\left(\frac{A + B + C}{3}\right)^3} = F$$

It will be seen that the fitness of the first three cases in the table is 1. If the system is independent the efficiency depends on genic balance. A 2-2-2 system would "work overtime" and a 10-10-10 system would "stop work early." Other systems are genically unbalanced and inefficient. It is easy to understand why a 2-6-6 system would be in trouble because of a bottleneck effect. A 10-6-6 system,

however, should perhaps have no trouble--there will be only an excess of the A product. However, this may disturb the functioning of the B and C genes. If we consider the poor adaptation of trisomes we see that some sort of mechanism like this is acting. Maize trisomes, for example, have reductions in yield from about 20 to over 80 percent.

If we multiply the frequencies of the genotypes by their fitness we can obtain values for the average fitness of the population. The randomly mating population has a fitness of .9416 and the average fitness of the inbred lines obtained from it is only .8787 (the computations required even for this simple model are lengthy and cannot be given here--xeroxed copies will be sent on request). The difference between the two values is significant but not great. If we consider that the total fitness of a plant is the product of the fitness of several systems then this difference is compounded--thus if there are 10 systems, $(.9416)^{10} = .5478$ vs. $(.8787)^{10} = .2744$.

Inbreeding tends to increase the genic imbalance. The balance may be restored when two inbred lines are crossed (2-2-10 x 10-10-2 = 6-6-6).

It is known that related species may have greatly different amounts of DNA and this has been suggested to mean that they are geared to different levels of replication. Corn Belt corn was produced by crossing Northern Flints and Southern Dents. It is possible that they had different optimum levels. The result of crossing them is suggested by the following:

	Level of Replication					
	1	2	3	4	5	6
Race #1	1/16	4/16	6/16	4/16	1/16	0
Race #2	0	1/16	4/16	6/16	4/16	1/16
Hybrid	1/32	5/32	10/32	10/32	5/32	1/32

The variance of the hybrid is increased and there is a greater potential for genic imbalance in the inbred lines extracted from the hybrid than from either parental race.

The testing of this hypothesis is difficult and the mathematical model needs to be refined. This model of heterosis does not exclude the co-existence of other models.

G. G. Doyle

Allelism of pg14 and g5

Four plants heterozygous for pg14 (Peterson, P. A., Genetics 38:682-683, 1953) were pollinated by homozygous g5 (Beckett, Coe, and Neuffer, MNL 47:147-148) and the F_1 was tested in the sandbench. All four families segregated 1 normal to 1 pale green mutable, so it is evident that pg14 and g5 are allelic.

J. B. Beckett

Narrow leaf probably on the long arm of chromosome 10

Three crosses of +/nl x TB-10Lb segregated narrow leaf seedlings in the sandbench, while +/nl x TB-10Sc (one cross) gave normal seedlings only, so it appears that nl is located on 10L rather than on 10S as previously believed (Beckett, MNL 49:131-134). Progenies were small, so confirmation is needed.

J. B. Beckett

Position of rgd, po, and hcf*-26 with respect to TB-6Sa

In field tests in 1977, po +/+ rgd x heterozygous TB-6Sa segregated a rather low frequency of male-sterile plants (i.e., polymitotic hypoploids). Several other plants were hypoploids with 50% pollen abortion. Two of these, when self-pollinated, gave progenies segregating a total of 3 normal and 31 ragged seedlings in sandbench tests. Therefore, po is uncovered by TB-6Sa and rgd is proximal, confirming previous reports (Phillips, Patterson and Buescher, MNL 51:49-52; Palmer and Dempsey, MNL 42:76-77).

Crosses involving +/hcf*-26 x TB-6Sa uncovered, at a somewhat low rate, yellow-green seedlings with high chlorophyll fluorescence under long wave ultraviolet light, thus confirming that hcf*-26 is on the short arm of chromosome 6 beyond the breakpoint of TB-6Sa, as reported last year (Leto and Miles, MNL 51: 57-59).

J. B. Beckett and K. Leto

Interactions among endosperm mutants for amylose synthesis and total starch content

A wide range of percent amylose and total percent starch has been reported among the major endosperm mutants, including ae, du, fl, fl2, h, o2, su, and su2. Multiple combinations of corn endosperm mutants affecting amylose-amylopectin ratios have been shown to have considerable genetic information. We studied the ten possible double combinations of endosperm mutant genes among 5 genes, including fl2, h, o2, su2, and du all in the inbred line W64A background. Interactions were studied through a diallel analysis (Table 1). A wide range of mean values for five variables measured was observed (Table 2).

Although differences among the ten double mutants were significant for all five variables, a Duncan multiple range test indicated no significant differences existed between any two means for the variables studied except percent amylose. Variance associated with general combining ability (GCA) for amylose synthesis was four times larger than for specific combining ability (SCA), and both were significant at the 1% level. We would suggest that the multiple combinations of endosperm mutants could be effective in upgrading percent amylose. Both GCA and SCA for percent starch were significant at the 5% level. Variance associated with GCA for 100 kernel weight was not significant; however, SCA for the same trait was significant. Specific gene combinations could change morphological traits.

GCA estimates of the endosperm mutant for the five variables reflect the general characteristics of each mutant involved in the double combinations (Table 3). The results imply an inverse relationship of percent amylose and percent starch, although the magnitude of the effect was different among the five mutants. The results could also suggest that it may be possible to find a favorable gene which is more efficient in total starch synthesis with no or little reduction in amylose synthesis. The incorporation of the selected favorable gene or genes and the ae gene may be considered as a possible step to increase both amylose and starch content.

The 10 double mutants were regrouped into 5 double mutant groups, each group representing one of the double combinations involving fl2, h, o2, su2, or du. The correlation coefficients for the 5 groups were computed separately (Table 4). It could also reflect the characteristics of the mutants involved in double combinations. A very high positive correlation between 100 kernel weight and starch weight/100 kernels for all the 5 double mutant groups was indicated. Correlations between percent amylose and percent starch were not significant or were significantly negatively correlated. It is not likely to upgrade both percent amylose and percent starch through double combinations of genes. However, in our data we could assume a possible candidate for incorporation with ae is the h gene, since

Table 1. Analysis of variance for 10 double mutants in the W64A background.

		Mean Squares				
	df	% Amylose	% Starch	Amylose Weight 100k	Starch Weight 100k	100 Kernel Weight
Double mutant	9	60.30**	27.91**	0.515**	2.54*	3.56*
GCA	4	103.44**	9.85*	0.900**	2.30*	3.21
SCA	5	25.78**	14.45*	0.206^{ns}	2.74*	3.83*
Error	10	0.54	2.65	0.076	0.64	0.97

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

NS Non-significant

Table 2. Mean values for five variables, % amylose, % starch, amylose, wt/100 kernels, starch wt/100 kernels, and 100 kernel weight.

	% Amylose	% Starch	Amylose Weight /100 k (grams)	Starch Weight /100 k (grams)	100 k Weight (grams)
f12 h	26.4 (10)	68.65 (6)	3.017 (10)	11.428 (3)	16.665 (2)
f12 02	27.35 (8)	68.75 (5)	2.563 (7)	9.371 (10)	13.545 (10)
f12 su2	29.15 (6)	71.55 (3)	2.814 (2)	9.655 (7)	14.130 (9)
f12 du	26.9 (9)	69.90 (4)	3.106 (9)	11.507 (2)	16.425 (3)
h o2	29.0 (7)	72.50 (1)	3.255 (8)	11.203 (4)	15.445 (5)
h su2	29.8 (5)	71.60 (2)	3.802 (3)	12.774 (1)	17.840 (1)
h du	37.4 (3)	66.60 (8)	3.602 (6)	9.648 (8)	14.560 (4)
o2 su2	40.9 (1)	65.60 (10)	4.227 (1)	10.332 (5)	15.755 (4)
o2 du	38.2 (2)	67.30 (7)	3.650 (5)	9.555 (9)	14.195 (8)
su2 du	36.9 (4)	66.25 (9)	3.679 (4)	9.962 (6)	15.035 (6)
S. D.	0.73	1.63	0.28	0.80	0.98

Table 3. GCA estimates for endosperm mutants in the W64A background.

	% Starch	% Amylose	Starch Wt./100 Kernels (grams)	Amylose Wt./100 Kernels (grams)	100 Kernel Weight
f12	+1.17	-6.33	-0.07	-0.06	-0.23
h	+1.22	-2.08	+0.96	+0.06	+1.02
o2	-0.40	+2.22	-0.57	+0.07	-0.83
su2	-0.11	+2.67	+0.18	+0.35	+0.44
du	-1.88	+3.53	-0.50	+0.18	-0.41
S.E(Gi-Gj)	0.94	0.42	0.46	0.16	0.56

Table 4. Correlation coefficients among 5 groups of double mutants.

	Gene involving one of combination				
Comparision	f12	h	o2	su2	du
% amylose vs. % starch	NS	NS	-0.78*	-0.91**	NS
% amylose vs. amylose wt/100k	NS	NS	0.89**	0.71*	0.76*
% starch vs. starch wt/100k	NS	NS	NS	NS	0.85**
wt. 100 k vs. amylose wt/100k	0.97**	NS	0.74*	NS	NS
wt. 100 k vs. starch wt/100k	0.86**	0.96**	0.90**	0.97**	0.98**

*, ** Denotes significance at the 0.05 and 0.01 percent level of probability, respectively.

it had the highest positive GCA estimate for percent starch and starch weight/100 kernels and also appeared to have no reduction in amylose weight/100 kernels. Correlation between percent amylose and percent starch was not significant for the double combination group involving h. We don't have data to support this assumption, and further studies should be conducted in this regard.

H. S. Chang and M. S. Zuber

Nucleus and cytoplasm in the segregation and effects of iojap

White seedlings in the progeny of crosses of ij ij ear parents by + + pollen were demonstrated by M. M. Rhoades (Cold Spring Harbor Symp. Quant. Biol. 11:202, 1946) to be the result of changes in maternally inherited (plastid) properties, occurring somatically during ear development. L. B. Mazoti (see MNL 50:58, 1976) has demonstrated that not all + + pollen parents yield white seedlings in such crosses, and that progeny tests of plants from crosses of ij ij x + + (white-negative) strains do not reveal hidden (i.e., suppressed) white lineages. In addition, Mazoti has observed that Euchlaena cytoplasm confers much-reduced expression of ij. Possible nuclear and cytoplasmic influences on white seedlings and on iojap segregation ratios have been examined in a range of strains and cytoplasms.

Are white-negative strains common? Six inbred strains were tested by crossing onto ij ij ear parents and sampling to test for green vs. white seedlings:

Male parent	No. ears tested	Green seedlings	White seedlings
K55	33	969	2
Ky21	7	164	0
Mo17	4	142	1
Oh51A	11	366	37
Tr	14	413	1
W23 (A C R-g)	5	114	0

Of the six strains, only Oh51A was consistently white-positive.

Do different cytoplasmic origins affect ij segregation differentially? Highly converged strains of two different inbred lines, K55 and Tr, each with six different cytoplasms, were crossed by ij ij, and the F_1's were carried to F_2 by crossing to the F_1 involving the inbred itself, or to the F_1 involving the other inbred (Table 1). While there was an overall deviation from 25% that is highly significant (22.18%), it is apparently consistent through all the families (heterogeneity is not high in the sub-families), and there is no consistent nucleus- or cytoplasm-specific deviation.

Does prior exposure of the cytoplasm to ij ij desensitize it to the effects of ij ij in subsequent segregations? Inbred lines were crossed by ij ij males and onto sibling +/ij and ij ij ear parents in a family segregating 1:1 (from +/ij x ij ij), and the F_1's were selfed (Table 2). There was no consistent pattern of effect of ij ij exposure on the segregation ratios.

Except for the finding that five of the six inbred strains tested are white-negative, these experiments identify no nuclear or cytoplasmic alteration of iojap segregations.

E. H. Coe, Jr.

Table 1. F2 segregations of ij from hybrids with K55 and Tr inbreds in six different cytoplasms.

Cytoplasm	Ear parent	Pollen parent	+	ij	$X^2_{3:1}$
K55	K55/ij	K55/ij	140	53	0.832
T	"	"	146	34	3.585
S	"	"	253	65	3.526
C	"	"	277	59	9.921**
Wn	"	"	164	27	12.023**
249	"	"	147	52	0.240
		Sum	1127	292	2.234
				X^2_h =	16.625**
Tr	Tr/ij	Tr/ij	174	46	1.964
T	"	"	194	74	0.975
S	"	"	249	76	0.452
C	"	"	244	60	4.491*
Wn	"	"	230	82	0.274
249	"	"	418	135	0.102
		Sum	1509	473	3.074
				X^2_h =	7.479
K55	K55/ij	Tr/ij	215	71	0.005
T	"	"	71	29	0.853
S	"	"	126	33	1.528
C	"	"	161	38	3.700
Wn	"	"	187	37	8.595**
249	"	"	231	65	1.459
		Sum	991	273	0.290
				X^2_h =	9.043
Tr	Tr/ij	K55/ij	263	77	1.004
T	"	"	186	39	7.053**
S	"	"	233	69	0.746
C	"	"	142	50	0.111
Wn	"	"	240	63	2.861
249	"	"	76	23	0.165
		Sum	1140	321	0.055
				X^2_h =	5.200
	Grand Total		4767	1359	25.905**

Table 2. F2 segregations from hybrids with inbred lines and cytoplasms exposed or not exposed to ij ij.

Inbred	Cytoplasm	+	ij	$X^2_{3:1}$	
38-11	38-11	392	100	5.734**	
K55	K55	140	53	0.832	
N6	N6	584	147	9.325**	
Oh51A	Oh51A	117	45	2.000	
38-11	+/ij	362	123	2.755	
"	ij	308	107	3.035	
K55	+/ij	294	110	5.843*	
"	ij	68	24	0.058	
Kr	+/ij	238	46	11.737**	
"	ij	391	94	8.166**	
Ky21	+/ij	404	112	0.081	
"	ij	307	76	5.432*	
N6	+/ij	146	47	0.043	
"	ij	184	70	4.177*	
Tr	+/ij	240	47	11.383**	
"	ij	388	103	4.237*	
CI21E	ij	323	105	0.050	
Sum	+/ij	1843	523	10.577**	X^2_h = 21.804**
	ij	2480	748	5.751*	X^2_h = 11.053

Rescue of lethal defective kernel mutants by genetic manipulation

We have identified and catalogued 855 separate kernel mutants in progenies from material obtained by treatment of pollen with ethyl methanesulfonate according to the method of treatment described below. The development of treatment methods is outlined in recent publications (M. G. Neuffer, 1978, Genetics and Breeding of Maize, D. B. Walden, ed., and M. G. Neuffer and E. H. Coe, 1978, Maydica).

Paraffin oil method for pollen treatment

1. Suspend 0.11% by volume of ethyl methanesulfonate (Eastman #7830) in light domestic paraffin oil (Fisher Lot 722268) by vigorous stirring for an hour or more.
2. Mix EMS suspension with fresh pollen in a plastic vial with a cap, using at least 15 times as much oil as pollen.
3. Shake the pollen-oil mixture periodically to prevent the pollen from clumping in the bottom of the vial.
4. After 50 minutes apply the pollen-oil mixture to fresh silks using a #10 camel hair brush. Apply only enough oil to get a good distribution of pollen. Stirring with the brush is necessary between every application.

Extreme precautions (mixing under a hood, protective clothing, sanitary disposal, etc.) should be taken to protect all persons associated with the work, as chemical mutagens are potentially carcinogenic and are particularly penetrating in oil.

The kernel mutants include a wide range of types from extremely defective mutants to viable mutants such as su and wx. They can be grouped into three broad classes:

1. Those affecting only the endosperm (such as sh, su or wx)
2. Those affecting only the embryo (such as gm)
3. Those affecting both the endosperm and embryo (such as de).

The third class may be divided into (a) viable and (b) lethal types, lethality meaning failure to germinate.

Class 3b and possibly class 2 mutants are of particular interest because they represent a large group of mutants about which very little is known and which hold the promise of including some auxotrophs (Neuffer, 1978). Many of the lethal defective kernel mutants had small, partially-developed embryos indicating that failure was delayed allowing a certain amount of growth. This suggests the possibility that some of these mutants might be rescued by supplying the missing gene product either by genetic manipulation with B-A translocations or by culturing immature embryos on supplemented media.

To identify the proper classes for study, the kernel mutants were screened as indicated in Figure 1.

In the group, 491 mutants incapable of producing viable plants were identified. Of these, 159 were selected for intensive investigation as indicated in the report that follows. A total of 396 kernel mutants including many of the 855 classified above and some from other experiments were tested for location to chromosome arm using J. B. Beckett's series of B-A translocations. A summary of the results is shown in Table 1 (147 mutants located, 37.1%).

As we proceeded to locate the defective endosperm mutants to chromosome arm we recognized that the TB-A chromosome crosses produced genetic differences in the endosperm and embryo that could be used as a way of rescuing defective kernel mutants with lethal embryos. In the B-A translocation method for locating mutants

Figure 1. Classification of kernel mutants according to viability

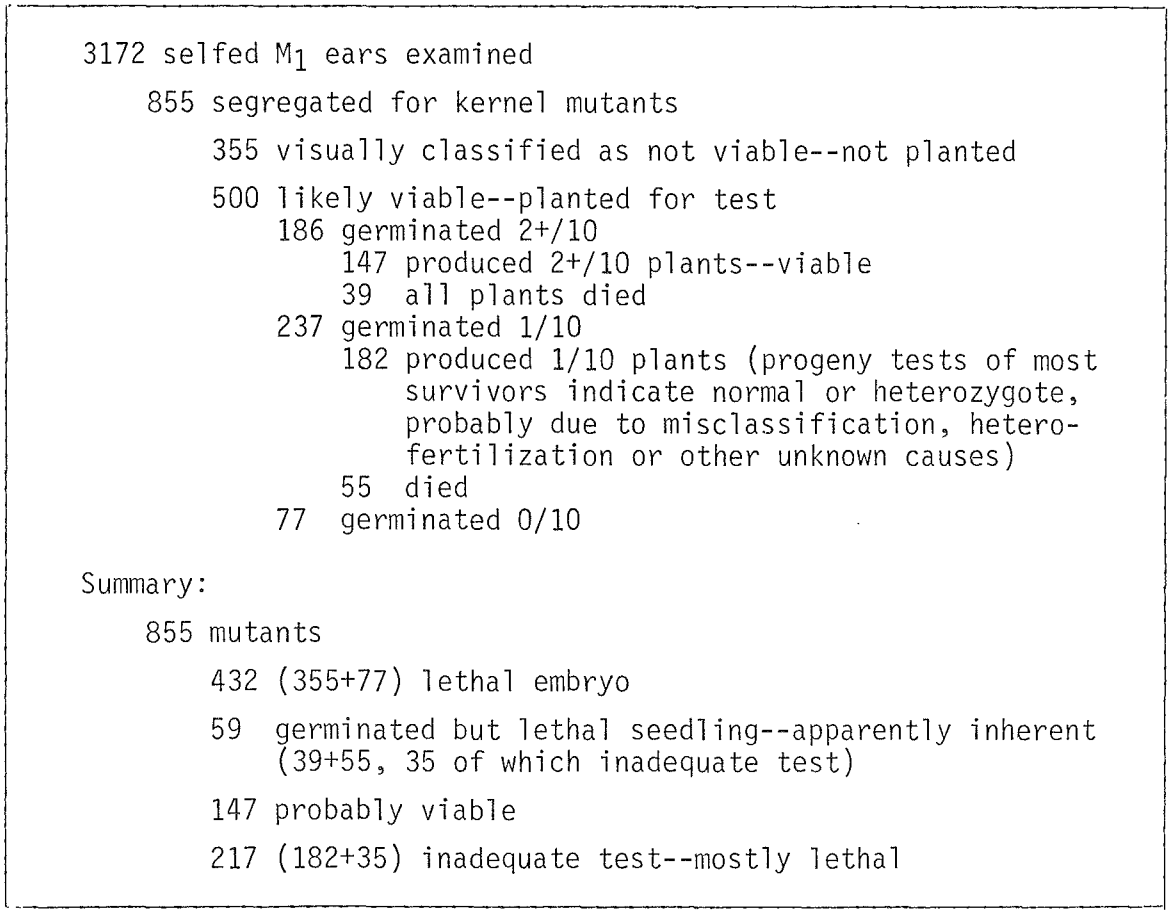

Table 1. Summary of kernel mutants located to chromosome arm

Number	Chromosome Short Arm	Long Arm
1	21	11
2	18	14
3	0	15
4	10	6
5	1	13
6	1	5
7	0	19*
8	0	1
9	4	3
10	1	4

*A certain number of these are not true locations. Hypoploidy for TB-7b produces kernels with small, opaque and sometimes defective endosperms.

developed by H. Roman and A. J. Ullstrup (Agron. J. 1951) and perfected by J. B. Beckett (Genetics 1972), each mutant is crossed by a series of B-A translocations in which each of the arms (currently 18 of 20 arms) of the A chromosome set (carrying non-mutant genes) is translocated to the centromere-carrying region of a B chromosome. Non-disjunction of the B centromere at the 2nd microspore division produces sperm nuclei with either 2 copies (duplication) or no copies (deficiency) of the particular chromosome arm involved. If the arm is the one on which the mutant is located, the mutant will be expressed in the cell lineages of the deficient sperm as a hemizygote (m/-).

Assuming a mutant female gamete in the locating cross (+/m x TB-A +/+), the consequences of non-disjunction in the male are such that: (a) the endosperm which receives no copy of the chromosome in question and is therefore mutant (hypoploid, m/m/-) is associated with an embryo that receives 2 copies and is non-mutant (hyperploid, m/+/+); or (b) an alternative in which the endosperm receives 2 copies and is non-mutant (hyperploid, m/m/+/+) and is associated with a mutant embryo receiving no copies of the locating chromosome (hypoploid, m/-), see Figure 2. This sets up a situation where a potentially lethal mutant embryo is associated in the same kernel with a genetically normal endosperm. The question is, will the normal endosperm feed the mutant embryo or vice versa in the reverse situation.

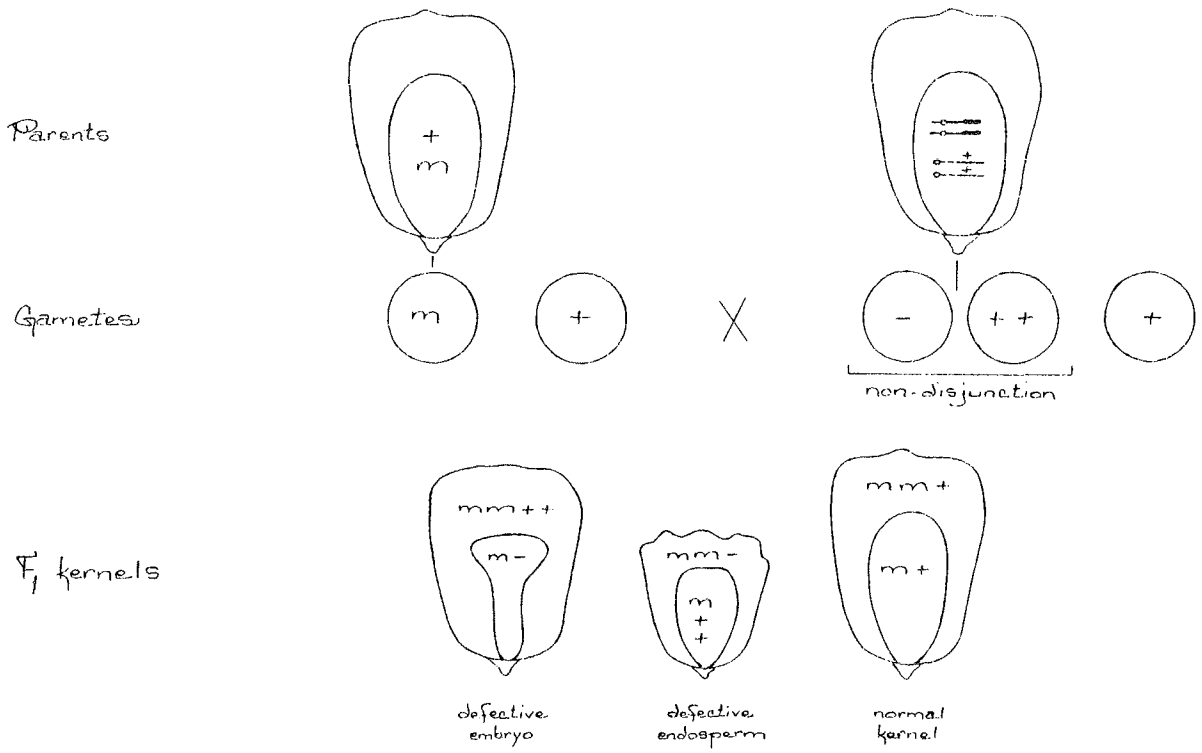

Figure 2. Results of crossing m/+ x TB-A for the appropriate chromosome arm.

Table 2. Listing of defective mutants tested for endosperm-embryo interaction in non-corresponding hyperploid-hypoploid combinations from the cross of +/mutant by B-A translocation of the proper constitution.

Mutant E No.	Chromosome Arm	Viability (Corresponding Emb-Endo)	Phenotype (Hyper endo, mm++/ Hypo emb, m-) Embryo	Seedling	Phenotype (Hypo endo, mm-/ Hyper emb, m++ Endosperm		Embryo
76B	7L	0	gm	0	m^+	←	N
198C	7L	0	gm	0	m^-	→	N
330D	3L	0	gm	0	m^+	←	N^-
627D	3L	0	N*?	leth	m^+	←	N
749	7L	0	gm	0	m^+	←	N
792	1S	0	gm	0	m^-	→	N
863	5L	0	gm	0	m^+	←	N
873	9S	+?	N*?	leth	m		N
974	7L	+	-N	leth	m		N
991	1S	0	N*?	leth	m^+	←	N
1045	5S	+(1)	-N?	0	m		N
1122	2S	+weak	small	leth	m	←	N^-
1160	5L	+?	gm	0	m		N
1208	1S	+?	-N?	0?	m		N
1283	3S	0	defective	0	m		N^-
1289	2S	0	gm	0	m^-	→	N
1296	6L	0	gm	0	m		N
1298	7L	0	gm	0	m^-	→	N
1299	5L	0	gm	0	m^-	→	N
1303	1L	0	small*	leth	m^-	→	N
1311	3L	0	gm	0	m^{--}	→	N
1312	2S	0	gm	0	m^-	→	N
1315	1L	0	N*	yg	m		leth
1316	2L	+	-gm	0	m		N?
1328	5L	+	N	N	m		N
1331	5L	0	gm	0	m		N
1332	7L	0	gm	0	m		N
1333	2S	0	N-gm	0	m		N
1339	3S	+wl	N*	pg leth	m		N
1341	7L	0?	N*	cb antho	m		N
1348	1S	0	gm	0	m		N

* possible case of + endosperm feeding m embryo; $^-$ column 4, possible case of + endosperm inhibiting m embryo; $\overset{\rightarrow}{\leftarrow}$ direction of advantage from endosperm:embryo interaction; ^{m} mutant phenotype; $^{m+}$ towards normal; $^{m-}$ extreme mutant; N^- less than normal; ^{l} luteus (yellow) seedling; ^{wl} yellowish white seedling; ^{yg} yellow green seedling; ^{gm} germless (or very small embryo); ^{pg} pale green seedling; $^{cb\ antho}$ anthocyanin crossbands on leaf; $^?$ designation still not clear.

Summary of Table 2 (see also Figure 2)

31 Mutants tested, located on 12 chromosome arms
- 22 inviable in homozygote (concordant)
- 9 with some degree of viability

Non-Mutant hyperploid endosperm (mm++)/Mutant hypoploid embryo (m-)
- 19 endosperm did not help embryo
- 7 helped to some degree (3 best: E1303, E1315, E1339)
- 5 endosperm inhibited embryo growth (E974 best)

Mutant hypoploid endosperm (mm-)/Non-mutant hyperploid embryo (m++)
- 14 no interaction
- 7 embryo helps endosperm
- 8 embryo inhibits or depletes endosperm
- 2 endosperm blocks growth of embryo (E1283, E1315)

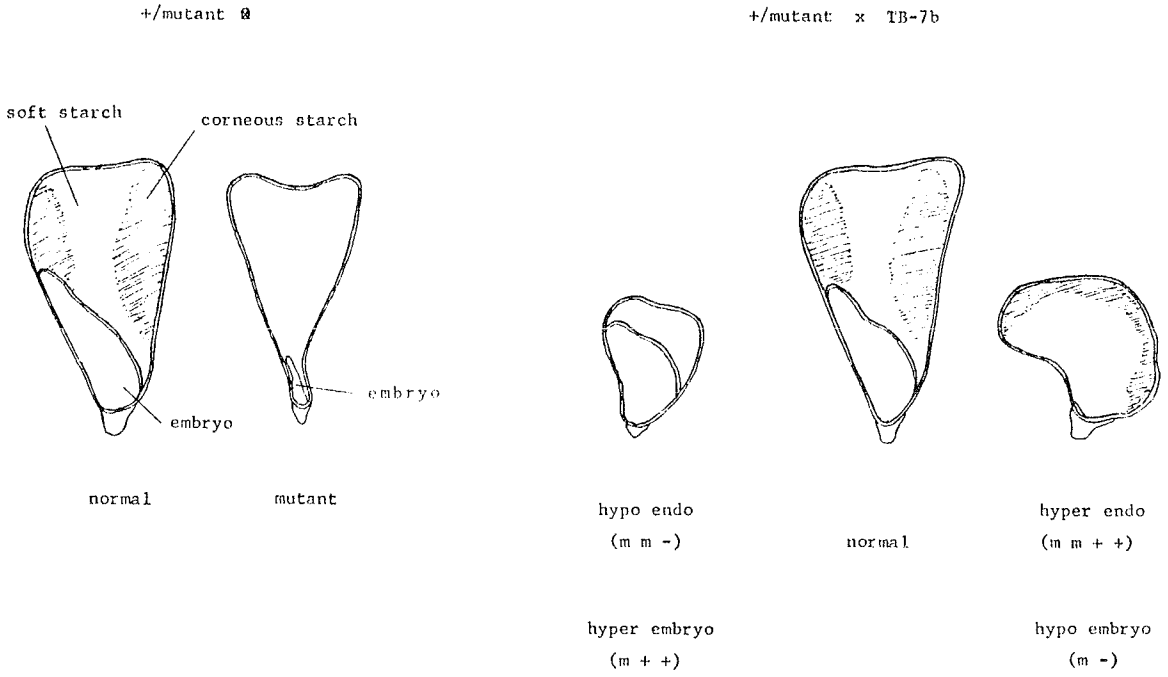

Figure 3. A diagrammatic representation of kernel cross-sections of mutant and hypoploid-hyperploid combinations for floury germless, one of the 8 mutant cases (listed in Table 2) in which the non-mutant embryo depleted the mutant endosperm.

Of the selected 159 mutants, 72 have been located to chromosome arm (45.3%) and of these, 31 have been tested for their response in both embryo-endosperm combinations described above. The results are seen in Table 2.

M. G. Neuffer, W. F. Sheridan and E. Bendbow

Rescue of lethal defective endosperm mutants by culturing immature embryos

Examination of kernels of EMS-induced lethal defective endosperm mutants has revealed that many of the mutants had partially developed embryos which had been interrupted at some time before kernel maturity, suggesting the possibility that the embryo in those cases had been sustained in its development either because the mutant did not control an earlier developmental stage and therefore was delayed in expression, or because some constituent had been supplied for a limited period by the surrounding cells. If the latter case should be true, there is hope that providing more of the required constituent will bring about more normal development of the embryo and, therefore, restore viability. Removal of the embryo from an immature mutant kernel at an early developmental stage and placing it on culture medium containing nutritional supplements may enable it to overcome the mutant defect, permitting the rescue of such mutants from lethality.

The procedure used to test this hypothesis was to plant stocks known to be segregating for each mutant, self-pollinate the plants and select for culturing at the earliest stage that the mutant kernels were distinguishable. Segregating ears

Table 1. Mutant immature embryo growth on basal and enriched media.

Mutant E. No.	Mutant Embryo: Age (Days)	Mutant Embryo: Length (mm)	Days Cultured	Growth response to media*: NH_4 Free Basal	Basal	Enriched	Mutant Phenotype
330	15	1-2	38	+	+	+	
627	16	1-2.5	37	+++	++	+	
740	16	3-4	32	+++	+++	+++	
744	15	1-2	21	++	++	++	
788	14	1.1	21	0	0	0	
792	16	1-2	36	(+	+	++)**	
873	15	2.5-3.5	21	++	+	+++	
874	13	1.5-3	23	+++	++	+	
883	16	3-4	22	+++	+++	+++	
888	15	2.5-3.5	21	+++	+++	+++	
890	15	4	35	+++	+++	+++	
912	16	2.5-3.5	20	+++	+++	+++	g/w stripe
923	17	2-4	21	+++	+++	+++	varieg.
931	14	1.5-3	21	+++	+++	+++	
932	14	1-2	21	++/+	+	+++	
933	15	2-3	21	+++	+++	+++	
936	14	1.5-2.5	21	+++	+++	+++	
948	15	1.5-2.5	9	cont.	++	cont.	
1009	18	3.5-5.0	21	+++	+++	+++	
1045	14	1.5-2.5	21	++	++	++	luteus
1054	14	2-3.5	36	+++	++	+++	
1058	13	tiny	28	0	0	0	
1076	15	2.5-4.0	9	cont.	+++	cont.	
1092	12	1.5-3	21	cont.+	cont.+	++	
1113	14	1.5-3	21	+++	+++	+++	
1121	18	4-5	20	++	++	+++	
1142	15	1-2	21	+++	+++	+++	
1156	17	1-2.5	21	++	++	++	
1162	15	1-2	21	++	++	+++	
1163	14	1.5-2.5	21	+++	+++	+++	
1167	14	1-3	21	+++	+++	+++	
1168	14	1-2	22	+++	+++	+++	
1176	18	1-2	21	++	+++	+++	
1202	15	1.5-3	21	+++	+++	+++	
1239	14	1.5-2.5	23	+++	+++	+++	
1255	24	1.5-3	21	+	0/+	++	
1283	14	1.5-2.5	21	+++	+++	+++	
1287	17	2.5	36	+/++	++	cont.	
1294	16	1.5-2.5	43	++	++	+	
1303	11	2-3.5	21	+++	+++	+++	
1311	13	tiny	21	+	+?	+	
	17	1	36	++	++	0	
1313	17	2.5-3.5	21	+++	+++	+++	
1319	16	2.5-5	21	cont.++	cont.++	++	
1324	15	2.5-3.5	21	+++	+++	+++	pg/w stripe
1331	14	1.5-2.5	21	+++	+++	+++	
1332	17	1-3	41	++	++	+	white
1339	14	2-5	21	+	+	+	
1369	14	1-2.5	21	+++	++	++	
1373	17	3-4	21	+++	+++	+++	
1380	15	1.5-2.5	16	cont.++	cont.++	cont.0	
1381	14	1.5-2	21	+	++	++	
1383	15	1.5-3.5	21	+++	+++	+++	
1385	14	1-1.5	21	+/0	0	0	
1387	15	1.5-2.5	21	++	++	++	
1391	15	1-2.5	21	+	++	++	
1392	14	1-3	21	++	+	++	
1394	15	1-2.5	21	++/+	+	+/0	
1395	15	1-2	21	++/+	++/+	++/+	
1399	14	1-1.5	21	0	0	0	
1406	17	1.5-3	21	++	++	+	
1411	17	1-2	21	++	+++	+++	
1415	16	1-2	21	++	++	+	white
1417	13/18	tiny/1.5-3	31/39	0/++	0/++	0/+++	
1419	14	tiny	21	cont.0	cont.?0	cont.0	
1420	21	3.5-5.5	21	++	+++	+++	
1421	24	2-4	22	++	+	++	white
1423	19	1.5-2	22	++	++	+	
1427	15	1.5-3	28	++	++	++	
1429	17	1.5-2.5	22	++	++/+	+	
1430	17	1-1.5	21	+	++/+	+++	
1431	14	1-2	22	++	+++	++	
1435	16	1-2	22	+	+	+	
1436	15	1-2	22	+	+	+	

*growth response:
0 embryo cultured--failed to enlarge
+ embryo enlarged--plumule or radical visible
++ small plantlets less than 5 cm high
+++ plants greater than 5 cm--weak to normal
tiny = too small to separate from endosperm--cultured endosperm-embryo mass
** roots only--weight comparison
cont. = cultures contaminated

were surface-sterilized and the immature embryos dissected out and placed individually into culture tubes, some containing mineral salts (T. Murashige and F. Skoog, Physiol. Plant., 1962) with 4% sucrose and 0.8% agar (basal medium) and some containing basal medium supplemented with amino acids, vitamins and nucleic acid bases (enriched medium). When possible, 10 mutant embryos from each segregating ear were cultured on each type of medium and 10 normal embryos from the same ear were cultured on the regular basal medium. The cultures were placed in a growth chamber with a mean temperature of 24 C and a light-dark regime of 16 hr: 8 hr for a period of three to five weeks, after which the growth response of the embryo on each medium was observed, the height in the tube measured, and fresh weights obtained of shoots and roots, if they had developed.

During the past two years, 81 lethal defective endosperm mutants have been cultured as described above. Results for the 72 mutants cultured during the summer of 1977 are presented in simplified form in the accompanying table.

About four-fifths of the mutants grew (responses ++ and +++) on two kinds of basal media (regular basal and ammonia-free basal) and on enriched medium. At the other extreme in response were nine mutants that did not grow on any of the media (responses 0 and +). Despite our assumption that it was unlikely that any mutants would grow only on basal media, nine mutants did display a preference for the unenriched media. Of particular interest are the few mutants that seem to prefer the ammonium-free basal medium (E627, 874, 1369, and 1394) and the few that grew only, or considerably better, on the enriched medium (E792, 1092?, 1121, 1255, 1162, 1417 and 1430). The mutants in the latter group are of special interest because it is among this type that auxotrophic mutants are expected to be found.

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Archaeological maize from Peru

Detailed studies of archaeological maize samples show that on the North Coast of Peru new types were introduced with each new cultural shift between 1050 B.C. and A.D. 600. Study of the 903 Gallinazo cobs excavated from the uppermost layers of the Huaca Prieta site, mostly a preceramic, pre-maize midden, demonstrates great contrast with earlier Cupisnique and Salinar maize arrays and with a later Moche sample. The Cupisnique material (161 specimens) comes from just north of Huaca Prieta (excavated by Junius Bird in the Chicama Valley just north of Trujillo, as were the Gallinazo specimens). Salinar maize is represented by over 200 whole cobs and fragments excavated by Michael West at Puerto Moorín in the Virú Valley. The Moche sample is from Huaca de la Cruz in the Virú Valley just south of Trujillo, excavated by Duncan Strong and Cliff Evans. Except for West's specimens, the collections have long been stored at the Botanical Museum of Harvard University and the Missouri Botanical Garden.

109 of the Gallinazo cobs were measured seven ways, and 21 of the 109 remain unclassified, being unique or intermediate. The measurements were chosen to be measurable on all specimens and to represent as many trends of variation as possible. Cupule outline drawings were also made. The rough data sheets are available; detailed definitions of the variables are forthcoming.

Maize appeared on the Peruvian coast with the start of Chavín influence on ceramics, textiles, metallurgy, etc. at about 1050 B.C. (uncorrected radiocarbon date). On the North Coast, the Chavín-like Cupisnique culture left four maize types at Huaca Prieta (Table 1); types CU-2 and CU-3 are closely related. Just to the south in the Virú Valley, the Salinar culture brought higher row-number

Table 1. The general ranges for 16 Peruvian North Coast archaeological maize types. All dimensions are in millimeters.

Culture	Type[1]	N[2]	Rachis diameter	Row number	Cupule width	Cupule length[3]	Cupule wing width[4]	Rachis segment length[5]	N'	Cob length[6]	N"
Moche											
	MO-1	4	6.9-9.6	8-10	5.3-7.1	2.3-3.3	.9-1.6	3.7-4.7	(4)	5.2-?9.6	(2)
Gallinazo											
	GA-1	15*	5.6-9.1	14-18	2.4-3.3	1.3-2.0	.4-.7	2.7-3.9	(12)	3.3-6.3	(5)
	GA-2	19*	7.6-9.9	12-14	3.7-4.3	1.2-1.9	.5-.7	3.0-3.7	(17)	2.8-5.9	(6)
	GA-3	10	4.2-6.3	10-12	2.5-3.3	1.2-1.9	.3-.7	2.6-3.2	(7)	2.8-3.5	(5)
	GA-4	19*	5.0-8.4	8-12	3.5-4.9	1.5-2.4	.5-1.0	3.1-3.9	(13)	2.5-6.3	(10)
	GA-5	18*	5.9-15.0	10-14	4.8-6.1	1.4-2.2	.8-1.3	3.3-4.1	(15)	2.8-8.1	(8)
	GA-6	7	5.4-11.7	12-14	3.5-5.0	2.1-3.4	.6-1.1	3.7-5.2	(5)	---	
Salinar											
	SA-1	2	12.1-16.3	22	3.1-4.2	1.3-1.5	.4-.5	2.4-3.5	(2)	6.0-7.2	(2)
	SA-2	2	11.9-15.4	26-30	2.3-2.8	1.4	.3	2.7-2.9	(2)	4.9-5.9	(2)
	SA-3	2	17.1-17.5	26-30	3.9-4.5	1.1-1.4	.5	2.4-2.7	(2)	5.4-7.1	(2)
	SA-4	2	9.2	16-18	2.8-3.0	1.2-1.8	.3-.5	2.6-3.4	(2)	5.7-7.2	(2)
	SA-5	2	9.4-12.8	16	3.7-3.8	1.0-1.2	.5-.7	2.4-3.1	(2)	7.1	(1)
Cupisnique											
	CU-1	2	9.8-11.4	20-24	3.0-3.6	1.2-2.2	.3-.5	3.1	(1)	4.9-6.2	(2)
	CU-2	8	7.5-10.4	18-22	2.3-3.1	1.5-1.8	.3-.6	2.5-3.6	(4)	4.0-6.0	(4)
	CU-3	8	6.4-8.0	14-16	2.0-3.3	---	.3-.6	---		4.5-5.4	(4)
	CU-4	3	5.2-7.1	12	2.4-3.0	1.3-1.6	.3-.7	2.6-3.1	(2)	3.5-5.7	(2)

* If N is over 11, the highest and/or lowest values may be omitted in the general range.

[1] CU-1 is like SA-1; CU-2 is like CU-3, SA-4 and GA-1; CU-4 is like GA-3; SA-5 is like GA-2.

[2] N for rachis segment length is N' since not all specimens have been measured, and for cob length it is N" since many cobs are broken.

[3] Cupule length is the cupule height of Galinat.

[4] The cupule wing is the same as the rachis flap of Nickerson.

[5] Galinat's internode is shorter than rachis segment length and longer than cupule length.

[6] Cob length is given only for entire or almost entire specimens.

types (SA-2 and SA-3) at about 200 B.C. with some specimens so extreme that the cobs are hollow and highly fasciated. Types SA-1, SA-2 and SA-3 may be only forms of one broad type.

Suddenly, the high row-number types disappeared, and at Huaca Prieta, three completely new types appeared with the succeeding Gallinazo culture (types GA-4, GA-5 and GA-6). At about A.D. 150, row numbers fell even more and cobs lengthened with the rise of Moche hegemony, at least in the Virú Valley.

Though no maize of the Middle Horizon (A.D. 550-900) from the Trujillo area has been studied, much evidence points to high row-number types, like the Salinar maize but higher yielding, being important just to the southeast at this time.

Elsewhere in Peru the abrupt changes observed on the North Coast are not seen. Maize from Wallace's Cerrillos site on the South Coast (stored at Harvard) is very like the Salinar and Cupisnique maize, but this evolves into Nazca maize without an abrupt shift (Grobman, Salhuana and Sevilla with Mangelsdorf, 1967, Races of Maize in Peru). On the Central Coast, the Lighthouse site excavated by Feldman

has Cerrillos-like maize, judging from photographs, but the nearby Aspero maize (excavated by Willey and Corbett, stored at Harvard) is very variable and includes specimens falling within the ranges of all the Gallinazo types (Towle, 1954, in Willey and Corbett, Early Ancón and Supe Culture). Kelley and Bonavia's Huarmey maize of the Central Coast (a few cobs remain at Harvard) is similarly variable.

Interpretation, though a bit premature, is possible. The samples of maize supposedly preceramic (pre-1750 B.C.) from Aspero and Huarmey seem to be considerably later, unless a very variable maize, with some cobs being as large as maize of two millenia later, is erased from the scene between 1750 and 1050 B.C. to be succeeded by a thoroughly different set of types. Chavín-related maize along all the coast had a similar range of variation and evolved into Salinar and Nazca maize. Gallinazo and, to a greater degree, Moche introduced new types more like maize found to the north both in archaeological sites and in farmers' fields. The interaction of the high and low row-number types may have led to a rapid evolution of Peruvian maize, as Mangelsdorf has suggested.

Robert McK. Bird

The CIMMYT maize germplasm catalog data base

Until recently the data pertaining to over 11,700 maize germplasm samples maintained by the International Maize and Wheat Improvement Center (CIMMYT) at El Batán, Mexico, have been difficult to obtain. The preparation of over 100 computer-readable files on cards and tapes from scores of field books, inventories and odd notes took two and a half years of work by Mario Gutierrez and myself. His role, besides regenerating the seed, was to prepare an inventory of the cans (up to 4.5 gallons in five cans per entry); mine was to obtain, complete and correct data on each entry for the Catalog Data Base.

The materials available at CIMMYT are almost entirely from Mexico, Central America, the Caribbean and eastern South America. Most are collections made in farmers' fields, markets or research institutions. About 700 entries are groups (narrowly-based composites representing very similar collections from a small geographic area) or composites-varieties (a range of materials produced by many breeding programs).

These entries were largely collected by three programs--CIMMYT, the joint Rockefeller Foundation-Mexican government project (two series) and a joint Brazil-US project based at Piracicaba. Many of the early Mexican collections are maintained only by the Instituto Nacional de Investigaciones Agricolas at Chapingo, Mexico. The second RF-Mexican program series and the CIMMYT series date from the 1956-1970 period. The Brazilian set (including many from neighboring countries) is mostly pre-1956. Almost all have been recently regenerated, although a large number of Andean collections were still being increased in 1976.

The information comes from collection notes, entry lists, can inventories, regeneration nurseries, agronomic, morphological and other studies, and there are files for group and composite-variety information, abbreviations and codes, and odd notes and synonyms.

Wherever possible original collection notes were transcribed, first onto forms, then onto cards, but often secondary sources were used. When the Brazilian germplasm bank was transferred to CIMMYT, only a few of the entry cards accompanied the entries, but other cards have been located and sent to CIMMYT since. The genealogies and complete names of the groups and composites-varieties are available in several files. Knowing the synonyms of the entries is important since many have several commonly-used labels; in parts of the Caribbean and Central America most of the collections have been described under a temporary collection number in numerous publications, but now carry a different number. For instance, Panama 22B, Panama 22P and Panama 22Q now are Panama 47, 131 and 180 respectively.

A preliminary catalog version was offset-printed in 1975; the files, and queries of the Data Base, are available from the Information Sciences/Genetic Resources Program of the University of Colorado. The punched cards are kept in the statistics section of CIMMYT, and the source documents are stored in the CIMMYT archive.

This belated note is submitted now because a longer report is still awaiting publication.

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Nomenclature for isozyme genes in maize

Formal genetic analyses have been reported for a number of maize isozymes over the past decade. Many isozyme systems have both multiple loci and multiple alleles. Since there has existed some confusion in the literature as to the proper conventions for nomenclature of isozyme genes and alleles, we describe below a few simple rules which should help clarify this situation. We also present here a list of the maize isozyme genes which have been studied in our laboratory. Only those isozymes which have been defined genetically are listed in Table I. Not included, therefore, are isozyme bands for which electrophoretic variants have not been found or for which formal genetic analysis has not yet been determined.

The references listed in Table I are those which define the isozyme system in terms of gel patterns, developmental and/or biochemical data. The abbreviation for maize catalase genes has been changed from *Ct* to *Cat* in order to avoid confusion with the older designation of clumped tassel as *Ct*. We have also changed the designation of maize aminopeptidase genes from *Lap* (leucine aminopeptidase) to *Amp* in order to reflect overlapping specificities toward amino acid-naphthylamides.

To conform with the adopted rules of nomenclature for defining maize genes we are adhering to the following system and suggest it for other isozyme genes. Preferably a three-letter abbreviation for the isozyme gene is used and is followed by the locus number. Both letter and number are italicized, the first letter being capitalized. When designating a particular allele, the gene symbol is followed by a dash and the allelic symbol (also italicized), which is usually a reference to the electrophoretic mobility of the isozyme gene product. For example, the *Cat1-F* allele codes for the catalase subunit CAT-1F.

Either a letter or a number can be used to denote the allelic isozyme products under a given set of electrophoretic conditions. Designating the band positions of a multi-locus isozyme system is also a matter of choice with the only rule which should be strictly adhered to being that the first genetically-defined isozyme locus to be reported is given the numerical designation 1, the second 2, etc., regardless of the isozyme band number which corresponds to the gene product. For example, *Mdh2* has two alleles, *Mdh2-m3* and *mdh2-m5*, which code for mitochondrial malate dehydrogenase isozymes mMDH-3 and mMDH-5, respectively.

Flexibility is allowed in the allelic designation in order to encompass differing degrees of complexity of the isozyme system. For isozymes which show distinct subcellular compartmentations, it is often useful to maintain this information in the allelic symbol by using m, g, s, or c for mitochondrial, glyoxysomal, soluble, or chloroplast.

Unfortunately, very few isozyme genes have been mapped to date. We are presently attempting to localize a number of the genes listed in Table I in order to extend their usefulness as biochemical-genetic markers.

Lila A. Ott and John G. Scandalios

Table I. Some isozyme genes of maize and their alleles.

Enzyme	Gene	(Former Designation)	Alleles	References
Alcohol dehydrogenase	*Adh1*	*Adh1*	*F, S*	5
	Adh2	*Adh2*	*F, S*	5
Aminopeptidase	*Amp1*	(*LapA*)	*F, S, V*	7, 10
	Amp2	(*LapD*)	*F, S*	7, 10
	Amp3	(*LapC*)	*F, S, I, V*	7, 10
	Amp4	(*LapB*)	*F, S*	7, 10
Amylase	*Amy1*	(*Amy1*)	*A, B*	2
	Amy2	(*Amy2*)	*A, B*	1
Catalase	*Cat1*	(*Ct1*)	*F, S, V, K, M*	4, 6
	Cat2	(*Ct2*)	*R, Z*	4, 8
Endopeptidase	*Enp1*	(*Ep1*)	*D, E, A, C, B, O*	3
Glutamate-oxaloacetic transaminase	*Got1*	(*Got1*)	*gF, gS*	9
	Got2	(*Got2*)	*sF, sS*	9, 10
	Got3	(*Got3*)	*mF, mS*	9, 10
Malate dehydrogenase	*Mdh1*	(*Mdh1*)	*m1, m2*	11
	Mdh2	(*Mdh2*)	*m3, m5*	11
	Mdh3	(*Mdh3*)	*m1*	11
	Mdh4	(*Mdh4*)	*m3*	11

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The effect of light on subcellular distribution of catalase in the leaves of maize

One of the sources of hydrogen peroxide in plant tissues is the oxidation of glycolate. During the processes of photosynthesis glycolate is synthesized in chloroplasts and oxidized by glycolate oxidase after being transported into peroxisomes. Since photosynthesis provides substrates of glycolate oxidation, green leaves get more hydrogen peroxide than etiolated leaves. Therefore, it is reasonable to find more catalase present in peroxisomes of green leaves than in etiolated leaves.

The highly inbred maize strain W64A was used in these experiments to determine the subcellular distribution of catalase during the development of seedlings. The leaves of 5-day or 6-day-old green and etiolated seedlings were used to prepare homogenates separately for sucrose density gradient centrifugation. Catalase activity was found in peroxisomal and soluble fractions as expected, but in addition it was also found in the mitochondrial fractions. Green leaves have higher catalase activity in the peroxisomal fraction than the etiolated leaves (Table 1). The ratio of catalase activity in green leaves to that of etiolated leaves indicates that the effect of light is greater on the peroxisomes than on the mitochondrial and soluble fractions (Table 2). These results suggest that peroxisome development in light is one of the factors which elevate catalase activity in corn leaves, although there may be some other mechanisms involved. The genetic control and physiological function of the compartmentalized catalases is being investigated.

Table 1. The relative activity of catalase in subcellular compartments in primary leaves of 5-day-old seedlings.

SUBCELLULAR COMPARTMENT	Dark (5 day dark)	Light (3 day dark + 2 day light)
Microbody	6.24%	16.38%
Mitochondria	42.00%	29.02%
Soluble	30.30%	34.65%
Others	21.46%	19.95%

Table 2. The subcellular distribution of catalase in green and etiolated leaves of 6-day-old seedlings.

Subcellular compartment	Green leaves		Etiolated leaves		
	Relative activity	Absolute activity	Relative activity	Absolute activity	Ratio of absolute activity
Microbody	11.02%	0.0935	6.57%	0.0176	5.31
Mitochondria	37.91%	0.3216	40.83%	0.1094	2.94
Soluble	34.61%	0.2937	31.83%	0.0853	3.43
Others	16.46%	0.1396	20.77%	0.0557	2.50
Total	100.00%	0.8483	100.00%	0.2680	3.16

D. Y. Chang and John G. Scandalios

Expression and intracellular localization of superoxide dismutases

Maize superoxide dismutase has been resolved into five major electrophoretic forms by starch gel electrophoresis (Fig. 1). These have been tentatively labeled SOD-1, SOD-2, SOD-3, SOD-4, and SOD-5 in order of their migration to the anode, in accordance with the recommendations of the Subcommittee on Multiple Molecular Forms of Enzymes of the IUPAC-IUB (1971). All five isozymes are present in the liquid endosperm, scutellum, pericarp, root, primary leaf, coleoptile sheath, and mesocotyl, and show quantitative differences in their expression among these various tissues. Cell fractionation studies using sucrose gradient centrifugation have demonstrated the presence of a cyanide-resistant isozyme (SOD-3) in the mitochondrial fractions of the scutellum and epicotyl. In addition, SOD-1 (a cyanide-sensitive isozyme) was found to be associated with the chloroplasts and the etioplasts of developing maize leaves. Contrary to what has been previously reported (Giannopolitis and Ries, Pl. Physiol. 59:309, 1977), cyanide-sensitive superoxide dismutases were not found to be associated with the mitochondria. Furthermore, only a single electrophoretic form (SOD-1) was observed in the plastid fraction.

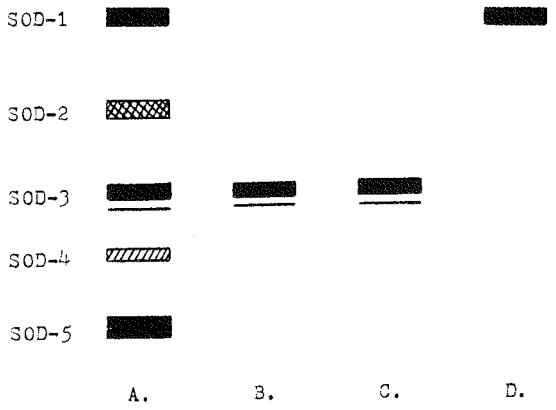

Fig.1: A. Zymogram showing superoxide dismutase isozymes from maize liquid endosperm; B. Gel assay in the presence of 2 mM cyanide; C. Mitochondrial fraction from 4-day old maize scutella; D. Chloroplast and etioplast fractions from 8-day old maize seedlings.

Further research with this system will be devoted in part to the characterization of the biochemical and genetic bases of these enzymes. In addition, experiments are being planned to study the physiological relationship between superoxide dismutase, catalase, and O_2^- generating systems within the cell.

James A. Baum and John G. Scandalios

Terminal inverted repeats in the unique DNAs associated with the S cytoplasm

Recently we reported finding unique, plasmid-like DNAs associated with mitochondria from the S-type of cytoplasmic male-sterile maize (PNAS 74:2904-2908). These unique DNAs have never been observed in mitochondrial DNA preparations from normal, T, C and EP cytoplasms. Conversely, they have been found in every S cytoplasm studied regardless of source or nuclear background. These associations have suggested a causal relationship between the unique DNAs and the S type of male sterility. Importantly, the unique DNAs are only isolated from mitochondrial preparations and not from chloroplasts or nuclei. Very recently, strict maternal transmission of the unique DNAs has been demonstrated.

The unique DNAs associated with the S cytoplasm (S-S and S-F) were isolated from mitochondria and separated by gel electrophoresis. Examination by electron microscopy revealed that the S-S and S-F DNAs were linear molecules of 6212 and 5227 base pairs, respectively. For comparison, the phage, ϕX174, contains a DNA molecule of 5375 base pairs which codes for nine genes.

When the S-S or S-F DNAs were denatured in 95% formamide and renatured in 50% formamide at room temperature for five minutes, lariat-like configurations were observed by electron microscopy of formamide spreads (Fig. 1). The lariat-like structure consisted of a short double-stranded (DS) stem and a large single-stranded (SS) loop. The double-stranded stem contained 195 and 168 base pairs for the S-S and S-F DNA, respectively, This constitutes 3.1 and 3.2 percent of the S-S and S-F molecules, respectively.

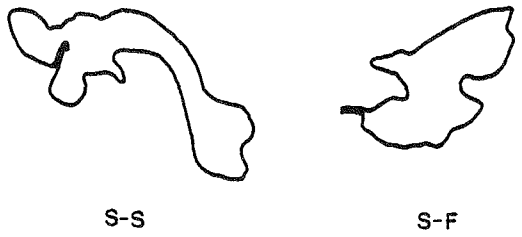

Figure 1. Tracing of unique DNA molecules (S-S and S-F) associated with mitochondria from the S cytoplasm after denaturation and reannealing. Loop portion is single-stranded while stem portion is double-stranded. Mounting was by the formamide technique.

The occurrence of stem-loop structures is interpreted as being due to terminal inverted repeats on the S-S and S-F molecules (Fig. 2). Intrastrand stem-loop structures form with first-order kinetics when the terminal inverted repeats pair. Figure 2 gives a diagrammatic presentation of the events leading to the formation of stem-loop structures.

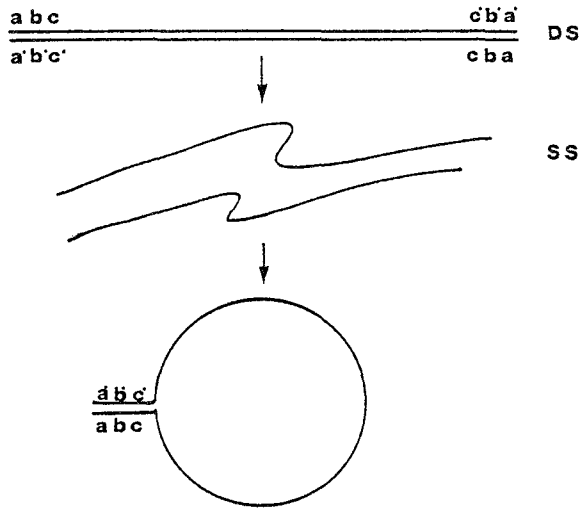

Figure 2. Diagrammatic representation of double-stranded (DS) DNA denatured (upper arrow) into single-stranded molecules (SS). Subsequent intrastrand reannealing (lower arrow) results in a single-stranded loop and a double-stranded stem.

The importance of terminal inverted repeats on the unique DNAs from the S cytoplasm is not clear. Inverted repeats are often prominently involved with insertional events in lower organisms. It is tempting to hypothesize that the unique DNAs described here are in some way related to the unstable nature of the S cytoplasm.

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Public inbred lines resistant to the heat and drought conditions encountered in the Southeast in 1977

As a part of an attempt to survey a wide range of materials for isozyme studies, about 250 public inbred lines were grown for increase last summer. These were mostly planted April 28, although 17 were planted about a week later. The full set included almost all commercially used public inbred lines of yellow and white field corn, sweet corn, and popcorn, except those from Florida, Louisiana, and the Harrow station in Canada. A number of historically important but now obsolete lines were included. A list is available on request. Most lines failed to set seed in quantity and quality sufficient for proper maintenance, despite irrigation,

Table 1. Lines apparently resistant to the extreme growth conditions encountered in North Carolina during the summer of 1977.

Line	Yield (0 = none 10=v.high)	Good Seed Quality	Flowering Date	Plant Ht. (Feet)	Ear Height (Feet)	Lodging (1= none 5=complete)
A635	5	√	7-4	5	2	2
CM7	8		6-20	5	2	2
CMV3	8		6-26	5	2	2
Co125	9	√	6-25	6	3	1
F7	8		6-24	5	2	5
H95	6		7-7	5	2	2
H99	7		7-1	5	2	1
I11-12E	9		7-12	6	3	2
Ind-B2	5	√	7-4	6	3	1
Ind-TR9-1-1-6	5	√	7-5	5	3	1
ND240	6	√	6-27	6	3	2
ND408	6	√	6-29	6	3	2
OH3167B	6		7-7	6	3	2
SD5	6		6-27	5	2	2
SD15	7	√	7-1	6	3	2
T8	9	√	7-13	6	3	1
T139	6	√	7-12	6	3	1
TX325	8	√	7-9	6	3	1
TX6252	6		7-11	6	4	2
V3	8		6-20	4	2	3
VA26	8		7-3	6	3	2
VA35	5	√	7-4	6	3	1
VA58	8		7-11	6	3	1
VA59	8	√	6-30	6	3	2
VA60	5	√	7-6	6	3	2
W59MHt	8		6-25	6	3	2
W117Ht	8		6-27	6	3	2
W629A	8	√	6-23	5	2	2

but several lines, scattered across maturity groupings and seed sources, did reasonably well. Since both plant breeders and geneticists may have an interest in inbreds able to better withstand extreme conditions, Table 1 summarizes the observations. (We obtained our materials, whenever possible, from the original source.)

M. M. Goodman and C. S. Stuber

Genetics of MDH

Analyses of crosses involving MDH variants have led to the conclusion that the inheritance of the mitochondrial MDH isozymes is largely governed by three nuclear loci (MdhA, MdhB, and MdhC in our temporary, laboratory designation), each with many multiple alleles. Hybrid dimers are formed between the allelic isozymes at each locus and between these three loci. The loci appear to be independent. Several of the hybrid isozymes formed as a result of interaction between various alleles at MdhC and common variants of MdhA and MdhB appear to be the actual basis for the hypothetical loci mdh1 and mdh3 of Yang et al. (P.N.A.S. 74:310-314, 1977). Our MdhA and MdhB appear to be identical to their mdh4 and mdh2, respectively.

It appears that a fourth locus, present in several inbred lines (Ky228, Mc5NT, Mo24W, W629A) and independent of MdhA and MdhB, affects the expression of all three of the mitochondrial Mdh loci (although not that of other allozymes associated with mitochondria), perhaps through a common effect on subunit structure, charge, or configuration or through a direct mitochondrial effect. When the allele involved is homozygous, each mitochondrial isozyme appears to migrate substantially further. In the heterozygous state, the effect is scarcely discernible.

Two nuclear loci govern the inheritance of the soluble Mdh isozymes, but less variation is present for these loci. They appear to be independent of the mitochondrial MdhA, MdhB, and MdhC loci, although the evidence in the case of one of the two soluble Mdh loci (MdhF) is much more convincing than the data accumulated thus far for the second (MdhD).

Our work has been based on coleoptiles homogenized in a sucrose-sodium ascorbate solution and run mostly with L-histidine-citric acid buffers ranging from pH 5.0 to pH 5.7. When ascorbic acid rather than its sodium salt is used in the homogenization process, the soluble MDH bands are selectively eliminated.

We have thus far demonstrated that at least 9 alleles exist at each of the MdhA and MdhB loci, including a "null" allele for each locus. Among U.S. materials, only 3 alleles are commonly found at either locus. Several commercial inbred lines are of special interest since they possess relatively rare allelic combinations. Ky21 and 4Co82 are each "null" for MdhB, the former possessing MdhA-1, resulting in a slow migrating allozyme; the latter having MdhA-10.5, which results in a fast migrating allozyme migrating close to the positions of the common soluble Mdh variants. H25 is "null" for MdhA but has MdhB-3 (equivalent to mdh2-m5 of Yang et al., 1977) and MdhC-18, an allele resulting in a very fast migrating band which almost reaches the edge of our 18.5 cm long, pH 5.7 gels. MdhC-18 is the less frequent of the two common MdhC alleles; we have rarely found three additional alleles at this locus among a wide array of Latin American collections. While we suspect that a "null" allele exists at this locus, we have not demonstrated that this is so.

The common, dark staining soluble MDH band is often found to be governed by the alleles of two loci. The most common variant (MdhD-12) of the darker staining of the loci is present in almost all common U.S. inbred lines except Tx303 and Tx325, which both have MdhD-14.5, a faster migrating variant, and which appear to be "null" for MdhF, the lighter staining soluble Mdh locus. Two additional rare variants of MdhD have been found among Latin American collections, but no null alleles have been detected. At present, it is uncertain whether the most frequent allele at the MdhF locus, which governs the lighter staining soluble MDH bands, is a "null" allele, MdhF-12, or MdhF-15, due to the almost universal occurrence of MdhD-12, which makes differentiation between MdhF-12 and MdhF-null difficult. Certainly MdhF-15 is not common among U.S. inbred lines, but is found in T232, F2 (a French line), Ill-12E (a component of the Stiff Stalk Synthetic), and Ill-13b (a sweet corn line). Three additional rare alleles of MdhF have been detected among Latin American collections.

By using trisomic stocks supplied by Dr. Lambert of the University of Illinois, we have been able to locate MdhB on chromosome 6. We were able to generate several trisomic plants having different sets of 3 different alleles at that locus. The assistance of Miss Wilma Hu, a research assistant of Dr. D. H. Timothy, Department of Crop Science, was essential to this work. We have yet to determine the chromosomal locations of the other Mdh loci.

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Resistance in sweet corn to Colletotrichum graminocolum

The inbreds PaIa453 ae du wx and PaIa5125 ae du wx were observed to differ in field reaction to Colletotrichum graminocolum (Ces) G. N. Nils., which causes anthracnose. PaIa5125 ae du wx appeared to have less disease. No clearly defined susceptible-resistant reaction was observed in either inbred. Therefore, resistance was defined as the relative production of lesions from a given concentration of inoculum. The objectives of this research were: 1) to determine if resistance could be distinguished on a basis of lesion production, and if so, 2) to determine the genetic basis of this resistance.

Experiments were conducted in the greenhouse and the field using both inbreds, the F_1 hybrid and 4 segregating populations. Inoculum was prepared from 8 isolates of C. graminocolum grown on PDA for 10 days. A standard inoculum concentration of 4.0×10^5 spores/ml was applied with a pressurized sprayer. The number of lesions was recorded on the 5th leaf of each plant in the greenhouse 10 days after inoculation. In the field, the number of lesions on the 6th leaf was recorded 10 days after inoculation. Results are presented in Tables 1 and 2.

Table 1. Mean and variance of the number of lesions and the number of lesions per dm^2 for inbreds, F_1 hybrid and 4 segregating populations is determined in the greenhouse.

	No. lesions		Lesions/dm^2	
	$\bar{x}$	σ^2	$\bar{x}$	σ^2
PaIa453 ae du wx (P_1)	80.3	3956.4	8.4	56.3
PaIa5125 ae du wx (P_1)	29.4	571.2	2.4	2.6
F_1	46.0	600.3	3.2	2.9
F_2	44.5	396.0	2.8	1.4
$P_1 \times F_1$	38.1	441.0	2.7	2.6
$F_1 \times P_1$	60.0	912.0	3.2	2.9
$F_1 \times P_2$	60.2	1036.8	3.9	2.9

Table 2. Mean and variance of the number of lesions and the number of lesions per dm^2 for inbreds, F_1 hybrid and 4 segregating populations as determined in the field.

	No. lesions		Lesions/dm^2	
	$\bar{x}$	σ^2	$\bar{x}$	σ^2
PaIa453 ae du wx (P_1)	139.7	12012.2	20.7	190.4
PaIa5125 ae du wx (P_2)	42.7	426.8	9.6	27.0
F_1	38.4	800.9	6.9	27.0
F_2	84.1	3956.4	15.6	171.6
$P_1 \times F_1$	75.1	3469.2	13.7	144.0
$F_1 \times P_1$	70.9	3981.6	12.5	134.6
$F_1 \times P_2$	105.9	3782.3	20.1	174.2

It appears that resistance determined on the basis of lesion production is present in the material tested (Tables 1, 2). A lower number of lesions was produced on PaIa5125 ae du wx when compared to PaIa453 ae du wx in both greenhouse and field inoculations. The F_1 responds similarly to PaIa5125 ae du wx in the field but appears intermediate in greenhouse inoculations. This may be due to fewer replications used in the greenhouse. Heritabilities were calculated for the segregating populations using the variance associated with PaIa5125 ae du wx as an estimate of environmental variance (Table 3).

Much of the variation in these traits appears to be of genetic origin. Based on these data this resistance may be controlled by as few as one gene. From the field data, a quantitative phenotypic response and genotype may be assigned to each inbred as follows:

Table 3. Heritabilities of the number of lesions and the number of lesions per dm^2 for 4 segregating populations inoculated in the field.[1]

	F_2	P_1xF_1	F_1xP_1	F_1xP_2
No. lesions	88.9	87.4	89.0	88.5
Lesions/dm^2	84.0	81.4	79.9	84.5

1. $\sigma_E^2 = 436.8$

PaIa453 ae du wx - A_1A_1; 139.7 lesions; 20.7 lesions/dm^2

PaIa5125 ae du wx - A_2A_2; 42.7 lesions; 6.9 lesions/dm^2

From this the mean number of lesions and mean number of lesions per dm^2 can be calculated for the segregating populations based on the proportion of genotypes present and assuming dominance:

	Genotype frequencies	No. lesions cal	No. lesions obs	Lesions/dm^2 cal	Lesions/dm^2 obs
F_2	0.25 (A_1A_1) + 0.75 (A_2-)	67.0	84.1	12.4	15.6
P_1 x F_1	0.5 (A_1A_1) + 0.5 (A_2-)	92.1	75.1	15.2	13.7
F_1 x P_1	0.5 (A_1A_1) + 0.5 (A_2-)	92.1	70.9	15.2	12.7
F_1 x P_2	1.0 (A_2-)	42.7	105.9	9.6	20.1

Other genes may influence lesion number in a quantitative manner of which only one was discernible in the material used in this study. It should be emphasized that plants exhibiting resistance as described here still sustain disease, but the reduction in inoculum efficiency compounded over several cycles of disease may be effective in preventing losses.

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Procedure for identification of publicly released sweet corn inbreds

Sweet corn inbreds released by breeders in the public sector will be designated by the two-letter postal abbreviation for the state in which the release is made followed by a number. Sweet corn inbreds released prior to Jan. 1, 1977 will retain their previous identification; however, the two-letter prefix may be added to sweet corn inbreds released before Jan. 1, 1977 that lacked prefix letters. To illustrate, sweet corn inbreds released by Illinois and Iowa, respectively, would be designated IL677 and IA5125.

This procedure was approved by the technical committee of USDA Regional Research Project NE-66. Members of the technical committee include a majority of sweet corn breeders in the United States. This procedure was adopted to prevent use of the same prefix by more than one state as has occurred in the past and to eliminate uncertainty concerning the state of origin that has occurred when sweet corn inbreds have been released with only numerical identification.

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A new paramutagenic allele at the R locus

In the 1975 Newsletter, occurrence of an aleurone pigment inhibitor which did not give Mendelian ratios was reported. It has since been determined that the plants generating this mutant phenotype had an ancestor from a common genetic stock at some point in their pedigree. In tests involving various sublines of this stock (A C R-g bz), colorless and mottled kernels segregated in the self-pollinated progeny of fully pigmented seeds. When several of the apparently colorless individuals (in a bz bz genotype, small bronze sectors on a colorless background are difficult to detect; therefore, some kernels which appear totally colorless may indeed be faintly mottled with bronze sectors) were selfed, mostly colorless offspring were produced along with a small number of fully pigmented and mottled kernels; there was no apparent ratio. By the second generation of inbreeding, all colorless offspring were obtained, some of which have bred true for two more generations. When a sample of these was testcrossed reciprocally to an r r stock, all progeny were colorless. In crosses of the colorless mutant with recessive stocks of all other aleurone color genes, pigmented offspring were produced. Thus, the mutant is an allele of R.

Among 14 sublines of the A C R-g bz stock tested, 12 generated colorless offspring in varying frequencies; the segregational patterns on the ears indicated that the event responsible for these mutants may be timed in terms of the development of the ear since in a substantial number of cases, the exceptional kernels arose predominantly on the lower half of the cob.

In order to examine the factors involved in generation of these R mutants, sibling plants of each of the 14 sublines were self-pollinated and crossed as females to a true-breeding colorless (mutant) line. Table 1 lists the results. The male parent in these crosses had no apparent effect on either the number of ears segregating for colorless or the frequency of colorless kernels. In family (subline) 4345, neither self-pollinated nor testcrossed ears produced any colorless while at the other extreme in family 4348, about 75% of the ears in each group segregated for the mutant phenotype. Among the remaining families also, the appearance of colorless seemed to be independent of the source of the male gamete.

On those ears which produced mutant kernels, the frequencies ranged from 0.2% to 68.1%. In any given family, there appeared to be no xenia but rather the range of colorless percentages was dependent upon the female parent and was characteristic for a given subline.

In all of the above tests, although the male parents were different, they arose from a common genetic stock. In one case when pollen from an unrelated A C R-r Bz stock was applied to silks of an A C R-g bz subline, the progeny segregated for self-colored, mottled and nearly colorless kernels. Extensive tests, however, have not been conducted to determine the effects of unrelated male parents on the production of colorless kernels in the A C R-g bz sublines.

Family	Total ears	self-pollinated					x colorless				
		No. of ears	No. of ears with colorless kernels	No. of ears without colorless kernels	% ears with colorless kernels	% colorless kernels-range	No. of ears	No. of ears with colorless kernels	No. of ears without colorless kernels	% ears with colorless kernels	% colorless kernels-range
4342	71	52	21	31	40.4	0.7-37.0	19	9	10	47.4	0.4-34.3
4343	62	41	21	20	51.2	0.3-68.1	21	16	5	76.2	1.4-40.2
4344	74	50	29	21	58.0	8.8-39.9	24	17	7	70.8	0.4-38.1
4345	67	48	0	48	0	0	19	0	19	0	0
4346	47	26	14	12	53.8	1.9-29.5	21	12	9	57.1	0.4-26.5
4347	60	39	8	31	20.5	4.0-29.1	21	5	16	23.8	0.9-26.8
4348	61	33	24	9	72.7	7.1-31.9	28	21	7	75.0	1.4-30.6
4349	16	12	5	7	41.7	14.4-33.6	4	4	0	100.0	0.3-36.8
4350	66	47	3	44	6.4	0.2-0.9	19	2	17	10.5	0.4-0.5
4351	61	31	19	12	61.3	0.4-46.8	30	20	10	66.7	0.5-34.0
4352	63	42	0	42	0	0	21	0	21	0	0
4353	58	37	7	30	18.9	13.4-21.8	21	9	12	42.9	0.9-21.4
4354-1	36	22	7	15	31.8	8.3-13.8	14	1	13	7.1	18.8
4354-2	75	45	22	23	48.9	2.7-38.3	30	15	15	50.0	11.9-35.9

Table 1- Distribution of colorless kernels on ears of A C R-g bz sublines self-pollinated and crossed as females to true-breeding colorless.

To determine whether there was an interaction between the newly arisen R mutants and the unrelated R alleles, true-breeding colorless lines were crossed as females with 12 full color stocks and the heterozygous progeny (all self-colored) backcrossed reciprocally to the colorless parent. One of the full color lines used was a W23 stock homozygous for an R-sc allele. On the ears resulting from the backcrosses of colorless females with heterozygous males in all cases except those involving the R-sc allele, there were few self-colored kernels on any ears and the pigmented ones ranged from deeply mottled to nearly colorless. The frequencies of the various color classes varied substantially; on some ears, the deeply mottled kernels comprised nearly 50% of the population while on others, the pigmented class was composed of mostly lightly mottled seeds with a few deeply mottled ones. As yet data on the precise frequencies of each class have not been compiled.

On the ears resulting from the backcross of the R-sc heterozygote as a male to the colorless line, self-colored and colorless kernels segregated in a 1:1 ratio but a few of the offspring on many ears were stippled. From the reciprocal cross of heterozygous females with colorless males, data so far are scant and no firm conclusions can be drawn.

The observations to date suggest that the newly arisen colorless mutant of R is paramutagenic. When it is heterozygous with a number of the R alleles tested, the pigmenting ability of that allele is reduced. The R-sc allele employed is not paramutable in this instance. Whether an inhibitor of R (I-R) is responsible for the few stippled kernels which arose in the backcross progeny of the R-sc heterozygote is not known. Stippled kernels have not been observed in the A C R-g bz stocks although stippling may not be distinguishable from light mottling in a bz bz genotype.

The original event responsible for the mottled and colorless kernels in the A C R-g bz sublines appears to be one which prevents the R allele from completely derepressing in the female. Reciprocal crosses between these stocks and an r

tester (and R deficiency) will provide better information regarding this question. Also, whether the R allele which mutates to colorless in the A C R-g bz stocks is paramutagenic in its pigmenting (derepressed) state or does not become so until after the mutational event has yet to be determined.

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The extent of sister chromatid cohesiveness in the centromere region of prophase II dyads

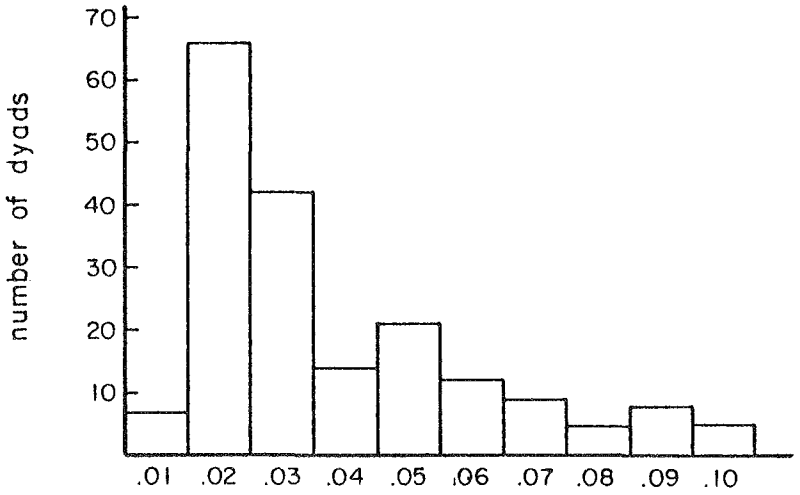
It has been suggested (M. P. Maguire, MGN 51:86-87, 1977) that maintenance of dyad integrity between anaphase I and anaphase II may depend upon the same mechanism as that which seems responsible for chiasma maintenance during the period between loss of the synaptonemal complex and anaphase I, namely sister chromatid cohesiveness. As observed cytologically at anaphase I in normal material, dyads are composed of four, usually highly condensed, chromatid arms which tend to diverge from tightly paired sister centromere regions. During interkinesis there is some decondensation of these arms followed by their recondensation during prophase II. During early prophase II in good preparations the four arms of each dyad can be traced.

If sister chromatids tend to cohere until anaphase II, except where they were separated at anaphase I as a result of crossing over, dyads would be expected to vary in their prophase II configuration in accordance with the position of their most proximal crossover in each arm. That is, a region of cohesiveness would be expected to extend from the centromere into each arm to the position of the most proximal crossover; an arm with no crossover should be cohesive throughout; a variety of complex configurations can be imagined for arms with multiple crossovers.

Camera lucida drawings were made of the dyads of 191 cells at early prophase II from four plants from diverse stocks (source indicated below). Measurements were made of the four arms of each dyad and of the regions where arms appeared to be cohesive. Where corresponding arms differed in length, the average length of the two was assigned to both. Total chromosome length was calculated for each cell; the extent of the apparently fused region at the intersection of the arms in each dyad (assumed to be the centromeric region) was divided by the total chromosome length of the cell in which it occurred. This operation was designed to give a value for extent of sister chromatid cohesiveness in this region adjusted for stage advancement, so that comparisons could be made among cells. The means and standard deviations for this value for the four plants are very similar:

		Extent fused region / Total chromosome length	
Plant no.	Stock	$\bar{X}$	s
825	Coop Inv 2a	.04	.0248
831	Maize chrom. 2-Tripsacum interchange stock	.04	.0250
841	Coop trisome 9	.04	.0204
885	Maize chrom. 2-Tripsacum interchange stock	.04	.0255

The overall mean of the extent of this fused region per dyad divided by the total chromosome length of the cell was .04 with standard deviation .0228, or an average of 20% per chromosome arm. Frequency distributions of classes of dyads with various extents of fusion/total length are shown in the following histogram.



Finally a rank difference correlation test of mean extent fusion versus total chromosome length of the cell was performed in search of a possible dependence of fusion extent on stage advancement. The rd from this calculation was -0.11 (not significant), suggesting no evidence here for such a dependence.

These results are not in accord with predictions of the hypothesis, if the genetic map is evenly distributed along the physical length of the chromosomes, and prophase II condensation is also even along chromosome length. In fact, if as usually anticipated, crossing over tends to be inhibited in centromere regions, the fit is even worse. If the hypothesis is to be seriously considered, the most reasonable speculation might be that sister chromatid cohesiveness tends to be lost to some extent proximal to crossovers during the anaphase I to prophase II interval.

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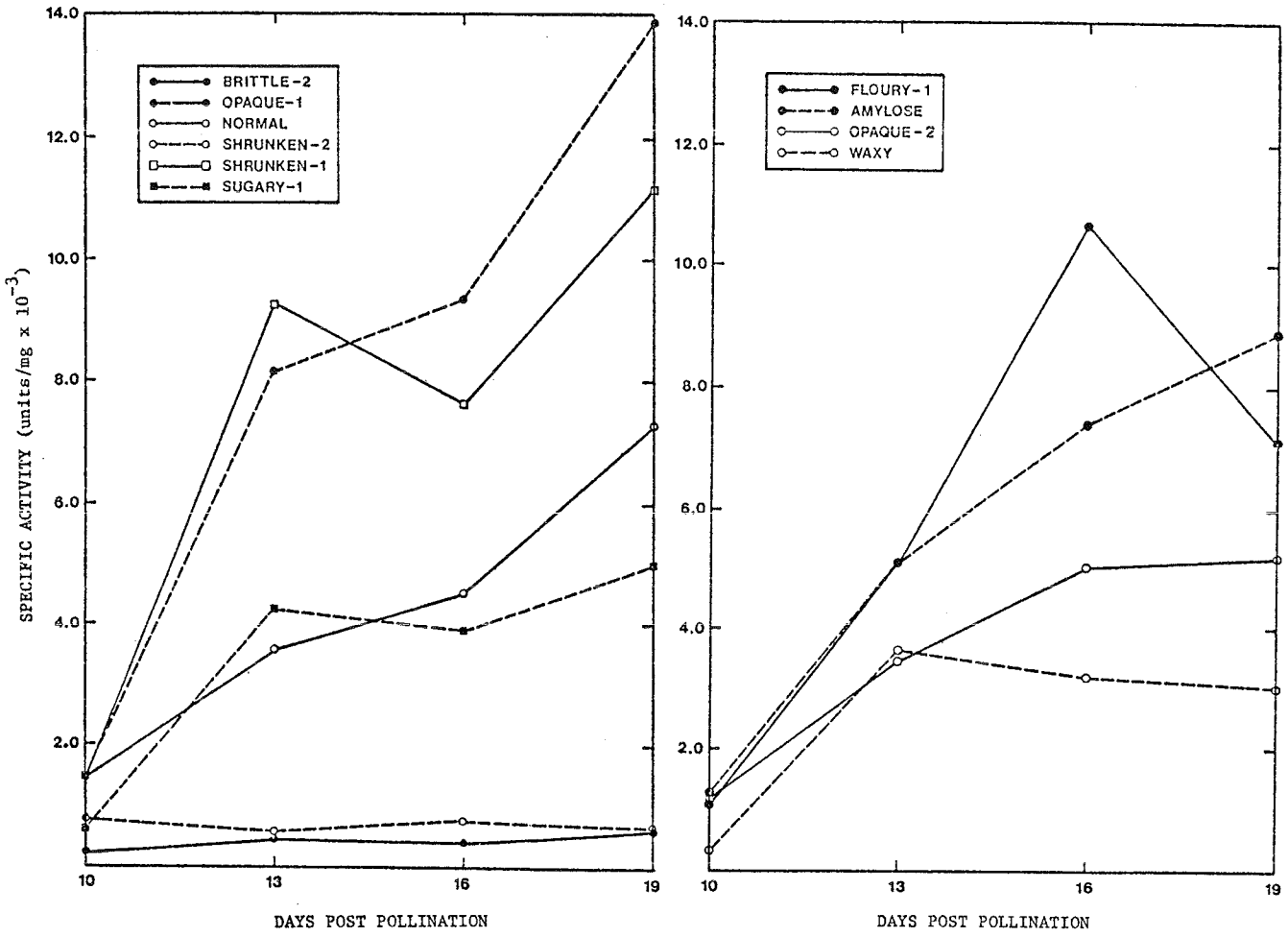
ADPGlucose pyrophosphorylase activity in various endosperm mutants

ADPGlucose pyrophosphorylase (E.C.2.7.7.9) catalyzes the conversion G-1-P + ATP $\longleftrightarrow$ ADPGlucose +PPi, which is the regulatory step in the formation of starch in plants. Activation by photosynthetic intermediates (3-PGA, fructose-1,6 diphosphate, fructose-6 phosphate) and inhibition by orthophosphate and ADP serve to control ADPGlucose pyrophosphorylase activity and, thus, the rate of starch accumulation in endosperm and leaf tissue.

The starch deficient mutants sh2 and bt2 represent two structural genes which code for ADPGlucose pyrophosphorylase in maize endosperm in maize endosperm (Hannah and Nelson, Plant Physiol. 53:297-302, 1975). The enzyme exists in two forms, A and B, in endosperm, which represent different aggregation states rather than different molecular species. The B form is a dimer composed of one subunit encoded by each structural gene while the A form is a tetramer composed of two subunits from each structural gene (Fuchs and Smith, Plant Physiol., submitted).

Comparisons of endosperm ADPGlucose pyrophosphorylase activities were made at 10, 13, 16 and 19 days post pollination among nine endosperm mutants and a

commercial hybrid. The mutants assayed were ae, bt2, fl, o, o2, sh2, su, and wx, while TX40 was used as the normal endosperm source. Ears were frozen when harvested and stored at -10 to -15 C until needed. The procedures for measuring ADPGlucose synthesis and isolation of ADPGlucose followed the method of Dickinson and Preiss (Arch. Biochem. Biophys. 130:119-128, 1969) as modified by Hannah and Nelson (Plant Physiol. 53:297-302, 1975) and Fuchs and Smith (Plant Physiol., submitted). One unit of enzyme activity is equal to 1 µmole ADPGlucose formed in 30 minutes. Protein was estimated by the method of Lowry et al. (J. Biol. Chem. 193:263-275, 1951) with BSA as the standard.



A survey of endosperm mutants assayed for total ADPGlucose pyrophosphorylase activity at 3 day intervals from 10 to 19 days post pollination.

The bt2 and sh2 mutants, which were known to affect ADPGlucose pyrophosphorylase, were the only mutants whose activity was significantly lower than normal (TX40) at all stages of development. In general, activity increased with age for the other genotypes, but appreciable variation in activity was observed over time. Opaque and sh had significantly higher activity than TX40, while su and wx were of interest since activity was essentially normal at 13 days post pollination but did not increase during the later developmental stages. Caution must be used in interpreting these data, however, as an inhibitor of ADPGlucose pyrophosphorylase

is present in the crude extract (Fuchs and Smith, Plant Physiol., submitted). Since these assays were run on crude extracts, it is not possible to conclude whether these observations represent differences in enzyme or inhibitor levels at this time.

James D. Smith and Roy L. Fuchs

Regulation of embryo growth by abscisic acid in vitro

The viviparous mutants of Zea mays are characterized by precocious germination 30-36 days after pollination, indicating a failure of the normal dormancy inducing mechanism. In addition to premature germination, all viviparous mutants cause obvious changes in seed and/or seedling phenotype. These pleiotropic effects allow the identification of homozygous viviparous seed on segregating ears to be made as early as 14 days post pollination.

Ears segregating for vp, vp2 and vp9 were harvested at various times from 14 to 30 days post pollination. Embryos were removed from surface sterilized seed and placed on Murashige-Skoog solidified agar medium with and without abscisic acid (ABA). A concentration of 3 mg% ABA was used since this completely inhibited growth of normal embryos. No other growth regulators were added. Explanted embryos were incubated for 20 days at 34C and 100fc illumination in a day:night regime of 12:12. Results are presented in Table 1.

Table 1. Growth of embryos cultured for 20 days with and without ABA in the medium

Genotype	Growth +ABA	Growth -ABA
vp-1/vp-1	+*	+
normal segregates	-	+
vp-2/vp-2	-	+**
normal segregates	-	+
vp-9/vp-9	-	+**
normal segregates	-	+

* vp-1 growth was retarded but not inhibited at 3 mg% ABA
** vp-2 and vp-9 seedlings are albino but grew at near normal rate.
- = no visible growth

Table 2. ABA content of 20 day old maize embryos as determined by gas chromatography

Genotype	ABA (mg) Bound	Free	Total	% of normal
vp-1/vp-1	0.008	0.021	0.029	90.6
vp-9/vp-9	0.003	0.009	0.012	37.5
normal	0.006	0.026	0.032	

In a second series of experiments, ABA levels of excised 20-day-old embryos were determined by gas chromatography. Preliminary results are shown in Table 2. Homozygous vp seeds were identified by their lack of anthocyanins in full aleurone color genetic background (A A2 C R). Homozygous vp2 and vp9 seeds were identified on segregating ears by their failure to produce endosperm carotenoids in a yellow (Y) genetic background.

Robertson (Genetics 40:745-760, 1955) grouped the viviparous mutants into two classes on the basis of their pleiotropic effects, but our preliminary data suggest that three, perhaps four, classes exist.

Class one mutants have normal carotenoid content in the endosperm and produce normal green seedlings and plants when homozygous. However, they fail to produce anthocyanin in the aleurone in the presence of the genes required for aleurone color. The vp mutant, which belongs to this class, produces normal amounts of ABA,

and growth of homozygous vp embryos in culture is not inhibited by ABA at concentrations that prevent growth of normal embryos. We suggest that class one mutants are defective for a specific ABA receptor.

Class two mutants fail to develop carotenoids in endosperm and leaf tissue, and the seedlings are albino when grown under normal illumination. These seedlings are not deficient for chlorophyll, but the chlorophyll is subject to photodestruction in the absence of carotenoids (Anderson and Robertson, Plant Physiol. 35:531-534, 1960). Our preliminary studies with two mutants of this class, vp2 and vp9, show that both are ABA deficient. Embryo ABA content of these mutants is 25-30% of that found in non-viviparous embryos of the same ears at various stages of development tested to date. In addition, the presence of ABA in the culture medium completely inhibits growth of vp2 and vp9 embryo explants. Since ABA and carotenoids have common precursers (Milborrow, Ann. Rev. Plant Physiol. 25:259-307, 1974, and Treharne et al., Phytochemistry 5:581-587, 1966) we suggest that the class two mutants will all be found to be deficient for ABA. The five mutants in this class (vp2, vp5, vp7, vp9 and w3) provide an opportunity to study ABA synthesis using a genetic dissection approach.

Class three mutants have pleiotropic effects on seedlings but do not affect carotenoids or anthocyanins in the seed. Two mutants (vp6 and vp8) fall into this category. While we have no data, as yet, regarding the response of these mutants to ABA, it is consistent with our model to propose that they may affect steps in ABA synthesis beyond the carotenoid branch point.

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Ellagic acid in maize

Ellagic acid is present in crude extracts of some of the maize stocks that we are analyzing. The stocks are of diverse origin, and we have not yet correlated the presence or absence of ellagic acid with any specific gene action. We have found it in several tissues, including anthers and pollen. It can be recognized on a chromatogram by its soft violet fluorescence in U.V., changing to very pale yellow on fuming with ammonia. Rf values are 0.27 in EtOH-HCOOH-H_2O (10:2:3) and 0.0 in 15% HOAc. Ellagic acid is an important constituent of tannins, and Bate-Smith has reported that it is frequently present in acid hydrolysates of the leaves of dicotyledons (J. Linn. Soc., Bot. 58:95, 1962) but that it is not present in monocotyledons (J. Linn. Soc., Bot 60:325, 1968). Its presence in some maize tissue crude extracts is therefore quite interesting.

Oldriska Ceska

Multiple gene combinations - flavonoid factors

We are attempting to derive stocks in which all the genetic factors involved in flavonoid synthesis and distribution are of known origin. We are concentrating in particular on 14 loci:

A, A2, A3, B, Bz, Bz2, C, C2, In, P, Pl, Pr, R, and Sm,

and on variants at these loci that are distinguishable by some visual or chemical means. We are interested in determining the extent to which variants of one locus can affect the expression of other loci, and we hope also to find other, as yet unrecognized, genetic factors involved in flavonoid synthesis. The number of different stocks that we could theoretically derive is astronomical, thus it may

take a while to get all the combinations that we would like to have. By next year we hope to have a list of those combinations already made, for those who may like to have some seed. They will not necessarily be in any particular inbred background, however.

E. Derek Styles

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Isolation and recovery of protoplasts from young leaf tissue

Working from the literature, we have obtained high yields of viable protoplasts using the following scheme with single cross hybrid maize, grown under standard greenhouse conditions.

At 21 days after seeding, plants are placed in a dark humid chamber for 16 hours.

Unrolled leaves surrounding the growing point are carefully dissected out, and leaf sections are floated on a solution of: 0.5% Onozuka R-10, 9% mannitol, and salts (Zapata et al., Plant Sci. Lett. 8:119, 1977) at pH 5.5.

The solution is shaken at 125 rpm for 2.5 to 3 hours at 23 C, and leaf sections are separated from the enzyme solution containing the protoplasts by passage through a double layer of cheesecloth.

Additional debris is removed by passage of the solution containing the protoplasts through an 85μ nylon filter, and protoplasts are collected by centrifugation at low speed (300xg for 5 minutes).

Protoplasts are washed twice in Zapata salts, 9% mannitol, and the final pellet is resuspended in the same solution.

This scheme yields 1×10^7 protoplasts/gm fr. wt. of tissue. This yield compares favorably with earlier reports of protoplast yields from maize: 2.5×10^4 protoplasts/gm (K. L. Giles, Plant Cell Phys. 15:281), 2×10^6 protoplasts/gm (H. C. Aldace et al., Tissue and Cell 9:167, 1977).

Gail Meadows and D. B. Walden

Shoot tip culture: Possibilities for the propagation of clonal lines

Shoot tip culture is used extensively to propagate clones of ornamental plants and a few crop plants. Its use in grass crops appears untried, but because it could be a source of more stable lines than through callus cultures it deserves some attention. The procedure utilizes the fact that stems possess axillary buds which, following release from apical dominance, can produce new shoots. These can then be used to generate secondary axillary buds. A scheme for such procedure is illustrated on the following page.

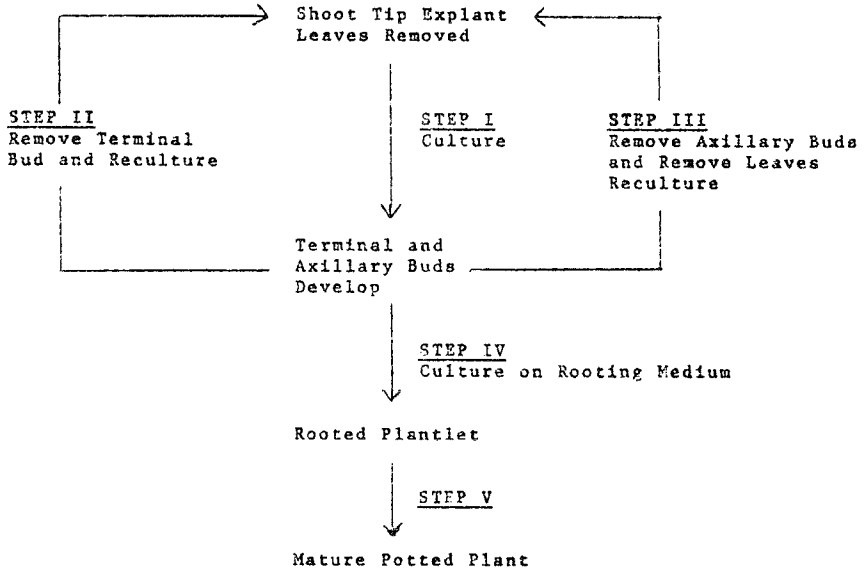
Some preliminary tests to determine how corn would respond in such a culture protocol generated the following observations:

Step I: Young 10-20 day shoot tips with all leaves removed possessing up to 10 axillary buds plus a terminal apex can be successfully cultured on appropriate sterile media. Axillary buds develop profusely and can be subcultured (Step III). Variations in this response appear to be stock-related and decidedly media-related.

Step III: Successful culture of the primary axillary buds is now routine though as yet second-order axillary buds have not been successfully subcultured.

Step IV: Roots do not initiate on the media used in Step I. Therefore a transfer is required. Apparently, the temperature and day length conditions are important for this step.

Step V: To date 6 plants derived from axillary buds have been successfully potted. We are following their development.



Despite the limited success of this procedure, a number of problems remain. For example, axillary buds in corn are probably not easily diverted from their normal developmental pattern as is the case in other plants. Axillary buds, at least by the 20-30 day stage, are probably differentiated as tiller, ear-shoot or some intermediate condition. The reversion of these buds to freely leaf-initiating meristems may prove difficult.

K. Raman

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Identity and sources of a sugary enhancer gene significant for sweet corn quality

Tests with sweet corns from two different sources have led to the identification of a gene, new to the maize list, that is of special interest because of a favorable effect on table quality. One source of the gene was two commercial hybrids, Tendertreat E. H. and Kandy Korn E. H. (E. H. = Everlasting Heritage), and the other was the Illinois sweet corn inbred line 677a. The gene is termed sugary enhancer and is tentatively symbolized se.

Outcrosses of the above three sugary (su) stocks were made to three dent corn inbred lines (182E, W22, W23), three popcorn inbred lines (Sg 18, I20, I53) and the flint variety, Canada Longfellow. Selfs and testcrosses to Ill.677a and also light yellow endosperm derivatives of Tendertreat E. H. (TT) and Kandy Korn E. H. (KK) of the resulting hybrids showed that Tendertreat E. H. and Kandy Korn E. H. were heterozygous and Ill. 677a was homozygous for the se gene. An overt phenotypic effect of se in homozygous condition is dilution of yellow pigmentation in the endosperm. The gene when homozygous does not change kernel form, but preliminary estimates indicate that it reduces kernel size about 10%. The diluting action of se on yellow endosperm varies considerably according to the genetic background. The kernels on segregating ears derived from crosses of TT, KK, or Ill.677a to one or another of the popcorn strains Sg18, I28, and I53 were distributed between two fairly well defined classes, dark and light yellow endosperm. These matings regularly gave the results expected if the light yellow phenotype was homozygous for a recessive gene (se) that assorts independently of the sugary (su) factor. It was noteworthy that the results from se/+ heterozygotes

involving the Ill.677a line as a parent in the outcrosses paralleled those from se/+ plants derived from the corresponding matings of the popcorns with TT and KK.

Three examples of the seed distributions observed following Su Se/su se x su se matings are:

	Non-sugary		sugary	
	Dk yell (Su Se)	Lt yell (Su se)	Dk yell (su Se)	Lt yell (su se)
(W22/Ill.677a) x TT Lt.	43	30	47	54
(I53/Ill.677a) x KK Lt.	35	28	44	43
(Ill.677a/Sg 18) x Ill.677a	32	32	39	40

The dark and light yellow kernel classes in populations resulting from outcrosses of TT, KK, and Ill.677a to the three dent inbred strains used, and also to Canada Longfellow, rather than to the popcorns, often were weakly defined or overlapping. Thus, the se gene frequently does not give an easily perceived phenotypic effect.

Light yellow endosperm derivatives from selfed TT or KK plants regularly gave only light yellow offspring when crossed with each other and also when outcrossed to the Ill.677a line. These matings provide confirmatory evidence that the Tendertreat E. H. and Kandy Korn E. H. commercial hybrids and the Ill.677a inbred strain carry the se gene in common.

Circumstantial evidence might suggest that the mutant gene here termed sugary enhancer (se) falls in one or another of the already established "soft" endosperm categories, such as floury or opaque. The se gene, for instance, dilutes yellow endosperm pigmentation, like floury-1, floury-2, and the several opaque factors. Furthermore, J. W. Gonzalez, A. M. Rhodes and D. B. Dickinson (Plant Physiol. 58:28-32) report that one of the three stocks in the near ancestry of Ill.677a which, as noted above, is homozygous se, was Bolivia 1035, a Carioco floury corn. The present writer's tests show, however, that Ill.677a, TT, and KK do not carry a gene which, in the absence of sugary, gives either a floury or an opaque phenotype.

TT, KK, and Ill.677a were pollinated with pollen from a floury-1 line. The endosperms of the resulting kernels regularly were translucent, i.e., non-floury. Non-floury and floury seeds were present in equal numbers in the F_2 offspring of these hybrids as expected from floury heterozygotes. In contrast, the F_2 populations derived from crosses between light yellow derivatives of TT, KK or Ill.677a and the popcorn inbred lines Sg 18, I28, or I53, although segregating for dark and light yellow endosperm, were non-floury throughout. These observations show that TT, KK, and Ill.677a do not carry floury-1, nor any other floury (or opaque) gene that regularly conditions soft endosperm. The evidence does not exclude the possibility, however, that in certain uncommon genetic environments, such as that of Bolivia 1035, for example, the se gene might yield a recognizable floury phenotype. Decisive evidence on the question whether se represents a new locus or is a novel type of allele at an already known locus must await the results of chromosomal site studies. The floury-2 (fl2) locus cannot be the se site because se assorts independently of su, whereas fl2 and su are linked with about 8% recombination between them. Provisional application of the symbol se to the sugary enhancer assumes that the gene resides at a previously unmarked locus.

Gonzalez, et al. (loc. cit.) found that Ill.677a kernels have an exceptionally high sucrose content at 18 days after pollination. The sucrose level was comparable to that in shrunken-2 (Supersweet) at this developmental stage. Unlike the effect of the sh2 gene, however, the high sucrose content was not associated with a large reduction in water soluble polysaccharides (phytoglycogen). We have

observed that a high level of sweetness is characteristic of light yellow endosperm derivatives of Tendertreat E. H. and Kandy Korn E. H. also, but not of the dark yellow derivatives, of these se/+ plants at 20-25 days post-pollination. Thus increased sweetness appears to be a characteristic effect of the sugary enhancer gene in sugary kernels.

Hybrids between Ill.677a and light yellow derivatives of TT or KK (which produce only light yellow kernels) were found to be outstanding in table quality. Palatability of these se se homozygotes substantially exceeded that of Tendertreat E. H. and Kandy Korn E. H. (se/+ heterozygotes) in the judgment of several persons. The superiority is associated not only with greater sweetness but also with a certain "crispness" of endosperm texture not characteristic of currently grown sweet corns.

I am indebted to Bruce Ashman, Robert I. Brawn, W. C. Feyerabend, and Stuart Smith for seed used in this study.

R. A. Brink

Genes that retard water loss in maturing sweet corn kernels

The water content of sweet corn at the time it is harvested for fresh use or preserving (17-24 days after pollination, depending on variety and temperature) is near 70%. Slow-drying is one of the important effects of the sugary-1 (su) gene as seed development advances. The soft, fully distended condition of the su kernel when it is most desirable for use as food is transitory. The seed later becomes firm and wrinkled, and eventually, in the ripe ear, hard and glassy.

Mutant genes that intensify the water retaining effect of the su factor, and thus prolong the harvesting period, are of interest as a possible means of significantly expanding the food uses of sugary corn. The writer has given special attention to three genes among the several that alter sugary-1 expression in this way, namely, floury-2 (f12), opaque-7 (o7) and, as noted in the immediately preceding news item, sugary enhancer (se).

Quantitative data on the retarding effect of the f12 gene on water loss in sugary and non-sugary kernels at Madison, Wisconsin, in 1977, are presented in Figure 1. In this experiment su f12 and su F12 plants derived from hybrids between W64A f12 and Seneca Chief, a commercial sweet corn hybrid, were crossed with W64A f12 and the standard dent corn inbred line W64A (F12), respectively. The su f12/W64A Su f12 hybrid was then pollinated using pollen from a closely related su f12 stock, and the su F12/W64A Su F12 hybrid was pollinated with su F12 pollen of similar derivation. Thus about 3/4 of the residual inheritance carried by the resulting four classes of kernels, Su F12, Su f12, su F12, and su f12 was derived from the W64A inbred strain, and the balance from Seneca Chief. Collections for moisture determination were begun at 30 days following pollination, the earliest time at which the Su and su kernels could be clearly distinguished from each other

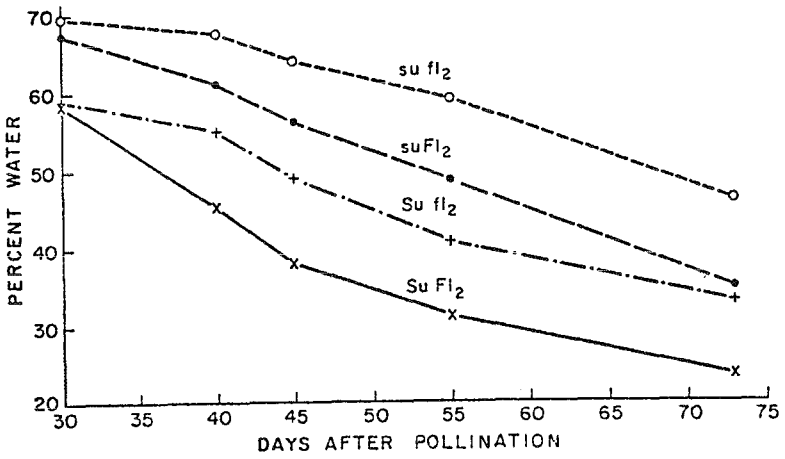

Figure 1. - The percent water in the four classes of kernels resulting from otherwise comparable (su fl_2/ Su fl_2)♀ × su fl_2 ♂ and (su Fl_2/ Su Fl_2)♀ × su Fl_2 ♂ matings.

on the ear. Weights of fresh and oven-dried kernels were taken on two independently collected 100-kernel samples (each based on 4 ears) of each of the four classes of seed, at 30, 40, 45, 55 and 73 days after pollination.

The retarding effect of su on water loss is shown by the positions of the Su f12 and su f12 entries in Figure 1. At 55 days after pollination, for example, the su seeds contain 17.7% more water than their non-sugary counterparts. A large difference (11%) persists even to 73 days.

The water content of Su kernels is already about 10% lower at 30 days than that of su seeds, as shown by the points of origin of the four lines in Figure 1. In contrast, the differentiating effect of f12 in this respect is little expressed until after 30 days. The f12 gene alone however, strongly retards water loss. At 73 days after pollination the moisture content of floury-2 (Su f12) kernels is 33.2%, a value which is about the same as that of sugary (su F12) kernels (34.5%) at this stage.

Especially noteworthy is the fact that f12 interacting with su holds the kernel water content above 60% up to 45 days after pollination. This means that, insofar as moisture content at the picking stage determines the suitability of sweet corn for food, f12 in conjunction with su extends the harvesting period to 40 days or more, and thus well beyond the upper limit for ordinary sweet corn of about 25 days.

Numerical data have not been taken on the effects of the su o7 and su se genotypes on water loss from maturing kernels. Field observations indicate, however, that the retarding action of both o7 and se are pronounced. Furthermore, J. W. Gonzalez, A. W. Rhodes and D. B. Dickinson (Plant Physiol. 58:28-32) report that I11.677a sugary seeds which, as shown in the preceding news item, are homozygous for the sugary enhancer (se) gene, tend similarly to retain moisture as development advances. Older data show that the waxy gene (wx) in combination with su, in contrast, has only a small effect on water retention in the maturing seed (R. H. Andrew, R. A. Brink and N. P. Neal, J. Agric. Res. 69:355-371).

The marked increase in toughness and resistance to mastication of the pericarp that occurs from about 20 days post-pollination onward need not be a deterrent to using more mature sweet corn for food. The kernels on older husked ears can be scarified at the crown with a sharp instrument and the endosperms and embryos can then be squezed out with a suitably shaped hand tool, leaving much of the pericarp on the cob. This operation can be carried out efficiently, however, only on kernels that are soft and fully distended. The writer has processed ears from su f12 and su se plants in this way up to 40 days after pollination without difficulty. The kernels on ordinary sweet corn, on the other hand, are too firm at 40 days to permit this operation. The procedure whereby endosperm and embryo were separated from pericarp is similar in certain respects to the factory process whereby cream-style corn is prepared for canning, and so lends itself to mechanization. A desirable departure from the usual commercial procedure, however, is scarification of the crown instead of cutting of the upper part of the kernel with its tough outer covering, before squeezing out the embryo and remaining endosperm.

Freeing sweet corn from the limitations as a food imposed by a pericarp that becomes coarsely fibrous and tough as kernel development advances is a useful gain in itself. Greater flexibility in time of harvesting is thus made possible. The amount and character of the food products obtainable also are expanded. Mention is made in the immediately preceding news item that certain sugary-sugary enhancer (se se) homozygotes, for example, were of excellent table quality when harvested at about 20 days after pollination. A different product is forthcoming at later developmental stages. Preliminary trials show that canned or frozen corn from su f12 or su se plants picked at 40 days and processed so as to leave much of the pericarp on the cob was less sweet than corresponding 20-day corns but had a pleasing texture and mellow taste. It is evident from the results of these tests

that incorporation in the sugary-1 genotype of one or another gene, like sugary enhancer, opaque-7, or floury-2, might significantly expand the range of nutritious and palatable food products that can be obtained from corn.

Attempts to broaden the culinary use of sweet corn by incorporating in the su genotype a second mutant that significantly delays water loss from the endosperm during kernel maturation encounters important mechanical and biological production problems. These include injury during machine harvesting and processing of the seed and fitness of the double mutants to meet the various challenges of the external environment. Perhaps the seed injury problem can be made manageable by the development of inbred lines with husks loose enough during ripening to facilitate more rapid field curing before machine picking.

Major components of the fitness system in sweet corn relate to seed germination and seedling establishment and reaction of the plant to ear and stalk rotting organisms.

The three mutants mentioned above, sugary enhancer, opaque-7, and floury-2, in homozygous condition reduce mature seed weight and, when in combination with the sugary gene, impair germination and seedling development. The su gene alone lowers mature seed weight about 20%, as compared with Su kernels on segregating ears. The results of a 1977 experiment on the weight of mature seeds from selfed su/+ and su fl2/+ plants in which the normal (+) parents in each set were nine inbred dent corn strains are instructive in showing the adverse effect of the fl2 gene on sugary kernel development. If the mature "weight index" of the +/+ (i.e., non-sugary, non-floury-2) kernels on segregating ears is taken as 100, then the mean weight index for the su/su (non-floury) seeds was 80.6 and that for su fl2/su fl2 kernels was 60.9. (The su and fl2 genes are linked with about 8% crossing over between them. No adjustment in calculating the weight index was made for the recombinants, so the 60.9 value is a maximum).

It was found that the fl2 gene alone on the background of W64A, a dent inbred line, reduced mature seed weight about 20%, as was the case also with the su gene alone, as noted earlier. Thus the adverse effects of the su and fl2 genes on mature seed weight are additive. Reduction of the seed weight index from 80 for the su or the fl2 gene alone to about 60 for the su fl2 double mutant results in a mature, dry kernel that borders on chaffiness in some genetic backgrounds. The yield of edible products would be expected to decline accordingly, and seed germination and plant establishment would be less reliable. These results make it doubtful that homozygous su fl2 strains would be economically viable.

Tests comparable in scale to those of fl2 on seed weight, in combination with su, have not been made with opaque-7 and sugary enhancer. Preliminary observations indicate, however, that whereas both these genes reduce the dry weight of mature sugary seeds, the net effects in this respect are significantly less than that of the fl2 factor.

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The cytological basis of the failure of non-disjunction associated with a B-10 translocation

During a routine propagation (r-g x r-g 10^{B} B10 (R-scm) B^{10} (R-scm)) of translocation TB-10(30), an ear with an unusual segregation pattern was observed. It contained only two seed classes (colored and colorless) instead of four as found

on the sib ears (Table 1). The two missing classes are the ones with discordant embryo and endosperm phenotypes, which are the descendants of pollen grains whose B^{10} undergoes non-disjunction during the second pollen mitosis. It is speculated

Table 1. Segregation of seed types following r-g r-g x r-g 10^B B^{10} (R-scm) B^{10} (R-scm) crosses (where 10^B and B^{10} are chromosomes of TB-10(30)).

		Number of kernels			
Progeny identification	Endosperm: Embryo:	r* r	Scm Scm	Scm r	r Scm
Normal ear		61	57	67	118
Exceptional ear		26	199	0	0

*"r" designates absence of anthocyanin (endosperm or embryo) and "Scm" designates anthocyanin pigmentation conditioned by R-scm (endosperm or embryo).

that B^{10} in the paternal plant of this exceptional ear had lost its non-disjunctional activity. Furthermore, the fact that both B^{10}'s of this plant were unable to non-disjoin, having been able to in the previous generation, suggests 10^B as responsible for such failure. This conjecture is substantiated by the fact that B^{10} non-disjunction was restored after incorporation of intact B's into the plants growing from the seeds of the exceptional ear.

The failure of 10^B to promote the non-disjunction of B^{10} may be due to a structural mutation that occurred either prior to or after the second pollen mitosis. According to the first explanation, 10^B would have been defective ever since or shortly after it was isolated. Its defectiveness had not been identified due to the presence of intact B's in the same stock. When B's disappeared as a result of successive test-crosses and preferential fertilizations toward the endosperm, B^{10} became unable to non-disjoin. The latter model predicts that 10^B possessed the ability of inducing B^{10} non-disjunction prior to the second pollen division but it changed its structure and lost such ability afterwards. Results of the following experiments are in agreement with the first explanation:

(1) Chromosome counts of root tips from the colored seeds on the exceptional ear gave an expected number, 13 out of 14 being 21 and one being 20, while the counts from seeds with colored embryo but colorless endosperm on the normal sib ears are unexpected, 15 out of 33 being 22, 16 being 24 and the last two being 25 and 27, respectively. Since their karyotype is supposed to be identical to the paternal parent (namely 10 10^B B^{10} B^{10}), the expected chromosome number is 22. Without exception, the extra chromosomes are telocentric.

(2) Test-crosses (r-g r-g x r-g 10^B B^{10} (R-scm) B^{10} (R-scm)) of seeds with colored embryo but colorless endosperm from two normal sib ears gave nearly 1:1 (33:27) proportion of normal (same as the paternal ear) and abnormal (same as the exceptional ear) ears. This ratio is not unexpected if the paternal parent contained two B chromosomes. Each derived 10^B B^{10} microspore would carry a single B which would undergo non-disjunction at the second pollen division as did B^{10}. The resulting two B^{10}'s and two B's would then move with 50% chance to the same or different poles. The former yields a sperm which possesses both B's and B^{10}'s and fertilizes preferentially the egg; the second sperm, devoid of both chromosomes, joins with the polar nuclei. The latter results in two sperms which carry either two B's or two B^{10}'s and conjugate with the egg randomly (Carlson, 1969, Genetics 62:543-554). As a consequence, a little more than 50% of the 10^B B^{10} B^{10} embryos

would contain two B's and the remaining embryos would not. Following test-crosses as staminate plants, the former would give rise to the normal ears and the latter to the abnormal.

(3) Nine seeds with colored embryo but colorless endosperm on a normal sib ear were germinated, root tips were excised, and the seedlings were transplanted to the field and then test-crossed on r-g r-g plants at maturity. Four of these contained 22 chromosomes and gave rise to abnormal ears similar to the exceptional ones. Four others possessed 24 chromosomes, all of which resulted in normal ears. The last one had 23 chromosomes and its test-crossed ear was likewise normal but the frequency of discordant seeds was only 60% of the regular normal ears.

The cytological analysis of 10^B structure yields an interesting result. Assuming one breakage on the B long arm, as in most of the other 37 B-10 translocations isolated by the writer, the pachytene figure of 10 10^B B^{10} tassels should contain a trivalent synapsing in a T-shaped configuration. Pairing on two of the arms of this configuration should be complete because they represent the homologous pairing between the 10 portions of TB-10(30) and chromosome 10. Pairing on the third arm, however, would not be complete since this consists of two non-homologous B portions. The B portion associated with B^{10} consists of the short arm, the centromere and the proximal end of the long arm. The B portion born on 10^B includes the distal end of the long arm. The pairing pattern observed contradicts this expectation. The three arms of the T-shaped trivalent paired perfectly. As was assumed, the break on B is on the euchromatic region of the long arm close to the heterochromatic block and that on chromosome 10 is at about 0.12 10L. B^{10} possesses an expected structure, but, surprisingly, 10^B carries the same B fragment as B10, including the short arm, the centromere and the euchromatic region of the B long arm.

This cytological picture furnishes several interesting views:

(1) It provides an answer to the question why 10^B cannot promote B^{10} non-disjunction. 10^B lacks the distal tip of the B long arm which is essential for B non-disjunction (Ward, 1973, Genetics 73:387-391).

(2) 10^B is a dicentric chromosome, containing the B and 10 centromeres.

(3) Since 10^B carries the same B portion as B^{10}, it may be able to non-disjoin as does B^{10} in the presence of intact B's. Yet, the preliminary test of this possibility gave a negative result. A more critical test is being currently undertaken.

(4) 10^B may originate from either the effect of x-irradiation during the initial isolation of this translocation or as the result of non-homologous crossing-over between proximal and distal portions of the B long arm. Study of these two possibilities is underway.

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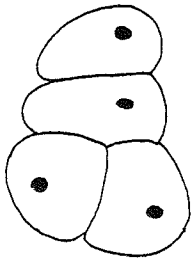
Induction of meiotic mutants by EMS

A search for meiotic mutants is being undertaken among plants of the M_3 generation following treatment of pollen with EMS. To date, sporocytes of 27 mutants which exhibited ovule abortion and/or abnormal kernel development have been examined. Of these, at least two appear to be new mutants.

One affects meiosis in several ways and is variable in its expression. Two or four univalents are sometimes present during diakinesis, prometaphase I and metaphase I. Whether the univalents result from the failure of homologous chromosomes to pair or from desynapsis is not yet known. Lagging chromosomes (usually 1



nucleolus

Figure 1

Figure 2

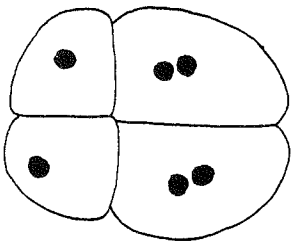


Figure 3

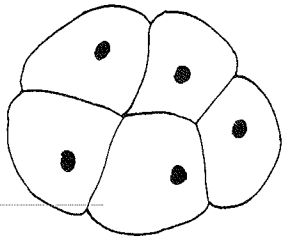


Figure 4

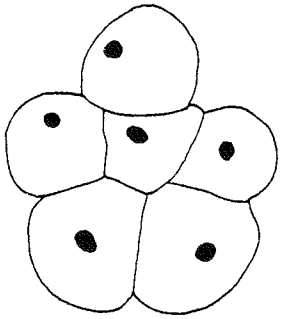


Figure 5

or 2, but sometimes 3 or 4) have been observed at both meiotic anaphases and telophases.

Occasionally, the axis of one or both of the metaphase II spindles is oriented in the same direction as, instead of at right angles to, that of the metaphase I spindle. As a result, quartets of the type shown in Figure 1, or linear quartets (Figure 2) are formed. Some quartets contain three normal microspores and one without a nucleolus, or two binucleolar microspores and two without nucleoli. Other quartets contain one, two, or more apparently normal nucleoli in addition to the four which are usually present (e.g., Figure 3). Clusters of five to eight microspores which have arisen from a single sporocyte are frequently observed (Figures 4 and 5). In some clusters, each cell has a nucleolus (Figures 4 and 5) while in others some cells may lack a nucleolus. The cells comprising the abnormal quartets and groups of more than four microspores are often unequal in size (Figures 2, 3, 4 and 5). Pollen and ovule abortion and the proportions of normal and defective kernels on ears are variable.

The presence of more than four nucleoli per quartet or group of microspores suggests that an extra replication of the nucleolus organizer, and possibly of the whole chromosome complement, has occurred at some stage. Attempts to determine whether the extra cells in clusters such as those in Figures 4 and 5 arise from supernumerary mitoses and if so, whether each cell receives a complete set of chromosomes, have been hampered by a tendency of cells which are near the end of the second meiotic division to fall apart when squashes are made; it may be necessary to resort to sectioning in order to examine these stages more fully. No mitoses have been observed during or after the late quartet stage. It has also still to be established whether there is more than one mutation affecting meiosis in the mutant stock or not.

The second mutant has not been studied in detail. It is completely male sterile and has poor seed set. Meiosis proceeds normally through leptonema to the early synizetic stage. Thereafter, a long stage follows during which the chromosomes become indistinct and appear to fuse with the nucleolus. Tassel samples with later stages are not yet available. The meiocytes may degenerate or they may undergo a modified type of meiosis similar to that shown by sticky chromosome homozygotes; however, there are features which differ from Beadle's description of the latter mutant.

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*The courtesy of the Department of Genetics and Development during a sabbatical tenure at the University of Illinois is greatly appreciated

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During summer 1975, 1,050 kernels from an unmarked cannister, assumed to contain "airline" corn (dc-8), were planted in an experimental plot in our Newfoundland research area. From onset of germination, however, it became apparent that the assumption was unfounded, as the growing stalks did not display the characteristic rounded fuselage or silver color. Following appearance of third and fourth leaflets the whole crop disappeared, although all plants had appeared highly viable and disease-resistant. It was tentatively concluded that the crop contained the lethal mutant disintegrating (dig) until an irate farmer 5 miles away called the experiment station to report his field of soybeans had 1,050 hardy corn plants growing there. Subsequent calls from a geographic area leading back to the station indicated the errant maize had traversed a hemizaggous route at 2-day intervals prior to rooting in the soybeans.

The crop was transferred back to the experiment station medium using the method of Cope et al. (MNL 01: 1-2, 1000 B.C.). A 20-foot barricade with #25 steel-gauze canopy was used to contain the plants while admitting proper light (1 roamin' candle) and ventilation. It was noted at first tassel that plants would, overnight, disengage primary roots and, whirling upper leaves, fly through the field to re-engage at new terminals (due to this propensity, keeping accurate pedigree numbers was impossible; however, all plants appear to be homozygous and display the same responses).

This corn was finally determined to be the outcross of airline corn x maize bearing the galloping gene, gap. Because of ear length and row number, it is now known that the female parent was twa-747 rather than dc-8 as originally suspected. The strain is now designated helicopter corn because of its mode of travel. 500 kernels were requisitioned by the Dept. of Defense for further study. Sample stock is available through any travel agency for anyone who can catch the plants.

M. U. Thant*
*Formerly your devoted typist, MN

ADDENDUM

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Studies on R-nj

Variability in the expression of the navajo gene in the R-nj:Illinois isolate in stock 2 background was examined. There were wide variations in the extent of anthocyanin pigmentation and eleven different types of expression ranging from "silk scar color only" to "almost whole kernel colored" were recovered. Progeny testing of the isolated phenotypes revealed that the variations in the R-nj expression are not heritable and are due to developmental conditions. A kernel with the minimum amount of aleurone pigmentation may, in the next generation, yield the whole spectrum of variations.

The R-nj gene showed dosage effects on aleurone pigmentation. Homozygous R-nj stock with three doses of R-nj in the triploid aleurone is uniformly and more intensely pigmented than kernels with two and one dose of the gene in heterozygous combination with r. Extent of pigmented area in the aleurone was found to be extremely variable in all three R-nj dosages. The effect of the heterochromatic knob (K) on the same arm of the chromosome, on the intensity of color and extent of pigmentation was also studied, but no detectable difference was observed for the presence or absence of K.

When R-nj was crossed reciprocally with marbled (R-mb), another pattern allele, and the F_1 testcrossed to r, an interesting situation was noticed. There is no dominance relationship between the two and both the phenotypes are expressed on the kernel. However, when the female carried the R-nj allele in homozygous or heterozygous condition, the majority of the kernels on crossed ears showed R-nj phenotype regardless of whether R-nj was present in the endosperm tissue or not. The situation suggests a pre-setting effect of the R-nj allele implying that the developmental information is pre-programmed in the mother and passed on to the offspring.

Recombinational analysis of the R-nj allele carried out with the help of a proximal marker, g (golden plant color), and a distal marker, K (heterochromatic knob), and one contamination marker, y (white kernel), revealed that R-nj represents a compound form with two closely linked but separable components, the self-color (Sc) component responsible for pigmentation of the whole kernel, and the navajo (Nj) responsible for restriction of aleurone color to the crown region of the kernel. Nj gene is most probably regulatory in nature controlling onset, termination, or period of Sc action. Sixty-four putative self-color recombinants were obtained from screening of 65,119 kernels from 430 ears of the testcross G R-nj K/g r-g k. Progeny testing of these suspected cases revealed that four were genuine self-color recombinants involving crossing over between G and K and also between silk and anther color and seed color. The probable gene order of this region was G Sc Nj K. In another testcross involving the mutable R-nj (R-nj Mp), two genuine self-colored exceptions were isolated from among 4,246 kernels. This further confirms the compoundness of the R-nj allele.

D. Kumar and K. R. Sarkar

Reversed germ orientation

A mutation, disturbing the orientation of germ in relation to cob, i.e., the embryo facing the stalk end of the ear as opposed to the tip end in normal situation, has been recovered in the course of a mutagenesis study. Progeny raised from these reversed embryo cases produced ears with all the germs facing the base of the ear or having both normal and abnormal germ orientation with variable

frequencies. The mutant showed poorer seed viability and weaker plant vigor as compared to normal sibs. A similar mutation disturbing the germ orientation was reported earlier by Brieger (MNL 20:55, 1948) and Joachim (MNL 29:53, 1955; 30:84-85, 1956). Joachim designated this trait as "reversed germ" and explained their occurrence as due to the development of both the florets in an earshoot resulting in two kernels facing each other. She observed that in Country Gentleman sweet corn 50% of kernels were reversed in the part of the ear where no distinct rows could be detected.

In our case, germ reversal was not always associated with crowding of kernels and irregularities of rows as reversal was noticed in many straight-rowed ears. The reversed position of the germ in relation to the ear shoot seems to be due to modified orientation of the ovule. Differential growth of integuments and nucellus at the posterior side of the ovule during normal development pushes the ovule to one side by 90^{o}. If the ovule is pushed on the other side to the same extent, the germ would show 180^{o} turning in respect to the normal position in mature cobs. This probably is the cause for origin of reversal in the reversed germ cases. Several kernels were found in few ears where this reversal was incomplete, showing part of the embryo on top of the kernels. The position of the embryo in relation to the silk scar was found to be unchanged. Hence, other possibilities like the twisting of the pedicel to bring about 180^{o} turning of the germ to face downward or interchange of relative position of embryo and endosperm initials in the embryo sac, i.e., turning of embryo sac within the ovule, seem to be less likely.

It is a maternal plant character and in crosses with normal orientation, expression of this trait depends on the female parent. When reversed embryo was used as female, the ears in F_1 plants showed this trait but were all normal when used as male. In F_2, a 3:1 segregation for normal and abnormal ears was obtained. Penetrance of this trait seemed to be complete but the ears exhibiting this characteristic showed variable expressivity of this gene. In the testcrosses an expected 1:1 segregation was realised depending on the choice of the female parent. The percentage of reversed germ kernels in the ear could be progressively increased through selection, suggesting the presence of modifiers. Among ears showing this trait in F_2 74% had less than 10% reversed germ kernels and only 8% had more than half of the kernels in the ear with reversed germ orientation.

We propose a three letter gene symbol, rgo, for this reversed germ orientation trait.

J. K. S. Sachan and K. R. Sarkar

C-banding pattern in some Indian and exotic strains of maize and teosinte

The Himalayan strains of maize, especially Sikkim primitives (SP 1 and SP 2) have aroused considerable interest in recent years among crop plant evolutionists. In order to obtain an explanation of the presence of primitive maize in the North-Eastern Himalayan region, cytological studies were undertaken. Distribution of constitutive heterochromatin was studied in some Indian and exotic strains of maize and teosinte during mitosis. Table 1 gives an idea of the C-banding pattern of some of these strains.

There are striking differences in the pattern of C-banding between primitive forms of maize and teosinte. The chromosomes of primitive strains of maize carry fewer and relatively smaller bands than in the evolved races of maize and teosinte. Apart from the conspicuous bands which are found at the terminal and sub-terminal regions, smaller and less distinct bands were also observed at the median and sub-median positions on chromosomes in teosinte and some strains of maize.

Table 1. C-banding pattern in maize and teosinte chromosomes

Strains	Source of material	C-banding			
		Interphase		Metaphase	
		Shape	Number	Large	Small
Races from the Americas					
Palomero Toluqueno	Mexico 6, Mexico	round, spherical	0-2	1-2	-
Palomero Toluqueno	Mexico 5, Mexico	"	0-4	3-5	-
Imbricado	Boyaca 406	"	0-3	1-3	-
Confite Morocho	CIMMYT, Mexico	"	2-4	3-4	-
Nal-Tel	Yucatan 7	"	5-14	12-13	2-4
Pira	Boyaca 462	"	6-22	17-19	2-3
Celaya	Guanajuato 28	"	9-17	17-19	3-6
Strains from N-E Himalayan regions					
SP 1	Sikkim, India	"	6-10	10-12	-
SP 2	Sikkim, India	"	10-12	12-14	-
Sikkim Collect. #1	Sikkim India	"	3-6	5-8	-
Naya Bunglow	Meghalaya, India	"	3-5	4-5	-
Other cultivated lines and varieties					
K.T. 41	Kanpur, India	"	6-10	10-12	-
Stock 2	CM 105, India	"	5-7	10-12	-
Golden Cross Bantam	U.S.A.	"	4-10	8-10	5-6
Zairaisu	Japan	"	4-13	10-12	-
Teosinte					
6 collections	Mexico	Irregular, angular	9-24	17-25	2-13

The banding pattern in chromosomes of the Himalayan races SP 1 and SP 2, which are derivatives of a widely grown cultivated variety variously named as Murli, Muralia, Poorvi Botapa, etc. in Sikkim, Meghalaya, and other parts of North-eastern Himalayan regions, do not correspond to those in the Mexican primitives. SP 1 and SP 2 chromosomes carried as many as 14 bands. Other collections from Meghalaya and Sikkim, however, showed fewer C-bands.

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Gene sequence from preferential segregation frequencies

Presence of the heterochromatic knob (K) on chromosome 10 leads to preferential segregation of the knob bearing chromosome. Frequencies of transmission of the individual genes in such stocks may be taken to serve as indices for their distance from K as the differences in these frequencies are due to differential crossing over between the genes and K. An attempt to determine the gene sequence in two R-ch isolates yielded the following data:

Table 1. Preferential segregation frequencies for g and anthocyanin in the test-cross progenies from G R-ch K / g r-g k

	Transmission frequency for K-linked gene	
	New Mexico R-ch1	New Mexico R-ch2
G (non-golden)	0.6724	0.7316
Glm (glume color)	0.7150	0.7662
Nr (nodal ring)	0.7197	0.7717
P (anther color)	0.7267	0.7568
Si (silk color)	0.7395	0.7777
Lm (leaf margin color)	0.7468	0.7691
S (seed color)	0.7497	0.8238
Population size	4,851	1,555
Gene order:	G-Glm-Nr-P-Si-Lm-S-K	G-P-Glm-Lm-Nr-Si-S-K

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Pollination effects on the grain yield and the qualities of the fertilized plants, II: self pollination with o2 pollen vs. cross-pollination with o2+ pollen

It is well known that the "opaque-2" gene in homozygosis reduces the yield of grain. It is accepted that this yield reduction can be due, partly, to a defective action of this gene in the genesis of the seed and, partly, to a minor capacity of the vegetative part of the plant. To know the relative importance of these two factors, the plants of three different single crosses, homozygous for the gene "opaque-2", were divided in two classes, in relation with the class of pollination they received: (a) plants cross-pollinated, with pollen from normal plants; (b) Self pollinated (plants pollinated with "opaque-2" pollen. Both treatments of each hybrid were set up as one trial of 6 replications (21 plants per replication). For each treatment, the grain yield and the behavior of the plants, after maturity of the grain, were measured.

As expected, the plants pollinated with normal pollen produced vitreous kernels, and the plants pollinated with opaque-2 pollen produced opaque kernels. For opaque-2 treatment, yield was 12.4% higher in one hybrid ($p < 0.05$), but 3.32% and 6.55% smaller in the other two hybrids. These two last differences were at probability levels of 60% and 20% respectively.

After maturity of the grain, the following characters of the plant were measured:

(1) Number of plants remaining green (10 days after maturity, in two hybrids, and 40 days after maturity in the other one): The number was significantly greater in one opaque-2 pollen treatment ($p < 0.001$). For the other two hybrids, all the plants of both treatments had all the leaves dry. The number of plants with "green stalk" was greater in the three opaque-2 treatments. In two of the hybrids the difference was significant at the 0.01 level.

(2) Proportion of dissolved solids in the stalk juice, measured with a field refractometer (only the stalks that remained green after maturity were observed): The opaque-2 treatment was significantly higher in two hybrids ($p < 0.05$), and smaller in the third one (40% level).

These differences in grain yield and plant characters, related with the genetical differences of the fertilizing pollen, suggest that the genetic information that the pollen brought to the embryo and to the endosperm affected not only the vigor of the seed but also the physiology of the female parent plant. Mariano Blanco, on his Doctoral thesis (University of Zaragoza, May, 1972), describes his experiments with several inbreds and one single cross of normal genotype. He found that the character "refractometrical reading of stalk juice" of different plants of the same stock was influenced by the origin of the pollen and the type of fertilization: selfed plants had higher refractometrical readings than cross-pollinated plants, and different plants of the same stock had different readings when pollinated with pollen of different inbreds. Then, he pointed out: "The influence of different pollen seems to affect the chemical composition and proportions of the components of the dry matter of the plant. . . Such influences and inmediat reaction should be kept in mind because they can affect the yields in trials." We think that studies of the interaction between the seed and the mother plant offer possibilities for research in plant physiology and plant phenogenesis.

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Relationship between pericarp thickness and several characteristics of maize kernels

Pericarp thickness of two groups of single cross maize hybrids (164 experimental and 30 commercial) and 79 parental inbred lines, grown in 1976 in performance tests and inbred nursery at VIMMES, was measured using the method described by Helm and Zuber (Crop Sci. 1972, 12:428-430). Pericarp thickness varies from 48 to 122 microns in hybrids, and from 42 to 124 microns in inbreds. Relationships between pericarp thickness and grain yield, kernel length, kernel width, kernel cross-section size, kernel weight, kernel volume, and kernel row number of hybrids were studied (Table 1).

Table 1. Relationships between pericarp thickness and several characteristics in 164 experimental (a) and 30 commercial maize single crosses(b).

Relationships		$\bar{x}$	$\bar{y}$	r	Coeff. of regression	T	F
Grain yield:Peric.thickness (t/ha-x) (microns - y)	a	10.45	77	-0.02	-0.16	-0.19	0.04
	b	12.21	77	0.27	2.90	1.80	2.31
Kernel length: - " - - mm	a	12.2	77	-0.10	-0.01	-1.29	1.67
	b	11.6	77	0.23	0.01	1.45	1.57
Kernel width: - " - - mm	a	8.1	77	0.33***	0.02	4.39	19.3
	b	8.7	77	0.61***	0.23	4.05	16.4
Kernel cross section-mm^2 : - " -	a	37	77	0.38***	0.18	5.16	26.6
Kernel volume: - " - - mm^3	a	277	77	0.22**	0.79	2.06	8.2
	b	295	77	0.60***	1.78	4.01	16.1
Kernel weight: - " - -grammes	a	0.36	77	0.42***	0.00	3.00	9.0
	b	0.36	77	0.51**	0.00	3.18	10.1
Kernel row number: - " -	a	17	77	-0.38***	-0.05	-3.40	26.6

Pericarp thickness of the two groups of hybrids does not correlate significantly to grain yield (r = -0.02 and 0.27 respectively), and to kernel length (r = 0.10 and 0.23). Significant positive correlation was found between pericarp thickness and kernel width (r = 0.33*** and 0.66***), size of kernel cross section (r = 0.38*** (Fig. 1), kernel weight (0.42*** and 0.51***) and kernel volume (r = 0.22** and 0.60***). Significant negative correlation (Fig. 2) was found between pericarp thickness and kernel row number.

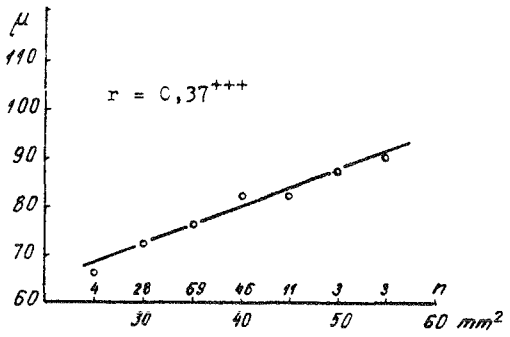

Fig. 1. Relationship between the size of kernel cross sections (width x thickness) in mm^2, and pericarp thickness in microns of 164 hybrids.

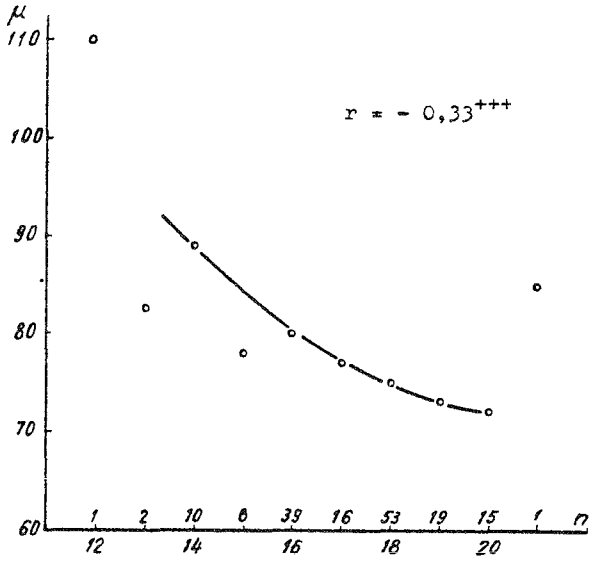

Fig. 2. Relationship between kernel row number and pericarp thickness of 164 hybrids.

Purdy and Crane (Crop Sci. 1967, pp. 294-297) reported that the pericarp thickness is an important factor in moisture loss from the maize kernel after physiological maturity. Data obtained in this experiment show that an increasing of the kernel row number and kernel length, and a decreasing of the kernel width, contribute to thinner pericarp in breeding for high yielding and fast drying maize.

Trifon M. Georgiev

Rapid measurement of mature maize pericarp

Pericarp thickness mean values from 6 measurements, and pericarp thickness mean value from 2 measurements (at the middle of germinal, and the middle of abgerminal side of the maize kernel) in 164 single crosses and in 79 parental inbred lines were compared.

Very high significant positive correlation was found between the two mean values in inbreds (r = 0.988***) and in single crosses (r = 0.935***) (Figures 1 and 2).

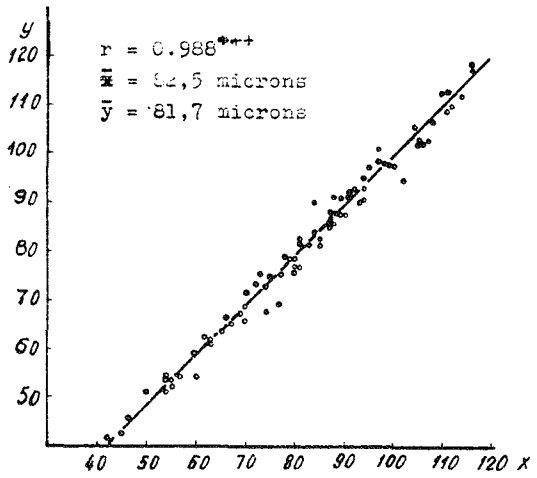

Fig. 1. Relationship between mean values of 6 measurements (x) and mean values of 2 measurements of pericarp thickness (y) in 79 maize inbred lines.

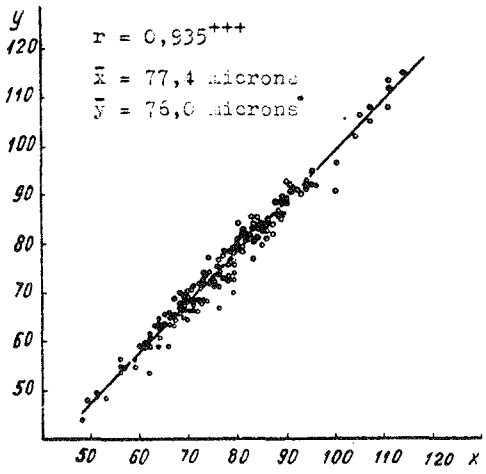

Fig. 2. Relationship between mean values of 6 measurements (x) and mean values of 2 measurements (y) of pericarp thickness in 164 maize single crosses.

While the 6 measurements give exact information on pericarp thickness at the tip, middle and top parts of the kernel for detailed morphological studies of maize inbreds and hybrids, the two less time-consuming measurements give satisfactory data for pericarp thickness on germinal and abgerminal sides of kernel in comparative inbred and hybrid studies.

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Studies on sucrose synthetase in the developing endosperms and embryos of normal and shrunken genotypes

The Sh locus on chromosome 9 has been shown to be the structural gene for the major form of sucrose synthetase in the developing endosperm of maize (Chourey and Nelson, 1976, Biochem. Genet. 14:1041). This form of the enzyme was found missing in the shrunken (sh/sh) endosperm. The residual sucrose synthetase activity in the mutant endosperm was thought to be due to another locus elsewhere in the genome. According to this, the normal endosperm should have both forms of the enzyme unless some complex gene interaction/regulation is taking place. However, to date, it has not been possible to conclusively demonstrate the presence of two forms. The observations described in this report concern the characterization of sucrose synthetase activity in normal and shrunken endosperms and normal embryos.

The enzyme sucrose synthetase is known to catalyze sucrose synthesis as well as sucrose cleavage. Both these reactions can be readily assayed. Experiments were done to determine the pH optimums for the two reactions. Sucrose synthesis and sucrose cleavage reactions showed two distinctly different pH optimums; but no differences were found for the two genotypes as well as the embryos (Table 1). It was noticed during these studies that the HEPES buffer which was used for all the experiments described in the previous report (Biochem. Genet. 14:1041) was inhibitory in action as compared to the tris-chloride buffer (same pH). In addition, the

Table 1. Properties of sucrose synthetase in normal and shrunken endosperms and normal embryos.

Criteria	+/+ Endosperm	sh/sh endosperm	Embryo
1. Ammonium sulphate cut	30-50%	30-50%	30-50%
2. Biogel A 1.5 column, Peak sp. act. tube #	34-35	33-34	33-34
3. Electrophoretical	Sh protein band	forward band (F.B.)	F.B.
4. Immunochemical*	similar	similar	similar
5. pH optimums for:			
Sucrose synthesis	9.0	9.0	9.0
Sucrose cleavage	6.0	6.0	6.0
6. Km's for:			
UDP-Glucose x 10^{-3} M	0.435	0.215	0.55
Fructose x 10^{-3} M	2.93	2.68	2.67
UDP x 10^{-3} M	0.85	0.62	0.47
Sucrose x 10^{-3} M	185.0 (192.0 for the Sh protein)	213.0	202.0
7. Developmental Profile	Same for the two genotypes.		Not done.

* As judged by immuno-inactivation of the enzyme activity by the antiserum.

sucrose cleavage reaction was previously underestimated in view of the low pH optimum described here. This necessitated a re-examination of the enzyme activity in various shrunken mutants (Table 2) using glycine-NaOH buffer for sucrose synthesis and MES buffer for sucrose cleavage.

All the sh mutants described in Table 2 are of independent origin and have a residual activity of less than 5% as compared to the normal endosperm. The embryos of both the genotypes have approximately 15% enzyme activity (as measured in either direction) of the normal endosperm. Various properties of the enzyme in the endosperm and embryo are listed in Table 1. The sh/sh endosperm enzyme is distinguishable from the Sh/Sh endosperm by only two criteria: 1) a very slight difference in electrophoretic mobility and 2) that the former shows a lesser amount of sucrose synthetase protein by electrophoretic as well as immunochemical criteria.

Table 2. Sucrose synthetase activity in 22 day old endosperms of normal and spontaneously induced shrunken mutants.

Stock	Synthesis (pH 9.0) Sp. Act.*	%	Cleavage (pH 6.0) Sp. Act.*	%
Normal-W22	1080	100	1568	100
shrunken-W22	48.5	4.88	78.5	5.05
shrunken-7196	29.0	2.92	36.9	2.37
shrunken-7205	50.4	4.67	79.5	5.07
shrunken-7342	29.9	2.77	42.6	2.72
shrunken-7611	41.8	3.87	53.6	3.42
shrunken-7650	56.0	5.18	76.7	4.89
shrunken-7731	60.4	5.59	83.1	5.30
Normal-W22 28 day	1417	100	2058	100
shrunken-7321 28 day	25.5	1.8	36.2	1.76

* Specific Activity: nmoles of sucrose or reducing sugar synthesized per mg of protein per minute.

Immunochemical cross-reactivity was judged in the past by Ouchterlony double diffusion tests. No evidence of the presence of any protein that will cross-react with the Sh protein was seen in the sh endosperm and the embryos (of either genotype). In the present studies we have assayed the same property by immuno-inactivation of the enzyme activity after the treatment of an enzyme with an antiserum. A complete inactivation of the enzyme activity was observed when crude preparations of Sh or sh endosperm or embryos were treated with an antiserum raised against the partially purified Sh protein. The antiserum raised against the partially purified sucrose synthetase from sh/sh endosperm, when treated similarly, also led to the complete inactivation of enzyme activity. Reasons for the discrepancy between the two immunological tests are not known. The developmental profile was monitored starting from 4 days through 48 days after pollination and no qualitative differences between the two genotypes were observed.

Prem S. Chourey and Oliver E. Nelson

Interallelic complementation at the sh locus at the enzyme level

Interallelic complementation leading to restoration of the normal phenotype in hetero-allelic combinations of some of the EMS induced sh mutants was reported previously (Chourey, 1971, Genetics 68:435). These studies were, however, restricted to the phenotype and the protein level as the enzymatic nature of the gene product was not known. Recent observations (Chourey and Nelson, 1976, Biochem. Genet. 14:1041) showing the product of the Sh locus (Sh protein) to be the enzyme sucrose synthetase has enabled us to examine some of the EMS induced sh mutants and their hybrids at the enzyme level (Table 1). The following observations are noteworthy:

Table 1. Sucrose synthetase activity in 22 day old endosperms of normal, EMS induced shrunken mutants and their hybrids.

Genotype	Phenotype	Synthesis (pH 9.0) Sp. Act.*	Synthesis (pH 9.0) %	Cleavage (pH 6.0) Sp. Act.*	Cleavage (pH 6.0) %
+/+-W22	Normal	994.0	100	1555.0	100
sh/sh-W22	Mutant	48.5	4.88	78.5	5.05
sh/sh-FFF	Mutant	63.9	6.43	91.9	5.91
sh/sh-CCC	Mutant	49.9	5.02	66.6	4.28
sh/sh-SSS	Mutant	301.0	30.3	73.2	4.71
sh/sh-FFC	Mutant	74.7	6.92	115.7	7.38
sh/sh-FFS	Normal	148.9	14.98	158.5	10.19
sh/sh-SSF	Normal	235.0	23.64	184.0	11.83
sh/sh-SSC	Normal	254.0	25.55	155.5	10.80

* Specific activity: nmoles of sucrose or reducing sugar synthesized per mg of protein per minute.

The mutants sh-F and sh-C are similar to the reference allele of sh mutant (W22 background) as regards amounts of sucrose synthetase activity. The same is true of sh-S if its sucrose synthetase activity is assayed in the direction of sucrose degradation (in the presence of UDP). The genetic basis of this residual enzyme activity in these three mutants is not known at this time, as each of

sh-F, sh-C and sh-S is known to code the sh locus specific protein (Chourey and Schwartz, 1971, Mutation Res. 12:151). The sh-S mutant, however, is genetically as well as biochemically unique as compared to all the sh mutants that we have studied so far. The enzyme specified by this mutant showed as much as 30% (as compared to the normal endosperm) activity when assayed in the direction of sucrose synthesis. The protein coded by sh-S locus is probably responsible for most of this enzyme activity. The sucrose synthesis and sucrose cleavage properties of sucrose synthetase are believed to be due to the same protein molecule in various plants including maize. Thus, it seems, there is a differential alteration in the rates of sucrose synthesis and sucrose cleavage in the enzyme molecule coded by the sh-S mutant. The occurrence of such a qualitative change in the enzyme in the sh mutant, which was initially selected on the basis of its shrunken phenotype, further substantiates that the Sh locus is the structural gene for sucrose synthetase.

The phenotype of the F_1 hybrid kernel produced by crossing sh-S with either sh-C or sh-F is indistinguishable from the wild type (Sh) kernel. No other hetero-allelic combination of sh mutants complements to produce the wild type phenotype. The endosperm extracts of the complementing hybrids (sh-S x sh-F or sh-S x sh-C) when assayed for sucrose synthesis showed activities that were lower than the sh-S but were higher than the sh-F or sh-C homozygotes. Thus the complementing heterozygotes, which have a wild type phenotype, have a lower enzyme activity than one of the parental types which is mutant in phenotype. However, when the same hybrids were assayed for sucrose cleavage the results were different. The complementing heterozygotes show a distinct elevation in the enzyme activity as compared to the parental types. A slight elevation is also observed for the noncomplementing heterozygote, viz. sh-F x sh-C. It is possible that the critical limit of the enzyme activity to restore the wild type phenotype lies somewhere between the values 7% and 10%, as compared to the wild type.

The physiological mode of sucrose synthetase reaction, i.e., whether sucrose cleavage or sucrose synthesis is the in vivo reaction in a developing kernel, is controversial. The following observations of this investigation are interpreted to suggest that sucrose cleavage is the critical in vivo reaction to mobilize photosynthesized sucrose for starch biosynthesis. It is possible that the sh-S mutant is shrunken in phenotype because of a significant reduction in the catalysis of the sucrose cleavage. Note that the rate of sucrose synthesis is not limiting as it is even higher in this mutant than in the complementing hybrids which have a wild type phenotype. In other organisms, interallelic complementation leading to wild type phenotype has been associated with an elevation in the enzyme activity as compared to the parental types. It is observed that such an elevation takes place in the hybrids only when the enzyme activity was assayed for sucrose cleavage.

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III. CYTOGENETIC WORKING MAP

Thanks to the efforts of a number of cooperators who prepared and forwarded compilations of their information, it has been possible to assemble tabulations of some key data and to draw preliminary cytogenetic maps. The following were put together hastily from the information supplied, and are reproduced to stimulate discussion and to invite further information. The work of synthesis was done as "volunteer" assignments by the following: Maguire (chromosomes 1 and 3); Patterson (2 and 4); Burnham and Phillips (5, 6 and 7); Coe and Beckett (8, 9 and 10). We apologize to cooperators for any violence done to the information that was supplied (and I to the committee for any undue nonchalance on my part in re-composing their work--E.H.C.).

Please note:

1. Some data do not combine to make consistent maps; substantial revisions can be anticipated in chromosomes 3, 6 and 10 as information develops further.
2. These are preliminary tabulations and working maps, hastily put together; they need more data and more analysis.
3. Additional information, tabulated in the work format of the tables, should be sent to Coe for next year's compilation.

J. B. Beckett, C. R. Burnham, E. H. Coe, M. P. Maguire,
E. B. Patterson and R. L. Phillips

Chromosome 1, short arm

	0 sr	1 vp5	(14) ag	15 ga6	19 zb4	23 ms17	24 ts2	26 P	28 zl	56 as	(58) pa	Ref.
T1-10g(1S.80,10L.21)								19.1				2
T1-2d(1S.78,2L.56)								20.9				2
T1-2c(1S.77,2L.33)		1.4					16.8	17.8				1
"	T	1.0	1.8									7
TB-1Sb-2Lc(1S.05, 1S.77,2L.33) Df	T		vp5									6
T1-5i(1S.71,5S.74)								17.2				2
T1-4b(1S.55,4L.83)								7.3				2
TB-1Sb-2L4464(1S.05, 1S.53,2L.28) Df	T		vp5									6
T1-9c(1S.48,9L.22)								0.8	T	?		1
T1-2b(1S.43,2S.36)							0.8	3.8	T	?		1
"								5.0				2
T1-6c(1S.25,6L.27)								7.7	T	?		1,2
T1-5b(1S.17,5L.10)								21.8	T	?		1,2
T1-3a(1S.15,3L.17)								10.1	T	?		1,2
TB-1Sb(1S.05) Df	sr	vp5			T	?						3,8

Chromosome 1, long arm

	64 hm	81 br	85 Vg	86 f	104 an	106 bz2	(108) ad	119 Ts3	(124) tb	127 Kn	(128) (lw,Adh)	135 gs	(154) vp8	158 Ts6	161 bm2	Ref.
TB-1La(1L.2) Df	hm	br			an	bz2					lw	gs	vp8		bm2	3,7,8
T1-6b(1L.25,6sat.)Burnh.	T	3.8														5
T1-7c(1L.39,7L.14)		0.0											34.3		44.7	8
T1-3(5267)/(5242), Dp 1L.72-.90											T Adh T					4
T1-3(5476)/(5242) Dp 1L.66-.90											T Adh T					4
TB-1La-3L(5267), Df 1L.2-.72											T +					4
TB-1La-5S(8041), Df 1L.2-.80						bz2					T +					4,9

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CHROMOSOME 2

Rearrangement	Cytological Positions		lg	gl2	B	sk	ts	-o-	v4	w3	Ht	Ch	Reference
2-5g	2S.79	5S.24		2.1 T 4.2									7
2-3e	2S.76	3L.48 T 0.4		3.9									7
Inv. 2a	2S.7	2L.8							34.5		Inv	30.4	1
Inv. 2a				Order:gl2-Inv-B							3.3 Inv	17.0	8
Inv. 2a	(Inv/Inv)			gl2-Ht = 7 ;	B-Ch = 32								8
2-6b	2S.69	6L.49		3.9 T 0.9									1
2-3(5304)	2S.62	3L.29		2.8 T 4.6									7
Inv. 2b	2S.50	2L.15			17 Inv			Inv	5.5				6
2-5(4741)	2S.47	5L.47			5.1	3.7 T							7
2-3c	2S.46	3S.52		0.5 T 4.9									1
2-3c				0.5 T 2.5									7
TB-3La-2S(6270)	2S.46	3L.1-.60	lg	gl2	b	sk wt T							3, 11, 9
2-3(6862)	2S.39	3L.20			11.6	8.7 T							7
1-2b	2S.36	1S.43			5.3 T	1.4							1
2-9a	2S.36	9L.58				0.5							1
2-9a						1.2 T							7
1-2(5255)	2S.31	1S.25				9.3 T							7
2-9b	2S.18	9L.22					5.0	T	7.8				2, 1
1-2(4937)	2S.15	1L.10				14.7		T	6.5				7
2-5b	2L.06	5S.09							5.0				1
2-5a	2L.14	5L.15						T	7.3				10
2-10a	2L.16	10L.55					13.5	T	6.5				2, 1
2-4d	2L.17	4L.45					9.6	T	8.8				1
TB-3La-2L(7285)	2L.26	3L.1-.39						T	v4				3, 11, 9
TB-1Sb-2L(4464)	2L.28	1S.05-.53						T	v4				3, 11, 9
2-4a	2L.30	4L.21					12.9	T	1.0				1
1-2c	2L.33	1S.77					8.5		0.3				1
TB-1Sb-2Lc	2L.33	1S.05-.77							+	T w3			3, 11, 9
2-10(5561)	2L.35	10S.16				26.5			3.8	T			7
2-7b	2L.37	7L.12					15.3	T	5.4				1
2-6c	2L.37	6L.25					12.3	T	1.7				1, 4, 5
2-6a	2L.40	6S.50							1.1				1, 4, 5
2-6d	2L.41	6L.45					26.6	T	4.2				1
2-3b	2L.45	3L.08							4.0	T			2, 1
2-7c	2L.47	7S.34							1.0	T			1
2-10(8219)	2L.50	10L.35									2.2		13
1-2d	2L.56	1S.78									5.5		13
2-4l	2L.56	4S.51									8.4		13
2-8(051-15)	2L.62	8L.48									16.7		13
2-3d	2L.67	3L.48									16.4		13
2-4f	2L.75	4L.12							22.3	T			1
2-4b	2L.81	4L.53							5.6		15.0		1, 13
2-4c	2L.81	4S.09							19.0			34.2	1
2-4c									24.6	1.4 T			12
2-9d	2L.83	9L.27							17.4	0.5 T			12

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Chromosome 3

	0	18	26	31	(38)	40	(45)	46	48	49	50	55	72	(75)	83	(86)	111	111+	122	128		Ref
	cr	d	ra2	Cg	cl	rt	Rf	Lg3	Rg	Rp3	gl6	ts4	ba	yl0	lg2	na	a	sh2	et	ga7	(vp)	
T3-7b(3S.92,7L.03)		0.4										27.3			43.8	43.5						1
T1-3d(1L.61,3S.75)		0.6	T	?								25.3			33.3	43.5						1
T3-6b(3S.73,6S.82sat)B.		0.4																				1
T2-3c(2S.46,3S.52)		0.8										17.7			35.4	33.5						1
T3-4(5156)(3S.47,4L.67)				(T	E4)																	2
TB-3Sb uncovers	cr	d	ra2	Cg	cl		T				+											5
T3-6a(3L.06,6L.30)		18.0											?	T	12.0	33.0	45.0					1
T2-3b(2L.45,3L.08)												1.1										1
T3-9c(3L.09,9L.12)					4.6	T		?						20.3	9.6						3.5	4,7
TB-3La(3L.10) uncovers						+		T			gl6	ts4	ba	yl0	lg2	na	a	sh2	et		vp	4,5
T3-9a(3L.11,9L.16)		34.0										2.9			25.0		45.1				10.1	1,4
T3-8b(3L.16,8L.23)		17.6										0.0			14.8	33.8						1
TB-3Lc uncovers											+	+ T	ba	yl0	+	na	a	sh2	et			5
T3-10a(3L.16,10L.22)												10.4			17.2	31.0	48.6					1
T1-3a(1S.15,3L.17)		23.4										4.2	T		5.9	12.9	46.6					1
T3-10b(3L.19,10L.27)												0.8	T			32.2	41.1					1
T3-10c(3L.22,10L.30)												0.7					39.0					1
T3-7a(3S.25,7L.18)		20.2										5.0			15.9		52.5					1
T3-5a(3L.28,5L.60)		24.5											?	T	7.9							1
T3-8a(3L.41,8L.61)		29.5										2.5	T		5.7	11.6	34.4					1
T3-9b(3L.48,9L.53)												32.1			7.9	T	18.0					1
T2-3d(2L.67,3L.48)																13.0	T 7.1					1
T2-3e(2S.76,3L.48)																7.5	T 20.7					1
T2-3a(2S.9,3L.6)Burnh.												15.1	T			10.4	21.6					1
TB-3La-2S6270(3L.1, 3L.60,2S.46) uncovers															?	T	+		+			6
T3-5b(3L.61,5L.57)																4.8	T 19.0					1
T3-5c(3L.62,5L.27)																11.7	T 12.6					1
TB-1La-3L5267(1L.20, 1L.72,3L.73) uncovers															?	T	a					3

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CHROMOSOME 4

Rearrangement	Cytological Positions		Ts5	su	-o-	gl4	Tu	gl3	c2	dp	References
4-6b	4S.80	6L.16	1.6 T	8.6							2, 3
1-4a	4S.69	1L.51	0.0	3.5							2, 3
4-10c	4S.64	10L.18		1.1	T		24.8				2, 3
4-8a	4S.59	8L.19	4.0	1.6							2, 3
4-5c	4S.34	5L.27		1.1	T (S)		26.4				2, 3
4-7a	4S.32	7L.06	10.6	0.6							2
2-4g	4S.31	2L.13	7.1 T	2.7							2, 3
4-9g	4S.27	9L.27		3.3	T		22.1				2, 3
TB-4a	4S.25			su bt2	T						4
4-5d	4S.21	5L.22		3.4	T (S)		21.5				2, 3
4-8b	4S.18	8L.16		5.7	T						2, 3
2-4c	4S.09	2L.81		9.2	T (S)		30.8				1, 2, 3
TB-9Sb-4L(6222)	4L.03	9S.40-.68			T (L)	gl4		gl3	c2	dp	4, 6
TB-7Lb-4L(4698)	4L.08	7L.30-.74			T (L)	gl4		gl3	c2	dp	6
TB-9Sb-4L(6504)	4L.09	9S.40-.83			T (L)	gl4		gl3	c2	dp	4, 6
2-4f	4L.12	2L.75		6.1	T		19.3				1, 2, 3
4-9d	4L.12	9L.17		3.8	T		21.2				2, 3
TB-1La-4L(4692)	4L.15	1L.20-.46			T	gl4		gl3	c2	dp	4, 6
4-10b	4L.15	10L.60		4.0	T (L)						2, 3
4-9a	4L.16	9L.58		9.8	T (L)		14.1				2, 3
2-4a	4L.21	2L.30		3.3	T (L)		14.0				1, 2, 3
4-6a	4L.37	6L.43		4.9	T (L)		14.6				2, 3
2-4d	4L.45	2L.17					0.2 T	5.4			1, 2, 3
2-4b	4L.53	2L.81						15.2 T			1, 2, 3
4-5b	4L.76	5L.68						3.0 T			2, 3
1-4b	4L.83	1S.55						8.9	3.5 (T)		5
4-9b	4L.90	9L.29						18.5	15.6 T		5

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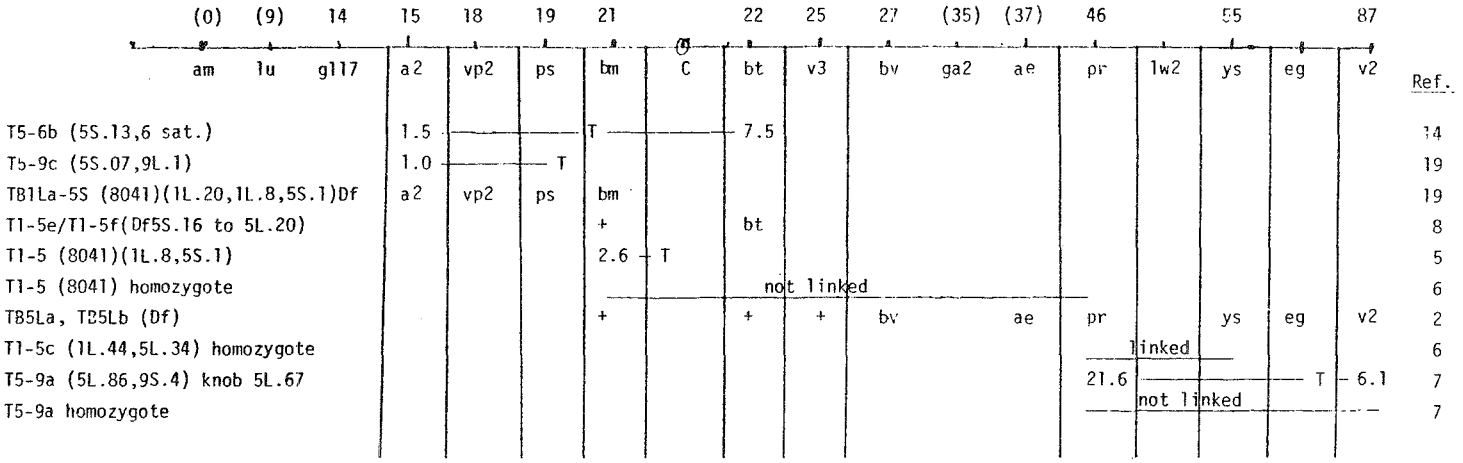
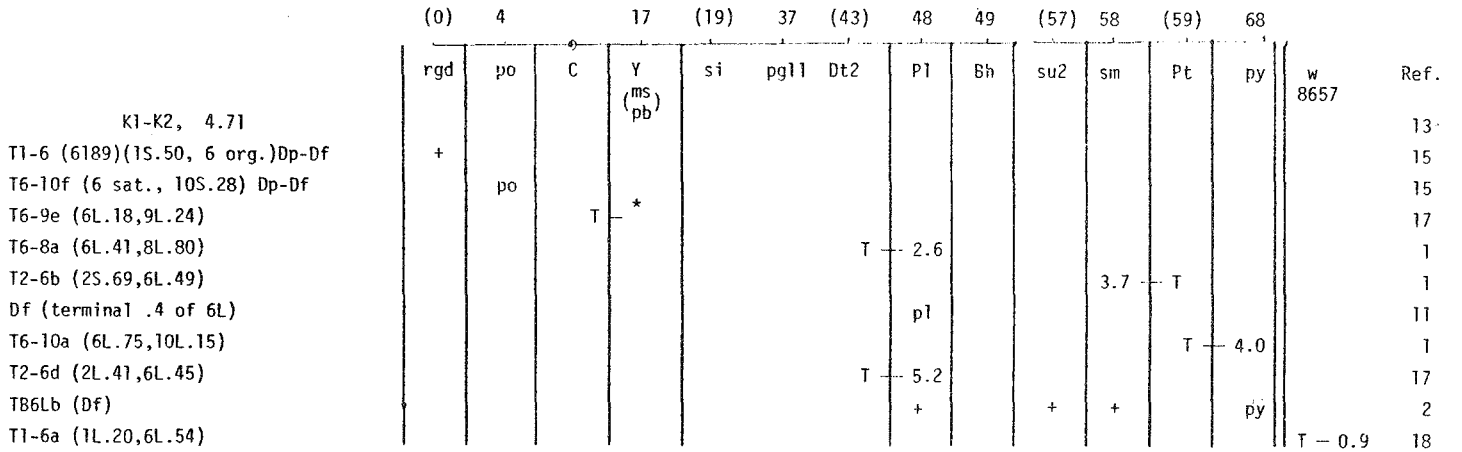
Chromosome 5

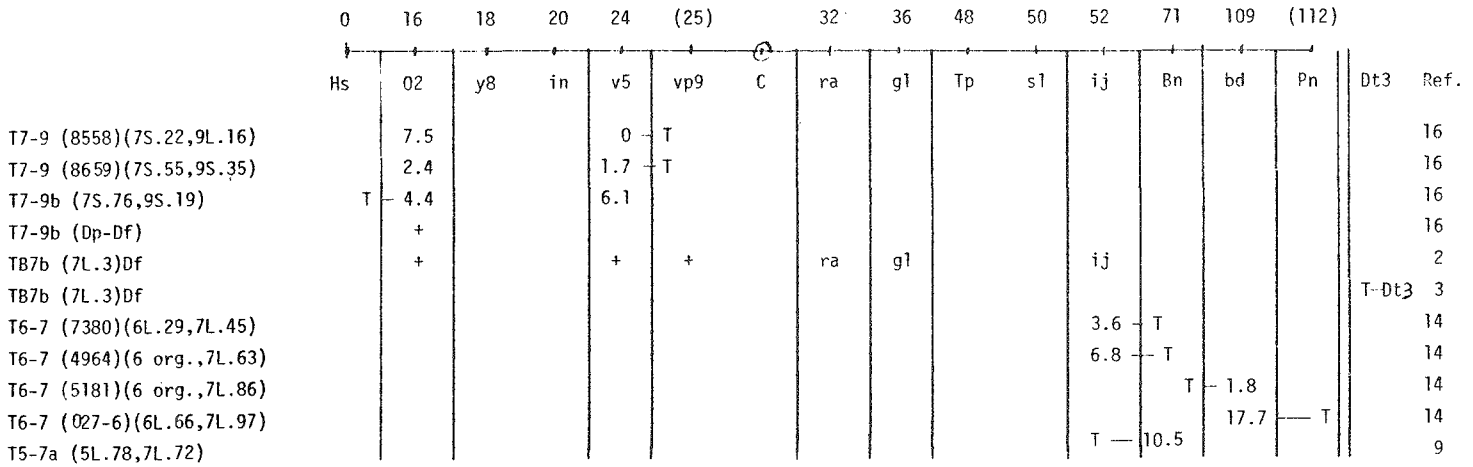


Chromosome 6



* Y linked with wx(9s)

Chromosome 7



Breakpoints listed are ones considered to be the most accurate, generally from Longley, 1961.

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Chromosome 8

	0 v16	14 ms8	28 j	Ref
T3-8a(3L.41,8L.61)	T	7.2		2
TB-8La(8L.7) uncovers	v16	ms8	j	1
T6-8a(6L.41,8L.80)	T	7.8		2

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Chromosome 9

	0 Dt	pyd	7 yg2	wd	26 c	29 sh	31 bz	40 Mr	44 bp	53 lo2	ga8	59 wx	62 d3	(64) pg12	(65) ar	66 v	67 ms2	69 gl15	79 bk2	104 Wc	134 Bf	138 bm4	Ref
Terminal Df 9S Knob		+	+	+																			6
Df Knob & thread		pyd	+	+																			6
Df distal half first chromomere		pyd	yg2	wd	+																		6
Df through sixth chr.		pyd	yg2	wd	+																		6
TB-9Sb-4L6504(9S.40, 9S.83,4L.09)unc.		T	yg2		c	sh																	5
T4-9(6504)(4L.09, 9S.83)Dp Df unc.			?	T		+																	10
T7-9(7074)(7L.03,9S.80)			?	T	0.6																		11
In9a(9S.7,9L.9)Dp-Df unc.							bz	In	?													bm4	9
TB-9Sb-4L6222(9S.40, 9S.68,4L.03) unc.		T	yg2		c	sh																	5
T4-9(6222)(4L.03,9S.68)					3.3	T	0.3																8
T8-9(4453)(8L.86,9S.68)Dp-Df unc.						sh	T	?															10
T8-9(5300)(8L.85,9S.43)												0.0	T	?									7,11
T5-9a(5L.86,9S.4)Burnham												2.0	T	?									2
TB-9Sb(9S.4) uncovers		pyd	yg2	wd	c	sh	bz				T	0.7				29.6							3,4
T5-9(4871)(5L.71,9S.38)												0.4											7
T6-9b(6L.10,9S.37)												3.8	T	?									2
T8-9(034-6)(8L.17,9S.34)												0.8											7
T2-9(6656)(2L.32,9S.31)						11						1.4	T	?									8
T8-9(6673)(8L.35,9S.31)												2.9	T	?									11
T4-9(5657)(4L.33,9S.25)					12.4							1.8	T	?									8
T3-9(5775)(3L.09,9S.24)												1.4	T	?									11
T7-9b(7S.76,9S.19)													T	?									8
T9-10b(9S.13,10S.40)												5.7											2
T7-9a(7L.63,9S.07)												0.3											8
TB-9Lc uncovers														+	T	ar	v	ms2	gl15	bk2	Bf	bm4	4
T1-9a(1S.13,9L.15)												11.2											2
T3-9a(3L.11,9L.16)												3.6		T		?							2
T1-9a/c (Df 9L.15 to .22)															ar	+							12
TB-9La(9L.4) uncovers																+	+	+	T	bk2	Bf	bm4	4
In9a(9S.7,9L.9)Dp-Df unc.							bz	In	?													bm4	9

References:

1. Burnham, C. R., Amer. Nat. 68:81, 1934.
2. Anderson, E. G., Genetics 23:307, 1938.
3. D. S. Robertson.
4. Beckett, J. B., MNL 49:130, 1975.
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6. McClintock, B., Genetics 29:478, 1944.
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10. E. H. Coe, Jr.
11. Patterson, E. B., MNL 26:8, 1952.
12. Turcotte, E., MNL 30:164, 1956.

Chromosome 10

	0	(12)			33							41	43	57	61	63	73	80	92	99	Ref
	Rp	oy	y9	C	zn	du	(li,bf2,ms10,nl)					Tp2	g	r	Lc	Mst	w2	o7	sr2	12	
TB-10Sc uncovers		oy	y9	T	+	+	+	+		+				43.0							1,2,4
TB-10L18 & 19 uncover			+		T	zn	du		bf2				g	r					sr2		3,4
TB-10L26 uncovers					+	T	du						g	r							3
TB-10L22 uncovers					+		+	T	li	bf2			g	r					sr2		3
TB-10Lb uncovers					+		+	T	li	bf2	ms10	nl	g	r			w2				3,4,5
T8-10b(8ctr.,10ctr.)					?	T	6.9						9.5	23.2							7
T6-10a(6L.75,10L.15)													10.6	12.0							6
"					?	T	5.9						9.6	23.7							7
T4-10c(4S.64,10L.18)									?	T			8.3	19.8							7
T3-10a(3L.16,10L.22)					?	T	4.2						15.7	27.7							7
T3-10b(3L.19,10L.27)					?	T	12.5						18.9	22.1							7
TB-10L20, et al., uncover					+	+	+	+		?	T		g	r							3
T6-10b(6L.12,10L.29)										?	T		2.5	18.6							7
T1-10f(1S.04,10L.30)													0.0	4.4							7
T3-10c(3L.22,10L.30)										?	T		6.5	22.8							7
T1-10e(B98)(1L.16,10L.31)										?	T		7.6	15.7							6
"										?	T		2.9	17.3							7
T1-10a(1L.29,10L.33)										?	T		15.3	34.3							7
TB-10La(10L.35)		32.5	20.9		+	+	+	12.3	+		T		g	r			w2		sr2		2
T2-10a(2L.16,10L.55)										?	T		1.8	9.6							7,8
T4-10b(4L.15,10L.60)										?	T		4.3	13.6							6
"										?	T		1.6	8.6							7,8
TB-10L32 uncovers					+	+	+	+					+	T r							3
T2-10(5651)(2S.71,10L.62)													0.3	T 4.2							6
T1-10d(A84)(1L.50,10L.68)										?	T		2.1	7.9							6
T1-10c(1L.43,10L.74)														0.0							7
T5-10(4384)(5L.13,10L.79)													0.0	T 2.3							6

References:

1. Beckett, J. B., MNL 47:145, 1973.
2. Robertson, D. S., unpublished.
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4. Beckett, J. B., unpublished.
5. Beckett, J. B., MNL 49:130, 1975.
6. Dooner, H., MNL 45:231, 1971.
7. Anderson, E. G., and H. H. Kramer, Genetics 39:506, 1954.
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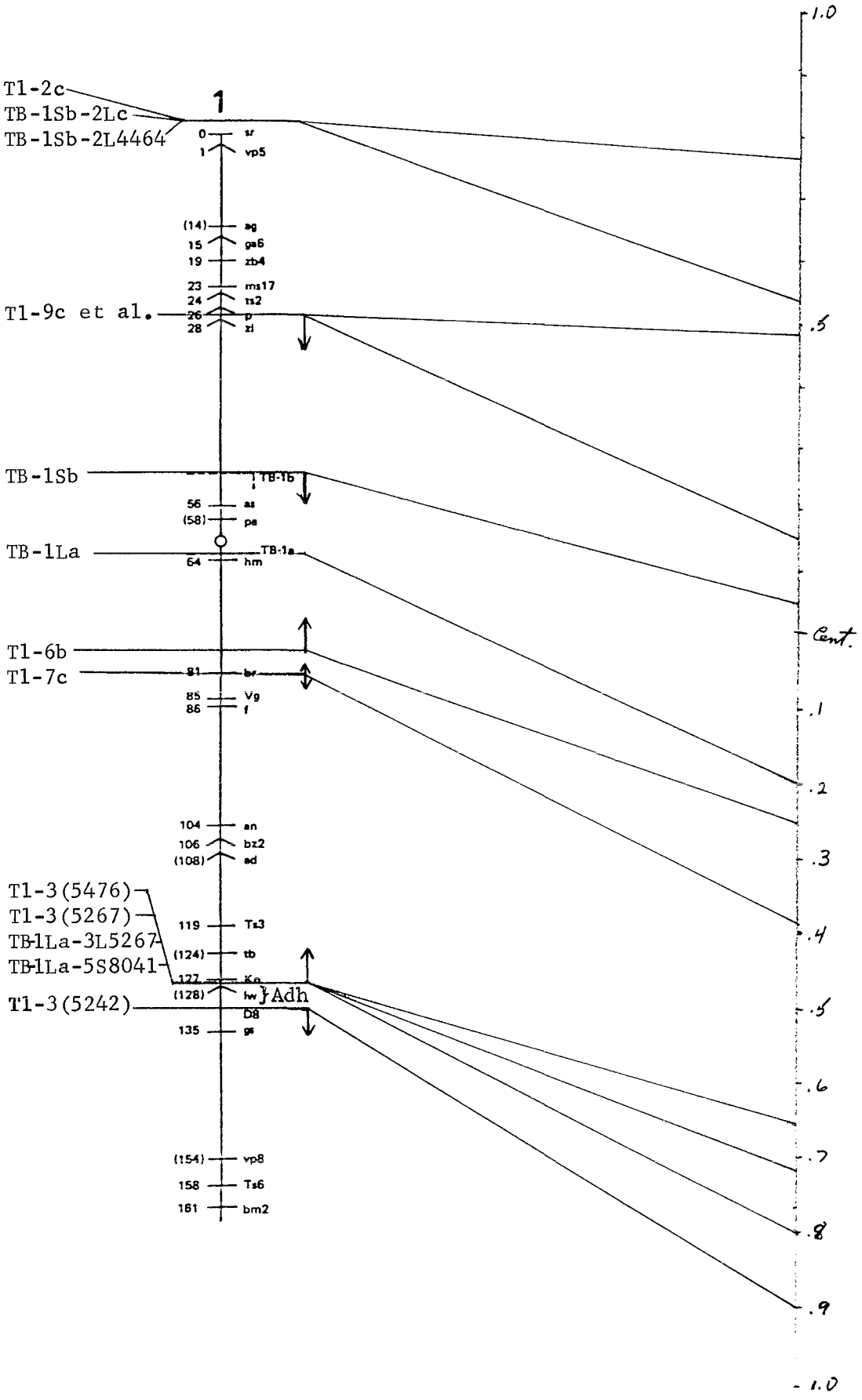
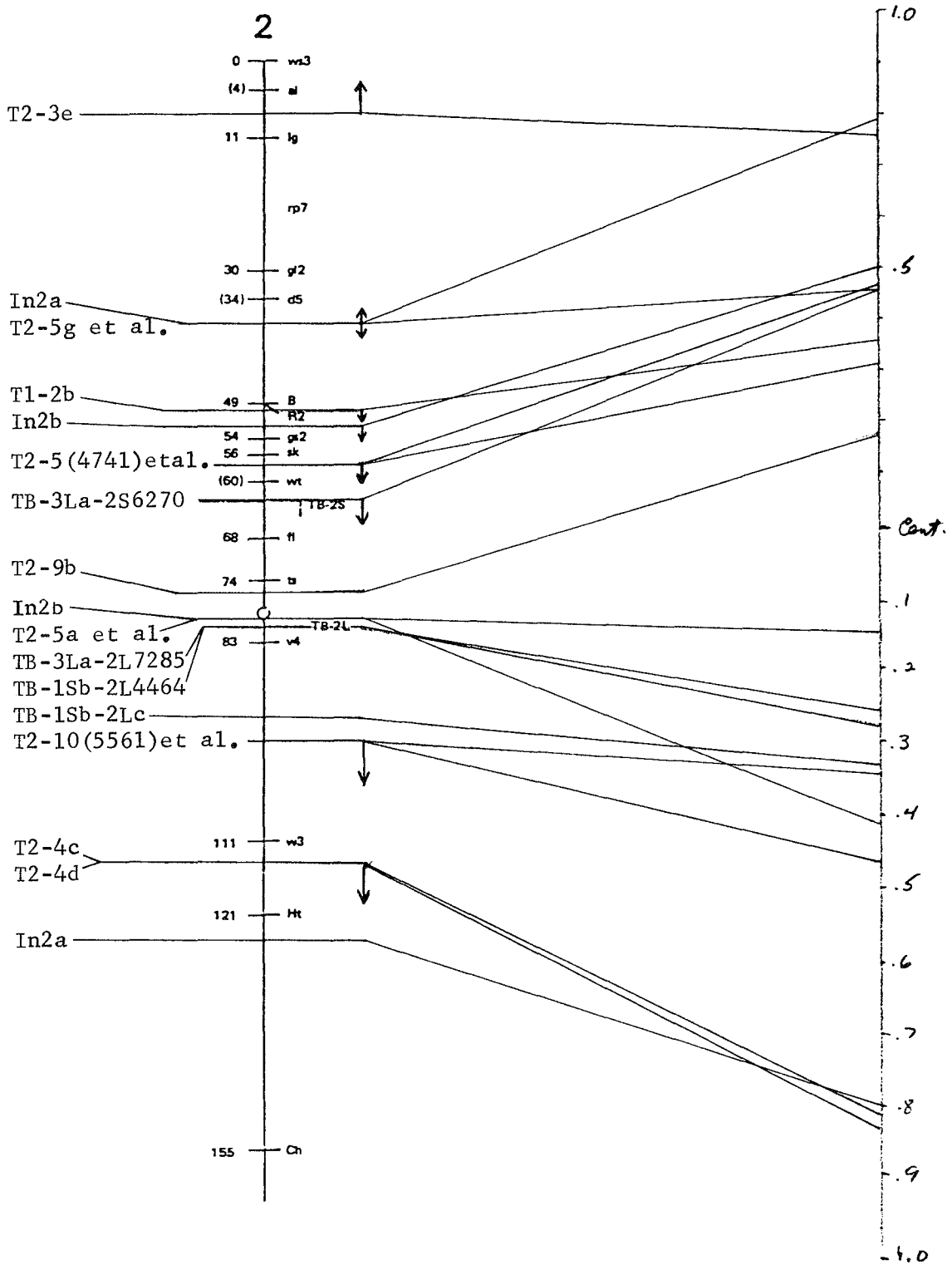
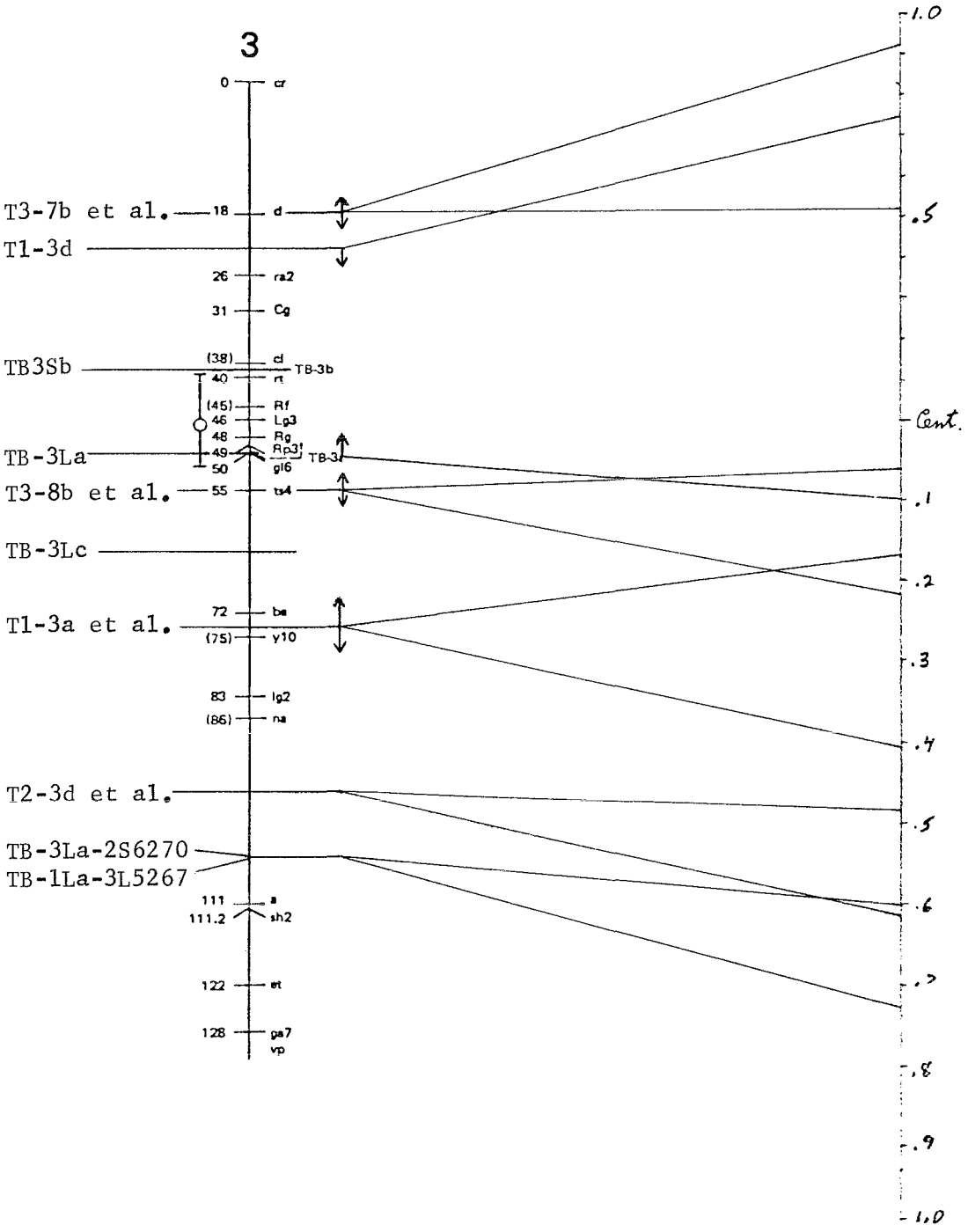
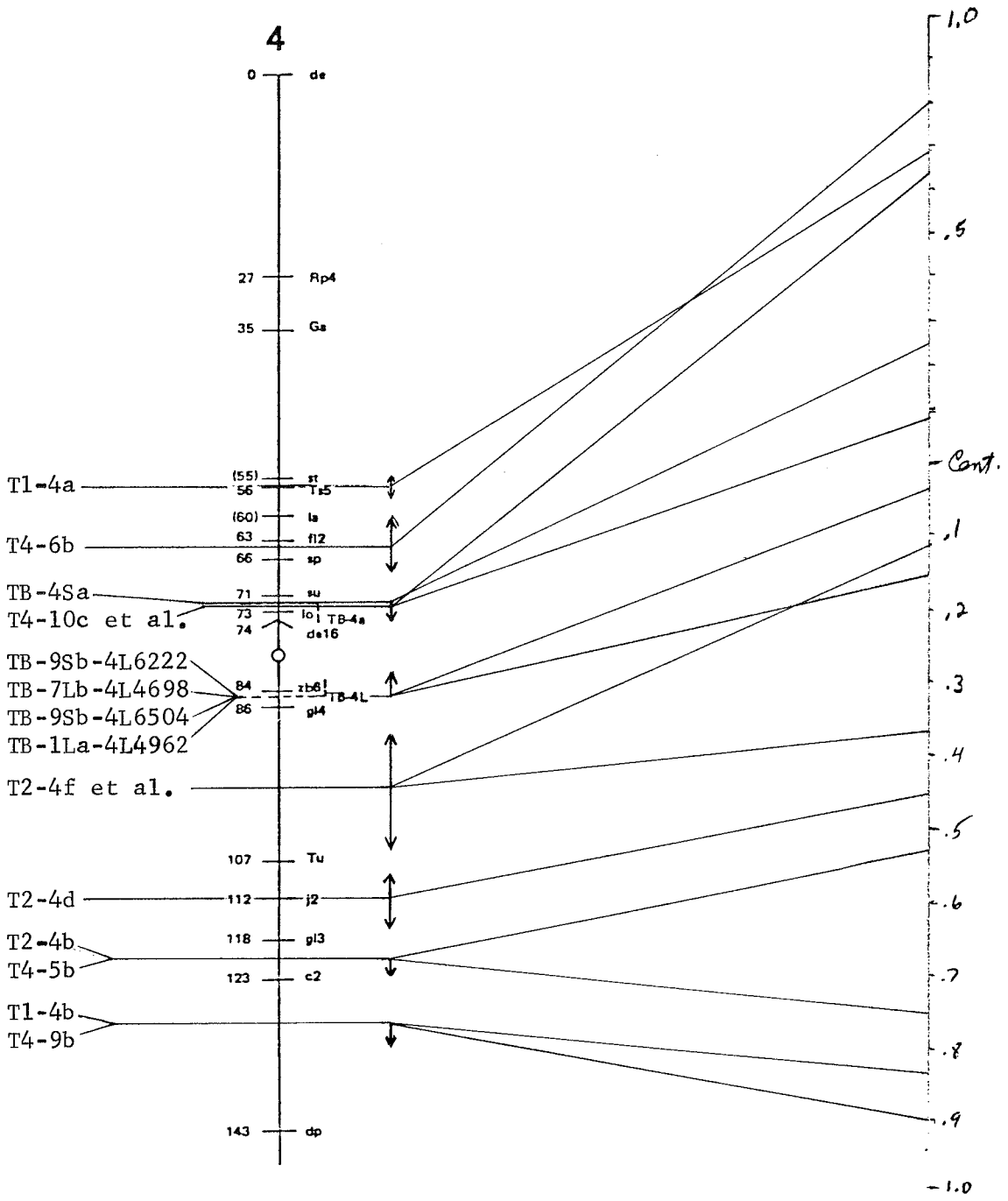
New jeans?

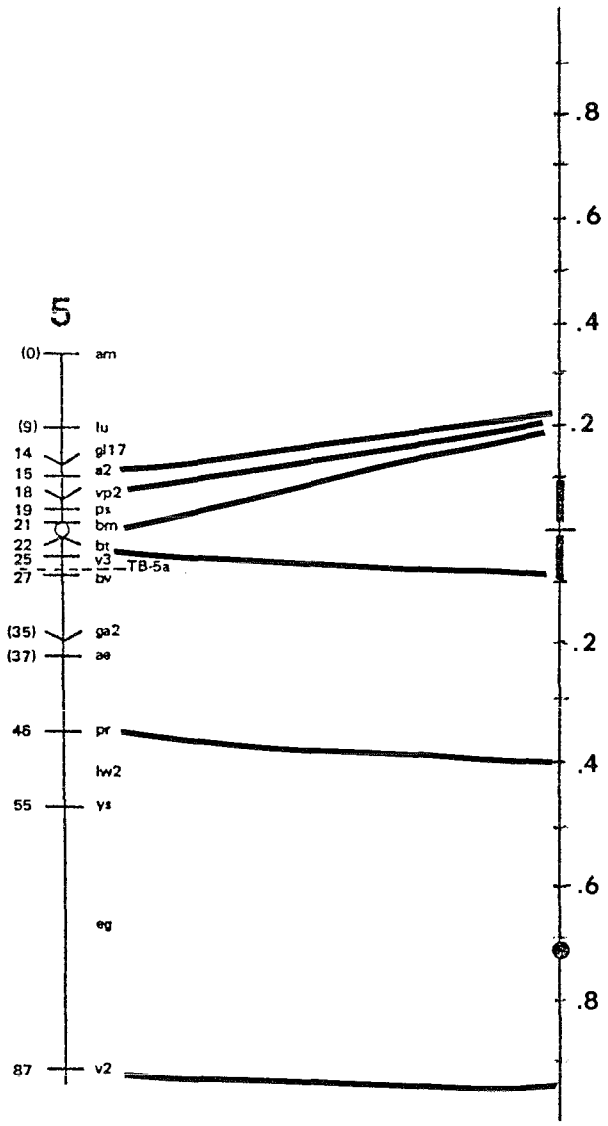
Map them!

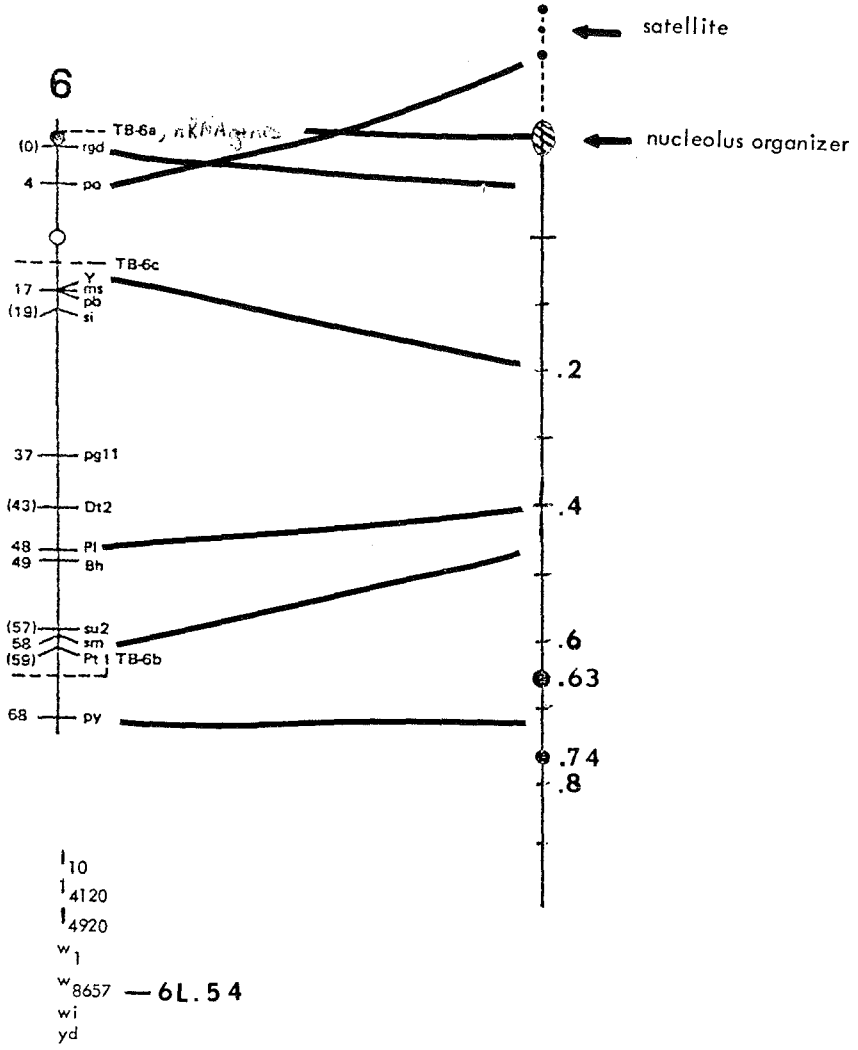

1
T1-2c
TB-1Sb-2Lc
TB-1Sb-2L4464
0 sr
1 vp5
(14) ag
15 ga6
19 zb4
23 ms17
24 ts2
26 p
28 zl
T1-9c et al.
TB-1Sb
TB-1b
56 as
(58) pa
TB-1La
TB-1a
64 hm
T1-6b
T1-7c
81 br
85 Vg
86 f
104 an
106 bz2
(108) ad
T1-3(5476)
T1-3(5267)
TB-1La-3L5267
TB-1La-5S8041
T1-3(5242)
119 Ts3
(124) tb
127 Ka
(128) lw
Adh
D8
135 gs
(154) vp8
158 Ts6
161 bm2
1.0
.5
Cent.
.1
.2
.3
.4
.5
.6
.7
.8
.9
1.0


2
0 ws3
(4) al
11 lg
rp7
30 gl2
(34) d5
49 B
R2
54 gs2
56 sk
(60) wt
TB-2S
68 fl
74 ts
TB-2L
83 w4
111 w3
121 Ht
155 Ch
T2-3e
In2a
T2-5g et al.
T1-2b
In2b
T2-5(4741)etal.
TB-3La-2S6270
T2-9b
In2b
T2-5a et al.
TB-3La-2L7285
TB-1Sb-2L4464
TB-1Sb-2Lc
T2-10(5561)et al.
T2-4c
T2-4d
In2a
1.0
.5
Cent.
.1
.2
.3
.4
.5
.6
.7
.8
.9
1.0

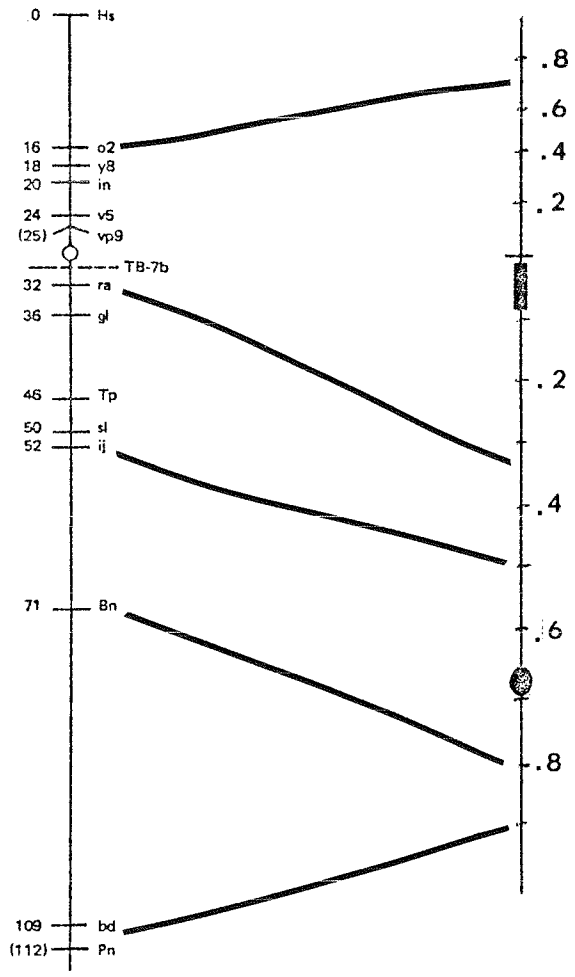

3
0 cr
18 d
26 ra2
31 Cg
(38) cl
40 rt
(45) Rf
46 Lg3
48 Rg
49 Rp3
50 gl6
55 ts4
72 ba
(75) y10
83 lg2
(86) na
111 a
111.2 sh2
122 et
128 pa7
vp
T3-7b et al.
T1-3d
TB3Sb
TB-3b
TB-3La
TB-3
T3-8b et al.
TB-3Lc
T1-3a et al.
T2-3d et al.
TB-3La-2S6270
TB-1La-3L5267
1.0
.5
Cent.
.1
.2
.3
.4
.5
.6
.7
.8
.9
1.0


4
0
de
27
Rp4
35
Ga
(55)
56
st
Ts5
(60)
la
63
fl2
66
sp
71
su
73
74
lo
TB-4a
de16
84
zb6
TB-4L
86
gl4
107
Tu
112
j2
118
gl3
123
c2
143
dp
T1-4a
T4-6b
TB-4Sa
T4-10c et al.
TB-9Sb-4L6222
TB-7Lb-4L4698
TB-9Sb-4L6504
TB-1La-4L4962
T2-4f et al.
T2-4d
T2-4b
T4-5b
T1-4b
T4-9b
1.0
.5
Cent.
.1
.2
.3
.4
.5
.6
.7
.8
.9
1.0

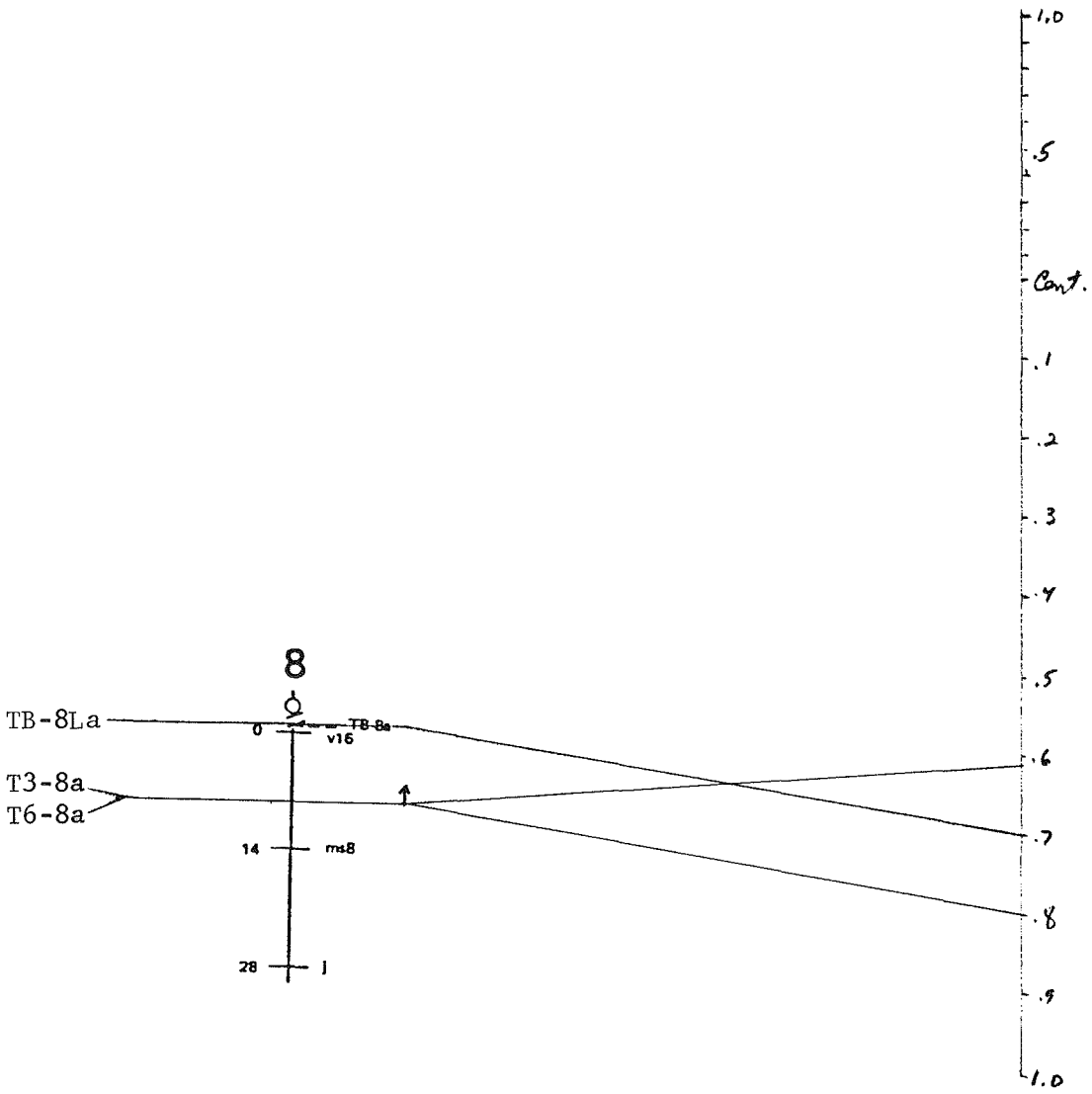

5n
(0)
am
(9)
lu
14
gl17
15
a2
18
vp2
19
ps
21
bm
22
bt
25
v3
27
bv
TB-5a
(35)
ga2
(37)
ae
46
pr
lw2
55
ys
eg
87
v2
.8
.6
.4
.2
.2
.4
.6
.8

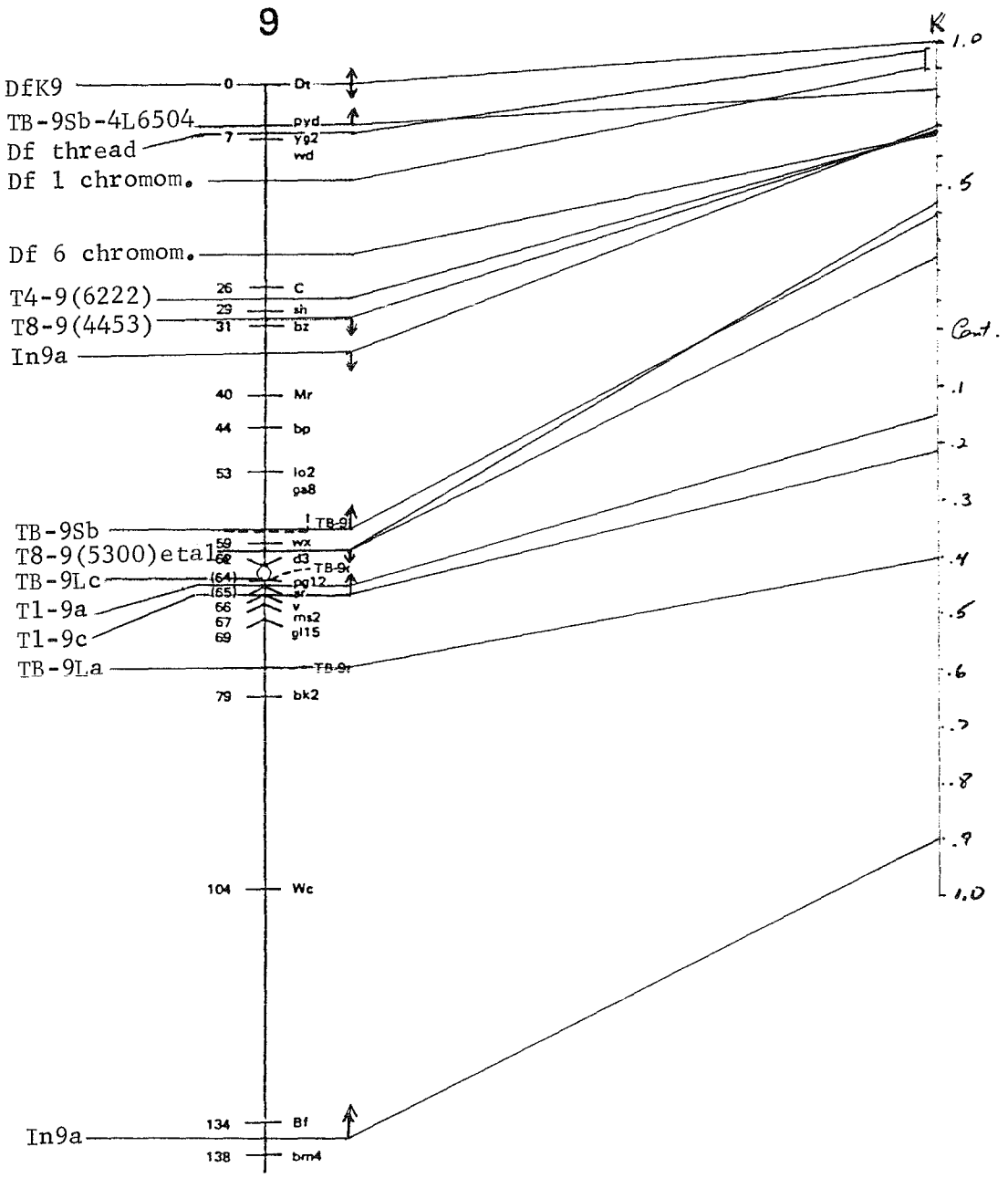

6
TB-6a
(0) rgd
4 po
TB-6c
17 Y ms pb
(19) si
37 pg11
(43) Dt2
48 Pl
49 Bh
(57) su2
58 sm
(59) Pt 1 TB-6b
68 py
satellite
nucleolus organizer
.2
.4
.6
.63
.74
.8
l10
l4120
l4920
w1
w8657
wi
yd
—6L.54

7

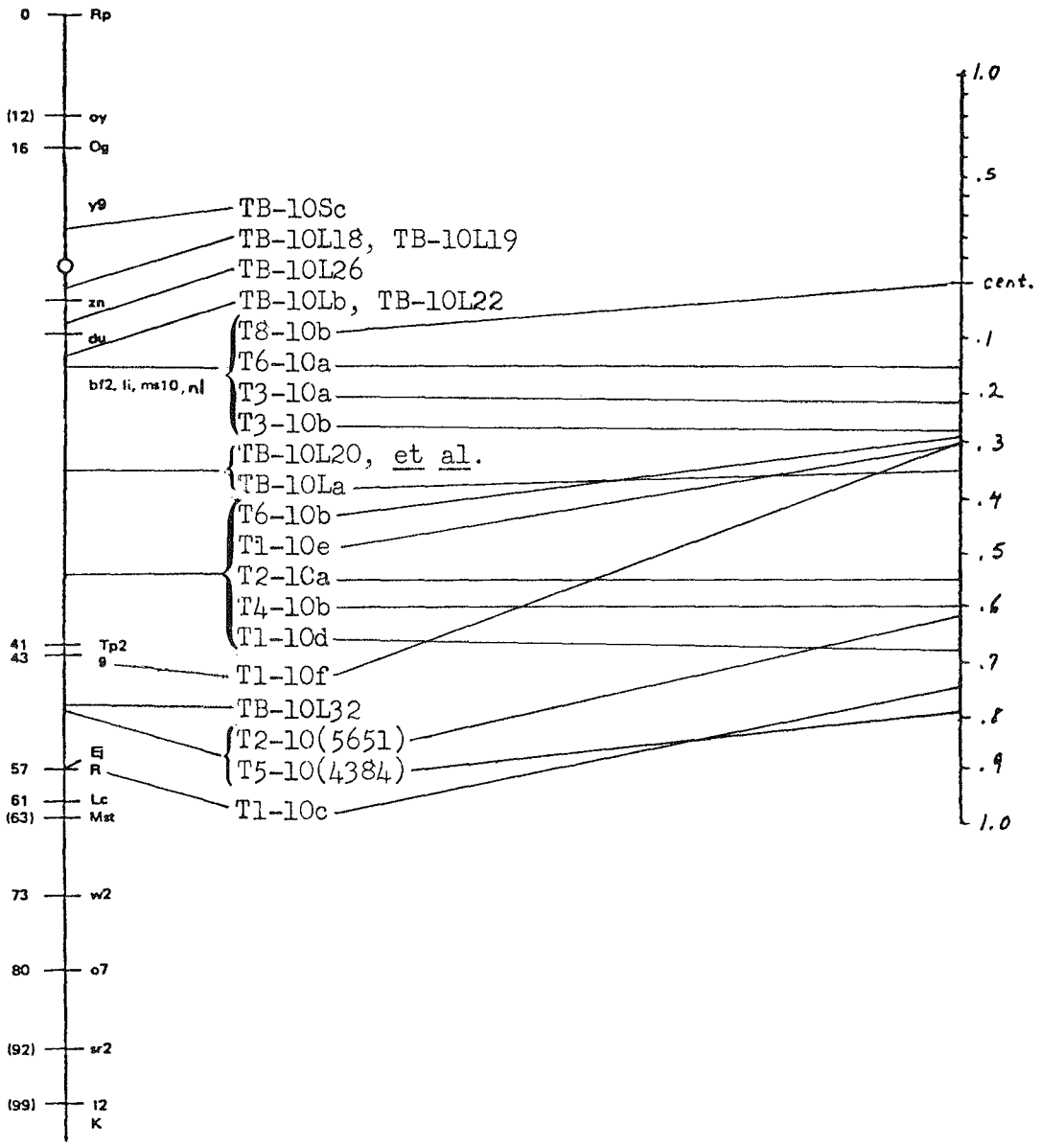


Dt_3 —— **Distal to .3**
E_1
g_2
va_1


8
TB-8La
T3-8a
T6-8a
0
TB-8a
v16
14
m-8
28
1
1.0
.5
Cent.
.1
.2
.3
.4
.5
.6
.7
.8
.9
1.0


9
K
1.0
.5
Cent.
.1
.2
.3
.4
.5
.6
.7
.8
.9
1.0
DfK9
TB-9Sb-4L6504
Df thread
Df 1 chromom.
Df 6 chromom.
T4-9(6222)
T8-9(4453)
In9a
TB-9Sb
T8-9(5300)etal
TB-9Lc
T1-9a
T1-9c
TB-9La
In9a
0 Dt
pyd
7 Yg2
wd
26 C
29 sh
31 bz
40 Mr
44 bp
53 lo2
ga8
TB-9l
59 wx
62 d3
TB-9k
(64) pg12
(65) sr
66 v
67 ms2
69 gl15
TB-9r
79 bk2
104 Wc
134 Bf
138 brn4

10


0 Rp
(12) oy
16 Og
y9
zn
du
bf2, li, ms10, nl
41
43 Tp2
g
57 Ej
R
61 Lc
(63) Mst
73 w2
80 o7
(92) sr2
(99) 12
K
TB-10Sc
TB-10L18, TB-10L19
TB-10L26
TB-10Lb, TB-10L22
T8-10b
T6-10a
T3-10a
T3-10b
TB-10L20, et al.
TB-10La
T6-10b
T1-10e
T2-10a
T4-10b
T1-10d
T1-10f
TB-10L32
T2-10(5651)
T5-10(4384)
T1-10c
1.0
.5
cent.
.1
.2
.3
.4
.5
.6
.7
.8
.9
1.0

IV. NEWS LETTER FILES

Following are additions and corrections for the "archival" list of materials of the Maize News Letter and related cooperation. C. R. Burnham provided several very helpful leads and items; a scan of the papers of E. G. Anderson identified some others. Starred items (*) have not been located; if you have other files or more information, please let me know. The previous list is in MNL 50:2-4.

Date	From	Pp.	Contents
7 Mar 23	R. A. Emerson, Cornell	6	Factor notation
*23 Nov 29	?	?	Summarization of linkage (Ref. 19 Dec 29)
19 Dec 29	G. W. Beadle, Cornell	1	Summarization of linkage
5 Feb 30	G. W. Beadle, Cornell	1	Summarization of linkage
* ?	?	?	"Cooperation planned at Sixth Congress" (Ref. MNL 14:56; is this a written item?)
* ?	?	?	Grant support (Ref. MNL 14:56)
* 1 Apr 34	?	?	"Rockefeller Grant available" (Ref. MNL 14:56; is this a written item?)
17 Sep 35	R. A. Emerson, Cornell	1	Disease resistance tests
5 Jan 37	D. G. Langham, Cornell	1	(Corrected date)
1 Apr 41	A. C. Fraser, Cornell	56	(Corrected date)
* ?	?	?	Call for v. 18 (assumed)
31 Jan 44	R. A. Emerson, Cornell	32	(Corrected date)
* ?	?	?	Call for v. 19 (assumed)
* ?	?	?	Call for v. 20 (assumed)
* ?	?	?	Call for v. 24 (assumed)
* ?	?	?	Call for v. 25 (assumed)
17 Mar 52	H. H. Smith, Cornell	76	(Corrected date)
15 Mar 56	M. M. Rhoades, Illinois	164	(Pp.)

E. H. Coe, Jr.

55 YEARS AGO

The letter from R. A. Emerson on the following pages has been reproduced from a carbon copy in E. G. Anderson's files. Dated March 7, 1923, it poses a need for consideration of genetic symbolization and nomenclature among corn geneticists, and requests their comments and ideas.

Department of Plant Breeding

New York State College of Agriculture
Cornell University
Ithaca, N. Y.

March 7, 1923

To Students of Corn Genetics : -

Recent developments in corn genetics studies make it important that those of us who are working with this material agree on a factor notation that shall be as nearly uniform as possible. It seems wise to follow the notation used by the Drosophila workers, in so far as practical, tho, in some respects, their usage is perhaps no more nearly consistent than our own.

Some of the problems that deserve attention are outlined below.

We now have several instances of practically indistinguishable phenotypes that are differentiated from the normal by distinct genes. We have heretofore designated white seedling by w, but there are certainly six and presumably many more non-allelomorphic genes for white seedling. Shall we attempt to find distinctive names for all these or call them all w and identify them further by number ? If the latter plan, which I personally prefer, is adopted, shall we use Arabic or Roman numerals and shall they be written as subscripts or full size numerals: w1, w2, w3; wI, wII, wIII; w_1, w_2, w_3; w_I, w_{II}, w_{III} ?

It has been proposed that Roman numerals, either as subscripts or not, be used for any such gene which has not as yet been placed in a linkage group and that Arabic subscripts indicating the group number be employed for such as have been so placed. As more

and more of these genes are located in linkage groups, the Roman numerals would be replaced by Arabic subscripts. Thus, w_I (or w_1), w_2, w_{VI} (or w_{VI}) might ultimately be written as w_7, w_2, w_{10}, etc. Is there any advantage in indicating this much of the linkage relations of genes by the gene symbol ? Is there any more reason for showing something of a gene's linkage relations by its symbol than for indicating by a characteristic suffix that a gene is one of duplicate or of complementary factors ? Is there any more reason for indicating group relations of such genes as the several ones for white seedling than for liguleless leaf, shrunken endosperm, etc., say: lg_5, sh_2, etc. ?

In case group relations are to be indicated by a numerical subscript, what shall be done with the three w genes that are now almost certainly known to belong to the Y - Pl group ? Shall we use the symbols w_{2a}, w_{2b}, w_{2c} (assuming that the Y - Pl group is regarded as the second one) ?

If numerical subscripts or full size figures are used to identify such genes as those for white seedling, we should doubtless adopt the same plan for defective endosperm (de), zebra striping (zb), zigzag culm (now z and zg), virescent seedling (v), pale green seedling (pg), piebald (pb), tassel seed (now ts and to, with one or more unnamed).

But how about colorless aleurone ? Should we abandon the symbols a, c, r, I, that have an established place in the literature of corn genetics and use say: a_1, a_2, a_3, A_4, or perhaps c_1, c_2, c_3, C_4 ? If group relations were to be indicated here, we might have a_7, a_{1a}, a_3, A_{1b} or c_7, c_{1a}, c_3, C_{1b}. Our present An and Rr pairs affect plant, silk, and anther, and pericarp colors in

various ways while Co and Ii are not known to do so, which may be reason enough for not bringing the aleurone factors under this scheme. The relation of Aa to plant, silk, anther, pericarp, and aleurone colors is a simple one and, therefore, presents no serious difficulties, but the R^r, R^g, r^r, r^g, r^{ch} series would be difficult unless the interpretation of multiple allelomorphism, be changed to one of close linkage of several distinct genes.

I assume that the plan outlined in this letter need not change our symbols for the several forms of dwarf plant; dwarf (d), brachytic (ba), nana (na), brevis (br), etc., because these dwarfs are sufficiently unlike morphologically to be more or less readily distinguishable. If, however, as seems probable now, there are two indistinguishable dwarfs, they will, I assume, have to be known as d_1 and d_2, or perhaps as dI and dII until their group relations are established.

There is being prepared now a general paper on corn linkage in which a summary of all available information is to be presented. It seems wise to me to number the linkage groups, whether or not any genes are to be given group numbers. In what order shall the groups be numbered ? Shall priority of publication of any linkage determine the numerical order ? Or shall the order be determined arbitrarily ? While priority of publication might seem the better basis, there are certain difficulties. Both Eyster and Hutchison, while not numbering the groups, discussed them in the supposed order of priority, as follows: C - wx, g - R, su - Tu, B - lg, Y - Pl, P - f. Jones gave the same order for the first three of this group. But Kempton in March 1917, published a brief

paper showing unmistakably a linkage between yellow endosperm and white seedling. Whether this paper was overlooked by Eyster, Hutchison, and Jones or whether it was left out of account because of doubt as to what white seedling gene was concerned or as to whether one or more than one yellow gene, as suggested by Kempton, was involved, makes little difference. The point is that if priority were taken as the basis for numbering the groups and they had been numbered by Eyster and Hutchison, someone might now or later claim the right to change the Y - Pl group from No. 5 to No. 2. Or if we now, on the basis of priority of publication, decide to call the Y - Pl group No. 2, what is to prevent someone later claiming that the g - R group is No. 1, thus making C - wx and Y - Pl Nos. 2 and 3, respectively, on the basis that what has recently been regarded as a multiple allelomorph series, namely, R^r, R^g, r^r, r^g, r^{ch}, etc., is better interpreted as very close linkage between genes for aleurone, leaf, silk, anther, and pericarp color, and citing Webber's 1906 paper as the first one on corn linkage ? I would personally prefer to have the corn-genetics men in this country adopt group numbers arbitrarily and then adhere to them rather than to have the numbers changed later by the discovery of some earlier paper. I suggest, therefore, that we number the groups in the order given by Eyster and by Hutchison, as follows:

1- C - wx
2- g - R
3- su - Tu
4- B - lg
5- Y - Pl
6- P - f

These are the only published groups so far as I know, except gs - zm to which apparently an also belongs, and it is not certainly known that this group may not belong to one of the six listed above. The same is true of d - pg, fr - gl - v, and doubtless others. Perhaps the A - v group (Stroman) should be No. 7, because A almost certainly does not belong with any of the six groups listed above. It might be better, however, to assign no numbers to groups other than the six listed above until the newer groups have been tested further.

Another problem is bothering some of us. Shall we continue to use bi-literal symbols for genes as we have usually done in the past, or adopt the recommendation of the Naturalists' committee to use single-letter symbols ? It is my understanding that the practice of Genetics of setting the second letter of a bi-literal symbol as a subscript was adopted by Dr. Shull because of this rule. Thus, our B (plant color), bl (blotched leaf), bc (brachytic culm), bh (blotched aleurone), etc., which have no close relation one to another, all become B symbols with literal subscripts to distinguish one from another. I fear this usage will be confusing. Would it not be likely to give the idea that the several "B" symbols stand for similar phenotypes just as our proposed W_1, W_2, W_3, etc., do ? If the corn men desire to stick to the use of bi-literal symbols, we shall probably have to refrain from publishing in Genetics or have our symbols changed by the editor when papers are published there. It is interesting to note that, while Genetics has been consistent in setting the second letter of two-letter symbols as subscripts in the text, it has allowed the form we are accustomed to/stand in drawings, as seen in some Drosophila papers.

It is true that certain difficulties are avoided by making the second letter of a bi-literal symbol a subscript. If a genetic formula be written, thus, blgpr, it may be wholly unintelligible to us. Following the usage insisted on by Genetics, it would become $bl_g p_r$, $bl_g pr$, or $blg p_r$, etc., which mean very different types. We have accomplished the same thing as you know by writing these formulae bl g pr, b lg p r, b l g pr, etc. I personally very much prefer our usage to that of Genetics, but if the majority of corn men think best to adopt the plan followed by Genetics, I shall use it. I hope that we can determine soon what is best for us to do for I have a paper in press now that I shall want to revise if we adopt the Genetics plan.

I am sending this to a considerable number of corn-genetics workers. When I have received replies from the majority, I may want to refer some of our problems to the chairman of the Naturalists' committee with the suggestion that he consider the advisability of referring it to the committee for consideration.

Sincerely,

RAE:V (signed) R. A. Emerson

V. REPORT OF MAIZE GENETICS COOPERATION STOCK CENTER

During 1977 the Maize Genetics Cooperation received 143 requests for maize genetic stocks. There were 107 (75%) domestic and 36 (25%) foreign requests. Requests from geneticists constituted 56%, plant breeders 15%, plant physiologists 16%, and educational 13%. A total of 1407 seed packets were sent to fill these requests, 64% for domestic and 36% for foreign use. The distribution of seed packets by use was geneticists 74%, plant breeders 7%, plant physiologists 9%, and educational 10%.

Each year stocks are sent in by maize geneticists to become a part of the collection. The following have been received during the past several years and are on the stock list or will be placed there when adequate seed is available.

Trait	Symbol	Contributor
Alcohol dehydrogenase	Adh-1S; Adh1-P Adh-1F825; Adh2-Gamma	M. Freeling
Disease lesion mimic-1	Les	M. G. Neuffer
Disease lesion mimic-2	Les2	"
Expanded glume	eg	C. R. Burnham
Golden plant-5	g5	J. B. Beckett
Grassy tiller	gt	D. Shaver
Floury-3	fl3	O. E. Nelson, Jr.
Indeterminant gametophyte	ig	J. L. Kermicle
Lethal leaf spot	Lls	F. Troyer
Lethal ovule	lo2	O. E. Nelson, Jr.
Opaque-5	o5	D. Robertson
Opaque-7	o7	K. McWhirter
Oil yellow (dominant allele)	Oy-yg	M. G. Neuffer
Helminthosporium maydis resistant	rhm	D. Smith
Multiple marker stock	bz2 a c2 a2pr y c bz wx r	E. H. Coe, Jr.
Corn-Tripsacum translocation	22^{T}+++ / ws3 lg1 gl2	M. Maguire
Endosperm mutants converted to W23 and W64a		D. Garwood
Alleles at the waxy locus		O. E. Nelson, Jr.
B-A translocations		J. B. Beckett
Trisomics 1, 2, 4, 6, 7		G. Doyle
Tetraploid stocks		D. Shaver

Chromosome Aberrations - Supplied by D. B. Walden; originally from R. Morris:

1-7-3691-1 (1L.06;7L.81)
2-7-3692-1 (2S.10;7L.02)
3-8-3687 (3L.25;8L.88)
4-7-3686 (4S.81;7S.06)
5-7-3699 (5S.46;7L.07)
5-7-3703 (5S.67;7L.81)
5-10-3693 (5L.67;10L.51)
6-9-3698 (6L.11;9L.28)
8-10-3697 (8L.10;10L.18)
9-10-3688 (9S.49;10L.02)

A list of reciprocal translocation stocks available from the Maize Cooperation is in News Letter 43, 1969, or is available upon request.

Requests for stocks and correspondence relative to the stock center should be addressed to:

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

Catalogue of Stocks

Chromosome 1

sr zb4 P-WW
sr P-WR
sr P-WR an gs bm2
sr P-WR an bm2
sr P-RR gs bm2
sr P-WR bm2
vp5
zb4 ms17 P-WW
zb4 ms17 P-WW rs2
zb4 ts2 P-WW br f bm2
zb4 ts2 P-WW bm2
zb4 P-WW
zb4 P-WW br
zb4 P-WW br f bm2
zb4 P-WW bm2
ms17
ts2 P-RR
ts2 P-WW bm2
ts2 br f bm2
P-CR
P-RR
P-RW
P-CW
P-MO
P-VV
P-RR as br f an gs bm2
P-RR br f an gs bm2
P-RR an ad bm2
P-RR an gs bm2
P-RR ad bm2
P-WR an Kn bm2
P-WR an ad bm2
P-WR an bm2
P-WR ad bm2
P-WR br Vg
P-WR br f gs bm2
P-WW rs2
P-WW rs2 br f
P-WW as br f bm2
P-WW hm br f
P-WW br f ad bm2
P-WW br f bm2
P-WW br f an gs bm2
P-WW br Vg
as
as rs2
rd-Hy
br f
br f Kn
br f Kn Ts6
br f Kn bm2

Chromosome 1 (continued)

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

Chromosome 2

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

Chromosome 2 (continued)

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

Chromosome 2 (continued)

LES
TB-3La-2S6270
Primary Trisomic 2

Chromosome 3

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

Chromosome 3 (continued)

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

Chromosome 4

Rp4
Ga
Ga su

Chromosome 4 (continued)

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

Chromosome 4 (continued)

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

Chromosome 5

lu
lu sh4
ms13
gl17
gl17 A2 pr ; A C R
gl17 a2 ; A C R
gl17 a2 bt ; A C R
gl17 a2 bt v2 ; A C R
A2 vp7 pr ; A C R
A2 bm bt pr ys ; A C R
A2 bm pr ; A C R
A2 bm pr ys ; A C R
A2 bm pr ys eg ; A C R
A2 bm pr v2 ; A C R
A2 bt v3 pr ; A C R
A2 bt pr ; A C R
A2 bt pr ys ; in A C R
A2 v3 pr ; A C R
A2 pr ; A C R
A2 pr na2 ; A C R
A2 pr ys ; A C R
a2 ; A C R
a2 ; A C R B Pl
a2 bm bt bv pr ; A C R
a2 bm bt pr ; A C R
a2 bm bt pr ys ; A C R
a2 bm pr ys ; A C R
a2 bm pr v2 ; A C R
a2 bt v3 pr ; A C R
a2 bt pr ; A C R
a2 bt v2 ; A C R
a2 v3 pr ; A C R
a2 pr ; A C R
vp2
vp2 pr
vp2 gl8
vp7
bm yg
bt
ms5
v3

Chromosome 5 (continued)

td ae
ae
sh4
gl8
na2
lw2
ys
eg
v2
yg
ms13
v12
br3
TB-5La
TB-5Lb
Primary Trisomic 5

Chromosome 6

rgd po y
rgd po Y
rgd Y
po = ms6
po y pl
po y Pl
po Y pl
y = pb = w-m
y l10
y l11
y l12
y w15
y pb4
y pb4 pl
y pb4 Pl
y si
y wi Pl
y Dt2 ; a-m A2 C R
y pg11 ; Wx pg12
y pg11 wi ; wx pg12
Y pg11 ; Wx pg12
y pg11 ; wx pg12
Y pg11 ; wx pg12
y pl
y Pl
y Pl Bh ; c sh wx A A2 R
y su2
Y l10
Y l12
Y pb4
Y wi pl
Y wi Pl
Y su2
wi

Chromosome 6 (continued)

Pl Dt2 ; a-m A2 C R
pl sm ; P-RR
Pl sm ; P-RR
Pl sm py ; P-RR
Pt
w
w14
ms6
2NOR ; a2 bm pr v2
Primary Trisomic 6

Chromosome 7

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

Chromosome 7 (continued)

va
Dt3 ; a-m A2 C R
v*-8647
yel*-7748
TB-7Lb (7L.30)
Primary Trisomic 7

Chromosome 8

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

Chromosome 9

yg2 C sh bz ; A A2 R
yg2 C sh bz wx ; A A2 R
yg2 C-I sh bz wx ; A A2 R
yg2 C sh bz wx K-L9 ; A A2 R
yg2 C bz wx ; A A2 R
yg2 c sh bz wx ; A A2 R
yg2 c sh wx ; A A2 R
yg2 c sh wx gl15 ; A A2 R
yg2 c sh wx gl15 K-L9 ; A A2 R-g
yg2 c bz wx ; A A2 R
wd-Ring C-I ; A A2 R
C sh bz ; A A2 R
C sh bz wx ; A A2 R
C-I sh bz wx ; A A2 R
C sh bz wx gl15 bm4 ; A A2 R
C sh ; A A2 R
C sh wx ; A A2 R
C wx ar ; A A2 R
C-I sh wx v ; A A2 R
C sh wx K-L9 ; A A2 R
C sh ms2 ; A A2 R
C bz Wx ; A A2 R
C Ds Wx ; A A2 R y
C Ds wx ; A A2 R pr
C-I Ds wx ; A A2 R
C-I ; A A2 R
C ; A A2 R
C ; A A2 R B Pl
C wx ; A A2 R

Chromosome 9 (continued)

C wx ; A A2 R B Pl
C wx ; A A2 R b Pl
C wx ; A A2 R B pl
C-I wx ; A A2 R y
C-I wx ; A A2 R y B pl
C wx ar da ; A A2 R
C wx v ; A A2 R
C wx v ; A A2 R Pl
C wx gl15 ; A A2 R
C wx gl15 ; A A2 R pr
C wx Bf ; A A2 R
c sh bz wx ; A A2 R y
c sh wx ; A A2 R
c sh wx v ; A A2 R
c sh wx gl15 ; A A2 R
c sh wx gl15 bk2 ; A A2 R
c sh wx gl15 Bf ; A A2 R
c sh wx bk2 ; A A2 R
c ; A A2 R
c wx ; A A2 R y
c wx v ; A A2 R
c wx gl15 ; A A2 R
c wx Bf ; A A2 R
c wx bk2 ; A A2 R
sh
sh wx v
sh wx d3
sh wx pg12 gl15 ; y pg11
lo2
wx*
wx-a
w11
wx d3
wx d3 w11
wx d3 v gl15
wx d3 gl15
Wx pg12 ; y pg11
wx pg12 ; y pg11
Wx pg12 ; Y pg11
wx pg12 ; Y pg11
wx pg12 bm4 ; y pg11
wx v
wx bk2
wx bk2 bm4
wx Bf
wx Bf bm4
d3
v
gl15
gl15 Bf

* Additional waxy alleles available from collection of O. E. Nelson.

Chromosome 9 (continued)

gl15 bm4
bk2 Wc
Wc
bm4
bm4 Bf
16
17
yel*-034-16
w*-4889
w*-8889
w*-8951
w*-8950
w*-9000
TB-9La (9L.40)
TB 9Sb (9S.40)
Primary Trisomic 9

Chromosome 10

oy
oy R ; A A2 C
oy bf2
oy ms11
oy bf2 R ; A A2 C
oy bf2 ms10
oy zn R ; A A2 C
oy du R ; A A2 C R
oy du r ; A A2 C
oy sr2
oy zn
Og
Og du R ; A A2 C
ms11
ms11 bf2
bf2
bf2 zn
bf2 li g r ; A A2 C
bf2 g R sr2 ; A A2 C
bf2 g r sr2 ; A A2 C
bf2 r sr2 ; A A2 C
nl zn g R ; A A2 C
nl g R ; A A2 C
nl g r ; A A2 C
nl g R sr2 ; A A2 C
y9
li zn g r ; A A2 C
li g R ; A A2 C
li g r ; A A2 C
li g r v18 ; A A2 C
li g R v18 ; A A2 C
ms10
du

Chromosome 10 (continued)

du v18
du o7
du g r ; A A2 C
du sr2
zn
zn g
zn g R sr2 ; A A2 C
zn g r ; A A2 C
Tp2 g r ; A A2 C
g R sr2 ; A A2 C
g r ; A A2 C
g r sr2 ; A A2 C
g r sr2 l ; A A2 C
g R-g sr2 ; A A2 C
g R-g sr2 v18 ; A A2 C
g R-g K10 ; A A2 C
g R-g sr2 ; A A2 C
g R-r K10 ; A A2 C
g r-r sr2 ; A A2 C
Ej r-r ; A A2 C
Ej r-r sr2 ; A A2 C
r sr2 l ; A A2 C
R-g ; A A2 C
r-g sr2 ; A A2 C
r K10 ; A A2 C
r-g ; A A2 C
r-r ; A A2 C
R-mb ; A A2 C
R-nj ; A A2 C
R-r ; A A2 C
R-r (Boone) ; A A2 C
R-lsk ; A A2 C
R-sk-mc.2 ; A A2 C
R-sk ; A A2 C
R-st ; A A2 C
R-st Mst
R-st Mst o7
Lc
w2
w2 l
o7
o7 ; o2
l
v18
Mst
l yel*-5344
yel*-8721
yel*-8454
yel*-8793
TB-10La (10L.35)
TB-10Sc
Primary Trisomic 10

Unplaced Genes

dv
dy
el
h
l4
LES2
Rs
v13
ws ws2
ub
zb
zb2
zb3
zn2
l*-4923
nec*-8376

Multiple Gene Stocks

A A2 C R-g Pr B Pl
A A2 C R-g Pr B pl
A A2 C r-g Pr B Pl
A A2 C r-g Pr B pl
A A2 c R-g Pr B pl
A A2 C R-r Pr B Pl
A A2 C R-r Pr B pl
A A2 C R-r Pr b Pl
A A2 c R-r Pr B Pl
A A2 C r-r Pr B Pl
A A2 c r-r Pr B Pl
A A2 C R Pr
A A2 C R Pr wx
A A2 C R Pr wx gl
A A2 C R Pr wx y
A A2 C R pr
A A2 C R pr y gl
A A2 C R pr y wx
A A2 C R pr y wx gl
A A2 c R Pr y wx
A A2 C r Pr y wx
bz2 a c2 a2 y c r
a su A2 C R
bm2 lg a su pr y gl j wx g
colored scutellum
lg gl2 wt ; a Dt A2 C R
lg su bm2 y gl j
su y wx a A2 C R-g pr
y wx gl
hm hm2
ts2 ; sk

Popcorns

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

Exotics and Varieties

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

Tetraploid Stocks

P-RR
P-VV
Ch
B Pl
a A2 C R Dt
su
pr ; A A2 C R
y
gl
ij
Y sh wx
sh bz wx
wx
g A A2 C R
A A2 C R B Pl

Cytoplasmic Steriles and Restorers

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

Waxy Reciprocal Translocations

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

Inversions

Inv.1a (1S.30-L.50)
Inv.1c (1S.35-L.01)
Inv.1d (1L.55-L.92)
Inv.1L-5131-10 (1L.46-L.82)
Inv.2a (2S.70-L.80)
In2d Inv.2-3713 (2S.93-L.65)
In2e Inv.2-3778 (2S.44-L.84)
Inv.2S-L8865 (2S.06-L.05)
In2h 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)
In4d Inv.4b (4L.40-L.96)
Inv.4c (4S.86-L.62)
Inv.4e (4L.16-L.81)
Inv.5-8623 (5S.67-L.69)
Inv.6-8452 (6S.77-L.33)
Inv.6-8604 (6S.85-L.32)
Inv.6-3712 (6S.76-L.63)
Inv.7b (7S.32-L.30)
Inv.7L-5803 (7L.17-L.61)
Inv.7-8540 (7L.12-L.92)
Inv.7-3717 (7S.32-L.30)
Inv.8a (8S.38-8L.15)
Inv.9a (9S.70-L.90)
Inv.9b (9S.05-L.87)
Inv.9c (9S.10-L.67)

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E. H. Coe, Jr.

ERRATA

50:28-29 Replace W59M with W182BN
51:5-6 " " " "
51:71 3d paragraph should read:

"Heritability estimates were obtained according to the equation $h^2 = (x_o - \bar{x})/(x_p - \bar{x})$, where x_o = mean of the selected population, x_p = mean of the individuals selected from the original source population, and $\bar{x}$ = mean of the original source population."

Dr. R. G. Palmer calls attention to Plant Breeding Symposium II, sponsored by Iowa State University at the new Iowa State Center, to be held March 12-16, 1979. The symposium will review plant breeding advances of the past 15 years, and evaluate areas of future potential. Ten half-day sessions will consider the following topics:

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2. Exotic Germplasm; Resources and Utilization
3. Application of Tissue Culture to Plant Improvement
4. Morphological and Physiological Traits
5. Selection and Breeding Methods
6. Chromosomal and Cytoplasmic Manipulations
7. Breeding for Stress Environments
8. Pest Resistance: Pathology, Entomology
9. Development of Plants for Multiple-Cropping Systems
10. Improvement of Nutritional Quality

For registration information please contact: Dr. K. J. Frey, Agronomy Dept., Iowa State University, Ames, Iowa 50011; telephone is (515) 294-7607.

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