MAIZE GENETICS COOPERATION

NEWSLETTER

78

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NOTE: The 2005 Maize Meeting will be held at Lake Geneva, Wisconsin, March 10-13.

I. Foreword

The Maize Genetics Cooperation Newsletter exists for the benefit of the maize community as an informal vehicle for communication. Its inception and continuation has been to foster cooperation among those interested in investigating maize. This cooperation has distinguished our field from others and as a consequence has moved it forward at a pace greater than would have occurred otherwise. Your submissions are encouraged to disseminate knowledge about our field that might otherwise go unrecorded.

Because maize is both a commercial species and a genetic model system, the danger exists that the sharing of research materials might be diminished. It is imperative for us to work together to prevent this from occurring. Certainly, basic findings should be transferred to the industrial sector and basic advances in industry should be shared with the academic community for the benefit of both. Published materials must be shared for research purposes with the only restriction being against commercial use.

We remind the readers that contributions to the Newsletter do not constitute formal publications. Citations to them should be accompanied by permission from the authors if at all possible. Notes can be submitted at any time and are entered into MaizeDB. The deadline for the next print copy, volume 79, is January 1, 2005. Electronic submission is encouraged by sending your contributions as attachments, or as text of an email, to Newsletter@chaco.agron.missouri.edu. Submissions must require minimal editing to be accepted.

We encourage the community to carry studies of general scientific interest to the formal literature. However, there is a great need to share technical tips, protocols, mutant descriptions, map information, ideas and other isolated information useful in the lab and field.

As in the past, Shirley Kowalewski has been responsible for assembly and correcting of the copy. She has performed this task with speed, precision and a great sense of humor. The maize community owes her much gratitude for her continued service in this capacity.

Mary Polacco
James A. Birchler
Co-editors

AMES, IOWA
Iowa State University

Analysis of the maize *Myb* gene superfamily: conserved motifs and functional characterization

--Jiang, C, Peterson, T

Myb proteins are defined by a highly conserved DNA-specific binding domain termed *Myb*, which is composed of approximately 50 amino acids with constantly spaced tryptophan residues. *Myb* genes encode one of the largest families of diverse transcription factors in plants. However, with the exception of a few well-studied cases, little is known about the functions of most *Myb* genes. In our study, we attempted to classify and predict the functions of *Myb* genes from maize and other plants. First, we clustered closely-related *Myb* genes into subgroups on the basis of similarity and phylogeny. We found that those *Myb* genes with similar function were clustered within the same subgroup by consulting the related published literatures. Interestingly, AtMyb33, 65, 101, 104 and At3g60460 were complementary, with few mismatches, to *Arabidopsis Myb* microRNA (noncoding RNA) miR159 (Rhoades et al., Cell 110:513-520, 2002). These five *Arabidopsis Mybs* are located in the same subgroup in our analysis. This clustering further provides a line of evidence for the reliability of the subgroup designations in our analysis. In addition, gene structure analysis shows that *Myb* genes in the same subgroup have conserved exon-intron structures and intron phases. Second, we used the motif-searching program MEME to identify conserved motifs in the C-terminal regions of the Myb proteins in each subgroup. We identified 38 candidate motifs with e-value <= 1e-10. Motifs were subjected to the similarity score test program PlotSimilarity from the GCG package from Genetics Computer Group (http://www.accelrys.com/products/gcg_wisconsin_package/), and the nonsynonymous substitution test using program YN00 from the PAML package (Yang and Nielsen, Mol Biol Evol, 17:32-43, 2000). These tests eliminated 20 putative motifs, leaving a total of 18 qualified motifs. These 18 motifs were found to be specific to each subgroup. To further test the specificity of these motifs, we performed a homology search in the Swiss-Prot database and the EST database from GenBank. Only genes containing *Myb* domains were detected in both datasets. For some motifs, no hits were detected in the EST search, possibly due to low expression levels. Finally, we predicted the functions of a large proportion of previously uncharacterized *Myb* genes. The resulting functional classification table may provide a useful starting point for determination of *Myb* gene function (Jiang, Gu and Peterson, submitted).

BELGRADE, ZEMUN, YUGOSLAVIA
Maize Research Institute (Zemun Polje)

White dent population from the Yugoslav variety collection carries the *Ga* gene

--Vancetovic, J, Vidakovic, M, Vidakovic, M, Rosulj, M

Since the discovery of the *Ga* genes of incompatibility in maize (Demerec, Zeits i Abst u Verer 50:281-291, 1929; Schwartz, Proc. Natl. Acad. Sci. USA 36:719-724, 1950) their importance in protecting specialty types of corn (popcorn, white corn, etc.) from the other predominant types has been recognized. A carrier of the *Ga* gene can pollinate any other corn, but can be pollinated only by a carrier of the same dominant allele. It should be noted that the allele *Ga1-S* shows partial dominance (Nelson, Genetics 37:101-124, 1952). Once introduced into a population, the *Ga* gene, being dominant, spreads very quickly and soon reaches homozygosity. Incorporating *Ga* genes into the specialty types of corn is a prerequisite for keeping their purity during mass production.

Here we report the discovery of a white dent population carrying the *Ga* gene in maize.

The accession number 1862 of the Yugoslav variety collection – population Beli rani brzak, from Bela Crkva, Banat, was included in a search for the restorer cytoplasm (Vidakovic et al., p. 138 in XVIIth Conference on Maize and Sorghum EUCARPIA, 1996). In 1993, two plants from this population were crossed by hand pollination, as females, with the tester B73 Ms10/ms10. A very small amount of seed was recorded from this cross. The next year, in the isolation field, plants from those two crossed ears were backcrossed with the Ms10/ms10 tester as females. Again, the number of successfully crossed ears was small; for the original ear 5, and for the second 3, instead of about 20, as was usually obtained in the rest of the experiment. A mixture of approximately the same amount of seed from backcrossed ears was made, and in 1995 the test for the presence of the restoring cytoplasm of about 60 plants was planted. On average, 1/8 ms10/ms10 sterile plants was expected from this type of cross.

The test was 100% fertile, indicating restoring cytoplasm is present. But in the further stages of testing (selfing of individual plants and outcrossing them to the ms10/ms10 tester), this hypothesis was rejected.

What was noticed is that the test was very late in maturity (the latest of all), and above all that all ears in the open pollination were white kerneled. How was this possible, since the surrounding maize was mostly yellow kerneled, and both testers, for crossing and backcrossing, were all yellow kerneled?

We supposed that the population might contain one of the *Ga* genes, and that in the first cross, since only a small amount of seed was obtained, unintended selfing occurred instead of crossing, while in the backcross, as the population is very late, even one badly cut tassel could pollinate the rest of the plants.

To test this hypothesis, in 1996 we took the original seed from the accession, and made pedigree crosses (plant to plant) with yellow dent maize of BSSS and Lancaster origin. Where the population was used as a male, selfing of the particular plants was made. Results of crosses indicated the presence of the *Ga* gene. When population 1862 was used as a female, only a few seeds were obtained from all of the crosses, while when used as a male, a full seed set was obtained on the BSSS and Lancaster lines.

In 1997, an additional testing was made with the popcorn lines BP1 and BP2, known for carrying the *Ga* genes, as well as with the stocks provided by the courtesy of Dr. Marty Sachs, Urbana, Illinois, to find which of the *Ga* genes is in question. Reciprocal crosses were made with four selfed progenies (from 1996) of the original population, a small amount of seed obtained from the cross of the population as a female with yellow dent corn in 1996, yellow dent lines B-84 (BSSS) and MV^2-4-2 (Lancaster), and the following US stocks: *(Ga1-S Su1)self*, *(Ga1 su1)self*, and *(Ga1 M1 Su1)self*. Success of the crosses is given in percentages in Table 1.

Table 1. Success of the reciprocal crosses with the investigated population and different sources of *Ga* genes. The seed set of the appropriate cross is given in %, except for the small quantities, where it is given in kernels (ker.).

Male*	1	2	3	4	5	6	7	8	9	10	11	12
Female*												
1					1 ker.		80	100	15 ker.	100	100	100
2					0	0	100	100	0	80	100	100
3					0		80	100	0	30	85	80
4					0	40	100	100	10 ker.	100	90	100
5	100	100	75				95					
6	60	3 ker.	75	60			85					
7	100	100	100	100	70	90						
8	100	70	100	100								
9		100	100	100								
10	100	80	100	90								
11	80	80	80	10								
12	80	100	100	80								

Genotypes are: 1-1862/1; 2-1862/2; 3-1862/3; 4-1862/4; 5-B-84; 6-MV^2-4-2; 7-1862xYellow; 8-(Ga1-S Su1)self*, 9-*(Ga1su1)self*, 10-*(Ga1M1Su1)self*, 11-BP1, 12-BP2
1-4 are selfed progenies from the pop. 1862. 5 and 6 are yellow dent maize lines. 7 is from the few kernels obtained from the cross of pop. 1862 and yellow dent maize. 16 and 17 are the ZP popcorn lines.

Based on the results shown in Table 1, it seems that our population contains the gene *Ga1-S* (Marty Sachs, personal communications).

A sample of this population was sent to Urbana, Illinois in 1996.

It should be noted that the population is not yet homozygous for the *Ga* gene. In 2000, we made a series of crosses of 12 selfed families from the population, as females, with the red kerneled line as a male. Among 12 families, only one exhibited a seed set of 285 yellow kernels. But, since once a *Ga* gene enters a population, it spreads to total homozygosity (Nelson, 1952), so probably after a few additional multiplications the gene in this population will be fixed.

From a breeding standpoint, this variety is a potential source for extracting medium-to-late white kerneled inbred lines, protected from the pollen of yellow-kerneled materials.

F2 population size for resistance to root and stalk lodging in maize

--Stojkov, S, Rosulj, M, Stankovic, G

Root and stalk lodging is one of the most important traits in commercial maize breeding. One of the main causes of increased root and stalk lodging is the presence of pathogens from the species *Fusarium*, so the maize breeder must pay special attention to resistance to these pathogens during the process of selection. An F2 population is a generation of maximum gene recombination. The influence of F2 population size has been studied in many papers for grain yield and other traits, but there is no literature on the optimal size of the F2 population necessary to develop hybrid combinations resistant to pathogens of maize root and stalk lodging. The objectives of this study were to estimate changes in genetic parameters with changes in population sizes and to obtain an F2 population size adequate for traits such as resistance to root and stalk lodging

The genetic material evaluated in the present study was F2 population S-5892 derived from a cross of two inbred lines, L588 and B92. Inbred line L588 is a dent type, derived from crossing B84 x Yugoslavian germplasm. FAO maturity group is 550. L588 has good general combining ability and is tolerant to root and stalk lodging. Public inbred line B92 is a semi-dent type. FAO maturity group is 700. B92 has excellent general combining ability, but is sensitive to root lodging in Yugoslavian conditions. The F1 generation L588 x B92 was self-pollinated in 1992 to obtain an F2 population. In 1993, 500 S0 plants from F2 population S-5892 were self-pollinated (plants were randomly selected) and crossed to six plants of inbred line L1325 as a tester. Inbred line L1325 is a flint type, derived from Argentinean germplasm. FAO maturity group is 450. L1325 shows high heterotic effects with both L588 and B92 inbred lines, and is tolerant to root lodging.

A total of 500 entries (half-sib progenies) were evaluated within 25 sets (Cochran and Cox, 1957). Each set consisted of 20 half-sib progenies completely randomised within each of three replications. The entries were grown at Zemun Polje, Velika Plana, Indjija and Becej in 1994, and Zemun Polje, Velika Plana and Becej in 1995. A plot consisted of 9.20m long hand-planted rows with 0.70m between rows. Over-planted plots were thinned to a uniform plant density of approximately 62.112 plants ha^{-1}. All experiments were machine-cultivated and manually weeded as necessary for proper weed control. Data were collected at harvest for root and stalk lodging according to the following scale: 1-stalk broken bellow the tassel, 2-stalk broken above the ear, 3-stalk broken at the level of the ear, 4-stalk broken bellow the ear, 5-totally lodged stalk.

The analysis of data was based on plot means. Data were analysed by pooling over sets and combining across environments. From a basic population size of 500 half-sib progenies (25 sets with 20 half-sib progenies), 53,130 (25/5) populations with a size of 100 half-sib progenies, 3.268,760 (25/10) populations with a size of 200 half-sib progenies and 3.268,760 (25/15) populations with a size of 300 half-sib progenies were obtained by computer simulation. From the total number of combinations, 30 samples for each population sizes were randomly selected (except the 500 where only one sample is possible).

Comparisons of mean values between different population sizes were done by t or t′ test in relation to whether variances were homogenous or not (Steel and Torrie, Principles and procedures of statistics, Mc.-Graw-Hill Book Co., New York, 1960). Half-sib family means from each sample were used to construct the distribution histogram for each population. The Komogorov-Smirnov one-sample test was applied to test distribution. Values of D that are significant indicate non-normality of the distribution (Snedecor and Cochran, Statistical Methods, 8th ed., Iowa State University Press, Ames, 1989).

The analyses of individual populations pooled over sets and

combined across environments were calculated to partition the within population variation for each population size into environments, sets, environments x sets interaction, replications, genotypes, genotypes x environments and error sources of variation. Genotypes x environments interaction mean squares were used to test significance of the genotype source of variation. Error mean squares were used to test significance of the genotypes x environments interaction source of variation.

Estimates of genetic variance components were calculated by equating observed mean squares with expected mean squares and solving the resulting system of equations. Heritability was estimated on a half-sib progeny mean basis within each population size. Genetic variance component and heritability estimates were declared significant if their values were 2 times greater than their standard errors (Falconer, Introduction to quantitative genetics, Longman, London and New York, 1989). Additive, dominance, and epistatic variance components are confounded in the genetic variance estimates for half-sib families; hence, heritability estimates should be considered an upper limit of the narrow-sense heritability (Lamkey and Hallauer, Maydica, 32:64-78, 1999).

Average estimates ranged from 1.712 for 200 HS progenies population size to 1.730 for 100 HS progenies population size. The maximum (1.826) and minimum (1.631) sample average estimate was found in the population size of 100 HS progenies (Table 1). There is no significant difference for average estimates between estimated population sizes (Table 2).

Table 1. Mean values and standard errors for different population sizes.

Sample	Population size			
	100	200	300	500
1	1.757±0.027	1.724±0.026	1.738±0.028	1.717±0.028
2	1.778±0.031	1.719±0.029	1.676±0.028	
3	1.721±0.028	1.748±0.026	1.665±0.029	
4	1.728±0.029	1.656±0.027	1.714±0.028	
5	1.656±0.030	1.677±0.031	1.702±0.028	
6	1.853±0.029	1.681±0.028	1.782±0.029	
7	1.669±0.026	1.755±0.029	1.762±0.028	
8	1.768±0.028	1.725±0.028	1.732±0.027	
9	1.725±0.026	1.729±0.027	1.738±0.028	
10	1.766±0.030	1.660±0.028	1.729±0.028	
11	1.653±0.030	1.732±0.029	1.705±0.029	
12	1.751±0.025	1.723±0.027	1.701±0.028	
13	1.713±0.029	1.725±.0.029	1.755±0.028	
14	1.666±0.029	1.629±0.027	1.686±0.028	
15	1.660±0.027	1.686±0.027	1.710±0.027	
16	1.789±0.028	1.741±0.028	1.765±0.028	
17	1.750±0.031	1.741±0.028	1.733±0.029	
18	1.692±0.027	1.729±0.027	1.720±0.028	
19	1.728±0.027	1.756±0.028	1.692±0.028	
20	1.631±0.030	1.718±0.027	1.676±0.029	
21	1.774±0.026	1.690±0.030	1.717±0.028	
22	1.726±0.029	1.740±0.028	1.710±0.027	
23	1.762±0.027	1.697±0.028	1.694±0.028	
24	1.698±0.025	1.752±0.028	1.764±0.027	
25	1.798±0.031	1.676±0.028	1.728±0.028	
26	1.709±0.029	1.710±0.029	1.737±0.027	
27	1.826±0.029	1.673±0.026	1.689±0.029	
28	1.641±0.032	1.724±0.027	1.733±0.027	
29	1.738±0.028	1.709±0.028	1.762±0.029	
30	1.743±0.026	1.741±0.028	1.710±0.029	
Max	1.826	1.756	1.782	
Min	1.629	1.629	1.665	
Average	1.730	1.712	1.720	1.717

Table 2. Differences between mean values from estimated population sizes.

Population size	100	200	300	500
100	1.730	0.018^{ns}	0.010^{ns}	0.013^{ns}
200		1.712	0.008^{ns}	0.005^{ns}
300			1.720	0.003^{ns}
500				1.717

ns = statistically non-significant difference

According to the Komogorov-Smirnov test, a lower value of parameter D indicates a greater normality of distribution. The values of parameter D became greater with the decrease of population size (500 HS, D = 0.0469; 300 HS, D = 0.0529; 200 HS, D = 0.0543; 100 HS D = 0.0469), but there is no evidence of statistically significant deviation from normality in any sample (Table 3).

Genetic variability of estimated half-sib progenies was at a satisfactory level for all population sizes investigated. Estimates of genetic variances were statistically significant for all samples and population sizes and ranged from 0.192 for 100 HS progenies population size to 0.232 for 300 HS progenies population size (Tables 4, 5, 6 and 7).

Values for genetic x environment variance interaction were also statistically significant for all samples and population sizes and ranged from 0.257 for 200 HS progenies population size to 0.283 for 100 HS population size (Tables 4, 5, 6 and 7). Statistically significant estimates of heritability were found for all samples in all population sizes. Their values ranged from 0.585 (100 HS progenies population size) to 0.647 (500 HS progenies population size) (Tables 4, 5, 6 and 7).

Table 3. Values of parameter D from the Komogorov-Smirnov one sample test for different population sizes.

Sample	Population size			
	100	200	300	500
1	0.0522	0.0491	0.0443	0.0469
2	0.0734	0.0592	0.0503	
3	0.0637	0.0474	0.0665	
4	0.0937	0.0540	0.0537	
5	0.0568	0.0660	0.0543	
6	0.0841	0.0513	0.0490	
7	0.1004	0.0496	0.0640	
8	0.0615	0.0538	0.0432	
9	0.0691	0.0552	0.0534	
10	0.0551	0.0535	0.0365	
11	0.0794	0.0420	0.0533	
12	0.0628	0.0557	0.0634	
13	0.0720	0.0504	0.0604	
14	0.0807	0.0506	0.0503	
15	0.0543	0.0420	0.0504	
16	0.0650	0.0552	0.0479	
17	0.0555	0.0538	0.0513	
18	0.0592	0.0504	0.0522	
19	0.0613	0.0521	0.0633	
20	0.0976	0.0506	0.0527	
21	0.0670	0.0659	0.0505	
22	0.0514	0.0505	0.0512	
23	0.0589	0.0501	0.0438	
24	0.0830	0.0522	0.0613	
25	0.0517	0.0849	0.0513	
26	0.0823	0.0522	0.0532	
27	0.0590	0.0501	0.0579	
28	0.0514	0.0659	0.0589	
29	0.0727	0.0503	0.0491	
30	0.0630	0.0646	0.0494	
Average	0.0679	0.0543	0.0529	0.0469
Probability	0.95 0.99	0.95 0.99	0.95 0.99	0.95 0.99
Critical value	0.1223 0.1512	0.0860 0.1071	0.0706 0.8720	0.0547 0.0678

Table 4. Estimate of genetic variance, genetic x environment interaction variance, heritability, and their standard error for a population size of 100 half-sib progenies.

Population size – 100 half-sib progeny

Sample	σ^2_G	$SE\sigma^2_G$	σ^2_{GXE}	$SE\sigma^2_{GXE}$	h^2	SEh^2
1	0.167	0.042	0.223	0.025	0.583	0.146
2	0.191	0.052	0.320	0.029	0.541	0.146
3	0.213	0.049	0.243	0.026	0.627	0.145
4	0.196	0.049	0.343	0.027	0.586	0.146
5	0.159	0.046	0.320	0.028	0.511	0.146
6	0.221	0.052	0.327	0.027	0.616	0.145
7	0.218	0.048	0.210	0.025	0.652	0.145
8	0.165	0.044	0.313	0.027	0.550	0.146
9	0.196	0.045	0.207	0.024	0.638	0.145
10	0.189	0.050	0.303	0.029	0.549	0.146
11	0.234	0.055	0.323	0.028	0.616	0.145
12	0.202	0.045	0.237	0.024	0.651	0.145
13	0.211	0.051	0.293	0.028	0.597	0.145
14	0.232	0.055	0.377	0.027	0.618	0.145
15	0.187	0.045	0.127	0.026	0.602	0.145
16	0.078	0.031	0.323	0.026	0.368	0.148
17	0.235	0.057	0.400	0.029	0.599	0.145
18	0.148	0.039	0.173	0.026	0.549	0.146
19	0.248	0.053	0.200	0.026	0.671	0.145
20	0.194	0.045	0.290	0.028	0.625	0.145
21	0.236	0.055	0.187	0.025	0.627	0.145
22	0.177	0.044	0.377	0.027	0.585	0.146
23	0.140	0.036	0.230	0.026	0.574	0.146
24	0.184	0.050	0.187	0.024	0.542	0.146
25	0.174	0.045	0.370	0.029	0.559	0.146
26	0.154	0.043	0.347	0.027	0.527	0.146
27	0.202	0.055	0.287	0.027	0.540	0.146
28	0.196	0.047	0.423	0.030	0.600	0.145
29	0.197	0.045	0.297	0.026	0.633	0.145
30	0.239	0.056	0.233	0.025	0.620	0.145
Min	0.078	0.031	0.127	0.024	0.527	0.145
Max	0.247	0.057	0.423	0.030	0.651	0.146
Average	0.193	0.048	0.283	0.027	0.585	0.146

Table 5. Estimate of genetic variance, genetic x environment interaction variance, heritability, and their standard error for a population size of 200 half-sib progenies.

Population size – 200 half-sib progeny

Sample	σ^2_G	$SE\sigma^2_G$	σ^2_{GXE}	$SE\sigma^2_{GXE}$	h^2	SEh^2
1	0.196	0.032	0.147	0.025	0.627	0.103
2	0.229	0.038	0.307	0.027	0.626	0.103
3	0.216	0.034	0.240	0.025	0.653	0.103
4	0.222	0.036	0.247	0.026	0.644	0.103
5	0.235	0.041	0.333	0.029	0.597	0.103
6	0.205	0.034	0.243	0.026	0.613	0.103
7	0.251	0.040	0.240	0.027	0.649	0.103
8	0.220	0.036	0.290	0.026	0.623	0.103
9	0.256	0.038	0.200	0.025	0.684	0.103
10	0.221	0.036	0.253	0.027	0.626	0.103
11	0.235	0.038	0.290	0.027	0.630	0.103
12	0.248	0.038	0.293	0.026	0.666	0.103
13	0.196	0.035	0.347	0.027	0.577	0.104
14	0.263	0.040	0.217	0.026	0.678	0.103
15	0.252	0.039	0.203	0.026	0.672	0.103
16	0.232	0.038	0.253	0.027	0.636	0.103
17	0.257	0.040	0.247	0.027	0.662	0.103
18	0.211	0.034	0.237	0.025	0.637	0.103
19	0.196	0.034	0.293	0.027	0.591	0.103
20	0.181	0.031	0.227	0.025	0.602	0.103
21	0.240	0.040	0.293	0.028	0.618	0.103
22	0.242	0.039	0.317	0.027	0.642	0.103
23	0.253	0.040	0.263	0.027	0.653	0.103
24	0.217	0.036	0.297	0.026	0.621	0.103
25	0.274	0.042	0.273	0.026	0.675	0.103
26	0.257	0.040	0.267	0.027	0.653	0.103
27	0.228	0.035	0.177	0.025	0.662	0.103
28	0.267	0.040	0.207	0.025	0.690	0.102
29	0.245	0.039	0.270	0.027	0.645	0.103
30	0.247	0.039	0.267	0.026	0.656	0.103
Min	0.181	0.031	0.147	0.025	0.591	0.102
Max	0.274	0.042	0.347	0.029	0.690	0.103
Average	0.227	0.037	0.257	0.026	0.630	0.103

Table 6. Estimate of genetic variance, genetic x environment interaction variance, heritability, and their standard error for a population size of 300 half-sib progenies.

Population size – 300 half-sib progeny

Sample	σ^2_G	$SE\sigma^2_G$	σ^2_{GXE}	$SE\sigma^2_{GXE}$	h^2	SEh^2
1	0.204	0.028	0.267	0.026	0.609	0.084
2	0.156	0.024	0.287	0.026	0.562	0.085
3	0.210	0.030	0.273	0.027	0.599	0.085
4	0.267	0.034	0.250	0.027	0.666	0.084
5	0.246	0.032	0.273	0.026	0.655	0.084
6	0.240	0.032	0.343	0.027	0.627	0.084
7	0.224	0.030	0.323	0.026	0.632	0.084
8	0.252	0.032	0.220	0.026	0.668	0.084
9	0.248	0.032	0.290	0.026	0.654	0.084
10	0.204	0.028	0.290	0.026	0.608	0.084
11	0.269	0.034	0.270	0.027	0.658	0.084
12	0.261	0.033	0.210	0.026	0.674	0.084
13	0.217	0.029	0.227	0.026	0.632	0.084
14	0.260	0.033	0.263	0.027	0.659	0.084
15	0.208	0.028	0.243	0.026	0.618	0.084
16	0.216	0.029	0.267	0.026	0.635	0.084
17	0.207	0.029	0.267	0.027	0.607	0.085
18	0.251	0.033	0.267	0.027	0.644	0.084
19	0.243	0.032	0.257	0.027	0.647	0.084
20	0.232	0.031	0.323	0.026	0.636	0.084
21	0.268	0.034	0.323	0.027	0.656	0.084
22	0.232	0.031	0.273	0.026	0.637	0.084
23	0.232	0.030	0.247	0.026	0.653	0.084
24	0.254	0.033	0.287	0.026	0.657	0.084
25	0.227	0.029	0.227	0.026	0.650	0.084
26	0.195	0.028	0.287	0.026	0.595	0.085
27	0.202	0.028	0.230	0.026	0.619	0.084
28	0.259	0.034	0.277	0.027	0.650	0.084
29	0.204	0.028	0.230	0.026	0.624	0.084
30	0.282	0.035	0.280	0.027	0.668	0.084
Min	0.156	0.024	0.210	0.026	0.562	0.084
Max	0.281	0.035	0.343	0.027	0.674	0.084
Average	0.232	0.031	0.269	0.026	0.637	0.084

Table 7. Estimate of genetic variance, genetic x environment interaction variance, heritability, and their standard error for a population size of 500 half-sib progenies

Population size – 500 half-sib progeny

Sample	σ^2_G	$SE\sigma^2_G$	σ^2_{GXE}	$SE\sigma^2_{GXE}$	h^2	SEh^2
1	0.203	0.022	0.263	0.026	0.647	0.066

These results point to the possibility of working with a lower number of plants per F2 population for traits such as tolerance to root and stalk lodging.

In the material of the MRI gene bank, only imidazoline resistance was found among a group of total and selective broad-spectrum herbicides

--Vancetovic, J, Vidakovic, M, Stefanovic, L, Simic, M

In view of the increasing need for tolerant maize genotypes to broad-spectrum herbicides, especially because of the marketing of transgenic corn resistant to herbicides (Roundup ready, Liberty link) without reliable laws regulating GMOs in Europe, we felt a need to investigate our existing germplasm variability for mutants potentially resistant to the useful broad-spectrum herbicides. Use of mutants has a few advantages in comparison with GMOs; regulation is much clearer, no special isolations are needed, and there is no danger of them being forbidden for any reason.

We chose 11 herbicides that are potentially useful in maize for the investigation (Table 1).

We made 11 bulks to sample our existing genetic variability (MRI gene bank with 5437 active accessions). Each bulk consisted of 20 kernels of each heterogeneous accession (OP variety, race, synthetic) and 10 kernels of each inbred line. Each bulk was about 20 kg in weight.

Table 1. Applied herbicides and dosages on the material of the Gene Bank of MRI "Zemun Polje"-Belgrade.

Herbicide	Active ingredient	Manufacturer	Dosage applied
Arsenal	imazapyr (250g/l)	Cyanamid, New Jersey	750-1500g/ha a.T.
Agil	propaquizafop (100g/l)	Novartis, Basel	80-150g/ha a.T.
Gallant	haloksifop-etoksi-etil (125g/l)	Dow AgroSciences, Vienna	62.5-187.5g/ha a.T.
Focus ultra	cyklosidim (200g/l)	BASF, Ludwigshafen	100-400g/ha a.T.
Fusilade super	fluazifop-p-buthyl (125g/l)	Zeneca, Fernhurst	125-500g/ha a.T.
Furore super	fenoxaprop-p-ethyl (75g/l)	AgrEvo Hoechst Schering, Berlin	75-150g/ha a.T.
Leopard	quizalofop-p-ethyl (50g/l)	Agon Chemicals, Ashdad	25-200g/ha a.T.
Pantera	quizalofop-p-tefuryl (40g/l)	Uniroyal Chemical, USA	40-60g/ha a.T.
Pivot	imazethapyr (100g/l)	Cyanamid, New Jersey	80-200g/ha a.T.
Roundup	glyphosate-isopropyl ammonium salt (480g/l)	Monsanto, Brisel	960-5760g/ha a.T.
Select super	clethodim (120g/l)	Tomen Agro, San Francisco	96-240g/ha a.T.

In the spring of 2001 these bulks were sown very densely (plant to plant) during an optimal sowing period, in an area of no more than 2 ares each. The first spraying with the chosen herbicides was done when the plants were at the stage of 3-4 leaves. Spraying with each herbicide was subsequently repeated 1 or 2 times because of the late emerging plants (the escapes).

Only for herbicide 9 (Pivot) were resistant plants actually found. This herbicide also had the longest period of activity (10-15 days). About 30 plants showed full resistance to Pivot, of which 6 were outcrossed to the nonresistant testers. About 40% of the resistant plants showed male sterility. Seeds from fertile resistant plants and sterile resistant plants (from the open-pollination) were collected separately.

In 2002, testcrosses of the 6 resistant plants with nonresistant testers were sown, as well as the open-pollinated seed from fertile and sterile resistant plants. Treatment was done with the Pivot herbicide, at the same dosage as the previous year. It seems that the gene(s) involved are dominant, since all treated material was resistant. Among the plants from the open-pollination of resistant sterile plants, only 1 exhibited male sterility (from a total of 60 plants in 2002), meaning that there is no correlation between Pivot resistance and some type of male sterility, as it may have seemed in the previous year. Nevertheless, sibbing was done on that 1 sterile plant, as well as selfing of testcrosses with nonresistant testers. This will allow, in 2004 (the material was planted in 2003, but a strong storm destroyed it all), investigation of the mode of inheritance of this resistance.

A high incidence of the sources of cytoplasmic male sterility (cms) in the Maize Research Institute (MRI) gene bank

--Vancetovic, J, Vidakovic, M, Vidakovic, M, Rosulj, M

Finding a restorer cytoplasm, which exists only theoretically, as proposed by Hermsen (Euphytica 14:221-224, 1965; Euphytica Supp. 1:63-67, 1968), would allow easier and genetically cleaner hybrid seed production based on male sterility in a plant species than any other system imposed so far. Only a limited amount of research has been done in this area, and all of the results were negative (Kohel and Richmond, Crop Sci. 3:361-362, 1963, on cotton; Rutger and Jensen, Euphytica 16:350-353, 1967, on barley; and Washnok, MNL46:25-27, 1972, on maize). This encouraged us to start a huge experiment, and search all of our gene bank for the presence of the restorer cytoplasm for the gene *ms10*. Unfortunately, we have not found it. So the objective of the original study was quite different from the results reported herein, not to find new sources of cms, but to find the restoring cytoplasm for the gene *ms10* in maize.

In a search for the restorer cytoplasm (Vidakovic et al., J. Hered. 93:444-447, 2002) for the gene *ms10*, each accession from the MRI gene bank was crossed with a heterozygous *Ms10/ms10* tester. The plants from the two crossed ears of each heterogeneous accession (OP varieties, races, synthetics), and from one ear of the crossed lines, were subsequently backcrossed with the *Ms10/ms10* tester. Apart from the *Ms10/*ms10 genetic constitution, the heterogeneous tester consisted of various genotypes from different maturity groups, in order to cover almost all of the vegetation span of the gene bank accessions. Crossing and backcrossing was done in isolation, with detasselled gene bank accessions used as females. Twenty plants of the first cross per each ear were used for backcrossing within each accession. Approximately the same number of kernels from the backcrossed ears were mixed within an accession, and subsequently planted in a population of about 60 plants for testing for the presence of the restoring cytoplasm for the *ms10* gene. Indication of the presence of the restoring cytoplasm would be a 100% fertile test, and average expected frequency of the sterile *ms10/ms10* plants was 1/8 for each accession.

Restorer cytoplasm wasn't found, but a high incidence of the sources of cytoplasmic male sterility was obvious within our gene bank. Since plants within each test were descendants from a single ear, they all shared the same cytoplasm. As an indication that an accession contains a sterile cytoplasm we took the presence of more than 30% sterile plants in the test, which is significantly higher than the theoretical 1/8 which would arise from *ms10/ms10* plants only. In Table 1 there is a list of accessions where sterile cytoplasm was indicated.

In the year 2000, spare seed from the crosses and/or backcrosses of these accessions was used for multiplication by open-pollination (cytoplasm is only transmitted through female plants). Genotypes with more than 40-50% sterile plants in the phase of multiplication (from the original seed lots) were chosen for testing of the type of cytoplasm involved (+- in Table 1).

Some more sources of sterile cytoplasms were indicated from an unexpected source – from so-called test-candidates for the restoring cytoplasm, which were 100% fertile in the test. Within those tests, selfing of individual plants and outcrossing to the *ms10/ms10* tester was performed. A 100% fertile self plus 100% sterile or 1:1 (Ft:St) segregating test would indicate the presence of the restorer cytoplasm. Such pairs of selfs-tests we did not find, but in some of the selfed progenies an excess of sterile plants was observed, far greater than 0.25, which would be expected if the original plant was heterozygous for the *ms10* gene. These progenies were also included in the multiplication field in 2000, and few were (Table 2) chosen for the test of the type of cytoplasmic sterility (based, again, on more than 40-50% sterile plants in the phase of multiplication).

Table 1. Accessions with more than 30% sterile plants in an *ms10* restorer cytoplasm test, indicating the presence of the sterile cytoplasm.

Kol-Int(-l)†	Acc.no	Name of accession	Provenience	LB‡	+§	Remark
Kol	36	Domaci bijeli	Orahovo	LB	+	
Kol	146	Zuti tvrdunac	Sipovo		+	
Kol	239	Mnogoredi zuti zuban			+	¶
Kol	420	Zuti tvrdunac	Kabas	LB		
Kol	536	Zlatni zuban	Prislonica		+	¶
Kol	1041	Zuti poluzuban	Prosenikovo	LB		
Kol	1084	10-redi	Zeta	LB		
Kol	1232	Saradan rani	Boljevic	LB		¶
Kol	1299	Srednje seme	Bjelo Polje	LB		
Kol	1326	Klek	Prigradna		+	
Kol	1385	Domaci	Goles		+	
Kol	1393	Domaci	Spajici	LB		
Kol	1415	Domaci bijeli	Bogomilovici			
Kol	1416	Domaci bijeli	Pjesivci	LB		
Kol	1436	Domaci	Sinj	LB		
Kol	1575	Bosanac	Zenica	LB		
Kol	1609	Beli	Kalna	LB		
Kol	1729	Poljski osmak	Visegrad	LB		
Kol	1922	Domaci slatki	Sinj			
Kol	1944	Domaci tvrdunac	Sipovo		+	
Kol	1947	Cado	Sipovo		+	
Kol	2170	Domaca bakrena trdinka	Radovljica	LB		
INT	2975	Mestnaja kavkazkaja zeltaja	Kavkaz	LB		
INT	3497	Nebraska long ear Xchalq-composite	Mexico		+	#
INT	3506	Amarillo bajio	Mexico		+	
INT	3635	Korom abad 3	Iran			#
INT	3727	Cuarentin 1938x45	Argentina		+	††
INT	3730	Cuarentin 1939x35	Argentina		+	††
INT	3732	Cuarentin 1938x33	Argentina			††
INT	3734	Cuarentin 1932x45	Argentina		+	††
INT	3735	Cuarentin 1933x39	Argentina		+	††
INT	3938	XIX/44 Synthetic	USA		+	
INT	4581	Jordi	Jordan		+	
INT	4961	Hasuri	Gruzia		+	¶
INT	4965	Ahmeta S. Birkiani	Gruzia			
INT	5012	Kremnistaja belaja	Gruzia			
INT	5267	PD 1109 population	DDR			
INT	5283	PD 1156 population	DDR		+	¶
INT	5307	PD 1302 population	DDR		+	¶
INT	5313	PD 1416 population	DDR		+	
INT	5399	Brzovec	Bulgaria	LB		
INT	6100	Voronjezskij M 52 synth.	Former SSSR		+	
INT	6651	Pool 42 (NTR-2)	Mexico			
INT	7106	MG 91 862 population	Bari g.bank-Italy		+	#
INT	7154	MG 91 912 population	Bari g.bank-Italy	LB		
INT	7224	MG 91 774 population	Bari g.bank-Italy	LB		
INT-L	1472	R-563	Bari g.bank-Italy	LB		
INT-L	4884	GR.38868	Greece		+	
INT-L	5771	WCB-27	Greece		+	
INT-L	5856	Rt 1	Tchecoslovachia		+	
INT-L	5857	CE-178Rf	Tchecoslovachia		+	
INT-L	5860	RT 2	Tchecoslovachia	LB	+	
INT-L	6136	Rt 11	Tchecoslovachia	LB	+	
INT-L	6137	Rt 25	Tchecoslovachia	LB	+	
INT-L	6276	SR 10 (flint)	Poland		+	

† Kol=Collection of OP varieties from the territory of the former Yugoslavia
INT=Heterozygous foreign material
INT-L=Foreign and domestic inbred lines
‡ LB=Sterile plants in the test (some or all) exhibited the late break of sterility
§ +=Chosen for the sterility type classification
¶ = 1 of 2 progenies gave 100% St test
# = The test was almost 100% sterile
††= Originally received as segregating for St plants

In the multiplication field, progenies were also included from the ears of the sterile plants found in the accessions of the Yugoslav Collection, taken from the open-pollination. Again, for further classification, only progenies with more than 40% sterile plants were chosen, indicating the presence of the sterile cytoplasm, and not solely some of the *ms* genes. These accessions are listed in Table 3.

Kol 146 will be included twice in the classification test (Table 1 and 3), chosen by the 2 criteria (descendants are from the 2 different ears from this variety).

Table 2. Selfed candidates for the restoring cytoplasm chosen by the excess of sterile plants in the selfed progenies (significantly greater than 0.25).

Kol-Int(-l) †	Acc.no	Name of accession	Provenience	+‡
Kol	237	Zuti zuban from sister crosses	Zemun Polje	+
Kol	393	Belo staro seme	Rznic	+
Kol	547	Krupni staklarac	Vica	+
Kol	842	Crveni kukuruz	Jastrebarsko	+
Kol	1387	Domaci bosanski	Bielo Bucje	+
Kol	2154	Doma i zuti	Prilep	+
INT	5019	Ambrolauri S. Nikorcminda popul.	Gruzia	+
INT	7106	MG 91 862 population	Bari g.bank-Italy	+
INT-L	5767	W 182B	Greece	+
INT-L	5981	SV 59	Tchecoslovachia	+

† Kol=Collection of OP varieties from the territory of the former Yugoslavia
INT=Heterozygous foreign material
INT-L=Foreign and domestic inbred lines
‡ +=Chosen for the sterility type classification

Table 3. Progenies chosen for sterility classification from the sterile plants from the open-pollination of the accessions of the Yugoslav Collection, based on an excess of more than 40-50% sterile plants during multiplication.

Kol	Acc.no	Name of accession	Provenience	+†
Kol	146	Zuti tvrdunac	Sipovo	+
Kol	326	Zuti tvrdunac	Novi Sad	+
Kol	1127	RB 10 Var. vulgata	Bitolsko	+
Kol	1172	RB 55 Var. vulgata	Probistip	+
Kol	1258	Domaci beli	Crmnica	+
Kol	1385	Domaci beli	Goles	+
Kol	1613	Beli	Svrljig	+
Kol	1882	Domaci D 1597	Gracac	+
Kol	2100	Bosanski zutac	Staro Sipovo	+

† +=Chosen for the sterility type classification

The high incidence of sterile cytoplasms in our gene bank is somewhat surprising. We have chosen 50 sources for a test of the type of cytoplasmic male sterility, but it seems that the number of the cms sources is at least twice that high, giving a number of about 100 independent sources of cms. In comparison with the total active number (about 5000) of accessions in our gene bank (accessions that are actually present), this gives a total of 2%. It raises a question of whether there may be any evolutionary significance of a high number of, mostly, populations with cytoplasmic male sterility, possibly in making some self-compatibility barrier protecting such a population from foreign pollen not carrying the appropriate restorer (Rf) gene.

Sterile plants from the accessions chosen are crossed for further classification in 2002 with 3 heterozygous testers: *RfT/rfT*, *RfC/rfC* and *RfS/rfS*. Field classification for these 3 types of cms will be done in 2004, since our breeding nursery in 2003 was destroyed by a storm.

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Screening maize genotypes for resistance to *Fusarium verticillioides* comparing in vivo plant response and in vitro bioassays

--Balconi, C, Lupotto, E, Lanzanova, C, Conti, E, Gualdi, L, Mazzoleni, A, Valoti, P, Motto, M

The availability of reliable methods for the screening and evaluation of maize genotypes for improving tolerance to

Fusarium attacks is an invaluable tool in breeding programmes to increase crop protection against fungal infection. Numerous *Fusarium* species are widespread pathogens in cereals. They infect small grain cereals (wheats, barley, oats, rice, triticale, sorghum, millet) and maize (mostly *F. verticillioides*), causing root, stem, and ear rot diseases in both temperate and semi-tropical areas of the world. Some *Fusarium* strains also produce mycotoxins that can be formed in infected plants before harvesting, or in grains during post-harvest storage. The occurrence of mycotoxins in cereal grains is a great concern worldwide, because their presence in feed and foods is often associated with chronic or acute mycotoxicoses in livestock and also in humans. In this respect, the development of improved maize genotypes with increased resistance to *F. verticillioides* using breeding and biotechnology strategies is important. The aim of our research is to develop a rapid and reliable screening method to evaluate maize genotypes for resistance to the infection of this pathogen.

For this purpose, six inbred lines released by the Maize Section of Bergamo (Lo904, Lo1010, Lo1096, Lo1067, Lo1095, Lo1124) (Bertolini et al., Maydica 45:73-87, 2000) were used as the experimental material. The inbreds were tested in field experiments with Silk Channel Inoculation Assay (SCIA) on adult plants, and by in vitro bioassays with Kernel Inoculation Assay (KIA) to follow the progression of *F. verticillioides* infection in inoculated maize germinating seeds. The results of the in vivo and in vitro experiments were compared i) to evaluate if the in vitro bioassays reflect in vivo plant response to *Fusarium* attack, and ii) to offer a rapid screening test for genotype evaluation.

For both SCIA and KIA protocols *F. verticillioides* was grown on PDA plates at 26 C until the mycelium covered the surface of the plate, and used for fresh spore inoculum production. For the field experiments, plants were hand-pollinated and SCIA applied at two different stages of kernel development at 3 and 6 days after pollination (DAP); controls were non-inoculated and sterile water-inoculated plants. According to previous information (see Reid, Hamilton, and Mather, Technical Bulletin 1996-5E, research branch, Agriculture and Agri-Food Canada, 1996, method for *F. graminearum*), amounts of 2 ml, containing two spore concentrations of 10^5 and $5x10^5$/ml, were employed for the SCIA tests. Inoculation was performed by spore injections into the silk channel of each primary ear. Ears were manually harvested, hand de-husked; the severity of ear rot symptoms was evaluated using rating scales based on the percentage of kernels with visible symptoms of infection, such as rot and mycelium growth. As reported by Reid et al. (1996), the visual rating scale consists of 7 classes based on percentage of visibly infected kernels (Disease Severity Rating-DSR-: 1=0%-no infection; 2=1-3%; 3=4-10%; 4=11–25%; 5=26-50%, 6=51-75%; 7=76-100%). Individual ear ratings using a visual scale, as described above, allowed a discernible screening of the six genotypes tested for *Fusarium* resistance. The kernel developmental stage at inoculation was discovered to be an important technical parameter: for all the genotypes tested, inoculations gave the best results when applied at 3 DAP. At this stage silks are elongated and green. Disease severity scores were lower for ears of the same genotypes infected at 6 DAP: at this latter stage silks appear wilted and began silk senescence, a physiological change apparently altering the suitability of silk for permitting the growth of ear-rotting organisms. The spore concentration of 10^5/ml, applied for inoculation at 3DAP, gave maximum differentiation among genotypes, ranging from the most resistant Lo904 (DSR: 2.58 ± 0.49), to the most susceptible Lo1124 (DSR: 6.42 ± 0.66). Together with Lo904, Lo1010 and Lo1096 appeared to be more resistant (DSR-range: 2.58-3.89) than Lo1124, Lo1067 and Lo1095 (DSR-range:5.7-6.42). For all genotypes, the non-inoculated and sterile water-inoculated plants, as controls, showed no infection, with disease severity ratings around 1.

To obtain the optimal conditions for the in vitro KIA bioassays, sterile kernels were inoculated with a conidial suspension of the fungus by adding 125, 1250 or 12500 spores/seed. Inoculated and control kernels (sterile water inoculated) were allowed to continue germination for 7 days. For each genotype, records of the percent of maize kernels with visible fungus colonization and an "infection score", were registered. The "infection score" rating scale consists of 6 classes related to kernel surface infection extent (Infection Score-IS-: 0=0% infected seed surface; 1=5-10%; 2=10-30%; 3=30-50%; 4=50-75%, 5=75-100%; 6=100% heavily infected). The progression of the infection on the developing vegetative tissues (radicle, coleoptile) was also recorded. Results indicated that an inoculum of 125 spores/seed was optimal to discriminate genotype response by the in vitro KIA bioassay. All Lo inbred lines tested showed a high percentage of seed colonized, but variability was observed for the extent of infection during the 7 days after inoculation. Lo904 and Lo1096 were resistant for all periods tested (IS-range: 1.14-1.75). On the other hand, Lo1095 and Lo1067 appeared significantly more susceptible 3 days after inoculation (IS-range: 2.33-4.58), and showed a very heavy contamination 7 days after inoculation (IS-range: 4.76-5.88). Interestingly, for these four genotypes, the indications obtained by in vitro KIA bioassays reflect field observations of resistance/sensitivity to *F.verticillioides.* Lo1010 and Lo1124, under the bioassay conditions previously described, showed response to *Fusarium* attack with scores intermediate between the most resistant and the most susceptible genotypes. Experiments are in progress to evaluate if higher or lower spore concentrations applied to KIA may improve the degree of differentiation among genotype response.

In conclusion, the results suggest that in vitro bioassays on mature kernels appear to be, after additional testing and standardization, a useful tool for screening and selfing maize plants with a superior level of protection against *Fusarium verticillioides.*

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Cloning and characterization of the *glossy1* gene of maize

--Sturaro, M, Hartings, H, Motto, M

The *glossy-1* (*gl1*) locus of maize is required for the formation of the epicuticular wax layer of young plants. *gl1* mutant seedlings can be visually identified because of their glossy leaf surface which is different from the dull surface of wild-type seedlings. The *gl1* locus was isolated by transposon tagging. Seven unstable mutations, *gl1-m1* to *gl2-m9* were induced in a parental strain carrying an active transposable *Activator* (*Ac*) element in the unstable *wx-m7* allele. Genetic tests on the *gl1-m5* allele indicated that it was not caused by the *Ac* element but by the insertion of the transposable element *Enhancer/Suppressor-Mutator* (*En/Spm*). A *Hin*dIII restriction fragment segregating with

the mutant phenotype was identified, by Southern analysis, using sequences from the *En/Spm* element as a probe. Part of the fragment was cloned and was shown to carry part of the unstable *gl2-m5* allele. These *gl1* sequences were used to identify a genomic fragment carrying the wild-type allele and to isolate its corresponding cDNA sequence.

Computer-aided analysis of the genomic sequence permitted us to identify the putative exons encompassing the *gl1* transcript. A database search for proteins homologous to the deduced *gl1* polypeptide bolstered the postulated mRNA sequence. On the basis of these data two primers were designed to isolate the full coding sequence of *gl1* by RT-PCR. A single 2,056 bp fragment including a 1,866 nucleotide long ORF was amplified from RNA extracted from wild-type seedling leaves. An in-frame stop codon is present 87 bp upstream of the ATG start codon of the main ORF. No alternative translation start sites can be found, indicating that the amplified fragment includes the complete coding region. Putative CAAT- and TATA-box motifs can be found in the promoter sequence 200 bp and 146 bp upstream of the ATG start codon, respectively, while a putative polyadenylation site is present 312 bp downstream of the translation stop codon.

Translation of the 1,866 nucleotide long open reading frame present in the *gl1* cDNA sequence gives rise to a putative polypeptide of 621 amino acids with an apparent molecular weight of 69.6 kDa and pI of 9.89. Hydropathy analysis predicts the presence of several transmembrane domains in the N-terminal region of the Gl1 polypeptide, as well as a hydrophilic C-terminal domain. Furthermore, a histidine-rich motif characteristic of a family of membrane-bound desaturases/hydroxylases is present in the N-terminal part.

A database search for proteins homologous to Gl1 with the TBLASTX algorithm revealed several sequences exhibiting high levels of similarity with the query sequence used. In particular, a putative polypeptide of 619 amino acids encoded by a cDNA from *O. sativa* (AK060786) showed 84% identity over its entire coding sequence. Furthermore, significant homologies, with a 67% identity score, were found with the products of two other rice cDNAs (AK066569 and AK070469), with the *WAX2* locus of *Arabidopsis thaliana* encoding a protein involved in cuticle synthesis (62% identity), and a partial polypeptide (L33792) derived from *Senecio odora* (55% identity). From this analysis it was argued that the highest degree of homology consistently regards the C-terminal part of the deduced proteins.

A comparison of the deduced Gl1 protein sequence and the product of the *A. thaliana CER1* locus, a putative aldehyde decarbonylase active in the epicuticular wax biosynthesis pathway, reveals an overall identity of 35%. This similarity score is significantly lower than the degree of similarity encountered between the putative Gl1 and *A. thaliana* WAX2 proteins (62%). Since previous results had attributed to the *gl1* locus a role as a *CER1*-orthologue from maize (Hansen et al., Plant Physiol. 113:1091-1100, 1997), we further investigated amino acid sequence similarities among a restricted group of Gl1-homologous sequences by means of phylogenetic analysis. These analyses suggest the presence of two groups of protein sequences, the former containing the CER1 protein as a founder sequence, the latter including the WAX2 sequence. Interestingly, the Gl1 sequence shows a high level of homology with the members of the WAX2 group, while a second maize sequence (GenBank AY104752) is located within the CER1 group, with which it shares 55% amino acid identity. Hence, phylogenetic analysis indicates that the *gl1*-related sequences can be divided into two subgroups, each comprising genes from at least three species: maize, rice, and *Arabidopsis*.

In conclusion, the genomic and cDNA sequences we have isolated differ from the putative *gl1* gene and transcript previously identified (Hansen et al., 1997). The protein encoded by *gl1* shows significant homology with the entire sequence of the *WAX2* gene product of *Arabidopsis*, involved in both cutin synthesis and cuticular wax production.

CHISINAU, MOLDOVA
Institute of Genetics

Effect of low positive temperature on callusogenesis and somatic embryogenesis of maize

--Climenco (Kravchenko), OA, Jacota, AG

The purpose of this study was to determine sensitivity of maize to cold during callus and somatic embryo formation. Fourteen inbred lines (MK01, W47, Rf7, XL12, 092, A239, P101, Co125, MK159, MK390, 459, F2, Mo17, B73) were used as experimental material. Tissue cultures were initiated from 12-day-old embryos (total number = 1951). Immature embryos and callus cultures were treated by low positive temperature (+10 C+12 C) for 24, 48 and 72 hours. The data were processed by two-factor analysis of variance.

The results indicate that genotype, temperature and genotype x temperature interaction effects are highly ($P<0.001$) significant for callusogenesis and somatic embryogenesis. It was found that in most of the cases, exposure to low temperature results in a decrease in frequency of callus and somatic embryo formation, with the exception of 459, A239 and XL12 inbred lines which had higher values of these characters in comparison with the control variants. It should be noted that somatic embryogenesis frequency of the inbreds 459, MK159, 092, P101, MK01, F2, Rf7, MK390 proved to be less than 20%. The data also indicate that the other inbreds (Co125, A239, XL12, W47, Mo17, B73) were more resistant to low positive temperature (the frequency of somatic embryogenesis was higher than 30%). The other result was that for each of the 14 inbreds the best combinations of factors studied were also determined.

Male maize gametophytes as a possible test system for radioecological monitoring

--Kravchenko, AN

In this study, male maize gametophytes taken from several zones of Moldova with different levels of radioactive pollution were analyzed. By means of morphocytological analysis three groups of characters were determined. These characters' variability showed the degree of pollen sensitivity to different levels of radioecological pollution. The first group includes morphological characters of pollen grain such as perimeter, area, the smallest and the biggest diameter, and eccentricity. The second group includes morphocytochemical characters of the vegetative cell nucleus (size, the DNA quantity and chromatin dispersion). The third group includes the same characters but for the generative cell nucleus. It appears from this study that the first group of characters has the highest degree of variability and the

third group the lowest one. Generally, results indicate that the male gametophyte of maize can be used in the elaboration of a test system for radioecological monitoring.

Effect of radioactive pollution of soil on maize chromosomes

--Kravchenko, AN, Saltanovici, TI, Rojnevschi, MK

The aim of this study was to determine the effect of radioactive pollution on ontogenesis of maize F_1 hybrid M291MB. This hybrid was cultivated in six zones of Moldova, which differ in the level of radioactive pollution. The results indicate that variability of chromosome aberration frequency was determined by place of cultivation. The analysis of chromosome aberration frequency and spectrum showed such abnormalities as chromosome lagging, chromosome and chromatid bridges and fragments. We also observed the appearance of cells with two nuclei and nuclei with two or more nucleoli. This is possibly due to an increase of the RNA synthesis level, and appears to be connected with the activation of reparation systems. Thus, chronic irradiation of plants causes chromosome aberrations and results in gene pool degradation of maize.

A new aspect of the shape effect of maize and chickpea plants

--Maslobrod, S, Ganea, A, Grati, M, Corlateanu, L, Romanova, I

Under the shape effect we understand the change of structure and function of the object of inanimate or animate nature under the influence of factors induced by geometrical figures such as cylinders, cones, pyramids, cupolas, etc. (Akimov, A.E., 1995; Narimanov, A.A., 2001). According to modern views, the torsion field of left (***l***) or right (***d***) rotation generated by these figures is laid on the basis of the shape effect (Akimov A.E., 1995). This is an informational field, it cannot be completely reduced to known physical fields (electromagnetic and gravitational), it has a high penetrating capacity and spreads with speed, exceeding then the velocity of light (Akimov, A.E., 1995).

For the first time, we have shown that under the influence of the torsion fields of these figures on the seeds of different plant species, the architectonics (structural leftness and rightness) of the seedlings change in absolute accordance with the vector (leftness and rightness) of rotation of the torsion field (Maslobrod, S.N., 2001). It is well known that ***l*** and ***d*** forms of plants differ according to the adaptive potential – ecological resistance and productivity (Maslobrod, S.N. et al., 2002; Sulima, U.G., 1970).

Taking into account the wide presentation of different geometrical figures of abiotic and biotic origin in nature, and the different morphogenetic influences of their torsion fields on plant objects, we have advanced the supposition about the shape effect as a factor of ecology and plant growth (Maslobrod, S.N., 2001). Developing this idea, we have made an attempt to check the presence of the shape effect on plants, not only on the level of organisms, but also on the level of chromosomes.

For this we have used the criterion of the number of chromosome aberrations (NCA) in primary rootlets of seedlings (Pausheva Z. P., 1974). The materials for the tests were: the maize hybrids MK01i x 2.9M and M215, and the chickpea cultivar Krasnokutskii 195. The control was the NCA in the rootlets of seedlings from normal seeds and seeds which were exposed to presowing gamma-irradiation with doses of 250 Gy (MK01i x 2.9M and Krasnokutskii 195) and 500 Gy (M215). The seeds were germinated in growing-boxes, filled with black soil (chernozem). During the whole period of seed germination (up to 72 hours), short cylinders (SC) and long cylinders (LC) were placed above the growing-boxes. SC and LC have diameters less and more than half of the cylinders length (Akimov, A.E., 1995), and they generate, by our data, ***l*** and ***d*** torsion fields, accordingly (Maslobrod, S.N., 2001).

The main results of the action of torsion fields of SC and LC (Table 1) were:

Normal seeds. There was an essential increase of NCA in chickpea and maize in comparison with the control. There were no differences between the SC and LC variants. A tendency to exceed the LC variant was observed..

Gamma-irradiated seeds. The reduction of NCA compared to the control (gamma-irradiated seeds without the influence of the torsion fields) was obtained with the use of SC (the differences are significant for chickpea and maize M215), and the increase of NCA was obtained with the use of LC (the differences are significant for maize M215). There were observed differences between the SC and LC variants (significant for all objects).

Table 1. The number of chromosome aberrations (NCA) in maize seeds (hybrids MK01i x 2.9M and M215) and chickpea seeds (cultivar Krasnokutskii 195) under the influence of the torsion fields of short (SC) and long (LC) cylinders.

Variant	Object	n	NCA,%	Student criterion	n	NCA,%	Student criterion
		Normal seeds			Gamma-irradiated seeds		
Control	MK01i x	326	1.8±0.5	$t_{1.2}$=5.12	856	20.6±1.3	$t_{2.3}$=1.96
SC	2.9M	345	10.4±1.6	$t_{1.3}$=5.80	488	18.0±1.7	$t_{2.3}$=2.70
LC		445	10.0±1.4		402	25.3±2.1	
Control	Krasnokutskii	940	1.1±0.3	$t_{1.2}$=4.18	462	11.7±1.5	$t_{1.2}$=2.73
SC	195	287	4.5 ±0.8	$t_{1.3}$=4.52	320	6.2±1.3	$t_{2.3}$=3.79
LC		405	6.7±1.2		385	14.3±1.7	
Control	M215	460	2.2±0.4		362	28.2±2.3	$t_{1.2}$=4.36
SC					477	16.0±1.6	$t_{1.3}$=2.43
LC					244	35.3±1.8	$t_{2.3}$=8.04

Note: n – number of cells analyzed in ana-telophase; t teor. by P_{05} = 1.96

Thus, on the examples of representatives of cereals and legumes, a previously unknown shape effect on plants was revealed at the chromosomal level. The NCA in primary rootlets of seedlings from normal seeds is sharply increased, and in seedlings from gamma-irradiated seeds this number is either reduced (with the influence of the left torsion field of SC on seeds) or increased (with the influence of the right torsion field of LC). Evidently the dose of presowing gamma-irradiation of seeds also influences this effect (the differences were more contrasting on maize with a dose of 500 Gy, than with 250 Gy). A modifying influence of genotype is not expected.

In light of the data obtained our point of view about the shape effect as a factor of ecology and plant growth has received further confirmation. The data obtained presume that torsion fields of geometrical figures (cylinders, in particular) can evidently be considered as a new, non-traditional radio protective and mutagenic factor of natural and artificial origin.

"Memory" of the system of two swelling seeds of maize and distant transmission of structural bioisomerism from one seedling to the other, determined by this "memory" under stress conditions

--Maslobrod, S, Ganea, A, Corlateanu, L

We have shown (Maslobrod, S.N. et al., 1992), that the mirror symmetrical seedlings (MSS) – left (***l***) seedling and right (***d***) seedling (***ld*** and ***dl*** pairs) - mainly grow from a pair of seeds with adjoining germs. The effect of mirror symmetrization (EMS) is inherent in different plant species, and depends on genotype and the physiological state of the seed pair. It reflects the power of interaction between the components of the elementary phytocenological cell and can be linked with genotype competitiveness and productivity (Maslobrod, S.N. et al., 2002). Our task was to ascertain: 1. Whether the EMS is kept when the seeds from the pair are germinated separately after their joint swelling during some period of time and thus to determine the exposition at which the memory of the system (microphytocenosis) of seeds begins to develop according to the criterion of the EMS. 2. Whether the structural-functional state of the component of the pair changes under the stress influence on the other component of the pair, which is disconnected in space, i.e. if there is a nonlocal connection between them.

Pairs of maize seeds were formed (hybrids M450, M215, Debut). They were germinated in vegetative vessels with soil. The number of every variant repetition (number of pairs) was from 100 to 500. Pairs were germinated under normal temperature (1), and after 24 hours of swelling, one seed was taken from these pairs of seeds and transplanted into another vessel. Next, all the seeds were germinated under normal temperature (2). The seed that was taken out was exposed to low temperature stress (+3 C for 5 hours), then all seeds were germinated at normal temperature (3). The percentage correlation of the sum of the pairs (PSP) with the different and the same signs of bioisomerism was determined (***ld***+***dl***) : (***ll***+***dd***). Determination of the seedling pair bioisomerism was made according to the initial (before separation) seeds of every pair. ***L*** and ***d*** seedlings of maize have the first real leaf turned up counter-clockwise and clockwise accordingly (Sulima, U.G., 1972).

It was revealed that in normal conditions (1) the EMS was displayed clearly (for M450, M215 and Debut, the PSP was 54.5 : 45.5; 56.6 : 43.4; 61.2 : 38.8, respectively). In the second variant, the EMS remained at the normal level (for the same hybrids the PSP was 54.4 : 45.6; 59.5 : 40.5; 64.0 : 36.0). Under low temperature stress the PSP has moved considerably to ll and dd pairs (38.5 : 61.5; 43.6 : 56.4; 43.4 : 56.6).

It can be assumed that seeds joined in pairs for 24 hours provide the formation of programme "memory" in seeds as the system of future bioisomerism of seedlings, and the stress promotes the "imposing" of this "memory" of the experienced seed (inductor) upon the normal seed (receiver). In connection with this, an experiment was conducted with the seeds of Debut hybrid, in which the seeds taken out from each pair were exposed to additional stresses besides low temperature stress: high temperature stress (+45 C for 5 hours), chemical stress (1M NaCl for 5 hours), radiation stress (gamma-irradiation with a dose of 250 Gy) and to the influence of torsional fields of geometrical figures (short cylinders, SC, and long cylinders, LC), taking into account the ability of these fields to induce chromosome aberrations in the rootlets of seedlings. The size of SC and LC and the results of the experiment are reported in the preceding article.

The results obtained in the first experiment were confirmed: with all types of stress influence on seeds taken from each pair, the EMS, i.e. the number of MSS, is reduced (Fig. 1). There is a leveling of PSP in the SC variant. To some extent this conforms with our data about the less pronounced ability of the SC field to induce a bioeffect compared to the LC field (see the aforementioned article).

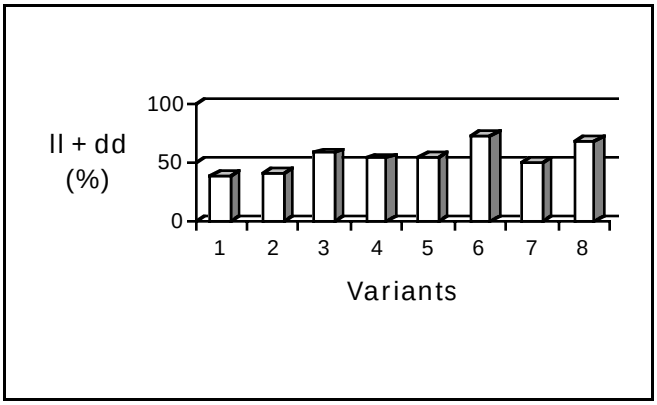

Figure 1. Relation of number of seedling pairs with the same sign of bioisomerism (***ll*** + ***dd***) to the common number of pairs (***ll*** +***dd*** +***ld***+***dl***). 1 - Control – a pair without seed separation. 2 - Norm - a pair with seed separation without stress. 3, 4, 5, 6, 7, 8 - +3 C, +45 C, NaCl, 250 Gy, SC, LC – stress influence on one seed separated from the pair.

The use of the Debut seeds later aided in the study of some aspects of the results observed:

1. If both seeds from the pair are exposed to the stress (for example, to the low temperature or gamma-irradiation), the EMS doesn't disappear (PSP is 58.7:41.3; 61.3:38.7, accordingly). These data confirm the fact of transmission of bioisomerism from one seedling to the other only when the stress is monopolar (on one seed from the pair).

2. The "memory" of the system of seeds is formed beginning with the 5 hours of their joint swelling (PSP 58.0 : 42.0), but transmission of bioisomerism in stress conditions (for example, low temperature) does not take place yet (PSP is 15.8 : 44.2). It begins during the 24 hours of joint seed soaking. Evidently, early "memory" of phytocenosis as a certain energy-informational volume image of the future seedling does not yet have enough resistance for the following distant address-aimed transmission under the stress of conditions.

3. The strength of the stress also influences the effect of distant transmission. So under 1 hour of chemical stress this transmission does not happen (PSP – 54.7 : 46.3), and with a dose of 100 Gy the effect is less marked than with a dose of 250 Gy (PSP – 43.9 : 56.1 and 27.8 : 72.3).

The results described in plants have been obtained for the first time. They clearly testify to the presence of a nonlinear structural-functional connection between components of a seed pair that were disconnected in space. An informational long-lasting action of the same type was theoretically (Bell's theorem) and experimentally proved in quantum mechanics on the level of elementary particle interaction. On the basis of our data, the supposition can be made that the "memory" of phytocenosis, consisting of the pair of adjoining seeds in the initial stage, is formed with the help of energy-informational images of the future

seedlings, and these images are able to be realized in the substantial structure of the object (seedling and, probably, adult plant) locally and remotely during ontogenesis.

COLUMBIA, MISSOURI
University of Missouri

One mutant rescues another: *orange pericarp* restores DIMBOA production to *bx*

--Wright, A

In maize, indole is a precursor to the antifeeding compound DIMBOA. The indole is produced from a modified TrpA protein (indole synthase). The *bx* (benzoxazinineless) phenotype is due to mutation in the indole synthase gene, although DIMBOA can be produced in *bx* seedlings from exogenous indole (Melanson et al., Proc. Natl. Acad. Sci. USA 94:13345-13350, 1997)

Since the *orange pericarp* (*orp1, orp2*) mutant accumulates indole due to mutation in the tryptophan synthase B subunit (Wright et al., Plant Cell 4:711-719, 1992), it might be reasonable to expect DIMBOA to be produced in *bx*, *orp1, orp2* seedlings. To test this possibility I obtained from an F2 family (segragating for *orange pericarp* and *bx*) a self-pollination that was homozygous *bx* and segregating *orange pericarp*. Ten-day old coleoptiles from *bx* that were also *orange pericarp* showed a positive blue color reaction to 5% methanolic ferric chloride. This color reaction was nearly as intense as non-mutant, although it was absent in *bx* which were not *orange pericarp*. This finding is consistent with previous work demonstrating indole as a precursor to DIMBOA.

CÓRDOBA, ARGENTINA
Universidad Nacional de Córdoba

Preliminary studies on combining ability effects for canopy temperature in corn (*Zea mays* L.)

--Biasutti, CA, Alemanno, GA

Canopy temperature has been indicated as a trait to discriminate among drought tolerant and non-tolerant maize genotypes. There are no reports on the gene action responsible for canopy temperature in corn. The objective of this work was to study the gene action for canopy temperature in maize and its impact on grain yield.

Ninety-three breeding lines were screened during two consecutive years for canopy temperature (CT). A hand-held infrared thermometer was employed to take canopy temperature. Temperature was recorded in breeding trials from 10 plants of each of 2 replications of each line in each year. Five measures were taken form the same plant during the vegetative phase in each line. Temperature was recorded around noon (11 – 13 hs) on sunny cloudless days. Medium values for each line were utilized to classify them as high (HCT) or low (LCT) canopy temperature lines. No other agronomic attributes were considered when the lines were selected for CT. The inbreds 6A, 11 and 86C were classified as LCT and inbreds 83A, 48B and 70A as HCT. All inbreds were derived from the same population by the pedigree selection method.

During the summer of 2001/2002 fifteen single cross hybrids among the six inbred lines selected for high or low canopy temperature were produced by the diallel-mating scheme.

In the summer of 2002/2003, a field experiment with 23 entries (15 single crosses, 6 parental inbreds and 2 checks) was conducted at the Experimental Station of Facultad de Ciencias Agropecuarias de Córdoba, in Córdoba province. The design was in randomized complete block, with three replications in plots 7 m long and spaced 0.7 apart, with 46.6 plants per plot. Canopy temperature of all entries was recorded in the same way as described above. Grain yield was recorded on a plot basis and corrected at 14% grain moisture.

A preliminary analysis of variance was made for CT and GY. The general combining ability (GCA) and specific combining ability (SCA) information were obtained by using the method 2 model I analysis of Griffing (Austr. J. Biol. Sci. 9:463-493, 1956), for the variables canopy temperature (CT) and grain yield (GY).

The inbred and hybrid means for canopy temperature and grain yield are in Table 1. The analysis of variance of the diallel crosses indicated significance for GCA and SCA for the two traits analyzed (Table 2). The canopy temperature and yield mean squares for SCA were larger than for GCA. The proportion of GCA in relation to SCA can be calculated by GCA/(GCA+SCA) (Baker, Crop Sci. 18:553-536, 1978). The values of this relation

Table 1. Means of 6 maize inbred lines and 15 diallel crosses evaluated for canopy temperature and grain yield.

Genotypes	Canopy Temperature °C	Grain yield q/ha
6A	23.46	13.69
11	23.73	47.45
86C	24.70	32.74
83A	28.03	43.81
48B	28.33	10.71
70A	28.27	29.76
6A x 11	27.62	53.19
6A x 86C	27.32	47.41
6A x 83A	26.10	49.75
6A x 48B	25.26	42.44
6A x 70A	27.85	47.66
11 x 86C	26.54	46.67
11 x 83A	27.54	31.54
11 x 48B	25.52	42.22
11 x 70A	25.86	45.82
86C x 83A	27.64	72.62
86C x 48B	26.28	73.03
86C x 70A	25.70	53.66
83A x 48B	25.56	46.87
83A x 70A	24.80	63.91
48B x 70A	25.10	61.48
LSD	1.35	13.24

Table 2. Analysis of variance of 15 diallel crosses among six maize inbred lines for canopy temperature (CT) and grain yield (GY), according to Method 2 of Griffing (1956).

Source	d.f.	Mean squares*	
		CT	GY
Genotypes	20	6.48	764.63
GCA	5	2.89	487.81
SCA	15	7.68	856.90
Error	40	0.67	64.40

*F tests were significant in all instances at P<0.01.

for CT and yield were 26 and 36% respectively, indicating that nonadditive genetic effects are very important. It is known that, in maize, nonadditive effects are generally more important for yield than for other traits, as indicated by Hallauer and Miranda (Quantitative analysis in maize breeding, Iowa State Univ. Press, Ames, p. 468, 1988). The greater expression of nonadditive effects for yield must be partially due to the presence of deleterious genes that cause inbreeding depression.

Low CT Inbred lines showed negative GCA effects, whereas high CT lines showed positive GCA effects (Table 3). Inbred 83A (HCT) had the largest GCA estimate for CT. Inbred 48B had the lesser GCA estimate for CT and a large negative GCA estimate for yield.

Table 3. Estimates of general and specific combining ability effects for inbred lines and for hybrids, respectively, for canopy temperature and grain yield in maize.

Genotypes	Canopy Temperature	Grain Yield
6A	-0.333	-6.372
11	-0.399	-0.558
86C	-0.107	5.007
83A	0.495	4.187
48B	0.080	-3.918
70A	0.264	1.655
6A x 11	2.104	14.57
6A x 86C	1.512	3.230
6A x 83A	-0.310	6.390
6A x 48B	-0.735	7.186
6A x 70A	1.671	6.833
11 x 86C	0.798	-3.323
11 x 83A	1.196	-17.633
11 x 48B	-0.408	1.153
11 x 70A	-0.252	-0.820
86C x 83A	1.003	17.880
86C x 48B	0.058	26.396
86C x 70A	-0.705	1.453
83A x 48B	-1.263	1.056
83A x 70A	-2.207	12.523
48B x 70A	-1.492	18.199

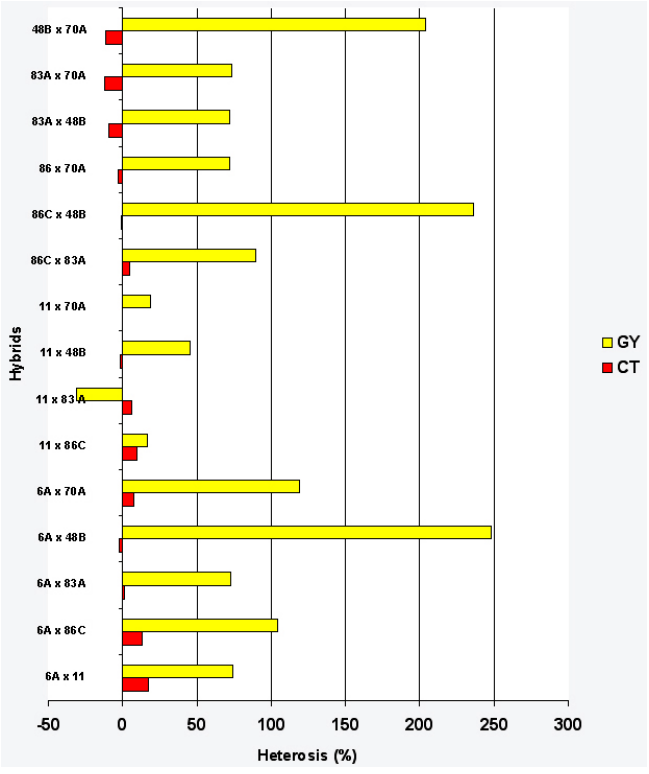

Figure 1. Heterosis for canopy temperature (CT) and grain yield (GY) in 15 maize hybrids.

The SCA estimates obtained by the Griffing (1956) method are equivalent to the specific heterosis obtained by the Gardner and Eberhart (Biometrics 22:439-452, 1966) analysis. Crosses with major positive heterosis for grain yield showed little or negative heterosis for canopy temperature (Figure 1). According to the data analyzed we can conclude that there is variability for CT among the inbred lines and so it is possible to select for low or high canopy temperature in breeding lines. High yield heterosis was found in LCT x HCT crosses. Further research is needed, with data from different environments, to confirm or negate these conclusions.

CORVALLIS, OREGON
Oregon State University

Re-targeting of a duplicated gene product and the evolution of novel gene function

--Kulhanek, D, Keppinger, J, Rivin, C

The tetraploid ancestry of maize and related species has resulted in a genome consisting of many collinear chromosome segments carrying duplicated genes. The retention of such a high proportion of these duplicated genes is likely to derive from a variety of selection mechanisms. Our studies of the duplicate maize genes *cpx1* and *cpx2* suggest that they have diverged in function by a change in protein targeting that is unique to the Maydeae.

Maize *cpx1* and *cpx2* occupy syntenous positions on chromosomes 2 and 10 and encode almost identical enzymes for coproporphyinogen III oxidase (AKA coprogen, CPX or CPO), a critical step in the synthesis of chlorophyll and heme (Figure 1). Both genes are expressed at similar levels in all tissues. To date, CPX activity has been found exclusively in the plastids in plants. Like all other plants, the *cpx1* gene encodes an enzyme with an N-terminal extension that functions as a plastid transit peptide. However, the *cpx2* gene is deleted for all but a small remnant of this targeting information. At the same time, small insertions and deletions create a new contiguous 5' ORF for *cpx2* that has the features of a mitochondrial targeting peptide (Fig. 2). To test for targeting, we fused the first exon of *cpx1* and *cpx2* to the *GFP* gene and introduced the fusions biolistically into maize leaves. The *cpx1-GFP* localized to the chloroplasts and the *cpx2-GFP* localized to mitochondria (see Williamson et al., MNL 2002).

The sequenced plant genomes, rice and *Arabidopsis*, have only a *cpx1*-type gene containing a plastid targeting peptide. This is also the only type of cpx found in barley and wheat. Using PCR, we looked for the presence of the *cpx2*-type gene in a variety of monocots, including various *Zea* species, *Z. mays* subspecies and *Tripsacum dactyloides*. Both types of genes are found within the Maydeae, but we have no evidence for *cpx2* outside this group of plants. The characteristic first exon of each gene was sequenced from *Tripsacum* and several *Zea* species. There was strong conservation of the *cpx1* and *cpx2* types of targeting information, as well as the beginning of the enzymatic region of the proteins.

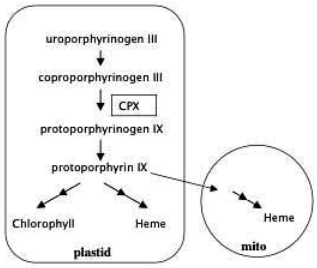

Figure 1. Tetrapyrrole synthesis in higher plants is divided between the plastid and the mitochondrion

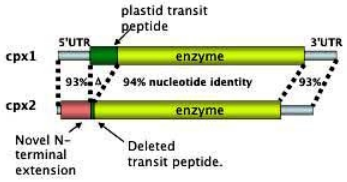

Fig 2. Comparison of cpx1 and cpx2 cDNAs Nucleotide identity is >90% throughout, but the transit peptide sequence is deleted from cpx2 and a novel ORF encodes an N-terminal extension that can direct mitochondrial localization.

The maintenance and expression of a *cpx2* gene in maize and its relatives implies that it has selective value, but the role of this protein is mysterious. The substrate for CPX is thought to be solely in the plastid. A mitochondrially localized *CPX2* suggests that earlier steps of heme biosynthesis may occur uniquely in the mitochondria of maize and its relatives. A null mutant for the *cpx1* gene was obtained by TUSC screening with the help of Pioneer HiBred Int'l. This mutant lacks all chlorophyll and is necrotic in the light due to the accumulation of phototoxic tetrapyrrole intermediates. However, seedlings and callus cultures can be maintained indefinitely in the dark on culture media, implying a capacity to synthesize heme (and not chlorophyll) in the absence of *CPX1*. We have created double mutants of *cpx1* and *cpx2* to test whether this growth capacity in the dark depends on the *cpx2* gene contributing to the production of heme. We are also looking at the effect of a *cpx2* mutation on the necrotic phenotype of the *cpx1* mutant in the light, to learn if *CPX2* may act to scavenge and detoxify tetrapyrrole intermediates in the cytoplasm or mitochondria.

Tousled-like kinases of maize are up-regulated in dividing tissue and can functionally complement the *tousled* mutant of *Arabidopsis*

--Owusu, EO, Rivin, CJ

The Tousled-like kinases (TLKs) are an evolutionarily conserved family of ser/thr kinases that localize to the nucleus. The first member of this gene family was isolated in *Arabidopsis thaliana* via a T-DNA insertion (Cell 75:939-950). *Arabidopsis* Tousled (TSL) mutants have a dramatic floral phenotype, showing a decrease in the number of sepals, petals and stamens, and a split gynoecium. Work on TLKs in mammals, *C.elegans* and *Drosophila* suggests that TLKs are involved in chromatin remodeling and transcriptional regulation during development. In mammals, activity of TLK proteins is highest during S phase of the cell cycle, but there is not pronounced regulation at the expression level.

In maize, there are 3 *TLK* genes designated *zmTLK1*, *zmTLK2* and *zmTLK3*. Like the *Arabidopsis* gene, the *zmTLK*s are expressed in all tissues at a low level, and are more highly expressed in floral tissue. To test whether *zmTLK* expression is generally up-regulated in dividing tissues, expression of the cyclin B and *zmTLK* genes were examined in multiplex RT-PCR. Both sets of genes are expressed at a higher level in all dividing tissues, i.e. root tip, dividing leaf, young ears and tassels and 9 DAP endosperm as compared to non-dividing tissues like expanding and differentiating leaf sections, silks, culm, and differentiating root sections, though differences in the level of mRNA are more pronounced for cyclinB than for any of the *zmTLK*s.

All *zmTLK* genes encode C-terminal catalytic domains with high homology to TSL (84% identity and 93% similarity). The amino acid sequences of the regulatory N-terminal are far less conserved in relation to *Arabidopsis*, but they share many structural features, such as nuclear localization signals and a series of coiled-coil domains. Using the floral dip method, TSL mutant plants were transformed with and fully complemented by overexpression constructs of *zmTLK2*, indicating that all the key regions are conserved between the *Arabidopsis* protein and *zmTLK2* and that they function in the same pathway.

Two classes of Tousled-like kinases indicate two rounds of gene duplication

--Owusu, EO, Rivin, CJ

The three *TLK* genes of maize were originally mapped to three loci, 1.12, 4.05 - 4.06 and 5.02 - 5.03 on the 1995 UMC map (Helentjaris et al., MNL 69, 1995). Sequence data obtained from genomic and cDNA clones from inbred B73 indicate that the three maize Tousled-like kinases, designated *zmTLK1*, *zmTLK2* and *zmTLK3*, fall into two classes. The *zmTLK2* and *zmTLK3* genes are 96% identical over their catalytic regions, while *zmTLK1* shares only 83% identity in the same region. (The complete *zmTLK* cDNA sequence has now been deposited in Genbank, accession number AY496080).

To learn something about the origins of these genes, we asked whether the identical genes were found on the syntenous chromosome locations on chromosomes 1 and 5, or on the non-syntenous positions 1 and 4 or 4 and 5. Using the oat-maize hybrid lines developed by Dr. R. L. Phillips (Plant Physiol. 125:1216-1235, 2001) we mapped *zmTLK1* to maize chromosome 1. The two nearly identical *zmTLK2* and *3* genes were found on the non-syntenous positions on chromosomes 4 and 5. These positions suggest that the two different classes were present in the tetraploid maize ancestor, and that a second duplication occurred to create the closely related *zmTLK2* and *3*. This model is also supported by the finding that both classes of *TLK* genes are present in *Z. luxurians*, *Z. diploperennis*, *Z. mays mexicana* and *Tripsacum dactyloides*.

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Electrophoretic analysis of progeny of maize matroclinal haploids

--Dzubetskiy, BV, Satarova, TN, Cherchel, VYu, Klyavzo, SP

Intensifying the breeding process in maize requires methods for rapid establishment of homozygotic lines. Inbreeding is the most widely used method to obtain homozygotic material, but requires several years. In contrast, obtaining androgenic and matroclinal haploids and doubling their chromosomes requires only 1-2 years, and should lead to complete homozygosity.

Distinguishing matroclinal haploids through genetic markers created by S. Chase (Bot. Rev. 35:117-167, 1969) allows selection of naturally occurring haploids that appear as a result of the development of unfertilized egg cells. Crossing an original population with a line containing dominant genes of plumule coloration allows visual separation of seeds with haploid embryos from hybrid seeds.

The method of genetic markers developed in detail by E. Zabirova and O. Shatskaya (Proc. Int. Symp.: Genetics, Selection and Technology of Corn Cultivation, Krasnodar, Russia: 1999, 219-227, 1999) was used to obtain haploids. Intercrossing of different lines derived from Lancaster germplasm previously created the population Synt 1950/1952, which then became the source for obtaining haploids. The line, Krasnodarskiy Embryo Marker - 1 (ZMK-1), kindly donated by Dr. E. Zabirova and Dr. O. Shatskaya, was used as a pollinator in order to recognize haploids. In our experiment, 1266 kernels were examined after crossing with the marker line. The frequency of haploids was 1.8% of the total number of kernels observed. Only 54.8% were confirmed as haploids among the separated seeds with possible haploid embryos that had germinated in soil. Diploidization of haploids was carried out at the 3-5-leaf stage, with a 0.125% solution of colchicine, according to the technique of E. Zabirova et al. (Maize and Sorghum 4:17-19, 1996). The efficiency of diploidization was 10.5%, but only two dihaploids were self-pollinated owing to the difference in dates between tasseling and ear flowering. Thus, we obtained dihaploid (autodiploid) lines DKh 50/02 and DKh 55/02 from the initial population Synt1950/1952.

Zein, the main storage protein in corn grain, demonstrates extremely high heterogeneity, and includes up to 22 bands in its electrophoretic spectrum. Examination of the spectra of zein reveals the level of heterozygosity of many loci dispersed among several chromosomes. Of lines that are homozygous in zein-coding loci (evaluated by electrophoresis), the great majority of self-pollinated lines show no variability in morphological and agronomic characteristics. In heterozygotes, bands of both maternal and paternal forms are present in the isozyme spectrum (Asika, Poperelya, Control of genetic purity of maize hybrids by electrophoretic analysis, Methodological recommendations, Min. of Agricult., Moscow, 1991). We investigated the reduction of electrophoretic band diversity of zein components in matroclinal dihaploid lines DKh50/02 and DKh55/02 compared to the source population.

The electrophoretic analysis of zein was carried out using polyacrylamide gel with an acetic acid-glycine buffer. The acid buffer and high concentrations of urea and 2-merca-proethanol separated the storage proteins into polypeptides, each representing the product of a single gene or a cluster locus (Poperelya, Asika, Lazarev, 1991, A.C. (Patent) USSR N 16822899). For the line DKh50/02, 80 kernels resulting from the second self-pollination and 10 kernels from the first self-pollination of the line DKh55/02 were analyzed. Forty kernels of the initial population Synt 1950/1952 were analyzed.

In 1950/52, there were four isozyme patterns (biotypes) containing more than one kernel (with 2,3,5 or 6 kernels each). The other 24 kernels each showed a unique pattern. This demonstrates that the initial population was highly heterogeneous for the polypeptides of zein (Fig. 1). In contrast, all the kernels of the dihaploid line DKh50/02 revealed the same isozyme pattern (Fig. 2). All the kernels of the other dihaplod line (DKh55/02) revealed a uniform isozyme pattern that was different from that of DKh50/02 (Fig. 3). The spread of some individual bands was also reduced in the dihaploid lines compared to the initial

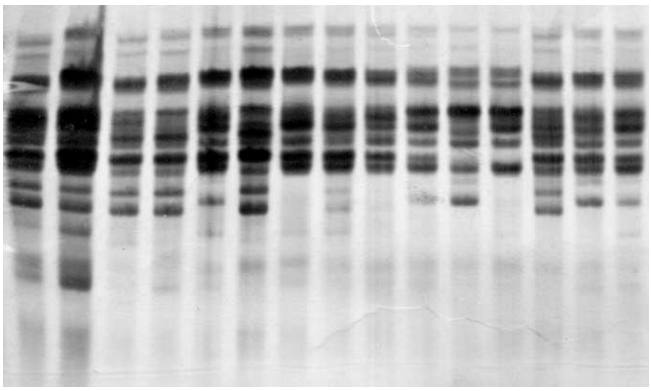

Figure 1.

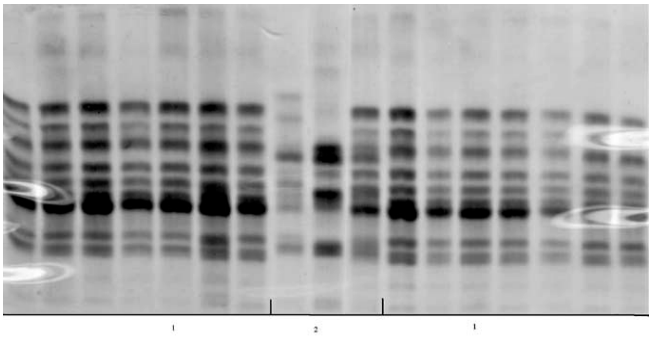

Figure 2.

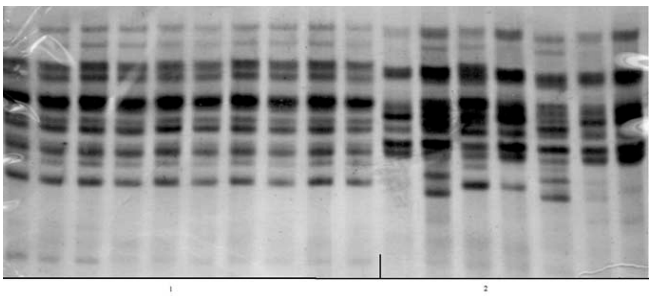

Figure 3.

population, and some bands present in the initial population were not present in the dihaploids.

The uniformity of the protein components of zein, as the main storage protein and one with complex composition, confirms that the matroclinal haploid lines identified by the genetic marker were homozygous.

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High resolution crossover maps for each bivalent of *Zea mays* using recombination nodules

--Anderson, LK, Doyle, GG, Brigham, B, Carter, J, Hooker, KD, Lai, A, Rice, M, Stack, SM

Recombination nodules (RNs) are closely correlated with crossing over, and, because they are observed by electron microscopy of synaptonemal complexes (SCs) in extended pachytene chromosomes, RNs provide the highest resolution cytological marker that is currently available for defining the frequency and distribution of crossovers along the length of chromosomes. Using the maize inbred line KYS, we prepared an SC idiogram in which each SC was identified by relative length and arm ratio. This idiogram is very similar to published idiograms of maize pachytene chromosomes based on squashes. We further confirmed identification of each SC with its linkage group by examining inversion heterozygotes. We mapped 4267 RNs on 2080 identified SCs to produce high resolution maps of RN frequency and distribution on each bivalent. RN frequencies are closely correlated with both chiasma frequencies and SC length. The total length of the RN recombination map is about two-fold shorter than most maize linkage maps, but there is good correspondence between the relative lengths of the different maps when individual bivalents are considered. Each bivalent has a unique distribution of crossing over, but all bivalents share a high frequency of distal RNs and a severe reduction of RNs at and near kinetochores. The frequency of RNs at knobs is either similar to or higher than the average frequency of RNs along the SCs. We suggest a model for knob structure that may explain this observation. These RN maps represent a measure of crossing over along maize bivalents that is independent from and complementary to linkage maps. RN maps can be used to evaluate patterns of crossing over and genetic interference.

This work is reported fully in: Anderson, L.K., G.G. Doyle, B. Brigham, J. Carter, K.D. Hooker, A. Lai, M. Rice, and S.M. Stack, 2003. High resolution crossover maps for each bivalent of *Zea mays* using recombination nodules. Genetics 165:849-865.

Acknowledgements. We thank Ben Burr and Ed Coe for providing KYS seeds. This work was supported by a grant from the National Science Foundation (MCB-9728673). K.D.H., A.L. and B.B. were supported by grants from the National Science Foundation for Research Experience for Undergraduates.

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Integrating genetic linkage maps with pachytene chromosome structure in maize

--Anderson, LK, Salameh, N, Bass, HW, Harper, LC, Cande, WZ, Weber, G, Stack, SM

Integrating genetic linkage maps with chromosome structure has been an important objective ever since it was demonstrated that genes occur in a fixed order on chromosomes. Linkage maps are defined by the percentage of recombination between markers [as expressed in centiMorgans (cM)] and reveal the linear order of markers. However, they do not contain in (positions on chromosomes) or as a physical length (number of formation on the actual physical distance between markers, whether that distance is expressed as a cytological length DNA base pairs). This is because crossing over is not evenly distributed along chromosomes. Crossing over is suppressed in heterochromatin and centromeres, and crossing over is variable even in euchromatin where most crossing over occurs. As a result, linkage maps cannot be simply overlaid on chromosomes to determine the physical position of genes. One way to integrate linkage maps with chromosome structure is to utilize high-resolution cytological markers of crossing over, such as recombination nodules (RNs). RNs are proteinaceous, multi-component, ellipsoids approximately 100 nm in diameter, which are found in the central region of synaptonemal complexes (SCs) between homologous chromosomes (bivalents) at pachytene. Evidence that RNs mark crossover sites include the close correspondence between the frequency and distribution of RNs compared to chiasmata, the presence of an essential crossover protein (MLH1p) in RNs, and the presence of MLH1p/RNs at chiasma sites. Because RNs can be observed only by electron microscopy of SCs in elongate pachytene bivalents, RNs represent the highest resolution markers available for determining the chromosomal location of crossing over. Each RN represents one crossover between two homologous non-sister chromatids, which yields two recombinant and two parental chromosomes that is, by definition, equivalent to 50 cM on a linkage map. On this basis, the frequency of RNs can be converted to cM and used to prepare a detailed map of recombination along the physical length of each of the ten pachytene chromosomes/SCs in maize. Because RN maps relate the amount of recombination to cytological position along pachytene chromosomes and linkage maps report the amount of recombination relative to genes or other markers, it is now possible to combine these two approaches to directly relate genetically-mapped markers to cytological position. We have used this procedure to predict the physical position of genetically mapped core bin markers on each of the ten chromosomes of maize. We tested our predictions for chromosome 9 using seven genetically-mapped, single-copy markers that were independently mapped on pachytene chromosomes using in situ hybridization. The correlation between the predicted and observed locations was very strong

(r^2 = 0.996), indicating a virtual 1:1 correspondence. Thus, this new, high-resolution, cytogenetic map enables one to predict the chromosomal location of any genetically mapped marker in maize with a high degree of accuracy. This work has been accepted for publication in Genetics.

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The large second intron of the MADS box transcription factor gene *ZmMADS2* is transcribed in maize tissues where the protein seems not to be required

--Dresselhaus, T, Schreiber, DN

We are interested in the function of MADS box transcription factor genes in both male and female gametophytes (pollen and embryo sac, respectively; see also Schreiber, Dresselhaus, Flowering Newsl. 35:8-16, 2003) and have cloned 7149 bp of the maize MADS box gene *ZmMADS2* (GenBank accession # AY227363). *ZmMADS2* consists of eight exons (E1-E8; see Figure 1) and encodes a class II MADS domain protein of the MIKC-type. This type of MADS domain protein contains an N-terminal MADS box (M: in *ZmMADS2* encoded by exon 1), followed by an intervening region (I: in *ZmMADS2* encoded by exon 2), a keratine-like box (K: in *ZmMADS2* encoded by exons 3-5) and a highly variable C-terminus (C: in *ZmMADS2* encoded by exons 6-8). The length of the transcript is around 1.3 kb and the size of the encoded protein is 240 aa.

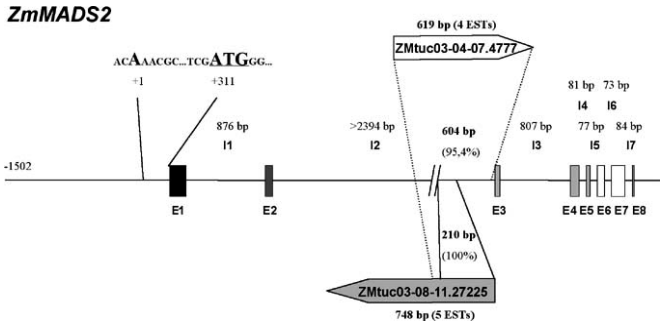

Figure 1. Genomic structure of the MADS box transcription factor gene *ZmMADS2*. The *ZmMADS2* gene consists of eight exons (E1 – E8) and seven introns (I1 - I7). The second intron (I2) is unusually large. The size of all seven introns is indicated. The MADS box (M) is encoded by exon 1 (black box), the intervening region (I) by exon 2 (dark grey box), the keratine-like box (K) by exons 3-5 (light grey boxes) and the highly variable C-terminus (C) by exons 6-8 (white boxes). The ATG START codon is located 311 bp downstream of the transcription start point. The 3' region of the second intron (I2) is transcribed in various maize tissues (see text) as indicated by the two TUCs (tentative unique contigs), each represented by four and five ESTs, respectively.

ZmMADS2 was previously reported to be specifically expressed in maize pollen and during maize pollen tube growth (Heuer et al., Sex. Plant Reprod. 13:21-27, 2000). A more detailed expression study revealed that *ZmMADS2* is also weakly expressed in root tips and strongly expressed in endothecium and connective tissues of the anther at one day before dehiscence, and in mature pollen after dehiscence. Development of both anthers and pollen was arrested at one day before dehiscence in transgenic plants expressing the 3' part of the *ZmMADS2*-cDNA in antisense orientation, indicating that the protein is required for anther dehiscence and pollen maturation. High amounts of a ZmMADS2-GFP fusion protein accumulate in degenerating nuclei of endothecial and connective cells of the anther, but could not be detected in germinated pollen tubes (Schreiber et al., Plant Physiol. 134:1069-1079, 2004).

Here, we report about the second intron of the *ZmMADS2* gene. Concerning MADS box genes from other plant species, this intron, with more than 2.4 kbp, is unusually large. After obtaining 2394 bp, we have stopped sequencing the second intron. The isolation of independent clones from a genomic library of the maize inbred line A188 suggests that the intron is even much bigger. None of the genomic clones isolated after screening a genomic library with the 3' UTR of *ZmMADS2* contained the 5'UTR, although the clones were relatively large in size with a length varying between 11 and 23 kbp. Instead, we found that the 3' part of the second intron is transcribed in various maize tissues. One end of TUC (tentative unique contig) ZMtuc03-08-11.27225 (748 bp), a contig of five ESTs (expressed sequence tags), is, over a length of 210 bp, 100% identical to the 3' end of the second intron of *ZmMADS2* (Figure 1). Adding the remaining 538 bp of ZMtuc03-08-11.27225 to the intron sequence of I2 increases the size of the intron to almost 3 kbp, although we assume that the intron is even much larger. Three of five ESTs were obtained from roots and thus overlap with the expression of *ZmMADS2*, while the two remaining ESTs were isolated from seedlings and the cell division part of the 6th leaf, thus tissues where *ZmMADS2* transcript could not be detected. It is unlikely, that the promoter of *ZmMADS2*, which is located a few kbp upstream of the 3' end of I2, influences transcription from this region. It is more likely that promoter(-like) elements in I2 are responsible for the expression. Interestingly, ZMtuc03-08-11.27225 does not contain an obvious open reading frame (ORF) and thus may not encode a protein, but instead be required for processing and stability of the *ZmMADS2* transcript. Transgenic maize plants have shown that ZmMADS2 is required for anther and pollen maturation, but neither for root development nor for cell division, where low or no *ZmMADS2* transcript amounts could be detected. From the expression of ZMtuc03-08-11.27225 in these tissues, we suggest that the intron-transcript could be involved in *ZmMADS2* transcript stability/degradation with the aim of silencing ZmMADS2 activity in those tissues where the protein is not required.

ZmMADS2 is not a single copy gene. Using the 3'UTR as a probe, we obtained at least three bands after digesting DNA of the inbred line A188 with various restriction enzymes (data not shown). This could be one explanation for the finding that we identified a second TUC, ZMtuc03-04-07.4777 (619 bp), transcribed from the intron of a MADS box gene allelic to *ZmMADS2*. Like ZMtuc03-08-11.27225, ZMtuc03-04-07.4777 does not contain an obvious ORF and thus might also be involved in transcript stability/gene regulation. Sequence identity between ZMtuc03-04-07.4777 and the 3'end of I2 of ZmMADS2 as well as with one end of ZMtuc03-08-11.27225 is almost 96% over the whole contig. All ESTs of ZMtuc03-04-07.4777 were obtained from mixed BMS (Black Mexican Sweet) tissue and suspension cultures and therefore could also represent a DNA

polymorphism. The genomic sequence of *ZmMADS2*, as well as the full length cDNA, were obtained from other lines, namely inbred line A188, and the ESTs of ZMtuc03-08-11.27225 were from cultivars W23 and F2.

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Differentiation process of maize root cells at reduced temperature

--Akimova, GP, Sokolova, MG, Makarova LE, Nechaeva, LV

The present work was aimed at the investigation of cooling impact on the speed of maize root cell differentiation based on biochemical indices. We took into account differences between varieties, which play an important role in the plants' response to reduced temperature (MNL 76:35-36, 2002).

The tests were conducted on 48-hour-old seedlings. Maize varieties under investigation were characterized by different growth reaction to temperature decrease (MNL 71:93,1997); in particular, the highly resistant variety (Omskaya 2) has demonstrated stationary growth at intense temperature decrease, that is, the processes of cell formation and the beginning of their extension were equally slowed down. The non-resistant variety (Uzbekskaya tooth-like) demonstrates deregulation of these processes.

A temperature of 10 C was proven to slow down the accumulation of basic components of cell capsules (pectins, hemicelluloses, cellulose, lignin). At the same time the resistant variety (Omskaya 2) in the zone of cells, which completed extension (in 48 hours at 10 C), showed that the speed of accumulation of the components mentioned dropped in direct proportion to the deceleration of root growth (Table 1). The cells, which started extension at a temperature of 10 C, exhibited a significant decrease in the speed of pectin accumulation, but an increase in the content of hemicelluloses, cellulose and lignin. The latter, in our view, characterizes a higher degree of differentiation of these cells at 10 C and apparently represents a peculiarity of the resistant variety.

Table 1. Content of soluble ethers of hydroxycinnamic acids (HCA) and major components of capsules of maize seedling root cells.

Variant	Root zones *	Pectins	Hemicelluloses	Cellulose	Lignin	HCA
		g X 10^{-11} /cell	g X 10^{-11} /cell	g X 10^{-11} /cell	% of weight of dry cell substances	g X 10^{-6} /cell
Omskaya 2						
27 C, 6 h	1	38.5±2.4	63.4±5.1	31.5±2.5	2.0	194.43±15.7
	2	66.4±3.1	131.0±7.5	68.1±3.5	2.4	987.48±73.4
10 C, 48 h	1	32.0±1.3	66.1±1.7	32.8±1.6	2.8	216.94±11.4
	2	68.0±2.8	150.4±12.5	70.2±3.7	3.1	333.26±35.1
Uzbekskaya tooth-like						
27 C, 8 h	1	36.0±1.1	96.6±3.2	54.0±0.7	4.2	362.48±22.3
	2	86.8±5.5	348.5±25.0	195.0±14.1	4.2	1647.74±102.0
10 C, 96 h	1	72.7±1.5	161.7±2.4	81.0±2.2	4.1	1100.07±138.0
	2	86.0±5.3	417.6±28.7	244.3±16.8	12.8	2040.61±144.0

*1, 2 – beginning and end of cell extension.

In the non-resistant variety the content of pectin in the capsules of cells, which started extension at 10 C, increased considerably in control plants, whereas lignin content in percentage to the cell dry substance content did not change. Such a proportion between the components evidently brings about higher plasticity of the capsules, which results in a significant increase of the cell volume. The proportion of the capsules' major components (Uzbekskaya tooth-like variety) in the cells that completed extension, changed towards larger lignin accumulation with simultaneous reduction of pectin content at a low positive temperature. The capsules of these cells acquired intensified rigidity, which increased with the root growth and at 10 C resulted in the deceleration of cell growth; consequently, it took considerably longer for the cells to reach certain dimensions than it did in optimal conditions (96 hours of growth at 10 C versus 8 hours of growth at 27 C).

Accumulation of phenolic compounds (lignin predecessors) increased in the cells under extension in both varieties. Nevertheless, in the non-resistant variety metabolism of hydroxycinnamic acids is significantly shifted towards the increase of content of soluble ethers, in the resistant variety – towards that of lignin. In the cells that completed growth, the balance of consumption of oxycinnaroic acids is shifted towards lignin synthesis in both varieties (Table 1).

Thus, peculiarities of cell capsule formation (linked to the change of speed of the capsule's accumulation of major components) at temperature reduction represent one of the key factors of cell extension.

Cold adaptation induction of AOX isophorm in maize

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Antipina, A, Truhin, A, Popov, V

Higher plants have two mitochondrial electron transport pathways from ubiquinone to oxygen. One is an electron transfer through the cytochrome pathway coupled to ATP production and inhibited by cyanide. The alternative pathway branches from the cytochrome pathway and donates electrons to oxygen directly by alternative oxidase (AOX) (Moore and Siedow, Biochim. Biophys. Acta. 1059:121-140, 1991). The alternative pathway is not coupled with ATP synthesis, but can be induced in response to stress or inhibition of the main electron transfer pathway. When plants are exposed to low temperatures, they produce reactive oxygen species (ROS) such as superoxide and hydrogen peroxide (Prasad et al., Plant Cell. 6:65-74, 1994). ROS damages membranes and other cell components. It has generally been proposed that increased AOX activity could be used to help maintain normal levels of metabolites and to reduce levels of reactive oxygen species (ROS) during the cold. In maize the part of electron partitioning to the alternative pathway was significantly increased under low temperatures, though it may not correlate with tolerance to cold (Ribas-Carbo et al., Plant Physiol. 122:199-204, 2000). A nuclear gene encodes alternative oxidase. The genes of AOX consist of a small multigene family. Three members were found in soybean (Whelan et al., Planta 198:197-201, 1996), rice (Ito et al., Gene 203:121-129, 1997; Saika et al., Genes. Genet. Syst. 77:31-38, 2002) and four members in *Arabidopsis* (Saisho et al., Plant Mol. Biol. 35:585-596, 1997) and mango (Considine et al., Plant Physiol. 2001 126:1619-1629). In maize a set of isophorms of AOX have also been demonstrated (Karpova et al., Plant Cell. 14:3271-3284, 2002).

The alternative oxidase protein levels in the mitochondria were studied after short cold stress at 2 C, as well as after adaptation during 7 days at 12 C. Four day etiolated maize seedlings grown at 20 C were used as control. Mitochondria were isolated

and purified as described earlier (Borovskii et al., BMC Plant Biol. 2:5, 2002). The expression of AOX proteins was performed by immunoblotting with AOX specific antibodies (kindly provided by Dr. T. E. Elthon, University of Nebraska).

Immunoblotting revealed that the level of AOX protein is raised under low temperature adaptation (Fig. 1). It is easy to

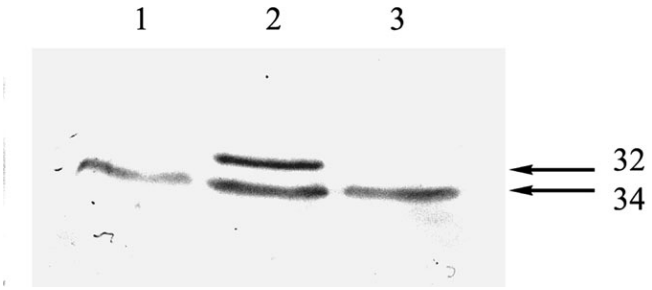

Figure 1. Western analysis of maize mitochondrial AOX proteins. Mitochondria were isolated from the control (1), 7 days cold (12 C) acclimated (2) and cold (2 hours at 2^0C) stressed (3) seedlings. Molecular weights of AOX isophorms are indicated.

see that most of the total AOX level increased on account of augmentation of the content of the 34 kD isophorm. The level of 32 kD isophorm characteristic for control seedling mitochondria was not increased. The differential increase of the 34 kD isophorm level made us suspect that these proteins are independently regulated, and may have specific and different functions during adaptation of maize to cold temperature. A similar situation appeared in rice, where only the 34 kD isophorm of AOX had a correlation with low temperature tolerance and the 32 kD did not (Abe et al., FEBS Lett. 527:181-185, 2002). It has also been reported that the capacity of the alternative pathway is greater in winter wheat than in spring wheat (McCaig and Hill, Can. J. Bot. 55:549-555, 1977). Alternatively in maize, the alternative pathway was significantly increased in the more chilling-sensitive variety (Ribas-Carbo et al., Plant Physiol. 122:199-204, 2000). The exact role of AOX and other energy dissipative pathways, such as external and internal rotenone-insensitive dehydrogenases or uncoupling protein (UCP) activity during stress, still need to be defined.

This work was supported by the Russian Fund of Basic Research (project 02-04-48599).

Drought induction of AOX level in maize was accompanied by lowering of AOX respiration activity

--Stupnikova, I, Borovskii, G, Korotaeva, N, Antipina, A, Truhin, A, Popov, V

There are two mitochondrial electron transport pathways from ubiquinone to oxygen in plants. The alternative pathway branches from the cytochrome pathway and donates electrons to oxygen directly by alternative oxidase (AOX) (Moore and Siedow, Biochim. Biophys. Acta. 1059:121-140, 1991). The alternative pathway is not coupled with ATP synthesis, but can be induced in response to stress or inhibition of the main electron transfer pathway (Wagner and Moore, Biosci. Rep. 17:319-333, 1997). When plants are exposed to stresses, drought for instance, they produce reactive oxygen species (ROS) such as superoxide and hydrogen peroxide (Prasad et al., Plant Cell. 6:65-74, 1994). ROS damages membranes and other cell components. It has generally been proposed that increased AOX activity could be used to help maintain normal levels of metabolites and to reduce levels of reactive oxygen species (ROS) during the stress. We propose that drought will increase the AOX respiration level and the quantity of the protein in mitochondria of maize seedlings.

The alternative oxidase protein levels in the mitochondria were studied after drought stress at 22 C for 1 or 2 days. Four day etiolated maize seedlings grown at 22 C were used as control. Mitochondria were isolated and purified as described earlier (Borovskii et al., BMC Plant Biol. 2:5, 2002). The expression of AOX proteins was performed by immunoblotting with AOX specific antibodies (kindly provided by Dr. T. E. Elthon, University of Nebraska). It had been shown previously that after 24 hours of drought stress, water content in the seedlings decreased from 90.6 % to 87.5%, and after 48 hours, water content decreased to 82.7 % (Korotaeva et al., MNL 77:34-35, 2003). Respiration of isolated mitochondria was performed as described by Korotaeva et al. (Russ. J. Plant Physiol. 48:798-803, 2001) using malate as substrate and KCN as the cytochrome pathway of respiration inhibitor. To measure potential activity of AOX respiration, it was activated by pyruvate and dithiotreithol.

Immunoblotting analysis revealed that the quantity of AOX protein increased under dehydration stress (Fig. 1). An accumulation of oxidized dimers of AOX was shown. No new isophorm induction appeared. Nevertheless, in spite of the accumulation of AOX protein, the potential alternative respiration of isolated mitochondria decreased during dehydration: from 62±7% at V_4 respiration in control seedlings, to 36±4% at 48h of dehydration. At the same time, the level of respiration without activation was not seriously changed: from 26±4%(control) to 22±5% (48h of dehydration). The results suggest that an increase in the AOX protein level is not enough to provide high alternative respiration activity during drought stress, and made

Figure 1. Western analysis of maize mitochondrial AOX proteins. Mitochondria were isolated from the control (1), 24h (2) or 48h (3) of drought stressed seedlings.

us doubt the role of AOX during dehydration stress itself. It may be that higher AOX activity is favorable in adaptation when oxidative stress, which accompanies dehydration, is not strong.

This work was supported by the Russian Fund of Basic Research (project 02-04-48599).

Are there some species-specific differences in DNA import activity in mitochondria?

--Nepomnyaschih, DV, Koulintchenko, MV, Konstantinov, YM

It has been shown previously (MNL 64:67-68, 1990) that bacterial vector plasmids pBR322 and pBR327 can effectively penetrate into intact mitochondria of seedlings and serve as templates for DNA synthesis. Using the well-defined mitochondrial linear plasmid of 2.3 kb, we showed the existence of an active, transmembrane potential-dependent mechanism of DNA uptake into potato tuber mitochondria (Koulintchenko et al., EMBO J. 22:1245-1254, 2003). The DNA import is restricted to double-strand DNA, but has no sequence specificity. The process is most efficient with linear DNA fragments up to a few kilobase pairs. It has been concluded that DNA transport into mitochondria might represent a physiological phenomenon with some yet unknown relevance.

The aim of the present work was to investigate the potential differences in DNA import activity between mitochondria from maize seedling and potato tuber mitochondria.

Mitochondria were isolated from potato (*Solanum tuberosum*) tubers as described (Koulintchenko et al., EMBO J. 22:1245-1254, 2003). Maize mitochondria were isolated from 4-day-old etiolated seedlings of hybrid VIR42 MV by the standard method of differential centrifugation. The substrate DNA used for mitochondrial import assays was the *orf1* fragment of 2.3 kb linear plasmid from maize (Leon et al., 1989; DDBJ/EMBL/GeneBank accession No. X13704). To obtain the radioactive linear fragment, 50 ng of unlabeled PCR product and the primers (ATGACAAAGACATATAGAATATCC / TTATTCCT TTTCGCGCTGCATT) were used for a single PCR cycle in which a 10 min elongation in an unlabeled dATP-deprived reaction medium containing 100 mCi of [α-32 P]dATP (3000 Ci/mmole) per 50 µl was followed by the addition of 0.2 mM unlabeled dATP, and a further 5 min elongation.

Standard mitochondrial import of DNA was carried out in a solution (import buffer) containing 40 mM potassium phosphate, pH 7.0, and 0.4 M sucrose. The samples (200 µl) containing 1-5 ng of 32P-labeled DNA and purified mitochondria (ca 300 µg of protein) were incubated at 25 C for 45 or 90 min under mild shaking. Following addition of 200 µg of DNase I and 10 mM $MgCl_2$, the incubation was continued for 20 min in the same conditions. Mitochondria were subsequently washed three times by resuspension in 1 ml of solution containing 10 mM potassium phosphate, 300 mM sucrose, 10 mM EDTA, 10 mM EGTA, 0.1% (w/v) BSA, 5 mM glycine, pH 7.5, and centrifuged for 5 min at 10 000 g. The final pellets were extracted with one volume of buffer containing 10 mM Tris-HCl, 1 mM EDTA, 1% (w/v) SDS, pH 7.5, and one volume of phenol. The nucleic acids recovered in the aqueous phase were ethanol-precipitated, fractionated by electrophoresis on a 1% (w/v) agarose gel and transferred onto a nylon membrane (Hybond N+, Amersham Biosciences) for autoradiography.

Figure 1 shows the uptake of 2.3 kb maize mitochondrial plasmid DNA by potato tuber and maize seedling intact mitochondria. Under the conditions used, maize mitochondria import DNA much more actively in comparison with potato mitochondria. To test the functional state of both potato and maize mitochondria, we also measured their respiratory control ratio (RCR), i.e. the ratio between the respiration rate of the mitochondria upon addition of ADP (150 µ) (state 3 respiration) and the respiration rate in the absence of additional ADP (state 4 respiration). RCR was rather high (3-4) with succinate-supported respiration for both plant species mitochondria. There was no detectable influence of succinate on DNA import activity (Fig. 1). The possibility that mitochondria use some endogeneous respiration substrates (like long chain fatty acids or others) under these conditions couldn't be excluded. The differences in DNA import activity of potato and maize mitochondria might be explained by the theory that the functional state of plant mitochondria may somehow affect DNA incorporation into organelles. Further experiments are required to clarify the question of whether some species-specific differences exist in mitochondrial DNA import activity depending on plant developmental stage, tissue specialization (storage or meristematic), etc. As a whole, the studies on DNA import activity in mitochondria in a number of plant species could result in new mitochondrial transformation strategies to improve some agronomically important traits encoded by the mitochondrial genome.

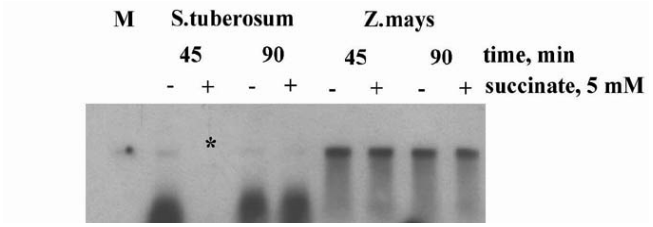

Figure 1. Uptake of DNA in potato (*Solanum tuberosum*) and corn (*Zea mays*) freshly isolated intact mitochondria. *Line where DNA wasn't loaded.

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Characterization of callusogenesis in A344, A344-CP5, Sg25 inbred lines and their hybrids

--Shmakov, VN, Mashnenkov, AS, Konstantinov, YM

The aim of the present work was to try to reveal some polymorphism if any in A344, A344-CP5, Sg25 inbred lines and their hybrids on the level of in vitro cell culture. It was shown previously that the subline A344-CP5 causes heterosis in the hybrid, including the initial line A344 (Mashnenkov, MNL 56:92-98, 1982). Callus culture for all genotypes studied was initiated from scutellum and seedling shoots. The explants consisted of scutellum and embryo excised from mature kernels of maize under aseptic conditions. Previously, kernels were submerged for about 8 h in 3% H_2O_2 in the dark. Then kernels were surface sterilized in 0.1% mercuric chloride with 0.1% saponin for 20 min, and subsequently rinsed three times with sterile water. Excised explants were transferred to vessels for plant tissue culture, containing 20 ml of culture medium under aseptic con-

ditions as blocks of 3-4 explants each. The vessels were incubated at 24±1 C in the dark. The callus induction and maintenance medium was MS basal salt containing 0.5 mg/l each of thiamine HCl, pyridoxine HCl and nicotinic acid, 80 mg/l inositol, 1 g/l each of casein hydrolysate and L-proline, 30 g/l sucrose, 2 mg/l 2,4D, and 0.2 mg/l BA.

A344-CP5 subline and hybrid A344 x 165 had the best characteristics of callusogenesis based on shoots as an explant (Table 2). It was observed that there was no case for callusogenesis on leaf explants (Table 2). It was also rather rare for epicotyls.

Table 1. Callusogenesis and seedling development characteristics of genotypes measured after 2 days of cultivation.

Genotypes	No. of explants	No. of developed seedlings	Length of shoot, mm	Length of root, mm	Initiation of callus, days	Volume of scutellum, mm^3
A344-CP5	9	9	8 ± 0.5	-	9	233 ± 22.3
A344	10	10	6 ± 0.9	-	8	232 ± 34.0
A344 X A344-CP5	10	9	11± 1.2	5 ± 0.6	9	163 ± 22.7
Sg 25	10	8	7 ± 1.4	3 ± 0.3	8	190 ± 27.0
A344 X Sg 25	10	8	12 ± 0.7	3 ± 0.5	8	267 ± 29.2
A344 X 165	10	10	12 ± 1.6	2 ± 0.3	9	228 ± 28.7

Table 2. Shoot explant callusogenesis characteristics of genotypes studied.

Genotypes	Number of explants	Initiation of leaf callus, days	Initiation of epicotyl callus, days	Initiation of callus from the upper segment of the hypocotyls, days	Initiation of callus from the medium segment of the hypocotyls, days	Initiation of callus from the lower segment of the hypocotyls, days
A344-CP5	12	-	12	6	3-6	6
A344	12	-	-	6	6	6
A344 X A344-CP5	12	-	-	6	3-10	6-12
Sg 25	12	-	-	-	-	-
A344 X Sg 25	12	-	-	-	3-12	6
A344 X 165	12	-	12	6-12	6	6

The calli growth on scutellum as an explant was relatively weak for all 6 genotypes studied (Table 1). Among all genotypes, only subline A344-CP5 demonstrated sufficient callus formation (Table 1). Sg25 inbred line scutellum couldn't serve as material for calli initiation at all. The longevity of calli cultivation on the medium we used in these experiments was restricted by about 2 weeks. After this period of calli cultivation, their growth was stopped as a result of cell damage.

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The effects of the glutathione redox system on DNA binding activity of nuclear DNA topoisomerase I

--Subota, IY, Katyshev, AI, Tarasenko, VI, Senghenko, LP, Kobzev, VF, Konstantinov, YM

Redox regulation of DNA binding activity of transcription factors involving modification of conservative cysteine residues in the protein molecule is considered to be one of the key mechanisms of gene expression control (Martin et al., J. Biol. Chem. 271:25617-25623, 1996; Akamatsu et al., J. Biol. Chem. 272:14497-14500, 1997). We have previously shown that DNA topoisomerase I (topo I) from carrot mitochondria, and probably the enzymes from mitochondria of other plant species, can be regarded as redox sensitive enzymes. Existence of evolutionary conservative cysteine residues which could potentially form disulfide bridges in functionally important domains of carrot protein was demonstrated (Konstantinov et al., Proc. Russian Acad. Sci. 377:263-265, 2001). Hence, mitochondrial topo I is a very plausible candidate for involvement in the mechanism of redox control of expression in the organelles. Recent data showing influence of redox conditions on DNA binding of maize mitochondrial topo I (MNL 77:37-38, 2003) also point to the possibility of complex participation of the enzyme in gene expression regulation. It is still unclear if the demonstrated redox dependence of DNA binding activity of mitochondrial topo I could be a common characteristic of plant enzymes from different cellular locations. In principle, orchestrated redox modulation of nuclear and mitochondrial topo I activities could be one of the ways to coordinate expression of mitochondrially located proteins encoded in the nucleus and mitochondria. To verify the hypothesis about the existence of a mechanism of redox control of nuclear topo I activity we have studied the influence of such physiological redox agents as glutathione on the DNA binding activity of nuclear topo I.

The nuclei were isolated from 4-day-old etiolated maize seedlings of hybrid VIR42 MV as described by Mascarenhas et al. (Methods in Enzymology 31:558-565, 1974) with some modifications. The method of topo I isolation was the same as described earlier (MNL 74:33, 2000) with the additional step of purification on non-denaturing PAGE. EMSA was carried out as described (Ikeda and Gray, Mol. Cell. Biol. 19:8113-8122, 1999) with minor modifications. Assay employed double stranded DNA probes containing *p53* and *atp9* gene promoter fragments. Oxidized and reduced glutathione were used in the experiments in 5 mM concentration.

Figure 1 shows the influence of two forms of glutathione on nuclear topo I DNA binding activity towards the probe containing a fragment of the *p53* gene promoter sequence. The presence of oxidized glutathione (GSSG) caused a significant increase in DNA binding (Fig.1, I-III). However, addition of the reduced form of glutathione (GSH) did not result in any detectable effect.

Surprisingly, the effects of the glutathione redox system on DNA binding activity of nuclear topo I were quite different when the oligonucleotide containing core sequence of the *atp9* gene promoter was used as a DNA probe (Fig.2, I-VI). A supplementary protein-DNA complex appeared upon addition of GSH into retardation medium (Fig. 2, VI). In our previous report (MNL 77:37-38, 2003), we showed the same phenomenon of stimulation of DNA binding activity of mitochondrial topo I in the presence of GSH. It should be noted that when using the *atp9* probe, the addition of GSSG, on the contrary, caused an inhibitory effect on DNA binding activity of nuclear topo I, consisting of the complete loss of slower migrating retardation complexes (Fig. 2, IV-VI).

Consequently, such a universal physiological redox modulator as glutathione has a pronounced influence on DNA binding activity of DNA topoisomerase I of nuclear origin. In our opinion, the data received speak well for the existence of a redox regulation mechanism involving not only modulation of the activity of transcriptional factors, but also a topo I redox modulation mechanism based on modification of cysteine residues in the protein molecule of nuclear and mitochondrial DNA topoi-

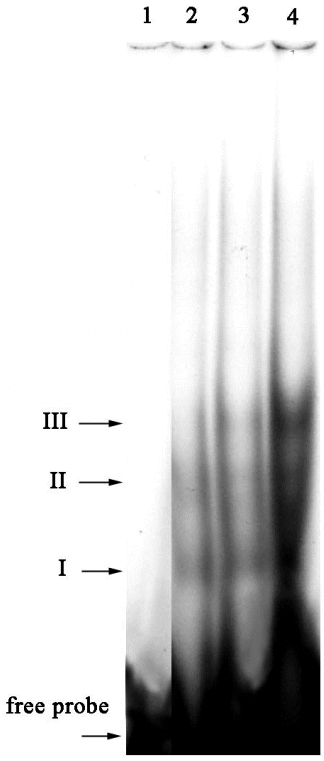

Figure 1. The effect of reduced and oxidized glutathione on DNA binding activity of nuclear topoisomerase I with the *p53* gene promoter fragment. 1) control (-topo I), 2) topo I, 3) topo I + GSH, 4) topo I + GSSG.

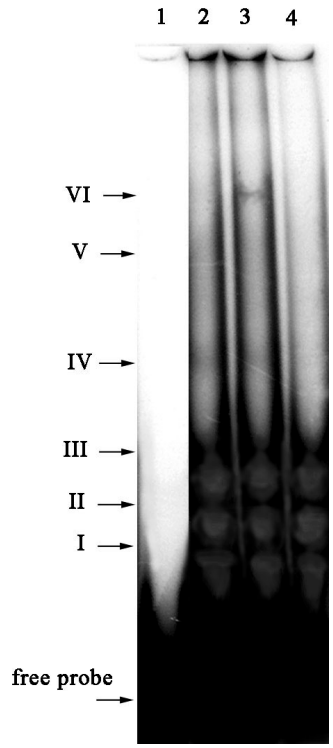

Figure 2. The effect of reduced and oxidized glutathione on DNA binding activity of nuclear topoisomerase I with the *atp9* gene promoter fragment. 1) control (-topo I), 2) topo I, 3) topo I + GSH, 4) topo I + GSSG.

somerase. Future experiments on site-directed mutagenesis will allow discovery of the possible role of individual cysteine residues in redox modulation of DNA binding and catalytic activity of topo I. Additional experiments are needed to elucidate the cause of the demonstrated differences in effects of the glutathione redox system on nuclear topo I DNA binding activity when using different DNA substrates.

Financial support from the Russian Foundation for Basic Research (Grant No. 01-04-48162) is acknowledged.

Isolation of a redox dependent mitochondrial DNA binding protein which binds specifically to the *coxI* promoter

--Tarasenko, VI, Subota, IY, Kobzev, VF, Konstantinov, YM

Although the plant mitochondrial promoter sequences are relatively well characterized, at the current time little is known about trans-factors controlling expression of the mitochondrial genome. There is only one report on isolation of mitochondrial DNA binding protein from a monocotyledon plant (wheat). This protein binds to the coxII gene upstream region and seems to be involved in transcription initiation (Ikeda and Gray, Mol. Cell. Biol. 19:8113-8122, 1999). In yeast and animal mitochondria, activity of a single transcription factor is sufficient for accurate initiation of transcription of all genes (Jang and Jaehning, J. Biol. Chem. 266:22671–22677, 1991; Fisher et al., Mol. Cell. Biol. 8:3496–3504, 1988). The authors proposed that isolated wheat protein also plays a role of such an universal transcription factor. On the other hand, the existence of multiple gene-specific mitochondrial transcription factors has been proposed on the basis of specific dependence of *coxII* expression in *Zea perennis* on the nuclear MCT locus (Newton et al., EMBO J. 14:585, 1995). Nothing is known about physiological conditions that can influence the activity of mitochondrial DNA binding proteins.

Both our research group (Konstantinov et al., Biochem. Mol. Biol. Intern. 36:319-326, 1995) and other investigators (Wilson et al., Eur J. Biochem. 242:81-85, 1996) have demonstrated the existence of redox regulation of transcription in plant mitochondria. An activation of RNA synthesis in organello in maize under oxidizing conditions and its suppression under reducing conditions was detected. Now we are trying to identify protein factors that can mediate this redox regulation. Previously, we have shown redox dependence of mitochondrial DNA topoisomerase I activity (Konstantinov and Tarasenko, MNL 73:39-40, 1999). In this letter, we report partial purification of a DNA binding protein that binds specifically to the *coxI* promoter region and also possesses properties of a redox dependent protein.

The mitochondria were isolated from 4-day-old etiolated seedlings of hybrid VIR42 MV and lysed by high salt and Triton X-100. The supernatant was applied on a DEAE-Toyopearl chromatography column. EMSA was carried out as described by Kagoshima et al. (J. Biol. Chem. 271:33074-33082, 1996) with minor modifications. We used a PCR-generated 230 bp EMSA probe containing the upstream region of the *coxI* gene (C1). This region includes two sites of transcription initiation (-326 and -231 relative to ATG). Another probe used (C3) contained a similar region of the *coxIII* gene (210 bp) which also comprises two initiation sites (-360 and -320 relative to ATG). The reason for use of such probes was the possibility that some additional motifs located upstream or downstream of the core promoter sequence may be important for specific binding.

Some fractions eluted from DEAE-Toyopearl contained activity which formed two complexes in EMSA when C1 was used as a probe (Fig. 1). These retardation complexes were stable in high salt and in the presence of non-specific competitor DNA, demonstrating the specificity of the interaction. Surprisingly, we have not detected any DNA binding activity when the C3 probe was used instead of C1. In another assay, the presence of unlabeled C3 probe, as well as a 34 bp oligonucleotide contained the *atp9* promoter core sequence in 50-fold excess in retardation reaction of DEAE fractions with labeled C1, did not affect the efficiency of DNA binding. These data strongly suggest that isolated protein demonstrates specific binding to the promoter region of the *coxI* gene, but doesn't possess an ability to bind to all mitochondrial promoter sequences. Based on this fact, it is possible to speculate that in maize mitochondria, unlike the situation existing in animal and yeast organelles, there is more than one transcriptional factor, and transcriptional regulation might be gene-specific.

In further experiments we tested the influence of reduced (GSH) and oxidized (GSSG) glutathione on DNA binding activity of the protein (Fig. 2). Some decrease of DNA binding activity was detected in the presence of GSH. Addition of GSSG caused significant changes in the DNA binding pattern. A faster migrating band (Fig. 2, III) emerged, whereas two bands detected in the absence of the agent (Fig. 2, I and II) disappeared. Moreover, intensity of this newly formed retardation complex was higher than that of two complexes detected when GSSG was omitted. Such DNA binding modulation could be explained by changes in the protein-DNA complex conformation caused by reduction/oxidation of cysteine residues in the protein molecule.

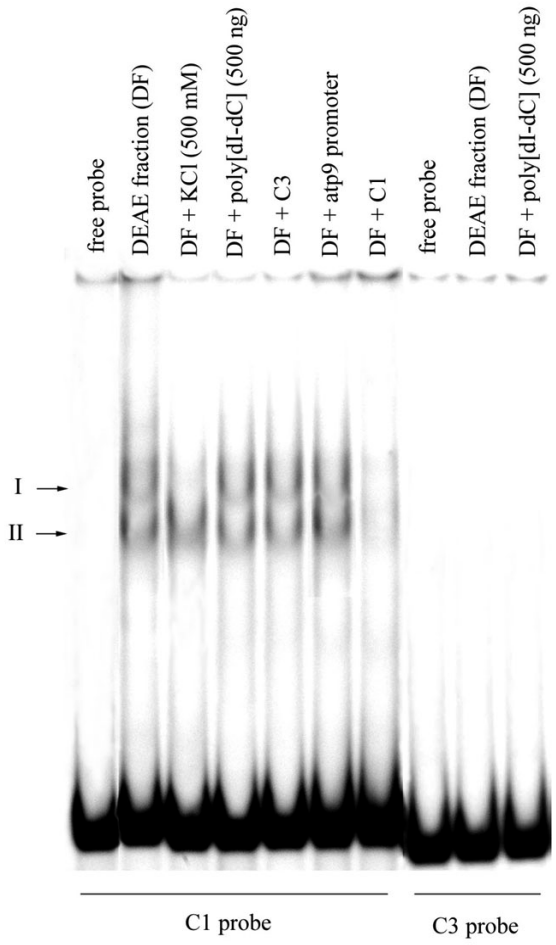

Figure 1. DNA binding activity exhibited by protein fractions isolated by chromatography on DEAE-Toyopearl (DF, eluted with 0.4 M KCl). Competition study using non-specific DNA competitor and probes containing mitochondrial promoter sequences. C1 – upstream region of the *coxI* gene (230 bp); C3 – upstream region of the *coxIII* gene (210 bp); *atp9* – core promoter of the *atp9* gene (34 bp, -288 relative to ATG). Unlabeled competitors were present in the reactions in a 50-fold excess relative to the labeled probe. Arrows indicate the positions of two DNA-protein complexes and the free radiolabeled C1 and C3 probes.

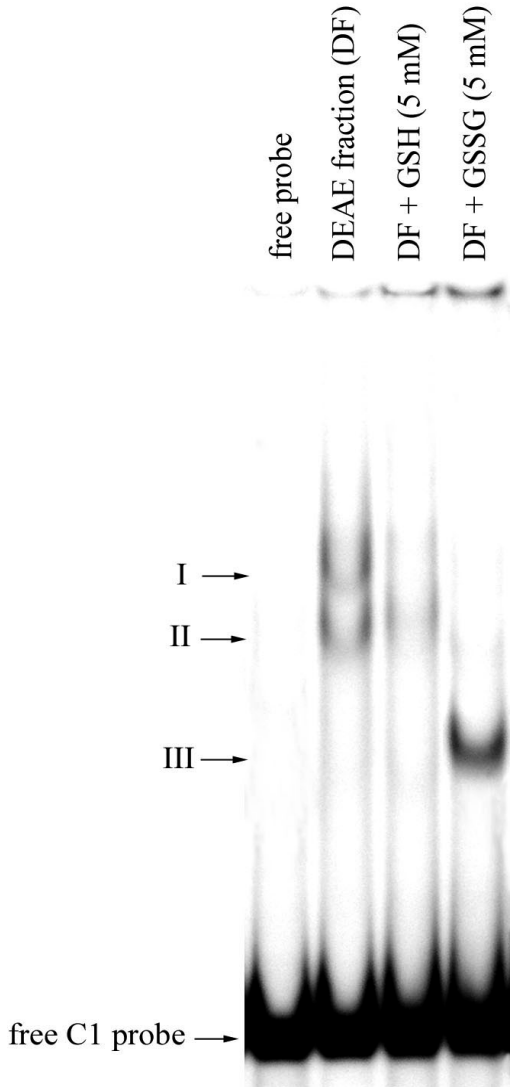

Figure 2. The effect of reduced and oxidized glutathione on DNA binding activity recovered in DEAE-Toyopearl protein fractions. Arrows indicate the positions of three DNA-protein complexes and the free radiolabeled C1 probe.

In conclusion, we have shown that reduced and oxidized forms of glutathione modulate the activity of the mitochondrial DNA binding protein that specifically recognizes the *coxI* promoter region. Such modulation potentially could play an important role in regulation of expression of mitochondrially encoded proteins, especially of respiratory chain components, in response to changes in the redox state of the chain. Additional investigations are needed to verify this hypothesis. Further puri-

fication and biochemical characterization of the protein is underway in our laboratory.

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AFLP analysis of the DNA in subline A344-CP5

--Baschalkhanov, SI, Mashnenkov, AS, Kobzev, VF, Katyshev, AI, Konstantinov, YM

The maize subline A344-CP5 causes heterosis in the hybrid including the initial line A344 (Mashnenkov, NGCNL, 1982, 56: 92-98). The aim of the present work was to study genetic polymorphism in an A344-CP5 subline, an A344 inbred line and their hybrid using amplified fragment length polymorphism (AFLP) markers.

DNA was extracted from individual *Z. mays* shoots (70-100 mg of fresh tissue) using the modified CTAB method (Doyle and Doyle, Phytochem. Bull. 19:11-15, 1987). The DNA preparations were treated with RNAse E (Sigma) and extracted with chloroform. After ethanol precipitation, the DNA pellets were resuspended in 50 µl of 10 mM Tris-HCl, pH 7.5. Then DNA was quantified by agarose gel electrophoresis.

AFLP reactions were carried out according to Vos et al. (Nucleic Acids Res. 23:4407, 1995) with minor modifications.

The template DNA was digested with *Eco*RI and *Tru*9I (an isoshizomer of *Mse*I). Then *Eco*RI and *Mse*I adapters were ligated onto corresponding sticky ends. First, PCR was performed with primers complementary to the ligated adapters with no selective nucleotides. Selective PCR was carried out with primers posessing three selective bases each. Eight combinations of selective primers were used: M-CTG + E-ACG, M-CTG + E-AGC, M-CTG + E-ACC, M-CTG + E-AGG, M-CAG + E-ACG, M-CAG + E-AGC, M-CAG + E-ACC, M-CAG + E-AGG where M and E are sequences of the *Mse*I and *Eco*RI adapters respectively. The selective PCR products were radiolabeled by direct incorporation of α-^{32}P-dATP (20 µCi per reaction). The resulting AFLP fragments were resolved on a sequencing gel and visualized by autoradiography.

It is known that 150 bands are sufficient for reliable estimates of genetic similarity (Pejic et al., Theor. Appl. Genet. 97:1248-1255, 1998). In our AFLP analysis we detected 258 bands (Fig. 1). This allowed us to reliably distinguish the subline A344-CP5 from inbred line A344 and their hybrid. In the case of the hybrid, a significant number of unique bands were revealed. Such results could be explained, presumably, by substantial DNA rearrangements in the hybrid genome. Thus, apart from the existence of sufficient genetic similarity of two parent inbred lines, crossing between them is accompanied by serious genomic DNA reorganization. We suggest that such DNA reorganization possibly is one of the important factors for the realization of the heterotic effect.

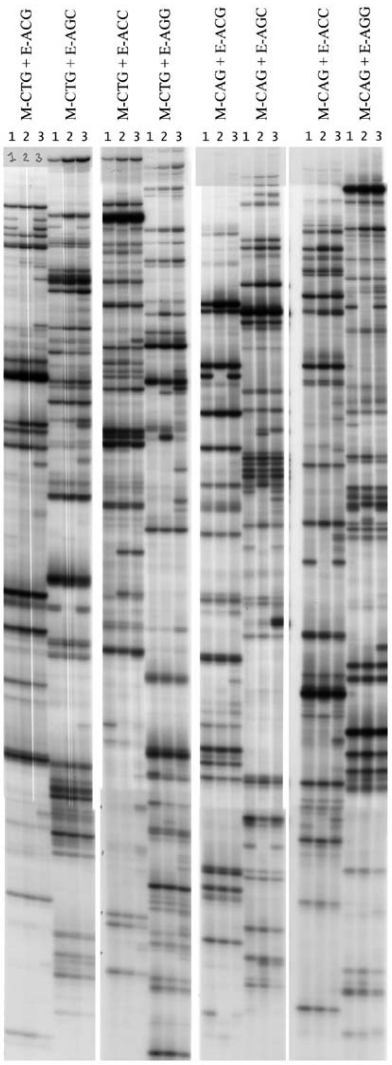

Figure 1.

ITHACA, NEW YORK
Cornell University

Identification of photographs from the Barbara McClintock papers on the National Library of Medicine website

--Kass, LB

While writing and conducting research for an intellectual biography of Barbara McClintock (Kass 1999, 2000, Kass and Murphy 2003, Kass and Provine 1997, 1999), I have located documents that clear up some confusion surrounding dates and identities for some photographs in the Barbara McClintock Papers at the American Philosophical Society Library, Philadelphia, Pennsylvania. The National Library of Medicine (NLM) websites have posted photographs from that collection:
<http://profiles.nlm.nih.gov/LL/Views/AlphaChron/alpha/10013/>
< http://profiles.nlm.nih.gov/LL/Views/Exhibit/other/visuals.html >

In a number of instances dates and identities for these photographs listed on the NLM website are imprecise or missing. Some photos depict McClintock's Cornell University colleagues during the Golden Age of Corn Genetics, 1928-1935 (Rhoades

1984, Kass and Bonneuil in press). Along with these are photos of McClintock after she left Cornell and accepted an appointment at the University of Missouri in 1936 (Kass unpublished MS). There are also a few photos taken with her siblings.

I have been fortunate to find documents at other institutions and archives that aid in more accurately dating the photographs listed on the NLM website. Additionally, colleagues who knew the persons who are misidentified or unidentified in the published photographs assisted with more accurate identifications. Below I share (in bold) additional details (sources in parentheses) for several published photos that currently appear on the NLM website and in Comfort (2001). More detailed information about sources for dating these photographs will be made available upon request.

The Barbara McClintock Papers
Photographic prints (Alphabetical Listing on NLM websites):

<http://profiles.nlm.nih.gov/LL/B/B/P/N/_/llbbpn.jpg> [Barbara McClintock] [ca. 1920s]
Comfort (2001) approximately dates this close-up photo as McClintock in the 1920s.
Barbara McClintock at the University of Missouri, February 1938 (Western Historical Manuscript Collections, Columbia Missouri; and George P. Rédei, University of Missouri, Columbia, Missouri; also see *Stadler Genetics Symposia* 1976: 3, where a photo is published of Stadler's Genetics Group at Missouri in 1938, including McClintock [as she appears in this close-up web-photo], W. Ralph Singleton [who provided the group photograph to George Rédei], and Ernest Sears [who identified members in the group]).

<http://profiles.nlm.nih.gov/LL/B/B/P/Y/_/llbbpy.jpg> [Barbara McClintock wearing Groucho Marx glasses] [ca. 1985]
Comfort (2001) dates this photo as McClintock ca. 1986.
Barbara McClintock, Cold Spring Harbor, October 14, 1983, photo by Susan G. Cooper. (Susan G. Cooper, Kass interview, March 18, 1997, at Cold Spring Harbor Laboratory).

<http://profiles.nlm.nih.gov/LL/B/B/Q/R/_/llbbqr.jpg> [Cornell University] [1929 or 1930]
Faculty, staff, students and collaborators in the Plant Breeding Department, Cornell University [spring 1929 or fall 1930]. Back row center, left to right: L. F. Randolph, Rollins Adams Emerson and George Beadle. Front Row: Marcus Rhoades 5th from right. (R. P. Murphy and Plant Breeding Department Archives, Cornell University).

<http://profiles.nlm.nih.gov/LL/B/B/P/J/_/llbbpj.jpg> [Group from Cornell University, including: Charles Burnham, Marcus Rhoades, Ralph Emerson, Almiro Blumenschien and George Beadle] (1928)
Corn geneticists in the Plant Breeding Department, Cornell University, 1929. Top Row, left to right: Rollins Adams Emerson, unidentified, Marcus M. Rhoades, Harold M. Perry, L. F. Randolph, Charles Russell Burnham. Bottom Row, left to right: Hsien W. Li, George Beadle, Ernest Dorsey (W. B. Provine's McClintock File, and Plant Breeding Department Archives, Cornell University; Henry M. Munger, and R. P. Murphy, Cornell University). note: Rollins Adams Emerson was misidentified as Ralph Emerson in this photograph. Burnham was an NRC Fellow at Cornell from July to December 1929, therefore the photo is more accurately dated 1929 (Cornell University Archives). Almiro Blumenschein [note spelling] was born in 1931, therefore, he could not possibly be in this 1929 photograph (North Carolina State University Archives).

<http://profiles.nlm.nih.gov/LL/B/B/Q/J/_/llbbqj.jpg> [McClintock with an unidentified man at the Stadler Symposium, University of Missouri-Columbia] (April 1978)
The man is George P. Rédei, Department of Agronomy, University of Missouri. The venue is Ed Coe's laboratory, 210 Curtis Hall, University of Missouri. Photo taken during Stadler Genetics Symposium, 1978 (Richard Whalen, South Dakota State University; Ed Coe, George P. Rédei, University of Missouri).

<http://profiles.nlm.nih.gov/LL/B/B/Q/D/_/llbbqd.jpg> [Portrait of McClintock Siblings, circa 1957] [ca. 1957]
Comfort (2001) approximately dates this photo of McClintock in the 1950's.
Portrait of McClintock Siblings, May 18, 1947 (California Institute of Technology Archives).

I would be most grateful to Cooperators who can assist with placing names on unidentified persons or dating photos. Please contact me by email at lbk7@cornell.edu, phone 607-255-4876, or by post at Department of Plant Biology, 228 Plant Science Building, Cornell University, Ithaca NY 14853-5908.

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JOHNSTON, IOWA
Pioneer Hi-Bred International

Use of anthocyanin pigmentation as a diagnostic tool to visualize drought stress in maize

--Habben, J

Visualization of drought stress in maize typically involves observing symptoms such as reduced plant height, as well as leaf rolling and leaf senescence. While these are useful traits to assess the differential response of germplasm to drought, they do not offer a tissue or cellular level understanding of the spatial and temporal response of plants to water deficits. In an effort to enhance our understanding of this facet of drought, we created transgenic maize that expresses a construct composed of a drought responsive promoter (*rab17*) fused to a chimeric transcription factor (*CRC*) whose product induces anthocyanin biosynthesis.

In maize, it is well known that *rab17* responds to increased levels of ABA (Busk, PK et al., Plant J. 11:1285-1295, 1997) and that ABA accumulates in drought stressed plants (Zeevaart, JAD and Creelman, RA, Annu. Rev. Plant Physiol. Plant Mol. Biol. 39:439-473, 1988). It is also well known that anthocyanin synthesis requires regulatory genes from both the *c1* and *r* families (reviewed in Mol, J et al., Trends Plant Sci. 3:212-217, 1998). At Pioneer, a chimeric *C1* and *R*-paralog fusion gene (*CRC*) has been created and its product demonstrated to induce synthesis of anthocyanins in maize (Bruce, W et al., Plant Cell 12:65-80, 2000). Thus, our intent was to combine these two well-characterized genetic elements to produce transgenic maize that would synthesize anthocyanins in tissues which perceive water deficits.

Ninety events containing the *rab17:CRC* construct were generated in a genetic background that does not normally produce anthocyanins. As an initial test of construct efficacy, intact auricles of several T0 events were incubated in either water (-ABA) or 20 µM ABA (+ABA). Within 24 hours, it was readily apparent that anthocyanin synthesis was induced in auricles exposed to ABA but not in control auricles (Fig. 1A): thus demonstrating the inducibility of the construct by this phytohormone. Approximately one week prior to anthesis, a subset of events was exposed to a water deficit (WD) while a second set of (clonal) events remained well watered (WW). Within 48 hours, anthocyanin pigmentation was observed in drought stressed leaves (Fig. 1B), stalks (Fig. 1C), and tassels (image not shown). In mature T1 kernels, pigmentation was primarily observed in the embryo and aleurone (Fig. 1D). Although embryos contained a high concentration of anthocyanin pigment, this concentration was not so great that it caused embryo lethality in subsequent generations.

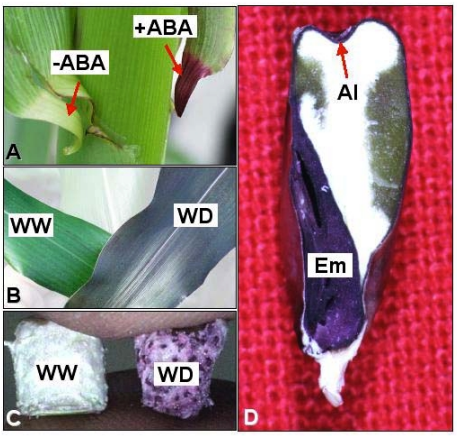

Figure 1

Figure 1. Images of anthocyanin deposition in plant parts of *rab17:CRC* events. A. Auricles of a T0, V5 plant incubated in either water (-ABA) or 20 µM ABA (+ABA). B. Ear leaves of T0 plants exposed to well watered (WW) or water deficit (WD) conditions. C. Cross section of stalks from T0 plants exposed to well watered (WW) or water deficit (WD) conditions. D. Longitudinal section of a mature T1 kernel; Em = embryo, Al = aleurone.

We further substantiated the utility of these transgenic events as a drought visualization tool by looking for anthocyanin deposition in organs that have previously been demonstrated to have elevated ABA levels under water deficit conditions. First, it has been shown that in developing ears exposed to drought stress there is a significant increase in ABA levels in apical kernels of drought stressed plants relative to their well watered controls (Wang, Z et al., Ann. Bot. 90:623-630, 2002). Fig. 2A shows that kernels from ear tips of *rab17:CRC* plants exposed to a drought stress have an increase in pigmentation relative to their well watered counterparts. Second, it is well established that ABA mediates stomatal closure in response to drought stress in plants (reviewed in Schroeder, J *et al*., Annu. Rev. Plant Physiol. Plant Mol. Biol. 52:627-658, 2001). We performed epidermal peels on transgenic leaves exposed to drought and well watered conditions and determined that in contrast to stomata exposed to full irrigation (image not shown), those exposed to water deficits accumulate anthocyanins (albeit in the subsidiary cells; Fig. 2B).

We also discovered anthocyanin pigmentation in cell types that, to the best of our knowledge, have not previously been noted for their accumulation of ABA. For example, Fig. 2C shows a very specific deposition of pigment adjacent to the silk abscission zone in the proximal end of post-pollinated silks. This pigmentation sector occurs independently of whether plants are well watered or drought stressed. Based on these observations, we hypothesize that the formation of the abscission zone initi-

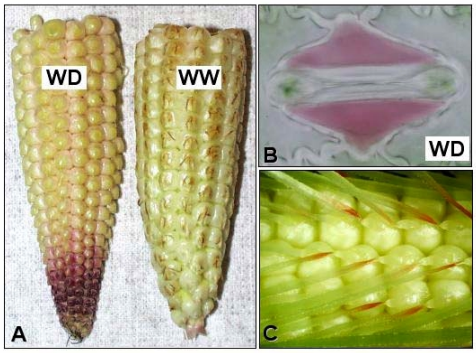

Figure 2

Figure 2. Images of anthocyanin deposition in T1 *rab17:CRC* events. A. Ear tips from 11 DAP plants exposed to water deficit (WD) or well watered (WW) conditions. B. Stomata from an epidermal peel of an event exposed to water deficit (WD) conditions. C. Proximal ends of post pollinated silks (3 DAP).

ates the dehydration of the silk, which in turn triggers an increase in ABA levels and subsequent activation of anthocyanin biosynthesis.

For decades, maize geneticists have exploited anthocyanin pigmentation as an in planta colorimetric assay to visualize various molecular mechanisms, thereby obviating the need for complex analytical techniques. As demonstrated here, by placing anthocyanin synthesis under the control of a well-characterized drought stress promoter, we can create a diagnostic tool that should have similar applicability to visualize the molecular mechanisms of the drought response in maize.

ms23 and *ms35* are alleles of the same gene that maps to chromosome 8

--Trimnell, MR, Fox, TW, Albertsen, MC

As we reported in MNL 76:37-38, *ms23* and *ms35* were found to be allelic, although their reported map locations indicated they were not on the same chromosome (see MNL 62:71 and MNL 69:126-128). In our 1999 Hawaii winter nursery, a family segregating for the *ms35-6011* mutation was sampled for SSR mapping (described in MNL 76:38) to confirm the mutant's reported map location on 9L (MNL 69:126-128). Results of our SSR mapping showed linkage with two markers, both of which map to chromosome 8. To verify this linkage, the segregating *ms35* family was replanted in a subsequent nursery and resampled for mapping. Again, the same two chromosome 8 markers showed linkage. Below is the composite data for these two mapping experiments.

Marker	Recomb. Alleles	Mutant Plants	% Recombination
umc1075	19	44	21.6
bnlg1194	29	38	26.3

These data show that, although not tightly linked to the SSR markers in this population, the *ms35* mutation maps on chromosome 8, not the long arm of chromosome 9 as previously reported. To confirm this map position, a family segregating for the *ms23* mutant (allelic to *ms35-6011*) also was sampled for SSR mapping. As shown below, the same two chromosome 8 markers with linkage to the *ms35-6011* allele also were linked to the *ms23* allele. The differences in % recombination result from a smaller sample size and from the two alleles being in different mapping populations. The key point is that two independent populations show linkage of a male sterile allele with the same two SSR markers on chromosome 8.

Marker	Recomb. Alleles	Mutant Plants	% Recombination
umc1075	3	23	6.5
bnlg1194	4	24	8.3

Independently, but concurrently with the *ms23* and *ms35-6011* mapping studies, we mapped a previously unmapped male-sterile mutant, *ms*-6059* (described by the late Dr. Earl Patterson in MNL 69:126-128), to chromosome 8. It showed linkage with the *umc1075* SSR marker (% Recombination = 16.7). This mutant was testcrossed with *ms23* in our 2001 Hawaii winter nursery. The progeny were grown in our 2002 Johnston nursery, and the mutants were found to be allelic (data shown below). In each testcross, the female parent was homozygous recessive for the indicated allele, and the male parent was heterozygous.

Female	Male	Progeny		X^2(1:1, P>0.050=3.84)
ms-6059*	*ms23*	37 Fertiles	40 Steriles	0.12
ms23	*ms*-6059*	21 Fertiles	12 Steriles	2.45

We conclude that *ms*-6059* is allelic to *ms23*, and that its proper allelic designation is *ms23-6059*. We also conclude that results of the allelism tests with *ms23*, *ms23-6059*, and *ms35-6011*, coupled with the mapping of these mutations in three different mapping populations that show linkage to the same markers on the same chromosome, indicate that *ms23*, *ms23-6059*, and *ms35-6011* are alleles of the same gene that maps to chromosome 8. We propose that subsequent gene lists show *ms23* = *ms35*. The following male-sterile alleles currently represent the same gene family: *ms23*; *ms23-6059*; *ms35-6011*; *ms35-6018*; *ms35-6027*; *ms35-6031*.

The question remains as to how two different sets of A-B interchanges could have led to an error in the chromosome placement of two different alleles. One answer would involve analyzing these interchange stocks with SSR's to determine the actual chromosome composition of the interchanged chromosomes, if these stocks were still available. This would reveal whether there had been additional rearrangements that might have involved chromosome 8 and that previously had been undetected. If this were true, then perhaps the *ms23* alleles are located in such a region. Otherwise, this points out the caution that should be applied to mapping data obtained without independent corroboration.

New chromosome 4 male-sterile mutant: *ms52*

--Trimnell, MR, Fox, TW, Albertsen, MC

During the 1991 Hawaii winter nursery season, George Peverly pointed out to MCA some male-sterile plants segregating in a backcrossing line. We planted remnant seed of this line in our 1992 Johnston, IA, nursery and designated it *ms*-PM91A*. The planted rows segregated for the male-sterile phenotype, and male-sterile plants were crossed with A632 and B73. These F1 ears were self-pollinated and then grown in our 1994 Johnston nursery. We planted several selfed ears in our nursery, and they segregated as follows:

Genotype	# Fertile Plts	# Sterile Plts	X^2(3:1, P>0.050=3.84)
ms-PM91A*/A632)1	13	3	0.33
ms-PM91A*/A632)2	13	4	0.02
ms-PM91A*/B73)1	17	2	2.12
ms-PM91A*/B73)2	18	6	0.00

In the 2000 Johnston, IA, nursery, we planted an F2 segregating family to determine the chromosome arm map location of the mutant as part of our standard procedure in working with previously unmapped male-sterile mutants. First, we determine the chromosome arm location, and then we conduct the appropriate allele testcrosses. Leaf punches were taken from 24 male-sterile plants and from 24 male-fertile plants for DNA isolation. Ninety-six SSR markers were used to genotype these samples. Shown below are four chromosome 4 markers that show linkage to the mutation.

Marker	Recomb. Alleles	Mutant Plts	% Recombination (based on mutant segregants)
bnlg1265	1	22	2.2
bnlg1755	2	19	5.3
bnlg1189	6	18	16.7
phi438301	10	24	20.8

The *ms*-PM91A* mutation is close to the centromere, and it appears to be on the short arm of chromosome 4.

After receiving the mapping data, we testcrossed *ms*-PM91A* with the mapped recessive male-sterile mutants found on chromosome 4 (*ms30*), as well as the unmapped male-sterile mutants (*ms27*, *ms31*). The resultant progeny were grown in our 2002 Johnston, IA, nursery. At least 40 plants were observed for each testcross; all testcross progeny were found to be fertile, indicating that *ms*-PM91A* was not allelic to *ms27*, *ms30*, or *ms31*. Our new designation for male-sterile mutant *ms*-PM91A* is *ms52-PM91A*.

New male-sterile mutant alleles

--Trimnell, MR, Fox, TW, Albertsen, MC

As part of our ongoing effort to map new male-sterile loci in maize, we have identified 16 new alleles of known male-sterile genes. Eight of the mutants (*ms*-6026*, *ms*-6048*, *ms*-6052*, *ms*-6054*, *ms*-6057*, *ms*-6060*, *ms*-6061*, *ms*-6065*) were identified by the late Dr. Earl Patterson (MNL 69:126-128) and given to us in the early 1990's. Two of the mutants (*ms*-MG04*, *ms*-MG07*) were identified by Major Goodman in the course of his studies on cytoplasmic diversity among exotic maize lines and given to MCA in the early 1980's. Mutant *ms*-SB370* was identified by MRT and MCA in a Mutator population originally constructed by Dr. Steve Briggs when he was with Pioneer. The mutant designated *ms*-DR87B* was found segregating in the *Ac*-tester line, *r-sc:m3*. Mutants *ms*-PR92* and *ms*-NP92* were identified by Evan Elsing and MCA among segregating breeding material in our 1992 Hawaii nurseries. Included also are two mutants from chemical mutagenesis. Mutant *ms*-HC8-4-4-1* originated from an inbred treated with sodium azide by Ken Hibberd, a fellow graduate student with MCA at the University of Minnesota in the late 1970's. Mutant *ms*-G39-4* originated from an inbred treated with EMS by MCA in 1983. Segregations of these male-sterile mutations as F2 families are shown below.

Genotype	Year Identified	Year Grown	# Fertile Plants	# Sterile Plants	X^2(3:1)[+]
ms-6026*/A632)1	--	1997	10	5	0.56
ms-6026*/B73)1	--	1997	15	1	3.00
ms-6048*/A632)1	--	1997	13	5	0.07
ms-6048*/B73)1	--	1995	13	1	2.38
ms-6052*/A632)1	--	1997	12	4	0.00
ms-6052*/B73)1	--	1997	9	7	3.00
ms-6054*/A632)1	--	1998	9	2	0.27
ms-6054*/B73)1	--	1998	11	3	0.10
ms-6057*/A632)1	--	1997	16	1	3.31
ms-6057*/B73)1	--	1997	9	5	0.86
ms-6060*/A632)1	--	1997	12	5	0.18
ms-6060*/B73)1	--	1997	11	4	0.02
ms-6061*/A632)1	--	1997	13	1	2.38
ms-6061*/B73)1	--	1997	10	4	0.10
ms-6065*/A632)1	--	1997	9	7	3.00
ms-6065*/B73)1	--	1997	10	6	1.33
ms-MG04*/A632)1	--	1998	9	6	1.80
ms-MG04*/B73)1	--	1998	15	4	0.16
ms-MG07*/A632)1	--	1998	12	3	0.20
ms-MG07*/B73)1	--	1997	12	3	0.20
ms-SB370* F2	1991	1992	26	8	0.04
ms-DR87B*/A632)1	1989	1995	16	1	3.31
ms-DR87B*/B73)2	1989	1993	14	4	0.07
ms-NP92*/A632)1	1992	1995	6	5	2.45
ms-NP92*/B73)1	1992	1995	9	6	1.80
ms-PR92*/A632)1	1992	1995	15	1	3.00
ms-PR92*/B73)1	1992	1995	16	3	0.86
ms-HC8-4-4-1*/A632)1	1978	1997	9	4	0.23
ms-HC8-4-4-1*/B73)1	1978	1997	12	3	0.20
ms-G39-4*/A632)1	1984	1999	18	2	2.40
ms-G39-4*/B73)1	1984	1997	18	6	0.00

[+](3:1, P>0.050=3.84)

Between 1997 and 2003, we planted F2 families in Hawaii and Johnston, IA, to determine the map location of these mutants. As part of our standard procedure in working with previously unknown male-sterile mutants, we first determine the chromosome arm location for a given mutant, and then we conduct the appropriate allele testcrosses. Leaf punches were taken from 24 male-sterile plants and from 24 male-fertile plants in the mapping families for DNA isolation. Ninety-six SSR markers, dispersed throughout the genome, were used to genotype these samples. SSR mapping results are as follows:

Family	Linked Markers	% Recombination[++]	Mutant Map Position

ms-6026*	*phi364545*	10	
ms-6026*	*phi452693*	35	6L
ms-6048*	*bnlg653*	0	5L
ms-6052*	*umc1736*	0	2L
ms-6054*	*umc1736*	4.2	
ms-6054*	*umc2205*	26.1	2L
ms-6057*	*bnlg1129*	6.5	9L
ms-6060*	*phi331888*	16.7	5
ms-6061*	*phi331888*	0	
ms-6061*	*bnlg653*	2.2	5L
ms-6065*	*bnlg1065*	25	
ms-6065*	*bnlg1129*	16.7	9L
ms-MG04*	*phi96100*	25	
ms-MG04*	*bnlg1064*	18.8	
ms-MG04*	*bnlg1396*	31.1	2S
ms-MG07*	*cyp6*	22.5	7
ms-SB370*	*bnlg653*	4.5	5L
ms-DR87B*	*umc1075*	6.8	
ms-DR87B*	*bnlg1194*	0	8S
ms-NP92*	*bnlg1189*	8.3	
ms-NP92*	*bnlg2244*	0	4L
ms-PR92*	*bnlg1056*	21.7	
ms-PR92*	*bnlg1065*	4.3	8L
ms-HC8-4-4-1*	*phi059*	*2.1*	
ms-HC8-4-4-1*	*bnlg1079*	*4.2*	
ms-HC8-4-4-1*	*mgs1*	*8.3*	*10*
ms-G39-4*	*bnlg619*	*10.4*	
ms-G39-4*	*phi236654*	*20*	*9L*

++% Recombination was determined using segregation scores only from mutants.

After receiving the mapping data, we made testcrosses with all of the known recessive male-sterile genes that mapped to the same respective chromosome as these mutants, as well as crossing with the unmapped recessive male-sterile genes (ms27, ms31). The resultant progeny were grown in our 2001, 2002, or 2003 Johnston nursery. The male fertility phenotype of at least 40 plants was observed for most of the testcrosses. The results of reciprocal testcrosses of those mutants that showed allelism with known male steriles are shown below, along with their respective allele designations.

Female	*Male*	*Progeny*	*_2(1:1)*	*Allele Designation*
ms-6026*	*ms50*	*34 Fertiles:27 Steriles*	*0.80*	
ms50	*ms*-6026*	*23 Fertiles:35 Steriles*	*2.48*	*ms50-6026*
ms-6048*	*ms5*	*33 Fertiles:32 Steriles*	*0.02*	
ms5	*ms*-6048*	*44 Fertiles:31 Steriles*	*2.25*	*ms5-6048*
ms-6052*	*ms33*	34 Fertiles:35 Steriles	0.01	
ms33	*ms*-6052*	46 Fertiles:17 Steriles	13.35	
ms-6052*	*ms33-EA89A*	20 Fertiles:20 Steriles	0.00	
ms33-EA89A	*ms*-6052*	22 Fertiles:21 Steriles	0.02	*ms33-6052*
ms-6054*	*ms38*	18 Fertiles:23 Steriles	0.61	
ms38	*ms*-6054*	21 Fertiles:21 Steriles	0.00	*ms38-6054*
ms-6057*	*ms25-YA85A*	33 Fertiles:39 Steriles	0.50	*ms25-6057*
ms13	*ms*-6060*	23 Fertiles:30 Steriles	0.92	*ms13-6060*
ms-6061*	*ms5*	35 Fertiles:28 Steriles	0.78	
ms5	*ms*-6061*	23 Fertiles:33 Steriles	1.79	*ms5-6061*
ms-6065*	*ms25-YA85A*	33 Fertiles:37 Steriles	0.23	
ms25-YA85A	*ms*-6065*	23 Fertiles:25 Steriles	0.08	*ms25-6065*
ms-MG04*	*ms40*	23 Fertiles:24 Steriles	0.02	
ms40	*ms*-MG04*	20 Fertiles:22 Steriles	0.10	*ms40-MG04*
ms-MG07*	*ms7*	30 Fertiles:15 Steriles	5.00	
ms7	*ms*-MG07*	18 Fertiles:23 Steriles	0.61	*ms7-MG07*
ms-SB370*	*ms5*	36 Fertiles:39 Steriles	0.12	
ms5	*ms*-SB370*	18 Fertiles:27 Steriles	1.80	*ms5-SB370*
ms-DR87B*	*ms23*	67 Fertiles:15 Steriles	1.97^{+++}	
ms23	*ms*-DR87B*	48 Fertiles:44 Steriles	0.17	*ms23-DR87B*
ms-NP92*	*ms30*	20 Fertiles:25 Steriles	0.56	*ms30-NP92*
ms-PR92*	*ms8*	32 Fertiles:34 Steriles	0.06	
ms8	*ms8-PR92*	32 Fertiles:28 Steriles	0.27	*ms8-PR92*
ms-HC8-4-4-1*	*ms11*	25 Fertiles:20 Steriles	0.56	
ms11	*ms*-HC8-4-4-1*	25 Fertiles:19 Steriles	0.82	*ms11-HC8-4*
ms-G39-4*	*ms25-YA85A*	5 Fertiles:10 Steriles	1.67	
ms25-YA85A	*ms*-G39-4*	22 Fertiles:20 Steriles	0.10	*ms25-G39-4*

(All females homozygous for mutation unless noted otherwise; all males heterozygous for mutation)

$^{+++}$Chi square value calculated for a 3:1 expected segregation ratio as female was heterozygous for the mutation in this testcross only.

LLAVALLOL, ARGENTINA
Universidad Nacional de la Plata y Cigen (CONICET-UNLP-CIC)

Quality maize hybrids developed in Argentina: chemical evaluation of endosperm

--Corcuera, VR, Bernatené, EA, Naranjo, CA

Although less than 20% of Argentina´s maize production is used in manufacturing, it is convenient to attend to the industrial requirements during the breeding process in improving protein quality and content, as well as starch quality, to obtain new more highly valued commodities. Kernel quality homogeneity of the new hybrids is also a very important concern for maize breeders. Since 1990, inbreds were obtained by Schull´s method from eleven foundational populations of *waxy*, *opaque2* and normal

endosperm maizes. Materials were conducted ear-per-row during the first inbreeding generations and later using balanced composites. Several single-crosses amongst developed inbreds were made. Oil content (%), protein content (%), starch content (%), as well as density, were measured at the lab through a non-destructive assay using a NIR device model Isotec 1227. Measurements were carried out on samples of F2 kernels taken from the 106 experimental single crosses and flint maize testers grown in two field trials at Llavallol and Castelar during two consecutive growing seasons (2001/02 and 2002/03). These precommercial hybrids differed from the rest by the nature of their endosperm, and based on this, they can be classified as waxy, high quality protein, and waxy and high quality protein (double recessive). Each sample consisted of 80 grams of whole grain from each location. Data obtained for each hybrid in both locations were averaged. Starches from a flint hybrid (3193), two double recessive hybrids (3165, 3152) and a waxy hybrid (3187) were isolated and purified in treatments with $NaHSO_3$ 0.005M, successive washes with NaCl, ethanol-water, and washed and dried (Salmoral et al., 2000). Molecular fractioning was performed according to the difference of solubility method in water-buthanol. Quantification and spectrophotometric profiles (λ_{max}) were done using iodine-potassium iodide reagent in a saturated solution of $CaCl_2$ using a Shimatzu spectrophotometer. Seventy-two hybrids (26 waxy, 14 high quality protein, 29 double recessive and 3 flint hybrids) showed ≥ 5% oil content, ranging from 3.52 to 6.41%, with an average of 5.24%, if all of the hybrids evaluated are considered. Protein content ranged from 8.2 to 12.6%, average: 10.25%. Twenty-six hybrids had ≥ 10.5% protein content (16 waxy, 3 high quality protein, 5 double recessive and 2 flint hybrids).

Starch content varied from 68.6 to 74.1%, average: 71.6%. Ninety-eight hybrids had ≥ 70% starch content (50 waxy, 40 double recessive, 6 high quality protein and 2 flint hybrids).
Density values fluctuated from 1.169 to 1.313 with an average of 1.264. The lowest value was found in a double recessive hybrid, and the highest in a waxy endosperm hybrid. When the different types of hybrids were considered individually, it was found that ***waxy hybrids*** have an average oil content of 5.03 ± 0.43, an average protein content of 10.00 ± 0.97, an average starch content of 71.44 ± 1.10 and an average density of 1.28 ± 0.21. The average oil content for ***high quality protein hybrids*** is 5.53 ± 0.43, the average protein content is 10.03 ± 0.56, the average starch content is 71.63 ± 0.70 and the average density is 1.24 ± 0.18. The average oil content observed for ***double recessive hybrids*** is 5.39 ± 0.37, the average protein content is 9.83 ± 0.69, the average starch content is 71.91± 1.22 and the average density is 1.25 ± 0.29.

Pairwise correlations amongst the endosperm chemical components considered above were calculated. No correlation was observed between oil and protein (r = 0.12), but statistically significant ones were found for oil-starch (r = -0.24, p ≥ 0.05), starch-protein (r = -0.71, p ≥ 0.05), oil-density (r = -0.35, p ≥ 0.01) and starch-density (r = -0.53, p ≥ 0.05) (see Table1).

Regression equations were also calculated:

oil (x) - protein (y) $y' = 9.0905 + 0.17880 * oil$
oil (x) - starch (y) $y' = 75.809 + (-0.8684) * oil$
oil (x) - density (y) $y' = 1.3093 + (-0.0057) * oil$
starch (x) - protein (y) $y' = 57.595 + (-0.6664) * starch$
starch (x) - density (y) $y' = 1.9193 + (-0.0089) * starch$

Table 1. Simple correlations between kernel chemical quality parameters.

Correlation	General	Waxy	Opaque2	Double recessive
starch-protein	- 0.71 **	- 0.76 **	0.36	- 0.68 **
starch-oil	- 0.24 *	- 0.34 *	0.31	- 0.37*
starch-density	- 0.53 **	- 0.46 **	0.49	0.40 *
protein-oil	0.12	0.08	0.45	0.18
protein-density	0.19	0.27 *	0.31	0.03
oil-density	- 0.35 *	- 0.12	0.48	- 0.15

Starch, oil and protein content of the endosperm were also measured in the parent inbreds, although they are not published here. In general terms, when the values found for the hybrids are compared to those of the inbreds, it can be stated that oil and starch content do not vary too much amongst them, but protein content in F2 kernels is always markedly lower than that found in the respective inbreds. So, the "dilution effect" observed for protein content in F2´s employed as animal or human feed could be overcome by developing high protein content inbreds to be used as the female parent in the crosses. Otherwise, the results obtained from the preliminary studies on starches showed that the flint hybrid 3193 had a 75% amylopectin and 8.04% amylose content. An 89% amylopectin content and an 11% amylose content was found in the waxy hybrid 3187, and an 80-81% amylopectin content and a 19-20% amylose content were detected in the double recessive hybrids 3165 and 3152. The higher amylopectin content is also confirmed through the high ramification degree (A: $\lambda_{max}/\lambda_{max}$ spectrum shoulder) that varied from 1.20 to 1.35.

Finally, it can be said that many hybrids tested in the field trials show an exceptional kernel quality based on their oil, protein and starch content, which makes them more than suitable to be used for animal and human feed or as industrial staples.

Kernel quality, evolutive cycle length and yield of waxy, high protein quality and double recessive inbreds developed in Argentina

--Corcuera, VR, Bernatené, EA, Naranjo, CA

During 1990, a maize quality breeding programme was initiated at the Instituto Fitotecnico de Santa Catalina aimed at obtaining high quality protein, waxy and double recessive hybrids capable of being used for human and animal nutrition, as well as new staples for several industries. Amongst other traits, evolutive cycle length was measured in the foundational materials, backcrosses, inbreds and different types of hybrids derived from them, through Heat Unit Requirements (HUR) and number of days to mid silking (R1 stage according to Hanway scale). In this paper, we present briefly the results obtained for evolutive cycle length to R1, yield and chemical nature of the endosperm of fifteen inbreds developed at the Institute and evaluated in field trials at Llavallol (35º S, 58º W) during three consecutive growing seasons (2000/1, 2001/2 and 2002/3). The materials were arranged in a 3 replicate complete randomised block design. Each plot consisted of a single row of 5.5 m, sown at a density of 71,500 plants/ha. The chemical composition and density of the grains was measured using a NIR device model Isotec 1227. Heat unit requirements in each inbred were mesasured on the basis of the individual plant, according to the USWB method corrected by 2.0 C as follows:

$$USWB_{2,0} = \{t_{min} + [30 - (t_{max} - 30) \times 2.0]\} / 2 - 10$$

t_{min}: minimum temperature (10 C)
t_{max}: maximum temperature

Number of days to mid-silking were also evaluated for each genotype. Yield was measured considering kernel weight/ear (15% humidity), number of ears per plant and number of plants/ha.

Most of the waxy maize used as foundationals in 1990 were precocious (418-518 C for silking), whilst the foundational high quality protein maize showed a long evolutive cycle (780-870 C for silking). Although inbreeding tends to lengthen evolutive cycle, it was possible to obtain through selection amongst and within families, short or medium evolutive cycle, high quality protein (472-676 C, 47 to 64 days) and waxy maize inbreds (505-681 C, 48 to 65 days). On the other hand, whether double recessive inbreds (high quality protein and modified starch) showed short or medium evolutive cycle length, some denoted a long one (490-715 C, 51 to 68 days), (see Table 1 and Table 2).

Table 1. Evolutive cycle of inbred lines evaluated in Llavallol during 3 consecutive years.

		2000/1		2001/2		2002/3	
Genotype	Endosperm	HUR (ºC)	Days	HUR (ºC)	Days	HUR (ºC)	Days
3088	opaque2	559.0	58.0	593.9	51.0	656.2	64.0
3139a	opaque2	535.0	56.0	596.0	51.0	586.4	57.0
3142a	opaque2	506.0	53.0	624.1	60.0	596.3	59.0
3141a	opaque2	545.0	56.0	571.4	47.0	676.0	64.0
3138a	opaque2	472.0	50.0	577.5	48.0	564.0	56.0
3022c	waxy	577.0	59.0	664.7	60.0	522.1	52.0
3024a	waxy	538.0	56.0	580.4	48.0	535.7	53.0
3074c	waxy	513.0	53.0	602.9	56.0	595.6	58.0
3072a	waxy	551.0	57.0	628.6	57.0	616.4	60.0
3020a	waxy	505.0	53.0	613.9	55.0	680.9	65.0
3096b	wx/o2	567.0	58.0	619.3	55.0	586.4	57.0
3096c	wx/o2	490.0	51.0	611.8	55.0	680.9	65.0
3135a	wx/o2	588.0	60.0	646.4	58.0	605.5	59.0
3136b	wx/o2	609.0	62.0	699.3	62.0	714.6	68.0
3137a	wx/o2	586.0	60.0	680.1	60.0	594.1	66.0

Table 2. Evolutive cycle and yield (on average) evaluated in Llavallol during 3 consecutive years.

Genotype	Endosperm	HUR (ºC)	Days	Yield (kg/ha)
3088	opaque2	603.0	58	2502
3139	opaque2	572.3	55	5076
3142	opaque2	575.3	57	5863
3141	opaque2	597.3	56	6506
3138	opaque2	537.6	51	4862
3022c	waxy	588.0	57	3932
3024a	waxy	551.2	52	1430
3074c	waxy	570.5	56	4218
3072	waxy	598.6	58	4075
3020a	waxy	599.9	58	6077
3096b	wx/o2	590.9	57	7722
3096c	wx/o2	594.2	57	7720
3135	wx/o2	613.3	59	3932
3136b	wx/o2	674.3	64	5148
3137	wx/o2	620.1	62	4301

In general terms, selection by precocity associated to quality breeding was successful, and unpublished data from a single hybrid field trial evaluated in Llavallol during the growing season 2002/3 showed that the evolutive cycle length of these was dominated by the most precocious parent of the cross.

The yield of the inbreds studied ranges from 1,430 kg/ha to 7,722 kg/ha, with an average of 4,891 kg/ha. If the genotypes are grouped according to the nature of their endosperm, the average yields are 4,962 kg/ha (2,502 to 6,506 kg/ha) for high quality protein inbreds, 3,946 kg/ha (1,430 to 6,077 kg/ha) for waxy inbreds and 5,765 kg/ha (3,932 to 7,722 kg/ha) for double recessive inbreds (see Table 2).

Protein, starch, oil content and density were measured in ten inbreds and data are shown in Table 3. Five inbreds had ≥5% oil content, 7 had ≥10.5% protein content and 5 had ≥70% starch. The highest density values correspond to opaque2 or double recessive genotypes, whilst lower values were observed in waxy inbreds, which is surely related to the chemical compositions of the starch (see Table 3). The correlation analysis showed that only oil content is related to yield (r = 0.49) whilst the correlation index starch-protein was r = -0.59. Considering the yield and the protein content of the inbreds studied, it can be stated that protein yield fluctuates from 149 to 873 kg of protein / ha.

Table 3. Chemical composition of the endosperm of ten inbreds evaluated in Llavallol during 3 consecutive years.

Genotype	Endosperm	Oil (%)	Protein (%)	Starch (%)	Density
3088	opaque2	5.39	12.4	67.5	1.309
3022	waxy	5.24	10.9	70.7	1.25
3024a	waxy	5.06	10.4	72.1	1.289
3074c	waxy	5.54	9.5	72.2	1.277
3072	waxy	3.64	10	70.9	1.267
3020a	waxy	4.55	12.7	69.6	1.241
3096b	wx/o2	4.21	11.4	69.1	1.276
3096c	wx/o2	4.51	10.8	69.3	1.264
3136b	wx/o2	4.35	10.5	70.2	1.323
3137	wx/o2	5.19	8.6	69.9	1.283

In brief, precocious, high yielding and high kernel quality inbreds could be developed using Schull´s method and conducting the materials ear-per-row during the first inbreeding generations, and later using balanced composites selecting within and amongst families.

Maize quality breeding: plant and prolificity traits of single-crosses obtained and tested in Argentina

--Bernatené, EA, Corcuera, VR, Naranjo, CA

A maize quality breeding programme has been conducted at the Instituto Fitotecnico de Santa Catalina since 1990. Several inbreds have been developed and used to obtain single-cross hybrids, which have been tested in multi-location field trials since 2001/02. These hybrids can be classified by the nature of their endosperm into 3 categories: 1) waxy, 2) high quality protein and 3) waxy and high quality protein (double recessive) hybrids. Waxy hybrids have a naturally modified starch (high amylopectin) that constitutes an excellent staple to be transformed for technological objectives and it is also partially useful for feed, and the textile, paper and adhesive industries. In turn, high quality protein hybrids differ from the rest by the high content of lysine and tryptophan that enhances their biological value. As a result, they are excellent for use in pig and poultry feed as a partial solution to starvation in humans.

Several single-cross hybrids and their parent inbreds were tested during the growing season of 2002/03. Of these, fifteen hybrids (five of each category) and their parents were studied using some plant and prolificity traits. The trial was sown in a 3-replicate completely randomized block design at a density of 71,500 plants/ha on October 22 at Llavallol (35º S - 58º W), province of Buenos Aires (Argentina).

Measurements for each trait were taken on the basis of the individual plant. Each plot consisted of a row of 5.5 m. Three

plant traits were considered: a) plant height (PH), b) ear insertion height (EIH) and c) number of leaves above the uppermost ear (LUE) as well as one prolificity trait: number of ears/plant (EP). Routine statistical analysis was done according to the classical methodology proposed by Falconer. Degree of heterosis was calculated on the basis of the highest parent value (HP-heterosis).

If data obtained for each descriptor in each group of hybrids are averaged, the high quality protein hybrids are 204.3 cm tall, waxy hybrids are 210.8 cm tall and the double recessive 239.6 cm tall. Waxy hybrids are more prolific (1.5 ears/plant) than high quality protein hybrids (1.1 ears/plant) but similar to double recessive hybrids (1.4 ears/plant). Detailed information is shown in Tables 1 to 3.

Table 1. Degree of heterosis in modified starch hybrids. Data taken at Llavallol (Province of Buenos Aires) during the growing season 2002/3.

		Evaluated Trait			
Genotype	Generation	PH	EIH	LUE	EP
	P1	185.74	56.57	4.73	2.00
	P2	160.84	49.53	4.22	1.91
3168	MP	173.29	53.05	4.48	1.96
	F1 (2002/3)	218.10	83.70	4.50	1.20
	HP-Heterosis %	17.42	47.95	-4.86	-40.00
	P1	138.25	42.72	4.42	1.61
	P2	160.84	49.53	4.22	1.91
3170	MP	149.55	46.13	4.32	1.76
	F1 (2002/3)	203.9	77.1	4.6	1.3
	HP-Heterosis %	26.77	55.66	4.07	-31.93
	P1	156.66	40.13	5.28	1.77
	P2	185.74	56.57	4.73	2.00
3172	MP	171.20	48.35	5.00	1.89
	F1 (2002/3)	192.2	78.1	4.5	1.90
	HP-Heterosis %	3.47	38.05	-14.77	-5.00
	P1	152.14	51.32	4.05	1.91
	P2	167.66	47.09	5.22	1.71
3175	MP	159.90	49.21	4.64	1.81
	F1 (2002/3)	208.70	81.60	4.50	1.60
	HP-Heterosis %	24.47	59.00	-13.79	-16.23
	P1	152.14	51.32	4.05	1.91
	P2	207.48	63.69	6.68	2.04
3176	MP	179.81	57.51	5.37	1.98
	F1 (2002/3)	231.30	101.30	5.00	1.70
	HP-Heterosis %	11.48	59.05	-25.14	-16.66

P1: Female parent
P2: Male parent
MP: Mid-parent value
F1: Hybrid value
HP-Heterosis: High-parent heterosis %

PH: Plant height (cm)
EIH: Ear insertion height (cm)
LUE: Leaves above the uppermost ear
EP: Ears per plant

Degree of heterosis is also presented in the attached tables. All hybrids evaluated showed lower LUE and EP than their respective parents. On the other hand, hybrid vigor is strongly expressed, as expected, through PH and EIH.

In summary, it can be said that the hybrids studied showed regular size and adequate ear insertion height that will facilitate their cultivation. Waxy and double recessive hybrids studied differed because of their prolificity, which is higher than in current maizes commonly grown in Argentina.

Table 2. Degree of heterosis in high quality protein hybrids. Data taken at Llavallol (Province of Buenos Aires) during the growing season 2002/3.

		Evaluated Trait			
Genotype	Generation	PH	EIH	LUE	EP
	P1	172.05	39.32	6.53	2.32
	P2	156.80	55.38	5.39	1.40
3145	MP	164.43	47.35	5.96	1.86
	F1 (2002/3)	216.30	86.40	5.70	1.00
	HP-Heterosis %	25.71	56.01	-12.71	-56.89
	P1	172.05	39.32	6.53	2.32
	P2	169.60	51.19	5.87	1.70
3146	MP	170.83	45.26	6.20	2.01
	F1 (2002/3)	237.60	99.20	5.70	1.50
	HP-Heterosis %	38.09	93.78	-12.71	-35.34
	P1	169.60	51.19	5.87	1.70
	P2	151.07	41.35	5.67	1.58
3147	MP	160.34	46.27	5.77	1.64
	F1 (2002/3)	190.90	78.30	5.10	1.10
	HP-Heterosis %	12.55	52.95	-13.11	-35.29
	P1	151.07	41.35	5.67	1.58
	P2	169.60	51.90	5.87	1.70
3150	MP	160.34	46.62	5.77	1.64
	F1 (2002/3)	186.30	75.80	5.50	1.10
	HP-Heterosis %	9.84	46.05	-6.30	-35.29
	P1	151.07	41.35	5.67	1.58
	P2	156.80	55.38	5.39	1.40
3151	MP	153.94	48.37	5.53	1.49
	F1 (2002/3)	190.40	61.80	5.10	1.00
	HP-Heterosis %	21.42	11.59	-10.05	-36.70

P1: Female parent
P2: Male parent
MP: Mid-parent value
F1: Hybrid value
HP-Heterosis: High-parent heterosis %

PH: Plant height (cm)
EIH: Ear insertion height (cm)
LUE: Leaves above the uppermost ear
EP: Ears per plant

Table 3. Degree of heterosis in high quality protein and modified starch (double recessive) hybrids. Data taken at Llavallol (Province of Buenos Aires) during the growing season 2002/3.

		Evaluated Trait			
Genotype	Generation	PH	EIH	LUE	EP
	P1	169.46	52.89	5.44	1.99
	P2	141.57	48.82	4.17	1.04
3158	MP	155.52	50.86	4.81	1.52
	F1 (2002/3)	205.50	85.10	5.00	1.70
	HP-Heterosis %	21.26	60.89	-8.08	-14.57
	P1	188.73	73.50	5.40	1.59
	P2	169.46	52.89	5.44	1.99
3160	MP	179.10	63.20	5.42	1.79
	F1 (2002/3)	259.60	105.80	6.20	1.50
	HP-Heterosis %	37.55	43.94	13.97	-24.62
	P1	170.47	69.89	5.52	1.92
	P2	168.64	48.70	5.82	1.30
3161	MP	169.55	59.30	5.67	1.61
	F1 (2002/3)	268.90	106.70	5.70	1.20
	HP-Heterosis %	57.74	52.66	-2.06	-37.50
	P1	172.38	44.55	4.76	1.19
	P2	169.46	52.89	5.44	1.99
3163	MP	170.92	48.72	5.10	1.59
	F1 (2002/3)	233.90	102.00	5.20	1.50
	HP-Heterosis %	35.68	92.85	-4.41	-24.62
	P1	168.04	48.70	5.82	1.30
	P2	141.57	48.82	4.17	1.04
3164	MP	154.81	48.76	5.00	1.17
	F1 (2002/3)	230.30	89.70	4.90	1.10
	HP-Heterosis %	37.05	83.73	-15.80	-15.38

P1: Female parent
P2: Male parent
MP: Mid-parent value
F1: Hybrid value
HP-Heterosis: High-parent heterosis %

PH: Plant height (cm)
EIH: Ear insertion height (cm)
LUE: Leaves above the uppermost ear
EP: Ears per plant

Behaviour of maize inbreds and single-crosses to spontaneous infections of *Puccinia sorghi* Schw.

--Sandoval, MC, Corcuera, VR, Bernatené, EA, Naranjo, CA

During 1990, at the Instituto Fitotécnico de Santa Catalina, a maize quality breeding plan aimed at obtaining high nutritional and high added value commercial maize hybrids was initiated. Studying the behaviour of the inbreds and single crosses in relation to the most important diseases constitutes a main objective of the program. During the growing season of 2002/03, 32 inbreds and 60 hybrids sown in a three-replicate randomized block design field trial at Llavallol at a density of 71,500 plantas/ha were studied for their behaviour to spontaneous infection of *Puccinia sorghi* Schw. (Figure 1), the etiological agent responsible for "common leaf rust". The maize genotypes studied may be classified by the nature of their endosperm as: a) waxy, b) high quality protein and c) waxy and high quality protein (double recessive). In maize regions with temperate winters like Argentina, the whole cycle of the disease may be verified. In our country, severity levels for common rust are near 40%.

Figure 1. Detail of uredosporic pustules of *P. sorghi*, developed (during the reproductive stage of cultivation) on the leaf of a waxy maize hybrid.

The following parameters were analyzed: 1) Type or degree of infection based on a modified Cobb´s scale

severity%	**Type of Infection**	**Behaviour to *P. sorghi***
less than 5	I	Very resistant
6 to 10	II	Resistant
11 to 25	III	Mid-resistant
26 to 40	IV	Mid-susceptible
41 to 65	V	Susceptible
66 to 100	VI	Very susceptible

and 2) percentage severity index (PSI) according to Wheeler (1969) and calculated as follows: sum of numerical ratings x 100/No of plants scored x Maximum score on scale.

The PSI (%) varied from 0 to 55 within the inbreds analyzed. Nine percent of them (3022c, 3074c and 3072) were very resistant, 28% resistant, 31% moderately resistant, 16% moderately susceptible (3096c, 3141, 3002c, 3074a and 3142), 3% susceptible (3014) and 13% immune (3139 II and 3024). The PSI (%) ranged from 0 to 32 in the hybrids. Eighty-three percent of them were very resistant, 3% resistant (precommercial hybrids 3222** and 3222***), 2% moderately resistant (precommercial hybrid 3181**) and 12% immune (precommercial hybrids 3231, 3191*, 3165, 3164, 3165* and 3215).

Pairwise correlations between PSI and potential yield of the single-crosses evaluated were performed. A negative correlation value (r: -0.265) was found, although it is not statistically significant. The fact that in some genotypes PSI was zero, does not let us assert categorically the existence of immunity to illness, as this can also be due to the presence of some other mechanism of evasion that was not considered; this would be worth evaluating in depth in our next studies. All the same, the behaviour of most of the genotypes under evaluation may be detected according to PSI values obtained and their behaviour to the spontaneous presence of *P. sorghi.*

Genomic affinities between maize and *Zea perennis* using classical and molecular cytogenetics (GISH-FISH)

--Gonzalez, G, Confalonieri, V, Naranjo, CA, Comas, C, Poggio, L

Zea mays ssp. *mays* (maize) is an allotetraploid (2n=20), and its genomic formula according to Naranjo et al. (1990) is AmAmBmBm. *Zea perennis* is an alloautooctoploid (2n=40) with a genomic formula ApApA'pA'p Bp1Bp1Bp2Bp2. These taxa, as well as the rest of the members of the genus *Zea*, are segmental cryptic polyploids, with diploidized behavior and basic number x=5 (Naranjo et al., 1990 Acad. Nac. Cs. Ex. Fís. y Nat., Bs. As. 5:43-53).

The aim of this work was to discover the homology between maize and *Zea perennis*. For this reason, their genomic affinities were analyzed through: a) The meiotic behavior of both parents (maize and *Zea perennis*) and their artificial hybrid (2n=30), through classical cytogenetics. b) The molecular affinities, at the medium and highly repetitive DNA level by genomic in situ hybridization (GISH). c) The physical mapping of the ribosomal sequence 45S (pTa71) using FISH.

The materials used were *Zea perennis* (from the location Piedra Ancha) and *Zea mays* ssp. *mays* (cv. 6482), both grown at the Instituto Fitotécnico Santa Catalina (Llavallol, 35º S – 58º W).

a) Classical cytogenetic studies: In order to know the meiotic behavior in metaphase I, chromosome preparations were made using young anthers from maize, *Zea perennis* and its F1 hybrids, which were dyed with acetic hematoxilin (2%).

This study showed a regular pairing of the chromosomes in maize, and the more frequent meiotic configuration was 10 II. The meiosis of *Zea perennis* showed a configuration of 5 IV + 10 II in most of the cells analyzed. In the F1 hybrid *Zea perennis* x *Zea mays* ssp. *mays* (2n=30), whose proposed genomic formula is AmApAp´Bp1Bp2Bm (Naranjo et al., 1990), the more frequent meiotic configuration was 5 III +5 II + 5 I.

b) GISH studies:

b.1) Metaphase maize chromosomes were hybridized with labeled total genomic DNA from *Zea perennis*. In this experiment, a scattered signal of hybridization was observed all over the chromosomes except for two pairs, the metacentric chromosome pairs I and II (Figure 1), which show weak hybridization signal. This showed that these maize chromosomes have some

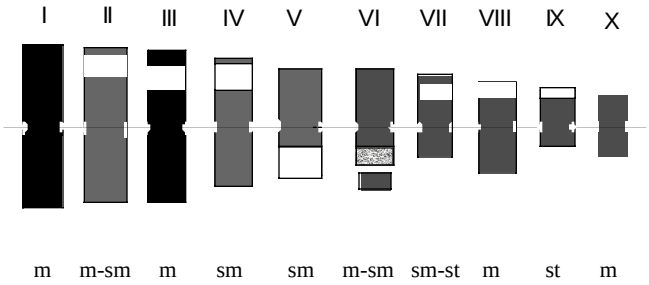

Figure 1. Idiogram of *Zea mays* ssp. *mays* (cv 6482) showing that the chromosome pairs I and II show the lowest molecular homology with *Zea perennis* (in black). White bands are knob locations. The dotted band is the location of the 45S ribosomal sequence (pTa71). m: metacentric. sm: submetacentric. st: subtelocentric.

sequences that are not shared with *Zea perennis* genome. In this experiment, it was also observed that the heterochromatic knobs of maize (DAPI+ bands), composed of a 180 bp sequence highly tandem repeated, do not show a hybridization signal with the genomic DNA of *Zea perennis*.

b.2) A hybridization experiment using labeled genomic DNA of maize and non-labeled genomic DNA as blocking in a proportion of 30:1 was carried out on interphasic and metaphasic maize cells. This experiment showed weak signals of hybridization on all the chromosomes except at the knobs zones (DAPI +) which exhibited an intense hybridization signal. This confirms that *Zea perennis* lacks the 180 pb sequence which is constitutive of maize knobs. This fact had already been observed in previous experiments of Southern-Blot and GISH (Poggio et al., 1999, Genome 42: 993-1000).

b.3) The metaphasic chromosomes of *Zea perennis* were hybridized with the labeled genomic DNA of maize. In this experiment, signals of dispersed hybridization throughout all the chromosomes were observed and none of them displayed a pattern of differential hybridization.

b.4) Meiotic cells (metaphase I) of the artificial F1 hybrid *Zea mays* ssp. *mays* x *Zea perennis* (2n=30) were hybridized with the total genomic DNA of maize. In this case, it was observed that the chromosomes that remained univalents at the meiotic configurations described above belong to maize (since they show a strong hybridization signal), and the bivalent chromosomes correspond to *Zea perennis* (as they show a weak hybridization signal).

These results confirm the origin of the meiotic configurations postulated in previous works.

c) FISH studies: Using the ribosomal sequence 45S of wheat (pTa71) as a probe.

c.1) This probe was hybridized on chromosomes of maize and two signals of hybridization were observed on the short arms of chromosome pair number 6.

c.2) Then, the probe was hybridized on metaphasic chromosomes of *Zea perennis.* In this case, four signals of hybridization were detected.

c.3) Finally, metaphases I of the artificial F1 hybrid *Zea perennis* x *Zea mays* ssp. *mays* (2n=30) were hybridized with the probe pTa71. This experiment showed three signals of hybridization which, in most of the cells, were located on a trivalent; whereas in the rest of the cells, two signals were located on a bivalent and only one signal on a univalent. These results indicate that the 45S ribosomic DNA sequence is present in the genomes that show the greatest degree of pairing during meiosis.

The results obtained by GISH and FISH were summarized through an idiogram of maize which shows those chromosomes with less molecular affinity with *Zea perennis*, the location of knobs and the physical mapping of the 45S ribosomal sequence (Figure 1).

These results allow us to conclude that an important divergence exists between the genomes of *Zea mays* ssp. *mays* and *Zea perennis* because of: a) the lack of hybridization observed on four chromosomes of maize (pairs I ad 2) when hybridized with *Zea perennis* DNA; b) the dispersed hybridization signals of the genomic maize DNA on *Zea perennis* chromosomes; and c) the lack of the 180 pb sequence of knobs in *Zea perennis*.

The classical and molecular cytogenetic studies (GISH-FISH) done on the F1 hybrid *Zea perennis* x *Zea mays* ssp. *mays* allow us to conclude that: a) The univalents come from maize and the bivalents from *Zea perennis* (by GISH); b) the fact that the 45S ribosomal DNA hybridized, in most of the cases, on a trivalent demonstrates that they are formed by two chromosomes of *Zea perennis* and a chromosome of maize. In addition, this result indicates that this sequence is located in the genomes with a greater degree of pairing in the hybrid (named genome A).

All the results indicate that *Zea perennis* (2n=40) is an autoalloctoploid in which one of its genomes would have been more compatible to the ancestor of the present maize. These results and the most frequent meiotic configurations observed in the hybrid, confirm the genomic formulas postulated in previous works (Naranjo et al., 1990; Poggio et al., 2000 Genet. Mol. Biol. 23:1021-1027) (Figure 2).

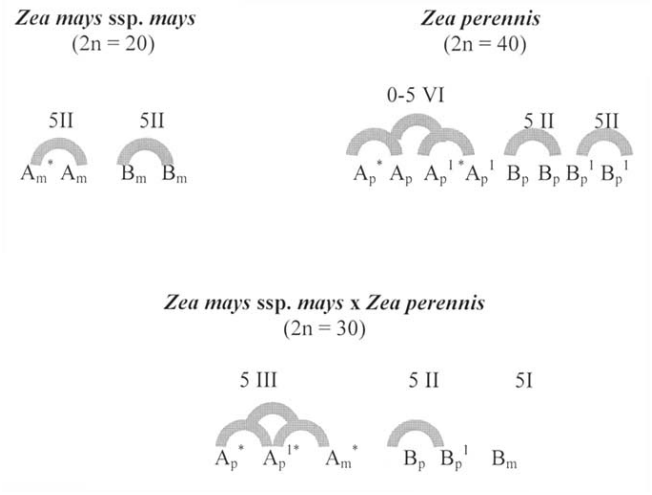

Figure 2. The most frequent meiotic configurations observed in *Zea mays* ssp. *mays*, *Zea perennis* and their F1 artificial hybrid. Arches show the more frequent meiotic associations. Asterisks indicate the genomes that possess the 45S (pTa71) ribosomal sequence. III: trivalent; II: bivalent; I: univalent.

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Germination of Andean Patagonian maize (*Zea mays* ssp. *mays*) inbreds selected for cold tolerance

--Huarte, HR, Jatimliansky, JR, Molina, MC

Maize is one of the most economically important crops in the world. In both hemispheres, the sowing region is located between 47 C and 30 C (temperate and sub-tropical areas). This distribution is due to the tropical origin of the species, which makes it sensitive to low temperatures. Cold susceptibility is one of the greatest constraints that limits maize's spread to new sowing areas for both commercial or subsistence purposes. Brandolini et al. (Brandolini, A., et al., Euphytica 111:33-41, 2000) pointed out that the ability of a determined genotype to germinate, emerge and grow vigorously under low temperatures, constitutes a remarkable characteristic the impact of which extends beyond the cold regions. Effectively, the use of cold tolerant maize genotypes in the most suitable regions for cultivation offers some agronomic advantages such as an earlier sowing date, and its introduction into regions currently considered marginal because of their low temperatures makes maize cultivation more successful.

In the case of maize, 10 C is commonly accepted as a base temperature (Tb) and 25 C as an optimum temperature for growth (To). Nevertheless, Rench (1973) determined 7.2 C to be the base temperature for growing. Several studies reported the existence of genetic variation both for germination and growth under low temperature conditions (Eagles, H and Brooking, IR, Euphytica 30:755-763, 1981). There is also evidence that genetic cold stress tolerance could be found in exotic germplasm such as those obtained from the Andean region (Brooking, IR Maydica 35:35-40, 1990). Due to the facts mentioned above, the objectives of this study were: I) Identifying within the materials evaluated, those able to germinate under low temperatures. II) Elucidating whether the eventual ability to germinate under low temperatures is due to a lower Tb for germination.

Seeds of 12 different genotypes were used. The materials were multiplied in the experimental field of the Instituto Fitotécnico de Santa Catalina (IFSC) (latitude 34° 48 S, longitude 58° 31 W) Argentina, during the growing season of 2000/01. Genotypes labelled SC from 1 to 7 are short cycle materials belonging to populations from NW Argentina Patagonian and the Xth Chilean region. SC 9 is a mid cycle genotype obtained at the IFSC. Two French inbreds (L 10 and L 11) and an American one (B 73) were used as testers.

Five replicates of 20 seeds each were incubated at constant temperatures from 10 C (± 0.5) up to 35 C (± 0.5 C) with intervals of 5 C. Testing lasted 7 days when incubation temperatures were 20 C or higher, and 14 and 28 days when germination occurred at 15 C or 10 C, respectively. Seeds were placed in 9 cm diameter Petri dishes on two discs of filter paper and hydrated with 6 ml of distilled water (equilibrated at the corresponding temperature). Seeds with a clearly visible radicle protrusion were considered germinated.

The following variables were considered:

I) Final germination percentages at 10 and 25 C
II) Germination index proposed by Smith & Millet (Smith, PE and Millet, AH, Proc. Am. Soc. Hort. Sci. 84:480-484, 1964) was calculated as follows.[$\sum$(number of seeds germinated on a given day)(number of days after planting)]/ total number of seeds germinated at the end of the trial
III) The base temperature for germination (50 %) was calculated for each of the materials included in this study.

Data obtained in I and II were analyzed by ANOVA and Tukey´s test (α=0.05).

Significant differences (P<0.05) among the genotypes evaluated were found in germination under stress treatment as well as the control test (25 C). Under stress treatment, Andean inbreds showed germination percentages higher than the testers (P<0.05)(Table 1). The germination index (GI) obtained for SC 1 and SC 3 under stress treatment also exceeded the testers, denoting a higher germination rate (P<0.05)(Table 1).

Table 1. Mean germination values and germination index at 10 C for the inbreds evaluated. S/C, stress/control ratios.

Genotypes	Germination (%)		Germination Index (days)	
	Stress	S/C	Stress	S/C
SC 1	99 a	0.99	8.4 a	3.91
SC 3	78 ab	0.83	8.4 a	3.51
SC 4	93 a	0.93	10.6 ab	3.51
SC 5	15 ef	0.15	15.2 d	4.43
SC 6	93 a	0.96	9.6 ab	6.01
SC 7	79 ab	1.13	10.4 ab	3.68
SC 9	46 cde	0.48	13.5 cd	4.15
SC 12	50 bc	0.92	11.4 bc	3.16
L 10	35 def	0.40	14.6 d	5.50
L 11	2 f	0.03	10.4 ab	5.71
B 73	21def	0.31	11 bc	4.36
Mean	60.45	0.67	11.18	4.34
Tukey(P<0.05)	4.79		4.80	

Stress: Germination test at 10 C for 28 days.
Control: Germination test at 25 C for 7 days
Different letters within the same column designate significant differences (p<0.05) according to Tukey´s test

The germination temperature base for all Andean inbreds was lower than that calculated for the testers and other genotypes of different origin (SC 9 and SC 12)(Table 2). In this sense the values calculated for this parameter in some Andean materials (SC 7, SC 4 and SC 1) are substantially lower than those previously reported for maize (e.g.= 7.2 or 10 C)

Table 2. Temperature base for germination of the genotypes evaluated.

Genotype	Temperature base for germination (°C)	Genotype	Temperature base for germination (°C)
SC 7	4.63	L 10	8.97
SC 1	5.49	SC 12	9.56
SC 4	5.56	B 73	9.68
SC 6	6.5	L 11	10.89
SC 3	8.39	SC 9	11.01
SC 5	8.49		

Germination percentages at 10 C of the Andean-Patagonian inbreds were greater than those from the selected testers employed (Table 1). In addition, one of these inbreds, SC 1, also showed the highest germination index.

SC 1, SC 4 and SC 6 temperature base for germination was 3 C lower than for B73 (Table 2). This fact is in accordance with the higher GI observed in some Andean inbreds exposed to stress treatment. Data presented in this communication show that some Andean-Patagonian inbreds are promising basic materials for developing new cold tolerant hybrids.

Evaluation of seedling growth of maize inbreds (*Zea mays* ssp. *mays*) selected from Andean-Patagonian populations for cold tolerance

--Huarte, HR, Jatimliansky, JR, Molina, MC

Maize is considered a cold susceptible crop with relatively high optimum temperatures for germination, development and dry matter accumulation (Miedema, P., Adv. Agron. 35:93-129, 1982). During the early vegetative stage, plants are susceptible to stress by low temperatures. Actual agronomic practices such as no-tillage and earliest sowing date during the growing season, increase the probability and extent of damage by cold.

The no-tillage system has spread to a great number of cultivation areas, and when this practice is used, it is very common to record soil temperatures lower than those observed under the moldboard plowing system, due to the presence of stubble on the soil surface (Griffith, DR, et al, Agron. Journal 65:321-326, 1973). A lower soil temperature increases the time to seedling emergence and consequently the risk of losses by pathogen attack (Shaw, RH, Corn and Corn Improvement, American Society of Agronomy, Madison Wisconsin, pp. 609-638, 1988), and weed competence is also greater (Olson, RA and Sander, DH, Corn and Corn Improvement, American Society of Agronomy, Agronomy Monograph 18, Madison Wisconsin, pp. 639-686, 1988). Although early sowing dates maximize the duration of the growing stage and promote yield increase (Laurer, JG, et al., Agron. Journal 91:834-839, 1999), they also increase the possibility that germination and early growing stages occur at suboptimal temperatures. The situation described above is enough to show the need for developing maize germplasm tolerant to low temperatures during crop establishment (Lee, EA, et al., Crop Sci. 42:1919-1929, 2002). Brandolini et al. (Brandolini, A, et al., Euphytica 111:33-41, 2000), stated that tolerance to cold temperature stress could be found in exotic germplasm, for example, that of the Andean region. Therefore, the objective of this work was to compare the seedling growth of several germplasms at 10 C (stress temperature) and at 25 C (control temperature).

Six short cycle populations from NW Argentine Patagonia and Xth region of Chile (SC1/ SC7), a mid cycle (SC9) and a long vegetative cycle (SC12) both from temperate origins were used. As controls, three cold tolerant inbreds were used, one introduced from the USA (B73) and the remainder introduced from France (L10 and L11).

Plant growth rates at 10 C (PGR_{10}), at 25 C (PGR_{25}) and their ratio (PGR_{10}/PGR_{25}) were evaluated. The design was a DCA with five replications for both stress (10 C ±1 C) and control treatments (25 C±1 C). Twenty-five seeds placed in nine cm Petri dishes on two discs of filter paper composed each plot. Seeds were hydrated with six ml of distilled water and re-hydrated when necessary. Incubation took place in darkness for twenty-eight days (stress treatment) and seven days (control treatment). Each germinated seed was transferred from the Petri dish to a plastic tray until the end of the experiment. At that time, the epicotyl and the radicle were excised and dried in an oven at 80 C for 24 h, and weighed to obtain the dry matter per plot. Data were subjected to analysis of variance (ANOVA). If significant differences were detected, treatment means were separated using HSD Tukey´s test (α=0.05).

PGR_{10} for SC1 was greater than those of the testers (P<0.05)(Figure 1A). On the other hand, PGR_{25} for B73 was higher than those scored for Andean inbreds (P<0.05)(Figure 1B). PGR_{10}/PGR_{25} ratios for the Andean inbreds showed a good performance. However, none of them exceeded L10 tester ratios (Figure 1C). Results suggest that some Andean-Patagonian inbreds are promising materials for developing new cold tolerant hybrids.

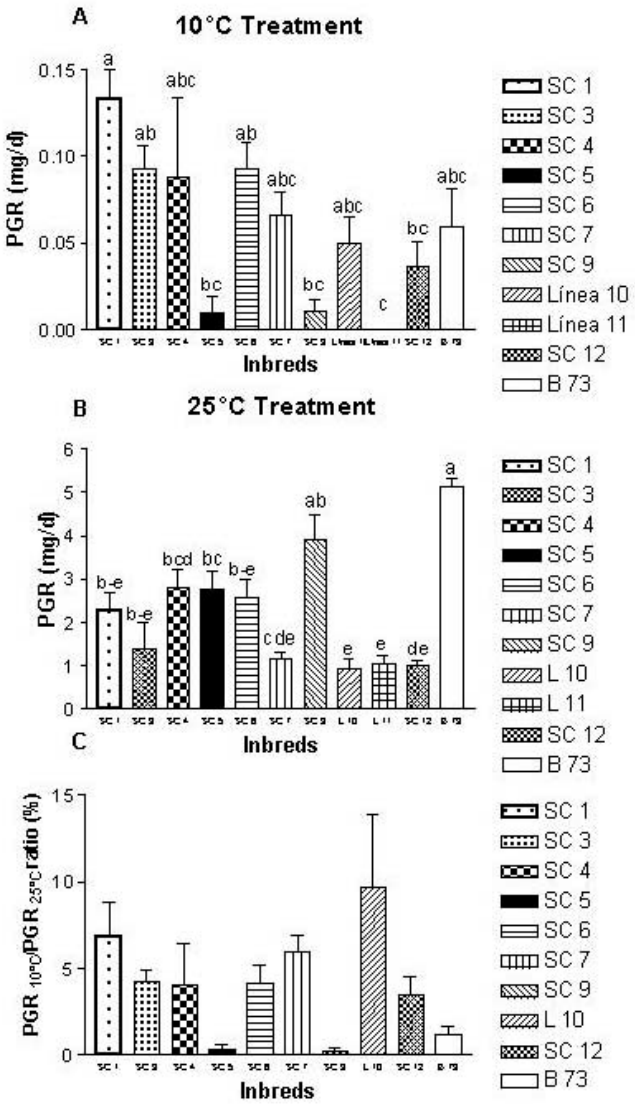

Figure 1. Plant growth rate (mg/d) of inbreds at 10 C treatment (A) and at 25 C treatment (B): Figure 1 C represents the ratio between plant growth rate at 10 C and 25 C.

Confirmatory factor analysis of morphological variables of the ear and kernel in popcorn cultivars

--Burak, R, Broccoli, AM

In order to confirm the relationship between morphological variables of the ear and kernel of popcorn cultivars (*Zea mays* L.), experimental trials were developed in 1999, 2000 and 2001 in Esteban Echeverría and Luján (Buenos Aires, Argentina). These areas belong to a landscape called " Pampa Ondulada", with hills that have the best soils for agriculture, classified as molisols, with 4.5 % organic matter.

Seventeen popcorn cultivars, commercial hybrids and native free pollination varieties, were planted, with a density of 70,000 plants/ha. Field trials were conducted under random complete blocks in a factorial arrangement: 17 treatments x 3 repetitions x 2 locations x 3 years. Experimental units were two rows of 5 m in length, with a distance between rows of 0.70 m.

Harvest maturity was reached at 135 days after sowing. Then kernels were homogenized at 14% moisture, and 306 measures of each variable were further developed.

The variables analyzed were:

Grain yield, kg/experimental unit (YIELD): this variable wsere measured by cutting grains of all kernels of each experimental unit and recording the weight of homogenized grains at 14 % moisture.

Expansion volume, gr/cc (EXVOL): volume of grains expanded in standard conditions (Dofing et al., 1990), obtained using a pop machine with temperature control by thermostat and temperature sensor under the plate. Expansion volume value was obtained by the relationship between the exploded sample volume measured in a test tube of 1,000 ml, and the volume of 30 cc of seed without exploding of the same sample, measured in a test tube of 150 ml.

Grain roundness index (GRI): the higher expansion volume usually attained from samples with medium to small kernels, rounder than the average, has been reported in classic papers, indicating the roundness index GRI, which is the relationship between thickness (KTH), width (KW) and length (KL) of the seed.

$$GRI = KTH / KW + KL$$

Harvest index (HI): calculated by the ratio No. of ears/No. of plants by experimental unit.

Percentage of cob (COB): calculated by the ratio weight of the kernels/weight of the ears.

Kernel volume in cubic centimeters (KXCC): Amount of grain in 1ml of volume.

Kernel per row (KFIL): mean of grain in the row of each experimental unit.

Longitude ear (EARL): measured in cm from the base to the apex of the ear.

Diameter ear (EARD): diameter in mm of the middle part of the ear.

To confirm the relationships among variables, factorial analysis by the principal component method was used, according to this model:

$$y_{ij} = \mu_j + l_{1j} * F_{i1} + l_{2j} * F_{i2} + l_{3j} * F_{i3} + ... + lm_j * F_{im} + \varepsilon_{ij}$$

y_{ij} = Value of the *i*th observation of the *j*th measured variable.
μ_j = Means of the *j*th variable.
F_{ik} = Value of the *i*th observation on the *k*th common factor.
l_{kj} = Regression coefficient of the *k*th common factor for predicting *j*th variable (loading factor).
ε_{ij} = Value of the *i*th observation on the *k*th only factor.
m = Number of common factors.

Statistical analyses were run with SAS/STAT software (Version 8.1), the FACTOR PROCEDURE (See appendix SAS program). The initial factor method: principal component variance with N=3 factor shows that the explained variance by each factor is: factor 1 = 4.37, factor 2 = 1.22 and factor 3 = 0.896, with a total of 6.478285. Variance is retained by two first and second factors, therefore these are the only factors considered in analysis. Kaiser's measure equaled 0.83245, which shows an optimal adaptation of the sample.

Commonalities are common variance estimations between variables and mean proportion of variance, with each variable contributing to the final solution. Commonalities less than 0.5 are considered as lacking sufficient explanation; as shown in Figure 1, with the exception of IH and COB, the rest of the variables are significantly contributing to the final solution.

The load factors matrix rotates; it is multiplied per one orthogonal matrix. Therefore, this allows a better interpretation of common factors, due to the simplification of the structure of these, and re-distributes variance since the former to the latter for achieving a simplest and, theoretically, a more significant pattern of factors. Table 1 shows rotated factors and non-rotating factors. Variances explained by each factor after rotation are similarly arranged (2.918 for factor 1 and 2.664 for factor 2).

Rotation by the varimax method (orthogonal transformation matrix) used in this work, gives a clearer separation of factors. Trends show that there are high factorial loads close to 1 or –1), and another near 0 in each column of the matrix. This means a clear association, negative or positive, between variable and factor; near zero involves an absence of association. To evaluate the contribution of each variable to the final solution, an empiric rule is applied. This suggests that factorial loads plus $\pm$ 0.3 represent a minimal level, $\pm$ 0.4 are important and $\geq \pm$ 0.5 are considered significant. Table 1 shows rotated loads which each one of the variables develops on factors and common variance estimations among these variables (commonality). Variables KXCC, EXVOL and GRI make positive and significant loads on factor 1 (an unobserved variable that includes expansion capacity and its components), while EARL, EARD, YIELD, KFIL and IH develop positive loads on factor 2 (an unobserved variable that represents yield and components). Excepting IH, the rest of the variables associated with factor load negatively on factor 1, resulting in antagonism with factor 1 (Fig. 1). Also, variables IH and COB do not influence factor 1. IH loads positively on factor 2, while COB negatively. IH could be taken as a selection character for improving yield without affecting the expansion capacity of grain.

Table 1. Unrotated and rotated (Method varimax,. orthogonal transformation matrix) factor pattern, (N=2) and commonality estimates.

	Unrotated		Rotated (Method varimax)		
Variables	Factor 1	Factor 2	Factor 1	Factor 2	Communality
KXCC	-0.557	0.598	0.815	0.062	0.668
EXVOL	-0.625	0.480	0.788	-0.067	0.626
GRI	-0.758	0.022	0.573	-0.498	0.576
EARL	0.851	0.013	-0.616	0.586	0.724
EARD	0.820	-0.190	-0.733	0.417	0.716
YIELD	0.801	0.299	-0.385	0.762	0.729
KFIL	0.809	0.158	-0.488	0.665	0.681
IH	0.436	0.488	0.0102	0.655	0.429
COB	-0.454	-0.481	0.0081	-0.662	0.438
Variance explained by each factor	4.368	1.214	2.918	2.664	Total = 5.582

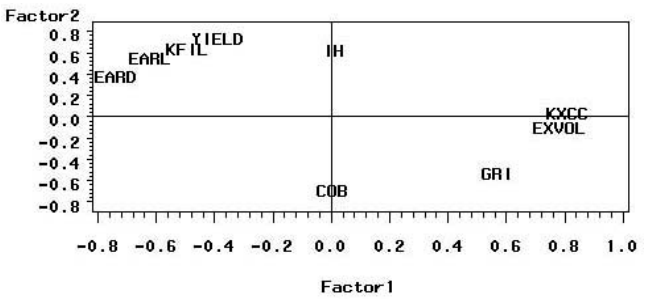

Figure 1. Plot of factor pattern for factor 1 and factor 2.

Appendix: SAS program.

```
 DATA pop;
 TITLE 'popcorn';
 INPUT YIELD KFIL EARL  EARD  EXVOL  GRI   COB   KXCC  IH;
 CARDS;
        3.12  26   12.5   27   33.01  0.39  20.7  5.75  0.98
         .    .    .     .     .      .     .     .     .
         .    .    .     .     .      .     .     .     .
        3.63  35   16.2   31   15.31  0.25  19.9  4.16  0.91
run;
PROC FACTOR DATA=pop res N=3;
     title 'method principal component unrotated' res N=3;
VAR YIELD KFIL EARL EARD EXVOL GRI COB KXCC IH;
run;
PROC FACTOR DATA=pop res N=2
out=sal_pop outstat=estad;
     title 'method principal component unrotated' N=2;
VAR YIELD KFIL EARL EARD EXVOL GRI COB KXCC IH;
run;
data estad;
set estad;
where _type_='PATTERN';
run;
proc transpose data=estad
   out=trans1(rename=(_name_=variable));
run;
data anotar1;
set trans1;
xsys='2'; ysys='2';
text=variable;
x=factor1; y=factor2;
run;
symbol1 v=none;
proc gplot data=trans1 anno=anotar1;title ' ';
plot factor2*factor1=1/vref=0 href=0;
run;
symbol1 v=plus c=red;
proc gplot data=sal_pop;
title ' ';
plot factor2*factor1=1;
run;
PROC FACTOR DATA=pop N=2 rotate=varimax preplot plot
reorder round out=sal_pop outstat=estad;
     title 'metohd principal component' N=2;
VAR YIELD KFIL EARL EARD EXVOL GRI COB KXCC IH;
run;
data estad;
set estad;
where _type_='PATTERN';
run;
proc transpose data=estad
   out=trans1(rename=(_name_=variable));
run;
data anotar1;
set trans1;
xsys='2'; ysys='2';
text=variable;
x=factor1; y=factor2;
run;
symbol1 v=none;
proc gplot data=trans1 anno=anotar1;title ' ';
plot factor2*factor1=1/vref=0 href=0;
run;
symbol1 v=plus c=red;
proc gplot data=sal_pop;
title ' ';
plot factor2*factor1=1;
run;
proc cluster data=sal_pop outtree=tree_pop method=single;
var factor1 factor2;
run;
proc tree data=tree_pop;
*out=out n=2;
copy factor1 factor2;
run;
symbol1 v=circle c=black;
symbol2 v=star c=black;
symbol3 v=plus c=black;
legend1 frame cframe=white cborder=black
position=center value=(justify=center);
axis1 minor=none label=angle=90 rotate=0=;
axis2 minor=none;
proc gplot;
plot factor1*factor2=cluster/frame cframe=white
vaxis=axis1 haxis=axis2 legend=legend1;
*title 'analisys'
*title2 'Single Linkage';
*title3 'cluster with data';
run;
proc cluster data=sal outtree=tree method=ward;
var factor1 factor2;
run;
proc tree out=out n=2;
copy factor1 factor2;
run;
symbol1 v=circle c=black;
symbol2 v=star c=black;
symbol3 v=plus c=black;
legend1 frame cframe=white cborder=black
position=center value=(justify=center);
axis1 minor=none label=(angle=90) rotate=0;
axis2 minor=none;
proc gplot;
plot factor1*factor2=cluster/frame cframe=white
vaxis=axis1 haxis=axis2 legend=legend1;
*title 'analisys'
*title2 'Single Linkage';
*title3 'cluster with data';
run;
```

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On the nature of plant chromosome transition state: gravity, plant vigor and chromosome aberration (an hypothesis)

--Pan, D

Chromosome aberrations were commonly observed in seeds carried into space and later germinated on earth. These aberrations were generally scored in root meristem cells, microspores, and consisted of chromosome fragments and recombinations. Significant chromosome abnormalitites, including fragmented chromosomes and chromosomal bridges, were found in roots of plant seedlings in space. Simultaneously, the space-flown seeds, under appropriate conditions, can genetically alter plant growth and productivity into a vigorous state. Neither the reasons for increases in chromosomal disturbance nor the mechanism for plant vigor observed from the space-flown seeds are known at present time. Generally, it is believed that cosmic radiation is one possibility, since ionizing radiation is known to cause such disturbances; but results obtained from space experiments with the specific aim of answering this question provided no evidence to support the notion that radiation effect is a major factor responsible for chromosome aberration from space flown seeds. Nevertheless, it has been suggested that weightlessness or space flight may affect chromosomal structure not directly but by increasing the likelihood of damage by altering sensitivity to other mutagenic factor(s). Thus, apparently, chromosome aberration induced by space or the vigor of plants derived from space treated seeds cannot simply be interpreted based on the widely accepted genetic concepts. In this note, we wish to submit and put forward an hypothesis that explains the possibility of an unknown chromosome state that can give rise to chromosome aberrations and also can genetically transform plants into a vigorous state in weightless conditions. The hypothesis is described with the following statements:

(1) A chromosome is defined as a single physical identity.

(2) Gravity factor means either gravity force or gravity mass (based on String theory).
(3) In this hypothesis, the transition between two states of chromosome structure is under influence only by gravity factor. Other physical factors, such as the effect of cosmic rays or HZE particles, are considered separately.

Let us first consider that the chromosome structure can exist in two different states, symbolized as $Chr_e \longleftrightarrow Chr_s$ and are assumed to be reversible. Chr_e : Chromosome structure on earth. Chr_s : Chromosome structure in space. The transition of chromosome structure in space will vary according to the altitude during the seeds travel in space. Therefore, the symbol of chromosome structure in space can be written as $Chr_{s(ro\text{----}>rn)(Ts)}$. where r is the altitude of space and varies from 0 to n length of altitude; and Ts is Transition State. For example, currently most of the experimental plant seeds are traveling in space about 250 to 450 miles above the earth. The equation of the transition of chromosome structure from the Chr_e state to the Chr_s state under the influence of the gravity factor while traveling in space can be written as:

$$Chr_e \longleftrightarrow Chr_{s(r0 \text{---}> rn)(Ts)} \qquad (1)$$

$$Chr_{s(r0 \text{---}>rn)(Ts)} / Chr_e \approx P$$

P: is a Transition Constant.

The equation (1) demonstrates that gravity factor governs the equilibrium between two states of the chromosome structure. Therefore, P: (the transition constant) is variable as a function of the gravity factor. According to the present hypothesis, there are two possibilities for the appearance of chromosome aberration from space treated seeds. The first possibility is that the chromosome aberration appeared directly from the transition state of the chromosome structure itself ($Chr_{s(r0 \text{-----}> rn)(Ts)}$) without the effect of other physical factors such as cosmic rays or HZE particles. It could also be that the vigor of plant growth and development of space treated seeds comes from the transition state of the chromosome structure. The second possibility for the chromosome aberration is the result of the combining effect of other physical factors and weightlessness. This kind of alteration of chromosome structure as the result of influence of space conditions can be written as:

$$Chr_e \longleftrightarrow Chr_{s(r0\text{- -->} rn)\,(Ts)} \longrightarrow Chr_{s(r0 \text{---}> rn\,)\,(Cry)}$$

$Chr_{s(r0 \text{--->}rn)\,(cry)}$ is designated as the cryptic state of $Chr_{s(r0\text{--->}rn)\,(Ts)}$, and it is an irreversible transition process. All negative effects observed from the growth and development of space treated seeds were the result of the phenotype of chromosome aberration derived from the cryptic state of the chromosome. A complete and detailed report of this hypothesis will be published elsewhere. Due to the current advancement of the isolating technique of corn chromosomes, such as the B chromosome, the present hypothesis is therefore testable. We will attempt to demonstrate the reality of the presence of the transition states of chromosome structure. This theory is established in memory of the late Dr. Oliver E. Nelson.

Phenotypic variations of maize after dry seed exposure to space conditions

--Pan, D, Zhou, W

It is known that space treated seeds usually show a pleiotropic effect on the phenotype of the plant during growth and development. The mechanism of the effect of space conditions on plant seeds is not really understood at the present time. In 2002, we sent seven maize inbred lines onboard the Space Shuttle Endeavor STS-112 to the International Space Station (ISS) for examination of the effect of space conditions on these inbred lines. After keeping the seeds on the ISS for ten days, the retrieved seeds were grown on earth and compared with Earth control seeds. Following is a summary of the observations on the phenotypes after germination and growth.

1. Generally, the inbred lines B73, W540 and A619 showed more vigor than their control counterparts during germination.
2. After space exposure, B73 had more tillers than control seeds.
3. One of the space treated inbred lines, A619, showed a mutagenic effect on the development of a purple strip on the shoot during growth.
4. On the ears of some of the space treated W540 inbred lines, the genetic effective kernels were found, and it was a lethal defect.
5. Space treated inbred line W22 r-r seeds showed early purple color intensified on the shoot during growth.
6. Space treated seeds of inbred-lines A619 and A631 showed earlier pollen shedding than Earth control seeds.

Generally speaking, some of the space treated seeds showed a variety of effects on phenotypes during germination and growth. The genetic consequence of the effect of space conditions on the seeds of some inbred lines is now under investigation.

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***ra1-154*, a new allele of the *ra1* mutant**

--Pilu R, Cassani E, Sirizzotti A, Landoni M

The ramosa maize mutant described here was originally observed in a selfed B73 family. This suggests that the mutation occurred spontaneously in the previous generation. In this mutant, originally named *ra*-154*, indeterminant branches form in place of spikelet pairs on the main axes of the normal tassel and ear. In contrast to normal tassels in which prominent branches emerge from the base of the inflorescence, the *ra*-154* tassels were branched along their entire length (Fig. 1). The genetic analysis of the *ramosa* mutant was based on the ascertainment of increased primary branching on the tassel at anthesis (Fig. 1A). Segregation analysis of the mutant phenotype based on this trait indicated that *ra*-154* was inherited as a monogenic recessive trait (Table 1).

To ascertain the relationship of the newly discovered ramosa mutation to previously described *ramosa1* (*ra1*) and *ramosa2* (*ra2*) maize mutations, we crossed these mutants inter se in all

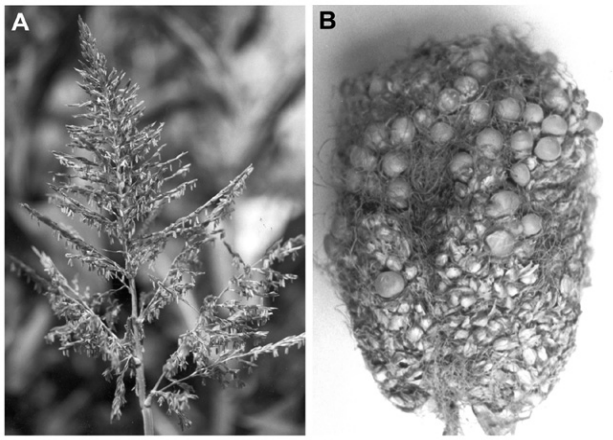

Figure 1. A, *ra*-154* tassel at anthesis, showing increased primary branching on the central spike; B, *ra*-154* ear at maturity.

Table 1. Segregation of the ramosa phenotype observed in genetic testing.

cross	segregation			
	wild-type	mutant	χ2 value	p
+/ra-154* selfed	108	50	3.13	0.10-0.50
+/ra-154* X *ra*-154/ ra*-154*	122	112	0.42	0.70-0.50
ra-154/ ra*-154* selfed	-	47	-	-

pairwise combinations to assay their pattern of complementation. The results obtained show that the *ra1* mutant failed to complement *ra*-154*, suggesting an allelic relationship. To corroborate this data, the map position for the ramosa mutation was achieved by the analysis of simple sequence repeat (SSR) marker-distribution in a F_2 segregating population, consisting of 60 plants obtained from the selfing of B73 *ra*-154/ra*-154* x W22 plants. A polymorphism for the markers *umc1666* and *bnlg1792* established the position of the mutation on bin 7.02, at a distance of about 3 cM from *bnlg1792* and about 8 cM from *umc1666* (recombinant values were converted to map distances using MAPMAKER 3). These data confirm the map position of *ra*-154* in the same genomic region of the *ra1* alleles previously described.

On the whole, these data indicate that the *ra*-154* mutant bears a lesion in the *ra1* gene. According to the guidelines indicated by the Maize-GDB we renamed the new mutation by the provisional designation *ra1-154*.

The *a1-eap* allele of *a1* represents a specific marker for the selection of embryogenetic mutants

--Gavazzi, G, Stucchi, C, Pilu, R

In some of our stocks, we occasionally observed seeds with a colorless aleurone and a ring of color surrounding the embryonic axis. A closer scrutiny under low magnification shows that this ring is due to pigmentation of a thin layer of cells of the scutellum surrounding the embryonic axis along its entire length or its apical portion (Fig. 1).

Sometimes an additional layer of cells is formed on the side of the scutellum facing the endosperm. After several rounds of selection we obtained a line, referred to as *eap* (*embryonic axis profile*), with this trait expressed in all seeds. When this line was crossed to an ACR line, homozygous for all genes required for aleurone pigmentation, the F1 was uniformly colored, whereas the F2 segregated colored and colorless seeds, the latter again showing the embryonic trait, in a 3 to 1 ratio. This observation suggested that this trait was due to a monogenic recessive color mutant. We then applied pollen of a single *eap* male parent to silks of five color tester lines, each one homozygous recessive for one of the genes required for seed pigmentation and homozygous dominant for the other color genes. These lines are here referred to as *c1*, *c2*, *a1*, *a2* or *r* testers. The resulting F1 seeds were homogeneously colored, and the F2 progenies segregated colored and colorless seeds in a 9 to 7 ratio, as expected if two genes responsible for seed pigmentation are segregating in each of the families scored (Table 1). A χ^2 test for 9:7 segregation fits the data except for the F2 where *a2* is segregating. Since in this case there is a significant excess of colorless seeds over the expected 7/16, this discrepancy could indicate preferential transmission of the *a2* marked chromosome. If we assume that the *eap* phenotype requires at least one dose of each of the color genes, the colorless seeds recovered in the F2 should be *eap* in 3 out of 7 cases, whereas the remaining 4/7 should be totally devoid of pigment. However the proportion of *eap* among the colorless seeds was consistently less than the expected 3/7. This could be the result of poor expression of this marker in a different genetic background.

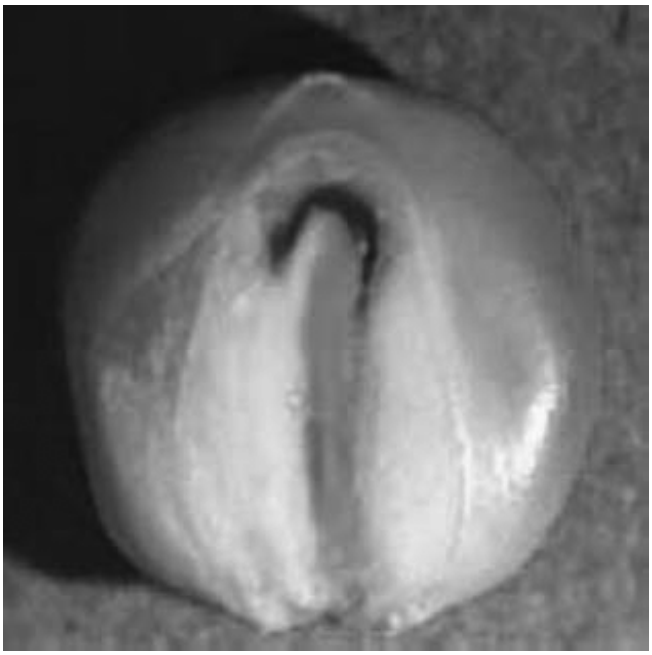

Figure 1.

Table 1. Segregation obtained in the F2 of crosses of five different color tester lines with the same male parent homozygous for *eap*.

cross	coded # of F1	F2 segregation		n	χ^2 (9:7)	P value
		color	colorless			
c1 x *eap*	9167	904	679	1583	0.48	.50-.30
c2 x *eap*	9168	1507	1188	2695	0.12	.80-.70
a1 x *eap*	9169	1775	1427	3202	0.87	.50-30
a2 x *eap*	9170	1514	1541	3055	56.5	< 0.01
r x *eap*	9171	450	342	792	0.01	.90-.80

An unplanned cross of *eap* females with a heterozygous TB-3La stock revealed unexpectedly that the *eap* phenotype is uncovered by this translocation. Since *a1* maps on 3L, we tried to establish the chromosomal location of *eap* with those microsatellites that define bin 3.09 where *a1* is positioned. An SSR analysis was performed on the F2 progeny of the cross *ACR*-r x *eap*. Two of the eight microsatellites tested on this segregating population showed polymorphism. Of the 62 individuals tested with the p-umc2008 marker only one recombinant was recovered, whereas five out of 36 recombinants were obtained with the p-umc1641 marker. These results place *eap* in a region where *a1* resides, with a recombination frequency between *eap* and *umc2008* and *umc1641* of 1.2 and 6.8 cM, respectively. The original *a1* tester employed in the complementation test was derived by conversion of *a1* into an *ACR-r* W22 inbred line.

We then crossed the *eap* line with other sources of *a1*, i.e. homozygous *a1R-sc*, *a1Mum2*, *a1Mum3*, *a1sh2* stocks, and we obtained progeny ears consisting of homogeneously colorless seeds. These results, together with the assignment of *eap* to the same bin where *a1* is located, support the conclusion that the *eap* phenotype is due to a recessive allele of *a1* that we accordingly named *a1-eap.* The apparent complementation of *a1-eap* with the *a1* tester in the W22 background remains to be elucidated.

Because of its phenotype the *a1-eap* allele can be used as a visible embryonic marker for the detection of mutants impaired in the development of the apical shoot. To find out the feasibility of this strategy we made use of the recently isolated *sml* gene (Pilu et al., Plant Physiology 128:502-511, 2003). The shootless phenotype caused by disruption of SAM formation, is due to the synergistic interaction of mutations of two genetic loci. Seedlings homozygous for both *sml* (shoot meristemless) and the unlinked *dgr* (distorted growth) loci have a SAMless phenotype, seedlings *Dgr/- sml/sml* are impaired in their morphogenesis to different extents, whereas the *dgr* mutation alone does not lead to a recognizable phenotype. The F2 obtained by selfing the progeny of homozygous *a1-eap Sml Dgr* females to *A1/A1 dgr/dgr Sml/sml* male parents consists of two kinds of progeny ears, one with all seeds homogeneously *eap* and another one segregating *eap/Eap* in a 3 to 1 ratio. Upon germination, seeds of the first class gave rise to normal seedlings whereas those of the second class yielded normal or a majority (80%) of abnormal (*sml* or *dgr*) seedlings following their separation into *eap* and *Eap*, respectively.

We take this result as evidence that the homozygous double mutant *a1-eap sml* exhibits a negative interaction, causing suppression of the embryonic axis pigmentation. Presence of a functional SAM thus seems necessary for cells around the embryonic axis to become competent to express pigment or, alternatively, that a functional SAM or a signal it elaborates, is required for pigment formation. We think that this observation represents useful information for the selection of mutants impaired in the shoot meristem formation or establishment. Mutants of the SAM could in fact be searched for in the selfed progeny of homozygous *a1-eap* seeds following chemical or transpositional mutagenesis by selecting exceptional M2 ears exhibiting segregation of the *eap* trait.

The *fdl* (*fused leaves*) mutation affects shoot apex organization and seedling growth

--Adamo, A, Dall'Aglio, C, Dolfini, S, Gavazzi, G, Consonni, G

Maize embryogenesis results in the formation of a massive cotiledonary structure, the scutellum, and a well differentiated embryonic axis, comprising the root and the shoot apex. The coleoptilar ring starts from a ridge on the face of the scutellum encircling the shoot apex, and consists of a sheathing structure surrounding the stem tip and the embryonic leaves, except for a small pore. At germination, the plumule and the coleoptile elongate. Initially the coleoptile grows faster than the plumule, but when it reaches the surface of the soil and is thus exposed to light, it stops growing and the plumule is extruded from the tip.

The *fused leaves (fdl)* mutant was isolated from an active *Spm* line by Dr. Jane Langdale (Oxford University), and behaves as a monogenic recessive mutant. The main effects of the mutation are detectable starting from the plumule emergence to the four leaves stage. Homozygous *fdl* seedlings are retarded in their germination and growth when compared to wild-type siblings. In addition, the first two mutant leaves appear curly, as a result of their growth while still enclosed within the coleoptile. Opening of the mutant coleoptile occurs with an irregular lateral fracture in contrast to the clear-cut hole formed in the wild-type. Regions of fusion between the coleoptile and the first leaf and, occasionally, between the first and the second leaf are also observed. At the emergence of the fourth leaf, the plant acquires a normal phenotype with expanded leaves, thus appearing indistinguishable from wild-type plants. Homozygous mutant plants can be grown to maturity and selfed.

A histological analysis was performed to find out if embryo development is also affected. We examined transversal sections of mutant and wild-type shoot apex at 14 dap (days after pollination). Interestingly in the mutant embryo, a file of coleoptilar cells occupied an ectopic position, invading the central region of the shoot apex, which is normally occupied by the leaf primordia. Accordingly, in embryo rescue tests, excised 12 dap embryos transferred to an MS medium, yielded the expected *fdl* phenotype.

We also investigated the effects of endogenous auxin application (IAA) on development of immature wild-type and mutant embryos (12 dap). Embryos were excised from segregating ears and transferred to MS medium or to MS medium containing IAA. IAA did not show a significant effect on wild-type shoot and root growth, whereas in the *fdl* mutants an increase in both seedling and primary root elongation was observed, particularly evident in the primary root. Wild-type and mutant embryos were cultivated on media containing the synthetic auxin 2,4-D. The *fdl* mutant phenotype can be mimicked in wild-type embryos grown in the presence of 2,4-D, but no phenotypic effects were detected in mutant embryos.

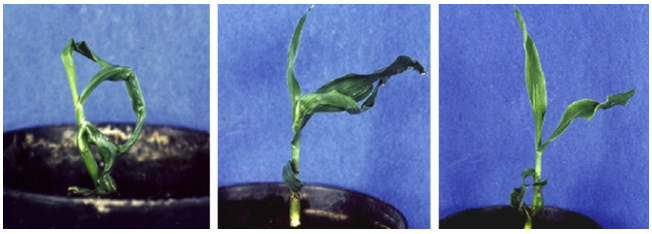

Figure 1. Phenotypes of the *fdl* mutant at different developmental stages.

Taken together these observations suggest that the mutation has a pleiotropic effect on both embryo and seedling growth. *fdl* has been located, by SSR analysis, on the long arm of chromosome 7 between *umc1342* and *umc1125*. Molecular genetic analysis indicates that the mutation is caused by insertion of a *Spm* element in the *fdl* locus. Efforts aimed at cloning the gene are underway.

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Characterization of new *vp* mutants not impaired in carotenogenesis

--Giulini, A, Bodei, S, Sirizzotti, A, Pilu, R, Burr, B, Gavazzi, G

The plant hormone abscisic acid (ABA) controls many aspects of plant growth and development under a diverse range of environmental conditions. To identify new genes functioning in ABA signalling and biosynthesis we collected a sample of 24 new mutants, derived from our collection (13) or from Dr. B. Burr (7) and Dr. D. Styles (4). Ten of these mutants are blocked in the synthesis of carotenoids, yielding albino seedlings. They represent mutational lesions on the early carotenoid pathway with down effects on ABA synthesis, whereas 14 have green seedlings and are thus not blocked in carotenogenesis. These presumably represent mutational lesions in steps of the hormone synthesis downstream of xanthophyll production, or they are response mutants impaired in the ABA receptor(s) or in the signal transduction. The data we present in this note refer to this second group of mutants.

Six of the 14 mutants isolated are allelic to *vp1* (3L), three mutants (*vpD**, *vp374**, *vp390**) represent another complementation group, do not complement *vp1* or *vp8* (1L) and are located on chromosome 1L, as determined by crosses with the B-A translocation stocks. Their allelism test with *vp14* (1L) is underway. *vp394**, whose chromosome location has not been determined, is not on chromosome 1L or 3L, thus representing a different gene from the previous two. *vp105** is not allelic to *vp1*, *vp8* and *vp10* and has a unique phenotype consisting of a green shoot apex with occasional vivipary. The last of this class, *rea*, shows a characteristic red embryonic axis and occasional vivipary. It is uncovered by TB-3L-2S and TB-3La, but it is not allelic to *vp1* on 3L. Its endogenous ABA content is normal.

Viability of the homozygous mutants is variable. Of those tested so far, *rea* is perfectly viable, *vp390**, *vp394** and *vp105** are lethals, while about one-half of the *vp374** mutants (13/29) can be rescued, and we succeeded in obtaining a homozygous ear (Fig.1). Mutants deficient in ABA biosynthesis or in response to ABA signals show a difference in their sensitivity to exogenous hormone. Whereas those ABA deficients, grown in the presence of ABA are highly inhibited in their growth, those insensitive are inhibited to a much lesser extent. We tested a sample of mutants of the first class (green seedling) and they all appear less inhibited, even though to a variable extent, than their wild-type counterparts (Table 1).

An interesting result was obtained when the sensitivity of a double mutant, *vp1-rea*, was compared to the one of the single mutants. The double mutant, in fact, appears significantly more

Figure 1.

Table 1. Response to exogenous ABA (10µM) of homozygous *vp* mutants and normal siblings obtained by selfing +/*vp* plants. The response is expressed as seedling elongation on media with ABA/ seedling elongation on media without ABA X 100. Length measurements were taken after 10 days of culture of immature embryos (30 DAP).

Mutant	F2 Segregat. (%)	Response to exogenous ABA	
		wt	*vp*
*vp374**	21.4	6.3	50.6
*vp390**	16.4	7.9	12.3
*vpD**	18.7	15.3	20.6
*vp426**	24.7	4.3	26.2
*vp105**	15.8	7.5	40.3

insensitive than either mutant taken alone (Fig. 2), a result suggesting an additive effect of *vp1* and *rea* on the decrease in sensitivity to the hormone. This would be expected if the two genes act on separate pathways of the transduction of the ABA signal. This conclusion corroborates previous results (Sturaro et al., J. Exp. Bot. 47:755-762, 1996) indicating that *vp1* and *rea* differ in their pattern of ABA inducible genes.

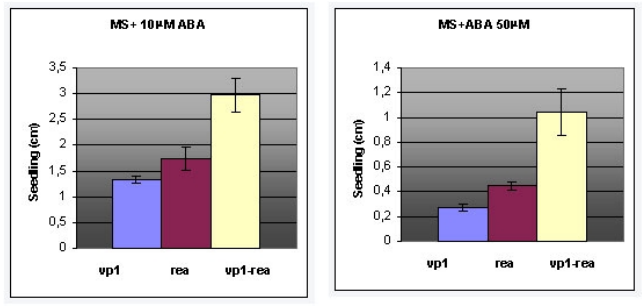

Figure 2. Effect of exogenous ABA at 10 or 50µM on growth of single (*vp1* and *rea*) versus double (*vp-rea*) mutants. Growth is expressed as seedling elongation on media with ABA/ seedling elongation on media without ABA X100. Length measurements were taken after 10 days of culture of immature embryos (30 DAP).

To test how the other mutants respond to ABA in terms of ABA responding genes, we started an RT-PCR analysis of *vp374**. 30-day-old embryos of the mutant were collected, incubated on solidified MS medium without or with ABA (10mM) for 48h, and then assayed for the presence of globulin, an ABA inducible gene. The result (data not shown) indicates that the mutant maintains its capacity to respond to ABA by inducing globulin. This test will be extended to the other mutants.

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Nucleotide sequence analysis of SCAR markers from *Zea mays* somaclones (*Zea mays* L.)

--Osipova, ES, Troitskij, AV, Dolgikh, YuI, Shamina, ZB, Gostimskij, SA

The deviations of the somaclones from the initial line A188 at the molecular level have been determined by RAPD- and ISSR-analysis (Osipova et al., MNL 74:52, 2000). Based on some specific RAPD- and ISSR-fragments, which were typical both for individual somaclones and for the groups of related ones, six SCAR-markers (Sequence Characterized Amplified Region) were produced (Osipova et al., MNL 77:53, 2003).

When we compared the nucleotide sequence of the SCAR-markers, using GenBank and PlantGDB (URL: http://www.ncbi.nlm.nih.gov/BLAST/ and http://www.zmdb.iastate.edu/cgi-bin/PlantGDBblast/ZMDB?db= Zea+mays) the most interesting results were obtained for the M-10 fragment. This marker contains the famous retrotransposon Opie-1 5'LTR (90% identity, error probability $3e^{-54}$) (Fig. 1).

The M-10 fragment was also homologous to part of the gene, which corresponded to the first exon of *Zea mays* ribosomal protein – L26 mRNA (Fig. 2). The deletion, consisting of 23 nucleotides and resulting in the shift of the reading frame, was found in the M-10 sequence, which corresponded to the L26 gene. A discrepancy of the sequences at the end of this

```
          >gi|3452304|gb|AF050451.1|AF050451   Zea mays retrotransposon Opie-1 5' LTR,
        partial sequence
                     Length = 1231

          Score =  220 bits (111), Expect = 3e-54
          Identities = 171/190 (90%), Gaps = 2/190 (1%)
          Strand = Plus / Minus

M-10:   46  acacaactcag--agttctctactcaaatggagctctagttgctatcacaaagaatcgaa 103
            ||||||||||  ||||| | ||| |||||||||||||| |||||||| ||||||||| ||
Opie-1: 776 acacaactcagcgagttcacaacttaaatggagctctaattgctatcgcaaagaatcaaa 717


M-10:   104 tgcgcggaaatgaggtcttggtgcttaggaatgctcaagggatgcttggtgtactcctcc 163
            |||||||||  || |||||||||||||||||||| |  | |||||||||||| |||||||
Opie-1: 716 tgcgcggaatcgaagtcttggtgcttaggaatgcttagagaatgcttggtgtgctcctcc 657


M-10:   164 atgcgcctaggggtccctttttatagctccaaggcagctaggagccgttgagagcattcca 223
            ||||||||||||||| ||||||||| |||||||||||||||||||||||| ||||||||
Opie-1: 656 atgcgcctaggggtccttttttatagccccaaggcagctaggagccgttgagtgcattcca 597


M-10:   224 ggaaggcaat 233
            ||||||||||
Opie-1: 596 ggaaggcaat 587
```

Figure 1. Finding the retrotransposon Opie-1 5'LTR in SCAR marker M-10.

```
M-10                              CCGCTGCCATGAAGCGCAATCCCCGCATCACGAGCTCCCGCCGGAAGTGC 400
Zea_mays_ribosomal_protein_L26    CCGCCGCCATGAAGCGCAATCCCCGCGTCACGAGCTCCCGCCGGAAGTGC 96
                                  **** ******************** ************************

M-10                              AGCAAGGCGCACTTCACGGCCCCGTCCTCCGTCCGCTGCGTTCTCATGTC 450
Zea_mays_ribosomal_protein_L26    CGCAAGGCGCACTTTACGGCCCCGTCCTCCGTCCGCCGCGTTCTCATGTC 146
                                  ************* ******************** *************

M-10                              CGACGGGCTGTCGACGGAGCTCC-----------------------ATCC 477
Zea_mays_ribosomal_protein_L26    CGCGGGGCTGTCGACGGAGCTCCGCCACAAGTACAACGTGCGCTCCATCC 196
                                  **  ******************                        ****

M-10                              CGATCCACAAGGACGACGAGGTGCAGGTCGTGCGCGGCACCTACAAAGGC 527
Zea_mays_ribosomal_protein_L26    CGATCCGCAAGGACGACGAGGTGCAGGTCGTGCGCGGCACCTACAAGGGC 246
                                  ****** ************************************ ***

M-10                              CGTGAGGGGAAGGTGGTGCAGGTGTACCACCGCCGCTGGGTCATCCACGT 577
Zea_mays_ribosomal_protein_L26    CGTGAGGGGAAGGTGGTGCAGGTGTACCGCCGCCGCTGGGTCATCCACGT 296
                                  ************************** ********************

M-10                              CGAGCGGATCACACGCGAGAAGGTGAACAGCTCTGTGCCTGGATGAGCGA 627
Zea_mays_ribosomal_protein_L26    CGAGCGGATCACCCGCGAGAAGGTGAACGGCTC----------------- 329
                                  ************ *************** ****

M-10                              TCATCCAAGAGATAGACTAGGCTCTATGCCTGGATGAGCGATCCGCAAGG 677
Zea_mays_ribosomal_protein_L26    -------------------------------------------------
```

Figure 2. Finding the part of the gene sequence encoding the first exon of *Zea mays* ribosomal protein L26 mRNA (marked in grey) in SCAR marker M-10.

gene encoding site and fragment M-10 was also noticed. Thus, we can assume that such destructive changes in this gene resulted from somaclonal variation within the process of cultivation.

When analyzing the Leb marker, it was found that this marker could contain either two unannotated repeats or the site homologues of the unknown rice gene. There are hypothetical acceptor and donor sites in the Leb sequence at the bounds of the identity segment (Fig. 3). Perhaps this fragment has some homology with the analogous maize genes which haven't been sequenced yet.

Some repeating sequences were revealed in markers QR-A and QR-2. As a result of sequencing, it was found that the QR-2 fragment (1018 bp), which was characteristic of the somaclones, differed from the QR-A fragment (1428 bp), which was characteristic of the initial line A188 (Fig. 4). They differed in the deletion located from the 490 to 944 position, which was probably the result of somaclonal variation.

TGTATAGACTCATCAAAAGCCTGGACCCATGGGGAGCCCATGATGATTTCCGGGGAAGACAGGCTTCGGCCCCGTAGACCAGGAAGAACGGGGTATCCCC
GGTGGCACGGCTGGGTGTTGTCCGGTTCCCCCATAGCACGGATGGGAGTTCACTGACCCAATTCGCACCATGCTTCTTCAAGTAATCGTAGGTGCGCGTC
TTGAGTCCCCTGAGGATCTCTGCATTTGCCTTTCCAACTTGGCCATTACTCCTGGGGTGGGCCACGGACGCGAAGCAGAGCTGGGTGTCGATGCCCTTGC
AATACTCCTGGAAGATCTGACTTGTAAACTGGGTTCCATTGTCCGTAATGATACGGTTTGGGACCCTAAATCTTCAGACGATGGACTTGAGGAAGGCGAC
AGCGGCACCCTAGGTGATGTTGACCACAGGGGTGGCCTTTGGCCACTTGGTGAACTTGTCGATGGTGACGAAGAGGTACCGGTACCCGCCAACGGCCCTG
GGAAATGGTCCCAGGATATCCACCCCCCATATGACGAATGGCCAGGAGGGTGGAATCATCTTCAGAATCTAAGCTGGTGTGTGTATCTGCTTTGCGTGGA
ATTAACACGCCTTGCAGGACTTTACCATCTCAGCTGCATCCTGGAGAGCGGTTGGCCAGTAGAAGCCATGCCAGAAGGCCTTACCGACCAGTGTGCAGGA
TGATGAATGACTTCCGCACTCTACTCCATGGATCTCCGTGAGCAACTCACGGCCCTCCTCCTGGGTAATGCACCGCATGTGAATGTCGTTGGCGCCGCGG
TGGTAGAGATCCCCTTCCACCACCGTGTAGCATTTAGCCAACCGCACTATGCGCTCTGCAGACACATGGTCTTCAGGAAGGATATTCTCCTTCAGGTAGT
CCCGGATCTCGGAGACCCATGCATCGGGACCACTCTG

Figure 3. Finding the hypothetical acceptor and donor sites (marked in dark grey) in the SCAR marker Leb located at the ends of the identified segment. The site homologues of the unknown rice gene is marked in grey.

```
                ++++++++++++++++++++++++++++++++++++++ ++++  ++++++ ++++++++
QR-2 (420)  AATCCCGGTGATCTACATCCCTGCCATCTTGGCTGGCAGAAACgaGAGTGCCGCTGAATT
QR-A (420)  AATCCCGGTGATCTACATCCCTGCCATCTTGGCTGGCAAAAAC--GAGTGCTGCTGAATT

                ++++++++++++
QR-2 (480)  CAAACACAGCTAA----------------------------------------------
QR-A (478)  CAAACACAGCTAAgggcctgtttggaagtagagttattccatagttttcatgcaatacca

QR-2 (493)  ------------------------------------------------------------
QR-A (538)  tagtatatagaaataccatggtatttcaaacccaaagctgtttggttggactttagaaaa

QR-2 (493)  ------------------------------------------------------------
QR-A (598)  cagagttttgaataccatagttttatcaataccatggtatttctgtggtattgaaaactg

QR-2 (493)  ------------------------------------------------------------
QR-A (658)  aagtccagaccaaagtttttcctttcttgcgcgtgttggactgcaccattatctttttct

QR-2 (493)  ------------------------------------------------------------
QR-A (718)  gcgctttactagttttgcttcatgtccgattggtccagatgttgacaatgtcttgtttac

QR-2 (493)  ------------------------------------------------------------
QR-A (778)  ggaaaaacctgtgacttatgtgacatggctccaaacatgtggtagtatctagggatacct

QR-2 (493)  ------------------------------------------------------------
QR-A (838)  tggttttaccttgggattagtaaaagggtagtattatgtcataacaattgttagactacg

                                                            +++++++++
QR-2 (493)  ----------------------------------------------------TACTTGGTT-
QR-A (898)  gtatttctaaaccataggtttacaaaattgtgtttccaaacagggcctaaTACTTGGTTg

         ++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
QR-2 (502)  CCCTGTTCTCTTGCTCCAGTCAACTACTTCCATCCACCAGGATGTAGATCCTAATGCTGG
QR-A (958)  CCCTGTTCTCTTGCTCCAGTCAACTACTTCCATCCACCAGGATGTAGATCCTAATGCTGG
```

Figure 4. Comparison of nucleotide sequences of SCAR markers QR-2 and QR-A.

The OPC-09 marker could contain either two unannotated repeats or the site homologues of the rice gene gag/pol, which is typical for encoding sites of retrotransposons (Table 1).

The results of our analysis demonstrated that these fragments mostly contained the repeats (Table 1). Such repeating sequences in specific RAPD- and ISSR-fragments were characteristic for somaclones. This corresponded to the available data of other researchers. In accordance to these, the somaclonal changes more often take place in the non-encoding sites of the genome.

Table 1. Analysis of nucleotide sequences of SCAR markers.

Name of the SCAR marker	Hits according to BLAST (NCBI)	Repeats according to Repeatmasker	Conclusion
M10 (806 bp)	There are substantial hits in *Zea mays* ribosomal protein L26 mRNA and *Zea mays* retrotransposon Opie-1 5' LTR.	There is a masked repeat (2-236 bp) characteristic of cereals.	Known repeats and pseudogenes are possible. There is a frameshift in exon.
Leb (937 bp)	There are two hits in *Z. mays*, both in unannotated parts.	There is a masked repeat (63-761 bp) characteristic of cereals.	Two new repeats or genes are possible.
OPC-09 (640 bp)	There are two hits in *Z. mays*, both in unannotated parts.	No repeats were found.	Two new repeats or the destructive gene, gag/pol, characteristic of retrotransposons are possible.
QR-2 (1018 bp)	No hits were found.	No repeats were found.	New repeat is possible.
QR-A (1428 bp)	No hits were found.	No repeats were found.	New repeat is possible.
Q-20 (796 bp)	No hits were found.	No repeats were found.	No data.
NO-15 (204 bp)	No hits were found.	No repeats were found.	No data.

No homologues were revealed for the NO-15 and Q-20 markers. This allows us to assume these fragments were specific for the somaclones investigated.

To clarify the nature of the Q-20 marker and its distribution in the genome, the Southern-blot hybridization analysis was applied. Hybridization of the Q-20 probe revealed one major 550 bp fragment at the second group of somaclones. For the R105 regenerant, the same fragment was also found in a high-molecular zone that assumes the duplication of this fragment (Fig. 5).

Thus, by means of sequencing SCAR markers, which were created on the basis of some polymorphic RAPD- and ISSR-fragments, some homology with known sequences of *Zea mays* and some other cereals was found. The changes were revealed both among repeating and unique sequences. The finding of the retrotransposon in the M-10 fragment corresponds to the assumption about the possible activation of some mobile element in in vitro culture. This may be one of the reasons for the appearance of some genetic changes.

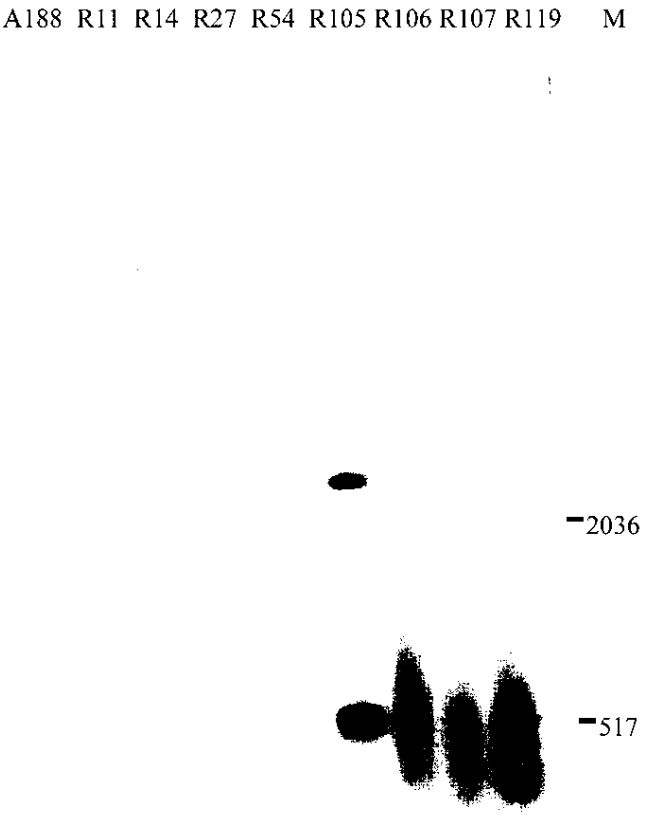

Figure 5. Southern-blot analysis of *Eco*RI, *Eco*RV, *Xba*I-digested DNA hybridised to the alpha ^{32}P-dATP-labelled Q-20 SCAR probe. A188 – the initial maize inbred line; R11-R54 – the somaclones of the first group, obtained after two months subculturing; R105-R119 – the somaclones of the second group, obtained after eight months subculturing; M – the molecular-weight marker (1 kb DNA ladder "GIBCO BRL").

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Maize chromosome polymorphism: A comparison between pachytene chromosomes and mitotic chromosomes using C-banding and FISH of 5S rDNA and Cent-4 probes in tropical hybrids

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Pachytene chromosomes have been considered the most appropriate stage for maize cytogenetics, due to the presence of easily detectable components, such as heterochromatic knobs, centric heterochromatin and enlarged chromomeres. Accurate identification of mitotic chromosome markers is highly desirable for studies involving somatic tissues and for identification of polymorphism among maize varieties.

In this note, we report some aspects of the investigation of the karyotypes of two tropical maize hybrids by conventional pachytene analysis, C-banding of mitotic metaphases and fluorescence in situ hybridization (FISH) of a 5S rDNA probe, as a marker for chromosome 2 (Mascia et al., Gene 15:7--20, 1981), and of a chromosome 4 centromere-specific repeat, designated

Cent-4 (Page et al., Genetics 159:291-302, 2001). The hybrids studied (441123 x 4443 and 441311 x 442612) were obtained in our laboratory by crossing sister inbred lines, derived from a flint maize variety (Jac Duro, Sementes Agroceres, Brazil). Their knob composition (see Table 1) is quite similar, except for chromosome 9, which is homozygous for K9S in the hybrid 441131 x 442612 and heterozygous in 441123 x 4443. In the present note, we aimed to emphasize the importance of the use of in situ hybridization of specific chromosome markers to resolve a problem of identification of chromosomes 2 and 4 in these materials.

A method previously described for root pretreatment and fixation (Bertão and Aguiar-Perecin, Cariologia 55:115-119, 2002) was employed to obtain metaphase spreads used in the C-banding (carried out as described in this reference) and FISH procedures. Preparations of microsporocytes for analysis of pachytene chromosomes were made according to current routine (Dempsey, The Maize Handbook, New York: Springer-Verlag, pp. 432-441, 1994).

Arm lengths of pachytene and C-banded mitotic metaphase chromosomes were determined from photographic prints and the chromosomes were identified according to their relative length and arm ratio, as described in the literature (Neuffer et al., Mutants of Maize, New York: Cold Spring Harbor Press, 1997; Aguiar-Perecin and Vosa, Heredity 54:37-42, 1985). Relative lengths of the mitotic chromosomes and C-bands were expressed as percent of the length of chromosome 10.

The 5S rDNA probe was a fragment amplified by PCR using the primers 5'GTGCGATCATACCAGC(AG)(CT)TAATGAACCGG3' and 5'GAGGTGCAACACGAGGACTTCCCAGGAGG3' (see Gottlob-McHugh et al., Genome 33:486-494, 1990). The Cent-4 probe (cloned in Bluescript and kindly provided by Dr J. A. Birchler, University of Missouri, USA) was amplified by PCR using M13 forward and M13 reverse primers. The Cent-4 sequence was labeled with biotin-14-dATP using the BioNick labeling system (Life Technologies), and the 5S rDNA probe with digoxigenin-11-dUTP, by random primed DNA labeling (Roche). In situ hybridization was performed as described (Schwarzacher and Heslop-Harrison, Practical in situ hybridization, Oxford: Bios, 2000), with slight modifications. Fluorescein-conjugated sheep anti-digoxigenin (Roche) was used to detect the rDNA sites. Three antibodies were necessary for a consistent detection of Cent-4 sequences: mouse anti-biotin, TRITC-conjugated rabbit anti-mouse and TRITC-conjugated swine anti-rabbit (DAKO). Chromosomes were counterstained with DAPI.

Figure 1 shows that in situ hybridization using fluorescein-labeled 5S rDNA revealed that the probe was localized on the long arm of a pair of chromosomes with submedian centromeres (arm ratio = 1.43 at mitotic metaphase and 1.71 at pachytene). The FISH signal of the TRITC-labeled cent-4 sequence was detected on the centromere of a pair of chromosomes with arm ratio values of 1.25 at mitotic metaphase and 1.30 at pachytene. Interstitial signals were not observed, as reported for other genotypes investigated by Page et al. (2001). Due to these arm ratio values and relative lengths, as well, these chromosomes had been identified as 4 and 5, respectively, in a previous paper (Bertão and Aguiar-Perecin, 2002). We reconsidered this interpretation, considering that 5S rDNA has been mapped to chromosome 2 (Mascia et al., 1981) and that the Cent-4 sequence was identified on the centromere of chromosome 4 (Page et al., 2001). Based on this conclusion, we organized Table 1, in which the knob composition, relative lengths and arm ratios of mitotic chromosomes, and arm ratios of pachytene chromosomes are shown. Figure 2a illustrates the C-banded karyotype of hybrid 441123 x 4443. Most of the values in Table 1 are coherent with the ones described in the literature, except for chromosomes 2 and 4. The arm ratio value of chromosome 2 at pachytene (1.71) seems rather high as compared with the range of 1.14-1.42 reported in the literature (Neuffer et al., 1997). Also, the range reported for arm ratio of chromosome 4 (1.63 to 2.0) is higher than our finding (1.30). The main point is that generally, the centromere is located in a position more submedian in chromosome 4 than in chromosome 2, and this is not the case in the materials investigated and also in other related lines (not shown). Figures 2b and c show these chromosomes at pachytene. Chomosome 5, which possesses a knob on the long arm (Figure 2d) is also shown, because it had been interpreted as number 2 in our previous paper. In this case, its arm ratio at pachytene (1.13) is consistent with the pattern described in the literature. As shown in Figure 2a and Table 1, in mitotic metaphases this chromosome is larger than chromosomes 2, 3 and 4 and its arm ratio is 1.35, due to the presence of the C-band in the long arm. In addition, the length and arm ratio of the other chromosomes, estimated on C-banded metaphases of hybrid 441123 x 4443, are coherent with values previously described in a comparative study between knobless chromosomes of Ceremonial race and the karyotype of a highly knobbed stock of Zapalote Chico (Aguiar-

Table 1. Biometrical analysis of mitotic C-banded metaphase and pachytene chromosomes of the hybrid 441123x4443.

Parameters	Chromosomes #												
	1	2	3		4	5	6	7		8	9		10
			KL+	KL–		KL+	KL2+ /L3+	KS+ / KL+	KS-/KL+	KL1 +/L2 +	KS+	KS -	
Metaphase													
RL	181.11	150.13	155.35	151.31	145.18	157.49	126.81	160.47	147.03	138.31	138.56	110.25	100
AR	1.18	1.43	1.82	1.72	1.25	1.35	1.75	1.64	2.38	2.49	0.88	1.63	1.74
C-band RL			15.91			20.36	12.03	19.89 / 27.09	/27.09	21.03	27.29		
Pachytene													
AR	1.28	1.71	2.59		1.30	1.13	6.73	2.17		3.82	1.47		2.68
Knob *			M			M	S / S	M / L		M / S	L		

RL = Relative lengths of chromosomes and C-bands expressed as percent of the length of chromosome 10.
AR = Arm ratio (long arm/short arm).
* Knob size = L, large; M, medium; S, small.
#: Numbers refer to chromosomes; knob location: (+), presence of knob; (-), absence of knob; knobs at 6L2, 6L3, and 8L1, 8L2 observed at pachytene, appear as a single band on mitotic chromosomes.

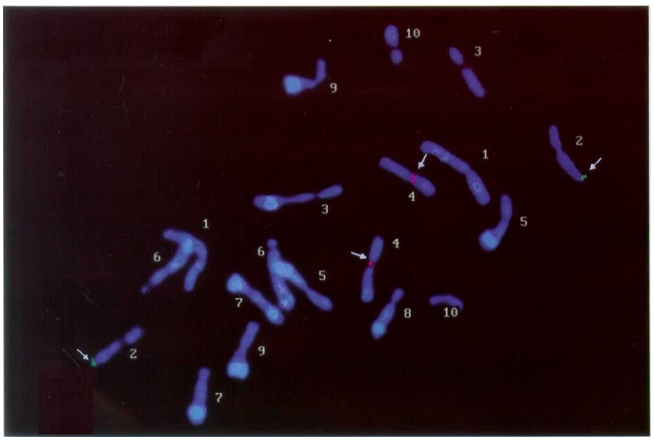

Figure 1. Mitotic metaphase of hybrid 441131 x 442612. Fluorescence *in situ* hybridization of 5S rDNA (green) and Cent-4 sequence (red). Chromosomes are counterstained with DAPI. DAPI bands correspond to knobs visualized at pachytene.
One chromosome is missing (number 8).

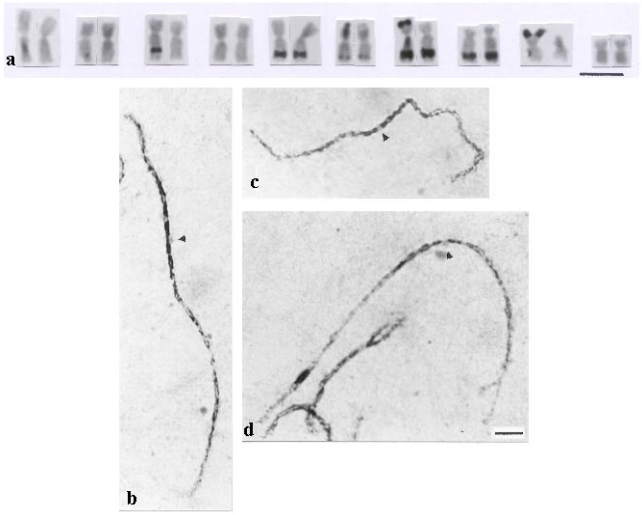

Figure 2. C-banded metaphase of hybrid 441123 x 4443 (a); pachytene chromosomes: chromosome 2 (b), chromosome 4 (c), chromosome 5 (d). Bars = 5um. Pachytene chromosomes are in the same magnification; arrowheads indicate the centromeres.

Perecin and Vosa, 1985). Also, relative lengths of C-bands are quite correspondent to knob sizes visualized at pachytene and expressed arbitrarily as large, medium and small, as seen in Table 1.

The present study emphasizes the importance of the use of molecular markers for cytogenetic analysis of maize chromosome polymorphism, that must be due not only to the presence of detectable knobs. We could speculate that variable content of different classes of repetitive could modify the gross morphology of the chromosomes as discussed here for chromosome 2 and 4, considering, for instance, that retrotransposon insertions have increased the size of the maize genome during its evolution (see San Miguel et al., Nature Genetics 20:43-45, 1998).

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High-resolution physical mapping of the maize genome and sequencing a part thereof

--Bharti, AK, Wei, F, Butler, E, Yu, Y, Goicoechea, JL, Kim, H, Fuks, G, Nelson, W, Hatfield, J, Gundlach, H, Karlowski, WM, Raymond, C, Towey, S, Jaffe, D, Nusbaum, C, Birren, B, Mayer, K, Soderlund, C, Wing, RA, Messing, J

Due to its economic significance, maize (>2 Gb) is likely to be the next cereal to be sequenced after rice (0.4 Gb). Sequencing the maize genome will present a new challenge not only because it is 5-times larger but also because it contains many gene families, tandemly arrayed and nested repeat sequences. Since maize genetics is so well advanced, it will aid in developing a map-based sequencing approach, which will further provide an arsenal for functional genomics. As a beginning towards sequencing the maize genome, out of a set of 156 BACs chosen, we have already sequenced 48 random clones. In addition, we have also sequenced 355,294 BAC ends yielding random small fragments of an average size of 627 bp (Table 1), which represents >11% of the maize genome (223 Mb). To establish a link between sequences and the genetic map, the same clones that have been sequenced at their ends have also been fingerprinted. DNA fingerprinting of highly redundant BAC libraries yields bins of overlapping BAC clones in a high-throughput fashion. Physical linkage of large bins of BACs can then be established to the genetic map by detection of genetic markers contained within individual BAC clones either by filter hybridization or by PCR-related methods. In the previous NSF-funded Maize Mapping Project (MMP: www.genome.arizona.edu/fpc/maize), 10,913 such BAC clones (anchored by 1,937 markers) have already been identified. Since these anchored BACs are positioned within contigs, the entire contig can be linked to the map (Fig. 1). A limiting factor to the anchoring process is marker density and the size of BAC contigs. The maize genetic map is about 2,000 cM, which would translate into roughly one cM per Mb. Given the current marker density and the preference for at least two markers per contig, it becomes necessary to generate contigs that are very large in size. For

Table 1. Current status of fingerprinting and sequencing of Zea mays ssp. mays cv. B73 BAC Libraries.

High Information Content Fingerprinting (HICF)	
Passed QC (presence of internal standards and expected vector bands)	"382,696"
"Genome Coverage based on 2,036 Mb coverage by agarose fpc"	28.2x
BAC End Sequencing (BES)	
Submitted to GenBank	"355,294"
Average read length submitted to GenBank	627 bp
"Genome Coverage based on 2,036 Mb coverage by agarose fpc"	11% (223 Mb)
BAC Sequencing	
Submitted to GenBank (28 Phase 1 and 20 Phase 2)	48

this we need a high resolution fingerprinting method that is capable of identifying even small overlaps between neighboring BACs, thereby resulting in large contigs.

Earlier, MMP had generated a genetically anchored physical map using *Hin*dIII-digested agarose-based fingerprints of BACs offering 21.5x physical coverage of the genome. Manual editing of the agarose fpc assembly has resulted in an increase in the number of contigs with >200 overlapping BAC clones from 272 to 390 and a decrease in the total number of contigs from 4,518 to 3,488 (1,446 anchored contigs). To further reduce the number of contigs and also enlarge their sizes, a high-resolution fluorescent fingerprinting method known as HICF (High Information Content Fingerprinting), has been carried out for the same BAC libraries (NSF-B73 and CHORI-201). A total of 305,849 BAC clones have already been fingerprinted and assembled into contigs by the HICF method whose results (Table 2) can be accessed via an interactive website (www.genome.arizona.edu/fpc_hicf/maize). Using the BAC nomenclature and address, all agarose-based contigs are being aligned to the HICF contigs (Fig. 1).

HICF is based on simultaneous restriction of the DNA with a type IIS restriction enzyme (*Earl*) along with a 4-base cutter (*Taql*) followed by labeling the ends with base-specific fluorescent dyes and resolving the fragments within a 35-500 bp range along with internal size standards. Based on simulations, it has been observed that the agarose fingerprinting (~40 bands/BAC)

Table 2. Current status of the agarose and the HICF fpc builds.

Particulars	"Agarose fpc build (June 06, 2003)"	"HICF fpc build (December 15, 2003)"
Number of successful fingerprints	"292,039"	"305,849"
"Genome Coverage (Based on 2,036 Mb Coverage by Agarose fpc and Av. Insert Size of BAC Libraries as 150 kb)"	21.5x	22.5x
Number of contigs	"3,488*"	"4,681"
Number of markers	"15,422"	"15,403"
Number of anchored contigs	"1,446"	"2,010"
Number of singletons	"14,482"	"33,566"
Contigs with >200 BACs	390	188
Contigs with 101 to 200 BACs	528	829
Contigs with 51 to 100 BACs	570	979
Contigs with 26 to 50 BACs	461	694
Contigs with 10 to 25 BACs	400	514
Cut off	e-12 (~70% overlap)	e-48 (~58% overlap)
Tolerance (resolution)	7 (=7 bp)	7 (=0.35 bp)

*After manual editing

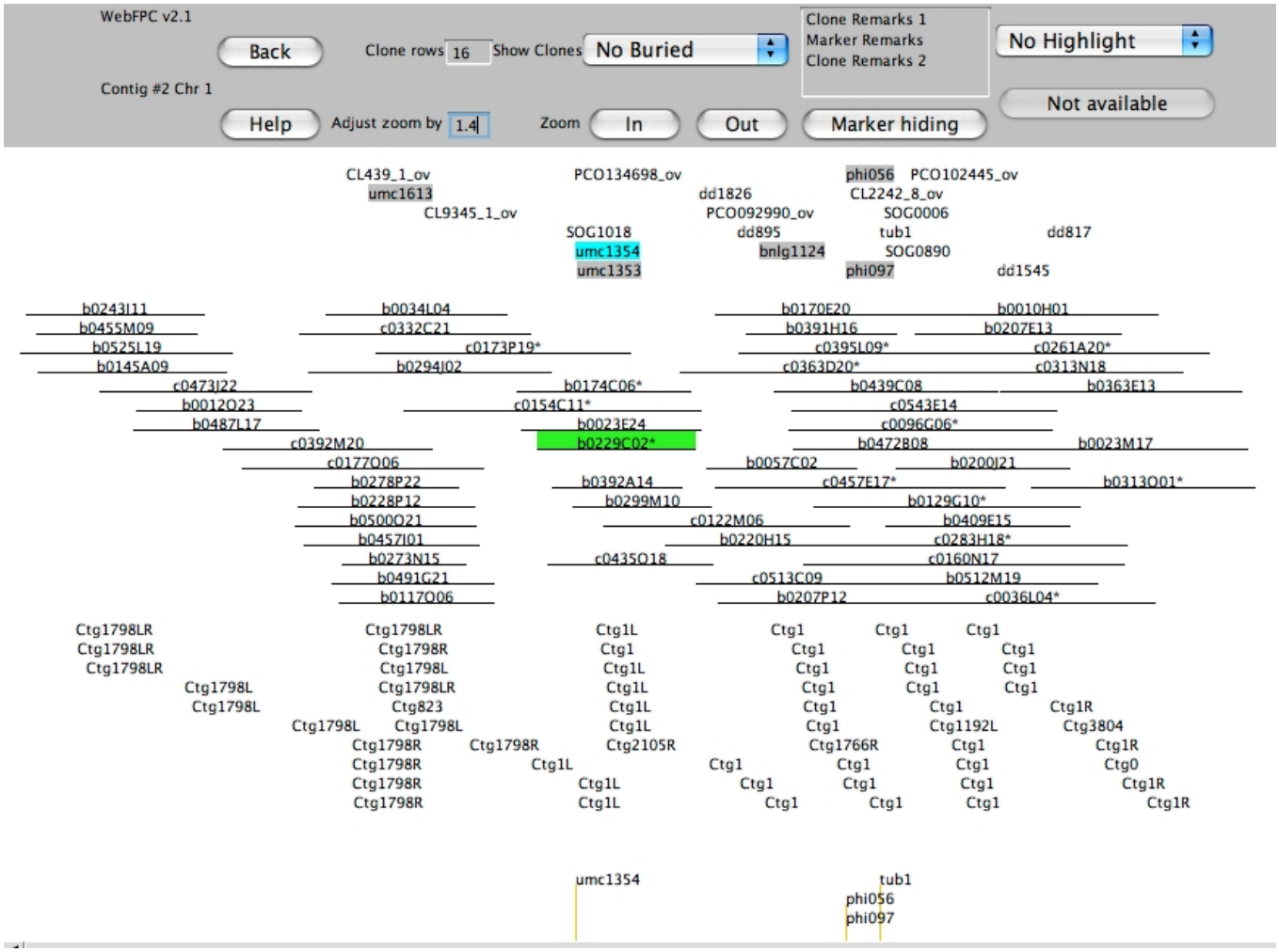

Figure 1. The HICF Contig# 2 (Chr 1) consists of two agarose contigs Ctg#1 and Ctg#1798. The clear-cut boundary between the two agarose contigs (both from maize chromosome 1) indicates a clean merge. The BAC clone b0229C02 (highlighted) has been anchored to chromosome 1 by the marker *umc1354* (highlighted).

requires a clone overlap of ~70% to achieve a medium cutoff (e-12). With the HICF (~120 bands/BAC), assembly can be achieved at a much lower cutoff (e-48), which requires a slightly lesser overlap of ~58%. Moreover, a resolution of as low as 0.35 bp is being used for the HICF fpc build as opposed to 7 bp for the agarose fpc build. Due to the restriction site bias, it is more likely that HICF will also cover those regions of the maize genome that have fewer *Hin*dIII sites. The HICF method, originally designed for gel-based sequencers (ABI377) at DuPont, has been adapted successfully for use with capillary sequencers (ABI3700). The fluorescent trace output is extracted using ABI GeneScan Analysis software. In order to call peaks and produce fpc-compatible files, the perl script provided by DuPont is being utilized with their peak-scoring scheme (with modifications). Typically the observed band sizes are off by 2 to 6 bp from the expected simulated digests. However a very good consistency in the sizes between duplicated runs has been observed in the HICF data being generated from different machines. As far as reproducibility of band sizes are concerned, in the observed vector band sizes from >72,000 HICF fingerprints, there is a maximum average size standard deviation of 0.11 bp.

Presence of the expected BAC vector bands along with the internal size standard peaks are being used as the two quality control (QC) steps for analysis of the entire HICF data set. As of now, the HICF success rate is ~86%, which has yielded 382,696 successful fingerprints that pass both the above criteria (Table 1). The current HICF fpc build has been made with data from 305,849 clones after screening for fingerprints with band count <175, which has resulted in 4,681 contigs (Table 2). There are numerous examples indicating that HICF can determine overlaps where the agarose method cannot (Fig. 1). Thus, the HICF method is likely to significantly reduce the number of contigs in the physical map by successfully joining distinct agarose-based contigs along with singletons. Furthermore, because a fraction of BAC end sequences is conserved in orthologous positions in the rice and maize genomes, additional contigs are expected to get anchored to the maize map due to the rice-maize synteny. It is also expected that resolution of the HICF-generated contigs would be high enough to select the entire Minimum Tiling Path (MTP) at once, thereby providing a minimal BAC clone set of <20,000 clones representing the entire maize genome. This single filter clone set available for hybridization would be a powerful tool in the hands of researchers interested in identifying a desired locus from within the maize genome. This would greatly facilitate cloning of desired marker-assisted traits.

Table 3. Repeat content of 43 BACs analyzed.

Number of repeat hits	"4,364"
Average number of repeat hits per BAC	101
Average length of repeat hit	"1,186 bp"
Total number of nucleotides	"7,086,942 bp"
Number of nucleotides as repeats	"5,178,209 bp"
Percentage of nucleotides as repeats	73%

Table 4. Distribution of the 73% hit in terms of total length and number of hits among each repeat category.

	No. of hits	% of hit no.	Length of hit (bp)	% of hit length
Class I elements (retroelements)	"3,660"	83.87	"4,969,641"	95.97
Class II elements (DNA transposons)	550	12.60	"160,301"	3.11
Ribosomal	5	0.00	"1,327"	0.03
Telomeric repeats	18	0.00	"2,622"	0.05
Centromeric repeats	95	0.02	"35,447"	0.68
Other repeats	32	0.01	"8,871"	0.17

Besides the development of a BAC-based genetic map of maize, we are also assembling information about the repeat sequences of the genome by de novo repeat detection from the sequenced BACs. This updated repeat database would be very useful for future comparative genomics work. Though it has been suggested that the maize genome is composed of small gene islands interspersed between large oceans of repetitive DNA, a more detailed picture of this organization is yet to emerge. Annotation of an initial set of random BACs sequenced indicate that large repeat-free open spaces vary from region to region in the genome. Another very interesting observation that could be drawn from the analysis is that most repeat elements are shorter than those existing in the repeat collection. This points towards a high incidence of fragmentation of repeats within the maize genome. Comparison of 43 sequenced BACs to the updated repeat database reveals a total repeat content of 73% (Table 3), out of which nearly 96% of the sequence length is comprised of retroelements and only about 3% constitutes DNA transposons (Table 4). In addition to the 48 BACs already sequenced, sequencing of 108 more clones will enable us to assemble a more complete set of repeat elements and assess their abundance.

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The distribution of chromosome breaks in radiation hybrid lines from chromosome 3

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The distribution of breaks induced by radiation is believed to be randomly distributed along a DNA molecule. Therefore the number of breaks in an interval is dependent on the size of that interval. This principle forms the basis for radiation hybrid (RH) mapping and has been supported by work in a number of animal systems. Our lab has been engaged in developing RH lines derived from oat x maize chromosome addition lines. RH mapping panels for maize chromosomes 1, 2, 3, 4, 6 and 9 have been produced from the RH lines. Chromosome breaks in these lines were distributed reasonably evenly across the chromosomes, except for chromosome 3 RH lines. Over two-thirds (15/22) of the chromosome 3 RH lines have a break between markers *umc1223* and *mmp9* (data not shown). These markers are located at positions 234.4 and 262.9 cM on the chromosome 3 IBM2 genetic map; this distance is approximately 3.5% of the total genetic length of this chromosome. Because the probability of a radiation-induced break is proportional to the physical size of a region, this result suggests that a large proportion of chromosome 3 sequence resides in this small genetic interval. We are beginning to analyze this region first by correlating this region with the cytological map, and second by examining the sequence composition of the region.

Locations of breaks in RH lines were mapped relative to two translocations on chromosome 3. The translocation breakpoints in TB-3La and TB-3Sb have previously been placed on the Pioneer 1999 consensus map. This provided us with information that helped us develop markers to map the breakpoints on our RH lines. Five loci were selected that spanned a genetic interval where the two translocation breakpoints were expected to lie.

These loci and their map positions were *mmp69* (215.60), *mmp29* (228.50), *AY110403* (238.00), *AY110297* (244.70), and *AY110151* (254.60). Invader Assays were developed for these loci by Third Wave Technologies Inc. Invader Assays are quantitative assays that can be used to determine the copy number of a sequence. We have previously evaluated this assay by determining the copy number of individual *r1* transcription units in a series of *R-mb* derivative alleles that have one, two or three copies of the transcription unit (R. Okagaki, unpublished data). A sequence that was on the translocated portion of 3L would be present in three copies in line TB-3La and in two copies in line TB-3Sb. A sequence that was present in two copies in lines TB-3La and TB-3Sb would not be present on either translocated arm. *AY110403* and *AY110297* were present in two copies in both TB-3La and TB-3Sb; therefore these two markers located at map positions 238.00 and 244.70 lay between the two translocation breakpoints. *mmp29* at map position 228.50 was present in three copies in TB-3Sb and two copies in TB-3La. This placed the breakpoint in TB-3Sb between *mmp29* and *AY110403*. Three copies of *AY110151* were detected in TB-La and two copies in TB-3Sb. The breakpoint in TB-3La lay between *AY110297* and *AY110151*. Data analysis for marker *mmp69* has not been completed.

Markers *mmp29*, *AY110403*, *AY110297*, and *AY110151* were then mapped on the RH lines. Assays were performed on up to 14 RH lines to determine the presence versus absence of each marker in a RH line. The bottom part of Figure 1 gives the mapping data from RH lines. The current set of RH lines divides

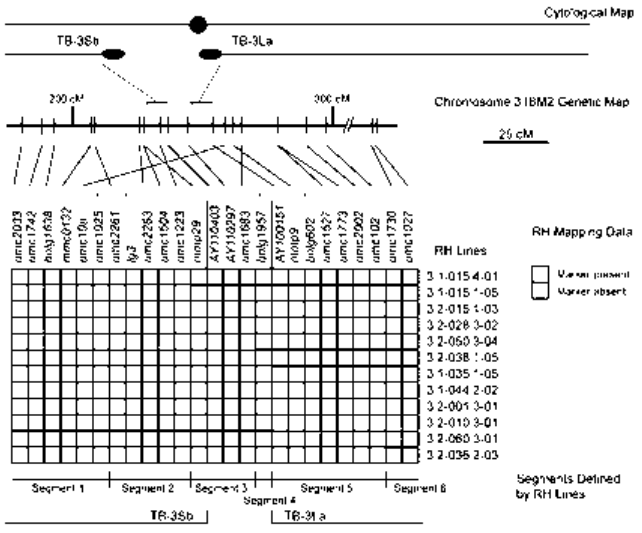

Figure 1. Comparison of genetic and radiation hybrid mapping with cytological landmarks

chromosome 3 into six segments. The TB-3Sb breakpoint is in segment 3, and the TB-3La breakpoint falls in segments 3, 4 or 5. The middle portion of Figure 1 shows the locations of markers on the IBM2 map. Positions for markers *umc10a, umc1223* and *umc1683* were inconsistent between the two maps. The discrepancy was small for *umc1223* and *umc1683*. Markers with an asterisk have not been placed on the IBM2 map. The top portion of Figure 1 depicts the two TB translocations used in this work and chromosome 3. The arm ratio of chromosome 3 is 2.0, and the region between the two translocation breakpoints is 20 to 25% of the chromosome length. The combined data show that the majority of chromosome breaks in our material occur in a region bounded by, or close to, the region defined by TB-3Sb and TB-3La.

Data from RH lines and cytological work suggests that this region of chromosome 3 accounts for much of the sequence on the chromosome while contributing little to the length of the genetic map. It seems possible that this region is largely composed of repetitive sequences. We are currently investigating this question. One preliminary Southern blot experiment has been conducted to compare the amount of the retroelement *Grande1* in different RH lines. The signal intensity from line 3.1-015.4-01, which carries the long arm plus a proximal segment of 3S, was approximately twice that of line 3.1-035.1-05, which carries only long arm sequences. There are control experiments that must be completed, and additional work with other repetitive sequences remains to be done. However, if the initial results are supported, then a large fraction of the repetitive sequences along chromosome 3 are clustered in one region of the chromosome.

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Sequence comparisons of six mitochondrial genomes from maize and teosinte

--Fauron, C, Minx, P, Gibson, M, Allen, J, Clifton, SW, Newton, K

Plant mitochondrial genomes are unusual in their diversity of structure and rapidity of change. Previously, only six mitochondrial genomes (mtDNA) had been completely sequenced: the liverwort, *Marchantia polymorpha*, three dicots, *Arabidopsis thaliana*, *Beta vulgaris*, *Brassica napus,* and a monocot, *Oryza sativa*. We have sequenced another six monocots within the genus *Zea* including fertile (NB and NA genotypes) and male sterile (cms-T and cms-C genotypes) maize and two teosintes from section Luxuriantes, *Zea luxurians* and *Zea perennis*. In addition, the mtDNA sequences of a related grass, *Sorghum bicolor*, is complete but still duplications have not been fully defined.

The sequencing of the *Zea mays* mitochondrial genomes from NB (GenBank Accession AY506529), NA and cms-C generated circular maps of 569,630, 701,046 and 739,719 base pairs respectively; *Zea luxurians* and *Zea perennis* mtDNAs also generate circular maps which are 539,347 and 570,353 bp long respectively. Sequencing of cms-T is 95% complete. Sequence comparisons reveal major rearrangements, large duplications, various foreign DNA insertions, but few differences in gene content.

This work has been supported by National Science Foundation grant DBI-0110168.

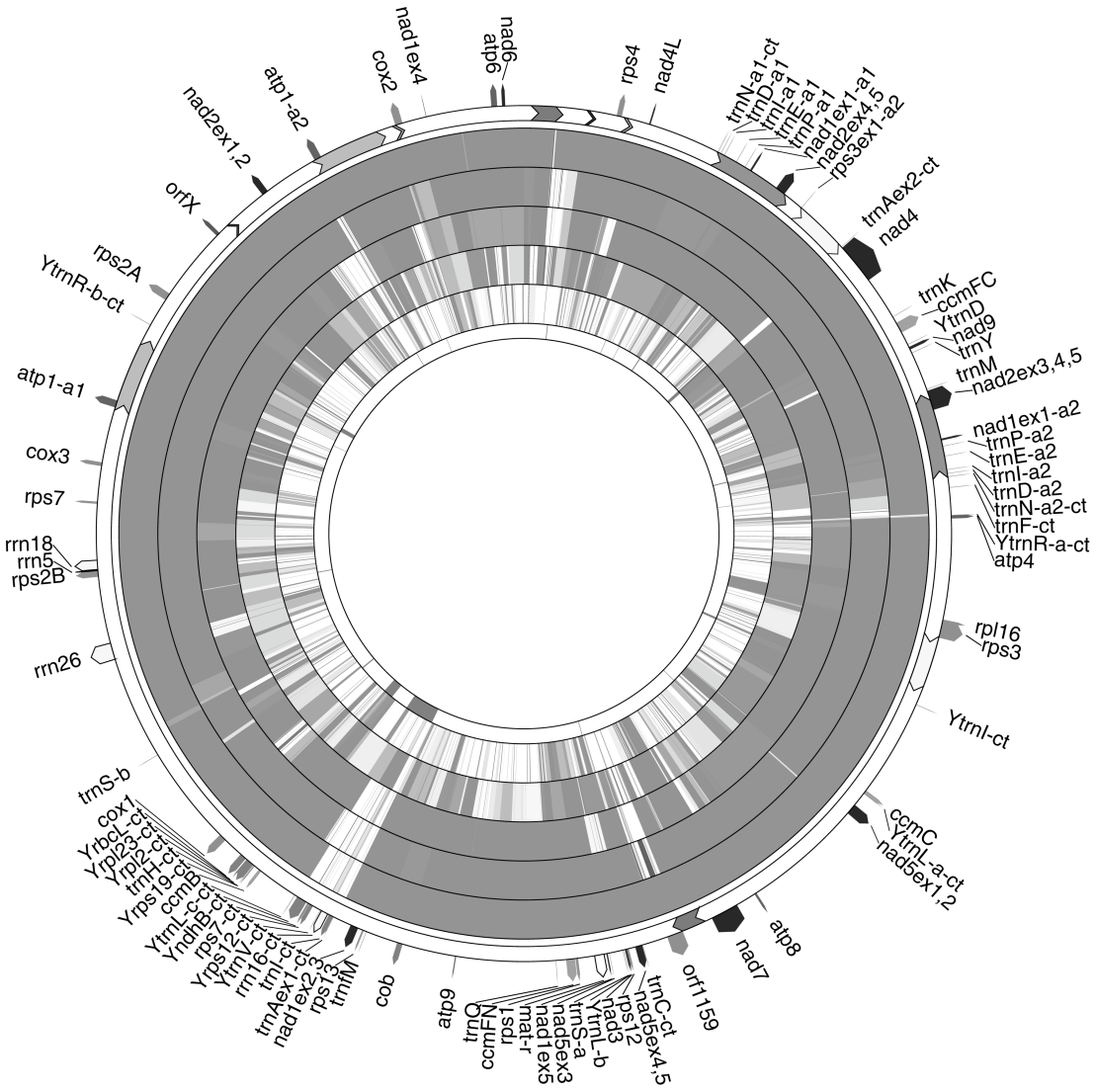

Figure 1. **Comparison of sequence similarity between *Zea mays* cytotype NB mitochondrial genome and other *Zea* mitochondrial genomes.**

The outer circle shows genes of *Zea mays mays* NB mtDNA and the outer ring contains NB mtDNA large repeats (500bp long and higher). Going towards the center of the circle the rings respectively represent NA mtDNA, CMS-C mtDNA, CMS-T mtDNA, *Zea perennis* mtDNA, *Sorghum bicolor* mtDNA. The inner rings shows regions of chloroplast homology to NB mtDNA with matches at least 80% identity and lengths of at least 100 bp. The gray scale indicates percent of similarity: from black (100% similarity) to white (below 60% similarity) identified from a BLASTN search (WashU BLAST 2.0) comparing each genome with the maize NB genome.

Table 1. List of known genes from maize NB mitochondrial genome also located on Figure 1.

Start	End	Gene	Product
19813	20889	rps4	ribosomal protein S4
26910	27212	nad4L	NADH dehydrogenase subunit 4L
42663	42734	trnN-a1-ct	tRNA-Asn
43435	43362	trnD-a1	tRNA-Asp
44163	44236	trnI-a1	tRNA-Ile
47148	47077	trnE-a1	tRNA-Glu
49198	49272	trnP-a1	tRNA-Pro
50490	50875	nad1 ex1-a1	exon 1 of NADH dehydrogenase subunit 1
59338	57194	nad2 ex4, 5-a1	exons 4 and 5 of NADH dehydrogenase subunit 2
64041	63968	rps3 ex1-a2	exon 1 of ribosomal protein S3
75891	75858	trnA ex2-ct	exon 2 of tRNA-Ala
84976	76135	nad4	NADH dehydrogenase subunit 4
94329	94257	trnK	tRNA-Lys
95774	98073	ccmFC	cytochrome c biogenesis FC
100905	100978	ΨtrnD	pseudo tRNA-Asp
101544	102116	nad9	NADH dehydrogenase subunit 9
102895	102977	trnY	tRNA-Tyr
111207	111278	trnM	tRNA-Met
111614	115826	nad2 ex3, 4, 5	exons 3, 4 and 5 of NADH dehydrogenase subunit 2
122530	122145	nad1 ex1-a2	exon 1 of NADH dehydrogenase subunit 1
123822	123748	trnP-a2	tRNA-Pro
125872	125943	trnE-a2	tRNA-Glu
128857	128784	trnI-a2	tRNA-Ileu
129585	129658	trnD-a2	tRNA-Asp
130357	130286	trnN-a2-ct	tRNA-Asn
132679	132607	trnF-ct	tRNA-Phe
138560	138489	ΨtrnR-ct	pseudo tRNA-Arg
139212	138553	atp4	ATPase subunit 4
161291	160734	rpl16	ribosomal protein L16
164661	161140	rps3	ribosomal protein S3
179691	179618	ΨtrnI-ct	pseudo tRNA-Ileu
200741	201463	ccmC	cytochrome c biogenesis C
201500	201584	trnL-a-ct	pseudo tRNA-Leu
204632	206947	nad5 ex1, 2	exons 1 and 2 of NADH dehydrogenase subunit 5
233200	232739	atp8	ATPase subunit 8
243574	237577	nad7	NADH dehydrogenase subunit 7
250363	253842	orf1159	known as orf1 in S2 plasmid
259354	259284	trnC-ct	tRNA-Cys
260942	259447	nad5 ex4, 5	exons 4 and 5 of NADH dehydrogenase subunit 5
262761	262384	rps12	ribosomal protein S12
263162	262806	nad3	NADH dehydrogenase subunit 3
263358	263239	ΨtrnL-b	pseudo tRNA-Leu
263694	263607	trnS-a	tRNA-Ser
266615	266594	nad5 ex3	exon 3 of NADH dehydrogenase subunit 5
267232	266974	nad1 ex5	exon 5 of NADH dehydrogenase subunit 1
269763	267745	mat-r	maturase
273846	273328	rps1	ribosomal protein S1
275849	273987	ccmFN	cytochrome c biogenesis FN
278064	277993	trnQ	tRNA-Gln
299462	299238	atp9	ATPase subunit 9
310906	312072	cob	apocytochrome b
318974	319047	trnfM	tRNA-fMet
319613	319963	rps13	ribosomal protein S13
320929	322595	nad1 ex2, 3	exons 2 and 3 of NADH dehydrogenase subunit 1
326638	326601	trnA ex1-ct	exon 1 of tRNA-Ala
327726	326704	trnI-ct	chloroplast tRNA-Ile
328030	329520	rrn16-ct	16S ribosomal RNA
329822	329751	ΨtrnV-ct	pseudo tRNA-Val
331579	332379	Ψrps12 ex2, 3-ct	Pseudo rps12
332438	332908	rps7-ct	chloroplast ribosomal protein S7
333208	335444	ndhB-ct	chloroplast NADH dehydrogenase subunit 2
336020	336100	ΨtrnL-c-ct	pseudo tRNA-Leu
346739	346550	Yrps19-ct	pseudo ribosomal protein rps19
344813	345433	ccmB	cytochrome c biogenesis B
346870	346943	trnH-ct	tRNA-His
348473	347007	Ψrpl2-ct	pseudo ribosomal protein L2
348769	348494	Ψrpl23-ct	pseudo ribosomal protein L23
350442	349025	ΨrbcL-ct	pseudo ribulose biphosphate carboxylase
354865	356451	cox1	cytochrome c oxidase subunit 1
378035	377949	trnS-b	tRNA-Ser
400215	403766	rrn26	26S ribosomal RNA
419314	418064	rps2B	ribosomal protein S2
419508	419383	rrn5	5S ribosomal RNA
421587	419620	rrn18	18S ribosomal RNA
433538	433984	rps7	ribosomal protein S7
442367	441570	cox3	cytochrome c oxidase subunit 3
455877	454351	atp1-a1	ATPase subunit 1
473442	473369	ΨtrnR-b-ct	pseudo tRNA-Arg
481148	479553	rps2A	ribosomal protein S2
497812	496997	orfX	homologous to mtt gene
511384	510018	nad2 ex1, 2	exon 1 and 2 of NADH dehydrogenase subunit 2
524814	523288	atp1-a2	ATPase subunit 1
541960	543536	cox2	cytochrome c oxidase subunit 2
548714	548772	nad1 e 4	exon 4 of NADH dehydrogenase subunit 1
562726	563958	atp6	ATPase subunit 6
565003	565665	nad6	NADH dehydrogenase subunit 6

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Progress of maize breeding and production increase in North Korea

--Kim, SK, Han, HJ, Kim, P

Maize is a staple food crop in North Korea, along with rice. It is grown on approximately 700,000ha, with about 200,000ha being on the hilly side, and rice is grown on 600,000ha. Seventy percent of the population of 23 million North Koreans depends on maize as the main food. While South Korea, with a population of 47 million, is the second largest importer of maize in the world after Japan. Historic floods in 1995 and 1996, and hail and drought in 1997, along with economic difficulties, caused the country of North Korea to be at the center of world concern. When the country had severe economic difficulties, scientists of South and North Korea, with strong support from both governments, and Koreans inside and outside of Korea, agreed to increase maize production in the shortest possible time, and also develop better high yielding and more stable maize cultivars for the country. The major constraints in maize production were lack of fertilizers and high quality F1 seeds, and the solid cropping of maize with legumes. The North and South Korean team collaborating on super-maize research has taken two prolonged approaches: increase grain production with the hybrid Suwon 19, which was developed in South Korea in 1976 by the senior author after his education in the USA, and initiate an extensive maize breeding program nationwide. The results of six years of the collaborative works (1998-2003) show that North Korea has very favorable environments for maize cultivation. The total production of maize and other foods (rice and potato) has increased from two million tons to four million tons within the project period. Purity improvement of the F1 hybrid, intercropping of soybean into maize fields, reduction of the population density of maize plants, and increased availability of fertilizers were aided by the government of South Korea as well as by NGOs. The increase in total production was also influenced by the promotion of disease free potato seeds and the advancement of agriculture as the No.1 policy of the government, as well as the introduction of the bonus system to encourage more cooperative farms. Although the total production has been doubled, it is still short one million tons for the population size. Approximately half of the 3000 cooperative farms have

used Suwon 19 for grain production. The collaborative team has tested a total of 31,000 crosses at 25 stations in North Korea and 7 in South Korea. Ten outstanding hybrids have been selected from the on-farm testings. F1 seed production for larger scale testings has been carried out. The results of genetic studies for tolerance of new maize inbreds to major biotic (*Exserohilum turcicum*, stalk rots, stem borers) and abiotic stresses (drought and N use efficiency) will increase the stability of maize.

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Research not done; ideas left over

--Chase, SS

INBREDS FROM MEIOCYTES: As a corn breeder, have you ever seen a super plant and wished you had the inbred lines to reproduce it? I'll tell you what to do; and you can figure out how. Take a meiocyte, in the male or female line; completely inhibit crossing-over; separate the sister cells of Division I, one from the other keeping both; culture each cell of each pair separately to produce an embryo or set of embryos; grow these to maturity. Each will be a homozygous diploid; each crossed with its mate will reproduce the elite genotype. You can have dozens of complementary pairs of inbreds for each original elite individual or hybrid!

PARTHENOGENESIS: What happens to the extra male sperm in maize parthenogenesis? It does not go to fuse with the polar nuclei and the other sperm nucleus--the endosperm is triploid, not tetraploid. Is it left totally out of the system? I think it likely that it goes to the suspensor. Parthenogenetic rates increase when pollination is delayed. Perhaps the sperm enters the egg after the egg has achieved readiness for division. And the egg divides before syngamy can be effected. This could also account for the fate of the female nucleus in cases of 'androgenesis'.

DIPLOIDY AND ILLEGITIMATE CROSSING-OVER: In the evolution of control of DNA by the organism, the step that made maize heterosis possible (and also diploidy and higher plants and animals) was prevention of crossing-over of homologous chromosomes at all somatic and mitotic stages of the sporophyte; crossing-over is limited to meiosis. 'Illegitimate' crossing-over can occasionally occur. Can it be forced and used as a breeder's tool? Illegitimate crossing-over leads to chimeral homozgosity.

TEOSINTE AS FOOD PLANT: Teosinte, ancestor of maize, is an unlikely food grain as is. Perhaps the hydroxide treatment of maize grain was invented for application to teosinte grain before maize evolved. Both maize and tortillas originated in Meso-America. Perhaps the tortilla came first.

Twins, pseudo-twins, and other seedling and kernel abnormalities in maize: a long term record

--Chase, SS; Kernan, P

In the search for parthenogenetic and androgenetic seedlings, starting at Ames in 1946 and continuing at DeKalb, many thousands of thousands of seedlings were examined by the senior reporter and colleagues. Many unusual individuals were preserved for drawing. Presented here are drawings, some representative of their class, a few directly depicting individuals. The frequency of 'haploidy' in maize, averaged among progenies, is about one per thousand seedlings. Many of the abnormalities reported here, found in progenies of normal maize (excluded here are abnormalities following mutagenic treatments or from known aberrant stocks), were less frequent; some were very rare indeed.

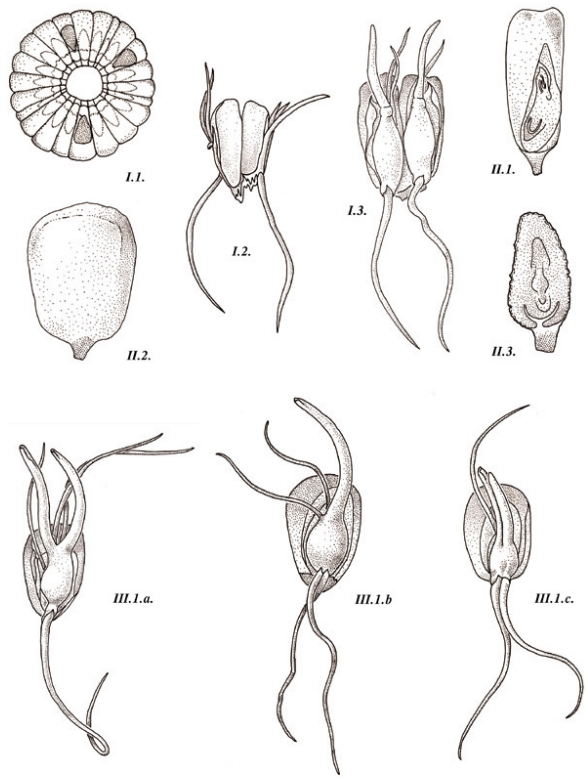

Fig I. Up-side-down embryo; faces base of ear. Common in Country Gentleman Sweetcorn; occasional generally. From second, usually sterile floret of pair.

Figs I.2&3 Back-to-back; and, side-by-side kernels and seedlings. Common in Country Gentleman Sweetcorn; occasional generally.

Fig II.1 Reversed embryo. Rare; a lethal. Embryo faces inwards. Can be 'rescued' by excision.

Fig II.2 Embryoless or germless. Occasional in some strains. Endosperm appears normal.

Fig II.3 Endospermless. Occasional in some strains; a lethal. Embryo can be 'rescued.'

(Not shown) Heterofertilized kernels. First noted by Sprague. Occasional to frequent in some strains.

(Not shown) Kernel with embryos derived from twin embryo sacs. Observed only once.

Fig III.1.a Pseudo-twin. Seedlings forked at scutellar node, with two (mirror image) plumules. Occasional.

Fig III.1.b Pseudo-twin. Seedling forked below scutellar node, with two radicles. Occasional.

Fig III.1.c Pseudo-twin. Seedling with double plumules and double radicles. Rare.

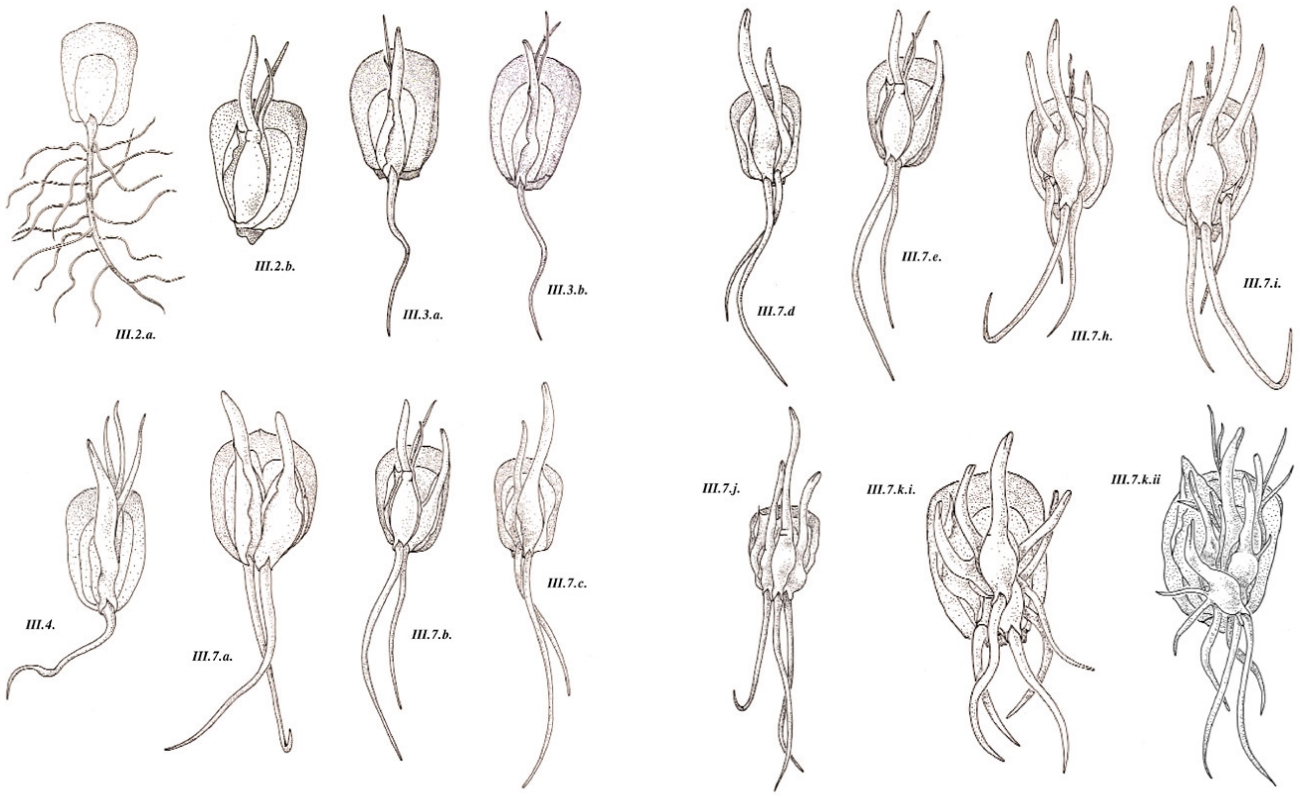

Fig III.2.a Plumuleless. Root system only developing. Rare. (See: Evans & Kermicle, 2001).

Fig III.2.b Rootless. Plumule and scutellar node only developing. Rare. (Evans & Kermicle) Occasionally caused by insect damage. Seedling can be 'rescued.'

Fig III.3.a Parthenogenetic monoploid (haploid). Frequency varies widely with genotype of pollinator and female parent (averaging about 1/1,000).

Fig III.3.b 'Androgenetic' or 'paternal' monoploid (haploid). Very rare in most genotypes (about 1/60,000). The terms 'androgenetic' and 'paternal' are qualified as the cytoplasm of these plants is derived from the female parent.

(Not shown) Parthenogenetic diploid. Originates by early chromosome doubling of a maternal monoploid or by derivation from an unreduced egg cell.

(Not shown) Parthenogenetic diploid. From a tetraploid parent (See: Randolph).

Fig III.4 Triploid. Occasional. (A few triploid plants can generally be found in any field of corn.) Formed by fertilization of an egg by an unreduced sperm; or by fertilization of an unreduced egg by a normal sperm.

(Not shown) Viviparous seedlings. Seedlings sprouting on ear; no dormancy.

Fig III.7.a Diploid/diploid twin. Rare.

Fig III.7.b Diploid/maternal monoploid twin. Rare.

Fig III.7.c Diploid/'paternal' monoploid twin. Very rare.

Fig III.7.d Triploid/maternal monoploid twin. Very rare indeed.

Fig III.7.e Maternal monoploid/maternal monoploid twin. Rare.

(Not shown) Maternal monoploid/'paternal' monoploid twin. Very rare indeed. (Gerrish, 1956)

(Not shown) Diploid/'paternal' monoploid twin. Very rare.

Fig III.7.h Maternal monoploid/diploid/maternal monoploid triplet. Very rare indeed.

Fig III.7.i Maternal monoploid/diploid/'paternal' monoploid triplet. Very rare indeed.

Fig III.7.j 'Paternal' monoploid/ diploid/'paternal' monoploid triplet. Seen once by Rhoades, once by Chase; also seen, as also many of the above twins and triplets, in 'ig' progeny.

Figs III.7.k.i&ii. Kernels with polyembryos. Two kernels seen; drawn here.

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The usefulness of multiple index improvement of a diallel of (7x6) corn lines

--Paraschivu, I, Aurelian, M, Paraschivu, M, Bonea, D, Gavrilescu, L, Matei, G

The research was done in the irrigated field of the corn improvement lab on a brown-red soil from S.C.D.A., Simnic-Craiova, Romania. Our objective was to characterize inheritance of rot resistance of the corn stalk and of senescence in a diallel of (7x6) corn lines. The experiment lasted three years; we used artificial inoculation with Fusarium moniliforme, the fungus which is most prevalent in the southern part of Romania and which causes stalk rot.

Table 1. The F1 hybrid classification using multiple index values.

No.	Combination	The complex index classification Production x Mechanical resistance	Average values Stalk rot resistance 1-9 (FAO)	Classification	Average values Senescence 1-9 (FAO)	Classification	The multiple index values 2x4x6	Classification
0	1	2	3	4	5	6	7	8
1	A 239 x A632	1	5.2	7	3.2	9	63	5
2	A 239x CH 539-9	24	6.0	13	3.1	8	2496	22
3	A 239x ND 481	14	6.1	14	5.3	23	4508	27
4	A 239x Oh 561	11	4.8	4	2.7	4	176	8
5	A 239x A 619	38	5.2	7	2.9	6	1596	19
6	A 239x CH 592-13-2	10	4.3	1	2.7	4	40	3
7	A632x A 239	13	6.0	13	3.7	14	2366	21
8	A632xCH539-9	5	5.9	12	4.0	16	960	17
9	A632x ND 481 000	30	6.3	16	5.7	26	12480	38
10	A632x Oh 561	6	5.4	8	3.4	11	528	13
11	A632x A 619	20	5.5	9	3.4	11	1980	20
12	A632x CH 592-13-2	27	5.7	11	3.7	14	4158	26
13	CH 539-9x A 239	25	5.1	6	2.8	5	750	16
14	CH 539-9x A632	21	4.8	4	2.6	3	252	10
15	CH 539-9x ND 481	26	6.3	16	5.4	24	9984	32
16	CH 539-9x Oh 561	4	5.4	8	3.1	8	256	11
17	CH 539-9x A 619 000	39	6.3	16	4.3	19	11856	36
18	CH 539-9x CH 592-13-2	35	5.4	8	3.5	12	3360	24
19	ND 481x A 239	31	6.0	13	4.3	19	7657	30
20	ND481xA632 **000**	34	6.2	15	5.1	22	11220	35
21	ND 481xCH 539-9 **000**	32	6.8	19	4.7	21	12768	39
22	ND 481xOh 561	29	6.6	18	4.5	20	10440	33
23	ND 481x A 619 000	42	7.2	21	6.3	28	24696	42
24	ND 481x CH 592-13-2	22	5.7	11	5.1	22	5324	29
25	Oh 561x A 239 ***46.7**	2	5.5	9	2.6	3	54	4
26	Oh 561x A632	12	6.1	14	3.8	15	2250	23
27	Oh 561x CH 539-9	17	4.9	5	3.0	7	5950	15
28	Oh 561x ND 481	19	5.6	10	4.6	21	3390	25
29	Oh 561x A 619	9	5.6	10	2.9	6	540	14
30	Oh 561x CH 592-13-2	18	6.6	3	2.5	2	108	7
31	A 619 x A 239 ***38.3**	3	4.8	4	2.3	1	12	1
32	A 619x A632	15	5.2	7	2.5	2	210	9
33	A 619x CH 539-9	33	5.9	12	3.6	13	5148	28
34	A 619x ND 481 **000**	36	7.2	21	6.1	27	20414	41
35	A 619x Oh 561	8	4.8	4	2.5	2	64	6
36	A 619x CH 592-13-2	37	6.1	14	4.1	17	8806	31
37	CH 592-13-2xA239 ***33.6**	16	4.4	2	2.3	1	32	2
38	CH 592-13-2xA632	23	5.1	6	3.1	8	1104	18
39	CH 592-13-2x	41	6.2	15	4.1	17	10445	34
40	CH 592-13-2xND481;**000**	28	6.9	20	5.6	25	14000	40
41	CH 592-13-2 x Oh 561	7	5.2	7	3.3	10	490	12
42	CH 592-13-2x A 619; **000**	40	6.4	14	4.2	18	12240	37

We selected 7 corn lines with diverse resistance to Fusarium stalk rot. The resistance scoring used the following notation scale (Figure 1).

Figure 1. *Fusarium* stalk rot is scored using the FAO 1-9 scale. Senescence is scored using the FAO 1-9 scale.

The best resistance to Fusarium stalk rot was found in the hybrids A239 x CH593-13-2 and CH593-3-2 x A239 (direct and reciprocal hybrid) with the average values equal to 4.3 and 4.4, and multiple index values equal to 40 and 32. Sensitivity to stalk rot and senescence is transmitted by ND-481. All hybrid combinations of this line (ND 481) have the highest multiple index values (Table 1).

The best resistance to senescence was found in the hybrids A619 x A239 and CH593-13-2 x A239, with an average value equal to 2.3, and multiple index values of 12 and 32 (Table 1).

Effect of different moisture conditions on total chlorophyll content

--Gavrilescu, L, Bonea, D, Dobre, M, Paraschivu, M

Within the Southern Zone of Oltenia, only two years out of ten are favorable for the corn crop due to the higher temperatures and the lack of rainfall. Many other specialists in our country, as well as abroad (Morizet et al., 1990), have studied genetic and physiological drought tolerance (Terbea et al., 1994; Burzo et al., 1999).

This paper deals with the influence of plant density (40,000; 50,000; 60,000 plants/ha) and crop moisture conditions (irrigated and unirrigated) on the total chlorophyll content from leaves of 4 corn hybrids (F322, F376, Olt, Cocor). The experiment was performed at the Research Station of Simnic for three years. Climatically, 1999 was favorable for the corn crop, 2000 was extremely dry and 2001 was intermediate. Results are averages for the three years of the experiment.

Our results show:

(1) Highest total chlorophyll content was found for the irrigated plants (Figures 1-4).

(2) Regarding total chlorophyll content, the best results were given by the F376 (irrigated) hybrid, followed by Cocor.

(3) The best results for drought tolerance and total chlorophyll content combined were given by the F322 hybrid (unirrigated).

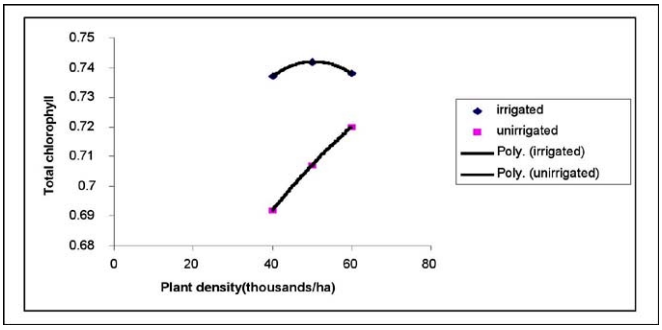

Figure 1. Total chlorophyll content for the F322 corn hybrid.

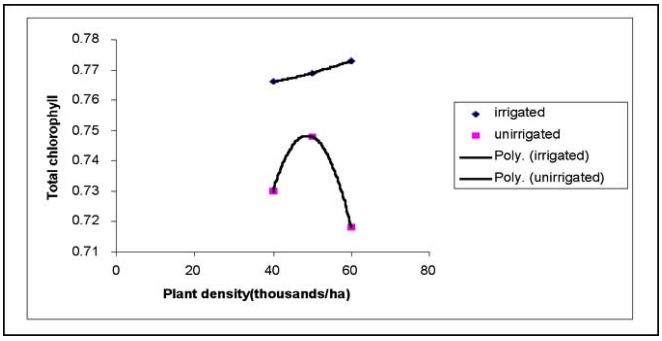

Figure 2. Total chlorophyll content for the F376 corn hybrid.

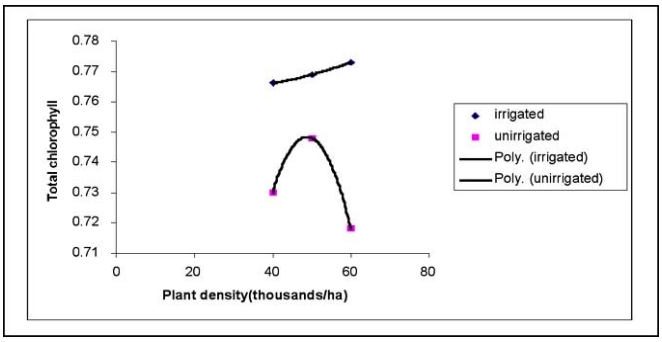

Figure 3. Total chlorophyll content for the Olt corn hybrid.

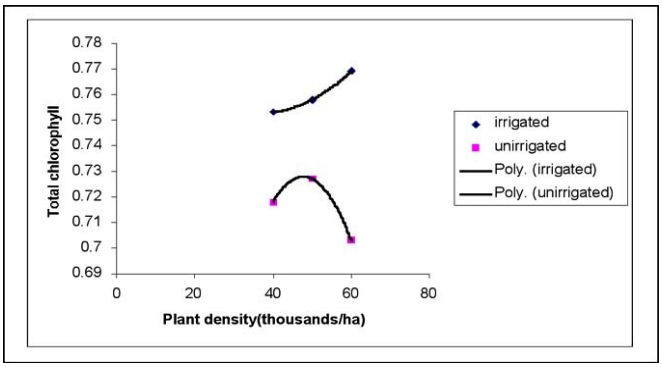

Figure 4. Total chlorophyll content for the Cocor corn hybrid.

The optimal density for the highest values of total chlorophyll in irrigated plants is between 50,000 and 60,000 plants/ha. In unirrigated plants, the maximum total chlorophyll was recorded for a density of 50,000 plants/ha. It was also observed that a density of 60,000 plants/ha caused a more intense shadowing of neighboring plants, resulting in the lower photosynthetic absorption emphasized in the graphics.

The influence of low temperatures on seed germination

--Bonea, D, Urechean, V, Paraschivu, M

The study of the local germplasm is imposed by the necessity of creating productive maize hybrids with resistance to low temperatures, with a view to selecting tolerant parental forms.

The tolerance of some local populations from the Research Station in the Simnic-Craiova region to low temperatures was characterized for effect of cold on seed germination.

For those ten local populations tested for their reaction at low temperatures, the rate of seed germination in three conditions has been established: (1) the control variant – temperatures of 23±1 C, in laboratory conditions; (2) low constant temperatures for seven days, in a refrigerator, at 4±1 C; (3) alternating temperatures of 12 hours at temperatures of 23±1 and 12 hours at temperatures of 4±1 C.

Seeds were germinated on paper filter in Petri plates under the treatments (Table 1).

Table 1. The influence of temperature on the rate of germination in local populations.

Treatment	Population	Germination		s
		%	± d	
a_0 (control)	b_1	58.3	-26.7	000
	b_2	90	+5	
	b_3	96.6	+11.6	*
	b_4	86.6	+1.6	
	b_5	98.3	+13.3	*
	b_6	81.6	-3.4	
	b_7	75	-10	
	b_8-contr.	85		
	b_9	88.3	+3.3	
	b_{10}	93.3	+8.3	
a_1 (low constant temperature)	b_1	45	-30	000
	b_2	70	-5	
	b_3	91.6	+16.6	**
	b_4	80	+5	
	b_5	96.6	+21.6	***
	b_6	63.3	-11.7	0
	b_7	63.3	-11.7	0
	b_8-contr.	75		
	b_9	56.6	-18.4	000
	b_{10}	91.6	+16.6	**
a_2 (alternating temperatures)	b_1	60	-26.6	000
	b_2	88.3	+1.7	
	b_3	95	+8.4	
	b_4	86.6	0	
	b_5	76.6	+10	
	b_6	85	-1.6	
	b_7	78.3	-8.3	
	b_8-contr.	86.6		
	b_9	75	-11.6	0
	b_{10}	90	+3.4	

LD5%=10.36; LD1%=13.82; LD0.1%=18

The influence of the planting density and soil moisture on the foliar surface index and the net rate of assimilation

--Gavrilescu, L, Bonea, D, Dobre, M, Paraschivu, M

Former research carried out at the Research Station of Simnic have shown that planting density is the major factor that influences the physiological processes which, in turn, determine the level of the yields.

The present paper deals with four corn hybrids (F322, F376, Olt, Cocor) planted at different densities (40,000; 50,000; 60,000) and in two soil moisture conditions (irrigated and unirrigated) within the 1999-2001 period, on a luvic brown-reddish soil with a 5.8 pH. Climatically, 1999 was relatively favorable for the corn crop, and 2000 was extremely dry, with a large soil water deficit and high air temperatures. In 2001, a soil moisture deficit was recorded beginning with the end of July and August, as well as very high air temperatures.

The foliar surface of each plant (FSI) was calculated per square meter of soil and as average values over three years.

The study carried out with the irrigated crop (Fig. 1) emphasizes the role of water for optimal physiological processes. There was a 58% increase in the FSI with the F322 hybrid at a 50,000 plant density in comparison with a 40,000 plant density, followed by the Olt hybrid which, with the same plant density, had a 39% increase in comparison with the minimum density (40,000). The F376 and Cocor hybrids have a FSI of 25-26% with the maximum density in comparison with the minimum density.

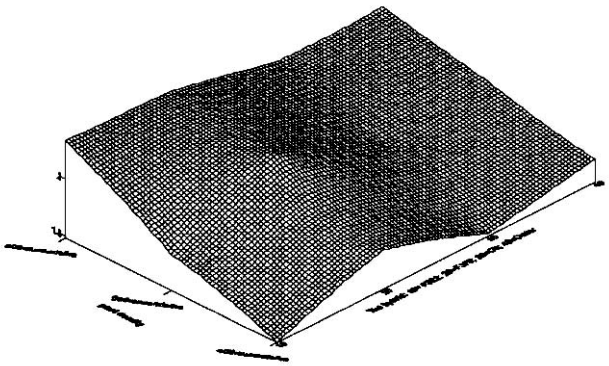

Figure 1. The FSI in function of the plant density and corn hybrid with irrigated plot

With the unirrigated crop (Fig. 2) the FSI values range between 1.16-1.68 m^2/m^2 with the F322 hybrid (49%); between 1.64 – 2.02 m^2/m^2 with F376 (23%); between 1.25 – 1.72 m^2/m^2 with Olt (37%) and between 1.46 – 1.89 m^2/m^2 with the Cocor hybrid (29%).

It can be noted that there is a difference between irrigated and unirrigated, as well as between densities, of approximately 14% with F322, 3% with F376 and 2% with Olt.

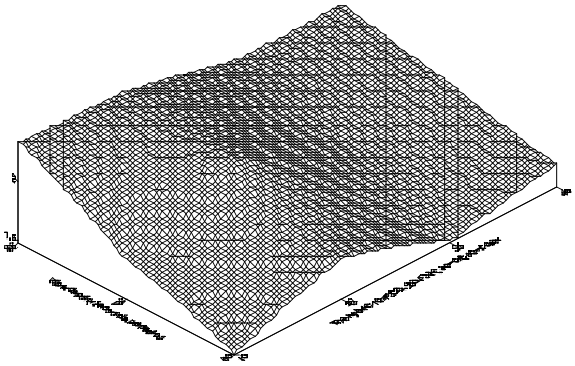

Figure 2. The FSI in function of the plant density and corn hybrid with unirrigated plot

We conclude that (1) The FSI increases with the plant density, the maximum value being recorded with the maximum density (60,000), both with irrigated and unirrigated plants; (2) With regard to the ISF, the best results were given by F322 (irrigated) and F376 (urirrigated).

TALLAHASSEE, FLORIDA
Florida State University

The *Single myb histone* (*Smh*) gene family of maize; detection of two PCR products from maize with primers for *Smh1*

--Figueroa, DM, Bass, HW

The *Single myb histone* (*Smh*) genes of maize make up a small gene family that was discovered as part of a screening for telomeric protein genes (Marian et al., Plant Physiol 133:1336-1350, 2003). The SMH proteins may be unique to higher plants. We have classified them as having a unique triple-motif protein organization – an N-terminal single MYB-like/SANT domain, a centrally located motif homologous to the globular domain of the linker histone (H1/H5), and a near-C-terminal coiled-coil domain. The presence and arrangement of this triple-motif are diagnostic for SMH proteins. GenBank accession numbers and ChromDB synonyms for the maize *Smh* genes are summarized in Table 1. We found a similar *Smh* gene family in *Arabidopsis* (SMH gene family page at TAIR, www.arabidopsis.org).

Table 1. Maize *Smh* genes.

cDNA	GenBank	ChromDB synonyms (at www.chromdb.org)
Smh1	AY271659	SMH101, HON107
Smh2		SMH102, hon112
Smh3	AY280629	SMH103
Smh4	AY280631	SMH104, HON108
Smh5	AY280630	SMH105
Smh6 AY280632	SMH106	

In addition to the five genes for which full-length cDNAs had been obtained, we found evidence for a sixth *Smh* gene by PCR analysis with primers for *Smh1*. The six *Smh* loci were numbered in sequential pairs to reflect our idea that they represent three closely related pairs of genes – *Smh1/2*, *Smh3/4*, and *Smh5/6* as described by Marian et al. (2003). The pairs are presumed to be duplicate but unlinked. *Smh1/2* and *Smh5/6* are phylogenetically more closely related to each other than to *Smh3/4*. *Smh3/4* show the most frequent hits in BLAST searches of ESTs.

PCR primers designed to detect *Smh1* but not *Smh3-6* amplified two products using inbred lines B73 or Mo17 (see Fig 1A & GenBank sequences AY328854 for B73 Smh1-STS and AY328855 for B73 Smh2-STS). The two bands of the STS doublet segregated independently in the IBM recombinant inbred lines and were mapped by Marian et al. (2003) using the IBM DNA mapping kit. The map position of *Smh2*-STS allowed for positional cloning of the *Smh2* gene.

The lack of a cDNA or EST corresponding to the *Smh2* locus initially indicated that the *Smh2*-STS might be from a non-functional gene or a rarely expressed gene. We checked a number of different maize lines to see how often the SMH1-57710/11 primer pair would produce the PCR product doublet. As shown in the Figure 1A-C, we found that all of the 11 lines of *Zea* sp.

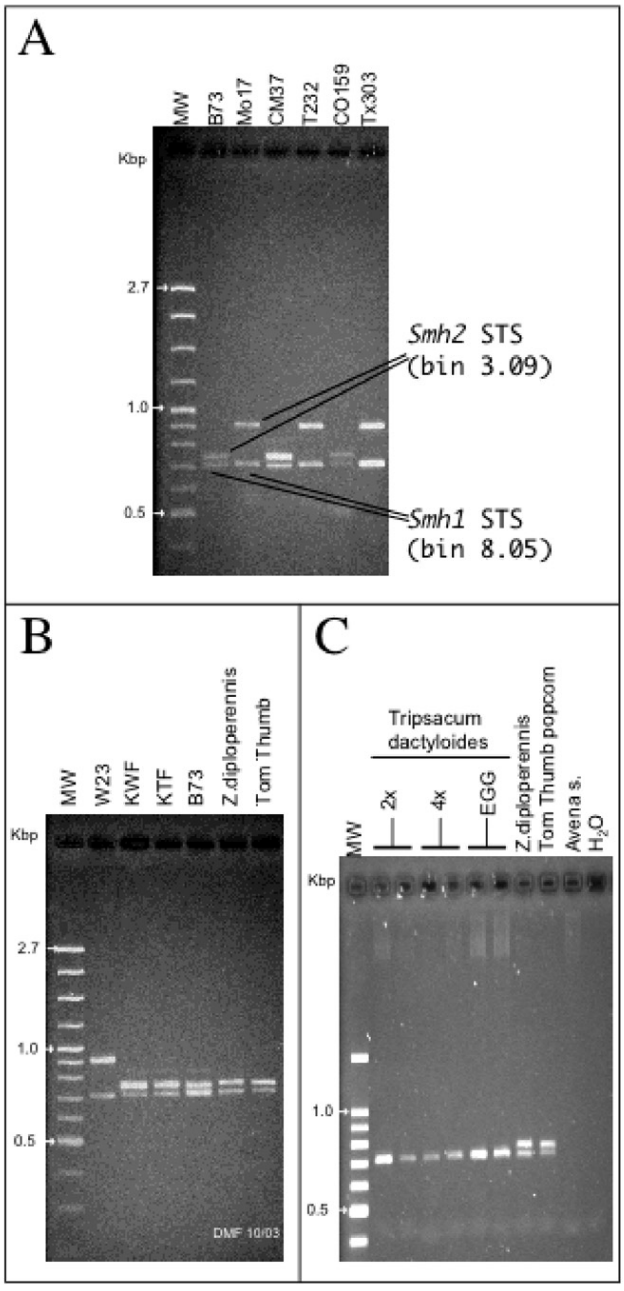

Figure 1.

tested produced two abundant PCR products (the lines are B73, Mo17, CM37, T232, CO159, TX303, W23, Knobless Tama Flint, Knobless Wilbur's Flint, *Zea diploperennis*, and Tom Thumb pop corn). In contrast, DNA from *Tripsacum dactyloides* produced a single band, whereas oat (Fig. 1C, *Avena sativa*) DNA and a no-template control reaction (Fig 1C, H2O) did not produce detectable PCR products. The single band from *Tripsacum* was amplified from leaf DNA isolated from three different sources of *T. dactyloides* (2x-diploid and 4x-tetraploid from M. Eubanks, and EGG-Eastern gamma grass from USDA). Further analysis of the *Tripsacum*-derived PCR products may help clarify whether *Tripsacum* has one or both members of the *Smh1/2* pair. A recent EST for a partial *Smh2* cDNA has been observed (see ChromDB SMH102/hon112), indicating that the *Smh2* locus may house a functional gene.

TALLAHASSEE, FLORIDA
Florida A & M University
Florida State University
ST. PAUL, MINNESOTA
University of Minnesota

FISH analysis of retroelement distribution patterns along mitotic chromosomes.

--Bassie, YR, Onokpise, OU, Odland, WE, Bass, HW

The distribution of retroelements was investigated by FISH analysis of mitotic chromosomes. PCR products specific for *prem2*, *opie2a*, and *zeon1* were cloned into plasmids and used to make FISH probes following the direct-labeled procedure for chromosome painting described by Koumbaris & Bass (Plant J. 35:647-659, 2003). The chromosomes were obtained as flow-sorted mitotic root-tip chromosomes (courtesy of Dr. Arumugunathan). The slides hybridized with *prem2* and *zeon1* contained maize chromosome 9 from an oat-x-maize addition line. The slide hybridized with *opie2a* contained total maize chromosomes from B73.

Each hybridization contained two fluorescent probes - an A488-labeled probe for the retroelement (either *prem2*, *opie2a*, or *zeon1*) and a centromere-specific oligonucleotide CentC probe, MCCY (Koumbaris & Bass, MNL 76, 2002). Following overnight hybridization, the slides were washed, DAPI-stained, and imaged by deconvolution microscopy. Representative FISH patterns are shown in Figure 1 (wavelengths = rows; retroelement experiments = columns). As described by Mroczeck & Dawe (Genetics 165:809, 2003), retroelements often appear under-represented at centromeres (c) and knobs (k) compared to DAPI-normalized bulk chromatin on meiotic chromosomes. This pattern (Fig 1; * indicates area of staining gap) is also clear on mitotic chromosomes hybridized with *prem2* or *opie2a*.

In contrast, the *zeon1* FISH staining pattern did not show a conspicuous reduction in signal intensity across the centromere of maize chromosome 9. The basis for these varied patterns remains unknown, but the *zeon1* family may prove informative on the relationship between heterochromatin and retroelement sequences in the maize genome. In addition, understanding the distribution of retroelement families should contribute to a better understanding of the structure and evolution of the maize genome.

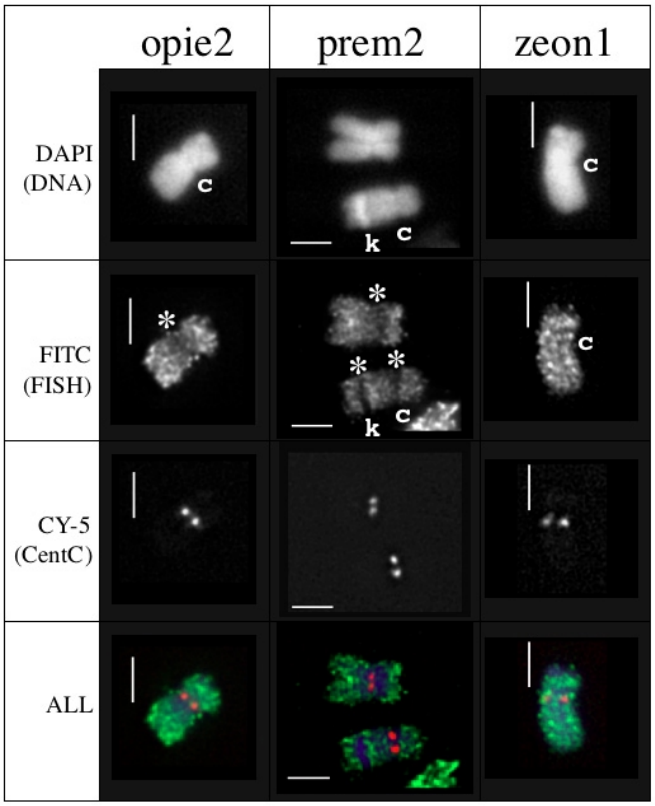

Figure 1.

This work was supported in part by a fellowship to YRB from the Consortium for Plant Biotechnology Research (CBPR).

TÜBINGEN, GERMANY
Eberhard-Karls-University

Isolation of a new root mutant *rum1* affected in lateral and seminal root initiation

--Woll, K, Hochholdinger, F

The early root system of maize consists of embryonically formed primary and seminal roots. Both root types form lateral roots a few days after emergence (Hochholdinger et al.: Trends Plant Sci., in press, 2004).

Two thousand *Mu*-tagged F_2-families of maize were visually screened for aberrant root phenotypes on 10-day-old seedlings which led to the identification of a new monogenic recessive mutant. The new root mutant *rum1* (*rootless with undetectable meristems 1*) is affected in lateral root initiation at the primary root and is completely devoid of seminal roots (Fig. 1). Histological analyses of cross sections obtained from embryos 30 days after pollination indicated that mutant in contrast to wild-type embryos do not develop seminal root primordia (Fig. 2). The previously described lateral root initiation mutant *lrt1* (Hochholdinger and Feix, Plant J. 16:247-255, 1998) does not initiate lateral roots at the primary and seminal roots and is affected in crown root formation at the coleoptilar node. In contrast to the phenotype of *lrt1*, the mutant *rum1* does not form seminal roots but normal crown roots at the coleoptilar node. Hence, the mutant *rum1* defines a new developmental window of root for-

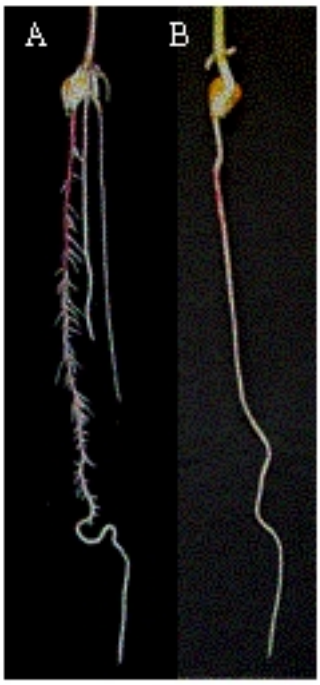

Figure 1. Wild-type (A) and *rum1* (B) seedlings 12 days after germination.

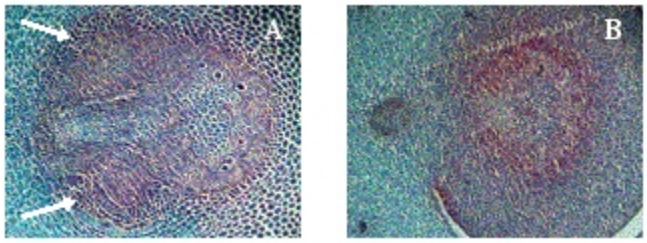

Figure 2. Cross section of embryos 30 days after pollination. Arrows in wild-type (A) indicate position of seminal root primordia which are missing in *rum1* (B).

mation comprising late embryogenesis (seminal roots) and early postembryonic root development (lateral roots on the primary root). A detailed molecular and genetic analysis of this new mutant is in progress.

URBANA, ILLINOIS
Maize Genetics Cooperation • Stock Center

The *Spotted-dilute* controlling element system revisited

--Stinard, PS

Sastry and Kurmi (MNL44:101-105, 1970) describe a mutable system at the *r1* locus involving three elements: the receptor haplotype *R1-r(sd2)* (*spotted dilute2*), the autonomous controlling element *Spf* (spotting factor), which elicits aleurone color mutability at *R1-r(sd2)*, and *Dil* (diluting factor), which suppresses background aleurone color of *R1-r(sd2)*. In the absence of *Spf* and *Dil*, kernels bearing the *R1-r(sd2)* haplotype are near full colored; in the presence of *Dil*, *R1-r(sd2)* kernels have pale aleurone color; in the presence of *Spf*, *R1-r(sd2)* kernels are dark with barely distinguishable darker sectors; and in the presence of both *Spf* and *Dil*, *R1-r(sd2)* kernels have dark sectors on a pale background. Full colored revertants of *R1-r(sd2)* can be isolated from mutable lines. The apparent progenitor haplotype of *R1-r(sd2)* is *R1-r(Cornell)* (Stadler and Emmerling, MNL 30:61-63, 1956), which may be identical to *R1-r(standard)*. Sastry and Kurmi performed tests indicating that *Spf* elicits mutability at *a2-m1::dSpm*. We performed additional tests (described elsewhere in this newsletter) that demonstrate that *Spf* and *En/Spm* are functionally equivalent at eliciting mutability at both *R1-r(sd2)* and various *dSpm/I* receptor alleles. We also demonstrated that *Dil* is very tightly linked, if not identical to, the *r1* haplotype-specific aleurone color inhibitor *Inr1* (also described elsewhere in this newsletter).

What sets the *Spotted-dilute* system apart from other controlling element systems are certain peculiar behaviors described by Sastry and Kurmi. They indicate that after *Spf* and *Dil* have segregated away from *R1-r(sd2)*, *R1-r(sd2)* is not capable of responding to either *Spf* or *Dil* once these factors have been reintroduced. However, as long as at least one of the two factors is present, *R1-r(sd2)* will also show a response to the other factor once it is reintroduced. They also indicate that *R1-ch* (most likely the *R1-ch(Stadler)* haplotype) responds to *Spf* and *Dil* regardless of whether either factor was previously present in the *R1-ch* line. We performed several experiments in order to investigate these unusual behaviors. We obtained two *R1-r(sd2) Dil Spf* lines from Jerry Kermicle, one with *R1-r(sd2)* linked to K10, and one with *R1-r(sd2)* residing on a normal chromosome 10. Both lines segregated for *R1-r(sd2)* (the other *r1* haplotype present was *r1-r* on N10), *Spf*, and *Dil*. From these lines, we isolated three homozygous *r1-r* N10 lines: one homozygous for both *Spf* and *Dil*, one segregating for *Spf* without *Dil*, and one homozygous for *Dil* and segregating for *Spf*. A W22 *r1-g(Stadler)* line without *Spf* and *Dil* was used as a control. We also developed the following *R1-r(sd2)* testers: *R1-r(sd2)* N10 with *Dil*, *R1-r(sd2)* N10 without *Dil*, and *R1-r(sd2)* K10 without *Dil*. Crosses were made of the *r1-r* lines onto the *R1-r(sd2)* testers and onto *R1-ch(Stadler)* in various combinations, and the data obtained are summarized in Table 1.

Some of our results conflict with those of Sastry and Kurmi. Crosses of *r1 Dil* (no *Spf*) onto the *R1-r(sd2)* N10 tester without *Dil* yielded pale colored kernels. Crosses of *r1 Spf* (no *Dil*) onto the *R1-r(sd2)* N10 tester without *Dil* yielded colored kernels with darker sectors. Finally, crosses of *r1 Dil Spf* onto the *R1-r(sd2)* N10 tester without *Dil* yielded pale colored kernels with sectors. In other words, *R1-r(sd2)* that has been separated from *Spf* and *Dil* does respond to both *Spf* and *Dil* separately or in combination when these factors are reintroduced, providing that *R1-r(sd2)* is carried on an N10 chromosome.

Crosses of *Dil* and *Spf* on to the *R1-r(sd2)* K10 tester without *Dil* gave results that are tantalizing but incomplete. All crosses intoducing *Dil* resulted in pale aleurone color, indicating that the presence of K10 has no effect on the response of *R1-r(sd2)* to *Dil*. However, none of the crosses introducing *Spf* resulted in sectored kernels. Control crosses of most of the plants used in the K10 crosses to an N10 version of *R1-r(sd2)* were not made because the K10 result was unanticipated. Thus, all of the putative *r1 Dil Spf* parents crossed to the K10 line were not independently verified to carry *Spf*, although it is likely that they do since the line was homozygous for *Dil* and *Spf* when tested in the previous generation. However, all *r1 Spf* (no *Dil*) parents were independently tested, and all of those known to carry *Spf* did not induce mutability when crossed to the K10 line.

Table 1. Kernel segregations on ears of crosses of *r1* with various combinations of *Dil* and *Spf* to various *R1-r(sd2)* testers and *R1-ch(Stadler)*.

male parent	male genotype[1]	kernel segregations [2] on crosses to *R1-r(sd2)* N10 / *r1-g* N10 tester	kernel segregations on crosses to *R1-r(sd2)* K10 / *r1-g* N10 tester	kernel segregations on crosses to *R1-r(sd2)* N10 *Dil* / r1-g N10 *dil* tester	kernel segregations on crosses to *R1-ch (Stadler)* tester
2003-962-2	*r1 Dil ?Spf*				Pale Cl
2003-962-3	*r1 Dil ?Spf*		Pale Cl, cl; K10 ratio		Pale Cl
2003-962-4	*r1 Dil ?Spf*		Pale Cl, cl; K10 ratio		Pale Cl
2003-962-5	*r1 Dil Spf*	Pale Cl sec, cl			Pale Cl
2003-962-6	*r1 Dil ?Spf*		Pale Cl, cl; K10 ratio		Pale Cl
2003-962-9	*r1 Dil ?Spf*		Pale Cl, cl; K10 ratio		Pale Cl
2003-962-10	*r1 Dil ?Spf*		Pale Cl, cl; K10 ratio		Pale Cl
2003-962-11	*r1 Dil ?Spf*		Pale Cl, cl; K10 ratio		Pale Cl
2003-963-3	*r1 Dil spf*	Pale Cl, cl			Pale Cl
2003-963-7	*r1 Dil Spf/spf*	Pale Cl, Pale Cl sec, cl			Pale Cl
2003-963-9	*r1 Dil spf*	Pale Cl, cl			Pale Cl
2003-963-10	*r1 Dil Spf/spf*	Pale Cl, Pale Cl sec, cl			Pale Cl
2003-963-11	*r1 Dil Spf/spf*	Pale Cl, Pale Cl sec, cl			
2003-964-1	*r1 dil Spf/spf*	Cl, sec base, cl		Cl, sec base, Pale Cl, sec, cl	Cl
2003-964-2	*r1 dil Spf*	Cl, sec base, cl		Cl, sec base, Pale Cl sec, cl	Cl
2003-964-4	*r1 dil Spf*		Cl, cl; K10 ratio	Cl, sec base, Pale Cl sec, cl	Cl
2003-964-7	*r1 dil Spf/spf*		Cl, cl; K10 ratio	Cl, sec base, Pale Cl, sec. cl	Cl
2003-964-8	*r1 dil spf*	Cl, cl		Cl, Pale Cl, cl	Cl
2003-911-3	*r1 dil spf*	Cl, cl		Cl, Pale Cl, cl	Cl
2003-911-9	*r1 dil spf*		Cl, cl 1:1 ratio = N10 crossover	Cl, Pale Cl, cl	Cl

[1]Male genotypes are based solely upon the results of test crosses made this generation. In the previous generation, family 2003-962 tested homozygous for both *Spf* and *Dil*, 2003-963 tested homozygous for *Dil* and segregating for *Spf*, and 2003-964 tested no *Dil* and segregating for *Spf*. 2003-911 is the W22 *r1-g(Stadler)* tester (no *Dil* or *Spf*).

[2]Pale Cl = pale aleurone color
cl = colorless aleurone
Cl = colored aleurone
sec = sectored aleurone
sec base = colored aleurone with visible darker sectors at kernel base

We tentatively conclude that *R1-r(sd2)* that has been separated from *Spf* and *Dil* does not respond to *Spf* when it is reintroduced either alone or in combination with *Dil*, when *R1-r(sd2)* resides on a K10 chromosome. However, *R1-r(sd2)* does respond to *Dil* regardless of whether *R1-r(sd2)* resides on an N10 or K10 chromosome. These results will have to be tested more thoroughly because there exists the possibility that the *R1-r(sd2)* K10 haplotype that we tested has lost its ability to respond to *Spf* for reasons other than the presence of K10. We derived our *R1-r(sd2)* K10 line from a single stable pale kernel segregant from an ear segregating for *Spf* and *Dil*. It is possible that this kernel was stable not because it lacked *Spf*, but rather because a mutation event or an imprecise excision event resulted in the formation of a nonresponsive *R1-r(sd2)* derivative. We plan to test other, independent *R1-r(sd2)* K10 derivatives, as well as create new *R1-r(sd2)* K10 derivatives by crossing over using the N10 source of *R1-r(sd2)* that is known to respond to *Spf* and an *r1* K10 line. We also plan to separate the nonresponsive *R1-r(sd2)* from K10 by crossing over to determine whether it can regain its ability to respond to *Spf* when carried on an N10 chromosome.

K10 is known to affect meiotic drive (Hiatt and Dawe, Genetics 164:699-709, 2003) and paramutation at the *r1* locus (Brink, Mutation Res 8:285-302, 1969). Our results suggest another possible effect of K10, the repression of transposable element excision. Whether this effect is confined to *R1-r(sd2)* or whether it could affect other mutable *r1* haplotypes or other linked or unlinked receptor loci is not known at this time. Clearly, the presence of a linked K10 does not automatically shut down transposable element excision events at *r1*. Descendants of the original *R1-r(sd2)* K10 mutational event retain mutability at *R1-r(sd2)* in the presence of *Spf* until *Spf* has segregated away. K10 also has no appreciable effect on mutability of *R1-st* (Brink, 1969). If the K10 effect is real, then apparently the mutable *R1-r(sd2)* on K10 is in a certain "state" in the presence of *Spf*; this state is reset once *Spf* has segregated away, and is not regained when *Spf* is reintroduced. This "state" could be a particular chromatin structure maintained by an *Spf* product such as a transposase that is irreversibly lost when the product is no longer present. Much remains to be learned about this system.

The *Spotted-dilute* system arose in a line in which *R1-r(Cornell)* was carried on a K10 chromosome (Stadler and Emmerling, MNL 30:61-63, 1956). If the K10 effect is real, its presence could be the reason that Sastry and Kurmi found that *Spf* did not induce mutability in *R1-r(sd2)* after *Spf* had been separated from it and then reintroduced. However, we found that the presence of K10 has no effect on the response of *R1-r(sd2)* to *Dil*, and this result conflicts with that of Sastry and Kurmi. Finally, we found that *Dil* suppresses aleurone color in crosses to *R1-ch(Stadler)*, but no combination of *Spf* with or without *Dil* induced mutability of *R1-ch(Stadler)*, and this also conflicts with Sastry and Kurmi. However, since Sastry and Kurmi did not indicate their source of *R1-ch*, it is possible that they used a different *r1* haplotype that happens to respond to *Spf*.

Finally, some comments should be made about what structural features of *R1-r(sd2)* could account for the phenotypes produced by its interactions with *Spf* and *Dil*. First, *Dil* seems to be similar, if not identical to *Inr1*. *Inr1* seems to work by suppressing *S* (seed color) subcomponents of certain *r1* haplotypes (Stinard and Sachs, J Hered 93:421-428, 2002). It is particularly effective at suppressing *S* subcomponents of inverted repeat haplotypes that have a single functional *S* subcomponent such as *R1-ch(Stadler)* and *R1-d(Catspaw)*. It does not, however, significantly reduce aleurone color when crossed to *R1-r(standard)*, a haplotype with two functional *S* subcomponents. In the case of *R1-r(standard)*, it could be that *Inr1* suppresses one *S* subcomponent, but not the other. If the lesion in *R1-r(sd2)* is a defective transposable element insertion into a nonsuppressible *S* subcomponent that knocks out its expression, then in the absence of *Inr1/Dil* and the autonomous element *Spf*, the suppressible *S* subcomponent would be expressed and give rise to near full color aleurone. In the presence of *Inr1/Dil*, the suppressible *S* subcomponent would be suppressed, giving rise to weak or no aleurone color. However, in the presence of both *Inr1/Dil* and *Spf*, excision of the defective transposable element inserted at the nonsuppressible *S* subcomponent would give rise to purple tissue sectors carrying a functional *S* subcomponent on a pale suppressed *S* background. This model will be further tested.

Spf from the *Spotted-dilute* system is a member of the *En/Spm* controlling element family

--Stinard, PS

The *Spotted-dilute* controlling element system was originally characterized as a three element system involving the autonomous element *Spf* (*spotting factor*), a receptor at the *r1* locus in the haplotype *R1-r(sd2)* (*spotted dilute2*), and a third factor called *Dil* (*diluting factor*) that suppresses background aleurone coloration in crosses involving *R1-r(sd2)* (Sastry and Kurmi, MNL 44:101-105, 1970). Another haplotype that responds to *Spf*, *R1-r(sd4)* has apparently been lost. There are several reported peculiarities of this system that set it apart from other controlling element systems, and these peculiarities will be addressed in a separate report in this MNL. Sastry and Kurmi tested *Spf* against the *Ac Ds* system and found no interaction. However, preliminary data showed that lines carrying *Spf* induced mutability at *a2-m1::dSpm*. Fincham and Sastry (Annu Rev Genet 8:15-50, 1974) cite control of *R1-r(sd2)* by *Spm*. In contrast, Singh, Sachan, Guha, and Sarkar (MNL 49:45-49, 1975) obtained negative results when they tested *Spf* against *c2-m2::dSpm*. In order to resolve these inconsistencies and simplify analysis of the *Spotted-dilute* system, several tests were conducted to explore the relationship between the *Spotted-dilute* and *En/Spm* systems.

In pilot projects to find appropriate combinations of autonomous elements and receptors for both systems, crosses of *Spf* were made to *dSpm/I* testers, and crosses of *En/Spm* were made to *R1-r(sd2)*. For crosses of *Spf* to *a1-m1-5719::dSpm sh2*, 14 plants were obtained that showed mutability at both *R1-r(sd2)* and *a1-m1-5719::dSpm*, and 5 were obtained that showed mutability at neither. There were no instances of mutability at one locus, but not the other. For crosses of *Spf* to *wx1-m8::Spm-I8*, 4 plants were obtained that showed mutability at both *R1-r(sd2)* and *wx1-m8::Spm-I8*, and 6 that showed mutability at neither. Again, there were no instances of mutability at one locus but not the other.

For crosses of *a1 Spm-S Sh2 wx1-m8::Spm-I8* to *R1-r(sd2)*, 8 out of 9 plants showed mutability at both *wx1-m8::Spm-I8* and *R1-r(sd2)*. 1 plant showed mutability at *R1-r(sd2)* but not at *wx1-m8::Spm-I8*. The possibility that the latter event represents an excision resulting in a stable null derivative of *wx1-m8::Spm-I8* has not been ruled out. Finally, for crosses of *o2-m12::Spm* to *R1-r(sd2)*, 8 out of 8 plants showed mutability at both loci.

These results are highly suggestive of *Spf* and *R1-r(sd2)* being members of the *En/Spm* controlling element system. To demonstrate this more conclusively, followup crosses were made involving *Spm*, *wx1-m8::Spm-I8*, and *R1-r(sd2)* for the ease of scoring mutability at *wx1* and *r1* simultaneously on individual kernels. The cross was set up as follows: [*R1-r(sd2) Wx1 A1 Sh2* X *R1-r(sd2) wx1-m8::Spm-I8 a1 Spm-S Sh2*] X *R1-r(sd2) wx1-m8::Spm-I8 A1 Sh2 Dil*. Two ears resulting from this cross segregated 1:1 for mutable and stable aleurone color indicating segregation for one *Spf* element, and approximately 1:1 for starchy and waxy due to heterozygosity at the *wx1* locus (there was a shortage of waxy kernels in one cross resulting in a deviation from 1:1 significant at the 5%, but not the 1% level). The waxy kernel class was scored for mutability at the *wx1* locus indicative of the presence of *En/Spm*. Complete kernel counts are given in Table 1. Of the kernels scorable for both *wx1* and *r1* mutability, 223 showed mutability at both loci and 226 showed mutability at neither (with no exceptions), demonstrating a complete correlation between the presence of *En/Spm* and *Spf* in a population of 449 individuals. Additional tests will be conducted in order to boost these numbers, but it is safe to conclude that *En/Spm* and *Spf* are functionally equivalent.

Table 1. Counts of kernels from the cross [*R1-r(sd2) Wx1 A1 Sh2* X *R1-r(sd2) wx1-m8::Spm-I8 a1 Spm-S Sh2*] X *R1-r(sd2) wx1-m8::Spm-I8 A1 Sh2 Dil*.

female parent	cl mutable; wx mutable	cl mutable Wx	cl stable; wx stable	cl stable Wx
2003-852-2	135	134	137	139
2003-852-10	88	123	89	120
Totals	223	257	226	259

1:1:1:1 χ^2 = 4.679 ($p<0.05$)
1:1 χ^2 for Wx to wx = 4.652 ($p<0.05$)
1:1 χ^2 for cl mutable to cl stable = 0.026 (NS)

New *inr1* and *inr2* alleles

--Stinard, PS

inr1 and *inr2* are loci with dominant alleles that suppress aleurone color in crosses to specific *r1* haplotypes (Stinard and Sachs, J Hered 93:421-428, 2002). To date, two dominant *inr1* alleles, *Inr1-Ref* and *Inr1-JD*, and one dominant *inr2* allele, *Inr2-JD*, have been described. We report the identification of two likely *inr1* alleles and one additional *inr2* allele from diverse sources.

Dil is a dominant inhibitor of aleurone color originally described as one of the factors in the *Spotted-dilute* controlling element system (Sastry and Kurmi, MNL 44:101-105, 1970). Crosses to various *r1* haplotypes give a similar pattern of response as *Inr1* and *Inr2* alleles, so mapping crosses of *Dil* were set up with *Inr1-Ref*, *Inr1-JD*, and *Inr2-JD* to test whether *Dil* maps to the same chromosomal location as these factors. Testcrosses involving Dil and Inr2-JD indicated independent assortment of these two factors (data not shown). However, the testcrosses of *Dil* involving *Inr1-Ref* and *Inr1-JD* (Testcross: [*Inr1 R1-Randolph* X *Dil R1-r(sd2)*] X *inr1 R1-Randolph*) showed very tight linkage (*Inr1-ref* crosses yielded 3,756 pale and colorless kernels, and no full colored exceptions; *Inr1-JD* crosses yielded 3,355 pale and colorless kernels and 1 full colored exception that could be a contaminant). Combining the data from both sets of *Inr1* crosses yields an *Inr1 Dil* linkage distance of less than 0.03 centiMorgans. It is likely that *Inr1* and *Dil* map to the same location, and that they are allelic. The full colored exception will be tested for contamination markers next summer.

It was reported by Stinard (MNL 77:77-79, 2003) that an *Fcu* line obtained from Peter Peterson of Iowa State University (74-1033-8@) elicits dark purple sectoring on a pale purple background in crosses to *R1-ch(Stadler)* and other *r1* haplotypes that are susceptible to aleurone color inhibition by dominant *Inr1* and *Inr2* alleles. It was not known whether the suppression of background aleurone color was an inherent property of *Fcu*, or whether the *Fcu* line carried dominant inhibitors of aleurone color. In order to test for inhibitors, the *Fcu* line was crossed to *Inr1-JD* and *Inr2-JD*, followed by a backcross by *R1-ch(Stadler)*. The *Inr2-JD* backcrosses showed independent segregation of two dominant aleurone color inhibitors (data not shown). However, the *Inr1-JD* backcrosses indicated tight link-

age between *Inr1* and a dominant inhibitor carried by the *Fcu* line (Table 1; full colored putative crossovers were counted twice in linkage calculations to account for the lack of ability to score the *Inr1 Inr** crossover class). The full colored kernels that were observed could be crossovers, contaminants, or they could represent reversion events at *Fcu*. The frequency of full colored kernels in these crosses (1.7×10^{-3}) is close to the observed reversion rate for *Fcu* (3.0×10^{-3}; reported elsewhere in this MNL). Additional linkage tests will be conducted in a background free of *Fcu*. It is likely that the inhibitor present in the *Fcu* line is an allele of *inr1*, but a more definitive answer will have to wait until these additional tests have been completed.

Table 1. Counts of pale purple stable, pale purple sectored, and full colored kernels from the cross [*Inr1-JD R1-Randolph fcu* X *Inr* r1-g Fcu*] X *inr1 R1-ch(Stadler) fcu*.

Female parent	No. pale purple stable	No. pale purple with purple sectors	No. full colored	1:1 χ^2 stable:sectored
2002P-195-1	124	120	2	0.066 (NS)
2002P-195-3	146	149	0	0.031 (NS)
2002P-195-5	163	195	0	2.860 (NS)
2002P-195-6	136	135	0	0.004 (NS)
2002P-195-7	120	142	1	1.847 (NS)
2002P-195-8	74	110	0	7.043 (p<0.01)
2002P-195-9	89	97	0	0.344 (NS)
Totals	852	948	3	5.121 (p<0.05)

Percentage crossing over between *Inr1* and *Inr** = 0.33 +/- 0.14.

During the course of our studies of *inr2*, which maps to the long arm of chromosome 9, we performed crosses to determine whether *inr2* lies proximally or distally to the TB-9Lc breakpoint, 9L.10. We were surprised when reciprocal crosses of Jack Beckett's TB-9Lc *Wc1* line to a N9 *R1-Randolph* line yielded colorless kernels when the TB-9Lc line was used as a female, but segregated for large pale and colorless kernels and smaller full colored kernels when the TB-9Lc line was used as a male. These results are indicative of a dominant inhibitor of *R1-Randolph* carried on the B-9Lc chromosome. The two kernel color classes are due to nondisjunction of the B centromeres at the mitotic division of the generative nucleus during pollen grain development (Beckett, J Hered 69:27-36, 1978). The larger pale and colorless kernels presumably have hyperploid endosperms carrying two B-9Lc chromosomes and hypoploid embryos carrying no B-9Lc chromosome. Conversely, the smaller full colored kernels presumably have hypoploid endosperms carrying no B-9Lc chromosome and hyperploid embryos carrying two B-9Lc chromosomes. The chromosomal makeup of these progeny kernels remains to be confirmed. The inhibitor in the TB-9Lc stock was tested for linkage with the *inr2* locus by the following testcross: [*r1* TB-9Lc *Inr** X *R1-Randolph* N9 *Inr2-JD*] X *R1-Randolph* N9 *inr2*. Of a total of 4,648 progeny kernels, all were pale purple or colorless, and none were full colored. Since it maps to the same location as *inr2* and shares the same properties as *Inr2-JD*, it is very likely that the inhibitor in the TB-9Lc stock is an allele of *inr2*, and we have named it *Inr2-9Lc*.

Three point linkage data for *wx1 inr2 v30*

--Stinard, PS

Dominant mutant alleles at the *inr2* locus suppress aleurone color in crosses to specific *r1* haplotypes (Stinard and Sachs, J Hered 93:421-428, 2002). Previous work (Stinard, MNL 77:76-66, 2003) places *inr2* on the long arm of chromosome 9, approximately 23 cM from *wx1*. In order to refine this placement, we conducted a three point linkage test of *wx1*, *inr2*, and *v30*, the latter locus being on the long arm of chromosome 9 in the vicinity of where *inr2* would be expected to map. The linkage testcross and results are indicated in Table 1. All lines are homozygous for the suppressible haplotype *R1-Randolph* to allow scoring for *inr2*. Kernels were scored for *wx1* and *inr2* on the test cross ears at maturity, and these kernels were subsequently planted in a cold sand bench and the resulting seedlings were scored for *v30*.

Table 1. Three point linkage data for *wx1*, *inr2*, and *v30*.

Testcross: [*Wx1 Inr2-JD V30 R1-Randolph* X *wx1 inr2 v30 R1-Randolph*] X *wx1 inr2 v30 R1-Randolph*.

Region	Phenotype	No.	Totals
0	Wx Inr V	897	
	wx inr v	938	1835
1	Wx inr v	261	
	wx Inr V	282	543
2	Wx Inr v	106	
	wx inr V	106	212
1 + 2	Wx inr V	23	
	wx Inr v	21	44

Map distance *wx1—inr2* = 22.3 +/- 0.8 cM
Map distance *inr2—v30* = 9.7 +/- 0.6 cM
Map distance *wx1—v30* = 32.0 +/- 0.9 cM

This linkage test establishes the global gene order as *wx1 inr2 v30*. The *wx1-inr2* distance (22.3 +/- 0.8 cM) agrees closely with those obtained previously (22.6 +/- 0.9 cM and 22.9 +/- 1.2 cM). The *wx1-v30* distance (32.0 +/- 0.9 cM) agrees with that indicated on the 1993 genetic map of chromosome 9 (31 cM; Neuffer et al., Mutants of Maize, Cold Spring Harbor Laboratory Press, 1997). *inr2* maps about 9.7 cM proximally to *v30*, in the vicinity of *bk2*.

The controlling elements *Fcu* and *arv-m594* map to chromosome 2

--Stinard, PS

Fcu was first identified by Gonella and Peterson (Genetics 85:629-645, 1977) as the factor responsible for aleurone color sectoring at the *r1* locus in Cuna tribal maize from Colombia. The *Fcu* system was found to be comprised of two elements: a responsive *r1* haplotype, *r1-cu*, and the controlling element *Fcu*. *r1-cu* produces a variable pale aleurone coloration in the absence of *Fcu*, but produces sectors of full color pigmentation on a pale background in the presence of *Fcu*. Two other *r1* haplotypes, *R1-r(sd2) (spotted dilute2*; also referred to as *R-r#2*; Gonella and Peterson, Molec Gen Genet. 167:29-36, 1978) and *R1-mo(cu)* (Gonella and Peterson, MNL 50:61-63, 1976) were subsequently found to produce sectoring in the presence of *Fcu* as well.

In MNL 77:77-79, we reported that the haplotypes *R1-ch(Stadler)*, *R1-d(Catspaw)*, *R1-Randolph*, *R1-r(Venezuela*

412-PI302347), and *R1-r(Venezuela559-PI302355)* also respond to *Fcu*. It is not clear whether the response at the *r1* locus is a direct one, or whether *Fcu* elicits its phenotype through interaction with intermediaries such as enhancers or suppressors of *r1* aleurone color expression.

Other genetic factors called *arv-m*'s (mutable amplifiers of aleurone color in certain *R1-Venezuela* haplotypes; Kermicle, MNL 77:52, 2003) also elicit aleurone color sectoring in crosses to the same haplotypes and may be similar, if not identical, to *Fcu*. As a first attempt at characterizing the similarities and differences between these controlling elements, *Fcu* (isolate PAP74-1033-8 received from Peter Peterson of Iowa State University) and *arv-m594* (an *arv-m* element isolated from one of Brink's *R1-r(Venezuela594-PI302363)* lines, Stock Center ID# X235B) were mapped using a series of *wx1* marked A-A translocations. Mapping crosses involving the translocations T1-9c, T1-9(4995), T1-9(8389), T3-9(8447), T3-9(8562), T4-9e, T4-9(5657), T5-9c, T5-9a, T6-9b, T7-9(4363), T7-9a, T8-9d, T9-10b, and T9-10(8630) failed to show linkage between *wx1* and *Fcu* (data not shown). Mapping crosses involving the translocations T1-9c, T1-9(5622), T1-9(8389), T3-9(8447), T3-9(8562), T4-9e, T4-9(5657), T5-9(022-11), T5-9a, T6-9e, T7-9(4363), T7-9a, T8-9d, T9-10(059-10), and T9-10b failed to show linkage between *wx1* and *arv-m594*. However, mapping crosses involving T2-9c, T2-9b, and T2-9d showed linkage of *wx1* with both *Fcu* (Table 1) and *arv-m594* (Table 2).

Table 1. Two point linkage data for *wx1* and *Fcu* in crosses involving T2-9 translocations.

Testcross: [*Wx1* T *fcu r1* X *wx1* N *Fcu r1*] X *wx1-m8* N *fcu R1-r(sd2)*

	Region 0		Region 1		
Translocation	Wx fcu	wx Fcu	wx fcu	Wx Fcu	% recombination *wx1—Fcu*
T2-9c (2S.49; 9S.33)[1]	484	335	176	354	39.3 +/- 1.3
T2-9b (2S.18; 9L.22)[2]	596	586	110	114	15.9 +/- 1.0
T2-9d (2L.83; 9L.27)[3]	637	623	103	95	13.6 +/- 0.9

[1]Total of 4 crosses.
[2]Total of 7 crosses.
[3]Total of 5 crosses.

Table 2. Two point linkage data for *wx1* and *arv-m594* in crosses involving T2-9 translocations.

Testcross: [*wx1* T *arv r1* X *Wx1* N *arv-m594 R1-r(Venexuela594-PI302363)*] X *wx1-m8* N *arv R1-r(sd2)*

	Region 0		Region 1		
Translocation	Wx arv-m	wx arv	wx arv-m	Wx arv	% recombination *wx1—arv-m594*
T2-9c (2S.49; 9S.33)[1]	738	829	418	281	30.8 +/- 1.0
T2-9b (2S.18; 9L.22)[2]	422	370	118	149	25.2 +/- 1.3
T2-9d (2L.83; 9L.27)[3]	676	603	60	72	9.4 +/- 0.8

[1]Total of 6 crosses.
[2]Total of 4 crosses.
[3]Total of 4 crosses.

Both *Fcu* and *arv-m594* show the same general patterns of linkage with *wx1* in the T2-9 translocations (Tables 1 and 2). It should be noted that the T2-9c data show distortions in the ratios of waxy to nonwaxy kernels indicative of the transmission of duplicate-deficient chromosomal segments through the female. The linkage was tightest with T2-9d (2L.83; 9L.27; 13.6 and 9.4 centiMorgans, respectively) and weakest with T2-9c (2S.49; 9S.33; 39.3 and 30.8 cM, respectively), indicating that both factors probably reside on the long arm of chromosome 2. However, the linkage values are clearly not identical for the two factors, all linkage values lying well outside each other's standard errors in side by side comparisons. These differences could be due to differences in chromosomal location for the two factors, but they could also be due to differences in genetic background between the lines, which are known to affect linkage values. No conclusions as to whether *Fcu* and *arv-m594* map to the exact same chromosomal location can be drawn based on these data. In order to resolve this question, direct mapping crosses will be made between *Fcu* and *arv-m594*, as well as mapping crosses of *Fcu* and *arv-m594* with the chromosome 2 marker stock *fl1 v4 w3 Ch1*.

The isolation and characterization of *Fcu* revertants

--Stinard, PS

No full colored germinal revertants have been reported from the *Fcu* controlling element system (Gonella and Peterson, Molec Gen Genet 167:29-36, 1978). Of 30 full colored kernels isolated from crosses involving the *Fcu* responsive haplotype *r1-cu* and *Fcu*, all turned out to be nonrevertant, and had appeared to be full colored due to the variable high background of aleurone color expression by *r1-cu*. We recently found that additional *r1* haplotypes respond to *Fcu*, and some of these haplotypes have a paler aleurone color background than *r1-cu* (Stinard, MNL 77:77-79, 2003). We proposed that mutability, and therefore reversion events, in the *Fcu* system is most likely due to changes at *Fcu* and not changes at the *r1* locus. In order to isolate *Fcu* revertants for further study, we made crosses of an *r1-g Fcu* line onto a responsive pale *r1* haplotype, *R1-r(Venezuela559-PI302355)*. Many full colored putative single kernel revertants were isolated from such crosses, and are summarized in Table 1. Many stable pale kernels that might represent *Fcu* losses were also observed, but these were not counted separately. Kernel counts are grouped by *Fcu* male parent because the possibility exists that multiple kernel revertants in such crosses could arise from revertant tassel sectors and might represent a single reversion event. If one counts each putative revertant as an independent event, the frequency of reversion observed in this population is 3.0×10^{-3}. However, if one considers multiple revertant kernels arising from one male parent as single events, the reversion rate becomes 8.1×10^{-4}. These two frequencies provide logical bounds for the rate of *Fcu* reversion in this population. All of these putative revertants need to be grown out and tested for the presence of contamination markers (*y1* and *wx1* from the *Fcu* parent) in order to rule out the possibility of contamination. However, if any of these putative revertants are real, it is clear that reversion is occurring at *Fcu* (or some other locus that interacts with *Fcu*) and not at the *r1* locus since the *r1-g* haplotype in the *Fcu* parent is not mutable in the presence of *Fcu*.

One of the *Fcu* male parents (2003-2653-6) used in these crosses was found to be heterozygous for a reversion event that must have arisen unnoticed in the previous generation. Kernel counts from this male parent are summarized in Table 2. Outcrosses of this particular parent to the *R1-r(Venezuela 559-PI302355)* tester resulted in 1:1 segregation for full colored and sectored/pale kernels. The self-pollinated ear from the

Table 1. Counts of full colored putative single kernel revertants from the cross *R1-r(Venezuela559-PI302355 fcu* X *r1-g Fcu wx1 y1*.

Fcu male parent	female parent	No. sectored and pale kernels	No. full colored kernels
2003-2650-6	2655-1	248	1
	2655-9	142	1
2003-2650-8	2640-2	261	0
	2640-3	195	1
	2640-8	131	1
2003-2650-9	2640-9	279	0
	2641-9	211	3
2003-2650-10	2643-1	141	0
	2643-5	178	0
	2643-9	137	0
	2656-9	123	0
	2656-10	135	0
2003-2651-1	2655-7	199	0
	2656-7	177	1
	2658-1	208	4
	2658-3	180	1
	2658-4	253	0
	2658-5	296	0
	2658-12	208	1
	2672-6	269	3
2003-2651-3	2640-1	183	1
	2640-11	214	0
	2654-1	218	0
	2654-8	217	1
	2654-9	264	0
	2654-10	298	2
	2654-12	162	0
	2657-2	215	0
	2657-4	155	0
	2657-7	202	0
2003-2651-9	2656-4	145	0
	2656-5	246	0
	2656-6	289	4
2003-2651-10	2641-2	284	0
	2642-7	180	0
	2642-8	173	1
	2657-5	118	0
	2657-6	150	0
2003-2651-11	2654-3	278	0
	2654-6	222	2
	2654-7	324	1
	2654-11	198	1
2003-2652-2	2643-2	113	0
	2643-3	165	0
	2643-4	140	0
	2643-8	126	0
	2643-11	133	0
	2655-8	152	0
2003-2653-2	2655-2	260	2
	2655-5	292	0
	2655-6	278	0
	2657-10	201	2
	2658-8	165	1
	2658-10	285	0
2003-2653-5	2642-2	179	0
	2642-6	196	0
	2642-9	137	0
	2642-10	168	0
	2642-12	138	0
	2657-1	141	0
2003-2653-10	2641-3	155	2
	2641-8	146	0
Grand Total		12,276	37

Frequency of reversion = 3.0 X 10^{-3}

Table 2. Counts of kernels from crosses involving the heterozygous *Fcu* revertant parent 2003-2653-6. Test cross: *R1-r(Venezuela559-PI302355 fcu* X *r1-g*; *Fcu/Fcu-R*; *wx1 y1*.

female parent	No. sectored and pale kernels	No. full colored kernels
2640-5	120	162
2655-8	121	105
2655-10	110	88
2657-8	84	109
Totals	435	464

1:1 χ^2 = 0.935 (not significant)

male parent plant was homozygous for the *y1* and *wx1* contamination markers, ruling out contamination of the *Fcu* parent line. The reproducibility of the 1:1 segregation in four separate outcrosses rules out contamination arising during pollination, as well as contamination occurring in the *R1-r(Venezuela 559-PI302355)* parent line. This event is the first proven instance of reversion in the *Fcu* system, and the revertant has been named *Fcu-R2003-2653-6*. Crosses are planned to determine whether *Fcu-R2003-2653-6* maps to the same chromosomal location as *Fcu*, or whether it represents a transpositional event or a change at an unlinked locus. The other putative single kernel revertants will be subjected to the same analysis if they prove not to be contaminants.

Three point linkage data for *su3* T4-9g *wx1*

--Stinard, PS

su3 and *su4* are a duplicate factor pair that give a sugary kernel phenotype when both factors are homozygous mutant (Stinard, MNL 63-64, 2002). Two point linkage data were reported for *su3* and *wx1* (T4-9g) in 1992 (6.5 +/- 2.6 cM; Stinard, MNL 66:4-5). It is likely that the *su3* and *wx1* T4-9g stocks used in these crosses carried *su4* as well since 1:1 segregation of *Su3* and *su3* was observed in the modified backcross. Separate lines of *su3 Su4* and *Su3 su4* have now been isolated, and linkage tests of *su3* with *wx1* T4-9g have been redone using these new lines. The testcross and results are presented in Table 1. The *su3 wx1* linkage distance from these crosses (7.7 +/- 2.5 cM) agrees closely with that obtained from the 1992 data (6.5 +/- 2.6 cM). These data also provide a test of linkage of *wx1* with T4-9g (1.9 +/- 1.1 cM), which differs somewhat from that reported by Anderson et al. (4.19 +/- 1.55 cM; MNL 39:106-109, 1965).

Table 1. Counts of phenotype segregations for individual ears of self-pollinated plants grown from kernels of the testcross *Su3 su4 Wx1* N X [*Su3 Su4 wx1* T4-9g X *su3 Su4 Wx1* N].

Region 0		Region 1		Region 2		Regions 1 and 2	
su N Wx	Su T wx	su T wx	Su N Wx	su N wx	Su T Wx	su T Wx	Su N wx
82	62	4	4	0	2	1	0

Map distance *su3*—T = 5.8 +/- 1.9 cM
Map distance T—*wx1* = 1.9 +/- 1.1 cM
Map distance su3—*wx1* = 7.7 +/- 2.1 cM

Additional linkage tests of *waxy1* marked reciprocal translocations at the MGCSC

--Jackson, JD, Stinard, P, Zimmerman, S

In the collection of A-A translocation stocks maintained at MGCSC is a series of *waxy1*-linked translocations that are used for mapping unplaced mutants. Also, new *wx1*-linked translocations are being introduced into this series and are in a conversion program to convert each translocation to the inbred backgrounds M14 and W23. These inbred conversions are then

crossed together to produce vigorous F1's to fill seed requests. Over the years, pedigree and classification problems arose during the propagation of these stocks. We have been able to sort through the problem ones, and can now supply good sources proven by linkage tests to include the correct translocated chromosomes. Additional pedigree information on bad sources is available should anyone want to check on sources supplied to them previously by the Stock Center.

Previously we reported the linkage results for some of these stocks (MNL72:81-82; MNL73:88-89; MNL74:67-69; MNL75:68-71; MNL76:65-67; MNL77:79). Below is a summary of additional translocation stocks we have completed testing. Additional translocation stocks will be tested as time allows.

Table 1. *wx1* T6-9(4778) (6S.80; 9L.30)

A) The F1 source showed linkage of *wx1* with *hcf26.*

2 point linkage data for *hcf26-wx1* T6-9(4778)
Testcross: [*Hcf26 wx1* T6-9(4778) x *hcf26 Wx1* N] x *hcf26 wx1* N

source:93-458-2 x 459 ^F1

Region	Phenotype	No.	Totals
0	hcf Wx	4342	
	+ wx	3681	8023
1	+ Wx	274	
	hcf wx	129	403

% recombination *hcf26-wx1*= 4.8± 0.2

Allelism testing of viviparous stocks with pink scutellum in the Maize Coop phenotype only collection

--Jackson, JD

This report summarizes allele testing of viviparous stocks characterized by phenotype only in the Maize Genetics Coop Stock Center collection. Some of these mutants have been found in other Coop stocks and some have been sent in by cooperators over the years. Crosses were made as follows: (+/*ps**) x (+/*ps1*) or (+/*ps1*) x (+/*ps**) and ears were scored at maturity for the pink scutellum phenotype. Proposed designations have been assigned to these new alleles and they have been placed on our stocklist. It is expected that with further sorting and allelism testing of viviparous stocks characterized by phenotype only, additional alleles of characterized mutants will be discovered and placed in the main collection.

previous designation	allelism test with *ps1*	new designation	MGCSC: stock number
*pale y-vp**-84-5266-5	6 positive	*ps1*-84-5266-5	525A
*vp**-85-3011-11	4 positive	*ps1*-85-3011-11	525B
*ps**-Mu85-3061-21	6 positive	*ps1*-Mu85-3061-21	525C
*vp**-85-3135-4	7 positive	*ps1*-85-3135-4	525D
*vp**-85-3182-6	4 positive	*ps1*-85-3182-6	525E
*ps**-85-3288-28	3 positive	*ps1*-85-3288-28	525F
*vp**-85-3422-13	2 positive	*ps1*-85-3422-13	525G
*ps**-85-3492-36	3 positive	*ps1*-85-3492-36	525H
*ps-vp**-85-86-3567-1	6 positive	*ps1*-85-86-3567-1	525I
*ps**-Mu86-1105-1	3 positive	*ps1*-Mu86-1105-1	525J
*ps**-86-1105-2	6 positive	*ps1*-86-1105-2	525K
*ps**-86-1352-4	2 positive	*ps1*-86-1352-4	525M
*vp-Y**-86-1361-7	5 positive	*ps1*-86-1361-7	525N
*vp(ps)**-86-1449-3	5 positive	*ps1*-86-1449-3	525L
*ps**-86-1499-3	2 positive	*ps1*-86-1499-3	525O
*vp(ps)**-86-1565-17	4 positive	*ps1*-86-1565-17	526A
*ps**-86-87-1742-18	3 positive	*ps1*-86-87-1742-18	526B
*ps**-90-3222-27	6 positive	*ps1*-90-3222-27	526C
*ps**-90-91-8549-7	2 positive	*ps1*-90-91-8549-7	526D
*ps**-96-5032-6	2 positive	*ps1*-96-5032-6	526E
*ps**-98-5691-5	4 positive	*ps1*-98-5691-5	526F
*ps**-99-2157-1	2 positive	*ps1*-99-2157-1	526G
*ps**-MGD-A-53-17	3 positive	*ps1*-2001-4095-1	5107 (MGD project)

Allelism testing of miscellaneous stocks in the Maize Coop phenotype only collection

--Jackson, JD

This report summarizes allele testing of miscellaneous stocks characterized by phenotype only in the Maize Genetics COOP Stock Center collection. Over the years, some of these mutants have been found in other COOP stocks and some have been sent in by cooperators. In most cases crosses were made between known heterozygotes and homozygous plants. Plants were scored at the seedling stage and again at maturity. Proposed new designations have been assigned to these alleles. The stocks with positive tests have been increased and placed on our stocklist. It is expected that with further sorting and allelism testing of mutations characterized by phenotype only, additional alleles of characterized mutants will be discovered and placed in the main collection.

POSITIVE TESTS:

previous designation	allelism test with *al1*	new designation	MGCSC: stock number
*lw**-1998-2	positive(+/lw*) x (+/al1)	*al1*-1998-2	203C

previous designation	allelism test with *y1-gbl1*	new designation	MGCSC: stock number
*g**-N2236	positive: (+/g*) x (+/*y1-gbl1*)	*y1-gbl1*-N2236	602H
pg-N547B*	positive; (+/pg*) x *y1-gbl1*; (+/pg*) x (+/*y1-gbl1*)	*y1-gbl1*-N547B	602I

previous designation	allelism test with *wlu2*	new designation	MGCSC: stock number
*wl**-N629A	positive; (+/*wlu2*) x (+/wl*)	*wlu2-N629A*	727BB

NEGATIVE TESTS

previous designation	allelism test with *zn2*	allelism test with *zb1*	allelism test with *zb3*	allelism test with *zb4*	allelism test with *zb7*
zn-PI228181*	negative: (+/zn*) x zn2; zn2 x (+/zn*)	negative: zn* x zb1	negative: zn* x zb3		
zn-78-695*		negative: (+/zn*) x zb1	negative: (+/zn*) x zb3	negative: (+/zn*) x zb4	negative: (+/zn*) x zb7

previous designation	allelism test with *ws3*	allelism test with *yg2*	allelism test with *g1*	allelism test with *g2*	
ws-N537D*	negative: (+/ws*) x ws3; (+/ws3) x (+/ws*)	negative: (+/ws*) x yg2; (+/yg2) x (+/ws*)			
v-N2260*	negative: v* x ws3	negative: (+/yg2) x v*; v* x yg2	negative:	negative:	

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Recent studies of CMS-S *restorers-of-fertility* have led to a change in nomenclature

--Gabay-Laughnan, S, Chase, CD

In maize genetics nomenclature, loci are indicated in lower case italics, e.g., the *rf3* locus. Alleles at a locus are also indicated in italics, with the first letter capitalized for dominant alleles, e.g., the *Rf3* allele. Restoring alleles for CMS-S act in haploid pollen, i.e., fertility restoration is gametophytic (Buchert, Proc. Natl. Acad. Sci. USA 47:1436-1440, 1961), and dominance relationships of restoring and nonrestoring alleles cannot be directly assessed (Kamps et al., Genetics 142:1001-1007, 1996). Based on our finding that a homozygous-lethal restoring allele is recessive to the nonrestoring allele in diploid pollen produced

by CMS-S tetraploid plants (Wen et al., Genetics 165:771-779, 2003), we have adopted the symbol *restorer-of-fertility lethal* (*rfl*) for homozygous-lethal restorers. Therefore, the symbol *rfl1* replaces the previously published symbol *RflII* (Laughnan and Gabay, Genetics and the Biogenesis of Cell Organelles, Ohio State Univ. Press, Columbus, pp. 330-349, 1975), while the symbol *rfl2* replaces the previously published symbol *RfVI* (Laughnan and Gabay, 1975). In addition, we have adopted the symbol *restorer-of-fertility viable* (*rfv*) for homozygous-viable *restorer-of-fertility* alleles that arise via spontaneous mutation or transposon mutagenesis because these alleles are also likely to be recessive. The symbol *rfv1* replaces the previously published symbol *RfIV (*Laughnan and Gabay, 1975). The symbol *restorer-of-fertility nonfunctional* (*rfn*) has been adopted for restorers that circumvent S cytoplasm pollen abortion but result in shed pollen that is nonfunctional (Gabay-Laughnan and Laughnan, Maydica 28:251-263, 1983); the symbol *rfn1* replaces the previously published symbol *Rf-nf 81-296-12* (Gabay-Laughnan, Maydica 42:163-172, 1997). The mutation by which a gene is identified is designated the reference allele. We will refer to that allele by the symbol -1, e.g., *rfl1-1*, *rfl2-1*. Because there are multiple, independent *restorer-of-fertility* loci for CMS-S, we anticipate that we will have a series of genes with the *rfl*, *rfv*, and *rfn* designations. New alleles at these loci that have arisen by independent mutational events will be identified by a laboratory number that indicates the year of isolation and the row number, e.g., *rfl1-99829* and *rfl2-911066.* The symbol *rfl2-911066* replaces the previously published symbol *Rf*-91-1066-3* (Gabay-Laughnan and Chase, MNL 74:73-74, 2000). Restorer alleles that have yet to be placed to chromosome, and tested for allelism with those already mapped and designated, will be given temporary symbols that also indicate the year of isolation and the row number, e.g. *rfl*-003379*, and *rfv*-991181.*

CMS-S *restorers-of-fertility* from multiple sources cluster on chromosome 2L

--Gabay-Laughnan, S, Chase, CD

In the CMS-S system, expression of a novel chimeric gene in the mitochondria results in the collapse of starch-filling pollen and a male-sterile phenotype. Loss-of-function mutations in nuclear genes required for mitochondrial gene expression behave as *restorer-of-fertility* alleles, disrupting expression of the CMS-S gene and the male fertility trait. Restoring alleles also disrupt expression of essential mitochondrial genes. These mutations are visible in pollen because it is haploid; they are tolerated in pollen because late-stage pollen development and pollen germination do not require mitochondrial respiration (Wen et al., Genetics 165:771-779, 2003).

The first nuclear gene capable of restoring fertility to CMS-S male-sterile maize plants was designated *Rf3* (Duvick, Adv. Genet. 13:1-56, 1965). The *rf3* locus has been mapped to the long arm of chromosome 2 (2L) via the use of a chromosome 2 inversion and reciprocal translocations involving 2L (Laughnan and Gabay, Maize Breeding and Genetics, John Wily, New York, pp. 427-446, 1978; Gabay-Laughnan et al., in press). These studies placed *rf3* proximal to the 2L.80 breakpoint. Through the use of RFLP markers, *rf3* was positioned 4.3cM distal to the *whp1* locus and 6.4cM proximal to the *bnl17.14* locus (Kamps and Chase, Theor. Appl. Genet. 95:525-531, 1997).

Since *Rf3* maps to 2L, this is usually the first chromosome arm that we test for linkage with newly arising CMS-S restorers. New restorers are located on a variety of chromosomes (reviewed by Gabay-Laughnan et al., Advances in Cellular and Molecular Biology of Plants. Volume 2: Molecular Biology of the Mitochondria, Kluwer, Dordrecht, Netherlands, pp. 395-432, 1995; Chase and Gabay-Laughnan, in press); while at least 47 of them map to chromosome arms other than 2L, there is a cluster of spontaneous restorers mapping to this arm. The spontaneously arisen restorers *rfl1*, *rfl2* and *rfn1* all map to 2L. The *rfl1* locus exhibits linkage to the *whp1* locus, thus placing it in the region of the *rf3* locus (Wen et al., 2003). Direct tests of allelism have established that *rf3, rfl1, rfl2 and rfn1* are distinct genetic loci. One spontaneous Oh51A restorer allele, *rfl1-99829*, has been mapped to the *rfl1* locus while two Oh51A restorers, *rfl2-911066* and *rfl2-921663,* map to the *rfl2* locus. An additional Oh51A restorer allele, *rfl*00-130*, has just recently been mapped to 2L. Its relationship to the other 2L restorers is as yet unknown. In addition, three of the new restorers recovered from transposon-active lines map to 2L; *rfl2-99114* from an *Ac-Ds* screen maps to the *rfl2* locus while the unplaced alleles *rfv*-991181*, also from an *Ac-Ds* screen, and *rfl*-003379* from an *I-En* (*Spm*) screen map to 2L. Tests for allelism of the unplaced restorers on chromosome 2L with *Rf3*, *rfl1*, *rfl2* and *rfn1* are underway. Thus, represented among the cluster of restorers on 2L is the entire range of types observed to occur: dominant and recessive; naturally occurring, spontaneously arising and transposon induced; homozygous viable, homozygous lethal and nonfunctional.

By genetic means, we can estimate the distance between *Rf3* and the other restorers on 2L. Plants carrying *Rf3* are intercrossed with plants carrying one of the new restorers. The resulting kernels are planted and, at maturity, pollen is examined to identify plants carrying both restorers. These are plants exhibiting 100%, or nearly 100%, normal pollen. Such plants are testcrossed with pollen from a normal-cytoplasm nonrestoring tester plant, and at least 200 kernels from each ear are planted. The resulting plants are scored for male sterility vs. male fertility by tassel examination at maturity. Fertile tassels are cut off and sterile or immature tassels are left. The plants are scored at least every other day until all reach maturity. The number of male-fertile plants is determined by counting the detasseled plants. The number of male-sterile plants is determined by counting the plants with tassels remaining.

Through use of this method, we have determined the positions of *rfn1*, *rfv*-991181*, *rfl1*, and *rfl2* relative to *rf3*. *rfn1* and *rfv*-991181* are very closely linked to *rf3*. *rfn1* and *rf3* exhibit about 1.5% recombination, while *rfv*-991181* and *rf3* exhibit about 1% recombination. We cannot determine whether these two restorers are on the same side of the *rf3* locus by this technique. The *rfl1* and *rfl2* loci each exhibit approximately 10% recombination with *rf3*. We cannot determine the order of these two loci, but they are on the same side of the *rf3* locus.

Interestingly, two restorers for CMS-T maize, *Rf8* and *Rf**, either one of which can substitute for *Rf1* to partially restore fertility, are either alleles or tightly linked genes on 2L near the *rf3* locus (Wise and Pring, Proc. Natl. Acad. Sci. USA 99:10240-10242, 2002; D. Pei and R. P. Wise unpublished observations).

We propose to exploit our collection of restorer alleles for CMS-S maize residing on the long arm of chromosome 2 (2L) in

order to characterize and clone nuclear genes that function in mitochondrial gene expression. In CMS-S maize, the biological system, the genetics, and the genomics tools come together, providing an unprecedented opportunity to conduct the molecular-genetic dissection of mitochondrial function in a higher eukaryote.

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Maize pachytene chromosome images straighten through use of digital software

-- Carvalho, CR and Saraiva, LS

The study of meiotic chromosomes of maize (*Zea mays* L.) in the pachytene stage has provided a great deal of information and allowed cytogeneticists to clarify many of the main genetic mechanisms. However, the benefits of long morphology also have created problems in analysis due to the sinuosity and overlapping in this meiotic stage. With the advances in the development of digital tools for systems of image analysis associated with cytogenetics, the morphology of the chromosomes has been more accurately resolved.

The objective of the present paper was to utilize a digital tool with an algorithm of linearity of curvilinear structure to improve the study of maize pachytene chromosomes. Each bivalent was straightened and arranged as a karyogram. Spikes previously selected in the pachytene stage were fixed in a fresh methanol-acetic acid (3:1) solution and stored at –20 C. The anthers were mechanically macerated with a mini-mixer and the pollen grain mother cells (PMC) recovered after being filtered in a nylon mesh (100 µm pore). PMCs were macerated in 10 µL enzymatic solution (Flaxzyme™) plus 140 µL distilled water at 35 C for 60 minutes. After washing in 0.075 M KCl solution and centrifuging, the PMCs were fixed again. A cell dissociation technique, dripping of the suspension and air-drying (Caixeta and Carvalho, Cytologia, 66:173-176, 2001), was utilized for slide preparations. The material was stained with 3% Giemsa solution.

The pachytene images were captured with an immersion objective (100 X) and with a video camera (CCD) attached to an Olympus™ BX 60 and digitalized in a Macintosh™ (G4) computer. The images of each bivalent were individualized at pachytene using the software SXM-Image (Barrett and Carvalho, Chromosome Research, 11:83-88, 2003) for application of the digital straightening tool for curvilinear objects.

The chromosomes at pachytene (Figure 1) were considered adequate to test the tool for straightening curved objects. Figure 2 shows the pachytene chromosomes straightened with the use of the digital tool with an algorithm for the straightening of sinuous structures. It can be observed in this figure that after application of the straightening tool the chromosomes did not lose their longitudinal pattern and the original morphology.

This tool also allowed idiogram assemblage of pachytene chromosomes straightened (Figure 3). This idiogram represents the 10 bivalents at pachytene, emphasizing the knob regions, the nucleolus organizer region (NOR) on bivalent number 6 and the linear arrangement of the chromomeres. This methodology was adequate for application in meiotic karyogram assemblages in routine, as well as in comparative,

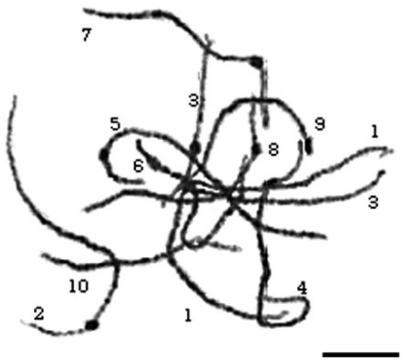

Figure 1. Maize pachytene chromosome obtained by air-drying technique with enzymatic maceration and stained with Giemsa solution. Bar = 5 µm.

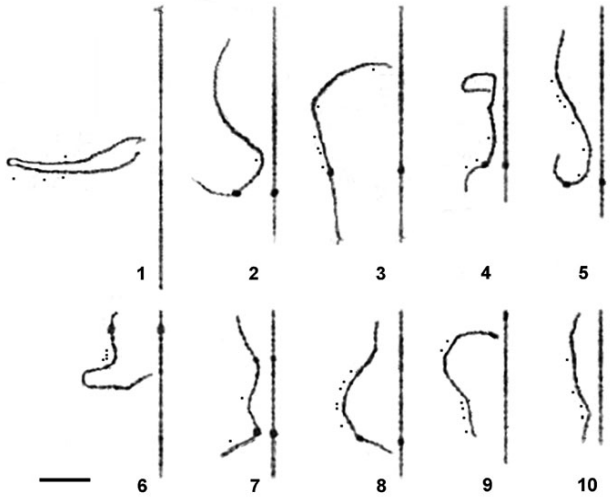

Figure 2. Maize karyogram of pachytene chromosomes isolated by digital process (from figure 1), showing original shape (left) and after application of the straightening tool (right). The black spots correspond to the overlapping chromosome regions. Bar = 5 µm.

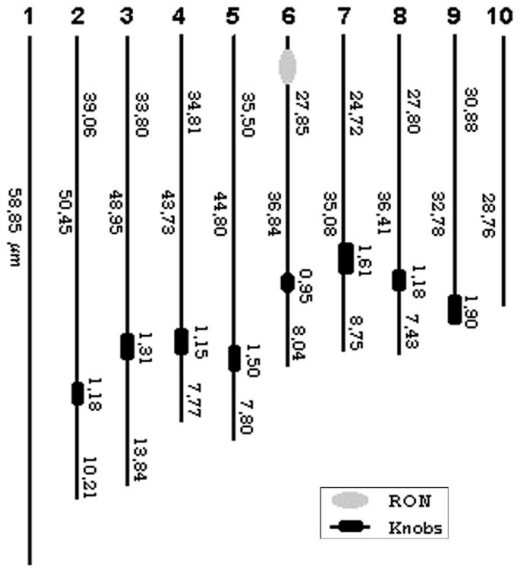

Figure 3. Idiogram of the 10 bivalents containing measures and some specific features of each straightened chromosome. NOR = nucleolus organizer region.

analyses of structural variations, chromomeric mapping and banding pattern among bivalents. This work was supportated by a grant from the FAPEMIG, CNPq, Brazil

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Colors and their role in survival

--Galinat, W

Plants may absorb certain rainbow colors after the evolution of genes necessary for their synthesis because some colors are part of certain vitamin chemistry essential to either or both the plant and/or its herbivore consumer, as pointed out by Jeff Cole. A good example of that is vitamin A derived from the carotene in the roots of carrots and in the endosperm of yellow corn kernels. Vitamin A deficiency results in night blindness and degeneration of skin tissue as occurs in elderly people. Certain colors of solar radiation may have deadly effects on exposed living things. The best example is ultra-violet (U.V.) radiation, which in unprotected white skin causes sunburn and sometimes skin cancer. Protection from U.V. damage is necessary. U.V. resistance may come from purple-violet colors in the target which reflects those colors away so the would be target survives and the radiation is blocked. Here on Earth, life is usually protected from dangerous U.V. radiation by an ozone layer of pale blue gas extending from nine to 18 miles high. In blocking U.V. radiation from the sun, it also tends to maintain seasonal temperatures on Earth. Purple colors in food plants such as cabbage, lettuce and even corn will give the reflective type of U.V. resistance. In people, purple clothes and suntan lotions may prevent or reduce sunburn on the skin. White people seem to object to coloring their skin purple but melanin is equally effective and survival of the fittest has blessed many people with this, especially those from tropical areas. In artic areas, people seem to need the U.V. radiation to photosynthesize vitamin D, giving strong bones to fight a cold rugged environment.

In plants, especially trees, red and yellow color, as seen in the beautiful autumn leaves in the northern United States and Canada, seems to be effective in attracting the red and yellow heat rays of the sun sufficiently to extend the growing season, especially for Maple trees, so that the range of their habitat extends northward so much that its leaves have become a Canadian symbol. In another example, the leaves of corn races adapted to cold high elevations in parts of Mexico and South America have become inherently red, apparently to solve the same heat problem.

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Conversions of the interchanges in reciprocal chromosomal translocations in maize to homozygosity for linkages to genes

--Findley, WR, Jr., Jones, M

Reciprocal chromosomal translocations have been successfully used to establish linkage relationships between translocation breakpoints and the genes that control important traits such as blight and virus resistance (Findley et al., Crop Sci. 18:608-611, 1973; Jenkins et al., Agron. J. 49:197-201, 1957). In 1946 the Agricultural Research Service, U. S. Department of Agriculture and the California Institute of Technology established a cooperative project to irradiate maize seeds during the atomic bomb explosion on the Bikini atoll. From this project, a large number of irradiated maize materials containing chromosomal translocations and chromosomal rearrangements were released and are available from the California Institute of Technology, Pasadena, California 91109 and the Maize Genetics Stock Center, S-123 Turner Hall, 1102 South Goodwin Avenue, Urbana, IL 61801-4798 (Longley, ARS 34-16, 1961; Freeling and Walbot, The Maize Handbook, Springer-Verlag, NY, p. 364, 1994). However, use of these stocks in linkage studies has been tedious and slow because microscopic examinations of pollen grains were needed to determine which plants carried the translocations.

In a heterozygous plant at the pachytene stage of microsporogenesis, chromatids with the reciprocally translocated chromosome segements pair with the homologous chromosome segements of their normal counterparts. This pairing of homologous segements leads to a configuration of a cross in the chromatids with the translocation breakpoints located at the center. Disjunction of the chromatids may occur along alternate or adjacent planes resulting in spores where one fourth of them are homozygous translocations, one fourth are homozygous normals, and one half contain unbalanced chromosome complements. Pollen grains with these unbalanced chromosomes do not accumulate starch and appear clear when viewed with a 40X microscope. This phenomenon was used to identify plants heterozygous for the translocation. These plants were self pollinated to obtain progeny plants that segregated as homozygous translocations, homozygous normals, and heterozygous translocations. When these progeny plants were testcrossed to a normal line, the homozygous translocations were differentiated from the homozygous normals by the 50% seed set of the semi-sterile progeny versus the completely fertile progeny. To avoid microscopic examination of pollen grains and simplify the selection process for useful genotypes, all translocations were converted to homozygosity.

Most of the translocation stocks listed in Table 1 were obtained from Dr. W. A. Russell, Iowa State University in the early 1960's. Additional stocks were later obtained from the Maize Genetics Stock Center, Urbana, Illinois. Conversion to homozygosity of the translocation stocks was initiated during the season of 1985. During the conversion each stock was backcrossed to inbred M14 a sufficient number of times to obtain at least 10 doses of the recurrent parent. Marker genes *su1, y1 o2,* and *wx* were incorporated into the stock translocations and inbred M14 to mark the short arm of chromosome 4 (near centromere), long arm of chromosome 6 at .10, short arm of chromosome 7 at .16 and the short arm of chromosome 9 at .56. Some stocks were developed by repeated backcrosses to semi-sterile types; others by sib-mating semi-sterile types to plants classified as homozygous normal. In the development of these latter stock translocations, a close linkage with the translocation or marker gene was assumed. However, no amount of backcrossing to the recurrent parent would overcome linkages between blocks of genes that are close to points of chromosomal interchange (Jenkins et al., 1957).

Table 1. Reciprocal chromosomal translocations homozygous for the interchange.

Translocation	Interchange
$y_1M14^{11*}T1\text{-}6c$	1S.25-6L.27
$wM14^{10}T1\text{-}9c$	1S.48-9L.22
$su_1M14^{10}T1\text{-}4a$	1L.51-4S.69
$su_1M14^{10}T1\text{-}4d$	1L.27-4L.30**
$wxM14^{10}T2\text{-}9b$	2S.18-9L.22
$wxM14^{10}T2\text{-}9c$	2S.49-9S.33
$wxM14^{10}T2\text{-}9d$	2L.83-9L.27
$su_1M14^{11}T2\text{-}4l$	2L.59-4S.40**
$o_2M14^{11}T2\text{-}7c$	2L.47-7S.34
y_1 $M14^{10}T3\text{-}6c$	3S.56-6L.54
$wxM14^{10}T3\text{-}9(8447)$	3S.44-9L.14
$wxM14^{10}T3\text{-}9(6722)$	3S.66-9S.66
wx $M14^{10}T3\text{-}9b$	3L.48-9L.53
$wxM14^{10}T3\text{-}9g$	3L.40-9L.14
$su_1M14^{10}T4\text{-}5(5529)$	4S.37-5L.46
$su_1M14^{10}T4\text{-}7(48\text{-}40\text{-}8)$	4S.32-7L.64
$su_1M14^{10}T4\text{-}8a$	4S.59-8L.19
$su_1M14^{10}T4\text{-}8(5339)$	4S.22-8L.71
$wxM14^{10}T4\text{-}9e$	4S.53-9L.26
wx $M14^{10}T4\text{-}9g$	4S.27-9L.27
$su_1M14^{16}T4\text{-}5j$	4L.36-5L.36**
$y_1M14^{10}T4\text{-}6a$	4L.37-6L.43
$su_1M14^{10}T4\text{-}10(6587)$	4L.55-10L.51
$o_2M14^{10}T5\text{-}7(064\text{-}18)$	5S.61-7S.49
$wxM14^{10}T5\text{-}9(8854)$	5S.33-9S.36
$o_2M14^{10}T5\text{-}7c$	5L.42-7L.72
$wxM14^{10}T5\text{-}9a$	5L.69-9S.17
$wxM14^{12}T6\text{-}9a$	6S.79-9L.40
$y_1M14^{11}T6\text{-}9d$	6S.73-9L.82
$y_1M14^{10}T6\text{-}10(5253)$	6S.80-10L.41
$y_1M14^{10}T6\text{-}7(4594)$	6L.52-7S.67
$wxM14^{12}T6\text{-}9b$	6L.10-9S.37
$wxM14^{11}T7\text{-}9b$	7S.76-9S.19
$o_2M14^{10}T7\text{-}8(038\text{-}8)$	7L.52-8L.46
$wxM14^{10}T7\text{-}9a$	7L.63-9S.07
$wxM14^{11}T7\text{-}9(027\text{-}9)$	7L.61-9S.18
$o_2M14^{10}T7\text{-}10(019\text{-}3)$	7L.17-10L.47
$wxM14^{10}T8\text{-}9b$	8S.67-9L.75
$wxM14^{11}T8\text{-}9(4643)$	8S.37-9L.11
$wxM14^{10}T8\text{-}9(6673)$	8L.35-9S..31
$wxM14^{10}T8\text{-}9(6921)$	8L.85-9L.15
$wxM14^{11}T9\text{-}10b$	9S.13-10S.40
$wxM14^{10}T9\text{-}10(059\text{-}10)$	9S.31-10L.53
$wxM14^{10}T9\text{-}10(8630)$	9S.28-10L.37
$wxM14^{11}T9\text{-}10(4303\text{-}9)$	9L.26-10S.44

*Doses of M14
**Breakage points reported in Freeling and Walbot (1994). Other breakage points in A. E. Longley (1961).

The use of these converted translocations for linkage studies can be enhanced by the following procedures. Cross lines to a series of translocation stocks to mark each chromosome arm at least twice. Use F1's or backcrosses to inbred M14 with an appropriate marker gene to express the trait and show linkage to a chromosome arm. Use translocations with breakpoints near the middle of the arm to test the entire chromosome arm. Determine gene placement to a chromosome arm by the presence or absence of the trait and the presence or absence of the translocation. Confirm an association of a gene to a chromosome arm by comparing its reaction to other chromosome stocks that have at least one arm in common with each other. Incorporate marker genes to identify the chromosome arm involved in expression of the trait. Lastly, use additional translocation stocks with breakpoints near the calculated gene site to produce more precise determinations.

In evaluation of the data, the frequency of plants showing reaction to the studied trait will indicate the degree of linkage to a chromosomal breakpoint. The degree of linkage also may be estimated by the frequency of plants that show the expected reaction to a trait (Findley et al., 1973). When appropriate marker stocks are used in gene linkage studies, the significance of the results may be determined by analyses of mean ratings for the observed traits. Presently, tests for the whole chromosome arm are possible with the forty-five converted translocation stocks, except for chromosome 6 and perhaps 10. However, to convincingly mark the short arm of chromosomes 3, 5, 8 and 9 two other stocks such as 3S.44 and 3S.56 and 8S.37 and 8S.67 may be required. Thirty-five homozygous translocation stocks were submitted to the Maize Genetics Stock Center in 1994, and the remaining 10 in 2002. In 2003, all 45 homozygous stocks should be available from the Maize Genetics Stock Center.

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Maize Genetics Cooperation • Stock Center

USDA/ARS/MWA - Soybean/Maize Germplasm, Pathology & Genetics Research Unit

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12,413 seed samples have been supplied in response to 291 requests, for 2003. This includes one large request from a collaborator on the Maize Gene Discovery Project that totaled 6,773 packets. A total of 83 requests were received from 21 foreign countries. Approximately 90% of our requests were received by electronic mail or through our order form on the World Wide Web. Popular stock requests include the IBM RIL mapping populations, Hi-II lines, *ig1* lines, transposable element lines, and Maize Gene Discovery Project lines.

Approximately 10.5 acres of nursery were grown this summer at the Crop Sciences Research & Education Center located at the University of Illinois. Dry weather in the early spring allowed the timely planting of our first nurseries, but cool weather delayed the planting of our later nurseries. After a cool spring, growing conditions were excellent, and overall we had a good pollination season. Additional irrigation was not necessary.

Special plantings were made of several categories of stocks:

1. In the 'Phenotype Only' collection is a series of stocks donated to the Stock Center by Dr. Gerry Neuffer upon his retirement. We have made available an additional 250 stocks in this series.

2. Plantings were also made from donated stocks from the collections of Jack Beckett (B-A translocation lines), Jim Birchler (inbred tetraploid lines), Ed Coe (*ppg2*, *cb2*, and *rgh2* alleles), Hugo Dooner (*bz1* alleles), Greg Doyle (inversions), Jerry Kermicle (extensive collection of Brink's *R1* alleles), Bruce May and Rob Martienssen (*mn** mutants from the MTM project), Scott Poethig (*epc1* alleles), Margaret Smith (male sterile cytoplasm lines), Derek Styles (*Ufo1* and *P1* alleles), Bill Tracy (inbred conversions of *su1* and *sh2*), and others. New mutable *r1* haplotypes were obtained from the North Central Regional Plant Introduction Station. We expect to receive additional accessions of stocks from maize geneticists within the upcoming year.

3. We conducted allelism tests of several categories of mutants with similar phenotype or chromosome location. We found additional alleles of *pink scutellum1*, *yellow endosperm1, albescent1 and white luteus2.* In this manner, we hope to move more stocks from our vast collection of unplaced uncharacterized mutants into the main collection.

4. We conducted linkage tests of several mutants and controlling elements using *waxy1*-marked A-A translocations. A more precise location was determined for *inhibitor of r1 aleurone color2* on the long arm of chromosome 9.

5. Two acres were devoted to the propagation of the large collection of cytological variants, including A-A translocation stocks and inversions. In this collection is a series of *waxy1*-marked translocations that are used for mapping unplaced mutants. Over the years, pedigree and classification problems arose during the propagation of these stocks. We were able to sort through the problem ones, and we can now supply good sources proven by linkage tests to include the correct translocated chromosomes. Additional translocation stocks were tested this last year. Results of these tests will be reported in the next issue of the Maize Genetics Cooperation Newsletter.

6. Stocks produced from the NSF project "Maize Gene Discovery, Sequencing and Phenotypic Analysis" (see: http://zmdb.iastate.edu/ or http://www.maizegdb.org/documentation/mgdp/) were grown this summer. Approximately 30% of these represented plants that originally had to be outcrossed, and needed to be selfed to analyze for mutant segregation. The remaining 70% were seed increases that were planted from those families that originally yielded poorly. These increases help to maintain adequate seed stock to fill future requests. Additionally, we grew 3,308 families of this material to screen for new adult plant mutant traits (see below).

We received 294 IBM (B73 Mo17 intermated population) recombinant inbred lines from Mike Lee and Georgia Davis. We received enough seeds of each line to distribute directly. We are also filling requests directly from seeds of transposed *Ac* lines provided by Tom Brutnell and chromatin stocks received from Vicki Chandler.

We have received 1644 lines from the B73 and B73xMo17 EMS materials from Dr. Gerry Neuffer (Regulation of Inflorescence Architecture in Maize project). There are sufficient seed for most of them to be distributed. We also have recently received approximately 1200 lines of A619 EMS materials from Torbert Rocheford that will be ready for distribution in the near future.

We continue to grow a winter nursery of 0.5 acres at the Illinois Crop Improvement Association's facilities in Juana Díaz, Puerto Rico. We had an excellent winter crop last year, and all indications are that the crop will perform well this year after a rainy start. We plan to continue growing our winter nurseries at this location.

The NSF project "Maize Gene Discovery, Sequencing and Phenotypic Analysis" has, to date, generated 38,093 stocks that have been sent to the Stock Center. All of these stocks were screened for ear and kernel mutants at the Stock Center; families with ample seed supplies had two samples removed for additional trait screening. The first sample from each family was sent to UC Berkeley for seedling mutant screening. From the second sample, 3,308 families of the most genetically active grids were planted and screened (by us, other project members and colleagues in the Maize Genetics community) at the University of Illinois for adult plant mutant traits. This was an organized mutant hunt and was very successful in the discovery of novel adult plant mutants. We plan to organize another mutant hunt next summer. The remaining seed generated by this project was placed into cold storage to fulfill requests. Results from the mutant screenings can be found at the MaizeGDB RescueMu Phenotype Database (http://www.maizegdb.org/rescuemu-phenotype.php). Future work will involve increasing stocks as necessary to maintain seed supply for requests and continue scoring these stocks for kernel and adult plant mutant traits.

Early this year, we hired Jason Carter as a new IT Specialist. We also purchased and deployed a state-of-the-art Apple Xserve, which now serves as a central repository for all of the Stock Center's data, providing greater flexibility and data security. We are now actively developing a robust, relational database for our stock inventory, as well as powerful, easy-to-use tools for maintaining, using, and distributing the data it stores. We are also cooperating with the MaizeGDB.org project in Ames, IA to provide it with a direct feed of information from our database. We have also worked with the MaizeGDB team to enhance our on-line stock listings and provide a shopping-cart style ordering system. See http://www.maizegdb.org/stock.php for stock search and browsing tools that are now provided by MaizeGDB.

Marty Sachs Philip Stinard Janet Day Jackson Shane Zimmerman Jason Carter

CHANGES TO OUR CATALOG OF STOCKS SINCE MNL77
(For a complete list of our stocks, see: http://maizegdb.org/cgi-bin/stockcatalog.cgi)

CHROMOSOME MARKERS

104E ids1-rgoVI
104H ids1-Burr
106A ppg2-N7A
107C P1-rw
204E rgh2-N1013A
206F ms33-6052
206G ms33-6054
218DA Ht1-Ladyfinger
225D Tertiary trisomic 1; v4 w3; B-1Sb-2L(4464) V4 W3
225E Telo2 w3; W3
318A ig1
328B Tertiary trisomic 3; B-3Sb Cl1; cl1
328C Tertiary trisomic 3; y10 a1; B-3Lc Y10 A1
333D sh2
407D su1
411C su1-N1161A
503G ms13-6060
503H ms5-6048
503I ms5-6061
503J ms5-6062
525A ps1-84-5266-5
525B ps1-85-3011-11
525C ps1-Mu85-3061-21
525D ps1-85-3135-4
525E ps1-85-3182-6
525F ps1-85-3288-28
525G ps1-85-3422-13
525H ps1-85-3492-36
525I ps1-85-86-3567-1
525J ps1-Mu86-1105-1
525K ps1-86-1105-2
525L ps1-86-1449-3
525M ps1-86-1352-4
525N ps1-86-1361-7
525O ps1-86-1499-3
526A ps1-86-1565-17
526B ps1-86-87-1742-18
526C ps1-90-3222-27
526D ps1-90-91-8549-7
526E ps1-96-5032-6
526F ps1-98-5691-5
526G ps1-99-2157-1
602C y1
602I y1-gbl1-N547B
604G y1 si1-at
608K ms50-6026
608L ms50-6055
610G hcf34-N1269C
614D Tertiary trisomic 6; w15 y1; B-6Ld W15 Y1
708BA bd1-2
712C ms22
713A Bn1 y1
727BB wlu2-N629A
803C gl18-5249
811A ms23-6011
811B ms23-6018
811C ms23-6027
811D ms23-6031
811E ms23-6059
811F ms23
811G d12-N203D
906I ms25-6057
906J ms25-6065
906K ms48-6049
932A Bz1-W22
932B bz1-E1
932C bz1-E2
932D bz1-E3
932E bz1-E4
932F bz1-E5
932G bz1-E6
932H bz1-E7
932I bz1-E8
932J bz1-E9
932K bz1-E10
932L bz1-m2(D3)
932M bz1-m2(D4)
932N bz1-m2(D5)
932O bz1-S2114(Ac)
932P Bz1(Dp26)
932Q bz1-m(Dp26)-m1
932R bz1-R
932S C1 sh1 bz1-R wx1
X03H ms24
X03I ms47-6039
X13H r1-g(Stadler)
X20K R1-g:1
X20L R1-g:8
X20M R1-g:8:pale
X20N R1-ch(pale):g5 wx1
X20O R1-r(Venezuela694#16037) arv-m694
X20P R1-st(Bolivia494)
X20Q R1-st(Bolivia707)
X20R R1-st(Bolivia706)
X20S R1-st(Chile370)
X20T R1-st(Argentina216/62)
X20U R1-st(Bolivia781)
X234Q R1-r(Venezuela530-PI302354)
X235B R1-r(Venezuela594-PI302363); arv-m594
X236G R1-g(New Mexico-PI218153)
X24D R1-st(Laughnan)
X24E R1-st(Peru715)
X24F R1-st(Chile369)
X24G R1-st(Bolivia1520)
X24H r1-sc:m6::Ds
X24I r1-g(Nc)3-5
X29F r1-m(Ames17331)
X29G r1-m(PI485274)
X29H r1-m(PI571748)
X29I r1-m(PI571802)
X29J r1-m(PI572146)
X29K r1-m(PI572173)

UNPLACED MUTANT

U740J vp14-2274

TOOLKIT

T3301Q mon00004::Ac
T3301ZB mon03078::Ac
T3301ZC mon03082::Ac
T3301ZE mon00038::Ac
T3301ZF mon00060::Ac
T3301ZG mon00092::Ac
T3301ZH mon00152::Ac
T3301ZI mon00164::Ac
T3301ZJ mon00178::Ac
T3301ZK mon00186::Ac
T3301ZL mon00192::Ac
T3301ZM mon00236::Ac
T3301ZN mon00238::Ac

CHROMDB STOCKS

3201-01 T-MCG3348.02
3201-02 T-MCG3955.01
3201-03 T-MCG3361.01
3201-03.1 T-MCG3361.05
3201-03.2 T-MCG3361.08
3201-04 T-MCG3571.03
3201-05 T-MCG2822.01
3201-06 T-MCG3322.04
3201-07 T-MCG3480.04
3201-08 T-MCG3772.05
3201-09 T-MCG3571.11
3201-10 T-MCG3571.37
3201-11 T-MCG4268.12
3201-12 T-MCG4268.16
3201-13 T-MCG4268.23
3201-14 T-MCG3818.11
3201-15 T-MCG3818.15
3201-16 T-MCG3818.18
3201-17 T-MCG3818.19

PHENOTYPE ONLY

adherent leaf
3609E ad*-N503
3610I ad*-N785B

albescent seedling
3611B al*-N427B

albino seedling
3507F w*-N172
3506H w*-N32

brittle endosperm
5603A bt*-Alexander

brown kernel
3606G bnk*-N1314A

brown midrib
5803J bm*-86-87-8875-6
5803K bm*-2001PR-1

collapsed endosperm
3601O cp*-N1055B
3602E cp*-N1170A
3603L cp*-N1419
3603O cp*-N1517C
3603Q cp*-N1541
3601J cp*-N948A

crinkly
3708R Cr*-N1454
3708S cr*-N2326
3708T cr*-N2329

cross banded
3708E Cb*-N1456
3708G cb*-N1517A
3708H cb*-N1519A
3708K cb*-N1959

defective crown
3605I dcr*-N1402A

defective kernel
338-40 dek*-MTM8237
338-41 dek*-MTM8333
438-07 dek*-MTM11230
3706B de*-N1194C
3701D de*-N293C
3702G de*-N878A
3703L de*-N1004A
3703M de*-N1006
3704A de*-N1014A
3704B de*-N1025A
3704D de*-N1035
3704E de*-N1051A
3704H de*-N1081
3704M de*-N1118A
3705C de*-N1139
3705D de*-N1141
3705N de*-N1181
3706D de*-N1221A
3706J de*-N1342B
3707B de*-N1367A
3707C de*-N1370
3707E de*-N1395A
3707I de*-N1408A
3707J de*-N1413
3702K de*-N921A
3703B de*-N944A
3703C de*-N947A
3703F de*-N958
5609C dek*-74-0060-4-Alex

dented kernel
3606B dnt*-N1064A

discolored kernel
3605P dsc*-N1321A
3605R dsc*-N1580A

distorted
4008B dst*-N196
4008BA dst*-N398B
4008D dst*-N676
4008E dst*-N1190B

dwarf
4404H d*-N720F
4402M d*-N307C
4405A d*-N1013B
4405F d*-N1219B
4405H d*-N1296B
4405I d*-N1319C
4405J d*-N1336C
4405L D*-N1452
4405M D*-N1591
4406F d*-N2252
4406J d*-N2256
4310M ty*-N1315C

etched
3804D et*-N1048
3802I et*-N634C
3804F et*-N1137A

flecked leaf
3411H flk*-N537B
4008NA flk*-N629E

floury
3705A de*-N1128
3901C fl*-N904A
3901G fl*-N1225A

germless
3807K gm*-N1488B
3807L gm*-N1532

glossy leaf
411D gl*-N1827B
512S gl*-N1843C
512Q gl*-N2476
512R gl*-N531B

green stripe
4009K gs*-N921B
4009M gs*-N1164D
4010D gs*-N2335

hairy sheath
4011E Hs*-N1539
4011F Hs*-N1610

knotted
4011M Kn*-HsuA1191

lesion
3908G les*-N2012
3908H les*-N2013
3908I les*-N2015
3908N Les*-N2320
3909H Les*-NA7145

luteus yellow seedling
3810I l*-N1206B
3810K l*-N1253C
3811C l*-N1974
3809M l*-N447A
3809O l*-N507B
3808C l*-N61B
3810G l*-N939C

many tillers
4209B tlr*-N2244

miniature kernel
3807S Mn*-N2423A
138-01 mn*-MTM181
138-02 mn*-MTM520
138-04 mn*-MTM534
138-05 mn*-MTM787
138-06 mn*-MTM791
138-07 mn*-MTM794
138-08 mn*-MTM801
138-09 mn*-MTM1232
138-10 mn*-MTM1595
138-11 mn*-MTM2009
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138-16 mn*-MTM2798
138-17 mn*-MTM2803
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138-19 mn*-MTM4286
138-20 mn*-MTM4290
138-23 mn*-MTM4517
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138-40 mn*-MTM4887: lp*-4887
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238-04 mn*-MTM5178
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238-06 mn*-MTM5222
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338-46 mn*-MTM10149
338-47 mn*-MTM10162
338-48 mn*-MTM10828
438-02 mn*-MTM11125
438-08 mn*-MTM11267: wrk*-MTM11267
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638-23 mn*-MTM19011
638-24 mn*-MTM19065
638-25 mn*-MTM19190
638-26 mn*-MTM19374
638-27 mn*-MTM19758
638-28 mn*-MTM19760
638-29 mn*-MTM20315
638-30 mn*-MTM20748
638-31 mn*-MTM20789
638-32 mn*-MTM21039
638-35 mn*-MTM21188
638-36 mn*-MTM21227
638-37 mn*-MTM21234
638-38 mn*-MTM21670
638-43 mn*-MTM21782
638-44 mn*-MTM21787
638-45 mn*-MTM21788
638-48 mn*-MTM21991
638-49 mn*-MTM22116
638-50 mn*-MTM22152
638-52 mn*-MTM22320
638-53 mn*-MTM22426
638-54 mn*-MTM22433
638-55 mn*-MTM22438

narrow leaf
3910C nl*-N323A
3910N nl*-N631
3911D nl*-N685A
3911L nl*-N1965
3911N Nl*-N2422

necrotic
4101N nec*-N291
4102J nec*-N473A
4104C nec*-N720C
4104O nec*-N1613
3709F stk*-N198A

opaque
3902D o*-N883A
3902M o*-N933
3903I o*-N1009
3903J o*-N1016
3903L o*-N1034
3904B o*-N1043
3904D o*-N1049
3904H o*-N1096A
3904J o*-N1103A
3904O o*-N1131A
3905B o*-N1148A
3905G o*-N1194A

3905I o*-N1205
3906A o*-N1242A
3906E o*-N1247
3907F o*-N1368
3907H o*-N1384A
3907I o*-N1388A

pale green
4203D pg*-N267
4203G pg*-N287A
4203H pg*-N293A
4203K pg*-N300
4203N pg*-N343
4204D pg*-N371A
4204O pg*-N422A
4205O pg*-N505
4207N pg*-N668
4302C pg*-N1087B
4302D pg*-N1103B
4302G pg*-N1180B
4302H pg*-N1185C
4302L pg*-N1269B
4302M pg*-N1277A
4303B pg*-N1379B
4303E pg*-N1483C
4303F pg*-N1549
4303N pg*-N1846A
4304F pg*-N2265

pale-pale green
4305K ppg*-N1082B
4305L ppg*-N1238B
4305M ppg*-N1248D

patched leaf
4105D ptc*-N522A
4105F ptc*-N724A

pygmy
4408B py*-N2238

ragged
4106H rgd*-N2334

rough
3806O rgh*-N1325
3806P rgh*-N1351

rolled leaf
3912G rld*-N956B

scarred kernel
3806S scr*-N1363A

semi-dwarf
4408H sdw*-N2299

shredded
4107A Shr*-N2477

shrunken
4006J sh*-N1178A
4006O sh*-N1337
4007A sh*-N1340
4007C sh*-N1364A
4007E sh*-N1421

small kernel
3705I de*-N1153
3707F de*-N1396A
4001B smk*-N257A
4001C smk*-N283C
4001D smk*-N304B
4002A smk*-N1017A
4002E smk*-N1036
4002H smk*-N1070
4002I smk*-N1072
4002J smk*-N1079
4002K smk*-N1082A
4002M smk*-N1087A
4003B smk*-N1140
4003D smk*-N1146A
4003H smk*-N1182A
4003I smk*-N1184
4003J smk*-N1187A
4003M smk*-N1236
4004A smk*-N1372
4004C smk*-N1382
4004G smk*-N1580B

small plant
4410E smp*-N706A
4410L smp*-N1143B
4408O smp*-N114B
4402N d*-N309
4404C d*-N597A
4410I smp*-N866
4409F smp*-N216
4409G smp*-N240

small seedling
4411O sms*-N479
4412E sms*-N687B
4412F sms*-N782
4412G sms*-N815B
4412H sms*-N1051B
4412I sms*-N1055C
4412K sms*-N1146B
4412M sms*-N1244C
4412N sms*-N1396B

speckled kernel
3805L spk*-N1263B

streaked leaf
3711F stk*-N1402B

striped leaf
3709EA str*-N424B
3711G stp*-N1475

terminal ear
4012O te*-N2293

unbranched
4012W ub*-N2263

virescent
4010B gs*-N2258
4305D pgv*-N51A
4501B v*-N13
4501C v*-N15A
4502O v*-N139A
4502P v*-N142 vir
4503F v*-N183A
4504A v*-N284
4504I v*-N354B
4504N v*-N391A
4506F v*-N608C
4509E v*-N1024B
4509G v*-N1064B
4509I v*-N1090B
4509J v*-N1114B
4509N v*-N1137B
4510F v*-N1316B
4510G v*-N1364B

viviparous kernel
5907G y-vp*-Alex 68-195

white luteus
3708L wl*-N1962
4112F wl*-N1118B
4112G wl*-N1123B
4112H wl*-N1138B
4112I wl*-N1148B
4112J wl*-N1170B
4112L wl*-N1261C
4208A wl*-N1373C
4208C wl*-N1393B

white stripe
4211C wst*-N1466
4211E wst*-N1472

white tipped leaf
4211N wt*-N662A

wrinkled kernel
3606R wr*-N1300A

yellow green
4307G yg*-N662B
4307L yg*-N1234E
4308A Yg*-N1582
4308B Yg*-N1584
4308H yg*-N1832
4309A yg*-N2269
4309E yg*-N2328

yellow stripe
3812L ys*-N2303

CATALOG OF STOCKS

CHROMOSOME 1 MARKERS

101A sr1 zb4 p1-ww
101B sr1 P1-wr
101C sr1 p1-ww
101D sr1 P1-rr
101F sr1 ts2 P1-rr
102A Ws4-N1589
102D Blh1-N1593
102F ms28
102G zb3
102H hcf6-N228B
102I hcf7-N1029D
103D vp5
103DA vp5-DR3076
103DB vp5-86GN4
103DC vp5-86GN3
103DD vp5-86GN6
103DE vp5-86GN11
103DF vp5-Mumm-1
103DG vp5-N81
103E zb4 ms17 p1-ww
104A Ts3
104F ms*-6034
104G ms*-6044
105A zb4 p1-ww
105B zb4 P1-wr
105C zb4 p1-ww br1
105E ms17 P1-wr
105F ms17 p1-ww
106B ts2 P1-rr
106C Glb1-0
106D Glb1-0; Glb2-0
106E ts2-N2409
106F wlu7-N1930
106G v35-N55
106H rgd3-N766B
107A P1-cr
107B P1-rr
107C P1-rw
107D P1-cw
107E P1-mm
107F P1-vv::Ac
107G P1-or
107H p1-ww
109A gs1-PI228173
109B gs1-PI262495
109C gs1-PI267181
109D P1-rr ad1 bm2
109E P1-wr br1 f1
110A P1-wr an1 Kn1 bm2
110D P1-wr an1 bm2
110E P1-wr ad1 bm2
110F P1-wr br1 Vg1
110H P1-wr br1 f1 bm2
110K P1-wr br1
111B hcf3-N846B
111C hcf3-N1242B
111D hcf44-N1278B
111F Les20-N2457
111G rs2
111H Les5-N1449
112B p1-ww br1 f1 bm2
112E as1
112H p1-ww br1
112I p1-ww br1 gs1 bm2
113B rd1
113BA rd1-Wasnok
113C br1 f1
113E br1 f1 Kn1
113K hm1; hm2
113L Hm1; hm2
114C br1 bm2
114D Vg1
114E Vg1; su1
114F br2 hm1; Hm2
114G br2 hm1; hm2
115C v22-8983
115CA v22-055-4
115D bz2; A1 A2 C1 Pr1 R1
115E bz2-mVW2::Mu1
115F bz2-mVW4::MuDR
115J bz2-m::Ds; A1 A2 C1 C2 Pr1 R1
116A bz2-m::Ds; A1 A2 Ac C1 C2 Pr1 R1
116C an1 bm2
116D def(an1..bz2)-6923; A1 A2 Bz1 C1 C2 Pr1 R1
116G an1
116GA an1-93W1189
116I bz2 gs1 bm2 Ts6; A1 A2 Bz1 C1 C2 R1
117A br2
117D tb1
117DA tb1-8963
117E Kn1

118B Kn1 bm2
118C lw1
118CA lw1-3108
118CB lw1-6474
118J Adh1-3F1124r53
118K Adh1-1S5657; Adh2-33
118L Adh1-3F1124::Mu3
118M Adh1-3F1124r17
118N Adh1-IL14H; su1
118O Adh1-Cm
118P Adh1-FCm
118Q Adh1-Ct
119A Adh1-1S; Adh2-1P
119B vp8
119C gs1
119D gs1 bm2
119E Ts6
119F bm2
119H Adh1-FkF(gamma)25; Adh2-N
119J Adh1-Fm335::Ds1
119K Adh1-Fm335RV1
119L Adh1-2F11::Ds2
119M Adh1-1F725
120A id1
120B nec2-8147
120C ms9
120CA ms9-6032
120CB ms9-6037
120CC ms9-6042
120D ms12
120E v22-055-4 bm2
120F Mpl1-Sisco
120G Mpl1-Freeling
121A ms14
121AA ms14-6005
121B br2-mi8043
121C D8
121D lls1
121DA lls1-N501B
121E ty*-8446
121G ct2
121GA ct2-rd3
124A v*-5688
124B j*-5828
124C w*-8345
124CA w*-013-3
124CB w*-8245
124D v*-5588
124E w*-018-3
124F w20-4791
124G w*-6577
124H w24-8054
124I v32-032-3
124J v*-8943
125A Les2-N845A
125B Mpl1-Jenkins
125C hcf13-N1097B
125D hcf41-N1275C
125E hcf50-N1481
125F hcf2-N506C
125G hcf31-N1268B
126A bz2 gs1 bm2; A1 A2 Bz1 C1 C2 R1
126B id1-N2286A
126C dek1-N928A
126D dek1-N971
126E dek32-N1322A
126F o13
126H P1-vv::Ac bz2-m::Ds
126I P1-vv::Ac
126J P1-ww-1112
126K P1-ovov-1114
126L P1-rr-4B2
126M P1-vv-5145
126N dek1-N1348
126O dek1-N1394
126P dek1-N1401
127A bz2 zb7-N101 bm2
127B dek1-N792
127C dek2-N1315A
127D dek22-N1113A
127E f1
127F Msc1-N791A
127G Tlr1-N1590
127I gt1
128A ij2-N8
128B l16-N515
128C l17-N544
128D pg15-N340B
128E pg16-N219
128G py2-N521A
128H spc2-N262A
129A w18-N495A
129AA w18-571C
129B wlu5-N266A
129C zb7-N101
129D emp1-R
129E ptd1-MS1568
129F dek*-MS2115
129G dek*-MS6214
130A o10-N1356
130B cp3-N888A
130BA cp3-N888A; mn4-N888C
130C id1-NA972
130D dek1-PB388
130E dek1-DR1129
130F ht4
6502A P1-ww-4Co63
6502C P1-ovov-CFS-29
6502D P1-rr(11)-CFS-33
6502E P1-rr(10)-CFS-36
6502F P1-rr(4-5)-CFS-47
6502G P1-rr(9)-CFS-53
6502I P1-rr(8-9)-CFS-75
6502K P1-vv-CFS-96
6502L P1-vv-CFS-110
6502M P1-vv-CFS-116
6502N P1-ovov-CFS-124
6502O P1-vv-CFS-138
6502P P1-rr(7)-CFS-140
6502Q P1-vv-CFS-155
6502R P1-o-grained-red-CFS-167
6502S P1-r pale(8)-CFS-181
6502T P1-rr(9)-CFS-186
6502U P1-vv-CFS-226
6502V P1-vv-CFS-245
6502W P1-vv-CFS-246
6502X P1-vv-CFS-249
6502Y P1-vv-CFS-252
6502Z P1-vv-CFS-255
6502ZA P1-vv-CFS-256
6502ZB P1-vv-CFS-259
6503A P1-rr(11)-CFS-272
6503B P1-vv-CFS-273
6503C P1-vv-CFS-278
6503D P1-vv-CFS-279
6503E P1-vv-CFS-281
6503F P1-vv-CFS-282
6503G P1-vv-CFS-283
6503H P1-vv-CFS-284
6503I P1-r pale(5)-CFS-285
6503J P1-vv-CFS-286 (Brazil)
6503K P1-mm-CFS-286
6503L P1-mm-CFS-287
6503M P1-mm-CFS-289
6503N P1-mm-CFS-290
6503O P1-mm-CFS-291
6503P P1-mm-CFS-292
6503R P1-mm-CFS-294
6503S P1-mm-CFS-297
6503T P1-mm-CFS-301
6503U P1-rw(9)-CFS-302
6503V P1-rr(11)-CFS-303
6503W P1-rr(10)-CFS-305
6503X P1-rr(10)-CFS-315
6503ZA P1-rr(2)-CFS-319
6503ZB P1-rr(8)-CFS-320
6503ZC P1-rr(7)-CFS-321
6504A P1-rw(8)-CFS-324
6504B P1-rw(6-7)-CFS-325
6504C P1-rr(9)-CFS-327
6504D P1-rw(7)-CFS-330
6504E P1-rw(9)-CFS-332
6504F P1-rw(8)-CFS-334
6504G P1-o-grained-red-CFS-335
6504H P1-rw(5-6)-CFS-336
6504I P1-rw(7-9)-CFS-342
6504J P1-rr(5)-CFS-345
6504K P1-rw(7)-CFS-350
6504L P1-rr-CFS-360
6504M P1-rw(5)-CFS-369
6504N P1-ww(1)-CFS-376
6504O P1-vv-CFS-497
6504Q P1-rr(11)-CFS-548

CHROMOSOME 2 MARKERS

201A mrl1-IHO
201B hcf106-Mum1::Mu1; hcf106c
201C hcf106-Mum2::Mu1; hcf106c
201D hcf106-Mum3::Mu1; hcf106c
201F ws3 lg1 gl2 b1
201G sm2-Brawn180
201H sm2-Brawn189
201I sm2-Brawn190
201J sm2-Brawn191
201K sm2-Brawn188
202A lg1-PI200299
202B lg1-PI262493
202C lg1-32TaiTaiTaSarga
202D lg1-ZCXGRB
202E lg1-64-4
202F fl1-o8
202G lg1-56-3037-5
202H Gn1-R
202I Gn1-DS
202J lil1-1
203B al1
203BA al1-Brawn
203BB al1-y3
203C al1-1998-2
203D al1 lg1
203G al1-y3 gl2
204A al1-lty3
204B hcf1-N490B
204C Wab1
204D B1'; mop1-1
205A al1 lg1 gl2
205B lg1
205C lg1 gl2
205G al1 gl2 B1
206A lg1 gl2 B1
206C D10-N2428
206D Wrp1-NA1163
206E oro2
207A w3-y11
207B ts1-0174
207C ts1-Anderson
207D ts1-69-Alex-MO17
208B lg1 gl2 B1 sk1
208C lg1 gl2 B1 sk1 v4
208D lg1 gl2 B1 v4
208E lg1 gl2 b1
208H gl2-Salamini
209A gl11-N352A
209E lg1 gl2 b1 sk1
209I gl2-Parker's Flint
210E gl2-3050-3
210F gl2-PI200291
210G gl2-PI239114
210H gl2-PI251009
210I gl2-PI251885
210J gl2-PI251930
210K gl2-PI262474
210L gl2-PI262493
210N gl2-N718
210O gl2-N239
211A lg1 gl2 b1 fl1
211H gl2 wt1
212B lg1 gl2 b1 fl1 v4
212D lg1 gl2 b1 v4
213B lg1 gl2 wt1
213F lg1 B1-v::Bg Ch1
213H lg1 gl2 B1-v::Bg
214A wt1-PI251939
214B lg1 b1 gs2
214C d5
214D gl11 B1
214E B1 ts1
214J sk1
214L lg1 gl2 mn1
215A gl14
215B gl11
215C wt1
215CA wt1-N472A
215CB wt1-N666B
215CC wt1-N178C
215CD wt1-N136A
215D mn1
215E fl1
215EA fl1-o4
215G fl1 v4
215H wt1 gl14
216A fl1 v4 Ch1
216D fl1 w3
216E fl1 v4 w3
216G fl1 v4 w3 Ch1
217A ts1
217B v4
217G v4 Ch1
217H ba2 v4
217I Les10-NA607
217J Les11-N1438
217K Les15-N2007
217L Les18-N2441
217M Les19-N2450
217N cpc1-N2284B
218A w3
218C w3 Ch1
218D Ht1-GE440
218DA Ht1-Ladyfinger
218DB Ht1
218E ba2
218G B1-Peru; A1 A2 C1 C2 r1-r
218GA B1-Peru; A1 A2 C1 C2 R1-r
218H w3-8686
218I w3-86GN12
218J w3-Kermicle-1
219A B1-Peru; A1 A2 C1 C2 r1-g
219B b1; A1 A2 C1 C2 r1-g
219C Ch1
219D Ht1 Ch1
219F B1-Peru; A1 A2 C1 C2 bz2 r1-g
219G B1-Bolivia-706B; A1 A2 C1 C2 r1-g

219H B1-Bolivia; A1 A2 C1 C2 Pl1-Rhoades Pr1 r1-g
219I B1-I; A1 A2 C1 C2 Pl1-Rhoades r1-r
219J B1-I; A1 A2 C1 C2 Pl1-Rhoades r1-g
219K B1-S; R1-g pl1-McClintock
219L B1-S; R1-r pl1-McClintock
220A Les1-N843
220B ws3 lg1 gl2; Alien Addition T2-Tripsacum
220D hcf15-N1253A
220F os1
221A gs2
221AA gs2-0229
221C wlv1-N1860 Ch1
221G wlv1-N1860
224B v*-5537
224H whp1; A1 A2 C1 R1 c2 gl1 in1
224I ws3-7752
224J sr5-7335
224K glnec*-8495
224L ws3-8949
224M ws3-8991
224N ws3-8945
226A ws3-N2357
226B b1-m1::Ds1; A1 A2 C1 C2 r1-g
226C b1-md2::Ds1; A1 A2 C1 C2 r1-g
226D b1-Pm5; A1 A2 C1 C2 r1-g
226E b1-Perum216; A1 A2 C1 C2 r1-g
227A dek3-N1289
227B dek4-N1024A
227C dek16-N1414
227D dek23-N1428
227E Les4-N1375
227I nec4-N516B
227K et2-2352
227L et2-91g6290-26
228A l18-N1940
228B spt1-N464
228C ws3-N453A
228CA ws3-N605A
228E B1-Bh
228F ms33-6019
228G ms33-6024
228H ms33-6029
228I ms33-6038
228J ms33-6041
229A rf3 Ch1
229B v24-N424
229BA v24-N576A
229BB v24-N588A
229BC v24-N350
229C w3 rf3 Ch1
229E emp2-MS1047
229F dek*-MS1365
229G dek*-MS4160
229H dek*-MS2159
229J dek*-PIE

CHROMOSOME 3 MARKERS

301A cr1
301B bif2-N2354
301C spc3-N553C
301D Wi2-N1540
301E rd4
301F ns1-R; ns2-R
302A d1-6016
302AA d1-N446
302AB d1-N339
302B d1 rt1
302E d1-tall
303A d1 rt1 Lg3-O
303F g2
303FA g2-pg14::I
303FB g2-v19
303FD g2-56-3040-14
303FE g2-59-2097
303FF g2-94-1478
303G g2 d1
304A d1 ys3
304F d1 Lg3-O ys3
304G Lg3-O Rg1
304I d1 h1
305A d1 Lg3-O
305D d1 Rg1
305K d1 cl1; Clm1-4
306A Rp3-A
306F ref1-MS1185
307A Sdw2-N1991
307C pm1
308B d1 ts4
308E ra2
308F ra2 Rg1
308G ra2-D
309A a1-m3::Ds Sh2
309B a1-m1-5718::dSpm
309C a1-m1-5719A1::dSpm
309D a1-m1-5719A1::dSpm; Mod Pr1
309E a1 Sh2; Spm-w
309F a1-m2-8417::dSpm
309G a1-m2(os)-o1
309H a1-m2-7991A-o2
309I a1-m2-7995::dSpm
309J a1-m2-7977B::dSpm
309K a1-m2-8012A-p1
309L a1 Sh2; Spm-s
309M a1-m1-5719A1::dSpm sh2
309N a1-m2-7995B
309O a1-m1-5996-4::dSpm
309P a1-m1-5719A1::dSpm; Spm-i
309Q a1-m5::Spm-w; Spm-s
309S a1-m2-8411A::Spm-w Sh2
309T a1-m2-7981B6::Spm-w
309U a1-m2-8409::Spm-i
309V a1-m5::Spm-w Sh2
309W a1-m2-8011::Spm-w Sh2
309X a1 Sh2; Spm-w-8745
309Y a1 Sh2; Spm-i
309Z a1-m1-5720-o2
310C ra2 lg2
310D Cg1
311A cl1
311AA cl1-N2
311B cl1; Clm1-2
311BA cl1-7716; Clm1-2
311C cl1; Clm1-3
311D cl1-p; Clm1-4
311E rt1
311F ys3
311G Lg3-O ys3
312A Les14-N2004
312B Les17-N2345
312D Lg3-O
312G brn1-R
312H g2 brn1-R
312I brn1-R cr1
312J brn1-R ra2 lg2
312K brn1-Nelson
312L brn1-3071
312M ms23
313A gl6
313AA gl6-gl7
313AB gl6-N672B
313D ms3
313DA ms3-6008
313DB ms3-6009
313DC ms3-6043
313DD ms3-6020
314A gl6 lg2 A1; A2 C1 C2 R1
314C gl6 lg2 a1-m et1; A2 C1 C2 Dt1 R1
314F Rg1 gl6 lg2
314G gl6 lg2
315B Rg1 gl6
315C Rg1
315D A1-b(P415); A2 C1 C2 R1
315I A1-m2(os)-p1
315J A1-m2(os)-r2
315K a1-m2-7991A-o1
315L a1-m2-7991A-p2
315M a1-m2-7991A-p3
315N a1-m2-7991A-p4
315O a1-m2-7991A-p4b
315P a1-m2-7991A-p5
315Q a1-m2-8010A-o2
315R A1-m3-r1a sh2-m1::Ds
315S a1-m5-o1
315T a1-m5-o2
315U A1-m5-r1
315V A1-m5-r4
315W A1-m5-r5
316A ts4
316B a1-N796
316C dek5-N1339A
316D a1-mt2
316E a1-mt3
316F a1-mt4
316G a1-mt5
316H a1-mt6
316I a1-mt7
316J a1-mt8
316K a1-mt11
316L a1-mt13
316M a1-mt15
316N a1-mt16
316O a1-mt18
316P a1-mt19
317F gl6 ts4 lg2
317I a1-m1-5996-4m::dSpm; Spm
317J a1-m2::Spm-s; Spm-w
317K a1-m2-7991A::Spm-s
317L a1-m2-8004::dSpm
317M a1-m2-8010A::Spm-s
317N a1-m2-8011::Spm-w
317O a1-m2-8012A
317P a1-m2-8147
317Q a1-m2-8167::dSpm
317R a1-m2-8414C
317S a1-m2-8549C
317T a1-m5::Spm-w Sh2
317U a1-m5::Spm-w sh2-1
317W a1-m1-5720::Spm
317X a1-m1-6078::dSpm
317Y a1-m2-8409-2
317Z A1 def-1260
318A ig1
318B ba1
318C y10-7748
318D hcf19-N1257A
318E sh2-N391B
318EA sh2-N2307
318F sh2-N2340
318G na1
318H vp1-Mc
318I y10-8624
319A lg2 A1-b(P415) et1; A2 C1 C2 Dt1 R1
319C lg2 a1-m et1; A2 C1 C2 R1 dt1
319D lg2 a1-m et1; A2 C1 C2 Dt1 R1
319F lg2 a1-st et1; A2 C1 C2 Dt1 R1
319G lg2 a1-st et1; dt1
320A lg2
320B lg2-PI184281
320C lg2 na1
320D lg2-podcorn
320E et1
320F A1 sh2; A2 C1 C2 R1 b1 pl1
320K sh2-94-1001-11
320L sh2-94-1001-58
320M sh2-94-1001-1003
320N a3-Styles; B1-b Pl1-Rhoades r1-g
320O a3-Styles; B1-b Pl1-Rhoades R1-nj
321A A1-d31; A2 C1 C2 R1
321B lg2 a1; A2 C1 C2 R1 dt1
321C lg2 A1-b(P415) et1; A2 C1 C2 R1 dt1
321D a1-m4::Ds; A2 C1 C2 R1
321E a1-rUq; A2 C1 C2 R1
321F a1-Mum1; A2 C1 C2 R1
321H a1-Mum3; A2 C1 C2 R1
321I a1-Mum4; A2 C1 C2 R1
321J a1-Mum5; A2 C1 C2 R1
321K a1-rUq; Uq1
321L a1-rUq(flow); Uq1
322A A1-d31 sh2; A2 C1 C2 R1 dt1
322B A1-d31 sh2; A2 C1 C2 Dt1 R1
322C A1-Mum3-Rev; A2 C1 C2 R1
322F a1-m; A2 C1 R1 b1 dt1 pl1
322I et1-24
322J et1-27
322K et1-34
322L et1-2162
322M et1-2320
322N et1-2424
322O et1-2457
322P et1-3191
322Q et1-3328
322R et1-5079
322S et1-84-6013
322T et1-88g-9733
322U et1-43
323A a1-m; A2 C1 C2 Dt1 R1
323D a1-m sh2; A2 C1 C2 Dt1 R1
323E a1-m et1; A2 C1 C2 Dt1 R1
323G a1-m1::rDt (Neuffer); A2 C1 C2 Dt1 R1
323H a1-st; A2 C1 C2 Mrh R1 dt1
323I a1-m1::rDt (Neuffer); A2 C1 C2 R1 dt1
324A a1-st; A2 C1 C2 Dt1 R1
324B a1-st sh2; A2 C1 C2 Dt1 R1
324E a1-st et1; A2 C1 C2 Dt1 R1
324G a1-st; A2 C1 C2 R1 dt1
324H a1 et1; A2 C1 C2 R1 dt1
324I a1-st et1; A2 C1 C2 R1 dt1
324J A2; C1 C2 R1 a1-sh2-del-Robertson
324K a1-Mus1; A2 C1 C2 R1
324L a1-Mus2; A2 C1 C2 R1
324M a1-Mus3
324N a1-Mus4
325A a1-p et1; A2 C1 C2 R1 dt1
325B a1-p et1; A2 C1 C2 Dt1 R1
325C a1-x1; A2 C1 C2 R1
325D a1-x3; A2 C1 C2 R1
325E A1 ga7; A2 C1 C2 R1
325G a3
325I a1-p; A2 C1 C2 Dt1 R1
325J a1-p; A2 C1 C2 Pr1 R1 dt1
325K a1-m3::Ds sh2-m1::Ds; A2 Ac C1 C2 R1

326A sh2-Elmore
326AA sh2-Garwood
326AB sh2-60-156
326B vp1
326BA vp1-Mum3
326BC vp1-86N6
326BD vp1-86GN14
326BE vp1-86GN18
326BF vp1-86GN19
326BG vp1-Mum2
326BH vp1-Mum1::Mu
326C Rp3
326D te1-1
326DA te1-Forester
326DB te1-Grogan
329A v*-9003
329B v*-8623
329C w21-022-15
329D yd2
329E w*-8336
329F yg*-W23
329G w*-062-3
329H v*-8609
329HA v*-8959
329I pg2
329K yel*-8630
329L yel*-5787
330A h1
330G a1-mrh; A2 C1 C2 Mrh R1
330H A1-b(P415) Ring 3; A2 C1 C2 R1
330I a1-Mum2; A2 C1 C2 MuDR R1
330J a1-Mum2; A2 C1 C2 R1
330K a1 sh2; A2 C1 C2 R1 dt1
330L a1-mrh; A2 C1 C2 R1
332B dek5-N874A
332C dek24-N1283
332D Wrk1-N1020
332F gl19-N169
332G dek6-N627D
332H dek17-N330D
332I Lxm1-N1600
332M Spc1-N1376
332N wlu1-N28
332S Mv1
333A dek5-25
333AA dek5-MS33
333B te1-Galinat
333C dek5-Briggs-1998-1
333D sh2

CHROMOSOME 4 MARKERS

401A Rp4-A
401AB Rp4-B
401C Ga1 su1
401D Ga1-S
401E Ga1-S; y1
401I ga1 su1
401J Ga1-M
401K Ga1-S su1
402A st1
402D Ts5
402E ms30-6028
402F hcf23-N1261A
403A Ts5 fl2
403B Ts5 su1
403C su1-F37
403D su1-PI228183
404A su3-5081; su4-5081
404B su3-89-1303-18; su4-89-1303-18
404C su3-94-4079-6; su4-94-4079-6
404D su3-85-3113-11; su4-85-3113-11
404E su3-87-2340-36; su4-87-2340-36
405B la1-PI239110
405BB la1-Funk:2232
405BC la1-N2020
405BD la1-N2276B
405BE la1-PI184284
405D la1-R su1 gl3
405G la1-R su1 gl4
406C fl2
406CA fl2-DR9234
406D fl2 su1
407D su1
407DA su1-N86
407DB su1-N2316
407DC su1-BKG489-13
407DD su1-Pl
407DE su1-R2412
407DF su1-N896A
407DG su1-N1161A
407DH su1-N2313
407DI su1-N2314
407DJ su1-N959
407DK su1-N1968
407DL su1-N1994
407E su1-am
407F su1-am; du1
408B bm3-Burnham su1
408C su1 zb6
408E bm3-91598-3
408J su1 ra3
408K su1; se1
408L su1 zb6 Tu1
409A su1-st
409B su1-66
409C su1-P
409D su1-5051
409F su1-28510
409G su1-28511
409H su1-28512
409I su1-28513
409J su1-28515
409K su1-28516
409L su1-28517
409M su1-28518
409N su1-28519
409O su1-28520
409P su1-30394
409Q su1-30397
409R su1-30398
409S su1-30399
409T su1-30400
409U su1-30401
409V su1-Bn2
410D su1 zb6 gl3
410E su1-A3
410F su1-4582::Mu1
410G su1-8064
410H su1-2401
410I su1-3837
410J su1-7110
410K su1-2857
410L su1-2859
410M su1-90-1101.1
410N su1-83-3383-4
410O su1-87-2046-27
410P su1-85-3217-10
410Q su1-84-5167-6
410R su1-84-5267-18
410S su1-85-3436-29
411A su1-8908
411B su1 gl4 o1
411F gl7 su1 v17
412C su1 gl3
412G su1 gl4 Tu1
413A su1 o1
413B su1 gl4
413D su1 C2-Idf1(Active-1); A1 A2 C1 R1
413F su1 de*-414E
413G v23 Su1 gl3; bm*-COOP
414A bt2
414AA bt2-Williams
414AB bt2-60-158
414AC bt2-9626
414AD bt2-5288
414B gl4
414BA gl4-Stadler
414BB gl4-gl16
414BD gl4-N525A
414C gl4 o1
414E de*-414E
415A j2
415B o1-N1243
415C o1-N1478A
415D bt2-8132
416A Tu1-A158
416B Tu1-l(1st)
416C Tu1-l(2nd)
416D Tu1-d
416E Tu1-md
416F Tu1 gl3
417B v8
417C gl3
417D o1 gl3
417E gl3-N531
418A gl3 dp1
418B c2; A1 A2 C1 R1
418D C2-Idf1(Active-1); A1 A2 C1 R1
418E dp1
418F o1
418G v17
419A v23-8914
419E gl7
419F Dt6 gl3 C2; A2 C1 R1 a1-m
419G Dt6 C2; A2 C1 R1 a1-m
419H c2-m1::Spm; A1 A2 C1 R1
419I c2-m2::dSpm c2-m3::Mpi1
419J c2-Mum1
419K c2-m2::dSpm; Spm-s
419L c2-m881058Y::IRMA; En Med wx1-m8::Spm-l8
419M c2-m3::Mpi1
420A su1 Dt4 C2; A2 C1 R1 a1-m
420C nec*-rd
420CA nec*-016-15
420D yel*-8957
420F dp*-4301-43
420G w*-9005
420H Dt4 C2; A2 C1 R1 a1-m
424C gl3-64-4
424D gl3-56-3120-2
424E gl3-56-3129-27
424F gl3-60-2555
424G gl3-PI183683
424H gl3-PI251928
424I gl3-PI251938
424J gl3-PI254858
424K gl3-PI267180
424L gl3-PI267219
424M gl3-PI311517
424N gl3-15
426A Gl5 Su1; gl20
426B gl3-PI251941
426D cp2-N1324A
427A cp2-o12
427AA cp2-N211C
427AB cp2-N1875A
427AC cp2-MS2608
427AD cp2-N912
427B dek25-N1167A
427C Ysk1-N844
427D orp1-N1186A; orp2-N1186B
427E dek8-N1156
427F dek10-N1176A
427G Ms41-N1995
427H dek31-N1130
427I Sos1-ref
428A gl5 Su1; gl20
428C nec5-N642
428D spt2-N1269A
428E wt2-N10
428F lw4; Lw3
428G bx1
428H gl5 su1; gl20
428L dsc1-MS2058

CHROMOSOME 5 MARKERS

501A am1 a2; A1 C1 C2 R1
501B lu1
501D ms13
501E gl17
501F gl17-N260B
501G gl17 a2; A1 C1 C2 R1
501I am1
502B A2 ps1-Sprague pr1; A1 C1 C2 R1
502C D9-N2319
502D A2 bm1 pr1; A1 C1 C2 R1
502E Ms42-N2082
502F Nl2-N1445
502G A2 Bt1 ga10
502H hcf21-N1259A
503A A2 bm1 pr1 ys1; A1 C1 C2 R1
503B hcf43-N1277B
503C a2-mu1::Mu1
503D a2-mu2
503E a2-mu3
503F A2 pac1-ref; A1 C1 C2 R1-r b1
503FA A2 pac1-ref; A1 B1-Peru C1 C2 Pl1-Rhoades r1-r
504A A2 bt1 pr1; A1 C1 C2 R1
504C A2 bm1 pr1 zb1; A1 C1 C2 R1
504E A2 bt1; A1 C1 C2 R1
505B A2 pr1 ys1; A1 C1 C2 R1
505C A2 bt1 pr1 ga*-Rhoades; A1 C1 C2 R1
505D pr1-N1515A
505E pr1-N1527A
506A A2 v3 pr1; A1 C1 C2 R1
506B A2 pr1; A1 C1 C2 R1
506C A2 pr1 v2; A1 C1 C2 R1
506D na2 A2 pr1; A1 C1 C2 R1
506F A2 pr1 v12; A1 C1 C2 R1
506L A2 br3 pr1; A1 C1 C2 R1
507A a2; A1 C1 C2 R1
507AA a2-Mus2; A1 C1 C2 R1
507AB a2-Mus3; A1 C1 C2 R1
507AC a2-Mus1; A1 C1 C2 R1
507F a2 bm1 bt1 ga*-Rhoades; A1 C1 C2 R1
507G a2 bm1 bt1; A1 C1 C2 R1
507H A2 bv1 pr1; A1 C1 C2 R1
507I a2-m4::Ds; wx1-m7::Ac7
508A a2 bm1 bt1 pr1; A1 C1 C2 R1
508C a2 bm1 bt1 bv1 pr1; A1 C1 C2 R1
508F a2 bm1 pr1 ys1; A1 C1 C2 R1
508H a2-Mum1

508I a2-Mum2
508J a2-Mum3
508K a2-Mum4
508L bv1 pr1
509G a2-m1::dSpm Bt1
509H a2-m1(II)::dSpm(class II)
509I pr1-m1
509J pr1-m2
509K a2-m1(ps)
509L a2-m1::dSpm; Spm-s
509M a2-m5::dSpm
509N A2-m1(os)-r1
510A a2 bm1 pr1 v2; A1 C1 C2 R1
510D a2 pr1 gl8; A1 C1 C2 R1
510E a2 ae1 pr1 gl8; A1 C1 C2 R1
510G a2 bm1 pr1 eg1; A1 C1 C2 R1
511C a2 bt1 pr1; A1 C1 C2 R1
511F a2 bt1 Pr1 ga*-Rhoades; A1 C1 C2 R1
511H a2 bt1; A1 C1 C2 R1
512C a2 bt1 pr1 ga*-Rhoades; A1 C1 C2 R1
512D vp2-N1136B
512E Wi4-N2445A
512F pb4
512G gl8-N166A
512H v13
512I lw2-vp12
513A a2 pr1; A1 C1 C2 R1
513C a2 pr1 v2; A1 C1 C2 R1
513D A2 pr1 sh4; A1 C1 C2 R1
513E a2 pr1 v12; A1 C1 C2 R1
514A a2 bm1 pr1; A1 C1 C2 R1
514B ae1-PS1
514C ae1-PS2
514D ae1-PS3
514E ae1-PS4
514F ae1-PS5
514G ae1-PS6
514H ae1-PS7
514I ae1-PS8
514J ae1-PS9
514K ae1-PS10
514L ae1-PS11
514M Ae1-5180-r4
514N bt1-m1::dSpm
514O bt1-m2
514P bt1-m3::dSpm
514Q bt1-m4::Ds
514R Bt1-m1-r1
515A vp2
515AA vp2-DR5180
515AB a2 vp2-green mosaic; A1 C1 C2 R1
515C ps1-Sprague
515CA ps1-8776
515CB ps1-881565-2M
515CC ps1-N80
515CD ps1-8205
515D bm1
515E bt1-N1992
515F bt1-N2308
515G bt1-N2309
516B bt1-R
516BA bt1-Elmore
516BB bt1-C103
516BC bt1-Singleton
516BD bt1-sh3
516BE bt1-sh5
516BF bt1-Eldridge
516BH bt1-6-783-7
516BI bt1-Vineyard
516BJ bt1-T
516BK bt1-W187R
516BL bt1-3040
516BM bt1-N797A
516C ms5
516D td1 ae1
516DA td1-Nickerson
516G A2 bm1 pr1 yg1; A1 C1 C2 R1
517A v3
517AB v3-8982
517B ae1
517BA ae1-EMS
517BB ae1-PS12
517BC ae1-PS13
517BD ae1-PS14
517BE ae1-PS15
517BF ae1-PS16
517BH ae1-Elmore
517E ae1 pr1 gl8
518A sh4
518AA sh4-Rhoades
518AB sh4-o9
518B gl8-Salamini
518BA gl8-R
518BB gl8-6:COOP
518BC gl8-6:Salamini
518BD gl8-10:COOP
518BE gl8-PI180167
518C na2
518D lw2
519A ys1
519AA ys1-W23
519AB ys1-5344
519AC ys1-N755A
519AD ys1-74-1924-1
519B eg1
519C v2
519D yg1
519E A2 pr1 yg1; A1 C1 C2 R1
519F A2 pr1 gl8; A1 C1 C2 R1
519H zb1
519I zb1-2
520A hcf38-N1273
520B v12
520C br3
520F A2 Dap1; A1 C1 C2 R1
520G A2 pr1 Dap1; A1 C1 C2 R1
520H Dap1-2
520I ae1-1979-7
520J ae1-MOEWS
520K ae1-1981-MuT
521A nec3-N409
521B Nec*-3-9c
521C nec*-8624
521D nec*-T5-9(5614)
521E nec*-7476
521F nec*-6853
521G nec*-7281
521H nec*-8376
521I v*-6373
521J yg*-8951
521K lw3; lw4
521L w*-021-7
521N lnec*-5931
521NA lnec*-8549
521P lw3; Lw4
524A v*-PI267226
524B les*-3F-3330
527A dek18-N931A
527B dek9-N1365
527C dek26-N1331
527D dek27-N1380A
527E grt1-N1308B
527F nec7-N756B
527G dek33-N1299
527H Msc2-N1124B
527I ppg1-N199
527J nec6-N493
528A Hsf1-N1595
528B wgs1-N206B
528C anl1-N1634
528CA anl1-330C
528E prg1-MS8186
528F ren1-MS807
528H dek*-MS2146
528I dek*-MS1182
529A anl1-N1643
529B anl1-N1645
529C anl1-N1671
529D anl1-N1685
529E anl1-N1691
529F anl1-N1673

CHROMOSOME 6 MARKERS

601C rgd1 y1
601D rgd1-N372B
601F po1-ms6 y1 pl1
601H rhm1 rgd1 y1
601I rhm1 y1 l11
601J Wsm1 Mdm1; Wsm2 Wsm3
601K wsm1 mdm1; wsm2 wsm3
601L Mdm1 y1
602A po1-ms6 wi1 y1
602C y1
602D rhm1 Y1
602H y1-N2236
602J y1-w-mut
602K y1-gbl
602L y1-pb1
602M y1-8549
602N y1-Caspar
602O y1-0317
602P y1-129E
603A y1 l10
603AA y1 l10-1359
603B y1 l11-4120
603C y1 l12-4920
603D w15-8896 y1
603H mn3-1184 y1
604A y1-87-2307-1
604D y1 l15-Brawn1
604F y1 si1-mssi
604FA y1 si1-ts8
604FB y1 si1-Sam
604G y1 si1-at
604H y1 ms1
604HA y1 ms1-Robertson
604I Y1 ms1
604IA ms1-6050
605A wi1 y1
605C y1 pg11; Wx1 pg12
605E wi1 Y1 Pl1
605F wi1 Y1 pl1
605G l3
605H pg11-M14; pg12-KYS
605I pg11-Oh43; pg12-KYS
606A Y1 pg11-4484; Wx1 pg12-4484
606AA pg11-8925; pg12-8925
606AB pg11-48-040-8; pg12-48-040-8
606AC pg11-8563; pg12-8563
606AD pg11-8322; pg12-8322
606B y1 pg11; pg12 wx1
606C Y1 pg11; pg12 wx1
606E y1 pl1
606F y1 Pl1
606I y1 pg11 su2; Wx1 pg12
607A y1 Pl1-Bh1; A1 A2 C2 R1 c1 sh1 wx1
607C y1 su2
607E y1 pl1 su2 v7
607H y1 Pl1-Bh1; A1 A2 C2 R1 Wx1 c1 sh1
607I y1 Pl1-Bh1; A1 A2 C2 R1 c1 sh1 skb1 wx1
607J sm1-Brawn168
607K sm1-Brawn178
607L sm1-Brawn184
608A gs3-N268
608C sbd1-N2292
608D Les13-N2003
608F y1 pl1 w1
608G Y1 l11
608H y1-m1301::dSpm
608I Rp8-A
608J Rp8-B
609D Y1 su2
609DA Y1 su2-89-1273
609DB su2-PS1
609DC su2-PS2
609DD su2-1979-5
609DE su2-87-2279-12
609DF su2-1981
609DG su2-1982
609DH su2-0203
609DI su2-PI193430
609DJ su2-1979-1
609F ms1-Albertsen
610B Dt2 Pl1; A2 C1 C2 R1 a1-m
610F Y1 pl1 su2 v7
610G hcf34-N1269C
610H Y1 Dt2 pl1; A2 C1 C2 R1 a1-m
610I hcf36-N1271B
610J hcf48-N1282C
610K hcf26-N1263C
610L hcf323
610M hcf5-N510C
611A Pl1 sm1; P1-rr
611D Pt1
611E Y1 pl1 w1
611EA w1-7366
611I sm1 tan1-py1; P1-rr
611K Y1 Pl1 w1
611L w1; l1
611M afd1
611N sr4-N65A
611O o14-N924
612A w14
612B po1
612BA po1-ms6
612C l*-4923
612D oro1
612DA oro1-6474
612I tan1-py1
612J w14-8657
612K w14-8050
612L w14-6853
612M w14-025-12
612N w14-1-7(4302-31)
612O yel*-1-7(4302-31)
613A 2NOR y1; A1 C1 C2 R1 a2 bm1 pr1 v2 wx1
613D vms*-8522
613F w14-8613
613I tus*-5267
613J gm*-6372
613L w*-8954
613M yel*-039-13
613N yel*-7285
613O l*-4-6(4447)
613P yel*-8631
613T pg11-6656; pg12-6656
627A dek28-N1307A

627B dek19-N1296A
627C vp*-5111
627G dek*-MS1104; l*-1104

CHROMOSOME 7 MARKERS

701B In1-D
701D o2
701E o2-Mum1
701F Hs1
702A o2 v5
702B o2 v5 ra1-Ref gl1
702I In1-Brawn
703A o2 v5 gl1
703B De*-B30
703C o2-m(r); Bg
703D o2 ra1-Ref gl1
703E o2-R; Bg
703F o2-m12::Spm
703G o2-m2::Ds; Ac
703H o2-m5::Ac
703J Rs1-O
703JA Rs1-1025::Mu6/7
703K Rs1-Z
704B o2 ra1-Ref gl1 sl1
704C o2-NA696
704D o2-NA697
704E gl1-m8
704F ms22-6036
704H o2-orange
704I gl1-PI267186
705A o2 gl1
705B o2 gl1 sl1
705D o2 bd1
706A o2 sl1
706B vp9-Bot100
707A y8 v5 gl1
707B in1; A1 A2 C1 C2 R1 pr1
707C in1 gl1; A1 A2 C1 C2 R1 pr1
707D v5
707E vp9-R
707EA vp9-3111
707EB vp9-86GN9
707EC vp9-86GN15
707F y8 gl1
707G in1 gl1; A1 A2 C1 C2 Pr1 R1
708A ra1-Ref
708AA ra1-PI262495
708AB ra1-PI184279
708AC ra1-PI239103
708AD ra1-PI267181
708AE ra1-PI267184
708AF ra1-63-3359
708B bd1-N2355
708C o15-N1117
708D y8-lty2
709A gl1
709AA gl1-56-3013-20
709AB gl1-56-3122-7
709AC gl1-PI183644
709AD gl1-PI218043
709AE gl1-PI251652
709AF gl1-PI257507
709AG gl1-Istra
709AH gl1-BMS
709AI gl1-7L
709AJ gl1-9:COOP
709AK gl1-N212
709AL gl1-N269
709AM gl1-N345B
709C gl1-m
710A gl1 Tp1
710B gl1 mn2
710E o5 gl1
710I gl1 Bn1
710J gl1-N271
710K gl1-dy
710L gl1-PI218038
711A Tp1
711B ij1-ref::Ds
711C ij1-60-2454-20
711G ts*-br
712A ms7
712AA ms7-6007
712B ms7 gl1
713A Bn1
713E Bn1 bd1
713H Bn1 ij1
713I bd1 Pn1
714A Pn1
714B o5
714BA o5-PS3038
714BB o5-N76B
714BC o5-N874B
714C o5-N1241
714D va1
715A Dt3; A2 C1 C2 R1 a1-m
715C gl1 Dt3; A2 C1 C2 R1 a1-m
716A v*-8647
716B yel*-7748
716C dlf1-N2389A
716D dlf1-N2461
716F Les9-N2008
727A dek11-N788
727B wlu2-N543A
727D v27-N590A
727DA v27-N53B
727DB v27-N413C
727E gl1-cgl
727F Rs4-N1606
727G Rs1-O o2 v5 ra1-Ref gl1
727H ms34-6004
727I ms34-6010
727J ms34-6013
727K ms34-6014
728A Px3-6
728B ptd2-MS3193
728C mn2-cp1
728D sh6-8601
728E sh6-N1295
728F ren2-NS326
728G dek*-MS2082
728H dek*-MS5153

CHROMOSOME 8 MARKERS

801A gl18-g
801B v16
801I yel*-024-5
801K v16 ms8
802A rgh1-N1285
802B emp3-N1386A
802C Ht2
802G ms43
802H gl18-PI262473
802I gl18-PI262490
803A ms8
803B nec1-025-4
803D gl18-g ms8
803F nec1-7748
803G nec1-6697
804A v21-A552
804B dp*-8925
804C tb*-poey1013
805A fl3
805C gl18-g v21-A552
805E el1
805G ms8 j1
808A ct1
808B Lg4-O
808C Htn1
808D epc1-W23
808G Epc1-W23
810A v16 j1; l1
810B j1
810C j1-JSM
827A dek20-N1392A
827B dek29-N1387A
827C Bif1-N1440
827CA Bif1-N2001
827D Sdw1-N1592
827E Clt1-N985
827F pro1-N1058
827G pro1-N1121A
827H pro1-N1528
827I pro1-N1533
827J wlu3-N203A
827K pro1
827L pro1-Tracy
828A ats1
828C pro1-N1154A
828D pro1-NA342
828E pro1-N1530

CHROMOSOME 9 MARKERS

901B yg2 C1 sh1 bz1; A1 A2 C2 R1
901C yg2 C1 sh1 bz1 wx1; A1 A2 C2 R1
901E yg2 C1 bz1 wx1; A1 A2 C2 R1
901H yg2 C1 Bz1; A1 A2 C2 R1
902A yg2 c1 sh1 bz1 wx1; A1 A2 C2 R1
902B yg2 c1 sh1 wx1; A1 A2 C2 R1
902C yg2 c1 sh1 wx1 gl15-Hayes; A1 A2 C2 R1
902D yg2 c1 sh1 Bz1 wx1 gl15 K9S-s; A1 A2 C2 R1
902E C1 sh1 Bz1-McC1; A1 A2 C2 R1
902F bz1-m13::dSpm
902G C1 sh1 bz1 wx1; A1 A2 C2 R1 Spm
902H bz1-m13::dSpm; Spm
902I bz1-m13CS1
902J bz1-m13CS3
902K bz1-m13CS4
902L bz1-m13CS5
902M bz1-m13CS6
902N bz1-m13CS7
903A C1 sh1 bz1; A1 A2 C2 R1
903B C1 sh1 bz1 wx1; A1 A2 C2 R1
903D C1-I sh1 bz1 wx1; A1 A2 C2 R1
903E bz1-m13CS8
903F bz1-m13CS10
903G bz1-m13CS11
903H bz1-m13CS12
904B C1 sh1; A1 A2 C2 R1
904C C1 sh1 wx1; A1 A2 C2 R1
904D C1 wx1 ar1; A1 A2 C2 R1
904F C1 sh1 bz1 gl15 bm4; A1 A2 C2 R1
904G rgo1-Sarkar
905A C1 sh1 wx1 K9S-l; A1 A2 C2 R1
905C C1 bz1 Wx1; A1 A2 C2 R1
905D C1 sh1 wx1 K9S-l; A1 A2 C2 K10-I R1
905E C1 sh1 wx1 v1; A1 A2 C2 R1
905G C1 bz1 wx1; A1 A2 C2 R1
905H c1 sh1 wx1; A1 A2 C2 R1-scm2 b1
905I ms45-6040
906A C1 wx1; A1 A2 C2 DsI Pr1 R1 y1
906B C1 wx1; A1 A2 C2 DsI R1 Y1 pr1
906C C1-I Wx1; A1 A2 C2 DsI R1
906D C1-I; A1 A2 C2 R1 y1
906G C1-I Sh1 Bz1 Wx1; DsI
906H C1 Sh1 bz1 wx1; Ac
907A C1 wx1; A1 A2 C2 R1
907E C1-I wx1; A1 A2 C2 R1 y1
907G c1-p; A1 A2 B1-b C2 R1 pl1
907H c1-n; A1 A2 C2 R1 b1 pl1
907I C1-S wx1; A1 A2 C2 R1
908A C1 wx1 da1 ar1; A1 A2 C2 R1
908B C1 wx1 v1; A1 A2 C2 R1
908D C1 wx1 gl15; A1 A2 C2 R1
908F C1 wx1 da1; A1 A2 C2 R1
908H Bf1-99-2070-8
909A C1 wx1 Bf1-ref; A1 A2 C2 R1
909B c1 bz1 wx1; A1 A2 C2 R1
909C c1 sh1 bz1 wx1; A1 A2 C2 R1
909D c1 sh1 wx1; A1 A2 C2 R1
909E c1 sh1 wx1 v1; A1 A2 C2 R1
909F c1 sh1 wx1 gl15; A1 A2 C2 R1
909G hcf42-N1276B
910B c1 sh1 wx1 gl15 Bf1-ref; A1 A2 C2 R1
910D c1; A1 A2 C2 R1
910G C1 sh1-bz1-x2 Wx1; A1 A2 C2 R1
910H C1 sh1-bz1-x3; A1 A2 C2 R1
910I sh1-bb1981 bz1-m4::Ds
910IA sh1-bb1981 bz1-m4::Ds; Ac
910L yg2-str
911A c1 wx1; A1 A2 C2 R1
911B c1 wx1 v1; A1 A2 C2 R1
911C c1 wx1 gl15-Hayes; A1 A2 C2 R1
911D Fas1
911E sem1-1364
911F def(Bf1..bm4)044-4
912A sh1
912AA sh1-1746
912AB sh1-9026-11
912AC sh1-3-6(6349)
912AD sh1-60-155
912AE sh1-EMS
912AF sh1-4020
912AG sh1-9552
912AH sh1-9626
912AI sh1-3017
912AJ sh1-6
912B sh1 wx1 v1
912E lo2
912H lo2 wx1
913C sh1 l7
913D sh1 l6
913E baf1
913F yg2-Mum1
913G yg2-Mum2
913H yg2-Mum3
913I yg2-Mum4
913J yg2-Mum5
913K yg2-Mum6
913L yg2-Mum7
913M yg2-Mum8
913N yg2-Mum9
913O yg2-DR83-106-3
913P yg2-DR83-106-5
914A wx1 d3-COOP
914B dek12-N1054
914K Wc1-ly; Y1
914L bz1-Mus1
914M bz1-Mus2

914N bz1-Mus3
914O bz1-Mus5
914P bz1-Mus6
914Q bz1-Mus7
914R bz1-Mus10
915A wx1
915B wx1-a
915C w11
915D wx1-N1050A
915E wx1-Alexander
915F wx1-N1240A
916A wx1 v1
916B wx1 v1-JRL
916C wx1 bk2
916E wx1 v1 gl15
916G Trn1-N1597
916H v31-N828
916I d3-8201
917A wx1 Bf1-ref
917C v1
917D ms2
917DA ms2-6002
917DB ms2-6012
917E gl15-Sprague
917EA gl15-Lambert
917EB gl15-KEW
917F d3-COOP
917FA d3-d2
917FB d3-015-12
917FC d3-072-7
917FD d3-8054
917FF d3-d2-Harberd
917FG d3-d2-Phillips
917FH d3-N660B
918A gl15 Bf1-ref
918B gl15 bm4
918C bk2 Wc1
918D Wc1
918F Wx1 Bf1-ref
918G Wc1 Bf1-ref bm4
918GA Wc1-Wh Bf1-ref bm4
918K bk2 v30
918L wx1 Wc1
919A bm4
919B Bf1-ref bm4
919C l6
919D l7
919G l6; l1
919I Bf1-DR-046-1
919J bz1-Mum9; MuDR
919K bz1-Mum4::Mu1
919L bz1-Mum1
919M bz1-Mum2
919N bz1-Mum3
919O bz1-Mum5
919P bz1-Mum6
919Q bz1-Mum7
919R bz1-Mum8
919S bz1-Mum9
919T bz1-Mum10
919U bz1-Mum11
919V bz1-Mum12
919W bz1-Mum15
919X bz1-Mum16
919Y bz1-Mum18
920A yel*-034-16
920B w*-4889
920C w*-8889
920E w*-8950
920F w*-9000
920G Tp3L-9SRhoades
920L ygzb*-5588
920M wnl*-034-5
920N pyd1
923A wx1-a
923B wx1-B
923C wx1-B1
923D wx1-B2::TouristA
923E wx1-B3::Ac
923F wx1-B4::Ds2
923G wx1-B6
923H wx1-B7
923I wx1-B8
923J wx1-BL2
923K wx1-BL3
923L wx1-C
923M wx1-C1
923N wx1-C2
923O wx1-C3
923P wx1-C4
923Q wx1-C31
923R wx1-C34
923S wx1-F
923T wx1-90
923U wx1-H
923V wx1-H21
923W wx1-I
923X wx1-J
923Y wx1-M
923Z wx1-m1::Ds
923ZA wx1-m6R
923ZB wx1-m6NR
923ZC wx1-m8::Spm-I8
923ZD wx1-P60
923ZE wx1-R
923ZF wx1-Stonor
924A Ring 9S Wd1 C1-I; wd1 C1
924B C1-I Ring 9S; A1 A2 C2 R1
924C yg2
924D wd1
924E wd1 C1 sh1 bz1
924F tiny fragment 9 Sh1 Bz1; C1 sh1 bz1 wx1
924G C1-I Bz1; Ac DsI
924H c1 sh1 bz1 wx1; Ac
925A bz1-m1::Ds wx1-m9::Ac
925B wx1-m9::Ac
925C bz1-m2::Ac
925D Wx1-m9r1
925E bz1-m2(DII)::Ds wx1-m6::Ds
925F C1 sh1 bz1 wx1-m8::Spm-I8
925H bz1-m2(DI)::Ds wx1; R1-sc:124
925I c1-m2::Ds Wx1; Ac
925J c1-m858::dSpm wx1
925K c1-m1::Ds
926A sh1-m5933::Ds
926B Sh1-r3(5933)
926C Sh1-r6(5933)
926D Sh1-r7(5933)
926E Sh1-r8(5933)
926F Sh1-r9(5933)
926G Sh1-r10(5933)
926H Sh1-r11(5933)
926I sh1-m6233::Ds
926J Sh1-r1(6233)
926K Sh1-r2(6233)
926L C1-I sh1-m6258::Ds
926M Sh1-m6258-r1
926N Sh1-r6795-1
926O bz1-m5::Ac
926P Bz1-wm::Ds1
926Q Bz1-m1-p
926R Bz1-m2-r1
926S Bz1-m2(DII)-r1
926T Bz1-m2(DII)-r2
926U Bz1-m2(DII)-r3
926V sh1-bb1981 Bz1-m4-p1
926W sh1-bb1981 Bz1-m4-r6851
926X sh1-bb1981 Bz1-m4-r7840B
926Y sh1-bb1981 Bz1-m4-r8332
926Z Bz1-m5-p1
926ZA Bz1-m5-r1
926ZB Bz1-m5-r2
927A dek12-N873
927B dek13-N744
927C dek30-N1391
927D Les8-N2005
927E Zb8-N1443
927H C1 Dt7; A2 C2 R1 a1-r
927I G6-N1585
927K Rld1-N1990
927L Rld1-N1441
928A yg2-N27
928AA yg2-N585
928AB yg2-N697
928AC yg2-N610
928B wlu4-N41A
928C ms20
928G c1-m5::Spm wx1-m8::Spm-I8; A1 A2 C2 R1
928H wx1-m7::Ac7
928I C1 bz1-mut::rMut; A1 A2 Bz2 C2 Mut R1
928J C1 bz1-(r)d; A1 A2 C2 R1
928K C1 Sh1 bz1-s; A1 A2 C2 Mut R1
928L ms45-6006
928M ms35-6011
928N ms35-6018
928O ms*-6021
928P ms*-6022
928Q ms35-6027
928R ms35-6031
928S ms*-6046
928T ms*-6047
929E Dp9
930A wx1-Mum1
930B wx1-Mum2
930C wx1-Mum3
930D wx1-Mum4
930E wx1-Mum5::Mu
930F wx1-Mum6
930G wx1-Mum7
930H wx1-Mum8
930I wx1-Mum9
930J wx1-Mum10
930K wx1-Mum11
930L wx1-Mus16
930M wx1-Mus181
930N wx1-Mus215
931A Wx1-m5::Ds
931B wx1-m6::Ds
931C wx1-m6-o1
931D Wx1-m7-i1
931E Wx1-m8-r10
931F Wx1-m9-r3
931G Wx1-m9-r4
931H wd1-Mus1
931I wd1-Mus2
931J wd1-Mus3
931K wd1-Mus4
931L wd1-Mus5
931M wd1-Mus6

CHROMOSOME 10 MARKERS

X01A oy1-Anderson
X01AA oy1-yg
X01AB oy1-8923
X01B oy1 R1; A1 A2 C1 C2
X01C oy1 bf2
X01E oy1 bf2 R1; A1 A2 C1 C2
X02C oy1 zn1 R1; A1 A2 C1 C2
X02E oy1 du1 r1; A1 A2 C1 C2
X02G oy1 zn1
X02H Oy1-N1459
X02I Oy1-N1538
X02J Oy1-N1583
X02K Oy1-N1588
X02L Oy1-N1989
X03A sr3
X03B Og1
X03D Og1 R1; A1 A2 C1 C2
X03E oy1 y9
X03F Inr1-Ref
X03G Ufo1
X04A Og1 du1 R1; A1 A2 C1 C2
X04B ms11
X04BA ms11-6051
X04D bf2
X04DA bf2-N185A
X04E du1-8501
X04F du1-8802
X05A Og*-0376
X05B Gs4-N1439
X05E bf2 sr2
X05G bf2 g1 R1-r; A1 A2 C1 C2
X05H r1 Sn1-coop; rea1
X05I r1 Sn1-bol1
X05J r1 Sn1-bol2
X05K r1 Sn1-bol3
X06A bf2 r1 sr2; A1 A2 C1 C2
X06C nl1 g1 R1; A1 A2 C1 C2
X06F bf2 R1 sr2; A1 A2 C1 C2
X07A nl1 g1 r1; A1 A2 C1 C2
X07C y9
X07CA y9-y12
X07D nl1
X08A vp10
X08B vp10-86GN5
X08C vp10-TX8552
X08F li1
X08FA li1-ILL90-243Teo
X09B li1 g1 R1; A1 A2 C1 C2
X09EA g1-g4
X09EB g1-56-3005-24
X09EC g1-1-7(X-55-16)
X09ED g1-68-609-13
X09EE g1-ws2
X09EF g1-PI262473
X09F ms10
X09FA ms10-6001
X09FB ms10-6035
X09G li1 g1 r1; A1 A2 C1 C2
X10A du1
X10AA du1-PS1
X10AB du1-PS2
X10AC du1-PS3
X10AD du1-PS6
X10AE du1-PS4
X10AF du1-PS5
X10AG du1-8801
X10AH du1-84-5350-31
X10D du1 g1 r1; A1 A2 C1 C2
X10F zn1
X10FA zn1-N25
X10G du1 v18
X11A zn1 g1
X11D Tp2 g1 r1; A1 A2 C1 C2
X11E g1 R1 sr2; A1 A2 C1 C2
X11F g1 r1; A1 A2 C1 C2
X11H zn1 R1-r; A1 A2 C1 C2
X11I Tp2 g1 sr2
X12A g1 r1 sr2; A1 A2 C1 C2
X12C g1 R1-g sr2; A1 A2 C1 C2
X12E g1 R1; A1 A2 C1 C2
X13D g1 r1-r sr2; A1 A2 C1 C2

X13E g1 r1-ch; A1 A2 C1 C2 wx1
X13G R1-p
X14A r1-r lsr1-Ej; A1 A2 C1 C2
X14E r1; A1 A2 C1 C2 wx1
X14F v18 r1; A1 A2 C1 C2
X14I r1-sc:m3::Ds
X14J R1-nj::Ac
X14K r1-Del902
X14L r1-g; A1 A2 C1 C2
X15B l1 r1 sr2; A1 A2 C1 C2
X15C R1-g; A1 A2 C1 C2
X15D r1-ch; A1 A2 C1 C2
X15F lsr1 R1-g Sr2
X15G isr1 r1-g sr2
X15H isr1 R1-r(Venezuela628-PI302369)
X15HA isr1 R1-r(Venezuela628-PI302369) sr2
X15I isr1 R1-nj Mst1
X16B r1 K10-I; A1 A2 C1 C2
X16C R1-ch; A1 A2 C1 C2 Pl1
X16CA R1-ch(Stadler)
X16D r1 sr2; A1 A2 C1 C2
X16E r1 K10-II; A1 A2 C1 C2
X16F R1 K10-II; A1 A2 C1 C2
X17B r1-r; A1 A2 C1 C2
X17C R1-mb; A1 A2 C1 C2
X17D R1-nj; A1 A2 C1 C2
X17E R1-r; A1 A2 C1 C2
X17F R1-scm3
X18A R1-lsk; A1 A2 C1 C2
X18B R1-sk:nc-2; A1 A2 C1 C2
X18C R1-st; A1 A2 C1 C2
X18D R1-sk; A1 A2 C1 C2
X18E R1-st Mst1
X18G R1-scm2; A1 A2 C1 C2 bz2
X18H R1-nj; A1 A2 C1 C2 bz2
X18I r1; A1 A2 C1 C2
X19A R1-sc:124
X19B w2
X19BA w2-Burnham
X19BB w2-2221
X19C l1 w2
X19D o7
X19E R1-r Lc1-Ecuador; b1
X19F r1 w2
X19G r1-n19 Lc1; b1
X19H r1-g:e Lc1; b1
X20B l1
X20C v18
X20I R1-d(Arapaho)
X20J R1-d(Catspaw)
X230A R1-r(Black Beauty Pop)
X230B R1-r(Burnham#2)
X230C R1-r(Cornell)
X230D R1-r(Ecuador1172)
X230E R1-r(Ethiopia-PI193658)
X230F R1-r(India6-PI166163)
X230G R1-r(India6-PI210551)
X230H R1-r(Kansas-PI222629)
X230I R1-r(MacDonald College)
X230J R1-r(Missouri-PI221889)
X230K R1-r(Oklahoma-PI213748)
X230L R1-r(Oklahoma-PI213757)
X230M R1-r(Turkey13-PI174414)
X230N R1-r(Turkey18-PI179131)
X230O R1-r(Turkey22-PI183773)
X230P R1-g(Argentina-PI162573)
X230Q R1-g(Arizona-PI213729)
X230R R1-g(Arizona-PI213738)
X230S R1-g(Arizona-PI218162)
X230T R1-g(Arizona-PI218164)
X231A R1-g(Arizona-PI218175)
X231B R1-g(Arizona-PI218178)
X231C R1-g(Black Mexico)
X231D R1-g(Bolivia1160)
X231E R1-g(Burnham#1)
X231F R1-g(Canada-PI214199)
X231G R1-g(North Dakota-PI213799)
X231H R1-g(North Dakota-PI213807)
X231I R1-g(South Dakota-PI213779)
X231J R1-g(South Dakota-PI213787)
X231K R1-g(Ethiopia32-PI197503)
X231L R1-g(Guatemala5A-Mangelsdorf2837)
X231M R1-g(India6-PI166161)
X231N R1-g(Iowa-PI217411)
X231O R1-d(Acoma)
X231P R1-d(Pony)
X231Q R1-d(Tomi)
X231R R1-d(Valley)
X231S R1-d(Winnebago)
X231T R1-g(Guerrero10)
X232A R1-r(Chiapas70)
X232B R1-g(Ecuador887#6723)
X232C R1-r(Ecuador929-Pl302341)
X232D R1-g(Venezuela903-Pl302393)
X232E R1-g(Bolivia494)
X232F R1-g(Bolivia705)
X232G R1-g(Argentina216/62-A)
X232H R1-g(BrazilCMI56)
X232I R1-g(BrazilCMI54)
X232J R1-g(Brazil4980)
X232K R1-g(Brazil1963)
X232L R1-g(Brazil3359)
X232M R1-g(Brazil5042)
X232N R1-g(Brazil5011)
X232O R1-g(ParaguayCMI128)
X232P R1-r(Argentina167/62)
X232Q R1-g(Bolivia707#6769)
X232R R1-g(Bolivia716#6759)
X232S R1-g(Bolivia724)
X232T R1-g(Bolivia1004)
X233A R1-g(Bolivia1520)
X233B R1-g(Brawn)
X233C R1-g(Peru San Miguel)
X233D R1-r(Venezuela694#16037)
X233E R1-ch(New Mexico-PI218151) K10-I; Pl1
X233F R1-ch(New Mexico-PI218159) K10-I; Pl1
X233G R1-ch(Pueblo); pl1
X233H R1-r(Venezuela760#16029)
X233I R1-g(New Mexico-PI218150)
X233J R1-g(New Mexico-PI218168)
X233K R1-g(Oklahoma-PI213756)
X233L R1-g(1302-Mangelsdorf2995)
X233M R1-g(Peru1304-Mangelsdorf2993)
X233N R1-g(Peru1595A-Mangelsdorf3013)
X233O R1-g(Turkey8-PI167989)
X233P R1-g(Washington-PI217489)
X233Q R1-nj(North Dakota-CuduD12-PI222285)
X233R R1-nj(New Mexico-PI218170)
X233S R1-st(2-COOP)
X233T R1-g(Bolivia473)
X234A R1-g(Bolivia716)
X234B R1-g(Chile370)
X234C R1-g(Chile406)
X234D R1-g(Ecuador592)
X234E R1-g(Peru568)
X234F R1-g(Peru1182)
X234G R1-g(Peru Corongo-ANC120)
X234H R1-g(Peru Corongo-ANC120#907)
X234I R1-g(Peru Corongo-ANC150)
X234J R1-g(Peru Huarmey)
X234K R1-g(Guerrero23)
X234L R1-r(Ecuador318-PI302308)
X234M R1-r(Ecuador731-PI302327)
X234N R1-r(Venezuela412-PI302347)
X234O R1-r(Venezuela455-PI302348)
X234P R1-r(Venezuela497-PI302351)
X234Q R1-r(Venezuela530-PI302354)
X234R R1-r(Venezuela559-PI302355)
X234S R1-r(Venezuela457-PI302356)
X234T R1-r(Venezuela590-PI302362)
X235A R1-r(Venezuela594-PI302363); Arv1
X235B R1-r(Venezuela594-PI302363); arv1-m594
X235C R1-r(Venezuela702-PI302370)
X235D R1-g(Venezuela753-PI302381)
X235E R1-r(Venezuela760-PI302383)
X235F R1-r(Colombia1424)
X235G R1-r(Colombia1816)
X235H R1-r(Colombia1817)
X235I R1-r(Colombia1818)
X235J R1-g(Mexico27)
X235K R1-g(Mexico33)
X235L R1-g(Mexico40)
X235M R1-g(Aguas Calientes27)
X235N R1-r(Aguas Calientes39)
X235O R1-g(Aguas Calientes39)
X235P R1-g(Guanajuato97)
X235Q R1-g(San Juan del Rio)
X235R R1-r(Maiz Morado)
X235S R1-g(Bolivia661#7534)
X235T R1-g(Venezuela628#16038)
X236A R1-g(Argentina60/62)
X236B R1-g(Argentina216/62-B)
X236C R1-r(Venezuela628-PI302369)
X236D R1-r(Venezuela1543)
X236E R1-g(New Mexico-PI218143)
X236F R1-g(New Mexico-PI218148)
X236G R1-g(New Mexico-PI218153)
X236H R1-g(New Mexico-PI218157)
X236I R1-g(New Mexico-PI218169)
X236J R1-g(New Mexico-PI218170)
X236K R1-g(New Mexico-PI218173)
X236L R1-g(Washington-PI217488)
X236M R1-nj(F C Anderson)
X236N R1-nj(Illinois-Emmerling Trisomic)
X236O R1-st(3-COOP)
X236P R1-g(Peru1083)
X236Q R1-g(Guerrero24)
X236R R1-g(Guanajuato31)
X236S R1-g(Colima10)
X236T R1-r(Venezuela459#16039)
X24A cm1
X24B lep*-8691
X24C v*-8574
X25A R1-scm2; A2 C1 C2 a1-st
X25B R1-scm2; A1 A2 C1 c2
X25C R1-sc:122; A1 A2 C1 C2 pr1
X25D R1-scm2; A1 C1 C2 a2
X25E R1-scm2; A1 A2 C2 c1
X26A r1-X1 / R1; A1 A2 C1 C2
X26B R1-scm2; A1 A2 C1 C2
X26C R1-sc:122; A1 A2 C1 C2
X26D R1-sc:5691; A1 A2 C1 C2
X26E R1-scm2; A1 A2 C1 C2 pr1 wx1
X26F R1-scm2; A1 A2 C1 C2 In1-D
X26G R1-scm2; A1 A2 C1 c2-m2::dSpm
X26H R1-scm2; A1 A2 C1 C2 wx1
X27A dek14-N1435
X27B dek15-N1427A
X27C w2-N1330
X27D Les6-N1451
X27E gl21-N478B; gl22-N478C
X27F Vsr1-N1446
X27G Oy1-N700
X27H orp2-N1186B; orp1-N1186A
X27I l19-N425
X27J l13-N59A
X27K v29-N418
X27L Les12-N1453
X28B R1-scm2; a1-m1::rDt (Neuffer)
X28C R1-nj(Cudu); A1 A2 C1 C2
X28D Vsr*-N716
X28E Les3
X28F cr4-6143
X28G R1-nj(Chase); A1 A2 C1 C2
X28I R1-scm2; A2 C1 C2 a1-m1-5719::dSpm
X28J R1-scm2; A1 A2 C1 C2 bz1
X29A ren3-MS1339
X29B dek*-MS2181
X29C cr4-N590C
X29D cr4-N647
X29E cr4-N411
X35A Rp1-A
X35B Rp1-B
X35C Rp1-C
X35D Rp1-D
X35E Rp1-J
X35F Rp1-K
X35G Rp1-M
X35H Rp1-Kr3
X35I Rp1-Kr4
X35J Rp1-Kr1J92
X35K Rp1-Kr1J6
X35L Rp1-G
X35M Rp5
X35N Rp1-D Rp1-J
X35O Rp1-C Rp1-J
X35P Rp1-F Rp1-J
X35Q Rp1-C Rp1-F Rp1-J
X35R Rp1-G Rp1-I
X35S Rp1-F Rp1-G Rp1-J
X35T Rp1-D Rp1-G Rp1-J
X35U Rp5 Rp1-D
X35V Rp5 Rp1-G
X35W Rp5 Rp1-C Rp1-G Rp1-J

UNPLACED GENES

U140A aph1
U140AA Aph1
U140C l4
U140G ms22
U140H ms24
U140I zn2-94-234
U240A Les7-N1461
U240D o11
U240E zn2
U240F zn2-PI251887
U240G zn2-PI236997
U240H zn2-PI239110
U240I zn2-56-3012-10
U340D ws1-COOP ws2-COOP
U340DA ws1-Pawnee ws2-Pawnee
U340H oro4
U340I Mei1-mei025
U440B gl13
U440C hcf49-N1480
U440D ub1-76C
U440E frz1
U440F mg1-Sprague
U540A dv1

U540B dy1
U640A dy1-Doyle
U640B dy1-Russian
U640C pam1
U640D pam2
U640E ada1
U640F atn1 Adh1-1S5657
U740A abs1-PI254851
U740C lty1
U740F pi1 pi2
U740G Fbr1-N1602
U740H ad2-N2356A
U740I ba3
U740K Rp7
U840A csp1-NA1173
U840B blc1-Tracy
U840D Les21-N1442
U840F agt1
U840G Wi3-N1614
U840H nld1-N2346
U840I Mc1
U840J hcf16
U940A Ht3
U940B dsy2
U940C v25-N17
U940D hcf11-N1250A
U940E hcf17
U940F hcf73
U940G Glb2-0

MULTIPLE GENES

M141A A1 A2 B1 C1 C2 Pl1 Pr1 R1-g
M141AA A1 A2 B1 C1 C2 Pl1-Rhoades Pr1 R1-g
M141B A1 A2 B1 C1 C2 pl1 Pr1 R1-g
M142A A1 A2 b1 C1 C2 pl1 R1-r
M142B a1 A2 b1 C1 C2 pl1 R1-r
M142C A1 a2 b1 C1 C2 pl1 R1-r
M142D A1 A2 b1 bz1 C1 C2 pl1 R1-r
M142E A1 A2 b1 bz2 C1 C2 pl1 R1-r
M142F A1 A2 b1 c1-p C2 pl1 R1-r
M142G A1 A2 b1 C1-I C2 pl1 R1-r Wx1
M142H A1 A2 b1 C1 c2 pl1 R1-r
M142I A1 A2 b1 C1 C2-Idfm pl1 R1-r
M142J A1 A2 b1 C1 C2-Idf1(Active-1) pl1 R1-r
M142K A1 A2 b1 C1 C2 pl1 pr1 R1-r
M142L A1 A2 b1 C1 C2 gl1 in1 pl1 R1-r
M142M A1 A2 b1 C1 C2 In1-D pl1 R1-r
M142N A1 a2 bt1 C1 C2 pr1 R1
M142O C1 sh1 bz1 wx1; A1 A2 C2 R1-r
M142P c1 sh1 wx1; A1 A2 C2 R1-r
M142Q yg2 c1 sh1 wx1; A1 A2 C2 R1-g
M142R A1 A2 b1 C1-I C2 pl1 R1-r wx1
M142S su1 c2; A1 A2 C1 R1-r
M142T A1 A2 b1 C1 C2 pl1 r1-g
M142U A1 A2 b1 C1 C2 pl1 r1-r
M142V A1 A2 C1 C2 R1-nj
M142W A1 A2 C1 C2 R1-st
M142X A1 A2 b1 C1 C2 Pl1 r1-g
M142Y A1 A2 B1 C1 C2 Pl1 r1-g
M142Z a1-st A2 b1 C1 C2 pl1 R1-scm2
M142ZA A1 a2 b1 C1 C2 pl1 R1-scm2
M142ZB b1 bz1 C1 pl1 R1-scm2 sh1
M142ZC A1 A2 b1 bz2 C1 C2 pl1 R1-scm2
M142ZD A1 A2 b1 c1-n C2 pl1 R1-scm2
M142ZE A1 A2 b1 c1-p C2 pl1 R1-scm2
M241A A1 A2 B1 C1 C2 Pl1 Pr1 r1-g
M241C A1 A2 B1 C1 C2 Pl1 Pr1 R1-r
M241D A1 A2 b1 C1 C2 Pl1-Rhoades r1-g
M242A A1 A2 b1 C1 c2 pl1 R1-scm2
M242B A1 A2 b1 C1 C2 pl1 pr1 R1-scm2
M242C in1 gl1; A1 A2 b1 C1 C2 pl1 R1-scm2
M242D a1 sh2; A2 b1 C1 C2 pl1 R1-scm2
M242E c1 sh1 wx1; A1 A2 b1 C2 pl1 R1-scm2
M242F su1 c2; A1 A2 b1 C1 pl1 R1-scm2
M242G A1 A2 b1 C1 C2 pl1 R1-scm2
M242H A1 A2 b1 C1 C2 pl1 r1-g
M242I A1 A2 b1 C1 C2 pl1 r1-r
M242J c1 sh1 R1-sc
M340A A1 A2 B1 c1 C2 pl1 Pr1 R1-g
M340B A1 A2 B1 c1 C2 Pl1 Pr1 R1-g
M340C A1 A2 b1 c1 C2 pl1 Pr1 R1-g
M341B A1 A2 B1 C1 C2 pl1 Pr1 R1-r
M341C A1 A2 b1 C1 C2 Pl1 Pr1 R1-r
M341CA A1 A2 b1 C1 C2 Pl1-Rhoades Pr1 R1-r
M341D A1 A2 B1 c1 C2 Pl1 Pr1 R1-r
M341F A1 A2 b1 C1 C2 pl1 Pr1 R1-r
M441B A1 A2 B1 C1 C2 pl1 Pr1 R1-r wx1
M441D A1 A2 B1 C1 C2 Pl1 Pr1 r1-r
M441F A1 A2 b1 C1 C2 pl1 Pr1 R1-g wx1
M541B A1 A2 b1 C1 C2 pl1 Pr1 R1-g
M541F a1 A2 C1 C2 R1-nj
M541G A1 a2 C1 C2 R1-nj
M541H A1 A2 c1 C2 R1-nj
M541I A1 A2 C1-I C2 R1-nj
M541J A1 A2 C1 c2 R1-nj
M541K A1 A2 C1 C2-Idf1(Active-1) R1-nj
M541L A1 A2 bz1 C1 C2 Pr1 R1-nj
M541M A1 A2 Bz1 C1 C2 pr1 R1-nj
M541N A1 A2 C1 C2 gl1 in1 R1-nj
M541O A1 A2 C1 C2 In1-D R1-nj
M541P ae1 wx1
M641C A1 A2 b1 C1 C2 pl1 Pr1 R1-r wx1
M641D A1 A2 C1 C2 Pr1 r1 wx1 y1
M641E A1 A2 C1 C2 r1-g wx1 y1
M641F r1-g y1; A1 A2 C1 C2
M641G sm1-Brawn184; sm2-Brawn184
M741A A1 A2 b1 C1 C2 pl1 Pr1 r1-g wx1
M741B Stock 6; A1 A2 B1 C1 C2 Pl1 R1-r
M741C Stock 6; A1 A2 B1 C1 C2 pl1 R1-r
M741F Stock 6; A1 A2 C1 C2 pl1 R1-g y1
M741G Stock 6; A1 A2 C1-I C2 pl1 R1-g wx1 y1
M741H Stock 6; A1 A2 B1 C1 C2 Pl1 R1-nj
M741I Stock 6; A1 A2 C1 C2 R1
M841A A1 A2 C1 C2 pr1 R1 su1
M841B f1 wx1
M841C v4 wx1
M841D v2 wx1
M841F A1 A2 bz2 C1 C2 R1-scm2 wx1
M841G A1 A2 C1 c2 R1-scm2 wx1
M841H gl6 wx1
M841I su1 wx1
M841J v16 wx1
M841K gl4 wx1
M841L gl2 lg1 wx1
M941A A1 A2 c1 C2 Pr1 R1 wx1 y1
M941B Mangelsdorf's tester; a1 bm2 g1 gl1 j1 lg1 pr1 su1 wx1 y1
M941BA Mangelsdorf's tester + R1-nj
M941C a1 Dt1 gl2 lg1 wt1
M941D gl1 wx1 y1
M941E gl8-R wx1 y1
M941F sm1; wx1
MX40A A1 A2 C1 C2 P1-vv::Ac r1-sc:m3::Ds
MX40B A1 A2 Ac2 bz2-m::Ds C1 C2 R1
MX40C A1 A2 C1 C2 r1-sc:m3::Ds Ac8168-9
MX40D P1-vv::Ac r1
MX41A A1 A2 C1 C2 gl1 pr1 R1 wx1 y1
MX41B A1 A2 C1 C2 gl1 pr1 R1 su1 wx1 y1
MX41C a1 a2 bz1 bz2 c1 c2 pr1 r1 wx1 y1
MX41D a1 A2 C1 C2 gl1 pr1 R1 su1 wx1 y1
MX41E a1-m1-n A2 C1 C2 R1 wx1-m8::Spm-I8

B-CHROMOSOME

B542A Black Mexican Sweet; B chromosomes present
B542B Black Mexican Sweet; B chromosomes absent
B542C L289; B chromosomes present
B542D L289; B chromosomes absent

Trisomic
123A trisomic 1.
223A trisomic 2
328A trisomic 3
422A trisomic 4
523A trisomic 5
615A trisomic 6
718A trisomic 7
807A trisomic 8
922A trisomic 9
X23A trisomic 10

TETRAPLOID

N102A Autotetraploid; A1 A2 B1 C1 C2 Pl1 Pr1 R1
N102D Autotetraploid; A1 A2 C1 C2 R1
N102E Autotetraploid; B chromosomes present
N102EA Autotetraploid; B chromosomes present
N102F Autotetraploid; A1 a2 C1 C2 R1
N103A Autotetraploid; P1-rr
N103B Autotetraploid; P1-vv::Ac
N103C Autotetraploid; P1-ww
N103D Autotetraploid; P1-wr
N103E Autotetraploid; P1-mm
N104A Autotetraploid; su1
N104B Autotetraploid; A1 A2 C1 C2 pr1 R1
N105B Autotetraploid; wx1 y1
N105D Autotetraploid; A1 a2 bt1 C1 C2 R1
N105E Autotetraploid; bt1
N106C Autotetraploid; wx1
N107B Autotetraploid; W23
N107C Autotetraploid; Synthetic B
N107D Autotetraploid; N6

CYTOPLASMIC-STERILE/RESTORER

C736A R213 (N); mito-N Rf1 rf2
C736AB R213 (T) Sterile; cms-T Rf1 rf2
C736B Ky21 (N); mito-N Rf1 Rf2 Rf3 RfC
C736C B37 (N); mito-N rf1 Rf2 rf3 rfC
C736CA B37 (T) Sterile; cms-T rf1 Rf2
C736CB B37 (T) Restored; cms-T Rf1 Rf2
C736E Tr (N); mito-N Rf3 rfC rfT
C736F W23 (N); mito-N rf1 Rf2 rf3 RfC
C736FA W23 (N); mito-N rf1 Rf2 rf3 RfC
C736G B73 (N); mito-N rf1 Rf2 rf3 rfC
C736H L317 (N); mito-N rf3 RfC rfT
C836A Wf9 (T) Sterile; cms-T rf1 rf2
C836B Wf9 (N); mito-N rf1 rf2 rf3 rfC
C836C Wf9 (T) Restored; cms-T Rf1 Rf2 rf3 rfC
C836D Wf9 (S) Sterile; cms-S rf1 rf2 rf3 rfC
C836E Mo17 (T) Sterile; cms-T rf1 Rf2 rf3 rfC
C836F Mo17 (N); mito-N rf1 Rf2 rf3 rfC
C836G Mo17 (C) Sterile; cms-C rf1 Rf2 rf3 rfC
C836H Mo17 (S) Sterile; cms-S rf1 Rf2 rf3 rfC
C936D K55 (N); mito-N Rf1 Rf2 rf3 RfC
C936DA K55 (N); mito-N Rf1 Rf2 rf3 RfC
C936F N6 (N); mito-N rf1 Rf2 rf3 RfC
C936FA N6 (N); mito-N rf1 Rf2 rf3 RfC
C936G N6 (T) Sterile; cms-T rf1 Rf2
C936H N6 (T) Restored; cms-T Rf1 Rf2
C936I SK2 (N); mito-N rf1 Rf2 rf3 rfC
C936J SK2 (T) Sterile; cms-T rf1 Rf2
C936K SK2 (T) Restored; cms-T Rf1 Rf2
C936M 38-11 (N); mito-N rf1 Rf2 rf3 rfC
CX36A N6 (C) Restored; cms-C rf1 Rf2 rf3 RfC
CX36B N6 (S) Sterile; cms-S rf1 Rf2 rf3 RfC
CX36C B37 (C) Sterile; cms-C rf1 Rf2 rf3 rfC
CX36D B37 (S) Sterile; cms-S rf1 Rf2 rf3 rfC

CYTOPLASMIC TRAIT

C337A NCS2
C337B NCS3

TOOLKIT

T0318AA TB-3Ld Ig1; ig1 R1-nj
T0318AB cms-L; ig1 R1-nj
T0318AC cms-MY; ig1 R1-nj
T0318AD cms-ME; ig1 R1-nj
T0318AE cms-S; ig1 R1-nj
T0318AF cms-SD; ig1 R1-nj
T0318AG cms-VG; ig1 R1-nj
T0318AH cms-CA; ig1 R1-nj
T0318AI cms-C; ig1 R1-nj
T0318AJ cms-Q; ig1 R1-nj
T0940A Hi-II Parent A (for producing embryogenic callus cultures)
T0940B Hi-II Parent B (for producing embryogenic callus cultures)
T0940C Hi-II A x B (for producing embryogenic callus cultures)
T0940D KYS (for chromosome observations in pachytene microsporocytes)
T0940E Mu off; a1-Mum2 A2 C1 C2 R1
T3301A bti00191::Ac
T3301B bti00194::Ac
T3301C bti00207::Ac
T3301D bti00220::Ac
T3301E bti00225::Ac
T3301F bti00226::Ac
T3301G bti00228::Ac
T3301H bti00245::Ac
T3301I bti00252A::Ac
T3301J bti00257::Ac
T3301K bti9576::Ac
T3301L bti99221::Ac
T3301M mon003073::Ac
T3301N mon003077::Ac
T3301O mon002901::Ac
T3301P mon003068::Ac
T3301Q mon00004::Ac
T3301R mon00044::Ac
T3301S bti00209::Ac
T3301T bti00230::Ac
T3301W mon00068::Ac
T3301X mon00098::Ac
T3301Y mon00110::Ac
T3301Z mon00126::Ac
T3301ZA mon00212::Ac
T3301ZB mon03078::Ac
T3301ZC mon03082::Ac
T3301ZE mon00038::Ac
T3301ZF mon00060::Ac
T3301ZG mon00092::Ac
T3301ZH mon00152::Ac
T3301ZI mon00164::Ac
T3301ZJ mon00178::Ac
T3301ZK mon00186::Ac
T3301ZL mon00192::Ac
T3301ZM mon00236::Ac
T3301ZN mon00238::Ac
T3302A Inv1m; P1-vv::Ac bz2-m::Ds
T3302C T1-2b; P1-vv::Ac bz2-m::Ds
T3302D T1-2(036-7); P1-vv::Ac bz2-m::Ds
T3302E T1-2c; P1-vv::Ac bz2-m::Ds
T3302F T1-3(5883); P1-vv::Ac bz2-m::Ds
T3302G T1-3k; P1-vv::Ac bz2-m::Ds
T3302H T1-3(5597); P1-vv::Ac bz2-m::Ds
T3302I T1-3(5982); P1-vv::Ac bz2-m::Ds
T3302J T1-4i; P1-vv::Ac bz2-m::Ds
T3302K T1-4(064-20); P1-vv::Ac bz2-m::Ds
T3302L T1-4(4308); P1-vv::Ac bz2-m::Ds
T3302M T1-4(8602); P1-vv::Ac bz2-m::Ds
T3302N T1-4b; P1-vv::Ac bz2-m::Ds
T3302O T1-5(5525); P1-vv::Ac bz2-m::Ds
T3303A T1-5(6899); P1-vv::Ac bz2-m::Ds
T3303B T1-5b; P1-vv::Ac bz2-m::Ds
T3303C T1-5(4613); P1-vv::Ac bz2-m::Ds
T3303D T1-5(5045); P1-vv::Ac bz2-m::Ds
T3303E T1-5(043-15); P1-vv::Ac bz2-m::Ds
T3303F T1-5(5512); P1-vv::Ac bz2-m::Ds
T3303G P1-vv::Ac; T1-6(5495) (6S.80; 1.S.25) bz2-m::Ds
T3303H P1-vv::Ac; T1-6(e**) bz2-m::Ds
T3303I T1-6(028-13); P1-vv::Ac bz2-m::Ds
T3303J T1-6(7352); P1-vv::Ac bz2-m::Ds
T3303K T1-6(7097); P1-vv::Ac bz2-m::Ds
T3303L T1-7(4405); P1-vv::Ac bz2-m::Ds
T3303M T1-7i; P1-vv::Ac bz2-m::Ds
T3303N T1-7(4837); P1-vv::Ac bz2-m::Ds
T3303O T1-7(010-12); P1-vv::Ac bz2-m::Ds
T3304A T1-8(6591); P1-vv::Ac bz2-m::Ds
T3304B T1-8(4685); P1-vv::Ac bz2-m::Ds
T3304C T1-8(4307-4); P1-vv::Ac bz2-m::Ds
T3304D T1-9(7535); P1-vv::Ac bz2-m::Ds
T3304E T1-9(8302); P1-vv::Ac bz2-m::Ds
T3304F T1-9(6762); P1-vv::Ac bz2-m::Ds
T3304G T1-10g; P1-vv::Ac bz2-m::Ds
T3304H T1-10f; P1-vv::Ac bz2-m::Ds
T3304I bz2-m::Ds
T3304J Inv1m; P1-vv::Ac r1-sc:m3::Ds
T3304K Inv1a; P1-vv::Ac r1-sc:m3::Ds
T3304M T1-2c; P1-vv::Ac r1-sc:m3::Ds
T3305A T1-3(5597); P1-vv::Ac r1-sc:m3::Ds
T3305B T1-4i; P1-vv::Ac r1-sc:m3::Ds
T3305C T1-4(064-20); P1-vv::Ac r1-sc:m3::Ds
T3305F T1-4b; P1-vv::Ac r1-sc:m3::Ds
T3305H T1-5(6899); P1-vv::Ac r1-sc:m3::Ds
T3305J T1-5(4613); P1-vv::Ac r1-sc:m3::Ds
T3305M T1-6(5495); P1-vv::Ac r1-sc:m3::Ds
T3305N T1-6e; P1-vv::Ac r1-sc:m3::Ds
T3305O T1-6(028-13); P1-vv::Ac r1-sc:m3::Ds
T3306C T1-7(4444); P1-vv::Ac r1-sc:m3::Ds
T3306D T1-7(4405); P1-vv::Ac r1-sc:m3::Ds
T3306H T1-8(6591); P1-vv::Ac r1-sc:m3::Ds
T3306L T1-9(8302); P1-vv::Ac r1-sc:m3::Ds
T3306M T1-9(6762); P1-vv::Ac r1-sc:m3::Ds
T3306N T1-10g; P1-vv::Ac r1-sc:m3::Ds
T3307A Ac8178-2S
T3307D Ac8163-3S
T3307F Ac8183-3
T3308A Ac8200-4S
T3308B Ac6076-5L
T3308D Ac8175-5S
T3308E Ac8193-5S
T3308F Ac8179-5L
T3308G Ac8181-5L
T3308H Ac8186-5L
T3309A Ac8196-5L
T3309B Ac6062-6L
T3309C Ac6063-6
T3309D Ac8172-6L
T3309E Ac8184-6
T3310A Ac8161-7
T3310B Ac8173-7L
T3310D Ac8190-7
T3310E Ac8194-7
T3310F Ac8185-7L
T3311A Ac8162-8
T3311B Ac8182-8L
T3311D Ac6059-10S
T3311F Ac8180-10
T3312A Ds-1S1 P1-vv::Ac Dek1
T3312B Ds-1S2 P1-vv::Ac Dek1
T3312C Ds-1S3 P1-vv::Ac Dek1
T3312D Ds-1S4 P1-vv::Ac Dek1
T3312E Ds-1L1 P1-vv::Ac Bz2
T3312F Ds-1L3 Bz2; Ac
T3312G Ds-2S1 B1-Peru; P1-vv::Ac
T3312I Ds-2S3 B1-Peru; P1-vv::Ac
T3312J Ds-2S4; P1-vv::Ac
T3312L Ds-3L1 A1 Sh2; P1-vv::Ac
T3312M Ds-3L2 A1 Sh2; P1-vv::Ac
T3312O Ds-4L1 C2; P1-vv::Ac
T3312P Ds-4L3 C2; P1-vv::Ac
T3312Q Ds-4L4 C2; P1-vv::Ac
T3312S Ds-4L6 C2; P1-vv::Ac
T3312T Ds-4L7 C2; P1-vv::Ac
T3312U Ds-5L1 A2 Pr1 Bt1; P1-vv::Ac
T3312V Ds-5S1 A2 Pr1 Bt1; P1-vv::Ac
T3312W Ds-5S2 A2 Pr1 Bt1; P1-vv::Ac
T3312Y Ds-9S1 C1-I wx1; Ac
T3312Z Ds-10L2 R1-sc; P1-vv::Ac

B-A TRANSLOCATIONS (BASIC SET)

122A TB-1La
122B TB-1Sb
222A TB-1Sb-2L4464
222B TB-3La-2S6270
327A TB-3La
327B TB-3Sb
421A TB-4Sa
423E TB-4Lf
522A TB-5La
522C TB-5Sc
614B TB-6Sa
614C TB-6Lc
717A TB-7Lb
719A TB-7Sc
809A TB-8Lc
922B TB-9Lc Wc1
922D TB-9Sd
X21B TB-10L19
X22A TB-10Sc

B-A TRANSLOCATIONS (OTHERS)

122C TB1-Lc
123B TB-1La Bz2; bz2-m
126G TB-1Sb P1-vv::Ac bz2-m::Ds A1 A2 Bz1 C1 C2 R1
221I TB-2Sa B1-Peru
221J TB-2Sb
225A TB-3La-2L7285
225B TB-1Sb-2Lc
320P TB-1La-3Le
320Q TB-5La-3L(1)
320R TB-5La-3L(2)
320S TB-5La-3L(3)
327C TB-3Lc
327D TB-3Ld
329Z T3-B(La); T3-B(Sb)
331A TB-1La-3L5267
331B TB-1La-3L4759-3
331C TB-1La-3L5242
331E TB-3Lf
331F TB-3Lg
331G TB-3Lh
331H TB-3Li
331I TB-3Lj
331J TB-3Lk
331K TB-3Ll
331L TB-3Lm
420B TB-9Sb-4L6504
420I TB-9Sb-4L6222
421B TB-1La-4L4692
421C TB-7Lb-4L4698
421D TB-4Sa Su1; su1
423A TB-4Lb
423B TB-4Lc
423C TB-4Ld
423D TB-4Le
423F TB-1Sb-2L4464-4f
425A TB-4Sg
425B TB-4Lh
425C TB-4Li
428I Dt6 TB-4Sa
522B TB-5Lb
522D TB-5Ld
528D TB-1La-5S8041
614A TB-6Lb
627E TB-6Lc Dt2; A2 C1 C2 R1 a1-m
719B TB-7Sc Vp9; vp9
720A TB-7Lb Dt3; a1-m1::rDt (Neuffer)
806A TB-8La
806B TB-8Lb
921A TB-9La
921B TB-9Sb
921C TB-9Lc
922C TB-9Sb C1-I
929A IsoB9-9 isochromosome Type 1
929B IsoB9-9 isochromosome Type 2
929C T9-B(La); T9-B(Sb)
929D IsoB9-9 isochromosome (original)
929F T9-B (La + Sb)
929G TB-9Sb; T9-8(4453)
929H TB-9Sb; T9-3(6722)
929I TB-9Sb-1866
929J TB-9Sb-1852
929K TB-9Sb-2150
929L TB-9Sb-14
929M TB-9Sb-2010
TX40D TB-1Sb P1-vv::Ac r1-sc:m3::Ds
TX40E TB-3La a1-m Dt1

TX40F TB-8Lc Ac2 bz2-m::Ds
TX40G TB-9Sd a1-m Dt1
TX40H TB-9Lc Ac8168-9 r1-sc:m3::Ds
TX40I TB-10L18 P1-vv::Ac r1-sc:m3::Ds
X21A TB-10La
X21C TB-10Ld
X22B T1La-B-10L18
X22C TB-10Lb
X30A TB-10L1
X30B TB-10L2
X30C TB-10L3
X30D TB-10L4
X30E TB-10L5
X30F TB-10L6
X30G TB-10L7
X31A TB-10L8
X31B TB-10L9
X31C TB-10L10
X31D TB-10L11
X31E TB-10L12
X31G TB-10L14
X31H TB-10L15
X31I TB-10L16
X31J TB-10L17
X32A TB-10L18
X32C TB-10L20
X32D TB-10L21
X32E TB-10L22
X32F TB-10L23
X32G TB-10L24
X32H TB-10L25
X32I TB-10L26
X32J TB-10L27
X32K TB-10L28
X33A TB-10L29
X33B TB-10L30
X33C TB-10L31
X33D TB-10L32
X33F TB-10L34
X33G TB-10L35
X33H TB-10L36
X34A TB-10L37
X34B TB-10L38

INVERSION

I143A Inv1a (1.S.30; 1.L.50)
I143B Inv1c (1.S.30; 1.L.01)
I143C Inv1d (1.L.55; 1.L.92)
I143D Inv1k (1.L.46; 1.L.82)
I243A Inv2b (2S.06; 2L.05)
I243B Inv2h (2L.13; 2L.51)
I343A Inv3a (3L.38; 3L.95)
I343B Inv3b (3L.21; 3L.70)
I343C Inv3c (3L.05; 3L.95)
I343D Inv3(8582) (3S.55; 3L.82)
I344A Inv9a (9S.70; 9L.90)
I443A Inv4b (4S.10; 4L.12)
I443B Inv4c (4S.89; 4L.62)
I443C Inv4a (4L.30; 4L.90)
I443D Inv4d (4L.40; 4L.96)
I443E Inv4f (4L.17; 4L.63)
I444A Inv2a (2S.70; 2L.80)
I543A Inv4e (4L.16; 4L.81)
I543B Inv5a (5S.05; 5L.72)
I743A Inv5(8623) (5S.67; 5L.69)
I743B Inv6d (6S.70; 6L.33)
I743C Inv6(3712) (6S.76; 6L.63)
I743D Inv6a (6S.76; 6L.63)
I843A Inv6e (6S.80; 6L.32)
I943A Inv7f (7L.17; 7L.61)
I943B Inv7(8540) (7L.12; 7L.92)
I943C Inv7(3717) (7S.32; 7L.30)
I943E Inv7a (7L.05; 7L.95)
IX43A Inv8a (8S.30; 8L.15)
IX43B Inv9b (9S.05; 9L.87)

RECIPROCAL TRANSLOCATIONS (wx1 AND Wx1 MARKED)

wx01A T1-9c (9L.22; 1.S.48); wx1
wx01B T1-9(5622) (9L.12; 1.L.10); wx1
wx02A T1-9(4995) (9S.20; 1.L.19); wx1
wx02AA T1-9(4995) (9S.20; 1.L.19); wx1
wx03A T1-9(8389) (9L.13; 1.L.74); wx1
wx04A T2-9c (9S.33; 2S.49); wx1
wx05A T2-9b (9L.22; 2S.18); wx1
wx06A T2-9d (9L.27; 2L.83); wx1
wx07A T3-9(8447) (9L.14; 3S.44); wx1
wx08A T3-9(c**) (9S.20; 3S.15); wx1
wx09A T3-9(8562) (9L.22; 3L.65); wx1
wx10A T4-9e (9L.26; 4S.53); wx1
wx11A T4-9g (9L.27; 4S.27); wx1
wx12A T4-9(5657) (9S.25; 4L.33); wx1
wx13A T4-9b (9L.29; 4L.90); wx1
wx14A T5-9c (9L.10; 5S.07); wx1
wx14B T5-9(022-11) (9L.27; 5S.30); wx1
wx15A T5-9(4817) (9S.07; 5L.06); wx1
wx16A T5-9d (9L.10; 5L.14); wx1
wx17A T5-9a (9S.17; 5L.69); wx1
wx18A T6-9(4778) (9L.30; 6S.80); wx1
wx19A T6-9a (9L.40; 6S.79); wx1
wx19B T6-9e (9L.24; 6L.18); wx1
wx20A T6-9b (9S.37; 6L.10); wx1 y1
wx21A T6-9(4505) (9ctr.00; 6L.13); wx1
wx22A T7-9(4363) (9ctr.00; 7ctr.00); wx1
wx23A T7-9a (9S.07; 7L.63); wx1
wx24A T8-9d (9S.16; 8L.09); wx1
wx25A T8-9(6673) (9S.31; 8L.35); wx1
wx26B T9-10(059-10) (9S.31; 10L.53); wx1
wx27A T9-10b (9S.13; 10S.40); wx1
Wx30A T1-9c (9L.22; 1.S.48); Wx1
Wx30B T1-9(4995) (9S.20; 1.L.19); Wx1
Wx30C T1-9(8389) (9L.13; 1.L.74); Wx1
Wx31A T2-9c (9S.33; 2S.49); Wx1
Wx31B T2-9b (9L.22; 2S.18); Wx1
Wx31C T2-9d (9L.27; 2L.83); Wx1
Wx32A T3-9(8447) (9L.14; 3S.44); Wx1
Wx32B T3-9(8562) (9L.22; 3L.65); Wx1
Wx32C T3-9(c**) (9S.20; 3S.15); Wx1
Wx33A T4-9e (9L.26; 4S.53); Wx1
Wx33B T4-9(5657) (9S.25; 4L.33); Wx1
Wx33C T4-9g (9L.27; 4S.27); Wx1
Wx34A T5-9c (9L.10; 5S.07); Wx1
Wx34B T5-9(4817) (9S.07; 5L.06); Wx1
Wx34C T4-9b (9L.29; 4L.90); Wx1
Wx35A T5-9(8386) (9S.13; 5L.87); Wx1
Wx35B T5-9a (9S.17; 5L.69); Wx1
Wx35C T5-9d (9L.10; 5L.14); Wx1
Wx36A T6-9(4778) (9L.30; 6S.80); Wx1
Wx37A T6-9(8768) (9S.61; 6L.89); Wx1
Wx37B T7-9(4363) (9ctr.00; 7ctr.00); Wx1
Wx37C T6-9(4505) (9ctr.00; 6L.13); Wx1
Wx38A T7-9a (9S.07; 7L.63); Wx1
Wx38B T8-9d (9S.16; 8L.09); Wx1
Wx38C T8-9(6673) (9S.31; 8L.35); Wx1
Wx39A T9-10(8630) (9S.28; 10L.37); Wx1
Wx39B T9-10b (9S.13; 10S.40); Wx1

PHENOTYPE ONLY

aberrant seedling
abbt*-N399B
abbt*-N454C
abbt*-N594B
abbt*-N595B
abbt*-N712B

absence of leaf blade
bladeless*-87-2406-23

adherent leaf
ad*-87-2285-18
ad*-N111
ad*-N194
ad*-N1958
ad*-N253
ad*-N273B
ad*-N316
ad*-N377B
ad*-N452E
ad*-N512B
ad*-N551B
ad*-N556D
ad*-N582
ad*-N605B
ad*-N640
ad*-N664
ad*-N682B
ad*-N767
ad*-N877B
ad*-N984B

adherent tassel
ad*-N613B

albescent
al*-1479
al*-84-5020-32
al*-PI245132
wh top*-Bauman

albino seedling
nlw*-85-3357-17
peach-albino*-N1983B
peach-albino-mutable*-87-2209-30
w*-002-12
w*-005-19
w*-009-6
w*-010-4
w*-011-11
w*-017-14-A
w*-017-14-B
w*-020-9
w*-034-16
w*-037-14
w*-039-15
w*-2065
w*-2246
w*-3858
w*-4670
w*-4873
w*-5201
w*-5255
w*-5267
w*-56-3003-12
w*-5602
w*-5622
w*-5787
w*-5863
w*-6293
w*-6504
w*-6575
w*-7165
w*-7219
w*-7281
w*-74-1674-1
w*-78-297-3
w*-8105W
w*-8129
w*-8147
w*-8201
w*-84-5205-46
w*-84-5222-30
w*-85-3359-11
w*-85-3552-25
w*-85-3559-30
w*-8529
w*-8549
w*-8569
w*-86-1078-6
w*-86-1265-30
w*-86-2222-5
w*-8630
w*-8635
w*-8637
w*-8670
w*-87-2215-8
w*-8925
w*-8963
w*-8970
w*-8977
w*-8992
w*-9235
w*-B-75
w*-BYD
w*-Canario Hembrilla Enano
w*-Fino
w*-MontenegrinFlint
w*-N103
w*-N109
w*-N115
w*-N1158B
w*-N126A
w*-N137D
w*-N145
w*-N147B
w*-N167
w*-N176
w*-N178A
w*-N1834
w*-N1839
w*-N1847
w*-N1849
w*-N1854
w*-N186
w*-N1865

w*-N1890
w*-N1909
w*-N191
w*-N1915
w*-N192
w*-N21A
w*-N22
w*-N220
w*-N224
w*-N23
w*-N24
w*-N278A
w*-N285
w*-N304A
w*-N318
w*-N332
w*-N335
w*-N346A
w*-N355
w*-N364
w*-N367B
w*-N404
w*-N405A
w*-N413B
w*-N42
w*-N428B
w*-N430B
w*-N436A
w*-N456A
w*-N457
w*-N491B
w*-N5
w*-N509B
w*-N516A
w*-N524A
w*-N532
w*-N536A
w*-N540B
w*-N547A
w*-N558B
w*-N563A
w*-N569B
w*-N574
w*-N58
w*-N587C
w*-N593B
w*-N6
w*-N613C
w*-N621B
w*-N627B
w*-N67A
w*-N682A
w*-N704
w*-N708A
w*-N727A
w*-N729A
w*-N736A
w*-N77
w*-N804B
w*-N829B
w*-N883B
w*-N917B
w*-PI184276
w*-PI193438
w*-PI201543
w*-PI213747
w*-PI228176
w*-PI228179
w*-PI228183
w*-PI232961
w*-PI232965
w*-PI232968
w*-PI232972
w*-PI239103
w*-PI239110
w*-PI251009
w*-PI251885
w*-PI251930
w*-PI251932
w*-PI254851
w*-PI267162
w*-PI267179
w*-PI267204
w*-Singleton-16
w*-Singleton-22
w*-Singleton-24
w*-Singleton-25
w*-Singleton-31
w*-Tama
w*-wh-mut
wh*-053-4
wh*-2083
wh*-89-578-6

barren stalk
ba*-1447
ba*-68-679-8
ba*-74-304-12
ba*-74-369-2
ba*-PI200290
ba*-PI218135
ba*-PI239105
ba*-PI251885
ba-ub*-94-4712

bilateral coleoptile
blc*-N743C

bleached leaf
Blh*-N1455
blh*-N203B
blh*-N2302B
blh*-N2359
Blh*-N2421
blh*-N265A

blotched aleurone
Bh*-86-1381-1
Bh*-SF98-12
Bh-Tu*-Mumm

blotched leaf
bl*-N1278A
bl*-N43
red leaf blotch*-PI213779
yel-spl*-N152

brachytic plant
br*-2180
br*-78-136KEW
br*-Brawn219-221
br*-Brawn227-229
br*-Brawn230
br*-Brawn231-233
br*-Brawn235-237
br*-Brawn259-260
br*-Brawn261-262
br*-Brawn263-266
br*-Brawn267-268
br*-Brawn269-271
br*-Brawn272-273
br*-Brawn274-275
br*-OSIJEK-Yugoslavia
br*-PI228171
br*-PI239105
br*-Singleton-8
br*-Singleton1969-252
td*-PI262476

brevis plant
bv*-N2283

brittle endosperm
bt*-011-11
bt*-1979-14
bt*-1979-16
bt*-1982
bt*-4380
bt*-4539
bt*-4973
bt*-60-151
bt*-8101
bt*-8102
bt*-83-84-3541-1
bt*-84-4
bt*-84-5
bt*-84-5091-9
bt*-84-5257-1
bt*-84-6
bt*-85-3096-6
bt*-85-3098-15
bt*-85-3099-16
bt*-85-3372-27
bt*-87-2132-39
bt*-87-2297-1
bt*-87-88-2630-28
bt*-88-3177-14
bt*-88-3177-2
bt*-88-3177-7
bt*-8804
bt*-8805
bt*-89-1265-18
bt*-90286
bt*-A4109
bt*-Briggs-1998-1
bt*-F-15
bt*-F-23
bt*-F-31
bt*-F-34
bt*-F-36
bt*-F-8
bt*-F10
bt*-Panzio
bt*-PetersonResHy
bt*-PI200197
bt*-PI251887
bt-gm*-84-5045-39
bt-gm*-85-3017-24
bt-sh*-PI251930

brittle stalk
bk*-N888D

brown endosperm
brn*-1981-1
brn*-1981-2
brn*-1981-3
brn*-1981-4
brn*-84-23
brn-bt*-81-F-24

brown kernel
bnk*-N747B
lt-brn-sml*-86-1302-37

brown midrib
bm*-N2331B
bm*-PI228174
bm*-PI251009
bm*-PI251893
bm*-PI251930
bm*-PI262480
bm*-PI262485
bm*-PI267186

brown pericarp
bp*-Coates
bp*-Lima100
bp*-Lima94
bp*-PI183639

burned leaf
les*-Funk-4
les*-PI262474

chromosome breaking
Chrom-breaking*-Mu

clasping leaf
clsp*-87-2320-9
clsp*-88-89-3522-1

collapsed endosperm
cp*-N1076A
cp*-N1078B
cp*-N1092A
cp*-N1104B
cp*-N1229A
cp*-N1255B
cp*-N1275A
cp*-N1293
cp*-N1294
cp*-N1311C
cp*-N1313
cp*-N1318
cp*-N1319A
cp*-N1338
cp*-N1369
cp*-N1379A
cp*-N1385
cp*-N1393A
cp*-N1399A
cp*-N1405A
cp*-N1417
cp*-N1430
cp*-N1436A
cp*-N1527B
cp*-N2356B
cp*-N524E
cp*-N628
cp*-N863A
cp*-N886
cp*-N918A
cp*-N935
cp*-N936A
cp*-N937A
cp*-N968A
cp*-N991
cp*-N992

colored leaf
lc*-PI239110

colored plumule
Pu*-1976-RYDCO

colorless aleurone
cl*-85-86-3559-1
cl*-86-1478-16
cl*-N1333A
cl*-N1345A
cl*-N1346A
cl*-N720E
cl*-N795
cl*-N801
cl*-N818A

cl-crown-pale-base*-85-86-3558-23
r*-86-1590-6

colorless floury
clf*-N2425B

crinkled leaf
cr*-97P-111
cr*-98-1698
cr*-N769B

crossbanded leaf
cb*-N1620A
Cb*-N2290B
cb*-N696D
cb*-N719A

crumpled kernel
crp*-N1429A
crp*-N2207
dnj*-N1534

defective crown
dcr*-N1053A
dcr*-N1158A
dcr*-N1176B
dcr*-N1233A
dcr*-N1409
dcr*-N871A
dcr*-N925A

defective kernel
de*-1276
de*-17
De*-1976-RYDCO
de*-2080
de*-2192
de*-2424
de*-2915
de*-2919
de*-3188
de*-4309
de*-5044Hagie
de*-85-86-3567-35
de*-8505
de*-8507
de*-8508
de*-86-1472-6
de*-8808
de*-8809
de*-8810
de*-8811
de*-8818
de*-N1002A
de*-N1007A
de*-N1057B
de*-N1089
de*-N1101
de*-N1122A
de*-N1136A
de*-N1142
de*-N1149A
de*-N1162
de*-N1166
de*-N1177A
de*-N1196
de*-N1310B
de*-N1329A
de*-N1336B
de*-N1345B
de*-N1390A
de*-N1400
de*-N1403
de*-N1420
de*-N1520B
de*-N1897
de*-N2022
de*-N232B
de*-N260D
de*-N279B
de*-N296C
de*-N307D
de*-N400A
de*-N408D
de*-N513B
de*-N528C
de*-N540A
de*-N573A
de*-N660C
de*-N674A
de*-N748B
de*-N751A
de*-N760B
de*-N877A
de*-N891A
de*-N902A
de*-N903
de*-N929
de*-N932
de*-N939A
de*-N970A
de*-N979A
de*-N981A
de-sml*-8813
de-sml*-8814
de-sml*-8815
de-sml*-8816
de-sml*-8817
def*-8101
def*-8102
def*-8103
def*-8104
def*-8105
def*-8106
def*-8107
def*-8108
def*-8109
def*-8110
def*-8111
def*-8112
def*-8113
def*-8114
def*-8116
def*-8118
def*-8119
def*-8120
def*-8121
def*-8122
def*-8123
def*-8125
def*-8126
def*-8127
def*-8128
def*-8130
def*-8131
def*-8132
def*-8134
def*-8136
def*-8137
def*-8138
def*-8201
def*-84-22
def*-84-28
def*-84-29
def*-84-30
def*-84-31
def*-84-37
def*-84-40
def*-84-41
def*-84-45
def*-84-48
def*-84-49
def*-84-53
def*-84-54
def*-84-58
def*-84-60
dek*-1979-32
dek*-1981-1
dek*-74-0060-4
dek*-84-14
dek*-86-1496-35
dek*-8902
dek*-8903
dek*-8904
dek*-99-6273-1
dek*-F-16
dek*-PS602
wrinkled-de*-86-1473-5
wrinkled-gm*-86-1582-32

defective tassel
Tp*-54-55-Jos
Tp*-PI213734
Tp*-Pk41-Jos
Tp*-T8-Jos
Tp*-Tenn61

dented kernel
dnt*-N1185A
dnt*-N1326
dnt*-N884A

dilute aleurone color
dil*-N452D
dil*-N524C
dil*-N743A

discolored kernel
dsc*-N1084
dsc*-N1135A
dsc*-N1302
dsc*-N749
pig*-84-5080-18
pig*-86-1178-6
pig-gm*-1979-51
pig-gm*-1979-52
pig-gm*-1979-9
pig-gm*-1981-A
pig-gm*-1981-B
pig-gm*-1982-3
pig-gm*-5020-14
pig-gm*-84-5078-10
pig-gm*-86-1200-3
pig-gm*-87-2275-15
pig-gm*-87-2305-22
pig-gm*-Briggs 1998-1
pig-gm*-Briggs 1998-2
pig-gm*-PI251930
ptd-dek*-1976-RYDCO
ptd-dek*-1981
ptd-dsc*-87-2490-22
sml-pig-gm*-88-89-3554-44

distichous ear
distichous*-68-1227

distorted
dst*-N444A

distorted segregation
off-ratio*-85-3255-6
off-ratio*-86-1155-1
wx-off-ratio*-86-1110-4

dull endosperm
du*-Sprague

dwarf plant
d*-018-3
d*-119
d*-136-220
d*-1821
d*-2108
d*-2201
d*-2447-8
d*-3-eared-JC
d*-3047
d*-5312
d*-56-3037-23
d*-60-2428
d*-64-4156-1
d*-74-1701-5
d*-75-6071-1
d*-76-1304-9
d*-76-2186
d*-78-282-3
d*-78-286-1
d*-78-286-5
d*-85-3081-33
d*-87-2198-36
d*-Brawn254-258
d*-gl11
d*-MarovacWhiteDent
d*-N1095B
d*-N1352B
d*-N157B
d*-N1883
d*-N1895
d*-N197A
d*-N203D
d*-N2295
d*-N282
d*-N299B
d*-N403B
d*-N454A
d*-N518A
d*-N549B
d*-N604
d*-N629B
d*-N699B
d*-N994B
d*-N998B
d*-PI180231
d*-PI183644
d*-PI184286
d*-PI213769
d*-PI228169
d*-PI228171
d*-PI239110
d*-PI245132
d*-PI251652
d*-PI251656
d*-PI251885
d*-PI254854
d*-PI262495
d*-PI267219
d*-rosette
d*-shlf-9-436-1
d*-su
d*-su2
d*-Teo
d*-ts1

ectopic coleoptile
ect*-N641B

erect leaf
dge*-N2410

etched endosperm
et*-3130
et*-3576
et*-5191
et*-6-9321-1
et*-73-766-1
et*-8-M-4
et*-84-5266-26
et*-84-5270-40
et*-85-86-3518-21
et*-86-1493-6
et*-8616
et*-87-2349-13
et*-88-89-3525-22
et*-88-89-3554-33
et*-89-90-1547-19
et*-89-90-1548-13
et*-90-3222-13
et*-Mu1767
et*-Mu2349
et*-N1001A
et*-N1078A
et*-N1332
et*-N1361
et*-N164B
et*-N185B
et*-N357C
et*-N403A
et*-N489A
et*-N509A
et*-N514A
et*-N516C
et*-N518B
et*-N561B
et*-N571A
et*-N586A
et*-N615A
et*-N617
et*-N629F
et*-N643A
et*-N670A
et*-N680C
et*-N701A
et*-N702A
et*-N723A
et*-N724D
et*-N745
et*-N76D
et*-N789
et*-N798A
et*-N818B
et*-N837A
et*-N861
et*-N864A
et*-N868A
Et*-N876A
et*-N953A
et*-N965
et*-Osturana
et-de*-88-89-3526-8
et-gm*-86-1475-34
et-gm*-86-87-1742-38
et-gm*-87-2502-19
et-mutable*-87-2519-31
granular-o*-84-5274-30
sh*-N972A
sml-et*-85-3522-29
su-sh-et*-98-1887-1

faded leaf
fd*-N1938

fine stripe leaf
str*-PI228164

flecked leaf
flk*-N403C
flk*-N527A
flk*-N564B
flk*-N570A
flk*-N630B
flk*-N653A

flint kernel
flint*-87-2126-22

floury endosperm
fl*-67-412
fl*-78-513
fl*-83-3386-19
fl*-84-44
fl*-8515
fl*-Mojo
fl*-N1145A
fl*-N1163
fl*-N1208A
fl*-N1287
fl*-N1308A
fl*-N1333B
fl*-N1426
fl*-N7B-65-1294
fl*-N872A
fl*-shoepeg
fl*-sucaxo
fl-cap*-1981
fl-cap*-66-519-1
fl-de*-8905
sml-fl-cap*-1981

germless
brn-gm*-85-3315-6
brn-gm*-85-86-3587-46
brn-gm*-85-86-3595-3
brn-gm*-86-1161-5
emb*-85-3100-32
emb*-85-3378-8
gm*-1387
gm*-1979-11
gm*-1979-53
gm*-5234
gm*-84-5087-4
gm*-8510
gm*-86-1011-2
gm*-86-1013-4
gm*-86-1097-3
gm*-86-1335-1
gm*-86-1591-7
gm*-86-87-1742-18
gm*-87-2456-9
gm*-N1292
gm*-N1303
gm*-N1311B
gm*-N1312
gm*-N1319B
gm*-N1390C
gm*-N198C
gm*-N869A
gm*-N928B
o-gm*-83-3398-6
o-gm*-84-33
o-gm*-84-44
o-gm*-98-5733-1
pr-gm*-86-1109-1
sh-gm*-84-5045-32
sh-gm*-88-3082-4
sml-dsc-gm*-95W-240
sml-o-gm*-86-1323-4
w-o-gm*-85-3135-4
w-o-gm*-86-1349-1
w-o-gm*-88-3270-10
y-gm*-85-3288-28

glassy endosperm
ae*-6921
ae*-84-7
ae*-92-1365-3
ae*-96-1449-1
ae*-Briggs 1998-1
ae*-Mu32

glossy leaf
gl*-1-3(5476)
gl*-218-1
gl*-32TaiTaiTaSarga
gl*-4339
gl*-5201
gl*-5249
gl*-56-3023-6
gl*-56-3023-9
gl*-56-3036-7
gl*-6
gl*-60-2484-8
gl*-63-2440-8
gl*-85-3095-12
gl*-8654
gl*-87-2215-30
gl*-87-2215-8
gl*-87-2278-34
gl*-88-3142-4
gl*-97P-261-5
gl*-Bizika
gl*-gl12
gl*-LGC-117
gl*-LGC-27
gl*-Loesch
gl*-Manglesdorf
gl*-Moritsa
gl*-N168
gl*-N203C
gl*-N356
gl*-N546C
gl*-N616A
gl*-N656A
gl*-N681A
gl*-N696E
gl*-PI184286
gl*-PI200203
gl*-PI228177
gl*-PI232974
gl*-PI239101
gl*-PI239110
gl*-PI251885
gl*-PI251933
gl*-PI262474
gl*-PI262476
gl*-PI262494
gl*-PI262500
gl*-PI267203
gl*-PI267209
gl*-PI267212
gl*-STL
gl-nec*-N516D

gnarled plant
Gn*-sgl

gravity non-responsive primary root
agt*-N491C

green striped leaf
gs*-98-5700-5
gs*-N359A
gs*-N484A

gritty leaf
gtl*-N2297

high chlorophyll fluorescence
hcf*-88-3005-3

knotted husks
mwp*-Nelson

lazy plant
la*-N2333B

lemon white
lw*-1979-45
lw*-1981-10
lw*-1998-1
lw*-73-2548
lw*-82-1
lw*-85-3076-28
lw*-85-3252-5
lw*-86-87-1828-7
lw*-87-2407-36
lw*-88-3177-2
lw*-89-90-3609-5
lw*-B73
lw-y-pg*-1998-4
lw-y-pg*-Funk-81-5
lw-y-pg*-PI200303
pale-y*-1981
pale-y*-83-84-3549-13
pale-y*-84-5082-33
pale-y*-84-5167-48
pale-y*-84-5288-19
pale-y*-85-3005-22
pale-y*-85-3006-30
pale-y*-85-3007-40
pale-y*-85-3010-40
pale-y*-85-3016-15
pale-y*-85-3017-31
pale-y*-85-3065-25
pale-y*-85-3069-6
pale-y*-85-3087-29
pale-y*-85-3377-2
pale-y*-86-1155-3
pale-y*-87-88-2679-1
pale-y*-88-89-3551-35
pale-y*-89-1313-3
pale-y*-89-90-1525-23
pale-y*-90-3220-1
pale-y*-90-3220-26
w*-N677
w*-N70
wh*-BMS-Rhoades
y-pg*-1981-17
y-pg*-84-5275-14
y-pg*-85-3042-7
y-pg*-85-3044-34
y-pg*-85-3078-41
y-pg*-85-3562-31
y-pg*-85-86-3533-9
y-pg*-86-1151-7
y-pg*-86-87-1723-27
y-pg*-87-2160-16

lesion
les*-2119
les*-74-1873-9
les*-ats

Les*-N1378
Les*-N1450
les*-N2290A
Les*-N2420
Les*-N502C

liguleless
Lg*-64-36
lg*-PI228170

loose pericarp
lsp*-N1045

luteus yellow seedling
l*-009-2
l*-009-6
l*-017-3
l*-025-4
l*-062-3
l*-2215
l*-2673
l*-4356
l*-4545
l*-4871
l*-5-9b[X-7-39]
l*-549-1 Derived Flint
l*-56-3003-12
l*-570-2 Cincantin
l*-5783-straw
l*-62-489-2
l*-6474
l*-6923
l*-6973
l*-7165
l*-7281
l*-73-563
l*-77-564-2
l*-7748
l*-8321
l*-8376
l*-84-5205-13
l*-84-5225-33
l*-8495
l*-85-3215-2
l*-85-3225-4
l*-85-3457-40
l*-85-3513-1
l*-85-3541-20
l*-86-1112-1
l*-86-1354-9
l*-8613
l*-8634
l*-88-89-3555-13
l*-89-90-1552-10
l*-8966
l*-8970
l*-d-8694
l*-leng
l*-LGC-43
l*-LGC-74
l*-Moritza (Bulgaria)
l*-N104
l*-N113
l*-N1145B
l*-N119
l*-N124B
l*-N129
l*-N137B
l*-N140
l*-N171A
l*-N175
l*-N1806B
l*-N1838
l*-N1878
l*-N188A
l*-N1908
l*-N1920
l*-N195
l*-N209
l*-N218
l*-N231
l*-N251
l*-N31
l*-N336
l*-N347
l*-N368B
l*-N392A
l*-N416A
l*-N438A
l*-N496B
l*-N52
l*-N523
l*-N606
l*-N612B
l*-N62
l*-N691A
l*-N703
l*-N730
l*-PI183642
l*-PI183643
l*-PI193433
l*-PI193435
l*-PI193436
l*-PI195245
l*-PI213737
l*-PI213745
l*-PI218038
l*-PI228183
l*-PI239110
l*-PI239114
l*-PI251884
l*-PI254854
l*-PI254856
l*-PI262495
l*-PI267215
l*-PI267226
l*-Rumanian Flint
l*-Tama
l*-y wx 6-9b
l-nec*-2001-519
pyg*-N761
y-l*-85-3234-6
y-l*-8910 Briggs
yd*-87-2278-34
yel*-5344
yel*-8721
yel*-8793
yg*-8962

male sterile
Ms*-2471
ms*-6015
ms*-6025
ms*-6026
ms*-6033
ms*-6039
ms*-6045
ms*-6048
ms*-6049
ms*-6052
ms*-6053
ms*-6054
ms*-6055
ms*-6057
ms*-6058
ms*-6059
ms*-6060
ms*-6061
ms*-6062
ms*-6064
ms*-6065
ms*-6066
ms*-N2415
Ms*-N2474
ms*-N2484
ms*-N352C
ms*-N45B
ms*-PI217219
ms-si*-355

many tillers
tlr*-N2243

marbled aleurone
Dap*-3
dap*-86-8126-2
Dap*-89-3177.0
Dap*-89-3177.5
Dap*-89-3178.3
Marbled*-Sprague

miniature kernel
de*-N663C
mn*-1981-51
Mn*-866248U
mn*-87-2215-17
mn*-87-2346-20
mn*-87-2347-36
mn*-87-2422-14
mn*-88-3177-2
mn*-88-89-3509-40
mn*-88-89-3564-25
mn*-N1536
Mn*-N273C
mn*-N378C
mn*-N894
mn*-PI239110
mn*-PI245132
sml-k*-97-4784-1

mosaic aleurone color
msc*-N593A

mottled aleurone
Mt*-2313
Mt*-65-2238
Mt*-Sprague

mottled kernel
Mt*-N1343A

multiple aleurone layer
Mal*-Galinat
Mal*-Nelson
Mal*-PI515052

nana plant
na*-N1519D

narrow leaf
nl*-5688
nl*-N232A
nl*-N410B
nl*-N462B
nl*-N543B
nl*-N622B
nl*-N625
nl*-N727B
nl*-N732A
nl*-N797B
nl*-PI245132
stf*-N601
stk*-N363

necrotic leaf
ll*-N248A
ll*-N264
ll*-N417D
ll*-N623
nec*-011-7
nec*-017-3
nec*-4871
nec*-4889
nec*-5588 early
nec*-5619
nec*-5876
nec*-77-549-2
nec*-77-574-1
nec*-8624
nec*-8737
nec*-fraz
nec*-N1119B
nec*-N1185B
nec*-N1487
nec*-N193
nec*-N200B
nec*-N215F
nec*-N283B
nec*-N419
nec*-N430A
nec*-N465
nec*-N468
nec*-N490A
nec*-N510A
nec*-N541B
nec*-N545B
nec*-N559
nec*-N562
nec*-N581
nec*-N596C
nec*-N599A
nec*-N650D
nec*-N666A
nec*-N712C
nec*-N811B
nec*-N814
nec*-PI228174
nec*-PI267184
nec*-Vasco
nec-pg*-PI239116
shootless*-99-677-6

necrotic leaf tips
nec*-N669

oil yellow plant
oy*-N2360A

opaque endosperm
lrg-o-crown*-89-1275-17
o*-1979-54
o*-1981-11-Fox-19
o*-1981-3-Fox-7
o*-1981-5-Fox-9
o*-1981-6-Fox-10
o*-1981-8-Fox-15
o*-1982
o*-1982-2-Fox-13
o*-2-Fox-6
o*-3015
o*-73-798-1
o*-76GH-76
o*-8129
o*-82:288-1
o*-83-84-3549-39
o*-84-5025-15

o*-84-5025-17
o*-84-5025-8
o*-84-5044-35
o*-84-5091-13
o*-84-5094-4
o*-84-5095-23
o*-84-5117-16
o*-84-5261-37
o*-84-5270-40
o*-84-5282-27
o*-84-5295-13
o*-84-5321-28
o*-84-5324-29
o*-84-8a
o*-85-3084-8
o*-85-3088-3
o*-85-3335-35
o*-86-87-1767-10
o*-87-2285-33
o*-87-2350-2
o*-88-89-3550-27
o*-97-4784-6
o*-Briggs-1998-1
o*-BS20-Fox-3
o*-Fox-12
o*-N1008A
o*-N1037A
o*-N1039
o*-N1046
o*-N1065A
o*-N1074A
o*-N1119A
o*-N1172A
o*-N1189A
o*-N1195A
o*-N1209
o*-N1218
o*-N1228
o*-N1244A
o*-N1245
o*-N1298
o*-N1301
o*-N1304
o*-N1310A
o*-N1320A
o*-N1355
o*-N1358
o*-N1422
o*-N1433
o*-N436C
o*-N491A
o*-N696A
o*-N829C
o*-N870
o*-N885A
o*-N895
o*-N899
o*-N906A
o*-N908
o*-N910
o*-N915
o*-N930
o*-N938A
o*-N941
o*-N948B
o*-N969A
o*-N973
o*-N989
o*-N995A
o*-N996
o*-N999A
o*-PI195245
o*-PI200285
o-de*-1981-9-Fox-18
o-dek*-6
o-dek*-87-2279-12
o-sh*-86-1297-2
o-sh*-F1979-19
os*-2162
pro*-Mu1
sml-o*-87-88-2692-5
sml-o*-PI195243

orobanche
oro*-6577
oro*-69-9291-8
oro*-84-5080-15
oro*-85-3087-3
oro*-85-3106-41
oro*-85-3113-11
oro*-88-3237-31
oro*-88-89-3550-32

pale aleurone
pa*-N893A
pa*-N917A
pale-Cl*-86-1476-14
pale-Cl*-LGC65
pale-Cl-gm*-84-5251-1

pale aleurone, with pigmented sectors
pa-Cl*-m-86-1474-39
pa-Cl*-m-86-1478-4
pa-Cl*-m-87-2224-33

pale crown
pa-crown*-85-86-3558-23

pale green plant
pg*-56-3012-10
pg*-8321
pg*-Hy2 Nob 7-5
pg*-LGC-61
pg*-N1074B
pg*-N607
pg*-N671

pale green seedling
pas*-90-3222-13
pg*-2142
pg*-5619
pg*-6372
pg*-69-5079-2
pg*-6923
pg*-7122
pg*-8129
pg*-84-5234-29
pg*-8412
pg*-8911
pg*-8959
pg*-Caspar
pg*-Fino
pg*-N102
pg*-N11
pg*-N1161B
pg*-N12
pg*-N123C
pg*-N124A
pg*-N127
pg*-N1322B
pg*-N1389B
pg*-N146A
pg*-N1476
pg*-N147A
pg*-N150A
pg*-N155A
pg*-N156B
Pg*-N1604
pg*-N161
pg*-N181
pg*-N1821
pg*-N1822A
pg*-N1824
pg*-N1825
pg*-N1827A
pg*-N1866
pg*-N1881
pg*-N1885
pg*-N1983A
pg*-N213
pg*-N215B
pg*-N222
pg*-N247B
pg*-N272C
pg*-N296A
pg*-N338A
pg*-N346B
pg*-N349
pg*-N35
pg*-N357B
pg*-N361A
pg*-N362A
pg*-N36A
pg*-N375B
pg*-N379
pg*-N380
pg*-N381
pg*-N384B
pg*-N40
pg*-N408C
pg*-N417A
pg*-N421
pg*-N429B
pg*-N441
pg*-N445
pg*-N450
pg*-N452C
pg*-N459
pg*-N45A
pg*-N46
pg*-N469
pg*-N478A
pg*-N481
pg*-N484B
pg*-N485
pg*-N506A
pg*-N507A
pg*-N511
pg*-N514B
pg*-N520
pg*-N524B
pg*-N526C
pg*-N535
pg*-N550
pg*-N556B
pg*-N558A
pg*-N570C
pg*-N590B
pg*-N596B
pg*-N597B
pg*-N59B
pg*-N600A
pg*-N603
pg*-N615B
pg*-N618
pg*-N619
pg*-N638
pg*-N639
pg*-N641A
pg*-N654B
pg*-N660A
pg*-N663B
pg*-N673A
pg*-N683A
pg*-N686B
pg*-N701B
pg*-N719C
pg*-N71A
pg*-N724B
pg*-N725A
pg*-N73A
pg*-N76A
pg*-N805
pg*-N812C
pg*-N816A
pg*-N836
pg*-N855C
pg*-N884B
pg*-N896B
pg*-N897B
pg*-N906B
pg*-PI183648
pg*-PI193424
pg*-PI251930
pg*-PI262473
pg*-PI262495
pg*-PI267162
pg*-PI267215
pg-nec*-RJL-6527

pale pale green leaf
ppg*-N1474B
ppg*-N1963
ppg*-N393B
ppg*-N406A
ppg*-N427A
ppg*-N449
ppg*-N458B
ppg*-N881B

pale yellow endosperm
lw*-8509
lw*-8513
lw*-8514
pale-endo*-73-3
pale-endo*-73-4004
pale-y*-83-3382-16
pale-y*-83-3382-18
pale-y*-83-84-3548-25
pale-y*-84-5103-16
pale-y*-85-3016-30
pale-y*-85-3036-38
pale-y*-85-3134-46
pale-y*-85-3374-13
pale-y*-85-3511-18
pale-y*-86-1155-2
pale-y*-87-2339-10
pale-y*-87-2350-2
pale-y*-87-2350-25
pale-Y*-87-2422-14
pale-y-gm*-Rsssc-77-110
pale-y-o*-84-5288-2
pale-y-o*-86-1296-27
y*-84-5272-12
y*-84-5288-1
y*-85-3041-2
y*-85-3087-12
y*-85-3125-7

papyrescent glumes
en*-Sprague

patched leaf
ptc*-N238A
ptc*-N444B

ptc*-N611
ptc*-N904B

piebald leaf
pb*-2-7-4400
pb*-87-2442-5
pb*-N1386C

pigmy plant
py*-N656B
py*-N714

pitted kernel
ptd*-N1425A
ptd*-N660E
ptd*-N738B
ptd*-N855A
ptd*-N901A
ptd*-N923

polytypic ear
pt*-McClintock
pt*-Mu
pt*-N868B

purple pericarp
Pl*-CFS-69

ragged leaf
rgd*-N2266

ragged seedling
rgd*-N203E
rgd*-N2290C
rgd*-N261B
rgd*-N378B

ramosa
ra*-412E
ra*-4889

red aleurone
pr*-N707A
pr*-N850

red pericarp
r*-ch-Burbank-CFS-80
r*-ch-PI213730

red seedling leaf
red-leaf*-86-1569-7

red silk scar
red-silk-scar*-MTC

reduced pollen fertility
ga*-0188
ga*-0213
ga*-0648
ga*-3615
ga*-91-5197-2
ga*-94-764
Ga*-Yugoslavia

rolled leaf
rld*-N1405B
rld*-N1525
Rld*-N2465
rld*-N556C

rough kernel
rgh*-N1060
rgh*-N1164B
rgh*-N1210
rgh*-N1306A
rgh*-N1524
rgh*-N799A
rgh*-N802
rgh*-N882
rgh*-N974A

shrunken kernel
pale-y-su-sh*-88-3133-28
sh*-1979-10
sh*-1981-14
sh*-1982-2
sh*-2927-Mumm
sh*-2928-Mumm
sh*-83-3328-24
sh*-84-3
sh*-84-5248-20
sh*-84-5317-44
sh*-85-3045-7
sh*-85-3104-27
sh*-85-3112-20
sh*-85-3375-38
sh*-8502
sh*-8503
sh*-8506
sh*-8511
sh*-8517
sh*-86-1565-17
sh*-87-2045-25
sh*-87-2045-6
sh*-87-2050-1
sh*-87-2050-3
sh*-87-2213-19
sh*-87-2215-12
sh*-87-2355-29
sh*-87-2406-3
sh*-87-2496-21
sh*-88-89-3540-1
sh*-8806
sh*-8807
sh*-8906
sh*-8907
sh*-97P-29-5
sh*-F-11
sh*-F-2
sh*-F-25
sh*-KERR
sh*-N1080
sh*-N1105B
sh*-N1147
sh*-N1320B
sh*-N1341
sh*-N1366
sh*-N1519B
sh*-N1969
sh*-N208C
sh*-N252B
sh*-N399A
sh*-N627A
sh*-N689
sh*-N741
sh*-N742
sh*-N750
sh*-N757
sh*-N785A
sh*-N819
sh*-N849
Sh*-N881A
sh*-N887A
sh*-N911
sh*-N961
sh*-PI596356
sh*-RJL
sh-bt*-85-3392-31
sh-crown*-Briggs-1998-1
sh-de*-6607
sh-de*-RSSSC-117
sh-fl*-9180
sh-fl*-9392
sh-o*-87-2410-24
sh-o*-87-2455-7
sh-wx*-F-18
su-sh*-F-5

silky
si*-0443
si*-0503
si*-8104
si*-N1967A

small kernel
smk*-N1003
smk*-N1019
smk*-N1057A
smk*-N1085A
smk*-N1115A
smk*-N1160
smk*-N1165A
smk*-N1168A
smk*-N1203
smk*-N1437
smk*-N1529
smk*-N215D
smk*-N227B
smk*-N320
smk*-N433A
Smk*-N845B
smk*-N890A
smk*-N909A
smk*-N942
smk*-N943
smk*-N994A

small plant
d*-N1074C
d*-N137C
d*-N149
d*-N164A
d*-N188B
D*-N2023
d*-N208B
d*-N210
d*-N2254
d*-N262C
d*-N287B
d*-N305
d*-N328
d*-N394
d*-N524D
d*-N528B
d*-N553D
d*-N707B
D*-N987B
smp*-N121
smp*-N135B
smp*-N153B
smp*-N156A
smp*-N183B
smp*-N1954
smp*-N272A
smp*-N306
smp*-N452B
smp*-N586B
smp*-N600B
smp*-N602
Smp*-N842

small seedling
d*-N155B
d*-N230A
d*-N254
d*-N266B
d*-N293B
d*-N408A
d*-N429A
d*-N526B
smp*-N1956
smp*-N276B
smp*-N279A
smp*-N630C
smp*-N751B
sms*-N1221B
sms*-N1964
sms*-N1971
sms*-N204B
sms*-N252A
sms*-N311C
sms*-N369B
sms*-N566
sms*-N570B
sms*-N666C
sms*-N680B

speckled leaf
spc*-N112
spc*-N1814
spc*-N198B
spc*-N357A
spc*-N370

spotted aleurone
cl-mut*-85-86-3564-1
cl-mut*-97-4782-9
cl-mut*-99-2170
coarse-mutable*-86-1417-7
Dt*-a; a1-m
Dt*-b; a1-m
Dt*-c; a1-m
Dt*-d; a1-m
Dt*-e; a1-m
Dt*-f; a1-m
Dt*-g; a1-m
Dt*-h; a1-m
Dt*-i; a1-m
fine mut*-86-1283-45
spk*-N551A
spk*-N600C
spk*-N687A

spotted leaf
les*-74-1820-6
spt*-N278B
spt*-N412A
spt*-N513A
spt*-N579B
spt*-N939B

stiff leaf
stf*-N1092C
stf*-N235B

streaked leaf
stk*-N1143C
stk*-N351
stk*-N368A
stk*-N433B
stk*-N584A
stk*-N587D
stk*-N589
stk*-N670B
stk*-N769C

stk*-N777B
stk*-N812B
stk*-N835A
stk*-N925B

striate leaf
Sr*-N2430
sr*-N675B

stubby plant
stb*-N938C

sugary kernel
su*-1979-8
su*-83-3383-21
su*-84-5350-2
su*-85-3133-32
su*-8504
su*-8803
su*-89-1279-14
su*-L874261
su*-N1040
su*-N236C
su*-N748A
su*-N817
su-sh*-F-22

tassel seed
Ts*-N1374
ts*-PI251881
ts*-PI267209
ts*-Sprague

tasselless
tls*-Funk
tls*-Va35

tiny plant
ty*-N1975
ty*-N215A
ty*-N236B
ty*-N326C
ty*-N702B

torn leaf
Trn*-N2438

translucent leaf
trans-leaf*-56-3122-7
trans-leaf*-68F-958
trans-leaf*-78-314
trans-leaf*-79-6533
trans-leaf*-PI228176

tube leaf
fused-leaves*-N36B
fused-leaves*-N835B
fused-leaves*-PI228170

tunicate
Tu*-5090B

unpaired rows
up*-Shirer

virescent seedling
l*-N184
pg*-N1171D
pg*-N39
v*-002-17
v*-007-18
v*-022-17
v*-025-4
v*-037-5
v*-1-2(5376)
v*-1-9(5622)
v*-2-9(5257)
v*-388-Sprague
v*-4308
v*-4698
v*-5-10(5355)
v*-5287
v*-5413
v*-5575
v*-56-3012-10
v*-5828
v*-60-151
v*-60-2397-15
v*-65-1433
v*-7230
v*-7281
v*-7312
v*-74-1690-1
v*-74-1873-1
v*-74-1948-1
v*-77-549-2
v*-8070
v*-8129
v*-8201
v*-8339
v*-8522
v*-8613
v*-8654
v*-8743
v*-8806
v*-8957
v*-8958
v*-9026
v*-Funk-84-13
v*-Funk-84-9
v*-leng
v*-LGC-111
v*-LGC-142
v*-LGC-98
v*-N1007B
v*-N1029B
v*-N1085B
v*-N110
v*-N1133B
v*-N1135B
v*-N1136C
v*-N114A
v*-N1151
v*-N116
v*-N1177B
v*-N1214B
v*-N125
v*-N1268A
v*-N128
v*-N131
v*-N133
v*-N134
v*-N135A
v*-N143B
v*-N153A
v*-N158
v*-N16
v*-N171B
v*-N179
v*-N1799
v*-N1806A
v*-N1836
v*-N183A
v*-N187
v*-N1873
v*-N1886
v*-N19
v*-N1912
v*-N1966
v*-N201
v*-N206A
v*-N2260
v*-N229
v*-N243
v*-N245
v*-N246
v*-N26
v*-N260C
v*-N280
v*-N289
v*-N29
v*-N298
v*-N303
v*-N330B
v*-N34
v*-N341
v*-N352B
v*-N358C
v*-N366
v*-N376
v*-N378A
v*-N395
v*-N397
v*-N398A
v*-N400B
v*-N414
v*-N41B
v*-N422B
v*-N423
v*-N439A
v*-N447B
v*-N463
v*-N467
v*-N470A
v*-N473B
v*-N499
v*-N50
v*-N517
v*-N51B
v*-N526A
v*-N528A
v*-N529B
v*-N53A
v*-N54A
v*-N54B
v*-N560
v*-N587A
v*-N620
v*-N621A
v*-N634A
v*-N64A
v*-N655A
v*-N65B
v*-N660D
v*-N661A
v*-N674B
v*-N678B
v*-N698B
v*-N699A
v*-N69A
v*-N710B
v*-N713B
v*-N724E
v*-N728
v*-N735
v*-N748C
v*-N75
v*-N756A
v*-N766C
v*-N770
v*-N779A
v*-N7B
v*-N806C
v*-N820
v*-N826
v*-N829A
v*-N839
v*-N840
v*-N84B
v*-N878B
v*-N891C
v*-N892B
v*-N909B
v*-N947C
v*-N956C
v*-N970B
v*-N972B
v*-N998D
v*-N999B
v*-pb-3019-16
v*-PI180165
v*-PI180231
v*-PI183640
v*-PI183648
v*-PI185851
v*-PI195244
v*-PI195245
v*-PI200197
v*-PI200201
v*-PI218042
v*-PI228174
v*-PI228176
v*-PI232974
v*-PI236996
v*-PI239105
v*-PI239114
v*-PI239116
v*-PI251883
v*-PI251891
v*-PI251930
v*-PI254856
v*-PI254857
v*-PI262476
v*-PI262487
v*-PI262489
v*-PI267184
v*-PI267209
v*-PI267212
v*-PI270293
v*-Pollacsek
v*-RumanianFlint
v*-Singleton-22
v*-Singleton-34
Vsr*-N1447
wst*-N643B

viviparous kernel
pale-vp*-87-2286-1
pale-vp*-87-2286-18
pale-vp*-87-2286-2
pale-vp*-87-2286-25
pale-vp*-87-2286-3
pale-y*-84-5027-22
pale-y*-84-5032-21
pale-y-vp*-83-3100-31
pale-y-vp*-83-3124-33
pale-y-vp*-84-5266-5
pale-y-vp*-85-3140-15
pale-y-vp*-85-3240-5
pale-y-vp*-85-3267-6
pale-y-vp*-85-3267-9
pale-y-vp*-85-3385-34
pale-y-vp*-86-1316-27
pale-y-vp*-88-3177-14
ps*-85-3288-28
ps*-85-3492-36

ps*-85-86-3567-1
ps*-86-1105-2
ps*-86-1352-4
ps*-86-1499-3
ps*-86-87-1742-18
ps*-89-90-1588-37
ps*-90-3222-27
ps*-90-91-8549-7
ps*-96-5032-6
ps*-98-5691-5
ps*-99-2157-1
ps*-Mu85-3061-21
ps*-Mu86-1105-1
vp(ps)*-86-1449-3
vp(ps)*-86-1565-17
vp*-0118
vp*-0315
vp*-2-8c
vp*-2000PR-1
vp*-73-30173
vp*-8101
vp*-8104
vp*-8106
vp*-8107
vp*-8108
vp*-8109
vp*-8110
vp*-8111
vp*-8112
vp*-8113
vp*-8114
vp*-8115
vp*-8116
vp*-8117
vp*-8201
vp*-8203
vp*-8204
vp*-8208
vp*-8209
vp*-8210
vp*-8211
vp*-84-5079-29
vp*-84-5279-29
vp*-84-5315-29
vp*-8418
vp*-8420
vp*-85-3011-11
vp*-85-3017-9
vp*-85-3040-29
vp*-85-3042-7
vp*-85-3099-16
vp*-85-3135-4
vp*-85-3182-6
vp*-85-3250-1
vp*-85-3339-25
vp*-85-3422-13
vp*-85-86-3567-20
vp*-86-1109-1
vp*-86-1407-15
vp*-86-1573-27
vp*-87-2146-18
vp*-87-2213-19
vp*-87-2224-3
vp*-87-2274-37
vp*-87-2299-1
vp*-87-2339-1
vp*-88-89-3555-1
vp*-88-89-8625-5
vp*-89-1181-8
vp*-89-1279-14
vp*-89-90-1561-18
vp*-93-1017-2
vp*-95-2086-1
vp*-Funk-8101
vp*-N702C
vp*-PI183642
vp*-PI185847
vp*-PI200204
vp*-PI254854
vp*-PI430482
vp-de*-87-2406-23
vp-Y*-86-1267-31
vp-Y*-86-1361-7
w-vp*-84-5020-4
w-vp*-85-3014-6
w-vp*-85-3304-13
w-vp*-91-1859-8
w-vp*-91-2544-7
w-vp*-92-1408-1
y-vp*-0730
y-vp*-1982-1
y-vp*-1982-2
y-vp*-1999-059-10
y-vp*-2062-Coop
y-vp*-60-153
y-vp*-6961
y-vp*-73-2656
y-vp*-80-6118
y-vp*-81-5
y-vp*-8102
y-vp*-8103
y-vp*-8105
y-vp*-8206
y-vp*-8207
y-vp*-83-1A
y-vp*-83-3101-36
y-vp*-8336
y-vp*-84-13
y-vp*-8419
y-vp*-85-3572-30
y-vp*-8512
Y-vp*-87-2339-10
y-vp*-87-2340-36
y-vp*-8701
y-vp*-88-89-3563-33
y-vp*-88-89-3613-25
y-vp*-99-2226-1

waxy endosperm
wx*-0208
wx*-98-1406-6
wx*-N66C

white cap kernel
Wc*-1982-1
Wc*-DC
Wc*-Funk-81-22
Wc*-Funk-81-23
wc*-N1206A
wc*-N1349
wc*-N897A

white endosperm
y*-1979-46
y*-1981
y*-1981-14
y*-1981-18
y*-1982-3
y*-73-2
y*-73-2262-1
y*-73-2262-2
y*-73-2394
y*-73-324-1
y*-73-4035
y*-73-426
y*-73-6
y*-84-8b
y*-87-2201-3
y*-Funk-81-12
y*-Funk-81-13
y*-Funk-81-2
y*-Funk-81-20
y*-Funk-81-9
y*-Sprague
y*-syn-DOCI
y*-Williams-60-154

white luteus seedling
w*-5413
wl*-N1
wl*-N1213B
wl*-N122
wl*-N126B
wl*-N1270A
wl*-N1350B
wl*-N1384B
wl*-N144
wl*-N165A
wl*-N18
wl*-N1803
wl*-N1819
wl*-N1844
wl*-N1848
wl*-N1855
wl*-N1857
wl*-N1863
wl*-N189
wl*-N1928
wl*-N1931
wl*-N1949
wl*-N217A
wl*-N221
wl*-N241
wl*-N255
wl*-N283A
wl*-N290
wl*-N299A
wl*-N311B
wl*-N313
wl*-N315
wl*-N345A
wl*-N358A
wl*-N362B
wl*-N38B
wl*-N4
wl*-N401
wl*-N408B
wl*-N415
wl*-N416B
wl*-N44
wl*-N442
wl*-N448
wl*-N455
wl*-N466
wl*-N47
wl*-N500
wl*-N502B
wl*-N508
wl*-N538A
wl*-N551C
wl*-N554A
wl*-N567
wl*-N575A
wl*-N60
wl*-N629A
wl*-N636
wl*-N637A
wl*-N646
wl*-N648
wl*-N654A
wl*-N663A
wl*-N686A
wl*-N698A
wl*-N709B
wl*-N711A
wl*-N720B
wl*-N758A
wl*-N981B
wl*-N998C

white margins
whm*-N1462
whm*-N1464
whm*-N1465B
whm*-N1470

white sheath
ws*-N1979
ws*-N537D

white stripe leaf
li*-PI262476
str*-PI262474
str*-X09
ws*-N1815
wst*-N1469
wst*-N1877
wst*-N248B
wst*-N413A
wst*-N454B
wst*-N548
wst*-N564A
wst*-N696B

white stripe leaf (iojap-like)
ij*-N504A

white striped seedling
ij-mos*-8624
j*-N793
stk*-N359B
str*-2104-4 EBP
str*-2116-1 EBP
str*-5120B-Teo
str*-6-10-4307
str*-78-314-1
str*-78-314-4
str*-78-314-5
str*-84-5222-7
str*-86-1494-27
str*-PI262495
str-et*-PI184276
wst*-N173B
wst*-N190A
wst*-N66B

white tipped leaf
wt*-N308
wt*-N432A
wt*-N580B
wt*-N650A

wrinkled kernel
wr*-N1075A
wr*-N1389A
wr*-N156C
wr*-N553A
wr*-N612A

yellow green leaf
l*-N906C
pastel*-1-6-5495
ppg*-N1484
pyg*-N1266A
pyg*-N223
pyg*-N307B

pyg*-N321
pyg*-N460
yg*-0130
yg*-4369
yg*-4889
yg*-5-8(5575)
yg*-56-3021-18
yg*-6697
yg*-68-1429
yg*-6853
yg*-74-1827-1
yg*-77-585
yg*-8105
yg*-8379
yg*-8622
yg*-8631
yg*-8682
yg*-8692
yg*-8946
yg*-910J
yg*-B73
yg*-Caspar
yg*-N1314B
yg*-N1315B
yg*-N157A
yg*-N1800
yg*-N1840
yg*-N1910
yg*-N1948
yg*-N2021
yg*-N2246
Yg*-N2294
yg*-N37
yg*-N389B
yg*-N38A
yg*-N685B
yg*-N706B
yg*-N72
yg*-N769A
yg*-PI180231
yg*-PI228174
yg*-PI239114
yg*-PI267206
yg*-PI267224
yg*-Singleton-127
yg*-Singleton-23
yg*-Singleton-30
yg-nec*-95-5320-7
yg-nec*-Singleton-29
Yg-str*-Mu

yellow stripe leaf
gs*-68-1354
ys*-1479
ys*-5-8(5575)
ys*-67-2403
ys*-68-1354
ys*-8912
ys*-N139B
ys*-N326A
ys*-N71B
ys*-PI228180
ys*-PI262172
ys*-PI262475
ys*-PI267219
ys*-whorled

zebra necrotic leaf
zn*-8637
zn*-BYD
zn*-N230B
zn*-N342A
zn*-N354A
zn*-N372A
zn*-N451
zn*-N571D

zebra striped seedling
zb*-89-3137-5
zb-gl*-2187

V. COMMUNITY IBM (cIBM2004) MAPS

Map score data for the community IBM maps were supplied by various cooperators (Table 1) and are based on the IBM-94 panel of 94 stocks. These lines are a subset of the high resolution IBM-302 mapping population used to anchor the physical map (Cone et al., Plant Physiol 130:1686-1696, 2002). The IBM recombinant inbreds were developed from the Syn 5 generation, following 4 intermatings of B73xMo17 (Lee, M et al., Plant Mol Biol 48:453-461, 2002). Seed for the IBM-94 (Table 2) is distributed by the Maize Genetics Cooperation Stock Center <http://maizegdb.org/cgi-bin/stockcatalog.cgi?id=1>. DNA samples of the IBM-94 and the parent lines, along with protocols, are distributed at the University of Missouri-Columbia (<http://www.maizemap.org/dna_kits.htm>. Map score data submitted over the web <http://www.maizemap.org/CIMDE/cimde.html > will return map results within a week after submission; loci are placed based on a framework of 250 loci, also previously placed on the high resolution IBM maps. Each new marker is assigned to chromosome and mapped on the individual chromosomes using the build and place commands of Mapmaker.

With permission of individual cooperators, the community map will be published annually to MaizeGDB. Loci submitted to MaizeGDB are vetted, in consultation with the contributors, for nomenclature, and probe details, including relationships of probes to physical map probes. The first community map was provided to MaizeGDB Dec 2003 and is called cIBM2003. Information about associations to the physical map are additionally provided to Cari Soderland via the IBM neighbors (Polacco, M et al., Plant and Animal Genomes XII Conference, 2004, Maize Workshop < http://63.141.253.172/12/abstracts/W34_PAG12_159.html> for inclusion in the maize FPC product <http://www.genome.arizona.edu/fpc/maize/> and to the Maize Mapping Project iMap representation <http://www.maizemap.org/imap.htm>.

Table 1. Cooperators providing raw map data include:

Ivan Acosta, Yale University, New Haven, CT: *hlm2, tip1*

Hank Bass, University of Georgia, Athens, GA: *fsu1b(smh2), smh1.*

Tom Brutnell, Cornell University, Ithaca, NY <http://bti.cornell.edu/Brutnell_lab2/Projects/Tagging/BMGG_pro_tagging.html>: *bti00191a::Ac, bti00191b::Ac, bti03525::Ac, bti03526::Ac, bti03545::Ac, bti03557::Ac, bti03616::Ac, bti03702::Ac, bti03811::Ac, bti31132a::Ac, bti31132b::Ac, bti31192::Ac, bti99224::Ac, btilc175::Ac, mon00004::Ac, mon00030::Ac, mon00038::Ac, mon00044::Ac, mon00060::Ac, mon00072::Ac, mon00084::Ac, mon00088::Ac, mon00092::Ac, mon00106::Ac, mon00108::Ac, mon00122::Ac, mon00128::Ac, mon00150::Ac, mon00152::Ac, mon00178::Ac, mon00186::Ac, mon00192::Ac, mon00200::Ac, mon00218::Ac, mon00236::Ac, mon03068::Ac, mon03077::Ac, mon03078::Ac, mon03080::Ac.*

ChromDB <http://www.chromdb.org>, University of Arizona, Tucson, AZ. Chromatin RFLP: *ago108, bnlg182, brd101, brd102, brd103, chb101a, chb101b, chb102, chc101a, chc101b, chr106a, chr106b, chr109a, chr109b, chr110a, chr110b, chr111, chr112a, chr112b, chr113, chr116a, chr116b, chr117a, chr117b, chr117c, chr117d, chr118, chr118, chr119, chr120, chr122, chr124, chr125a, chr125b, chr126a, chr126b, crd101, dmt101, dmt102a, dmt102b, dmt103a, dmt103b, dmt103c, epl101, fie2, gta105, gta106b, gta107a, gta107b, gtb101, gtc101, gtc102, gtd101, gte101, gte102, hac101a, hac101b, haf101, hag101, hag102, hag103a, hag103b, hag105, hda102, hda108, hda109, hda110, hdt102, hdt103, hdt105, hmga101, hon104a, hon104b, hon105, hon106a, hon106b, hon107a, hon107b, hon110, hxa102a, hxa102b, mbd101a, mbd101b, mbd105, mbd106, mbd108, mbd109, mbd116, mbd119, mbd121, mez1, nfa101, nfa102a, nfa102b, nfa104, nfc101, nfc103a, nfc103b, nfc104a, nfc104b, nfc104c, nfc105, nfd101a, nfd101b, nfd102, nfd104a, nfd104c, nfd104d, nfd104e, nfd105, nfd106, nfd107, nfd108, nfd109, nfd110, nfd114, nfe101, sdg101, sdg102a, sdg102b, sdg102c, sdg104, sdg105a, sdg106, sdg107, sdg108a, sdg108b, sdg110, sdg111a, sdg113, sdg115, sdg116a, sdg116b, sdg117a, sdg117b, sdg118, sdg119, sdg123, sdg129, sgb101, sgb103, sgf101, smh4, vef101a, vef101b.*

Genoplante, France <http://genoplante-info.infobiogen.fr>. RFLP related to Overgos on the physical map: *gpm1, gpm10, gpm11, gpm12, gpm13, gpm14, gpm15, gpm16, gpm2, gpm3, gpm4, gpm5, gpm6, gpm7, gpm8, gpm9*

Peter Rogowsky, EDP ENS-Lyon, Lyon France: *ensl015, ensl016.*

Mike McMullen, USDA-ARS Columbia, MO. SNPs related to Overgos on the Physical map: *AI714808, AI737325, AI861369, AW036917, AW172071, AW231791, AW258116, AY103622, AY103942, AY103944, AY104188, AY104234, AY104686, AY105043, AY105132, AY105205, AY105451, AY105479, AY105872, AY106026, AY106137, AY106170, AY106313, AY106318, AY106439, AY106518, AY106592, AY106674, AY106825, AY107012, AY107018, AY107034, AY107053, AY107079, AY107121, AY107128, AY107193, AY107200, AY107292, AY107329, AY107414, AY107489, AY107496, AY107517, AY107629, AY107682, AY107743, AY107844, AY107881, AY107910, AY107911, AY108545, AY108825, AY108844, AY109061, AY109096, AY109128, AY109538, AY109804, AY110113, AY110240, AY110782, AY110827, AY110835, AY110853, AY111089, AY111254, AY111333, AY111822, AY111877, AY111962, AY112119, AY112175, AY112199, AY112283, AY112355, BE518809, BG266188*

Sahtoh Namiko, Cold Spring Harbor Laboratory, Cold Spring Harbor, NY: *shpl1.*

Wolfgang Werr, Roman Zimmerman, Institut für Entwicklungsbiologie,Universtaat Köln, Köln, Germany: *nact11, nact12, nact15, nact16a, ns1*

Table 2. The IBM-94 panel includes the below lines:

MO001 MO005 MO007 MO008 MO010 MO012 MO014 MO015 MO016 MO017 MO021 MO022 MO023 MO024 MO025 MO027 MO028 MO029 MO030 MO031 MO032 MO033 MO034 MO035 MO039 MO040 MO043 MO044 MO045 MO046 MO048 MO051 MO052 MO054 MO055 MO057 MO058 MO060 MO061 MO062 MO063 NO066 MO067 MO074 MO075 MO076 MO077 MO079 MO262 MO264 MO265 MO266 MO267 MO269 MO272 MO275 MO276 MO281 MO284 MO287 MO288 MO296 MO297 MO298 MO309 MO310 MO311 MO315

MO317 MO321 MO322 MO323 MO325 MO326 MO328 MO337 MO341 MO344 MO345 MO346 MO352 MO354 MO355 MO357 MO360 MO364 MO365 MO368 MO369 MO378 MO379 MO382 MO383 MO384
These line numbers are identical to the corresponding line in the larger panel of stocks, IBM 302, where seed is also maintained at the Stock Center.

Acknowledgements. This report would not have been possible without the considerable contributions of Karen Cone, Mike McMullen, Georgia Davis, Seth Haverman and Hector Sanchez-Villeda towards the design and implementation of the CIMDE (Community IBM map data entry) software package (Sanchez-Villeda et al., Bioinformatics 19:2022-2030, 2003) and the encouragement of Ed Coe, PI of the Maize Mapping Project, NSF DBI 9872655, which funded the software resources used for these maps.

Maps Legend.
Bin edges are indicated to the left of each chromosome; asterisks indicate a core marker. Positions of off-frame loci are indicated as an interval value, in parentheses, and refer to the interval 'down' from the framework locus to their immediate left. Note, due to the inter-matings in this population, these map coordinates are not centiMorgan (cM) units as defined in the classical sense.

Submitted by
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USDA-ARS
University of Missouri

cIBM2004 CHROMOSOME 1

BIN	Position	Locus	Locus Off Frame
1.00	0	***umc1354***	
1.01	2.3	***tub1***	
	6.9	***dmt103b***	
	10.5	***umc1566***	*(17.4)mon03077::Ac*
	44.2	***lim179***	*(7.7)AY107629*
	68.1	***umc1977***	
	80.4	***AY110853***	
1.02	90.5	***umc157a***	
	107.3	***csu1171***	
	134.6	***bnlg1953***	*(2.3)mon03080::Ac*
	180.6	***mon00192::Ac, mon00106::Ac***	
1.03	180.7	***umc76a***	
	185.9	***umc1403***	
	234.1	***AY106592***	
	243.0	***asg35b***	
	254.0	***AY110240***	
	257.1	***AY107489***	*(4.8)bnlg182*
	262.5	***umc1598***	
	274.5	***mmp100***	
1.04*	295.7	***umc1169***	*(0.6)chr125b*
	310.2	***bnl9.11b***	*(3.1)AY103942*
1.05	324.6	***csu3***	*(5.5)btilc175::Ac*
	339.0	***umc1515***	
	342.1	***nfd104c***	
	346.1	***AY106439***	
	347.2	***AY107682***	
	358.7	***hac101b***	
1.06*	367.4	***mbd106***	
	369.8	***umc1972***	*(1.1)gpm4*
	385.5	***mbd119***	
	392.3	***asg58***	
	404.1	***nact15***	
	427.2	***ntf1***	
	448.5	***umc192***	*(56.1)mon00186::Ac*
1.07	449.1	***asg62***	
	510.4	***bnlg1556***	
	520.3	***dmt103c***	
	537.9	***hon105***	*(8.7)bti03557::Ac*
	547.4	***bnlg1025***	
1.08	561.1	***umc128***	*(0.6)AY105132*
	568.2	***bnlg2228***	
	570.7	***chr124***	
	574.3	**bti03545::Ac**	
	577.4	***sdg123***	
	586.5	***umc1991***	
	606.2	***nfc103a***	
1.09	611.1	***cdj2***	*(3.7)AY106137*

	625.8	***umc2047***	
	639.7	***AY112283***	
1.10	644.4	***umc107a***	
	650.8	***chb101b, chr106a***	
	650.9	***nfa103a***	
	656.2	***AY104234***	
	660.6	***hxa102b***	
	662.5	***vef101b***	
1.11*	671.6	***lim39***	*(21.7)mon00200::Ac*
	696.7	***umc1421***	*(2.7)hon110*
	714.2	***AY109096***	
	727.0	***AY106825, umc1630***	
	730.6	***AY112175***	
	741.5	***AY109128***	
	772.3	***phi064***	
1.12	790.7	***bnl6.32***	
	792.6	***umc1605***	
	801.7	***AY104686***	

cIBM2004 CHROMOSOME 2

BIN	Position	Locus	Locus Off Frame
2.00	0	***isu53a***	
2.01*	22	***umc1165, bti00191a::Ac***	*(42.1)sgb101*
2.02*	64.7	***umc1824a***	
	78.4	***dmt102b***	
	84.2	***eks1***	*(22.0)gpm7*
2.03	110.1	***umc6a***	
	128.3	***nfd102***	
	139.4	***mmp33***	
	152.2	***sdg104***	
	157.1	***AY107034***	
	159.8	***sdg107***	
2.04*	161.3	***phi109642***	
	169.2	***AY103944***	
	172	***umc1326***	
	174.3	***hag103a***	
	192.1	***AI714808***	*(4.9)bti03811*
	197.4	***umc2030***	*(1.0)sdg102a*
	200	***hda102***	*(0.9)AY112119*
2.05	206.1	***umc131***	
	207.4	***zpu1***	*(0.4)AY107012*
	212.6	***ns1***	
	214.6	***AY111877***	
2.06*	217.3	***pbf1***	
	226.9	***umc1080***	*(0.5)sdg119*
2.07*	238.9	***umc2129***	*(3.1)AI861369*
	244	***umc1890***	
	255.7	***nfc104b***	
	259.4	***sdg106, nfd101b***	*(11.7)chr119,(9.7)sdg116b*
	271.8	***umc1560***	
2.08	274.7	***asg20***	
	286.8	***mon00084::Ac***	
	296.4	***hda109, chr122***	
	303.1	***umc1604***	
	308	***hag105***	
	312.4	***chc101b***	
	326	***mmc0381***	
	336.2	***gpm16***	
2.09	346	***umc49a***	
	355.3	***umc1252***	
	380.9	***BG266188***	
	401.4	***bnlg469b***	
2.10*	437.1	***AY106674***	
	446.7	***lim104***	*(0.9)chr106b*
	452.4	***umc1696***	

cIBM2004 CHROMOSOME 3

BIN	Position	Locus	Locus Off Frame
3.01*	0	***umc1931***	
	4.4	***bnl8.15***	*()AY106313*
	17.9	***AY112199***	
	20.5	***umc2049***	
3.02	40.1	***csu32a***	
	58.7	***umc1886***	
3.03	86.1	***asg24a***	
	104.7	***lim66***	
3.04	124.5	***asg48***	
	137.4	***umc1608***	
	147.7	***nfc104c***	*(2.3)haf101,(12.6)AY107193*
	160.9	***mmc0132***	*(3.8)chr126b, (3.4)bti03702::Ac*
	169.7	***umc1449***	*(4.8)mbd105,(4.6)chr110a,(4.5)AY111333*
	176.3	***hac101a***	
	179.9	***AY110827, gpm14***	
3.05	184.4	***umc102***	*(7.6)bti03526::Ac*
	190.7	***chr109b, umc1102***	*(35.1)mon00178::Ac,(0.5)nact16a*
	227.9	***csu636***	
3.06*	236.5	***umc1539***	
	249.9	***asg39***	
	251.9	***AY106026***	
	309.4	***csu1183***	
	314.4	***sdg113***	
	320.4	**bti31083::Ac**	
	330.9	***asg7b***	
3.07	333.2	***bnl6.16a***	
	348.2	***AY107018***	
	369.4	***umc1404***	
	377	***sdg117a***	
	378.8	***nfc101, nfc105***	
	380.6	***hon108***	
	382.3	***AY106518***	
3.08	387.2	***umc17a***	
	389	***AW258116***	
	403.5	***gpm3***	
	408.7	***mmc0251***	*(12.7)sdg115*
	424	***umc1273***	
3.09	428.6	***umc63a***	
	436.7	***csu845***	*(13.5)AY111254*
	476.5	***lim182***	
	491.1	***bnlg1754, fsu1b***	
	527.2	***umc1641***	
	532.4	**bti03616::Ac**	
	538.3	***umc1594***	

cIBM2004 CHROMOSOME 4

BIN	Position	Locus	Locus Off Frame
4.01*	0	***msf1***	
	20.8	***bx4***	
	43.1	***umc1759***	
4.02	45.6	***php20725a***	
	67.4	***umc1943***	
4.03	104.8	***umc31a***	*(1.5)chr117c*
	116.6	***umc1926***	
	147.6	***umc2039***	
	212.5	***bnl8.45c***	
4.04*	280	***bnlg490***	
4.05*	286.1	***sdg108a***	
	292.1	***AY107128***	
	301.5	***csu509***	
	307.9	***chr112a, hda108***	
	310.8	***umc1511***	
	313.1	***nfa104***	
	314.8	***nfd104e***	*(0.5)AY105043*
4.06*	321	***umc1945***	
	335.7	***nfd105***	
	344	***mpik3***	
4.07	371.4	***bti31094::Ac, umc66a***	
	377.2	***umc2038***	
	380.4	***mon00128::Ac***	
	382.9	***mon03078::Ac***	
	387.1	***bnl5.24b***	
	397.4	**bti00207::Ac**	
4.08*	398.7	***umc1775***	
	409.4	***umc1808***	
	448.9	***ufg23***	*(13.7)nfd106, (12.8)bti00245::Ac*
	463.1	***umc1842***	*(1.0)mbd121, (1.9)mon00150::Ac*
4.09	468.2	***umc52***	*(6.5)AY111962*
	475.9	***umc1999***	*(2.1)nfd107*
	492.4	***AY107200***	*(7.4)chb102*
	501.4	***mbd116***	*(14.5)bti31192::Ac*
	517.6	***sbp2***	*(4.4)AY107910*
4.10	530.4	***php20608a***	
	547.8	***umc1109***	*(15.2)AY111822*
	563.6	***hon106b***	
	574.2	***bip2***	
	577.9	***umc1707***	

cIBM2004 CHROMOSOME 5

BIN	Position	Locus	Locus Off Frame
5.00	0	***umc1253***	
	21.4	***tip1, umc1260***	
5.01	23.2	***npi409***	
	31.5	***lim407***	
	61.2	***umc2036***	*(4.0)hxa102a*
5.02*	85.9	***asg73***	
	90.5	***chb101a***	
	94	***umc1587***	*(23.4)ago108,(23.3)AY110835*
5.03*	119	***bnlg1879***	
	133	***rz474a***	*(1.2)mbd109*
	138.3	***nfc103b***	
	150	***umc2035***	
	158.8	***hag101***	
	165.7	***crd101***	
	168.8	***gpm5***	
	173	***umc1609***	
	178.3	***AY107414***	
	182.2	***shpl1***	
	187.1	***sdg129, AY107844***	
	187.2	***gtc101, gtc102***	
	188.3	***hon106a***	
	190.7	***bnlg1902***	
5.04*	199.3	***mon00030::Ac***	*(2.8)nfd108*
	203.7	***umc1990***	
	218.4	***AY105205***	
	225.7	***umc1349***	
	233.3	***mon00152::Ac***	
5.05*	242.3	***umc1482***	
	252.7	***nfe101***	*(15.0)AY107329,(13.5)AY111089*
	269.7	***nbp35***	*(0.6)gte102*
	281.8	***mon00044::Ac***	
5.06	290	***umc126a***	
	299.7	***nfd104a***	
	316	***bnlg609***	
	326.7	***sdg117b***	
5.07	347.1	***umc108***	
	397.9	***bnlg1118***	
	412.3	***bnlg118***	
5.08*	443.5	***umc1225***	
	445.2	***mmp175***	
	454.4	***nfd109***	
5.09	463.3	***php10017***	

cIBM2004 CHROMOSOME 6

BIN	Position	Locus	Locus Off Frame
6.00	0	***umc1143***	
6.01	19.9	***hon104b, umc85a***	*sdg102b*
	27.1	***bnlg1867***	*(1.5)gpm8*
	31.1	***nfa101***	*(0.6)mez1,(0.5)mon00038::Ac*
	32.3	***AY107121***	
6.02*	36.1	***umc1006***	
	40.7	***sdg102c***	
	49.5	***csu923***	
6.04	61	***umc65a***	
	75.6	***umc1857***	*(1.5)nact11,(1.2)AY108825,(1.8)AY109804*
	78.5	***sgf101***	
	85.6	***gta107b, gta105***	
	90.5	***bti31132a::Ac, bti31132b::Ac***	
	103.3	***umc2006***	*(7.8)AY107053*
6.05*	111.7	***isu111a***	
	114.8	***AY105479***	*(0.7)AY107517*
	116.9	***chr117d***	
	121.4	***uaz280c***	
	130.6	***umc1388***	
	143.6	***pmg1***	*(1.5)mbd101b*
	152.4	***chr116a, AY107881***	
	169.6	***AW036917***	
	192.5	***uaz121a***	
	199.4	***gpm9***	
6.06	213.3	***umc38a***	
	216.3	***umc1762***	
	218	***sdg111a, gtb101***	
	231.3	***lim379***	
	236.6	***lim151***	*(0.9)gte101*
6.07	257	***umc132a***	*(0.7)hdt103*
	260.2	***nfa102a***	
	265.5	***mlg3***	
	309.1	***umc1350***	*(35.3)chr118,(35.3)chr121*
6.08*	346.2	***umc2059***	

cIBM2004 CHROMOSOME 7

BIN	Position	Locus	Locus Off Frame
7.00	0	***csu582***	
	42.8	***umc1378***	*(1.2)gpm12*
	63.9	***umc1672***	
7.01	80.8	***asg8***	
	91.8	***php20581a***	
	102.8	***hda110***	
	122	***dmt101***	
	123.7	***o2, hon102***	
7.02	124.3	***asg34a***	
	142.5	***sdg101***	
	150.4	***cyp6***	*(0.6)AY109061*
	150.9	***AY106170***	
	152	***epl101, vef101a***	
	153.1	***hag102, sdg110***	
	169.2	***umc1983***	
	185.5	***mon03068::Ac***	
	194.6	***bnlg1808***	
7.03*	196.9	***mmp127***	
	233	***bnlg1070***	
	241.8	***mbd108***	
	243	***brd103***	*(1.6)bti31211::Ac*
	245.4	***sdg116a***	
	247.7	***mon00072::Ac***	
	254.9	***umc56***	
	261.3	***brd101, nfd101a***	*(3.2)AY107911*
	279.8	***tif1***	
7.04*	290.2	***umc1710***	
	313.2	***bnlg1666***	
	320.8	***chr111***	
	342.9	***mon00088::Ac***	
	345.7	***umc1708***	
	355.6	***AY108844***	
	368.5	***mon00060::Ac***	
	381	***umc1412***	
	389	***gpm2***	
7.05	395.9	***umc245***	*(7.0)chc101a*
	405.4	***phi069***	*(6.0)AY106318*
	449.3	***umc1406***	
7.06	455.3	***umc168***	
	459.7	***nfc104a***	

cIBM2004 CHROMOSOME 8

BIN	Position	Locus	Locus Off Frame
8.01	0	***npi220a***	
	9.3	***csu319***	
	35	***umc1592***	
	57.9	***umc1327***	
	76.4	***mmp85***	
8.02*	82	***hon107b***	
	83.9	***gpm11***	
	86.6	***cdo460***	
	107.3	***mmp166***	
	111.1	***chr117a***	
	126.8	***AI737325***	
	143.8	***nfd110, nfd114***	
	143.9	***umc124a***	
	145.6	***umc1530***	
	166.5	***gpm10, AY110113***	
8.03	166.6	***chr110b, chr126a***	
	169.4	**bti03525::Ac**	
	174.5	***umc1910***	
	180.4	***AW172071***	
	197.9	***umc1457***	
	201.7	***AY108545, mbd101a***	
8.04*	204.1	***AW231791, AY107079***	
	213.8	***sdg105a***	
8.05*	216.1	***umc1130***	
	229.6	***chr117b***	*(6.1)nfa102b*
	237.5	***umc1889***	*(0.6)hdt102*
	242.2	***chr112b***	
	242.8	***hon107a***	
	243.4	***fsu1a***	
8.06*	264.4	***sdg118, umc1149***	
	287.2	***hdt105***	
	294.4	***chr116b***	
	300.1	***bnlg1031***	*(18.8)mon00108::Ac*
8.07*	328.3	***umc1268***	
8.08	335.2	***npi414a***	
	364.8	***umc1933***	*(20.2)mon00122::Ac*
8.09	395.1	***agrr21***	
	469.4	***bnlg1131***	

cIBM2004 CHROMOSOME 9

BIN	Position	Locus	Locus Off Frame
9.01	0	***umc109***	*(9.6)AY112355*
	47.4	***lim343***	
	50.6	***ufg41***	
	54.8	***gta106b, gta107a***	
	69.5	***umc1967***	
	71.2	***AY107496***	
9.02	73.5	***bz1***	
	90.6	***chr113***	
	93.5	***csu471***	
	99	***AY103622***	
	118.4	***bnlg1401***	
	149.5	***AY105451***	
9.03	159	***wx1***	
	160.9	***mon00236***	
	162.8	***umc1258***	*(1.1)hon104a*
	167.9	***gpm6***	
	174	***chr120***	
	175.3	***nact12***	*(5.7)gtd101,(5.7)ensl015,(5.7)ensl016,(5.8)*
	181.9	***umc1271***	*AY107743,(5.7)BE518809*
	189.9	***rz682***	
9.04*	196.6	***bnlg1209***	
	208.2	***AY110782***	
	209.4	***umc1120***	
9.05	215.7	***umc95***	
	218.6	***chr125a***	
	225.7	***mmp151d***	
	248.2	***csu634***	
	253.7	***ufg24***	
9.06*	269	***gpm1, mmp142***	
	293.5	***AY107292***	
	305.8	***mmp131***	*(4.9)AY104188*
	313.7	***mmp168***	*(3.4)nfd104d,(19.2)mon00004*
9.07	333.5	***asg12***	
	346.3	***bnlg619***	
	359	***brd102, mon00092***	
	378.5	***umc1137***	*(25.8)dmt103a*
	408.6	***umc1505***	

cIBM2004 CHROMOSOME 10

BIN	Position	Locus	Locus Off Frame
10.00	0	***mmp48a***	
	10	***php20753a***	
10.01	14	***php20075a***	
	39.5	***umc2018***	*(2.1)bti99224::Ac*
10.02	48.5	***npi285a***	
	76.1	***umc2069***	
10.03	80.8	***umc130***	*(8.9)chr109a,(4.5)AY105872*
	90.2	***sdg108b***	
	92.9	***umc1345***	
	97.7	***hlm2***	
	100.8	***gpm13, fie2***	
10.04	102.5	***umc64a***	
	108.1	***umc1995***	
	116.1	***AY109538***	
	125.6	***umc1330***	
	131.6	***hag103b***	
10.05	136.3	***umc259a***	
	148	***umc1506***	*(28.1)gpm15*
10.06*	177.3	***bnl10.13a***	
	187.9	***bnl17.02***	
	219.4	***umc1993***	*(5.6)dmt102a*
10.07	245.2	***bnl7.49a***	
	255	***bnlg1677***	*(0.4)sgb103*
	311.2	***bnlg1450***	*(33.7)mon00218::Ac*
	315.8	***bti00191b::Ac***	*(0.4)sgb103*
	341.1	***asg19b***	*(33.7)mon00218::Ac*

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VIII. AUTHOR INDEX
(* identifies articles authored in this Newsletter)

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Cooperators (that means you) need the Stock Center.
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(1) Send stocks of new factors you report in this Newsletter or in publications, and stocks of new combinations, to the collection.

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MaizeGDB needs Cooperators (this means you) to:

(1) Look up "your favorite gene or expression" in **MaizeGDB** and send refinements and updates to via the public annotation "button" at http://www.maizegdb.org.

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