the second meiotic division takes place; the chromatids are randomly distributed at AII, giving rise to 100% anomalous tetrads. Mutant plants are completely male- and female-sterile.

This meiotic mutation is controlled by a single recessive gene (the segregation in M_3-M_4 plants heterozygous for this mutation was 64 normal:15 mutant plants, χ^2 = 1.52, P = 0.1 - 0.15).

This mutation causing meiotic sterility was designated "the absence of the first division," and its symbol is afd-W23 (W23 is line Wisconsin 23 where this mutation first appeared). This new type of meiotic mutation has not been described in the literature. Meiotic mutants in plant and animal species are listed in a reference (Ontogenesis 6: 2, 1975). However, the first division was experimentally substituted by the second division by Astaurov (Cytogenetica razhvitiya tutovogo shelkopryada, M. "Nauka," 1968) in Bombyx mori and by Maguire (Chromosoma 48:2, 1974) in Zea mays L. The mechanisms of the substitutions are different from the mechanism we describe for afd-W23.

This type of meiotic mutation might have been involved in the course of evolution of apomictic plants as a cytogenetic mechanism underlying the gradual transition from meiosis to mitosis.

I.N. Golubovskaya

INSTITUTO DE GENETICA
Facultad de Agronomia, Maracay, Venezuela

Location of gl12 — A-B chromosome translocations were used to locate gl12 in a mutant stock received from the Maize Genetics Coop.

The data from the F_1 progenies presented in Table 1 clearly indicate the long arm of chromosome 3 as the carrier of the gl12 locus. After this result we looked to relate gl12 with Rg, lg2 and ra2.

Table 1. Results of the F_1 from gl12 gl12 x the A-B translocations.

Translocations	Breakpoint	Gl12	gl12	Total
B-1a	1L.2	37	0	37
B-1b	1S.05	169	0	169
B-3a	3L.1	88	15	103
B-4a	4S.25	232	0	232
B-7b	7L.3	62	0	62
B-8a	8L.7	119	0	119
B-9a	9L.5	121	0	121
B-9b	9S.4	146	0	146
B-10a	10L.35	59	0	59

Table 2. Backcross segregation: Gl12 gl12 Rg rg x gl12 rg.

Gl12 Rg	Gl12 rg	gl12 Rg	gl12 rg	Total
84	12	3	116	215

$$\text{Recombination} = \frac{15}{215} = 0.07.$$

Table 3. F_2 segregation: Gl12 gl12 Lg2 lg2 selfed.

F_2 families	a_1 Gl12 Lg2	a_2 Gl12 lg2	a_3 gl12 Lg2	a_4 gl12 lg2
73.1049	67	31	20	1
73.1050	65	22	20	1
Total	132	53	40	2

$$c = \frac{a_1 \times a_4}{a_2 \times a_3} = \frac{264}{2120} = 0.1245;\ \text{recombination fraction} = 0.23.$$

Table 4. F_2 segregation: Gl12 gl12 Ra2 ra2 selfed.

F_2 family	Gl12 Ra2	gl12 Ra2	Gl12 ra2	gl12 ra2	Total
73.1051	63	23	18	1	105

$$c = \frac{a_1 \times a_4}{a_2 \times a_3} = 0.1521;\ \text{recombination fraction} = 0.25.$$

Livia B. de Lares and Dora M. de Zerpa

Location of gl14 — Homozygous gl14 from the Maize Genetics Coop was crossed with A-B and reciprocal interchanges. The F_1 and testcross data are presented in Tables 1 and 2.

Table 1. F_1 progenies (gl14 x A-B translocations).

Translocation	Breakpoint	Gl14	gl14
B-1b	1L.05	226	0
B-3a	3L.01	115	0
B-4a	4S.25	56	0
B-7b	7L.3	24	0
B-8a	8L.7	28	0
B-9a	9L.5	77	0
B-9b	9S.4	9	0
B-10a	10L.35	124	0

Table 2. Backcross segregations: (A-translocations x gl14) x gl14.

Reciprocal translocation	Breakpoint	Gl gl Tt*	Gl gl tt	gl gl Tt	gl gl tt
wx 1-9c	1S.48; 9L.22	1	3	12	7
wx 1-9(8389)	1L.74; 9L.13	21	12	4	3
wx 2-9b	2S.18; 9L.22	37	17	2	22
2-9c	2S.49; 9S.33	26	17	1	7
		53	36	21	39
wx 3-9a	3L.11; 9L.16	2	3	0	0
wx 3-9b	3L.48; 9L.53	6	5	5	6
wx 3-9c	3L.09; 9L.12	6	9	5	14
3-9e	3L.02; 9L.29	17	9	6	13
wx 4-9b	4L.90; 9L.29	14	15	2	6
wx 4-9g	4S.27; 9L.27	3	2	3	2
wx 4-9(5657)	4L.33; 9S.25	15	14	17	13
wx 5-9a	5L.69; 9S.17	11	18	9	3
wx 5-9c	5S.07; 9L.10	3	3	3	2
wx 6-9a	6S.79; 9L.40	7	5	3	7
wx 6-9b	6S.10; 9S.37	17	8	15	10
wx 7-9a	7L.63; 9S.07	9	18	23	18
7-9c	7L.14; 9L.22	26	22	13	10
wx 7-9(4363)	7 cent; 9 cent.	11	8	25	18
wx 8-9(6673)	8L.35; 9S.31	8	11	20	16

*Tt = heterozygous translocation, semisterile; tt = homozygous for normal chromosomes, fertile.

Table 3. Backcross progenies: (gl14 x wx 2-9b) x gl14; (gl14 x 2-9c) x gl14; (gl14 x 2-9d) x gl14.

Translocation	Breakpoint	Gl gl Tt*	Gl gl tt	gl gl Tt	gl gl tt
wx 2-9b	2S.18; 9L.22	37	17	2	22
		10	5	5	6
		19	1	17	42
		48	12	4	25
		28	4	3	19
2-9c	2S.49; 9S.33	26	17	1	7
		27	13	11	11
		26	23	10	28
		26	19	13	25
		8	7	3	8
2-9d	2L.83; 9L.27	11	17	9	32
		18	14	19	36

*Tt = heterozygous translocation, semisterile; tt = homozygous for normal chromosomes, fertile.

The data indicated linkage between gl14 and T wx 2-9b and 2-9c, both with break points on short arm of chromosome 2; consequently, additional backcross progenies (Table 3 on previous page) were raised involving the named interchanges plus T2-9d (2L.83; 9L.27).

We found the classification of gl14 to be difficult and not always satisfactory because of the frequent doubtful expression of the character. The detection of linkage and the χ^2 partition of its components showed that Gl14-gl14 segregation constituted an important source of deviation (Tables 4 and 6). Notwithstanding, backcross progenies involving T wx 2-9b showed significant deviation from independent inheritance due to joint segregation (Table 4).

Table 4. Detection of linkage between gl14 and T wx 2-9b: analysis of χ^2 by orthogonal functions.*

	Gl14 gl14 Tt	Gl14 gl14 tt	gl14 gl14 Tt	gl14 gl14 tt	Total
Observed	142 (a_1)	39 (a_2)	31 (a_3)	114 (a_4)	326
Expected (with no linkage)	81.5	81.5	81.5	81.5	326

Segregation for Gl14-gl14:

	χ^2	DF	Probability
$\chi^2_{gl} = \frac{(a_1 + a_2 - a_3 - a_4)^2}{n} = \frac{(36)^2}{326} = \frac{1296}{326} =$	3.97	1	0.05- 0.02

Segregation for Tt:

	χ^2	DF	Probability
$\chi^2_T = \frac{(a_1 - a_2 + a_3 - a_4)^2}{n} = \frac{(20)^2}{326} = \frac{400}{326} =$	1.22	1	0.30 - 0.20

Joint segregation:

	χ^2	DF	Probability
$\chi^2_L = \frac{(a_1 - a_2 - a_3 + a_4)^2}{n} = \frac{(186)^2}{326} = \frac{34596}{326} =$	106.12	1	Extremely small
Total	111.31	3	Extremely small

*Mather K., 1938. The measurement of linkage in heredity. London: Methuen & Co. Ltd.

The linkage relationship between gl14 and the wx 2-9b breakpoint indicated by the backcross data in Table 4 was confirmed by information obtained from F_2 families (Tables 5 and 6). Classification of F_2 kernels for waxy and normal endosperm corresponds with the separation of TT from tt; both genotypes are fully fertile, but TT can be identified by its association with waxy endosperm. In this way we were able to ascribe a genotype to each class of the F_2 (Table 5) and detect linkage (Table 6).

Table 5. Families of the F_2: wx 2-9b x gl14 selfed.

Classes		73-1013	73-1014	73-1015	Total
	TT*	17	14	0	
Gl14	Tt**	48	46	49	180 (a_1)
	tt***	11	9	1	21 (a_2)
	TT*	5	6	7	
gl14	Tt**	10	11	10	49 (a_3)
	tt***	27	12	22	61 (a_4)

*waxy endosperm and fully fertile
**normal endosperm and semisterile
***normal endosperm and fertile

Table 6. Detection of linkage from the F_2: gl14 x wx 2-9b selfed and X^2 analysis.

	a_1	a_2	a_3	a_4	Total
Observed	180	21	49	61	311
Expected (with no linkage)	174.935	58.315	58.315	19.435	311

$$X^2_{gl} = \frac{(a_1 + a_2 - 3a_3 - 3a_4)^2}{3n} \qquad X^2_T = \frac{(a_1 - 3a_2 + a_3 - 3a_4)^2}{3n}$$

$$X^2_L = \frac{(a_1 - 3a_2 - 3a_3 + 9a_4)^2}{9n}$$

	X^2	DF	Probability
Segregation for Gl14 gl14	17.83	1	0.01
Segregation for Tt	0.30	1	0.70 - 0.50
Joint segregation	96.23	1	Extremely small

Backcross and F_2 data and analyses indicate that gl14 is located on chromosome 2, probably on the short arm .

Dora M. de Zerpa and Livia B. de Lares

INSTITUTO FITOTÉCNICO DE SANTA CATALINA
Llavallol, F.N.G.R., Argentina

Hereditable character conditioned by nuclear units and genes that do not respond to mendelian inheritance — One multiple dominant line, A C R Pr B Pl, derived from Doctor Randolph's pedigree 1877 (year 1933), was maintained by inbreeding in the Instituto Fitotécnico de Santa Catalina. This subline was considered in 1953 as inductive of mutations (Mazoti, Caryologia VI, Suppl. pp. 709-715, 1954) It gave origin, by crossing, to the dominant inhibitor of aleurone color C-I-7001 (referred to in MNL 40:62 as C^{IP}) which is an allele of gene C, at locus 26 of chromosome 9. The "multiple dominant" subline produced a new dominant inhibitor mutant of aleurone color located near the gene wx at an approximate distance of 26 units from C-I-7001. The presence of a dominant activator Ac* and a recessive li* is necessary for the gene, C*-IE-7002, to manifest its dominant inhibitor action of the aleurone color; for this reason I think that this gene has originated from the transposition of the structural genes of a possible operon C to a contiguous place of a foreign operator.

In Table 1, (published in MNL 41:88) we find that the data manifest the relation of 0.685 colorless aleurone, 0.315 aleurone color, for repulsion association of two inhibitor dominant genes of aleurone color, C-I-7001 and C*-IE-7002 at a distance of approximately 26% of crossing-over, having in backcross the recessive activator gene Li* and having present in all cases the dominant activator Ac*.

The location of gene C*-IE-7002 was done by the cross indicated in Table 2. From the analysis of Table 2 we can judge that in the majority of the classes the deviations are not significant and that the great deviations which manifest themselves in the less frequent classes (0.1% double crossingover) were a mistake in the classification into normal kernels and shrunken endosperm which would greatly modity the value of x^2.

In progenies derived from the same progenitors, both having the same isogenic constitution and the same mendelian relation, 3/4 aleurone color and 1/4 colorless aleurone should be obtained in all progenies; however, here we obtained variable segregations in the ear according to the different areas (1/2 right or 1/2 left of the ear) and in other cases an excess of colorless aleurone kernels.