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

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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 χ^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.

Table 1. C-I-7001 +/C C*-IE-7002, Li* li* X C +/C +, li* li*.

Progeny	Phenotype: Colorless aleurone	Phenotype: Colored aleurone	N-1	χ^2 relation 0.685:0.315	P
1	219	116	1	1.51	ns
2	98	47	1	0.05	ns
3	234	117	1	0.54	ns
4	276	123	1	0.08	ns
5	266	116	1	0.22	ns
6	128	54	1	0.28	ns
7	256	123	1	0.15	ns
8	213	100	1	0.02	ns
9	228	101	1	0.09	ns
10	189	117	1	6.43	<0.01
11	266	109	1	1.02	ns
	Total χ^2		11	10.46	ns
Deviation	2.373	1.121	1	0.55	ns
Heterogeneity			10	9.91	ns

Table 2. C-I-7001 + + +/C sh wx C*-IE-7002, Li* li* X C sh wx, li* li*.

Phenotype			Frequency calculation	Observed	Calculated	P
Colorless	+	+	0.37750	190	212.5	ns
Colorless	sh	wx	0.20575	98	115.63	ns
Color	sh	wx	0.18875	104	106.07	ns
Colorless	+	wx	0.10525	91	59.15	<0.001
Color	sh	+	0.10500	62	59.01	ns
Color	+	+	0.01700	9	9.55	ns
Colorless	sh	+	0.00050	4	0.28	<0.001
Color	+	wx	0.00025	4	0.14	<0.001
				562	561.98	

Tables 3 and 4 show the variable segregation according to the different sectors or areas of the ear in progenies derived from the backcross +/C*-IE-7002, Li* li* X li* li*.

In Table 5 we can observe a great variability in the segregation of colored and colorless kernels.

Table 3. +/C*-IE-7002, Li* li* X +/+, li* li*: Area (1), seven rows of the middle left side of the ear; (2), seven rows of the middle right side.

Progeny		Aleurone Colored	Colorless	Total	Deviation from mendelian segregation χ^2	P
1	I(1)	120	98	218	46.2	<0.001
	D(2)	156	46	202	0.5	ns
		276	144	420	19.3	<0.001

Discrepancy between middle left side and middle right side of the ear $\chi^2 = 22.89$; $P<0.001$.

Table 4. +/C*-IE-7002, Li* li* X +/+, li* li*: Areas as in Table 3.

Progeny	Aleurone Colored	Colorless	Total	Deviation from mendelian segregation χ^2	P
I(1)	128	90	218	30.8	<0.001
D(2)	148	56	204	0.79	ns
	276	146	422		

Discrepancy between middle left side and middle right side of the ear $\chi^2 = 8.91$; $P<0.01$.

Table 5. Backcross +/C*-IE-7002, Li* li* X +/+, li* li*.

Progeny	Phenotype Colored aleurone	Colorless aleurone	Deviation from mendelian segregation χ^2	P
74-7950	192	62	0.04	ns
74-7122	227	150	43.96	<0.001
74-7122	171	155	88.38	<0.001
72-7952	217	117	17.92	<0.001
72-7000	262	178	56.48	<0.001
72-7000	289	137	11.64	<0.001
(a)			218.01	

These variable deviations are attributed to the gene or a chromosomic sector of the locus C*-IE-7002 that produces its active replies with a frequency not synchronized with the chromosomic division; for this reason it would stay free in the cellule, and its distribution during the cellular multiplication would be a matter of hazard, with the activator genes Ac* and li* necessary to manifest its activity. In M.N.L. (40:62-63, 1966; 41:87-91, 1967) the hypothesis based on replies of DNA was suggested in order to interpret this phenomenon; however, at that time we had neither located the gene C*-IE-7002 nor found genes Ac* and li*. For this reason the actual interpretation differs from the previous ones, for the gene C-I-7001 (C^{IP}) would not have had its origin in teosinte, but in the subline derived from Dr. Randolph's dominant multiple pedigree 1877 (1933).

The gene that would produce "replies" would not be the gene C-I-7001, but it could be the gene C*-IE-7002, linked to the preceding one. In order to explain the hypothesis based on replies of DNA it is not essential that the "bits" of DNA be autoduplicable; it is sufficient for it to be produced by a chromosomic sector which corresponds to the gene C*-IE-7002.

The hypothesis that those free units of DNA would explain the contiguous phenomena of apparent paramutation, mosaicism, no mendelian segregations and genetic instability is affirmed in this work.

I think that the locus C could be a "normal" operon (Jacob, F., Science 152: 1470-78, 1966) in which its structural genes are capable of producing an enzyme that would inhibit the aleurone color but are repressed by the normal regulator in an inducible system that does not have the "inducer." The recessive gene c would be the operon C with the regulator genes which are suppressed or absent. The gene C-I would be the operon C with the operator suppressed or absent. This hypothesis would satisfy the degree of dominance of the series of alleles of the C locus. From its analysis we could interpret that the gene C*-IE-7002 is a translocation of structural genes to a contiguous place of a foreign active operator; for this reason it would not act as the dominant gene C-I, which cannot be repressed since it lacks an operator. In this case the gene C*-IE-7002, which has an operator, would need the genes Ac* and li* for its regulation. This hypothesis would be true if the aleurone color manifested itself in the presence of a deficiency at the C locus. This could not be true according to McClintock (Cold Spring Harbor Symp. Quant. Biology XVI:13-44, 1951) but the hypothesis would be possible according to Coe (Genetics 47:779-783, 1962).

I think that it is possible that Dr. Randolph's line can be related to the one studied by McClintock (material which was never introduced in this Institute) and that the transposition phenomenon based on the breakage-fusion-bridge cycle may have given rise to many sublines with structural changes. In the light of the genetic regulation phenomena which are now known, such position effects could turn a character conditioned by only one gene of good expression and penetration into a fluctuating character, offering advantages in the evolution and selection of the organisms (Mazoti, 1945, Revista Argentina de Agronomía t.12, No. 3, p. 181). Perhaps with this system of transposition of chromosomic sectors the regulation of the reply of the genes or of a little sector of DNA can also be altered, causing a "self-infection" of unpredictable consequences for the organism that may change the concept about the basis of selection (Rendel, Proc. Nat. Acad. Sci. 64: 578-583, 1969) or perhaps may annul the normal inhibition of the cellular multiplication (Mazoti, 1963, Revista Facultad de Agronomía, 3a época, t. XXXIX, pp. 63-68).

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Information or seed for lines Ind.AH83 and CI617 — Iowa Stiff Stalk Synthetic was developed in 1933 and 1934 by recombining 16 lines selected by various corn breeders as being stiff stalked (Sprague, G. F., J. Amer. Soc. Agron. 38:108-117, 1946). The 16 lines recombined to form Iowa Stiff Stalk Synthetic were: Ia.I159, Ia.I224, Ia.Os420, Ia.WD456, Ind.461-3, Ill.12E, CI617, CI540, Ill. Hy, Oh3167B, Ind.AH83, Ind.Tr9-1-1-6, F1B1-7-1, A3G-3-1-3, CI187-2 and LE23. Iowa Stiff Stalk Synthetic has been used extensively in the Iowa Corn Breeding Program in basic breeding studies evaluating recurrent selection procedures for the improvement of breeding populations. It has been shown to be good for general combining ability in crosses with other varieties and also as a source population for lines having good general combining ability (e.g., B14, B37, B73 and N28).

Iowa Stiff Stalk Synthetic was developed about 40 years ago, and it is impossible to determine how many times the synthetic has been reproduced to maintain its viability. Consequently, I have been attempting to reassemble the original lines used in the formation of the synthetic variety. I am interested in resynthesizing Iowa Stiff Stalk Synthetic from the original lines to determine how its