(Floury fl/fl x floury "Blanco") F_2 = all floury

(Floury fl/fl x floury "Blanco") x Flint = Table 1

Table 1

fl/fl^a (female) x Flint

Progeny	Floury	Flint
1	111	137
2	90	84
3	99	96
4	95	102
Total	395	419

It would be interesting to compare the percentage of lysine in fl and fl^a in order to be able to establish a possible case of genic action by intrachromosomal duplication. (fl^a duplicate = fl)?

Luis B. Mazoti

2. Further studies on the effects of the paramutagenic gene c^{IP}.

In 1966 (MNL 40:62) I described a new paramutagenic gene which is very stable, has normal viability and is localized at the locus C. This new paramutagenic gene c^{IP} produces in its alleles the mutational sequences: $C^i \rightarrow c^i$ and $c^i \rightarrow c^I$.

Further studies show that:

a. The mutation rate of C^i to c^{im} (m = mutation) due to the paramutagenic gene c^{IP} is 33%. This mutation rate is homogeneous in various progenies, and does not produce mosaicism phenomena (Table 1).

Table 1

Results of the cross: $\underline{c}^{IP}/\underline{C}^{i}$ x $\underline{c}^{i}/\underline{c}^{i}$, with $\underline{C}^{i}$ to $\underline{c}^{i}$ mutation

Progeny	Colorless aleurone	Colored aleurone	Ratio
1	219	116	1.88 : 1
2	98	47	2.08 : 1
3	234	117	2.01 : 1
4	276	123	2.19 : 1
5	266	116	2.29 : 1
6	128	54	2.37 : 1
7	256	123	2.08 : 1
8	213	100	2.13 : 1
9	228	101	2.28 : 1
10	189	117	1.61 : 1
11	266	109	2.44 : 1
Total	2,373	1,123	

b. The mutation rate of $\underline{c}^{i}$ to $\underline{c}^{Im}$ is heterogeneous in the various progenies and shows mosaicism phenomena on the ears (Table 2).

Table 2

Results of the cross: $\underline{c}^{IP}/\underline{c}^{i}$ x $\underline{C}^{i}/\underline{C}^{i}$, with $\underline{c}^{i}$ to $\underline{c}^{I}$ mutation

Progeny	Colorless aleurone	Colored aleurone	Ratio
1	143	117	1.2 : 1
2	140	110	1.2 : 1
3	219	161	1.3 : 1
4	176	97	1.8 : 1
5	224	86	2.6 : 1
6	136	29	4.6 : 1
Total	1, 038	600	

c. For initiation of instability in the genes $\underline{c}^{i}$ and $\underline{C}^{i}$, the presence of the paramutagenic gene $\underline{c}^{IP}$ is required. Nevertheless, this instability, once acquired, continues at least for two generations without the presence of the paramutagenic gene. This instability is the product of mutations and reversion(s). The reversion of the mutant gene $\underline{c}^{Im}$ to its original standard form $\underline{C}^{iR}$ (R = reversion) in the absence of the paramutagenic gene was detected with the following method (crossing-over is ignored due to its low frequency in relation to the mutation rate):

$$\frac{c^{i}\quad sh}{c^{IP}\quad Sh} \times \frac{C^{i}\quad Sh}{C^{i}\quad Sh}$$

$$\downarrow$$

$$\frac{c^{Im}\quad sh}{C^{i}\quad Sh} \times \frac{C^{i}\quad sh}{C^{i}\quad sh} \longrightarrow$$

100 C Sh

56 C sh (reversion)

0 c Sh

51 c sh

$$\frac{c^{i}\quad sh}{C^{i}\quad Sh}$$

$$\frac{c^{IP}\quad Sh}{C^{i}\quad Sh}$$

The ears with large sectors of aleurone color (containing C sh kernels) could be due to a reversion of $\underline{c}^{Im}$ to $\underline{C}^{iR}$.

d. Mutation of $\underline{c}^{i}$ to $\underline{c}^{Im}$ in the absence of the paramutagenic gene $\underline{c}^{IP}$ was detected by means of the following method:

$$\frac{c^{i}\quad sh}{c^{IP}\quad Sh} \times \frac{C^{i}\quad Sh}{C^{i}\quad Sh}$$

$$\downarrow$$

$$\frac{c^{i}\quad sh}{C^{i}\quad Sh} \times \frac{C^{i}\quad sh}{C^{i}\quad sh} \longrightarrow$$

216 C Sh

14 C Sh dilute

75 C sh

102 C sh dilute

51 c sh (delayed effect)

0 c Sh

$$\frac{c^{IP}\quad Sh}{C^{i}\quad Sh}$$

$$\frac{c^{Im}\quad sh}{C^{i}\quad Sh}$$

The colorless (c sh) kernels could be interpreted as a delayed mutation of c^i to c^{Im}, induced by the paramutagenic gene c^{IP}.

e. The reversion and mutation of the same C^i allele in the absence of c^{IP} (which is responsible for initiation of gene instability) was detected by means of the following method:

$$\frac{c^{IP}\ \ Sh}{C^i\ \ Sh} \times \frac{c^i\ \ sh}{c^i\ \ sh}$$

$$\frac{c^{im}\ \ Sh}{c^i\ \ sh} \times \frac{c^i\ \ sh}{c^i\ \ sh} \longrightarrow \begin{array}{l} 44\ c^i\ Sh \text{ (reversion)} \\ 102\ c^i\ Sh \\ 139\ c^i\ sh \\ 1\ C^i\ sh \end{array}$$

$$\frac{c^{IP}\ \ Sh}{c^i\ \ sh}$$

$$\frac{C^i\ \ Sh}{c^i\ \ sh}$$

The colored (C Sh) kernels could be the reversion product of the mutant c^{im} to its original C^i (designated as C^{iR}). The C^{iR} kernels were saved and crossed again with c^i sh; the results are indicated in Table 3.

Table 3

Results of the cross: $\frac{C^{iR}\ \ Sh}{c^i\ \ sh} \times \frac{c^i\ \ sh}{c^i\ \ sh}$

Progeny	C Sh	C Sh dilute	c Sh*	C sh	c sh
1	50	47	35	1	134
2	53	28	53	1	104
3	68	20	48	1	78
Total	171	95	136	3	316

*The c Sh class of colorless kernels could be due to a second mutation of C^{iR} to c^{im2}.

Summary:

In the presence of the paramutagenic gene c^{IP}, the following mutational sequences have been obtained: $C^i \longrightarrow c^{im}$ and $c^i \longrightarrow c^{Im}$.

In the absence of the paramutagenic c^{IP} gene, the following mutational sequences have been obtained: $c^{im} \longrightarrow c^{iR}$, $c^i \longrightarrow c^{Im}$, and $c^{im} \longrightarrow C^{iR} \longrightarrow c^{im2}$. Symbols: C^i, c^I, c^I = standard alleles; m = first mutation; m2 = second mutation; R = reversion.

The present results may be interpreted in accordance with the hypothesis already described (MNL 40:62, 1966) as an excessive replication of DNA segment(s) of the paramutagenic gene c^{IP}. This segment(s) could have the power of self duplication and interaction with the C locus either in an attached or in a free state, not necessarily released into the cytoplasm. These findings suggest the possibility that a gene of a higher organism may originate episome-like particles.

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1. Pale phenotypes at the A_2 locus.

A number of distinct pale phenotypes, representing a wide spectrum of qualitative differences in anthocyanin coloration, have been isolated at the A_2 locus. These arose from a newly induced unstable a_2 mutant, a_2 m(1 1511), but are themselves stable. They fall into a sequential series of pigment types from very light pales to darker shades. (Other phenotypes representing unrelated forms of phenotypic expression have also been isolated.)

Differences in pale phenotypes may be due to one of two alternatives: (1) differential placement of the I(nr)* element (Peterson, 1966) within the A_2 locus - the position hypothesis or (2) qualitative differences in the composition of the I(nr) element - the composition hypothesis. The position hypothesis may be tested by subjecting pales of different origin to crossover tests. Differential placement would be expected to yield full color types.

It is interesting to note that in a study of the a_1 - Dt system, Professor Rhoades found novel types at the a_1 locus that had not previously been recorded in natural populations. Similar types of variants have arisen at the A_2 and Wx loci following their exposure to the Ac-Ds system (McClintock, 1951). It is evident that systems such as a_1 - Dt, Ac-Ds and En-I can significantly influence types of variation originating at a locus.

*I(nr) = suppresses gene action but does not respond to En.