Table 2

Response to Bh (Blotched) of i and c mutations

Mutagen	Mutant	Bh (Blotched)	
		Male	Female
Diethyl sulfate (DES)	i-1	-	Completely colored
Ethyl methane sulfonate (EMS)	i-2	Colorless	Colorless
Radiation (gamma rays)	i-3	'blotched'	'blotched'
EMS	c-1	Colored	Colored
"	c-2	-	'blotched'
"	c-3	-	-
"	c-4	-	Colorless
"	c-5	Colorless	Colorless
"	c-6	Slightly 'blotched'	Colorless
"	c-7	-	Colorless
"	c-8	Colorless	Colorless
"	c-9	Slightly 'blotched'	-

We are repeating these experiments to confirm our results.

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2. Breeding behaviour of mutations from I and C.

Colored kernels isolated from the progeny of the cross $\frac{\text{I Sh Bz Wx}}{\text{I Sh Bz Wx}}$ (seed treated) x C sh bz wx generally do not breed true for i mutation. Out of 16 kernels isolated this way, only 3 bred true for the change I to i. In a new series from 1442 progenies, 22 kernels were isolated, none of these showed the change from I to i on further testing. In contrast, $\frac{\text{C Sh Bz Wx}}{\text{C Sh Bz Wx}}$ (seeds treated) x c sh Bz wx yielded 6 colorless kernels in 794 progenies. Out of these 6, 3 failed to propagate but the other three had changed from C to c and bred true. Essentially then the findings are the same as those reported last year, i.e.:

(i) There is a very high proportion of non-concordant changes of *I*. *C*, in general, yields concordant changes.

(ii) The mutation rate of *I* and *i* must be much lower than *C* to *c*.

All the observations are suggestive that *I* and *C* may occupy different loci and may in fact have a functional relationship that is yet to be clarified.

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3. The basis of somatic instability in maize.

Various mutagenically-effective treatments for the Sh_2 locus failed to produce any back mutation of the closely-linked A_1 *Ds* marker complex. Nor were any *Ac*-like elements generated (Maize News Letter 42:6-7). An excision-repair model is proposed to explain the stability of *Ds* (mutations) in the absence of *Ac* and its high mutability in the presence of *Ac*. According to this model, *Ds* is considered to be a mutation that produces a specific kink in the chromosome. The *Ds* mutation can occur once or more than once in a structural gene and also anywhere in the genome. The presence of this kink is specifically recognized by the product of *Ac*. The product of *Ac* may be visualized as an excision enzyme concerned with monitoring the fidelity of the chromosomes and excising the *Ds*-type damage. *Ac* can occur in either active or inactive phase and probably in more than one location. Two major states of *Ds* are assumed to present themselves somewhat differently to the *Ac* excision enzyme. The Dissociation-type state is excised as a chromosome break and the back-mutation type is excised so that it is repairable. Intracistronic recombination and therefore the site-referability of different *Ds* 'insertions' as observed by Nelson (cited in Dawson, G.W.P. 1966. The Physiology of Gene and Mutation Expression. Proc. Symp. Prague 67-70) is also readily explained. Under the present scheme, the phenomenon of transposition cannot be explained as a unique transfer of material substance from one location to the other but rather as the occurrence of a new *Ds* kink at another location (presumably in the wake of chromosomal breakage).

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