

(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 Sh₂ locus failed to produce any back mutation of the closely-linked A₁Ds 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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