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1. a_1 --mutable of the En system.

The colorless types arising from $a_1^{m(dense)}$:

The original mutable allele, $a_1^{m(dense)}$, is recognized by its heavily mutating pattern. From testcrosses of this allele, altered patterns of mutability (timing and frequency changes) are recovered. In addition, a high number of colorless kernels arise. These colorless types do not mutate in the presence of En, i.e. their color potential is lost; and, therefore, a permanent change at the locus has occurred. These are designated $a_1^{m(nr)}$ (non-responsive) to distinguish them from $a_1^{m(r)}$ (respond) types that have a different origin and are mutable when En is present. En was not detected at the a_1 locus of these $a_1^{m(nr)}$ types. This is in contrast to Dr. Brink's comparable stable colorless type of the P^{VV} series that does possess M_p near the P locus.

A changed En - En^{mod.}:

En^{mod.} is differentiated from the normal En in its effect on the $a_1^{m(r)}$ allele since it induces a very low rate of mutability. From this En^{mod.}, however, somatic sectors and germinal mutations of high rates of mutability do occur.

Independent En effect on $a_1^{m(fine)}$:

The $a_1^{m(fine)}$ mutation is autonomously controlled and is represented by a fine clear mutable pattern. It is stable in that it gives primarily $a_1^{m(fine)}$ progeny in outcrosses. The presence of Independent En (Inde.-En) with the $a_1^{m(fine)}$ allele results in a very dense pattern--a pattern that is similar to the original autonomous dense allele. In addition to this pattern change, the combination of $a_1^{m(fine)}$ and Inde.-En results in a high rate of colorless types, which have been tested and found to be $a_1^{m(nr)}$ types. Thus, the presence of Inde.-En causes the $a_1^{m(fine)}$ allele to change to a non-responding type at a high rate. It, also, causes the loss of all color potential of the $a_1^{m(fine)}$ allele. The presence of this additional En with the autonomously mutating $a_1^{m(fine)}$ allele results in a higher and often earlier mutability which is in contrast to the characteristic responses of higher dosages of mutators, which cause later and, therefore, lighter patterns.

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