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Abstract

The alteration of a complementation pattern among *pyr-3* mutants of Neurospora

Dutta, S. K. and V. W. Woodward. The alteration of a complementation pattern among pyr-3 mutants of Neurospora.

Based on the presence or absence of aspartate transcarbamylase (ATC) activity, pyr-3 mutants fall into two classes: those with and those lacking in vitro activity. Most of the ATC$^-$ mutants are non-complementing whereas all of the ATC$^+$ types complement with two ATC$^-$ mutants (KS-43 and pyr-3d). R. H. Davis and V. W. Woodward have proposed that the pyr-3 locus specifies two active sites on one protein, one effecting the conversion of carbamylphosphate (CAP) and aspartic acid to ureidosuccinic acid and the other the synthesis of CAP, presumably from CO_2, NH_3 and ATP. It has been postulated that the non-complementing, ATC$^-$ mutants possess either no ATC protein or ATC protein damaged at both sites; the ATC$^+$ mutants have ATC protein damaged only at the CAP-synthesizing site, and mutants KS-43 and pyr-3d contain ATC protein damaged at the transcarbamylase site.

Evidence to this end was obtained (Woodward and Davis 1963 Heredity 18: 21) by changing one of the ATC$^-$, non-complementing mutants, by ultraviolet irradiation, to an ATC$^+$, complementing type. According to ATC activity, complementation, and suppressibility by a mutant known to suppress only ATC$^+$ types, the new mutation took on the qualities of the ATC$^+$ mutants. The evidence supported the idea that one of the damaged, enzymatic sites had been repaired by partial reverse mutation.

The present paper concerns the successful attempt to alter the same non-complementing, ATC$^-$ mutant (KS-23) at the second active site. All mutants were marked with second morphological mutations (col-4 and al-2). ATC$^-$, col-4 mutants were treated with an LD-50 dose of ultraviolet and over-plated onto minimal agar covered with conidia of ATC$^+$, al-2 mutants. Colonies emerging on such plates were either reversions or heterocaryons. Heterocaryon formation was verified by the recovery of both homocaryons and the reconstruction of a heterocaryon between an ATC$^+$ mutant and one of the homocaryotic components.

Three ATC$^-$, non-complementing mutants (KS-23, KS-6 and KS-139) were irradiated and over-plated onto conidia of five ATC$^+$ mutants (KS-10, 16, 20, 48 and 125). Six heterocaryons from 615 colonies were recovered; four of the heterocaryons resulted from KS-23 + KS-125 and two were from KS-6 + KS-125. Homocaryons derived from these heterocaryons were shown to be of two types: one, the original ATC$^+$ mutant, and the other a mutant capable of forming heterocaryons with ATC$^+$ mutants. By the criterion of complementation, the second component resembles KS-43 and pyr-3d, since it failed to complement the original KS-43, while the other homocaryon (KS-125) did.

Tests are under way to determine whether the alteration in complementation pattern results from mutation within the original mutation (primary site, partial revertant) or from mutation at another site (secondary site, partial revertant).

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