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Aspergillus nidulans pyrE and pyrF

Abstract

Sequencing of a gene cloned by complementation of a *pyr* mutant in Glasgow strain G190 shows this mutant to be *pyrE*, encoding dihydroorotate dehydrogenase. This discovery necessitates a revision of the genetic map derived from crosses with this strain.

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Brief Notes

Aspergillus nidulans pyrE and *pyrF*

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Sequencing of a gene cloned by complementation of a *pyr* mutant in Glasgow strain G190 shows this mutant to be *pyrE*, encoding dihydroorotate dehydrogenase. This discovery necessitates a revision of the genetic map derived from crosses with this strain.

The *pyrE* and *pyrF* mutants of *A. nidulans* were deduced by Palmer and Cove (1975 Mol. Gen. Genet. 138: 243-255) to be deficient, respectively, in dihydroorotate dehydrogenase and orotate phosphoribosyl transferase. They were reported to map on chromosome I:

anA 9.3 pyrE 6.2 adG 9.0 pyrF 6.5 luA

Subsequent work involved Cambridge strain 595 (= Glasgow G190, and FGSC A721), original genotype: *anA1 pyrE8 luA1 yA2; cnxH5*. Arst 1988 (Mol. Gen. Genet. 213: 545-547) mapped the *pyr* mutation in this strain:

anA		adG		davA		**pyr?**		luA		ornD
	10.0		0.6		0.4		5.0		2.0	
	(9/102, 14/128)		(19/3088)		(12/2728)		(6/74, 3/102, 4/82)		(3/149)	

From this map it was concluded that the mutation in this strain must be *pyrF* rather than *pyrE*, and both Glasgow and FGSC strains designations were "corrected" accordingly. However, the "*pyrF*" gene that was cloned by complementation of this mutation (Aleksenko *et al.* 1999 Mol. Microbiol. 33:599-611) has now been sequenced and proves to encode dihydroorotate dehydrogenase, so the original description of the strain as carrying *pyrE8* was correct, and the fault lies in the mapping.

Arst (1988) used two crosses with the disputed strain to map these mutants, all data being obtained by selection of a class of recombinants and subsequent analysis of outside markers, which gave a consistent order in every case. On the other hand, Palmer and Cove gave no details of their crosses or number of progeny analysed. It should also be noted that Palmer and Cove's map position for *pyrD* has also been revised in subsequent work (Aleksenko *et al.* 1999 Mol. Microbiol. 33:599-611), and that, *pyrG*, which was reported to be unlinked to *galD*, is now agreed to be quite close to it (Kafer and May 1988 Fungal Genet. Newslett. 35:13-15).

It therefore seems prudent to accept the Arst data and conclude that *pyrE* maps between *davA* and *luA*, while the position of *pyrF* is unknown. Unfortunately, no authentic *pyrF* mutant strains are now available - unless any reader knows better.