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## Abstract

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Revised allelism relationships among Aspergillus meth and gal mutants

A. methD10 and methH2 are allelic

Gajewski and Litwinska 1968 (Mol. Gen. Genet. 102:210-220) isolated a number of methionine auxotrophs, including one allele at a new locus designated methD. This mutant was reported to map on linkage group III, 7.7% from argB and 12% from methH. Since methH is 7% to the left of argB2, methD was assumed to be to its right, but Caddick and Arst 1986 (Genet. Res. Camb. 47:83-91) have now mapped methD10 to the left of argB2, in a position which should put it close to methH.

I have retested the complementation of methD10 and methH2 in heterokaryons established on MM + methionine and transferred to plain MM: no growth resulted. In addition, a cross between strains carrying the two mutants gave no meth^+ recombinants in 10^4 hybrid ascospores. I conclude that these mutants are allelic and propose that they should be regarded as methH alleles since that is the locus that has been mapped for some time. Since Gajewski and colleagues have shown that methionine suppressors are very readily obtained, it is more than likely that their original complementation and mapping results were the product of an undetected spontaneous suppressor.

I have also checked the phenotype of both mutants: Paszewski and Grabski 1975 (J. Bacteriol. 124:893-904) reported that meth-2 and meth-10 are the only methionine mutants failing to respond to homocysteine, which places their deficiencies in the final step of methionine biosynthesis - homocysteine methylation. This lack of response is confirmed, as is the fact that partial growth is shown by methH10 on choline, suggesting that this mutant is leaky and can respond to increased concentrations of a methyl donor.

B. *gal-4* and *gal-7*

C.F. Roberts 1963 (J. Gen. Microbiol. 31:45-58 and Ph.D. thesis, Glasgow 1961) originally found that these two leaky galactose mutants showed only partial complementation and were apparently closely linked: he concluded that the mutations were allelic. He also concluded from a haploidization that *gal-4* was probably in linkage group I, but the then switched to mitotic crossing-over experiments with *gal-7* and showed that this mutant could not be on either arm of chromosome I, and a further haploidization with *gal-7* suggested, on the basis of 8 segregants, that it might be on VIII.

Some time ago, I tried to retest the location of *gal-7*, and in two haploidizations concluded that it was probably in linkage group IV, but neither experiment was without its difficulties (either of *gal* classification or of allele ratios) and a third haploidization was totally inconclusive. No meiotic linkage of *gal-7* to linkage group markers has been found over the years, although sane *gal-7* is present on a number of mapping strains. I also failed to find linkage with markers on IV in one cross.

Lacking any hard evidence, in the latest edition of Genetic Maps (Cold Spring Harbor 1986) I have designated *gal-7* as belonging to a separate locus: *galH*, possibly located on IV.

Classification of galactose non-utilizers appears to be a common problem, variable from cross to cross. It seem likely that these mutants are subject to suppression by unsuspected markers in the stocks: it might be worthwhile to test for the effect of *ssbA* (sorbitol suppressor - E. Käfer 1986, FGN 33:27-28) on galactose mutants. - - - Institute of Genetics, University of Glasgow, Glasgow G11 5JS, Scotland