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Abstract

Among several amino acid analogue sensitive mutants, D.E.A. Catcheside selected mts(MN1) and mts(MN9) via their 5-methyltryptophan sensitive phenotype (1966, Ph.D thesis, University of Birmingham).

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Among several amino acid analogue sensitive mutants, D.E.A. Catcheside selected mts(MN1) and mts(MN9) via their 5-methyltryptophan sensitive phenotype (1966, Ph.D thesis, University of Birmingham). While mts(MN1) was located to the right of ylo-1 of linkage group VI by Catcheside, mts(MN9) was not assigned to any locus.

Both in Neurospora crassa and in yeast, mutants defective in general or cross-pathway control of amino acid synthesis display amino acid analogue sensitivity. mts(MN1) and mts(MN9) were therefore tested for their regulatory capacity. Both turned out to be defective in cross-pathway control (for mts(MN1) see Koch and Barthelmess, 1987, FGN 33:30-32).

Mutant strains carrying the mts(MN9) mutation failed to derepress the ornithine carbamoyltransferase of arginine synthesis, the leucine aminotransferase of leucine synthesis and the saccharopine dehydrogenase of lysine synthesis under arginine, asparagine, histidine, methionine, threonine, tyrosine and tryptophan limitation, respectively. In this respect, mts(MN9) resembled a typical cpc-1 allele (Barthelmess, 1982, Genet. Res. 39:169-185). The effect on the remaining basal enzyme activity, however, was not as pronounced as found for most cpc-1 alleles, e.g. j5 (Barthelmess, 1982), CD15 or CD86 (Davis, 1979, Genetics 93:557-575), but resembled j9, a less stringent cpc-1 allele (Barthelmess, 1982). In agreement with this, the mts(MN9) mutant was able to grow like cpc-1 (j9) on medium supplemented with 0.03% glycine, a condition that does not allow growth of cpc-1 alleles j5 or CD15 (Barthelmess, 1986, Mol. Gen. Genet. 203:533-537).

Crosses with mts(MN9) produced many unpigmented ascospores. This was a first hint that the mts(MN9) strain might carry a chromosomal aberration. When mts(MN9) was crossed with cpc-1 (CD86 or CD15) only three classes of segregants were observed: the two parental classes and a new class of slow germinating ascospores with slow vegetative growth. The latter is presumed to be a duplication bearing class, while the unpigmented ascospores probably represent the corresponding segregants carrying a deficiency. The wild type did not segregate. This suggests that mts(MN9) is involved in a translocation and is linked with cpc-1. Since further mapping studies indicated linkage of mts(MN9) with pan-1 as well as met-5 on linkage group IV, it is assumed that linkage groups IV and VI are affected by the translocation. Marker studies to find out which of the two is the donor chromosome were not performed. Very helpful discussions with Dr. Perkins made us aware of the fact that the data so far available do not allow us to draw any conclusions on the precise nature of the translocation.

Complementation studies including mts(MN9), cpc-1 (CD86 and j5) and mts(MN1) were performed as already described for mts MNl) (Koch and Bartheless, 1987). The mutants were recessive to their respective wild type alleles, but complementation of the amino acid analogue sensitive phenotype was not observed in heterocaryons carrying mutant alleles simultaneously. These findings suggest that cpc-1, mts(MN9) as well as mts(MNl) belong to the same complementation group. - - - Institut für Angewandte Genetik, Universität Hannover, 3000 Hannover FRG. Supported by the Deutsche Forschungsgemeinschaft.