The nomenclature of *mts* and *cpc* mutants of Neurospora.

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Mutants defective in general amino acid control in Neurospora have been designated *cpc* for cross pathway control (Barthelmess 1982, Genet. Res. 39:169-185, Davis 1979, Genetics 93:557-575). More recently, Koch and Barthelmess (1987, FGN 33:30-32, 1988, FGN 35:22-23) have shown that mutants sensitive to 5-methyl tryptophan and designated *mts* are *cpc-1* alleles. Despite the priority of *mts*, on the grounds of apt description, I propose that the designation *mts* lapse and *cpc* be used for this set of alleles.

Mutants of Neurospora supersensitive to 5-methyl tryptophan (5MT) were isolated as a tool to assist selection of mutants defective in the regulation of tryptophan biosynthesis (Catcheside 1966, PhD Thesis, University of Birmingham). It had been found that Neurospora was resistant to high concentrations of tryptophan analogues, including saturated solutions of 5MT, and it was reasoned that if sensitive mutants could be obtained they should permit selection of mutants defective in tryptophan pathway control as second site mutations restoring resistance.

Twelve mutants sensitive to 5MT were obtained from a single experiment. Eleven of these, MN1 to MN8, MN10, MN13 and MN16, were tentatively assigned to a single locus, *mts*, 0.5 units distal to *ylo-1* on linkage group VI, on the basis that each recombined with a frequency of 0.03% or less with MN1. Crosses between MN9 (three strains) and MN1 (two strains) yielded 2.1%, 3.7% and 5.1% MT resistant progeny. Koch and Barthelmess (1988) reported that MN9 fails to complement either MN1 or *cpc-1* alleles, indicating each of these three mutations to be allelic despite the relatively high recombination frequency between MN1 and MN9. Koch and Barthelmess also reported that MN9 contains a duplication generating chromosomal rearrangement involving linkage groups IV and VI. Barthelmess and Krüger (unpublished) investigated the expression of *cpc-1* in MN9. When total RNA of MN9, wild-type and *cpc-1* mutant strains was investigated in Northern blots, no hybridization with a radiolabelled probe of the *cpc-1* gene was found for MN9. This would indicate that the *cpc-1* gene is involved in the rearrangement of MN9. In view of this, all of the original 5MT resistant mutants were rechecked. Crosses to Emerson wild types show the aberration associated with MN9 to be present in the original isolate and all tested progeny of backcrosses to wild type. MN3, MN4 and MN5 also yield unpigmented spores in crosses to wild type, indicating they too contain chromosomal rearrangements. However, there is no evidence of a chromosomal rearrangement in the remaining nine MN alleles. Information on the plieotropic phenotype of MN1 has been published (Catcheside 1971, Proc. Austral. Biochem. Soc. 4:17, 1978, Neurospora Newsl. 25:17-18). The enhanced sensitivity of MN1 to 8-aza-adenine suggests that cross pathway control may extend beyond the regulation of amino acid pathways in Neurospora.