

cpl-1: A Neurospora mutant sensitive to chloromphenicol

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Abstract

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NEW MUTANTS AND STOCKS

Chalmers, J.H. and P. St. Lawrence²

cpl-1: A *Neurospora* mutant
sensitive to chloromphenicol.

Wild type *Neurospora* is relatively resistant to most of the antibiotics and inhibitors which have been used to select mitochondrial mutants in yeast and other organisms (Thayer, 1969, *Neurospora Newsl.* 15: 20; Chalmers, 1974, *Neurospora Newsl.* 21: 20; Al-Saqur, 1975, *Neurospora Newsl.* 22: 6). This resistance may be due either to permeability barriers or to an alternate terminal oxidase which bypasses most of the mitochondrial electron transport chain (Lambowitz and Slayman, 1971, *J. Bact.* 108: 1087). We have selected mutants which lock this alternate pathway and as a result are hypersensitive to antimycin A even on fermentable media (Chalmers, 1974, *Genetics* 78: 543; Edwards *et al.*, 1976, in "Genetics and Biogenesis of Chloroplasts and Mitochondria", Th. Bucher, *et al.*, eds. North Holland Press) by starvation for inositol in the presence of low levels of the drug. One such mutant, ANTAS6, was found also to be inhibited by chloromphenicol. Because other antimycin A sensitive mutants are not appreciably more sensitive to chloromphenicol than is wild type, the mutant was renamed cpl-1.

The cpl-1 mutant was induced by U.V. light in a sn₁cr strain (Perkins, 1971, *Neurospora Newsl.* 18: 12) which also carried inl (JH319) and trp-3 (td120). Inositol starvation was continued for 5 days at 30°C in Vogel's Medium N, supplemented with 200 µg/ml of L-tryptophan and 0.25 µg/ml of antimycin A. Surviving conidia were plated on Vogel's medium without the drug, but with the addition of inositol (150 µg/ml) and tryptophan. Colonies obtained after 2-3 days of growth were replica plated on medium containing 0.3 µg/ml of antimycin A. Putative mutants were crossed to an Oak Ridge wild type to restore mycelial morphology.

The cpl-1 mutant is inhibited by about 1 µg/ml of antimycin A and by less than 0.5 mg/ml of chloromphenicol (wild type is resistant to 4 mg/ml chloromphenicol). The cytochrome spectrum of the mutant resembles that of wild type when both are grown on minimal medium without drugs. Although other antimycin A sensitive mutants lock the cyanide- and azide-insensitive alternate oxidase, cpl-1 retains it and will express the oxidase when incubated with chloromphenicol (2 mg/ml) for a few hours.

To test whether the observed sensitivity was due to an alteration in cytosolic ribosomes, the incorporation of ³H-leucine was studied with and without 1 or 2 mg/ml of chloromphenicol or 100 µg/ml of cycloheximide (Hawley and Greenawalt, 1970, *J. Biol. Chem.* 248: 3574). Over a 30 minute period, no significant differences between cpl-1 and wild type were seen.

The nature of the cpl-1 mutation is unknown. It has been mapped to linkage Group VI, and displays 42% recombination with trp-2 and 24% with ylo-1. Two known modifiers of permeability mod-5 (Barratt and St. Lawrence, 1969, *Neurospora Newsl.* 15: 15) and mts (Catcheside, 1978, *Neurospora Newsl.* 25: 17) also map to linkage Group VI, but allelism tests have not been performed. The mutant readily reverts with both U.V. and "nitrosoguanidine". We have examined a number of such revertants, induced in both monocytons and heterocytons, for non-Mendelian inheritance, but to date only nuclear mutations have been observed; this may be a function of the genetic background. cpl-1 should be a useful mutant for studies on mitochondrial biosynthesis. (Supported in part by Training Grant TØ1-5-GM00367.) ■ ■ ■ Department of Biochemistry, Baylor College of Medicine, Houston, TX 77030; Department of Genetics, University of California, Berkeley, CA 94720.