Fungal Genetics Reports

Volume 26 Article 2

# cpl-1: A Neurospora mutant sensitive to chloromphenicol

J. H. Chalmers
*Baylor College of Medicine*

P. St. Lawrence
*University of California*



Recommended Citation
Chalmers, J. H., and P.S. Lawrence (1979) "cpl-1: A Neurospora mutant sensitive to chloromphenicol," *Fungal Genetics Reports*: Vol. 26, Article 2. https://doi.org/10.4148/1941-4765.1690

# cpl-1: A Neurospora mutant sensitive to chloromphenicol

**Abstract**
*cpl-1*: A Neurospora mutant sensitive to chlaromphenicol [sic]

Chalmers, J.H! ond P. St. Lawrence[2]

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

Wild type Neurosporo is relatively resistant to most of the antibiotics and inhibitors which hove 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 on alternate terminal oxidase which bypasses most of the mitochondriol electron transport chain (Lambowitz and Slayman, 1971, J. Bact. 108: 1087). We lave selected mutants which lock this alternate pathway and as a result ore hypersensitive to ontimycin 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 ontimycin 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 sn,cr strain (Perkins, 1971, Neurorporo 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 conidio were plated on Vogel's medium without the drug, but with the addition of inositol (150 µg/ml) ond 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 ofantimycin A and by less than 0.5 mg/ml of chloromphenicol (wild type is resistant to 4 mg/ml chloramphenicol). The cytochrome spectrum of the mutant resembles that of wild type when both ore grown on minimal medium without drugs. Although other ontimycin A sensitive mutants lock the cyanide-b ond azide-insensitive alternote oxidase, cpl-1 retains it ond will express the oxidase when incubated with chloromphenicol (2 mg/ml) for a few hours.

To test whether the observed sensitivity woo due to on alteration in cytosolic ribosomes, the incorporation of $^{3}$H-leucine was studied with and without 1 or 2 mg/ml of chloramphenicol 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 ond 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 Newrl. 25: 17) also map to linkage Group VI, but allelism tests hove not been performed. The mutant readily reverts with both U.V. and "nitrosoguanidine". We have examined a number of such revertants, induced in both monocaryons and heterocaryons, for non-Mendelion inheritance, but to dote only nuclear mutations have been observed; this may be a function of the genetic background. cpl-1 should be a useful mutant for studier on mitochondriol biosynthesis. (Supported in part by Training Grant TØI-5-GM00367.) [1] Department of Biochemistry, Baylor College of Medicine, Houston, TX 77030; [2]Department of Genetics, University of California, Berkeley, CA 94720.