cpl-1: A Neurospora mutant sensitive to chloromphenicol

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Abstract

cpl-1: A Neurospora mutant sensitive to chloromphenicol [sic]
NEW MUTANTS AND STOCKS

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\(cpl-1\): 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 lack this alternate pathway and are therefore hypersensitive to o-nitroguanidine even on fermentable media (Chalmers, 1974, Genetics 78: 543; Edwards \textit{et al.}, 1976, in "Genetics and Biogenesis of Chloroplasts and Mitochondria" , Th. Bucher, \textit{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 o-nitroguanidine 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 \(spcr\) strain (Perkins, 1971, Neurospora NewsL. 18: 12) which also carried \(inl\) (JH319) and \(trp-3\) (td120). Inositol starvation was continued for 5 days at \(30^\circ\)C in Vogel's Medium N, supplemented with \(200 \mu\text{g/ml}\) of L-tryptophan and \(0.25 \mu\text{g/ml}\) of o-nitroguanidine. Surviving conidia were plated on Vogel's medium without the drug, but with the addition of inositol (\(150 \mu\text{g/ml}\)) and o-nitroguanidine. Colonies obtained after 2-3 days of growth were replica plated on medium containing \(0.3 \mu\text{g/ml}\) of o-nitroguanidine. Putative mutants were crossed to an Oak Ridge wild type to restore mycelial morphology.

The \(cpl-1\) mutant is inhibited by about \(1 \mu\text{g/ml}\) of o-nitroguanidine and by \(0.5 \mu\text{g/ml}\) of chloromphenicol (wild type is resistant to \(4 \mu\text{g/ml}\) chloromphenicol). The cytochrome spectrum of the mutant resembles that of wild type when both are grown on minimal medium without drugs. Although other o-nitroguanidine sensitive mutants lack the cytochrome-oxidase-insensitive alternate oxidase, \(cpl-1\) retains it and will express the oxidase when incubated with chloromphenicol (\(2 \mu\text{g/ml}\)) for a few hours.

To test whether the observed sensitivity was due to an alteration in cytosolic ribosomes, the incorporation of \(^{3}\text{H}-\text{leucine was studied with and without 1 or 2 \mu\text{g/ml}\) of chloromphenicol or 100 \mu\text{g/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 \(yla-1\). Two known modifiers of permeability mod-5 (Barrott and St. Lawrence, 1969, Neurospora NewsL. 15: 15) and \(mnts\) (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 monokaryons and heterokaryons, 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 T32-GM00367.)

\[\text{Supplement 1: 32-42.}\]