Fungal Genetics Reports

Volume 34 Article 10

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Recommended Citation

Martinelli, S. D., and A. Zamir (1987) "Variable susceptibility of laboratory strains of Aspergillus nidulans to hygromycin B and other ribosomal antibiotics," *Fungal Genetics Reports*: Vol. 34, Article 10. https://doi.org/10.4148/1941-4765.1556

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Abstract

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We have been using the ribosomal antibiotics cycloheximide (CHX), paromomycin (PAR) hygromycin (HYG) and geneticin (GEN) to select mutants with altered ribosomal proteins and have found that a random selection of genetically marked strains of *A. nidulans* have widely differing responses to these compounds, when added to solid medium. The majority of strains gave the same range of responses as the standard Glasgow strain with the *biA1* mutation and from which many Aspergillus strains are derived by mutation and recombination. Particularly notable exceptions are strains SDM12 (FGSC A397), SDM108 (FGSC A64) and SDM10 (isolated from a cross between SDM12 and SDM315 [*yA2 phenA2*]), which are hypersensitive to 2mM PAR, 200 uM GEN and 300 uM HYG. Strains SDM108 and SDM10 are also very sensitive to 0.3 mg/ml CHX. Master strain D is fairly susceptible to these antibiotics but not to the extent of the aforementioned.

In crosses with these strains the hypersensitive phenotype segregates as follows:

1. Hygromycin resistance

strain SDM390 (*fwA1 pabaA1 sB43 alX4*) X strain SDM12 (*pantoB100*)
phenotype: normal sensitivity hypersensitive
segregation: 1:l

2. Hygromycin and cycloheximide resistance

strain SDM108 (*wA3 biA1 riboE6*) X strain SDM10 (*phenA2*)
phenotype: hypersensitive
hygromycin resistance to hypersensitivity: 1:7
cycloheximide resistance to hypersensitivity 1:3

The result from the latter cross was particularly surprising since neither parent exhibited any resistance to cycloheximide and yet the combination could produce resistant progeny presumably resulting from complementary gene action. The resistance was associated with an abnormal morphology, slower growth and poor conidiation. Attempts to back-cross progeny have failed owing to the infertility of these strains. Again because of fertility and reluctance to form diploid strains, it is not known whether the sensitivity of strains SDM12 and SDM108 is allelic. The hygromycin resistance may have been generated by three complementing genes.

The variable sensitivity is important for two reasons. Firstly, our attempts to demonstrate the inheritance of aminoglyoside resistance mutations isolated in strain SDM 108 have been hampered by the difficulty of finding other strains with the same resistance genotype as SDM108, for the purpose of outcrossing. Secondly, several workers are using the bacterial hygromycin B resistance as a marker in transformation experiments, but they may have unwittingly picked relatively resistant strains for their experiments. If we can map the hypersensitivity to a linkage group, it should be easy to introduce this allele into the recipient strains used by other laboratories in a controlled way without affecting the fertility of those strains (strain SDM108 and its derivatives are very infertile).

Careful screening of stock strains is recommended before commencing any work with antibiotics. - - - Dept. of Biology, Birkbeck College, University of London, Malet Street, London WClE 7HX, England