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A mutant strain of *Aspergillus nidulans* is hypersensitive to cycloheximide

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Abstract

We routinely use a strain derived from the master strain A513 (FGSC) for mitotic linkage studies in our laboratory. This strain carries the *ActA1* mutation on linkage group III, which should confer resistance to actidione (trade name for cycloheximide). However, we have observed that our strain 513s is hypersensitive to actidione in comparison with a wild-type strain at the *ActA1* locus (our collection).

A mutant strain of *Aspergillus nidulans* is hypersensitive to cycloheximide

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We routinely use a strain derived from the master strain A513 (FGSC) for mitotic linkage studies in our laboratory. This strain carries the *ActA1* mutation on linkage group III, which should confer resistance to actidione (trade name for cycloheximide). However, we have observed that our strain 513s is hypersensitive to actidione in comparison with a wild-type strain at the *ActA1* locus (our collection).

In the haploid, the *ActA1* mutation can be selected by an actidione concentration in complete medium of 1.25 g/l (Warr and Roper 1965. J. Gen. Microbiol. 40:273-281). We have tested by point inoculation the actidione resistance of 513s and the wild type strain to different doses ranging from 0.0075 g/l to 1.5 g/l. The growth of 513s was completely inhibited at all these doses except at the lowest one, where a small colony developed. The wild-type strain grew up to 0.75 g/l; therefore, the difference between the two strains was about 100 times.

A similar result has been obtained by Dr. R. Crebelli at the Istituto Superiore di Sanità (personal communication), while FGSC reports the original A513 is still fully resistant to cycloheximide (personal communication).

We made a cross between the ActA⁺ strain 35 (*anA1 pabaA1; yA2; methG1; nicA2; sC12*; our collection) and 513s, selecting for actidione-resistant ascospores at a dose of 1.25 g/l. 26 actidione-resistant, well-conidiated colonies developed from 2430 crossed viable ascospores plated. These data suggest that the determinant of the actidione hypersensitivity maps about 1 cM from the *ActA1* locus.