

Date: Fri, 21 Oct 1994 12:27:38 -0700 (PDT)
From: Christopher John Roberts <roberts@darkwing.uoregon.edu>
Subject: Strain question

Hi Craig,

I have a question about a strain that I received from the FGSC.

FGSC #3915, met-7 thi-3, allele NM251 18558

I studied this strain as part of a study of the effect of methionine starvation on DNA methylation levels. Starvation of methionine auxotrophs for methionine causes reduced DNA methylation due to a reduction of the intracellular SAM pools. I showed this for representative mutants from all of the met complementation groups.

My question has to do with an observation I've made about crosses involving the above strain. I crossed this strain to a wild type strain from our collection which contained the al-2 mutation. I picked a progeny from that cross that was both al-2 and met-7 (strain C22-19), and crossed this back to the original met-7 strain (#3915). So this cross was heterozygous for al-2, and I would predict that half the progeny from the subsequent cross would be al+ and half al-2. What I found was a high frequency of al+/al-2 mosaic cultures arising from single germinated ascospores (these results were reported in a paper from our lab, Foss et al., (December, 1993) Science 262:1737). Subsequently we have found that the strain #3915 has a mutation unrelated to met-7 that has the following phenotypes:

1. ascospores with aberrant morphology.
 2. high frequency of aneuploidy
 3. at present it is unclear whether the mutation is a recessive trait or is partially dominant.
- It does appear to be a single mutation. A sister spore of C22-19, called C22-18, behaved normally when backcrossed to strain #3915, i.e., no aneuploids were seen, and all ascospores were of normal shape.

Ann Hagemann in our lab has done some microscopy, and it appears that the aneuploidy arises from the aberrant formation of ascospores, such that some of the spores get 3 nuclei (these are preliminary results).

My question is, do you know of any mutations of Neurospora that give these phenotypes, and is it possible that the genetic background of strain #3915 might explain where this mutation came from? Since the markers for #3915 all checked out (i.e., met-, thi-, mtA) it seems unlikely that the wrong strain was sent.

Any information or thoughts about this you might have will be greatly appreciated. Thanks for your consideration.

Regards,

Chris