

1: Genetics 1998 Aug;149(4):1777-86

Analysis of fluG mutations that affect light-dependent conidiation in *Aspergillus nidulans*.

Yager LN, Lee HO, Nagle DL, Zimmerman JE.

Department of Biology, Temple University, Philadelphia, Pennsylvania 19122, USA.
lyager@thunder.ocis.temple.edu

Conidiation in *Aspergillus nidulans* is induced by exposure to red light but can also be induced by blue light in certain mutant strains. We have isolated a mutation in the fluG gene that abolishes responsiveness to red light but does not affect the response to blue light. It has been shown that the veA1 (velvet) mutation allows conidiation to occur in the absence of light. We have identified three other fluG mutations that suppress the veA1 phenotype; these double mutants do not conidiate in the dark. The mutations described here define two new phenotypic classes of fluG alleles that display abnormal responses to light. We have characterized these mutations with respect to their molecular identity and to their effect on fluG transcription. Although it has been shown that fluG is required for the synthesis of an extracellular factor that directs conidiation, we do not detect this factor under conditions that promote conidiation in the veA1 suppressors. Furthermore, extracellular rescue is not observed in fluG deletion strains containing the wild-type veA allele. We propose that a genetic interaction between fluG and veA influences the production of the extracellular signal and regulates the initiation of conidiation.

2: Prog Ind Microbiol 1994;29:429-54

Sexual sporulation.

Champe SP, Nagle DL, Yager LN.

Waksman Institute, Rutgers University, Piscataway, NJ 08855.

3: Mol Cell Biol 1992 Sep;12(9):3827-33

Isolation of a gene required for programmed initiation of development by *Aspergillus nidulans*.

Adams TH, Hide WA, Yager LN, Lee BN.

Department of Biology, Texas A&M University, College Station 77843.

In contrast to many other cases in microbial development, *Aspergillus nidulans* conidiophore production initiates primarily as a programmed part of the life cycle rather than as a response to nutrient deprivation. Mutations in the acoD locus result in "fluffy" colonies that appear to grow faster than the wild type and proliferate as undifferentiated masses of vegetative cells. We show that unlike wild-type strains, acoD deletion mutants are unable to make conidiophores under optimal growth conditions but can be induced to conidiate when growth is nutritionally limited. The requirement for acoD in conidiophore development occurs prior to activation of brlA, a primary regulator of development. The acoD transcript is present both in vegetative hyphae prior to developmental induction and in developing cultures. However, the effects of acoD mutations are