Nitrate Repression of Xanthine Dehydrogenase II in *Aspergillus nidulans*

By C. SCAZZOCCHIO and F. B. HOLL (*Department of Genetics, University of Cambridge, Cambridge CB4 1XH, U.K.*)

There are two xanthine dehydrogenases in *Aspergillus nidulans*. Xanthine dehydrogenase I is induced by uric acid. Xanthine dehydrogenase II has also a nicotinate dehydrogenase activity and is induced by nicotinate. Genetic evidence indicates that these enzymes share a peptide and a cofactor. Both enzymes are repressed by ammonia. Xanthine dehydrogenase II is also repressed by nitrate. The mechanism of this specific repression was studied in mutant *apl^r*, which is constitutive, but not de-repressed, for xanthine dehydrogenase II. A nitrite reductase-*apl^r* double mutant (*nirA₂-apl^r*) is repressible by nitrate, indicating that it is not through conversion into ammonia that nitrate represses. A *nir⁻¹* (nitrate reductase- and nitrite reductase-non-inducible)-*apl^r* double mutant is de-repressed, whereas a *nir^c₁-apl^r* (mutant *nir^c₁* is a constitutive allele of mutant *nir⁻¹*) double mutant is normally repressed by nitrate. It is concluded that both nitrate and a protein under the control of the *nir* locus are necessary for the repression of xanthine dehydrogenase II. The introduction of a *nirD* mutation (*nirD* is the nitrate reductase structural gene) into an *apl^r* strain showed that this protein necessary for repression is nitrate reductase itself. Some of the *nirD-apl^r* double mutants belong to the de-repressed class, and one belongs to the super-repressed class. As the absence of the nitrate reductase protein results in de-repression it is concluded that the mechanism of control is negative.

Some speculation on the physiological role of nitrate repression of xanthine dehydrogenase can be put forward. Xanthine dehydrogenase II is also a nicotinate dehydrogenase. Growth on nitrate as nitrogen source demands NADPH as electron donor. Repression of xanthine dehydrogenase II by nitrate saves nicotinate for the synthesis of NADPH.

This work is supported by the Science Research Council. F. B. H. gratefully acknowledges support by a Donald Vernon Snider Memorial Fellowship and by a Medical Research Council of Canada Research Studentship.

Alcohol and Amine Catabolism in the Fungus *Aspergillus nidulans*

By M. M. PAGE and D. J. COVE (*Department of Genetics, University of Cambridge, Cambridge CB4 1XH, U.K.*)

Aspergillus nidulans can utilize methylamine and ethylamine as sources of nitrogen, and ethanol and ethylamine as sources of carbon. Mutant strains unable to utilize acetate as a carbon source are, in addition, unable to utilize either ethanol or ethylamine, indicating that the catabolism of these latter two compounds involves common intermediates. Mutant strains able to utilize acetate but unable to utilize ethanol and/or ethylamine have been isolated. At least four genes are involved. Mutation in two (*mauA* and *mauB*) can lead to the inability to use either methylamine or ethylamine as nitrogen source, and ethylamine as a carbon source, while not affecting the use of ethanol. Mutation in the *ald* gene can prevent the use of either ethylamine or ethanol as carbon source while not affecting the use of methylamine or ethylamine as nitrogen source. *ald* mutants excrete acetaldehyde when growing on ethanol. Strains mutant in the fourth (*alc*) gene are of two types. One class is able to utilize ethylamine as a nitrogen and carbon source, and is cross-fed by *ald* mutants, whereas the other class can only use ethylamine as a nitrogen source.

These findings are consistent with the independent conversion of ethanol and ethylamine into acetaldehyde, which is then converted into acetate. An alcohol dehydrogenase has been detected in wild-type, *mauA*, *mauB* and *ald* strains, but is absent from *alc* strains. A monoamine oxidase has been detected in wild-type, *ald* and *alc* strains. This activity is absent from *mauA* and *mauB* strains. Attempts to detect an acetaldehyde-oxidizing enzyme have met with only limited success. A low activity has been detected in wild-type and the cross-fed class of *alc* mutants, but no activity can be detected in the other class of *alc* mutants or in *ald* mutants.

If the enzymes of alcohol oxidation form a complex, and acetaldehyde remains bound to this, the above observations may be accounted for, as may the finding that exogenous acetaldehyde fails to act as a carbon source and that ethylamine is a much poorer carbon source than ethanol.

We acknowledge gratefully the financial support of the Science Research Council.