These results suggest that the addition of nitrate increases the flow of metabolites through the pentose phosphate pathway, and that this flow is regulated both by enzyme activities and by the availability of a product of nitrate metabolism.

O. H. thanks the Medical Research Council for a research studentship.

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Studies of Temperature-Sensitive *cnx* Mutants in the Fungus *Aspergillus nidulans*

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In *Aspergillus nidulans*, mutation in any one of five genes (designated *cnx*) can lead both to the loss of nitrate reductase and xanthine dehydrogenase activity, and to the inability to utilize both nitrate and hypoxanthine as nitrogen sources (Pateman *et al.*, 1964). Strains mutant in one of those genes (*cnxE*) are partially repaired for both growth and enzyme activity by high concentrations of molybdate (Arst *et al.*, 1970). It has therefore been proposed that some or all of the *cnx* genes specify a molybdenum-containing cofactor necessary for both nitrate reductase and xanthine dehydrogenase activity.

To determine whether the *cnx* genes specify protein components of nitrate reductase or enzymes required for the synthesis of the cofactor from the metabolic pool, the temperature-stability *in vitro* of nitrate reductase from temperature-sensitive *cnx* mutants has been studied. Temperature-sensitive mutations in all of the five *cnx* genes are not yet available, but it has been shown that those in one (*cnxH*) result in the production of a nitrate reductase markedly less stable than that of the wild-type. In two others (*cnxE* and *cnxF*) no differences in stability have been demonstrated. It therefore appears that the molybdenum cofactor contains at least one protein component, but that several enzymically catalysed modifications are necessary for its synthesis.

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Chlorate Toxicity in the Fungus *Aspergillus nidulans*

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It has been shown that a number of both assimilatory and dissimilatory (respiratory) nitrate reductases are also able to reduce chlorate (Pichinoty, 1964; Pichinoty & Méténier, 1966; MacDonald & Coddington, 1968). Mutant strains of *Ustilago maydis* lacking nitrate reductase are found to be resistant to chlorate, which is toxic to wild-type strains (Lewis & Fincham, 1970); and strains of *Escherichia coli* and a number of related species, selected for resistance to chlorate toxicity, have been shown to lack nitrate reductase (Piéchaud *et al.*, 1967). It has therefore been proposed that chlorate is not itself toxic, but is rendered toxic by nitrate reductase-catalysed conversion into chlorite, which is equally toxic to strains possessing or lacking nitrate reductase.

In *Aspergillus nidulans*, mutation in a number of different genes can effect nitrate assimilation (Pateman *et al.*, 1967). These include five *cnx* genes, which specify a cofactor required by nitrate reductase (Pateman *et al.*, 1964), the structural gene for nitrate reductase, *nia*, and the *nir* gene, which specifies a protein necessary for induction of nitrate reductase synthesis. Mutations in any of these seven genes can abolish nitrate reductase activity. It has been shown, however, that not all such mutations lead to chlorate resistance. Further, mutations in the *nia* gene have now been obtained that render strains resistant to chlorate only on certain nitrogen sources. In addition, chlorate-resistant strains have been obtained that appear normal with respect to nitrate assimilation.

As a result of these findings it appears likely that the chlorate is toxic to *Aspergillus*, not principally because it is converted into chlorite, but instead because chlorate interferes with the breakdown of organic nitrogen compounds to provide sources of nitrogen. Some evidence is available that suggests that chlorate might in this respect mimic nitrate, which may also control the breakdown of such compounds.

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