Chambers, J. A. A. **The *i* phenotype of *Neurospora crassa*.**

The *i* mutant of *N. crassa* is an enhancer of the *am* mutant phenotype; i.e., the leaky growth of *am* mutants is blocked in the *i,am* double mutant. The *i* single mutant is also unable to use proline as sole nitrogen source. Further studies have shown that it is also unable to use urocanate, methionine, alanine, isoleucine, valine, B.S.A., hypoxanthine, uridine or urea as sole nitrogen source. Although the *i* mutant cannot use B.S.A. or methionine as sole nitrogen source, it can use either as sole sulphur source.

Synthesis of proline oxidase in wild type is weakly induced by proline and repressed by ammonium (Facklam and Marzluf 1978, Biochem. Genet. 16:343). However, in *i* the enzyme is present in cultures grown on ammonia as sole nitrogen source and rapidly repressed upon transfer to a proline-containing medium.

The most likely explanation for the leaky phenotype of *am* mutants is the existence of an alternative pathway. Two possibilities are: a) the NAD-dependent glutamate dehydrogenase, although the properties of the Neurospora enzyme and evidence from *Aspergillus nidulans* make this unlikely, or b) a more complex pathway involving the enzymes glutamine synthetase and glutamate : oxoglutarate amino transferase (Hammett and More 1980, Biochem. Biophys. Res. Commun. 92:127).

Any explanation for the *i* phenotype requires that this postulated alternative pathway be affected as well as the synthesis of the nitrogen metabalite which causes ammonium repression. Recent studies of the regulation of uricase synthesis have shown that glutamine is the effector in repression (Wang and Marzluf 1979, Molec. gen. Genet. 176:385). These observations in connection with the tight linkage of *i* to *gln* suggest that the *i* phenotype may be due to a defective glutamine synthetase. - - - Department of Genetics, The University of Leeds, Leeds LS2 9JT, United Kingdom.