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# Further properties of the i (en-am-1) mutant

## Abstract

Further properties of the *i (en-am-1)* mutant

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Following the report of J. A. A. Chambers (1980 Neurospora Newsl. 27: 17) the following further information may be of interest.

## Further properties of the *i (en-am-1)* mutant.

I first observed the effect of a linked mutation interacting with *am* in 1949 during analysis of a cross of *am* (32213) x *arg-9* (E5041). The mutation, called *i*, came from the ES041 parent; it may have been already present in its wild type progenitor or it may have arisen at the same time as the *arg-9* mutation (E5041) was bred from a culture exposed to radiation in the Eniwetok atomic bomb test; the mutant is not at present in the stock list). It interacts with NADP-glutamate dehydrogenase-deficient am alleles to give a phenotype characterized by great sensitivity to inhibition by ammonium ions. I keep am *i* doubles on glutamate as sole N-source. In liquid culture 0.5 mM $NH_4Cl$ inhibits growth (measured as dry weight after 3 days at 25°) by 80 to 90 per cent when 0.1 M glutamate is the main nitrogen source.

R. R. Burk, in his Ph.D. thesis (Cambridge, 1965; see also Burk 1364 Neurospora Newsl. 6: 27) reported that single mutant *i*, though generally cryptic, was unable to use L-proline as sole N-source. I find that wild type grows poorly in liquid N-free Vogel's supplemented with 20 mM L-proline and *i* does not grow at all, On sorbose agar test plates, with the same added N-source, *i* is characterized by rather rapidly spreading but very thin growth.

Some years later I noticed that, unlike true wild types, *i* segregants were not inhibited at all by 0.02 M glycine added to test plates to sharpen the am auxotrophy. Glycine-resistance and proline nonutilization segregated together in the fairly limited number of asci scored. I have recently confirmed this observation and also shown that proline nonutilization and glycine-resistance are correlated with resistance to both 0.5 mM DL-ethionine and 0.2 mM DL-*p*-fluorophenylalanine, both of which completely inhibit wild type on sorbose-agar test plates.

From a cross of *am i* with wild type, 17 completely germinated asci consisted of 7 parental ditypes and 10 tetratypes (looser linkage than has been found in the past). Segregants from two PD and five tetratype asci were tested for inhibition by the analogues. From each tetratype ascus one of the two $am^+$ segregants was resistant to both analogues and was also proline-nonutilizing and glycine-resistant. No such segregants were found in the parental ditypes. Two of the drug-resistant (putative *i*) segregants have been checked by crossing to *am* and both gave *am i* recombinants at about the expected frequency. Four completely germinated asci from the *trans* cross *am* + x + *i*, were all parental ditypes. All the *am* segregants (as well as the + *i* parent) were resistant to both analogues, as well as being glycine-resistant and proline-nonutilizing.

I postulate that the *i* mutation (called *en-am-1* by N. Dunn-Coleman, *personal communication)* is permanently repressed with respect to one or more amino acid uptake systems as well as in some way preventing ammonia utilization by the alternative amination system (glutamine synthetase plus GOGAT) which normally permits leaky growth when NADP-glutamate dehydrogenase is lost.

Chambers (op. cit.) suggests that *i* may be a mutation in the glutamine synthetase gene *gln*. From a cross *am gln* x *am i* I found 0.67% (9/1342) *am* $gln^+ i^+$ recombinants, corresponding to 1.3 map units between *gln* and *i*. This seems high for alleles, but a connection between *gln* and *i* is not ruled out.

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