
Page 353, lines 14–26 should read:

tryp-1 locus exhibit a variety of phenotypes with alterations in any or all of the activities of the complex (DeMoss, Jackson and Chalmers 1967). A major class of these mutants lacks all three activities and accumulates the catalytically inactive tryp-2 gene product (Arroyo-Begovich and DeMoss 1969; Chalmers and DeMoss 1970). When extracts of these tryp-1 and tryp-2 mutants are mixed, an active wild-type complex is generated in vitro, indicating that the genetic change in these strains leads to the production of completely inactive gene products (Arroyo-Begovich and DeMoss 1969; Chalmers and DeMoss 1970).

A number of observations have been made in Neurospora that mutations which cause complete loss of enzyme function (Seale 1968), or loss of antigenic crossreaction (Terry 1966), or pleiotropic defects in multienzyme complexes (Case and Giles 1968) are often suppressed by super suppressors analogous to the nonsense suppressors described in bacteria (Garen 1968) and in yeast (Hawthorne 1969).