In S. cerevisiae the non-protein amino acid gamma-aminobutyric acidplays a role in nitrogen utilization and oxidative stress tolerance. GABA accumulation occurs through permease-mediated uptake by Uga4p, Put4p, and Gap1p, or intracellular production via glutamate degradation by the glutamate decarboxylase Gad1p. GABA degradation into succinate is a two-step process mediated by the gene products of UGA1 and UGA2. UGA1 encodes a 4-aminobutyrate aminotransferase that deaminates GABA to succinate semialdehyde which in turn is converted to succinate by the succinate semialdehyde dehydrogenaseencoded by UGA2. S. cerevisiae cells in which GABA degradation is blocked are more sensitive to oxidative stress and can no longer grow on GABA as their sole nitrogen source. The presence of GABA causes an increase in expression of UGA1 and UGA2 which is mediated by the transcriptional activators Uga3p, Dal81p, and Gln3p. Uga3p and Dal81p bind to upstream activation sites called the UAS-GABA element found in the promoters of GABA regulated genes.. Gln3p is involved in a more general nitrogen regulation of transcription through binding of the promoter element UAS-GATA. Levels of UGA2 transcript are also upregulated under conditions of oxidative stress.GABA-transaminases have been identified in various organisms, including UGA1 homologs from yeasts Ustilago maydis and Aspergillus nidulans. Mutations in the human GABA-aminotransferase gene, ABAT, have been associated with the disease GABA-transaminase deficiency, the clinical features of which include developmental and neurological abnormalities.About glutamate degradation In S. cerevisiae, the main pathway for glutamate degradation is catalyzed by the glutamate dehydrogenase encoded by GDH2. However, glutamate can also by degraded into gamma-aminobutyrateby the glutamate decarboxylase Gad1p and then converted into succinate by the enzymes encoded by UGA1 and UGA2. Glutamate degradation by this pathway and expression of its genes have been shown to be important for oxidative stress tolerance. Conditions of oxidative stress elevate the transcript levels of GAD1 and UGA2. UGA1 and UGA2 expression is also upregulated in the presence of GABA which is mediated by the transcriptional activators Uga3p and Uga35p/Dal81p, 6). These transcription factors bind to upstream activation sites in the promoters of GABA-regulated genes known as the UAS-GABA. Regulation of Gad1p is suggested to be linked to calcium levels as the protein is able to bind calmodulin. S. cerevisiae cells in which this pathway is blocked are more sensitive to oxidative stress and can no longer grow on GABA as their sole nitrogen source.