When optimal sources of nitrogen are unavailable, S. cerevisiae is able to utilize arginine as its sole nitrogen source. Arginine catabolism begins in the cytosol with the hydrolysis of arginine by the Car1p arginaseto form urea and ornithine. Ornithine is then transaminated by the Car2p ornithine amino transferaseinto L-glutamate gamma-semialdehyde, which in turn spontaneously forms L-delta-1-pyrroline-5-carboxylate. Subsequently, p5c is converted into proline by the p5c reductasePro3p. In the absence of oxygen arginine degradation does not proceed further and the pathway is as shown here. If oxygen is present, proline is converted to glutamate via the proline utilization pathwayin the mitochondria.CAR2 gene expression is regulated in a manner very similar to that of CAR1. An upstream repression site in the CAR2 promoter is bound by the global repressor Ume6p, which forms a complex with Sin3p and Rpd3p that downregulates CAR2 expression. This repression is balanced by binding of the global transcriptional activators Abf1p and Rap1p at an upstream activation site. The balance between positive and negative control by these global transcription factors is tipped toward induction when arginine is present and toward repression when it is not. The presence of arginine also induces the binding of the transcriptional activators Arg80p, Arg81p, and Mcm1p. The presence of allophanate, a degradation product of urea, increases CAR2 expression through the two positive regulators, Dal81p and Dal82p. Unlike many of the genes involved in arginine degradation, CAR2 is not sensitive to nitrogen catabolite repression.S. cerevisiae cells deficient in Car2p are unable to grow on ornithine, and presumably arginine, as their sole nitrogen source. In humans, deficiency of the CAR2 homolog, ornithine-delta-aminotransferase, results in the progressive blinding disorder, gyrate atrophy of the choroid and retina.