The ADE2 gene encodes phosphoribosylaminoimidazole carboxylase, which catalyzes the sixth step in the de novo biosynthesis of purine nucleotides. ADE2 transcription is regulated specifically by adenine and also by general amino-acid control. Gene expression is repressed in the presence of adenine, activated in the absence of adenine, and also slightly increased in conditions of amino-acid starvation. Maximal ADE2 expression is dependent on the transcriptional activators Gcn4p, Bas1p, and Pho2p, which bind to 5'-TGACTC-3' sites in the ADE2 promoter. Gcn4p upregulates ADE2 expression during amino acid starvation, a condition which inhibits Gcn4p degradation. Bas1p binds the ADE2 promoter in both repressing and derepressing growth conditions, but binding increases slightly when adenine is limiting. Under derepressing conditions, Pho2p also binds the ADE2 promoter in a Bas1p-dependent manner, and transcription is induced. Derepression by Bas1p and Pho2p also requires the purine biosynthesis pathway intermediates 5-phosphoribosyl-4-succinocarboxamide-5-aminoimidazole and 5-phosphoribosyl-4-carboxamide- 5-aminoimidazole, which are thought to promote interaction between these two activators.Mutations in ADE2 lead to the accumulation of purine precursors in the vacuole, which causes the colony to be red in color. This pigmentation phenotype is widely utilized as a marker for genetic selection and screening.