Transcriptional regulation is an important mechanism for controlling carbon metabolism in Saccharomyces cerevisiae. The Snf1p kinase complex, which phosphorylates serine and threonine residues, is essential for regulating the transcriptional changes associated with glucose derepression through its activation of the transcriptional activators Cat8p and Sip4p, and its deactivation of the transcriptional repressor Mig1p. The complex has also been shown to be involved in multiple processes, including phosphorylation of histone H3; direct regulation of RNA polymerase II holoenzyme; regulation of translation, glycogen biosynthesis, and lipid biosynthesis; and regulation of general stress responses, response to salt stress and response to heat stress. The active Snf1p kinase complex is a heterotrimeric complex composed of Snf1p, the catalyticsubunit; Snf4p, a regulatorysubunit; and one of three possible beta subunitswhich appear to tether Snf1p and Snf4p together and also may determine substrate specificity of the Snf1p kinase complex.The Snf1p kinase complex belongs to a highly conserved family of serine/threonine protein kinases, and homologs to each of the subunitshave been found in all eukaryotes, including plants and mammals.Snf4p appears to activate Snf1p by inhibiting the autoinhibitory interaction of the C-terminusof Snf1p with the catalytic N-terminus of Snf1p, but Snf4p does not influence the phosphorylation state of Snf1p. Snf4p is associated with Snf1p in cells grown in both high and low levels of glucose. SNF4 is not regulated by glucose repression, but binding of Snf4p to Elc1p inhibits the degradation of Snf4p.snf4 null mutants are viable, but are unable to grow on maltose or on non-fermentable carbon sources. As compared to wild type, snf4 null mutants display increased temperature sensitivity, an extended generational life span, a shortened G1 phase and longer S and G2 phases, and decreased production of Cdc28p during growth in glucose-limited cultures, and they also lack peroxisomes.Snf4p has similarity to human PRKAG2, mutations in which are associated with hypertrophic cardiomyopathy and Wolff-Parkinson-White syndrome. Snf4p also contains a region with homology to the known mammalian Elongin C binding proteins, and this region is necessary for the interaction between Snf4p and Elc1p. The growth defects of snf4 null mutants are complemented by Arabidopsis thaliana SNF4 and Candida albicans SNF4.