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.Snf1p is known or predicted to phosphorylate a wide range of substrates, including histone H3Mig1p, Snf1p, Sip1p, Sip2p, Gal83p, Gln3p, Hsf1p, Cat8p, and Sip4p. The kinase activity of Snf1p is under multiple types of regulation. Its N-terminal catalytic domain appears to be autoinhibited by binding to its C-terminal regulatory domain under high-glucose conditions. Under low-glucose conditions, the catalytic domain is bound by Snf4p, which alleviates the autoinhibition from the Snf1p regulatory domain. Std1p has also been shown to enhance the kinase activity of Snf1p. Snf1p is activated by phosphorylation on threonine 210 by either Sak1p, Tos3p, or Elm1p, and is deactivated by the dephosphorylase Glc7p/Reg1p. Although Snf1p is a member of the family of AMP-activated protein kinases, Snf1p is not directly regulated by an AMP signal and SNF1 transcription is not regulated by glucose repression.snf1 null mutants are viable, but are unable to grow on sucrose, galactose, maltose, melibiose or nonfermentable carbon sources. snf1 null mutants also display sporulation defects and don't contain any detectable peroxisomes. Overproduction of Snf1p causes accelerated aging.Snf1p has similarity to human PRKAA2, which is implicated in pancreatic carcinoma and may be an important target for drug development against diabetes, obesity, and other diseases. The growth defects of snf1 null mutants are complemented by tobacco NPK5 and potato StubSNF1.