BCY1 encodes the regulatory subunit of cAMP-dependent protein kinase, the effector kinase of the Ras-cAMP signaling pathway. PKA activity regulates processes involved in cell growth and response to nutrients and stress. In the absence of cAMP, Bcy1p inhibits PKA activity by forming an inactive heterotetrameric complex with the PKA catalytic subunits, in which two regulatory subunits bind to two catalytic subunits. In the presence of cAMP, binding of cAMP to Bcy1p causes its dissociation from the complex as a homodimer, releasing the catalytic subunits as two active monomer. PKA is able to phosphorylate its own regulatory subunit, and the phosphorylation state of Bcy1p has been shown to affect its affinity for the PKA catalytic subunits. Phosphorylation of Bcy1p by other kinases, such as Yak1p and Mck1p, affects its cellular localization. External stimuliresult in these kinases phosphorylating an N-terminal localization domain in Bcy1p, and the phosphorylated form of Bcy1p translocates from the nucleus to the cytoplasm via an interaction with the protein Zds1p. Bcy1p function is independent of its location, but the relocalization of Bcy1p to the cytoplasm may serve to recruit PKA to a specific subset of target proteins. Increased temperature also leads to higher expression of the BCY1 gene. Null mutations in BCY1 lead to constitutive PKA activity, which results in such phenotypes as reduced glycogen accumulation, impaired growth on a variety of carbon sources, temperature sensitivity, and sensitivity to nitrogen starvation. PKA is conserved from yeast to man, and BCY1 homologs have been identified in fission yeast, flies, worms, mice, pigs, cows, and humans. In humans, four regulatory subunit genes falling into two classes, RI and RII, have been identified, and Bcy1p is structurally and functionally similar to the mammalian RII class of PKA regulators.