Snf12p is a component of SWI/SNF, and is required for maintaining the full structural integrity of the complex. Snf12p binds to sequences in the activation domain of transcriptional activator Gcn4p, which contributes to the ability of Gcn4p to recruit the entire SWI/SNF complex to gene promoters. The requirement for Snf12p in assisting transcriptional activation appears to depend on both the activator protein and promoter involved. snf12 null mutants are viable, but temperature-sensitive, and also display defects in HO and HIS3 transcription, decreased structural integrity of the SWI/SNF complex, defective binding of the SWI/SNF complex to SNZ1 promoters, and hypersensitivity to 3-aminotriazole. Snf12p is similar to Rsc6p and human SMARCD2, and homologs are also present in Ashbya gossypii, Caenorhabditis elegans, and mice, indicating that Snf12p may belong to a family of related genes encoding proteins with analogous functions.By regulating the structure of chromatin, chromatin remodeling complexes, all of which contain an ATPase as a central motor subunit, perform critical functions in the maintenance, transmission, and expression of eukaryotic genomes. The SWI/SNF chromatin remodeling complex is involved in DNA replication, stress response, and transcription, and binds DNA nonspecifically, altering nucleosome structure to facilitate binding of transcription factors. For some genes, transcriptional activators are able to target the SWI/SNF complex to upstream activation sequencesin the promoter. The SWI/SNF chromatin remodeling complex family contains two evolutionary conserved subclasses of chromatin remodeling factors, one subfamily includes yeast SWI/SNF, fly BAP, and mammalian BAF, and the other subfamily includes yeast RSC, fly PBAP, and mammalian PBAF. It appears that some human SWI/SNF subunits act as tumor suppressors and there is also evidence that human SWI/SNF subunits are involved in controlling cell growth via their interaction with other tumor suppressors. Expression of adenovirus E1A oncoproteins, which are regulators of cellular and viral transcription, in Saccharomyces cerevisiae requires the function of the SWI/SNF complex, and expression of E1A in wild-type cells leads to a specific loss of SWI/SNF dependent transcription. These results suggest that the SWI/SNF complex is a target of these oncoproteins in mammalian cells and that the disruption of normal cell cycle control by E1A may be due in part to altered activity of the SWI/SNF complex.