Snf6p is a component of SWI/SNF, present at two copies per complex. Expression of Snf6p appears to be constitutive, and the protein is required for maintaining the full structural integrity of SWI/SNF. Snf6p can bind either free or nucleosomal DNA, and is involved in promoting the DNA-binding of SWI/SNF. Snf6p thereby affects transcription at a variety of promoters, and may be involved in negative regulation of chromatin silencing. snf6 null mutants are viable but defective in the derepression of SUC2 transcription, cannot grow anaerobically on raffinose and galactose, or aerobically on glycerol, display impaired association between myc-Snf2p and Snf5p, and also show increased resistance to cisplatin, one of the most widely-used anticancer drugs. Homozygous diploid snf6 null mutants fail to sporulate and display a low budding index. Phenotypes of snf6 nulls are partially suppressed by cyc8 mutations.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.