Snf11p is a component of the SWI/SNF complex, and can function as a transcriptional activator in genetic assays. Two copies of Snf11p occur per SWI/SNF complex. Snf11p interacts with an evolutionarily-conserved 40-residue sequence in Snf2p, suggesting that Snf11p homologs may exist in other organisms. snf11 null mutants are viable, but display hypersensitivity to calcium and to calcofluor white. snf11 nulls also display reduced phosphate levels in the mannoproteins on the cell wall surface, and homozygous diploid deletion strains display a high budding index.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.