Swi1p is a component of the SWI/SNF complex, binds promoter activation domains, and is required for transcription of a diverse set of genes, including HO and Ty retrotransposons. Swi1p is also required for normal growth and initiation of meiosis. swi1 null mutants are viable, but display sporulation defects, slow growth, decreased levels of Adh2p, and decreased heat-shock induction of HSP82. swi1 mutants are also defective in mating-type switching, are highly sensitive to gamma-rays, and display increased rates of spontaneous oligomycin resistance resulting from mutations in the mitochondrial DNA. swi1 mutations are supressed by spt2 mutations, and are synthetically lethal in combination with dst1 null mutations. Swi1p is a member of a family of DNA-binding proteins that includes Drosophila melanogaster DRI, human retinol-binding proteins RBP1 and RBP2, human SMCX, which is associated with X-linked mental retardation, and mouse Jumonji, which is required for neural tube formation. Swi1p is also similar to human ARID1A, and Hansenula polymorpha and Candida albicans Swi1p. Swi1p also contains several regions which are similar to D. melanogaster Engrailed.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.