Arp7p and Arp9p are nuclear actin-related proteins that form a stable heterodimer which is an essential component of both the SWI/SNF and RSC chromatin remodeling complexes. The C-termini of Arp7p and Arp9p are both required for association of the Arp7p/Arp9p heterodimer with the RSC complex. Genetic analyses indicate that the Arp7p/Arp9p heterodimers may also cooperate with Nhp6ap and Nhp6bp to facilitate proper chromatin architecture. Depending on the genetic background tested, arp7 and arp9 null mutants are each either inviable or show greatly impaired growth with mutant phenotypes similar to those seen in snf2 nulls, such as an inability to grow on non-fermentable carbon sources. The temperature sensitivity of either arp7 or arp9 conditional mutants is suppressed by the overproduction of Nhp6ap.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.