

Human papillomavirus-16 E7 protein inhibits the DNA interaction of the TATA binding transcription factor

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Previous studies have shown that the HPV-16 E7 protein interacts with TBP. This interaction was found to take place through residues in the carboxy terminal half of E7, mutation of which resulted in weaker transforming activity. In addition, binding of E7 to TBP was found to be increased following protein kinase CK2 (casein kinase II) phosphorylation of E7, and mutation of this CK2 site also reduces E7's transforming activity. To date, however, there is no information on the effects of E7 upon TBP function. In order to address this we have performed a series of assays to investigate the effects of E7 upon the ability of human and *S. pombe* TBP to bind DNA. We show that HPV-16 E7 is indeed a potent inhibitor of TBP DNA binding activity. Further, this activity of E7 is increased following CK2 phosphorylation of E7, consistent with it having an increased affinity for TBP. Finally, a mutant E7 protein defective in its ability to bind TBP, has no effect upon TBP binding to DNA.

These result