The human papillomavirus type 18 E6 oncoprotein induces Vascular Endothelial Growth Factor 121 (VEGF121) transcription from the promoter through a p53-independent mechanism

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Résumé en anglais	Altered angiogenic response is associated with high-grade cervical dysplasia and with invasive squamous carcinoma of the cervix. Vascular Endothelial Growth Factor (VEGF) is one of the most potent inducers of angiogenesis and is up-regulated in carcinoma of the cervix. Infection by high-risk human papillomavirus and persistent expression of viral oncogene E6 are etiologically linked to the development of cervical cancer. E6 is able to immortalize cells and induce malignant transformation by inactivating p53. In cervical cancer, regulation of VEGF expression is poorly described. Thus, we investigated whether E6 oncoprotein could regulate VEGF expression in HPV18-positive cervical cancer-derived HeLa cells harboring a wild-type p53. The alternative splicing of vegf mRNA renders three major isoforms of 121, 165 and 189 amino-acids in humans. We have designed isoform specific real time QRT-PCR assays to quantitate vegf transcripts and VEGF121 was the predominant isoform. Silencing HPV18 E6 mRNA with specific siRNA reduced VEGF121 expression by at least 50% whereas silencing of p53 did not alter its expression. Treatment with cycloheximide did not inhibit E6-induced VEGF121 expression. Collectively, these results suggest that HPV18 E6 oncoprotein contributes to tumor angiogenesis by inducing VEGF transcription from the promoter in a p53-independent manner.
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