Functional interaction between human papillomavirus type 16 E6 and E7 oncoproteins and cigarette smoke components in lung epithelial cells

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The smoking habit is the most important, but not a sufficient cause for lung cancer development. Several studies have reported the human papillomavirus type 16 (HPV16) presence and E6 and E7 transcripts expression in lung carcinoma cases from different geographical regions. The possible interaction between HPV infection and smoke carcinogens, however, remains unclear. In this study we address a potential cooperation between tobacco smoke and HPV16 E6 and E7 oncoproteins for alterations in proliferative and tumorigenic properties of lung epithelial cells. A549 (alveolar, tumoral) and BEAS-2B (bronchial, non-tumoral) cell lines were stably transfected with recombinant pLXSN vectors expressing HPV16 E6 and E7 oncoproteins and exposed to cigarette smoke condensate (CSC) at different concentrations. HPV16 E6 and E7 expression was associated with loss of p53 stability, telomerase (hTERT) and p16INK4A overexpression in BEAS-2B cells as demonstrated by quantitative real-time polymerase chain r