Tobacco smoke activates human papillomavirus 16 p97 promoter and cooperates with high-risk E6/E7 for oxidative DNA damage in lung cells

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© 2015 Peña et al. We have previously shown a functional interaction between human papillomavirus type 16 (HPV-16) E6 and E7 oncoproteins and cigarette smoke condensate (CSC) in lung cells suggesting cooperation during carcinogenesis. The molecular mechanisms of such interaction, however, remain to be elucidated. Here we first present evidence showing that cigarette smoke condensate (CSC) has the ability to activate the HPV-16 p97 promoter by acting on the long control region (LCR) in lung epithelial cells. Interestingly, we observed that CSC-induced p97 promoter activation occurs in a dose-dependent manner in both tumor A-549 (lung adenocarcinoma), H-2170 (bronchial carcinoma), SiHa or Hela (cervical carcinoma) cells but not in non-tumor BEAS-2B (bronchial) or NL-20 (alveolar) lung cells unless they ectopically expressed the HPV-16 E6 and E7 oncogenes. In addition, we also observed a significant increase of primary DNA damage in tumor and non-tumor CSC-treated lung cells expressing HP