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Importance of HPV-16 E1 expression in cervical carcinogenesis

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Statement of the Problem: High-risk human papillomavirus (HPV) infections cause transformation of the host cells and cervical carcinoma by down-regulating and inhibiting host regulatory proteins such as p53 and pRb by overexpressing the viral oncoproteins E6 and E7. However, the E1 protein which is the only enzyme encoded by HPV has also been shown to cause DNA instability leading to integration of the virus into the host genome and triggering carcinogenic events. The purpose of this study was to determine the expression level of E1 in HPV-16 positive patients across all clinical stages.

Methodology & Theoretical Orientation: Total RNA was extracted from 39 HPV-16 positive patients across normal, precancerous and cancer stages of cervical carcinoma. Patient RNA samples were then subjected to cDNA synthesis. HPV-16 E1 mRNA level was detected for each patient by droplet digital PCR.

Findings: A significant increase in E1 expression in correlation with disease progression between SCC samples and normal samples (p<0.05) and CIN 2/3 and normal samples (p<0.01) was found. The average relative E1 mRNA level in clinical samples was 0.18 for normal, 0.41 for CIN 1, 0.65 for CIN 2/3 and 0.79 for SCC.

Conclusion & Significance: In this study, we characterized the expression of E1 mRNA in clinical samples ranging from normal, precancerous and cancer stages. We were able to determine that E1 is expressed at significantly different levels in normal patients and cancer patients. Further studies in a larger cohort, as well as the functional roles of E1 are warranted, and could lead to targeted treatment of HPV16 infection. Collectively, we have characterized, for the first time, the expression profile of HPV16-E1 in different

stages of cervical cancer disease progression.

Biography

Fern Baedyananda is a PhD candidate at Chulalongkorn University. She is extremely passionate about the causes of oncogenesis, especially ones of viral origin. She has been researching HPV since the beginning of her PhD and currently on antiviral drugs, especially ones that can target HPV, however the mechanism in which HPV causes carcinogenesis is not fully understood. Using patient cervical samples, she has constructed a new model around a viral oncoprotein that could prove to be a promising target for therapy.

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