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# Sequence Analysis of the Effect of HPV-16 E1 Variation on Cervical Carcinogenesis

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**Abstract :** High-risk human papillomavirus (HPV) infections cause transformation of the host cells 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 the integration of the virus into the host genome and triggering carcinogenic events. A 63bp duplication in the E1 helicase region has been detected in European patients. However, the clinical prognosis of these patients is still controversial. This study was performed to determine the presence of the HPV-16 E1 63bp duplication in patient cervical samples in Thai women and determine the sequence of the variant in the Thai population. Detection of the HPV-16 E1 duplication in the helicase region was performed in 90 patient cell samples across normal, cervical intraepithelial neoplasia I-III, and squamous cervical carcinoma stages by PCR. The PCR products were purified and sequenced to determine the presence of duplication variants.The variant form was found in 10% of all CIN 1 patients. In this study, the presence of the 63 bp duplication variant in the Thai population was found to be present and was further characterized. Interestingly, all samples that exhibited the variant form of HPV-16 E1 were classified as CIN I. Presence of the variant, constricted to mild dysplasia signifies the importance of HPV-16 E1 in carcinogenesis.

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