OPINION

hnRNP E1 controls the expression of the *HPV16* oncogene and prevents cervical cancer

Sander Gliboff

Gliboff S. *hnRNP E1* controls the expression of the *HPV16* oncogene and prevents cervical cancer J. Clin. Genet. Genom. 2022;5(2):1-2.

ABSTRACT

Heterogeneous nuclear ribonucleoprotein *E1*, also known as *hnRNP E1*, is an essential RNA-Binding Protein (RBP) that is essential for the growth of tumors. The development of HPV-associated malignancy and the expression of the HPV gene are both influenced by the presence of several RNA/DNA binding sites and various RBPs on the *Human papillomavirus 16* (*HPV16*). It is yet unknown how *hnRNP E1* affects *HPV16* oncogenes in the growth of cervical lesions. A community-based cohort was established in Shanxi Province, China, with a total of 816 participants who had cervical lesions of various grades. The relationship between the expression of *hnRNP E1* mRNA and cervical lesions was examined using data from the Gene Expression Omnibus (GEO) and The Cancer Genome Atlas (TCGA) databases. We created cells that have *hnRNP E1* up- and down-regulated. Cell counting kit-8, analyses from flow cytometry, and chromatin immunoprecipitation sequencing were all used to assess *hnRNP E1* functions. Our findings demonstrated that the degree of the cervical lesions had a linear relationship with *hnRNP E1* expression. Cervical lesions could be more likely to develop in those with low expression of *HPV16 E2*, high expression of *E6*, and a low ratio of *E2* to *E6*. *HPV16* oncogene expression and *hnRNP E1* expression were associated. Dopaminergic synapses, the Wnt signalling system, gnRH secretion, and the mTOR signalling pathway were all impacted by *hnRNP E1*-relevant genes. Significantly reducing *HPV16 E6* expression, inducing apoptosis, arresting the cell cycle at the G0/G1 stage, and inhibiting cell proliferation were all effects of *hnRNP E1*. Our findings suggest that *hnRNP E1* could suppress the expression of the *HPV16 E6* oncogene and prevent cervical cancer development, which provides new insight into how to stop the carcinogenicity of HPV across a variety of diseases by controlling RNA-binding proteins.

Key Words: *Malignancy; Immunoprecipitation; Chromatin; Carcinogenicity; Papillomavirus; Ribonucleoprotein*

INTRODUCTION

The fourth most common kind of cancer among women diagnosed worldwide is cervical cancer. Cervical cancer incidence in China has considerably grown, in contrast to declining patterns in Western nations. In China, it is anticipated that there will be 0.06 million fatalities and 0.11 million new cases by 2020. With 5.42 linked deaths per 100,000 women in 2014, Shanxi Province in China has a notably high incidence of cervical cancer cases, more than twice the national norm. More than 50% of HPV cases are caused by high-risk persistent HPV infections, particularly *HPV16* infections, which are crucial in the development of cervical cancer. The *HPV16* genome, which is made up of 8000 base pairs of double-stranded circular DNA, can be broken down into early genes (*E1, E2, E4, E5, E6,* and *E7*), late genes (*L1 and L2*), and long regulatory sections. An essential carcinogenic protein is *HPV16 E6*, which promotes and maintains

Editorial Office, Journal of Clinical Genetics and Genomics, UK

Correspondence: Sander Gliboff , Editorial Office, Journal of Clinical Genetics and Genomics, UK , Email: sandergliboff@gmail.com

Received:6 April, 2022, Manuscript No. PULJCGG-22-5786; Editor assigned:8 April 2022, Pre-QC No. PULJCGG-22-5786 (PQ); Reviewed:- 12 April 2022, QC No. PULJCGG-22-5786 (Q); Revised:15 April 2022; Manuscript No. PULJCGG-22-5786 (R); Published:-20 April 2022, DOI: 10.37532. puljcgg.22.5(2)1-2

cellular transformation. Particularly, *E6* and *E7* expressions are required to promote cell proliferation in infected tissues as well as the formation of high-grade lesions and cancer. The primary inhibitor of the production of the oncogenes *E6* and *E7* is thought to *be HPV-16 E2*. A crucial step in cervical carcinogenesis is the integration of the *HPV16* genome into host chromosomes, which typically results in disruption of *E2*, loss of *E6* regulation, and subsequent *E6* overexpression. Due to their significance in HPV integration and carcinogenesis, *E2* and *E6* have received a lot of attention as crucial genes for integration. However, RNA processing within cells, which is often altered by RNA binding proteins, is necessary for the regulation of HPV gene expression (*RBPs*). Heterogeneous nuclear ribonucleoproteins (*hnRNPs*), which act as RBPs, play a variety of roles in the metabolism of nucleic acids. The *hnRNP E1* gene encodes a protein with three K homology (KH) domains. To increase the specificity and affinity of RNA/DNA binding, it is necessary. Early promoter p97 and late promoter p670 are the two distinctive promoters of *HPV-16*. The *HPV16* genome also has two polyadenylation sites, three 5′ splice sites, and two 3′ splice sites. These locations offer the structural underpinnings for targeted binding to several RBPs and associated protein complexes. According to studies, *hnRNP* E1 is implicated in a number of pathogenic disorders. RBPs, also known as heterogeneous nuclear ribonucleoproteins (*hnRNPs*), have a range of functions in the metabolism of nucleic acids. Three K homology (KH) domains are present in the protein that the *hnRNP E1* gene encodes. It is required to boost the RNA/DNA binding's specificity and affinities. The two different promoters of *HPV16* are early promoter p97 and late promoter p670. Three 5′ splice sites, two 3′ splice sites, and two polyadenylation sites are also present in the *HPV16* genome. These sites provide the structural basis for specific binding to various RBPs and related protein complexes. Studies have shown that *hnRNP E1* is connected to a number of pathogenic diseases.

We predicted that *hnRNP E1* may be important for *HPV16* oncogene expression and cervical carcinogenesis based on the specificity with which the distinctive KH structural domains of *hnRNP* E1 connect with RNA or DNA and taking into account that *HPV16* provides RNA or DNA binding sites. Our earlier population-based findings demonstrated a strong correlation between the development of cervical cancer and *hnRNP E1, HPV16 E2,* and *E6*. In the current work, we examined the variations in *hnRNP E1* expression at various cervical pathological stages. We further investigated *hnRNP E1*'s probable role and mechanism in cervical lesions *in vitro*. Our findings might serve as a starting point for identifying new *HPV16* molecular targets as well as prognostic and predictive biomarkers for cervical cancer.