

## Investigation of Tumor Suppressor miR-383 Expression in HPV-related Malignancies

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### Research Article

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### ABSTRACT

Although genetic changes were investigated in disease development previously, we now know that epigenetic changes also play a critical role in diseases. In addition to DNA methylation and histone modification, changes in non-coding RNA expression also contribute to pathological processes as epigenetic modulators. miRNAs are conserved molecules that bind complementarily to their encoded target mRNAs, thereby performing post-transcriptional regulation. MicroRNA-383 (miR-383) functions as a tumor suppressor in various cancers, but its expression pattern in HPV-associated cervical lesions remains unclear. This exploratory study aimed to investigate whether miR-383 expression is altered in HPV-positive cervical tissues and to generate hypotheses for future mechanistic studies. In this study, miR-383 expression levels were analyzed using qRT-PCR in paired HPV-positive cervical tissue and adjacent normal tissue samples from 30 patients. Our preliminary findings suggest a potential trend toward miR-383 upregulation, warranting further validation in larger cohorts. The observed upregulation may reflect a compensatory host response against viral oncogenesis, potentially through miR-383-mediated repression of oncogenic targets such as SFN and PD-L1, suggesting its role in restraining early cervical carcinogenesis and highlighting its potential as a biomarker in HPV-related disease progression.



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## 1. Introduction

HPV (Human Papillomavirus) is a sexually transmitted infection that can cause cancer and precancerous lesions. [1] Cervical cancer (CC), which is the most common HPV-related cancer, is the fourth most frequently diagnosed and leads to high mortality in women, second only to breast cancer [2]. The pathogenesis is largely mediated by the viral oncoproteins E6 and E7, which disrupt key cellular mechanisms, leading to genomic instability and unchecked proliferation [3]. Emerging evidence highlights the involvement of microRNAs (miRNAs) in HPV-induced oncogenesis, where the aberrant expression of specific miRNAs can promote tumor growth, evade immune detection, and reduce treatment efficacy [4].

MicroRNAs (miRNAs), are single-stranded RNA molecules consisting of 20-23 nucleotides. These molecules, belonging to the small non-coding RNA family, regulate gene expression post-transcriptionally [5]. miRNAs bind to target mRNA, either degrading it or repressing translation. microRNA (miRNA) dysregulation on the pathogenesis and progression of cervical cancer, primarily driven by persistent high-risk human papillomavirus (HR-HPV) infection. Key miRNAs, including miR-21, miR-9, miR-34a, and miR-29, are highlighted for their distinct roles in tumorigenesis. MiR-21 is consistently upregulated in HPV-positive lesions and cervical tumors, where it promotes cancer progression by targeting tumor suppressors like PTEN and PDCD4, thereby enhancing cell proliferation, immune evasion,

and inflammation via the NF- $\kappa$ B pathway, solidifying its potential as a diagnostic and prognostic biomarker [6, 7]. The function of miR-9 is context-dependent, acting as an *oncomiR* in squamous cell carcinoma (SCC) by suppressing TWIST1 and CDH1 to induce epithelial-mesenchymal transition (EMT) and invasion, while in adenocarcinoma (AC), downregulation of miR-9 facilitates EMT via CDH2 upregulation, suggesting a tumor-suppressive role [8]. In contrast, miR-34a and miR-29 are frequently downregulated in cervical carcinogenesis. MiR-34a suppression, mediated by HPV E6-induced p53 degradation, leads to increased cell proliferation and invasion through the WNT1/ $\beta$ -catenin pathway [9, 10]. MiR-29a acts as a tumor suppressor by targeting genes like SIRT1 and HSP47, inhibiting migration, invasion, and EMT, with its loss contributing to metastasis [11, 12]. While these miRNAs offer promising avenues as biomarkers for early detection and potential therapeutic targets significant challenges remain. These include methodological standardization in miRNA detection, accounting for patient heterogeneity, and overcoming delivery and specificity hurdles for miRNA-based therapies. Therefore, miRNAs are poised to become valuable complementary tools in cervical cancer diagnosis and treatment, but require further validation and integration with conventional clinical practices [13, 14].

MiR-383 functions as a tumor inhibitor miRNA and downregulation of miR-383 is a common characteristic of

many types of cancer [4]. Recent study suggests that miR-383 expression is implicated in the pathogenesis and in predicting molecular progression, cellular proliferation, and patient prognosis in various carcinomas. Reduced levels of miR-383 are associated with diminished prognostic outlooks. In vitro studies demonstrate that miR-383 mediates its anti-tumor activity by binding to specific proteins and molecules, thereby influencing critical signaling pathways in cancer cells [4]. Although there are no studies investigating changes in miR383 expression in patients with HPV infection, it has been studied in many types of cancer.

In cervical cancer, miR-383 functions as a crucial tumor suppressor, with its expression significantly downregulated in cancerous tissues and cell lines [15]. Reduction in miR-383 levels is associated with poorer patient prognosis. Its tumor-suppressive function is primarily inhibited by the long non-coding RNA LINC01128, which binds to and "sponges" miR-383-5p. This interaction leads to the increased expression of the oncogenic protein Stratifin (SFN), a direct target of miR-383-5p, thereby promoting cancer progression. Consequently, restoring miR-383 expression presents a promising potential therapeutic strategy for cervical cancer [15].

To date, no study has examined miR-383 expression specifically in HPV-associated cervical lesions. Therefore, we conducted an exploratory analysis to address this gap in the literature. Given the lack of prior evidence, the present work was designed as a hypothesis-generating investigation rather than a confirmatory trial. Accordingly, miR-383 expression was compared between healthy and diseased tissues from HPV-positive individuals. The primary aim was to evaluate whether miR-383 expression levels could contribute to risk classification and diagnosis.

## 2. Materials and Methods

### 2.1. Collection and Storage of Samples

Within the scope of study, diseased and healthy samples taken from HPV-positive women by conization method were used. In this study, 30 individuals were recruited. The inclusion criteria was positivity for at least one HPV type of the individuals in the patient group. The HPV genotypes of the patients range from HPV-16, HPV-18, HPV-31, HPV-39, HPV-51, HPV-52, HPV-66, and HPV-68. Some patients have infections with more than one genotype. The study group consisted of women who applied to Sivas Cumhuriyet University Hospital Department of Gynecology and Obstetrics for routine screening. Ethics committee approval was obtained for the samples of patient to be used in the study (File number: 2019-10/01).

The severity of abnormal cellular changes in cervical lesions is determined by a more detailed examination

called colposcopy. One of the methods used to treat precancerous lesions or CINs detected by colposcopy is conization. Conization is the process of removing a portion of the cervix under local or general anesthesia. The tissue samples for our study consisted of biopsy samples obtained from suitable patients using the cone biopsy (conization) method. Accordingly, healthy and diseased tissues from the same individual were placed in Eppendorf tubes containing RiboSaver (GeneAll, Cat No: 351-001), a protective fluid for genetic material RNA, and stored at -80°C until the experiments.

### 2.2. RNA isolation and qRT-PCR

Isolation of total RNA containing miRNA from diseased and healthy tissue samples taken from the same individual and preserved in RiboSaver was performed using the Tissue Total RNA Isolation Protocol of the Total RNA Kit (EcoPURE, Cat No: E2075). The protocol is suitable for fresh or frozen tissue samples up to 30 mg, and since the collected sample amounts did not exceed 30 mg, the protocol was applied exactly as described. Accordingly, after the tissue samples were fragmented with a scalpel, 400 µl of EcoPURE Lysis/Binding Buffer was immediately added to the fragmented tissue samples using an appropriate amount of liquid nitrogen. The homogenized mixture was transferred to 1.5 ml RNase-free tubes and vortexed for 10-15 seconds. To separate cellular debris and the pellet, the lysate was centrifuged at maximum speed for 2 minutes at room temperature. The supernatant was transferred to a new RNase-free tube and its quantity was noted. An equal volume of absolute EtOH was added to the supernatant and vortexed for 10 seconds. The subsequent steps were the same as the protocol applied to blood and smear samples. The resulting total RNA containing miRNA was dissolved in 80 µl of EcoPURE Elution Buffer and stored at -80°C until the next experimental step.

RNA concentration was measured using Qubit 3.0 Fluorometer and concentrations were equalized to 100 ng for cDNA synthesis. For expression level analysis, cDNA synthesis was performed using the VitaScript FirstStrand cDNA Synthesis Kit (Procomcure). PCR reactions were performed using specific primers GAPDH (gene ID:2597) as reference genes in accordance with the 2x Magic SYBR Green Mix (Procomcure) protocol. GAPDH primer sequences are F: 5'-ATGTTCCAATATGATTCCACCC-3' and R: 5'-ATGAGTCCTTCCACGATACC-3'. miR-383 primer sequences are F: 5'-CTTCCCAAGAGTTTCACT-3' and R: 5'-CCACTCCAGTCCACCAAAT-3'. The RT-PCR conditions for three-step amplification reaction were determined as follows: denaturation at 95°C for 5 minutes, 40 cycles at 95°C for 10 seconds, 55-60°C for 15 seconds and 72°C for 20 seconds, and final extension 72°C for 2 minutes.

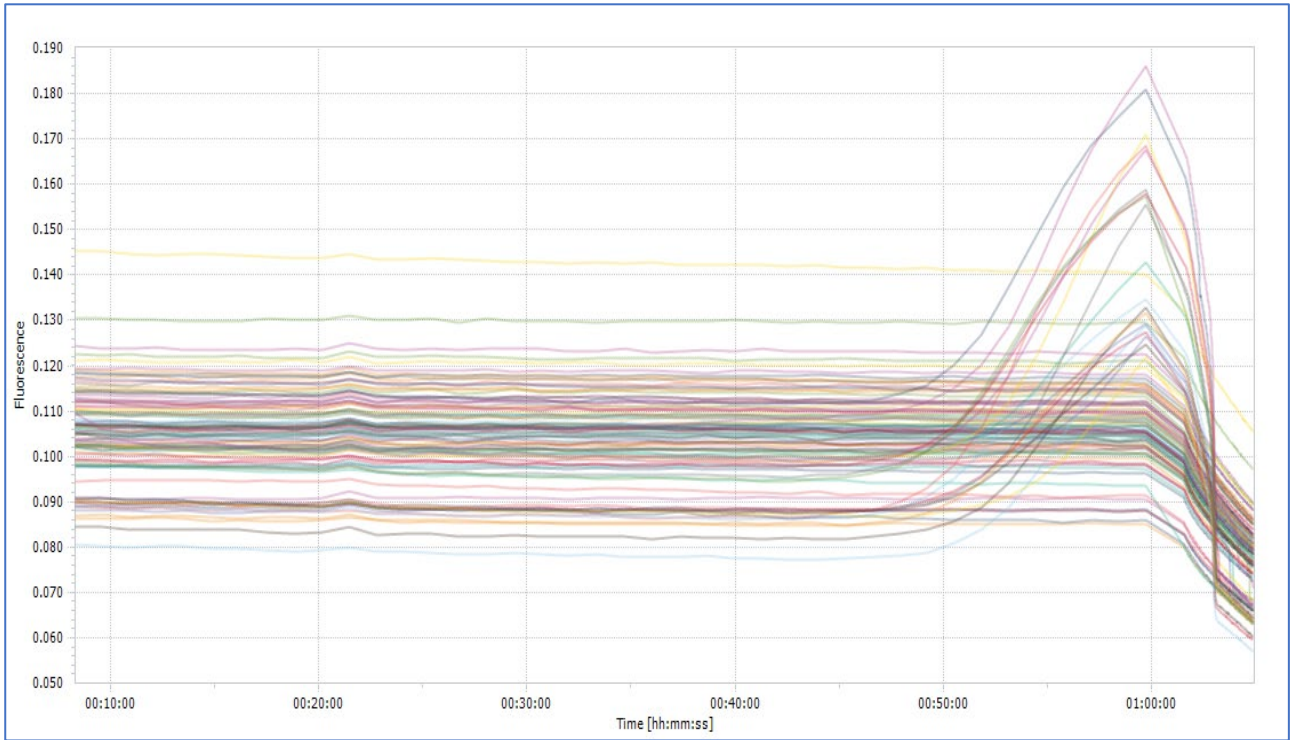


Figure 1: Cq values obtained from qPCR results.

### 2.3. Statistical Analysis

The raw data obtained as a result of RT-PCR experiments were analyzed using  $\Delta\Delta CT$  method and student t-test ( $p < 0.05$ ) was used for statistical significance.

### 3. Results and Discussion

The patient group of our study consisted diseased and neighboring healthy tissue samples taken from HPV-positive individuals by conization method were also examined.

Patients were evaluated according to their educational level, place of residence, smoking and oral contraceptive use and CIN grades. All individuals included in our study are married, have children, and have no family history of cervical cancer. The demographic and clinical data of our study group are summarized in Table 1.

miR-383 expression levels in HPV samples were determined by qPCR analysis. Details of the Cq peaks from the RT-PCR results are given in Figure 1. Statistical analysis of the data was performed using the  $\Delta\Delta CT$  method with the “miScript miRNA PCR Data Analysis” and “RT2 Profiler PCR Data Analysis” software (<https://geneglobe.qiagen.com/tr/analyze>).

Table 1: Demographic and clinical data of patient

	Cases (n=30)
Age (mean $\pm$ SD)	39.74 $\pm$ 7.17
Educational level n (%)	
Primary school	11 (36,66)
Secondary school	8 (26,67)
High school	8 (26,67)
University	3 (10)
Place of residence, n (%)	
City center	24 (80)
County	6 (20)
Employment status, n (%)	
Employed	5 (16,67)
Not employed	25 (83,33)
Smoker, n (%)	
Absent	21 (70)
Present	9 (30)
Oral contraceptive use, n (%)	
Absent	28 (93,33)
Present	2 (6,67)
CIN grade, n (%)	
CIN 1	14 (46,67)
CIN 2	13 (43,33)
CIN 3	3 (10)

An increase in miR-383 expression was observed in HPV tissue samples (fold change=1.38, p=0.463273) (Table 2). Fold change values are shown in Figure 2, details with Average  $\Delta Cq$  in Figure 3, and finally details for  $2^{-\Delta Cq}$  in Figure 4.

Table 2. Fold regulation, fold change, and p-value showing the change in MiR383 expression levels according to qPCR analysis results in the study sample.

miRNA	Fold Regulation	Fold Change	p-Value
Mir383	1.38	1.38	0.463273

\*P<0.05 was considered statistically significant.

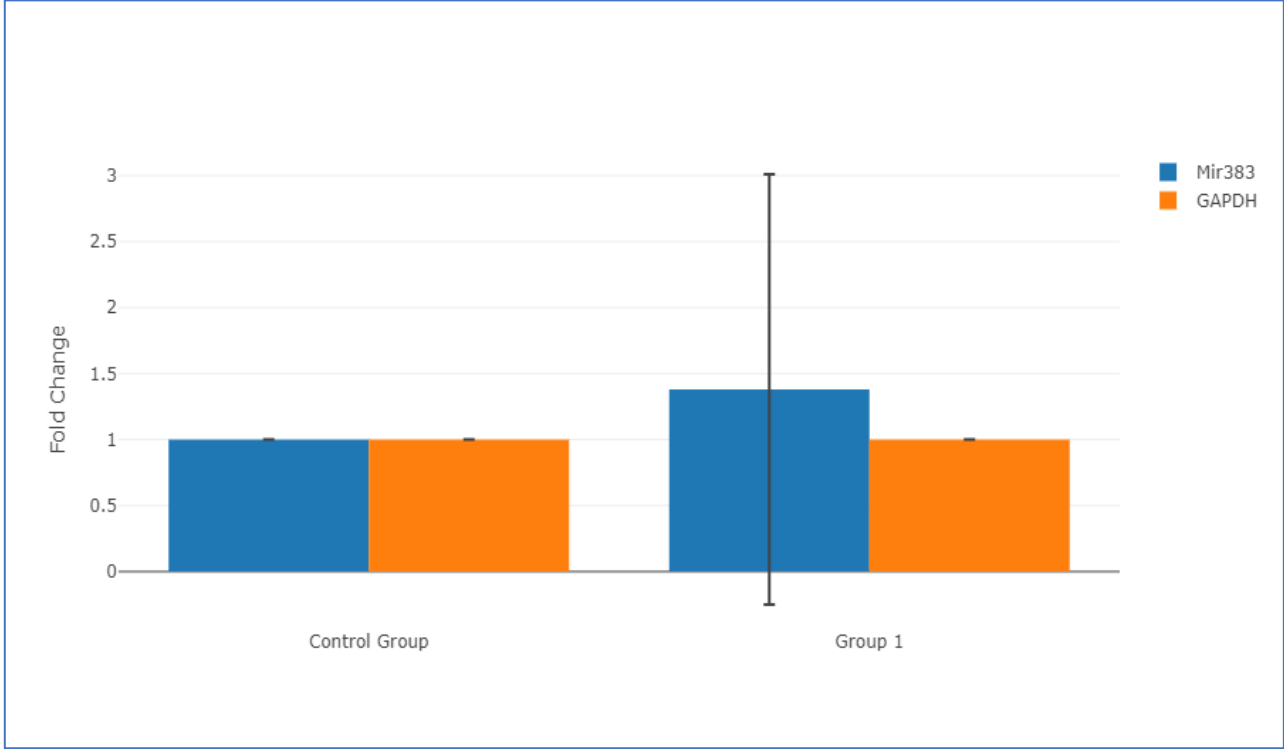


Figure 2. Comparison of fold change values of miR-383 in HPV-affected and healthy tissues.

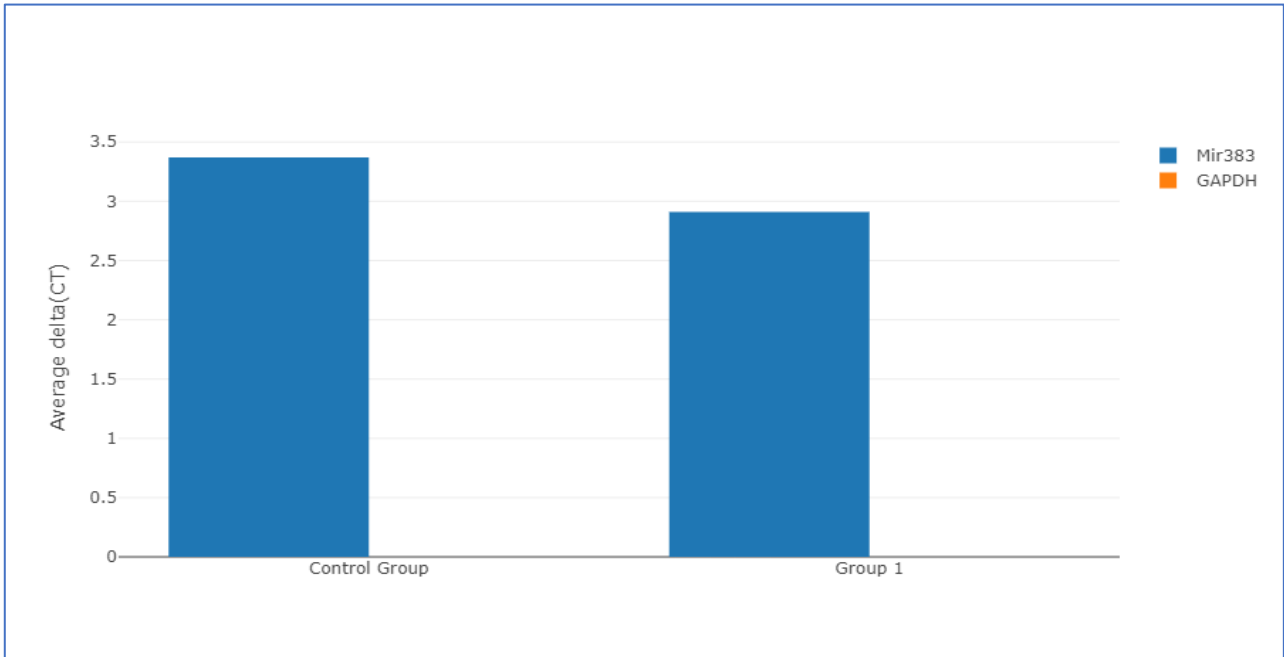


Figure 3. Comparison of miR-383 average  $\Delta Cq$  values in HPV-affected and healthy tissues.

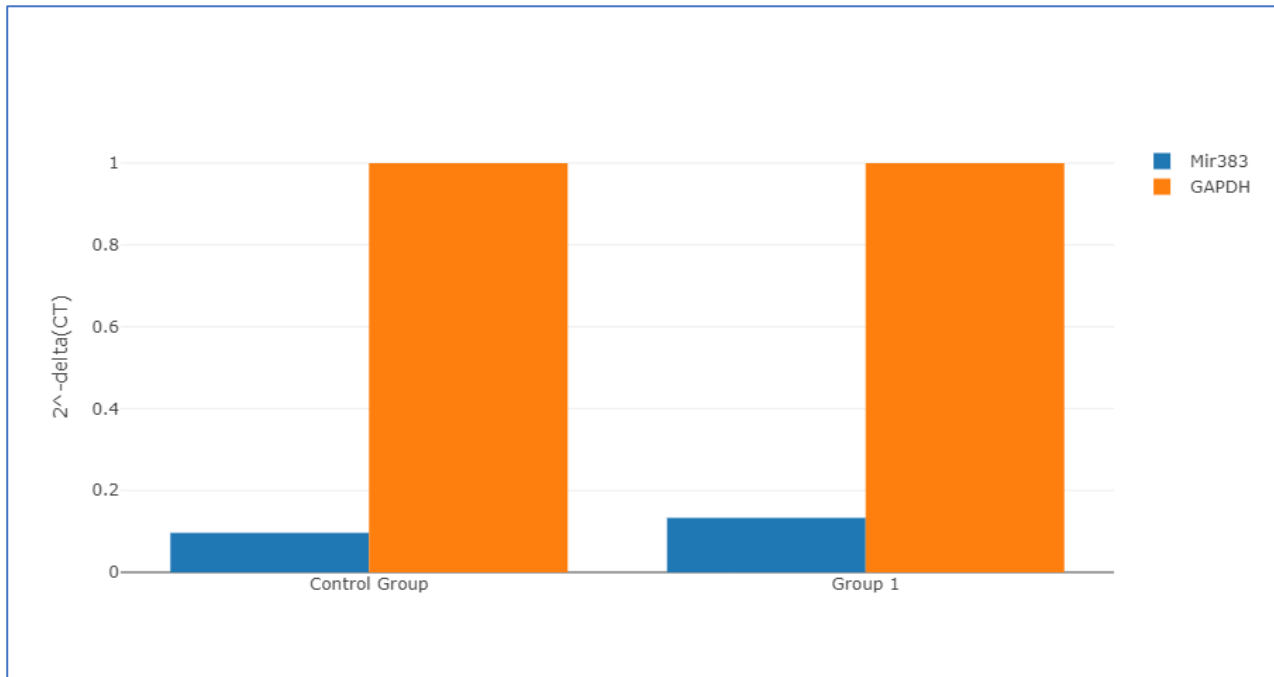


Figure 4. Comparison of  $2^{-\Delta\Delta Cq}$  values of miR-383 in HPV-infected and healthy tissues.

### 3.1 Discussion

Cervical cancer is a major health problem for women worldwide. It is largely caused by HPV infections and has a high mortality rate. Not all HPV infections lead to cancer, and understanding the pathogenesis of how cancer develops is critical. Therefore, we investigated the expression of miR-383, involved in epigenetic cancer-preventive pathway, in HPV-positive individuals. To our knowledge, this is one of the first studies to report miR-383 expression levels specifically in HPV-positive patients. As an exploratory study, our findings should be interpreted as hypothesis-generating. The observed upregulation of miR-383 (fold change = 1.38,  $p = 0.463$ ), although not statistically significant, provides preliminary evidence for a potential host response against HPV-induced oncogenesis. However, due to the limited size of our study group ( $n=30$ ), this difference was not statistically significant. The absence of a significant difference may also reflect the early disease stage of our cohort (predominantly CIN 1–2), as miR-383 downregulation might occur only in advanced carcinomas.

Analysis of the study group's clinical and demographic characteristics showed that some participants presented with multiple HPV infections. Comparison of gene expression levels based on HPV type indicated no statistically significant effect of multiple infections relative to single-type infections. This observation aligns with epidemiological data suggesting that co-infections do not synergistically elevate the risk of cancer development [16]. Additionally, factors including education level, occupation, and geographic location did not reveal any significant differences between the patient cohort.

MicroRNAs (miRNAs) play a critical role in the initiation and progression of cervical cancer (CC) by acting as key regulators of gene expression. In CC, the persistent infection with high-risk human papillomavirus (HR-HPV)

disrupts the normal miRNA profile, leading to the dysregulation of oncogenic and tumor-suppressive miRNAs. For example, miR-34a, a direct transcriptional target of p53, is frequently downregulated in HPV-positive lesions due to E6-mediated p53 degradation, which promotes cell cycle progression and inhibits apoptosis [17, 18]. Similarly, miR-218 is suppressed in CC, resulting in increased expression of its target LAMB3 and enhanced cell migration and invasion [19]. Other miRNAs such as miR-100 are significantly reduced in cervical tumors, leading to overexpression of PLK1 and accelerated cell proliferation. Furthermore, miR-199a and miR-9 are markedly upregulated in CC tissues and are associated with lymph node metastasis and poor prognosis [20, 21]. Collectively, these miRNA alterations contribute to hallmark cancer processes—including sustained proliferation, evasion of apoptosis, and metastasis—thereby playing a central role in cervical carcinogenesis and disease progression.

MicroRNA-383 (miR-383) is divided into two subtypes, miR-383-3p and miR-383-5p. One of the few studies on the role of miR-383 in cervical cancer shows that its target gene is Stratifin (SFN). miR-383-5p's established direct target is the 3'UTR of SFN (Stratifin) mRNA. The oncogenic effects of SFN overexpression are primarily mediated through a specific molecular axis involving the long non-coding RNA LINC01128. LINC01128, upregulated in cervical cancer, acts as a competitive endogenous RNA (ceRNA) by "sponging" miR-383-5p, thereby sequestering it and inhibiting its normal regulatory function [22]. The suppression of miR-383-5p leads to the subsequent overexpression of its direct target, Stratifin (SFN). SFN promotes key oncogenic processes such as cell proliferation, cancer stem cell development, and epithelial-mesenchymal transition (EMT). This LINC01128/miR-383-5p/SFN axis critically drives cervical

cancer progression [22]. Consequently, experimental restoration of miR-383-5p expression has been shown to exert anti-tumor effects by repressing SFN, highlighting its potential as a therapeutic target.

In cervical cancer, PD-L1 (programmed death-ligand 1) is frequently overexpressed, contributing to tumor immune evasion and promoting malignant phenotypes such as proliferation and invasion. Dong et al. (2018) demonstrated that PD-L1 is directly targeted and suppressed by a group of tumor suppressor microRNAs, including miR-383, miR-140, miR-142, and miR-340. Specifically, miR-383 binds to the 3'-UTR of PD-L1 mRNA, thereby inhibiting its expression. Loss of miR-383 expression in cervical cancer tissues correlates with PD-L1 upregulation and poorer patient survival, highlighting its role as a key epigenetic regulator of PD-L1. Restoration of miR-383 reduces PD-L1 levels and suppresses cancer cell growth and invasion, suggesting that miR-383 acts as a tumor suppressor partly through its negative regulation of PD-L1 [23].

Collectively, these studies show that miR-383 inhibits tumorigenesis in cervical cancer by suppressing PD-L1 and SFN. In our study, the observed increase in miR-383 expression (fold change = 1.38) in HPV-infected tissues suggests a possible compensatory host response that may restrain early carcinogenesis. However, this finding was not statistically significant, likely due to the limited sample size (n = 30) and the heterogeneity of HPV genotypes and lesion grades among participants.

Future studies should address these limitations by recruiting larger, well-stratified patient cohorts according to HPV type and CIN grade and performing functional assays (e.g., luciferase reporter, Western blot) to confirm direct targeting of SFN and PD-L1 by miR-383 in HPV-positive cervical cells. Also, evaluating the prognostic value of miR-383 expression levels in predicting disease progression or treatment response is warranted. Future prospective studies should also examine circulating miR-383 levels in serum or cervical swabs as a non-invasive screening biomarker. Moreover, investigating the interplay between HPV-encoded miRNAs (e.g., HPV-16 miR-H1) and host miR-383 could reveal novel regulatory networks. Such investigations would further clarify the clinical utility of miR-383 as a biomarker or therapeutic target in HPV-related malignancies.

The exploratory nature of this study limits definitive conclusions; however, it lays the groundwork for future confirmatory studies with larger sample sizes and functional validation. In conclusion, this exploratory study identifies miR-383 as a candidate tumor suppressor in HPV-related cervical lesions and provides a rationale for further investigation into its clinical utility as a biomarker or therapeutic target.

### Conflict of Interest

There are no conflicts of interest in this work.

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### Ethical Approval

Ethics committee approval was obtained for the patient samples to be used in the study (File number: 2019-10/01).

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