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Computational Identification of HPV-Related Molecular Drivers in Cervical Cancer

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ABSTRACT

Background & Objective: Cervical cancer is the fourth most prevalent cancer among women, accounting for about 8% of all female cancer-related deaths. Human Papillomavirus (HPV), a sexually transmitted infection, plays a significant role in its development. This study aims to investigate HPV's role in cervical cancer pathogenesis using a bioinformatics approach.

Materials & Methods: This computational study utilized transcriptomic data related to cervical cancer and Host-Pathogen Protein-Protein Interactions (HP-PPIs) of HPV. Gene expression data were sourced from the TCGA database, while HPV HP-PPIs were retrieved from HPIDB v.3.0. Differentially Expressed Genes/Proteins (DEGs/DEPs) were identified using the EdgeR library, applying a threshold of absolute log fold change greater than 2 and P-value less than 0.05. DEGs/DEPs were filtered based on HP-PPIs to identify interactions with HPV proteins. A protein-protein interaction (PPI) network was constructed to identify hub genes/proteins relevant to cervical cancer, and pathway enrichment analysis was conducted using Enrichr software.

Results: The analysis identified 661 interactions between DEGs/DEPs and HPV proteins. Notably, the VE6 and VE7 genes in HPV showed the highest degree of interaction with human genes. Key human genes such as MYC, ACTB, JUN, HDAC1, CCND1, and HSPA5 were significant in the interaction network. Pathway enrichment analysis indicated involvement in pathways like Epstein-Barr virus infection and viral carcinogenesis.

Conclusion: This research highlights essential hub genes and pathways in cervical cancer influenced by HPV, emphasizing the need for further in vitro and in vivo validation.

Keywords: Cervical cancer, Human papillomavirus, Bioinformatics, Differential expressed genes

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1. Introduction

Cervical cancer ranks as the fourth most prevalent cancer among women, recording 604,127 novel instances in the year 2020, along with a tally of over 341,831 fatalities **(1,2)**. This fatality figure constitutes approximately 8% of the aggregate annual count of female cancer-related deaths. It is noteworthy that the lion's share, about 84%, of incident cases as well as 88% of mortalities stemming from cervical cancer were concentrated within economies categorized as low- and middle-income nations **(3)**. Human Papillomavirus (HPV), an infection transmitted through sexual contact, is an infectious agent of notes. Its ability to establish a sustained infection, especially involving high-risk strains such as HPV 16 and 18, manifests a profound correlation with cervical cancer development **(4)**. The two HPV subtypes in combination account for approximately 70-75% of all cases of cervical carcinoma, in addition to contributing to a range of 40-60% of its precursor lesions. Observations stemming from epidemiological research indicate that virtually the entirety of cervical cancer instances, close to 100%, are attributed to HPV. Notably, a small subset of tumors, around 5%, does not show a connection to persistent HPV infection **(5,6)**. The virus typically invades the mucocutaneous epithelium, generating viral particles within mature epithelial cells. This subsequently triggers an alteration in the regulation of the cell cycle, fostering unregulated cellular proliferation. This process culminates in the accrual of genetic harm **(7,8)**.

In addressing the complex landscape of cervical cancer, a bioinformatics approach emerges as a valuable tool. Leveraging computational analysis and large-scale data integration, bioinformatics offers a means to unravel intricate relationships between HPV infection and cervical cancer development. By deciphering genomic, proteomic, and transcriptomic information, this approach strives to identify key molecular players and pathways underlying HPV's contribution to oncogenesis **(9-11)**.

The connection between cervical cancer and HPV infection continues to be a subject of active investigation and continuous discussion within the scientific community. While there have been suggestions of a potential association, the primary genes and molecular mechanisms by which HPV might contribute to or affect the development of cervical cancer have not been completely clarified. In this particular research, we employed a bioinformatics strategy to investigate potential connections between cervical cancer and HPV infection. The aim of the study to elucidate the molecular mechanisms underlying HPV involvement in cervical cancer progression through a comprehensive bioinformatics analysis.

2. Materials and Methods

Data collection

This computational study leverages transcriptomic data. The employed data in this research is divided into two categories, one pertaining to the host, in this case, human, and the other concerning the HPV. The human gene expression data linked with cervical cancer is sourced from The Cancer Genome Atlas (TCGA) database (https://portal.gdc.cancer.gov/). We specifically downloaded transcriptome data for cervical cancer samples from TCGA. This data includes gene expression profiles for thousands of genes across hundreds of patients with cervical cancer. Conversely, for collecting HPV-associated host proteins, the interactions between Host and Pathogen Proteins (HP-PPIs) specific to HPV were procured from the Host-Pathogen Interaction Database (HPIDB v.3.0). We specifically extracted interactions between all HPV subtypes and human proteins from HPIDB v.3.0 (https://hpidb.igbb.msstate.edu/). This ensures a comprehensive analysis of potential interactions across the diverse HPV strains.

Analysis of data

The gene expression information in cervical cancer was obtained from the TCGA database **(12)**. The entire cervical cancer dataset, along with the corresponding clinical details, was accessed using the R package TCGA biolinks **(13)**. The inclusion criteria consisted of the following: patients diagnosed with cervical cancer, patients with accessible gene expression data in the TCGA database, samples that had sufficient quality and quantity of RNA for gene expression analysis, complete clinical data associated with each sample (including patient demographics, disease stage, and treatment information), and the availability of matched normal tissue samples for comparison, if applicable. A total of 307 tissue samples from the cervix, consisting of 304 cancerous samples and 3 normal samples, were included in the study. The EdgeR library in the R programming language, in conjunction with the DGEList function **(14)**, was utilized to perform differential expression analysis. Subsequently, Differentially Expressed Genes/Proteins (DEGs/DEPs) meeting criteria of an absolute log fold change greater than 2 and an adjusted P-value less than 0.05 were selected. The analysis retained only genes accountable for encoding proteins.

Interactions between DEGs/DEPs of host and HPV proteins

As previously mentioned, the HP-PPIs of HPV were obtained from the HPIDB **(15)**. In this stage, the DEGs/DEPs were screened based on the HP-PPIs of HPV, with the intention of retaining only those interactions that were shared between the DEGs/DEPs of the human host and HPV proteins concerning cervical cancer. The interactions of genes/proteins between the human host and HPV in the context of cervical cancer were imported into Cytoscape and visualized **(16)**. Subsequently, the Network Analyzer plugin was applied to analyze this network, utilizing metrics like Degree, and Neighborhood Connectivity to evaluate the significance and roles of specific genes/proteins in the interactions between the human host and HPV in cervical cancer.

Interactions of DEGs/DEPs in host

For this step, DEGs/DEPs of the host that interact with HPV proteins were considered. Initially, the Protein-Protein Interactions (PPIs) network was assembled using STRING (https://string-db.org/) and visualized via Cytoscope software **(17)**, maintaining a minimum required interaction score of 0.4 in STRING. The network was then evaluated using the Network Analyzer plugin, with metrics like Degree, and Neighborhood Connectivity to identify the hub genes/proteins within the human host in cervical cancer.

Pathway enrichment analysis of DEGs/DEPs in host

KEGG serves as a comprehensive database for the systematic examination of high-level genome pathways associated with genes. Enrichr software was employed **(18)** to perform pathway enrichment analysis for DEGs/DEPs, applying a statistical significance cut-off of adjusted P-value less than 0.05.

3. Results

Analysis of data

After obtaining and processing the gene expression data related to cervical cancer from the TCGA

database, DEGs/DEPs were identified within the dataset. The comprehensive analysis revealed a total of 4458 DEGs/DEPs, including 1803 genes/proteins exhibiting an up-regulated pattern and 2655 genes/proteins displaying a down-regulated pattern. The details of these identified DEGs/DEPs can be found in Supplementary File 1.

Interactions between DEGs/DEPs of host and HPV proteins

Following the acquisition of DEGs/DEPs, the HP-PPIs associated with HPV were extracted from the HPIDB database. After this, the DEGs/DEPs underwent a filtering process, which incorporated the HP-PPIs pertaining to HPV. Through the analysis, a total of 661 interactions were recognized as common between the DEGs/DEPs originating from the human host and the proteins of HPV cervical cancer (as detailed in Supplementary File 2). These interactions were then visualized using Cytoscape (Figure 1).

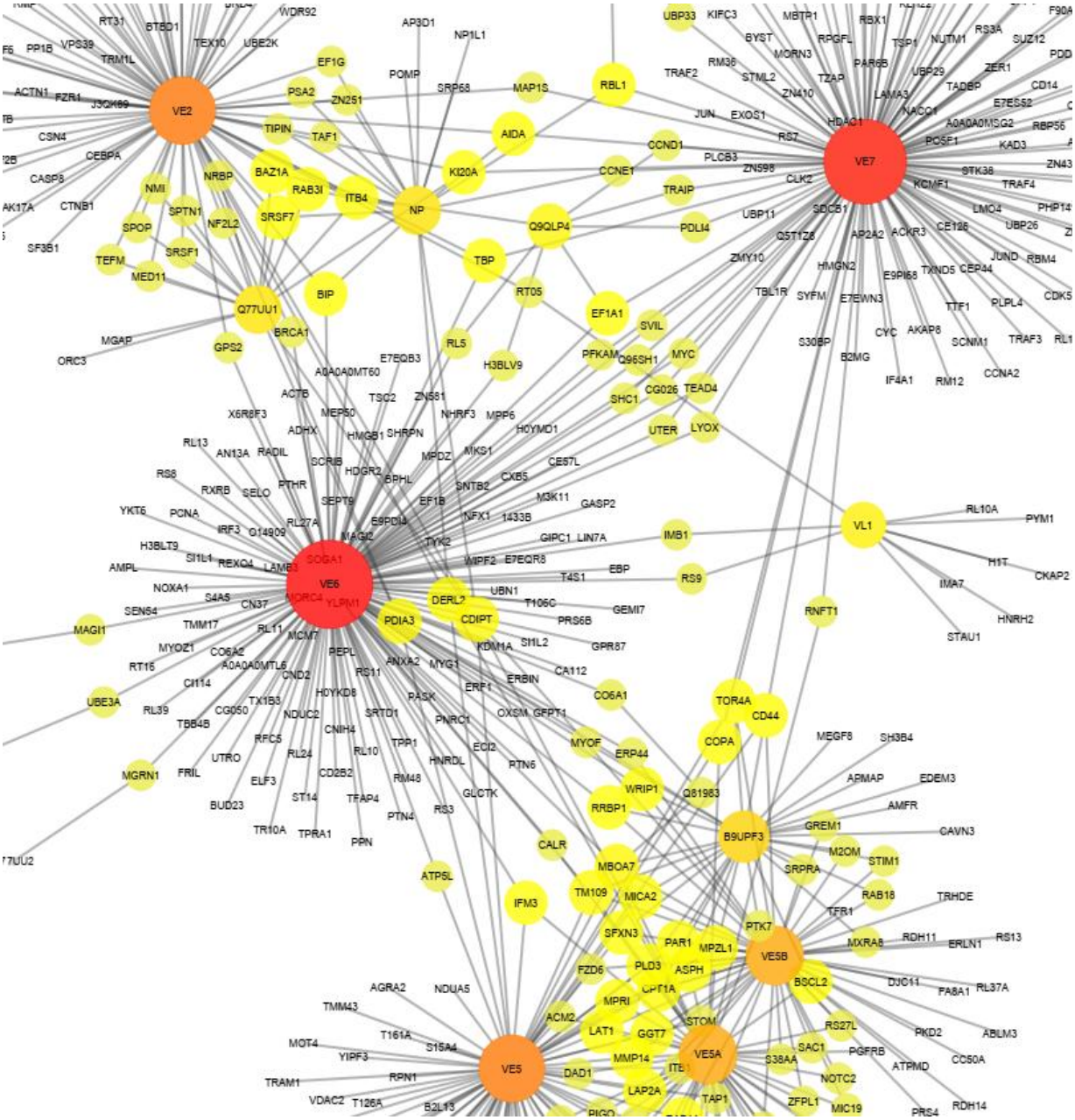


Figure 1. Network of interactions between host and pathogen genes. In this figure, Circle nodes represent the host and pathogen genes, while the colors ranging from white, yellow, orange and red indicate the degree of the nodes, ranging from low to high, in terms of genes and pathways. The nodes with a higher degree, indicated by a redder color, while those with a lower degree, represented by a whiter color

The resulting network was further examined employing Network Analyzer. Considering the findings, it is noteworthy that the genes VE6 and VE7 in HPV exhibited the greatest degree of connectivity and displayed the highest number of interactions with human genes (Table 1).

Table 1. List of effective and candidate genes in the interaction between human and pathogen genes

Name	Degree	NeighborhoodConnectivity
VE6	148	1.32
VE7	136	1.26
VE2	81	1.58
VE5	80	1.96
VE5A	54	2.25

Interactions of DEGs/DEPs in host

As highlighted in the preceding section, a collective sum of 4458 DEGs/DEPs was mined from the host in the context of cervical cancer. These specific DEGs/DEPs were then subjected to the STRING database to evaluate the interactions that transpire among proteins. The resultant PPIs extracted from this process (Figure 2)

Underwent comprehensive analysis utilizing NetworkAnalyzer. The findings of this analysis revealed that genes, namely MYC, ACTB, JUN, HDAC1, CCND1 and HSPA5, are the most significant genes within the network. These genes exhibited a notably higher degree of interaction with other genes in comparison to their counterparts (Figure 3 and Table 2).

Table 2. List of key genes after human interaction network analysis

Name	Degree	Neighborhood Connectivity
MYC	79	18.68
ACTB	78	18.21
JUN	54	21.83
HDAC1	50	21.14
CCND1	48	23.35

Pathway enrichment analysis of DEGs/DEPs in host

Enrichr was employed to carry out pathway enrichment analysis for the identified DEGs/DEPs. The results highlight the top five pathway terms: Epstein-Barr virus infection, viral carcinogenesis, Human papillomavirus infection, Cell cycle, and Protein processing in endoplasmic reticulum, as presented in Table 3.

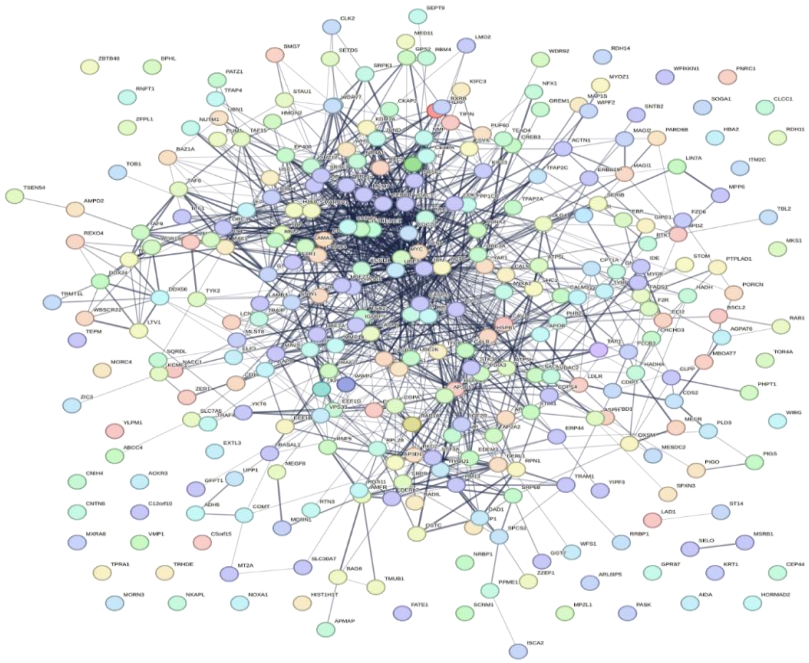


Figure 2. Interactions between selected human genes. This figure illustrates the protein-protein interactions derived from STRING (https://string-db.org/), showcasing the complex relationships and interactions among the selected human genes. Each node represents a specific protein, while the edges indicate the interactions between them. The visualization emphasizes the interconnected nature of these proteins, which may play significant roles in various biological processes and pathways relevant to human health and disease

Table 3. A list of some enriched biological pathways

Term	Adjusted P-value	Genes
Epstein-Barr virus infection	5.49E-12	PDIA3;JUN;CDKN1B;HDAC1;TAP2;TAP1;TRAF2;TYK2;CCNA2;CCND3;MAVS;SNW1;CASP8;CCND1;IRF3;CCNE1;TRAF3;MYC;CASP3;CYCS;CALR;CD44
Viral carcinogenesis	5.49E-12	JUN;CDKN1B;TBP;HDAC1;ACTN1;GTF2B;SCRIB;UBE3A;TRAF2;CDC20;CCNA2;RBL2;CREB3;CCND3;SNW1;CASP8;RBL1;CCND1;IRF3;CCNE1;TRAF3;CASP3
Human papillomavirus infection	2.45E-10	CDKN1B;HDAC1;LAMA3;UBE3A;THBS1;CCND3;PARD6B;CASP8;CCND1;CASP3;MAGI1;TBP;LAMB3;FZD6;TSC2;SCRIB;TYK2;CCNA2;RBL2;CREB3;RBL1;IRF3;NFX1;CCNE1;TRAF3
Cell cycle	6.86E-09	CDKN1B;PCNA;MCM7;HDAC1;RBX1;CDC20;CCNA2;RBL2;CCND3;FZR1;RBL1;CCND1;CCNE1;MYC;ORC3
Protein processing in endoplasmic reticulum	7.21E-09	PDIA3;EDEM3;TRAM1;HSPA5;WFS1;AMFR;RPN1;DERL1;DERL2;TRAF2;RRBP1;RNF5;RBX1;DAD1;STT3A;HYOU1;CALR

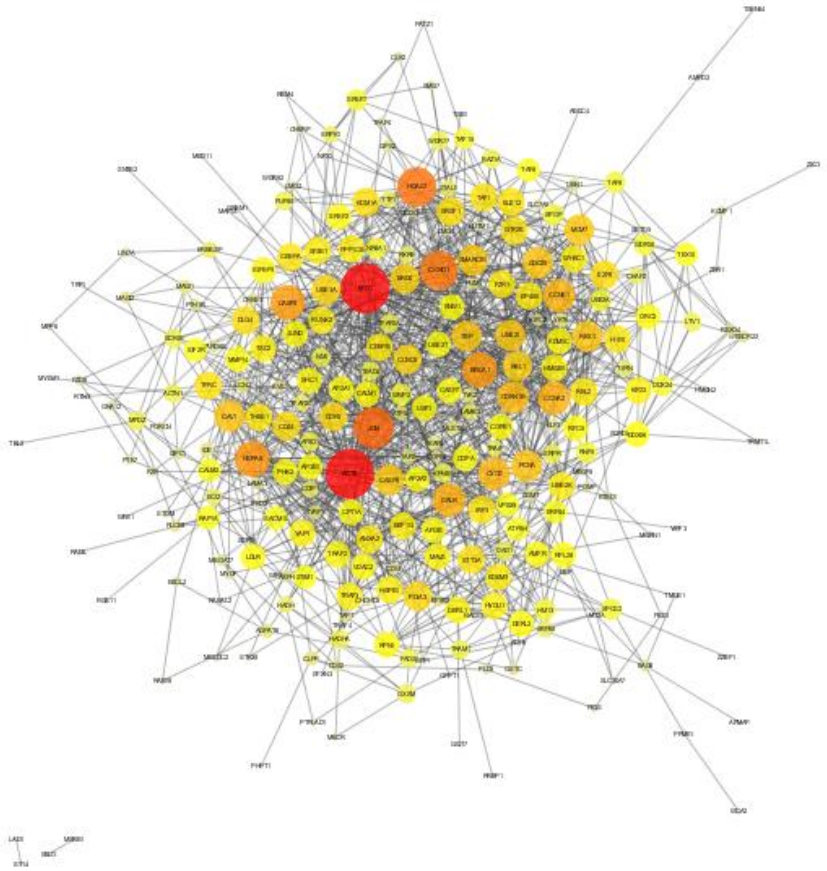


Figure 3. Network analysis on human genes. In this figure, Circle nodes represent the human genes, while the colors ranging from white, yellow, orange and red indicate the degree of the nodes, ranging from low to high, in terms of genes. The nodes with a higher degree, indicated by a bigger circle, while those with a lower degree, represented by a smaller circle

4. Discussion

Cervical cancer ranks fourth in prevalence among women. About 8% of all female cancer-related deaths are attributed to cervical cancer. HPV, a sexually transmitted infection, is a significant factor in cervical cancer. High-risk strains like HPV 16 and 18, which can establish persistent infections, are strongly linked to cervical cancer development, contributing to 70-75% of cases and 40-60% of precursor lesions. In

tackling the complexities of cervical cancer, bioinformatics proves valuable. Using computational analysis and comprehensive data integration, bioinformatics helps unravel the intricate relationship between HPV infection and cervical cancer **(19,20)**. By examining transcriptomic data, this study aims to identify crucial molecular players and pathways involved in HPV-driven oncogenesis.

In this study, the Epstein-Barr virus infection, viral carcinogenesis, Human papillomavirus infection are most significant pathways. The Epstein-Barr Virus (EBV) is classified as a member of the gamma-herpes virus family and was initially identified as the first oncogenic virus affecting humans. EBV is accountable for causing conditions like infectious mononucleosis and several types of cancer affecting both lymphoid and epithelial tissues. These cancers encompass B-cell lymphomas such as Burkitt lymphoma, Hodgkin lymphoma, and post-transplant lymphoproliferative disorder. Additionally, EBV's presence has been observed in other cancer types, including prostate, breast, oral, and salivary gland cancers. While there is documentation of EBV's existence in cervical cancer and its precursor lesions (CIN), the findings in the literature vary, and the direct causal role of EBV in the development of cervical cancer has not yet been definitively established **(21,22)**.

Viral carcinogenesis is a complex process involving the transformation of normal cells into cancerous ones due to the activity of oncogenic viruses. One such virus that has been extensively studied in relation to cervical cancer is the High-Risk Human Papillomavirus (hrHPV). The pathway leading to viral carcinogenesis involves various molecular mechanisms that contribute to the development and progression of cancer. This response will provide an overview of the viral carcinogenesis pathway, its association with cervical cancer, and insights from the published articles provided. The viral carcinogenesis pathway, particularly in the context of cervical cancer, primarily revolves around the activity of hrHPV. HPV are DNA viruses known to infect mucosal and cutaneous epithelial tissues. They have been classified into different types based on their genomic sequences. Among them, hrHPV types, such as HPV16 and HPV18, are strongly associated with the development of cervical cancer. The key molecular events involved in the viral carcinogenesis pathway include the expression of viral oncoproteins, disruption of cell cycle control, inhibition of apoptosis, and induction of genetic instability **(23,24)**.

In this study, VE6 and VE7 in HPV exhibited the highest number of interactions with human genes. The role of the E6 and E7 oncoproteins of HPV in cervical cancer is significant and has been extensively studied in various published articles. These oncoproteins play a crucial role in cervical cancer development and progression. The E6 and E7 oncoproteins of HPV have been recognized as key players in cervical cancer hallmarks and potential targets for therapy. They are known to be involved in the transformation of cervical cells, disrupting normal cellular processes and contributing to oncogenesis. E6 targets the tumor suppressor protein p53 for degradation, impairing its ability to induce apoptosis in damaged cells. E7 interacts with retinoblastoma (pRb) and other related proteins, disrupting cell cycle regulation and promoting abnormal cell division **(25,26)**. These interactions collectively contribute to the transformation of cervical epithelial cells into cancerous cells.

Aldo, in this study, MYC and ACTB have the highest number of interactions with other genes in human and considered as hub genes. The MYC gene plays a significant role in cervical cancer. The integration of the HPV viral genome into chromosome 8 at 8q24 activates the MYC proto-oncogene, contributing to oncogenesis. A study revealed a long-range chromatin interaction between the integrated HPV fragment, MYC gene, and 8q24.22, further confirming this relationship **(27)**. The dysregulation of MYC expression is associated with increased cell proliferation, survival, and angiogenesis in cervical cancer cells.

ACTB, also known as Beta-actin, is involved in various cellular processes, including cell structure, motility, and intracellular trafficking. While specific information about ACTB's direct role in cervical cancer was not found in the provided articles, its involvement in these cellular processes suggests potential contributions to tumor growth and metastasis **(24)**.

5. Conclusion

The primary objective of the research is to explore the relationship between HPV infection and the development of cervical cancer. The study's specific emphasis is on pinpointing central genes and essential pathways within cervical cancer that might be impacted by HPV's potential involvement in the development of the disease. The results of this investigation rely on the utilization of bioinformatics techniques and computational resources. Consequently, both in vitro and in vivo assessments are essential to validate and confirm the findings presented in this study.

6. Declarations

Acknowledgments

Not applicable.

Ethical Considerations

This study utilized publicly available transcriptomic data and protein-protein interaction databases, which

do not involve human subjects or animal experimentation.

Authors' Contributions

Conceptualization: MA, Methodology: MA, BA, Investigation: MA, Visualization: BA, Project administration: MR, Supervision: MR, MA, Writing – original draft: BA, Writing- review & editing: MR, MA.

Conflict of Interest

The authors declare that there are no conflicts of interest related to this study. All authors have disclosed any potential financial or personal relationships that could be perceived as influencing the research.

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