

## HUMAN PAPILLOMAVIRUS (HPV): STRUCTURE, PATHOGENESIS, DIAGNOSIS AND PREVENTION.

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**Abstract:** Human papillomaviruses (HPVs) are among the most widespread sexually transmitted infections worldwide, with significant oncogenic potential, particularly in the development of cervical carcinoma. HPV is a small, non-enveloped, double-stranded DNA virus with tropism to epithelial cells, where it can cause latent, productive, or transforming infections. More than 200 genotypes of HPV have been identified, of which high-risk types such as HPV-16 and HPV-18 are strongly associated with cervical and other anogenital cancers. Early detection and prevention strategies, including molecular diagnostics, cytological screening, and vaccination, have significantly reduced HPV-related disease burden in many countries. However, in developing regions, including Uzbekistan, challenges remain in terms of population awareness, accessibility of diagnostic methods, and implementation of vaccination programs. This article reviews the structure and replication of HPV, its role in carcinogenesis, diagnostic approaches, and current prevention strategies, highlighting the importance of strengthening HPV control measures in Central Asia.

**Keywords:** Human papillomavirus, HPV, cervical cancer, oncogenic types, diagnosis, prevention, vaccination

### Introduction (Literature Review)

Human papillomavirus (HPV) represents one of the most clinically significant viruses due to its high prevalence and established link with oncogenesis. According to the World Health Organization (WHO), HPV infection is the most common viral sexually transmitted disease, affecting nearly 80% of sexually active individuals at some point in their lifetime. Globally, more than 200 HPV genotypes have been identified, classified into low-risk and high-risk groups based on their oncogenic potential.

Low-risk HPV types (such as HPV-6 and HPV-11) are typically associated with benign lesions, including anogenital warts and respiratory papillomatosis. In contrast, high-risk types (HPV-16, HPV-18, HPV-31, HPV-45, among others) are implicated in the development of precancerous lesions and invasive carcinomas, particularly of the cervix, vulva, anus, penis, and oropharyngeal region. Cervical cancer remains the fourth most common cancer among women worldwide, with approximately 600,000 new cases and 340,000 deaths annually, the vast majority of which occur in low- and middle-income countries.

The viral genome is a circular double-stranded DNA of approximately 8,000 base pairs, organized into early (E) and late (L) regions. The early genes (E1, E2, E5, E6, E7) are essential for viral replication and modulation of host cell cycle control, while the late genes (L1, L2) encode structural proteins of the viral capsid. In particular, the E6 and E7 oncoproteins of high-risk HPV types interfere with tumor suppressor proteins p53 and Rb, leading to uncontrolled cell proliferation and malignant transformation.

In recent decades, significant progress has been made in HPV prevention and control. The introduction of prophylactic vaccines targeting the most oncogenic HPV types has proven to be a milestone in reducing HPV-related disease incidence. Furthermore, advancements in molecular diagnostics, such as PCR-based genotyping, have improved the sensitivity and specificity of HPV detection, enabling earlier intervention.

Despite these advances, challenges persist in many countries, including Uzbekistan, where limited awareness, insufficient vaccination coverage, and lack of regular screening programs hinder effective HPV control. Strengthening educational campaigns, expanding access to vaccination, and implementing routine screening are crucial steps toward reducing HPV-associated disease burden in the region.

## Materials and Methods

This article is based on a comprehensive review of scientific literature and epidemiological data on human papillomavirus (HPV). Published articles, systematic reviews, and reports from the World Health Organization (WHO), the Centers for Disease Control and Prevention (CDC), and other peer-reviewed journals were analyzed to evaluate HPV structure, pathogenesis, diagnostic strategies, and prevention methods.

For the structural and molecular aspects of HPV, virology textbooks and recent molecular biology studies were reviewed to provide updated insights into viral genome organization and protein function. The oncogenic mechanisms were examined by analyzing studies describing the interaction of HPV oncogenes (E6, E7) with tumor suppressor proteins p53 and Rb.

Epidemiological data, including HPV prevalence, genotype distribution, and cervical cancer incidence, were retrieved from global cancer statistics (GLOBOCAN 2020) and regional health databases when available. Special emphasis was placed on comparing data from high-income countries with that of low- and middle-income countries, particularly Central Asia, to highlight disparities in HPV burden and control.

Diagnostic strategies were evaluated by reviewing studies on cytology (Pap smear), histopathology, molecular methods (polymerase chain reaction, HPV DNA testing), and immunohistochemical approaches. Screening guidelines from international organizations were considered to assess the sensitivity and specificity of each diagnostic modality.

Preventive strategies, including prophylactic vaccination, were analyzed using data from clinical trials, population-based studies, and vaccination program reports. Studies focusing on vaccine coverage, public acceptance, and long-term efficacy were incorporated to assess the current status of HPV vaccination worldwide.

The analysis also included health policy reports and regional studies addressing HPV-related challenges in Uzbekistan and neighboring countries. Since local data are limited, global trends

were used as a comparative framework to highlight the need for improved HPV surveillance and control in Central Asia.

### Results and Discussion.

The analysis of high-risk human papillomavirus (HPV) types revealed a strong association with cervical carcinoma. As shown in the figure, HPV-16 accounted for the largest proportion of cases, representing approximately 55–60% of cervical cancers. HPV-18 was the second most frequent genotype, contributing around 15–20%. Other types such as HPV-31, HPV-33, and HPV-45 demonstrated lower but clinically significant prevalence rates.

These findings are consistent with previous epidemiological studies, which have identified HPV-16 and HPV-18 as the primary oncogenic drivers of cervical cancer. The predominance of HPV-16 highlights its aggressive nature and capacity for persistent infection, while HPV-18 is often linked with adenocarcinoma subtypes. The relatively smaller but notable contribution of HPV-31, HPV-33, and HPV-45 indicates that broader vaccine coverage may be necessary to achieve effective cervical cancer prevention.

Overall, the results emphasize the critical role of HPV-16 and HPV-18 in cervical carcinogenesis and underline the importance of targeted screening programs and vaccination strategies focused on these genotypes.

### Conclusion.

This study highlights the critical role of high-risk HPV types, particularly HPV-16 and HPV-18, in the development of cervical carcinoma. The predominance of HPV-16 underscores its highly oncogenic potential, while HPV-18 remains a significant contributor, especially in adenocarcinoma cases. Although other types such as HPV-31 and HPV-45 occur less frequently, their presence indicates that they should not be overlooked in preventive strategies. Overall, the findings emphasize the necessity of targeted vaccination and screening programs, with a focus on HPV-16 and HPV-18, while also considering broader coverage to protect against additional oncogenic HPV types. Strengthening early detection and immunization strategies could substantially reduce the global burden of cervical cancer.

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