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Human Papillomavirus Associated Oral Cancer Epidemiology and Public Health Implications

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Abstract

Human papillomavirus (HPV) is an important etiological factor in head and neck cancer. Its role is particularly well established in HPV-associated oropharyngeal squamous cell carcinoma. Persistent infection with high-risk HPV types, especially HPV16, can contribute to malignant transformation by disrupting important cellular regulatory pathways. The epidemiology of oral and oropharyngeal HPV infection differs across populations. Sexual behavior, tobacco exposure, and demographic characteristics can influence infection patterns. HPV is also detected in oral cavity cancers. However, its direct causal role in oral cancer remains less certain than its established role in oropharyngeal cancer. Recent studies have reported considerable geographical differences in HPV prevalence and genotype distribution. These findings highlight the need for standardized detection methods and reliable population-based surveillance. HPV vaccination provides an important opportunity for prevention. Studies have reported a lower prevalence of vaccine-type oral HPV among vaccinated populations. However, limited vaccine coverage and gaps in public awareness continue to affect prevention efforts. Combining HPV vaccination with oral health promotion and tobacco and alcohol control may help reduce the future burden of HPV-associated cancers. Further epidemiological and longitudinal studies are needed to better understand persistent oral HPV infection and its contribution to cancer development.

1. Introduction

Human Papillomavirus (HPV) infection has become an important part of the epidemiology of head and neck cancer. The association is particularly strong with Oropharyngeal Squamous Cell Carcinoma (OPSCC). Persistent infection with high-risk HPV, especially HPV16, is an established etiological factor in this cancer. In contrast, the relationship between HPV and cancers of the oral cavity remains less certain. This distinction is important because oral cancer, oropharyngeal cancer, and HPV-associated head and neck cancer are sometimes discussed as a single disease group. However, these cancers differ in their anatomical origin and biological characteristics. Current evidence supports a strong causal role for HPV in a substantial proportion of tonsillar and base-of-tongue cancers. The evidence for a direct causal role in cancers of the anterior oral cavity remains inconsistent (Kaur et al., 2024; Vani et al., 2024). The global importance of HPV-associated cancer extends beyond cancer biology. HPV infection is common in the general population. Most infections are transient and are cleared naturally. Only a small proportion of infections persist and may contribute to malignant transformation. Prevention is therefore an important part of HPV-associated cancer control. HPV vaccination can reduce infection with oncogenic HPV types. Public education and sexual-health awareness can also support prevention. Tobacco control remains important because tobacco exposure continues to contribute to the overall burden of head and neck cancer. Early recognition of suspicious oral and oropharyngeal lesions may support timely diagnosis. Appropriate HPV testing is also important for accurate disease classification. The public-health relevance of HPV-associated cancer has increased with changes in the epidemiology of OPSCC. Traditional head and neck

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cancers have been strongly associated with tobacco and alcohol exposure. HPV-associated OPSCC shows a different epidemiological pattern. HPV16 is the predominant type detected in HPV-positive OPSCC. A large systematic review and meta-analysis of 134 studies involving 12,139 OPSCC cases reported a pooled HPV prevalence of 48.1%. The prevalence was highest in North America at 72.6%. HPV16 was the most frequently detected genotype among HPV-positive tumors (Lu et al., 2024).

Global estimates have further demonstrated the contribution of HPV to the burden of head and neck cancer. Earlier IARC-based estimates attributed approximately 38,000 head and neck cancers each year to HPV. Around 29,000 of these cases were estimated to occur in the oropharynx (de Martel et al., 2017). More recent analyses have also shown substantial geographical variation. A global analysis estimated approximately 42,000 HPV-attributable oropharyngeal cancers in 2012. The corresponding attributable fraction was 42.7%. The attributable burden was higher in developed regions and was greater among men than women (Lu et al., 2022). The epidemiological transition is also relevant in countries where tobacco-related head and neck cancer remains common. India provides an important example. A 2024 systematic review and meta-analysis included 54 eligible Indian studies. Thirty-four high-quality studies were included in the quantitative analysis. The pooled HPV prevalence among head and neck cancer patients was 20% (95% CI, 12–32%). HPV prevalence was 16% in oral cancer and 22% in oropharyngeal cancer. It was 29% in laryngeal cancer. However, the HPV-attributable fraction based on markers of active carcinogenesis was much lower in oral cancer than in oropharyngeal cancer. The estimated attributable fraction based on E6/E7 mRNA was 3.36% for oral cancer compared with 12.54% for oropharyngeal cancer (Vani et al., 2024). These findings show that HPV-associated cancer should not be considered a single disease entity. Anatomical site is an important factor when interpreting HPV epidemiology. The method used for HPV detection can also influence prevalence estimates. Viral genotype and evidence of viral transcription provide additional information about the biological importance of infection. Host characteristics and exposure to tobacco and alcohol may further influence the epidemiological pattern. A clear distinction between HPV detection and HPV-driven carcinogenesis is therefore essential when interpreting epidemiological studies. This review examines the epidemiology and biological basis of HPV-associated oral and oropharyngeal cancer. It discusses major risk factors and geographical variation. Diagnostic considerations and current prevention strategies are also examined. Particular attention is given to vaccination, surveillance, and future research needs. These approaches may help reduce the growing burden of HPV-associated head and neck cancer.

2. HPV Infection and the Biological Basis of Oral Carcinogenesis

2.1 Oral HPV infection and transmission

HPV is a small, non-enveloped, double-stranded DNA virus with a genome of approximately 8 kb. More than 200 HPV genotypes have been identified. Some of these types infect mucosal surfaces. High-risk mucosal types, particularly HPV16, are strongly associated with HPV-related carcinogenesis. In contrast, low-risk types such as HPV6 and HPV11 are mainly associated with benign epithelial lesions rather than invasive cancer. Oral HPV infection can occur through intimate sexual contact, including oral sex. Other routes of transmission may also contribute. However, their relative importance is less clear. Epidemiological studies have consistently linked oral HPV infection with sexual exposure and a higher number of sexual partners. Smoking and concurrent genital HPV infection have also been associated with oral HPV infection. The natural history of oral HPV infection differs from that of cervical HPV infection. Oral infection is generally less common than genital infection. Most oral infections are also transient. A prospective Australian cohort of 704 adults detected oral HPV infection in 10.7% of participants at baseline. Among participants who were HPV-negative at baseline, 25% acquired a new oral HPV infection during 24 months of follow-up. Of those included in the clearance analysis, 78% cleared the infection during follow-up (Antonsson et al., 2021). Recent longitudinal evidence further highlights the importance of persistence. A multinational cohort of men was followed for a median of 44.8 months. The study identified 739 oncogenic oral HPV infections. Among incident infections, 78% were transient and 6.6% persisted for more than 24 months. Persistence was higher among infections already present at study entry. In this group, 24.3% persisted beyond 24 months. For HPV16, persistence beyond 24 months occurred in 14.1% of incident infections and 21.6% of prevalent infections (Dickey et al., 2025). These findings are important when interpreting oral HPV data. Detection of HPV DNA in the oral cavity does not indicate the presence of cancer. It also does not mean that cancer will develop. Most oral HPV infections are cleared. Persistent infection is more relevant to carcinogenesis and therefore requires greater attention in epidemiological studies.

2.2 E6, E7 and disruption of cellular regulation

The carcinogenic activity of high-risk HPV is mainly mediated by the viral oncoproteins E6 and E7. E6 promotes the degradation of the tumor suppressor p53 through the E6-associated protein pathway. This weakens cellular responses to DNA damage and reduces apoptosis. E7 targets the retinoblastoma protein (pRb). This disrupts the pRb–E2F regulatory pathway and promotes inappropriate cell-cycle progression (Narisawa-Saito & Kiyono, 2007). Loss of functional pRb activity leads to increased

expression of p16. This response forms the basis for the use of p16 immunohistochemistry as a surrogate marker of HPV-associated Oropharyngeal Squamous Cell Carcinoma (OPSCC). However, p16 positivity alone does not always demonstrate transcriptionally active HPV. Some tumors may show p16 overexpression without being driven by HPV. Therefore, p16 results should be interpreted together with the anatomical site and clinical context. HPV-specific testing may also be required in selected cases (Fakhry, Lacchetti, Rooper, et al., 2018). The difference between HPV DNA detection and biologically active infection is particularly important in epidemiological research. HPV DNA may reflect a transient infection rather than a tumor-driving infection. Detection of E6/E7 mRNA provides stronger evidence of viral activity. Other concordant HPV-specific biomarkers can also strengthen the evidence for a causal relationship.

2.3 Oral cavity versus oropharyngeal cancer

The anatomical distinction between oral cavity and oropharyngeal cancer is essential when interpreting HPV epidemiology. The oral cavity includes the anterior tongue, buccal mucosa, gingiva, floor of the mouth, hard palate, and related structures. The oropharynx includes the tonsils, base of the tongue, soft palate, and posterior pharyngeal structures. High-risk HPV, particularly HPV16, has a well-established association with cancers of the tonsils and base of the tongue. The evidence for a direct causal role in anterior oral cavity cancer is much weaker. A 2024 systematic review examined the causal relationship between HPV and oral cavity cancer. The review included 15 studies and found that HPV detection was more common among oral cancer cases than controls. The pooled odds ratio was 5.36. However, none of the included studies examined E6/E7 mRNA. The authors also identified problems related to inconsistent measurements, confounding, and the lack of high-quality HPV-specific biomarkers. Therefore, the available evidence was not sufficient to establish a causal relationship between HPV and oral cavity cancer (Kaur et al., 2024). This distinction should remain clear throughout epidemiological and public-health discussions. HPV-driven oropharyngeal cancer represents a well-established disease pathway (Figure 1). In contrast, detection of HPV in an oral cavity tumor should not automatically be interpreted as evidence of HPV-driven carcinogenesis. Evidence of transcriptionally active HPV is more informative than HPV DNA detection alone. This distinction is particularly important when estimating the HPV-attributable burden of oral cancer.

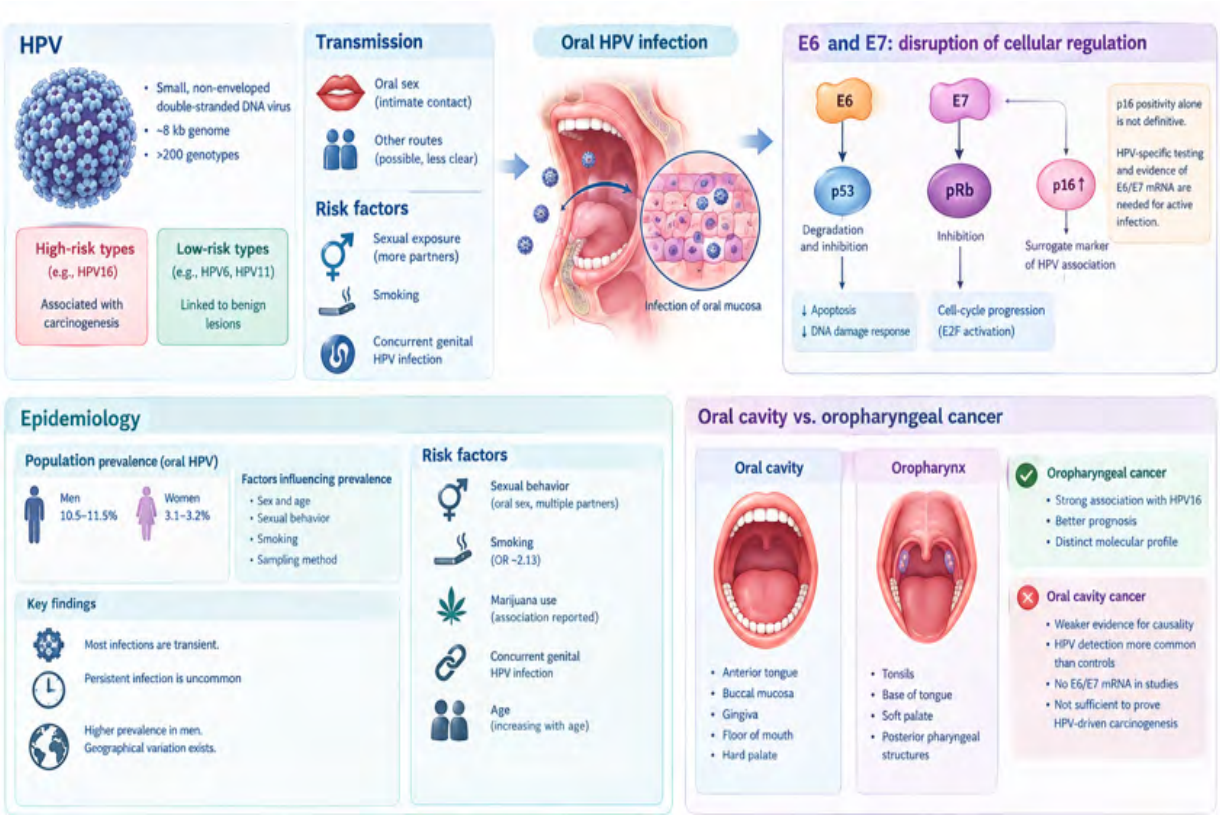


Figure 1: High-risk HPV oncoproteins E6 and E7 disrupt cellular tumor-suppressor pathways by promoting p53 inactivation and pRb inhibition. Increased p16 expression can serve as a surrogate marker of HPV-associated disease. However, p16 positivity alone does not establish transcriptionally active HPV infection. The epidemiological section highlights differences in oral HPV prevalence, persistence, and associated risk factors. The final panel distinguishes the anatomical sites and evidence linking HPV more strongly with oropharyngeal cancer.

3. Epidemiology of Oral and Oropharyngeal HPV Infection

3.1 Population prevalence

Population-based studies show considerable variation in oral HPV prevalence. Differences are observed according to sex, age, geography, sexual behavior, smoking, and sampling methods. In the United States, NHANES 2011–2014 data showed an overall oral HPV prevalence of 11.5% among men and 3.2% among women aged 18–69 years. High-risk oral HPV was detected in 7.3% of men and 1.4% of women. HPV16 was detected in 1.8% of men and 0.3% of women. These findings are relevant because HPV-positive oropharyngeal squamous cell carcinoma (OPSCC) occurs more frequently in men. Oral HPV infection was also associated with sexual and behavioral factors. Among men with concurrent genital HPV infection, oral HPV prevalence was 19.3%. This was much higher than the 4.4% observed among men without concurrent genital infection (Sonawane et al., 2017). A recent systematic review of studies published through October 2024 reported oral HPV prevalence ranging from 0.67% to 11.89% in healthy populations. The mean prevalence was approximately 5%. Prevalence was generally higher in men than in women. High-risk HPV prevalence ranged from 0.5% to 4.7%, with HPV16 being the most frequently detected type (Maltseva et al., 2025). Differences between studies do not necessarily represent true geographical variation. Sampling methods can have a major influence on HPV detection. Oral rinse, saliva, tonsillar swabs, and tissue samples may produce different results. PCR platforms, primer sets, genotype panels, and sampling frequency can also affect prevalence estimates. Differences in age and sexual behavior may further contribute to the observed heterogeneity.

3.2 Risk factors for oral HPV infection

Smoking may affect both HPV acquisition and persistence. Tobacco exposure can alter local immune responses and damage the oral epithelium. It may also promote a local inflammatory environment that supports viral persistence. The relationship between smoking and oral HPV may therefore involve more than direct effects on viral transmission. NHANES data provide further evidence for the role of behavioral factors. Oral HPV prevalence was higher among men who reported heavy cigarette smoking. Higher prevalence was also observed among current marijuana users and individuals with a greater number of lifetime sexual partners (Sonawane et al., 2017). Age may also influence oral HPV prevalence. The age pattern differs from that observed for cervical HPV infection. Oral HPV can be acquired throughout adulthood. Cumulative exposure and differences in viral clearance may contribute to infection patterns in middle-aged and older adults. Longitudinal studies also show that new infections continue to occur during adulthood. Persistent infection is particularly important because of its potential role in HPV-associated cancer.

3.3 Epidemiology of HPV-associated cancer

The epidemiology of HPV-positive OPSCC differs from that of traditional tobacco-associated head and neck cancer. HPV-positive tumors are more frequently observed in men. Cervical lymph-node involvement can also be prominent even when the primary tumor is small or clinically occult. HPV-positive tumors have a distinct molecular profile and show different clinical outcomes compared with HPV-negative disease. A large international individual-patient-data analysis included 7,654 patients with oropharyngeal cancer who had available p16 and HPV results. The study showed a clear relationship between combined p16 and HPV status and overall survival. Five-year overall survival was 81.1% in patients with p16-positive/HPV-positive tumors. It was 40.4% in patients with p16-negative/HPV-negative tumors. Patients with discordant p16 and HPV results had intermediate survival outcomes (Mehanna et al., 2023). HPV16 remains the dominant genotype in HPV-associated OPSCC. A systematic review and meta-analysis of nine studies included 1,310 patients with HPV16-associated disease and 219 patients with HPV-non16 disease. The pooled 5-year overall survival was 83.4% for HPV16-associated OPSCC and 69.3% for HPV-non16 disease (Shenker, Razavian, D'Agostino, et al., 2024). These findings show that HPV status has clinical relevance beyond epidemiological classification. The combined assessment of HPV and p16 status can provide important prognostic information. HPV subtype may also contribute to differences in clinical outcomes. Therefore, accurate HPV classification is important for epidemiological studies as well as clinical evaluation.

4. Geographical and Population-Level Variation

The HPV-attributable fraction of oropharyngeal cancer varies substantially between geographical regions. A recent systematic review and meta-analysis included 134 studies and 12,139 cases of oropharyngeal SCC. The pooled HPV prevalence was 48.1% globally. North America had the highest pooled prevalence at 72.6%. HPV16 represented the most common HPV type, and the estimated HPV-attributable fraction was highest in North America (Lu et al., 2024). The geographical pattern reflects differences in sexual behavior, tobacco exposure, HPV prevalence, healthcare access, diagnostic practices, and population structure. The interaction between HPV and traditional risk factors also differs between populations. A separate Indian systematic review involving 33 studies reported HPV prevalence ranging from 0% to 86.6% across individual studies. Several studies described HPV-positive tumors in younger patients and reported advanced disease and nodal involvement. However, the authors emphasized substantial

uncertainty in prevalence estimates and did not identify consistent survival differences between HPV-positive and HPV-negative disease in the Indian setting (Nandi et al., 2021). Another Indian meta-analysis published in 2024 included 33 studies and 4,654 head and neck cancer patients and reported a pooled HPV prevalence of 33% (95% CI, 25.8–42.6%). Considerable regional variation was observed, with an estimated prevalence of 47% in eastern India and 19.8% in western India (Satapathy et al., 2024). The differences between the Indian meta-analyses illustrate an important methodological issue. HPV prevalence depends strongly on study selection, anatomical site, specimen type, PCR methodology, and criteria for defining HPV positivity. Therefore, a single national prevalence estimate should be interpreted cautiously. The growing contribution of HPV to oropharyngeal cancer has created a shift in the traditional understanding of head and neck cancer. Tobacco and alcohol remain major causes of oral cavity cancer, while HPV has become increasingly important for tonsillar and base-of-tongue cancers. This epidemiological separation has consequences for prevention because tobacco control alone cannot address the entire burden of HPV-associated disease. At the same time, an increase in HPV-associated OPSCC should not be interpreted as evidence that all oral cancers are becoming HPV-driven. Current evidence continues to support a much stronger causal relationship between HPV and oropharyngeal cancer than between HPV and anterior oral cavity cancer.

5. HPV Detection, Diagnosis and Clinical Significance

HPV detection can be performed using PCR-based methods, hybridization assays, sequencing, p16 immunohistochemistry, and detection of viral transcripts. The choice of assay strongly influences epidemiological estimates. For population surveillance, oral rinse and saliva samples are attractive because collection is non-invasive and can be repeated. However, detection of HPV DNA in saliva does not establish persistent infection or tumor causality. A positive result should therefore not be interpreted in the same manner as HPV positivity in a tumor specimen. For cancer diagnosis, the anatomical site is critical. The College of American Pathologists guideline endorsed by ASCO recommends determination of HPV tumor status in newly diagnosed OPSCC. p16 immunohistochemistry can be used as a surrogate marker in appropriate oropharyngeal tumors, with positivity defined by strong nuclear and cytoplasmic staining involving at least 70% of tumor cells (Fakhry, Lacchetti, & Perez-Ordonez, 2018). HPV-specific testing may be required when the clinical and pathological findings are discordant. p16 should not automatically be considered synonymous with HPV infection in every head and neck tumor. This limitation becomes particularly important when evaluating non-oropharyngeal tumors.

HPV-positive OPSCC generally demonstrates a different clinical behavior from HPV-negative disease. The improved outcomes observed in HPV-positive disease have led to substantial interest in treatment de-escalation. However, HPV status should not be considered in isolation. The HNCIG-EPIC-OPC individual-patient-data analysis demonstrated that combined p16 and HPV status was more informative than either marker alone. The best outcomes occurred in tumors positive for both p16 and HPV, while discordant tumors had poorer outcomes (Mehanna et al., 2023). HPV subtype may also influence prognosis. A meta-analysis found better 5-year overall survival for HPV16-associated OPSCC than for HPV-non16 disease (Shenker, Razavian, D'Agostino Jr, et al., 2024). In contrast, the prognostic significance of HPV in oral cavity cancer remains uncertain. A systematic review and meta-analysis of non-oropharyngeal head and neck cancers found no significant overall-survival association between HPV positivity and oral cavity cancer, reinforcing the need to avoid extrapolating the strong prognostic effect observed in OPSCC to all oral cancers (Sahovaler et al., 2020).

6. Public Health Implications and Prevention

HPV vaccination represents the most important primary prevention strategy for HPV-associated malignancy. Current vaccines target HPV types responsible for a large proportion of HPV-associated cancers. Although the strongest evidence base initially focused on cervical and anogenital disease, accumulating evidence supports a protective effect against oral vaccine-type HPV infection. A systematic review and meta-analysis involving 15,240 participants found that vaccinated individuals were 46% less likely to have oral vaccine-type HPV infection than unvaccinated individuals (Tsentemeidou et al., 2021). Population-level evidence is also evolving. A 2026 analysis of nationally representative NHANES data reported a weighted prevalence of vaccine-targeted oral HPV infection of 1.9% among U.S. adults aged 18–64 years. Lower prevalence was observed in vaccinated individuals, while higher prevalence was associated with smoking, alcohol use, and multiple oral-sex partners (Byrd et al., 2026). Direct evidence showing a reduction in oropharyngeal cancer incidence after HPV vaccination requires long-term follow-up because the interval between infection and cancer development can span many years. Therefore, the absence of immediate cancer-incidence reductions should not be interpreted as evidence that vaccination has no effect on HPV-associated oropharyngeal cancer.

7. Future Public Health Directions

One of the major limitations in HPV-associated oral cancer research is methodological heterogeneity. Studies differ in anatomical definitions, tissue sampling, oral rinse collection, PCR primers, HPV genotype panels, p16 interpretation, and confirmation of transcriptionally active infection. These differences partly explain the wide variation in reported prevalence. Future epidemiological studies should clearly distinguish oral cavity cancer from

oropharyngeal cancer. HPV DNA detection should preferably be complemented by biomarkers capable of identifying biologically active infection. Standardized reporting of HPV genotype, p16 status, E6/E7 transcription, smoking, alcohol exposure, sexual behavior, age, sex, and tumor subsite would improve comparability. Cross-sectional studies provide prevalence estimates but cannot adequately characterize persistence. Longitudinal cohorts are therefore needed to determine which oral HPV infections persist, which factors promote persistence, and which molecular changes precede malignant transformation. Recent longitudinal studies have already demonstrated that most incident infections clear while a smaller fraction persists for prolonged periods. A clinically useful risk-prediction model may eventually combine persistent HPV16 detection with viral load, E6/E7 expression, host immune markers, tobacco exposure, age, and molecular changes in epithelial cells.

The development of non-invasive biomarkers represents an important research direction. Saliva is particularly attractive because it can be collected repeatedly without tissue biopsy. Salivary HPV DNA, HPV RNA, circulating tumor DNA, methylation signatures, extracellular vesicles, and immune markers are being investigated as potential biomarkers. However, analytical sensitivity alone is not sufficient. A useful screening biomarker must distinguish transient infection from clinically meaningful persistence and must demonstrate that early detection improves outcomes. Large prospective studies will be required before population-based oral HPV screening can be considered. The long latency between HPV infection and cancer development makes cancer-incidence endpoints difficult to evaluate in vaccine studies. National cancer registries linked with immunization records could provide important evidence over time. The epidemiological effect of vaccination may first become visible through reductions in vaccine-type oral HPV infection. Subsequent reductions in precancerous molecular changes and eventually HPV-associated OPSCC incidence could provide a sequence of population-level indicators. Global surveillance should also monitor vaccine uptake by sex, age, socioeconomic status, and geographical region. Such information can identify populations in which HPV-associated cancer prevention remains inadequate. HPV-associated oral and oropharyngeal cancer should be incorporated into broader national cancer-control strategies. Vaccination, tobacco control, alcohol-risk reduction, oral-health services, cancer registries, pathology capacity, and public education should operate as complementary components. Countries with high tobacco-related oral cancer burden may require a dual approach. HPV vaccination can address future HPV-associated disease, while tobacco and areca-nut control remain essential for reducing existing oral cavity cancer risk.

8. Conclusion

HPV has emerged as an important cause of oropharyngeal cancer, particularly cancers of the tonsil and base of the tongue, although its causal contribution to oral cavity cancer remains less certain. Persistent high-risk HPV infection, especially HPV16, can promote carcinogenesis through E6 and E7 oncoprotein-mediated disruption of key cellular regulatory pathways. The epidemiology of HPV-associated cancer varies considerably across populations, highlighting the need for standardized HPV detection, improved surveillance, and region-specific research. HPV vaccination represents an important long-term prevention strategy, while tobacco, alcohol, and other established oral cancer risk factors should continue to be addressed. Continued integration of vaccination, oral-health services, cancer surveillance, and public awareness may reduce the future burden of HPV-associated head and neck cancers.

Declarations

Ethics approval statement

Not applicable

Consent to participate

Not applicable

Consent to publish

Not applicable

Data Availability Statement

The data are available from the corresponding author upon reasonable request

Competing Interests

The authors declare that they have no conflict of interest

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