Emphasizing HPV infection and the molecular processes in oral cancer development

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Abstract

This research examines the epidemiological features and biomolecular mechanisms contributing to developing precancerous and cancerous conditions in oral lesions associated with Human Papillomavirus (HPV) infections. Existing literature highlights HPV's role in potentially malignant oral disorders. Consequently, the underlying biomolecular processes can initiate or contribute to benign lesions and oral carcinogenesis.

Keywords: HPV, Cancer, Infection

Introduction

Human Papillomavirus (HPV) infections represent a significant global health challenge, being associated with a wide range of diseases, including various types of cancer (1–9). HPV is a family of DNA viruses that show a particular tropism for epithelial cells of the skin and mucous membranes (10–20). There are over 200 HPV genotypes, some classified as low-risk (LR) and others as high-risk (HR) for cancer development (21–30).

Epidemiology

HPV infections are prevalent and often asymptomatic (31–41). It is estimated that about 80% of sexually active individuals will contract an HPV infection at some point in their lives (42–50). High-risk infections, such as those caused by HPV-16 and HPV-18 genotypes, are responsible for most cases of cervical cancer and are also associated with anogenital tumors, including those of the anus, vulva, vagina, and penis (51–60).

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Additionally, HPV has been identified as a significant etiological factor in head and neck cancers, particularly oropharyngeal cancer (61–70).

Epidemiology of Oral Cancer

Oral squamous cell carcinoma (SCC) is the sixth most common cancer worldwide, with an exceptionally high prevalence in Central-Southern and Eastern Asia (71–80). Mortality from oral cancer is significantly high, especially in Asian countries, where diagnosis often occurs in the advanced stages of the disease (81–90). Major risk factors include tobacco use, alcohol, betel quid, poor oral hygiene, malnutrition, and viral and bacterial infections (91–100, 294). These factors contribute to the malignant transformation of oral cells, leading to the development of SCC (101–110).

Role of HPV in Oral Carcinogenesis

HPVs are oncogenic viruses that can cause benign hyperplastic lesions, precancerous lesions, and cancerous lesions (111–120). The HPV genome comprises double-stranded DNA and encodes structural and regulatory proteins (121–130). The HPV E6 and E7 proteins are crucial in carcinogenesis. They inactivate the tumor suppressor proteins pRb and p53, promoting tumor growth (131–140). This inactivation allows cells to proliferate uncontrollably, leading to tumor formation (141–150).

Molecular Mechanisms

HPV E1 and E2 proteins are necessary for viral replication (151–166). The E5 protein activates the epidermal growth factor receptor (EGFR), promoting cell proliferation (167–173). The integration of HPV DNA into the host genome and the expression of E6 and E7 proteins are key events in cellular transformation (174–184). These proteins interact with cellular pathways, altering cell cycle regulation and promoting the survival of tumor cells (185–193).

Diagnosis and Detection

Molecular tests for HPV search for viral DNA sequences in tissues (194–198). PCR is a widely used technique for amplifying and detecting specific DNA sequences (199,200). In the context of HPV, PCR can be used to identify the presence of viral DNA in tissue samples (201,202). This technique is relatively simple and rapid, but its sensitivity can vary depending on experimental conditions and sample quality. PCR is often used as an initial screening method for the presence of HPV in clinical samples (203,204). Digital droplet PCR (ddPCR) represents an evolution of traditional PCR and is considered one of the most sensitive techniques for detecting and quantifying viral DNA (205,206). ddPCR divides the sample into thousands of droplets, each containing a single DNA molecule (207). This approach allows for more precise quantification of viral DNA, even at low concentrations. ddPCR can detect a few copies of viral DNA, making it particularly useful for the early diagnosis and prognostic evaluation of patients with head and neck SCC (208). The precision and sensitivity of ddPCR are crucial for an accurate diagnosis, which can significantly improve patient treatment and prognosis (209–212).

Controversies and Challenges

The link between HPV and oral squamous cell carcinoma (SCC) is debated due to variations in sampling and detection methods (213,214). Key factors include the transmission of HPV through saliva and its presence in asymptomatic individuals (215). The prevalence of HPV in healthy oral mucosa varies, and there is no vertical transmission from mother to fetus, posing additional challenges in managing HPV infection. Further research is needed to resolve these issues and develop more effective management strategies.

Implications for Prevention

Developing preventative and therapeutic methods requires an understanding of the molecular underpinnings of HPV infection and its role in mouth carcinogenesis (217,218). HPV vaccination might considerably decrease the incidence of mouth cancer linked to HPV. Advanced molecular diagnostic tools can yield more precise prognostic information (222)and improve the clinical care of patients with head and neck SCC. Prevention through immunization and early diagnosis is essential to lower the illness burden and increase survival rates.

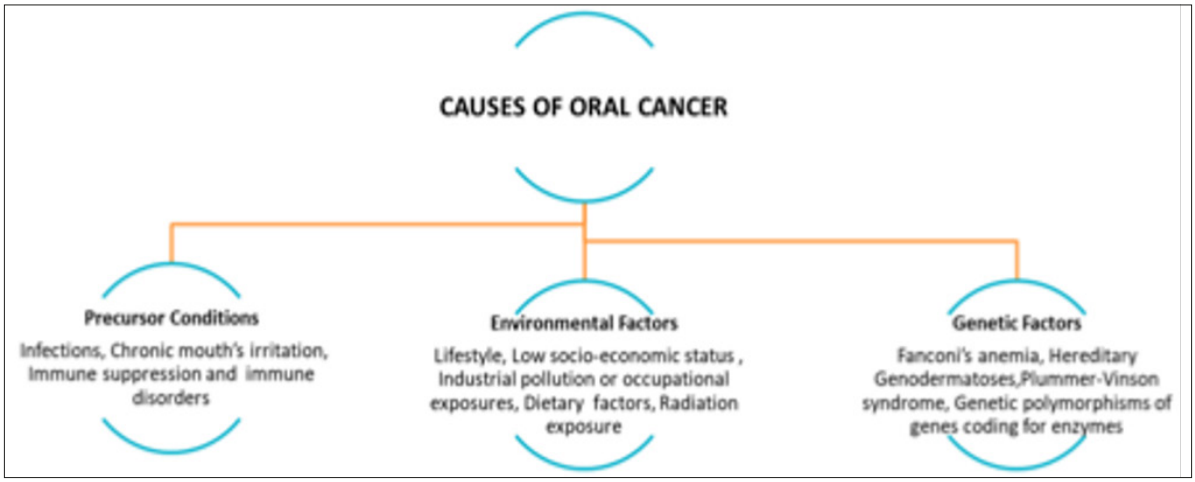

Figure 1

Preventive Strategies

One of the best ways to lower the incidence of HPV-associated mouth cancer is by HPV vaccination (223). High-risk oncogenic HPV infections, which account for a sizable portion of occurrences of mouth cancer, can be prevented with the use of current vaccinations (224). About 70% of cases of HPV-associated oral cancer are caused by HPV types 16 and 18, which are the targets of these vaccinations, which are intended to elicit an immune response against these viruses (225, 291).

Efficacy of HPV Vaccines

HPV vaccines, such as Gardasil and Cervarix, have shown high efficacy in preventing high-risk oncogenic HPV infections. Clinical studies indicate that these vaccines can reduce the incidence of precancerous lesions and associated tumors (226–228). Vaccination is also effective in preventing HPV infections in high-risk populations, such as young women and adolescents. Early vaccination, before exposure to the virus, is critical to maximize vaccine efficacy (229). The future of HPV vaccination includes the development of new vaccines that offer broader protection against a more significant number of HPV types (205,230–232). For example, Gardasil 9 protects against nine high-risk HPV types, compared to the four types covered by the original version of Gardasil. This increased coverage can potentially prevent an even more significant percentage of HPV-associated cancer cases. Additionally, research continues to explore the use of HPV vaccines to prevent other types of cancer beyond cervical and oral cancers, such as anal, vulvar, and vaginal cancers (233–235, 292).

Global Vaccine Coverage

Even though HPV vaccinations are effective, vaccination rates vary widely worldwide. High vaccination coverage rates have been attained in several high-income nations, including Australia and the United Kingdom, with encouraging outcomes in lowering HPV infections and precancerous lesions (236). Nonetheless, vaccination rates are still low in many other areas. Due to obstacles such as the vaccine's high cost, inadequate healthcare infrastructure, and low vaccination knowledge, immunization is still not readily accessible or acceptable in many places (237).

Educational Efforts and Health Policies

Health policies and focused educational initiatives are required to increase vaccination coverage and lower the prevalence of mouth cancer (238). Campaigns to raise public knowledge of the advantages of HPV vaccination and the dangers of HPV infections should be a part of educational initiatives. Parents, teenagers, and medical professionals should all be the focus of these programs to ensure they have the knowledge they need to encourage vaccination (239). By offering free or inexpensive vaccinations, including vaccination into public health initiatives, and establishing infrastructure for vaccine delivery and distribution, health policy should work to increase the accessibility and affordability of HPV vaccines (240). Policies should also address cultural obstacles and vaccination-related worries to boost public trust in the efficacy and safety of vaccinations (240–242).

Treatment of Oral Cancer

The location of the tumor, the patient's overall health, and the stage of the disease all contribute to the complicated treatment of oral cancer. Surgery, radiation, chemotherapy, and targeted treatments are available. Every strategy has benefits and drawbacks, and therapies are frequently combined to optimize effectiveness and enhance patient outcomes (243–246).

- Surgery is usually the initial treatment for oral cancer, mainly if the tumor is accessible and confined. The primary tumor and, if required, any adjacent lymph nodes that could be impacted must be removed. If the tumor is large, more involved operations, such as removing a portion of the tongue, jaw, or other anatomical components, can be necessary. Surgery may be followed by further treatments to get rid of any leftover tumor cells (247–249).
- Radiotherapy: This method kills tumor cells using high-energy radiation. It can be administered alone or in conjunction with chemotherapy and surgery. Radiotherapy is especially helpful for treating regions that are hard to access through surgery or shrinking the tumor before surgery (250–253). However, it has serious adverse effects, such as taste changes, xerostomia (dry mouth), and mucositis (inflammation of the mucous membranes) (254–263).
- Chemotherapy: This treatment uses medications to eradicate or inhibit malignant cell development (264–270). It can be given intravenously, orally, or by other means. To boost the effectiveness of the treatment, chemotherapy and radiation are sometimes combined (chemoradiotherapy) (271–275). Chemotherapy, however, can cause serious adverse effects, such as immunosuppression, nausea, vomiting, and hair loss (276–283).
- Targeted Therapies: A class of medical interventions known as targeted therapies target specific molecular targets implicated in the development and metastasis of cancer (284–290). Targeted treatments for oral cancer include cetuximab and other inhibitors of the epidermal growth factor receptor (EGFR). By blocking signals that encourage tumor cell development, these medications increase the effectiveness of therapy. To improve patient results, targeted therapies can be used with other treatments.

Future Research

To create more efficient management plans for HPV infection, additional study is required to resolve the issues and shed light on the disputes surrounding the virus. Addressing this global health issue requires ongoing research and innovation in diagnosis and treatment. Future research should concentrate on locating particular HPV biomarkers, creating fresh, focused treatments, and refining immunization plans.

9. Conclusions

The article provides a thorough summary of HPV infections and the molecular processes that contribute to mouth cancer. Understanding these pathways is essential for improving HPV-associated oral cancer prevention, diagnosis, and therapy. Further research is needed to address issues related to the virus and

resolve disagreements. Diagnostic and therapeutic research and innovation must continue to address this global health concern.

Abbreviations

HPV	Human Papillomavirus
SCC	Squamous Cell Carcinoma
LR	Low-Risk
HR	High-Risk
EGFR	Epidermal Growth Factor Receptor
PCR	Polymerase Chain Reaction
ddPCR	Digital Droplet Polymerase Chain Reaction
EGFR	Epidermal Growth Factor Receptor

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Conflicts of Interest

The authors declare no conflict of interest.

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