

□ Human Papillomavirus Type 16 (HPV16)

- Extracellular vesicular Wnt7b mediates HPV E6-induced cervical cancer angiogenesis by activating the β -catenin signaling pathway
- Non-replicating Newcastle Disease Virus as an adjuvant for DNA vaccine enhances antitumor efficacy through the induction of TRAIL and granzyme B expression
- Enhancement of therapeutic DNA vaccine potency by melatonin through inhibiting VEGF expression and induction of antitumor immunity mediated by CD8+ T cells
- NF- κ B potentiates tumor growth by suppressing a novel target LPTS
- Sensitivity and specificity of antibodies against HPV16 E6 and other early proteins for the detection of HPV16-driven oropharyngeal squamous cell carcinoma
- miR-34a and its novel target, NLRC5, are associated with HPV16 persistence
- Evidence for possible role of toll-like receptor 3 mediating virus-induced progression of pituitary adenomas
- Tobacco exposure results in increased E6 and E7 oncogene expression, DNA damage and mutation rates in cells maintaining episomal human papillomavirus 16 genomes

Human papillomavirus type 16 (HPV16) is a high-risk oncogenic strain of the human papillomavirus (HPV) family, with a strong tropism for squamous epithelial cells and a well-established role in the development of several human cancers.

Clinical significance:

HPV16 is the most carcinogenic HPV subtype.

It is responsible for approximately:

50–60% of cervical squamous cell carcinomas,

85–90% of HPV-positive oropharyngeal cancers,

and a substantial proportion of anal, vulvar, vaginal, and penile cancers.

Oncogenic mechanism:

HPV16 encodes the E6 and E7 oncoproteins, which respectively inactivate tumor suppressor proteins p53 and Rb, leading to cellular immortalization, genomic instability, and malignant transformation.

Transmission:

Primarily through sexual contact.

Persistent infection with high-risk types such as HPV16 is the key factor for oncogenesis.

Detection:

Identified by PCR-based HPV genotyping, in situ hybridization, or immunohistochemistry (e.g., p16 overexpression as a surrogate marker).

Prevention:

Highly effective vaccines (e.g., bivalent, quadrivalent, and nonavalent) include coverage for HPV16

and can prevent associated malignancies when administered prior to exposure.

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