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# "Comparison of Chromosomal Aneuploidy in HPV-Positive and HPV-Negative Oral Squamous Cell Carcinoma Cell Lines"

**Background:** Oral squamous cell carcinoma (OSSC) is the 16th most prevalent cancer worldwide and represents the most common type of head and neck cancer.[1] Although tobacco and alcohol use are the most commonly cited risk factors, the human papillomavirus (HPV) is believed to be associated with up to 70% of OSCCs in the United States.[2] While there are over 200 of these viruses, HPV16 in particular is present in over 90% of HPV-positive cases.[3] HPV has been demonstrated to induce chromosomal instability in various cancers, and similar mechanisms may contribute to the pathogenesis of oral squamous cell carcinoma (OSCC).[4] This study investigated the relationship between HPV status and the degree of aneuploidy in two of the most common and well established OSCC cell lines: HPV-negative CAL27 and HPV-positive SCC090.

**Methods:** Chromosomes were captured during mitosis using colcemid treatment, and cells were prepared for analysis using standard cytogenetic techniques.[5] Total chromosome counts were determined by light microscopy examination with 23 CAL27 cells and 34 SCC090 cells analyzed and categorized by ploidy status.

**Results:** HPV-negative CAL27 cells demonstrated predominantly diploid states, with 78.3% (18/23) maintaining normal chromosome numbers (46). In contrast, HPV-positive SCC090 cells showed dramatically increased chromosomal instability with only 17.6% (6/34) retaining diploidy. The majority of SCC090 cells exhibited significant aneuploidy, including 23.5% hypotriploidy (58-68 chromosomes), 17.6% hypertriploidy (70-80 chromosomes), and 17.6% tetraploidy and above (≥ 92 chromosomes). Triploid and near-triploid populations were observed exclusively in the HPV-positive cell line.

**Conclusions:** HPV infection is associated with substantial chromosomal instability in OSCC cells, with HPV-positive cells showing a marked increase in aneuploidy compared to HPV-negative cells. These findings suggest that HPV-mediated chromosomal instability may contribute to oral cancer progression and could have implications for precision-medicine treatment strategies targeting chromosomally unstable tumors.[6]

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[1] Ferlay J, Ervik M, Lam F, Laversanne M, Colombet M, Mery L, Piñeros M, Znaor A, Soerjomataram I, Bray F (2024). Global Cancer Observatory: Cancer Today. Lyon, France: International Agency for Research on Cancer. (https://gco.iarc.who.int/today). Accessed 7/14/2025.

[2] Centers for Disease Control and Prevention. HPV and Oropharyngeal Cancer. (https://www.cdc.gov/cancer/hpv/basic_info/hpv_oropharyngeal.htm). Accessed 7/14/2025.

[3] PDQ® Adult Treatment Editorial Board. PDQ Oropharyngeal Cancer Treatment. Bethesda, MD: National Cancer Institute. (https://www.cancer.gov/types/head-and-neck/patient/adult/oropharyngeal-treatment-pdq). Accessed 7/14/25.

[4] Korzeniewski N, Spardy N, Duensing A, Duensing S (2011). Genomic instability and cancer: Lessons learned from human papillomaviruses. Cancer Lett. 305 (2): pp 113-122.

[5] Howe B, Umrigar A, Tsien F (2014). Chromosome preparation from cultured cells. J Vis Exp. 83 (Jan 28): e50203.

[6] Tsien F (2020). Cytogenetics in precision medicine. Clinical Precision Medicine. Ch 1: pp 1-10.