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“The Role of Antiretroviral Therapy in Susceptibility to Oral Human Papillomavirus (HPV) Infection”

Despite the success of antiretroviral therapy (ART) in controlling human immunodeficiency virus (HIV) infection and extending the lives of people living with HIV (PLWH), risk of human papillomavirus (HPV)-associated head and neck squamous cell carcinoma (HNSCC) remains elevated in this population. Research by PD/PIs Cameron, Hagensee, and others has suggested that ART itself may predispose PLWH to oral HPV risk, and temporal associations between ART initiation and oral HPV acquisition and presentation of associated lesions have been demonstrated; however, the factors related to this residual risk of HPV+ HNSCC in PLWH remain unclear. This project aims to determine the susceptibility of oral HPV infection as a function of exposure to various branded ART medications (including Biktarvy, Genvoya, Cabenuva, Symtuza, Tivicay, and others), and their individual components (reverse transcriptase inhibitors, protease inhibitors, fusion inhibitors, and others).

To address this, saliva samples have been collected from PLWH in New Orleans, Louisiana stabilized by various ART regimens. Samples were tested for HPV using polymerase chain reaction (PCR) that detects a range of HPV genotypes, including oral-specific HPV. Results were stratified by demographic traits, HPV vaccine status (Merck Gardasil 9), and specific ART regimen of each patient, then analyzed to understand whether these factors contribute to an increased risk of HPV infection.

Of the 206 participants enrolled to date, the average age is 49 (range, 29-66), 51% are male, 85% are black (remaining 15% white), 40% are vaccinated against HPV, and 56% are taking Biktarvy. There was no association between oral HPV and age, gender, sex assigned at birth, or branded ART regimen (Biktarvy vs. Other). Strikingly, participants with a history of HPV vaccination were more likely to have oral HPV than those who are vaccine naive (43% vs. 33). However, this research is still ongoing, and further evidence is required to substantiate these preliminary findings. When combined with the broader efforts of PD/PIs Cameron, Hagensee, and Meyers to understand the interplay between HIV, ART, oral tissue architecture, and HPV on the NIH 1R01DE033878-01 grant, new opportunities to prevent HNSCC that take into consideration the effects of ART on patient risk will be revealed.