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

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Introduction

Despite the success of antiretroviral therapy (ART) in controlling human immunodeficiency virus (HIV) infection and extending the lives of people living with HIV (PLWH), the 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 as well as presentation of associated lesions have been demonstrated; however, the factors related to this residual risk of HPV+ HNSCC in PLWH remain unclear.

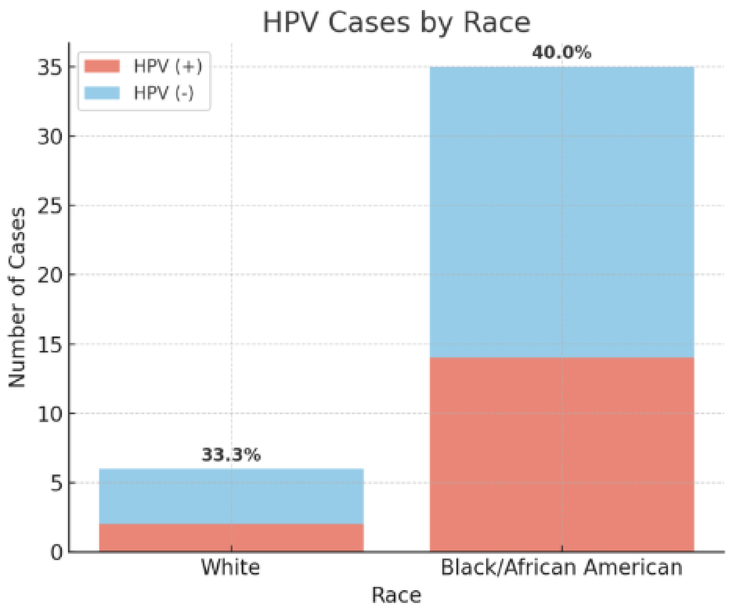
This project aims to determine the risk of oral HPV infection as a function of exposure to various ART medications (including Biktarvy, Genvoya, Symtuza, Tivicay, and others), their individual components (reverse transcriptase inhibitors, protease inhibitors, fusion inhibitors, and others), and demographics/medical history like race, gender, and vaccination status with the Merck Gardasil-9 HPV vaccine.

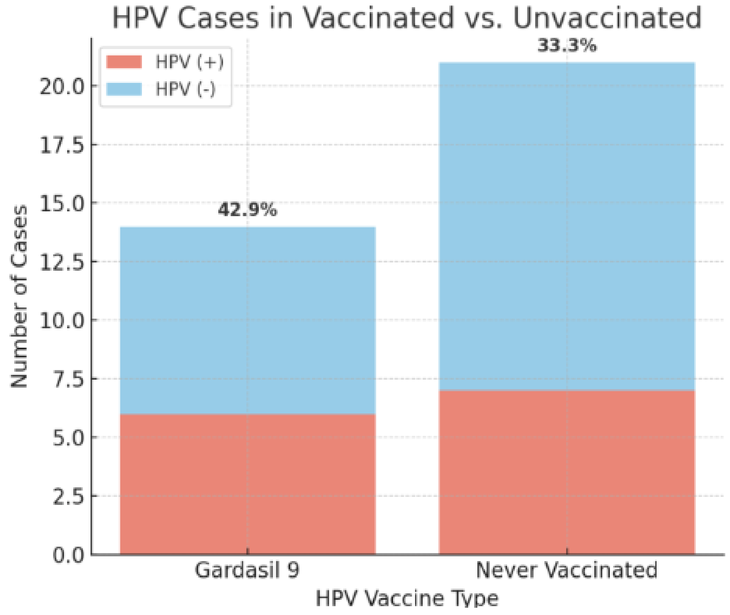
Methods

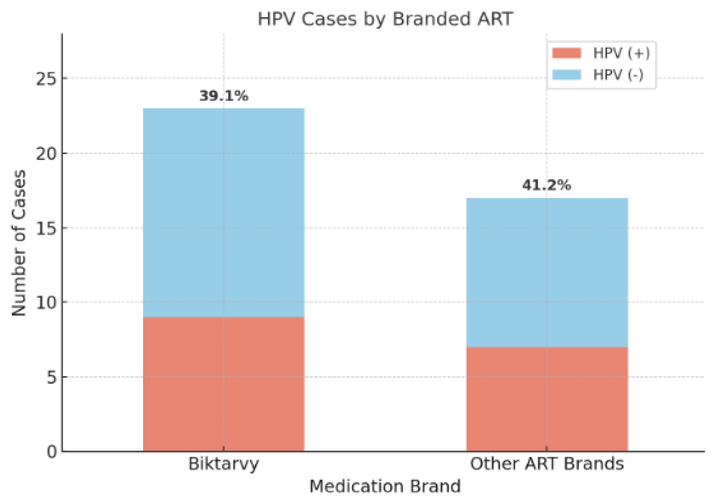
206 saliva samples were collected from PLWH stabilized by various ART regimens in New Orleans, Louisiana. Samples were tested for HPV using polymerase chain reaction (PCR). Results were stratified by demographic traits, HPV vaccine status, and specific ART regimen of each patient, then analyzed to understand the contribution of these factors to risk of HPV infection.

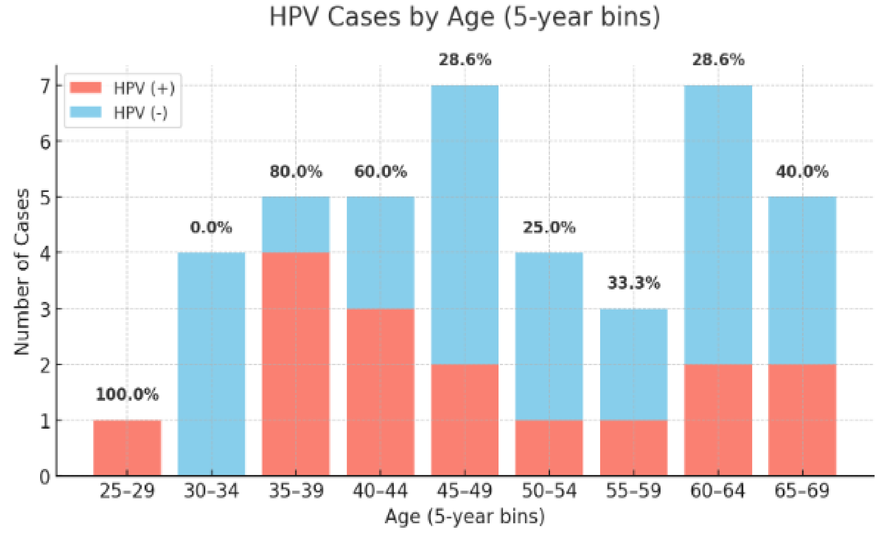
Of the 206 participants enrolled to date, 41 samples were processed and analyzed for this project. The average age is 49 (range, 29-66), 51% are male, 85% are black (remaining 15% white), 40% are vaccinated against HPV(Gardasil-9), and 56% are taking Biktarvy.

Results









Discussion

Based on this subset of patients, there is no association between HPV and age, gender, sex assigned at birth, or branded ART regimen (Biktarvy vs. Other). Strikingly, participants with a history of HPV vaccination were 9.52% more likely to have HPV than those who are vaccine naïve (Chi-square, p = 0.11), with a residual risk of the Gardasil-9 vaccine to HPV infection of 15.4%.

However, this research is still ongoing, and further evidence is required to substantiate these preliminary findings. The sample size analyzed is modest, leading to limited statistical power. Moreover, samples from patients on a pre-HIV infection prophylactic ART regimen (PrEP) are still yet to be included (to exclude HIV as a factor of HPV risk); a greater quantity of samples from patients across the spectrum of ART regimens is needed to demonstrate effects of individual medication components on oral HPV acquisition; and delineation between HPV genotypes is crucial in determining the risk of developing pathologies like HNSCC. 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 consider the effects of ART on patient risk will be revealed.