(LGAIN= 14.1; p= 0.017). This difference was primarily driven by an increase in Treg frequencies (HGAIN= 4.00%, LGAIN= 2.38%, No AIN= 1.88%; p= 0.048), and participants with HIV16/18 also had significantly higher Treg frequencies (HPV16/18= 4.02%, No HPV16/18= 2.68%; p= 0.021). Th17 frequencies did not vary with HPV status, HPV16/18 positivity or AIN stage. No mucosal parameters were associated with anorectal HIV RNA detection.

Conclusion HPV infection and high-grade AIN were associated with diffuse mucosal Treg infiltration, reducing the mucosal Th17/Treg ratio in participants with high-grade AIN. While changes in T cell immunology did not increase HIV RNA shedding in ART+ MSM, their effect on ART-naïve men or HIV susceptibility in HIV-negative men will be interesting areas for future research.

Disclosure No significant relationships.

**Background**

Antibody-dependent cell-mediated cytotoxicity (ADCC) mediated by natural killer (NK) cells plays a critical role in HIV-1 infection. As a novel subset of dendritic cells (DCs), 6-sulfo LacNAc-expressing DCs (slanDCs) also express CD16. However, the levels of slanDC-mediated ADCC during HIV-1 infection are not well addressed.

**Methods**

Forty-five HIV-1-infected subjects were enrolled and 19 HIV-1 negative individuals were used as healthy controls (HCs). The complex of gp120 and anti-gp120 was used to stimulate peripheral blood mononuclear cells (PBMCs) and the level of TNF-alpha secreted by slanDCs was detected using intercellular staining of flow cytometer.

**Results**

The counts of slanDCs in HIV-1-infected and treatment naïve patients were significantly lower than those of HCs and those receiving anti-retrovirus therapy (ART) (P=0.0331, P<0.0001). The number of slanDCs in HIV-1-infected patients with ART was significantly higher than those who did not receive ART, indicating that ART could help HIV-1-infected individuals to recover the number of slanDCs. The level of slanDC-mediated ADCC evaluated as the level of TNF-alpha production by slanDCs stimulated by the complex of gp120-anti-gp120, was significantly lower in HIV-1-infected subjects as compared with HCs and those receiving ART (P=0.0011, P=0.0002). The expression of CD16 (MFI) by slanDCs from HIV-1-infected patients receiving ART was significantly higher than that from HIV-1-infected untreated and HCs (P=0.0014, P=0.0003), and the expression of CD16 (MFI) in slanDC was positively correlated with the ADCC effect (P<0.0001).

**Conclusion**

The slanDC-mediated ADCC existed in HIV-1-infected patients and the level could be enhanced by ART, suggesting an alternative pathway involved in ADCC in HIV-1 infection.

**Disclosure**

No significant relationships.