There is a growing body of data demonstrating an increased risk for HIV acquisition in the presence of HPV infection, but the mechanism of this relationship is unclear. This study investigated the impact of HPV infection on both genital inflammation and HIV risk in the CAPRISA 004 trial.

Baseline cervicovaginal lavage specimens collected from 737 HIV-uninfected women were analysed to determine the prevalence of HPV infection. Clinical, reproductive, demographic and behavioral data were captured. The presence of DNA from 37 HPV genotypes was assessed using Linear Array, and the concentrations of 48 relevant cytokines were quantified by multiplexed ELISA assays. The presence of HIV was measured monthly using two rapid tests and confirmed by western blot and PCR.

Of the 737 eligible participants, 74% had prevalent HPV-infection (95% CI: 71–77%). Participants with prevalent HPV infection were 2.8 times more likely to acquire HIV infection compared to those with no HPV infection (95% CI: 1.3–5.9; p = 0.007). HIV risk was independent of the oncogenicity of HPV strains at baseline [(HPV oncogenic strains HR 2.5 (95% CI 1.0–6.2) vs non-oncogenic strains HR 2.1 (95% CI 0.9–5.1)], and was also increased in the presence of multiple concurrent infections (HR 3.1; 95% CI 1.4–6.8).

No cytokine signatures were associated with prevalent HPV infection. The use of tenofovir gel did not prevent HPV infection.

These data confirm a relationship between HPV infection and increased risk for HIV acquisition, and underscores the need to define the underlying biological mechanisms to inform targeted interventions in settings that bear a high burden of both infections.

**Abstracts**

### S17.2 HPV INFECTION, GENITAL INFLAMMATION, AND HIV RISK IN THE CAPRISA 004 TRIAL

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There is a growing body of data demonstrating an increased risk for HIV acquisition in the presence of HPV infection, but the mechanism of this relationship is unclear. This study investigated the impact of HPV infection on both genital inflammation and HIV risk in the CAPRISA 004 1% TFV gel trial.

Baseline cervicovaginal lavage specimens collected from 737 HIV-uninfected women were analysed to determine the prevalence of HPV infection. Clinical, reproductive, demographic and behavioral data were captured. The presence of DNA from 37 HPV genotypes was assessed using Linear Array, and the concentrations of 48 relevant cytokines were quantified by multiplexed ELISA assays. The presence of HIV was measured monthly using two rapid tests and confirmed by western blot and PCR.

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These data confirm a relationship between HPV infection and increased risk for HIV acquisition, and underscores the need to define the underlying biological mechanisms to inform targeted interventions in settings that bear a high burden of both infections.

### S17.4 USING DATA ON PATHOGENESIS AND EPIDEMIOLOGY TO INFORM ANAL CANCER SCREENING STRATEGIES: DATA FROM THE STUDY OF PREVENTION OF ANAL CANCER (SPANC)

Mary Poynten*, On behalf of the SPANC study team. The SPANC study team includes Andrew Gruich, Mary Poynten, Jeff Jin, David Templeton, Garrett Prestage, Dorothy Machakel, (Kirby Institute, UNSW, Sydney, Australia); Andrew Carr, Winnie Tong, (St Vincent’s Hospital, Sydney); Christopher Fairley (Melbourne Sexual Health Centre, Melbourne); Richard Hillman, Kirsten Howard, Kristen McCaffrey (Sydney University); Annabelle Farrowarth, Jennifer Roberts (Douglas Hand-Moor Pathology, Sydney); Suzanne Garland, Sepehr Tabrizi, Alyssa Comoll (Royal Women’s Hospital, Melbourne); Geoff Honnor, Kathy Triffit (Community representatives).

**Background**

HPV vaccination of school-aged boys will prevent anal cancer in future generations. Vaccination of gay men up to age 26 is recommended in several jurisdictions, but vaccination is generally not recommended at older ages because of a concern of possible lack of efficacy due to past or current HPV infection. Anal cancer screening, based on the model of cervical cancer screening, has also been proposed as a means to reduce morbidity.

**Methods**

The Study of the Prevention of Anal Cancer (SPANC) is a three-year prospective study of the natural history of anal HPV infection and cancer precursors in HIV-negative and -positive gay men aged ≥35 years. At each visit all men receive an anal swab for cytology and HPV genotyping, followed by high resolution anoscopic-directed biopsy for histological assessment.

**Results**

At the end of June 2015, 595 men had been enrolled. Median age was 49 and 35.3% were HIV-positive. Men of all ages enrolled in SPANC were likely to report multiple sexual partners in the past 6 months (overall 73.4% of 35–44 year olds decreasing to 62.1% of 65+ year olds, p trend = 0.03). The prevalence of HPV16, the genotype responsible for >90% of anal cancer, was 29.4% in 35–44, 30.8% in 45–54, 34.2% in 55–64 and 19.0% in 65+ year olds (p trend = 0.54), with no difference by HIV status. The incidence of HPV16 decreased with age from 5.6/100 person years (PY) in 35–44 year olds to 2.9/100PY in 55–64 year olds. There was no incident HPV16 in men aged 65+ (p trend = 0.059). At baseline, the prevalence of...