oral high grade intraepithelial neoplasia (HSIL) was higher in HIV-positive than in the HIV-negative participants (46.9% vs 32.3%, p < 0.001). Among those with HSIL at baseline, the clearance rate was similar between HIV-positive and -negative participants (38.4 vs 38.0 per 100PY, p = 0.636).

Conclusions Gay men in SPANC reported multiple sexual partners across the adult age-range, and incident HPV16 continued to be detected in men up to their seventh decade of life. This suggests that HPV vaccination of adult gay men may prevent infection and have a role in cancer prevention. Anal HSIL is highly prevalent, particularly among HIV-positive men, but there are high rates of clearance without treatment. These data suggest that a screening test which can distinguish persistent from transient HSIL is required. The role of HPV biomarkers in identifying those HSIL lesions most likely to persist should be investigated.

Oral Presentations

001 - Spread of antimicrobial-resistant gonorrhoea

**001.1 EVOLUTION AND SPREAD OF ANTIBIOTIC-RESISTANT GONORRHOEA**

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**Introduction** Neisseria gonorrhoeae has developed resistance to all classes of antimicrobials that have been used to treat it and strains that are resistant to multiple classes of antimicrobials have evolved. Only one antimicrobial (ceftriaxone) can now be used for empirical treatment in many countries. Hence, it is important to understand the dynamics and drivers of resistance evolution.

**Methods** First, we estimated rates of resistance evolution from antimicrobial resistance surveillance data from the USA and from England and Wales for heterosexual men (HetM) and men who have sex with men (MSM). Second, we developed dynamic transmission models to reconstruct the observed dynamics of N. gonorrhoeae transmission and resistance evolution in both HetM and MSM.

**Results** We found that resistance to ciprofloxacin and cefixime initially spreads exponentially at rates between 0.2 and 2.4 per year. These rates suggest that the proportion of resistant strains doubles every 3 to 35 months. We found lower rates of spread in HetM (0.2 to 0.8) compared with MSM (0.9 to 2.4). The models show that the treatment rate is the driving force for the spread of resistance.

**Conclusion** There is a trade-off in optimising the treatment rate to provide individual patient care to all those who are infected and to keep the spread of resistance as low as possible. These findings have implications for developing antimicrobial treatment strategies and point-of-care tests to detect resistance.

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