**Introduction** Herpes simplex virus type 1 (HSV-1) has emerged as the leading cause of first episode genital herpes among adolescents and young adults. The natural history of genital and oral shedding after first episode genital HSV-1 must be understood to provide risk assessment for sexual partners.

**Methods** Persons with laboratory documented first clinical episode genital HSV-1 infection obtained daily self-collected genital and oral swabs for 20 day sessions starting 2 months and 11 months after the first genital HSV-1 outbreak. HSV was detected in swabs using real-time quantitative HSV-1 PCR. Rates of genital and oral shedding were compared during the first and second swabbing sessions using Poisson regression models.

**Results** Of 52 persons who completed the first swabbing session, 27 (52%) were HSV-1 seronegative (primary infection) at presentation and 25 (48%) were HSV-1 seropositive at initial presentation. Twenty-five persons completed both sessions (11–12 months). Genital HSV-1 shedding was detected in 176 (12.2%) of 1438 swabs from the first shedding session, and declined to 46 (7.1%) of 645 swabs in the second session. The rate of genital lesions was 61 of 1595 (3.8%) days in the first session and 12 of 746 (1.6%) days in the second session. Oral shedding was detected infrequently, with 3.6% of swabs HSV-1 positive in the first session, and 3.3% during the second session. Oral lesions were rare in both sessions.

**Conclusions** Genital HSV-1 shedding occurs substantially less frequently than genital HSV-2 shedding, and declines in the first year after genital HSV-1 acquisition. These results will inform counselling messages about risk of sexual transmission to persons with first episode genital HSV-1 infection.

---

**011.2 REPEAT SYPHILIS IS ASSOCIATED WITH AN ALTERED IMMUNE PROFILE**

Chris Kenyon, Achilleas Tsoumanis, Kara Osbak, Maarjan Van Esbroeck, Tanja Cruydi, Luc Keitens. Institute of Tropical Medicine, Antwerp, Belgium

10.1136/sextrans-2017-053264.62

**Introduction** There may be a difference in the immune and inflammatory response to repeat compared to initial syphilis.

**Methods** Prospective study: We prospectively recruited patients with a new diagnosis of syphilis, described their clinical and demographic characteristics and tested their plasma for IFNa, IFNg, IL-1b, IL-12p40, IL-12p70, IP-10, MCP-1, MIP-1a, MIP-1b, IL-4, IL-5, IL-6, IL-7, IL-8, IL-10, IL-17A (Luminex multiplex assay (EMD Millipore) and their serum with a quantitative Rapid Plasma Reagin (RPR) test (Macrovue, Becton, Dickinson) at baseline pre-treatment and 6 months following therapy. The Mann-Whitney U-test was used to assess if cytokine levels and RPR titres differed between those with initial and repeat syphilis sampled at baseline and 6 month time points. Retrospective study: We compared RPR response kinetics between initial and repeat syphilis in persons attending our HIV/STI clinic.

**Results** Prospective study: 91 individuals, 36 with initial- and 55 with repeat syphilis, were included in the study. At baseline visit those with initial syphilis were more likely to be symptomatic and had higher levels of IL-8, IL-10 and MIP-1b. By the 6 month visit IL-10 remained higher in those with initial syphilis. Median RPR titres were higher at baseline in the repeat compared to the initial infection groups in those with symptomatic (primary and/or secondary) syphilis (1/128 [IQR 1/64-1/256] vs. 1/64 [IQR 1/16-1/128], p=0.016) but not those with latent syphilis. Retrospective study: Syphilis was diagnosed in 1 027/12 520 individuals tested. Repeaters had higher RPR titres at diagnosis and a stepwise increase in RPR titre with number of previous syphilis episodes. They had a different RPR response kinetic: they were less likely to be serofast and less likely to serorevert than initial syphilis. Not one of those with 4 or more episodes of syphilis seroreverted.

**Conclusion** Repeat syphilis has a different clinical presentation and immunological response than initial infection. We discuss the implications for clinicians and epidemiologists.

---

**011.3 EXTREME HETEROGENEITY IN VAGINAL MICROBIAL KINETICS WITHIN AND ACROSS WOMEN**

1JP Hughes, 1,2Anna Wald, 1,2DN Fredricks. 1Fred Hutchinson Cancer Research Centre, USA; 2University of Washington, USA

10.1136/sextrans-2017-053264.63

**Introduction** Incidental bacterial vaginosis (BV) is associated with a shift in the vaginal microbiota from lactobacillus predominant to a diverse community of anaerobic bacteria. The kinetics of this shift are poorly described. We sought to identify the dynamics of incident BV.

**Methods** Twenty women with frequent BV (>3 episodes per year) self-collected vaginal swabs every 8 hours for 60 days. Swabs were analysed with quantitative PCR targeting Lactobacillus iners, Lactobacillus jensenii, Lactobacillus crispatus, Gardnerella vaginalis, Atopobium vaginae, bacterial vaginosis associated bacterium-2 (BVAB2), and Megaspheera spp. We defined bacteria as absent, low (4 DNA copies), moderate (104–108 DNA copies) or high-level (108–1011 DNA copies). Participants kept detailed diaries regarding sexual behaviour, menstruation, antibiotic use and vulvovaginal symptoms.

**Results** We noted three states of the vaginal microbiota: three women had high-level lactobacilli with intermittent, transient low-level G. vaginalis throughout the sampling period (State 1); five women had high-level lactobacilli with persistent, fluctuating low to moderate-level G. vaginalis and other BV associated anaerobic species throughout the sampling period (State 2); two women had polymicrobial colonisation with high-level G. vaginalis and other BV associated species, and intermittent, transient low-level, or persistent moderate-level Lactobacillus jensenii and crispatus throughout the sampling period (State 3). Ten women shifted between States 2 and 3 on at least one occasion. Extremely rapid transition from State 2 to 3 over.

**Conclusions** The vaginal microbiota is extremely dynamic and BV develops over narrow time intervals. Low-levels of BV associated species in the vagina may be a risk factor for rapid, incident BV. Future studies will identify drivers of shifts in the vaginal bacterial biota.