(STs) previously reported in China (ST1, ST2, ST4, ST9, ST22, ST47), but also eight novel lineages, were detected. Only some quinolone-resistant isolates had acid amino substitutions in ParC (Ser83Leu in UPa) and ParE (Val417Thr in UPa and the novel Thr417Val in UUA), whereas the mechanism(s) for the remaining strains remains unclear. Although several isolates were non-susceptible to macrolides, mutations in 23S rRNA or substitutions in L4/L22 were not detected.

Conclusion This is the first study analysing susceptibility of *Ureaplasma* spp. isolates detected in Switzerland and the clonal distribution outside China. Resistance rates are low compared to other surrounding countries, but the empirical use of quinolones is compromised. We hypothesise that some hyperepidemic STs (e.g., ST4) spread worldwide via sexual intercourse. Large combined microbiological and clinical studies should address this important aspect.

**P06 - Genital microbiome**

**P06.01 WOMEN OF DUTCH ETHNIC ORIGIN HAVE LOWER PREVALENCE OF VAGINAL MICROBIOME DYSBIOSIS THAN WOMEN OF OTHER ETHNIC ORIGIN RESIDING IN AMSTERDAM**

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Introduction American women of African or Hispanic ancestry have increased risk of vaginal microbiome dysbiosis compared to women of European or Asian ancestry. However, the association between vaginal microbiome composition and ethnicity within Europe is largely unknown. We investigated this association in Amsterdam, The Netherlands.

Methods Non-pregnant women (18–34 years, n = 564) representing six ethnic origins (Dutch, South-Asian/Indonesian Surinamese, African Surinamese, Ghanaian, Turkish, and Moroccan) were cross-sectionally selected from the ongoing HELIUS multi-ethnic cohort study in Amsterdam for vaginal microbiome analysis. Extracted DNA from self-sampled vaginal swabs was sequenced targeting the V3/V4 region of the 16S rRNA gene and using the Illumina MiSeq platform, and sequence reads were clustered using hierarchical clustering.

Results Clustering of 502/564 samples with sufficient read counts resulted in microbiome clusters dominated by *Lactobacillus crispatus* (n = 120), *L. iners* (n = 168), *L. jensenii* (n = 8), *L. gasseri* (n = 10), *Streptococcus agalactiae* (n = 8), *Bifidobacteriaceae/Bifidobacterium* spp. (n = 10), *Gardnerella vaginalis* (n = 78), and a mixture of anaerobes (n = 100), respectively. Microbiome composition was significantly associated with ethnic origin (P = 0.002). Women of Dutch ethnic origin had the highest prevalence of L.crispatus-dominated microbiome (40% vs 16–26% in the other ethnic groups), the lowest prevalence of *L. iners*-dominated microbiome (28% vs 31–39% in the other groups), and the lowest prevalence of clusters dominated by *G. vaginalis* or a mixture of anaerobes (25% vs 30–45% in other groups). Turkish women and South-East Asian/Indonesian Surinamese women had the highest prevalence of *L. iners*-dominated microbiome (38% and 39%, respectively), and women from African descent (African Surinamese and Ghanaian women) the highest prevalence of clusters dominated by *G. vaginalis* or a mixture of anaerobes (48% and 44%, respectively).

Conclusion This large multi-ethnic study shows that dysbiotic vaginal microbiome compositions are significantly increased in women of non-Dutch ethnic origin. Therefore, these women may be at increased risk of STI acquisition and adverse reproductive health outcomes.

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**P06.02 LONGITUDINAL CERVICOVAGINAL MICROBIOME MEASUREMENTS OF WOMEN BEFORE AND AFTER HIV-SEROCONVERSION**

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Introduction Bacterial vaginosis (BV) by Nugent scoring is associated with enhanced acquisition and cervicovaginal shedding of HIV, but longitudinal molecular studies of these relationships are scarce.

Methods HIV-negative (n = 397) female sex workers in Kigali, Rwanda, were followed for two years. Demographic, behavioural, clinical, HIV, sexually transmitted infection, and cervicovaginal microbiota data were collected at regular intervals. The cervicovaginal microbiota were characterised by Nugent scoring, Amsel criteria (pH >4.5, a positive whiff test and presence of >20% clue cells on wet mount; two or three criteria indicated BV diagnosis), and phylogenetic 16S DNA microarray.

Results During follow-up, 19 women seroconverted for HIV. The associations between BV by Nugent or Amsel criteria and subsequent HIV seroconversion did not reach statistical significance (aOR = 1.56 (95% CI 0.51–4.77) and aOR = 4.85 (95% CI 0.59–39.90), respectively). For 10/19 women, phylogenetic microbiome composition was available before and after seroconversion, with a median of 324 days (range 42–386) before and 196 days (range 121–492) after seroconversion. Before seroconversion, none of the women had a *L. crispatus*-dominated microbiome, four had a *L. iners*-dominated microbiome, four a moderately dysbiotic microbiome and two severe dysbiosis. The microbiome composition of five women remained stable before and after seroconversion, four shifted to (more severe) dysbiosis, and one shifted from dysbiosis to a *L. iners*-dominated microbiome. After seroconversion, phylogenetic microbiome composition was available for all 19 women. 26% had a *L. iners*-dominated microbiome, 32% moderate dysbiosis, and 42% severe dysbiosis.

Conclusion In this population of high risk women, the association between BV and subsequent HIV seroconversion did not reach statistical significance, but statistical power was limited.
The molecular microbiome analysis showed that high levels of L. crispatus may protect against HIV acquisition and that recently acquired HIV infection may make women more prone to dysbiosis. More research is needed to confirm these relationships and determine causality.

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**P06.03** INCREASED G. VAGINALIS CLADE DIVERSITY IS ASSOCIATED WITH PENILE VAGINAL SEX AND BACTERIAL VAGINOSIS

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**Introduction** While BV is considered to be polymicrobial, some investigators consider *Gardnerella vaginalis* to be integral to its pathogenesis. *G. vaginalis* is however, also detected in women without BV. Recent evidence indicates different *G. vaginalis* clades exist, but it is unclear how these may be associated with the pathogenesis of BV.

**Methods** Established qPCR and multiplex assays were used to determine the association between *G. vaginalis* load and 4 clades of *G. vaginalis* with onset of penile-vaginal sex and BV in two distinct study populations. The WOW study investigated incident BV in women having sex with women (WSW); 378 longitudinal samples were selected from 51 WSW who developed incident BV and 51 who did not. 178 samples were selected from 42–27–21 year old female students without BV from the Fuss study: 15 women had no prior sexual experience with others, 15 had only engaged in non-coital activities and 12 had engaged in penile-vaginal sex.

**Results** *G. vaginalis* load was higher in women with BV [n = 37; log$_{10}$ median load = 6.2 (IQR = 6.5)] compared to those without BV (n = 156; log$_{10}$ median load = 3.2 (IQR = 4.8); p = 0.0001) in the WOW population. No difference in *G. vaginalis* load was found between women with no history of penile-vaginal sex [n = 40; log$_{10}$ median load = 4.1 (IQR = 3.3)] compared to women engaging in penile-vaginal sex [n = 35; log$_{10}$ median load = 4.1 (IQR = 4.8); p = 0.548] in the Fuss population. WOW participants with BV were more likely to have multiple *G. vaginalis* clades (88.6%; 95% CI = 0.74–0.95) compared to participants without BV (60.3%; 95% CI = 0.52–0.68, p = 0.0013). Multiple clades of *G. vaginalis* were also more common in Fuss participants who engaged in penile-vaginal sex (64.5%; 95% CI = 0.47–0.79) compared who had not (34.5%; 95% CI = 0.20–0.53, p = 0.0379).

**Conclusion** Penile-vaginal sex was associated with increased *G. vaginalis* clade diversity in young women without BV. Increased *G. vaginalis* loads and increased clade diversity were associated with BV in WSW.

**Disclosure of interest statement** No pharmaceutical grants were received in the development of this study.

**P06.04** GARDNERELLA VAGINALIS PRESENCE IN VAGINAL DYSBIOSIS: A SECONDARY ANALYSIS

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**Introduction** It has been hypothesised that *Gardnerella vaginalis* (GV) is necessary for the development of bacterial vaginosis (BV), and BV is associated with an increase of GV abundance and/or biofilm formation. We conducted a secondary analysis using data from multiple studies to investigate the first two hypotheses.

**Methods** Gram-stained Nugent scores and log-transformed bacterial counts obtained by in-house quantitative PCR for selected *Lactobacillus* species, GV and *Atopobium vaginae* (AV) counts were available for 1577 samples of women from Belgium (n = 469), Tanzania (n = 204), South Africa (n = 439), Kenya (n = 369), and Rwanda (n = 96). We determined the presence and median bacterial counts by Nugent score category using univariate analysis stratified by country.

**Results** Using Nugent scores, 1054(67%), 125(8%), and 398 (25%) samples had normal, intermediate and BV microbiota, respectively. GV presence was associated with BV in all countries (Chi$^2$: p < 0.001). The median GV counts were higher for samples with intermediate-score (Kruskal-Wallis: p = 0.001) and BV-score (p = 0.001) compared to samples with normal-score, with no difference between samples with intermediate-score and BV-score (p = 0.459). Only 25(6%) of the 398 samples with BV-score were negative for GV by PCR compared to 30(24%) with intermediate-score, and 663(63%) with normal-score. Of the 25 samples with BV and no presence of GV, AV was detected in 13 (52%). The AV presence and counts in the 25 samples were lower compared to BV-positive-GV-positive samples (88%) (Chi$^2$: p < 0.001; Kruskal-Wallis: p < 0.001) whereas AV prevalence and counts were higher compared to BV-negative samples (20%) (Chi$^2$: p < 0.001; Kruskal-Wallis: p < 0.001).

**Conclusion** We confirm that GV presence and higher GV loads are strongly correlated with BV by Nugent score. Half of the samples of women with GV-negative dysbiosis had AV present. Future research is needed to investigate the role of GV and/or AV-associated biofilm in BV and to evaluate the role of threshold of GV and AV for potential PCR based diagnostic testing for BV.

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