to go for clinical collection. Aim of this study was to evaluate the performance of a new Home–based Self Vaginal FLOQSwab™ (HBSVF - COPAN Italia, Brescia) in combination with a commercially available real-time PCR assay, Anyplex II STI-7 (Seegene, Seoul, Korea) which detects seven major pathogens in a single reaction (*Chlamydia trachomatis* - CT, *Neisseria gonorrhoeae* - NG, *Trichomonas vaginalis* - TV, *Mycoplasma hominis* - MH, *Mycoplasma genitalium* - MG, *Ureaplasma urealyticum* - UU, and *Ureaplasma parvum* - UP).

Methods A total of 78 asymptomatic donors, employees of a private industry (aged 18 to 45 years) were voluntarily enrolled to STIs screening. The subjects answered to a standardise anonymized questionnaire regarding the easy of use of self vaginal collection. The new HBSV swab was collected in a domestic context by following the detailed "how to use it" instructions. After collection, the HBSV swabs were shipped at room temperature to the laboratory in Pievesestina and processed within five weeks. The threshold cycle value (Ct) of a human genomic target (internal control, IC) and Ct of pathogens (CT, NG, TV, MH, MG, UU, UP) were taken as parameters to assess respectively, the efficiency of self-sampling and presence of any inhibitor effects, the stability of nucleic acids on dry swabs.

Results no failure results have been observed, the IC of all samples were amplified (average Ct 30). The real time PCR assay was able to identified 2/78 CT, 4/78 UU, 40/78 UP, 6/78 MH, 1/78 TV positive patients. No MG and NG positive patients have been detected. Women reported self-collection with HBSV easy and comfortable (100%).

Conclusion the new HBSV device showed excellent recovery and stability of nucleic acid of STI pathogens up to 5 weeks at room temperature. The HBSV is suitable for screening of STIs with real-time PCR assay.

P1.58 LACTIC ACID EXERTS ANTI-*CHLAMYDIA TRACHOMATIS* ACTIVITY ON THE EPITHELIUM BY REDUCING HOST CELL PROLIFERATION

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Introduction Epidemiological studies have demonstrated that the vaginal microbiota can significantly impact the risk of acquiring sexually transmitted infections. The human vagina often contains *Lactobacillus* spp., which produce lactic acid and create an acidic environment (pH 3.5–4) thought to reduce vaginal STIs. Unlike high d-lactate producers, *Lactobacillus* spp. that produce low amounts or no d-lactate, while achieving low pH do not reduce *Chlamydia trachomatis* infectivity. Further, exposure to culture supernatants from d-lactate producing *Lactobacillus* spp. reduces epithelial cell proliferation. We tested if low proliferation affects infection.

Methods A 3D model of A2EN cervical epithelial cells was exposed to lactic acid (D, L or D/L) at concentrations that produce pH 7, 5.5 and 4 or to several *Lactobacillus* spp. conditioned media (LCM) and infected with *C. trachomatis* serovar L2. Lysates from these A2EN cells were used to infect HeLa cells, and IFUs counted to determine infectivity. 2D A2EN cells were exposed to lactic acid, proliferation chemical inhibitors or LCM followed by infection with *C. trachomatis* L2. Proliferation and infectivity were evaluated by microscopy.

Results At pH 4, d-lactate and LCMs from high d-lactate producing vaginal *Lactobacillus* spp. afforded maximal protection compared to l-lactate. Interestingly, high infectivity was observed with HCl at pH 4, indicating that pH alone is not responsible for this protection. Exposure to d-lactate or LCMs reduced cell proliferation. Chemical cell proliferation inhibitors dramatically reduced *C. trachomatis* infectivity.

Conclusion These results suggest a differential role for vaginal *Lactobacillus* spp. in protecting against *C. trachomatis* infections and potentially other STIs. This protection is driven by the production of d-lactate, which acts on epithelial cells by inhibiting cell proliferation, which appears to be required for infection.

P1.59 GENOMIC CHARACTERISATION OF URETHRITIS-ASSOCIATED *NEISSERIA MENINGITIDIS*

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Introduction Mainly case reports have shown that *N. meningitidis*, typically a resident of the oropharynx and the causative agent of meningococcal meningitis and meningococcemia, is capable of invading and colonising the urogenital tract. This can result in urethritis, akin to the syndrome caused by *N. gonorrhoeae*, the etiologic agent of gonorrhoea. Recently, meningococcal strains associated with outbreaks of urethritis were reported to share genetic characteristics with gonococcus, raising the question of the extent to which these strains contain features that promote adaptation to the genitourinary niche, making them "gonococcus-like" and distinguishing them from other *N. meningitidis*.

Methods A total of 31 urethritis-associated *N. meningitidis*, representing multiple serogroups and independently collected over a decade and 3 continents, underwent genome sequencing and analysis. The genomes were compared with serogroup-matched *N. meningitidis* strains isolated from carriage and invasive disease and *N. gonorrhoeae* strains isolated from men with urethritis.

Results Intact nitrite reductase (AniA), disrupted factor-H binding protein (fHbp), and the lack of capsule are features previously speculated to promote urogenital colonisation. However, we found that a considerable number (n=11) of meningococcal urethritis isolates harbour mutations in AniA predicted to result in truncated peptides and a minority (n=4) of these isolates contained alleles associated with frameshifted fHbp. We noted substantial diversity in the capsule biosynthetic locus, including intact, disrupted, and absent capsules, indicating urogenital colonisation is possible across a range of capsular phenotypes.

Conclusion The meningococcal urethritis strains in this study do not share the allelic patterns of AniA, fHbp, or the capsule locus previously reported for urethritis-associated *N. meningitidis*. The allelic patterns likely reflect diversity in the underlying meningococcal population, rather than novel adaptation to the urogenital tract.