therapy (ART). Our hypothesis is that any association between STIs and VF would be confounded by drug use.

**Methods** The OHTN Cohort Study follows people receiving HIV care in Ontario. STI results and viral load (VL) data were retrieved via linkage with the provincial laboratory. We restricted analyses to 2610 MSM who completed>=1 annual questionnaire in 2008–2014 and had two consecutive VL<50 within a six-month period on ART. VF was defined as a single VL>=1000 or two consecutive VLs>=200. Periods of STI exposure were set around the diagnosis dates for each STI. We modelled STI diagnosis exposures and drug use as time-varying covariates on risk of VF using Cox regression adjusting for age, region and income as confounders. Our model allowed for repeat STI exposures and repeated VF events using the marginal means/rates model.

**Results** There were 472 VFs with a 24 month cumulative incidence of 12.1% (95%CI 11.1, 13.1). VFs at time of a new chlamydia or gonorrhoea infection were close to nil. We did not observe an increased risk of VF at the time of a new syphilis infection (HR=1.2 95% CI 0.8, 2.0; aHR=1.1 95% CI 0.7, 1.7). Risk was higher among drug users (non-injection aHR=1.4 95% CI 1.1, 1.8; injection aHR=1.8 95% CI 1.1, 2.6). There was no significant interaction but some evidence of positive confounding between syphilis and VF by drug use.

**Conclusion** Regardless of drug use, we did not find an association between a new STI diagnosis and increased risk of VF among men on suppressive ART. Our data are limited by possible misclassification of STI exposures, because not all men were tested, and among those diagnosed, exact dates of acquisition were unknown.

## P2.37 PRESENCE OF GENITAL *CHLAMYDIA TRACHOMATIS* SEROTYPE L2 INFECTION IN SOUTH AFRICAN WOMEN

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**Introduction:** *Chlamydia trachomatis* serotype-L, lymphogranuloma venereum (LGV), is a well-recognised infection among men who have sex with men in developed nations. In Africa, LGV is an uncommon but recognised cause of genital ulcer disease in men and women. The presence of genital infection in African women is unknown.

**Methods** In this pilot study we evaluated the presence of *C. trachomatis* serotype-L in 55 vaginal specimens that tested positive for *C. trachomatis*. These specimens were obtained in several studies over the period 2012–2016 that recruited women visiting a mobile health clinic in rural Mopani District (n=25) and in various settings in Pretoria: a tertiary obstetrics and gynaecology clinic (n=14), an antiretroviral treatment (ART) clinic (n=10) and a sexually transmitted infections (STI) clinic (n=6). Presence of serovar type-L of *C. trachomatis* was assessed using a targeted PCR assay and confirmed by whole-genome sequencing (WGS) of DNA from the clinical specimen.

**Results** We identified serotype-L *C. trachomatis* infection by targeted PCR in 8 cases. All of these women had presented with vaginal discharge at either the ART (n=5) or STI (n=3) clinic. Two women had co-infection with *Neisseria gonorrhoeae*, two with *Mycoplasma genitalium* and two with *Trichomonas vaginalis*. WGS of 5 specimens confirmed the presence of the L2 serovar. Also, one mixed infection of serovars L2 and E (minority) was observed.

**Conclusion** This pilot study demonstrates the presence of symptomatic cervical infection by *C. trachomatis* of serotype-L2 in African women. This confirms one report of (chronic) genital infection in African women from more than two decades ago. The significance of this observation is to be determined with regards to virulence, morbidity, distribution across the population and clinical management in the current context of the syndromic approach

## P2.38 MICROBIOLOGICAL ANALYSIS FROM A PHASE II STUDY IN ADULTS EVALUATING SINGLE DOSES OF GEPOTIDACIN (GSK2140944) IN THE TREATMENT OF UNCOMPLICATED UROGENITAL GONORRHOEA CAUSED BY *NEISSERIA GONORRHOEAE*

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**Introduction** Gepotidacin (GEP), a novel triazaacenaphthylene antibacterial, inhibits bacterial DNA replication. A Phase 2 study evaluated GEP as a single oral dose (1.5 or 3g) in subjects with urogenital gonorrhoea.

**Methods** Pre-dose specimens were obtained for culture and susceptibility testing by agar dilution. Microbiological success (MS), was culture confirmed eradication of *N. gonorrhoeae* (GC) at test-of-cure (TOC), 3–7 days post dose, in the microbiological evaluable (ME) population which consisted of all randomised subjects with culture confirmed urogenital gonorrhoea at baseline, who received any dose of GEP and returned for TOC.

**Results** Against 69 GC isolates recovered from baseline urogenital specimens in the ME population, GEP minimum inhibitory concentration [MIC (μg/mL)] range was ≤0.06–1 and MIC90 was 0.5. Resistance (R) to comparators were 33%, 28%, 20%, 0%, 0% and 0% for ciprofloxacin (CIP), penicillin, tetracycline, ceftriaxone, cefixime and spectinomycin, respectively. 2 isolates had elevated azithromycin MICs (MICs=2). Overall MS was 96% (66/69) in the ME population. PK/PD analysis showed 100% (61/61) MS when the free area under the curve/MIC ratio (*f*AUC/MIC) was ≥48. MS decreased to 63% (5/8) at *f*AUC/MICs ≤24. All isolates from the 3 urogenital failures were CIP-R, had a baseline GEP MIC=1 and a pre-existing D86N mutation in ParC, a critical residue in GEP binding. 2 were treated with a 3g GEP dose (*f*AUC/MICs=24) and 1 was treated with a 1.5g GEP dose (*f*AUC/MIC=12). 5 additional isolates with D86N were MS (2 at GEP MIC=1, 3 at GEP MIC ≤0.25). Isolates from 2 failed subjects (3g GEP dose) demonstrated R emergence to GEP (MICs increased ≥32 fold) and had an additional mutation (A92T) in GyrA, also located in GEP binding pocket.

**Conclusion** Subjects with *f*AUC/MICs ≥48 were MS, including 3 with D86N (*f*AUC/MICs ≥96). Further study of GEP, in the treatment of gonorrhoea is warranted, including demonstration that higher exposures suppress R in key isolate subsets.