O05.6 DISTINCT GROWTH AND GENOME PROFILES ARE PRESENT IN CLINICAL ISOLATES FROM WOMEN WHO FAIL TO RESOLVE GENITAL CHLAMYDIA INFECTION AFTER AZITHROMYCIN TREATMENT

[1]A Lawrence, [2]JS Hocking, [1]B Wee, [2]L Vodstrcil, [3]P Timms, [4]S Tabrizi, [1]WM Huston*. [1]*Institute of Health and Biomedical Innovation, Queensland University of Technology*; [2]*Centre for Epidemiology and Biostatistics, Melbourne School of Population and Global Health, University of Melbourne*; [3]*University of Sunshine Coast*; [4]*Royal Women's Hospital*

10.1136/sextrans-2015-052270.108

Introduction Treatment failure to be appears to occurring in women with genital *Chlamydia trachomatis* after azithromycin treatment. This study aimed to characterise the molecular basis of treatment failure by characterising clinical isolates from these women who failed to resolve the infection.

Methods A selection of 6 clinical isolates were cultured and whole genome sequence examined. Cultures were conducted in variety of cell lines, and in the presence of a novel anti-chlamydial drug to examine differences in growth and drug responses between isolates from treatment failure cases and treatment resolved cases.

Results The clinical isolates showed distinct growth profiles in different cell lines, with variation in infectious progeny yield and kinetics. Cervical, endometrial, gastrointestinal and fibroblast cell lines were compared and distinctions in growth were observed. There were no genome mutations in the ribosome loci in any isolate that would confer a direct resistance to azithromycin. However, there was less susceptibility to a novel anti-chlamydia drug by a clinical isolate from a treatment failure case as well as higher growth yield in the absence of the drug.

Conclusion Distinct phenotypic growth traits were observed in clinical treatment failure isolates of *C. trachomatis* that could underlie the mechanism of azithromycin treatment failure clinically.

Disclosure of interest statement This study was funded by the NHMRC.

O06 - STI therapy: new and old

O06.1 THE EFFICACY OF PRISTINAMYCIN FOR *MYCOPLASMA GENITALIUM* – AN INCREASING MULTIDRUG RESISTANT PATHOGEN

[1,2,3]CS Bradshaw*, [4,5]J Twin, [1,3]M Bissessor, [1,2]TRH Read, [6]JJ Jensen, [1,2]CK Fairley, [4,5,6]SM Garland, [1,2]MY Chen, [1]K Worthington, [1]M Grant, [4,5,6]SN Tabrizi. [1]*Melbourne Sexual Health Centre, Alfred Health, Melbourne, Australia*; [2]*Central Clinical School, Monash University, Melbourne, Australia*; [3]*School of Population and Global Health, University of Melbourne, Melbourne, Australia*; [4]*Department of Microbiology and Infectious Diseases, Royal Women's Hospital, Melbourne, Australia*; [5]*Murdoch Childrens Research Institute, Melbourne, Australia*; [6]*Staten Serum Institute, Copenhagen, Denmark*

10.1136/sextrans-2015-052270.109

Introduction To determine the efficacy of pristinamycin-based regimens for *M. genitalium*-infections failing prior regimens and to examine the presence of 23S rRNA and ribosomal protein gene mutations and their association with treatment failure.

Methods In 2013 *M. genitalium*-infected men and women attending Melbourne Sexual Health Centre who failed azithromycin and moxifloxacin were treated with pristinamycin 1g qid for 10 days with a test-of-cure (TOC) 3–4 weeks post-pristinamycin. From December 2014 pristinamycin was prescribed 1g bid for 10 days second-line following azithromycin failure. Pre- and post-treatment samples were stored and sequenced to detect 23S rRNA and ribosomal protein gene mutations, as potential markers of pristinamycin resistance.

Results By March 2015 37 *M. genitalium*-infected patients had received pristinamycin: 32 males (10 rectal; 22 urine samples) and 5 females (1 rectal; 3 urine; 1 cervical). TOC data are available on 25 patients at abstract submission: 20 were cured (80%; 95% CI 61–92%) and 5 (20%; 8–39%) failed pristinamycin. Failure rates in the 16 patients treated with 1g qid 10 days were 12% (n = 2), and 33% (n = 3) in the 9 treated with 1g bd 10 days, p = 0.23. Of the 5 pristinamycin failures; 2 were cured with moxifloxacin, 3 failed moxifloxacin and are awaiting TOCs following solithromycin or combined doxycycline/pristinamycin. Mutations in 23S rRNA and ribosomal protein genes were associated with failure of pristinamycin.

Conclusion Increasing reports of azithromycin and moxifloxacin failure for *M. genitalium*-infections necessitates evaluation of new agents. We present some of the earliest data on the use of pristinamycin for *M. genitalium*. Treatment failure occurred when delivered as monotherapy following failure of prior regimens. Current data on use of combined pristinamycin and doxycycline as a second line regimen after azithromycin failure will be available for presentation. Resistance mutations in the 23S rRNA and ribosomal protein genes are associated with pristinamycin failure.

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

O06.2 HIGH *IN VITRO* SUSCEPTIBILITY TO THE NOVEL SPIROPYRIMIDINETRIONE AZD0914 AMONG 873 CONTEMPORARY CLINICAL *NEISSERIA GONORRHOEAE* ISOLATES IN 21 EUROPEAN COUNTRIES DURING 2012–2014

[1]M Unemo*, [1]J Ringlander, [2]C Wiggins, [1]H Fredlund, [1]S Jacobsson, [2]M Cole, the European Collaborative Group (Eszter Balla, Christopher Barbara, Maria Jose Borrego, Tatiana Brilene, Stephanie Chisholm, Tania Crucitti, Alje van Dam, Steen Hoffmann, Samo Jeverica, Peter Kohl, Panayiota Maikanti, Beata Mlynarczyk-Bonikowska, Gatis Pakarna, Peter Pavlik, Angelika Stary, Paola Stefanelli, Guðrún Svanborg, Gaute Syversen, Eva Tzelepi, Julio Vazquez). [1]*WHO Collaborating Centre for Gonorrhoea and Other Sexually Transmitted Infections, National Reference Laboratory for Pathogenic Neisseria, Department of Laboratory Medicine, Clinical Microbiology, Faculty of Medicine and Health, Örebro University, Örebro, Sweden*; [2]*Sexually Transmitted Bacteria Reference Unit, Public Health England, Colindale, London, UK*

10.1136/sextrans-2015-052270.110

Introduction Resistance in *Neisseria gonorrhoeae* has emerged to all antimicrobials available for treatment of gonorrhoea. The first gonococcal strains with high-level resistance to ceftriaxone, the last option for first-line empirical antimicrobial monotherapy, were recently described. Consequently, new treatment options are essential. In this study, the *in vitro* activity of the novel spiropyrimidinetrione AZD0914, a DNA topoisomerase II inhibitor, among contemporary consecutive clinical *N. gonorrhoeae* isolates obtained in 21 European countries was investigated and compared to the activities of antimicrobials currently or previously recommended for treatment.

Methods Consecutive clinical *N. gonorrhoeae* isolates (n = 873) cultured in 21 European countries during 2012–2014 were examined for their susceptibility to AZD0914. The MICs of AZD0914 were determined using agar dilution method. For comparison, the MICs of ceftriaxone, cefixime, azithromycin