(NG). Antimicrobial resistant (AMR) NG is a global public health concern, which may emerge *de novo* or be imported to the UK when individuals infected abroad have subsequent sexual partners at home. We investigated whether patients who reported sex outside the UK ('sex abroad') were more or less likely to be diagnosed with AMR NG.

Methods Logistic regression was used to model the association between reported recent sex abroad and decreased susceptibility (DS) to ceftriaxone (MIC (mg/L)>0.015) and cefixime (0.125) and azithromycin AMR (>1) stratifying by sexual orientation (men who have sex with men (MSM) and heterosexual men and women) from isolates in England and Wales collected within the Gonococcal Resistance to Antimicrobials Surveillance Programme, 2004–2015.

Results Over 10% of MSM and heterosexuals reported sex abroad. Among heterosexuals, infection with a strain of NG with DS to ceftriaxone was associated with sex abroad after adjusting for potential confounders: ceftriaxone (DS prevalence, adjusted odds ratio (95% confidence interval)): 14%, 1.8 (1.3–2.3). Infection with NG DS/AMR to cefixime or azithromycin was not associated with reported sex abroad after adjusting for potential confounders: cefixime 4%, 1.6 (0.9–2.7); azithromycin 2%, 1.5 (0.7–3.3). For MSM, no association was found between infections with DS/AMR NG and sex abroad.

Conclusion In the UK, heterosexuals with NG infection who report sex abroad are at a higher risk of DS to ceftriaxone, suggesting that sex abroad might be the source of some AMR NG within heterosexual networks and highlighting the importance of condom use for travellers. In contrast, DS/AMR NG was not associated with sex abroad among MSM, which might reflect established AMR within MSM networks in the UK. Genetic comparison of these isolates using whole genome sequencing might further elucidate how AMR NG is imported and disseminated in the UK.

O05.6 THE IMPACT OF A RAPID GENOTYPIC *NEISSERIA GONORRHOEAE* ASSAY ON TARGETED CIPROFLOXACIN THERAPY

[1]Lao-Tzu Allan-Blitz, [2]Romney M Humphries, [2]Peera Hemarajata, [3]Ashima Bhatti, [4]Jeffrey D Klausner. *[1]David Geffen School of Medicine, Ucla, Los Angeles, USA; [2]Department of Laboratory Medicine, University of California Los Angeles, Los Angeles, USA; [3]Fielding School of Public Health, University of California Los Angeles, Los Angeles, USA; [4]Division of Infectious Diseases: Department of Medicine, University of California Los Angeles, Los Angeles, USA*

10.1136/sextrans-2017-053264.29

Introduction Multidrug-resistant *N. gonorrhoeae* infections are a threat to public health. In November 2015, UCLA Health began routine gyrase A (*gyr*A) genotyping all *N. gonorrhoeae* positive specimens, and reporting genotype and treatment recommendations for wild-type infections. Physicians were educated about wild-type *gyr*A genotypes predicting ciprofloxacin susceptibility. In May 2016 we began sending electronic reminders to providers of genotype results and treatment recommendations.

Methods We reviewed records for all laboratory confirmed *N. gonorrhoeae* cases from January 1st 2015 - November 30th2016. Infections in different anatomic sites were considered unique infections, while unique infections in a single patient on the same date were considered a case. Empiric therapy was defined as treatment within one day of specimen collection.

Results Among 381 patients (32% HIV infected) there were 411 cases and 459 anatomic site-specific *N. gonorrhoeae* infections. Of cases, 290 (71%) were treated non-empirically. The average time to treatment among non-empirically treated cases (n=256) was 5.2 days (SD 4 days). After November 2015, there were 319 infections: 131 (41%) were wild-type *gyr*A genotypes, 92 (29%) mutant, 92 indeterminate and 4 were not attempted. Of the 92 indeterminate results 68 (74%) were from the pharynx, compared to 24 (26%) from other sites (p-value<0.001). Among non-empirically treated cases, ceftriaxone was used in 119 (96%) of 124 before versus 132 (72%) of 184 after assay introduction (p-value<0.001). Among 59 non-empirically treated wild-type *gyr*A infections, 17 (29%) were treated with ciprofloxacin; 2 (9%) of 23 before electronic reminders began compared to 15 (50%) of 30 after (p-value=0.001), six cases had missing data. There was no ciprofloxacin use prior to assay implementation.

Conclusion A large health system successfully implemented routine *N. gonorrhoeae gyr*A genotyping with a reduction in ceftriaxone use. Targeted ciprofloxacin therapy increased with the use of electronic provider reminders.

Oral Presentation Session 6

Host-Pathogen Interactions and Vaginal/ Urethral Microbiota

O06.1 INVESTIGATING THE INTERACTION OF THE STEALTH PATHOGEN AND CAUSATIVE AGENT OF SYPHILIS, *TREPONEMA PALLIDUM*, WITH HUMAN PLATELETS

[1]Brigette Church, [2]John R Webb, [1]Caroline E Cameron. *[1]University of Victoria, Victoria, Canada; [2]BC Cancer Agency, Victoria, Canada*

10.1136/sextrans-2017-053264.30

Introduction *Treponema pallidum* ssp. *pallidum*, the causative agent of syphilis, is a highly invasive pathogen that interacts with a diverse repertoire of host cells during infection. The pathogen invades immunologically privileged sites and crosses the placental, blood-brain, endothelial and blood-retina barriers to establish widespread infection. *Treponema pallidum* disseminates via the circulatory and lymphatic systems, avoiding the prevalent inflammatory reactions raised against other blood-borne pathogens. In this study we investigate if *T. pallidum* uses an interaction with human platelets, key mediators of homeostasis and immune surveillance, to facilitate host persistence. We demonstrate that *T. pallidum* adheres to human platelets enabling survival for an extended period, and we discuss how this interaction may aid *T. pallidum* pathogenesis.

Methods Platelet rich plasma prepared from donor blood was incubated under host-mimicking microaerophilic conditions with viable *T. pallidum*, followed by examination for *T. pallidum*-platelet interactions via darkfield microscopy and flow cytometry analyses. Viability was confirmed using microscopic and fluorescent staining methodologies.

Results *Treponema pallidum* binds both the rounded and spread morphologies of activated platelets via a polar tip structure, maintaining a firm tether under fluidic conditions. A lack of interaction between heat-killed *T. pallidum* and platelets confirmed specificity and identified heat-labile *T. pallidum* surface components as mediators of this interaction. Viability assays illustrated *T. pallidum* retained viability in platelet rich plasma for >3 days under these conditions.
Conclusion The demonstration in this study of (1) prolonged *T. pallidum* survival within human platelet rich plasma and (2) *T. pallidum*-platelet interactions indicates that platelets do not exhibit a direct antimicrobial effect on *T. pallidum* and that *T. pallidum* mediates a strong and specific interaction with human platelets. These findings may reveal a novel mechanism of host survival employed by this elusive pathogen.

O06.2 INITIAL INTERACTIONS OF HERPES SIMPLEX VIRUS WITH HUMAN SKIN DENDRITIC CELLS

[1]Cunningham AL, [1]M Kim, [1]Truong NR, [1]Sandgren KJ, [1]Harman AN, [1]Bertram KM, [2]L Bosnjak, [1]N Nasr, [3]N Olbourne, [4]S Sawleshwarkar, [2]V James, [5]K Mckinnon, [6]Cohen RC. *[1]The Westmead Institute For Medical Research And University of Sydney, Westmead, Australia; [2]The Westmead Institute for Medical Research, Westmead, Australia; [3]Sydney Institute of Plastic and Reconstructive Surgery, Sydney, Australia; [4]Western Sydney Sexual Health Centre, Parramatta, Australia; [5]Western Sydney Sexual Health Centre, Westmead, Australia; [6]Children's Hospital At Westmead, Westmead, Australia*

10.1136/sextrans-2017-053264.31

Introduction HSV2 initially infects the stratified squamous epithelium of the anogenital mucosa prior to entering nerve endings, resulting in lifelong latent infection of neurons in the dorsal root ganglia. We have recently reported that topical application of HSV-1 to the inner surface of human foreskin explants, simulating *in vivo* infection, infects epidermal Langerhans cells (LCs) which then emigrate into the dermis. Here they formed large cell clusters with dermal dendritic cells (DCs). HSV-expressing LC fragments were observed inside the dermal DCs/macrophages.
Methods To define the mechanism of this interaction, we isolated LCs and dermal DCs from large human abdominal skin specimens by flow sorting. LCs were infected with HSV2 and co-cultured with dermal DCs.
Results All infected LCs developed apoptosis and fragments of them were observed within the dermal DC cytoplasm. HSV infected LCs expressed several chemokines as RNA and protein, with corresponding receptors expressed on dermal DC subsets. These DCs also expressed several phagocytic/apoptotic receptors for phosphatidylserine. In genital herpes lesions the selective contact of CD8 T cells with one of three dermal DC subsets was observed. The distribution of CD4 T cells and contact with these DC subsets is eventually being studied.
Conclusion Thus, we conclude that a viral antigen relay takes place whereby HSV infected LCs undergo apoptosis and are taken up by dermal DCs by phagocytosis for subsequent antigen presentation, probably via different pathways for CD4 and CD8 T cells. As dendritic cells are key targets for the new generation of vaccine adjuvants these studies define potential cellular targets for mucosal vaccines.

O06.3 ANALYSIS OF THE *TREPONEMA PALLIDUM* PROTEOME FOR EVIDENCE OF HOST PROTEIN MIMICRY; IDENTIFICATION OF A MECHANISM FOR BACTERIAL PERSISTENCE AND ESTABLISHMENT OF LATENCY DURING SYPHILIS INFECTION?

Caroline Cameron, Simon Houston. *University of Victoria, Victoria, Canada*

10.1136/sextrans-2017-053264.32

Introduction The causative agent of syphilis, *Treponema pallidum*, is a highly invasive pathogen that can establish lifelong latency. Experimental evidence generated by our laboratory demonstrates a subset of *T. pallidum* proteins exhibits mimicry of host proteins, a strategy that may be used by *T. pallidum* to evade detection by the immune system and establishment of latency. Here we analysed all *T. pallidum* proteins of unknown function to assess the complete repertoire of potential host protein mimics expressed by this stealthy and highly successful pathogen.
Methods Amino acid sequences of 327 functionally unannotated protein-coding genes from *T. pallidum ssp. pallidum* (Nichols strain) were submitted to the protein fold recognition server, Phyre2. For each *T. pallidum* protein, the 20 top-ranked template matches and structural models were obtained. To identify potential *T. pallidum* host protein mimics, we analysed the source organism and functions of all high-confidence template proteins used for modelling (confidence scores/90%; alignment coverage/10%).
Results High-confidence structural predictions were generated for 51% of *T. pallidum* proteins with no assigned function (167/327). Analysis of these 167 functionally unannotated proteins identified a range of *T. pallidum* proteins predicted to adopt structural folds similar to domains from host proteins central to the processes of homeostasis and self-recognition, including Toll-like receptors, extracellular matrix components, and proteins involved in cell-signalling, complement and blood coagulation pathways.
Conclusion Our analyses have identified a complement of potential host protein mimics within *T. pallidum*. This novel finding will provide significant insight into *T. pallidum* virulence mechanisms for mediating host attachment and subverting host recognition, thereby aiding establishment of persistent infection. Our results also illustrate the power of molecular modelling for enhancing our understanding of microbial pathogenesis and disease establishment for bacterial pathogens with unique proteomes.

O06.4 HIGHER LEVELS OF A CYTOTOXIC PROTEIN, VAGINOLYSIN, IN LACTOBACILLUS-DEFICIENT COMMUNITY STATE TYPES IN THE VAGINAL MUCOSA

[1]Rebecca G Nowak, [2]Tara M Randis, [2]Purnahamsi Desai, [3]Xin He, [1]Courtney K Robinson, [2]Adam J Ratner, [1]Jacques Ravel, [1]Rebecca M Brotman. *[1]University of Maryland Baltimore, Baltimore, USA; [2]New York University, New York, USA; [3]University of Maryland, College Park, USA*

10.1136/sextrans-2017-053264.33

Introduction Bacterial cytotoxic proteins, such as vaginolysin (VLY) produced by *Gardnerella vaginalis*, are thought to be virulence factors that *in vitro* alter cell integrity and local immunity. VLY may play a significant role in bacterial vaginosis (BV), therefore we assessed whether *G. vaginalis* dominant