treatment efficacy. It also highlights the sub-optimal efficacies of emerging treatments currently in clinical trial to treat extragenital gonorrhoeal infections and suggest optimisation during clinical trials could potentially improve the efficacy of these treatments to reach the marketplace while balancing patients side effects.

S02.3 MOLECULAR TESTING FOR STI ANTIMICROBIAL RESISTANCE: TACKLING A MOVING TARGET

D Whiley*. *The University of Queensland, Brisbane, Australia*

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The gonococcus is now presenting some serious public health and clinical challenges. Notably, antibiotic-resistant *Neisseria gonorrhoeae* strains continue to emerge and spread and now threaten key gonorrhoea treatments, including ceftriaxone. Alternate treatment strategies are needed. Individualized therapy, whereby a rapid resistance test is used to inform individual patient treatment, is one proposed strategy. The use of individualised therapy in now already embedded in treatment of antibiotic (azithromycin)-resistant *Mycoplasma genitalium* infection, but is still in its infancy for the gonococcus. Key challenges exist, including knowledge gaps relating resistance mechanisms, the fact that sequence targets may change over time. These hurdles are not insurmountable, and can potentially be overcome via enhanced international collaboration and sequence/data sharing.

S02.4 CONCORDANCE BETWEEN GENOTYPIC AND PHENOTYPIC RESISTANCE, AND IMPLICATIONS FOR SURVEILLANCE OF AMR

M Cole*. *Public Health England, UK*

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Culture-based phenotypic antimicrobial susceptibility testing (AST) is the gold-standard method for determining *Neisseria gonorrhoeae* antimicrobial resistance (AMR). AMR surveillance can be enhanced by the molecular detection of resistance directly from clinical specimens, usually by nucleic-acid amplification techniques (NAATs). AMR surveillance is then possible in areas that struggle with obtaining viable cultures. NAAT AMR detection can be more sensitive and quicker than culture-based AST and can subsequently improve sample size and representativeness. Available NAATs do not however reliably detect all resistance markers, so some selection is required.

Whole-genome sequencing (WGS) is normally performed using cultured isolates and this enables detection of all known resistance markers. If the sequence is linked to culture, then the data can be used to detect unknown resistance determinants. Additional advantages of using WGS for AMR surveillance activities include the identification of AMR clones and transmission networks to further enhance *N. gonorrhoeae* molecular epidemiology.

Genotypic and phenotypic resistance concordance is high for some antimicrobials such as ciprofloxacin, so using genotypic, in place of phenotypic methods, can be done with confidence. For some antimicrobials, such as cefixime and ceftriaxone, resistance can be multi-factorial so multiple loci

detection/sequencing and appropriate algorithms need to be applied to predict resistance. Even then, the predicted resistance may not always correlate with the MIC. Azithromycin can also be a challenge as the exact mechanisms of resistance have not been identified for all isolates.

As genotypic methods alone cannot detect the emergence of novel resistance and not all resistance mechanisms are known, then appropriate sampling would be required if NAATs or WGS were to be solely used in surveillance studies. It is likely that AST via phenotypic methods has a role in AMR surveillance for some time to come, but AMR surveillance would certainly be enhanced by genotypic resistance detection.

S03.1 ROLE OF MYCOPLASMA GENITALIUM IN SYMPTOMS AND SYNDROMES IN CISGENDER WOMEN

M Trent*. *Johns Hopkins Schools of Medicine and Public Health, Baltimore, Maryland, USA*

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Mycoplasma genitalium (MG) has been a known pathogen affecting reproductive health outcomes since the 1980s. However, it was not officially considered in the Centers for Disease Control and Prevention (CDC) STI treatment guidelines until 2015. The paucity of population-based epidemiological data and clinical outcomes data from randomized controlled trials limits the scope of the CDC recommendations. While increased attention to MG as a pathogen has been driven by complicated presentations of urethritis in cisgender males, MG also has significant implications for sexual and reproductive health outcomes in cisgender women, including maternal-child health outcomes. Recurrent and persistent infection may be associated with ongoing inflammation in the female genital tract that may increase secondary STI and HIV infection risk due to resulting shifts in vagina microbiota. While there are no public health control programs in the United States for MG, several other countries have recognized the importance of prioritizing MG as a public health issue. In this session, we will examine MG in the context of adolescent and young adult cisgender women's health disparities and discuss the potential for strategic interventions in practice.

S03.2 WHAT IS THE ROLE OF QUINOLONE RESISTANCE TESTING IN THE MANAGEMENT OF M. GENITALIUM?

G Murray*. *Royal Women's Hospital, Melbourne, Australia*

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In the wake of escalating macrolide resistance, resistance to fluoroquinolones has emerged as a problem requiring urgent attention. The mechanisms of resistance for macrolides (single nucleotide mutations in the 23S rRNA gene) are well defined both in vitro and clinically through analysis of treatment failure; resistance-guided therapy based on these mutations can achieve first line cure levels of over 90%. This contrasts with our understanding of the mechanisms of fluoroquinolone resistance, where multiple single nucleotide polymorphisms in the *parC* gene have been postulated to contribute, but evidence supporting a role of each mutation is limited. Currently

there are a number of commercial assays reporting various mutations linked to fluoroquinolone resistance. This talk will look at the evidence supporting the use of these assays and how these assays may contribute to patient management.

S03.3 MYCOPLASMA GENITALIUM: NEW TREATMENT APPROACHES

C Bradshaw*. *Alfred Health and Monash University, Melbourne, Australia*

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Mycoplasma genitalium is an increasingly prominent STI notable for its limited susceptibility to antimicrobials and increasing acquisition of antimicrobial resistance. While AMR varies globally, rising rates significantly impacts on the efficacy of available antimicrobials and clinicians now commonly infections that challenging to treat. This talk will discuss the range of current and new treatment strategies including resistance-guided treatment strategies, combination treatment approaches, repurposed antimicrobials and new agents

S03.4 M. GENITALIUM: IS IT TIME FOR SURVEILLANCE?

L Manhart*. *University of Washington, Seattle, Wa, USA*

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First identified in 1980, *Mycoplasma genitalium* is a recognized cause of male urethritis and is associated with increased risk of female upper reproductive tract disease and sequelae. Coinciding with the recognition of these disease associations is the emergence of significant antimicrobial resistance and potentially untreatable infections. Globally, from 2016–2017, half (51.4%) of *M. genitalium* infections were resistant to macrolides, ~9% contained fluoroquinolone resistance-associated mutations, and markers of dual class resistance were detected in ~4% (Machalek, 2020). Yet these estimates are derived largely from dedicated research studies, which are not always able to recruit and enroll representative samples of the population at risk. Surveillance systems overcome some of these limitations and are beginning to be implemented in several countries. In the US, a pilot sentinel surveillance system to monitor the prevalence of infection, and track changes in antimicrobial resistance was launched in 2020. Remnant specimens from patients tested for *Neisseria gonorrhoeae* and/or *Chlamydia trachomatis* attending eight sexual health clinics in four geographic regions of the US are tested for *M. genitalium* using the Aptima *Mycoplasma genitalium* assay (Hologic, Inc.). Each clinic submits 400 specimens: 200 from male sex at birth patients; 200 from female sex at birth patients (100 symptomatic and 100 asymptomatic for each sex) for a total of 1600 specimens per year. Macrolide and fluoroquinolone antibiotic resistance markers at positions 2058 and 2059 in *M. genitalium* 23S rRNA and in *parC* mRNA, respectively, will be assessed. The prevalence of *M. genitalium* infections, prevalence of antibiotic resistance, and characteristics associated with resistant infections from the 2020 surveillance cycle will be presented. These results will be discussed in the context of surveillance efforts in other settings.

S04.2 HOW SHOULD ANTIMICROBIAL RESISTANCE IN NEISSERIA GONORRHOEAE INFLUENCE PID TREATMENT

J Ross*. *University Hospital Birmingham NHS Trust, Birmingham, UK*

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Rising levels of antibiotic resistance in uncomplicated *N. gonorrhoeae* infection have led to changes in recommended treatment and suggestions for new management paradigms. It is, however, unclear how these new approaches to treatment should be reflected in managing gonococcal pelvic inflammatory disease (PID). *Gonorrhoea* remains an important but relatively uncommon cause of PID for which treatment is often commenced prior to identification of the causative organisms. Those at highest risk (severe symptoms, partner with *gonorrhoea*, gram negative diplococci on microscopy) should have therapy which includes cover for *N. gonorrhoeae*. Newer agents, such as lefamulin, gepotidacin and zoliflodacin have not been evaluated in women with pelvic infections but frequently have a wide spectrum of activity covering not just *gonorrhoea* but many of the other bacteria associated with PID, and achieve good tissue levels in the upper genital tract. However, optimal dosing regimens for PID to achieve clearance of infection but minimise the risk of developing future resistance need to be determined.

S04.4 EVIDENCE BASED APPROACHES TO PREVENTING PELVIC INFLAMMATORY DISEASE (PID) AND ITS SEQUELAE

P Oakeshott*. *St George's University of London, London, UK*

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There are many challenges to preventing pelvic inflammatory disease (PID). The case definition is unclear: clinically diagnosed PID may not be confirmed by laparoscopy/endometrial biopsy/imaging. Both PID and its main cause *Chlamydia trachomatis* infection can be asymptomatic, and often no clear pathogen is identified. Most women (>85%) with *chlamydia* infection do not get PID. Finally, uptake of *chlamydia* screening is often low, and treatment of those infected and their sexual partners can be challenging.

A 2016 Cochrane review found four trials (in 21, 686 women) which examined the effect of *chlamydia* screening on PID: Scholes, Ostergaard, Oakeshott, Andersen. Overall, the risk of PID was lower in women in intervention than control groups: RR 0.68, 0.49 to 0.94. But in the two trials at low risk of detection bias (Oakeshott, Andersen) the RR was 0.8, 0.55 to 1.17, compatible with no effect. Thus, although detection and treatment of *chlamydia* infection can reduce the risk of PID in an individual woman, the size of the effect is uncertain. It is also unclear whether *chlamydia* screening to prevent PID reduces rates of infertility and ectopic pregnancy.

Our recent 'Test n Treat' trial of free rapid *chlamydia* tests and same day treatment in 500 sexually active, ethnically diverse teenagers, highlights the problems of persuading those at risk to get screened. Despite a 6.2% prevalence of *chlamydia* (and 0.6% *gonorrhoea*), test uptake was only 13%. Interviews suggested this was due to not feeling at risk, perceptions of stigma and little knowledge of STIs. However, £5 notes can be effective incentives, and enabled us to recruit