conferred by a mutation in the 23s domain of ribosomal RNA. Resistance to cefixime, however, is more complex and involves two regions of the penA gene, which encodes penicillin binding protein 2. One of several mutations in the non-mosaic and mosaic regions of the penA gene have been implicated in conferring resistance. In the United Kingdom, implementation of point-of-care methods for determination of resistance patterns may improve antimicrobial stewardship at the expense of excess costs, though as testing becomes cheaper such methods are predicted to be cost-saving. Similar results have been reported in the United States. Extensive work is underway in Australia to evaluate resistance guided therapy among various populations and the implications for co-infection with other sexually transmitted infections. In this presentation, we summarize the current state diagnostic tests for genetic markers of resistance in *Neisseria gonorrhoeae*, the implementation thus far in different countries, and how such tests can be used to guide antimicrobial therapy with the hopes of stemming the rising tide of multidrug-resistant strains.

S09.2 RESISTANCE-GUIDED TREATMENT OF CHLAMYDIA

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Despite being one of the most common STIs globally, true antimicrobial resistance to recommended regimens for *Chlamydia trachomatis* is a rare occurrence. Concern about treatment failure has arisen because of high repeat *C. trachomatis* infection rates both in men and women. However, only few strains isolated from clinical treatment failure have been found to demonstrate multi-drug resistance in vitro, including resistance to macrolides and tetracyclines, and this resistance usually exhibits a heterotypic pattern. Treatment failure could be explained by differences in the efficacy of azithromycin and doxycycline, especially in rectal infections. If rectal infection is more difficult to treat with azithromycin than cervical infection, then auto-inoculation from rectal to cervical site could contribute to repeat cervical infection in women. Moreover, factors such as immune response and drugs pharmacokinetics may also play a role in treatment failure. Repeat infection does not necessarily mean treatment failure; recurrent *C. trachomatis* infections can result from reinfection from an untreated partner or new infection from a sexual partner or could be a false-positive diagnosis due to the detection of non-viable *chlamydia* acid nucleic that is still clearing after treatment. It is also possible that in the absence of genotyping, cases of lymphogranuloma venereum (LGV) will be missed, leading to treatment failure. The identification of L genovars should be performed early (preferably within 1 week) because LGV treatment requires 21 days of doxycycline compared with 7 days for an infection with a non-L genovar. Ideally, routine LGV molecular diagnostics should be available in all countries, where they could be centralized at some national reference or similar type of expert laboratory. Different in-house or laboratory-developed NAATs have been designed and used for LGV diagnosis. The recent availability of commercial real-time PCR kits will allow more laboratories to implement routine LGV diagnosis to guide treatment.

S09.3 MYCOPLASMA GENITALIUM: RESISTANCE-GUIDED TREATMENT STRATEGIES

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Syndromic treatment strategies for *Mycoplasma genitalium* have low efficacy in the context of rising antimicrobial resistance. The first generation of resistance assays that indicate macrolide susceptibility/resistance have substantially improved first line cure. However rising AMR and intrinsic resistance to many commonly used antimicrobials have substantially limited treatment options. This talk will cover the efficacy of current RGT strategies using available antimicrobials and adherence to and tolerability of this approach. With a clear need to adapt treatment strategies to rising AMR, new strategies may include quinolone resistance and near patient testing

S09.4 PREVALENCE OF MOLECULAR MARKERS FOR AZITHROMYCIN RESISTANCE FOUND IN *TREPONEMA PALLIDUM* IN BRITISH COLUMBIA, CANADA

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Introduction In 2000, BC Centre for Disease Control (BCCDC) executed a mass treatment campaign with azithromycin in an attempt to curb a syphilis outbreak. As a result of mass treatment, syphilis case numbers decreased temporarily, but within two years the outbreak began to ramp up again. An antibiotic resistance study was performed on selected samples from the past 20 years in British Columbia to understand the local trends of azithromycin-resistant syphilis in BC.

Materials/Methods BCCDC Public Health Laboratory selected positive (n=273) samples from 2000 to 2019 for azithromycin resistance testing. A two reaction RFLP (Restriction Fragment Length Polymorphism) panel was designed to detect two possible mutations in the 23S rRNA gene of *Treponema pallidum*, which confer azithromycin resistance.

Results Among samples tested, over 75% contained one of the two mutations leading to macrolide resistance. The majority of the samples had the A2058G mutation. Only four of the screened samples tested positive for the A2059G mutation. None of the samples that were tested contained both resistance-conferring mutations in the 23S rRNA gene. To note only about 8.5% (5/59) of specimens collected in the first 10 years were drug resistant. Although case of azithromycin-resistant *T. pallidum* were detected in 2000, almost no additional resistant cases were detected among selected specimens for the next 10 years, except four cases in year 2004. In contrast, the drug resistance prevalence drastically increased to 93.9% (201/214) over the next 10 years (2010–2019).

Conclusions While azithromycin is not first or second choice in recommended syphilis treatment regimen in British Columbia, it is a drug of choice for many countries in other parts of the world; increasing azithromycin drug resistance, therefore, is a real global concern. In this presentation we will discuss the positivity rate for azithromycin resistance-conferring point mutations observed in our positive *T. pallidum* PCR samples.