B), and a control group of 74 with a CTX MIC of <0.125 $\mu\text{g/ml}$ (group C), were included. All isolates were characterised using antibiograms, conventional *penA* mosaic gene PCR, and genotyping by NG-multi-locus variable-number tandem repeat analysis (MLVA). *PenA* mosaic positive isolates and a strict selection of the remaining isolates were further characterised by NG-multilocus sequence typing (MAST) and sequencing of ESC resistance determinants (*penA*, *mtrR*, and *porB1b*).

Results The majority of the isolates in group A ($n=47$; 64%) but only 11% ($n=6$) of the isolates in group B contained a *penA* mosaic allele. No *penA* mosaic-containing isolate was identified in group C (see Abstract O1-S03.03 figure 1). All the 53 *penA* mosaic isolates had an identical *penA* sequence (type XXXIV) and were assigned to the same MLVA cluster, which additionally included three isolates that were susceptible to CTX (MIC <0.125 $\mu\text{g/ml}$). Within this MLVA cluster, 46 (87%) of the *penA* mosaic isolates were assigned NG-MAST ST1407, and the remaining 7 isolates had closely related STs. All these *penA* mosaic isolates contained a *mtrR* promoter deletion and in 52/53 isolates the *porB1b* alterations G101K and A102N were found. Decreased susceptibility to cefixime and ceftriaxone (MIC ≥ 0.016 $\mu\text{g/ml}$) was found in 50/53 and 44/53 isolates, respectively. The mosaic *penA* MLVA cluster, containing ST1407 (87%) and closely related STs (13%), represented Dutch homosexual men (66%), patients with frequent chlamydia co-infection (32%), and commercial sex workers (7%).

Abstract O1-S03.03 Figure 1 GGD.

Conclusions A strong correlation was found between the decreased ESC susceptibility and a NG *penA* mosaic strain (ST1407) that was highly prevalent among visitors of the STI clinic Amsterdam. This strain was identified in many other countries. The rapid spread of this NG strain might be facilitated by high-risk sexual behaviour and should be monitored closely to identify potential treatment failure.

O1-S03.04 CORE GROUPS, ANTIMICROBIAL RESISTANCE AND REBOUND IN GONORRHOEA

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Background *Neisseria gonorrhoeae* (NG) is a major cause of sexually transmitted infection worldwide. Surveillance data from North America suggest that incidence has increased in recent years, after initially falling in the face of intensified control efforts, as anti-

microbial resistance in NG has increased. We evaluated the likely mechanisms behind such rebound using simple compartmental models, and explored the implications of such rebound for disease control practice.

Methods We evaluated the impact of risk-focussed treatment strategies on long-term gonorrhoea trends using risk-structured susceptible-infectious-susceptible" (SIS) compartmental models that included and excluded the possibility of antibiotic resistance in gonorrhoea transmission and control. We also examined optimal treatment strategies to minimise gonorrhoea rates when more than one antibiotic is available.

Results Model projections, consistent with previous work, showed that when antibiotic resistance is not possible, strategies that focus on treatment of highest risk individuals (the so-called "core group"), result in collapse of gonorrhoea transmission see Abstract O1-S03.04 figure 1. In contrast, in the presence of antimicrobial resistance, a focus on the core group causes rebound in incidence, with maximal dissemination of antibiotic resistance. When two antibiotics are available for treatment, we found that random assignment of treatment was most effective at delaying rebound in overall rates in the population, while the current strategy, which is to switch first-line treatment when a threshold level of resistance is reached, produced the quickest rebound.

Conclusions While previous models have shown that the targeted treatment of core-group individuals is the most effective at lowering rates of gonorrhoea, our model suggests that core group-focused treatment strategies efficiently disseminate antimicrobial resistant strains of NG, with rebound in gonorrhoea rates. This paradox poses a great dilemma to the control and prevention of gonorrhoea, especially when development of new antibiotic classes has lagged in recent years and vaccine development for gonorrhoea still faces many challenges. Our study highlighted the need for focus on non-antimicrobial strategies for the prevention and control of gonorrhoea.

Abstract O1-S03.04 Figure 1 Prevalence over time with risk group-focused treatment strategies.

O1-S03.05 COMMUNITY-ACQUIRED METHICILLIN-RESISTANT AND SUSCEPTIBLE *STAPHYLOCOCCUS AUREUS* AMONG MEN WHO HAVE SEX WITH MEN

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Background Community Acquired Methicillin Resistant *Staphylococcus aureus* (CA-MRSA) has been found more often among men who have sex with men (MSM) in some studies (USA). This study assesses the prevalence and sexual risk factors for CA-MRSA

and methicillin susceptible *Staphylococcus aureus* (MSSA) colonisation and infection among MSM in Amsterdam, the Netherlands. **Methods** MSM attending the sexually transmitted infections outpatient clinic in Amsterdam were invited to participate in this study and divided in two groups: (1) MSM with clinical signs of a skin/soft tissue infection (symptomatic group) and (2) MSM without clinical signs of such infections (asymptomatic group). Demographic characteristics, medical history, sexual behaviour, history of sexual contacts and known risk factors for colonisation with *S aureus* were collected through a self-completed questionnaire. Swabs were collected from the anterior nasal cavity, throat, perineum, penile glans and, if present, from infected skin lesions. Culture for *S aureus* was done on blood agar plates and for MRSA broth on selective chromagar plates after enrichment in broth. If MRSA was found, the sex partners of the index patient were invited for screening for MRSA.

Results Between October 2008 and April 2010 a total of 214 MSM were included in the study: 76 into the symptomatic group and 138 MSM into the asymptomatic group. The prevalence of MSSA in the nose was 36% (78/214) and in skin lesions 36% (27/76). The prevalence of MRSA was 0.9% (2/214). Both MRSA cases, one asymptomatic and one symptomatic, were HIV positive. The asymptomatic MRSA carrier had been hospitalised the previous year. None of the four sexual contacts that could be traced were colonised by MRSA. The symptomatic MRSA case had a soft tissue infection in the genital area; in this case also the nasal cavity, perineum and glans penis were positive for MRSA. No sexual contacts could be traced. There were no significant differences in age, sexual risk behaviour, drug use, history or diagnoses of sexual transmitted diseases, circumcision status or hygiene behaviour between those with and without a genital *S aureus* infection, but those infected with *S aureus* were significantly more often HIV infected (55% vs 34%; $p<0.01$).

Conclusion CA-MRSA among MSM STI outpatient clinic visitors in Amsterdam is rare. There were no indications for sexual transmission of MRSA or MSSA in this population.

01-S03.06 EVIDENCE OF CIRCULATING MACROLIDE RESISTANCE IN MYCOPLASMA GENITALIUM INFECTIONS AND DEVELOPMENT OF A RAPID ASSAY TO DETECT RESISTANCE

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Background *Mycoplasma genitalium* (Mg) is a sexually transmitted bacterium causing urethritis, cervicitis and longer term sequelae such as pelvic inflammatory disease. At Melbourne Sexual Health Centre (MSHC), Australia, from December 2007 to December 2009, 111 *M genitalium* infected patients (86 males/25 females) were treated with 1 g azithromycin with a cure rate of 69% (95% CI 60% to 77%). Resistance to macrolide antibiotics such as azithromycin occurs via mutations of the Mg 23S rRNA gene in response to exposure of sub-therapeutic doses of the drug. It is our hypothesis that the unacceptably high rate of treatment failures seen at this clinic is due to macrolide resistant Mg strains circulating in the community. To test this theory, we determined whether resistance was present in initial consult samples collected at MSHC, and we developed a rapid assay able to detect resistance during routine diagnostics.

Methods A subset of 83 Mg positive samples from patients taken prior to azithromycin was analysed in this study (62 males/20 females); 56 of these patients were subsequently cured by azithro-

mycin and 27 cases failed azithromycin. DNA sequencing was then carried out to determine each respective 23S rRNA sequence type. A real-time PCR assay coupled with high resolution melt analysis was developed to detect the mutational changes in the Mg 23S rRNA gene, dubbed MARS (Macrolide Antibiotic Resistance Screen), and was tested against a panel of known macrolide resistant Mg isolates and the subset of samples from MSHC.

Results In total, 16/83 (19%) of the pre-treatment samples tested possessed 23S rRNA mutations conveying macrolide resistance; significantly more patients who failed 1 g azithromycin had pre-existing macrolide mutations (12/27; 44%) compared to those who were cured by 1 g azithromycin (4/56; 7%); $p<0.0001$ (Abstract O1-S03.06 table 1). The MARS assay that was developed was able to identify when a 23S rRNA mutation was present in 100% of these samples.

Conclusions This data shows compelling evidence that macrolide resistance is circulating in certain populations and is attributing to a significant level of treatment failures seen in an Australian sexual health clinic. The development of a rapid molecular assay to detect resistance provides the means for clinicians to choose a more appropriate second line treatment option such as moxifloxacin, and thereby reduce transmission of resistant strains and avoiding sequelae associated with persistent infection.

Abstract O1-S03.06 Table 1 23S rRNA gene mutations present in a subset of 2007–2009 Mg positive samples from initial consult from MSHC

Sequence type	Patients failing azithromycin, n = 26 (%)	Patients cured by azithromycin, n = 56 (%)	p Value	Detection with MARS
Detection of any 23S rRNA gene mutation	12 (44)	4 (7)	<0.0001	100%
A2059G	7	3	0.01	
A2058G	4	1	0.04	
A2059C	1	—	—	
Wild type	14	52	<0.0001	100%

Epidemiology oral session 4: STI and HIV among youth

01-S04.01 INCREASING ADOLESCENT HIV PREVALENCE IN NORTHEASTERN ZIMBABWE: EVIDENCE OF LONG-TERM SURVIVORS OF MOTHER TO CHILD TRANSMISSION

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Background Longitudinal data from eastern Zimbabwe suggested an increase in HIV prevalence among 15 to 17 year olds between 2005 and 2008. Prevalence increased from 1.2% to 2.6% among adolescent males and from 2.2% to 2.8% in females (see Abstract O1-S04.01 figure 1). This is surprising given a general trend of decreasing HIV prevalence in the past decade associated with a reduction in sexual risk behaviour. It is unknown whether the increase is the result of resurgence in risky sexual behaviour, or long-term survival of infants infected perinatally during the early 1990s when prevalence was increasing exponentially among pregnant women in Zimbabwe

Methods We use data from the Manicaland HIV/STD Prevention Project collected between 2006 and 2008 to test hypotheses indicating whether adolescent HIV infections are likely sexually acquired or perinatally acquired. We use Fisher's exact test and relative risk regression to examine the association between