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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¹J Twin, ²C Bradshaw, ³S Garland, ²C Fairley, ⁴J Jensen, ³S Tabrizi. ¹Murdoch Children's Research Institute, Melbourne, Australia; ²Melbourne Sexual Health Centre, Australia; ³Royal Women's Hospital, Australia; ⁴Statens Serum Institut, Denmark

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 01-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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¹J Eaton, ²F Takavarasha, ¹S Gregson, ¹T Hallett, ²P Mason, ¹L Robertson, ¹C Schumacher, ²C Nyamukapa, ¹G Garnett. ¹School of Public Health, Imperial College London, London, UK; ²Biomedical Research and Training Institute, Zimbabwe

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