to a high of 94.9% in 1997–1998 ($p < 0.0001$). For 2692 infections with documented dates of treatment, the median time from specimen collection to treatment was 21 days (75th percentile: 33 days).

Conclusions This high school STD screening and treatment program achieved high treatment rates for both CT and NGU, although the treatment effort required strong commitments of various individuals from the screening program, the administrations in participating schools, and the local health services. Differences in treatment rates per school year reflected the dynamic interplay of these various individuals and organisations as well as the commonly high absentee, truancy, and dropout rates among students in the school district.

03-S4.06 PREDICTORS OF CLINICAL TREATMENT FAILURE AMONG MEN WITH IDIOPATHIC NGU

doi:10.1136/sextrans-2011-050109.126

¹D V Colombaro, ¹L E Manhart, ¹C M Wetmore, ²M. S Lowens, ¹N A Kay, ¹P A Totten, ²M R Golden. ¹University of Washington Seattle, USA; ²Public Health-Seattle & King County, University of Washington, Seattle, USA

Background Up to half of men with nongonococcal urethritis (NGU) have no known aetiology, yet still receive syndromic treatment. Identifying characteristics associated with clinical treatment failure may aid in determining the aetiology of these cases.

Methods From 1 January 2007 to 31 December 2010, 553 men entered a randomised double-blind treatment trial for NGU at the Public Health Seattle & King County STD clinic in Washington. Eligible men had visible urethral discharge or ≥ 5 PMNs/high power field on a Gram stained slide of urethral exudates. Men were randomised to either 1 g single dose azithromycin or 100 mg doxycycline twice daily for 7 days. Chlamydia trachomatis, *Neisseria gonorrhoeae*, and *Trichomonas vaginalis* were assessed by TMA (Gen-Probe, Inc., San Diego, CA); *Mycoplasma genitalium* was assessed by an in-house PCR assay. Ureaplasmas were detected by culture and speciated by a Ureaplasma urealyticum-biovar two specific PCR. Men negative for all pathogens were considered idiopathic and invited to return 2–5 weeks after enrolment. Clinical treatment failure was defined as visible urethral discharge or ≥ 5 PMNs. We evaluated baseline demographic and clinical characteristics, self-reported sexual history at enrolment, sexual practices between visits and depression as potential correlates of clinical treatment failure using log binomial regression.

Results Of the 430 (81%) men with NGU who returned for follow-up, 202 (47%) were considered idiopathic at baseline. Enrollees were 68% white and 27% black. Age ranged from 19 to 62. Fifty-one men (25%) with idiopathic NGU experienced clinical failure. In multivariate analyses, purulent discharge at enrolment more than doubled the risk of failure (ARR=2.5, 95% CI: 1.4% to 4.4%) and black men were nearly twice as likely as non-blacks to have treatment failure (ARR=1.8, 1.1 to 2.8). Age, socioeconomic status, number of partners in last 2 months, sexual orientation, sexual behaviour (anal/vaginal sex, unprotected sex between visits), depression, and other baseline clinical characteristics were not associated with treatment failure see Abstract 03-S4.06 table 1.

Conclusions Treatment failure was common among men with idiopathic NGU and associated with black race and purulent discharge at enrolment. The association with purulent discharge suggests an etiologic agent that evokes a robust immune response. Insofar as race defines sexual networks, an etiologic agent present in the network may explain the observed differential risk of persistent NGU.

Abstract 03-S4.06 Table 1 Measures of risk for clinical failure among men treated for idiopathic NGU

Characteristics	Univariate analysis		Multivariate analysis	
	RR (95% CI)	p Value	RR (95% CI)	p Value
Demographics				
Age	0.99 (0.97 to 1.02)	0.591		
Black race	1.66 (1.02 to 2.70)	0.042	1.76 (1.11 to 2.80)	0.016
Low socioeconomic status*	1.24 (0.76 to 2.02)	0.394		
Baseline signs and symptoms				
PMNs ≥ 10	1.40 (0.85 to 2.31)	0.190		
Visible discharge	1.66 (0.65 to 4.21)	0.286		
Purulent discharge on exam	1.75 (0.71 to 4.28)	0.223	2.51 (1.44 to 4.35)	0.001
Discharge amount on exam		0.789		
Small	Referent	–		
Moderate	1.18 (0.70 to 1.99)	0.536		
Large	0.87 (0.25 to 3.08)	0.834		
Depression (CES-D score ≥ 10)	1.02 (0.56 to 1.87)	0.936		
Sexual behaviours				
Sex with a man in last 12 months	0.94 (0.56 to 1.59)	0.819		
Any anal insertive partners in last 2 months	0.88 (0.51 to 1.53)	0.654		
Any vaginal sex partners in last 2 months	0.79 (0.48 to 1.29)	0.343		
Number sex partners in last 2 months	1.02 (0.96 to 1.09)	0.535		
Any unprotected sex between visits	1.06 (0.56 to 1.98)	0.864		

*As measured by ability to pay full price for the clinic visit vs payment on a sliding scale.

Clinical sciences oral session 5—treatment: Syphilis, Herpes, & Bacterial Vaginosis

03-S5.01 IMPACT OF AIC316, A NOVEL ANTIVIRAL HELICASE-PRIMASE INHIBITOR, ON GENITAL HSV SHEDDING: RANDOMISED, DOUBLE-BLIND, PLACEBO-CONTROLLED TRIAL

doi:10.1136/sextrans-2011-050109.127

¹A Wald, ²S Stoelben, ³S Tyring, ⁴T Warren, ⁵C Johnston, ¹M L Huang, ²B Timmler, ²H Ruebsamen-Schaeff, ⁶L Corey, ²A Birkmann. ¹University of Washington, Seattle, USA; ²AiCuris GmbH & Co. KG Wuppertal, Germany; ³University of Texas Health Science Center Houston, USA; ⁴Westover Heights Clinic, Portland, USA; ⁵University of Washington, Seattle, USA; ⁶Fred Hutchinson Cancer Research Center, University of Washington, Seattle, USA

Background Current treatments for HSV infection are imperfect, do not completely abrogate viral shedding or transmission, and do not interrupt HSV-2—HIV interactions. AIC316 is a helicase-primase inhibitor, a new class of anti-HSV compounds that has a distinct mode of action from currently available nucleoside analogues.

Methods We investigated the safety and efficacy of AIC316 in patients with genital HSV-2 infection in a Phase 2, randomised, multicenter, parallel, double-blind, placebo-controlled trial of 4 different doses of AIC316 administered orally for 4 weeks. Participants were randomised 1:1:1:1 to one of the following dose groups: 5 mg given once daily (qd), 25 mg qd, 75 mg qd, 400 mg given once-a-week, or matching placebo. Participants in the once daily dose groups received a loading dose. During the trial period participants obtained a swab of genital secretions daily and maintained a diary of genital lesions. The primary endpoint was the rate of genital HSV shedding measured by HSV DNA PCR in the AIC316 groups vs placebo.