Epidemiology poster session 1: STI trends: HPV

P1-S1.51 PREVALENCE OF ANOGENITAL WARTS AMONG STD CLINIC PATIENTS-STD SURVEILLANCE NETWORK, USA, JANUARY 2010–SEPTEMBER 2010

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Background STD clinics routinely provide diagnostic and treatment services for anogenital warts. With the availability and increasing use of a highly effective quadrivalent vaccine against the HPV types associated with 90% of anogenital warts, the impact on patients presenting with anogenital warts to STD clinics may be significant. To be positioned to estimate the population-level impact of HPV vaccine on STD clinics, we conducted a baseline cross sectional analysis of patients with anogenital wart-related visits.

Methods We reviewed STD clinic data collected on patients seen by a clinician from 1 January 2010 to 30 September 2010 in 11 sites (38 clinics) participating in the STD Surveillance Network (SSuN)—Seattle, WA (1 clinic); San Francisco, CA (1); Los Angeles, CA (12); Denver, CO (1); Chicago, IL (5); New Orleans, LA (1); Birmingham, AL (1); Richmond, VA (3); Baltimore, MD (2); Philadelphia, PA (2); New York City, NY (9). SSuN uses a collaboratively developed protocol to collect demographic, risk behaviour, and clinical data on all patients with anogenital warts at participating STD clinics. The unit of analysis was unique patients; patients were considered to have anogenital warts if warts were identified at any visit.

Results Among SSuN sites, 3–13% (median 4%) of STD clinic patients had anogenital wart-related visits, with 5063 patients presenting for 6989 visits. Among patients with anogenital warts, 20% of the patients had multiple anogenital warts-related visits (range 2–26 visits). Overall, the median prevalence rate was 2% (range 1–5%) for women and 6% (range 4–22%) for men. By age and sex, median prevalence rates were highest among women aged 20–24 at 3% (range 1–7%) and among men aged 25–29 at 8% (range 5–25%). Among men who have sex with men (MSM), the median prevalence was 7% (range 4–18%) and among men who have sex with women only (MSW) it was 6% (range 3–23%). Of patients with anogenital warts, 40% were African American, 32% were white, 21% were Hispanic compared to all clinic patients who were 58% African American, 18% white, and 18% Hispanic. 59% received treatment and most treatment (97%) was provider applied.

Conclusions The prevalence of anogenital warts among women is low in STD clinics. It may thus be difficult to monitor the impact of the HPV vaccine in women in these settings. However, the higher prevalence in MSM and MSW suggest that these clinics may provide settings in which to monitor anogenital warts in men.

P1-S1.52 INCIDENCE OF ANAL HPV AND HPV-RELATED SEQUELAE IN HIV-INFECTED AND UNINFECTED US ADOLESCENTS

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Background Immunodeficiency related to HIV infection may place HIV-infected youth at increased risk of human papillomavirus (HPV) infection and anal dysplasia. Our objective was to determine incidence of anal HPV infection and related sequelae, and factors associated with these outcomes, among adolescents who are HIV-infected or -uninfected but at-risk.

Methods We analysed data from the Reaching for Excellence in Adolescent Care and Health Project. Adolescents age 12–18 years who were behaviourally HIV-infected (n=319) or -uninfected but at risk (n=177) were recruited at 15 US sites from 1996 to 1999. Incidence rates for anal HPV, high risk anal HPV, anogenital warts, and anal dysplasia were calculated using Poisson modelling. Factors associated with these outcomes were explored using Cox proportional hazards modelling.

Results Mean age at entry was 16.8 years, and mean follow-up time for detection of anal HPV infection was 22.4 months (SD 10.8). Most participants (76%) were female, and 70% were black non-Hispanic. HIV-infected women (vs -uninfected women) had higher incidence of anal HPV (30 vs 14 per 100 person-years; p=0.002), high risk anal HPV (12 vs 5.3 per 100 person-years; p=0.04), and anogenital warts (6.7 vs 1.6 per 100 person-years; p=0.002) but not anal dysplasia. Although incidence rates of these outcomes were consistently higher among HIV-infected vs -uninfected men, they did not achieve statistical significance. Factors associated with incident anal HPV, high risk anal HPV, anogenital warts, and anal dysplasia in women are shown in the Abstract P1-S1.52 table 1 below. No factors were associated with any outcome in men.

Abstract P1-S1.52 Table 1 Factors associated with incident anal HPV and HPV-related sequelae among women

	HIV-uninfected		HIV-infected	
Outcome	**Predictor**	**HR (95% CI)**	**Predictor**	**HR (95% CI)**
Anal HPV	Cervical HPV infection	2.45 (1.01 to 5.92)	None	
High risk anal HPV	None		Smoker	3.46 (1.21 to 9.89)
			Late (vs early) CDC disease stage	2.80 (1.18 to 6.67)
Anogenital warts	None		Cervical HPV infection	4.28 (1.29 to 14.19)
			HIV viral load	1.55 (1.12 to 2.17)
Anal dysplasia	None		Late (vs early) CDC disease stage	7.02 (2.18 to 22.59)
			Ever had high risk anal HPV infection	3.72 (1.52 to 9.12)

Conclusions HIV-infected women, when compared to HIV-uninfected women, had higher rates of HPV and related sequelae. Because HIV-infected youth are at increased risk of HPV and related disease, enhanced HPV prevention efforts, such as vaccination, are warranted for this group.

P1-S1.53 ASSESSING HPV GENOTYPE PREVALENCE IN AUSTRALIAN WOMEN BY INDIGENOUS ETHNICITY PRE-VACCINATION

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Background A government funded HPV vaccination program was implemented across Australia from April 2007. The aim of this study was to ascertain whether HPV genotype prevalence, prior to

vaccination, differed significantly by Indigenous status, age group, Pap cytology status or area of residence.

Methods We used women attending for routine Pap smear, (April 2005—February 2008), as the sampling frame with 34 sites across Australia selected to include adequate numbers of Indigenous and remote-dwelling women. The final recruitment of 2620 (mean 33, range 15—66 years) included 26% (684) indigenous. DNA extracts from Thin-Prep specimens were screened by HPV AMPLICOR (Roche) and in-house HPV PCR/ELISA, with positives genotyped by LINEAR ARRAY HPV genotyping test (Roche).

Results The overall prevalence of HPV infection was 39% (95% CI—36.8 to 40.6), with high risk (HR) HPV detected in 26% (95% CI—24.8 to 28.2). Single infections were detected in 17% (95% CI—15.8 to 18.7). While multiple infections were common overall at 22%, there was no difference in proportion of multiple HPV carriage between indigenous and non-indigenous (58% of HPV positive non-Indigenous women and 56% of HPV positive Indigenous women had multiple types detected). As with single infections, multiple type infections were less prevalent with increasing age. The six most common genotypes were—HPV 16 (8.3%), 51 (5.1%), 53 (4.7%), 62 (4.3%), 89 (3.9%) and 52 (3.8%). Age-specific HPV prevalence rates were similar for Indigenous and non-Indigenous women aged ≤30, but higher for Indigenous women aged 31—40, particularly for non-vaccine targeted HR-HPV genotypes. By HPV clades for this age group, indigenous women were significantly more likely to have α 7 HPV ((45, 39, 59, 68 or 70 without 18) OR 1.9 (1.1 to 3.3) p=0.03) or α 5 group (HPV51, 26, 69, 82) with OR 2.1 (1.1 to 4.3) p=0.02). There was no significant association between Indigenous status and detection of HPV from the α 9 clade (31, 33, 35, 52, 58, 67), with or without HPV 16. Overall, HR-HPV prevalence increased from 21% in women with normal cytology, to 81% in those with high-grade lesions/cancer.

Conclusions Cross-sectional prevalence of HR-HPV was high in Australian women, with vaccine preventable genotypes observed in 13% of all women (25% in <25 year olds). Vaccination should significantly reduce vaccine related HPV infection and disease in Australian women, irrespective of indigenous status or area of residence.

P1-S1.54 HIGH-RISK HUMAN PAPILLOMA VIRUS (HPV) TYPES PREVALENCE IN 20—64-YEARS-OLD WOMEN; SLOVENIAN NATIONAL HPV PREVALENCE STUDY, 2010

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Background To estimate prevalence of 14 high-risk human papilloma virus (HPV) types among Slovenian women screened for cervical cancer.

Methods In 2010, we conducted a cross-sectional study in a convenience sample of 4469 women 20—64 years old, who were eligible for a preventive cytological examination of the cervical smear according to the criteria of the Slovenian National Cervical Cancer Screening Program, presented during the study period within a network of 16 outpatient gynaecology services with a nationally wide geographical coverage and consented to participate. We used three-step HPV genotyping strategy on cervical smear specimens positive with Digene Hybrid Capture 2 HPV DNA Test and/or Abbot Real Time High Risk HPV Test. Infection with high-risk HPV types was defined as the presence of one or more of the following 14 HPV types—HPV16, HPV18, HPV31, HPV33, HPV35, HPV39, HPV45, HPV51, HPV52, HPV56, HPV58, HPV59, HPV66 and HPV68. The owerall prevalence of high-risk HPV types as well as individual high-risk HPV types was estimated with 95% CIs Statistical analyses were performed using the STATA package version 10.0.

Results Prevalence of cervical infection with any high-risk HPV type examined was 13.1% (CI—12.1 to 14.0), prevalence of infection with HPV16 only was 3.5% (CI—3.0 to 4.1) and prevalence of infection with HPV18 was 1.0 (CI—0.7 to 1.3). The corresponding age specific prevalence estimates decreased with age and were the highest among 20—24 years old women—26.0% (CI 22.4 to 29.5), 9.2% (CI 6.8 to 11.5) and 1.9% (CI 0.8 to 3.0), respectively. Overall prevalence of infection with any high-risk HPV type examined was the lowest among participants without evidence of cervical disease 10.8% (CI 9.9 to 11.8) and increased with the severity of cervical disease to 72.5% (CI 61.7 to 83.3) in women with low grade squamous intraepithelial lesion (LSIL) and 83.7% (CI 72.2 to 95.2) in women with high grade squamous intraepithelial lesion (HSIL). Corresponding HPV16 prevalence estimates were 2.5% (CI 2.0 to 3.0), 26.1 (CI 15.5 to 36.7) and 41.9% (CI 26.5 to 57.2), and corresponding HPV18 prevalence estimates were 0.9% (CI 0.6 to 1.2), 7.3% (CI 1.0 to 13.5) and 7.0% (CI 0.0 to 14.9).

Conclusions Our results provide baseline high-risk HPV types prevalence estimates and will inform future monitoring of the impact of HPV vaccination program, including possible replacement of non-vaccine HPV types and design of effective cervical cancer screening strategies.

P1-S1.55 HIGHER SEROPREVALENCE IS ASSOCIATED WITH HPV INFECTIONS OF MUCOSAL EPITHELIUM AND INFECTIONS AT MULTIPLE SITES IN MEN

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Background Previously we observed elevated HPV seroprevalence in men who had sex with men (MSM) and men who had sex with men and women (MSMW) compared to men who had sex with women (MSW). We hypothesise that the higher seroprevalence is associated with HPV infection at multiple anatomic sites, and the presence of mucosal epithelium infection as opposed to keratinised epithelium infection. We assessed associations between HPV seropositivity and prevalent HPV infection at anal canal and external genitalia in men to test the hypothesis.

Methods Enrolment data for 1663 men, including 1474 MSW, 83 MSM and 106 MSMW, were analysed. HPV L1 VLP-based ELISA was used for serum antibody testing and Linear Array for HPV DNA testing. Associations were estimated for HPV 6 and 16, respectively, using logistic regression. MSM and MSMW were combined to form the group MSM.

Results Overall HPV 6 and 16 seroprevalence was 9.9% and 14.1%. HPV 6 and 16 DNA was present in 2.2% and 2.7% of anal samples, and 6.4% and 7.3% of genital samples. Seroprevalence of HPV 6 and 16 was significantly higher in MSM compared to MSW (32.8 vs 6.9%; 34.4 vs 11.5%). Similarly, HPV 6 and 16 DNA was more frequently detected in anal samples (9.5 vs 1.2%; 9.0 vs 1.8%) and genital samples (7.4 vs 6.2%; 9.0 vs 7.1%) of MSM than MSW. Men with simultaneous anal and genital HPV infection (AOR—9.20 and 6.79) or anal HPV infection alone (AOR—19.93 and 3.19) were significantly more likely to be HPV 6 and 16 seropositive. There was no association of seropositivity with prevalent genital HPV 6 or 16 infection. Strong positive associations were detected in MSW and MSM who were anal HPV 6 positive, regardless of coinfection with genital HPV 6. In contrast, no association was observed among MSW with anal HPV 16, with or without genital coinfection.