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.