**Results** The majority of the 11876 participants included in the analysis were White, employed, and median age was 33 years. In 2013, overall and undiagnosed HIV prevalence was 13.6% (106/782) and 3.2% (25/782) respectively. Overall undiagnosed fraction remained unchanged: 34% (45/131) in 2000 and 24% (25/106) in 2013. Undiagnosed fraction among sexual health clinic non-attenders in last year remained unchanged: 62% (23/37) in 2000; 59% (10/17) in 2013. HIV testing in the last year increased: 26% (263/997) to 60% (467/777); among undiagnosed HIV+ men, it increased from 28.6% (10/35) to 66.7% (16/24). Compared to men aged >45, men aged 15–25 (AOR: 7.47, 95% CI: 1.56–35.74); compared to sexual health clinic attenders in the last year, non-sexual health clinic attenders (AOR: 4.39, 95% CI: 1.90–10.16) were more likely to have undiagnosed HIV.
**Conclusions** HIV testing has increased yet undiagnosed HIV remains unchanged. Strategies to increase HIV testing among young MSM and in non-sexual health clinics should be developed and evaluated.
**Declaration of interest statement** AMJ has been a Governor of the Wellcome Trust since 2011. The other authors declare that they have no conflicts of interest.

### O20.6 FIRST FORCED SEX AND SEXUAL BEHAVIOUR AFFECTING PREVALENCE OF HIV/AIDS AMONG MSM IN SOUTH INDIA

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**Background** Forced sex is the risk factor for psychological morbidities, HIV, and other sexually transmitted infections. Limited study on MSM are available in India and no systematic attempt has been made to know the impact of first forced sex with male and HIV. Therefore, the present study examined the prevalence of the first forced sex and its linkage with HIV infection in South India.
**Methods** The present study has been used data from the cross sectional survey known as Integrated Behavioural and Biological Assessment during 2009–10. The survey was conducted in the selected districts of states, Andhra Pradesh, Tamil Nadu and Maharashtra. The sample size of MSM was 3875. Bivariate and multivariate logistic regression analysis were used.
**Results** Those MSM who have reported of their first forced sex with males, are found more likely to be HIV positive (34.69% vs. 29.06% and OR = 1. 297, $p < 0.05$) as compared to those MSM who did not have first forced sex with male. In Tamil Nadu, prevalence of first forced sex with male among MSM highest in Salem (57.1%) followed by Madurai (56.4%), and Dharmapuri (51.2%). In Andhra Pradesh, 23.5% MSM have had first forced sex with male in Hyderabad followed by Guntur (16.8%), East Godavari (8.8%) and Vizag (4.0%).
**Conclusion** The present study has found that first forced sexual intercourse with a male is a significant risk factor for the HIV infection among MSM in South India. Therefore, there is an urgent need to control the prevalence of first forced sex and transmission of HIV infection.
**Disclosure of interest** N/A.

## O21 - HIV and co-morbidity

### O21.1 PLATELET DERIVED SOLUBLE GLYCOPROTEIN VI DECREASES PRIOR TO CORONARY EVENT IN HIV POSITIVE PATIENTS

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**Introduction** Platelets play a key role in coronary artery disease (CAD). Glycoprotein VI (GPVI) is a platelet specific collagen receptor which is shed when activated. Soluble (s) GPVI is associated with CAD in the general population and lower levels have been found in patients taking abacavir. This trial was performed to determine if sGPVI was predictive of CAD in HIV.
**Methods** 24 HIV+ subjects with CAD (HIV+ cases) with stored plasma available in the 12 months before CAD diagnosis were age and sex matched 1:2 with 46 HIV+ subjects without CAD (HIV+ controls). 41 HIV negative controls (healthy controls) were used as comparators. HIV+ patients had two samples analysed; 12 and 1 month before CAD diagnosis; healthy controls had a single sample analysed (202 samples in total). sGPVI was determined by ELISA.
**Results** Of the combined HIV+ subjects 63 [90%] were male; mean 51 years; 92.8% taking antiretrovirals. HIV+ subjects (combined HIV+ cases and HIV+ controls) were more likely to smoke (34 [30.6%] v's 3 [7.3%], $p < 0.001$) than healthy controls. HIV+ cases were hypertensive (13 [54.1%] v's 5 [10.8%], $p < 0.001$) and had a family history of CAD (12 [52.1%] v's 9 [25.0%], p 0.033) at higher rates than HIV+ controls. sGPVI was higher in HIV+ subjects (combined) then healthy controls (129.9 ng/ml [SD 59.5] v's 84.4 ng/ml [SD 46.1], $p < 0.001$). 12 months before event there was no difference in sGPVI between HIV+ cases and HIV+ controls (123.2 ng/ml [SD 61.7] v's 137.8 ng/ml [SD 63.5], p 0.369). 1 month before event sGPVI was significantly lower in HIV+ cases (111.1 ng/ml [SD 45.0] v's 143.9 ng/ml [SD 56.1], p 0.016).
**Conclusion** HIV+ subjects have higher sGPVI than healthy controls; sGPVI is lower prior to CAD event in HIV+. sGPVI may play an important role in promoting CAD in HIV.
**Disclosure of interest statement** No commercial funding was involved in this project.

### O21.2 ONE PROFILE OR MANY? PLASMA BIOMARKERS CXCL10, SCD163 AND SCD14 REVEAL DISTINCT ASSOCIATIONS WITH HIV TREATMENT RESPONSE, CHOICE OF TREATMENT REGIMEN, AND CARDIOVASCULAR RISK FACTORS

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**Introduction** Persistent systemic immune activation despite effective HIV treatment may be revealed by measuring plasma 'biomarker' levels. Here we investigate three established biomarkers within a well-characterised HIV cohort.
**Methods** Plasma sCD14, sCD163 and CXCL10 levels were measured by ELISA methods in 475 consecutive patients with documented CVD risk (age, ethnicity, gender, smoking, blood pressure, BMI, fasting metabolic profile), as well as HIV treatment history and immunological/virological outcomes, and analysed using multiple regression analysis.
**Results** All biomarkers were reduced with higher CD4 counts ($p < 0.05$), but showed distinct associations with virological response: CXCL10 strongly correlated with viral load ($p < 0.001$), sCD163 was significantly reduced among 'aviremic' patients only ($p = 0.02$), while sCD14 was unaffected by virological status under 10000 cpms ($p > 0.2$) however sCD14 was increased if HIV RNA viral load was >10000 cpm ($p = 0.003$). The choice of HIV treatment did not affect CXCL10, however, higher sCD163 was associated with PI's ($p = 0.05$) and lower sCD14 was associated with integrase inhibitors ($p = 0.02$). Several CVD risk factors were associated with sCD163 (age, ethnicity, HDL, BMI), with a favourable influence of Framingham score <10% ($p = 0.04$). Soluble CD14 levels were higher among smokers ($p = 0.002$), with no effect of other CVD risk factors, except age ($p = 0.045$), or overall Framingham score.
**Conclusion** These biomarkers reveal remarkably distinct associations, with levels of CXCL10 most readily explained by routinely monitored variables (viral load, CD4 counts), while sCD163 levels appear to reflect a deeper level of virological suppression as well as the influence of CVD risk factors. Levels of sCD14, which have been linked to overall mortality risk, are least associated with routinely monitored variables, with evidence of specific effects of smoking and integrase inhibitor therapy that warrant further investigation.
**Disclosure of interest statement** None to disclose.

## O21.3 PRE-THERAPY INFLAMMATION/COAGULATION ACTIVATION AND LONG-TERM CD4 RESPONSE TO ANTIRETROVIRAL THERAPY

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**Background** Pre-antiretroviral therapy (ART) inflammation/coagulation activation biomarkers predict clinical outcomes, but whether they predict CD4 response to ART initiation is unknown.
**Methods** Study cohort was a subset of 2 international trials, SMART (evaluating continuous versus interrupted ART) and FIRST (evaluating 3 first-line ART regimens with ≥2 classes). At the start of follow-up (baseline), participants had to be ART-naïve/off ART, have C-reactive protein (CRP), interleukin-6 (IL-6) and D-dimer measured and be (re)initiating ART. Using random effects linear models, we assessed the association between quartiles of each of the baseline biomarker and change in CD4 to up to 24 months after ART-initiation. Analyses adjusted for baseline CD4, study arm, follow-up time and other known confounders. Sensitivity analyses included separate analyses by trials and excluding the interrupted ART arm in SMART.
**Results** Overall, 1084 individuals (659 from SMART (26% ART naïve) and 425 from FIRST) met the eligibility criteria, providing 8264 CD4 measurements. 75% were male with the mean age of 42 years, 37% and 47% were white and black respectively, and 10% and 33%, respectively, were hepatitis B and C positive. The median (inter-quartile range) baseline CD4 (cells/$mm^3$) were 360 (265–473) overall and 416 (350–530) and 100 (22–300) in SMART and FIRST, respectively. All of the biomarkers were inversely associated with baseline CD4 in FIRST but not in SMART. Curves of CD4 change over time by pre-ART biomarker quartiles significantly overlapped for all biomarkers. In adjusted models, there was no significant relationship between baseline biomarker levels and mean change in CD4 (P for trend: CRP: 0.97; IL-6: 0.25 and D-dimer: 0.29). Sensitivity analyses yielded similar results.
**Conclusion** Pre-ART inflammation/coagulation activation markers do not predict CD4 response to ART. These biomarkers appear to influence the risk of clinical outcomes through mechanisms other than by blunting long-term CD4 gain.
**Disclosure** The SMART and FIRST studies were funded by grants from US National Institutes of Health (NIH). Kirby Institute, UNSW Australia, is funded through the Australian Government Department of Health and Ageing. No other disclosures.

## O21.4 DECREASED CELLULAR ENERGY IN THE BRAIN FRONTAL WHITE MATTER IS ASSOCIATED WITH PAST IMMUNOSUPPRESION IN CHRONIC HIV INFECTION

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**Background** Long-term brain neurochemical changes are not clearly understood in HIV-infected adults who are ageing and are otherwise clinically stable.
**Methods** Forty men (mean age = 54 ± 7), 20 HIV-infected (70% with historical AIDS, nadir CD4 ≤350, all on antiretroviral treatment and virally suppressed), and 20 demographically comparable HIV-uninfected controls were enrolled into a prospective observational cohort study. All underwent standard neuropsychological testing to determine the level of neurocognitive performance over an 18-month period, and a proton magnetic resonance spectroscopy scan of the brain frontal white matter to assess *in vivo* neurochemical information. Clinically relevant neuropsychological change over the study period was determined using neurocognitive norms for change. Major brain metabolites: Creatine, N-Acetyl Aspartate, Choline, Glutamate/Glutamine and Myo-Inositol were fitted in jMRUI in reference to water. Brain metabolites' change was determined using linear regression with a time effect, a group effect and a time*group interaction effect. Data were randomly selected from the larger baseline cohort (N = 120) for this preliminary analysis.
**Results** Over the 18 months period, neurocognitive performance change did not differ between HIV-infected and controls. Creatine significantly decreased in the HIV-infected compared to the controls ($p < 0.03$). Myo-Inositol and Glutamate/Glutamine increased as a function of time ($p < 0.0001$) but equally in both groups. When adjusting our analyses for age, we corroborated an age*group effect ($p < 0.03$) on reduced N-Acetyl Aspartate in the HIV-infected participants, while the decreasing Creatine