Abstracts presented at the
22nd International Workshop on Co-morbidities
and Adverse Drug Reactions in HIV
30 November–4 December 2020
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Non-alcoholic fatty liver disease (NAFLD) in HIV-monoinfected patients: results from the European ECHAM study

P Ingiliz1, L Assoumou2, S De Wit3, P-M Girard4, MA Valentin5, C Katlama6, C Neessi7, P Campa8, AD Hufner9, J Schulze zur Wiesch10, H Rougier4, J-P Bastard11, S Mauss9, L Serfaty12, V Ratziu12, Y Menu11, D Costagliola2, G Behrens12, M Lemoine1, J Capeau7, the ANRS-ECHAM group

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Background: Metabolic liver disease and unexplained transaminase elevations are common features in mono-infected people living with HIV (PLWH) on antiretroviral treatment (ART). They generally result from the presence of non-alcoholic fatty liver disease (NAFLD), a growing concern in this population. However, the real impact and role of NAFLD in inducing advanced liver disease in PLWH remains unknown.

Methods: We conducted a multicentre European study (ECHAM) in order to determine the prevalence and risk factors of liver steatosis and fibrosis in ART-controlled monoinfected PLWH over 40 years of age with either a metabolic syndrome (MetS) or persistently elevated liver enzymes or clinical lipodystrophy. Participants were prospectively enrolled and had a full liver and metabolic assessment including magnetic resonance imaging proton density fat fraction (MRI-PDFF), vibration-controlled transient elastography with also determination of CAP values (VCTE, Fibroscan®) and serological markers. Following our validation study, MRI-PDFF and CAP were used as the best diagnostic methods for the detection of moderate-to-severe liver steatosis and aspartate-aminotransferase (AST) to platelet ratio-index (APRI) of significant fibrosis.

Results: From March 2014 to November 2015, we enrolled 442 participants and analysed 402 of them: male (85%), median age 55 years (IQR 50–61), median BMI 26.0 kg/m² (23.6–28.7), median CD4 cell count 630/mm³ (510–832), 67% met the definition of MetS. Significant liver steatosis (MRI-PDFF ≥10%), fibrosis (APRI ≥0.5), cirrhosis (APRI ≥2) were observed in 36%, 29% and 2%, respectively. Multivariable analysis identified seven factors independently associated with steatosis: ALT (OR: 1.23 [1.16–1.31] per additional 5U), CD4 T-cell count (OR: 4.04 [1.92–8.51] per additional 500/mm³), triglycerides (OR: 1.48 [1.18–1.84] per additional 10U), ferritin (OR: 1.05 [1.03–1.07] per additional 10U), triglycerides (OR: 1.48 [1.18–1.84] per additional 10U), leptin (≥3.2 µg/l, OR: 2.12 [1.14–3.93]), HDL (<1 mmol/l for men, <1 mmol/l for women, OR: 1.83 [1.03–3.27]), non-CC PNPLA3 genetic polymorphism (OR: 1.92 [1.11–3.33]). Otherwise, three independent factors were associated with significant liver fibrosis: duration of ART exposure (OR: 1.07 [1.02–1.12] per additional year), insulin resistance (HOMA ≥2.5, 2.04 [1.25–3.35]) and MRI-PDFF ≥10% (OR: 3.21 [1.99–5.18]).

Using MRI-PDFF as a reference, the CAP technique (cutoff 280 dB/m), had good accuracy (0.86 [0.82-0.90]) for the diagnosis of moderate-to-severe steatosis.

Conclusions: MRI-PDFF and CAP (280 dB/m threshold) are valuable tools for identification of steatosis in ART-controlled monoinfected PLWH with either a metabolic syndrome or unexplained liver enzyme elevation or lipodystrophy. Significant liver steatosis is frequent and is associated with classic features of the metabolic syndrome and a high CD4 cell count. Liver fibrosis is common and some patients develop cirrhosis. Independent risk factors for liver fibrosis are duration of ART exposure and insulin resistance together with hepatic steatosis, indicating that metabolic liver disease and ART participate in liver fibrosis in this at-risk population which therefore requires screening and monitoring.
ABSTRACT 002

Antiviral Therapy 2020; 25 Suppl 1:A2

Progression of liver disease in the post direct-acting antiviral (DAA) era of HCV therapy among those with and without HIV

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¹NIAID, National Institutes of Health, Bethesda; ²CCMD, National Institutes of Health, Bethesda; ³Institute of Human Virology, University of Maryland, Baltimore, USA

Objectives: Highly effective direct-acting antiviral (DAA) therapy has led to successful HCV clearance for the overwhelming majority of treated patients with and without HIV. HIV coinfection has been shown to increase rates of liver disease progression in chronic HCV, yet little is known about long-term prognosis for persons living with HIV (PLWH) after HCV clearance. This study aims to characterize all-cause mortality and progression of liver disease in patients with chronic HCV with and without HIV in the post-DAA era.

Methods: We conducted a prospective longitudinal cohort study of 443 participants infected with HCV (n=199 coinfected with HIV) in the greater Washington, DC region. Participants were seen annually for laboratory assessments, HIV–HCV viral load determination and HCC screening as indicated. Progression of liver disease was defined as a new development of varices, ascites, jaundice, portal hypertension, hepatic encephalopathy, hepatocellular carcinoma, liver transplant or cirrhosis after enrolment. We used SAS JMP version 14 for statistical analyses.

Results: At time of enrolment, median age was 58 years. The median duration of follow-up was 39.4 months. The cohort was 74% male and 81% Black. More than half of participants (72%) achieved sustained virological response (SVR) either at enrolment or prior to progression of liver disease, censoring or death, and 16% had a diagnosis of cirrhosis at baseline.

There was no difference between PLWH and those without HIV with regard to all-cause mortality, progression of liver disease or incidence of liver-related events (death or disease progression). HCV viral clearance with achievement of SVR was associated with reduced mortality (P<0.0001), reduced progression of liver disease (P<0.0001) and reduced incidence of liver-related events (P<0.0001). Further, presence of cirrhosis was also associated with liver disease progression events (P<0.0001) and incidence of any liver-related event (P<0.0001) as well as mortality (P=0.005). BMI category, sex, history of diabetes, current smoking and history of intravenous drug use were also evaluated and were not found to be associated with overall incidence of liver-related events. See Figure 1.

Conclusions: This study affirms the importance of HCV treatment and subsequent viral clearance in protecting against progression of liver disease and mortality. In this cohort study, PLWH had similar long-term outcomes and HIV was not shown to affect liver disease progression or mortality.
Distinct lipidomic signatures between persons living with HIV and seronegative persons: combined analysis of the ACTG 5260s and the MACS/WIHS Combined Cohort Studies


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Objectives/aims: HIV and antiretroviral treatment (ART) cause metabolic derangements, but the distinct effects of HIV infection and ART on specific metabolic pathways are not known. We assessed the association of HIV and ART on the metabolome and lipidome.

Methods: We performed widely-targeted metabolic and lipidomic profiling of plasma from ART-naive persons living with HIV (PLHIV) before and after initial ART therapy in the AIDS Clinical Trials Group study A5260s and in HIV-seronegative participants from the Multicenter AIDS Cohort Study/Women’s Interagency HIV Study Combined Cohort. For this analysis, PLHIV who did not meet criteria for successful treatment (HIV RNA level <20 copies/ml by 24 and through 96 weeks of ART with no ART interruptions) were excluded. HIV-seronegative participants were frequency-matched by age, sex, race and body mass index (BMI). Metabolites and lipid subspecies were measured via mass spectrometry (Sciex 6500+ QTRAP). Orthogonal partial least squares discriminant analysis (OPLS-DA) and volcano plots were used to assess if differences in metabolites and lipid subspecies discriminated between HIV-seronegative participants and ART-naive PLHIV as well as ART groups.

Results: Of 435 participants, 218 were PLHIV (75 randomized to RAL-based, 77 to DRV/r-based, 66 to ATV/r-based ART) and 217 were HIV-seronegative. All PLHIV received a backbone of tenofovir/emtricitabine. Prior to ART initiation, PLHIV were slightly younger (37 versus 44 years), compared with seronegative participants, but race, sex, BMI and history of dyslipidemia were similar between groups. In addition, 58 (27%) PLHIV had CD4 count <200 cells/mm³ and 49 (22.5%)...
had an HIV RNA level \(>100,000\) copies/ml. OPLS-DA indicated that metabolites and lipid subspecies could distinguish between individual comparisons of HIV-seronegative participants with ART-naive PLHIV, PLHIV after 96 weeks of raltegravir (RAL)-based ART and PLHIV after 96 weeks of darunavir/ritonavir (DRV/r) or atazanavir/ritonavir (ATV/r)-based ART. Volcano plots of the metabolites identified by OPLS-DA with variable importance in projection (VIP) values \(>1\) demonstrated that ART-naive PLHIV had a unique lipidomic imprint primarily driven by higher levels of triacylglycerols (TAGs) having polyunsaturated fatty acid (FA) side chains (Figure 1A), suggesting an inflammatory predisposition. RAL-based ART was associated with a shift in the lipidome such that unsaturated TAGs were lower compared with HIV-seronegative participants (Figure 1B). However, DRV/r- or ATV/r-based ART promoted a shift in the lipidome characterized by both higher unsaturated TAGs and higher saturated TAGs with shorter mono/saturated FAs, compared with HIV-seronegative participants (Figure 1C), suggesting both a lipogenic and inflammatory propensity.

**Conclusion:** ART-naive PLHIV have a unique lipidomic signature primarily composed of higher unsaturated TAGs, suggesting an inflammatory state ameliorated by RAL-based ART. In PLHIV on ART, we observed ART class lipidomic response differences (RAL versus DRV/r or ATV/r) compared with HIV-seronegative persons that may identify individuals at higher risk for derangements in specific metabolic pathways involving lipogenesis.
ABSTRACT 004

Antiviral Therapy 2020; 25 Suppl 1:A5

Relationships between T-cell activation, RANKL and OPG expression and low bone mineral density in HIV+ and HIV- subjects in the HIV UPBEAT cohort

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Introduction: Persistent immune activation, despite antiretroviral therapy (ART), in people with HIV (PWH) has been implicated in the development of serious non-AIDS events including low bone mineral density (BMD). T-cells express receptor activator of NF-kb ligand (RANKL) and osteoprotegrin (OPG), the cytokines controlling bone resorption and formation, respectively. We explored relationships between T-cell activation, RANKL and OPG expression and low BMD in PWH and HIV- subjects within the HIV UPBEAT cohort.

Methods: HIV UPBEAT, a single-site, prospective, cohort study, recruited PWH and HIV- subjects from similar backgrounds. Demographics, clinical history, lumbar spine (LS) and femoral neck (FN) BMD measured by dual X-ray absorptiometry (DXA) were recorded. T-cell activation (CD4/CD8+CD38+HLA-DR+) and T-cell RANKL and OPG expression were evaluated in cryopreserved PBMCs by flow cytometry. Low BMD was defined as T-score <-1 in those >40 years or Z-score <-2 in those <40 years. Logistic regression was used to explore associations between covariates and LS or FNBMD, respectively.

Results: Of 219 subjects, 107 (48.8%) were PWH (68% male, 34% African, age 47 [39–53] years, 30% smokers, 100% on ART) and 112 were HIV- (48% male, 17% African, age 50 [44–56] years, 15% smokers). Compared with the HIV- group, PWH had an increased prevalence of low BMD (40 [37%] versus 14 [11%], P=0.001) and higher CD4/CD8+ T-cell activation (both P<0.004).

Activated CD4/CD8+ T-cells had significantly higher RANKL, but not OPG, expression resulting in a higher RANKL:OPG ratio compared with total CD4/CD8+ T-cell RANKL:OPG (P<0.001; Figure 1). Despite higher CD4/CD8+ activation in PWH, neither CD4/CD8+ RANKL/OPG expression nor respective RANKL:OPG ratios differed between PWH and HIV- subjects. In the subgroup with low LSBMD, CD4/CD8+ RANKL, but not OPG, expression was altered, largely attributable to increased CD4/CD8+ RANKL expression in PWH (Figure 1).

Low LSBMD was associated with being HIV+ (OR 5.76 [2.37, 14.0]; P<0.001) and increased total CD4/CD8+ RANKL expression (OR [per 5% increase] 5.25 [5.05, 5.45], P=0.01 and 5.20 [5.05, 5.40], P=0.01, respectively).

In analysis adjusted for HIV+ status, age, gender, ethnicity, BMI and smoking status, being HIV+ was independently associated with low LSBMD (OR 3.30 [1.34, 8.14]; P=0.008). Both higher CD4+ (OR 5.19 [5.03, 5.35]; P=0.02) or CD8+ (OR 5.15 [5.0, 5.30]; P=0.02) RANKL expression remained independently associated with low LSBMD. In our final model, both being HIV+ and higher CD4/CD8+ RANKL expression were independently associated with low LSBMD.

We did not find any associations between HIV status, CD4/CD8+ RANKL or OPG expression and FNBMD.

Conclusions: Despite effective ART, PWH who had low LSBMD had significant increases in CD4/CD8+ RANKL expression, without corresponding OPG increases compared with HIV- subjects. This data supports a role for the dysregulation of T-cell RANKL/OPG axis in bone loss in PWH, in particular at the LS, which may be driven by persistent T-cell activation.
Figure 1. T-cell RANKL and OPG Expression

Figure 1 shows RANKL and OPG expression (panel A) and the RANKL-OPG ratio (B) in total CD4+ and activated (CD38+ HLA-DR+) CD4+ T-cells and RANKL and OPG expression (panel C) and the RANKL-OPG ratio (D) in total CD8+ and activated (CD38+ HLA-DR+) CD8+ T-cells. Panel E and G show CD4+ and CD8+ RANKL expression in PWL and HIV- subjects stratified by the presence of low or normal LS BMD respectively. Similarly, panels F and H show CD4+ and CD8+ OPG expression in PWL and HIV- subjects stratified by the presence of low or normal LS BMD respectively.
ABSTRACT 005

Weight gain and waist-to-height ratio in people living with HIV after switching to integrase strand transfer inhibitors in Guatemala

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¹Unidad de Atención Integral de VIH e Infecciones Crónicas “Dr. Carlos Rodolfo Mejía Villatoro”, Hospital Roosevelt, Guatemala; ²Department of Medicine, Division of Infectious Diseases, Washington University in Saint Louis

Objectives: Ever since the inclusion of integrase strand transfer inhibitors (INSTI) as first-line therapy for HIV, low-to-middle income countries began simplifying antiretroviral therapy (ART) regimens to INSTI regimens to reduce costs and improve treatment availability. However, undesired weight gain and increased waist adiposity associated with INSTI use remains a concern. To date there are no studies relating these outcomes to INSTI use in Guatemala or comparing their effect with older regimens. Our objective is to compare weight gain in PLWH switched to INSTI with PLWH that did not switch treatment and to determine risk factors associated with significant weight gain and high waist-to-height ratio (WTH) in PLWH receiving care in Hospital Roosevelt in Guatemala.

Methods: Data from a prospective cohort of PLWH enrolled between July and March 2020 was analysed. We included all PLWH over the age of 18 with over 6 months of ART use and with a 6-month follow-up. Participants included in the switch group were prescribed INSTI for at least 6 months. Normality was assessed with K-S and Shapiro Wilk test. Independent samples t-test was used to determine mean difference (MD) in the weight change experienced between groups. Demographics, lifestyle, HIV parameters, anthropometrics and treatment history were compared based on significant weight gain and high WTH ratio using univariable logistic regression. Significant weight gain was defined as weight at follow-up over 5% from weight at enrolment and high WTH ratio was defined as WTH ratio above the cutoff for gender (0.579 for men, 0.587 for women).

Results: Of 166 participants enrolled, 55.4% were males and 14% had indigenous background. High WTH ratio was found in 33.7% participants and 20.5% gained over 5% of their weight at enrolment. Median follow-up time was 6.2 months. Forty-one

<table>
<thead>
<tr>
<th>Variable</th>
<th>SWG*</th>
<th>No SWG</th>
<th>p-value</th>
<th>High WTH ratio</th>
<th>Normal WTH ratio</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female gender</td>
<td>12(35.3%)</td>
<td>62 (47.0%)</td>
<td>0.250</td>
<td>36(64.3%)</td>
<td>38(34.5%)</td>
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</tr>
<tr>
<td>Age over 50 years</td>
<td>6(17.6%)</td>
<td>33 (25.0%)</td>
<td>0.497</td>
<td>16(28.6%)</td>
<td>23(20.9%)</td>
<td>0.333</td>
</tr>
<tr>
<td>Smoking</td>
<td>0(0%)</td>
<td>18 (13.6%)</td>
<td>0.026</td>
<td>5(8.9%)</td>
<td>13(11.8%)</td>
<td>0.571</td>
</tr>
<tr>
<td>Alcohol use</td>
<td>17 (50.0%)</td>
<td>36 (27.3%)</td>
<td>0.014</td>
<td>13(23.2%)</td>
<td>40(36.4%)</td>
<td>0.113</td>
</tr>
<tr>
<td>Switched to INSTI based regimen</td>
<td>7 (20.6%)</td>
<td>34 (23.8%)</td>
<td>0.658</td>
<td>12 (21.4%)</td>
<td>29 (26.4%)</td>
<td>0.570</td>
</tr>
<tr>
<td>Eats fruits and vegetables</td>
<td>2 (5.9%)</td>
<td>8 (6.1%)</td>
<td>1.000</td>
<td>5 (8.9%)</td>
<td>5 (4.5%)</td>
<td>0.308</td>
</tr>
<tr>
<td>Sustained Viral suppression</td>
<td>26 (76.5%)</td>
<td>115 (87.1%)</td>
<td>0.175</td>
<td>45 (80.4%)</td>
<td>96 (87.3%)</td>
<td>0.257</td>
</tr>
<tr>
<td>Low physical activity</td>
<td>25 (73.5%)</td>
<td>95 (72.0%)</td>
<td>1.000</td>
<td>49 (87.5%)</td>
<td>71 (64.5%)</td>
<td>0.002</td>
</tr>
</tbody>
</table>

*At follow up **Significant weight gain
participants (24%) switched to INSTI-based ART. Mean weight change was +1.90 kg in the switch-group and +1.21 kg in the no-switch group without difference among groups (MD=0.065, 95% CI: -1.39, 0.094; P=0.086). In the univariable analysis, alcohol use and smoking at follow-up were associated with significant weight gain. Female gender and low physical activity were associated with high WTH ratio (Table 1). Switching to INSTI-based regimens was not significantly different based on high WTH ratio or significant weight gain.

Discussion: Our findings are consistent with other studies that report weight gain over 1 kg after 6 months on INSTI-based regimens. However, we found no association that weight gain experienced in the switch group was different than in the no-switch group. Demographic and lifestyle factors were significantly associated with outcomes, which further implicate their influence in body composition regardless of ART. Further studies and longer follow-up times are needed to determine the long-term INSTI effect in our cohort.
**ABSTRACT O06**

*Antiviral Therapy* 2020; 25 Suppl 1:A9

Persistence of abnormal central fat distribution in adults with HIV acquired from early childhood

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**Objective:** Antiretroviral (ARV)-associated effects on body composition and obesity remain a significant concern among people living with HIV (PLWHIV). In adults infected from birth or in early life, little is known of the long-term effects of extensive ARV exposure on body composition and metabolism. This study explores the changes in body fat distribution and associated metabolic factors in relation to ARV exposure.

**Methods:** Body composition of young adults who acquired HIV early in life (n=70; perinatal or transfusion-acquired) was cross-sectionally compared to that of age- and sex-matched healthy controls (n=47). As part of a natural history cohort study, longitudinal body composition data were available for a subset of PLWHIV (n=40). Regional body fat distribution, particularly trunk-limb fat ratio, was obtained using dual energy X-ray absorptiometry (DEXA). Waist-hip ratio (WHR), BMI, fasting glucose, insulin, and lipids, total CD4, viral RNA and detailed ARV exposure history were obtained.

**Results:** Although PLWHIV had similar BMI relative to controls, WHR, trunk-limb fat ratio, HOMA-IR and triglycerides were significantly greater in PLWHIV (Table 1). WHR was positively correlated with cumulative exposure to PIs (r=0.275; P=0.02) as well as NRTIs and NNRTIs (P<0.04). The specific ARV agents significantly correlated with WHR were tenofovir DF (r=0.358; P=0.002) and nevirapine (r=0.275; P=0.02). Trunk-limb fat ratio was positively correlated with cumulative years on nelﬁnavir (r=0.346; P<0.003) and NRTIs (r=0.314; P=0.008), specifically stavudine (r=0.424; P=0.0003) and tenofovir DF (r=0.238; P<0.05). Neither WHR nor trunk-limb fat ratio were related to cumulative exposure to INSTIs.

Longitudinal analysis revealed similar CD4 T-cell count and rates of viral suppression over a median follow-up of 7 years (Table 1). Significant increases were observed in WHR, trunk-limb fat ratio, percentage body fat and percentage trunk fat. Interestingly, the rates of overweight (28% versus 53%) and obesity (12.5% versus 25%) doubled and increases in BMI correlated with longer exposure to certain ARVs during follow-up. Particularly, changes in BMI were positively correlated with longer exposure to abacavir (r=0.395; P=0.01), lamivudine (r=0.546; P=0.0003) and raltegravir (r=0.329; P=0.04). Change in trunk-limb fat ratio was positively correlated with longer exposure to stavudine (r=0.398; P=0.01) and didanosine (r=0.385; P=0.02).

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**Table 1. (Abstract O06)**

<table>
<thead>
<tr>
<th></th>
<th>Cross-sectional</th>
<th></th>
<th>HIV+ Longitudinal</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HIV+ (n=70)</td>
<td>Control (n=47)</td>
<td>Baseline</td>
<td>Last Visit</td>
</tr>
<tr>
<td>Age, y</td>
<td>26.7 ± 5.6</td>
<td>26.5 ± 4.8</td>
<td>19.7 ± 5.7</td>
<td>27.1 ± 5.3b</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>26.8 ± 7.0</td>
<td>26.2 ± 4.9</td>
<td>23.4 ± 5.3</td>
<td>26.9 ± 6.3b</td>
</tr>
<tr>
<td>Waist-hip ratio</td>
<td>0.91 ± 0.08a</td>
<td>0.84 ± 0.08</td>
<td>0.91 ± 0.08</td>
<td>0.93 ± 0.09b</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>3.9 ± 3.1a</td>
<td>2.5 ± 1.6</td>
<td>5.4 ± 8.3</td>
<td>4.8 ± 3.6</td>
</tr>
<tr>
<td>Triglyceride, mg/dL</td>
<td>120 ±102a</td>
<td>68 ± 38</td>
<td>161 ± 141</td>
<td>130 ± 109</td>
</tr>
<tr>
<td>CD4 T-cell, cells/mm³</td>
<td>542 ±358b</td>
<td>830 ± 305</td>
<td>603 ± 445</td>
<td>633 ± 370</td>
</tr>
<tr>
<td>HIV suppressed, n (%)</td>
<td>38 (54)</td>
<td></td>
<td>20 (51)</td>
<td>19 (49)</td>
</tr>
<tr>
<td>Percentage body fat</td>
<td>29.4 ± 10.5</td>
<td>28.2 ± 8.2</td>
<td>23.6 ± 10.5</td>
<td>29.2 ± 11.0b</td>
</tr>
<tr>
<td>Percentage trunk fat</td>
<td>27.9 ± 10.7</td>
<td>25.4 ± 8.3</td>
<td>22.3 ± 10.7</td>
<td>28.7 ± 11.3b</td>
</tr>
<tr>
<td>Trunk-limb fat ratio</td>
<td>0.98 ± 0.3a</td>
<td>0.76 ± 0.2</td>
<td>0.99 ± 0.37</td>
<td>1.08 ± 0.37b</td>
</tr>
</tbody>
</table>

Note: Statistics are reported as mean ± standard deviation, except as indicated.

a Significantly different from controls at *p* < 0.05

b Significantly different from baseline at *p* < 0.05
P=0.01) but was inversely correlated with emtricitabine 
(r=-0.326; P=0.04). Change in WHR was not related to 
ARV exposure during follow-up.

**Conclusion:** This study presents strong evidence for 
sustained alterations in body fat distribution with per-
sistent or worsening central adiposity in young adults 
with life-long HIV. Not only did the effects of ARV 
agents previously recognized to contribute to lipodys-
trophy persist in adulthood, but more contemporary 
ARVs were associated with weight gain. As this cohort 
ages, further evaluation of life-long ARV use and meta-
bolic risk factors are warranted to mitigate the risks of 
cardiovascular disease and the health consequences of 
obesity.
ABSTRACT 007

Antiviral Therapy 2020; 25 Suppl 1:A11

Greater increases over time in BMI, waist circumference and subcutaneous and visceral fat area among men with HIV

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Objectives: Adipose tissue (AT) abnormalities are common in people with HIV (HIV+) and are associated with metabolic disease. AT area and density may provide complimentary information. In the traditional obesity paradigm, increases in AT area are associated with decreases in density due to lipid engorgement of adipocytes. We assessed longitudinal changes in AT area and density by HIV serostatus among men in the Multicenter AIDS Cohort Study (MACS).

Methods: Men who completed MACS sub-studies CVD2 (2011–2013, 40–70 years old, no known cardiovascular disease) and CVD3 (2015–2017) underwent measurement of visceral (VAT) and subcutaneous (SAT) AT area (cm²) and density (Hounsfield Units [HU]) from single-slice L4-L5 abdominal CT scans. Wilcoxon rank sum tests compared between-group parameters.

Results: At CVD2, HIV+ (n=201) and HIV-negative (HIV-, n=126) men had median age 52 and 57 years, BMI 26 kg/m² (both groups), 35% and 28% were Black, and 12% and 7% were Hispanic, respectively. HIV+ men had current median CD4+ T-lymphocyte count 642 cells/µl, 88% HIV-1 RNA <50 copies/ml, 10 years on highly active antiretroviral therapy (HAART, 89% with historical thymidine analogue NRTI exposure). Current ART use was 51% PI-, 43% NNRTI- and 16% INSTI-based. VAT area (HIV- 137 cm², HIV+ 158 cm²; P=0.16) and density (HIV- -86 HU, HIV+ -87 HU; P=0.30) were similar by HIV serostatus, but HIV- men had greater SAT area (209 versus 170 cm²; P=0.0001) and less dense SAT (-96 versus -93 HU; P=0.04).

Over a median of 4.8 years, HIV+ men experienced small but significant increases in BMI and waist circumference, with a trend towards greater SAT and VAT area gain than HIV- men. However, changes in VAT and SAT density were similar for HIV+ and HIV- men (Table 1). Concordance in directionality of changes in SAT and VAT area (HIV+ 71%, HIV- 77%) and density (92% for both) were high, suggesting frequency of redistribution from subcutaneous to visceral depots was low and not significantly more common among HIV+ men.

Conclusions: Although HIV+ men were younger and had less SAT at study start, HIV+ men had larger increases in BMI, waist circumference, SAT area and VAT area than HIV- men over a similar time interval. Changes in VAT and SAT density did not differ by HIV serostatus, with all men generally experiencing decreases in AT density, as expected accompanying adipocyte hypertrophy during weight gain.

Table 1. (Abstract 007)
ABSTRACT 008

**Antiviral Therapy 2020; 25 Suppl 1:A12**

Expansion of CD4+ T effector memory CD45RA+ (TEMRA) cells is associated with incident diabetes in veterans with HIV

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**Objectives:** Differences in the relative distribution of T-cell subsets have been linked to a higher prevalence of cardiovascular and metabolic diseases in the general population. Given the persistent immune activation associated with HIV infection, we assessed whether T-cell subsets were associated with incident diabetes in HIV-positive and HIV-negative veterans.

**Methods:** We performed flow cytometry and functional assays on peripheral blood mononuclear cells collected from HIV-positive and HIV-negative participants in the Veterans Aging Cohort Study between 2005 and 2007 to characterize CD4+ and CD8+ memory (central, effector and effector RA+ [TEMRA]), CD57+, CD28-, and TH1, TH2, and TH17 CD4+ T-cells. We used two definitions of TEMRA cells: CD4+CD45RA+CD28-CD57+ and CD4+CD45RA+CD27- (Table 1). Cases of incident diabetes were identified by two-physician chart adjudication. Individuals were followed until the onset of diabetes, death or censored on 9/30/2015. We assessed HIV-positive and HIV-negative veterans separately using Cox proportional hazards models with incident diabetes as the outcome and T-cell subset as the main exposure, adjusted for age, race, time updated body mass index, cytomegalovirus serostatus, hepatitis C virus serostatus, alcohol use, high-density lipoprotein, low-density lipoprotein, total cholesterol, circulating inflammatory markers (interleukin-6, d-dimer and soluble CD14) and viral load and antiretroviral therapy use (HIV-positive only). We report the hazard ratio (HR)

### Table 1. Cox proportional hazards models, stratified by HIV status, assessing the relationship of baseline T cell subsets and incident diabetes adjusted for age, race, cytomegalovirus serostatus, viral load and antiretroviral therapy use (HIV-positive only), high-density lipoprotein, low-density lipoprotein, total cholesterol, time updated body mass index, hepatitis C virus, history of alcohol abuse, and circulating concentrations of interleukin-6, D-dimer and soluble CD14. CI, confidence interval; SD, standard deviation

<table>
<thead>
<tr>
<th>T cell subset</th>
<th>HIV-negative (N = 578)</th>
<th>HIV-positive (N = 1259)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hazard Ratio per SD increment (C1)</td>
<td>P value</td>
</tr>
<tr>
<td>CD4+ T cell subset</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CD4+CD45RA-CD28-CD57+ (TEMRA)</td>
<td>1.06 [0.77, 1.44]</td>
<td>0.73</td>
</tr>
<tr>
<td>CD4+CD27- (TEMRA)</td>
<td>1.10 [0.93, 1.30]</td>
<td>0.27</td>
</tr>
<tr>
<td>CD4+CD28+</td>
<td>1.03 [0.76, 1.40]</td>
<td>0.86</td>
</tr>
<tr>
<td>CD4+CD57+CD28+</td>
<td>1.01 [0.87, 1.18]</td>
<td>0.85</td>
</tr>
<tr>
<td>CD8+ T cell subset</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CD8+CD45RA-CD28-CD57+ (TEMRA)</td>
<td>0.88 [0.73, 1.07]</td>
<td>0.21</td>
</tr>
<tr>
<td>CD8+CD27- (TEMRA)</td>
<td>0.82 [0.67, 1.01]</td>
<td>0.06</td>
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<tr>
<td>CD8+CD28+</td>
<td>0.87 [0.70, 1.08]</td>
<td>0.22</td>
</tr>
<tr>
<td>CD8+CD57+CD28+</td>
<td>1.04 [0.89, 1.22]</td>
<td>0.60</td>
</tr>
</tbody>
</table>
for incident diabetes per standard deviation increment in the T-cell subset.

**Results:** A total of 1,259 HIV-positive and 578 HIV-negative individuals were diabetes free at baseline (date of blood collection) and there were 238 incident diabetes events (133 [10.6%] in HIV-positive and 105 [18.2%] in HIV-negative) over a median follow-up time of 8.6 years. Persons with HIV had a median age of 51 years, were 69% Black, 97% male, 65% virologically suppressed and 84% on antiretroviral therapy. HIV-negative individuals had a median age of 52 years, were 68% Black and 90% male. In the fully adjusted model, the risk of incident diabetes increased with higher baseline proportion of CD4+ TEMRA cells using both definitions (above) in HIV-positive persons only (HR 1.16 [1.00, 1.34]; P=0.05 and HR 1.20 [1.04, 1.38]; P=0.01; Table 1). The diabetes risk associated with CD4+ TEMRA was present even after adjustment for CMV serostatus, a known cause of TEMRA cell inflation. No other T-cell subsets were significantly associated with incident diabetes in HIV-positive or HIV-negative individuals.

**Conclusions:** In an observational cohort of veterans with and without HIV, higher baseline CD4+ TEMRA cells was associated with an increased risk of incident diabetes in HIV-positive individuals. HIV infection appears to drive expansion of terminally differentiated CD4+ memory T-cells that may predispose to the development of diabetes. Further studies in this cohort will use unsupervised analysis techniques to further characterize T-cells associated with diabetes and other comorbidities.
ABSTRACT P01

*Antiviral Therapy* 2020; 25 Suppl 1:A15

Weight gain after initiation of antiretroviral therapy in acute HIV-1

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**Background:** Excess weight gain with integrase strand transfer inhibitors (INSTIs) has been reported in some people with chronic HIV. In antiretroviral therapy (ART)-naive people, greater weight gain over 18 months was reported with dolutegravir than other agents. We hypothesized that initiating an INSTI-based regimen during acute HIV infection (AHI) would result in more weight gain than a non-INSTI-based regimen and INSTIs other than elvitegravir (EVG) would be associated with greater weight gain than EVG.

**Methods:** We performed a retrospective, observational, single centre chart review analysis of adults with AHI (Feibig Stages 1–5) who were initiated on ART and followed for 48 (±12) weeks. Changes in weight between people on INSTI- versus non-INSTI regimens were compared, and in a subgroup analysis, EVG versus non-EVG and tenofovir alafenamide (TAF) versus non-TAF were compared. Chi-square, t-test or Wilcoxon rank sum test were used, when appropriate.

**Results:** Overall median (IQR) weight change in 61 people on INSTI was 4.53 (1.22–8.36; within-group *P*<0.0001) kg. Median weight change in 58 people initiated on INSTI was 4.66 (1.22–8.43; *P*<0.0001) kg versus 1.64 (-3.08–6.57; *P* = 0.75) kg in 3 people not on INSTI (between-group *P* = 0.33). Median weight change on EVG was 4.40 (0.91–6.71; *P*<0.0001) kg versus 7.10 (4.97–13.15; *P* = 0.0001) kg for non-EVG INSTIs (between-group *P* = 0.008; Figure 1). Median weight change on TAF (*n* = 33) was 2.66 (0.81–7.53; *P* = 0.002) kg versus 5.31 (3.72–9.34; *P*<0.0001) kg in non-TAF recipients (*n* = 25; between-group *P* = 0.06). Lower baseline CD4+ T-cell count correlated with greater weight gain (*P* = 0.012). No association between weight gain and race (*P* = 0.930) or gender (*P* = 0.379) was noted.

**Conclusions:** People initiating ART during AHI gained weight over 48 weeks, with persons taking INSTIs gaining more weight, though this finding did not reach statistical significance due to small sample size. Amongst INSTI-treated persons, those not on EVG gained more weight than those on EVG. While the benefits of starting ART during AHI on immune system preservation and reservoir should not be underscored, risk and consequences of weight gain following ART initiation should be discussed when initiating ART during AHI.

![Figure 1. (Abstract P01)](image)
ABSTRACT P02

Antiviral Therapy 2020; 25 Suppl 1:A16

Body weight and lipid changes after switch to dolutegravir-based regimens in IDU and non-IDU

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Objectives: Increasing evidence from clinical trials and observational studies shows an association between use of integrase strand transfer inhibitors (INSTI), especially dolutegravir (DTG), and weight gain in people with HIV (PWH). Some studies have pointed out the importance of factors like female gender and Black African ethnicity in determining greater weight gain with DTG. Data on people with a history of intravenous drug use (IDU) is lacking. The aim of this study was to evaluate weight and lipid changes following switch to DTG over 96 weeks in IDU and non-IDU.

Methods: We conducted an observational, retrospective analysis on all subjects enrolled in the UCD ID cohort who were switched to DTG. Weight and lipids (total cholesterol, LDL, HDL, total cholesterol/HDL ratio) were recorded at the time of switch, 48 and 96 weeks post-switch, alongside subject’s demographic and clinical data.

Paired sample t-test was used to analyse weight and lipid changes within each group at 48 and 96 weeks post-switch. Non-parametric Mann-Whitney test was used to assess the difference in weight and lipid changes between IDU and non-IDU at 48 and 96 weeks post-switch to DTG.

Results: Overall, 138 were included in the analysis. Of these, 96 patients (53 IDU and 43 non-IDU) had weight recorded at baseline and 48 weeks, and 90 patients (43 IDU and 47 non-IDU) had weight recorded at baseline and 96 weeks. All patients were of Caucasian ethnicity. Demographic and clinical characteristics of the study population are summarized in Table 1.

The median (IQR) weight at 48 weeks was significantly higher than at baseline in both groups (IDU: 65 kg [56.3–76.9] to 69.5 kg [59.7–81.6]; P 0.007; non IDU: 73.45 kg [66.82–82.45] to 74.25 kg [67.02–86.87]; P 0.003). The median (IQR) weight at 96 weeks was significantly higher in non-IDU only (73.4 kg [66.3–82] to 77.6 kg [70.55–86.7]; P<0.001).

There was not a significant between-group difference in % median (IQR) weight change between baseline and 48 weeks (IDU: 2.26% [-3.81–14.93]; non-IDU: 3.43% [-0.88–8.12]; P 0.954), and between baseline and 96 weeks (IDU: -0.48% [-6.06–10]; non-IDU: 5.52% [0.72–13.33]; P 0.346). Of note, weight change in non-IDU at 96 weeks was >5%, which is generally considered clinically significant, whereas IDU were more likely to lose weight at 96 weeks, although with a high within-group variability. No significant change in lipid parameters between baseline and 48/96 weeks was observed in IDU and non-IDU.

Conclusions: A significant median weight gain at 48 weeks was observed following switch to DTG in both IDU and non-IDU, without any difference in % weight change between the two groups. Our results suggest that weight gain following switch to DTG might be a widespread phenomenon in patients with different sociodemographic characteristics.

Table 1. (Abstract P02)

<table>
<thead>
<tr>
<th>Patient’s characteristics</th>
<th>IDU (N=73)</th>
<th>NON IDU (N=66)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years): median [IQR]</td>
<td>48 [34–53.5]</td>
<td>49 [36–50]</td>
</tr>
<tr>
<td>Gender at birth (Male): n (%)</td>
<td>43 (59.7%)</td>
<td>50 (76.9%)</td>
</tr>
<tr>
<td>Risk factor for transmission in non-IDU [WAS: n (%)]</td>
<td>-</td>
<td>30 (60%)</td>
</tr>
<tr>
<td>Duration of HIV infection (years): median [IQR]</td>
<td>15 [10.7–20]</td>
<td>10 [7–14]</td>
</tr>
<tr>
<td>NADPH CYP450 count (n/ml): median [IQR]</td>
<td>206 [127, 317]</td>
<td>330 [211, 412]</td>
</tr>
<tr>
<td>CYP450 count at switch (n/ml): median [IQR]</td>
<td>479 [286, 642]</td>
<td>662 [432, 718]</td>
</tr>
<tr>
<td>Detectable viral load at switch: n (%)</td>
<td>31 (41.5%)</td>
<td>36 (54.5%)</td>
</tr>
<tr>
<td>NRTI backbone pre-switch: n (%)</td>
<td>13 (17.8%)</td>
<td>30 (45.5%)</td>
</tr>
<tr>
<td>NRTI backbone post-switch: n (%)</td>
<td>25 (34.2%)</td>
<td>32 (48.5%)</td>
</tr>
<tr>
<td>Third agent pre-switch: n (%)</td>
<td>21 (28.8%)</td>
<td>19 (28.8%)</td>
</tr>
</tbody>
</table>

Table 1. Demographic and clinical characteristics of study population. ELI: endocytosis ligand inhibitor; U11: integrase strand transfer inhibitor; PI: protease inhibitor; dNRTI: non-nucleoside reverse transcriptase inhibitor; NNRTI: non-nucleoside reverse transcriptase inhibitor; TDF: tenofovir disoproxil fumarate; FTC: emtricitabine; TMC: tenofovir alafenamide. *other medications included abacavir/lamivudine or raltegravir/virolug regimen.
ABSTRACT P03

Antiviral Therapy 2020; 25 Suppl 1:A17

Effect of tesamorelin on diabetic retinopathy and glycaemic control in HIV-infected subjects with diabetes and central adiposity

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Objectives/aim: People living with HIV (PLWH) have increased risk for excess visceral adipose tissue (VAT) which has been associated with increased risk of cardiometabolic and hepatic diseases (non-alcoholic steatohepatitis). Tesamorelin is a synthetic analogue of growth hormone releasing hormone, approved for treatment of excess VAT in PLWH. Placebo-controlled Phase III trials of tesamorelin in over 800 PLWH showed a minimal effect on haemoglobin A1c (HbA1c) in a population without diabetes mellitus (DM), but its safety in PLWH with DM has not been studied. Therefore, we conducted a Phase IV randomized, placebo-controlled, double-dummy trial in PLWH with excess VAT and DM to determine the effect of tesamorelin on glycaemic control and progression of diabetic retinopathy (DR).

Methods: PLWH with excess VAT and stable DM were randomized 2:1 to tesamorelin (2 mg/day, subcutaneous injection) or placebo for 36 months, with longitudinal assessments of waist circumference (WC) and serum IGF-1 levels. DR was assessed by Early Treatment Diabetic Retinopathy Study (ETDRS) Person scale, at baseline and at 6, 12, 24 and 36 months. Significant progression was defined as 3-step or greater progression of DR in at least one eye. Glycaemic control was assessed by change in HbA1c.

Results: Overall, 127 participants were treated for an average of 16.8 ± 12.6 months. The study population was 87.4% male, with a mean age of 55.2 ± 8.9 years and mean WC of 111.36 ± 14.40 cm. Mean HbA1c at baseline was 7.2 ± 1.3% and 7.1% had DR. IGF-1 was increased from baseline in the treatment group compared with placebo throughout all 36 months, suggesting participant adherence to tesamorelin. At 12 months, WC tended to decrease in both the tesamorelin group (-3.06 ± 6.13 cm, P=0.5) and the placebo group (-1.49 ± 10.53 cm, P=0.5), though there was no significant difference between groups (P=0.7). No participant in either arm had development or progression of DR during the treatment period and there were no significant changes in HbA1c from baseline at 12, 24 or 36 months (Table 1).

Discussion/conclusion: No difference in progression of retinopathy or glycaemic control was observed over 36 months in PLWH with excess VAT and DM randomized to tesamorelin compared with placebo, providing additional evidence for the safety of tesamorelin in this population.

Table 1. Effect of tesamorelin on DR and HbA1c (Abstract P03)

<table>
<thead>
<tr>
<th></th>
<th>Tesamorelin</th>
<th>Placebo</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>% of participants with ≥3-step progression on ETDRS Person (compared with baseline)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Both eyes [n]</td>
<td>0 [18]</td>
<td>0 [14]</td>
<td>1.0</td>
</tr>
<tr>
<td>One eye [n]</td>
<td>0 [18]</td>
<td>0 [14]</td>
<td>1.0</td>
</tr>
<tr>
<td>Difference of HbA1c from baseline (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12 months [n]</td>
<td>0.09 ±0.86 (39)</td>
<td>0.14 ±0.76 (29)</td>
<td>0.98</td>
</tr>
<tr>
<td>24 months [n]</td>
<td>0.07 ±1.02 (20)</td>
<td>0.36 ±1.03 (20)</td>
<td>0.25</td>
</tr>
<tr>
<td>36 months [n]</td>
<td>0.00 ±1.64 (26)</td>
<td>0.62 ±1.75 (20)</td>
<td>0.39</td>
</tr>
</tbody>
</table>
ABSTRACT P04

Antiviral Therapy 2020; 25 Suppl 1:A18

Dietary habits and impact on cardiovascular disease risk in HIV infection

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Background: People living with HIV (PWH) have twice the risk of cardiovascular disease (CVD) compared with the general population with data on dietary intake, a measure of socioeconomic status and a contributor to CVD risk, limited in PLWH. We aimed to investigate differences in dietary intake, calculated by food frequency questionnaire (FFQ), between PLWH and CVD risk matched controls and examine associations between HIV and subclinical CVD measured by coronary CT angiography (CCTA), adjusting for these differences.

Methods: The UPBEAT CAD substudy, examining CVD risk in PWH, enrolled participants with and without HIV over 40 years with no CVD history, matched on HIV status and CVD risk factors. Participants underwent an FFQ and CCTA to assess for subclinical CVD. Nutritional data were calculated using Nutritics dietetics software (Dublin 2020). Data are reported as median (IQR). Between-group comparisons and associations between variables and subclinical CVD were calculated using Mann–Whitney U test and logistic regression, respectively.

Results: 99 participants were included, of which 51 were PWH, median age was 49.8 (45.6–55.8) years, 73.5% male, 76.5% Caucasian and 22.4% were current smokers. PWH had lower HDL cholesterol (1.27 [1.0–1.3] versus 1.4 [1.1–1.6] in HIVneg; P=0.017), were less likely to have a family history of CVD (37% versus 58% in HIVneg; P=0.036) and more likely to be on statin therapy (49%, versus 12%, respectively; P<0.01). Other demographics and cardiovascular risk factors were similar between the groups.

Based on FFQ, PLWH had less daily intake of protein (93.2 [83.7–141.6] g versus 127.2 [98.8–181.4] g; P=0.043), caffeine (382.7 [235–1,197] mg versus 3,325 [123–18,633] mg; P=0.049) and alcohol (4 [0.02–13.48] g versus 8.9 [2.9–15.39] g; P=0.035) in PWH and HIVneg, respectively. There was no difference in total daily calorie, carbohydrate, sugar, fibre, cholesterol and fat intake between groups.

Prevalence of any coronary plaque was similar between the two groups (PWH; 33%, HIVneg 40%; P=0.494). On univariate analysis comprising the whole cohort, there was no association between either food group intake (Figure 1) or HIV status with presence of total plaque (OR 0.75 [95% CI 0.329, 1.711]) or non-calcified plaque (OR 3.1 [95% CI 0.712, 13.6]; P=0.132). Adjusting for difference in dietary intake between the groups, there was no association between food intake and presence of total plaque (OR 0.85 [95% CI 0.43, 1.69]) or non-calcified plaque (OR 1.12 [95% CI 0.37, 3.58]; P=0.85).

Figure 1. (Abstract P04)
two groups, HIV status remained not associated with either total plaque (OR 0.874 [95% CI 0.351, 2.181]) or non-calcified plaque (OR 0.474 [95% CI 0.100, 2.255]).

Conclusions: These results, the first to examine dietary impact on CVD risk in PWH, suggest differences in dietary intake may not predict subclinical CVD in PWH.
ABSTRACT P05

Antiviral Therapy 2020; 25 Suppl 1:A20

Frailty in ageing people living with HIV: a matched controlled study

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Objective: Data on the relationship between HIV infection and frailty syndrome are limited. We compared the prevalence of frailty among middle-aged people living with HIV (PLHIV) with controlled HIV-unexposed individuals.

Methods: This cross-sectional multicentric study consecutively included 200 PLHIV from the ANRS EP58 HAND 55-70 Study and 1,000 HIV-uninfected individuals from the French national CONSTANCE cohort, matched on age, sex and education level. To be included, PLHIV were aged 55–70 years, had controlled HIV disease (HIV viral load <50 copies/ml and a lymphocyte T-CD4 level >200 cells/µl for the last 24 and 12 months, respectively). A pre-defined quota of a third of participants per 5 years age ranges (55–59; 60–64; 65–70) were included. Our main outcome was a measure of frailty (>2 items) and pre-frailty (one or two items) using a proxy of the 5-item Fried score. Multivariate logistic regression was performed to assess the association between HIV and frailty/pre-frailty, adjusting for demographic, social, behavioural and comorbidity confounders. This study was sponsored by Inserm-ANRS (Institut national de la santé et de la recherche médicale - France RÉcherche Nord & Sud Sida-hiv Hepatites).

Results: Full outcome measures were available for 192 PLHIV and 822 HIV-unexposed individuals. The median age was 62 years and 84.9% were male. Among PLHIV, the median CD4 cell count was 645.5 cells/µl. The prevalence of frailty was 5.73% in PLHIV versus 1.73% in controls, and of pre-frailty 57.29% versus 52.19%. HIV was statistically associated with prefrailty/frailty when adjusting on the matching variables of age, gender and education level (OR=1.92; CI95%=1.40, 2.62). After adjusting for social and behavioural factors and comorbidities, HIV was not significantly associated with prefrailty/frailty (OR=1.26; 95% CI=0.87, 1.83). Frailty/prefrailty in PLHIV was associated with active smoking, chronic kidney disease, depression and abstinence in alcohol use disorder.

Conclusions/discussion: Prevalence of frailty is increased in an ageing PLHIV with well controlled HIV disease, but other factors than HIV are predominant. Longitudinal studies are however still needed in middle-aged PLHIV with controlled HIV viraemia to assess possible links between HIV and frailty.
The impact of COVID-19 and its response on the psychosocial wellbeing and medical care among persons living with HIV and COPD

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1Division of Pulmonary, Allergy and Critical Care, University of Pittsburgh; 2Department of Medicine, University of Pittsburgh

Aims: People with COPD and persons with HIV have been recognized as medically and socially vulnerable populations during the COVID-19 pandemic. Data on the impact of the pandemic and its response on the psychosocial wellbeing and medical care of persons living with HIV and COPD is lacking. We evaluated the mental health and health-care utilization consequences during the pandemic in this population.

Methods: We surveyed Pittsburgh HIV Lung Cohort participants from May through July 2020. Demographic and clinical data included age, sex, race and smoking history. Anxiety, depression and insomnia during the pandemic were evaluated using the General Anxiety Disorder-7 scale, Patient Health Questionnaire-9 and Insomnia Severity Index. The survey assessed for COVID-19 symptoms, health-care access/utilization and risk/protective behaviours. Continuous variables were compared between participants with and without COPD using t-test and Mann–Whitney test as appropriate and categorical variables were compared using Fisher’s exact test. Multiple and ordinal logistic regression was used to evaluate the association of COPD and any interaction effect by HIV status with anxiety, depression, insomnia, general health status, health-care utilization and risk/protective behaviours. All statistical tests were two-sided.

Results: 136 individuals were included: age 57.9 ± 9.5 years; 76.5% male; 60.3% Caucasian and 39% Black; 47% current or former smokers. Forty-two (30.9%) respondents screened positive for anxiety disorders, 35 (25.7%) had major depressive disorder and 7 (5.1%) reported insomnia. Median scores and proportions across categories by severity of symptoms did not differ by COPD status. Of 22 participants with new or worsening symptoms that could be related to COVID-19, only 10 sought medical care. Participants with COPD reported similar levels of concern about seeking care due to potential COVID-19 exposures (30.0% versus 42.9%; P=0.64), interruptions in medical care (30.4% versus 27.4%; P=0.801) and a trend to less delay in diagnostic testing as those without COPD (8.7% versus 20.4%; P=0.247). Individuals with COPD were more likely to have sought emergent or urgent care since March (30.4% versus 8.0%; P=0.007). Although all respondents reported practicing social-distancing and masking, the majority (119; 87.5%) had in fact continued interacting with people outside the household and 38 (27.9%) joined large gatherings. Those with COPD reported similar risk behaviour as those without. There was no interaction effect by HIV status. See Table 1.

Conclusions: Anxiety, depression and concern of COVID-19 exposure were prevalent among persons with and without COPD or HIV. Many respondents experienced interruptions in medical care and diagnostic testing and concern about health-care-associated COVID-19 exposure, but this was not more severe in

Table 1. (Abstract O09)

<table>
<thead>
<tr>
<th></th>
<th>All Participants</th>
<th>Participants with COPD</th>
<th>Participants without COPD</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Used telemedicine since March 1st</td>
<td>79 (59.1%)</td>
<td>12 (55.6%)</td>
<td>66 (59.4%)</td>
<td>0.972</td>
</tr>
<tr>
<td>Had an ED/urgent care visit since March 1st</td>
<td>16 (11.8%)</td>
<td>7 (30.4%)</td>
<td>9 (8.5%)</td>
<td>0.007</td>
</tr>
<tr>
<td>Experienced interruptions of medical care</td>
<td>59 (27.9%)</td>
<td>7 (30.4%)</td>
<td>51 (27.4%)</td>
<td>0.901</td>
</tr>
<tr>
<td>Experienced delay in diagnostic testing</td>
<td>25 (18.4%)</td>
<td>2 (8.7%)</td>
<td>23 (20.4%)</td>
<td>0.247</td>
</tr>
<tr>
<td>Experienced difficulty obtaining medications</td>
<td>2 (1.5%)</td>
<td>0</td>
<td>2 (1.8%)</td>
<td>1</td>
</tr>
<tr>
<td>Worried about seeking care that is not related to COVID-19 symptoms due to concern for COVID-19</td>
<td>89 (65.4%)</td>
<td>11 (50.0%)</td>
<td>78 (68.9%)</td>
<td>0.640</td>
</tr>
<tr>
<td>Delayed seeking care due to concerns for COVID-19</td>
<td>9 (6.8%)</td>
<td>2 (8.7%)</td>
<td>7 (6.2%)</td>
<td>0.648</td>
</tr>
<tr>
<td>Visited a gym, restaurant, bar, or movie theater</td>
<td>30 (23.0%)</td>
<td>7 (30.4%)</td>
<td>23 (20.4%)</td>
<td>0.493</td>
</tr>
<tr>
<td>Visited family or friends</td>
<td>119 (87.5%)</td>
<td>19 (82.8%)</td>
<td>100 (88.6%)</td>
<td>0.489</td>
</tr>
<tr>
<td>Visited an event or gathering with ≥15 people</td>
<td>38 (27.9%)</td>
<td>8 (34.8%)</td>
<td>30 (26.8%)</td>
<td>0.450</td>
</tr>
<tr>
<td>Touched on an airplane since March 1st</td>
<td>5 (3.7%)</td>
<td>1 (4.4%)</td>
<td>4 (3.5%)</td>
<td>0.363</td>
</tr>
</tbody>
</table>

22nd International Workshop on Co-morbidities and Adverse Drug Reactions in HIV
those with COPD or HIV. Despite likely higher risk of poor outcome in those with COPD, individuals with COPD were not more likely to curtail social interactions than those without COPD. HIV status also did not seem to modify the impact of COVID-19 on behaviours in this group.
ABSTRACT O10
Antiviral Therapy 2020; 25 Suppl 1:A23
Faster decline in lung function in treated HIV-positive versus HIV-negative AGEhIV cohort participants independent of smoking behaviour
SO Verboeket1,2, A Boyd1,4, FW Wit2,3, E Verheij2, MF Schim van der Loeff3,4, N Kootstra5, M van der Valk6, RP van Steenwijk7, MB Drummond8, GD Kirk8, P Reiss1,2,3, the AGEhIV Cohort Study

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Objectives/aim: We previously reported forced vital capacity (FVC) to be lower in HIV-positive versus HIV-negative participants with limited smoking exposure at time of enrolment in the AGEhIV cohort study. We now evaluate longitudinal changes in spirometry indices, accounting for smoking and other risk factors.

Methods: Pre-bronchodilator spirometry measurements from biennial AGEhIV cohort study visits over a median 6 years were analysed. Adjusted declines in 1-second forced expiratory volume (FEV1), FVC and FEV1/FVC were modelled using linear mixed-effects models and compared between HIV and smoking categories. Rates of FEV1 and FVC decline were evaluated in relation to CD4 and CD8 T-cell counts, C-reactive protein (CRP), interleukin-6, soluble CD14, soluble CD163 and intestinal fatty acid-binding protein levels in separate models.

Results: 500 HIV-positive and 481 HIV-negative participants were included, with median baseline age 53.2 versus 52.5 years (P=0.2), 89% versus 85% male (P=0.04), 89% versus 94% White (P<0.001), and 159 (32%) HIV-positive and 183 (38%) HIV-negative participants never smoked. HIV-positive participants were virally suppressed during 95% of study visits. Adjusted yearly declines in FEV1 and FVC were greater in HIV-positive than HIV-negative participants. Compared with HIV-negative participants, HIV-positive participants had an overall adjusted additional decline in FEV1 of 10.4 ml/year, P=0.0005 and FVC of 11.5 ml/year, P=0.01 (FEV1/FVC 0.07 %/year, P=0.3), with a similar trend for never smokers (FEV1 6.0 ml/year, P=0.1; FVC 9.1 ml/year, P=0.1; FEV1/FVC 0.00 %/year, P=0.9). Higher CRP
levels during follow-up were associated with accelerated declines in $\text{FEV}_1$ and FVC among HIV-positive participants.

**Conclusion(s)/discussion:** Treated HIV infection was associated with faster declines in both $\text{FEV}_1$ and FVC, but not $\text{FEV}_1$/FVC. These changes were not only dependent of smoking and may be partly driven by ongoing interstitial or (small-)airway damage, potentially related to increased inflammation.
ABSTRACT O11
Antiviral Therapy 2020; 25 Suppl 1:A25
Gp120/CXCR4 axis promotes uncoupling of NOS3 in HIV smokers
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Purpose: Human immunodeficiency virus (HIV)-associated pulmonary artery hypertension (HIV-PAH) occurs approximately in 1 out of every 200 people with HIV (PWH), which is 100–1,000 times higher than the rate of PAH in general population. Further, disproportionately higher number of PWH are current smokers than the general population. However, the role of cigarette smoking (CS) in the pathogenesis of HIV-PAH is still elusive. The current hypothesis was that CS increases the interaction between the HIV protein, envelope glycoprotein 120 (gp120) and the host chemokine receptor, C-X-C motif chemokine receptor 4 (CXCR4), which disproportionately impairs the vasodilatory function of pulmonary artery endothelial cell (PAEC) in HIV and therefore increases the risk of HIV-PAH.

Methods: Smokers and non-smokers with and without HIV-1 infection were recruited at the University of Alabama at Birmingham 1917 clinic. All HIV-positive patients were on ART and had low blood viral load. Endothelial function in the participants was recorded by evaluating flow mediated dilation (FMD) and measuring in plasma, the key readouts of the endothelial nitric oxide synthase (NOS3) signalling, a crucial enzyme involved in vascular dilation. To determine the role of CS and gp120/CXCR4 axis in endothelial dysfunction, rat pulmonary artery endothelial cells (RPAEC) were exposed to 2% cigarette smoke extract (CSE), 50 ng/ml gp120, in the presence or absence of the CXCR4 antagonist, 25 μM AMD3100 (Plerixafor). Subsequently, NOS3 expression and function was assessed in these cells.

Results: Compared with non-smokers or HIV-negative smokers, PWH who smoke (HIV-smokers) had attenuated response to FMD. The plasma levels of nitrates/nitrites and cyclic guanosine monophosphate (cGMP), a readout of NOS-derived vasodilator, nitric oxide, were low, while the levels of peroxynitrite (highly reactive oxidant formed from nitrite and hydrogen peroxide) were elevated in HIV-smokers. In vitro, exposure to CSE increased the expression of CXCR4 in RPAEC. The RPAECs challenged with gp120 in the presence of CSE, had reduced nitrite/nitrate and cGMP, and instead had increased production of NOS3-derived superoxide and peroxynitrite. This uncoupling of NOS3 activity from producing nitric oxide to superoxide and peroxynitrite was mediated by gp120+CSE dependent decrease in the phosphorylation of NOS3 at Ser1177 and increase in the phosphorylation at Thr495 (two key post-translational modifications that control NOS3 activity). The effects of gp120+CSE on NOS3 were inhibited by treating the RPAECs with the CXCR4 inhibitor, AMD3100.

Conclusions: Together, the study demonstrated that CS dependent gp120/CXCR4 signalling cascade is an important driver of endothelial dysfunction in HIV and the inhibition of CXCR4 by AMD3100 can be a potential therapeutic target for HIV-PAH.
ABSTRACT O12

Antiviral Therapy 2020; 25 Suppl 1:A26

Anti-inflammatory effects of arabinoxylan rice bran supplementation in participants with treated, suppressed HIV infection and inadequate immune reconstitution: a randomized, double-blind trial

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Objectives/aims: Arabinoxylan rice bran (MGN-3/ Biobran, Daiwa Pharmaceutical) has shown immunomodulatory effects, including augmentation of NK cell activity, inhibition of HIV replication in vitro in mononuclear cells, enhancement cytotoxic CD8 T-cell activity in HIV-infected patients, reduced exercise- and lipopolysaccharide-induced inflammation in an animal model, and reduced high sensitivity C-reactive protein (hsCRP) in irritable bowel syndrome. We evaluated the anti-inflammatory effects of arabinoxylan rice bran supplementation in virologically suppressed participants with HIV who had incomplete immune reconstitution. Markers of inflammation, microbial translocation and monocyte/macrophage activation were measured including soluble CD14 (sCD14), soluble CD163 (sCD163), lipopolysaccharide binding protein (LBP), high-sensitivity C-reactive protein (hsCRP) and interleukin-6 (IL-6). Our primary hypothesis was that arabinoxylan rice bran supplementation would reduce sCD14 levels.

Methods: We conducted a randomized, double-blind, placebo-controlled trial of 27 adults with HIV on antiretroviral therapy (ART) with CD4+ cell counts of 100–350 cells/mm3, who had been on ART with HIV-1 RNA ≤50 copies/ml for at least 24 weeks prior to entry. Participants were randomized to receive two 500 mg capsules of arabinoxylan rice bran three times daily (n=13) or two placebo capsules three times daily (n=14) for 12 weeks. Fasting blood samples were obtained in the morning at entry and week 12. Soluble CD14, sCD163, LBP and IL-6 were measured by ELISA (R&D Systems).

A total sample of 24 participants (12 per arm) would detect a clinically relevant difference of 0.07 log10 in sCD14 (and other biomarkers) between treatment versus placebo arms with 90% power and a 0.05 two-sided type I error rate, assuming the sd of the changes in log10 sCD14 from baseline to week 12 is 0.05 for both groups.

Absolute change in sCD14 (and other biomarkers) from baseline to week 12 was compared between the treatment arm and the placebo arm by a two-sided, two-sample t-test. The primary efficacy analysis was a modified intent-to-treat analysis, which included all randomized participants for whom baseline and week 12 samples were available for assays.

Results: A total of 24 participants were included in our analyses (12 rice bran and 12 placebo). Three participants were lost to follow-up. Those included had chronic HIV infection, with a median ART duration of 144 months (range 24–444). The median baseline CD4 cell count was 244 cells/mm3 (range 117–354). There was no statistically significant difference in the change in sCD14, sCD163, IL-6, hsCRP or LBP levels from baseline to 12 weeks between the treatment and placebo group (Table 1). Treatment was well tolerated with no withdrawals due to side effects.

Discussion: We found no evidence of a beneficial effect of 12 weeks of arabinoxylan rice bran supplementation on markers of inflammation and monocyte/macrophage activation in virologically suppressed participants with HIV who had inadequate immune reconstitution.

Table 1. (Abstract O12)

<table>
<thead>
<tr>
<th>Biomarker</th>
<th>Baseline</th>
<th>Week 12</th>
<th>Absolute change</th>
<th>p-value*</th>
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<tbody>
<tr>
<td>Soluble CD14 (ng/mL)</td>
<td></td>
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</tr>
<tr>
<td>Arabinoxylan Rice Bran</td>
<td>1660.5 ± 547.6</td>
<td>1653.5 ± 830.1</td>
<td>-6.0 (± 668)</td>
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<td>Placebo</td>
<td>1435.8 ± 629.6</td>
<td>1470.8 ± 741.2</td>
<td>40.8 (± 407)</td>
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<tr>
<td>Soluble CD163 (ng/mL)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arabinoxylan Rice Bran</td>
<td>752.2 ± 159.7</td>
<td>752.6 ± 204.3</td>
<td>26.4 (± 165)</td>
<td>0.96</td>
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<td>Placebo</td>
<td>753.7 ± 296.4</td>
<td>753.5 ± 307.8</td>
<td>29.9 (± 166)</td>
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<tr>
<td>IL-6 (ng/mL)</td>
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<tr>
<td>Arabinoxylan Rice Bran</td>
<td>5.7 ± 5.2</td>
<td>5.9 ± 7.3</td>
<td>0.2 (± 5.5)</td>
<td>0.31</td>
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<tr>
<td>Placebo</td>
<td>2.9 ± 1.0</td>
<td>8.6 ± 17.7</td>
<td>5.7 (± 18)</td>
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<tr>
<td>hsCRP (mg/L)</td>
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<tr>
<td>Arabinoxylan Rice Bran</td>
<td>4.6 ± 9.1</td>
<td>2.0 ± 2.5</td>
<td>-2.6 (± 7.0)</td>
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<td>12.5 ± 32.6</td>
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<td>Lipoprotein binding protein (ng/mL)</td>
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<tr>
<td>Arabinoxylan Rice Bran</td>
<td>20.2 ± 7.8</td>
<td>25.6 ± 11.9</td>
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<td>18.9 ± 8.3</td>
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<tr>
<td>CD4 (cells/mm³)</td>
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<td></td>
<td></td>
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<tr>
<td>Arabinoxylan Rice Bran</td>
<td>246.9 ± 75.2</td>
<td>279.5 ± 97.0</td>
<td>32.6 (± 22)</td>
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<tr>
<td>Placebo</td>
<td>248.4 ± 64.4</td>
<td>259.3 ± 96.5</td>
<td>-8.1 (± 35)</td>
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</tbody>
</table>

* All values are mean (± standard deviation)

* Two-tailed T-test evaluating the difference in absolute changes between the treatment groups
ABSTRACT P06

The influence of HIV infection and antiretroviral treatment on pulmonary function in individuals in an urban setting in sub-Saharan Africa

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Background and objective: With the roll-out of antiretroviral treatment (ART) life expectancy of people with HIV and hence morbidity from non-communicable diseases, including pulmonary diseases, have increased. This study aims to investigate whether HIV and ART are associated with pulmonary function, taking into account the role of tuberculosis (TB).

Methods: HIV-positive adults (ART-naive, on first- or second-line ART), and age and sex matched HIV-negative controls were included in a cross-sectional study in Johannesburg, South Africa. Spirometry was performed to determine lung function, measuring the forced expiratory volume in one second (FEV₁), the forced vital capacity (FVC) and the FEV₁/FVC ratio before (pre) and after (post) short-acting bronchodilator. The association between HIV, ART and pulmonary function was analysed using linear regression models, adjusting for age, gender, body surface area (BSA), TB and pulmonary risk factors.

Results: Overall, 548 participants (62% women) were included with a mean age of 38 (sd 9.5) years. No effect of HIV or ART on post-FEV₁ was observed in age, gender and BSA adjusted analysis. Additional adjustment for TB resulted in a higher post-FEV₁ in HIV-positive participants on ART compared with HIV-negative participants, whereas TB was associated with a decline in FEV₁. No effect of HIV and ART on post-FEV₁/FVC was observed.

Conclusions: HIV infection and ART use were not associated with a decline in pulmonary function in this urban African population. TB showed a mediating effect on the association between HIV, ART and pulmonary function.
ABSTRACT P07

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Predictors of long-term progression to chronic kidney disease for people living with HIV in Ghana

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Background: HIV infection is associated with an increased risk of progression to chronic kidney disease (CKD) and this risk is highest in people of West African descent. While antiretroviral treatment has improved outcomes overall in this domain, tenofovir and other antiretroviral therapies (ART) have shown independent associations with renal pathology including CKD and tubular dysfunction (TD). However, to date there is a limited body of research available exploring whether such findings remain consistent across sub-Saharan African populations, which has the highest rates of HIV globally. This study assessed the relationships between key factors such as ART drugs and hepatitis B virus (HBV) status with CKD and TD progression in a population based in central Ghana.

Methods: This single-centre longitudinal study enrolled patients with HIV taking ART in Ghana between 2003 and 2018. All participants required at least two eGFR measurements, one of which upon commencing ART and the second at least 3 years after this date. Hepatitis B status and ART regimen were noted for each participant. In a subgroup of these patients, markers of tubular dysfunction were additionally noted for. Multilevel model linear regression was carried out to determine predictors of worsening eGFR function. Cox proportional hazards modelling was carried out to assess for development of CKD 3 and TD.

Results: Use of tenofovir was associated with greater levels of annual eGFR decline compared with zidovudine, with a statistically significant mean difference of -1.08 ml/min/1.73 m² per year (95% CI -1.92, -0.24). Despite this, there was no statistically significant difference in the incidence of rapid progression CKD across NRTI drugs.

Nevirapine use was also associated with a statistically significant increased annual eGFR decline compared with efavirenz use (-0.78 per year [95% CI -1.39, -0.17]) and protease inhibitors (-1.55 per year, 95% CI -2.68, -0.41).

Negative HBV status was associated with a statistically significant increased annual eGFR decline compared with positive HBV status (-1.25 per year, 95% CI -2.20, -0.29) and unknown HBV status (-1.38 per year, 95% CI -2.01, -0.74). See Table 1.

Conclusions: Increased rates of eGFR decline amongst PLH in Ghana have strong statistical associations

Table 1. Factors associated with eGFR change per year – results from multi-level linear regression model incorporating all eGFR measurements recorded over a 15-year period (Abstract P07)
with both their particular HAART drug regimen and HBV status. The findings add to an important base of research investigating the links between tenofovir and renal pathology in sub-Saharan African settings. Positive HBV status had associated with more favourable eGFR change compared with negative HBV status. While this result may have been particularly vulnerable to sampling bias due to a large percentage of participants without HBV status available (45.5%), the finding may warrant further investigation.
ABSTRACT P08

Antiviral Therapy 2020; 25 Suppl 1:A30

Greater incident proteinuria in pre-diabetic men with HIV than without HIV: the Multicenter AIDS Cohort Study

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Background: In the general population, prediabetes (pre-DM) is associated with proteinuria, which is a major risk factor for chronic kidney disease. While people living with HIV (PLWH) have a higher risk of proteinuria compared with HIV-seronegative individuals, it is unknown whether among those with pre-DM, incident proteinuria differs by HIV serostatus.

Methods: Urine protein and creatinine were measured at each semi-annual visit among men in the Multicenter AIDS Cohort Study (MACS; April 2006–present). Men with an observed case of confirmed pre-DM on or after April 2006 (baseline visit), no prevalent proteinuria at the baseline visit and no use of anti-diabetic medications at the baseline visit or during follow-up, were included in the analysis. Confirmed pre-DM was defined as fasting glucose (FG) of 100–125 mg/dl confirmed within a year by an additional FG 100–125 mg/dl or haemoglobin A1c 5.7–6.4%. Incident proteinuria was defined as protein-to-creatinine ratio >200 mg/g, confirmed at a subsequent visit within a year. We used Poisson regression models to determine whether incident proteinuria in pre-DM participants differed by HIV serostatus, and among men with HIV, whether factors related to HIV treatment and disease severity were related to incident proteinuria.

Results: Between 2006 and 2019, of the 1,138 men with pre-DM (547 with HIV [MWH], 591 without HIV), 161 (14%) developed incident proteinuria (121 [22%] with HIV [MWH], 40 [7%] men without HIV) over a median of 10 years of follow-up (IQR: 5–12 years). After adjustment for age, race/ethnicity, education, smoking status, hepatitis C virus serostatus, BMI, dyslipidaemia, hypertension and eGFR ever <60 ml/min/1.73 m², MWH had an incidence of proteinuria that was 4.2 times (95% CI: 2.8, 6.4) greater than that of men without HIV ($P<0.01$). Among MWH, history of AIDS ($P=0.06$) and current CD4 cell count <500/mm³ ($P<0.01$) were both associated incident proteinuria in multivariable models, whereas HIV viraemia (>500 copies/ml) and cumulative exposure to tenofovir, stavudine or protease inhibitors showed no association.

Conclusions: Among men with pre-DM, men with HIV have a fourfold higher risk of incident proteinuria compared with men without HIV. Strategies to preserve renal function should be evaluated in this population.
ABSTRACT P09

Antiviral Therapy 2020; 25 Suppl 1:A31

Risk factors, screening, diagnosis and treatment of osteoporosis in HIV-infected adults in an HIV primary care clinic

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Objectives/aim: The HIV-positive population is ageing and with that comes emergent comorbidities such as osteoporosis. The prevalence of osteoporosis in people living with HIV (PLHIV) has been shown to be three times greater than HIV-uninfected controls. Despite often pressing needs in primary care, screening and treating chronic diseases such as osteoporosis is becoming a crucial part of care. For this reason, our objective was to assess osteoporosis risk factors, screening, diagnosis and treatment for bone disease among patients with HIV over age 50 at a multidisciplinary HIV primary care clinic.

Methods: A retrospective chart review was completed at the John Ruedy Clinic, a low-barrier HIV clinic at St. Paul’s Hospital in Vancouver, BC, Canada. HIV-positive patients ≥50 years old with ≥1 annual appointment with their clinic physician between 1 June 2016 and 1 June 2019 were included.

Results: We analysed data from 146 patients, representing 25% of the clinic’s population of patients ≥50 years old. The majority of patients were male (n=134, 92%), with a median age of 55 years and median of 15 years since HIV diagnosis. In addition to age and HIV status, patients had a median of 3 osteoporosis risk factors, and 145 patients (99%) had ≥1 risk factor. All screening was done with dual-energy X-ray absorptiometry (DXA) scans ordered by a physician. A total of 93 patients (63%) were not screened, 15 patients (10%) had previously been diagnosed with osteoporosis and 39 patients (27%) were screened with DXA. Of those 39 patients screened with DXA, 7 patients (18%) had normal bone mineral density, 22 patients (56%) were diagnosed with osteopenia and 10 patients (26%) were diagnosed with osteoporosis. Management of the 25 patients with osteoporosis included no treatment (n=6, 24%), vitamin D or calcium supplementation (n=4, 16%) and bisphosphonate treatment (n=15, 60%). Of those with osteoporosis, 17 patients were on TDF and 10 patients (59%) were changed to another antiretroviral for bone health.

Discussion/conclusions: This study showcases the high frequency of risk factors for osteoporosis in PLHIV at the John Ruedy Clinic who are ≥50 years old. Despite this, rates of screening and treatment for osteoporosis were low. Similar findings have been described elsewhere and this may be a common problem in primary care clinics for PLHIV. While the osteoporosis rates in this sample was similar to prevalence rates described in the literature, this is likely an underestimate due to under-screening of patients with a number of risk factors. Treatment uptake for osteoporosis in this population was found to be low and it is an important area to address. Optimizing the use of the multidisciplinary team in a coordinated approach to screening and treatment can help to comprehensively manage osteoporosis in this population.
ABSTRACT P10

Antiviral Therapy 2020; 25 Suppl 1:A32

Evaluation of a decentralized model of HCV treatment in a disadvantaged, inner city population; efficacy and effectiveness

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Introduction: Despite great advances in the treatment of chronic hepatitis C infection, the disease persists in many people who are not stably engaged with the health-care system. We tested the efficacy and effectiveness of a decentralized care model for HCV treatment in this population.

Methods: We designed a care model in which the primary caregiving role was assumed by a community-based, non-governmental organization (COPE) rather than the medical centre (Jacobi). COPE has performed HCV point-of-care testing (Orasure) in non-medical settings in New York City since 2012, with a seropositivity rate of 12.1% in 7,141 tests performed through 12/31/2020. In this IRB-approved study performed in the Bronx, NY in 2018–2019, subjects provided written, informed consent for serotesting and limited demographic information, plus oral consent for further evaluation, if seropositive. Upon PCR confirmation of active infection, a reflex panel of laboratory tests and an abdominal ultrasound examination were performed, then the subject was evaluated medically. Medical appropriateness and potential exclusionary comorbidities or drug interactions were determined by the physician, therapy was prescribed, and follow-up was coordinated through patient navigation from COPE, who provided other patient support as needed individually. The control group included 429 HCV+ subjects treated with dual antiviral agents between 2014–2016, in a hospital clinic. Treated subjects were matched with controls on a 1:1 basis, controlling for sex, race, HIV infection, presence or absence of advanced fibrosis and age within 5 years.

Results: A total of 126 seropositive subjects were found in 1,199 tested (10.5%). Almost all were aware of their hepatitis infections and 19 had received some form of HCV treatment at some point in the past. HCV RNA was detected in 53/104 seropositives tested. Evaluation was completed and treatment initiated in 31. End of treatment responses were seen in 90% in the study group versus 87% in matched controls (Chi Sq 0.161, P=0.69), while SVR was documented in 74% of the study group versus 87% in controls (Chi Sq 1.65, P=0.20), the former due to loss to follow-up and not to viral rebound.

Conclusion: A strikingly high proportion of antibody-positive subjects with undetectable RNA contents were seen (49%), possibly reflecting the influence of active infection on all-cause mortality. There was a high proportion of dropouts throughout the treatment cascade, though viral suppression was detected on PCR testing at least once in every treated subject. Viral outcomes using this treatment model did not differ from those in a demographically comparable treatment group treated at an academic medical center. A community-centric treatment model may successfully treat HCV infection in around one half of a population that is not being seen in a formal health-care setting.
ABSTRACT O13

Antiviral Therapy 2020; 25 Suppl 1:A33

Exposure to HIV pre-exposure prophylaxis (PrEP) decreases leukocyte mitochondrial function and gene expression profiles in vitro and alters the lipidome

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Objectives: The use of antiretroviral therapy (ART) as pre-exposure prophylaxis (PrEP) is an effective strategy for prevention of HIV acquisition. The cellular and metabolic consequences of PrEP exposure, however, have not been sufficiently described. We explored the in vitro effects of PrEP on immune cell transcriptional profiles and mitochondrial function, and the in vivo longitudinal consequences of PrEP exposure on inflammatory biomarkers and advanced lipid profiles in a cohort of people initiating PrEP (n=15).

Methods: Peripheral blood mononuclear cells (PBMCs) were isolated from people without HIV and exposed to tenofovir disoproxil fumarate (TDF) or emtricitabine (FTC) overnight. Mitochondrial mass and function were measured by flow cytometry and the Agilent XFp analyzer. Monocyte-derived macrophages (MDMs) were differentiated in 20% autologous serum for 5 days in the presence or absence of TDF or FTC, and surface marker expression, lipid uptake and efferocytosis were measured by flow cytometry. MDM gene expression was profiled using RNAseq. Plasma biomarkers were measured by ELISA and lipids were measured using mass spectrometry and the Lipidyzer platform. For statistical analyses, unpaired t-tests were used for in vitro experiments and paired t-tests were used to compare longitudinal changes in participant plasma biomarker and lipid levels.

Results: PBMCs exposed to TDF or FTC had decreased maximal oxygen consumption rate (OCR) and reduced mitochondrial mass. Exposure to PrEP medications also increased reactive oxygen species (ROS) production from monocyte subsets. Compared with MDMs cultured in medium alone, cells differentiated in the presence of TDF (829 genes) or FTC (888 genes) had significant changes in gene expression. Further, MDMs differentiated in the presence of PrEP medications had decreased mitochondrial mass and displayed increased lipid uptake and reduced efferocytosis. These functional differences were related to changes in gene expression (MerTK, COX5A/B, COX6A1, NDUFB9, STAT2, MYD88, NLRP3, SOCS6, SOD2) and protein expression (for example, CD36, Scavenger receptor A). Plasma lipid levels were also altered in vivo in individuals receiving PrEP (TDF+FTC), including significant increases in hexosylceramide (HCER) species that have been linked to cardiometabolic changes. We observed increased sVCAM-1 levels following initiation of PrEP compared with baseline. We also found that exposure of an endothelial cell line to TDF or FTC resulted in increased mRNA expression of VCAM-1 in vitro.

Conclusions: Exposure of leukocytes to TDF or FTC resulted in decreased mitochondrial function and altered functional and transcriptional profiles. These findings may have important implications for the metabolic and immunological consequences of PrEP in populations at risk for HIV acquisition. Further studies should explore the consequences of newer PrEP medications on in vitro and in vivo immune cell function and the lipidome.
ABSTRACT O14

Antiviral Therapy 2020; 25 Suppl 1:A34

Associations between testosterone usage and electrocardiographic QT interval duration in men living with and without HIV

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Objectives: Testosterone usage (T-use) may alter risk factors for sudden cardiac death in men living with HIV (MLWH). Electrocardiographic QT interval prolongation, which could potentiate ventricular arrhythmias, has previously been associated with HIV infection and, separately, associated with low testosterone levels. We investigated whether T-use shortens the QT interval duration in MLWH and HIV-uninfected (HIV-) men.

Methods: We utilized data from the Multicenter AIDS Cohort Study, a prospective, longitudinal study of HIV infection among men who have sex with men. Multivariable linear regression analyses were used to evaluate associations between T-use and QTc duration.

Results: T-use was more common in MLWH compared with HIV- men (19% versus 9%). In a multivariable regression analysis, T-use was associated with a 5.7 msec shorter QT interval (95% CI -9.5, -1.9; P=0.003); in comparison, HIV infection was associated with a 4.3 msec longer QTc (95% CI 2.1, 6.5; P<0.001). Recent T-use, compared with prior T-use, was also associated with a 6.9 msec shorter QTc (95% CI -13, -1.1; P=0.02). In contrast, there was no significant difference in QTc (beta=0.98 msec; P=0.65) between men with no T-use compared to men with prior T-use. There was no interaction between recent T-use and HIV serostatus (P=0.22).

Conclusions: This study is the first known analysis of T-use and QTc interval in MLWH. Overall, our data demonstrate that recent T-use is associated with a shorter QTc interval. Increased T-use duration above a threshold of ≥50% of visits in the preceding 5 years was associated with a shorter QTc interval while lesser T-use duration was not. Given the association of longer QTc with HIV infection and the increased prevalence of hypogonadism and low T-levels in MLWH, the shorter QTc associated with T-use observed in this study supports a protective effect on ventricular repolarization that may counteract the potentially deleterious prolongation seen with HIV infection. Further research is needed to investigate prognostic implications of T-use on clinical arrhythmic outcomes in MLWH.
ABSTRACT O15

Antiviral Therapy 2020; 25 Suppl 1:A35

Subclinical atherosclerosis in persons with HIV is associated with anti-cytomegalovirus CD4+ T-cells

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Objectives/aim: Persons with HIV (PWH) have a twofold increased risk of cardiovascular disease (CVD) compared with the general population, which persists despite suppression of plasma viraemia on antiretroviral therapy (ART) and is not explained by traditional cardiovascular risk factors including age, race, obesity, dyslipidaemia and smoking status. Accumulating evidence indicates that chronic innate and adaptive immune activation contribute to the development of CVD in PWH, which may be exacerbated by inflated T-cell responses to cytomegalovirus (CMV). In this study, we assessed relationships between circulating CD4+ T-cell subsets, including anti-CMV CD4+ T-cells, with subclinical atherosclerosis among PWH on long-term ART.

Methods: 70 PWH on a single regimen of efavirenz, emtricitabine and tenofovir with sustained plasma viral suppression and 30 HIV-negative controls, all without known CVD were included in this study. We measured CMV viraemia and CMV IgG titres. We also measured the proportion of CD4+ T-cell subsets by flow cytometry (CD3, CD4, CD8, CD45RO, CCR7, CX3CR1, CD69, CD57). CMV-specific T-cells were measured using the DYSNTHSTRYV-MHC tetramer in 10 participants with HLA-DR7. Brachial artery flow-mediated vasodilation (FMD) and carotid plaque burden were measured by ultrasound. We used linear regression models to assess the relationship of immune parameters with cardiovascular end points. Lastly, immunohistochemical and immunofluorescence stains were used to localize immune cells, including CX3CR1+ CD4+ T-cells, within coronary plaques (n=3 per group).

Results: PWH had a higher proportion of circulating CX3CR1+GPR56+CD57 (henceforth referred to as C-G-C) CD4+ T-cells (3.9% [IQR 1.7–15.1%] compared with 1.3% [IQR 0.7–4.2%] in HIV-negative controls; P=0.02). A median of 14.4% [IQR 4.7–32.7%] of the C-G-C CD4+ T-cells in the PWH had antigen receptors that recognized a single CMV glycoprotein-B epitope. A higher number of carotid plaques was positively associated with the percentage of C-G-C CD4+ T-cells (P<0.05) after adjusting for age, sex, BMI, fasting LDL, hypertension and smoking status in PWH (Figure 1). This relationship was not observed in the HIV-negative individuals. We found no association between C-G-C CD4+ T-cells and FMD. Notably, CX3CR1+ CD4+ T-cells were present in coronary plaques of PWH who died of sudden cardiac death. While the coronary lesions were at a similar advanced stage, plaques from PWH had higher percentage of CX3CR1+ (0.8 versus 0.13% median cells/mm²; P=0.09), and CD4+ T-cells (0.1 versus 0.01% median cells/mm²; P=0.09).

Conclusion(s)/discussion: Circulating C-G-C CD4+ T-cells can be CMV-specific, and this subset is significantly higher and associated with greater plaque burden in PWH. Furthermore, CX3CR1+ CD4+ T-cells are present in coronary plaque tissue from PWH and may contribute to atherosclerosis progression. Further studies will determine the functional characteristics and receptor specificity of C-G-C CD4+ T-cells present in atheroma, and whether anti-CMV therapies may have a role in reducing CVD burden in PWH.
Figure 1. Carotid plaques are associated with CX3CR1<sup>+</sup>-GPR56<sup>+</sup>-CD57<sup>+</sup> (C=Г=Г) CD4<sup>+</sup> T-cells.

(A) Relationship between circulating CD4<sup>+</sup> subsets (CD4<sub>T_EMSA</sub>, CD4<sub>T_EM</sub>, CD4 naïve, HLA-D<sup>+</sup> CD4<sup>+</sup>, CD69<sup>+</sup> CD4<sup>+</sup>, CD57<sup>+</sup> CD4<sup>+</sup>, CX<sub>CR1</sub><sup>+</sup> CD4<sup>+</sup>, GPR56<sup>+</sup> CD4<sup>+</sup> and C=Г=Г CD4<sup>+</sup> T-cells) and carotid plaque number after adjustment for sex, age, body mass index (BMI), fasting LDL, hypertension and smoking status.

(B) Removal of age from the adjusted model in (A)

(C) Circulating C=Г=Г CD4<sup>+</sup> T-cells were significantly higher in PWH with carotid plaque (p<0.05) (C). *p<0.05, **p<0.01.
ABSTRACT 016

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Early ART initiation may preserve influenza vaccine response durability

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Objectives/aims: Ageing and immunosenescence predict poor seasonal influenza vaccine responsiveness. We sought to determine whether treated HIV infection is also associated with poor vaccine responsiveness, and whether delayed antiretroviral therapy (ART) initiation or persistent immune activation affect vaccine response in this setting.

Methods: People with HIV (PWH) maintaining ART-mediated viral suppression >1 year and HIV-negative controls, aged 40–65 years, all CMV-seropositive and enriched for HIV risk factors, received seasonal influenza vaccination between 2014–2018. Total IgG titre against each year’s vaccine antigens were assessed at baseline, 1 and 4 months (M1 and M4, respectively). PWH were stratified by timing of ART initiation (within 6 months of HIV infection [early ART] versus later), and among later initiators, by nadir CD4 count (≥350, 200–350, <200 cells/mm³). Plasma KT ratio, IP-10, sCD14, sCD163, IL-6, sTNFR2 and suPAR were assessed at baseline. Antibody titre (reported as log-EC50) changes after vaccination were assessed with linear mixed-models, adjusted for demographics and health-related behaviours. Timepoint-by-group and biomarker-by-timepoint interaction terms were used to assess between-group differences and the impact of biomarkers on vaccine response at each timepoint.

Results: Of 164 PWH and 41 HIV-negative participants, median age was 54 years, 91% were men and 61% were White. Of HIV-negatives, 56% were MSM, 34% were current smokers, 15% had distant IDU and 41% had >100 lifetime male sexual partners. Of the PWH, 34 were early ART initiators, and the remainder had a range of nadir CD4 counts: >350 (n=32), 200–350 (n=43) and <200 cells/mm³ (n=55). Median duration of viral suppression was 8 years (IQR 5–11 years). Flu-specific IgG titres increased from baseline to M1 similarly in all groups. While there was no evidence for a decay in titre between M1 to M4 in HIV- and early ART initiators, late ART initiators experienced significant declines (P<0.04 for all). The extent of titre decay from M1 to M4 was impacted by ART initiation timing: compared with HIV-, the early ART group had a similar slope of decay (P=0.66), but the combined later ART groups experienced a significantly greater rate of decline (P=0.02). Among all participants, higher sCD14 and IP-10, but not other biomarkers, were associated with greater rates of titre decline M1 to M4 (P=0.05 and P=0.03), but were modest in effect size (9% greater rate of titre decline per 1 IQR increase in either biomarker). See Figure 1.

Conclusions: Compared with adults without HIV, ART-suppressed PWH have similar early humoral responses to influenza vaccination, but only those who initiate ART within the first 6 months of infection appear to maintain similar response durability at 4 months. While the clinical implications of these findings remain unclear, some pathways of immune activation appear to be associated with shorter response durability.
Figure 1. (Abstract O16)

A. Change in Influenza Titer by HIV Group

B. Change in Influenza Titer by HIV Group

* <0.05
** <0.01
*** <0.001

Log EC50

HIV-Negative
Early ART
Late ART, CD4 nadir >200
Late ART, CD4 nadir 200-350
Late ART, CD4 nadir >350
ABSTRACT P11

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Plasma LAG3 and subclinical coronary artery disease in the Multicenter AIDS Cohort Study

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Objectives: The objectives of this study were 1) to determine whether plasma LAG3, a T-cell inhibitor associated with cardiovascular disease in the general population, differed by HIV serostatus and 2) to ascertain whether LAG3 was associated with moderate to severe coronary artery stenosis, independent of systemic inflammation, in the Multicenter AIDS Cohort Study (MACS).

Methods: Plasma specimens collected 12 ± 8 months before coronary CT angiography were tested for LAG3 in men enrolled in the MACS Cardiovascular Substudy. LAG3 was natural log transformed to correct for skewness and to fit a normal distribution. Multivariable linear regression was used to determine if HIV serostatus was associated with log LAG3 concentrations, independent of age, race, study center, systolic blood pressure, antihypertensive medication use, diabetes medication use, fasting glucose, HDL and total cholesterol, lipid lowering medication use, body mass index and smoking status (current, former or never). Multivariable logistic regression estimated the odds of moderate to severe coronary artery stenosis related to each log increase in LAG3 concentrations, independent of demographic and cardiovascular risk factors, as well as systemic inflammation biomarkers (IL-6, ICAM-1, sTNFαRI, sTNFαRII, sCD163 and sCD14). In models that included only men with HIV, HIV-associated variables were included as follows: history of an AIDS defining malignancy or opportunistic infection, current and nadir CD4+ T-cell count, current viral load and ART use duration.

Results: 58% (410/704) of the study participants were men with HIV, who were younger (mean age 52 versus 55 years) and less often reported non-Hispanic White race (50% versus 69%) than men without HIV. In a univariate analysis, median (interquartile range [IQR]) LAG3 in men with HIV (7.1 pg/ml [natural log transformed; 6.4, 8.1]) was lower than in men without HIV (7.5 [6.7–7.3]; P < 0.01). However, in a model adjusted for covariates, HIV serostatus was not significantly associated with log LAG3 concentrations. Greater age was associated with greater log LAG3 concentrations (P < 0.01). Non-Hispanic Black race and current smoking status were associated with lower log LAG3 concentrations (P < 0.05 for both). In a model that included only men with HIV, current antiretroviral (ART) therapy use was associated with lower log LAG3 concentrations (P = 0.04). In adjusted models with and without inflammatory markers, no significant association between log LAG3 concentrations and moderate to severe coronary stenosis was observed.

Conclusion: Despite biological plausibility and previous studies in the general population that showed the association of plasma LAG3 with CVD, we did not detect any association between LAG3 concentrations and either HIV serostatus or significant subclinical atherosclerosis.
ABSTRACT P12

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Risk factors for severe hypertriglyceridaemia in people living with HIV in Guatemala

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Objectives: Hypertriglyceridaemia is more prevalent in the Hispanic population living in the US and in Latin America in general. People living with HIV (PLWH) have increased risk of hypertriglyceridaemia due to antiretroviral therapy (ART) effects. Adverse health outcomes correlate with triglyceride levels and aggravation of this condition into severe hypertriglyceridaemia demands aggressive treatment. Severe hypertriglyceridaemia lacks comprehensive data sets in PLWH describing the condition and its risk factors. We aimed to identify risk factors for severe hypertriglyceridaemia in PLWH receiving care at Hospital Roosevelt in Guatemala.

Methods: Baseline data from a prospective cohort study of PLWH enrolled between July 2019 and March 2020 was analysed. We included PLWH 18 years of age or older receiving ART over the last 6 months. Severe hypertriglyceridaemia was defined as fasting serum triglycerides >500 mg/dl from a lipid panel taken up to 4 months before or 1 month after enrolment. Demographics, alcohol use, smoking, history of comorbidities, HIV parameters (CD4 and viral load history), anthropometrics and ART at enrolment were compared based on severe hypertriglyceridaemia using univariable analysis; variables with a P<0.03 were included in a multivariable regression model.

Results: Of 673 participants included, 54.7% were males with a median age of 40 years (IQR 32–49) and 106 (15.8%) had indigenous background. Severe hypertriglyceridaemia was identified in 35 (5%) cases. The majority of ART regimens were efavirenz-based (396, 58.8%), dolutegravir-based (162, 24.1%), lopinavir/ritonavir-based (73, 10.8%), nevirapine-based (32, 4.7%) accounting for 98% of ART prescribed. In the univariable analysis, male gender, diabetes, high waist to stature ratio (WTS) and efavirenz-based current ART were all associated with severe hypertriglyceridaemia and were included in the multivariable analysis (Table 1). Dolutegravir was associated with not having severe hypertriglyceridaemia (OR 0.29, 95% CI 0.087, 0.95; P=0.043). All variables included in the multivariable analysis were associated with severe hypertriglyceridaemia, with a relative higher risk conferred by high WTS and diabetes (Table 1).

Discussion: To our knowledge, this is the first study assessing demographic and clinical variables with severe hypertriglyceridaemia in PLWH in the region. Severe hypertriglyceridaemia has been associated with male gender and diabetes in other studies, consistent with the findings of this cohort. While both protease

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Table 1. (Abstract P12)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Poor Triglyceridemia (mg/dl)</th>
<th>Median (IQR)</th>
<th>Univariable analysis</th>
<th>Multivariable analysis</th>
</tr>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>p-value</td>
<td>OR*</td>
</tr>
<tr>
<td>Male gender</td>
<td>27 (77.1%)</td>
<td>347 (54.4%)</td>
<td>0.009</td>
<td>3.49</td>
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<td></td>
<td>7 (20.0%)</td>
<td>35 (5.5%)</td>
<td>0.004</td>
<td>3.73</td>
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<tr>
<td>Diabetes</td>
<td>31 (88.6%)</td>
<td>446 (69.9%)</td>
<td>0.020</td>
<td>4.01</td>
</tr>
<tr>
<td>Abnormal WTS Ratio</td>
<td>27 (77.1%)</td>
<td>369 (57.8%)</td>
<td>0.033</td>
<td>2.40</td>
</tr>
<tr>
<td>Efavirenz-based ART</td>
<td>3 (8.6%)</td>
<td>159 (24.9%)</td>
<td>0.040</td>
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</tr>
<tr>
<td>Dolutegravir-based ART</td>
<td>4 (11.4%)</td>
<td>69 (10.8%)</td>
<td>0.785</td>
<td></td>
</tr>
<tr>
<td>Nevirapine-based ART</td>
<td>0 (0%)</td>
<td>32 (5.0%)</td>
<td>0.194</td>
<td></td>
</tr>
<tr>
<td>Excessive alcohol use</td>
<td>4 (11.4%)</td>
<td>45 (7.1%)</td>
<td>0.312</td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>5 (14.3%)</td>
<td>78 (12.2%)</td>
<td>0.790</td>
<td></td>
</tr>
<tr>
<td>&gt;150 min of exercise</td>
<td>1 (2.9%)</td>
<td>41 (6.4%)</td>
<td>0.717</td>
<td></td>
</tr>
</tbody>
</table>

*Severe hypertriglyceridemia **Odds ratio ***95% Confidence interval
inhibitors and efavirenz have been linked with severe hypertriglyceridaemia, in our cohort only efavirenz had a significant association with this outcome. This may be related to specific genetic and metabolic profiles for our population, specifically regarding variations in the cytochrome P450 enzymes. However, further studies are needed to evaluate the effect severe hypertriglyceridaemia has on cardiovascular risk in this cohort.
ABSTRACT P13

*Antiviral Therapy* 2020; 25 Suppl 1:A42

Predictors of atherosclerosis in people with HIV who do not currently qualify for statin therapy

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Objectives/aim: People with HIV are at increased risk for cardiovascular disease (CVD). This study aimed to describe factors associated with atherosclerosis in people with HIV who do not currently qualify for statin therapy.

Methods: Participants with controlled HIV (suppressed viral load, antiretroviral therapy for >6 months) who were at moderate cardiovascular risk (10-year Framingham risk score 10–15%) with no indication for statin therapy were recruited from a single centre in Australia and 4 centres in Switzerland. All participants had carotid intima media thickness (cIMT) measured, a physical exam and fasting bloods. Elevated cIMT was defined as >1.0 mm and is indicative of subclinical carotid atherosclerosis.

Results: Participants (n=83) were predominantly Caucasian (88%), male (99%), with a median age of 54 years (IQR 50–58). 27 (32.5%) were current smokers, 26 (31%) had a positive family history of CVD and 23 (27%) had been previously diagnosed with hypertension. All participants were on antiretroviral therapy with a suppressed viral load. Median CD4 cell count was 610 cells/ul (IQR 417–761). The median cIMT at the common carotid artery was 0.7 mm (IQR 0.6–0.8). cIMT correlated with age, high-density lipoprotein (HDL) cholesterol, albumin and high sensitivity C-reactive protein, with non-significant trends towards correlation with IL-6 and platelet count. Ten (12%) participants had a cIMT >1.0 mm. Factors associated with a cIMT >1.0 mm included total cholesterol (OR 2.3, P=0.035), low-density lipoprotein cholesterol (OR 2.5, P=0.046), HDL cholesterol (OR 13.9, P=0.027) and current abacavir use (OR 6.9, P=0.007).

Conclusion: In people with well controlled HIV, dyslipidaemia and exposure to abacavir is associated with an increase in atherosclerosis and cardiovascular risk.
ABSTRACT P14

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Impact of rosuvastatin on pulse wave velocity (PWV) in men with HIV at moderate cardiovascular risk; a sub-study of a multinational, randomized, double blind, placebo-controlled trial

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Aim: People with HIV are at increased risk for cardiovascular disease (CVD). This substudy aimed to determine the effect of rosuvastatin on PWV in HIV.

Methods: Men with well controlled HIV (VL <20, ART for >6 months) at moderate cardiovascular risk (10-year Framingham risk score 10–15%) and no indication for statins (n=55) were randomized 1:1 to rosuvastatin (n=28) or matched placebo (n=27). Participants had PWV, carotid intima media thickness and fasting bloods at baseline, week 48 and 96. The primary endpoint was change in PWV from baseline to week 96. Factors associated with baseline PWV were evaluated.

Results: Participants were predominantly Caucasian (50 [90.9%]), median age 54 years (IQR 51–58). Seventeen (30.9%) were smokers, median systolic blood pressure 130 mmHg (IQR 120–135). Baseline variables were well matched across the arms. There was no difference in change in PWV from baseline to week 96 between the arms; rosuvastatin =0.4 m/s (IQR -0.1–1.0) versus placebo =0.4 m/s (IQR -0.2–1.2); P=0.92. Rosuvastatin led to significant reductions in total cholesterol (-1.3 mmol/l [-1.7–-0.9] versus placebo =0.0 [-0.5–0.4]; P<0.001). Baseline PWV was associated (P<0.05) with age, smoking, body mass index and intima media thickness but not with cell count or CD4 nadir.

Non-severe events were more frequent with rosuvastatin. Two participants developed new type 2 diabetes, four experienced a CVD event and one an asymptomatic raise in CK to 9,000; all in the rosuvastatin arm.

Conclusions: In men with HIV at moderate CVD risk rosuvastatin had no effect on PWV but was associated with significant side effects.
ABSTRACT P15

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One-year statin persistence and adherence in adults with HIV in the United States

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**Background:** Statin persistence and adherence are low among US adults. Many adults with HIV have high cardiovascular disease risk and may benefit from statin therapy. Most individuals with HIV in the US have high adherence to antiretroviral therapy (ART) but less is known about their statin persistence and adherence. We compared statin persistence and adherence between adults with and without HIV. Statin adherence was compared with ART adherence among adults with HIV.

**Methods:** We analysed claims data from adults in the MarketScan database who initiated statin therapy between 2007 and 2016. People with HIV (n=5,619) were frequency matched 1-to-4 to those without HIV (n=22,476) based on age, sex and calendar year of statin initiation. Statin persistence was defined by having dispensed statin medication during the last 90 days of the 365 days following initiation. High statin adherence was defined as proportion of days covered (PDC) ≥80% during the 365 days following initiation. The minimum PDC was calculated for all ART drugs among people with HIV.

**Results:** The mean age of the study population was 51 years and 85.8% were men. Statin persistence was higher among adults with versus without HIV (72.8% versus 65.2%, multivariable-adjusted prevalence ratio 1.13, 95% CI 1.11, 1.15). Among those who were persistent, a higher proportion of people with versus without HIV had high statin adherence (69.6% versus 59.9%, multivariable-adjusted prevalence ratio 1.16, 95% CI 1.13, 1.19). Among people with HIV and high ART adherence (PDC≥0.90), 34.6% had a PDC for statin therapy <0.80.

**Conclusions:** A high proportion of adults with HIV have low adherence to statin therapy. Patient-centred approaches are needed to increase statin adherence among people with HIV.
Comparing the prevalence of hypertension between HIV-positive and HIV-negative adults: a global systematic review and meta-analysis of cross-sectional studies

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Objectives/aim: As life expectancy among people living with HIV (PLHIV) has increased dramatically since effective antiretroviral therapy was introduced, ageing PLHIV are increasingly affected by non-communicable diseases, including hypertension. Hypertension, though often asymptomatic, is a key risk factor for other conditions like chronic kidney disease and cardiovascular disease. In recent years, several observational studies have found a differing prevalence of hypertension among PLHIV compared with HIV-negative individuals, with some finding an elevated burden and others finding a reduced burden. However, this evidence has not been systematically assessed, undermining efforts to develop hypertension prevention and care strategies. We aimed to perform the first global systematic review and meta-analysis of hypertension prevalence by HIV status.

Methods: We searched MEDLINE, EMBASE, Global Health and Cochrane Central Register of Controlled Trials to 23 August 2019, and reference lists of included articles, to identify published cross-sectional studies presenting hypertension prevalence by HIV status among adults aged over 15. Our risk of bias assessments addressed adequacy of sample sizes, participant selection and HIV and hypertension status measurement.

We pooled risk ratios (RRs) for prevalent hypertension with random effects models. We also explored whether any difference in hypertension prevalence was associated with study-level factors through sub-group analyses and meta-regression. Our sensitivity analyses examined the impact of removing studies at high risk of bias and applying the Hartung-Knapp modification to account for small sample sizes. The study followed PRISMA guidelines and is registered with PROSPERO (CRD42019151359).

Figure 1. [Abstract O17]
Results: Of 20,305 studies identified, 51 were eligible (1,943,047 participants). Overall, global hypertension prevalence was lower among PLHIV than HIV-negative individuals (RR: 0.90, 95% confidence interval [CI]: 0.84, 0.97, I²=97%). The relationship varied by continent (Figure 1; HPN: hypertension), with prevalence higher among PLHIV in North America (1.13, 1.00, 1.27) and lower among PLHIV in Africa (0.75, 0.66, 0.84) and Asia (0.77, 0.63, 0.95). Meta-regression confirmed that the risk ratio was higher for North America than Africa (coefficient: 1.46, 95% CI: 1.16, 1.83). Removing two studies at high risk of bias indicated that the risk ratio for Europe was also higher than the risk ratio for Africa (1.28, 1.04, 1.57). Our results were robust to use of the Hartung-Knapp modification.

Conclusion(s)/discussion: Our findings demonstrate that the relative vulnerability of PLHIV to hypertension differs by region. This suggests that policy for hypertension prevention and care will require tailoring to local needs. Our results also highlight the need for further studies of PLHIV and matched negative controls, in order to better understand the dynamics and mechanisms of observed context-specific trends and shape care accordingly. Efforts to safeguard the long-term quality of life of PLHIV will rely on multidisciplinary work to respond to the varying challenges posed by comorbidities.
ABSTRACT O18
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Carotid atherosclerosis in suppressed HIV patients and in a healthy sample: prediction by cardiovascular risk equations
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Background: Carotid plaque assessment may improve cardiovascular (CV) risk prediction and could reclassify patients to a higher risk, which would benefit of intensive preventive measures. The aim of the study was to assess the relation between carotid plaque and several CV risk equations in an HIV-infected suppressed sample and to compare with a healthy sample.

Methodology: Cross-sectional study of two samples matched by age and sex: (1) HIV-patients randomly selected from an HIV Unit in 2014–16; (2) community-based sample randomly selected from Girona (Spain) in 2016. All individuals were aged 30 to 80 years and had no history of CV disease. HIV patients had undetectable viral load on the last 6 months. The presence of plaque in the common, bulb and internal carotid was assessed by B-mode ultrasound. 10-year CV risk was assessed by 5 equations: three designed for the general population (Framingham, REGICOR [Framingham calibrated and validated for the Spanish population] and Systematic Coronary Risk Evaluation [SCORE]) and calibrated to HIV-infected population (COMVIH [Framingham calibrated to Spanish HIV population] and the reduced D:A:D).

Results: 758 participants were included (379 per group), 78% men, age 50 (10). HIV-sample: CD4 718 (338) cell/ul, duration of antiretroviral therapy 14.7 (6.6) years. There were no differences between samples in the presence of carotid plaque (HIV 33.2%, control 31.3%; adjusted odds ratio [95% CI] 1.15 [0.79, 1.68]).

Figure 1. (Abstract O18)
All three CV risk equations for the general population showed that HIV-infected patients have a slight but significantly higher risk of future severe CV outcomes than controls (Framingham [7.52 versus 6.48], REGICOR [2.67 versus 2.16], SCORE [0.40 versus 0.26]; P≤0.001 for all comparisons). However, there were no significant differences between groups in the percentage of HIV-infected patients or controls in different risk categories for Framingham (P=0.843), REGICOR (P=0.375) or SCORE (P=1.000). The COMVIH equation in the HIV-infected group classified a higher number of patients in the moderate and high CV risk categories. The presence of carotid plaque increased alongside the increase in risk category, with no significant differences according to HIV serostatus. When CV risk equations for general population were applied, among 18% to 27% of participants classified in the low CV risk category had carotid plaque; the percentage of carotid plaque in those classified in the moderate risk category was between 47% to 76%. These percentages were similar when COMVIH or reduced D:A:D were used. See Figure 1. 

Conclusions: An important number of participants classified in the low and moderate CV risk categories by all the equations and in both groups had carotid plaque. Assessment of carotid plaque by ultrasound may be useful for identifying HIV-infected patients who could benefit from more aggressive management of preventive measures.
ABSTRACT O19  
Antiviral Therapy 2020; 25 Suppl 1:A49

Selective drop-out of HIV-positive AGEhIV Cohort participants may bias estimates of long-term adverse health effects of ageing with HIV

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Aims: Attrition is an important challenge for longitudinal studies, especially when attrition is associated with the studied exposure or outcome variables (that is, selective drop-out). This problem is likely to occur in cohort studies aimed at evaluating clinical outcomes in ageing people with HIV (PWH). A higher burden of co- and multimorbidity in ageing PWH could lead to earlier study discontinuation and thereby to biased estimates of incident comorbidities in PWH. This analysis aims to (1) quantify attrition and its reasons and (2) determine factors possibly associated with attrition in the AGEhIV Cohort Study.

Methods: From October 2010 through September 2012 589 HIV-positive and 550 lifestyle comparable HIV-negative participants, aged ≥45 years, were included into the cohort. Participants underwent extensive health evaluation (±1.5 h) during biennial study visits. In this analysis follow-up data through October 2019 were included. Attrition was defined as not having attended a study-visit without advance notice within 6 months of its scheduled date, or withdrawal of consent. Follow-up data were analysed in intervals of 2 years. Observation time was censored at time of attrition, death or the last scheduled study-visit, whichever occurred first. Complementary log-log regression was used to (1) estimate hazard ratios (HRs) for attrition during each interval, comparing HIV-positive and HIV-negative participants, and (2) assess factors possibly associated with attrition, including HIV-status, socio-demographics, having ≥1 comorbidities and depressive symptoms (categorized as ≤8, 9–15 or ≥16 points on the Center for Epidemiologic Studies Depression [CES-D] questionnaire).

Results: During the 9-year observation period, attrition occurred in 178 (29.8%) HIV-positive and 90 (16.4%) HIV-negative participants, of whom 87 (48.9%) and 41 (45.6%), respectively, withdrew consent. Reasons for withdrawal were more commonly health-related (for example, study-visit was too strenuous or poor health condition prevented study participation) in HIV-positive (n=33, 37.9%) versus HIV-negative (n=8, 19.5%; P=0.01) participants. Thirty HIV-positive and 8 HIV-negative participants died. HIV-status was associated with attrition (univariable HRHIV1.9, 95% CI=1.5, 2.5; P<0.001; Figure 1), this effect was attenuated after adjustment for gender, age at enrolment, non-Dutch origin, level of education, and having ≥1 comorbidity or depressive symptoms at a study-visit (multivariable HHRHIV1.5, 95% CI=1.1, 2.0; P=0.003). Non-Dutch origin, lower educational level, having ≥1 comorbidities or severe depressive symptoms (≥16 points on the CES-D) each were also independently associated with attrition, without significant interactions with HIV-status for any of these factors.

Conclusions: HIV-positive status was independently associated with greater risk of attrition, with poor health often reported as the reason. This was only partly explained by having greater numbers of comorbidities or depressive symptoms, each of which were equally associated with attrition in HIV-positive and negative participants. Indicators of poor health not captured by our study may contribute to the increased attrition rate among PWH. These findings suggest that selective drop-out may lead to underestimates of long-term adverse health effects of ageing in PWH.

Figure 1. [Abstract O19]
ABSTRACT O20

Antiviral Therapy 2020; 25 Suppl 1:A50

Choline-containing lipid species are associated with hepatic steatosis in people with HIV

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Objectives/aims: People with HIV (PWH) have a higher prevalence of non-alcoholic fatty liver disease (NAFLD) than HIV-negative persons. The mechanisms that give rise to this disparity are not known but may be due to virus-specific effects, antiretroviral therapy or changes in the intestinal microbiome. Collectively, these factors contribute to alterations of the plasma lipidome that promote lipid accumulation, inflammation and fibrosis in the liver. A more complete understanding of the plasma lipidome will provide targets for specific therapies and diagnostics for NAFLD in PWH. In this study, we define the plasma lipidome associated with NAFLD as compared with other ectopic lipid depots in PWH.

Methods: Adult PWH on antiretroviral therapy with sustained viral suppression were recruited at a large academic medical centre. Participants underwent non-contrasted abdominal CT scans and the size and/or density of skeletal muscle, liver and adipose tissue were measured. Fasted plasma was collected from all participants for untargeted liquid chromatography-mass spectrometry lipidomics profiling. The abundance of lipid species relative to the size and/or density of tissues as measured on CT scan was assessed using linear regression modelling.

Results: The mean age of the cohort was 46 years old and BMI was 33.3. 78% of participants were male and 53% were White. The average CD4+ T-cell count was 498 cells/mm³ and the average duration of antiretroviral therapy was 8.6 years. A higher abundance of choline-containing species, including phosphatidylcholine and lysophosphatidylcholine was associated with lower levels of hepatic steatosis (as measured by higher CT attenuation; Figure 1). Conversely, higher abundance of triglycerides and oxidized triglyceride species was associated with greater hepatic steatosis. Visceral fat attenuation showed similar relationships with triglycerides and choline-containing lipid species, whereas visceral fat volume and pericardial fat volume had an inverse relationship with these species.

Discussion: A decreased abundance of plasma choline-containing species and increased levels of triglyceride species is associated with hepatic steatosis in PWH. Plasma choline deficiency has previously been shown to be associated with hepatic steatosis and cardiovascular disease in HIV and could be secondary to intestinal dysbiosis. Ongoing studies will determine whether changes in the intestinal microbiome are associated with a ‘pro-NAFLD’ plasma lipidome in PWH.
ABSTRACT P16

Antiviral Therapy 2020; 25 Suppl 1:AS1

No difference in monocyte cholesterol metabolism gene expression in people living with HIV and matched uninfected controls: the HIV UBPEAT CAD Substudy


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Objectives/aims: People living with HIV (PLWH) have an increased risk of coronary artery disease (CAD) with previous studies reporting monocyte gene expression consistent with high intracellular cholesterol in treatment-naive PLWH which improves but does not normalize with ART. We aimed to examine differences in cholesterol metabolism gene expression in a cohort of treated PLWH with CAD risk factor (CADRF) matched uninfected controls.

Methods: The UBPEAT CAD substudy enrolled 100 participants of the HIV UPBEAT study, a prospective longitudinal cohort of PLWH and uninfected controls with over 10 years follow-up. Participants were over the age of 40 years, had no known history of CAD and were closely matched for HIV serostatus and traditional CADRF.

Quantitative polymerase chain reaction used to assess the expression of 17 cholesterol metabolism genes in monocytes isolated from fasting fresh blood samples. Gene expression was reported relative to 3 housekeeping genes. Between group differences was assessed using Mann–Whitney U test and analysis of covariance to adjust for confounders (SPSS vers 24).

Results: 99 participants were included in the analysis. Median age was 50.81 (46.29, 56.24) years, 71.71% male, 76.76% Caucasian and 48.48% were current smokers. PLWH had lower HDL cholesterol (HIV+ 1.27 [1.0, 1.3]; HIV- 1.4 [1.1, 1.6]; P=0.017) and more likely to be on statin therapy (HIV+ 49%, HIV- 12%; P<0.01). Other demographics and CADRF were similar between groups.

There was no significant difference between groups in expression of cholesterol sensing (SCAP: HIV+ 0.07 [0.05, 0.11], HIV- 0.07 [0.05, 0.08]; P=0.425, SREBF1: HIV+ 0.013 [0.01, 0.02], HIV- 0.010 [0.01, 0.02]; P=0.28, MBTPS1: HIV+ 0.03 [0.02, 0.04], HIV- 0.03 [0.02, 0.04]).

<table>
<thead>
<tr>
<th>Gene</th>
<th>HIV Negative Estimated Mean (95% CI)</th>
<th>HIV Positive Estimated Mean (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCAP</td>
<td>0.0088 (0.0796, 0.0404)</td>
<td>0.0779 (0.0041, 0.0392)</td>
<td>0.237</td>
</tr>
<tr>
<td>SREBF1</td>
<td>0.0131 (0.0119, 0.0150)</td>
<td>0.0117 (0.0099, 0.0138)</td>
<td>0.34</td>
</tr>
<tr>
<td>SREBF2</td>
<td>0.0198 (0.0198, 0.0229)</td>
<td>0.0219 (0.0198, 0.0239)</td>
<td>0.391</td>
</tr>
<tr>
<td>NBTPS1</td>
<td>0.0080 (0.0210, 0.0016)</td>
<td>0.0080 (0.0074, 0.0086)</td>
<td>0.42</td>
</tr>
<tr>
<td>NBTPS2</td>
<td>0.0048 (0.0033, 0.0064)</td>
<td>0.0058 (0.0041, 0.0077)</td>
<td>0.513</td>
</tr>
<tr>
<td>PPARG</td>
<td>0.0055 (0.0171, 0.0117)</td>
<td>0.0092 (0.0170, 0.0115)</td>
<td>0.293</td>
</tr>
<tr>
<td>NSD1</td>
<td>0.0053 (0.0042, 0.0064)</td>
<td>0.0053 (0.0042, 0.0064)</td>
<td>0.395</td>
</tr>
<tr>
<td>ULP</td>
<td>0.0038 (0.0018, 0.0058)</td>
<td>0.0035 (0.0021, 0.0054)</td>
<td>0.353</td>
</tr>
</tbody>
</table>

Model adjusted for HDL and Statin use. CI: Confidence Interval (lower bound, upper bound)
0.025 [0.02, 0.05]; \( P = 0.49 \), PPARA: HIV- 0.006 [0.004, 0.014], HIV+ 0.005 [0.003, 0.015]; \( P = 0.34 \), NR1H3: HIV- 0.004 [0.002, 0.006], HIV+ 0.004 [0.002, 0.006]; \( P = 0.93 \), LPL: HIV- 0.0017 [0.0006, 0.0037], HIV+ 0.0015 [0.0006, 0.0060]; \( P = 0.67 \), cholesterol uptake (LDLR: HIV- 0.0435 [0.0300, 0.0532], HIV+ 0.0400 [0.0254, 0.0548]; \( P = 0.79 \), CD36: HIV- 0.0175 [0.0058, 0.0460], HIV+ 0.0320 [0.0054, 0.0933]; \( P = 0.67 \), synthesis (HMGCR: HIV- 0.0141 [0.0499, 0.0378], HIV+ 0.0175 [0.0046, 0.0782]; \( P = 0.72 \), PMVK: HIV- 0.0146 [0.0099, 0.0318], HIV+ 0.0150 [0.0107, 0.0239]; \( P = 0.72 \), ACAT2: HIV- 0.0097 [0.0066, 0.0136], HIV+ 0.0105 [0.0065, 0.0150]; \( P = 0.737 \) or efflux genes (ABCA1: HIV- 0.0006 [0.0002, 0.0014], HIV+ 0.0007 [0.0003, 0.0018]; \( P = 0.323 \), ABCG1: HIV- 0.0047 [0.0033, 0.0094], HIV+ 0.0069 [0.0029, 0.0094]; \( P = 0.978 \), SCARB1: HIV- 0.0214 [0.0146, 0.0344], HIV+ 0.0200 [0.0147, 0.0305]; \( P = 0.722 \).

After adjustment for HDL and statin use, there remained no significant association between HIV serostatus and cholesterol metabolism gene expression (Table 1).

**Conclusions/discussion:** In a cohort of treatment-experienced PLWH and CADRF matched controls, there was no significant difference in monocyte cholesterol gene expression suggesting a persistent dysfunctional intracellular cholesterol metabolism may not contribute to the increased risk of CAD observed in stable, ART-treated PLWH.
Prevalence of silent atherosclerosis and other comorbidities in an outpatient cohort of adults living with HIV: associations with HIV parameters and biomarkers

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Background: People living with HIV (PLWH) are at risk of non-infectious comorbidities. It is important to individualize those at higher risk.

Methods: In a single-centre cohort of PLWH, we performed a cross-sectional analysis of comorbidities, diagnosed according to standard procedures. The primary end point was the prevalence of subclinical carotid/coronary atherosclerosis. Secondary end points were its association with selected inflammatory/immune activation biomarkers and with other comorbidities. Associations were examined using Chi-2 or Fisher’s exact test for categorical variables and Student or Wilcoxon tests for quantitative variables, and a stepwise multivariate logistical model was performed for further exploration.

Results: Among 790 participants (median age: 49.8 years [IQR: 44.5–55.6], 77.1% males, median CD4: 536/mm³ [IQR: 390–754], 83.6% with undetectable viral load), asymptomatic atherosclerosis was found in 26% and was associated in multivariate analysis with older age, longer known duration of infection, longer exposure to antiretroviral drugs and ever exposure to stavudine, higher sCD14 and lower adiponectin levels. Hypertension was found in 33.5% of participants, diabetes in 19.4%, renal impairment in 14.6%, elevated LDL-cholesterol in 13.3%, elevated triglyceride/HDL-cholesterol ratio in 6.6% and osteoporosis in 7.9%. The presence of two or more comorbidities was found in 42.1% of participants and was associated in multivariate analysis with older age, longer exposure to antiretrovirals, ever exposure to stavudine and higher sCD14 levels. Comorbidities were diversely associated with biomarkers: osteoporosis with higher IL-6, renal impairment with higher sCD14, hypertension with higher D-dimer, diabetes and elevated triglyceride/HDL-cholesterol ratio both with lower adiponectin and lower 25-hydroxy-vitamin D.

Conclusions: Asymptomatic atherosclerosis and multimorbidity were frequent in a cohort of middle-aged, well-controlled, PLWH and were associated with traditional and HIV-specific factors. Associations between morbidities and inflammatory/immune activation biomarkers were diverse.
ABSTRACT P18
Antiviral Therapy 2020; 25 Suppl 1:A54

Higher interactions of leukocytes and platelets with the endothelium as a potential hint of cardiovascular risk in ABC-treated HIV patients

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Aims: Abacavir (ABC) has been linked with a higher risk of myocardial infarction. We have demonstrated in vitro, in cells from healthy donors, that clinical concentrations of ABC, but not of tenofovir disoproxil fumarate (TDF), have pro-inflammatory effects, inducing leukocyte-endothelium interactions and pro-thrombotic actions, causing the interplay of platelets with endothelial cells and leukocytes (specifically with neutrophils). Furthermore, ABC promoted thrombus formation in a well-established murine in vivo model. Thus, given that platelets and their interactions with other vascular cells play an important role in thrombus formation that can lead finally to myocardial infarction, the aim of the present study was to test the pro-inflammatory and pro-thrombotic status of HIV patients undergoing ABC versus tenofovir (TFV), including TDF or tenofovir alafenamide (TAF), treatment by analysing polymorphonuclear (PMN)- and/or platelet-endothelium interactions in cells isolated from blood of these two groups of HIV patients.

Methods: This is a non-randomized prospective observational study in which we used leukocytes and platelets from blood withdrawn from HIV-patients at Hospital Clinico Universitario de Valencia who had been receiving treatment, for at least 6 months, with an ART regime that included either ABC or TFV. Interactions of isolated PMNs, rolling and adhesion, and isolated platelets, adhesion, with a non-infected endothelium monolayer were evaluated by means of a parallel-plate flow chamber system. Platelets were labelled with an anti-CD41 (specific platelet marker) antibody linked to Alexa-Fluor®488 in order to visualize them by Epifluorescence microscopy.

Results: 50 patients were included in the study, 25 of whom were receiving ABC and 25 of whom were receiving TFV. There were no significant differences in demographic and cardiovascular risk parameters between the two groups. PMN rolling (Figure 1A) and adhesion (Figure 1B) along the endothelium were significantly higher in the ABC group than in the TFV group. Moreover, the number of platelets adhering to endothelial cells was higher in the ABC versus TFV group (Figure 1C).

Conclusions: Treatment with ABC enhances PMN-endothelium interactions, thus promoting the initial phases of the inflammatory process. Furthermore, it induces platelet adhesion to endothelial cells, which is an important step in thrombus formation. Our results give support to the increased risk of myocardial infarction observed in ABC-treated HIV patients.

Figure 1. (Abstract P18)
ABSTRACT P19

Antiviral Therapy 2020; 25 Suppl 1:A55

Protective effects of lamivudine, emtricitabine, didanosine, raltegravir and atazanavir on platelet aggregation

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1University of Valencia, Valencia, Spain

Objectives/aims: Cardiovascular toxicity associated with combined antiretroviral therapy has been attributed mainly to nucleoside reverse transcriptase inhibitors (NRTI; specifically abacavir), though particular drugs from other families (non-nucleoside reverse transcriptase inhibitors [NNRTI], chemokine co-receptor 5 antagonist [aCCR5], protease inhibitors [PI] and integrase strand transfer inhibitors [INSTI]) may also produce this side effect as they are administered in combination. This study evaluates the actions of clinically relevant concentrations of antiretrovirals belonging to the different families on platelet aggregation – key step in the transition from inflammation to thrombus generation.

Methods: We employed platelet-rich plasma (PRP) from healthy donors from Hospital Clínico Universitario de Valencia, who had not been receiving drugs for at least 10 days. Platelet aggregation in response to positive stimulus such as ADP (1 mM) or collagen (2 µg/ml) was assessed by an impedance aggregometer (Chrono-Log Model 590-2D) in the presence of selected antiretroviral drugs (4 min treatment): the NRTIs abacavir (5 µg/ml), tenofovir (0.3 µg/ml), didanosine (1.8 µg/ml), emtricitabine (2.5 µg/ml) and lamivudine (2.3 µg/ml); the INSTIs raltegravir (5 µg/ml) and elvitegravir (10 µg/ml); the aCCR5 maraviroc (5 µg/ml); the PI lopinavir (25 µg/ml) and atazanavir (25 µg/ml) and the NNRTIs efavirenz (25 µg/ml) and rilpivirine (1 µg/ml). AUC (area under curve) is the parameter shown as it includes both amplitude and slope of the curve.

Results: In contrast to what was expected, none of the drugs potentiated platelet aggregation induced by ADP or collagen. However, some of them had a protective action. Particularly lamivudine and raltegravir reduced platelet aggregation induced by ADP (Figure 1A) whereas emtricitabine, didanosine and atazanavir inhibited that caused by collagen (Figure 1B). On the other hand, the rest of antiretroviral drugs analysed did not induce significant changes in platelet aggregation stimulated by ADP or collagen.

Conclusions/discussion: Our results suggest that lamivudine, emtricitabine, didanosine, atazanavir and raltegravir could be safer alternatives than other antiretroviral drugs used in acquired immune deficiency syndrome treatment such as abacavir which have been correlated with the appearance of cardiovascular side effects.
ABSTRACT P20

Antiviral Therapy 2020; 25 Suppl 1:A56

Changes in inflammatory markers after switching to elvitegravir/cobicistat/emtricitabine/tenofovir alafenamide in virologically suppressed older people living with HIV

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Aim: Antiretroviral therapy reduces both immune activation and systemic inflammation, yet, little data exist on these indices in older people living with HIV. We examined the effect of age on plasma biomarkers of monocyte activation (sCD14, sCD163), and systemic (IL-6, hscRP, sTNFR-I, TNFR-2 and D-dimer) and vascular (Lp-PLA2) inflammation in virologically suppressed adults living with HIV who switched from a stable regimen to elvitegravir/cobicistat/emtricitabine/tenofovir alafenamide in two randomized, controlled trials (GS-US-292-1823, GS-US-292-1826).

Methods: Samples were included from participants who were on abacavir/lamivudine or emtricitabine/tenofovir disoproxil fumarate plus a 3rd agent for ≥6 months and switched to elvitegravir/cobicistat/emtricitabine/tenofovir alafenamide, received at least one dose of study medication, and had both baseline and at least one post-baseline value at weeks 12 or 24 for any biomarker. Samples from participants who had an HIV-1 RNA ≥50 copies/ml at any time point were excluded. Biomarker levels were measured at baseline and at weeks 12 and 24. We compared baseline levels and change from baseline between two age groups (<60 years, ≥60 years) using the Wilcoxon rank sum test.

Results: 235 participants were included (<60 years, 119; ≥60 years, 116): 14% women, 10% Black, 30% current smokers, 29% statin users and 45% with 10-year cardiovascular risk ≥10%. Median baseline CD4 count (651 cells/μl) was similar in both groups. Baseline levels IL-6, TNFRs 1 and 2, D-dimer and sCD163 were significantly higher in the ≥60 years old group compared with levels in the <60 years. In the total population, median levels of IL-6, D-dimer, TNFRs 1 and 2, and Lp-PLA2 increased from baseline through week 24. There were no differences in the median change in biomarkers between age groups at weeks 12 and 24 (P>0.05) except for sCD163 (P=0.007 and 0.03, respectively). By stepwise regression model, age was not a significant predictor for percent change in any of the biomarkers through week 24. See Table 1.

Conclusions: Although there were differences in biomarkers between age groups at baseline, age was not associated with longitudinal biomarker changes in virologically suppressed adults after switching to elvitegravir/cobicistat/emtricitabine/tenofovir alafenamide.

Table 1. Baseline and change in biomarkers by age group (Abstract P20)

<table>
<thead>
<tr>
<th>Biomarkers</th>
<th>Median (Q1, Q3)</th>
<th>&lt;60 years</th>
<th>≥60 years</th>
<th>P-value*</th>
<th>Overall</th>
<th>&lt;60 years</th>
<th>≥60 years</th>
<th>P-value*</th>
<th>Overall</th>
</tr>
</thead>
<tbody>
<tr>
<td>hsCRP (μg/ml)</td>
<td>1.4 (0.8, 4.0)</td>
<td>1.5 (0.7, 4.6)</td>
<td>2.1 (1.0, 4.6)</td>
<td>0.03</td>
<td>1.7 (0.8, 4.1)</td>
<td>1.5 (0.7, 4.6)</td>
<td>0.03</td>
<td>1.8 (0.8, 4.1)</td>
<td>1.6 (0.7, 4.7)</td>
</tr>
<tr>
<td>IL-6 (pg/ml)</td>
<td>7.7 (12.2, 27.7)</td>
<td>7.2 (10.8, 27.7)</td>
<td>7.7 (12.2, 27.7)</td>
<td>0.0001</td>
<td>2.1 (1.4, 3.5)</td>
<td>2.1 (1.4, 3.5)</td>
<td>0.0001</td>
<td>2.1 (1.4, 3.5)</td>
<td>2.1 (1.4, 3.5)</td>
</tr>
<tr>
<td>D-dimer (ng/ml)</td>
<td>275 (203, 443)</td>
<td>286 (209, 464)</td>
<td>275 (203, 443)</td>
<td>&lt;0.0001</td>
<td>332 (229, 483)</td>
<td>332 (229, 483)</td>
<td>&lt;0.0001</td>
<td>332 (229, 483)</td>
<td>332 (229, 483)</td>
</tr>
<tr>
<td>sCD14 (ng/ml)</td>
<td>1656 (1440, 1803)</td>
<td>1652 (1337, 1803)</td>
<td>1656 (1440, 1803)</td>
<td>0.88</td>
<td>1636 (1402, 1803)</td>
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</tr>
<tr>
<td>sCD40L (pg/ml)</td>
<td>657 (596, 800)</td>
<td>733 (580, 927)</td>
<td>657 (596, 800)</td>
<td>0.008</td>
<td>678 (596, 927)</td>
<td>678 (596, 927)</td>
<td>0.008</td>
<td>678 (596, 927)</td>
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</tr>
<tr>
<td>Lp-PLA2 (ng/ml)</td>
<td>107 (59, 225)</td>
<td>96 (46, 229)</td>
<td>107 (59, 225)</td>
<td>0.18</td>
<td>108 (59, 229)</td>
<td>108 (59, 229)</td>
<td>0.18</td>
<td>108 (59, 229)</td>
<td>108 (59, 229)</td>
</tr>
<tr>
<td>sTNFR-I (pg/ml)</td>
<td>227 (102, 229)</td>
<td>245 (124, 225)</td>
<td>227 (102, 229)</td>
<td>&lt;0.0001</td>
<td>252 (124, 225)</td>
<td>252 (124, 225)</td>
<td>&lt;0.0001</td>
<td>252 (124, 225)</td>
<td>252 (124, 225)</td>
</tr>
</tbody>
</table>

*P-value for difference between age groups by 2-sided Wilcoxon rank sum test

We used a multivariate, stepwise regression model to identify factors associated with percent change in biomarkers. Demographics, disease characteristics, prior HIV treatment duration, CD4 cell count, eGFR, lipid profiles, CVD risk score, diabetes, hypertension, hyperlipidaemia, current smoking status, baseline statin use and baseline third agent were included in the stepwise regression models.

Table 1.

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ABSTRACT P21

Antiviral Therapy 2020; 25 Suppl 1:A57

Attitudes toward COVID-19 among people living with HIV attending a multiethnic outpatient centre in Houston, Texas

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Objectives: The evolving outbreak of coronavirus 2019 (COVID-19) may impact people with HIV (PWH) in unique ways, beyond the increased risk of medical complications. Social distancing measures resulting in social isolation and interruptions in medical care may disproportionately affect PWH. The purpose of this study is to capture and understand how COVID-19 is affecting PWH in a low-income, multiethnic urban health centre in Houston, Texas.

Methods: Cross-sectional survey of adult PWH who attend Thomas Street Health Center for outpatient care and volunteered to complete a short telephone questionnaire. Descriptive statistics were calculated for all patient characteristics and survey responses.

Results: Of the 188 participants, 26% were women, 3.9% were transgender women, 33% were non-Hispanic Black and 57.4% Hispanic. The median (interquartile range) age was 49 (40–56) years; 73% had 1 or more comorbidities. Overall, most (82%) people had not experienced difficulty accessing HIV medicines; 25% of patients reported difficulty accessing medical care and failing to attend clinic due to fear of exposure to COVID-19. Most (82.8%) reported being affected by COVID-19 in their daily life; 14% reported being extremely affected. 64% feared getting COVID-19, though a minority feared they were at increased risk of becoming sick, a finding especially true among Black (22.4%) and Hispanic individuals (33.9%) compared with White participants (15.4%). Nearly half (48.4%) reported increased anxiety. More than a third (37.8%) reported feeling depressed, more frequently reported among Black (46.6%) compared with Hispanic individuals (34.8%) and White participants (23%); 31% felt more alone; 90% reported not seeking behavioural health resources. Only 10% reported increased use in alcohol or illegal substances. One in four did not have access to an electronic device for a telemedicine visit. Of the 14% who had lost their job after the pandemic started, 61% were Hispanic.

Conclusions: PWH experienced a wide range of effects from the COVID-19 pandemic. It is important to recognize the socioeconomical and psychological implications that will be seen in disadvantaged communities of PWH as a consequence of COVID-19.