With improved treatment, women living with HIV (WLHIV) are increasingly becoming pregnant. Studies have shown suboptimal viral suppression following pregnancy. In addition, protease inhibitors (PI) have been associated with preterm birth (PTB).

**Methods.** We studied WLHIV with at least 2 live births while on the PHACS SMARTT study. We compared CD4 counts and viral loads (VL) between the first and second pregnancies using Wilcoxon rank-sum tests. We then examined trends in these measures over all reported pregnancies using mixed-effect linear regression models adjusting for maternal age and birth year, with a random effect to account for repeated measures in the same woman over time. Finally, we evaluated the association of PI or non-PI use during pregnancy with PTB, using GEE logistic regression models to adjust for pregnancy number, maternal age, and birth year.

**Results.** Between 2007 and 2018, 699 women had >1 pregnancy while on study, with a total of 1642 children. Their mean CD4 counts remained stable over repeat pregnancies. Their mean log VL decreased between the first and second pregnancies, both early and late in pregnancy (-0.42 cp/mL and -0.16 cp/mL respectively, P < 0.001 for each), but increased by 0.61 cp/mL (P < 0.001) between the end of the first pregnancy and early in the next pregnancy. Most women had VL suppression during pregnancy with VL rebound by the next pregnancy (figure). A majority of women (55%) received a PI in both their first and second pregnancies, with an increase in PTB rate of 4.3%, whereas those who changed from a PI to a non-PI had a decrease of 4.7% (table). Changing to a PI resulted in a stable rate, whereas remaining on a non-PI resulted in a drop of 2%. In adjusted models including all pregnancies, first trimester PI use was associated with an increased rate of PTB (adjusted OR 1.35; 95% CI 1.02, 1.97).

**Conclusion.** Most WLHIV achieved VL suppression during pregnancy, but many had a VL rebound after pregnancy. First trimester PI use was associated with higher risk of PTB.

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**Table:** Paired Group and PTB

| Paired Group | Percent of PTB | Difference |
|--------------|----------------|------------|
| First/second | first          | second     | Difference |
| Pregnancy    | pregnancy      | pregnancy |            |
| Non-PI/Non-PI (n = 103) | 11.7% | 9.7% | -2.0% |
| Non-PI/PI (n = 91) | 15.4% | 15.4% | 0% |
| On PI/Non-PI (n = 86) | 16.3% | 11.6% | -4.7% |
| On PI/Stayed on PI (n = 351) | 14.8% | 19.1% | +4.3% |

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**Figure:** Percent of women with unsuppressed VL (>400 cp/mL)

**Disclosures.** E. Chadwick, Abbott Labs: Shareholder, stock dividends. AbbVie: Shareholder, stock dividends. R. B. Van Dyke, Gildai Sciences: Grant Investigator, Research grant.

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**396. Body Fat Redistribution/Accumulation, Pancreatic Disorders, Musculoskeletal Disorders, IRIS, Severe Systemic Rash and Hypersensitivity Reactions Following Initiation of Commonly Prescribed Antiretrovirals**

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**Session:** 115. HIV-Related Comorbidities and Complications

**Background.** Dolutegravir (DTG), elvitegravir (EVG), raltegravir (RAL), and dolutegravir (DRV) are commonly used for the treatment of HIV. We assessed the frequency of 6 select disorders after prescription of DTG, EVG, RAL, or DRV-based regimens.

**Methods.** HIV-positive patients in the OPERA® Observational Database initiating DTG-, EVG-, RAL-, or DRV-containing regimens were included. Disorders of interest were body fat redistribution/accumulation, pancreatic disorders, and musculoskeletal disorders, as defined in Figures 2–3, as well as immune reconstitution inflammatory syndrome (IRIS), severe systemic rash and hypersensitivity reaction (HSR). Baseline patient characteristics and disorder history were described. The proportion of patients with disorders of interest during follow-up were compared between core agents for each disorder. All events occurring during follow-up were considered prevalent, while incident disorders excluded patients with any history of disorder. To account for multiple comparisons, the Sidak Correction was applied (adjusted α level: 0.017).

**Results.** Out of 22,674 patients, 7,860 (35%) initiated DTG, 9,738 (43%) EVG, 1,600 (7%) RAL, and 3,477 (15%) DRV. Baseline demographic and clinical characteristics varied by core agent initiated (Figure 1). Compared with DTG, history of body fat redistribution/accumulation was less frequent in patients initiating EVG, and more frequent in patients initiating RAL (Figure 2). EVG users also had a lower prevalence during follow-up than DTG users (Figure 3). However, there was no difference in new onset of body fat redistribution/accumulation between groups (Figure 3). No difference in prevalent or incident pancreatic or musculoskeletal disorders was detected between core agents (Figure 3). IRIS, severe systemic rash, and HSR occurred in no more than 2 patients per core agent group, with no difference detected between groups.

**Conclusion.** Incident body fat redistribution/accumulation, pancreatic disorders, musculoskeletal disorders, IRIS, severe systemic rash, and HSR were rare in this large cohort of patients initiating DTG, EVG, RAL, or DRV. Despite some channeling of patients with a disorder history towards DTG and RAL use, the likelihood of new events did not differ by core agent.

**Figure 1. Baseline Demographic and Clinical Characteristics**

**Figure 2. Baseline history of body fat redistribution/accumulation, pancreatic disorders and musculoskeletal disorders**

**Disclosures.** E. Chadwick, Abbott Labs: Shareholder, stock dividends. AbbVie: Shareholder, stock dividends. R. B. Van Dyke, Gildai Sciences: Grant Investigator, Research grant.
937. Virally Suppressed PLH Switching From Abacavir to Tenofovir Alafenamide Did Not Have Changes in Immune Activation or Inflammation
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Session: 115. HIV-Related Comorbidities and Complications
Friday, October 5, 2018: 8:45 AM

Background. Abacavir (ABC) use has been associated with increased risk of myocardial infarction in persons living with HIV (PLH). Its mechanism is unknown, but may involve immune activation inflammation, and/or altered platelet reactivity. In the current analysis, we compared changes in biomarkers of immune activation and inflammation associated with increased cardiovascular (CV) mortality in virally suppressed PLH who switched off ABC to tenofovir alafenamide (TAF) to those who remained on ABC.

Methods. In a randomized, double-blinded, active-controlled trial (GS US 311-1717), virally suppressed PLH on a stable regimen containing ABC plus lamivudine (3TC) were randomly assigned (1:1) to maintain therapy or to switch to TAF plus emtricitabine (FTC) while continuing their third agent. At baseline (BL) and weeks 4, 12, 24, and 48 plasma markers (IL-6, hsCRP, D-Dimer, sCD14, sCD163, TNF-R1, and TNF-R2) were measured by ELISA; Lp-PLA2 levels were measured by the Plac assay.

Results. Of 556 PLH randomized, 548 had samples available for biomarker assessment (TAF: 274; ABC: 274), both arms were of similar CD4 (median 671 cells/μL), age (median 52 years), race (73% white), but there were fewer women in the TAF arm; levels of Lp-PLA2 >7.5 is increased CV risk). BL biomarker concentrations were similar between arms: 0.015) at baseline (BL). Mean BL ASCVD scores were 7.9 in both arms (>7.5 is increased CV risk). BL biomarker concentrations were similar between arms: most had high concentrations of Lp-PLA2 (>250 mg/mL, 96%) and one-third had elevated hsCRP levels >3 mg/L (34%). After switching from ABC to TAF, sCD14 had increased MI risk may involve an alternative etiology.

Conclusion. Prior to switching from ABC to TAF, virally suppressed PLH with mean ASCVD scores of 7.9 had elevated levels of CV risk markers (Lp-PLA2 and hsCRP). Switching off ABC to TAF was not associated with any meaningful change in markers of immune activation or inflammation, suggesting that the ABC-associated increased MI risk may involve an alternative etiology.