Table 2: Demographics Among HCV RNA + (*n* = 488)

Demographics	n (%)
People Born 1945–1965	178 (36.5%)
People Not Born 1945–1965	310 (63.5%)
Women Age 18–45	77 (15.8%)
Known IDU	154 (31.6%)
People Not Born 1945–1965 With No Known IDU	179 (36.7%)

Disclosures. **C. A. Chastain**, Gilead Sciences, Inc.: Grant Investigator and Research Contractor, Grant recipient and Research support. **J. Johnson**, Gilead Sciences, Inc.: Grant Investigator, Grant recipient. **K. Miller**, Gilead Sciences, Inc.: Grant Investigator, Grant recipient. **J. H. Han**, Gilead Sciences, Inc.: Grant Investigator, Grant recipient. **W. H. Self**, Gilead Sciences, Inc.: Grant Investigator, Grant recipient.

933. Serum Albumin Is Associated With Higher Inflammation and Carotid Atherosclerosis in Treated HIV Infection

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Background. Lower serum albumin has recently been associated with cardiovascular disease and non-AIDS malignancies in HIV. This analysis explores the associations between serum albumin and markers of inflammation and atherosclerosis.

Methods. We conducted a nested study within in the SATURN-HIV trial in which 147 HIV+ adults on stable antiretroviral therapy (ART), were virally suppressed, and had an LDL-cholesterol level <130 mg/dL were randomized to 10 mg daily rosuvastatin or placebo. Measures of serum albumin, carotid intima media thickness (IMT, surrogate marker of atherosclerosis), inflammation, T-cell and monocyte immune activation were assessed at baseline, 24, 48, and 96 weeks later. Spearman correlations and linear-mixed effects models with random intercept and slope were conducted to assess associations with albumin.

Results. Mean age was 45 years, 80% were male, 69% were African American, and 46% were receiving protease inhibitors. Mean serum albumin was not significantly different between the groups at any time points (4.05–4.08 g/dL in statin arm vs. 4.01–4.11 g/dL in placebo arm, $P = 0.08$–0.35). Low serum albumin significantly correlated with elevated levels of interleukin-6 (IL6), d-dimer, fibrinogen, and high sensitivity C-reactive protein (hsCRP) at all time points ($P \leq 0.04$). Low serum albumin also correlated with higher inflammatory monocytes (CD14+CD16+) at week 24 and week 96 ($P \leq 0.03$) but not with markers of T-cell activation at any time point ($P \geq 0.10$). Lower baseline albumin significantly predicted larger changes in IMT ($P = 0.03$), IL6, d-dimer, tumor necrosis factor-α receptor 1, fibrinogen, and hsCRP ($P \leq 0.02$) over 96 weeks. After adjusting for age, gender, smoking, body mass index, vascular cell adhesion molecule and creatinine clearance, every 1 g/dL decrease in albumin remained associated with a 0.5 mm increase in IMT over 96 weeks ($P = 0.05$).

Conclusion. Lower serum albumin in controlled HIV is associated with higher markers of chronic inflammation, hypercoagulation, and monocyte activation, which could explain the prior observation that albumin predicts non-AIDS events in HIV. Our findings suggest that serum albumin may predict progression of carotid atherosclerosis independent of traditional risk factors.

Disclosures. **G. A McComsey**, Gilead: Consultant, Consulting fee and Research support. Merck: Consultant, Consulting fee and Research support. Viiv: Consultant, Consulting fee and Research support. Roche: Research Contractor, Research support. Astellas: Research Contractor, Research support.

934. Incidence of Skin and Soft-tissue Infection in People Living With HIV in a Large Urban Public Health Care System in Houston, Texas, 2009–2014

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Background. Skin and soft-tissue infections (SSTIs) disproportionately impact patients with HIV. Recent declines have been noted in the incidence of SSTIs in the non-HIV population. We set out to study the epidemiology and microbiology of SSTIs in a population of 8,597 patients followed for HIV primary care in a large urban county system from January 1, 2009 to December 31, 2014.

Methods. SSTIs were identified from the electronic medical record (EMR) by the use of ICD-9 billing codes. Charts were reviewed to confirm the diagnosis of acute SSTI and abstract culture and susceptibility data. We calculated yearly SSTI incidence using Poisson regression with clustering by patient.

Results. 2202 SSTIs were identified. Of 503 (22.8%) cultured SSTIs, 332 (66.0%) included *S. aureus* as a pathogen, of which 287/332 (86.4%) featured *S. aureus* as the sole pathogen. Of *S. aureus* isolates with susceptibilities, 231/331 (69.8%) were methicillin-resistant, and the proportion did not vary by year (P = NS).

The observed incidence of SSTI was 78.0 per 1,000 person-years (95% CI 72.9–83.4) and declined from 96.0 infections per 1,000 person-years in 2009 to 56.5 infections per 1,000 person years in 2014 ($P < 0.001$). Other significant predictors of SSTI incidence in both univariate as well as multivariate analysis included CD4 count, viral load, and being a Spanish-speaking Hispanic.

Conclusion. Although SSTI rates in a large urban HIV-infected outpatient cohort declined approximately 40% between 2009 and 2014, SSTIs remain a significant problem.

Figure 1. SSTI incidence per 1000 person-years, 2009–2014

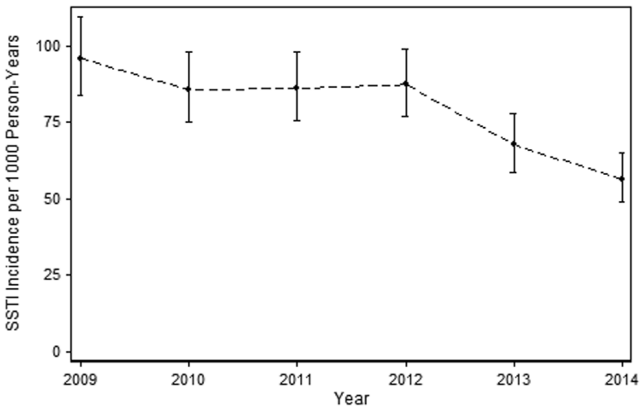

Table 1: Univariate and Multivariate Poisson Regression Results

Factor	Univariate IRR (95% CI)	Multivariate IRR (95% CI)
Male gender	1.06 (0.92–1.22)	–
MSM	1.09 (0.95–1.25)	–
Race/ethnicity		
White/Asian/other	Ref	Ref
Non-Hispanic African-American	0.92 (0.76–1.11)	0.86 (0.71–1.04)
English-speaking Hispanic	0.88 (0.69–1.12)	0.88 (0.69–1.12)
Spanish-speaking Hispanic	0.46¥ (0.36–0.60)	0.48¥ (0.37–0.62)
CD4 count (cells/μL)		
100+	Ref	Ref
50–100	1.56* (1.19–2.04)	1.23 (0.93–1.62)
<50	2.28¥ (1.80–2.90)	1.49* (1.16–1.92)
Viral load (copies/mL)		
<1,000	Ref	Ref
1,000+	2.17¥ (1.91–2.46)	1.90¥ (1.66–2.19)
Year		
2009	Ref	Ref
2010	0.89 (0.75–1.06)	0.92 (0.78–1.10)
2011	0.90 (0.75–1.07)	0.96 (0.80–1.15)
2012	0.91 (0.76–1.09)	0.99 (0.83–1.19)
2013	0.71¥ (0.58–0.85)	0.77* (0.64–0.93)
2014	0.59¥ (0.49–0.71)	0.65¥ (0.54–0.79)

*$P < 0.01$.

¥$P < 0.001$.

Disclosures. **C. Arias**, Merck & Co., Inc.: Grant Investigator, Research support. MeMed: Grant Investigator, Research support. Allergan: Grant Investigator, Research support

935. Repeat Pregnancies Among Women Living With HIV: Evaluating Temporal Changes in HIV Disease Status and Exploring the Association Between Preterm Birth and Protease Inhibitor Use in Pregnancy

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Background. 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 first 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 $\log_{10}$ 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.

Table: Paired Group and PTB

Paired Pregnancy Regimen Group	Percent of PTB		
First/second pregnancy	first pregnancy	second pregnancy	Difference
Non-PI/Non-PI (*n* = 103)	11.7%	9.7%	-2.0%
Non-PI/On 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%

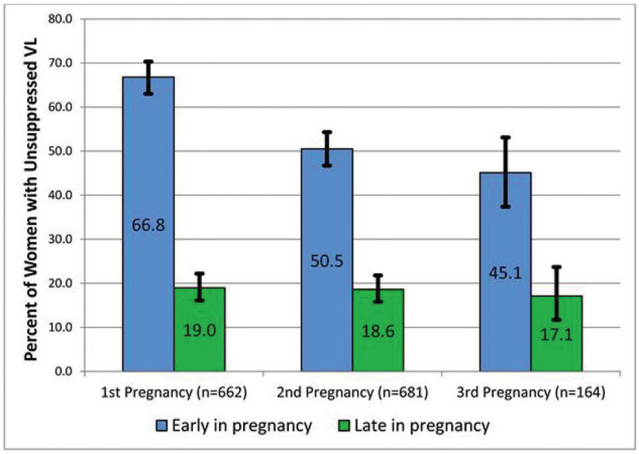

Figure: Percent of women with unsuppressed VL (>400 cp/mL)

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

936. 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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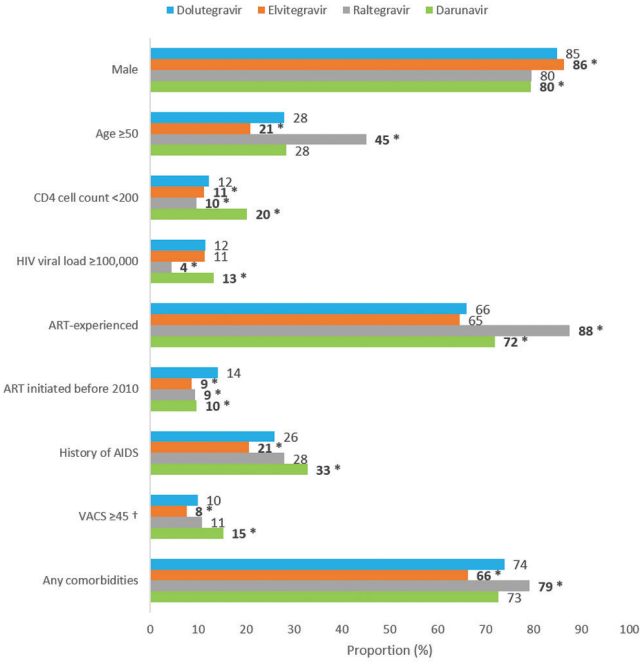
Background. Dolutegravir (DTG), elvitegravir (EVG), raltegravir (RAL), and darunavir (DRV) are commonly used for the treatment of HIV. We assessed the frequency of 6 select disorders after prescription of DTG-, EVG-, RAL-, or DRV-based regimens.

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.

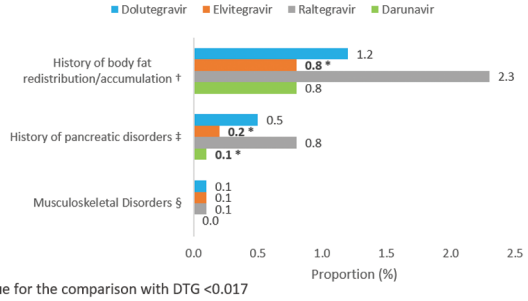
Conclusion. Incident body fat redistribution/accumulation, pancreatic disorders, musculoskeletal disorders, IRIS, severe systemic rash, and HSR were rare in this large cohort of patients initiating DTG, EVG, RAL, or DRV. Despite some channeling of patients with a disorder history towards DTG and RAL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

Figure 1. Baseline Demographic and Clinical Characteristics



* P-value for the comparison with DTG <0.017
† VACS Mortality Index: Scored by summing pre-assigned points for age, CD4 count, HIV-1 RNA, hemoglobin, platelets, aspartate and alanine transaminase, creatinine, and viral hepatitis C infection. A higher score is associated with a higher risk of 5-year all-cause mortality.

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



*** P-value** for the comparison with DTG <0.017
† Body Fat Redistribution/Accumulation: diagnosis of "lipohypertrophy", "lipoaccumulation", "hyperadiposity", "lipoatrophy", or "lipodystrophy"
‡ Pancreatic Disorders: diagnosis of "pancreatitis" or pancreatic adverse elevation (lipase >3X ULN)
§ Musculoskeletal Disorders: diagnosis of "Rhabdomyolysis" or musculoskeletal adverse elevations (CPK ≥10X ULN)