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 IDŬ	154 (31.6%)
People Not Born 1945–1965 With No Known IDU	179 (36.7%)

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

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Session: 115. HIV-Related Comorbidities and Complications Friday, October 5, 2018: 8:45 AM

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 ($P \le 0.04$). Low serum albumin also correlated with higher inflammatory monocytes (CD14+CD16+) at week 24 and week 96 ($P \le 0.03$) but not with markers of T-cell activation at any time point ($P \ge 0.10$). Lower baseline albumin significantly predicted larger changes in IMT (P = 0.03), IL6, d-dimer, tumor necrosis factor- α receptor 1, fibrinogen, and hsCRP ($P \le 0.02$) 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 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.

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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 Vagish Hemmige, MD¹, Cesar Arias, MD, PhD, FIDSA², Siavash Pasalar, PhD³ and Thomas P. Giordano, MD, MPH, FIDSA⁴, ¹Division of Infectious Diseases, Montefiore Medical Center, Bronx, New York, ²Microbiology and Molecular Genetics, University of Texas McGovern Medical School, Houston, Texas, ³HarrisHealth, Houston, Texas and ⁴Department of Medicine, Section of Infectious Diseases, Baylor College of Medicine, Houston, Texas

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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%)

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.

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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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