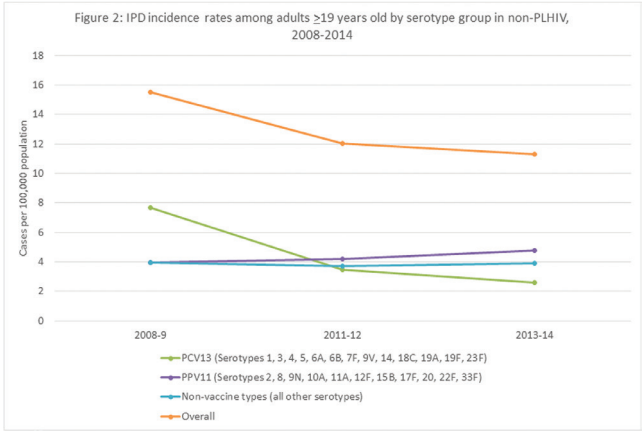

Disclosures. **L. Harrison**, GSK: Scientific Advisor, Consulting fee; **W. Schaffner**, Pfizer: Scientific Advisor, Consulting fee; Merck: Scientific Advisor, Consulting fee; Novavax: Consultant, Consulting fee; Dynavax: Consultant, Consulting fee; Sanofi-pasteur: Consultant, Consulting fee; GSK: Consultant, Consulting fee; Seqirus: Consultant, Consulting fee

1812. Cancer Mortality among Persons with Human Immunodeficiency Virus Infection in New York City, 2001–2015

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Background. With the prolonged life-span of persons with HIV (PWH) due to anti-retroviral therapy, their cancer burden has increased. Cancer continues to be a leading cause of death among PWH. Studying cancer mortality can inform and guide the development of cancer screening and prevention strategies for PWH.

Methods. We analyzed data for all persons > = 13 years who were diagnosed with HIV from 2001 to 2015 and reported to the New York City (NYC) HIV surveillance registry (HSR). Using the HSR and the underlying cause of death obtained from the NYC vital statistics registry and the National Death Index, we examined age-specific and age-standardized mortality rates from cancer and compared time trends of deaths due to HIV-related8 cancer to deaths from non-HIV-related cancers.

Results. There were 34,190 deaths reported among 154,688 PWH of whom nearly half (n = 16,804; 49.1%) died due to HIV (excluding HIV-related cancers). Among all deaths, HIV was the leading cause, followed by cancer (both HIV and non-HIV-related) (n = 5,271; 15.4%) and cardiovascular disease (n = 3,724, 10.9%). The top three causes of non-HIV-related cancer deaths were lung cancer (n = 1,040; 19.7%), liver cancer (n = 552; 10.5%), and colorectal cancer (n = 315; 5.6%). Although the mortality rate among PWH decreased over time (24.4 to 13.9 per 1,000 person-years from 2001 to 2015), the proportion of deaths attributable to all cancers increased (10.6% in 2001 to 19.9% in 2015, p < .0001). This increase was driven by non-HIV-related cancers (6.1% of all deaths in 2001 to 15.8% in 2015, p < .0001). The mean age increased from 2001 to 2015 among the dead (46 to 56 years) and among the censored (35 to 49 years). After controlling for demographic factors, transmission risk, and last CD4 count, the hazard ratio for cancer deaths was higher among people who inject drugs (HR = 1.5; 95% CI = 1.4–1.7) and those with last CD4 count < 200 (HR = 9.3; 95% CI = 8.3–10.5).

Conclusion. Although mortality rates are decreasing in PWH, deaths due to non-HIV-related cancers are increasing. The upward trend in the mean age suggests that aging may be contributing to this increase. Routine screening for liver and colon cancers along with smoking cessation may reduce lung, liver and colon cancer deaths.

Disclosures. **All authors:** No reported disclosures.

1813. Fatty Liver Disease in HIV: Predictors and Response to Statin Therapy

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Background. Liver disease has emerged as a leading cause of mortality and morbidity in HIV. Much of the current challenge in liver disease is related to nonalcoholic fatty liver disease (NAFLD). In HIV-uninfected populations, statin therapy has been suggested as potential intervention, but no such data is available in HIV. The aims of this study are to investigate the effect of rosuvastatin on hepatic steatosis in HIV infection, measured by Liver Fat Score (LFS) and to assess the natural history and predictors of changes in hepatic steatosis over 96 weeks.

Methods. This is a secondary analysis of the SATURN-HIV trial, in which HIV+ adults on stable ART with HIV-1 RNA < 1,000 copies/mL and LDL-cholesterol < 130mg/dL were randomized to 10mg daily rosuvastatin or placebo. Changes in LFS and in markers of systemic inflammation and monocyte activation were assessed from entry through week 96. Spearman correlations, multivariable linear regression and logistic regression were used to study relationships among variables.

Results. Overall, 147 patients were randomized (n = 72 to rosuvastatin n = 75 to placebo); 78% were male, 68% African Americans, 8% had chronic hepatitis C and mean age and BMI were 46 years and 29 kg/mm^2. A significant increase in LFS over 96 weeks was seen in both the placebo and statin arms (p = 0.01 and p < 0.01 respectively, p = 0.49 between groups). Furthermore, the progression from no-steatosis (LFS ≤ -0.64) at baseline to steatosis (LFS > -0.64) at week 96 was higher in rosuvastatin arm (OR = 4.3, p = 0.03), and remained statistically significant after adjusting for demographics, HOMA (baseline and change over 96 weeks), hepatitis C, heavy alcohol use and HIV parameters. Baseline LFS was independently associated with IP-10 (β = 0.82, p = 0.03) and sCD163 (β = 0.43, p = 0.005), and the increase in LFS over 96 weeks was independently associated with IP-10 (β = 2.85, p = 0.02).

Conclusion. In HIV+ subjects on ART, hepatic steatosis increased over time, regardless of statin treatment, and was independently associated with markers of immune activation. The progression from non-steatosis to hepatic steatosis was greater on statin. Despite its effective role in reducing cardiovascular disease risk and inflammation, statin therapy does not appear effective in hepatic steatosis.

Disclosures. **C. O. Hileman**, Gilead: Medical Advisory Board, Research support; **G. Mccomsey**, Gilead: Consultant, Consulting fee and Research support; BMS: Consultant, Consulting fee and Research support; GSK/ViiV: Consultant, Consulting fee and Research support; ICON: Consultant, Consulting fee; Merck: Investigator, Research support

1814. Leveraging the ART Advantage: diabetes and hypertension along the HIV care cascade in rural South Africa

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Session: 227. HIV: Co-morbidities and Co-infections
Saturday, October 7, 2017: 10:30 AM

Background. Participation in antiretroviral therapy (ART) programs has been associated with greater utilization of care for diabetes and hypertension in rural South Africa. However, there is limited data about whether this apparent "ART advantage" translates into improved chronic disease management indicators.

Methods. The Health and Aging in Africa: a Longitudinal Study of an INDEPTH Community in South Africa (HAALSI) is a cohort of 5,059 adults >40 in Agincourt. The study collects data on demographics, healthcare utilization, height, weight, blood pressure (BP), and blood glucose. HIV infection, HIV-1 RNA viral load (VL) and ART drug levels are tested via dried blood spots. We defined hypertension (HTN) based on measured BP or self-report of diagnosis by a healthcare provider or use of antihypertensive medication and diabetes (DM) by measured glucose or self-report of diagnosis by a healthcare provider or the use of DM medications. Our primary predictor of interest was stage along the HIV care cascade (HIV-, HIV+ not on ART, ART with a detectable VL, and with a suppressed VL). We compared the proportion in each subgroup who were aware of and treated for their hypertension or diabetes diagnosis, and fit adjusted linear regression models to estimate differences in systolic BP and glucose among those with diagnosed HTN or DM.

Results. Rates of HTN and DM were higher in HIV- than those with a suppressed VL (HTN: 68.4% v. 46.4%, DM: 12.9% vs.. 8.8%, respectively). However, the suppressed VL group had higher crude rates of awareness of HTN diagnosis and treated HTN as compared with the HIV- group (Aware: 69.9% vs.. 65.2%, p = 0.118; Treated: 50.2% vs.. 46.4%, p = 0.002). There were no significant differences in awareness or treatment rates for DM. In adjusted linear regression models among those with diagnosed HTN or DM, having a suppressed VL was associated with lower mean systolic BP (-5.94 mm Hg, 95% CI: -9.68 – -2.20) and lower mean glucose (-3.74 mmol/L, 95% CI: -5.95 – -0.58), compared with being HIV-. This effect was preserved in models restricted to overweight and obese participants.

Conclusion. The HIV care delivery platform in South Africa appears to offer a vehicle for healthcare delivery for other chronic conditions. Future studies are needed to assess causality of these relationships, and to determine optimal methods of integrating chronic disease with HIV management.

Disclosures. **All authors:** No reported disclosures.