Table 1. Demographic characteristics

Characteristic	Patients with HIV (n=90,900)	Patients without HIV or ART (n=17,946,580)
Sex		
Male	55,260 (60.8%)	7,776,510 (43.4%)
Female	35,640 (39.2%)	10,170,070 (56.6%)
Age, y		
18-44	29,010 (31.9%)	6,965,350 (38.8%)
45-64	39,060 (43.0%)	5,781,260 (32.2%)
65+	22,820 (25.1%)	5,199,970 (29.0%)
Race		
White	56,310 (62.0%)	14,129,350 (78.7%)
African American	24,720 (27.2%)	2,042,660 (12.5%)
Other	9,870 (10.9%)	1,570,670 (8.8%)
ART Treatment	35,610 (39.2%)	-
Obesity (BMI ≥ 30.0 kg/m^2)	36,280 (40.0%)	8,261,270 (46.0%)
Hypertension	45,010 (49.5%)	6,842,690 (38.1%)
Hyperlipidemia	44,980 (49.5%)	6,241,010 (34.8%)
Tobacco Smoker	49,690 (54.7%)	5,233,790 (29.2%)
Alcoholism	11,210 (12.3%)	683,660 (3.8%)
Hepatitis C	9,230 (10.2%)	213,890 (1.2%)
T2DM	20,080 (22.1%)	2,674,460 (14.9%)

BMI, Body mass index; T2DM, type 2 diabetes mellitus
Other includes patients whose race was recorded as "Hispanic/Latino", "Asian", "Multi-racial", or "Other", as well as patients with two or more races on record

Table II. Group-specific prevalence of T2DM

Characteristic	Patients with HIV (n=90,900)	Patients without HIV or ART (n=17,946,580)
Overall	22.1% (20,080/90,900)	14.9% (2,679,490/17,946,580)
Sex		
Male	20.8% (11,510/55,260)	16.5% (1,279,050/7,776,510)
Female	24.0% (8,570/35,640)	13.7% (1,395,410/10,170,070)
Age, y		
18-44	7.2% (2,090/29,010)	3.7% (254,850/6,965,350)
45-64	25.6% (9,980/39,060)	16.1% (931,640/5,781,260)
65+	38.8% (8,850/22,820)	28.6% (1,487,980/5,199,970)
Race		
White	21.3% (12,000/56,310)	14.1% (1,998,860/14,129,350)
African American	21.7% (5,360/24,720)	19.1% (430,010/2,042,660)
Other	27.6% (2,720/9,870)	4.7% (245,590/1,570,670)
ART Treatment		
Yes	17.6% (6,280/35,610)	-
No	24.9% (13,790/55290)	
Obese		
Yes	30.8% (11,190/36,280)	22.8% (1,880,130/8,261,270)
No	16.3% (8,890/54,620)	8.2% (794,330/9,685,310)
Hypertension		
Yes	37.7% (16,950 /45,010)	32.2% (2,201,130/6,842,690)
No	6.8% (3,130/45,890)	4.3% (473,330/11,103,890)
Hyperlipidemia		
Yes	35.1% (15,800/44,980)	32.5% (2,025,950/6,241,010)
No	9.3% (4,280/45,920)	5.5% (648,510/11,705,570)
Tobacco Smoker		
Yes	24.6% (12,210/49,690)	18.6% (974,260/5,233,790)
No	19.1% (7,870/41,210)	13.4% (1,700,200/12,712,790)
Alcoholism		
Yes	24.4% (2,730/11,210)	18.0% (122,990/683,660)
No	21.8% (17,350/79,690)	14.8% (2,551,480/17,269,920)
Hepatitis C		
Yes	29.5% (2,720/9,230)	25.3% (53,910/213,890)
No	21.2% (17,360/81,670)	14.8% (2,620,550/17,732,690)

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

341. Risk of Type 2 Diabetes Mellitus after Antiretroviral Therapy Initiation in Individuals Living with HIV in the United States

Ricky Hsu, MD[1]; Laurence Brunet, PhD[2]; Jennifer S. Fusco, BS[2]; Karam Mounzer, MD[3]; Vani Vannappagari, MBBS, MPH, PhD[4]; Cassidy Henegar, PhD[4]; Jean A. van Wyk, MB,ChB[4]; Lloyd D. Curtis, MA BM BCh MRCP[5]; Janet Lo, MD[6] and Gregory Fusco, MD, MPH[2]; [1]NYU Langone Medical Center – AIDS Healthcare Foundation, New York, New York; [2]Epividian, Inc., Durham, North Carolina; [3]Philadelphia FIGHT, Philadelphia, Pennsylvania; [4]ViiV Healthcare, Research Triangle Park, North Carolina; [5]GSK, Uxbridge, UK; [6]Harvard Medical School, Boston, Massachusetts

Session: 44. HIV Complications: Cardiovascular, Metabolic, and Other Complications
Thursday, October 3, 2019: 12:15 PM

Background. Limited data exist on the risk of type 2 diabetes mellitus (T2DM) with the use of integrase inhibitors. We assessed the risk of incident T2DM with antiretroviral therapy (ART).

Methods. ART-naïve (ART-N) and -experienced, suppressed (ART-ES; baseline viral load 13 years of age initiating dolutegravir (DTG), elvitegravir/cobicistat (EVG/c), raltegravir (RAL) or boosted darunavir (bDRV) in the OPERA® cohort. After excluding prevalent prediabetes/T2DM and missing baseline covariates, incidence rates of T2DM (i.e., diagnosis, antidiabetic drug, and/orHbA1C >6.5%) were estimated with Poisson regression. The association between core agents and incident T2DM was estimated with multivariate Cox proportional hazards regression adjusted for age, sex, race, HCV co-infection and BMI at baseline. Median (IQR) absolute BMI change from baseline was evaluated at 6, 12, 18, and 24 months in those who developed incident T2DM and those who did not. All analyses were stratified by ART experience.

Results. Individuals prescribed these ART regimens varied significantly (Figure 1). Overall, incidence rate per 1,000 person-years was low for T2DM (ART-N IR: 9.1; 95% CI: 7.5, 10.9; ART-ES IR: 13.3; 95% CI: 11.6, 15.2; Figure 2). Among ART-N initiators, no statistical difference was observed in the risk of progression to T2DM between DTG and EVG/c (aHR: 0.70; 95% CI: 0.47, 1.05) or bDRV (aHR: 0.53; 95% CI: 0.26, 1.04); RAL could not be evaluated due to the small number of T2DM events. Among ART-ES initiators; no difference was observed between DTG and EVG/c (aHR: 0.96; 95% CI: 0.70, 1.33), RAL (aHR: 1.17; 95% CI: 0.70, 1.96) or bDRV (aHR: 0.90; 95% CI: 0.57, 1.42) (Figure 3). A greater absolute change in BMI was observed for ART-N initiators developing T2DM at all timepoints; reaching statistical significance at 12 and 18 months (Figure 4). No differences were observed for ART-ES initiators.

Conclusion. Incident T2DM was uncommon among ART-N and ART-ES persons initiating DTG, EVG/c, RAL or bDRV in this large clinical population. None of the comparisons between DTG and other core agents showed a statistically significant increased risk of T2DM. However, due to the small number of events in the ART-N population differential risk cannot be excluded and monitoring HbA1c remains prudent.

Figure 1. Baseline demographic and clinical characteristics

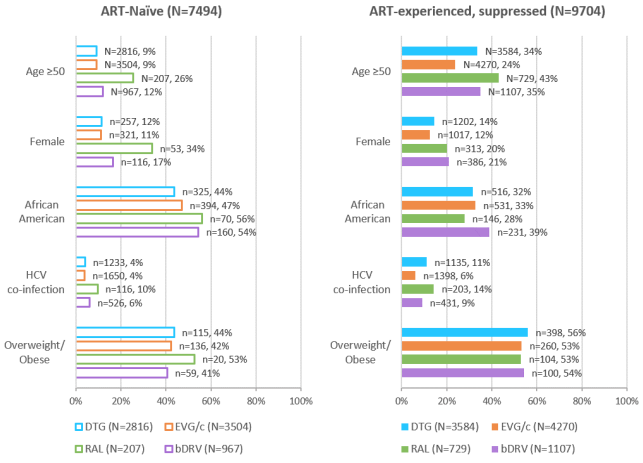


Figure 2. Incidence rates of T2DM and 95% confidence intervals, per 1,000 person-years

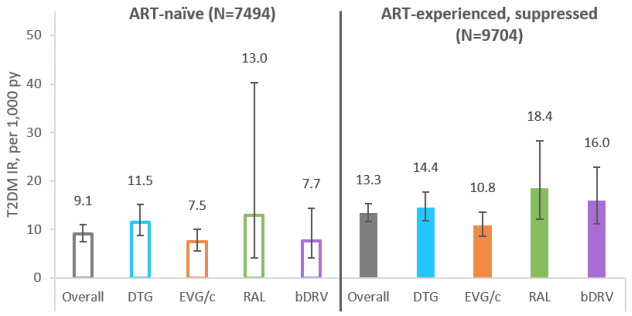
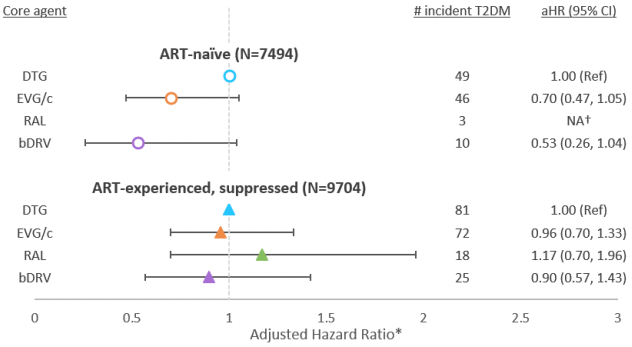
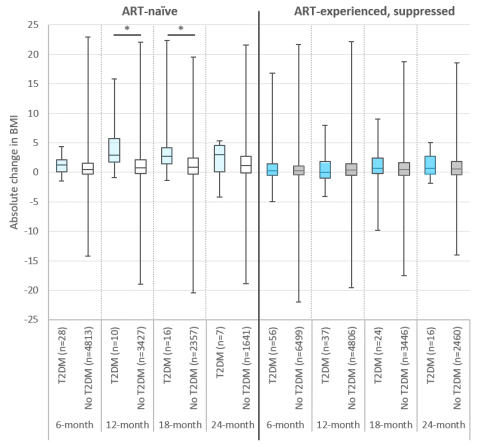


Figure 3. Adjusted hazard ratios* for the association between core agent and T2DM



* Obtained from Cox proportional hazards models adjusted for age, sex, race/ethnicity, HCV co-infection and BMI at baseline
† RAL was not included in the ART-naïve model due to the small number of incident T2DM

Figure 4. Overall changes in BMI from baseline to specific time points during follow-up†



* p-value <0.05
†Among those with a BMI measured both at baseline and at the time point of interest (±3 months)

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

342. The Impact of Glycemic Control on CD4 Cell Count in Persons Living with HIV and Diabetes Mellitus—Washington, DC

Lindsey J. Powers Happ, MPH[1]; Anne K. Monroe, MD, MSPH[1]; Heather A. Young, PhD, MPH[1]; Yan Ma, PhD, MA, MS[1]; Alan E. Greenberg, MD, MPH[2]; Debra A. Benator, MD[3]; Michael A. Horberg, MD[4] and Amanda Castel, MD, MPH[1]; [1]The George Washington University, Washington, DC; [2]George Washington University Milken Institute School of Public Health, Washington, DC; [3]Washington DC VA Medical Center, Washington DC; [4]Kaiser Permanente, Rockville, Maryland

Session: 44. HIV Complications: Cardiovascular, Metabolic, and Other Complications
Thursday, October 3, 2019: 12:15 PM

Background. Among persons living with HIV (PLWH) with type 2 diabetes mellitus (DM) there is limited research on the effect of DM control on CD4 count. Current guidelines recommend that PLWH with DM maintain a hemoglobin A1c (HbA1c) <7%. This analysis examined the impact of HbA1c on trends in CD4 count among PLWH receiving care in Washington, DC.

Methods. We used data from the DC Cohort, a longitudinal observational cohort of patients receiving HIV care at 14 clinics between 2011–2018. Participants with DM on an ongoing antiretroviral regimen with ≥1 year of follow-up, ≥2 HbA1c results, and ≥2 CD4 count results were included. Participants were compared based on the most recent HbA1c result categorized into one of three control levels control: strict, HbA1c < 7.5%; moderate, HbA1c between 7.5–9.0%; and uncontrolled, HbA1c >9.0%. All statistical tests were performed within the framework of the linear mixed-effects (LME) model. The rates of increase in CD4 count by DM control were compared using an LME model with random slopes and random intercepts, adjusted for sex, BMI, nadir CD4, a history of AIDS, or cancer diagnosis.

Results. Among 554 participants (median age 53.5; 70.8% male; 82.7% Black), there were 5,138 total CD4 count measurements. In unadjusted analysis, participants with moderate or uncontrolled HbA1c had higher mean CD4 counts over the follow-up period than those with strict HbA1c control (strict: 690 cells/μL, moderate: 712 cells/μL uncontrolled: 711 cells/μL; $P = 0.0156$ strict vs. moderate, 0.049 strict vs. uncontrolled). All DM control groups had a similar temporal increase over time in CD4 count ($P = 0.46$). In multivariate analysis, only moderate vs. strict control showed a significant difference in CD4 count (mean difference=18.1; $P = 0.02$). Results showed CD4 count change was not affected by the duration of HIV diagnosis or diabetes diagnosis. See Table 1 for additional results.

Conclusion. PLWH and DM with moderate HbA1c control had higher CD4 counts than those with strict HbA1c control and similar CD4 counts compared with those with uncontrolled HbA1c levels, while the rate of increase in CD4 count was similar in the three groups. These results show that moderate DM control may benefit CD4 count, which should be considered when revising DM control guidelines for PLWH.

Table 1. Univariate and multivariate models of average mean difference in CD4 cell count

Univariate Models

	Category	Average mean difference in CD4 count cells/μL (SE)	p-value*
HbA1c group	Uncontrolled (>9.0%)	22.2 (9.5)	**0.049**
	Moderate Control (7.5-9.0%)	21.8 (7.9)	**0.016**
	Strict Control (<7.5%)	reference	
Age at enrollment (median 53.5 years)	<Median	reference	
	≥ Median	-42.6 (26.1)	0.11
Sex	Females	168.9 (28.3)	**<0.0001**
	Males	reference	
Race/ethnicity	Non Hispanic black	reference	
	All other race/ethnicities	-28.9 (35.1)	0.411
Most recent BMI category	≥30 kg/m2	83.2 (14.8)	**<0.0001**
	25-30 kg/m2	36.8(11.6)	**0.002**
	<25 kg/m2	reference	
Diabetes diagnosis at enrollment	No	56.4 (29.6)	0.057
	Yes	reference	
On a non-insulin medication	No	-17.0 (9.2)	0.064
	Yes	reference	
On insulin	No	-7.0(11.2)	0.535
	Yes	reference	
Duration of HIV diagnosis (median 14.3 years)	<Median	3.7 (26.6)	0.891
	≥ Median	reference	
Nadir CD4 (median 281.5 cells/μL)	<Median	-288.1 (23.6)	**<0.0001**
	≥ Median	reference	
Duration of ARV use in database (median 4.4 years)	<Median	-36.2 (26.5)	0.172
	≥ Median	reference	
Has a cancer diagnosis	Yes	-118.9 (21.6)	**<0.0001**
	No	reference	
Has an AIDS defining diagnosis	Yes	-188.7 (33.5)	**<0.0001**
	No	reference	
Hypertension	Yes	35.6 (14.7)	**0.016**
	No	reference	
Dyslipidemia	Yes	20.4 (15.1)	0.178
	No	reference	
Multivariate Model **			
HbA1c group	Uncontrolled (>9.0%)	16.0(9.3)	0.085
	Moderate Control (7.5-9.0%)	18.1 (7.8)	**0.02**
	Stringent Control (<7.5%)	reference	

All statistical tests performed within the framework of the linear mixed effects model.
*p values in bold represent variables significant a p value of <0.05; p values for variables with more than two groups are adjusted for multiple comparisons using the Tukey method.
**Multivariate model was first fit with all variables found to be significant in univariate analysis. Hypertension was no longer significant in the multivariate model.
Final multivariate model reported was adjusted for sex, most recent BMI category, continuous nadir CD4, having a history of AIDS, and having a cancer diagnosis

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

343. T-cell Subsets Associated with Diabetes in Veterans with and without HIV

Samuel Bailin, MD[1]; Kathleen McGinnis, DrPH, MS[2]; Wyatt J. McDonnell, PhD[1]; Kaku So-Armah, PhD[3]; Melissa Wellons, MD[1]; Margaret Doyle, PhD[4]; Simon Mallal, MBBS[1]; Amy Justice, MD, PhD[5]; Matthew Freiberg, MD[1] and John Koethe, MD, MSCI[1]; [1]Vanderbilt University Medical Center, Nashville, Tennessee; [2]VA Connecticut Healthcare System, West Haven, Connecticut; [3]Boston University School of Medicine, Boston, Massachusetts; [4]University of Vermont, Colchester, Vermont; [5]Yale School of Medicine, West Haven, Connecticut

Session: 44. HIV Complications: Cardiovascular, Metabolic, and Other Complications
Thursday, October 3, 2019: 12:15 PM

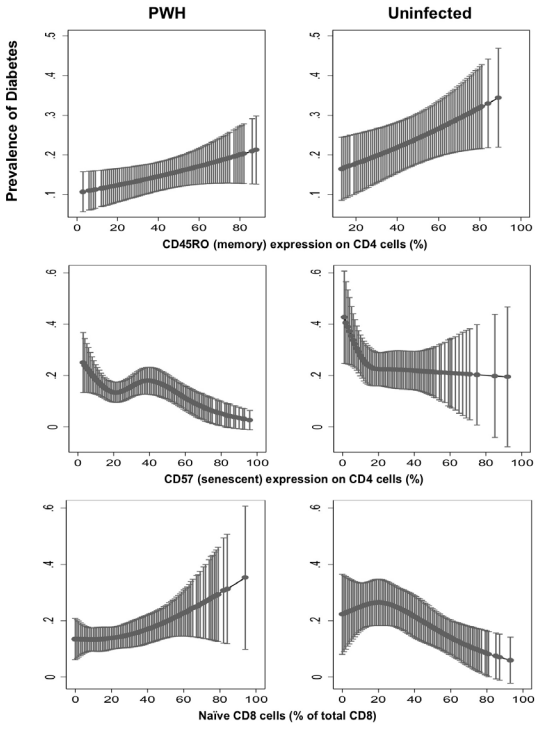
Background. Depletion of naïve CD4$^+$ T cells and elevated adaptive immune activation are hallmarks of HIV infection. Higher proportions of memory CD4$^+$ T cells are associated with prevalent diabetes in the general population, but few studies of persons with HIV (PWH) exist.

Methods. We analyzed data from 1532 PWH and 836 uninfected veterans in the longitudinal Veterans Aging Cohort Study (VACS), which archived peripheral mononuclear cells from these veterans between 2005 and 2007. We used flow cytometry to phenotype CD4$^+$ and CD8$^+$ T cells, including naïve, activated CD38$^+$, senescent CD57$^+$, total memory, and memory subsets. Prevalent diabetes (at blood collection) was identified in the VA electronic medical record using random glucose, hemoglobin A1c, ICD-9 codes, and medication. Cases were validated by two-physician chart review. We used multivariate logistic regression models adjusted for age, gender, body mass index, race/ethnicity, unhealthy alcohol use, hepatitis C, CMV status, and viral suppression stratified by HIV status to identify T-cell subsets associated with diabetes in PWH and uninfected.

Results. The cohort was 95% male, 68% African-American, and 22% diabetic. Higher CD4$^+$CD45RO$^+$ memory T cells were associated with prevalent diabetes in the uninfected and in PWH ($P = 0.03$ and $P = 0.07$, respectively; Figure A). Among subsets, diabetes was associated with higher transitional memory CD4$^+$ T cells in the uninfected ($P = 0.01$), but higher central memory cells ($P = 0.02$) and lower effector memory cells ($P = 0.04$) in PWH. T effector memory RA$^+$ cells were not associated with diabetes. Lower senescent CD4$^+$CD57$^+$ T cells were associated with diabetes in both PWH and uninfected ($P = 0.03$ and $P = 0.04$, respectively; Figure B), but results for naïve CD8$^+$ T cells diverged: diabetes was associated with higher naïve CD8$^+$cells in PWH but lower in uninfected ($P = 0.01$ and $P < 0.01$, respectively; Figure C). We assessed interaction by HIV status in a pooled model, which was only significant for the naïve CD8$^+$ T cells ($P = 0.01$).

Conclusion. The adaptive immune profile associated with prevalent diabetes was similar by HIV status and characterized by a shift in CD4$^+$ T cells from senescent to memory phenotypes, suggesting that chronic immune activation contributes to the higher risk of diabetes in PWH.

Figure: Adjusted prevalence of diabetes estimated for each T cell subset in PWH and uninfected Veterans



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