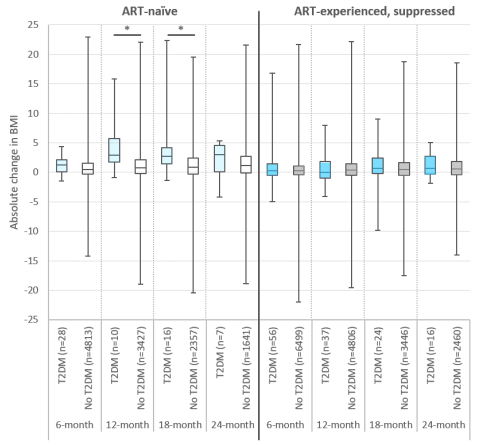
Figure 4. Overall changes in BMI from baseline to specific time points during follow-up†



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

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

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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

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

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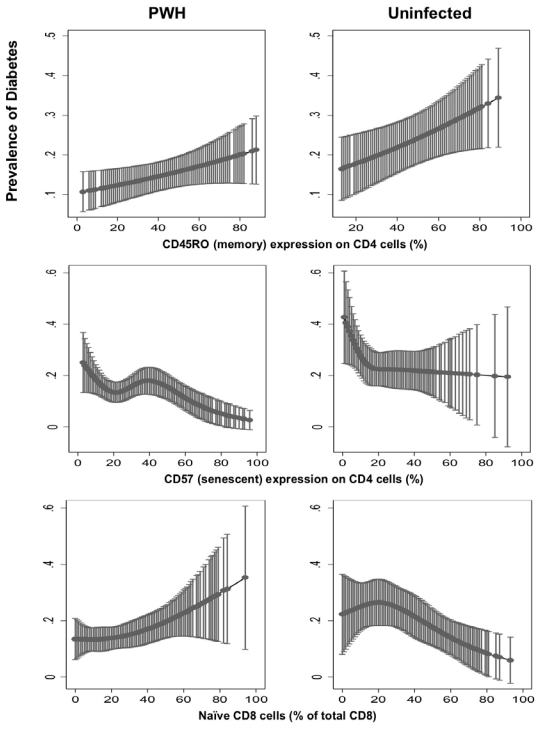
Background. Depletion of naïve CD4$^+$ T cells and elevated adaptive immune activation are hallmarks of HIV infection. Higher proportions of memory CD4$^+$ T cells are associated with prevalent diabetes in the general population, but few studies of persons with HIV (PWH) exist.

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



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