Cardiovascular Risk Profile: A Clinic-Based Sample of Youth Living with HIV in the US

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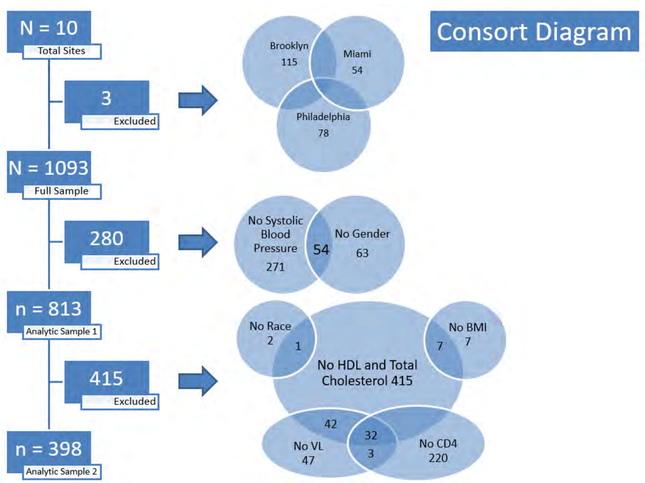
BACKGROUND

- Accelerated atherosclerosis has been found in young individuals with HIV.
- Previous research indicates that lower CD4 count and higher viral load (VL) significantly increased the risk of cardiovascular disease (CVD); however, this link is largely under-investigated among youth living with HIV (YLH).
- We examined whether detectable VL and low CD4 count have significant associations with increased CVD risk among YLH aged 14-26 years old.

METHODS

- This study used electronic health records from the *Adolescent Medicine Trials Network* 154 Cascade Monitoring baseline data.
- Multivariable linear regression was used to assess the association between detectable VL and CD4 of ≤200 with *Cardiac Risk Score1* (for those who had data on systolic blood pressure, cigarette smoking, diabetes, and anti-hypertensive medication use, *n* =813) and *Cardiac Risk Score2* (for those who had data on systolic blood pressure, cigarette smoking, diabetes, anti-hypertensive medications use, total cholesterol, and HDL cholesterol, *n* = 398) adapted from the Framingham gender-specific algorithm.



Our findings showed that detectable VL was associated with significantly increased risk of CVD, both at the bivariate level and in multivariable linear regression models. These findings contribute to our understanding of the broad spectrum of the VL-CVD risk relationship.

RESULTS

- The sample was predominantly Black and male with mean age of 21y. Overall, 47.8% had a detectable VL and 8.6% had a baseline CD4 of ≤ 200 indicating immune dysfunction.
- In bivariate analyses, we found significant associations between VL and CVD risk as measured by both cardiac risk scores (*Cardiac Risk Score1*, $p < 0.001$; *Cardiac Risk Score2*, $p < 0.01$).
- In the multivariable linear regression models, after adjusting for demographic and clinical covariates, we found that for a 1000-point increase in VL copies/mL, there was a 38% increase in the likelihood of having CVD risk (*Cardiac Risk Score2, B* = 0.038, 95% CI = 0.011, 0.066; $p < 0.01$.
- A small effect was observed for VL on increased CVD risk when measuring the strength of this association ($\beta = 0.134$). We observed similar findings with *Cardiac Risk Score1.*

Multivariable Linear Regression Models by *Cardiac Risk Score1*

Model 1 with Cardiac Risk Score1

	Model 1A with VL Results				Model 1C with VL and CD4 Results			
	Unstandardized			Standardized	Unstandardized			Standardized
	B	SE	95% CI	β	B	SE	95% CI	β
VL	0.002	0.001	(0.000, 0.003)	0.067**	0.002	0.001	(0.001, 0.004)	0.088**
CD4	---	---	---	---	0.000	0.000	(-0.001, 0.001)	0.015

Covariates: Age, Race/Ethnicity, Gender, ART Medication, Substance Abuse Diagnostics, BMI

Multivariable Linear Regression Models by *Cardiac Risk Score2*

Model 2 with Cardiac Risk Score2

	Model 2A with VL Results				Model 2C with VL and CD4 Results			
	Unstandardized			Standardized	Unstandardized			Standardized
	B	SE	95% CI	β	B	SE	95% CI	β
VL	0.038	0.014	(0.011, 0.066)	0.134**	0.040	0.016	(0.009, 0.071)	0.146*
CD4	---	---	---	---	-0.005	0.007	(-0.019, 0.010)	-0.035

Covariates: Age, Race/Ethnicity, Gender, ART Medication, Substance Abuse Diagnostics, BMI

CONCLUSIONS

- Higher plasma viral loads were associated with a small but statistically significant increased risk of CVD, independent of exposure to ART and presence of demographic and clinical covariates.
- Our findings demonstrate the independent contribution of detectable VL on cardiovascular risk and highlight the importance of maintaining VL suppression, monitoring metabolic health, and promoting self-management in YLH.

CONTACT INFORMATION

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