

Body composition and coronary artery plaque burden amongst an AAPI cohort

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Introduction

Existing studies identifying a positive correlation between BMI or central adiposity and coronary artery disease (CAD) often collect data post-mortem or via computed tomography angiography, and do not adequately represent Native Hawaiian and Other Pacific Islanders (NHPI) amongst cohorts.

Conversely, low body fat and BMI have been associated with the highest mortality in CAD patients¹.

Glucagon-like peptide-1 agonists (GLP-1a) reduce fat, lean mass, and cardiovascular death in obese patients with CAD².

This study aims to determine the significance and utility of body composition on the presence and severity of CAD via invasive coronary angiography amongst a predominantly Asian American and Pacific Islander population.

Methods

Investigated adults presenting for coronary catheterization.

Collected BMI; waist, hip, and forearm circumference; skinfold caliper measurements.

Assessed comorbidities, medications and illicit drug usage.

Coronary angiograms were reviewed and quantified for the number of significantly stenosed vessels, stent placement, and presence of chronic total occlusions (CTO). Stenoses >50% were defined as significant.

Results

NHPI participants had a significantly greater waist circumference, averaging 16.3 cm greater than non-NHPI participants. No significant interaction was found between NHPI status and the overall presence of CAD.

GLP-1a use was not associated with the presence of CAD, but tobacco and statin use showed significant associations.

NHPIs had the highest incidence of hyperlipidemia, hypertension, and kidney disease, while having the lowest rates of statin use, and highest use of GLP-1a.

Table 1. Prevalence of comorbidities, drug, and medication use across study participants

	NHPI (n=19)	White (n=13)	Asian (n=17)	Total (n=49)
Diabetes	52.6%	23.1%	52.9%	44.9%
Hyperlipidemia	89.5%	61.5%	88.2%	81.6%
Hypertension	94.7%	84.6%	82.4%	87.8%
Chronic Kidney Disease	47.4%	23.1%	17.6%	30.6%
Preexisting CAD	57.9%	61.5%	52.9%	57.1%
Tobacco	42.1%	53.8%	52.9%	49.0%
GLP-1a	31.6%	0.0%	11.8%	16.3%
Statin	63.2%	69.2%	82.4%	71.4%

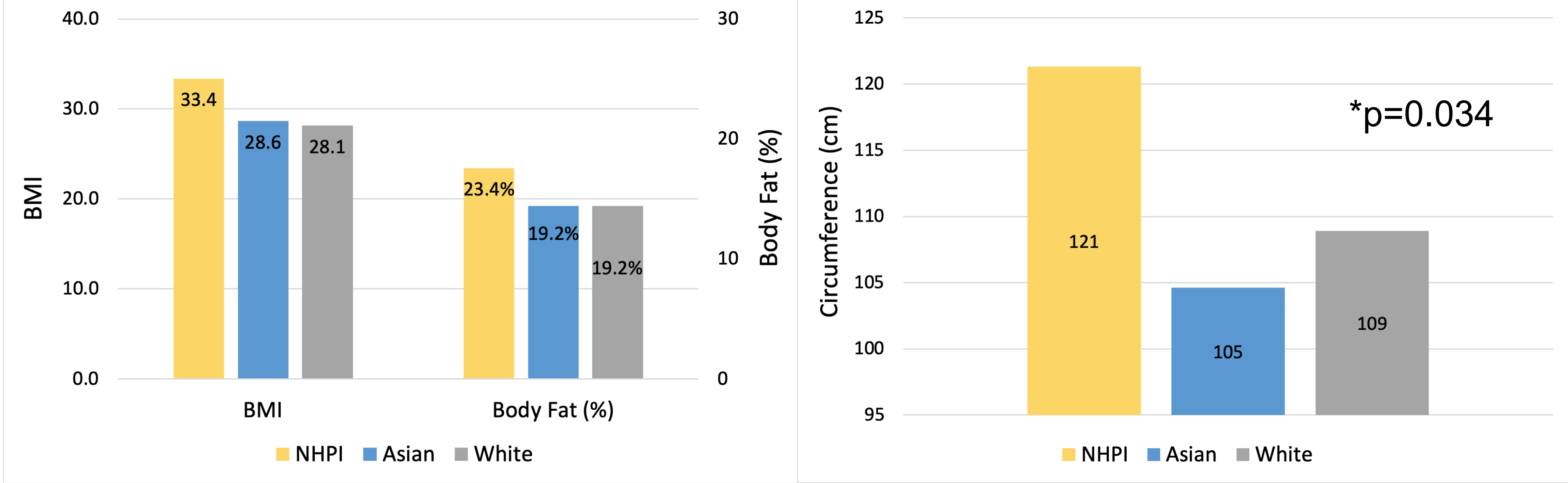

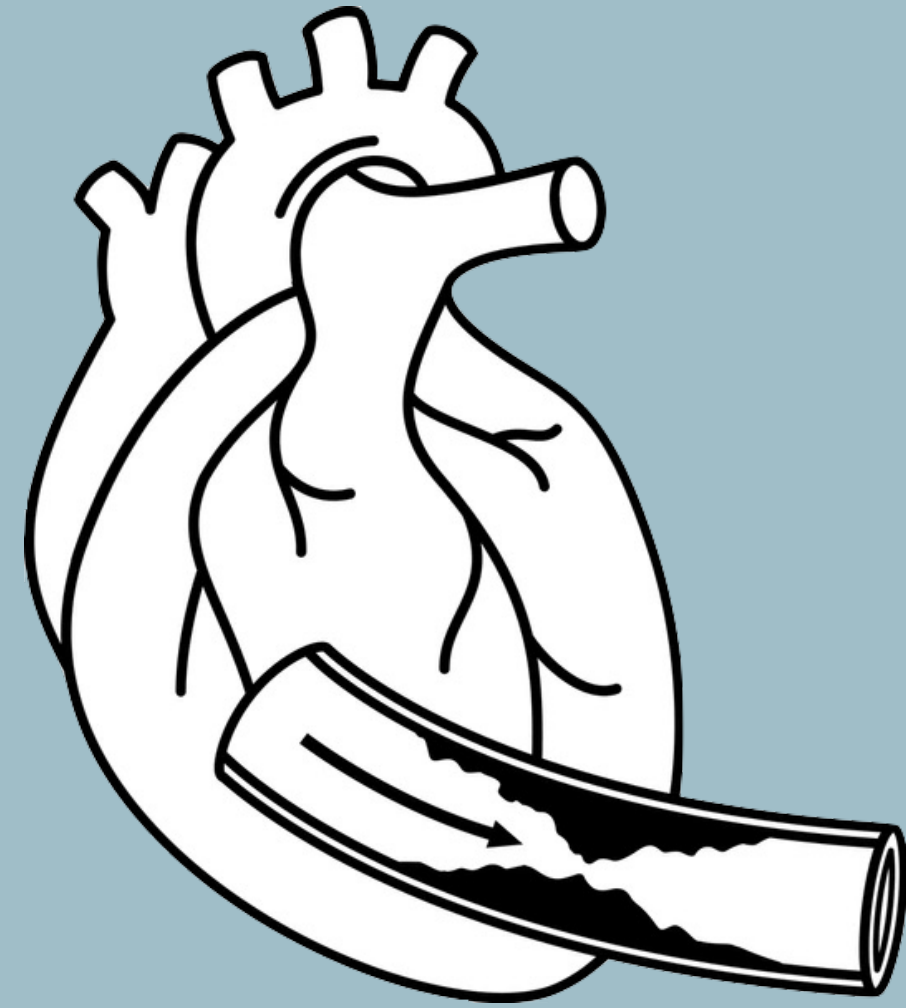


Figure 1. BMI and body fat stratified by ethnic group **Figure 2.** Waist circumference stratified by ethnic group




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Body composition was not found to be associated with the presence or severity of coronary artery plaque burden



Despite an increased mean waist circumference, NHPI participants had a lower incidence of CTO



ANOVA Linear Regression: Number of Stenotic Vessels and Body Fat Composition

Model		Sum of Squares	df	Mean Square	F	p
M₁	Regression	3.034	1	3.034	1.132	0.293
	Residual	125.946	47	2.680		
	Total	128.980	48			

ANOVA Linear Regression: Number of Stenotic Vessels and BMI

Model		Sum of Squares	df	Mean Square	F	p
M₁	Regression	1.784	1	1.784	0.659	0.421
	Residual	127.195	47	2.706		
	Total	128.980	48			

ANOVA Linear Regression: Number of Stenotic Vessels and Waist Circumference

Model		Sum of Squares	df	Mean Square	F	p
M₁	Regression	5.857	1	5.857	2.236	0.142
	Residual	123.122	47	2.620		
	Total	128.980	48			

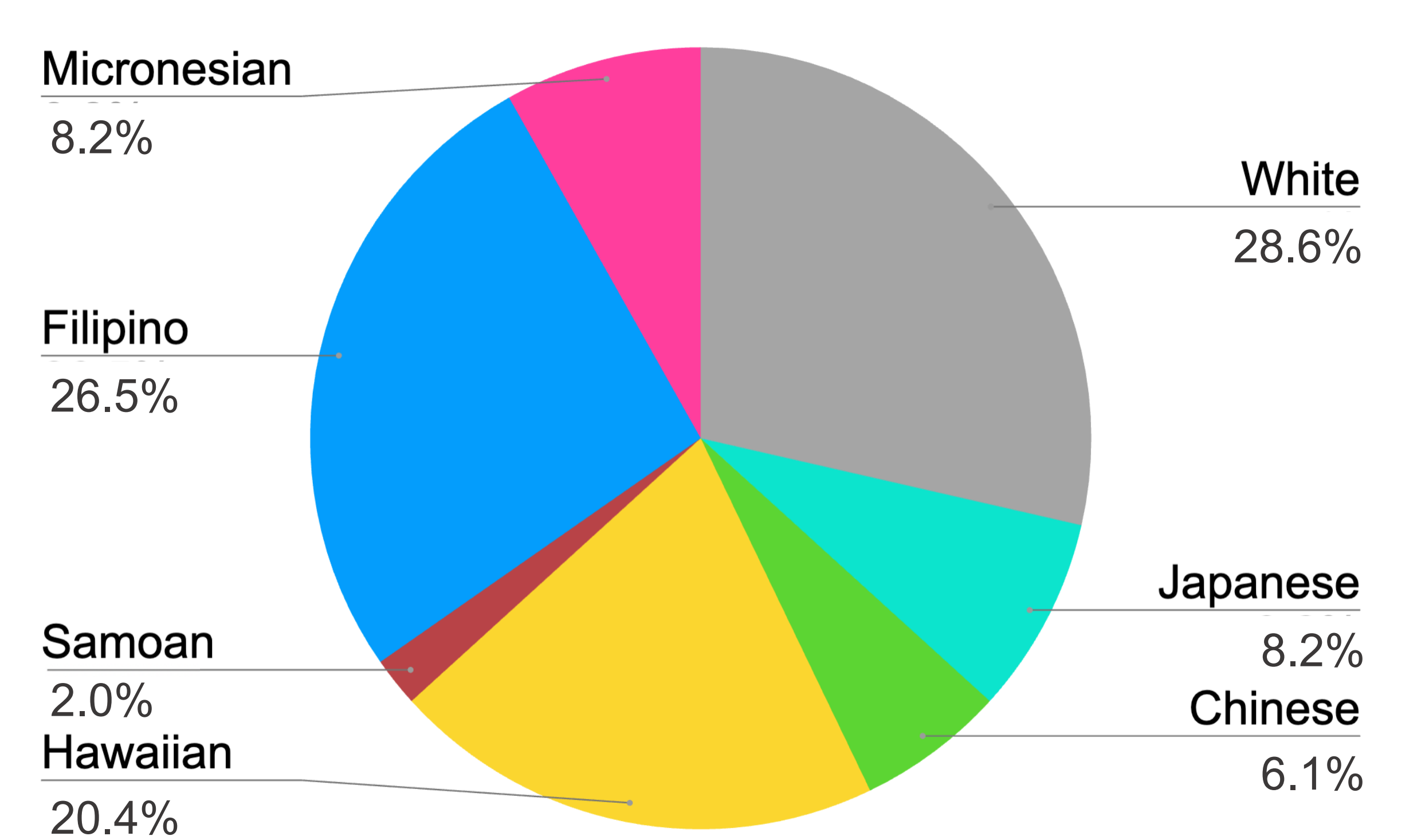


Figure 3. Ethnic distribution of participants

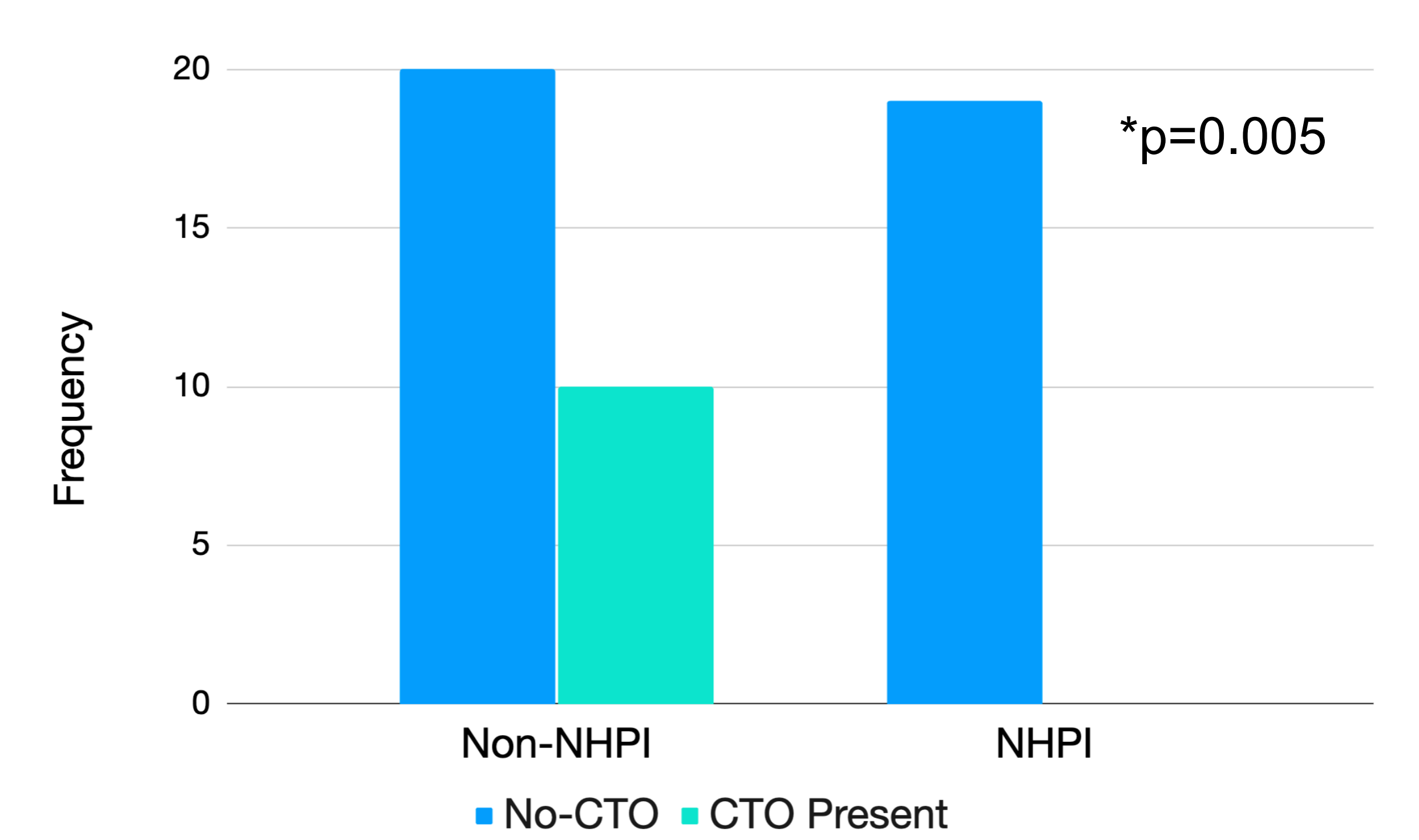


Figure 4. CTO prevalence by NHPI status

Discussion

While previous studies link central adiposity to CAD, our findings suggest these metrics are not associated with significant stenotic disease and may be less useful amongst AAPI patients undergoing invasive angiography.

The significant association of tobacco use with the presence and extent of CAD aligns with known pathophysiological mechanisms that promote atherosclerosis, however nonsignificance amongst other traditional risk factors in this study imply limitation in sample size and statistical power.

NHPI participants had a lower prevalence of CTO, despite similar rates of CAD overall. This may reflect the lower age at enrollment of NHPIs compared to other ethnic groups (61 vs 67 years). Whether additional genetic or socioeconomic factors unique to NHPIs alter coronary physiology or the natural disease progression of CTOs, or if the high rate of GLP-1a use affected CTO progression remains uncertain.

Future research should further explore the impact GLP1a have on plaque progression and stabilization, differences in adiposity between NHPIs and other ethnic groups, and whether the validity of traditional body composition metrics in predicting cardiovascular disease remains consistent amongst AAPI cohorts.

Acknowledgements

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