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Diet quality, inflammation, and the ankle brachial index in adults with or without cardiometabolic conditions

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Abstract

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Statement of authorship

J.M. researched the literature, developed the study concept and design, analyzed and interpreted the data, and organized and wrote the manuscript. D.S.A. assisted with statistical analysis, provided data verification and interpretation, and critically revised and edited the manuscript. F.B.H., K.L.T., and R.C.K. contributed to study design and concept, data interpretation, and careful revision of the manuscript. S.C., M.G., and A.M.S.-R. contributed to the interpretation of data and critically reviewed and edited the manuscript. J.M. had full access to all the data in the study and takes responsibility for its integrity and the data analysis. All authors contributed meaningful intellectual content to the manuscript and read and approved the final version.

Conflict of interest

The authors have no conflicts to report.

Background and aims—Diet quality may influence non-traditional cardiovascular disease (CVD) risk factors – namely, C-reactive protein (CRP) and the ankle-brachial index (ABI). Preexisting traditional cardiometabolic conditions may confound this association. We aimed to determine whether diet quality was associated with high-risk CRP or ABI, independently from traditional cardiometabolic risk factors.

Methods—Baseline data were analyzed from US-Hispanics/Latinos aged 18–74y without previously-diagnosed CVD participating in the population-based Hispanic Community Health Study/Study of Latinos cohort. Included were 14,623 participants with CRP data, and 7,892 participants (45y) with ABI data. Diet quality was measured with the Alternate Healthy Eating Index (AHEI).

Results—Nearly 35% of Hispanics/Latinos had high-risk CRP concentration and 6.3% had high-risk ABI (peripheral artery disease (PAD): 4.2%; arterial stiffness: 2.1%). After adjusting for sociodemographic and lifestyle factors, diabetes, hypertension, hypercholesterolemia, and obesity, the odds (95% confidence interval) of having high-risk ABI were 37% (5, 44%) lower per 10-unit increase in AHEI ($p=0.018$). The association was marginally significant for PAD (0.77 (0.58, 1.00); $p=0.05$), and non-significant for arterial stiffness ($p=0.16$). Each 10-unit increase in AHEI was associated with 21% (10, 30%) lower odds of high-risk CRP ($p=0.0002$) after similar adjustments. There were no significant interactions between AHEI and age, sex, ethnicity, smoking, or pre-existing cardiometabolic conditions for associations with ABI. The association between AHEI and high-risk CRP was stronger for those with diabetes (p -interaction<0.0001), obesity (p -interaction=0.005), or ages 45–74y (p -interaction=0.011).

Conclusions—Higher diet quality is associated with lower inflammation and less adverse ABI among Hispanics/Latinos, independently from traditional cardiometabolic risk factors.

Keywords

diet quality; inflammation; ankle brachial blood pressure index; subclinical vascular disease; peripheral arterial disease; HCHS/SOL

Introduction

Non-traditional risk factors of cardiovascular disease (CVD) may improve the prediction of CVD risk when assessed together with traditional risk factors such as biological and cardiometabolic markers^{1–4}. Two of these novel non-traditional CVD risk factors are C-reactive protein (CRP), an inflammatory marker, and the ankle brachial blood pressure index (ABI), a marker of subclinical vascular disease, namely peripheral arterial diseases (PAD) or arterial stiffness. Each marker has been estimated to be an independent risk factor for cardiovascular events^{2, 3, 5, 6}. Because of the potential role of non-traditional risk factors on CVD-risk prediction, it is imperative to identify health behaviors that may prevent them.

Consuming a healthy diet is well known to prevent and control traditional cardiometabolic risk factors for CVD, such as type 2 diabetes, obesity, hypertension, and high blood cholesterol⁷. The literature on diet and non-traditional markers is sparser. While the majority of studies suggest a significant inverse association between indices of diet quality and CRP^{8–18}, some have shown null results^{19, 20} or associations modified by other risk

factors²¹. Population-based studies on diet quality and ABI measures are limited and are more inconsistent^{22,23,24,15, 25}.

Discrepancies in findings may be due to variations in study design and sample size, characteristics of the population, statistical model adjustment, and definition of diet quality. Several indices of diet quality have been used to study diet-disease associations. Particularly, the Alternate Healthy Eating Index (AHEI) captures multiple foods and nutrients with recent and strong evidence of association with lower disease risk, and it uses absolute measures to define the components according to cutoffs relevant to disease risk^{26–28}. Furthermore, there is ample evidence that traditional and non-traditional biological risk markers co-occur^{4, 29}. Thus, statistical models testing the association between diet quality and non-traditional risk factors, especially those of cross-sectional design, should adjust for traditional cardiometabolic factors to properly control for confounding and better determine the strength of the association independently from traditional markers^{4, 29}. Few of the aforementioned studies on diet quality and CRP or subclinical vascular disease fully adjusted for cardiometabolic conditions^{11, 17, 29}.

Thus, this study aimed to determine the extent to which AHEI is associated with high-risk CRP and ABI (PAD and arterial stiffness), after adjusting for traditional cardiometabolic risk factors. Secondly, we aimed to determine if traditional risk factors modulated the association between diet and CRP or ABI. We investigated these questions in the Hispanic Community Health Study/Study of Latinos (HCHS/SOL), a cohort that shows low to moderate scores of diet quality³⁰ and high prevalence of cardiometabolic risk factors³¹. Notably, US-Hispanics/Latinos present high CRP concentrations^{32–34} and high-risk ABI levels³³. Determining the association of diet with non-traditional CVD risk factors can reinforce the merits of following a healthy diet as a clinically-relevant primordial and primary behavioral strategy that could prevent further CVD.

Materials and Methods

Study population and data collection

Baseline data collected between 2008 and 2011 from the Hispanic Community Health Study/Study of Latinos (HCHS/SOL) were used for this analysis. HCHS/SOL is a community-based prospective cohort study of 16,415 Hispanic/Latino individuals³⁵. Potentially eligible individuals were identified from randomly selected households, following a stratified two-stage area probability sampling of household addresses in each of four U.S. field centers (Chicago, IL; Miami, FL; Bronx, NY; and San Diego, CA). Eligible participants had to be community-dwelling men and women (not institutionalized or on active military duty), living in the identified household, ages 18–74y, self-identified as having Hispanic/Latino ethnicity (hereafter referred using their country of origin), able to attend a field center clinic, and not planning to move within 6 months. All participants signed informed consent. The institutional review boards of each field center, coordinating center, reading centers, and the National Heart, Lung, and Blood Institute approved this study.

Data collection has been described in detail previously^{31, 35}. Briefly, standardized clinical assessments and interviews were conducted by centrally-trained personnel in the participant's preferred language during a visit to one of the study centers. Participants self-reported their demographic and socioeconomic characteristics, medical diagnoses and use of medications (self-reported and scanned by interviewer), and family history of main chronic diseases, including coronary heart disease.

Dietary assessment and definition of diet quality

Dietary assessment and the definition of AHEI have been described in detail previously^{30, 36, 37}. Briefly, two 24-hour recalls were administered in the participant's language of preference, one in-person at the baseline visit and one via telephone or in-person within 5–90 days of the baseline visit. We excluded recalls with energy intake <1st or >99th sequence-sex-specific percentile, or deemed unreliable by the interviewer. Foods and nutrients were analyzed using the multiple-pass methods of the Nutrition Data System for Research (NDSR) software version 11 from the Nutrition Coordinating Center at University of Minnesota, which includes Hispanic/Latino foods.

The AHEI-2010 includes 11 dietary components: vegetables without potatoes, whole fruit without fruit juice, whole grains, sugar sweetened beverages and fruit juice, nuts and legumes, red and processed meat, *trans* fat, omega-3 fatty acids, polyunsaturated fatty acids, sodium, and alcohol. Each component was created by adding the corresponding NDSR food subgroups. Predicted usual intake amounts for each component were then estimated using the National Cancer Institute method. Each component was scored from 0–10 as continuous (prorated intermediate values) from minimal to maximal observance of the recommended amount of each item. Lastly, individual components' scores were summed. AHEI scores ranged from 0 to 110 (unhealthiest to healthiest).

Outcomes definition

A Roche Modular P Chemistry Analyzer was used to analyze serum high-sensitivity CRP (Roche Diagnostics Indianapolis, IN). CRP values were categorized as either low-risk (<1.0mg/L), moderate-risk (1.0–3.0mg/L), elevated-risk (>3.0–10.0mg/L), or acute inflammation (>10.0mg/L). A dichotomous high-risk CRP category was defined as >3.0mg/L versus low-risk ≤ 3.0mg/L³⁸.

The ankle brachial index was measured using standardized procedures as previously described³³. Participants aged ≥45y were measured for appropriate cuff size, and four cuffs were placed on each ankle and each upper arm. While the participant was lying down and rested, systolic blood pressures were measured once, starting with the right arm, at the bilateral brachial, anterior tibial, and posterior tibial arteries. Limb-specific ABI was computed as the highest ipsilateral ankle artery pressure divided by the highest brachial artery pressure. This ankle-to-arm SBP ratio was used as the final ABI, using the lower of the two limb values. PAD was defined as having an ABI ≤0.90 (based on evidence of a doubling in risk of total mortality, cardiovascular mortality, and major coronary events)³⁹, and arterial stiffness was defined as ABI ≥1.40 (based on evidence of increased risk of poor arterial compressibility resulting from stiffness and calcification)^{39–41}. Given that both

classifications reflect elevated risk for cardiovascular death or event^{39–41}, and following previous examples^{42, 43}, a combined category of high-risk ABI included those with either PAD or arterial stiffness.

Covariate definitions

The definition of lifetime cigarette packs-years has been previously described⁴⁴. Participants were asked if they had ever smoked at least 100 cigarettes in their entire life, as well as use of cigars and pipes, number of cigarettes per day, age at smoking initiation, and periods of smoking cessation and use of smoking-cessation aids. Lifetime pack-years was estimated as the difference from the age the participant began smoking to either the participant's age at baseline examination or the age that the participant quit smoking (adjusted for any years without smoking during the time period), multiplied by the average number of cigarettes smoked per day. The Global Physical Activity Questionnaire was used to assess physical activity⁴⁵. Self-reported hours of physical activity and sedentary behavior were converted into metabolic equivalents, and categorized as high, moderate, or low levels.

BMI was calculated as weight in kilograms (measured to the nearest 0.1kg in a Tanita body composition analyzer) divided by height in meters squared (measured to the nearest centimeter). Waist circumference and height were measured using an anthropometric tape. The average of three blood pressure observations, measured with an automatic sphygmomanometer after a quiet rest, was recorded. Fasting blood samples were collected soon after arrival and shipped to the central laboratory for analysis. A Roche Modular P Chemistry Analyzer was used to analyze serum triglycerides, serum HDL-C, serum LDL-C, and plasma glucose (Roche Diagnostics Indianapolis, IN). A Tosoh G7 automated high-performance liquid chromatography analyzer (Tosoh Bioscience Inc., San Francisco, CA) was used to measure glycosylated hemoglobin (HgA1C) in EDTA whole blood.

Diabetes was defined as having fasting glucose ≥ 126 mg/dL (for fasting time ≥ 8) or fasting glucose ≥ 200 mg/dL (for fasting time ≥ 8) or post-oral glucose tolerance test ≥ 200 mg/dL or HgA1C $\geq 6.5\%$, or a self-report of medically-diagnosed diabetes, or medication use for high blood sugar or diabetes. Obesity was defined as BMI ≥ 30 . Hypertension was defined as blood pressure $\geq 140/90$ or use of hypertension medication. Hypercholesterolemia was defined having a total cholesterol ≥ 240 mg/dL, or LDL-C ≥ 160 mg/dL, or HDL-C <40 mg/dL, or use of lipid-lowering medications.

Statistical analysis

For the CRP analysis, we excluded 1,504 of the 16,415 HCHS/SOL participants because of pre-existing coronary heart disease (CHD) or cerebrovascular disease as defined by abnormalities detected in an electrocardiogram report (indicative of possible old myocardial infarction), or self-report of angina, heart attack, stroke, mini-stroke or transient ischemic attack, or procedure (angioplasty, stent, bypass) in the heart or to the arteries of the neck. Additionally, 152 participants with missing dietary data to calculate AHEI and 136 with missing CRP values were excluded. Thus, data from 14,623 participants were available for CRP analysis.

By HCHS/SOL design, 60% participants (n=9,714) were ≥45y; of these, 9,706 participants had ABI measures. We excluded 1,201 participants with pre-existing CHD or cerebrovascular disease as defined above, an additional 536 individuals who self-reported a previous diagnosis of PAD, and 77 participants with missing AHEI data. Thus, a total of 7,892 participants were used in the ABI analysis.

Differences in characteristics by categories of CRP and ABI were performed using age-adjusted survey regression for continuous variables, and chi-square tests for categorical variables. Odds ratios (OR) and 95% confidence intervals (CI) of having high-risk CRP or high-risk ABI levels as dichotomous outcomes were estimated from survey logistic regression models, for each 10-unit increase of AHEI, controlling for age, sex, ethnic background, center, family history of CHD, total energy intake, household income, marital status, health insurance, years living in the US, physical activity, cigarette packs per year, and use of non-steroidal anti-inflammatory drugs (NSAID). An additional model adjusted for four cardiometabolic risk factors: diabetes, hypertension, hypercholesterolemia, and obesity. The generalized logit function was used in logistic regressions testing the odds of multiple categories of CRP (low, moderate, elevated, and acute) and ABI (normal, PAD, arterial stiffness). A linear regression model tested the association between 10 units of AHEI and CRP as a continuous outcome adjusted for the same covariates. The interaction terms between AHEI and ethnic background, and with traditional CVD risk factors (age, sex, cigarette use, physical activity, diabetes, hypertension, hypercholesterolemia, and obesity) were tested in logistic regression models. Sensitivity analyses were conducted: 1) by using abdominal obesity (waist circumference >102 cm for men, >88 cm for women) instead of BMI cutoffs to define obesity, and 2) excluding those with acute inflammation in the CRP analysis, or reclassifying the normal ABI category as two categories: normal (1.0 ≤ABI<1.4) or intermediate-risk (0.90<ABI<1.0) values, to create a 4-level ABI outcome. All analyses accounted for clustering and stratification and were weighted to adjust for sampling probability of selection and non-response, using complex survey procedures in SAS software v9.4 (SAS Institute, Cary, NC). A significance level of $p<0.05$ was used.

Results

Over a third of the study sample had high-risk concentrations of CRP, and 6.3% had high-risk ABI, of which 4.2% were classified with PAD and 2.1% with arterial stiffness (Table 1). High-risk concentrations of CRP were more likely to be observed among individuals who were older, female, divorced/widowed, with a family history of CHD, lower household income, or had more years of residency in the US. Individuals with high-risk CRP were more likely to report using anti-inflammatory drugs, be exposed to smoking for ≥10 years, have low physical activity levels, lower mean AHEI, and be of Mexican, Puerto Rican or Cuban origin.

High-risk levels of ABI, PAD, and arterial stiffness were more often observed among individuals who were older, divorced/widowed, with smoking exposure ≥10 years, fewer years of residency in the US, low physical activity levels, and lower AHEI scores. Cuban ethnicity was predominant among those with high-risk ABI and PAD, whereas arterial stiffness was more prevalent among adults of Mexican and Cuban origin. Women were more

likely to be classified with PAD but less likely to have arterial stiffness. Having health insurance was more likely among those with arterial stiffness. All four cardiometabolic risk factors were more prevalent among those with high-risk CRP concentrations and those with high-risk ABI.

In analyses relating AHEI with study outcomes, interaction tests between AHEI and ethnic background were non-significant ($p=0.22$ for CRP and $p=0.75$ for ABI); thus all models presented are for all backgrounds combined. The likelihood of having high-risk CRP concentrations was 23% lower for every 10 units of AHEI score, after adjusting for sociodemographic and lifestyle factors (OR (95%CI)=0.77 (0.68, 0.86); $p<0.0001$) (Figure 1). Adjusting for diabetes, hypercholesterolemia, hypertension, and obesity slightly attenuated this association, but it remained significant (0.79 (0.70, 0.90); $p=0.0002$). When high-risk CRP was categorized as moderate-risk, elevated-risk, and acute concentrations, significant associations with AHEI remained for elevated-risk (0.80 (0.68, 0.94); $p=0.007$) and acute (0.68, (0.54, 0.85); $p=0.001$) but not for moderate-risk (0.97 (0.85, 1.10); $p=0.61$) in the fully-adjusted model. In sensitivity analysis, results were identical when we adjusted for abdominal obesity rather than BMI-defined categories, or if we excluded those with acute inflammation from the high-risk category (data not shown). Analysis with CRP as a continuous outcome showed a significant inverse association with every 10 units of AHEI (β (SE) = -0.06 (0.03); $p=0.0004$).

We tested interactions between traditional CVD risk factors and AHEI in association with high-risk CRP concentrations. Significant interactions were observed for age category (18–44y vs. 45–74; p -interaction=0.011), diabetes (p -interaction<0.0001), and obesity (p -interaction =0.005) (Table 2). Significant lower odds of high-risk CRP with more favorable AHEI were observed for those aged 45–74 (0.73 (0.63, 0.85); $p<0.0001$), or those with obesity (0.69 (0.57, 0.83); $p=0.0001$), but not for those aged 18–44 or without obesity. The association between AHEI and high-risk CRP for individuals classified as overweight ($25\text{kg/m}^2 \leq \text{BMI} < 30\text{kg/m}^2$) was significant but weaker than for obesity (0.76 (0.63, 0.92); $p=0.005$). The likelihood of high-risk CRP was lower with higher AHEI for individuals regardless of the presence or absence of diabetes; however, the point estimate of the association was stronger for those with diabetes compared to those without (0.69 (0.53, 0.90) vs. 0.81 (0.71, 0.93); Table 2). The point estimate was stronger for those with untreated diabetes (0.60 (0.42, 0.86)), which was nearly half of those with diabetes. Other interactions with AHEI tested for the outcome of CRP (i.e. sex, cigarette use, physical activity, hypertension, or hypercholesterolemia) were not statistically significant.

The association between AHEI and high-risk ABI was not significant (OR=0.79 (0.61, 1.02); $p=0.07$) in the model adjusted for sociodemographic and lifestyle factors (Figure 2). After adjusting for the four cardiometabolic risk factors, each 10 units of AHEI were associated with 37% (5, 44%) lower odds of having high-risk ABI (OR=0.73 (0.56, 0.95); $p=0.02$). The association remained marginally significant for PAD alone (0.77 (0.58, 1.00); $p=0.05$), whereas results were similar but did not achieve statistical significance for arterial stiffness (0.68 (0.40, 1.16); $p=0.16$) in the fully-adjusted model. Results were similar when adjusting for abdominal obesity, as well as when reclassifying ABI as normal, intermediate-

risk (non-significant), PAD, and arterial stiffness (data not shown). None of the tested interactions with traditional CVD risk factors were deemed significant at the $p < 0.05$ level.

Discussion

In this study, we observed that consuming a healthier diet was associated with 21% lower likelihood of having high-risk inflammation and 26% lower likelihood of having high-risk ABI level, after controlling for the traditional cardiometabolic risk factors prevalent in this Hispanic/Latino population. These results support a growing body of evidence on the favorable role of healthy diets in lowering the odds of non-traditional risk factors of CVD. This is important because these emerging factors have been associated with stronger CVD-risk prediction, thus identifying primordial and primary behaviors that could curb these risk factors may help further lower CVD risk.

The role of diet quality on the inflammation marker CRP has been well established in multiple observational studies, with most studies suggesting that more favorable diets may be associated with less inflammation^{8–18}. The magnitude of this association in our study was similar to the results from another study in a cohort of white and black adults that used AHEI as a measure of diet quality^{12, 13}. On the other hand, our results were directionally similar but weaker than the results from a cohort of predominantly white nurses that also used AHEI¹⁰. Both prior studies adjusted only for BMI, rather than for the more complete set of cardiometabolic factors that were controlled in the current study. We noticed that the AHEI-CRP association was slightly attenuated when we adjusted for all four cardiometabolic risk factors. The few studies that have adjusted for multiple traditional risk factors have detected significant associations between diet quality and CRP^{11, 17}, as we did. Moreover, Chrysoshoou *et al* observed that the association remained significant for individuals with hypertension, diabetes, hypercholesterolemia, or those who smoked, when analyzed separately¹¹. We did not detect a significant interaction with smoking, neither with sex as previously reported²¹. Nonetheless, results suggest that the odds of inflammation, as measured by CRP, can be lower in people with or without diabetes or obesity, who follow a healthier diet. Furthermore, our results suggest that the benefits of a healthy diet on CRP may be even more pronounced for those with diabetes, especially if untreated, with obesity, or older than 45y.

Importantly, this is one of the few reports of an association of diet quality and inflammation among Hispanics/Latinos. Studies among Puerto Rican middle-aged adults failed to detect an association between a diet score that reflected adherence to American Heart Association (AHA) dietary recommendations and CRP^{20, 46}. It is possible that the food components or the emphasis given to each food in AHEI influence inflammation more than those on the AHA guidelines. For example, fruit and vegetable intake has been strongly related to lower CRP⁴⁷. While AHEI includes each of these food categories separately in their scores, the AHA diet score combined them. Similarly, AHEI, but not the AHA score, includes a separate category for nuts and legumes, which have been associated to lower inflammation⁴⁸.

Our results on the association of diet quality and ABI (combining PAD and arterial stiffness) were significant after adjusting for traditional cardiometabolic risk factors. Separate analysis of each condition showed a marginal association with PAD and non-significant association with arterial stiffness. Inverse significant associations between diet quality (measured by adherence to the 2010 Dietary Guidelines) and arterial stiffness were shown in the Framingham Heart Study; however this study measured arterial stiffness using a different method than ours (tonometry) ²². Studies analyzing PAD separately are inconsistent. While the PREDIMED trial showed that a Mediterranean diet supplemented with either nuts or olive oil reduced the incidence of PAD compared to a low-fat diet ²⁴, the Multi-Ethnic Study of Atherosclerosis failed to detect any association in cross-sectional analysis ^{15, 25}. Differences in diet quality scores and analytic design may explain these inconsistent results.

Single nutrients and foods have been connected to ABI-related outcomes, suggesting possible mechanisms for the observed associations. For example, after adjusting for hypertension, diabetes, and coronary artery disease, Lane *et al* found that vitamins A, B₆, C, and E, fiber, folate, and omega-3 fatty acids were associated with lower odds of PAD in NHANES ²⁹. Naqvi *et al* subsequently repeated a similar analysis in NHANES and showed that further adjustments for physical activity and energy intake produced null results for these same nutrients ⁴⁹. We adjusted for similar covariates and the significant association with high-risk ABI was sustained, but we observed marginal associations between AHEI and PAD, and null associations with arterial stiffness; however, we may have been limited by the low sample size for these outcomes. Some other specific foods and nutrients that have been related to lower PAD or arterial stiffness in other cross-sectional studies and smaller trials include nuts ^{50, 51}; legumes ⁵²; blueberries ⁵³; linolenic acid and saturated fatty acids (inversely) ⁵⁴. Omega-3 fatty acids do not seem to confer any benefits on ABI-related markers ^{54, 55}. Some of these foods and nutrients are comprised by AHEI. Our results suggest that an overall healthy diet quality encompassing multiple foods and nutrients operating synergistically may be as important in sustaining beneficial levels of this emerging CVD risk factor as consuming these single foods. We did not detect significant interactions with any of the traditional CVD risk factors; however small sample size may have limited the power for this.

Our study is limited by its cross-sectional design, which cannot help establish causality. Yet, the consistency of associations in the literature (particularly for CRP), and the adjustment for prevalent cardiometabolic conditions, helps strengthen the notion that diet quality may contribute to non-traditional CVD risk factors. We were also limited by the small number of cases categorized with PAD or arterial stiffness. Combining them as a general high-risk ABI category helped increase statistical power and we were able to detect a significant association with this outcome. Measures of odds ratios may slightly overestimate the strength of association when the outcome is frequent, such as high-risk CRP among those with cardiometabolic factors. Still, estimating the OR can appropriately capture differences in exposure between groups. We used only one diet quality score, and it is possible that other foods or nutrients, or the weight given to these in other diet quality scores, may impact CRP and ABI differently. Finally, while we used dichotomous definitions for the traditional risk factors as previously suggested by other risk assessment studies^{1, 7}, it may be useful for future studies to consider duration and severity of these conditions. We focused our analysis

on two emerging risk factors, but other potential factors correlated to arterial stiffness, such as chronic kidney disease, should be explored.

A major strength of this study is that we appropriately adjusted for traditional cardiometabolic factors to control for confounding due to the co-occurrence of traditional and non-traditional markers, and to better determine the independent association of AHEI with non-traditional markers. The several sensitivity analyses conducted also helped confirm the robustness of our results. Finally, testing for interaction with traditional risk factors showed the importance of a healthy diet among those with major CVD risk factors (diabetes, obesity, and older age).

In conclusion, higher diet quality is associated with lower inflammation and ABI among Hispanics/Latinos, independently from traditional cardiometabolic risk factors. Promoting a healthy overall diet is a clinically-relevant primordial behavioral strategy that may help lower CVD-risk related to emerging factors in a population that already presents high prevalence of cardiometabolic markers.

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Highlights

- Higher diet quality is associated with less adverse ankle brachial index
- Higher diet quality is associated with lower inflammation (C-reactive protein)
- Better diet quality may help lower CRP for those with diabetes, obesity, age=45–75y
- Associations were independent from pre-existing traditional cardiometabolic factors
- A healthy overall diet may help lower emerging CVD-risk factors among Hispanics

Figure 1. Odds ratio (95% confidence interval) for high C-reactive protein for each 10-unit increase in AHEI in HCHS/SOL 2008–2011

Reported as Odds Ratio (95% CI) with reference category as normal CRP (<1.0mg/L when contrasting moderate-risk, elevated-risk, or acute concentrations; or <3.0mg/L when contrasting combined high-risk CRP concentrations). Model 1 was adjusted for age, sex, background, family history of CHD, center, energy intake, household income, marital status, years of living in the US, health insurance, physical activity, cigarettes packs per year, use of nonsteroidal anti-inflammatory drugs. Model 2 was additionally adjusted for diabetes, hypertension, hypercholesterolemia, obesity. n=14,623.

Abbreviations: AHEI, alternate healthy eating index; CRP, C-reactive protein.

Figure 2. Odds ratio (95% confidence interval) for high-risk ankle brachial index, peripheral artery disease, or arterial stiffness for each 10-unit increase in AHEI among adults ≥ 45 y in HCHS/SOL 2008–2011

Figure 2: Reported as Odds Ratio (95% CI) with reference category as normal ABI (>0.90 or <1.40 mg/L). Model 1 was adjusted for age, sex, background, family history of CHD, center, energy intake, household income, marital status, years of living in the US, health insurance, physical activity, cigarettes packs per year, use of nonsteroidal antiinflammatory drugs. Model 2 was additionally adjusted for diabetes, hypertension, hypercholesterolemia, obesity. ABI was measured among participants aged ≥ 45 y. $n=7,892$. Abbreviations: AHEI, alternative healthy eating index; ABI, Ankle-Brachial Index; PAD, peripheral artery disease.

Characteristics of adults without previously-diagnosed CVD by low-risk and high-risk levels of emerging CVD risk factors, HCHS/SOL 2008–2011^a**Table 1**

		Inflammation (n=14,623)		Ankle Brachial Index (n=7,892) ^e			
		Low- or moderate-risk CRP	High-risk CRP	Low-risk ABI	High-risk ABI (PAD, arterial stiffness) ^f	PAD ^f	Arterial Stiffness ^f
n, %		9,217; 65.2	5,406; 34.8	7426; 93.7	466; 6.3	328; 4.2	138; 2.1
Age, mean (y)		39.0 (0.26)	42.3 (0.36) [*]	55.3 (0.16)	59.3 (0.66) [*]	58.9 (0.71)	60.0 (1.25) [*]
Females, %		46.0	65.7 [*]	55.2	57.7	69.9	32.8 [*]
Years living in the US, (y)		7.1 (0.55)	7.6 (0.62) [*]	9.4 (2.6)	8.7 (3.0) [*]	8.6 (3.0)	9.1 (3.6) [*]
Marital status							
	Single	36.7	31.9 [*]	17.4	18.8 [*]	21.3	14.0 [*]
	Married/Partner	49.5	50.1	55.1	44.4	39.6	54.2
	Divorced/Widowed	13.9	18.0	27.5	36.8	39.2	31.9
Income							
	<\$30,000	57.8	65.0 [*]	63.0	66.3	65.5	67.9
	\$30,000	35.4	29.2	31.0	25.2	23.8	28.0
	Missing	6.8	5.8	6.1	8.5	10.6	4.1
Family history of CHD, %		25.5	29.8 [*]	39.7	44.9	49.7	35.1 [*]
Health insurance, %		48.0	49.6	54.0	58.6	53.0	70.4 [*]
Use of NSAID, %		13.4	18.6 [*]	19.4	16.2	13.8	21.2
Cigarettes packs per year							
	>0 and <10 years	23.4	22.2 [*]	19.2	17.3 [*]	20.6	10.5 [*]
	10 years	12.3	15.2	25.2	32.7	37.8	22.0
Physical activity ^b							
	High	15.1	11.3 [*]	8.9	5.4 [*]	4.0	8.1 [*]
	Moderate	47.0	41.6	42.3	35.6	36.3	34.2
	Low	37.8	47.1	48.8	59.0	59.6	57.7

		Inflammation (n=14,623)		Ankle Brachial Index (n=7,892) ^e			
		Low- or moderate-risk CRP	High-risk CRP	Low-risk ABI	High-risk ABI (PAD, arterial stiffness) ^f	PAD ^f	Arterial Stiffness ^f
Center							
	Bronx	26.4	29.2 *	24.8	26.6 *	23.8	32.3 *
	Chicago	16.6	15.5	13.5	10.7	6.6	19.1
	Miami	28.2	31.5	35.8	47.0	54.6	31.6
	San Diego	28.9	23.7	25.9	15.7	15.0	17.0
Ethnic Background							
	Mexican	41.1	34.8 *	33.5	22.0 *	18.3	29.6 *
	Puerto Rican	13.7	17.3	17.1	18.1	17.5	19.4
	Cuban	18.7	21.7	25.8	38.0	42.4	29.2
	Dominican	9.4	10.7	8.9	6.6	6.2	7.4
	Central American	7.8	7.1	6.7	6.7	6.7	6.7
	South American	5.5	4.2	5.8	6.8	6.4	7.6
	Other/Mixed	3.9	4.2	2.2	1.8	2.5	0.24
AHEI score ^c							
	Mean (SD)	40.3 (0.29)	38.7 (0.32) *	41.3 (0.91)	39.5 (1.0) *	38.9 (1.0)	40.9 (1.2) *
Cardiometabolic risk factors ^d							
	Hypertension	18.1	28.2 *	42.0	61.8 *	64.6	56.1 *
	Hypercholesterolemia	37.1	48.0 *	52.1	61.3 *	60.0	64.0 *
	Diabetes	10.3	19.8 *	24.1	37.3 *	33.3	45.4 *
	Obesity	25.9	62.1 *	40.1	47.8 *	41.8	59.9 *

^a All analyses were weighted to adjust for sampling probability of selection and non-response. Except for age, all data is shown as percent or age-adjusted mean (41.1y for CRP and 55.6 for ABI) and 95% confidence interval. High-risk CRP was defined as >3.0 mg/L; high-risk ABI was defined as 0.90 or 1.40. High-risk ABI was further classified as PAD (ABI 0.90) or arterial stiffness (1.40). Participants were excluded if they had pre-existing coronary heart disease or cerebrovascular disease as defined by abnormalities detected in ECG report, or self-report of angina, heart attack, stroke, mini-stroke or transient ischemic attack, or procedure in the heart or to the arteries of the neck. Additionally, participants with self-reported PAD were excluded from the ABI subgroup analysis. Abbreviations: ABI, ankle brachial index; AHEI, alternate healthy eating index; CHD, coronary heart disease; CRP, C-reactive protein; NSAID, Nonsteroidal Anti-inflammatory Drugs; PAD, peripheral artery disease.

^qPhysical activity was assessed using the Global Physical Activity Questionnaire. Self-reported hours of sedentary behavior and activity at work, travel, and leisure were converted into METs (Metabolic Equivalent), and categorized as high, moderate, or low levels, as based on number of days spent doing physical activity at each designated intensity level.

^cAHEI is a score comprised of 11 food and nutrient components, ranging from 0–110 (unhealthiest to healthiest diet quality).

^dHypertension defined as blood pressure $\geq 140/90$ or use of medication. Hypercholesterolemia was defined as total cholesterol ≥ 240 mg/dL or LDL ≥ 160 mg/dL, or HDL < 40 mg/dL, or use of lipid-lowering medication. Diabetes was defined as fasting glucose ≥ 125 mg/dL, or post-oral glucose tolerance test > 199 mg/dL or A1C $\geq 6.5\%$, or self-reported diabetes, or use of high blood sugar/diabetes medications. Obesity was defined as BMI ≥ 30 kg/m².

^eABI was measured only among participants aged ≥ 45 y.

^fContrasted to low-risk ABI.

* $p < 0.05$.

Table 2

Odds ratio (95% confidence interval) for high C-reactive protein for each 10-unit increase in AHEI stratified by age category, diabetes status, and obesity status; HCHS/SOL, 2008–2011^a

		Low- or moderate-risk CRP (≤3mg/dL)	High-risk CRP (>3mg/L)	
			OR (95% CI)	p-value
Diabetes ^b	No	1.00	0.81 (0.71, 0.93)	0.003
	Yes	1.00	0.69 (0.53, 0.90)	0.006
Obesity ^c	No	1.00	0.87 (0.74, 1.02)	0.08
	Yes	1.00	0.69 (0.57, 0.83)	0.0001
Age	18–44y	1.00	0.87 (0.72, 1.05)	0.14
	45–74y	1.00	0.73 (0.63, 0.85)	0.0001

^aReported as Odds Ratio (95% CI) and *p*-value. Adjusted for age, sex, background, family history of CHD, center, energy intake, household income, marital status, years of living in the US, health insurance, physical activity, cigarettes packs per year, use of nonsteroidal anti-inflammatory drugs, diabetes (except when stratified for diabetes), hypertension, hypercholesterolemia, obesity (except when stratified for obesity). n=14,623. Abbreviations: AHEI, alternate healthy eating index; CRP, C-reactive protein.

^bDiabetes was defined as fasting glucose >125 mg/dL, or post-oral glucose tolerance test >199 mg/dL or A1C>6.5%, or self-reported diabetes, or use of high blood sugar/diabetes medications.

^cObesity was defined as BMI>30kg/m².