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Association between diet quality indices and arterial stiffness in youth with type 1 diabetes: SEARCH for Diabetes in Youth Nutrition Ancillary Study

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Abstract

Aims: We studied the association of three distinct diet quality indices and two measures of arterial stiffness in youth and young adults (YYA) aged 10 to 30 with T1D.

Methods: Cross-sectional (n=1,421) and longitudinal (n=520) analyses were conducted in T1D YYA participating in the SEARCH for Diabetes in Youth Study. The diet quality indices included the Dietary Approaches to Stop Hypertension (DASH) index, the Healthy Eating Index 2015 (HEI-2015), and a modified Mediterranean Diet Quality Index (mKIDMED). Arterial stiffness was measured with pulse wave velocity (PWV) and augmentation index (AIx) obtained using a SphygmoCor-Vx device and tonometer.

Results: Average diet quality was moderate to poor, with mean scores of 41 (DASH, range 0-80), 55 (HEI-2015, range 0-100), 3.7 (mKIDMED, range −3-12). None of the diet quality scores was associated with the central PWV or Aix, independent of demographic, clinical and lifestyle factors, body mass index and HbA1c. Longitudinal data yielded consistent findings with cross-sectional results.

Conclusions: This study suggests that diet quality may not function as an independent risk factor for arterial stiffening in YYA with T1D. These findings do not diminish the importance of consuming a quality diet for the management of diabetes, as demonstrated in previous work.

Keywords

diet quality; dietary pattern; arterial stiffness; diabetes; epidemiology; adolescents and young adults

1. Introduction

Individuals with diabetes are at greater risk of cardiovascular disease (CVD) compared to the general population (1,2). A multitude of factors are independently associated with increased CVD risk in persons with diabetes including poor glycemic control (3), dyslipidemia (4), hypertension (5), and central obesity (6). Moreover, there is longitudinal evidence that traditional CVD risk factors contribute to subclinical cardiovascular changes that may be detected well-before clinical manifestations occur (7,8). Increased arterial stiffness (AS), a subclinical indicator of cardiovascular dysfunction (9), is independently prognostic of CVD morbidity and mortality (10,11) and is worsened in youth with diabetes (12,13). Uncovering modifiable risk factors of AS would open up potentially new and earlier avenues for prevention of CVD in persons with diabetes.

In individuals without diabetes, evidence suggests an association between AS and diet patterns (14–17) as healthy dietary components including fruits, vegetables, dairy foods, and fish have been favorably associated with decreased AS (14,15). A limited number of small-scale studies have examined the relationship between AS and healthful dietary patterns, such as the Dietary Approaches to Stop Hypertension (DASH) diet, which is recommended by

national guidelines targeting cardiovascular risk management (18). A longitudinal study showed favorable effects of a low sodium DASH diet in sodium sensitive adults (19). Likewise, a randomized, crossover study resulted in a positive association of the DASH diet containing lean beef in adults (20) and augmentation index (AIx), a measure of systemic AS. Additionally, in a cross-sectional study, a Mediterranean-type diet was related to lower AIx in healthy children (21).

Evidence in populations with diabetes is scarce. One study of middle-aged adults largely comprised of persons with type 2 diabetes (T2D) reported low fat dairy and vegetable intakes were inversely associated with pulse wave velocity (PWV), a measure of central AS (22). In one of the few studies focusing on adolescents with type 1 diabetes (T1D), a dietary pattern high in sugar-sweetened and diet beverages, eggs, potatoes and fatty meats was positively associated with AIx but not PWV (23). The SEARCH for Diabetes in Youth Nutrition Ancillary Study (SNAS) provides a unique opportunity because the availability several dietary indices reflective of high-quality diets, including the DASH score, a Mediterranean diet quality measure, the mKIDMED index and the Healthy Eating Index (HEI) 2015 allow a robust estimation of diet quality within SNAS (24–28).

The purpose of this study was to examine the association between diet quality indices and two measures of AS, PWV and Aix, in youth and young adults (YYA) with T1D using cross-sectional and longitudinal data. We hypothesized that greater diet quality as measured by higher DASH, mKIDMED, and HEI-2015 score would be associated with lower PWV and lower AIx.

2. Material and Methods

2.1 Study Design

The SEARCH for Diabetes in Youth study is a multicenter study, that began in 2001, of physician-diagnosed diabetes mellitus in youth younger than age 20 at diagnosis. SEARCH identified cases that were prevalent in 2001 and additionally cases that were incident in 2002-2005 (SEARCH Phase 1) (29,30). The design of SEARCH is shown in Supplementary Figure 1.

In SEARCH Phase 2, the surveillance effort included incident diabetes cases in 2006-2009. Additionally, participants enrolled in Phase 1 were invited to 12-, 24- and 60-month follow-up visits during Phase 2. In SEARCH Phase 3 (funding period 2010-2015), the surveillance effort included incident diabetes diagnosed between 2010 and 2014. Additionally, SEARCH participants from SEARCH 1 and 2 aged 10 years or older with at least 5 years of diabetes duration were invited for another study visit consisting of questionnaires including a food frequency questionnaire (FFQ) physical examinations, and laboratory measures. This group is referred to as the SEARCH Cohort Study and provides the basis of this manuscript. At each study visit, participants completed questionnaires, including a food frequency questionnaire (FFQ), physical examinations and a fasting blood draw were conducted. Participants were on average 17.7 years of age at the time of the cohort visit (age range 10 to 30 years). Data collection sites were located in South Carolina, Ohio, Colorado, Washington and California. The study was approved by and followed procedures in accordance with the

ethical standards of the respective local Institutional Review Boards. Parents of participants under age 18 provided written informed consent while participants provided assent; all participants aged 18 years or older provided written informed consent.

2.2 Diet assessment and dietary indices

Dietary intake was assessed at the baseline (first) visit for each SEARCH participant and at the 12-, 24- month visits and at the cohort visit using a FFQ (31). Dietary assessment was not conducted on children under 10 years of age. These analyses use data from the cohort visit (1) for cross-sectional analyses because of the larger sample size and thus larger power, and (2) from up to four visits (including baseline and follow-up visits as available) for longitudinal assessment of diet. Thus, for the longitudinal analyses, the dietary exposure assessment preceded PWV and AIx outcome assessment.

In brief, the FFQ consisted of 85 food lines (i.e. questions about one or multiple foods or beverages being consumed) for which the participant indicated whether the item was consumed in the past week (“yes/no”) and if yes, how many days and the average portion size. Portion size was queried for each line item as a number or as “very small,” “small,” “medium” or “large” relative to pictures of food. A final open-ended question queried all other foods that a participant might want to report. The FFQ was primarily self-administered after staff instruction.

Food groups were created by either collapsing food lines based on their major components or by disaggregating composite foods into basic foods. The dietary intake data were analyzed using Nutrition Data System for Research (NDSR 2014) developed by the Nutrition Coordinating Center (NCC) at the University of Minnesota, Minneapolis, MN. The SEARCH FFQ has been validated in this sample and has been shown to have good relative validity compared to three 24 hour recalls for the majority of food groups and nutrients used in this analysis (32).

Three dietary indices were used to evaluate diet quality in this study; DASH, HEI-2015, and mKIDMED, described previously in detail (33). These indices were chosen because they represent a) adherence to an overall high-quality diet based on the Dietary Guidelines for Americans (HEI-2015), and b) two specific diets patterns that have been shown to be favorably associated with cardiovascular disease (DASH and Mediterranean Diet). All indices were coded based on food item, food group and nutrient data from the SEARCH FFQ. Adherence to the DASH diet was assessed with an index variable based on the algorithms developed by Gunther et al, resulting in an overall DASH adherence score that ranged from 0 to 80 (34). The HEI-2015 evaluates diet quality relative to the 2015-2020 Dietary Guidelines for Americans (26) and the score was calculated following the method described by the National Cancer Institute (35), resulting in an overall HEI-2015 that ranged from 0 to 100 (26,36). The mKIDMED score characterizes the degree to which dietary intake in children or adolescents resembles a Mediterranean diet (27,37–39). Items denoting a negative connotation with respect to a Mediterranean diet were assigned a value of -1, whereas those with a positive aspect were scored +1, yielding a total range of negative three (−3) to 12. Higher scores on all dietary indices indicate higher diet quality.

2.3 Arterial stiffness measures

At the SEARCH cohort visit assessments were conducted with the participants metabolically stable. Non-invasive measures of arterial stiffness including central PWV and AIx were ascertained using a SphygmoCor device and tonometer. Measurements were obtained at a stable room temperature after 10 minutes of rest. The details of the arterial stiffness measures are described elsewhere (26,40). Briefly, carotid-femoral PWV(along the trunk) measures the pulse transit time from the carotid artery to the femoral artery and is a measure of central arterial stiffness in a large, elastic artery and predicts future cardiovascular disease events and mortality (12). Three separate recordings were taken at each site, averaged and reported in m/sec. Higher PWV values indicate increased arterial stiffness. AIx is a measure of wave reflections influenced by central stiffness and also is associated with all-cause mortality in adults (41). Higher AIx also indicates stiffer vessels. All analyses of PWV included adjustment for mean arterial pressure and heart rate. AIx was adjusted for mean arterial pressure and height (40). About 5-6% of the T1D sample exhibited complications, including retinopathy and diabetic kidney disease at the time of this study (42).

2.4 Assessment of covariates

Covariates were identified a priori based on hypothesized associations informed by the literature. Race and ethnicity were obtained through self-report using standard Census questions (43) Current smoking, parental education and household income (as reported by parents of minors or as reported by the young adult participants) were based on self-report. Physical activity and hours of watching television were assessed using questions identical to or slightly modified from the Youth Risk Behavior Surveillance System (YRBSS) (44,45). Physical examinations at the study visits were conducted according to standardized protocols by trained and certified staff. Height and weight were measured to the nearest 0.1 cm and 0.1 kg, respectively. BMI was calculated as weight (kg)/height squared (m^2) and converted to a BMI z-score (46). Participants were asked to be fasting at the study visit. Blood specimens were processed at the site and shipped within 24 hours to the Northwest Lipid Metabolism and Diabetes Research Laboratories in Seattle, WA. Ale was measured by a dedicated ion exchange high performance liquid chromatography instrument (TOSOH, Bioscience, Inc., San Francisco, CA). Measurements of plasma cholesterol, triglycerides, and high-density lipoprotein (HDL) cholesterol were performed on a Hitachi 917 autoanalyzer (Boehringer Mannheim Diagnostics, Indianapolis, IN). LDL cholesterol was calculated by the Friedewald equation for individuals with triglyceride concentration <400 mg/dL (4.52 mmol/L) and by the BetaQuantification procedure for those with triglyceride concentration ≥400 mg/dL (47).

2.5 Statistical analyses

In the current analysis, we used participants in the SEARCH cohort study who had developed incident diabetes in 2002-2006 or 2008. Supplementary Figure 2 illustrates our inclusion and exclusion criteria. We limited the analysis to individuals with T1D (n=2,713) age 10 or older (n=1,729) at the time of their cohort study visit because dietary intake was not collected in those younger than 10 years. We then excluded those with a missing FFQ (n=104), leaving 1,625 youth with dietary intake data for descriptive analyses. Among those

youth, 86 had no arterial stiffness data, and a remaining 118 had missing data for both outcomes (PWV and Aix) or for mean arterial pressure (a covariate included in all models). Thus, the cohort analysis is based on 1,421 YYA. Additionally, 520 of these participants had at least one previous FFQ, so they are included in an analysis of the association of longitudinally-assessed diet with outcomes.

Statistical analyses were performed using SAS software (version 9.2; SAS Institute, Cary, NC); statistical significance was concluded at $p<0.05$ except where otherwise stated. Descriptive statistics were computed for all exposures, outcomes, and adjustment variables. We examined cross-sectional associations between FFQ-based diet indices (DASH, mKIDMED, and HEI-2015) and measures of arterial stiffness (PWV and AIx) at the cohort visit. PWV was log transformed to improve normality because skewed distributions. We fit a series of multiple regression models for each exposure/outcome pair, with adjustment as follows. For PWV, model 1 was adjusted for mean arterial pressure and heart rate. For AIx, model 1 was adjusted for mean arterial pressure and height. For all outcomes, model 2 adjusted for model 1 covariates, FFQ-derived daily energy intake in kilocalories, demographic variables (age, sex, race/ethnicity, maximum parental education, income, and clinical site), and diabetes-related variables (diabetes duration and insulin regimen). Model 3 added lifestyle variables (smoking, physical activity, TV watching time) to model 2 and is the model considered the most salient. Additionally, we created several more complex models, all based on Model 3 plus add-ons, to consider the inclusions of specific other covariates, whose role in the model may however be considered controversial. Thus, Model 4 was based on Model 3 plus BMI z-score, Model 5 added HbA1c (%), Model 6 added BMI z-score and HbA1c(%), and Model 7 added BMI z-score, HbA1c and LDL/HDL ratio.

Additionally, to assess how long-term diet is associated with arterial stiffness, we constructed a longitudinally-assessed summary of each diet quality index (i.e., a time-weighted average over all available assessments of diet quality) for the participants with multiple measurements of diet quality leading up to the cohort visit. We used a stepwise approximation, assuming diet quality remained constant for half the time interval before and half the time interval after the visit an FFQ was completed. We fit multiple linear regression models for each outcome using each of the longitudinally-assessed dietary indices, adjusting for covariates as in the cross-sectional models.

Finally, it was of interest to explore potential effect modification by BMI and HbA_{1c}, so the models with longitudinally-assessed diet were expanded to allow for an interaction between BMI z-score and diet quality (added to Model 4) and an interaction between HbA_{1c}(%) and diet quality (added to Model 5).

3. Results

Demographic and clinical characteristics of the study sample are shown in Table 1. The average age at the cohort visit was approximately 18 years, with an almost equal distribution between females and males. The majority of participants were non-Hispanic white, and the average diabetes duration was 95 months (~7.9 years). More than half of the sample reported participating in vigorous physical activity more than 2 days per week and watching more

than 2 hours per day of TV and 13% reported currently smoking. Mean levels of lipid levels were 96 mg/dl for LDL cholesterol, 56 mg/dl for HDL-cholesterol, and the LDL/HDL ratio was 1.85. Mean HbA1c was 9.1% (ranging from 4.9 to 16.4%). Mean levels were 5.45 m/sec for PWV (range 1.1-22.5) and −2.59 for AIx (range −34-40). Dietary quality was low-to-moderate, with a mean DASH score of 40.4 (out of 80), ranging from 14 to 67, mean HEI-2015 of 55 (out of 100), ranging from 25 to 88), mean mKIDMED of 3.5 (out of 12), ranging from −2 to 10.

As shown in Table 2, in linear regression models of the cross-sectional data (Model 1), none of the three indices of diet quality were associated with PWV. When adjusted for demographic and clinical variables (Model 2) or lifestyle variables (Model 3) these results remained unchanged. Additional adjustment for BMI-z-score alone (Model 4), HbA1c alone (Model 5) or both (Model 6) did not alter the findings, nor did consideration of all these covariates together plus LDL/HDL ratio. With respect to the diet quality – AIx association, the unadjusted and the demographic/clinical adjusted model (Model 2) indicated a significant inverse association of mKIDMED with AIx. Adjustment for lifestyle variables however explained this association and diet quality did not reach statistical significance in any of the subsequent models. A final series of analyses exploring a potential non-linear relationship of dietary quality indices with measures of arterial stiffness with BMI z-score and an interaction with levels of glycemic control did not offer new insights, and the associations remained null (data not shown; p *for interaction* > 0.10). Lastly, these analyses were also conducted considering PWV arm and PWV foot as outcome measures, but the results were identical (data not shown).

Table 3 presents findings using the prospective data with longitudinally-assessed diet on participants who had at least two FFQs. As in the cross-sectional analysis, the results of the regression models did not indicate the presence of any statistically significant associations between diet quality indices and measures of arterial stiffness.

4. Discussion

Contrary to our hypothesis, this study did not find evidence for an association between diet quality characterized by three different diet quality indices, DASH, mKIDMED and HEI-2015, and two measures of arterial stiffness, PWV and AIx. Some precedent for a lack of association between dietary quality and arterial stiffening comes from earlier findings from our research group. In this same study of T1D, no association was detected between dietary fiber intake and central AS measured by PWV and with Aix (48). This is plausible given that higher diet quality is typically associated with higher fiber intake. Furthermore, in a separate investigation of biomarkers of inflammation, a similar null association of diet quality was observed with three biomarkers of inflammation (C-reactive protein, fibrinogen and interleukin-6) (33). All of the above findings are consistent with a trial in a group of middle aged individuals with diabetes (16 T1D, 100 T2D) evaluating the effect of increased fruit, vegetable and dairy intake on AS which did not see an effect of the intervention on changes in PWV or AIx (49).

Moreover, the evidence that exists on a relationship of dietary quality influencing vascular stiffness is not particularly clear for those with T1D. One cross-sectional study of youth with T1D found an empirically derived, unhealthy dietary pattern to be associated with AIx but not with PWV or Brachial Distensibility (23). The adjusted models also did not control for BMI z-score or any other traditional risk factors, in part because of how those characteristics had already factored into the creation of the dietary pattern. Furthermore, a cross-sectional study of adults with T1D using a traditional nutritional substitution model concluded that under isocaloric conditions, substituting fat or protein for carbohydrates would be associated with decreased AS, as was substituting protein for alcohol. However, these findings contradict another study that empirically derived dietary patterns and found that a dietary pattern with full-fat cheese and eggs and another dietary pattern with sweets were associated with less AS (50). Lastly, a recent investigation in the SEARCH study on added sugar intake (which is inversely related to diet quality) in relation to central or peripheral PWV and AIx revealed no association in the entire sample of T1D cross-sectionally or with longitudinally assessed added sugar intake but did find evidence for significant effect-modification by BMI z-score such that a positive association of added sugars with PWV was seen in a group with a lower BMI z-score (51). This finding held in both cross-sectional analyses and in analyses with longitudinally measured diet and BMI. However, future analyses in SEARCH will allow a fully specified longitudinal analysis assessing the impact of changes in diet and their duration on changes on AS.

The aforementioned mixed findings thus raise the question of which causal pathways toward arterial stiffening may be influenced by the quality of dietary intake and what type of dietary intake characteristics, if any, may specifically play a role in any causal constellations. A significant number of distinct pathways exist and include overproduction of collagen and diminished synthesis of elastin, dysregulation of matrix metalloproteases, dysfunction of the endothelium (in part due to advanced glycation end-products creating cross-links in tissue proteins), dysfunctional neuroendocrine signaling, increased inflammation, hypertrophy of the arterial wall muscle due to increased blood pressure, increased autonomic tone, elevated glucose and insulin levels, and genetics (52). In persons with T1D, the elevated glucose levels likely play a dominant role in any causal constellation leading to AS, given that improvements in glycemic control have been consistently associated with better vascular outcomes (53). This raises the question as to how much causal variation could hypothetically be attributed to other pathways and which of these in turn are associated with dietary intake.

Previous work showing strong associations of high-quality diet with outcomes has focused on (1) lipid levels and glycemic control including significant inverse cross-sectional associations of diet quality measured by DASH adherence with total cholesterol, LDL cholesterol and apolipoprotein B, (2) inverse cross-sectional and longitudinal associations of mKIDMED and total cholesterol and LDL cholesterol, and (3) positive associations of increased intake of sugar-sweetened beverages with total and LDL cholesterol (24,27,54–56). Given that only a minority of the pathways underlying the arterial stiffening process seem to be influenced by the role of lipoprotein cholesterols (52), the totality of evidence suggests that if diet quality is associated with cardiovascular disease in persons with T1D, the main causal route may be through pathways that are directly influenced by adverse lipid profiles and less so with other pathways leading to arterial stiffness. More evidence for this

hypothesis is provided by the aforementioned trial among adults with type 1 and type 2 diabetes that examined the impact of increased fruit, vegetable and dairy intake (which achieved a significantly increased quality of dietary intake) and interestingly demonstrated no impact on arterial stiffness while exhibiting a strong impact on a measure of subclinical atherosclerosis, leading to reduced measures of the common carotid artery intima media thickness (57).

There are several limitations to the present study. The dietary assessment relied on self-reported intake using an FFQ and while this instrument has shown good reliability and validity we cannot exclude the potential for error or systematic bias (32,58). While our analyses included both cross-sectional and longitudinally-assessed diet, we were not yet in a position to evaluate the association of dietary quality (and change therein) on change in AS measures. Our cross-sectional analyses were not able to assess temporal relationships, whereas the longitudinal analyses were somewhat limited by the restricted sample size. Furthermore, the longitudinal analyses relied on an average of 7.7 years of follow-up (minimum 2.1, maximum 12.6) which is less than ideal given that subclinical disease such as arterial stiffness tends to develop only after many years of exposure.

Among the strengths of this study is that it has one of the largest diverse samples of persons with T1D observed over time that has been carefully characterized with respect to dietary intake and health. Furthermore, we used three different measures of dietary quality rather than focusing on a single dietary index and our previous work established the ability of these indices to characterize dietary quality in T1D populations with a high level of validity (24,55,59,60). The level of dietary intake quality in our sample of T1D was generally very similar to that of samples of youth and young adults with and without diabetes in the US (61–63). The consistency observed across dietary quality indices enhances the credibility of the findings, similar to other studies (33,64). Lastly, this study evaluated both central AS with the PWV measurement as well as AIx which measures both peripheral and central stiffness and captures a part of arterial stiffness not captured by PWV.

In conclusion, this study did not find an association between three distinct diet quality measures and AS in YYA with T1D, but findings of this study do not diminish the importance of quality of dietary intake for glycemic control and lipoprotein levels, both important in the management of T1D (24,27,54–56). Moreover, there is substantial evidence that the quality of dietary intake is amenable to improvements through interventions among persons with T1D (57,63,65,66). Replication of these findings using longitudinal data focusing on changes in AS, which this study is currently collecting, will allow a more robust observational evaluation of this question. Nonetheless, our current study provides valuable information in a large, representative sample of individuals with T1D who may not have access to such interventions.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

1. Diet is associated with cardiovascular disease risk in youth with type 1 diabetes
2. Here, diet quality is measured with 3 different diet quality indices
3. Diet quality may not function as an independent risk factor for arterial stiffening
4. Cross-sectional and longitudinal analyses yield consistent results

Table 1.

Demographic and clinical characteristics of youth with type 1 diabetes age 10 years or older in the SEARCH Nutrition Ancillary Study (n=1,421)

Variable	Mean (SD) or N(%)
Age at corresponding visit, mean (SD)	17.7 (4.2)
DM duration, in months, mean (SD)	94.6 (22.9)
Female, N (%)	733 (51.6%)
Non-Hispanic White	1107 (77.9%)
Parental education	
Less than High School	51 (3.6%)
High School graduate	156 (11.1%)
Some college	440 (31.4%)
Bachelor's degree or more	756 (53.9%)
Income	
<$25,000	192 (13.6%)
$25-49,000	238 (16.8%)
$50-74,000	214 (15.1%)
$75,000+	539 (38.1%)
Don't know/Refused	231 (16.3%)
Insulin dose per kg	0.85 (0.39)
Kilocalories kcal	1693 (755)
Smoking	
Never	980 (70.3%)
Former	230 (16.5%)
Current	185 (13.3%)
Watch television 2 or more hours/day	750 (53.4%)
Vigorous physical activity >2 d/wk	820 (58.2%)
BMI z-score	0.62 (0.93)
HDL-cholesterol (mg/dL)	56 (14)
LDL-cholesterol (mg/dL)	96 (28)
Triglycerides (mg/dL)	93 (73)
LDL/HDL ratio	1.85 (0.77)
HbA1c %	9.1 (1.8)
Mean Arterial pressure	81.1 (9.07)
Heart rate	68.76 (11.45)
Height (m)	1.67 (0.12)
Exposure variables	
DASH score (range 0-80)	40.4 (8.7)
mKIDMED score (range −3 to 12)	3.64 (2.08)
HEI-2015 (range 0-100)	55.1 (11.2)
Longitudinally assessed DASH[1]	40.6 (6.5)

Variable	Mean (SD) or N(%)
Longitudinally assessed mKIDMED[1]	3.5 (1.5)
Longitudinally assessed HEI 2015[1]	54.0 (8.6)
Outcome variables	
PWV (carotid-femoral, m/sec)	5.45 (1.18)
AIx, %	−2.59 (10.48)

[1]Sample size: 520

DASH: Dietary Approaches to Smiddle Hypertension

HEI: Healthy Eating Index

mKIDMED: modified Mediterranean Diet index

Table 2.

Cross-sectional association of DASH, mKIDMED and HEI-2015 with arterial stiffness in youth with type 1 diabetes aged 10 years or older in the SEARCH Nutrition Ancillary Study (n=1,333 for PWV, n=1,137 for AIx)

	DASH			mKIDMED			HEI-2015		
	Beta	**SE**	**p**	**Beta**	**SE**	**p**	**Beta**	**SE**	**p**
PWV (carotid-femoral) m/sec (log transformed)									
Model1	0.00004	0.0006	0.95	0.0033	0.0025	0.19	0.0001	0.0005	0.82
Model2	0.0002	0.0006	0.70	−0.0015	0.0026	0.56	0.0001	0.0004	0.81
Model3	0.0003	0.0006	0.58	−0.0013	0.0027	0.63	0.0002	0.0005	0.69
Model4	0.0002	0.0006	0.73	−0.0022	0.0026	0.40	0.00006	0.0005	0.89
Model5	0.0003	0.0006	0.60	−0.0014	0.0027	0.59	0.0002	0.0005	0.73
Model6	0.0002	0.0006	0.73	−0.0022	0.0026	0.40	0.00006	0.0005	0.89
Model7	0.0001	0.0006	0.84	−0.0023	0.0027	0.39	−.00002	0.0005	0.97
AIx, %									
Model1	−.0383	0.0324	0.24	−.2689	0.1358	0.0479	−.0349	0.0252	0.17
Model2	−.0135	0.0329	0.68	−.2791	0.1490	0.06	−.0195	0.0256	0.45
Model3	0.0073	0.0338	0.83	−.1980	0.1532	0.20	−.0029	0.0263	0.91
Model4	0.0096	0.0339	0.78	−.1857	0.1538	0.23	−.0013	0.0264	0.96
Model5	0.0111	0.0339	0.74	−.1820	0.1538	0.24	0.0002	0.0264	0.99
Model6	0.0131	0.0340	0.70	−.1714	0.1543	0.27	0.0015	0.0265	0.95
Model7	0.0116	0.0342	0.73	−.1587	0.1551	0.31	0.0037	0.0267	0.89

DASH: Dietary Approaches to Smiddle Hypertension

AIx: Augmentation Index

HEI: Healthy Eating Index

mKIDMED: modified Mediterranean Diet index

PWV: Pulse wave velocity

Model 1: Heart rate, mean arterial pressure for PWV; Height, mean arterial pressure for AIx

Model 2: Model 1 plus age, sex, race, parental education, income, site, disease duration, insulin regimen, and kcal

Model 3: Model 2 plus smoking, vigorous physical activity, TV watching time

Model 4: Model 3 plus BMI z-score

Model 5: Model 3 plus HbA1c

Model 6: Model 3 plus BMI z-score and HbA1c

Model 7: Model 3 plus BMI z-score, HbA1c and LDL/HDL ratio

Table 3.

Association of longitudinally assessed diet characterized by DASH, mKIDMED and HEI-2015 with arterial stiffness in youth with type 1 diabetes aged 10 years or older in the SEARCH Nutrition Ancillary Study (n=486 for PWV, n=433 for AIx)

	DASH			mKIDMED			HEI-2015		
	Beta	**SE**	**p**	**Beta**	**SE**	**p**	**Beta**	**SE**	**p**
PWV (carotid-femoral) m/sec									
Model1	−.0003	0.0014	0.83	0.0004	0.0059	0.95	0.001	0.0010	0.26
Model2	0.0002	0.0014	0.90	−.0030	0.0061	0.63	0.001	0.0010	0.29
Model3	0.0009	0.0015	0.55	−.0007	0.0063	0.91	0.002	0.0011	0.15
Model4	0.0007	0.0014	0.62	−.0024	0.0062	0.70	0.001	0.0011	0.27
Model5	0.0009	0.0015	0.55	−.0006	0.0063	0.93	0.002	0.0011	0.14
Model6	0.0008	0.0014	0.60	−.0020	0.0062	0.74	0.001	0.0011	0.22
Model7	0.0004	0.0015	0.79	−.0025	0.0064	0.70	0.001	0.0011	0.23
AIx, %									
Model1	−.0527	0.0718	0.460	−.1027	0.3097	0.74	0.0299	0.05324777	0.57
Model2	0.0079	0.0753	0.92	−.0174	0.3287	0.96	0.0412	0.05611368	0.46
Model3	0.0720	0.0777	0.35	0.2209	0.3364	0.51	0.0787	0.05699901	0.17
Model4	0.0734	0.0779	0.35	0.2296	0.3384	0.50	0.0806	0.05734777	0.16
Model5	0.0764	0.0778	0.33	0.2465	0.3373	0.47	0.0863	0.05737150	0.13
Model6	0.0772	0.0780	0.32	0.2514	0.3392	0.46	0.0874	0.05766340	0.13
Model7	0.1021	0.0787	0.20	0.3196	0.3454	0.34	0.0989	0.05814964	0.09

DASH: Dietary Approaches to Smiddle Hypertension

AIx: Augmentation Index

HEI: Healthy Eating Index

mKIDMED: modified Mediterranean Diet index

PWV: Pulse wave velocity

Model 1: Heart rate, mean arterial pressure for PWV; Height, mean arterial pressure for AIx

Model 2: Model 1 plus age, sex, race, parental education, income, site, disease duration, insulin regimen, and kcal

Model 3: Model 2 plus smoking, vigorous physical activity, TV watching time

Model 4: Model 3 plus BMI z-score

Model 5: Model 3 plus HbA1c

Model 6: Model 3 plus BMI z-score and HbA1c

Model 7: Model 3 plus BMI z-score, HbA1c and LDL/HDL ratio