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# Associations Between Adherence to the Dietary Approaches to Stop Hypertension (DASH) diet and Six Glucose Homeostasis Traits in the Microbiome and Insulin Longitudinal Evaluation Study (MILES)

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## Abstract

**Background and Aims:** The DASH diet conveys protection against type 2 diabetes mellitus (T2D) Via plant-based and non-plant-based recommendations. Research has not identified which glucose homeostasis pathways are improved. We examined associations between adherence to a DASH diet and six glucose homeostasis traits, probing whether associations could be attributed to the plant-based (DASH-P) and/or non-plant based (DASH-NP) components.

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AUTHORSHIP

G.R, A.C.W. and M.O.G. made substantial contributions to conception and design, data acquisition, analysis, and interpretation. G.R. and A.C.W drafted the article and all authors provided critical revisions with important intellectual content. All authors gave final approval of the version to be published. A.C.W gives agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.



CONFLICTS OF INTEERST

None.

**Methods:** We included data from 295 adults without T2D (age 59.3±9.00 years; 63.46% non-Hispanic White and 36.54% African American, self-reported race ancestry) participating in the Microbiome and Insulin Longitudinal Evaluation Study (MILES). An oral glucose tolerance test (OGTT) yielded fasting plasma glucose, insulin, C-peptide, and insulin secretion, sensitivity, and disposition index. Habitual dietary intake was assessed by food frequency questionnaire (FFQ). Associations between DASH components and glucose homeostasis traits were examined, controlling for demographics, body mass index (BMI), physical activity, and energy intake. For significant associations, the models were repeated with scores for DASH-P and DASH-NP as predictors in the same model.

**Results:** DASH and DASH-P scores were inversely associated with fasting plasma glucose (DASH:$\beta$=−0.036±0.012,$P$=0.005;DASH-P: $\beta$=−0.04±0.017,$P$=0.002), and positively associated with insulin sensitivity (DASH:$\beta$=0.022±0.012,$P$=.042;DASH-P:=0.036±0.015,$P$=0.014). The DASH score was also associated with disposition index ($\beta$=0.026±0.013,$P$=0.038), but this association did not reach significance with DASH-P ($\beta$=0.035±0.018,$P$=0.051). No associations were observed with DASH-NP score (all $P$>.05).

**Conclusions:** DASH diet is associated with improvement in specific glucose homeostasis traits, likely arising from increased plant-based foods. Such research may help tailor future dietary advice to specific metabolic risk, and to food groups most effective at improving these.

### Keywords

DASH diet; plant-based; Type 2 diabetes; glucose homeostasis; oral glucose tolerance test

## Introduction

Low dietary quality is a major modifiable risk factor for type 2 diabetes (T2D). The Dietary Approaches to Stop Hypertension, or DASH diet, encourages the consumption of vegetables, fruits, and whole grains, and seeks to reduce the intake of red meat, dairy, sugar-sweetened beverages (SSBs), and sweets. In addition to beneficial effects on blood pressure[1-3], adoption of, and adherence to, a DASH dietary pattern has been shown to improve glycemic control[4] and has been associated with a reduced risk of T2D[5]. Although the primary diagnostic marker of T2D is fasting hyperglycemia[6], the earliest prognostic perturbation is reduced insulin sensitivity (i.e., increased insulin resistance), which predicts the development of T2D in normoglycemic individuals more than a decade before clinical diagnosis in most cases[7]. Increased insulin secretion follows, predicting the development of T2D approximately three to five years before onset[7, 8]. If these physiologic changes fail to compensate for reduced insulin sensitivity, hyperglycemia will arise. Both elevated insulin secretion[9] and hyperinsulinemia[10] can predict dysglycemia independent of insulin sensitivity. Therefore, to effectively tailor preventive advice to individuals' metabolic dysfunction, risk factors for T2D should be examined for associations with multiple glucose homeostasis traits independently, each indicating distinct (but correlated) aspects of dysfunction.

Data from randomized controlled trials (RCTs) has shown that adoption of a DASH diet improves fasting insulin levels, but does not improve fasting glucose levels or

HOMA-IR; a measure of insulin resistance[4]. Observational studies typically assess habitual diet, and thereby may provide insights into the effects of long-term adherence to a DASH diet. Data from such studies not only show a direct association between a DASH diet and reduced fasting insulin[11], but also suggest that the DASH diet may reduce insulin resistance[11-13]. While RCTs have typically relied on measures of fasting insulin and/or glucose to estimate HOMA-IR, the PREMIER trial, which used frequently-sampled intravenous glucose tolerance testing (FSIGT) to more directly measure insulin sensitivity[14, 15], reported improvements in insulin sensitivity following adoption of the DASH diet.[16] Further investigation is needed to determine which components of glucose and insulin homeostasis improve with adherence to a DASH diet and account for the associated protection from T2D.

Recent meta-analyses have reported inverse associations between plant-based diets and incident T2D.[17, 18] Similarly, multiple randomized controlled trials and cohort studies have reported consistent associations between plant-based diets and improved glycemic control.[19-22] Notably, data attributing T2D risk reduction to the non-plant based components of DASH is less consistent. For example, while recent meta-analyses have noted a significant association between reduced red meat consumption and T2D risk reduction, [23-25] one study was susceptible to residual confounding[23], while another attributed protection to the consequent increased consumption of plant-based foods[24], which others did not control for[25]. Similarly, while SSB intake is associated with increased diabetes risk,[26] the authors noted residual confounding;[26] the extent to which this relationship is causal remains controversial[27]. Importantly, it has been noted that associations between individual food groups and disease risk are strongly confounded by overall dietary pattern, [28] and we are not aware of any studies which have sought to discriminate between the roles of plant- and non-plant based food intake in T2D risk within the context of the DASH diet specifically.

The goals of the current analysis were to examine the associations between the DASH dietary recommendations and six traits related to glucose homeostasis: fasting plasma glucose, insulin, and C-peptide, as well as insulin secretion, insulin sensitivity, and disposition index from an OGTT, using data from a multi-racial sample of older US adults. We also aimed to determine whether any associations could be attributed to the plant *vs* the non-plant based components of a DASH diet.

## METHODS

### Participants

Recruitment methods for the Microbiome and Insulin Longitudinal Evaluation Study (MILES) have been described in detail elsewhere[29]. The cohort consists of 353 participants (129 African American and 224 non-Hispanic White ancestry participants by self-report) aged 40-80 years living in North Carolina. Exclusion criteria at the time of enrollment included (1) severe illness (e.g., actively treated cancer), (2) use of medication known to alter the microbiome (e.g., antibiotics, metformin and proton pump inhibitors[30, 31]) within the prior month, (3) current use of oral steroids, (4) inflammatory bowel disease, (5) previous surgery for weight loss, (6) chronic constipation or diarrhea requiring

prescription therapy, (7) current pregnancy, (8) end stage renal disease, (9) self-reported heavy alcohol use, and (10) the presence of diabetes determined either by self-reported history or point of care fasting glucose ≥ 126 mg/dl. Eligible individuals were invited to participate in a baseline clinic visit, where they underwent an OGTT, completed food frequency questionnaires, and had anthropometric measures taken.

For our analyses, participants were excluded for missing dietary intake data and/or implausible energy intake, defined as <= 600 or ≥ 6000 kcals/day for men, and <= 400 ≥ 4000 kcals/day for women (N=20), and missing data on key covariates (N=44) yielding a final sample for analysis of N=295.

All subjects gave written informed consent prior to participation, and the study was approved by Institutional Review Boards at participating centers.

## Measures

**Glucose homeostasis traits:** Following an overnight fast, venous blood was drawn before, 30 minutes after, and 120 minutes after oral ingestion of a 75-gram glucose load. Fasting insulin, and C-peptide values were derived via immunoassay and glucose concentrations via the hexokinase G-6-PDH method. Insulin sensitivity/resistance was calculated using the Matsuda insulin sensitivity index (ISI), which correlates strongly with insulin sensitivity as quantified by euglycemic clamp (r=0.7-0.8[32-34]). Insulin secretion was calculated as the ratio of the area under the curve (AUC) for insulin from baseline to 30 minutes to the AUC for glucose for the same period ($AUC\text{-}Ins_{0\text{-}30}/AUC\text{-}Glu_{0\text{-}30}$), a value that correlates strongly (r= ~0.7[34]) with the first phase of insulin secretion from the intravenous glucose tolerance test. Disposition index (DI), which represents the value of insulin secretion accounting for the degree of compensation for insulin resistance, was calculated as the product of insulin sensitivity and insulin secretion ($ISI \times AUC\text{-}Ins_{30}/AUC\text{-}Glu_{30}$),.

In sensitivity analyses, dysglycemia was defined as the presence of fasting or 2-hour glucose levels in the prediabetic or diabetic range. Though diabetes at baseline was an exclusionary criterion, OGTT performed during the study found that 25 individuals had 2-h glucose levels of 11.1 mmol/l or greater, and three individuals had fasting glucose levels slightly greater than 7.0 mmol/l. These 28 participants (8%) were classified as having diabetes. None of these individuals were taking antidiabetic medication. An additional 136 participants (40%) were classified as having prediabetes based on impaired fasting glucose (IFG) (5.6–6.9 mmol/l, 73 individuals; 22%) or impaired glucose tolerance (IGT) (2-h glucose 7.8–11.0 mmol/L, 27 individuals) or both IFG and IGT (36 individuals; 11%).

**Dietary intake:** Habitual dietary intake over the past year was assessed using an FFQ. Participants completed the FFQ electronically with study staff available to answer questions. Participants were asked to record the frequency of consumption and the usual portion size of 124 food and drink items using the most recent version of the Diet History Questionnaire (DHQ) at the time of the study's inception: the DHQ II. The DHQ is developed by the National Cancer Institute[35]. Frequency was assessed via 11 options, which ranged from "never" to "≥2 times/day" for foods, and "rare or never" to "≥6 times/day" for beverages.

Portion size options differed by food type (e.g., number of slices for bread, number of tablespoons for cream cheese). Participants were also asked "modifier questions" to clarify aspects of certain foods that can be key drivers of overall dietary pattern (e.g., fat content of bacon, whole grain content of bread) and about their use of dietary supplements. At the time of the baseline visit, the most recent DHQ version was used.

Diet*Calc software was used to generate habitual intake of 176 micro- and macro-nutrients and 124 foods and beverages, based on the USDA's MyPyramid Equivalents Database and Food Patterns Equivalents Database. An overall DASH diet score was calculated as the sum of quintile scores for the following 8 components: (1) fruit and fruit juice, (2) vegetables, (3) whole grains, (4) nuts and legumes, (5) low-fat dairy, (6) sodium (reverse scored), (7) red and processed meat (reverse scored), and (8) SSBs (reverse scored). Scores were separated into quintiles for components 1-4 (fruits and fruit juice, vegetables, whole grains, and nuts and legumes). Although we did not ask if participants were attempting to adhere to a specific dietary pattern, as these scores measure how closely habitual diet reflects the DASH recommendations, scores were summed to reflect adherence to the plant-based components of DASH (DASH-P). Quintiles for components 5-8 (low-fat dairy, sodium, red and processed meat and SSBs) were summed to reflect adherence to the non-plant based (or other) components of DASH (DASH-NP).

**Demographic factors:** Self-reported age, sex, race, education, and income levels were recorded via electronic questionnaires with study staff available to answer questions.

**Anthropometric measures:** Height and weight were recorded by trained study staff using a stadiometer and a calibrated scale. Body mass index (BMI) was calculated as weight in kilograms (kg) divided by height in meters (m) squared ($kg/m^2$). For sensitivity analyses, overweight/obese was defined using the CDC criteria[36] as BMI $\geq$ 25 $kg/m^2$, while BMI < 25 $kg/m^2$ was considered normal weight.

**Physical Activity—**The physical activity survey employed in MILES is the well-validated instrument used in the Multi-Ethnic Study of Atherosclerosis (MESA)[37]. The MESA Typical Week Physical Activity Survey was based on the Cross-Cultural Activity Participation Study,[38] and implemented as has been previously described.[39] We analyzed total physical activity as the sum of light, moderate, and vigorous physical activity in metabolic equivalents (MET) minutes per week.

## ANALYSES

All analyses were conducted using the latest version of R software (version 4.0.5.)[40].

### Descriptive Statistics.

Demographic information, stratified by quintile of total DASH score, was calculated as mean (+/− standard deviation; SD) or total number (N) and percentage (%) in Table 1. Crude differences between the 1$^{st}$ and 5$^{th}$ DASH quintiles (i.e., without controlling for covariates) were conducted using *unpaired Wilcoxon rank sum for* continuous variables (and chi-squared tests of difference for categorical variables.

### Associations of adherence to a DASH diet with glucose homeostasis traits.

Glucose homeostasis traits were transformed to normality using an inverse normal transformation with a blom constant[41, 42]. Main effects of DASH score on each glucose homeostasis trait were examined using linear regression models, with each of the six glucose homeostasis traits as outcomes in separate models, and DASH score as predictor. All models controlled for age, sex, race, BMI, physical activity, education level, income level, and energy intake (kilocalories per day; kcal/day) and are presented in Table 2. Given that our six glucose homeostasis traits were interdependent and highly correlated (see Supplementary Table 1), we did not correct these models for multiple testing. We used the variance inflation factor (VIF) to measure the extent of collinearity among the covariates in our regression models, setting VIF values at or above 5 as the cutoff for concern for multicollinearity. Our analyses yielded VIF values 1.04-1.66, detecting no significant presence of collinearity among the covariates.

Sensitivity analyses were conducted by first stratifying by dysglycemia (normoglycemic *vs* dysglycemic), and subsequently by BMI (normal weight *vs* overweight/obese), each as defined above, and by additionally removing BMI as a covariate from the models on both the MILES cohort as a whole, and when stratified by glycemic status.

### Associations of DASH subscores (DASH-P and DASH-NP) with Glucose Homeostasis.

Where a significant association was found between DASH diet score and an individual glucose homeostasis trait, the linear regression models were re-run specifying DASH-P and DASH-NP scores as predictors in the same model.

Finally, the potential role of individual DASH food groups was probed by re-running any significant associations between glucose homeostasis and either DASH-P or DASH-NP using the food groups which were aggregated to form the respective DASH sub-score as collective predictors.

## RESULTS

Adherence to a DASH diet was not associated with age, race, or education level (all $P>.05$; Table 1). However, those with higher DASH scores were more likely to be female ($\chi^2=6.78$; df=1, $P=.009$; Table 1), have higher PA levels (W= 1274, $P<.001$; Table 1) and higher BMI (W= 2316, $P=.006$; Table 1).

### Associations of adherence to a DASH diet with glucose homeostasis traits.

In multivariable linear regression models for the whole MILES population, the DASH index was inversely associated with fasting plasma glucose ($\beta = -0.036\pm 0.012$, $P=0.005$; Table 2), and positively associated with both insulin sensitivity ($\beta = 0.022\pm0.012$, $P=.042$; Table 2), and disposition index ($\beta = 0.026\pm0.013$, $P=0.038$; Table 2). Adherence to a DASH diet was not associated with fasting insulin ($\beta = -0.01\pm0.01$, $P=0.287$; Table 2), fasting C-peptide ($\beta = -0.018\pm0.01$, $P=0.943$; Table 2), nor insulin secretion ($\beta = 0.0003\pm0.012$, $P=0.978$; Table 2).

When analyses were stratified by dysglycemia status, only the association between a DASH diet and fasting glucose in normoglycemic individuals remained ($\beta$ = −0.026±0.013, $P$=0.046; Table 2), while in BMI-stratified analyses, the DASH diet was not significantly associated with any of the glucose homeostasis traits (all $P$>.05; Table 2). Models that were run without BMI as a covariate showed the same pattern of results for the MILES population as a whole (Supplemental Table 2). Moreover, and without BMI as a covariate but when stratified by glycemic status, these models showed a highly similar pattern of results to models that controlled for BMI, although there was an incremental increase in the association of DASH diet with C-peptide, which now reached significance in participants with dysglycemia ($\beta$ = −0.035±0.016, $P$=0.035; Supplemental Table 2).

### Associations of DASH-P and DASH-NP with Glucose homeostasis.

Fasting plasma glucose and insulin sensitivity (separately) showed significant associations with DASH-P (fasting plasma glucose: $\beta$ = −0.054±0.017, $P$=0.002; insulin sensitivity: $\beta$ = 0.036±0.015, $P$=0.014; Table 3). Associations between these traits were significant for DASH score for nut intake (fasting plasma glucose: $\beta$ = −0.094±0.044, $P$=0.004; insulin sensitivity: $\beta$=−0.104±0.037, $P$=0.005; Table 4). The DASH score for fruit intake was significantly associated with insulin sensitivity ($\beta$=0.142±0.042, $P$=0.049; Table 4) and showed a trend association with fasting glucose ($\beta$=0.081±0.041, $P$=0.052; Table 4). No associations were found between these traits and DASHscore for vegetable or whole grain intake (all P>.05; Table 4).

None of the glucose homeostasis measures were significantly associated with DASH-NP (all $P$>.05; Table 3).

## DISCUSSION

Substantial evidence indicates that adherence to the DASH diet provides reduces the risk of incident T2D[2-5]. However, few studies have examined the specific alterations in glucose homeostasis than can benefit from adherence to this dietary pattern, and none have examined whether the plant-based vs. non-plant-based components of a DASH diet confer differential levels of protection. Using data from participants in the MILES study, who underwent a two-hour OGTT, we examined the association of DASH diet scores with six glucose homeostasis measures. Our analyses found that higher DASH diet scores were associated with improved fasting plasma glucose, insulin sensitivity, and disposition index, but were not associated with fasting insulin, fasting C-peptide, nor insulin secretion. When stratifying total DASH score into two separate component scores, one representing the sum of plant-based DASH components and one representing the sum of other (non-plant based) DASH components, our analyses revealed that only the former was associated with improved glucose homeostasis, implying that any protection from T2D conferred by the DASH diet arises from an increase in plant-based foods.

The metabolic dysfunction underlying T2D precedes the onset of clinical disease by over a decade.[7] The earliest detectable change is an increase in insulin resistance[7] (i.e., decreased insulin sensitivity). In our study, insulin sensitivity, as measured by the Matsuda index from an OGTT, was positively associated with dietary alignment with

DASH recommendations. These findings support data from observational studies which have shown that habitual adherence to a DASH diet pattern is associated with lower HOMA-IR[11-13], and data from the PREMIER RCT which found improvements in insulin sensitivity (measured directly by FSIGT)[16]. Together, these results raise the question of whether the failure of RCT data to show changes in insulin resistance with adoption of a DASH diet[2-4] is due to (1) the relatively short time of trials (range: 3-24 weeks); and /or (2) the use of HOMA-IR as a measure of insulin resistance. While HOMA-IR demonstrates moderate correlation with results from euglycemic clamp,[14] individual variability may be as high as 30%[14], and addition of HOMA-IR to the Matsuda index does not improve the classification of insulin resistance, whereas the reverse doses[15]. HOMA-IR may be a poor representation of insulin resistance[43]. Variability in HOMA-IR is largely driven by variability in fasting insulin, which itself reflects not only insulin resistance but also insulin secretion and insulin clearance [44]. Thus, while HOMA-IR can predict incident T2D,[45-47] it may be less sensitive to the small decrements in insulin sensitivity that mark the earliest stages of pre-diabetes, when compared to results of glucose tolerance tests.

Alongside decreased insulin sensitivity, the pre-diabetic state is marked by compensatory increases in both overall[48] and glucose-stimulated[49] insulin secretion. While we did not find the association between adherence to a DASH diet and fasting insulin to be significant, it is possible that our analysis lacked power. However, our results were in the same direction as data from both RCTs and observational studies, which corroborate that a DASH diet reduces fasting insulin.[2-4, 11-13]

We did observe a positive association between adherence to a DASH diet and disposition index. Disposition index measures insulin secretion in the context of insulin resistance. As such, our findings suggest that adherence to a DASH diet is associated with better $\beta$-cell function.[50, 51] Adequate $\beta$-cell stimulated insulin secretion in the early phases of insulin resistance can maintain glucose levels at elevated but subclinical levels[52] for several years[53], potentially preventing the onset of T2D. However, further deterioration of $\beta$-cell function is accompanied by marked increases in fasting plasma glucose, followed by decreases in acute phase glucose-stimulated insulin release, leading to higher post-prandial and fasting plasma glucose. What causes $\beta$-cell dysfunction in the pathogenesis of T2D is not well understood, but a contemporary hypothesis posits glucose toxicity as a major contributor[52, 54]. If this theory is correct and hyperglycemia contributes to $\beta$-cell dysfunction, our observation that adherence to a DASH diet is associated with both increased insulin sensitivity and improved markers of $\beta$-cell function makes our finding that adherence to a DASH diet is also associated with lower fasting plasma glucose expected. However, this is a novel finding that previous studies did not observe[2-4, 11-13] and which therefore also requires further confirmation.

Finally, we found similar associations between a score representing the plant-based components of DASH and both insulin sensitivity and lower fasting plasma glucose, but we did not observe this association with a similar score representing the non-plant-based components of DASH. In addition, the plant-based score showed a trend towards association with disposition index. As both diet scores were associated with insulin sensitivity, but not insulin secretion, a more modest association with disposition index was to be expected.

Previous data strongly support the role of plant-based diets in protecting against T2D[17, 18, 24], and improvements in glucose homeostasis,[19-22] while support for causal associations between T2D risk and red meat intake,[23-25] SSB consumption,[26, 27] and dairy intake is less consistent. In our study, and within the context of a DASH diet specifically, the plant-based components were only associated with glucose homeostasis parameters indicative of improved glucose control. These data add weight to growing support for a plant-based approach for preventing T2D, and suggest that the greatest reduction in T2D risk is achieved by increasing plant-based foods, not by increasing low-fat dairy, or reducing sodium, red meat, and SSB intake. Given the associations of these foods with other chronic diseases, focusing on plant-based foods may be a valuable initial strategy for those most at risk T2D, and can later be supplemented by other focused nutritional modifications.

While our study benefited from a multi-racial population and high-quality phenotyping afforded by OGTT, a few important limitations should be noted. The practical demands of delivering the OGTT limited our sample size and thereby limited study power, especially for stratified analyses, as well as examining the association of the individual plant-based DASH components with glucose homeostasis. Despite the use of a well-validated FFQ[55, 56], the usual limitations of self-reported nutritional data apply, such as a tendency to under-report intake, a bias that is only partially overcome by controlling for energy intake in our statistical models[57]. Finally, we cannot rule out possible residual confounding, and its cross-sectional design precluded causal inferences.

In conclusion, there is strong evidence that adherence to a DASH diet prevents incident T2D.[2-4, 11-13] The development of diabetes is marked by sequential, heterogenous aberrances in several markers of glucose homeostasis, and our findings suggest differential associations of each of these markers with a DASH diet, hinting at protection during early stages of pathology preceding clinical dysglycemia. Our analyses also show that increasing consumption of plant-based components of DASH is associated with better glucose homeostasis, but the same is not true with decreasing consumption of the non-plant based components of DASH. Taken together, our study provides hope that in the future, dietary advice may be tailored to the specific metabolic perturbations an individual is experiencing, and to the food groups most efficacious at ameliorating these imbalances, to better reduce the burden of T2D.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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## HIGHLIGHTS

- Type 2 Diabetes (T2D) is a heterogenous condition, with perturbations across multiple metabolic pathways that arise at different times during disease progression.
- While there is robust evidence that The Dietary Approaches to Stop Hypertension (DASH) diet conveys protection from type 2 diabetes (T2D) via lower fasting insulin levels, there are little data on whether DASH improves other markers of T2D risk.
- We examined cross-sectional associations between adherence to a DASH diet and six traits related to glucose homeostasis.
- DASH adherence was associated fasting plasma glucose, insulin sensitivity, and disposition index, but not fasting insulin nor insulin resistance - suggesting that DASH provides protection during the early, but not later, stages of the pathology preceding clinical dysglycemia.

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**Table 1:**

Mean +/− standard deviation (SD), or frequencies (+ percentage) for demographic, dietary and glucose homeostasis characteristics, by DASH diet score quintiles

| | DASH quintile (score range) | | | | | P |
|---|---|---|---|---|---|---|
| | 1 (11-21) | 2 (21-24) | 3 (24-26) | 4 (26-29) | 5 (29-37) | 1st vs. 5 th |
| N | 79 | 78 | 52 | 69 | 55 | |
| *Demographic, anthropometric and lifestyle information* | | | | | | |
| Age, y | 59.25 (1.12) | 58.44 (9.38) | 59.90 (8.85) | 59.65 (9.00) | 61.15 (8.60) | .30 |
| Sex, female | 40 (50.63%) | 47 (60.26%) | 34 (65.38%) | 42 (60.87%) | 41 (74.55%) | .009 |
| Race, AA | 27 (34.18%) | 34 (34.59%) | 21 (40.38%) | 23 (33.33%) | 14 (25.45%) | .38 |
| Education | | | | | | .31 |
| High school or less | 11 (13.92%) | 9 (11.54%) | 1 (1.92%) | 4 (5.80%) | 3 (5.46%) | |
| Some college | 22 (27.85%) | 25 (32.05%) | 19 (36.54%) | 14 (20.29%) | 17 (30.91%) | |
| College | 28 (35.44%) | 26 (33.33%) | 21 (40.38%) | 26 (37.68%) | 16 (29.09%) | |
| Post-graduate education | 18 (22.78%) | 18 (23.08) | 11 (21.15%) | 25 (36.23%) | 19 (34.55%) | |
| BMI | 29.68 (6.30) | 29.92 (6.50) | 30.06 (8.23) | 26.66 (5.50) | 27.80 (7.34) | .006 |
| Exercise, MET min/week | 1470.51 (1945.18) | 1675.62 (1848.20) | 2162.35 (2443.83) | 2943.47 (3375.73) | 3291.81 (4322.35) | <.001 |
| *Glucose homeostasis traits* | | | | | | |
| Fasting glucose (mg/dL) | 100.84 (9.52) | 99.50 (12.43) | 98.40 (10.83) | 96.51 (9.92) | 96.22 (11.08) | .01 |
| Fasting insulin (μU/mL) | 13.83 (13.46) | 12.58 (8.44) | 11.14 (7.09) | 10.55 (7.69) | 9.45 (6.13) | .007 |
| Fasting C-Peptide (ng/mL) | 2.61 (1.26) | 2.41 (1.21) | 2.33 (1.14) | 2.21 (1.06) | 2.05 (0.86) | .003 |
| Insulin sensitivity index | 4.31 (3.29) | 4.31 (2.64) | 5.05 (3.96) | 5.62 (3.80) | 6.06 (4.37) | .002 |
| Insulin secretion | 0.46 (0.32) | 0.50 (0.33) | 0.42 (0.22) | 0.43 (0.31) | 0.35 (0.26) | .002 |
| Disposition index | 1.48 (0.85) | 1.65 (0.78) | 1.65 (0.89) | 1.78 (0.88) | 1.65 (0.84) | .20 |
| *Dietary intake* | | | | | | |
| DASH, total score | 18.22 (2.13) | 22.11 (0.83) | 24.45 (0.50) | 26.87 (0.79) | 30.93 (2.20) | <.001 |
| DASH-P | 8.47 (2.76) | 10.46 (2.74) | 12.85 (2.57) | 13.25 (2.58) | 16.44 (2.31) | <.001 |
| DASH-NP | 9.75 (2.77) | 11.66 (2.54) | 11.60 (2.53) | 13.62 (2.53) | 14.49 (2.50) | <.001 |
| Energy intake, kcal/day | 1676.36 (809.38) | 1766.38 1024.94) | 2102.16 (922.75) | 1782.08 (966.44) | 1905.41 (768.83) | .03 |

*Abbreviations*: AA: African-American; BMI: Body mass index, DASH: Dietary approaches to stop hypertension; DASH-P: DASH plant index; DASH-NP: DASH non-plant index; kcal: kilocalories

Note: P-values represent 1st vs. 5th quintile. Continuous variables derived from an unpaired Wilcoxon rank sum test on untransformed variables, and categorical variables derived from a chi-square test of differences

**Table 2:**

Parameter estimates from multivariable linear regression models associating total DASH- score with glucose homeostasis traits, in the whole MILES sample and stratified by dysglycemia and weight status

| | | | Glycemic category | | | | Weight category | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | All participants (N=339) | | Normoglycemic (N=174) | | Dysglycemic (N=159) | | Normal weight (N= 101) | | Overweight/obese (N=232) | |
| | β (SE) | *P* | β (SE) | *P* | β (SE) | *P* | β (SE) | *P* | β (SE) | *P* |
| Fasting glucose | **−0.036 (0.012)** | **.005** | **−0.026 (0.013)** | **.046** | −0.019 (0.014) | .183 | −0.023 (0.020) | .251 | −0.041 (0.016) | .098 |
| Fasting insulin | −0.01 (0.01) | .287 | 0.005 (0.150) | .729 | −0.014 (0.013) | .296 | −0.011 (0.019) | .577 | −0.006 (0.012) | .647 |
| Fasting C-peptide | −0.018 (0.01) | 0.943 | 0.0003 (0.015) | .982 | −0.020 (0.013) | .106 | −0.009 (0.018) | .601 | −0.017 (0.013) | .180 |
| Insulin sensitivity | **0.022 (0.011)** | **.042** | 0.001 (0.014) | .918 | 0.024 (0.013) | .068 | 0.017 (0.019) | .377 | 0.018 (0.012) | .149 |
| Insulin secretion | 0.0003 (0.012) | .978 | 0.013 (0.017) | .458 | −0.011 (0.017) | .53 | −0.002 (0.022) | .941 | 0.007 (0.015) | .659 |
| Disposition index | **0.026 (0.013)** | **.038** | 0.021 (0.172) | .172 | 0.013 (0.014) | .374 | 0.020 (0.022) | .359 | 0.028 (0.016) | .074 |

Note: All models control for age, sex, race, body mass index (BMI), education level, income, physical activity and energy intake. **Significant results (*P*<.05) in bold.**

**Table 3:**

Parameter estimates from multivariable linear regression models associating DASH-P and DASH-NP scores with select glucose homeostasis traits

| | DASH-P | | DASH-NP | |
|---|---|---|---|---|
| | **β (SE)** | ***P*** | **β (SE)** | ***P*** |
| Fasting glucose | **−0.054 (0.017)** | **.002** | −0.013 (0.020) | .519 |
| Insulin sensitivity | **0.036 (0.015)** | **0.014** | 0.003 (0.017) | .851 |
| Disposition index | 0.035 (0.018) | 0.051 | 0.016 (0.020) | .425 |

Note: All models control for age, sex, race, body mass index (BMI), education level, income, physical activity and energy intake. **Significant results (*P*<.05) in bold**. *Abbreviations*: DASH: Dietary approaches to stop hypertension; DASH-P: DASH plant index; DASH-NP: DASH non-plant index

**Table 4:**

Parameter estimates from multivariable linear regression models associating DASH scores for nut intake, fruit intake, vegetable intake and whole grain intake with select glucose homeostasis traits

| | Fasting glucose | | Insulin sensitivity | |
|---|---|---|---|---|
| | **β (SE)** | ***P*** | **β (SE)** | ***P*** |
| Nut score | **−0.094 (0.044)** | **.0035** | **−0.104 (0.037)** | **.005** |
| Fruit score | 0.081 (0.041) | 0.052 | **0.142 (0.042)** | **.049** |
| Vegetable score | 0.019 (0.043) | 0.658 | −0.031 (0.043) | .555 |
| Whole grain score | −0.054 (0.42) | 0.204 | −0.044 (0.043) | .723 |

Note: All models control for age, sex, race, body mass index (BMI), education level, income, physical activity and energy intake. **Significant results (*P*<.05) in bold**.

*Abbreviations*: DASH: Dietary approaches to stop hypertension