Published in final edited form as:
Public Health Nutr. 2009 December ; 12(12): 2493–2503. doi:10.1017/S1368980009991261.

Dietary patterns associated with metabolic syndrome, sociodemographic and lifestyle factors in young adults: the Bogalusa Heart Study

Priya R Deshmukh-Taskar[1], **Carol E O'Neil**[2], **Theresa A Nicklas**[1,*], **Su-Jau Yang**[1], **Yan Liu**[1], **Jeanette Gustat**[3], and **Gerald S Berenson**[3]

[1]Children's Nutrition Research Center, Department of Pediatrics, Baylor College of Medicine, 1100 Bates Avenue, Houston, TX 77030-2600, USA

[2]Louisiana State University AgCenter, Louisiana State University, Baton Rouge, LA, USA

[3]Tulane School of Public Health and Tropical Medicine, New Orleans, LA, USA

Abstract

Objective—To examine the association between dietary patterns (DP) and risk for metabolic syndrome (MetS); and to identify differences in DP by socio-economic, demographic and lifestyle factors.

Design—Dietary intake (from an FFQ), anthropometric/biochemical parameters and sociodemographic/lifestyle information (from a self-reported questionnaire) were evaluated, using a cross-sectional design. Statistical methods included principal component factor analysis, analysis of covariance and linear regression. All analyses were covariate-adjusted.

Setting—The Bogalusa Heart Study (1995–1996), USA.

Subjects—Young adults (19–39 years; *n* 995; 61 % females/39 % males; 80 % whites/20 % blacks) from a semi-rural southern US community were examined.

Results—The 'Western Dietary Pattern' (WDP) consisted of refined grains, French fries, high-fat dairy foods, cheese dishes, red meats, processed meats, eggs, snacks, sweets/desserts, sweetened beverages and condiments. The 'Prudent Dietary Pattern' (PDP) consisted of whole grains, legumes, vegetables, fruits, 100 % fruit juices, low-fat dairy products, poultry, clear soups and low-fat salad dressings. The DP explained 31 % of the dietary intake variance. Waist circumference ($P = 0{\cdot}02$), triceps skinfold ($P = 0{\cdot}01$), plasma insulin ($P = 0{\cdot}03$), serum TAG ($P = 0{\cdot}05$), and the occurrence of MetS ($P = 0{\cdot}03$) were all inversely associated with PDP. Insulin sensitivity ($P < 0{\cdot}0005$) was positively associated with PDP. Serum HDL cholesterol ($P = 0{\cdot}05$) was inversely associated with WDP. Blacks consumed more servings from WDP than whites ($P = 0{\cdot}02$). Females consumed more servings from PDP than males ($P = 0{\cdot}002$). Those with >12 years of education consumed more servings from PDP than their counterparts ($P < 0{\cdot}0001$). Current smokers consumed more servings from WDP than current non-smokers ($P < 0{\cdot}0001$). Physically very active young adults consumed fewer servings from WDP than their sedentary counterparts ($P = 0{\cdot}02$).

Conclusions—More studies are warranted to confirm these findings in other populations.

*Corresponding author: tnicklas@bcm.tmc.edu.

Keywords

Dietary patterns; Metabolic syndrome; Young adults; Blacks; Whites; Demographics; Socio-economic status; Lifestyle factors

Studies involving dietary patterns (DP) and their association with diseases have several benefits over the conventional approach, which has focused largely on the effects of single nutrients or individual foods(1,2). As the measurement of diet is complex, and foods are typically consumed in combinations, the combined effect of nutrients and foods can be observed only when DP are examined(1,2). Moreover, results from DP analyses are more helpful in disseminating diet-related messages to consumers that they may be more likely to adhere to rather than those related to single foods or nutrients(3). DP have also been related to selected biomarkers of dietary exposure(1,2) and have been reported to contribute in the development or prevention of CHD and type 2 diabetes mellitus (T2DM)(4).

Recent focus has been on the occurrence of metabolic syndrome (MetS), a constellation of metabolic abnormalities including central obesity, elevated blood levels of CHD-promoting lipids, hypertension, insulin resistance and hyperglycaemia(5). In adults, MetS increases the risk of CHD by two-fold and the risk for T2DM by five-fold(5–7). The age-adjusted prevalence of MetS in US adults (≥20 years) participating in the 2003–2006(8) National Health and Nutrition Examination Survey (NHANES) was 34 % *v.* 29·2 % reported in the 1988–1994 NHANES(9). Among young adults (20–39 years), the prevalence of MetS has increased from 10·8 % (in 1988–1994)(9) to 15·6 % (in 2003–2006) in females(8), and from 15·7 % (in 1988–1994)(9) to 20·3 % (in 2003–2006)(8) in males. Young adulthood is an important period of transition from adolescence into adulthood, when individuals begin to live an independent life. Pressures of independence, hurried lifestyles and providing support for new families may lead to shifts in their dietary and lifestyle patterns. Consequently, unhealthy dietary habits such as skipping breakfast(10), relying on fast food(11) and eating outside home(12) are prevalent among young adults. Moreover, individuals from rural and semi-rural US communities tend to have poorer dietary and health habits because of their lower socio-economic status (SES) (13–15). It is therefore critical to examine the DP of young adults and their relation to risk factors for chronic diseases in order to administer effective dietary and lifestyle prevention and treatment programmes for metabolic disorders such as the MetS, in this age group.

Despite the rising prevalence of MetS, few recent studies have examined the role of DP and their relationship with MetS(16–20); their results, in general, showed that healthy DP were inversely associated with the occurrence of MetS in adults(16–20). However, to date only one Bogalusa Heart Study (BHS)(21) has examined the relationship of diet with MetS in young adults, and showed that lower fruit and vegetable consumption and higher sweetened beverage consumption were independently associated with one to two risk factors for MetS(21). Yet, the above-mentioned BHS explored the association of only single food groups rather than DP with MetS.

The present study, although an extension of the previous BHS(21), had two additional objectives. First, it aimed to identify various DP among young adults and to examine the association of these DP with the risk factors of MetS. Second, because food consumption differs by SES and demographic factors such as gender and ethnicity(13), and the occurrence of MetS is related to lifestyle factors such as physical inactivity, smoking and alcohol consumption (22), the present study also assessed SES, and demographic and lifestyle differences among the DP in these young adults.

Methods

Study design and participants

The BHS was conducted in the semi-rural community of Washington Parish, Bogalusa, which is 70 miles north of New Orleans, LA(23). The study began in 1973 as a long-term epidemiological investigation of cardiovascular risk factors and their environmental determinants in a bi-racial (black/white) paediatric population. Eventually, the study was expanded to include observations of young adults. Details of the BHS study design, participation rates and protocols are presented elsewhere(23). Data for the present study were collected during a follow-up post-high school cross-sectional survey conducted in 1995–1996 on young adults aged 19–39 years (mean age 30 (SD 5·1) years). Data on ninety-four subjects were excluded from an initial sample of 1089 subjects: i.e. females with energy intakes <2092 kJ (500 kcal) or >14644kJ (3500 kcal)(2) (*n* 47), males with energy intakes <3347kJ (800 kcal) or >16736kJ (4000 kcal)(2) (*n* 23) and pregnant and/or lactating females (*n* 24). The final sample (*n* 995) was 61 % females and 39 % males, with 80 % whites and 20 % blacks. The present study was approved by the Tulane Medical Center Institutional Review Board, and written informed consent was obtained from all the participants.

Measurements

Dietary measures—All young adults from the present study completed the youth/adolescent questionnaire (YAQ), a 131 food-item, self-administered, semi-quantitative food-frequency questionnaire(24,25). The YAQ is valid and reliable for use in epidemiological studies(24, 25). Briefly, this questionnaire included specific foods/beverages (including alcohol) along with a commonly used unit or portion size. Each food/beverage item provided three to six possible responses, ranging from 'never or less than once a month' to 'five or more times per day.' Participants indicated how often, on average, they had consumed a given amount of the specified food/beverage during the past year. Usual portion sizes were calculated for each of the food/beverage items. The selected frequency choice indicated by the participants for each food/beverage was converted to daily intake, e.g. one serving/week was converted to 0·14 serving/d. Food/beverage items were then grouped into specific food categories (as reported earlier)(13), and were further categorised based on food characteristics, e.g. refined or whole grains, low-fat or high-fat dairy foods and so on. Thirty-six food groups from the YAQ were identified for the analyses, of which twenty-four food groups representing the DP from the current study are presented in Table 1.

Anthropometric measures—Duplicate measures of all anthropometric parameters were collected by trained examiners using standardised protocols(23); least-square means and their standard errors are presented. Height was measured to the nearest 0·1 cm on a stadiometer and weight was measured to the nearest 0·1 kg on a balance-beam metric scale. The National Heart, Lung and Blood Institute reference standards(26) were used to classify participants' BMI (weight (kg)/height^2 (m^2)) into normal weight (BMI ≥ 18·5 and ≤24·9kg/m^2) or overweight/obese (BMI ≥ 25 kg/m^2). Young adults who were underweight (BMI < 18·5 kg/m^2) were included with those in the normal weight category. Waist circumference was measured midway between the rib cage and superior border of the iliac crest. Hip circumference was measured at the greater trochanters. Waist-to-hip ratio was calculated. Triceps skinfold was measured to the nearest millimetre with Lange skinfold calipers (Cambridge Scientific Industries, Inc., Cambridge, MD, USA). A description of the reproducibility of these measures used in the BHS is discussed elsewhere(23).

Laboratory measures—Venous blood was collected following a 12h fast. Plasma glucose concentrations were measured by the glucose oxidase method using a Beckman glucose analyzer (Beckman Instruments, Fullerton, CA, USA); a commercial radioimmunoassay kit

measured plasma immunoreactive insulin concentration (Padebas Pharmacia, Piscataway, NJ, USA). Indices of insulin sensitivity were calculated according to the Quantitative Insulin Sensitivity Check Index (QUICKI) formula (=1/(log fasting plasma glucose (mg/dl) + log fasting plasma insulin (μU/ml)); higher QUICKI values indicate greater insulin sensitivity) (27); and the Homeostasis Model Assessment of insulin resistance (HOMA-IR) formula (= (fasting plasma glucose (mg/dl) × fasting plasma insulin (μU/ml))/405; higher HOMA values indicate greater insulin resistance)(28). Serum total cholesterol and TAG concentrations were measured using enzymatic procedures on the Abbott VP instrument (Abbott Laboratories, North Chicago, IL, USA), and serum LDL cholesterol (LDL-C) and HDL cholesterol (HDL-C) were analysed using a combination of heparin–calcium precipitation and agar–agarose gel electrophoretic procedures(29). Blind duplicates were used for quality control for all analyses (23). Right arm systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured in triplicate by trained nurses(23), using the first and fifth Korotkoff phase readings with the participant sitting relaxed, and also using the automated instrument (the readings of which were used in the present study). Means of all replicate measures were used for statistical analyses.

Diagnostic criteria for metabolic syndrome—Several definitions exist for MetS(30). The International Diabetes Federation (IDF) criteria help to generate greater prevalence estimates for MetS (especially, in the European population) by using central obesity as a mandatory criterion. Conversely, the revised Adult Treatment Panel (ATP III)(5) criteria mandate the selection of a wider range of risk factors for identifying individuals with MetS (as discussed below), with no single mandatory criterion. Although, in the USA, both the IDF and revised ATP III criteria identify mostly the same people, the IDF criteria have a lower predictive power for coronary events(30). Therefore, in the present study, the young adults were classified as having MetS using the revised ATP III criteria(5,30). Data on medications prescribed to the young adults to increase serum HDL-C and/or lower serum TAG were not available in the present data set. Therefore, we slightly modified the criteria for serum TAG and HDL-C from the original revised ATP III criteria(5,30).

The criteria for classifying young adults with MetS for the present study were ≥3 of the following risk factors: (i) abdominal obesity (waist circumference ≥ 102 cm in males and ≥88 cm in females); (ii) high serum TAG (≥150 mg/dl); (iii) low serum HDL-C (<40 mg/dl in males or <50 mg/dl in females); (iv) high blood pressure (≥130 or ≥85 mmHg or those taking medications for hypertension); and (v) high fasting plasma glucose (≥100 mg/dl or those taking medications, i.e. oral hypoglycaemic agents/insulin).

Demographic, socio-economic status and lifestyle information—Participants completed a questionnaire eliciting information on their age, gender, ethnicity, smoking status (i.e. non-smoker, current smoker and ex-smoker) and alcohol intake (based on the frequency, type and length of alcohol use during the past 12 months). White/black males and females were classified into four ethnicity × gender groups. The SES of the young adults was determined using income (i.e. ≤$15 000, $15 001–30 000, $30 001–45 000, >$45 000) and education levels (i.e. ≤12 years or >12 years). To determine marital status, young adults were asked whether they were currently married and/or cohabiting or were single. Physical activity outside work was measured with a self-reported subjective rating on a 5-item Likert scale adapted from the Lipid Research Clinic's questionnaire(31). Participants were considered sedentary if they classified themselves as 1 or 2; were considered moderately active if they classified themselves as 3; and were considered very active if they classified themselves as 4 or 5 on the Likert scale. The test–retest reliability of this questionnaire has been reported to be high (r = 0·85). Also, this questionnaire has been noted to be significantly associated with a 4-week physical activity history(31).

Data analysis

The Statistical Analysis Software (version 8·2; SAS Inc., Cary, NC, USA)(32) was used to conduct data analyses. To identify the DP, principal components factor analysis was conducted. Factor analysis helps to summarise and refine large data sets containing several variables, simultaneously, into a small number of orthogonal variables named as 'patterns'. Factor analysis has earlier shown to have good reproducibility and validity with data from a food frequency questionnaire(33).

In the present study, thirty-six food groups from the YAQ were subjected to principal component factor analysis with varimax rotation to identify the DP. Specific food items were aggregated based on the degree to which the food items were correlated with one another in the data set. Eigenvalues (>1), the scree test (a graph from which the number of factors were chosen where the plot levelled off to a linear decreasing pattern) and interpretability of derived factors were used to derive the DP. Linear regression examined the association between DP and MetS risk factors (dependent variables). Analysis of covariance with Tukey–Kramer's *post-hoc* test was used to examine: (i) ethnicity × gender differences in the occurrence of metabolic risk factors (dependent variables); and (ii) differences in mean servings of foods from the DP (dependent variables) by SES, demographic and lifestyle characteristics. The mean number of servings of foods consumed from the DP was used in the latter analyses because factor scores by themselves have no biological meaning. The covariates varied for each analyses and included age, energy intake, gender, ethnicity, ethnicity × gender, SES, marital status, physical activity, smoking, alcohol consumption and BMI. Statistical significance was set at $P \leq 0{\cdot}05$.

Results

Identification of dietary patterns (Table 2)

Factor analysis retained two DP, which contained twenty-four of the original thirty-six food groups from the YAQ. The DP were labelled as: the 'Western Dietary Pattern' (WDP; consisting of refined grains, French fries, high-fat dairy products, dishes with cheese, red meats, processed meats, eggs, snacks, sweets and desserts, sweetened beverages and condiments) and the 'Prudent Dietary Pattern' (PDP; consisting of whole grains, legumes, vegetables (i.e. cruciferous, other leafy and dark-yellow vegetables), tomatoes, fruits, 100 % fruit juices, low-fat dairy products, poultry, clear soups and low-fat salad dressings). The WDP and the PDP explained 19 % and 12 % of the dietary intake variance, respectively.

Covariate-adjusted mean metabolic profiles of young adults (Table 3)

Among the four ethnicity × gender groups, white females had the lowest energy intake, waist circumference, waist-to-hip ratio and SBP; white males had the highest waist circumference and waist-to-hip ratio but the lowest serum HDL-C; and black males had the highest SBP. Compared to white males, white females had lower energy intake, BMI, waist circumference, waist-to-hip ratio, SBP, DBP, plasma glucose, serum total cholesterol, LDL-C and TAG and physical activity, but higher triceps skinfold and serum HDL-C. Compared to black males, black females had higher triceps skinfold and serum HDL-C, but lower waist-to-hip ratio, SBP and DBP. Compared to black females, white females had lower energy intake, BMI, waist circumference, waist-to-hip ratio, SBP, plasma insulin and serum HDL-C, but higher serum TAG. Compared to black males, white males had higher waist circumference and waist-to-hip ratio, but lower SBP and serum HDL-C.

The overall occurrence of MetS in young adults was 12·2 %, with 14·9 % in males *v.* 10·4 % in females ($P = 0{\cdot}03$, data not shown). No ethnic differences in the occurrence of MetS were

observed (12·8 % in whites *v*. 9·6 % in blacks ($P = 0·22$, data not shown)). However, black males had a higher occurrence of Mets than black females (15·4 % *v*. 5·8 %; $P = 0·03$).

Covariate-adjusted associations between dietary patterns and components of metabolic syndrome (Table 4)

Using the covariate-adjusted model (excluding BMI), waist circumference, triceps skinfold, plasma insulin and the occurrence of MetS were all inversely associated with the PDP. Insulin sensitivity was positively associated with the PDP. Serum TAG was negatively associated with both PDP and WDP. After adjusting for BMI in addition to other covariates, serum HDL-C was inversely associated with the WDP. The overall occurrence of MetS did not differ by the two DP.

Covariate-adjusted demographic, socio-economic status and lifestyle differences in dietary patterns (Table 5)

Overall, young adults consumed more servings from the WDP than the PDP (mean 9·8 (SD 0·2) *v*. 4·5 (SD 0·2); $P < 0·0001$, data not shown). Blacks consumed more servings from the WDP than whites, and females consumed more servings from the PDP than males. Whites (males and females) consumed fewer servings from the WDP than black females. White females consumed more servings from the PDP than white males. Older young adults (30–39 years) consumed more servings from the PDP than their younger age group counterparts (19–24 years).

A higher percentage of young adults reported to be in the income category of <$15 000 (27·8 %) compared to $30 001–45 000 (20·6 %; $P = 0·006$, data not shown). Young adults reporting an income level of >$45 000 consumed more servings of the PDP than those reporting lower income, who consumed more servings from the WDP (income model showed significance when adjusted for only gender, ethnicity and ethnicity × gender, but not other covariates). Young adults with >12 years of education consumed more servings from the PDP than those with an education ≤12 years, who consumed more servings from the WDP. Current smokers consumed more servings from the WDP than current non-smokers, who consumed more servings from the PDP. Those who were physically very active (level 5) consumed fewer servings from the WDP than those who were sedentary (level 2).

Discussion

Factor analysis discerned two prominent DP in the present study, the ‘WDP’ mainly characterised by high-fat and high-refined carbohydrate foods, and the ‘PDP’ mainly characterised by low-fat and low-refined carbohydrate foods. A growing body of evidence suggests that increased consumption of healthier foods, including fruits and vegetables(34–37), whole grains/cereals(38), dairy products(39) and other low-fat foods(40), may prevent chronic nutrition-related diseases mainly by their vitamin/mineral(39,41), phytochemical(41) and fibre content(42,43). For example, whole grains have lower glycaemic index and higher fibre content than refined grains, and their consumption may increase insulin sensitivity(43) and plasma levels of anti-inflammatory cytokines (e.g. adiponectin)(44) and reduce serum markers of systemic inflammation (e.g. C-reactive protein and tumour necrosis factor alpha-receptor 2)(38). The calcium content in dairy foods has been hypothesised to lower central obesity and insulin resistance(39,45). Increased consumption of fruits and vegetables has been associated with lower incidence of stroke(34), ischaemic heart disease(34), hypertension(35), T2DM(36) and increased satiety(37), that may help to reduce body weight. Conversely, consumption of energy-dense (i.e. high-fat and/or high-refined carbohydrate foods) may contribute to a surplus intake of ‘discretionary calories’ in the diet (46) and may contribute to the prevalence of overweight/obesity and related chronic diseases over time.

The present study found that several risk factors for CHD, T2DM and MetS were associated with the DP (especially, the PDP). The occurrence of MetS (i.e. more than or equal to three MetS risk factors) was inversely associated with the PDP; however, no association was noted in the occurrence of MetS with the WDP in this study. In a recent longitudinal study(16), participants who were in the highest quintile of the WDP scores (comprising of refined grains, processed meat, fried foods and red meat) had an 18 % greater risk of MetS than those in the lowest quintile for the WDP scores; however, in the same study, consumption of the PDP was not associated with MetS(16). In another cross-sectional study(19), a dietary pattern characterised by a healthy balanced diet (with a frequent intake of raw and salad vegetables, fruits, fish, pasta and rice, and low intake of fried foods, sausages, fried fish and potatoes) was inversely correlated with central obesity, plasma glucose and TAG, and positively correlated with plasma HDL-C. The above dietary pattern(19) was also negatively associated with the risk of having undiagnosed diabetes, and this association was independent of age, gender, smoking and obesity.

In our present study, the finding of serum HDL-C being inversely associated with the WDP (after controlling for BMI and other covariates) is not in agreement with earlier theories(47, 48). In general, diets high in saturated fatty acids tend to increase the cardio-protective serum HDL-C levels along with increasing other CHD-causing lipids (e.g. serum total cholesterol) (47). Conversely, low-fat, high-carbohydrate diets tend to decrease serum HDL-C, but also decrease serum total cholesterol and LDL-C(48).Whether the consumption of a mixture of high-fat and high-refined carbohydrate foods from the WDP led to the inverse association of serum HDL-C with the WDP, or whether the adjustment of BMI as a covariate in the model led to this finding, is not fully understood.

We also found an inverse association of serum TAG with both the DP. Diets high in refined carbohydrates and low in fat, tend to increase serum TAG owing to increased VLDL cholesterol TAG secretion, which is a result of increased hepatic fatty acid availability due to lower fatty acid oxidation(48). The WDP included a mixture of high-fat and high-refined carbohydrate foods that may have resulted in the inverse association of serum TAG with the WDP. Conversely, the inverse association of serum TAG with the PDP could be because of the consumption of low-refined carbohydrate foods (e.g. whole grains, legumes, vegetables and so on) as well as consumption of some low-fat foods (e.g. low-fat dairy products, poultry and low-fat salad dressings). Nevertheless, further investigation in this area is warranted.

Blacks and white males had more risk factors for MetS than white females in the present study. Further, blacks consumed more servings of the WDP, and females consumed more servings from the PDP. Earlier research has reported that blacks were less likely to modify meats to make them lower in fat and ate more fried foods than whites(49). Ethnic disparities in dietary intakes could be attributed to the fact that a larger number of blacks are in the lower SES group than whites(50) and hence may consume poorer diets(13,14) owing to either their inability to afford healthier foods(51), or may have a decreased accessibility to healthier foods(52). White females had the lowest BMI, waist circumference and waist-to-hip ratio than other ethnicity × gender groups, and consumed fewer servings from the WDP than black females. Black females (especially, from low SES groups) tend to be less health conscious than white females, and hence may be less likely to choose healthy dietary and lifestyle patterns(53,54).

In the present study, young adults in the higher SES group (especially, those with higher education) consumed more servings from the PDP. Similar results with respect to SES and food group consumption were suggested earlier(13,19). The relationship between higher SES and the consumption of a PDP could be because of increased knowledge and health awareness, or increased pressures of social acceptability that occur with the increasing SES, which may influence their food consumption habits(53). Among the lower SES groups, the increased cost

of healthier foods or decreased access to healthier foods may be important factors influencing their food choices(51,52).

Current smokers in the present study consumed more servings of the WDP than current non-smokers who consumed more servings from the PDP. Those young adults who reported to be in the highest level of physical activity consumed lesser number of servings from the WDP than those who reported to be less physically active. Often, healthy dietary and lifestyle habits tend to cluster together among individuals. In a recent study(55), relative to non-smokers, current smokers reported higher overall energy intake, higher percentages of energy from fat, sweets and alcohol, and a lower percentage of energy from protein among low-income women. Yet, our study failed to find differences in DP by alcohol consumption and marital status.

The use of factor analysis to identify DP is a major strength of the present study. Although factor analysis takes into account the issue of high inter-correlations of foods within the diet, decisions based on factor loadings may be subjective or arbitrary and can affect the study results and interpretation(56). Nevertheless, a recent study depicted that the young adult age-group frequently consumed less healthier foods(11) resembling the WDP from the current study, suggesting that the factor loadings from our study are robust and the DP are meaningful. Also, the similarity in the results on DP and MetS from our study to those reported in longitudinal (16) and cross-sectional studies(17–20) strengthens the present findings.

The present study has some limitations. Owing to its cross-sectional design, causal inferences cannot be made(57). Despite the large sample size, blacks were under-represented. The findings from this study may be specific to young adults of Bogalusa, and are not representative of national findings. The YAQ, which was originally developed for the dietary assessment of adolescents, was used for the dietary assessment of young adults in the current study. In comparison with a 24 h dietary recall, the YAQ has been more helpful in characterising snack food consumption among the young adults(58). Further, the energy intake when measured by YAQ or the 24 h dietary recall in young adults has been similar(58). Lastly, the dietary data used in our study were collected over 10 years ago; yet, it is not uncommon to publish results from long-term epidemiological studies with data that were collected earlier (e.g. the Framingham offspring cohort study)(20,59). The present BHS findings are thus still noteworthy, as they provide valuable information on the role of DP, MetS and its association with socio-demographic and lifestyle factors. These findings may also help to generate new hypotheses for future research.

Conclusions

Overall, DP are important in identifying relationships with occurrence of diseases such as the MetS. Specifically, a prudent balanced dietary pattern may be helpful in preventing MetS in this sample of BHS young adults. More studies are warranted to confirm these findings in other populations. Nonetheless, nutrition intervention programmes for young adults to promote healthy dietary and lifestyle habits tailored-based on their SES, demographic and lifestyle characteristics, may be beneficial.

Acknowledgments

The present study was supported by funds from the United States Department of Agriculture (USDA), Economic Research Service Cooperative Agreement No. 43-3AEL-2-80122; the National Heart, Lung and Blood Institute, grant HL-38844; National Institute of Aging grant AG-16592; and USDA Hatch 940-36-3104 Project #93673 and LAB93676#0199070. This work is a publication of the United States Department of Agriculture (USDA/ARS) Children's Nutrition Research Center, Department of Pediatrics, Baylor College of Medicine, Houston, TX. The contents of this publication do not necessarily reflect the views or policies of the USDA, nor does mention of trade names, commercial products, or organisations imply endorsement from the US government. The contributions of the authors were as follows: P.R.D-T. interpreted the data, designed and wrote the manuscript; C.O.N. reviewed and edited

the manuscript; T.A.N. designed the study and edited the manuscript; S.J.Y. and Y.L. conducted the statistical analyses; J.G. contributed to the initial conceptualising of the study; and G.S.B. directed and designed the study. None of the authors had a personal or financial conflict of interest. The authors thank the many people who contributed to the Bogalusa Heart Study collaborative effort. The authors also thank the children and young adults of Bogalusa, without whom this study would not have been possible. The authors would like to thank Ms Bee Wong for library assistance and Ms Pam Harris for submitting the manuscript.

References

1. Hu FB. Dietary pattern analysis: a new direction in nutritional epidemiology. Curr Opin Lipidol 2002;13:3–9. [PubMed: 11790957]
2. Willett, WC. Nutritional Epidemiology. Vol. 2nd ed.. New York, NY: Oxford University Press; 1998. p. 20-23.322
3. Freeland-Graves J, Nitzke S. Position of the American Dietetic Association: total diet approach to communicating food and nutrition information. J Am Diet Assoc 2002;102:100–108. [PubMed: 11794489]
4. Brunner EJ, Mosdøl A, Witte DR, Martikainen P, Stafford M, Shipley MJ, Marmot MG. Dietary patterns and 15-y risks of major coronary events, diabetes, and mortality. Am J Clin Nutr 2008;87:1414–1421. [PubMed: 18469266]
5. Grundy SM, Cleeman JI, Daniels SR, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement. Curr Opin Cardiol 2006;21:1–6. [PubMed: 16355022]
6. Hanley AJ, Karter AJ, Williams K, Festa A, D'Agostino RB Jr, Wagenknecht LE, Haffner SM. Prediction of type 2 diabetes mellitus with alternative definitions of the metabolic syndrome: the Insulin Resistance Atherosclerosis Study. Circulation 2005;112:3713–3721. [PubMed: 16344402]
7. Grundy SM. Metabolic syndrome pandemic. Arterioscler Thromb Vasc Biol 2008;28:629–636. [PubMed: 18174459]
8. Ervin, RB. Prevalence of Metabolic Syndrome Among Adults 20 Years of Age and Over, by Sex, Age, Race and Ethnicity, and Body Mass Index: United States, 2003–2006. National Health Statistics Reports. Hyattsville, MD: National Center for Health Statistics; 2009. no. 13
9. Ford ES, Giles WH, Mokdad AH. Increasing prevalence of the metabolic syndrome among US adults. Diabetes Care 2004;27:2444–2449. [PubMed: 15451914]
10. Nicklas TA, Myers L, Reger C, Beech B, Berenson GS. Impact of breakfast consumption on nutritional adequacy of the diets of young adults in Bogalusa, Louisiana: ethnic and gender contrasts. J Am Diet Assoc 1998;98:1432–1438. [PubMed: 9850113]
11. Pereira MA, Kartashov AI, Ebbeling CB, Van Horn L, Slattery ML, Jacobs Dr Jr, Ludwig DS. Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis. Lancet 2005;365:36–42. [PubMed: 15639678]
12. Clemens LH, Slawson DL, Klesges RC. The effect of eating out on quality of diet in premenopausal women. J Am Diet Assoc 1999;99:442–444. [PubMed: 10207396]
13. Deshmukh-Taskar P, Nicklas TA, Yang SJ, Berenson GS. Does food group consumption vary by differences in socioeconomic status, demographics and lifestyle factors in young adults? The Bogalusa Heart Study. J Am Diet Assoc 2007;107:223–234. [PubMed: 17258958]
14. Champagne CM, Bogle ML, McGee BB, Yadrick K, Allen HR, Kramer TR, Simpson P, Gossett J, Weber J. Dietary intake in the lower Mississippi delta region: results from the Foods of our Delta Study. J Am Diet Assoc 2004;104:199–207. [PubMed: 14760567]
15. Smith J, Lensing S, Horton JA, Lovejoy J, Zaghloul S, Forrester I, McGee BB, Bogle ML. Prevalence of self-reported nutrition-related health problems in the Lower Mississippi Delta. Am J Pub Health 1999;89:1418–1421. [PubMed: 10474563]
16. Lutsey PL, Steffen LM, Stevens J. Dietary intake and the development of the metabolic syndrome: the Atherosclerosis Risk in Communities study. Circulation 2008;117:754–761. [PubMed: 18212291]
17. Panagiotakos DB, Pitsavos C, Skoumas Y, Stefanadis C. The association between food patterns and the metabolic syndrome using principal components analysis: The ATTICA Study. J Am Diet Assoc 2007;107:979–987. [PubMed: 17524719]

18. Leite ML, Nicolosi A. Dietary patterns and metabolic syndrome factors in a non-diabetic Italian population. Public Health Nutr 2009;12:1494–1503. [PubMed: 19144241]
19. Williams DE, Prevost AT, Whichelow MJ, Cox BD, Day NE, Wareham NJ. A cross-sectional study of dietary patterns with glucose intolerance and other features of the metabolic syndrome. Br J Nutr 2000;83:257–266. [PubMed: 10884714]
20. Sonnenberg L, Pencina M, Kimokoti R, Quatromoni P, Nam BH, D'Agostino R, Meigs JB, Ordovas J, Cobain M, Millen B. Dietary patterns and the metabolic syndrome in obese and non-obese Framingham women. Obes Res 2005;13:153–162. [PubMed: 15761175]
21. Yoo S, Nicklas T, Baranowski T, Zakeri IF, Yang SJ, Srinivasan SR, Berenson GS. Comparison of dietary intakes associated with metabolic syndrome risk factors in young adults: the Bogalusa Heart Study. Am J Clin Nutr 2004;80:841–848. [PubMed: 15447888]
22. Zhu S, St-Onge MP, Heshka S, Heymsfield SB. Lifestyle behaviors associated with lower risk of having the metabolic syndrome. Metabolism 2004;53:1503–1511. [PubMed: 15536610]
23. Berenson GS. the Bogalusa Heart Study Investigators. Bogalusa Heart Study: a long-term community study of a rural biracial (black/white) population. Am J Med Sci 2001;322:293–300. [PubMed: 11876192]
24. Rockett HR, Breitenbach M, Frazier AL, Witschi J, Wolf AM, Field AE, Colditz GA. Validation of a youth/adolescent food frequency questionnaire. Prev Med 1997;26:808–816. [PubMed: 9388792]
25. Rockett HRH, Wolf AM, Colditz GA. Development and reproducibility of a food frequency questionnaire to assess diets of older children and adolescents. J Am Diet Assoc 1995;95:336–340. [PubMed: 7860946]
26. US Department of Health and Human Services, National Institutes of Health. How Are Overweight and Obesity Diagnosed. 2008 [accessed August 2009]. http://www.nhlbi.nih.gov/health/dci/Diesease/obe/obe_daignosis.html
27. Katz A, Nambi SS, Mather K, Baron AD, Follmann DA, Sullivan G, Quon MJ. Quantitative insulin sensitivity check index: a simple, accurate method for assessing insulin sensitivity in humans. J Clin Endocrinol Metab 2000;85:2402–2410. [PubMed: 10902785]
28. Mathews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and β-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 1985;28:412–419. [PubMed: 3899825]
29. Srinivasan, SR.; Berenson, GS. Serum lipoprotein in children and methods for study. In: Lewis, LA., editor. CRC Handbook of Electrophoresis. Lipoprotein Methodology and Human Studies. Boca Raton, FL: CRC Press; 1983. p. 185-204.
30. Assmann G, Guerra R, Fox G, Cullen P, Schulte H, Willett D, Grundy SM. Harmonizing the definition of the metabolic syndrome: comparison of the criteria of the Adult Treatment Panel III and the International Diabetes Federation in United States American and European populations. Am J Cardiol 2007;99:541–548. [PubMed: 17293200]
31. Ainsworth BE, Jacobs DR Jr, Leon AS. Validity and reliability of self-reported physical activity status: the Lipid Research Clinics questionnaire. Med Sci Sports Exerc 1993;25:92–98. [PubMed: 8423761]
32. SAS Inc. Statistical Analysis Software (SAS) Version 8·2. Cary, NC: 1999.
33. Hu FB, Rimm E, Smith-Warner SA, Feskanich D, Stampfer MJ, Ascherio A, Sampson L, Willett WC. Reproducibility and validity of dietary patterns assessed with a food-frequency questionnaire. Am J Clin Nutr 1999;69:243–249. [PubMed: 9989687]
34. Bazzano LA, He J, Ogden LG, Loria CM, Vupputuri S, Myers L, Whelton PK. Fruit and vegetable intake and risk of cardiovascular disease in US adults: the first National Health and Nutrition Examination Survey Epidemiologic Follow-up Study. Am J Clin Nutr 2002;76:93–99. [PubMed: 12081821]
35. Alonso A, de la Fuente C, Martin-Arnau AM, de Irala J, Martinez JA, Martinez-Gonzalez MA. Fruit and vegetable consumption is inversely associated with blood pressure in a Mediterranean population with a high vegetable-fat intake: the Seguimiento Universidad de Navarra (SUN) Study. Br J Nutr 2004;92:311–319. [PubMed: 15333163]

36. Williams DE, Wareham NJ, Cox BD, Byrne CD, Hales CN, Day NE. Frequent salad vegetable consumption is associated with a reduction in the risk of diabetes mellitus. J Clin Epidemiol 1999;52:329–335. [PubMed: 10235173]
37. Rolls BJ, Ello-Martin JA, Tohill BC. What can intervention studies tell us about the relationship between fruit and vegetable consumption and weight management? Nutr Rev 2004;62:1–17. [PubMed: 14995052]
38. Qi L, van Dam RM, Liu S, Franz M, Mantzoros C, Hu FB. Whole-grain, bran, and cereal fiber intakes and markers of systemic inflammation in diabetic women. Diabetes Care 2006;29:207–211. [PubMed: 16443861]
39. Zemel MB, Richards J, Milstead A, Campbell P. Effects of calcium and dairy on body composition and weight loss in African-American adults. Obes Res 2005;13:1218–1225. [PubMed: 16076991]
40. Johnston CS, Tjonn SL, Swan PD. High-protein, low-fat diets are effective for weight loss and favorably alter biomarkers in healthy adults. J Nutr 2004;134:586–591. [PubMed: 14988451]
41. Tucker KL. Dietary intake and coronary heart disease: a variety of nutrients and phytochemicals are important. Curr Treat Opt Cardiovasc Med 2004;6:291–302.
42. Hallfrisch J, Behall KM. Mechanisms of the effects of grains on insulin and glucose responses. J Am Coll Nutr 2000;19:S320–S325.
43. Steffen LM, Jacobs DR Jr, Murtaugh MA, Moran A, Steinberger J, Hong CP, Sinaiko AR. Whole grain intake is associated with lower body mass and greater insulin sensitivity among adolescents. Am J Epidemiol 2003;158:243–250. [PubMed: 12882946]
44. Yannakoulia M, Yiannakouris N, Melistas L, Kontogianni MD, Malagaris I, Mantzoros CS. A dietary pattern characterized by high consumption of whole-grain cereals and low-fat dairy products and low consumption of refined cereals is positively associated with plasma adiponectin levels in healthy women. Metabolism 2008;57:824–830. [PubMed: 18502266]
45. Pereira MA, Jacobs DR Jr, Van Horn L, Slattery ML, Kartashov AI, Ludwig DS. Dairy consumption, obesity, and the insulin resistance syndrome in young adults: the CARDIA Study. JAMA 2002;287:2081–2089. [PubMed: 11966382]
46. UD Department of Agriculture & US Department of Health and Human Services. Dietary guidelines for Americans. 2005 [accessed June 2009]. http://www.healthierus.gov/dietaryguidelines/2005
47. Samaha FF. Effect of very high-fat diets on body weight, lipoproteins, and glycemic status in the obese. Curr Atheroscler Rep 2005;7:412–420. [PubMed: 16255998]
48. Pelkman CL, Fishell VK, Maddox DH, Pearson TA, Mauger DT, Kris-Etherton PM. Effects of moderate-fat (from monounsaturated fat) and low-fat weight-loss diets on the serum lipid profile in overweight and obese men and women. Am J Clin Nutr 2004;79:204–212. [PubMed: 14749224]
49. Gans KM, Burkholder GJ, Risica PM, Lasater TM. Baseline fat-related dietary behaviors of white, Hispanic, and black participants in a cholesterol screening and education project in New England. J Am Diet Assoc 2003;103:699–706. [PubMed: 12778040]
50. Goodman E, Adler NE, Daniels SR, Morrison JA, Slap GB, Dolan LM. Impact of objective and subjective social status on obesity in a biracial cohort of adolescents. Obes Res 2003;11:1018–1026. [PubMed: 12917508]
51. Drewnowski A. Obesity and the food environment: dietary energy density and diet costs. Am J Prev Med 2004;27:S154–S162.
52. Baker EA, Schootman M, Barnidge E, Kelly C. The role of race and poverty in access to foods that enable individuals to adhere to dietary guidelines. Prev Chronic Dis 2006;3:A76. [PubMed: 16776877]
53. Colhoun, H.; Prescott-Clarke, P. Health Survey for England 1994. London: HMSO; 1996. cited in McLaren L, Kuh D. Women's body dissatisfaction, social class, and social mobility. Soc Sci Med 2004;58:1575–1584. [PubMed: 14990360]
54. Duncan GE, Anton SD, Newton RL Jr, Perri MG. Comparison of perceived health to physiological measures of health in black and white women. Prev Med 2003;36:624–628. [PubMed: 12689808]
55. Delahanty JC, DiClemente CC, Havas S, Langenberg P. Smoking status and stages of change for dietary behaviors among WIC women. Am J Health Behav 2008;32:583–593. [PubMed: 18442338]
56. Martinez ME, Marshall JR, Sechrest L. Invited commentary: factor analysis and the search for objectivity. Am J Epidemiol 1998;148:17–19. [PubMed: 9663398]

57. Gordis, L. Epidemiology. Vol. 2nd ed.. W. B Saunders Co; 2000. Case control and cross-sectional studies; p. 153-155.

58. Nicklas TA. Dietary studies of children and young adults (1973–1988): the Bogalusa Heart Study. Am J Med Sci 1995;310:S101–S108. [PubMed: 7503111]

59. Imamura F, Lichtenstein AH, Dallal GE, Meigs JB, Jacques PF. Confounding by dietary patterns of the inverse association between alcohol consumption and type 2 diabetes risk. Am J Epidemiol. 2009Epublication 8 May 2009

Table 1

Components of food groups* included in the DP from the YAQ: The Bogalusa Heart Study

Food groups	Foods included
1. Whole grains	Hot breakfast cereal (e.g. oatmeal, grits), dark breads, other grains (e.g. bulgur, kasha, couscous)
2. Refined grains	White bread, pita bread or toasts, muffins, cornbreads, bagels, biscuits or rolls, rice, noodles, pasta, pancakes, waffles, tortillas
3. Low-fat dairy products	Skim or 1 % milk, non-fat or low-fat yoghurt and cheese, other non-fat dairy products
4. High-fat dairy products	Whole or 2 % milk/chocolate milk, whipped cream, regular yoghurt, cheese, cottage cheese, cream cheese, pudding, frozen yoghurt, ice cream, milkshake or frappe
5. Fruits	Grapes, raisins, bananas, cantaloupes, melons, apples/apple sauce, pears, oranges, strawberries, peaches, plums, apricots
6. 100 % fruit juices	100 % fruit juices
7. Tomatoes	Tomatoes, tomato sauce, spaghetti sauce, salsa
8. Legumes	Beans, lentils, soybeans, peas, lima beans
9. Cruciferous vegetables	Broccoli, greens, coleslaw, kale
10. Green leafy vegetables	Spinach, lettuce, tossed salad
11. Dark-yellow/orange vegetables	Carrots, yams, sweet potatoes
12. Other vegetables	String beans, beets, corn, peppers, eggplant, zucchini, mixed vegetables, summer squash
13. French fries	French fries
14. Red meats	Beef, steak, lamb, pork, meatballs, meatloaf, ham
15. Processed meats	Processed meats, bacon, hot dogs, salami, bologna
16. Poultry	Chicken, turkey, chicken nuggets
17. Eggs	Eggs
18. Main dishes	
Dishes with cheese	Pizza, tacos/burritos, lasagna, baked ziti, macaroni and cheese, spaghetti, grilled cheese
Burgers and sandwiches	Cheese burger, hamburger, peanut butter sandwich, chicken/turkey sandwich, roast beef/ham sandwich, deli meat sandwich, tuna sandwich, other fish sandwich
19. Snacks	Potato chips, corn chips, nachos, popcorn, pretzels, crackers, peanuts, fun fruit, graham crackers, saltines, wheat thins
20. Sweets and desserts	Pop tarts, cakes, snack cakes, Twinkies, Danish pastries, pastries, donuts, cookies, brownies, pie, chocolates, candy bars, other candy such as mints, flavoured gelatin, pudding, frozen yoghurt, ice cream, milkshake, popsicles
21. Sweetened beverages	Soda, punch, lemonade, non-carbonated fruit drink, iced tea
22. Low-fat salad dressings	Low-fat salad dressing
23. Condiments	Brown gravy, ketchup, mayonnaise, added sugar
24. Low-fat soups	Clear soup, chicken noodle soup

DP, dietary patterns; YAQ, youth and adolescent food frequency questionnaire.

* Only twenty-four food groups identified in the DP (from Table 2) are discussed above from a total of thirty-six food groups from the YAQ.

Table 2

Identification of DP from factor loadings* for foods from the YAQ: The Bogalusa Heart Study

Food items	Factor loadings	
	WDP	PDP
1. Whole grains	–	0·46
2. Legumes	–	0·61
3. Cruciferous vegetables	–	0·70
4. Other vegetables	–	0·74
5. Green leafy vegetables	–	0·69
6. Dark-yellow vegetables	–	0·70
7. Tomatoes	–	0·58
8. Fruits	–	0·64
9. 100 % fruit juices	–	0·43
10. Low-fat dairy products	–	0·36
11. Poultry	–	0·40
12. Clear soups	–	0·36
13. Low-fat salad dressings	–	0·49
14. Refined grains	0·43	–
15. French fries	0·53	–
16. High-fat dairy products	0·53	–
17. Dishes with cheese	0·58	–
18. Red meats	0·50	–
19. Processed meats	0·59	–
20. Eggs	0·39	–
21. Snacks	0·53	–
22. Sweets and desserts	0·54	–
23. Sweetened beverages	0·44	–
24. Condiments	0·40	–
Variability explained	19 %	12 %

DP, dietary patterns; YAQ, youth and adolescent food frequency questionnaire; WDP, Western dietary pattern; PDP, prudent dietary pattern.

* Data (1–24) are factor loadings (correlation coefficients between the variables and factors) derived from principal component factor analysis. Absolute values of factor loadings <0·30 are indicated by '–' for simplicity.

Table 3

Covariate-adjusted metabolic profiles of young adults* (19–39 years) by ethnicity and gender: The Bogalusa Heart Study

	White males (*n* 311)		White females (*n* 486)		Black males (*n* 78)		Black females (*n* 120)		*P*-values†	
	Least-square mean	SE	Least-square mean	SE	Least-square mean	SE	Least-square mean	SE	Unadjusted model	Adjusted model
Age (years)	$30{\cdot}2^{a}$	0·3	$29{\cdot}6^{a,b}$	0·2	$30{\cdot}3^{a,b}$	0·6	$28{\cdot}5^{b}$	0·5	<0·01	
Total energy intake (kJ)‡	920^{a}	182·0	8155^{b}	156·1	9866^{a}	366·3	8966^{a}	294·0		<0·0001¶
Total energy intake (kcal)	2201^{a}	43·5	1949^{b}	37·3	2358^{a}	87·5	2143^{a}	70·3		<0·0001¶
Obesity measurements										
BMI (kg/m^2)§	$28{\cdot}2^{a}$	0·4	$25{\cdot}8^{b}$	0·3	$27{\cdot}8^{a,b}$	0·9	$29{\cdot}8^{a}$	0·7		<0·0001**
Waist circumference (cm)	$94{\cdot}8^{a}$	0·9	$79{\cdot}4^{b}$	0·8	$89{\cdot}3^{c}$	1·9	$88{\cdot}4^{c}$	1·5		<0·0001**
Waist-to-hip ratio	$0{\cdot}9^{a}$	0·0	$0{\cdot}76^{b}$	0·0	$0{\cdot}84^{c}$	0·0	$0{\cdot}8^{d}$	0·0		<0·0001**
Triceps skinfold (mm)	$18{\cdot}5^{a}$	0·7	$27{\cdot}2^{b}$	0·6	$16{\cdot}7^{a}$	1·4	$30{\cdot}0^{b}$	1·1		<0·0001**
Physical activity	$3{\cdot}4^{a}$	0·1	$3{\cdot}1^{b}$	0·5	$3{\cdot}6^{a}$	0·1	$3{\cdot}2^{a,b}$	0·1		<0·0001††
Blood pressure (mmHg)										
Systolic	$113{\cdot}3^{a}$	0·7	$106{\cdot}2^{b}$	0·6	$117{\cdot}0^{c}$	1·3	$109{\cdot}6^{d}$	1·1		<0·0001‡‡
Diastolic	$75{\cdot}8^{a}$	0·6	$71{\cdot}6^{b}$	0·5	$77{\cdot}4^{a}$	1·1	$72{\cdot}3^{b}$	0·9		<0·0001‡‡
Plasma glucose (mg/dl)	$81{\cdot}7^{a}$	0·8	$77{\cdot}5^{b}$	0·6	$81{\cdot}8^{a}$	1·5	$78{\cdot}9^{a,b}$	1·2		<0·0001‡‡
Plasma insulin (μU/ml)	$12{\cdot}3^{a,b}$	0·5	$11{\cdot}0^{a}$	0·5	$13{\cdot}9^{a,b}$	1·1	$13{\cdot}9^{b}$	0·9		<0·0001‡‡
Serum lipids										
Total cholesterol (mg/dl)	$200{\cdot}1^{a}$	2·7	$191{\cdot}2^{b}$	2·3	$199{\cdot}3^{a,b}$	5·4	$190{\cdot}0^{a,b}$	4·3		<0·02‡‡
HDL-C (mg/dl)	$41{\cdot}1^{a}$	0·8	$50{\cdot}3^{b}$	0·7	$48{\cdot}4^{b}$	1·6	$54{\cdot}6^{c}$	1·3		<0·0001‡‡
LDL-C (mg/dl)	$133{\cdot}3^{a}$	2·3	$120{\cdot}2^{b}$	2·0	$131{\cdot}0^{a,b}$	4·6	$122{\cdot}2^{b}$	3·7		<0·0001‡‡
TAG (mg/dl)	$139{\cdot}9^{a}$	7·6	$116{\cdot}8^{b}$	6·5	$108{\cdot}5^{a,b,c}$	15·3	$77{\cdot}7^{c}$	12·3		<0·0001‡‡
	n	%	*n*	%	n	%	*n*	%		
MetS//	46	14·8	56	11·5	12	$15{\cdot}4^{a}$	7	$5{\cdot}8^{b}$		0·03

HDL-C, HDL cholesterol; LDL.C, LDL cholesterol; MetS, metabolic syndrome.

^{a,b,c,d}Mean (percentage) values within a row with unlike superscript letters were significantly different ($P < 0{\cdot}01$).

*All young adults (*n* 995) were fasting before the blood draw.

†Least-square means for dependent variables compared by analysis of covariance and Tukey–Kramer's *post hoc* test.

‡1 kcal54194 kJ.

§Normal weight, BMI ≥ 18·5 and ≤24·9 kg/m^2 ; overweight/obese, BMI ≥ 25·0 kg/m^2.

//Diagnosis of MetS based on ≥3 of the following risk factors: waist circumference ≥102 cm in males and ≥88 cm in females; serum TAG ≥ 150 mg/dl; serum HDL-C < 40 mg/dl in males and <50 mg/dl in females; blood pressure ≥130 or ≥85 mmHg or taking medications for hypertension; and fasting plasma glucose ≥100 mg/dl or taking medications (oral hypoglycaemic agents/insulin).

¶Energy intake model adjusted for age, socio-economic status (SES), smoking status, alcohol intake and physical activity.

**Obesity measurement model(s) adjusted for energy intake, age, SES, smoking status, alcohol intake and physical activity.

††Physical activity model (1 = sedentary, 3 = moderately active and 5 = very active) adjusted for energy intake, age and SES.

##Blood/plasma/serum parameters model(s) adjusted for energy intake, age, SES, smoking status, alcohol intake and physical activity.

Table 4

Covariate-adjusted associations between risk factors for MetS* and DP in young adults (19–39 years)

	Model 1†				Model 2‡			
	PDP (*n* 995)		WDP (*n* 995)		PDP (*n* 995)		WDP (*n* 995)	
Risk factors (dependent variables)	**Std β**	***P* value**	**Std β**	***P* value**	**Std β**	***P* value**	**Std β**	***P* value**
Obesity measurements								
BMI§	−0·07	0·15	−0·05	0·42	–	–	–	–
Waist circumference	−0·10	0·02	−0·05	0·46	−0·04	0·009	0·00	1·00
Triceps skinfold	−0·11	0·01	−0·02	0·75	−0·06	0·17	0·02	0·62
Blood pressure								
Systolic	−0·05	0·29	−0·05	0·42	−0·03	0·51	−0·04	0·60
Diastolic	−0·01	0·80	0·00	0·99	0·01	0·86	0·17	0·80
Diabetes mellitus								
Plasma glucose	−0·03	0·52	−0·05	0·49	−0·01	0·83	−0·03	0·64
Plasma insulin	−0·10	0·03	−0·04	0·53	−0·07	0·09	−0·01	0·83
Insulin resistance: HOMA-IR	−0·07	0·11	−0·04	0·58	−0·04	0·33	−0·01	0·89
Insulin sensitivity: QUICKI	0·16	<0·0005	0·02	0·78	0·13	0·001	−0·01	0·82
Lipid profiles								
Total serum cholesterol	−0·01	0·88	−0·08	0·22	0·00	0·94	−0·08	0·27
Serum LDL-C	−0·01	0·91	−0·02	0·77	0·01	0·92	−0·01	0·93
Serum HDL-C	0·05	0·26	−0·11	0·10	0·03	0·45	−0·12	0·05
Serum TAG	−0·10	0·05	−0·13	0·05	−0·07	0·10	−0·12	0·07
MetS*								
≥3 risk factors	−0·10	0·03	−0·08	0·22	−0·07	0·07	−0·05	0·35
	OR	CI	OR	CI	OR	CI	OR	CI
MetS*								
(Reference = no MetS)	0·93	0·80, 1·07	0·93	0·80, 1·07	0·93	0·80, 1·07	0·93	0·80, 1·07

MetS, metabolic syndrome; DP, dietary patterns; PDP, prudent dietary pattern; WDP, Western dietary pattern; Std β, standardised β; HOMA-IR, Homeostasis

Model Assessment of insulin resistance; QUICKI, Quantitative Insulin Sensitivity Check Index; LDL-C, LDH cholesterol; HDL-C, HDL cholesterol.

*Diagnosis of MetS based on ≥3 of the following risk factors: waist circumference ≥ 102 cm in males and ≥88 cm in females; serum triacylglycerol ≥ 150 mg/dl; HDL-C < 40 mg/dl in males and <50 mg/dl in females; blood pressure ≥ 130 or ≥85 mmHg or taking medications for hypertension; and fasting plasma glucose ≥ 100 mg/dl or taking medications (oral hypoglycaemic agents/insulin).

†Model 1 adjusted for age, energy intake, ethnicity, gender, ethnicity × gender, socio-economic status (SES), physical activity, alcohol intake and smoking status.

‡Model 2 adjusted for BMI in addition to age, energy intake, ethnicity, gender, ethnicity × gender, SES, physical activity, alcohol intake and smoking status.

§BMI calculated as weight (kg)/height^2 (m^2) with normal weight defined as BMI ≥18·5 and ≤24·9 kg/m^2 ; and overweight/obese defined as BMI ≥ 25·0 kg/m^2.

Table 5

Covariate-adjusted demographic, SES and lifestyle differences in servings of foods from DP among young adults (19–39 years): The Bogalusa Heart Study

Characteristics of young adults	Sample size (*n*)*	Fully adjusted model† PDP Mean	SE	Fully adjusted model† WDP Mean	SE	Unadjusted‡/partially adjusted model§ PDP Mean	SE	Unadjusted‡/partially adjusted model§ WDP Mean	SE
Demographics									
Ethnicity									
Whites	7974·2		0·1	10·0	0·1	4·7	2·8	9·8	3·8
Blacks	1984·0		0·2	10·5	0·2	4·6	2·4	11·1	4·2
		$P = 0·46^{\dagger}$		$P = 0·02^{\dagger}$		$P = 0·74^{\ddagger}$		$P < 0·0001^{\ddagger}$	
Gender									
Males	3893·8		0·2	10·1	0·2	4·6	2·8	10·6	4·3
Females	6064·5		0·2	10·4	0·2	4·7	2·7	9·4	3·6
		$P = 0·002^{\dagger}$		$P = 0·18^{\dagger}$		$P = 0·45^{\ddagger}$		$P < 0·0001^{\ddagger}$	
Ethnicity × gender									
White males	$3113·8^{a}$		0·2	$10·1^{a}$	0·2	4·6	2·9	$10·3^{a}$	4·1
White females	$4864·6^{b}$		0·1	$10·0^{a}$	0·1	4·7	2·7	$9·0^{b}$	3·5
Black males	$783·7^{a,b}$		0·3	$10·2^{a,b}$	0·3	4·5	2·0	$11·5^{a}$	4·9
Black females	$1204·3^{a,b}$		0·3	$10·9^{b}$	0·3	4·6	2·7	$10·9^{a}$	3·8
		$P = 0·8^{\dagger}$		$P = 0·07^{\dagger}$		$P = 0·87^{\ddagger}$		$P < 0·0001^{\ddagger}$	
		a v. b, $P = 0·01^{\dagger}$		*a v. b*, $P = 0·01^{\dagger}$					
Age groups (years)									
19–24	$2083·5^{a}$		0·2	10·5	0·2	4·2	2·8	10·2	4·2
25–29	$2734·0^{a,b}$		0·2	10·3	0·2	4·7	2·8	10·0	4·0
30–34	$3024·4^{b}$		0·2	10·1	0·2	4·7	2·6	9·6	3·6
35–39	$2124·5^{b}$		0·2	10·2	0·2	4·9	2·6	9·6	3·9
		$P = 0·001^{\dagger}$		$P = 0·63^{\dagger}$		$P = 0·08^{\ddagger}$		$P = 0·30^{\ddagger}$	
SES*									
Income ($)									
≤ 15000	2623·8		0·2	10·2	0·2	$4·2^{a}$	2·2	$10·6^{a}$	4·1
15 001–30000	2594·0		0·2	10·4	0·2	$4·5^{a,b}$	2·9	$10·0^{a,b}$	3·9
30001–45000	1954·2		0·2	10·3	0·2	$4·8^{a,b}$	3·1	$9·6^{a,b}$	3·7
≥45001	2274·5		0·2	10·2	0·2	$5·1^{b}$	2·6	$9·0^{b}$	3·8
		$P = 0·10^{\dagger}$		$P = 0·88^{\dagger}$		$P < 0·001^{\S}$		$P < 0·05^{\S}$	
Education (years)									
≤12	2723·6		0·2	10·7	0·2	3·9	1·9	10·7	4·1
>12	6414·6		0·2	9·8	0·2	5·0	2·9	9·4	3·8
		$P < 0·0001^{\dagger}$		$P < 0·0001^{\dagger}$		$P < 0·0001^{\S}$		$P < 0·001^{\S}$	
Lifestyle factors									
Current smokers									
No	6654·07		0·2	9·9	0·1	4·9	2·7	9·5	3·9
Yes	3304·14		0·1	10·6	0·2	4·1	2·5	10·6	4·0
		$P < 0·0001^{\dagger}$		$P < 0·0001^{\dagger}$		$P < 0·0001^{\S}$		$P < 0·0001^{\S}$	
Current drinkers									
No	2614·5		0·2	10·3	0·2	4·6	2·7	9·7	3·9
Yes	7343·7		0·2	10·2	0·1	4·7	2·7	9·9	4·0
		$P = 0·74^{\dagger}$		$P = 0·57^{\dagger}$		$P = 0·56^{\S}$		$P = 0·38^{\S}$	
Physical activity (outside of work)*									
1 (sedentary)	613·8		0·3	$10·3^{a,b}$	0·3	4·5	2·5	10·1	3·8
2	1563·9		0·2	$10·8^{a}$	0·2	4·6	2·5	10·3	40

Characteristics of young adults	Sample size (*n*)*	Fully adjusted model†				Unadjusted‡/partially adjusted model§			
		PDP		WDP		PDP		WDP	
		Mean	SE	Mean	SE	Mean	SE	Mean	SE
3 (moderately active)	4574·0		0·2	10·3[a,b]	0·2	4·5	2·5	9·7	3·8
4	1554·4		0·2	10·2[a,b]	0·2	4·8	3·1	9·5	3·8
5 (very active)	1664·5		0·2	9·8[b]	0·2	50	30	10·0	4·6
		$P = 0·08$†		$P = 0·02$†		$P = 0·31$§		$P = 0·15$§	
Marital status*									
Unmarried	4524·3		0·2	10·2	0·2	4·6	2·5	10·1	4·2
Married	5334·0		0·2	10·3	0·2	4·6	2·9	9·6	3·7
		$P = 0·09$†		$P = 0·55$†		$P = 0·73$§		$P = 0·66$§	

SES, socio-economic status; DP, dietary patterns; PDP, prudent dietary pattern; WDP, Western dietary pattern.

[a,b]Mean values within a column with unlike superscript letters were significantly different by analysis of covariance and Tukey–Kramer's *post hoc* test ($P < 0·05$).

*Sample size differs from the original sample (*n* 995) due to missing data for some subjects for SES, physical activity and marital status.

†Fully adjusted model controlled for age, energy intake, gender, ethnicity, ethnicity × gender, SES, physical activity, smoking status, alcohol intake and marital status.

‡Unadjusted model.

§Partially adjusted model controlled for gender, ethnicity and ethnicity × gender.