

The Effect of Carbohydrate Amount, Quality and Type on Arterial Pulse Pressure in Cuban-Americans with and Without Type 2 Diabetes

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Abstract

Background: Arterial pulse pressure, the difference between systolic and diastolic blood pressure, has been used as an indicator (surrogate measure) of arterial stiffness. High arterial pulse pressure (>40) has been associated with increased cardiovascular disease and mortality. Several clinical trials have reported that the proportion of calories from carbohydrate has an effect on blood pressure. The primary objective of this study was to assess arterial pulse pressure and its association with carbohydrate quantity and quality (glycemic load) with diabetes status for a Cuban American population.

Methods: A single point analysis included 367 participants. There was complete data for 365 (190 with and 175 without type 2 diabetes). The study was conducted in the investigator's laboratory located in Miami, Florida. Demographic, dietary, anthropometric and laboratory data were collected. Arterial pulse pressure was calculated by the formula systolic minus the diastolic blood pressure. Glycemic load, fructose, sucrose, percent of average daily calories from carbohydrate, fat and protein, grams of fiber and micronutrient intakes were calculated from a validated food frequency questionnaire.

Results: The mean arterial pulse pressure was significantly higher in participants with (52.9 ± 12.4) than without (48.6 ± 13.4) type 2 diabetes. The odds of persons with diabetes having high arterial pulse pressure (>40) was 1.85 (95% CI = 1.09, 3.13); $p=0.023$. For persons with type 2 diabetes higher glycemic load was associated with lower arterial pulse pressure.

Conclusions: Arterial pulse pressure and diet are modifiable risk factors of cardiovascular disease. Arterial pulse pressure may be associated with carbohydrate intake differently considering diabetes status. Results may be due to individuals with diabetes following dietary recommendations. The findings of this study suggest clinicians take into consideration how medical condition, ethnicity and diet are associated with arterial pulse pressure before developing a medical nutrition therapy plan in collaboration with the client.

Keywords: Arterial pulse pressure; Carbohydrate quality; Glycemic load; Fructose; Cuban American; Type 2 Diabetes

Abbreviations: ADA: American Diabetes Association; BMI: Body Mass Index; CHD: Coronary Heart Disease; CVD: Cardiovascular Disease; DBP: Diastolic Blood Pressure; FFQ: Food Frequency Questionnaire; HDL-C: High Density Lipoprotein Cholesterol; Hg: Mercury; HTN: Hypertension; Kcal: Kilocalories; LDL-C: Low Density; Lipoprotein Cholesterol; PP: Arterial Pulse Pressure; SBP: Systolic Blood Pressure; WC: Waist Circumference; WHO: World Health Organization.

Introduction

Cardiovascular disease (CVD) is a major complication and a leading cause of early death among persons with diabetes. About 65% of the people with diabetes die from heart disease and stroke [1,2]. Death rates among adults with diabetes were 2-4 times higher from heart disease and about 2.8 times greater from stroke, when compared to individuals without diabetes [3,4]. Prevalence of diabetes adjusting for age differences for people 20 years or older was 8.2% for Cuban Americans as compared to 6.6% for non-Hispanic Whites [3]. Also alarming is the high mortality rate of Cuban Americans from diabetes (47 per 100,000 people) as opposed to (22 per 100,000) for non-Hispanic Whites [5]. Although, Cuban Americans represent the largest segment (21%) of the elderly population among all US ethnic/racial groups [6], adequate research has not been directed towards diabetes and CVD risk of this minority population.

Numerous studies suggest that a possible link between diabetes

and CVD could be due to stiffening of arteries. The progressive rise in systolic blood pressure (SBP) and decrease in diastolic blood pressure (DBP) can lead to an increased arterial pulse pressure (PP) which, in turn promotes stiffening of arteries [7]. As such, PP can be considered a risk factor for arterial stiffness [8-12]. Arterial stiffness is an independent CVD risk factor in patients with diabetes [13,14]. PP may serve as a cost-effective, preliminary screening tool for heart health, since arterial stiffness has been associated with CVD risk in patients with diabetes [13,14].

Dietary composition has been associated with CVD risk and the metabolic syndrome (a cluster of abnormalities resulting in insulin resistance) throughout the literature. Studies with the association of carbohydrate type and amount with blood pressure, a component

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of metabolic syndrome have come to different conclusions. The associations among PP, type 2 diabetes and carbohydrate intake have not been studied, particularly in a population at high risk for diabetes and CVD. Therefore, the aim of this study was to assess the effects of glycemic load, carbohydrate type, percent of calories from carbohydrate, with diabetes status on PP for a Cuban American population.

Materials and Methods

Design and study population

The present study is a single point analysis of Cuban Americans with and without type 2 diabetes from a dataset of cardiovascular risk factors, diabetes self management, acculturation, social support, depression and perceived stress collected at the investigator's laboratory at Florida International University (FIU). The purpose of the primary study was to generate hypotheses and examine associations between the persons with type 2 diabetes as compared to their controls (without diabetes). The study protocol was approved by the Institutional Review Board (IRB) at FIU. All participants read, understood and signed an informed consent form approved by the IRB.

Participants were recruited by sending letters randomized from local zip codes from two purchased mailing lists (*KnowledgeBase Marketing, Inc.*, Richardson, TX 75081.) of persons with and without type 2 diabetes. Trained interviewers screened respondents for heritage and diabetes status. Participants were selected from those responded to the mailings and met an initial screening of the inclusion criteria. Half the persons with type 2 diabetes were chosen first; then, those without diabetes were gender and age grouping matched to the first half of persons with diabetes. The next half of persons with diabetes was chosen and the last half of persons without diabetes was selected who matched persons with diabetes in age grouping and gender. Of the 388 respondents screened 367 subjects met the inclusion criteria.

Recruitment

KnowledgeBase Marketing, Inc provided mailing lists of Cuban Americans from Miami-Dade and Broward Counties. Approximately ten thousand letters were mailed. Three percent (N = 300) were undeliverable due to "unknown addresses". Respondents made contact by telephone and they were interviewed by staff for eligibility. An appointment was set for eligible respondents to meet at the investigator's Human Nutrition laboratory at Florida International University (FIU). The screening involved a standard questionnaire administered by telephone in either Spanish or English. This initial assessment determined eligibility based on self-reported Cuban heritage and diabetes status. A series of questions based on language indicators were asked by a trained interviewer to establish Cuban heritage. When the final sample size was achieved at 4% (N = 388) further recruitment was terminated. Of the 388 candidates who responded, 18 did not qualify and were excluded for the following reasons: two were not of Cuban heritage; seven had other chronic illnesses; and nine could not be matched by age grouping. There were complete data sets for 365 participants (190 with and 175 without type 2 diabetes).

The inclusion criteria were: a) Cuban Americans, self-reported with type 2 diabetes diagnosed by a physician for the group with diabetes, b) age ≥ 30 years, male and female, c) not pregnant or lactating, d) no thyroid disorders, and e) no major psychiatric disorders. For inclusion, controls met the same criteria as subjects with type 2 diabetes, except they were free of any type of diabetes. After enrollment and blood collection, persons found to have type 2 diabetes were removed from

the control group and added to the group with diabetes. They were given a letter with their laboratory report and advised to see their physician.

Data collection procedures

The demographics were collected in group settings. The protocol was explained and the IRB approved, informed consent was signed by each participant. Appointments were made for groups of participant weekly until a quota, based on a sample size determination, was reached and all data were collected. All written materials were provided in English or Spanish except for the food frequency questionnaire (FFQ). Trained bilingual interviewers were available to aid in the translation of the FFQ. Each participant was assigned a unique personal identification number (PIN) to ensure confidentiality. Data were collected from each participant in the principal investigator's laboratory. Blood collection and anthropometrics were conducted individually and privately with fully equipped blood collection and biometric measurement rooms. Measurements used for this study are described below.

Dietary and biometric measurements

All dietary variables were collected using the semi-quantitative food frequency questionnaire (FFQ) developed by Walter C. Willett. Food frequency questionnaires have been shown to be valuable tools in assessing long-term dietary intake. This FFQ has been extensively validated and standardized in several multiethnic population-based prospective and cross-sectional studies including Cuban Americans [15,16]. The FFQ is intended to measure usual and long-term intakes of foods that are relevant determinants of chronic diseases like T2D and CVD [17]. Participants self-reported average consumption of various foods over the past year, and chose frequencies ranging from 'never' to 'six or more servings' per day. Even though the questionnaire was self-administered, trained bilingual (English/Spanish) interviewers were available to answer questions. Average daily percent of calories from carbohydrate, type of carbohydrate (i.e. fructose, sucrose) and grams of fiber were the variables used from the results of the FFQ.

To estimate the quantity of carbohydrate consumed, the percent of calories from carbohydrate was calculated by daily intake of grams of carbohydrate multiplied by the energy unit of carbohydrate (4 kcal/g), divided by total daily calories (Kcal) then multiplied by 100. The glycemic load values, which estimates the quality and quantity of carbohydrate in the overall diet, were calculated using the glycemic index value (glucose was the reference) of the particular food multiplied by the grams of carbohydrate per serving of the food [16]. These values were each multiplied by the frequency of consumption, summed and divided by 100 to produce the dietary glycemic load (GL). The dietary glycemic index (GI) was then calculated by dividing the GL by the total amount of carbohydrate consumed in grams. To estimate carbohydrate type, fructose and sucrose divided by Kcal and multiplied by 1000.

Anthropometrics were taken in a private room adjoining the laboratory, for the participants' comfort and privacy. Height measurements were taken with the subject standing erect without shoes and for weight wearing light clothes using a SECA balance scale (Seca Corp, Columbia, MD). Body mass index (BMI) was calculated using formula weight/height^2 (kg/m^2) after converting weight in pounds to kilograms and height measured in inches to meters. Waist circumference to the nearest 0.1 cm was measured horizontally with a non-stretchable measuring tape placed midway between the 12th rib and iliac crest at minimal respiration.

Supine blood pressure was measured twice by auscultatory method and an average of the two readings was taken as the (SBP) and (DBP) in mmHg. PP was then calculated as the difference between SBP and DBP; (PP = SBP-DBP). Trained investigators measured blood pressure of each participant using a random zero sphygmomanometer (Tycos 5090-02 Welch Allyn Pocket Aneroid Sphygmomanometer, Arden, NC) and a stethoscope (Littmann Cardiology, 3M, St. Paul, MN). The appropriate adult-size arm cuff was used in order to record accurate measures. The size is determined by the width of the inflatable bladder, which is about 40% of the circumference from the midpoint of the arm. The measurements were read with the forearm and the manometer at the level of the heart. The cuff was placed at least one inch above the antecubital space on the right arm. The stethoscope head was applied firmly on the brachial artery of a slightly flexed arm. The cuff was inflated until the manometer indicator exceeded >180 mm Hg. Then pressure was released at a rate of 2 mm Hg per second. As the pressure fell, systolic blood pressure was determined at the point where the initial pulse sound was heard; Korotkoff's fifth phase was used to determine diastolic blood pressure. The first reading was measured at the beginning of the data collection, after the subject has been seated for 5 minutes rest, from the upper arm at the level of the heart. Two separate readings were taken from each participant at least 10 minutes apart and the average was recorded and used for data analysis.

A 20 ml sample of venous blood was collected from each subject after overnight fasting (at least 8 hours), by a certified phlebotomist. Each participant was instructed to refrain from smoking, drinking any beverages other than water; and to refrain from unusual physical activity in the morning before blood collection. For this study, blood samples were drawn into a vacutainer Serum Separator tubes (SST) and upon coagulation (30-45 minutes after venipuncture) were centrifuged at 1100 RCF (2,500 rpm) for 30 minutes. The serum was transferred into separate plastic tubes for glucose and lipid profile analysis. Blood samples were analyzed and reported by Laboratory Corporation of America (LabCorp®, Miami FL, USA).

Statistical analysis

Continuous variables were tested by Q-Q plots for normality. Variables that were not linear were transformed to achieve linearity. Database management and statistical analyses were performed with IBM SPSS version 18 (SPSS Inc, Chicago, IL). Descriptive statistics were performed and percentages were calculated for the demographic characteristics as a whole and stratified by diabetes status. Group means (student t-tests) were conducted to evaluate differences by diabetes status. Pearson correlation analyses were calculated to determine the relationship between PP and the variables of interest.

Variations in PP were assessed using the general linear model. Models with PP as the dependent variable were performed to evaluate the difference between participants with and without diabetes after adjusting for clinically significant covariates and their interactions. A binary variable for high PP (>40) versus normal was constructed for logistic regression analysis to determine the odds of persons with diabetes having a higher PP than persons without diabetes. General linear models were performed to assess the effect of carbohydrate, sugar type, and GL on PP by diabetes status. All models included age and gender. Covariates for full models included all potential confounders such as Kcal, smoking, BMI, sodium, hypertension medications, cholesterol medication, alcohol intake and energy intake. Final models contained all covariates with p-values ≤ 0.2. Significance was set at p < 0.05.

Table 1: Characteristics of the study participants.

Variables N =367	With Diabetes n=190	Without Diabetes n=177	p-value
Male	72(37.9)	59(33.3)	0.831
Female	118(62.1)	118(64.3)	
Age (years)	65.2±11.8	62.6±11.4	0.036
WC	105.5±14.5	100.0±12.4	<0.001
BMI	31.6±6.5	29.9±5.0	0.008
HTN (yes)	126(56.0)	99(44.0)	0.041
PP mm Hg	52.9±12.4	48.7±13.3	0.002
PP >40	159(83.7)	123(70.3)	0.002
SBP mm Hg	133±15.4	130±19.1	0.229
DBP mm Hg	79.7±9.1	81.7±9.4	0.040
Chol mg/dL	188±52.0	206±45.3	<0.001
TG	177±107	147±165	0.376
HDL-C	48.9±14.8	56.2±14.7	<0.001
LDL-C	99.0±45.0	114±46.5	0.002
% CHO	46.6±9.4	51.5±5	<0.001
Fiber (grams)	23.0±10.1	23.3±10.2	0.733
% Kcal Protein	19.8±4.0	17.3±3.1	<0.001
% Kcal Fat	34.2±7.5	31.3±5.9	<0.001
% Kcal SFA	10.7±2.6	10.4±2.5	<0.001
Current smoker	31(16.3)	30(16.9)	0.871
Cholesterol Meds	41(21.6)	33(18.6)	0.490
HTN Meds	103(54.2)	79(44.6)	0.067

Note: Binary values are given as N (%) within diabetes status and compared by the Chi-square test. The Pearson Chi-square P value is reported. Continuous variables are measured by the independent sample group mean test and reported as means ± standard deviation. P values are considered two-tailed and calculated at the 95th percentile.

Abbreviations: PP = arterial pulse pressure; GL = glycemic load; Kcal = kilocalories; Cholesterol Meds= cholesterol-lowering medications; HTN Med= hypertension medications; HTN = hypertension; BMI=body mass index; WC =waist circumference; SBP=systolic blood pressure; DBP=diastolic blood pressure; TC=total cholesterol; TG=triglycerides; HDL-C= high-density lipoprotein cholesterol; LDL-C=low-density lipoprotein cholesterol.

Results

The characteristics of the participants by diabetes status are shown in Table 1. Majority of the participants was female (over 60%); however, there was no significant difference in gender across diabetes status. Persons with diabetes were approximately 2 ½ years older, had higher WC, BMI and PP measurements than those without diabetes. A significantly higher percentage of persons with diabetes had lower HDL-C, and had a higher percentage of HTN than participants without diabetes. Persons without diabetes consumed a higher percentage of their calories from carbohydrate than persons with diabetes; but, there were no significant differences in daily fiber intakes.

The effect of diabetes status on PP was assessed with PP as a continuous, independent variable by the General Linear Model. Covariates considered included NSAIDs, hypertension, current

smoker, education, alcohol, sodium and BMI. The final model { $F = (5, 362) = 20.24$, $p < 0.001$; $R^2 = 0.182$ (Adjusted $R^2 = 0.173$)} contained age ($p < 0.001$), gender ($p = 0.215$), and hypertensive medications ($p = 0.003$). Individuals with type 2 diabetes were positively associated with PP { $B = 0.064(0.018, 0.111)$, $SE = 0.024$, $p = 0.007$ } (data not shown). The

same variables predicted the binary variable for PP (high >40) by logistic regression analyses. The odds of persons with diabetes having high PP was 1.85 (1.09, 3.13), $p = 0.023$. The model explained 16.5% of the estimated variance of high PP (Nagelkerke R^2) with 77.7% of the cases and controls classified correctly (data not shown).

Table 2: Effect of diabetes status and glycemic load on Arterial Pulse Pressure.

General Linear Model, dependent variable –ln PP					
Parameter	B	SE	p-value	95% Confidence Interval	
				Lower Bound	Upper Bound
Without Diabetes	0.035	0.048	0.466	-0.059	0.129
Female	-0.027	0.025	0.270	-0.076	0.021
No HTN meds	-0.073	0.024	0.003	-0.121	-0.026
Age	0.007	0.001	<0.001	0.004	0.009
GL-Q1	0.079	0.047	0.093	-0.013	0.171
GL-Q2	0.127	0.050	0.012	0.028	0.226
GL-Q3	0.071	0.052	0.172	-0.031	0.172
GL-Q4 (reference)					
Diabetes by GL-Q	$F = 3.83$; $p = 0.010$, estimated power = 81.8%, with $\alpha = 0.05$				

Model $F = (10, 354) = 9.43$, $p < 0.001$; R^2 Squared = 0.210 (Adjusted $R^2 = 0.188$)

Abbreviations: PP = arterial pulse pressure; HTN meds= hypertension medications; B=coefficient; SE=standard error, GL = glycemic load; Q = quartiles

Table 3: Effect of glycemic load stratified by diabetes status on Arterial Pulse Pressure.

Logistic regression, stratified by diabetes status: dependent variable PP>40					
Without diabetes	Wald (df)	SE	p-value	OR	95% CI
GL-Quartiles	6.52(3)	-	0.089	-	-
GL-Q1	1.88	0.57	0.170	0.46	0.12, 1.39
GL-Q2	3.92(1)	0.49	0.048	0.38	0.15, 0.99
GL-Q3	0.03(1)	0.50	0.855	1.10	0.41, 2.94
Gender-male	1.71(1)	0.41	0.191	0.58	0.26, 1.31
Age	11.7(1)	0.02	0.001	1.06	1.03, 1.10
HTN Med	2.49(1)	0.40	0.115	0.53	0.24, 1.17
Model: without diabetes: $\chi^2(n=175, 6) = 32.1$, $p < 0.001$, Nagelkerke $R^2 = 0.238$					
With diabetes	Wald (df)	SE	p-value	OR	95% CI
GL-Quartiles	8.56(3)	-	0.036	-	-
GL-Q1	6.84(1)	0.56	0.009	4.33	1.44, 13.0
GL-Q2	5.28(1)	0.61	0.022	4.10	1.23, 13.7
GL-Q3	2.70(1)	0.58	0.100	2.58	0.83, 7.96
Gender-male	2.04(1)	0.45	0.154	0.53	0.22, 1.27
Age	2.01 (1)	0.017	0.113	1.03	0.99, 1.06
HTN med	3.18 (1)	0.43	0.075	0.47	0.20, 1.08
Model: diabetes: $\chi^2(n=190, 6) = 17.2$, $p = 0.009$, Nagelkerke $R^2 = 0.147$					

Abbreviations: PP= arterial pulse pressure; HTN meds= hypertension medications; B=coefficient; SE=standard error; GL = glycemic load; Q = quartiles

The type of carbohydrate composition of the diet, diabetes status and its effect on PP was examined by general linear models. First, GL was examined by a full model with all potential confounders: age, gender, current smoker, education, BMI, HTN medications. The covariates smoking, education and BMI were not retained, since $p > 0.2$ and they did not improve the model. The final model is presented in Table 2. Glycemic load differed by diabetes status. Table 3 presents the effect of GL on high PP (>40), stratified by diabetes status. Individuals with type 2 diabetes with GL in the 1st and 2nd quartiles were 4 times more likely to have high PP; whereas this relationship was not significant for persons without diabetes.

General Linear models with PP as the dependent variable were conducted for fructose per Kcal and sucrose per Kcal with main and interactive effects of diabetes status. Covariates included age, gender, current smoker, education and HTN medications. Neither the reduced, nor the full models were significant. Several models were conducted to assess the effect of percent calories from carbohydrate considering diabetes status, age and gender on PP and no significant effect was found for percent calories from carbohydrate or its interaction with diabetes. Similar analyses were performed with percent of calories from protein, total fat, saturated fat; total fiber, monounsaturated fatty acids and total polyunsaturated fatty acids. Macronutrient composition, fiber, and type of fat did not affect PP, nor did their interactions with diabetes status.

Discussion

Although prospective clinical trials found reduction in blood pressure for lower carbohydrate higher protein diets for individuals with hypertension [18-20], they did not include persons with diabetes, nor did they measure a risk factor for arterial stiffness.

The present study is the first to examine and compare PP in persons with and without diabetes for a Cuban American sample. Even though approximately 65% of the population was female; there was no gender by diabetes status interaction and effect on PP. This finding is in accordance for a Chinese population, where no differences between genders for arterial stiffness were found [21]. On the contrary, ethnicity and gender were associated with arterial stiffness measured by PP; however, the participants were normotensive and young adults [22].

This study demonstrated that higher PP was independently associated in individuals, with type 2 diabetes; whereby, they had a significantly higher PP than those without diabetes. Our results suggested that persons with type 2 diabetes had greater stiffening of their arteries. This finding was in accordance with research that has suggested a close relationship between diabetes mellitus and increased arterial stiffening [23-25]. Contrary to a few studies that failed to detect any significant increase in the stiffness of the elastic artery in patients with diabetes [26,27], our results were aligned to Miyagi et al. [28], who demonstrated that PP was significantly associated with having diabetes. It is important to note that PP is a risk factor for arterial stiffness and that there are direct measures such as noninvasive ultrasonic imaging [23] and echo tracking systems [24]. However, our results using PP index were nonetheless similar to the results using direct measures. Moreover, several studies have used the PP index as an indicator of arterial stiffness with success [29-31].

It is generally acknowledged that there are many predisposing factors affecting the development of chronic diseases such as diabetes mellitus and CVD. Among these overlapping and concomitant factors are genetics, age, gender, diet, physical inactivity, drug interactions,

socio-economic status, stress, education and many other biological, psychological and social factors; which may be contributing to the remainder of the variance in PP. These interactions are difficult to assess and the mechanism is usually complex as they work synergistically with other major risk factors to exacerbate the condition.

Diabetes is an independent risk factor for CVD and it has been established that when patients with diabetes develop vascular abnormalities they sustain a worse prognosis for survival than do CVD patients without diabetes [26-28]. Our study suggests that PP should also be considered as an important index along with other measures of risk assessment like cigarette smoking, elevated blood pressure, serum lipids levels and hyperglycemia. Moreover, our results support the use of PP as an important risk assessment tool for individuals with diabetes. We reported almost twice the odds of higher PP for those with than those without diabetes. Arterial stiffness, a non-invasive measure and a risk factor for arterial stiffness, would provide evidence indicative of the development of atherosclerosis before the presentation of symptoms of CVD.

Notwithstanding are the effects of diet composition on blood pressure and arterial stiffness for persons with and without diabetes. Higher insulin response and lower plasma glucose responses for persons with type 2 diabetes were found for a higher protein (30% compared to 15%), lower carbohydrate diet (55% compared to 40 %) over a 5 week period [32]; however, the investigators did not measure blood pressure. Systolic blood pressure was lowered by 1.3 mm Hg for non-hypertensive adults and 2.9 mm Hg for hypertensive adults who consumed the low-carbohydrate diet as compared to controls without weight loss for a six-week trial [33]. Other prospective trials found reductions in blood pressure for lower carbohydrate, higher protein diets for persons with hypertension [18-20].

Studies comparing effect of low GL versus low fat diets with blood pressure in obese adults are few and have drawn different conclusions ranging from lower blood pressure with the low GL-diet [34] to no difference in blood pressure [35]. There is similar controversy between high-protein versus a high-fiber, high-carbohydrate diet for normotensive adults. In a randomized 8-week trial, for obese women, diastolic blood pressure was reduced more with a high protein diet than the high-fiber, high-carbohydrate diet [36]. On the other hand, no difference in blood pressure was found for overweight/obese adults with type 2 diabetes in a 12-month trial for the high-carbohydrate, high fiber diet or the high-protein diet [37].

Diet is a modifiable risk factor for CVD. Our study found persons with diabetes who consumed higher GL may have a lower risk of CVD (greater probability for lower PP). The results were not significant for participants without diabetes. Ethnically suitable dietary plans for adults with type 2 diabetes may be different from the diets of healthy individuals. The American Diabetes Association (ADA) recommends monitoring carbohydrate intake by counting carbohydrate serving for persons with diabetes [38]. The narrower standard deviation of carbohydrate intake for participants with diabetes, in this study, suggests persons with diabetes may be more inclined to measure their carbohydrate intake as compared to those without diabetes. The ADA further suggested estimation and intake of low glycemic index or glycemic load of foods (a ranking of how foods affect blood glucose) may provide additional benefits. Percent of calories from saturated fat in the diet has been recommended by the ADA to be not more than 7%; yet, both persons with and without diabetes in this study consumed 10% or more of calories from saturated fat. The cardioprotective dietary

recommendation for daily requirements of fiber intake (25-30 grams) was not met by either group [39].

In this study, glycemic load but not carbohydrate type were associated with PP for persons with diabetes and this association was not observed in persons without diabetes. Although the quality and quantity of carbohydrate intake, captured by glycemic load (GL), has been reported to reduce the metabolic syndrome in a national prospective study [40], blood pressure was not significantly lowered. In contrast, a low-glycemic load diet improved blood pressure more than those in a low-fat diet in a randomized parallel-design of overweight young adults [34]. Dietary guidelines for low carbohydrate diets to improve blood glucose may be at the expense of arterial health for some persons with diabetes. Glycemic load was inversely associated with PP for participants with type 2 diabetes. Since GL is a measure of quantity and quality of carbohydrate, these findings suggest that persons with diabetes may be restricting carbohydrate intake in an attempt to follow medical advice for dietary guidelines.

There are several limitations of this study. First, since this is a single time point study, causality cannot be assumed. Second, the sample of Cuban Americans may not be representative of Cuban Americans living in Florida or the United States. Third, although PP has been considered a risk factor for arterial stiffness, we did not use pulse wave pressure, which has been considered the gold standard [41]. Finally, the source of fructose was not determined. A strength of this study was that PP measurement could be easily taken by auscultatory method (a fitted cuff, stethoscope and sphygmomanometer); whereas pulse wave pressure requires a medical setting and specific instruments. Additionally, this study addressed a gap in the literature regarding carbohydrate quality and a risk factor for arterial stiffness by diabetes status.

Conclusions

Elevated pulse pressure and diet are modifiable risk factors. Our study suggests that PP should be considered as a technique in developing a plan towards risk reduction. This measurement may have particular usefulness for persons with type 2 diabetes. Prospective studies are needed to clarify whether this technique has value in the early detection and screening of CVD. The impact of carbohydrate intake and blood pressure for persons with and without diabetes has yet to be resolved. Future studies of dietary composition and diabetes status association with PP are needed. It is possible that the relationship among carbohydrate type and amount (GL) with diabetes status and PP was due to individuals with diabetes following medical advice and dietary guidelines. The findings of this study suggest clinicians take into consideration how the medical condition, ethnicity and the current diet is associated with PP before developing a medical nutrition therapy plan in collaboration with the client.

Competing Interests

The authors have no competing interests. The funding sources had no role in the design, conduct, analyses, and the final report of the study.

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References

1. National Institute of Diabetes and Digestive and Kidney Diseases (2008) National Diabetes Statistics, 2007 fact sheet. Bethesda, MD; U.S. Department of Health and Human Services, National Institutes of Health.
2. Engelgau MM, Geiss LS, Saaddine JB, Boyle JP, Benjamin SM, et al. (2004) The Evolving Diabetes Burden in the United States. *Ann Intern Med* 140: 945-950.
3. Centers for Disease Control and Prevention (2008) National diabetes fact sheet: General information and national estimates on diabetes in the United States, 2007. Atlanta, GA; U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.
4. Howard BV, Rodriguez BL, Bennett PH, Harris MI, Hamman R, et al. (2002) Prevention Conference VI: Diabetes and cardiovascular disease: Writing group I: Epidemiology. *Circulation* 105: e132-e137.
5. Office of Minority Health Disparities, 2007. Highlights in Minority Health and Health Disparities.
6. US Census Bureau. Current Population Survey, 2002 (2002) [database online]. Washington, DC; US Census Bureau.
7. Domanski M, Norman J, Wolz M, Mitchell G, Pfeffer M (2001) Cardiovascular risk assessment using pulse pressure in the first national health and nutrition examination survey (NHANES I). *Hypertension* 38: 793-797.
8. Assmann G, Cullen P, Evers T, Petzinna D, Schulte H (2005) Importance of arterial pulse pressure as a predictor of coronary heart disease risk in PROCAM. *Eur Heart J* 26: 2120-2126.
9. Miwa Y, Tsushima M, Arima H, Kawano Y, Sasaguri T (2004) Pulse pressure is an independent predictor for the progression of aortic wall calcification in patients with controlled hyperlipidemia. *Hypertension* 43: 536-540.
10. Danchin N, Benetos A, Lopez-Sublet M, Demicheli T, Safer M, et al. (2004) Aortic pulse pressure is related to the presence and extent of coronary artery disease in men undergoing diagnostic coronary angiography: a multicenter study. *Am J Hypertens* 17:129-133.
11. Millar JA, Lever AF, Burke V (1999) Pulse pressure as a risk factor for cardiovascular events in the MRC Mild Hypertension Trial. *J Hypertens* 17: 1065-1072.
12. Weber T, Auer J, O'Rourke MF, Kvas E, Lassnig E, et al. (2004) Arterial stiffness, wave reflections, and the risk of coronary artery disease. *Circulation* 109: 184-189.
13. Schram MT, Kostense PJ, van Dijk RA, Dekker JM, Nijpels G, et al. (2002) Diabetes, pulse pressure and cardiovascular mortality: the Hoorn Study. *J Hypertens* 20: 1743-1751.
14. Cockcroft JR, Wilkinson IB, Evans M, McEwan P, Peters JR, et al. (2005) Pulse pressure predicts cardiovascular risk in patients with type 2 diabetes mellitus. *Am J Hypertens* 18: 1463-1467.
15. Nath SD, Huffman FG (2005) Validation of a semiquantitative food frequency questionnaire to assess energy and macronutrient intakes of Cuban Americans. *Int J Food Sci Nutr* 56: 309-314.
16. Willett WC, Sampson L, Stampfer MJ, Rosner B, Bain C, et al. (1985) Reproducibility and validity of a semiquantitative food frequency questionnaire. *Am J Epidemiol* 122: 51-65.
17. Willett W (1998) *Nutritional Epidemiology*. Oxford University Press, New York.
18. Burke V, Hodgson JM, Beilin LJ, Giangulioi N, Rogers P, et al. (2001) Dietary protein and soluble fiber reduce ambulatory blood pressure in treated hypertensives. *Hypertension* 38: 821-826.
19. He J, Gu D, Wu X, Chen J, Duan X, et al. (2005) Effect of soybean protein on blood pressure: A randomized, controlled trial. *Ann Intern Med* 143: 1-9.
20. Hodgson JM, Vurke V, Beilin LJ, Puddey IB (2006) Partial substitution of carbohydrate intake with protein intake from lean red meat lowers blood pressure in hypertensive persons. *Am J Clin Nutr* 83: 780-787.
21. Avolio AP, Deng FQ, Li WQ, Luo YF, Huang ZD, et al. (1985) Effects of aging on changing arterial distensibility in populations with high and low prevalence

- of hypertension: comparison between rural and urban communities in China. *Circulation* 71(92): 202-210.
22. Hlaing WH, Prineas RJ (2006) Arterial stiffness variations by gender in African-American and Caucasian Children. *J Natl Med Assoc* 98: 181-189.
23. Salomaa V, Riley W, Kark JD, Nardo C, Folsom AR (1995) Noninsulin dependent diabetes mellitus and fasting glucose and insulin concentrations are associated with arterial stiffness indexes: the ARIC study. *Circulation* 91: 1432-1443.
24. Emoto M, Nishizawa Y, Kawagishi T, Maekawa K, Hiura Y, et al. (1998) Stiffness indexes beta of the common carotid and femoral arteries are associated with insulin resistance in NIDDM. *Diabetes Care* 21: 1178-1182.
25. Vavuranakis M, Stefanadis C, Triandaphyllidi E, Toutouzas K, Toutouzas P (1999) Coronary artery distensibility in diabetic patients with simultaneous measurements of luminal area and intracoronary pressure: evidence of impaired reactivity to nitroglycerin. *J Am Coll Cardiol* 34: 1075-1081.
26. Kool MJ, Lambert J, Stehouwer CD, Hoeks AP, Struijker Boudier HA, et al. (1995) Vessel wall properties of large arteries in uncomplicated IDDM. *Diabetes Care* 18: 618-624.
27. McVeigh G, Brennan G, Hayes R, Cohn J, Finkelstein S, et al. (1993) Vascular abnormalities in non-insulin dependent diabetes mellitus identified by arterial waveform analysis. *Am J Med* 95: 424-430.
28. Miyagi T, Muratani H, Kimura Y, Fukiyama K, Kawano Y, et al. (2002) Increase in pulse pressure relates to diabetes mellitus and low HDL cholesterol, but not to hyperlipidemia in hypertensive patients aged 50 years or older. *Hypertens Res* 25: 335-341.
29. Safar ME (1989) Pulse pressure in essential hypertension: clinical and therapeutical implications. *J Hypertens* 7: 769-776.
30. Darme B, Girerd X, Safar M, Cambien F, Guize L (1989) Pulsatile versus steady component of blood pressure: a cross-sectional analysis and a prospective analysis on cardiovascular mortality. *Hypertension* 13: 392-400.
31. Liao D, Arnett DK, Tyroler HA, Riley WA, Chambless LE, et al. (1999) Arterial stiffness and the development of hypertension. The ARIC study. *Hypertension* 34: 201-206.
32. Gannon MC, Nuttall FQ (2004) Effect of a high-protein, low-carbohydrate diet on blood glucose control in people with type 2 diabetes. *Diabetes* 53: 2375-2382.
33. Appel LJ, Sacks FM, Carvey VJ, Obarzanek E, Swain JF, et al. (2005) Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids: Results of the OmniHeart Randomized Trial. *JAMA* 294: 2455-2466.
34. Pereira MA, Swain J, Goldfine AB, Rifai N, Ludwig DS (2004) Effect of a low-glycemic load diet on resting expenditure and heart disease risk factors during weight loss. *JAMA* 292: 2482-2490.
35. Ebbeling CB, Leidig MM, Sinclair KB, Seger-Shippie LB, Feldman HA, et al. (2005) Effects of an ad libitum low-glycemic load diet on cardiovascular disease risk factors in obese young adults. *Am J Clin Nutr* 81: 976-982.
36. Te Morenga LA, Levers MT, Williams SM, Brown RC, Mann J (2011) Comparison of high protein and high fiber weight-loss diets in women with risk factors for the metabolic syndrome: a randomized trial. *Nutrition Journal* 10: 40.
37. Larsen RN, Mann NJ, Maclean E, Shaw JE (2011) The effect of high-protein, low-carbohydrate diets in the treatment of type 2 diabetes: a 12 month randomized control trial. *Diabetologia* 54: 731-740.
38. American Diabetes Association (2010) Standards of Medical Care in Diabetes – 2010. *Diabetes Care* 33: S11-S61.
39. American Dietetic Association (2006) American Association publishes evidence-based nutrition practice guidelines for registered dietitians.
40. McKeown NM, Meigs JB, Liu S, Wilson PW, Jacques PF (2002). Whole-grain intake is favorably associated with metabolic risk factors for type 2 diabetes and cardiovascular disease in the Framingham Offspring Study. *Am J Clin Nutr* 76: 390-398.
41. Lim HS, Lip GY (2008) Arterial stiffness: beyond pulse wave velocity and its measurement. *J Hum Hypertens* 22: 656-658.